

Missouri Department of Health and Senior Services
Division of Community and Public Health
Section for Environmental Public Health
Bureau of Environmental Epidemiology
Environmental Public Health Tracking Program



MISSOURI EPHT DATA & STATISTICAL GUIDE





Published By:

**Missouri Department of Health and Senior Services (DHSS)
Division of Community and Public Health (DCPH)
Section for Environmental Public Health (SEPH)
Bureau of Environmental Epidemiology (BEE)
Environmental Public Health Tracking (EPHT) Program
P.O. Box 570
Jefferson City, Missouri 65102-0570**

Telephone: 573.751.6102

Fax: 573.526.6946

E-mail Address: EPHTN@health.mo.gov

Web Site: <http://www.health.mo.gov/EPHT/>

In partnership with the Centers for Disease Control and Prevention (CDC)
Environmental Public Health Tracking Branch
Cooperative Agreement #5U38EH000188-05

Web Published in Jefferson City, Missouri
October 2014



Table of Contents

Point of Contact.....	1
Media Contact.....	1
Data Partners.....	2
Data Sources and References.....	4
Measures.....	7
Indicators and Nationally Consistent Data and Measures.....	11
Metadata.....	23
Diseases and Conditions Reportable In Missouri.....	24
Missouri’s Statutes, Rules, and Regulations Pertaining to Specific Data.....	25
Frequently Asked Data Questions.....	29
Acronyms Used in this Guide.....	33
Guide Development and Maintenance.....	36
Appendixes:	
A. Centers for Disease Control and Prevention Standards for Nationally Consistent Data and Measures within the Environmental Public Health Tracking Network [Version 3.0 June 20, 2013]	
B. National EPHT NCDM “How-To” Guides	
C. Tracking Network Metadata Content Guidance Document	
D. Missouri Code of State Regulations (CSR) – 19 CSR 20.20, Reporting Communicable, Environmental and Occupational Diseases	
E. List of Diseases and Conditions Reportable In Missouri	

Point of Contact

The Missouri Department of Health and Senior Services' (DHSS) Environmental Public Health Tracking (EPHT) Program is responsible for obtaining and maintaining the data sets and analyses located on the Missouri EPHT Network Portal.

For more information, please contact:

Roger W. Gibson, MPH
Environmental Public Health Tracking Program Manager

Missouri Department of Health and Senior Services
Division of Community and Public Health
Section for Environmental Public Health
Bureau of Environmental Epidemiology
930 Wildwood Drive
P.O. Box 570
Jefferson City, Missouri 65102-0570

Telephone: 573.751.6102
Fax: 573.526.6946

E-mail Address: roger.gibson@health.mo.gov
Web Site: <http://www.health.mo.gov/EPHT/>

Media Contact

For media inquiries, please contact:

Ryan Hobart
Communications Director

Missouri Department of Health and Senior Services
Office of Public Information
912 Wildwood Drive
P.O. Box 570
Jefferson City, Missouri 65102-0570

Telephone: 573.751.6062
Fax: 573.751.6010

E-mail Address: ryan.hobart@health.mo.gov
Web Site: <http://www.health.mo.gov>

Data Partners

The Missouri EPHT Network Portal hosts, displays, and links to data from a wide variety of program partners. These partners include:

- Missouri Department of Health and Senior Services (DHSS) bureaus and programs, such as:
 - Bureau of Environmental Epidemiology (BEE)
 - Health and Risk Assessment Program (HRAP)
 - Healthy Indoor Environments (HIE)
 - Environmental Surveillance (ES)
 - Bureau of Health Care Analysis & Data Dissemination (BHCADD)
 - Bureau of Vital Statistics (BVS)
 - Bureau of Vital Records (BVR)
 - Bureau of Environmental Health Services (BEHS)
 - Office of Epidemiology for Public Health Practice
 - Bureau of Cancer and Chronic Disease Control (BCCDC)
- Missouri Department of Natural Resources (DNR) units and programs, such as:
 - Division of Environmental Quality (DEQ)
 - Air Pollution Control Program (APCP)
 - Environmental Services Program (ESP)
 - Hazardous Waste Program (HWP)
 - Land Reclamation Program (LRP)
 - Solid Waste Management Program (SWMP)
 - Water Protection Program (WPP)
 - Public Drinking Water Branch (PDWB)
 - Water Pollution Control Branch (WPCB)
 - Regional and Satellite Offices
- Missouri Department of Conservation (MDC)
- Missouri Department of Social Services (DSS) - MO HealthNet
- Missouri Department of Agriculture (MDA)
- Missouri Department of Elementary and Secondary Education (DESE)
- Missouri Department of Economic Development (DED)
- Missouri Department of Transportation (MoDOT)
- Missouri Census Data Center (MCDC)
- Missouri Housing Development Commission (MHDC)
- Missouri Cancer Registry and Research Center (MCR-ARC)
- Office of Social and Economic Data Analysis (OSED)
- Area Agencies on Aging (AAA)
- Local Public Health Agencies (LPHAs)
- Agency for Toxic Substances and Disease Registry (ATSDR)
- National Aeronautics and Space Administration (NASA)
- National Center for Environmental Health (NCEH)
- National Institute for Environmental Health Sciences (NIEHS)
- National Institute for Occupational Safety and Health (NIOSH)
- National Oceanic and Atmospheric Administration (NOAA)

- National Weather Service (NWS)
- National Program of Cancer Registries (NPCR)
- United States (US) Department of Agriculture (USDA)
- US Department of Energy (DOE)
- US Department of Health and Human Services (HHS)
 - Centers for Disease Control and Prevention (CDC)
- US Department of Housing and Urban Development (HUD)
- US Department of Interior (DOI)
 - United States Fish and Wildlife Service (USFWS)
 - United States Geological Survey (USGS)
- US Department of Transportation (DOT)
- US Environmental Protection Agency (EPA)
 - Region 7 Office - Kansas City
 - Office of Air and Radiation (OAR)
 - Office of Chemical Safety and Pollution Prevention (OCSP)
 - Office of Enforcement and Compliance Assurance (OECA)
 - Office of Environmental Information (OEI)
 - Office of Solid Waste and Emergency Response (OSWER)
 - Office of Water (OW)
- State Environmental Health Indicators Collaborative (SEHIC)
- Council of State and Territorial Epidemiologists (CSTE)
- Association of State and Territorial Health Officers (ASTHO)
- North American Association of Central Cancer Registries (NAACCR)
- Surveillance Epidemiology and End Results (SEER)

More information on Missouri EPHT partners can be obtained from the Missouri EPHT Network Portal website, <http://health.mo.gov/living/environment/epht/index.php> or by contacting the Missouri EPHT Program Manager.

Data Sources and References

The Missouri EPHT Network Portal uses multiple data sources and references to create data sets, analyses, tables, charts, graphs, and tools. Specific data sources and references by content area are detailed below:

- Air Quality:
 - Missouri DNR:
 - Air Sampling Results
 - Missouri Emissions Inventory System (MOEIS)
 - Missouri Environmental Emergency Response Tracking System (MEERTS)
 - LRP Mining Database
 - MoDOT
 - CDC:
 - National EPHT Network Portal
 - US EPA:
 - AIRNow
 - AirExplorer
 - Air Quality Index (AQI)
 - Air Quality System (AQS) Database
- Acute Myocardial Infarction Hospitalizations:
 - BHCADD:
 - Patient Abstract System (PAS)
 - Emergency Department (ED)
 - Missouri Information for Community Assessment (MICA)
 - Office of Epidemiology:
 - Behavioral Risk Factor Surveillance System (BRFSS)
 - BCCDC:
 - Heart Disease and Stroke Prevention Program
- Asthma:
 - BHCADD:
 - PAS
 - ED
 - MICA
 - Office of Epidemiology:
 - BRFSS
 - BCCDC:
 - Asthma Prevention and Control Program
 - DESE:
 - Missouri School Health Profiles
- Birth Defects:
 - BHCADD:
 - PAS
 - ED
 - MICA

- BVS
- BVR
 - Birth Defects Registry
 - Missouri Electronic Vital Records (MoEVR) System
- Office of Epidemiology:
 - BRFSS
- the Hope program (formerly Children with Special Health Care Needs)
- Cancer:
 - MCR-ARC
 - BHCADD:
 - PAS
 - ED
 - MICA
 - BVS
 - Office of Epidemiology:
 - BRFSS
 - BCCDC
- Carbon Monoxide:
 - BEE
 - ES - Carbon Monoxide Surveillance
 - BHCADD:
 - PAS
 - ED
 - MICA
 - BVS
 - BVR
 - MoEVR
- Childhood Lead Poisoning:
 - BEE
 - HIE Program
 - Childhood Lead Poisoning Prevention Program (CLPPP)
 - Adult Blood Lead Epidemiology and Surveillance (ABLES)
 - BHCADD:
 - PAS
 - ED
 - MICA
 - BVS
- Housing
 - MCDC
 - OSEDA
 - MHDC
 - Point in Time Homeless Population Counts
 - US Census Bureau
 - US HUD
- Poverty
 - MCDC

- DED
- OSEDA
- US Census Bureau

- Reproductive Outcomes:
 - BHCADD:
 - PAS
 - ED
 - MICA
 - BVS
 - BVR
 - Birth Defects Registry
 - MoEVR
 - Office of Epidemiology:
 - BRFSS
 - Women's Health

- Vital Statistics:
 - BHCADD:
 - PAS
 - ED
 - MICA
 - BVS
 - BVR
 - MoEVR

- Water Quality:
 - Missouri DNR:
 - Safe Drinking Water Information System (SDWIS)
 - Aquatic Invertebrate Sampling (AQUID)
 - Well Information Management System (WIMS)
 - MDC
 - MDA
 - BEE
 - HRAP
 - HIE
 - ES
 - Private Drinking Water
 - BEHS
 - On-Site Wastewater Treatment Program
 - US EPA

More information on Missouri EPHT data sources can be obtained from the Missouri EPHT Network Portal website, <http://health.mo.gov/living/environment/epht/index.php> or by contacting the Missouri EPHT Program Manager.

Measures

¹mea·sure *noun* \ˈme-zhər, ˈmā-\

Definition of MEASURE

1 a (1) : an adequate or due portion (2) : a moderate degree; *also* : MODERATION, TEMPERANCE (3) : a fixed or suitable limit : BOUNDS <rich beyond *measure*>

b : the dimensions, capacity, or amount of something ascertained by *measuring*

c : an estimate of what is to be expected (as of a person or situation)

d (1) : a *measured* quantity (2) : AMOUNT, DEGREE



- Definition provided by Merriam-Webster®

Analyzing raw data to create measures allows values to be calculated and assigned to each condition or situation. These values assist in monitoring and evaluating the potential future risk, as well as the effectiveness of interventions and preventative actions. These values usually appear as summary characteristics or statistics; such as a sum, percentage, or rate and are commonly known as measures. The EPHT Network creates measures for each indicator within each content area.

In general, measures are commonly used for:

- Incidence – the rate of occurrence or influence of the risk of developing some new condition within a specified period of time.
- Prevalence – the percentage of a population that is affected with a particular cause/condition at a given time period.
- Morbidity – the relative incidence of disease that alters health and quality of life.
- Mortality – the number of deaths in a given location or time period.

Explanations of the types of measures available across the EPHT Network include:

- **Counts**

A count is the sum of occurrence of a cause/condition. Counts are calculated by adding the total value for each individual, group, and/or location.

$$\text{value} + \text{value} = \text{count}$$

Example:

In Missouri during calendar year 2008, there were 7,830 males and 5,560 females that were hospitalized for an acute myocardial infarction.

$$7,830 + 5,560 = 13,390$$

The count is 13,390. This means that 13,390 people in Missouri were admitted to the hospital for an acute myocardial infarction in 2008.

- **Averages**

An average is a single value that represents the general significance of a set of unequal values. Averages are calculated by adding the values for each individual, group, and/or location, then dividing the sum by the number of values.

$$(\text{value} + \text{value}) / \text{count of values} = \text{average}$$

Example:

In Missouri there were 824 babies born with a birth defect in 2004, 965 in 2005, and 827 in 2006.

$$(824 + 965 + 827) / 3 = 872$$

The average is 872. This means that on average, 872 babies were born with a birth defect in Missouri in each year between the years 2004 and 2006.

- **Percentages**

A percentage is a part of a whole value expressed in hundredths. A percentage is calculated by dividing the value of the part by the value of the whole, then multiplying the product by 100.

$$\frac{\text{Value of Part}}{\text{Value of Whole}} \times 100 = \text{percentage}$$

The percent sign (%), is a mathematical symbol that indicates the preceding number is divided by one hundred.

Example:

In Missouri there were 8,266 people hospitalized for Asthma in 2008. In Clay County, Missouri there were 287 people hospitalized for Asthma in 2008.

$$\frac{287}{8,266} \times 100 = 3.47\%$$

The percentage is 3.47%. This means that 3.47% of all Missourians hospitalized for Asthma in 2008 are from Clay County, Missouri.

- **Rates**

A rate is a measure of the frequency of occurrence of a cause/condition. Rates are calculated by dividing a numerator by a denominator, then multiplying the product by a constant.

$$\frac{\text{numerator}}{\text{denominator}} \times \text{constant} = \text{rate}$$

The numerator is the number of people affected by a specific cause/condition. The denominator is the total number of people potentially affected by the same specific cause/condition; this is sometimes shown as the “at-risk population”. The constant is a number chosen to give the result an understandable context, typically this number is shown in thousands (e.g. 1,000: 10,000: 100,000).

Example:

In calendar year 2009, 92,697 Missouri children less than six years old were tested to determine their blood lead level. According to the US Census Bureau, 445,566 children less than six years old resided in Missouri.

$$\frac{92,697}{445,566} \times 10,000 = 2,080.43$$

The rate is 2,080.43 per 10,000. This means that for every 10,000 children less than six years old living in Missouri in 2009; 2,080 were tested for the presence of lead in their blood. This value could also be stated as approximately 1/5th or 20%.

There are several different types of rates. The most common are:

- Crude
Crude rates are the overall frequency which has not been adjusted for significant factors which might have influenced the rate. Crude rates are recommended as a summary measure when it is not necessary to adjust or accommodate for other factors.
- Adjusted
Adjusted rates have been statistically modified to eliminate the effect of different distributions in the different populations. This allows health measures such as rates of diseases and deaths to be compared between several communities with different groups. The most common factor used to adjust rates is age; other factors can also be used, such as race or gender.
- Aggregated
Aggregate rates are calculated by summing or combining multiple data elements. The practice of using aggregated data is sometimes done to increase statistical power when the amount of data may be limited. It may also be used when displaying the data element individually could potentially compromise confidentiality or provide identifying information on

a specific demographic or geography. For example, if a county had a specific health condition with only one case for a specific race or gender, that rate would be aggregated by all races and/or genders before being displayed.

A rolling rate is another example of an aggregated rate. Rolling rates are calculated across a time period that will overlap another time period. For example, data may be cumulated for the time period of 2000 – 2002, 2001 – 2003, and 2002 – 2004. Rolling rates are most often displayed in three, five, and ten year intervals.

Aggregated data is sometimes referred to as cumulative or cumulated data.

Some rates may include a confidence interval. A confidence interval is a range around a value that conveys how reliable and stable the value is. In general, the smaller the confidence interval range is the more reliable and stable the value will be. For example, a 95% confidence interval can be thought of as a range of values or interval that contains the “true value” 95% of the time. If the analysis was conducted 100 times, 95 of those times the final value would fall within the range and 5 of those times the final value would fall either higher or lower than the range. Confidence intervals are sometimes referred to as “margins of error”.

Indicators and Nationally Consistent Data and Measures

in·di·ca·tor *noun* \ˈin-də-,kā-tər\
Definition of INDICATOR

1 : one that **indicates**: as

a : an index hand (as on a dial) : **POINTER**

b (1) : **GAUGE** 2b, **DIALECT** 4a (2) : an instrument for automatically making a diagram that indicates the pressure in and volume of the working fluid of an engine throughout the cycle

2 a : a substance (as litmus) used to show visually (as by change of color) the condition of a solution with respect to the presence of a particular material (as a free acid or alkali)

b : **TRACER** 4b

3 : an organism or ecological community so strictly associated with particular environmental conditions that its presence is **indicative** of the existence of these conditions

4 : any of a group of statistical values (as level of employment) that taken together give an **indication** of the health of the economy



- Definition provided by Merriam-Webster®

EPHT Network content has been conceptually divided into hazard, exposure, and health outcome areas. Content workgroups (CWG), comprised of state and local health professionals, focused on developing measures specific to one of these groups.

Additionally, the content is divided conceptually into indicator areas. These areas represent high-level concepts within each content team domain. Within each indicator, the content workgroups developed one or more measures that represent specific ways the indicator can be measured in time and place. The majority of all measures for an indicator are reported in a single table using a standard template.

The Missouri EPHT Network Portal follows the requirements and recommendations detailed in the National EPHT's "Centers for Disease Control and Prevention Standards for Nationally Consistent Data and Measures within the Environmental Public Health Tracking Network" [Version 3.0 | June 20, 2013] and "how-to" guides (see Appendix) for the creation of indicators and Nationally Consistent Data and Measures (NCDM).

Indicators and NCDMs available on Missouri’s EPHT Network Portal, by content area, include:

<i>Acute Myocardial Infarction</i>	
Indicator	Measure
Hospitalizations for Acute Myocardial Infarction (AMI)	Annual number of hospitalizations for AMI by state and county
	Annual average daily number of hospitalizations for AMI, by month by state and county
	Annual maximum daily number of hospitalizations for AMI by month by state and county
	Annual minimum daily number of hospitalizations for AMI by month by state and county
	Annual rate of hospitalization for AMI among persons 35 and over by age group (total, 35-64, 65+) per 10,000 population by state and county
	Annual age-adjusted rate of hospitalization for AMI persons 35 and over per 10,000 population by state and county

The data used to calculate and/or compile these measures was provided by DHSS/ BHCADD.

<i>Air Quality</i>	
Indicator	Measure
Ozone – Days above regulatory standard	Annual number of days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard, by county, and Metropolitan Statistical Area (MSA) (where monitors exist)
	Annual number of person-days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard, by county, and MSA (where monitors exist)
Particulate Matter (PM_{2.5})- Days above regulatory standard	Annual percent of days with PM _{2.5} levels over the National Ambient Air Quality Standard, by county (where monitors exist)
	Annual person-days with PM _{2.5} over the National Ambient Air Quality Standard, by county (where monitors exist)

Annual PM_{2.5} Level	Annual average ambient concentrations of PM _{2.5} (based on seasonal averages and daily measurement), by county (where monitors exist)
	Annual percentage of population living in counties exceeding the National Ambient Air Quality Standard (compared to percentage of population living in counties that meet the standard, and the percentage of the population living in counties without PM _{2.5} monitors), by state

The data used to calculate and/or compile these measures was provided by the Missouri DNR, US EPA, and the CDC's National EPHT Network.

<i>Asthma</i>	
Indicator	Measure
Hospitalizations for Asthma	Annual number of hospitalizations for asthma by state and county
	Annual average daily number of hospitalizations for asthma, by month by state and county
	Annual maximum daily number of hospitalizations for asthma by month by state and county
	Annual minimum daily number of hospitalizations for asthma by month by state and county
	Annual rate of hospitalization for asthma by age group (total, 0-4, 5-14, 15-34, 35-64, and 65+) per 10,000 population by state and county
	Annual age-adjusted rate of hospitalization for asthma per 10,000 population by state and county

The data used to calculate and/or compile these measures was provided by DHSS/ BHCADD.

<i>Birth Defects</i>	
Indicator	Measure
Prevalence of Birth Defects	5 year prevalence of Anencephaly per 10,000 live births by state & county
	5 year prevalence of Spina Bifida (without anencephaly) per 10,000 live births by state & county

Prevalence of Birth Defects – <i>continued</i>	5 year prevalence of Hypoplastic Left Heart Syndrome per 10,000 live births by state & county
	5 year prevalence of Tetralogy of Fallot per 10,000 live births by state & county
	5 year prevalence of Transposition of the Great Arteries (vessels) per 10,000 live births by state & county
	5 year prevalence of Cleft Lip with or without Cleft Palate per 10,000 live births by state & county
	5 year prevalence of Cleft Palate without Cleft Lip per 10,000 live births by state & county
	5 year prevalence of Hypospadias per 10,000 live male births by state & county
	5 year prevalence of Gastroschisis per 10,000 live births by state & county
	5 year prevalence of Upper Limb Deficiencies per 10,000 live births by state & county
	5 year prevalence of Lower Limb Deficiencies per 10,000 live births by state & county
	5 year prevalence of Trisomy 21 per 10,000 live births by state & county and by maternal age at delivery (<35, ≥ 35)

The data used to calculate and/or compile these measures was provided by DHSS/BVS, BVR, and BHCADD.

Cancer	
Indicator	Measure
Incidence of Selected Cancers	5 year number of cases of Mesothelioma by state
	5 year age-adjusted incidence rate of Mesothelioma per 100,000 population by state
	Annual number of cases of Melanoma of the Skin by state
	5 year number of cases of Melanoma of the Skin by state and county
	Annual age-adjusted incidence rate of Melanoma of the Skin per 100,000 population by state
	5 year age-adjusted incidence rate of Melanoma of the Skin per 100,000 population by state and county
	Annual number of cases of Liver and Intrahepatic Bile Duct Cancer by state

Incidence of Selected Cancers – <i>continued</i>	5 year number of cases of Liver and Intrahepatic Bile Duct Cancer by state and county
	Annual age-adjusted incidence rate of Liver and Intrahepatic Bile Duct Cancer per 100,000 population by state
	5 year age-adjusted incidence rate of Liver and Intrahepatic Bile Duct Cancer per 100,000 population by state and county
	Annual number of cases of Kidney and Renal Pelvis Cancer by state
	5 year number of cases of Kidney and Renal Pelvis Cancer by state and county
	Annual age-adjusted incidence rate of Kidney and Renal Pelvis Cancer per 100,000 population by state
	5 year age-adjusted incidence rate of Kidney and Renal Pelvis Cancer per 100,000 population by state and county
	Annual number of cases of Breast Cancer in females by Age group (<50, ≥50, total) by state
	5 year number of cases of Breast Cancer in females by Age group (<50, ≥50, total) by state and county
	Annual age-adjusted incidence rate of Breast Cancer in females per 100,000 population by Age group (<50, ≥50, total) by state
	5 year age-adjusted incidence rate of Breast Cancer in females per 100,000 population by Age group (<50, ≥50, total) by state and county
	Annual number of cases of Lung and Bronchus Cancer by state
	5 year number of cases of Lung and Bronchus Cancer by state and county
	Annual age-adjusted incidence rate of Lung and Bronchus per 100,000 population by state
	5 year age-adjusted incidence rate of Lung and Bronchus Cancer per 100,000 population by state and county
	Annual number of cases of Bladder Cancer (including in situ) by state
	5 year number of cases of Bladder Cancer (including in situ) by state and county
	Annual age-adjusted incidence rate of Bladder Cancer (including in situ) per 100,000 population by state
	5 year age-adjusted incidence rate of Bladder Cancer (including in situ) per 100,000 population by state and county

Incidence of Selected Cancers – <i>continued</i>	Annual number of cases of Brain and other nervous systems Cancer by state
	5 year number of cases of Brain and other nervous systems Cancer by state and county
	Annual age-adjusted incidence rate of Brain and other nervous system Cancer per 100,000 population by state
	5 year age-adjusted incidence rate of Brain and other nervous system Cancer per 100,000 population by state and county
	Annual number of cases of Brain and Central Nervous System Cancer in children (<15 years and <20 years) by state
	Annual Age-adjusted incidence rate of Brain and Central Nervous System Cancer in children (<15 years and <20 years) per 1,000,000 population by state
	Annual number of cases of Thyroid Cancer by state
	5 year number of cases of Thyroid Cancer by state and county
	Annual age-adjusted incidence rate of Thyroid Cancer per 100,000 population by state
	5 year age-adjusted incidence rate of Thyroid Cancer per 100,000 population by state and county
	Annual number of cases of Non-Hodgkin's Lymphoma by state
	5 year number of cases of Non-Hodgkin's Lymphoma by state and county
	Annual age-adjusted incidence rate of Non-Hodgkin's Lymphoma per 100,000 population by state
	5 year age-adjusted incidence rate of Non-Hodgkin's Lymphoma per 100,000 population by state and county
	Annual number of cases of Leukemia by state
	5 year number of cases of Leukemia by state and county
	Annual age-adjusted incidence rate of Leukemia per 100,000 population by state
	5 year age-adjusted incidence rate of Leukemia per 100,000 population by state and county
	Annual number of Leukemia in children (<15 years and <20 years) by state
	Annual age-adjusted incidence rate of Leukemia in children (<15 years and <20 years) per 1,000,000 population by state

Incidence of Selected Cancers – <i>continued</i>	Annual number of cases of Chronic Lymphocytic Leukemia by state
	Annual age-adjusted incidence rate of Chronic Lymphocytic Leukemia per 100,000 population by state
	Annual number of cases of Acute Myeloid Leukemia by state
	Annual age-adjusted incidence rate of Acute Myeloid Leukemia per 100,000 population by state
	Annual number of Acute Myeloid Leukemia in children (<15 years and <20 years) by state
	Annual age-adjusted incidence rate of Acute Myeloid Leukemia in children (<15 years and <20 years) per 1,000,000 population by state
	Annual number of cases of Acute Lymphocytic Leukemia in children (<15 years and <20 years) by state
	Annual age-adjusted incidence rate of Acute Lymphocytic Leukemia in children (<15 years and <20 years) per 1,000,000 population by state
	Annual number of cases of Oral Cavity and Pharynx Cancer by state
	5 year number of cases of Oral Cavity and Pharynx Cancer by state and county
	Annual age-adjusted incidence rate of Oral Cavity and Pharynx Cancer per 100,000 population by state
	5 year age-adjusted incidence rate of Oral Cavity and Pharynx Cancer per 100,000 population by state and county
	Annual number of cases of Larynx Cancer by state
	5 year number of cases of Larynx Cancer by state and county
	Annual age-adjusted incidence rate of Larynx Cancer per 100,000 population by state
	5 year age-adjusted incidence rate of Larynx Cancer per 100,000 population by state and county
	Annual number of cases of Esophagus Cancer by state
	5 year number of cases of Esophagus Cancer by state and county
	Annual age-adjusted incidence rate of Esophagus Cancer per 100,000 population by state
	5 year age-adjusted incidence rate of Esophagus Cancer per 100,000 population by state and county

Incidence of Selected Cancers – <i>continued</i>	Annual number of cases of Pancreas Cancer by state
	5 year number of cases of Pancreas Cancer by state and county
	Annual age-adjusted incidence rate of Pancreas Cancer per 100,000 population by state
	5 year age-adjusted incidence rate of Pancreas Cancer per 100,000 population by state and county

The data used to calculate and/or compile these measures was provided by MCR-ARC.

<i>Carbon Monoxide</i>	
Indicator	Measure
Hospitalizations for Carbon Monoxide (CO) Poisoning	Annual number of hospitalizations for CO poisoning by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent) by state
	Annual crude rate of hospitalization for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent) by state
	Annual age-adjusted rate of hospitalization for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent) by state
Emergency Department Visits for Carbon Monoxide Poisoning	Annual number of emergency department visits for CO Poisoning by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent) by state
	Annual crude rate of emergency department visits for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent) by state
	Annual age-adjusted rate of emergency department visits for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent) by state
Carbon Monoxide Poisoning Mortality	Annual number of deaths from CO poisoning by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent) by state

Carbon Monoxide Poisoning Mortality - <i>continued</i>	Annual crude rate of death from CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent) by state
	Annual age-adjusted rate of death from CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent) by state
Reported Exposure to Carbon Monoxide	Annual number of unintentional CO exposures reported to poison control centers by resulting health effect and treatment in a healthcare facility by state
	Annual crude rate of unintentional CO exposures reported to poison control centers per 100,000 population by resulting health effect and treatment in a healthcare facility by state
Home Carbon Monoxide Detector Coverage	Annual percent of Behavioral Risk Factor Surveillance System (BRFSS) respondents reporting at least one CO detector in their household by state

The data used to calculate and/or compile these measures was provided by DHSS/BEE, BVS, BVR, and BHCADD.

<i>Childhood Lead Poisoning*</i>	
Indicator	Measure
Testing Coverage and Housing Age	3 year testing period by annual birth cohort number of children born in the same year and tested for lead before age 3 by state and county
	3 year testing period by annual birth cohort percent of children born in the same year and tested before age 3 by state and county
	Annual number of children younger than 5 years living in poverty (as measured in 2000 census) by state and county
	Annual percent of children younger than 5 years living in poverty (as measured in 2000 census) by state and county
	Annual number of homes built before 1950 (as measured in the 2000 Census) by state and county
	Annual percent of homes built before 1950 (as measured in the 2000 Census) by state and county

*The Childhood Lead Poisoning measures can be displayed as the one indicator described above or as two indicators splitting the age of housing measures from the testing and poverty measures. The two indicators would be (1) Testing Coverage and

(2) Age of Housing. At the time of this publication, revised and new Childhood Lead Poisoning indicators are under review by the CWG.

The data used to calculate and/or compile these measures was provided by DHSS/BEE, MCDC, and the US Census Bureau.

Reproductive Outcomes & Vital Statistics	
Indicator	Measure
Prematurity	Annual percent of preterm (less than 37 weeks gestation) live singleton births by state and county
	5 year annual average percent of very preterm (less than 32 weeks gestation) live singleton births by state and county
Low Birthweight	Annual percent of low birthweight (less than 2500 grams) live term singleton births by state and county
	5 year annual average percent of very low birthweight (less than 1500 grams) live singleton births by state and county
Mortality	5 year annual average infant (less than 1 year of age) Mortality Rate per 1,000 live births by state and county
	5 year annual average neonatal (less than 28 days of age) Mortality Rate per 1,000 live births by state and county
	5 year annual average perinatal (equal to or greater than 28 weeks gestation to less than 7 days of age) Mortality Rate per 1,000 live births (plus fetal deaths equal to or greater than 28 weeks gestation) by state and county
	5 year annual average postneonatal (equal to or greater than 28 days to less than 1 year of age) Mortality Rate per 1,000 live births
Fertility	Annual total Fertility Rate per 1,000 women of reproductive age by state and county
Sex Ratio at Birth	Annual male to female sex ratio at birth (term singletons only) by state and county

The data used to calculate and/or compile these measures was provided by DHSS/BVS, BVR, and BHCADD.

*Water Quality***

Indicator	Measure
Arsenic Level and Potential Population Exposures	Annual distribution of number of community water systems by mean arsenic concentrations (micrograms per liter) by year by state
	Annual distribution of number of people served by community water systems by mean arsenic concentrations (micrograms per liter) by year by state
	Annual distribution of number of community water systems by maximum arsenic concentrations (micrograms per liter) by year by state
	Annual distribution of number of people served by community water systems by maximum arsenic concentrations (micrograms per liter) by year by state
	Quarterly distribution of number of community water systems by mean arsenic concentrations (micrograms per liter) by quarter by state
	Quarterly distribution of number of people served by community water systems by mean arsenic concentrations (micrograms per liter) by quarter by state
Nitrate Level and Potential Population Exposures	Annual distribution of number of community water systems by mean nitrate concentrations (milligrams per liter) by year by state
	Annual distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per liter) by year by state
	Annual Distribution of number of community water systems by maximum nitrate concentrations (milligrams per liter) by year by state
	Annual Distribution of number of people served by community water systems by maximum nitrate concentrations (milligrams per liter) by year by state
	Quarterly distribution of number of community water systems by mean nitrate concentrations (milligrams per liter) by quarter by state
	Quarterly distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per liter) by quarter by state
Disinfection Byproducts (DBP) Level and Potential Population Exposure (TTHM)	Annual distribution of number of community water systems by mean trihalomethane (THM) concentrations (micrograms per liter) by year by state

Disinfection Byproducts (DBP) Level and Potential Population Exposure (TTHM) - <i>continued</i>	Annual distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter) by year by state
	Annual distribution of number of community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter) by year by state
	Annual Distribution of number of people served by community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter) by year by state
	Quarterly distribution of number of community water systems by mean trihalomethane concentrations (micrograms per liter) by quarter by state
	Quarterly distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter) by quarter by state
Disinfection Byproduct: Levels and Potential Population Exposures (HAA5)	Annual distribution of number of community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter) by year by state
	Annual distribution of number of people served by community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter) by year by state
	Annual distribution of number of community water systems by maximum haloacetic acids (HAA5) concentrations (micrograms per liter) by year by state
	Annual distribution of number of people served by community water systems by maximum haloacetic acids (HAA5) concentrations (micrograms per liter) by year by state
	Quarterly distribution of number of people served by community water systems by mean haloacetic acids concentrations (micrograms per liter) by quarter by state
	Quarterly distribution of number of people served by community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter) by quarter by state
Public Water Use	Annual number of people receiving water from community water systems by state

** At the time of publication of this document, these water measures and additional water measures were under review by the CWG.

The data used to calculate and/or compile these measures was provided by DNR.

Complete NCDM files are compiled and submitted to CDC following the EPHT Content Workgroup recommendations and “how-to” guides (see Appendix). These files, as well as Extensible Markup Language (XML) Schema files, are available for download on Missouri’s EPHT network portal at <http://health.mo.gov/living/environment/epht/>.

Metadata

Metadata is “data about data.” It assists in the understanding of data by describing the content, quality, condition, access, and other characteristics of the data.

Questions answered by metadata include:

- Why was the data created or collected?
- How was the data created or collected?
- Who created or collected the data?
- When was the data last updated?
- How can the data be obtained?

All data included on the EPHT Network Portal must contain metadata that adheres to the Federal Geographic Data Committee (FGDC) Content Standard for Digital Geospatial Metadata (FGDC-STD-001-1998). Initially created for geospatial data, this standard was identified as robust enough to describe non-geospatial data. Tracking Network stakeholders have developed a profile from this standard that includes all required elements of the standard and additional elements identified by the stakeholders necessary to describe EPHT Network resources.

You can learn more at the FGDC homepage (<http://www.fgdc.gov/>) or from the Tracking Network Metadata Content Guidance Document (see Appendix).

The Missouri EPHT Network Portal includes a metadata search tool. Missouri’s EPHT metadata is available at <http://health.mo.gov/living/environment/epht/>. National EPHT metadata is available at <http://ephtracking.cdc.gov/showIndicatorsData.action>.

Diseases and Conditions Reportable In Missouri

The reporting of cases of disease is important in the planning and evaluation of prevention and control programs, the assurance of appropriate medical treatment, and in the detection of common-source outbreaks. In Missouri, the authority to require notification of cases of disease is the responsibility of DHSS.

DHSS has compiled a listing of 91 conditions and/or diseases that must be reported. These conditions and/or diseases are detailed in the Missouri Code of State Regulations, 19 CSR 20.20, Reporting Communicable, Environmental, and Occupational Diseases (see Appendix).

Reporting of cases of diseases and related conditions is a vital step in controlling and preventing the spread of disease. The data obtained from mandatory reporting by physicians, clinicians, and other health providers is used to:

- provide the basis for determining public health priorities;
- observe and establish trends in the incidence and prevalence of disease;
- identify potential disease outbreaks;
- plan and implement prevention and control programs;
- geographically distribute resources; and
- evaluate the success or failure of prevention and control programs.

Additional information on mandatory reporting of conditions and/or diseases in Missouri is available at <http://health.mo.gov/living/healthcondiseases/communicable/communicabledisease/manuals.php>. Questions regarding mandatory reporting should be addressed to the Division of Community and Public Health via e-mail to info@health.mo.gov or by calling 573.751.6113.

Public Health Surveillance is defined as the ongoing and systematic collection, analysis, and interpretation of outcome specific data for use in the planning, implementation, and evaluation of public health practice.

A surveillance system includes the functional capacity for data collection and analysis as well as the timely dissemination of these data to persons who can undertake effective prevention and control activities.¹

¹ Thacker SB. Historical development. In: Teutsch SM, Churchill RE, eds. Principles and Practice of Public Health Surveillance. New York, NY: Oxford University Press; 1994:3.

Missouri's Statutes, Rules, and Regulations Pertaining to Specific Data

Please Note: This listing provides a reference to the statutes, rules, and regulations that affect the most commonly requested data. This listing is not all-inclusive of all potential state statutes, rules, and/or regulations that could apply to a particular situation or request. Each request for data made to DHSS will be carefully reviewed and evaluated prior to the release of any data.

Vital Records Data

Missouri State Statutes and Code of Regulations allow for the release of record level vital records data by the Missouri Department of Health and Senior Services. The below listed statutes only apply to vital events occurring within Missouri's borders. The records of vital events that occur to Missouri residents in other states are the property of the state where the events take place.

193.045.2(4), RSMo, authorizes the state registrar to provide to the state or local health agencies copies of or data derived from certificates and reports required under sections 193.005 to 193.325, deemed necessary for state or local health planning and program activities...such copies or data shall remain the property of the department and the uses made of them shall be governed by the state registrar.

193.245(1), RSMo, the department to disclose upon request, a listing of persons who are born or who die on a particular date, but no information from the record other than the name and date of such birth or death shall be disclosed.

193.245(2), RSMo, allows the department to authorize disclosure of information contained in vital records for legitimate research purposes.

193.255.4, RSMo, authorizes the state registrar, upon request by federal, state, local and other public or private agencies, to furnish copies or data of any other vital statistics... for statistical or administrative purposes upon such terms or conditions as may be prescribed by regulation, provided that such copies or data shall not be used for purposes other than those for which they were requested unless so authorized by the state registrar.

19 CSR 10-10.090 Access to Vital Records: (1) (B) 3. No data shall be furnished from records for research purposes until the state registrar of vital records has received and approved a formal request for the research project. (1) (B) 2. The term research means a systematic study designed to develop or contribute to generalizable knowledge. The term generalizable means to emphasize the general character rather than specific details of, to formulate general principles or inferences from particulars. (1) (D) authorizes the state registrar or the local custodian – when deemed in the public interest and not for purposes of commercial solicitation or private gain – to furnish copies of

records or data from records to public agencies administering health, welfare, safety, law enforcement, education or public assistance programs and to private agencies approved by the state registrar.

Under section 610.035, RSMo, the department is prohibited from disclosing any Social Security number of a living person unless such disclosure is permitted by federal law, federal regulation, or state law.

Section 208.120, RSMo prohibits the department from disclosing any information obtained by them in the discharge of their official duties relative to the identity of applicants for, or recipients of, benefits or the contents of any records (e.g., Medicaid, Food Stamps). Public assistance information can be provided on de-identified records only.

45 C.F.R. Part 160 and Part 164. Vital Records requestors for research or administrative purposes will only be provided access to the minimum information necessary to achieve their specific research or administrative requests. Requestors are prohibited from disclosing any information that would identify a person and are also prohibited from the re-release of the data provided.

Patient Abstract System (PAS) Data

Missouri State Statutes and Code of Regulations allow for the release of PAS data by DHSS. The Department and other "public health authorities" are authorized to utilize PAS information for epidemiologic studies and for surveillance. The below listed statutes apply to Missouri residents only.

192.067(1), RSMo, the department, for purposes of conducting epidemiological studies to be used in promoting and safeguarding the health of the citizens of Missouri ... is authorized to receive information from patient medical records.

192.067(2), RSMo, Medical information...may be released by the department only in a statistical aggregate form that precludes and prevents the identification of patient, physician, or medical facility except that medical information may be shared with other public health authorities and coinvestigators of a health study if they abide by the same confidentiality restrictions required of the department of health and senior services... The department of health and senior services, public health authorities and coinvestigators shall use the information collected only for the purposes provided ...

192.665(9), RSMo, "Patient abstract data", data submitted by hospitals which includes but is not limited to date of birth, sex, race, zip code, county of residence, admission date, discharge date, principal and other diagnoses, including external causes, principal and other procedures, procedure dates, total billed charges, disposition of the patient and expected source of payment with sources categorized according to Medicare,

Medicaid, other government, workers' compensation, all commercial payors coded with a common code, self-pay, no charge and other.

192.667(7), RSMo, Information obtained by the department under the provisions of section 192.665 and this section shall not be public information...The department of health and senior services may authorize the use of the data by other research organizations pursuant to the provisions of section 192.067... The department shall not release data in a form which could be used to identify a patient. Any violation of this subsection is a class A misdemeanor.

19 CSR 10-33.010 Reporting Patient Abstract Data by Hospitals and Ambulatory Surgical Centers: (1)(A) Coinvestigator means any person or organization that applies to the department to be a coinvestigator of an epidemiological study; (C) Epidemiological study means research using patient abstract data to understand, promote or safeguard the health of a defined population. No marketing study or study designed to use data on a specific provider shall be considered an epidemiological study; (M) Public health authority means a federal, state or local governmental agency which has as its mission and responsibility the promotion and safeguarding of the public's health.

19 CSR 10-33.010 Reporting Patient Abstract Data by Hospitals and Ambulatory Surgical Centers: (12) Any person may apply to the department to be a coinvestigator of an epidemiological study using patient abstract data. A research protocol shall be submitted which includes all of the following: (A) A description of the proposed study; (B) The purpose of the study; (C) A description of the data elements needed for the study; (D) A description of a tape or a report if either is required; (E) A statement indicating whether the study protocol has been reviewed and approved by an institutional review board; (F) A description of data security procedures, including who shall have access to the data; and (G) A description of the proposed use and release of the data.

19 CSR 10-33.010 Reporting Patient Abstract Data by Hospitals and Ambulatory Surgical Centers: (13) The director of the department shall appoint a data release advisory committee composed of three (3) persons representing the health care industry and three (3) persons representing researchers and consumers. The advisory committee shall review all research protocols of persons applying to be a coinvestigator of an epidemiological study using patient abstract data. The advisory committee shall make a recommendation to the director whether the coinvestigator protocol should be accepted, accepted with conditions, or rejected. The committee shall consider: (A) The review made by the staff of the department; (B) Whether the proposed study meets the definition of an epidemiological study; (C) The potential for the coinvestigator or any other person to use the data for nonepidemiological purposes; (D) The professional expertise of the applicant to conduct the study; (E) The appropriateness of the proposed study design; (F) The willingness and ability of the applicant to protect the identity of any patient, physician, or provider; and (G) The data security measures and final disposition of the data proposed.

19 CSR 10-33.010 Reporting Patient Abstract Data by Hospitals and Ambulatory Surgical Centers: (14) The coinvestigator shall agree to the confidentiality, security and release of data requirements imposed by the department and shall agree to the review and oversight requirements imposed by the department.

19 CSR 10-33.010 Reporting Patient Abstract Data by Hospitals and Ambulatory Surgical Centers: (15) Data released to the coinvestigator shall not be rereleased in any form by the coinvestigator without the prior authorization of the department. Authorization for subsequent release of the data shall be considered only if the proposed release does not identify a patient, physician or provider.

19 CSR 10-33.010 Reporting Patient Abstract Data by Hospitals and Ambulatory Surgical Centers: (16) The following data elements permit identification of a patient, physician or provider, and are not to be rereleased by a coinvestigator: patient name; patient Social Security number; any datum which applies to fewer than three (3) patients, physicians or providers; physician number; provider number; and a quantity figure if one (1) entity contributes more than sixty percent (60%) of the amount.

19 CSR 10-33.010 Reporting Patient Abstract Data by Hospitals and Ambulatory Surgical Centers: (17) The department shall release only those patient abstract data elements to the coinvestigator which the department determines are essential to the study. The Unique Physician Identification Number (UPIN) associated with any patient abstract data shall not be released to any coinvestigator. If the research being conducted by a coinvestigator requires a physician number, the department may create a unique number which is not the UPIN. The department shall not provide information which links the unique number to the name of the physician.

19 CSR 10-33.010 Reporting Patient Abstract Data by Hospitals and Ambulatory Surgical Centers: (18) No epidemiological study conducted with a coinvestigator shall be approved unless the department determines that: (A) The epidemiological study has public benefit sufficient to warrant the department to expend resources necessary to oversee the project with the coinvestigator; (B) The department has sufficient resources available to oversee the project with the coinvestigator; and (C) The data release advisory committee reviewed the study and the director of the department authorized approval.

19 CSR 10-33.010 Reporting Patient Abstract Data by Hospitals and Ambulatory Surgical Centers: (19) Public health authorities and coinvestigators receiving data shall be informed by the department of the penalty for violating section 192.067, RSMo.

Frequently Asked Data Questions

When I run a query on Missouri's EPHT Network Portal, why do some of the values display as stars?

The query results will display stars (**) when the count or rate in certain cells has been suppressed either because the observed number of events is very small and not appropriate for publication, or it could be used to calculate the number in a cell that has been suppressed. Suppression is a statistical practice that is used to protect patient confidentiality and potentially identifying information by withholding or excluding small numbers within a specific demographic or geography.

Why isn't all the data the same on both the Missouri and National EPHT Network Portals?

There are many scientifically valid reasons that the data presented on the Missouri and National EPHT Network Portals may not be identical, including:

- Not every state or government health agency collects data on every condition and/or disease. Even when data is collected for the same condition and/or disease, different data elements may be required.
- Condition and/or disease definitions can vary between jurisdictions.
- Data element definitions can vary between jurisdictions.
- Conditions and/or diseases may have drastic seasonal variations across geographic areas.
- Different datasets and/or sources may have been used.
- Suppression of small cell values or complimentary suppression may be used.
- Data may be aggregated by different ages, races, or other demographic.

Specific questions regarding data differences should be addressed to the Missouri EPHT Program Manager via e-mail at EPHTN@health.mo.gov or by calling 573.751.6102.

Why isn't record level data for health conditions available on Missouri's EPHT Network Portal?

Patient level records are not public information, and may be shared only with other public health authorities and coinvestigators of a health study if they abide by the same confidentiality restrictions required by DHSS under section 192.067, RSMo.

In addition, federal law protects patient privacy. All requests for health information must be reviewed and approved by governing bodies. Agreements between agencies protect information and how it is used. All users must sign confidentiality agreements to ensure privacy and information must be stored in a secure environment. Access to this level of data will always be restricted and strictly controlled with all agencies and individuals held accountable by law.

What is the “Secure” EPHT Network Portal?

Missouri’s Secure EPHT Network Portal provides the same information as the public portal. The only difference between the two portals is that data available on the secure portal has not been suppressed. This data is available only to those people who have a legitimate need-to-know, such as other public health authorities or in some instances, coinvestigators of a health study.

To request a secure user account, please complete and submit the “Secure User Access” request available at <http://health.mo.gov/living/environment/epht/index.php>. After submitting the request, the local security officer will contact you for further information.

Questions regarding the process of obtaining a secure user account should be addressed to the Missouri EPHT Program Manager via e-mail at EPHTN@health.mo.gov or by calling 573.751.6102.

The data I want isn’t available on the Missouri EPHT Network Portal. How can I request it?

To submit a special request for specific data for research, a principal investigator must submit a completed Application for Missouri Vital Records or Patient Abstract System Data for Research Purposes. The application requires detailed information about the study protocol, justification for all data elements requested (each data element must be related to the hypotheses), and measures to ensure the confidentiality and security of the data. All information must be clear, consistent and specific. General descriptions do not allow accurate assessment of the value of the study or the need for the data items. Release of data from vital records and/or the Patient Abstract System by DHSS is granted to an agency/institution for the sole purpose of the research project described in the protocol application. The applicant will be required to complete and sign an Agreement for Oversight. All persons that will have access to the data must be listed in the application and will be required to sign the Confidentiality Pledge prior to being granted access to the study data.

It is the principal investigator's responsibility to design a valid study that would make a contribution to public health, and it is not the department's role to help refine a faulty study or a poorly described study until it meets generally acceptable scientific standards. Protocols of this nature will be rejected and further processing of such applications will be discontinued. An application will be immediately rejected if it is determined that:

1. it does not clearly describe a well-designed research or epidemiologic study,
2. the data will be used for commercial or marketing purposes, or private gain,
3. being a co-investigator would overburden the department, or

4. there is reason to believe that confidentiality of the data would be jeopardized by its release.

Researchers interested in obtaining DHSS data should first familiarize themselves with the data sets prior to designing their studies (see Data and Surveillance Systems available at <http://health.mo.gov/data/index.php>). Only those data elements related to the hypotheses and necessary for the study should be requested. The principal investigator will be notified of any discrepancy between the list of data elements requested in the research protocol and those determined by DHSS staff to be needed. Vital Records and Patient Abstract Data custodian contact information may be found at <http://health.mo.gov/data/pdf/contactus.pdf>.

Additional information on Missouri DHSS data release policies, procedures, and guidelines are available at <http://health.mo.gov/data/policies.php>.

Suggestions for adding additional data sets, sources, and/or content areas to Missouri's EPHT Network Portal should be addressed to the Missouri EPHT Program Manager via e-mail at EPHTN@health.mo.gov or by calling 573.751.6102.

Can I share data that I've obtained from Missouri's EPHT Network Portal?

You can share data obtained from Missouri's public EPHT Network Portal; however, sharing of data obtained by special request or from Missouri's Secure EPHT Network Portal is forbidden.

Releasing, sharing, or publishing DHSS-provided data or subsets of such data to any person or entity not directly identified in the study personnel section of the application or annual review form is not allowed.

Analytic tables, graphs, charts, or maps produced from DHSS-provided data for analytic purposes are allowable and not considered re-release.

What criteria are used to determine if a research study will be approved by DHSS?

Studies and/or research projects must meet the following specific standards and criteria:

- be scientifically valid and statistically sound;
- contribute to public health practice;
- not use Missouri Department of Health and Senior Services' resources unreasonably and unnecessarily;
- be conducted ethically and with integrity;
- be in compliance with state and federal statutes and regulations, including confidentiality provisions;
- be reviewed by the Missouri Department of Health and Senior Services' Institutional Review Board when required; and

- be consistent with Missouri Department of Health and Senior Services' policy.

Is there a cost for accessing the data on the Missouri EPHT Network Portal?

There is no cost for accessing data on Missouri's EPHT Network Portal; however, special requests for data may have a cost associated. In an effort to recover the service cost incurred for staff time and other expenses involved in data delivery, DHSS may charge fees for data and services based on the fee schedule (<http://health.mo.gov/data/pdf/datafeepolicy.pdf>). Fees are assessed for preparation of data based on programming time and materials. Payment is required before data files can be released.

Questions regarding Missouri's EPHT Network Portal should be addressed to the Missouri EPHT Program Manager via e-mail at EPHTN@health.mo.gov or by calling 573.751.6102.

Acronyms Used in this Guide

Acronym	Meaning
AAA	Area Agencies on Aging
ABLES	Adult Blood Lead Epidemiology and Surveillance
AMI	Acute Myocardial Infarction
APCP	Air Pollution Control Program
AQI	Air Quality Index
AQS	Air Quality System
AQUID	Aquatic Invertebrate Database
ASTHO	Association of State and Territorial Health Officers
ATSDR	Agency for Toxic Substances and Disease Registry
BCCDC	Bureau of Cancer and Chronic Disease Control
BEE	Bureau of Environmental Epidemiology
BEHS	Bureau of Environmental Health Services
BHCADD	Bureau of Health Care Analysis & Data Dissemination
BRFSS	Behavioral Risk Factor Surveillance System
BVR	Bureau of Vital Records
BVS	Bureau of Vital Statistics
CDC	Centers for Disease Control and Prevention
cL	centiliter
CLPPP	Childhood Lead Poisoning Prevention Program
CO	Carbon Monoxide
CSR	Code of State Regulations
CSTE	Council of State and Territorial Epidemiologists
CWS	Community Water System
CWG	Content Workgroup
DBP	Disinfection Byproduct
DCPH	Division of Community and Public Health
DED	Department of Economic Development
DEQ	Division of Environmental Quality
DESE	Missouri Department of Elementary and Secondary Education
DHSS	Department of Health and Senior Services
dL	deciliter
DNR	Department of Natural Resources
DOE	US Department of Energy
DOI	Department of Interior
DOT	Department of Transportation
DSS	Department of Social Services
ED	Emergency Department
EPA	Environmental Protection Agency

Acronym	Meaning
EPHI	environmental public health indicator
EPHT	Environmental Public Health Tracking
ES	Environmental Surveillance
ESP	Environmental Services Program
FGDC	Federal Geographic Data Committee
HAA5	Haloacetic Acids
HHS	Health and Human Services
HIE	Healthy Indoor Environments
HRAP	Health and Risk Assessment Program
HUD	Housing and Urban Development
HWP	Hazardous Waste Program
ICD	International Classification of Diseases
LPHAs	Local Public Health Agencies
LRP	Land Reclamation Program
MCDC	Missouri Census Data Center
MCL	maximum contaminant level
MCR-ARC	Missouri Cancer Registry and Research Center
MDA	Missouri Department of Agriculture
MDC	Missouri Department of Conservation
MEERTS	Missouri Environmental Emergency Response Tracking System
MHDC	Missouri Housing Development Commission
MICA	Missouri Information for Community Assessment
mL	milliliter
MoDOT	Missouri Department of Transportation
MOEIS	Missouri Emissions Inventory System
MoEVR	Missouri Electronic Vital Records
MSA	Metropolitan Statistical Area
NAACCR	North American Association of Central Cancer Registries
NASA	National Aeronautics and Space Administration
NCDM	Nationally Consistent Data & Measures
NCEH	National Center for Environmental Health
NIEHS	National Institute for Environmental Health Sciences
NIOSH	National Institute for Occupational Safety and Health
NOAA	National Oceanic and Atmospheric Administration
NPCR	National Program of Cancer Registries
NWS	National Weather Service
OAR	Office of Air and Radiation
OCSP	Office of Chemical Safety and Pollution Prevention
OECA	Office of Enforcement and Compliance Assurance
OEI	Office of Environmental Information

Acronym	Meaning
OSEDA	Office of Social and Economic Data Analysis
OSWER	Office of Solid Waste and Emergency Response
OW	Office of Water
PAS	Patient Abstract System
PDWB	Public Drinking Water Branch
PM _{2.5}	Particulate Matter 2.5 micrometers or less
ppb	part per billion
ppm	part per million
RSMo	Revised Statutes of Missouri
SDWIS	Safe Drinking Water Information System
SEPH	Section for Environmental Public Health
SEER	Surveillance Epidemiology and End Results
SEHIC	State Environmental Health Indicators Collaborative
SWMP	Solid Waste Management Program
THM	Trihalomethane
TTHM	Total Trihalomethanes
US	United States
USDA	United States (US) Department of Agriculture
USFWS	United States Fish and Wildlife Service
USGS	United States Geological Survey
WIMS	Well Information Management System
WPCB	Water Pollution Control Branch
WPP	Water Protection Program
XML	Extensible Markup Language
ZCTA	ZIP Code Tabulation Areas

Guide Development and Maintenance

This guide will continue to be revised and updated, as needed, as the program progresses. As part of the EPHT program's ongoing self-assessment, DHSS will meet periodically with partner entities throughout the year and review this guide to determine whether revisions are needed. Revisions will be published annually.

The Data & Statistical Guide is posted on the DHSS Internet site and available at: <http://health.mo.gov/living/environment/epht/index.php>.

 please consider the environment before printing

Appendix A

Centers for Disease Control and Prevention
Standards for Nationally Consistent Data
and Measures within the
Environmental Public Health Tracking Network
[Version 3.0 | June 20, 2013]

**Centers for Disease Control and Prevention Standards
for Nationally Consistent Data and Measures within
the Environmental Public Health Tracking Network**

Version 3.0
June 20, 2013

**Environmental Health Tracking Branch
Division of Environmental Hazards and Health Effects
National Center for Environmental Health
Centers for Disease Control and Prevention**

Foreword

This document was first published in March, 2008, setting the standards for the first Nationally Consistent Data and Measures (NCDMs) for the National Environmental Health Tracking Program. The purpose of these NCDMS was to ensure compatibility and comparability of data and measures useful for understanding the impact of our environment on our health. Version 2.0

- reflect the lessons learned in implementing the first NCDMs across local, state, and national tracking networks
- improve the utility of specific measures
- identify recommended temporal and spatial resolution, specifically for health outcomes, based on confidentiality protection needs and data steward requests

Specific updates included in version 2 include:

- Clarified description of process for creating and adopting the first set of NCDMs
- Clarified the meaning of indicator, measure, and data within the Tracking Network
- Added columns to the table summarizing the indicators and measures in order to identify
 - minimum temporal and geographic resolution
 - data source
 - grantee requirements
- Updated indicator templates to reflect minimum temporal and geographic resolution at which measures are to be displayed on public portals

Version 3.0 includes a change from required to optional for the Fertility indicator and documentation for NCDMs adopted since the release of version 2 in August 2011.

- Hospitalizations and ED visits for heat
- ED visits for asthma
- Blood lead levels by birth cohort and annual blood lead levels
- Updates to drinking water NCDMs

Table of Contents

Introduction

Section 1: Summary of Environmental Public Health Tracking's Standards for Nationally Consistent Data and Measures (NCDMs)

- A. Acute Myocardial Infarction
- B. Air Quality
- C. Asthma
- D. Birth Defects
- E. Cancer
- F. Carbon Dioxide Poisoning
- G. Childhood Lead Poisoning
- H. Drinking Water
- I. Heat
- J. Reproductive Health Outcomes

Section 2: Indicator Templates

- A. Acute Myocardial Infarction
- B. Air Quality
- C. Asthma
- D. Birth Defects
- E. Cancer
- F. Carbon Dioxide Poisoning
- G. Childhood Lead Poisoning
- H. Drinking Water
- I. Heat
- J. Reproductive Health Outcomes

Introduction

Environmental Public Health Tracking is the ongoing collection, integration, analysis, interpretation, and dissemination of data from environmental hazard monitoring, human exposure, and health effects surveillance. In financial year 2002, Congress appropriated funds to the Centers for Disease Control and Prevention (CDC) to develop a national environmental public health tracking network and to improve environmental health capacity at the state and local level.

CDC established its National Environmental Public Health Tracking Program with the following goals:

1. Build a sustainable national environmental public health tracking network (Tracking Network);
2. Enhance environmental public health tracking workforce and infrastructure;
3. Disseminate information to guide policy, practice, and other actions to improve the Nation's health;
4. Advance environmental public health science and research;
5. Foster collaboration among health and environmental programs.

In 2006, CDC transitioned from a piloting and planning phase to implementation. The network was envisioned as a web-based, secure, distributed network of standardized electronic health and environmental data. Sixteen states and New York City were funded in August 2006 to construct state-wide (city-wide) networks that will be components of the national network and to participate in a collaborative process to develop network standards development process. Additional funding from Congress allowed CDC to add 6 more states in 2009 and 1 in 2010.

As part of the implementation process, CDC established a Content Work Group (CWG) to:

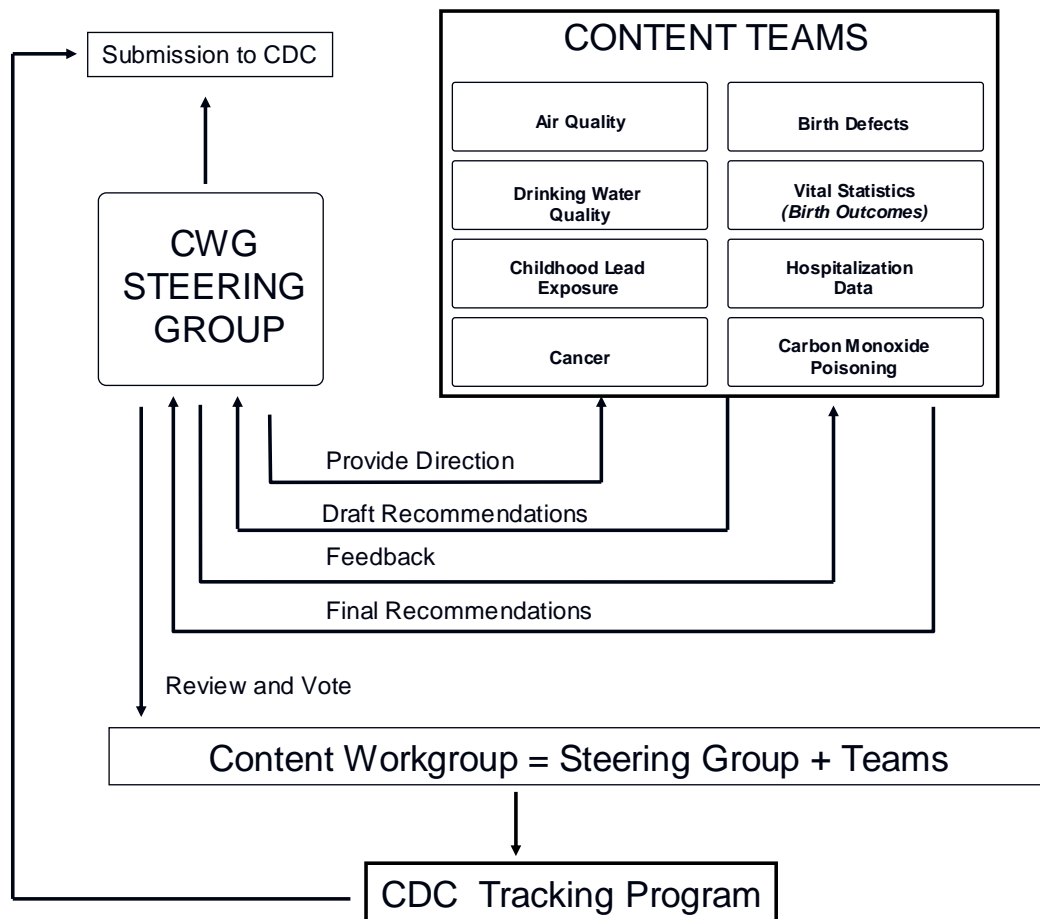
1. Identify and recommend core measures for the Tracking Network;
2. Examine the availability and applicability of existing data and identify approaches for deriving or collecting needed data;
3. Identify and adapt standards and guidelines to facilitate nationally consistent data collection and ensure compatibility with existing standards efforts;
4. Recommend metadata elements to describe data quality;
5. Identify and recommend methods and tools for data integration, analysis and presentation.

The CWG structure included a steering group made up of the principal investigators for grantee health departments and academic partners. Content-specific teams advised the steering group. These teams included content experts from: grantee states, cities and academic partners; non-funded states and cities; CDC; other government agencies including the Environmental Protection Agency (EPA), the National Aeronautics and Space Administration (NASA), the US Geological Survey (USGS) and the National Institutes of Health (NIH); and non-governmental organizations including the American Association of Poison Control Centers (AAPCC), the National Birth Defects Prevention Network (NBDPN), the National Association of Health Data Organizations (NAHDO), the National Association for Public Health Statistics and Information

Systems (NAPHSIS) and the North American Association of Central Cancer Registries (NAACCR).

Eight content teams were established, and each provided recommendations to CDC via the steering group for an initial set of Nationally Consistent Data and Measures (NCDMs)(Figure 1). NCDMs consist of measures, grouped by indicators, and the data required to generate them. A measure is a summary characteristic or statistic, such as a sum, percentage, or rate. There may be several measures of a specific indicator which when considered in conjunction fully describe the indicator. An indicator is one or more items, characteristics or other things that will be assessed and that provide information about a population's health status, their environment, and other factors with the goal allowing us to monitor trends, compare situations, and better understand the link between environment and health. It is assessed through direct and indirect measures (e.g. levels of a pollutant in the environment as a measure of possible exposure) that describe health or a factor associated with health (i.e., environmental hazard, age) in a specified population. In general, content teams focused on developing measures specific to one of these areas, but they also considered both proven and potential linkages to the other areas.

Figure 1: Content Work Group (CWG) Structure and Process, 2006 - 2010



Recommendations from content teams were separated into two parts; the first part concerned indicators, measures, and how-to-guides which described the methods for extracting necessary data and generating the measures. The second part was a data dictionary which described the data to be shared with CDC. Recommendations were reviewed by the CWG Steering Group for scientific rigor, utility for Tracking, and feasibility of each grantee generating the measures and where specified providing data to CDC for use on the National Tracking Portal.

This document provides an updated summary of the NCDMs adopted by CDC as Tracking standards. Section One of this document includes tables that summarize the indicators and measures and identify the requirements of Tracking grantees for creating measures and providing data to CDC. These Tracking standards incorporate discussions among the CWG steering group as well as the recommendations of content teams concerning the use of existing national datasets, where relevant.

Section Two includes the indicator templates originally developed by the teams and updated by CDC. An indicator template describes the indicator’s measures and their deviations, uses, and limitations. Although teams generally adhered to the template there was some minor variation in

the submitted documents. In creating this document original recommendations were modified to ensure compatibility with the National Network and consistency across NCDMs.

Details regarding the data needed to generate the measures are provided in the how-to-guides, data dictionaries, and schemas available from the CDC Tracking Program. Each set of documentation represents a data feed needed to generate one or more measures.

SECTION ONE: SUMMARY OF NATIONALLY CONSISTENT DATA AND MEASURES

This section lists all NCDMs for the Tracking Network by indicator and measure name. The minimum temporal and geographic resolutions are provided for the display of each required measure. These resolutions were selected to provide the most granular view of the measure possible while considering the rarity of the outcome being measured and data steward requirements. Grantees able to publish more temporally or geographically resolved measures are encouraged to do so. Grantees unable to publish at least the minimum temporal and geographic resolutions should provide written documentation to CDC Tracking Program. **The temporal and geographic resolutions of the measures in this document are not necessarily the temporal and spatial resolution of the data requirements. Information about the required fields and resolution of the data to generate the measures are provided in the how-to-guides and data dictionaries.** The source of the data required to generate each measure at the national level is provided in the summary table. Some data are provided by state and local grantees while other data are provided by national partners. Each measure is also listed as either required or optional for Tracking Grantees. Required means the grantees must (1) provide the data to CDC Tracking Program if the data are not available nationally and (2) publish the measure on their state or local portals.

Content Domain: Heart Attacks or Acute Myocardial Infarction (AMI)

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Heart Attacks	Number of hospitalizations for heart attack	Annual	State and county	Grantee Provided	Required
	Average daily number of hospitalizations for heart attack, by month	Annual	State and county	Grantee Provided	Optional
	Maximum daily number of hospitalizations for heart attack by month	Annual	State and county		
	Minimum daily number of hospitalizations for heart attack by month	Annual	State and county		
	Rate of hospitalization for heart attack among persons 35 and over by age group (total, 35-64, 65+) per 10,000 population	Annual	State and county	Grantee Provided	Required
	Age-adjusted rate of hospitalization for heart attack persons 35 and over per 10,000 population	Annual	State and county		

Content Domain: Air Quality

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
<u>Ozone—Days Above Regulatory Standard</u>	Number of days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard	Annual	County	Nationally Derived	Required
	Number of person-days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard	Annual	County		
<u>Fine Particle (PM2.5)—Days Above Regulatory Standard</u>	Percent of days with PM2.5 levels over the National Ambient Air Quality Standard (NAAQS)	Annual	County	Nationally Derived	Required
	Number of person-days with PM2.5 over the National Ambient Air Quality Standard (NAAQS)	Annual	County		
<u>Annual PM2.5 Level</u>	Average ambient concentrations of PM 2.5 in micrograms per cubic meter (based on seasonal averages and daily measurement)	Annual	County	Nationally Derived	Required
	Percent of population living in counties exceeding the National Ambient Air Quality Standard (compared to percent of population living in counties that meet the standard and percent of population living in counties without PM2.5 monitoring)	Annual	State		

Content Domain: Asthma

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
<u>Hospitalizations for Asthma</u>	Number of hospitalizations for asthma	Annual	State and county	Grantee Provided	Required
	Average daily number of hospitalizations for asthma, by month	Annual	State and county	Grantee Provided	Optional
	Maximum daily number of hospitalizations for asthma by month	Annual	State and county		
	Minimum daily number of hospitalizations for asthma by month	Annual	State and county		
	Rate of hospitalization for asthma by age group (total, 0-4, 5-14, 15-34, 35-64, and 65+) per 10,000 population	Annual	State and county	Grantee Provided	Required
	Age-adjusted rate of hospitalization for asthma per 10,000 population	Annual	State and county		
<u>Emergency Department Visits for Asthma</u>	Annual number of emergency department visits for asthma	Annual	State and county	Grantee Provided	Required
	Average number of emergency department visits for asthma as primary diagnosis per month	Annual	State and county		
	Annual crude rate of emergency department visits for asthma by age group (total, 0-4, 5-14, 15-34, 35-64, and 65+) per 10,000 population by age group	Annual	State and county		
	Annual age-adjusted rate of emergency department visits for asthma by age groups (total, 0-4, 5-14, 15-34, 35-64, and 65+) per 10,000 population	Annual	State and county		

Content Domain: Birth Defects

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
Prevalence of Birth Defects	Prevalence of Anencephaly per 10,000 live births	5 year	State and county	Grantee Provided	Required
	Prevalence of Spina Bifida (without Anencephaly) per 10,000 live births over	5 year	State and county		
	Prevalence of Hypoplastic Left Heart Syndrome per 10,000 live births	5 year	State and county		
	Prevalence of Tetralogy of Fallot per 10,000 live births	5 year	State and county		
	Prevalence of Transposition of the Great Arteries (vessels) per 10,000 live births	5 year	State and county		
	Prevalence of Cleft Lip with or without Cleft Palate per 10,000 live births	5 year	State and county		
	Prevalence of Cleft Palate without Cleft Lip per 10,000 live births	5 year	State and county		
	Prevalence of Hypospadias per 10,000 live male births	5 year	State and county		
	Prevalence of Gastroschisis per 10,000 live births	5 year	State and county		
	Prevalence of Upper Limb Deficiencies per 10,000 live births	5 year	State and county		
	Prevalence of Lower Limb Deficiencies per 10,000 live births	5 year	State and county		
	Prevalence of Trisomy 21 per 10,000 live births by maternal age at delivery (<35 and >=35)	5 year	State and county		

Content Domain: Cancer

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
<u>Incidence of Selected Cancers</u>	Number of cases of Mesothelioma	5 year	State	Nationally Derived	Required
	Age-adjusted incidence rate of Mesothelioma per 100,000 population	5 year	State		
	Number of cases of Melanoma of the Skin	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Melanoma of the Skin per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Liver and Intrahepatic Bile Duct Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Liver and Intrahepatic Bile Duct Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Kidney and Renal Pelvis Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Kidney and Renal Pelvis Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Breast Cancer in females by Age group (<50, ≥50, total)	Annual	State		
		5 year	State and county		
Age-adjusted incidence rate of Breast Cancer in females per 100,000 population by Age group (<50, ≥50, total)	Annual	State			

		5 year	State and county		
Number of cases of Lung and Bronchus Cancer		Annual	State		
		5 year	State and county		
Age-adjusted incidence rate of Lung and Bronchus Cancer per 100,000 population		Annual	State		
		5 year	State and county		
Number of cases of Bladder Cancer (including in situ)		Annual	State		
		5 year	State and county		
Age-adjusted incidence rate of Bladder Cancer (including in situ) per 100,000 population		Annual	State		
		5 year	State and county		
Number of cases of Brain and other nervous systems Cancer		Annual	State		
		5 year	State and county		
Age-adjusted incidence rate of Brain and other nervous systems Cancer per 100,000 population		Annual	State		
		5 year	State and county		
Number of cases of Brain and Central Nervous System Cancer in children (<15 years and <20 years)		Annual	State		
Age-adjusted incidence rate of Brain and Central Nervous System Cancer in children (<15 years and <20 years) per 1,000,000 population		Annual	State		
Number of cases of Thyroid Cancer		Annual	State		
		5 year	State and county		
Age-adjusted incidence rate of Thyroid Cancer per 100,000		Annual	State		

population	5 year	State and county		
Number of cases of Non-Hodgkin's Lymphoma	Annual	State		
	5 year	State and county		
Age-adjusted incidence rate of Non-Hodgkin's Lymphoma per 100,000 population	Annual	State		
	5 year	State and county		
Number of cases of Leukemia	Annual	State		
	5 year	State and county		
Age-adjusted incidence rate of Leukemia per 100,000 population	Annual	State		
	5 year	State and county		
Number of Leukemia in children (<15 years and <20 years)	Annual	State		
Age-adjusted incidence rate of Leukemia in children (<15 years and <20 years) per 1,000,000 population	Annual	State		
Number of cases of Chronic Lymphocytic Leukemia	Annual	State		
Age-adjusted incidence rate of Chronic Lymphocytic Leukemia per 100,000 population	Annual	State		
Number of cases of Acute Myeloid Leukemia	Annual	State		
Age-adjusted incidence rate of Acute Myeloid Leukemia per 100,000 population	Annual	State		
Number of Acute Myeloid Leukemia in children (<15 years and <20 years)	Annual	State		

	Age-adjusted incidence rate of Acute Myeloid Leukemia in children (<15 years and <20 years) per 1,000,000 population	Annual	State		
	Number of cases of Acute Lymphocytic Leukemia in children (<15 years and <20 years)	Annual	State		
	Age-adjusted incidence rate of Acute Lymphocytic Leukemia in children (<15 years and <20 years) per 1,000,000 population	Annual	State		
<u>Incidence of Selected Cancers</u>	Number of cases of Oral Cavity and Pharynx Cancer	Annual	State	Nationally Derived	Optional
		5 year	State and county		
	Age-adjusted incidence rate of Oral Cavity and Pharynx Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Larynx Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Larynx Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Esophagus Cancer	Annual	State		
		5 year	State and county		
	Age-adjusted incidence rate of Esophagus Cancer per 100,000 population	Annual	State		
		5 year	State and county		
	Number of cases of Pancreas Cancer	Annual	State		
		5 year	State and county		
Age-adjusted incidence rate of Pancreas Cancer per 100,000 population	Annual	State			
	5 year	State and county			

Content Domain: Carbon Monoxide

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
<u>Hospitalizations for Carbon Monoxide (CO) Poisoning</u>	Number of hospitalizations for CO poisoning by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State	Grantee Provided	Required
	Crude rate of hospitalization for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
	Age-adjusted rate of hospitalization for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
<u>Emergency Department Visits for CO Poisoning</u>	Number of emergency department visits for CO Poisoning by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State	Grantee Provided	Optional
	Crude rate of emergency department visits for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
	Age-adjusted rate of emergency department visits for CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional	Annual	State		

	non-fire related, and unknown intent)				
<u>CO Poisoning Mortality</u>	Number of deaths from CO poisoning by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State	Nationally Derived	Required
	Crude rate of death from CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
	Age-adjusted rate of death from CO poisoning per 100,000 population by cause/intent (unintentional fire-related, unintentional non-fire related, and unknown intent)	Annual	State		
<u>Reported Exposure to CO</u>	Number of unintentional CO exposures reported to poison control centers by resulting health effect and treatment in a healthcare facility	Annual	State	Nationally Derived	Optional
	Crude rate of unintentional CO exposures reported to poison control centers per 100,000 population by resulting health effect and treatment in a healthcare facility	Annual	State		
<u>Home CO Detector Coverage</u>	Percent of Behavioral Risk Factor Surveillance System (BRFSS) respondents reporting at least one CO detector in their household	Annual	State	Nationally Derived	Optional

Content Domain: Childhood Lead Poisoning

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
<u>Testing and Housing Age</u>	Number of children born in the same year and tested	Annual	State and county	Nationally Derived	Required
	Percent of children born in the same year and tested	Annual	State and county		
	Number of homes built before 1950 (as measured in the 2000 Census)	Annual	State and county		
	Percent of homes built before 1950 (as measured in the 2000 Census)	Annual	State and county		
	Number of children younger than 5 years living in poverty (as measured in 2000 census)	Annual	State and county		Optional
	Percent of children younger than 5 years living in poverty (as measured in 2000 census)	Annual	State and county		
<u>Blood Lead Levels by Birth Cohort</u>	Number of children born in the same year and tested	Annual	State and county	Nationally Derived	Required
	Percent of children born in the same year and tested	Annual	State and county		
	Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$	Annual	State and county		
	Percent of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$	Annual	State and county		
	Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$, by blood lead level category	Annual	State		
	Percent of children born in the same	Annual	State		

	year and tested with confirmed blood lead levels ≥ 10 $\mu\text{g/dL}$, by blood lead level category				
	PROPOSED *Number of children born in the same year and tested with blood lead levels between 5 and <10 $\mu\text{g/dL}$	Annual	State and county		
	PROPOSED*Percent of children born in the same year and tested with blood lead levels between 5 and <10 $\mu\text{g/dL}$	Annual	State and county		
<u>Annual Blood Lead Levels</u>	Number of children tested, by age group	Annual	State and county	Nationally Derived	Required
	Percent of children tested, by age group	Annual	State and county		
	Number of children tested with confirmed blood lead levels ≥ 10 $\mu\text{g/dL}$, by age group	Annual	State and county		
	Percent of children tested with confirmed blood lead levels ≥ 10 $\mu\text{g/dL}$, by age group	Annual	State and county		
	Number of children tested with confirmed blood lead levels ≥ 10 $\mu\text{g/dL}$ by blood lead level category, by age group	Annual	State		
	Percent of children tested with confirmed blood lead levels ≥ 10 $\mu\text{g/dL}$, by blood lead level category, by age group	Annual	State		
	PROPOSED *Number of children tested with blood lead levels between 5 and <10 $\mu\text{g/dL}$	Annual	State and county		
	PROPOSED*Percent of children tested with blood lead levels between 5 and <10 $\mu\text{g/dL}$	Annual	State and county		

Content Domain: Climate Change

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
<u>Heat Stress Hospitalizations</u>	Number of hospitalizations for heat stress	Annual from May–September	State and national	Grantee Provided	Required
	Crude rate of hospitalization for heat stress by age groups (total, 0–4, 5–14, 15–34, 35–64, and 65+) per 100,000 population	Annual from May–September	State and national		
	Age-adjusted rate of hospitalization for heat stress (by age groups 0–4, 5–14, 15–34, 35–64, and 65+) per 100,000 population	Annual from May–September	State and national		
<u>Heat Stress Emergency Department Visits for Heat Stress</u>	Annual number of emergency department visits for heat stress	Annual from May–September	State and county	Grantee Provided	Required
	Annual crude rate of emergency department visits for heat stress by age group (total, 0–4, 5–14, 15–34, 35–64, and 65+) per 100,000	Annual from May–September	State and county		
	Age-adjusted rate of emergency department visits for heat stress by age groups (total, 0–4, 5–14, 15–34, 35–64, and 65+) per 100,000 population	Annual from May–September	State and county		

Content Domain: Drinking Water

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
<u>Atrazine Level and Potential Population Exposures</u>	Distribution of number of Community Water Systems (CWS) by mean atrazine concentration (micrograms per liter)	Quarterly	County	Grantee Provided	Required
	Distribution of number of CWS by maximum atrazine concentration (micrograms per liter)	Annual	County		
	Distribution of number of CWS by mean atrazine concentration (micrograms per liter)	Annual	County		
	Mean concentration of atrazine (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by mean atrazine concentration (micrograms per liter)	Quarterly	County		
	Distribution of number of people served by CWS by maximum atrazine concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean atrazine concentration (micrograms per liter)	Annual	County		
<u>Arsenic Level and Potential Population Exposures</u>	Distribution of number of community water systems by mean arsenic concentrations (micrograms per liter)	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean arsenic concentrations (micrograms per liter)	Annual	State		
	Distribution of number of community water systems by maximum arsenic concentrations (micrograms per liter)	Annual	State		

	Distribution of number of people served by community water systems by maximum arsenic concentrations (micrograms per liter)	Annual	State		
	Mean concentration of Arsenic (micrograms per liter) at CWS-level	Annual	State		
<u>Di (2-Ethylhexyl) phthalate (DEHP) Level and Potential Population Exposures</u>	Distribution of number of Community Water Systems (CWS) by maximum DEHP concentration (micrograms per liter)	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean DEHP concentration (micrograms per liter)	Annual	County		
	Mean concentration of DEHP (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum DEHP concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean DEHP concentration (micrograms per liter)	Annual	County		
<u>Nitrate Level and Potential Population Exposures</u>	Distribution of number of community water systems by mean nitrate concentrations (milligrams per liter)	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per liter)	Annual	State		
	Distribution of number of community water systems by maximum nitrate concentrations (milligrams per liter)	Annual	State		
	Distribution of number of people served by community water systems by maximum nitrate concentrations (milligrams per liter)	Annual	State		

	Distribution of number of community water systems by mean nitrate concentrations (milligrams per liter)	Quarterly	State		
	Distribution of number of people served by community water systems by mean nitrate concentrations (milligrams per liter)	Quarterly	State		
	Mean concentration of nitrate (milligrams per liter) at CWS-level	Annual	State		
<u>Disinfection Byproducts (DBP) Level and Potential Population Exposure (TTHM)</u>	Distribution of number of community water systems by mean trihalomethane (THM) concentrations (micrograms per liter)	Annual	State	Grantee Provided	Required
	Distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter)	Annual	State		
	Distribution of number of community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter)	Annual	State		
	Distribution of number of people served by community water systems by maximum trihalomethane (THM) concentrations (micrograms per liter)	Annual	State		
	Distribution of number of community water systems by mean trihalomethane concentrations (micrograms per liter)	Quarterly	State		
	Distribution of number of people served by community water systems by mean trihalomethane (THM) concentrations (micrograms per liter)	Quarterly	State		

<u>Disinfection Byproduct: Levels and Potential Population Exposures (HAA5)</u>	Distribution of number of community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter)	Annual	State	Grantee Provided	Required
	Mean concentration of HAA5 (micrograms per liter) at CWS-level	Annual	State		
	Distribution of number of community water systems by maximum haloacetic acids (HAA5) concentrations (micrograms per liter)	Annual	State		
	Distribution of number of CWS by maximum TTHM concentration (micrograms per liter)	Annual	State		
	Distribution of number of people served by community water systems by mean haloacetic acids (HAA5) concentrations (micrograms per liter)	Quarterly	State		
	Distribution of number of CWS by mean TTHM concentrations (micrograms per liter)	Quarterly	State		
	Distribution of number of CWS by mean TTHM concentration (micrograms per liter)	Annual	State		
	Mean concentration (micrograms per liter) of TTHM at CWS-level	Annual	State		
<u>Public Water Use</u>	Number of people receiving water from community water systems	Annual	State	Grantee Provided	Required
<u>Combined Radium-226 and -228 Levels and Potential Population</u>	Distribution of number of Community Water Systems (CWS) by maximum Radium concentration picoCuries per Liter	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean Radium concentration picoCuries per Liter	Annual	County		

<u>Exposure</u>	Mean concentration of Radium picoCuries per Liter at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum Radium concentration picoCuries per Liter	Annual	County		
	Distribution of number of people served by CWS by mean Radium concentration picoCuries per Liter	Annual	County		
<u>Tetrachloroethene (PCE) Levels and Potential Population Exposure</u>	Distribution of number of Community Water Systems (CWS) by maximum PCE concentration (micrograms per liter)	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean PCE concentration (micrograms per liter)	Annual	County		
	Mean concentration of PCE (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum PCE concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean PCE concentration (micrograms per liter)	Annual	County		
<u>Trichloroethene (TCE) Levels and Potential Population Exposure</u>	Distribution of number of CWS by maximum TCE concentration (micrograms per liter)	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean TCE concentration (micrograms per liter)	Annual	County		
	Mean concentration of TCE (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum TCE concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean TCE concentration (micrograms per liter)	Annual	County		

<u>Uranium Levels and Potential Population Exposure</u>	Distribution of number of Community Water Systems (CWS) by maximum Uranium concentration (micrograms per liter)	Annual	County	Grantee Provided	Required
	Distribution of number of CWS by mean Uranium concentration (micrograms per liter)	Annual	County		
	Mean concentration of Uranium (micrograms per liter) at CWS-level	Annual	County		
	Distribution of number of people served by CWS by maximum Uranium concentration (micrograms per liter)	Annual	County		
	Distribution of number of people served by CWS by mean Uranium concentration (micrograms per liter)	Annual	County		

Content Domain: Reproductive Health Outcomes

Indicator	Measure	Temporal Resolution	Geographic Resolution	Source of Data for National Network	Grantee Required
<u>Prematurity</u>	Percent of preterm (less than 37 weeks gestation) live singleton births	Annual	State and county	Nationally Derived	Required
	Percent of very preterm (less than 32 weeks gestation) live singleton births	5 year Annual Average	State and county		
<u>Low Birthweight</u>	Percent of low birthweight (less than 2500 grams) live term singleton births	Annual	State and county	Nationally Derived	Required
	Percent of very low birthweight (less than 1500 grams) live singleton births	5 year Annual Average	State and county		
<u>Mortality</u>	Average Infant (less than 1 year of age) Mortality Rate per 1000 live births	5 year Annual Average	State and county	Nationally Derived	Required
	Average Neonatal (less than 28 days of age) Mortality Rate per 1000 live births	5 year Annual Average	State and county		
	Average Perinatal (equal to or greater than 28 weeks gestation to less than 7 days of age) Mortality Rate per 1000 live births (plus fetal deaths equal to or greater than 28 weeks gestation)	5 year Annual Average	State and county		
	Average Postneonatal (equal to or greater than 28 days to less than 1 year of age) Mortality Rate per 1000 live births	5 year Annual Average	State and county		
<u>Fertility</u>	Total Fertility Rate per 1000 women of reproductive age	Annual	State and county	Nationally Derived	Optional
<u>Sex Ratio at Birth</u>	Male to Female sex ratio at birth (term singletons only)	Annual	State and county	Nationally Derived	Required

SECTION TWO: INDICATOR TEMPLATES

This section contains an indicator template for each indicator and corresponding measures listed in section one. The indicator template provides basic information about the indicator including:

1. Measures
2. Derivations of the measures
3. Units
4. Geographic Scope
5. Geographic Scale
6. Time Period
7. Time Scale
8. Rationale
9. Use of the Measure
10. Limitations of the Measure
11. Data Sources
12. Limitations of Data Sources
13. References

Additional information about the underlying data needed for the indicator and steps for extracting the data and generating the measures can be found in the how-to-guides and data dictionaries.

CONTENT DOMAIN: HEART ATTACK
INDICATOR: HOSPITALIZATIONS FOR HEART ATTACK

Type of EPHT Indicator	Health Outcome
Measures	<ol style="list-style-type: none"> 1. Number of hospitalizations for acute myocardial infarction (AMI) 2. Minimum daily number of hospitalizations for AMI by month 3. Maximum daily number of hospitalizations for AMI by month 4. Average daily number of hospitalizations for AMI by month 5. Crude rate of hospitalizations for AMI among persons 35 and older by age group (total, 35-64, 65+) per 10,000 population 6. Annual age-adjusted rate of hospitalizations for AMI among persons 35 and older per 10,000 population <p>When supported by sufficient data volume, the measures may also be reported stratified by sex, race, and ethnicity.</p>
Derivation of Measures	<p>Numerator: Resident hospitalizations for AMI, ICD-9-CM: 410.00–410.92 by gender and total for state and by county</p> <p>Denominator: Midyear resident population by gender, for state and by county</p> <p>Adjustment: Age-adjustment by the direct method to Year 2000 U.S. Standard population</p>
Unit	Hospital admission (categorized by discharge diagnosis)
Geographic Scope	State and national (tracking network states)
Geographic Scale	State and county
Time Period	Hospital admissions from January 1 through December 31 for each year, 2000–current
Time Scale	Daily, monthly, and annually (as appropriate for the measure)
Rationale	<p>There currently is no single AMI surveillance system in place in the United States, nor does such a system exist for coronary heart disease (CHD) in general. Mortality is the sole descriptor for national data for AMI. Estimates of incidence and prevalence of AMI and CHD are largely based on survey samples (e.g., NHANES) or large cohort studies such as the Atherosclerosis Risk in Communities (ARIC) study.</p> <p>In 2007, the American Heart Association estimated 565,000 new attacks and 300,000 recurrent attacks of MI annually (National Heart, Lung, and Blood Institute: based on unpublished data from the ARIC study and the Cardiovascular Health Study [CHS]). Among</p>

	<p>Americans aged ≥ 20 years, new and recurrent MI prevalence for both men and women represented 3.7% of the U.S. population, or 7,900,000 (4.9 million men and 3.0 million women). Corresponding prevalence by race and ethnicity is 5.4% for white men, 2.5% for white women, 3.9% for black men, and 3.3% for black women.</p> <p>The well-documented risk factors for AMI include diabetes, hypertension, obesity, hypercholesterolemia, and cigarette smoking. Increasingly, investigators both in the United States and abroad have shown significant relationships between air pollutants and increased risk of AMI and other forms of CHD. Studies have often focused on persons aged >65 years. A number of epidemiologic studies have reported associations between air pollution (ozone, PM₁₀, CO, PM 2.5, SO₂) and hospitalizations for AMI and other forms of heart disease. Models have demonstrated increases in AMI hospitalization rate in relation to fine particles (PM_{2.5}), particularly in sensitive subpopulations such as the elderly, patients with pre-existing heart disease, and particularly persons who are survivors of MI or persons with COPD. An increase of 10 ug/m³ in PM 2.5 was associated with a 4.5% elevation in risk of acute ischemic coronary events (unstable angina and AMI) (95% CI, 1.1–8.0). Mortality statistics have been linked for a 16-year period to chronic exposure of multiple air pollutants in 500,000 adults residing throughout the United States. Each 10 ug/m³ in annual PM_{2.5} was related to a 12% increased mortality risk.</p>
<p>Use of the Measures</p>	<p>Developing a standardized analytic method for AMI hospital admissions among residents in each state will provide more uniform information for multiple users at the national, state, and local levels. These measures will allow monitoring of trends over time, identify high risk groups, and inform prevention, evaluation, and program planning efforts.</p> <p>These measures will address the following surveillance functions:</p> <ul style="list-style-type: none"> • Examination of time trends in AMI hospitalizations. • Identification of seasonal trends. • Assessment of geographic differences in hospitalizations. • Evaluation of differences in AMI hospitalizations by age, gender, and race/ethnicity. • With further analysis ... evaluation of disparities in AMI hospitalizations by factors such as age, race/ethnicity, gender, education, and/or income. • Determination of populations in need of targeted interventions. • Identification of possible environmental relationships that warrant further investigation or environmental public health action when AMI data are linked with environmental variables.

<p>Limitations of the Measures</p>	<p>Hospitalization data for AMIs omit persons who do not receive medical care or who are not hospitalized, including those who die in emergency rooms, in nursing homes, or at home without being admitted to a hospital, and those treated in outpatient settings.</p> <p>Differences in rates by time or area may reflect differences or changes in diagnostic techniques and criteria and in the coding of AMI or in medical care access.</p> <p>Differences in rates by area may be due to different sociodemographic characteristics and associated behaviors.</p> <p>When rates across geographic areas are compared, a variety of non-environmental factors, such as access to medical care and diet, can affect the likelihood of persons hospitalized for AMI.</p> <p>Reporting rates at the state and/or county level will not show the true AMI burden at a more local level (i.e., neighborhood).</p> <p>Reporting rates at the state and/or county level will not be resolved geographically enough to be linked with many types of environmental data.</p> <p>When looking at small geographic levels (e.g., ZIP code), users must consider appropriate cell suppression rules imposed by the data providers or individual state programs.</p> <p>Although duplicate records and transfers from one hospital to another are excluded, the measures are based upon events, not individuals, because no unique identifier is always available. When multiple admissions are not identified, the true prevalence will be overestimated.</p> <p>Even at the county level, the measures generated will often be based upon numbers too small to report or present without violating state and federal privacy guidelines and regulations. Careful adherence to cell suppression rules in cross tabulations is necessary, and methods to increase cell sizes by combining data across time (e.g., months, years) and geographic areas may be appropriate.</p>
<p>Data Sources</p>	<p>Numerator: State inpatient hospitalization data (using admission date)</p> <p>Denominator: U.S. Census Bureau population data</p>
<p>Limitations of Data Sources</p>	<p>State hospital discharge data: Using a measure of all AMI hospitalizations will include some</p>

	<p>transfers between hospitals for the same person for the same AMI event. Variations in the percentage of transfers or readmissions for the same AMI event may vary by geographic area and impact rates. However, efforts were made to identify and exclude transfers based on unique identifiers consisting of date of birth, zip code, gender, and encrypted social security number when available.</p> <p>Without reciprocal reporting agreements with abutting states, statewide measures and measures for geographic areas (e.g., counties) bordering other states may be underestimated because of health care utilization patterns.</p> <p>Each state must individually obtain permission to access and, in some states, provide payment to obtain the data.</p> <p>Veterans Affairs, Indian Health Services, and institutionalized (prison) populations are not usually included in hospitalization datasets.</p> <p>Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers to hospitalize patients</p> <p>Street address is not available in many states.</p> <p>Sometimes mailing address of patient is listed as the residence address of the patient.</p> <p>Patients may be exposed to environmental triggers in multiple locations, but hospital discharge geographic information is limited to residence.</p> <p>Since the data capture hospital discharges (rather than admissions), patients admitted toward the end of the year and discharged the following year will be omitted from the current year dataset.</p> <p>Data will need to be de-duplicated (i.e., remove duplicate records for the same event).</p> <p>There is usually a two-year lag period before data are available from the data owner.</p> <p>Census data: Available only every 10 years; thus, postcensal data will be estimated for calculating rates for years following the census year.</p> <p>Postcensal estimates at the ZIP code level are not available from the Census Bureau. These estimates should be extrapolated or purchased</p>
--	--

	from a vendor.
References	<ol style="list-style-type: none"> 1. Rosamond, W., et al., Heart disease and stroke statistics--2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. <i>Circulation</i>, 2007. 115(5): p. e69–171. 2. Boland, L.L., et al., Occurrence of unrecognized myocardial infarction in subjects aged 45 to 65 years (the ARIC study). <i>Am J Cardiol</i>, 2002. 90(9): p. 927–31. 3. Thom, T., et al., Cardiovascular disease in the United States and preventive approaches, in <i>Hurst's The Heart, Arteries and Veins</i>, V. Fuster, R. Alexander, and R. O'Rourke, Editors. 2001, McGraw-Hill: New York, NY. 4. Jones, D.W., et al., Risk factors for coronary heart disease in African Americans: the atherosclerosis risk in communities study, 1987–1997. <i>Arch Intern Med</i>, 2002. 162(22): p. 2565–71. 5. Kannel, W.B., et al., Menopause and risk of cardiovascular disease: the Framingham study. <i>Ann Intern Med</i>, 1976. 85(4): p. 447–52. 6. Pope, C.A., 3rd, et al., Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. <i>Circulation</i>, 2004. 109(1): p. 71–7. 7. Vermylen, J., et al., Ambient air pollution and acute myocardial infarction. <i>J Thromb Haemost</i>, 2005. 3(9): p. 1955–61. 8. Pope, C.A., 3rd, et al., Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. <i>Circulation</i>, 2006. 114(23): p. 2443–8 9. von Klot, S., et al., Ambient air pollution is associated with increased risk of hospital cardiac readmissions of myocardial infarction survivors in five European cities. <i>Circulation</i>, 2005. 112(20): p. 3073–9.

CONTENT DOMAIN: AIR QUALITY
INDICATOR: OZONE-DAYS ABOVE REGULATORY
STANDARD

Type of EPHT Indicator	Hazard
Measures	<ol style="list-style-type: none"> 1. Number of days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard (NAAQS) 2. Number of person-days with maximum 8-hour average ozone concentration over the National Ambient Air Quality Standard (NAAQS)
Derivation of Measures	<p>This overview provides the key technical points in how EPA and CDC processed EPA’s air quality data for use in the EPHT air indicators.</p> <p>Processing raw data First, EPA extracts the air quality data from the Air Quality System (AQS). EPA uses the following steps in developing the air data and measures for EPHT air quality indicators.</p> <p>Step 1: EPA accesses daily maximum 8-hour average ozone concentrations (ppm) (parameter code ‘44201’ and duration code ‘W’) and supplemental data fields (e.g. latitude, longitude, elevation) for all the monitoring sites across the US from the EPA’s Data Mart. The data are obtained only from monitors that are designated as Federal Reference Methods or equivalent. The data include any flagged values associated with exceptional events (high winds, fires, construction, etc) regardless of concurrence by the EPA Regional Office. EPA retains data from monitors that meet the minimum data completeness criteria set forth in the national air quality standard (i.e. if valid 8-hour averages are available for at least 75% of possible hours in a day or the maximum 8-hour average is above ozone 8-hr NAAQS).</p> <p>Step 2: For each monitoring site, retain the maximum concentration at the site for each monitored day. The pollutant occurrence code (poc) which distinguishes multiple monitors at a single site is listed in the output data set.</p> <p>Step 3: Site-level daily monitoring data are used to create ozone 8-hr maximum daily county-level dataset. Daily county-level dataset is created by retaining the maximum concentration among all monitors within the county for each monitored day. The county-level daily dataset is used to create number of days and number of person-days with ozone levels over the daily NAAQS measures.</p>

	<p>Creating Measures</p> <p>Step 3: Ozone levels decrease significantly in the colder parts of the year in many areas, ozone is required to be monitored at monitoring sites only during the ozone season, which is defined on a state by state basis. Only counties that have at least 75% of the days monitored during the ozone seasons are considered complete. The measures are computed only for counties that satisfy the completeness criteria.</p> <p><i>Number of days with Ozone levels over the NAAQS:</i></p> <p>Step 4: Select counties which pass the completeness criteria mentioned in Step 3.</p> <p>Step 5: To calculate the annual number of days over the daily NAAQS, sum the number of days with ozone levels over the daily 8-hr NAAQS for the entire year.</p> <p><i>Number of person-days with ozone levels over the NAAQS:</i></p> <p>Step 4: To calculate Person-days with ozone levels over the daily 8-hr NAAQS, multiply the number of days over the daily NAAQS by the total population of the county.</p>
Units	<ol style="list-style-type: none"> 1. Exceedance days 2. Population-weighted exceedance days
Geographic Scope	United States
Geographic Scale	County (where monitors exist)
Time Period	2001-current
Time Scale	Calendar year
Rationale	<p>According to the published literature, air pollution is associated with premature death, increased rates of hospitalization for respiratory and cardiovascular conditions, adverse birth outcomes, and lung cancer (2, 3). Air pollution places a large economic burden on the country. In a report prepared for the American Lung Association,(2) estimated that air pollution related illness was estimated to cost approximately \$100 billion annually (2) (1988 dollars) in the United States, with an estimated number of excess deaths ranging from 50,000 to 100,000 annually (3). More than half of the U.S. population, approximately 159 million persons, live in counties with unhealthy levels of air pollution in the form of either ozone or particulate matter (1). Elevated pollution levels depend on sources, transport, season geography, and atmospheric conditions. Each part of the country has its own level of pollution concentrations that can be exacerbated by many conditions, including stagnation, fire, or wind. The seasons for peak concentrations also vary between geographical regions. (4)</p> <p>The Clean Air Act, which was last amended in 1990, requires EPA to set NAAQS for widespread pollutants from numerous and diverse sources</p>

	<p>considered harmful to public health and the environment. The Clean Air Act established two types of national air quality standards. Primary standards set limits to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly. Secondary standards set limits to protect public welfare, including visibility impairment and damage to animals, crops, vegetation, and buildings. (5)</p> <p>Our indicator is based on comparing measured levels of ozone by county to the primary ozone 8-hr NAAQS, which is set at 75 ppb. The Clean Air Act requires periodic review of the science upon which the standards are based and the standards themselves. Primary air quality standards indicate the acceptable level of substances in the air before harm will occur based on proven scientific and medical research. State governments also set air quality standards. In several cases, California's standards or other benchmarks are more stringent than the EPA NAAQS.</p>
<p>Use of Measure</p>	<p>The indicator for the number of days with maximum 8-hour average ozone concentration over the standard is similar to EPA's analyses on number of days with air quality index (AQI) levels higher than 100 (for ozone) – see www.epa.gov/airtrends/aqi_info.html. This measure is consistent with the EPA and state AQI program efforts to communicate an area's air quality levels to the public. In addition, this indicator can be used to inform policy makers and the public of the degree of hazard within a state (by county or MSAs with monitors) during a year. For example, the number of days per year that ozone is higher than the NAAQS can be used to communicate to sensitive populations (such as asthmatics) the number of days that they may be exposed to unhealthy levels of ozone; this is the same level used in the air quality alerts that inform these sensitive populations when and how to reduce exposure. See http://www.epa.gov/air/airtrends/2007/report/groundlevelozone.pdf and http://www.epa.gov/air/airtrends/aqtrnd00/pdffiles/aqioz.pdf. In the use of the measure, it is important to explain that not all counties have monitors although most populated areas are monitored.</p>

<p>Limitations of The Measure</p>	<p>Since ozone levels decrease significantly in the colder parts of the year in many areas, ozone is required to be monitored only during the ozone season., which are designated on a State by State basis.(6)</p> <p>The number of high ozone days per year varies, which makes tracking trends over time difficult to analyze or interpret. The variability results from the following: a) the number of high ozone days is related to temperature; there will be more high days in hotter summers; and b) there are a small number of events per year, so for statistical reasons this type of measure will bounce around more than an average. c) When creating measures, we only consider monitors with 75% completeness during the ozone season and ozone seasons are designated on a state by state basis.</p> <p>Variation within counties may exist but will not be captured in this measure. Within these areas, the monitor with the highest reading on any day is used in the measure. Larger areas will have a broader range of pollution values and perhaps more monitors that may measure a high value on a given day. Thus, day and person-day estimates for larger areas may be biased higher than estimates for smaller areas. The relative variation among county populations in many states may be large enough relative to the variation in the number of days greater than the ozone NAAQS that the population component can dominate the calculation of the number of person-days. Thus, careful investigation of the underlying data to properly identify changes in population and air quality is needed when comparing person-days in space and time.</p> <p>The data for this indicator represent only counties that have air monitors; thus the data tend to reflect urban air quality (where most people live). Although populations in areas without monitors also may be exposed to ozone that exceeds the standard, they are not counted. The number of days that exceed the EPA NAAQS or other health benchmarks does not provide information regarding the severity (max concentrations) of potential exposures. The relationship between ambient concentrations and personal exposure is largely unknown and variable depending upon pollutant, activity patterns, and microenvironments.</p> <p>This indicator is not for use compliance determination with NAAQS or reasonable further progress toward attaining compliance.</p>
<p>Data Sources</p>	<p>Air quality data: EPA Air Explorer http://epa.gov/mxplorer/index.htm</p>
<p>Limitations of Data Sources</p>	<p>The AQS monitoring data, which are used in the calculation of measures, are not present for all counties and days.</p>
<p>References</p>	<ol style="list-style-type: none"> 1. American Lung Association. State of the Air 2004; 2004 [cited 2008 Dec 4]. Available from: http://lungaction.org/reports/sota04_full.html 2. Cannon J. The Health Costs of Air Pollution: A Survey of

	<p>Studies Published 1984– 1989. New York: American Lung Association; 1990.</p> <ol style="list-style-type: none">3. Dockery DW and Pope CA. Acute respiratory effects of particulate air pollution. <i>Annu Rev Public Health</i> 1994;15:107–132.4. US Environmental Protection Agency. US EPA general site on ozone effects. Available from: http://www.epa.gov/air/ozonepollution/health.html5. Criteria document for ozone NAAQS: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=1499236. Ozone Season definition by state: http://www.epa.gov/ttn/naaqs/ozone/ozonetech/40cfr58d.htm
--	---

CONTENT DOMAIN: AIR QUALITY
INDICATOR: PM_{2.5}—DAYS ABOVE REGULATORY STANDARD

Type of EPHT Indicator	Hazard
Measures	<ol style="list-style-type: none"> 1. Percent of days with PM_{2.5} levels over the National Ambient Air Quality Standard (NAAQS) 2. Number of person-days with PM_{2.5} over the National Ambient Air Quality Standard (NAAQS)
Derivation of Measures	<p>This overview provides the key technical points in how EPA and CDC processed EPA’s air quality data for use in the EPHT air indicators.</p> <p>Processing raw data: First, EPA extracts the air quality data from the Air Quality System (AQS). EPA uses the following steps in developing the air data and measures for EPHT air quality indicators.</p> <p>Step 1: EPA accesses PM_{2.5} daily concentrations ($\mu\text{g}/\text{m}^3$) (parameter code ‘88101’ and duration code ‘7’) and daily maximum 8-hour average ozone concentrations (ppm) (parameter code ‘44201’ and duration code ‘W’) and supplemental data fields (e.g. latitude, longitude, elevation) for all the monitoring sites across the US from the EPA’s Data Mart. The data are obtained only from monitors that are designated as Federal Reference Methods or equivalent. The data include any flagged values associated with exceptional events (high winds, fires, construction, etc) regardless of concurrence by the EPA Regional Office.</p> <p>Step 2: For each monitoring site, retain the maximum concentration at the site for each monitored day. The pollutant occurrence code (poc) which distinguishes multiple monitors at a single site is listed in the output data set.</p> <p>Step 3: Site-level daily monitoring data are used to create 24-hr maximum daily county-level PM_{2.5} dataset. Daily county-level dataset is created by retaining the maximum concentration among all monitors within the county for each monitored day. The county-level daily dataset is used to create percent of days and number of person-days with PM_{2.5} levels over the daily NAAQS measures.</p> <p>Creating Measures <i>Percent of days with PM_{2.5} levels over the NAAQS:</i> Step 4: To calculate the annual percent of days over the daily NAAQS, sum the number of days with PM_{2.5} levels over the daily NAAQS and</p>

	<p>divide by the total number of monitored days. Multiply this exceedance fraction by 100 to get percent of days.</p> <p>Number of person-days with PM_{2.5} levels over the NAAQS: Step 5: To calculate person-days with PM_{2.5} levels over the NAAQS multiply the exceedance fraction from Step 4 by 365 to get the annual days and then multiply by the total population of the county.</p> <p>For PM_{2.5} - days above regulatory standard indicator, tracking portal only displays counties that have year-round monitoring.</p>
Unit	<ol style="list-style-type: none"> 1. Exceedance days 2. Population weighted exceedance days
Geographic Scope	Contiguous United States
Geographic Scale	County (where monitors exist)
Time Period	2001-current
Time Scale	Calendar year
Rationale	<p>According to the published literature, air pollution is associated with premature death, increased rates of hospitalization for respiratory and cardiovascular conditions, adverse birth outcomes, and lung cancer (2,3,4). Air pollution places a large economic burden on the country. In a report prepared for the American Lung Association, (2) estimated that air pollution related illness was estimated to cost approximately \$100 billion annually (2) (1988 dollars) in the United States, with an estimated number of excess deaths ranging from 50,000 to 100,000 annually (3). More than half of the U.S. population, approximately 159 million persons, live in counties with unhealthy levels of air pollution in the form of either ozone or particulate matter (1). Elevated pollution levels depend on sources, transport, season geography, and atmospheric conditions. Each part of the country has its own level of pollution concentrations that can be exacerbated by many conditions, including stagnation, fire, or wind. The seasons for peak concentrations also vary between geographical regions.</p> <p>The Clean Air Act, which was last amended in 1990, requires EPA to set NAAQS for widespread pollutants from numerous and diverse sources considered harmful to public health and the environment. The Clean Air Act established two types of national air quality standards. Primary standards set limits to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly. Secondary standards set limits to protect public welfare, including visibility impairment and damage to animals, crops, vegetation, and buildings.</p> <p>Our indicator is based on comparing measured levels of PM_{2.5} by county to the 24-hr NAAQS for PM_{2.5}, which is set at 35 $\mu\text{g}/\text{m}^3$. The</p>

	<p>Clean Air Act requires periodic review of the science upon which the standards are based and the standards themselves. Primary air quality standards indicate the acceptable level of substances in the air before harm will occur based on proven scientific and medical research. State governments also set air quality standards. In several cases, California's standards or other benchmarks are more stringent than the EPA NAAQS. (5)</p>
<p>Use of the Measure</p>	<p>This indicator can be used to inform the public and policy makers of the degree of potential exposures within a state (for counties with monitors) during a year. For example, the percentage of days per year that PM_{2.5} is higher than the NAAQS can be used to communicate to sensitive populations (such as asthmatics) the percentage of days that they may be exposed to unhealthy levels of PM_{2.5}; this is similar to the level used in the Air Quality Alerts that inform these sensitive populations when and how to reduce exposure.</p> <p>The number of person-days may be directed toward policy makers who are interested in roughly comparing population exposure between areas, to determine the areas most in need of prevention and pollution control activities.</p>
<p>Limitations of the Measure</p>	<p>The data for this indicator represent highly populated counties that have PM_{2.5} monitors. As a result, the data tend to reflect urban air quality and longer-term average air quality levels. Populations in counties without monitors may also be exposed to concentrations that exceed a standard.</p> <p>The percentage of days during which the EPA NAAQS or other health benchmarks are exceeded does not provide information regarding the severity (maximum concentrations) of potential exposures. Even with these limitations, trends in PM_{2.5} levels are a useful measure to describe public health concerns within these areas. We identify several limitations with this indicator below.</p> <p>This indicator is based on the percentage of high days rather than the total number of high days to highlight the fact that PM_{2.5} monitors follow different operating schedules. Most operate on a once-every-third day schedule, but a small proportion operates on a daily or once-every-sixth day schedule. Because most of the monitors do not take measurements every day, the number of short-term events (e.g., days in which the NAAQS is exceeded) is uncertain, and except where PM_{2.5} levels vary uniformly throughout the year, estimating short-term measures that are representative of short-term exposures over a year is complex. To address this limitation, the measure can be based on the percentage of monitored days. It should be noted that state air programs will be evaluating the daily PM_{2.5} NAAQS by using a frequency-based analysis to determine whether areas within the state</p>

	<p>attain this NAAQS.</p> <p>Populations in counties without monitors may be exposed to concentrations that exceed a standard. Person-day estimates for larger, highly populated counties may be biased higher than estimates for smaller and lower populated counties. The indicator uses the highest value of all monitors in the area so that larger counties with more monitors may have a broader range of pollution values and greater potential to measure a high day than smaller counties with fewer monitors</p> <p>The relationship between ambient concentrations and personal exposure is largely unknown, and it varies depending upon pollutant, activity patterns, and microenvironments.</p> <p>Because the number of high PM_{2.5} days per year can vary considerably, tracking trends over time needs to be done carefully. The variability results because: the number of high PM_{2.5} days is related to meteorological factors (e.g., temperature and mixing heights), and few events occur per year, so that this type of extreme value measure will vary considerably for statistical reasons. When creating measures, we only consider monitors, which have at least 11 observations per calendar quarter.</p>
Data Sources	<p>Air-quality data: EPA Air Explorer http://epa.gov/mxplorer/index.htm</p> <p>Population data: county population data can be found at http://www.census.gov/popest/counties/CO-EST2006-01.html</p>
Limitations of Data Sources	<p>Air-monitoring data provides information regarding concentrations around the specific location of each monitor. For PM_{2.5} this can be a rather large area, except when unusual local emissions (agricultural fires) occur. Within-county variation in concentrations will likely exist but will not be captured in this measure. Many PM_{2.5} monitors operate once-every third day (some once-every-sixth day); a few monitors operate every day.</p>
References	<ol style="list-style-type: none"> 1. American Lung Association. State of the Air 2004; 2004 [cited 2008 Dec 4]. Available from: http://lungaction.org/reports/sota04_full.html 2. Cannon J. The Health Costs of Air Pollution: A Survey of Studies Published 1984– 1989. New York: American Lung Association; 1990. 3. Dockery DW and Pope CA. Acute respiratory effects of particulate air pollution. Annu Rev Public Health 1994;15:107–132.

	<ol style="list-style-type: none">4. Schwartz, J. Air pollution and hospital admissions for heart disease in eight U.S. counties. <i>Epidemiology</i> 1999;10:17–22. 5. U.S. Environmental Protection Agency. U.S. EPA Criteria Document for PM. Available from: Volume 1 <u>VOL I FINAL PM AQCD OCT2004.PDF</u> and Volume 2 <u>VOL II FINAL PM AQCD OCT2004.PDF</u>
--	--

CONTENT DOMAIN: AIR QUALITY
INDICATOR: ANNUAL PM_{2.5} LEVEL

Type of EPHT Indicator	Hazard
Measure	<ol style="list-style-type: none"> 1. Annual average ambient concentrations of PM_{2.5} in micrograms per cubic meter (based on seasonal averages and daily measurement) 2. Annual percent of population living in counties exceeding the National Ambient Air Quality Standard (compared to percent of population living in counties that meet the standard and percent of population living in counties without PM_{2.5} monitoring)
Derivation of Measure	<p>First, EPA extracts the air quality data from the Air Quality System (AQS). EPA uses the following steps in developing the air data and measures for EPHT air quality indicators.</p> <p>Processing raw data</p> <p>Step 1: EPA accesses PM_{2.5} daily concentrations (mcg/m³) (parameter code ‘88101’ and duration code ‘7’) and supplemental data fields (e.g. latitude, longitude, elevation) for all the monitoring sites across the US from the EPA’s Data Mart. The data are obtained only from monitors that are designated as Federal Reference Methods or equivalent. The data include any flagged values associated with exceptional events (high winds, fires, construction, etc) regardless of concurrence by the EPA Regional Office.</p> <p>Step 2: For each monitoring site, retain the maximum concentration at the site for each monitored day. The pollutant occurrence code (poc) which distinguishes multiple monitors at a single site is listed in the output data set.</p> <p>Creating Measures</p> <p>Step 3: The annual average measures of PM_{2.5} are created using the site-level daily monitoring data. Only monitors that have at least 11 observations for each of the four calendar quarters are considered complete. The annual averages are computed only for monitors that satisfy the completeness criteria.</p> <p><i>Annual average ambient concentrations of PM_{2.5} measure:</i></p> <p>Step 4: Select monitors with complete quarterly and annual data using the site-level monitoring data.</p> <p>Step 5: Calculate the quarterly average for each calendar quarter and then compute the annual average for each monitor with four valid quarters by averaging the quarterly averages. If a county has more than one monitor then the maximum annual average among monitors with complete (4 valid quarters) data is assigned as the annual average for that county.</p>

	<p><i>Annual percent of population living in counties exceeding the NAAQS (compared to percent of population living in counties that meet the standard and percent of population living in counties without PM_{2.5} monitoring) measure:</i></p> <p>Step 6a: This is a state-level measure and uses the county-level annual average concentrations calculated in step 3.</p> <p>Step 6b: To calculate the annual percent of population living in counties that exceed the annual NAAQS, sum the population of all counties that exceed the annual NAAQS and divide by the total population of the state. Multiply this fraction by 100 to get percent.</p> <p>Step 6c: To calculate the annual percent of population living in counties that meet the annual NAAQS, sum the population of all counties that meet the annual NAAQS and divide by the total population of the state. Multiply this fraction by 100 to get percent.</p> <p>Step 6d: To calculate the annual percent of population living in counties that do not have complete monitors, sum the population of all counties that do not have complete monitors and divide by the total population of the state. Multiply this fraction by 100 to get percent.</p>
Unit	<ol style="list-style-type: none"> 1. Microgram per cubic meter ($\mu\text{g}/\text{m}^3$) 2. Population proportion by hazard level
Geographic Scope	Contiguous United States
Geographic Scale	County (where monitors exist)
Time Period	2001- current
Time scale	Calendar year
Rationale	<p>According to work conducted by Pope et al. (1), long-term exposure to PM_{2.5} is related to many adverse health conditions. Each 10 $\mu\text{g}/\text{m}^3$ elevation in PM_{2.5} is related to an 8% increase in lung cancer mortality, a 6% increase in cardiopulmonary mortality, and a 4% increase in death from general causes.(2)</p> <p>The annual average provides an indication of the long-term trends in overall PM_{2.5} burden, relevant to its long-term effects.</p> <p>The percent of the population living in counties that exceed the standard provides an indication of the population at risk for long-term exposure.</p> <p>Note: these indicators are similar to indicators developed by EPA and state air quality agencies for use in air quality stats and trends analyses and reports (see www.epa.gov/airtrends)</p>
Use of The Measure	This indicator can be used to inform policy makers and the public about the degree of potential exposures to fine particles within a state during a year and over time (trends). This is appropriate, as many existing health studies have found the strongest association with health outcomes based on long-

	<p>term studies; thus, EPA developed the annual NAAQS at 15 ug/m³. The indicator (annual average PM_{2.5} concentrations) can be compared to the National Ambient Air Quality Standard (NAAQS) level of 15 ug/m³ or other health-based standards (although not in a regulatory manner) to communicate the degree of public health concern to policy makers and the general public. (3)</p>
Limitations of the Measure	<p>This measure provides a general indication of the overall trend in annual PM_{2.5} concentrations. It may be affected by density and placement of monitors, and coverage will vary across the country and within states. It does not directly reflect exposure. Certain geographic areas, such as those near busy roads, are likely to have higher values.</p> <p>When creating measures we only consider monitors that have at least 11 observations per calendar quarter. It is important to understand that this indicator is not for use—compliance determination with NAAQS or reasonable further progress toward attaining compliance.</p> <p>The relationship between ambient concentrations and personal exposure is largely unknown, and it varies depending upon pollutant, activity patterns, and microenvironments.</p> <p>The percent of state population living in counties with no PM_{2.5} measurements must always be considered when attempting to estimate the proportion of population at risk.</p>
Data Sources	<p>EPA Air Quality System Monitoring Data, State Air Monitoring Data. http://www.epa.gov/air/data/aqsdb.html</p>
Limitations of Data Sources	<p>Air monitoring data provides information regarding concentrations around the specific location of each monitor. For PM_{2.5} this can be a rather large area, except when unusual local emissions (agricultural fires) occur. Within-county variation in concentrations will likely exist but will not be captured in this measure. Many PM_{2.5} monitors operate once-every-third day (some once-every-sixth day) and a few measure every day</p>
References	<p>Dockery DW and Pope CA. Acute respiratory effects of particulate air pollution. Annu Rev Public Health 1994;15:107–132.</p> <p>Cannon J. The Health Costs of Air Pollution: A Survey of Studies Published 1984– 1989. New York: American Lung Association; 1990.</p> <p>U.S. Environmental Protection Agency. U.S. EPA Criteria Document for PM. Available from: Volume 1 VOL_I_FINAL_PM_AQCD_OCT2004.PDF and Volume 2 VOL_II_FINAL_PM_AQCD_OCT2004.PDF</p>

CONTENT DOMAIN: ASTHMA
INDICATOR: HOSPITALIZATIONS FOR ASTHMA

Type of EPHT Indicator	Health Outcome
Measures	<ol style="list-style-type: none"> 1. Number of hospitalizations for asthma 2. Minimum daily number of hospitalizations for asthma by month 3. Maximum daily number of hospitalizations for asthma by month 4. Average daily number of hospitalizations for asthma by month 5. Crude rate of hospitalization for asthma by age group (total, 0-4, 5-14, 15-34, 35-64, and 65+) per 10,000 population 6. Age-adjusted rate hospitalizations for asthma per 10,000 population (all ages) <p>When supported by sufficient data volume, the measures may also be reported stratified by sex, race, and/or ethnicity.</p>
Derivation of Measures	<p>Numerator: Resident hospitalizations for asthma, ICD-9-CM: 493.XX.</p> <p>Denominator: Midyear resident population.</p> <p>Adjustment: Age-adjustment by the direct method to Year 2000 U.S. Standard population</p>
Unit	Hospital admission (categorized by discharge diagnosis)
Geographic Scope	State and national (tracking network states)
Geographic Scale	State and county
Time Period	Hospital admissions from January 1 through December 31 for each year, 2000–current
Time Scale	Daily, monthly, and annually (as appropriate for the measure)
Rationale	<p>In 2004, 20.5 million people in the United States reported having asthma. In 2003, there were more than 574,000 hospitalizations for asthma. In 2002, there were more than 4,200 deaths in which asthma was the underlying cause. Asthma is the leading chronic health condition among children. There are also large racial, income, and geographic disparities in poor asthma outcomes. Asthma causes lower quality of life, preventable undesirable health outcomes, and large direct and indirect economic costs. Environment attributable fractions of the 1988–1994 economic costs for asthma were 39.2% for children aged <6 years and 44.4% for children aged 6–16 year, costing more than \$400 million for each age group.</p> <p>A number of epidemiologic studies have reported associations between air pollution exposures and asthma. The association between ambient</p>

	<p>air particulate matter (PM) concentrations and asthma, including increased hospital admissions, is well documented. Models demonstrate 5–20% increases in respiratory-related hospital admissions per 50µg/m³ of PM₁₀ and 5–15% per 25µg/m³ of PM_{2.5}, with the largest effect on asthma admissions.</p> <p>In the eastern United States, summer ozone pollution was associated with more than 50,000 hospital admissions per year for asthma and other respiratory emergencies. Large multi-city and individual city studies found a positive association between ozone and total respiratory hospital admissions, including asthma, especially during the warm season. Among U.S. and Canadian studies, the ozone-associated increase in respiratory hospital admissions ranged from 2-30% per 20 ppb (24 hour), 30 ppb (8-hour) or 40 ppb (1-hour) increment of ozone in warm seasons.</p> <p>In 2000, the Institute of Medicine concluded that allergens produced by cats, cockroaches, and house dust mites exacerbates asthma, as does exposure to environmental tobacco smoke (ETS) in pre-school aged children. A 2005 California Air Resources Board report concluded that ETS exacerbates asthma in children and adults (CARB, 2005). That report also estimated 202,300 childhood asthma episodes occur each year in the United States as a result of exposure to ETS.</p>
<p>Use of the Measures</p>	<p>Developing a standardized analytic method for asthma hospital admissions among residents in each state will provide more uniform information for multiple users at the national, state, and local levels. These measures will allow monitoring of trends over time, identify high risk groups, and inform prevention, evaluation, and program planning efforts.</p> <p>These measures will address the following surveillance functions:</p> <ul style="list-style-type: none"> • How many hospitalizations for asthma occur in every month? • Is there a seasonal or temporal trend of asthma hospitalizations? • What’s the distribution of asthma hospitalizations by place of residence? • How do hospitalizations for asthma differ between geographic areas (e.g., ZIP code, county, state, region)? • With further analysis ... Are there disparities in asthma hospitalizations by factors such as age, race, ethnicity, gender, education, and/or income?

	<ul style="list-style-type: none"> • Which populations need targeted interventions? • When asthma data are linked with environmental variables, do the linked measures identify environmental relationships that warrant further investigation or environmental public health action?
<p>Limitations of the Measures</p>	<p>Hospitalization data, by definition, do not include asthma among individuals who do not receive medical care or who are not hospitalized, including those who die in emergency rooms, in nursing homes, or at home without being admitted to a hospital, and those treated in outpatient settings.</p> <p>Differences in rates by time or area may reflect differences or changes in diagnostic techniques and criteria and in the coding of asthma.</p> <p>Reporting rates at the state and/or county level will not show the true asthma burden at a more local level (i.e., neighborhood).</p> <p>Differences in rates by area may be due to different sociodemographic characteristics and associated behaviors.</p> <p>When rates across geographic areas are compared, many non-environmental factors, such as access to medical care and diet, can affect the likelihood of a person being hospitalized for asthma.</p> <p>Reporting rates at the state and/or county level will not be resolved geographically enough to be linked with many types of environmental data.</p> <p>When looking at small geographic levels (e.g., ZIP code), users must consider appropriate cell suppression rules imposed by the data providers or individual state programs.</p> <p>Although duplicate records and transfers from one hospital to another are excluded, the measures are based upon events, not individuals, because no unique identifier is always available. When multiple admissions are not identified, the true prevalence will be overestimated.</p> <p>Even at the county level, the measures generated will often be based upon numbers too small to report or present without violating state and federal privacy guidelines and regulations. Careful adherence to cell suppression rules in cross tabulations is necessary, and methods to increase cell sizes by combining data across time (e.g., months, years) and geographic areas may be appropriate.</p>
<p>Data Sources</p>	<p>Numerator: State inpatient hospitalization data (using admission date)</p>

	<p>Denominator: US Census Bureau population data</p>
<p>Limitations of Data Sources</p>	<p>State hospital discharge data: The use of a measure of all asthma hospitalizations will include some transfers between hospitals for the same person for the same asthma event. Variations in the percentage of transfers or readmissions for the same asthma event may vary by geographic area and impact rates. However, efforts were made to identify and exclude transfers based on unique identifiers consisting of date of birth, zip code, gender, and encrypted social security number when available.</p> <p>Without reciprocal reporting agreements with abutting states, statewide measures and measures for geographic areas (e.g., counties) bordering other states may be underestimated because of health care utilization patterns.</p> <p>Each state must individually obtain permission to access and, in some states, provide payment to obtain the data.</p> <p>Veterans Affairs, Indian Health Services, and institutionalized (prison) populations are excluded.</p> <p>Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers to hospitalize patients</p> <p>Street address is not available in many states.</p> <p>Sometimes mailing address of patient is listed as the residence address of the patient.</p> <p>Patients may be exposed to environmental triggers in multiple locations, but hospital discharge geographic information is limited to residence.</p> <p>Since the data capture hospital discharges (rather than admissions), patients admitted toward the end of the year and discharged the following year will be omitted from the current year dataset.</p> <p>Data will need to be de-duplicated (i.e., remove duplicate records for the same event).</p> <p>There is usually a two-year lag period before data are available from the data owner.</p>

	<p>Census data: Available only every 10 years; thus, postcensal data must be estimated when rates for years following the census year are calculated.</p> <p>Postcensal estimates at the ZIP code level are not available from the Census Bureau. These need to be extrapolated or purchased from a vendor.</p>
References	<ol style="list-style-type: none"> 1. Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System (BRFSS) Prevalence Data. 1999–2010 November 16, 2011 [cited 2012 July 2]; Available from: http://www.cdc.gov/asthma/brfss/default.htm#00. 2. Mannino, D.M., et al., Surveillance for asthma—United States, 1960–1995. <i>MMWR CDC Surveill Summ</i>, 1998. 47(SS-1): p. 1–28. 3. Mannino, D.M., et al., Surveillance for asthma—United States, 1980–1999. <i>MMWR Surveill Summ</i>, 2002. 51(1): p. 1–13. 4. Britton, J. and S. Lewis, Epidemiology of Childhood Asthma, in <i>Asthma: Epidemiology, Anti-Inflammatory Therapy and Future Trends</i>, M. Gjembycz and B. O'Connor, Editors. 2000, Birkhäuser Basel: Switzerland. p. 25–56. 5. Gold, D.R. and R. Wright, Population disparities in asthma. <i>Annu Rev Public Health</i>, 2005. 26: p. 89–113. 6. Lanphear, B.P., et al., Residential exposures associated with asthma in US children. <i>Pediatrics</i>, 2001. 107(3): p. 505–11. 7. Lanphear, B.P., et al., Contribution of residential exposures to asthma in us children and adolescents. <i>Pediatrics</i>, 2001. 107(6): p. E98. 8. Redd, S.C., Asthma in the United States: burden and current theories. <i>Environ Health Perspect</i>, 2002. 110 Suppl 4: p. 557–60. 9. Arif, A.A., J.E. Rohrer, and G.L. Delclos, A population-based study of asthma, quality of life, and occupation among elderly Hispanic and non-Hispanic whites: a cross-sectional investigation. <i>BMC Public Health</i>, 2005. 5: p. 97. 10. Jorres, R.M.H., Atmospheric pollutants, in <i>Asthma: Basic Mechanisms and Clinical Management</i>, P. Barnes, I. Rodger, and N. Thomson, Editors. 1998, Academic Press: London. p. 589–596. 11. Trasande, L. and G.D. Thurston, The role of air pollution in asthma and other pediatric morbidities. <i>J Allergy Clin Immunol</i>, 2005. 115(4): p. 689–99. 12. Jaffe, D.H., M.E. Singer, and A.A. Rimm, Air pollution and emergency department visits for asthma among Ohio Medicaid

	<p>recipients, 1991–1996. Environ Res, 2003. 91(1): p. 21–8.</p> <p>13. U.S. Environmental Protection Agency, Air Quality Criteria for Particulate Matter (Final Report, Oct 2004), 2004, U.S. Environmental Protection Agency. EPA 600/P-99/002aF-bF: Washington, DC.</p> <p>Institute of Medicine, Committee on the Assessment of Asthma and Indoor Air. Division of Health Promotion. Disease Prevention. Clearing the Air: Asthma and Indoor Air Exposures 2000, Washington, DC: The National Academies Press.</p>
--	--

Indicator Template
Content Area: Asthma
Indicator: Emergency Department Visits for Asthma
Environmental Public Health Tracking

Type of EPHT Indicator	Health outcome
Measures	<ol style="list-style-type: none"> 1. Annual age-adjusted rate of emergency department visits for asthma per 10,000 population 2. Annual crude rate of emergency department visits for asthma per 10,000 population 3. Annual number of emergency department visits for asthma 4. Average Number of emergency department visits for asthma as primary diagnosis per month
Derivation of Measure(s)	<p><i>Numerator:</i></p> <ul style="list-style-type: none"> • Emergency Department Visits during a calendar year with asthma (ICD-9-CM 493) as the primary diagnosis (includes records for ED Visits resulting in a hospitalization) • Both inpatient and outpatient records with duplicates removed and transfers to other hospitals included <p><i>Denominator:</i></p> <ul style="list-style-type: none"> • Annual population estimates for state and county from U.S. Census Bureau <p><i>Adjustment:</i></p> <ul style="list-style-type: none"> • Age-adjustment by the direct method to the Year 2000 US Standard population • U.S. 2000 standard population by age categories from Surveillance Epidemiology and End Results (SEER), National Cancer Institute
Unit	<ol style="list-style-type: none"> 1. Age-adjusted rate per 10,000 population 2. Rate per 10,000 population 3. Number 4. Number
Geographic Scope	State and national
Geographic Scale	Residents of jurisdiction – State, County
Time Period	Hospital admissions between January 1 to December 31, inclusive, for each year, 2000–
Time Scale	Daily, monthly, and annually (as appropriate for the measure)
Rationale	Asthma continues to be a serious public health problem that affects over 23 million people including 7 million children in the United States. In 2008,

there were 456,000 hospitalizations and 1.8 million emergency department visits (ED) for asthma.³ Asthma is the leading chronic health condition among children.⁴ There are also large racial, income, and geographic disparities in poor asthma outcomes.⁵ Asthma causes lower quality of life, preventable undesirable health outcomes, and large direct and indirect economic costs.

As a chronic respiratory disease, asthma attacks interfere with everyday activities. According to NCHS National Health Interview Survey, there were 10.5 million missed school days among children age 5–17 years and over 14.5 million missed work days in adults age 18 years or over in 2008. In 2007, there were over 3,400 deaths in which asthma was the underlying cause.

Environment Attributable Fractions of the 1988-1994 economic costs for asthma were 39.2% for children <6 years of age and 44.4% for 6- to 16-year-olds, costing more than \$400 million for each age group. According to a more recent estimation 30% of asthma exacerbations among children were related to the environment. This was associated with an annual cost of \$2.0 billion. Despite the availability of effective prevention measures, asthma associated costs are increasing.

Associations between environmental exposures and asthma have been consistently demonstrated. Many outdoor air pollutants have been associated with increased asthma ED visits. There is strong scientific evidence for direct associations between increased ozone concentrations and increases in asthma ED visits, in children and adults. In one study, asthma ED visits increased by 33 percent when daily 1-hour maximum ozone concentrations exceeded 75 ppb. Associations between asthma-related ED visits and ambient air particulate matter—both PM₁₀ and PM_{2.5}—have been repeatedly confirmed, and are especially robust for children. Other pollutants related to higher asthma ED visit totals include carbon monoxide (CO), nitrogen dioxide (NO₂), and pollution from coal and petrochemical sources. Other outdoor environmental triggers for asthma ED visits in children include weed and tree pollen, and ambient temperature. Increased asthma ED visits has also been associated with environmental tobacco smoke (ETS). Asthma ED visits in children are consistently higher in the fall, co-occurring with the start of the school year; increases in asthma ED visits in children have been shown to be related to increased respiratory viral infections. The state emergency department visit data is electronically maintained and is available in almost every state in the U.S. Data stewards for 18 grantees maintain ED data.

The data has comparable basic information about each visit and can provide a better tracking measure of asthma burden than inpatient hospitalization data on its own. These measures can be used to evaluate the impact of ambient air pollution on respiratory health of children and adults. Also, the measures can be used for better resource management to further reduce the asthma related

	<p>expenditures. Combined with inpatient asthma data, emergency department data will provide more complete spatial and temporal trends for asthma.</p> <p>Additionally, emergency department visits are believed to be largely preventable if managed properly through the use of Asthma Action Plans and avoiding environmental triggers. This offers an outcome that may be a more measurable indicator of environmental events and of public health intervention</p>
<p>Use of the Measure</p>	<p>The development of a single analytic method for asthma emergency department visits among persons living in state will inform multiple users:</p> <p><i>State:</i></p> <ul style="list-style-type: none"> • May be linked with other risk factors such as air pollution to identify susceptible populations and explore ecologic relationships • Allows for a better understanding of what the asthma surveillance data represents when interpreting number of inpatient hospitalizations • Permits the monitoring of trends temporally and spatially <p><i>National:</i></p> <ul style="list-style-type: none"> • It will allow for comparison across states which can be used to target interventions (especially for CDC and EPA). <p><i>Public:</i></p> <ul style="list-style-type: none"> • Public and concerned community members will be able to view the Tracking Network webpage and learn the annual rate of asthma emergency department visits and burden of asthma is high in their community from.
<p>Limitations of the Measure</p>	<ul style="list-style-type: none"> • Numbers may be too small in rural areas to calculate stable rates. • These measures do not account for other causes (triggers) of asthma or other reasons for visiting the ED. • The timing of the exposure may not correspond with the timing of the asthma exacerbation leading to the ED visit. • Individuals may have asthma exacerbations due to exposure to an environmental risk factor that does not result in an ED visit and thus are not captured in this measure. • Cannot combine counts from asthma ED visit measure with counts from asthma hospitalization measure because records for ED patients who are subsequently hospitalized are already counted as hospitalizations (i.e., would result in double-counting of events). • Differences in rates by time or area may reflect differences or changes in diagnostic techniques and criteria and in the coding of asthma. • Reporting rates at the state and/or county level will not show the true asthma burden at a more local level (i.e. neighborhood).

	<ul style="list-style-type: none"> • Differences in rates by area may be due to different socio-demographic characteristics and associated behaviors. • When comparing rates across geographic areas, a variety on non-environmental factors, such as access to medical care and diet, can impact the likelihood of persons hospitalized for asthma. • Reporting rates at the state and/or county level will not be geographically resolved enough to be linked with many types of environmental data. • When looking at small geographic levels (e.g. ZIP code), users must take into consideration appropriate cell suppression rules imposed by the data providers or individual state programs. • Although duplicate records and transfers from one hospital to another are excluded, the measures are based upon events, not individuals, because no unique identifier is always available. When multiple admissions are not identified, the true prevalence will be overestimated. • Even at the county level it can be expected that the measures generated will often be based upon numbers too small to report or present without violating state and federal privacy guidelines and regulations. Careful adherence to cell suppression rules in cross tabulations is necessary and methods to increase cell sizes by combining data across time (e.g., months, years) and geographic areas may be appropriate.
Data Sources	<p><i>Numerator:</i> State inpatient emergency department data <i>Denominator:</i> US Census Bureau population data</p>
Limitations of Data Sources	<p><i>State emergency department data:</i></p> <ul style="list-style-type: none"> • State emergency department data • Need to obtain permission to use; not publicly available • ED visits for asthma are only one piece of a larger picture that describes asthma burden. • Veteran’s Administration, Indian Health Service and institutionalized (e.g. prison) populations are excluded • In-state residents who visit in surrounding states would not be included unless states have emergency department data sharing agreements. • Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers. • Do not have a zip code for all patients. • Sometimes mailing address of patient (e.g., P.O. Box) is listed as the residence address of the patient • Patients may be exposed to environmental triggers in multiple locations, but ED geographic information is limited to residence. • Data will need to be de-duplicated using a standardized method. <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the

	Census Bureau. These need to be extrapolated or purchased from a vendor.
Related Indicators	<ul style="list-style-type: none"> • Hospitalizations for Asthma • Asthma Prevalence among Adults and Children
References	<ol style="list-style-type: none"> 1. Summary Health Statistics for U.S. Adults: National Health Interview Survey, 2008, Tables 3 and 4. http://www.cdc.gov/nchs/data/series/sr_10/sr10_242.pdf 2. Summary Health Statistics for U.S. Children: National Health Interview Survey, 2008, Table 1. http://www.cdc.gov/nchs/data/series/sr_10/sr10_244.pdf 3. Akinbami LJ, Moorman JE, Liu X. Asthma Prevalence, Health Care Use, and Mortality: United States, 2005–2009. National Health Statistics Reports; No 32. Hyattsville, MD: National Center for Health Statistics, 2011. 4. Britton JR, Lewis SA, Epidemiology of childhood asthma. In Asthma: Epidemiology, Anti-Inflammatory Therapy and Future Trends; MA Giembycz and BJ O’Connor (Eds.),. Switzerland: Birkhäuser Verlag, 2000, pp. 25-56. 5. Gold DR, Wright R, Population disparities in asthma. Annu. Rev. Public Health 2005; 26: 89-113. 6. Lanphear BP, Aligne CA, Auinger P, et al., Residential exposures associated with asthma in US children. Pediatrics 2001; 107: 505-511. 7. Lanphear BP, Kahn RS, Berger O, et al., Contribution of residential exposures to asthma in US children and adolescents. Pediatrics 2001; 107: e98. 8. Redd SC. Asthma in the United States: Burden and current theories. Environ Health Perspect 2002; 110 (Suppl 4): 557-60. 9. Arif AA, Rohrer JE, Delclos GL. A population-based study of asthma, quality of life, and occupation among elderly Hispanic and non-Hispanic whites: a cross-sectional investigation. BMC Public Health 2005; 5: 97. 10. Pruss-Ustun A, Corvalan C. Preventing disease through health environments. Towards an estimate of the environmental burden of disease. World Health Organization. 2006. 11. Landrigan PJ, Schechter CB, et al. Environmental Pollutants and Disease in American Children: Estimates of Morbidity, Mortality, and Costs for Lead Poisoning, Asthma, Cancer, and Developmental Disabilities. Environ Health Perspect. 2002;110:721-728. 12. Bahadori K, Doyle-Waters MM, Marra C, Lynd L, Alasaly K, Swiston J, FitzGerald JM. Economic burden of asthma: a systematic review. BMC Pulm Med. 2009 May 19;9:24.

13. Babin SM, Burkhom HS, Holtry RS, *et al.* Pediatric patient asthma-related emergency department visits and admissions in Washington, DC, from 2001-2004, and associations with air quality, socio-economic status and age group. *Environ Health*. 2007; 6: 9.
14. Peel JL, Tolbert PE, Klein M, *et al.* Ambient air pollution and respiratory emergency department visits. *Epidemiology*. 2005; 16: 164-174.
15. Stieb DM, Burnett RT, Beveridge RC, *et al.* Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. *Environ Health Perspect*. 1996; 104: 1354-60.
16. Tolbert PE, Mulholland JA, Macintosh DL, *et al.* Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia. *Am J Epidemiol*. 2000; 151: 798-810.
17. Norris G, VoungPong SN, Koenig JQ, *et al.* An association between fine particles and asthma emergency department visits for children in Seattle. *Environ Health Perspect*. 1999; 107: 489-93.
18. Sun HL, Chou MC, Lue KH. The relationship of air pollution to ED visits for asthma differs between children and adults. *Am J Emerg Med*. 2006; 24: 709-13.
19. Slaughter JC, Kim E, Sheppard L, *et al.* Association between particulate matter and emergency room visits, hospital admissions and mortality in Spokane, Washington. *J Expo Anal Environ Epidemiol*. 2005; 15: 153-9.
20. Villeneuve PJ, Chen L, Rowe BH, *et al.* Outdoor air pollution and emergency department visits for asthma among children and adults: A case-crossover study in northern Alberta, Canada. *Environ Health*. 2007; 6:40.
21. Teach SJ, Crain EF, Quint DM, *et al.* Indoor environmental exposures among children with asthma seen in an urban emergency department. *Pediatrics*. 2006; 117: S152-8.
22. Vargas PA, Brenner B, Clark S, *et al.* Exposure to environmental tobacco smoke among children presenting to the emergency department with acute asthma: A multicenter study. *Pediatr Pulmonol*. 2007; 42: 646-55.
23. Baibergenova A, Thabane L, Akhtar-Danesh N, *et al.* Effect of gender, age, and severity of asthma attack on patterns of emergency department visits due to asthma by month and day of the week. *Eur J Epidemiol*. 2005; 20: 947-56.
24. Silverman RA, Ito K, Stevenson L, *et al.* The relationship of fall school opening and emergency department asthma visits in a large metropolitan area. *Arch Pediatr Adolesc Med*. 2005; 159: 818-23.
25. Clark NA, Demers PA, Karr CJ, Koehoorn M, Lencar C, Tamburic L, Brauer M. Effect of early life exposure to air pollution on development

	<p>of childhood asthma. Environ Health Perspect. 2010;118(2):284-90.</p> <p>26. Expert Panel Report 3. Guidelines for the Diagnosis and Management of Asthma. Bethesda, MD: US Department of Health and Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute, National Asthma Education and Prevention Program, 2007.</p>
--	---

CONTENT DOMAIN: BIRTH DEFECTS

INDICATOR: PREVALENCE OF BIRTH DEFECTS

Type of EPHT Indicator	Health Outcome
Measure	<p>Five year prevalence rates of 12 birth defects per 10,000 live births.</p> <ol style="list-style-type: none"> 1. Anencephaly 2. Spina bifida (without anencephaly) 3. Hypoplastic left heart syndrome 4. Tetralogy of Fallot 5. Transposition of the great arteries (vessels) 6. Cleft lip with or without cleft palate 7. Cleft palate without cleft lip 8. Hypospadias (male births only) 9. Gastroschisis 10. Upper limb deficiencies 11. Lower limb deficiencies 12. Trisomy 21 <ul style="list-style-type: none"> ○ Among mothers <35 years of age at delivery ○ Among mothers ≥35 years of age at delivery <p>Five year prevalence rates at the state level are reported stratified by maternal age at delivery, maternal ethnicity/race, and infant sex. Five year prevalence rates at the county level are reported stratified by one demographic variable at a time: maternal age at delivery, maternal ethnicity/race, or infant sex.</p>
Derivation of Measure(s)	<p>Denominator is composed of all live-born infants in geographic region of interest during a calendar year.</p> <p>Numerator is composed of all live-born infants, fetal deaths (where available), and terminations (where available) with birth defect 'X' in the geographic region of interest during a calendar year.</p> <p>For states that ascertain fetal deaths and/or terminations, two sets of birth prevalence estimates are to be calculated for each birth defect—one including and one excluding fetal deaths and/or terminations.</p> <p>Diagnosis of cases may be made up to one year of age—ascertainment may be at any time.</p>
Unit	Defect present at birth
Geographic Scope	State and National (tracking network states)
Geographic Scale	State, county
Time Period	1998-current
Time Scale	Five year

<p>Rationale</p>	<p>Birth defects pose a significant public health problem. One in 33 babies is born with a structural birth defect in the United States. Birth defects are a leading cause of infant mortality; they are also responsible for considerable morbidity and disability with enormous economic and social costs. A lifetime of medical care and special education for a single child can cost more than \$500,000.</p> <p>Approximately 60% of birth defects are of unknown etiology. The ambient environment remains a source of great public concern, but few environmental exposures have been well-studied. Most birth defects likely will be explained by a complex interaction between genetic predispositions and environmental factors. However, before the ability to conduct studies to explore these interactions is achieved, linking birth defects–outcome data with environmental hazard or exposure data is critical. The first step in effecting successful linkages of these data is the existence of high-quality birth defects prevalence data for which the geospatial and temporal patterns and distributions can be monitored. The environmental public health tracking (EPHT) initiative is well-positioned to bring together birth prevalence data from its state partners to begin analyses of these patterns, which will provide important clues to public health officials and researchers.</p>
<p>Use of the Measure</p>	<p>The basic procedure for calculating birth prevalence is the same for all the suggested birth defects. Once the input data are appropriately prepared, birth prevalence will be calculable for all defects at the same time.</p> <p>State Allow for consistent and rapid method for calculating and displaying (using GIS) prevalence at selected geographical areas (i.e., county level).</p> <p>Allow for a better understanding of spatial and temporal patterns of selected birth defects.</p> <p>National Allow for comparison of birth prevalence across states, which can be used to target interventions. Any comparison of birth prevalence, however, will need to account for the variability in data collection methods between state surveillance systems. (See “Limitations of Data Sources” below and introductory text in appended team recommendations).</p> <p>Local Concerned community members will be able to view the tracking network Web page to see the birth prevalence of selected birth defects (while protecting confidentiality) at specified geographical areas. A</p>

	<p>public health message will help interpret the results and provide more information on selected birth defects and prevention measures (i.e., folic acid for prevention of neural tube defects, smoking and clefts, alcohol and fetal alcohol syndrome, and known teratogenic medications). A link to a list of known teratogens can be provided to users.</p>
Limitations of the Measure	<p>Ideally, incidence rates would be used instead of birth prevalence to measure birth defects occurrence. The numerator of the incidence would be the number of new cases of birth defect A in an area and time period and the denominator would be the number of conceptions at risk for developing birth defect A in that area and time period. Because both the number of conceptions and the number of cases “lost” through spontaneous abortions (as well as terminations and later fetal losses depending on the source of ascertainment for the specific surveillance system) is unknown, incidence cannot be calculated. Birth prevalence is the only appropriate measure that can be reported for birth defects occurrence.</p> <p>It is not feasible, at this time, to recommend that individual-level birth defects surveillance data be made available on even a secure national portal. Most states have strict guidelines with respect to confidentiality, and even the publication of birth prevalence data based on <5 cases in a geographic region is generally not done.</p>
Data Sources	<p>State birth defects surveillance systems: The data sources that contribute to birth defects surveillance systems include the following (this varies by system type):</p> <ul style="list-style-type: none"> • Vital records • Hospital records (discharge summaries or disease indices, nursery logs, NICU logs) • Administrative databases (Medicaid, state hospital discharge, HMO) • Specialty data sources (specialty clinics, programs for children with special health care needs) • Prenatal diagnostic centers or genetics clinics • Clinical examination • Local or national laboratories for cytogenetic testing <p>Denominator data will come from state vital records—number of live births, by year, by maternal age, and by race/ethnicity. These data may be aggregated and provided to the birth defects surveillance system for calculating birth prevalence, or it may be made available on an individual level to the birth defects surveillance system. This varies by state.</p>
Limitations of Data Sources	<p>All states in the US do not have a birth defects surveillance program. Among those that do, there is significant variability between surveillance systems. These include:</p>

	<ul style="list-style-type: none"> • Ascertainment method (active, passive, passive with follow-up/verification) <ul style="list-style-type: none"> ○ Primary differences are with data sources, coding, availability of verbatim description, and case verification • Ascertainment of spontaneous fetal deaths and variability in gestational age for inclusion. • Ascertainment of prenatally diagnosed cases and elective terminations • Case definitions • Classification as isolated, multiple, or syndromic <p>Data for specific birth defects may not be collected by each state or may only have been collected recently, limiting historical data for that birth defect.</p> <p>Address data tend to be based on address at delivery, not conception (more relevant time period for birth defects-related exposure).</p> <p>Approximately 50% of birth defects surveillance systems do not geocode their address data.</p>
--	---

CONTENT DOMAIN: CANCER

INDICATOR: INCIDENCE OF SELECTED CANCERS

Type of EPHT Indicator	Health Outcome
Measure	<ol style="list-style-type: none"> 1. Annual number of cases for selected cancers, by state 2. Annual age-adjusted incidence rate for selected cancers per 100,000 population or per 1,000,000 for childhood cancers (<15 & <20 years of age), by state 3. Average annual number of cases for selected cancers over five year period, by county 4. Age-adjusted incidence rate for selected cancers per 100,000 population over a five year period, by county <p>Measures for each of the selected cancer types are provided by sex and race/ethnicity groups. Some measures are also provided by age group as defined below.</p>
Derivation of Measure(s)	<p>Numerator is composed of counts of unique invasive primary incident cases of cancer “x” (bladder cancer also includes in situ) diagnosed during a specified calendar year or five year period within residents of a specified geographic region. Incident cancer data were originally collected by state and regional cancer registries. It is proposed that data for the National EPHT Network be obtained from the NCI and CDC joint venture, State Cancer Profiles.</p> <p>Denominator is composed of counts of the population residing in the geographic region of interest during a specified calendar year or five year period. Population data were originally collected by the U.S. Census. For these national cancer indicators, population data is obtained from the NCI and CDC’s State Cancer Profiles, which use U.S. Census data as modified by SEER.</p> <p>Rates will be age-adjusted to year 2000 U.S. standard population.</p> <p>Cancer types:</p> <p>Mesothelioma: SEER Recode B 36010. ICD-O-3 codes: histologies 9050-9055. Malignant cases: ICD behavior code ‘3’.</p> <p>Melanoma of the skin*: SEER Recode B 25010. ICD-O-3 codes: primary site C440-C449, histologies 8720-8790. Invasive melanoma (behavior code ‘3’).</p>

Liver & Intrahepatic Bile Duct: SEER Recode B 21071, 21072. ICD-O-3 codes: primary sites C220, C221; excludes histologies: 9590-9989, 9050-9055, and 9140. Malignant cases: ICD behavior code '3'.

Kidney & Renal Pelvis: SEER Recode B 29021, 29022. ICD-O-3 codes: C649, C659; excludes histologies: 9050-9055, 9140, 9590-9989. Malignant cases: ICD behavior code '3'.

Oral Cavity & Pharynx: SEER Recode B Site Groups 20010-20100 (20010, 20020, 20030, 20040, 20050, 20060, 20070, 20080, 20090, 20100). ICD-O-3 site codes: C000-C009, C019-C069, C079-C119, C129-C140, C142-C148; excludes histologies 9050-9055, 9140, 9590-9989.

Esophageal: SEER Recode B 21010. ICD-O-3 site codes: C150-C159; excluding histologies 9050-9055, 9140, 9590-9989.

Pancreas: SEER Recode B 21100. ICD-O-3 codes: C250-C259; excluding histologies 9050:9055, 9140, 9590:9989.

Larynx: SEER Recode B 22020. ICD-O-3 codes: C320-C329; excluding histologies 9050:9055, 9140, 9590:9989.

Lung & Bronchus: SEER Recode B 22030. ICD-O-3 Site codes C340-C349; excludes histologies 9050-9055, 9140, 9590-9989.

Breast (female):** SEER Recode B 26001. ICD-O-3 Site codes C500-C509; excludes histologies 9050-9055, 9140, 9590-9989.

Bladder: SEER Recode B 29010. ICD-O-3 Site codes C670-C679; excludes histologies 9050-9055, 9140, 9590-9989. [includes invasive and in-situ]

Brain & ONS*:** SEER Recode B 31010, 31040. ICD-O-3 Site codes C700-C709, C710-C719, C720-C729; excludes histologies 9050-9055, 9140, 9590-9989.

Thyroid: SEER Recode B 32010. ICD-O-3 Site codes C739; excludes histologies 9050-9055, 9140, 9590-9989.

Non-Hodgkin Lymphoma: SEER Recode B 33041, 33042. ICD-O-3 codes: histology 9590-9596, 9670-9671, 9673, 9675, 9678-9680, 9684, 9687, 9689-9691, 9695, 9698-9702, 9705, 9708-9709, 9714-9719, 9727-9729; histology 9823 or 9827 in all sites except C420, C421, C424.

	<p>Leukemia: SEER Recode B 35011, 35012, 35013, 35021, 35022, 35023, 35031, 35041, 35043. ICD-O-3 codes: <u>ALL</u> – histology 9826,9835-9837; <u>Other lymphocytic</u> – histology 9820, 9832-9834, 9940; <u>Acute monocytic</u> – histology 9891; <u>CML</u> – histology 9863, 9875, 9876, 9945, 9946; <u>Other</u> – histology 9860, 9930, 9801, 9805, 9931, 9733, 9742, 9800, 9831, 9870, 9948, 9963, 9964. Site codes C420, C421, C424 – histology 9827. (Also include codes for CLL and AML.)</p> <p>Chronic Lymphocytic Leukemia (CLL): SEER Recode B 35012. ICD-O-3 codes: C420, C421, C424 with histology 9823.</p> <p>Acute Myeloid Leukemia (AML): SEER Recode B 35021. ICD-O-3 codes: histology 9840, 9861, 9866, 9867, 9871-9874, 9895-9897, 9910, 9920.</p> <p>Child cancers: SEER ICC3 childhood cancer codes http://seer.cancer.gov/iccc/iccc3.html</p> <p>NOTE: SEER Recode B (Dec 2003) http://seer.cancer.gov/siterecode_b/icdo3_d12192003/ Tobacco-related cancers: consistent with SEER Recode B, CWG Cancer Team NCDM specifies Histology Exclusions 9050-9055 (Mesothelioma), 9140 (Kaposi Sarcoma), 9590-9989 (Lymphoma, Leukemia, Miscellaneous). * Grantee portals may choose to additionally display In-situ cases, both disaggregated and aggregated with invasive cases (“All combined”). ** Breast – Malignant/invasive only: The NEPHTN Metadata state “Counts and rates for in situ breast cancer cases among women are presented; these are reported separately and are not included in counts or rates for the "All Sites" category.” (CDC-EHTB plans to delete this sentence from national portal Metadata.) The NCDM states “Numerator is composed of counts of unique invasive primary incident cases of cancer ...” (in “Derivation of Measure”). Grantee portals may choose to additionally display In-situ cases, both disaggregated and aggregated with invasive cases (“All combined”). *** Brain/ONS – Malignant/invasive only: The NEPHTN Metadata state “Incidence data on nonmalignant primary brain and central nervous system (CNS) tumors are available on this Web site.” (CDC-EHTB plans to delete this sentence from national portal Metadata.) The NCDM states “Numerator is composed of counts of unique invasive primary incident cases of cancer ...” (in “Derivation of Measure”).</p>
Unit	Newly reported cancer case
Geographic Scope	State and national (tracking network states)
Geographic Scale	State and county.
Time Period	2000-current
Time Scale	Annual and 5 year period
Rationale	Approximately 1.4 million Americans are expected to be diagnosed with cancer during 2007. The National Cancer Institute (NCI) estimated that in January 2003, there were approximately 10.3 million living Americans with a history of cancer. The risk of being

diagnosed with cancer increases as a person ages, and 77 % of all cancers are diagnosed in Americans age 55 years or older. Cancer, a diverse group of diseases characterized by the uncontrolled growth and spread of abnormal cells, is believed to be caused by both external and internal risk factors.

