

LCP ECOLOGICAL RISK ASSESSMENT FOR EMERGENCY REMOVAL ACTIVITIES

This document was presented to describe sampling and analyses conducted for the emergency removal aspect of the LCP Site. As stated in the ecological risk assessment these data were collected and the report submitted for the purpose of investigating eminent and substantial risk to human health and the environment.

Reference is made to a biological opinion and section 7 consultation. To date, no consultation has been requested by the EPA.

The selection of reference areas is critical and assurances should be made that these areas (Troupe Creek and the Satilla River) are appropriate and historical documentation shows these have not been impacted. A description for the inclusion of these areas is necessary to fully evaluate their applicability as reference areas.

There are numerous contaminants that are known from the site that need to be examined. A complete description of the contaminants of concern (COC's) is necessary to evaluate potential environmental impacts. There are PAH's, lead, sodium and other contaminants that have been tentatively identified emanating from the site. The nature and extent of these contaminants has not been defined to date. Several inferences are made regarding several different Aroclors but the source and or extent have not been made clear. Testing representative biota for PCB congeners would aid in determining specific physiological testing to identify sub-lethal impacts to the biota.

The effects of PCBs do not necessarily relate to direct toxicity. Other methodology should be used to evaluate potential impacts to individuals and populations from the contaminants emanating from this locale. The congener specific analyses performed by Skidaway Institute show dechlorination may be occurring within the marsh and thereby changing the composition of the contaminants. Additionally, several Aroclors have been discussed and there seems to be some confusion as to which Aroclor was tested from a particular spot. Aroclor 1268 is a unique aroclor yet there is not a discussion as to potential metabolites and there are no data within the literature discussing toxicity or other adverse impacts of this aroclor. The use of the aroclor may have changed to composition of congeners as this aroclor was exposed to high temperatures, pressure and mechanical stress. The testing for only Aroclor 1268 or 1260 identify the body burden of these contaminants but do not address the metabolites or their potential

influence on biological processes. Furthermore, when addressing the issues of the COC's it is imperative to investigate possible synergistic, additive, antagonistic or agonistic effects of the combination of the COC's.

In the investigation composite samples of edible tissues was made of specific fishes. Use of edible tissue data is essential to evaluate human health concerns, however, to be conservative regarding environmental impacts it would be prudent to use individual samples (whole body) to assess potential bioaccumulation of the COC's.

It is stated "Measurement endpoints should be linked to the assessment endpoints by the mechanism of toxicity and the route of exposure." The data are not specific in identifying the complete exposure pathways nor the mechanism of toxicity. As described above, little is known of the toxicity of Aroclor 1268 and the primary COC's (mercury, PCB's, PAH's and lead) have varying impacts on biota with low toxicity. Moreover, without identifying appropriate measurement endpoints for chronic effects it would not be advisable to speculate on chronic effects as an order of magnitude less than the acute testing values. This assumes a linear relationship between the acute concentrations measuring lethality and chronic conditions that should be based on anatomical, physiological and populational impacts.

Throughout the risk assessment it is stipulated that PCB's, and mercury are reproductive, behavioral and developmental toxins that only under unique conditions cause direct mortality. Following this it is determined that acute/imminent threat exposure are sufficient to cause direct mortality and/or a short term dose will result in chronic mortality related to response or reproductive failure. These statements indicate chronic and sub-lethal testing methodology be instituted to determine the long term effects of the COC's.

Behavioral impacts would be anticipated from the COC's and their initial identified levels. These could severely impact threatened and endangered species that have unique feeding behaviors dependent on physiological mechanisms known to be sensitive to the COC's.

