HUMAN HEALTH BASELINE RISK ASSESSMENT FOR THE ESTUARY, OPERABLE UNIT 1 MARSH TRESPASSER, FISH AND SHELLFISH CONSUMER, CLAPPER RAIL CONSUMER

Revision 4 dated ~~December 2010~~July 2011

LCP CHEMICALS SUPERFUND SITE
BRUNSWICK, GEORGIA

Prepared for:

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~~December 2010~~July 2011

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HUMAN HEALTH BASELINE RISK ASSESSMENT FOR THE ESTUARY, OPERABLE UNIT 1
MARSH TRESPASSER, FISH AND SHELLFISH CONSUMER, CLAPPER RAIL CONSUMER
Revision ~~14~~
LCP Chemicals Superfund Site
Brunswick, Georgia

~~December 2010~~
July 2011

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1.0 INTRODUCTION

1.1 Overview

This report, which has been prepared by Environmental Planning Specialists Inc. (EPS) with assistance from Ted Simon LLC on behalf of the LCP Steering Committee, provides a human health baseline risk assessment (HHBRA) for Operable Unit 1 (OU1) of the LCP Chemicals Superfund Site in Brunswick, Georgia. This report is specific to marsh trespasser and consumption of fish, shellfish and clapper rail. These scenarios were requested by the US Environmental ~~Proection~~Protection Agency (USEPA) to evaluate any potential health risks associated with incidental or purposeful ingestion of estuarine biota from the LCP marsh and contact with LCP marsh sediment.

1.2 Timeline

Arcadis Geraghty Miller, Inc. previously prepared a draft HHBRA in 1997 and a revised HHBRA in 1999 (Geraghty & Miller, 1999), but at the time the estuary HHBRA was linked to the upland assessment (the upland is now recorded as Operable Unit 3). The USEPA segregated these into two OUs in late 2005, and subsequently requested a stand-alone HHBRA for the estuary (OU1) for the listed scenarios. Previous drafts of a stand-alone HHBRA were prepared by Ted Simon LLC and EPS in March 2008, October 2008 ~~and~~ , July 2009~~. This version addresses comments issued by USEPA in a letter to Honeywell dated December 10, 2009. In addition, in an email dated January 7, 2010, USEPA informed Honeywell that USEPA would perform an evaluation of the data and provide Honeywell with the summary statistics and exposure point concentrations (EPC) to be used in the revised HHBRA. The USEPA provided those summary statistics to Honeywell in a letter dated July 1, 2010. There were subsequently several rounds of correspondence between Honeywell and USEPA regarding those summary statistics, culminating in a final transmittal of the summary statistics by USEPA in a letter dated October 20, 2010 (USEPA, 2010a).~~, and December 2010. The USEPA issued comments on the December 2010 draft in a letter to Honeywell dated May 17, 2011. Honeywell subsequently met with representatives of the USEPA and the Georgia Environmental Protection Division (GAEPD) on June 8, 2011 to discuss and resolve several outstanding issues. This version addresses the latest USEPA comments and incorporates the agreements reached at the June 8, 2011 meeting.

1.3 Purpose

The overall goal of this risk assessment is to develop essential scientific information that can be used in decision-making regarding the LCP Chemicals Site estuary in support of

an evaluation of the need for remedial action. To accomplish this goal, the specific objective of this assessment is to quantitatively evaluate whether constituents of potential concern (COPCs) detected in post-removal action sediment and consumable marsh biota at the property present a potential exposure[1] and health risk[2] to future trespassers of the property or consumers of LCP marsh biota. Note that a separate assessment is also being performed for the ecological receptors.

This document serves as a comprehensive update to past drafts of the HHBRA for OU1. Certain elements of the HHBRA may be described by reference to past HHBRA submissions, where agreement had been reached with USEPA on key elements of the assessment (such references should assist in the review of this version).

1.4 Report Organization

To the degree possible, all methods and procedures used in this evaluation are consistent with standard USEPA methods and procedures.

The report consists of the following sections:

1. *Introduction*. Report objectives; general approach~~.~~
2. *Pertinent Background Information*. Summary of historical land uses; description of the physical setting; description of the occurrence of chemicals at the property; and summary of environmental investigations~~.~~
3. *Data Analysis*. Description of the data selection and exclusion process for the risk analysis~~.~~
4. *Exposure Assessment*~~.~~
5. *Toxicity Assessment*~~.~~
6. *Risk Characterization*~~.~~
7. *Development of Remedial Goal Options*~~.~~
8. *Uncertainty Assessment*~~.~~
9. ~~*Risk Management Considerations*.~~
10. ~~9.~~ *References*~~.~~

[1] Exposure occurs when a person comes into direct contact with a chemical in an environmental medium (*e.g.,* soil, air). Exposure is quantified as the concentration of a chemical contacted in a medium averaged over the duration of the contact.

[2] Health risk is the probability of one or more harmful health effects occurring at either a measured or assumed level of exposure.

2.0 PERTINENT BACKGROUND INFORMATION

2.1 Site Background and History

The LCP property is located in Brunswick, Georgia and occupies approximately 813 acres.[3] Approximately 114 acres comprised the main contiguous area of former manufacturing operations at the Site (called the 'upland' area), while 670+ acres is occupied by tidal marshlands.

The upland area has been employed for industrial uses since 1919, beginning with the Atlantic Richfield Company (ARCO), who built a petroleum refining operation on the property. In 1937, 1942, and 1950, the Georgia Power Company (Georgia Power) acquired portions of the property. From 1941 to 1955, Dixie Paint and Varnish Company (subsequently the Dixie O'Brien Corporation and eventually a wholly owned subsidiary of the O'Brien Corporation) produced paints and varnishes on a portion of the property south of the Georgia Power site. In the mid 1950's, Allied Chemical (now Honeywell) acquired almost the entire property, and utilized it primarily for the production of caustic solutions, hydrogen gas, and chlorine gas. In 1979, LCP Chemicals-Georgia (LCP) acquired the property and continued the chlor-alkali manufacturing processes until operations ceased in early 1994. Honeywell repurchased the property in 1998 and currently owns the property.

Glynn County Planning Commission Land Use Maps show the property zoned as industrial property for both current and future use. Intended future land use for the property is continued industrial use.

2.2 Trespasser Access

The LCP marsh is surrounded primarily by industrial property. Access is limited by gate from the upland but accessible by watercraft from the Turtle River and marsh creeks. The upland and marsh are bordered by a county land disposal facility and a pistol firing range to the north, the Brunswick Pulp and Paper/Georgia-Pacific mill to the south, and Ross Road on the east and is defined as an industrial property. Access to the marsh from the upland is limited by fencing, onsite ~~personal~~personnel and security patrols during off hours.

[3] Based upon an updated property boundary survey by EMC Engineering Services, Inc. (2007).

3.0 DATA ANALYSIS

3.1 Overview

Analytical data from sediment and biota samples collected in the ~~in the~~ LCP marsh were used to identify constituents of potential concern (COPC) and to evaluate human exposure to those COPC. The initial data analysis for this HHBRA, including the identification of COPC and the derivation of exposure point concentrations (EPCs), was conducted by USEPA, and the results provided to Honeywell for use in the risk assessment (USEPA, 2010a).

3.2 Marsh Sediment

The sediment dataset used in this analysis was limited to samples of surface sediment (upper 15 cm) from the years 2000 through 2007 (i.e., following the marsh removal action of 1998-99). Sediment samples from the Turtle River and Purvis Creek domains were excluded as these areas remain inundated at low tide and afford no opportunity for exposure. Each result was treated as an individual sample; no averaging was performed. Sampling locations are shown in Figure 1.

Identification of COPC was conducted by comparing the maximum detected concentration of each constituent with the higher of: two-times the mean constituent-specific background concentration[4] (inorganics only) or the appropriate USEPA Regional Screening Level (RSL) for residential soil (USEPA, 2010b). Consistent with USEPA Region 4 guidance, RSLs based on non-cancer endpoints were adjusted to a target hazard quotient of 0.1 by dividing the RSL value by 10 (USEPA, 2000). It should be noted that the residential RSL for Aroclor 1254 was used to screen Aroclor 1268 since no values specific to Aroclor 1268 exist. Additional discussion of Aroclor 1268 toxicity is provided in ~~Section~~Sections 5 and 8.

Per USEPA Region 4 guidance, risk from carcinogenic polyaromatic hydrocarbons (cPAHs) was assessed in terms of benzo(a)pyrene toxic equivalents (BaP TEQ) ~~(USEPA, 2000b~~ rather than individual PAHs (USEPA, 2000b). The derivation of the B(a)P TEQ is provided in Table 2.

[4] ~~39~~Background concentrations for sediment were taken from the Human Health Baseline Risk Assessment for Marsh Sediment and Upland Soil, LCP Chemicals Site (Geraghty & Miller, 1999). These data represent the average concentration from a total of 38 background ~~marsh~~surface sediment samples ~~were~~ collected ~~by PTI Environmental Services (PTI, 1997). in~~ Jointer Creek (22 samples) and Clubbs Creek (16 samples), although not all analytes were included in all samples. For COPC selection, two-times the average background value was compared with the maximum detected concentration of inorganic constituents from site samples.

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Regarding Aroclors, early testing for the full Aroclor suite demonstrated that only Aroclor 1268 was present in the marsh sediment and in biota. Hence, subsequent sampling was limited to Aroclor 1268. Polychlorinated Biphenyl (PCB) homologue analysis of sediment and biota were presented in Kannan et al. (1997) and Kannan et al. (1998). The homologue proportions are substantially similar to the proportions in Aroclor 1268. More recent work indicates the same conclusions (Sajwan et al., 2008; Cumbee et al., 2008; Pulster and Maruya, 2008; Pulster et al., 2005).

For chemicals identified as ~~-~~COPC based on the screening described above USEPA's ProUCL software version 4.00.02 (USEPA, 2007) was used to calculate EPCs. For each COPC dataset, the ProUCL software evaluates the data distribution (e.g., normal versus lognormal), the proportion of the samples reported as non-detect, and the total number of samples, and provides a recommendation for a specific statistical method as the basis for the EPC. These recommendations were followed in all cases. The ProUCL EPC recommendations are summarized in Table 1. Detailed output from the ProUCL software is provided in Appendix A.

3.3 Seafood Tissue

The occurrence data for the constituents detected in finfish and shellfish collected from the Brunswick area and the Turtle River adjacent to the LCP Site are presented in Table 3. Only samples collected from ~~locations~~the LCP portion of the Turtle River estuary, identified as "Zone D (section of Turtle River from GA Highway 303 to Channel Marker 9)", "Zone H~~", "Zone I", and "~~ (Purvis Creek), and "Zone I" (Gibson Creek) were included. These fish and shellfish were collected between 2002 and 2006 following guidance provided in *Recommendations For A Fish Tissue Monitoring Strategy For Freshwater Lakes, Rivers, And Streams* from the Georgia Department of Natural Resources (GA-DNR) (FTAC, 1992~~) and is~~). The datasets are comprised of between 8 and 31 composite samples per species. The data consist of analytical results from fish species likely to be consumed by humans (e.g., red drum, spotted seatrout) as well as those less likely to be consumed (e.g., spot, striped mullet). The likelihood of consumption of a given species is based on a relative species harvest analysis of the Marine Recreational Fisheries Statistics Survey (MRFSS) data from 2001 through 2005. In addition, it continues to be common knowledge among recreational anglers that red drum and seatrout are more highly sought than are mullet or spot, both as game fish and fish for consumption. Additional discussion of the use of the MRFSS data is provided in Section 4.5. In addition to finfish, samples of ~~Blue Crab and White Shrimp~~blue crab and white shrimp were obtained and analyzed for PCBs, mercury, and other inorganics.

The COPC selection process applied for the seafood tissue data involved comparison of maximum detected constituent concentrations in fish and shellfish to USEPA Region 3 RSLs for fish ingestion (USEPA, 2010c). Following USEPA Region 4 guidance, for non-~~carcinogencs~~carcinogens, one-tenth of the fish ingestion RSL was used for screening (USEPA, 2000). It should be noted that the fish ingestion RSL for Aroclor 1254 was used to screen Aroclor 1268 since no values specific to Aroclor 1268 exist. Additional discussion of Aroclor 1268 toxicity is provided in Section 5. If the maximum detected concentration exceeded the RSL, the chemical was retained as a COPC. COPCs in finfish include Aroclor 1268 and mercury. COPCs in shellfish include Aroclor 1268, mercury, copper and zinc. The screening of COPCs in finfish and shellfish is provided in Table 3.