Major risk factors for cancer include tobacco use, diet, exercise, and sun exposure (Clapp, Howe, Jacobs). For example, male smokers are about 23 times more likely to develop lung cancer than male non-smokers. Researchers have also identified genetic risks for cancer. Female first degree relatives (mother, sisters, and daughters) of women with breast cancer are about twice as likely to develop breast cancer as women who do not have a family history of breast cancer (*Cancer Facts and Figures, 2007*; ACS, 2007).

However, the etiology of many cancer types is not well established. The physical environment (e.g., air quality, chemical pollution, and water quality) remains a source of great public concern but few community-level environmental exposures have been well-studied. Studies of occupational cohorts have identified numerous suggestive epidemiological associations between certain occupational exposures and elevated cancer rates. After reviewing the evidence regarding the causes of cancer in the United States, Doll and Peto published a seminal article in 1981 estimating that 35% of all U.S. cancer deaths were attributable to diet, 30% to smoking, 4% to occupation, and 2% to pollution. While some authors have agreed with Doll and Peto (Ames and Gold 1998), and others have cautioned against their approach: “there is substantial evidence that occupational and environmental exposures contribute to the burden of cancer” (Clapp, Howe, and Jacobs 2006).

One way to assess cancer burden is to study geographic variation. In recent years, geographic information systems (GIS) have become an important tool for health and environmental research. GIS can extend the analysis of data beyond simple mapping by enabling the linkage, visualization, and analysis of multiple layers of health and environmental data from both spatial and temporal perspectives.

One important use of geographic analysis of health data is in the analysis of regional variations in cancer mortality and incidence. The National Cancer Institute’s *Atlas of Cancer Mortality for U.S. Counties: 1950–1969* (Mason et al. 1975), represented the first effort to map cancer mortality data at the county level throughout the United States. In 1999, the national level analysis of cancer mortality was updated by the NCI (*Atlas of Cancer Mortality in the United States, 1950–94*, Devesa et al. 1999). More recently, multiple Web-

	<p>based data query systems have made U.S. cancer incidence and mortality datasets and or maps available at the county (NCI/CDC State Cancer Profiles: http://statecancerprofiles.cancer.gov/; NCI SEER data: http://seer.cancer.gov/data/; NJ DHSS cancer online: http://www.cancer-rates.info/nj/) and/or state level (NAACCR CINA+ Online: http://www.cancer-rates.info/naacccr/ ; CDC U.S. Cancer Statistics: http://apps.nccd.cdc.gov/uscs/).</p>
<p>Use of the Measure</p>	<p>At the local and state levels, the EPHT Network will:</p> <p>Allow interested persons to obtain information on environmental exposures (air pollution and drinking water quality) and cancer or other health outcomes (birth defects, asthma, and birth weight) for a selected geographic area and time interval. Standard suppression rules will be used to prevent the release of information that might reveal the identity of any person diagnosed with cancer. Public health messages will help interpret the results and provide linkages to additional information on cancer prevention, cancer etiology, and cancer treatment options. While many of these diverse health and environmental datasets are already available to the public, they are not currently available through “one-stop-shopping” via the Internet.</p> <p>Improve access to metadata regarding multiple health outcome datasets and environmental exposure datasets for public health practitioners and researchers. Enhanced access will provide better understanding of the strengths and limitations of the available datasets and may increase the use of the collected data.</p> <p>Allow for a better understanding of spatial and temporal patterns of selected cancers suggested to be linked to environmental exposures within states.</p> <p>At the national level, the EPHT Network will:</p> <p>Enhance the opportunity for multi-state epidemiological research by improving access to cancer incidence rates and environmental exposure information. This could be particularly helpful for uncommon cancer types or sub-types whereby incidence is too small for meaningful ecological studies in individual states.</p>
<p>Limitations of the Measure</p>	<p>Counts and rates will be calculated based upon residential address at time of diagnosis. No information is available on prior residences.</p> <p>Geocoding accuracy, level of geocoding, and geocoding completeness may vary by time and space. This could potentially create geographically non-random errors in calculated rates of cancer.</p> <p>No personal exposure information will be available, including smoking history, diet, lifestyle, or history of cancer.</p>

	<p>Data that will reveal the identity of any individual diagnosed with cancer can not be released. Suppression rules will govern the release of small case counts.</p> <p>No information will be available on the latency of cancer cases.</p>
Data Sources	National Cancer Institute, Surveillance Epidemiology and End Results; CDC National Program of Cancer Registries
Strengths and Limitations of Data Sources	<p>All of the 16 states and the 1 city participating in the EPHT Network are working with their state and/or regional cancer registry program(s). Registry training, data collection, data coding, data cleaning, and quality control programs are highly standardized and subject to annual evaluation. Documentation is available online from the North American Association of Centralized Cancer Registries (NAACCR). http://www.naacr.org/index.asp?Col_SectionKey=7&Col_ContentID=135.</p> <p>State cancer registry programs may vary, however, regarding the availability and quality of residential address information collected and completeness of geocoding efforts.</p>

**CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: HOSPITALIZATIONS FOR CARBON MONOXIDE
POISONING**

Type of EPHT Indicator	Health Outcome/Exposure
Measures	<ol style="list-style-type: none"> 1. Number of hospitalizations for carbon monoxide (CO) poisoning 2. Crude rate of hospitalization for CO poisoning per 100,000 population 3. Age-adjusted rate of hospitalization for CO poisoning per 100,000 population
Derivation of measure	<p>Numerator: Resident hospitalizations for CO poisoning that meet the 1998 CSTE case definition for public health surveillance for a “Confirmed” or “Probable” case of acute CO poisoning in administrative data sets.</p> <p>Frequencies for three unique groups:</p> <ol style="list-style-type: none"> 1. Unintentional, non-fire related 2. Unintentional, fire-related 3. Unknown intent <p>Denominator: Midyear resident population</p> <p>Adjustment: Age-adjustment by the direct method to year 2000 US Standard Population</p>
Unit	Hospital admission (categorized by discharge diagnosis)
Geographic Scope	State and national (tracking network states)
Geographic Scale	State; county when feasible
Time Period	2000-current
Time Scale	Calendar year

<p>Rationale</p>	<p>Carbon monoxide (CO) is an odorless, colorless gas that usually remains undetectable until exposure results in injury or death. Each year in the United States, an estimated 10,000 persons seek medical attention or lose at least one day of normal activity because of CO intoxication. There is limited information on CO hospitalization. In Florida, 1,494 were hospitalized with a diagnosis of CO poisoning from 1999–2007. Out of which 10% (n=143) were unintentional fire-related, 33% (n=493) were unintentional non-fire-related, and 17% (n=256) were from unknown cause of CO poisoning. During 2000–2009, a total of 68,316 CO exposures were reported to poison centers across United States.</p> <p>Persons hospitalized with CO poisoning are among the most severely poisoned cases. Unintentional CO poisoning is almost entirely preventable. These data are available in most states.</p>
<p>Use of the Measure</p>	<p>These data can be used to assess the burden of severe CO poisoning, monitor trends over time, identify high-risk groups, and enhance prevention, education, and evaluation efforts.</p>
<p>Limitations of the Measure</p>	<p>Hospitalization data, by definition, do not include: persons treated in outpatient settings (e.g., emergency departments, urgent care clinics, clinicians’ offices or hyperbaric chambers but not hospitalized); persons who call poison control centers and are managed at the scene, and/or receive medical care but are not hospitalized; persons who do not seek any medical care; or persons who die immediately from CO exposure without medical care.</p>
<p>Data Sources</p>	<p>Numerator: State inpatient hospital discharge data Denominator: U.S. Census Bureau population data</p>
<p>Limitations of the Data Source</p>	<p>The use and quality of ICD9-CM coding varies across jurisdictions; this is especially true of the codes used to describe how an injury occurs, indicated as E-codes. Examples of this variation include:</p> <ul style="list-style-type: none"> • The number of diagnostic fields available to specify cause of the injury; • Whether E-codes are mandated; • The completeness and quality of E-coding; for example, the reliability of ICD-9-CM coding to distinguish between cases of CO poisoning that are intentional or unintentional, and/or fire-or non-fire related <p>The toxic effects of CO exposure are nonspecific and easily misdiagnosed when CO exposure is not suspected. These misdiagnosed cases will not be counted.</p> <p>These data usually do not include data from federal facilities such as Veteran’s Administration hospitals.</p>

	<p>These data usually include only cases of state residents treated within the state. Health-care access is not restricted to these political boundaries so patients hospitalized for CO poisoning in another state may not be counted in their own state. Likewise, they may not be counted in the jurisdiction in which they were treated. Currently, few states have access to, or agreements to obtain, hospital discharge data from other states where their state residents may be hospitalized. To the extent that patients are treated out of state, there is undercounting of the rate of state residents poisoned by CO.</p> <p>Differences in rates between jurisdictions may reflect differences in hospital admissions practices for treating persons with severe CO poisoning. For example, some facilities may routinely admit all patients treated with hyperbaric oxygen; other facilities may release patients treated with hyperbaric oxygen after the treatment is completed if they are in stable condition.</p> <p>Race and ethnicity are important risk factors for CO poisoning, yet, many hospitalization data sets do not contain these data. Those that do may have data quality issues.</p> <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census Bureau. These need to be extrapolated or purchased from a vendor.
References	<ol style="list-style-type: none"> 1. Centers for Disease Control and Prevention, Perspectives in Disease Prevention and Health Promotion Carbon Monoxide Intoxication—A Preventable Environmental Health Hazard MMWR, 1982. 31(39): p. 529–31. 2. Centers for Disease Control Prevention, Carbon monoxide exposures—United States, 2000–2009. MMWR, 2011. 60(30): p. 1014–7. 3. Harduar-Morano, L. and S. Watkins, Review of unintentional non-fire-related carbon monoxide poisoning morbidity and mortality in Florida, 1999–2007. Public Health Rep, 2011. 126(2): p. 240–50. 4. King, M.E. and S.A. Damon, Attitudes about carbon monoxide safety in the United States: results from the 2005 and 2006 Health Styles Survey. Public Health Rep, 2011. 126 Suppl 1: p. 100–7.

CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: EMERGENCY DEPARTMENT VISITS FOR CARBON MONOXIDE POISONING

Type of EPHT Indicator	Health Outcome
Measures	<ol style="list-style-type: none"> 1. Number of emergency department (ED) visits for CO poisoning 2. Crude rate of ED visits for CO poisoning per 100,000 population 3. Age-adjusted rate of ED visits for CO poisoning per 100,000 population
Derivation of measure	<p>Numerator: Resident emergency department visits for CO poisoning that meet the 1998 CSTE case definition for public health surveillance for a “Confirmed” or “Probable” case of acute CO poisoning in administrative data sets.</p> <p>Frequencies for three unique groups:</p> <ol style="list-style-type: none"> 1. Unintentional, non-fire related 2. Unintentional, fire-related 3. Unknown intent <p>Denominator: Midyear resident population <i>Adjustment:</i> Age-adjustment by the direct method to year 2000 US Standard Population</p>
Unit	Emergency department visit
Geographic Scope	State and national (tracking network states)
Geographic Scale	State
Time Period	2000-current
Time Scale	Calendar year

<p>Rationale</p>	<p>Carbon Monoxide (CO) poisoning is preventable; nonetheless, unintentional, non-fire-related CO poisoning is responsible for approximately 15,000 emergency department visits and nearly 500 deaths annually in the United States. During 2004–2006, an estimated average of 20,636 ED visits for nonfatal, unintentional, non-fire-related CO exposures occurred each year. Approximately 73% of these exposures occurred in homes, and 41% occurred during winter months (December–February). Prevention efforts targeting residential and seasonal CO exposures can substantially reduce CO-related morbidity. During 2000–2009, a total of 68,316 CO exposures were reported to poison centers across United States.</p> <p>Persons admitted to emergency departments and diagnosed with CO poisoning range from suspected exposure to severe poisonings that may result in treatment and release, hospitalization, or death. Emergency department visits represent patients not counted in other clinical settings. Unintentional CO poisoning is usually preventable. Emergency department data are available in more than 50% of the states and that number is increasing.</p>
<p>Use of the Measure</p>	<p>These data can be used to assess the burden of CO poisoning and to monitor trends over time as well as to identify high risk groups, and enhance prevention, education, and evaluation efforts.</p>
<p>Limitations of the Measure</p>	<p>Measures based on emergency department data alone may underestimate its prevalence because these data may not include persons that are managed at the scene, persons who do not seek any medical care, persons admitted without first visiting an emergency department, or persons who die immediately from CO exposure without medical care.</p>
<p>Data sources</p>	<p>Numerator: State emergency department visit data</p> <p>Denominator: U.S. Census Bureau population data</p>
<p>Limitations of the Data Source</p>	<p>Emergency department data have limitations for comparisons across jurisdictions because the use and quality of ICD-9-CM coding may vary across jurisdictions; this is especially true of the codes used to describe how an injury occurs, indicated as E-codes. Examples of this variation include:</p> <ul style="list-style-type: none"> • The number of diagnostic fields available to specify cause of the injury vary from nine to unlimited (in some states reaching more than 100); • E-codes are mandated in some jurisdiction but not in others;

	<ul style="list-style-type: none"> • The completeness and quality of E-coding vary by hospital as well as jurisdiction. In addition, the reliability of ICD-9-CM coding to distinguish between cases that are intentional or unintentional, fire-related, or of unknown intent is undocumented; • States are inconsistent in the use of intent codes. <p>The toxic effects of CO exposure are nonspecific and easily misdiagnosed when CO exposure is not suspected. These misdiagnosed cases will not be counted.</p> <p>These data usually do not include data from federal facilities such as Veteran's Administration hospitals.</p> <p>These data usually include only cases of state residents who were treated within the state. Health care access is not restricted to these political boundaries so people discharged from the emergency department for CO poisoning in another state will neither be counted in their own state nor in the jurisdiction in which they were treated. Currently, few states have access to, or agreements to obtain, their emergency department data from other states in which their residents may have received treatment. To the extent that patients are treated out of state, there is undercounting of the rate of residents poisoned by CO.</p> <p>Regional variation between emergency departments in diagnosing CO poisoning may exist.</p> <p>Many emergency department visit data sets do not contain race or ethnicity information and those that do may have data quality issues. Yet, these characteristics are known risk factors for CO poisoning.</p> <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census Bureau. These need to be extrapolated or purchased from a vendor.
References	<ol style="list-style-type: none"> 1. Centers for Disease Control and Prevention. Perspectives in Disease Prevention and Health Promotion Carbon Monoxide Intoxication—A Preventable Environmental Health Hazard MMWR Morb Mortal Wkly Rep 1982;31(39):529–31. 2. Centers for Disease Control Prevention. Nonfatal, unintentional,

	<p>non-fire-related carbon monoxide exposures—United States, 2004-2006. <i>MMWR Morb Mortal Wkly Rep</i> 2008;57(33):896–9.</p> <ol style="list-style-type: none"><li data-bbox="597 302 1487 407">3. Hampson NB. Emergency department visits for carbon monoxide poisoning in the Pacific Northwest. <i>J Emerg Med</i> 1998;16(5):695–8.<li data-bbox="597 428 1487 491">4. Kao LW, Nanagas KA. Carbon monoxide poisoning. <i>Emerg Med Clin North Am</i> 2004;22(4):985–1018.<li data-bbox="597 512 1487 617">5. Partrick M, Fiessler F, Shih R, Riggs R, Hung O. Monthly variations in the diagnosis of carbon monoxide exposures in the emergency department. <i>Undersea Hyperb Med</i> 2009;36(3):161–7.
--	---

**CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: CARBON MONOXIDE POISONING MORTALITY**

Type of EPHT Indicator	Health Outcome
Measures	<ol style="list-style-type: none"> 1. Number of deaths from CO poisoning 2. Crude rate of death from CO poisoning per 100,000 population 3. Age-adjusted rate of death from CO poisoning per 100,000 population
Derivation of measure	<p>Numerator: Resident deaths from CO poisoning for three unique groups:</p> <ol style="list-style-type: none"> 1. Unintentional, non-fire related 2. Unintentional, fire-related 3. Unknown intent <p>Denominator: Midyear resident population</p> <p>Adjustment: Rates age-adjusted by the direct method to the Year 2000 U.S. Standard Population</p>
Unit	Deaths due to CO poisoning
Geographic Scope	State and National
Geographic Scale	State
Time Period	2000-current
Time Scale	Calendar year

<p>Rationale</p>	<p>CO is an odorless, colorless gas that usually remains undetectable until exposure results in injury or death. Carbon monoxide (CO) poisoning is a leading cause of unintentional poisoning deaths in the United States. CO poisoning is preventable; nonetheless, unintentional, non–fire-related CO poisoning is responsible for approximately 15,000 emergency department visits and nearly 500 deaths annually in the United States. During 1999–2004, CO poisoning was listed as a contributing cause of death on 16,447 death certificates in the United States and 2,631 (16%) were classified as both unintentional and non-fire-related deaths. The annual average age-adjusted death rate in the U.S. was 1.5 deaths per million persons. The US Consumer Product Safety Commission’s historical data indicate that there is a statistically significant increasing trend in non-fire CO fatalities from 1999 through 2007. In 2007, 183 unintentional consumer product–related, non–fire-related CO deaths were reported. Out of which heating systems were associated with the largest percentage of non-fire CO poisoning fatalities at 38 percent (estimated 70 deaths); Engine-Driven Tools-related CO fatalities were also associated with 38 percent (69 deaths), and the remaining six product categories [Charcoal Grills or Charcoal (7 deaths); Ranges, Ovens (7 deaths); Water Heaters (3 deaths); Grills, Camp Stoves (3 deaths); Other Products (1 death); and Multiple Products (24 deaths)] combined were associated with a total of 25 percent.</p> <p>Death is the most severe outcome of CO poisoning. Unintentional CO poisoning deaths are almost entirely preventable. Most localities have access to data on their resident deaths.</p>
<p>Use of the Measure</p>	<p>These data can be used to assess the burden of severe CO poisoning, monitor trends over time, and enhance prevention, education, and evaluation efforts.</p>
<p>Limitations of the Measure</p>	<p>This measure understates the burden of CO poisoning because most cases do not result in death. Rates can be misleading (i.e., do not reflect risk of occurrence) if a relatively large proportion of deaths occur to non-residents poisoned within the jurisdiction (they are excluded from the rate calculation). Death investigation laws vary by locale.</p>
<p>Data Sources</p>	<p>Numerator: Death certificate records from vital statistics agency</p> <p>Denominator: Population counts or estimates from the U.S. Bureau of the Census</p>

<p>Limitations of the Data Source</p>	<p>Death investigation laws vary by locale. In addition, variations may occur between localities in how medical examiners/coroners/physicians assign intentionality. Thus an area where the ME/coroner/physician is disinclined to attribute a CO poisoning to suicide will have a higher unintentional CO poisoning death rate than a comparable locale. Finally, CO poisonings that are unrecognized by the ME/coroner/physician will be attributed to other causes.</p>
<p>References</p>	<ol style="list-style-type: none"> 1. Centers for Disease Control Prevention, Carbon monoxide--related deaths--United States, 1999-2004. MMWR Morb Mortal Wkly Rep, 2007. 56(50): p. 1309-12. 2. Centers for Disease Control Prevention, Unintentional non-fire-related carbon monoxide exposures--United States, 2001-2003. MMWR Morb Mortal Wkly Rep, 2005. 54(2): p. 36-9. 3. Mott, J.A., et al., National vehicle emissions policies and practices and declining US carbon monoxide-related mortality. JAMA, 2002. 288(8): p. 988-95. 4. Hnatov, MV. Non-Fire Carbon Monoxide Deaths Associated with the Use of Consumer Products 2007 Annual Estimates. Bethesda, MD: US Consumer Product Safety Commission. Available at: http://www.cpsc.gov/library/foia/foia11/os/co10.pdf. Accessed July 18, 2012

CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: REPORTED EXPOSURE TO CARBON MONOXIDE

Type of Indicator	Exposure, Health Outcome
Measures	<ol style="list-style-type: none"> 1. Number of unintentional CO exposures reported to poison control centers by resulting health effect and treatment in a healthcare facility 2. Crude rate of unintentional CO exposures reported to poison control centers per 100,000 population by resulting health effect and treatment in a healthcare facility
Derivation of measures	<p>Number of reported cases of unintentional carbon monoxide exposure stratified by presence of subsequent health effect and consequential treatment in a healthcare facility</p> <p>Denominator used is Midyear resident population</p>
Unit	Reported exposure to CO
Geographic Scope	State and national (tracking network states)
Geographic Scale	County
Time Period	2000- current
Time scale	Annual
Rationale	<p>PCCs serve the public and healthcare providers in the management of actual or potential exposure to hazardous substances, including CO. PCC calls are fielded by certified specialists in poisoning information (SPIs), and recorded in a standard electronic format. Regional PCC data are centralized nationally by AAPCC annually.</p> <p>PCC calls provide information about CO exposure that may not otherwise be captured in hospital discharge data or emergency department data. These include events where CO exposure was detected but did not result in symptoms, where symptoms were mild and did not require follow-up in a health care facility, and where the event resulted in symptoms but the patient refused to seek medical treatment. Two state-based evaluations (Connecticut [1] and Wisconsin [2]) found minimal overlap between persons using PCCs</p>

	<p>and persons treated in emergency departments. As such, tracking of PCC calls in addition to indicators of mortality, hospitalizations, and emergency room visits provides a more complete picture of the public health burden of CO exposure.</p>
Use of the Measure	<p>These data may be used to estimate the population's exposure to CO and to monitor trends over time. They may also be used to estimate symptomatic CO exposures among exposed persons who may not be treated in a health care facility and therefore would not be captured in other health outcome datasets.</p>
Limitations of the Measure	<p>Exposure status should not be considered confirmed. In some cases, ambient air sampling results or the patient's lab results may be reported in the case notes but only when this information is available or provided to the SPI. In addition, it should be noted that because they may contain identifiable and sensitive information, SPI notes are removed from case records by regional PCCs before submitting to the AAPCC and are therefore unavailable at the national level.</p> <p>Not all potentially hazardous CO exposures will be captured by PCC calls. For example, cases of moderately elevated exposure in the home are unlikely to be recognized if there are no acute symptoms and a CO alarm is not installed. Moreover, knowledge, attitudes, and practices around the use of PCCs likely vary both within and across jurisdictions. In the event of suspected exposure, callers may first notify their local fire department or call 911 or even their utility provider; in either case, the regional PCC may not be simultaneously notified. Practices by health care providers that use PCCs are also likely to vary from one jurisdiction to another. Generally speaking, healthcare providers use the PCC as a resource in the diagnosis and treatment of poisonings; in addition, in New York City, where CO poisoning was designated as an immediately reportable condition in 2004, the PCC plays an integral role in the management of reports from healthcare providers and in the rapid referral of the fire department for investigation at the site of exposure for the prevention of secondary cases (3). For these reasons, caution should be exercised in comparing rates of reported exposure across states.</p>
Data Sources	<p>Numerator: PCC calls (usually in standard Toxicall database)</p> <p>Denominator: U.S. Census Bureau population data</p>
Limitations of the Data Sources	<p>SPIs are not required to collect patient state/ZIP code unless the patient is the caller. Using caller state/ZIP code to determine</p>

	<p>residency may cause the number of calls pertaining to state residents to be overestimated—for example, when the caller is an out-of-state health care provider.</p> <p>The number of cases may differ slightly between datasets obtained directly from the state’s PCC and the national AAPCC dataset for that state; this is typically due to calls that are re-routed to another state when the state’s PCC is overloaded. The AAPCC national dataset is corrected for such instances.</p> <p>Age adjustment is not recommended since age is often estimated (such as "Adult > 19" or “50s”).</p>
<p>References</p>	<ol style="list-style-type: none"> 1. Toal B. Comparison of Three CO Databases in Connecticut [PowerPoint presentation]. EPHT Web Seminar; 2006 June. 2. Bekkedal M, Sipsma K, Stremski ES, Malecki KC, Anderson HA. Evaluation of five data sources for inclusion in a statewide tracking system for accidental carbon monoxide poisonings. WMJ. 2006 Mar;105(2):36-40. 3. Wheeler K, Kass D, Hoffman R, Vecchi M, Allocca A. Preventing CO poisoning: tracking the impact of legislative and regulatory changes in New York City [PowerPoint Presentation]. Annual Meeting of the Council of State and Territorial Epidemiologists; 2006 June.

**CONTENT DOMAIN: CARBON MONOXIDE
INDICATOR: HOME CARBON MONOXIDE DETECTOR
COVERAGE**

Type of Indicator	Intervention
Measure	Percent of Behavioral Risk Factor Surveillance System (BRFSS) respondents reporting at least one CO detector in their household
Derivation of Measure	<p>Numerator: The number of respondents reporting CO detector in household</p> <p>Denominator: The number of respondents reporting CO detector in household plus respondents reporting no CO detector in household</p> <p>Proportion is adjusted using the survey's household weight</p>
Unit	CO detector presence
Geographic Scope	State and national (tracking network states)
Geographic Scale	State
Time Period	2004; States' BRFSS surveys should include this question every 3–5 years and/or when implementing interventions, such as new legislation, to increase the use of CO alarms
Time Scale	Annual
Rationale	Correctly installed and maintained CO detectors can prevent injury and death from exposure to CO.
Use of the Measure	<p>Collected data will determine the occurrence of CO detectors in homes. These data also can be combined with other data collected by the BRFSS survey, including respondent demographics (e.g., age, sex, and race of survey respondents and age and sex composition of household), socioeconomic characteristics (e.g., insurance status), and relevant health and prevention risk factors (e.g., smoking status, presence of fire alarms). The results of these analyses can be used to target and evaluate public health prevention strategies.</p> <p>Notes about conducting the analysis:</p>

	<p>BRFSS data should be analyzed by experts in analysis of sample survey data and the software available to conduct this type of analysis (e.g., SUDAAN and SAS survey procedures).</p> <p>The BRFSS survey is designed so that the primary sampling unit is the respondent. As such, BRFSS data are typically directly weighted to account for sampling error based on data collected at the individual level. However, the question about CO detectors is based on the household rather than the individual as the sampling unit. Using the weighting designed for individuals may bias the prevalence estimate of household risk factors. The indicator will therefore use a weight based on the potential error associated with sampling the household rather than the individual.</p>
<p>Limitations of the Measure</p>	<p>Carbon monoxide alarms must be properly installed and maintained to be effective; a single question does not capture information about either. Maine has developed two questions that can be asked to get supplemental information on maintenance:</p> <ol style="list-style-type: none"> 1. Is your carbon monoxide detector battery powered or have a battery for back-up power? <p><u>Response categories</u>: Yes; No; Don't Know; Refused</p> <ol style="list-style-type: none"> 2. When was the last time you checked the batteries? <p><u>Response categories</u> (Read only if needed): Within the past year; More than a year; Don't know/Not sure; Refused</p>
<p>Data Sources</p>	<p>BRFSS state-added question from the Indoor Air Pollution Module, question number 4:</p> <p><i>A carbon monoxide or CO detector checks the level of carbon monoxide in your home. It is not a smoke detector. Do you have a carbon monoxide detector in your home?</i></p>
<p>Limitations of the Data Resources</p>	<p>While the data collection methods are standardized to allow comparisons between states, there may still be bias introduced by “house-effects”—that is, the variation introduced by different organizations and individuals implementing the survey for different states.</p> <p>The BRFSS questionnaire is available in English or Spanish language versions; persons who are not conversationally fluent in English (or Spanish in the states that offer the Spanish-language option) are not eligible. This population of non-English speakers may differ systematically from English speakers in health and behavior</p>

	<p>characteristics, including the presence of a CO detector in their homes.</p> <p>The BRFSS is a telephone survey. While the effect of telephone non-coverage on estimates derived from BRFSS is small, the population without telephones is not likely representative of the general population. In particular, this population is less likely to have a CO detector in the household; therefore, these results should not be generalized to populations without telephone coverage.</p> <p>An increasing number of households use telephone technology that may result in changes in the population sampled and therefore may make the survey results less reliably generalized and introduce other bias. Two examples are:</p> <ol style="list-style-type: none">1. Households with cellular telephones and no traditional telephone. These households are not in the sampling frame for the BRFSS2. Households that use Caller ID to screen calls; their members may be less likely to pick up the call. <p>Surveys based on self-reported information are likely less accurate than those based on physical measurements. However, when measuring change over time, this type of bias is likely to be constant and therefore not a factor in trend analysis.</p>
--	---

**CONTENT DOMAIN: CHILDHOOD LEAD POISONING
INDICATOR: TESTING COVERAGE AND HOUSING AGE**

Type of EPHT Indicator	Hazard /Intervention
Measures	<ol style="list-style-type: none"> 1. Number of children born in the same year and tested for lead before age 3 2. Percent of children born in the same year and tested before age 3 3. Number of homes built before 1950 (as measured in the 2000 Census) 4. Percent of homes built before 1950 (as measured in the 2000 Census)
Derivation of Measure(s)	<p>Use birth year cohort to calculate the percentage of children with at least one test prior to age 36 months.</p> <p>Use 2000 Census, Summary file 3, to calculate the percentage of pre-1950 housing units</p>
Unit	Proportion of houses by age-based hazard assessment
Geographic Scope	State and national
Geographic Scale	county and state
Time Period	2000-
Time Scale	annual; birth cohort
Rationale	<p>Elevated BLLs in young children have been associated with adverse health effects ranging from learning impairment and behavioral problems to death. Because children may have elevated BLLs and not have any specific symptoms, CDC recommends a blood-lead test for young children at risk for lead poisoning. Risk factors identified in the National Health and Nutrition Examination Surveys (NHANES) include living in housing built before 1950, especially deteriorating condition, being African American and living in a family in poverty.</p> <p>Many states have adopted a targeted testing strategy (test children at high risk), and some states recommend universal testing (test all young children). Nevertheless, studies have documented low blood-lead testing rates among children at high risk. CDC recommends that state and local childhood lead poisoning prevention programs (CLPPPs) evaluate testing among high-risk populations. All CLPPPs have assessed testing in their states but many methods have been used and it is not possible to compare across states.</p> <p>CLPPPs also administer education campaigns for physicians and parents about childhood lead poisoning to enable them to identify</p>

	<p>children at risk.</p> <p>For both universal testing plans and targeted testing plans, children should be tested at least once before the age of 3 years. Some states require more than one test between the ages of 6 and 36 months. Using a birth cohort, the number of children born in a specific year tested before the age of 36 months can be determined.</p>
<p>Use of the Measure</p>	<p>State Identify populations that are not being tested adequately and improve testing</p> <p>Allow for a better understanding of what the blood-lead surveillance data represent</p> <p>National Allow for comparison across states; such comparison can be used to target interventions (especially CDC, EPA, HUD)</p> <p>Public/parents Determine if their community is at risk and the percentage of children being tested. There will be a public health message which will help interpret the results and provide more information on lead sources and prevention.</p> <p>Health care providers Identify children who should be tested for lead by identifying high-risk communities</p>
<p>Limitations of the Measure</p>	<p>This measure estimates testing rates in children living in communities which may be at greater risk of exposure due to older housing. It is a surrogate for a child's risk of lead poisoning due to lead paint in the home. A more direct measure would be based on individual children and the actual age of their housing.</p> <p>Some tested children's addresses are not in the CLPPP data system, while only the provider's address is provided for other children. This can result in some tests being attributed to the wrong county or not being counted at all.</p> <p>Counties are not homogenous with respect to the distribution of lead hazards or risk factors for lead exposure.</p> <p>Using number of pre-1950s housing from Census does not account for houses that have been renovated or have had lead removed.</p> <p>This measure does not account for other lead sources in the community.</p>

	<p>Children may be exposed to lead paint in neighboring counties (visiting family, day care)</p> <p>Many states require children be tested more than once. This indicator does not determine how many children are tested more than once to meet such state requirements.</p>
Data Sources	<ul style="list-style-type: none"> • Childhood Blood Lead Surveillance Data • US Census (Summary file 3) for total number of housing units and number of pre-1950 units • Vital statistics birth data for number of births
Limitations of Data Sources	<p>Childhood Blood Lead Surveillance Data</p> <ul style="list-style-type: none"> • Surveillance data are not randomly sampled or representative of the population. • Addresses for all children tested are not included. • Address of the treating clinic is listed sometimes as the address of the child. • De-duplication by a standardized method will be required • Race and ethnicity are not always captured. <p>Census data</p> <ul style="list-style-type: none"> • Data are available only every 10 years. • Does not have information on renovation of pre 1950 housing is not available. • Does not have information on the condition of the housing is not available. • Address level information on the year the housing was built is not available. <p>Vital Statistics Birth Data</p> <ul style="list-style-type: none"> • Children may move to another county after birth

**CONTENT DOMAIN: CHILDHOOD LEAD POISONING
INDICATOR: BLOOD LEAD LEVELS BY BIRTH COHORT**

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Exposure
Measure(s)	<ol style="list-style-type: none"> 1. Number of children born in the same year and tested , by county and state 2. Percent of children born in the same year and tested, by county and state 3. Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by county and state 4. Percent of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by county and state 5. Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by blood lead level category³, by state 6. Percent of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by blood lead level category³, by state <p>¹ The current blood lead reference level is $5 \mu\text{g/dL}$ based on National Health and Nutrition Examination Survey (NHANES) 2007 – 2008 and 2009 – 2010 data published in the Fourth National Report on Human Exposure to Environmental Chemicals, and updated in 2012. Blood Lead Levels (BLLs) are confirmed if there is either: (1) one elevated venous test or (2) two elevated capillary and/or unknown tests at least 1 day but less than 12 weeks apart.</p> <p>²Details about selecting the appropriate test to classify a child are in the “How-To-Guide for Creating CLP-2 datasets.”</p> <p>³ BLL categories (in units of $\mu\text{g/dL}$) are <10, $10-<15$, $15-<20$, $20-<25$, $25-<45$, $45-<70$, and ≥ 70. An additional category for unconfirmed single capillary or unknown specimen tests is used to calculate the total number of children tested. Data are presented by categories at the state level only.</p>

Derivation of Measure(s)	<p>Create CLP-2 (county level) dataset using the <u>“How-To-Guide for Creating CLP-2 datasets.”</u></p> <ul style="list-style-type: none"> • Select children’s records from childhood lead poisoning database. • Classify test results. • Aggregate by county of residence and birth cohort. • Merge with total number of county to obtain the denominator. <p><u>From CLP-2 dataset, calculate the measures:</u></p> <ol style="list-style-type: none"> 1. Number of children born in the same year and tested, by county and state <ul style="list-style-type: none"> • Sum all BLL categories including the unconfirmed 2. Percent of children born in the same year and tested, by county and state <ul style="list-style-type: none"> • Divide number of children tested by the total number of children in the birth cohort 3. Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by county and state <ul style="list-style-type: none"> • Sum number of children in BLL categories $\geq 10 \mu\text{g/dL}$ (BLLs10_14,...,BLLs70), excluding unconfirmed 4. Percent of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by county and state <ul style="list-style-type: none"> • Divide number of children tested with BLLs $\geq 10 \mu\text{g/dL}$ by the total number of children tested and multiply by 100 5. Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by blood lead level category³, by state <ul style="list-style-type: none"> • Sum number of children by BLL categories $\geq 10 \mu\text{g/dL}$ (BLLs10_14,...,BLLs70), excluding unconfirmed 6. Percent of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by blood lead level category³, by state <ul style="list-style-type: none"> • BLL Categories = Divide number of children for each BLL category by the total number of children tested and multiply by 100
Unit	Number and percent
Geographic Scope	State or National
Geographic Scale	County or State (measures 1-4 available by county and state; measures

	5 and 6 available by state)
Time Period	2000 (or first available) to current
Time Scale	Annual birth cohort
Rationale	<p>Blood lead levels in young children have been associated with adverse health effects ranging from learning impairment and behavioral problems to death. No threshold for adverse effects has been identified. Because children may have elevated BLLs and not have any specific symptoms, CDC recommends blood lead testing for young children at risk for lead poisoning. The risk factors identified by the National Health and Nutrition Examination Surveys (NHANES) include living in housing built before 1950, especially housing in deteriorating condition, being African American, and living in poverty.</p> <p>Many states have adopted a targeted testing strategy (i.e., test children at high risk), whereas some states recommend universal testing (i.e., test all children), either statewide or within high-risk counties and cities. For both universal and targeted testing strategies, children should be tested at least once before the age of 3 years. Some states require more than one test between the ages of 6 and 36 months. In all states, a blood lead test is required for Medicaid-eligible children at 12 and 24 months of age.</p> <p>CDC updated its recommendations on children’s blood lead levels in May 2012. The new recommendation is based on the U.S population of children aged 1-5 years who are in the top 2.5% of children tested for lead in their blood. This reference value is the 97.5th percentile, which is currently 5 µg/dL based on NHANES 2007 – 2008 and 2009 – 2010 data (CDC, 2012). The recommendation that chelation therapy should be considered for children with BLLs ≥45 µg/dL has not changed. BLL results ≥70 µg/dL represent a medical emergency. Many states initiate environmental investigations at either BLLs ≥20 µg/dL or persistent BLL results that are 15-19 µg/dL</p> <p>This indicator uses a birth cohort approach. Using these measures, it is possible to determine how many children born in a specific year were tested before the ages of 3 and how many of those tested had an elevated BLL. For children with more than one test before the age of 3, this indicator uses the highest venous specimen result or if there is no venous specimen the highest confirmatory capillary/unknown result. Using the highest results allows for examination of the peak BLLs for the birth cohort. Inclusion of multiple cohorts will allow for the evaluation of trends in testing and BLLs greater than the reference value.</p>

<p>Use of the Measure(s)</p>	<ul style="list-style-type: none"> • To identify and monitor temporal and spatial changes in BLL testing and -BLLs by birth cohort. • To better understand BLL surveillance data when interpreting number of -BLLs. • To compare testing and BLLs within and across states for the purpose of targeting interventions. Comparisons should only be made between areas with similar testing and reporting rules. • To link data on risk factors and compare risk factors within and across states. • To guide interventions and allocation of resources related to BLL testing and prevention of lead exposure in young children.. • To develop and support public health policy and legislation related to BLL testing and prevention of childhood lead poisoning. • To monitor progress towards eliminating BLLs $\geq 5 \mu\text{g/dL}$, the current reference value (NHANES 2007 – 2008 and 2009 – 2010 data).
<p>Limitations of the Measure(s)</p>	<ul style="list-style-type: none"> • The analysis uses the county of the child’s residence at the time of the test, which may be different from the county where the child was exposed to lead. • Counties are not homogenous with respect to the distribution of lead hazards or risk factors for lead exposure. • Number and percent of BLLs cannot be interpreted as prevalence or incidence for the population. • State to state comparisons must be made cautiously and require additional information about the states’ testing practices, confirmatory testing practices, and reporting laws. • Because the capillary test is subject to contamination it can result in a false positive BLL. The number and percent of BLLs may be overestimated when non-venous test results are used.
<p>Data Sources</p>	<p>Childhood Blood Lead Surveillance Data Vital Statistics Birth Data</p>
<p>Limitations of Data Sources</p>	<p>Childhood Blood Lead Surveillance Data</p> <ul style="list-style-type: none"> • Surveillance data are not randomly sampled or representative of the population. • Complete residential addresses are not available for all children tested. • Sometimes the address of the provider or another address is listed as the child’s address when the data is not provided by the reporting authority. <p>Vital Statistics Birth Data</p> <ul style="list-style-type: none"> • The number of children born from Vital Statistics does not include children who have moved in or out of the area since birth. Therefore, as a denominator, it may under or over

	estimate the number of children in a birth cohort.
Presentation	<p>Small numbers of children tested, births, or BLLs may exist when the measures are calculated at the county levels. These small numbers are not accurate estimates for childhood lead poisoning in these polygons. In addition, these small numbers will require additional data processing steps to preserve confidentiality. One or more of the following methods can be used:</p> <ul style="list-style-type: none"> • Suppression of small numbers, • Aggregation of neighboring geographic units. • Aggregation to a lower resolved geographic level unit, • Aggregation of successive birth cohorts. <p>Data on blood lead levels are presented by categories at the state level only.</p> <p>This indicator should be displayed with information about the lead testing program, including:</p> <ul style="list-style-type: none"> • State and/or local testing policies or strategies (i.e., targeted or universal) • CDC-funded Childhood Lead Poisoning Prevention Program • Minimum BLL reported by laboratories to state or local lead program
Related Indicators	Blood Lead Testing and Housing Age Annual Blood Lead Levels
References	Centers for Disease Control and Prevention (CDC). 2012. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in “Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention”.

**CONTENT DOMAIN: CHILDHOOD LEAD POISONING
INDICATOR: ANNUAL BLOOD LEAD LEVELS**

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Exposure
Measure(s)	<p>1. Number of children tested, by age group¹, by county and state</p> <p>2. Percent of children tested, by age group¹, by county and state</p> <p>3. Number of children tested with confirmed blood lead levels ≥ 10 $\mu\text{g/dL}$^{3,4}, by age group¹, by county and state</p> <p>4. Percent of children tested with confirmed blood lead levels ≥ 10 $\mu\text{g/dL}$^{3,4}, by age group¹, by county and state</p> <p>5. Number of children tested with confirmed blood lead levels ≥ 10 $\mu\text{g/dL}$ by blood lead level category^{2,3,4}, by age group¹, by state</p> <p>6. Percent of children tested with confirmed blood lead levels ≥ 10 $\mu\text{g/dL}$, by blood lead level category^{2,3,4}, by age group¹, by state</p> <p>¹Measures are available stratified by two age groups: <36 months and 36 to <72 months</p> <p>²The current blood lead reference level is 5 $\mu\text{g/dL}$ based on National Health and Nutrition Examination Survey (NHANES) 2007 – 2008 and 2009 – 2010 data published in the Fourth National Report on Human Exposure to Environmental Chemicals, and updated in 2012. Blood Lead Levels (BLLs) ≥ 10 $\mu\text{g/dL}$ are confirmed if there is either: (1) one elevated venous test or (2) two elevated capillary and/or unknown tests at least 1 day but less than 12 weeks apart.</p> <p>³Details about selecting the appropriate test to classify a child are in the “How-To-Guide for Creating CLP-4 datasets.”</p> <p>⁴ BLL categories (in units of $\mu\text{g/dL}$) are <10, 10-14, 15-19, 20-24, 25-44, 45-69, and ≥ 70. An additional category for unconfirmed elevated capillary or unknown specimen tests is used to calculate the total number of children tested. Confirmed BLLs $\geq 10\mu\text{g/dL}$ and BLLs 5-9$\mu\text{g/dL}$, reflecting the NHANES reference value, will be included by Spring 2013. Data on confirmed BLLs $\geq 10\mu\text{g/dL}$ will be presented by blood lead categories at the state level only.</p>

Derivation of Measure(s)	<p>Create CLP-4 (county level) dataset using the “<u>How-To-Guide for Creating CLP-4 datasets.</u>”</p> <ul style="list-style-type: none"> • Select children’s records from childhood lead poisoning database. • Classify test results. • Aggregate by county of residence and year • Merge with total number of children by county to obtain the denominator. <p><u>From CLP-4 dataset, calculate the measures:</u></p> <ol style="list-style-type: none"> 1. Number of children tested <ul style="list-style-type: none"> • Sum all BLL categories including the unconfirmed 2. Percent of children tested <ul style="list-style-type: none"> • Divide number of children tested by the total number of children 3. Number of children tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$⁴ <ul style="list-style-type: none"> • Sum number of children in BLL categories $\geq 10 \mu\text{g/dL}$ (BLLs 10-14,...,BLLs70), excluding unconfirmed 4. Percent of children tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$⁴ <ul style="list-style-type: none"> • Divide number of children tested with blood lead levels $\geq 10 \mu\text{g/dL}$ by the total number of children tested and multiply by 100 5. Number of children tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$⁴ <ul style="list-style-type: none"> • Sum number of children for each BLL category 6. Percent of children tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$⁴ <ul style="list-style-type: none"> • Divide number of children for each BLL category by the total number of children tested and multiply by 100
Unit	Number and percent
Geographic Scope	State or National
Geographic Scale	County or State (measures 1-4 available at county and state; measures 5 and 6 available only at state)
Time Period	2000 to current
Time Scale	Annual
Rationale	Blood lead levels in children have been associated with adverse health effects ranging from learning impairment and behavioral problems to death. Lead can affect almost every organ and system in your body. The

	<p>effects of lead are the same whether it enters the body through breathing or swallowing. Small children can be exposed by eating lead-based paint chips, chewing on objects painted with lead-based paint or swallowing house dust or soil that contains lead. Children are more vulnerable to lead poisoning than adults. The main target for lead toxicity is the nervous system in young children. A child who swallows large amounts of lead may develop blood anemia, severe stomachache, muscle weakness, and brain damage. If a child swallows smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, lead can affect a child's mental and physical growth.</p> <p>Since children may have higher BLLs and not display any specific symptoms, CDC recommends blood lead testing for young children at risk for lead poisoning. The risk factors identified by the National Health and Nutrition Examination Surveys (NHANES) include living in housing built before 1950, especially housing in deteriorating condition, being African American, and living in poverty.</p> <p>States have developed and implemented assessment protocols for children to determine the need for a blood lead test. For both universal and targeted testing strategies, children should be tested at least once before the age of 3 years. Some states require more than one test between the ages of 6 and 36 months. Children not tested before the age of 3 should be tested at least once before the age of 6. In all states, a blood lead test is required for Medicaid-eligible children at 12 and 24 months.</p> <p>CDC updated its recommendations on children's blood lead levels in May 2012. The new recommendation is based on the U.S population of children aged 1-5 years who are in the top 2.5% of children tested for lead in their blood. This reference value is the 97.5th percentile, which is currently 5 µg/dL based on NHANES 2007 – 2008 and 2009 – 2010 data (CDC, 2012). The recommendation that chelation therapy should be considered for children with BLLs ≥45 µg/dL has not changed. BLL results ≥70 µg/dL represent a medical emergency. Many states initiate environmental investigations at either BLLs ≥20 µg/dL or persistent BLL results that are 15-19 µg/dL.</p> <p>This indicator provides information on the number of children tested each year and the number of those children tested with confirmed blood lead levels above 10 µg/dL. This information is used to direct resources for testing and management of elevated cases and be linked with environmental or the risk factor data to monitor trends over time.</p>
--	--

Use of the Measure(s)	<ul style="list-style-type: none"> • To identify and monitor temporal and spatial changes in BLL testing and confirmed BLLs $\geq 10\mu\text{g/dL}^4$ by year. • To better understand BLL surveillance data when interpreting number of confirmed BLLs $\geq 10\mu\text{g/dL}^4$. • To compare testing and BLLs within and across states for the purpose of targeting interventions. Comparisons should only be made between areas with similar testing and reporting rules. • To link data on risk factors and compare risk factors within and across states. • To guide interventions and allocation of resources related to BLL testing and prevention of EBLLs in children. • To develop and support public health policy and legislation related to BL testing and prevention of childhood lead exposure. • To monitor progress towards eliminating BLLs $\geq 5\ \mu\text{g/dL}$, the current reference value (NHANES 2007 – 2008 and 2009 – 2010 data).
Limitations of the Measure(s)	<ul style="list-style-type: none"> • The analysis uses the county of the child’s residence at the time of the test, which may be different from the county where the child was exposed to lead. • Counties are not homogenous with respect to the distribution of lead hazards or risk factors for lead exposure. • Number and percent of EBLLs through surveillance data cannot be interpreted as prevalence or incidence for the population as a whole • State to state comparisons must be made cautiously and require additional information about the states’ testing practices, confirmatory testing practices, and reporting laws. • Because the capillary test is subject to contamination it can result in a false positive EBLL. The number and percent of EBLLs would be overestimated if unconfirmed, non-venous test results are used.
Data Sources	Childhood Blood Lead Surveillance Data Census Population Data: Vintage bridged-race post-censal population estimates: http://www.cdc.gov/nchs/nvss/bridged_race.htm
Limitations of Data Sources	Childhood Blood Lead Surveillance Data <ul style="list-style-type: none"> • Surveillance data are not randomly sampled or representative of the population. • Complete residential addresses are not available for all children tested. • If the child’s address is not provided the address of the provider may be used.
Related Indicators	Blood Lead Testing and Housing Age Blood Lead Levels by Birth Cohort
References	Centers for Disease Control and Prevention (CDC). 2012. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in “Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention”.

INDICATOR TEMPLATE
CONTENT AREA: CLIMATE AND HEALTH
INDICATOR: HEAT STRESS HOSPITALIZATIONS

Type of EPHT Indicator	Health outcome
Measures	<ol style="list-style-type: none"> 1. Age-adjusted rate of hospitalization for heat stress per 100,000 population 2. Crude rate of hospitalization for heat stress per 100,000 population 3. Number of hospitalizations for heat stress
Derivation of Measure(s)	<p><i>Numerator:</i> Hospital admissions having any ICD-9 code in the range of 992.0-992.9, or cause of injury code E900.0 or E900.9, EXCLUDING cases with a code of E900.1 (man-made source of heat) anywhere in the record.</p> <p><i>Denominator:</i> Midyear resident population, by gender, for state and by county</p> <p><i>Adjustment:</i> Age-adjustment by the direct method to year 2000 US standard population</p>
Unit	<ol style="list-style-type: none"> 1. Age-adjusted rate per 100,000 population 2. Rate per 100,000 population 3. Number
Geographic Scope	State and national
Geographic Scale	Residents of jurisdiction – State
Time Period	Hospital admissions between May 1 to September 30, inclusive, for each year, 2000–
Time Scale	May–September of each data year
Rationale	<p>The Intergovernmental Panel on Climate Change (IPCC) projects with “virtual certainty” suggest that climate change will cause more frequent, more intense, and longer heat waves (1). Any individual, regardless of age, sex or health status can develop heat stress if engaged in intense physical activity and/or exposed to environmental heat (and humidity). Physiologic mechanisms maintain the core body temperature (i.e., the operating temperature of vital organs in the head or trunk) in a narrow optimum range around 37 °C (98.6 °F). When core body temperature rises, the physiologic response is to sweat and circulate blood closer to the skin's surface to increase cooling. If heat exposure exceeds the physiologic capacity to cool, and core body temperature rises, then a range of heat-related symptoms and conditions can develop. Heat stress or Heat-related illness ranges from mild heat edema and rash, heat syncope, heat cramps, to the most common type, heat exhaustion (2). Heat-related cramps, rash, and edema are relatively minor readily treatable conditions; however, they should be used as important warning signs to immediately remove the affected individual from the exposure situation.</p>

Heat cramps are brief, intermittent, and often severe muscular cramps occurring typically in muscles that are fatigued by heavy work (2). Individuals with heat cramp can also exhibit hyponatremia, hypochloremia (which are low serum sodium and chloride levels).

Heat syncope is a temporary loss of consciousness as a result of prolonged heat exposure (2). Individuals adapt to hot, humid environment by dilation of cutaneous vessels in the skin to radiate heat. Peripheral vasodilation along with blood volume loss, results in lowering the blood pressure which can result in inadequate central venous return and cerebral perfusion, causing light-headedness and fainting.

Heat exhaustion is a consequence of extreme depletion of blood plasma volume, which may be coincident with hyponatremia and/or peripheral blood pooling (2). Heat exhaustion often does not present with definitive symptoms and may be misdiagnosed, often as an acute viral illness. Symptoms include mild disorientation, generalized malaise, weakness, nausea, vomiting, headache, tachycardia (rapid beating of the heart), and hypotension. Because untreated heat exhaustion can progress to heat stroke, the most serious form of heat-related illness, treatment should begin at the first signs of heat exhaustion (3).

Heat stroke is an extreme medical emergency that if untreated can result in death or permanent neurological impairment (2). Heat stroke occurs when a person's core body temperature rises above 40 °C (104 °F) as a result of impaired thermoregulation. High core body temperature and disseminated intravascular coagulation results in cell damage in vital organs, such as the brain, liver, and kidneys, which can lead to serious illness and death (3). Death may occur rapidly due to cardiac failure or hypoxia, or it can occur days later as a result of renal failure due to dehydration and/or rhabdomyolysis (i.e., the breakdown of muscle fibers with release into the circulation of muscle fiber contents, some of which are toxic to the kidney and can cause kidney damage) (4). Heat stroke is typically divided into two types. The two types are in general clinically the same, except that the individuals/population groups affected require medical interventions specific to their unique physiology and medical status (3). "Exertional Heat Stroke," as the name implies, involves strenuous physical activity under high temperature conditions to which the heat stroke victim was not acclimatized, and usually affects healthy young adults, such as athletes, outdoor laborers and soldiers. "Classic" heat stroke, by definition does not involve exertion, and usually affects susceptible individuals, such as infants and young children, the elderly, or people with chronic illness. Because heat stroke, even if treated, can have a death rate as high as 33%, and up to 17% of heat stroke survivors suffer permanent damage, measures should be taken to prevent heat-related illness, especially among vulnerable populations.

The relationship between extreme heat and increased daily morbidity and mortality is well established. This indicator captures hospital admissions directly attributed to heat stress (e.g., heat illness, heat stroke, and hyperthermia). It is a measure that can

	be tracked easily and consistently across geography and time, and acts as a sentinel for the broader range of heat-related illness that is not recognized and/or coded as such.
Use of the Measure	Heat stress can manifest in a number of clinical outcomes, and people with chronic health problems (e.g., cardiovascular disease, diabetes, obesity) are more susceptible to the effects of heat than healthy individuals. For these reasons, heat stress may not be listed as the primary diagnosis. This indicator therefore includes all cases where heat stress is explicitly listed as the primary diagnosis or any other diagnosis. Increases in the rates of hospital admission for heat stress are one potential impact of rising global temperatures. Tracking these data can help document changes over place and time, monitor vulnerable areas, and evaluate the results of local climate-adaptation strategies.
Limitations of the Measure	Periods of extreme heat are frequently associated with increases in hospital visits and admissions for many causes. This measure does not capture the full spectrum of heat stress, especially where exposure to excess heat is not explicitly documented.
Data Sources	<i>Numerator:</i> State inpatient hospital discharge data (using admission date) <i>Denominator:</i> US Census Bureau population data
Limitations of Data Sources	<i>State hospital discharge data:</i> <ul style="list-style-type: none"> • Using a measure of all heat stress hospitalizations will include some transfers between hospitals for the same individual for the same heat stress event. Variations in the percentage of transfers or readmissions for the same heat stress event may vary by geographic area and impact rates. • Without reciprocal reporting agreements with abutting states, statewide measures and measures for geographic areas (e.g., counties) bordering other states may be underestimated because of health care utilization patterns. • Each state must individually obtain permission to access and, in some states, provide payment to obtain the data. • Veterans Affairs, Indian Health Services and institutionalized (e.g. Prison) populations are excluded. • Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers to hospitalize patients • Street address is currently not available in many states. • Sometimes mailing address of patient is listed as the residence address of the patient • Patients may be exposed to environmental triggers in multiple locations, but hospital discharge geographic information is limited to residence. • Since the data captures hospital discharges (rather than admissions), patients admitted toward the end of the year and discharged the following year will be omitted from the current year dataset

	<ul style="list-style-type: none"> • Data will need to be de-duplicated (i.e. remove duplicate records for the same event) • There is usually a two year lag period before data are available from the data owner. <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census
Related Indicators	<ul style="list-style-type: none"> • Heat vulnerability • Heat-related mortality • Temperature distribution • Emergency department visits for heat stress
References	<ol style="list-style-type: none"> 1. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. 2007. Human health In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE. , editors. Climate Change 2007: Impacts, Adaptation and Vulnerability Contribution of Working Group II to: Fourth Assessment Report of the Intergovernmental Panel on Climate Change. New York: Cambridge University Press. pp. 391–431. 2. Rosen’s Emergency Medicine: Concepts and Clinical Practice. 2010. Chapter 139: Heat illness. In JA Marx Editor-in-Chief; RS Hockberger & RM Walls Senior Editors; JG Adams ... [et al] Editors (7th ed). Philadelphia: Mosby Elsevier. 3. American Medical Association. Heat-related Illness During Extreme Weather Emergencies (Report 10 of the Council on Scientific Affairs (A97), 1997; www.ama-assn.org/ama/pub/category/13637.html). 4. Centers for Disease Control and Prevention. Heat-related deaths--Los Angeles County, California, 1999-2000, and United States, 1979-1998. MMWR 2001;50(29):623-6.

INDICATOR TEMPLATE
CONTENT AREA: CLIMATE AND HEALTH
INDICATOR: EMERGENCY DEPARTMENT VISITS FOR HEAT STRESS

Type of EPHT Indicator	Health outcome
Measures	<ol style="list-style-type: none"> 1. Annual age-adjusted rate of emergency department visits for heat stress per 100,000 population 2. Annual crude rate of emergency department visits for heat stress per 100,000 population 3. Annual number of emergency department visits for heat stress
Derivation of Measure(s)	<p><i>Numerator:</i></p> <ul style="list-style-type: none"> • Patients treated in an Emergency Department (ED) having any ICD-9 code in the range of 992.0-992.9, or cause of injury code E900.0 or E900.9. • Cases with a code of E900.1 (man-made source or heat) anywhere in the record are <u>excluded</u>. • <p><i>Denominator:</i> Midyear resident population, by gender, for state and by county</p> <p><i>Adjustment:</i></p> <ul style="list-style-type: none"> • Age-adjustment by the direct method to the Year 2000 US Standard population • U.S. 2000 standard population by age categories from Surveillance Epidemiology and End Results (SEER), National Cancer Institute
Unit	<ol style="list-style-type: none"> 5. Age-adjusted rate per 100,000 population 6. Rate per 100,000 population 7. Number
Geographic Scope	State and national
Geographic Scale	State (annual), County (aggregate years)
Time Period	Hospital admissions between May 1 to September 30, inclusive, for each year, 2000–
Time Scale	May–September of each data year
Rationale	<p>The Intergovernmental Panel on Climate Change (IPCC) projects with “virtual certainty” suggest that climate change will cause more frequent, more intense, and longer heat waves (1). Any individual, regardless of age, sex or health status can develop heat stress if engaged in intense physical activity and/or exposed to environmental heat (and humidity). Physiologic mechanisms maintain the core body temperature (i.e., the operating temperature of vital organs in the head or trunk) in a narrow optimum range around 37 °C (98.6 °F). When core body temperature rises, the physiologic response is to sweat and circulate blood closer to the skin's surface to increase cooling. If heat exposure exceeds the physiologic capacity to cool, and core body temperature rises, then a range of heat-related symptoms and conditions can develop. Heat stress or Heat-related illness ranges from mild heat edema, rash, heat syncope, heat cramps, to the most common type,</p>

heat exhaustion (2). Heat-related cramps, rash, and edema are relatively minor readily treatable conditions; however, they should be used as important warning signs to immediately remove the affected individual from the exposure situation.

Heat cramps are brief, intermittent, and often severe muscular cramps occurring typically in muscles that are fatigued by heavy work (2). Individuals with heat cramp can also exhibit hyponatremia, hypochloremia, and low serum sodium and chloride levels.

Heat syncope is a temporary loss of consciousness as a result of prolonged heat exposure (2). Individuals adapt to hot, humid environment by dilation of cutaneous vessels in the skin to radiate heat. Peripheral vasodilation along with blood volume loss, results in lowering the blood pressure which can result in inadequate central venous return and cerebral perfusion, causing light-headedness and fainting.

Heat exhaustion is a consequence of extreme depletion of blood plasma volume, which may be coincident with hyponatremia and/or peripheral blood pooling (2). Heat exhaustion often does not present with definitive symptoms and may be misdiagnosed, often as an acute viral illness. Symptoms include mild disorientation, generalized malaise, weakness, nausea, vomiting, headache, tachycardia (rapid beating of the heart), and hypotension. Because untreated heat exhaustion can progress to heat stroke, the most serious form of heat-related illness, treatment should begin at the first signs of heat exhaustion (3).

Heat stroke is an extreme medical emergency that if untreated can result in death or permanent neurological impairment (2). Heat stroke occurs when a person's core body temperature rises above 40 °C (104 °F) as a result of impaired thermoregulation. High core body temperature and disseminated intravascular coagulation results in cell damage in vital organs, such as the brain, liver, and kidneys, which can lead to serious illness and death (3). Death may occur rapidly due to cardiac failure or hypoxia, or it can occur days later as a result of renal failure due to dehydration and/or rhabdomyolysis (i.e., the breakdown of muscle fibers with release into the circulation of muscle fiber contents, some of which are toxic to the kidney and can cause kidney damage) (4). Heat stroke is typically divided into two types. The two types are in general clinically the same, except that the individuals/population groups affected require medical interventions specific to their unique physiology and medical status (3). "Exertional Heat Stroke," as the name implies, involves strenuous physical activity under high temperature conditions to which the heat stroke victim was not acclimatized, and usually affects healthy young adults, such as athletes, outdoor laborers and soldiers. "Classic" heat stroke, by definition does not involve exertion, and usually affects susceptible individuals, such as infants and young children, the elderly, or people with chronic illness. Because heat stroke, even if treated, can have a death rate as high as 33%, and up to 17% of heat stroke survivors suffer permanent be taken to prevent heat-related illness, especially among vulnerable populations.

The relationship between extreme heat and increased daily morbidity and mortality is well established. This indicator captures hospital admissions *directly* attributed to heat

	stress (e.g., heat illness, heat stroke, and hyperthermia). It is a measure that can be tracked easily and consistently across geography and time, and acts as a sentinel for the broader range of heat-related illness that is not recognized and/or coded as such.
Use of the Measure	<p>Heat stress can manifest in a number of clinical outcomes, and people with chronic health problems (e.g., cardiovascular disease, diabetes, obesity) are more susceptible to the effects of heat than healthy individuals. For these reasons, heat stress may not be listed as the primary diagnosis. This indicator therefore includes all cases where heat stress is explicitly listed as the primary diagnosis or any other diagnosis.</p> <p>Increases in the rates of ED visits for heat stress are one potential impact of rising global temperatures. Tracking these data can help document changes over place and time, monitor vulnerable areas, and evaluate the results of local climate-adaptation strategies.</p>
Limitations of the Measure	Periods of extreme heat are frequently associated with increases in hospital visits and admissions for many causes. This measure does not capture the full spectrum of heat-stress, where exposure to excess heat is not explicitly documented.
Data Sources	<p><i>Numerator:</i> State emergency department data</p> <p><i>Denominator:</i> US Census Bureau population data</p>
Limitations of Data Sources	<p><i>Emergency Department data:</i></p> <ul style="list-style-type: none"> • Data are not available for all states. • Number of diagnostic fields in hospital records varies from state to state. Utilization of EDs varies geographically. <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year.
Related Indicators	<ul style="list-style-type: none"> • Heat vulnerability • Heat-related mortality • Temperature distribution • Heat stress hospitalizations
References	<ol style="list-style-type: none"> 1. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. 2007. Human health In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE. , editors. Climate Change 2007: Impacts, Adaptation and Vulnerability Contribution of Working Group II to: Fourth Assessment Report of the Intergovernmental Panel on Climate Change. New York: Cambridge University Press. pp. 391–431. 2. Rosen’s Emergency Medicine: Concepts and Clinical Practice. 2010. Chapter 139: Heat illness. In JA Marx Editor-in-Chief; RS Hockberger & RM Walls Senior Editors; JG Adams ... [et al] Editors (7th ed). Philadelphia: Mosby Elsevier. 3. American Medical Association. Heat-related Illness During Extreme Weather Emergencies (Report 10 of the Council on Scientific Affairs (A97), 1997; www.ama-assn.org/ama/pub/category/13637.html). 4. Centers for Disease Control and Prevention. Heat-related deaths--Los Angeles County, California, 1999-2000, and United States, 1979-1998. MMWR 2001; 50(29):623-6.

CONTENT DOMAIN: COMMUNITY WATER

INDICATOR: ATRAZINE

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Quarterly distribution of number of Community Water Systems (CWS) by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 2. Yearly distribution of number of CWS by maximum atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 3. Yearly distribution of number of CWS by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 4. Mean concentration of atrazine at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 1. Quarterly distribution of number of people served by CWS by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 2. Yearly distribution of number of people served by CWS by maximum atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 3. Yearly distribution of number of people served by CWS by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine).
Derivation of Measures	Atrazine measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	µg/L of Atrazine
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Atrazine and Public Health</p> <p>Atrazine is a widely used herbicide active against broadleaf and grassy weeds. Atrazine was first registered as an herbicide in 1958. More than 70 million pounds have been applied</p>

annually in recent years, with about 75% of corn cropland receiving treatment. In addition to agricultural uses, atrazine is used in residential turf applications and on golf courses and sod farms to control weeds. Atrazine and its degradation products are the most commonly detected pesticides in ground and surface waters (Barr et al., 2007). The frequent detection of atrazine and its degradation products in streams, rivers, groundwater, and reservoirs is related directly to the volume of its use, its persistence in soils due to its resistance to photolysis and hydrolysis, and its ability to travel within water systems (Nelson et al., 2001). In water systems, atrazine is transformed over time by various chemical reactions into other compounds or its degradation products or metabolites, including dealkylated compounds such as desethylatrazine (DEA), desisopropylatrazine (DIA), and diaminochlorotriazine (DACT). In soil, atrazine degrades slowly to dealkylated compounds, which have half-lives of several months. Bacteria and plants can metabolize atrazine to hydroxylated products. In plants, atrazine is absorbed by the root system and tends to form hydroxylated metabolites that cannot be removed by washing contaminated vegetables (Nelson et al., 2001). Atrazine does not bioaccumulate. Studies suggest that in animals, the degradation products that retain the chlorine have biologic activity similar to that of atrazine, while the hydroxylated metabolites do not retain its biologic activity (Nelson et al., 2001). Use of atrazine in the presence of nitrogen fertilizers, has raised a possibility of N-nitrosation in soil (DeMarini and Zahm, 1999). There may also be endogenous formation of N-nitrosoatrazine from precursors ingested in the diet and drinking water. For the general population, drinking water is an infrequent source of atrazine exposure, but estimates of seasonal intakes from drinking water in a small number of communities have exceeded the recommended limits (U.S. EPA, 2003). As a result, atrazine use has progressively been restricted in an effort to reduce surface and ground water contamination.

In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, atrazine was detected in 888 systems serving greater than 34 million people (EPA, 2009). Concentrations of atrazine were greater than the MCL in 98 systems serving 3.1 million people. Atrazine was the second highest occurring regulated synthetic organic chemical found based on the percent of detections found from the 6 Year Review data (EPA, 2009).

While it is used on many crops, atrazine has not been found in many food samples, and then only at very low levels. Therefore, it is very unlikely that people would be exposed to atrazine by eating crops from atrazine-accumulated soil.

Most people are not exposed regularly to atrazine. People living near areas where atrazine was applied to crops may be exposed through contaminated drinking water. Atrazine has been found at about 20 Superfund sites in the United States. People living near those sites may be exposed to higher levels of atrazine. Factory workers who work with atrazine may be exposed to higher amounts of atrazine than other workers. The government has estimated that approximately 1,000 people may be exposed to atrazine in this way (ATSDR, 2003).

Applicators of atrazine may be exposed dermally and by inhalation. Atrazine is well absorbed orally, metabolized, and then eliminated in the urine over a few days (Bradway et al., 1982; Catenacci et al., 1993; Timchalk et al., 1990).

Metabolism of atrazine and its degradation products is complex and results in many potential metabolites (Barr et al., 2007). As many as 8-12 metabolites of atrazine have been identified in animals and humans, with recent studies showing DACT as the primary

metabolite (Barr et al., 2007); therefore, earlier biomonitoring studies measuring atrazine mercapturate alone misrepresent and underestimate total atrazine exposure. Panuwet et al., (2008) developed an analytical method that measures the seven primary urinary metabolites of atrazine, which are: hydroxyatrazine, DACT, DIA, DEA, desethylatrazine mercapturate, atrazine mercapturate, and atrazine itself.

Human health effects of atrazine at environmental doses or at biomonitored levels from environmental exposure are unknown. In mammalian studies, atrazine is rated as having low acute toxicity. Atrazine product formulations can be mild skin sensitizers and irritants. Some human ecologic and epidemiologic studies of reproductive and cancer outcomes have shown either positive or no associations, but effects are difficult to attribute due to lack of exposure markers or due to mixed chemical or pesticide exposures (ATSDR, 2003; Gammon et al., 2005; Sathiakumar and Delzell, 1997). Studies of couples living on farms that use atrazine for weed control found an increase in the risk of pre-term delivery. These studies are difficult to interpret because most of the farmers were men who may have been exposed to several types of pesticides. A meta-analysis linked hypospadias to parental exposure to pesticides with possible endocrine-mediated effects (Rocheleau et al., 2009). Some epidemiological studies that looked at the potential impact of prenatal exposure to atrazine or its products of environmental degradation on pregnancy outcomes in the general population observed higher rates of babies born small-for gestational age (SGA) (Munger et al., 1997, Villanueva et al., 2005; Ochoa-Acuna et al., 2009). They also linked exposure of mothers who lived closer to sites with high atrazine concentrations with a higher risk of gastroschisis (Waller et al., 2010). Most of these studies were retrospective and relied on ecological assessment of exposure to atrazine. However, the most recent study that measured urinary biomarkers of prenatal atrazine exposure and was based on a prospective population-based cohort found associations between environmental exposure to atrazine and adverse effects on fetal growth, specifically birth weight, birth length, and small head circumference (Chevrier et al., 2011). Atrazine is not mutagenic and is not considered genotoxic. The International Agency for Research on Cancer (IARC) considers atrazine not classifiable with respect to human carcinogenicity, and the EPA considers atrazine unlikely to be a human carcinogen. However, IARC recommends future research to characterize the ability of atrazine to interfere with the hypothalamic-pituitary-ovarian axis in women. This research would help determine whether atrazine is a mammary carcinogen in women. Another area for future research is to explore atrazine's ability to alter immune and aromatase function in humans. Additional information is available from U.S. EPA at: <http://www.epa.gov/pesticides/>; from ATSDR at: <http://www.atsdr.cdc.gov/toxpro2.html>, and IARC at <http://www.iarc.fr/>

Children are likely to be exposed to atrazine in the same way as adults, primarily through contact with dirt that contains atrazine or by drinking water from wells that are contaminated with the herbicide. Little information is available about the effects of atrazine in children. Maternal exposure to atrazine in drinking water has been associated with low fetal weight and heart, urinary, or limb defects in humans. It is not known whether atrazine or its metabolites can be transferred from a pregnant mother to a developing fetus through the placenta or from a nursing mother to her offspring through breast milk.

Biomonitoring Information

Urinary levels of atrazine mercapturate reflect recent exposure. In the NHANES 2001–2002 subsample, levels of atrazine mercapturate were generally not detectable (CDC, 2005). In small studies of Maryland residents in 1995–1996 (MacIntosh et al., 1999) and 83

Minnesota children with multiple urine collections during 1997 (Adgate et al., 2001), atrazine mercapturate was infrequently detected at the detection limit of 0.3 µg/L. In a study of 60 farm worker children, atrazine was detected in only four children (Arcury et al., 2007). Using immunoassay atrazine equivalents (detected mostly as atrazine mercapturate), the urinary geometric mean levels for herbicide applicators in Ohio and Wisconsin were about 6 µg/L (Hines et al., 2003; Perry et al., 2000). The geometric mean of urinary atrazine mercapturate was 1.2 µg/L in 15 farmers studied several days after spraying the pesticide (Curwin et al., 2005). In a small number of field workers, urinary concentrations ranged from 5-1756 µg/L (Lucas et al., 1993). However, biomonitoring studies that have evaluated only one urinary metabolite of atrazine (such as atrazine mercapturate) probably underestimated exposure (Barr et al., 2007).

Finding measurable amounts of atrazine or its metabolites in urine does not mean that the levels of atrazine and its metabolites (e.g., atrazine mercapturate) cause an adverse health effect. Biomonitoring studies on levels of atrazine mercapturate provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of atrazine than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

Sources of Atrazine

Atrazine is the common name for an herbicide that is widely used to kill weeds. It is used mostly on farms. Pure atrazine—an odorless, white powder—is not very volatile, reactive, or flammable. It will dissolve in water. Atrazine is made in the laboratory; it does not occur naturally.

Atrazine is used on crops such as sugarcane, corn, pineapples, sorghum, and macadamia nuts, and on evergreen tree farms and for evergreen forest re-growth. It has also been used to keep weeds from growing on both highway and railroad rights-of-way. Some of the trade names of atrazine are Aatrex®, Aatram®, Atratol®, and Gesaprim®. The scientific name for atrazine is 6-chloro-N-ethyl-N'-(1-methylethyl)-triazine-2,4-diamine. Atrazine is a Restricted Use Pesticide, which means that only certified herbicide users may purchase or use it. Certification for the use of atrazine is obtained through the appropriate state office where the herbicide user is licensed. Atrazine is usually used in the spring and summer months. For it to be active, atrazine needs to dissolve in water and enter the plants through their roots. It then acts in the shoots and leaves of the weed to stop photosynthesis. Atrazine is taken up by all plants, but in plants not affected by atrazine, it is broken down before it can affect photosynthesis. The application of atrazine to crops as an herbicide accounts for almost all of the atrazine that enters the environment, but some may be released from manufacture, formulation, transport, and disposal.

Any atrazine that is washed from the soil into streams and other bodies of water will stay there for a long time, because chemical breakdown is slow in rivers and lakes. It also will persist for a long time in groundwater. This is one reason why atrazine is found commonly in the water collected from drinking water wells in some agricultural regions.

If atrazine enters the air, it can be broken down by reactions with other reactive chemicals in the air. However, sometimes atrazine is on particles such as dust. When this happens, breakdown is not expected. Atrazine is removed from air mainly by rainfall. When atrazine is on dust particles, the wind can blow it long distances from the nearest application area.

	<p>For example, atrazine has been found in rainwater more than 180 miles (300 kilometers) from the nearest application area.</p> <p>Atrazine does not tend to accumulate in living organisms such as algae, bacteria, clams, or fish, and, therefore, does not tend to build up in the food chain.</p> <p>Atrazine Regulation and Monitoring</p> <p>Congress established the Safe Drinking Water Act in 1974, which set enforceable Maximum Contaminant Levels (MCLs) and non-enforceable Maximum Contaminant Level Goals (MCLGs) for certain, specified contaminants. In the case of atrazine in drinking water, EPA has set an MCL of 3 µg/L. Atrazine is designated as a Restricted Use Pesticide, which means that only certified pesticide applicators can use atrazine. The Occupational Safety and Health Administration (OSHA) has set a limit of 5 milligrams of atrazine per cubic meter of workplace air (5 mg/m³) for an 8-hour workday and 40-hour work week. EPA has determined maximum levels allowed in foods of 0.02-15 parts atrazine per million parts of food (0.02-15 ppm).</p>
Use of Measure	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to atrazine at different concentrations. • Maximum concentrations provide information on the peak potential exposure to atrazine at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of the Measure	<p>The current measures are derived for CWS only. Private wells are another important source of population exposure to atrazine in some agricultural regions. Transient non-community water systems, which are regulated by EPA, may also be an important source of atrazine exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be converted directly to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Ground water systems may have many wells with different atrazine concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the atrazine concentration of people served by wells with higher atrazine concentrations.</p> <p>Exposure may be higher or lower than estimated if data from multiple entry points for water with different atrazine levels are averaged to estimate levels for the PWS.</p>

Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. Adgate JL, Barr DB, Clayton CA, Eberly LE, Freeman NC, Liroy PJ, et al. Measurement of children's exposure to pesticides: analysis of urinary metabolite levels in a probability-based sample. <i>Environ Health Perspect</i> 2001;109(6):583-590. 2. Agency for Toxic Substances and Disease Registry (ATSDR). 2003. Toxicological Profile for Atrazine. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. 3. Arcury TA, Grzywacz JG, Barr DB, Tapia J, Chen H, Quandt SA. Pesticide urinary metabolite levels of children in eastern North Carolina farmworker households. <i>Environ Health Perspect</i> 2007;115(8):1254-1260. 4. Barr D.B., P. Panuwet, J.V. Nguyen, S. Udunka, L.L. Needham. Assessing exposure to atrazine and its metabolites using biomonitoring. <i>Environmental Health Perspectives</i> 2007, Vol. 115, No. 10, 1474-1478. 5. Bradway DE, Moseman RF. Determination of urinary residue levels of the N-dealkyl metabolites of triazine herbicides. <i>J Agric Food Chem</i> 1982;30(2):244-247. 6. Catenacci G, Barbieri F, Bersani M, Ferioli A, Cottica D, Maroni M. Biological monitoring of human exposure to atrazine. <i>Toxicol Lett</i> 1993;69(2):217-222. 7. Centers for Disease Control and Prevention (CDC). Third National Report on Human Exposure to Environmental Chemicals. Atlanta (GA). 2005. 3/11/09 8. Chevrier C., G. Limon. C. Monfort, f. Rouget, R. Garlantezec, C. Petit, G. Durand, S. Cordier. Urinary biomarkers of prenatal atrazine exposure and adverse birth outcomes In the PELAGIE Birth Cohort. <i>Environmental Health Perspectives</i> 2011, March 2 (doi:10.1289/ehp.1002775) 9. Curwin BD, Hein MJ, Sanderson WT, Barr DB, Heederik D, Reynolds SJ, et al. Urinary and hand wipe pesticide levels among farmers and nonfarmers in Iowa. <i>J Expo Anal Environ Epidemiol</i> 2005;15(6):500-508. 10. DeMarini DM, Zahm SH. Atrazine IARC Monographs 73, 1999. 11. Gammon DW, Aldous CN, Carr WC Jr, Sanborn JR, Pfeifer KF. A risk assessment of atrazine use in California: human health and ecological aspects. <i>Pest Manag Sci</i> 2005;61(4):331-355. 12. Hines CJ, Deddens JA, Striley CA, Biagini RE, Shoemaker DA, Brown KK, et al. Biological monitoring for selected herbicide biomarkers in the urine of exposed custom applicators: application of mixed-effect models. <i>Ann Occup Hyg</i> 2003;47(6):503-517. 13. Lucas AD, Jones AD, Goodrow MH, Saiz SG, Blewett C, Seiber JN, et al. Determination of atrazine metabolites in human urine: development of a biomarker of exposure. <i>Chem Res Toxicol</i> 1993;6(1):107-116. 14. MacIntosh DL, Needham LL, Hammerstrom KA, Ryan PB. A longitudinal investigation of selected pesticide metabolites in urine. <i>J Expo Anal Environ Epidemiol</i> 1999;9(5):494-501. 15. Munger R., P. Isacson, S. Hu, T., Burns, J. Hanson, C. F. Lynch et al., Intrauterine growth retardation in Iowa communities with herbicide-contaminated drinking water supplies. <i>Environmental Health Perspectives</i>, 1997; Vol., 105, 308-314. 16. Ochoa-Acuna H., J. Frankenberger J., L. Hahn, C. Carbajo. Drinking-water herbicide exposure in Indiana and prevalence of small-for-gestational-age and preterm delivery. <i>Environmental Health Perspectives</i> 2009; Vol. 117, 10, 1619-1624. 17. Panuwet R, J. V. Nguyen, P. Kuklenyik, S. O. Udunka, L.L. Needham, D. B. Barr. Quantification of atrazine and its metabolites in urine by on-line solid-phase extraction-high-performance liquid chromatography-tandem mass spectrometry. <i>Anal Bioanal Chem</i> 2008; 391: 1931-1939.

	<p>18. Rocheleau C. M., P. A. Romitti, L. K. Dennis. Pesticides and hypospadias: a meta-analysis. <i>J Pediatr Urol</i> 2009; Vol. 5, 17-24.</p> <p>19. Perry M, Christiani D, Dagenhart D, Tortorelli J, Singzoni B. Urinary biomarkers of atrazine exposure among farm pesticide applicators. <i>Ann Epidemiol</i> 2000;10(7):479.</p> <p>20. Sathiakumar N, Delzell E. A review of epidemiologic studies of triazine herbicides and cancer. <i>Crit Rev Toxicol</i> 1997;27(6):599-612.</p> <p>21. Timchalk C, Dryzga MD, Langvardt PW, Kastl PE, Osborne DW. Determination of the effect of tridiphane on the pharmacokinetics of [14C]-atrazine following oral administration to male Fischer 344 rats. <i>Toxicology</i> 1990;61(1):27-40.</p> <p>22. U.S. Environmental Protection Agency (U.S. EPA). Interim Reregistration Eligibility Decision For Atrazine. Case No. 0062. 2003. Available at URL: http://www.epa.gov/oppsrrd1/REDS/atrazine_ired.pdf . 3/11/09</p> <p>23. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.</p> <p>24. Villanueva C. M., G. Durand, M. B. Coutte, C. Chevrier, S. Cordier. Atrazine in municipal drinking water and risk of low birth weight, preterm delivery, and small-for-gestational-age status. <i>Occupational Environ Med</i> 2005; Vol. 62, 6, 400-405.</p> <p>25. Waller S. A., K. Paul, S. E. Peterson, J. E. Hitti. Agricultural-related chemical exposures, season of conception, and risk of gastroschisis in Washington State. <i>Am J Obstet Gynecol</i> 2010, Vol. 202, 241.</p>
--	--

CONTENT DOMAIN: COMMUNITY WATER

INDICATOR: ARSENIC

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of Community Water Systems (CWS) by maximum arsenic concentration (cut-points: 0-5, >5-10, >10-30, >30 µg/L arsenic). 2. Yearly distribution of number of CWS by mean arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic). 3. Mean concentration of arsenic at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of people served by CWS by maximum arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic). 2. Yearly distribution of number of people served by CWS by mean arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic).
Derivation of Measures	<p>Arsenic measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.</p>
Units	Concentration of arsenic, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Arsenic and Public Health</p> <p>Exposures to higher than average levels of arsenic can come from elevated localized soil and ground water concentrations from application and runoff of</p>

	<p>arsenical pesticides and leachate from coal ash and landfills (ATSDR 2005). Exposure to hundreds of micrograms per liter of arsenic found in drinking water of Taiwan, Chile, Argentina, Mexico, Bangladesh, and India has been associated with many adverse health effects including lung, bladder, liver and skin cancers (NRC, 1999; Rahman et al. 2005; Salazar et al. 2004; Fazal et al., 2001). Arsenic has been identified as a human carcinogen by the International Agency for Research in Cancer (IARC) (IARC, 2004). Other adverse health effects include nausea, cardiovascular disease, (Chen et al., 2007; Chih-Hao et al., 2007; Bunderson et al., 2004), developmental and reproductive effects (Hopenhayn et al., 2003; Ahmad et al., 2001)), Diabetes Mellitus (Rahman et al., 1998), and skin keratosis and hyperpigmentation (Kapaj et al., 2006).</p> <p>Measured arsenic concentrations in finished drinking water can be used to understand the distribution of potential arsenic exposure levels for populations served by community water supplies. These measures allow for comparison of potential for arsenic exposures between the populations served by different water systems and water sources over time, and potentially across demographic groups.</p> <p>Sources of Arsenic</p> <p>Arsenic compounds (As (III) and As (V)) are found in both ground water and surface waters. The primary sources are geologic formations from which arsenic can be dissolved. Higher levels of arsenic tend to be found in ground water (e.g. aquifers) as compared to surface waters (e.g., lakes, rivers).</p> <p>Arsenic Regulation and Monitoring</p> <p>In 2001 EPA reduced the regulatory drinking water standard Maximum Contaminant Level (MCL) to 10 µg/L from 50 µg/L (effective January 23, 2006) on the basis of bladder and lung cancer risks (EPA 2001a). The cancer risks were extrapolated from the Taiwanese (Chen et al. 1985) study to U.S. risks. Lowering the MCL from 50 to 10 ppb statistically reduces bladder and lung cancer mortality and morbidity by 37-56 cancers a year in the U.S. (EPA 2001b). Based on the current understanding of the health impacts from arsenic exposure, the potential for adverse health effects from drinking water exposure to arsenic is very low for most municipal drinking water systems.</p>
<p>Use of Measure</p>	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to arsenic at different concentrations. • Maximum concentrations provide information on the peak potential exposure to arsenic at the state level. • Mean concentrations at the CWS level provide information on potential

	exposure at a smaller geographic scale.
Limitations of The Measure	Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	<p>Samples are taken once a year (surface sources), once every three years (groundwater sources), or once every nine years (for sources with a waiver). Frequency of sampling is based on compliance with the MCL; the lower the measured concentration the fewer samples will be taken and some years there may be no sampling for arsenic.</p> <p>Ground water systems may have multiple wells with different arsenic concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the arsenic concentration of people served by wells with higher arsenic concentrations.</p> <p>Exposure may be higher or lower than estimated if data from multiple entry points for water with different arsenic levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. Ahmad SA, Sayed MH, Barua S, Khan MH, Faruquee MH, Jalil A, Hadi SA, Talukder HK., 2001. Arsenic in drinking water and pregnancy outcomes. <i>Environmental Health Perspectives</i>; 109(6):629-31. 2. ATSDR 2005. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. Draft for Public comment. September 2005. Available at http://www.atsdr.cdc.gov/toxprofiles/tp2.html 3. Bunderson M, Brooks DM, Walker DL, Rosenfeld ME, Coffin JD, Beall HD., 2004. Arsenic exposure exacerbates atherosclerotic plaque formation and increases nitrotyrosine and leukotriene biosynthesis. <i>Toxicology and Applied Pharmacology</i> 2004 Nov 15;201(1):32-9. 4. Chen C-J, Chuang Y-C, Lin T-M, Wu H-Y. Malignant neoplasms among residents of a blackfoot disease-endemic area in Taiwan: high-arsenic well water and cancers. <i>Cancer Res.</i> 1985;45:5895–5899. 5. Chen Y., Factor-Litvak P., Howe GR., Graziano JH., Brandt-Rauf P., Parvez F., van Geen A., Ahsan H., 2007. Arsenic exposure from drinking water, dietary intakes of B vitamins and folate, and risk of high blood pressure in Bangladesh: a population-based, cross-sectional study. <i>American Journal of Epidemiology</i>, Mar 1;165(5):541-52 6. Chih-Hao Wang, Chuhsing Kate Hsiao, Chi-Ling Chen, Lin-I Hsu, Hung-Yi

Chiou, Shu- Yuan Chen, Yu-Mei Hsueh, Meei-Maan Wu, Chien-Jen Chen 2007. A review of the epidemiologic literature on the role of environmental arsenic exposure and cardiovascular diseases. *Toxicology and Applied Pharmacology* 222 (2007) 315–326.

7. Fazal MA, Kawachi T, Ichion E. Extent and severity of groundwater arsenic contamination in Bangladesh. *Water International* (2001) 26:370–79

8. Hopenhayn C, Ferreccio C, Browning SR, Huang B, Peralta C, Gibb H, Hertz-Picciotto I., 2003. Arsenic exposure from drinking water and birth weight. *Epidemiology.*;14(5):593-602.

9. IARC 2004. Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 84. Some Drinking Water Disinfectants and Contaminants, including Arsenic. September Lyon, pp. 39-267. Available at <http://monographs.iarc.fr/ENG/Monographs/vol84/volume84.pdf>

10. Kaltreider RC, Davis AM, Lariviere JP, Hamilton JW. 2001. Arsenic alters the function of the glucocorticoid receptor as a transcription factor. *Environ. Health Perspect* 109:245-251.

11. Kapaj S., Peterson H., Liber K., Bhattacharya P., 2006. Human health effects from chronic Arsenic poisoning-A review, *Journal of Environmental Science and Health, Part A*;41(10):2399-2428.

12. Karagas M, Stukel TA, Tosteson TD. 2002. Assessment of cancer risk and environmental levels of arsenic in New Hampshire. *Int. J. Hyg. Environ. Health* 205, 85-94.

13. NCI .2003. Ries LAG, Eisner MP, Kosary CL, Hankey BF, Miller BA, Clegg L, Mariotto A, Fay MP, Feuer EJ, Edwards BK (eds). *SEER Cancer Statistics Review, 1975-2000*, National Cancer Institute (NCI). Bethesda, MD, http://seer.cancer.gov/csr/1975_2000/, 2003. http://seer.cancer.gov/csr/1975_2000/results_single/sect_01_table.01.pdf

14. National Research Council. *Arsenic in Drinking Water*, National Academy Press. 1999.

15. Rahman M., Tondel M., Ahmad SA., Axelson O., 1998. Diabetes Mellitus associated with Arsenic exposure in Bangladesh. *American Journal of Epidemiology*, Vol 48, no2, pp 198-203.

16. Rahman MM, Sengupta MK, Ahamed S, et al. Arsenic contamination of groundwater and its health impact on residents in a village in West Bengal, India. *Bulletin of the World Health Organization* (2005) 83:49–57.

17. Salazar et al. 2004. p53 Expression in circulating lymphocytes of non-melanoma skin cancer patients from an arsenic contaminated region in Mexico. A pilot study. *Molecular and Cellular Biochemistry* 255:25-31.

18. U.S. EPA 2000a. Arsenic in drinking water proposal, 65 FR 38888 at 38894 and 38897, June 22, 2000, available at <http://www.epa.gov/fedrgstr/EPA-WATER/2000/June/Day-22/w13546.htm> and http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=2000_register&docid=00-13546-filed.pdf

19. U.S. EPA 2000b. Arsenic in Drinking Water Rule Economic Analysis December 2000, available at <http://www.epa.gov/safewater/arsenic/history.html>

- | | |
|--|---|
| | <p>20. U.S. EPA 2001a. Arsenic in drinking water final rule, 66 FR 6976. Available at http://www.epa.gov/fedrgstr/EPA-WATER/2001/January/Day-22/w1668.htm</p> <p>21. U.S. EPA 2001b. Quick Reference Guide for Arsenic. EPA 816-F-01-004. Available at http://www.epa.gov/safewater/arsenic/pdfs/quickguide.pdf</p> <p>22. U.S. EPA 2001c. Arsenic in drinking water Technical fact sheet. Available at http://www.epa.gov/ogwdw/arsenic/regulations_techfactsheet.html</p> <p>23. U.S. EPA 2004. Estimated per Capita Water Ingestion and Body Weight in the United States – an Update. EPA-822-R-00-001. October 2004. Available at http://epa.gov/waterscience/criteria/drinking/percapita/2004.pdf</p> <p>24. U.S. EPA 2005. Factoids: Drinking Water and Ground Water Statistics for 2004. EPA Office of Water. EPA 816-K-05-001. May 2005. Available at http://www.epa.gov/safewater/data/pdfs/data_factoids_2004.pdf</p> |
|--|---|

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: DI(2-ETHYLHEXYL)PHTHALATE (DEHP)
ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of Community Water Systems (CWS) by maximum DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP). 2. Yearly distribution of number of CWS by mean DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP). 3. Mean concentration of DEHP at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 4. Yearly distribution of number of people served by CWS by maximum DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP). 5. Yearly distribution of number of people served by CWS by mean DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP).
Derivation of Measures	DEHP measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	DEHP, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Di (2-ethylhexyl)phthalate and Public Health</p> <p>DEHP is the most commonly used of a group of related chemicals called phthalates or phthalic acid esters. Some people who drink water containing DEHP well in excess of the maximum contaminant level (MCL) for many years may have problems with their livers or could experience reproductive difficulties and may have an increased risk of getting cancer. (U.S.EPA, 2010)</p>

In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, DEHP was detected in 3,098 systems, which collectively serve more than 45 million people (EPA, 2009). Concentrations of DEHP were greater than the MCL in 460 systems serving 11.5 million people. DEHP was the highest occurring regulated synthetic organic chemical found based on the percent of detections found from the 6 Year Review data. This contamination could be due, in part, to sample contamination from older generation laboratory and field sampling equipment made of plastics that contained and released phthalates (EPA, 2009).

Most of what we know about the health effects of DEHP comes from studies of rats and mice given high amounts of DEHP. Brief oral exposure to very high levels of DEHP damaged sperm in mice. Although the effect reversed when exposure ceased, sexual maturity was delayed in the animals. High amounts of DEHP damaged the liver of rats and mice. Whether or not DEHP contributes to human kidney damage is unclear.

The Department of Health and Human Services has determined that DEHP may reasonably be anticipated to be a human carcinogen. The EPA has determined that DEHP is a probable human carcinogen. These determinations were based entirely on liver cancer in rats and mice. The International Agency for Research on Cancer has stated that DEHP cannot be classified as to its carcinogenicity to humans.

People are exposed through ingestion, inhalation, and, to a lesser extent, dermal contact with products that contain phthalates. For the general population, dietary sources have been considered as the major exposure route, followed by inhaling indoor air. Infants may have relatively greater exposures from ingesting indoor dust containing some phthalates (Clark et al., 2003). Human milk can be a source of phthalate exposure for nursing infants (Calafat et al., 2004; Mortensen et al., 2005). The intravenous or parenteral exposure route can be important in patients undergoing medical procedures involving devices or materials containing phthalates. In settings where workers may be exposed to higher air phthalate concentrations than the general population, urinary metabolite and air phthalate concentrations are roughly correlated (Liss et al., 1985; Nielsen et al., 1985; Pan et al., 2006). Phthalates are metabolized and excreted quickly and do not accumulate in the body (Anderson et al., 2001).

Biomonitoring Information

Four metabolites of DEHP were measured for the Fourth National Report on Human Exposure to Environmental Chemicals: mono-(2-ethyl-5-hexyl) phthalate (MEHP), mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP), mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP) and mono-(2-ethyl-5-carboxypentyl) phthalate (MECPP). MEHP is primarily formed by the hydrolysis of DEHP in the gastrointestinal tract and then absorbed. By contrast, DEHP present in medical devices and parenteral delivery systems results in the

diester parent compound, rather than the monoester metabolite. being directly introduced into the blood. After parenteral administration hydrolysis of DEHP most likely also occurs in the blood, and subsequent metabolism is similar to that following ingestion (Koch et al., 2005a, 2005b, 2005c). MEOHP, MEHHP, and MECPP are produced by the oxidative metabolism of MEHP and are present at roughly three- to five-fold higher concentrations than MEHP in urine (Barr et al., 2003; Fromme et al., 2007; Koch et al., 2003). MEHP is the putative toxic metabolite of DEHP. Liver toxicity, decreased testicular weight, and testicular atrophy have been observed in rodents fed high doses over a short term or with chronic dosing (McKee et al., 2004; NTP-CERHR, 2000c, 2006). In contrast, marmoset monkeys fed high dose DEHP for longer than a year did not demonstrate testicular or liver toxicity (NTP-CERHR, 2006). Very high doses of DEHP have suppressed estradiol production in female rats (Lovecamp-Swan and Davis, 2003). The U.S. Food and Drug Administration determined that in adults, the amounts of DEHP or MEHP received from intravenous delivery systems or blood transfusions (DEHP is hydrolyzed to MEHP in stored blood) would result in short-term elevations similar to background levels (FDA, 2001). However, critically ill neonates and infants receiving selected or multiple intensive procedures, such as exchange transfusions, extracorporeal membrane oxygenation, and parenteral nutrition, could receive higher exposures than the general population (Calafat et al., 2004; FDA, 2001; Loff et al., 2000; Weuve et al., 2006).

The levels of MEHP reported in NHANES 1999-2000, 2001-2002, and 2003-2004 appear roughly comparable to those reported previously in several small U.S. studies involving adults (Blount et al., 2000), pregnant women in New York City (Adibi et al., 2003), and low income African-American women in Washington, DC (Hoppin et al., 2002). In another sample of men attending an infertility clinic, the median and 95th percentile values of urinary MEHP were similar, but MEHHP and MEOHP were about three to five times higher than comparable values found in males in two NHANES survey periods (1999-2000, 2001-2002) (CDC, 2005; Hauser et al., 2007). In separate analyses of NHANES 1999-2000 and NHANES 2001-2002, the adjusted geometric mean levels of urinary MEHP were significantly higher in children compared with adolescents and adults, and in females compared with males (CDC, 2005; Silva et al., 2004). Studies of hospitalized neonates have reported urinary geometric mean levels of MEHP, MEOHP, and MEHHP that were two to five times higher, or more (depending on the intensity of DEHP-product exposure), than the geometric means of children in the NHANES subsamples for all three survey periods (Calafat et al., 2004; Weuve et al., 2006). Small studies of plasma and platelet donors have reported very high levels of MEHP, MEOHP, MEHHP and MECPP in urine collected shortly after these procedures (Koch et al., 2005b, 2005c). Finding a measurable amount of one or more DEHP metabolites in urine does not mean that the levels of the metabolites or the parent compound cause an adverse health effect. Biomonitoring studies on levels of urinary DEHP metabolites provide physicians and public health

	<p>officials with reference values so that they can determine whether people have been exposed to higher levels of DEHP than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.</p> <p>Sources of DEHP</p> <p>Phthalates are industrial chemicals, often called <i>plasticizers</i>, that are added to plastics make them more flexible and resilient. Phthalates are also used in other applications as solubilizing and stabilizing agents. Numerous products contain phthalates: adhesives; automotive plastics; detergents; lubricating oils; some medical devices and pharmaceuticals; plastic raincoats; solvents; vinyl tiles and flooring; and personal-care products, such as soap, shampoo, deodorants, lotions, fragrances, hair spray, and nail polish. Phthalates are often used in polyvinyl chloride-type plastics, such as plastic bags, garden hoses, inflatable recreational toys, blood product storage bags, intravenous medical tubing, and toys (ATSDR, 2001, 2002). Because they are not chemically bound to the plastics to which they are added, phthalates can be released into the environment during use or disposal of the product. Various phthalate esters have been measured in specific foods, indoor and ambient air, indoor dust, water sources, and sediments (Clark et al., 2003).</p> <p>DEHP is primarily used to produce flexibility in plastics, mainly polyvinyl chloride, which is used for many consumer products, toys, packaging film, and blood product storage and intravenous delivery systems. Concentrations in plastic materials may reach 40% by weight. DEHP has been removed from or replaced in most toys and food packaging in the United States. Following ingestion, DEHP is metabolized to more than 30 metabolites which are rapidly eliminated in urine, and in humans, as glucuronide conjugates (Albro et al., 1982; Albro and Lavenhar, 1989; ATSDR, 2002; Peck and Albro, 1982). The major source of di(2-ethylhexyl) phthalate in drinking water is discharge from rubber and chemical factories (U.S. EPA, 2010).</p> <p>DEHP Regulation and Monitoring</p> <p>The EPA limits the amount of DEHP that may be present in drinking water to 6 parts of DEHP per billion parts of water (6 ppb), or 6 ug/L.</p> <p>The Occupational Safety and Health Administration (OSHA) sets a maximum average of 5 milligrams of DEHP per cubic meter of air (5 mg/m³) in the workplace during an 8-hour shift. The short-term (15-minute) exposure limit is 10 mg/m³.</p>
<p>Use of Measure</p>	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the

	<p>number of people potentially exposed to DEHP at different concentrations.</p> <ul style="list-style-type: none"> • Maximum concentrations provide information on the peak potential exposure to DEHP at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells may be another source of population exposure to DEHP. Transient non-community water systems, which are regulated by EPA, may also be an important source of DEHP exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Ground water systems may have many wells with different DEHP concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the DEHP concentration of people served by wells with higher DEHP concentrations.</p> <p>Exposure may be higher or lower than estimated if data from multiple entry points for water with different DEHP levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. Adibi JJ, Perera FP, Jedrychowski W, Camann DE, Barr D, Jacek R, et al. Prenatal exposures to phthalates among women in New York City and Krakow, Poland. <i>Environ Health Perspect</i> 2003;111(14):1719-1722. 2. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for di-n-butyl phthalate update [online]. 2001. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp135.html. 4/20/09 3. Agency for Toxic Substances and Disease Registry (ATSDR). 2002. Toxicological Profile for Di(2-ethylhexyl) phthalate. Update. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. 4. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for di(2-ethylhexyl)phthalate update [online]. 2002. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp9.html. 4/20/09. 5. Albro PW, Corbett JT, Schroeder JL, Jordan S, Matthews HB. Pharmacokinetics, interactions with macromolecules and species differences in metabolism of DEHP. <i>Environ Health Perspect</i> 1982;45:19-25. 6. Albro PW and Lavenhar SR. Metabolism of di(2-ethylhexyl) phthalate. <i>Drug Metab Rev</i> 1989;21:13-34. 7. Anderson WA, Castle L, Scotter MJ, Massey RC, Springall C. A biomarker approach to measuring human dietary exposure to certain phthalate diesters. <i>Food</i>

Addit Contam 2001;18(12):1068- 1074.

8. Barr DB, Silva MJ, Kato K, Reidy JA, Malek NA, Hurtz D, et al. Assessing human exposure to phthalates using monoesters and their oxidized metabolites as biomarkers. *Environ Health Perspect* 2003;111(9):1148-1151.
9. Blount BC, Silva MJ, Caudill SP, Needham LL, Pirkle JL, Sampson EJ, et al. Levels of seven urinary phthalate metabolites in a human reference population. *Environ Health Perspect* 2000;108(10):979-982.
10. Calafat AM, Slakman AR, Silva MJ, Herbert AR, Needham LL. Automated solid phase extraction and quantitative analysis of human milk for 13 phthalate metabolites. *J Chromatogr B* 2004;805:49-56.
11. Calafat AM, Needham LL, Silva MJ, Lambert G. Exposure to di- (2-ethylhexyl) phthalate among premature neonates in a neonatal intensive care unit. *Pediatrics* 2004;113:e429-e434.
12. Centers for Disease Control and Prevention (CDC). Third National Report on Human Exposure to Environmental Chemicals. Atlanta (GA). 2005.
13. Clark K, Cousins IT, Mackay D. Assessment of critical exposure pathways. In Staples CA (ed), *The Handbook of Environmental Chemistry, Vol.3, Part Q: Phthalate Esters*. 2003;New York, Springer, pp. 227-262.
14. Food and Drug Administration (FDA). Safety assessment of di(2-ethylhexyl)phthalate (DEHP) released from PCV medical devices. 2001 [online]. Available at URL: <http://www.fda.gov/cdrh/ost/dehp-pvc.pdf>. 4/20/09
15. Fromme H, Bolte G, Koch HM, Angerer J, Boehmer S, Drexler H, et al. Occurrence and daily variation of phthalate metabolites in the urine of an adult population. *Int J Hyg Environ Health* 2007;210:21-33.
16. Hauser R, Meeker JD, Singh NP, Silva MJ, Ryan L, Duty S, et al. DNA damage in human sperm is related to urinary levels of phthalate monoester and oxidative metabolites. *Hum Reprod* 2007; 22(3):688-695.
17. Hoppin JA, Brock JW, Davis BJ, Baird DD. Reproducibility of urinary phthalate metabolites in first morning urine samples. *Environ Health Perspect* 2002; 110(5):515-518.
18. Koch HM, Bolt HM, Preuss R, Angerer J. New metabolites of di(2-ethylhexyl)phthalate (DEHP) in human urine and serum after single oral doses of deuterium-labeled DEHP. *Arch Toxicol* 2005a; 79:367-376.
19. Koch HM, Bolt HM, Preuss R, Eckstein R, Weisback V, Angerer J. Intravenous exposure to di(2-ethylhexyl)phthalate (DEHP) : metabolites of DEHP in urine after a voluntary platelet donation. *Arch Toxicol* 2005b; 79:689-693.
20. Koch HM, Angerer J, Drexler H, Eckstein R, Weisbach V. Di(2-ethylhexyl)phthalate (DEHP) exposure of voluntary plasma and platelet donors. *Int J Hyg Environ Health* 2005c;208:489-498. Koch HM, Drexler H, Angerer J. Internal exposure of nursery- school children and their parents and teachers to di(2-ethylhexyl) phthalate (DEHP). *Int J Hyg Environ Health*. 2004; 207:15-22.
21. Koch HM, Rossbach B, Drexler H, Angerer J. Internal exposure of the general population to DEHP and other phthalates - determination of secondary and primary phthalate monoester metabolites in urine. *Environ Res* 2003; 93:177-185.
22. Liss GM, Albro PW, Hartle RW, Stringer WT. Urine phthalate determinations as an index of occupational exposure to phthalic anhydride and di (2-ethylhexyl) phthalate. *Scand J Work Environ Health* 1985;11(5):381-387.
23. Loff S, Kabs F, Witt K, Sartoris J, Mandl B, Niessen KH, et al. Polyvinylchloride infusion lines expose infants to large amounts of toxic plasticizers. *J Pediatr Surg* 2000; 35(12):1775-1781.
24. Lovekamp-Swan T, Davis BJ. Mechanisms of phthalate ester toxicity in the

female reproductive system. *Environ Health Perspect* 2003; 111(2):139-145.

25. McKee RH, Butala JH, David RM, Gans G. NTP center for the evaluation of risks to human reproduction reports on phthalates: addressing the data gaps [review]. *Reprod Toxicol* 2004; 18(1):1- 22.

26. Mortensen GK, Main KM, Andersson A-M, Leffers H, Skakkebaek NE. Determination of phthalate monoesters in human milk, consumer milk, and infant formula by tandem mass spectrometry (LC-MS-MS). *Anal Bioanal Chem* 2005;382:1084- 1092.

27. Nielsen J, Akesson B, Skerfving S. Phthalate ester exposure—air levels and health of workers processing polyvinylchloride. *Am Ind Hyg Assoc J* 1985;46(11):643-647.

28. NTP-CERHR. National Toxicology Program-Center for the Evaluation of Risks to Human Reproduction. Monograph on the Potential Human Reproductive and Developmental Effects of Di (2-ethylhexyl) Phthalate (DEHP). Research Triangle Park (NC). 2000c [online]. Available at URL: <http://cerhr.niehs.nih.gov/chemicals/dehp/dehp-eval.html>. 6/2/09

29. NTP-CERHR. National Toxicology Program-Center for the Evaluation of Risks to Human Reproduction. Draft Update Monograph on the Potential Human Reproductive and Developmental Effects of Di (2-ethylhexyl) Phthalate (DEHP). Research Triangle Park (NC). 2006 [online]. Available at URL: <http://cerhr.niehs.nih.gov/chemicals/dehp/dehp-eval.html>. 6/2/09

30. Pan G, Hanaoka T, Yoshimura M, Zhang S, Wang P, Tsukino H, et al. Decreased serum free testosterone in workers exposed to high levels of di-n-butyl phthalate (DBP) and di-2-ethylhexylphthalate (DEHP): a cross-sectional study in China. *Environ Health Perspect* 2006; 114(11):1643-1648.

31. Peck CC, Albro PW. Toxic potential of the plasticizer di (2-ethylhexyl) phthalate in the context of its disposition and metabolism in primates and man. *Environ Health Perspect* 1982; 45:11-17.

32. Silva MJ, Barr DB, Reidy JA, Malek NA, Hodge CC, Caudill SP, et al. Urinary levels of seven phthalate metabolites in the U.S. population from the National Health and Nutrition Examination Survey (NHANES) 1999-2000 [published erratum appears in *Environ Health Perspect* 2004; 112(5):A270]. *Environ Health Perspect* 2004; 112(3):331-338.

33. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.

34. U.S. EPA, Basic Information about Di (2- ethylhexyl) phthalate in Drinking Water. 2010. http://water.epa.gov/drink/contaminants/basicinformation/di_2-ethylhexyl_phthalate.cfm

35. Weuve J, Sanchez GN, Calafat AM, Schettler T, Green RA, Hu H, et al. Exposure to phthalates in neonatal intensive care unit infants: urinary concentrations of monoesters and oxidative metabolites. *Environ Health Perspect* 2006; 114(9):1424-1431.

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: DISINFECTION BYPRODUCTS
ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Quarterly distribution of number of Community Water Systems (CWS) by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 2. Yearly distribution of number of CWS by maximum HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 3. Yearly distribution of number of CWS by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 4. Mean concentration of HAA5 at CWS-level, by year. 5. Quarterly distribution of number of CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 6. Yearly distribution of number of CWS by maximum TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 7. Yearly distribution of number of CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 8. Mean concentration of TTHM at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 9. Quarterly distribution of number of people served by CWS by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 10. Yearly distribution of number of people served by CWS by maximum HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 11. Yearly distribution of number of people served by CWS by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) mg/L HAA5). 12. Quarterly distribution of number of people served by CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 13. Yearly distribution of number of people served by CWS by maximum TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM). 14. Yearly distribution of number of people served by CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) mg/L TTHM).

	(>80-100), (>100) mg/L TTHM).
Derivation of Measures	Disinfection byproducts measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Trihalomethanes comprise chloroform, bromodichloromethane, dibromochloromethane, bromoform and their sum, denoted total trihalomethanes (TTHM). Haloacetic acids comprise trichloroacetic acid, dichloroacetic acid, monochloroacetic acid, dibromoacetic acid, monobromoacetic acid, and their sum, denoted HAA5. Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	concentration of HAA5, µg/L concentration of TTHM, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	2002 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Disinfection By Products and Public Health</p> <p>Disinfection byproducts (DBP) are formed when disinfectants used to inactivate microbial contaminants in water react with materials, primarily organic matter, in the water (Bellar et al. 1974, Rook 1974, Cedergren et al. 2002, Sadiq and Rodriguez 2004). Several hundred DBPs in over a dozen chemical classes have been identified (Woo et al. 2002, Krasner et al. 2006). Most commonly, DBPs form when chlorine reacts with naturally occurring organic matter in the source water.</p> <p>DBPs have been associated with both cancer and adverse pregnancy outcomes. High DBP levels, mainly for THMs, have been linked to bladder, colon and rectal cancer (King and Marrett 1996, Cantor et al. 1998, Amy et al. 2005, Villanueva et al. 2004, Villanueva et al. 2007), with bladder cancer reported most frequently. Although findings about adverse pregnancy outcomes have been less definitive, DBPs have been implicated in fetal loss (Swan et al. 1998, Waller et al. 1998, King et al. 2000, Dodds et al. 2004) and a variety of adverse birth outcomes involving growth (Bove et al. 1995, Gallagher et al. 1998, Wright et al. 2004, Infante-Rivard 2004, Toledano et al. 2005) and birth defects (Dodds et al. 1999, Klotz and Pyrch 1999, Dodds and King 2001, Cedergren et</p>

al. 2002, Shaw et al. 2003). In contrast, however, other research has found little effect on birth outcomes (Savitz et al., 2006).

Animal, microbial, in vitro and modeling studies have also pointed to toxicity or carcinogenicity of a wide variety of DBPs (Boorman 1999, Komulainen 2004). Numerous studies have indicated that different DBPs among the THMs and HAAs have different health effects. A number of studies have suggested that iodinated and brominated DBPs are more toxic than their chlorinated counterparts (Plewa et al. 2002, 2004, Richardson 2005). It is therefore appropriate that the tracking network follow individual DBP species and not just class totals (*c.f.* Singer 2006).

Sources of DBPs

DPB levels tend to be highest in water derived from surface sources because ground water generally has little organic matter (Symons et al. 1975, Whitaker et al. 2003). Ground water can, however, produce relatively high levels of the more brominated DBPs when the water, due either to geological circumstances (Whitaker et al. 2003) or salt water intrusion in coastal areas (von Gunten 2003), has elevated levels of bromide.

Bromate and chlorite are formed primarily after disinfection by ozone and chlorine dioxide, respectively. Sampling for these DBPs is required only for treatment plants that use the disinfectants that form them. Ozonation and chlorine dioxide are less common mechanisms of disinfection so these two DBPs will not be tracked initially. The disinfection processes that produce these two byproducts are likely to be used more often in the future so bromate and chlorite should be considered for eventual incorporation into the tracking network.

DBP Regulation and Monitoring

Safe Drinking Water Act (SDWA) regulation of DBPs began with the 1979 Total Trihalomethane Rule. This rule set an interim MCL for total trihalomethanes (TTHM), defined as the sum of four trihalomethanes, of 0.10 mg/L for community water systems (CWS) serving 10,000 or more people and using a disinfectant. The Stage 1 Disinfectants and Disinfection Byproducts Rule of 1998 (US EPA 1998) reduced the MCL for TTHM to 0.080 mg/L, added MCLs for the sum of five haloacetic acids (HAA5) of 0.060 mg/L, bromate of 0.010 mg/L and chlorite of 1.0 mg/L, and increased the scope of the rule to cover all CWS that disinfect. The rule had phased compliance with a date of 1 January 2002 for public water systems (PWS) with 10,000 or more people with a surface water or ground water under direct influence source and a date of 1 January 2004 for all other affected PWSs. The Stage 2 Disinfectants and Disinfection Byproducts Rule of 2006 (US EPA 2006) did not alter MCLs but did change how compliance with MCLs will be calculated and requires that PWSs evaluate their distribution systems for appropriate sampling locations. The results of this evaluation may affect the number and location of samples.

	<p>The scope of the rule also increased to cover consecutive systems that receive finished water from other systems. The first reporting deadline for compliance with the Stage 2 rule was in 2006 but it will be a number of years before the rule requires the new compliance calculations based on routine DBP samples.</p> <p>Currently, therefore, Safe Drinking Water Act standards exist for two classes of halogenated organic DBPs, trihalomethanes (THM) and haloacetic acids (HAA), and for two inorganic compounds, bromate and chlorite (US EPA, 2007). Given the near ubiquity of chlorine disinfection, the THMs and HAAs are useful indicators of risk for other DBPs because they occur at high levels and are easily measured.</p> <p>In summary, evidence suggests that disinfection byproducts adversely affect human health. The THMs and HAAs are the most commonly formed DBPs that are routinely tracked in state Safe Drinking Water Act databases. Measures based on these contaminants thus provide a window into potential human exposure to DBPs in publicly provided drinking water. They show where people are potentially exposed to high levels of DBPs. These water supply systems are candidates for enhancement of source water quality, infrastructure improvements or other interventions to reduce DBP exposure.</p>
Use of Measure	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to nitrate at different concentrations. • Maximum concentrations provide information on the peak potential exposure to nitrate at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Transient non-community water systems, which are regulated by EPA, may also be an important source of DBPs exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Safe Drinking Water Act compliance data include only a handful of the hundreds of known DBPs (Weinberg et al. 2002), most of which occur in chemical classes other than THMs and HAAs. While compliance sampling for THMs and HAAs is directed at the DBPs thought to be most commonly produced by chlorination, non-regulated DBPs exist even among the THMs and HAAs.</p>

Concern has also been expressed about iodinated THMs and HAAs which, while present in lower concentrations than the brominated and chlorinated THMs, are thought to be toxic at lower doses (e.g. Plewa et al. 2004).

THMs and HAAs may not be the most satisfactory indicators of DBP levels in waters subject to alternative disinfection methods that produce different DBPs in different proportions than chlorination (Richardson 2002, Weinberg et al. 2002) and may result in high levels of unregulated DBPs. Little is known about the quantitative occurrence of these DBPs in the distribution system (Richardson et al. 2002, Krasner et al. 2006). While the health effects of different DBPs may vary, with some suspected to be hazardous, few have been characterized for their effects on human health (Woo et al. 2002).

Correlations among different DBPs can be relatively low (King et al. 2004, Rodriguez et al. 2004a) so that the measured concentrations of THMs and HAAs may not be good predictors of exposure to other DBPs or overall DBP exposure. THM4 or HAA5, which are the only available data in some state databases, may therefore tell little about the relative concentrations of the THMs or HAAs.

DBP levels vary seasonally (Singer et al. 1981, Whitaker et al. 2003, Rodriguez et al. 2004b). Quarterly samples may not capture maximum levels and may not even adequately reflect short term levels. They may therefore be inadequate for estimating exposure during critical periods of a pregnancy, which may be as short as two to three weeks, especially if peak exposure matters more than average exposure. Furthermore, these fluctuations make it difficult to characterize levels with a single number such as an annual average and thus pose challenges to the development of meaningful synopses of patterns and trends.

DBP levels are spatially and temporally labile within a distribution system (Rodriguez et al. 2004b). THM levels increase with time after disinfection and therefore with distance from the treatment plant (Chen and Weisel 1998, Rodriguez and Sérodes 2001). HAA levels may increase or decrease (Chen and Weisel 1998, Rodriguez et al. 2004b), depending upon distribution system conditions. Rechlorination further increases DBP levels. For all but small distribution systems it is therefore impossible to adequately characterize DBP levels with a single value. DBP sampling locations may change over time, making it more difficult to compare measurements from year to year. Better estimation of DBP levels will require spatial and hydraulic modeling of distribution systems.

Water supply systems sample for DBPs on different schedules that range from quarterly to triennially. Different sampling frequencies complicate comparisons among different water supply systems. Long intervals between samples, although allowed only where THM and HAA levels have been found to be well under the MCL, create greater uncertainty about levels between sampling dates

	<p>and require stronger assumptions when estimating exposure during short term events such as pregnancies. When allowed, annual or triennial monitoring takes place during the month of warmest weather and may therefore overestimate average DBP levels.</p> <p>Water supply systems that have disinfection waivers generally have no DBP sample results. While the default assumption that these water supply systems have DBP concentrations of zero is generally reasonable, low levels of DBPs can be found in raw ground water, e.g., from surface contamination or from movement of chlorinated water from onsite wastewater treatment systems into ground water.</p> <p>Human behavior greatly influences exposure, complicating efforts to estimate exposure from tap water measurements (Nieuwenhuijzen et al. 2000, Kaur et al. 2004, Nuckols et al. 2005). Among the influences on exposure are showering and bathing time, consumption of tap water, use of bottled water, and exposure to water at workplaces or other locations outside the home. Moreover, ascertaining DBP levels in drinking water does not address other routes of exposure such as swimming (Villanueva et al. 2007, Zwiener et al. 2007). This consideration is not strictly a limitation of the measure but pertains to using the measure as an indicator of exposure.</p> <p>Some state SDWA databases may contain only totals for THMs and HAAs and may not record sample results for individual DBPs. Measures involving individual THMs and HAAs cannot be calculated for these states.</p>
<p>Related Indicators</p>	<p>Public Water Use</p>
<p>References</p>	<ol style="list-style-type: none"> 1. Amy G., G. Craun, S. W. Krasner, K. P. Cantor, M. Hildesheim, P. Weyer, and W. D. King. 2005. Improved exposure assessment on existing cancer studies. Awwa Research Foundation, Denver, Colorado. 2. Bellar, T. A., J. J. Lichtenberg, and R. C. Kroner. 1974. The occurrence of organohalides in chlorinated drinking waters. <i>Journal American Water Works Association</i> 66:703-706. 3. Boorman, G. A., V. Dellarco, J. K. Dunnick, R. E. Chapin, S. Hunter, F. Hauchman, H. Gardner, M. Cox, and R. C. Sills. 1999. Drinking water disinfection byproducts: Review and approach to toxicity evaluation. <i>Environmental Health Perspectives</i> 107 (suppl 1):207-217. 4. Bove, F. J., M. C. Fulcomer, J. B. Klotz, J. Esmart, E. M. Dufficy, and J. E. Savrin. 1995. Public drinking water contamination and birth outcomes. <i>American Journal of Epidemiology</i> 141:850-862. 5. Cantor, K. P., C. F. Lynch, M. Hildesheim, M. Dosemeci, J. Lubin, M. Alavanja, and G. Craun. 1998. Drinking water source and chlorination byproducts I. Risk of bladder cancer. <i>Epidemiology</i> 9:21-28. 6. Cedergren, M. I., A. J. Selbing, O. Lofman, and B. A. J. Kallen. 2002. Chlorination byproducts and nitrate in drinking water and risk for congenital cardiac defects. <i>Environmental Research</i> 89:124-130. 7. Chen, W. J., and C. P. Weisel. 1998. Halogenated DBP concentrations in a distribution system. <i>Journal American Water Works Association</i> 90:151-163. 8. Dodds, L., W. King, C. Woolcott, and J. Pole. 1999. Trihalomethanes in public

- water supplies and adverse birth outcomes. *Epidemiology* 10:233-237.
9. Dodds, L., and W. D. King. 2001. Relation between trihalomethane compounds and birth defects. *Occupational & Environmental Medicine* 58:443-446.
 10. Dodds, L., W. D. King, A. C. Allen, B. A. Armson, D. B. Fell, and C. Nimrod. 2004. Trihalomethanes in public water supplies and risk of stillbirth. *Epidemiology* 15:179-186.
 11. Gallagher, M. D., J. R. Nuckols, L. Stallones, and D. A. Savitz. 1998. Exposure to trihalomethanes and adverse pregnancy outcomes. *Epidemiology* 9:484-489.
 12. Infante-Rivard, C. 2004. Drinking water contaminants, gene polymorphisms, and fetal growth. *Environmental Health Perspectives* 112:1213-1216.
 13. Kaur, S., M. J. Nieuwenhuijsen, and P. Steer. 2004. Exposure of pregnant women to tap water related activities. *Occupational and Environmental Medicine* 61:454-460.
 14. King, W. D., L. Dodds, and A. C. Allen. 2000. Relation between stillbirth and specific chlorination by-products in public water supplies. *Environmental Health Perspectives* 108:883-886.
 15. King, W. D., L. Dodds, B. A. Armson, A. C. Allen, D. B. Fell, and C. Nimrod. 2004. Exposure assessment in epidemiologic studies of adverse pregnancy outcomes and disinfection byproducts. *Journal of Exposure Analysis and Environmental Epidemiology* 14:466-472.
 16. King, W. D., and L. D. Marrett. 1996. Case-control study of bladder cancer and chlorination by-products in treated water (Ontario, Canada). *Cancer Causes & Control* 7:596-604.
 17. Klotz, J. B. and L. A. Pynch. 1999. Neural tube defects and drinking water disinfection by-products. *Epidemiology* 10:383-90.
 18. Komulainen, H. 2004. Experimental cancer studies of chlorinated by-products. *Toxicology* 198(1-3):239-48.
 19. Krasner, S. W., H. S. Weinberg, S. D. Richardson, S. J. Pastor, R. Chinn, M. J. Scrimanti, G. D. Onstad, and A. D. Thruston, Jr. 2006. The occurrence of a new generation of disinfection byproducts. *Environmental Science & Technology* 40:7175-7185.
 20. Nieuwenhuijsen, M. J., M. B. Toledano, and P. Elliott. 2000. Uptake of chlorination disinfection by-products; a review and a discussion of its implications for exposure assessment in epidemiological studies. *Journal of Exposure Analysis & Environmental Epidemiology* 10:586-599.
 21. Nuckols, J. R., D. L. Ashley, C. Lyu, S. M. Gordon, A. F. Hinckley, and P. Singer. 2005. Influence of tap water quality and household water use activities on indoor air and internal dose levels of trihalomethanes. *Environmental Health Perspectives* 113:863-870.
 22. Plewa, M. J., Y. Kargalioglu, D. Vankerk, R. Minear, and E. D. Wagner. 2002. Mammalian cell cytotoxicity and genotoxicity analysis of drinking water disinfection by-products. *Environmental & Molecular Mutagenesis* 40:134-142.
 23. Plewa, M. J., E. D. Wagner, S. D. Richardson, A. D. Thruston, Jr., Y.-T. Woo, and A. B. McKague. 2004. Chemical and biological characterization of newly discovered iodoacid drinking water disinfection byproducts. *Environmental Science & Technology* 38:4713-4722.
 24. Richardson, S. D. 2005. New disinfection by-product issues: Emerging DBPs and alternative routes of exposure. *Global NEST Journal* 7:43-60.
 25. Richardson, S. D., J. E. Simmons, and G. Rice. 2002. Disinfection byproducts: The next generation. *Environmental Science & Technology* 36:198A-205A.
 26. Roberts, M. G., P. C. Singer, and A. Obolensky. 2002. Comparing total HAA and

total THM concentrations using ICR data. *Journal American Water Works Association* 94:103-114.

27. Rodriguez, M. J., and J. B. Serodes. 2001. Spatial and temporal evolution of trihalomethanes in three water distribution systems. *Water Research* 35:1572-1586.
28. Rodriguez, M. J., M. Huard, and J. B. Sérodes. 2004a. Experimental study of the formation of chlorination by-products in potable water of Quebec City, Canada. *Bulletin of Environmental Contamination & Toxicology* 72:211-218.
29. Rodriguez, M. J., J. B. Sérodes, and P. Levallois. 2004b. Behavior of trihalomethanes and haloacetic acids in a drinking water distribution system. *Water Research* 38:4367-4382.
30. Rook, J. J. 1974. Formation of haloforms during chlorination of natural waters. *Water Treatment and Examination* 23:234-243.
31. Sadiq, R., and M. J. Rodriguez. 2004. Disinfection by-products (DBPs) in drinking water and predictive models for their occurrence: a review. *Science of the Total Environment* 321:21-46.
32. Savitz, D. A., P. C. Singer, A. H. Herring, K. E. Hartmann, H. S. Weinberg, and C. Makarushka. 2006. Exposure to drinking water disinfection by-products and pregnancy loss. *American Journal of Epidemiology* 164:1043-1051.
33. Shaw, G. M., D. Ranatunga, T. Quach, E. Neri, A. Correa, and R. R. Neutra. 2003. Trihalomethane exposures from municipal water supplies and selected congenital malformations. *Epidemiology* 14:191-199.
34. Singer, P. C. 2006. DBPs in drinking water: Additional scientific and policy considerations for public health protection. *Journal American Water Works Association* 98:73-80.
35. Singer, P. C., J. J. Barry III, G. M. Palen, and A. E. Scrivner. 1981. Trihalomethane formation in North Carolina drinking waters. *Journal American Water Works Association* 73:392-401.
36. Swan, S. H., K. Waller, B. Hopkins, G. Windham, L. Fenster, C. Schaefer, and R. R. Neutra. 1998. A prospective study of spontaneous abortion: relation to amount and source of drinking water consumed in early pregnancy. *Epidemiology* 9:126-133.
37. Symons, J. M., T. A. Bellar, J. J. Carswell, J. DeMarco, K. L. Kropp, G. G. Robect, D. R. Seeger, C. J. Slocum, B. L. Smith, and A. A. Stevens. 1975. National organics reconnaissance survey for halogenated organics. *Journal American Water Works Association* 67:634-647.
38. Toledano, M. B., M. J. Nieuwenhuijsen, N. Best, H. Whitaker, P. Hambly, C. de Hoogh, J. Fawell, L. Jarup, and P. Elliott. 2005. Relation of trihalomethane concentrations in public water supplies to stillbirth and birth weight in three water regions in England. *Environmental Health Perspectives* 113:225-232.
39. US EPA (United States Environmental Protection Agency). 1998. Stage 1 Disinfectants and Disinfection byproducts rule. *Federal Register* 63:69389-69476.
40. US EPA (United States Environmental Protection Agency). 2006. Stage 2 Disinfectants and Disinfection byproducts rule. *Federal Register* 71:387-493.
41. US EPA (United States Environmental Protection Agency). 2007. National Water Program Fiscal Year 2007 Guidance, Appendix A: Program Activity Measures Supporting the National Water Program for FY 2007. US EPA Web site at http://www.epa.gov/ocfo/npmguidance/owater/2007/2007_appendixa.ppt (accessed 18/15/07).
42. Villanueva, C. M., K. P. Cantor, S. Cordier, J. J. K. Jaakkola, W. D. King, C. F. Lynch, S. Porru, and M. Kogevinas. 2004. Disinfection byproducts and bladder

- cancer: A pooled analysis. *Epidemiology* 15:357-367.
43. Villanueva, C. M., K. P. Cantor, J. O. Grimalt, N. Malats, D. Silverman, A. Tardon, R. Garcia-Closas, C. Serra, A. Carrato, G. Castano-Vinyals, R. Marcos, N. Rothman, F. X. Real, M. Dosemeci, and M. Kogevinas. 2007. Bladder cancer and exposure to water disinfection by-products through ingestion, bathing, showering, and swimming in pools. *American Journal of Epidemiology* 135:148-156.
 44. von Gunten, U. 2003. Ozonation of drinking water: Part II. Disinfection and by-product formation in presence of bromide, iodide, or chlorine. *Water Research* 37:1469-1487.
 45. Waller, K., S. H. Swan, G. DeLorenze, and B. Hopkins. 1998. Trihalomethanes in drinking water and spontaneous abortion. *Epidemiology* 9:134-140.
 46. Weinberg H. S., S. W. Krasner, S. D. Richardson, and A. D. Thruston, Jr. 2002. The occurrence of disinfection by-products (DBPs) of health concern in drinking water: results of a nationwide DBP occurrence study; EPA/600/R-02/068. US EPA, Athens, Georgia.
 47. Whitaker, H., M. J. Nieuwenhuijsen, N. Best, J. Fawell, A. Gowers, and P. Elliot. 2003. Description of trihalomethane levels in three UK water suppliers. *Journal of Exposure Analysis and Environmental Epidemiology* 13:17-23.
 48. Woo, Y.-T., D. Lai, J. L. McClain, M. K. Manibusam, and V. Dellarco. 2002. Use of mechanism-based structure-activity relationships analysis in carcinogenic potential ranking for drinking water disinfection by-products. *Environmental Health Perspectives* 110 (suppl 1):75-87.
 49. Wright, J. M., J. Schwartz, and D. W. Dockery. 2004. The effect of disinfection by-products and mutagenic activity on birth weight and gestational duration. *Environmental Health Perspectives* 112:920-925.
 50. Zwiener, C., S. D. Richardson, D. M. De Marini, T. Grummt, T. Glauner, and F. H. Frimmel. 2007. Drowning in disinfection byproducts? Assessing swimming pool water. *Environmental Science & Technology* 41:363-372.

CONTENT DOMAIN: COMMUNITY WATER

INDICATOR: NITRATE

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 15. Quarterly distribution of number of Community Water Systems (CWS) by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 16. Yearly distribution of number of CWS by maximum nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 17. Yearly distribution of number of CWS by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 18. Mean concentration of nitrate at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 19. Quarterly distribution of number of people served by CWS by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 20. Yearly distribution of number of people served by CWS by maximum nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 21. Yearly distribution of number of people served by CWS by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate).
Derivation of Measures	Nitrate measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	Concentration of nitrate, mg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	Nitrates and Public Health

Nitrate was first identified as a public health threat in drinking water in 1945 when high nitrate levels from private wells were shown to cause methemoglobinemia or “blue baby syndrome” in infants who received formula made from well water. When an individual is exposed to nitrate it can be converted to nitrite (NO_2^-) in the body and then oxidize the ferrous iron (Fe^{+2}) in deoxyhemoglobin in the blood to form methemoglobin containing ferric iron (Fe^{+3}). Methemoglobin cannot transfer oxygen to tissues; thus nitrate or nitrite can starve the body of oxygen and produce a clinical condition known as cyanosis, where the lips and extremities turn gray or blue. Infants younger than four months of age are more sensitive than adults, and can develop “blue baby” syndrome from intake of nitrate higher than 10 mg/L nitrate or 45 mg/L nitrate–nitrogen. Blue baby syndrome is fatal in about ten percent of the cases (ATSDR, 2007). Usually there are no outward signs of cyanosis at methemoglobin levels below 20 percent (Dabney et al, 1990).

In addition, there is some evidence to suggest that exposure to nitrate in drinking water is also associated with adverse reproductive outcomes such as spontaneous abortions, intrauterine growth retardation, and various birth defects such as anencephaly, related to fetal exposures to nitrate. However, the evidence is inconsistent (Manassaram et al, 2006).

Similarly, long term exposure to higher nitrate levels in drinking water has been suggested as a risk factor for cancer. Cancer at several sites (i.e. gastric, colorectal, bladder, urothelial, brain, esophagus, ovarian and non-Hodgkins lymphoma) have been shown to be associated with nitrate in drinking water in some studies (Sandor et al, 2001; Weyer et al, 2001; Gulis et al, 2002; De Roos et al, 2003; Volkmer et al, 2005; Ward et al, 2005b; Chiu et al, 2007;). Other studies have not found any association (Ward et al, 2003; Ward et al, 2005, 2005c; Ward et al, 2006; Zeegers et al, 2006). Significant regional differences in cancer risk may occur (Mueller et al, 2001). Occupational exposures are also of concern as nitrate fertilizer workers have shown increased risk for stomach cancer (Zandjani et al. 1994).

Sources of Nitrate

Nitrate is the most commonly found contaminant in groundwater aquifers worldwide (Ward, 2005 from: Spalding and Exner 1993). Nitrate (NO_3^-) originates in drinking water from nitrate-containing fertilizers, sewage and septic tanks, and decaying natural material such as animal waste. Nitrate is very soluble in water, can easily migrate, and does not evaporate (EPA Consumer Fact Sheet). Anthropogenic sources of nitrates are increasing resulting in increased nitrate levels in water resources. Surface water and shallow wells in both rural and urban areas can be affected. Consequently, private wells are especially vulnerable to excess levels of nitrates. Excess levels of nitrate and nitrite can occur in community water supplies. A U.S. Geological Survey (USGS) study found nitrate levels exceeded regulatory monitoring standards in 2% of a sample of 242 public drinking water wells between 1992 and 1999 (Squillace et al, 2002). Levels of nitrates in private wells are less well known; private wells are not regularly monitored and are often more vulnerable to higher levels of nitrates because they draw water from shallower groundwater aquifers. The

	<p>USGS estimates approximately 22% of domestic wells in agricultural areas of the U.S. exceed the MCL (Ward, 2007).</p> <p>Nitrate Regulation and Monitoring</p> <p>Congress established the Safe Drinking Water Act in 1974, which set enforceable Maximum Contaminant Levels (MCLs) and non-enforceable Maximum Contaminant Level Goals (MCLGs) for certain specified contaminants. In the case of nitrate in drinking water, the MCLG of 10 mg/L (ppm) was established from human data from studies of methemoglobinemia in young children. (Johnson and Kross 1990; Walton, 1950). The MCL is also set at 10 ppm, and any exceedance of the MCL is potentially serious as there is no additional margin of safety between the MCLG and the MCL. (Ward, 2005). The MCLG and MCL for nitrite are 1 mg/L. While evidence to suggest MCL exposures for chronic health endpoints remains inconclusive, there is some evidence to suggest that chronic exposure to nitrate levels below the MCL may be of concern (Ward, 2005).</p>
Use of Measure	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to nitrate at different concentrations. • Maximum concentrations provide information on the peak potential exposure to nitrate at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells are another important source of population exposure to nitrate. Transient non-community water systems, which are regulated by EPA, may also be an important source of nitrate exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Nitrate levels can vary substantially in groundwater; thus high levels may not be captured by even quarterly sampling. Estimates of the number of people potentially exposed may be unreliable as they are based on estimates made by the water system operator. Concentrations in drinking water cannot be directly converted to exposure because overall water consumption, and the proportion of water consumed that comes from the tap is quite variable (EPA 2004). In systems that have more than one Entry point to the Distribution system, the actual nitrate level at any given house is a mixture of the levels from all contributing sources. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to</p>

	<p>underestimate the nitrate concentration of people served by wells with higher nitrate concentrations.</p> <p>Exposure may be higher or lower than estimated if data from multiple entry points for water with different nitrate levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use
References	<p>51. ATSDR Case Studies in Environmental Medicine: Nitrate/Nitrite Toxicity. http://www.atsdr.cdc.gov/HEC/CSEM/nitrate/index.html Downloaded 08/07/07</p> <p>52. Bosch, H. M., A. B. Rosenfield, R. Huston, H. R. Shipman, and F. L. Woodward. 1950. Methemoglobinemia and Minnesota well supplies. <i>Am. Water Works Assoc J</i> 42:161-170.</p> <p>53. Chiu HF, Tsai SS, Yang CY. 2007. Nitrate in drinking water and risk of death from bladder cancer: an ecological case-control study in Taiwan. <i>J Toxicol Environ Health A</i> 70(12):1000-1004.</p> <p>54. Coss A, Cantor KP, Reif JS, Lynch CF, Ward MH. 2004. Pancreatic cancer and drinking water and dietary sources of nitrate and nitrite. <i>Am J Epidemiol</i> 159(7):693-701.</p> <p>55. Dabney BJ, Zelarney PT, Hall AH. 1990. Evaluation and treatment of patients exposed to systemic asphyxiants. <i>Emerg Care Q</i> 6(3):65-80</p> <p>56. De Roos AJ, Ward MH, Lynch CF, Cantor KP. 2003. Nitrate in public water supplies and the risk of colon and rectum cancers. <i>Epidemiology</i> 14(6):640-649.</p> <p>57. Gulis G, Czompolyova M, Cerhan JR. 2002. An ecologic study of nitrate in municipal drinking water and cancer incidence in Trnava District, Slovakia. <i>Environ Res</i> 88(3):182-187.</p> <p>58. Johnson CJ and Kross BC. 1990. Continuing importance of nitrate contamination of groundwater and wells in rural areas. <i>Am J Ind Med</i> 18(4):449-456.</p> <p>59. Mueller BA, Newton K, Holly EA, Preston-Martin S. 2001. Residential water source and the risk of childhood brain tumors. <i>Environ Health Perspect</i> 109(6):551-556.</p> <p>60. Ruckart PZ, Henderson AK, Black ML, Flanders WD. 2007. Are nitrate levels in groundwater stable over time? <i>J Expo Sci Environ Epidemiol</i> Apr 11; [Epub ahead of print]</p> <p>61. Sandor J, Kiss I, Farkas O, Ember I. 2001. Association between gastric cancer mortality and nitrate content of drinking water: ecological study on small area inequalities. <i>Eur J Epidemiol</i> 17(5):443-447.</p> <p>62. U.S. Environmental Protection Agency Office of Water: Candidate Contaminants List. http://www.epa.gov/safewater/ccl/index.html Downloaded 08/02/07</p> <p>63. U.S. Environmental Protection Agency. Office of Water (4606) Occurrence Estimation Methodology and Occurrence Findings Report for the Six-Year Review of Existing National Primary Drinking Water Regulations. EPA-815-R-03-006 www.epa.gov June 2003. http://www.epa.gov/safewater/standard/review/pdfs/support_6yr_occurrencemethods_final.pdf Downloaded 08/02/07</p> <p>64. U.S. Environmental Protection Agency (2007b): Technical Factsheet on: Nitrate/Nitrite. http://www.epa.gov/safewater/dwh/t-ioc/nitrates.html Downloaded 08/07/07</p> <p>65. Volkmer BG, Ernst B, Simon J, Kuefer R, Bartsch G Jr, Bach D, Gschwend JE. 2005. Influence of nitrate levels in drinking water on urological malignancies: a community-based cohort study. <i>BJU Int</i> 95(7):972-976.</p> <p>66. Walton, G. 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. <i>Am J Public Health</i> 41:986-996.</p> <p>67. Ward MH, Cantor KP, Riley D, Merkle S, Lynch CF. 2003. Nitrate in public water</p>

	<p>supplies and risk of bladder cancer. <i>Epidemiology</i> 14(2):183-190.</p> <p>68. Ward MH, Heineman EF, McComb RD, Weisenburger DD. 2005. Drinking water and dietary sources of nitrate and nitrite and risk of glioma. <i>J Occup Environ Med</i> 47(12):1260-1267.</p> <p>69. Ward MH, deKok TM, Levallois P, Brender J, Gulis G, Nolan BT, VanDerslice J. 2005b. Workgroup report: Drinking-water nitrate and health--recent findings and research needs. <i>Environ Health Perspect</i> 113(11):1607-1614.</p> <p>70. Ward MH, Heineman EF, McComb RD, Weisenburger DD. 2005c. Drinking water and dietary sources of nitrate and nitrite and risk of glioma. <i>J Occup Environ Med</i> 47(12):1260-1267.</p> <p>71. Ward MH, Cerhan JR, Colt JS, Hartge P. 2006. Risk of non-Hodgkin lymphoma and nitrate and nitrite from drinking water and diet. <i>Epidemiology</i> 17(4):375-382.</p> <p>72. Weyer PJ, Cerhan JR, Kross BC, Hallberg GR, Kantamneni J, Breuer G, Jones MP, Zheng W, Lynch CF. 2001. Municipal drinking water nitrate level and cancer risk in older women: the Iowa Women's Health Study. <i>Epidemiology</i> 12(3):327-338.</p> <p>73. Zandjani F, Hogsæet B, Andersen A, Langard S. 1994. Incidence of cancer among nitrate fertilizer workers. <i>Int Arch Occup Environ Health</i> 66:189-93.</p> <p>74. Zeegers MP, Selen RF, Kleinjans JC, Goldbohm RA, van den Brandt PA. 2006. Nitrate intake does not influence bladder cancer risk: the Netherlands cohort study. <i>Environ Health Perspect</i> 114(10):1527-1531.</p>
--	---

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: PUBLIC WATER USE
ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Exposure
Measures	22. Number of people receiving water from community water systems.
Derivation of Measures	This measure will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format.
Units	1. Number of people
Geographic Scope	State
Geographic Scale	State
Time Period	2009 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Public Water Use and Public Health</p> <p>The public water use index provides some data to explore the relative importance of community water supplies as sources of drinking water and to provide context for subsequent community drinking water system (CWS) indicators. SDWA collects data for a number of different types of public water systems of which community water systems (CWS) are a sub-set. The community water systems represent non-transient public water systems that serve year round community residents and are the focus of the initial indicators. The range of state populations served by CWS as their primary residential drinking water source varies from 95% to as low as 40% within the United States. Understanding the relative population coverage of these indicators by state helps to understand representativeness of these data for prioritization and evaluation across the United States and within individual states and communities.</p>
Use of Measure	<p>This measure can be useful in providing data for surveillance purposes.</p> <ul style="list-style-type: none"> • Estimated population potentially exposed to contaminants in CWS.
Limitations of The Measure	The current measure is derived for CWS only. Private wells are another important source of population exposure to water contaminants. Transient non-community water systems, which are regulated by EPA, may also be an important source of potential exposure.
Data Sources	State grantee
Limitations of Data Sources	Population estimates are rough and may overestimate or underestimate the number of affected people.

Related Indicators	All other community water indicators.
Additional Information	<p>1. U.S. Environmental Protection Agency, <i>Water On Tap</i>, Office of Water (4601) EPA 816-K-09-002, December 2009. http://water.epa.gov/drink/guide/upload/book_waterontap_full.pdf</p> <p>2. U.S. Environmental Protection Agency, Public Drinking Water Systems: Facts and Figures http://water.epa.gov/infrastructure/drinkingwater/pws/factoids.cfm</p> <p>3. U.S. Environmental Protection Agency, Public Drinking Water Systems Programs. http://water.epa.gov/infrastructure/drinkingwater/pws/index.cfm</p>

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: COMBINED RADIUM-226 AND -228
ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of Community Water Systems (CWS) by maximum Radium concentration (cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium). 2. Yearly distribution of number of CWS by mean Radium concentration (cut-points: cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium). 3. Mean concentration of Radium at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 4. Yearly distribution of number of people served by CWS by maximum Radium concentration (cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium). 5. Yearly distribution of number of people served by CWS by mean Radium concentration (cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium).
Derivation of Measures	Combined Radium-226 and -228 measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	pCi/L combined Radium-226 & -228
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Radium-226 and -228 and Public Health</p> <p>Radium is a naturally occurring silvery-white radioactive metal that can exist in several forms called isotopes. Radium is produced constantly by the radioactive decay of uranium and thorium. Uranium and thorium are found in small amounts in most rocks and soil. Some of the radiation from radium is being released constantly into the environment. It is this radioactive decay that causes concern</p>

about the safety of radium and all other radioactive substances. Two of the main radium isotopes found in the environment are radium-226 and radium-228. The decay of radium-226 results in the formation of radon which exists as a gas and is mobile in environmental media. Radium has been used as a radiation source for treating cancer, in radiography of metals, and combined with other metals as a neutron source for research and radiation instrument calibration. Until the 1960s, radium was a component of the luminous paints used for watch and clock dials, instrument panels in airplanes, military instruments, and compasses (ATSDR, 2010).

Everyone is exposed to low levels of radium in the air, water, and food. Higher levels may be found in the air near industries that burn coal or other fuels or near sites that mine or mill uranium. It also may be found at higher levels in drinking water from groundwater wells. Miners, particularly miners of uranium and hard rock, are exposed to higher levels of radium. It may also be found at radioactive waste disposal sites (ATSDR, 1990).

It is not known whether long-term exposure to radium at the levels that are normally present in the environment (for example, 1 pCi of radium per gram of soil) is likely to result in harmful health effects. However, exposure to higher levels of radium over a long period of time may result in harmful effects including anemia, cataracts, fractured teeth, cancer (especially bone cancer), and death. Patients who were injected with radium in Germany, from 1946 to 1950, for the treatment of certain diseases including tuberculosis were significantly shorter as adults than people who were not treated. Some of these health effects may take years to develop and mostly are due to gamma radiation. Radium gives off gamma radiation, which can travel fairly long distances through air. Therefore, just being near radium at the high levels that may be found at some hazardous waste sites may be dangerous to your health.

Exposure to high levels of radium results in an increased incidence of bone, liver, and breast cancer. The EPA and the National Academy of Sciences, Committee on Biological Effects of Ionizing Radiation, has stated that radium is a known human carcinogen.

Biomonitoring Information

Urine tests can determine if you have been exposed to radium. Another test measures the amount of radon (a breakdown product of radium) in exhaled air. Both types of tests require special equipment and cannot be done in a doctor's office. These tests cannot tell how much radium you were exposed to, nor can they be used to predict whether you will develop harmful health effects (ATSDR, 1990). Levels of radium in the U.S. population are unknown.

Sources of Radium

Radium forms from the decay of uranium or thorium in the environment.

	<p>Radium -226 is formed from the decay of uranium-238; Radium-228 is formed from the decay of thorium. Radium is abundant in low levels everywhere because it originates from uranium which is commonly found in all rocks, soil and water. (EPA, 2010)</p> <p>Radium Regulation and Monitoring The EPA has set a drinking water limit of 5 picocuries per liter (5 pCi/L) for radium-226 and radium-228 (combined) (EPA, 2009). A gross alpha particle activity measurement may be substituted for the required radium-226 measurement provided that the measured gross alpha particle activity does not exceed 5 pCi/L. The EPA lifetime exposure cancer risk estimate for radium at the MCL, is approximately 1-2 cases per 10,000 people.</p> <p>Monitoring frequency Once a CWS has satisfied initial monitoring requirements (4 quarterly samples at every entry point to the distribution system within the first quarter after initiating the source); the required frequency for Combined Radium-226 and -228 monitoring is once every three years if the average of the initial monitoring results for the contaminant is greater than one-half the MCL but at or below the MCL. States may allow CWS to reduce the frequency of monitoring from once every three years to once every six or nine years at each sampling point, if the average of the initial monitoring results for each contaminant is below the detection limit. If a system has a monitoring result that exceeds the MCL while on reduced monitoring, the system must collect and analyze quarterly samples at that sampling point until the system has results from four consecutive quarters that are below the MCL, unless the system enters into another schedule as part of a formal compliance agreement with the State (CFR, 2002).</p>
<p>Use of Measure</p>	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to combined Radium-226 and -228 at different concentrations. • Maximum concentrations provide information on the peak potential exposure to combined Radium-226 and -228 at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
<p>Limitations of The Measure</p>	<p>The current measures are derived for CWS only. Private wells may be another source of population exposure to combined Radium-226 and -228. Transient non-community water systems, which are regulated by EPA, may also be an important source of combined Radium-226 and -228 exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating</p>

	populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	<p>The required monitoring frequency for combined Radium-226 and -228 is infrequent and may be as intermittent as every nine years; therefore most states will have very little data on this contaminant.</p> <p>Ground water systems may have multiple wells with different combined Radium-226 and -228 concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the combined Radium-226 and -228 concentrations of people served by wells with higher combined Radium-226 and -228 concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different combined Radium-226 and -228 levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use; Uranium
References	<ol style="list-style-type: none"> 1. Agency for Toxic Substances and Disease Registry (ATSDR). Toxic Substances Portal. Radium. 2010. Available at: http://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=154 2. Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for Radium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service Available at: http://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=790&tid=154 3. Code of Federal Regulations (CFR), 2002. Title 40 Protection of the Environment Chapter I--Environmental Protection Agency Part 141--National Primary Drinking Water Regulations 141.26 Monitoring frequency and compliance requirements for radionuclides in community water systems. Available at: URL: http://www.access.gpo.gov/nara/cfr/waisidx_02/40cfr141_02.html 4. U.S. Environmental Protection Agency (U.S. EPA). Radiation Protection, Radium, 2010. Available at: http://www.epa.gov/radiation/radionuclides/radium.html 5. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.

CONTENT DOMAIN: COMMUNITY WATER

INDICATOR: TETRACHLOROETHENE (TETRACHLOROETHYLENE) (PCE)

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 6. Yearly distribution of number of Community Water Systems (CWS) by maximum PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE). 7. Yearly distribution of number of CWS by mean PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE). 8. Mean concentration of PCE at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 9. Yearly distribution of number of people served by CWS by maximum PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE). 10. Yearly distribution of number of people served by CWS by mean PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE).
Derivation of Measures	PCE measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	PCE, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be the approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Tetrachloroethene (PCE) and Public Health</p> <p>Tetrachloroethene (PCE) is a volatile halogenated short-chain hydrocarbon. Tetrachloroethene is used in dry cleaning, metal cleaning, the synthesis of other chemicals, and household products such as water repellants, silicone lubricants, and spot removers. PCE is produced and used in high volumes in the U.S. and has been detected in urban and ambient air and occasionally in soils and drinking water most likely contaminated by industrial discharge (Moran et al., 2007; Rowe et al., 2007). Because of its volatility, this solvent does not persist in the soil or water following the discontinuation of contamination.</p>

Inhalation is the most common exposure route for the general population including indoor sources from paints, adhesives, and cleaning solutions. Volatilization from contaminated water (e.g., shower water) as well as the use of household products containing this solvent can result in higher indoor than outdoor air concentrations (ATSDR, 1997; Martin et al., 2005). Nearby dry cleaning establishments, industries producing PCE, and contaminated waste disposal sites can also contribute to human exposure (Armstrong and Green, 2004; ATSDR, 1997 and 2000; Schreiber et al., 1993; Wallace et al., 1991). Drinking water may contribute to exposure when underground drinking water supplies have been contaminated. Workers in industries such as dry cleaning, aircraft maintenance, electronics manufacturing, and chemical production may be exposed by inhalation or by dermal contact with PCE. The EPA has established drinking water standards and other environmental standards for PCE, and the FDA regulates PCE and trichloroethene as indirect food additives. Workplace standards have been established by OSHA, and ACGIH has recommended occupational guidelines and biological exposure indices for monitoring workers. Human health effects from PCE at low environmental doses or at biomonitored levels from low environmental exposures are unknown. PCE is well absorbed by ingestion and inhalation, and animal studies have demonstrated that liquid forms can be dermally absorbed. Following absorption, part of the solvent dose is excreted into expired air; for PCE, about 97-99% of the dose is eliminated unmetabolized into expired air, though it has an elimination half-life of several days (ATSDR 1997; Monster, 1986). The retained solvent can undergo hepatic metabolism. PCE is metabolized to trichloroacetic acid and trichloroethanol, which are eliminated in the urine. Accidental or intentional high dose acute exposure by ingestion or inhalation can result in loss of motor coordination, somnolence, and unconsciousness. Inhaling high doses of PCE may also produce cardiac arrhythmias attributed to enhanced sensitivity to catecholamines. High dose acute exposure to PCE has resulted in reversible kidney impairment, and prolonged, low level PCE exposure has been associated with altered renal enzyme excretion and liver enlargement (ATSDR, 1997). Chronic occupational exposure to PCE may be associated with mild degrees of neurological impairments, including reaction times, verbal skills, cognitive ability, and motor function (Armstrong and Green, 2004). Various epidemiologic studies of chronic PCE exposure in dry cleaning workers found increased incidences of esophageal and cervical cancers and non-Hodgkins lymphoma, but confounding exposures (e.g., other solvents and trichloroethene) were likely (IPCS, 2006). In animal studies, PCE-induced kidney and liver tumors and caused leukemia (IARC, 1995). IARC classifies PCE as a probable human carcinogen, and NTP classifies it as reasonably anticipated to be a human carcinogen (IARC, 1995; NTP, 2004). Additional information about these solvents is available from ATSDR at: <http://www.atsdr.cdc.gov/toxpro2.html>.

In an analysis of occurrence data from the EPA 6 Year Review of National

Primary Drinking Water Regulations, PCE was detected in 1,262 systems serving close to 32 million people (EPA, 2009). Concentrations of PCE were greater than the MCL in 241 systems serving close to 15 million people. PCE was the fifth highest occurring regulated volatile organic chemical found based on the percent of detections found from the 6 Year Review data (EPA, 2009).

Biomonitoring Information

Levels of halogenated solvents in blood reflect recent exposure. In the NHANES 2003-2004 subsample, the level of blood PCE for adults at the 75th percentile of the U.S. population appear similar to the levels at the 75th percentile reported for non-smoking adults in a subsample of NHANES 1999-2000 participants (CDC, 2009; Lin et al., 2008) and were similar or slightly less than levels reported in a nonrepresentative subsample of the earlier NHANES III (1988-1994) (Ashley et al., 1994; Churchill et al., 2001). A recent study of low income, urban children in the Midwest reported slightly lower median PCE levels (Sexton et al., 2005; Sexton et al., 2006) than the NHANES III levels (Ashley et al., 1994; Churchill et al., 2001).

Comparatively higher blood levels of PCE and trichloroethene have been noted for urban and industrial residential settings than for rural settings (Barkley et al., 1980; Begerow et al., 1996; Brugnone et al., 1994). Residing near dry-cleaning facilities or storing recently dry-cleaned clothes at home can contribute to increased blood PCE levels (Begerow et al., 1996; Popp et al., 1992). In contrast, PCE blood levels in occupationally exposed workers have been reported to be many thousand times higher than the general population (Begerow et al., 1996; Furuki et al., 2000; Monster et al., 1983). The occupational biological exposure index associated with an 8-hour exposure of 25 ppm is 500 µg/L PCE in blood (ACGIH, 2007). Non-occupational exposures are usually well below this level. Finding a measurable amount of any of these solvents in blood does not mean that the level of the solvent causes an adverse health effect. Biomonitoring studies of blood halogenated solvents can provide physicians and public health officials with reference values so that they can determine whether or not people have been exposed to higher levels of halogenated solvents than levels found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

Sources of PCE

The major source of PCE in drinking water is discharge from factories and dry cleaners. A federal law called the Emergency Planning and Community Right to Know Act requires facilities in certain industries, which manufacture, process, or use significant amounts of toxic chemicals, to report annually on their releases of these chemicals. For more information on the uses and releases of chemicals in your state, contact the Community Right-to-Know Hotline: (800) 424-9346 (EPA, 2010).

