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FINAL REMEDIAL INVESTIGATION & RISK ASSESSMENT **REPORT CENTRAL LANDFILL OPERABLE UNIT 2 JOHNSTON, RHODE ISLAND VOLUME V OF V**

PREPARED FOR:

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APPENDIX F

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ECOTOXICOLOGICAL EFFECTS ASSESSMENT

APPENDIX F

TABLE OF CONTENTS

Page

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APPENDIX F ECOTOXICOLOGICAL EFFECTS ASSESSMENT

1.00 INTRODUCTION

As part of the Stage II ERC, detailed toxicological profiles were prepared which review known toxicological effects of the COCs. Using the toxicological profiles as a basis, the toxicological effects assessment puts the primary COCs within the context of the OU2 Study Area in terms of exposure potential and potential effects on receptors expected to utilize the site. GZA reviewed available literature regarding toxicological effects of the COCs (VOCs, P AHs, PCBs and pesticides, metals, and ammonia) on aquatic or terrestrial species (or similar organisms) that inhabit the OU2 exposure areas.

2.00 VOLATILE ORGANIC COMPOUNDS

Limited data are available regarding the toxicity of VOCs on ecological receptors. In general, VOCs must be present at high concentrations in order to cause adverse effects in animals. The following is a brief synopsis of toxicity studies regarding several of the VOC Contaminants of Potential Ecological Concern (COPECs) in sediment or surface water of the CLF Drainage areas.

In a gavage study involving rats and mice, high concentrations of chlorobenze reduced the survival rate for male rats (IRIS, 1999). Chlorobenzene (>99% pure) was administered by gavage in com oil to groups of rats and mice (50/sex/dose). Male and female rats and female mice groups received doses of 60 or 120 mg/kg, and male mice groups received doses of 30 or 60 mg/kg. Untreated groups of rats and mice served as controls. Chlorobenzene was administered five times per week for 103 weeks. Only the high dose male rats had statistically significant mortality (52% survival rate) compared to control groups. However, no chlorobenzene-related signs of clinical toxicity were observed in the rats (IRIS, 1999).

In a study addressing the carcinogenicity of 1,4-dichlorobenzene in rodents, female rats and male and female mice were gavaged with 300 and 600 mg/kg/day and male rats were gavaged with 150 and 300 mg/kg/day (ORNL, 1999a). Untreated rats and mice were used as controls. Higher percentages of mononuclear cell leukemia in male rats, of hepatocellular carcinomas in male mice, and of hepatocellular adenomas in female mice were found in high dose groups compared to control groups.

There is no evidence that acetone is carcinogenic to animals. However, adverse reproductive effects may occur at high concentrations. Doses greater than $3 \frac{\varrho}{\kappa}$ day during pregnancy were associated with spermatogenetic effects, reduced reproductive index, and decreased pup survival of rodents (ORNL, 1999b).

The low levels of VOCs in sediment and surface water from the OU2 Study Area are not expected to be carcinogenic or cause adverse effects in aquatic or terrestrial ecological receptors.

3.00 SEMIVOLATILE ORGANIC CONTAMINANTS

The following discussion focuses on several semivolatile organic contaminants that were considered to be of potential ecological concern in the OU2 Study Area.

Butylbenzylphthalate

Butylbenzylphthalate was considered a potential contaminant of ecological concern in surface water from Sedimentation Pond 4, Upper Simmons Reservoir, and Almy Watershed. In a review article by Staples et al., 1997 on the toxicity of 18 phthalate esters to aquatic organisms, lower molecular weight esters including BBP were found to be acutely or chronically toxic to aquatic algae, invertebrates, and fish. Based on the review of various studies regarding BBP aquatic toxicity, acute toxicity in aquatic organisms was found to result from BBP concentrations of 0.21 to 5.3 mg/L, and chronic toxicity resulted from concentrations from 0.075 mg/1 to 3.5 mg/1. Compared to the ranges reported in Staples et al., 1997, maximum butylbenzylphthalate concentrations of 0.004, 0.0048, and 0.01 from Upper Simmons Reservoir, Almy Watershed, and Sedimentation Pond 4, respectively are not expected to cause chronic or acute toxic effects in aquatic receptors.