As with the marsh sediment data, EPCs in fish and shellfish were calculated using USEPA's ProUCL software version 4.00.02. Table 3 provides summary data, COPC selection, and EPCs for chemicals in fish and shellfish.

3.4 Clapper Rail (Rallus longirostris) Tissue

Clapper rail are small game birds living on the Atlantic coast (Figure 2). Clapper rail tissue was collected by USEPA from July through August 1995. A total of 16 clapper rail samples were obtained by USEPA sampling personnel from the most highly contaminated portion of the LCP marsh prior to the removal action. USEPA also collected 7 clapper rail from an off-Site reference area along Troup Creek. The USEPA sampling and analysis protocol included analysis for PCBs (specifically Aroclor 1268) and mercury. For purposes of the human health risk assessment, only the data from the breast tissue (the tissue generally consumed by humans) were included in the data set, providing a sample number of 14. The occurrence summaries for the clapper rail constituent concentrations at the Site are presented in Table 3. For screening of COPCs, the USEPA Region 3 RSLs for fish ingestion were used. ProUCL version 4.00.02 was used to calculate EPCs. It should be noted that for Aroclor 1268, ProUCL recommended an EPC based on the 99% Chebychev method, which corresponded to a value 19.94 mg/kg. However, this value exceeds the maximum detected concentration of 19.42 mg/kg. The maximum ~~detetected~~detected concentration was used for the intake calculations.

EPS

4.0 EXPOSURE ASSESSMENT

4.1 Overview

An exposure assessment was conducted as part of the health risk assessment to evaluate the potential exposure pathways at the LCP Site. An exposure pathway is defined by the following four elements: (1) a source and mechanism of constituent release to the environment; (2) an environmental transport medium for the released constituent; (3) a point of potential contact with the contaminated medium (the exposure point); and (4) an exposure route at the exposure point. The purpose of the exposure assessment is to estimate the way a population may potentially be exposed to constituents at a site. The conceptual site model (CSM) discussed below is specific to contact with the marsh sediment and fish and game consumption. The general CSM was presented in the earlier risk assessment previously reviewed by USEPA Region 4 and Georgia Environmental Protection Division (GAEPD) (Geraghty & Miller, 1999).

4.2 Conceptual Site Model

The conceptual site model provides the framework of the risk assessment. Generally, it characterizes the primary and secondary potential sources and release mechanisms and identifies the primary exposure points, receptors, and exposure routes. Receptors may include any living organism (plant, animal, and human). This risk assessment focuses on potential human exposure to COPCs detected in sediment and biota collected at, and adjacent to, the LCP Site. Exposure points are places or "points" where exposure could potentially occur, and exposure routes include the basic pathways through which COPCs may potentially be taken up by the receptor. Please note that the risk evaluation for fish and shellfish consumption in this section includes only these direct consumption pathways for contacting chemicals. Figure 3 shows a diagram of the simplified conceptual site model for the marsh trespasser and fish and game consumers.

Although analytical data for surface water do exist, it is not appropriate to include ingestion of surface water in a tidal marsh because the concentrations of whatever might be in the water would change with each tidal cycle, and any measured concentration would be meaningless relative to long term exposure. The existing surface water data for Aroclor 1268 at 12 locations ranges from non-detect to 0.18 micrograms per liter (µg/L). Aroclor 1268 is more similar toxicologically to Aroclor 1016 than to Aroclor 1254. The recreational water PRG for Aroclor 1016 obtained from the RAIS website based on noncancer effects in 790 µg/L. The recreational water PRG for Aroclor 1016 obtained from the RAIS website based on cancer effects for Aroclor 1016

is 66 μg/L. Both are orders of magnitude above the maximum detected surface water concentration of 0.18 μg/L.

A similar issue exists with respect to the evaluation of dermal contact with surface water. In addition, the implementation memo for *Risk Assessment Guidance for Superfund (RAGS), Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)* (RAGS-E) (USEPA 2004a) indicates that for chemicals such as PCBs that have permeability coefficient (Kp) values outside the effective prediction domain, quantitative estimates of risk may be inaccurate. Hence, the appropriate qualitative statement of risk is that there may be some risk from dermal contact with surface water for the marsh trespasser; however, the ever-changing, tidally influenced and unknown concentrations in surface water and the lack of a credible exposure assessment methodology preclude any meaningful quantitative risk estimate for this pathway.

4.3 General Exposure Assumptions

To provide some understanding of the range of exposures and consequent risks, scenarios based on both reasonable maximum exposure (RME) and central tendency exposure (CTE) were evaluated. Standard default values for assessing risk that generally lead to the RME risk estimates were used (USEPA, 1991, 1997a). In several guidance documents, USEPA indicated that the RME approach is incomplete by presenting only a point estimate of risk with no indication of where it falls within the risk distribution (USEPA, 1992, 1997a, 2000).

The concept of RME provides an estimate of the highest reasonable exposure possible to an individual. Such an individual is defined as the RME receptor and is generally considered to be at the 90th percentile of the exposure distribution or higher whereas CTE provides a midrange estimate.

4.4 Marsh Sediment Exposure Assumptions and Exposure Model

It is important to note that exposure to sediment is not similar to exposure to surface soil. In fact, Region 4 EPA's Supplemental Guidance to RAGS indicates that in most cases, it is unnecessary to assess the risk of human exposure to sediment (USEPA, 2000b). Sediment occupies the skin surface for only a brief time before one's foot is moved into the water column and sediment is rinsed away. In addition, exposure to surface soil occurs by incidental ingestion from the hands. In the case of sediment, the water washes away or mixes the sediment on the hands and feet as they are withdrawn. In the case of an individual becoming really muddy, it is unlikely that this individual would put his hands on his face. There are subjective reports of soil in the

mouth being gritty and unpleasant in quantities as low as 10 mg (Kissel et al., 1996; Holmes et al., 1999). An individual crabbing or moving through the marsh would be reluctant to place his or her filthy hands near the face. Perhaps some tiny bits of mud caught beneath fingernails might later make its way to a receptor's mouth and be ingested. Regardless of these practical behaviors, sediment ingestion rates were assumed to be similar to those for residential soil - 100 mg/day for adult and adolescent receptors ~~and 200 mg/day for child receptors~~. Given the nature of sediment in the marsh, as discussed above, ~~these are~~this is a conservative assumption~~s~~.

This is a tidal marsh. Instead of seasonal periods, the marshes are covered by water about every 12 hours. Some areas of the marshes will be wet for longer periods than others depending on their elevation relative to the tidal change. Drought conditions do not affect the degree of diurnal seawater inundation of the marsh.

The marsh is a difficult place to negotiate on foot with any modicum of safety. There are many warnings about the dangers of "ploof" mud in the local newspapers along the Georgia and South Carolina coast. The sediment in the coastal marsh is often just like quicksand and individuals who choose to walk in the marsh may sink up to their waists or deeper. ~~For this reason, an exposure frequency of 6 days per year was chosen based on discussions with USEPA and GAEPD personnel.~~Based on discussions with USEPA and GAEPD personnel, exposure frequencies of 52 days per year and 6 days per year were selected for the RME and CTE trespasser receptors, respectively.

Exposure concentrations in sediment are also different than those in soil because of the high water content of sediment. In ecological risk assessment, moisture content of sediment and soil samples is routinely used to adjust laboratory-reported dry weight concentrations to wet weight concentrations. That procedure was also performed here because the receptor contacts wet sediment and hence, wet weight concentrations are more representative of the actual exposure situation. Please note that this adjustment is appropriate for dermal exposure to sediment but not for ingestion exposure. This method was used by USEPA in 2004 to assess sediment exposure by the dermal route to hydrophobic chemicals such as Aroclor 1268 and PAHs (USEPA, 2004b). Basically, the concentration is reduced by the percent of water as follows:

$$Concentration\,(wet\ weight) = \frac{Concentration\,(dry\ weight)}{100\% + Percent\ \ Moisture} \qquad \text{(Eq. 1)}$$

Hydrophobic chemicals will tend to distribute among the various size particles of sediment according to the organic carbon content of the particular size fraction. This estimation is necessary because the upper bound of skin loading depends on particle

size. This would be the case for Aroclor 1268 and PAHs. Metals in sediment would likely not show as much size partitioning. As a conservative measure, 100% of the total mercury present was assumed to be methylmercury. A size partitioning factor can calculated as follows:

$$Partition\ Factor = \frac{Percent\ Size\ Fraction_i}{\sum_{i=1}^{n} Percent\ Size\ Fraction} \quad \text{(Eq. 2)}$$

Equations 1 and 2 were combined as:

$$Effective\ Conc.\ (wet\ wt) = \frac{Concentration\ (dry\ weight)\ x\ Partition\ Factor}{Percent\ Size\ Fraction\ x\ (100\% + Percent\ \ Moisture)} \quad \text{(Eq.3)}$$

Grain size fractions along with total organic carbon (TOC) measurements were available for 26 separate sampling locations from the 2006 sampling event. Particles less than 0.075 mm in diameter are those that adhere to the skin to the greatest extent (USEPA, 2004b). Size fractions were available for these data for grain sizes greater than 0.075 mm in addition to separation into coarse, medium and fine sand as well as fines and gravel. There were statistically-significant correlations between TOC and the various sediment types in the sample (Table 4). TOC was positively correlated with fines and with gravel and negatively correlated with medium sand and fine sand. The conclusion is that the organic carbon in the sample is primarily in the fines. These small particles would also be trapped on the surface of the gravel particles and hence, be entrained in the gravel sample. It was assumed that all organic carbon in each sample was present as fines.

The organic carbon in fines was adjusted upward by dividing percent TOC by percent fines. Percent moisture was obtained from another set of 26 separate locations also obtained in 2006 (Table 5). Percent moisture showed a low variability and the mean of these data were used to represent percent moisture in all sediment.

Equation 3 was used to calculate effective concentrations using the EPC values. Effective concentrations were determined for Aroclor 1268 and carcinogenic PAHs only.

Calculation of Dermal Absorption per Event

The dermal absorbed dose per event was calculated as:

$$DA_{event} = C_{Effective} \cdot 10^{-6}\ {}^{kg}/_{mg} \cdot SAF \cdot ABS_i \quad \text{(Eq. 4)}$$

Table C-4 in RAGS-E gives the maximum particle loading per size of particle (USEPA, 2004a). The average maximum loading for particles less than 0.075 mm, i.e. the fines, calculated from Exhibit C-4 in RAGS-E is 13 mg/cm^2. This was used as the value for

SAF, skin adherence factor. Note that this value is quite similar to that for Children-in-Mud from Exhibit C-3 in RAGS-E. Also note this value is about 20-fold higher than the value of 0.07 mg/cm^2 usually used for soil dermal pathway. Table 6 provides the calculation of DA_{event}.

"ABS Fraction" is the dermal absorption fraction for the COPC as reported by USEPA (2010b). These values are 0.14 for Aroclor 1268 and 0.13 for PAHs, including the benzo(a)pyrene equivalents used herein. For all metals evaluated in this risk assessment, dermal ABS Fraction values of zero were assigned per USEPA (2010b).

For the metals, DA_{event} was calculated using the EPCs without adjusting to an effective concentration.

For completeness, a sample calculation for DA_{event} for Aroclor 1268 is provided below.

$$DA_{event} = \frac{Partition\ Factor}{\%Size\ Fraction} \times \frac{EPC}{100\% + \%Moisture} \times 10^{-6\ kg/mg} \times SAF \times ABS_i$$

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This is a combination of equations 1-4 above.