	<p>PCE Regulation and Monitoring</p> <p>The EPA limits the amount of PCE that may be present in drinking water to 5 parts of PCE per billion parts of water (5 ppb), or 5 ug/L.</p>
Use of Measure	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to PCE at different concentrations. • Maximum concentrations provide information on the peak potential exposure to PCE at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells may be another source of population exposure to PCE. Transient non-community water systems, which are regulated by EPA, also may be an important source of PCE exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Ground water systems may have multiple wells with different PCE concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the PCE concentration of people served by wells with higher PCE concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different PCE levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. ACGIH. TLVs and BEIs Based on the documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. 2007. Signature Publications. Cincinnati OH. p.104. 2. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for tetrachloroethylene update. 1997 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp18.html. 4/22/09 3. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for Tetrachloroethylene update. 2000 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp14.html. 4/22/09 4. Armstrong SR, Green LC. Chlorinated hydrocarbon solvents. Clin Occup Environ Med 2004;4(3):481-496. 5. Ashley DL, Bonin MA, Cardinali FL, McCraw JM, Wooten JV. Blood concentrations of volatile organic compounds in a nonoccupationally exposed US

- population and in groups with suspected exposure. *Clin Chem* 1994;40(7 Pt 2):1401-1404.
6. Barkley J, Bunch J, Bursey JT, Castillo N, Cooper SD, Davis JM, et al. Gas chromatography mass spectrometry computer analysis of volatile halogenated hydrocarbons in man and his environment—a multimedia environmental study. *Biomed Mass Spectrom* 1980;7(4):139-147.
 7. Begerow J, Jermann E, Keles T, Freier I, Ranft U, Dunemann L. Internal and external tetrachloroethene exposure of persons living in differently polluted areas of Northrhine-Westphalia (Germany). *Zentralbl Hyg Umweltmed*. 1996;198(5):394-406.
 8. Brugnone F, Perbellini L, Guiliari C, Cerpelloni M, Soave M. Blood and urine concentrations of chemical pollutants in the general population. *Med Lav* 1994; 8(5):370-389.
 9. Centers for Disease Control and Prevention (CDC). Fourth National Report on Human Exposure to Environmental Chemicals, 2009. Available at: <http://www.cdc.gov/exposurereport/pdf/FourthReport.pdf>
 10. Churchill JA, Ashley DL, Kaye WE. Recent chemical exposures and blood volatile organic compound levels in a large population- based sample. *Arch Environ Health* 2001; 56(2):157-166.
 11. Furuki K, Ukai H, Okamoto S, Takada S, Kawai T, Miyama Y, Mitsuyoshi K, et al. Monitoring of occupational exposure to tetrachloroethene by analysis for unmetabolized tetrachloroethene in blood and urine in comparison with urinalysis for trichloroacetic acid. *Int Arch Occup Environ Health*. 2000; 73(4):221-227.
 12. International Agency for Research in Cancer (IARC). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals Vol. 63, 1995. Available at: <http://monographs.iarc.fr/ENG/Monographs/vol63/mono63.pdf>
 13. International Programme on Chemical Safety (IPCS). Concise International Chemical Assessment Document 68-Tetrachloroethene. 2006 [online]. Available at URL: [http:// www.inchem.org/documents/cicads/cicads/cicad68.htm](http://www.inchem.org/documents/cicads/cicads/cicad68.htm). 4/22/09
 14. Lin YS, Egeghy PP, Rappaport SM. Relationships between levels of volatile organic compounds in air and blood from the general population. *J Exp Sci Environ Epidemiol* 2008; 18:421-429.
 15. Martin SA, Simmons MB, Ortiz-Serrano M, Kendrick C, Gallo A, Campbell J, et al. Environmental exposure of a community to airborne trichloroethylene. *Arch Environ Occup Health* 2005; 60(6):341-316.
 16. Monster AC. Biological monitoring of chlorinated hydrocarbon solvents. *J Occup Med* 1986; 28:583-588.
 17. Monster AC, Regouin-Peters W, Van Schijndel A, van der Tuin J. Biological monitoring of occupational exposure to tetrachloroethene. *Scand J Work Environ Health* 1983; 9:273-281.
 18. Moran MJ, Zogorski JS, Squillace PJ. Chlorinated solvents in groundwater of the United States. *Environ Sci Technol* 2007; 41:74-81.
 19. National Toxicology Program (NTP). Report on Carcinogens, 11th ed. 2004. [online]. Available at URL: <http://ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s066dich.pdf>. 4/22/09
 20. Popp W, Muller G, Baltes-Schmitz B, Wehner B, Vahrenholz C, Schmieding W, et al. concentrations of tetrachloroethene in blood and trichloroacetic acid in urine in workers and neighbours of dry-cleaning shops. *Int Arch Occup Environ Health* 1992; 63:393- 395.
 21. Rowe BL, Toccalino PL, Moran MJ, Zogorski JS, Price CV. Occurrence and

	<p>potential human-health relevance of volatile organic compounds in drinking water from domestic wells in the United States. <i>Environ Health Perspect</i> 2007; 115(11):1539-1546.</p> <p>22. Schreiber JS, House S, Prohonic E, Smead G, Hudson C, Styk M, et al. An investigation of indoor air contamination in residences above dry cleaners. <i>Risk Anal</i> 1993; 13(3):335-344. Sexton K, Adgate JL, Church TR, Ashley DL, Needham LL, Ramachandran, et al. Children's exposure to volatile organic compounds as determined by longitudinal measurements in blood. <i>Environ Health Perspect</i> 2005; 113(3):342-349.</p> <p>23. Sexton K, Adgate JL, Fredrickson AL, Ryan AD, Needham LL, Ashley DL. Using biologic markers in blood to assess exposure to multiple environmental chemicals for inner-city children 3-6 years of age. <i>Environ Health Perspect</i> 2006; 114(3):453-459.</p> <p>24. U.S. Environmental Protection Agency (U.S. EPA). Basic Information about Tetrachloroethylene in Drinking Water, 2010. Available at:</p> <p>25. http://water.epa.gov/drink/contaminants/basicinformation/tetrachloroethylene.cfm</p> <p>26. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.</p> <p>27. Wallace L, Nelson W, Ziegenfus R, Pellizzari E, Michael L, Whitmore R, et al. The Los Angeles TEAM Study: Personal exposures, indoor-outdoor air concentrations, and breath concentrations of 25 volatile organic compounds. <i>J Exp Anal Environ Epidemiol</i> 1991; 1(2):157-192.</p>
--	---

CONTENT DOMAIN: COMMUNITY WATER

INDICATOR: TRICHLOROETHENE (TRICHLOROETHYLENE) (TCE)

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 3. Yearly distribution of number of CWS by maximum TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE). 4. Yearly distribution of number of CWS by mean TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE). 5. Mean concentration of TCE at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 6. Yearly distribution of number of people served by CWS by maximum TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE). 7. Yearly distribution of number of people served by CWS by mean TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE).
Derivation of Measures	TCE measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	TCE, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be the approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Trichloroethene (TCE) and Public Health</p> <p>Trichloroethene (TCE) is a volatile halogenated short-chain hydrocarbon. TCE is used primarily as an industrial degreaser, solvent, and in the synthesis of other chemicals. In the past, it was used in dry cleaning, food processing, household cleaners, and as a general anesthetic. TCE is produced and used in high volumes in the U.S. and has been detected in urban and ambient air and occasionally soils and drinking water most likely contaminated by industrial discharge (Moran et al., 2007; Rowe et al., 2007). Because of its volatility, this solvent does not persist in the soil or water following the discontinuation of contamination.</p>

Drinking or breathing high levels of TCE may cause nervous system effects, liver and lung damage, abnormal heartbeat, coma, and possibly death (ATSDR, 2003). Inhalation is the most common exposure route for the general population including indoor sources from paints, adhesives, and cleaning solutions. Volatilization from contaminated water (e.g., shower water) as well as the use of household products containing this solvent can result in higher indoor than outdoor air concentrations (ATSDR, 1997b; Martin et al., 2005). Nearby dry cleaning establishments, industries producing this solvent, and contaminated waste disposal sites can also contribute to human exposure (Armstrong and Green, 2004; ATSDR, 1997a, 1997b, and 2000; Schreiber et al., 1993; Wallace et al., 1991). Drinking water may contribute to exposure when underground drinking water supplies have been contaminated. Workers in industries such as dry cleaning, aircraft maintenance, electronics manufacturing, and chemical production may be exposed by inhalation or dermal contact. The EPA has established drinking water standards and other environmental standards for TCE, and the FDA regulates TCE as an indirect food additive. OSHA has established workplace standards, and ACGIH has recommended occupational guidelines and biological exposure indices for monitoring workers (ACGIH, 2007). Human health effects from TCE at low environmental doses or at biomonitored levels from low environmental exposures are unknown. TCE is well absorbed by ingestion and inhalation, and animal studies have demonstrated that liquid forms can be dermally absorbed. Following absorption, part of the solvent dose is excreted into expired air (ATSDR 1997a; Monster, 1986). The retained solvent can undergo hepatic metabolism. TCE is metabolized to trichloroacetic acid and trichloroethanol, which are eliminated in the urine. Accidental or intentional high dose acute exposure by ingestion or inhalation can result in loss of motor coordination, somnolence, and unconsciousness. Inhaling high doses of TCE may also produce cardiac arrhythmias attributed to enhanced sensitivity to catecholamines. Prolonged, low level exposure to TCE has been associated with altered renal enzyme excretion and liver enlargement (ATSDR, 1997a, b). Chronic occupational exposure to TCE may be associated with mild degrees of neurological impairments, including reaction times, verbal skills, cognitive ability and motor function (Armstrong and Green, 2004). In animal studies, TCE induced kidney and liver tumors; and caused lung and testicular tumors (IARC, 1995). A recent EPA toxicological review (EPA/635/R-09/011F) characterized TCE as carcinogenic in humans by all routes of exposure (EPA, 2011). For cancer, the inhalation unit risk is 2×10^{-2} per ppm [4×10^{-6} per $\mu\text{g}/\text{m}^3$], based on human kidney cancer risks (Charbotel et al.; 2006) and adjusted, using human epidemiologic data, for potential risk for non-Hodgkin lymphoma (NHL) and liver cancer. The oral unit risk for cancer is 5×10^{-2} per mg/kg/day, resulting from physiologically based pharmacokinetic model-based route-to-route extrapolation of the inhalation unit risk based on the human kidney cancer risks (Charbotel et al. 2006) and adjusted, using human epidemiologic data, for potential risk for NHL and liver cancer. There is high confidence in these unit

	<p>risks for cancer, as they are based on good quality human data, as well as being similar to unit risk estimates based on multiple rodent bioassays. Evidence is sufficient to conclude that TCE operates through a mutagenic mode of action for kidney tumors. Evidence is insufficient and TCE-specific quantitative data are lacking on early-life susceptibility.</p> <p>Additional information about TCE is available from ATSDR at: http://www.atsdr.cdc.gov/toxpro2.html.</p> <p>In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, TCE was detected in 1,013 systems serving 29.5 million people (EPA, 2009). Concentrations of TCE were greater than the MCL in 195 systems serving close to 12 million people. TCE was the fifth highest occurring regulated volatile organic chemical found based on the percent of population served by systems with at least one sample detection found from the 6 Year Review data (EPA, 2009).</p> <p>Biomonitoring Information Levels of halogenated solvents in blood reflect recent exposure. Blood levels of TCE were generally not detected in the NHANES 2003-2004 subsample and were detected infrequently in previous U.S. surveys (CDC, 2009).</p> <p>Comparatively higher blood levels of tetrachloroethene and TCE have been noted for urban and industrial residential settings than for rural settings (Barkley et al., 1980; Begerow et al., 1996; Brugnone et al., 1994). Finding a measurable amount of any of these solvents in blood does not mean that the level of the solvent causes an adverse health effect. Biomonitoring studies of blood halogenated solvents can provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of halogenated solvents than levels found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.</p> <p>Sources of TCE TCE does not occur naturally in the environment. However, it has been found in underground water sources and many surface waters as a result of the manufacture, use, and disposal of the chemical (ATSDR, 2003).</p> <p>TCE Regulation and Monitoring The EPA has set a maximum contaminant level for TCE in drinking water of 0.005 milligrams per liter (0.005 mg/L) or 5 parts of TCE per billion parts water. The EPA has also developed regulations for the handling and disposal of trichloroethylene.</p> <p>OSHA has set an exposure limit of 100 parts of TCE per million parts of air (100 ppm) for an 8-hour workday, 40-hour work week (ATSDR, 2003).</p>
Use of Measure	<p>These measures assist by providing data that can be used for surveillance purposes.</p>

	<ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to TCE at different concentrations. • Maximum concentrations provide information on the peak potential exposure to TCE at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	The current measures are derived for CWS only. Private wells may be another source of population exposure to TCE. Transient non-community water systems, which are regulated by EPA, also may be an important source of TCE exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	Ground water systems may have multiple wells with different TCE concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the TCE concentration of people served by wells with higher TCE concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different TCE levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. ACGIH. TLVs and BEIs Based on the documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. 2007. Signature Publications. Cincinnati OH. p.104. 2. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for tetrachloroethylene update. 1997a [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp18.html. 4/22/09 3. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for trichloroethylene update. 1997b [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp19.html. 4/22/09 4. Agency for Toxic Substances and Disease Registry (ATSDR). ToxFAQs™ for Trichloroethylene (TCE), July 2003. Available at: http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=172&tid=30 5. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for Methylene chloride update. 2000 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp14.html. 4/22/09 6. Armstrong SR, Green LC. Chlorinated hydrocarbon solvents. Clin Occup Environ Med 2004;4(3):481-496. 7. Barkley J, Bunch J, Bursey JT, Castillo N, Cooper SD, Davis JM, et al. Gas chromatography mass spectrometry computer analysis of volatile halogenated hydrocarbons in man and his environment—a multimedia environmental study.

- Biomed Mass Spectrom 1980;7(4):139-147.
8. Begerow J, Jermann E, Keles T, Freier I, Ranft U, Dunemann L. Internal and external tetrachloroethene exposure of persons living in differently polluted areas of Northrhine-Westphalia (Germany). *Zentralbl Hyg Umweltmed.* 1996;198(5):394-406.
 9. Brugnone F, Perbellini L, Guiliari C, Cerpelloni M, Soave M. Blood and urine concentrations of chemical pollutants in the general population. *Med Lav* 1994;8(5):370-389.
 10. Centers for Disease Control and Prevention (CDC). Fourth National Report on Human Exposure to Environmental Chemicals, 2009. Available at: <http://www.cdc.gov/exposurereport/pdf/FourthReport.pdf>
 11. Charbotel B, Fevotte J, Hoirs M, Martin JL, Bergeret A. Case-control study on renal cell cancer and occupational exposure to trichloroethylene. Part II: Epidemiological aspects. *Ann. Occup. Hyg.* Vol. 50, No. 8, pp. 777-787, 2006.
 12. International Agency for Research in Cancer (IARC). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals Vol. 63, 1995. Available at: <http://monographs.iarc.fr/ENG/Monographs/vol63/mono63.pdf>
 13. Martin SA, Simmons MB, Ortiz-Serrano M, Kendrick C, Gallo A, Campbell J, et al. Environmental exposure of a community to airborne trichloroethylene. *Arch Environ Occup Health* 2005;60(6):341-316.
 14. Monster AC. Biological monitoring of chlorinated hydrocarbon solvents. *J Occup Med* 1986;28:583-588.
 15. Monster AC, Regouin-Peeters W, Van Schijndel A, van der Tuin J. Biological monitoring of occupational exposure to tetrachloroethene. *Scand J Work Environ Health* 1983;9:273-281.
 16. Moran MJ, Zogorski JS, Squillace PJ. Chlorinated solvents in groundwater of the United States. *Environ Sci Technol* 2007;41:74-81.
 17. National Toxicology Program (NTP). Report on Carcinogens, 11th ed. 2004. [online]. Available at URL: <http://ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s066dich.pdf>. 4/22/09
 18. Rowe BL, Toccalino PL, Moran MJ, Zogorski JS, Price CV. Occurrence and potential human-health relevance of volatile organic compounds in drinking water from domestic wells in the United States. *Environ Health Perspect* 2007;115(11):1539-1546.
 19. Schreiber JS, House S, Prohonic E, Smead G, Hudson C, Styk M, et al. An investigation of indoor air contamination in residences above dry cleaners. *Risk Anal* 1993;13(3):335-344.
 20. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.
 21. U.S. Environmental Protection Agency (U.S. EPA). Toxicological Review of Trichloroethylene (CAS No. 79-01-76) In Support of Summary Information on the Integrated Risk Information System (IRIS), September 2011. EPA/635/R-09/011F
<http://www.epa.gov/IRIS/toxreviews/0199tr/0199tr.pdf>.
 22. Wallace L, Nelson W, Ziegenfus R, Pellizzari E, Michael L, Whitmore R, et al. The Los Angeles TEAM Study: Personal exposures, indoor-outdoor air concentrations, and breath concentrations of 25 volatile organic compounds. *J Exp Anal Environ Epidemiol* 1991;1(2):157-192.

CONTENT DOMAIN: COMMUNITY WATER

INDICATOR: URANIUM (U)

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of Community Water Systems (CWS) by maximum Uranium concentration (cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium). 2. Yearly distribution of number of CWS by mean Uranium concentration (cut-points: cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium). 3. Mean concentration of Uranium at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 4. Yearly distribution of number of people served by CWS by maximum Uranium concentration (cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium). 5. Yearly distribution of number of people served by CWS by mean Uranium concentration (cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium).
Derivation of Measures	Uranium measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	Uranium, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Uranium (U) and Public Health</p> <p>Uranium is a silver-white metal that is extremely dense and weakly radioactive. It usually occurs as an oxide and is extracted from ores containing less than 1% natural uranium. Natural uranium is a mixture of three isotopes: 238U (greater than 99%), 235U (about 0.72%), and 234U (about 0.01%). Uranium has many commercial uses, including nuclear weapons, nuclear fuel, in some ceramics,</p>

and as an aid in electron microscopy and photography. Depleted uranium (DU) refers to uranium in which the proportions of ²³⁵U and ²³⁴U isotopes have been reduced compared with the proportion in natural uranium. Since the 1990's, DU has been used by the military in armor-piercing ammunition and as a component of protective armor for tanks. Natural and depleted uranium are primarily chemical toxicants, with radiation playing a minor role or no role at all (ATSDR, 2009).

Everyone is exposed to uranium in food, air, and water as part of the natural environment. (ATSDR, 2009). Variable concentrations of uranium occur naturally in drinking water sources. In some locations the natural concentrations may have increased due to mining and milling of uranium. Thus, the primary exposure sources for non-occupationally exposed persons are likely dietary and drinking water. Populations most heavily exposed to uranium are those employed in mining and milling operations, or in uranium enrichment and processing activities (ATSDR, 2009). In workplaces that involve uranium mining, milling, or processing, human exposure occurs primarily by inhaling dust and other small particles. Exposure to DU may occur in military personnel from retention of internal shrapnel that contains DU or exposure to dust generated from ammunition impact.

Absorption of uranium compounds is low by all routes of exposure (i.e., ingestion, inhalation, and skin contact). Depending upon the specific compound and solubility, 0.1%-6% of an ingested dose may be absorbed. Inhaled uranium-containing particles are retained in the lungs, where limited absorption occurs (less than 5%). After long term or repeated exposure, kidneys, liver, and bones can accumulate uranium with the largest amounts being stored in bones (Li et al., 2005). Uranium is eliminated in feces and urine; about 50% of the absorbed dose is eliminated in the urine within the first 24 hours. After exposure to soluble uranium salts, the initial half-life of uranium is about 15 days (Bhattacharyya et al., 1992), which represents distribution and excretion, with much slower elimination from bone. After inhalation, the half-life of insoluble uranium in the lungs is several years (Durakovic et al., 2003).

Human health effects from uranium at low environmental doses or at biomonitored levels from low environmental exposures are unknown. Health outcomes that may occur with uranium overexposure, based on both observed human effects and animal studies, include non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity. Studies of persons with chronic exposure to elevated uranium salts in drinking water have shown changes in urinary biomarkers potentially associated with impaired kidney function (Kurttio et al., 2006). IARC and NTP have no ratings for uranium human carcinogenicity. Radiation risks from exposure to natural uranium are very low. Alpha radiation (such as that from uranium) is classified as a human carcinogen. However, human studies have not found elevated rates of cancer from uranium exposure, and high-dose animal studies have not found cancer

following inhalation, oral, or dermal exposure to uranium.

Workplace air standards and guidelines for external exposure to soluble and insoluble uranium compounds have been established by OSHA and ACGIH, respectively. Drinking water and other environmental standards have been established by U.S. EPA. Information about external exposure (i.e., environmental levels) and health effects is available from ATSDR at: <http://www.atsdr.cdc.gov/toxpro2.html>.

In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, uranium was detected in 4,101 systems serving close to 55 million people (EPA, 2009). Concentrations of uranium were greater than the MCL in 448 systems serving close to 8.4 million people (EPA, 2009).

Biomonitoring Information

Levels of urinary uranium reflect recent and ongoing or accumulated exposure. A previous nonrandom subsample from NHANES III (n = 499) (Ting et al., 1999) and other small populations have shown urinary concentrations that are similar to those in NHANES 1999-2000, 2001-2002, and 2003-2004 (Dang et al., 1992; Galletti, 2003; Karpas et al., 1996; Tolmachev et al., 2006). Older studies have demonstrated urinary uranium concentrations that are consistent with levels in the U.S. population, in that the levels were below their respective detection limits (Byrne et al., 1991; Hamilton et al., 1994; Komaromy-Hiller et al., 2000). In a study of 105 persons exposed to natural uranium in well water, urinary levels of uranium were as high as 9.55 µg/L (median 0.162 µg/L) (Orloff et al., 2004). Eighty-five percent of those levels were above the 95th percentile of the NHANES 1999-2000 population. The U.S. Nuclear Regulatory Commission (NRC) has set an action level of 15 µg/L urinary uranium to protect people who are occupationally exposed (NRC, 1978). Finding a measurable amount of uranium in urine does not mean that the level of uranium causes an adverse health effect. Biomonitoring studies on levels of uranium provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of uranium than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

Sources of Uranium

Uranium is a naturally-occurring element found in the earth's crust. It is naturally abundant in rocks, soil and water. Significant concentrations of uranium can occur in phosphate rock deposits, and in minerals such as pitchblende and uraninite. The total amount of Uranium on earth stays virtually the same because it has such a long half-life (4.47x10⁹ years for U-238) (EPA, 2010).

	<p>Uranium Regulation and Monitoring</p> <p>The EPA limits the amount of uranium that may be present in drinking water to 30 ug/L (EPA, 2009). A gross alpha particle activity measurement may be substituted for the required uranium measurement provided that the measured gross alpha particle activity does not exceed 15 pCi/l.</p> <p>Monitoring frequency</p> <p>Once a CWS has satisfied initial monitoring requirements (4 quarterly samples at every entry point to the distribution system within the first quarter after initiating the source); the required frequency for Uranium monitoring is once every three years if the average of the initial monitoring results for the contaminant is greater than one-half the MCL but at or below the MCL. States may allow CWS to reduce the frequency of monitoring from once every three years to once every six or nine years at each sampling point, if the average of the initial monitoring results for each contaminant is below the detection limit. If a system has a monitoring result that exceeds the MCL while on reduced monitoring, the system must collect and analyze quarterly samples at that sampling point until the system has results from four consecutive quarters that are below the MCL, unless the system enters into another schedule as part of a formal compliance agreement with the State (CFR, 2002).</p>
Use of Measure	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to Uranium at different concentrations. • Maximum concentrations provide information on the peak potential exposure to Uranium at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells may be another source of population exposure to Uranium. Transient non-community water systems, which are regulated by EPA, may also be an important source of Uranium exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	<p>State grantee</p>
Limitations of Data Sources	<p>The required monitoring frequency for Uranium is infrequent (every 3 to 6 years) and may be as intermittent as every nine years; therefore most states will have very little data on this contaminant.</p> <p>Ground water systems may have multiple wells with different Uranium concentrations that serve different parts of the population. Compliance samples</p>

	are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the Uranium concentrations of people served by wells with higher Uranium concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different Uranium levels are averaged to estimate levels for the PWS.
Related Indicators	Public Water Use; combined Radium-226 and -228
References	<ol style="list-style-type: none"> 1. Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological Profile for uranium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. 2. Bhattacharyya MH, Breitenstein BD, Metivier H, Muggenburg BA, Stradling GN, Volf V. Guidebook for the treatment of accidental internal radionuclide contamination of workers. In: Gerber GB, Thomas RG, eds. Radiation protection dosimetry. Vol. 41 (1). Kent (England): Nuclear Technology Publishing; 1992. pp. 1-49. 3. Byrne AR, Benedik L. Uranium content of blood, urine and hair of exposed and non-exposed persons determined by radiochemical neutron activation analysis, with emphasis on quality control. <i>Sci Total Environ</i> 1991;107:143-157. 4. Centers for Disease Control and Prevention (CDC). Third National Report on Human Exposure to Environmental Chemicals. Atlanta (GA). 2005. 4/20/09 5. Code of Federal Regulations (CFR), 2002. Title 40 Protection of the Environment Chapter I--Environmental Protection Agency Part 141--National Primary Drinking Water Regulations 141.26 Monitoring frequency and compliance requirements for radionuclides in community water systems. Available at: URL: http://www.access.gpo.gov/nara/cfr/waisidx_02/40cfr141_02.html 6. Dang HS, Pullat VR, Pillai KC. Determining the normal concentration of uranium in urine and application of the data to its biokinetics. <i>Health Phys</i> 1992;62:562-566. 7. Durakovic A, Horan P, Dietz LA, Zimmerman I. Estimate of the time zero lung burden of depleted uranium in Persian Gulf War veterans by the 24-hour urinary excretion and exponential decay analysis. <i>Mil Med</i> 2003;168(8):600-605. 8. Ejnik JW, Carmichael AJ, Hamilton MM, McDiarmid M, Squibb K, Boyd P, et al. Determination of the isotopic composition of uranium in urine by inductively coupled plasma mass spectrometry. <i>Health Phys</i> 2000;78:143-146. 9. Galletti M, D'Annibale L, Pinto V, Cremisini C. Uranium daily intake and urinary excretion: a preliminary study in Italy. <i>Health Phys</i> 2003;85:228-235. 10. Gwiazda RH, Squibb K, McDiarmid M, Smith D. Detection of depleted uranium in urine of veterans from the 1991 Gulf War. <i>Health Phys</i> 2004;86:12-18. 11. Hamilton EI, Sabbioni E, Van der Venne MT. Element reference values in tissues from inhabitants of the European community. VI. Review of elements in blood, plasma and urine and a critical evaluation of reference values for the United Kingdom population. <i>Sci Total Environ</i> 1994;158:165-190. 12. Karpas Z, Halicz L, Roiz J, Marko R, Katorza E, Lorber A, et al. Inductively coupled plasma mass spectrometry as a simple, rapid, and inexpensive method for determination of uranium in urine and fresh water: comparison with LIF. <i>Health Phys</i> 1996;71(6):879-885. 13. Komaromy-Hiller G, Ash KO, Costa R, Howerton K. Comparison of representative ranges based on U.S. patient population and literature reference intervals for urinary trace elements. <i>Clin Chim Acta</i> 2000;296(1-2):71-90. 14. Kurttio P, Auvinen A, Salonen L, Saha H, Pekkanen J, Makelainen I, et al. Renal effects of uranium in drinking water. <i>Environ Health Perspect</i> 2002;110(4):337-342.

	<p>15. Kurttio P, Harmionen A, Saha H, Salonen L, Karpas Z, Komulainen H, Auvinen A. Kidney toxicity of ingested uranium from drinking water. <i>Am J Kidney Dis</i> 2006;47(6):972-982.</p> <p>16. Li WB, Roth P, Wahl W, Oeh U, Hollriegl V, Paretzke HG. Biokinetic modeling of uranium in man after injection and ingestion. <i>Radiat Environ Biophys</i> 2005;44:29-40.</p> <p>17. May LM, Heller J, Kalinsky V, Ejnik J, Cordero S, Oberbroekling KJ, et al. Military deployment human exposure assessment: urine total and isotopic uranium sampling results. <i>J Toxicol Environ Health A</i> 2004;67(8-10):697-714.</p> <p>18. Orloff KG, Mistry K, Charp P, Metcalf S, Marino R, Shelly T, et al. Human exposure to uranium in groundwater. <i>Environ Res</i> 2004;94:319-326.</p> <p>19. Ough EA, Lewis BM, Andrews WS, Bennett LG, Hancock RG, Scott K. An examination of uranium levels in Canadian forces personnel who served in the Gulf War and Kosovo. <i>Health Phys</i> 2002;82(4): 527-532.</p> <p>20. Ting BG, Paschal DC, Jarrett JM, Pirkle JL, Jackson RJ, Sampson EJ, et al. Uranium and thorium in urine of United States residents: reference range concentrations. <i>Environ Res</i> 1999;81:45-51.</p> <p>21. Tolmachev S, Kuwabara J, Noguchi H. concentration and daily excretion of uranium in urine of Japanese. <i>Health Phys</i> 2006;91(2):144-153.</p> <p>22. U.S. Nuclear Regulatory Commission (NRC). Regulatory Guide 8.22-Bioassay at uranium mills. Washington (DC): NRC; July 1978.</p> <p>23. U.S. Environmental Protection Agency . Radiation Protection, Uranium, 2010. Available at: http://www.epa.gov/radiation/radionuclides/uranium.html</p> <p>24. U.S. Environmental Protection Agency. The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.</p>
--	--

CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES
INDICATOR: PREMATUREITY

Type Of EPHT Indicator	Health Outcome
Measure	1. Percent of preterm (less than 37 weeks gestation) live singleton births 2. Percent of very preterm (less than 32 weeks gestation) live singleton births
Derivation of Measure	1. Number of live singleton births before 37 weeks of gestation to resident mothers, divided by total number of live singleton births to resident mothers 2. Number of live singleton births before 32 weeks of gestation to resident mothers, divided by total number of live singleton births to resident mothers
Unit	1. Preterm live singleton births 2. Very preterm live singleton births
Geographic Scope	State and national
Geographic Scale	State and County
Time Period	2000-current
Time Scale	Preterm: Annual Very Preterm: 5 yr annual average

<p>Rationale</p>	<p>Preterm birth (at less than 37 completed weeks of gestation and among all births regardless of plurality) affects more than 500,000, or 12.5%, of live births in the United States and is a leading cause of infant mortality and morbidity (8, 9, 13). Of those births, the majority (about 84%) of premature babies are born <i>moderately preterm</i> (between 32 and 36 completed weeks of gestation). The remaining 16% of those are born <i>very preterm</i> (at less than 32 weeks of gestation), representing more than 80,000, or 2%, of live births in the United States. Of those infants born very preterm, about 63% are born between 28–31 weeks of gestation, and about 37% are born at less than 28 weeks of gestation.</p> <p>The preterm birth rate rose 18% between 1990 and 2004 (from 10.6% in 1990 to 12.5% in 2004) and more than 30% since 1981 (from 9.4%) (9). For 2003–2004, increases were seen among both moderately preterm and very preterm births. The percentage of infants born very preterm increased from 1.92% to 2.01% between 1990 and 2004 (9); it also increased between 2003 and 2004 from 1.97% to 2.01%, respectively.</p> <p>Preterm birth rates are higher among black mothers compared to Hispanic and white mothers. Between 2002 and 2003, the rates increased for the three largest race and ethnic groups: non-Hispanic white (11.0 to 11.3%), non-Hispanic black (17.7 to 17.8%), and Hispanic (11.6 to 11.9 %) (9). Since 1990, preterm birth rates have risen by one-third (about 33%) for non-Hispanic white births (from 8.5%) and by 8% for Hispanic births (11.0%). In contrast, preterm rates among non-Hispanic black infants have declined slightly over this period (from 11.9%). However, the preterm birth risk of non-Hispanic blacks continues to be substantially higher than the risk of other race and ethnic groups. Of particular concern is the very preterm rate, about twice as high among non-Hispanic black infants compared to non-Hispanic white and Hispanic births (3.99% compared to 1.6% and 1.73%, respectively).</p> <p>Preterm birth is a leading cause of infant mortality, morbidity, and long-term disability (8, 9, 13, 14). All infants born preterm are at risk for serious health problems; however, those born earliest are at greater risk of medical complications, long-term disabilities, and death.</p> <p>Studies have shown that infants born prematurely, especially those with VLBW, have an increased risk for neurological problems ranging from attention deficit hyperactivity disorder to cerebral palsy or mental retardation compared with infants born at term gestation (1, 6, 8, 14). Preterm birth is associated with nearly half of all congenital neurological defects such as cerebral palsy (9); it is also associated with congenital gastrointestinal defects such as gastroschisis.</p> <p>Preterm infants are at greater risk for serious health problems for several reasons: the earlier an infant is born, the less it will weigh, the less developed its organs will be, and the more medical complications it will likely face later in life. Very preterm infants have the greatest risk of death and lasting disabilities, including mental retardation, cerebral palsy, respiratory (premature lung) and gastrointestinal problems (including birth defects such as gastroschisis), and vision and hearing loss. Preterm births account for health care expenditure of more than \$3 billion per year (14).</p>
-------------------------	---

Studies have shown that major risk factors associated with preterm birth include (2, 4, 7, 8, 10, 14):

1. Plural births
2. Previous preterm birth
3. Certain uterine or cervical abnormalities of the mother
4. Mother's age, race, poverty (for example, black women, women younger than 17 and older than 35 years, and poor women are at greater risk than other women)
5. Male fetal gender (associated with singleton preterm birth)
6. Certain lifestyles and environmental factors, including:
 - Late or no prenatal care,
 - Maternal smoking, alcohol consumption (especially in early pregnancy), illegal drug use, exposure to the medication diethylstilbestrol (DES), domestic violence, lack of social support, stress, long working hours with long periods of standing, being underweight before pregnancy, obesity, marital status, and spacing (less than 6–9 months between giving birth and the beginning of the next pregnancy),
 - Neighborhood-level characteristics,
 - Environmental contaminants (e.g., exposure to air pollution and drinking water contaminated with chemical DBP or lead).

Certain medical conditions during pregnancy (e.g., infections, diabetes, hypertension, blood clotting disorders/thrombophilia, vaginal bleeding, certain birth defects of the fetus) may also increase the risk of preterm birth.

The strength of the association of each of these risk factors with preterm birth varies, and remains a subject of significant debate in the literature (14).

The rise in the occurrence of multiple/plural births, which are much more likely than singleton births to be preterm, influenced the overall preterm birth rate over the past two decades. However, preterm rates for singleton births have also increased, up to 11% since 1990 (9). This increase in singleton preterm births was only in infants born moderately preterm; the singleton very preterm birth rate declined slightly, from 1.69% in 1990 to 1.61% in 2004.

Preterm births are associated with many modifiable risk factors, and prevention of preterm births may greatly contribute to the overall reduction in infant illness, disability, and death. Several studies are being conducted to improve our understanding of the precise causes of preterm births, especially those with VLBW, and to learn how to prevent them. These studies look at how genes, maternal stress, race, occupational and environmental factors, and infections may contribute to preterm birth (8). Better understanding of the specific causes of preterm births is needed before tailored interventions can be developed.

Neighborhood-level characteristics have proven to be useful predictors of preterm birth risks (10). Neighborhoods are the geographic units where interventions can be targeted, and those interventions can be an effective way to reduce preterm birth rates and other adverse birth outcomes. Neighborhood-level characteristics contributing to prematurity include the social, economic, and environmental risk factors such as certain aspects of the built

	<p>environment.</p> <p>Preterm births data are readily available in all state health departments and can be used to examine trends. These trends may reflect the contributions of environmental exposures and other modifiable risks to preterm births. These trends can also be used to evaluate the effectiveness of existing and new prevention programs.</p> <p>“<i>Live birth</i> means the complete expulsion or extraction from its mother of a product of human conception, irrespective of the duration of pregnancy, which, after such expulsion or extraction, breathes, or shows any other evidence of life, such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles, whether or not the umbilical cord has been cut or the placenta is attached. Heartbeats are to be distinguished from transient cardiac contractions; respirations are to be distinguished from fleeting respiratory efforts or gasps.” All states require the reporting of live births regardless of length of gestation or birth weight (3).</p>
<p>Use Of The Measure</p>	<p>These measures can be utilized to enhance public health prevention actions and interventions, and inform policy makers and the public regarding risk factors management and mitigation.</p>
<p>Limitations Of The Measure</p>	<p>Uncertainties associated with gestational age estimates: The interval between the first day of the mother’s last normal menstrual period (LMP) and the day of birth is one method used to determine the gestational age of the newborn. However, this measurement is subject to error for many reasons, including imperfect maternal recall or misidentification of the LMP due to postconception bleeding, delayed ovulation, or intervening early miscarriage (9). Thus, for the purpose of calculating national statistics of preterm births, these data are being edited for gestational ages that are clearly inconsistent with the infant’s plurality and birth weight, but substantial inconsistencies in the data still persist (9).</p> <p>The National Center for Health Statistics (NCHS) and most state vital records offices report gestational age based on an algorithm that uses both the mother’s reported last normal menses and the clinician’s estimate of gestational age. The LMP indicator is used unless its value appears to be inconsistent with birthweight, falls outside likely parameters, or was not reported. If any of these circumstances exist, the clinical estimate is used. Nationwide in 2004, approximately 5.9% of gestational age values were based on the clinical estimate (9).</p> <p>Changes in reporting of the gestational age over time may affect trends in preterm birth rates, especially by race (9). These reporting problems may occur more frequently among some subpopulations and among births with shorter gestations.</p>

	<p>Difficulties of interpreting preterm and very preterm birth rates: The preterm birth rates might be an indicator of pregnancy outcome that does not necessarily predict the true health risk associated with early birth. Preterm rates based on live singleton births may be affected by maternal characteristics; a low preterm birth rate might indicate a low-risk population, and a high preterm birth rate might indicate maternal characteristics that predispose to preterm birth.</p>
Data Sources	<p>Birth certificate data from Vital Statistics state systems (both numerator and denominator);</p> <p>National Vital Statistics System (NVSS), CDC, NCHS http://www.cdc.gov/nchs/VitalStats.htm;</p> <p>CDC Wonder: Natality Data Request, CDC http://wonder.cdc.gov/natality.html</p> <p>CDC GIS Reproductive Health Atlas: http://cdc.gov/reproductivehealth/gisatlas/index.htm</p>
Limitations Of Data Sources	<p>Vital statistics data are readily available, of high quality, and useful for various purposes, including public health surveillance; however, they cannot be correctly interpreted unless various qualifying factors and classification methods are considered (see “Limitations of the Measure”). The factors to be considered will vary depending on the intended use of the data; however, most of the limiting factors result from imperfections in the original records, and they should not be ignored. Yet, their existence does not lessen the value of the data for calculating/estimating this measure.</p> <p>One important limitation of the national data is the timeliness of when the data are available. The national file cannot be compiled until all states have submitted their data. Often times there is delay of 2-3 years before national statistics are available. There are also some differences between national data and state data handling of unknowns, imputation rules, and close out dates. There may be differences or delays in processing resident births that occur out of state. These process issues, along with the need to close off national statistics at specified intervals following a reporting period, may lead to small discrepancies between national data compiled by NCHS and data maintained by state vital statistics registries.</p>
Related Indicators	<p>Low birthweight</p>
References	<ol style="list-style-type: none"> 1. Ananth C. W., Joseph K. S., Oyelese Y., Demissie K., Vintzileos A. M. Trends in Preterm Birth and Perinatal Mortality Among Singletons: United States, 1989 through 2000. <i>Obstet Gynecol</i>, 2005, Vo. 105, No. 5, 1084-1091 2. Blackmore C. A. and Rowley D. L. 1994. Preterm Birth. Editors: Wilcox L.S. and Marks J S. In: <i>From Data to Action CDC’s Public Health Surveillance for Women, Infants, and Children. CDC’s Maternal & Child Health Monograph 1994.</i> Centers for Disease Control and Prevention, Atlanta Georgia

3. Centers for Disease Control and Prevention, National Center for Health Statistics (NCHS), NCHS Definitions. Available from: <http://www.cdc.gov/nchs/datawh/nchsdefs/list.htm> Last accessed: June 19, 2007
4. Cooperstock M. and Campbell J. Excess Males in Preterm Birth: Interactions with Gestational Age, Race, and Multiple Birth. *Obstet Gynecol* 1996, 88: 189-193
5. Hamilton B.E., Martin J.A., and Ventura S.J.: Births: Preliminary Data for 2005. Health E-Stats. Released November 21, 2006. Available from: <http://www.cdc.gov/nchs/products/pubs/pubd/hestats/prelimbirths05/prelimbirths05.htm>
6. Healthy People 2010, Volume 2, Objective 16: Maternal, Infant, and Child Health. <http://www.healthypeople.gov/Document/HTML/Volume2/16MICH.htm>
7. Joseph K. S., Allen A. C., Dodds L., Vincer M. J., and Armson B. A. Causes and Consequences of Recent Increases in Preterm Birth Among Twins. *Obstet Gynecol* 2001, Vol. 98, No. 1, 57-64
8. March of Dimes, 2007. Available from: <http://www.marchofdimes.com>
9. Martin J.A., Hamilton B. E., Sutton P., Ventura S. J., Menacker F., and Kimeyer S. Births: Final Data for 2004. *National Vital Statistics Reports*, Vol. 55, No 1. Hyattsville, MD: National Center for Health Statistics. 2006. Available from: http://www.cdc.gov/nchs/data/nvsr/nvsr55/nvsr55_01.pdf
10. Messer L. C., Buescher P. A., Laraia B. A., and Kaufman J. S. Neighborhood-Level Characteristics as Predictors of Preterm Birth: Examples from Wake County, North Carolina. *SCHS Studies*, No. 148, November 2005. North Carolina Public Health, State Center for Health Statistics, Raleigh, NC. Available from: <http://www.schs.state.nc.us/SCHS/>
11. Monaghan S. C., Little R. E., Hulchiy O., Strassner H., and Gladen B. C. Preterm Birth in Two Urban Areas of Ukraine. *Obstet Gynecol* 2000, Vol. 95, No. 5, 752-755 et al., 2000
12. Petrini J. R., Callaghan W. M., Klebanoff M., Green N. S., Lackritz E. M., Howse J. L., Schwartz R. H., and Damus K. Estimated Effect of 17 Alpha-Hydroxyprogesterone Caproate on Preterm Birth in the United States. *Obstet Gynecol* 2005, 105: 267-272
13. Tan H, Wen S. W., Mark W., Fung K. F. K., Demissie K., and Rhoads G. G. The Association Between Fetal Sex and Preterm Birth in Twin Pregnancies. *Obstet Gynecol* 2004, Vol. 103, No. 2, 327-332
14. Weismiller D. G. Preterm Labor. *American Family Physician*, 1999, Vol. 59, No. 3

CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES
INDICATOR: LOW BIRTHWEIGHT

Type Of EPHT Indicator	Health Outcome
Measure	1. Percent of low birthweight (less than 2500 grams) live term singleton births 2. Percent of very low birthweight (less than 1500 grams) live singleton births
Derivation of Measure	Number of singleton infants live born at term (at or above 37 completed weeks of gestation) with a birthweight of less than 2,500 grams, divided by the total number of singleton infants live born at term to resident mothers Number of live singleton births with a birthweight of less than 1,500 grams, divided by total number of live singleton births to resident mothers
Unit	LBW: live singleton term births VLBW: live singleton births
Geographic Scope	State and national
Geographic Scale	State and County
Time Period	2000-current
Time Scale	Low birthweight: Annual Very low birthweight: 5 yr annual average
Rationale	<p>LBW, a weight of less than 2,500 grams, or 5 pounds, 8 ounces, at birth (regardless of gestational age and plurality), affects about 1 of every 13 babies born each year in the United States (7). Studies have shown that LBW is an important predictor of future morbidity and mortality. Note however, that the percent of LWB babies among all births (a percentage that is confounded by gestational age and plurality) is not recommended as a population-level measure of perinatal morbidity and mortality (1, 11). It is not recommended as a measure because preterm delivery, decreased fetal growth, and genetically determined small body size commonly occur in LBW infants (1). Compared to infants of normal weight, LBW infants may be at increased risk of perinatal morbidity, infections, and the longer-term consequences of impaired development such as delayed motor and social development or learning disabilities. Mortality risk is lowest for infants born weighing 3,500–4,500 grams (8).</p> <p>Nationally, the percentage of LBW infants (regardless of gestational age and plurality) has been increasing steadily; it reached 8.2% of all births in 2005, the highest level reported since 1968 (4). The 2005 rate was 17% higher than the 1970 (7%) rate, which was 22% higher than the 1984 low (6.7%). In addition, this rate is 64% higher than the Healthy People 2010 goal of 5% (5). The percentage of LBW births also increased among singleton births, from 5.9% in 1990 to 6.31% in 2004 (7% increase).</p> <p>Increases in the multiple birth rate, obstetric interventions (e.g., induction of labor and</p>

cesarean delivery), older maternal age at childbearing, and increased use of infertility therapies likely have affected the trends toward lower birthweights (8). Environmental exposures have also been implicated as possible risk factors for LBW, but the magnitude of the contribution to these increased rates remains relatively uncertain. The percentage of LBW increased among each of the largest racial and ethnic groups: non-Hispanic whites (from 7.0% in 2003 to 7.2% in 2004), non-Hispanic blacks (from 13.6% in 2003 to 13.7% in 2004), and Hispanics (from 6.7% in 2003 to 6.8% in 2004) (8).

LBW in singleton births rose between 2003 and 2004 among non-Hispanic white and Hispanic infants; the increase for non-Hispanic black infants was not statistically significant (8). Since 1990, singleton LBW rates have risen 8% and 14% for Hispanic and non-Hispanic white infants, respectively; the rates have declined 2% among non-Hispanic black infants.

The youngest and oldest mothers are the most likely to deliver LBW infants. In 2004, the lowest LBW levels were reported for women aged 25–34 years (7.3% for women aged 25–29 years and 7.5% for women 30–34 year old); the highest LBW levels were for teenagers younger than 15 years (13.6%) and women aged 45–54 years (21.2%) (8). However, much of the elevated LBW risk among older mothers can be attributed to their higher multiple birth rates; in fact, the LBW rate declined from 21% to 10% for the oldest mothers of singleton births.

LBW rates also vary widely between states or reporting areas (8). In 2004, more than 10% of all infants born in Alabama, Louisiana, Mississippi, South Carolina, and the District of Columbia were LBW., This compares with less than 6.5% of newborns in Alaska, Maine, Oregon, Vermont, and Washington that were LBW. Different demographic characteristics of these populations, including maternal age, race, or ethnicity, may explain some of these differences.

Infants weighing less than 1,500 grams, or 3 pounds, 4 ounces, at birth are considered VLBW (3); most of them are also premature (born before 37 weeks gestation). (Note that the percent of VLBW babies among all births is also confounded by plurality; therefore, the percent of VLBW births among singleton births is recommended as a population-level measure of prematurity.) Studies have shown that the infant's birthweight is a predictor of future morbidity and mortality (8), especially for VLBW infants. VLBW infants have about a 25% chance of dying in the first year of life; this risk is estimated to be about 100 times higher for VLBW infants than for normal-weight infants ($\geq 2,500$ grams) (8). VLBW infants have an increased risk for developing neurological and intellectual problems (including attention deficit hyperactivity disorder, cerebral palsy, developmental delay and mental retardation), visual problems (including blindness), hearing loss, infections, and chronic lung diseases compared with infants of normal weight or infants born at term gestation (2, 5, 6, 7).

Nationally, the percentage of VLBW infants (regardless of plurality) increased slightly from 1.45% in 2003 to 1.49% in 2005, and has increased from 1.27% in 1990 (5). The 2005 rate is 66% higher than the Healthy People 2010 goal of 0.9% (5). The VLBW has

increased since 1990 among whites, blacks, Puerto Ricans, American Indians, and other population groups (5). For 2004–2005, increases in VLBW rates were statistically significant for non-Hispanic black infants but not for non-Hispanic white infants (8).

The increase in the rate of multiple births, in which the infants tend to be much smaller than in singleton births, has likely affected the upward trend in the VLBW rate (8). However, the VLBW rate among singleton births also increased slightly from 1.12% in 2004 to 1.14% in 2005 (8).

Increases in obstetric interventions (e.g., induction of labor and cesarean delivery), teenage pregnancy, and older maternal age at childbearing likely contributed to the increased VLBW rates. Teen mothers, especially those younger than aged 15 years, have a higher chance of giving birth to a VLBW infant. Environmental exposures, including exposure to air pollution, drinking water contaminated with chemical DBP, and exposure to pesticides, have also been implicated as possible risk factors for VLBW, but the exact magnitude of the contribution to the increased VLBW rates remains relatively uncertain

Birthweight is a multifactorial and heterogeneous birth outcome. Birthweight of an infant is directly related to its gestational age. As noted above, multiple births are usually LBW, even those delivered at term. Therefore, the focus of the measure is restricted to singleton term births. As such, the measure distinguishes between preterm and multiple birth categories and decreased fetal growth that may be affected by other risk factors, including environmental factors.

LBW rate is associated with many modifiable risk factors, and preventing LBW may contribute to the overall reduction in infant illness, disability, and death. Several studies are being conducted that may help understand the biological, social, and environmental factors that contribute to LBW births and learn how to prevent them. These studies look at how genes, hormonal changes, maternal stress, race, occupational and environmental factors, and infections may contribute to prematurity and LBW (7). Specific causes of LBW births must be better understood before tailored interventions can be developed.

Neighborhood-level characteristics have proven to be useful predictors of LBW risks (9). Neighborhoods are the geographic units where interventions can be targeted, and those interventions can be an effective ways to reduce LBW rates, infant mortality, and other adverse birth outcomes. Neighborhood-level characteristics contributing to LBW include social, economic, and environmental risk factors, such as certain aspects of the built environment.

The percentage of LBW among term singleton births is a useful and feasible measure of perinatal health. LBW, gestational age, and plurality data are readily available in all state health departments, and can be used to examine trends that occur over time and space. These trends may reflect the contributions of environmental exposures and other modifiable risk factors for LBW.

Exposure to air pollution (both indoor and outdoor) and drinking water contaminated with

	<p>chemical DBPs or lead may serve as examples of environmental risk factors. Maternal smoking, alcohol consumption, or inadequate weight gain are associated with an increased risk of intrauterine growth retardation and LBW. Socioeconomic factors, including low income and lack of education, are reported as risk factors for LBW (10).</p> <p>Women younger than 15 years or older than 35 years, unmarried mothers, and women who have had previous preterm birth are at increased risk of having LBW babies. Women who experience excessive stress, domestic violence, or other abuse also may be at increased risk of having a LBW baby (7).</p> <p>“<i>Live birth</i> means the complete expulsion or extraction from its mother of a product of human conception, irrespective of the duration of pregnancy, which, after such expulsion or extraction, breathes, or shows any other evidence of life, such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles, whether or not the umbilical cord has been cut or the placenta is attached. Heartbeats are to be distinguished from transient cardiac contractions; respirations are to be distinguished from fleeting respiratory efforts or gasps.” All states require the reporting of live births, regardless of length of gestation or birth weight (3).</p> <p><i>Birthweight</i> is the first weight of the newborn obtained after birth (3).</p> <p><i>Low birthweight</i> is defined as less than 2,500 grams or 5 pounds, 8 ounces (3). Before 1979, low birthweight was defined as 2,500 grams or less.</p> <p><i>Very low birthweight</i> is defined as less than 1,500 grams or 3 pounds, 4 ounces (3). Before 1979, very low birthweight was defined as 1,500 grams or less.</p> <p><i>Term birth</i> is defined here as the birth at or above 37 completed weeks of gestation.</p>
<p>Use Of The Measure</p>	<p>This indicator can be used to influence public health prevention actions and interventions and policy makers and inform the public regarding risk factors management and mitigation.</p> <p>The LBW measure can be used to track the perinatal health in states, regions, counties, and smaller geographic areas or communities, as needed. Baseline data can be used to monitor changes or trends.</p> <p>This measure can also be used to evaluate the effectiveness of existing and new prevention programs.</p>
<p>Limitations Of The Measure</p>	<p>Difficulties of interpreting LBW birth rates among term singleton births: Using LBW rates alone as a pregnancy outcome measure might not inform the user about the true health risk associated with LBW.</p> <p>Difficulties of interpreting VLBW birth rates: Although the percentage of VLBW births has increased during the past 20 years, in large part this could be due to improvements in fetal health. Conditions that may have resulted in a fetal death decades ago might today result in fetal survival and a live VLBW birth (6).</p>

	<p>Recommendations: LBW rates should be interpreted with caution. The LBW rate should be only one of the reproductive outcome measures being tracked, and it should be accompanied by the infant mortality rate (neonatal and postneonatal), fetal death rate if reliable, and morbidity measures. If feasible, an infant’s anthropometric parameters should also be monitored; this could include a reduced head circumference measure because smaller head size may predict lower IQ and cognitive abilities and may be associated with ADD/ADHD.</p>
Data Sources	<p>Birth certificate data from Vital Statistics state systems (both numerator and denominator)</p> <p>National Vital Statistics System (NVSS), CDC, NCHS; CDC Wonder: Natality Data Request, CDC http://wonder.cdc.gov/natality.html</p> <p>CDC GIS Reproductive Health Atlas: http://cdc.gov/reproductivehealth/gisatlas/index.htm</p>
Limitations Of Data Sources	<p>Although vital statistics data are readily available, of high quality, and otherwise useful for various purposes, including public health surveillance, they cannot be correctly interpreted unless various qualifying factors and classification methods are considered (see also “Limitations of the Measure”). The factors to be considered will vary, depending of the intended use of the data; however, most of the limiting factors result from imperfections in the original records, and they should not be ignored. Yet, their existence does not lessen the value of the data for the purpose of calculating this measure. At the minimum, the following data quality attributes should be evaluated: completeness of registration, reporting and quality control procedures, and records geocoding procedures and quality.</p> <p>One important limitation of the national data is the timeliness of when the data are available. The national file cannot be compiled until all states have submitted their data. Often times there is delay of 2-3 years before national statistics are available. There are also some differences between national data and state data handling of unknowns, imputation rules, and close out dates. There may be differences or delays in processing resident births that occur out of state. These process issues, along with the need to close off national statistics at specified intervals following a reporting period, may lead to small discrepancies between national data compiled by NCHS and data maintained by state vital statistics registries.</p>
Related Indicators	<p>Prematurity</p>
References	<ol style="list-style-type: none"> 1. Adams M., Andersen A-M. N., Andersen P. K., Haig D., Henriksen T. B., Hertz-Picciotto I., Lie R. T., Olsen J., Skjerven R., and Wilcox A. Sostrup Statement on Low Birthweight. Int J Epidemiol 2003, 32: 884-885 2. Ananth C. W., Joseph K. S., Oyelese Y., Demissie K., Vintzileos A. M. Trends in Preterm Birth and Perinatal Mortality Among Singletons: United States, 1989 through 2000. Obstet Gynecol,2005, Vo. 105, No. 5, 1084-1091 3. Centers for Disease Control and Prevention, National Center for Health Statistics (NCHS), NCHS Definitions. Available from:

<http://www.cdc.gov/nchs/datawh/nchsdefs/list.htm> Last accessed: June 19, 2007

4. Hamilton B.E., Martin J.A., and Ventura S.J.: Births: Preliminary Data for 2005. *Health E-Stats*. Released November 21, 2006. Available from: <http://www.cdc.gov/nchs/products/pubs/pubd/hestats/prelimbirths05/prelimbirths05.htm>
5. Healthy People 2010, Volume 2, Objective 16: Maternal, Infant, and Child Health. <http://www.healthypeople.gov/Document/HTML/Volume2/16MICH.htm>
6. Kitchen WH, Permezel MJ, Doyle LW, Ford GW, Rickards AL, and Kelly EA. Changing obstetric practice and 2-year outcome of the fetus of birth weight under 1000 g. *Obstet Gynecol*. 1992 Feb;79(2):268-75.
7. March of Dimes, 2007. Available from: <http://www.marchofdimes.com>
8. Martin J.A., Hamilton B. E., Sutton P., Ventura S. J., Menacker F., and Kimeyer S. Births: Final Data for 2004. *National Vital Statistics Reports*, Vol. 55, No 1. Hyattsville, MD: National Center for Health Statistics. 2006. Available from: http://www.cdc.gov/nchs/data/nvsr/nvsr55/nvsr55_01.pdf
9. Messer L. C., Buescher P. A., Laraia B. A., and Kaufman J. S. Neighborhood-Level Characteristics as Predictors of Preterm Birth: Examples from Wake County, North Carolina. *SCHS Studies*, No. 148, November 2005. North Carolina Public Health, State Center for Health Statistics, Raleigh, NC. Available from: <http://www.schs.state.nc.us/SCHS/>
10. Preliminary Results from the Central Valley/South Coast Children’s Environmental Health Demonstration Project – Supplement to July 2006 Report, California Environmental Health Tracking Program, 2006. Available from: <http://www.catracking.com/cvsc>
11. Wilcox A. J. On the Importance – and the Unimportance – of Birthweight. *Int J Epidemiol* 2001, 30: 1233-1241

CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES
INDICATOR: MORTALITY (USING PERIOD LINKED BIRTH/INFANT DEATH APPROACH)

Type of EPHT Indicator	Health Outcome
Measures	<ol style="list-style-type: none"> 1. Average Infant (less than 1 year of age) Mortality Rate per 1000 live births 2. Average Neonatal (less than 28 days of age) Mortality Rate per 1000 live births 3. Average Perinatal (equal to or greater than 28 weeks gestation to less than 7 days of age) Mortality Rate per 1000 live births (plus fetal deaths equal to or greater than 28 weeks gestation) 4. Average Postneonatal (equal to or greater than 28 days to less than 1 year of age) Mortality Rate per 1000 live births
Derivation of Measures	<ol style="list-style-type: none"> 1. Infants: Number of deaths occurring in infant residents under 1 year of age (under 366 days during a leap year) in a given year divided by the number of live births in the same year. 2. Neonates: Number of deaths occurring in infant residents less than 28 days of age in a given year divided by the number of live births in the same year 3. Perinates: Number of fetal deaths in infant residents greater than or equal to 28 weeks gestation plus infant deaths less than 7 days old in a given year divided by the number of live births plus fetal deaths at greater than or equal to 28 weeks gestation in the same year 4. Postneonates: Number of deaths occurring in infant residents at 28 days to less than 1 year of age (under 366 days during a leap year) in a given year divided by the number of live births in the same year <p>Both birth and death counts are geographically classified based on maternal residence at the time of birth.</p>
Units	<ol style="list-style-type: none"> 1. Deaths per 1,000 live births 2. Deaths per 1,000 live births 3. Deaths per 1,000 live births plus fetal deaths at 28 or greater weeks gestation 4. Deaths per 1,000 live births
Geographic Scope	State and national
Geographic Scale	State and County
Time Period	2000-current
Time Scale	Five year

<p>Rationale</p>	<p>Fetuses and young children may be particularly susceptible to harmful effects of environmental contaminants. Many environmental contaminants have been proposed to be particularly toxic in utero; many cross the placenta and make their way into the circulatory system of the developing fetus. However, specific health effects are often not well understood for years. Therefore, gross indicators of childhood health—such as mortality—should be tracked as part of an EPHT system. Furthermore, data on births and deaths in a region may be far more complete than data on other health-related events.</p> <p>Overall, congenital malformations, deformations, and chromosomal abnormalities are the leading cause of infant deaths (20.1% of deaths) (1). Disorders related to short gestation and LBW are second, making up 16.6% of deaths. However, importantly, cause of death varies over the first year of life, and combining all causes obscures the fact that sudden infant death syndrome is the leading cause of death in the postneonatal period.</p> <p>Disorders related to short gestation and LBW are the leading cause of neonatal death (24.3% of deaths) (1). This is in contrast to the leading cause of postneonatal death, which is sudden infant death syndrome (21.8%). Congenital malformations, deformations, and chromosomal abnormalities are the second-leading cause of neonatal deaths (21.4%) and postneonatal deaths (17.5%) (1).</p> <p>Restricting infant mortality to deaths during the perinatal, neonatal, or postneonatal period may limit the etiologic heterogeneity inherent in a gross measure such as overall infant mortality. Also, it may be more likely that infants who died within 7 or 28 days, respectively, were living in reasonable proximity to where they were born, making ecological associations with environmental exposures potentially more meaningful. Specifically, exclusion of infants who died within 28 days might reduce etiologic heterogeneity due to differences in early prenatal care and other non-environmental factors likely to influence neonatal survival.</p> <p>When a fetus or an infant dies around the time of labor and delivery, it is not always clear whether to classify this event as a live birth and infant death, or a fetal death. Diagnostic ability for detecting signs of life, such as breathing or beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles after expulsion or extraction from the mother may vary across obstetric clinics.</p> <p>Unexplained fetal death and death related to growth restriction are the leading causes of fetal loss (2). Fetal death is an important contribution</p>
-------------------------	--

	<p>to reproductive loss, with the rate being many times higher than the rate of sudden infant death syndrome among infants (1). Although the rate of late fetal loss (greater than or equal to 28 weeks gestation) has been decreasing in past decades, the rate of intermediate fetal loss (20–27 weeks gestation) has remained relatively constant (3). Markers of increased risk for fetal loss include pre-pregnancy obesity, lower socioeconomic status, non-Hispanic black race, and advanced maternal age.</p>
Use of the Measure	<p>Identifying populations with higher infant, neonatal, perinatal, and postneonatal mortality rates may indicate where potential environmental problems are. It will assist in targeting outreach intervention activities and improve our understanding of geographic variation, time trends, and demographic patterns of infant death.</p>
Limitations of the Measure	<p>An important limitation of this health outcome measure is the heterogeneity in its etiology. Environmental exposure-related causes of infant death are only one piece of a puzzle that includes many other factors, such as access to and quality of health care, competency in childcare, and understanding of injury prevention.</p> <p>The maternal residence during pregnancy and the infant’s residence during the first year of life are critical data for linking deaths to environmental hazards/exposures; these residences may differ from maternal residence at birth or infant residence at death. The mother may have lived far from the place at which she gave birth during part or all of the pregnancy. The infant who died may have been born and lived for a major portion of its life far from the place of death; it may be less likely that neonates and perinates who died were born and lived far from the place of death.</p> <p>NCHS currently uses a period linkage approach that links death certificates to birth certificates. This approach would allow stratification of deaths according to place of birth. However, it does not address the possibility that migration across states or other geographies occurred <i>during</i> pregnancy or infancy.</p>
Data Sources	<p>Local, state, or national vital statistics systems (birth, death, and fetal death records)</p>
Limitations of Data Sources	<p>It may be reasonable to assume universal reporting of live births and infant deaths in the United States; however, some births/deaths may be excluded because of the difficulty in distinguishing a death shortly after birth as a live birth; a death soon after birth might be reported as a fetal death rather than as a live birth and infant death. In addition, some fetal deaths may be missed in some regions, although those occurring at greater than or equal to 28 weeks are less likely to be missing.</p> <p>Data on fetal death certificates may not provide all the information that</p>

	<p>can be collected from birth certificates linked to infant deaths within 7 days; however, many variables used for environmental health tracking (maternal race/ethnicity and age, place of residence) have relatively complete reporting on the fetal death certificate.</p> <p>Births and deaths will be tabulated according to maternal race/ethnicity, using linked data from birth certificates.</p>
<p>References</p>	<ol style="list-style-type: none"> 1. Heron M. Deaths: Leading Causes for 2004. National Vital Statistics Reports; vol. 56, no. 5. Hyattsville, Maryland: National Center for Health Statistics. 2007. Available from: http://www.cdc.gov/nchs/data/nvsr/nvsr56/nvsr56_05.pdf 2. Fretts, RC. Etiology and prevention of stillbirth. Am J Obstet Gynecol. 193(6): 1923-35. 2005. 3. MacDorman MF, Hoyert DL, Martin JA, Munson ML, Hamilton BE. Fetal and perinatal mortality, United States, 2003. Natl Vital Stat Rep. 2007 Feb 21;55(6):1-17.

CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES

INDICATOR: FERTILITY

Type of EPHT Indicator	Health outcome
Measure	Total Fertility Rate per 1000 women of reproductive age
Derivation of Measure(s)	TFR = sum of age-specific fertility rates * 5
Unit	Rate per 1,000 women of reproductive age
Geographic Scope	State and national
Geographic Scale	State and County
Time Period	2000-current
Time Scale	Year
Rationale	<p>The cause of approximately 10% of fertility problems is unknown, and environmental contaminants, including endocrine disruptors, have been considered major contributors. The case of diethylstilbestrol revealed that environmental contamination can have multi-generational effects on reproduction that should be studied and tracked long-term. Several indicators have been used to track fertility on a global, national, state, and local level. Indicators most commonly used are the general fertility rate (GFR), which is defined as the number of live births divided by the total number of women of reproductive age (aged 15–44 years), and the total fertility rate (TFR).</p> <p>The TFR differs from the GFR in that it adjusts for age-specific differences in fertility. It also shows the potential impact of current fertility patterns on reproduction, allowing for more valid comparisons of rates across time and space.</p> <p><i>Fecundity:</i> The physical ability of a woman or couple to conceive and carry a child to term birth.</p> <p><i>Fertility:</i> The ability to conceive a child.</p>
Use of the Measure	The TFR indicates the average number of births to a hypothetical cohort of 1,000 women if they experienced the age-specific birth rates observed in a given year. Understanding the geographic distribution and trends in fertility will provide basic descriptive clues to changes that may be influenced by environmental risk factors. As more is learned regarding the link between adverse exposures and fertility, these rates will provide important background information about how fertility varies geographically in relation to changes in potentially related environmental risk factors and how it has varied over time within the United States. Similar to the GFR, the TFR may not be

	specific enough to permit tracking of specific changes related to environmental risk factors. However, if the estimate of 10% is correct, this measure can be used with other measures, including ambient concentrations of pollutants, to examine potential associations with population-level changes in fertility and generate some well-informed hypotheses or areas for future investigations.
Limitations of the Measure	The fertility measure is influenced by social/demographic choices for reproduction, maternal age, parity, and social class measures, as well as the use of contraception and infertility treatments leading to multiple births. These factors all may determine variations in overall fertility across populations and geographic locations; therefore social and demographic factors would need to be controlled for to examine any environmental effects on total fertility.
Data Sources	<p>Numerator: U.S. National Center for Health Statistics—Vital Statistics Reports and/or state-specific vital statistics (for more recent years of data)</p> <p>Denominator: U.S. Census Bureau</p>
Limitations of Data Sources	National-level data sources may differ slightly from state-level vital statistics data sources

CONTENT DOMAIN: REPRODUCTIVE HEALTH OUTCOMES
INDICATOR: SEX RATIO AT BIRTH AMONG SINGLETON BIRTHS

Type of EPHT Indicator	Health outcome
Measure	Male to Female sex ratio at birth (term singletons only)
Derivation of Measure(s)	Sex ratio=total males/total females at birth among term singleton births only
Unit	Ratio
Geographic Scope	State and national
Geographic Scale	State and county
Time Period	2000-current
Time Scale	Year
Rationale	Population growth is, in part, related to the number of live male children (1). Numerous studies have reported changes in the ratio of males to females at birth; many of the studies have found a reduction in male relative to female births in different countries throughout the world (2-5). Although the mechanism that determines the sex of the infant is not completely understood, some (6-12), but not all (3-4), have suggested that environmental hazards can affect the number of males. Biological parent(s) and/or the fetus can come in contact with and become exposed to different hazards referred to as endocrine disruptors (7-8, 10, 12). Fewer males are conceived when exposure to endocrine disruptors results in a decrease in testosterone. Because states have accurate Vital Statistics (VS) records on the sex of live births, changes over time in the sex ratio of infants can be measured as the ratio of males to females. This ratio of total males/total females born in a pre-defined polygon (e.g., state, county, ZIP code, census tract, block group) at a certain time (one birth year or multiple years) is referred to as the Sex Ratio (SR).
Use of the Measure	The SR can be used to monitor the proportion of males to females in states, counties, or smaller-resolution polygons, when data are available and such analyses are justified. Baseline data can be used to determine if the proportion of males is changing over time. When the number of male births is the same as the number of female births, the SR is equal to 1.000. Many studies have observed baseline SR values that are usually higher than 1.000, and closer to 1.050(1, 3, 13). In 2002, the U.S. SR was 1.048 (1). If the SR is decreasing over time, the implication is that fewer males than females are born for that period of time. If consistent decreases in the SR occur, this outcome could be used to determine if such changes are the result of environmental hazards that can disrupt the endocrine system or some other

	physiological system related directly or indirectly to the expression of the neonates' sex at birth.
Limitations of the Measure	Unfortunately, other factors besides endocrine disruptors can affect the expression of sex (6, 13-15). Decreases in male births inversely related to parental smoking, gestation length, parental age, and birth order. Reproductive practices and social morays regarding sex preferences—males over females, for example, can affect the observed SR (3, 4, 7). Case-control studies have to be carried out to determine if decreases in the SR over time are due to contact with and exposure to endocrine disruptors; but effect modifiers have to be controlled in order to understand this relationship, factors that modify it need to be better accounted for. (8).
Data Sources	State's VS data, CDC Wonder, CDC VS data, and U.S. Census 2000 data in Summary File (SF) 1.
Limitations of Data Sources	There may be discrepancies between national and state data as noted in the templates for measures of prematurity and growth retardation above.
References	<ol style="list-style-type: none"> 1. Mathews TJ, Brady E, Hamilton, E. Trend analysis of the sex ratio at birth in the United States. <i>National Vital Statistics Reports; volume 53, number 20</i>. Hyattsville, Maryland: National Center for Health Statistics. 2005. 2. Grech V, Vassallo-Agius P, Savona-Ventura C. Secular trends in sex ratios at birth in North America and Europe over the second half of the 20th century. <i>J Epidemiol Community Health</i> 2003;57:612-5. 3. Marcus M, Kiely J, Xu F, et al. Changing sex ratio in the United States, 1969-1995. <i>Fertil Steril</i> 1998;70:270-3. 4. Martuzzi M, Di Tanno N, Bertollini R. Declining trends of male proportion at birth in Europe. <i>Arch Environ Health</i> 2001;56:358-364. 5. Parazzini F, La Vecchia C, Levi F, et al. Trends in male: female ratio among newborn infants in 29 countries from five continents. <i>Hum Reprod</i> 1998;13:1394-6. 6. Fukuda M, Fukuda K, Shimizu T, et al. Parental preconceptional smoking and male: female ratio of newborns. <i>Lancet</i> 2002;359:1407-8. 7. Garry VF, Holland SE, Erickson LL, et al. Male reproductive hormones and thyroid function in pesticide applicators in the Red River Valley of Minnesota. <i>J Toxicol Environ Health Part</i>

	<p>A 2003;66:965-86.</p> <ol style="list-style-type: none"> 8. Gomez del Rio I, Marshall T, Tsai P, et al. Number of boys born to men exposed to polychlorinated biphenyls. <i>Lancet</i> 2002;360:143-4. 9. Karmaus W, Huang S, Cameron L. Parental concentration of dichlorodiphenyl dichloroethene and polychlorinated biphenyls in Michigan fish eaters and sex ratio in offspring. <i>J Occup Environ Med</i> 2002;44:8-13. 10. Mackenzie CA, Lockridge A, Keith M. Declining sex ratio in a first nation community. <i>Environ Health Perspect</i> 2005;113:1295-8. 11. Sakamoto M, Nakano A, Akagi H. Declining Minamata male birth ratio associated with increased male fetal death due to heavy methylmercury pollution. <i>Environ Res</i> 2001;87:92-8. 12. Weisskopf MC, Anderson HA, Hanrahan LP, et al. Decreased sex ratio following maternal exposure to polychlorinated biphenyls from contaminated Great Lakes sport-caught fish: a retrospective cohort study. <i>Environ Health</i> 2003;2:2. 13. Vatten LJ, Skjærven R. Offspring sex and pregnancy outcome by length of gestation. <i>Early Hum Dev</i> 2004;76:47-54. 14. Juntunen KST, Kvist AP, Kauppila AJI. A shift from a male to a female majority in newborns with increasing age of grand multiparous women. <i>Hum Reprod</i> 1997;12:2321-3. 15. Nicolich MJ, Huebner WW, Schnatter AR. Influence of parental and biological factors on the male birth fraction in the United States: An analysis of birth certificate data from 1964 through 1988. <i>Fertil Steril</i> 2000;73:487-92.
--	---

Appendix B

National EPHT “How-To” Guides

Content Domain: Emergency Department Visits

**Recommendations for
Nationally Consistent Data and Measures**

***Abbreviated* Part 2: Recommended Data Sets**

Version History v3.27	
Field Name	Description
RACE	Updated RACE variable to OPTIONAL
Version History v3.26	
Field Name	Description
INCIDENTCOUNTFIRE	Updated the “ Code Scheme ” to clarify the use of “-999” value. The updated text now states: “If -999= Count is recorded in INCIDENTCOUNTUNKNOWN”
INCIDENTCOUNTNONFIRE	Updated the “ Code Scheme ” to clarify the use of “-999” value. The updated text now states: “If -999= Count is recorded in INCIDENTCOUNTUNKNOWN”
Version History v3.25	
Field Name	Description
HEALTHOUTCOMEID	Field has been modified to include all health outcome ED Visits as: 1= asthma 3= carbon monoxide poisoning 4= heat stress illness
COUNT_FIRE_H	Renamed INCIDENTCOUNTFIRE
COUNT_NONFIRE_H	Renamed INCIDENTCOUNTNONFIRE
COUNT_UNK_H	Renamed as INCIDENTCOUNTUNKNOWN
Version History v3.24	
Field Name	Description
DAILYAVG	Field has been deleted from the data set
Version History v3.23	
Field Name	Description
MONTHLYMIN	Field has been deleted from the data set
MONTHLYMAX	Field has been deleted from the data set
Version History v3.22	
Field Name	Description
MONTHLYMIN	-999 has been added to denote missing values (9999 was used previously)
MONTHLYMAX	-999 has been added to denote missing values (9999 was used previously)
DAILYAVG	Field has been deleted from the schema. CDC will calculate from MONTHLYHOSP and # days in given month

Version History v3.21	
Field Name	Description
COUNTY	A code indicating county unknown ('U') was added.
COUNTY	FIPS code changed from 3 digit to 5 digit
RACEETHNICITYREPORTED	Code set was modified to differentiate between variable collection methods 1=yes separate 2= yes combined 3= no
TRANSFEREXCLUSION	9=unknown deleted since exclusion of transfers will never be unknown
EXCLUSIONMETHOD	9 changed to Not Applicable

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Emergency Department (ED) Data

Characteristic	Description
Data Source	State and Local Data Systems
Purpose	This data set will be used to calculate incidence measures for emergency department (ED) visits related to unintentional CO poisoning as described in the Part 1 package, for use on the national public portal.
Geographic Level	The smallest geographic unit to be represented in this data set is the county.
Restrictions	This is a restricted access data set. Data will be displayed via the national public portal only when sufficient conditions have been met to protect data privacy. Only registered users will have direct access to this data set via the national secure portal.

NOTE: When preparing the data file, the sequence of data elements (i.e. field names) in the data file can be found in the schema for each health outcome. For example: The sequence of data elements (i.e. field) for health outcome CO poisoning:

```

<ROWIDENTIFIER>2</ROWIDENTIFIER>
<AGEGROUP>19</AGEGROUP>
<COUNTY>06001</COUNTY>
<EDVISITYEAR>2000</EDVISITYEAR>
<EDVISITMONTH>01</EDVISITMONTH>
<ETHNICITY>H</ETHNICITY>
<HEALTHOUTCOMEID>3</HEALTHOUTCOMEID>
<MONTHLYVISITS >10000</MONTHLYVISITS >
<RACE>W</RACE>
<RACEETHNICITYREPORTED>2</RACEETHNICITYREPORTED>
<SEX>M</SEX>
<INCIDENTCOUNTFIRE>0</INCIDENTCOUNTFIRE>
<INCIDENTCOUNTNONFIRE>0</INCIDENTCOUNTNONFIRE>
<INCIDENTCOUNTUNKNOWN>0</INCIDENTCOUNTUNKNOWN>
    
```


**ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA DICTIONARY FOR AGGREGATE DATA
EMERGENCY DEPARTMENT (ED) VISITS**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length
AGEGROUP	Five-year age group of individuals hospitalized for the condition indicated by HEALTHOUTCOMEID	Integer	1= 0–4 years 2= 5–9 years 3= 10–14 years 4= 15–19 years 5= 20–24 years 6= 25–29 years 7= 30–34 years 8= 35–39 years 9= 40–44 years 10= 45–49 years 11= 50–54 years 12= 55–59 years 13= 60–64 years 14= 65–69 years 15= 70–74 years 16= 75–79 years 17= 80–84 years 18= 85+ years 19= Unknown	1–19	2
COUNTY	County of residence	String	FIPS U=Unknown	00000– 99999 U	5
EDVISITMONTH	Month of ED visit	String	mm	01–12	2
EDVISITYEAR	Year of ED visit	Integer	yyyy	20XX	4

**ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA DICTIONARY FOR AGGREGATE DATA
EMERGENCY DEPARTMENT (ED) VISITS**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length
ETHNICITY (optionally reported)	Ethnicity of individuals	Text	H=Hispanic NH=Non-Hispanic U=Unknown	H, NH, U	2
EXCLUSIONMETHOD	Variable used to identify & exclude transfers from dataset	Integer	1= Unique ID 2= Source of Admission 3= Disposition 9= Not applicable	1-3, 9	1
HEALTHOUTCOMEID	Health outcome (Asthma, Carbon monoxide poisoning, or Heat stress illness)	Integer	1=Asthma 3=Carbon monoxide poisoning 4=Heat stress illness	1, 3, 4	1
INCIDENTCOUNTFIRE (For CO ED Visits ONLY)	Number of unintentional fire-related CO poisoning ED visits	Integer	nnnnnn If -999= Count is recorded in INCIDENTCOUNTUNKNOWN	0 to 999999, -999	6
INCIDENTCOUNTNONFIRE (For CO ED Visits ONLY)	Number of unintentional, non-fire related CO poisoning ED visits	Integer	nnnnnn If -999= Count is recorded in INCIDENTCOUNTUNKNOWN	0 to 999999, -999	6
INCIDENTCOUNTUNKNOWN (For CO ED Visits ONLY)	Number of CO poisoning ED visits where the cause was undetermined	Integer	nnnnnn	0 to 999999	6
MONTHLYVISITS	Number of monthly hospitalization events	Integer	nnnnn	0000-99999	5

**ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA DICTIONARY FOR AGGREGATE DATA
EMERGENCY DEPARTMENT (ED) VISITS**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length
OUTOFSTATEEXCLUSION	Exclusion of residents admitted to hospitals located in another state	Integer	1= Yes 2= No	1, 2	1
RACE (OPTIONAL)	Race group of individual admitted	Text	W=White B=Black O=Other U=Unknown	W, B, O, U	1
RACEETHNICITYREPORTED	Race and ethnicity fields reported in source data	Integer	1=Yes separate 2=Yes combined 3=No	1, 2, 3	1
SEX	Sex of individuals admitted	Text	M=Male F=Female U=Unknown	M, F, U	1
STATE <i>(For Header section only; Not a data element)</i>	Patient's state of residence	String	FIPS		2
TRANSFEREXCLUSION	Exclusion of transfers from dataset	Integer	1= Yes 2= No	1, 2	1

Content Domain: Hospitalizations

Recommendations for Nationally Consistent Data and Measures

***Abbreviated* Part 2: Recommended Data Sets**

Version History v3.26	
Field Name	Description
INCIDENTCOUNTFIRE	Updated the “ Code Scheme ” to clarify the use of “-999” value. The updated text now states: “If -999= Count is recorded in INCIDENTCOUNTUNKNOWN”
INCIDENTCOUNTNONFIRE	Updated the “ Code Scheme ” to clarify the use of “-999” value. The updated text now states: “If -999= Count is recorded in INCIDENTCOUNTUNKNOWN”
ADMISSIONYEAR	To match the field in the XML schema, name of the variable changed to YEARADMITTED
Version History v3.25	
Field Name	Description
HEALTHOUTCOMEID	Field has been modified to include all health outcome hospitalizations as: 1= asthma 2= acute myocardial infarction 3= carbon monoxide poisoning 4= heat stress illness
COUNT_FIRE_H	Renamed as INCIDENTCOUNTFIRE
COUNT_NONFIRE_H	Renamed as INCIDENTCOUNTNONFIRE
COUNT_UNK_H	Renamed as INCIDENTCOUNTUNKNOWN
Version History v3.24	
Field Name	Description
DAILYAVG	Field has been deleted from the data set
Version History v3.23	
Field Name	Description
MONTHLYMIN	Field has been deleted from the data set
MONTHLYMAX	Field has been deleted from the data set
Version History v3.22	
Field Name	Description
MONTHLYMIN	-999 has been added to denote missing values (9999 was used previously)
MONTHLYMAX	-999 has been added to denote missing values (9999 was used previously)
DAILYAVG	Field has been deleted from the schema. CDC will calculate from MONTHLYHOSP and # days in given month
Version History v3.21	

Field Name	Description
COUNTY	A code indicating county unknown ('U') was added.
COUNTY	FIPS code changed from 3 digit to 5 digit
RACEETHNICITYREPORTED	Code set was modified to differentiate between variable collection methods 1=yes separate 2= yes combined 3= no
TRANSFEREXCLUSION	9=unknown deleted since exclusion of transfers will never be unknown
EXCLUSIONMETHOD	9 changed to Not Applicable

ENVIRONMENTAL PUBLIC HEALTH TRACKING

AGGREGATE DATA SET SUMMARY

HOSPITALIZATIONS

Asthma and Acute Myocardial Infarction

Characteristic	Description
Data Source	State Hospital Discharge Data Systems
Purpose	This data set will be used to calculate measures related to asthma, acute myocardial infarction, carbon monoxide poisoning, and heat stress illness hospitalizations as described in the Part 1 package, for use on the national public portal.
Geographic Level	The smallest geographic unit to be represented in this data set is the county.
Restrictions	This is a restricted access data set. Data will be displayed via the national public portal only when sufficient conditions have been met to protect data privacy. Only registered users will have direct access to this data set via the national secure portal.

NOTE: When preparing the data file, the sequence of data elements (i.e. field names) in the data file can be found in the schema for each health outcome. For example: The sequence of data elements (i.e. field) for health outcome CO poisoning:

```
<RowIdentifier>3</RowIdentifier>
<AdmissionMonth>01</AdmissionMonth>
<AgeGroup>19</AgeGroup>
<County>06001</County>
<Ethnicity>H</Ethnicity>
<ExclusionMethod>9</ExclusionMethod>
<HealthOutcomeID>3</HealthOutcomeID>
<MonthlyHosp>10000</MonthlyHosp>
<OutOfStateExclusion>1</OutOfStateExclusion>
<Race>W</Race>
<RaceEthnicityReported>2</RaceEthnicityReported>
<Sex>M</Sex>
<TransferExclusion>1</TransferExclusion>
<YearAdmitted>2000</YearAdmitted>
<IncidentCountFire>0</IncidentCountFire>
<IncidentCountNonFire>0</IncidentCountNonFire>
<IncidentCountUnknown>0</IncidentCountUnknown>
```

**ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA DICTIONARY FOR AGGREGATE DATA
HOSPITALIZATIONS**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length
ADMISSIONMONTH	Month of hospital admission	String	mm	01–12	2
AGEGROUP	Five-year age group of individuals hospitalized for the condition indicated by HEALTHOUTCOMEID	Integer	1= 0–4 years 2= 5–9 years 3= 10–14 years 4= 15–19 years 5= 20–24 years 6= 25–29 years 7= 30–34 years 8= 35–39 years 9= 40–44 years 10= 45–49 years 11= 50–54 years 12= 55–59 years 13= 60–64 years 14= 65–69 years 15= 70–74 years 16= 75–79 years 17= 80–84 years 18= 85+ years 19= Unknown	1–19	2

**ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA DICTIONARY FOR AGGREGATE DATA
HOSPITALIZATIONS**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length
COUNTY	County of residence	String	FIPS U= Unknown	00000–99999 U	5
ETHNICITY (optionally reported)	Ethnicity of individuals	Text	H= Hispanic NH= Non-Hispanic U= Unknown	H, NH, U	2
EXCLUSIONMETHOD	Variable used to identify & exclude transfers from dataset	Integer	1= Unique ID 2= Source of Admission 3= Disposition 9= Not applicable	1-3, 9	1
HEALTHOUTCOMEID	Health outcome (Asthma, Acute myocardial infarction, Carbon monoxide poisoning, or Heat stress illness)	Integer	1= Asthma 2= Acute myocardial infarction 3= Carbon monoxide poisoning 4= Heat stress illness	1, 2, 3, 4	1
INCIDENTCOUNTFIRE (For CO Hospitalizations ONLY)	Number of unintentional fire-related CO poisoning hospitalizations	Integer	nnnnnn If -999= Count is recorded in INCIDENTCOUNTUNKNOWN	0 to 999999, -999	6
INCIDENTCOUNTNONFIRE (For CO Hospitalizations ONLY)	Number of unintentional, non-fire related CO poisoning hospitalizations	Integer	nnnnnn If -999= Count is recorded in INCIDENTCOUNTUNKNOWN	0 to 999999, -999	6

**ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA DICTIONARY FOR AGGREGATE DATA
HOSPITALIZATIONS**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length
INCIDENTCOUNTUNKNOWN (For CO Hospitalizations ONLY)	Number of CO poisoning hospitalizations where the cause was undetermined	Integer	nnnnnn	0 to 999999	6
MONTHLYHOSP	Number of monthly hospitalization events	Integer	nnnn	0000–9999	4
OUTOFSTATEEXCLUSION	Exclusion of residents admitted to hospitals located in another state	Integer	1= Yes 2= No	1, 2	1
RACE (optionally reported)	Race of individuals	Text	W= White B= Black O= Other U= Unknown	W, B, O, U	1
RACEETHNICITYREPORTED	Race and ethnicity fields reported in source data	Integer	1= Yes separate 2= Yes combined 3= No	1, 2, 3	1
SEX	Sex of individuals admitted	Text	M= Male F= Female U= Unknown	M, F, U	1
STATE (For Header section only; Not a data element)	State of residence	String	FIPS		2

**ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA DICTIONARY FOR AGGREGATE DATA
HOSPITALIZATIONS**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length
TRANSFEREXCLUSION	Exclusion of transfers from dataset	Integer	1= Yes 2= No	1, 2	1
YEARADMITTED	Year of hospital admission	Integer	yyyy	20XX	4

HOW-TO GUIDE

Asthma Emergency Department (ED) Visits

Environmental Public Health Tracking

07-02-2013

Data Source	Emergency Department (ED) Visits
NCDM Requirements	<ul style="list-style-type: none"> • Health Outcome (Asthma) • State/County of Residence • ED Visit Year/Month • Age Group • Sex • Race/Ethnicity (optional) • Transfers not to be excluded • Out-state residents to be excluded • ED visits to federal facilities to be excluded • ED visits of residents to out-of-state hospitals are to be optionally included
Measures Generated	<ul style="list-style-type: none"> • Annual Number of ED Visits by age group, sex, race/ethnicity*, and county and state • Annual Crude (unadjusted) Rate of ED Visits by age group, sex, race/ethnicity*, and county and state • Annual Age-Adjusted Rate of ED Visits for all ages by sex, race/ethnicity*, and county and state • (optional**) Average Number of ED Visits per Month by age group, sex, race/ethnicity*, and county and state • (optional**) Daily Number of ED Visits by age group, sex, race/ethnicity*, and county and state <p>* measures by race/ethnicity are optional</p> <p>** optional measures for state portal only and not submitted to CDC</p>
Definitions	<p>■ <i>Asthma</i>: A common chronic inflammatory disease of the airways characterized by variable and recurring symptoms, airflow obstruction, and bronchospasm and by reversible obstruction of the airways; ICD-9 493.XX.</p> <p><i>Duplicate record</i>: More than one record for the same person with the same ED Visit data (e.g., sex, date of birth, admission/ED Visit date, and zip code have exact same information). Duplicate records may also be due to continuation of data beyond a single line. In this case, duplicates may be identified using a record sequence number.</p> <p><i>ED Visit date</i>: The calendar date of the ED Visit:</p> <ul style="list-style-type: none"> ■ Day (optional)

	<ul style="list-style-type: none"> ▪ Month (required) ▪ Year (required) <p><i>ED Visit Year:</i> An ED Visit for asthma during a specific calendar year. ED Visit year is based only upon the calendar year of the Visit, even when discharge and/or release year is different.</p> <p><i>Emergency Department Visit:</i> Treatment in a hospital emergency department. This should include both patients who are treated and released and those that are admitted as inpatients from the emergency department.</p> <p><i>Hospital Transfers:</i> The practice of discharging a patient from one facility and readmitting them to a second facility within 48 hours.</p> <p><i>ICD-9-CM code:</i> International Classification of Diseases, 9th Revision, Clinical Modification</p> <p><i>Multiple visits:</i> More than one ED Visit for the same person for the same diagnosis code occurring on different dates and related to a separate event within a given year. Multiple ED Visits are considered separate events if they occurred more than 48 hours apart.</p> <p><i>Observation Stay:</i> This is an alternative to inpatient admission that exists in some facilities but for EPHT is considered in ED Visit statistics. Observation Stays may originate as an ED Visit or directly as an Observation Stay. Note that the definition of an Observation Stay may not be standard across hospitals, and Observation Stays may not be recorded across states in a consistent manner.</p> <p><i>Primary Diagnosis Code:</i> The first diagnosis field(s) of the coded clinical record (i.e., primary or principal diagnosis). Presently, the code is represented by an ICD-9-CM code (the International Classification of Diseases, 9th Revision, Clinical Modification). The code for asthma is 493.XX.</p> <p><i>Resident:</i> A person who resides in the grantee's state/county (permanently or for an extended period) at the time of the ED Visit.</p>
How-to-Guide Requirements and Cautions	<ul style="list-style-type: none"> • This How-to-Guide has two purposes: 1) Guide the user in the development of the XML dataset using the SAS code, located on SharePoint, for submission to CDC; 2) Describe the calculation steps for creating the Nationally Consistent Data Measures (NCDMs). Grantees should use the How-to-Guide in order to ensure that the calculation of measures are consistent with those required by CDC in the preparation of their own computer code. • Note that one set of SAS code (not provided by CDC's EPHT program) is designed to handle the creation of XML files for all hospitalization (inpatient) outcomes (separate code is available for all ED Visit outcomes). The code manages the differences in definitions between the outcomes and generates a separate file for each outcome by each single year. • This How-to-Guide and the optional SAS code not provided by CDC's EPHT program) presume that the user has removed duplicate records while keeping multiple ED Visits. A case should be counted once per ED Visit; de-duplication of

records to achieve this goal should be conducted at the discretion of the data owners, managers, and/or analysts.

- ED Visits include both patients who are admitted to the hospital through the emergency department (inpatients) and those who are treated and released (outpatients); therefore, both inpatient and outpatient data are required for this indicator. If identified and/or stored separately, observation stay data should be included as well.
- In the event that an ED Visit occurred at the end of a calendar year and the discharge date occurred in the following year, the dataset that includes the discharge date will be required before the dataset can be considered complete.
- The How-to-Guide steps do not incorporate data suppression and/or aggregation rules. Suppression guidelines are separately applied by CDC for the national portal and by grantees for state portals.
- Admissions of residents to out-of-state hospitals are not required to be included. However, when available these admissions should be included. It is noted that some states must include out-of-state admissions of its residents. Use the “OUTOFSTATEEXCLUSION” variable in the dataset to capture whether out-of-state admissions are included or not (the Data Dictionary and schema provide for formal notation in the dataset on whether these admissions are included). Be certain to use footnotes and metadata to acknowledge the disposition of these admissions.
- ED Visits for individuals who are not state residents should be excluded. If a data steward’s database includes these cases, exclude them from the EPHT database. If they cannot be excluded, footnotes and metadata should acknowledge that these cases are included.
- Patients transferred from or to other acute care facilities should be included. Indicate in footnotes and/or metadata if transfers are excluded.
- Patients with an ED Visit at a federal facility should not be included. If a data steward’s database includes these cases, exclude them from the EPHT database. If they cannot be excluded, footnotes and metadata should acknowledge that these cases are included.
- Although hospital discharge data are collected using a standard format across states, there are considerable differences in the variable attributes; for example, response categories may differ between states for “source of admission” and “disposition” variables. These differences may reflect how certain variables are collected, whether the reporting of a variable (for example patient name or race) is mandatory, and/or differences in data availability and access agreements. The number of diagnosis fields available in the discharge data also varies by state, ranging from nine to an unlimited number. In addition, the data vary by state in regard to data quality such as the validity or completeness of specific fields. In all cases, the data analyst should work closely with the data managers in order to understand the nuances of the data.

	<ul style="list-style-type: none"> The Data Dictionary in SharePoint should be referred to for the standardized definitions and notations of the variables to be submitted to CDC.
<p>NOTE FOR NON-SAS USERS: Grantees not using SAS should use the steps outlined below for guidance on important issues such as population requirements and should refer to the Data Dictionary for specifications of the required fields. The data file should be converted to the .XML file format and the required header inserted into the XML file, according to the Schema found on SharePoint. In addition, refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data Dictionary).</p> <p>NOTE FOR SAS USERS: SAS code is not developed or distributed by CDC.</p>	
<p>Section A: CREATION OF REQUIRED DATA FILE FOR NCDMs</p>	
<p>Step #1</p>	<p><i>Identifying the data sources for ED Visits:</i></p> <p>ED Visits include both patients who are treated and released in the ED (outpatients) and who are admitted as inpatients through the emergency department; therefore, <u>both</u> inpatient and outpatient data files are required for this indicator. If identified separately, observation stay data files are also required.</p>
<p>Step #2</p>	<p><i>Identifying ED Visits for asthma</i></p> <p>a. From ED data, select all records that meet the following criteria:</p> <ul style="list-style-type: none"> Select all records with asthma (ICD-9-CM = 493) listed as the first-listed/primary/principal diagnosis. Exclude all records where the State of residence is not your state. <p>b. From inpatient hospitalization data, select all records that meet the following criteria:</p> <ul style="list-style-type: none"> Select all records with asthma (ICD-9-CM = 493) listed as the first-listed/primary/principal diagnosis. Restrict the dataset to patients who were admitted from an ED using the following criteria: <ul style="list-style-type: none"> point of origin code indicates emergency department, or CPT codes: 99281-99285, or revenue codes: 0450-0459, or positive ED charges These criteria are consistent with the criteria used by AHRQ (see: http://www.hcup-us.ahrq.gov/db/vars/siddistnote.jsp?var=hcup_ed). Exclude all records where the State of residence is not your state. <p>c. From Observation Stay data</p> <p>In states where observation stays are identified separately, include these observation stay records with ED Visits. Not all states require the reporting of observation stay records. Contact data stewards to determine whether records for observation stays are collected and if so, if the records are located with outpatient or inpatient records, or in</p>

	<p>a separate file. Observation Stays can be identified by selecting all the records that meet the following criteria:</p> <ul style="list-style-type: none"> • Select all records with asthma (ICD-9-CM = 493) listed as the first-listed/primary/principal diagnosis and <ul style="list-style-type: none"> ○ revenue code: 762, or ○ positive OS charge when revenue codes not available, or ○ CPT codes: 99217-99220 or 99234-9923 • These criteria are consistent with the criteria used by AHRQ (see: http://www.hcup-us.ahrq.gov/db/vars/siddistnote.jsp?var=hcup_ed).
Step #3	<p><i>Identifying the required data file content</i></p> <p>Each record should include the following variables:</p> <ul style="list-style-type: none"> • Primary diagnosis code • Date of admission • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient's sex • Patient's race (optional) – White, Black, Other, Unknown • Patient's ethnicity (optional) – Hispanic, Non-Hispanic, Unknown • County of residence • State of residence <p><i>Data Specifications</i></p> <p>Refer to the Data Dictionary in order to conform with the coding specifications required for the NCDM variables. Note that the county FIPS code is 5 digits and that the first two digits will be the same as the state FIPS code. Note also that some variables are optional.</p> <ul style="list-style-type: none"> • For SAS users, the admission date (and discharge and birth date) variable is acceptable in the following formats: <ul style="list-style-type: none"> ○ SAS DATE FORMAT ○ MMDDYYYY ○ MMDDYY ○ MM/DD/YYYY ○ MM-DD-YYYY ○ DDMONYYYY ○ DDMONYY ○ YYYYMMDD ○ DDMONYYYY:00:00:00 • The base format for counts and population data should be by 5-year age groups beginning 0-4 and ending with 85+. ED Visit counts must be submitted to CDC by these 5-year age groups (see Data Dictionary). Refer to the measure-specific step for the appropriate age groups for calculation and

	<p>presentation. In summary, the ED Visit and population age-groups required for the calculation and presentation of measures are:</p> <ul style="list-style-type: none"> ○ Asthma counts to CDC: 5-yr age-groups (0–4, 5–9 ... 85+) ○ Asthma age-specific rate presentation: 0–4, 5–14, 15–24, 25–34, 35–44, 45–54, 55–64 and 65+ ○ Asthma crude and age-adjusted rates: 5-yr age groups <ul style="list-style-type: none"> ● Race and ethnicity variables are optional. Therefore, data files and counts and measures may be generated without specifying race or ethnicity if these data are missing or considered unreliable/inaccurate. If race and ethnicity data are reported, the NCDMs will only be generated when the source variables for race and ethnicity are separate variables. The NCDMs will not be generated if race and ethnicity are reported as a combined race/ethnicity variable. If race and ethnicity data are being provided make sure that the coding structure conforms to that described in the Data Dictionary. <ul style="list-style-type: none"> ○ Race: White; Black; Other; Unknown. ○ Ethnicity: Hispanic; non-Hispanic; Unknown. <p>Please consult your data steward and data managers to understand what types of ED Visits are included and excluded (e.g., resident out-of-state ED Visits) and the available variables and coding system (e.g., some data stewards may code race and ethnicity as one variable whereas others may code them as separate variables).</p>
Step #4	<p><i>Selecting records for year of interest:</i> 2000–present calendar year. The SAS program (not provided by CDC) prepares one NCDM file for each year, as required for CDC submission.</p> <p>Most ED Visit data are released in annual discharge-based datasets. In order to have complete admission date information using discharge-based datasets, it is necessary to have the dataset of the year of interest <u>and</u> the subsequent year. Since hospital data is based on discharge year and the EPHT dataset is to be based on admission date, patients discharged in current year (i.e., 2006) but admitted to the ED in the previous year (i.e., 2005) should be counted as 2005 ED Visits.</p>
Step #5	<p><i>Removal of duplicates:</i> This How-to-Guide and accompanying SAS code presumes that the user has already removed any duplicate records, while keeping multiple ED Visits.</p> <p>The following variables may be used to identify duplicate records: hospital code, medical record number, admission date, discharge date, date of birth, sex, and zip code.</p> <p>Duplicate records may also be due to continuation of data beyond a single record line. In this case, duplicates may be identified using a record sequence number.</p> <p>GO TO SECTION B FOR INSTRUCTIONS ON CREATING AN XML FILE IF NOT USING SAS.</p> <p>GO TO SECTION C OR INSTRUCTIONS ON CREATING AN XML FILE IF USING SAS.</p>
<p>Section B: CREATION OF XML DATA FILE FOR NCDMS WHEN NOT USING OPTIONAL</p>	

SAS CODE	
Step #1	<p><i>Required data file:</i> Each record should include the following variables:</p> <ul style="list-style-type: none"> • Primary diagnosis code • Date of visit • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient's sex • Patient's race (optional) – White, Black, Other, Unknown • Patient's ethnicity (optional) – Hispanic, Non-Hispanic, Unknown • County of residence • State of residence
Step #2	Create required fields according to the specifications of each field provided in the Data Dictionary.
Step #3	Convert the data file to the .XML file format and insert the required header into the XML file, according to the Schema found on SharePoint. Refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data Dictionary).
Step #4	<p>Submit completed XML file to CDC using PHIN-MS.</p> <p><i>GO TO SECTION D FOR INSTRUCTIONS ON CALCULATING MEASURES FOR GRANTEE PORTALS.</i></p>
Section C: CREATION OF XML DATA FILE AND NCDM FILE FOR NCDMS USING OPTIONAL SAS CODE	
Step #1	<p><i>Create SAS Datasets:</i> Be sure to make copies of the inpatient and outpatient data before proceeding. If the ED Visit data is not in SAS format, convert it to SAS format and save it as a permanent dataset for creating the XML and measures. Keep the following variables in the SAS dataset :</p> <ul style="list-style-type: none"> • Primary diagnosis code • Date of admission • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient's sex • Patient's race and ethnicity (<i>optional</i>) • County of residence • State of residence

Step #2	<p><i>Create XML File:</i> After creating the SAS datasets, download ED-NCDM containing the recommended SAS code and Users Guide. These can be found on SharePoint. The current version will only create xml files (not measures).</p> <p>Unzip the files and read and follow the User’s Guide.</p> <p>Click ED-NCDM.msi to launch the setup wizard. Double click the shortcut icon to launch the program.</p> <p>XML files for each single year of data will be generated for one outcome, as required for CDC submission.</p>
Step #3	<p><i>Creating Measures using SAS:</i> SAS menu options 3–9 generate the required NCDMs. These options are currently non-operational. These options will follow the steps described below in Section D. If selecting menu options 3–9, this SAS program will generate a final output in the form of MS Excel spreadsheet.</p> <p>Keep only those records that meet the following criteria:</p> <ul style="list-style-type: none"> • First-listed diagnosis code = 493 • State = Your state • Date of Birth (if available and being used to calculate patient age) is not missing • Patient’s age at the time of ED Visit is not missing • Date of Discharge is not missing • Date of Admission is not missing. <p><i>Population Data:</i> US Census bureau residential population data for state and county. Intercensal population estimates should be used for the intercensal years, and population extrapolations for postcensal years.</p>
Section D: GENERATE MEASURES FOR GRANTEE PORTALS	
ANNUAL NUMBER OF ED VISITS	
Step #1	Annual Number of ED Visits by sex and total
	<p><i>State:</i> Calculate the number of visits during the year of interest by sex (male, female, and unknown /missing). Then sum the number of visits across sex (male + female + unknown/missing) for the total annual number of ED Visits for the state.</p> <p><i>County:</i> Calculate the number of visits during the year of interest by county of residence and sex (male, female, unknown/missing). Then sum the number of visits across sex (male + female + unknown/missing) to get the total annual number of ED Visits by county of residence.</p>
Step #2	Annual number of ED Visits by race and total (<i>optional</i>)
	<p><i>State:</i> Calculate the number of visits during the year of interest by race (white, black, other, unknown). Then sum the number of visits across race (white + black + other + unknown) for the total annual number of ED Visits for the state.</p> <p><i>County:</i> Calculate the number of visits during the year of interest by county of residence and race (white, black, other, unknown). Then sum the number of visits across race (white + black + unknown) to get the total annual number of ED Visits by</p>

	county of residence.
Step #3	Annual number of ED Visits by age groups and total
	<p><i>State:</i> Calculate the number of visits during the year of interest for each 5-year age specific category (0–4, 5–9... 85+). Then sum the number of visits across all age groups to get the total annual number of ED Visits for the state.</p> <p><i>County:</i> Calculate the number of visits during the year of interest by county of residence and each 5-year age category (0–4, 5–9... 85+). Then sum the number of visits across all age categories to get the total annual number of ED Visits for each county of residence.</p>
<i>MONTHLY NUMBER OF ED VISITS (optional)</i>	
Step #4	Average Number of ED Visits per Month (<i>optional</i>)
	<p>NOTE: Average number of ED Visits per month is <u>not</u> a required NCDM and is not submitted to CDC or required to be placed on grantee portals. Because of the potential future use of this measure, it is included in the How-to-Guide.</p> <p><i>State:</i> Calculate the number of ED Visits for the state for a given month during the year of interest. Then divide the monthly totals by the number of days in that month (i.e. the denominator for January would be 31), adjusting for leap years when necessary.</p> <p><i>County:</i> Calculate the number of ED Visits by county of residence for the year of interest. Then divide the monthly total by the number of days in that month (i.e. the denominator for January would be 31), adjusting for leap years when necessary.</p>
<i>DAILY NUMBER OF ED VISITS (optional)</i>	
Step #5	<p>NOTE: Daily number of ED Visits is <u>not</u> a required NCDM and is not submitted to CDC or required to be placed on grantee portals. Because of the potential future use of this measure, it is included in the How-to-Guide.</p> <p>Sum the total number of ED Visits for each day by sex during the year of interest for the entire state by sex. Add the daily number of male, female, and unknown (including missing sex information) to obtain the total number of daily admissions. Repeat the above by race/ethnicity and 5-year age groups to calculate the daily number of ED Visits by race/ethnicity and age groups.</p>
ANNUAL UNADJUSTED (CRUDE) RATE OF ED VISITS	
Step #6	Annual ED Visit rate by sex and total per 10,000 population
	<p>Exclude any observation where sex is unknown or missing. Sum the ED Visits for male and female to obtain the total ED Visits for each age group.</p> <p>Use U.S. Census Bureau residential population data for state and county (see Section C, Step #3).</p> <p><i>State (required)</i></p> <ul style="list-style-type: none"> <i>Numerator:</i> The annual number of ED Visits for males, females, and total for

	<p>the year of interest</p> <ul style="list-style-type: none"> • <i>Denominator:</i> The population for the state for males, females, and total. • <i>Constant:</i> 10,000 • <i>Formulas:</i> <p>Unadjusted (Crude) Rate for males per 10,000 people = # of male ED Visits / total male state population × 10,000</p> <p>Unadjusted (Crude) Rate for females per 10,000 people = # of female ED Visits / total female state population × 10,000</p> <p>Unadjusted (Crude) Rate for total population per 10,000 people = (# male + # female) annual ED Visits / total state population × 10,000</p> <p><i>County (required)</i></p> <ul style="list-style-type: none"> • <i>Numerator:</i> The annual number of ED Visits for males, females, and total by county of residence for the year of interest. • <i>Denominator:</i> The population for each county of residence in the state for males, females, and total. • <i>Constant:</i> 10,000. • <i>Formulas:</i> <p>Unadjusted (Crude) Rate for males per 10,000 people = # of male ED Visits for each county of residence / total male county population × 10,000</p> <p>Unadjusted (Crude) Rate for females per 10,000 people = # of female ED Visits for each county of residence / total female county population × 10,000</p> <p>Unadjusted (Crude) Rate for total population per 10,000 people = (# of male annual ED Visits + # of female annual ED Visits) for each county of residence / total county population × 10,000</p>
--	--

ANNUAL AGE ADJUSTED RATE OF ED VISITS

Step #7	Annual age-adjusted rate of ED Visits by sex and total per 10,000 population
---------	--

	<p>Exclude any ED Visit observation where sex is unknown or missing. Sum the admissions for male and female to obtain the totals for each age-group. To calculate age-specific rates (for the 5-year age categories 0-4, 5-9...85+), use US Census bureau residential population data for denominators. Intercensal population estimates should be used for the intercensal years. The standard population should be the 2000 U.S. Standard Population divided into 18 age groups (http://seer.cancer.gov/stdpopulations/). For tutorial on age-adjustment see http://seer.cancer.gov/seerstat/tutorials/aarates/definition.html.</p> <p><i>State:</i></p> <ul style="list-style-type: none"> • Calculate age-specific rates for male, female and total for 5-year age groups (0-4,
--	---

	<p>5–9, ..., 85+) by dividing the number of state ED Visits in that age group and sex by the Census state population of same age group and sex.</p> <ul style="list-style-type: none"> • Compute age-adjustment population weights for male, female and total for 5-year age groups using the 2000 US Standard population as follows: Age-adjusted weight = age-specific std pop/total std pop. • Multiply the age-specific rate × age adjustment weight for each age group for male, female and total. • Compute age-adjusted ED Visit rate for male, female and total by summing the product of the previous step for each age group i: $\sum(\text{rate}_i \times \text{weight}_i)$ <p><i>County:</i></p> <ul style="list-style-type: none"> • Calculate age-specific rates for male, female and total for 5-year age groups (0-4, 5-9, ..., 85+) by dividing the number of county ED Visits in that age group and sex by the Census county population of same age-age group and sex. • Compute age-adjustment population weights for male, female and total for 5-year age groups using the 2000 US Standard population as follows: Age-adjusted weights = age-specific std pop/total std pop. • Multiply the age-specific rate × age adjustment weight for each age group for male, female and total. • Compute age-adjusted ED Visit rate for male, female and total by summing the product of the previous step for each age group i: $\sum(\text{rate}_i \times \text{weight}_i)$ <p><i>Confidence Intervals (optional):</i></p> <ul style="list-style-type: none"> • 95% confidence intervals may be calculated. <p>Lower Confidence Limit (LCL) = [age-adjusted rate – { 1.96 × age-adjusted rate / SQRT (Number of ED Visits)}]</p> <p>Upper Confidence Limit (UCL) = [age-adjusted rate + { 1.96 × age-adjusted rate / SQRT (Number of ED Visits)}]</p> <p>Please Note: With small numbers of ED Visits (i.e. ED Visits < 20), calculation methods assuming a non-normal distribution may be more appropriate.</p>
Section E: PRESENTATION & DISPLAY	
Aggregation & Suppression	Follow your state’s rules, laws, and regulations as well as rules and agreements between you and your data partner(s) in determining whether and when small cell values need to be suppressed.
Visual display	<p>If using optional SAS code, export SAS datasets to Microsoft Excel® for creating charts or for formatting the computed values for presentation.</p> <p>Aggregations calculated under the “Data Measurements” should be displayed, using Microsoft Excel® or equitable spreadsheet product, by state at a minimum and if available and appropriate by county. Recommended spreadsheet displays include:</p>

	<ul style="list-style-type: none"> • Annual number of asthma ED Visits by state and county • Unadjusted (crude) rate of asthma ED Visits by state and county • Age-Adjusted rate of asthma ED Visits by state and county • Average number of daily visits per month by state and county <p>Annual number of ED Visits can be displayed by showing sex on x-axis and number of ED Visits on y-axis. Similarly sex on x-axis can be replaced by race or age groups to display the number of ED Visits by race or age-group. Displays by race and sex are optional.</p> <p>These bar charts can be created by using any spreadsheet application or by using SAS.</p> <p>Pie charts and bar charts should be used as supplementary visual displays in conjunction with spreadsheets for aggregated calculations.</p> <p>Mapping of calculated counts and rates should be done on the county level. Recommended maps include:</p> <ul style="list-style-type: none"> • Annual number of asthma ED Visits by county per year • Age-Adjusted rate of asthma Ed Visits by county per year <p>Public can view bar charts and map showing the state and county level asthma measures. These visual displays will show whether the selected geographical area has a high, medium high, medium low, or low ED Visits rate. The public will also be able to see the links to other related information from various national, state and local sources.</p> <p>Mapping the rate of ED Visits per 10,000 residents will allow users to assess the level of environmentally related risk factors in their residential geographic area as well as in the surrounding geographical area. Future mapping may include other risk factors such as poverty and other potential risk factors. One limitation of mapping is that not all sources of asthma risk factors may be mapped. For example, indoor mold, dust, and pollen.</p>
Interpretation	<p><i>Small Numbers:</i> Measures that generate counts or rates based upon numbers that are small and potentially violate state privacy guidelines can collapse data across years or geographic units to generate data that can be released.</p> <p><i>Measures for multiple years:</i> The how-to-guide steps can be repeated for additional years of ED Visit data. Multi-year ED Visit data can be merged to create one dataset. Add the number of ED Visits for each year in multi-year cohort and divide by the number of years to calculate an average annual number of ED Visits.</p>

Indicator Template
Content Area: Asthma
Indicator: Emergency Department Visits for Asthma
Environmental Public Health Tracking

Type of EPHT Indicator	Health outcome
Measures	<ol style="list-style-type: none"> 1. Annual age-adjusted rate of emergency department visits for asthma per 10,000 population 2. Annual crude rate of emergency department visits for asthma per 10,000 population 3. Annual number of emergency department visits for asthma
Derivation of Measure(s)	<p><i>Numerator:</i></p> <ul style="list-style-type: none"> • Emergency department visits during a calendar year with asthma (ICD-9-CM 493) as the primary diagnosis (include records for ED Visits resulting in a hospitalization) • Both inpatient and outpatient records with duplicate* records removed and transfers to other hospitals included. <p>*Duplicate records refer to more than one record for the same person for the same event (with the same ED Visit data e.g., sex, date of birth, admission/ED Visit date, and Zip Code have exact same information).</p> <p><i>Denominator:</i></p> <ul style="list-style-type: none"> • Annual population estimates for state and county from U.S. Census Bureau <p><i>Adjustment:</i></p> <ul style="list-style-type: none"> • Age-adjustment by the direct method to the Year 2000 US Standard population
Unit	<ol style="list-style-type: none"> 1. Age-adjusted rate per 10,000 population 2. Rate per 10,000 population 3. Number
Geographic Scope	State and county
Geographic Scale	Residents of jurisdiction – State, County
Time Period	Emergency department visits with admission dates from January 1 through December 31, inclusive, for each year
Time Scale	Annual
Rationale	Asthma continues to be a serious public health problem; asthma prevalence increased from 7.3% in 2001 to 8.4% in 2010. ¹ In 2010, more than 25 million people including 7 million children (0–17 years) had asthma. ¹ In 2008, there were 456,000 hospitalizations and 1.8 million emergency department visits (ED) for asthma. ² Asthma is a leading chronic health condition among children. The

greatest rise in asthma rates was among black children (almost a 50% increase) from 2001 through 2009.² There are also large racial, income, and geographic disparities in poor asthma outcomes.^{1,6}

As a chronic respiratory disease, asthma can interfere with everyday activities. According to CDC Vital Signs 2011 report, more than half (59%) of children and one-third (33%) of adults who had an asthma attack missed school or work because of asthma in 2008.³ In 2007, there were over 3,400 deaths in which asthma was the underlying cause.³

Despite the availability of effective prevention measures, asthma-associated costs are increasing. Asthma cost the US about \$3,300 per person with asthma each year from 2002 to 2007 in medical expenses.³ Medical expenses associated with asthma increased from \$48.6 billion in 2002 to \$50.1 billion in 2007.³

Environment Attributable Fractions of the 1988–1994 economic costs for asthma were 39.2% for children <6 years of age and 44.4% for 6–16years of age, costing more than \$400 million for each age group.⁴

Associations between environmental exposures and asthma have been consistently demonstrated.^{6,7,8,9} Many outdoor air pollutants have been associated with increased asthma ED visits.^{10,11,12,13,14} There is strong scientific evidence for direct associations between increased ozone concentrations and increases in asthma ED visits, in children and adults.^{11,12} In one study, asthma ED visits increased by 33 percent when daily 1-hour maximum ozone concentration exceeded 75 ppb;⁹ another study reported 26% increase in ED visits when the daytime mean ozone concentration exceeded 60ppb.¹⁰ Associations between asthma-related ED visits and ambient air particulate matter—both PM₁₀ and PM_{2.5}—have been repeatedly observed, and are especially robust for children.^{12, 13} Other pollutants related to higher asthma ED visit totals include carbon monoxide (CO), nitrogen dioxide (NO₂), and pollution from automobiles, coal, and petrochemical sources.^{14,15} Other outdoor environmental triggers for asthma ED visits in children include pollen, and ambient temperature. Increased asthma ED visits has also been associated with environmental tobacco smoke (ETS).¹⁶

The state emergency department visit data are electronically maintained and are available in almost every state in the U.S. The data have comparable basic information about each visit and can provide a more sensitive tracking measure of asthma exacerbation than inpatient hospitalization. These measures can be used to evaluate the impact of ambient air pollution on respiratory health of children and adults. Also, the measures can be used for better resource management to further reduce asthma-related expenditures. Combined with inpatient asthma data, emergency department data will provide more complete spatial and temporal trends for asthma.

Use of the Measure

The development of a single analytic method for asthma emergency department visits among persons living in state will inform multiple users:

	<p><i>State:</i></p> <ul style="list-style-type: none"> • May be linked with other risk factors such as air pollution to identify susceptible populations and explore ecologic relationships • Allows for a better understanding of what the asthma surveillance data represents when interpreting number of inpatient hospitalizations • Permits the monitoring of trends temporally and spatially <p><i>National:</i></p> <ul style="list-style-type: none"> • It will allow for comparison across states which can be used to target interventions (especially for CDC and EPA). <p><i>Public:</i></p> <ul style="list-style-type: none"> • Public and concerned community members will be able to view the Tracking Network webpage and learn the annual rate of asthma emergency department visits and burden of asthma in their state or county.
<p>Limitations of the Measure</p>	<ul style="list-style-type: none"> • Numbers may be too small in rural areas to calculate stable rates. • The timing of the exposure may not correspond with the timing of the asthma exacerbation leading to the ED visit. • Individuals may have asthma exacerbations due to exposure to an environmental risk factor that does not result in an ED visit and thus are not captured in this measure. • Differences in rates by time or area may reflect differences or changes in diagnostic techniques and criteria and in the coding of asthma. • Reporting rates at the state and/or county level will not show the true asthma burden at a more local level (i.e., neighborhood). • Differences in rates by area may be due to different socio-demographic characteristics and associated behaviors. • When comparing rates across geographic areas, a variety of non-environmental factors, such as access to medical care, can impact the likelihood of persons treated at ED for asthma. • Reporting rates at the state and/or county level may not have sufficient geographic resolution to be linked with many types of environmental data. • When looking at small geographic levels users must take into consideration appropriate cell suppression rules imposed by the data providers or individual state programs. • Although duplicate records for the same ED visit are excluded, the measures are based upon events, not individuals, because no unique identifier is always available. When multiple admissions for the same person during the year are not identified, the resulting rate is not the proportion of the population that has an asthma ED visit. Rather it is the number of events per 10,000 population which is an overestimate of the proportion. Even at the county level, it can be expected that the measures generated will often be based upon numbers too small to report or present without violating state and federal privacy guidelines and regulations. Careful adherence to cell

	<p>suppression rules in cross tabulations is necessary and methods to increase cell sizes by combining data across time (e.g., months, years) and geographic areas may be appropriate.</p>
Data Sources	<p><i>Numerator:</i> State emergency department data <i>Denominator:</i> US Census Bureau population data</p>
Limitations of Data Sources	<p><i>State emergency department data:</i></p> <ul style="list-style-type: none"> • ED visits for asthma are only one piece of a larger picture that describes asthma burden. • Veteran’s Administration, Indian Health Service and institutionalized (e.g., prisoners) populations are excluded • In-state residents who visit an ED in surrounding states would not be included unless states have emergency department data sharing agreements. • Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers. • Sometimes mailing address of patient (e.g., P.O. Box) is listed as the residence address of the patient • Patients may be exposed to environmental triggers in multiple locations, but ED geographic information is limited to residence.
Related Indicators	<ul style="list-style-type: none"> • Asthma prevalence among adults • Asthma prevalence among children • Hospitalizations for asthma
References	<ol style="list-style-type: none"> 1. Akinbami LJ, Moorman JE, Bailey C, Zahran HS, King M, Johnson CA, Liu X. Trends in Asthma Prevalence, Health Care Use, and Mortality in the United States, 2001–2010. 2. Akinbami LJ, Moorman JE, Liu X. Asthma Prevalence, Health Care Use, and Mortality: United States, 2005–2009. National Health Statistics Reports; No 32. Hyattsville, MD: National Center for Health Statistics, 2011. 3. Centers for Disease Control and Prevention. Vital Signs report 2011: Asthma in the US. http://www.cdc.gov/vitalsigns/Asthma/index.html 4. Lanphear BP, Aligne CA, Auinger P, et al. Residential exposures associated with asthma in US children. <i>Pediatrics</i> 2001; 107: 505-511. 5. Britton JR, Lewis SA. Epidemiology of childhood asthma. In <i>Asthma: Epidemiology, Anti-Inflammatory Therapy and Future Trends</i>; MA Gienbycz and BJ O’Connor (Eds.),. Switzerland: Birkhäuser Verlag, 2000, pp. 25-56. 6. Lanphear BP, Kahn RS, Berger O, et al., Contribution of residential exposures to asthma in US children and adolescents. <i>Pediatrics</i> 2001; 107: e98. 7. Redd SC. Asthma in the United States: Burden and current theories. <i>Environ Health Perspect</i> 2002; 110 (Suppl 4): 557-60. 8. Peel JL, Tolbert PE, Klein M, <i>et al.</i> Ambient air pollution and respiratory emergency department visits. <i>Epidemiology</i>. 2005; 16: 164-174. 9. Stieb DM, Burnett RT, Beveridge RC, <i>et al.</i> Association between ozone and

asthma emergency department visits in Saint John, New Brunswick, Canada. *Environ Health Perspect.* 1996; 104: 1354-60.

10. Tolbert PE, Mulholland JA, Macintosh DL, *et al.* Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia. *Am J Epidemiol.* 2000; 151: 798-810.
11. Sun HL, Chou MC, Lue KH. The relationship of air pollution to ED visits for asthma differs between children and adults. *Am J Emerg Med.* 2006; 24: 709-13.
12. Norris G, VoungPong SN, Koenig JQ, *et al.* An association between fine particles and asthma emergency department visits for children in Seattle. *Environ Health Perspect.* 1999; 107: 489-93.
13. Slaughter JC, Kim E, Sheppard L, *et al.* Association between particulate matter and emergency room visits, hospital admissions and mortality in Spokane, Washington. *J Expo Anal Environ Epidemiol.* 2005; 15: 153-9.
14. Villeneuve PJ, Chen L, Rowe BH, *et al.* Outdoor air pollution and emergency department visits for asthma among children and adults: A case-crossover study in northern Alberta, Canada. *Environ Health.* 2007; 6:40.
15. Clark NA, Demers PA, Karr CJ, Koehoorn M, Lencar C, Tamburic L, Brauer M. Effect of early life exposure to air pollution on development of childhood asthma. *Environ Health Perspect.* 2010;118(2):284-90.
16. Teach SJ, Crain EF, Quint DM, *et al.* Indoor environmental exposures among children with asthma seen in an urban emergency department. *Pediatrics.* 2006; 117: S152-8.

HOW-TO GUIDE
Asthma Hospitalizations
Environmental Public Health Tracking
07-18-2013

Data Source	Inpatient Hospitalization Admissions
NCDM Requirements	<ul style="list-style-type: none"> • Health Outcome = Asthma • State/County of Residence • Admission Year/Month • Age Group • Sex • Race/Ethnicity (optional) • Transfers not to be excluded • Out-of-State residents to be excluded • Admissions to federal facilities to be excluded • Admissions of residents to out-of-state hospitals are to be optionally included
Measures	<ul style="list-style-type: none"> ▪ Annual Number of Hospital Admissions by age group, sex, race/ethnicity*, and county and state. ▪ Annual Crude (unadjusted) Rate of Hospital Admissions for all ages by sex, race/ethnicity*, and county and state ▪ Annual Age-Adjusted Rate of Hospital Admissions for all ages by sex, race/ethnicity*, and county and state ▪ (<i>optional**</i>) Average Number of Hospitalizations per Month by age group, sex, race/ethnicity*, and county and state ▪ (<i>optional**</i>) Daily Number of Hospitalizations by age group, sex, race/ethnicity*, and county and state <p>*Measures by race/ethnicity are optional</p> <p>**Optional measures are for state portal only and not submitted to CDC</p>
Definitions	<p><i>Admission date:</i> The date of the hospital admission; month, day, and year. Month and year are required.</p> <p><i>Asthma:</i> A chronic inflammatory pulmonary disorder that is characterized by reversible obstruction of the airways; ICD-9 493.XX.</p> <p><i>Discharge date:</i> The date of discharge from hospital. When the date of the hospital discharge is not available, use the admission date, if available.</p> <p><i>Duplicate records:</i> More than one record for the same person with the same hospital admission data (e.g., where sex, date of birth, admission date, and zip code have exactly same information).</p> <p><i>Event/Event Year:</i> A hospital admission for asthma results with a primary diagnosis of</p>

	<p>493.XX during a specific calendar year. Event year is based only upon admission year, even when discharge year is different.</p> <p><i>Hospital Transfers:</i> Generally, a patient discharged from one facility and readmitted to a second facility on the same day.</p> <p><i>Hospitalization/ Hospital Admission:</i> Condition of being placed (Admission) or treated as a patient in an acute care hospital for treatment as an inpatient. Treatment as an out-patient is not considered to be hospitalization. To be considered as inpatient Hospitalization, a minimum stay is required (often over 23 hours).</p> <p><i>Multiple admissions:</i> Second or subsequent admission for the same person for the same primary diagnosis code but on a different date and related to a separate event within a given year. Multiple admissions are considered separate events (generally at least 48 hours apart).</p> <p><i>Out-of-State admissions:</i> When a resident of the grantee state is admitted to a hospital located in another state (usually an abutting state).</p> <p><i>Primary Diagnosis Code:</i> The first diagnosis field(s) of the coded clinical record (i.e., primary or principal diagnosis). Presently, the code is represented by an ICD-9-CM code (the International Classification of Diseases, 9th Revision, Clinical Modification). For asthma that code is 493.XX.</p> <p><i>Resident:</i> Any person with a residential address in the county/state of the grantee at the time of the hospital admission.</p>
<p>How-to-Guide Requirements and Cautions</p>	<ul style="list-style-type: none"> • This How-to-Guide has two purposes: 1) Guide the user in the development of the XML dataset using the SAS code, located on SharePoint, for submission to CDC; 2) Describe the calculation steps for creating the Nationally Consistent Data Measures (NCDMs). Grantees should use the How-to-Guide in order to ensure that the calculation of measures are consistent with those required by CDC in the preparation of their own computer code. • Note that one set of SAS code (not provided by CDC’s EPHT program) is designed to handle the creation of XML files for all hospitalization (inpatient) outcomes (separate code is available for all ED Visit outcomes). The code manages the differences in definitions between the outcomes and generates a separate file for each outcome by each single year. • This how-to-guide and accompanying SAS code (not provided by CDC’s EPHT program) presume that the user already removed any duplicate records (see definitions for more information), while keeping multiple admissions. • Hospitalizations due to transfers between acute care hospitals are not excluded in the counts/measures to be generated. Therefore, for consistency, it is advised that transfers not be excluded. An algorithm to exclude transfers is underdevelopment. NOTE: The Date Dictionary includes two variables regarding the exclusion of transfers. These are placeholders only (to be activated in future work) and the SAS code will automatically set the codes for these variables the same for all grantees.

These variables do not need to be a part of your SAS dataset. If they are present, the program will ignore them.

- The data source is an inpatient discharge dataset but the EPHT dataset is based upon date of admission because of the goal of relating a hospitalization event with an environmental event. Therefore, the hospitalization counts and measures require the development of an admission dataset. For admissions at the end of a calendar year where the discharge date is in the following year, that latter year's discharge dataset will be required before the admission dataset for the preceding year can be considered complete.
- Data suppression/aggregation rules are not incorporated into the SAS code. Suppression guidelines are currently applied by CDC for the national portal.
- The steps for estimating age-adjusted rates are not the same for asthma and acute myocardial infarction. This is because the age groups are different, requiring steps for population weighting in the case of acute myocardial infarction.
- Admissions of residents to out-of-state hospitals are not required to be included. However, when available these admissions should be included. It is noted that some states must include out-of-state admissions of its residents. Use the “**OUTOFSTATEEXCLUSION**” variable in the dataset to capture whether out of-state admissions are included or not (the Data Dictionary and schema provide for formal notation in the dataset on whether these admissions are included). Be certain to use footnotes and metadata to acknowledge the disposition of these admissions.
- Admissions to federal facilities, such as Veteran's Hospitals, are not included. Be certain to inform CDC if your state requires that your dataset includes admissions to federal facilities so that the measures can be appropriately footnoted.
- The optional measures of Monthly Average Number of Hospitalizations and Daily Number of Hospitalizations are only intended for inclusion on state portals and as optional measures. These are not to be submitted to CDC.
- The Data Dictionary should be referred to for the standardized definitions and notations of the variables to be submitted to CDC.

NOTE FOR NON-SAS USERS: Grantees not using SAS should use the steps outlined below for guidance on important issues such as population requirements and should refer to the Data Dictionary for specifications of the required fields.

The data file should be converted to the .XML file format and the required header inserted into the XML file, according to the Schema found on SharePoint. In addition, refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data Dictionary).

NOTE FOR SAS USERS: SAS code is not developed or distributed by CDC.

STEPS FOR CREATING SAS HOSPITALIZATION DATASET

Step #1

Source of Data: Individual level state inpatient hospital admission data based on primary diagnosis.

Please consult your data steward and data managers to understand the variables and coding system, specifically for race and ethnicity variables. In some states these variables may be coded as one variable whereas in others they are coded as separate variables.

Years of Interest: 2000–present calendar year; The SAS program (not provided by CDC) prepares one NCDM file for each year, as required for CDC submission.

Most hospitalization data is released in annual discharge-based datasets. In order to have complete admission date information using discharge-based datasets, it is necessary to have the dataset of the year of interest and the subsequent year. For example, to obtain all admissions during 2005 using discharge based datasets, it will be necessary to have both the 2005 and the 2006 discharge datasets for admissions that occurred in 2005 but were not reported until release of the 2006 discharge dataset. For this example, 2005 data should not be submitted prior to receipt of the 2006 discharge dataset from the data steward.

Removal of Duplicates: This how-to-guide and accompanied SAS code presumes that the user has already removed any duplicate records, while keeping multiple admissions.

Data Specifications: Refer to the Data Dictionary in order to conform to coding specifications required for the NCDM variables.

NOTE: county FIPS code is 5 digits and that the first two digits will be the same as the state FIPS code. Also, some variables are optional.

Select all hospital records that meet the following criteria:

- Exclude all records where the State of residence is not your state.
- Since hospitalizations data are based on discharge year and the EPHT dataset is to be based on admission date, patients discharged in current year (i.e., 2006) but admitted in the previous year (i.e., 2005) should be counted as 2005 hospitalizations. The admission date (and discharge and birth date) variable is acceptable in the following formats:
 - SAS DATE FORMAT
 - MMDDYYYY
 - MMDDYY
 - MM/DD/YYYY
 - MM-DD-YYYY
 - DDMONYYYY
 - DDMONYY
 - YYYYMMDD
 - DDMONYYYY:00:00:00

SAS Dataset: Make a copy of the hospital admission data before proceeding to next step. If the admission data are not in SAS format, convert it to SAS format and save it as a permanent dataset for creating the XML and measures. Keep the following variables in

	<p>the SAS dataset :</p> <ul style="list-style-type: none"> ○ Primary diagnosis code ○ Date of admission ○ Date of discharge ○ Patient date of birth <u>OR</u> Age at admission ○ Patient’s sex ○ Patient’s race (optional) - White, Black, Other, Unknown ○ Patient's ethnicity (optional) - Hispanic, Non-Hispanic, Unknown ○ County of residence ○ State of residence <p><i>Population Data:</i> US Census bureau residential population data for state and county. Intercensal population estimates should be used for the intercensal years, and population extrapolations for postcensal years.</p> <p><i>Go to Step # 2.</i></p>
<p>Step #2</p>	<p>The base format for counts and population data should be by 5-year age groups for asthma beginning 0–4 and ending with 85+.</p> <p>Hospitalization counts must be submitted to CDC by these 5-year age groups (see Data Dictionary). For the calculation of measures and presentation, the age-groups of interest for various asthma measures are different because of the nature of the diseases. Refer to the measure-specific step for the appropriate age groups for calculation and presentation. In summary, the hospitalization and population age-groups required for the calculation and presentation of measures are:</p> <p>Asthma counts to CDC: 5-yr age-groups (0–4, 5–9 ... 85+)</p> <p>Asthma age-specific rate presentation: 0–4, 5–14, 15–24, 25–34, 35–44, 45–54, 55–64 and 65+</p> <p>Asthma crude and age-adjusted rates: 5-yr age groups</p> <p>Race and ethnicity variables are optional. Therefore, counts and measures may be generated without specifying race or ethnicity if these data are missing or considered unreliable/inaccurate. If race and ethnicity data are reported, the NCDMs will only be generated when the source variables for race and ethnicity are separate variables. The NCDMs will not be generated if race and ethnicity are reported as a combined race/ethnicity variable. If race and ethnicity data are being provided, be sure that the coding structure conforms to that laid out in the Data Dictionary.</p> <p>Race: White; Black; Other; Unknown.</p> <p>Ethnicity: Hispanic; non-Hispanic; Unknown.</p> <p><i>Go to Step # 3.</i></p>
<p>STEPS FOR GENERATING NCDM REQUIREMENTS</p> <p><i>(Grantees not using SAS should refer to the steps below for conversion of their data file to XML format)</i></p>	

Step #3	<p>After creating the SAS datasets, download IP-NCDM containing the recommended SAS code and Users Guide. These can be found on SharePoint. The current version will only create xml files (not measures).</p> <p>Unzip the files and read and follow the User’s Guide. Click IP-NCDM.msi to launch the setup wizard. Double click the shortcut icon to launch the program.</p> <p>XML files for each single year of data will be generated for one outcome, as required for CDC submission.</p> <p>SAS code to generate measures is not yet operational.</p> <p><i>NOTE:</i> Single year files for individual outcomes must be submitted, as specified in the data call letter</p> <p>Steps #4 through #12 should be used to calculate the NCDMs to ensure consistency between grantees.</p>
STEPS FOR COMPLETING SPECIFIC MEASURES	
Step #4	<p>SAS menu options 3–9 generate measures beyond those required for the national portal but are currently non-operational. These options will follow the steps below (4b through 12). These steps may also be used to generate measures outside of SAS. If selecting menu options 3–9, this SAS program will generate a final output in the form of MS Excel spreadsheet. A PDF file containing histograms will also be created.</p> <p>Flag all admissions where primary diagnosis code is “493” by creating a variable (<i>for example Ishospital</i>) that takes the value of 1, if admission is due to diagnosis code “493”; else its value is 2.</p> <p>Keep only those records that meet the following criteria:</p> <ul style="list-style-type: none"> - Principal diagnosis code = 493 - State = Your state - Date of Birth (if available and being used to calculate patient age) is not missing - Patient’s age at the time of hospital admission is not missing - Date of Discharge is not missing - Date of Admission is not missing. <p>Go to Step # 5.</p>
ANNUAL NUMBER OF HOSPITAL ADMISSIONS	
Step #5	Annual Number of Hospital Admissions by sex and total
Step #5a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by sex (male, female and unknown (including missing)).</p> <p>Sum the number of male, female and unknown sex hospital admissions to get the total number of hospital admissions for the year.</p>
Step #5b	<i>County:</i> Sum the total number of admissions by county of residence for year of interest

	<p>by sex [male, female and unknown (including missing)].</p> <p>Sum the number of male, female and unknown sex hospital admissions to get the total number of hospital admission by county for the year.</p>
Step #6	Annual Number of Hospital Admissions by race and total
Step #6a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by race categories.</p> <p>Sum the number of hospital admissions to get the total number of asthma hospital admission for the year.</p>
Step #6b	<p><i>County:</i> Sum the total number of admissions for by county of residence for year of interest by race categories.</p> <p>Sum the number of hospital admissions to get the total number of hospital admission by county and race for the year.</p>
Step #7	Annual Number of Hospital Admissions by age groups and total
Step #7a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by the specific 5-year age groups created in step #2.</p> <p>Sum the number of hospital admissions for each age group to get the total number of hospital admission for the year.</p>
Step #7b	<p><i>County:</i> Sum the total number of admissions for year of interest for each county by the specific 5-year age groups created in step #2.</p> <p>Sum the number of hospital admissions for each age group to get the total number of hospital admission for the year.</p>
DAILY NUMBER OF HOSPITAL ADMISSIONS (NOT A REQUIRED NCDM)	
Step #8	<p>NOTE: Daily number of admissions is not a required NCDM and is not submitted to CDC or required to be placed on state portals. Because of the potential future use of this measure. It remains in the How-to-Guide and SAS code.</p> <p>Sum the total number of admission for each day by sex during the year of interest for entire state to get the daily number of admissions by sex. Add the daily number of male, female and unknown (including missing sex information) to obtain the total number of daily admissions. Repeat the above by race and 5-year age-groups to calculate the daily number of hospital admissions by race and age groups.</p>
ANNUAL AGE-SPECIFIC HOSPITAL ADMISSIONS RATE	
Step #9	Annual age-specific hospital admission rate by sex
Step #9a	<p><i>Create the numerator data:</i> Sum the number of hospitalizations in the state for the year of interest in each of the 5-year age-groups for both male and female. Exclude any observation where sex is unknown or missing. Sum the admissions for male and female to obtain the total admissions for each age-group.</p>
Step #9b	<p><i>Create the denominator data:</i> Sum the population in the state or county for year of interest in each 5-year age group for both male and female. Sum male and female</p>

	population to obtain the total state population for each age-group.
Step #9c	Merge both numerator and denominator data by state of residence, year of hospital admissions, age-group and sex.
Step #9d	<i>For asthma presentation:</i> Compute age-specific rates for male, female and total for age-groups 0-4, 5-14, 15-24, 25-34, 35-44, 45-54, 55-64 and 65+ by dividing the number of hospital admissions in that age group and sex by the population of same age-age group and sex. For example, to calculate the age-specific rates of admissions in 5-14 year old male divide the annual number of hospital admissions in 5-14 years old male by the population of 5-14 years old male.
Step #9e	All rates are to be presented as per 10,000 population. Multiply the rates calculated in step 9d by 10,000 to obtain rate of admission per 10,000 population
Step #9f	Upper and lower confidence limits (95% confidence interval) for age-specific rates may be computed. For each age-specific rate computed in step 9d, compute Lower Confidence Limit (LCL) and Upper Confidence Limit (UCL) as follows LCL = [age-specific rate – {1.96 * age-specific rate/SQRT (Number of Admissions)}] UCL = [age-specific rate + {1.96 * age-specific rate/SQRT (Number of Admissions)}]
ANNUAL UNADJUSTED (CRUDE) RATE OF HOSPITAL ADMISSIONS	
Step #10a	Exclude any observation where sex is unknown or missing. <i>Asthma:</i> Create the numerator data (referred to in Step #2) to obtain the annual number of hospital admissions by sex for both male and female across all ages. Sum the male and female number of admissions to obtain total admissions for both sexes.
Step #10b	<i>Asthma:</i> Create the denominator data as referred to in step #2 across all ages. Sum the population for male, female and both sex for the year of interest.
Step #10c	Merge both numerator and denominator data by state of residence, year of hospital admissions and sex.
Step #10d	Compute the annual unadjusted rate of hospital admissions as follows: Unadjusted Admission Rate (Male) = (# of Male Admissions/Male Population) Unadjusted Admission Rate (Female) = (# of Female Admissions/Female Population) Unadjusted Admission Rate (Total) = (Total Admissions/ State Population) Multiply the above computed rated by 10,000 to obtain the number of admissions per 10,000 population.
ANNUAL AGE ADJUSTED RATE OF HOSPITAL ADMISSIONS	
Step #11	Annual Age Adjusted Rate of Hospital Admissions for State by Sex
Step #11a	<i>Asthma:</i> Calculate the age specific rates as described in steps 10a through 10d using 5 yr age groups (0–4, 5–9 ... 85+) for male, female and both sexes.

Step #11b	To calculate age-specific rates (for the 5-year age categories 0–4, 5–9...85+); use U.S. Census bureau residential population data for denominators. Intercensal population estimates should be used for the intercensal years. . The standard population should be the 2000 U.S. Standard Population divided into 18 age groups. The link for the 2000 U.S. Standard Population is: http://seer.cancer.gov/stdpopulations/). After downloading, combine the '0' age group with the '1–4' age group.
Step #11c	Merge both numerator and denominator data by age group and sex.
Step #11d	<p>Compute the age-adjusted population weights using the 2000 US population as the standard.</p> <p><i>Asthma:</i> Compute the age-adjustment weights of hospital admissions using 2000 US Standard Population by age group for males, females, and both sexes as follows:</p> <p>Age-adjusted weights = age-specific std pop/total std pop, where the total weight for all ages is 1.0.</p>
Step #11e	<p>Compute the age-adjusted hospital admissions rate:</p> $\text{Age-adjusted rate} = \text{Sum of age-specific rate} \times \text{age adjusted weight}$ <p>For tutorial on age-adjustment see http://seer.cancer.gov/seerstat/tutorials/aarates/definition.html .</p>
Step #11f	<p>95% confidence intervals may be computed.</p> $\text{LCL} = [\text{age-adjusted rate} - \{1.96 \times \text{age-adjusted rate}/\text{SQRT}(\text{Number of Admissions})\}]$ $\text{UCL} = [\text{age-adjusted rate} + \{1.96 \times \text{age-adjusted rate}/\text{SQRT}(\text{Number of Admissions})\}]$ <p>NOTE: With small numbers of hospitalizations (e.g., <20), calculation methods assuming a non-Normal distribution may be more appropriate.</p>
Step #11f	To calculate the Annual age-adjusted rate of hospital admissions by County, follow steps 11a through 11f using the same 2000 US standard population.
PRESENTATION	
Step #12a	Export SAS datasets to Microsoft Excel® for creating charts or for formatting the computed values for presentation
Step #12b	<p>Annual number of hospital admissions can be displayed by showing sex on x-axis and number of admissions on Y-axis. Similarly sex on X-axis can be replaced by race or age groups to display the number of admissions by race or age-group.</p> <p>These bar charts can be created by using any spreadsheet application or by using SAS.</p>
Additional visual display	<p>Public can view histograms and map showing the state and county level asthma measures. These visual displays will show whether the selected geographical area has a high, medium high, medium low or low hospital admission rate. The public will also be able to see the links to other related information from various national, state and local sources.</p> <p>Mapping the rate of hospital admissions per 10,000 residents will allow users to assess the level of environmentally related risk factors in their residential geographic area as</p>

	<p>well as in the surrounding geographical area. Future mapping may include other risk factors such as poverty and other potential risk factors. One limitation of mapping is that not all sources of asthma or myocardial infarction risk factors may be mapped. For example, indoor mold, dust, and pollen.</p>
<p>Interpretation</p>	<p>Small Numbers: Measures that generate counts or rates based upon numbers that are small and potentially violate state privacy guidelines can collapse data across years or geographic units to generate data that can be released.</p> <p>Measures for multiple years: The how-to-guide steps can be repeated for additional years of hospital admission data. Multi-year hospital admission data can be merged to create one dataset. Add the number of hospital admissions for each year in multi-year cohort and divide by the number of years to calculate an average annual number of hospital admissions.</p>

Indicator Template
Content Area: Asthma
Indicator: Hospitalizations for Asthma
Environmental Public Health Tracking

Type of EPHT Indicator	Health outcome
Measures	1. Age-adjusted rate of hospitalization for asthma per 10,000 population 2. Crude rate of hospitalization for asthma per 10,000 population 3. Number of hospitalizations for asthma
Derivation of Measure(s)	<i>Numerator:</i> Resident hospitalizations for asthma, ICD-9-CM: 493.XX by gender and total for state and by county <i>Denominator:</i> Midyear resident population, by gender, for state and by county <i>Adjustment:</i> Age-adjustment by the direct method to year 2000 US standard population
Unit	1. Age-adjusted rate per 10,000 population 2. Rate per 10,000 population 3. Number
Geographic Scope	State and national
Geographic Scale	Residents of jurisdiction – State, County
Time Period	Hospital admissions between January 1 to December 31, inclusive, for each year, 2000–
Time Scale	Daily, monthly, and annually (as appropriate for the measure)
Rationale	<p>In 2004, 20.5 million people in the U.S. reported having asthma. In 2003, there were over 574,000 hospitalizations for asthma. In 2002, there were over 4,200 deaths in which asthma was the underlying cause. Asthma is the leading chronic health condition among children. There are also large racial, income, and geographic disparities in poor asthma outcomes. Asthma causes lower quality of life, preventable undesirable health outcomes, and large direct and indirect economic costs. Environment Attributable Fractions of the 1988-1994 economic costs for asthma were 39.2% for children <6 years and 44.4% for 6–16 year olds, costing more than \$400 million for each age group.</p> <p>A number of epidemiologic studies have reported associations between air pollution exposures and asthma. The association between ambient air particulate matter (PM) concentrations and asthma, including increased hospital admissions, is well documented. Models demonstrate 5–20% increases in respiratory-related hospital admissions per 50µg/m³ of PM₁₀ and 5–15% per 25µg/m³ of PM_{2.5}, with the largest effect on asthma admissions.</p> <p>In the Eastern United States, summer ozone pollution was associated with more than 50,000 hospital admissions per year for asthma and other respiratory emergencies. Large multi-city and individual city studies found a positive association between ozone and total respiratory hospital admissions, including asthma, especially during the warm season. Among US and Canadian studies, the ozone-associated increase in respiratory hospital admissions ranged from 2-30%</p>

	<p>per 20 ppb (24 hour), 30 ppb (8 hour) or 40 ppb (1 hour) increment of ozone in warm seasons.</p> <p>In 2000, the Institute of Medicine cited sufficient evidence to conclude that allergens produced by cats, cockroaches and house dust mites caused asthma exacerbations as did exposure to environmental tobacco smoke (ETS) in preschool aged children. A 2005 California Air Resources Board report noted that there is sufficient evidence to conclude that ETS causes asthma exacerbation in children and adults (CARB, 2005). That report also estimated 202,300 excess childhood asthma episodes occur each year in the U.S. as a result of exposure to ETS.</p>
<p>Use of the Measure</p>	<p>The development of a standardized analytic method for asthma hospital admissions among residents in each state will inform multiple users at the national, state, and local levels. These measures will allow the monitoring of trends over time, identify high risk groups, and inform prevention, evaluation and program planning efforts.</p> <p>These measures will address the following surveillance functions:</p> <ul style="list-style-type: none"> • How many hospitalizations for asthma occur in every month? • Is there a seasonal or temporal trend of asthma hospitalizations? • What's the distribution of asthma hospitalizations by place of residence? • How do hospitalizations for asthma differ between geographic areas (e.g. zip code, county, state, or region)? • With further analysis ... Are there disparities in asthma hospitalizations by factors such as age, race, ethnicity, gender, education, and/or income? • Which populations are in need of targeted interventions? • When asthma data are linked with environmental variables, do the linked measures identify environmental relationships warranting further investigation or environmental public health action?
<p>Limitations of the Measure</p>	<ul style="list-style-type: none"> • Hospitalization data, by definition, does not include asthma among individuals who do not receive medical care or who are not hospitalized, including those who die in emergency rooms, in nursing homes, or at home without being admitted to a hospital, and those treated in outpatient settings. • Differences in rates by time or area may reflect differences or changes in diagnostic techniques and criteria and in the coding of asthma. • Reporting rates at the state and/or county level will not show the true asthma burden at a more local level (i.e. neighborhood). • Differences in rates by area may be due to different socio-demographic characteristics and associated behaviors. • When comparing rates across geographic areas, a variety of non-environmental factors, such as access to medical care and diet, can impact the likelihood of persons being hospitalized for asthma. • • Reporting rates at the state and/or county level will not be geographically resolved enough to be linked with many types of environmental data. • When looking at small geographic levels (e.g. ZIP code), users must take into consideration appropriate cell suppression rules imposed by the data

	<p>providers or individual state programs.</p> <ul style="list-style-type: none"> • Although duplicate records and transfers from one hospital to another are excluded, the measures are based upon events, not individuals, because no unique identifier is ever? available. When multiple admissions are not identified, the true prevalence will be overestimated. • Even at the county level it can be expected that the measures generated will often be based upon numbers too small to report or present without violating state and federal privacy guidelines and regulations. Careful adherence to cell suppression rules in cross tabulations is necessary and methods to increase cell sizes by combining data across time (e.g., months, years) and geographic areas may be appropriate.
Data Sources	<p><i>Numerator:</i> State inpatient hospitalization data (using admission date) <i>Denominator:</i> US Census Bureau population data</p>
Limitations of Data Sources	<p><u>State hospital discharge data:</u></p> <ul style="list-style-type: none"> • Using a measure of all asthma hospitalizations will include some transfers between hospitals for the same individual for the same asthma event. Variations in the percentage of transfers or readmissions for the same asthma event may vary by geographic area and impact rates. • Without reciprocal reporting agreements with abutting states, statewide measures and measures for geographic areas (e.g., counties) bordering other states may be underestimated because of health care utilization patterns. • Each state must individually obtain permission to access and, in some states, provide payment to obtain the data. • Veterans Affairs, Indian Health Services and institutionalized (prison) populations are excluded. • Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers to hospitalize patients • Street address is currently not available in many states. • Sometimes mailing address is listed as the residence address of the patient • Patients may be exposed to environmental triggers in multiple locations, but hospital discharge geographic information is limited to residence. • Since the data captures hospital discharges (rather than admissions), patients admitted toward the end of the year and discharged the following year will be omitted from the current year dataset • Data will need to be de-duplicated (i.e. remove duplicate records for the same event) • There is usually a two year lag period before data are available from the data owner. <p><u>Census data:</u></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census Bureau. These need to be extrapolated or purchased from a vendor.
Related Indicators	<ul style="list-style-type: none"> • Asthma prevalence among adults • Asthma prevalence among children • Emergency department visits due to asthma

References

1. Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System (BRFSS) Prevalence Data. 1999–2010 November 16, 2011 [cited 2012 July 2]; Available from: <http://www.cdc.gov/asthma/brfss/default.htm#00>.
2. Mannino, D.M., et al., Surveillance for asthma—United States, 1960–1995. *MMWR CDC Surveill Summ*, 1998. 47(SS-1): p. 1–28.
3. Mannino, D.M., et al., Surveillance for asthma—United States, 1980–1999. *MMWR Surveill Summ*, 2002. 51(1): p. 1–13.
4. Britton, J. and S. Lewis, Epidemiology of Childhood Asthma, in *Asthma: Epidemiology, Anti-Inflammatory Therapy and Future Trends*, M. Giembycz and B. O'Connor, Editors. 2000, Birkhäuser Basel: Switzerland. p. 25–56.
5. Gold, D.R. and R. Wright, Population disparities in asthma. *Annu Rev Public Health*, 2005. 26: p. 89–113.
6. Lanphear, B.P., et al., Residential exposures associated with asthma in US children. *Pediatrics*, 2001. 107(3): p. 505–11.
7. Lanphear, B.P., et al., Contribution of residential exposures to asthma in us children and adolescents. *Pediatrics*, 2001. 107(6): p. E98.
8. Redd, S.C., Asthma in the United States: burden and current theories. *Environ Health Perspect*, 2002. 110 Suppl 4: p. 557–60.
9. Arif, A.A., J.E. Rohrer, and G.L. Delclos, A population-based study of asthma, quality of life, and occupation among elderly Hispanic and non-Hispanic whites: a cross-sectional investigation. *BMC Public Health*, 2005. 5: p. 97.
10. Jorres, R.M.H., Atmospheric pollutants, in *Asthma: Basic Mechanisms and Clinical Management*, P. Barnes, I. Rodger, and N. Thomson, Editors. 1998, Academic Press: London. p. 589–596.
11. Trasande, L. and G.D. Thurston, The role of air pollution in asthma and other pediatric morbidities. *J Allergy Clin Immunol*, 2005. 115(4): p. 689–99.
12. Jaffe, D.H., M.E. Singer, and A.A. Rimm, Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991–1996. *Environ Res*, 2003. 91(1): p. 21–8.
13. U.S. Environmental Protection Agency, Air Quality Criteria for Particulate Matter (Final Report, Oct 2004), 2004, U.S. Environmental Protection Agency. EPA 600/P-99/002aF-bF: Washington, DC.
14. Institute of Medicine, Committee on the Assessment of Asthma and Indoor Air. Division of Health Promotion. Disease Prevention. *Clearing the Air: Asthma and Indoor Air Exposures 2000*, Washington, DC: The National Academies Press.

Carbon monoxide poisoning Hospitalizations and Emergency Department Visits Data Submission

Fall 2014 Data Call

We received a few comments from grantees regarding Carbon monoxide (CO) poisoning data submission and How-to guides. There were some typos and some unclear language in the How-to guide and, therefore, we have made a couple of edits, which are listed below along with some clarification. Please note that no changes were made to how the data should be processed or submitted, including no changes to the ICD-9 codes used for selection and categorization of CO cases.

1. When selecting hospitalizations or emergency department (ED) visits for CO poisoning, you need to select **ANY** of the ICD-9-CM diagnosis codes as principal diagnosis, injury cause, or other diagnoses. We have updated page 1 of the CO Hospitalizations How-to guide to correct this error. This information was also provided in the Data Call Webinar presentation, slide 10, which is available below and on SharePoint.
2. **ONLY** submit new years of hospitalizations and ED visits data for CO poisoning. For example: during the Fall 2013 data call, if you submitted CO poisoning data till 2011; then you need to submit **ONLY** new years of data i.e., 2012 and 2013 (if available). We have updated page 3 of the How-to guide so that this clear.