Sediment testing within the "marsh" to a depth of 18" is not sufficient. The report indicates that "PCB concentrations increased from 0.25 mg/kg at the surface to 5.4 mg/kg at depth." without specifying the depth. It is assumed the depth was to the 18" level. Interestingly enough this is the same approximate

depth that the root bed and mat of the *Spartina* extends. It would seem reasonable that degradation of the PCB's, PAH's and possible methylation of mercury could occur within this depth and that actions requiring the elimination of this layer may yield contaminants at higher levels. Furthermore, the products found within this layer may have a higher propensity to be bioavailable. Deeper sediment testing would be recommended to further identify and characterize the nature and extent of the COC's as well as sub surface water flow and potential transport of the COC's. The core sample from Purvis Creek indicated the mercury concentration increased with depth. This is an important finding when looking at the overall health and activities within the potential area of concern. That is, dredging activities are being planned and are occurring within the potential area of concern. This coupled with the releases occurring for many years would lead to a hypothesis that depositional zones could contain high levels of COC's and future activities may cause a bolus release of these through resuspension and disturbance.

Several references have been made to the "co-location" of the COC's and for the most part this seems to hold. However, there are areas that have not been investigated and the full extent of the contamination and the identification of the contaminants has not been addressed.

Limited sampling (6 samples) within the "marsh area" for organo-mercury samples showed all samples had methyl mercury and these samples varied in there concentration. This would indicate further delineation of the site and/or a mechanism to determine methylation rates need to be performed.

Dioxin analyses are based on TEQ's and need to be clarified when considering degradation compounds of the PCB's and the potential dechlorination of organo-chlorines as described by preliminary data from the Skidaway Institute investigations. Dibenzo dioxins and dibenzo furan analyses should be displayed and then total "dioxin" levels reported for the nature and extent of the contamination within the marsh.

The models used to produce the "high" concentration maps should be explained. The limited sampling performed in the marsh north of the outfall canal and the depositional zones do not lend themselves to broad interpretations of the dispersion pattern. Without identification of potential areas of certain COC's (i.e. lead and PAH's) there is a high degree of uncertainty in trying to depict the contaminant pattern within the marsh and depositional zones. Additionally, maps have been generated on contaminants

that have not had adequate tests performed nor a source area identified (e.g. PCB 1260).

Food Web and biotic testing

The food web model does not include incidental ingestion of water and sediments for the avian, reptilian and mammalian species. Inclusion of these items would be the appropriate modeling for conservative estimates of risk. The LOAEL's derived from a "calculated" value from the food web model do not address the chronic effects associated with mercury, pcb's, pah's and lead. In the food web model incidental exposure to sediments through the feeding on crustaceans was not included. Depurating the animals prior to analyses could severely underestimate the quantity of ingested contaminants for the avian and reptilian models. Additionally, without examining the roots and cleaning of the Spartina the exposure scenario is again underestimated for the herbivores (Manatee) which are in residence throughout the year.

A species diversity index should accompany the food web model. It is important to identify which species may have been extirpated from the "site" due to low tolerance and if the reference areas are comparable. This will also aid in determining the prey base for the food web surrogates as the availability, density and composition of the prey base directly influences exposure scenarios and behavior.

The environmental stressors impeding on the surviving species at the site should be evaluated through explicit testing procedures as indicated in the outline presented in 1995. These testing procedures and their rationale are still applicable to the site. Furthermore, to establish risks to the environment it is essential to understand the full extent of chronic conditions that are occurring at the site. It is known that continuous exposure to low stress levels will raise the baseline stress response and the magnitude of response to further stressors is reduced. That is, accommodation may be occurring in the resident species and they are not functioning to their full capacity. Many of the species of concern are long lived and impacts to them would not be manifest for many years or are transgenerational. The lipid reduction and overall health of the specimens collected are indicative of severe environmental stress. Tolerance thresholds may have been met and exceeded for many of the species depicted within the food web model and their responses may not have been fully evaluated. To determine reproductive incompetency appropriate tests (enclosure) should be performed. Small sample sizes and body (egg) burdens are necessary, however, further

identification of developmental and ontogenetic changes are necessary.

In determining the "LOELs" it is necessary to find the level of contamination that produces a risk factor of one. Comparisons can then be made and further identification of the "plume" of contamination be determined.