The calculation of DA_{event} for Aroclor 1268 is as follows:

$$DA_{event} = \frac{Partition\ Factor}{\%Size\ Fraction} \times \frac{EPC}{100\% + \%Moisture} \times 10^{-6\ kg/mg} \times SAF \times ABS_i$$

$$= 7.55\% \times \frac{2.571\ mg/kg}{100\% + 67.82\%} \times 10^{-6\ kg/mg} \times 13\ mg/cm^2 \times 0.14$$

$$= 7.55\% \times 1.53\ mg/kg \times 10^{-6\ kg/mg} \times 13\ mg/cm^2 \times 0.14$$

$$= 2.11E-07$$

Calculation of Dermal and Oral Doses

The exposure assumptions for the marsh trespasser scenario are shown in Table 7.

The Dermal Absorbed Dose (DAD) is calculated as:

$$DAD = \frac{DA_{event} \times EF \times ED \times EV \times SA}{BW \times AT} \qquad \text{(Eq. 5)}$$

EF is the exposure frequency in days/yr, ED is the exposure duration in years, EV is the events/day and SA is the skin surface area. BW is body weight and AT is averaging time. The following inputs were used for the RME receptors:

	EF	~~6~~52 days/yr
	ED	30 yr for the adult
		10 yr for the adolescent
	~~EV~~	~~2 yr~~BW 70 kg for the ~~child~~adult
	~~BW~~	~~1/day~~
		~~70kg,~~ 45 kg ~~or 15 kg~~

~~(adult;~~for the adolescent~~; child)~~

AT	25550 days for cancer
	ED*365 for noncancer
SA	3870 cm^2 for the adult
	2559 cm^2 for the adolescent
	~~1101 cm^2 for the adult~~

SA was determined as the skin surface area of the feet and lower legs. These values were 3870 cm^2 for adults~~,~~ and 2559 cm^2 for adolescents ~~and 1101 cm^2 for children~~. They were suggested by GAEPD and were calculated based on Exhibit C-1 of RAGS-E. For Aroclor 1268 and PAHs, it was assumed that only the fines clung to the skin.

The oral dose is given by:

$$Oral\ Dose\left(\frac{mg}{kg-day}\right)=\frac{C_{sed}\times IR_{sed}\times EF\times ED\times CF}{BW\times AT} \quad \text{(Eq. 6)}$$

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C_{sed} is the concentration in sediment in mg/kg and IR_{sed} is the sediment ingestion rate in mg/day. CF is a conversion factor to obtain the appropriate units.

IR_{sed}	100 mg/day (adults and adolescents)
~~IR_{sed}~~	~~200 mg/day (children)~~
CF	1*E*-06 kg/mg

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For noncarcinogens, Eq. 6 was applied to adults~~,~~ and adolescents ~~and children~~ separately. For carcinogens, the dose was apportioned to each age group separately. ~~The use of only 2 years as the ED for the child, as noted above, is based on the fact that children less than four are very unlikely to wade in the soft mud of a tidal marsh.~~ The dermal-specific toxicity criteria are then applied to Eq. 5 to obtain the dermal risk estimate. The oral toxicity criteria are applied to Eq. 6 to obtain the oral risk estimate. The lifetime receptor cancer risk was calculated by combining the risk for the individual age categories. To achieve a residential lifetime span of 30 years, the adult risk was multiplied by 0.~~6~~67 and added to the ~~child risk and~~ adolescent risk (RME receptors only).

Tables 8a and 8b (RME and CTE cases, respectively) provide the intake doses of carcinogens and resulting cancer risk estimates for the Marsh Trespasser scenario.

Tables 9a and 9b provide the intake doses of systemic toxicants and resulting noncancer hazard indices for the Marsh Trespasser scenario.

4.5 Fish Consumption Exposure Assumptions and Exposure Model

For the fish consumption risk assessment, both RME and CTE exposure assumptions (Table 10) were developed from USEPA (1997a) and other sources (DHHS, 1999; Appendix ~~A~~B). The goal in providing both RME and CTE risk estimate is to inform the risk decision makers about the potential range of risks associated with the site (USEPA, 1992; 2000).

Fish Consumption Rates

In this risk assessment for fish consumption, values reflecting the southeastern United States were used to represent recreational fish consumers (USEPA, 1997a). ~~Site-specific~~As an additional measure, information on seafood consumption from the Brunswick area obtained by the Agency for Toxic Substances and Disease Registry (ATSDR) and the Glynn County Health Department ~~in 1997~~(GCHD) was used to develop exposure assumptions for ~~subsistence~~hypothetical "high quantity"[5] fish consumers.

In 1998, the ~~Glynn County Health Department~~ ATSDR and GCHD conducted a survey to assess ~~fish~~ consumption of locally harvested seafood and mercury intake (DHHS, 1999). ~~Data on fish consumption from this survey was used to develop exposure assumptions for the so-called subsistence consumer. Because no clear definition exists for the subsistence fish consumer and because the Brunswick population contained some self-identified subsistence fish consumers, this dataset was used as a basis for the subsistence exposure factors.~~ Because this study included two self-identified "subsistence"[6] fishers, this dataset was used as a basis for the fish ingestion rates for the hypothetical high quantity fish consumer receptor. These estimates are shown in Table 10 and their derivation is presented in Appendix ~~A~~B.

Proportions of Species Consumed

The Marine Recreational Fisheries Statistics Program of the Office of Science and Technology within National Oceanic and Atmospheric Administration (NOAA) conducts

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[5] The term "high quantity" is used in this risk assessment to describe consumers who consume more locally-caught fish than the typical recreational angler.
[6] The GCHD/ATSDR study (2000) states that "subsistence fishers catch seafood as the primary source of their dietary protein."

the Marine Recreational Fisheries Statistics Survey (MRFSS) to produce catch, effort and participation estimates and to provide biological, social and economic data (NMFSS, 2007). USEPA made use of these data obtained from 1986 to 1993 to determine estimates of consumption of marine fish (USEPA, 1997a).

The MRFSS consists of a telephone survey and an intercept or creel survey conducted on two-month intervals. These two-month intervals are called waves. The period of two months was chosen because it was the maximum time for easy recall of past fishing trips. The intercept data from 2001 through 2005 was used here. These data are freely available on the internet (NMFSS, 2007).

A recent study by the National Academy of Science revealed that the MRFSS was flawed in its execution and the data generated are inaccurate and biased (NAS, 2006a). The criticisms by the NAS were several: (1) sampling and statistical issues, such as failure to include anglers with access to private property and the use of different survey methods in different states; (2) lack of reliable human dimensions data, such as social, behavioral, attitudinal and economic data; (3) lack of coordination between federal and state personnel and "balkanization" of the survey methods and designs; and (4) the need for improved communication and outreach with anglers.

Even if the MRFSS data were reliable, its use would entail an estimation of consumption from the harvest. Others have attempted to perform this estimation and there is considerable uncertainty in the procedure (Rupp et al., 1980; ChemRisk, 1992; Ebert et al., 1993; Price et al., 1994). If MRFSS data from a sufficiently large area is included, it is appropriate to use MRFSS data to obtain the relative abundance of species in the overall catch. The proportion of various species in the MRFSS data would reflect both the relative abundance of various species and angler success. Table 11 shows the average percentage of the various species of fish caught by coastal Georgia anglers between 2001 and 2005 developed from the MRFSS data. The MRFSS data is available from the NOAA Fisheries website (http://www.st.nmfs.gov) as SAS export files.

Because the concentrations of COPCs are different in different species of fish, likely due to their feeding strategies, it is important to weight the species-specific exposure point concentrations according to angler success and preferences. This procedure is made quite simple by the use of a Fraction Ingested (FI) term applied to individual fish species as shown on Tables 12a-c,13a-c,14a-c, and 15a-c.

Concentrations in Finfish and Shellfish

Exposure point concentrations in fish were the 95% UCL of the arithmetic mean concentration calculated by a variety of statistical methods that were recommended by ProUCL. ~~The~~These values are shown in Table 3.

The effects of attenuation processes which would reduce the concentrations in fish and shellfish over time are not considered. Because the COPCs have been present at the Site for many years, any attenuation by fate and transport mechanisms is already reflected in the on-Site concentrations and in the EPCs. ~~With the exception of mercury that may be deposited in the marsh and nearby water bodies by atmospheric deposition, COPCs in fish (e.g. Aroclor 1268) may continue to decline over time. This attenuation is discussed more fully in Section 8.~~

4.6 Dose Calculation for Fish Consumption

The exposure dose was estimated for carcinogens as follows:

$$ADD\left(\frac{mg}{kg-day}\right) = \sum_{i=1}^{species} \frac{P_i \times C_i \times FCR \times EF \times ED \times CF}{BW \times AT} \qquad \text{(Eq. 7)}$$

where,

C_i	=	Concentration in ith fish species (mg/kg)
P_i	=	Proportion of the ith species in the total catch (%)
FCR	=	Fish Consumption Rate (g/day)
EF	=	Exposure Frequency (days/yr)
ED	=	Exposure Duration (yr)
BW	=	Body Weight (kg)
CF	=	Conversion Factor (kg/g) = 1E-03
AT	=	Averaging Time (days)

The unit analysis for Eq. 7 is as follows:

$$ADD\left(\frac{mg}{kg-day}\right) = \sum_{i=1}^{species} \frac{\% \times {mg}/{kg} \times {g}/{d} \times {d}/{yr} \times yr \times {kg}/{g}}{kg \times d} = \frac{mg}{kg-d}$$

The exposure dose was estimated for noncarcinogens as follows:

$$ADD\left(\frac{mg}{kg-day}\right) = \sum_{i=1}^{species} \frac{P_i \times C_i \times FCR \times CF}{BW} \qquad \text{(Eq. 8)}$$

The unit analysis for Eq. 8 is as follows:

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$$ADD\left(\frac{mg}{kg-day}\right)=\sum_{i=1}^{species}\frac{\%\times {}^{mg}/_{kg}\times {}^{g}/_{d}\times {}^{kg}/_{g}}{kg}=\frac{mg}{kg-d}$$

Details of the risk estimation for consumption of finfish are provided in Tables 12a-c (RME Recreational), 13a-c (CTE Recreational), 14a-c (RME ~~Subsistance~~High Quantity), and 15a-c (CTE ~~Subsistance).~~High Quantity). Details of the risk estimation for consumption of shellfish are provided in Tables 16 (RME) and 17 (CTE).

4.7 Clapper Rail Exposure Assumptions and Exposure Model

Residents living in the vicinity of the LCP Site could potentially obtain game from areas adjacent to the marsh. Similar to the seafood scenario, it is unlikely that individuals would hunt an appreciable amount in the vicinity of the Site due to the close proximity of more desirable and accessible areas. The USEPA and GAEPD requested at the time of the previous risk assessment (Geraghty & Miller, 1999) that potential risks associated with ingestion of clapper rails (*Rallus longirostris*) obtained in the vicinity of the LCP Site be evaluated. According to United States Fish and Wildlife (USFWS) representatives, although the clapper rail is hunted, individuals do not commonly consume clapper rails due to their small size and lack of culinary satisfaction (Bowers, 1997, as cited in Geraghty & Miller, 1999). However, as a conservative measure in response to the request by USEPA and GAEPD, potential risks associated with clapper rail ingestion were assessed in the previous risk assessment and also here.

~~Specific data regarding the amount of clapper rail ingestion were not available. This was not surprising, however, since local sportsmen and the GA DNR indicated that clapper rail are generally hunted for sport and not as an edible game bird. An informal internet search using Google® found two recipes for clapper rail breasts -- one where the tiny morsels were wrapped in bacon and served on a bed of rice; mention was made of the darkness of the breast meat and its gamey taste.~~

In order to estimate an ingestion rate for clapper rail, it was assumed that a wildlife consumer would obtain 10% of total game ingestion solely from clapper rail obtained near the LCP site. Data for total game ingestion were obtained from Table 11-6 in USEPA's *Exposure Factors Handbook* (USEPA, 1997a). The CTE value was assumed to be the mean and the RME value was assumed to be the mean plus two standard errors. The standard error was greater than the mean in all cases. Refer to the Section 8.7 for a discussion of how this issue contributes to the uncertainty of the RME risk estimates. Consumption for adults, adolescents and children were calculated in terms of g/day. Similar to the previous risk assessment, it was assumed that 10% of these

ingestion rates reported in the Exposure Factors Handbook represented clapper rail consumption. The consumption rate estimation is shown in Table 18. The details of the intake dose and risk/hazard calculation are shown in Table 19 (RME) and Table 20 (CTE).