If you have additional questions about CO poisoning hospitalizations and ED visits data submission, please contact us at: TrackingSupport@cdc.gov

Hospitalizations and Emergency Department Data

- When selecting cases from Hospitalizations and ED data, use:

Health Outcome	Diagnosis code selection
Asthma	Primary diagnosis code
CO Poisoning	ANY primary or other diagnosis code
Heart Attack	Primary diagnosis code
Heat Stress	ANY primary or other diagnosis code

HOW-TO GUIDE

Carbon Monoxide Poisoning Emergency Department (ED) Visits

Environmental Public Health Tracking

07-18-2013

Data Source	Emergency Department (ED) Visits
NCDM Requirements	<ul style="list-style-type: none"> • Health Outcome (Carbon Monoxide (CO) Poisoning) • State/County of Residence • ED Visit Year/Month • Age Group • Sex • Race/Ethnicity (optional) • Transfers not to be excluded • Out-state residents to be excluded • ED visits to federal facilities to be excluded • ED visits of residents to out-of-state hospitals are to be optionally included
Measures Generated	<ul style="list-style-type: none"> • Age-adjusted rate of ED visits for unintentional carbon monoxide poisoning per 100,000 population, stratified by cause: fire related, non-fire related, or unknown • Crude rate of ED visits for unintentional carbon monoxide poisoning per 100,000 population, stratified by cause: fire related, non-fire related, or unknown • Number of ED visits for carbon monoxide poisoning, stratified by cause: fire related, non-fire related, or unknown
Definitions	<p><i>Carbon monoxide (CO) Poisoning:</i> This indicator tracks acute, unintentional carbon monoxide poisoning resulting in ED treatment. Carbon monoxide is an odorless, colorless gas that is the byproduct of combustion, which preferentially binds to hemoglobin and therefore displaces oxygen in the blood stream. Carbon monoxide is the leading cause of acute, unintentional poisoning and death (excluding alcohol and drug-related intoxication).</p> <p><i>Duplicate record:</i> More than one record for the same person with the same ED Visit data (e.g., sex, date of birth, admission/ED Visit date, and zip code have exact same information). Duplicate records may also be due to continuation of data beyond a single line. In this case, duplicates may be identified using a record sequence number.</p> <p><i>ED Visit date:</i> The calendar date of the ED Visit:</p> <ul style="list-style-type: none"> ▪ Day (optional) ▪ Month (required) ▪ Year (required) <p><i>ED Visit Year:</i> An ED Visit for CO Poisoning during a specific calendar year. ED Visit year is based only upon the calendar year of the Visit, even when discharge and/or</p>

	<p>release year is different.</p> <p><i>Emergency Department Visit:</i> Condition of being treated in an acute care hospital in an emergency department (ED) for treatment as an outpatient, or placed in an acute care hospital (admitted) as an inpatient subsequent to treatment in the ED.</p> <p><i>Hospital Transfers:</i> The practice of discharging a patient from one facility and readmitting them to a second facility within 48 hours.</p> <p><i>ICD-9-CM code:</i> International Classification of Diseases, 9th Revision, Clinical Modification</p> <p><i>Multiple visits:</i> Second or subsequent ED Visit for the same person for the same diagnosis code occurring on different dates and related to a separate event within a given year. Multiple ED Visits are considered separate events if they occurred more than 48 hours apart.</p> <p><i>Observation Stay:</i> This is an alternative to inpatient admission that exists in some facilities but for EPHT is considered in ED Visit statistics. Observation Stays may originate as an ED Visit or directly as an Observation Stay. Note that the definition of an Observation Stay may not be standard across hospitals, and Observation Stays may not be recorded across states in a consistent manner.</p> <p><i>Primary Diagnosis Code:</i> Presently, diagnosis codes are represented by ICD-9-CM codes (the International Classification of Diseases, 9th Revision, Clinical Modification). Carbon monoxide is classified as any primary or other diagnosis code of 986, or cause of injury code E868.2, E868.3, E868.8, E868.9, E982.0, or E982.1. Cases with any intentional cause of carbon monoxide poisoning (E952.0, E952.1) or other intentional injury (E950.0-E979.9, E990.0-E999) anywhere in the record are <u>excluded</u>.</p> <p><i>Resident:</i> A person who resides in the grantee's state/county (permanently or for an extended period) at the time of the ED Visit.</p>
<p>How-to-Guide Requirements and Cautions</p>	<ul style="list-style-type: none"> • This How-to-Guide has two purposes: 1) Guide the user in the development of the XML dataset using the SAS code, located on SharePoint, for submission to CDC; 2) Describe the calculation steps for creating the Nationally Consistent Data Measures (NCDMs). Grantees should use the How-to-Guide in order to ensure that the calculation of measures are consistent with those required by CDC in the preparation of their own computer code. • Note that one set of SAS code (not provided by CDC's EPHT program) is designed to handle the creation of XML files for all hospitalization (inpatient) outcomes (separate code is available for all ED Visit outcomes). The code manages the differences in definitions between the outcomes and generates a separate file for each outcome by each single year. • This How-to-Guide and the optional SAS code not provided by CDC's EPHT program) presume that the user has removed duplicate records while keeping multiple ED Visits. A case should be counted once per ED Visit; de-duplication of records to achieve this goal should be conducted at the discretion of the data owners, managers, and/or analysts.

- ED Visits include both patients who are admitted to the hospital through the emergency department (inpatients) and those who are treated and released (outpatients); therefore, both inpatient and outpatient data are required for this indicator. If identified and/or stored separately, observation stay data should be included as well. Note: This guidance provides instruction on how to select ED Visit cases from ED datasets, inpatient datasets, and observation stay datasets.
- In the event that an ED Visit occurred at the end of a calendar year and the discharge date occurred in the following year, the dataset that includes the discharge date will be required before the dataset can be considered complete.
- The How-to-Guide steps do not incorporate data suppression and/or aggregation rules. Suppression guidelines are separately applied by CDC for the national portal and by grantees for state portals.
- Admissions of residents to out-of-state hospitals are not required to be included. However, when available these admissions should be included. It is noted that some states must include out-of-state admissions of its residents. Use the “OUTOFSTATEEXCLUSION” variable in the dataset to capture whether out-of-state admissions are included or not (the Data Dictionary and schema provide for formal notation in the dataset on whether these admissions are included). Be certain to use footnotes and metadata to acknowledge the disposition of these admissions.
- ED Visits for individuals who are not state residents should be excluded. If a data steward’s database includes these cases, exclude them from the EPHT database. If they cannot be excluded, footnotes and metadata should acknowledge that these cases are included.
- Patients transferred from or to other acute care facilities should be included. Indicate in footnotes and/or metadata if transfers are excluded.
- Patients with an ED Visit at a federal facility should not be included. If a data steward’s database includes these cases, exclude them from the EPHT database. If they cannot be excluded, footnotes and metadata should acknowledge that these cases are included.
- Although hospital discharge data are collected using a standard format across states, there are considerable differences in the variable attributes; for example, response categories may differ between states for “source of admission” and “disposition” variables. These differences may reflect how certain variables are collected, whether the reporting of a variable (for example patient name or race) is mandatory, and/or differences in data availability and access agreements. The number of diagnosis fields available in the discharge data also varies by state, ranging from nine to an unlimited number. In addition, the data vary by state in regard to data quality such as the validity or completeness of specific fields. In all cases, the data analyst should work closely with the data managers in order to understand the nuances of the data.
- The Data Dictionary in SharePoint should be referred to for the standardized

	definitions and notations of the variables to be submitted to CDC.																
<p>NOTE FOR NON-SAS USERS: Grantees not using SAS should use the steps outlined below for guidance on important issues such as population requirements and should refer to the Data Dictionary for specifications of the required fields. The data file should be converted to the .XML file format and the required header inserted into the XML file, according to the Schema found on SharePoint. In addition, refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data Dictionary).</p> <p>NOTE FOR SAS USERS: SAS code is not developed or distributed by CDC.</p>																	
Section A: CREATION OF REQUIRED DATA FILE FOR NCDMS																	
Step #1	<p><i>Identifying the data sources for ED Visits:</i></p> <p>ED Visits include both patients who are treated and released in the ED (outpatients) and who are admitted as inpatients through the emergency department; therefore, <u>both</u> inpatient and outpatient data files are required for this indicator. If identified separately, observation stay data files are also required.</p>																
Step #2	<p><i>Identifying ED Visits for CO Poisoning</i></p> <p>a. From ED data, Select cases having any of the following ICD-9 codes as principal diagnosis, injury cause, or other diagnoses:</p> <table border="0" style="margin-left: 40px;"> <thead> <tr> <th style="text-align: left;">Code</th> <th style="text-align: left;">Description</th> </tr> </thead> <tbody> <tr> <td>986</td> <td>Toxic effect of carbon monoxide</td> </tr> <tr> <td>E868.2</td> <td>Accidental poisoning by motor vehicle exhaust gas</td> </tr> <tr> <td>E868.3</td> <td>Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels</td> </tr> <tr> <td>E868.8</td> <td>Accidental poisoning by carbon monoxide from other sources</td> </tr> <tr> <td>E868.9</td> <td>Accidental poisoning by carbon monoxide, unspecified source</td> </tr> <tr> <td>E982.0</td> <td>Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted</td> </tr> <tr> <td>E982.1</td> <td>Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted</td> </tr> </tbody> </table> <ul style="list-style-type: none"> • Exclude all records where the State of residence is not your state. • <i>Remove and exclude</i> records having any intentional cause of carbon monoxide poisoning (E952.0, E952.1) or other intentional injury (E950.0–E979.9, E990.0–E999). <p>Of the selected cases, identify and categorize any accidental injury/poisoning due to fire and flames (E890–E899). These cases will comprise the “unintentional, fire related” subset of this measure.</p> <ul style="list-style-type: none"> • Of the remaining selected cases, identify and categorize cases of carbon monoxide poisoning due to all other causes (E818, E825, E838, E844, E867, or E868). These cases will comprise the “unintentional, non-fire related” subset of this measure. Note: If a record has both a fire related and other cause of CO poisoning, classify as “unknown.” 	Code	Description	986	Toxic effect of carbon monoxide	E868.2	Accidental poisoning by motor vehicle exhaust gas	E868.3	Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels	E868.8	Accidental poisoning by carbon monoxide from other sources	E868.9	Accidental poisoning by carbon monoxide, unspecified source	E982.0	Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted	E982.1	Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted
Code	Description																
986	Toxic effect of carbon monoxide																
E868.2	Accidental poisoning by motor vehicle exhaust gas																
E868.3	Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels																
E868.8	Accidental poisoning by carbon monoxide from other sources																
E868.9	Accidental poisoning by carbon monoxide, unspecified source																
E982.0	Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted																
E982.1	Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted																

Categorize all remaining cases, including those without E-coding, as “unknown.”

b. From ED data, select all records that meet the following criteria:

Code	Description
986	Toxic effect of carbon monoxide
E868.2	Accidental poisoning by motor vehicle exhaust gas
E868.3	Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels
E868.8	Accidental poisoning by carbon monoxide from other sources
E868.9	Accidental poisoning by carbon monoxide, unspecified source
E982.0	Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted
E982.1	Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted

- *Remove and exclude* records having any intentional cause of carbon monoxide poisoning (E952.0, E952.1) or other intentional injury (E950.0–E979.9, E990.0–E999).

Of the selected cases, identify and categorize any accidental injury/poisoning due to fire and flames (E890–E899). These cases will comprise the “unintentional, fire related” subset of this measure.

- Of the remaining selected cases, identify and categorize cases of carbon monoxide poisoning due to all other causes (E818, E825, E838, E844, E867, or E868). These cases will comprise the “unintentional, non-fire related” subset of this measure. *Note:* If a record has both a fire related and other cause of CO poisoning, classify as “unknown.”

Categorize all remaining cases, including those without E-coding, as “unknown.”

- Restrict the dataset to patients who were admitted from an ED using the following criteria:
 - point of origin code indicates emergency department, or
 - CPT codes: 99281–99285, or
 - revenue codes: 0450–0459, or
 - positive ED charges
- These criteria are consistent with the criteria used by AHRQ (see: http://www.hcup-us.ahrq.gov/db/vars/siddistnote.jsp?var=hcup_ed).
- Exclude all records where the State of residence is not your state.

c. From Observation Stay data

In states where observation stays are identified separately, include these observation stay records with ED Visits. Not all states require the reporting of observation stay records. Contact data stewards to determine whether records for observation stays are

	<p>collected and if so, if the records are located with outpatient or inpatient records, or in a separate file. Observation Stays can be identified by selecting all the records that meet the following criteria:</p> <table border="0"> <thead> <tr> <th style="text-align: left;">Code</th> <th style="text-align: left;">Description</th> </tr> </thead> <tbody> <tr> <td>986</td> <td>Toxic effect of carbon monoxide</td> </tr> <tr> <td>E868.2</td> <td>Accidental poisoning by motor vehicle exhaust gas</td> </tr> <tr> <td>E868.3</td> <td>Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels</td> </tr> <tr> <td>E868.8</td> <td>Accidental poisoning by carbon monoxide from other sources</td> </tr> <tr> <td>E868.9</td> <td>Accidental poisoning by carbon monoxide, unspecified source</td> </tr> <tr> <td>E982.0</td> <td>Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted</td> </tr> <tr> <td>E982.1</td> <td>Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted</td> </tr> </tbody> </table> <ul style="list-style-type: none"> • AND <ul style="list-style-type: none"> ○ revenue code: 762, or ○ positive OS charge when revenue codes not available, or ○ CPT codes: 99217–99220 or 99234–9923 • <i>Remove and exclude</i> records having any intentional cause of carbon monoxide poisoning (E952.0, E952.1) or other intentional injury (E950.0–E979.9, E990.0–E999). <p>Of the selected cases, identify and categorize any accidental injury/poisoning due to fire and flames (E890–E899). These cases will comprise the “unintentional, fire related” subset of this measure.</p> <ul style="list-style-type: none"> • Of the remaining selected cases, identify and categorize cases of carbon monoxide poisoning due to all other causes (E818, E825, E838, E844, E867, or E868). These cases will comprise the “unintentional, non-fire related” subset of this measure. Note: If a record has both a fire related and other cause of CO poisoning, classify as “unknown.” <p>Categorize all remaining cases, including those without E-coding, as “unknown.”</p> <ul style="list-style-type: none"> • These criteria are consistent with the criteria used by AHRQ (see: http://www.hcup-us.ahrq.gov/db/vars/siddistnote.jsp?var=hcup_ed). 	Code	Description	986	Toxic effect of carbon monoxide	E868.2	Accidental poisoning by motor vehicle exhaust gas	E868.3	Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels	E868.8	Accidental poisoning by carbon monoxide from other sources	E868.9	Accidental poisoning by carbon monoxide, unspecified source	E982.0	Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted	E982.1	Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted
Code	Description																
986	Toxic effect of carbon monoxide																
E868.2	Accidental poisoning by motor vehicle exhaust gas																
E868.3	Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels																
E868.8	Accidental poisoning by carbon monoxide from other sources																
E868.9	Accidental poisoning by carbon monoxide, unspecified source																
E982.0	Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted																
E982.1	Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted																
Step #3	<p><i>Identifying the required date file content</i></p> <p>Each record should include the following variables:</p> <ul style="list-style-type: none"> • Any primary or other diagnosis code • Date of admission • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient’s sex 																

- Patient's race (optional) – White, Black, Other, Unknown
- Patient's ethnicity (optional) – Hispanic, Non-Hispanic, Unknown
- County of residence
- State of residence

Data Specifications

Refer to the Data Dictionary in order to conform with the coding specifications required for the NCDM variables. Note that the county FIPS code is 5 digits and that the first two digits will be the same as the state FIPS code. Note also that some variables are optional.

- For SAS users, the admission date (and discharge and birth date) variable is acceptable in the following formats:
 - SAS DATE FORMAT
 - MMDDYYYY
 - MMDDYY
 - MM/DD/YYYY
 - MM-DD-YYYY
 - DDMONYYYY
 - DDMONYY
 - YYYYMMDD
 - DDMONYYYY:00:00:00
- The base format for counts and population data should be by 5-year age groups beginning 0–4 and ending with 85+. ED Visit counts must be submitted to CDC by these 5-year age groups (see Data Dictionary). Refer to the measure-specific step for the appropriate age groups for calculation and presentation. In summary, the ED Visit and population age-groups required for the calculation and presentation of measures are:
 - CO Poisoning counts to CDC: 5-yr age-groups (0–4, 5–9 ... 85+)
 - CO Poisoning age-specific rate presentation: 0–4, 5–14, 15–24, 25–34, 35–44, 45–54, 55–64 and 65+
 - CO Poisoning crude and age-adjusted rates: 5-yr age groups
- Race and ethnicity variables are optional. Therefore, data files and counts and measures may be generated without specifying race or ethnicity if these data are missing or considered unreliable/inaccurate. If race and ethnicity data are reported, the NCDMs will only be generated when the source variables for race and ethnicity are separate variables. The NCDMs will not be generated if race and ethnicity are reported as a combined race/ethnicity variable. If race and ethnicity data are being provided make sure that the coding structure conforms to that described in the Data Dictionary.
 - Race: White; Black; Other; Unknown.
 - Ethnicity: Hispanic; non-Hispanic; Unknown.

Please consult your data steward and data managers to understand what types of ED

	Visits are included and excluded (e.g., resident out-of-state ED Visits) and the available variables and coding system (e.g., some data stewards may code race and ethnicity as one variable whereas others may code them as separate variables).
Step #4	<p><i>Selecting records for year of interest:</i> 2000–present calendar year. The SAS program (not provided by CDC) prepares one NCDM file for each year, as required for CDC submission.</p> <p>Most ED Visit data are released in annual discharge-based datasets. In order to have complete admission date information using discharge-based datasets, it is necessary to have the dataset of the year of interest <u>and</u> the subsequent year. Since hospital data is based on discharge year and the EPHT dataset is to be based on admission date, patients discharged in current year (i.e., 2006) but admitted to the ED in the previous year (i.e., 2005) should be counted as 2005 ED Visits.</p>
Step #5	<p><i>Removal of duplicates:</i> This How-to-Guide and accompanying SAS code presumes that the user has already removed any duplicate records, while keeping multiple ED Visits.</p> <p>The following variables may be used to identify duplicate records: hospital code, medical record number, admission date, discharge date, date of birth, sex, and zip code.</p> <p>Duplicate records may also be due to continuation of data beyond a single record line. In this case, duplicates may be identified using a record sequence number.</p> <p>GO TO SECTION B FOR INSTRUCTIONS ON CREATING AN XML FILE IF NOT USING SAS.</p> <p>GO TO SECTION C OR INSTRUCTIONS ON CREATING AN XML FILE IF USING SAS.</p>
Section B: CREATION OF XML DATA FILE FOR NCDMS WHEN NOT USING OPTIONAL SAS CODE	
Step #1	<p><i>Required date file:</i> Each record should include the following variables:</p> <ul style="list-style-type: none"> • Any primary or other diagnosis code • Date of visit • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient’s sex • Patient’s race (optional) – White, Black, Other, Unknown • Patient’s ethnicity (optional) – Hispanic, Non-Hispanic, Unknown • County of residence • State of residence
Step #2	Create required fields according to the specifications of each field provided in the Data Dictionary.
Step #3	Convert the data file to the .XML file format and insert the required header into the XML file, according to the Schema found on SharePoint. Refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data

	Dictionary.
Step #4	Submit completed XML file to CDC using PHIN-MS. <i>GO TO SECTION D FOR INSTRUCTIONS ON CALCULATING MEASURES FOR GRANTEE PORTALS.</i>
Section C: CREATION OF XML DATA FILE AND NCDM FILE FOR NCDMS USING OPTIONAL SAS CODE	
Step #1	<p><i>Create SAS Datasets:</i> Be sure to make copies of the inpatient and outpatient data before proceeding. If the ED Visit data is not in SAS format, convert it to SAS format and save it as a permanent dataset for creating the XML and measures. Keep the following variables in the SAS dataset :</p> <ul style="list-style-type: none"> • Any primary or other diagnosis code • Date of vist • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient’s sex • Patient’s race and ethnicity (<i>optional</i>) • County of residence • State of residence
Step #2	<p><i>Create XML File:</i> After creating the SAS datasets, download ED-NCDM containing the recommended SAS code and Users Guide. These can be found on SharePoint. The current version will only create xml files (not measures).</p> <p>Unzip the files and read and follow the User’s Guide.</p> <p>Click ED-NCDM.msi to launch the setup wizard. Double click the shortcut icon to launch the program.</p> <p>XML files for each single year of data will be generated for one outcome, as required for CDC submission.</p>
Step #3	<p><i>Creating Measures using SAS:</i> SAS menu options 3–9 generate the required NCDMs. These options are currently non-operational. These options will follow the steps described below in Section D. If selecting menu options 3–9, this SAS program will generate a final output in the form of MS Excel spreadsheet.</p> <p>Keep only those records that meet the following criteria:</p> <ul style="list-style-type: none"> • Any primary or other diagnosis codes for CO poisoning • State = Your state • Date of Birth (if available and being used to calculate patient age) is not missing • Patient’s age at the time of ED Visit is not missing • Date of Discharge is not missing • Date of Admission is not missing. <p><i>Population Data:</i> US Census bureau residential population data for state and county. Intercensal population estimates should be used for the intercensal years, and population</p>

	extrapolations for postcensal years.
Section D: GENERATE MEASURES FOR GRANTEE PORTALS	
ANNUAL NUMBER OF ED VISITS	
Step #1	Annual Number of ED Visits by cause (fire-related, non-fire related, and unknown) and total
	<p><i>State:</i> Calculate the number of visits during the year of interest by cause (fire-related, non-fire related, and unknown). Then sum the number of visits across causes (fire-related, non-fire related, and unknown) for the total annual number of ED Visits for the state.</p> <p><i>County:</i> Calculate the number of visits during the year of interest by county of residence and cause (fire-related, non-fire related, and unknown). Then sum the number of visits across cause (fire-related, non-fire related, and unknown) to get the total annual number of ED Visits by county of residence.</p>
Step #2	Annual number of ED Visits by race (<i>optional</i>) and total
	<p><i>State:</i> Calculate the number of visits during the year of interest by race (white, black, other, unknown). Then sum the number of visits across race (white + black + other + unknown) for the total annual number of ED Visits for the state.</p> <p><i>County:</i> Calculate the number of visits during the year of interest by county of residence and race (white, black, other, unknown). Then sum the number of visits across race (white + black + unknown) to get the total annual number of ED Visits by county of residence.</p>
MONTHLY NUMBER OF ED VISITS (<i>optional</i>)	
Step #4	Average Number of ED Visits per Month (<i>optional</i>)
	<p>NOTE: Average number of ED Visits per month is <u>not</u> a required NCDM and is not submitted to CDC or required to be placed on grantee portals. Because of the potential future use of this measure, it is included in the How-to-Guide.</p> <p><i>State:</i> Calculate the number of ED Visits for the state for a given month during the year of interest. Then divide the monthly totals by the number of days in that month (i.e. the denominator for January would be 31), adjusting for leap years when necessary.</p> <p><i>County:</i> Calculate the number of ED Visits by county of residence for the year of interest. Then divide the monthly total by the number of days in that month (i.e. the denominator for January would be 31), adjusting for leap years when necessary.</p>
DAILY NUMBER OF ED VISITS (<i>optional</i>)	
Step #5	<p>NOTE: Daily number of ED Visits is <u>not</u> a required NCDM and is not submitted to CDC or required to be placed on grantee portals. Because of the potential future use of this measure, it is included in the How-to-Guide.</p> <p>Sum the total number of ED Visits for each day by cause during the year of interest for the entire state by cause. Add the daily number of male, female, and unknown</p>

	(including missing sex information) to obtain the total number of daily admissions. Repeat the above by race/ethnicity and 5-year age groups to calculate the daily number of ED Visits by race/ethnicity and age groups.
ANNUAL UNADJUSTED (CRUDE) RATE OF ED VISITS	
Step #6	Annual ED Visit rate by cause (fire-related, non-fire related, and unknown) and total per 100,000 population
	<p>Sum number of CO poisoning cases by cause (fire-related, non-fire related, and unknown) and sum all CO poisoning cases to obtain total number.</p> <p>Use U.S. Census Bureau residential population data for state and county (see Section C, Step #3).</p> <p><i>State (required)</i></p> <ul style="list-style-type: none"> • <i>Numerator:</i> The annual number of ED Visits state by cause and total for the year of interest • <i>Denominator:</i> The population for the by year of interest • <i>Constant:</i> 100,000 • <i>Formulas:</i> <p>Unadjusted (Crude) Rate for total population per 100,000 people = (# male + # female) annual ED Visits / total state population × 100,000</p> <p><i>County (required)</i></p> <ul style="list-style-type: none"> • <i>Numerator:</i> The annual number of ED Visits state by cause and total by county of residence for the year of interest. • <i>Denominator:</i> The population for each county of residence in the state • <i>Constant:</i> 100,000. • <i>Formulas:</i> <p>Unadjusted (Crude) Rate for total population per 100,000 people = (# of male annual ED Visits + # of female annual ED Visits) for each county of residence / total county population × 100,000</p>
ANNUAL AGE ADJUSTED RATE OF ED VISITS	
Step #7	Annual age-adjusted rate of ED Visits by cause (fire-related, non-fire related, and unknown) and total per 100,000 population
	<p>Obtain the totals for each age-group. To calculate age-specific rates (for the 5-year age categories 0-4, 5-9...85+), use US Census bureau residential population data for denominators. Intercensal population estimates should be used for the intercensal years. The standard population should be the 2000 U.S. Standard Population divided into 18 age groups (http://seer.cancer.gov/stdpopulations/). For tutorial on age-adjustment see http://seer.cancer.gov/seerstat/tutorials/aarates/definition.html.</p> <p><i>State:</i></p> <ul style="list-style-type: none"> • Calculate age-specific rates for CO poisoning cause and total for 5-year age groups (0-4, 5-9, ..., 85+) by dividing the number of state ED Visits in that age group by

	<p>the Census state population of same age group.</p> <ul style="list-style-type: none"> • Compute age-adjustment population weights total for 5-year age groups using the 2000 US Standard population as follows: Age-adjusted weight = age-specific std pop/total std pop. • Multiply the age-specific rate × age adjustment weight for each age group for total. • Compute age-adjusted ED Visit rate for total by summing the product of the previous step for each age group i: $\sum(\text{rate}_i \times \text{weight}_i)$ <p><i>County:</i></p> <ul style="list-style-type: none"> • Calculate age-specific rates total for 5-year age groups (0-4, 5-9, ..., 85+) by dividing the number of county ED Visits in that age group and sex by the Census county population of same age-age group and sex. • Compute age-adjustment population weights total for 5-year age groups using the 2000 US Standard population as follows: Age-adjusted weights = age-specific std pop/total std pop. • Multiply the age-specific rate × age adjustment weight for each age group • Compute age-adjusted ED Visit rate for total by summing the product of the previous step for each age group i: $\sum(\text{rate}_i \times \text{weight}_i)$ <p><i>Confidence Intervals (optional):</i></p> <ul style="list-style-type: none"> • 95% confidence intervals may be calculated. <p>Lower Confidence Limit (LCL) = [age-adjusted rate – { 1.96 × age-adjusted rate / SQRT (Number of ED Visits) }]</p> <p>Upper Confidence Limit (UCL) = [age-adjusted rate + { 1.96 × age-adjusted rate / SQRT (Number of ED Visits) }]</p> <p>Please Note: With small numbers of ED Visits (i.e. ED Visits < 20), calculation methods assuming a non-normal distribution may be more appropriate.</p>
Section E: PRESENTATION & DISPLAY	
Aggregation & Suppression	Follow your state’s rules, laws, and regulations as well as rules and agreements between you and your data partner(s) in determining whether and when small cell values need to be suppressed.
Visual display	<p>If using optional SAS code, export SAS datasets to Microsoft Excel® for creating charts or for formatting the computed values for presentation.</p> <p>Aggregations calculated under the “Data Measurements” should be displayed, using Microsoft Excel® or equitable spreadsheet product, by state at a minimum and if available and appropriate by county. Recommended spreadsheet displays include:</p> <ul style="list-style-type: none"> • Annual number of CO Poisoning ED Visits by cause, state, and county • Unadjusted (crude) rate of CO Poisoning ED Visits by cause, state, and county

	<ul style="list-style-type: none"> • Age-Adjusted rate of CO Poisoning ED Visits by cause, state, and county • Average number of daily visits per month by cause, state, and county <p>Annual number of ED Visits can be displayed by showing sex on x-axis and number of ED Visits on y-axis. Similarly sex on x-axis can be replaced by race or age groups to display the number of ED Visits by race or age-group. Displays by race and sex are optional.</p> <p>These bar charts can be created by using any spreadsheet application or by using SAS.</p> <p>Pie charts and bar charts should be used as supplementary visual displays in conjunction with spreadsheets for aggregated calculations.</p> <p>Mapping of calculated counts and rates should be done on the county level. Recommended maps include:</p> <ul style="list-style-type: none"> • Annual number of CO Poisoning ED Visits by county per year • Age-Adjusted rate of CO Poisoning Ed Visits by county per year <p>Public can view bar charts and map showing the state and county level CO Poisoning measures. These visual displays will show whether the selected geographical area has a high, medium high, medium low, or low ED Visits rate. The public will also be able to see the links to other related information from various national, state and local sources.</p> <p>Mapping the rate of ED Visits per 100,000 residents will allow users to assess the level of environmentally related risk factors in their residential geographic area as well as in the surrounding geographical area. Future mapping may include other risk factors such as poverty and other potential risk factors. One limitation of mapping is that not all sources of CO Poisoning risk factors may be mapped. For example, indoor mold, dust, and pollen.</p>
Interpretation	<p><i>Small Numbers:</i> Measures that generate counts or rates based upon numbers that are small and potentially violate state privacy guidelines can collapse data across years or geographic units to generate data that can be released.</p> <p><i>Measures for multiple years:</i> The how-to-guide steps can be repeated for additional years of ED Visit data. Multi-year ED Visit data can be merged to create one dataset. Add the number of ED Visits for each year in multi-year cohort and divide by the number of years to calculate an average annual number of ED Visits.</p>

Indicator Template
Content Area: Carbon Monoxide Poisoning
Indicator: Carbon Monoxide Poisoning Emergency Department Visits

Environmental Public Health Tracking

Type of EPHT Indicator	Health outcome and exposure
Measures	<ol style="list-style-type: none"> 1. Age-adjusted rate of emergency department visits for CO poisoning per 100,000 population 2. Crude rate of emergency department visits for CO poisoning per 100,000 population 3. Number of emergency department visits for CO Poisoning
Derivation of Measure(s)	<p><i>Numerator:</i> Resident emergency department visits for CO poisoning that meet the 1998 CSTE case definition for public health surveillance for a "Confirmed" or "Probable" case of acute CO poisoning in administrative data sets.</p> <p>Frequencies for three unique groups: Unintentional, non-fire related Unintentional, fire-related Unknown intent</p> <p><i>Denominator :</i> Midyear resident population <i>Adjustment:</i> Age-adjustment by the direct method to year 2000 US Standard Population</p>
Unit	<ol style="list-style-type: none"> 1. Age-adjusted rate per 100,000 population 2. Rate per 100,000population 3. Number
Geographic Scope	State and national
Geographic Scale	Residents of jurisdiction – State, County
Time Period	Hospital admissions between January 1 to December 31, inclusive, for each year, 2000–
Time Scale	Calendar year
Rationale	<p>Carbon Monoxide (CO) poisoning is preventable; nonetheless, unintentional, non-fire-related CO poisoning is responsible for approximately 15,000 emergency department visits and nearly 500 deaths annually in the United States. During 2004–2006, an estimated average of 20,636 ED visits for nonfatal, unintentional, non-fire-related CO exposures occurred each year. Approximately 73% of these exposures occurred in homes, and 41% occurred during winter months (December–February). Prevention efforts targeting residential and seasonal CO exposures can substantially reduce CO-related morbidity. During 2000–2009, a total of 68,316 CO exposures were reported to poison centers across United States.</p> <p>Persons admitted to emergency departments and diagnosed with CO poisoning that ranges from suspected exposure to severe poisonings that may result in treatment and release, hospitalization, or death. Emergency department visits represent</p>

	patients not counted in other clinical settings. Emergency department data are available in more than 50% of states and that number is increasing.
Use of the Measure	These data can be used to assess the burden of severe CO poisoning, monitor trends over time, identify high-risk groups, and enhance prevention, education, and evaluation efforts.
Limitations of the Measure	<p>This data may not include:</p> <ul style="list-style-type: none"> • Persons who call poison control centers and are managed at the scene, and/or receive medical care but are not treated at the emergency department • persons who do not seek any medical care • persons who die immediately from CO exposure without medical care
Data Sources	<p><i>Numerator:</i> State inpatient hospital discharge data <i>Denominator:</i> US Census Bureau population data</p>
Limitations of Data Sources	<p><i>State hospital discharge data:</i> The use and quality of ICD-9-CM coding varies across jurisdictions; this is especially true of the codes used to describe how an injury occurs, indicated as E-codes. Examples of this variation include:</p> <p>The number of diagnostic fields available to specify cause of the injury; Whether E-codes are mandated; The completeness and quality of E-coding; for example, the reliability of ICD-9-CM coding to distinguish between cases of CO poisoning that are intentional or unintentional, and/or fire-or non-fire related</p> <p>The toxic effects of CO exposure are nonspecific and easily misdiagnosed when CO exposure is not suspected. These misdiagnosed cases will not be counted.</p> <p>These data usually do not include data from federal facilities such as Veteran's Administration hospitals, Indian Health Services, or institutionalized populations (e.g., prisons).</p> <p>These data usually include only cases of state residents treated within the state. Health-care access is not restricted to these political boundaries so patients arriving at emergency departments for CO poisoning in another state may not be counted in their own state. Likewise, they may not be counted in the jurisdiction in which they were treated. Currently, few states have access to, or agreements to obtain, emergency department data from other states where their state residents may be hospitalized. To the extent that patients are treated out of state, there is undercounting of the rate of state residents poisoned by CO.</p> <p>Race and ethnicity are important risk factors for CO poisoning, yet many hospitalization data sets do not contain these data. Those that do may have data quality issues.</p> <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census Bureau. These need to be extrapolated or purchased from a vendor.

Related Indicators	<ul style="list-style-type: none"> • Age-adjusted rate of hospitalization for CO poisoning per 100,000 population • Crude rate of hospitalization for CO poisoning per 100,000 population • Number of hospitalizations for CO poisoning • Annual number of deaths from CO poisoning • Annual crude rate of death from CO poisoning • Annual age-adjusted rate of death from CO poisoning
References	<ol style="list-style-type: none"> 1. Centers for Disease Control and Prevention. Perspectives in Disease Prevention and Health Promotion Carbon Monoxide Intoxication—A Preventable Environmental Health Hazard MMWR Morb Mortal Wkly Rep 1982;31(39):529–31. 2. Centers for Disease Control Prevention. Nonfatal, unintentional, non-fire-related carbon monoxide exposures—United States, 2004-2006. MMWR Morb Mortal Wkly Rep 2008;57(33):896–9. 3. Hampson NB. Emergency department visits for carbon monoxide poisoning in the Pacific Northwest. J Emerg Med 1998;16(5):695–8. 4. Kao LW, Nanagas KA. Carbon monoxide poisoning. Emerg Med Clin North Am 2004;22(4):985–1018. 5. Partrick M, Fiessler F, Shih R, Riggs R, Hung O. Monthly variations in the diagnosis of carbon monoxide exposures in the emergency department. Undersea Hyperb Med 2009;36(3):161–7.

HOW-TO GUIDE

Carbon Monoxide Poisoning Hospitalizations

Environmental Public Health Tracking

07-18-2013

Data Source	Inpatient Hospitalization Admissions
NCDM Requirements	<ul style="list-style-type: none">• Health Outcome = Carbon monoxide (CO) poisoning• State/County of Residence• Admission Year/Month• Age Group• Sex• Race/Ethnicity (optional)• Transfers not to be excluded• Out-of-State residents to be excluded• Admissions to federal facilities to be excluded• Admissions of residents to out-of-state hospitals are to be optionally included
Measures	<ul style="list-style-type: none">• Age-adjusted rate of hospitalization for unintentional carbon monoxide poisoning per 100,000 population, stratified by cause: fire related, non-fire related, or unknown• Crude rate of hospitalization for unintentional carbon monoxide poisoning per 100,000 population, stratified by cause: fire related, non-fire related, or unknown• Number of hospitalizations for carbon monoxide poisoning, stratified by cause: fire related, non-fire related, or unknown
Definitions	<p><i>Admission date:</i> The date of the hospital admission; month, day, and year. Month and year are required.</p> <p><i>Discharge date:</i> The date of discharge from hospital. When the date of the hospital discharge is not available, use the admission date, if available.</p> <p><i>Duplicate records:</i> More than one record for the same person with the same hospital admission data (e.g., where sex, date of birth, admission date, and zip code have exactly same information).</p> <p><i>Event/Event Year:</i> A hospital admission for CO Poisoning results with a primary or other diagnosis during a specific calendar year. Event year is based only upon admission year, even when discharge year is different.</p> <p><i>CO Poisoning:</i> This indicator tracks acute, unintentional carbon monoxide poisoning resulting in hospitalization. Carbon monoxide is an odorless, colorless gas that is the byproduct of combustion, which preferentially binds to hemoglobin and therefore displaces oxygen in the blood stream. Carbon monoxide is the leading cause of acute, unintentional poisoning and death (excluding alcohol and drug-related intoxication).</p> <p><i>Hospital Transfers:</i> Generally, a patient discharged from one facility and readmitted to a</p>

	<p>second facility on the same day.</p> <p><i>Hospitalization/ Hospital Admission:</i> Condition of being placed (Admission) or treated as a patient in an acute care hospital for treatment as an inpatient. Treatment as an out-patient is not considered to be hospitalization. To be considered as inpatient Hospitalization, a minimum stay is required (often over 23 hours).</p> <p><i>Multiple admissions:</i> Second or subsequent admission for the same person for the same primary diagnosis code but on a different date and related to a separate event within a given year. Multiple admissions are considered separate events (generally at least 48 hours apart).</p> <p><i>Out-of-State admissions:</i> When a resident of the grantee state is admitted to a hospital located in another state (usually an abutting state).</p> <p><i>Primary Diagnosis Code:</i> Presently, diagnosis codes are represented by ICD-9-CM codes (the International Classification of Diseases, 9th Revision, Clinical Modification). Carbon monoxide is classified as any primary or other diagnosis code of 986, or cause of injury code E868.2, E868.3, E868.8, E868.9, E982.0, or E982.1. Cases with any intentional cause of carbon monoxide poisoning (E952.0, E952.1) or other intentional injury (E950.0-E979.9, E990.0-E999) anywhere in the record are <u>excluded</u>.</p> <p><i>Resident:</i> Any person with a residential address in the county/state of the grantee at the time of the hospital admission.</p>
<p>How-to-Guide Requirements and Cautions</p>	<ul style="list-style-type: none"> • This How-to-Guide has two purposes: 1) Guide the user in the development of the XML dataset using the SAS code, located on SharePoint, for submission to CDC; 2) Describe the calculation steps for creating the Nationally Consistent Data Measures (NCDMs). Grantees should use the How-to-Guide in order to ensure that the calculation of measures are consistent with those required by CDC in the preparation of their own computer code. • Note that one set of SAS code (not provided by CDC’s EPHT program) is designed to handle the creation of XML files for all hospitalization (inpatient) outcomes (separate code is available for all ED Visit outcomes). The code manages the differences in definitions between the outcomes and generates a separate file for each outcome by each single year. • This how-to-guide and accompanying SAS code (not provided by CDC’s EPHT program) presume that the user already removed any duplicate records (see definitions for more information), while keeping multiple admissions. • Hospitalizations due to transfers between acute care hospitals are not excluded in the counts/measures to be generated. Therefore, for consistency, it is advised that transfers not be excluded. An algorithm to exclude transfers is underdevelopment. NOTE: The Date Dictionary includes two variables regarding the exclusion of transfers. These are placeholders only (to be activated in future work) and the SAS code will automatically set the codes for these variables the same for all grantees. These variables do not need to be a part of your SAS dataset. If they are present, the program will ignore them. • The data source is an inpatient discharge dataset but the EPHT dataset is based upon

	<p>date of admission because of the goal of relating a hospitalization event with an environmental event. Therefore, the hospitalization counts and measures require the development of an admission dataset. For admissions at the end of a calendar year where the discharge date is in the following year, that latter year's discharge dataset will be required before the admission dataset for the preceding year can be considered complete.</p> <ul style="list-style-type: none"> • Data suppression/aggregation rules are not incorporated into the SAS code. Suppression guidelines are currently applied by CDC for the national portal. • Please note that the steps for estimating age-adjusted rates are not the same for different health outcomes. • Admissions of residents to out-of-state hospitals are not required to be included. However, when available these admissions should be included. It is noted that some states must include out-of-state admissions of its residents. Use the “OUTOFSTATEEXCLUSION” variable in the dataset to capture whether out of state admissions are included or not (the Data Dictionary and schema provide for formal notation in the dataset on whether these admissions are included). Be certain to use footnotes and metadata to acknowledge the disposition of these admissions. • Admissions to federal facilities, such as Veteran’s Hospitals, are not included. Be certain to inform CDC if you state requires that your dataset includes admissions to federal facilities so that the measures can be appropriately footnoted. • The Data Dictionary should be referred to for the standardized definitions and notations of the variables to be submitted to CDC.
--	--

NOTE FOR NON-SAS USERS: Grantees not using SAS should use the steps outlined below for guidance on important issues such as population requirements and should refer to the Data Dictionary for specifications of the required fields.

The data file should be converted to the .XML file format and the required header inserted into the XML file, according to the Schema found on SharePoint. In addition, refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data Dictionary).

NOTE FOR SAS USERS: SAS code is not developed or distributed by CDC.

STEPS FOR CREATING SAS HOSPITALIZATION DATASET

<p>Step #1</p>	<p><i>Source of Data:</i> Individual level state inpatient hospital admission data based on primary diagnosis.</p> <p>Please consult your data steward and data mangers to understand the variables and coding system, specifically for race and ethnicity variables. In some states these variables may be coded as one variable whereas in others they are coded as separate variables.</p> <p><i>Years of Interest:</i> 2000–present calendar year; (<u>submit all new years of data, which was</u></p>
----------------	--

previously not submitted) The SAS program (not provided by CDC) prepares one NCDM file for each year, as required for CDC submission.

Most hospitalization data is released in annual discharge-based datasets. In order to have complete admission date information using discharge-based datasets, it is necessary to have the dataset of the year of interest and the subsequent year. For example, to obtain all admissions during 2005 using discharge based datasets, it will be necessary to have both the 2005 and the 2006 discharge datasets for admissions that occurred in 2005 but were not reported until release of the 2006 discharge dataset. For this example, 2005 data should not be submitted prior to receipt of the 2006 discharge dataset from the data steward.

Removal of Duplicates: This how-to-guide and accompanied SAS code presumes that the user has already removed any duplicate records, while keeping multiple admissions.

Data Specifications: Refer to the Data Dictionary in order to conform to coding specifications required for the NCDM variables.

NOTE: county FIPS code is 5 digits and that the first two digits will be the same as the state FIPS code. Also, some variables are optional.

Select all hospital records that meet the following criteria:

- Exclude all records where the State of residence is not your state.
- Since hospitalizations data are based on discharge year and the EPHT dataset is to be based on admission date, patients discharged in current year (i.e., 2006) but admitted in the previous year (i.e., 2005) should be counted as 2005 hospitalizations. The admission date (and discharge and birth date) variable is acceptable in the following formats:
 - SAS DATE FORMAT
 - MMDDYYYY
 - MMDDYY
 - MM/DD/YYYY
 - MM-DD-YYYY
 - DDMONYYYY
 - DDMONYY
 - YYYYMMDD
 - DDMONYYYY:00:00:00

SAS Dataset: Make a copy of the hospital admission data before proceeding to next step. If the admission data are not in SAS format, convert it to SAS format and save it as a permanent dataset for creating the XML and measures. Keep the following variables in the SAS dataset:

- **Any** or Primary diagnosis code
- Date of admission
- Date of discharge
- Patient date of birth OR Age at admission
- Patient's sex
- Patient's race (optional) - White, Black, Other, Unknown

	<ul style="list-style-type: none"> ○ Patient's ethnicity (optional) - Hispanic, Non-Hispanic, Unknown ○ County of residence ○ State of residence <p><i>Population Data:</i> US Census bureau residential population data for state and county. Intercensal population estimates should be used for the intercensal years, and population extrapolations for postcensal years.</p> <p><i>Go to Step # 2.</i></p>
Step #2	<p>The base format for counts and population data should be by 5-year age groups for CO Poisoning beginning 0–4 and ending with 85+.</p> <p>Hospitalization counts must be submitted to CDC by these 5-year age groups (see Data Dictionary). For the calculation of measures and presentation, the age-groups of interest for various CO Poisoning measures are different because of the nature of the diseases. Refer to the measure-specific step for the appropriate age groups for calculation and presentation. In summary, the hospitalization and population age-groups required for the calculation and presentation of measures are:</p> <p>CO Poisoning counts to CDC: 5-yr age-groups (0–4, 5–9 ... 85+)</p> <p>CO Poisoning age-specific rate presentation: 0–4, 5–14, 15–24, 25–34, 35–44, 45–54, 55–64 and 65+</p> <p>CO Poisoning crude and age-adjusted rates: 5-yr age groups</p> <p>Race and ethnicity variables are optional. Therefore, counts and measures may be generated without specifying race or ethnicity if these data are missing or considered unreliable/inaccurate. If race and ethnicity data are reported, the NCDMs will only be generated when the source variables for race and ethnicity are separate variables. The NCDMs will not be generated if race and ethnicity are reported as a combined race/ethnicity variable. If race and ethnicity data are being provided, be sure that the coding structure conforms to that laid out in the Data Dictionary.</p> <p>Race: White; Black; Other; Unknown.</p> <p>Ethnicity: Hispanic; non-Hispanic; Unknown.</p> <p><i>Go to Step # 3.</i></p>
<p>STEPS FOR GENERATING NCDM REQUIREMENTS</p> <p><i>(Grantees not using SAS should refer to the steps below for conversion of their data file to XML format)</i></p>	

Step #3	<p>After creating the SAS datasets, download IP-NCDM containing the recommended SAS code and Users Guide. These can be found on SharePoint. The current version will only create xml files (not measures).</p> <p>Unzip the files and read and follow the User’s Guide. Click IP-NCDM.msi to launch the setup wizard. Double click the shortcut icon to launch the program.</p> <p>XML files for each single year of data will be generated for one outcome, as required for CDC submission.</p> <p>SAS code to generate measures is not yet operational.</p> <p><i>NOTE:</i> Single year files for individual outcomes must be submitted, as specified in the data call letter</p> <p>Steps #4 through #12 should be used to calculate the NCDMs to ensure consistency between grantees.</p>
---------	--

STEPS FOR COMPLETING SPECIFIC MEASURES

Step #4	<p>SAS menu options 3–9 generate measures beyond those required for the national portal but are currently non-operational. These options will follow the steps below (4b through 12). These steps may also be used to generate measures outside of SAS. If selecting menu options 3–9, this SAS program will generate a final output in the form of MS Excel spreadsheet. A PDF file containing histograms will also be created.</p> <ul style="list-style-type: none"> - Select cases having any of the following ICD-9 codes as principal diagnosis, injury cause, or other diagnoses: <table border="0" style="margin-left: 40px;"> <thead> <tr> <th style="text-align: left;">Code</th> <th style="text-align: left;">Description</th> </tr> </thead> <tbody> <tr> <td>986</td> <td>Toxic effect of carbon monoxide</td> </tr> <tr> <td>E868.2</td> <td>Accidental poisoning by motor vehicle exhaust gas</td> </tr> <tr> <td>E868.3</td> <td>Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels</td> </tr> <tr> <td>E868.8</td> <td>Accidental poisoning by carbon monoxide from other sources</td> </tr> <tr> <td>E868.9</td> <td>Accidental poisoning by carbon monoxide, unspecified source</td> </tr> <tr> <td>E982.0</td> <td>Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted</td> </tr> <tr> <td>E982.1</td> <td>Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted</td> </tr> </tbody> </table> - State = Your state - Date of Birth (if available and being used to calculate patient age) is not missing - Patient’s age at the time of hospital admission is not missing - Date of Discharge is not missing - Date of Admission is not missing. <p><i>Remove and exclude</i> records having any intentional cause of carbon monoxide poisoning</p>	Code	Description	986	Toxic effect of carbon monoxide	E868.2	Accidental poisoning by motor vehicle exhaust gas	E868.3	Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels	E868.8	Accidental poisoning by carbon monoxide from other sources	E868.9	Accidental poisoning by carbon monoxide, unspecified source	E982.0	Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted	E982.1	Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted
Code	Description																
986	Toxic effect of carbon monoxide																
E868.2	Accidental poisoning by motor vehicle exhaust gas																
E868.3	Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels																
E868.8	Accidental poisoning by carbon monoxide from other sources																
E868.9	Accidental poisoning by carbon monoxide, unspecified source																
E982.0	Poisoning by motor vehicle exhaust gas, undetermined whether accidentally or purposefully inflicted																
E982.1	Poisoning by other carbon monoxide source, undetermined whether accidentally or purposefully inflicted																

	<p>(E952.0, E952.1) or other intentional injury (E950.0–E979.9, E990.0–E999).</p> <p>Of the remaining records, identify and categorize any accidental injury/poisoning due to fire and flames (E890–E899). These cases will comprise the “unintentional, fire related” subset of this measure.</p> <p>Of the remaining records, identify and categorize cases of carbon monoxide poisoning due to all other causes (E818, E825, E838, E844, E867, or E868). These cases will comprise the “unintentional, non-fire related” subset of this measure. Note: If a record has both a fire related and other cause of CO poisoning, classify as “unknown.”</p> <p>Categorize all remaining cases, including those without E-coding, as “unknown.”</p> <p>Assign geography by state and county of patient’s residence.</p> <p>Go to Step # 5.</p>
ANNUAL NUMBER OF HOSPITAL ADMISSIONS	
Step #5	Annual Number of Hospital Admissions by sex and total
Step #5a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by sex (male, female and unknown (including missing)).</p> <p>Sum the number of male, female and unknown sex hospital admissions to get the total number of hospital admissions for the year.</p>
Step #5b	<p><i>County:</i> Sum the total number of admissions by county of residence for year of interest by sex [male, female and unknown (including missing)].</p> <p>Sum the number of male, female and unknown sex hospital admissions to get the total number of hospital admission by county for the year.</p>
Step #6	Annual Number of Hospital Admissions by race and total
Step #6a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by race categories.</p> <p>Sum the number of hospital admissions to get the total number of CO Poisoning hospital admission for the year.</p>
Step #7	Annual Number of Hospital Admissions by age groups and total
Step #7a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by the specific 5-year age groups created in step #2.</p> <p>Sum the number of hospital admissions for each age group to get the total number of hospital admission for the year.</p>
DAILY NUMBER OF HOSPITAL ADMISSIONS (NOT A REQUIRED NCDM)	
Step #8	<p>NOTE: Daily number of admissions is not a required NCDM and is not submitted to CDC or required to be placed on state portals. Because of the potential future use of this measure. It remains in the How-to-Guide and SAS code.</p> <p>Sum the total number of admission for each day by sex during the year of interest for entire state to get the daily number of admissions by sex. Add the daily number of male,</p>

	female and unknown (including missing sex information) to obtain the total number of daily admissions. Repeat the above by race and 5-year age-groups to calculate the daily number of hospital admissions by race and age groups.
ANNUAL AGE-SPECIFIC HOSPITAL ADMISSIONS RATE	
Step #9	Annual age-specific hospital admission rate by sex
Step #9a	<i>Create the numerator data:</i> Sum the number of hospitalizations in the state for the year of interest in each of the 5-year age-groups for both male and female. Exclude any observation where sex is unknown or missing. Sum the admissions for male and female to obtain the total admissions for each age-group.
Step #9b	<i>Create the denominator data:</i> Sum the population in the state for year of interest in each 5-year age group for both male and female. Sum male and female population to obtain the total state population for each age-group.
Step #9c	Merge both numerator and denominator data by state of residence, year of hospital admissions, age-group and sex.
Step #9d	<i>For CO Poisoning presentation:</i> Compute age-specific rates for male, female and total for age-groups 0–4, 5–14, 15–24, 25–34, 35–44, 45–54, 55–64 and 65+ by dividing the number of hospital admissions in that age group and sex by the population of same age-age group and sex. For example, to calculate the age-specific rates of admissions in 5–14 year old male divide the annual number of hospital admissions in 5–14 years old male by the population of 5–14 years old male.
Step #9e	All rates are to be presented as per 100,000 population. Multiply the rates calculated in step 9d by 100,000 to obtain rate of admission per 100,000 population
Step #9f	Upper and lower confidence limits (95% confidence interval) for age-specific rates may be computed. For each age-specific rate computed in step 9d, compute Lower Confidence Limit (LCL) and Upper Confidence Limit (UCL) as follows LCL = [age-specific rate – {1.96 * age-specific rate/SQRT (Number of Admissions)}] UCL = [age-specific rate + {1.96 * age-specific rate/SQRT (Number of Admissions)}]
ANNUAL UNADJUSTED (CRUDE) RATE OF HOSPITAL ADMISSIONS	
Step #10a	Exclude any observation where sex is unknown or missing. <i>CO Poisoning:</i> Create the numerator data (referred to in Step #2) to obtain the annual number of hospital admissions by sex for both male and female across all ages. Sum the male and female number of admissions to obtain total admissions for both sexes. Exclude any observation where sex is unknown or missing
Step #10b	<i>CO Poisoning:</i> Create the denominator data as referred to in step #2 across all ages. Sum the population for male, female and both sex for the year of interest.
Step #10c	Merge both numerator and denominator data by state of residence, year of hospital admissions and sex.

Step #10d	<p>Compute the annual unadjusted rate of hospital admissions as follows:</p> <p>Unadjusted Admission Rate (Male) = (# of Male Admissions/Male Population)</p> <p>Unadjusted Admission Rate (Female) = (# of Female Admissions/Female Population)</p> <p>Unadjusted Admission Rate (Total) = (Total Admissions/ State Population)</p> <p>Multiply the above computed rates by 100,000 to obtain the number of admissions per 100,000 population.</p>
ANNUAL AGE ADJUSTED RATE OF HOSPITAL ADMISSIONS	
Step #11	Annual Age Adjusted Rate of Hospital Admissions for State by Sex
Step #11a	<i>CO Poisoning</i> : Calculate the age specific rates as described in steps 10a through 10d using 5 yr age groups (0–4, 5–9 ... 85+) for male, female and both sexes.
Step #11b	To calculate age-specific rates (for the 5-year age categories 0–4, 5–9... 85+); use U.S. Census bureau residential population data for denominators. Intercensal population estimates should be used for the intercensal years. . The standard population should be the 2000 U.S. Standard Population divided into 18 age groups. The link for the 2000 U.S. Standard Population is: http://seer.cancer.gov/stdpopulations/). After downloading, combine the '0' age group with the '1–4' age group. Calculate annual warm-season rates per 100,000 residents, adjusted for age, by state and county. Note: County-level measures may require aggregation of years due to small numbers.
Step #11c	Merge both numerator and denominator data by age group and sex.
Step #11d	<p>Compute the age-adjusted population weights using the 2000 US population as the standard.</p> <p><i>CO Poisoning</i> : Compute the age-adjustment weights of hospital admissions using 2000 US Standard Population by age group for males, females, and both sexes as follows:</p> <p>Age-adjusted weights = age-specific std pop/total std pop, where the total weight for all ages is 1.0.</p>
Step #11e	<p>Compute the age-adjusted hospital admissions rate:</p> <p>Age-adjusted rate = Sum of age-specific rate × age adjusted weight</p> <p>For tutorial on age-adjustment see http://seer.cancer.gov/seerstat/tutorials/aarates/definition.html .</p>
Step #11f	<p>95% confidence intervals may be computed.</p> <p>LCL = [age-adjusted rate – {1.96 × age-adjusted rate/SQRT (Number of Admissions)}]</p> <p>UCL = [age-adjusted rate + {1.96 × age-adjusted rate/SQRT (Number of Admissions)}]</p> <p>NOTE: With small numbers of hospitalizations (e.g., <20), calculation methods assuming a non-Normal distribution may be more appropriate.</p>
Step #11f	To calculate the Annual age-adjusted rate of hospital admissions by County (if needed), follow steps 11a through 11f using the same 2000 US standard population.
PRESENTATION	

Step #12a	Export SAS datasets to Microsoft Excel® for creating charts or for formatting the computed values for presentation
Step #12b	<p>Annual number of hospital admissions can be displayed by showing sex on x-axis and number of admissions on Y-axis. Similarly sex on X-axis can be replaced by race or age groups to display the number of admissions by race or age-group.</p> <p>These bar charts can be created by using any spreadsheet application or by using SAS.</p>
Additional visual display	<p>Public can view histograms and map showing the state or county level CO Poisoning measures. These visual displays will show whether the selected geographical area has a high, medium high, medium low or low hospital admission rate. The public will also be able to see the links to other related information from various national, state and local sources.</p> <p>Mapping the rate of hospital admissions per 100,000 residents will allow users to assess the level of environmentally related risk factors in their residential geographic area as well as in the surrounding geographical area. Future mapping may include other risk factors such as poverty and other potential risk factors. One limitation of mapping is that not all sources of CO Poisoning risk factors may be mapped.</p>
Interpretation	<p>Small Numbers: Measures that generate counts or rates based upon numbers that are small and potentially violate state privacy guidelines can collapse data across years or geographic units to generate data that can be released.</p> <p>Measures for multiple years: The how-to-guide steps can be repeated for additional years of hospital admission data. Multi-year hospital admission data can be merged to create one dataset. Add the number of hospital admissions for each year in multi-year cohort and divide by the number of years to calculate an average annual number of hospital admissions.</p>

Indicator Template
Content Area: Carbon Monoxide Poisoning
Indicator: Carbon Monoxide Poisoning Hospitalizations

Environmental Public Health Tracking

Type of EPHT Indicator	Health outcome/Exposure
Measures	<ol style="list-style-type: none"> 1. Age-adjusted rate of hospitalization for CO poisoning per 100,000 population 2. Crude rate of hospitalization for CO poisoning per 100,000 population 3. Number of hospitalizations for CO poisoning
Derivation of Measure(s)	<p><i>Numerator:</i> Resident hospitalizations for CO poisoning that meet the 1998 CSTE case definition for public health surveillance for a "Confirmed" or "Probable" case of acute CO poisoning in administrative data sets.</p> <p>Frequencies for three unique groups:</p> <ul style="list-style-type: none"> Unintentional, non-fire related Unintentional, fire-related Unknown intent <p><i>Denominator :</i> Midyear resident population</p> <p><i>Adjustment:</i> Age-adjustment by the direct method to year 2000 US Standard Population</p>
Unit	<ol style="list-style-type: none"> 1. Age-adjusted rate per 100,000population 2. Rate per 100,000population 3. Number
Geographic Scope	State and national
Geographic Scale	Residents of jurisdiction – State, County
Time Period	Hospital admissions between January 1 to December 31, inclusive, for each year, 2000–
Time Scale	Calendar year
Rationale	<p>Carbon monoxide (CO) is an odorless, colorless gas that usually remains undetectable until exposure results in injury or death. Each year in the United States, an estimated 10,000 persons seek medical attention or lose at least one day of normal activity because of CO intoxication. There is limited information on CO hospitalization. In Florida, 1,494 were hospitalized with a diagnosis of CO poisoning from 1999–2007. Out of which 10% (n=143) were unintentional fire-related, 33% (n=493) were unintentional non-fire-related, and 17% (n=256) were from unknown cause of CO poisoning. During 2000–2009, a total of 68,316 CO exposures were reported to poison centers across United States.</p> <p>Persons hospitalized with CO poisoning are among the most severely poisoned cases. Unintentional CO poisoning is almost entirely preventable. These data are available in most states.</p>
Use of the Measure	These data can be used to assess the burden of severe CO poisoning, monitor trends over time, identify high-risk groups, and enhance prevention, education, and evaluation efforts.

Limitations of the Measure	Hospitalization data, by definition, do not include: persons treated in outpatient settings (e.g., emergency departments, urgent care clinics, clinicians' offices or hyperbaric chambers but not hospitalized); persons who call poison control centers and are managed at the scene, and/or receive medical care but are not hospitalized; persons who do not seek any medical care; or persons who die immediately from CO exposure without medical care.
Data Sources	<i>Numerator:</i> State inpatient hospitalization data (using admission date) <i>Denominator:</i> US Census Bureau population data
Limitations of Data Sources	<p><i>State hospital discharge data:</i></p> <p>The use and quality of ICD-9-CM coding varies across jurisdictions; this is especially true of the codes used to describe how an injury occurs, indicated as E-codes. Examples of this variation include:</p> <ul style="list-style-type: none"> • The number of diagnostic fields available to specify cause of the injury; • Whether E-codes are mandated; • The completeness and quality of E-coding; for example, the reliability of ICD-9-CM coding to distinguish between cases of CO poisoning that are intentional or unintentional, and/or fire-or non-fire related <p>The toxic effects of CO exposure are nonspecific and easily misdiagnosed when CO exposure is not suspected. These misdiagnosed cases will not be counted.</p> <p>These data usually do not include data from federal facilities such as Veteran's Administration hospitals, Indian Health Services, or institutionalized populations (e.g., prisons).</p> <p>These data usually include only cases of state residents treated within the state. Health-care access is not restricted to these political boundaries so patients hospitalized for CO poisoning in another state may not be counted in their own state. Likewise, they may not be counted in the jurisdiction in which they were treated. Currently, few states have access to, or agreements to obtain, hospital discharge data from other states where their state residents may be hospitalized. To the extent that patients are treated out of state, there is undercounting of the rate of state residents poisoned by CO.</p> <p>Differences in rates between jurisdictions may reflect differences in hospital admissions practices for treating persons with severe CO poisoning. For example, some facilities may routinely admit all patients treated with hyperbaric oxygen; other facilities may release patients treated with hyperbaric oxygen after the treatment is completed if they are in stable condition.</p> <p>Race and ethnicity are important risk factors for CO poisoning, yet, many hospitalization data sets do not contain these data. Those that do may have data quality issues.</p> <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census

	Bureau. These need to be extrapolated or purchased from a vendor.
Related Indicators	<ul style="list-style-type: none"> • Age-adjusted rate of emergency department visits for CO poisoning per 100,000 population • Crude rate of emergency department visits for CO poisoning per 100,000 population • Number of emergency department visits for CO poisoning • Annual number of deaths from CO poisoning • Annual crude rate of death from CO poisoning • Annual age-adjusted rate of death from CO poisoning
References	<ol style="list-style-type: none"> 1. Centers for Disease Control and Prevention, Perspectives in Disease Prevention and Health Promotion Carbon Monoxide Intoxication—A Preventable Environmental Health Hazard MMWR, 1982. 31(39): p. 529–31. 2. Centers for Disease Control Prevention, Carbon monoxide exposures—United States, 2000–2009. MMWR, 2011. 60(30): p. 1014–7. 3. Harduar-Morano, L. and S. Watkins, Review of unintentional non-fire-related carbon monoxide poisoning morbidity and mortality in Florida, 1999–2007. Public Health Rep, 2011. 126(2): p. 240–50. 4. King, M.E. and S.A. Damon, Attitudes about carbon monoxide safety in the United States: results from the 2005 and 2006 Health Styles Survey. Public Health Rep, 2011. 126 Suppl 1: p. 100–7.

**Erratum:
Hospitalizations Data Call**

Document type: How-to-guide

Document Name: CO_Hosp_How_to_Guide_July 2014.pdf

Health Outcome: CO

Data Type: Hospitalizations

1. Page 1: Definitions: Event/Event Year: A hospital admission for CO Poisoning results with a primary diagnosis of ~~493.xx~~ during a specific calendar year. Event year is based only upon admission year, even when discharge year is different.
Correction: ~~493.xx~~ Update: Event/Event Year: A hospital admission for CO Poisoning results with a primary or any other diagnosis during a specific calendar year. Event year is based only upon admission year, even when discharge year is different.
2. A simplified tabular format for showing ICD-9-CM codes for fire, non-fire, and unknown cases is given below:

Exposure route strata
CO Related – Unintentional/Non Fire-Related: E868.2-E868.9; excluding any (E890-E899) or (E950-E979.9 or E990-E999)
CO Related – Unintentional/Fire-Related: E890-E899; excluding any (out-of-state) or (E950-E979.9 or E990-E999)
CO Related – Unknown Exposure/Intent: <ul style="list-style-type: none"> • 986, excluding any (E868.2-E868.9) or (E890-E899) or (E950-E979.9 or E990-E999) • E982.0-E982.1, excluding any (E868.2-E868.9) or (E890-E899) or (E950-E979.9 or E990-E999) • Cases with <u>both</u> E868.2-E868.9 <u>and</u> E890-E899, excluding any (E950-E979.9 or E990-E999)

Note: Above suggestions were provided by EPHT grantees WA (Steve Macdonald) and ME (Cathy Decker).

HOW-TO GUIDE

Acute Myocardial Infarction (AMI) Hospitalizations

Environmental Public Health Tracking

07-18-2013

Data Source	Inpatient Hospitalization Admissions
NCDM Requirements	<ul style="list-style-type: none"> • Health Outcome = Acute Myocardial Infarction (AMI) • State/County of Residence • Admission Year/Month • Age Group • Sex • Race/Ethnicity (optional) • Transfers not to be excluded • Out-of-State residents to be excluded • Admissions to federal facilities to be excluded • Admissions of residents to out-of-state hospitals are to be optionally included
Measures	<ul style="list-style-type: none"> • Annual Number of Hospital Admissions by age group, sex, race/ethnicity*, and county and state. • Annual Crude (unadjusted) Rate of Hospital Admissions for all ages by sex, race/ethnicity*, and county and state • Annual Age-Adjusted Rate of Hospital Admissions for all ages by sex, race/ethnicity*, and county and state • (<i>optional**</i>) Average Number of Hospitalizations per Month by age group, sex, race/ethnicity*, and county and state • (<i>optional**</i>) Daily Number of Hospitalizations by age group, sex, race/ethnicity*, and county and state <p>* measures by race/ethnicity are optional</p> <p>** optional measures are for state portal only and not submitted to CDC</p>
Definitions	<p><i>Acute Myocardial Infarction (AMI):</i> Irreversible death of heart muscle as a consequence of prolonged loss of blood supply; ICD-9 410.XX.</p> <p><i>Admission date:</i> The date of the hospital admission; month, day, and year. Month and year is required.</p> <p><i>Discharge date:</i> The date of discharge from hospital. When the date of the hospital discharge is not available, use the admission date, if available.</p> <p><i>Duplicate records:</i> More than one record for the same person with the same hospital admission data (e.g., where sex, date of birth, admission date, and zip code have</p>

	<p>exactly same information).</p> <p><i>Event/Event Year:</i> A hospital admission for AMI results with a primary diagnosis of 410.XX during a specific calendar year. Event year is based only upon admission year, even when discharge year is different.</p> <p><i>Hospital Transfers:</i> Generally, a patient discharged from one facility and readmitted to a second facility on the same day.</p> <p><i>Hospitalization/ Hospital Admission:</i> Condition of being placed (Admission) or treated as a patient in an acute care hospital for treatment as an inpatient. Treatment as an out-patient is not considered to be hospitalization. To be considered as inpatient Hospitalization, a minimum stay is required (often over 23 hours).</p> <p><i>Multiple admissions:</i> Second or subsequent admission for the same person for the same primary diagnosis code but on a different date and related to a separate event within a given year. Multiple admissions are considered separate events (generally at least 48 hours apart).</p> <p><i>Out-of-State admissions:</i> When a resident of the grantee state is admitted to a hospital located in another state (usually an abutting state).</p> <p><i>Primary Diagnosis Code:</i> The first diagnosis field(s) of the coded clinical record (i.e., primary or principle diagnosis). Presently, the code is represented by an ICD-9-CM code (the International Classification of Diseases, 9th Revision, Clinical Modification). For myocardial infarction that code is 410.XX.</p> <p><i>Resident:</i> Any person with a residential address in the county/state of the grantee at the time of the hospital admission.</p>
<p>How-to-Guide Requirements and Cautions</p>	<ul style="list-style-type: none"> • This How-to-Guide has two purposes: 1) Guide the user in the development of the XML dataset using the SAS code, located on SharePoint, for submission to CDC. 2) Describe the calculation steps for creating the Nationally Consistent Data Measures (NCDMs). Grantees should use the How-to-Guide in order to ensure that the calculation of measures are consistent with those required by CDC in the preparation of their own computer code. • Note that one set of SAS code is designed to handle the creation of XML files for all hospitalization (inpatient) outcomes (separate code is available for all ED Visit outcomes). The code manages the differences in definitions between the outcomes and generates a separate file for each outcome by each single year. • This how-to-guide and accompanying SAS code presume that the user already removed any duplicate records (see definitions for more information), while keeping multiple admissions. • Hospitalizations due to transfers between acute care hospitals are not excluded in the counts/measures to be generated. Therefore, for consistency, it is advised that transfers not be excluded. An algorithm to exclude transfers is underdevelopment. NOTE: The Date Dictionary includes two variables regarding the exclusion of transfers. These are placeholders only (to be activated in future work) and the SAS code will automatically set the codes for these

variables the same for all grantees. These variables do not need to be a part of your SAS dataset. If they are present, the program will ignore them.

- The data source is an inpatient discharge dataset but the EPHT dataset is based upon date of admission because of the goal of relating a hospitalization event with an environmental event. Therefore, the hospitalization counts and measures require the development of an admission dataset. For admissions at the end of a calendar year where the discharge date is in the following year, that latter year's discharge dataset will be required before the admission dataset for the preceding year can be considered complete.
- Data suppression/aggregation rules are not incorporated into the SAS code. Suppression guidelines are currently applied by CDC for the national portal.
- The steps for estimating age adjusted rates are not the same for asthma and acute myocardial infarction. This is because the age-groups are different, requiring steps for population weighting in the case of acute myocardial infarction.
- Admissions of residents to out-of-state hospitals are not required to be included. However, when available these admissions should be included. It is noted that some states must include out-of-state admissions of its residents. Use the "OUTOFSTATEEXCLUSION" variable in the dataset to capture whether out of-state admissions are included or not (the Data Dictionary and schema provide for formal notation in the dataset on whether these admissions are included). Be certain to use footnotes and metadata to acknowledge the disposition of these admissions.
- Admissions to federal facilities, such as Veteran's Hospitals, are not included. Be certain to inform CDC if you state requires that your dataset includes admissions to federal facilities so that the measures can be appropriately footnoted.
- The optional measures of Monthly Average Number of Hospitalizations and Daily Number of Hospitalizations are only intended for inclusion on state portals and as optional measures. These are not to be submitted to CDC.
- The Data Dictionary should be referred to for the standardized definitions and notations of the variables to be submitted to CDC.

NOTE FOR NON-SAS USERS: Grantees not using SAS should use the steps outlined below for guidance on important issues such as population requirements and should refer to the Data Dictionary for specifications of the required fields.

The data file should be converted to the .XML file format and the required header inserted into the XML file, according to the Schema found on SharePoint. In addition, refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data Dictionary).

NOTE FOR SAS USERS: SAS code is not developed or distributed by CDC.

STEPS FOR CREATING SAS HOSPITALIZATION

<p>Step #1</p>	<p><i>Source of data:</i> Individual level state inpatient hospital admission data based on primary diagnosis at an acute care facility.</p> <p>Please consult your data steward and data mangers to understand the variables and coding system, specifically for race and ethnicity variables. In some states these variables may be coded as one variable whereas in others they are coded as separate variables.</p> <p><i>Years of interest:</i> 2000–present calendar year. The SAS program (not provided by CDC) prepares one NCDM file for each year, as required for CDC submission.</p> <p>Most hospitalization data is released in annual discharge-based datasets. In order to have complete admission date information using discharge-based datasets, it is necessary to have the dataset of the year of interest <u>and</u> the subsequent year. For example, to obtain all admissions during 2005 using discharge based datasets, it will be necessary to have both the 2005 and the 2006 discharge datasets for admissions that occurred in 2005 but were not reported until release of the 2006 discharge dataset. For this example, 2005 data should not be submitted prior to receipt of the 2006 discharge dataset from the data steward.</p> <p><i>Removal of duplicates:</i> This how-to-guide and accompanied SAS code presumes that the user has already removed any duplicate records, while keeping multiple admissions.</p> <p><i>Data Specifications:</i> county FIPS code is 5 digits and that the first two digits will be the same as the state FIPS code. Also, some variables are optional.</p> <p>Select all hospital records that meet the following criteria:</p> <ul style="list-style-type: none"> • Exclude all records where the State of residence is not your state. • Since hospital data is based on discharge year and the EPHT dataset is to be based on admission date, patients discharged in current year (i.e., 2006) but admitted in the previous year (i.e., 2005) should be counted as 2005 hospitalizations. The admission date (and discharge and birth date) variable is acceptable in the following formats: <ul style="list-style-type: none"> ○ SAS DATE FORMAT ○ MMDDYYYY ○ MMDDYY ○ MM/DD/YYYY ○ MM-DD-YYYY ○ DDMONYYYY ○ DDMONYY ○ YYYYMMDD ○ DDMONYYYY:00:00:00 <p><i>SAS Dataset:</i> Make a copy of the hospital admission data before proceeding to next step. If the admission data are not in SAS format, convert it to SAS format and save it as a permanent dataset for creating the XML and measures. Keep the following variables in the SAS dataset:</p>
----------------	---

	<ul style="list-style-type: none"> • Primary diagnosis code • Date of admission • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient's sex • Patient's race (optional) - White, Black, Other, Unknown • Patient's ethnicity (optional) - Hispanic, Non-Hispanic, Unknown • County of residence • State of residence <p><i>Population Data:</i> U.S. Census bureau residential population data for state and county. Intercensal population estimates should be used for the intercensal years, and population extrapolations for postcensal years.</p> <p><i>Go to Step # 2.</i></p>
Step #2	<p>The base format for counts and population data should be by 5-year age groups for AMI beginning 0-4 and ending with 85+. Hospitalization counts must be submitted to CDC by these 5-year age groups (see Data Dictionary). For the calculation of measures and presentation, the age-groups of interest for various asthma and acute myocardial infarction measures are different because of the nature of the diseases. Refer to the measure-specific step for the appropriate age groups for calculation and presentation. In summary, the hospitalization and population age-groups required for the calculation and presentation of measures are:</p> <p>Acute myocardial infarction counts to CDC: 5-yr age groups (0–4, 5–9 ... 85+)</p> <p>Acute myocardial infarction age-specific rate presentation: 0–34, 35–44, 45–54, 55–64, 65–74, 75–84, and 85+</p> <p>Acute myocardial infarction crude and age-adjusted rates: 35+</p> <p>Race and ethnicity variables are optional. Therefore, counts and measures may be generated without specifying race or ethnicity if these data are missing or considered unreliable/inaccurate. If race and ethnicity data are reported, the NCDMs will only be generated when the source variables for race and ethnicity are separate variables. The NCDMs will not be generated if race and ethnicity are reported as a combined race/ethnicity variable. If race and ethnicity data is being provided, be sure that the coding structure conforms with that laid out in the Data Dictionary.</p> <p>Race: White; Black; Other; Unknown.</p> <p>Ethnicity: Hispanic; non-Hispanic; Unknown.</p> <p><i>Go to Step # 3.</i></p>
<p>STEPS FOR GENERATING NCDM REQUIREMENTS</p> <p><i>(Grantees not using SAS should refer to the steps below for conversion of their data file to XML format)</i></p>	

Step #3	<p>After creating the SAS datasets, download IP-NCDM containing the recommended SAS code and Users Guide. These can be found on SharePoint. The current version will only create xml files (not measures).</p> <p>Unzip the files and read and follow the User’s Guide. Click IP-NCDM.msi to launch the setup wizard. Double click the shortcut icon to launch the program.</p> <p>XML files for each single year of data will be generated for one outcome, as required for CDC submission.</p> <p>SAS code to generate measures is not yet operational.</p> <p><i>NOTE:</i> Single year files for individual outcomes must be submitted, as specified in the data call letter</p> <p>Steps #4 through #12 should be used to calculate the NCDMs to ensure consistency between grantees.</p>
STEPS FOR COMPLETING SPECIFIC MEASURES	
Step #4	<p>SAS menu options 3–9 generate measures beyond those required for the national portal but are currently non-operational. These options will follow the steps below (4b through 12). These steps may also be used to generate measures outside of SAS. If selecting menu options 3–9, this SAS program will generate a final output in the form of MS Excel spreadsheet. A PDF file containing histograms will also be created.</p> <p>Flag all admissions where primary diagnosis code is “410” by creating a variable (<i>for example Ishospital</i>) that takes the value of 1, if admission is due to diagnosis code “493”; else its value is 2.</p> <p>Keep only those records that meet the following criteria:</p> <ul style="list-style-type: none"> - Principal diagnosis code = 410 - State = Your state - Date of Birth (if available and being used to calculate patient age) is not missing - Patient’s age at the time of hospital admission is not missing - Date of Discharge is not missing - Date of Admission is not missing. <p>Go to Step # 5.</p>
ANNUAL NUMBER OF HOSPITAL ADMISSIONS	
Step #5	Annual Number of Hospital Admissions by sex and total
Step #5a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by sex (male, female and unknown (including missing)).</p> <p>Sum the number of male, female and unknown sex hospital admissions to get the total number of hospital admissions for the year.</p>
Step #5b	<i>County:</i> Sum the total number of admissions by county of residence for year of interest

	<p>by sex [male, female and unknown (including missing)].</p> <p>Sum the number of male, female and unknown sex hospital admissions to get the total number of hospital admission by county for the year.</p>
Step #6	Annual Number of Hospital Admissions by race and total
Step #6a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by race categories.</p> <p>Sum the number of hospital admissions to get the total number of AMI hospital admission for the year.</p>
Step #6b	<p><i>County:</i> Sum the total number of admissions for by county of residence for year of interest by race categories.</p> <p>Sum the number of hospital admissions to get the total number of hospital admission by county and race for the year.</p>
Step #7	Annual Number of Hospital Admissions by age groups and total
Step #7a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by the specific 5-year age groups created in step #2.</p> <p>Sum the number of hospital admissions for each age group to get the total number of hospital admission for the year.</p>
Step #7b	<p><i>County:</i> Sum the total number of admissions for year of interest for each county by the specific 5-year age groups created in step #2.</p> <p>Sum the number of hospital admissions for each age group to get the total number of hospital admission for the year.</p>
DAILY NUMBER OF HOSPITAL ADMISSIONS (NOT A REQUIRED NCDM)	
Step #8	<p>NOTE: Daily number of admissions is not a required NCDM and is not submitted to CDC or required to be placed on state portals. Because of the potential future use of this measure. It remains in the How-to-Guide and SAS code.</p> <p>Sum the total number of admission for each day by sex during the year of interest for entire state to get the daily number of admissions by sex. Add the daily number of male, female and unknown (including missing sex information) to obtain the total number of daily admissions. Repeat the above by race and 5-year age-groups to calculate the daily number of hospital admissions by race and age groups.</p>
ANNUAL AGE-SPECIFIC HOSPITAL ADMISSIONS RATE	
Step #9	Annual age-specific hospital admission rate by sex
Step #9a	<p><i>Create the numerator data:</i> Sum the number of hospitalizations in the state for the year of interest in each of the 5-year age-groups for both male and female. Exclude any observation where sex is unknown or missing. Sum the admissions for male and female to obtain the total admissions for each age-group.</p>
Step #9b	<p><i>Create the denominator data:</i> Sum the population in the state or county for year of interest in each 5-year age group for both male and female. Sum male and female</p>

	population to obtain the total state population for each age-group.
Step #9c	Merge both numerator and denominator data by state of residence, year of hospital admissions, age-group and sex.
Step #9d	<i>For acute myocardial infarction presentation:</i> Compute age-specific rates for male, female and total for age-groups 0–34, 35–44, 45–54, 55–64, 65–74, 75–84, and 85+ by dividing the number of hospital admissions in that age group and sex by the population of same age-group and sex.
Step #9e	All rates are to be presented as per 10,000 population. Multiply the rates calculated in step 9d by 10,000 to obtain rate of admission per 10,000 population.
Step #9f	Upper and lower confidence limits (95% confidence interval) for age-specific rates may be computed. For each age-specific rate computed in step 9d, compute Lower Confidence Limit (LCL) and Upper Confidence Limit (UCL) as follows LCL = [age-specific rate – { 1.96 × age-specific rate/SQRT (Number of admissions)}] UCL = [age-specific rate + { 1.96 × age-specific rate/SQRT (Number of admissions)}]
ANNUAL UNADJUSTED (CRUDE) RATE OF HOSPITAL ADMISSIONS	
Step #10a	Exclude any observation where sex is unknown or missing. <i>Acute myocardial infarction:</i> Create the numerator data (referred to in Step #2) to obtain the annual number of hospital admissions by sex for both male and female for ages 35+ only. Sum the male and female number of admissions to obtain total admissions for both sexes.
Step #10b	<i>Acute myocardial infarction:</i> Create the denominator data as referred to in step #2 for ages 35+ only. Sum the population for male, female and both sex for the year of interest.
Step #10c	Merge both numerator and denominator data by state of residence, year of hospital admissions and sex.
Step #10d	Compute the annual unadjusted rate of hospital admissions as follows: Unadjusted Admission Rate (Male) = (# of Male Admissions/ Male Population) Unadjusted Admission Rate (Female) = (# of Female Admissions/ Female Population) Unadjusted Admission Rate (Total) = (Total Admissions/ State Population) Multiply the above computed rates by 10,000 to obtain the number of admissions per 10,000 population.
ANNUAL AGE ADJUSTED RATE OF HOSPITAL ADMISSIONS	
Step #11	Annual Age Adjusted Rate of Hospital Admissions for State by Sex
Step #11a	<i>Acute myocardial infarction:</i> Calculate the age specific rates as described in steps 10a through 10d using 5 yr age groups for 35+ only (35–39, 40–44 ... 85+) for male, female and both sexes.

Step #11b	To calculate age-specific rates (for the 5-year age categories 0–34, 35–39...85+); use U.S. Census bureau residential population data for denominators. Intercensal population estimates should be used for the intercensal years. The standard population should be the 2000 U.S. Standard Population divided into 18 age groups. The link for the 2000 U.S. Standard Population is: http://seer.cancer.gov/stdpopulations/).
Step #11c	Merge both numerator and denominator data by age group and sex.
Step #11d	<p>Compute the age-adjusted population weights using the 2000 US population as the standard.</p> <p><i>Acute myocardial infarction:</i> Because age-adjustment is based on the 35+ population only, the age-adjusted weights must be normalized. The total weight for ages 35+ must now be 1.0.</p> <p>Refer to the following article for the method to normalize: http://www.cdc.gov/nchs/data/statnt/statnt20.pdf</p>
Step #11e	<p><i>Compute the age-adjusted hospital admissions rate:</i></p> <p>Age-adjusted rate = Sum of age-specific rate × age adjusted weight</p> <p>For tutorial on age-adjustment see: http://seer.cancer.gov/seerstat/tutorials/aarates/definition.html</p>
Step #11f	<p>95% confidence intervals may be computed.</p> <p>LCL = [age-adjusted rate – { 1.96 × age-adjusted rate/SQRT (Number of Admissions)}]</p> <p>UCL = [age-adjusted rate + { 1.96 × age-adjusted rate/SQRT (Number of Admissions)}]</p> <p>NOTE: With small numbers of hospitalizations (e.g., <20), calculation methods assuming a non-Normal distribution may be more appropriate.</p>
Step #11f	To calculate the Annual age-adjusted rate of hospital admissions by County, follow steps 11a through 11f using the same 2000 US standard population.
PRESENTATION	
Step #12a	Export SAS datasets to Microsoft Excel® for creating charts or for formatting the computed values for presentation
Step #12b	<p>Annual number of hospital admissions can be displayed by showing sex on x-axis and number of admissions on y-axis. Similarly sex on x-axis can be replaced by race or age groups to display the number of admissions by race or age-group.</p> <p>These bar charts can be created by using any spreadsheet application or by using SAS.</p>
Additional visual display	<p>Public can view histograms and map showing the state and county level AMI measures. These visual displays will show whether the selected geographical area has a high, medium high, medium low or low hospital admission rate. The public will also be able to see the links to other related information from various national, state and local sources.</p> <p>Mapping the rate of hospital admissions per 10,000 residents will allow users to assess</p>

	<p>the level of environmentally related risk factors in their residential geographic area as well as in the surrounding geographical area. Future mapping may include other risk factors such as poverty and other potential risk factors. One limitation of mapping is that not all sources of myocardial infarction risk factors may be mapped. For example, indoor mold, dust, and pollen.</p>
<p>Interpretation</p>	<p>Small Numbers: Measures that generate counts or rates based upon numbers that are small and potentially violate state privacy guidelines can collapse data across years or geographic units to generate data that can be released.</p> <p>Measures for multiple years: The how-to-guide steps can be repeated for additional years of hospital admission data. Multi-year hospital admission data can be merged to create one dataset. Add the number of hospital admissions for each year in multi-year cohort and divide by the number of years to calculate an average annual number of hospital admissions.</p>

Indicator Template
Content Area: Heart Attack
Indicator: Hospitalizations for Heart Attack

Environmental Public Health Tracking

Type of EPHT Indicator	Health outcome
Measures	<ol style="list-style-type: none"> 1. Age-adjusted rate of hospitalization for heart attack among persons 35 and over per 10,000 population 2. Crude rate of hospitalization for heart attack among persons 35 and over per 10,000 population 3. Number of hospitalizations for heart attack
Derivation of Measure(s)	<p><i>Numerator:</i> Resident hospitalizations for Heart Attacks or Acute Myocardial Infarction (AMI), ICD-9-CM: 410.00 – 410.92 by gender and total for state and by county</p> <p><i>Denominator:</i> Midyear resident population, by gender, for state and by county</p> <p><i>Adjustment:</i> Age-adjustment by the direct method to Year 2000 US Standard population</p>
Unit	<ol style="list-style-type: none"> 1. Age-adjusted rate per 10,000 population 2. Rate per 10,000 population 3. Number
Geographic Scope	State and national
Geographic Scale	Residents of jurisdiction – State, County
Time Period	Hospital admissions between January 1 to December 31, inclusive, for each year, 2000–
Time Scale	Annual
Rationale	<p>There currently is no single Heart Attack, also known as Acute Myocardial Infarction (AMI), surveillance system in place in the US, nor does this exist for coronary heart disease (CHD) in general. Mortality is the sole descriptor for national data for AMI. Estimates of incidence and prevalence of AMI and CHD are largely based on survey samples (e.g., National Health and Nutrition Examination Survey) or large cohort studies such as the Atherosclerosis Risk in Communities (ARIC) study.</p> <p>In 2007 the American Heart Association estimated 565,000 new attacks and 300,000 recurrent attacks of acute myocardial infarction annually (National Heart, Lung, and Blood Institute: based on unpublished data from the ARIC study and the Cardiovascular Health Study (CHS)). Among Americans age 20 and older, new and recurrent MI prevalence for both men and women represented 3.7% of the US population or 7,900,000 individuals (4.9 million men and 3.0 million women). Corresponding prevalence by race and gender is 5.4% for white males, 2.5% for white females, 3.9% for black males and 3.3% for black females.</p> <p>The well documented risk factors for AMI include diabetes, hypertension, obesity, hypercholesterolemia, and cigarette smoking. Increasingly investigators both in the</p>

	<p>U.S. and abroad have shown significant relationships between air pollutants and increased risk of AMI and other forms of CHD. Studies have often focused on elderly individuals (>65 years). A number of epidemiologic studies have reported associations between air pollution (ozone, PM₁₀, CO, PM_{2.5}, SO₂) and hospitalizations for AMI and other forms of heart disease. Models have demonstrated increases in AMI hospitalization rate in relation to fine particles (PM_{2.5}) particularly in sensitive subpopulations such as the elderly, patients with pre-existing heart disease, especially those who are survivors of AMI or those with COPD. An increase of 10 ug/m³ in PM_{2.5} levels was associated with a 4.5% elevation in risk of acute ischemic coronary events (unstable angina and AMI) (95% CI, 1.1–8.0). Mortality statistics have been linked for a 16 year period to chronic exposure to multiple air pollutants in 500,000 adults who resided in all 50 states. Each 10 ug/m³ in annual PM_{2.5} was related to a 12% increased mortality risk.</p>
<p>Use of the Measure</p>	<p>The development of a standardized measures method for AMI hospital admissions among residents in each state will inform multiple users at the national, state, and local levels. These measures, and associated indicators, will allow for monitoring of trends over time and have the potential to identify high risk groups not reflected in current national data. These data may also inform prevention, evaluation and program planning efforts.</p> <p>These measures will address the following surveillance functions:</p> <ul style="list-style-type: none"> • Examination of time trends in AMI hospitalizations. • Identification of seasonal trends. • Assessment of geographic differences in hospitalizations. • Evaluation of differences in AMI hospitalizations by age, gender, and race/ethnicity. • Determination of populations in need of targeted interventions • Identification of possible environmental relationships warranting further investigation or environmental public health action, when AMI data are linked with environmental variables,
<p>Limitations of the Measure</p>	<ul style="list-style-type: none"> • Hospitalization data for Heart Attacks omits individuals who do not receive medical care or who are not hospitalized, including those who die in emergency rooms, in nursing homes, or at home without being admitted to a hospital, and those treated in outpatient settings. • Differences in rates by time or area may reflect differences or changes in diagnostic techniques and criteria and in the coding of AMI or in medical care access. • Differences in rates by area may be due to different socio-demographic characteristics and associated behaviors. • When comparing rates across geographic areas, a variety of non-environmental factors, such as access to medical care and diet, can impact the likelihood of persons hospitalized for AMI. • Reporting rates at the state and/or county level will not show the true AMI burden at a more local level (i.e. neighborhood).

	<ul style="list-style-type: none"> • Reporting rates at the state and/or county level will not be geographically resolved enough to be linked with many types of environmental data. • When looking at small geographic levels (e.g. zip code), users must take into consideration appropriate cell suppression rules imposed by the data providers or individual state programs. • Although duplicate records and transfers from one hospital to another are excluded, the measures are based upon events, not individuals, because no unique identifier is always available. When multiple admissions are not identified, the true prevalence will be overestimated. • Even at the county level it can be expected that the measures generated will often be based upon numbers too small to report or present without violating state and federal privacy guidelines and regulations. Careful adherence to cell suppression rules in cross tabulations is necessary and methods to increase cell sizes by combining data across time (e.g., months, years) and geographic areas may be appropriate.
Data Sources	<p><i>Numerator:</i> State inpatient hospitalization data (using admission date) <i>Denominator:</i> US Census Bureau population data</p>
Limitations of Data Sources	<p><i>State hospital discharge data:</i></p> <ul style="list-style-type: none"> • Using a measure of all AMI hospitalizations will include some transfers between hospitals for the same individual for the same AMI event. Variations in the percentage of transfers or readmissions for the same AMI event may vary by geographic area and impact rates. • Without reciprocal reporting agreements with abutting states, statewide measures and measures for geographic areas (e.g., counties) bordering other states may be underestimated because of health care utilization patterns. • Each state must individually obtain permission to access and, in some states, provide payment to obtain the data. • Veterans Affairs, Indian Health Services and institutionalized (prison) populations are not usually included in hospitalization datasets. • Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers to hospitalize patients • Street address is currently not available in many states. • Sometimes mailing address of patient is listed as the residence address of the patient • Patients may be exposed to environmental triggers in multiple locations, but hospital discharge geographic information is limited to residence. • Since the data captures hospital discharges (rather than admissions), patients admitted toward the end of the year and discharged the following year will be omitted from the current year dataset • Data will need to be de-duplicated (i.e., remove duplicate records for the same event) • There is usually a two year lag period before data are available from the data owner. <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census

	Bureau. These need to be extrapolated or purchased from a vendor.
Related Indicators	<ul style="list-style-type: none"> • Annual average ambient concentration of PM_{2.5} in microgram per cubic meter • Number of days with maximum 8-hour average ozone concentration over the National Ambient Air Quality standard • Number of person-days with maximum 8-hour average ozone concentration over the National Ambient Air Quality standard • Number of person-days with PM_{2.5} levels over the National Ambient Air Quality standard • Percent days with PM_{2.5} levels over the National Ambient Air Quality standard
References	<ol style="list-style-type: none"> 1. Rosamond, W., et al., Heart disease and stroke statistics--2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. <i>Circulation</i>, 2007. 115(5): p. e69–171. 2. Boland, L.L., et al., Occurrence of unrecognized myocardial infarction in subjects aged 45 to 65 years (the ARIC study). <i>Am J Cardiol</i>, 2002. 90(9): p. 927–31. 3. Thom, T., et al., Cardiovascular disease in the United States and preventive approaches, in <i>Hurst's The Heart, Arteries and Veins</i>, V. Fuster, R. Alexander, and R. O'Rourke, Editors. 2001, McGraw-Hill: New York, NY. 4. Jones, D.W., et al., Risk factors for coronary heart disease in African Americans: the atherosclerosis risk in communities study, 1987–1997. <i>Arch Intern Med</i>, 2002. 162(22): p. 2565–71. 5. Kannel, W.B., et al., Menopause and risk of cardiovascular disease: the Framingham study. <i>Ann Intern Med</i>, 1976. 85(4): p. 447–52. 6. Pope, C.A., 3rd, et al., Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. <i>Circulation</i>, 2004. 109(1): p. 71–7. 7. Vermeylen, J., et al., Ambient air pollution and acute myocardial infarction. <i>J Thromb Haemost</i>, 2005. 3(9): p. 1955–61. 8. Pope, C.A., 3rd, et al., Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. <i>Circulation</i>, 2006. 114(23): p. 2443–8. 9. von Klot, S., et al., Ambient air pollution is associated with increased risk of hospital cardiac readmissions of myocardial infarction survivors in five European cities. <i>Circulation</i>, 2005. 112(20): p. 3073–9.

INDICATOR TEMPLATE
CONTENT AREA: CLIMATE AND HEALTH
INDICATOR: EMERGENCY DEPARTMENT VISITS FOR HEAT STRESS

Environmental Public Health Tracking

Type of EPHT Indicator	Health outcome
Measures	<ol style="list-style-type: none"> 1. Annual age-adjusted rate of emergency department visits for heat stress per 100,000 population 2. Annual crude rate of emergency department visits for heat stress per 100,000 population 3. Annual number of emergency department visits for heat stress
Derivation of Measure(s)	<p><i>Numerator:</i></p> <ul style="list-style-type: none"> • Patients treated in an Emergency Department (ED) having any ICD-9 code in the range of 992.0-992.9, or cause of injury code E900.0 or E900.9. • Cases with a code of E900.1 (man-made source or heat) anywhere in the record are <u>excluded</u>. <p><i>Denominator:</i> Midyear resident population, by gender, for state and by county</p> <p><i>Adjustment:</i></p> <ul style="list-style-type: none"> • Age-adjustment by the direct method to the Year 2000 US Standard population • U.S. 2000 standard population by age categories from Surveillance Epidemiology and End Results (SEER), National Cancer Institute
Unit	<ol style="list-style-type: none"> 1. Age-adjusted rate per 100,000 population 2. Rate per 100,000 population 3. Number
Geographic Scope	EPHT grantee states with hospitalization data
Geographic Scale	State
Time Period	Ed visits between May 1 to September 30, inclusive, for each year, 2000–
Time Scale	May–September of each data year
Rationale	The Intergovernmental Panel on Climate Change (IPCC) projects with “virtual certainty” suggest that climate change will cause more frequent, more intense, and longer heat waves (1). Any individual, regardless of age, sex or health status can develop heat stress if engaged in intense physical activity and/or exposed to environmental heat (and humidity). Physiologic mechanisms maintain the core body temperature (i.e., the operating temperature of vital organs in the head or trunk) in a narrow optimum range around 37 °C (98.6 °F). When core body temperature rises, the physiologic response is to sweat and circulate blood closer to the skin's surface to increase cooling. If heat

exposure exceeds the physiologic capacity to cool, and core body temperature rises, then a range of heat-related symptoms and conditions can develop. Heat stress or Heat-related illness ranges from mild heat edema, rash, heat syncope, heat cramps, to the most common type, heat exhaustion (2). Heat-related cramps, rash, and edema are relatively minor readily treatable conditions; however, they should be used as important warning signs to immediately remove the affected individual from the exposure situation.

Heat cramps are brief, intermittent, and often severe muscular cramps occurring typically in muscles that are fatigued by heavy work (2). Individuals with heat cramp can also exhibit hyponatremia, hypochloremia, and low serum sodium and chloride levels.

Heat syncope is a temporary loss of consciousness as a result of prolonged heat exposure (2). Individuals adapt to hot, humid environment by dilation of cutaneous vessels in the skin to radiate heat. Peripheral vasodilation along with blood volume loss, results in lowering the blood pressure which can result in inadequate central venous return and cerebral perfusion, causing light-headedness and fainting.

Heat exhaustion is a consequence of extreme depletion of blood plasma volume, which may be coincident with hyponatremia and/or peripheral blood pooling (2). Heat exhaustion often does not present with definitive symptoms and may be misdiagnosed, often as an acute viral illness. Symptoms include mild disorientation, generalized malaise, weakness, nausea, vomiting, headache, tachycardia (rapid beating of the heart), and hypotension. Because untreated heat exhaustion can progress to heat stroke, the most serious form of heat-related illness, treatment should begin at the first signs of heat exhaustion (3).

Heat stroke is an extreme medical emergency that if untreated can result in death or permanent neurological impairment (2). Heat stroke occurs when a person's core body temperature rises above 40 °C (104 °F) as a result of impaired thermoregulation. High core body temperature and disseminated intravascular coagulation results in cell damage in vital organs, such as the brain, liver, and kidneys, which can lead to serious illness and death (3). Death may occur rapidly due to cardiac failure or hypoxia, or it can occur days later as a result of renal failure due to dehydration and/or rhabdomyolysis (i.e., the breakdown of muscle fibers with release into the circulation of muscle fiber contents, some of which are toxic to the kidney and can cause kidney damage) (4). Heat stroke is typically divided into two types. The two types are in general clinically the same, except that the individuals/population groups affected require medical interventions specific to their unique physiology and medical status (3). "Exertional Heat Stroke," as the name implies, involves strenuous physical activity under high temperature conditions to which the heat stroke victim was not acclimatized, and usually affects healthy young adults, such as athletes, outdoor laborers and soldiers. "Classic" heat stroke, by definition does not involve exertion, and usually affects susceptible individuals, such as infants and young children, the elderly, or people with chronic illness. Because heat stroke, even if treated, can have a death rate as high as 33%, and up to 17% of heat stroke survivors suffer permanent be taken to prevent heat-

	<p>related illness, especially among vulnerable populations.</p> <p>The relationship between extreme heat and increased daily morbidity and mortality is well established. This indicator captures hospital admissions <i>directly</i> attributed to heat stress (e.g., heat illness, heat stroke, and hyperthermia). It is a measure that can be tracked easily and consistently across geography and time, and acts as a sentinel for the broader range of heat-related illness that is not recognized and/or coded as such.</p>
Use of the Measure	<p>Heat stress can manifest in a number of clinical outcomes, and people with chronic health problems (e.g., cardiovascular disease, diabetes, obesity) are more susceptible to the effects of heat than healthy individuals. For these reasons, heat stress may not be listed as the primary diagnosis. This indicator therefore includes all cases where heat stress is explicitly listed as the primary diagnosis or any other diagnosis.</p> <p>Increases in the rates of ED visits for heat stress are one potential impact of rising global temperatures. Tracking these data can help document changes over place and time, monitor vulnerable areas, and evaluate the results of local climate-adaptation strategies.</p>
Limitations of the Measure	<p>Periods of extreme heat are frequently associated with increases in hospital visits and admissions for many causes. This measure does not capture the full spectrum of heat-stress, where exposure to excess heat is not explicitly documented.</p>
Data Sources	<p><i>Numerator:</i> State emergency department data <i>Denominator:</i> US Census Bureau population data</p>
Limitations of Data Sources	<p><i>Emergency Department data:</i></p> <ul style="list-style-type: none"> • Data are not available for all states. • Number of diagnostic fields in hospital records varies from state to state. Utilization of EDs varies geographically. <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year.
Related Indicators	<ul style="list-style-type: none"> • Heat vulnerability • Heat-related mortality • Temperature distribution • Heat stress hospitalizations
References	<ol style="list-style-type: none"> 1. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. 2007. Human health In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE. , editors. Climate Change 2007: Impacts, Adaptation and Vulnerability Contribution of Working Group II to: Fourth Assessment Report of the Intergovernmental Panel on Climate Change. New York: Cambridge University Press. pp. 391–431. 2. Rosen’s Emergency Medicine: Concepts and Clinical Practice. 2010. Chapter 139: Heat illness. In JA Marx Editor-in-Chief; RS Hockberger & RM Walls Senior Editors; JG Adams ... [et al] Editors (7th ed). Philadelphia: Mosby Elsevier.

3. American Medical Association. Heat-related Illness During Extreme Weather Emergencies (Report 10 of the Council on Scientific Affairs (A97), 1997; www.ama-assn.org/ama/pub/category/13637.html).

4. Centers for Disease Control and Prevention. Heat-related deaths--Los Angeles County, California, 1999-2000, and United States, 1979-1998. MMWR 2001; 50(29):623-6.

HOW-TO GUIDE

Heat Stress Emergency Department (ED) Visits

Environmental Public Health Tracking

07-02-2013

Data Source	Emergency Department (ED) Visits
NCDM Requirements	<ul style="list-style-type: none"> • Health Outcome = Heat Stress • State/County of Residence • ED Visit Year/Month • Age Group • Sex • Race/Ethnicity (optional) • Transfers not to be excluded • Out-state residents to be excluded • ED visits to federal facilities to be excluded • ED visits of residents to out-of-state hospitals are to be optionally included
Measures Generated	<ul style="list-style-type: none"> • Annual* Number of ED Visits by age group, sex, race/ethnicity**, and county and state • Annual Crude (unadjusted) Rate of ED Visits by age group, sex, race/ethnicity**, and county and state • Annual Age-Adjusted Rate of ED Visits for all ages by sex, race/ethnicity*, and county and state <p>*For Heat stress illness ANNUAL numbers and rates include ED visits between <u>May 1 to September 30</u> of reporting year</p> <p>**measures by race/ethnicity are optional</p>
Definitions	<p><i>Duplicate record:</i> More than one record for the same person with the same ED Visit data (e.g., sex, date of birth, admission/ED Visit date, and zip code have exact same information). Duplicate records may also be due to continuation of data beyond a single line. In this case, duplicates may be identified using a record sequence number.</p> <p><i>ED Visit date:</i> The calendar date of the ED Visit:</p> <ul style="list-style-type: none"> ▪ Day (optional) ▪ Month (required) ▪ Year (required) <p><i>ED Visit Year:</i> An ED Visit for Heat Stress during a specific calendar year. ED Visit year is based only upon the calendar year of the Visit, even when discharge and/or release year is different.</p> <p><i>Emergency Department Visit:</i> Treatment in a hospital emergency department. This</p>

	<p>should include both patients who are treated and released and those that are admitted as inpatients from the emergency department.</p> <p><i>Event/Event Year:</i> A hospital admission for Heat Stress results with a primary diagnosis of 992.0-992.9 or cause of injury code E900.0 or E900.9 (cases with a code E900.1 are excluded) during May 1 to September 30 of a specific calendar year. Event year is based only upon admission year, even when discharge year is different.</p> <p><i>Heat Stress:</i> Heat stress is defined as a constellation of explicit effects of hot weather on the body including heat stroke and sunstroke (hyperthermia), heat syncope/collapse, heat exhaustion, heat cramps, heat fatigue, heat edema, and other/unspecified clinical effects attributed to excessive heat exposure. For Heat Stress illness measures only include cases that occurred during May 1 to September 30 of each calendar year.</p> <p><i>Hospital Transfers:</i> The practice of discharging a patient from one facility and readmitting them to a second facility within 48 hours.</p> <p><i>ICD-9-CM code:</i> International Classification of Diseases, 9th Revision, Clinical Modification</p> <p><i>Multiple visits:</i> More than one ED Visit for the same person for the same diagnosis code occurring on different dates and related to a separate event within a given year. Multiple ED Visits are considered separate events if they occurred more than 48 hours apart.</p> <p><i>Observation Stay:</i> This is an alternative to inpatient admission that exists in some facilities but for EPHT is considered in ED Visit statistics. Observation Stays may originate as an ED Visit or directly as an Observation Stay. Note that the definition of an Observation Stay may not be standard across hospitals, and Observation Stays may not be recorded across states in a consistent manner.</p> <p><i>Primary Diagnosis Code:</i> Presently, diagnosis codes are represented by ICD-9-CM codes (the International Classification of Diseases, 9th Revision, Clinical Modification). Heat stress is classified as any primary or other diagnosis code in the range of 992.0-992.9, or cause of injury code E900.0 or E900.9. Cases with a code of E900.1 (man-made source or heat) anywhere in the record are <u>excluded</u>.</p> <p><i>Resident:</i> A person who resides in the grantee's state/county (permanently or for an extended period) at the time of the ED Visit.</p>
<p>How-to-Guide Requirements and Cautions</p>	<ul style="list-style-type: none"> • This How-to-Guide has two purposes: 1) Guide the user in the development of the XML dataset using the SAS code, located on SharePoint, for submission to CDC; 2) Describe the calculation steps for creating the Nationally Consistent Data Measures (NCDMs). Grantees should use the How-to-Guide in order to ensure that the calculation of measures are consistent with those required by CDC in the preparation of their own computer code. • Note that one set of SAS code (not provided by CDC's EPHT program) is designed to handle the creation of XML files for all hospitalization (inpatient) outcomes (separate code is available for all ED Visit outcomes). The code manages the differences in definitions between the outcomes and generates a

separate file for each outcome by each single year.

- This How-to-Guide and the optional SAS code not provided by CDC’s EPHT program) presume that the user has removed duplicate records while keeping multiple ED Visits. A case should be counted once per ED Visit; de-duplication of records to achieve this goal should be conducted at the discretion of the data owners, managers, and/or analysts.
- ED Visits include both patients who are admitted to the hospital through the emergency department (inpatients) and those who are treated and released (outpatients); therefore, both inpatient and outpatient data are required for this indicator. If identified and/or stored separately, observation stay data should be included as well.
- In the event that an ED Visit occurred at the end of a calendar year and the discharge date occurred in the following year, the dataset that includes the discharge date will be required before the dataset can be considered complete.
- The How-to-Guide steps do not incorporate data suppression and/or aggregation rules. Suppression guidelines are separately applied by CDC for the national portal and by grantees for state portals.
- Admissions of residents to out-of-state hospitals are not required to be included. However, when available these admissions should be included. It is noted that some states must include out-of-state admissions of its residents. Use the “OUTOFSTATEEXCLUSION” variable in the dataset to capture whether out-of-state admissions are included or not (the Data Dictionary and schema provide for formal notation in the dataset on whether these admissions are included). Be certain to use footnotes and metadata to acknowledge the disposition of these admissions.
- ED Visits for individuals who are not state residents should be excluded. If a data steward’s database includes these cases, exclude them from the EPHT database. If they cannot be excluded, footnotes and metadata should acknowledge that these cases are included.
- Patients transferred from or to other acute care facilities should be included. Indicate in footnotes and/or metadata if transfers are excluded.
- Patients with an ED Visit at a federal facility should not be included. If a data steward’s database includes these cases, exclude them from the EPHT database. If they cannot be excluded, footnotes and metadata should acknowledge that these cases are included.
- Although hospital discharge data are collected using a standard format across states, there are considerable differences in the variable attributes; for example, response categories may differ between states for “source of admission” and “disposition” variables. These differences may reflect how certain variables are collected, whether the reporting of a variable (for example patient name or race) is mandatory, and/or differences in data availability and access agreements. The number of diagnosis fields available in the discharge data also varies by state,

	<p>ranging from nine to an unlimited number. In addition, the data vary by state in regard to data quality such as the validity or completeness of specific fields. In all cases, the data analyst should work closely with the data managers in order to understand the nuances of the data.</p> <ul style="list-style-type: none"> The Data Dictionary in SharePoint should be referred to for the standardized definitions and notations of the variables to be submitted to CDC. 																										
<p>NOTE FOR NON-SAS USERS: Grantees not using SAS should use the steps outlined below for guidance on important issues such as population requirements and should refer to the Data Dictionary for specifications of the required fields. The data file should be converted to the .XML file format and the required header inserted into the XML file, according to the Schema found on SharePoint. In addition, refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data Dictionary).</p> <p>NOTE FOR SAS USERS: SAS code is not developed or distributed by CDC.</p>																											
<p>Section A: CREATION OF REQUIRED DATA FILE FOR NCDMs</p>																											
<p>Step #1</p>	<p><i>Identifying the data sources for ED Visits:</i></p> <p>ED Visits include both patients who are treated and released in the ED (outpatients) and who are admitted as inpatients through the emergency department; therefore, <u>both</u> inpatient and outpatient data files are required for this indicator. If identified separately, observation stay data files are also required.</p>																										
<p>Step #2</p>	<p><i>Identifying ED Visits for Heat Stress</i></p> <p>a. Select cases having any of the following ICD-9 codes as a principal diagnosis, injury cause, or other diagnoses:</p> <table border="0" data-bbox="516 1150 1161 1680"> <thead> <tr> <th style="text-align: left;">Code</th> <th style="text-align: left;">Description</th> </tr> </thead> <tbody> <tr><td>992.0</td><td>Heat stroke and sunstroke</td></tr> <tr><td>992.1</td><td>Heat syncope</td></tr> <tr><td>992.2</td><td>Heat cramps</td></tr> <tr><td>992.3</td><td>Heat exhaustion from water depletion</td></tr> <tr><td>992.4</td><td>Heat exhaustion from salt depletion</td></tr> <tr><td>992.5</td><td>Heat exhaustion, unspecified</td></tr> <tr><td>992.6</td><td>Heat fatigue, transient</td></tr> <tr><td>992.7</td><td>Heat edema</td></tr> <tr><td>992.8</td><td>Other specified heat effects</td></tr> <tr><td>992.9</td><td>Unspecified effects of heat and light</td></tr> <tr><td>E900.0</td><td>Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)</td></tr> <tr><td>E900.9</td><td>Effect from unknown cause of excessive heat</td></tr> </tbody> </table> <ul style="list-style-type: none"> Remove any records having ICD-9 code E900.1 (man-made source of heat) as a cause of injury or other diagnosis. If data are not already limited to treatment/admission dates in the months of May through September, exclude cases outside this range. 	Code	Description	992.0	Heat stroke and sunstroke	992.1	Heat syncope	992.2	Heat cramps	992.3	Heat exhaustion from water depletion	992.4	Heat exhaustion from salt depletion	992.5	Heat exhaustion, unspecified	992.6	Heat fatigue, transient	992.7	Heat edema	992.8	Other specified heat effects	992.9	Unspecified effects of heat and light	E900.0	Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)	E900.9	Effect from unknown cause of excessive heat
Code	Description																										
992.0	Heat stroke and sunstroke																										
992.1	Heat syncope																										
992.2	Heat cramps																										
992.3	Heat exhaustion from water depletion																										
992.4	Heat exhaustion from salt depletion																										
992.5	Heat exhaustion, unspecified																										
992.6	Heat fatigue, transient																										
992.7	Heat edema																										
992.8	Other specified heat effects																										
992.9	Unspecified effects of heat and light																										
E900.0	Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)																										
E900.9	Effect from unknown cause of excessive heat																										

- Exclude all records where the State of residence is not your state.

b. From inpatient hospitalization data, select cases having any of the following ICD-9 codes as a principal diagnosis, injury cause, or other diagnoses:

Code	Description
992.0	Heat stroke and sunstroke
992.1	Heat syncope
992.2	Heat cramps
992.3	Heat exhaustion from water depletion
992.4	Heat exhaustion from salt depletion
992.5	Heat exhaustion, unspecified
992.6	Heat fatigue, transient
992.7	Heat edema
992.8	Other specified heat effects
992.9	Unspecified effects of heat and light
E900.0	Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)
E900.9	Effect from unknown cause of excessive heat

- *Remove* any records having ICD-9 code E900.1 (man-made source of heat) as a cause of injury or other diagnosis.
- If data are not already limited to treatment/admission dates in the **months of May through September**, exclude cases outside this range.
- Restrict the dataset to patients who were admitted from an ED using the following criteria:
 - point of origin code indicates emergency department, or
 - CPT codes: 99281–99285, or
 - revenue codes: 0450–0459, or
 - positive ED charges
- These criteria are consistent with the criteria used by AHRQ (see: http://www.hcup-us.ahrq.gov/db/vars/siddistnote.jsp?var=hcup_ed).
- Exclude all records where the State of residence is not your state.

c. From Observation Stay data

In states where observation stays are identified separately, include these observation stay records with ED Visits. Not all states require the reporting of observation stay records. Contact data stewards to determine whether records for observation stays are collected and if so, if the records are located with outpatient or inpatient records, or in a separate file. Observation Stays can be identified by selecting all the records that meet the following criteria:

Select cases having any of the following ICD-9 codes as a principal diagnosis, injury cause, or other diagnoses:

	<table border="0"> <thead> <tr> <th style="text-align: left;">Code</th> <th style="text-align: left;">Description</th> </tr> </thead> <tbody> <tr><td>992.0</td><td>Heat stroke and sunstroke</td></tr> <tr><td>992.1</td><td>Heat syncope</td></tr> <tr><td>992.2</td><td>Heat cramps</td></tr> <tr><td>992.3</td><td>Heat exhaustion from water depletion</td></tr> <tr><td>992.4</td><td>Heat exhaustion from salt depletion</td></tr> <tr><td>992.5</td><td>Heat exhaustion, unspecified</td></tr> <tr><td>992.6</td><td>Heat fatigue, transient</td></tr> <tr><td>992.7</td><td>Heat edema</td></tr> <tr><td>992.8</td><td>Other specified heat effects</td></tr> <tr><td>992.9</td><td>Unspecified effects of heat and light</td></tr> <tr><td>E900.0</td><td>Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)</td></tr> <tr><td>E900.9</td><td>Effect from unknown cause of excessive heat</td></tr> </tbody> </table> <ul style="list-style-type: none"> • <i>Remove</i> any records having ICD-9 code E900.1 (man-made source of heat) as a cause of injury or other diagnosis. • If data are not already limited to treatment/admission dates in the months of May through September, exclude cases outside this range. • AND <ul style="list-style-type: none"> ○ revenue code: 762, or ○ positive OS charge when revenue codes not available, or ○ CPT codes: 99217–99220 or 99234–9923 • These criteria are consistent with the criteria used by AHRQ (see: http://www.hcup-us.ahrq.gov/db/vars/siddistnote.jsp?var=hcup_ed). 	Code	Description	992.0	Heat stroke and sunstroke	992.1	Heat syncope	992.2	Heat cramps	992.3	Heat exhaustion from water depletion	992.4	Heat exhaustion from salt depletion	992.5	Heat exhaustion, unspecified	992.6	Heat fatigue, transient	992.7	Heat edema	992.8	Other specified heat effects	992.9	Unspecified effects of heat and light	E900.0	Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)	E900.9	Effect from unknown cause of excessive heat
Code	Description																										
992.0	Heat stroke and sunstroke																										
992.1	Heat syncope																										
992.2	Heat cramps																										
992.3	Heat exhaustion from water depletion																										
992.4	Heat exhaustion from salt depletion																										
992.5	Heat exhaustion, unspecified																										
992.6	Heat fatigue, transient																										
992.7	Heat edema																										
992.8	Other specified heat effects																										
992.9	Unspecified effects of heat and light																										
E900.0	Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)																										
E900.9	Effect from unknown cause of excessive heat																										
Step #3	<p><i>Identifying the required date file content</i></p> <p>Each record should include the following variables:</p> <ul style="list-style-type: none"> • Any primary or other diagnosis code • Date of admission • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient’s sex • Patient’s race (optional) – White, Black, Other, Unknown • Patient’s ethnicity (optional) – Hispanic, Non-Hispanic, Unknown • County of residence • State of residence <p><i>Data Specifications</i></p> <p>Refer to the Data Dictionary in order to conform with the coding specifications required for the NCDM variables. Note that the county FIPS code is 5 digits and that the first two digits will be the same as the state FIPS code. Note also that some variables are</p>																										

	<p>optional.</p> <ul style="list-style-type: none"> • For SAS users, the admission date (and discharge and birth date) variable is acceptable in the following formats: <ul style="list-style-type: none"> ○ SAS DATE FORMAT ○ MMDDYYYY ○ MMDDYY ○ MM/DD/YYYY ○ MM-DD-YYYY ○ DDMONYYYY ○ DDMONYY ○ YYYYMMDD ○ DDMONYYYY:00:00:00 • The base format for counts and population data should be by 5-year age groups beginning 0–4 and ending with 85+. ED Visit counts must be submitted to CDC by these 5-year age groups (see Data Dictionary). Refer to the measure-specific step for the appropriate age groups for calculation and presentation. In summary, the ED Visit and population age-groups required for the calculation and presentation of measures are: <ul style="list-style-type: none"> ○ Heat Stress counts to CDC: 5-yr age-groups (0–4, 5–9 ... 85+) ○ Heat Stress age-specific rate presentation: 0–4, 5–14, 15–24, 25–34, 35–44, 45–54, 55–64 and 65+ ○ Heat Stress crude and age-adjusted rates: 5-yr age groups • Race and ethnicity variables are optional. Therefore, data files and counts and measures may be generated without specifying race or ethnicity if these data are missing or considered unreliable/inaccurate. If race and ethnicity data are reported, the NCDMs will only be generated when the source variables for race and ethnicity are separate variables. The NCDMs will not be generated if race and ethnicity are reported as a combined race/ethnicity variable. If race and ethnicity data are being provided make sure that the coding structure conforms to that described in the Data Dictionary. <ul style="list-style-type: none"> ○ Race: White; Black; Other; Unknown. ○ Ethnicity: Hispanic; non-Hispanic; Unknown. <p>Please consult your data steward and data managers to understand what types of ED Visits are included and excluded (e.g., resident out-of-state ED Visits) and the available variables and coding system (e.g., some data stewards may code race and ethnicity as one variable whereas others may code them as separate variables).</p>
Step #4	<p><i>Selecting records for year of interest:</i> 2000–present calendar year. The SAS program (not provided by CDC) prepares one NCDM file for each year, as required for CDC submission.</p> <p>Most ED Visit data are released in annual discharge-based datasets. In order to have complete admission date information using discharge-based datasets, it is necessary to</p>

	have the dataset of the year of interest <u>and</u> the subsequent year. Since hospital data is based on discharge year and the EPHT dataset is to be based on admission date, patients discharged in current year (i.e., 2006) but admitted to the ED in the previous year (i.e., 2005) should be counted as 2005 ED Visits.
Step #5	<p><i>Removal of duplicates:</i> This How-to-Guide and accompanying SAS code presumes that the user has already removed any duplicate records, while keeping multiple ED Visits.</p> <p>The following variables may be used to identify duplicate records: hospital code, medical record number, admission date, discharge date, date of birth, sex, and zip code.</p> <p>Duplicate records may also be due to continuation of data beyond a single record line. In this case, duplicates may be identified using a record sequence number.</p> <p>GO TO SECTION B FOR INSTRUCTIONS ON CREATING AN XML FILE IF NOT USING SAS.</p> <p>GO TO SECTION C OR INSTRUCTIONS ON CREATING AN XML FILE IF USING SAS.</p>
Section B: CREATION OF XML DATA FILE FOR NCDMS WHEN NOT USING OPTIONAL SAS CODE	
Step #1	<p><i>Required data file:</i> Each record should include the following variables:</p> <ul style="list-style-type: none"> • Any primary or other diagnosis code • Date of visit • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient's sex • Patient's race (optional) – White, Black, Other, Unknown • Patient's ethnicity (optional) – Hispanic, Non-Hispanic, Unknown • County of residence • State of residence
Step #2	Create required fields according to the specifications of each field provided in the Data Dictionary.
Step #3	Convert the data file to the .XML file format and insert the required header into the XML file, according to the Schema found on SharePoint. Refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data Dictionary.
Step #4	<p>Submit completed XML file to CDC using PHIN-MS.</p> <p><i>GO TO SECTION D FOR INSTRUCTIONS ON CALCULATING MEASURES FOR GRANTEE PORTALS.</i></p>
Section C: CREATION OF XML DATA FILE AND NCDM FILE FOR NCDMS USING OPTIONAL SAS CODE	
Step #1	<i>Create SAS Datasets:</i> Be sure to make copies of the inpatient and outpatient data before

	<p>proceeding. If the ED Visit data is not in SAS format, convert it to SAS format and save it as a permanent dataset for creating the XML and measures. Keep the following variables in the SAS dataset :</p> <ul style="list-style-type: none"> • Any primary or other diagnosis code • Date of admission • Date of discharge • Patient date of birth <u>OR</u> Age at admission • Patient’s sex • Patient’s race and ethnicity (<i>optional</i>) • County of residence • State of residence 																										
Step #2	<p><i>Create XML File:</i> After creating the SAS datasets, download ED-NCDM containing the recommended SAS code and Users Guide. These can be found on SharePoint. The current version will only create xml files (not measures).</p> <p>Unzip the files and read and follow the User’s Guide.</p> <p>Click ED-NCDM.msi to launch the setup wizard. Double click the shortcut icon to launch the program.</p> <p>XML files for each single year of data will be generated for one outcome, as required for CDC submission.</p>																										
Step #3	<p><i>Creating Measures using SAS:</i> SAS menu options 3–9 generate the required NCDMs. These options are currently non-operational. These options will follow the steps described below in Section D. If selecting menu options 3–9, this SAS program will generate a final output in the form of MS Excel spreadsheet.</p> <p>Keep only those records that meet the following criteria:</p> <ul style="list-style-type: none"> • First-listed diagnosis code <table data-bbox="516 1276 1161 1801" style="margin-left: 40px;"> <thead> <tr> <th style="text-align: left;">Code</th> <th style="text-align: left;">Description</th> </tr> </thead> <tbody> <tr><td>992.0</td><td>Heat stroke and sunstroke</td></tr> <tr><td>992.1</td><td>Heat syncope</td></tr> <tr><td>992.2</td><td>Heat cramps</td></tr> <tr><td>992.3</td><td>Heat exhaustion from water depletion</td></tr> <tr><td>992.4</td><td>Heat exhaustion from salt depletion</td></tr> <tr><td>992.5</td><td>Heat exhaustion, unspecified</td></tr> <tr><td>992.6</td><td>Heat fatigue, transient</td></tr> <tr><td>992.7</td><td>Heat edema</td></tr> <tr><td>992.8</td><td>Other specified heat effects</td></tr> <tr><td>992.9</td><td>Unspecified effects of heat and light</td></tr> <tr><td>E900.0</td><td>Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)</td></tr> <tr><td>E900.9</td><td>Effect from unknown cause of excessive heat</td></tr> </tbody> </table> <ul style="list-style-type: none"> • <i>Remove</i> any records having ICD-9 code E900.1 (man-made source of heat) as a 	Code	Description	992.0	Heat stroke and sunstroke	992.1	Heat syncope	992.2	Heat cramps	992.3	Heat exhaustion from water depletion	992.4	Heat exhaustion from salt depletion	992.5	Heat exhaustion, unspecified	992.6	Heat fatigue, transient	992.7	Heat edema	992.8	Other specified heat effects	992.9	Unspecified effects of heat and light	E900.0	Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)	E900.9	Effect from unknown cause of excessive heat
Code	Description																										
992.0	Heat stroke and sunstroke																										
992.1	Heat syncope																										
992.2	Heat cramps																										
992.3	Heat exhaustion from water depletion																										
992.4	Heat exhaustion from salt depletion																										
992.5	Heat exhaustion, unspecified																										
992.6	Heat fatigue, transient																										
992.7	Heat edema																										
992.8	Other specified heat effects																										
992.9	Unspecified effects of heat and light																										
E900.0	Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)																										
E900.9	Effect from unknown cause of excessive heat																										

	<p>cause of injury or other diagnosis.</p> <ul style="list-style-type: none"> • If data are not already limited to treatment/admission dates in the months of May through September, exclude cases outside this range. • State = Your state • Date of Birth (if available and being used to calculate patient age) is not missing • Patient’s age at the time of ED Visit is not missing • Date of Discharge is not missing • Date of Admission is not missing. <p><i>Population Data:</i> US Census bureau residential population data for state and county. Intercensal population estimates should be used for the intercensal years, and population extrapolations for postcensal years.</p>
Section D: GENERATE MEASURES FOR GRANTEE PORTALS	
ANNUAL NUMBER OF ED VISITS	
Step #1	Annual Number of ED Visits by sex and total
	<p><i>State:</i> Calculate the number of visits during the year of interest by sex (male, female, and unknown /missing). Then sum the number of visits across sex (male + female + unknown/missing) for the total annual number of ED Visits for the state.</p> <p><i>County:</i> Calculate the number of visits during the year of interest by county of residence and sex (male, female, unknown/missing). Then sum the number of visits across sex (male + female + unknown/missing) to get the total annual number of ED Visits by county of residence.</p>
Step #2	Annual number of ED Visits by race and total (<i>optional</i>)
	<p><i>State:</i> Calculate the number of visits during the year of interest by race (white, black, other, unknown). Then sum the number of visits across race (white + black + other + unknown) for the total annual number of ED Visits for the state.</p> <p><i>County:</i> Calculate the number of visits during the year of interest by county of residence and race (white, black, other, unknown). Then sum the number of visits across race (white + black + unknown) to get the total annual number of ED Visits by county of residence.</p>
Step #3	Annual number of ED Visits by age groups and total
	<p><i>State:</i> Calculate the number of visits during the year of interest for each 5-year age specific category (0–4, 5–9... 85+). Then sum the number of visits across all age groups to get the total annual number of ED Visits for the state.</p> <p><i>County:</i> Calculate the number of visits during the year of interest by county of residence and each 5-year age category (0–4, 5–9... 85+). Then sum the number of visits across all age categories to get the total annual number of ED Visits for each county of residence.</p>
MONTHLY NUMBER OF ED VISITS (<i>optional</i>)	

Step #4	Average Number of ED Visits per Month (<i>optional</i>)
	<p>NOTE: Average number of ED Visits per month is <u>not</u> a required NCDM and is not submitted to CDC or required to be placed on grantee portals. Because of the potential future use of this measure, it is included in the How-to-Guide.</p> <p><i>State:</i> Calculate the number of ED Visits for the state for a given month during the year of interest. Then divide the monthly totals by the number of days in that month (i.e. the denominator for January would be 31), adjusting for leap years when necessary.</p> <p><i>County:</i> Calculate the number of ED Visits by county of residence for the year of interest. Then divide the monthly total by the number of days in that month (i.e. the denominator for January would be 31), adjusting for leap years when necessary.</p>
<i>DAILY NUMBER OF ED VISITS (optional)</i>	
Step #5	<p>NOTE: Daily number of ED Visits is <u>not</u> a required NCDM and is not submitted to CDC or required to be placed on grantee portals. Because of the potential future use of this measure, it is included in the How-to-Guide.</p> <p>Sum the total number of ED Visits for each day by sex during the year of interest for the entire state by sex. Add the daily number of male, female, and unknown (including missing sex information) to obtain the total number of daily admissions. Repeat the above by race/ethnicity and 5-year age groups to calculate the daily number of ED Visits by race/ethnicity and age groups.</p>
ANNUAL UNADJUSTED (CRUDE) RATE OF ED VISITS	
Step #6	Annual ED Visit rate by sex and total per 100,000 population
	<p>Exclude any observation where sex is unknown or missing. Sum the ED Visits for male and female to obtain the total ED Visits for each age group.</p> <p>Use U.S. Census Bureau residential population data for state and county (see Section C, Step #3).</p> <p><i>State (required)</i></p> <ul style="list-style-type: none"> • <i>Numerator:</i> The annual number of ED Visits for males, females, and total for the year of interest • <i>Denominator:</i> The population for the state for males, females, and total. • <i>Constant:</i> 100,000 • <i>Formulas:</i> <p>Unadjusted (Crude) Rate for males per 100,000 people = # of male ED Visits / total male state population × 100,000</p> <p>Unadjusted (Crude) Rate for females per 100,000 people = # of female ED Visits / total female state population × 100,000</p> <p>Unadjusted (Crude) Rate for total population per 100,000 people = (# male + # female) annual ED Visits / total state population × 100,000</p>

	<p><i>County (required)</i></p> <ul style="list-style-type: none"> • <i>Numerator:</i> The annual number of ED Visits for males, females, and total by county of residence for the year of interest. • <i>Denominator:</i> The population for each county of residence in the state for males, females, and total. • <i>Constant:</i> 100,000. • <i>Formulas:</i> <p>Unadjusted (Crude) Rate for males per 100,000 people = # of male ED Visits for each county of residence / total male county population × 100,000</p> <p>Unadjusted (Crude) Rate for females per 100,000 people = # of female ED Visits for each county of residence / total female county population × 100,000</p> <p>Unadjusted (Crude) Rate for total population per 100,000 people = (# of male annual ED Visits + # of female annual ED Visits) for each county of residence / total county population × 100,000</p>
<p>ANNUAL AGE ADJUSTED RATE OF ED VISITS</p>	
<p>Step #7</p>	<p>Annual age-adjusted rate of ED Visits by sex and total per 100,000 population</p>
	<p>Exclude any ED Visit observation where sex is unknown or missing. Sum the admissions for male and female to obtain the totals for each age-group. To calculate age-specific rates (for the 5-year age categories 0-4, 5-9...85+), use US Census bureau residential population data for denominators. Intercensal population estimates should be used for the intercensal years. The standard population should be the 2000 U.S. Standard Population divided into 18 age groups (http://seer.cancer.gov/stdpopulations/). For tutorial on age-adjustment see http://seer.cancer.gov/seerstat/tutorials/aarates/definition.html.</p> <p><i>State:</i></p> <ul style="list-style-type: none"> • Calculate age-specific rates for male, female and total for 5-year age groups (0–4, 5–9, ..., 85+) by dividing the number of state ED Visits in that age group and sex by the Census state population of same age group and sex. • Compute age-adjustment population weights for male, female and total for 5-year age groups using the 2000 US Standard population as follows: $\text{Age-adjusted weight} = \text{age-specific std pop} / \text{total std pop}.$ • Multiply the age-specific rate × age adjustment weight for each age group for male, female and total. • Compute age-adjusted ED Visit rate for male, female and total by summing the product of the previous step for each age group i: $\sum(\text{rate}_i \times \text{weight}_i)$ <p><i>County:</i></p> <ul style="list-style-type: none"> • Calculate age-specific rates for male, female and total for 5-year age groups (0-4, 5-9, ..., 85+) by dividing the number of county ED Visits in that age group and

	<p>sex by the Census county population of same age-age group and sex.</p> <ul style="list-style-type: none"> • Compute age-adjustment population weights for male, female and total for 5-year age groups using the 2000 US Standard population as follows: Age-adjusted weights = age-specific std pop/total std pop. • Multiply the age-specific rate \times age adjustment weight for each age group for male, female and total. • Compute age-adjusted ED Visit rate for male, female and total by summing the product of the previous step for each age group i: $\sum(\text{rate}_i \times \text{weight}_i)$ <p><i>Confidence Intervals (optional):</i></p> <ul style="list-style-type: none"> • 95% confidence intervals may be calculated. <p>Lower Confidence Limit (LCL) = [age-adjusted rate – { 1.96 \times age-adjusted rate / SQRT (Number of ED Visits)}]</p> <p>Upper Confidence Limit (UCL) = [age-adjusted rate + { 1.96 \times age-adjusted rate / SQRT (Number of ED Visits)}]</p> <p>Please Note: With small numbers of ED Visits (i.e. ED Visits < 20), calculation methods assuming a non-normal distribution may be more appropriate.</p>
Section E: PRESENTATION & DISPLAY	
Aggregation & Suppression	Follow your state’s rules, laws, and regulations as well as rules and agreements between you and your data partner(s) in determining whether and when small cell values need to be suppressed.
Visual display	<p>If using optional SAS code, export SAS datasets to Microsoft Excel® for creating charts or for formatting the computed values for presentation.</p> <p>Aggregations calculated under the “Data Measurements” should be displayed, using Microsoft Excel® or equitable spreadsheet product, by state at a minimum and if available and appropriate by county. Recommended spreadsheet displays include:</p> <ul style="list-style-type: none"> • Annual number of Heat Stress ED Visits by state and county • Unadjusted (crude) rate of Heat Stress ED Visits by state and county • Age-Adjusted rate of Heat Stress ED Visits by state and county • Average number of daily visits per month by state and county <p>Annual number of ED Visits can be displayed by showing sex on x-axis and number of ED Visits on y-axis. Similarly sex on x-axis can be replaced by race or age groups to display the number of ED Visits by race or age-group. Displays by race and sex are optional.</p> <p>These bar charts can be created by using any spreadsheet application or by using SAS.</p> <p>Pie charts and bar charts should be used as supplementary visual displays in conjunction with spreadsheets for aggregated calculations.</p> <p>Mapping of calculated counts and rates should be done on the county level.</p>

	<p>Recommended maps include:</p> <ul style="list-style-type: none"> • Annual number of Heat Stress ED Visits by county per year • Age-Adjusted rate of Heat Stress Ed Visits by county per year <p>Public can view bar charts and map showing the state and county level Heat Stress measures. These visual displays will show whether the selected geographical area has a high, medium high, medium low, or low ED Visits rate. The public will also be able to see the links to other related information from various national, state and local sources.</p> <p>Mapping the rate of ED Visits per 100,000 residents will allow users to assess the level of environmentally related risk factors in their residential geographic area as well as in the surrounding geographical area. Future mapping may include other risk factors such as poverty and other potential risk factors. One limitation of mapping is that not all sources of Heat Stress risk factors may be mapped. For example, indoor mold, dust, and pollen.</p>
Interpretation	<p><i>Small Numbers:</i> Measures that generate counts or rates based upon numbers that are small and potentially violate state privacy guidelines can collapse data across years or geographic units to generate data that can be released.</p> <p><i>Measures for multiple years:</i> The how-to-guide steps can be repeated for additional years of ED Visit data. Multi-year ED Visit data can be merged to create one dataset. Add the number of ED Visits for each year in multi-year cohort and divide by the number of years to calculate an average annual number of ED Visits.</p>

HOW-TO GUIDE
Heat Stress Hospitalizations
Environmental Public Health Tracking
07-01-13

Data Source	Inpatient Hospitalization Admissions
NCDM Requirements	<ul style="list-style-type: none"> • Health Outcome = Heat Stress • State/County of Residence • Admission Year/Month • Age Group • Sex • Race/Ethnicity (optional) • Transfers not to be excluded • Out-of-State residents to be excluded • Admissions to federal facilities to be excluded • Admissions of residents to out-of-state hospitals are to be optionally included
Measures	<ul style="list-style-type: none"> ▪ Annual Number of Hospital Admissions by age group, sex, race/ethnicity*, and county and state. ▪ Annual Crude (unadjusted) Rate of Hospital Admissions for all ages by sex, race/ethnicity*, and county and state ▪ Annual Age-Adjusted Rate of Hospital Admissions for all ages by sex, race/ethnicity*, and county and state <p>*Measures by race/ethnicity are optional</p>
Definitions	<p><i>Admission date:</i> The date of the hospital admission; month, day, and year. Month and year are required.</p> <p><i>Discharge date:</i> The date of discharge from hospital. When the date of the hospital discharge is not available, use the admission date, if available.</p> <p><i>Duplicate records:</i> More than one record for the same person with the same hospital admission data (e.g., where sex, date of birth, admission date, and zip code have exactly same information).</p> <p><i>Event/Event Year:</i> A hospital admission for Heat Stress illness results with a primary diagnosis of 493.XX during May 1 – September 30 of a specific calendar year. Event year is based only upon admission year, even when discharge year is different.</p> <p><i>Heat Stress:</i> Heat stress is defined as a constellation of explicit effects of hot weather on the body including heat stroke and sunstroke (hyperthermia), heat syncope/collapse, heat exhaustion, heat cramps, heat fatigue, heat edema, and other/unspecified clinical effects attributed to excessive heat exposure. It only includes cases that occurred during May 1 to September 30 of each calendar year. Heat stress is classified as any primary or other diagnosis code in the range of 992.0–992.9, or cause of injury code E900.0 or E900.9.</p>

	<p>Cases with a code of E900.1 (man-made source or heat) anywhere in the record are <u>excluded</u>.</p> <p><i>Hospital Transfers:</i> Generally, a patient discharged from one facility and readmitted to a second facility on the same day.</p> <p><i>Hospitalization/ Hospital Admission:</i> Condition of being placed (Admission) or treated as a patient in an acute care hospital for treatment as an inpatient. Treatment as an out-patient is not considered to be hospitalization. To be considered as inpatient Hospitalization, a minimum stay is required (often over 23 hours).</p> <p><i>Multiple admissions:</i> Second or subsequent admission for the same person for the same primary diagnosis code but on a different date and related to a separate event within a given year. Multiple admissions are considered separate events (generally at least 48 hours apart).</p> <p><i>Out-of-State admissions:</i> When a resident of the grantee state is admitted to a hospital located in another state (usually an abutting state).</p> <p><i>Primary Diagnosis Code:</i> Presently, diagnosis codes are represented by ICD-9-CM codes (the International Classification of Diseases, 9th Revision, Clinical Modification). Heat stress is classified as any primary or other diagnosis code in the range of 992.0-992.9, or cause of injury code E900.0 or E900.9, Cases with a code of E900.1 (man-made source or heat) anywhere in the record are <u>excluded</u>.</p> <p><i>Resident:</i> Any person with a residential address in the county/state of the grantee at the time of the hospital admission.</p>
<p>How-to-Guide Requirements and Cautions</p>	<ul style="list-style-type: none"> • This How-to-Guide has two purposes: 1) Guide the user in the development of the XML dataset using the SAS code, located on SharePoint, for submission to CDC; 2) Describe the calculation steps for creating the Nationally Consistent Data Measures (NCDMs). Grantees should use the How-to-Guide in order to ensure that the calculation of measures are consistent with those required by CDC in the preparation of their own computer code. • Note that one set of SAS code (not provided by CDC’s EPHT program) is designed to handle the creation of XML files for all hospitalization (inpatient) outcomes (separate code is available for all ED Visit outcomes). The code manages the differences in definitions between the outcomes and generates a separate file for each outcome by each single year. • This how-to-guide and accompanying SAS code (not provided by CDC’s EPHT program) presume that the user already removed any duplicate records (see definitions for more information), while keeping multiple admissions. • Hospitalizations due to transfers between acute care hospitals are not excluded in the counts/measures to be generated. Therefore, for consistency, it is advised that transfers not be excluded. An algorithm to exclude transfers is underdevelopment. NOTE: The Date Dictionary includes two variables regarding the exclusion of transfers. These are placeholders only (to be activated in future work) and the SAS code will automatically set the codes for these variables the same for all grantees.

	<p>These variables do not need to be a part of your SAS dataset. If they are present, the program will ignore them.</p> <ul style="list-style-type: none"> • The data source is an inpatient discharge dataset but the EPHT dataset is based upon date of admission because of the goal of relating a hospitalization event with an environmental event. Therefore, the hospitalization counts and measures require the development of an admission dataset. For admissions at the end of a calendar year where the discharge date is in the following year, that latter year’s discharge dataset will be required before the admission dataset for the preceding year can be considered complete. • Data suppression/aggregation rules are not incorporated into the SAS code. Suppression guidelines are currently applied by CDC for the national portal. • Please note that the steps for estimating age-adjusted rates are not the same for different health outcomes. • Admissions of residents to out-of-state hospitals are not required to be included. However, when available these admissions should be included. It is noted that some states must include out-of-state admissions of its residents. Use the “OUTOFSTATEEXCLUSION” variable in the dataset to capture whether out of-state admissions are included or not (the Data Dictionary and schema provide for formal notation in the dataset on whether these admissions are included). Be certain to use footnotes and metadata to acknowledge the disposition of these admissions. • Admissions to federal facilities, such as Veteran’s Hospitals, are not included. Be certain to inform CDC if your state requires that your dataset includes admissions to federal facilities so that the measures can be appropriately footnoted. • The Data Dictionary should be referred to for the standardized definitions and notations of the variables to be submitted to CDC.
--	--

NOTE FOR NON-SAS USERS: Grantees not using SAS should use the steps outlined below for guidance on important issues such as population requirements and should refer to the Data Dictionary for specifications of the required fields.

The data file should be converted to the .XML file format and the required header inserted into the XML file, according to the Schema found on SharePoint. In addition, refer to the crosswalk between the Schema and Data Dictionary for identification of the necessary field names for the XML file (located in the "Aggregate Data Set Summary" section of the Data Dictionary).

NOTE FOR SAS USERS: SAS code is not developed or distributed by CDC.

STEPS FOR CREATING SAS HOSPITALIZATION DATASET

Step #1	<p><i>Source of Data:</i> Individual level state inpatient hospital admission data based on primary diagnosis.</p> <p>Please consult your data steward and data mangers to understand the variables and</p>
---------	---

coding system, specifically for race and ethnicity variables. In some states these variables may be coded as one variable whereas in others they are coded as separate variables.

Years of Interest: 2000–present calendar year. The SAS program (not provided by CDC) prepares one NCDM file for each year, as required for CDC submission.

Most hospitalization data is released in annual discharge-based datasets. In order to have complete admission date information using discharge-based datasets, it is necessary to have the dataset of the year of interest and the subsequent year. For example, to obtain all admissions during 2005 using discharge based datasets, it will be necessary to have both the 2005 and the 2006 discharge datasets for admissions that occurred in 2005 but were not reported until release of the 2006 discharge dataset. For this example, 2005 data should not be submitted prior to receipt of the 2006 discharge dataset from the data steward.

Removal of Duplicates: This how-to-guide and accompanied SAS code presumes that the user has already removed any duplicate records, while keeping multiple admissions.

Data Specifications: Refer to the Data Dictionary in order to conform to coding specifications required for the NCDM variables.

NOTE: county FIPS code is 5 digits and that the first two digits will be the same as the state FIPS code. Also, some variables are optional.

Select all hospital records that meet the following criteria:

- Exclude all records where the State of residence is not your state.
- Since hospitalizations data are based on discharge year and the EPHT dataset is to be based on admission date, patients discharged in current year (i.e., 2006) but admitted in the previous year (i.e., 2005) should be counted as 2005 hospitalizations. The admission date (and discharge and birth date) variable is acceptable in the following formats:
 - SAS DATE FORMAT
 - MMDDYYYY
 - MMDDYY
 - MM/DD/YYYY
 - MM-DD-YYYY
 - DDMONYYYY
 - DDMONYY
 - YYYYMMDD
 - DDMONYYYY:00:00:00

SAS Dataset: Make a copy of the hospital admission data before proceeding to next step. If the admission data are not in SAS format, convert it to SAS format and save it as a permanent dataset for creating the XML and measures. Keep the following variables in the SAS dataset:

- Any primary or other diagnosis code
- Date of admission
- Date of discharge

	<ul style="list-style-type: none"> ○ Patient date of birth <u>OR</u> Age at admission ○ Patient’s sex ○ Patient’s race (optional) - White, Black, Other, Unknown ○ Patient's ethnicity (optional) - Hispanic, Non-Hispanic, Unknown ○ County of residence ○ State of residence <p><i>Population Data:</i> U.S. Census bureau residential population data for state and county. Intercensal population estimates should be used for the intercensal years, and population extrapolations for postcensal years.</p> <p><i>Go to Step # 2.</i></p>
Step #2	<p>The base format for counts and population data should be by 5-year age groups for heat stress beginning 0–4 and ending with 85+.</p> <p>Hospitalization counts must be submitted to CDC by these 5-year age groups (see Data Dictionary). For the calculation of measures and presentation, the age-groups of interest for various Heat Stress measures are different because of the nature of the diseases. Refer to the measure-specific step for the appropriate age groups for calculation and presentation. In summary, the hospitalization and population age-groups required for the calculation and presentation of measures are:</p> <p>Heat stress counts to CDC: 5-yr age-groups (0–4, 5–9 ... 85+)</p> <p>Heat Stress age-specific rate presentation: 0–4, 5–14, 15–24, 25–34, 35–44, 45–54, 55–64 and 65+</p> <p>Heat stress crude and age-adjusted rates: 5-yr age groups</p> <p>Race and ethnicity variables are optional. Therefore, counts and measures may be generated without specifying race or ethnicity if these data are missing or considered unreliable/inaccurate. If race and ethnicity data are reported, the NCDMs will only be generated when the source variables for race and ethnicity are separate variables. The NCDMs will not be generated if race and ethnicity are reported as a combined race/ethnicity variable. If race and ethnicity data are being provided, be sure that the coding structure conforms to that laid out in the Data Dictionary.</p> <p>Race: White; Black; Other; Unknown.</p> <p>Ethnicity: Hispanic; non-Hispanic; Unknown.</p> <p><i>Go to Step # 3.</i></p>
<p>STEPS FOR GENERATING NCDM REQUIREMENTS</p> <p><i>(Grantees not using SAS should refer to the steps below for conversion of their data file to XML format)</i></p>	

Step #3	<p>After creating the SAS datasets, download IP-NCDM containing the recommended SAS code and Users Guide. These can be found on SharePoint. The current version will only create xml files (not measures).</p> <p>Unzip the files and read and follow the User’s Guide. Click IP-NCDM.msi to launch the setup wizard. Double click the shortcut icon to launch the program.</p> <p>XML files for each single year of data will be generated for one outcome, as required for CDC submission.</p> <p>SAS code to generate measures is not yet operational.</p> <p><i>NOTE:</i> Single year files for individual outcomes must be submitted, as specified in the data call letter</p> <p>Steps #4 through #12 should be used to calculate the NCDMs to ensure consistency between grantees.</p>
---------	--

STEPS FOR COMPLETING SPECIFIC MEASURES

Step #4	<p>SAS menu options 3–9 generate measures beyond those required for the national portal but are currently non-operational. These options will follow the steps below (4b through 12). These steps may also be used to generate measures outside of SAS. If selecting menu options 3–9, this SAS program will generate a final output in the form of MS Excel spreadsheet. A PDF file containing histograms will also be created.</p> <p>Flag all admissions where primary diagnosis code is “992.0-992.9 or E900.0 or E900.9 (exclude E900.1)” by creating a variable (<i>for example Ishospital</i>) that takes the value of 1, if admission is due to diagnosis codes targeted; else its value is 2.</p> <p>Select cases having any of the following ICD-9 codes as a principal diagnosis, injury cause, or other diagnoses:</p> <table style="margin-left: 40px;"> <thead> <tr> <th style="text-align: left;">Code</th> <th style="text-align: left;">Description</th> </tr> </thead> <tbody> <tr><td>992.0</td><td>Heat stroke and sunstroke</td></tr> <tr><td>992.1</td><td>Heat syncope</td></tr> <tr><td>992.2</td><td>Heat cramps</td></tr> <tr><td>992.3</td><td>Heat exhaustion from water depletion</td></tr> <tr><td>992.4</td><td>Heat exhaustion from salt depletion</td></tr> <tr><td>992.5</td><td>Heat exhaustion, unspecified</td></tr> <tr><td>992.6</td><td>Heat fatigue, transient</td></tr> <tr><td>992.7</td><td>Heat edema</td></tr> <tr><td>992.8</td><td>Other specified heat effects</td></tr> <tr><td>992.9</td><td>Unspecified effects of heat and light</td></tr> <tr><td>E900.0</td><td>Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)</td></tr> <tr><td>E900.9</td><td>Effect from unknown cause of excessive heat</td></tr> </tbody> </table> <ul style="list-style-type: none"> - State = Your state - Date of Birth (if available and being used to calculate patient age) is not missing - Patient’s age at the time of hospital admission is not missing 	Code	Description	992.0	Heat stroke and sunstroke	992.1	Heat syncope	992.2	Heat cramps	992.3	Heat exhaustion from water depletion	992.4	Heat exhaustion from salt depletion	992.5	Heat exhaustion, unspecified	992.6	Heat fatigue, transient	992.7	Heat edema	992.8	Other specified heat effects	992.9	Unspecified effects of heat and light	E900.0	Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)	E900.9	Effect from unknown cause of excessive heat
Code	Description																										
992.0	Heat stroke and sunstroke																										
992.1	Heat syncope																										
992.2	Heat cramps																										
992.3	Heat exhaustion from water depletion																										
992.4	Heat exhaustion from salt depletion																										
992.5	Heat exhaustion, unspecified																										
992.6	Heat fatigue, transient																										
992.7	Heat edema																										
992.8	Other specified heat effects																										
992.9	Unspecified effects of heat and light																										
E900.0	Health effect caused by excessive heat due to weather (e.g., sunstroke, ictus solaris/heatstroke)																										
E900.9	Effect from unknown cause of excessive heat																										

	<ul style="list-style-type: none"> - Date of Discharge is not missing - Date of Admission is not missing. <p><i>Remove any records having ICD-9 code E900.1 (man-made source of heat) as a cause of injury or other diagnosis. <u>If data are not already limited to admission in the months of May through September, exclude cases outside this range.</u> Assign geography by state and county of patient's residence.</i></p> <p><i>Go to Step # 5.</i></p>
ANNUAL NUMBER OF HOSPITAL ADMISSIONS	
Step #5	Annual Number of Hospital Admissions by sex and total
Step #5a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by sex (male, female and unknown (including missing)).</p> <p>Sum the number of male, female and unknown sex hospital admissions to get the total number of hospital admissions for the year.</p>
Step #5b	<p><i>County:</i> Sum the total number of admissions by county of residence for year of interest by sex [male, female and unknown (including missing)].</p> <p>Sum the number of male, female and unknown sex hospital admissions to get the total number of hospital admission by county for the year.</p> <p><i>Number of Heat Stress hospitalizations at county level may not be displayed due to small numbers.</i></p>
Step #6	Annual Number of Hospital Admissions by race and total
Step #6a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by race categories.</p> <p>Sum the number of hospital admissions to get the total number of heat stress hospital admission for the year.</p>
Step #7	Annual Number of Hospital Admissions by age groups and total
Step #7a	<p><i>State:</i> Sum the total number of admissions for year of interest for the state by the specific 5-year age groups created in step #2.</p> <p>Sum the number of hospital admissions for each age group to get the total number of hospital admission for the year.</p>
DAILY NUMBER OF HOSPITAL ADMISSIONS (NOT A REQUIRED NCDM)	
Step #8	<p>NOTE: Daily number of admissions is not a required NCDM and is not submitted to CDC or required to be placed on state portals. Because of the potential future use of this measure. It remains in the How-to-Guide and SAS code.</p> <p>Sum the total number of admission for each day by sex during the year of interest for entire state to get the daily number of admissions by sex. Add the daily number of male, female and unknown (including missing sex information) to obtain the total number of daily admissions. Repeat the above by race and 5-year age-groups to calculate the daily number of hospital admissions by race and age groups.</p>

ANNUAL AGE-SPECIFIC HOSPITAL ADMISSIONS RATE	
Step #9	Annual age-specific hospital admission rate by sex
Step #9a	<p><i>Create the numerator data:</i> Sum the number of hospitalizations in the state for the year of interest in each of the 5-year age-groups for both male and female. Exclude any observation where sex is unknown or missing. Sum the admissions for male and female to obtain the total admissions for each age-group.</p> <p>NOTE: Remember to includes Heat Stress cases from May 1 to September 30 of each reporting year</p>
Step #9b	<i>Create the denominator data:</i> Sum the population in the state for year of interest in each 5-year age group for both male and female. Sum male and female population to obtain the total state population for each age-group.
Step #9c	Merge both numerator and denominator data by state of residence, year of hospital admissions, age-group and sex.
Step #9d	<i>For heat stress presentation:</i> Compute age-specific rates for male, female and total for age-groups 0–4, 5–14, 15–24, 25–34, 35–44, 45–54, 55–64 and 65+ by dividing the number of hospital admissions in that age group and sex by the population of same age-age group and sex. For example, to calculate the age-specific rates of admissions in 5–14 year old male divide the annual number of hospital admissions in 5–14 years old male by the population of 5–14 years old male.
Step #9e	All rates are to be presented as per 100,000 population. Multiply the rates calculated in step 9d by 100,000 to obtain rate of admission per 100,000 population
Step #9f	<p>Upper and lower confidence limits (95% confidence interval) for age-specific rates may be computed.</p> <p>For each age-specific rate computed in step 9d, compute Lower Confidence Limit (LCL) and Upper Confidence Limit (UCL) as follows</p> <p>LCL = [age-specific rate – { 1.96 * age-specific rate/SQRT (Number of Admissions)}]</p> <p>UCL = [age-specific rate + { 1.96 * age-specific rate/SQRT (Number of Admissions)}]</p>
ANNUAL UNADJUSTED (CRUDE) RATE OF HOSPITAL ADMISSIONS	
Step #10a	<p>Exclude any observation where sex is unknown or missing.</p> <p><i>Heat Stress:</i> Create the numerator data (referred to in Step #2) to obtain the annual number of hospital admissions by sex for both male and female across all ages. Sum the male and female number of admissions to obtain total admissions for both sexes.</p>
Step #10b	<p><i>Heat Stress:</i> Create the denominator data as referred to in step #2 across all ages. Sum the population for male, female and both sex for the year of interest.</p> <p>NOTE: Remember to includes Heat Stress cases from May 1 to September 30 of each reporting year</p>
Step #10c	Merge both numerator and denominator data by state of residence, year of hospital admissions and sex.

Step #10d	<p>Compute the annual unadjusted rate of hospital admissions as follows:</p> <p>Unadjusted Admission Rate (Male) = (# of Male Admissions/Male Population)</p> <p>Unadjusted Admission Rate (Female) = (# of Female Admissions/Female Population)</p> <p>Unadjusted Admission Rate (Total) = (Total Admissions/ State Population)</p> <p>Multiply the above computed rates by 100,000 to obtain the number of admissions per 100,000 population.</p>
ANNUAL AGE ADJUSTED RATE OF HOSPITAL ADMISSIONS	
Step #11	Annual Age Adjusted Rate of Hospital Admissions for State by Sex
Step #11a	<i>Heat Stress</i> : Calculate the age specific rates as described in steps 10a through 10d using 5 yr age groups (0–4, 5–9 ... 85+) for male, female and both sexes.
Step #11b	<p>To calculate age-specific rates (for the 5-year age categories 0–4, 5–9... 85+); use U.S. Census bureau residential population data for denominators. Intercensal population estimates should be used for the intercensal years. . The standard population should be the 2000 U.S. Standard Population divided into 18 age groups. The link for the 2000 U.S. Standard Population is: http://seer.cancer.gov/stdpopulations/). After downloading, combine the '0' age group with the '1–4' age group. Calculate annual warm-season rates per 100,000 residents, adjusted for age, by state and county.</p> <p><i>NOTE</i>: County-level measures may require aggregation of years due to small numbers.</p>
Step #11c	Merge both numerator and denominator data by age group and sex.
Step #11d	<p>Compute the age-adjusted population weights using the 2000 US population as the standard.</p> <p><i>Heat Stress</i>: Compute the age-adjustment weights of hospital admissions using 2000 US Standard Population by age group for males, females, and both sexes as follows:</p> <p>Age-adjusted weights = age-specific std pop/total std pop, where the total weight for all ages is 1.0.</p> <p><i>NOTE</i>: Remember to include Heat Stress cases from May 1 to September 30 of each reporting year</p>
Step #11e	<p>Compute the age-adjusted hospital admissions rate:</p> <p>Age-adjusted rate = Sum of age-specific rate × age adjusted weight</p> <p>For tutorial on age-adjustment see http://seer.cancer.gov/seerstat/tutorials/aarates/definition.html .</p>
Step #11f	<p>95% confidence intervals may be computed.</p> <p>LCL = [age-adjusted rate – { 1.96 × age-adjusted rate/SQRT (Number of Admissions)}]</p> <p>UCL = [age-adjusted rate + { 1.96 × age-adjusted rate/SQRT (Number of Admissions)}]</p> <p><i>NOTE</i>: With small numbers of hospitalizations (e.g., <20), calculation methods assuming a non-Normal distribution may be more appropriate.</p>

Step #11f	To calculate the Annual age-adjusted rate of hospital admissions by County (if needed), follow steps 11a through 11f using the same 2000 US standard population.
PRESENTATION	
Step #12a	Export SAS datasets to Microsoft Excel® for creating charts or for formatting the computed values for presentation
Step #12b	Annual number of hospital admissions can be displayed by showing sex on x-axis and number of admissions on Y-axis. Similarly sex on X-axis can be replaced by race or age groups to display the number of admissions by race or age-group. These bar charts can be created by using any spreadsheet application or by using SAS.
Additional visual display	Public can view histograms and map showing the state or county level Heat Stress measures. These visual displays will show whether the selected geographical area has a high, medium high, medium low or low hospital admission rate. The public will also be able to see the links to other related information from various national, state and local sources. Mapping the rate of hospital admissions per 100,000 residents will allow users to assess the level of environmentally related risk factors in their residential geographic area as well as in the surrounding geographical area. Future mapping may include other risk factors such as poverty and other potential risk factors. One limitation of mapping is that not all sources of heat stress risk factors may be mapped. For example, indoor mold, dust, and pollen.
Interpretation	Small Numbers: Measures that generate counts or rates based upon numbers that are small and potentially violate state privacy guidelines can collapse data across years or geographic units to generate data that can be released. Measures for multiple years: The how-to-guide steps can be repeated for additional years of hospital admission data. Multi-year hospital admission data can be merged to create one dataset. Add the number of hospital admissions for each year in multi-year cohort and divide by the number of years to calculate an average annual number of hospital admissions.

INDICATOR TEMPLATE
CONTENT AREA: CLIMATE AND HEALTH
INDICATOR: HEAT STRESS HOSPITALIZATIONS

Environmental Public Health Tracking

Type of EPHT Indicator	Health outcome
Measures	<ol style="list-style-type: none"> 1. Age-adjusted rate of hospitalization for heat stress per 100,000 population 2. Crude rate of hospitalization for heat stress per 100,000 population 3. Number of hospitalizations for heat stress
Derivation of Measure(s)	<p><i>Numerator:</i> Hospital admissions having any ICD-9 code in the range of 992.0-992.9, or cause of injury code E900.0 or E900.9, EXCLUDING cases with a code of E900.1 (man-made source of heat) anywhere in the record.</p> <p><i>Denominator:</i> Midyear resident population, by gender, for state and by county</p> <p><i>Adjustment:</i> Age-adjustment by the direct method to year 2000 US standard population</p>
Unit	<ol style="list-style-type: none"> 1. Age-adjusted rate per 100,000 population 2. Rate per 100,000 population 3. Number
Geographic Scope	EPHT grantee states with hospitalization data
Geographic Scale	State
Time Period	Hospital admissions between May 1 to September 30, inclusive, for each year, 2000–
Time Scale	May–September of each data year
Rationale	The Intergovernmental Panel on Climate Change (IPCC) projects with “virtual certainty” suggest that climate change will cause more frequent, more intense, and longer heat waves (1). Any individual, regardless of age, sex or health status can develop heat stress if engaged in intense physical activity and/or exposed to environmental heat (and humidity). Physiologic mechanisms maintain the core body temperature (i.e., the operating temperature of vital organs in the head or trunk) in a narrow optimum range around 37 °C (98.6 °F). When core body temperature rises, the physiologic response is to sweat and circulate blood closer to the skin's surface to

increase cooling. If heat exposure exceeds the physiologic capacity to cool, and core body temperature rises, then a range of heat-related symptoms and conditions can develop. Heat stress or Heat-related illness ranges from mild heat edema and rash, heat syncope, heat cramps, to the most common type, heat exhaustion (2). Heat-related cramps, rash, and edema are relatively minor readily treatable conditions; however, they should be used as important warning signs to immediately remove the affected individual from the exposure situation.

Heat cramps are brief, intermittent, and often severe muscular cramps occurring typically in muscles that are fatigued by heavy work (2). Individuals with heat cramp can also exhibit hyponatremia, hypochloremia (which are low serum sodium and chloride levels).

Heat syncope is a temporary loss of consciousness as a result of prolonged heat exposure (2). Individuals adapt to hot, humid environment by dilation of cutaneous vessels in the skin to radiate heat. Peripheral vasodilation along with blood volume loss, results in lowering the blood pressure which can result in inadequate central venous return and cerebral perfusion, causing light-headedness and fainting.

Heat exhaustion is a consequence of extreme depletion of blood plasma volume, which may be coincident with hyponatremia and/or peripheral blood pooling (2). Heat exhaustion often does not present with definitive symptoms and may be misdiagnosed, often as an acute viral illness. Symptoms include mild disorientation, generalized malaise, weakness, nausea, vomiting, headache, tachycardia (rapid beating of the heart), and hypotension. Because untreated heat exhaustion can progress to heat stroke, the most serious form of heat-related illness, treatment should begin at the first signs of heat exhaustion (3).

Heat stroke is an extreme medical emergency that if untreated can result in death or permanent neurological impairment (2). Heat stroke occurs when a person's core body temperature rises above 40 °C (104 °F) as a result of impaired thermoregulation. High core body temperature and disseminated intravascular coagulation results in cell damage in vital organs, such as the brain, liver, and kidneys, which can lead to serious illness and death (3). Death may occur rapidly due to cardiac failure or hypoxia, or it can occur days later as a result of renal failure due to dehydration and/or rhabdomyolysis (i.e., the breakdown of muscle fibers with release into the circulation of muscle fiber contents, some of which are toxic to the kidney and can cause kidney damage) (4). Heat stroke is typically divided into two types. The two types are in general clinically the same, except that the individuals/population groups affected require medical interventions specific

	<p>to their unique physiology and medical status (3). “Exertional Heat Stroke,” as the name implies, involves strenuous physical activity under high temperature conditions to which the heat stroke victim was not acclimatized, and usually affects healthy young adults, such as athletes, outdoor laborers and soldiers. “Classic” heat stroke, by definition does not involve exertion, and usually affects susceptible individuals, such as infants and young children, the elderly, or people with chronic illness. Because heat stroke, even if treated, can have a death rate as high as 33%, and up to 17% of heat stroke survivors suffer permanent damage, measures should be taken to prevent heat-related illness, especially among vulnerable populations.</p> <p>The relationship between extreme heat and increased daily morbidity and mortality is well established. This indicator captures hospital admissions directly attributed to heat stress (e.g., heat illness, heat stroke, and hyperthermia). It is a measure that can be tracked easily and consistently across geography and time, and acts as a sentinel for the broader range of heat-related illness that is not recognized and/or coded as such.</p>
<p>Use of the Measure</p>	<p>Heat stress can manifest in a number of clinical outcomes, and people with chronic health problems (e.g., cardiovascular disease, diabetes, obesity) are more susceptible to the effects of heat than healthy individuals. For these reasons, heat stress may not be listed as the primary diagnosis. This indicator therefore includes all cases where heat stress is explicitly listed as the primary diagnosis or any other diagnosis.</p> <p>Increases in the rates of hospital admission for heat stress are one potential impact of rising global temperatures. Tracking these data can help document changes over place and time, monitor vulnerable areas, and evaluate the results of local climate-adaptation strategies.</p>
<p>Limitations of the Measure</p>	<p>Periods of extreme heat are frequently associated with increases in hospital visits and admissions for many causes. This measure does not capture the full spectrum of heat stress, especially where exposure to excess heat is not explicitly documented.</p>
<p>Data Sources</p>	<p><i>Numerator:</i> State inpatient hospital discharge data (using admission date)</p> <p><i>Denominator:</i> US Census Bureau population data</p>
<p>Limitations of Data Sources</p>	<p><i>State hospital discharge data:</i></p> <ul style="list-style-type: none"> Using a measure of all heat stress hospitalizations will include some transfers between hospitals for the same individual for the same heat stress event. Variations in the percentage of transfers or readmissions for the same heat stress event may vary by geographic area and impact rates.

	<ul style="list-style-type: none"> • Without reciprocal reporting agreements with abutting states, statewide measures and measures for geographic areas (e.g., counties) bordering other states may be underestimated because of health care utilization patterns. • Each state must individually obtain permission to access and, in some states, provide payment to obtain the data. • Veterans Affairs, Indian Health Services and institutionalized (e.g. Prison) populations are excluded. • Practice patterns and payment mechanisms may affect diagnostic coding and decisions by health care providers to hospitalize patients • Street address is currently not available in many states. • Sometimes mailing address of patient is listed as the residence address of the patient • Patients may be exposed to environmental triggers in multiple locations, but hospital discharge geographic information is limited to residence. • Since the data captures hospital discharges (rather than admissions), patients admitted toward the end of the year and discharged the following year will be omitted from the current year dataset • Data will need to be de-duplicated (i.e. remove duplicate records for the same event) • There is usually a two year lag period before data are available from the data owner. <p><i>Census data:</i></p> <ul style="list-style-type: none"> • Only available every 10 years, thus postcensal estimates are needed when calculating rates for years following the census year. • Postcensal estimates at the ZIP code level are not available from the Census
Related Indicators	<ul style="list-style-type: none"> • Heat vulnerability • Heat-related mortality • Temperature distribution • Emergency department visits for heat stress
References	<ol style="list-style-type: none"> 1. Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. 2007. Human health In: Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE. , editors. Climate Change 2007: Impacts, Adaptation and Vulnerability Contribution of Working Group II to: Fourth Assessment Report of the Intergovernmental Panel on Climate Change. New York: Cambridge University Press. pp. 391–431. 2. Rosen’s Emergency Medicine: Concepts and Clinical Practice. 2010. Chapter 139: Heat illness. In JA Marx Editor-in-Chief; RS Hockberger & RM Walls Senior Editors; JG Adams ... [et al] Editors (7th ed).

Philadelphia: Mosby Elsevier.

3. American Medical Association. Heat-related Illness During Extreme Weather Emergencies (Report 10 of the Council on Scientific Affairs (A97), 1997; www.ama-assn.org/ama/pub/category/13637.html).
4. Centers for Disease Control and Prevention. Heat-related deaths--Los Angeles County, California, 1999-2000, and United States, 1979-1998. *MMWR* 2001;50(29):623-6.

How-to-Guide Birth prevalence of birth defect X per 10,000 live births Environmental Public Health Tracking	
Step #1	Note relevant case definition – see Appendix A for details.
Step #2	<p>NOTE: Steps 2 – N are focusing on hypoplastic left heart syndrome. Please see Appendix B for sample SAS code for systems using ICD-9-CM and CDC BPA Codes, for all 12 defects of interest.</p> <p><i>Using your numerator dataset:</i></p> <p>Create binary variable for presence of the defect. If you are working with a file with one record per subject, but multiple variables for diagnoses, you may use an array to pull out the presence of the diagnostic code of interest. For example, if there are 25 variables for birth defect codes, DX1-DX25, the SAS code may look something like this:</p> <pre> array defect (25) dx1-dx25; do i = 1 to 25; if defect (i) = 7467 then HLHS=1; (or if using BPA codes, "if defect (i) = 746700 then HLHS=1;") end;</pre> <p>This will result in the variable HLHS taking the value of 1 if the infant has the HLHS diagnostic code in any of its birth defect diagnosis variables.</p>
Step #3	<p>Cross-tabulate a categorical variable for year of birth with the HLHS variable. Output the frequencies into a dataset. For example; if the year of birth variable is called YEAR_NUMER (2000-2004), the SAS code will look something like this:</p> <pre> proc freq data=numerators; tables YEAR_NUMER *HLHS/out=one; run;</pre> <p>The output dataset will contain four variables, YEAR_NUMER, HLHS, COUNT, and PERCENT, with 10</p>

	<p>records (2 records per year because HLHS is coded either 1 or missing).</p> <p>Rename the COUNT variable COUNT_NUMER. This will be necessary later on because we will be merging this data set with numerator counts with a dataset with denominator counts, and if the variable is called COUNT in both datasets, then the values for COUNT in the second dataset in the merge statement will overwrite the values for COUNT in the first dataset.</p>
<p>Step #4</p>	<p><i>Using your denominator data of live births</i></p> <p>Get a frequency of live births by year and output the frequencies into a dataset. For example, if the year of birth variable in the denominator data is also called YEAR_DENOM (2000-2004), the SAS code will look something like this:</p> <pre>proc freq data=denominators; tables YEAR_DENOM/out=two; run;</pre> <p>Rename the COUNT variable COUNT_DENOM.</p>
<p>Step #5</p>	<p>Rename the year variables in the two output datasets that have been created, to have the same name (such as YEAR). Ensure that they are of the same variable type (character or numeric) and of the same length.</p> <p>Sort both datasets by YEAR.</p> <p>Merge the datasets by YEAR.</p> <p>Your resulting dataset will have 10 records in total, 2 per year, with the variables COUNT_NUMER and COUNT_DENOM containing the values that will be used to calculate prevalence.</p>
<p>Step #6</p>	<p>In this merged dataset, create a new variable called PREVALENCE in which you divide the COUNT_NUMER variable by the COUNT_DENOM variable and multiply by 10,000. This will give the birth prevalence per 10,000 live births. The rows where HLHS=1 are the only meaningful observations in this dataset.</p>

<p>Step #7</p>	<p>Stratification</p> <p>Assuming the numerator and denominator datasets have a maternal race/ethnicity variable available, each defined by the same four levels: Non-Hispanic White, Non-Hispanic Black, Hispanic, and Other.</p> <p>Steps 7-10 show how to calculate annual birth prevalence stratified by race/ethnicity. The procedure would be identical for the other stratification factors of infant sex and maternal age (18-20, 21-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-59). These factors can be collapsed if data are too sparse within these strata for calculation or display.</p> <p><i>Using your numerator dataset</i></p> <p>Cross-tabulate a categorical for maternal race/ethnicity by the categorical variable for year of birth by the HLHS variable. Output the frequencies into a dataset. For example; if the race/ethnicity variable is called RACE4_NUMER and the other variables are as defined previously, the SAS code will look something like this:</p> <pre>proc freq data=numerators; tables RACE4_NUMER*YEAR_NUMER *HLHS/out=one; run;</pre> <p>The output dataset will contain five variables, RACE4_NUMER, YEAR_NUMER, HLHS, COUNT, and PERCENT, with 40 records.</p> <p>Rename the COUNT variable COUNT_NUMER_RACE. This will be necessary later on because we will be merging this data set with numerator counts with a dataset with denominator counts, and if the variable is called COUNT in both datasets, then the values for COUNT in the second dataset in the merge statement will overwrite the values for COUNT in the first dataset.</p>
<p>Step #8</p>	<p><i>Using your denominator data of live births</i></p> <p>Get a frequency of live births by maternal race/ethnicity and year and output the frequencies into a dataset. For example; if the race/ethnicity</p>

	<p>variable is called RACE4_DENOM and year is as defined previously, the SAS code will look something like this:</p> <pre>proc freq data=denominators; tables RACE4_DENOM*YEAR_DENOM/out=two; run;</pre> <p>Rename the COUNT variable COUNT_DENOM_RACE.</p>
Step #9	<p>Rename the year and race variables in the two output datasets that have been created, to have the same name (such as YEAR and RACE). Ensure that they are of the same variable type (character or numeric) and of the same length.</p> <p>Sort both datasets by YEAR and RACE.</p> <p>Merge the datasets by YEAR and RACE.</p> <p>Your resulting dataset will have 40 records in total, 2 per year per race, with the variables COUNT_NUMER_RACE and COUNT_DENOM_RACE containing the values that will be used to calculate birth prevalence by maternal race/ethnicity.</p>
Step #10	<p>In this merged dataset, create a new variable called PREVALENCE_RACE in which you divide the COUNT_NUMER_RACE variable by the COUNT_DENOM_RACE variable and multiply by 10,000. This will give the birth prevalence per 10,000 live births of each maternal race/ethnicity. The rows where HLHS=1 are the only meaningful observations in this dataset.</p>
Step #11	<p>Repeat Steps 7-10 for stratification by other factors.</p>
Ideas for Public health messages:	<p>This has not been adequately discussed within our group to make recommendations.</p>
Presentation	<p>Data presentation should include details about the states' ascertainment system (active, passive, passive with follow-up) as well as birth outcomes ascertained by the system.</p> <p>Confidentiality: The birth defects team has substantial concerns regarding data confidentiality, even though we have proposed a rate-based indicator/measure for presentation. Consistent with most state surveillance system's confidentiality</p>

	guidelines, we propose that any birth prevalence estimate based on less than 5 cases be suppressed.
Interpretation	<p>Birth prevalence is the preferred measure for quantifying the occurrence of birth defects in a population. Interpretation of these data will have to be made given a complete understanding of the variability between birth defects surveillance programs in terms of the three principal issues discussed in the beginning of this report.</p> <ul style="list-style-type: none">• What are the birth defects ascertained by the system?• How are the birth defects ascertained?• Among whom are the birth defects ascertained? <p>Any interpretation of a comparison of birth prevalence between states or any attempt to combine data between states to derive a birth prevalence estimate for a larger geographic area MUST fully consider the heterogeneity between systems. Such comparisons or combinations are generally considered inappropriate. In the context of specific research projects, "national" birth defects prevalence estimates have been calculated (26).</p>

Pilot Testing

Indicator/Measure Pilot Testing Birth prevalence of birth defect X per 10,000 live births Environmental Public Health Tracking					
Pilot testing within Team	<p>Following group discussion and the development of preliminary plans, Miland Palmer from UT wrote SAS code to calculate birth prevalence based on BPA codes. This initial code was shared among the group and volunteers from NY, FL, and NH (and MA (external to our working group)) volunteered to field test the code. NY, FL, and NH reported on the results of their field testing at our face-to-face meeting. UT reported on some lessons learned and other important issues to keep in mind when sharing code.</p> <p>Our overall conclusion from the process of SAS code development and indicator pilot testing was that explicit documentation is more important than perfect SAS code. Each system will need to make modifications according to their system’s specific issues. This package therefore includes the code from UT, FL, and NY, but emphasizes the documentation and guidance that is necessary to implement a consistent calculation of birth prevalence and stratified birth prevalence.</p> <p>Most of the details pertaining to specific variable field specifications will be included in Part 2 of this recommendation package, “Recommended Data Sets”.</p>				
	<table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 50%; vertical-align: top;">Utah (BPA codes)</td> <td style="width: 50%; vertical-align: top;"> <p>Utah emphasized that the metadata for each state will be essential to allow users to determine the extent to which data are comparable.</p> </td> </tr> <tr> <td style="width: 50%; vertical-align: top;">New York (BPA codes)</td> <td style="width: 50%; vertical-align: top;"> <p>Because NY uses a relational database (not a single flat file), the code was modified to accommodate this system of data storage.</p> <ul style="list-style-type: none"> • Clarified the need for better variable definition in our how-to-guide – for example, what should be the upper limit of interest for maternal age? </td> </tr> </table>	Utah (BPA codes)	<p>Utah emphasized that the metadata for each state will be essential to allow users to determine the extent to which data are comparable.</p>	New York (BPA codes)	<p>Because NY uses a relational database (not a single flat file), the code was modified to accommodate this system of data storage.</p> <ul style="list-style-type: none"> • Clarified the need for better variable definition in our how-to-guide – for example, what should be the upper limit of interest for maternal age?
Utah (BPA codes)	<p>Utah emphasized that the metadata for each state will be essential to allow users to determine the extent to which data are comparable.</p>				
New York (BPA codes)	<p>Because NY uses a relational database (not a single flat file), the code was modified to accommodate this system of data storage.</p> <ul style="list-style-type: none"> • Clarified the need for better variable definition in our how-to-guide – for example, what should be the upper limit of interest for maternal age? 				

		<ul style="list-style-type: none"> • Array size – UT had used 17 – NY needed 25 fields for defect diagnostic codes. • Calculation of rate was modified such that missing data were not dropped out.
	<p>Florida (ICD-9-CM codes)</p>	<p>Because FL uses a different coding system, they modified the UT SAS code to accommodate the ICD-9-CM coding system.</p> <ul style="list-style-type: none"> • FL needed to create a denominator dataset – with counts for all strata of interest. Initial code assumed denominator data (either count data or individual-level data) were available to the state birth defects program. • Changed missing values from . to 0 so that the rate could be calculated.
	<p>New Hampshire (BPA codes)</p>	<p>Because New Hampshire works with an off-site contractor who does not use SAS for the storage and analysis of data, the NH field test consisted of sharing the guidelines and templates for the final data tables with the contractor to determine if that information could be generated by their system. Several issues emerged:</p> <ul style="list-style-type: none"> • NH does not have a sufficient number of annual births (n=14,000) to produce annual birth prevalence estimates. They are likely to generate estimates for several years of aggregation. • Resolution – state and

		county <ul style="list-style-type: none">• Minor corrections to the codes may be necessary.• Race is not consistently captured in NH; stratification by race will not be possible.
Pilot testing outside of Team	Massachusetts (at the request of BD Team member, New Hampshire)	Awaiting results of pilot testing

Appendix A: Birth Defect Case Definitions

Prior to the calculation of birth prevalence by the birth defects surveillance system, clarification and refinement of the case definition for each of the 12 priority birth defects is necessary. Below are the relevant data the surveillance system needs in order to implement the application of the appropriate case definition. Some of these case definitions were adapted from a recent surveillance report by the Metropolitan Atlanta Congenital Defects Program (MACDP).(27)

All cases must be diagnosed on or before the infant's first birthday, although may be ascertained at any time.

Anencephaly: This category comprises anencephaly and acrania. While true acrania can result from failure of the bones of the skull to form, rather than failure of the neural tube to close, the terms are often used interchangeably in medical records, and true acrania is quite rare. If both anencephaly and spina bifida are present, the infant, fetus or child is counted in the category of anencephaly.

ICD-9-CM Codes: 740.0-740.1

740.0	Anencephalus Acrania Hemiancephaly Hemicephaly
740.1	Craniorachischisis

CDC/BPA Codes: 740.00-740.10

740.000	Absence of brain
740.010	Acrania
740.02	Anencephaly
740.030	Hernianencephaly, hemicephaly
740.080	Other
740.100	Craniorachischisis

Spina bifida (without anencephaly): This category comprises spina bifida at any level, with or without hydrocephalus or Arnold-Chiari malformation; specifically, meningocele, myelocele, myelomeningocele, lipomeningocele, lipomyelocele, and lipomyelomeningocele. This category excludes infants with anencephaly (740.00-740.10). If both anencephaly and spina bifida are present, the infant, fetus, or child is included in the anencephaly category. This category excludes spina bifida occulta (756.100).

ICD-9-CM Codes: 741.0, 741.9 without 740.0-740.10

741.0	Spina bifida with hydrocephalus Arnold-Chiari syndrome, type II Chiari malformation, type II
-------	--

- Any condition classifiable to 741.9 with any condition classifiable to 742.3
- 741.9 Spina bifida without mention of hydrocephalus
Hydromeningocele (spinal)
Hydromyelocele
Meningocele (spinal)
Meningomyelocele
Myelocele
Myelocystocele
Rachischisis
Spina bifida (aperta)
Syringomyelocele

CDC/BPA Codes: 741.00-741.99 without 740.0-740.10

- 741.000 Spina bifida aperta, any site, with hydrocephalus
- 741.010 Spina bifida cystica, any site, with hydrocephalus and Arnold-Chiari malformation
Arnold-Chiari malformation, NOS
- 741.020 Spina bifida cystica, any site, with stenosed aqueduct of Sylvius
- 741.030 Spina bifida cystica, cervical, with unspecified hydrocephalus
Spina bifida cystica, cervical, with hydrocephalus but without mention of Arnold-Chiari malformation or aqueduct stenosis
- 741.040 Spina bifida cystica, thoracic, with unspecified hydrocephalus, no mention of Arnold-Chiari
- 741.050 Spina bifida cystica, lumbar, with unspecified hydrocephalus, no mention of Arnold-Chiari
- 741.060 Spina bifida cystica, sacral, with unspecified hydrocephalus, no mention of Arnold-Chiari
- 741.070 Spina bifida of any site with hydrocephalus of late onset
- 741.080 Other spina bifida, meningocele of specified site with hydrocephalus
- 741.085 Spina bifida, meningocele, cervicothoracic, with hydrocephalus
- 741.086 Spina bifida, meningocele thoracolumbar, with hydrocephalus
- 741.087 Spina bifida, meningocele, lumbosacral with hydrocephalus
- 741.090 Spina bifida of any unspecified type with hydrocephalus
- 741.900 Spina bifida (aperta), without hydrocephalus
- 741.910 Spina bifida (cystica), cervical, without hydrocephalus
- 741.920 Spina bifida (cystica), thoracic, without hydrocephalus
- 741.930 Spina bifida (cystica), lumbar, without hydrocephalus
- 741.940 Spina bifida (cystica), sacral, without hydrocephalus
- 741.980 Spina bifida, other specified site, without hydrocephalus
Includes: cervicothoracic, thoracolumbar, lumbosacral
- 741.985 Lipomyelomeningocele
- 741.990 Spina bifida, site unspecified, without hydrocephalus (myelocoele, myelomeningocele, meningomyelocele)

Hypoplastic left heart syndrome: This category comprises hypoplastic left heart syndrome (HLHS) with or without an additional VSD.

ICD-9-CM Codes: 746.7

746.7 Hypoplastic Left Heart Syndrome: Atresia, or marked hypoplasia, of aortic orifice or valve, with hypoplasia of ascending aorta and defective development of left ventricle (with mitral valve atresia)

CDC/BPA Codes: 746.700

746.700 Hypoplastic Left Heart Syndrome

Tetralogy of Fallot: This category comprises tetralogy of Fallot (TOF), TOF with absent pulmonary valve, pulmonary atresia with a VSD (including TOF-pulmonary atresia), pulmonary atresia with a VSD and multiple aortopulmonary collaterals (also known as pseudotruncus), double-outlet right ventricle of TOF type.

ICD-9-CM Codes: 745.11, 745.2

745.11 Double Outlet Right Ventricle (only include Double Outlet Right Ventricle, TOF type)

745.2 Tetralogy of Fallot

CDC/BPA Codes: 745.200, 745.210, 745.180, 746.000+745.400, 747.310

745.200 Fallot's Tetralogy

745.210 Fallot's Pentalogy

745.180 Other specified transposition of the great vessels (only include Double Outlet Right Ventricle, TOF type)

746.000 + 745.400 Pulmonary valve atresia and ventricular septal defect

747.310 Pulmonary artery atresia and ventricular septal defect

Important to Note:

ICD-9-CM codes can not be used to reliably identify records with pulmonary atresia and ventricular septal defect (sometimes referred to as Tetralogy of Fallot with pulmonary atresia).

Double Outlet Right Ventricle (DORV) is coded using ICD9-CM code 745.11 (Double Outlet Right Ventricle) and CDC/BPA code 745.180 (Other specified transposition of the great vessels). All DORV variants are grouped under one code. If DORV cases are to be analyzed, we suggest a distinction be made among the variants: DORV-TGA type, DORV-TOF type, and DORV with ventricular septal defect (VSD). We recommend that DORV-TGA type be analyzed with transposition of the great arteries and DORV-TOF type be analyzed with Tetralogy of Fallot.

Classification of DORV variants was recently conducted on Metropolitan Atlanta Congenital Defects Program (MACDP) surveillance data. Roughly 50% of the records were classified as DORV-TGA type, 30% as DORV-VSD type,

and 20% as DORV-TOF type. This classification required MACDP to examine clinical details on the surveillance records (such as echocardiograph reports) that were collected during data abstraction. It could not have been accomplished using a computer algorithm.

If a surveillance system is unable to distinguish among the DORV variants, we recommend excluding ICD9-CM code 745.11 and CDC/BPA code 745.180 from analyses.

Transposition of the great arteries (vessels): This category comprises all types of transposition of the great arteries with concordant atrioventricular connections (dextrotransposition of the great arteries [d-TGA]) with or without ventricular septal defect or left ventricular outflow tract obstruction (pulmonary valve or infundibular stenosis), double-outlet right ventricle (DORV) with malposed great arteries, and unspecified d-TGA. The category does not include other types of DORV or corrected (L-transposition) of the great arteries.

ICD-9-CM Codes: 745.10, 745.11, 745.19

745.10 Complete transposition of the great vessels
745.11 Double Outlet Right Ventricle (only include Double Outlet Right Ventricle, TGA type)
745.19 Other transposition of the great vessels

CDC/BPA Codes: 745.10-745.19

745.100 Transposition of the great vessels, no VSD
745.110 Transposition of the great vessels, VSD
745.180 Other specified transposition of the great vessels (only include Double Outlet Right Ventricle, TGA type)
745.190 Unspecified transposition of the great vessels

Cleft lip with or without cleft palate: This category includes cleft lip with or without an associated cleft hard or soft palate, cleft alveolar ridge, and cleft gum.

ICD-9-CM Codes: 749.1, 749.2

749.1 Cleft lip
Cheiloschisis
Congenital fissure of lip
Harelip
Labium leporinum
749.10 Cleft lip, unspecified
749.11 Unilateral, complete
749.12 Unilateral, incomplete
749.13 Bilateral, complete
749.14 Bilateral, incomplete
749.2 Cleft palate with cleft lip
Cheilopalatoschisis

749.20	Cleft palate, with cleft lip, unspecified
749.21	Unilateral, complete
749.22	Unilateral, incomplete
749.23	Bilateral, complete
749.24	Bilateral, incomplete
749.25	Other combinations

CDC/BPA Codes: 749.10-749.29

749.100	Cleft lip, unilateral
749.110	Cleft lip, bilateral
749.120	Cleft lip, central
749.190	Cleft lip, NOS (no fused lip) Cleft gum
749.200	Cleft lip, unilateral, with any cleft palate
749.210	Cleft lip, bilateral, with any cleft palate
749.220	Cleft lip, central, with any cleft palate
749.290	Cleft lip, NOS, with any cleft palate

Cleft palate without cleft lip: This category comprises cleft hard or soft palate that is not associated with a cleft lip. The category does not include isolated cleft uvula (749.080) that is not associated with cleft lip or other cleft palate.

ICD-9-CM Codes: 749.0

749.0	Cleft palate, unspecified
749.01	Unilateral, complete
749.02	Unilateral, incomplete Cleft uvula
749.03	Bilateral, complete
749.04	Bilateral, incomplete

CDC/BPA Codes: 749.000-749.090

749.000	Cleft hard palate, unilateral
749.010	Cleft hard palate, bilateral
749.020	Cleft hard palate, central
749.030	Cleft hard palate, NOS
749.040	Cleft soft palate, alone, unilateral
749.050	Cleft soft palate, alone, bilateral
749.060	Cleft soft palate, alone, central
749.070	Cleft soft palate, alone, NOS
749.090	Cleft palate, NOS Palatoschisis

Hypospadias: This category comprises all degrees of hypospadias, with or without associated chordee. The category does not include epispadias, or chordee without associated hypospadias.

ICD-9-CM Codes: 752.61

752.61 Hypospadias

CDC/BPA Codes: 752.600-752.607, 752.625-752.627

- 752.600 Hypospadias (alone), NOS
- 752.605 1^o, glandular, coronal
- 752.606 2^o, penile
- 752.607 3^o, perineal, scrotal
- 752.625 Cong. chordee with 1^o, coronal hypospadias
- 752.626 Cong. chordee with 2^o, penile hypospadias
- 752.627 Cong. chordee with 3^o, perineal,scrotal hypospadias

Gastroschisis: This category comprises gastroschisis only. The category does not include omphalocele, umbilical or epigastric hernia, limb-body wall complex, or other specified and unspecified abdominal wall defects. Infants, fetuses, or children in whom the diagnosis of gastroschisis could not reliably be distinguished from omphalocele should not be included. If a surveillance system uses ICD9-CM codes, it must also use procedure codes or surgical codes, or conduct case verification/follow-up to accurately make this distinction.

ICD-9-CM Codes: 756.79

- 756.79 Other congenital anomalies of abdominal wall
 - Exomphalos
 - Gastroschisis
 - Omphalocele
 - Excludes umbilical hernia (551-553 with .1)

Additional surgical/procedure codes that can help identify cases of gastroschisis:

CDC/BPA Codes: 756.71

- 756.71 Gastroschisis

Upper limb deficiencies: This category includes complete or partial absence of the upper arm (humerus), lower arm (radius and/or ulna), wrist (carpals), hand (metacarpals), or fingers (phalanges).

ICD-9-CM Codes: 755.20-755.29

- 755.2 Reduction deformities of upper limb
- 755.20 Unspecified reduction deformity of upper limb
 - Ectromelia NOS of upper limb
 - Hemimelia NOS of upper limb
 - Shortening of arm, congenital
- 755.21 Transverse deficiency of upper limb
 - Amelia of upper limb
 - Congenital absence of:
 - fingers, all (complete or partial)
 - forearm, including hand and fingers

- upper limb, complete
Congenital amputation of upper limb
Transverse hemimelia of upper limb
- 755.22 Longitudinal deficiency of upper limb, NEC
Phocomelia NOS of upper limb
Rudimentary arm
- 755.23 Longitudinal deficiency, combined, involving humerus, radius,
and ulna (complete or incomplete)
Congenital absence of arm and forearm (complete or
incomplete) with or without metacarpal deficiency and/or
phalangeal deficiency, incomplete
Phocomelia, complete, of upper limb
- 755.24 Longitudinal deficiency, humeral, complete or partial (with or
without distal deficiencies, incomplete)
Congenital absence of humerus (with or without absence of
some [but not all] distal elements)
Proximal phocomelia of upper limb
- 755.25 Longitudinal deficiency, radioulnar, complete or partial (with or
without distal deficiencies, incomplete)
Congenital absence of radius and ulna (with or without absence
of some [but not all] distal elements)
Distal phocomelia of upper limb
- 755.26 Longitudinal deficiency, radial, complete or partial (with or
without distal deficiencies, incomplete)
Agenesis of radius
Congenital absence of radius (with or without absence of some
[but not all] distal elements)
- 755.27 Longitudinal deficiency, ulnar, complete or partial (with or
without distal deficiencies, incomplete)
Agenesis of ulna
Congenital absence of ulna (with or without absence of some
[but not all] distal elements)
- 755.28 Longitudinal deficiency, carpals or metacarpals, complete or
partial (with or without incomplete phalangeal deficiency)
- 755.29 Longitudinal deficiency, phalanges, complete or partial
Absence of finger, congenital
Aphalangia of upper limb, terminal, complete or partial
- Excludes:*
terminal deficiency of all five digits (755.21)
transverse deficiency of phalanges (755.21)

CDC/BPA Codes: 755.200-755.290

If description of the condition includes amniotic or constricting bands use additional code, 658.800 (Only use 658.800 if another reportable defect is present). Excludes shortening of upper limb (use 755.580) or hypoplasia of upper limb (use 755.585).

- 755.200 Absence of upper limb
Absent: humerus (total or partial), radius, ulna and hand
Includes: amelia of upper limb, NOS infants with rudimentary or nubbin fingers attached to stump of humerus or shoulder girdle
- 755.210 Absence of upper arm and forearm
Absent: humerus (total or partial), radius and ulna (total or partial)
Present: hand (total or partial)
Includes: phocomelia of upper limb, NOS; intercalary reduction defect of upper limb, NOS
- 755.220 Absence of forearm only or upper arm only
Absent: radius and ulna
Present: humerus, hand (total or partial)
or
Absent: humerus
Present: radius, ulna, and hand
- 755.230 Absence of forearm and hand
Absent: radius and ulna (total or partial) and hand
Includes: infants with rudimentary or nubbin fingers attached to stump of forearm or elbow
- 755.240 Absence of hand or fingers
Absent: hand or fingers (total or partial) not in conjunction with ray or long bone reduction
Includes: rudimentary or nubbin fingers;
absent individual phalanges;
absent or missing fingers, NOS
Excludes: isolated absent or hypoplastic thumb (use 755.260)
- 755.250 Split-hand malformation
Absent: central fingers (third with or without second, fourth) and metacarpals (total or partial)
Includes: monodactyly; lobster-claw hand
Excludes: isolated absent central fingers without metacarpal defects (use 755.240)
- 755.260 Preaxial longitudinal reduction defect of upper limb
Absent: radius (total or partial) and/or thumb with or without second finger (total or partial)
Includes: isolated absent or hypoplastic thumb;
radial ray defect, NOS
- 755.265 Longitudinal reduction defect of upper limb, NOS
Includes: absent forearm long bone with absent fingers, NOS
- 755.270 Postaxial longitudinal reduction defect of upper limb
Includes: isolated absent ulna (total or partial);
absent fifth with or without fourth finger (total or partial) only if ulna or fifth ± fourth metacarpal also totally or partially absent;
ulnar ray defect, NOS
- 755.280 Other specified reduction defect of upper limb
- 755.285 Transverse reduction defect of upper limb, NOS
Includes: congenital amputation of upper limb, NOS

755.290 Unspecified reduction defect of upper limb

Lower limb deficiencies: This category includes complete or partial absence of the upper leg (femur), lower leg (tibia and/or fibula), ankle (tarsals), foot (metatarsals), or toes (phalanges).

ICD-9-CM Codes: 755.30-755.39

- 755.3 Reduction deformities of lower limb
755.30 Unspecified reduction deformity of lower limb
Ectromelia NOS of lower limb
Hemimelia NOS of lower limb
Shortening of leg, congenital
755.31 Transverse deficiency of lower limb
Amelia of lower limb
Congenital absence of:
 foot
 leg, including foot and toes
 lower limb, complete
 toes, all, complete
Transverse hemimelia of lower limb
755.32 Longitudinal deficiency of lower limb, NEC
Phocomelia NOS of lower limb
755.33 Longitudinal deficiency, combined, involving femur, tibia, and fibula (complete or incomplete)
Congenital absence of thigh and (lower) leg (complete or incomplete) with or without metacarpal deficiency and/or phalangeal deficiency, incomplete
Phocomelia, complete, of lower limb
755.34 Longitudinal deficiency, femoral, complete or partial (with or without distal deficiencies, incomplete)
Congenital absence of femur (with or without absence of some [but not all] distal elements)
Proximal phocomelia of lower limb
755.35 Longitudinal deficiency, tibiofibular, complete or partial (with or without distal deficiencies, incomplete)
Congenital absence of tibia and fibula (with or without absence of some [but not all] distal elements)
Distal phocomelia of lower limb
755.36 Longitudinal deficiency, tibia, complete or partial (with or without distal deficiencies, incomplete)
Agenesis of tibia
Congenital absence of tibia (with or without absence of some [but not all] distal elements)
755.37 Longitudinal deficiency, fibular, complete or partial (with or without distal deficiencies, incomplete)
Agenesis of fibula

- 755.38 Congenital absence of fibula (with or without absence of some [but not all] distal elements)
Longitudinal deficiency, tarsals or metatarsals, complete or partial (with or without incomplete phalangeal deficiency)
- 755.39 Longitudinal deficiency, phalanges, complete or partial
Absence of toe, congenital
Aphalangia of lower limb, terminal, complete or partial
Excludes:
terminal deficiency of all five digits (755.31)
transverse deficiency of phalanges (755.31)

CDC/BPA Codes: 755.30-755.39

If description of condition includes amniotic or constricting bands use additional code, 658.800 (Only use this code if another reportable defect is present). Excludes shortening of lower limb (use 755.680) and hypoplasia of lower limb (use 755.685).

- 755.300 Absence of lower limb
Absent: femur (total or partial), tibia, fibula, and foot
Includes: amelia of lower limb, NOS infants with rudimentary or nubbin toes attached to stump of femur or pelvic girdle
- 755.310 Absence of thigh and lower leg
Absent: femur (total or partial), tibia and fibula (total or partial)
Present: foot (total or partial)
Includes: phocomelia of lower limb, NOS;
intercalary reduction defect of lower limb, NOS
- 755.320 Absence of lower leg only or femur only
Absent: tibia and fibula
Present: femur, foot (total or partial)
or
Absent: femur
Present: tibia, fibula, and foot
- 755.330 Absence of lower leg and foot
Absent: tibia and fibula (total or partial), foot
Includes: infants with rudimentary or nubbin toes attached to stump of leg or knee
- 755.340 Absence of foot or toes
Absent: foot or toes (total or partial) not in conjunction with ray or long bone reduction
Includes: rudimentary or nubbin toes;
absent individual phalanges;
absent or missing toes, NOS
Excludes: isolated absent or hypoplastic great toe (use 755.365)
- 755.350 Split-foot malformation
Absent: central toes (third with or without second, fourth) and metatarsals (total or partial)

Includes: monodactyly; lobster claw foot
Excludes: isolated absent central toes without metatarsal defects (use 755.340)

Note: preaxial lower limb reductions can occur with split-hand malformations of the upper limb and these lower limb defects should be coded 755.365.

- 755.360 Longitudinal reduction defect of lower limb, NOS
Includes: absent long bone of leg with absent toes, NOS
- 755.365 Preaxial longitudinal reduction defect of lower limb
Absent: tibia (total or partial) and/OR great toe with or without second toe (total or partial)
Includes: isolated absent or hypoplastic great toe; tibial ray defect, NOS
- 755.366 Postaxial longitudinal reduction defect of lower limb
Includes: isolated absent fibula (total or partial); absent fifth with or without fourth toe (total or partial) only if fibula or fifth ± fourth metatarsal also totally or partially absent; fibular ray defect, NOS
- 755.380 Other specified reduction defect of lower limb
- 755.385 Transverse reduction defect of lower limb, NOS
Includes: congenital amputation of lower limb, NOS
- 755.390 Unspecified reduction defect of lower limb

Trisomy 21 (Down Syndrome): These categories comprise karyotypes documenting full trisomy 21, translocation trisomy 21, or mosaic trisomy 21, and diagnoses of any of these trisomies 21 for which the karyotype is not stated in the medical record. The category does not include suspected trisomy 21 or features characteristic of Down syndrome for which the karyotype was not evaluated.

ICD-9-CM Codes: 758.0

- 758.0 Down syndrome
Mongolism
Translocation Down syndrome
Trisomy:
21 or 22
G

CDC/BPA Codes: 758.00-758.09

- 758.000 Down syndrome, karyotype trisomy 21
758.010 Down syndrome, karyotype trisomy G, NOS
758.020 Translocation trisomy – duplication of a 21
758.030 Translocation trisomy – duplication of a G, NOS
758.040 Mosaic Down syndrome
758.090 Down syndrome, NOS

Appendix B: Selected literature reviews

NOTE: This review of the literature of environmental risk factors associated with the 12 selected birth defects is not meant to be comprehensive.

Anencephaly

Although not many studies are available examining anencephaly and environmental risk factors, those that are available are suggestive of possible risks which warrant further investigation as well as surveillance. For the few studies out there, living near a TRI facility (28), maternal residence within 1,000 m of agricultural pesticide application (29), maternal exposure to nitrates in the drinking water above the MCL as well as a dose response of nitrates in the drinking water obtained from groundwater (30) were all associated with anencephaly. Paternal smoking was also found to increase risk (31). In occupational studies, maternal occupation in agriculture or industry (32), maternal occupation in agriculture alone (33), and maternal occupational exposure to electromagnetic fields (34) were suggestive for an increase in risk to anencephaly. Paternal occupations that applied pesticides (33), had exposure to solvents (35); (36), and had exposure to electromagnetic fields (34) were suggestive for an increased risk.

- Texas birth defect registry – living near a TRI facility OR=1.4 (28)
- In Shanghai China paternal smoking was associated with an increased risk (OR=2.1) (31)
- In California, maternal residences within 1,000 m of agricultural pesticide applications found a suggestive increased risk for the following physicochemical categories: amides (OR=2.2 95%CI 0.8, 5.9), benzimidazoles (OR=1.8 95%CI 0.7, 4.7) (29)
- Case-control study conducted in California examining nitrates in drinking water: maternal exposure to nitrate above the MCL of 45 mg/L OR=4.0 95%CI 1.0,15.4 when examined by water type, groundwater with increasing nitrate levels had an increased risk (5-15 mg/L OR=2.1 95%CI 1.1,4.1; 16-35 mg/L OR= 2.3 95%CI 1.1,4.5; 36-67 mg/L OR=6.9 95%CI 1.9,24.9) (30)
- Maternal occupational exposure to 0.1 μ T electromagnetic fields in Norway: >24 hours/week exposure suggestive increased risk OR=1.11 95%CI 0.35, 3.48 (34)
- Paternal occupational exposure to 0.1 μ T electromagnetic fields in Norway: 4-24 hours/week exposure OR=1.52 95%CI 1.15, 2.02 (34)
- Based on three states of the Mexican Republic, maternal occupation in industry or agriculture 3 months prior to conception to one month after conception had an increased risk OR=6.5 95%CI 1.4,29.6 (32); maternal occupation in agriculture OR= 3.67 95%CI 1.02,13.14 and paternal application of pesticides suggestive increased risk OR=2.50 95%CI 0.58,7.08 (33)
- Paternal occupation examined for Texas birth defect registry 1981-1986 paternal occupations exposed to solvents OR=2.53 95%CI 1.56,4.10

(among the solvents painters the greatest OR=3.43 95%CI 1.83-6.43)
(35)

- Meta-analysis on paternal exposure to organic solvents based on 3 studies (1 case-control, 2 cohort studies) OR=2.18 95%CI 1.52, 3.11 (36)

Spina Bifida

Although not many studies are available examining spina bifida and environmental risk factors, those that are available are either suggestive of possible risks or did not find an association. Environmental public health surveillance for spina bifida would be beneficial to further identify potential risk factors that can be examined more closely with research. In New Zealand no association was found with spraying 2,4,5-trichlorophenoxyacetic acid (37). Environmental exposures that suggest a possible increased risk of spina bifida include living near a TRI facility (28), highest tertile (40+ppb) for TTHM in drinking water (38), maternal residence within 1,000 m of agricultural pesticide application (29) or agricultural chemical exposure opportunity (39). Maternal occupational exposures that may be associated include agricultural workers, metalworkers, construction workers, industrial workers, cleaning workers, and exposure to electromagnetic fields (34, 40, 41). Paternal occupations or occupational exposures that may be associated with spina bifida include bricklayers, agriculture exposures, painters, and exposure to organic solvents (36, 40-42)

- Texas birth defect registry – living near a TRI facility OR=1.3 (28)
- New Jersey Birth Defects Registry – public monitoring data for water TTHM highest tertile (40+ ppb) POR (prevalence odds ratio) = 1.7 95% CI 0.8,3.1 (38)
- In Shanghai China paternal smoking was associated with an increased risk (OR=1.9) (31)
- No association in New Zealand with spraying of 2,4,5-trichlorophenoxyacetic acid and spina bifida (37)
- In California, maternal residences within 1,000 m of agricultural pesticide applications found an increased risk for the following physicochemical categories: amides (OR=3.3 95%CI 1.2,9.3), benzimidazoles (OR=2.7 95%CI 1.1, 6.5) (29)
- New Brunswick, Canada case-control study that developed an index to assess pesticide exposure called the agricultural chemical exposure opportunity (ACEO) index. Found fairly strong association between spina bifida without hydrocephalus and the ACEO index. (39)
- Maternal and/or Paternal occupational exposure in Spain, Sweden, and Hungary (40): suggestive increased OR's for women in agricultural occupations in Sweden (OR=1.8 95%CI 0.8, 4.2) and Spain (OR=2.2 95%CI 0.8, 5.9), in Hungary increased OR's for female metalworkers (OR=3.0 95%CI 1.1, 8.8) construction workers (OR=2.3 95%CI 1.0, 5.3) and other industrial workers (OR=1.4 95%CI 1.0,2.0). For paternal exposures, bricklayers in Spain (OR=2.8 95%CI 1.4, 5.4) other occupations in Sweden, Spain, and Hungary had some suggestive

increased odds, but nothing consistent among the three countries (e.g., paternal agriculture in Sweden and Hungary; Swedish and Spanish painters, printers, and paper and plastic workers; Hungary industrial workers and transport workers)

- Maternal occupational exposure in The Netherlands: women working in agriculture occupations increased risk (OR=3.4 95%CI 1.3,9.0) and suggestive increased risk among cleaning women (OR= 1.7 95%CI 0.9,3.4)(43)
- Maternal occupational exposure to 0.1 μ T electromagnetic fields in Norway: >24 hours/week exposure OR=2.33 95%CI 1.10, 4.94 (34)
- Paternal occupation as a painter had an increased risk with OR=3.21 among a population-based registry in British Columbia (42)
- Norwegian farmers – parents identified as farmers (mainly orchards or greenhouses) increased risk (OR=2.76 95%CI 1.07,7.13) (41)
- Meta-analysis on paternal exposure to organic solvents based on 3 studies is suggestive OR=1.59 95%CI 0.99,2.56 (36)

Neural Tube Defects (as a grouping)

- Texas birth defect registry – no association found with living proximity to hazardous waste site and NTD's (28); found elevated OR's living near TRI facility for women \geq 35 years of age (OR=2.7 95%CI 1.4, 5.0) and white non-Hispanic women (OR= 1.8 95%CI 1.1, 2.8) (28)
- New Jersey Birth Defects Registry – prevalence odds ratios of public monitoring data for water TTHM 40+ ppb concurrent with first trimester 2.1 95%CI 1.1, 4.0 (restricted to subjects with known residency at conception and to cases with isolated defects (38)
- Mexican American women along the Texas-Mexico border case-control studies: PCB exposure found not to be associated with NTD's (44); did find and increased risk among maternal occupations exposed to solvents, cleaning, and health care (45); biomarkers of exposure blood lead > 5 μ g/dL and urinary mercury >5.61 μ g/dL were suggestive with increased risk in NTD's (OR = 1.5 95%CI 0.6, 4.3; OR= 1.8 95%CI 0.8, 3.7; respectively) no relation found for urinary arsenic or urinary cadmium (46)
- Case-control study based on MACDP for years 1968-1980 Offspring of mothers employed in a nursing occupation during periconceptual period had an increased risk of anencephaly or spina bifida RR=2.00 95%CI 1.01, 4.30 (47)
- Paternal exposure to chlorophenolate wood preservatives in the sawmill industry saw an increased risk for anencephaly and spina bifida for birth cohort in British Columbia for the years 1952-1988 (obtained from abstract could not get actual numbers) (48)
- Based on the California births and fetal deaths from 1989-1991 paternal occupational groups associated with statistical increased risk in NTD's cooks, janitors and cleaners and farm workers (OR's 2.9, 2.5, 2.1, respectively); suggestive increased risk in adjusted models for spina

bifida for technical/sales/administrative (OR=1.8 95%CI 1.0, 3.3) and military (OR=3.0 95%CI 1.0,9.1) (49)

Hypoplastic Left Heart Syndrome (HLHS)

No studies were identified that examined environmental exposures and HLHS, a severe heart defect necessitating surgery for survival. However, several studies were identified examining occupational exposures. Maternal exposure to solvents and cluster living in the same area as wells maternal painting were suggestive increased risk for HLHS (50). Paternal occupations and occupational exposures that may be at increased risk of HLHS include paternal paint stripping with family history of cardiac defects (51) and exposure to solvents (52-55). Given the hints supplied by the occupational studies, further investigations and more importantly, environmental public health surveillance may be advantageous for advancing the knowledge about this birth defect.

- In the Baltimore-Washington Infant Study paternal paint stripping with family history of cardiac defects found increase in HLHS (OR=11.9; 95% CI 2.4-60.0)(51); exposure to solvents increased the risk of HLHS (RR=3.4)(52-55), exposure to solvents/degreasing agents attributable fraction 4.6% 95%CI 3.2, 6.0 (54); maternal exposure to solvents OR=3.33; 95%CI 0.96, 11.55 and cluster living in same area, mother's paintings OR= 2.73; 95%CI 1.04, 7.14 (56)
- Maternal occupational exposures to disinfectants, pesticides, dyes, lacquers or paints, and anesthetic gases were rare in a case-control study in Finland thus did not find and increased risk for HLHS (50)

Tetralogy of Fallot (ToF)

Although not many studies are available examining ToF and environmental risk factors, those that are available are suggestive of possible risks which warrant further investigation as well as environmental public health surveillance. Increasing levels of ambient carbon monoxide was found to be associated with an increased risk of ToF. Maternal exposure to hair dyes (54) and organic solvents is suggestive (55). In a rat model, nitrofen was observed to induce ToF (57).

- No effects due to caffeine (dietary) found in the National Birth Defects Prevention Study (58)
- From Texas birth defects registry; carbon monoxide found associated (OR=2.04; 95%CI 1.26, 3.29) (59)
- Infants with Down Syndrome – smoking associated with ToF OR=4.6 95%CI1.2-17.0 (60)
- Baltimore-Washington Infant Study exposure to hair dye (RR=1.6; attributable fraction 4.1% 95%CI 2.5, 5.7) (54); organic solvents (RR=2.7) (55)

- Rat model: nitrofen (a diphenyl ether herbicide) was observed to induce congenital cardiovascular anomalies at 11th day of gestation in Sprague-Dawley rats with ToF one of the common cardiovascular defects detected. (57)

Transposition of the great arteries (vessels)

In the BWIS, transposition of the great arteries was associated with organic solvents (55) and pesticides (61).

Congenital Heart Defects (as a grouping)

- Southern California from the California Birth Defects Monitoring Program, conotruncal defects in general (included ToF in case definitions) increased with 2nd month of gestation ozone exposures (OR=1.36; 95% CI 0.91, 2.03) (49)
- Increasing level of total trihalomethanes in drinking water (general grouping, major cardiac defects) (exploratory in nature, used CI's at 50%, 90% and 99%)(62)
- Study in Nova Scotia (63) and Santa Clara, CA (64) did not find an association for trihalomethanes
- Conal malformations (includes ToF, transposition, truncus arteriosus) in Finland case control study – maternal occupational exposure to dyes, lacquers or paints during 1st trimester OR=2.9; 95%CI 1.2, 7.5 however, maternal exposure to plastic raw materials, disinfectants, pesticides, microwave ovens or video display terminals at work were not associated (65)

Orofacial clefts (as a grouping)

There are two main types of orofacial clefts: cleft lip and cleft palate. Cleft lip is the congenital failure of the maxillary and median nasal processes to fuse, forming a groove or fissure in the lip; cleft palate is the congenital failure of the palate to fuse properly, forming a grooved depression or fissure in the roof of the mouth.(66) They can occur individually, together, or in conjunction with other birth defects. Due to their distinct etiology, cleft lip with cleft palate and cleft lip without cleft palate are often grouped together as **cleft lip with or without cleft palate (CLP)**. **Cleft palate without cleft lip (CP)** is classified as a separate birth defect. Twice as many boys are born with CLP as girls and the incidence of cleft lip is highest in Asian and American Indian populations and lowest in blacks.(66, 67) CP is a multifactorial condition affecting girls more frequently than boys.

OFCs have been extensively researched exploring potential demographic, genetic, and environmental risk factors. OFCs are likely caused by a combination of genetic susceptibility and environmental (nongenetic) factors. In addition, the literature about the risk factors for OFCs provides ample evidence to support gene-environment interactions as the underlying causes

of clefting.(68) Therefore, environmental public health surveillance for OFCs would be beneficial to better understand potential risk factors, especially those that can be prevented.

Advanced maternal age was identified as a risk factor in some studies (68) but other studies found no association with increasing maternal age. (69) Other maternal factors that have been extensively studied for their associations with OFCs include: smoking, alcohol consumption, medication use, use of retinoic acid, certain illnesses, socioeconomic status, stress, body mass index, exposures to environmental and occupational chemicals, exposure to agricultural pesticides, and dietary or nutritional factors (66, 68). Demographic factors that do not appear to contribute to risk for OFCs include season, geographic location, social class, parity, and paternal age.(66) However, CLP and CP occurrences were found to be correlated with increasing birth order.(70) Low socioeconomic status, when adjusted for race/ethnicity, multivitamin/mineral supplement intake, cigarette smoking and binge drinking, was not associated with increased risk of OFCs (66). However, a Scottish study found the association between OFCs and socioeconomic status (without adjusting for other factors); in this study, the pattern was stronger for CLP than for CP. (70)

Several studies have reported that maternal occupations, including hairdressing, agriculture, leather or shoe manufacturing and exposure to pesticides, lead, and aliphatic acids increased risk of OFCs (71) however, other studies failed to demonstrate a link between pesticides exposure and risk for OFCs. Occupational exposure to organic solvents (including xylene, toluene, and acetone) has been reported to increase risk for OFCs.(71, 72) However, living in proximity to hazardous waste sites does not appear to increase risks for OFCs.(73) Studies have also failed to demonstrate conclusive evidence of an effect of exposure to drinking water chlorination and chlorination by-products on clefting.(66)

- Maternal occupational exposure to glycol ethers, chemicals found in a wide range of domestic and industrial products, has been found to be statistically significantly associated with OFCs (OR=2.03; 95% CI=1.11-3.73), after adjustment for several potential confounders; risk tended to increase with exposure level, especially of an isolated cleft lip.(74) However, other researchers suggest that the current scientific evidence is insufficient to determine whether occupational exposure to glycol ethers cause human congenital malformation due to potential methodological problems with those studies.(75)
- The results of studies on the relationship between maternal exposure to organic solvents and the development of OFCs have been inconsistent. One French case-control study found a statistically significant association between OFCs and occupational exposure (during the first two months after conception) to halogenated aliphatic solvents only (OR=4.40; 95% CI=1.41-16.15), after controlling for potential confounders, such as sex of child, family history, and

- maternal epilepsy. (76) In another case-control study conducted in France (77), the risk of OFCs was associated with maternal occupational exposure to 1) oxygenated solvents (for CLP: OR=1.8; 95% CI= 1.1-2.9 and for CP: OR= 1.4; 95% CI=0.7—2.7); 2) chlorinated solvents (for CLP: OR=9.4; 95% CI=2.5-35.3 and for CP: OR=3.8; 95% CI= 0.7-20.7); and 3) petroleum solvents (for CLP: OR=3.6; 95% CI=1.5-8.8 and for CP: OR=1.2; 95% CI=0.3-4.9). In this study, the risk of OFCs increased linearly with level of exposure within the three categories of considered oxygenated solvents: aliphatic alcohols, glycol ethers, and other oxygenated solvents, including esters, ketones, and aliphatic aldehydes. The low number of cases and the problem of multiple chemical exposures require that these findings be interpreted with caution.
- A case-control study conducted in Brazil (78) has identified the following environmental risk factors for OFCs: residential proximity to industrial installations (OR=3.32; 95% CI=2.18-5.05) and the combined effect of exposure to household insecticides and urban vector control pesticides spraying (OR=5.73; 95% CI=2.51-11.28). Maternal occupation strongly associated with OFCs included domestic services (OR=2.89; 95% CI=1.76-4.86). However, the results of this study should be interpreted with caution due to the lack of any control for confounders, such as other occupational exposures.
 - Analysis of data from two population-based Californian case-control studies (73) have found little or no increased risk for maternal residence in a census tract containing a hazardous waste site for several birth defects, including clefts (OR=1.2; 95% CI=0.8-1.8), neural tube defects (OR=0.9; 95% CI=0.7-1.3), and heart defects (OR=1.3; 95% CI= 0.8-2.1), after controlling for several potential confounders. However, elevated risks were seen for neural tube defects (OR=2.1; 95% CI=0.6-7.6) and heart defects (OR=4.2; 95% CI=0.7-26.5) for maternal residence within 0.25 mile of a National Priority List site.
 - The role of maternal occupational exposure during the first trimester of pregnancy in the occurrence of OFCs was investigated among women enrolled in a multi-center European case-referent study between 1989-1992.(71) After adjusting for confounding factors, only CP was significantly associated with maternal occupation in services, such as hairdressing (OR= 5.1; 95% CI=1.0-26.0) and housekeeping (OR=2.8; 95% CI= 1.1-7.2). The results of this study suggest that the following occupational exposures are associated with OFCs: aliphatic aldehydes (OR=2.1; 95% CI=0.8-5.0) and glycol ethers (OR=1.7; 95% CI=0.9-3.3) for CLP and exposure to lead compounds (OR=4.0; 95% CI=1.3-12.2), biocides (OR=2.5; 95% CI= 1.0-6.0), antineoplastic drugs (OR=5.0; 95% CI=0.8-34.0), trichloroethylene (OR=6.7; 95% CI=0.9-49.7), and aliphatic acids (OR=6.0; 95% CI=1.5-22.8) for CP only. However, due to the small number of cases, these results must be interpreted with caution. Nevertheless, these

results identify some chemicals that have already been known or suspected as reproductive or developmental toxins.

Hypospadias

Concern regarding environmental risk factors for hypospadias has largely been confined to pesticides with potential endocrine disrupting effects.(79-91). Better international surveillance of hypospadias has been strongly encouraged.(92)

Gastroschisis

The birth prevalence of gastroschisis is increasing in the U.S. and world-wide over the past decades and this increase cannot be fully explained by a systematic shift in the classification of abdominal wall defects; the speed at which the increase has occurred might suggest environmental rather than genetic risk factors alone.(93) Prevalence of gastroschisis varies widely by geographic location, both within and between different countries(94). Two studies indicated that gastroschisis was more likely to occur in rural than urban areas(95, 96) The reason for the increasing prevalence of the defect is mainly unknown, and therefore, environmental public health surveillance for gastroschisis would be beneficial to further identify potential risk factors that can be examined more closely with future research. Further elucidation of risk factors for gastroschisis, especially preventable risk factors is therefore, warranted.

Although few studies have examined occurrence of gastroschisis and environmental risk factors, those that are available suggest risks that warrant further investigation as well as surveillance. Furthermore, incorporating genetic analyses into birth defect cluster investigations may increase our understanding of both genetic and environmental risk factors and their potential interactions for gastroschisis.

Young maternal age has consistently been reported as a risk factor for gastroschisis.(93, 97-99) Other risk factors include maternal tobacco smoking(100), use of recreational drugs, both illicit as well as maternal use of common vasoactive over-the-counter medications, including analgesics (such as aspirin) and decongestants. (60, 101, 102) In an animal model, gastroschisis resulted from exposure of pregnant mice to carbon monoxide (CO) in combination with a low protein and low zinc diet. The results of a case-control study which evaluated this model suggest that young mothers are at increased risk of having an infant with gastroschisis if they smoke and are also malnourished.(103) A change in paternity (childbearing with different fathers) has also been implicated as a risk factor suggesting that the immune system of the mother may play a role in the development of gastroschisis.(104) Maternal occupational exposure has also been linked to gastroschisis. One study has suggested a link between increased gastroschisis risk and commercial and sales work of mothers. (105)

Another study found an association between gastroschisis and maternal exposure to solvents and colorants. (101) Residence within 3 km of a hazardous waste landfill site was associated with odds ratio of borderline significance for gastroschisis (OR=3.19; 95% CI= 0.95-10.77). (106)

Upper limb deficiencies / Lower limb deficiencies

Limb deficiencies have been associated with parental exposure to pesticides (107, 108) and employment in agriculture (41, 109).

Trisomy 21

Although Trisomy 21 is a chromosomal defect, there is still debate in the scientific literature regarding a potential role of the environment (18-20, 110)

Appendix C: SAS Code Examples

UT Sample SAS Code (Miland Palmer)

```

/*****
/** SAS Code to create Prevalence rates for specific birth defects **/
/** Before executing this code be sure that your data is in the **/
/** proper format and that it contains the necessary variables and **/
/** information **/
/** The code for each defect is the same, only defect specific **/
/** information such as code ranges and labels are different **/
*****/
*****/
/**Data Specifications - Main defect data set should contain one **/
/** record for each case with variables for the following: **/
/** year of birth, gender, Mom's age, and Moms race. **/
/** This SAS code was built for summarized denominator data with **/
/** Summary counts by year for maternal age, maternal race, and **/
/** infant gender. Take care the your denominator data set and **/
/** main defect data set have the same coding system. **/
/** reference codes can be dealt with and changed in the code **/
/** below. If you have questions about this code please contact **/
/** Miland Palmer - mpalmer@utah.gov (801)257-0566 ext. 218 **/
*****/
*****/
/*Define Library EPHTN This should be the Location of your main dataset */
libname ephtn 'P:\MP_EPHTN_Prevalence\DataSets';
/*Mark all cases that should be counted in each specific defect group*/
data ephtn.maindefects (drop=i TOF1 TOF2);
set ephtn.maindefects;
array defect(17)dx1-dx17; /*check each defect for code in range*/
do i=1 to 17;
if defect(i)=746700 then
    hlhs=1; /*Set defect variable to appropriate value for
HLHS*/

if hlhs<1 then hlhs=0; /*If defect variable is blank set it to 0 for ease of
process*/
if 740010<=defect(i)<=740100 then /*Repeat each of above steps for
anencephaly*/
    anencephaly=1;

if anencephaly<1 then anencephaly=0;
if 741000<=defect(i)<=741999 then /*Repeat each of above steps for Spina
bifida*/
    SpinaBifida=1;

if anencephaly=1 or SpinaBifida<1 /*Excludes Spina bifida with anencephaly and
sets blank*/
then SpinaBifida=0; /*spina bifida variables to 0 for ease of
process*/

if defect(i)in(745200,745210,745180,747310) then /*Repeat each of above steps
for TOF*/
    TOF=1;
if defect(i)=746000 then /*Looks for 746000 pulmonary valve atresia*/
    TOF1=1;
if defect(i)=745400 then /*Looks for 745400 VSD*/
    TOF2=1;
if TOF1=1 and TOF2=1 then /*If pulmonary valve atresia code and VSD
code are present */
    TOF=1; /*Count as TOF case*/

```

Birth Defects, How to guide
Final Draft: 2008

```
if TOF<1 then TOF=0;
if 745100<=defect(i)<=745199 then /*Repeat each of above steps for TGA*/
    TGA=1;

if TGA<1 then TGA=0;
if 749000<=defect(i)<=749090 then /*Repeat each of above steps for Cleft Lip*/
    CleftLip=1;
if CleftLip<1 then CleftLip=0;

if 749000<=defect(i)<=749090 then /*Repeat each of above steps for Cleft
Palate*/
    CleftPalate=1;
if CleftPalate<1 then CleftPalate=0;
if 752600<=defect(i)<=752607 or 752625<=defect(i)<=752627 then /*Repeat each
of above steps for Hypospadias*/
    Hypospadias=1;
if Hypospadias<1 then Hypospadias=0;
if defect(i)=756710 then /*Repeat each of above steps for Gastroschisis*/
    Gastroschisis=1;
if Gastroschisis<1 then Gastroschisis=0;
if 755200<=defect(i)<=755290 then /*Repeat each of above steps for Upper
Limb*/
    UpperLimb=1;
if UpperLimb<1 then UpperLimb=0;
if 755300<=defect(i)<=755399 then /*Repeat each of above steps for Lower
Limb*/
    LowerLimb=1;
if LowerLimb<1 then LowerLimb=0;
if 758000<=defect(i)<=758099 then /*Repeat each of above steps for Trisomy21*/
    T21=1;
if T21<1 then T21=0;

end;
run;
/*****Macro to calculate specific rates*****/
/*****Overall Rate Calculation*****/
%macro Rates(dfct,defectname);
proc freq data=ephtn.maindefects; /*Obtain frequency counts of defect by year*/
tables childdobyear*&dfct /out=one; /*Output frequencies to table work.one*/
where &dfct=1;
run;
data ephtn.&dfct (keep=year count_numer); /*Rename year and count variables in
work.one to make merge with */
set work.one; /*denominator data possible output overall counts to maincount
dataset*/
rename count=count_numer;
rename childdobyear=year;
run;
/*****Rates by Race*****/
proc freq data=ephtn.maindefects; /*Obtain frequency counts of defect by year
by race*/
tables racerpt*childdobyear*&dfct/out=one; /*Output frequency counts to
work.one dataset*/
where &dfct=1;
run;
data work.one; /*Rename variables in work.one for creation of race dataset for
later merge*/
set work.one;
rename count=Count_numer_race;
rename childdobyear=year;
run;
proc sort data=work.one; /*Sort race counts by year to prepare data to be
transposed*/
```

Birth Defects, How to guide
Final Draft: 2008

```
by year;
run;
proc transpose data=work.one out=ephtn.&dfct.race (drop=_name_ _label_);
var count_numer_race; /*Transpose race counts so data can be later merged with
denominator data*/
id racerpt;          /*in one summary table with all stratified rates*/
by year;
run;
data ephtn.&dfct.race; /*Rename coded race variables so they match the
denominator data*/
set ephtn.&dfct.race;
rename _6=WhiteNumer;
rename _9=OtherNCNumer;
rename _4=HispanNumer;
rename _2=AsianNumer;
rename _1=NativeNumer;
rename _3=BlackNumer;
run;
/*****Rates by infant sex*****/
proc freq data=ephtn.maindefects; /*Obtain frequency counts of defect by year
and infant sex*/
tables gender*childdobyear*&dfct/out=one; /*Output counts to work.one
dataset*/
where &dfct=1;
run;
data work.one; /*Rename count variables for creation of sex dataset and for
later merge*/
set work.one;
rename count=Count_numer_gender;
rename childdobyear=year;
run;
proc sort data=work.one; /*Sort sex counts by year to prepare data to be
transposed*/
by year;
run;
proc transpose data=work.one out=ephtn.&dfct.sex (drop=_name_ _label_);
var count_numer_gender; /*Transpose sex data so it can be merged with
denominator data*/
id gender;          /*to form one summary table of stratified rates*/
by year;
run;
data ephtn.&dfct.sex; /*Rename coded sex variables so they match the
denominator data*/
set ephtn.&dfct.sex;
rename _1=MaleNumer;
rename _2=FemaleNumer;
rename _3=IndeterminsexNumer;
rename _4=Missingsexnumer;
run;
/*****Rates by Maternal Age*****/
proc freq data=ephtn.maindefects; /*Obtain frequency counts of defect by year
and maternal age*/
tables agegroup*childdobyear*&dfct/out=one; /*Output counts to work.one
dataset*/
where hlhs=1;
run;
data work.one; /*Rename count variables for creation of maternal age dataset and
for later merge*/
set work.one;
rename count=Count_numer_agegroup;
rename childdobyear=year;
run;
```

Birth Defects, How to guide
Final Draft: 2008

```
proc sort data=work.one; /*Sort maternal age counts by year to prepare data to
be transposed*/
by year;
run;
proc transpose data=work.one out=ephtn.&dfct.age (drop=_name_ _label_);
var count_numer_agegroup; /*Transpose maternal age data so it can be merged with
denominator data*/
id agegroup; /*to form one summary table of stratified rates*/
by year;
run;
data ephtn.&dfct.age; /*Rename coded sex variables so they match the
denominator data*/
set ephtn.&dfct.age;
rename _1=NumerLess20;
rename _2=Numer20to24;
rename _3=Numer25to29;
rename _4=Numer30to34;
rename _5=Numer35over;
rename _6=MissingAge;
run;
/*Create a dataset that has the rates for each group in a variable along with a
descriptive variable*/
/*That shows the numerator and denominator for each group. Depending on the
purpose of the output */
/*This table could be formatted or exported and presented in a more meaningful
format.*/
data ephtn.prevalence&dfct (keep=year Prevall calcprevall prevless20
calcless20
prev20to24 calc20to24 prev25to29 calc25to29 prev30to34 calc30to34
prev35over calc35over
prevMale calcprevMale prevFemale calcprevFemale prevIndet calcprevIndet
prevmissex calcprevmissex prevWhite calcprevwhite prevblack
calcprevblack
prevHisp calcprevHisp prevAsain calcprevAsain prevNative calcprevNative
prevOtherNC
calcOtherNC); /*Merge defect counts by year and stratification with
denominator data*/
merge ephtn.&dfct ephtn.&dfct.age ephtn.&dfct.race
ephtn.&dfct.sex ephtn.denom9405;
by year;
/*calculate overall prevalence per 10,000*/
prevall=((count_numer/total)*10000);
label prevall='Overall Prevalence';
calcprevall="("||trim(left(count_numer))||"/"||trim(left(total))||)";
label calcprevall='Overall Calc.';
/*Calculate age prevalence rates*/
prevless20=((numerless20/denomless20)*10000);
calcless20="("||trim(left(numerless20))||"/"||trim(left(denomless20))||)";
prev20to24=((numer20to24/denom20to24)*10000);
calc20to24="("||trim(left(numer20to24))||"/"||trim(left(denom20to24))||)";
prev25to29=((numer25to29/denom25to29)*10000);
calc25to29="("||trim(left(numer25to29))||"/"||trim(left(denom25to29))||)";
prev30to34=((numer30to34/denom30to34)*10000);
calc30to34="("||trim(left(numer30to34))||"/"||trim(left(denom30to34))||)";
prev35over=((numer35over/denom35over)*10000);
calc35over="("||trim(left(numer35over))||"/"||trim(left(denom35over))||)";
/*Calculate Sex prevalence rates*/
prevMale=((MaleNumer/MaleDenom)*10000);
calcprevMale="("||trim(left(Malenummer))||"/"||trim(left(Maledenom))||)";
prevFemale=((femalenummer/femaledenom)*10000);
calcprevFemale="("||trim(left(femalenummer))||"/"||trim(left(femaledenom))||)";
prevIndet=((IndeterminsexNumer/IndeterminsexDenom)*10000);
```


Birth Defects, How to guide
Final Draft: 2008

```
calcprevIndet="( (trim(left(IndeterminsexNumer)) || "/" || trim(left(Indeterminsex  
Denom)) || " ) )";  
prevmisssex=( (missingsexnumer/missingsexdenom) *10000 );  
calcprevmissex="( (trim(left(missingsexnumer)) || "/" || trim(left(missingsexdenom  
)) || " ) )";  
/*Calculate Race prevalence rates*/  
prevWhite=( (whitenummer/whitedenom) *10000 );  
calcprevwhite="( (trim(left(whitenummer)) || "/" || trim(left(whitedenom)) || " ) )";  
prevblack=( (blacknumer/blackdenom) *10000 );  
calcprevblack="( (trim(left(blacknumer)) || "/" || trim(left(blackdenom)) || " ) )";  
prevHisp=( (Hispsnumer/hispdenom) *10000 );  
calcprevHisp="( (trim(left(hispsnumer)) || "/" || trim(left(hispdenom)) || " ) )";  
prevAsain=( (Asainnumer/Asaindenom) *10000 );  
calcprevAsain="( (trim(left(Asainnumer)) || "/" || trim(left(Asaindenom)) || " ) )";  
prevNative=( (Nativenummer/Nativedenom) *10000 );  
calcprevNative="( (trim(left(Nativenummer)) || "/" || trim(left(Nativedenom)) || " ) )";  
prevOtherNC=( (OtherNCnumer/OtherNCdenom) *10000 );  
calcOtherNC="( (trim(left(OtherNCnumer)) || "/" || trim(left(OtherNCdenom)) || " ) )";  
run;  
/*Print the output dataset*/  
ods html path='P:\MP_EPHTN_Prevalence\Output\' body="&dfct.prevalence.html";  
title "Prevalence of &defectname";  
proc print data=ephtn.prevalence&dfct noobs ;  
var year Prevall calcprevall prevless20 calcless20  
prev20to24 calc20to24 prev25to29 calc25to29 prev30to34 calc30to34  
prev35over calc35over  
prevMale calcprevMale prevFemale calcprevFemale prevIndet calcprevIndet  
prevmisssex calcprevmissex prevWhite calcprevwhite prevblack  
calcprevblack  
prevHisp calcprevHisp prevAsain calcprevAsain prevNative calcprevNative  
prevOtherNC  
calcOtherNC;  
  
run;  
ods html close;  
%mend Rates;  
/*Call above macro for each defect %rates(DefectVariable,Defect Name)*/  
%rates(hlhs,Hypoplastic Left Heart Syndrome);  
%rates(anencephaly,Anencephaly);  
%rates(SpinaBifida,Spina bifida);  
%rates(TOF,Tetralogy of Fallot);  
%rates(TGA,Transposition of Great Arteries);  
%rates(CleftLip,Cleft lip with or without cleft palate);  
%rates(CleftPalate,Cleft palate alone);  
%rates(Hypospadias,Hypospadias);  
%rates(UpperLimb,Upper limb deficiencies);  
%rates(LowerLimb,Lower limb deficiencies);  
%rates(T21,Trisomy 21 'Down Syndrome');
```

FL Sample SAS code (Kim Hauser, Jason Salemi) – Part 1

```

/*****
FLORIDA EPHTN 1-Creating Dataset for Use in SAS
*****/

options nofmterr;

libname ephtn "C:\Birth Defects Center\EPHT-AS\SAS Code\Output";
libname bdr "C:\Birth Defects Center\FBDR Data\SAS Datasets";

/* Making a quick research dataset for use */

%let years =
1998,1999,2000,2001,2002,2003
;

%let regvars =
fbdr_id incldx1-incldx24 bthflag regflag
;

%let bvsvars =
sex race_eth mdoofday mdoobmo mdoobyr doofday doobmo doobyr
;

/* Creating the necessary datasets */

proc sql;
    create table ephtn.maindefects as
    select *
        from bdr.final_98_04 (where=(dataset in (&years))
                                keep= dataset certno &regvars)
as a
        inner join
        bdr.birth_98_03 (where=(dataset in (&years))
                                keep= dataset certno &bvsvars) as b
        on a.dataset = b.dataset and a.certno = b.certno;
quit;

/* Modifying the defect dataset to match coding system required by program */

data ephtn.maindefects;
    set ephtn.maindefects;
    mdoob= mdy(mdoobmo,mdoofday,mdoobyr);
    doob= mdy(doobmo,doofday,doobyr);
    calcMomAge= int((doob-mdoob)/365.25);
    if calcMomAge= . then agegroup= 6;
    else if calcMomAge < 20 then agegroup= 1;
    else if 20 <= calcMomAge < 25 then agegroup= 2;
    else if 25 <= calcMomAge < 30 then agegroup= 3;
    else if 30 <= calcMomAge < 35 then agegroup= 4;
    else if calcMomAge >= 35 then agegroup= 5;
    if sex= . then gender= 4;
    else if sex= 1 then gender= 1;
    else if sex=2 then gender= 2;
    if race_eth= 1 then racerpt= 6;
    else if race_eth= 2 then racerpt= 3;
    else if race_eth= 3 then racerpt= 4;
    else if race_eth= 4 then racerpt= 2;
    else if race_eth= 5 then racerpt= 1;
    else if race_eth in (6,7,.) then racerpt= 9;
    childdobyear= dataset;

```

Birth Defects, How to guide
Final Draft: 2008

```
run;

/* Determining the denominator data (live births) */

proc freq data=ephtn.maindefects;
    tables childdobyear*bthflag / out=tot (drop=percent bthflag);
    tables childdobyear*agegroup*bthflag / out=age (drop=percent bthflag );
    tables childdobyear*gender*bthflag / out=gender (drop=percent bthflag );
    tables childdobyear*racerpt*bthflag / out=racerpt (drop=percent bthflag
);
run;

proc transpose data=age out=aget (drop=_name_ _label_);
var count;
id agegroup;
by childdobyear;
run;

data aget;
set aget;
rename _1=DenomLess20;
rename _2=Denom20to24;
rename _3=Denom25to29;
rename _4=Denom30to34;
rename _5=Denom35over;
rename _6=MissingAge;
run;

proc transpose data=gender out=gendert (drop=_name_ _label_);
var count;
id gender;
by childdobyear;
run;

data gendert;
set gendert;
rename _1=MaleDenom;
rename _2=FemaleDenom;
rename _3=IndeterminsexDenom;
rename _4=MissingsexDenom;
run;

proc transpose data=racerpt out=racerptt (drop=_name_ _label_);
var count;
id racerpt;
by childdobyear;
run;

data racerptt;
set racerptt;
rename _6=WhiteDenom;
rename _9=OtherNCDenom;
rename _4=HispanDenom;
rename _2=AsianDenom;
rename _1=NativeDenom;
rename _3=BlackDenom;
run;

data ephtn.denom9803;
merge tot racerptt aget gendert;
by childdobyear;
rename childdobyear=year COUNT=TOTAL;
run;
```

Birth Defects, How to guide
Final Draft: 2008

```
/*
proc means data=ephtn.maindefects sum;
    class childdoby year agegroup gender racerpt;
    var bthflag;
    types childdoby year childdoby* (agegroup gender racerpt);
    output out=ephtn.denom9803_2 (drop=_type_ _freq_) sum=LiveBirths;
run;
*/

/* Limiting the dataset following creation of the denominator so that
   SAS processing is quicker
*/

data ephtn.maindefects;
    set ephtn.maindefects (where=(regflag=1));
run;

/*
data denom;
input year TOTAL NativeDenom AsianDenom BlackDenom HispDenom WhiteDenom
OtherNCDenom DenomLess20 Denom20to24
    Denom25to29 Denom30to34 Denom35over MissingAge MaleDenom FemaleDenom
MissingsexDenom;
datalines;
1998 195344 588 4080 43379 39509 107421 367 25598 47892 52423 42961
    26426 44 100189 95154 1
1999 196699 554 4340 44008 41452 106031 314 25243 48884 52122 43131
    27275 44 100582 96114 3
2000 203732 672 4929 46114 45823 105777 417 25494 51136 53299 45014
    28727 62 103964 99765 3
2001 204653 696 5101 45698 49528 103012 618 24463 52105 52461 46148
    29453 23 104686 99963 4
2002 204510 669 5317 44992 51521 101302 709 23375 51995 52706 46916
    29489 29 104788 99716 6
2003 211204 759 5709 46029 54753 103262 692 22965 54078 54458 48877
    30797 29 108466 102737 1
;
run;
*/
```

FL Sample SAS code (Kim Hauser, Jason Salemi) – Part 2

```

/*****
*****/
/*****
*****/
/** Purpose:
**/
/**   SAS Code to create prevalence rates for specific birth defects.
**/
/** -----
----- **/
/** Input Data Required:
**/
/**   Before executing this code be sure that:
**/
/**
**/
/**   1) You have a birth defects dataset in the proper format
**/
/**       a) Dataset needs to be 1 record per infant, w/ all dx codes
**/
/**       b) Need coded variables for maternal age/race, & infant gender
**/
/**           Age--> (1)<20; (2)20-24; (3)25-29; (4)30-34; (5)35+; (6)missing
**/
/**           Race-> (1)NatAm; (2)Asian; (3)Black; (4)Hispanic; (6)White;
(9)Other/Miss   **/
/**           Sex--> (1)Male; (2)Female; (3)Indeterminate/Ambiguous;
(4)Missing     **/
**/
/**   2) You have a denominator dataset with same coding of age/race/gender
**/
/**       a) Need one observation per year
**/
/**       b) Variables include:
**/
/**           year, TOTAL, NativeDenom, AsianDenom, BlackDenom,
**/
/**           HispDenom, WhiteDenom, OtherNCDenom, DenomLess20, Denom20to24,
**/
/**           Denom25to29, Denom30to34, Denom35over, MissingAge, MaleDenom,
**/
/**           FemaleDenom, IndeterminsexDenom, MissingsexDenom
**/
/** -----
----- **/
/** Necessary Program Modifications:
**/
/** -----
===== **/
/**   --> PLEASE ENTER YOUR PROGRAM SPECIFIC INFO IN THE %let STATEMENTS
BELOW <--   **/
/** -----
===== **/
/**   This will allow the program to be specific to your table names,
diagnosis   **/
/**   coding system, etc.
**/
/** -----
----- **/

```

Birth Defects, How to guide
Final Draft: 2008

```
/** Details:
**/
/** Created by -----> Miland Palmer -mpalmer@utah.gov (801)257-0566
ext. 218 **/
/** Modified by -----> Jason Salemi -jsalemi@health.usf.edu (813)259-8801
**/
/*****
*****/
/*****
*****/

/*****
*****/
/** BEGIN ENTERING PARAMETERS SPECIFIC TO YOUR PROGRAM
**/
/*****
*****/

/*Define Library EPHTN This should be the folder location of your main dataset
*/
libname ephtn "C:\Birth Defects Center\EPHT-AS\SAS
Code\Output";

/* Enter your state */
%let state= Florida
;

/* Enter the name of the dataset housing your diagnosis codes */
%let numerator_table_name= maindefects
;

/* Enter the name of the dataset housing your denominator data */
%let denominator_table_name= denom9803
;

/* Enter the total number of diagnosis code variables in your dataset */
%let num_dxcodes= 24
;

/* Enter a "1" if your defect variables are character(text) or "2" if numeric
*/
%let dx_type= 1
;

/* Enter the prefix for your diagnosis code variables - i.e. dx if named dx1-
dx20 */
%let dx_prefix= incldx
;

/* For the following, please enter the specific code indicative of each defect
Please place double quotes (") around each code and separate multiple
codes by a space or comma.
--> 7400 7401 (not acceptable, needs quotes)
--> "7400 7401" (not acceptable, each code needs quotes around it)
--> "7400", "7401" (acceptable)
--> "7400" "7401" (acceptable)
*/
%let hlhs_codes= "7467"
;
%let anencephaly_codes= "7400", "7401"
;
%let SpinaBifida_codes= "741"
;
```

Birth Defects, How to guide
Final Draft: 2008

```

%let TOF_codes=          "7452"
;
%let PulmAtresia_codes=  "74601"
;
%let vsd_codes=         "7454"
;
%let TGA_codes=         "7451"
;
%let CleftLip_codes=    "7491", "7492"
;
%let CleftPalate_codes= "7490"
;
%let Hypospadias_codes= "75261"
;
%let Gastroschisis_codes= "7567"
;
%let UpperLimb_codes=   "7552"
;
%let LowerLimb_codes=   "7553"
;
%let T21_codes=        "7580"
;

/*****
*****/
/**          END ENTERING PARAMETERS SPECIFIC TO YOUR PROGRAM
**/
/*****
*****/

options nofmterr;

/* Creates variables marking all cases that should be counted in each specific
defect group
--> (1)defect (0)no defect
*/
data ephnt.&numerator_table_name (drop=i TOF1 TOF2 tester);
    set ephnt.&numerator_table_name;

    /* First, converting diagnosis codes into character format for uniform
processing.
    Then, checks across all diagnosis codes for codes specific to each
defect group
    (as specified by each state at the beginning of the program).
    */

    tester= &dx_type;
    if tester= 1 then do;
        array defect1(&num_dxcodes) $ &dx_prefix.1-
&dx_prefix.&num_dxcodes;
        end;
    else if tester= 2 then do;
        array defect2(&num_dxcodes) &dx_prefix.1-
&dx_prefix.&num_dxcodes;
        end;

    do i=1 to &num_dxcodes;
        defect&dx_type(i)= compress(trim(strip(defect&dx_type(i))) || "
");
        defect&dx_type(i)= compress(defect&dx_type(i),".");

        *=====*;
        *          Hypoplastic left heart syndrome      *;

```

Birth Defects, How to guide
Final Draft: 2008

```
*=====*;
if defect&dx_type(i) in: (&hlhs_codes) then hlhs=1;
    if hlhs<1 then hlhs=0;

*=====*;
*           Anencephaly           *;
*=====*;
if defect&dx_type(i) in: (&anencephaly_codes) then anencephaly=1;
    if anencephaly<1 then anencephaly=0;

*=====*;
*           Spina bifida (w/o anencephaly)           *;
*=====*;
if defect&dx_type(i) in: (&SpinaBifida_codes) then SpinaBifida=1;
    if anencephaly=1 or SpinaBifida<1 then SpinaBifida=0;

*=====*;
*           Tetralogy of Fallot           *;
*=====*;
if defect&dx_type(i) in: (&TOF_codes) then TOF=1;
    if defect&dx_type(i) in: (&PulmAtresia_codes) then TOF1=1;
    if defect&dx_type(i) in: (&vsd_codes) then TOF2=1;
if TOF1=1 and TOF2=1 then TOF=1;
    if TOF<1 then TOF=0;

*=====*;
*           Transposition of the great arteries           *;
*=====*;
if defect&dx_type(i) in: (&TGA_codes) then TGA=1;
    if TGA<1 then TGA=0;

*=====*;
*           Cleft lip w/ and w/o cleft palate           *;
*=====*;
if defect&dx_type(i) in: (&CleftLip_codes) then CleftLip=1;
    if CleftLip<1 then CleftLip=0;

*=====*;
*           Cleft palate w/o cleft lip           *;
*=====*;
if defect&dx_type(i) in: (&CleftPalate_codes) then CleftPalate=1;
    if CleftLip=1 or CleftPalate<1 then CleftPalate=0;

*=====*;
*           Hypospadias           *;
*=====*;
if defect&dx_type(i) in: (&Hypospadias_codes) then Hypospadias=1;
    if Hypospadias<1 then Hypospadias=0;

*=====*;
*           Gastroschisis           *;
*=====*;
if defect&dx_type(i) in: (&Gastroschisis_codes) then
Gastroschisis=1;
    if Gastroschisis<1 then Gastroschisis=0;

*=====*;
*           Upper limb defects           *;
*=====*;
if defect&dx_type(i) in: (&UpperLimb_codes) then UpperLimb=1;
    if UpperLimb<1 then UpperLimb=0;

*=====*;
```


Birth Defects, How to guide
Final Draft: 2008

```

*           Lower limb defects           *;
*=====*;
if defect&dx_type(i) in: (&LowerLimb_codes) then LowerLimb=1;
    if LowerLimb<1 then LowerLimb=0;

*=====*;
*           Trisomy 21 - Down Syndrome   *;
*=====*;
if defect&dx_type(i) in: (&T21_codes) then T21=1;
    if T21<1 then T21=0;

end;
run;

/* Macro create to calculate prevalence rates (no confidence intervals) for
each defect group:
(1) overall
(2) by maternal age
(3) by maternal race
(4) by infant gender
Final prevalence datasets and web pages are created for each defect group.
*/

ods listing close; *Supresses printing to the output window;
%macro Rates(dfct,defectname);

    /* Determining counts of each defect overall and by breakdown variables
    */
    proc freq data=ephtn.&numerator_table_name;
        tables childdobyear*&dfct
            /out=&dfct (drop=percent &dfct rename=(childdobyear=year
count=NumerAll));
        tables childdobyear*racerpt*&dfct
            /out=race (drop=percent &dfct
rename=(childdobyear=year count=count_numer_race));
        tables childdobyear*gender*&dfct
            /out=gender (drop=percent &dfct
rename=(childdobyear=year count=count_numer_gender));
        tables childdobyear*agegroup*&dfct
            /out=age (drop=percent &dfct
rename=(childdobyear=year count=count_numer_agegroup));
        where &dfct=1;
    run;

    /* Modifying COUNTS BY MATERNAL RACE dataset and prepaing for merge */
    proc transpose data=race out=&dfct.race (drop=_name_ _label_);
        var count_numer_race;
        id racerpt;
        by year;
    run;

    data &dfct.race;
        set &dfct.race;
        rename _6=NumerWhite;
        rename _9=NumerOtherNC;
        rename _4=NumerHisp;
        rename _2=NumerAsian;
        rename _1=NumerNative;
        rename _3=NumerBlack;
    run;

    /* Modifying COUNTS BY INFANT GENDER dataset and prepaing for merge */
    proc transpose data=gender out=&dfct.sex (drop=_name_ _label_);
        var count_numer_gender;

```

Birth Defects, How to guide
 Final Draft: 2008

```

      id gender;
      by year;
run;

data &dfct.sex;
  set &dfct.sex;
  rename _1=NumerMale;
  rename _2=NumerFemale;
  rename _3=NumerIndet;
  rename _4=NumerMissingsex;
run;

/* Modifying COUNTS BY MATERNAL AGE dataset and prepaing for merge */
proc transpose data=age out=&dfct.age (drop=_name_ _label_);
  var count_numer_agegroup;
  id agegroup;
  by year;
run;

data &dfct.age;
  set &dfct.age;
  rename _1=NumerLess20;
  rename _2=Numer20to24;
  rename _3=Numer25to29;
  rename _4=Numer30to34;
  rename _5=Numer35over;
  rename _6=NumerMissingAge;
run;

/* Create a dataset that has the rates for each group in a variable along
with a descriptive variable */
/* that shows the numerator and denominator for each group. Depending on
the purpose of the output, */
/* this table could be formatted or exported and presented in a more
meaningful way. */

proc sort data=ephtn.&denominator_table_name;
  by year;

data prevalence&dfct
  (keep=year prevall
  prevless20 prev20to24 prev25to29 prev30to34 prev35over
prevMissingage
  prevMale prevFemale prevIndet prevMissingsex
prevWhite prevBlack prevHisp prevAsian prevNative
prevOtherNC
  calcall
  calcless20 calc20to24 calc25to29 calc30to34 calc35over
calcMissingage
  calcMale calcFemale calcIndet calcMissingsex
calcWhite calcBlack calcHisp calcAsian calcNative
calcOtherNC);

/* Merge defect counts by year and breakdown variables with
denominator data */
merge &dfct
  &dfct.age
  &dfct.race
  &dfct.sex
  ephtn.&denominator_table_name;
  by year;

```

Birth Defects, How to guide
Final Draft: 2008

```

/* For those levels where there were no cases, change the "." to a
"0" */
array miss(*) _numeric_;
do i=1 to dim(miss);
    if miss(i)= . then miss(i)= 0;
end;

length calcall
    calcless20 calc20to24 calc25to29 calc30to34 calc35over
calcmissex
    calcMale calcFemale calcIndet calccalcMissex
    calcWhite calcBlack calcHisp calcAsian calcNative
calcOtherNC $12;

/* Calculate prevalence rates, calculation fields, etc */
array prevs(*) prevall
    prevless20 prev20to24 prev25to29 prev30to34
prev35over prevmissex
    prevMale prevFemale prevIndet
calcprevMissex
    prevWhite prevBlack prevHisp prevAsian
prevNative prevOtherNC;
array calcs(*) $ calcall
    calcless20 calc20to24 calc25to29 calc30to34
calc35over calcmissex
    calcMale calcFemale calcIndet
calccalcMissex
    calcWhite calcBlack calcHisp calcAsian
calcNative calcOtherNC ;
array numer(*) Numerall
    Numerless20 Numer20to24 Numer25to29 Numer30to34
Numer35over Numermissex
    NumerMale NumerFemale NumerIndet
calcNumerMissex
    NumerWhite NumerBlack NumerHisp
NumerAsian NumerNative NumerOtherNC;
array denom(*) TOTAL
    DenomLess20 Denom20to24 Denom25to29
Denom30to34 Denom35over MissingAge
    MaleDenom FemaleDenom IndetDenom
MissingsexDenom
    WhiteDenom BlackDenom HispDenom
AsianDenom NativeDenom OtherNCDenom;

do i=1 to dim(prevs);
    prevs(i)=((numer(i)/denom(i))*10000);          *Prevalence
rate calculation;
    if prevs(i)= . then prevs(i)= 0;              *Recode rates
with 0 cases;
    prevs(i)=put(prevs(i),7.2);                  *Format rate
to 2 decimal places;

    *Create calculation variables in NUMERATOR/DENOMINATOR
format;

    calcs(i)="(|trim(left(put(numer(i),comma12.0))||"/"||trim(left(put(denom(i),comma12.0))||")");
    calcs(i)= translate(calcs(i),"0",".");        *Replace "."
in calculation field with "0";
end;
run;

/* Re-ordering variables in a more presentable way */

```

Birth Defects, How to guide
Final Draft: 2008

```
data ephtn.prevalence&dfct;
    length Defect $30;
    retain year prevall calcall prevless20 calcless20 prev20to24
calc20to24 prev25to29 calc25to29
        prev30to34 calc30to34 prev35over calc35over
prevMissingage calcMissingage prevMale calcMale
        prevFemale calcFemale prevIndet calcIndet
prevMissingsex calcMissingsex prevWhite calcWhite
        prevBlack calcBlack prevHisp calcHisp prevAsian
calcAsian prevNative calcNative prevOtherNC
        calcOtherNC;
    set prevalence&dfct;
    Defect= "&dfct";
run;

/* Print the output dataset in HTML format - for each defect */
ods html path='C:\Birth Defects Center\EPHT-AS\SAS Code\Output'
body="&dfct.prevalence.html";
    title "&state.'s Prevalence Rate of &defectname (per 10,000)";
    proc print data=ephtn.prevalence&dfct noobs ;
    run;
ods html close;
%mend Rates;

/* Call above macro for each defect --> %rates(DefectVariable,Defect Name) */

%rates(Hlhs,          Hypoplastic Left Heart Syndrome);
%rates(Anencephaly,  Anencephaly);
%rates(SpinaBifida,  Spina bifida);
%rates(TOF,          Tetralogy of Fallot);
%rates(TGA,          Transposition of Great Arteries);
%rates(CleftLip,     Cleft Lip w/ or w/o Cleft Palate);
%rates(CleftPalate, Cleft Palate w/o Cleft Lip);
%rates(Hypospadias,  Hypospadias);
%rates(UpperLimb,    Upper Limb Deficiencies);
%rates(LowerLimb,    Lower Limb Deficiencies);
%rates(T21,          Trisomy 21 [Down Syndrome]);
%rates(Gastroschisis, Gastroschisis);

/* Combine all defect-specific tables together into a combined dataset */

data ephtn.prevalance_final;
    set ephtn.prevalencehlhs          ephtn.prevalenceanencephaly
ephtn.prevalenceSpinaBifida          ephtn.prevalenceTOF
        ephtn.prevalenceTGA          ephtn.prevalenceCleftLip
ephtn.prevalenceCleftPalate          ephtn.prevalenceHypospadias
        ephtn.prevalenceUpperLimb    ephtn.prevalenceLowerLimb
ephtn.prevalenceT21                  ephtn.prevalenceGastroschisis;
run;
```

NY Sample SAS Code For Relational Databases (Phil Cross)

```
libname x "C:\Anna_Temp_";

libname sybasedb sybase
        user=axv03 password=xxxxxxx
        database=cehcmr server=sybaseHINPROD;

/*-----Select BPA's from CmCBPA and cases from Cmcases-----*/

proc sql;

        *Select BPA's---;
create table t1 as
select      distinct caseno, BPA
from        sybasedb.CmCBPA
where       BPAInd='C'
and         caseno between '1994000000' and '2005999999';

        *select cases-----;
create table t2 as
select a.caseno, substr(a.caseno,1,4) as doyear, bcno, BPA
from t1 left join sybasedb.Cmcases as a
on a.caseno=t1.caseno
where caseInd='C'
and BCNo between '000001' and '888887';
quit;

/*-----Create variable 'Malf'-----*/;
data temp1;
length malf $50;
set t2;
select;

when (BPA eq '746700') malf='hlhs';
when ('740000'<=BPA<='740100') malf='anencephaly';
when ('741000'<=BPA<='741999') malf='Spina Bifida';
when (BPA in ('745200', '745210', '746840')) malf='TOF';
when (BPA='746000') malf='TOF1';
when ('745480'<=BPA<='745490') malf='TOF2';
when ('745100'<=BPA<='745199') malf='TGA';
when ('749000'<=BPA<='74909') malf='Cleft Palate';
when ('749100'<=BPA<='749290') malf='Cleft Lip';
when ('752600'<=BPA<='752607') malf='Hypospadias';
when ('752620'<=BPA<='752627') malf='Hypospadias';
when (BPA eq '756710') malf='Gastroschisis';
when ('755200'<=BPA<='755290') malf='UpperLimb';
when ('755300'<=BPA<='755399') malf='LowerLimb';
when ('758000'<=BPA<='758099') malf='T21';
otherwise;
end;
if (malf>' ');
run;

/*-----Remove dups by caseno and malf-----*/;

proc sort nodupkey;
by caseno malf;
```

Birth Defects, How to guide
Final Draft: 2008

run;

```
/*-----If a case has anencephaly and Spina Bifida,  
      then count only once as anencephaly-----*/;
```

```
data anencep spina;  
set temp1;  
keep caseno;  
if malf='anencephaly' then output anencep;  
if malf='Spina Bifida' then output spina;  
run;
```

```
proc sql;  
create table dups as  
select caseno, count(*) as count  
from temp1  
where malf in ('anencephaly', 'Spina Bifida')  
group by caseno  
having count gt 1;  
quit;
```

```
proc sql;  
create table temp2 as  
select *  
from temp1;  
delete from temp2  
where (caseno in (select caseno from dups) and malf in ('Spina Bifida'));  
quit;
```

```
/*---If a case has TOF1 AND TOF2 then count as TOF,  
      if a case only has TOF1 or TOF2 then delete-----*/;
```

```
proc sql;  
create table dups1 as  
select caseno, count(*) as count  
from temp1  
where malf in ('TOF1', 'TOF2')  
group by caseno  
having count eq 1;  
quit;
```

```
proc sql;  
create table temp3 as  
select *  
from temp1;  
delete from temp3  
where (caseno in (select caseno from dups1) and malf in ('TOF1', 'TOF2'));  
quit;
```

```
data temp3;  
set temp3;  
if malf='TOF1' or malf='TOF2' then malf='TOF';  
run;
```

```
proc sort data=temp3 nodupkey;  
by caseno malf;  
run;
```

```
/*-----Create format for Mother's age group-----*/;
```

```
proc format;  
value $age
```

Birth Defects, How to guide
Final Draft: 2008

```
'1'-'19'='1'  
'20'-'24'='2'  
'25'-'29'='3'  
'30'-'34'='4'  
'35'-'55'='5'  
other, ' '= '6';  
run;  
/*-----Link with bc data-----*/;  
proc sql;  
create table x.temp4 as  
select a.*, b.*  
from temp3 as a, x.bc_data as b  
where a.bcno=b.bcno  
and a.dobyyear=b.doby;  
quit;  
  
/*-----Create counts for all malformations (Numerator)-----*/;  
  
proc report data=x.temp4 out=x.numer headline headskip spacing=1 nowd missing;  
columns malf dobyy racerpt agegroup gender N;  
define malf/group;  
define dobyy /group;  
define racerpt/across width=10;  
define agegroup/across format=$age. width=10;  
define gender/across width=10;  
run;  
  
/*-----Link with denominator data and calculate prevalences-----*/;  
  
proc sql;  
  
create table x.report as  
  
select malf, a.doby, round((a.n/b.n)*10000, .1) as prevall,  
  
'('||trim(left(put(a.n,5)))||'/'||trim(left(put(b.n, 10.)))||')' as  
calcprevall,  
  
round((_C3_/AsianDenom)*10000, .1) as prevAsian,  
'('||trim(left(put(_C3_,5.))||'/'||trim(left(put(AsianDenom, 10.))||')' as  
calcprevAsian,  
  
round((_C4_/HispanDenom)*10000, .1) as prevHispan,  
'('||trim(left(put(_C4_,5.))||'/'||trim(left(put(HispanDenom, 10.))||')' as  
calcprevHispan,  
  
round((_C5_/NativeDenom)*10000, .1) as prevNative,  
'('||trim(left(put(_C5_,5.))||'/'||trim(left(put(NativeDenom, 10.))||')' as  
calcprevNative,  
  
round((_C6_/WhiteDenom)*10000, .1) as prevWhite,  
'('||trim(left(put(_C6_,5.))||'/'||trim(left(put(WhiteDenom, 10.))||')' as  
calcprevWhite,  
  
round((_C7_/BlackDenom)*10000, .1) as prevBlack,  
'('||trim(left(put(_C7_,5.))||'/'||trim(left(put(BlackDenom, 10.))||')' as  
calcprevBlack,  
  
round((_C8_/otherNCDenom)*10000, .1) as prevOtherNC,  
'('||trim(left(put(_C8_,5.))||'/'||trim(left(put(OtherNcDenom, 10.))||')' as  
calcPrevOther,  
  
round((_C9_/denomless20)*10000, .1) as prevless20,
```

Birth Defects, How to guide
Final Draft: 2008

```
'('||trim(left(put(_C9_,5.))||'/'||trim(left(put(Denomless20, 10.))||'))' as
calcprevLess20,

round((_C10_/denom20to24)*10000,.1) as prev20to24,
'('||trim(left(put(_C10_,5.))||'/'||trim(left(put(Denom20to24, 10.))||'))' as
calcprev20to24,

round((_C11_/denom25to29)*10000,.1) as prev25to29,
'('||trim(left(put(_C11_,5.))||'/'||trim(left(put(Denom25to29, 10.))||'))' as
calcprev25to29,

round((_C12_/denom30to34)*10000,.1) as prev30to34,
'('||trim(left(put(_C12_,5.))||'/'||trim(left(put(Denom30to34, 10.))||'))' as
calcprev30to34,

round((_C13_/denom35over)*10000,.1) as prev35over,
'('||trim(left(put(_C13_,5.))||'/'||trim(left(put(Denom35over, 10.))||'))' as
calcprev35over,

round((_C14_/denomAgeMissing)*10000,.1) as prevAgeMissing,
'('||trim(left(put(_C14_,5.))||'/'||trim(left(put(DenomAgeMissing, 10.))||'))'
as calcprevAgeMiss,

round((_C15_/Missingsexdenom)*10000,.1) as prevmissex,
'('||trim(left(put(_C15_,5.))||'/'||trim(left(put(Missingsexdenom, 10.))||'))'
as calcprevmissex,

round((_C16_/Maledenom)*10000,.1) as prevmale,
'('||trim(left(put(_C16_,5.))||'/'||trim(left(put(Maledenom, 10.))||'))' as
calcprevmale,

round((_C17_/femaledenom)*10000,.1) as prevfemale,
'('||trim(left(put(_C17_,5.))||'/'||trim(left(put(femaledenom, 10.))||'))' as
calcprevfemale

from x.numer as a, x.denom1 as b
where a.dobyy=b.dobyy;
quit;

/*-----Print the report.Format the columns.
      Each malformation on a separate page-----*/;

ods html file='C:\Anna_temp_\report1.xls';

proc report data=x.report out=x.report1 nowd headline headskip ls=256 ps=100
split='*';
title1 'New York State Department of Health';
title2 'Congenital Malformation Registry';
title3 'Prevalence per 10,000 Live Births';
title4 "Report was created &sysdate.";

columns malf dobyy ('Overall' prevall calcprevall)

("By Mother's Race" prevAsian calcprevAsian prevHispanic calcPrevHispanic prevNative
calcprevNative prevWhite CalcPrevWhite prevBlack CalcprevBlack prevOtherNC
calcprevOther )

("By Mother's Age" prevless20 calcprevless20 prev20to24 calcprev20to24
prev25to29 calcprev25to29 prev30to34 calcprev30to34 prev35over calcprev35over
prevagemissing calcprevagemis)
```


Birth Defects, How to guide
Final Draft: 2008

```
("By Infant sex" prevmale calcprevmale prevfemale calcprevfemale prevmissex  
calcprevmissex);
```

```
define malf/ width=12 order 'Malformation';  
define prevless20/'Prv*<20' width=4;  
define prev20to24/'Prv*20*to24' width=4;  
define prevall/'Prv*All' width=4;  
define prev25to29/'Prv*25*to29' width=4;  
define prev30to34/'Prv*30*to34' width=4;  
define prev35over/'Prv*>=35' width=4;  
define prevagemissing/'Prv*Age*Miss' width=4;  
define prevmale/'Prv*Male' width=4;  
define prevfemale/'Prv*Fem' width=4;  
define prevmissex/'Prv*Miss*Sex' width=4;  
define prevAsian/'Prv*Asia' width=4;  
define prevHisp/'Prv*Hisp' width=4;  
define prevotherNc/'Prv*Other' width=4;  
define prevWhite/'Prv*Whit' width=4;  
define prevBlack/'Prv*Blac' width=4;  
define prevnative/'Prv*Nati' width=4;  
  
define calcprevalasian/'Calc*Prv*Asian' width=12;  
define calcprevhisp/'Calc*Prv*Hisp' width=12;  
define calcprevNative/'Calc*Prv*Native' width=12;  
define calcprevWhite/'Calc*Prv*White' width=12;  
define calcprevBlack/'Calc*Prv*Black' width=12;  
define calcprevother/'Calc*Prv*Other' width=12;  
define calcprevall/'Calc*Prv*All' width=12;  
define calcprevless20/'Calc*Prv*<20' width=12;  
define calcprev20to24/'Calc*Prv*20to24' width=12;  
define calcprev25to29/'Calc*Prv*25to29' width=12;  
define calcprev30to34/'Calc*Prv*30to34' width=12;  
define calcprev35over/'Calc*Prv*>=35' width=12;  
define calcprevmissex/'Calc*Prv*Missex' width=12;  
define calcprevmale/'Calc*Prv*Male' width=12;  
define calcprevfemale/'Calc*Prv*Female' width=12;  
define calcprevagemis/'Calc*Prv*AgeMiss' width=12;  
  
break after malf/page;  
run;  
  
ods html close;
```

Appendix D: Classification of birth defects in National Birth Defects Prevention Study

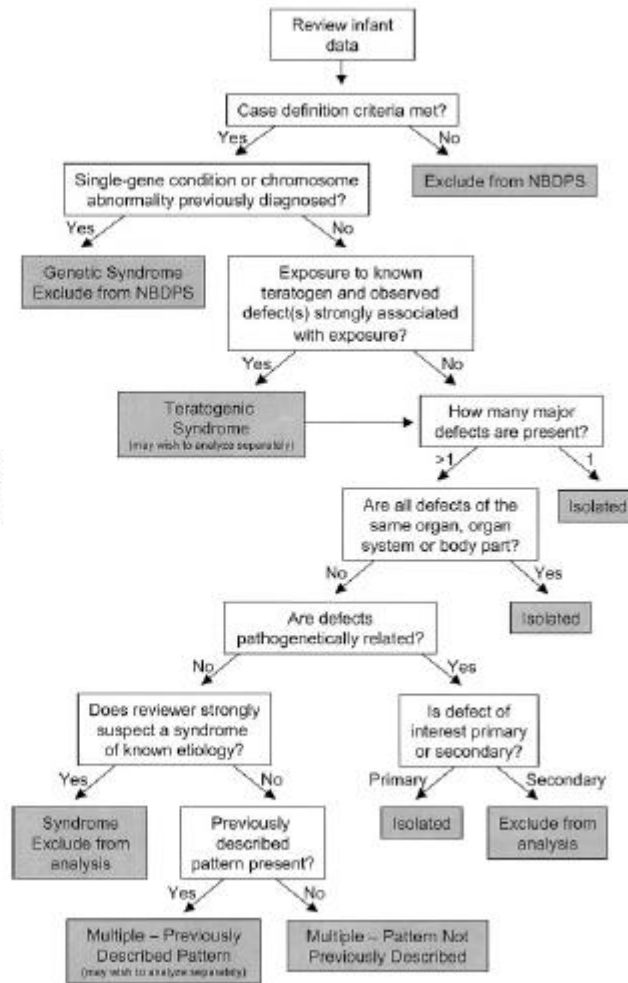


Figure 1. A summary of the process of determining whether an infant has the defect of interest as an isolated defect, as one of multiple congenital anomalies, or as component of a syndrome. Please refer to text for details on decision points.

* Source: Rasmussen et al. Guidelines for case classification for the National Birth Defects Prevention Study, **Birth Defects Res Part A**. 2003; 67:193-201

Appendix E: Template for output data set*

Year†	Overall		By Maternal Age										
			18-<20 years		20-24 years		25-29 years		30-34 years		35-39 years		40-
	Prev	Num/Denom	Prev	Num/Denom	Prev	Num/Denom	Prev	Num/Denom	Prev	Num/Denom	Prev	Num/Denom	Prev
2000													
2001													
2002													
2003													
2004													

Year†	Overall		By Maternal Race/Ethnicity							
			Non-Hispanic White		Non-Hispanic Black		Hispanic		Other Race/Ethnicity	
	Prev	Num/Denom	Prev	Num/Denom	Prev	Num/Denom	Prev	Num/Denom	Prev	Num/Denom
2000										
2001										
2002										
2003										
2004										

* See Recommendations, Part 2 "Recommended Datasets".

† Last five years of available data.

Table will continue with data by other stratification factors

References

1. National Birth Defects Prevention Network (NDBPN). Guidelines for Conducting Birth Defects Surveillance. Atlanta, GA: National Birth Defects Prevention Network, Inc.; June 2004.
2. Duke CW, Alverson CJ, Correa A. Fetal death certificates as a source of surveillance data for stillbirths with birth defects. *Public Health Rep* 2007;122(5):664-9.
3. Green HG, Nelson CJ, Gaylor DW, Holson JF. Accuracy of birth certificate data for detecting facial cleft defects in Arkansas children. *Cleft Palate J* 1979;16(2):167-70.
4. Gregg JB, Stanage WF, Johnson W. Birth certificate data: how reliable? *S D J Med* 1984;37(11):21-2.
5. Hexter AC, Harris JA. Bias in congenital malformations information from the birth certificate. *Teratology* 1991;44(2):177-80.
6. Honein MA, Paulozzi LJ. Birth defects surveillance: assessing the "gold standard". *Am J Public Health* 1999;89(8):1238-40.
7. Honein MA, Paulozzi LJ, Watkins ML. Maternal smoking and birth defects: validity of birth certificate data for effect estimation. *Public Health Rep* 2001;116(4):327-35.
8. Kirby RS. Birth certificates are an inappropriate source for identifying cases of birth defects in epidemiologic studies. *Int J Circumpolar Health* 2007;66(1):4; author reply 5-6.
9. Tairou F, De Wals P, Bastide A. Validity of death and stillbirth certificates and hospital discharge summaries for the identification of neural tube defects in Quebec City. *Chronic Dis Can* 2006;27(3):120-4.
10. Wang Y, Druschel CM, Cross PK, Hwang SA, Gensburg LJ. Problems in using birth certificate files in the capture-recapture model to estimate the completeness of case ascertainment in a population-based birth defects registry in New York State. *Birth Defects Res A Clin Mol Teratol* 2006;76(11):772-7.
11. Wang Y, Sharpe-Stimac M, Cross PK, Druschel CM, Hwang SA. Improving case ascertainment of a population-based birth defects registry in New York State using hospital discharge data. *Birth Defects Res A Clin Mol Teratol* 2005;73(10):663-8.
12. Watkins ML, Edmonds L, McClearn A, Mullins L, Mulinare J, Khoury M. The surveillance of birth defects: the usefulness of the revised US standard birth certificate. *Am J Public Health* 1996;86(5):731-4.
13. Bird TM, Hobbs CA, Cleves MA, Tilford JM, Robbins JM. National rates of birth defects among hospitalized newborns. *Birth Defects Res A Clin Mol Teratol* 2006;76(11):762-9.
14. Boulet SL, Correa-Villasenor A, Hsia J, Atrash H. Feasibility of using the national hospital discharge survey to estimate the prevalence of selected birth defects. *Birth Defects Res A Clin Mol Teratol* 2006;76(11):757-61.
15. Meyer RE, Sever LE. Towards a gold standard for national prevalence estimates of birth defects. *Birth Defects Res A Clin Mol Teratol* 2006;76(11):770-1.

16. National Birth Defects Prevention Network (NBDPN). State Birth Defects Surveillance Program Directory. *Birth Defects Res A Clin Mol Teratol* 2006;76(12):837-893.
17. Centers for Disease Control and Prevention. Metropolitan Atlanta Congenital Defects Program Defect Code List. In.
18. Halpern GJ, Stoupel EG, Barkai G, Chaki R, Legum C, Fejgin MD, et al. Solar activity cycle and the incidence of foetal chromosome abnormalities detected at prenatal diagnosis. *Int J Biometeorol* 1995;39(2):59-63.
19. Puri BK, Singh I. Season of birth in Down's syndrome. *Br J Clin Pract* 1995;49(3):129-30.
20. Tonelli M, Specchia C, Decarli A, Barlati S. De novo chromosomal abnormalities and month of conception. *Prenat Diagn* 2006;26(2):118-22.
21. Czeizel A. Definition of multiple congenital anomalies. *Acta Morphol Acad Sci Hung* 1981;29:251-259.
22. Khoury M, Botto L, Mastroiacovo P, Skjaerven R, Castilla E, Erickson J. Monitoring for multiple congenital anomalies: an international perspective. *Epidemiol Rev* 1994;16(335-350).
23. Rasmussen SA, Olney RS, Holmes LB, Lin AE, Keppler-Noreuil KM, Moore CA, et al. Guidelines for case classification for the National Birth Defects Prevention Study. *Birth Defects Research (Part A)* 2003;67:193-201.
24. Mason C, Kirby R, Sever L, Langlois P. Prevalence is the preferred measure of frequency of birth defects. *Birth Defects Res A Clin Mol Teratol* 2005;73(10):690-2.
25. Environmental Protection Agency. 2007 Report on the Environment: Science Report. 2007.
26. Canfield MA, Honein MA, Yuskiv N, Xing J, Mai CT, Collins JS, et al. National estimates and race/ethnic-specific variation of selected birth defects in the United States, 1999-2001. *Birth Defects Res A Clin Mol Teratol* 2006;76(11):747-56.
27. Correa A, Cragan JD, Kucik ME, Alverson CJ, Gilboa SM, Balakrishnan R, et al. Reporting birth defects surveillance data 1968-2003. *Birth Defects Res A Clin Mol Teratol* 2007;79(2):65-186.
28. Suarez L, Brender J, Langlois PH, Zhan FB, Moody K. Maternal Exposure to Hazardous Waste Sites and Industrial Facilities and Risk of Neural Tube Defects in Offspring. *Annals of Epidemiology* 2007;17(10):772-777.
29. Rull RP, Ritz B, Shaw GM. Neural tube defects and maternal residential proximity to agricultural pesticide applications. *Am J Epidemiol* 2006;163(8):743-53.
30. Croen LA, Todoroff K, Shaw GM. Maternal exposure to nitrate from drinking water and diet and risk for neural tube defects. *Am J Epidemiol* 2001;153(4):325-31.
31. Zhang J, Savitz DA, Schwingl PJ, Cai WW. A case-control study of paternal smoking and birth defects. *Int J Epidemiol* 1992;21(2):273-8.
32. Blanco Munoz J, Lacasana M, Borja Aburto VH, Torres Sanchez LE, Garcia Garcia AM, Lopez Carrillo L. Socioeconomic factors and the risk of anencephaly in a Mexican population: a case-control study. *Public Health Rep* 2005;120(1):39-45.

33. Lacasana M, Vazquez-Grameix H, Borja-Aburto VH, Blanco-Munoz J, Romieu I, Aguilar-Garduno C, et al. Maternal and paternal occupational exposure to agricultural work and the risk of anencephaly. *Occup Environ Med* 2006;63(10):649-56.
34. Blaasaas KG, Tynes T, Lie RT. Risk of selected birth defects by maternal residence close to power lines during pregnancy. *Occup Environ Med* 2004;61(2):174-6.
35. Brender JD, Suarez L. Paternal occupation and anencephaly. *Am J Epidemiol* 1990;131(3):517-21.
36. Logman JF, de Vries LE, Hemels ME, Khattak S, Einarson TR. Paternal organic solvent exposure and adverse pregnancy outcomes: a meta-analysis. *Am J Ind Med* 2005;47(1):37-44.
37. Hanify JA, Metcalf P, Nobbs CL, Worsley KJ. Aerial spraying of 2,4,5-T and human birth malformations: an epidemiological investigation. *Science* 1981;212(4492):349-51.
38. Klotz JB, Pynch LA. Neural tube defects and drinking water disinfection by-products. *Epidemiology* 1999;10(4):383-90.
39. White FM, Cohen FG, Sherman G, McCurdy R. Chemicals, birth defects and stillbirths in New Brunswick: associations with agricultural activity. *CMAJ* 1988;138(2):117-24.
40. Blatter BM, Roeleveld N, Bermejo E, Martinez-Frias ML, Siffel C, Czeizel AE. Spina bifida and parental occupation: results from three malformation monitoring programs in Europe. *Eur J Epidemiol* 2000;16(4):343-51.
41. Kristensen P, Irgens LM, Andersen A, Bye AS, Sundheim L. Birth defects among offspring of Norwegian farmers, 1967-1991. *Epidemiology* 1997;8(5):537-44.
42. Olshan AF, Teschke K, Baird PA. Paternal occupation and congenital anomalies in offspring. *Am J Ind Med* 1991;20(4):447-75.
43. Blatter BM, Roeleveld N, Zielhuis GA, Gabreels FJ, Verbeek AL. Maternal occupational exposure during pregnancy and the risk of spina bifida. *Occup Environ Med* 1996;53(2):80-6.
44. Suarez L, Gilani Z, Felkner M, Brender J, Henry J, Hendricks K. Exposure to polychlorinated biphenyls and risk of neural-tube defects in a Mexican American population. *Int J Occup Environ Health* 2005;11(3):233-7.
45. Brender J, Suarez L, Hendricks K, Baetz RA, Larsen R. Parental occupation and neural tube defect-affected pregnancies among Mexican Americans. *J Occup Environ Med* 2002;44(7):650-6.
46. Brender JD, Suarez L, Felkner M, Gilani Z, Stinchcomb D, Moody K, et al. Maternal exposure to arsenic, cadmium, lead, and mercury and neural tube defects in offspring. *Environmental Research* 2006;101(1):132-139.
47. Matte TD, Mulinare J, Erickson JD. Case-control study of congenital defects and parental employment in health care. *Am J Ind Med* 1993;24(1):11-23.
48. Dimich-Ward H, Hertzman C, Teschke K, Hershler R, Marion SA, Ostry A, et al. Reproductive effects of paternal exposure to chlorophenolate wood preservatives in the sawmill industry. *Scand J Work Environ Health* 1996;22(4):267-73.

49. Ritz B, Yu F, Fruin S, Chapa G, Shaw GM, Harris JA. Ambient air pollution and risk of birth defects in Southern California. *Am J Epidemiol* 2002;155(1):17-25.
50. Tikkanen J, Heinonen OP. Risk factors for hypoplastic left heart syndrome. *Teratology* 1994;50(2):112-7.
51. Correa-Villasenor A, Ferencz C, Loffredo C, Magee C. Paternal exposures and cardiovascular malformations. The Baltimore-Washington Infant Study Group. *J Expo Anal Environ Epidemiol* 1993;3 Suppl 1:173-85.
52. Loffredo CA. Epidemiology of cardiovascular malformations: prevalence and risk factors. *Am J Med Genet* 2000;97(4):319-25.
53. Botto LD, Correa A. Decreasing the burden of congenital heart anomalies: an epidemiologic evaluation of risk factors and survival. *Progress in Pediatric Cardiology* 2003;18(2):111-121.
54. Wilson PD, Loffredo CA, Correa-Villasenor A, Ferencz C. Attributable fraction for cardiac malformations. *Am J Epidemiol* 1998;148(5):414-23.
55. Ferencz C, Correa-Villasenor A, Loffredo C. Genetic and Environmental Risk Factors of Major Cardiovascular Malformations: The Baltimore-Washington Infant Study: 1981-1989. Armonk, NY: Futura Publishing Co; 1997.
56. Kuehl KS, Loffredo CA. A cluster of hypoplastic left heart malformation in Baltimore, Maryland. *Pediatr Cardiol* 2006;27(1):25-31.
57. Kim WG, Suh JW, Chi JG. Nitrofen-induced congenital malformations of the heart and great vessels in rats: an animal model. *J Pediatr Surg* 1999;34(12):1782-6.
58. Browne ML, Bell EM, Druschel CM, Gensburg LJ, Mitchell AA, Lin AE, et al. Maternal caffeine consumption and risk of cardiovascular malformations. *Birth Defects Res A Clin Mol Teratol* 2007;79(7):533-43.
59. Gilboa SM, Mendola P, Olshan AF, Langlois PH, Savitz DA, Loomis D, et al. Relation between ambient air quality and selected birth defects, seven county study, Texas, 1997-2000. *Am J Epidemiol* 2005;162(3):238-52.
60. Torfs CP, Christianson RE. Maternal risk factors and major associated defects in infants with Down syndrome. *Epidemiology* 1999;10(3):264-70.
61. Loffredo CA, Silbergeld EK, Ferencz C, Zhang J. Association of transposition of the great arteries in infants with maternal exposures to herbicides and rodenticides. *Am J Epidemiol* 2001;153(6):529-36.
62. Bove FJ, Fulcomer MC, Klotz JB, Esmart J, Dufficy EM, Savrin JE. Public drinking water contamination and birth outcomes. *Am J Epidemiol* 1995;141(9):850-62.
63. Dodds L, King W, Woolcott C, Pole J. Trihalomethanes in public water supplies and adverse birth outcomes. *Epidemiology* 1999;10(3):233-7.
64. Shaw GM, Malcoe LH, Milea A, Swan SH. Chlorinated water exposures and congenital cardiac anomalies. *Epidemiology* 1991;2(6):459-60.
65. Tikkanen J, Heinonen OP. Risk factors for conal malformations of the heart. *Eur J Epidemiol* 1992;8(1):48-57.
66. Texas Department of State Health Services. Birth Defects Risk Factor Series: Oral Clefts; 2005.
67. Dworkin P. Pediatrics. Second Edition ed: Williams & Wilkins; 1992.

68. Shaw GM. Epidemiologic characteristics of oral clefts. *Frontiers in Fetal Health* 2001;3(2):42.
69. Vieira AR, Orioli IM, Murray JC. Maternal age and oral clefts: a reappraisal. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2002;94(5):530-5.
70. Clark JD, Mossey PA, Sharp L, Little J. Socioeconomic status and orofacial clefts in Scotland, 1989 to 1998. *Cleft Palate Craniofac J* 2003;40(5):481-5.
71. Lorente C, Cordier S, Bergeret A, De Walle HE, Goujard J, Ayme S, et al. Maternal occupational risk factors for oral clefts. Occupational Exposure and Congenital Malformation Working Group. *Scand J Work Environ Health* 2000;26(2):137-45.
72. Wyszynski DF, Beaty TH. Review of the role of potential teratogens in the origin of human nonsyndromic oral clefts. *Teratology* 1996;53(5):309-17.
73. Croen LA, Shaw GM, Sanbonmatsu L, Selvin S, Buffler PA. Maternal residential proximity to hazardous waste sites and risk for selected congenital malformations. *Epidemiology* 1997;8(4):347-54.
74. Cordier S, Bergeret A, Goujard J, Ha MC, Ayme S, Bianchi F, et al. Congenital malformation and maternal occupational exposure to glycol ethers. Occupational Exposure and Congenital Malformations Working Group. *Epidemiology* 1997;8(4):355-63.
75. Maldonado G, Delzell E, Tyl RW, Sever LE. Occupational exposure to glycol ethers and human congenital malformations. *Int Arch Occup Environ Health* 2003;76(6):405-23.
76. Laumon B, Martin JL, Collet P, Bertucat I, Verney MP, Robert E. Exposure to organic solvents during pregnancy and oral clefts: a case-control study. *Reprod Toxicol* 1996;10(1):15-9.
77. Chevrier C, Dananche B, Bahuau M, Nelva A, Herman C, Francannet C, et al. Occupational exposure to organic solvent mixtures during pregnancy and the risk of non-syndromic oral clefts. *Occup Environ Med* 2006;63(9):617-23.
78. Leite IC, Paumgartten FJ, Koifman S. Chemical exposure during pregnancy and oral clefts in newborns. *Cad Saude Publica* 2002;18(1):17-31.
79. Bhatia R, Shiau R, Petreas M, Weintraub JM, Farhang L, Eskenazi B. Organochlorine pesticides and male genital anomalies in the child health and development studies. *Environ Health Perspect* 2005;113(2):220-4.
80. Brouwers MM, Feitz WF, Roelofs LA, Kiemeneij LA, de Gier RP, Roeleveld N. Risk factors for hypospadias. *Eur J Pediatr* 2007;166(7):671-8.
81. Carbone P, Giordano F, Nori F, Mantovani A, Taruscio D, Lauria L, et al. Cryptorchidism and hypospadias in the Sicilian district of Ragusa and the use of pesticides. *Reprod Toxicol* 2006;22(1):8-12.
82. Longnecker MP, Klebanoff MA, Brock JW, Zhou H, Gray KA, Needham LL, et al. Maternal serum level of 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene and risk of cryptorchidism, hypospadias, and polythelia among male offspring. *Am J Epidemiol* 2002;155(4):313-22.

83. Pierik FH, Burdorf A, Deddens JA, Juttmann RE, Weber RF. Maternal and paternal risk factors for cryptorchidism and hypospadias: a case-control study in newborn boys. *Environ Health Perspect* 2004;112(15):1570-6.
84. Recio R, Robbins WA, Borja-Aburto V, Moran-Martinez J, Froines JR, Hernandez RM, et al. Organophosphorous pesticide exposure increases the frequency of sperm sex null aneuploidy. *Environ Health Perspect* 2001;109(12):1237-40.
85. Safe SH. Endocrine disruptors and human health--is there a problem? An update. *Environ Health Perspect* 2000;108(6):487-93.
86. Salazar-Garcia F, Gallardo-Diaz E, Ceron-Mireles P, Loomis D, Borja-Aburto VH. Reproductive effects of occupational DDT exposure among male malaria control workers. *Environ Health Perspect* 2004;112(5):542-7.
87. Sanborn M, Kerr KJ, Sanin LH, Cole DC, Bassil KL, Vakil C. Non-cancer health effects of pesticides: systematic review and implications for family doctors. *Can Fam Physician* 2007;53(10):1712-20.
88. Taxvig C, Hass U, Axelstad M, Dalgaard M, Boberg J, Andeasen HR, et al. Endocrine-disrupting activities in vivo of the fungicides tebuconazole and epoxiconazole. *Toxicol Sci* 2007;100(2):464-73.
89. Weidner IS, Moller H, Jensen TK, Skakkebaek NE. Cryptorchidism and hypospadias in sons of gardeners and farmers. *Environ Health Perspect* 1998;106(12):793-6.
90. Weselak M, Arbuckle TE, Foster W. Pesticide exposures and developmental outcomes: the epidemiological evidence. *J Toxicol Environ Health B Crit Rev* 2007;10(1-2):41-80.
91. Carbone P, Giordano F, Nori F, Mantovani A, Taruscio D, Lauria L, et al. The possible role of endocrine disrupting chemicals in the aetiology of cryptorchidism and hypospadias: a population-based case-control study in rural Sicily. *Int J Androl* 2007;30(1):3-13.
92. Dolk H, Vrijheid M, Scott JE, Addor MC, Botting B, de Vigan C, et al. Toward the effective surveillance of hypospadias. *Environ Health Perspect* 2004;112(3):398-402.
93. DiTanna GL, Rosano A, Mastroiacovo P. Prevalence of gastroschisis at birth: retrospective study. *BMJ* 2002;327:1389-1390.
94. Texas Department of State Health Services. Birth Defects Risk Factor Series: Gastroschisis; 2005.
95. Forrester M, Merz R. Epidemiology of abdominal wall defects, Hawaii, 1986-1997. *Teratology* 1999;60(3):117-123.
96. Salihu HM, Pierre-Louis BJ, Druschel CM, RS K. Omphalocele and gastroschisis in the State of New York, 1992-1999. *Birth Defects Res A Clin Mol Teratol* 2005;67(9):630-636.
97. Calzolari E, Bianchi F, Dolk H, Milan M. Omphalocele and gastroschisis in Europe: a survey of 3 million births 1980-1990. EUROCAT Working Group. *Am J Med Genet* 1995;58(2):187-94.
98. Torfs CP, Velie EM, Oechsli FW, Bateson TF, Curry CJ. A population-based study of gastroschisis: demographic, pregnancy, and lifestyle risk factors. *Teratology* 1994;50(1):44-53.

99. Williams LJ, Kucik JE, Alverson CJ, Olney RS, Correa A. Epidemiology of gastroschisis in metropolitan Atlanta, 1968 through 2000. *Birth Defects Res A Clin Mol Teratol* 2005;73(3):177-83.
100. Goldbaum G, Daling J, Milham S. Risk factors for gastroschisis. *Teratology* 1990;42(4):397-403.
101. Torfs CP, Katz EA, Bateson TF, Lam PK, Curry CJ. Maternal medications and environmental exposures as risk factors for gastroschisis. *Teratology* 1996;54(2):84-92.
102. Werler MM, Sheehan JE, Mitchell AA. Maternal medication use and risks of gastroschisis and small intestinal atresia. *Am J Epidemiol* 2002;155(1):26-31.
103. Lam PK, Torfs CP. Interaction between maternal smoking and malnutrition in infant risk of gastroschisis. *Birth Defects Res A Clin Mol Teratol* 2006;76(3):182-6.
104. Chambers CD, Chen BH, Kalla J, Jernigan L, Jones KL. Novel risk factor in gastroschisis: change in paternity. *Am J Med Genet* 2007;143(7):653-659.
105. Hemminki K, Saloniemi I, Kyyronen P, Kekomaki M. Gastroschisis and omphalocele in Finland in the 1970s: prevalence at birth and its correlates. *J Epidemiol Community Health* 1982;36(4):289-93.
106. Dolk H, Vrijheid M, Armstrong B, Abramsky L, Bianchi F, Garne E, et al. Risk of congenital anomalies near hazardous-waste landfill sites in Europe: the EUROHAZCON study. *Lancet* 1998;352(9126):423-7.
107. Shaw GM, Wasserman CR, O'Malley CD, Nelson V, Jackson RJ. Maternal pesticide exposure from multiple sources and selected congenital anomalies. *Epidemiology* 1999;10(1):60-6.
108. Lin S, Marshall EG, Davidson GK. Potential parental exposure to pesticides and limb reduction defects. *Scand J Work Environ Health* 1994;20(3):166-79.
109. Engel LS, O'Meara ES, Schwartz SM. Maternal occupation in agriculture and risk of limb defects in Washington State, 1980-1993. *Scand J Work Environ Health* 2000;26(3):193-8.
110. Christianson RE, Sherman SL, Torfs CP. Maternal meiosis II nondisjunction in trisomy 21 is associated with maternal low socioeconomic status. *Genet Med* 2004;6(6):487-94.

Content Domain: Birth Defects

**Recommendations for
Nationally Consistent Data and Measures**

***Abbreviated* Part 2: Recommended Data Sets**

Version 4.0 of the Birth Defects Recommended Data Sets, Part 2 data dictionaries include information on the optionality and XML schema source for each data field.

ENVIRONMENTAL PUBLIC HEALTH TRACKING

BIRTH DEFECTS

Changes Incorporated in Versions 3.2 and 3.2.1

Field Name	Dictionaries Affected	Description
COUNTY	BD1 – BD3	The missing value code 'U' is now accepted for this field.
COUNTY	BD1 – BD3	The field length was expanded from 3 to 5 for reasons of compatibility with the existing XML schemas. The state FIPS code is now redundantly embedded in the county FIPS code.
MATERNAL RACE GROUP	BD2 – BD4	The code 'NS' (not submitted) has been explicitly added to the allowable codes for this field. This code is intended to allow the maternal race field to be selectively collapsed, for example, when maternal ethnicity is Hispanic. This code is not intended to indicate unknown race.
LIVE BIRTHS WITH DEFECT	BD1 – BD4	The unknown value code -999 is now accepted for this field, to cover situations in which a birth count is known for a demographic classification, but a corresponding birth defect count was not determined.
LIVE BIRTHS + FETAL DEATHS + TERMINATIONS WITH DEFECT	BD1 – BD4	The unknown value code -999 is now accepted for this field, to cover situations in which a birth count is known for a demographic classification, but a corresponding birth defect count was not determined.
LIVE BIRTHS + FETAL DEATHS + TERMINATIONS WITH DEFECT	BD1 – BD4	This field is now optional, in order to accommodate data submissions from states that conduct surveillance for live births only.

ENVIRONMENTAL PUBLIC HEALTH TRACKING

AGGREGATE DATA SET SUMMARY

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Characteristic	Description
Data Source	State Birth Defects Registries
Purpose	This data set will be used to calculate prevalence measures for birth defects as described in the Part 1 package, for use on the national public portal.
Geographic Level	The smallest geographic unit to be represented in this data set is the county.
Restrictions	This is a restricted access data set. Data will be displayed via the national public portal only when sufficient conditions have been met to protect data privacy. Only registered users will have direct access to this data set via the national secure portal.

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
Reference to a header document (MCN, etc.)						Required	ephtn-ph-BD1.xsd <xsd:element name="Header" type="headerType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
BIRTH DEFECT	Birth defect category.	Integer	1 = Anencephaly 2 = Spina bifida (w/o anencephaly) 3 = Hypoplastic left heart syndrome 4 = Tetralogy of Fallot 5 = Transposition of the great arteries (vessels) 6 = Cleft lip with or w/o cleft palate 7 = Cleft palate w/o cleft lip 8 = Hypospadias 9 = Gastroschisis 10 = Upper limb deficiencies 11 = Lower limb deficiencies 12 = Trisomy 21	1 - 12	2	Required	ephtn-ph-BD1.xsd <xsd:element name="BirthDefect" type="BirthDefectType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
STATE	State FIPS code.	String	FIPS	A valid state FIPS code.	2	Not specified in schema	ephtn-core.xsd <xsd:element name="StateFIPS Code" type="statecodeType">
COUNTY	County FIPS code over which birth defect cases and underlying birth populations are counted.	String	FIPS U = Unknown	A valid county FIPS code for the state, or 'U'.	5	Required	ephtn-ph-BD1.xsd <xsd:element name="Countycode" type="unknownCountyCodeType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
START DATE ¹	Date on which data aggregation begins.	Date	yyyymmdd		8	Required	ephtn-ph-BD1.xsd <xsd:element name="StartDate" type="NCDMdate" minOccurs="1" maxOccurs="1" />
END DATE ¹	Date on which data aggregation ends.	Date	yyyymmdd		8	Required	ephtn-ph-BD1.xsd <xsd:element name="EndDate" type="NCDMdate" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING**DATA DICTIONARY FOR AGGREGATE DATA****BIRTH DEFECTS****Birth Defect Counts, by Maternal Age and Infant Sex**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
SURVEILLANCE TYPE	State birth defects surveillance system uses only active case finding procedures, only passive reporting, or a combination of active and passive methods.	Text	A = active P = passive PF = passive with follow-up	A, P, PF	2	Required	ephtn-ph-BD1.xsd <xsd:element name="Surveillance" type="SurveillanceType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
ASCERTAINMENT	State birth defects surveillance system routinely ascertains cases of birth defects among live births only, or also covers other outcomes of pregnancy (fetal deaths and/or terminations).	Text	L = live births only LF = live births + fetal deaths LT = live births + pregnancy terminations LFT = live births + fetal deaths + pregnancy terminations	L, LF, LT, LFT	3	Required	ephtn-ph-BD1.xsd <xsd:element name="Ascertainment" type="AscertainmentType" minOccurs="1" maxOccurs="1" />
CODE SET	Indicates the standard under which birth defects cases are coded and classified.	Integer	1 = ICD-9-CM 2 = ICD-9-CM, CDC coding based on BPA	1, 2	1	Required	ephtn-ph-BD1.xsd <xsd:element name="CodeSet" type="CodeSetType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
MATERNAL AGE GROUP	Five-year maternal age intervals for which cases and underlying birth populations are counted.	Integer	1 = <20 years 2 = 20-24 years 3 = 25-29 4 = 30-34 5 = 35-39 6 = ≥40 9 = Unknown	1 - 6, 9	1	Required	ephtn-ph-BD1.xsd <xsd:element name="MaternalAgeGroup" type="MaternalAgeType" minOccurs="1" maxOccurs="1" />
INFANT SEX	Infant sex for which cases and underlying birth populations are counted.	Text	M = Male F = Female U = Unknown	M, F, U	1	Required	ephtn-ph-BD1.xsd <xsd:element name="InfantSex" type="InfantSexType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
TOTAL LIVE BIRTHS ²	Total number of live births.	Integer		0 to nnnnnnn	7	Required	ephtn-ph-bd-core.xsd <xsd:element name="TLB" type="sevenDigit NumberType" minOccurs="1" maxOccurs="1">

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
LIVE BIRTHS WITH BIRTH DEFECT	Number of cases of birth defect among live births only.	Integer	-999 = Unknown	0 to nnnn -999 Note: The missing value code -999 is appropriate when a birth count is known for a demographic classification, but the birth defect count is unknown.	4	Required	ephtn-ph-bd-core.xsd <xsd:element name="LBWBD" type="missingFourDigitNumberType" minOccurs="1" maxOccurs="1">

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
LIVE BIRTHS + FETAL DEATHS + TERMINATIONS WITH DEFECT	Number of cases of birth defect among live births plus fetal deaths and/or pregnancy terminations, in those states that ascertain cases among fetal deaths and/or terminations. (optional)	Integer	-999 = Unknown	0 to nnnn -999 Note: The missing value code -999 is appropriate when a birth count is known for a demographic classification, but the birth defect count is unknown.	4	Optional	ephtn-ph-bd-core.xsd <xsd:element name="LBFDTWD" type="missingFourDigitNumberType" minOccurs="0" maxOccurs="1">

- 1 Start dates and end dates will allow for aggregation of more than one year of data when necessary, as well as part-year aggregates.

-
- 2 This is the denominator for “prevalence” calculations. This data may not need to be duplicated as it may be available through some of the vital records datasets as part of EPHT.

ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA SET FROM WHICH MEASURES MAY BE DERIVED
BIRTH DEFECTS
Birth Defect Counts, by Maternal Age and Infant Sex

Referred to as Excel file: BD1.xls in the following discussion

Notes on Table Shell

1. Prevalence measures (and corresponding confidence intervals) can be directly calculated from the proposed data set, at both the level at which the data are provided (the finest level of breakdown) or at higher levels of aggregation (for example, all maternal ages). Because prevalence measures at higher levels require that numerators and denominators be calculated first (the prevalence measures cannot be directly aggregated) there is limited utility in including pre-calculated measures in this data set.
2. For each birth defect there is an exhaustive cross-classification between maternal five-year age group and infant sex, with $7 \times 3 = 21$ mutually exclusive classifications. It should be possible to assign each specific birth defect case to exactly one maternal age group \times infant sex classification. An example of the complete cross-classification structure appears in lines 3-23 of the table shell (for anencephaly).

ENVIRONMENTAL PUBLIC HEALTH TRACKING

AGGREGATE DATA SET SUMMARY

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Characteristic	Description
Data Source	State Birth Defects Registries
Purpose	This data set will be used to calculate prevalence measures for birth defects as described in the Part 1 package, for use on the national public portal.
Geographic Level	The smallest geographic unit to be represented in this data set is the county.
Restrictions	This is a restricted access data set. Data will be displayed via the national public portal only when sufficient conditions have been met to protect data privacy. Only registered users will have direct access to this data set via the national secure portal.

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
------------	-------------------	-----------	-------------	--------------	--------------	-------------	---------------

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
BIRTH DEFECT	Birth defect category.	Integer	1 = Anencephaly 2 = Spina bifida (w/o anencephaly) 3 = Hypoplastic left heart syndrome 4 = Tetralogy of Fallot 5 = Transposition of the great arteries (vessels) 6 = Cleft lip with or w/o cleft palate 7 = Cleft palate w/o cleft lip 8 = Hypospadias 9 = Gastroschisis 10 = Upper limb deficiencies 11 = Lower limb deficiencies 12 = Trisomy 21	1 - 12	2	Required	ephtn-ph-BD2.xsd <xsd:element name="BirthDefect" type="BirthDefectType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
STATE	State FIPS code.	String	FIPS	A valid state FIPS code.	2	Not specified in schema	ephtn-core.xsd <xsd:element name="StateFIPS Code" type="statecodeType">
COUNTY	County FIPS code over which birth defect cases and underlying birth populations are counted.	String	FIPS U = Unknown	A valid county FIPS code for the state, or 'U'.	5	Required	ephtn-ph-BD2.xsd <xsd:element name="Countycode" type="unknownCountyCodeType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
START DATE ¹	Date on which data aggregation begins.	Date	yyyymmdd		8	Required	ephtn-ph-BD2.xsd <xsd:element name="StartDate" type="NCDMdate" minOccurs="1" maxOccurs="1" />
END DATE ¹	Date on which data aggregation ends.	Date	yyyymmdd		8	Required	ephtn-ph-BD2.xsd <xsd:element name="EndDate" type="NCDMdate" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
SURVEILLANCE TYPE	State birth defects surveillance system uses only active case finding procedures, only passive reporting, or a combination of active and passive methods.	Text	A = active P = passive PF = passive with follow-up	A, P, PF	2	Required	ephtn-ph-BD2.xsd <xsd:element name="Surveillance" type="SurveillanceType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
ASCERTAINMENT	State birth defects surveillance system routinely ascertains cases of birth defects among live births only, or also covers other outcomes of pregnancy (fetal deaths and/or terminations).	Text	L = live births only LF = live births + fetal deaths LT = live births + pregnancy terminations LFT = live births + fetal deaths + pregnancy terminations	L, LF, LT, LFT	3	Required	ephntn-ph-BD2.xsd <xsd:element name="Ascertainment" type="AscertainmentType" minOccurs="1" maxOccurs="1" />
CODE SET	Indicates the standard under which birth defects cases are coded and classified.	Integer	1 = ICD-9-CM 2 = ICD-9-CM, CDC coding based on BPA	1, 2	1	Required	ephntn-ph-BD2.xsd <xsd:element name="CodeSet" type="CodeSetType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
MATERNAL ETHNICITY	Maternal Hispanic ethnicity for which cases and underlying birth populations are counted.	Text	H = Hispanic NH = non-Hispanic U = Unknown	H, NH, U	2	Required	ephtn-ph-BD2.xsd <xsd:element name="MaternalEthnicity" type="ethnicityType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
MATERNAL RACE GROUP	Maternal race group for which cases and underlying birth populations are counted.	Text	W = White B = Black O = Other U = Unknown NS = Not submitted	W, B, O, U, NS Note: The code 'NS' is intended to allow the maternal race field to be selectively collapsed, for example, when maternal ethnicity is Hispanic. This code is not intended to indicate unknown race.	2	Required	ephntn-ph-BD2.xsd <xsd:element name="MaternalRace" type="MaternalRaceType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
INFANT SEX	Infant sex for which cases and underlying birth populations are counted.	Text	M = Male F = Female U = Unknown	M, F, U	1	Required	ephtn-ph-BD2.xsd <xsd:element name="InfantSex" type="InfantSexType" minOccurs="1" maxOccurs="1" />
TOTAL LIVE BIRTHS ²	Total number of live births.	Integer		0 to nnnnnnn	7	Required	ephtn-ph-bd-core.xsd <xsd:element name="TLB" type="sevenDigitNumberType" minOccurs="1" maxOccurs="1">

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
LIVE BIRTHS WITH BIRTH DEFECT	Number of cases of birth defect among live births only.	Integer	-999 = Unknown	0 to nnnn -999 Note: The missing value code -999 is appropriate when a birth count is known for a demographic classification, but the birth defect count is unknown.	4	Required	ephtn-ph-bd-core.xsd <xsd:element name="LBWBD" type="missingFourDigitNumberType" minOccurs="1" maxOccurs="1">

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
LIVE BIRTHS + FETAL DEATHS + TERMINATIONS WITH DEFECT	Number of cases of birth defect among live births plus fetal deaths and/or pregnancy terminations, in those states that ascertain cases among fetal deaths and/or terminations. (optional)	Integer	-999 = Unknown	0 to nnnn -999 Note: The missing value code -999 is appropriate when a birth count is known for a demographic classification, but the birth defect count is unknown.	4	Optional	ephntn-ph-bd-core.xsd <xsd:element name="LBFDTWD" type="missingFourDigitNumberType" minOccurs="0" maxOccurs="1">

- 1 Start dates and end dates will allow for aggregation of more than one year of data when necessary, as well as part-year aggregates.

-
- 2 This is the denominator for “prevalence” calculations. This data may not need to be duplicated as it may be available through some of the vital records datasets as part of EPHT.

ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA SET FROM WHICH MEASURES MAY BE DERIVED
BIRTH DEFECTS

Birth Defect Counts, by Maternal Ethnicity/Race and Infant Sex

Referred to as Excel file: BD2.xls in the following discussion

Notes on Table Shell

1. Prevalence measures (and corresponding confidence intervals) can be directly calculated from the proposed data set, at both the level at which the data are provided (the finest level of breakdown) or at higher levels of aggregation (for example, all maternal ethnicity/race groups). Because prevalence measures at higher levels require that numerators and denominators be calculated first (the prevalence measures cannot be directly aggregated) there is limited utility in including pre-calculated measures in this data set.
2. For each birth defect there is an exhaustive cross-classification between maternal ethnicity/race and infant sex. A *fully expanded* ethnicity/race cross-classification structure would contain 3 ethnicity classes (H, NH, unknown) by 4 race classes (W, B, Other, unknown) for a total of $3 \times 4 = 12$ mutually exclusive classifications. However, because cases with maternal Hispanic ethnicity will not be simultaneously tabulated by race in this data set, the segment of the maternal ethnicity/race cross-classification structure covering these cases is collapsed from 4 classifications to just 1. The partially collapsed maternal ethnicity/race cross-classification structure thus has 9 mutually exclusive classifications. Since infant sex has 3 classes (M, F, unknown) the partially collapsed maternal ethnicity/race \times infant sex cross-classification structure has $9 \times 3 = 27$ mutually exclusive classifications. The partially collapsed structure remains exhaustive; it should be possible to assign each specific birth defect case to exactly one maternal ethnicity/race \times infant sex classification. An example of the partially collapsed cross-classification structure appears in lines 3-29 of the table shell (for anencephaly).

The maternal ethnicity/race data structure can be further collapsed to accommodate varying needs, but caution should be taken to ensure that

the cross-classification structure remains exhaustive and mutually exclusive.

ENVIRONMENTAL PUBLIC HEALTH TRACKING

AGGREGATE DATA SET SUMMARY

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Characteristic	Description
Data Source	State Birth Defects Registries
Purpose	This data set will be used exclusively to calculate prevalence of Trisomy 21 for two special maternal age groups (<35 years old, ≥35 years old) as described in the Part 1 package, for use on the national public portal.
Geographic Level	The smallest geographic unit to be represented in this data set is the county.
Restrictions	This is a restricted access data set. Data will be displayed via the national public portal only when sufficient conditions have been met to protect data privacy. Only registered users will have direct access to this data set via the national secure portal.

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
BIRTH DEFECT	Birth defect category.	Integer	12 = Trisomy 21	12 only	2	Required	ephtn-ph-BD3.xsd <xsd:element name="BirthDefect" type="Trisomy21BirthDefectType" minOccurs="1" maxOccurs="1" />
STATE	State FIPS code.	String	FIPS	A valid state FIPS code.	2	Not specified in schema	ephtn-core.xsd <xsd:element name="StateFIPSCode" type="statecodeType">

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
COUNTY	County FIPS code over which birth defect cases and underlying birth populations are counted.	String	FIPS U = Unknown	A valid county FIPS code for the state, or 'U'.	5	Required	ephtn-ph-BD3.xsd <xsd:element name="Countycode" type="unknownCountyCodeType" minOccurs="1" maxOccurs="1" />
START DATE ¹	Date on which data aggregation begins.	Date	yyyymmdd		8	Required	ephtn-ph-BD3.xsd <xsd:element name="StartDate" type="NCDMdate" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
END DATE ¹	Date on which data aggregation ends.	Date	yyyymmdd		8	Required	ephtn-ph-BD3.xsd <xsd:element name="EndDate" type="NCDMdate" minOccurs="1" maxOccurs="1" />
SURVEILLANCE TYPE	State birth defects surveillance system uses only active case finding procedures, only passive reporting, or a combination of active and passive methods.	Text	A = active P = passive PF = passive with follow-up	A, P, PF	2	Required	ephtn-ph-BD3.xsd <xsd:element name="Surveillance" type="SurveillanceType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
ASCERTAINMENT	State birth defects surveillance system routinely ascertains cases of birth defects among live births only, or also covers other outcomes of pregnancy (fetal deaths and/or terminations).	Text	L = live births only LF = live births + fetal deaths LT = live births + pregnancy terminations LFT = live births + fetal deaths + pregnancy terminations	L, LF, LT, LFT	3	Required	ephntn-ph-BD3.xsd <xsd:element name="Ascertainment" type="AscertainmentType" minOccurs="1" maxOccurs="1" />
CODE SET	Indicates the standard under which birth defects cases are coded and classified.	Integer	1 = ICD-9-CM 2 = ICD-9-CM, CDC coding based on BPA	1, 2	1	Required	ephntn-ph-BD3.xsd <xsd:element name="CodeSet" type="CodeSetType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
MATERNAL AGE GROUP	Two categories of maternal age for which cases and underlying birth populations are counted.	Integer	1 = < 35 years 2 = ≥ 35 years 9 = Unknown	1, 2, 9	1	Required	ephtn-ph-BD3.xsd <xsd:element name="MaternalAgeGroup" type="MaternalAgeTypeForTrisomy21" minOccurs="1" maxOccurs="1" />
MATERNAL ETHNICITY	Maternal Hispanic ethnicity for which cases and underlying birth populations are counted.	Text	H = Hispanic NH = non-Hispanic U = Unknown	H, NH, U	2	Required	ephtn-ph-BD3.xsd <xsd:element name="MaternalEthnicity" type="ethnicityType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
MATERNAL RACE GROUP	Maternal race group for which cases and underlying birth populations are counted.	Text	W = White B = Black O = Other U = Unknown NS = Not submitted	W, B, O, U, NS Note: The code 'NS' is intended to allow the maternal race field to be selectively collapsed, for example, when maternal ethnicity is Hispanic. This code is not intended to indicate unknown race.	2	Required	ephntn-ph-BD3.xsd <xsd:element name="MaternalRace" type="MaternalRaceType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
INFANT SEX	Infant sex for which cases and underlying birth populations are counted.	Text	M = Male F = Female U = Unknown	M, F, U	1	Required	ephtn-ph-BD3.xsd <xsd:element name="InfantSex" type="InfantSexType" minOccurs="1" maxOccurs="1" />
TOTAL LIVE BIRTHS ²	Total number of live births.	Integer		0 to nnnnnnn	7	Required	ephtn-ph-bd-core.xsd <xsd:element name="TLB" type="sevenDigitNumberType" minOccurs="1" maxOccurs="1">

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
LIVE BIRTHS WITH BIRTH DEFECT	Number of cases of birth defect among live births only.	Integer	-999 = Unknown	0 to nnnn -999 Note: The missing value code -999 is appropriate when a birth count is known for a demographic classification, but the birth defect count is unknown.	4	Required	ephtn-ph-bd-core.xsd <xsd:element name="LBWBD" type="missingFourDigitNumberType" minOccurs="1" maxOccurs="1">

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
LIVE BIRTHS + FETAL DEATHS + TERMINATIONS WITH DEFECT	Number of cases of birth defect among live births plus fetal deaths and/or pregnancy terminations, in those states that ascertain cases among fetal deaths and/or terminations. (optional)	Integer	-999 = Unknown	0 to nnnn -999 Note: The missing value code -999 is appropriate when a birth count is known for a demographic classification, but the birth defect count is unknown.	4	Optional	ephntn-ph-bd-core.xsd <xsd:element name="LBFDTWD" type="missingFourDigitNumberType" minOccurs="0" maxOccurs="1">

- 1 Start dates and end dates will allow for aggregation of more than one year of data when necessary, as well as part-year aggregates.