5.0 TOXICITY ASSESSMENT

5.1 Overview

This section discusses the two general categories of toxic effects (non-carcinogenic and carcinogenic) evaluated in risk assessments and the toxicity values used to calculate potential risks. Toxicity values for potential non-carcinogenic and carcinogenic effects are determined from available databases. For this risk assessment, toxicity values were first obtained from the USEPA's Integrated Risk Information System (IRIS). If toxicity criteria were not available in IRIS, other sources were consulted following a recommended hierarchy of toxicity values (USEPA, 2003).

5.2 General Toxic Effects

A distinction is made between carcinogenic and non-carcinogenic effects. For potential carcinogens, the previous regulatory guidelines (USEPA, 1989) use the linearized multistage model that assumes that any level of exposure to a carcinogen potentially could cause cancer. This point of view is changing and the 2005 Guidelines for Carcinogen Risk Assessment stress that knowledge of the mode of action is all important in the development of toxicity criteria (USEPA, 2005).

5.3 Non-Carcinogenic Effects

For many non-carcinogenic effects, protective mechanisms must be overcome before an effect is manifested. Therefore, a finite dose (threshold), below which adverse effects will not occur, exists for non-carcinogens. A single compound might elicit several adverse effects depending on the dose, the exposure route, the duration of exposure, and the susceptibility of the individual. Chemicals may exhibit their toxic effects at the point of application or contact (local effect), or they may exhibit effects at other sites (systemic effects) after they have been distributed throughout the body. Most chemicals can produce more than one type of toxic effect depending on the dose and the susceptibility of the exposed individual or receptor. The potential for non-carcinogenic effects is estimated by comparing a calculated exposure dose to an RfD or reference concentration (RfC) for each individual constituent. The RfD or RfC represents a daily exposure level which is designed to be protective of human health, even for sensitive individuals or subpopulations over a lifetime of exposure.

For a given chemical, the dose or concentration that elicits no adverse effect, usually in an animal bioassay, is referred to as the "no observed adverse effect level" (NOAEL). The lowest dose or concentration at which adverse effects are noticed is referred to as the “lowest observed adverse effect level” (LOAEL). Either the NOAEL or LOAEL is used to establish non-cancer toxicity values (RfDs) for oral or dermal exposure and

RfCs for inhalation exposure. The RfD and RfC represent a daily exposure level, within an order of magnitude, that is not expected to cause adverse health effects in any humans (USEPA, 1989). The RfD is an estimated oral dose of a chemical that is unlikely to cause adverse health effects. RfCs and unit risks are not discussed any further because none of the exposure scenarios in this risk assessment involve inhalation of chemicals. The uncertainty factor represents areas of uncertainty inherent in the extrapolation from the available data. The confidence levels (low, medium, high) assess the degree of confidence in the extrapolation of available data.

5.4 Carcinogenic Effects

Cancer induction in humans and animals by chemicals proceeds through a complex series of reactions and processes. Potentially carcinogenic chemicals may produce tumors at the point of application or contact, or they may produce tumors in other tissues after they have been distributed throughout the body. Some chemicals are associated only with one or two tumor types while others may cause tumors at many different sites.

One of the fundamental problems in cancer risk assessment is extrapolating from animal data to effects on humans. Typically, the USEPA extrapolates data from laboratory studies in which animals (usually rodents) have been exposed to the chemical in question. Epidemiological data are generally not used by USEPA to develop toxicity values because the studies do not have enough statistical power.

To develop cancer slope factors, USEPA extrapolates from observed laboratory animal data using mathematical models of dose-response. These models estimate a point-of-departure level, usually the 10% response level. The dose at the point-of-departure is known as the benchmark dose. Statistical 90% confidence limits around the point of departure level are developed and the slope of the line from the lower confidence limit on the benchmark dose through the origin is the slope factor. Hence, the cancer slope factor is the 95% upper bound on the slope of the dose-response curve in the low dose region. In the new Cancer Guidelines, USEPA recommends gaining an understanding of the mode of action in lieu of the default assumption of linearity (USEPA, 2005). Not all the values on IRIS reflect the emphasis on understanding the mode of action that is prescribed in the new Cancer Guidelines.

Chemical constituents are classified as known, probable, or possible human carcinogens based on a USEPA weight-of-evidence scheme in which chemicals are systematically evaluated for their ability to cause cancer in humans or laboratory

animals. The USEPA classification scheme (USEPA, 1989) contains five classes based on the weight of available evidence, as follows:

A known human carcinogen;
B probable human carcinogen:
B1 probable human carcinogen -- limited evidence in humans;
B2 probable human carcinogen -- sufficient evidence in animals and inadequate data in humans;
C possible human carcinogen -- limited evidence in animals;
D inadequate evidence to classify; and
E evidence of non-carcinogenicity.

This classification has been updated in USEPA's Guidelines for Carcinogen Risk Assessment (USEPA, 2005) and is slowly being replaced by the descriptors "*Carcinogenic to Humans*," "*Likely to Be Carcinogenic to Humans*," "*Suggestive Evidence of Carcinogenic Potential*," "*Inadequate Information to Assess Carcinogenic Potential*," and "*Not Likely to Be Carcinogenic to Humans*." IRIS remains to be updated in this regard.

5.5 Toxicity Values

Whenever possible, route-specific toxicity values have been used. However, toxicity values for dermal exposures have not yet been developed by USEPA; therefore, the oral toxicity values were used to derive adjusted toxicity values for use in assessing dermal exposure. The use of adjusted toxicity values represent the theoretical toxicity of the orally absorbed dose of the constituent based on the oral toxicity value and the assumed or measured gastrointestinal absorption (GI_{ABS}) in the study underlying the NOAEL or LOAEL. Thus, the calculated RfD and Cancer Slope Factor (CSF) values are:

$$RfD_a = RfD_o \times GI_{ABS} \quad \text{(Eq. 9a)}$$

$$CSF_a = CSF_o / GI_{ABS} \quad \text{(Eq. 9b)}$$

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This approach is discussed in detail in Appendix A of USEPA (1989) and in USEPA (2004a). Chemical-specific GI_{ABS} values were available for all COPCs in marsh sediment (USEPA, 2010b).

The hierarchy of sources of toxicity values recommended by USEPA was used to obtain toxicity criterion (USEPA, 2003) with the exception of Aroclor 1268. Toxicity profiles below indicate the source of toxicity criteria used in this risk assessment. A summary of the toxicity criteria used and their sources is presented in Table 21.

5.6 Aroclor 1268

IRIS contains values for the cancer slope factor for PCB mixtures and reference doses for Aroclor 1016 and Aroclor 1254 only. Both a cancer slope factor value and a reference dose for Aroclor 1268 are available in the peer reviewed literature. This source would be identified as ~~tier~~Tier 3 in USEPA's hierarchy of toxicity values. OSWER directive 9285.7-53 (USEPA, 2003) states in this regard:

> *Priority should be given to sources that provide toxicity information based on similar methods and procedures as those used for Tier I and Tier II, contain values which are peer reviewed, are available to the public, and are transparent about the methods and processes used to develop the values.*

Although there exist peer reviewed articles in the journal Regulatory Toxicology and Pharmacology on Aroclor 1268 that fulfill these requirements and do indeed use similar methods and processes, these values have not yet been placed in the IRIS database (Warren et al., 2004; Simon et al., 2007). Hence, the RfD value for Aroclor 1016 on the IRIS database (7E-05 mg/kg-day) was used as a surrogate toxicity criterion for Aroclor 1268. A further discussion of the choice of the Aroclor 1016 RfD and not the Aroclor 1254 RfD is presented below and in Section 8.

5.6.1 Cancer Slope Factor for Aroclor 1268

PCBs are classified as B2, a probable human carcinogen. The current PCB carcinogenicity assessment is based on dose-response cancer bioassays of Aroclor mixtures performed in rodents in 1996. USEPA used these studies to develop cancer slope factors (USEPA, 1996). Two slope factors were derived – one for high risk and persistence mixtures and the other for low risk and persistence mixtures. The values are 2.0 per mg/kg-day and 0.4 per mg/kg-day respectively. IRIS recommends using the high risk and persistence value for soil contact risk assessment. This value of 2.0 per

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mg/kg-~~d~~day was also used for contact with PCBs in marsh sediment and PCBs consumed in fish.

5.6.2 Reference Dose for Aroclor 1268

The determination of whether Aroclor 1268 is more similar on a toxicological basis to Aroclor 1016 or Aroclor 1254 would determine the choice of a surrogate toxicity value. As will be shown below, Aroclor 1268 is more similar on a toxicological basis to Aroclor 1016 than to Aroclor 1254. Hence, the RfD for Aroclor 1016 was used.

To examine the potential similarities between the three mixtures, three modes of action (MOAs) were considered:

- A dioxin-like MOA characterized by binding to the aryl hydrocarbon receptor and quantified by dioxin TEQ (van den Berg et al., 2006);
- An MOA based on binding to the ryanodine receptor and consequent interference with cellular calcium homeostasis (Pessah et al., 2006; Simon et al., 2007); and
- An MOA based on binding to trans-thyretin, a plasma thyroid binding protein, and subsequent increase metabolism of thyroxin (Chauhan et al., 2000).

In addition to these three MOAs, the effect of bioaccumulation and metabolism in humans was considered. Bioaccumulation and metabolism of PCBs was first quantified in the 1990s based on examination of tissue concentrations in relatively lightly exposed capacitor workers versus heavily exposed Yusho and Yucheng patients (Brown et al., 1989; Lawton et al., 1985a,b). A scheme of PCB metabolism was developed that now appears quite accurate when compared with recent data on congener measurements in humans (Brown, 1994; Brown et al., 2007; Park et al., 2007). Distribution data from Park et al. (2007) were normalized to the concentration of PCB153 in plasma because this is the most prevalent congener in humans. In this way, values between zero and one were developed for all 209 congeners. If a congener was not detected, it was assigned a value of zero. One can think of these values as a bioaccumulation "equivalent" for humans. The rationale for using this "bioaccumulation equivalence" scheme is that because PCBs tend to persist in humans, toxic effects are due to long term exposure.

Congener concentrations in Aroclor 1016, 1254 and 1268 were obtained from Anderson (1991) and Frame et al., (1996). For each MOA, the value of the relative potency of each congener was multiplied by the congener bioaccumulation equivalent and the congener concentration in Aroclor 1016, Aroclor 1254 and Aroclor 1268. For each

mixture, the sum of these values represented the potential for the particular Aroclor mixture to produce toxicity specific to each MOA. ~~Table 22 shows the amount of each bioaccumulated equivalent value in the mixture. As can be seen,~~ Aroclor 1254 has more of each type of bioaccumulated equivalent and contains about 1 order of magnitude more of both bioaccumulated neurotoxic equivalents and bioaccumulated thyroid hormone equivalents than either of the other two mixtures. Additional details of this analysis are provided in Section 8.

The conclusion is that the reference dose for Aroclor 1016 is more likely to reflect the toxicity to humans than is the reference dose for Aroclor 1254 and the RfD of 7E-05 mg/kg-~~d~~day was used as a surrogate toxicity criterion for Aroclor 1268.

5.7 Polycyclic Aromatic Hydrocarbons

These ubiquitous chemicals have a clear carcinogenic endpoint and are represented in the quantitative risk evaluation as benzo(a)pyrene equivalents. Benzo(a)pyrene has an oral cancer slope factor on IRIS and is classified as B2, a probable human carcinogen. IRIS indicates that human data on the carcinogenicity of benzo(a)pyrene is inadequate to demonstrate the chemical is responsible for human cancer. This assessment of inadequacy stems from the fact that benzo(a)pyrene occurs as part of a mixture of chemicals and may not be the sole carcinogen present. However, PAHs and benzo(a)pyrene occur in cigarette smoke, roofing tar and coke oven emissions, and few would argue that cigarette smoking and lung cancer are unrelated. Tumors have occurred in rodents from administration of benzo(a)pyrene by a variety of exposure routes. The data are considered sufficient for quantitative analysis and the oral cancer slope factor on IRIS is 7.3 per mg/kg-day.