-
- 2 This is the denominator for “prevalence” calculations. This data may not need to be duplicated as it may be available through some of the vital records datasets as part of EPHT.

ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA SET FROM WHICH MEASURES MAY BE DERIVED
BIRTH DEFECTS

**Trisomy 21 Counts, by Maternal Age, Maternal Ethnicity/Race, and
Infant Sex**

Referred to as Excel file: BD3.xls in the following discussion

Notes on Table Shell

1. Prevalence measures (and corresponding confidence intervals) can be directly calculated from the proposed data set, at both the level at which the data are provided (the finest level of breakdown) or at higher levels of aggregation (for example, all maternal ages). Because prevalence measures at higher levels require that numerators and denominators be calculated first (the prevalence measures cannot be directly aggregated) there is limited utility in including pre-calculated measures in this data set.
2. This table shell supports special tabulations for Trisomy 21 only, involving a cross-classification of broad maternal age groupings (< 35 years, ≥ 35 years, unknown) × maternal ethnicity/race × infant sex. The special maternal age groups are crossed with a partially collapsed maternal ethnicity/race structure (see note 2 [in this document] for table shell BD2.xls) and infant sex for a total of $3 \times 9 \times 3 = 81$ classifications. An example of the complete cross-classification structure appears in lines 3-83 of the table shell.

ENVIRONMENTAL PUBLIC HEALTH TRACKING

AGGREGATE DATA SET SUMMARY

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Characteristic	Description
Data Source	State Birth Defects Registries
Purpose	This data set will be used to calculate prevalence measures for birth defects as described in the Part 1 package, for use on the national public portal.
Geographic Level	The smallest geographic unit to be represented in this data set is the state.
Restrictions	This is a restricted access data set. Data will be displayed via the national public portal only when sufficient conditions have been met to protect data privacy. Only registered users will have direct access to this data set via the national secure portal.

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
------------	-------------------	-----------	-------------	--------------	--------------	-------------	---------------

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
BIRTH DEFECT	Birth defect category.	Integer	1 = Anencephaly 2 = Spina bifida (w/o anencephaly) 3 = Hypoplastic left heart syndrome 4 = Tetralogy of Fallot 5 = Transposition of the great arteries (vessels) 6 = Cleft lip with or w/o cleft palate 7 = Cleft palate w/o cleft lip 8 = Hypospadias 9 = Gastroschisis 10 = Upper limb deficiencies 11 = Lower limb deficiencies 12 = Trisomy 21	1 - 12	2	Required	ephtn-ph-BD4.xsd <xsd:element name="BirthDefect" type="BirthDefectType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
STATE	State FIPS code.	String	FIPS	A valid state FIPS code.	2	Not specified in schema	ephtn-core.xsd <xsd:element name="StateFIPS Code" type="statecodeType">
START DATE ¹	Date on which data aggregation begins.	Date	yyyymmdd		8	Required	ephtn-ph-BD4.xsd <xsd:element name="StartDate" type="NCDMdate" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
END DATE ¹	Date on which data aggregation ends.	Date	yyyymmdd		8	Required	ephtn-ph-BD4.xsd <xsd:element name="EndDate" type="NCDMdate" minOccurs="1" maxOccurs="1" />
SURVEILLANCE TYPE	State birth defects surveillance system uses only active case finding procedures, only passive reporting, or a combination of active and passive methods.	Text	A = active P = passive PF = passive with follow-up	A, P, PF	2	Required	ephtn-ph-BD4.xsd <xsd:element name="Surveillance" type="SurveillanceType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
ASCERTAINMENT	State birth defects surveillance system routinely ascertains cases of birth defects among live births only, or also covers other outcomes of pregnancy (fetal deaths and/or terminations).	Text	L = live births only LF = live births + fetal deaths LT = live births + pregnancy terminations LFT = live births + fetal deaths + pregnancy terminations	L, LF, LT, LFT	3	Required	ephntn-ph-BD4.xsd <xsd:element name="Ascertainment" type="AscertainmentType" minOccurs="1" maxOccurs="1" />
CODE SET	Indicates the standard under which birth defects cases are coded and classified.	Integer	1 = ICD-9-CM 2 = ICD-9-CM, CDC coding based on BPA	1, 2	1	Required	ephntn-ph-BD4.xsd <xsd:element name="CodeSet" type="CodeSetType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
MATERNAL AGE GROUP	Five-year maternal age intervals for which cases and underlying birth populations are counted.	Integer	1 = <20 years 2 = 20-24 years 3 = 25-29 4 = 30-34 5 = 35-39 6 = ≥40 9 = Unknown	1 - 6, 9	1	Required	ephtn-ph-BD4.xsd <xsd:element name="MaternalAgeGroup" type="MaternalAgeType" minOccurs="1" maxOccurs="1" />
MATERNAL ETHNICITY	Maternal Hispanic ethnicity for which cases and underlying birth populations are counted.	Text	H = Hispanic NH = non-Hispanic U = Unknown	H, NH, U	2	Required	ephtn-ph-BD4.xsd <xsd:element name="MaternalEthnicity" type="ethnicityType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
MATERNAL RACE GROUP	Maternal race group for which cases and underlying birth populations are counted.	Text	W = White B = Black O = Other U = Unknown NS = Not submitted	W, B, O, U, NS Note: The code 'NS' is intended to allow the maternal race field to be selectively collapsed, for example, when maternal ethnicity is Hispanic. This code is not intended to indicate unknown race.	2	Required	ephntn-ph-BD4.xsd <xsd:element name="MaternalRace" type="MaternalRaceType" minOccurs="1" maxOccurs="1" />

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
INFANT SEX	Infant sex for which cases and underlying birth populations are counted.	Text	M = Male F = Female U = Unknown	M, F, U	1	Required	ephtn-ph-BD4.xsd <xsd:element name="InfantSex" type="InfantSexType" minOccurs="1" maxOccurs="1" />
TOTAL LIVE BIRTHS ²	Total number of live births.	Integer		0 to nnnnnnn	7	Required	ephtn-ph-bd-core.xsd <xsd:element name="TLB" type="sevenDigitNumberType" minOccurs="1" maxOccurs="1">

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
LIVE BIRTHS WITH BIRTH DEFECT	Number of cases of birth defect among live births only.	Integer	-999 = Unknown	0 to nnnn -999 Note: The missing value code -999 is appropriate when a birth count is known for a demographic classification, but the birth defect count is unknown.	4	Required	ephtn-ph-bd-core.xsd <xsd:element name="LBWBD" type="missingFourDigitNumberType" minOccurs="1" maxOccurs="1">

ENVIRONMENTAL PUBLIC HEALTH TRACKING

DATA DICTIONARY FOR AGGREGATE DATA

BIRTH DEFECTS

Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and Infant Sex

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Optionality	Schema Source
LIVE BIRTHS + FETAL DEATHS + TERMINATIONS WITH DEFECT	Number of cases of birth defect among live births plus fetal deaths and/or pregnancy terminations, in those states that ascertain cases among fetal deaths and/or terminations. (optional)	Integer	-999 = Unknown	0 to nnnn -999 Note: The missing value code -999 is appropriate when a birth count is known for a demographic classification, but the birth defect count is unknown.	4	Optional	ephntn-ph-bd-core.xsd <xsd:element name="LBFDWD" type="missingFourDigitNumberType" minOccurs="0" maxOccurs="1">

- 1 Start dates and end dates will allow for aggregation of more than one year of data when necessary, as well as part-year aggregates.

-
- 2 This is the denominator for “prevalence” calculations. This data may not need to be duplicated as it may be available through some of the vital records datasets as part of EPHT.

ENVIRONMENTAL PUBLIC HEALTH TRACKING
DATA SET FROM WHICH MEASURES MAY BE DERIVED
BIRTH DEFECTS

**Birth Defect Counts, by Maternal Age, Maternal Ethnicity/Race, and
Infant Sex**

Referred to as Excel file: BD4.xls in the following discussion

Notes on Table Shell

1. Prevalence measures (and corresponding confidence intervals) can be directly calculated from the proposed data set, at both the level at which the data are provided (the finest level of breakdown) or at higher levels of aggregation (for example, all maternal ages). Because prevalence measures at higher levels require that numerators and denominators be calculated first (the prevalence measures cannot be directly aggregated) there is limited utility in including pre-calculated measures in this data set.
2. This table shell involves a simultaneous cross-classification of all maternal and infant demographic factors (maternal age in 5-year groupings, a partially collapsed maternal ethnicity/race classification structure, and infant sex) at the state-level only. (See note 2 [in this document] for table shell BD2.xls for a description of the partially collapsed maternal ethnicity/race structure having 9 classes.) The proposed cross-classification structure has $7 \times 9 \times 3 = 189$ classifications. Selected segments of the cross-classification structure appear in lines 3-62 of the table shell.
3. This is the only birth defects data set currently proposed that includes a cross-classification at the indicated level of demographic detail. While the special Trisomy 21 county-level data set (described above) also incorporates a simultaneous cross-classification of all demographic factors, the maternal age groupings proposed for that data set (< 35 years, ≥ 35 years) are very broad.

CONTENT DOMAIN: BIRTH DEFECTS

INDICATOR: PREVALENCE OF BIRTH DEFECTS

Type of EPHT Indicator	Health Outcome
Measure	<ul style="list-style-type: none"> • Average annual number of cases over a 5 year period • Prevalence per 10,000 live births over a five year period
Derivation of Measure(s)	<p>Denominator is composed of all live-born infants in geographic region of interest during a calendar year.</p> <p>Numerator is composed of all live-born infants, fetal deaths (where available), and terminations (where available) with birth defect 'X' in the geographic region of interest, during a calendar year.</p> <p>For states that ascertain fetal deaths and/or terminations, two sets of birth prevalence estimates are to be calculated for each birth defect -- including and excluding fetal deaths and/or terminations.</p> <p>Diagnosis of cases may be made up to one year of age – ascertainment may be at any time.</p>
Unit	Defect presence at birth (or fetal death/termination)
Geographic Scope	State and National (tracking network states)
Geographic Scale	State, county
Time Period	2000-
Time Scale	Calendar year
Rationale	<p>Birth defects pose a significant public health problem. One in 33 babies is born with a structural birth defect in the United States. Birth defects are a leading cause of infant mortality and responsible for considerable morbidity and disability with enormous economic and social costs. A lifetime of medical care and special education for a single child can cost over \$500,000.</p> <p>Approximately 60% of birth defects are of unknown etiology. The ambient environment remains a source of great public concern, but few environmental exposures have been well-studied. Most birth defects will likely be explained by a complex interaction between genetic predispositions and environmental factors. However, prior to the ability to conduct studies to explore these interactions, the linkage of birth defects outcome data with environmental hazard or exposure data is critical. The first step in effecting successful linkages of these data is the existence of high quality birth defects prevalence data for which the geospatial and temporal patterns and distributions can be monitored. The environmental public health tracking (EPHT) initiative is well-</p>

	<p>positioned to bring together birth prevalence data from its state partners to begin analyses of these patterns, which will provide important clues to public health officials and researchers.</p>
<p>Use of the Measure</p>	<p>The basic procedure for calculating birth prevalence is the same for all the suggested birth defects. Once the input data are appropriately prepared, birth prevalence will be calculable for all defects at the same time.</p> <p>State Allow for consistent and rapid method for calculating and displaying (using GIS) prevalence at selected geographical areas (i.e, county level).</p> <p>Allow for a better understanding of spatial and temporal patterns of selected birth defects.</p> <p>National Allow for comparison of birth prevalence across states which can be used to target interventions. Any comparison of birth prevalence, however, will need to account for the variability in data collection methods between state surveillance systems. (See “Limitations of Data Sources” below and introductory text in appended team recommendations).</p>
<p>Limitations of the Measure</p>	<p>Ideally, incidence rates would be used instead of birth prevalence to measure birth defects occurrence. The numerator of the incidence would be the number of new cases of birth defect A in an area and time period and the denominator would be the number of conceptions at risk of developing birth defect A in that area and time period. Because the both the number of conceptions is unknown and the number of cases “lost” through spontaneous abortions (as well as terminations and later fetal losses depending on the source of ascertainment for the specific surveillance system), incidence cannot be calculated. Birth prevalence is the only appropriate measure that can be reported for birth defects occurrence.</p> <p>It is not feasible, at this time, to recommend that individual-level birth defects surveillance data be made available on even a secure national portal. Most states have strict guidelines with respect to confidentiality and even the publication of birth prevalence data based on <5 cases in a geographic region is generally not done.</p>
<p>Data Sources</p>	<p>State birth defects surveillance systems: The data sources that contribute to birth defects surveillance systems include the following (this varies by system type):</p> <ul style="list-style-type: none"> • Vital records • Hospital records (discharge summaries or disease indices,

	<p>nursery logs, NICU logs)</p> <ul style="list-style-type: none"> • Administrative databases (Medicaid, state hospital discharge, HMO) • Specialty data sources (specialty clinics, programs for children with special health care needs) • Prenatal diagnostic centers or genetics clinics • Clinical examination • Local or national laboratories for cytogenetic testing <p>Denominator data will come from state vital records – number of live births, by year, by maternal age, and race/ethnicity. These data may be aggregated and provided to the birth defects surveillance system for calculating birth prevalence, or may be made available on an individual level to the birth defects surveillance system. This varies by state.</p>
<p>Limitations of Data Sources</p>	<p>All states in the US do not have a birth defects surveillance program. Among those that do, there is significant variability between surveillance systems. Refer to the introductory pages of the appended workgroup team recommendations for a more detailed discussion.</p> <ul style="list-style-type: none"> • Ascertainment method (active, passive, passive with follow-up/verification) <ul style="list-style-type: none"> ○ Primary differences are with data sources, coding, availability of verbatim description, and case verification • Ascertainment of spontaneous fetal deaths and variability in gestational age for inclusion. • Ascertainment of prenatally diagnosed cases and elective terminations • Case definitions • Classification as isolated, multiple or syndromic <p>Address data tends to be address at delivery not conception (more relevant time period for birth defects-related exposure).</p> <p>Approximately 50% of birth defects surveillance systems do not geocode their address data.</p>
<p>Related Indicators</p>	
<p>Recommendations for Future Development of Indicator and Measure</p>	<p>Ideally, through support from environmental public health tracking, birth defects surveillance systems will be able to conduct active surveillance of the 12 priority birth defects. Short of this goal, passive surveillance systems should be able to conduct follow-up/verification of cases of these 12 defects. The type of ascertainment system, as well as whether it ascertains prenatally diagnosed cases and elective terminations should be clearly indicated when the birth prevalence is presented.</p>

	<p>Future analyses may:</p> <ul style="list-style-type: none">• Examine spatial or temporal trends in birth defects prevalence. The Birth Defects Content Workgroup Team believes that such analyses, at this time, will be most interesting (and relevant) if conducted within state surveillance systems, rather than between states, due to the heterogeneity between systems with respect to ascertainment, as discussed above.• Explore ecologic associations between environmental hazards and prevalence of birth defects. This group is considering proposal of a second measure/indicator to be put into place in a second phase of development – involving linkage with environmental hazard data.• If states are able to successfully classify cases, birth prevalence would be calculated among isolated cases of each defect, and then for a group of cases with MCA (Multiple Congenital Anomalies). (See detailed discussion on page 4 of workgroup team recommendations) <p>Improve methods for collecting and reporting state birth defects data</p>
--	--

ENVIRONMENTAL PUBLIC HEALTH TRACKING**CHILDHOOD BLOOD LEAD POISONING****Summary for CLP2:****Blood Lead Levels by Birth Cohort, by County**

Characteristic	Description
Data Sources	CDC Lead Program; CLPP Programs; Tracking Grantees
Purpose	<p>This dataset allows public health professionals and researchers on the secure portal to evaluate screening practices and monitor progress towards eliminating lead poisoning among children. Also, this dataset will be used to develop indicators (with appropriate procedures to protect confidentiality) for the national public portal. This dataset can be linked to US Census data and other data sources to identify geographic areas where children are at risk for lead poisoning.</p> <p>This dataset is used to create the following indicators:</p> <ul style="list-style-type: none">• Blood Lead Levels by Birth Cohort
Restrictions	This is a restricted access dataset without appropriate confidentiality measures applied.

**CLP2: BLOOD LEAD LEVELS BY BIRTH COHORT, BY COUNTY
CHILDHOOD LEAD POISONING**

**DATA DICTIONARY FOR AGGREGATE DATA
Environmental Public Health Tracking**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Required
STATE	State FIPS code	String	nn	FIPS code	2	Required
CountyFIPS	County FIPS code	String	nnnnn U = unknown	FIPS code, U	5	Required
BCYear	Year in which birth cohort was born	Integer	nnnn	0-9999	4	Required

**CLP2: BLOOD LEAD LEVELS BY BIRTH COHORT, BY COUNTY
CHILDHOOD LEAD POISONING**

**DATA DICTIONARY FOR AGGREGATE DATA
Environmental Public Health Tracking**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Required
BLLCategory	Categorization of blood lead test results in units of $\mu\text{g}/\text{dL}$	Integer	1 = <5 2 = 5- <10 3 = 10-<15 4 = 15-<20 5 = 20-<25 6 = 25-<45 7 = 45-<70 8 = ≥ 70 9 = No testing in County 10 = County not in system	1-10	2	Required
NumChildrenTested	Number of children tested	Integer		0-nnnnn	5	Required

**CLP2: BLOOD LEAD LEVELS BY BIRTH COHORT, BY COUNTY
CHILDHOOD LEAD POISONING**

**DATA DICTIONARY FOR AGGREGATE DATA
Environmental Public Health Tracking**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Required
Confirmed	Classification of test result as either confirmed or unconfirmed	Integer	0=no, unconfirmed 1=yes, confirmed	0-1	1	Required
Specimen	Classification of test result by type of specimen used	Integer	1=venous 2=capillary 3=unknown specimen	1-3	1	Required

HOW-TO GUIDE: CREATING CLP-2 DATA SET CHILDHOOD LEAD POISONING

Environmental Public Health Tracking

Use of Dataset	<p>This dataset is used to create the following indicators:</p> <ul style="list-style-type: none"> • Blood Lead Levels by Birth Cohort <p><i>Templates describing each indicator and how to generate it are available.</i></p>
Definitions	<p>Blood Lead Level (BLL) Category (in units of $\mu\text{g/dL}$): <5, 5- <10, 10-<15, 15-<20, 20-<25, 25-<45, 45-<70, and ≥ 70.</p> <p>Child: Any person <36 months of age at the time of the blood lead test under consideration.</p> <p>Confirmed Blood Lead Level (BLL): A BLL is confirmed if there is either: (1) one venous test or (2) two capillary or tests of unknown type 1 day to <12 weeks apart.</p> <p>Method Limit of Detection (MDL): The minimum concentration of blood lead that can be determined with a 99% confidence that the true concentration of blood lead can be identified as greater than zero.</p> <p>Result: A quantifiable value or value below the method limit of detection (<MDL) from a blood lead test reported in micrograms per deciliter ($\mu\text{g/dL}$)</p> <p>Test: A blood sample that (1) produces a valid result (as defined above) and (2) was analyzed by a Clinical Lab Improvement Amendments certified facility or an approved portable device.</p> <p>Test Date: The date that the blood lead sample was drawn. When the date of the blood draw is not available, use the earliest date from the following: date of blood lead sample analysis, date of blood lead result report, or date sample was received by laboratory.</p> <p>Unconfirmed Blood Lead Level: A test result without a confirmatory test.</p>
PART 1	Create BLL file

<p>Step #1</p>	<p>Select the following records for BLL file</p> <p>Source: state/local child blood lead surveillance data Variables required to create file: child's date of birth; county, and state of child's residence on day of test; date blood lead test sample drawn or alternative date (see test date definition); sample type (venous, capillary, unknown); blood lead test result.</p> <p>Select blood lead testing records that meet the following criteria:</p> <p>Include:</p> <ul style="list-style-type: none"> • All children born in the birth cohort year chosen for analysis. • All tests where child's age at blood draw date is <36 months. <ul style="list-style-type: none"> ○ Use the test date and birth data to calculate child's age at time of test. When the date of the blood lead draw is not available, use the earliest date from the following: date of blood lead sample analysis, date of blood lead result report, or date received by laboratory. <p>Note: For children listed with provider's address, include those children in the file with county as the county corresponding to the provider's address. If neither address is provided, then list county as unknown.</p> <p>Exclude:</p> <ul style="list-style-type: none"> • Records for which a BLL was not valid. For example, exclude records where lab reported that the quantity of blood drawn was insufficient for analysis or the sample clotted. • Tests that were not done at a Clinical Lab Improvement Amendments certified facility or were not done using an approved portable device
<p>Step #2</p>	<p>Assign Unique ID Numbers</p> <p>Many young children have more than one blood test each year or over multiple years. To get a count of children tested (rather than total tests), and to assign each child to a BLL category:</p> <ol style="list-style-type: none"> 1. Each child should have a unique ID number. 2. All test results must be matched to the appropriate child ID number. <p>There can be one test or multiple lab results for a given child. There can be only one lab report record per child per sample date.</p> <p>If ID numbers have not been previously assigned, then assign an ID number to each child and match all records by child.</p>
<p>Step #3</p>	<p>Retain the following variables in the records selected</p> <p>ChildIDNumber, State, CountyFIPS, Birth_Date, Test_Date, Test_Type, Test_Result</p>
<p>Step #4</p>	<p>Create New variables</p>

	<p>BLLCategory</p> <p>Code scheme:</p> <p>1 = <5 2 = 5 - <10 3 = 10-<15 4 = 15-<20 5 = 20-<25 6 = 25-<25 7 = 45-<70 8 = ≥70</p> <p>Total tested for the year equals the sum of all BLL categories.</p> <p>Specimen variable identifies the test type for the test.</p> <p>Specimen 1=Venous 2= Capillary 3= Unknown</p> <p>Confirmed variable identifies status of child's results.</p> <p>Confirmed 0=no 1=yes 2=unknown</p>
<p>Step #5</p>	<p>Assign child to appropriate BLL category variable</p> <p>Classification scheme:</p> <ol style="list-style-type: none"> 1. Does child have a test result ≥ 10 $\mu\text{g}/\text{dL}$ from a venous specimen? <ol style="list-style-type: none"> a. Yes, use test result to classify child's BLL and set Specimen to venous and confirmed to yes. If child has more than one venous test ≥ 10 $\mu\text{g}/\text{dL}$, then use the highest test result. b. No, proceed to question 2. 2. Does child have a test result ≥ 10 $\mu\text{g}/\text{dL}$ from a capillary or unknown specimen? <ol style="list-style-type: none"> a. Yes, proceed to question 3. b. No, proceed to question 4. 3. Does the child have a confirmatory test (venous, capillary, or unknown

	<p>specimen) between 1 day and < 12 weeks after first test?</p> <ol style="list-style-type: none"> a. Yes, use the confirmatory test result to classify child's BLL (<10, 10-14, 15-19, 20-24, 25-44, 45-69, or ≥70). Set Specimen to capillary or unknown and confirmed to yes. If child has more than one set of elevated capillary or unknown specimen test and a confirmatory test, use the results from the highest confirmatory test to classify the child's BLL, Specimen, and confirmed. b. No, classify child by BLL category using the latest test result. Set Specimen to capillary or unknown and confirmed to no. <ol style="list-style-type: none"> 4. Does the child have a venous test < 10 µg/dL? <ol style="list-style-type: none"> a. Yes, classify child as 0 to <5 or 5 to <10. Retain Specimen as venous and confirmed as yes. b. No, proceed to question 5. 5. Does the child have two capillary or unknown specimen tests results < 10 µg/dL and between 1 day and < 12 weeks apart? <ol style="list-style-type: none"> a. Yes, classify child by BLL category using the latest test result. Specimen as capillary or unknown specimen and confirmed to yes. b. No, classify child by BLL category using the latest test result. Set Specimen as capillary or unknown specimen and confirmed to no. <p>Follow the same rules even if a child with more than one test has a different address for each test. Provide the county which corresponds to the selected test. Test results below the method limit of detection should be classified as <5 µg/dL.</p>
Step #6	<p>Confirm accuracy of state counties. Set to "U" for unknown if not included or are incorrect.</p>
Step #7	<p>Create variable "BCYear" using Birth Date to represent the year in which the child was born.</p>
Step #8	<p>Retain the following variables ChildIDNumber, State, CountyFIPS, BCYear, BLLCategory, Confirmed and Specimen</p>
PART 2	<p>Aggregate by county</p>
Step #1	<p>Create variable "NumChildrenTested" which represents the total number of children in each county, Year, and BLL category.</p>
Step #2	<p>Retain the following variables for the BLL file State, CountyFIPS, BCYear, BLLCategory, Count, Confirmed and Specimen</p>
Note:	<p>Part 3 are not required for data submission to CDC Tracking.</p>

Part 3	Merge CLP 2 with population data from Vital Statistics
Step #1	<p>Create the county birth data Source: Vital Statistics birth data</p> <ul style="list-style-type: none"> • Select all births for birth year = “YYYY” (matching lead birth cohort year) • Delete any counties that fall outside the state • Sum the number of births by county
Step #2	<p>Retain the following variables for the birth data Year, CountyFIPS, TotalBirths</p>
Step #3	<p>Create merged CLP2 and birth data Merge the CLP2 (created in Part 3) and birth data by county and birth year</p>
Step #4	<p>Retain the following variables for the merged child test/birth data: State, CountyFIPS, BCYear, BLLCategory, NumChildrenTested, TotalBirths, Confirmed and Specimen</p>

ENVIRONMENTAL PUBLIC HEALTH TRACKING

CHILDHOOD BLOOD LEAD POISONING

Summary for CLP4:

Annual Blood Lead Levels, by County

Characteristic	Description
Data Sources	CDC Lead Program; CLPP Programs; Tracking Grantees
Purpose	<p>This dataset allows public health professionals and researchers on the secure portal to evaluate screening practices and monitor progress towards eliminating lead poisoning among children. Also, this dataset will be used to develop indicators (with appropriate procedures to protect confidentiality) for the national public portal. This dataset can be linked to US Census data and other data sources to identify geographic areas where children are at risk for lead poisoning.</p> <p>This dataset is used to create the following indicators:</p> <ul style="list-style-type: none"> • Annual Blood Lead Levels
Restrictions	This is a restricted access dataset without appropriate confidentiality measures applied.

**CLP4: ANNUAL BLOOD LEAD LEVELS, BY COUNTY
CHILDHOOD LEAD POISONING**

**DATA DICTIONARY FOR AGGREGATE DATA
Environmental Public Health Tracking**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Required
STATE	State FIPS code	String	nn	FIPS code	2	Required
CountyFIPS	County FIPS code	String	nnnnn U = unknown	FIPS code, U	5	Required
YearTested	Year in which tests occurred	Integer	nnnn	0-9999	4	Required

**CLP4: ANNUAL BLOOD LEAD LEVELS, BY COUNTY
CHILDHOOD LEAD POISONING**

**DATA DICTIONARY FOR AGGREGATE DATA
Environmental Public Health Tracking**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Required
AgeGroup	Age in months of child at time of test	Integer	1 = 0-<12 2 = 12-<24 3 = 24-<36 4 = 36-<48 5 = 48-<60 6 = 60-<72 9 = unknown age 10=No Testing in county 11=County not in system	1-6, 9 – 11	2	Required

**CLP4: ANNUAL BLOOD LEAD LEVELS, BY COUNTY
CHILDHOOD LEAD POISONING**

**DATA DICTIONARY FOR AGGREGATE DATA
Environmental Public Health Tracking**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Required
BLLCategory	Categorization of blood lead test results in units of $\mu\text{g}/\text{dL}$	Integer	1 = <5 2 = 5 - <10 3 = 10-<15 4 = 15-<20 5 = 20-<25 6 = 25-<45 7 = 45-<70 8 = ≥ 70 9=No testing in county 10=County not in system	1 - 10	2	Required
Specimen	Classification of test result by type of specimen used	Integer	1=venous 2=capillary 3=unknown specimen	1-3	1	Required

**CLP4: ANNUAL BLOOD LEAD LEVELS, BY COUNTY
CHILDHOOD LEAD POISONING**

**DATA DICTIONARY FOR AGGREGATE DATA
Environmental Public Health Tracking**

Field Name	Field Description	Data Type	Code Scheme	Legal Values	Field Length	Required
Confirmed	Classification of test result as either confirmed or unconfirmed	Integer	0=no, unconfirmed 1=yes, confirmed	0-1	1	Required
NumChildrenTested	Number of children tested	Integer		0-nnnnn	5	Required

HOW-TO GUIDE: CREATING CLP-4 DATA SET CHILDHOOD LEAD POISONING

Environmental Public Health Tracking

Use of Dataset	<p>This dataset is used to create the following indicators:</p> <ul style="list-style-type: none"> • Annual Blood Lead Levels <p><i>Templates describing each indicator and how to generate it are available.</i></p>
Definitions	<p>Age group: Age in months of child at time of test (0-<12, 12-<24, 24-<36, 36-<48, 48-<60, and 60-<72)</p> <p>Blood Lead Level (BLL) Category (in units of $\mu\text{g}/\text{dL}$): 0 - <5, 5-<10, 10-<15, 15-<20, 20-<25, 25-<45, 45-<70, and ≥ 70.</p> <p>Child: Any person <72 months of age at the time of the blood lead test under consideration.</p> <p>Confirmed Blood Lead Level (BLL): A BLL is confirmed if there is either: (1) one venous test or (2) two capillary or tests of unknown type 1 day to <12 weeks apart.</p> <p>Method Limit of Detection (MDL): The minimum concentration of blood lead that can be determined with a 99% confidence that the true concentration of blood lead can be identified as greater than zero.</p> <p>Result: A quantifiable value or value below the method limit of detection (<MDL) from a blood lead test reported in micrograms per deciliter ($\mu\text{g}/\text{dL}$)</p> <p>Test: A blood sample that (1) produces a valid result (as defined above) and (2) was analyzed by a Clinical Lab Improvement Amendments certified facility or an approved portable device.</p> <p>Test Date: The date that the blood lead sample was drawn. When the date of the blood draw is not available, use the earliest date from the following: date sample was received by laboratory, date of blood lead sample analysis, or date of blood lead result report.</p> <p>Unconfirmed Blood Lead Level: A test result without a confirmatory test within 12 weeks.</p>

PART 1	Create BLL file
Step #1	<p>Select the following records for BLL file Source: state/local child blood lead surveillance data Variables required to create file: child's date of birth; county, and state of child's residence on day of test; date blood lead test sample drawn or alternative date (see test date definition); sample type (venous, capillary, unknown); blood lead test result.</p> <p>Select blood lead testing records that meet the following criteria:</p> <p>Include:</p> <ul style="list-style-type: none"> • All children tested in the year chosen for analysis. • All tests where child's age at blood draw date is <72 months. <ul style="list-style-type: none"> ○ Use the test date and birth date to calculate child's age at time of test. When the date of the blood lead draw is not available, use the earliest date from the following: date of blood lead sample analysis, date of blood lead result report, or date received by laboratory. <p>Note: For children listed with provider's address, include those children in the file with county as the county corresponding to the provider's address. If neither address is provided, then list county as unknown.</p> <p>Exclude:</p> <ul style="list-style-type: none"> • Records for which a BLL was not valid. For example, exclude records where lab reported that the quantity of blood drawn was insufficient for analysis or the sample clotted. • Tests that were not done at a Clinical Lab Improvement Amendments certified facility or were not done using an approved portable device
Step #2	<p>Assign Unique ID Numbers Many young children have more than one blood test each year or over multiple years. To get a count of children tested (rather than total tests), and to assign each child to a BLL category:</p> <ol style="list-style-type: none"> 1. Each child should have a unique ID number. 2. All test results must be matched to the appropriate child ID number. <p>There can be one test or multiple lab results for a given child. There can be only one lab report record per child per sample date.</p> <p>If ID numbers have not been previously assigned, then assign an ID number to each child and match all records by child.</p>
Step #3	<p>Retain the following variables in the records selected</p> <p>ChildIDNumber, State, CountyFIPS, Birth_Date, Test_Date, Test_Type, Test_Result</p>

<p>Step #4</p>	<p>Create New variables</p> <p>Variable called BLLCategory.</p> <p>Code scheme: 1 = <5 2 = 5 - <10 3 = 10-<15 4 = 15-<20 5 = 20-<25 6 = 25-<45 7 = 45-<70 8 = ≥70 9=No testing in county 10 = County not in system</p> <p>Total tested for the year equals the sum of all BLL categories.</p> <p>Specimen variable identifies the test type for the test.</p> <p>Specimen 1=Venous 2= Capillary 3= Unknown</p> <p>Confirmed variable identifies status of child's results.</p> <p>Confirmed 0=no 1=yes 2=unknown</p>
<p>Step #5</p>	<p>Assign child to appropriate BLL category.</p> <p>Classification scheme:</p> <ol style="list-style-type: none"> 1. Does child have a test result ≥ 10 $\mu\text{g/dL}$ from a venous specimen? <ol style="list-style-type: none"> a. Yes, use test result to classify child's BLL and set Specimen to venous and confirmed to yes. If child has more than one venous test ≥ 10 $\mu\text{g/dL}$, then use the highest test result. b. No, proceed to question 2.

	<ol style="list-style-type: none"> 2. Does child have a test result ≥ 10 $\mu\text{g/dL}$ from a capillary or unknown specimen? <ol style="list-style-type: none"> a. Yes, proceed to question 3. b. No, proceed to question 4. 3. Does the child have a confirmatory test (venous, capillary, or unknown specimen) between 1 day and < 12 weeks after first test? <ol style="list-style-type: none"> a. Yes, use the confirmatory test result to classify child's BLL (< 10, 10-14, 15-19, 20-24, 25-44, 45-69, or ≥ 70). Set Specimen to capillary or unknown and confirmed to yes. If child has more than one set of elevated capillary or unknown specimen test and a confirmatory test, use the results from the highest confirmatory test to classify the child's BLL, test type, and confirmed. b. No, classify child by BLL category using the latest test result. Set Specimen to capillary or unknown and confirmed to no. 4. Does the child have a venous test < 10 $\mu\text{g/dL}$? <ol style="list-style-type: none"> a. Yes, classify child as 0 to < 5 or 5 to < 10. Retain Specimen as venous and confirmed as yes. b. No, proceed to question 5. 5. Does the child have two capillary or unknown specimen tests results < 10 $\mu\text{g/dL}$ and between 1 day and < 12 weeks apart? <ol style="list-style-type: none"> a. Yes, classify child by BLL category using the latest test result. Set Specimen as capillary or unknown specimen and confirmed to yes. b. No, classify child by BLL category using the latest test result. Specimen as capillary or unknown specimen and confirmed to no. <p>Follow the same rules even if a child with more than one test has a different address for each test. Provide the county which corresponds to the selected test. Test results below the method limit of detection should be classified as < 5 $\mu\text{g/dL}$.</p>
Step #6	<p>Confirm accuracy of state counties. Set to "U" for unknown if not included or are incorrect.</p>
Step #7	<p>Create variable "AgeGroup" using Birth_Date and Test_Date. "AgeGroup" represents the child's age in months at the time of the blood lead test.</p> <p>1 = 0-< 12 2 = 12-< 24 3 = 24-< 36 4 = 36-< 48 5 = 48-< 60 6 = 60-< 72</p>

Step #8	Create variable “YearTested” using Test_Date to represent the year in which the child was tested.
Step #9	Retain the following variables ChildIDNumber, State, CountyFIPS, YearTested, AgeGroup, BLLCategory, Specimen, Confirmed
PART 2	Aggregate by county
Step #1	Create variable “NumChildrenTested” which represents the total number of children in each county, Year, AgeGroup, BLLCategory, Specimen, and Confirmed.
Step #2	Retain the following variables for the BLL file State, CountyFIPS, YearTested, AgeGroup, BLLCategory, Specimen, Confirmed, NumChildrenTested
Note:	Part 3 is not required for data submission to CDC Tracking.
Part 3	Merge CLP 4 with population data from Census
Step #1	Create the county population data Source: Vintage bridged-race postcensal population estimates available through NVSS: http://www.cdc.gov/nchs/nvss/bridged_race.htm
Step #2	Retain the following variables for the population data State, CountyFIPS, Year, AgeGroup, Population
Step #3	Create merged BLL and birth file Merge the BLL file (created in Part 3) and birth file by county, age group, and year
Step #4	Retain the following variables for the merged child test/population data State, CountyFIPS, YearTested, AgeGroup, BLLCategory, Specimen, Confirmed, NumChildrenTested, Population

CONTENT DOMAIN: CHILDHOOD LEAD POISONING INDICATOR: ANNUAL BLOOD LEAD LEVELS

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Exposure
Measure(s)	<ol style="list-style-type: none"> 1. Number of children tested, by age group¹, by county and state 2. Percent of children tested, by age group¹, by county and state 3. Number of children tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$^{3,4}, by age group¹, by county and state 4. Percent of children tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$^{3,4}, by age group¹, by county and state 5. Number of children tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$ by blood lead level category^{2,3,4}, by age group¹, by state 6. Percent of children tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$, by blood lead level category^{2,3,4}, by age group¹, by state\ 7. Number of children tested with blood lead levels between 5 and $<10 \mu\text{g/dL}$^{3,4,5}, by age group¹, by county and state 8. Percent of children tested with blood lead levels between 5 and $<10 \mu\text{g/dL}$^{3,4,5}, by age group¹, by county and state <p>Notes:</p> <p>¹Measures are available stratified by two age groups: <36 months and 36 to <72 months</p> <p>² The current blood lead reference level is $5 \mu\text{g/dL}$ based on National Health and Nutrition Examination Survey (NHANES) 2007 – 2008 and 2009 – 2010 data published in the Fourth National Report on Human Exposure to Environmental Chemicals, and updated in 2012. Blood Lead Levels (BLLs) $\geq 10 \mu\text{g/dL}$ are confirmed if there is either: (1) one elevated venous test or (2) two elevated capillary and/or unknown tests at least 1 day but less than 12 weeks apart.</p> <p>³ Details about selecting the appropriate test to classify a child are in the “How-To-Guide for Creating CLP-4 datasets.”</p> <p>⁴ BLL categories (in units of $\mu\text{g/dL}$) are <5, $5-<10$, , $10-14$, $15-19$, $20-24$, $25-44$, $45-69$, and ≥ 70. Data will be presented by blood lead categories at the state level only.</p>

	<p>⁵5 - < 10 µg/dL measures can be stratified by confirmed status on the public portal</p>
Derivation of Measure(s)	<p>Create CLP-4 (county level) dataset using the “How-To-Guide for Creating CLP-4 datasets.”</p> <ul style="list-style-type: none"> • Select children’s records from childhood lead poisoning database. • Classify test results. • Aggregate by county of residence and year • Merge with total number of children by county to obtain the denominator. <p><u>From CLP-4 dataset, calculate the measures:</u></p> <ol style="list-style-type: none"> 1. Number of children tested <ul style="list-style-type: none"> • Sum all BLL categories including the unconfirmed 2. Percent of children tested <ul style="list-style-type: none"> • Divide number of children tested by the total number of children 3. Number of children tested with confirmed blood lead levels ≥ 10 µg/dL⁴ <ul style="list-style-type: none"> • Sum number of children in BLL categories ≥ 10 µg/dL (BLLs 10-14,...,BLLs70), excluding unconfirmed 4. Percent of children tested with confirmed blood lead levels ≥ 10 µg/dL⁴ <ul style="list-style-type: none"> • Divide number of children tested with blood lead levels ≥ 10 µg/dL by the total number of children tested and multiply by 100 5. Number of children tested with confirmed blood lead levels ≥ 10 µg/dL⁴ <ul style="list-style-type: none"> • Sum number of children for each BLL category 6. Percent of children tested with confirmed blood lead levels ≥ 10 µg/dL⁴ <ul style="list-style-type: none"> • Divide number of children for each BLL category by the total number of children tested and multiply by 100 7. Number of children tested with blood lead levels between 5 and <10 µg/dL⁴, by age group, by county and state <ul style="list-style-type: none"> • Sum number of children in BLL categories 5 and < 10 µg/dL, including unconfirmed 8. Percent of children tested with blood lead levels between 5 and <10 µg/dL⁴, by age group, by county and state <ul style="list-style-type: none"> • Divide the number of children tested with BLLs between 5 and < 10 µg/dL by the total number of children
Unit	Number and percent
Geographic Scope	State or National
Geographic Scale	County or State (measures 1-4 available at county and state; measures 5 and 6 available only at state)

Time Period	2000 to current
Time Scale	Annual
Rationale	<p>Blood lead levels, even low levels, in children have been associated with adverse health effects ranging from learning impairment and behavioral problems to death. Lead can affect almost every organ and system in your body. The effects of lead are the same whether it enters the body through breathing or swallowing. Small children can be exposed by eating lead-based paint chips, chewing on objects painted with lead-based paint or swallowing house dust or soil that contains lead. Children are more vulnerable to lead poisoning than adults. The main target for lead toxicity is the nervous system in young children. A child who swallows large amounts of lead may develop blood anemia, severe stomachache, muscle weakness, and brain damage. If a child swallows smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, lead can affect a child's mental and physical growth.</p> <p>Since children may have higher BLLs and not display any specific symptoms, CDC recommends blood lead testing for young children at risk for lead poisoning. The risk factors identified by the National Health and Nutrition Examination Surveys (NHANES) include living in housing built before 1950, especially housing in deteriorating condition, being African American, and living in poverty.</p> <p>States have developed and implemented assessment protocols for children to determine the need for a blood lead test. For both universal and targeted testing strategies, children should be tested at least once before the age of 3 years. Some states require more than one test between the ages of 6 and 36 months. Children not tested before the age of 3 should be tested at least once before the age of 6. In all states, a blood lead test is required for Medicaid-eligible children at 12 and 24 months.</p> <p>CDC updated its recommendations on children's blood lead levels in May 2012. The new recommendation is based on the U.S population of children aged 1-5 years who are in the top 2.5% of children tested for lead in their blood. This reference value is the 97.5th percentile, which is currently 5 µg/dL based on NHANES 2007 – 2008 and 2009 – 2010 data (CDC, 2012). The recommendation that chelation therapy should be considered for children with BLLs ≥ 45 µg/dL has not changed. BLL results ≥ 70 µg/dL represent a medical emergency. Many states initiate environmental investigations at either BLLs ≥ 20 µg/dL or persistent BLL results that are 15-19 µg/dL</p> <p>This indicator provides information on the number of children tested each year, and the number of those children tested with confirmed blood lead levels above 5 µg/dL. This information is used to direct resources for testing and management of cases above the reference value and be linked with environmental or the risk factor data to monitor trends over time.</p>

Use of the Measure(s)	<ul style="list-style-type: none"> • To identify and monitor temporal and spatial changes in BLL testing and confirmed BLLs $\geq 5\mu\text{g/dL}$ by year. • To better understand BLL surveillance data when interpreting number of confirmed BLLs $\geq 5\mu\text{g/dL}$. • To compare testing and BLLs within and across states for the purpose of targeting interventions. Comparisons should only be made between areas with similar testing and reporting rules. • To link data on risk factors and compare risk factors within and across states. • To guide interventions and allocation of resources related to BLL testing and prevention of lead exposure in young children. • To develop and support public health policy and legislation related to BL testing and prevention of childhood lead exposure. • To monitor progress towards eliminating BLLs $\geq 5 \mu\text{g/dL}$, the current reference value (NHANES 2007 – 2008 and 2009 – 2010 data).
Limitations of the Measure(s)	<ul style="list-style-type: none"> • The analysis uses the county of the child’s residence at the time of the test, which may be different from the county where the child was exposed to lead. • Counties are not homogenous with respect to the distribution of lead hazards or risk factors for lead exposure. • Number and percent of BLLs through surveillance data cannot be interpreted as prevalence or incidence for the population as a whole • State to state comparisons must be made cautiously and require additional information about the states’ testing practices, confirmatory testing practices, and reporting laws. • Because the capillary test is subject to contamination it can result in a false positive BLL. The number and percent of BLLs would be overestimated if unconfirmed, non-venous test results are used.
Data Sources	<p>Childhood Blood Lead Surveillance Data Census Population Data: Vintage bridged-race post-censal population estimates available through NVSS: http://www.cdc.gov/nchs/nvss/bridged_race.htm</p>
Limitations of Data Sources	<p>Childhood Blood Lead Surveillance Data</p> <ul style="list-style-type: none"> • Surveillance data are not randomly sampled or representative of the population. • Complete residential addresses are not available for all children tested. • If the child’s address is not provided the address of the provider may be used.
Presentation	<p>Small numbers of children tested, births, or BLLs may exist when the measures are calculated at the county levels. These small numbers are not accurate estimates for childhood lead poisoning in these polygons. In addition, these small numbers will require additional data processing steps to preserve confidentiality. One or more of the following methods can be used:</p> <ul style="list-style-type: none"> • Suppression of small numbers, • Aggregation of neighboring geographic units • Aggregation to a lower resolved geographic level unit, • Aggregation of successive birth cohorts.

	<p>Data on confirmed levels are presented by categories at the state level only.</p> <p>This indicator should be displayed with information about the lead testing program, including:</p> <ul style="list-style-type: none"> • State and/or local testing policies or strategies (i.e., targeted or universal) • CDC-funded Childhood Lead Poisoning Prevention Program • Minimum BLL reported by laboratories to state or local lead program
Related Indicators	<p>Age of Housing</p> <p>Blood Lead Levels by Birth Cohort</p>
References	<p>Centers for Disease Control and Prevention (CDC). 2012. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in “Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention”.</p>

CONTENT DOMAIN: CHILDHOOD LEAD POISONING INDICATOR: BLOOD LEAD LEVELS BY BIRTH COHORT

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Exposure
Measure(s)	<ol style="list-style-type: none"> 1. Number of children born in the same year and tested , by county and state 2. Percent of children born in the same year and tested, by county and state 3. Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$², by county and state 4. Percent of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$², by county and state 5. Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$², by blood lead level category³, by state 6. Percent of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}$², by blood lead level category³, by state 7. Number of children tested with blood lead levels between 5 and $<10 \mu\text{g/dL}$^{2,4}, by county and state 8. Percent of children tested with blood lead levels between 5 and $<10 \mu\text{g/dL}$^{2,4}, by county and state <p>¹ The current blood lead reference level is 5 $\mu\text{g/dL}$ based on National Health and Nutrition Examination Survey (NHANES) 2007 – 2008 and 2009 – 2010 data published in the Fourth National Report on Human Exposure to Environmental Chemicals, and updated in 2012. Blood Lead Levels (BLLs) are confirmed if there is either: (1) one elevated venous test or (2) two elevated capillary and/or unknown tests at least 1 day but less than 12 weeks apart.</p> <p>²Details about selecting the appropriate test to classify a child are in the “How-To-Guide for Creating CLP-2 datasets.”</p> <p>³ BLL categories (in units of $\mu\text{g/dL}$) are <5, $5 - <10$, $10-<15$, $15-<20$, $20-<25$, $25-<45$, $45-<70$, and ≥ 70. Data are presented by categories at the state level only.</p> <p>⁴ $5 - < 10 \mu\text{g/dL}$ measures can be stratified by confirmed status on the public portal</p>

Derivation of Measure(s)	<p><u>Create CLP-2 (county level) dataset using the “How-To-Guide for Creating CLP-2 datasets.”</u></p> <ul style="list-style-type: none"> • Select children’s records from childhood lead poisoning database. • Classify test results. • Aggregate by county of residence and birth cohort. • Merge with total number of county to obtain the denominator. <p><u>From CLP-2 dataset, calculate the measures:</u></p> <ol style="list-style-type: none"> 1. Number of children born in the same year and tested, by county and state <ul style="list-style-type: none"> • Sum all BLL categories including the unconfirmed 2. Percent of children born in the same year and tested, by county and state <ul style="list-style-type: none"> • Divide number of children tested by the total number of children in the birth cohort 3. Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by county and state <ul style="list-style-type: none"> • Sum number of children in BLL categories $\geq 10 \mu\text{g/dL}$ (BLLs10_14,...,BLLs70), excluding unconfirmed 4. Percent of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by county and state <ul style="list-style-type: none"> • Divide number of children tested with BLLs $\geq 10 \mu\text{g/dL}$ by the total number of children tested and multiply by 100 5. Number of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by blood lead level category³, by state <ul style="list-style-type: none"> • Sum number of children by BLL categories $\geq 10 \mu\text{g/dL}$ (BLLs10_14,...,BLLs70), excluding unconfirmed 6. Percent of children born in the same year and tested with confirmed blood lead levels $\geq 10 \mu\text{g/dL}^2$, by blood lead level category³, by state <ul style="list-style-type: none"> • BLL Categories = Divide number of children for each BLL category by the total number of children tested and multiply by 100 7. Number of children tested with blood lead levels between 5 and <10, by county and state <ul style="list-style-type: none"> • Sum number of children in BLL categories 5 and < 10 $\mu\text{g/dL}$, including unconfirmed 8. Percent of children tested with blood lead levels between 5 and <10 $\mu\text{g/dL}$, by county and state <ul style="list-style-type: none"> • Divide the number of children tested with BLLs between 5 and < 10 $\mu\text{g/dL}$ by the total number of children
Unit	Number and percent

Geographic Scope	State or National
Geographic Scale	County or State (measures 1-4 available by county and state; measures 5 and 6 available by state)
Time Period	2000 (or first available) to current
Time Scale	Annual birth cohort
Rationale	<p>Blood lead levels in young children have been associated with adverse health effects ranging from learning impairment and behavioral problems to death. No threshold for adverse effects has been identified. Because children may have elevated BLLs and not have any specific symptoms, CDC recommends blood lead testing for young children at risk for lead poisoning. The risk factors identified by the National Health and Nutrition Examination Surveys (NHANES) include living in housing built before 1950, especially housing in deteriorating condition, being African American, and living in poverty.</p> <p>Many states have adopted a targeted testing strategy (i.e., test children at high risk), whereas some states recommend universal testing (i.e., test all children), either statewide or within high-risk counties and cities. For both universal and targeted testing strategies, children should be tested at least once before the age of 3 years. Some states require more than one test between the ages of 6 and 36 months. In all states, a blood lead test is required for Medicaid-eligible children at 12 and 24 months of age.</p> <p>CDC updated its recommendations on children's blood lead levels in May 2012. The new recommendation is based on the U.S population of children aged 1-5 years who are in the top 2.5% of children tested for lead in their blood. This reference value is the 97.5th percentile, which is currently 5 µg/dL based on NHANES 2007 – 2008 and 2009 – 2010 data (CDC, 2012). The recommendation that chelation therapy should be considered for children with BLLs ≥45 µg/dL has not changed. BLL results ≥70 µg/dL represent a medical emergency. Many states initiate environmental investigations at either BLLs ≥20 µg/dL or persistent BLL results that are 15-19 µg/dL</p> <p>This indicator uses a birth cohort approach. Using these measures, it is possible to determine how many children born in a specific year were tested before the ages of 3 and how many of those tested had an elevated BLL. For children with more than one test before the age of 3, this indicator uses the highest venous specimen result or if there is no venous specimen the highest confirmatory capillary/unknown result. Using the highest results allows for examination of the peak BLLs for the birth cohort. Inclusion of multiple cohorts will allow for the evaluation of trends in testing and BLLs greater than the reference value.</p>
Use of the	<ul style="list-style-type: none"> • To identify and monitor temporal and spatial changes in BLL testing and -BLLs by

Measure(s)	<p>birth cohort.</p> <ul style="list-style-type: none"> • To better understand BLL surveillance data when interpreting number of -BLLs. • To compare testing and BLLs within and across states for the purpose of targeting interventions. Comparisons should only be made between areas with similar testing and reporting rules. • To link data on risk factors and compare risk factors within and across states. • To guide interventions and allocation of resources related to BLL testing and prevention of lead exposure in young children. • To develop and support public health policy and legislation related to BLL testing and prevention of childhood lead poisoning. • To monitor progress towards eliminating BLLs ≥ 5 $\mu\text{g/dL}$, the current reference value (NHANES 2007 – 2008 and 2009 – 2010 data).
Limitations of the Measure(s)	<ul style="list-style-type: none"> • The analysis uses the county of the child's residence at the time of the test, which may be different from the county where the child was exposed to lead. • Counties are not homogenous with respect to the distribution of lead hazards or risk factors for lead exposure. • Number and percent of BLLs cannot be interpreted as prevalence or incidence for the population. • State to state comparisons must be made cautiously and require additional information about the states' testing practices, confirmatory testing practices, and reporting laws. • Because the capillary test is subject to contamination it can result in a false positive BLL. The number and percent of BLLs may be overestimated when non-venous test results are used.
Data Sources	<p>Childhood Blood Lead Surveillance Data Vital Statistics Birth Data</p>
Limitations of Data Sources	<p>Childhood Blood Lead Surveillance Data</p> <ul style="list-style-type: none"> • Surveillance data are not randomly sampled or representative of the population. • Complete residential addresses are not available for all children tested. • Sometimes the address of the provider or another address is listed as the child's address when the data is not provided by the reporting authority. <p>Vital Statistics Birth Data</p> <ul style="list-style-type: none"> • The number of children born from Vital Statistics does not include children who have moved in or out of the area since birth. Therefore, as a denominator, it may under or over estimate the number of children in a birth cohort.
Presentation	<p>Small numbers of children tested, births, or BLLs may exist when the measures are calculated at the county levels. These small numbers are not accurate estimates for childhood lead poisoning in these polygons. In addition, these small numbers will require additional data processing steps to preserve confidentiality. One or more of the following methods can be used:</p> <ul style="list-style-type: none"> • Suppression of small numbers, • Aggregation of neighboring geographic units.

	<ul style="list-style-type: none"> • Aggregation to a lower resolved geographic level unit, • Aggregation of successive birth cohorts. <p>Data on blood lead levels are presented by categories at the state level only.</p> <p>This indicator should be displayed with information about the lead testing program, including:</p> <ul style="list-style-type: none"> • State and/or local testing policies or strategies (i.e., targeted or universal) • CDC-funded Childhood Lead Poisoning Prevention Program • Minimum BLL reported by laboratories to state or local lead program
Related Indicators	Blood Lead Testing and Housing Age Annual Blood Lead Levels
References	Centers for Disease Control and Prevention (CDC). 2012. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in “Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention”.

Drinking Water Quality Nationally Consistent Data and Measures (NCDMs)

How-To Guide

This How-To Guide (Guide) provides a general outline of the steps required for processing state drinking water quality compliance datasets into NCDMs for the National Environmental Public Health Tracking Network (NEPHTN).

The Guide takes into account newly accepted XML schema changes, the addition of six new analytes (Atrazine, DEHP, PCE, TCE, Radium, and Uranium), and new locational information requirements for water systems. Prior to these changes, water quality NCDMs were tracked for four analytes: Nitrate, Arsenic, total Trihalomethanes (TTHM), and Haloacetic Acids (HAA5). As a convention, all ten analytes are treated generically in this Guide unless analyte-specific details are otherwise noted. State water quality Safe Drinking Water Act compliance databases are referred to as “SDWIS” (Safe Drinking Water Information System), since most grantee states use the SDWIS-State database. States that have a SDWIS-like database can assume that the term “SDWIS” refers to their water quality database(s) as well.

The Guide is organized into the following sections (e.g., major steps):

- I. Staging Table Development
- II. XML Dataset Development
- III. State-Level NCDM Development

I. Staging Table Development

This set of processing steps involves extracting the following types of records for the reporting period from SDWIS:

1. Water system descriptive and location information, i.e. the Inventory table; and
2. Water quality sampling results information, i.e. the “Sampling Results” table.

The reporting period is 1999 to the most current year having complete water quality information within SDWIS (e.g., 2011 for the 2012 data call).

The number of staging tables does not change with the new XML schema. The number of records changes greatly in the Sampling Results table, due to the addition of six new analytes. The structure of the Inventory staging table changes very slightly due to the introduction of geographic information.

See [Appendix A](#) for sample Staging Tables.

Steps for Staging Table Development

- a. Assemble “Inventory” table – The Inventory staging table has one record for each Community Water System (CWS) for each year that it actively provided service to its retail population, whether systems were active for an entire year or a portion therein. Given limitations in SDWIS the hope is that NCDMs will be generated for water systems currently active as well as those

that went inactive during the reporting period. CWS that are currently inactive most often consolidate with other water systems to mitigate ongoing and costly water quality issues.

Grantees, or cooperating data stewards at the state primacy agency, will extract the Inventory table directly from SDWIS or via data flow tailored for Tracking Water NCDMs by the USEPA Exchange Network. As with previous data calls, the Inventory staging table includes the following elements: system ID/name, year, principal city/county served, number of connections, population served, and primary type of source water.

Listed below are processing steps and associated data elements that change with the new XML schema:

1. For each inventory record, calculate geographic coordinates of approximate center of the retail service area. These coordinates will assist users in identifying water systems; they are not intended for water quality linkage analysis. Please see [Appendix B](#) - Guidance for Estimating Community Water System (CWS) Service Area Representative Point Locations for further reference.
 2. For each inventory record in which a representative geographic coordinate has been found, provide a code that describes the derivation technique used. These codes have been enumerated in the Data Dictionary and can be found in [Appendix B](#).
- b. Assemble water quality “Sampling Results” table – The Sampling Results staging table includes one record for each compliance sample for each of the ten tracked analytes that are attributable to each CWS in the Inventory table.

Water quality sampling data will be extracted directly from SDWIS or via data flow tailored for Tracking Water NCDMs by the USEPA Exchange Network. The structure of the Sampling Results staging table does not change with the new schema. Changes to the Sampling Results table come from appending water quality sampling results for the six new analytes. As with all previous data calls, the Sampling Results staging table includes the following elements: system ID, sampling date, sampling station ID, analyte code, concentration, concentration units, non/detection flag, and detection limit.

Listed below are some notable special cases and processing guidelines that should be adhered to when assembling the Sampling Results staging table:

- 1) All samples with results below the detection limit must have a non-zero and positive detection limit value provided. If a detection limit is not available from the source data, grantees are expected to estimate the detection limit from available data and/or provide a standard detection limit number. Guidance for determining detection limits when one is not provided is available in [Appendix D](#).
- 2) Compliance sampling done by drinking water wholesalers, that have interties with and sold to the CWS having a retail population, should be included in the Sampling Results staging

table, if SDWIS captures this information accurately and completely. Each importing CWS should be attributed with wholesalers' applicable sampling results data.

- 3) For Uranium samples that are reported in pCi/L, convert pCi to μg using the following conversion: $0.67 \text{ pCi}/\mu\text{g}$ (or $\text{pCi}/\text{L} \times 1.49 = \mu\text{g}/\text{L}$)

Note: The Water Team is tasked to develop SAS and SQL indicator calculation packages that take the two aforementioned Staging Tables as input and accomplishes Steps II and III below. These SAS and SQL programs will be developed by and shared among grantees. Furthermore, SQL programs will be developed with their use by CDC IT and/or Science Team in mind. Calculating summary-level measures at CDC using sample-level data submitted to CDC is a task in the work plan that will occur after the 2012 data call.

II. XML Dataset Development

This set of processing steps summarizes and formats the Staging Tables into two XML datasets that conform to the newly proposed XML schema. Please see Appendix E for example XML that have been validated for submission to CDC.

- a. The Inventory XML dataset is an annual list of each CWS that was actively delivering water to customers for the years of reporting; it is a direct copy in XML format of the Inventory staging table.
- b. The Water Quality XML dataset accommodates sample-level (optional) and summary-level (required) measures of water contaminant concentrations. This dataset embodies the largest changes to the XML schema and captures all of the information that was previously captured in the Annual and Quarterly Water Quality datasets of previous NEPHTN data calls. As an aside, this reduction of XML datasets also results in a reduction of accompanying metadata. The Water Quality dataset has a finite number of columns, and incorporates a hierarchical data structure, such that including additional analytes in future data calls will not require additional XML schema changes. The geographic unit of sample-level records is the sampling station. The geographic unit of summary-level records is the CWS. The reader is referred to the Data Dictionary as reference in further understanding this structure. The current Data Dictionary can be found on SharePoint at:

https://ephtn.sharepointsite.net/datasubmission/NCDM_Docs/Forms/AllItems.aspx.

Listed below are processing steps that are affected by changes to the new XML schema:

- 1) **(Optional)** Append all observations from Sampling Results staging table and code these records as "sample-level" data. At least for the 2012 data call, this step is only applicable to grantees who wish to pilot the submission of sample-level data to CDC.
- 2) Grouping by all analytes, all years, and all CWS in the Sampling Results staging table (or using the sample-level data that was added in [Step II.b.1](#) above), calculate annual mean and maximum concentration values, count of unique samples, count of unique sampling

stations, count of samples that resulted in non-detect, and date of last sample. Append these annual measures to dataset and code these records as “summary-level” data with an “annual” time period type. For samples coded as non-detect a concentration of half the detection limit is used before summarizing.

- 3) In the same manner as [Step II.b.2](#) above, calculate CWS-level quarterly mean concentration values, count of unique samples, count of unique sampling stations, count of samples that resulted in non-detect, and date of last sample for Nitrate, Atrazine, TTHM, and HAA5 only.

Special note for Steps II.b.2 & II.b.3 Above: For Nitrate, Arsenic, and the six new analytes, annual [and quarterly for Nitrate and Atrazine only] average concentration values are derived from first averaging by sampling station, then averaging by CWS. For disinfection-by-products (TTHM and HAA5) annual and quarterly average concentration values are derived from first averaging by day, then by CWS. Maximums for all 10 analytes are derived by taking the annual maximum for each CWS.

III. State-Level NCDM Development

This set of processing steps further summarizes the CWS-level measures (calculated in [Step II](#)) into statewide frequencies of water systems and summed population-served by analyte-specific concentration categories. The data produced in this step are not submitted to CDC.

Standard concentration categories have not been specified by CDC for each analyte in previous data calls. However, previous SAS indicator packages have suggested and used benchmarks relative to the regulatory Maximum Contaminant Level (MCL) for each analyte. In practice, grantees and CDC have been afforded the option of choosing which analyte-specific concentration categories work best for them.

Listed below are the steps for producing State-level annual and quarterly NCDMs:

- a. Joining the Inventory ([Step II.a](#)) and annual CWS-level records from the Water Quality dataset (see [Step II.b.2](#)) and grouping by: (1) analyte-specific concentration categories, (2) by all analytes, and (3) by all years, calculate State-level annual frequencies of CWS and summed population-served.
- b. In the same manner as [Step III.a](#) above, and using quarterly CWS-level records from the Water Quality dataset (see [Step II.b.3](#)), calculate state-level quarterly frequencies of CWS and summed population-served for Nitrate, Atrazine, TTHM, and HAA5 only.

Appendix A - Sample Staging Tables

PWS Inventory

PWSIDNumber	<u>YearAssociatedTo</u>	<u>YearPulled</u>	PWSName	PrincipalCountyServedFI PS	PrincipalCityFeatureID	TotalConnections	SystemPopulation	PrimarySourceCode	Latitude	Longitude	LocationDerivationCode
NH1234567	2011 0	<u>2012</u>	wild acres development	33003	873526	375	75 0	SWP	44.1 467	-72.5537	SA
NH0023010	2011 0	<u>2012</u>	green pine mobile home park	33019	873525	100	20 0	GW	42.9 725	-71.4385	MFL

Sampling Results

PWSIDNumber	DateSampled	SamplePointID	AnalyteCode	Concentration	ConcentrationUnits	NonDetectFlag	DetectionLimit
NH1234567	11/28/2001	501	1040	0	mg/L	1	0.001
NH1234567	10/9/2002	502	1005	5	µg/L	0	0.001
NH0012345	10/28/2003	501	2950	15	µg/L	0	0.001
NH0012345	10/25/2004	501	2456	25	µg/L	0	0.001

Appendix B

Guidance for Estimating Community Water System (CWS) Service Area Representative Point Locations

This appendix provides a recommended methodology for EPHT State grantees to estimate a central and representative point location for each CWS that is reported to CDC by grantees in annual data calls. The CDC public portal will display these point locations to orient users while navigating dynamic maps and to facilitate identification of a CWS relative to the approximate retail population in which it serves. It is expected that these point locations will be displayed in relatively low resolution at scales small enough to depict regional differences, like county-, state-, or national-scale maps. These locations are not expected to be precise enough or intended to be used in linking health outcome information to water quality measures; they are only intended for diagrammatic purposes.

Grantees are required to report to CDC representative service area locations for each CWS. Grantees, however, are not required to follow this methodology or to use the recommended data sources. This document is provided to enumerate the possible derivation methods, given grantees' collective experience and expertise and when assuming that the described data sources exist at grantee sites. It is not an exhaustive list of methods or data sources. Depending on capacity and data sources available at each individual state, a grantee may choose a different methodology or data sources to accomplish the required objective.

As per the Drinking Water XML schema, all point locations shall be reported in North American Datum 1983 decimal degrees coordinates.

General Methodology: Coordinates for all water system locations are compiled for each available data source. Data sources are prioritized in order of increasing precision. The Inventory dataset is updated according to this prioritization, using coordinates that are likely to be less precise, if coordinates of water systems from higher priority data sources are not available.

Complexities not addressed by this document: The Inventory dataset includes an annual record for each CWS, if the CWS was active for at least a portion of the reported year. This document, however, does not address annual changes in the water system location. If grantee water quality and location-related data sources can track annual changes in water system service area locations, then grantees are expected and it is their responsibility to accurately depict these changes in the Inventory dataset.

Ordered below are source datasets containing geographic locations for individual water systems, prioritized from highest to lowest spatial precision. Each source dataset has a corresponding LocationDerivationCode value, which should be used in the corresponding field of the Inventory dataset.

- I. *Service area polygon centroids* (LocationDerivationCode=SA) – Increasingly, States are working to assemble polygonal boundaries that approximate the retail service area of public water systems. As a GIS layer, the geometric center (or centroid) can be quickly calculated using common GIS tools. For example, the following article from the ESRI Support website describes

how to create and update two fields that describe the centroid of a polygonal layer: <http://support.esri.com/en/knowledgebase/techarticles/detail/32482>. For large water systems that have uneven population distribution, grantees may wish to use a population-weighted centroid, by intersecting water system polygons with Census Block centroids and proportionally weighting each centroid by its contribution to the total population within the service area polygon. Further population-weighted refinements can be accomplished by assuming that people only live within an arbitrary distance (e.g., 500ft) of a street centerline network, and similarly intersecting the resulting buffered segments with service area polygons.

- II. *Mean of water system facility locations* (LocationDerivationCode=MFL) – State drinking water primacy agencies typically track geographic locations of important facilities at public water systems. Some of these facility types are often situated proximate to the retail population. These include groundwater wellheads, treatment plants, and distribution system sampling stations. If it is assumed that these types of facilities are close to the retail population, then we can use their mean center as a proxy for the representative system location. However, because of the sensitivity of some of these facilities, a non-disclosure agreement may be required to release the facility locations to the grantee. Grantee liaisons to the State primacy agency should communicate and reiterate to the data steward that the use of sensitive coordinates is undertaken in a secure domain. Any derived locational information that is ultimately made public also completely masks the true geography of the original sensitive facility locations.

Per water system, a centroid or mean location can be calculated from a group of facility locations. If only one facility location is available and data stewards have strict confidentiality requirements for this location, grantees can reduce precision on the points (e.g., to the nearest hundredth of a decimal degree) or use the GeoMasking tool (See [Appendix C](#)) provided by the Geospatial Workgroup and downloadable on the EPHTN SharePoint site to randomly skew the point within an arbitrary distance threshold (e.g., between 200 and 500 meters). This tool requires the use of a polygonal layer within which the resulting point is constrained; county administrative boundaries trimmed of water features would serve this purpose satisfactorily.

- III. *Principal city served* (LocationDerivationCode=PCS) – The Geographic Names Information System (GNIS - <http://geonames.usgs.gov>) place code for each water system's principal city served is already an element in the Inventory dataset. Each GNIS entity has a corresponding latitude/longitude in NAD83 decimal degrees that can be used to approximate a water system's service area. Alternatively, grantees can use their own State's placename database or a geocoding service to derive coordinates from the principal city served
- IV. *Geocoded water system address* (LocationDerivationCode=GSH) – Water quality databases often include contact addresses for each CWS. Grantees can use in-house geocoding expertise or an external service like BatchGeo.com or Google Fusion Tables (http://earth.google.com/outreach/tutorial_fusion_yourowndata.html) to infer coordinate locations from the water system address. To filter contact addresses that are not proximate to the water system service area, compare the geocoded county of the contact address to the

5 July, 2012

principal county served (which is typically a substring within the PWSID). Contact addresses for CWS are usually proximate to their service area, but sometimes, and especially for private water systems that serve multiple jurisdictions, a water system contact address is the system's billing address and can be situated far from the retail service area.

- V. *Principal County served* (LocationDerivationCode=PNS) – Using GNIS or commercial data sources, grantees can make use of the centroid of county regions. In the absence of locations from the previous 4 data sources, this location might be useful for some states that have smaller counties, in which very few water systems serve the population of a single county. In western states this data source will not likely be useful, since these regions can be very large and can be expected to capture too many water systems.
- VI. *Other* (LocationDerivationCode=O) – Location derived by some method not outlined above, e.g., zip code, etc. Please specify what O is in your metadata file.

5 July, 2012

Appendix C - GeoMasking Tool

A PowerPoint demonstration of New York State Department of Health's GeoMasking Tool can be found on SharePoint at:

http://ephtn.sharepointsite.net/geowkgp/Community_Mapping/GeoMasking_Tool/GeoMask_Tool_7_11.pdf

The GeoMasking Tool described in the presentation above is available on SharePoint at:

http://ephtn.sharepointsite.net/geowkgp/Geo_Wiki/Community%20Mapping%20Team.aspx

5 July, 2012

Appendix D - Determining appropriate values for non-detects when no detection limit is provided.

This guidance is provided to assist grantees in substituting detection limit (DL) values in the Sampling Results Staging Table for observations in which DL values were not provided by their SDWIS data steward.

1. Using sampling observations that already have analyte-specific DLs specified, take the annual median DL for each unique lab and substitute this value for non-detect observations lacking a DL for the same year and same lab.
2. Using sampling observations that already have analyte-specific DLs specified, take the annual median DL and substitute this value for non-detect observations not updated in (1) and lacking a DL for the same year.
3. For the remaining non-detect observations lacking a DL and not updated in (1) or (2), please refer to the Excel file on Share Point, Analyte Detection Methods (https://ephtn.sharepointsite.net/datasubmission/NCDM_Docs/Analyte_detection_methods_1_6_2012.xlsx) for an appropriate DL.

Appendix E – Sample XML validated for submission to CDC

Pasted below are validated examples of the Inventory and Water Quality Levels datasets. Only one observation is included in the Inventory dataset and only two observations are included in the Water Quality Levels dataset (one for sample- and one summary-level data). Please see the Data Dictionary for additional description of allowable values and variable formats. Please refer to the actual schema files provided on the EPHTN SharePoint site as an authoritative resource for XML syntax, including element naming and sequence.

I. Inventory dataset:

```
<?xml version="1.0" encoding="windows-1252" ?>
<PWSInventory xmlns="http://www.ephtn.org/NCDM/ENV/PWSInventory">
  <Header>
    <MCN>4b956457-b578-41c4-b274-f067ba0430da</MCN>
    <JurisdictionCode>CA</JurisdictionCode>
    <ContentGroupIdentifier>PWSINVENTORY</ContentGroupIdentifier>
    <SubmitterInformation>
      <SubmitterEmailAddress>craig.wolff@cdph.ca.gov</SubmitterEmailAddress>
      <SubmitterName>Craig Wolff</SubmitterName>
      <SubmitterTitle>IT/GIS Director</SubmitterTitle>
    </SubmitterInformation>
    <StateFIPSCode>06</StateFIPSCode>
  </Header>
  <Dataset>
    <Row>
      <RowIdentifier>1</RowIdentifier>
      <PWSIDNumber>CA0103040</PWSIDNumber>
      <YearAssociatedTo>2011</YearAssociatedTo>
      <YearPulled>2011</YearPulled>
      <PWSName>NORRIS CANYON PROPERTY OWNERS ASSN.</PWSName>
      <PrincipalCountyServedFIPS>06001</PrincipalCountyServedFIPS>
      <PrincipalCityFeatureID>1658237</PrincipalCityFeatureID>
      <TotalConnections>19</TotalConnections>
      <SystemPopulation>100</SystemPopulation>
      <PrimarySourceCode>GW</PrimarySourceCode>
      <Latitude>
        <LatitudeRange>37.734364</LatitudeRange>
        <!-- comment about LatitudeNS element -->
      </Latitude>
      <Longitude>
        <LongitudeRange>-122.027303</LongitudeRange>
      </Longitude>
    </Row>
  </Dataset>
</PWSInventory>
```

5 July, 2012

```
<!-- comment about LongitudeNS element -->
</Longitude>
<LocationDerivationCode>SA</LocationDerivationCode>
</Row>
</Dataset>
</PWSInventory>
```

II. Water Quality Levels dataset (first row is an optional sample-level observation and second row is a summary-level observation)

```
<?xml version="1.0" encoding="windows-1252" ?>
<WaterQualityLevels xmlns="http://www.ephtn.org/NCDM/ENV/WaterQualityLevels">
  <Header>
    <MCN>f4ba15e2-3933-456f-a7d9-7edbac6fd19e</MCN>
    <JurisdictionCode>CA</JurisdictionCode>
    <ContentGroupIdentifier>WQL</ContentGroupIdentifier>
    <SubmitterInformation>
      <SubmitterEmailAddress>craig.wolff@cdph.ca.gov</SubmitterEmailAddress>
      <SubmitterName>Craig Wolff</SubmitterName>
      <SubmitterTitle>IT/GIS Director</SubmitterTitle>
    </SubmitterInformation>
    <StateFIPSCode>06</StateFIPSCode>
  </Header>
  <Dataset>
    <Row>
      <RowIdentifier>1</RowIdentifier>
      <PWSIDNumber>CA0103040</PWSIDNumber>
      <Year>2000</Year>
      <AnalyteCode>4006</AnalyteCode>
      <DateSampled>2000-08-08</DateSampled>
      <SamplePointID>0103040-001</SamplePointID>
      <DetectionLimit>0.000000</DetectionLimit>
      <NonDetectFlag>0</NonDetectFlag>
      <ConcentrationUnits>ug/L</ConcentrationUnits>
      <Concentration>4.5075</Concentration>
    </Row>
    <Row>
      <RowIdentifier>43571</RowIdentifier>
      <PWSIDNumber>CA0103040</PWSIDNumber>
```


5 July, 2012

```
<Year>2000</Year>  
<AnalyteCode>4006</AnalyteCode>  
<DateSampled>2000-08-08</DateSampled>  
<AggregationType>MX</AggregationType>  
<NumSamplingLocations>1</NumSamplingLocations>  
<SummaryTimePeriod>2000</SummaryTimePeriod>  
<NumSamples>1</NumSamples>  
<NumNonDetects>0</NumNonDetects>  
<ConcentrationUnits>ug/L</ConcentrationUnits>  
<Concentration>4.5075</Concentration>  
</Row>  
</Dataset>  
</WaterQualityLevels>
```

Content Domain: Drinking Water Quality

**Recommendations for
Nationally Consistent Data and Measures**

Part 2: Recommended Data Sets

Last Revised: January 14, 2014

**Environmental Public Health Tracking
Drinking Water Quality
PUBLIC DRINKING WATER QUALITY DATASET:
PWS Inventory, Drinking Water Quality Sampling Results**

Characteristic	Description
Data Sources	Safe Drinking Water Act/State Drinking Water Information System (SDWA/SDWIS), or SDWIS-like Data System
Purpose	<p>This data set contributes to the Environmental Public Health Tracking Network. The EPHT cooperative agreement states that “by September 30, 2008 [...all grantees must] track and make available core environmental health tracking measures on the State and National EPHT Network [...including ...] data/information on key water contaminants, as defined through the Content workgroup process.” The Content Workgroup Water Team identified initial contaminants of concern for the national EPHT program, identified nationally consistent data sources, and developed nationally consistent indicators and measures. This data set can be used to calculate the nationally consistent measures for the initial contaminants of concern.</p> <p>This data set contains the information needed to calculate Environmental Public Health Tracking (EPHT) measures of contaminants in public water supply for arsenic, disinfection byproducts, nitrates, atrazine, di(2-ethylhexyl) phthalate (DEHP), radium, tetrachloroethene (tetrachloroethylene) (PCE), trichloroethene (trichloroethylene (TCE), and uranium. Data are derived from state Safe Drinking Water Act databases. The data set consists of two tables:</p> <ol style="list-style-type: none"> 1. PWS Inventory. This file is required and contains descriptive and locational information about each public water system (PWS) with which water quality data is provided. This dataset should only include Community Water Systems (CWS) as defined and regulated by the Safe Drinking Water Act. It does not include Non-Transient Non-Community (NTNC) and Transient Non-Community water systems (TNC). There is one record for every year that a CWS was active, delivering drinking water to customers, and in which water quality data is complete. CWS that were once active and are currently inactive should be included if State's data support this scenario. 2. Drinking Water Quality Sampling Results. This file is required and contains one record for each community water system (CWS) for the mean and maximum concentrations per year of each of arsenic, disinfection byproducts, nitrates, atrazine, di(2-ethylhexyl) phthalate (DEHP), radium, tetrachloroethene (tetrachloroethylene) (PCE), trichloroethene (trichloroethylene (TCE), and uranium; and the mean concentrations per quarter of disinfection byproducts, nitrates and atrazine. This dataset also accommodates sample-level data and includes one record for each compliance sample for the same analytes used in calculating summary concentrations. Some fields (e.g. NumSamplingStations) are only included for summary-level data observations and belong to the schema group “SummaryLevelGroup”. Other fields (e.g. DetectionLimit) are only included for sample-level data observations and belong to the schema group “SampleLevelGroup”. Sample-level observations are not required as part of the Spring 2012 CDC data submission; Associated optional fields for sample-level data are indicated as such in the Optionality column.
Restrictions	This is <u>not</u> a restricted access data set.

PWS Inventory

Header				
Field Name/SchemaName	Field Description	Optionality	Format	Allowed Values
StateFIPSCode	State FIPS code	Required	AN(2)	FIPS State Code
Table Core		Optionality		
SchemaName	Field Description		Format	Allowed Values
PWSIDNumber	PWS identifier	Required	AN(9)	nine character value consisting of the 2 letter state abbreviation followed by 7 numbers
YearAssociatedTo	Year that these data are associated with regards to sampling results	Required	Text(4)	YYYY. 1999 through latest complete year (e.g. 2011)
YearPulled	Year that these data were pulled from state records	Required	Text(4)	YYYY. 1999 through latest year.
PWSName	Name of PWS	Required	AN(40)	Any; "U" = Unknown; "NS" = Not submitted
PrincipalCountyServedFIPS	Principal county FIPS served by the CWS	Required	AN(5)	Any; "U" = Unknown; "NS" = Not submitted
PrincipalCityFeatureID	Principal city, town or village Feature ID served by the CWS	Required	N(10)	9999999999;"-999" for Missing; "-888" for Not Submitted Feature ID can be obtained from: http://geonames.usgs.gov/domestic/download_data.htm
TotalConnections	Number of residential service connections	Required	N(7)	1-9999999"
SystemPopulation	Permanent population uniquely served by the CWS	Required	N(8)	10-99999999 "
PrimarySourceCode	Type of source	Required	AN(3)	GU = ground water under direct influence of surface water, GUP = purchased ground water under direct influence of surface water, GW = ground water, GWP = purchased ground water, SW = surface water, SWP = purchased surface water; "U" = Unknown; "NS" = Not submitted
Latitude	Latitude in NAD83 decimal degrees describing approximate center of retail service area of water system	Required	N(10)	00.0000000 to 90.0000000;"-99.99" for Missing; "-88.88" for Not Submitted.

Longitude	Longitude in NAD83 decimal degrees describing approximate center of retail service area of water system	Required	N(11)	-180.000000 to 180.000000; "-999" for Missing; "-888" for Not Submitted.
LocationDerivationCode	Code describing how approximate latitude/longitude location was derived	Required	AN(3)	SA = Service area polygon centroid; MFL = Mean of 1 or more facility locations that are expected to be proximate to service area extent; PCS = GNIS coordinates for Principal City Served; GSH = The geocoded address of water system headquarters; PNS - GNIS coordinates for Principal County Served; O= Other (e.g. zip code, etc.) "-999" = Missing; "-888" = Not Submitted; (See "Appendix A. Service Area Location Derivation Guidance of the How-To Guide" on EPHTN Share Point site for more information & guidance for deriving water system locations.)

Drinking water quality sampling results

Header					
Field Name/SchemaName	Field Description	Optionality	Schema Group	Format	Allowed Values
StateFIPSCode	State FIPS code	Required	NA	AN(2)	FIPS State Code
Table Core					
SchemaName	Field Description	Optionality	Schema Group	Format	Allowed Values
PWSIDNumber	PWS identifier	Required	NA	AN(9)	Nine character value consisting of the 2 letter state abbreviation followed by 7 numbers
Year	Year	Required	NA	Text(4)	YYYY; 1999 through latest complete year (e.g. 2011)
AnalyteCode	USEPA Analyte code for required constituents (arsenic, nitrate, TTHM, HAA5, atrazine, PCE, TCE, DEHP, radium, and uranium)	Required	NA	N(4)	1005=Arsenic; 2050=Atrazine; 2456=HAA5; 2950=TTHM; 2039=DEHP; 1040=Nitrate; 2987=PCE; 2984=TCE; 4010=Combined Radium 226 & 228; 4006=Uranium (see How-To-Guide for converting gross alpha particle activity to U in ug/L)
ConcentrationUnits	The analyte-specific units of summary-level measures and individual sample values as reported in the Concentration and DetectionLimit fields. Each analyte has a standard unit for this dataset.	Required	NA	AN(6)	"ug/L" (Arsenic, TTHM, HAA5, Atrazine, DEHP, PCE, TCE, uranium); "mg/L" (Nitrate as nitrogen); "pCi/L" (Radium)
Concentration	Reported summary-level concentration or reported concentration of sample	Required	NA	6.4	>0 for summary-level measure or sample-level concentration; -888, if sample-level data and NonDetectFlag=1
DateSampled	Date of sample (sample-level data) or Date last sampled (summary-level data)	Required	NA	YYYY-MM-DD	A valid date from 1/1/1999 through December 31 st of the latest complete year (e.g. 2011-12-31).

SamplePointID	Sampling station identifier for sample-level records only.	Optional	SampleLevelGroup	AN(20)	Character ID for sampling station; "-999" for missing; "-888" for Not Submitted
DetectionLimit	Sample detection limit	Optional	SampleLevelGroup	3.6	>0, if NonDetectFlag=1; -888, if NonDetectFlag=0.
NonDetectFlag	Flag to indicate whether sample resulted in a detection or not	Optional	SampleLevelGroup	N(1)	1=Sample was a non-detect; 0=Sample was a detection
AggregationType	The type of summary operation performed (i.e. mean or max) for summary-level data.	Required	SummaryLevelGroup	AN(3)	"X" = Mean; "MX" = Maximum
NumSamplingLocations	Number of compliance sampling locations available from which summary-level records were derived.	Required	SummaryLevelGroup	N(4)	1-9999; "-888" for Not Submitted
SummaryTimePeriod	Year or Quarter for summary-level data	Required	SummaryLevelGroup	AN(10)	YYYY for annual summarized values; YYYY-Q for quarterly summarized values
NumSamples	The number of samples that were used in calculating the mean/max for a given analyte during a quarter or year.	Required	SummaryLevelGroup	N(4)	1-XXXX

NumNonDetects	The number of samples that were non-detections for summary-level data.	Required	SummaryLevelGroup	N(4)	0-XXXX (XXXX must be no greater than NumSamples)
---------------	--	----------	-------------------	------	--

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: PUBLIC WATER USE
ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Exposure
Measures	1. Number of people receiving water from community water systems.
Derivation of Measures	This measure will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format.
Units	1. Number of people
Geographic Scope	State
Geographic Scale	State
Time Period	2009 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Public Water Use and Public Health</p> <p>The public water use index provides some data to explore the relative importance of community water supplies as sources of drinking water and to provide context for subsequent community drinking water system (CWS) indicators. SDWA collects data for a number of different types of public water systems of which community water systems (CWS) are a sub-set. The community water systems represent non-transient public water systems that serve year round community residents and are the focus of the initial indicators. The range of state populations served by CWS as their primary residential drinking water source varies from 95% to as low as 40% within the United States. Understanding the relative population coverage of these indicators by state helps to understand representativeness of these data for prioritization and evaluation across the United States and within individual states and communities.</p>
Use of Measure	<p>This measure can be useful in providing data for surveillance purposes.</p> <ul style="list-style-type: none"> • Estimated population potentially exposed to contaminants in CWS.
Limitations of The Measure	The current measure is derived for CWS only. Private wells are another important source of population exposure to water contaminants. Transient non-community water systems, which are regulated by EPA, may also be an important source of potential exposure.
Data Sources	State grantee

Limitations of Data Sources	Population estimates are rough and may overestimate or underestimate the number of affected people.
Related Indicators	All other community water indicators.
Additional Information	<ol style="list-style-type: none">1. U.S. Environmental Protection Agency, <i>Water On Tap</i>, Office of Water (4601) EPA 816-K-09-002, December 2009. http://water.epa.gov/drink/guide/upload/book_waterontap_full.pdf2. U.S. Environmental Protection Agency, Public Drinking Water Systems: Facts and Figures http://water.epa.gov/infrastructure/drinkingwater/pws/factoids.cfm3. U.S. Environmental Protection Agency, Public Drinking Water Systems Programs. http://water.epa.gov/infrastructure/drinkingwater/pws/index.cfm

CONTENT DOMAIN: COMMUNITY WATER**INDICATOR: ARSENIC****ENVIRONMENTAL PUBLIC HEALTH TRACKING**

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of Community Water Systems (CWS) by maximum arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic). 2. Yearly distribution of number of CWS by mean arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic). 3. Mean concentration of arsenic at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 4. Yearly distribution of number of people served by CWS by maximum arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic). 5. Yearly distribution of number of people served by CWS by mean arsenic concentration (cut-points: 0-5, >5-10, >10-20, >20-30, >30 µg/L arsenic).
Derivation of Measures	Arsenic measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	Concentration of arsenic, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year

<p>Rationale</p>	<p>Arsenic and Public Health</p> <p>Exposures to higher than average levels of arsenic can come from elevated localized soil and ground water concentrations from application and runoff of arsenical pesticides and leachate from coal ash and landfills (ATSDR 2005). Exposure to hundreds of micrograms per liter of arsenic found in drinking water of Taiwan, Chile, Argentina, Mexico, Bangladesh, and India has been associated with many adverse health effects including lung, bladder, liver and skin cancers (NRC, 1999; Rahman et al. 2005; Salazar et al. 2004; Fazal et al., 2001). Arsenic has been identified as a human carcinogen by the International Agency for Research in Cancer (IARC) (IARC, 2004). Other adverse health effects include nausea, cardiovascular disease, (Chen et al., 2007; Chih-Hao et al., 2007; Bunderson et al., 2004), developmental and reproductive effects (Hopenhayn et al., 2003; Ahmad et al., 2001), Diabetes Mellitus (Rahman et al., 1998), and skin keratosis and hyperpigmentation (Kapaj et al., 2006).</p> <p>Measured arsenic concentrations in finished drinking water can be used to understand the distribution of potential arsenic exposure levels for populations served by community water supplies. These measures allow for comparison of potential for arsenic exposures between the populations served by different water systems and water sources over time, and potentially across demographic groups.</p> <p>Sources of Arsenic</p> <p>Arsenic compounds (As (III) and As (V)) are found in both ground water and surface waters. The primary sources are geologic formations from which arsenic can be dissolved. Higher levels of arsenic tend to be found in ground water (e.g. aquifers) as compared to surface waters (e.g., lakes, rivers).</p> <p>Arsenic Regulation and Monitoring</p> <p>In 2001 EPA reduced the regulatory drinking water standard Maximum Contaminant Level (MCL) to 10 µg/L from 50 µg/L (effective January 23, 2006) on the basis of bladder and lung cancer risks (EPA 2001a). The cancer risks were extrapolated from the Taiwanese (Chen et al. 1985) study to U.S. risks. Lowering the MCL from 50 to 10 ppb statistically reduces bladder and lung cancer mortality and morbidity by 37-56 cancers a year in the U.S. (EPA 2001b). Based on the current understanding of the health impacts from arsenic exposure, the potential for adverse health effects from drinking water exposure to arsenic is very low for most municipal drinking water systems.</p>
<p>Use of Measure</p>	<p>These measures can assist by addressing the following surveillance functions:</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and

	<p>the number of people potentially exposed to arsenic at different concentrations.</p> <ul style="list-style-type: none"> • Maximum concentrations provide information on the peak potential exposure to arsenic at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	<p>Samples are taken once a year (surface sources), once every three years (groundwater sources), or once every nine years (for sources with a waiver). Frequency of sampling is based on compliance with the MCL; the lower the measured concentration the fewer samples will be taken and some years there may be no sampling for arsenic.</p> <p>Ground water systems may have multiple wells with different arsenic concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the arsenic concentration of people served by wells with higher arsenic concentrations.</p> <p>Exposure may be higher or lower than estimated if data from multiple entry points for water with different arsenic levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. Ahmad SA, Sayed MH, Barua S, Khan MH, Faruquee MH, Jalil A, Hadi SA, Talukder HK., 2001. Arsenic in drinking water and pregnancy outcomes. <i>Environmental Health Perspectives</i>; 109(6):629-31. 2. ATSDR 2005. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. Draft for Public comment. September 2005. Available at http://www.atsdr.cdc.gov/toxprofiles/tp2.html 3. Bunderson M, Brooks DM, Walker DL, Rosenfeld ME, Coffin JD, Beall HD., 2004. Arsenic exposure exacerbates atherosclerotic plaque formation and increases nitrotyrosine and leukotriene biosynthesis. <i>Toxicology and Applied Pharmacology</i> 2004 Nov 15;201(1):32-9. 4. Chen C-J, Chuang Y-C, Lin T-M, Wu H-Y. Malignant neoplasms among residents of a blackfoot disease-endemic area in Taiwan: high-arsenic well water and cancers.

- Cancer Res. 1985;45:5895–5899.
5. Chen Y., Factor-Litvak P., Howe GR., Graziano JH., Brandt-Rauf P., Parvez F., van Geen A., Ahsan H., 2007. Arsenic exposure from drinking water, dietary intakes of B vitamins and folate, and risk of high blood pressure in Bangladesh: a population-based, cross-sectional study. *American Journal of Epidemiology*, Mar 1;165(5):541-52
 6. Chih-Hao Wang, Chuhsing Kate Hsiao, Chi-Ling Chen, Lin-I Hsu, Hung-Yi Chiou, Shu-Yuan Chen, Yu-Mei Hsueh, Meei-Maan Wu, Chien-Jen Chen
2007. A review of the epidemiologic literature on the role of environmental arsenic exposure and cardiovascular diseases. *Toxicology and Applied Pharmacology* 222 (2007) 315–326.
 7. Fazal MA, Kawachi T, Ichion E. Extent and severity of groundwater arsenic contamination in Bangladesh. *Water International* (2001) 26:370–79
 8. Hopenhayn C, Ferreccio C, Browning SR, Huang B, Peralta C, Gibb H, Hertz-Picciotto I., 2003. Arsenic exposure from drinking water and birth weight. *Epidemiology*;14(5):593-602.
 9. IARC 2004. Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 84. Some Drinking Water Disinfectants and Contaminants, including Arsenic. September Lyon, pp. 39-267. Available at <http://monographs.iarc.fr/ENG/Monographs/vol84/volume84.pdf>
 10. Kaltreider RC, Davis AM, Lariviere JP, Hamilton JW. 2001. Arsenic alters the function of the glucocorticoid receptor as a transcription factor. *Environ. Health Perspect* 109:245-251.
 11. Kapaj S., Peterson H., Liber K., Bhattacharya P., 2006. Human health effects from chronic Arsenic poisoning-A review, *Journal of Environmental Science and Health, Part A*;41(10):2399-2428.
 12. Karagas M, Stukel TA, Tosteson TD. 2002. Assessment of cancer risk and environmental levels of arsenic in New Hampshire. *Int. J. Hyg. Environ. Health* 205, 85-94.
 13. NCI .2003. Ries LAG, Eisner MP, Kosary CL, Hankey BF, Miller BA, Clegg L, Mariotto A, Fay MP, Feuer EJ, Edwards BK (eds). SEER Cancer Statistics Review, 1975-2000, National Cancer Institute (NCI). Bethesda, MD, http://seer.cancer.gov/csr/1975_2000/, 2003.
http://seer.cancer.gov/csr/1975_2000/results_single/sect_01_table.01.pdf
 14. National Research Council. Arsenic in Drinking Water, National Academy Press. 1999.
 15. Rahman M., Tondel M., Ahmad SA., Axelson O., 1998. Diabetes Mellitus associated with Arsenic exposure in Bangladesh. *American Journal of Epidemiology*, Vol 48, no2, pp 198-203.
 16. Rahman MM, Sengupta MK, Ahamed S, et al. Arsenic contamination of groundwater and its health impact on residents in a village in West Bengal, India. *Bulletin of the World Health Organization* (2005) 83:49–57.
 17. Salazar et al. 2004. p53 Expression in circulating lymphocytes of non-melanoma skin cancer patients from an arsenic contaminated region in Mexico. A pilot study. *Molecular and Cellular Biochemistry* 255:25-31.
 18. U.S. EPA 2000a. Arsenic in drinking water proposal, 65 FR 38888 at 38894 and 38897, June 22, 2000, available at <http://www.epa.gov/fedrgstr/EPA-WATER/2000/June/Day-22/w13546.htm> and http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=2000_register&docid=00-13546-filed.pdf

	<p>19. U.S. EPA 2000b. Arsenic in Drinking Water Rule Economic Analysis December 2000, available at http://www.epa.gov/safewater/arsenic/history.html</p> <p>20. U.S. EPA 2001a. Arsenic in drinking water final rule, 66 FR 6976. Available at http://www.epa.gov/fedrgstr/EPA-WATER/2001/January/Day-22/w1668.htm</p> <p>21. U.S. EPA 2001b. Quick Reference Guide for Arsenic. EPA 816-F-01-004. Available at http://www.epa.gov/safewater/arsenic/pdfs/quickguide.pdf</p> <p>22. U.S. EPA 2001c. Arsenic in drinking water Technical fact sheet. Available at http://www.epa.gov/ogwdw/arsenic/regulations_techfactsheet.html</p> <p>23. U.S. EPA 2004. Estimated per Capita Water Ingestion and Body Weight in the United States – an Update. EPA-822-R-00-001. October 2004. Available at http://epa.gov/waterscience/criteria/drinking/percapita/2004.pdf</p> <p>24. U.S. EPA 2005. Factoids: Drinking Water and Ground Water Statistics for 2004. EPA Office of Water. EPA 816-K-05-001. May 2005. Available at http://www.epa.gov/safewater/data/pdfs/data_factoids_2004.pdf</p>
--	---

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: ATRAZINE
ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Quarterly distribution of number of Community Water Systems (CWS) by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 2. Yearly distribution of number of CWS by maximum atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 3. Yearly distribution of number of CWS by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 4. Mean concentration of atrazine at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 5. Quarterly distribution of number of people served by CWS by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 6. Yearly distribution of number of people served by CWS by maximum atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine). 7. Yearly distribution of number of people served by CWS by mean atrazine concentration (cut-points: 0-1, >1-3, >3-4, >4 µg/L atrazine).
Derivation of Measures	Atrazine measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	µg/L of Atrazine
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year

Rationale	<p>Atrazine and Public Health</p> <p>Atrazine is a widely used herbicide active against broadleaf and grassy weeds. Atrazine was first registered as an herbicide in 1958. More than 70 million pounds have been applied annually in recent years, with about 75% of corn cropland receiving treatment. In addition to agricultural uses, atrazine is used in residential turf applications and on golf courses and sod farms to control weeds. Atrazine and its degradation products are the most commonly detected pesticides in ground and surface waters (Barr et al., 2007). The frequent detection of atrazine and its degradation products in streams, rivers, groundwater, and reservoirs is related directly to the volume of its use, its persistence in soils due to its resistance to photolysis and hydrolysis, and its ability to travel within water systems (Nelson et al., 2001). In water systems, atrazine is transformed over time by various chemical reactions into other compounds or its degradation products or metabolites, including dealkylated compounds such as desethylatrazine (DEA), desisopropylatrazine (DIA), and diaminochlorotriazine (DACT). In soil, atrazine degrades slowly to dealkylated compounds, which have half-lives of several months. Bacteria and plants can metabolize atrazine to hydroxylated products. In plants, atrazine is absorbed by the root system and tends to form hydroxylated metabolites that cannot be removed by washing contaminated vegetables (Nelson et al., 2001). Atrazine does not bioaccumulate. Studies suggest that in animals, the degradation products that retain the chlorine have biologic activity similar to that of atrazine, while the hydroxylated metabolites do not retain its biologic activity (Nelson et al., 2001). Use of atrazine in the presence of nitrogen fertilizers, has raised a possibility of N-nitrosation in soil (DeMarini and Zahm, 1999). There may also be endogenous formation of N-nitrosoatrazine from precursors ingested in the diet and drinking water. For the general population, drinking water is an infrequent source of atrazine exposure, but estimates of seasonal intakes from drinking water in a small number of communities have exceeded the recommended limits (U.S. EPA, 2003). As a result, atrazine use has progressively been restricted in an effort to reduce surface and ground water contamination.</p> <p>In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, atrazine was detected in 888 systems serving greater than 34 million people (EPA, 2009). Concentrations of atrazine were greater than the MCL in 98 systems serving 3.1 million people. Atrazine was the second highest occurring regulated synthetic organic chemical found based on the percent of detections found from the 6 Year Review data (EPA, 2009).</p> <p>While it is used on many crops, atrazine has not been found in many food samples, and then only at very low levels. Therefore, it is very unlikely that people would be exposed to atrazine by eating crops from atrazine-accumulated soil.</p> <p>Most people are not exposed regularly to atrazine. People living near areas where atrazine was applied to crops may be exposed through contaminated drinking water. Atrazine has been found at about 20 Superfund sites in the United States. People living near those sites may be exposed to higher levels of atrazine. Factory workers who work with atrazine may be exposed to higher amounts of atrazine than other workers. The government has</p>
------------------	---

estimated that approximately 1,000 people may be exposed to atrazine in this way (ATSDR, 2003).

Applicators of atrazine may be exposed dermally and by inhalation. Atrazine is well absorbed orally, metabolized, and then eliminated in the urine over a few days (Bradway et al., 1982; Catenacci et al., 1993; Timchalk et al., 1990).

Metabolism of atrazine and its degradation products is complex and results in many potential metabolites (Barr et al., 2007). As many as 8-12 metabolites of atrazine have been identified in animals and humans, with recent studies showing DACT as the primary metabolite (Barr et al., 2007); therefore, earlier biomonitoring studies measuring atrazine mercapturate alone misrepresent and underestimate total atrazine exposure. Panuwet et al., (2008) developed an analytical method that measures the seven primary urinary metabolites of atrazine, which are: hydroxyatrazine, DACT, DIA, DEA, desethylatrazine mercapturate, atrazine mercapturate, and atrazine itself.

Human health effects of atrazine at environmental doses or at biomonitored levels from environmental exposure are unknown. In mammalian studies, atrazine is rated as having low acute toxicity. Atrazine product formulations can be mild skin sensitizers and irritants. Some human ecologic and epidemiologic studies of reproductive and cancer outcomes have shown either positive or no associations, but effects are difficult to attribute due to lack of exposure markers or due to mixed chemical or pesticide exposures (ATSDR, 2003; Gammon et al., 2005; Sathiakumar and Delzell, 1997). Studies of couples living on farms that use atrazine for weed control found an increase in the risk of pre-term delivery. These studies are difficult to interpret because most of the farmers were men who may have been exposed to several types of pesticides. A meta-analysis linked hypospadias to parental exposure to pesticides with possible endocrine-mediated effects (Rocheleau et al., 2009). Some epidemiological studies that looked at the potential impact of prenatal exposure to atrazine or its products of environmental degradation on pregnancy outcomes in the general population observed higher rates of babies born small-for gestational age (SGA) (Munger et al., 1997, Villanueva et al., 2005; Ochoa-Acuna et al., 2009). They also linked exposure of mothers who lived closer to sites with high atrazine concentrations with a higher risk of gastroschisis (Waller et al., 2010). Most of these studies were retrospective and relied on ecological assessment of exposure to atrazine. However, the most recent study that measured urinary biomarkers of prenatal atrazine exposure and was based on a prospective population-based cohort found associations between environmental exposure to atrazine and adverse effects on fetal growth, specifically birth weight, birth length, and small head circumference (Chevrier et al., 2011). Atrazine is not mutagenic and is not considered genotoxic. The International Agency for Research on Cancer (IARC) considers atrazine not classifiable with respect to human carcinogenicity, and the EPA considers atrazine unlikely to be a human carcinogen. However, IARC recommends future research to characterize the ability of atrazine to interfere with the hypothalamic-pituitary-ovarian axis in women. This research would help determine whether atrazine is a mammary carcinogen in women. Another area for future research is to explore atrazine's ability to alter immune and aromatase function in humans. Additional information is available from U.S. EPA at: <http://www.epa.gov/pesticides/> ; from ATSDR at: <http://www.atsdr.cdc.gov/toxpro2.html>, and IARC at <http://www.iarc.fr/>

Children are likely to be exposed to atrazine in the same way as adults, primarily through contact with dirt that contains atrazine or by drinking water from wells that are contaminated with the herbicide. Little information is available about the effects of atrazine in children. Maternal exposure to atrazine in drinking water has been associated with low fetal weight and heart, urinary, or limb defects in humans. It is not known whether atrazine or its metabolites can be transferred from a pregnant mother to a developing fetus through the placenta or from a nursing mother to her offspring through breast milk.

Biomonitoring Information

Urinary levels of atrazine mercapturate reflect recent exposure. In the NHANES 2001–2002 subsample, levels of atrazine mercapturate were generally not detectable (CDC, 2005). In small studies of Maryland residents in 1995–1996 (MacIntosh et al., 1999) and 83 Minnesota children with multiple urine collections during 1997 (Adgate et al., 2001), atrazine mercapturate was infrequently detected at the detection limit of 0.3 µg/L. In a study of 60 farm worker children, atrazine was detected in only four children (Arcury et al., 2007). Using immunoassay atrazine equivalents (detected mostly as atrazine mercapturate), the urinary geometric mean levels for herbicide applicators in Ohio and Wisconsin were about 6 µg/L (Hines et al., 2003; Perry et al., 2000). The geometric mean of urinary atrazine mercapturate was 1.2 µg/L in 15 farmers studied several days after spraying the pesticide (Curwin et al., 2005). In a small number of field workers, urinary concentrations ranged from 5–1756 µg/L (Lucas et al., 1993). However, biomonitoring studies that have evaluated only one urinary metabolite of atrazine (such as atrazine mercapturate) probably underestimated exposure (Barr et al., 2007).

Finding measurable amounts of atrazine or its metabolites in urine does not mean that the levels of atrazine and its metabolites (e.g., atrazine mercapturate) cause an adverse health effect. Biomonitoring studies on levels of atrazine mercapturate provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of atrazine than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

Sources of Atrazine

Atrazine is the common name for an herbicide that is widely used to kill weeds. It is used mostly on farms. Pure atrazine—an odorless, white powder—is not very volatile, reactive, or flammable. It will dissolve in water. Atrazine is made in the laboratory; it does not occur naturally.

Atrazine is used on crops such as sugarcane, corn, pineapples, sorghum, and macadamia nuts, and on evergreen tree farms and for evergreen forest re-growth. It has also been used to keep weeds from growing on both highway and railroad rights-of-way. Some of the trade names of atrazine are Aatrex®, Aatram®, Atratol®, and Gesaprim®. The scientific name for atrazine is 6-chloro-N-ethyl-N'-(1-methylethyl)-triazine-2,4-diamine. Atrazine is a Restricted Use Pesticide, which means that only certified herbicide users may purchase or use it. Certification for the use of atrazine is obtained through the appropriate state office

	<p>where the herbicide user is licensed. Atrazine is usually used in the spring and summer months. For it to be active, atrazine needs to dissolve in water and enter the plants through their roots. It then acts in the shoots and leaves of the weed to stop photosynthesis. Atrazine is taken up by all plants, but in plants not affected by atrazine, it is broken down before it can affect photosynthesis. The application of atrazine to crops as an herbicide accounts for almost all of the atrazine that enters the environment, but some may be released from manufacture, formulation, transport, and disposal.</p> <p>Any atrazine that is washed from the soil into streams and other bodies of water will stay there for a long time, because chemical breakdown is slow in rivers and lakes. It also will persist for a long time in groundwater. This is one reason why atrazine is found commonly in the water collected from drinking water wells in some agricultural regions.</p> <p>If atrazine enters the air, it can be broken down by reactions with other reactive chemicals in the air. However, sometimes atrazine is on particles such as dust. When this happens, breakdown is not expected. Atrazine is removed from air mainly by rainfall. When atrazine is on dust particles, the wind can blow it long distances from the nearest application area. For example, atrazine has been found in rainwater more than 180 miles (300 kilometers) from the nearest application area.</p> <p>Atrazine does not tend to accumulate in living organisms such as algae, bacteria, clams, or fish, and, therefore, does not tend to build up in the food chain.</p> <p>Atrazine Regulation and Monitoring</p> <p>Congress established the Safe Drinking Water Act in 1974, which set enforceable Maximum Contaminant Levels (MCLs) and non-enforceable Maximum Contaminant Level Goals (MCLGs) for certain, specified contaminants. In the case of atrazine in drinking water, EPA has set an MCL of 3 µg/L. Atrazine is designated as a Restricted Use Pesticide, which means that only certified pesticide applicators can use atrazine. The Occupational Safety and Health Administration (OSHA) has set a limit of 5 milligrams of atrazine per cubic meter of workplace air (5 mg/m³) for an 8-hour workday and 40-hour work week. EPA has determined maximum levels allowed in foods of 0.02-15 parts atrazine per million parts of food (0.02-15 ppm).</p>
<p>Use of Measure</p>	<p>These measures can assist by addressing the following surveillance functions:</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to atrazine at different concentrations. • Maximum concentrations provide information on the peak potential exposure to atrazine at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
<p>Limitations of the Measure</p>	<p>The current measures are derived for CWS only. Private wells are another important source of population exposure to atrazine in some agricultural regions.</p>

	<p>Transient non-community water systems, which are regulated by EPA, may also be an important source of atrazine exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be converted directly to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Ground water systems may have many wells with different atrazine concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the atrazine concentration of people served by wells with higher atrazine concentrations.</p> <p>Exposure may be higher or lower than estimated if data from multiple entry points for water with different atrazine levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. Adgate JL, Barr DB, Clayton CA, Eberly LE, Freeman NC, Lioy PJ, et al. Measurement of children's exposure to pesticides: analysis of urinary metabolite levels in a probability-based sample. <i>Environ Health Perspect</i> 2001;109(6):583-590. 2. Agency for Toxic Substances and Disease Registry (ATSDR). 2003. Toxicological Profile for Atrazine. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. 3. Arcury TA, Grzywacz JG, Barr DB, Tapia J, Chen H, Quandt SA. Pesticide urinary metabolite levels of children in eastern North Carolina farmworker households. <i>Environ Health Perspect</i> 2007;115(8):1254-1260. 4. Barr D.B., P. Panuwet, J.V. Nguyen, S. Udunka, L.L. Needham. Assessing exposure to atrazine and its metabolites using biomonitoring. <i>Environmental Health Perspectives</i> 2007, Vol. 115, No. 10, 1474-1478. 5. Bradway DE, Moseman RF. Determination of urinary residue levels of the N-dealkyl metabolites of triazine herbicides. <i>J Agric Food Chem</i> 1982;30(2):244-247. 6. Catenacci G, Barbieri F, Bersani M, Ferioli A, Cottica D, Maroni M. Biological monitoring of human exposure to atrazine. <i>Toxicol Lett</i> 1993;69(2):217-222. 7. Centers for Disease Control and Prevention (CDC). Third National Report on Human Exposure to Environmental Chemicals. Atlanta (GA). 2005. 3/11/09 8. Chevrier C., G. Limon. C. Monfort, f. Rouget, R. Garlantezec, C. Petit, G. Durand, S. Cordier. Urinary biomarkers of prenatal atrazine exposure and adverse birth outcomes in the PELAGIE Birth Cohort. <i>Environmental Health Perspectives</i> 2011, March 2 (doi:10.1289/ehp.1002775) 9. Curwin BD, Hein MJ, Sanderson WT, Barr DB, Heederik D, Reynolds SJ, et al. Urinary and hand wipe pesticide levels among farmers and nonfarmers in Iowa. <i>J Expo Anal Environ Epidemiol</i> 2005;15(6):500-508.

10. DeMarini DM, Zahm SH. Atrazine IARC Monographs 73, 1999.
11. Gammon DW, Aldous CN, Carr WC Jr, Sanborn JR, Pfeifer KF. A risk assessment of atrazine use in California: human health and ecological aspects. *Pest Manag Sci* 2005;61(4):331-355.
12. Hines CJ, Deddens JA, Striley CA, Biagini RE, Shoemaker DA, Brown KK, et al. Biological monitoring for selected herbicide biomarkers in the urine of exposed custom applicators: application of mixed-effect models. *Ann Occup Hyg* 2003;47(6):503-517.
13. Lucas AD, Jones AD, Goodrow MH, Saiz SG, Blewett C, Seiber JN, et al. Determination of atrazine metabolites in human urine: development of a biomarker of exposure. *Chem Res Toxicol* 1993;6(1):107-116.
14. MacIntosh DL, Needham LL, Hammerstrom KA, Ryan PB. A longitudinal investigation of selected pesticide metabolites in urine. *J Expo Anal Environ Epidemiol* 1999;9(5):494-501.
15. Munger R., P. Isacson, S. Hu, T., Burns, J. Hanson, C. F. Lynch et al., Intrauterine growth retardation in Iowa communities with herbicide-contaminated drinking water supplies. *Environmental Health Perspectives*, 1997; Vol., 105, 308-314.
16. Ochoa-Acuna H., J. Frankenberger J., L. Hahn, C. Carbajo. Drinking-water herbicide exposure in Indiana and prevalence of small-for-gestational-age and preterm delivery. *Environmental Health Perspectives* 2009; Vol. 117, 10, 1619-1624.
17. Panuwet R, J. V. Nguyen, P. Kuklenyik, S. O. Udunka, L.L. Needham, D. B. Barr. Quantification of atrazine and its metabolites in urine by on-line solid-phase extraction-high-performance liquid chromatography-tandem mass spectrometry. *Anal Bioanal Chem* 2008; 391: 1931-1939.
18. Rocheleau C. M., P. A. Romitti, L. K. Dennis. Pesticides and hypospadias: a meta-analysis. *J Pediatr Urol* 2009; Vol. 5, 17-24.
19. Perry M, Christiani D, Dagenhart D, Tortorelli J, Singzoni B. Urinary biomarkers of atrazine exposure among farm pesticide applicators. *Ann Epidemiol* 2000;10(7):479.
20. Sathiakumar N, Delzell E. A review of epidemiologic studies of triazine herbicides and cancer. *Crit Rev Toxicol* 1997;27(6):599-612.
21. Timchalk C, Dryzga MD, Langvardt PW, Kastl PE, Osborne DW. Determination of the effect of tridiphane on the pharmacokinetics of [14C]-atrazine following oral administration to male Fischer 344 rats. *Toxicology* 1990;61(1):27-40.
22. U.S. Environmental Protection Agency (U.S. EPA). Interim Reregistration Eligibility Decision For Atrazine. Case No. 0062. 2003. Available at URL: http://www.epa.gov/oppsrrd1/REDs/atrazine_ired.pdf . 3/11/09
23. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.
24. Villanueva C. M., G. Durand, M. B. Coutte, C. Chevrier, S. Cordier. Atrazine in municipal drinking water and risk of low birth weight, preterm delivery, and small-for-gestational-age status. *Occupational Environ Med* 2005; Vol. 62, 6, 400-405.
25. Waller S. A., K. Paul, S. E. Peterson, J. E. Hitti. Agricultural-related chemical exposures, season of conception, and risk of gastroschisis in Washington State. *Am J Obstet Gynecol* 2010, Vol. 202, 241.

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: DI(2-ETHYLHEXYL)PHTHALATE (DEHP)
 ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of Community Water Systems (CWS) by maximum DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP). 2. Yearly distribution of number of CWS by mean DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP). 3. Mean concentration of DEHP at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 4. Yearly distribution of number of people served by CWS by maximum DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP). 5. Yearly distribution of number of people served by CWS by mean DEHP concentration (cut-points: 0-2, >2-4, >4-6, >6-10, >10 µg/L DEHP).
Derivation of Measures	DEHP measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	DEHP, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Di (2-ethylhexyl)phthalate and Public Health</p> <p>DEHP is the most commonly used of a group of related chemicals called phthalates or phthalic acid esters. Some people who drink water containing</p>

DEHP well in excess of the maximum contaminant level (MCL) for many years may have problems with their livers or could experience reproductive difficulties and may have an increased risk of getting cancer. (U.S.EPA, 2010)

In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, DEHP was detected in 3,098 systems, which collectively serve more than 45 million people (EPA, 2009). Concentrations of DEHP were greater than the MCL in 460 systems serving 11.5 million people. DEHP was the highest occurring regulated synthetic organic chemical found based on the percent of detections found from the 6 Year Review data. This contamination could be due, in part, to sample contamination from older generation laboratory and field sampling equipment made of plastics that contained and released phthalates (EPA, 2009).

Most of what we know about the health effects of DEHP comes from studies of rats and mice given high amounts of DEHP. Brief oral exposure to very high levels of DEHP damaged sperm in mice. Although the effect reversed when exposure ceased, sexual maturity was delayed in the animals. High amounts of DEHP damaged the liver of rats and mice. Whether or not DEHP contributes to human kidney damage is unclear.

The Department of Health and Human Services has determined that DEHP may reasonably be anticipated to be a human carcinogen. The EPA has determined that DEHP is a probable human carcinogen. These determinations were based entirely on liver cancer in rats and mice. The International Agency for Research on Cancer has stated that DEHP cannot be classified as to its carcinogenicity to humans.

People are exposed through ingestion, inhalation, and, to a lesser extent, dermal contact with products that contain phthalates. For the general population, dietary sources have been considered as the major exposure route, followed by inhaling indoor air. Infants may have relatively greater exposures from ingesting indoor dust containing some phthalates (Clark et al., 2003). Human milk can be a source of phthalate exposure for nursing infants (Calafat et al., 2004; Mortensen et al., 2005). The intravenous or parenteral exposure route can be important in patients undergoing medical procedures involving devices or materials containing phthalates. In settings where workers may be exposed to higher air phthalate concentrations than the general population, urinary metabolite and air phthalate concentrations are roughly correlated (Liss et al., 1985; Nielsen et al., 1985; Pan et al., 2006). Phthalates are metabolized and excreted quickly and do not accumulate in the body (Anderson et al., 2001).

Biomonitoring Information

Four metabolites of DEHP were measured for the Fourth National Report on Human Exposure to Environmental Chemicals: mono-(2-ethyl-5-hexyl) phthalate (MEHP), mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP), mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP) and mono-(2-ethyl-5-carboxypentyl) phthalate (MECPP). MEHP is primarily formed by the hydrolysis of DEHP in the gastrointestinal tract and then absorbed. By contrast, DEHP present in medical devices and parenteral delivery systems results in the diester parent compound, rather than the monoester metabolite, being directly introduced into the blood. After parenteral administration hydrolysis of DEHP most likely also occurs in the blood, and subsequent metabolism is similar to that following ingestion (Koch et al., 2005a, 2005b, 2005c). MEOHP, MEHHP, and MECPP are produced by the oxidative metabolism of MEHP and are present at roughly three- to five-fold higher concentrations than MEHP in urine (Barr et al., 2003; Fromme et al., 2007; Koch et al., 2003). MEHP is the putative toxic metabolite of DEHP. Liver toxicity, decreased testicular weight, and testicular atrophy have been observed in rodents fed high doses over a short term or with chronic dosing (McKee et al., 2004; NTP-CERHR, 2000c, 2006). In contrast, marmoset monkeys fed high dose DEHP for longer than a year did not demonstrate testicular or liver toxicity (NTP-CERHR, 2006). Very high doses of DEHP have suppressed estradiol production in female rats (Lovecamp-Swan and Davis, 2003). The U.S. Food and Drug Administration determined that in adults, the amounts of DEHP or MEHP received from intravenous delivery systems or blood transfusions (DEHP is hydrolyzed to MEHP in stored blood) would result in short-term elevations similar to background levels (FDA, 2001). However, critically ill neonates and infants receiving selected or multiple intensive procedures, such as exchange transfusions, extracorporeal membrane oxygenation, and parenteral nutrition, could receive higher exposures than the general population (Calafat et al., 2004; FDA, 2001; Loff et al., 2000; Weuve et al., 2006).

The levels of MEHP reported in NHANES 1999-2000, 2001-2002, and 2003-2004 appear roughly comparable to those reported previously in several small U.S. studies involving adults (Blount et al., 2000), pregnant women in New York City (Adibi et al., 2003), and low income African-American women in Washington, DC (Hoppin et al., 2002). In another sample of men attending an infertility clinic, the median and 95th percentile values of urinary MEHP were similar, but MEHHP and MEOHP were about three to five times higher than comparable values found in males in two NHANES survey periods (1999-2000, 2001-2002) (CDC, 2005; Hauser et al., 2007). In separate analyses of NHANES 1999-2000 and NHANES 2001-2002, the adjusted geometric mean levels of urinary MEHP were significantly higher in children compared with adolescents and adults, and in females compared with males (CDC, 2005; Silva et al., 2004).

Studies of hospitalized neonates have reported urinary geometric mean levels of MEHP, MEOHP, and MEHHP that were two to five times higher, or more (depending on the intensity of DEHP-product exposure), than the geometric means of children in the NHANES subsamples for all three survey periods (Calafat et al., 2004; Weuve et al., 2006). Small studies of plasma and platelet donors have reported very high levels of MEHP, MEOHP, MEHHP and MECPP in urine collected shortly after these procedures (Koch et al., 2005b, 2005c). Finding a measurable amount of one or more DEHP metabolites in urine does not mean that the levels of the metabolites or the parent compound cause an adverse health effect. Biomonitoring studies on levels of urinary DEHP metabolites provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of DEHP than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

Sources of DEHP

Phthalates are industrial chemicals, often called *plasticizers*, that are added to plastics make them more flexible and resilient. Phthalates are also used in other applications as solubilizing and stabilizing agents. Numerous products contain phthalates: adhesives; automotive plastics; detergents; lubricating oils; some medical devices and pharmaceuticals; plastic raincoats; solvents; vinyl tiles and flooring; and personal-care products, such as soap, shampoo, deodorants, lotions, fragrances, hair spray, and nail polish. Phthalates are often used in polyvinyl chloride-type plastics, such as plastic bags, garden hoses, inflatable recreational toys, blood product storage bags, intravenous medical tubing, and toys (ATSDR, 2001, 2002). Because they are not chemically bound to the plastics to which they are added, phthalates can be released into the environment during use or disposal of the product. Various phthalate esters have been measured in specific foods, indoor and ambient air, indoor dust, water sources, and sediments (Clark et al., 2003).

DEHP is primarily used to produce flexibility in plastics, mainly polyvinyl chloride, which is used for many consumer products, toys, packaging film, and blood product storage and intravenous delivery systems. Concentrations in plastic materials may reach 40% by weight. DEHP has been removed from or replaced in most toys and food packaging in the United States. Following ingestion, DEHP is metabolized to more than 30 metabolites which are rapidly eliminated in urine, and in humans, as glucuronide conjugates (Albro et al., 1982; Albro and Lavenhar, 1989; ATSDR, 2002; Peck and Albro, 1982). The major source of di(2-ethylhexyl) phthalate in drinking water is discharge from rubber and chemical factories (U.S. EPA, 2010).

	<p>DEHP Regulation and Monitoring</p> <p>The EPA limits the amount of DEHP that may be present in drinking water to 6 parts of DEHP per billion parts of water (6 ppb), or 6 ug/L.</p> <p>The Occupational Safety and Health Administration (OSHA) sets a maximum average of 5 milligrams of DEHP per cubic meter of air (5 mg/m³) in the workplace during an 8-hour shift. The short-term (15-minute) exposure limit is 10 mg/m³.</p>
Use of Measure	<p>These measures can assist by addressing the following surveillance functions:</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to DEHP at different concentrations. • Maximum concentrations provide information on the peak potential exposure to DEHP at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells may be another source of population exposure to DEHP. Transient non-community water systems, which are regulated by EPA, may also be an important source of DEHP exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Ground water systems may have many wells with different DEHP concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the DEHP concentration of people served by wells with higher DEHP concentrations.</p> <p>Exposure may be higher or lower than estimated if data from multiple entry points for water with different DEHP levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use

References

1. Adibi JJ, Perera FP, Jedrychowski W, Camann DE, Barr D, Jacek R, et al. Prenatal exposures to phthalates among women in New York City and Krakow, Poland. *Environ Health Perspect* 2003;111(14):1719-1722.
2. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for di-n-butyl phthalate update [online]. 2001. Available at URL: <http://www.atsdr.cdc.gov/toxprofiles/tp135.html>. 4/20/09
3. Agency for Toxic Substances and Disease Registry (ATSDR). 2002. Toxicological Profile for Di(2-ethylhexyl) phthalate. Update. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.
4. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for di(2-ethylhexyl)phthalate update [online]. 2002. Available at URL: <http://www.atsdr.cdc.gov/toxprofiles/tp9.html>. 4/20/09.
5. Albro PW, Corbett JT, Schroeder JL, Jordan S, Matthews HB. Pharmacokinetics, interactions with macromolecules and species differences in metabolism of DEHP. *Environ Health Perspect* 1982;45:19-25.
6. Albro PW and Lavenhar SR. Metabolism of di(2-ethylhexyl) phthalate. *Drug Metab Rev* 1989;21:13-34.
7. Anderson WA, Castle L, Scotter MJ, Massey RC, Springall C. A biomarker approach to measuring human dietary exposure to certain phthalate diesters. *Food Addit Contam* 2001;18(12):1068- 1074.
8. Barr DB, Silva MJ, Kato K, Reidy JA, Malek NA, Hurtz D, et al. Assessing human exposure to phthalates using monoesters and their oxidized metabolites as biomarkers. *Environ Health Perspect* 2003;111(9):1148-1151.
9. Blount BC, Silva MJ, Caudill SP, Needham LL, Pirkle JL, Sampson EJ, et al. Levels of seven urinary phthalate metabolites in a human reference population. *Environ Health Perspect* 2000;108(10):979-982.
10. Calafat AM, Slakman AR, Silva MJ, Herbert AR, Needham LL. Automated solid phase extraction and quantitative analysis of human milk for 13 phthalate metabolites. *J Chromatogr B* 2004;805:49-56.
11. Calafat AM, Needham LL, Silva MJ, Lambert G. Exposure to di- (2-ethylhexyl) phthalate among premature neonates in a neonatal intensive care unit. *Pediatrics* 2004;113:e429-e434.
12. Centers for Disease Control and Prevention (CDC). Third National Report on Human Exposure to Environmental Chemicals. Atlanta (GA). 2005.
13. Clark K, Cousins IT, Mackay D. Assessment of critical exposure pathways. In Staples CA (ed), *The Handbook of Environmental Chemistry, Vol.3, Part Q: Phthalate Esters*. 2003;New York, Springer, pp. 227-262.
14. Food and Drug Administration (FDA). Safety assessment of di(2-ethylhexyl)phthalate (DEHP) released from PVC medical devices. 2001 [online]. Available at URL: <http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/psn/printer.cfm?id=77> 5/9/2013
15. Fromme H, Bolte G, Koch HM, Angerer J, Boehmer S, Drexler H, et al. Occurrence and daily variation of phthalate metabolites in the urine of an adult population. *Int J Hyg Environ Health* 2007;210:21-33.
16. Hauser R, Meeker JD, Singh NP, Silva MJ, Ryan L, Duty S, et al. DNA damage in human sperm is related to urinary levels of phthalate monoester and oxidative metabolites. *Hum Reprod* 2007; 22(3):688-695.
17. Hoppin JA, Brock JW, Davis BJ, Baird DD. Reproducibility of urinary phthalate

- metabolites in first morning urine samples. *Environ Health Perspect* 2002; 110(5):515-518.
18. Koch HM, Bolt HM, Preuss R, Angerer J. New metabolites of di(2-ethylhexyl)phthalate (DEHP) in human urine and serum after single oral doses of deuterium-labeled DEHP. *Arch Toxicol* 2005a; 79:367-376.
 19. Koch HM, Bolt HM, Preuss R, Eckstein R, Weisback V, Angerer J. Intravenous exposure to di(2-ethylhexyl)phthalate (DEHP) : metabolites of DEHP in urine after a voluntary platelet donation. *Arch Toxicol* 2005b; 79:689-693.
 20. Koch HM, Angerer J, Drexler H, Eckstein R, Weisbach V. Di(2-ethylhexyl)phthalate (DEHP) exposure of voluntary plasma and platelet donors. *Int J Hyg Environ Health* 2005c;208:489-498. Koch HM, Drexler H, Angerer J. Internal exposure of nursery-school children and their parents and teachers to di(2-ethylhexyl) phthalate (DEHP). *Int J Hyg Environ Health*. 2004; 207:15-22.
 21. Koch HM, Rossbach B, Drexler H, Angerer J. Internal exposure of the general population to DEHP and other phthalates - determination of secondary and primary phthalate monoester metabolites in urine. *Environ Res* 2003; 93:177-185.
 22. Liss GM, Albro PW, Hartle RW, Stringer WT. Urine phthalate determinations as an index of occupational exposure to phthalic anhydride and di (2-ethylhexyl) phthalate. *Scand J Work Environ Health* 1985;11(5):381-387.
 23. Loff S, Kabs F, Witt K, Sartoris J, Mandl B, Niessen KH, et al. Polyvinylchloride infusion lines expose infants to large amounts of toxic plasticizers. *J Pediatr Surg* 2000; 35(12):1775-1781.
 24. Lovekamp-Swan T, Davis BJ. Mechanisms of phthalate ester toxicity in the female reproductive system. *Environ Health Perspect* 2003; 111(2):139-145.
 25. McKee RH, Butala JH, David RM, Gans G. NTP center for the evaluation of risks to human reproduction reports on phthalates: addressing the data gaps [review]. *Reprod Toxicol* 2004; 18(1):1- 22.
 26. Mortensen GK, Main KM, Andersson A-M, Leffers H, Skakkebaek NE. Determination of phthalate monoesters in human milk, consumer milk, and infant formula by tandem mass spectrometry (LC-MS-MS). *Anal Bioanal Chem* 2005;382:1084- 1092.
 27. Nielsen J, Akesson B, Skerfving S. Phthalate ester exposure—air levels and health of workers processing polyvinylchloride. *Am Ind Hyg Assoc J* 1985;46(11):643-647.
 28. NTP-CERHR. National Toxicology Program-Center for the Evaluation of Risks to Human Reproduction. Monograph on the Potential Human Reproductive and Developmental Effects of Di (2-ethylhexyl) Phthalate (DEHP). Research Triangle Park (NC). 2000c [online]. Available at URL: <http://cerhr.niehs.nih.gov/chemicals/dehp/dehp-eval.html>. 6/2/09
 29. NTP-CERHR. National Toxicology Program-Center for the Evaluation of Risks to Human Reproduction. Draft Update Monograph on the Potential Human Reproductive and Developmental Effects of Di (2-ethylhexyl) Phthalate (DEHP). Research Triangle Park (NC). 2006 [online]. Available at URL: <http://cerhr.niehs.nih.gov/chemicals/dehp/dehp-eval.html>. 6/2/09
 30. Pan G, Hanaoka T, Yoshimura M, Zhang S, Wang P, Tsukino H, et al. Decreased serum free testosterone in workers exposed to high levels of di-n-butyl phthalate (DBP) and di-2-ethylhexylphthalate (DEHP): a cross-sectional study in China. *Environ Health Perspect* 2006; 114(11):1643-1648.
 31. Peck CC, Albro PW. Toxic potential of the plasticizer di (2-ethylhexyl) phthalate in

	<p>the context of its disposition and metabolism in primates and man. Environ Health Perspect 1982; 45:11-17.</p> <p>32. Silva MJ, Barr DB, Reidy JA, Malek NA, Hodge CC, Caudill SP, et al. Urinary levels of seven phthalate metabolites in the U.S. population from the National Health and Nutrition Examination Survey (NHANES) 1999-2000 [published erratum appears in Environ Health Perspect 2004; 112(5):A270]. Environ Health Perspect 2004; 112(3):331-338.</p> <p>33. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.</p> <p>34. U.S. EPA, Basic Information about Di (2- ethylhexyl) phthalate in Drinking Water. 2010. http://water.epa.gov/drink/contaminants/basicinformation/di_2-ethylhexyl_phthalate.cfm</p> <p>35. Weuve J, Sanchez GN, Calafat AM, Schettler T, Green RA, Hu H, et al. Exposure to phthalates in neonatal intensive care unit infants: urinary concentrations of monoesters and oxidative metabolites. Environ Health Perspect 2006; 114(9):1424-1431.</p>
--	--

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: TETRACHLOROETHENE (TETRACHLOROETHYLENE) (PCE)
ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of Community Water Systems (CWS) by maximum PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE). 2. Yearly distribution of number of CWS by mean PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE). 3. Mean concentration of PCE at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 4. Yearly distribution of number of people served by CWS by maximum PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE). 5. Yearly distribution of number of people served by CWS by mean PCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L PCE).
Derivation of Measures	PCE measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	PCE, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be the approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Tetrachloroethene (PCE) and Public Health</p> <p>Tetrachloroethene (PCE) is a volatile halogenated short-chain hydrocarbon. Tetrachloroethene is used in dry cleaning, metal cleaning, the synthesis of other chemicals, and household products such as water repellants, silicone lubricants, and spot removers. PCE is produced and used in high volumes in the U.S. and has been detected in urban and ambient air and occasionally in soils and drinking water most likely contaminated by industrial discharge (Moran et al., 2007; Rowe</p>

et al., 2007). Because of its volatility, this solvent does not persist in the soil or water following the discontinuation of contamination.

Inhalation is the most common exposure route for the general population including indoor sources from paints, adhesives, and cleaning solutions. Volatilization from contaminated water (e.g., shower water) as well as the use of household products containing this solvent can result in higher indoor than outdoor air concentrations (ATSDR, 1997; Martin et al., 2005). Nearby dry cleaning establishments, industries producing PCE, and contaminated waste disposal sites can also contribute to human exposure (Armstrong and Green, 2004; ATSDR, 1997 and 2000; Schreiber et al., 1993; Wallace et al., 1991). Drinking water may contribute to exposure when underground drinking water supplies have been contaminated. Workers in industries such as dry cleaning, aircraft maintenance, electronics manufacturing, and chemical production may be exposed by inhalation or by dermal contact with PCE. The EPA has established drinking water standards and other environmental standards for PCE, and the FDA regulates PCE and trichloroethene as indirect food additives. Workplace standards have been established by OSHA, and ACGIH has recommended occupational guidelines and biological exposure indices for monitoring workers. Human health effects from PCE at low environmental doses or at biomonitored levels from low environmental exposures are unknown. PCE is well absorbed by ingestion and inhalation, and animal studies have demonstrated that liquid forms can be dermally absorbed. Following absorption, part of the solvent dose is excreted into expired air; for PCE, about 97-99% of the dose is eliminated unmetabolized into expired air, though it has an elimination half-life of several days (ATSDR 1997; Monster, 1986). The retained solvent can undergo hepatic metabolism. PCE is metabolized to trichloroacetic acid and trichloroethanol, which are eliminated in the urine. Accidental or intentional high dose acute exposure by ingestion or inhalation can result in loss of motor coordination, somnolence, and unconsciousness. Inhaling high doses of PCE may also produce cardiac arrhythmias attributed to enhanced sensitivity to catecholamines. High dose acute exposure to PCE has resulted in reversible kidney impairment, and prolonged, low level PCE exposure has been associated with altered renal enzyme excretion and liver enlargement (ATSDR, 1997). Chronic occupational exposure to PCE may be associated with mild degrees of neurological impairments, including reaction times, verbal skills, cognitive ability, and motor function (Armstrong and Green, 2004). Various epidemiologic studies of chronic PCE exposure in dry cleaning workers found increased incidences of esophageal and cervical cancers and non-Hodgkins lymphoma, but confounding exposures (e.g., other solvents and trichloroethene) were likely (IPCS, 2006). In animal studies, PCE-induced kidney and liver tumors and caused leukemia (IARC, 1995). IARC classifies PCE as a probable human carcinogen, and NTP classifies it as reasonably anticipated to be a human carcinogen (IARC, 1995; NTP, 2004). Additional information about these solvents is available from ATSDR at: <http://www.atsdr.cdc.gov/toxpro2.html>.

In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, PCE was detected in 1,262 systems serving close to 32 million people (EPA, 2009). Concentrations of PCE were greater than the MCL in 241 systems serving close to 15 million people. PCE was the fifth highest occurring regulated volatile organic chemical found based on the percent of detections found from the 6 Year Review data (EPA, 2009).

Biomonitoring Information

Levels of halogenated solvents in blood reflect recent exposure. In the NHANES 2003-2004 subsample, the level of blood PCE for adults at the 75th percentile of the U.S. population appear similar to the levels at the 75th percentile reported for non-smoking adults in a subsample of NHANES 1999-2000 participants (CDC, 2009; Lin et al., 2008) and were similar or slightly less than levels reported in a nonrepresentative subsample of the earlier NHANES III (1988-1994) (Ashley et al., 1994; Churchill et al., 2001). A recent study of low income, urban children in the Midwest reported slightly lower median PCE levels (Sexton et al., 2005; Sexton et al., 2006) than the NHANES III levels (Ashley et al., 1994; Churchill et al., 2001).

Comparatively higher blood levels of PCE and trichloroethene have been noted for urban and industrial residential settings than for rural settings (Barkley et al., 1980; Begerow et al., 1996; Brugnone et al., 1994). Residing near dry-cleaning facilities or storing recently dry-cleaned clothes at home can contribute to increased blood PCE levels (Begerow et al., 1996; Popp et al., 1992). In contrast, PCE blood levels in occupationally exposed workers have been reported to be many thousand times higher than the general population (Begerow et al., 1996; Furuki et al., 2000; Monster et al., 1983). The occupational biological exposure index associated with an 8-hour exposure of 25 ppm is 500 µg/L PCE in blood (ACGIH, 2007). Non-occupational exposures are usually well below this level. Finding a measurable amount of any of these solvents in blood does not mean that the level of the solvent causes an adverse health effect. Biomonitoring studies of blood halogenated solvents can provide physicians and public health officials with reference values so that they can determine whether or not people have been exposed to higher levels of halogenated solvents than levels found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

Sources of PCE

The major source of PCE in drinking water is discharge from factories and dry cleaners. A federal law called the Emergency Planning and Community Right to Know Act requires facilities in certain industries, which manufacture, process, or use significant amounts of toxic chemicals, to report annually on their releases of these chemicals. For more information on the uses and releases of chemicals in your state, contact the Community Right-to-Know Hotline: (800) 424-9346 (EPA,

	<p>2010).</p> <p>PCE Regulation and Monitoring The EPA limits the amount of PCE that may be present in drinking water to 5 parts of PCE per billion parts of water (5 ppb), or 5 ug/L.</p>
Use of Measure	<p>These measures can assist by addressing the following surveillance functions:</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to PCE at different concentrations. • Maximum concentrations provide information on the peak potential exposure to PCE at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells may be another source of population exposure to PCE. Transient non-community water systems, which are regulated by EPA, also may be an important source of PCE exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Ground water systems may have multiple wells with different PCE concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the PCE concentration of people served by wells with higher PCE concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different PCE levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. ACGIH. TLVs and BEIs Based on the documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. 2007. Signature Publications. Cincinnati OH. p.104. 2. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for tetrachloroethylene update. 1997 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp18.html. 4/22/09 3. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for Tetrachloroethylene update. 2000 [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp14.html. 4/22/09 4. Armstrong SR, Green LC. Chlorinated hydrocarbon solvents. Clin Occup Environ Med

- 2004;4(3):481-496.
5. Ashley DL, Bonin MA, Cardinali FL, McCraw JM, Wooten JV. Blood concentrations of volatile organic compounds in a nonoccupationally exposed US population and in groups with suspected exposure. *Clin Chem* 1994;40(7 Pt 2):1401-1404.
 6. Barkley J, Bunch J, Bursey JT, Castillo N, Cooper SD, Davis JM, et al. Gas chromatography mass spectrometry computer analysis of volatile halogenated hydrocarbons in man and his environment—a multimedia environmental study. *Biomed Mass Spectrom* 1980;7(4):139-147.
 7. Begerow J, Jermann E, Keles T, Freier I, Ranft U, Dunemann L. Internal and external tetrachloroethene exposure of persons living in differently polluted areas of Northrhine-Westphalia (Germany). *Zentralbl Hyg Umweltmed.* 1996;198(5):394-406.
 8. Brugnone F, Perbellini L, Guiliari C, Cerpelloni M, Soave M. Blood and urine concentrations of chemical pollutants in the general population. *Med Lav* 1994; 8(5):370-389.
 9. Centers for Disease Control and Prevention (CDC). Fourth National Report on Human Exposure to Environmental Chemicals, 2009. Available at: <http://www.cdc.gov/exposurereport/pdf/FourthReport.pdf>
 10. Churchill JA, Ashley DL, Kaye WE. Recent chemical exposures and blood volatile organic compound levels in a large population- based sample. *Arch Environ Health* 2001; 56(2):157-166.
 11. Furuki K, Ukai H, Okamoto S, Takada S, Kawai T, Miyama Y, Mitsuyoshi K, et al. Monitoring of occupational exposure to tetrachloroethene by analysis for unmetabolized tetrachloroethene in blood and urine in comparison with urinalysis for trichloroacetic acid. *Int Arch Occup Environ Health.* 2000; 73(4):221-227.
 12. International Agency for Research in Cancer (IARC). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals Vol. 63, 1995. Available at: <http://monographs.iarc.fr/ENG/Monographs/vol63/mono63.pdf>
 13. International Programme on Chemical Safety (IPCS). Concise International Chemical Assessment Document 68-Tetrachloroethene. 2006 [online]. Available at URL: <http://www.inchem.org/documents/cicads/cicads/cicad68.htm>. 4/22/09
 14. Lin YS, Egeghy PP, Rappaport SM. Relationships between levels of volatile organic compounds in air and blood from the general population. *J Exp Sci Environ Epidemiol* 2008; 18:421-429.
 15. Martin SA, Simmons MB, Ortiz-Serrano M, Kendrick C, Gallo A, Campbell J, et al. Environmental exposure of a community to airborne trichloroethylene. *Arch Environ Occup Health* 2005; 60(6):341-316.
 16. Monster AC. Biological monitoring of chlorinated hydrocarbon solvents. *J Occup Med* 1986; 28:583-588.
 17. Monster AC, Regouin-Peeters W, Van Schijndel A, van der Tuin J. Biological monitoring of occupational exposure to tetrachloroethene. *Scand J Work Environ Health* 1983; 9:273-281.
 18. Moran MJ, Zogorski JS, Squillace PJ. Chlorinated solvents in groundwater of the United States. *Environ Sci Technol* 2007; 41:74-81.
 19. National Toxicology Program (NTP). Report on Carcinogens, 12th ed. 2010. [online]. Available at URL: <http://ntp.niehs.nih.gov/ntp/roc/twelfth/ListedSubstancesReasonablyAnticipated.pdf> 5/9/2013.

- | | |
|--|--|
| | <ol style="list-style-type: none">20. Popp W, Muller G, Baltes-Schmitz B, Wehner B, Vahrenholz C, Schmieding W, et al. concentrations of tetrachloroethene in blood and trichloroacetic acid in urine in workers and neighbours of dry-cleaning shops. <i>Int Arch Occup Environ Health</i> 1992; 63:393- 395.21. Rowe BL, Toccalino PL, Moran MJ, Zogorski JS, Price CV. Occurrence and potential human-health relevance of volatile organic compounds in drinking water from domestic wells in the United States. <i>Environ Health Perspect</i> 2007; 115(11):1539-1546.22. Schreiber JS, House S, Prohonic E, Smead G, Hudson C, Styk M, et al. An investigation of indoor air contamination in residences above dry cleaners. <i>Risk Anal</i> 1993; 13(3):335-344. Sexton K, Adgate JL, Church TR, Ashley DL, Needham LL, Ramachandran, et al. Children's exposure to volatile organic compounds as determined by longitudinal measurements in blood. <i>Environ Health Perspect</i> 2005; 113(3):342-349.23. Sexton K, Adgate JL, Fredrickson AL, Ryan AD, Needham LL, Ashley DL. Using biologic markers in blood to assess exposure to multiple environmental chemicals for inner-city children 3-6 years of age. <i>Environ Health Perspect</i> 2006; 114(3):453-459.24. U.S. Environmental Protection Agency (U.S. EPA). Basic Information about Tetrachloroethylene in Drinking Water, 2010. Available at:25. http://water.epa.gov/drink/contaminants/basicinformation/tetrachloroethylene.cfm26. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.27. Wallace L, Nelson W, Ziegenfus R, Pellizzari E, Michael L, Whitmore R, et al. The Los Angeles TEAM Study: Personal exposures, indoor-outdoor air concentrations, and breath concentrations of 25 volatile organic compounds. <i>J Exp Anal Environ Epidemiol</i> 1991; 1(2):157-192. |
|--|--|

CONTENT DOMAIN: COMMUNITY WATER

INDICATOR: NITRATE

ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Quarterly distribution of number of Community Water Systems (CWS) by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 2. Yearly distribution of number of CWS by maximum nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 3. Yearly distribution of number of CWS by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 4. Mean concentration of nitrate at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 5. Quarterly distribution of number of people served by CWS by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 6. Yearly distribution of number of people served by CWS by maximum nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate). 7. Yearly distribution of number of people served by CWS by mean nitrate concentration (cut-points: (0-3), (>3-5), (>5-10), (>10-20), (>20) mg/L nitrate).
Derivation of Measures	Nitrate measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	Concentration of nitrate, mg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year

<p>Rationale</p>	<p>Nitrates and Public Health</p> <p>Nitrate was first identified as a public health threat in drinking water in 1945 when high nitrate levels from private wells were shown to cause methemoglobinemia or “blue baby syndrome” in infants who received formula made from well water. When an individual is exposed to nitrate it can be converted to nitrite (NO_2^-) in the body and then oxidize the ferrous iron (Fe^{+2}) in deoxyhemoglobin in the blood to form methemoglobin containing ferric iron (Fe^{+3}). Methemoglobin cannot transfer oxygen to tissues; thus nitrate or nitrite can starve the body of oxygen and produce a clinical condition known as cyanosis, where the lips and extremities turn gray or blue. Infants younger than four months of age are more sensitive than adults, and can develop “blue baby” syndrome from intake of nitrate higher than 10 mg/L nitrate or 45 mg/L nitrate–nitrogen. Blue baby syndrome is fatal in about ten percent of the cases (ATSDR, 2007). Usually there are no outward signs of cyanosis at methemoglobin levels below 20 percent (Dabney et al, 1990).</p> <p>In addition, there is some evidence to suggest that exposure to nitrate in drinking water is also associated with adverse reproductive outcomes such as spontaneous abortions, intrauterine growth retardation, and various birth defects such as anencephaly, related to fetal exposures to nitrate. However, the evidence is inconsistent (Manassaram et al, 2006).</p> <p>Similarly, long term exposure to higher nitrate levels in drinking water has been suggested as a risk factor for cancer. Cancer at several sites (i.e. gastric, colorectal, bladder, urothelial, brain, esophagus, ovarian and non-Hodgkins lymphoma) have been shown to be associated with nitrate in drinking water in some studies (Sandor et al, 2001; Weyer et al, 2001; Gulis et al, 2002; De Roos et al, 2003; Volkmer et al, 2005; Ward et al, 2005b; Chiu et al, 2007;). Other studies have not found any association (Ward et al, 2003; Ward et al, 2005, 2005c; Ward et al, 2006; Zeegers et al, 2006). Significant regional differences in cancer risk may occur (Mueller et al, 2001). Occupational exposures are also of concern as nitrate fertilizer workers have shown increased risk for stomach cancer (Zandjani et al. 1994).</p> <p>Sources of Nitrate</p> <p>Nitrate is the most commonly found contaminant in groundwater aquifers worldwide (Ward, 2005 from: Spalding and Exner 1993). Nitrate (NO_3^-) originates in drinking water from nitrate-containing fertilizers, sewage and septic tanks, and decaying natural material such as animal waste. Nitrate is very soluble in water, can easily migrate, and does not evaporate (EPA Consumer Fact Sheet). Anthropogenic sources of nitrates are increasing resulting in increased nitrate levels in water resources. Surface water and shallow wells in both rural and urban areas can be affected. Consequently, private wells are especially vulnerable to excess levels of nitrates. Excess levels of nitrate and nitrite can occur in community water supplies. A U.S. Geological Survey (USGS) study found nitrate levels exceeded regulatory monitoring standards in 2% of a sample of 242 public drinking water wells between 1992 and 1999 (Squillace et al, 2002). Levels of nitrates in</p>
-------------------------	--

	<p>private wells are less well known; private wells are not regularly monitored and are often more vulnerable to higher levels of nitrates because they draw water from shallower groundwater aquifers. The USGS estimates approximately 22% of domestic wells in agricultural areas of the U.S. exceed the MCL (Ward, 2007).</p> <p>Nitrate Regulation and Monitoring</p> <p>Congress established the Safe Drinking Water Act in 1974, which set enforceable Maximum Contaminant Levels (MCLs) and non-enforceable Maximum Contaminant Level Goals (MCLGs) for certain specified contaminants. In the case of nitrate in drinking water, the MCLG of 10 mg/L (ppm) was established from human data from studies of methemoglobinemia in young children. (Johnson and Kross 1990; Walton, 1950). The MCL is also set at 10 ppm, and any exceedance of the MCL is potentially serious as there is no additional margin of safety between the MCLG and the MCL. 2002). The MCLG and MCL for nitrite are 1 mg/L. While evidence to suggest MCL exposures for chronic health endpoints remains inconclusive, there is some evidence to suggest that chronic exposure to nitrate levels below the MCL may be of concern (Ward, 2005).</p>
Use of Measure	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to nitrate at different concentrations. • Maximum concentrations provide information on the peak potential exposure to nitrate at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells are another important source of population exposure to nitrate. Transient non-community water systems, which are regulated by EPA, may also be an important source of nitrate exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Nitrate levels can vary substantially in groundwater; thus high levels may not be captured by even quarterly sampling. Estimates of the number of people potentially exposed may be unreliable as they are based on estimates made by the water system operator. Concentrations in drinking water cannot be directly converted to exposure because overall water consumption, and the proportion of water consumed that comes from the tap is quite variable (EPA 2004). In systems that have more than one Entry point to the Distribution system, the actual nitrate level at any given house is a mixture</p>

	<p>of the levels from all contributing sources. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the nitrate concentration of people served by wells with higher nitrate concentrations.</p> <p>Exposure may be higher or lower than estimated if data from multiple entry points for water with different nitrate levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. ATSDR Case Studies in Environmental Medicine: Nitrate/Nitrite Toxicity. http://www.atsdr.cdc.gov/HEC/CSEM/nitrate/index.html Downloaded 08/07/07 2. Bosch, H. M., A. B. Rosenfield, R. Huston, H. R. Shipman, and F. L. Woodward. 1950. Methemoglobinemia and Minnesota well supplies. <i>Am. Water Works Assoc J</i> 42:161-170. 3. Chiu HF, Tsai SS, Yang CY. 2007. Nitrate in drinking water and risk of death from bladder cancer: an ecological case-control study in Taiwan. <i>J Toxicol Environ Health A</i> 70(12):1000-1004. 4. Coss A, Cantor KP, Reif JS, Lynch CF, Ward MH. 2004. Pancreatic cancer and drinking water and dietary sources of nitrate and nitrite. <i>Am J Epidemiol</i> 159(7):693-701. 5. Dabney BJ, Zelarney PT, Hall AH. 1990. Evaluation and treatment of patients exposed to systemic asphyxiants. <i>Emerg Care Q</i> 6(3):65-80 6. De Roos AJ, Ward MH, Lynch CF, Cantor KP. 2003. Nitrate in public water supplies and the risk of colon and rectum cancers. <i>Epidemiology</i> 14(6):640-649. 7. Gulis G, Czompolyova M, Cerhan JR. 2002. An ecologic study of nitrate in municipal drinking water and cancer incidence in Trnava District, Slovakia. <i>Environ Res</i> 88(3):182-187. 8. Johnson CJ and Kross BC. 1990. Continuing importance of nitrate contamination of groundwater and wells in rural areas. <i>Am J Ind Med</i> 18(4):449-456. 9. Mueller BA, Newton K, Holly EA, Preston-Martin S. 2001. Residential water source and the risk of childhood brain tumors. <i>Environ Health Perspect</i> 109(6):551-556. 10. Ruckart PZ, Henderson AK, Black ML, Flanders WD. 2007. Are nitrate levels in groundwater stable over time? <i>J Expo Sci Environ Epidemiol</i> Apr 11; [Epub ahead of print] 11. Sandor J, Kiss I, Farkas O, Ember I. 2001. Association between gastric cancer mortality and nitrate content of drinking water: ecological study on small area inequalities. <i>Eur J Epidemiol</i> 17(5):443-447. 12. U.S. Environmental Protection Agency Office of Water: Candidate Contaminants List. http://www.epa.gov/safewater/ccl/index.html Downloaded 08/02/07 13. U.S. Environmental Protection Agency. Office of Water (4606) Occurrence Estimation Methodology and Occurrence Findings Report for the Six-Year Review of Existing National Primary Drinking Water Regulations. EPA-815-R-03-006 www.epa.gov June 2003. http://www.epa.gov/safewater/standard/review/pdfs/support_6yr_occurrencemethods_final.pdf Downloaded 08/02/07 14. U.S. Environmental Protection Agency (2007b): Technical Factsheet on: Nitrate/Nitrite. http://www.epa.gov/safewater/dwh/t-ioc/nitrates.html Downloaded 08/07/07 15. Volkmer BG, Ernst B, Simon J, Kuefer R, Bartsch G Jr, Bach D, Gschwend JE. 2005. Influence of nitrate levels in drinking water on urological malignancies: a community-based cohort study. <i>BJU Int</i> 95(7):972-976. 16. Walton, G. 1951. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. <i>Am J Public Health</i> 41:986-996.

- | | |
|--|---|
| | <ol style="list-style-type: none">17. Ward MH, Cantor KP, Riley D, Merkle S, Lynch CF. 2003. Nitrate in public water supplies and risk of bladder cancer. <i>Epidemiology</i> 14(2):183-190.18. Ward MH, Heineman EF, McComb RD, Weisenburger DD. 2005. Drinking water and dietary sources of nitrate and nitrite and risk of glioma. <i>J Occup Environ Med</i> 47(12):1260-1267.19. Ward MH, deKok TM, Levallois P, Brender J, Gulis G, Nolan BT, VanDerslice J. 2005b. Workgroup report: Drinking-water nitrate and health--recent findings and research needs. <i>Environ Health Perspect</i> 113(11):1607-1614.20. Ward MH, Heineman EF, McComb RD, Weisenburger DD. 2005c. Drinking water and dietary sources of nitrate and nitrite and risk of glioma. <i>J Occup Environ Med</i> 47(12):1260-1267.21. Ward MH, Cerhan JR, Colt JS, Hartge P. 2006. Risk of non-Hodgkin lymphoma and nitrate and nitrite from drinking water and diet. <i>Epidemiology</i> 17(4):375-382.22. Weyer PJ, Cerhan JR, Kross BC, Hallberg GR, Kantamneni J, Breuer G, Jones MP, Zheng W, Lynch CF. 2001. Municipal drinking water nitrate level and cancer risk in older women: the Iowa Women's Health Study. <i>Epidemiology</i> 12(3):327-338.23. Zandjani F, Hogsæt B, Andersen A, Langard S. 1994. Incidence of cancer among nitrate fertilizer workers. <i>Int Arch Occup Environ Health</i> 66:189-93.24. Zeegers MP, Selen RF, Kleinjans JC, Goldbohm RA, van den Brandt PA. 2006. Nitrate intake does not influence bladder cancer risk: the Netherlands cohort study. <i>Environ Health Perspect</i> 114(10):1527-1531. |
|--|---|

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: COMBINED RADIUM-226 AND -228
 ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of Community Water Systems (CWS) by maximum Radium concentration (cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium). 2. Yearly distribution of number of CWS by mean Radium concentration (cut-points: cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium). 3. Mean concentration of Radium at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 4. Yearly distribution of number of people served by CWS by maximum Radium concentration (cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium). 5. Yearly distribution of number of people served by CWS by mean Radium concentration (cut-points: 0-3, >3-5, >5-10, >10 pCi/L Radium).
Derivation of Measures	Combined Radium-226 and -228 measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	pCi/L combined Radium-226 & -228
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Radium-226 and -228 and Public Health</p> <p>Radium is a naturally occurring silvery-white radioactive metal that can exist in several forms called isotopes. Radium is produced constantly by the radioactive decay of uranium and thorium. Uranium and thorium are found in small</p>

amounts in most rocks and soil. Some of the radiation from radium is being released constantly into the environment. It is this radioactive decay that causes concern about the safety of radium and all other radioactive substances. Two of the main radium isotopes found in the environment are radium-226 and radium-228. The decay of radium-226 results in the formation of radon which exists as a gas and is mobile in environmental media. Radium has been used as a radiation source for treating cancer, in radiography of metals, and combined with other metals as a neutron source for research and radiation instrument calibration. Until the 1960s, radium was a component of the luminous paints used for watch and clock dials, instrument panels in airplanes, military instruments, and compasses (ATSDR, 2010).

Everyone is exposed to low levels of radium in the air, water, and food. Higher levels may be found in the air near industries that burn coal or other fuels or near sites that mine or mill uranium. It also may be found at higher levels in drinking water from groundwater wells. Miners, particularly miners of uranium and hard rock, are exposed to higher levels of radium. It may also be found at radioactive waste disposal sites (ATSDR, 1990).

It is not known whether long-term exposure to radium at the levels that are normally present in the environment (for example, 1 pCi of radium per gram of soil) is likely to result in harmful health effects. However, exposure to higher levels of radium over a long period of time may result in harmful effects including anemia, cataracts, fractured teeth, cancer (especially bone cancer), and death. Patients who were injected with radium in Germany, from 1946 to 1950, for the treatment of certain diseases including tuberculosis were significantly shorter as adults than people who were not treated. Some of these health effects may take years to develop and mostly are due to gamma radiation. Radium gives off gamma radiation, which can travel fairly long distances through air. Therefore, just being near radium at the high levels that may be found at some hazardous waste sites may be dangerous to your health.

Exposure to high levels of radium results in an increased incidence of bone, liver, and breast cancer. The EPA and the National Academy of Sciences, Committee on Biological Effects of Ionizing Radiation, has stated that radium is a known human carcinogen.

Biomonitoring Information

Urine tests can determine if you have been exposed to radium. Another test measures the amount of radon (a breakdown product of radium) in exhaled air. Both types of tests require special equipment and cannot be done in a doctor's office. These tests cannot tell how much radium you were exposed to, nor can they be used to predict whether you will develop harmful health effects (ATSDR,

	<p>1990). Levels of radium in the U.S. population are unknown.</p> <p>Sources of Radium Radium forms from the decay of uranium or thorium in the environment. Radium -226 is formed from the decay of uranium-238; Radium-228 is formed from the decay of thorium. Radium is abundant in low levels everywhere because it originates from uranium which is commonly found in all rocks, soil and water. (EPA, 2010)</p> <p>Radium Regulation and Monitoring The EPA has set a drinking water limit of 5 picocuries per liter (5 pCi/L) for radium-226 and radium-228 (combined) (EPA, 2009). A gross alpha particle activity measurement may be substituted for the required radium-226 measurement provided that the measured gross alpha particle activity does not exceed 5 pCi/L. The EPA lifetime exposure cancer risk estimate for radium at the MCL, is approximately 1-2 cases per 10,000 people.</p> <p>Monitoring frequency Once a CWS has satisfied initial monitoring requirements (4 quarterly samples at every entry point to the distribution system within the first quarter after initiating the source); the required frequency for Combined Radium-226 and -228 monitoring is once every three years if the average of the initial monitoring results for the contaminant is greater than one-half the MCL but at or below the MCL. States may allow CWS to reduce the frequency of monitoring from once every three years to once every six or nine years at each sampling point, if the average of the initial monitoring results for each contaminant is below the detection limit. If a system has a monitoring result that exceeds the MCL while on reduced monitoring, the system must collect and analyze quarterly samples at that sampling point until the system has results from four consecutive quarters that are below the MCL, unless the system enters into another schedule as part of a formal compliance agreement with the State (CFR, 2002).</p>
Use of Measure	<p>These measures can assist by addressing the following surveillance functions:</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to combined Radium-226 and -228 at different concentrations. • Maximum concentrations provide information on the peak potential exposure to combined Radium-226 and -228 at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells may be another source of population exposure to combined Radium-226 and -228. Transient non-community water systems, which are regulated by EPA, may also be an</p>

	important source of combined Radium-226 and -228 exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	<p>The required monitoring frequency for combined Radium-226 and -228 is infrequent and may be as intermittent as every nine years; therefore most states will have very little data on this contaminant.</p> <p>Ground water systems may have multiple wells with different combined Radium-226 and -228 concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the combined Radium-226 and -228 concentrations of people served by wells with higher combined Radium-226 and -228 concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different combined Radium-226 and -228 levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use; Uranium
References	<ol style="list-style-type: none"> 1. Agency for Toxic Substances and Disease Registry (ATSDR). Toxic Substances Portal. Radium. 2010. Available at: http://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=154 2. Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for Radium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service Available at: http://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=790&tid=154 3. Code of Federal Regulations (CFR), 2002. Title 40 Protection of the Environment Chapter I--Environmental Protection Agency Part 141--National Primary Drinking Water Regulations 141.26 Monitoring frequency and compliance requirements for radionuclides in community water systems. Available at: URL: http://www.access.gpo.gov/nara/cfr/waisidx_02/40cfr141_02.html 4. U.S. Environmental Protection Agency (U.S. EPA). Radiation Protection, Radium, 2010. Available at: http://www.epa.gov/radiation/radionuclides/radium.html 5. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.

CONTENT DOMAIN: COMMUNITY WATER INDICATOR: TRICHLOROETHENE (TRICHLOROETHYLENE) (TCE) ENVIRONMENTAL PUBLIC HEALTH TRACKING	
Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of CWS by maximum TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE). 2. Yearly distribution of number of CWS by mean TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE). 3. Mean concentration of TCE at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 4. Yearly distribution of number of people served by CWS by maximum TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE). 5. Yearly distribution of number of people served by CWS by mean TCE concentration (cut-points: 0-1, >1-2, >2-5, >5 µg/L TCE).
Derivation of Measures	TCE measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	TCE, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be the approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Trichloroethene (TCE) and Public Health</p> <p>Trichloroethene (TCE) is a volatile halogenated short-chain hydrocarbon. TCE is used primarily as an industrial degreaser, solvent, and in the synthesis of other chemicals. In the past, it was used in dry cleaning, food processing, household cleaners, and as a general anesthetic. TCE is produced and used in high volumes in the U.S. and has been detected in urban and ambient air and</p>

occasionally soils and drinking water most likely contaminated by industrial discharge (Moran et al., 2007; Rowe et al., 2007). Because of its volatility, this solvent does not persist in the soil or water following the discontinuation of contamination.

Drinking or breathing high levels of TCE may cause nervous system effects, liver and lung damage, abnormal heartbeat, coma, and possibly death (ATSDR, 2003). Inhalation is the most common exposure route for the general population including indoor sources from paints, adhesives, and cleaning solutions. Volatilization from contaminated water (e.g., shower water) as well as the use of household products containing this solvent can result in higher indoor than outdoor air concentrations (ATSDR, 1997b; Martin et al., 2005). Nearby dry cleaning establishments, industries producing this solvent, and contaminated waste disposal sites can also contribute to human exposure (Armstrong and Green, 2004; ATSDR, 1997a, 1997b, and 2000; Schreiber et al., 1993; Wallace et al., 1991). Drinking water may contribute to exposure when underground drinking water supplies have been contaminated. Workers in industries such as dry cleaning, aircraft maintenance, electronics manufacturing, and chemical production may be exposed by inhalation or dermal contact. The EPA has established drinking water standards and other environmental standards for TCE, and the FDA regulates TCE as an indirect food additive. OSHA has established workplace standards, and ACGIH has recommended occupational guidelines and biological exposure indices for monitoring workers (ACGIH, 2007). Human health effects from TCE at low environmental doses or at biomonitored levels from low environmental exposures are unknown. TCE is well absorbed by ingestion and inhalation, and animal studies have demonstrated that liquid forms can be dermally absorbed. Following absorption, part of the solvent dose is excreted into expired air (ATSDR1997a; Monster, 1986). The retained solvent can undergo hepatic metabolism. TCE is metabolized to trichloroacetic acid and trichloroethanol, which are eliminated in the urine. Accidental or intentional high dose acute exposure by ingestion or inhalation can result in loss of motor coordination, somnolence, and unconsciousness. Inhaling high doses of TCE may also produce cardiac arrhythmias attributed to enhanced sensitivity to catecholamines. Prolonged, low level exposure to TCE has been associated with altered renal enzyme excretion and liver enlargement (ATSDR, 1997a, b). Chronic occupational exposure to TCE may be associated with mild degrees of neurological impairments, including reaction times, verbal skills, cognitive ability and motor function (Armstrong and Green, 2004). In animal studies, TCE induced kidney and liver tumors; and caused lung and testicular tumors (IARC, 1995). A recent EPA toxicological review (EPA/635/R-09/011F) characterized TCE as carcinogenic in humans by all routes of exposure (EPA, 2011). For cancer, the inhalation unit risk is 2×10^{-2} per ppm [4×10^{-6} per $\mu\text{g}/\text{m}^3$], based on human kidney cancer risks (Charbotel et al.; 2006) and

adjusted, using human epidemiologic data, for potential risk for non-Hodgkin lymphoma (NHL) and liver cancer. The oral unit risk for cancer is 5×10^{-2} per mg/kg/day, resulting from physiologically based pharmacokinetic model-based route-to-route extrapolation of the inhalation unit risk based on the human kidney cancer risks (Charbotel et al. 2006) and adjusted, using human epidemiologic data, for potential risk for NHL and liver cancer. There is high confidence in these unit risks for cancer, as they are based on good quality human data, as well as being similar to unit risk estimates based on multiple rodent bioassays. Evidence is sufficient to conclude that TCE operates through a mutagenic mode of action for kidney tumors. Evidence is insufficient and TCE-specific quantitative data are lacking on early-life susceptibility. Additional information about TCE is available from ATSDR at: <http://www.atsdr.cdc.gov/toxpro2.html>.

In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, TCE was detected in 1,013 systems serving 29.5 million people (EPA, 2009). Concentrations of TCE were greater than the MCL in 195 systems serving close to 12 million people. TCE was the fifth highest occurring regulated volatile organic chemical found based on the percent of population served by systems with at least one sample detection found from the 6 Year Review data (EPA, 2009).

Biomonitoring Information

Levels of halogenated solvents in blood reflect recent exposure. Blood levels of TCE were generally not detected in the NHANES 2003-2004 subsample and were detected infrequently in previous U.S. surveys (CDC, 2009).

Comparatively higher blood levels of tetrachloroethene and TCE have been noted for urban and industrial residential settings than for rural settings (Barkley et al., 1980; Begerow et al., 1996; Brugnone et al., 1994). Finding a measurable amount of any of these solvents in blood does not mean that the level of the solvent causes an adverse health effect. Biomonitoring studies of blood halogenated solvents can provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of halogenated solvents than levels found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

Sources of TCE

TCE does not occur naturally in the environment. However, it has been found in underground water sources and many surface waters as a result of the manufacture, use, and disposal of the chemical (ATSDR, 2003).

TCE Regulation and Monitoring

The EPA has set a maximum contaminant level for TCE in drinking water of

	<p>0.005 milligrams per liter (0.005 mg/L) or 5 parts of TCE per billion parts water. The EPA has also developed regulations for the handling and disposal of trichloroethylene.</p> <p>OSHA has set an exposure limit of 100 parts of TCE per million parts of air (100 ppm) for an 8-hour workday, 40-hour work week (ATSDR, 2003).</p>
Use of Measure	<p>These measures can assist by addressing the following surveillance functions:</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to TCE at different concentrations. • Maximum concentrations provide information on the peak potential exposure to TCE at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells may be another source of population exposure to TCE. Transient non-community water systems, which are regulated by EPA, also may be an important source of TCE exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Ground water systems may have multiple wells with different TCE concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the TCE concentration of people served by wells with higher TCE concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different TCE levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. ACGIH. TLVs and BEIs Based on the documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. 2007. Signature Publications. Cincinnati OH. p.104. 2. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for tetrachloroethylene update. 1997a [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp18.html. 4/22/09 3. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for trichloroethylene update. 1997b [online]. Available at URL: http://www.atsdr.cdc.gov/toxprofiles/tp19.html. 4/22/09 4. Agency for Toxic Substances and Disease Registry (ATSDR). ToxFAQs™ for

Trichloroethylene (TCE), July 2003. Available at:

<http://www.atsdr.cdc.gov/toxfaqs/tf.asp?id=172&tid=30>

5. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for Methylene chloride update. 2000 [online]. Available at URL: <http://www.atsdr.cdc.gov/toxprofiles/tp14.html>. 4/22/09
6. Armstrong SR, Green LC. Chlorinated hydrocarbon solvents. *Clin Occup Environ Med* 2004;4(3):481-496.
7. Barkley J, Bunch J, Bursey JT, Castillo N, Cooper SD, Davis JM, et al. Gas chromatography mass spectrometry computer analysis of volatile halogenated hydrocarbons in man and his environment—a multimedia environmental study. *Biomed Mass Spectrom* 1980;7(4):139-147.
8. Begerow J, Jermann E, Keles T, Freier I, Ranft U, Dunemann L. Internal and external tetrachloroethene exposure of persons living in differently polluted areas of Northrhine-Westphalia (Germany). *Zentralbl Hyg Umweltmed*. 1996;198(5):394-406.
9. Brugnone F, Perbellini L, Guiliari C, Cerpelloni M, Soave M. Blood and urine concentrations of chemical pollutants in the general population. *Med Lav* 1994;8(5):370-389.
10. Centers for Disease Control and Prevention (CDC). Fourth National Report on Human Exposure to Environmental Chemicals, 2009. Available at: <http://www.cdc.gov/exposurereport/pdf/FourthReport.pdf>
11. Charbotel B, Fevotte J, Hoirs M, Martin JL, Bergeret A. Case-control study on renal cell cancer and occupational exposure to trichloroethylene. Part II: Epidemiological aspects. *Ann. Occup. Hyg.* Vol. 50, No. 8, pp. 777-787, 2006.
12. International Agency for Research in Cancer (IARC). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals Vol. 63, 1995. Available at: <http://monographs.iarc.fr/ENG/Monographs/vol63/mono63.pdf>
13. Martin SA, Simmons MB, Ortiz-Serrano M, Kendrick C, Gallo A, Campbell J, et al. Environmental exposure of a community to airborne trichloroethylene. *Arch Environ Occup Health* 2005;60(6):341-316.
14. Monster AC. Biological monitoring of chlorinated hydrocarbon solvents. *J Occup Med* 1986;28:583-588.
15. Monster AC, Regouin-Peeters W, Van Schijndel A, van der Tuin J. Biological monitoring of occupational exposure to tetrachloroethene. *Scand J Work Environ Health* 1983;9:273-281.
16. Moran MJ, Zogorski JS, Squillace PJ. Chlorinated solvents in groundwater of the United States. *Environ Sci Technol* 2007;41:74-81.
17. National Toxicology Program (NTP). Report on Carcinogens, 12th ed. 2010. [online]. Available at URL: <http://ntp.niehs.nih.gov/ntp/roc/twelfth/ListedSubstancesReasonablyAnticipated.pdf> 5/9/2013
18. Rowe BL, Toccalino PL, Moran MJ, Zogorski JS, Price CV. Occurrence and potential human-health relevance of volatile organic compounds in drinking water from domestic wells in the United States. *Environ Health Perspect* 2007;115(11):1539-1546.
19. Schreiber JS, House S, Prohonic E, Smead G, Hudson C, Styk M, et al. An investigation of indoor air contamination in residences above dry cleaners. *Risk*

	<p>Anal 1993;13(3):335-344.</p> <p>20. U.S. Environmental Protection Agency (U.S. EPA). The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.</p> <p>21. U.S. Environmental Protection Agency (U.S. EPA). Toxicological Review of Trichloroethylene (CAS No. 79-01-76) In Support of Summary Information on the Integrated Risk Information System (IRIS), September 2011. EPA/635/R-09/011F http://www.epa.gov/IRIS/toxreviews/0199tr/0199tr.pdf.</p> <p>22. Wallace L, Nelson W, Ziegenfus R, Pellizzari E, Michael L, Whitmore R, et al. The Los Angeles TEAM Study: Personal exposures, indoor-outdoor air concentrations, and breath concentrations of 25 volatile organic compounds. J Exp Anal Environ Epidemiol 1991;1(2):157-192.</p>
--	--

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: URANIUM (U)
 ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Yearly distribution of number of Community Water Systems (CWS) by maximum Uranium concentration (cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium). 2. Yearly distribution of number of CWS by mean Uranium concentration (cut-points: cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium). 3. Mean concentration of Uranium at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 4. Yearly distribution of number of people served by CWS by maximum Uranium concentration (cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium). 5. Yearly distribution of number of people served by CWS by mean Uranium concentration (cut-points: 0-5, >5-15, >15-30, >30 µg/L Uranium).
Derivation of Measures	Uranium measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	Uranium, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	1999 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Uranium (U) and Public Health</p> <p>Uranium is a silver-white metal that is extremely dense and weakly</p>

radioactive. It usually occurs as an oxide and is extracted from ores containing less than 1% natural uranium. Natural uranium is a mixture of three isotopes: ^{238}U (greater than 99%), ^{235}U (about 0.72%), and ^{234}U (about 0.01%). Uranium has many commercial uses, including nuclear weapons, nuclear fuel, in some ceramics, and as an aid in electron microscopy and photography. Depleted uranium (DU) refers to uranium in which the proportions of ^{235}U and ^{234}U isotopes have been reduced compared with the proportion in natural uranium. Since the 1990's, DU has been used by the military in armor-piercing ammunition and as a component of protective armor for tanks. Natural and depleted uranium are primarily chemical toxicants, with radiation playing a minor role or no role at all (ATSDR, 2009).

Everyone is exposed to uranium in food, air, and water as part of the natural environment. (ATSDR, 2009). Variable concentrations of uranium occur naturally in drinking water sources. In some locations the natural concentrations may have increased due to mining and milling of uranium. Thus, the primary exposure sources for non-occupationally exposed persons are likely dietary and drinking water. Populations most heavily exposed to uranium are those employed in mining and milling operations, or in uranium enrichment and processing activities (ATSDR, 2009). In workplaces that involve uranium mining, milling, or processing, human exposure occurs primarily by inhaling dust and other small particles. Exposure to DU may occur in military personnel from retention of internal shrapnel that contains DU or exposure to dust generated from ammunition impact.

Absorption of uranium compounds is low by all routes of exposure (i.e., ingestion, inhalation, and skin contact). Depending upon the specific compound and solubility, 0.1%-6% of an ingested dose may be absorbed. Inhaled uranium-containing particles are retained in the lungs, where limited absorption occurs (less than 5%). After long term or repeated exposure, kidneys, liver, and bones can accumulate uranium with the largest amounts being stored in bones (Li et al., 2005). Uranium is eliminated in feces and urine; about 50% of the absorbed dose is eliminated in the urine within the first 24 hours. After exposure to soluble uranium salts, the initial half-life of uranium is about 15 days (Bhattacharyya et al., 1992), which represents distribution and excretion, with much slower elimination from bone. After inhalation, the half-life of insoluble uranium in the lungs is several years (Durakovic et al., 2003).

Human health effects from uranium at low environmental doses or at biomonitored levels from low environmental exposures are unknown. Health outcomes that may occur with uranium overexposure, based on both observed human effects and animal studies, include non-malignant respiratory disease (fibrosis, emphysema) and nephrotoxicity. Studies of persons with

chronic exposure to elevated uranium salts in drinking water have shown changes in urinary biomarkers potentially associated with impaired kidney function (Kurttio et al., 2006). IARC and NTP have no ratings for uranium human carcinogenicity. Radiation risks from exposure to natural uranium are very low. Alpha radiation (such as that from uranium) is classified as a human carcinogen. However, human studies have not found elevated rates of cancer from uranium exposure, and high-dose animal studies have not found cancer following inhalation, oral, or dermal exposure to uranium.

Workplace air standards and guidelines for external exposure to soluble and insoluble uranium compounds have been established by OSHA and ACGIH, respectively. Drinking water and other environmental standards have been established by U.S. EPA. Information about external exposure (i.e., environmental levels) and health effects is available from ATSDR at: <http://www.atsdr.cdc.gov/toxpro2.html>.

In an analysis of occurrence data from the EPA 6 Year Review of National Primary Drinking Water Regulations, uranium was detected in 4,101 systems serving close to 55 million people (EPA, 2009). Concentrations of uranium were greater than the MCL in 448 systems serving close to 8.4 million people (EPA, 2009).

Biomonitoring Information

Levels of urinary uranium reflect recent and ongoing or accumulated exposure. A previous nonrandom subsample from NHANES III (n = 499) (Ting et al., 1999) and other small populations have shown urinary concentrations that are similar to those in NHANES 1999-2000, 2001-2002, and 2003-2004 (Dang et al., 1992; Galletti, 2003; Karpas et al., 1996; Tolmachev et al., 2006). Older studies have demonstrated urinary uranium concentrations that are consistent with levels in the U.S. population, in that the levels were below their respective detection limits (Byrne et al., 1991; Hamilton et al., 1994; Komaromy-Hiller et al., 2000). In a study of 105 persons exposed to natural uranium in well water, urinary levels of uranium were as high as 9.55 µg/L (median 0.162 µg/L) (Orloff et al., 2004). Eighty-five percent of those levels were above the 95th percentile of the NHANES 1999-2000 population. The U.S. Nuclear Regulatory Commission (NRC) has set an action level of 15 µg/L urinary uranium to protect people who are occupationally exposed (NRC, 1978). Finding a measurable amount of uranium in urine does not mean that the level of uranium causes an adverse health effect. Biomonitoring studies on levels of uranium provide physicians and public health officials with reference values so that they can determine whether people have been exposed to higher levels of uranium than are found in the general population. Biomonitoring data can also help scientists plan and conduct research on exposure and health effects.

	<p>Sources of Uranium</p> <p>Uranium is a naturally-occurring element found in the earth's crust. It is naturally abundant in rocks, soil and water. Significant concentrations of uranium can occur in phosphate rock deposits, and in minerals such as pitchblende and uraninite. The total amount of Uranium on earth stays virtually the same because it has such a long half-life (4.47x10⁹ years for U-238) (EPA, 2010).</p> <p>Uranium Regulation and Monitoring</p> <p>The EPA limits the amount of uranium that may be present in drinking water to 30 ug/L (EPA, 2009). A gross alpha particle activity measurement may be substituted for the required uranium measurement provided that the measured gross alpha particle activity does not exceed 15 pCi/l.</p> <p>Monitoring frequency</p> <p>Once a CWS has satisfied initial monitoring requirements (4 quarterly samples at every entry point to the distribution system within the first quarter after initiating the source); the required frequency for Uranium monitoring is once every three years if the average of the initial monitoring results for the contaminant is greater than one-half the MCL but at or below the MCL. States may allow CWS to reduce the frequency of monitoring from once every three years to once every six or nine years at each sampling point, if the average of the initial monitoring results for each contaminant is below the detection limit. If a system has a monitoring result that exceeds the MCL while on reduced monitoring, the system must collect and analyze quarterly samples at that sampling point until the system has results from four consecutive quarters that are below the MCL, unless the system enters into another schedule as part of a formal compliance agreement with the State (CFR, 2002).</p>
Use of Measure	<p>These measures can assist by addressing the following surveillance functions:</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to Uranium at different concentrations. • Maximum concentrations provide information on the peak potential exposure to Uranium at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Private wells may be another source of population exposure to Uranium. Transient non-community water systems, which are regulated by EPA, may also be an important source of Uranium exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations</p>

	in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.
Data Sources	State grantee
Limitations of Data Sources	<p>The required monitoring frequency for Uranium is infrequent (every 3 to 6 years) and may be as intermittent as every nine years; therefore most states will have very little data on this contaminant.</p> <p>Ground water systems may have multiple wells with different Uranium concentrations that serve different parts of the population. Compliance samples are taken at each entry point to the distribution system. In systems with separate wells serving some branches or sections of the distribution system, the system mean would tend to underestimate the Uranium concentrations of people served by wells with higher Uranium concentrations. Exposure may be higher or lower than estimated if data from multiple entry points for water with different Uranium levels are averaged to estimate levels for the PWS.</p>
Related Indicators	Public Water Use; combined Radium-226 and -228
References	<ol style="list-style-type: none"> 1. Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological Profile for uranium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. 2. Bhattacharyya MH, Breitenstein BD, Metivier H, Muggenburg BA, Stradling GN, Volf V. Guidebook for the treatment of accidental internal radionuclide contamination of workers. In: Gerber GB, Thomas RG, eds. Radiation protection dosimetry. Vol. 41 (1). Kent (England): Nuclear Technology Publishing; 1992. pp. 1-49. 3. Byrne AR, Benedik L. Uranium content of blood, urine and hair of exposed and non-exposed persons determined by radiochemical neutron activation analysis, with emphasis on quality control. Sci Total Environ 1991;107:143-157. 4. Centers for Disease Control and Prevention (CDC). Third National Report on Human Exposure to Environmental Chemicals. Atlanta (GA). 2005. 4/20/09 5. Code of Federal Regulations (CFR), 2002. Title 40 Protection of the Environment Chapter I--Environmental Protection Agency Part 141--National Primary Drinking Water Regulations 141.26 Monitoring frequency and compliance requirements for radionuclides in community water systems. Available at: URL: http://www.access.gpo.gov/nara/cfr/waisidx_02/40cfr141_02.html 6. Dang HS, Pullat VR, Pillai KC. Determining the normal concentration of uranium in urine and application of the data to its biokinetics. Health Phys 1992;62:562-566. 7. Durakovic A, Horan P, Dietz LA, Zimmerman I. Estimate of the time zero lung burden of depleted uranium in Persian Gulf War veterans by the 24-hour urinary excretion and exponential decay analysis. Mil Med 2003;168(8):600-605. 8. Ejniak JW, Carmichael AJ, Hamilton MM, McDiarmid M, Squibb K, Boyd P, et al. Determination of the isotopic composition of uranium in urine by inductively coupled plasma mass spectrometry. Health Phys 2000;78:143-146. 9. Galletti M, D'Annibale L, Pinto V, Cremisini C. Uranium daily intake and urinary

- excretion: a preliminary study in Italy. *Health Phys* 2003;85:228-235.
10. Gwiazda RH, Squibb K, McDiarmid M, Smith D. Detection of depleted uranium in urine of veterans from the 1991 Gulf War. *Health Phys* 2004;86:12-18.
11. Hamilton EI, Sabbioni E, Van der Venne MT. Element reference values in tissues from inhabitants of the European community. VI. Review of elements in blood, plasma and urine and a critical evaluation of reference values for the United Kingdom population. *Sci Total Environ* 1994;158:165-190.
12. Karpas Z, Halicz L, Roiz J, Marko R, Katorza E, Lorber A, et al. Inductively coupled plasma mass spectrometry as a simple, rapid, and inexpensive method for determination of uranium in urine and fresh water: comparison with LIF. *Health Phys* 1996;71(6):879-885.
13. Komaromy-Hiller G, Ash KO, Costa R, Howerton K. Comparison of representative ranges based on U.S. patient population and literature reference intervals for urinary trace elements. *Clin Chim Acta* 2000;296(1-2):71-90.
14. Kurttio P, Auvinen A, Salonen L, Saha H, Pekkanen J, Makelainen I, et al. Renal effects of uranium in drinking water. *Environ Health Perspect* 2002;110(4):337-342.
15. Kurttio P, Harmionen A, Saha H, Salonen L, Karpas Z, Komulainen H, Auvinen A. Kidney toxicity of ingested uranium from drinking water. *Am J Kidney Dis* 2006;47(6):972-982.
16. Li WB, Roth P, Wahl W, Oeh U, Hollriegel V, Paretzke HG. Biokinetic modeling of uranium in man after injection and ingestion. *Radiat Environ Biophys* 2005;44:29-40.
17. May LM, Heller J, Kalinsky V, Ejniak J, Cordero S, Oberbroekling KJ, et al. Military deployment human exposure assessment: urine total and isotopic uranium sampling results. *J Toxicol Environ Health A* 2004;67(8-10):697-714.
18. Orloff KG, Mistry K, Charp P, Metcalf S, Marino R, Shelly T, et al. Human exposure to uranium in groundwater. *Environ Res* 2004;94:319-326.
19. Ough EA, Lewis BM, Andrews WS, Bennett LG, Hancock RG, Scott K. An examination of uranium levels in Canadian forces personnel who served in the Gulf War and Kosovo. *Health Phys* 2002;82(4): 527-532.
20. Ting BG, Paschal DC, Jarrett JM, Pirkle JL, Jackson RJ, Sampson EJ, et al. Uranium and thorium in urine of United States residents: reference range concentrations. *Environ Res* 1999;81:45-51.
21. Tolmachev S, Kuwabara J, Noguchi H. concentration and daily excretion of uranium in urine of Japanese. *Health Phys* 2006;91(2):144-153.
22. U.S. Nuclear Regulatory Commission (NRC). Regulatory Guide 8.22-Bioassay at uranium mills. Washington (DC): NRC; July 1978.
23. U.S. Environmental Protection Agency . Radiation Protection, Uranium, 2010. Available at: <http://www.epa.gov/radiation/radionuclides/uranium.html>
24. U.S. Environmental Protection Agency. The Analysis of Regulated Contaminant Occurrence Data from public Water Systems in Support of the Second Six-year Review of National Primary Drinking Water Regulations. EPA-815-B-09-006, October 2009.

CONTENT DOMAIN: COMMUNITY WATER
INDICATOR: DISINFECTION BYPRODUCTS
 ENVIRONMENTAL PUBLIC HEALTH TRACKING

Type of EPHT Indicator	Hazard, Exposure
Measures	<p>Level of Contaminant in Finished Water</p> <ol style="list-style-type: none"> 1. Quarterly distribution of number of Community Water Systems (CWS) by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) µg/L HAA5). 2. Yearly distribution of number of CWS by maximum HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) µg/L HAA5). 3. Yearly distribution of number of CWS by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) µg/L HAA5). 4. Mean concentration of HAA5 (µg/L) at CWS-level, by year. 5. Quarterly distribution of number of CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) µg/L TTHM). 6. Yearly distribution of number of CWS by maximum TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) µg/L TTHM). 7. Yearly distribution of number of CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) µg/L TTHM). 8. Mean concentration of TTHM at CWS-level, by year. <p>Potential Population Exposure to Contaminants in Finished Water</p> <ol style="list-style-type: none"> 9. Quarterly distribution of number of people served by CWS by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) µg/L HAA5). 10. Yearly distribution of number of people served by CWS by maximum HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) µg/L HAA5). 11. Yearly distribution of number of people served by CWS by mean HAA5 concentration (cut-points: (0-15), (>15-30), (>30-45), (>45-60), (>60-75), (>75) µg/L HAA5). 12. Quarterly distribution of number of people served by CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) µg/L TTHM). 13. Yearly distribution of number of people served by CWS by maximum TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) µg/L TTHM). 14. Yearly distribution of number of people served by CWS by mean TTHM concentration (cut-points: (0-20), (>20-40), (>40-60), (>60-80), (>80-100), (>100) µg/L TTHM).

	(>100) µg/L TTHM).
Derivation of Measures	Disinfection byproducts measures will be developed from water system attribute and water quality data stored in state Safe Drinking Water Act (SDWA) databases such as the Safe Drinking Water Information System (SDWIS/State). Trihalomethanes comprise chloroform, bromodichloromethane, dibromochloromethane, bromoform and their sum, denoted total trihalomethanes (TTHM). Haloacetic acids comprise trichloroacetic acid, dichloroacetic acid, monochloroacetic acid, dibromoacetic acid, monobromoacetic acid, and their sum, denoted HAA5. Data will be cleaned and transformed to a standard format. Analytical results of drinking water samples (usually taken at entry points to the distribution system or representative sampling points after treatment) will be used in conjunction with information about each CWS (such as service population and latitude and longitude of representative location of the CWS service area) to generate the measures.
Units	concentration of HAA5, µg/L concentration of TTHM, µg/L
Geographic Scope	State and Community Water System by County
Geographic Scale	The finest detail will be approximate point location of the community water distribution system represented by water withdrawal point, water distribution extents, principal county served, or principal city served.
Time Period	2002 or earliest year available to most current year of data abstraction.
Time Scale	Calendar year
Rationale	<p>Disinfection By Products and Public Health</p> <p>Disinfection byproducts (DBP) are formed when disinfectants used to inactivate microbial contaminants in water react with materials, primarily organic matter, in the water (Bellar et al. 1974, Rook 1974, Cedergren et al. 2002, Sadiq and Rodriguez 2004). Several hundred DBPs in over a dozen chemical classes have been identified (Woo et al. 2002, Krasner et al. 2006). Most commonly, DBPs form when chlorine reacts with naturally occurring organic matter in the source water.</p> <p>DBPs have been associated with both cancer and adverse pregnancy outcomes. High DBP levels, mainly for THMs, have been linked to bladder, colon and rectal cancer (King and Marrett 1996, Cantor et al. 1998, Amy et al. 2005, Villanueva et al. 2004, Villanueva et al. 2007), with bladder cancer reported most frequently. Although findings about adverse pregnancy outcomes have been less definitive, DBPs have been implicated in fetal loss (Swan et al. 1998, Waller et al. 1998, King et al. 2000, Dodds et al. 2004) and a variety of adverse birth outcomes involving growth (Bove et al. 1995, Gallagher et al. 1998, Wright et al. 2004, Infante-Rivard 2004, Toledano et al. 2005) and birth defects (Dodds et al. 1999, Klotz and Pynch 1999, Dodds and King 2001, Cedergren et al. 2002, Shaw et al. 2003). In contrast, however, other research has found little effect on birth outcomes (Savitz et al., 2006).</p>

Animal, microbial, in vitro and modeling studies have also pointed to toxicity or carcinogenicity of a wide variety of DBPs (Boorman 1999, Komulainen 2004). Numerous studies have indicated that different DBPs among the THMs and HAAs have different health effects. A number of studies have suggested that iodinated and brominated DBPs are more toxic than their chlorinated counterparts (Plewa et al. 2002, 2004, Richardson 2005). It is therefore appropriate that the tracking network follow individual DBP species and not just class totals (*c.f.* Singer 2006).

Sources of DBPs

DPB levels tend to be highest in water derived from surface sources because ground water generally has little organic matter (Symons et al. 1975, Whitaker et al. 2003). Ground water can, however, produce relatively high levels of the more brominated DBPs when the water, due either to geological circumstances (Whitaker et al. 2003) or salt water intrusion in coastal areas (von Gunten 2003), has elevated levels of bromide.

Bromate and chlorite are formed primarily after disinfection by ozone and chlorine dioxide, respectively. Sampling for these DBPs is required only for treatment plants that use the disinfectants that form them. Ozonation and chlorine dioxide are less common mechanisms of disinfection so these two DBPs will not be tracked initially. The disinfection processes that produce these two byproducts are likely to be used more often in the future so bromate and chlorite should be considered for eventual incorporation into the tracking network.

DBP Regulation and Monitoring

Safe Drinking Water Act (SDWA) regulation of DBPs began with the 1979 Total Trihalomethane Rule. This rule set an interim MCL for total trihalomethanes (TTHM), defined as the sum of four trihalomethanes, of 0.10 mg/L for community water systems (CWS) serving 10,000 or more people and using a disinfectant. The Stage 1 Disinfectants and Disinfection Byproducts Rule of 1998 (US EPA 1998) reduced the MCL for TTHM to 0.080 mg/L, added MCLs for the sum of five haloacetic acids (HAA5) of 0.060 mg/L, bromate of 0.010 mg/L and chlorite of 1.0 mg/L, and increased the scope of the rule to cover all CWS that disinfect. The rule had phased compliance with a date of 1 January 2002 for public water systems (PWS) with 10,000 or more people with a surface water or ground water under direct influence source and a date of 1 January 2004 for all other affected PWSs. The Stage 2 Disinfectants and Disinfection Byproducts Rule of 2006 (US EPA 2006) did not alter MCLs but did change how compliance with MCLs will be calculated and requires that PWSs evaluate their distribution systems for appropriate sampling locations. The results of this evaluation may affect the number and location of samples. The scope of the rule also increased to cover consecutive systems that receive finished water from other systems. The first reporting deadline for compliance with the Stage 2 rule was in 2006 but it will be a number of years before the rule requires the new compliance calculations based on

	<p>routine DBP samples.</p> <p>Currently, therefore, Safe Drinking Water Act standards exist for two classes of halogenated organic DBPs, trihalomethanes (THM) and haloacetic acids (HAA), and for two inorganic compounds, bromate and chlorite (US EPA, 2007). Given the near ubiquity of chlorine disinfection, the THMs and HAAs are useful indicators of risk for other DBPs because they occur at high levels and are easily measured.</p> <p>In summary, evidence suggests that disinfection byproducts adversely affect human health. The THMs and HAAs are the most commonly formed DBPs that are routinely tracked in state Safe Drinking Water Act databases. Measures based on these contaminants thus provide a window into potential human exposure to DBPs in publicly provided drinking water. They show where people are potentially exposed to high levels of DBPs. These water supply systems are candidates for enhancement of source water quality, infrastructure improvements or other interventions to reduce DBP exposure.</p>
Use of Measure	<p>These measures assist by providing data that can be used for surveillance purposes.</p> <ul style="list-style-type: none"> • Distribution measures provide information on the number of CWS and the number of people potentially exposed to nitrate at different concentrations. • Maximum concentrations provide information on the peak potential exposure to nitrate at the state level. • Mean concentrations at the CWS level provide information on potential exposure at a smaller geographic scale.
Limitations of The Measure	<p>The current measures are derived for CWS only. Transient non-community water systems, which are regulated by EPA, may also be an important source of DBPs exposure. Measures do not account for the variability in sampling, numbers of sampling repeats, and variability within systems. Concentrations in drinking water cannot be directly converted to exposure, because water consumption varies by climate, level of physical activity, and between people (EPA 2004). Due to errors in estimating populations, the measures may overestimate or underestimate the number of affected people.</p>
Data Sources	State grantee
Limitations of Data Sources	<p>Safe Drinking Water Act compliance data include only a handful of the hundreds of known DBPs (Weinberg et al. 2002), most of which occur in chemical classes other than THMs and HAAs. While compliance sampling for THMs and HAAs is directed at the DBPs thought to be most commonly produced by chlorination, non-regulated DBPs exist even among the THMs and HAAs.</p> <p>Concern has also been expressed about iodinated THMs and HAAs which, while present in lower concentrations than the brominated and chlorinated THMs, are thought to be toxic at lower doses (e.g. Plewa et al. 2004).</p>

THMs and HAAs may not be the most satisfactory indicators of DBP levels in waters subject to alternative disinfection methods that produce different DBPs in different proportions than chlorination (Richardson 2002, Weinberg et al. 2002) and may result in high levels of unregulated DBPs. Little is known about the quantitative occurrence of these DBPs in the distribution system (Richardson et al. 2002, Krasner et al. 2006). While the health effects of different DBPs may vary, with some suspected to be hazardous, few have been characterized for their effects on human health (Woo et al. 2002).

Correlations among different DBPs can be relatively low (King et al. 2004, Rodriguez et al. 2004a) so that the measured concentrations of THMs and HAAs may not be good predictors of exposure to other DBPs or overall DBP exposure. THM4 or HAA5, which are the only available data in some state databases, may therefore tell little about the relative concentrations of the THMs or HAAs.

DBP levels vary seasonally (Singer et al. 1981, Whitaker et al. 2003, Rodriguez et al. 2004b). Quarterly samples may not capture maximum levels and may not even adequately reflect short term levels. They may therefore be inadequate for estimating exposure during critical periods of a pregnancy, which may be as short as two to three weeks, especially if peak exposure matters more than average exposure. Furthermore, these fluctuations make it difficult to characterize levels with a single number such as an annual average and thus pose challenges to the development of meaningful synopses of patterns and trends.

DBP levels are spatially and temporally labile within a distribution system (Rodriguez et al. 2004b). THM levels increase with time after disinfection and therefore with distance from the treatment plant (Chen and Weisel 1998, Rodriguez and Sérodes 2001). HAA levels may increase or decrease (Chen and Weisel 1998, Rodriguez et al. 2004b), depending upon distribution system conditions. Rechlorination further increases DBP levels. For all but small distribution systems it is therefore impossible to adequately characterize DBP levels with a single value. DBP sampling locations may change over time, making it more difficult to compare measurements from year to year. Better estimation of DBP levels will require spatial and hydraulic modeling of distribution systems.

Water supply systems sample for DBPs on different schedules that range from quarterly to triennially. Different sampling frequencies complicate comparisons among different water supply systems. Long intervals between samples, although allowed only where THM and HAA levels have been found to be well under the MCL, create greater uncertainty about levels between sampling dates and require stronger assumptions when estimating exposure during short term events such as pregnancies. When allowed, annual or triennial monitoring takes place during the month of warmest weather and may therefore overestimate average DBP levels.

	<p>Water supply systems that have disinfection waivers generally have no DBP sample results. While the default assumption that these water supply systems have DBP concentrations of zero is generally reasonable, low levels of DBPs can be found in raw ground water, e.g., from surface contamination or from movement of chlorinated water from onsite wastewater treatment systems into ground water.</p> <p>Human behavior greatly influences exposure, complicating efforts to estimate exposure from tap water measurements (Nieuwenhuijzen et al. 2000, Kaur et al. 2004, Nuckols et al. 2005). Among the influences on exposure are showering and bathing time, consumption of tap water, use of bottled water, and exposure to water at workplaces or other locations outside the home. Moreover, ascertaining DBP levels in drinking water does not address other routes of exposure such as swimming (Villanueva et al. 2007, Zwiener et al. 2007). This consideration is not strictly a limitation of the measure but pertains to using the measure as an indicator of exposure.</p> <p>Some state SDWA databases may contain only totals for THMs and HAAs and may not record sample results for individual DBPs. Measures involving individual THMs and HAAs cannot be calculated for these states.</p>
Related Indicators	Public Water Use
References	<ol style="list-style-type: none"> 1. Amy G., G. Craun, S. W. Krasner, K. P. Cantor, M. Hildesheim, P. Weyer, and W. D. King. 2005. Improved exposure assessment on existing cancer studies. Awwa Research Foundation, Denver, Colorado. 2. Bellar, T. A., J. J. Lichtenberg, and R. C. Kroner. 1974. The occurrence of organohalides in chlorinated drinking waters. <i>Journal American Water Works Association</i> 66:703-706. 3. Boorman, G. A., V. Dellarco, J. K. Dunnick, R. E. Chapin, S. Hunter, F. Hauchman, H. Gardner, M. Cox, and R. C. Sills. 1999. Drinking water disinfection byproducts: Review and approach to toxicity evaluation. <i>Environmental Health Perspectives</i> 107 (suppl 1):207-217. 4. Bove, F. J., M. C. Fulcomer, J. B. Klotz, J. Esmart, E. M. Dufficy, and J. E. Savrin. 1995. Public drinking water contamination and birth outcomes. <i>American Journal of Epidemiology</i> 141:850-862. 5. Cantor, K. P., C. F. Lynch, M. Hildesheim, M. Dosemeci, J. Lubin, M. Alavanja, and G. Craun. 1998. Drinking water source and chlorination byproducts I. Risk of bladder cancer. <i>Epidemiology</i> 9:21-28. 6. Cedergren, M. I., A. J. Selbing, O. Lofman, and B. A. J. Kallen. 2002. Chlorination byproducts and nitrate in drinking water and risk for congenital cardiac defects. <i>Environmental Research</i> 89:124-130. 7. Chen, W. J., and C. P. Weisel. 1998. Halogenated DBP concentrations in a distribution system. <i>Journal American Water Works Association</i> 90:151-163. 8. Dodds, L., W. King, C. Woolcott, and J. Pole. 1999. Trihalomethanes in public water supplies and adverse birth outcomes. <i>Epidemiology</i> 10:233-237. 9. Dodds, L., and W. D. King. 2001. Relation between trihalomethane compounds and birth defects. <i>Occupational & Environmental Medicine</i> 58:443-446. 10. Dodds, L., W. D. King, A. C. Allen, B. A. Armson, D. B. Fell, and C. Nimrod. 2004. Trihalomethanes in public water supplies and risk of stillbirth. <i>Epidemiology</i> 15:179-186.

11. Gallagher, M. D., J. R. Nuckols, L. Stallones, and D. A. Savitz. 1998. Exposure to trihalomethanes and adverse pregnancy outcomes. *Epidemiology* 9:484-489.
12. Infante-Rivard, C. 2004. Drinking water contaminants, gene polymorphisms, and fetal growth. *Environmental Health Perspectives* 112:1213-1216.
13. Kaur, S., M. J. Nieuwenhuijsen, and P. Steer. 2004. Exposure of pregnant women to tap water related activities. *Occupational and Environmental Medicine* 61:454-460.
14. King, W. D., L. Dodds, and A. C. Allen. 2000. Relation between stillbirth and specific chlorination by-products in public water supplies. *Environmental Health Perspectives* 108:883-886.
15. King, W. D., L. Dodds, B. A. Armson, A. C. Allen, D. B. Fell, and C. Nimrod. 2004. Exposure assessment in epidemiologic studies of adverse pregnancy outcomes and disinfection byproducts. *Journal of Exposure Analysis and Environmental Epidemiology* 14:466-472.
16. King, W. D., and L. D. Marrett. 1996. Case-control study of bladder cancer and chlorination by-products in treated water (Ontario, Canada). *Cancer Causes & Control* 7:596-604.
17. Klotz, J. B. and L. A. Pynch. 1999. Neural tube defects and drinking water disinfection by-products. *Epidemiology* 10:383-90.
18. Komulainen, H. 2004. Experimental cancer studies of chlorinated by-products. *Toxicology* 198(1-3):239-48.
19. Krasner, S. W., H. S. Weinberg, S. D. Richardson, S. J. Pastor, R. Chinn, M. J. Scilimenti, G. D. Onstad, and A. D. Thruston, Jr. 2006. The occurrence of a new generation of disinfection byproducts. *Environmental Science & Technology* 40:7175-7185.
20. Nieuwenhuijsen, M. J., M. B. Toledano, and P. Elliott. 2000. Uptake of chlorination disinfection by-products; a review and a discussion of its implications for exposure assessment in epidemiological studies. *Journal of Exposure Analysis & Environmental Epidemiology* 10:586-599.
21. Nuckols, J. R., D. L. Ashley, C. Lyu, S. M. Gordon, A. F. Hinckley, and P. Singer. 2005. Influence of tap water quality and household water use activities on indoor air and internal dose levels of trihalomethanes. *Environmental Health Perspectives* 113:863-870.
22. Plewa, M. J., Y. Kargalioglu, D. Vanker, R. Minear, and E. D. Wagner. 2002. Mammalian cell cytotoxicity and genotoxicity analysis of drinking water disinfection by-products. *Environmental & Molecular Mutagenesis* 40:134-142.
23. Plewa, M. J., E. D. Wagner, S. D. Richardson, A. D. Thruston, Jr., Y.-T. Woo, and A. B. McKague. 2004. Chemical and biological characterization of newly discovered iodoacid drinking water disinfection byproducts. *Environmental Science & Technology* 38:4713-4722.
24. Richardson, S. D. 2005. New disinfection by-product issues: Emerging DBPs and alternative routes of exposure. *Global NEST Journal* 7:43-60.
25. Richardson, S. D., J. E. Simmons, and G. Rice. 2002. Disinfection byproducts: The next generation. *Environmental Science & Technology* 36:198A-205A.
26. Roberts, M. G., P. C. Singer, and A. Obolensky. 2002. Comparing total HAA and total THM concentrations using ICR data. *Journal American Water Works Association* 94:103-114.
27. Rodriguez, M. J., and J. B. Serodes. 2001. Spatial and temporal evolution of trihalomethanes in three water distribution systems. *Water Research* 35:1572-1586.
28. Rodriguez, M. J., M. Huard, and J. B. Sérodes. 2004a. Experimental study of the formation of chlorination by-products in potable water of Quebec City, Canada. *Bulletin of Environmental Contamination & Toxicology* 72:211-218.
29. Rodriguez, M. J., J. B. Sérodes, and P. Levallois. 2004b. Behavior of trihalomethanes and haloacetic acids in a drinking water distribution system. *Water Research* 38:4367-4382.

30. Rook, J. J. 1974. Formation of haloforms during chlorination of natural waters. *Water Treatment and Examination* 23:234-243.
31. Sadiq, R., and M. J. Rodriguez. 2004. Disinfection by-products (DBPs) in drinking water and predictive models for their occurrence: a review. *Science of the Total Environment* 321:21-46.
32. Savitz, D. A., P. C. Singer, A. H. Herring, K. E. Hartmann, H. S. Weinberg, and C. Makarushka. 2006. Exposure to drinking water disinfection by-products and pregnancy loss. *American Journal of Epidemiology* 164:1043-1051.
33. Shaw, G. M., D. Ranatunga, T. Quach, E. Neri, A. Correa, and R. R. Neutra. 2003. Trihalomethane exposures from municipal water supplies and selected congenital malformations. *Epidemiology* 14:191-199.
34. Singer, P. C. 2006. DBPs in drinking water: Additional scientific and policy considerations for public health protection. *Journal American Water Works Association* 98:73-80.
35. Singer, P. C., J. J. Barry III, G. M. Palen, and A. E. Scrivner. 1981. Trihalomethane formation in North Carolina drinking waters. *Journal American Water Works Association* 73:392-401.
36. Swan, S. H., K. Waller, B. Hopkins, G. Windham, L. Fenster, C. Schaefer, and R. R. Neutra. 1998. A prospective study of spontaneous abortion: relation to amount and source of drinking water consumed in early pregnancy. *Epidemiology* 9:126-133.
37. Symons, J. M., T. A. Bellar, J. J. Carswell, J. DeMarco, K. L. Kropp, G. G. Robect, D. R. Seeger, C. J. Slocum, B. L. Smith, and A. A. Stevens. 1975. National organics reconnaissance survey for halogenated organics. *Journal American Water Works Association* 67:634-647.
38. Toledano, M. B., M. J. Nieuwenhuijsen, N. Best, H. Whitaker, P. Hambly, C. de Hoogh, J. Fawell, L. Jarup, and P. Elliott. 2005. Relation of trihalomethane concentrations in public water supplies to stillbirth and birth weight in three water regions in England. *Environmental Health Perspectives* 113:225-232.
39. US EPA (United States Environmental Protection Agency). 1998. Stage 1 Disinfectants and Disinfection byproducts rule. *Federal Register* 63:69389-69476.
40. US EPA (United States Environmental Protection Agency). 2006. Stage 2 Disinfectants and Disinfection byproducts rule. *Federal Register* 71:387-493.
41. US EPA (United States Environmental Protection Agency). 2007. National Water Program Fiscal Year 2007 Guidance. US EPA Web site at http://water.epa.gov/resource_performance/planning/upload/FY2007_NWPG_Final_Web.pdf (accessed 5/9/2013).
42. Villanueva, C. M., K. P. Cantor, S. Cordier, J. J. K. Jaakkola, W. D. King, C. F. Lynch, S. Porru, and M. Kogevinas. 2004. Disinfection byproducts and bladder cancer: A pooled analysis. *Epidemiology* 15:357-367.
43. Villanueva, C. M., K. P. Cantor, J. O. Grimalt, N. Malats, D. Silverman, A. Tardon, R. Garcia-Closas, C. Serra, A. Carrato, G. Castano-Vinyals, R. Marcos, N. Rothman, F. X. Real, M. Dosemeci, and M. Kogevinas. 2007. Bladder cancer and exposure to water disinfection by-products through ingestion, bathing, showering, and swimming in pools. *American Journal of Epidemiology* 135:148-156.
44. von Gunten, U. 2003. Ozonation of drinking water: Part II. Disinfection and by-product formation in presence of bromide, iodide, or chlorine. *Water Research* 37:1469-1487.
45. Waller, K., S. H. Swan, G. DeLorenze, and B. Hopkins. 1998. Trihalomethanes in drinking water and spontaneous abortion. *Epidemiology* 9:134-140.
46. Weinberg H. S., S. W. Krasner, S. D. Richardson, and A. D. Thruston, Jr. 2002. The occurrence of disinfection by-products (DBPs) of health concern in drinking water: results

- | | |
|--|--|
| | <p>of a nationwide DBP occurrence study; EPA/600/R-02/068. US EPA, Athens, Georgia.</p> <p>47. Whitaker, H., M. J. Nieuwenhuijsen, N. Best, J. Fawell, A. Gowers, and P. Elliot. 2003. Description of trihalomethane levels in three UK water suppliers. <i>Journal of Exposure Analysis and Environmental Epidemiology</i> 13:17-23.</p> <p>48. Woo, Y.-T., D. Lai, J. L. McClain, M. K. Manibusam, and V. Dellarco. 2002. Use of mechanism-based structure-activity relationships analysis in carcinogenic potential ranking for drinking water disinfection by-products. <i>Environmental Health Perspectives</i> 110 (suppl 1):75-87.</p> <p>49. Wright, J. M., J. Schwartz, and D. W. Dockery. 2004. The effect of disinfection by-products and mutagenic activity on birth weight and gestational duration. <i>Environmental Health Perspectives</i> 112:920-925.</p> <p>50. Zwiener, C., S. D. Richardson, D. M. De Marini, T. Grummt, T. Glauner, and F. H. Frimmel. 2007. Drowning in disinfection byproducts? Assessing swimming pool water. <i>Environmental Science & Technology</i> 41:363-372.</p> |
|--|--|

Analyte	EPA method number	Standard method number	Detection Level
Arsenic	EPA 200.5		1.4 ug/L
Arsenic	EPA 200.8 ICPMS Method		0.2 ppb
Arsenic	EPA 200.8 ICPMS Method		1 ppb
Arsenic	EPA 200.7 ICP Method		10 ppb
Arsenic	EPA 200.9		0.5 ug/L
Arsenic	EPA 206.2		1 ug/L
Arsenic	EPA 206.3		2 ug/L
Arsenic	EPA 206.4		10 ug/L
Arsenic		Standard Methods 3113 B	1 ug/L
Arsenic		Standard Methods 3114 B	???
Arsenic		Standard Methods 3120 B	8 ug/L
Arsenic		Standard Method 3125 B	0.02 ug/L
Arsenic		Standard Method 3500-As B	1ug/L
Atrazine	EPA 505		2.4ug/L
Atrazine	EPA 525.2		.078 ug/L
Atrazine	EPA 508.1		.003 ug/L
Atrazine	EPA 551.1		.082 ug/L
Atrazine	EPA 527		.036 ug/L
Atrazine	EPA 1699		14 pg/L
Atrazine	EPA 507		.015 ug/L
Bis(2-ethylhexyl)phthalate (DEHP)	EPA 1625		10 ug/L
Bis(2-ethylhexyl)phthalate (DEHP)	EPA 606		2 ug/L
Bis(2-ethylhexyl)phthalate (DEHP)	EPA 506		2.25 ug/L
Bis(2-ethylhexyl)phthalate (DEHP)	EPA 525.2		.46 ug/L
Bis(2-ethylhexyl)phthalate (DEHP)		Standard Methods 6410	2.5 ug/L
Haloacetic acids	EPA Method 552.1		0.32 ug/L for Dalapon, 0.24 ug/L for monobromoacetic acid
Haloacetic acids	EPA Method 552.2		0.204 ug/L for monobromoacetic acid
Haloacetic acids		Standard Method 6251B	0.5-30ppb
Nitrate	EPA Method 300.0		.002 mg/L
Nitrate	EPA Method 353.2		0.02 mg N/L
Nitrate, nitrite	EPA Method 300.1		0.010 mg N/L
Nitrate		Standard Methods 4110 B	3.7 ug/L

Nitrate		Standard Methods 4110 C	17 ug/L
Nitrate		Standard Methods 4500-NO3-B	????
Nitrate		Standard Methods 4500-NO3-C	0.05 to 2 mg NO ₃ ⁻ -N/L
Nitrate		Standard Methods 4500-NO3-D	0.14 to 1400 mg NO ₃ ⁻ -N/L
Nitrate		Standard Methods 4500-NO3-E	0.01 to 1.0 mg NO ₃ ⁻ -N/L
Nitrate		Standard Methods 4500-NO3-F	0.1 to 10 mg NO ₃ ⁻ -N/L
Nitrate		Standard Methods 4500-NO3-H	0.01 to 10 mg NO ₃ ⁻ -N/L
Nitrate		Standard Methods 4500-NO3-I	0.00025 to 10 mg NO ₃ ⁻ -N/L
Radium		Standard Methods 7500-RA B	????
Radium		Standard Methods 7500-RA C	.03 pCi/L
Tetrachloroethylene (PCE)	EPA 601		.03 ug/L
Tetrachloroethylene (PCE)	EPA 624		4.1 ug/L
Tetrachloroethylene (PCE)	EPA 1624		10 ug/L
Tetrachloroethylene (PCE)	EPA 502.2 by PID		.05 ug/L
Tetrachloroethylene (PCE)	EPA 502.2 by ELCD		.04 ug/L
Tetrachloroethylene (PCE)	EPA 524.2		.05 ug/L
Tetrachloroethylene (PCE)	EPA 524.3		.036 ug/L
Tetrachloroethylene (PCE)	EPA 551.1		.002 ug/L
Tetrachloroethylene (PCE)	EPA 8021B (by GC-ELCD)		.04 ug/L
Tetrachloroethylene (PCE)	EPA 8021B (by GC-PID)		.05 ug/L
Tetrachloroethylene (PCE)		Standard Methods 6200B	.04 ug/L
Tetrachloroethylene (PCE)		Standard Methods 6200C	.01 ug/L
Trichloroethylene (TCE)	EPA 601		.12 ug/L
Trichloroethylene (TCE)	EPA 624		1.9 ug/L
Trichloroethylene (TCE)	EPA 1624		10 ug/L
Trichloroethylene (TCE)	EPA 502.2 by PID		.02 ug/L
Trichloroethylene (TCE)	EPA 502.2 by ELCD		.01 ug/L
Trichloroethylene (TCE)	EPA 524.2		.02 ug/L
Trichloroethylene (TCE)	EPA 524.3		.035 ug/L
Trichloroethylene (TCE)	EPA 551.1		.002 ug/L
Trichloroethylene (TCE)	EPA 8021B (by GC-ELCD)		.01 ug/L
Trichloroethylene (TCE)	EPA 8021B (by GC-PID)		.02 ug/L
Trichloroethylene (TCE)		Standard Methods 6200B	.04 ug/L

Trichloroethylene (TCE)		Standard Methods 6200C	.01 ug/L
Trihalomethanes	EPA method 502.2		0.02 ug/L
Trihalomethanes	EPA method 524.2		<=0.2 ppb
Trihalomethanes	EPA method 524.3		0.025 ug/L
Trihalomethanes	EPA method 551.1		0.05 ug/L
Trihalomethanes	EPA method 601		0.05 ug/L
Trihalomethanes		Standard Methods 6200 B	0.12 ug/L
Trihalomethanes		Standard Methods 6200 C	0.01 ug/L
Trihalomethanes		Standard Methods 6232 B	0.1 to 200 ug/L
Trihalomethanes		Standard Methods 6232 C	According to SM, detection lir
Trihalomethanes		Standard Methods 6232 D	According to SM, detection lir
Uranium	EPA 200.8		.1 ug/L
Uranium	EPA 200.10		.03 ug/L
Uranium		Standard Methods 3125	.001 ug/L

www.nemi.gov is a website that has all of the methods. You can search by analyte and get the list of methods, a descriptive name, the

EPA approved method for drinking water compliance monitoring*

*No information on EPA approved THM methods

Does not include ASTM methods

Method description	Comments
AVICP-AES	
ICP/MS	Detection limit in water when DRC mode is used on the ICPMS, IDL = 0.025 ug/L
ICP/MS	Detection limit in water without DRC mode on the ICPMS
ICP	Detection limit in water; used when sample has high salt content (>500 ppm). Estimated detection level per Standard Methods = 50
Determination of trace elements by stabilized temperature graphite Furnace Atomic Absorption (GFAA)	
Arsenic by GFAA	
Arsenic by gaseous hydride generation and Atomic Absorption Spectrophotometry	
Metals in water by GFAA	
Manual hydride generation/atomic absorption spectrometric method	
Total recoverable metals in water by ICP	
ICP/MS	
Silver Diethyldithiocarbamate (detectic	Best when interferences are absent
Microextraction and Gas Chromatography system equipped with a linearized electron capture detector (ECD)	
Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry	
Liquid-Solid Extraction and Electron Capture Gas Chromatography	
Liquid/Liquid Extraction and Gas Chromatography with Electron Capture Detection	
Solid phase extraction and capillary column gas chromatography/mass spectrometry	
High resolution gas chromatograph/high resolution mass spectrometer	
Capillary column gas chromatography (GC) system equipped with a nitrogen-phosphorus detector (NPD).	
Capillary column gas chromatography (GC)	
Gas chromatography (GC) system equipped with an electron capture detector (ECD).	
Liquid-Liquid Extraction or Liquid Solid Extraction and Gas Chromatography with Photoionization Detection	
Liquid-Solid Extraction and Capillary Column Gas Chromatography/Mass Spectrometry	
Liquid-Liquid Extraction Gas Chromatographic/Mass Spectrometric Method	
Ion-Exchange Liquid-Solid Extraction and Gas Chromatography with an Electron Capture Detector	
Liquid-liquid extraction, derivitization and gas chromatography with electron capture detection	
Micro Liquid-Liquid Extraction Gas Chromatographic method	
Ion chromatography	
Colorimetric analysis. Nitrate rxn with	! Minimum reporting limit of 0.1 mg N/L
Ion chromatography method. Uses Dionix IC with an AS23 column	
Ion chromatography with chemical suppression of Eluent Conductivity	

Ion chromatography

UV spectrophotometric Very old (no longer approved)

Second-derivative UV Spectrophotometric Proposed method

Nitrate Electrode Method Several interferences (NO₂, Cl, S, Br, I, and others)

Cadmium reduction

Automated Cadmium reduction

Automated Hydrazine reduction

Cadmium reduction flow

Precipitation Method

Emanation Method

Purgeable Halocarbons via GC with Electrolytic conductivity (ELCD) or microcoulometric detector

Purgeable Organic Compounds via GC/MS

Volatile Organic Compounds by GC/MS

Purge and Trap Capillary Column Gas Chromatography with Photoionization and Electrolytic Conductivity Detectors in Series

Purge and Trap Capillary Column Gas Chromatography with Photoionization and Electrolytic Conductivity Detectors in Series

Purgeable Organic Compounds in Water by Capillary Column Gas Chromatography/Mass Spectrometry

Capillary Column Gas Chromatography/Mass Spectrometry

Liquid/Liquid Extraction and Gas Chromatography with Electron Capture Detection

Gas Chromatography Using Photoionization and/or Electrolytic Conductivity Detectors

Gas Chromatography Using Photoionization and/or Electrolytic Conductivity Detectors

Purge and Trap Capillary-Column GC/MS Method

Purge and Trap Capillary-Column GC Method

Purgeable Halocarbons via GC with Electrolytic conductivity (ELCD) or microcoulometric detector

Purgeable Organic Compounds via GC/MS

Volatile Organic Compounds by GC/MS

Purge and Trap Capillary Column Gas Chromatography with Photoionization and Electrolytic Conductivity Detectors in Series

Purge and Trap Capillary Column Gas Chromatography with Photoionization and Electrolytic Conductivity Detectors in Series

Purgeable Organic Compounds in Water by Capillary Column Gas Chromatography/Mass Spectrometry

Capillary Column Gas Chromatography/Mass Spectrometry

Liquid/Liquid Extraction and Gas Chromatography with Electron Capture Detection

Gas Chromatography Using Photoionization and/or Electrolytic Conductivity Detectors

Gas Chromatography Using Photoionization and/or Electrolytic Conductivity Detectors

Purge and Trap Capillary-Column GC/MS Method

Purge and Trap Capillary-Column GC Method

VOCs in water by GC/PID/ELCD

VOCs in water using GCMS Reporting limit is 0.5 ppb

Purgeable organic compounds in water by GCMS

chlorinated compounds in water using GC-ECD

purgeable halocarbons via GC with electrolytic conductivity (ELCD) or microcoulometric detector

Purge and Trap capillary-column GC/MS

Purge and Trap capillary-column gas chromatographic method

Detection levels highly dependent on characteristics of the gas

Liquid-liquid extraction gas chromatographic system used, the ratio of solvent to water, and

chromatographic method interferences present in the solvent

Purge and Trap Gas Chromatographic/Mass Spectrometric Method

Purge and Trap Gas Chromatographic

Metals in water by ICP/MS

: source of the method, the instrumentation, and the detection level

CUTPOINTS

Chemical Name	Chemical_ID	Cutpoint_ID	Cutpoint_Desc	Cutoff point for ND bin
Arsenic	1005	1	ND*	Arsenic 10 ug/L
Arsenic	1005	2	(0-5)	
Arsenic	1005	3	(>5-10)	
Arsenic	1005	4	(>10-20)	
Arsenic	1005	5	(>20-30)	
Arsenic	1005	6	(>30)	
HAA5	2456	1	ND	HAA5 10 ug/L
HAA5	2456	2	(0-15)	
HAA5	2456	3	(>15-30)	
HAA5	2456	4	(>30-45)	
HAA5	2456	5	(>45-60)	
HAA5	2456	6	(>60-75)	
HAA5	2456	7	(>75)	
Nitrate	1040	1	ND	Nitrate 1 mg/L
Nitrate	1040	2	(0-3)	
Nitrate	1040	3	(>3-5)	
Nitrate	1040	4	(>5-10)	
Nitrate	1040	5	(>10-20)	
Nitrate	1040	6	(>20)	
TTHM	2950	1	ND	TTHM 10 ug/L
TTHM	2950	2	(0-20)	
TTHM	2950	3	(>20-40)	
TTHM	2950	4	(>40-60)	
TTHM	2950	5	(>60-80)	
TTHM	2950	6	(>80-100)	
TTHM	2950	7	(>100)	
DEHP	2039	1	ND	DEHP 3 ug/L
DEHP	2039	2	(0 - 2)	
DEHP	2039	3	(>2 - 4)	
DEHP	2039	4	(>4- 6)	
DEHP	2039	5	(>6 - 10)	
DEHP	2039	6	(>10)	
Atrazine	2050	1	ND	Atrazine 2 ug/L
Atrazine	2050	2	(0 - 1)	
Atrazine	2050	3	(>1- 3)	
Atrazine	2050	4	(>3- 4)	

Atrazine	2050	5	(>4)	
TCE	2984	1	ND	TCE 2 ug/L
TCE	2984	2	(0 - 1)	
TCE	2984	3	(>1- 2)	
TCE	2984	4	(>2- 5)	
TCE	2984	5	(>5)	
PCE	2987	1	ND	PCE 2 ug/L
PCE	2987	2	(0 - 1)	
PCE	2987	3	(>1- 2)	
PCE	2987	4	(>2- 5)	
PCE	2987	5	(>5)	
Radium	4010	1	ND	Radium 2 pCi/L
Radium	4010	2	(0- 3)	
Radium	4010	3	(>3-5)	
Radium	4010	4	(>5-10)	
Radium	4010	5	(>10)	
Uranium	4006	1	ND	Uranium 5 ug/L
Uranium	4006	2	(0-5)	
Uranium	4006	3	(>5-15)	
Uranium	4006	4	(>15-30)	
Uranium	4006	5	(>30)	
N/A	5	1	N/A	

MCL		
Chemical Name	MCL	Unit
DEHP	6	ug/L
Atrazine	3	ug/L
TCE	5	ug/L
PCE	5	ug/L
Radium	5	pCi/L
Uranium	30	ug/L
Arsenic	10	ug/L
Nitrate	10	mg/L
TTHM	80	ug/L
HAA5	60	ug/L

***To populate the ND (non-detect) bin, you must select those values from CWS for which # of samples equals the # of non-detects. A CWS will only be designated as ND for an analyte if all samples for that time period (annual or quarterly) were ND and it is less than or equal to the 'cutoff point' for that analyte.**

Appendix C

Tracking Network Metadata Content Guidance Document

Metadata Content Guidance Document



STANDARDS AND NETWORK DEVELOPMENT

Metadata Content Guidance Document

Acknowledgments

We would like to thank the following people for their contributions to this document.

Name	Title	Organization	Role
Metadata Subgroup Leadership			
Kochukoshy Cheruvettolil	Associate	Ross & Associates Consulting	Facilitator
Gonza Namulanda	Public Health Informatics Fellow	Centers for Disease Control	Chair Author
Jeff Patridge	GIS Analyst	Missouri Office of Administration	Chair Author
Document Development Team			
Rodney Garland	Research Analyst	Oregon Public Health Division	Team Leader Author, Editor
Kim Kennedy	Consultant	Scientific Technologies Corporation	Contributor
Sanjaya Kumar	Research Scientist	New York State Dept. of Health	Author
Rene LeBlanc	Systems Analyst	Maine Dept. of Health and Human Services	Contributor
Sam LeFevre	Program Manager	Utah Dept. of Health	Team Leader Author
Lisa Parker	Database Analyst	Maine Dept. of Health and Human Services	Contributor
Chuck Shorter	Field Epidemiologist	Tulane University	Author
Jeanne Zborowski	Principle Investigator	University of Pittsburgh	Author
Metadata Subgroup Members			
Carol Bingaman	IT Consultant	Pennsylvania Dept. of Health	Contributor
Chris Duclos	GIS Manager	Florida Dept. of Health	Contributor
John Folsom	Database Administrator	Florida Dept. of Health	Contributor
Sara Kustron	GIS Coordinator	Massachusetts Dept. of Public Health	Contributor
Robert Levey	Computer Systems Manager	New York City Dept. of Health and Mental Hygiene	Contributor
Rashid Malik	DP Program Analyst	Maryland Dept. of Health and Mental Hygiene	Contributor
Richard Opiekun	Research Scientist	New Jersey Dept. of Health and Senior Services	Contributor
Judy Rager	Epidemiologist	University of Pittsburgh	Contributor
Misty Richard	Epidemiologist	New Hampshire Dept. of Health and Human Services	Contributor
Juley Rycheck	Epidemiologist	University of Pittsburgh	Contributor
Parveeza Shaikh	Database Specialist	Maryland Dept. of Health and Mental Hygiene	Contributor
Megan Tehan	Epidemiologist	New Hampshire Dept. of Health and Human Services	Contributor
Contractor Support			
Madhu Chaganthi	Network Architecture	Northrop Grumman	Support
Rhonda A. Martin	Project Manager	Northrop Grumman	Support
Crystal Spellman	Business Analyst	Northrop Grumman	Support

This page left intentionally blank



Table of Contents

Chapter 1: How to Use This Guide	1
1.1 What is the Purpose of This Guide?	1
1.2 Using This Guide	1
Chapter 2: Introduction	2
2.1 What is Metadata?.....	2
2.2 Why is Metadata Important to EPHT?	3
2.3 Who Creates Metadata Standards?	3
2.4 What is the FGDC Metadata Standard?	4
2.5 What is the EPHT Metadata Profile?	4
Chapter 3: Structure	5
3.1 Metadata Sections.....	6
Section 1: Identification Information.....	6
Section 2: Data Quality	6
Section 3: Spatial Data Organization.....	6
Section 4: Spatial Reference	6
Section 5: Entity and Attribute	6
Section 6: Distribution.....	6
Section 7: Metadata Reference	7
Section 8: Citation	7
Section 9: Time Period	7
Section 10: Contact	7
Chapter 4: Exceptions	8
4.1 What if I have latitudes and longitudes in my data set?	8
Chapter 5: Elements	9
5.1 Element Guidance Tables	9
5.2 Section 1: Identification Information	9
Originator	9
Publication Date	10
Title.....	11
URL	12
Abstract.....	14
Purpose	15
Supplemental Info	16
Currentness	17
Progress	18
Maintenance and Update Frequency	19
Spatial Domain (Composite Element).....	20
Theme Keyword Thesaurus.....	23
Theme Keyword	24
Place Keyword Thesaurus	25
Place Keyword.....	27

Table of Contents

Access Constraints.....	28
Use Constraints.....	30
Point of Contact (Composite Element).....	32
Security Classification System.....	33
Security Classification.....	34
Security Handling Description.....	36
Native Data Set Environment.....	37
5.3 Section 2: Data Quality.....	38
Logical Consistency Report.....	38
Completeness Report.....	40
Process Description.....	41
Process Date.....	43
5.4 Section 5: Entity and Attribute.....	44
Entity and Attribute Overview.....	44
Entity and Attribute Detail Citation.....	46
5.5 Section 6: Distribution.....	48
Distributor (Composite Element).....	48
Resource Description.....	49
Distribution Liability.....	50
Custom Order Process.....	52
5.6 Section 7: Metadata Reference.....	53
Metadata Date.....	53
Metadata Contact (Composite Element).....	54
Metadata Standard Name.....	55
Metadata Standard Version.....	56
Metadata Access Constraints.....	57
Metadata Use Constraints.....	58
5.7 Section 9: Time Period.....	59
Calendar Date.....	59
Beginning Date.....	60
Ending Date.....	61
Time of Day.....	62
Beginning Time.....	64
Ending Time.....	66
Section 10: Contact.....	68
Contact Organization.....	68
Contact Person.....	69
Contact Position.....	70
Address Type.....	71
Address.....	72
City.....	73
State or Province.....	74
Postal Code.....	75
Country.....	76
Contact Telephone Number.....	77
Contact TDD/TTY Telephone.....	78

Table of Contents

Contact FAX Number.....	79
Contact E-mail Address.....	80
Hours of Service	81
Contact Instructions.....	82
Chapter 6: Examples of Metadata Records	83
6.1 Cancer Dataset	83
EPHT Metadata:.....	83
6.2 Air Pollution Data	92
EPHT Metadata:.....	92
6.3 Asthma Hospitalizations Data	96
EPHT Metadata:.....	96
6.4 Drinking Water Data System	100
EPHT Metadata:.....	100
Chapter 7: Terms and Acronyms	106
Chapter 8: Citations and References	112
Appendix A: EPHTN Metadata Profile	113
Appendix B: Keywords from ISO 19115 Topic Categories	116



Chapter 1: How to Use This Guide

1.1 What is the Purpose of This Guide?

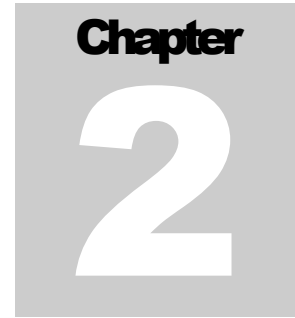
The purpose of this guide is to provide a user friendly manual that provides a brief introduction to metadata, the structure of metadata, and guidance for entering information required to create a metadata record for use in the Environmental Public Health Tracking Network (Tracking Network).



This document consists of seven chapters and one appendix. Chapters 1 through 3 provide basic information about metadata and Tracking Network concepts of metadata. Chapter 4 provides specific guidance with respect to entering content into a metadata record. Chapter 6 provides completed metadata records to illustrate what a final metadata entry should look like. Finally, chapters 7 and 8, as well as appendix A, provide background information about metadata and this guidance document.

1.2 Using This Guide

Although this guide contains basic information on metadata and the structure of metadata, its primary purpose is to provide detailed help on filling out the **content** of a metadata record. Therefore, the most relevant information is in Chapter 5. For specific guidance, use the Table of Contents to find the information needed.



Chapter 2: Introduction

2.1 *What is Metadata?*

A simple and often used definition of metadata is “data about data.” A commonly understood example of a type of metadata is the information one might use to search a computer catalog at your local library to find a book. In this catalog, a library patron might find information on the title, author, and abstract for the individual books found during a library search. These summaries would help determine which of the books contain the information of interest to the library patron conducting the search. Another way to define metadata is structured information that describes and makes it easier to retrieve or manage an information resource (NISO 2004).

These definitions, although useful, allow for variability in what type of information is collected to describe “data” or “information” and how metadata is gathered and presented. Therefore, the term “metadata” has different meanings to different organizations and professions. For example, the Open Management Group uses metadata to refer to computer-to-computer exchanges of information such as in the eXtended Markup Language (XML) Metadata Interchange (XMI). In the library environment, metadata commonly refers to a formal scheme to describe any type of object (NISO 2004).

These different needs and uses have resulted in several defined types of metadata. Dr. William Y. Arms, a recognized expert on digital library development from Cornell University, defined three types of metadata: descriptive, structural, and administrative (Arms, 2000). The National Information Standards Organization (NISO) also recognized these three types of metadata, but added the subtypes of rights management and preservation under administrative metadata (NISO 2004). The Getty Institute defined five types of metadata: administrative; descriptive; preservation; technical and use (Baca ed. 2000). Given all these types, needs, and definitions, it is no wonder there is confusion about what constitutes metadata.

For purposes of this document, metadata will be defined using the term “descriptive”. This is the most common type and use of metadata. The NISO (2004) definition of descriptive metadata is:

“Descriptive metadata describes a resource for purposes such as discovery and identification. It can include elements such as title, abstract, author, and keyword.”

This definition contains two key terms “describes” and “discovery”. These two key terms provide a framework for the form and use of descriptive metadata and places it in the context of a computer to human interaction. A metadata document that describes an object or resource can be searched and then discovered so that information can be found and evaluated.

With metadata defined and the type of metadata chosen (descriptive), a standard way to describe an object or resource is needed so that a systematic search can be conducted. To meet the particular needs for metadata by professionals, several organizations have developed “standards”. One simple definition of a standard within the context of metadata is a set of criteria, guidelines, and best practices for collecting information to describe an object or resource. A standard helps provide a framework so the information gathered and provided as metadata is similar, interoperable (able to be exchange between systems), and searchable.

2.2 Why is Metadata Important to EPHT?

Descriptive metadata is the “Backbone” of the Environmental Public Health Tracking Network. Its creation and maintenance is essential for the success of the Network. As a result, many of the early grant efforts have focused on its development.

Metadata is important to the Tracking Network for two reasons.

- ❑ It allows Network users to locate resources through a variety of means including keywords, geographic boundaries, and date and time.
- ❑ A Network user can determine the content of a resource, why created, how it was created, any limitations, access and use restrictions, data quality, and contact information. It helps a user to decide if a resource found on the Network is appropriate for the proposed use.

Data will be available through the Network only if it contains FGDC and Tracking Network-compliant metadata.

2.3 Who Creates Metadata Standards?

So who creates standards? Professional organizations and governmental institutions create standards. These different standards organizations create standards that meet the needs of their constituents. For descriptive metadata there are several organizations setting standards to meet their individual needs. Three such organizations that have created descriptive metadata standards are listed below.

- ☞ Dublin Core Metadata Initiative (Dublin Core; <http://dublincore.org/>) – Dublin Core’s mission statement states “The Dublin Core Metadata Initiative provides simple standards to facilitate the finding, sharing and management of information.” This is a widely used standard, particularly within the communities of library resources.
- ☞ International Organization for Standardization (ISO; <http://www.iso.org/iso/home.htm>–[ISO](#)) is an organization that works on standardizing many processes worldwide that benefits almost every sector of business, industry, and technology. ISO has also worked on standards for descriptive metadata for the general information documentation.
- ☞ Federal Geographic Data Committee (FGDC; <http://www.fgdc.gov/>) – FGDC is an organization devoted to promoting the coordinated development, use, sharing, and dissemination of geospatial (geographic) data. The FGDC standard for descriptive metadata is primarily focused for geospatial data and information, but can easily be adapted to more generic types of data and information.

2.4 What is the FGDC Metadata Standard?

The Tracking Network has adopted the FGDC metadata standard because it could capture both spatial and non-spatial information that may be present in EPHT data sets. In addition, federal agencies or any organization funded by those agencies is required under Executive Order 12906 to use this standard to capture information about geospatial datasets (www.archives.gov/federal-register/executive-orders/pdf/12906.pdf)

Currently the standard name is the Content Standard for Digital Geospatial Metadata (CSDGM), Version 2, (or FGDC-STD-001-1998). Eventually the FGDC standard will become the North American Profile (NAP), a profile compatible with ISO metadata standard 19115:2003. This work is being lead by the FGDC.

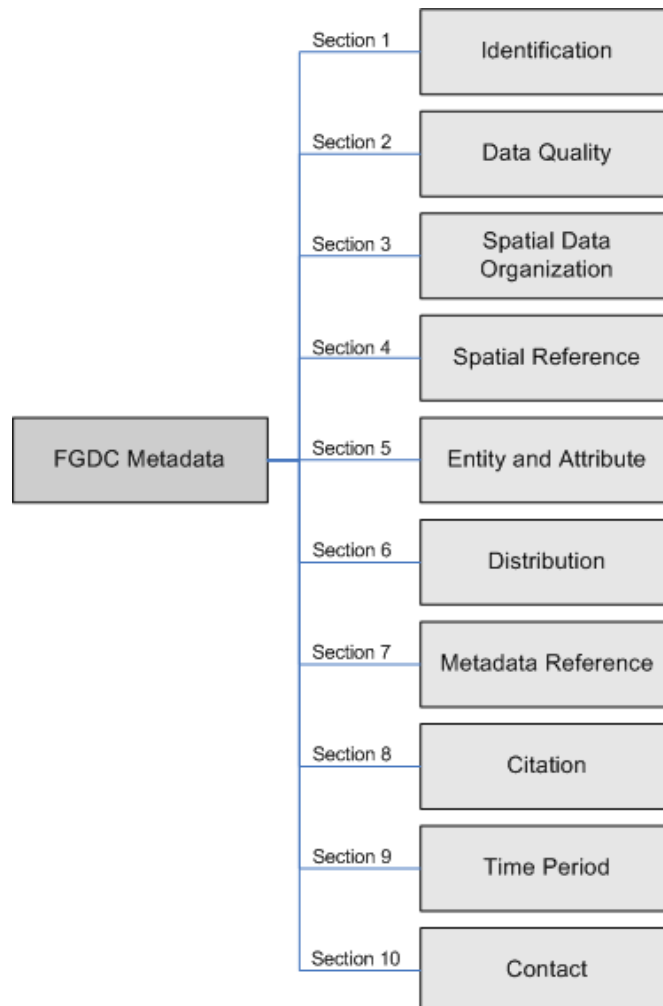
2.5 What is the EPHT Metadata Profile?

The EPHT Metadata Profile is a subset of the full FGDC Content Standard. It represents the minimum set of descriptive metadata elements that are required for making data resources available on the Environmental Public Health Tracking Network. EPHT Metadata Subgroup members, data stewards, and partners developed the profile. It contains all of the minimum elements required by FGDC and several optional elements that the group believed essential for documenting EPHT data.

Chapter 3

Chapter 3: Structure

Metadata (metadata from this point forward means metadata based on the FGDC Standard) consists of ten sections. All these sections have a hierarchy of parts called **elements**. Elements are the individual portions of the metadata standard that either form subsections or hold information entered by a user. A subsection is a **compound** element. Compound elements are placeholders within a section hierarchy that holds no information. All other elements contain information entered by a user. The figure below shows the names of the ten sections.



3.1 Metadata Sections

Each section of a metadata record has elements that will describe unique information on a data set. To describe any given data set not all of these sections are required. However, the totality of the sections and elements with those sections we cover almost all potential information needs for describing the majority of potential data sets. What follows are brief descriptions of the metadata sections.

Section 1: Identification Information

The Identification Information section is a central part of a metadata record. This section contains the basic information about a data set that will help a consumer of metadata determine the how, why, what, and when of a data set.

Section 2: Data Quality

The Data Quality section provides basic information about the quality of a dataset and helps the consumer of metadata determine if the dataset meets their basic quality requirements.

Section 3: Spatial Data Organization

The Spatial Data Organization section is the mechanism used to describe spatial (generally a Geographic Information System or GIS) data set. This section is not part of the EPHT Metadata Profile.

Section 4: Spatial Reference

The Spatial Reference section describes the coordinate system and other spatial information that is generally only applicable to a spatial (generally a GIS) data set. This section is not part of the EPHT Metadata Profile.

Section 5: Entity and Attribute

The Entity and Attribute section is likely the second most important part of a metadata record. It provides more detail on data set content or a link to other documents such as an existing data dictionary that provides information on the columns with a data set.

Section 6: Distribution

The Distribution section provides information on the organization and/or person to contact to obtain a data set, determine what the liability of distribution is, and how to order a dataset. Sometimes the distributor of a data set is not the same as the contact for the data set itself. The Tracking Network will only use the organization (corporate) contact because personal contact information within federal systems is Information in Identifiable Form (IIF) and is subject to additional security procedures.

Section 7: Metadata Reference

The Metadata Reference section provides information on the standard used for the metadata record itself, the record creation date, and who created the metadata record.

Section 8: Citation

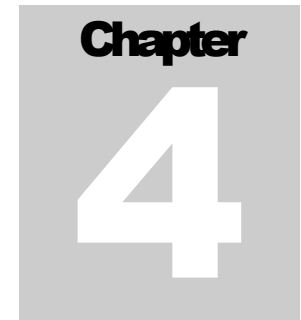
The Citation section provides the recommended reference for a data set. This section does not stand-alone but is part of other sections.

Section 9: Time Period

The Time Period section has information about the date and time of coverage of a data set.

Section 10: Contact

- The Contact section provides the basic contact information such as an organization name, phone number, and other information needed for a metadata consumer to contact an organization and/or individual to ask questions about the data set. The Contact section is used in multiple sections of a metadata record to provide contact information on the data owner, the creator of the metadata record, and the distributor of the data set itself, if these are different contact organizations and/or persons. The Tracking Network will only use the organization (corporate) contact because personal contact information within federal systems is Information in Identifiable Form (IIF) and is subject to additional security procedures.



Chapter 4: Exceptions

4.1 What if I have latitudes and longitudes in my data set?

There will be data sets with latitudes and longitudes as columns in the data set. This situation does not make the data a GIS layer, but does require that when the data is distributed, the data users know what datum the latitude and longitude is in. The most common datum's are: North American Datum of 1927 (NAD27), NAD83, and World Geodetic System of 1984 (WGS84). This scenario, assumes that the latitudes and longitudes are in correctly formatted decimal degrees.

For the above scenario there needs to be a method to inform the data users on the latitudes and longitudes datum. To do this, it is currently recommended that in the metadata element "Entity and Attribute Overview", the fact that latitudes and longitudes are columns in the data set be provided and the datum defined. This recommendation may change in the future as the Tracking Network Profile matures.



Chapter 5: Elements

5.1 Element Guidance Tables

The tables below provide details on each individual element and guidance on entering content into each element

5.2 Section 1: Identification Information

Originator

Element Name	Originator
Definition	The name(s) of the organization(s) that developed that data set.
Purpose and Meaning	Identify the developer of this dataset. Development means to create a new dataset using new or existing data, to edit an existing dataset or to compile a new dataset from existing data.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: The complete name of the organization(s) responsible for developing the dataset. Unknown: The developer of this dataset is unknown
Recommendations for Filling in the Entry	Should be the complete name of the organization that actually developed (built) this dataset, rather than the organization(s) that provided any source data used to create it. If the names of editors or compilers are provided, the name must be followed by “(ed.)” or “(comp.)” respectively.
Examples	Environmental Public Health Tracking Network, Misoretah Department of Health. U.S. Environmental Protection Agency (comp.).
Additional Resources	None
Other Comments	None
Things to Note	None

Publication Date

Element Name	Publication Date
Definition	The date when this dataset was published or otherwise made available for release.
Purpose and Meaning	Publication or release reference date. This date can be a version date as well.
Obligation	Mandatory
Occurrence	Single
Date Type	Date
Domain (Controlled Terms)	<p>Free Date: As complete a date as is available formatted as:</p> <ul style="list-style-type: none"> ▪ YYYYMMDD, where YYYY is the four-digit year, MM is the numeric value (1 – 12) for the month, and DD is the numeric value (01 – 31) for the day. Leave off the DD or MMDD if the month or day is unknown. <p>Unpublished Material: Use this value for planned or pending datasets. Change this value to the actual publication date when the dataset is completed.</p> <p>Unknown: The developer and publication date of this dataset is unknown</p>
Recommendations for Filling in the Entry	<p>Use the date of creation, modification, compilation of the dataset or the date of implementation onto the Tracking Network for this date.</p> <p>The date range (time domain) covered by the dataset should not be used.</p> <p>The FGDC specifies that the date format is YYYYMMDD</p>
Examples	<p><i>Only year is known:</i> 2007</p> <p><i>Year and month (January) are known:</i> 200701</p> <p><i>Year month and day are known (January 10):</i> 20070110</p>
Additional Resources	None
Other Comments	None
Things to Note	None

Elements

Title

Element Name	Title
Definition	The logical name by which the dataset is known.
Purpose and Meaning	Provides the name of the dataset.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: The complete name of the dataset. A dataset must have a name; therefore, no other options are available.
Recommendations for Filling in the Entry	This should be the complete logical name of the dataset. Provide the physical name of the dataset in the Native Data Set Environment element.
Examples	<i>Health Data:</i> Case Counts of Liver Cancers by County for Misoretah <i>Environmental Data:</i> Arsenic Levels in Drinking Water for Misoretah
Additional Resources	None
Other Comments	None
Things to Note	As a minimum, the name should include a theme (data subject).

URL

Element Name	URL
Definition	The name of an online computer resource that contains the dataset or application. Entries should follow the Uniform Resource Locator (URL) convention of the Internet.
Purpose and Meaning	The URL element is important for providing Tracking Network users with direct access to an online dataset or data resource described by the metadata record. URLs can provide access to a variety of data download, data clearinghouse and web-mapping services. Often web-based applications use this element as a means to directly link to a service or data layer. Complete this element if the dataset or resource is accessible via the Internet.
Obligation	Optional (Complete if applicable)
Occurrence	Single
Data Type	Text
Domain (Controlled Terms)	Free text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	<p>The URL should contain documentation of online linkage using the nomenclature that reflects the specifics of the resource (data, service, application). The use of a URL is encouraged within the Tracking Network to promote direct access to publicly available data sets and services.</p> <p>Providing online linkage for datasets that are not live services (static data) is straightforward. The user is only required to provide the URL link to the location of the file. Providing online linkage for live data and maps requires additional detail for correct consumption within other applications. ArcIMS Image Services, ArcIMS Feature Services, and WMS Image Services are classified as live mapping services. Each of these services may be consumed directly from a metadata record by web-based mapping applications if they are documented correctly. See examples below.</p>
Examples	<p><i>Static data:</i> (Health Data Example). The Pennsylvania Health Care Cost Containment Council (PHC4) provides inpatient hospitalization reports from 1998-2006 by county online. The URL for access is http://www.phc4.org/countyprofiles/.</p> <p><i>Live mapping services:</i> (Environmental Data Example) US EPA EnviroMapper Services For a service with the following parameters: <u>Server:</u> http://www.epa.gov/enviro <u>Service:</u> em The appropriate URL for the element would be: http://www.epa.gov/enviro/html/em/</p>

Elements

	To link directly to a map of regulated sites in Eighty Four, Pennsylvania within EnviroMapper, the complete link is: http://134.67.99.122/enviro/emef.asp?xl=-80.161079&yt=40.252019&xr=-80.027712&yb=40.096294
Additional Resources	None
Other Comments	None
Things to Note	None

Abstract

Element Name	Abstract
Definition	A brief narrative summary of the dataset.
Purpose and Meaning	This information provides a user with a brief description of the source and contents of the dataset
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	<p>The abstract should contain information about:</p> <ul style="list-style-type: none"> <input type="checkbox"/> subject, topic or theme of the dataset <input type="checkbox"/> the population included in or covered by the dataset; <input type="checkbox"/> the spatial domain (largest spatial unit; for example state) and scale (smallest spatial unit; for example county) of the dataset; and <input type="checkbox"/> temporal domain (range) and scale (time unit) of the dataset. <p>The abstract may also contain a brief description of the processes used to create or compile this dataset. Provide detailed description of the processes used in one or more Process Description elements.</p>
Examples	<p><i>Health Data:</i> This data set contains the annual case counts and standardized rates of liver cancers (ICDO-3 C220) among Misoretah residents for each county of the state of Misoretah from 1970 through 2007. Dataset compiled from Misoretah Cancer Registry data.</p> <p><i>Environmental Data:</i> Distribution point monitoring data from the Misoretah Division of Safe Drinking Water (MDSDW) used to compile average municipal water system arsenic concentration levels for all municipalities in the state of Misoretah. The dataset provide the number of samples, the earliest and latest sampling dates, and the minimum maximum, mean, median and standard deviation of the arsenic concentration.</p>
Additional Resources	None
Other Comments	None
Things to Note	As a minimum, the name should include a theme (data subject).

Purpose

Element Name	Purpose
Definition	A summary of the intentions with which the dataset was developed.
Purpose and Meaning	This section should address why the data set was developed and/or published. Generally, this will contain a reference to one or more state and/or national Tracking Network objectives.
Obligation	Mandatory
Occurrence	Single
Data Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	The purpose should be clear and concise. Information might include what were the objectives of the activities or research that resulted in this dataset; what objectives are served by presenting the data in digital (electronic) form; how should the data be used.
Examples	<p><i>Purpose:</i> To provide EPHT grantees, researchers, other public health professionals and the public with summary information on hospitalizations for asthma and myocardial infarction in the State of Mordana.</p> <p><i>Purpose:</i> To provide access to and enhance the use of information worldwide, advancing understanding of human interactions in the environment, and serving the needs of science, and public and private decision-making.</p> <p><i>Purpose:</i> To provide consultants, planners, and resource managers with information on wetland location and type. Data collected to meet U.S. Fish and Wildlife Service's mandate to map the wetland and deep-water habitats of the United States.</p>
Additional Resources	None
Other Comments	None
Things to Note	None

Supplemental Info

Element Name	Supplemental Information
Definition	Other descriptive information about the dataset.
Purpose and Meaning	This element is a text comment field in which to supply additional information about the dataset/resource not covered elsewhere. This includes related studies, dataset limitations, and notifications.
Obligation	Optional (Complete if applicable.)
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	Use this element to provide a more in depth discussion of the dataset or location. It might also be useful for a more in depth discussion of software tools made available to the Tracking Network by state or local health departments.
Examples	<p><i>Population/Health Example:</i> The data and documentation are visible through the United States-Mexico Demographic Data Viewer (US-MEX DDViewer) application at http://plue.sedac.ciesin.columbia.edu/plue/ddviewer/ddv30-USMEX/</p> <p><i>Environmental Resource Example:</i> (USGS Mineral Resources Data System) This file contains the software GSSEARCH, used to search, retrieve, and print the MRDS records. GSSEARCH is software developed at the U.S. Geological Survey as an outgrowth of a system to manage geologic bibliographic information. It supports fixed- or variable-length data and allows for full-text searching of specific indexed fields. It presents the selected records back to the user for perusal in both browse and detail formats. The records may also be printed or written to a disk file in four different formats: ASCII, fixed, comma delimited, and DBASE compatible.</p>
Additional Resources	None
Other Comments	None
Things to Note	None

Currentness

Element Name	Currentness
Definition	The basis on which the time period of content information is determined.
Purpose and Meaning	This element provides information on how “up-to-date” the dataset or data resource is.
Obligation	Mandatory
Occurrence	Single
Data Type	Free Text
Domain (Controlled Terms)	Publication Date: Used if the data is secondary or has been processed. As of Time Period End Date: Use this if the currentness as it applies to the source data.
Recommendations for Filling in the Entry	Information about the currentness of the dataset (how "up-to-date" is the dataset) is important to potential users. Most users are interested in the currentness of a dataset related to the "ground condition" (when the "real world" looked the way as described by the dataset). The Currentness element requires the producer to identify if the Time Period of Content dates and times refer to the ground condition or some other later time when the information was recorded, published, etc. If the data is secondary or been processed, then the phrase “Publication Date” should be used. Publication Data is reflective of processed data. “As of Time Period End Date” reflects the fact this is source data.
Examples	Publication Date As of Time Period End Date
Additional Resources	None
Other Comments	None
Things to Note	None

Progress

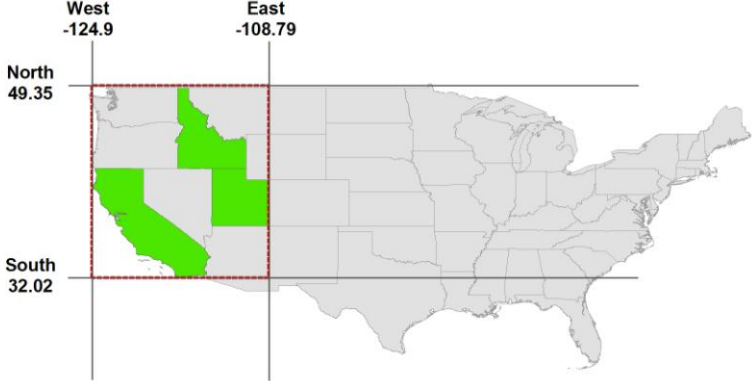
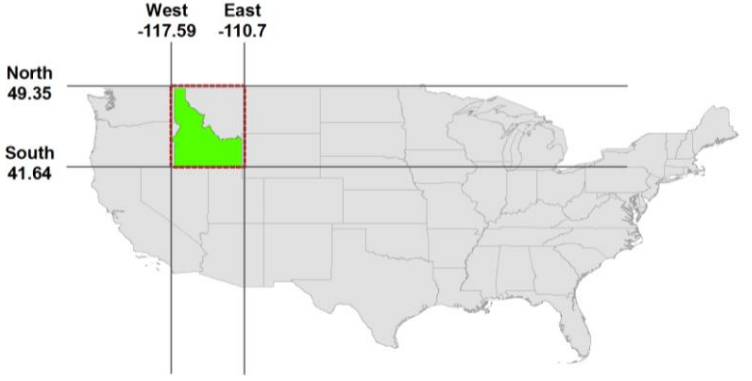
Element Name	Progress
Definition	The state of the dataset.
Purpose and Meaning	This information describes whether the dataset is in its final form, currently being added to, or if the data resource is in the planning stages.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	<p>Complete: The dataset is complete and ready for use. This does not imply that new data will not be added in the future.</p> <p>In Work: The dataset is not complete and is currently being edited or undergoing quality control.</p> <p>Planned: The dataset is currently being created.</p> <p>Free Text:</p>
Recommendations for Filling in the Entry	As metadata is released to the Tracking Network, certain datasets or resources might be in various stages of development. This element describes the current state of the dataset.
Examples	<p><i>Progress:</i> complete</p> <p><i>Progress:</i> planned</p>
Additional Resources	None
Other Comments	None
Things to Note	None

Maintenance and Update Frequency

Element Name	Maintenance And Update Frequency
Definition	The frequency that changes are made to the dataset after the initial dataset is complete.
Purpose and Meaning	Use this element to provide information for Tracking Network users as to the frequency of planned and expected updates to the described dataset.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Continually Daily Weekly Monthly Quarterly Annually Unknown As needed Irregular None planned Free text
Recommendations for Filling in the Entry	The update frequency will most likely depend on the type of data. For example, air quality data might be updated daily or weekly; hospitalizations for asthma might be updated quarterly. For some final datasets, perhaps no update is planned (e.g. mortality data for 2004)
Examples	See controlled terms.
Additional Resources	None
Other Comments	No definitions provided for the controlled terms, as they were considered self-evident.
Things to Note	None

Spatial Domain (Composite Element)

Element Name	<p>West Bounding Coordinate East Bounding Coordinate North Bounding Coordinate South Bounding Coordinate</p>
Definition	<p>The geographic area covered by the data set. For each of the individual coordinates it is either a latitude or longitude expressed in decimal degrees. West and East Bounding Coordinates are longitude and are negative for the United States. North and South Bounding Coordinates are positive latitudes for the United States.</p>
Purpose and Meaning	<p>The purpose of the spatial domain and its four elements is to “draw a box” around an area where data for the dataset is relevant. If a dataset is for an entire state, then a box is draw around the entire state. If a dataset contains only one county in a state, then a box is draw around only that county. For areas where there may be multiple, and potential widely dispersed, states that are part of a single dataset, a bounding box is drawn around the entire region. This may include state not included in the dataset.</p> <p>The purpose of spatial domain in the Tracking Network is that that it provides a basic spatial context to a dataset. The ultimate purpose is to provide a way in later version of the Network to search and discover metadata by using a service that allows for the inactive searching for metadata using a map tool.</p>
Obligation	Mandatory
Occurrence	Single
Date Type	Numeric in decimal degrees using the North American Datum of 1983.
Domain (Controlled Terms)	<p>-180.0 <= West Bounding Coordinate <= 180.0 -180.0 <= East Bounding Coordinate <= 180.0 -90.0 <= North Bounding Coordinate <= 90.0 -90.0 <= South Bounding Coordinate <= 90.0</p>
Recommendations for Filling in the Entry	<p>Without the consultation or use of GIS software, gathering-bounding coordinates can be difficult. It is recommended to initial seek help from a person familiar with geographic coordinates or have a table of bounding coordinates that will be repeatedly used for your area of interest. There is at least one freely available tool on the internet for determining basic boundary box coordinates. The additional resource section of this table provides a URL to the tool.</p> <p>Use decimal degrees. Use the minimum rectangular area that completely encloses the spatial domain of the dataset (i.e., a state, county, sub-county, or multi-state area). Recommend at least 2 and no more than 4 significant digits.</p>

<p>Examples</p>	<p>Regional Dataset:</p> <p>West Bounding Coordinate: -124.9 East Bounding Coordinate: -108.79 North Bounding Coordinate: 49.35 South Bounding Coordinate: 32.02</p>  <p>State Dataset:</p> <p>West Bounding Coordinate: -117.59 East Bounding Coordinate: -110.70 North Bounding Coordinate: 49.35 South Bounding Coordinate: 41.64</p> 
<p>Additional Resources</p>	<p>To determine basic bounding box coordinates use the FGDC tool: http://clearinghouse1.fgdc.gov/servlet/FGDCWizard.</p> <p>To convert Universal Transverse Mercator (UTM) coordinates to decimal degrees use the National Geodetic Survey conversion tool: http://www.ngs.noaa.gov/cgi-bin/utm_getgp.prl.</p> <p>To convert local State Plane coordinates to decimal degrees use the National Geodetic Survey conversion tool: http://www.ngs.noaa.gov/cgi-bin/spc_getgp.prl.</p>

Elements

	To convert latitude and longitude from degrees, minutes, and seconds format to decimal degrees use the FCC tool: http://www.fcc.gov/mb/audio/bickel/DDDMSS-decimal.html .
Other Comments	Spatial domain does not relate to the underlying geographic system of the data itself (if any) or any function of a Geographic Information System. It is always expressed in latitude and longitude using the North American Datum of 1983, and is always expressed in decimal degrees.
Things to Note	None

Theme Keyword Thesaurus

Element Name	Theme Keyword Thesaurus
Definition	Reference to a formally registered thesaurus or a similar authoritative source of theme keywords.
Purpose and Meaning	This source of a set of keywords and phrases is used to select the keywords that describe the content of a dataset.
Obligation	Mandatory
Occurrence	Multiple
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element
Recommendations for Filling in the Entry	Use at least, ISO 19115 Topic Category. Use other standard vocabularies/thesauri.
Examples	<p><i>For public health data:</i> ISO 19115. Public Health Information Network (PHIN) Vocabulary Standards and Specifications.</p> <p><i>For environmental data:</i> Consortium for International Earth Science Information Network (CIESIN) Indexing Vocabulary. Chemical Abstracts Service (CAS) CA Lexicon.</p>
Additional Resources	None
Other Comments	None
Things to Note	<p>Adding at least one keyword from ISO 19115 is in compliance with FGDC Version 3</p> <p>Thesaurus work in Public Health Information Network (PHIN), Vocabulary Access and Distribution System (VADS) may provide additional information in the future. See http://www.cdc.gov/PhinVSBrowser/StrutsController.do for more information of PHIN VADS.</p>

Theme Keyword

Element Name	Theme Keyword
Definition	Topic of the content of the dataset
Purpose and Meaning	This is a common-use word or phrase used to describe the general subject area of the dataset. Use a standardized set of key words and phrases to allow identification of dataset resources in any search. When users are searching for datasets, theme key words help eliminate resources that are of no interest.
Obligation	Mandatory
Occurrence	Multiple
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	Select terms covering the content of the dataset. Include broad and specific terms, and use controlled vocabularies/thesauri when possible. Include at least one ISO topic category referencing the ISO 19115 Thesaurus.
Examples	<p><i>For public health data:</i></p> <p>Cancer Birth defects Lead poisoning</p> <p><i>For environmental data:</i></p> <p>Natural Resources Toxics Ecology</p>
Additional Resources	None
Other Comments	None
Things to Note	<p>Adding at least one keyword from ISO 19115 complies with FGDC Version 3. See Appendix B of this Guidance Document for the ISO 19115 controlled terms.</p> <p>Thesaurus work in Public Health Information Network (PHIN), Vocabulary Access and Distribution System (VADS) may provide additional information in the future. See http://www.cdc.gov/PhinVSBrowser/StrutsController.do for more information of PHIN VADS.</p>

Place Keyword Thesaurus

Element Name	Place Keyword Thesaurus
Definition	Reference to a formally registered thesaurus or a similar authoritative source of theme keywords.
Purpose and Meaning	Place keywords are used for searching and discovering data based on a place name, such as the name of a state or a county. To help standardize the entry of place names a thesaurus is used. By using a thesaurus, all metadata creators will provide place names using the same system, thereby decreasing the potential for errors and the use of nonstandard names.
Obligation	Mandatory
Occurrence	Multiple
Date Type	Text
Domain (Controlled Terms)	<p>None: If no place name thesaurus is used for a group of place keywords then use “None”.</p> <p>GNIS: The Geographic Names Information System is the standard place name thesaurus for the United States.</p> <p>FIPS: The Federal Information Processing Standards is a numerical code assigned to U.S. Census Bureau areas.</p> <p>Free Text: User can write any other place name thesaurus used.</p>
Recommendations for Filling in the Entry	<p>Multiple place name thesauri can be used. Therefore, a metadata document can use GNIS as the thesaurus for a group of standardized place names and then use a local thesaurus or “None” for a group of place names that are only used locally.</p> <p>If is recommended to use at least the GNIS thesaurus for some place names for standardization. It is recommended to include at least one FIPS code for your place.</p>
Examples	GNIS FIPS None
Additional Resources	<p>The Geographic Names (GNIS) domestic names website: http://geonames.usgs.gov/ There you will find a searchable database of place names.</p> <p>Federal Information Processing Standards (FIPS) Website: http://www.itl.nist.gov/fipspubs/index.htm: FIPS codes for the United States.</p>
Other Comments	None

Elements

Things to Note	Thesaurus work in Public Health Information Network (PHIN), Vocabulary Access and Distribution System (VADS) may provide additional information in the future. See http://www.cdc.gov/PhinVSBrowser/StrutsController.do for more information of PHIN VADS.
----------------	---

Elements

Place Keyword

Element Name	Place Keyword
Definition	The geographic name of a location covered by a dataset (Includes city, county, state, state acronym, regional descriptions and references.)
Purpose and Meaning	<p>Place keywords are used for searching and discovering data based on a place name, such as the name of a state or a county. These keywords can come from thesauri or from the metadata creator.</p> <p>Place keywords are critical to finding resources for a particular area based on searching place names. Multiple place keywords can be entered. Therefore, if this were a dataset covering a region, all the states in that region are entered as individual keyword entries.</p>
Obligation	Mandatory
Occurrence	Multiple
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	<p>Provide full geographic name, acronyms, and FIPS codes.</p> <p>When entering state names, enter the full state name and the two-letter acronym.</p>
Examples	<p>Misoretah</p> <p>MH</p> <p>59</p>
Additional Resources	None
Other Comments	None
Things to Note	None

Access Constraints

Element Name	Access Constraints
Definition	Legal restrictions prerequisites for accessing the dataset. These include any access constraints applied to assure the protection of privacy or intellectual property, and any special restrictions or limitations on obtaining the dataset.
Purpose and Meaning	Used to identify any external restrictions on the access to the dataset. This usually applies to datasets that are exempt from public records laws such as endangered species, personal health, and intellectual properties. This element also provides an explanation for the security level applied to the dataset by describing the decision made for applying security restrictions.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free text: There are no controlled terms for this element
Recommendations for Filling in the Entry	<p>Identify the most common access restriction. Some datasets may be restricted due to sensitivity, whereas others might be considered draft and are not ready for distribution.</p> <p>For any single dataset, multiple access constraints may apply. Multiple constraints are shown as separate paragraphs within the Access Constraints narrative. Multiple constraints could include state or local standard access constraint language in combination with dataset specific constraints.</p> <p>Include any agency approval requirements (IRB, MOA, TPA, etc.). If agency approval is required, refer the user to the application process. Also, include any technology requirements (certification download, registration into a LDAP, etc.) for access. Direct the user to the protocols for completing those requirements.</p> <p>If local organization that governs a dataset has published access constraints, add a URL to those document(s) to the narrative.</p>
Examples	<p><i>For public health data:</i></p> <p>Data have been restricted due to the sensitive nature of the location information presented.</p> <p>Formal permission is required for access to this dataset. A formal IRB approval process is requirement to access this dataset. To inquire about getting IRB approval, contact the data steward listed in the Data contact section of this metadata. Additional information provided at: www.fakeurl.com/fake_instructions.html.</p>

Elements

	<i>For environmental data:</i> Data have been restricted due to the identification of sensitive habitats.
Additional Resources	None
Other Comments	None
Things to Note	None

Use Constraints

Element Name	Use Constraints
Definition	Restrictions and legal prerequisites for using the dataset after access is granted. These include any use constraints applied to assure the protection of privacy or intellectual property, and any special restrictions or limitations on using the data set.
Purpose and Meaning	To describe any restrictions to the usage of the data.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free text: There are no controlled terms for this element
Recommendations for Filling in the Entry	<p>Identify the most common use restriction. Like Access Constraints, more than one restriction may apply. Multiple use constraints can be separate documents that have information clarifying the use constraints or an URL to a document on use constraints.</p> <p>In some cases, the source data steward may have restrictions. For example, the source data steward may not allow their data be linked with other data that may result in identity of an individual. There may also be restrictions on using data in analysis and release of data to public. If the restriction standard is published in a public reference, then it should be mentioned here. Any licensing issues associated with use described. Add statements about inappropriate use.</p>
Examples	<p><i>For public health data:</i> Must read and fully comprehend metadata prior to data use. Acknowledgement of the Originator must be included when using the dataset as a source.</p> <p>Methods for collecting this data changed in 1990; therefore, data collected prior to 1990 should not be comparable to data collected after 1990.</p> <p><i>For environmental data:</i> Data only considered accurate to 5 meters. Data should not be used at scales greater than 1:24, 000.</p> <p><i>Other:</i> This data should not be used for any commercial gain or in support of commercial products (no implied endorsements), to direct or plan targeted advertising, etc. This data cannot be used to refute, contradict, or interfere with public health policy, programs, investigations, intervention actions, or health promotion activities.</p>

Elements

	<p>This dataset should not be linked with ___ data due to (privacy/national security/etc) concerns.</p> <p>This dataset links cancer data with drinking water data. It is inappropriate to use this data without understanding the limitations of the linkages made. Please consult the documentation at www.fake_website.com/fake_constraints.</p>
Additional Resources	None
Other Comments	None
Things to Note	None

Elements

Point of Contact (Composite Element)

Element Name	Point of Contact
Definition	Contact information for the organization that is knowledgeable about the dataset.
Purpose and Meaning	This is the contact information for the dataset owner. The actual information entered is described in the Contact section.
Obligation	Mandatory
Occurrence	Single
Date Type	Composite Element
Domain (Controlled Terms?)	None
Recommendations for Filling in the Entry	Refer to the Contact section elements.
Examples	None
Additional Resources	None
Other Comments	None
Things to Note	None

Security Classification System

Element Name	Security Classification System
Definition	Name of the security classification system used to classify this dataset.
Purpose and Meaning	Classified information is information that is sensitive in nature and is restricted for access purposes by law or regulation to classes of individuals who meet security clearance criteria. For the purposes of the Tracking Network, sensitive information is information that if misused could cause harm to subject individuals (the cases) or disrupt, hinder and prevent public health programs and activities.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms?)	<p>Free Text: Name of the Security Classification System.</p> <p>None: The system is not classified (Unclassified).</p> <p>Unknown: The system used is unknown</p> <p>The Security Subgroup will determine the security classification system. Therefore, these domains are preliminary.</p>
Recommendations for Filling in the Entry	Only datasets that contain information that can be used to identify the subject individuals (the cases) or could be used to contradict, disrupt, hinder or prevent public health actions or programs should be classified. If the dataset does not contain sensitive information, the dataset should not be classified. If the dataset does contain this information, the classification should use the Federal Information Security Oversight Office (ISOO)'s Classified National Security Information Directive Number 1, September 22, 2003.
Examples	Classified National Security Information Directive No 1. (32 CFR Parts 2001 and 2004, RIN 3095-AB18), September, 2003.
Additional Resources	<p>See: http://www.archives.gov/isoo/policy-documents/eo-12958-implementing-directive.html.</p> <p>see also: CIESIN's Guide to FGDC Compliant Metadata: 7.10 Metadata Security Information; http://sedac.ciesin.columbia.edu/metadata/guide/metadref.html</p> <p>see also: Dublin Core Metadata Initiative; http://dublincore.org/usage/meetings/2002/10/securityClassification.shtml</p>
Other Comments	None
Things to Note	None

Security Classification

Element Name	Security Classification
Definition	Name of the handling restriction on the dataset.
Purpose and Meaning	Provides the name of a security classification level that has a standard definition and associated levels of access authorization, data transmission requirements, data management requirements, and use constraints.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms?)	<p>Unclassified: Data are unrestricted and available to the public.</p> <p>Restricted: Available only to those who meet set criteria.</p> <p>Sensitive: This data is highly sensitive and not available on the Tracking Network portal.</p> <p>Confidential: National security information or material which requires protection and the unauthorized disclosure of which could reasonably be expected to cause damage to the national security.</p> <p>Secret: National security information or material which requires a substantial degree of protection and the unauthorized disclosure of which could reasonably be expected to cause serious damage to the national security</p> <p>Top Secret: National security information or material which requires the highest degree of protection and the unauthorized disclosure of which could reasonably be expected to cause exceptionally grave damage to the national security</p> <p>None: the system is not classified (Unclassified).</p> <p>The Security Subgroup will determine the security classification system. Therefore, these domains are preliminary.</p>
Recommendations for Filling in the Entry	For Tracking Network data, use either Restricted or Unclassified . If this element is not included, the data is assumed unclassified.
Examples	<i>For restricted datasets:</i> Restricted <i>For unclassified datasets:</i> Unclassified
Additional Resources	See: http://www.archives.gov/isoo/policy-documents/eo-12958-implementing-directive.html .

Elements

	<p>See also: CIESIN's Guide to FGDC Compliant Metadata: 7.10 Metadata Security Information; http://sedac.ciesin.columbia.edu/metadata/guide/metadref.html</p> <p>See also: Dublin Core Metadata Initiative; http://dublincore.org/usage/meetings/2002/10/securityClassification.shtml</p>
Other Comments	<p>If the dataset is classified Restricted, the <i>Security Handling Description</i> element will need to be completed. Access and use constraints will need to be specified in the <i>Access Constraints</i> element and the <i>Use Constraints</i> element to describe the criteria and requirements for the Restricted classification. Data not classified are assumed to be Unclassified.</p>
Things to Note	None

Security Handling Description

Element Name	Security Handling Description
Definition	Additional information about security restrictions
Purpose and Meaning	Provides a description of security requirements or restrictions imposed on both the distribution and use of the dataset. This information is supplemental to the Access Constraints and Use Constraints, and specifically addresses physical and data security requirements for the transmission, access, storage, and disposition of the dataset.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms?)	Free Text: Description of security requirements and restrictions. None: No security handling descriptions.
Recommendations for Filling in the Entry	This section should focus on the technology necessary for secure access, transmission, storage, and disposition of the dataset. The description may also include procedures for tracking and auditing data access and transactions; reporting data security breaches, unauthorized access, or security system failures; and procedures for destruction of the data.
Examples	This dataset must be maintained on a server or PC that is isolated from the Internet by a hardware-based firewall. This dataset must be encrypted before transmission.
Additional Resources	None
Other Comments	None
Things to Note	None

Native Data Set Environment

Element Name	Native Data Set Environment
Definition	Descriptions of the data set, including the name of the software, computer operating system, file name, and data set size.
Purpose and Meaning	The purpose of Native Data Set Environment is to provide basic information about a dataset's computer environment so a user can determine if they have the software and operating system to accommodate the dataset. This information can be useful to a metadata consumer for evaluating their computer capacity.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	None: Native data set environment known. Free Text
Recommendations for Filling in the Entry	Operating System and Version; Software and Version; File Name; File Size; Total Records in Dataset.
Examples	Microsoft Windows 2000; Microsoft Access 2003; BD3_MH_NCDM; Size: 524 kb; 3000 records. UNIX 03; Oracle 10g; CO_MH_NCDM; Size: 204 mb; 12000 records.
Additional Resources	None
Other Comments	Record the physical name of a dataset in the Resource Description element. Include the total number of records in the dataset.
Things to Note	None.

5.3 Section 2: Data Quality

Logical Consistency Report

Element Name	Logical Consistency Report
Definition	An explanation of the fidelity of relationships in the dataset and tests used.
Purpose and Meaning	<p>This element was developed to refer mostly to the geography of the data (i.e., polygons are closed and neat line-simplified, no duplicate features exist).</p> <p>The FGDC also states to use it for quality assurance, quality control information such as are the X values always between ‘0’ and ‘100’. Are all ‘Y’ values text format? Does value Z always equal the sum of values ‘R’ and ‘S’?</p>
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	<p>None: No logical consistency report needed.</p> <p>Free Text</p>
Recommendations for Filling in the Entry	<p>For the purposes of the Tracking Network, this should refer to a logical consistency report for the reference database(s) used to geo-reference a data set.</p> <p>This may also be the place to include any measures or tests of the sensitivity/specificity/accuracy of the geo-referencing process used and how those measures were determined. Example might include percentage of missing features, percentage of feature with missing feature values, percentage of mislabeled features or feature values, etc.</p> <p>NON-GEOGRAPHIC data (note: geo-referenced is not geographic) in the Network, measures of completeness and consistence should be in the “Completeness Report” element. This would apply to all health-outcome, bio-monitoring, environmental hazards, environmental monitoring, and population-based data.</p>
Examples	<p>This single precision coverage was built for points. There have been no edits to this coverage since the last build or clean.</p> <p>Point station locations verified using the 2002 Aerial Photography (1 ft).</p> <p>The data set was checked for topological consistency using the Arc/INFO commands BUILD and LABELERRORS. The NAWQA polygon attribute is no longer consecutive because of study units that have merged and study units deleted.</p>

Elements

	<p>Beginning with the 2005 data release, the spatial data are built according to the following logic: All geographic identifiers are assumed to be year 1990, if they match a year 1990 county FIPS code and the year 1990 Census tract identifier. Any identifiers that do not match year 1990 data are assumed year 2000 identifiers if they match a year 2000 designation for the corresponding type of geographic unit (Census tract). Any identifiers that do not match year 2000 or 1990 identifiers are assumed either to predate the 1990 data, or to be erroneous. The geographic components that are associated with these identifiers are not included in the spatial data. Some polygons overlap due to inconsistencies in the source geographic data; others overlap due to errors in the source data causing a given area to be included in multiple designations simultaneously.</p>
Additional Resources	None
Other Comments	Logical consistency can be performed by WAMS verification software (USGWS-NWI).
Things to Note	None

Completeness Report

Element Name	Completeness Report
Definition	Information about omissions, selection criteria, generalization, definitions used, and other rules used to derive the data set.
Purpose and Meaning	This element provides a location to describe the non-spatial aspects of data quality.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	None: No completeness report needed. Free Text
Recommendations for Filling in the Entry	NOTE the actual descriptions of processes are in the Process Description element. Use this section to provide measures of the process performance. Include a description of any data filtering applied (e.g., data only contains first diagnosis, first-admission, etc.) Include a description of source data incompleteness (e.g. percentage of records lacking a valid sex code). Include a description of geo-referencing accuracy. This is different that the completeness of the reference data set and should include a percentage of non-reference-able addresses and a description and ratio of data that was geo-referenced using less specific methods (e.g., to centroid). If descriptive statistics computed, describe the process used to compute those statistics. If unit conversions (e.g., Dx code to ICD10) applied, describe the capacity of the method used to compute the conversion.)
Examples	Data is available for sites in the ___ and ___ area. Missing data indicated by the code “999” in the appropriate fields. All wells measured in 1999, 2000, and 2001 are included. Public health providers located in the county of BIGCOUNTY in the state of Misoretah are not included in this dataset because the underlying site location data do not include these areas.
Additional Resources	None
Other Comments	None
Things to Note	None

Process Description

Element Name	Process Description
Definition	An explanation of the event and related parameters or tolerances.
Purpose and Meaning	The purpose of process description is to give an indication of how the dataset was created. It is useful in determining its fitness for purpose.
Obligation	Mandatory
Occurrence	Multiple
Date Type	Text
Domain (Controlled Terms)	None: No process description provided. Free Text
Recommendations for Filling in the Entry	<p>This is a repeating element. There should be at least one process description for a metadata record. Add additional process steps to show the history of process changes to the dataset. This element is closely tied to the Process Date element, which indicates the date of additional process step changes.</p> <p>Processes to consider for entry into this element are:</p> <ul style="list-style-type: none"> • Source material to describe where the data came from (source media type, domains, scales, acquisition, and quality control process) – Analytical Metadata • Process used to create the data including resolution of measurement, which includes information on: <ul style="list-style-type: none"> ○ Translation (data transaction from source to EPHTN; conversion units to standard units) ○ Geocoding and Geo-referencing (reference data, exception handling) ○ Aggregation ○ Computation (statistical summarization) • Methods for updating • Any quality assurance techniques
Examples	<p><i>Health Data:</i> Manually entered location of Rural Health Clinics from field collected data. The Misoretah Department of Health performed address geocoding. Points edited to the database by reference to digital color infrared photography, road, and street layer. Rural Health Clinics staffs for made field verification and edits where needed.</p> <p>The county, State, and national spreadsheets containing preterm infant delivery rates were loaded into Microsoft Excel. Values for "No Population" changed to -77777, values for "No events" changed to -88888, and values for "Insufficient data" changed to -99999. The FIPS code 12025 changed to 12086. Extra blank spaces that preceded the numbers deleted. The resulting files, one for counties, one for States, and one for the</p>

E l e m e n t s

	<p>nation saved to dBase IV files. Demographic group transposed the national statistics to list rates.</p> <p><i>Environmental Data:</i> The annual number of days that ozone levels exceeded EPA standards was summarized from the original database provided by the EPA. The original data source was for all monitoring station ozone data available in the State of Misoretah from 1996 to 2006. Each monitoring station had latitude and longitude assigned using EPA guidelines on spatial accuracy. The number of days that ozone levels exceeded EPA standards was summed for each station for each full year of data. For information on EPA ozone and ozone standards, please review the documents on the website: http://www.epa.gov/air/ozonepollution/.</p> <p>For additional information on the creation of this dataset, view the document at the following website: http://example.fake.website.com/fakereport.pdf.</p>
Additional Resources	None
Other Comments	None
Things to Note	None

Process Date

Element Name	Process Date
Definition	The date when the event was completed.
Purpose and Meaning	The purpose of process date is to provide the date of a process step. This date ties directly to the Process Description element.
Obligation	Mandatory
Occurrence	Multiple
Date Type	Date
Domain (Controlled Terms)	<p>Free Date: As complete a date as is available formatted as: YYYYMMDD, where YYYY is the four-digit year, MM is the numeric value (1 – 12) for the month, and DD is the numeric value (01 – 31) for the day. Leave off the DD or MMDD if the month or day not known.</p> <p>Unknown: The process date of this dataset is unknown</p>
Recommendations for Filling in the Entry	<p>Use the date of creation or modification of a process step. The first process date and its companion process description should be the date of the process if known, or the date of the creation of the metadata record that recorded the process.</p> <p>Subsequent process dates should reflect the date of changes to the process.</p> <p>The FGDC specifies that the date format is YYMMDD</p>
Examples	<p><i>Only year is known:</i> 2007</p> <p><i>Year and month (January) are known:</i> 200701</p> <p><i>Year month and day are known (January 10):</i> 20070110</p>
Additional Resources	None
Other Comments	None
Things to Note	None

5.4 Section 5: Entity and Attribute

Entity and Attribute Overview

Element Name	Entity and Attribute Overview
Definition	Detailed summary of the information contained in a data set.
Purpose and Meaning	An Entity is the table. An Attribute is the unique field (column) in a table. The purpose of this is to provide an overview of what is in a data set. What are the major types of data included in the data set, what are the key columns in the data set, were there codes used, and what important information about the data set you want the data consumer to know.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	None: No entity and attribute overview information provided. Free Text
Recommendations for Filling in the Entry	<p>The FGDC guidance states that the entity and attribute overview is appropriate when:</p> <ul style="list-style-type: none"> ○ Your data set is well documented in a data dictionary, data specification manual, or some other format, and you can provide a data consumer with a citation to this document, such as in a website link to the document. ○ You can adequately describe your data set in a short descriptive paragraph (or set of paragraphs within the EPHTN). The FGDC further suggests explaining here any unclear attribute labels and/or codes. <p>If it is desired to place detailed information on some specific columns of your data set you should include:</p> <ul style="list-style-type: none"> ○ The Attribute Label: the physical name of the column. ○ The Attribute Definition: the logical name and as much of a description as is needed to make it clear what the field contains. <p>Attribute Domain Values: should either include the attributes standardized vocabulary (such as codes and their meaning) or if the standardized vocabulary is publicly available online, the online documentation URL.</p>
Examples	<p><i>Population/Health Data:</i></p> <p>This data set is an aggregation table for Misoretah Cancers. The data set contains metrics (counts and statistics) aggregated by location, time, demographic, and diagnostic assignment fields. Assignment identities are County FIPS code, Year, 5-year age/sex groups, and SEER site codes. Vocabularies for FIPS codes are available at <u> </u>URL<u> </u>. The SEER site codes are available at <u> </u>URL<u> </u>. The available metrics are annual county count per year per age/sec group per diagnosis code</p>

Elements

	<p>and the standard error on the count. The computational methods for those metrics are available at ___URL___.</p> <p><i>Environmental Data:</i> This data set portrays Air monitoring sites that showed data exceeding a national standard, with information on location, land use of location, monitor type, monitor I.D., and what monitored.</p>
Additional Resources	None
Other Comments	This element is closely tied to the Entity and Attribute Detail Citation.
Things to Note	In the future, it may be possible to enter all information for each column of data in a data set. Within the FGDC standard there are the elements for going column by column and include definitions, code sets, and data types. For simplicity, the current version of the EPHTN profile does not included these options. Future versions may expand to include this option.

Entity and Attribute Detail Citation

Element Name	Entity and Attribute Detail Citation
Definition	Reference used to the complete description of the entity types, attributes, and attribute values for the data set.
Purpose and Meaning	The purpose of this is to provide a means to provide a web site to a document with a detailed description of the data set, including column descriptions, data types, codes used, or a formal citation that would provide a means to access such a document. Therefore, if a publicly available online data dictionary or other descriptive document exists for this dataset, reference that document's URL here.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	None: No entity and attribute detail citation provided. Free Text
Recommendations for Filling in the Entry	If a URL, please include the entire URL including the http or https section of the web address. In addition, provide some context on what the URL covers.
Examples	<p><i>Health Data:</i> The State of Misoretah's cancer registry uses the data standards described in the data dictionary of the North American Association of Central Cancer Registries. This data dictionary, as well as other support documents describing data quality and data standards can be found at: http://www.naacr.org/index.asp?Col_SectionKey=7&Col_ContentID=122</p> <p><i>Environmental Data:</i> The tables, columns, codes, and descriptions used by Misoretah Drinking Water Program are documented in the EPA, SDWIS/State data dictionary found at: https://iaspub.epa.gov/reports/rwservlet?edrreportpdf&19996</p> <p><i>Others:</i></p> <p>U.S. Department of Agriculture. 1975. Soil Taxonomy: A basic system of soil classification for making and interpreting soil surveys. Soil Conserv. Serv., U.S. Dep. Agric. Handb. 436.</p> <p>U.S. Department of Agriculture. 1992. Keys to Soil Taxonomy. SMSS Technical Monograph No. 19. Soil Surv. Staff, Soil Conserv. Serv.</p> <p>U.S. Department of Agriculture. 1993. National Soil Survey Handbook, title 430-VI. Soil Surv. Staff, Soil Conserv. Serv.</p>

Elements

Additional Resources	None
Other Comments	None
Things to Note	This is the location for providing citations to the structure and content of each column of data within a data set.

5.5 Section 6: Distribution

(The distribution section is optional can be repeating as many times as needed)

Distributor (Composite Element)

Element Name	Distributor
Definition	The party from whom the dataset may be obtained
Purpose and Meaning	This is the contact information for the organization(s) to contact to obtain the dataset. This may or may not be the same as the Point of Contact. See the Content section for a description of the actual information entered.
Obligation	Optional (Enter if Applicable)
Occurrence	Single
Date Type	Composite Element
Domain (Controlled Terms?)	None
Recommendations for Filling in the Entry	Refer to the Contact section elements.
Examples	None
Additional Resources	None
Other Comments	None
Things to Note	None

Resource Description

Element Name	Resource Description
Definition	The identifier by which the distributor knows the dataset.
Purpose and Meaning	Typically, a dataset may have a logical name and physical name. The complete logical name is given as the dataset title in the Title element. Record the physical name here.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms?)	Free Text: The complete physical name of the dataset.
Recommendations for Filling in the Entry	<p>This is the physical name and possible additional information such as the size of the file (32 Mb), and the file format (XML, SAS dataset, etc.). If this is a dynamically linked set of tables or files, you can provide the physical names of these tables and files as individual entries within the narrative.</p> <p>For an aggregation table of annual cancer case counts by SEER Site code by 5 year age/sex groups at the Census Block Group level, the physical table name might be</p>
Examples	<p>HO_CANCER_SITE_CBG_5AS</p> <p>FileOne.dbf; FileTwo.dbf; total size 32 Mb.</p> <p>XML file; 32 Mb.</p>
Additional Resources	None
Other Comments	None
Things to Note	None

Distribution Liability

Element Name	Distribution Liability
Definition	Statement of liability assumed by the distributor.
Purpose and Meaning	This is the distributor’s disclaimer statement.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms?)	Free Text: Description of the distribution liability. None: No Distribution Liability
Recommendations for Filling in the Entry	The distribution liability statement is a disclaimer for liability, reliability, damages and endorsements. Persons completing this element of metadata should check with their legal services to determine if the organization has a standard statement that meets the intent of this element.
Examples	<p>“In preparation of this data, every effort has been made to offer the most current, correct, complete and clearly expressed information possible. Nevertheless, some errors in the data may exist. In particular, but without limiting anything here, the ___ (agency) ___ disclaims any responsibility for source data, compilation and typographical errors and accuracy of the information that may be contained in this data. This data does not represent the official legal version of source documents or data used to compile this data. The ___ (agency) ___ further reserves the right to make changes to this data at any time without notice.</p> <p>Data compiled by the staff of the ___ (agency) ___ from a variety of source data, and are subject to change without notice. The ___ (agency) ___ makes no warranties or representations whatsoever regarding the quality, content, condition, functionality, performance, completeness, accuracy, compilation, fitness, or adequacy of the data.</p> <p>By using the data, you assume all risk associated with the acquisition, use, management, and disposition of data in your information system, including any risks to your computers, software or data being damaged by any virus, software, or any other file that might be transmitted or activated during the data exchange of this data. The ___(agency)___ shall not be liable, without limitation, for any direct, indirect, special, incidental, compensatory, or consequential damages, or third-party claims, resulting from the use or misuse of the acquired data, even if the ___(agency)___ has been advised of the possibility of such potential damages or loss.</p> <p>Format compatibility is the user’s responsibility.</p>

Elements

	<p>Reference herein to any specific commercial products, processes, services, or standards by trade name, trademark, manufacture, URL, or otherwise, does not necessarily constitute or imply its endorsement, recommendation or favoring by the ____ (agency) ____ . The view and opinions of the metadata compiler expressed herein do not necessarily state or reflect those of the ____ (agency) ____, or the data owners and shall not be used for advertising or product endorsement purposes.</p> <p>Use of the data with other data shall not terminate, void, or otherwise contradict this statement of liability.</p> <p>The sale or resale of the data, or any portions thereof, is prohibited unless with the express written permission of the ____ (agency or data stewards) ____ .</p> <p>“If errors or otherwise inappropriate information is brought to our attention, a reasonable effort will be made to fix or remove it. Such concerns should be addressed to the ____ (which contact point contained in the metadata) ____”</p>
Additional Resources	Contact your organizational legal support.
Other Comments	The example provided is a single statement that includes all components of the disclaimer. Component labels may also be used.
Things to Note	None

Custom Order Process

Element Name	Custom Order Process
Definition	Description of custom distribution services, and the terms and conditions for obtaining those services.
Purpose and Meaning	Use this element to provide users with instructions for ordering datasets that may not be directly available online (for example, datasets that require approval prior to access). This element describes the process for enrolling or setting up an account on the distributors secure data access module, completing a request for data, completing the review and approval process for that data request, and accessing the data through the process.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms?)	None: No custom ordering process. Free Text: Completely describe the custom order process.
Recommendations for Filling in the Entry	Pay close attention to needs like IRB approval and Trading Partner Agreements for ordering data.
Examples	This dataset requires prior approval by the national scientific advisory board. Please access the application and guidelines at ____.
Additional Resources	None
Other Comments	None
Things to Note	None

5.6 Section 7: Metadata Reference

Metadata Date

Element Name	Metadata Date
Definition	The date that the metadata were created or last updated.
Purpose and Meaning	The date that the metadata was created or last edited.
Obligation	Mandatory
Occurrence	Single
Date Type	Date
Domain (Controlled Terms)	Free Date
Recommendations for Filling in the Entry	Fill in date for year, month of year, and day of year. The recommended format for filling in date: YYYYMMDD.
Examples	<i>Year, month, and day (January 10):</i> 20070110
Additional Resources	None
Other Comments	None
Things to Note	None

Metadata Contact (Composite Element)

Element Name	Metadata Contact
Definition	The party responsible for the metadata information.
Purpose and Meaning	This is the contact information for the organization that created or maintains the metadata record. The Contact Section contains a description of the actual information entered.
Obligation	Mandatory
Occurrence	Single
Date Type	Composite Element
Domain (Controlled Terms?)	None
Recommendations for Filling in the Entry	Refer to the Contact section elements.
Examples	None
Additional Resources	None
Other Comments	None
Things to Note	None

Elements

Metadata Standard Name

Element Name	Metadata Standard Name
Definition	The name of the metadata standard used to document the data set.
Purpose and Meaning	The purpose of this is to provide the name of the standard used to create the metadata record. The user needs to know the standard to assess the information contained within.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	FGDC Content Standards for Digital Geospatial Metadata Free Text
Recommendations for Filling in the Entry	Recommend using “FGDC Content Standards for Digital Geospatial Metadata.”
Examples	FGDC Content Standards for Digital Geospatial Metadata
Additional Resources	The FGDC Content Standards can be found at the following website: http://www.fgdc.gov/standards/standards_publications/index.html
Other Comments	None
Things to Note	None

Metadata Standard Version

Element Name	Metadata Standard Version
Definition	Identification of the version of the metadata standard used to document the data set.
Purpose and Meaning	The purpose of this is to provide the name of the standard version used to create the metadata record.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	EPHT Metadata Profile Version 1.2 Free Text
Recommendations for Filling in the Entry	Recommend using “EPHT Metadata Profile Version 1.2”
Examples	EPHT Metadata Profile Version 1.2
Additional Resources	None
Other Comments	None
Things to Note	This element was inadvertently left out of the EPHT Metadata Creation Tool (MCT) and the EPHT Metadata Profile. Future releases will include this element. Until then, use Metadata Standard Name in the MCT.

Metadata Access Constraints

Element Name	Metadata Access Constraints
Definition	Restrictions and legal prerequisites for accessing the metadata. These include any access constraints applied to assure the protection of privacy or intellectual property, and any special restrictions or limitations on obtaining the metadata.
Purpose and Meaning	Provides information on restrictions and legal prerequisites for accessing the metadata (not the data).
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	<p>None: No restrictions on the viewing of a metadata document.</p> <p>Authorized User: For very specific reasons, the metadata record is restricted for viewing only by persons authorized via role-based security.</p> <p>Restricted: For very specific reasons, the metadata record is restricted to only internal viewing of the data owners only.</p> <p>The metadata access constraints will be determined in the future by the Metadata and Security subgroups. Therefore, these domains are preliminary.</p>
Recommendations for Filling in the Entry	The FGDC guidance states that with the exception of classified information and intellectual properties, the response is usually 'none'. Even if a data set is exempted from public record laws (endangered species locations, personal health data, etc.) the metadata is typically fully accessible.
Examples	<p>None</p> <p>Authorized User</p> <p>Restricted</p>
Additional Resources	None
Other Comments	The Metadata Subgroup would like to reiterate their continued support for open and unrestricted access to metadata provided to the Tracking Network.
Things to Note	None

Metadata Use Constraints

Element Name	Metadata Use Constraints
Definition	Restrictions and legal prerequisites for using the metadata after access are granted. These include any metadata use constraints applied to assure the protection of privacy or intellectual property, and any special restrictions or limitations on using the metadata.
Purpose and Meaning	Restrictions and legal prerequisites for using the metadata (not the data) after access granted.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	None: No metadata use constraints Free Text
Recommendations for Filling in the Entry	<p>The FGDC states that this may be applicable for the protection of privacy or intellectual properties. Note that although a dataset may be exempt from public access, the metadata seldom contains any protected information such as the location of an endangered species nesting site or the address of an AIDS patient.</p> <p>If there are no metadata use constraints, then this element should use “None”. If there are use constraints, explicitly state them. This statement should include the reason why the metadata document use is constrained and the actual constraint on the metadata record.</p>
Examples	<p>This metadata document is restricted to internal partners only due to ...</p> <p>None</p>
Additional Resources	None
Other Comments	The Metadata Subgroup would like to reiterate their continued support for open and unrestricted access to metadata provided to the Tracking Network.
Things to Note	None

5.7 Section 9: Time Period

Calendar Date

Element Name	Calendar Date
Definition	The year (optionally month or month and day).
Purpose and Meaning	Provides a means of entering a single date that communicates a single date for a pertinent characteristic of the dataset (e.g. the time period represented by the data in the dataset).
Obligation	Mandatory
Occurrence	Single
Date Type	Date
Domain (Controlled Terms)	<p>Unknown: The beginning date for the date range is unknown at this time</p> <p>Unpublished Material: The metadata references a dataset that is pending or in progress.</p> <p>Free Date</p>
Recommendations for Filling in the Entry	Fill in dates for year, month of year, or day of year. The recommended formats for filling in dates are: YYYY for years; YYYYMM for month of year; YYYYMMDD for day of the year
Examples	<p>The time period of coverage for a statewide childhood blood lead surveillance system that operated from 1990 to 2005:</p> <p>If only year is known - <i>1990</i></p> <p>If only month and year are known - <i>199006</i></p> <p>If day, month and year are known – <i>19900601</i></p>
Additional Resources	<p>FGDC Graphical Representation – Beginning Date</p> <p>http://www.fgdc.gov/csdgmgraphical/ideninfo/timepd/timeinfo/rnge/beginind.htm</p>
Other Comments	The Calendar Date is one component of the Time Period metadata element. The Time Period element provides information on how to fill out dates and times in the sections of the metadata where this information is required. As such, the Time Period element (including Calendar Date) is not a stand-alone element, but rather a guideline for sections requiring a specific temporal reference.
Things to Note	If this is a range of date and not a single date, then use Beginning Date and Ending Date elements.

Beginning Date

Element Name	Beginning Date
Definition	The first year (optionally month or month and day) of the event.
Purpose and Meaning	Provides a means of entering starting date information that communicates a date <i>range</i> (starting and ending dates) for a pertinent characteristic of the dataset (e.g. the time period represented by the data in the dataset). Used in conjunction with Ending Date to specify the time period of the characteristic specified.
Obligation	Mandatory
Occurrence	Single
Date Type	Date
Domain (Controlled Terms)	Unknown: The beginning date for the date range is unknown at this time Unpublished Material: The metadata references a dataset that is pending or in progress. Free Date
Recommendations for Filling in the Entry	Fill in dates for year, month of year, or day of year. The recommended formats for filling in dates are: YYYY for years; YYYYMM for month of year; YYYYMMDD for day of the year
Examples	The time period of coverage for a statewide childhood blood lead surveillance system that operated from 1990 to 2005: If only year is known: <i>1990</i> If only month and year are known: - <i>199006</i> If day, month and year are known: <i>19900601</i>
Additional Resources	FGDC Graphical Representation – Beginning Date http://www.fgdc.gov/csdgmgraphical/ideninfo/timepd/timeinfo/rnge/begind.htm
Other Comments	The Beginning Date is one component of the Time Period metadata element. The Time Period element provides information on how to fill out dates and times in the sections of the metadata where this information is required. As such, the Time Period element (including Beginning Date) is not a stand-alone element, but rather a guideline for sections requiring a specific temporal reference.
Things to Note	If this is for a single date then use the Calendar Date element.

Ending Date

Element Name	Ending Date
Definition	The last year (and optionally month, or month and day) for the event.
Purpose and Meaning	Provides a means of entering ending date information that communicates a date <i>range</i> (starting and ending dates) for a pertinent characteristic of the dataset (e.g. the time period represented by the data in the dataset). Used in conjunction with Beginning Date to specify the time period of the characteristic specified.
Obligation	Mandatory
Occurrence	Single
Date Type	Date
Domain (Controlled Terms)	<p>Unknown: The ending date for the date range is unknown at this time</p> <p>Present: Indicates that the dataset is currently being compiled. Replace with an actual date once on-going data collection is terminated.</p> <p>Free date</p>
Recommendations for Filling in the Entry	Fill in dates for year, month of year, or day of year. The recommended formats for filling in dates are: YYYY for years; YYYYMM for month of year; YYYYMMDD for day of the year
Examples	<p>The time period of coverage for a statewide childhood blood lead surveillance system that continues to operate: <i>Present</i></p> <p>The time period of coverage for a statewide childhood blood lead surveillance system that operated from 1990 to 2005 and has discontinued on-going data collection:</p> <p>If only year is known: <i>2005</i></p> <p>If only month and year are known: <i>200506</i></p> <p>If day, month and year are known: <i>20050601</i></p>
Additional Resources	FGDC Graphical Representation – Ending Date http://www.fgdc.gov/csdgmgraphical/ideninfo/timepd/timeinfo/rnge/endingd.htm
Other Comments	The Ending Date is one component of the Time Period metadata element. The Time Period element provides information on how to fill out dates and times in the sections of the metadata where this type of information is required. As such, the Time Period element (including Ending Date) is not a stand-alone element, but rather a guideline for sections requiring a specific temporal reference.
Things to Note	If this is for a single date then use the Calendar Date element.

Time of Day

Element Name	Time of Day
Definition	The hour (and optionally minute, or minute and second) of day.
Purpose and Meaning	Provides a means of entering starting time information that communicates a single time for a pertinent characteristic of the dataset (e.g. the time of day that an air-monitor takes samples at a single time of day).
Obligation	Optional
Occurrence	Single
Date Type	Time
Domain (Controlled Terms)	Unknown: The beginning date for the date range is unknown at this time Free time
Recommendations for Filling in the Entry	<p><i>Local Time:</i> values shall follow the 24-hour timekeeping system for local time of day in the hours, minutes, seconds, and decimal fractions of a second (to the precision desired) without separators convention: HHMMSSSS</p> <p><i>Local Time with Time Differential Factor:</i> recording time in local time and the relationship to Universal Time (Greenwich Mean Time), values shall follow the 24-hour timekeeping system for local time of day in hours, minutes, seconds, and decimal fractions of a second (to the resolution desired) without separators convention. This value shall be followed, without separators, by the time differential factor. The time differential factor expresses the difference in hours and minutes between local time and Universal Time. It is represented by a four-digit number preceded by a plus sign (+) or minus sign (-), indicating the hours and minutes the local time is ahead of or behind Universal Time, respectively. The general form is HHMMSSSSshhmm, where HHMMSSSS is the local time using 24-hour timekeeping (expressed to the precision desired), 's' is the plus or minus sign for the time differential factor, and hhmm is the time differential factor. This option allows producers to record local time and time zone information.</p> <p><i>Universal Time (Greenwich Mean Time):</i> recording time in Universal Time (Greenwich Mean Time), values shall follow the 24-hour timekeeping system for Universal Time of day in hours, minutes, seconds, and decimal fractions of a second (expressed to the precision desired) without separators convention, with the upper case letter "Z" directly following the low-order (or extreme right hand) time element of the 24-hour clock time expression. The general form is HHMMSSSSZ, where HHMMSSSS is Universal Time using 24-hour timekeeping, and Z is the letter "Z".</p>
Examples	Intermittent air monitor takes a single sample at 4:12:34 am EST

Elements

	(shown in local time): Hours only: 04 Hours and minutes: 0412 Hours and minutes and seconds: 041234
Additional Resources	FGDC Graphical Representation – Beginning Time http://www.fgdc.gov/csdgmgraphical/ideninfo/timepd/timeinfo/rnge/begint.htm
Other Comments	The Time of Day is one component of the Time Period metadata element. The Time Period element provides information on how to fill out dates and times in the sections of the metadata where this information is required. As such, the Time Period element (including Time of Day) is not a stand-alone element, but rather a guideline for sections requiring a specific temporal reference.
Things to Note	If this is a range of date and not a single date, then use Beginning Time and Ending Time elements that are part of the Beginning Date and Ending Date elements.

Beginning Time

Element Name	Beginning Time
Definition	The first hour (and optionally minute, or minute and second) of the day for the event.
Purpose and Meaning	Provides a means of entering starting time information that communicates a time <i>range</i> (starting and ending times) for a pertinent characteristic of the dataset (e.g. the time of day that an air-monitor taking intermittent samples took its first sample). Used in conjunction with Ending Time to specify the time period of the characteristic specified.
Obligation	Optional
Occurrence	Single
Date Type	Time
Domain (Controlled Terms)	Unknown: The beginning date for the date range is unknown at this time Free time
Recommendations for Filling in the Entry	<p><i>Local Time:</i> values shall follow the 24-hour timekeeping system for local time of day in the hours, minutes, seconds, and decimal fractions of a second (to the precision desired) without separators convention: HHMMSSSS</p> <p><i>Local Time with Time Differential Factor:</i> recording time in local time and the relationship to Universal Time (Greenwich Mean Time), values shall follow the 24-hour timekeeping system for local time of day in hours, minutes, seconds, and decimal fractions of a second (to the resolution desired) without separators convention. This value shall be followed, without separators, by the time differential factor. The time differential factor expresses the difference in hours and minutes between local time and Universal Time. It is represented by a four-digit number preceded by a plus sign (+) or minus sign (-), indicating the hours and minutes the local time is ahead of or behind Universal Time, respectively. The general form is HHMMSSSSshhmm, where HHMMSSSS is the local time using 24-hour timekeeping (expressed to the precision desired), 's' is the plus or minus sign for the time differential factor, and hhmm is the time differential factor. This option allows producers to record local time and time zone information.</p> <p><i>Universal Time (Greenwich Mean Time):</i> recording time in Universal Time (Greenwich Mean Time), values shall follow the 24-hour timekeeping system for Universal Time of day in hours, minutes, seconds, and decimal fractions of a second (expressed to the precision desired) without separators convention, with the upper case letter "Z" directly following the low-order (or extreme right hand) time element of the 24-hour clock time expression. The general form is HHMMSSSSZ, where HHMMSSSS is Universal Time using 24-hour timekeeping, and Z is the letter "Z".</p>

Elements

Examples	Intermittent air monitor took its first sample of the day at 4:12:34 am EST (shown in local time): Hours only: 04 Hours and minutes: 0412 Hours and minutes and seconds: 041234
Additional Resources	FGDC Graphical Representation – Beginning Time http://www.fgdc.gov/csdgmgraphical/ideninfo/timepd/timeinfo/rnge/begint.htm
Other Comments	The Beginning Time is one component of the Time Period metadata element. The Time Period element provides information on how to fill out dates and times in the sections of the metadata where this information is required. As such, the Time Period element (including Beginning Time) is not a stand-alone element, but rather a guideline for sections requiring a specific temporal reference.
Things to Note	If this is for a single date then use the Time of Day element that is part of the Calendar Date element.