5.8 Mercury

Mercury is known to exist in sediment in equilibrium between inorganic forms and methylmercury. For all exposure scenarios considered here - sediment exposure, fish consumption or clapper rail consumption - all mercury was assumed to be present as methylmercury. The reference dose for methylmercury is available on IRIS and is 1E-04 mg/kg-day. The RfD for methylmercury was completed in 2001 and is based on the occurrence of neurodevelopmental effects from several epidemiological studies. The development of the RfD is available on IRIS and also in USEPA's Mercury Study Report to Congress (USEPA, 1997b).

5.9 Aluminum

The primary toxicological effect of aluminum is neurotoxicity. This effect was first observed in patients in the early days of renal dialysis – patients developed dementia

within 6-9 months. Removal of aluminum from the dialysis fluid decreased the incidence of dementia. The critical endpoint for the provisional reference dose for aluminum is the occurrence of developmental neurotoxicity in mice observed in several studies. The LOAEL from the mouse studies was 100 mg/kg-day. The combined uncertainty factor was 100 resulting in an RfD of 1 mg/kd-day. The full derivation is provided in the professional peer-reviewed toxicity value (PPRTV) document for aluminum (USEPA, 2006).

5.10 Chromium

In keeping with previous versions of this HHBRA, total chromium detected in sediment was evaluated as hexavalent chromium (VI) for purposes of both COPC screening and risk characterization. The oral RfD is based on a NOAEL in a drinking water study in rats. This value was chosen rather than that of trivalent chromium (III) because it is lower (i.e., more conservative), reflecting the greater toxicity of chromium (VI) compared with chromium (III). The ~~May~~December 2010 version of the RSLT (USEPA, 2010b) incorporates a new oral cancer slope factor for chromium (VI). This value was developed by the New Jersey Department of Environmental Protection and is based on an increased incidence of tumors of the small intestine in mice exposed to chromium (VI) in a drinking water study conducted by the United States National Toxicology Program.

The use of these toxicity values for chromium (VI) makes for an extremely conservative assessment. Although the there are no site-specific data available on the speciation of chromium in the sediment in the LCP estuary, chromium (VI) was not known to be used in Site operations. Further, chromium (III) is strongly favored in natural waters and sediments because the concentrations of sediment constituents known to reduce chromium (VI) to chromium (III) generally far outweigh the concentrations of the few constituents known to oxidize chromium (III) to chromium (VI). Once reduced, chromium (III) is very stable in aquatic environments and highly unlikely to oxidize to chromium (VI). (James and Bartlett, 1983; Fendorf 1995; Weaver and Hochella, 2003).

5.11 Lead

Lead was identified as a COPC because maximum detected concentrations in sediment exceeded default screening levels. Because of its unique toxicological properties, lead is evaluated differently from other COPCs in the risk assessment process. Lead can produce a number of significant noncancer adverse effects, including effects on the gastrointestinal system, hematopoietic system, cardiovascular system, central and peripheral nervous system, and kidneys. Unlike other noncarcinogens, however, no

RfD has been developed for lead. Instead, the metric used to evaluate the toxicological significance of lead exposure is the 10 μg/dL blood lead "level of concern" established by the U.S. Centers for Disease Control (CDC, 1991). The USEPA has developed biokinetic models to estimate the effect of site- or media-specific lead exposure to changes in a receptor's baseline blood lead level (BLL) which can then compared to the 10 μg/dL level of concern.

The USEPA has established a residential soil screening level for lead of 400 mg/kg (USEPA, 1994) that is based on the biokinetic modeling described above such that a hypothetical child would have no more than a 5% risk of exceeding a blood lead level of 10 μg/dL. Although lead was identified as a COPC in marsh sediment based on the conservative screening approached used in this risk assessment, its EPC of 43.7 mg/kg (based on the 95% UCL) was nearly 10-times below the residential screening value of 400 mg/kg used by USEPA for residential land use. On this basis, no additional risk evaluation of lead in soil was performed.

5.12 Manganese

The RfD for manganese is based on dietary requirements in humans and a single epidemiological study from Greece. The value is 1.4E-01 mg/kg-day. Manganese does not appear to be carcinogenic and is classified in group D. Additional information is available on the IRIS database.

5.13 Thallium

The toxicity of thallium compounds was recently reviewed by the USEPA and it concluded that insufficient toxicological information exists to develop reliable quantitative dose-response estimates. As a result of that review, all toxicity values related to thallium were withdrawn from USEPA's IRIS database. For this risk assessment, the withdrawn RfD for thallium ~~chloride~~(soluble salts) was used. Previously, IRIS toxicity assessments were available for a number of thallium compounds; Thallium ~~chloride~~(soluble salts) was chosen because the water in the marsh and estuary is salt water. The withdrawn RfD is 6.5E-5 and based on a NOAEL in from rat subchronic study in which critical effect was elevation of serum enzymes. In the principal study, dose-related increases in alopecia, lacrimation, and exophthalmos were also observed. Thallium does not appear to be carcinogenic and is classified in group D. Additional information is available on the IRIS website.

6.0 RISK CHARACTERIZATION

6.1 Overview

This section discusses the potential risk to human health associated with sediment contact and fish and game consumption. A summary of risk estimates is presented in Table ~~23~~22.

6.2 General Concepts

Potential risks to human health can be evaluated quantitatively by combining potential exposure and toxicity data. A distinction is made between non-carcinogenic and carcinogenic endpoints, and two general criteria are used to describe risk: the hazard quotient (HQ) for non-carcinogenic effects; and excess lifetime cancer risk (ELCR) for constituents thought to be potential human carcinogens.

Exposure doses are averaged only over the exposure duration period to evaluate non-carcinogenic effects. The HQ is the ratio of the estimated exposure dose and the RfD for oral, dermal and inhalation exposures. An HQ greater than 1 indicates that the estimated potential exposure for that constituent exceeds the RfD. This ratio does not provide the probability of an adverse effect, but does reflect the concept of a threshold for the adverse effects. While an HQ value of less than 1 indicates that health effects are highly unlikely to occur, an HQ value that exceeds 1 does not suggest that health effects will occur. RfDs have been developed as protective estimates of the human threshold for adverse effects and have a margin of safety included. The RfD is a very good tool for CERCLA-type risk assessments that are ultimately used to develop a cleanup level with a high expectation of protectiveness. The RfD is a poor tool for determining whether actual human effects will occur. The sum of the HQs is the hazard index (HI).

A limitation with the hazard index approach is that the assumption of dose additivity is applied to compounds that produce different effects by different mechanisms of action. Consequently, the summing of hazard indices for a number of compounds that are not expected to induce the same type of effects or that do not act by the same mechanism or on the same target organ may overestimate the potential for adverse effects (USEPA, 1989). Consistent with USEPA risk assessment guidelines for chemical mixtures, in the event that a total HI exceeds 1, HQs should be segregated HQs by target organ (USEPA, 1989). In this risk assessment, this is not an issue because the two risk drivers mercury and Aroclor 1268, produce effects on the same target organ – the brain and nervous system.

The ELCR is an estimate of the potential increased risk of cancer resulting from lifetime exposure to constituents detected in media at the facility. Estimated doses, or intakes, for each constituent are averaged over the hypothesized lifetime of 70 years. It is assumed that a large dose received over a short period is equivalent to a smaller dose received over a longer period, as long as the total doses are equivalent. The ELCR, equal to the product of the exposure dose and the CSF, is estimated for each appropriate COPC in each medium. The risk values provided in this report are an indication of the potential increased risk from contact with Site media. Similar to RfDs, the cancer slope factor is a tool to develop protective cleanup levels, but a poor predictor of the actual occurrence of cancer in humans. Because ELCRs are probabilities, they can be summed across routes of exposure and COPCs to derive a "Total Site Risk" (USEPA, 1989). ELCR estimates are evaluated in the context of the risk range of 1 in 1,000,000 (10^{-6}) to 1 in 10,000 (10^{-4}) identified in the National Contingency Plan (NCP) (40 CFR Part 300).

6.3 RME Results – Marsh Trespasser

RME risk estimates and hazard indices were determined for ~~child,~~ adolescent, adult, and "lifetime" consumers in the marsh trespasser scenario (Table 8a, Table 9a, Table ~~23~~22). The RME cancer risk for the lifetime receptor is ~~2E-06.~~1E-05. The risk estimate~~s~~ for the ~~child and~~ adolescent ~~were summed and~~ was added to ~~60~~67% of the adult estimate. This procedure provides a value for exposure duration of 30 years with ~~2 years as a child,~~ 10 years as an adolescent and ~~18~~20 years as an adult. This value is at the ~~lower end~~mid-point of the risk range identified in the NCP. The RME hazard indices for the adult~~,~~ and adolescent~~, and child~~ receptors are 0.~~01, 06~~ and 0.~~01, and 0.04~~08, respectively. These are ~~all~~both below the regulatory threshold of unity.

6.4 CTE Results – Marsh Trespasser

CTE risk estimates and hazard indices were determined for ~~child,~~ adolescent, adult, and lifetime receptors in the marsh trespasser scenario (Table 8b, Table 9b, Table ~~23~~22). The CTE cancer risk for the lifetime receptor is ~~1E~~2E-07. This value is nearly 10-fold lower than the lower end of the risk range identified in the NCP. The CTE hazard indices for the adult~~,~~ and adolescent~~, and child~~ receptors are 0.~~002, 0.003,~~005 and 0.~~01~~006, respectively. These are ~~all~~both below the regulatory threshold of unity.

6.5 RME Results - Consumers of Recreationally Caught Fish

RME risk estimates and hazard quotients were determined for child, adolescent, adult, and lifetime consumers of fish. The estimated RME cancer risk for the lifetime fish consumer was 1E-04. The risk estimates for the child and adolescent were summed

and added to one-half of the adult estimate. This procedure provides a value for exposure duration of 30 years with 6 years as a child, 9 years as an adolescent, and 15 years as an adult. The RME hazard indices for the adult, adolescent and child receptors in the recreational fish consumption scenario were 3, 3 and 4 respectively. These calculations and results are shown in Tables 12a, b and c, and summarized in Table ~~23~~22.

Following USEPA Region 4 risk assessment guidance (USEPA 2000), Aroclor 1268 and mercury are identified as constituents of concern (COCs). The guidance indicates that the total HI may be separated into target organ-specific HIs. However, in this case, both PCBs and mercury affect the brain and nervous system and thus should not be separated. Although mercury is a significant contributor to the total HI, it seems likely that mercury would be difficult to clean up in fish due to atmospheric deposition and mercury cycling. A further discussion of mercury related to fish clean up is presented in Section~~s~~ 8 ~~and 9~~.

6.6 CTE Results - Consumers of Recreationally Caught Fish

The estimated CTE cancer risk for the lifetime recreational fish consumer is ~~1E-~~2E-05 estimated in a similar fashion as described for the RME results. The lifetime risk was estimated as a sum ~~as described previously~~ of the risk estimates for the child, adolescent, and adult. The CTE HIs for the adult, adolescent, and child receptors in this scenario are 0.8, 0.9, and 1, respectively. These low CTE risk and hazard estimates support the conclusion that no chemicals would be likely to be selected as COCs. These calculations and results are shown in Tables 13a, b and c, and summarized in Table ~~23~~22.

6.7 RME Results – ~~Subsistence~~Hypothetical High Quantity Consumers of Fish

The estimated RME cancer risk for the lifetime ~~subsistence~~high quantity fish consumer is 2E-04. The lifetime risk was estimated as a sum as described ~~previously.~~in Section 6.5. The RME HIs for the adult, adolescent, and child receptors in this scenario are 5, 5, and 8, respectively. These calculations and results are shown in Tables 14a, b and c, and summarized in Table ~~23~~22.

6.8 CTE Results – ~~Subsistence~~Hypothetical High Quantity Consumers of Fish

The estimated CTE cancer risk for the lifetime ~~subsistence~~high quantity fish consumer is 4E-05. The lifetime risk was estimated as a sum as described ~~previously.~~in Section 6.6. The CTE HIs for the adult, adolescent, and child receptors in this scenario are 2, 3, and 2 respectively. These calculations and results are shown in Tables 15a, b and c, and summarized in Table ~~23~~22.

6.9 RME Results – Consumers of Shellfish

The estimated RME cancer risk for the lifetime consumer of shellfish is 6E-05. The lifetime risk was estimated as a sum as described ~~previously~~ in Section 6.5. The RME hazard indices for the adult, adolescent, and child receptors in this scenario are 2, 0.7, and 4, respectively. Table 16 shows the calculations and results. A summary is also provided in Table ~~23~~22.

6.10 CTE Results – Consumers of Shellfish

The estimated CTE cancer risk for the lifetime consumer of shellfish is ~~6E~~9E-06. The lifetime risk was estimated as a sum as described ~~previously~~ in Section 6.6. The CTE hazard indices for the adult, adolescent, and child receptors in this scenario are 0.6, 0.2, and 2, respectively. Table 17 shows the calculations and results. A summary is also provided in Table ~~23~~22.

6.11 RME Results – Consumers of Clapper Rail

The estimate of RME cancer risk for the lifetime consumer of clapper rail is 1E-04. The lifetime risk was estimated as a sum as described ~~previously~~ in Section 6.5. The RME hazard indices for the adult, adolescent, and child receptors are 2, 1, and 1, respectively. Table 19 shows the calculations and results. A summary is also provided in Table ~~23~~22.

6.12 CTE Results – Consumers of Clapper Rail

The estimate of CTE cancer risk for the lifetime consumer of clapper rail is ~~5E~~8E-06. The lifetime risk was estimated as a sum as described ~~previously~~ in Section 6.6. The CTE hazard indices for the adult, adolescent, and child receptors are 0.4, 0.1, and 0.4, respectively. These are all below the regulatory threshold of unity. Table 20 shows the calculations and results. A summary is also provided in Table ~~23~~22.

7.0 DEVELOPMENT OF REMEDIAL GOAL OPTIONS

Consistent with USEPA Region 4 guidance (EPA 2000), a range of Remedial Goal Options (RGOs) is presented for each constituent identified as a COC. Region 4 guidance states:

> *Chemicals of Concern (COCs) are the Chemicals of Potential Concern (COPCs) that significantly contribute to a pathway in a use scenario for a receptor (e.g. hypothetical future child resident, current youth trespasser, current adult construction worker, etc.) that either (a) exceeds a 10-4 cumulative site cancer risk; or (b) exceeds a non-carcinogenic hazard index (HI) of 1. Note: generally, a 10-4 cumulative site risk level and an HI of 1 are used as the remediation "trigger." The exact level used as the "trigger" is at the discretion of the risk manager. The carcinogen "trigger" represents the summed risks to a receptor considering all pathways, media, and routes per land use scenario. The HI represents the total of the hazard quotients (HQs) of all COPCs in all pathways, media, and routes to which the receptor is exposed. If the HI exceeds 1.0, then more specific HIs should be developed by summing HQs of COPCs with Reference Doses (RfDs) based on toxic effects on the same target organs. This specific target-organ based HI should form the basis of COC selection. Chemicals are not considered as significant contributors to risk and therefore are not included as COCs if their individual carcinogenic risk contribution is less than 10-6 and their non-carcinogenic HQ is less than 0.1.*

Examination of Table ~~23~~22 indicates that the scenarios for which cancer risk estimates would trigger development of RGOs are that of the recreational fish consumer, with a lifetime risk of 1E-04 and HIs exceeding 1, the ~~subsistence~~hypothetical high quantity fish consumer with a lifetime risk of 2E-04 and HIs exceeding 1, the shellfish consumer with a HIs exceeding 1 for the adult and child receptors, and the clapper rail consumer, with a lifetime risk of 1E-04 and an HI exceeding 1 for the adult receptor. All of these values are just slightly above the trigger level of 1E-04. RME hazard indices in fish and game consumption scenarios would generally trigger RGO development as most of these are greater than unity. Risk estimates and hazard indices for the marsh trespasser scenario would not trigger RGO development.

Table ~~24a~~23a presents the cancer and non-cancer based RGOs for recreational finfish consumption; Table ~~24v~~23b presents the cancer and non-cancer based RGOs for ~~subsistence finfish consumption~~the hypothetical high quantity fish consumer; Table ~~24c~~23c presents the non-cancer based RGOs for shellfish consumption; Table ~~24d~~23d presents the cancer and non-cancer based RGOs for clapper rail consumption.

8.0 UNCERTAINTY ASSESSMENT

8.1 Overview

The risk estimates presented here are conservative estimates of potential risks associated with potential exposure to constituents detected in media at the LCP Site. Uncertainty is inherent in the risk assessment process, and a discussion of these uncertainties is presented in this section. Each of the three basic building blocks for risk assessment (monitoring data, exposure scenarios, and toxicity values) and for the exposure assessment (factors, models, and scenarios) contributes to the overall uncertainty.

Samples collected during site investigations were intended to characterize the nature and extent of potential contamination at the Site. Subsequently, most of the samples were collected from locations selected in a directed manner to accomplish this goal. Sampling locations selected in this way provide considerable information about the Site, but often tend to be concentrated in areas of higher levels of contamination. Therefore, data from sampling locations selected in this manner tend to overestimate constituent concentrations representative of the potential exposure area. This may not be as large an issue in this risk assessment because of the abundance of data at the LCP Site (Figure 1). Hence, this risk assessment (like others) is based on the assumption that the available sampling data adequately describe human contact with chemicals in environmental media at the LCP Site.

~~8.2 Decreasing Concentrations in Fish~~

~~In 1997, fish samples were obtained near the LCP facility and analyzed for total PCBs (Kannan et al., 1998). Two points are to be made herein. First, the homologue concentration observed in fish at that time was very similar to the homologue composition of Aroclor 1268 and thus, the extent of weathering in terms of changing PCB composition (and hence toxicity) is very small. Second, when one compares the concentrations in Blue Crab, Spotted Seatrout and Striped Mullet reported in Kannan et al. (1998) to the more recent samples used here, it is clear there has been considerable reduction in PCB concentrations (Table 25). The dataset used in this risk assessment was examined to determine if trend analysis could yield an estimate of the rate at which concentrations are changing in these fish. Generally speaking, there does not appear to be a trend in concentration, neither increasing nor decreasing. However, there were not sufficient numbers of samples of each species over time to be able to perform a credible analysis. Suffice it to say, that if environmental weathering of Aroclor 1268 is occurring, then concentrations in all fish may decrease over time, and the risk estimates~~

for fish and shellfish consumption presented here would likely be lower if this risk assessment exercise were to be repeated in the future.

8.2 Mercury is unlikely to decrease in fish. A discussion of mercury related to trophic level weighting method used by the GAEPD was presented in Section 9. While Aroclor 1268 appears to be decreasing in fishHypothetical High Quantity Fish Consumption

This risk assessment included an evaluation of hypothetical high quantity consumers of fish because the ATSDR/GCHD seafood survey (DHHS, 1999) included two Glynn County residents who identified themselves as "subsistence" fishers. Data from the ATSDR/GCHD survey were used to develop fish intake estimates consumers of locally caught fish that might have higher rates of consumption than is reflected by the rates for the recreational consumer obtained from EPA's Exposure Factors Handbook. The derivation of the fish ingestion rates for this receptor is described in Appendix B. However, because the ATSDR/ GCHD study only included information about the survey respondants' recent seafood consumption (including both finfish and shellfish) from all sources (i.e., locally harvested and purchased), not only fish harvested from the Turtle River or its tributaries, these intake estimates are likely to significantly overestimate finfish consumption from the areas in close proximity to the LCP site. In addition, the ATSDR/GCHD study included a small number of respondants over a short period of time which adds to the uncertainty about the use of these data to estimate dietary intakes over the extended time periods evaluated in this risk assessment.

Although the ATSDR/GCHD study included individuals that identified themselves as subsistence fishers, it seems unlikely that mercury levels will decrease due to continued atmospheric deposition unrelated to the LCP Site.

8.3 Subsistence Fish Consumption

It seems very unlikely that any of the Brunswick population could be considered subsistence fish consumers. One way to determineevaluate this is to compare the fish consumption rates among the Brunswick anglers included in the ATSDR/GCHD study to the recommended daily allowance (RDA) of protein. The recommended daily allowance of protein for adults and children greater than 1 year old is 0.8 g/kg-day (NAS, 2005).

One can divide the subsistence RME fish consumption rates (FCR) by body weight to obtain the FCR in g/kg-day. The respective values are 0.22, 0.23 and 0.35 g/kg-day for the adult, adolescent and child subsistence fish consumers, all less than the RDA. In contrast, the mean intake of four Columbia River tribes is 59 g/day and the 95th percentile is 170 g/day (CRITFC, 1994). In a 70 kg adult, these would correspond to

FCR values of 0.84 g/kg-day and 2.4 g/kg-day respectively. Note that these values are both greater than the RDA. Wolfe and Walker (1987) observed fish consumption rates up to 770 g/day in a study of 94 Alaskan communities, corresponding to 11 g/kg-day. Therefore, it seems very unlikely that individuals in the Brunswick population could be considered true subsistence fish consumers.

Another possible way to ~~determine if~~ evaluate whether or not subsistence anglers are present is to examine monetary incomes of anglers based on the zip codes provided in the MRFSS data. The zip codes would presumably not be biased or inaccurate. For this exercise, subsistence anglers were assumed to be represented by those harvesting Spot or Striped Mullet, fish that can be easily caught from shore and would tend to be targeted by subsistence anglers (as opposed to Spotted Seatrout or Red Drum). There were very few consumers of Striped Mullet and Spot. Census data can provide the average income per zip code. The average income of the zip codes of anglers harvesting Spot and Striped Mullet were obtained from databases maintained by the Missouri Census Data Center (MCDC, 2006). The average yearly income of the zip codes of the coastal Georgia residents harvesting Spot from 2001 to 2005 was $35,240. The average yearly income of the zip codes of the coastal Georgia residents harvesting Striped Mullet from 2001 to 2005 was $37,847. The average yearly income of all the coastal Georgia zip codes was $38,193. These income values seem quite similar.

Discussions with personnel at the Georgia DNR Coastal Resources Division suggest that the intercept survey was able to pick up all income levels and would include subsistence anglers if present (Spud Woodward, Kathy Knowlton, Georgia DNR, personal communication). It is interesting to note that of the group of nine anglers who harvested Spot from 2001 through 2005, only one came from Brunswick whereas four came from Savannah. The average zip code income of this single Brunswick angler was $23,898. The average zip code income of the Savannah anglers ranged from $18,830 to $60,182. In addition, there may be income variability within a single zip code but income data for smaller areas are not available.

It is possible that some subsistence anglers lived in the Savannah zip code in which the average income was $18,830. However, none of these anglers were from the Brunswick area and there remains no evidence that there were subsistence anglers in the Brunswick area.

~~8.4~~8.4.3 Choosing a Toxicity Criterion for Aroclor 1268

The determination of whether Aroclor 1268 is more similar on a toxicological basis to Aroclor 1016 or Aroclor 1254 would determine the choice of a surrogate toxicity value.

As will be shown below, Aroclor 1268 is more similar on a toxicological basis to Aroclor 1016 than to Aroclor 1254. Hence, the RfD for Aroclor 1016 was used.

To examine the potential similarities between the three mixtures, the metabolism and persistence of the various congeners in humans, the composition of the three Aroclor mixtures and three MOAs for the toxicity of PCBs were considered.

Metabolism and Persistence of Individual Congeners

Data for metabolism and persistence were obtained from Park et al. (2007) who examined serum PCB concentrations in 87 Korean volunteers. Table ~~26~~24 shows the lipid-normalized concentrations of congeners detected in serum along with the distribution. These values were obtained from Table 1 in Park et al (2007). The most abundant congener in human serum is PCB153 that has an average concentration of 39.2 ng/g lipid and comprises 22.6% of the total serum PCB concentration. The last column in Table ~~26~~24 labeled "Relative Persistence" is the ratio between the serum concentration of each congener and that of PCB153 to obtain a value reflecting the bio-persistence of each congener in the body relative to PCB153. These values are analogous to the familiar "TEF" scheme for the dioxin-like properties of PCBs.