Ending Time

Element Name	Ending Time
Definition	The last hour (and optionally minute, or minute and second) of the day for the event.
Purpose and Meaning	Provides a means of entering ending time information for a data element that communicates a time <i>range</i> (starting and ending times) for a pertinent characteristic of the dataset (e.g. the time of day that an air-monitor taking intermittent samples took its last sample). Used in conjunction with Beginning Time to specify the time period of the characteristic specified.
Obligation	Optional
Occurrence	Multiple
Date Type	Time
Domain (Controlled Terms?)	Unknown – The beginning date for the date range is unknown at this time Free time
Recommendations for Filling in the Entry	<p>-Local Time - values shall follow the 24-hour timekeeping system for local time of day in the hours, minutes, seconds, and decimal fractions of a second (to the precision desired) without separators convention: HHMMSSSS</p> <p>-Local Time with Time Differential Factor. Recording time in local time and the relationship to Universal Time (Greenwich Mean Time), values shall follow the 24-hour timekeeping system for local time of day in hours, minutes, seconds, and decimal fractions of a second (to the resolution desired) without separators convention. This value shall be followed, without separators, by the time differential factor. The time differential factor expresses the difference in hours and minutes between local time and Universal Time. It is represented by a four-digit number preceded by a plus sign (+) or minus sign (-), indicating the hours and minutes the local time is ahead of or behind Universal Time, respectively. The general form is HHMMSSSSshhmm, where HHMMSSSS is the local time using 24-hour timekeeping (expressed to the precision desired), 's' is the plus or minus sign for the time differential factor, and hhmm is the time differential factor. This option allows producers to record local time and time zone information.</p> <p>-Universal Time (Greenwich Mean Time). Recording time in Universal Time (Greenwich Mean Time), values shall follow the 24-hour timekeeping system for Universal Time of day in hours, minutes, seconds, and decimal fractions of a second (expressed to the precision desired) without separators convention, with the upper case letter "Z" directly following the low-order (or extreme right hand) time element of the 24-hour clock time expression. The general form is HHMMSSSSZ, where HHMMSSSS is Universal Time using 24-hour timekeeping, and Z is the letter "Z".</p>

Elements

Examples	Intermittent air monitor took its last sample of the day at 11:48:02 pm EST (shown in local time): Hours only: 23 Hours and minutes: 2348 Hours and minutes and seconds: 234802
Additional Resources	FGDC Graphical Representation – Ending Time http://www.fgdc.gov/csdgmgraphical/ideninfo/timepd/timeinfo/rnge/endingt.htm
Other Comments	The Ending Time is one component of the Time Period metadata element. The Time Period element provides information on how to fill out dates and times in the sections of the metadata where this information is required. As such, the Time Period element (including Ending Time) is not a stand-alone element, but rather a guideline for sections requiring a specific temporal reference.
Things to Note	None

Section 10: Contact

Contact Organization

Element Name	Contact Organization
Definition	The name of the organization(s) that developed the data set.
Purpose and Meaning	Provides the full name of the organization that is associated with the development of the dataset. Used in cases where the association of the organization to the dataset is more significant than the association of the person to the dataset. In the case of organizations where there is clearly a hierarchy present, list the parts of the hierarchy from largest to smallest, separated by full stops and a space.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element. User can write any information that is in accordance to their organization's naming policy.
Recommendations for Filling in the Entry	Recommend including the complete name of the organization.
Examples	New York State Department of Health. Bureau of Environmental and Occupational Epidemiology
Additional Resources	None
Other Comments	None
Things to Note	None

Contact Person

Element Name	Contact Person
Definition	The name of the individual to which the contact applies.
Purpose and Meaning	Provides the full name of the individual that is associated with the development of the dataset.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element. User can write any information that is in accordance to their organization's naming policy.
Recommendations for Filling in the Entry	While included as part of the EPHT Metadata Profile, the Tracking Network will only use the Organization (corporate) contact because personal contact information within federal systems is Information in Identifiable Form (IIF) and is subject to additional security procedures.
Examples	Firstname Lastname
Additional Resources	None
Other Comments	None
Things to Note	None

Elements

Contact Position

Element Name	Contact Position
Definition	The title of the individual (if applicable) who is the representative of the organization(s) that developed the dataset.
Purpose and Meaning	Provides the full position title of the individual who represents the organization that developed the dataset.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element. User can write any information that is in accordance to their organization's position title policy.
Recommendations for Filling in the Entry	Recommend including the complete position title of the individual named as contact person for the dataset.
Examples	Program Research Specialist –III GIS Analyst
Additional Resources	None
Other Comments	None
Things to Note	None

Address Type

Element Name	Address Type
Definition	Type of address of the organization(s) that developed the dataset.
Purpose and Meaning	To identify if the address provided in the “contact address” section is mailing or physical or mailing and physical address of the organization(s) that developed the dataset.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	<p>Mailing: The address is only used for mail delivery such as PO Box addresses.</p> <p>Physical: The address is of actual office location of the organization(s) that developed the dataset and there is a separate/different address for receiving the mail.</p> <p>Mailing and Physical: The address is used both for receiving the mail and is actual office location of the organization(s) that developed the dataset.</p> <p>Free Text: User can write any other information if the prior three domains do not describe their address type adequately.</p>
Recommendations for Filling in the Entry	None
Examples	<p>If the address provided in metadata is State Department of Health, PO Box 100, Albany, NY 12345; then select “mailing”</p> <p>If the address provided in metadata is State Department of Health, Room 99, 100 State Street, Albany, NY 12345 and mailing address is not the same, then select “physical”.</p> <p>If the same address receives mail (State Department of Health, Room 99, 100 State Street, Albany, NY 12345) then select “mailing and physical”.</p>
Additional Resources	None
Other Comments	None
Things to Note	None

Address

Element Name	Address
Definition	Contact address for organization that developed the dataset.
Purpose and Meaning	To provide the physical and/or mailing address of a contact.
Obligation	Mandatory
Occurrence	Single.
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element. User can write any information that is in accordance to their address type mentioned in “address type” field.
Recommendations for Filling in the Entry	It is recommended to include the street number and name (pre-directional, suffix, and post-directional as appropriate), post office box number, rural or highway contract route and box number), and secondary descriptor and number (e.g., suite or room number, floor) if needed.
Examples	90 State St. W. has four address components -- the street number “90”; the street name “State”; the street type “St.”; and the street direction “W.”
Additional Resources	http://www.usps.com/ncsc/lookups/usps_abbreviations.html#suffix Official United States Postal Service street suffixes http://www.usps.com/ncsc/lookups/usps_abbreviations.html#secunitdesig Official United States Postal Service secondary unit designators
Other Comments	None
Things to Note	None

Elements

City

Element Name	City
Definition	City name for contact address of the organization(s) that developed the dataset.
Purpose and Meaning	Provides the name of city where the organization that developed the dataset wants to accept physical mail.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element. User can write any information that is in accordance to their address type mentioned in “address type” field.
Recommendations for Filling in the Entry	Recommend including the complete city name.
Examples	New York City
Additional Resources	None
Other Comments	None
Things to Note	None

Elements

State or Province

Element Name	State or Province
Definition	State or province for contact address of the organization(s) that developed the dataset.
Purpose and Meaning	Provides the name of state/province where the organization that developed the dataset wants to accept physical mail.
Obligation	Mandatory
Occurrence	Single.
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element. User can write any information that is in accordance to their address type mentioned in “address type” field. Use the full state name or abbreviated name of state/province.
Recommendations for Filling in the Entry	Recommend including the complete state/province name.
Examples	New York or NY; Ontario or ON
Additional Resources	http://www.usps.com/ncsc/lookups/usps_abbreviations.html#states Official United States Postal Service abbreviations
Other Comments	None
Things to Note	None

Postal Code

Element Name	Postal Code
Definition	ZIP Code or Postal Code for contact address of the organization(s) that developed the dataset.
Purpose and Meaning	Provides the ZIP Code or Postal Code for contact address of the organization that developed the dataset.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element. User can write any information that is in accordance to their address type mentioned in “address type” field. It can be either five digits ZIP Code or ZIP+4 format for address in the United States. For Canada, it is six character alphanumeric Postal Code.
Recommendations for Filling in the Entry	None
Examples	12180 12180-2659 P8N 4G8
Additional Resources	http://zip4.usps.com/zip4/welcome.jsp Official United States Postal Service ZIP Code lookup. The site provides ZIP Code based on address or city or company name. http://www.canadapost.ca/Default.aspx Official Canada Post’s Postal Code lookup. The site provides quick search, advance search, rural address and P. O. Box search, reverse search and list of municipalities.
Other Comments	None
Things to Note	None

Elements

Country

Element Name	Country
Definition	Name of Country for contact address of the organization(s) that developed the dataset.
Purpose and Meaning	Provides the name of Country where the organization that developed the dataset wants to accept physical mail.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element. User can write any information that is in accordance to their address type mentioned in “address type” field.
Recommendations for Filling in the Entry	None
Examples	USA Canada
Additional Resources	None
Other Comments	None
Things to Note	None

Contact Telephone Number

Element Name	Contact Telephone Number
Definition	Contact voice telephone number of the organization(s) that developed the dataset.
Purpose and Meaning	Provides the contact telephone number by which dataset user can speak to an individual to find more information or answer to any question related to the dataset.
Obligation	Mandatory
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	Recommend including the country code, area code, and telephone number.
Examples	1 518 402 7990
Additional Resources	None
Other Comments	None
Things to Note	None

Elements

Contact TDD/TTY Telephone

Element Name	Contact TDD/TTY Telephone
Definition	Contact telephone number by which hearing-impaired individuals can contact the organization(s) that developed the dataset.
Purpose and Meaning	Provides the contact telephone number by which hearing-impaired dataset user can communicate with an individual to find more information or answer to any question related to the dataset.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	Recommend including the complete country code, area code, and telephone number.
Examples	1 518 402 7960
Additional Resources	None
Other Comments	None
Things to Note	None

Elements

Contact FAX Number

Element Name	Contact FAX Number
Definition	Contact telephone number of a facsimile machine of the organization.
Purpose and Meaning	Provides the contact telephone number of a facsimile machine by which data user can contact the organization(s) that developed the dataset.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	Recommend including the complete country code, area code, and telephone number.
Examples	1 518 402 7959
Additional Resources	None
Other Comments	None
Things to Note	None

Elements

Contact E-mail Address

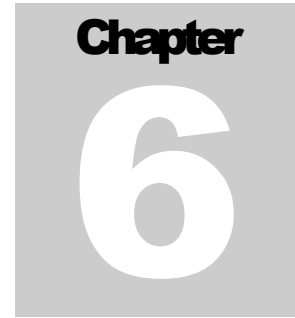
Element Name	Contact E-mail Address
Definition	Contact electronic mailbox address of the organization.
Purpose and Meaning	Provides the contact electronic mailbox address that data user can contact the organization(s) that developed the dataset.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	None
Examples	BEOEGIS@health.state.ny.us
Additional Resources	None
Other Comments	None
Things to Note	None

Hours of Service

Element Name	Hours of Service
Definition	Time period when individuals can speak to the organization.
Purpose and Meaning	Provides the information about days and time period when data user can speak to the organization(s) that developed the dataset.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	Recommend including the days, time, and time zone information.
Examples	Monday to Friday between 1:00 PM to 3:00 PM (Eastern Standard Time) Monday and Wednesday Between 9:00 AM to 11:00 AM EST
Additional Resources	None
Other Comments	None
Things to Note	None

Contact Instructions

Element Name	Contact Instructions
Definition	Supplemental instructions on how or when to contact the organization listed under the contact address.
Purpose and Meaning	Provides the information about how or when the data users can contact the organization(s) that developed the dataset.
Obligation	Optional
Occurrence	Single
Date Type	Text
Domain (Controlled Terms)	Free Text: There are no controlled terms for this element.
Recommendations for Filling in the Entry	Recommend including the detailed instructions, if any, for the users to follow before contacting the organization that developed the dataset.
Examples	<p>Contact data center</p> <p>Send any request to the agency by e-mail at the address listed under contact e-mail.</p> <p>For questions related to the data set access please contact the data center by e-mail at the address listed under contact e-mail and for all other data quality related questions please contact the Data Center by calling the number listed under contact telephone number between the service hours listed above.</p> <p>You can also send your questions /comments by fax at the number listed under contact fax number.</p>
Additional Resources	None
Other Comments	None
Things to Note	None



Chapter 6: Examples of Metadata Records

6.1 *Cancer Dataset*

Misoretah Cancer Counts by Site, Year, County, and Age-Sex Group

Theme keywords: Human, Health, Cancer

Abstract: This data set contains annual cancer case counts for primary cancers occurring among Misoretah residents aggregated by major cancer sites, by five year age-sex groupings, for each county in the State of Misoretah from 1973 through 2004. The cancer data was obtained from the Misoretah Cancer Registry and aggregated by the Misoretah Environmental Public Health Tracking Network. Population data obtained from commercially available census data and estimated by linear regression for intercensal years.

EPHT Metadata:

- [Identification Information](#)
- [Data Quality Information](#)
- [Entity and Attribute Information](#)
- [Distribution Information](#)
- [Metadata Reference Information](#)

Identification Information:

Citation:

Citation information:

Originators: The Misoretah Environmental Public Health Tracking Program (comp.), Environmental Epidemiology Program, Bureau of Epidemiology, Misoretah Department of Health

Title:

Misoretah Cancer Counts by Site, Year, County, and Age-Sex Group

Publication date: 200612

Online linkage:

Description:**Abstract:**

This data set contains annual cancer case counts for primary cancers occurring among Misoretah residents aggregated by major cancer sites, by five year age-sex groupings, for each county in the State of Misoretah from 1973 through 2004. The cancer data obtained from the Misoretah Cancer Registry and aggregated by the Misoretah Environmental Public Health Tracking Network. Population data obtained from commercially available census data and estimated by linear regression for intercensal years.

Purpose:

This data provides public health researchers, professionals, and the public with summary information about the rates of cancer by major site classifications.

Supplemental information:

The Misoretah Cancer Registry (MCR) site codes used as the major site aggregation code. Those codes are a modification of the National Cancer Institute (NCI): Surveillance, Epidemiology and End Results (SEER) program codes. Further information about the MCR found at <http://uuhsc.Misoretah.edu/MCR/>. Only primary diagnosis cases are included. Cases of secondary or subsequent cancers were excluded. The MCR provide the Misoretah EPHTN with annual updates after the MCR has validated the quality of the data and submitted to the SEER. The Misoretah EPHTN processes the data for geo-referencing and aggregation codes, before publishing a new cumulative aggregation data set. Records with counts less than 10 were masked.

The MCR serves as the official repository for statewide cancer data per the Misoretah Cancer Reporting Rule, R384-100, and a memorandum of Agreement between the Misoretah Department of Health and the University of Misoretah. The MCR operates as one of several population-based cancer registries under contract to the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute. The MCR follows the SEER data standards to provide high quality information on time trends in cancer incidence and survival rates for the nation. The MCR collects complete, timely and accurate cancer incidence, treatment and survival data for all SEER-reportable cancer cases in Misoretah.

The Misoretah Environmental Public Health Tracking Network (MEPHTN) obtains data from the MCR. The MEPHTN processed MCR data to create this dataset. In creating this dataset, the MEPHTN implemented MCR aggregation and data use requirements.

Time period of content:**Time period information:****Range of dates/times:**

Beginning date: 197301

Ending date: 200412

Currentness reference:

Publication Date

Status:

Progress: Complete

Maintenance and update frequency: Annually

Spatial domain:

Bounding coordinates:

West bounding coordinate: -114.042925

East bounding coordinate: -109.041501

North bounding coordinate: 42.001718

South bounding coordinate: 36.997693

Keywords:

Theme:

Theme keywords: Human, Health, Cancer

Theme keyword thesaurus: ISO 19115 Topic Category Thesaurus

Place:

Place keywords: Misoretah, MZ, 49

Place keyword thesaurus: Geographic Names Information (GNIS)

<http://geonames.usgs.gov/domestic/index.html>

Place keyword thesaurus: Federal Information Processing Standards (FIPS)

<http://www.itl.nist.gov/fipspubs/by-num.htm> FIPS PUB 5-2 Codes for the Identification of the States, The District of Columbia and the Outlying Areas of the United States, and Associated Areas.

<http://www.itl.nist.gov/fipspubs/fip5-2.htm>

Access constraints: This data is publicly available.

Use constraints:

NO-USE: This data may not be used in any way to imply MCR or Misoretah Department of Health (MDOH) endorsement of any research objective, commercial or for-profit venture or to advertise or support a commercial product, or to direct or plan targeted advertising.

This data may not be used to refute, contradict or interfere with public health policy, programs, investigations, intervention actions or health promotion activities conducted by the MCR or its agencies or any Misoretah State government agency or any local government public health agency in Misoretah.

This data may not be used to identify subjects of cancer case information or the individual or organization who reported the cancer case information.

PUBLICATION: The data user will comply with Misoretah Cancer Registry (MCR) rules for publication or presentation of this data or any results derived from this data. Publication approval of any manuscript or document must be accomplished prior to submission for publication. Data users will provide a copy of any publication draft or public presentation of this data or results derived from this data to the Misoretah Environmental Public Health Tracking Network (MEPHTN) that will coordinate MEPHTN and MCR approval to publish or present. See contact information in this metadata. The MCR requires 30 days to approve draft publications. The MCR will provide a response in writing to the data user.

RIGHT TO REFUSAL: The MCR and/or the MEPHTN retain the right to refusal for any publication or public presentation of the data or results derived from the data.

ACKNOWLEDGEMENT: Use of this data requires acknowledgement of the Misoretah Cancer Registry (MCR) and the Misoretah Environmental Public Health Tracking Network (MEPHTN) in any publications or public presentations of the data or results derived from the data.

Acknowledgement must be made that the research was supported by the Misoretah Cancer Registry, which is funded by Contract Number N01-PC-35141 from the National Cancer Institute with additional support from the Misoretah Department of Health and the University of Misoretah. Acknowledgement must be made that the research was supported by the Misoretah Environmental

EXAMPLES

Public Health Tracking Network, which is partially funded by the Centers for Disease Control and Prevention.

AUTHORSHIP: Authorship is required when either the MCR or the MEPHTN makes substantial contribution to the data.

AUDITS: The MCR and/or the MEPHTN retain the right to conduct on-site audits of the researcher with or without cause. Audits will be conducted after notification and during normal business hours by representatives of the MCR or MEPHTN. The audit will observe research practices for protecting data.

REPORTS: Data users must submit annual and final reports regarding the progress and or completion of research projects to the MCR.

Point of contact:

Contact information:

Contact organization primary:

Contact organization: The Misoretah Environmental Public Health Tracking Program

Contact position: Manager, Environmental Epidemiology Program

Contact address:

Address type: mailing and physical address

Address:

1234 Anyplace Street

City: Somewhere City

State or province: Misoretah

Postal code: 97531

Country: USA

Contact voice telephone: 123.456-7890

Contact TDD/TTY telephone:

Contact facsimile telephone: 123.456-0987

Contact electronic mail address: MEPHT@Misoretah.gov

Hours of service: 9:00 AM - 4:00 PM, Pacific Mountain Time

Contact instructions:

Security information:

Security classification system: None

Security classification: Unclassified

Security handling description: This data may be freely distributed. However, the use constraints apply to all recipients of this data.

Native data set environment:

LINUX; Oracle 10e; HO_CANCER_CNTY_YR_5AS_SITE; 35 mb; 100000 records.

[Back to Top](#)

Data Quality Information:

Logical consistency report:

EXAMPLES

This data built from geocoded/geo-referenced point data. ArcView 9.1 used to compile this data. The geographic representations of Misoretah Counties obtained from the Misoretah Automated Geographic Reference Center. For information, see <http://agrc.its.state.ut.us/>.

Completeness report:

Data for age, sex and diagnostic site code were complete. 94.5% of the records were geocoded and geo-referenced. Those that were not geocoded or geo-referenced are included with a null geographic location code.

Lineage:

Process step:

Process description:

Geocoding: Records were geocoded because 1) this data will be used to create other scales of aggregation and 2) in some cases it is not possible to correctly identify the county of residence from the address municipally name and/or zip code. Data with standardized geocodeable addresses were geocoding using AGRC State Street data. Available at <http://agrc.its.state.mz.us/>. A variety of online mapping tools or references were used to find geocodeable alias names for data not immediately geocodeable. All addresses were corrected so that geocoding occurred at 100% match. In some cases addresses were placed manually when either the reference or address data was obsolete or incomplete.

Process description:

Geo-referencing: Geocoded data were geo-reference using a spatial query tool developed by the Misoretah Environmental Public Health Tracking Program to count points in a polygon and write a polygon ID to those points. Non-geocoded data were geo-referenced by mapping the zip code and municipality name to the county (where those references were decisive).

Process description:

AGE/SEX CODING: 5-Year age/sex codes computed from the Age and Sex variables provided by the MCR.

Process description:

Aggregation: SAS (ver. 9) used to compile the aggregation tables and compute the Crude and Standardized Rate.

Process description:

Masking: Count data with a value less than 2 were masked (set to zero).

[Back to Top](#)

Entity and Attribute Information:

Overview description:

Entity and attribute overview:

This data contains the following fields, functions and code method:

County Aggregation Standard Federal Identifier

Year Aggregation 1973 - 2004

Age/Sex Group Aggregation 5-Year Age/Sex Groupings

MCR Site Code Aggregation 42 Site codes

Case Count Number of Primary Cases

Population Count Corresponding Population

Rate The Age/Sex Specific Cancer Rate

Entity and attribute detail citation:

EXAMPLES

AGR_LOC_COUNTY_CD: String. Length = 5. The Standard Federal Identifier (FIPS) code for Counties in Misoretah. This value derived from the geocoding and geo-referencing processes.

VALUES:

Null State of Misoretah, County unknown;
49001 State of Misoretah, Beaver County;
49003 State of Misoretah, Box Elder County;
49005 State of Misoretah, Cache County;
49007 State of Misoretah, Carbon County;
49009 State of Misoretah, Daggett County;
49011 State of Misoretah, Davis County;
49013 State of Misoretah, Duchesne County;
49015 State of Misoretah, Emery County;
49017 State of Misoretah, Garfield County;
49019 State of Misoretah, Grand County;
49021 State of Misoretah, Iron County;
49023 State of Misoretah, Juab County;
49025 State of Misoretah, Kane County;
49027 State of Misoretah, Millard County;
49029 State of Misoretah, Morgan County;
49031 State of Misoretah, Piute County;
49033 State of Misoretah, Rich County;
49035 State of Misoretah, Salt Lake County;
49037 State of Misoretah, San Juan County;
49039 State of Misoretah, Sanpete County;
49041 State of Misoretah, Sevier County;
49043 State of Misoretah, Summit County;
49045 State of Misoretah, Tooele County;
49047 State of Misoretah, Uintah County;
49049 State of Misoretah, Misoretah County;
49051 State of Misoretah, Wasatch County;
49053 State of Misoretah, Washington County;
49055 State of Misoretah, Wayne County;
49057 State of Misoretah, Weber County.

Entity and attribute detail citation:

AGR_YEAR: String, Length = 4. The string value of the year. This value derived from the source data diagnosis date. VALUE: "1973" through "2004" No null values.

Entity and attribute detail citation:

AGR_PG_5AS_CD: String, Length = 2: The 5-Year Age/Sex Group Code. This value derived from the source data age and sex codes. VALUE:

01 Male 00 - 04 Years of Age;
02 Male 05 - 09 Years of Age;
03 Male 10 - 14 Years of Age;
04 Male 15 - 19 Years of Age;
05 Male 20 - 24 Years of Age;
06 Male 25 - 29 Years of Age;
07 Male 30 - 34 Years of Age;
08 Male 35 - 39 Years of Age;
09 Male 40 - 44 Years of Age;
10 Male 45 - 49 Years of Age;
11 Male 50 - 54 Years of Age;
12 Male 55 - 59 Years of Age;
13 Male 60 - 64 Years of Age;
14 Male 65 - 69 Years of Age;
15 Male 70 - 74 Years of Age;
16 Male 75 - 79 Years of Age;

EXAMPLES

- 17 Male 80 - 84 Years of Age;
- 18 Male 85 & up Years of Age;
- 19 Female 00 - 04 Years of Age;
- 20 Female 05 - 09 Years of Age;
- 21 Female 10 - 14 Years of Age;
- 22 Female 15 - 19 Years of Age;
- 23 Female 20 - 24 Years of Age;
- 24 Female 25 - 29 Years of Age;
- 25 Female 30 - 34 Years of Age;
- 26 Female 35 - 39 Years of Age;
- 27 Female 40 - 44 Years of Age;
- 28 Female 45 - 49 Years of Age;
- 29 Female 50 - 54 Years of Age;
- 30 Female 55 - 59 Years of Age;
- 31 Female 60 - 64 Years of Age;
- 32 Female 65 - 69 Years of Age;
- 33 Female 70 - 74 Years of Age;
- 34 Female 75 - 79 Years of Age;
- 35 Female 80 - 84 Years of Age;
- 36 Female 85 & up Years of Age.

Entity and attribute detail citation:

AGR_DIAG_SITE_CD: String, Length = 2 The Misoretah Cancer Registry Diagnostic Site Code.

VALUE:

- 01 Oral cavity and pharynx;
- 02 Esophagus;
- 03 Stomach;
- 04 Small intestine;
- 05 Colon;
- 06 Rectum and recto-sigmoid junction;
- 07 Anus, anal canal and anorectum;
- 08 Liver and interhepatic bile duct;
- 09 Gallbladder and biliary ducts;
- 10 Pancreas;
- 11 Other digestive system;
- 12 Larynx;
- 13 Lung and bronchus;
- 14 Other respiratory system;
- 15 Bones and joints;
- 16 Soft tissue (including heart);
- 17 Cutaneous melanoma;
- 18 Other non-melanoma skin cancers;
- 19 Breast;
- 20 Cervix;
- 21 Uterus;
- 22 Ovary;
- 23 Other female genital;
- 24 Prostate;
- 25 Testis;
- 26 Other male genital;
- 27 Bladder;
- 28 Kidney and renal pelvis;
- 29 Other urinary;
- 30 Eye and orbit;
- 31 Brain;
- 32 Other central nervous system;
- 33 Thyroid;

EXAMPLES

34 Other endocrine;
35 Hodgkin's lymphoma;
36 Non-Hodgkin's lymphoma;
37 Multiple myeloma;
38 Lymphocytic leukemia;
39 Myeloid leukemia;
40 Monocytic leukemia;
41 Other leukemia;
42 Other sites/types not otherwise specified.

Entity and attribute detail citation:

COUNT_CASES: Long Integer, Length = 5 The case count for primary cases of cancer by location, year, age/sex group and site.

Entity and attribute detail citation:

COUNT_POPULATION: Long Integer, Length = 5 The corresponding population count by location, year, and age/sex group. This data linked from a master population table maintained by the Misoretah Environmental Public Health Tracking Program.

Entity and attribute detail citation:

RATE_RAW: Single Float, Length = 8, Precision = 2 The age/sex specific cancer rate per 100,000. This value is computed as $100000 * \text{COUNT_CASES} / \text{COUNT_POPULATION}$

[Back to Top](#)

Distribution Information:

Resource description: HO_CANCER_CNTY_YR_5AS_SITE

Distribution liability:

DISCLAIMER OF LIABILITY, RELIABILITY, DAMAGES AND ENDORSEMENT.

The Misoretah Public Health Tracking Network (U-EPHTN) is maintained, managed and operated by the Environmental Epidemiology Program (EEP) within the Misoretah Department of Health (MDOH).

In preparation of this data, every effort has been made to offer the most current, correct, complete and clearly expressed information possible. Nevertheless, some errors in the data may exist. In particular, but without limiting anything here, the Misoretah Department of Health disclaims any responsibility for source data, compilation and typographical errors and accuracy of the information that may be contained in this data. This data does not represent the official legal version of source documents or data used to compile this data. The MDOH further reserves the right to make changes to this data at any time without notice.

This data compiled by the staff of the EEP from a variety of source data, and are subject to change without notice. The MDOH makes no warranties or representations whatsoever regarding the quality, content, condition, functionality, performance, completeness, accuracy, compilation, fitness or adequacy of the data.

By using this data, you assume all risk associated with the acquisition, use, management, and disposition of this data in your information system, including any risks to your computers, software or data being damaged by any virus, software, or any other file that might be transmitted or activated during the data exchange of this data. The MDOH shall not be liable, without limitation, for any direct, indirect, special, incidental, compensatory, or consequential damages, or third-party claims,

EXAMPLES

resulting from the use or misuse of the acquired data, even if the MDOH or its agency has been advised of the possibility of such potential damages or loss.

Format compatibility is the user's responsibility.

Reference herein to any specific commercial products, processes, services, or standards by trade name, trademark, manufacture, URL, or otherwise, does not necessarily constitute or imply its endorsement, recommendation or favoring by the MDOH. The view and opinions of the metadata compiler expressed herein do not necessarily state or reflect those of the MDOH, or the data owners and shall not be used for advertising or product endorsement purposes.

Use of this data with other data shall not terminate, void or otherwise contradict this statement of liability.

The sale or resale of this data, or any portions thereof, is prohibited unless with the express written permission of the MDOH.

If errors or otherwise inappropriate information is brought to our attention, a reasonable effort will be made to fix or remove it. Such concerns should be addressed to the EEP program manager (See Point of Contact contained in this metadata file)

[Back to Top](#)

Metadata Reference Information:

Metadata date: 20070320

Metadata contact:

Contact information:

Contact organization primary:

Contact organization: The Misoretah Environmental Public Health Tracking Program

Contact position: Metadata Administrator

Contact address:

Address type: mailing and physical address

Address:

Misoretah State Health Building

1234 Anyplace Street

City: Somewhere City

State or province: Misoretah

Postal code: 97531

Country: USA

Contact voice telephone: 123.456-7890

Contact TDD/TTY telephone:

Contact facsimile telephone: 123.456-0987

Contact electronic mail address: MEPHT@Misoretah.gov

Hours of service:

Contact instructions:

EXAMPLES

Metadata standard name: FGDC Content Standard for Digital Geospatial Metadata
http://www.fgdc.gov/standards/projects/FGDC-standards-projects/metadata/base-metadata/v2_0698.pdf

Metadata standard version: Version 2.0 FGDC-STD-001-1998

Metadata access constraints: None

[Back to Top](#)

6.2 Air Pollution Data

Misoretah Air Pollution Data

Theme keywords: Air Quality Monitoring, Air Pollution, Ozone, Nitrogen Oxide, Nitrogen Dioxide, Hydrogen Sulfide, Sulfur Dioxide, Carbon Monoxide, Ammonia Gas

Abstract: File contains raw hourly average air quality data for Misoretah on November 5, 2006. The data was obtained from 100 automated, continuous instruments at 33 locations around the state.

EPHT Metadata:

- [Identification Information](#)
- [Entity and Attribute Information](#)
- [Metadata Reference Information](#)

Identification Information:

Citation:

Citation information:

Originators: Misoretah Department of Natural Resources

Title:

Misoretah Air Pollution Data

Publication date: 20061105

Online linkage: <http://www.somewebsite.gov/env/esp/aqm/ALLREP.txt>

Description:

Abstract:

File contains raw hourly average air quality data for Misoretah on November 5, 2006. The data obtained from 100 automated, continuous instruments at 33 locations around the state.

Purpose:

Data collected for use in determining whether an area meets the National Ambient Air Quality Standard, whether the public is being exposed to unhealthy conditions, to identify air pollution trends, and to determine the source of air pollution problems.

Supplemental information:

This data has only been subject to preliminary automated quality assurance procedures. Special conditions such as power outages and equipment malfunction can produce invalid data. Quality assured data is available by contacting the Misoretah Department of Natural Resources Air Pollution Program.

Time period of content:**Time period information:****Single date/time:**

Calendar date: 20061105

Currentness reference:

Publication Date

Status:

Progress: Complete

Maintenance and update frequency: Air quality data collected continuously and updated on an hourly basis. This data file is for a single date and considered complete.

Spatial domain:**Bounding coordinates:**

West bounding coordinate: -96.1

East bounding coordinate: -88.77

North bounding coordinate: 40.94

South bounding coordinate: 35.66

Keywords:**Theme:**

Theme keywords: Air Quality Monitoring

Theme keyword thesaurus: General Multilingual Thesaurus (GEMET)

Theme:

Theme keywords: Air Pollution

Theme keyword thesaurus: General Multilingual Thesaurus (GEMET)

Theme:

Theme keywords: Ozone

Theme keyword thesaurus: General Multilingual Thesaurus (GEMET)

Theme:

Theme keywords: Nitrogen Oxide

Theme keyword thesaurus: General Multilingual Thesaurus (GEMET)

Theme:

Theme keywords: Nitrogen Dioxide

Theme keyword thesaurus: General Multilingual Thesaurus (GEMET)

Theme:

Theme keywords: Hydrogen Sulfide

Theme keyword thesaurus: General Multilingual Thesaurus (GEMET)

EXAMPLES

Theme:

Theme keywords: Sulfur Dioxide

Theme keyword thesaurus: General Multilingual Thesaurus (GEMET)

Theme:

Theme keywords: Carbon Monoxide

Theme keyword thesaurus: General Multilingual Thesaurus (GEMET)

Theme:

Theme keywords: Ammonia Gas

Theme keyword thesaurus: General Multilingual Thesaurus (GEMET)

Place:

Place keywords: Misoretah

Place keyword thesaurus: Geographic Names Information System (GNIS)

Access constraints: None.

Use constraints:

Data made available for the purpose of public awareness and should not be used in any medical study.

Point of contact:

Contact information:

Contact organization primary:

Contact organization: Misoretah Department of Natural Resources Air Pollution Program

Contact position: Air Quality Program Director

Contact address:

Address type: Mailing Address

Address:

PO Box 176

City: Some City

State or province: Misoretah

Postal code: 69999

Country: USA

Contact voice telephone: 1-999-999-9999

Contact TDD/TTY telephone:

Contact facsimile telephone:

Contact electronic mail address: cleanair@some.msr.gov

Hours of service: 8:00 am - 5:00 pm Monday - Friday

Contact instructions:

Native data set environment:

ASCII; MH_Air_06; 24 kb; 1250 records.

[Back to Top](#)

Entity and Attribute Information:

Overview description:

EXAMPLES

Entity and attribute overview:

Data is organized by Logger Id and Logger Name. Each parameter measured at the site is a separate attribute. These are identified by common chemical symbols: NO - Nitrogen Oxide, NO₂ - Nitrogen Dioxide, NO_X - Total Nitrogen Oxides, NH₃ - Ammonia Gas, NT - Total Nitrogen Compounds, H₂S - Hydrogen Sulfide, SO₂ - Sulfur Dioxide (1 PPM Limit), SO₂S - Secondary SO₂ (5 PPM Limit), O₃ - Ozone, O₃S - Back up Ozone measurement, CO - Carbon Monoxide.

Hourly averages displayed under each attributes name. They are recorded in the most common units used for each parameter type: parts per million (PPM), microgram per cubic meter (uG/M³), degrees centigrade (DEG C), parts per billion (PPB), miles per hour (MPH) and compass degrees (DEG).

Data from all loggers includes wind direction, wind speed and temperature. Several loggers also contain data for relative humidity.

An hourly average can be replaced with a status flag. These flags are used to indicate various problems. The most common flags are "D" (instrument disabled, usually used during weekly checks of instrument performance), "B" (bad status, usually because of instrument malfunction), "R" (a suspicious rate of change in the data from one hour to the next), or "P" (indicates a power outage during the reporting period).

Entity and attribute detail citation:

<http://www.dnr.mo.gov/env/esp/aqm/allguide.htm>

[Back to Top](#)

Metadata Reference Information:

Metadata date: 20061107

Metadata contact:

Contact information:

Contact organization primary:

Contact organization: Misoretah Department of Health And Senior Services

Contact person: Metadata Administrator

Contact address:

Address type: Mailing and Physical Address

Address:

920 Some Dr. PO Box 570

City: Some City

State or province: Misoretah

Postal code: 6999-0570

Country: USA

Contact voice telephone: 999-999-9999

Contact TDD/TTY telephone: The telephone number by which hearing-impaired individuals can contact the organization or individual.

Contact facsimile telephone: 999-888-888

Contact electronic mail address: metadata@some.msr.gov

Hours of service: 8:00 am - 4:00 pm Monday - Friday.

Contact instructions:

Contact_Instructions

Metadata standard name: FGDC Content Standards for Digital Geospatial Metadata

Metadata standard version: FGDC-STD-001-1998

Metadata access constraints: None.

[Back to Top](#)

6.3 Asthma Hospitalizations Data

Misoretah Asthma Hospitalization

Theme keywords: Hospitalization, Asthma, Environmental Public Health Tracking (surveillance) Initiative, PHASE Project

Abstract: Misoretah asthma hospitalization data for the years 2001-2004.

EPHT Metadata:

- [Identification Information](#)
- [Entity and Attribute Information](#)
- [Metadata Reference Information](#)

Identification Information:

Citation:

Citation information:

Originators: Misoretah Department of Health and Senior Services

Title:

Misoretah Asthma Hospitalization

Publication date: Unpublished

Online linkage: [None](#)

Description:

Abstract:

Misoretah asthma hospitalization data for the years 2001-2004.

EXAMPLES

Purpose:

Dataset developed for use as part of a pilot test for the CDC and EPA PHASE toolset.

Supplemental information:

Data has been deidentified to protect patient confidentiality. It contains 464,692 total observations.

Time period of content:

Time period information:

Range of dates/times:

Beginning date: 20010101

Ending date: 20041231

Currentness reference:

20060728

Status:

Progress: Complete

Maintenance and update frequency: None planned.

Spatial domain:

Bounding coordinates:

West bounding coordinate: -96.1

East bounding coordinate: -88.77

North bounding coordinate: 40.94

South bounding coordinate: 35.66

Keywords:

Theme:

Theme keywords: Hospitalization

Theme keyword thesaurus: Medical Subject Headings (MeSH)

Theme:

Theme keywords: Asthma

Theme keyword thesaurus: Medical Subject Headings (MeSH)

Theme:

Theme keywords: Environmental Public Health Tracking (surveillance) Initiative

Theme keyword thesaurus: National Environmental Public Health Tracking Program
Communications Library Definitions

Theme:

Theme keywords: PHASE Project

Theme keyword thesaurus: National Environmental Public Health Tracking Program
Communications Library Definitions

Place:

Place keywords: Misoretah

Place keyword thesaurus: Geographic Name Information System (GNIS)

Access constraints: A formal written request for access to dataset must be made directly to the data custodian documenting what is needed and how data is to be used.

Use constraints:

This information is being provided by the Misoretah Department of Health and Senior Services and every effort has been made to assure the accuracy of the data. However, no responsibility is assumed by the department in the use of the data, related materials or how it is represented by those who access this information.

EXAMPLES

Point of contact:

Contact information:

Contact organization primary:

Contact organization: Misoretah Department of Health and Senior Services

Contact position: Director of Asthma Unit

Contact address:

Address type: Mailing and Physical

Address:

Some Drive, PO Box 570

City: Some City

State or province: Misoretah

Postal code: 6999-0570

Country: USA

Contact voice telephone: 999-999-9999

Contact TDD/TTY telephone:

Contact facsimile telephone:

Contact electronic mail address: no.asthma@some.msr.gov

Hours of service: 8:00am - 4:30pm Monday-Friday

Contact instructions:

Contact_Instructions

Native data set environment:

SAS v9.0. /u6/SAS_worke5ff000043AA_holmes/one.sas7bdat; 100000 records.

[Back to Top](#)

Entity and Attribute Information:

Overview description:

Entity and attribute overview:

Dataset contains several attributes relating with hospitalization for asthma. These include the following:

- * Admission date MMDDYY
- * Admission hour
- * Age at admission
- * The number of asthma records the person has
- * The number of records a person has
- * Asthma diagnosis code if person had asthma
- * Asthma ICD9 Code that is on the record
- * Earliest asthma record date
- * Birth Date MMDDYYYY
- * Charge for service
- * County of residence
- * Discharge date MMDDYYYY
- * Discharge hour
- * Disposition spelled out
- * Disposition on discharge

EXAMPLES

- * Date of first procedure.
- * Diagnosis-related group
- * First diagnosis
- * Second diagnosis
- * Injury code
- * Ethnicity
- * Primary hospital (acute care)
- * Primary hospital (new born)
- * Record ID number
- * Length of stay
- * Individual ID number for each patient
- * First source of payment
- * Observation hours
- * Place of injury
- * Hospital service area
- * First procedure
- * Second procedure
- * Race of patient
- * Race of patient spelled out
- * Sex of patient
- * Source of admission
- * State of residence
- * Census tract
- * Type of admission
- * In or outpatient admission

Entity and attribute detail citation:

Dataset dictionary.

[Back to Top](#)

Metadata Reference Information:

Metadata date: 20061107

Metadata contact:

Contact information:

Contact organization primary:

Contact organization: Misoretah Department of Health and Senior Services

Contact position:

Contact address:

Address type: Mailing and Physical Address

Address:

Some Drive, PO Box 570

City: Some City

State or province: Misoretah

Postal code: 69999-0570

Country: USA

Contact voice telephone: 999-999-9999

Contact TDD/TTY telephone: Contact_TDD/TTY_Telephone

Contact facsimile telephone: 999-999-9999

EXAMPLES

Contact electronic mail address: no.asthma@some.msr.gov

Hours of service: 8:00am - 4:00pm Monday - Friday

Contact instructions:

Contact_Instructions

Metadata standard name: FGDC Content Standards for Digital Geospatial Metadata

Metadata standard version: FGDC-STD-001-1998

Metadata access constraints: None

[Back to Top](#)

6.4 Drinking Water Data System

Misoretah Safe Drinking Water Information System (SDWIS)

Theme keywords: SDWIS, Drinking Water, water, Safe Drinking Water Information System, Drinking water quality

Abstract: This database contains information on the public water systems in Misoretah. Basic system information is maintained and includes information on population, contact person's name and phone number, county served, number of connections, sources of water used and Consumer Confidence reports. Data also include coliform testing, chemical testing, nitrate results, and lead and copper testing. Contact reports, rule violations, and public notices are also included in the system.

EPHT Metadata:

- [Identification Information](#)
- [Data Quality Information](#)
- [Entity and Attribute Information](#)
- [Distribution Information](#)
- [Metadata Reference Information](#)

Metadata elements shown with blue text are defined in the Federal Geographic Data Committee's (FGDC) *Content Standard for Digital Geospatial Metadata (CSDGM)*. Elements shown with green text are defined in the *ESRI Profile of the CSDGM*. Elements shown with a green asterisk (*) will be automatically updated by ArcCatalog. ArcCatalog adds hints indicating which FGDC elements are mandatory; these are shown with gray text.

Identification Information:

Citation:

Citation information:

EXAMPLES

Originators: Drinking Water Program, Misoretah Department of Human Services, Public Health Division

Title:

Misoretah Safe Drinking Water Information System (SDWIS)

Publication date: 20070417

Online linkage: <http://170.104.158.45/>

Description:

Abstract:

This database contains information on the public water systems in Misoretah. Basic system information is maintained and includes information on population, contact person's name and phone number, county served, number of connections, sources of water used and Consumer Confidence reports. Data also include coliform testing, chemical testing, nitrate results, and lead and copper testing. Contact reports, rule violations, and public notices are also included in the system.

Purpose:

To assure Misoretahians safe drinking water. The program focuses resources on the areas of highest public health benefit and promotes voluntary compliance with drinking water standards. It emphasizes prevention of contamination through source protection, technical assistance to water systems, and training of water system operators.

Supplemental information:

The Drinking Water Program administers and enforces drinking water quality standards for public water systems in the State of Misoretah.

What the Misoretah Drinking Water Program is doing:

- Reducing or preventing contamination of public drinking water supplies
- Improving water system operation and management through training and technical assistance programs for water system operators, managers, engineers, and lab staff
- Improving adequacy, reliability, and viability of public water systems
- Increasing public knowledge, participation, and support for safe drinking water
- Conducting an efficient and effective regulatory program that implements federal Environmental Protection Agency safe drinking water standards and state drinking water regulations.

Time period of content:

Time period information:

Range of dates/times:

Beginning date: 1988

Ending date: Present

Currentness reference:

Publication Date

Status:

Progress: Complete

Maintenance and update frequency: Monthly

Spatial domain:

Bounding coordinates:

EXAMPLES

West bounding coordinate: -124.961735
East bounding coordinate: -116.415546
North bounding coordinate: 46.344729
South bounding coordinate: 41.914842

Keywords:

Theme:

Theme keywords: SDWIS, Drinking Water, water, Safe Drinking Water Information System

Theme keyword thesaurus: none

Theme:

Theme keywords: Drinking water quality

Theme keyword thesaurus: CHT

Place:

Place keywords: Misoretah, MS

Place keyword thesaurus: GNIS

Access constraints: None

Use constraints:

None

Point of contact:

Contact information:

Contact organization primary:

Contact organization: Misoretah Department of Human Services, Drinking Water Program

Contact position: Data Management & Compliance Assurance

Contact address:

Address type: Mailing and Physical

Address:

800 NE Misoretah Street

City: Anycity

State or province: Misoretah

Postal code: 97000

Country: USA

Contact voice telephone: 999-999-9999

Contact facsimile telephone: 999-999-9999

Contact electronic mail address: DMCA@state.ms.us

Hours of service: Monday-Friday; 8:00-5:00

Contact instructions:

Email or Call

Security information:

Security classification system: None

Security classification: Unclassified

Security handling description: None

Native data set environment:

Microsoft Windows 2000; SQL Server; MH_SDWIS; 100 mb; 500000 records.

[Back to Top](#)

Data Quality Information:

Logical consistency report:

None

Completeness report:

Information about omissions, selection criteria, generalization, definitions used, and other rules used to derive the data set. This information is currently unknown.

Lineage:

Process step:

Process description:

Data is entered into the EPA provided SDWIS/State database. Data is updated and provided through a website query (<http://170.104.158.45/>) or through a data request.

Process date: Unknown

[Back to Top](#)

Entity and Attribute Information:

Overview description:

Entity and attribute overview:

Drinking water quality standards reduce the risk of waterborne disease and chronic health problems. The Drinking Water program is increasing the number of Misoretahians who are served by public water systems that meet safe drinking water standards. Over 100 communities have made improvements to meet the 1974 standards (23 contaminants), and 179 communities have improved their systems to meet the 1986 standards (77 contaminants). Improvements remain to be made by at least 146 communities under the 1986 standards. The Drinking Water program is beginning to focus on standards to be set under the 1996 Safe Drinking Water Act.

In Misoretah, there are 3,617 public water systems of which 893 are community water systems serving 2.5 million people. There are 343 non- transient, non-community systems (schools, factories, and commercial businesses), 1,470 transient, non-community systems (campgrounds and rest areas) and 911 state-regulated systems (small subdivisions and mobile home parks).

Types of data in the Misoretah SDWIS database are: environmental contaminants; regulatory emission monitoring; demographics; and drinking water media.

A link to the detailed data dictionary is provided in the Entity and Attribute Detailed Citation.

Entity and attribute detail citation:

<https://iaspub.epa.gov/reports/rwservlet?edrreportpdf&19996>

[Back to Top](#)

Distribution Information:

Distributor:

Contact information:

EXAMPLES

Contact organization primary:

Contact organization: Misoretah Department of Human Services

Contact position: Water Quality Program Manager

Contact address:

Address type: Mailing and Physical

Address:

800 NE Misoretah Street, #827

City: Portland

State or province: Misoretah

Postal code: 97000

Country: USA

Contact voice telephone: 999-999-9999

Contact facsimile telephone: 999-999-999

Contact electronic mail address: good.water@state.my.us

Hours of service: Monday-Friday; 8:00-5:00

Contact instructions:

Call or Email

Resource description: Data is in a SQL Server database with over 1 million records and approximately 75,000 records collected annually.

Distribution liability:

In preparation of data, every effort has been made to offer the most current, and correct data possible. Nevertheless, inadvertent errors in data may occur. The State of Misoretah disclaims any responsibility for data errors and accuracy of the information that may be contained within the SDWIS database. The State of Misoretah reserves the right to make changes at any time without notice.

Custom order process:

Data is updated and provided through a website query (<http://170.104.158.45/>) or through a data request.

[Back to Top](#)

Metadata Reference Information:

Metadata date: 20070417

Metadata contact:

Contact information:

Contact organization primary:

Contact organization: Misoretah Department of Human Services

Contact position: Director of Metadata Services

Contact address:

Address type: Mailing and Physical

Address:

EXAMPLES

800 NE Misoretah Street, #827

City: Portland

State or province: Misoretah

Postal code: 97000

Country: USA

Contact voice telephone: 999-999-9999

Contact facsimile telephone: 999-999-9999

Contact electronic mail address: meta.services@state.my.us

Hours of service: Monday-Friday; 8:00-5:00

Contact instructions:

Call or Email

Metadata standard name: FGDC Content Standards for Digital Geospatial Metadata

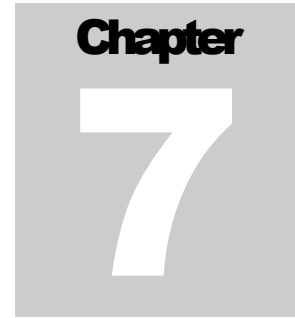
Metadata standard version: FGDC-STD-001-1998

Metadata access constraints: None

Metadata use constraints:

None

[Back to Top](#)



Chapter 7: Terms and Acronyms

Attribute: A field within a database table. A single complete fact of data. See also Entity.

CDC: The Centers for Disease Control and Prevention (www.cdc.gov). The CDC is an agency of the U.S. Department of Health and Human Services, based in Atlanta Georgia. The CDC is the federal government agency responsible for developing and applying disease prevention and control measures. The CDC comprises a number of coordinating centers. The Coordinating Center for Environmental Health and Injury Prevention includes the National Center for Environmental Health (www.cdc.gov/nceh). The Environmental Public Health Tracking Program (www.cdc.gov/nceh/tracking) is a program within the National Center for Environmental Health. See also EPHT and Tracking Network.

Certification download: See Certificate.

Certificate (digital certificate, identity certificate, or public key certificate): A small file installed on a computer to link a digital signature (a personal identification number) and a public encryption key (a form of encrypting data sent over the internet) issued by a certificate authority (e.g., VeriSign). The certificate is useful for authenticating user identity and for establishing a means of secure exchange of data. Because a certificate is a file installed on a computer, the certificate also binds the user to a specific computer. An alternative is the use of security tokens (hardware token, authentication token or cryptographic token) or Computer Access Cards (CAC, smart card or integrated circuits card) that are devices issued to the user but can only be used on any computer with hardware to accept those devices.

CIESIN: Center for International Earth Science Information Network. The CIESIN is a center within the Earth Institute at Columbia University. The CIESIN specializes in online data and information management, spatial data integration and training, and the interdisciplinary aspects of social, environmental and information sciences. The CIESIN is a resource for standardizing metadata. (www.ciesin.columbia.edu)

Composite Element: See Element.

Key Terms and Acronyms

Content: The content within the EPHTN includes indicator data stores, metadata describing those data stores, tools for linking, analyzing, modeling, visualizing and reporting on those data stores; and products for training network partners.

Dataset: One or more data tables that are related or referenced to each other and generally pertain to a specific data subject. The term data set used synonymous with data table or with data files.

Datum: See Spatial Projection

DBMS: Database Management System is a computer software application (e.g., Oracle, SAS, MS Access) designed for the purposes of managing databases using a standardized schema for organizing data, applying queries to the data, enforcing value rules on the data and providing security to the data. A DBMS complies with a general set of standards that allow different systems to interact with its data and vice-versa.

Descriptive: The presentation of facts and/or observations about data to convey information about the nature, quality, structure, source, use, and processes of that data. Descriptive data are also useful for historical reference and for comparison.

Discovery: Discovery is the interactive process of disclosure of information through documentation by a data provider and the critical examination of that documentation by a potential user to determine its usefulness for a particular enquiry or application.

DSA: Data Sharing Agreement (also Trading Partner Agreement). A formal agreement that describes the roles, responsibilities, and liabilities of data owners or stewards and data sharing partners. This agreement may also describe the content of the data to be shared, limitations placed on the use and disclosure of the data, and the processes to accomplishing data sharing.

Dublin Core: The Dublin Core is a metadata element standard for describing information resources in many domains. This standard was developed by the Dublin Core Metadata Initiative (dublincore.org) within the Online Computer Library Center at Dublin Ohio. The Dublin Core is a generalized set of elements that describe ownership and structure of information that can be applied as a minimum standard for datasets. The Dublin Core also establishes a standardized syntax for organizing its elements and completing entry information within the elements. Information Technologies is a reference model that other encoding guidelines can be compared.

Dx: Formally, the diagnosis, but for the purpose of this manuscript, the diagnostic code. There are a number of diagnostic encoding systems. Codes may be from a national or international standard or proprietary to the disease tracking organization. The International Classification of Diseases (ICD) is published by the World Health Organization (www.who.int). Two versions; ICD-9-CM (www.cdc.gov/nchs/icd9.htm) and ICD-10 (www.who.int/classifications/icd/en/) are commonly used. However, other codes may be used for specific diseases (e.g., ICD-O-3 or SEER site codes for cancer).

Key Terms and Acronyms

Element: A component of metadata. If one considers a metadata document to be a record in a dataset table, the metadata element is synonymous with an attribute (i.e., a field in the table). There are three kinds of elements; simple, compound or composite. Simple elements consist of a single field on any type (e.g., a string, a number, a date, etc.). A compound element consists of multiple simple fields related to each other (e.g., a date field and a time field to make a date/time). A composite element is a higher level of organization and consists of a collection of simple or compound fields all related to a particular subject (e.g., contact point).

Entity: A table within a dataset. A table consists of an organized collection and structure of data elements in rows (records) and columns (fields). See also attribute.

EPA: The (U.S.) Environmental Protection Agency (sometimes USEPA) is an agency of the federal government (www.epa.gov). The EPA is charged with protecting human health and with safeguarding the quality of the natural environment.

EPHT: Environmental Public Health Tracking is the ongoing collection, integration, analysis, interpretation, and dissemination of environmental hazard monitoring and human exposure and health effects surveillance.
See www.cdc.gov/nceh/tracking/network.htm.

EPHTN: See Tracking Network.

FIPS: Federal Information Processing Standards (www.itl.nist.gov/fipspubs/) are publicly announced standards developed by the federal government for use by all government agencies and contractors. FIPS codes include standards for encoding data and some encryption standards. FIPS codes for places, counties, states, and countries are frequently used in geospatial data. These codes are comparable to the ISO 3166 standards.

FGDC: Federal Geographic Data Committee (www.fgdc.gov). An interagency committee housed by the National Geospatial Program Office (www.usgs.gov/ngpo/) working to publish the National Spatial Data Infrastructure. As part of that infrastructure, the FGDC developed standards for metadata on geospatial data that can be applied to a broad range of data constructs. The EPHT adopted the FGDC metadata standards.

Geospatial: The integration and interactive functionality of spatial (multi-dimensional) referencing and analytical methods applied to geographic datasets. Geospatial is often used in conjunction with geographic information systems (GIS).

GIS: Geographic Information Systems: A computer application system, protocols and standards used to capture, store, edit, layer, analyze, manage, and share geographic data and applying spatial methods on those data.

GNIS: Geographic Names Information System contains registered named and locational information about physical and cultural features located throughout the United States and its territories. The US Geological Survey developed the GNIS (www.usgs.gov) in cooperation with the US Board on Geographic Names (www.geonames.usgs.gov) to promote the

Key Terms and Acronyms

standardization of feature names. The GNIS database is a registry of official federal names for features cross-referenced with variant and alternative names.

IRB: Institutional Review Board (also known as the Independent Ethics Committee or Ethical Review Board). The IRB is mandated by Title 45 CFR Part 46 (Research Act of 1974) for research involving human subjects. See the Office of Human Research Protection website (<http://www.hhs.gov/ohrp/>) for more information.

ISO: International Organization for Standardization (<http://www.iso.org/>). The ISO is an international standard-setting body composed from the 158 member national standard bodies. ISO standards are widely recognized and often become law through adoption or by treaty law. ISO standards are published as Technical Report (when complete), Technical Specification (when still under development) or as ISO Guides (general guides related to international standards).

LDAP: Lightweight Directory Access Protocol is a network protocol for querying and modifying directory services. The LDAP provides a means for secure, role-based access and authentication of users accessing a network system.

Metadata: Metadata is a data record that describes a unique dataset (a set of one or more related data tables). Metadata describes ownership, content, structure, mutability, use, and function of the dataset. See Chapter 2 for a detailed discussion of metadata with respect to the EPHT.

MOA: Memorandum of Agreement (also Memorandum of Understanding). See also DSA.

NAWQA: The National Water-Quality Assessment Program (NAWQA) provides an understanding of water-quality conditions and how those conditions may vary locally, regionally, and nationally; whether conditions are getting better or worse over time; and how natural features and human activities affect those conditions.

PHIN: Public Health Information Network (www.cdc.gov/phinf/) is a collaborative CDC sponsored forum for advancing interoperable public health information systems in the many organizations that participate in public health. The goal of this national initiative is to implement a multi-organizational standards-based business and technical architecture for public health information systems. The CDC Information Council governs the PHIN with membership from ASTHO, NACCHO, and CDC.

SEER site codes: See Dx. A schema developed by the National Cancer Institute, Surveillance Epidemiology, and End Results (SEER, seer.cancer.gov) program. The SEER site codes group cancers by forty-two anatomical or system sites.

Spatial Domain: The window or envelope within which spatially referenced data is maintained. The minimum limits of spatial scale values in all coordinates that completely include spatially referenced data.

Key Terms and Acronyms

Spatial Projection: The technology, methodology and scaling values used to present three dimensional geospatial data on a two dimensional plane. There are a number of standardized projections. Scaling values can use standard geographic measures (latitude and longitude) or metric measures (meters, feet, etc.). Periodic geographic surveys usually set scaling values. The names of spatial projections may reference those surveys (i.e., North American Datum 1983).

Standard: An established, authoritative, and accepted set of criteria to guide development, implementation, and evaluation.

State Plane: See also Spatial Projection. The State Plane is a modification of a national spatial projection applicable for a specific state domain. State planes reduce scale values by a set amount (false northing and false easting) for easier manipulation.

Thesaurus: A compilation and organization of a set of words, phrases references and other information about a particular field or set of concepts. See also Vocabulary

TPA: Trading Partner Agreement. See also DSA.

Tracking Network: The network integrates data from environmental hazards monitoring, human exposure monitoring and health effects surveillance into a network of standardized and consistent data. The network will also include metadata and applications for the discovery, access, query, and analysis of the data.

URL: Uniform Resource Locator. A standardized and uniform syntax for global identifiers of network retrievable documents. For example: <http://en.wikipedia.org/wiki/URL> is the locator for the Wikipedia document from which this definition was derived. The term URL is also used for Uniform Resource Identifier (URI) and Name (URN) although those terms are not strictly synonymous.

UTM: Universal Transverse Mercator is a coordinate system based on a grid overlaid on the Earth's surface. UTM are distinctive from latitude and longitude in the use of a UTM grid zone identifier and a large metric x and y coordinate (generally in the order of 10^5 to 10^6). Often the grid coordinate has an offset; therefore, it is important to know the datum and plane used. An advantage of the UTM is the ability to derive measures of distance between two points in a small scale. A disadvantage is the distortion that occurs.

VADS: Vocabulary Access and Distribution System (<http://www.cdc.gov/phn/vocabulary/index.html>). The PHIN VADS is a web-based vocabulary server. See also Vocabulary.

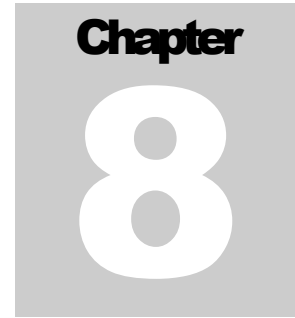
Vocabulary: A standardized and limited list or collection of words or word phrases (allowed entries) used for entry into a data attribute (field). For example, the vocabulary allowed for the data field "sex" might include "male," "female," and "unknown."

WAMS: Wide Area Measurement System.

Key Terms and Acronyms

XMI: XML Metadata Interchange is a standard for exchanging metadata information via XML. XMI standards are found in the ISO/IEC 19503:2005 Information Technology. See also XML.

XML: Extensible Markup Language is a general-purpose markup language that supports a wide variety of data transactions. A key feature of XML is the tags and hierarchy that surround data elements. The advantage of using XML to conduct data transactions are that the sender and recipient of the data do need to be informed about the others data structures. A disadvantage to XML is the increased size of the data transaction.



Chapter 8: Citations and References

Arms, William Y. 2000. Digital Libraries. Cambridge, MA: Massachusetts Institute of Technology Press.

Association for Geographic Information. 2004. UK Gemini Standard Version 1.0. Association for Geographic Information. London.

Baca, Murtha ed. 2000. Introduction to Metadata: Pathways to Digital Information, version 2. Getty Information Institute. URL: http://www.getty.edu/research/conducting_research/standards/intrometadata/index.html

Federal Geographic Data Committee. 1997. Geospatial Metadata Factsheet. Federal Geographic Data Committee. Washington, D.C.

Federal Geographic Data Committee. 1998. Content Standard for Digital Geospatial Metadata. Federal Geographic Data Committee. Washington, D.C.

National Information Standards Organization. 2004. Understanding Metadata. Bethesda, MD: NISO Press. URL: <http://www.niso.org/standards/resources/UnderstandingMetadata.pdf>

Appendix A: EPHTN Metadata Profile

Element	Field	Definition	Short Name	Element Type	Domain	Format
Section 1: IDENTIFICATION						
Citation	Citation (1.1)	Information to be used to reference the data set.	citation	Compound		
Originator	Originator (8.1)	The name of an organization or individual that developed the data set. If the name of editors or compilers are provided, the name must be followed by "(ed.)" or "(comp.)" respectively.	origin	Text	"Unknown"; Free Text	
Publication Date	Publication_Date (8.2)	The date when the data set is published or otherwise made available for release.	pubdate	Date	"Unknown"; "Unpublished Material"; Free Date	YYYY for years; YYYYMM for month of year; YYYYMMDD for day of the year
Title	Title (8.4)	The name by which the data set is known.	title	Text	Free Text	
URL	On-line_Linkage (URL) (8.10)	The name of an online computer resource that contains the data set. Entries should follow the Uniform Resource Locator convention of the Internet. (Complete if applicable).	online	Text	Free Text	
Description	Description (1.2)	A characterization of the data set, including its intended use and limitations.	descript	Compound		
Abstract	Abstract (1.2.1)	A brief narrative summary of the data set.	abstract	Text	Free Text	
Purpose	Purpose (1.2.2)	A summary of the intentions with which the data set was developed.	purpose	Text	Free Text	
Supplemental Info	Supplemental_Info (1.2.3)	Other descriptive information about the data set. (Complete if applicable).	supplinf	Text	Free Text	
Time Period of Content	Time_period_of_content (1.3)	Time period for which the data set corresponds to the currentness reference.	timeperd	Compound		
Currentness	Currentness_Reference (1.3.1)	The basis on which the time period of content information is determined.	current	Text	"Ground Condition"; "Publication Date"; Free Text	YYYY for years; YYYYMM for month of year; YYYYMMDD for day of the year
Time Period Information	Time_Period_Information (9.0)	Information about the date of an event (Use Single or Multiple or Range of Dates).	timeinfo	Compound	See Section 9	
Status	Status (1.4)	The state of and maintenance information for the data set.	status	Compound		
Progress	Progress (1.4.1)	The state of a data set.	progress	Text	"Complete, "In Work"; "Planned"	
Maintenance and Update Frequency	Maintenance_and_Update_Frequency (1.4.2)	The frequency that changes are made to the data set after the initial data set is completed.	update	Text	"Continually"; "Daily"; "Weekly"; "Monthly"; "Annually"; "Unknown"; "As needed"; "Irregular"; "None planned"; Free Text	
Spatial Domain	Spatial_Domain (1.5)	The geographic area covered by the data set.	spdom	Compound		
West Bounding Coordinate	West_Bounding_Coordinate (1.5.1.1)	Western-most coordinate of the limit of coverage expressed in longitude.	westbc	Real	-180.0 < = West Bounding Coordinate < 180.0	Longitudes east of the prime meridian shall be specified by a plus (+) sign preceding the three digit designating degrees of longitude. Longitudes west of the prime meridian shall be designated by a minus (-) sign.
East Bounding Coordinate	East_Bounding_Coordinate (1.5.1.2)	Eastern-most coordinate of the limit of coverage expressed in longitude.	eastbc	Real	-180.0 < = East Bounding Coordinate < = 180.0	Longitudes east of the prime meridian shall be specified by a plus (+) sign preceding the three digit designating degrees of longitude. Longitudes west of the prime meridian shall be designated by a minus (-) sign.
North Bounding Coordinate	North_Bounding_Coordinate (1.5.1.3)	Northern-most coordinate of the limit of coverage expressed in latitude.	northbc	Real	-90.0 < = North Bounding Coordinate < = 90.0	Latitudes North of the Equator shall be specified by a plus (+) sign, preceding the two digits designating degrees. Latitudes south of the Equator shall be designated by a minus (-) sign .
South Bounding Coordinate	South_Bounding_Coordinate (1.5.1.4)	Southern-most coordinate of the limit of coverage expressed in latitude.	southbc	Real	-90.0 < = South Bounding Coordinate < = 90.0	Latitudes North of the Equator shall be specified by a plus (+) sign, preceding the two digits designating degrees. Latitudes south of the Equator shall be designated by a minus (-) sign .

APPENDIX A

<i>Element</i>	<i>Field</i>	<i>Definition</i>	<i>Short Name</i>	<i>Element Type</i>	<i>Domain</i>	<i>Format</i>
Keywords	Keywords (1.6)	Words or phrases summarizing an aspect of the data set.	keywords	Compound		
Theme Keyword Thesaurus	Theme_Keyword_Thesaurus (1.6.1.1)	Reference to a formally registered thesaurus or a similar authoritative source of theme keywords.	themekt	Text	"None"; Free Text	
Theme Keyword	Theme_Keyword (1.6.1.2)	Common-use word or phrase used to describe the subject of the data set.	themekey	Text	Free Text	
Place Keyword Thesaurus	Place_Keyword_Thesaurus (1.6.2.1)	Reference to a formally registered thesaurus or a similar authoritative source of place keywords.	placekt	Text	"None"; "Geographic Names Information System"; Free Text	
Place Keyword	Place_Keyword (1.6.2.2)	The geographic name of a location covered by a data set. (Includes city, county, state, state acronym, regional descriptions and references).	placekey	Text	Free Text	
Access Constraints	Access_Constraints (1.7)	Restrictions and legal prerequisites for accessing the data set. These include any access constraints applied to assure the protection of privacy or intellectual property, and any special restrictions or limitations on obtaining the data set.	accostnt	Text	"None"; Free Text	
Use Constraints	Use_Constraints (1.8)	Restrictions and legal prerequisites for using the data set after access is granted. These include any use constraints applied to assure the protection of privacy or intellectual property, and any special restrictions or limitations on using the data set.	useco	Text	"None"; Free Text	
Point of Contact	Point_of_Contact (1.9)	Contact information for an organization that is knowledgeable about the data set.	ptcontac	Compound	See Section 10.	
Security Information	Security_Information (1.12)	Handling restrictions imposed on the data set because of national security, privacy, or other concerns.	secinfo	Compound		
Security Classification System	Security_Classification_System (1.12.1)	Name of the classification system.	secsys	Text	Free Text	
Security Classification	Security_Classification (1.12.2)	Name of the handling restrictions on the data set.	secclass	Text	"Top secret"; "Secret"; "Confidential"; "Restricted"; "Unclassified"; "Sensitive"; "Free text"	
Security Handling Description	Security_Handling_Description (1.12.3)	Additional information about the restrictions on handling	sechandl	Text	Free Text	
Native Data Set Environment	Native_Data_Set_Environment (1.13)	A description of the data set, including the name of the software, computer operating system, file name, and data set size.	native	Text	Free Text	
Section 2: DATA QUALITY INFORMATION						
Logical Consistency Report	Logical_Consistency_Report (2.2)	An explanation of the fidelity of relationships in the data set and tests used.	logic	Text	Free Text	
Completeness Report	Completeness_Report (2.3)	Information about omissions, selection criteria, generalization, definitions used, and other rules used to derive the data set.	complete	Text	Free Text	
Lineage	Lineage (2.5)	Information about the events, parameters, and source data which constructed the data set, and information about the responsible parties.	lineage	Compound		
Process Step	Process_Step (2.5.2)	Information about a single event.	procstep	Compound		
Process Description	Process_Description (2.5.2.1)	An explanation of the event and related parameters or tolerances.	procdesc	Text	Free Text	
Process Date	Process_Date (2.5.2.3)	The date when the event was completed.	procdte	Date	Free Date	
Section 5: ENTITY AND ATTRIBUTES						
Overview Description	Overview_Description (5.2)	Description of the entities, attributes, attribute values, and related characteristics encoded.	overview	Compound		
Entity and Attribute Overview	Entity_and_Attribute_Overview (5.2.1)	Detailed summary of the information contained in a data set.	eaover	Text	Free Text	
Entity and Attribute Detail Citation	Entity_and_Attribute_Detail_Citation (5.2.2)	Reference used to the complete description of the entity types, attributes, and attribute values for the data set.	eadetclt	Text	Free Text	
Section 6: DISTRIBUTION INFORMATION						
Distribution Information	Distribution_Information (6)	Information about the distributor of the options for obtaining the data	distifo	Compound		
Distributor	Distributor (6.1)	The part from whom the data set may be obtained	distrib	Compound	See Section 10.	
Resource Description	Resource_Description (6.2)	The identifier by which the distributor knows the data set.	resdesc	Text	Free Text	
Distribution Liability	Distribution_Liability (6.3)	Statement of the liability assumed by the distributor.	distlab	Text	Free Text	
Custom Order Process	Custom_Order_Process (6.5)	Description of custom distribution services available, and the terms and conditions for obtaining these services.	custom	Text	Free Text	

APPENDIX A

<i>Element</i>	<i>Field</i>	<i>Definition</i>	<i>Short Name</i>	<i>Element Type</i>	<i>Domain</i>	<i>Format</i>
Section 7: METADATA REFERENCE						
Metadata Date	Metadata_Date (7.1)	The date that the metadata were created or last updated.	metd	Date	Free Date	YYYY for years; YYYYMM for month of year; YYYYMMDD for day of the year
Metadata Contact	Metadata_Contact (7.4)	The party responsible for the metadata information.	metc	Compound	See Section 10	
Metadata Standard Name	Metadata_Standard_Name (7.5)	The name of the metadata standard used to document the data set.	metstdn	Text	FGDC Content Standards for Digital Geospatial Metadata; Free Text	
Metadata Access Constraints	Metadata_Access_Constraints (7.8)	Restrictions and legal prerequisites for accessing the metadata. These include any access constraints applied to assure the protection of privacy or intellectual property, and any special restrictions or limitations on obtaining the metadata.	metac	Text	Free Text	
Metadata Use Constraints	Metadata_Use_Constraints (7.9)	Restrictions and legal prerequisites for using the metadata after access is granted. These include any metadata use constraints applied to assure the protection of privacy or intellectual property, and any special restrictions or limitations on using the metadata.	metuc	Text	None; Free Text	
Section 9: TIME PERIOD INFORMATION (Enter EITHER a single Date OR multiple dates)						
Single Date	Single_Date (9.1)	Means of encoding a single date and time.	sngdate	Compound		
Calendar Date	Calendar_Date (9.1.1)	The year (optionally month or month and day).	caldate	Date	Unknown; Free Date	YYYY for years; YYYYMM for month of year; YYYYMMDD for day of the year
Time of Day	Single_Time (9.1.2)	The hour (and optionally minute, or minute and second) of day.	time	Time		
OR						
Multiple Dates	Multiple_Dates (9.2)	Means of encoding multiple individual dates. (Complete if applicable).	mdattm	Compound		
Range of Dates	Range_of_Dates (9.3)	Means of encoding a range of dates. (Complete if applicable).	rngdates	Compound		
Beginning Date	Beginning_Date (9.3.1)	The first year (optionally month or month and day) of the event.	begdate	Date	Unknown; Free Date	YYYY for years; YYYYMM for month of year; YYYYMMDD for day of the year
Beginning Time	Beginning_Time (9.3.2)	The first hour (and optionally minute, or minute and second) of the day for the event.	begtime	Time		
Ending Date	End_Date (9.3.3)	The last year (and optionally month or month and day) for the event.	enddate	Date	Unknown; Present; Free Date	YYYY for years; YYYYMM for month of year; YYYYMMDD for day of the year
Ending Time	End_Time (9.3.4)	The last hour (and optionally minute, or minute and second) of the day for the event.	endtime	Time		
Section 10: CONTACT INFORMATION						
Contact Information	Contact_Information (10.0)	This section provides a means of identifying individuals and organizations, and is used by other sections of the metadata standard. This section is never used alone.	cntinfo	Compound		
Contact Organization	Contact_Organization (10.2)	The name of the organization.	cntorg	Text	Free Text	
Contact Position	Contact_Position (10.3)	Title of the individual. (Complete if applicable).	cntpos	Text	Free Text	
Contact Address	Contact_Address (10.4)	The address for the organization.	cntaddr	Compound		
Address Type	Address_Type (10.4.1)	Address type.	addrtype	Text	Mailing; Physical; Mailing and Physical; Free Text	
Address	Address (10.4.2)	Contact address for organization.	address	Text	Free Text	
City	City (10.4.3)	Contact address city.	city	Text	Free Text	
State or Province	State_or_Province (10.4.4)	Contact address state or province.	state	Text	Free Text	
Postal Code	Postal_Code (10.4.5)	Contact address Zip or postal code.	postal	Text	Free Text	
Country	Country (10.4.6)	Contact address country.	country	Text	Free Text	
Contact Telephone Number	Contact_Voice_Telephone (10.5)	The telephone number by which individuals can speak to the organization.	cntvoice	Text	Free Text	
Contact TDD/TTY Telephone	Contact_TDD/TTY_Telephone (10.6)	The telephone number by which hearing-impaired individuals can contact the organization. (Complete if applicable).	cntidd	Text	Free Text	
Contact Fax Number	Contact_Facsimile_Telephone (10.7)	The telephone number of a facsimile machine of the organization. (Complete if applicable).	cntfax	Text	Free Text	
Contact E-mail Address	Contact_Electronic_Mail_Address (10.8)	The address of the electronic mailbox of the organization. (Complete if applicable).	cntemail	Text	Free Text	
Hours of Service	Hours_of_Service (10.9)	Time period when individuals can speak to the organization. (Complete if applicable).	hours	Text	Free Text	
Contact Instructions	Contact_Instructions (10.10)	Supplemental instructions on how or when to contact the organization. (Complete if applicable).	cntinst	Text	Free Text	
Template is based on a subset of the Federal Geographic Data Committee's "Content Standard for Digital Geospatial Metadata" Version 2 - 1998. (FGDC-STD-001 June 1998). The complete FGDC standard can be viewed at www.fgdc.gov/metadata/constan.html *Areas in gray are descriptive headings. Data entry elements are in white.						

Appendix B: Keywords from ISO 19115

Topic Categories

Code Number	Topic	Description
001	farming	rearing of animals and/or cultivation of plants
002	biota	flora and/or fauna in natural environments
003	boundaries	legal land descriptions
004	Climatology, Meteorology, Atmosphere	processes and phenomena of the atmosphere
005	economy	economic activities, conditions, and employment
006	elevation	height above or below the earth's surface
007	environment	environmental resources, protection, and conservation
008	geoscientific information	information pertaining to the earth sciences
009	health	health, health services, human ecology, and safety
010	imagery, base maps, earth cover	base maps
011	intelligence, military	military bases, structures, activities
012	inland waters	inland water features, drainage systems and characteristics
013	location	positional information and services
014	oceans	features and characteristics of salt water bodies
015	planning, cadastre	information used for future use of the land
016	society	characteristics of society and culture
017	structure	man-made construction
018	transportation	means and aids for conveying persons and/or goods
019	utilities, communication	energy, water and waste systems, and communications infrastructure

CHANGE REQUEST AND TRACKING FORM

Use this form to identify and describe a problem encountered when using the metadata user guide, or to describe a requested change to the user guide. If you have encountered multiple problems or have multiple change requests, use a separate form for each problem or request.

YOUR NAME	
YOUR EMAIL	
YOUR PHONE	
YOUR OPERATING ENVIRONMENT	
DATE	

PLEASE DESCRIBE PROBLEM OR REQUESTED CHANGE (Give as much detail as possible, use additional pages as necessary. If this request is for the application, please describe your actions that resulted in the problem. Include other software you had open at the time):

EMAIL COMPLETED FORM TO: METADATA USER GUIDE CHANGE MANAGER

Email: ephtmetadata@cdc.gov.

CHANGE MANAGER (only)	
RECEIVED	
REVIEWED	
ACTION	
COMPLETED	

Appendix D

Missouri Code of State Regulations – 19 CSR 20.20
Reporting Communicable, Environmental, and Occupational Diseases

Rules of Department of Health and Senior Services

Division 20—Division of Community and Public Health Chapter 20—Communicable Diseases

Title	Page
19 CSR 20-20.010 Definitions Relating to Communicable, Environmental and Occupational Diseases	3
19 CSR 20-20.020 Reporting Communicable, Environmental and Occupational Diseases	5
19 CSR 20-20.030 Exclusion From School and Readmission	8
19 CSR 20-20.040 Measures for the Control of Communicable, Environmental and Occupational Diseases	8
19 CSR 20-20.050 Quarantine or Isolation Practices and Closing of Schools and Places of Public and Private Assembly	10
19 CSR 20-20.060 Control Measures for Food Handlers	10
19 CSR 20-20.070 Duties of Local Health Departments	10
19 CSR 20-20.075 Confidentiality of Information Obtained for Reporting of Communicable, Environmental and Occupational Diseases and Conditions	10
19 CSR 20-20.080 Duties of Laboratories	11
19 CSR 20-20.090 Contact With Communicable Diseases by First Responders or Emergency Medical Person and Mortuary Personnel	12
19 CSR 20-20.091 Testing for Contagious or Infectious Disease	12
19 CSR 20-20.092 Blood-Borne Pathogen Standard Required for Occupational Exposure of Public Employees to Blood and Other Infectious Materials	12
19 CSR 20-20.100 Tuberculosis Testing for Residents and Workers in Long-Term Care Facilities and State Correctional Centers	13



**Title 19—DEPARTMENT OF
HEALTH AND SENIOR SERVICES**
**Division 20—Division of Community
and Public Health**
Chapter 20—Communicable Diseases

**19 CSR 20-20.010 Definitions Relating to
Communicable, Environmental and Oc-
cupational Diseases**

PURPOSE: This rule defines terminology used throughout this chapter and defines terms related to infectious waste.

- (1) Administrator is the person in charge of an institution, such as the chief executive officer, chairperson of the board, administrator, clinician in charge, or any equivalent position.
- (2) Adult respiratory distress syndrome (ARDS) is a syndrome with the following simultaneous characteristics:
 (A) Hypoxemia due to intrapulmonary shunting of blood;
 (B) Increased lung stiffness; and
 (C) Chest x ray evidencing diffuse infiltration.
- (3) Board is the State Board of Health.
- (4) Carrier is a person who harbors a specific infectious agent in the absence of discernible clinical disease and serves as a potential source or reservoir of infection for man.
- (5) Case, as distinct from a carrier, is a person in whose tissues the etiologic agent of a communicable disease is present and which usually produces signs or symptoms of disease. Evidence of the presence of a communicable disease also may be revealed by routine laboratory findings.
- (6) Cluster is a group of individuals who manifest the same or similar signs and symptoms of disease.
- (7) Communicable disease is an illness due to an infectious agent or its toxic products and transmitted, directly or indirectly, to a susceptible host from an infected person, animal or arthropod, or through the agency of an intermediate host or a vector, or through the inanimate environment.
- (8) Contact is a person or animal that has been in association with an infected person or animal and through that association has had the opportunity to acquire the infection.
- (9) Designated representative is any person or group of persons appointed by the director of the Department of Health and Senior Services to act on behalf of the director or the State Board of Health.
- (10) Director is the state Department of Health and Senior Services director.
- (11) Disinfection is the killing of pathogenic agents outside the body by chemical or physical means, directly applied.
 (A) Concurrent disinfection is disinfection immediately after the discharge of infectious material from the body of an infected person or after the soiling of articles with the infectious discharges.
 (B) Terminal disinfection is the process of rendering the personal clothing and immediate physical environment of a patient free from the possibility of conveying the infection to others after the patient has left the premises or after the patient has ceased to be a source of infection or after isolation practices have been discontinued.
- (12) Environmental and occupational diseases are illnesses or adverse human health effects resulting from exposure to a chemical, radiological or physical agent.
- (13) Exposure is defined as contact with, absorption, ingestion or inhalation of chemical, biologic, radiologic, or other physical agents by a human that results in biochemical, physiological or histological changes.
- (14) Food is any raw, cooked or processed edible substance, ice, beverage or ingredient used or intended for use in whole or in part for human consumption.
- (15) Heat exhaustion means a reaction to excessive heat marked by prostration, weakness and collapse resulting from dehydration.
- (16) Heat stroke means a severe illness caused by exposure to excessively high temperatures and characterized by severe headache; high fever with a dry, hot skin; tachycardia; and in serious cases, collapse, coma or death.
- (17) Hyperthermia means a physician-diagnosed case of heat exhaustion or heat stroke.
- (18) Hypothermia means a physician-diagnosed case of cold injury associated with a fall of body temperature to less than ninety-four and one-tenth degrees Fahrenheit (94.1°F) and resulting from exposure to a cold environment.
- (19) Immediately reportable diseases are those diseases or findings listed in 19 CSR 20-20.020(1)(A)–(C) and shall be reported at once, without delay and with a sense of urgency by means of rapid communication to the Missouri Department of Health and Senior Services or to the local public health agency, regardless of the day or hour.
- (20) Immunization is a treatment which renders an individual less susceptible to the pathologic effects of a disease or provides a measure of protection against the disease.
- (21) Infectious waste is waste capable of producing an infectious disease. For a waste to be infectious, it must contain pathogens with sufficient virulence and quantity so that exposure to the waste by a susceptible host could result in an infectious disease. Infectious waste generated by small quantity generators shall include the following categories:
 (A) Sharps—all discarded sharps including hypodermic needles, syringes and scalpel blades. Broken glass or other sharp items that have come in contact with material defined as infectious are included;
 (B) Cultures and stocks of infectious agents and associated biologicals—included in this category are all cultures and stocks of infectious organisms as well as culture dishes and devices used to transfer, inoculate and mix cultures; and
 (C) Other wastes—those wastes designated by the medical authority responsible (physician, podiatrist, dentist, veterinarian) for the care of the patient which may be capable of producing an infectious disease.
- (22) Institution is any public or private hospital, nursing home, clinic, mental health facility, home health agency, or medical or professional corporation composed of health care workers.
- (23) Invasive disease is caused by a pathogen that invades the bloodstream and/or normally sterile bodily fluids and has the potential to cause severe morbidity and/or mortality. Culturing organisms from blood, cerebrospinal fluid, joint fluid, or pleural fluid identifies invasive diseases. Examples of conditions caused by invasive organisms include:
 (A) *Haemophilus influenzae*—meningitis, occult febrile bacteremia, epiglottitis, septic arthritis, pericarditis, abscesses, empyema, and osteomyelitis;
 (B) *Streptococcus pneumoniae*—bacteremia, and meningitis;



(C) *Neisseria meningitidis*—meningitis with or without meningococemia, septicemia (purpura fulminans), bacteremia, pericarditis, myocarditis, arthritis, and epididymitis;

(D) *Streptococcus pyogenes* (group A)—bacteremia associated with cutaneous infection, deep soft tissue infection (necrotizing fasciitis), meningitis, peritonitis, osteomyelitis, septic arthritis, postpartum sepsis, neonatal sepsis, and non-focal bacteremia.

(24) Isolation is the separation for the period of communicability of infected individuals and animals from other individuals and animals, in places and under conditions as will prevent the direct or indirect transmission of the infectious agent from infected individuals or animals to other individuals or animals who are susceptible or who may spread the agent to others.

(25) Laboratory means a facility for the biological, microbiological, serological, chemical, immuno-hematological, biophysical, cytological, pathological, or other examination of materials derived from the human body for the purpose of providing information for the diagnosis, prevention, or treatment of any disease or impairment of, or the assessment of the health of a human. These examinations also include procedures to determine, measure, or otherwise describe the presence or absence of various substances or organisms in the body. Facilities only collecting or preparing specimens (or both) or only serving as a mailing service and not performing testing are not considered laboratories. Laboratory includes hand-held testing equipment. All testing laboratories must be certified under the Clinical Laboratories Improvement Amendment of 1988 (CLIA—42 CFR part 493).

(26) Local health authority is the city or county health officer, director of an organized health department or of a local board of health within a given jurisdiction. In those counties where a local health authority does not exist, the health officer or administrator of the Department of Health and Senior Services district in which the county is located shall serve as a local health authority.

(27) Local public health agency is a legally constituted body provided by a city, county or group of counties to protect the public health of the city, county or group of counties.

(28) Methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci (VRE), and nosocomial infection are:

(A) MRSA shall be defined as *S. aureus* strains that are resistant to oxacillin, nafcillin and methicillin; historically termed MRSA. These organisms are resistant to all β -lactam agents, including cephalosporins and carbapenems. (NOTE: MRSA isolates are often resistant to other multiple, commonly used classes of antimicrobial agents, including erythromycin, clindamycin, and tetracycline.)

(B) VRE shall be defined as enterococci that possess intrinsic or acquired resistance to vancomycin. Several genes, including *vanA*, *vanB*, *vanC*, *vanD*, and *vanE*, contribute to resistance to vancomycin in enterococci.

(C) Nosocomial infection shall be defined by the national Centers for Disease Control and Prevention and applied to infections within hospitals, ambulatory surgical centers, and other facilities.

(29) Outbreak or epidemic is the occurrence in a community or region of an illness(es) similar in nature, clearly in excess of normal expectancy and derived from a common or a propagated source.

(30) Period of communicability is the period of time during which an etiologic agent may be transferred, directly or indirectly, from an infected person to another person or from an infected animal to a person.

(31) Person is any individual, partnership, corporation, association, institution, city, county, other political subdivision authority, state agency or institution or federal agency or institution.

(32) Pesticide poisoning means human disturbance of function, damage to structure or illness which results from the inhalation, absorption or ingestion of any pesticide.

(33) Poisoning means injury, illness or death caused by chemical means.

(34) Quarantine is a restriction of movement of persons or animals that have been exposed to a communicable disease, but have not yet developed disease. The period of quarantine will not be longer than the entire incubation period of the disease. The purpose of quarantine is to prevent effective contact with the general population.

(A) Complete quarantine is a limitation of freedom of movement of persons or animals exposed to a reportable disease, for a period of time not longer than the entire incubation period of the disease, in order to prevent effective contact with the general population.

(B) Modified quarantine is a selective, partial limitation of freedom of movement of per-

sons or animals determined on the basis of differences in susceptibility or danger of disease transmission. Modified quarantine is designed to meet particular situations and includes, but is not limited to, the exclusion of children from school, the closure of schools and places of public or private assembly and the prohibition or restriction of those exposed to a communicable disease from engaging in a particular occupation.

(35) Reportable disease is any disease or condition for which an official report is required. Any unusual expression of illness in a group of individuals which may be of public health concern is reportable and shall be reported to the local health department, local health authority or the Department of Health and Senior Services by the quickest means.

(36) Small quantity generator of infectious waste is any person generating one hundred kilograms (100 kg) or less of infectious waste per month and as regulated in 10 CSR 80.

(37) Statewide pandemic is an outbreak of a particularly dangerous disease affecting a high proportion of the population, appearing in three (3) or more counties, as declared by the director of the Department of Health and Senior Services.

(38) Terrorist event is the unlawful use of force or violence committed by a group or individual against persons or property to intimidate or coerce a government, the civilian population, or any segment thereof, in furtherance of political or social objectives. Terrorist attacks are classified as chemical, biological, or radiological.

(A) Chemical means any weapon that is designed or intended to cause widespread death or serious bodily injury through the release, dissemination, or impact of toxic or poisonous chemicals or precursors of toxic or poisonous chemicals.

(B) Biological means any microorganism, virus, infectious substance, or biological product that may be engineered as a result of biotechnology, or any naturally occurring or bioengineered component of any such microorganism, virus, infectious substance, or biological product.

(C) Radiological means any weapon that is designed to release radiation or radioactivity at a level dangerous to human life.

(39) Toxic substance is any substance, including any raw materials, intermediate products, catalysts, final products or by-products of any manufacturing operation conducted in a commercial establishment that has the capacity



through its physical, chemical or biological properties to pose a substantial risk of death or impairment, either immediately or later, to the normal functions of humans, aquatic organisms or any other animal.

(40) Unusual diseases—Examples include, but are not limited to, the following:

- (A) Diseases uncommon to a geographic area, age group, or anatomic site;
- (B) Cases of violent illness resulting in respiratory failure;
- (C) Absence of a competent natural vector for a disease; or
- (D) Occurrence of hemorrhagic illness.

(41) Unusual manifestation of illness—Examples include, but are not limited to, the following:

- (A) Multiple persons presenting with a similar clinical syndrome at a steady or increasing rate;
- (B) Large numbers of rapidly fatal cases, with or without recognizable signs and symptoms;
- (C) Two (2) or more persons, without a previous medical history, presenting with convulsions;
- (D) Persons presenting with grayish colored tissue damage; or
- (E) Adults under the age of fifty (50) years, without previous medical history, presenting with adult respiratory distress syndrome (ARDS).

(42) Varicella (Chickenpox) severity of illness shall include the following categories:

- (A) Mild—less than fifty (50) lesions (able to count lesions within thirty (30) seconds);
- (B) Moderate—fifty to five hundred (50–500) lesions (anything in between mild and severe); and
- (C) Severe—more than five hundred (500) lesions (difficult to see the skin) or lesions with complications.

AUTHORITY: sections 192.006 and 260.203, RSMo 2000 and 192.020, RSMo Supp. 2006. This rule was previously filed as 13 CSR 50-101.010. Original rule filed July 15, 1948, effective Sept. 13, 1948. Rescinded and readopted: Filed Dec. 11, 1981, effective May 13, 1982. Amended: Filed Aug. 16, 1988, effective Dec. 29, 1988. Amended: Filed Aug. 14, 1992, effective April 8, 1993. Amended: Filed Sept. 15, 1995, effective April 30, 1996. Emergency amendment filed June 1, 2000, effective June 15, 2000, expired Dec. 11, 2000. Amended: Filed June 1, 2000, effective Nov. 30, 2000. Amended: Filed Oct. 1, 2004, effective April 30, 2005. Amended: Filed Feb. 15, 2006, effective Sept.*

30, 2006. Emergency amendment filed June 15, 2007, effective July 6, 2007, expired Jan. 1, 2008. Amended: Filed June 15, 2007, effective Jan. 30, 2008.

**Original authority: 192.006, RSMo 1993, amended 1995; 192.020, RSMo 1939, amended 1945, 1951, 2004; and 260.203, RSMo 1986, amended 1988, 1992, 1993.*

19 CSR 20-20.020 Reporting Communicable, Environmental and Occupational Diseases

PURPOSE: This rule designates the diseases, disabilities, conditions and findings that must be reported to the local health authority or the Department of Health and Senior Services. It also establishes when they must be reported.

PUBLISHER'S NOTE: The secretary of state has determined that the publication of the entire text of the material which is incorporated by reference as a portion of this rule would be unduly cumbersome or expensive. This material as incorporated by reference in this rule shall be maintained by the agency at its headquarters and shall be made available to the public for inspection and copying at no more than the actual cost of reproduction. This note applies only to the reference material. The entire text of the rule is printed here.

(1) The diseases within the immediately reportable disease category pose a risk to national security because they: can be easily disseminated or transmitted from person to person; result in high mortality rates and have the potential for major public health impact; might cause public panic and social disruption; and require special action for public health preparedness. Immediately reportable diseases or findings shall be reported to the local health authority or to the Department of Health and Senior Services immediately upon knowledge or suspicion by telephone (1 (800) 392-0272), facsimile or other rapid communication. Immediately reportable diseases or findings are—

(A) Selected high priority diseases, findings or agents that occur naturally, from accidental exposure, or as the result of a bioterrorism event:

- Anthrax
- Botulism
- Plague
- Rabies (Human)
- Ricin toxin
- Severe Acute Respiratory syndrome-associated Coronavirus (SARS-CoV) Disease
- Smallpox

Tularemia (pneumonic)

Viral hemorrhagic fevers (filoviruses (e.g., Ebola, Marburg) and arenaviruses (e.g., Lassa, Machupo))

(B) Instances, clusters, or outbreaks of unusual diseases or manifestations of illness and clusters or instances of unexplained deaths which appear to be a result of a terrorist act or the intentional or deliberate release of biological, chemical, radiological, or physical agents, including exposures through food, water, or air.

(C) Instances, clusters, or outbreaks of unusual, novel, and/or emerging diseases or findings not otherwise named in this rule, appearing to be naturally occurring, but posing a substantial risk to public health and/or social and economic stability due to their ease of dissemination or transmittal, associated mortality rates, or the need for special public health actions to control.

(2) Reportable within one (1) day diseases or findings shall be reported to the local health authority or to the Department of Health and Senior Services within one (1) calendar day of first knowledge or suspicion by telephone, facsimile or other rapid communication. Reportable within one (1) day diseases or findings are—

(A) Diseases, findings or agents that occur naturally, or from accidental exposure, or as a result of an undetected bioterrorism event:

Acute respiratory distress syndrome (ARDS) in patients under fifty (50) years of age (without a contributing medical history)

- Animal (mammal) bite, wound, humans
- Brucellosis
- Cholera
- Dengue fever
- Diphtheria
- Glanders
- Haemophilus influenzae*, invasive disease
- Hantavirus pulmonary syndrome
- Hemolytic uremic syndrome (HUS), post-diarrheal
- Hepatitis A
- Influenza-associated pediatric mortality (eighteen (18) years of age or younger)
- Influenza-associated public and/or private school closures
- Lead (blood) level greater than or equal to forty-five micrograms per deciliter (≥ 45 $\mu\text{g}/\text{dl}$) in any person equal to or less than seventy-two (≤ 72) months of age
- Measles (rubeola)
- Meningococcal disease, invasive
- Novel Influenza A virus infections, human
- Outbreaks (including nosocomial) or epidemics of any illness, disease or condition that may be of public health concern, including any illness in a food



- handler that is potentially transmissible through food
- Pertussis
- Poliomyelitis
- Poliovirus infection, nonparalytic
- Q fever
- Rabies (animal)
- Rubella, including congenital syndrome
- Shiga toxin-producing *Escherichia coli* (STEC)
- Shiga toxin positive, unknown organism
- Shigellosis
- Staphylococcal enterotoxin B
- Streptococcus pneumoniae*, drug resistant invasive disease
- Syphilis, including congenital syphilis
- T-2 mycotoxin
- Tetanus
- Tuberculosis disease
- Tularemia (non-pneumonic)
- Typhoid fever (*Salmonella typhi*)
- Vancomycin-intermediate *Staphylococcus aureus* (VISA), and Vancomycin-resistant *Staphylococcus aureus* (VRSA)
- Venezuelan equine encephalitis virus neuroinvasive disease
- Venezuelan equine encephalitis virus non-neuroinvasive disease
- Yellow fever
- (B) Diseases, findings or adverse reactions that occur as a result of inoculation to prevent smallpox, including but not limited to the following:
 - Accidental administration
 - Contact transmission (i.e., vaccinia virus infection in a contact of a smallpox vaccinee)
 - Eczema vaccinatum
 - Erythema multiforme (roseola vaccinia, toxic urticaria)
 - Fetal vaccinia (congenital vaccinia)
 - Generalized vaccinia
 - Inadvertent autoinoculation (accidental implantation)
 - Myocarditis, pericarditis, or myopericarditis
 - Ocular vaccinia (can include keratitis, conjunctivitis, or blepharitis)
 - Post-vaccinial encephalitis or encephalomyelitis
 - Progressive vaccinia (vaccinia necrosum, vaccinia gangrenosa, disseminated vaccinia)
 - Pyogenic infection of the vaccination site
 - Stevens-Johnson Syndrome
- (3) Reportable within three (3) days diseases or findings shall be reported to the local health authority or the Department of Health and Senior Services within three (3) calendar days of first knowledge or suspicion. These diseases or findings are—
 - Acquired immunodeficiency syndrome (AIDS)
 - Arsenic poisoning
 - California serogroup virus neuroinvasive disease
 - California serogroup virus non-neuroinvasive disease
 - Campylobacteriosis
 - Carbon monoxide poisoning
 - CD4+ T cell count
 - Chancroid
 - Chemical poisoning, acute, as defined in the most current ATSDR CERCLA Priority List of Hazardous Substances; if terrorism is suspected, refer to subsection (1)(B)
 - Chlamydia trachomatis*, infections
 - Coccidioidomycosis
 - Creutzfeldt-Jakob disease
 - Cryptosporidiosis
 - Cyclosporiasis
 - Eastern equine encephalitis virus neuroinvasive disease
 - Eastern equine encephalitis virus non-neuroinvasive disease
 - Ehrlichiosis, human granulocytic, monocytic, or other/unspecified agent
 - Giardiasis
 - Gonorrhea
 - Hansen’s disease (Leprosy)
 - Heavy metal poisoning including, but not limited to, cadmium and mercury
 - Hepatitis B, acute
 - Hepatitis B, chronic
 - Hepatitis B surface antigen (prenatal HBsAg) in pregnant women
 - Hepatitis B Virus Infection, perinatal (HBsAg positivity in any infant aged equal to or less than twenty-four (≤24) months who was born to an HBsAg-positive mother)
 - Hepatitis C, acute
 - Hepatitis C, chronic
 - Hepatitis non-A, non-B, non-C
 - Human immunodeficiency virus (HIV)-exposed newborn infant (i.e., newborn infant whose mother is infected with HIV)
 - Human immunodeficiency virus (HIV) infection, as indicated by HIV antibody testing (reactive screening test followed by a positive confirmatory test), HIV antigen testing (reactive screening test followed by a positive confirmatory test), detection of HIV nucleic acid (RNA or DNA), HIV viral culture, or other testing that indicates HIV infection
 - Human immunodeficiency virus (HIV) test results (including both positive and negative results) for children less than two (2) years of age whose mothers are infected with HIV
 - Human immunodeficiency virus (HIV) viral load measurement (including non-detectable results)
 - Hyperthermia
 - Hypothermia
 - Lead (blood) level less than forty-five micrograms per deciliter (<45 µg/dl) in any person equal to or less than seventy-two (≤72) months of age and any lead (blood) level in persons older than seventy-two (>72) months of age
 - Legionellosis
 - Leptospirosis
 - Listeriosis
 - Lyme disease
 - Malaria
 - Methemoglobinemia, environmentally-induced
 - Mumps
 - Mycobacterial disease other than tuberculosis (MOTT)
 - Occupational lung diseases including silicosis, asbestosis, byssinosis, farmer’s lung and toxic organic dust syndrome
 - Pesticide poisoning
 - Powassan virus neuroinvasive disease
 - Powassan virus non-neuroinvasive disease
 - Psittacosis
 - Rabies Post-Exposure Prophylaxis (Initiated)
 - Respiratory diseases triggered by environmental contaminants including environmentally or occupationally induced asthma and bronchitis
 - Rocky Mountain spotted fever
 - Saint Louis encephalitis/virus neuroinvasive disease
 - Saint Louis encephalitis virus non-neuroinvasive disease
 - Salmonellosis
 - Streptococcal disease, invasive, Group A *Streptococcus pneumoniae*, invasive in children less than five (5) years
 - Toxic shock syndrome, staphylococcal or streptococcal
 - Trichinellosis
 - Tuberculosis infection
 - Varicella (Chickenpox)
 - Varicella deaths
 - Vibriosis (non-cholera *Vibrio* species infections)
 - West Nile virus neuroinvasive disease
 - West Nile virus non-neuroinvasive disease
 - Western equine encephalitis virus neuroinvasive disease
 - Western equine encephalitis virus non-neuroinvasive disease
 - Yersiniosis
- (4) Reportable weekly diseases or findings shall be reported directly to the Department of Health and Senior Services weekly. These diseases or findings are:
 - Influenza, laboratory-confirmed



(5) Reportable quarterly diseases or findings shall be reported directly to the Department of Health and Senior Services quarterly. These diseases or findings are:

- Methicillin-resistant *Staphylococcus aureus* (MRSA), nosocomial
- Vancomycin-resistant enterococci (VRE), nosocomial

(6) A physician, physician's assistant, nurse, hospital, clinic, or other private or public institution providing diagnostic testing, screening or care to any person with any disease, condition or finding listed in sections (1)–(4) of this rule or who is suspected of having any of these diseases, conditions or findings, shall make a case report to the local health authority or the Department of Health and Senior Services, or cause a case report to be made by their designee, within the specified time.

(A) A physician, physician's assistant, or nurse providing care in an institution to any patient with any disease, condition or finding listed in sections (1)–(4) of this rule may authorize, in writing, the administrator or designee of the institution to submit case reports on patients attended by the physician, physician's assistant, or nurse at the institution. But under no other circumstances shall the physician, physician's assistant, or nurse be relieved of this reporting responsibility.

(B) Duplicate reporting of the same case by health care providers in the same institution is not required.

(7) Except for influenza, laboratory-confirmed and Varicella (Chickenpox); a case report as required in section (6) of this rule shall include the patient's name, home address with zip code, date of birth, age, sex, race, home phone number, name of disease, condition or finding diagnosed or suspected, the date of onset of the illness, name and address of the treating facility (if any) and the attending physician, any appropriate laboratory results, name and address of the reporter, treatment information for sexually transmitted diseases, and the date of report.

(A) A report of an outbreak or epidemic as required in subsections (1)(B) and (1)(C) of this rule shall include the diagnosis or principal symptoms, the approximate number of cases, the local health authority jurisdiction within which the cases occurred, the identity of any cases known to the reporter, and the name and address of the reporter.

(B) Influenza, laboratory-confirmed reporting as required in section (4) of this rule shall include the patient's age group (i.e., 0–4, 5–24, 25–64, and 65+ years) and serology/serotype (i.e., A, B, and unknown),

the local health authority jurisdiction within which the cases occurred, and the date of report. Aggregate patient data shall be reported weekly.

(C) Varicella (Chickenpox) reporting as required in section (3) of this rule shall include the patient's name, date of birth, vaccination history, and severity of illness; the local health authority jurisdiction within which the cases occurred, and the date of report.

(8) Any person in charge of a public or private school, summer camp or child or adult care facility shall report to the local health authority or the Department of Health and Senior Services the presence or suspected presence of any diseases or findings listed in sections (1)–(4) of this rule according to the specified time frames.

(9) All local health authorities shall forward to the Department of Health and Senior Services reports of all diseases or findings listed in sections (1)–(4) of this rule. All reports shall be forwarded according to procedures established by the Department of Health and Senior Services director as listed in sections (1)–(4). Reports will be forwarded immediately if a terrorist event is suspected or confirmed. The local health authority shall retain from the original report any information necessary to carry out the required duties in 19 CSR 20-20.040(2) and (3).

(10) Information from patient medical records received by local public health agencies or the Department of Health and Senior Services in compliance with this rule is to be considered confidential records and not public records.

(11) Reporters specified in section (6) of this rule will not be held liable for reports made in good faith in compliance with this rule.

(12) The following material is incorporated into this rule by reference:

(A) 2005 Agency for Toxic Substances and Disease Registry (ATSDR) 1825 Century Blvd., Atlanta, GA 30345, Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) Priority List of Hazardous Substances, available at: <http://www.atsdr.cdc.gov/cercla>. This rule does not incorporate any subsequent amendments or additions.

(13) Each hospital and ambulatory surgical center shall report on a quarterly basis antibiogram data for infection, not colonization, from all body sites monitored by that health

care facility. Antibiogram data to be reported shall include nosocomial methicillin sensitive *Staphylococcus aureus* (*S. aureus*), nosocomial *S. aureus*, nosocomial vancomycin sensitive enterococci, and nosocomial enterococci isolates. Data shall be reported directly to the Department of Health and Senior Services. Reporting shall include only a patient's first diagnostic nosocomial isolate per admission of *Staphylococcus aureus* (*S. aureus*) and enterococci and the isolates corresponding methicillin or vancomycin sensitivity; irrespective of location or of other antimicrobial sensitivity(ies). Intermediate methicillin or vancomycin sensitivity shall be reported as resistant (i.e., methicillin-resistant *Staphylococcus aureus* (MRSA) or vancomycin-resistant enterococci (VRE), respectively).

(A) Isolates from cultures performed for routine surveillance purposes are excluded from the requirement to report. Methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant enterococci (VRE) nosocomial infections to be reported to the Department of Health and Senior Services are limited to those body sites monitored by the individual hospital or ambulatory surgical center.

(B) Aggregate antibiogram data for patients' non-duplicative isolates, per admission, of nosocomial MRSA and VRE infections shall reflect susceptibility patterns and shall be reported as the:

1. Number of nosocomial isolates of *S. aureus* sensitive to methicillin (oxacillin, etc.);
2. Number of nosocomial isolates *S. aureus*;
3. Number of nosocomial isolates of enterococci sensitive to vancomycin; and
4. Number of nosocomial isolates enterococci.

(C) Aggregate data shall be reported for the quarters January–March, April–June, July–September, and October–December within ten (10) days of the end of the quarter. Each quarter's aggregate report shall include only those data that are available within a ten (10)-day reporting period from the end of that quarter.

AUTHORITY: sections 192.006, 210.040, and 210.050, RSMo 2000 and section 192.020, RSMo Supp. 2007. This rule was previously filed as 13 CSR 50-101.020. Original rule filed July 15, 1948, effective Sept. 13, 1948. Amended: Filed Sept. 1, 1981, effective Dec. 11, 1981. Rescinded and readopted: Filed Nov. 23, 1982, effective March 11, 1983. Emergency amendment filed June 10, 1983, effective June 20, 1983, expired Sept. 10,*



1983. Amended: Filed June 10, 1983, effective Sept. 11, 1983. Amended: Filed Nov. 4, 1985, effective March 24, 1986. Amended: Filed Aug. 4, 1986, effective Oct. 11, 1986. Amended: Filed June 3, 1987, effective Oct. 25, 1987. Emergency amendment filed June 16, 1989, effective June 26, 1989, expired Oct. 23, 1989. Amended: Filed July 18, 1989, effective Sept. 28, 1989. Amended: Filed Nov. 2, 1990, effective March 14, 1991. Emergency amendment filed Oct. 2, 1991, effective Oct. 12, 1991, expired Feb. 8, 1992. Amended: Filed Oct. 2, 1991, effective Feb. 6, 1992. Amended: Filed Jan. 31, 1992, effective June 25, 1992. Amended: Filed Aug. 14, 1992, effective April 8, 1993. Amended: Filed Sept. 15, 1994, effective March 30, 1995. Amended: Filed Sept. 15, 1995, effective April 30, 1996. Emergency amendment filed June 1, 2000, effective June 15, 2000, expired Dec. 11, 2000. Amended: Filed June 1, 2000, effective Nov. 30, 2000. Emergency amendment filed Dec. 16, 2002, effective Dec. 26, 2002, expired June 23, 2003. Amended: Filed Dec. 16, 2002, effective June 30, 2003. Amended: Filed Oct. 1, 2004, effective April 30, 2005. Amended: Filed Feb. 15, 2006, effective Sept. 30, 2006. Amended: Filed Nov. 15, 2007, effective May 30, 2008.

*Original authority: 192.006, RSMo 1993, amended 1995; 192.020, RSMo 1939, amended 1945, 1951, 2004; 210.040, RSMo 1941, amended 1993; and 210.050, RSMo 1941, amended 1993.

19 CSR 20-20.030 Exclusion From School and Readmission

PURPOSE: This rule requires the exclusion of persons from school who have a reportable disease or who are liable to transmit a reportable disease. The methods of readmission to school are also established.

Editor's Note: The secretary of state has determined that the publication of this rule in its entirety would be unduly cumbersome or expensive. The entire text of the material referenced has been filed with the secretary of state. This material may be found at the Office of the Secretary of State or at the headquarters of the agency and is available to any interested person at a cost established by state law.

(1) Persons suffering from a reportable disease or who are liable to transmit a reportable disease listed in 19 CSR 20-20.020(1)-(3) shall be barred from attending school.

(2) Any person excluded from school under section (1) of this rule may be readmitted to school by one (1) of the following methods:

(A) Certification in writing by an attending physician attesting to the person's noninfectiousness;

(B) After a period of time equal to the longest period of communicability of the disease as established in the 1990 fifteenth edition of the *Control of Communicable Diseases in Man* published by the American Public Health Association; the 1991 twenty-second edition of the *Report of the Committee on Infectious Diseases* published by the American Academy of Pediatrics; or the following recommendations of the Immunization Practices Advisory Committee published by the Centers for Disease Control in the *Morbidity and Mortality Weekly Report: General Recommendations on Immunization*, April 7, 1989; *Update on Adult Immunization*, November 15, 1991; *New Recommended Schedule for Active Immunization of Normal Infants and Children*, September 19, 1986; *Pertussis Vaccination: Acellular Pertussis Vaccine for Reinforcing and Booster Use—Supplementary ACIP Statement*, February 7, 1992; *Diphtheria, Tetanus and Pertussis: Recommendations for Vaccine Use and Other Preventive Measures*, August 8, 1991; *Haemophilus b Conjugate Vaccines for Prevention of Haemophilus influenzae Type b Disease Among Infants and Children Two Months of Age and Older*, January 11, 1991; *Immunization of Children Infected With Human Immunodeficiency Virus—Supplementary ACIP Statement*, April 1, 1988; *Immunization of Children Infected with Human T-Lymphotropic Virus Type III/Lymphadenopathy-Associated Virus*, September 26, 1986; *Prevention and Control of Influenza*, May 15, 1992; *Measles Prevention: Recommendations of the Immunization Practices Advisory Committee (ACIP)*, December 29, 1989; *Meningococcal Vaccines*, May 10, 1985; *Mumps Prevention*, June 9, 1989; *Pneumococcal Polysaccharide Vaccine*, February 10, 1989; *Poliomyelitis Prevention: Enhanced-Potency Inactivated Poliomyelitis Vaccine Supplementary—Statement*, December 11, 1987; *Poliomyelitis Prevention*, January 29, 1982; *Rabies Prevention*, March 22, 1991; *Rubella Prevention*, November 23, 1990; *Varicella-Zoster Immune Globulin for the Prevention of Chickenpox*, February 24, 1984; *Hepatitis B Virus: A Comprehensive Strategy for Eliminating Transmission in the United States Through Universal Childhood Vaccination*, November 22, 1991; *Plague Vaccine*, June 11, 1982; *Typhoid Immunization*, July 13, 1990; *Typhus Vaccine*, June 2, 1978; and *Yellow Fever Vaccine*, May 4, 1990; or

(C) When the local health authority declares that the designated health emergency

is ended, after consultation and concurrence of the director of the Department of Health or his/her designated representative.

AUTHORITY: sections 192.005.2. and 192.020, RSMo 1994.* This rule was previously filed as 13 CSR 50-101.041. Original rule filed Dec. 11, 1981, effective May 13, 1982. Amended: Filed Sept. 16, 1982, effective Jan. 14, 1983. Amended: Filed Aug. 4, 1986, effective Oct. 11, 1986. Amended: Filed April 4, 1988, effective June 27, 1988. Emergency amendment filed Jan. 13, 1989, effective Jan. 23, 1989, expired May 22, 1989. Amended: Filed Jan. 13, 1989, effective May 11, 1989. Amended: Filed Oct. 3, 1989, effective Feb. 25, 1990. Amended: Filed Nov. 2, 1990, effective March 14, 1991. Amended: Filed July 12, 1991, effective Oct. 31, 1991. Amended: Filed Aug. 14, 1992, effective Feb. 26, 1993.

*Original authority: 192.005.2., RSMo 1985, amended 1993 and 192.020, RSMo 1939, amended 1945, 1951.

19 CSR 20-20.040 Measures for the Control of Communicable, Environmental and Occupational Diseases

PURPOSE: This rule defines investigative and control measures for reportable diseases and establishes who is responsible for them.

Editor's Note: The following material is incorporated into this rule by reference:

1) **Morbidity and Mortality Weekly Report** (Atlanta: Centers for Disease Control).

In accordance with section 536.031(4), RSMo, the full text of material incorporated by reference will be made available to any interested person at the Office of the Secretary of State and the headquarters of the adopting state agency.

(1) In controlling the diseases and findings listed in 19 CSR 20-20.020, the director shall comply with the methods of control section of one (1) of the two (2) books listed in 19 CSR 20-20.030(2)(B) or the recommendations of the Immunization Practices Advisory Committee (ACIP) published by the Centers for Disease Control in the *Morbidity and Mortality Weekly Report* listed in 19 CSR 20-20.030(2)(B). The director shall use the legal means necessary to control, investigate, or both, any disease or condition listed in 19 CSR 20-20.020 which is a threat to the public health.

(2) It shall be the duty of the local health authority, the director of the Department of



Health or the director's designated representative on receiving a report of a communicable, environmental or occupational disease to—

(A) Inspect any premises that they have reasonable grounds to believe are in a condition conducive to the spread of any communicable disease;

(B) Confer with the physician, laboratory or person making the report;

(C) Collect for laboratory analysis any samples or specimens that may be necessary to confirm the diagnosis or presence of the disease or biological, chemical or physical agents and to determine the source of the infection, epidemic or exposure. Health program representatives and other personnel employed by the Department of Health, after training and certification to perform venipuncture, and after specific authorization from a physician, are authorized to perform venipuncture utilizing procedures within the scope of the training they have been given. The content and scope of this training shall be established by the Department of Health. Training shall be provided by a physician or his/her designee and the certificate shall be signed by the physician. Nothing in this rule shall limit the authority of local public health departments to establish their own training policies, with or without certification, or to limit their voluntary participation in the certification program developed by the Department of Health, nor shall it apply to venipuncture for other purposes;

(D) Make a complete epidemiological, environmental or occupational industrial hygiene investigation and record of the findings on a communicable disease or exposure report form;

(E) Establish and maintain quarantine, isolation or other measures as required;

(F) Provide the opportunity to be immunized to all contacts of persons suffering from those diseases for which there is a reliable and approved means of immunization;

(G) Establish appropriate control measures which may include isolation, quarantine, disinfection, immunization, closure of establishment and other measures considered appropriate by medical experts for the protection of public health;

(H) Establish, as the local health authority, whenever a case of unrecognized illness is reported or otherwise brought to the attention of the local health authority or the Department of Health and investigation presents symptoms of a communicable disease, but sufficient time has not elapsed to render a positive diagnosis, after consultation with the director or his/her designated representative, the control measures applicable in actual

cases of the suspected communicable disease, until a positive diagnosis can be established. If a disease proves to be noncommunicable, the temporary control measures shall be terminated at once;

(I) Assume direct responsibility as director of health to make necessary investigation and immediately institute appropriate control measures necessary for the protection of the public health in occurrence of outbreaks or unusual clusters of illness involving more than one (1) county or a general regional area; and

(J) Investigate, as the local health authority, the disease within the local jurisdiction with assistance from the director of the Department of Health or his/her designated representative when any outbreak or unusual occurrence of a reportable disease is identified through reports required by 19 CSR 20-20.020. If, in the judgment of the director, the disease outbreak or unusual occurrence constitutes a medical emergency, the director may assume direct responsibility for the investigation.

(3) It shall be the duty of the local health authority, upon identification of a case of a reportable disease or upon receipt of a report of that disease, to take actions and measures as may be necessary according to any policies which have been or may be established by the director of the Department of Health, within the provisions of section (2) and subsections (2)(A)–(J) of this rule.

(A) When the local health authority is notified of a reportable disease or has reason to suspect the existence of a reportable disease within the local jurisdiction, the local health authority, either in person or through a designated representative, shall make an investigation as is necessary and immediately institute appropriate control measures as set forth in section (2) and subsections (2)(A)–(J) of this rule.

(B) The local health authority shall use every reasonable means to determine the presence of a communicable disease or the source of any disease listed in 19 CSR 20-20.020 or of any epidemic disease of unknown cause. In the performance of this duty, the local health authority shall examine or cause to be examined any person reasonably suspected of being infected or of being a source or contact of infection and any person who refuses examination shall be quarantined or isolated.

(C) Control measures implemented by the local health authority shall be at least as stringent as those established by the director of the Department of Health and shall be subject to review and alteration by the director. If the

local health authority fails to carry out appropriate control measures, the director or his/her designated representative shall take steps necessary to protect the public health.

(4) It shall be the duty of the attending physician, immediately upon diagnosing a case of a reportable communicable disease, to give detailed instructions to the patient, members of the household and attendants regarding proper control measures. When a person dies while infected with a communicable disease, it shall be the duty of the attending physician to learn immediately who is to prepare the body for burial or cremation and then notify the funeral director, embalmer or other responsible person regarding the communicable disease the deceased had at the time of death. A tag shall also be affixed to the body providing the name of the communicable disease likely to have been present at the time of death.

(5) Every practitioner of the healing arts and every person in charge of any medical care facility shall permit the director of the Department of Health or the director's designated representative to examine and review any medical records which are in the practitioner's or person's possession or to which the practitioner or person has access, upon request of the director or the director's designated representative in the course of investigation of reportable diseases in 19 CSR 20-20.020.

AUTHORITY: sections 192.006 and 192.020, RSMo 2000. This rule was previously filed as 13 CSR 50-101.050. Original rule filed July 15, 1948, effective Sept. 13, 1948. Rescinded and readopted: Filed Dec. 11, 1981, effective May 13, 1982. Amended: Filed Sept. 16, 1982, effective Jan. 14, 1983. Amended: Filed March 21, 1984, effective July 15, 1984. Amended: Filed June 2, 1988, effective Aug. 25, 1988. Amended: Filed Nov. 15, 1989, effective Feb. 11, 1990. Amended: Filed Aug. 14, 1992, effective April 8, 1993. Amended: Filed Sept. 15, 1995, effective April 30, 1996. Emergency amendment filed June 13, 2002, effective July 1, 2002, expires Dec. 27, 2002. Amended: Filed June 13, 2002, effective Nov. 30, 2002.*

**Original authority: 192.006.1., RSMo 1993, amended 1995 and 192.020, RSMo 1939, amended 1945, 1951.*



19 CSR 20-20.050 Quarantine or Isolation Practices and Closing of Schools and Places of Public and Private Assembly

PURPOSE: This rule provides for the isolation or quarantine of persons and animals with a communicable disease and their contacts; it also authorizes the closing of schools and places of public and private assembly.

(1) The local health authority, the director of the Department of Health and Senior Services or the director's designated representative shall require isolation of a patient or animal with a communicable disease, quarantine of contacts, concurrent and terminal disinfection, or modified forms of these procedures necessary for the protection of the public health. The isolation of a patient, animal or contact shall be carried out according to the methods of control in 19 CSR 20-20.040(1).

(2) No person or animal infected with or suspected of having a communicable disease listed in 19 CSR 20-20.020(1)–(3) or any contact of a disease subject to quarantine or isolation shall move or be moved from one (1) health jurisdiction to another, unless necessary for medical care, without notice to and consent from the local health authority, the director of the Department of Health and Senior Services or the director's designated representative. If a person is moved for the reason of medical care, the health authority who ordered the isolation or quarantine shall be notified within seventy-two (72) hours.

(3) The local health authority, the director of the Department of Health and Senior Services or the director's designated representative is empowered to close any public or private school or other place of public or private assembly when, in the opinion of the local health authority, the director of the Department of Health and Senior Services or the director's designated representative, the closing is necessary to protect the public health. However, in a statewide pandemic, only the director of the Department of Health and Senior Services or the director's designated representative shall have the authority to close a public or private school or other place of public or private assembly. The director or designated representative shall consult with the local health authorities prior to any such closing. Any school or other place of public or private assembly that is ordered closed shall not reopen until permitted by whomever ordered the closure.

AUTHORITY: section 192.020, RSMo Supp. 2006. This rule was previously filed as 13 CSR 50-101.061. Original rule filed Dec. 11,*

1981, effective May 13, 1982. Emergency amendment filed June 15, 2007, effective July 6, 2007, expired Jan. 1, 2008. Amended: Filed June 15, 2007, effective Jan. 30, 2008.

**Original authority: 192.020, RSMo 1939, amended 1945, 1951, 2004.*

19 CSR 20-20.060 Control Measures for Food Handlers

PURPOSE: This rule establishes control measures for persons working with food products who are suspected of having a communicable disease.

(1) For the purpose of this rule, a communicable disease is defined as a disease transmitted through handling food.

(2) No person infected with a communicable disease, whether actively infected or a chronic carrier, and no person with any one (1) of the signs and symptoms listed in this section, shall engage in the production, preparation, manufacture, packaging, storage, sale, distribution or transportation of food. The following signs and symptoms indicate infection with a foodborne pathogen: diarrhea, vomiting, open skin sores, boils, fever, dark urine or jaundice, unless determined not to be caused by a pathogen able to be transmitted by food. The local health authority, the director of the Department of Health or the director's designated representative may order examinations necessary to determine the presence of a foodborne infection.

(3) Notice shall be sent immediately to the local health authority, to the director of the Department of Health or to the director's designated representative by any person responsible for the production, preparation, manufacture, packaging, storage, sale, distribution or transportation of food if any infection or disease known to be transmissible through food occurs on the premises or among the employees.

(4) When the possibility of transmission of infection is suspected in any person engaged in the production, preparation, manufacture, packaging, storage, sale, distribution or transportation of food; the local health authority, the director of the Department of Health or the director's designated representative is authorized to require any of the following measures:

(A) The immediate exclusion of that person from the production, preparation, manufacture, packaging, storage, sale, distribution or transportation of food;

(B) The immediate exclusion of the food supply concerned from distribution and use; and

(C) Adequate medical examination of that person and his/her associates, including necessary laboratory testing of blood, feces, sputum, throat cultures and other bodily secretions or excreta.

AUTHORITY: sections 192.005.2., 192.020, 196.045 and 196.225, RSMo 1994. This rule was previously filed as 13 CSR 50-101.071. Original rule filed Dec. 11, 1981, effective May 13, 1982. Amended: Filed Nov. 4, 1992, effective May 6, 1993.*

**Original authority: 192.005.2., RSMo 1985, amended 1993; 192.020, RSMo 1939, amended 1945, 1951; 196.045, RSMo 1943, amended 1993; and 196.225, RSMo 1939, amended 1977.*

19 CSR 20-20.070 Duties of Local Health Departments

PURPOSE: This rule establishes procedures for reporting communicable diseases to the Missouri Department of Health by local health departments.

(1) All local health authorities shall forward reports of all diseases and conditions mentioned in 19 CSR 20-20.020 to the Missouri Department of Health. These reports shall be forwarded within twenty-four (24) hours after they are received, according to procedures established by the Department of Health director. Local health authorities shall transcribe from the original reports information necessary to the conduct of their duties in 19 CSR 20-20.040(2), (2)(A)–(J), (3) and (3)(A)–(C) before forwarding the reports. All reports received by either the local health authority or the Department of Health are to be considered confidential records and not public records.

AUTHORITY: section 192.020, RSMo 1994. This rule was previously filed as 13 CSR 50-101.080. Original rule filed July 15, 1948, effective Sept. 13, 1948. Amended: Filed Dec. 11, 1981, effective May 13, 1982.*

**Original authority: 192.020, RSMo 1939, amended 1945, 1951.*

19 CSR 20-20.075 Confidentiality of Information Obtained for Reporting of Communicable, Environmental and Occupational Diseases and Conditions

PURPOSE: This rule requires local public health agencies to establish confidentiality



policies and procedures which are as stringent as Missouri Department of Health (MDOH) policies and procedures for information obtained for reporting of communicable, environmental and occupational diseases. It also requires establishment of security policies and procedures for access to MDOH information systems.

(1) Local public health agencies shall adopt and abide by confidentiality policies and procedures which are as stringent as Missouri Department of Health (MDOH) policies and procedures for information obtained for the reporting of communicable, environmental and occupational diseases defined in 19 CSR 20-20.020.

(2) Such information may be used only for investigation to determine the source of exposure and/or potential for spread; follow-up screening to monitor disease, exposure status, or communicability; counseling and patient education regarding the disease or condition and its prevention; administration of immunizations and/or prophylactic medications to the case or contacts; isolation and/or restriction of the client's or contact's activities; environmental assessment and other activities undertaken to eliminate the source of exposure; or epidemiologic analysis to determine trends in incidence, prevalence, treatment, disease progression, and/or risk factors associated with diseases.

(3) Local public health agencies shall forward reports to MDOH in accordance with 19 CSR 20-20.020. Otherwise, such information shall be released only in a statistical aggregate form that precludes and prevents the identification of an individual, physician, or medical facility except when such release is specifically authorized by law.

(4) Local public health agencies that access MDOH information systems shall establish security policies and procedures which are as stringent as MDOH policies and procedures to protect information systems against unauthorized data disclosure, modification, or destruction and to protect the integrity of the information system. Local public health agencies and employees who use MDOH information systems to perform their duties shall abide by MDOH policies and procedures for access to and use of information systems.

(5) Local public health agencies shall provide comprehensive training to employees on confidentiality and security policies, laws, and the administrative, civil, and criminal penalties for violations. Local public health agencies shall monitor employees to assure com-

pliance with confidentiality laws, rules, policies and procedures. Local public health agencies shall immediately report to MDOH any breaches of confidentiality and security as specified by MDOH policy.

(6) Contractors performing work for MDOH or local public health agencies that involves access to information obtained for the reporting of communicable, environmental and occupational diseases shall be required, through their contracts, to abide by sections (1)–(5) of this rule.

AUTHORITY: sections 191.656, 192.006, 701.328, RSMo Supp. 1998 and 167.183, 192.020, 192.067 and 192.802, RSMo 1994. Original rule filed Aug. 4, 1999, effective Jan. 30, 2000.*

**Original authority: 167.183, RSMo 1992; 191.656, RSMo 1988, amended 1992, 1993, 1996; 192.006, RSMo 1993, amended 1995; 192.020, RSMo 1939, amended 1945, 1951; 192.067, RSMo 1988; 192.802, RSMo 1992; and 701.328, RSMo 1993, amended 1998.*

19 CSR 20-20.080 Duties of Laboratories

PURPOSE: This rule establishes the responsibility of laboratories to report to the Missouri Department of Health and Senior Services specified results of tests and to submit isolates/specimens for certain diseases and conditions.

(1) The director, person in charge of any laboratory, or designee of the director or person in charge of any laboratory shall report to the local health authority or the Missouri Department of Health and Senior Services the result of any test that is positive for, or suggestive of, any disease or condition listed in 19 CSR 20-20.020. These reports shall be made according to the time and manner specified for each disease or condition following completion of the test and shall designate the test performed, all results of the test, including numeric results, if applicable, units of measure of the results, and reference ranges for normal and abnormal results, the name and address of the attending physician, the name of the disease or condition diagnosed or suspected, the date the test results were obtained, the name and home address (with zip code) of the patient and the patient's age, date of birth, sex, race, and ethnicity.

(2) In reporting findings for diseases or conditions listed in 19 CSR 20-20.020, laboratories shall report—

Arsenic—results of all biological specimens including time frame of urine specimen collection, if applicable;

Cadmium—results of all biological specimens including time frame of urine specimen collection, if applicable;

Carboxyhemoglobin proportion—all results;

Chemical/pesticide (blood or serum)—all results, including if none detected;

Lead level—results of all biological specimens;

Mercury—results of all biological specimens including time frame of urine specimen collection, if applicable; and

Methemoglobin proportion—all results.

(3) Isolates or specimens positive for the following reportable diseases or conditions must be submitted to the State Public Health Laboratory for epidemiological or confirmation purposes:

Anthrax (*Bacillus anthracis*)

Cholera (*Vibrio cholerae*)

Diphtheria (*Corynebacterium diphtheriae*)

Escherichia coli O157:H7

Haemophilus influenzae, invasive disease

Influenza Virus-associated pediatric mortality

Listeriosis

Malaria (*Plasmodium* species)

Measles (rubeola)

Mycobacterium tuberculosis

Neisseria meningitidis, invasive disease

Orthopoxvirus (smallpox/cowpox-vaccinia/monkeypox)

Other Shiga Toxin positive organisms

Pertussis (*Bordetella pertussis*)

Plague (*Yersinia pestis*)

Salmonella species

Severe Acute Respiratory Syndrome-associated Coronavirus (SARS-CoV) disease

Shigella species

Tularemia, pneumonic

Vancomycin-intermediate *Staphylococcus aureus* (VISA)

Vancomycin Resistant *Staphylococcus aureus*

(4) Every laboratory performing culture and sensitivity testing on human specimens in Missouri for health care facilities shall annually report these results to the Missouri Department of Health and Senior Services (MDHSS) for each facility provided this service. The data submitted should be in the format of antibiograms as defined by the Clinical and Laboratory Standards Institute (CLSI), M39-A2, Analysis and Presentation of Cumulative Antimicrobial Susceptibility Test Data. Only data from the first unique isolate from each patient should be included. Duplicate cultures must be excluded when compiling these antibiograms. The antibiograms for the preceding year are to be sent to MDHSS by July 1 of the following year



(ex: 2006 data, January 1, 2006–December 31, 2006, will be due on July 1, 2007).

AUTHORITY: section 192.006, RSMo 2000 and sections 192.020 and 192.131, RSMo Supp. 2007. This rule was previously filed as 13 CSR 50-101.090. Original rule filed July 15, 1948, effective Sept. 13, 1948. Amended: Filed Aug. 4, 1986, effective Oct. 11, 1986. Amended: Filed Aug. 14, 1992, effective April 8, 1993. Amended: Filed Sept. 15, 1995, effective April 30, 1996. Emergency rule filed June 1, 2000, effective June 15, 2000, expired Dec. 11, 2000. Emergency rescission filed June 2, 2000, effective June 15, 2000, expired Dec. 11, 2000. Previous version of rule rescinded filed June 1, 2000, effective Jan. 30, 2001. Readopted: Filed June 1, 2000, effective Nov. 30, 2000. Amended: March 14, 2003, effective Sept. 30, 2003. Amended: Filed March 14, 2003, effective Sept. 30, 2003. Amended: Filed April 15, 2005, effective Oct. 30, 2005. Amended: Filed Feb. 15, 2006, effective Sept. 30, 2006. Amended: Filed Nov. 15, 2007, effective May 30, 2008.*

**Original authority: 192.006, RSMo 1993, amended 1995; 192.020, RSMo 1939, amended 1945, 1951, 2004; and 192.131, RSMo 2004.*

19 CSR 20-20.090 Contact With Communicable Diseases by First Responders or Emergency Medical Person and Mortuary Personnel

PURPOSE: This rule defines the procedures for notification to a first responder or emergency medical person and mortuary personnel who are exposed to an individual who is human immunodeficiency virus seropositive, hepatitis B infected or infected with any other reportable communicable disease as listed in 19 CSR 20-20.020(1)–(5).

(1) The following definitions shall be used in administering this rule:

(A) Authorized personnel—any individual who has the authority to hire or fire and demote or promote employees for a corporation, entity or organization;

(B) Emergency medical person—a licensed attendant who has been specially trained in emergency cardiac and noncardiac care, and who has successfully completed an emergency service training program certified by the Department of Health as meeting the requirements of sections 190.100–190.190, RSMo and any individual providing emergency medical services who is licensed under Chapters 334 and 335, RSMo;

(C) Employee—a wage earner or volunteer providing emergency care;

(D) Employer—one who provides gainful work for wage earners and volunteers in the emergency care area;

(E) Exposure—any contact with an individual who is human immunodeficiency virus (HIV) seropositive or infected with any other reportable communicable disease as listed in 19 CSR 20-20.020(1)–(5), when the contact is consistent with the known means of transmission and occurs within the period of communicability of the disease;

(F) Facility—a facility licensed under Chapter 197 or 198, RSMo.

(G) First responder—an individual with training in first aid or emergency medical care, who is associated with a police department, sheriff's department, fire service or ambulance service and who is routinely dispatched to the scene of an accident or unforeseen emergency medical incident prior to or with the arrival of a licensed, staffed and equipped ambulance;

(H) Mortuary personnel—those persons having direct contact with a corpse prior to completion of embalming, cremating or enclosing the corpse in a sealed casket; and

(I) To notify—within forty-eight (48) hours after confirming potential exposure, the facility shall report the potential exposure by phone or in person to the employer(s)/funeral director of the potentially exposed employee(s)/mortuary personnel.

(2) If a facility admits a patient who was in an emergency rescue operation, received medical treatment or was transported to the facility by a first responder or an emergency medical person and is subsequently diagnosed as HIV seropositive or infected with any other reportable communicable disease as listed in 19 CSR 20-20.020(1)–(5), the facility, after confirming the presence of the disease, shall notify the employer(s) of the potentially exposed employee(s). The employer(s) shall be provided with the ambulance run number, police incident report or sufficient information to enable identification of the potentially exposed employee without reference to the patient's name. Notifications shall remain confidential and shall be released to authorized personnel only.

(3) If mortuary personnel remove a corpse from a facility or provide care to the corpse and the facility subsequently determines the presence at the time of death of HIV seropositivity or infection with any other reportable communicable disease as listed in 19 CSR 20-20.020(1)–(5), the facility shall notify the

funeral director of the mortuary personnel's contact.

(4) The employer/funeral director shall investigate the potential exposure of the employee/mortuary personnel to determine if it was consistent with the known means of transmission and occurred within the period of communicability of the disease in question.

(A) If the exposure was consistent with the known means of transmission and occurred within the period of communicability, the employer/funeral director shall notify the employee/mortuary personnel within forty-eight (48) hours.

(B) The employer/funeral director shall instruct the employee/mortuary personnel to contact the facility for medical direction.

AUTHORITY: sections 190.100–190.190 and 191.653, RSMo 1994. Original rule filed July 18, 1989, effective Nov. 11, 1989.*

**Original authority: 190.100, RSMo 1973, amended 1987, 1989; 190.105–190.115, RSMo 1973; 190.120, RSMo 1973, amended 1980; 190.125–190.135, RSMo 1973; 190.140, RSMo 1973, amended 1987; 190.141, RSMo 1989; 190.145, RSMo 1973, amended 1975; 190.150–190.160, RSMo 1973; 190.165, RSMo 1973, amended 1978; 190.171, RSMo 1978; 190.175–190.180, RSMo 1973; 190.185, RSMo 1973, amended 1989, 1993; 190.190, RSMo 1973; and 191.653, RSMo 1988.*

19 CSR 20-20.091 Testing for Contagious or Infectious Disease

PURPOSE: This rule determines the contagious or infectious diseases for which testing is reasonable and appropriate and which may be administered pursuant to section 191.631, RSMo.

(1) Tests for the following contagious or infectious diseases may be administered pursuant to sections 191.630 to 191.631, RSMo:

- (A) Hepatitis B;
- (B) Hepatitis C;
- (C) Syphilis; and/or
- (D) Human T-Cell Lymphotropic Virus (HTLV) I/II.

AUTHORITY: section 191.631, RSMo Supp. 2002. Original rule filed March 14, 2003, effective Sept. 30, 2003.*

**Original authority: 191.631, RSMo 2002.*

19 CSR 20-20.092 Blood-Borne Pathogen Standard Required for Occupational Exposure of Public Employees to Blood and Other Infectious Materials

PURPOSE: This rule establishes standards



for protection of public employees from occupational exposure to blood-borne pathogens in the workplace.

PUBLISHER'S NOTE: The secretary of state has determined that the publication of the entire text of the material which is incorporated by reference as a portion of this rule would be unduly cumbersome or expensive. Therefore, the material which is so incorporated is on file with the agency who filed this rule, and with the Office of the Secretary of State. Any interested person may view this material at either agency's headquarters or the same will be made available at the Office of the Secretary of State at a cost not to exceed actual cost of copy reproduction. The entire text of the rule is printed here. This note refers only to the incorporated by reference material.

(1) The blood-borne pathogen standard governing public employers in the state of Missouri having employees with occupational exposure to blood or other potentially infectious materials shall be the standard of the Occupational Safety and Health Administration as codified in 29 CFR 1910.1030. The Occupational Safety and Health Administration standard as codified in 29 CFR 1910.1030 is incorporated herein by reference.

(2) As part of the Occupational Safety and Health Administration blood-borne pathogen standard codified in 29 CFR 1910.1030, each public employer having employees with occupational exposure is required to establish a written Exposure Control Plan. Such plan shall include a requirement that the most effective available needleless systems and sharps with engineered sharps injury protection be included as engineering and work practice controls. However, such engineering controls shall not be required if:

(A) None are available in the marketplace; or

(B) An evaluation committee, as described in section 191.640.5, RSMo determines by means of objective product evaluation criteria that use of such devices will jeopardize patient or employee safety with regard to a specific medical procedure.

AUTHORITY: sections 191.640, RSMo Supp. 2002 and 192.006, RSMo 2000.* Original rule filed March 14, 2003, effective Sept. 30, 2003.

*Original authority: 191.640, RSMo 2001; 192.006, RSMo 1993, amended 1995.

19 CSR 20-20.100 Tuberculosis Testing for Residents and Workers in Long-Term Care Facilities and State Correctional Centers

PURPOSE: This rule establishes tuberculosis testing requirements for residents and workers in long-term care facilities and state correctional centers.

(1) General Requirements. Long-term care facilities and state correctional centers shall screen their residents and staff for tuberculosis using the Mantoux method purified protein derivative (PPD) five tuberculin unit (5 TU) test. Each facility shall be responsible for ensuring that all test results are completed and that documentation is maintained for all residents, employees, and volunteers.

(A) In interpreting this rule, long-term care facilities shall include employees, volunteers, and residents of residential care facilities I, residential care facilities II, intermediate care facilities and skilled nursing facilities as defined in section 198.006, RSMo.

(B) In interpreting this rule, state correctional centers shall include all employees and volunteers of the Missouri Department of Corrections and the residents of all correctional institutions operated by the Missouri Department of Corrections.

(C) Whenever tuberculosis is suspected or confirmed, or tuberculosis infection is diagnosed among residents, employees or volunteers, the Department of Health or local health authority shall be notified as required in 19 CSR 20-20.020(2).

(2) Long-Term Care Residents. Within one (1) month prior to or one (1) week after admission, all residents new to long-term care are required to have the initial test of a Mantoux PPD two (2)-step tuberculin test. If the initial test is negative, zero to nine millimeters (0–9 mm), the second test, which can be given after admission, should be given one to three (1–3) weeks later. Documentation of chest X ray evidence ruling out tuberculosis disease within one (1) month prior to admission, along with an evaluation to rule out signs and symptoms compatible with infectious tuberculosis, may be accepted by the facility on an interim basis until the Mantoux PPD two (2)-step test is completed.

(A) All skin test results are to be documented in millimeters (mm) of induration.

(B) Bacillus of Calmette and Guerin (BCG) vaccination shall not prevent residents from receiving a tuberculin test.

(C) A reaction of ten millimeters (10 mm) or more shall be considered as infected with *Mycobacterium tuberculosis* for an individual with a history of BCG vaccination.

(D) Evidence of tuberculosis infection is considered to be a reaction of five millimeters (5 mm) or more for all contacts to infectious tuberculosis or for an individual who is immunosuppressed or has abnormal chest X-ray findings consistent with old healed tuberculosis disease, and ten millimeters (10 mm) or more for all others.

(E) Residents with a negative, zero to nine millimeters (0–9 mm), Mantoux PPD two (2)-step test need not be routinely retested unless exposed to infectious tuberculosis or they develop signs and symptoms which are compatible with tuberculosis disease.

(F) Residents with a documented history of tuberculosis infection or an adequate course of preventive treatment shall not be required to be retested. Residents with a documented history of tuberculosis disease and adequate chemotherapy shall not be required to be retested. In the absence of documentation, a repeat test shall be required.

(G) All skin test results of five millimeters (5 mm) or more for contacts to infectious tuberculosis or for an individual who is immunocompromised, or ten millimeters (10 mm) or more for all others, shall require a chest X ray within one (1) week, or a review of the results of a chest X ray taken within the month prior to admission along with an evaluation to rule out signs and symptoms compatible with tuberculosis disease to rule out active pulmonary disease.

(H) Individuals with a positive finding presenting evidence of a recent, within one (1) month of the date of admission, chest X ray need not be given a new X ray. However, the results of the X ray must be reviewed in the light of the additional information of the identification of tuberculosis infection as indicated by the Mantoux PPD skin test.

(I) An individual who is skin-test positive with a normal chest X ray should be considered for preventive medication. Those who complete a recommended course of preventive treatment and those for whom preventive treatment is not medically indicated need have no further testing for tuberculosis unless signs and symptoms which are compatible with tuberculosis disease are present.

(J) All residents of long-term care facilities who are exposed to a case of infectious tuberculosis or who develop signs and symptoms which are compatible with tuberculosis disease shall be medically evaluated. All long-term care facility residents shall have a documented annual evaluation to rule out signs and symptoms of tuberculosis disease.

(3) Long-Term Care Employees and Volunteers. All new long-term care facility employees and volunteers who work ten (10)



or more hours per week are required to obtain a Mantoux PPD two (2)-step tuberculin test within one (1) month prior to starting employment in the facility. If the initial test is zero to nine millimeters (0–9 mm), the second test should be given as soon as possible within three (3) weeks after employment begins, unless documentation is provided indicating a Mantoux PPD test in the past and at least one (1) subsequent annual test within the past two (2) years. It is the responsibility of each facility to maintain a documentation of each employee's and volunteer's tuberculin status.

(A) All skin test results are to be documented in millimeters (mm) of induration.

(B) BCG vaccination shall not prevent employees and volunteers from receiving a tuberculin test.

(C) For an individual with a history of BCG vaccination, a reaction of ten millimeters (10 mm) or more shall be considered as infected with *Mycobacterium tuberculosis*.

(D) Evidence of tuberculosis infection is considered to be a reaction of five millimeters (5 mm) or more for all contacts to infectious tuberculosis or for an individual who is immunosuppressed or has abnormal chest X ray findings consistent with old healed tuberculosis disease, and ten millimeters (10 mm) or more for all others.

(E) Employees and volunteers with an initial zero to nine millimeters (0–9 mm) Mantoux PPD two (2)-step test shall be one (1)-step tuberculin tested annually and the results recorded in a permanent record.

(F) Employees and volunteers with a documented history of a positive Mantoux PPD test shall not be required to be retested. In the absence of documentation, a repeat test shall be required.

(G) All positive findings shall require a chest X ray to rule out active pulmonary disease.

(H) Individuals with a positive finding need not have repeat annual chest X rays. They shall have a documented annual evaluation to rule out signs and symptoms of tuberculosis disease.

(I) An individual who is skin-test positive with a normal chest X ray should be considered for preventive medication. Those who complete a recommended course of preventive medication need have no further testing for tuberculosis unless signs and symptoms which are compatible with tuberculosis disease are present.

(J) All employees and volunteers of long-term care facilities who are exposed to a case of infectious tuberculosis or who develop signs and symptoms which are compatible with tuberculosis disease shall be medically evaluated. All employees or volunteers of

these facilities shall have a documented annual evaluation to rule out signs and symptoms of tuberculosis disease.

(4) State Correctional Centers Residents. All residents of state correctional centers are required to obtain a Mantoux PPD two (2)-step tuberculin test upon admission to rule out tuberculosis. If the initial test is negative, zero to nine millimeters (0–9 mm), the second test should be given within ninety (90) days of entrance into the state correctional system.

(A) All skin test results are to be documented in millimeters (mm) of induration.

(B) BCG vaccination shall not prevent residents from receiving a tuberculin test.

(C) For an individual with a history of BCG vaccination, a reaction of ten millimeters (10 mm) or more shall be considered as infected with *Mycobacterium tuberculosis*.

(D) A positive test is defined as having a reaction of five millimeters (5 mm) or more for all contacts to infectious tuberculosis or for an individual who is immunosuppressed or has abnormal chest X ray findings consistent with old healed tuberculosis disease, and ten millimeters (10 mm) or more for all others.

(E) Individuals with an initial negative zero to nine millimeters (0–9 mm) Mantoux PPD two (2)-step test shall be one (1)-step tuberculin tested annually and the results recorded in a permanent record.

(F) Individuals with a documented history of a positive Mantoux PPD test shall not be required to be retested. In the absence of documentation, a repeat test shall be required.

(G) All positive findings shall require a chest X ray to rule out active pulmonary disease.

(H) Individuals with a positive finding need not have repeat annual chest X rays. They shall have a documented annual evaluation to rule out signs and symptoms of tuberculosis disease.

(I) An individual who is skin-test positive with a normal chest X ray should be considered for preventive medication. Those who complete a recommended course of preventive medication need have no further testing for tuberculosis unless signs and symptoms which are compatible with tuberculosis disease are present.

(J) All residents of state correctional centers who are exposed to a case of infectious tuberculosis or who develop signs and symptoms which are compatible with tuberculosis disease shall be medically evaluated. All residents shall have a documented annual evaluation to rule out signs and symptoms of tuberculosis disease.

(5) Missouri Department of Corrections New Employees and Volunteers. All new employees and volunteers who work ten (10) or more hours per week for the Missouri Department of Corrections are required to obtain a Mantoux PPD two (2)-step tuberculin test within three (3) weeks of starting employment. If the initial test is negative, zero to nine millimeters (0–9 mm), the second test should be given one to three (1–3) weeks after the initial test. It is the responsibility of each state correctional center to maintain documentation of each employee's or volunteer's tuberculin status.

(A) All skin test results are to be documented in millimeters (mm) of induration.

(B) BCG vaccination shall not prevent new employees and volunteers from receiving a tuberculin test.

(C) For an individual with a history of BCG vaccination, a significant reaction of ten millimeters (10 mm) or more shall be considered as infected with *Mycobacterium tuberculosis*.

(D) A positive test is defined as having a reaction of five millimeters (5 mm) or more for all contacts to infectious tuberculosis or for an individual who is immunosuppressed or has abnormal chest X ray findings consistent with old healed tuberculosis disease, and ten millimeters (10 mm) or more for all others.

(E) Employees and volunteers with a negative zero to nine millimeters (0–9 mm) Mantoux PPD two (2)-step test shall be one (1)-step tuberculin tested annually and the results recorded in a permanent record.

(F) Employees and volunteers with a documented history of a positive Mantoux PPD test shall not be required to be retested. In the absence of documentation, a repeat test shall be required.

(G) All positive findings shall require a chest X ray to rule out active pulmonary disease.

(H) Individuals with a positive finding need not have repeat annual chest X rays. They shall have a documented annual evaluation to rule out signs and symptoms of tuberculosis disease.

(I) An individual who is skin-test positive with a normal chest X ray should be considered for preventive medication. Those who complete a recommended course of preventive medication need have no further testing for tuberculosis unless signs and symptoms which are compatible with tuberculosis disease are present.

(J) All employees and volunteers of state correctional centers who are exposed to a case of infectious tuberculosis or who develop signs and symptoms which are compatible



with tuberculosis disease shall be medically evaluated. All employees and volunteers shall have a documented annual evaluation to rule out signs and symptoms of tuberculosis disease.

AUTHORITY: section 199.350, RSMo 1994. Original rule filed April 17, 1995, effective Nov. 30, 1995. Emergency amendment filed June 14, 2000, effective June 24, 2000, expired Feb. 22, 2001. Amended: Filed June 14, 2000, effective Nov. 30, 2000.*

**Original authority: 199.350, RSMo 1992.*

Appendix E

List of Diseases and Conditions Reportable In Missouri

Diseases and Conditions Reportable In Missouri (19 CSR 20-20.020)

Numbers in parenthesis represent ICD-9 and ICD-10 Codes

Report Diseases and Conditions to your local health agency or to:
Missouri Department of Health and Senior Services during business hours 573-751-6113,
after hours and on weekends 800-392-0272 or by fax 573-526-0235

1. Immediately reportable diseases or findings shall be reported to the local health authority or to the Department of Health and Senior Services immediately upon knowledge or suspicion by telephone, facsimile or other rapid communication. Immediately reportable diseases or findings are—

(A) Selected high priority diseases, findings or agents that occur naturally, form accidental exposure, or as the result of a bioterrorism event:

- Anthrax (022, A22)
- Botulism (005.1, A05.1)
- Plague (020, A20)
- Rabies (Human) (071, A82)
- Ricin Toxin (988, T62)
- Severe Acute Respiratory Syndrome-associated Coronavirus (SARS-CoV) Disease (480.3, J12.8)
- Smallpox (variola) (050, B03)
- Tularemia (pneumonic) (021.2, A21.2)
- Viral hemorrhagic fevers (filoviruses (e.g., Ebola, Marburg) and arenaviruses (e.g., Lassa, Machupo)) (078.7, 078.89, A96, A98, A99)

(B) Instances, clusters, or outbreaks of unusual diseases or manifestations of illness and clusters or instances of unexplained deaths which appear to be a result of a terrorist act or the intentional or deliberate release of biological, chemical, radiological, or physical agents, including exposures through food, water, or air.

(C) Instances, clusters, or outbreaks of unusual, novel, and/or emerging diseases or findings not otherwise named in this rule, appearing to be naturally occurring, but posing a substantial risk to public health and/or social and economic stability due to their ease of dissemination or transmittal, associated mortality rates, or the need for special public health actions to control.

2. Reportable within one (1) day diseases or findings shall be reported to the local health authority or to the Department of Health and Senior Services within one (1) calendar day of first knowledge or suspicion by telephone, facsimile or other rapid communication. Reportable within one (1) day diseases or findings are—

(A) Diseases, findings or agents that occur naturally, or from accidental exposure, or as a result of an undetected bioterrorism event:

- Acute respiratory distress syndrome (ARDS) in patients under fifty (50) years of age (without a contributing medical history)
- Animal (mammal) bite, wound, humans
- Brucellosis (023, A23)
- Cholera (001, A00)
- Dengue fever (065.4, A90, A91)
- Diphtheria (032, A36)
- Glanders (024, A24.0)
- *Haemophilus influenzae*, invasive disease (038.41, 041.5, 320.0, A41.3, J14, G00.0)
- Hantavirus pulmonary syndrome (079.81, 480.8, B33.8)
- Hemolytic uremic syndrome (HUS), post-diarrheal (283.11, D59.3)
- Hepatitis A (070.0, 070.1, B15)
- Influenza - associated pediatric mortality (18 years of age or younger) (487, J10)
- Influenza - associated public and/or private school closures (487, J10)
- Lead (blood) level greater than or equal to forty-five micrograms per deciliter ($\geq 45 \mu\text{g}/\text{dl}$) in any person equal to or less than seventy-two (≤ 72) months of age

- Measles (rubeola) (055, B05)
- Meningococcal disease, invasive (036, A39)
- Novel Influenza A virus infections, human (487, J10)
- Outbreaks (including nosocomial) or epidemics of any illness, disease or condition that may be of public health concern, including illness in a food handler that is potentially transmissible through food
- Pertussis (033.0, A37.0)
- Poliomyelitis (045, A80)
- Poliovirus infection, nonparalytic
- Q fever (083.0, A78)
- Rabies (animal)
- Rubella, including congenital syndrome (056, 771.0, B06, P35.0)
- Shiga toxin-producing *Escherichia coli* (STEC) (008.04, A04.3)
- Shiga toxin positive, unknown organism (005.8, 005.9, A04.8, A04.9)
- Shigellosis (004, A03)
- Staphylococcal enterotoxin B (988, T62)
- Streptococcus pneumoniae, drug resistant invasive disease (038.2, 481, 482.3, A40.3, J13)
- Syphilis, including congenital syphilis (090, 093-097, A50-A52)
- T-2 mycotoxins (989.7, 989.9, T64)
- Tetanus (037, A35)
- Tuberculosis disease (010-018, A15-A19)
- Tularemia (non-pneumonic) (021.3-9, A21.0-.1, A21.3-.9)
- Typhoid fever (*Salmonella typhi*) (002.0, A01.0)
- Vancomycin-intermediate *Staphylococcus aureus* (VISA), and Vancomycin-resistant *Staphylococcus aureus* (VRSA) (038.11, 041.11, A41.0, A49.0)
- Venezuelan equine encephalitis virus neuroinvasive disease (066.2, A92.2)
- Venezuelan equine encephalitis virus non-neuroinvasive disease (066.2, A92.2)
- Yellow fever (060.9, A95)

(B) Diseases, findings or adverse reactions that occur as a result of inoculation to prevent smallpox, including but not limited to the following:

- Accidental administration
- Contact transmission (i.e., vaccinia virus infection in a contact of a smallpox vaccinee)
- Eczema vaccinatum
- Erythema multiforme (roseola vaccinia, toxic urticaria)
- Fetal vaccinia (congenital vaccinia)
- Generalized vaccinia
- Inadvertent autoinoculation (accidental implantation)
- Myocarditis, pericarditis, or myopericarditis
- Ocular vaccinia (can include keratitis, conjunctivitis, or blepharitis)
- Post-vaccinial encephalitis or encephalomyelitis
- Progressive vaccinia (vaccinia necrosum, vaccinia gangrenosa, disseminated vaccinia)
- Pyogenic infection of the vaccination site
- Stevens-Johnson Syndrome

3. Reportable within three (3) days diseases or findings shall be reported to the local health authority or the Department of Health and Senior Services within three (3) calendar days of first knowledge or suspicion. These diseases or findings are—

- Acquired immunodeficiency syndrome (AIDS) (042, B20)
- Arsenic poisoning

(Continued on page 2)

- California serogroup virus neuroinvasive disease (062.5, A83.5)
- California serogroup virus non-neuroinvasive disease (062.5, A92.8)
- Campylobacteriosis (008.43, A04.5)
- Carbon monoxide poisoning
- CD4+ T cell count
- Chancroid (099.0, A57)
- Chemical poisoning, acute, as defined in the most current ATSDR CERCLA Priority List of Hazardous Substances; if terrorism is suspected, refer to subsection (1)(B)
- *Chlamydia trachomatis* infections (099.8, A56)
- Coccidioidomycosis (114, B38)
- Creutzfeldt-Jakob disease (046.1, A81.0)
- Cryptosporidiosis (007.4, A07.2)
- Cyclosporiasis (007.5, A07.8)
- Eastern equine encephalitis virus neuroninvasive disease (062.2, A83.2)
- Eastern equine encephalitis virus non-neuroninvasive disease (062.2, A92.8)
- Ehrlichiosis, human granulocytic, monocytic, or other/unspecified agent (082.40, 082.41, 082.49, A79.8, A79.9)
- Giardiasis (007.1, A07.1)
- Gonorrhea (098.0-098.3, A54.0-A54.2)
- Hansen's disease (Leprosy) (030, A30)
- Heavy metal poisoning including, but not limited to, cadmium and mercury
- Hepatitis B, acute (070.20, 070.21, 070.30, 070.31, B16)
- Hepatitis B, chronic (070.22, 070.23, 070.32, 070.33, 070.42, 070.52, B18.0, B18.1)
- Hepatitis B surface antigen (prenatal HBsAg) in pregnant women (070.20-070.23, 070.30-070.33, 070.42, 070.52, B16, B18.0, B18.1)
- Hepatitis B Virus Infection, perinatal (HbsAg positivity in any infant aged equal to or less than twenty-four (≤ 24) months who was born to an HbsAg-positive mother) (070.20-070.23, 070.30-070.33, 070.42, 070.52, B16, B18.0, B18.1)
- Hepatitis C, acute (070.41, 070.51, B17.1)
- Hepatitis C, chronic (070.44, 070.54, B18.2)
- Hepatitis non-A, non-B, non-C (070.9, B19)
- Human immunodeficiency virus (HIV)-exposed newborn infant (i.e., newborn infant whose mother is infected with HIV)
- Human immunodeficiency virus (HIV) infection, as indicated by HIV antibody testing (reactive screening test followed by a positive confirmatory test), HIV antigen testing (reactive screening test followed by a positive confirmatory test), detection of HIV nucleic acid (RNA or DNA), HIV viral culture, or other testing that indicates HIV infection
- Human immunodeficiency virus (HIV) test results (including both positive and negative results) for children less than two (2) years of age whose mothers are infected with HIV
- Human immunodeficiency virus (HIV) viral load measurement (including nondetectable results)
- Hyperthermia
- Hypothermia
- Lead (blood) level less than forty-five micrograms per deciliter ($<45 \mu\text{g}/\text{dl}$) in any person equal to or less than seventy-two (≤ 72) months of age and any lead (blood) level in persons older than seventy-two (>72) months of age
- Legionellosis (482.84, A48.1, A48.2)
- Leptospirosis (100, A27)
- Listeriosis (027.0, 771.2, A32, P37.2)
- Lyme disease (088.81, A69.2)
- Malaria (084, B50-B54)
- Methemoglobinemia, environmentally-induced
- Mumps (072, B26)
- Mycobacterial disease other than tuberculosis (MOTT) (031, A31)
- Occupational lung diseases including silicosis, asbestosis, byssinosis, farmer's lung and toxic organic dust syndrome
- Pesticide poisoning
- Powassan virus neuroinvasive disease (063.8, A83.8)
- Powassan virus non-neuroinvasive disease (063.8, A92.8)
- Psittacosis (073, A70)
- Rabies Post-Exposure Prophylaxis (Initiated) (V01.5 V04.5)
- Respiratory diseases triggered by environmental contaminants including environmentally or occupationally induced asthma and bronchitis
- Rocky Mountain spotted fever (082.0, A77.0)
- Saint Louis encephalitis virus neuroinvasive disease (062.3, A83.3)
- Saint Louis encephalitis virus non-neuroinvasive disease (062.3, A92.8)
- Salmonellosis (003, A02.0)
- Streptococcal disease, invasive, Group A (041.01, 034.1, A40.0, A49.1, A38)
- *Streptococcus pneumoniae*, invasive in children less than five (5) years (038.2, 481, 482.3, A40.3, J13)
- Toxic shock syndrome, staphylococcal or streptococcal (785.5, A48.3)
- Trichinellosis (124, B75)
- Tuberculosis infection (795.5, R76.1)
- Varicella (chickenpox) (052.1, 052.7, 052.8, 052.9)
- Varicella deaths (052, B01)
- Vibriosis (non-cholera *Vibrio* species infections) (005.4, .8, A05.3, .8)
- West Nile virus neuroinvasive disease (066.41, 066.42, A92.3)
- West Nile virus non-neuroinvasive disease (066.40, 066.49, A92.3)
- Western equine encephalitis virus neuroinvasive disease (062.1, A83.1)
- Western equine encephalitis virus non-neuroinvasive disease (062.1, A92.8)
- Yersiniosis (008.44, A04.6)

4. Reportable weekly diseases or findings shall be reported directly to the Department of Health and Senior Services weekly. These diseases or findings are—

- Influenza, laboratory-confirmed (487, J10)

5. Reportable quarterly diseases or findings shall be reported directly to the Department of Health and Senior Services quarterly. These disease or findings are—

- Methicillin-resistant *Staphylococcus aureus* (MRSA), nosocomial
- Vancomycin-resistant enterococci (VRE), nosocomial

NOTE: Cancer is also a reportable disease. Please refer to [CSR 70-21.010](#) for complete information.

Isolates or specimens positive for the following reportable diseases or conditions must be submitted to the State Public Health Laboratory for epidemiological or confirmation purposes:

- Anthrax (*Bacillus anthracis*)
- Cholera (*Vibrio cholerae*)
- Diphtheria (*Corynebacterium diphtheriae*)
- *Escherichia coli* O157:H7
- *Haemophilus influenzae*, invasive disease
- Influenza Virus-associated pediatric mortality
- Listeriosis
- Malaria (*Plasmodium* species)
- Measles (rubeola)
- *Mycobacterium tuberculosis*
- *Neisseria meningitidis*, invasive disease
- Orthopoxvirus (Smallpox/cowpox-vaccinia/monkeypox)
- Other Shiga Toxin positive organisms
- Pertussis (*Bordetella pertussis*)
- Plague (*Yersinia pestis*)
- Salmonella species
- Severe Acute Respiratory Syndrome-associated Coronavirus (SARS-CoV) disease
- Shigella species
- Tularemia, pneumonic
- Vancomycin-intermediate *Staphylococcus aureus* (VISA)
- Vancomycin Resistant *Staphylococcus aureus*

The reporting rule can be accessed at: <http://www.sos.mo.gov/adrules/csr/current/19csr/19c20-20.pdf>
AN EQUAL OPPORTUNITY/AFFIRMATIVE ACTION EMPLOYER
Service provided on a nondiscriminatory basis