Bioaccumulation and metabolism of PCBs was first quantified in the 1990s based on examination of tissue concentrations in relatively lightly exposed capacitor workers versus heavily exposed Yusho and Yucheng patients (Brown et al., 1989; Lawton et al., 1985a,b). Comparison of relative bio-persistence from Brown (1994) ~~appear~~appears to predict quite well the observed relative serum concentrations in Park et al (2007).

Congener Composition of the Aroclor Mixtures

The congener composition of Aroclor 1016 was obtained as the average percentage from Anderson (1991) and Frame et al., (1996). The congener composition of Aroclor 1254 was obtained as the average percentage from Anderson (1991), Frame et al., (1996) and Kodavanti et al. (2001). The congener composition of Aroclor 1268 was obtained from Anderson (1991). These are shown in Table ~~27~~25.

Modes of Action (MOAs) of PCBs Related to Systemic Toxicity

The three modes of action considered are:

- A dioxin-like MOA characterized by binding to the aryl hydrocarbon receptor and quantified by dioxin TEQ (van den Berg et al., 2006).
- A MOA based on binding to the ryanodine receptor and consequent interference with cellular calcium homeostasis (Pessah et al., 2006; Simon et al., 2007).

- A MOA based on binding to trans-thyretin, a plasma thyroid binding protein, and subsequent increase metabolism of thyroxin (Chauhan et al., 2000).

Congener concentrations in Aroclor 1016, 1254 and 1268 were obtained from Anderson (1991) and Frame et al., (1996). For each MOA, the value of the relative potency of each congener was multiplied by the congener bioaccumulation equivalent and the congener concentration in Aroclor 1016, Aroclor 1254 and Aroclor 1268. For each mixture, the sum of these values represented the potential for the particular Aroclor mixture to produce toxicity specific to each MOA. Table 26 shows the amount of each bioaccumulated equivalent value in the mixture. As can be seen, Aroclor 1254 has more of each type of bioaccumulated equivalent and contains about 1 order of magnitude more of both bioaccumulated neurotoxic equivalents and bioaccumulated thyroid hormone equivalents than either of the other two mixtures.

Mixture Toxicity Estimates for the Three MOAs

For each MOA, the value of the relative potency of each congener was multiplied by the congener relative persistence of that congener and the congener concentration in Aroclor 1016, Aroclor 1254 and Aroclor 1268. These calculations are shown in Table ~~28~~27 for all congeners that persist in the body based on Park et al. (2007) and comprise greater than 0.5% of any of the three Aroclor mixtures. In addition, the dioxin-like congeners PCB77, PCB81, PCB105, PCB114, PCB118, PCB123, PCB126, PCB156, PCB157, PCB167, PCB169 and PCB189 were included even if they were not persistent or were at very low percent composition values in the Aroclor mixtures. For each mixture, the sum of these values represented the potential for the particular Aroclor mixture to produce toxicity specific to each MOA. ~~The~~ Table 26 and the bottom row of Table ~~28 and Table 22~~27 show the amount of each bio-persistent equivalent value in the mixture. As can be seen, Aroclor 1254 has more of each type of bio-persistent equivalent. Aroclor 1254 contains at least two ~~order~~orders of magnitude more bio-persistent dioxin TEQ than either Aroclor 1016 or Aroclor 1268. Aroclor 1254 contains about 1 order of magnitude more bio-persistent Ca^{2+} neurotoxic equivalents than Aroclor 1016 and about 2 orders of magnitude more than Aroclor 1268. Aroclor 1254 contains 2 orders of magnitude more bio-persistent thyroid hormone equivalents than Aroclor 1016 and 4 orders of magnitude more than Aroclor 1268.

The reference doses for Aroclor 1016 and Aroclor 1254 on IRIS are based on the critical endpoints of reduced birth weights in monkeys for Aroclor 1016 and ocular, dermal and immune effects for Aroclor 1254. It is likely that the critical effect for Aroclor 1016 is based on either the Ca^{2+} endpoint or the thyroid disrupting endpoint (Simon et al., 2007;

Pessah et al., 2006; Dziennis et al., 2008; Lein et al., 2007; Howard et al., 2003; Kodavanti, 2005). It is likely that the critical endpoint for Aroclor 1254 is the Ca^{2+} endpoint.

Aroclor 1254 is orders of magnitude more toxic than either Aroclor 1016 or Aroclor 1268. The 3- to 4-fold difference in the RfD values is due to inconsistent application of extrapolation factors. In any case, the conclusion of the analysis is that the reference dose for Aroclor 1016 is more likely to reflect the toxicity to humans than is the reference dose for Aroclor 1254 and, hence, the RfD of 7E-05 mg/kg-dday was used as a surrogate toxicity criterion for Aroclor 1268.

8.58.4 Comparison of Noncancer Effects of PCBs in Monkeys and Humans

The current USEPA oral RfD for Aroclor 1254 is 2E-5 mg/kg-day and follows standard USPA USEPA guidance and procedures for the development of an RfD, and is based upon studies in monkeys by Arnold et al. (1993a,b) and Tryphonas et al. (1989, 1991a,b). The USEPA has interpreted these studies as indicating a LOAEL of 5.0 µg/kg-day based on ocular, dermal and immunological effects as the critical endpoints. From this LOAEL, an RfD of 0.02 µg/kg-day is calculated using a total Uncertainty Factor (UF) of 300 which was based on adopting a factor of 10 for sensitive individuals, 3 for interspecies extrapolation, 3 for use of a LOAEL instead of a NOAEL, and 3 for the use of subchronic rather than chronic data. The current USEPA oral RfD for Aroclor 1016 is 7E-5 mg/kg-day and was based from on a different series of monkey studies evaluating perinatal and neurobehavioral effects (Barsotti and Miller, 1984; Levin et al., 1988; Schantz et al., 1989; Schantz et al., 1991) that identified a NOAEL of 7 µg/kg-day to which a total uncertainty factor of 100 was applied.

While the monkey clearly shares a great many anatomical and physiological similarities with humans, this does not necessarily mean that primates and humans share a similar responsiveness to a particular chemical. When available, empirical comparisons of potency may provide an important test of the validity of the animal model being used to extrapolate safe human exposure levels. Interestingly in this instance, the responsiveness of the experimental model used to derive the RfD and its ability to reflect accurately the dose-toxicity relationships in humans can be examined because some of these monkey studies also provided tissue concentration data that corresponded to the daily applied dose. In the study by Tryphonas et al. (1991a,b), the observed oculodermal effects were associated with 5, 20, 40, or 80 µg/kg-day doses of Aroclor 1254 in the diet. The corresponding PCB serum concentrations at steady-state, achieved after about 10 months of treatment, were 10.4, 32.1, 68.1, and 105.1 ppb, respectively. Thus, if one were to assume that humans are as sensitive as the test

species, then obvious oculo-dermal effects should be evident in humans with PCB blood levels above 10 ppb and immune dysfunction would appear at PCB blood levels of about 70-100 ppb.

In contrast to the projections one would reach from the available PCB monkey studies, a review of the PCB clinical studies in human populations environmentally and occupationally exposed to PCBs clearly ~~indicate~~indicates that humans are not as sensitive to PCB-induced effects as are primates. For example, during the 1970s and 1980s, over 90% of the general US population had detectable PCB blood levels and almost 30% had blood levels greater than 1000 ppb (ATSDR, 1997). With almost 30% percent of the U.S. having serum PCB levels 200 times greater than those that produced discolored and disfigured nails, and eye swelling and discharge in monkeys, people displaying these symptoms would be common and visible effects evident.

In addition, studies of occupationally-exposed capacitor manufacturing workers have failed to document the same oculo-dermal findings upon which the RfD is based - some of the clinical studies of occupationally exposed individuals were comprised of workers with average PCB concentrations of 400 ppb, with some individuals with serum PCB levels of 3,250 ppb (Baker et al., 1980; Emmett et al., 1988a,b; Lawton et al., 1985a,b; James et al., 1993; ATSDR, 1997).

There are no studies evaluating the potential immune effects of PCBs in humans in the same way as the Tryphonas monkey studies; these kinds of tests are not performed in humans. However, there is information regarding the functional immune status of PCB-exposed individuals. In one study, responsiveness to immune challenge with mumps and trichophyton antigens was compared between PCB-exposed workers and non-exposed controls (Emmett et al., 1988b). These antigen challenge tests are instructive because, like the SRBC test used in the monkey studies, interaction of the three principal cells of the immune system (macrophages, T-lymphocytes, and B-lymphocytes) is required. No significant effects on responsiveness were noted, despite the fact that the capacitor workers had PCB serum levels much greater than those in the monkeys in the Tryphonas studies. Similarly, morbidity analyses of occupationally exposed groups found no associations between PCB exposure and leukocyte or differential blood counts (Fischbein et al., 1979; Baker et al., 1980; Maroni et al., 1981; Chase et al., 1982; Smith et al., 1982; Stark et al., 1986; James et al., 1993). Likewise mortality studies of these same groups of workers failed to find any increase in mortality from infectious disease (James et al., 1993). Again, individuals in some these workplaces had blood PCB levels that averaged hundreds of ppb with some individuals

reaching levels greater than 1,000 ppb (Lawton et al., 1985a,b; James et al., 1993; ATSDR, 1997).

The general appropriateness of the monkey as a model for PCB toxicity in humans can also be evaluated through examination of other toxicological endpoints. For example, Arnold et al. (1993a,b) found significantly diminished serum cholesterol levels among rhesus monkeys receiving 40 or 80 µg/kg-day Aroclor 1254. At least five studies have examined serum cholesterol and other lipids in PCB-exposed workers and compared them with controls (Baker et al., 1980; Chase et al., 1982; Smith et al., 1982; Emmett, 1985; Emmett et al., 1988a,b). None found a significant increase or decrease in serum cholesterol among PCB-exposed workers.

Arnold et al. (1995) conducted breeding experiments with male and female monkeys treated with 0, 5, 20, 40, or 80 µg/kg-day Aroclor 1254. After 37 months of exposure, females were bred with an untreated male. During the study, two of the monkeys in the high dose group had to be euthanized because they developed a severe wasting syndrome associated with the PCB exposure. In this study PCB treatment appeared to result in increased adverse reproductive outcomes, including decreased numbers of live births, increased suspected resorptions, and perhaps increased risk of post-partum death. Evidence of these effects appeared at the lowest PCB dosage in this study, 5 µg/kg-day. As with the oculo-dermal effects, these kinds of severe reproductive sequelae would be difficult to miss in humans with comparable or greater serum PCB levels. However, among women exposed occupationally to PCBs the only effect that has been observed is a slight decrease in infant birth weight (Taylor et al., 1989). In studies of women with environmental PCB exposure, no consistent effect on infant birth weight has been observed (Longnecker et al., 1997). Also, studies of birth outcomes have found no increased risk of spontaneous abortion or stillbirth attributable to PCB exposure (Longnecker et al., 1997). These comparisons indicate that monkeys are more sensitive to the reproductive effects of PCBs than humans.

Last, the comparison showing monkeys are particularly sensitive to PCBs that is the most convincing is that of lethality. In the study by Barsotti et al. (1976), one of nine monkeys treated with either 100 or 200 µg/kg-day died from toxicity during the course of the study. In studies by Tryphonas and co-workers (Tryphonas et al., 1986; Tryphonas et al., 1991a,b) these researchers suggest that doses between 80 µg/kg-day and 200 µg/kg-day can induce lethality following chronic exposures that produce blood levels of about 285 ppb at the lower dosage rate. In contrast, studies of PCB-exposed workers find no evidence of increased mortality, even among groups of workers with average PCB concentrations of 400 ppb or more and with individuals having serum PCB levels

as high as 3,250 ppb (Lawton et al., 1985a,b; James et al., 1993). Likewise the "wasting syndrome" described for these monkeys that led either to lethality has never been observed in humans (James, 1993; ATSDR, 1997). Thus, it is clear that monkeys do develop a number of frank adverse effects and may even die at PCB levels that were without any identifiable clinical effect in chronically exposed worker populations.

~~8.6~~8.5 Uncertainty Related to Aroclor 1268 Toxicity

The hazard indices for contact with marsh sediment and fish and game consumption presented in Table ~~23~~22 are artificially elevated due to the use of the RfD for Aroclor 1016 as a surrogate for that of Aroclor 1268.

The toxicity values and other toxicological information used in this report are likewise associated with significant uncertainty. In addition, humans are different than laboratory animals. The effects shown by the animals in the high dose studies are often very different than effects reported by humans in parallel epidemiology studies (e.g., Kimbrough et al 1999; Kimbrough and Krouskas, 2003).

This is indeed the case for PCBs. The noncancer RfD for Aroclor 1268 used here is based on those for Aroclor 1016 presented on IRIS. The monkeys used in the studies that support the IRIS noncancer toxicity values for PCBs are exquisitely more sensitive than humans to the effects of PCBs. The monkeys in these studies developed a "wasting" syndrome at PCB body burden levels about 100 fold lower than seen in occupational studies of humans – and these higher levels in humans were without apparent effect.

Regarding PCBs and cancer, a study by Kimbrough et al. (1999) indicates that PCBs may not cause cancer to the extent previously thought. The researchers conducted a mortality study of workers with at least 90 days exposure to PCBs between 1946 and 1977. For the 7,075 workers studied, vital status was obtained for 98.7 percent of the workers. This makes this study the largest cohort of male and female workers exposed to PCBs studied. The authors concluded that there were no "significant elevations in the site-specific cancer mortality of production workers."

As far as the cancer effects of PCBs, the extent of the contribution of dioxin-like and non-dioxin like PCBs to the development of cancer in the rats in the study supporting the IRIS PCB cancer slope factor remains unclear. The National Academy of Sciences recently released a draft review of USEPA's Dioxin Reassessment (NAS, 2006b). The review was highly critical and changes in the dioxin toxicity criteria will affect the evaluation for PCBs.

Hence, there is both scientific and regulatory/administrative uncertainty associated with the cancer slope factor and the reference doses for PCBs. In all likelihood, the values ~~on~~in IRIS are over-protective.

~~8.7~~8.6 Uncertainty in Exposure Estimates Related to Fish and Game Consumption

It is likely that the greatest uncertainty on the exposure side of this risk assessment is related to the amount of clapper rail eaten. It is difficult to find current estimates of their population size, hunting statistics or hunting lore. ~~As noted, an~~ Specific data regarding the amount of clapper rail ingestion were not available. This was not surprising, however, since local sportsmen and the GA-DNR indicated that clapper rail are generally hunted for sport and not as an edible game bird. An informal internet search ~~did turn up a couple of~~ using Google® found two recipes~~.~~ for clapper rail breasts – one where the tiny morsels were wrapped in bacon and served on a bed of rice; mention was made of the darkness of the breast meat and its gamey taste The birds are up to 400 g in size. The exposure assumptions used for clapper rail were obtained from USEPA (1997a) and were related to game in general. In addition, the mean game consumption rate in g/kg-day was provided along with a standard error of the mean. One notes in Table 18 that the standard errors were larger than the mean. Statistically speaking, that suggests that the mean consumption rate has a finite probability of being negative. Practically speaking, there is a great deal of uncertainty associated with the RME exposure estimates of clapper rail consumption.

Extrapolation of fish consumption rates between different age groups also bears considerable uncertainty. The survey of fish and game consumption practices conducted in Brunswick targeted adults. Ages were not reported in the data nor were individual fish consumption rates. In addition, the data were reported in three groups: < 1 meal per week, about 1 meal per week, and > 1 meal per week. These data obtained in adults were then applied to children without any changes to reflect possibly different preferences for fish that children might have. For example, the mean clapper rail consumption rate for children obtained from USEPA's *Exposure Factors Handbook* is one quarter that of adults (USEPA, 1997a; Table 18). If a child in the subsistence consumption scenario consumed one quarter of the amount of fish that an adult consumed, this value would be about 7 g/day (Table 10). Use of this value would reduce the estimated HI in the child subsistence fish consumption scenario from 8 to 5.6. Given the small size of clapper rail, it does seem likely that consumption rates would be lower than consumption rates for fish. How much lower is not known.

9.0 RISK MANAGEMENT CONSIDERATIONS

As discussed above in the Uncertainty Analysis section below, Aroclor-1268 concentrations in fish are likely to decrease over time. Because of mercury cycling and the global atmospheric reservoir of mercury, it seems unlikely that mercury concentrations will fall.

It is very unlikely that mercury in fish at Brunswick constitute a health risk. The GA-DNR has adopted a trophic level weighting approach for determining mercury-related fish advisories. The methodology is detailed in GA-DNR (2001). When one uses the same finfish data from 2002 and 2005 as was used here, the calculated Trophic Level Residue Value (TLRV) for a water body is less than the comparison value of 0.3 mg/kg in edible fish tissue. The concentration term used in this analysis is the geometric mean. USEPA's *Water Quality Criterion for the Protection of Human Health: Methylmercury, EPA-823-R-01-001* provides the scientific and regulatory support for this value (USEPA, 2001). A previous calculation of the TLRVs for the four water bodies near the LCP Site was conducted in 2006 and showed that all total TLRV values were less than 0.3 and that mercury-based fish advisories are not needed (Simon, 2006).

The Georgia Environmental Protection Division of the DNR is the regulatory authority that establishes whether the various water bodies in the state support their designated uses. If a water body does not support or only partially supports a designated use, the water body is considered impacted. Between 2004 and 2006, the GAEPD reclassified Gibson and Purvis Creeks near the LCP Site from the "not support" list based on levels of mercury in fish. In the GAEPD Summary of Changes, both creeks were "removed FCG(Hg) from not support list based on new fish tissue data that showed the trophic level weighted residue (TWR) is less than or equal to 0.3 mg/kg" (GAEPD, 2006). Hence, the GAEPD does not consider the current levels of mercury in fish near the LCP Site to be of concern.

Calculations performed with the child recreational fish consumer indicated that reduction of Aroclor-1268 concentrations in Red Drum, Southern Kingfish and Spotted Seatrout (accounting for 80% of consumption) to 1% of the current levels would lower the HI of the child RME recreational fish consumer from 4.3 down to 2.3.

Because GAEPD considers mercury in fish not to be a problem at the LCP Site and in the coastal waters around Brunswick (GAEPD, 2006), the main risk driver becomes Aroclor-1268. If the toxicity criteria for Aroclor-1268 from the peer reviewed scientific literature were used in lieu of the surrogate values for total PCBs and Aroclor-1016, the

risk estimates and hazard indices at the Site would be below levels of concern. Hence, the risks from Site exposure may be overly conservative due to the use of the IRIS toxicity values. Even the RfD value presented in the peer reviewed literature overestimates risk because it does not take into account the differences between humans and monkeys (Simon et al., 2007) (see Section 8).

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APPENDIX A

ProUCL OUTPUT

APPENDIX B

DEVELOPMENT OF RME AND CTE VALUES FOR ~~SUBSISTENCE~~HYPOTHETICAL HIGH QUANTITY FISH CONSUMERS

~~The Glynn County Health Department working with ATSDR developed values for~~ In 1999 the Agency for Toxic Substaces and Disease Registry (ATSDR) and the Glenn County Health Department (GCHD) conducted a survey that collected information on seafood consumption by Glenn County residents (DHHS, 1999). Because the ATSDR/GCHD seafood survey (DHHS, 1999) included two Glynn County residents who identified themselves as "subsistence" fishers, this risk assessment included an evaluation of hypothetical high quantity consumers of fish. Fish ingestion rates for this receptor scenario were derived using a Monte Carlo simulation based on data from several different sources, including locally relevant information from the ATSDR/GCHD study. This Appendix describes the derivation of these values.

The ATSDR/GCHD study produced information on the frequency of consumption of local fish and game from a target group of 211 individuals. The target group in Brunswick was limited to individuals who ~~consumed or caught fish from the Turtle River~~ lived in Glynn County~~, Georgia~~ for at least two consecutive years, had consumed or caught fish from the Turtle River or its tributaries in Glynn County, and had not been employed in an industry associated with occupational mercury exposure (DHHS, 1999). The frequency of consuming fish or game was assessed using both an interviewer-administered questionnaire and a dietary diary. 36% of the target population reported consuming seafood ~~or wild game~~(both locally caught and purchased) less than once per week~~.~~, 38% reported consumption about once per week~~.~~, 18% reported consumption more than once per week, and 8% ~~were missing data. These proportions were used to weight the choice of meal frequency distributions. The 8% missing data was assumed to be equally distributed among the other rate classes to yield 38% less than once per week, 41% about once per week and 21% more than once per week. The Brunswick data were obtained in 1998, about ten years ago. Poisson distributions were used to obtain a random selection of meal frequencies from the Brunswick data with rates~~ did not provide consumption frequency information.

For the Monte Carlo simulation, RiskAmp software[7] was used to generate a random selection of meal frequencies from the ATSDR/GCHD data based on Poisson distributions with lambda (i.e., expected) values of 2 meals/month, 4 meals per month and 7 meals per month (corresponding to the three ~~classes. Lognormal distributions~~

[7] RiskAmp is a commercially available Monte Carlo "add in" program for Microsoft Excel.

were fit to age- and gender-specific fish meal size values from USDA's Continuing Survey of Food Intake by Individuals (CSFII). Meal frequency andgroupings listed above). The proportions of survey respondants associated with each of these groupings (i.e., 38%, 41%, and 21%)[8] were used to weight the selection of meal frequency distributions.

Because the ATSDR/GCHD study only provided information on the frequency of seafood consumption by the local population, additional information on the portion size of fish consumed by individuals was also needed. The arithmetic mean and standard deviation of fish meal sizes, in units of grams, for children, adolescents, and adults were obtained from the U.S. Department of Agriculture's Continuing Survey of Food Intake by Individuals (CSFII) 1994-1996, 1998 (USDA, 2000). Using RiskAmp, lognormal distributions were fit to the age-specific fish meal size values obtained from the CSFII.

Using RiskAmp, values from the meal frequency distributions and values from the meal size distributions were multiplied to obtain a monthly fish consumption rate. 12 monthlyingestion rate distribution. These values were multipliedivided by 30.46 (the average number of days in a month) to yield distributions of daily fish ingestion rates, in units of grams/day, for children, adolescents, and adults. The 50th and 90th percentiles of these distributions were then adjusted by weighting factors for seasonal fish availability obtained from the Marine Recreational Fisheries Statistics Survey (MRFSS.

) data described in Section 4.5. The final daily fish ingestion rate for a given age group was assumed to be the average of the fish ingestion rates in these MRFSS intervals. For adults, adolescents and children, the RME and CTE fish consumption ingestion rate values were assumed to be the 90th and 50th and 90th percentiles, respectively, of the resulting distributions. These values are presented in Table B-1. This table also provides the input distributions and weighting factors required for the Monte Carlo simulation.

[8] The missing fish consumption rate information for 8% of the survey responders was assumed to be equally distributed among the other rate classes.

Table ~~AB~~-1 ~~gives the details of this calculation.~~. Derivation of Ingestion Rates for High Quantity Fish Consumers

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Meal Sizes (grams)[1]		
Age	**Arithmetic Mean**	**Standard Deviation**
0-6 years (Child)	54.5 g	42.7 g
7-16 years (Adolescent)	94.9 g	78.8 g
17-30 years (Adult)	134.6 g	111.9 g

[1] Data obtained from the USDA's Continuing Survey of Food Intake by Individuals 1994-1995, 1998 (USDA, 2000).

Meal Frequency[2]			
Survey Response	**<1/week**	**~ 1/week**	**>1/week**
Poisson Parameter[3]	2	4	7
Weighting Factor	38%	41%	21%

[2] Data obtained from ATSDR/ GCHD seafood survey (DHHS, 1999).

[3] Value corresponds to the approximate number of meals per month based on ATSDR/ GCHD survey responses.

Fish Availability Weighting Factor (unitless)[4]	
January – February	0.1
March – April – May	0.52
June – July – August	1
September – October	0.76
November – December	0.6

[4] Data for 2001-2005 harvest for Georgia obtained from the Marine Recreational Fisheries Statistics Survey online database (NMFS, 2007).

High Quantity Fish Ingestion Rates (grams/day)		
Age	**RME (90th %tile)**	**CTE (50th %tile)**
0-6 years (Child)	10	3
7-16 years (Adolescent)	18	11
17-30 years (Adult)	27	13

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