Phenol

Phenol was considered a contaminant of potential ecological concern in sediments and surface water of Sedimentation Ponds 3 & 4 and Stream Channels; however, the concentrations present in surface water and sediment from these exposure areas are not expected to cause toxic effects in aquatic receptors. Additionally, phenol is not suspected to be a carcinogen in animals. In a bioassay addressing the carcinogenicity of phenol (IRIS, 1999), mice and rats were administered analytical grade phenol (approximately 98.5% pure) in the drinking water at concentrations of 2500 or 5000 ppm for 103 weeks. Dose-related decreases in weight gain in treated mice were attributed to decreased water consumption. No other clinical signs of toxicity were observed, and mortality rates (approximately 14%) were comparable between experimental and control groups.

Benzo(a)anthracene, Benzo[b]pyrene, and Bezo[b]fluoranthene

Benzo(a)anthracene, benzo[b]pyrene, and bezo[b]fluoranthene were considered to be contaminants 6f potential ecological concern in sediments from Sedimentation Ponds 2 & 3 the Stream Channels, and the Upper Simmons Reservoir. These contaminants are polycyclic (or polynuclear) aromatic hydrocarbons (PAHs) which constitute a class of several thousand organic compounds composed of two or more fused aromatic rings. Although limited data are available on the toxicity of PAH compounds, benzo(a)anthracene, benzo[b]pyrene, and bezo[b]fluoranthene are considered possible or probable carcinogens (Menzie *et al.,* 1992).

Eisler (1987) describes toxicological generalizations regarding PAHs and aquatic organisms. Generally, the toxicity of PAHs tends to increase with increasing molecular weight. Some species of aquatic organisms rapidly bioconcentrate PAHs from low concentrations in the ambient medium. Uptake of PAHs is species specific, and is higher in algae, molluscs, and other species that are incapable of metabolizing PAHs. BCFs tend to increase with increasing molecular weight, increasing k_{ow} values, with time approaching an equilibrium level, with increases in dissolved organic matter in the medium, and with increases in the lipid content of the organism. Typical BCFs for PAHs in aquatic organisms are in the range of 10 to 100,000.

There are sufficient data to conclude that benzo $[a]$ anthracene, benzo $[a]$ pyrene, and benzo[6]fluoranthene, are carcinogenic to animals (IRIS, 1995; IRIS, 1996). Eisler (1987) presents the following chronic reference doses for carcinogenicity in rodents (from Lo and Sandi, 1978; Overcash, 1983): benzo[a]pyrene, 0.002 mg/kg body weight; benzo[a]anthracene, 2.0 mg/kg body weight, and benzo[b]fluoranthene, 40.0 mg/kg body weight.

A relevant report of PAH toxicity to birds was presented in Eisler (1987). In this study (Patton *et al.,* 1980), mallards were fed diets containing 4,000 mg PAH/kg (mostly as naphthalenes, naphthenes, and phenanthrene) for 7 months. Although no overt signs of toxicity were observed, liver weight and blood flow increased.

4.00 POLYCHLORINATED BIPHENYLS (PCBS)

All of the PCBs detected in surface water and sediments from the exposure areas were considered to be contaminants of potential ecological concern due to their potential to accumulate in the aquatic food web. The following is a general discussion of the biological and chemical processes that influence the bioconcentration and bioaccumulation of PCBs in the aquatic food web.

PCBs are highly lipophilic (lipid attracted) compounds, with a very low solubility in water and high solubility in nonpolar organic solvents [octanol/water partition coefficient ($log K_{ow}$) $= 6.04$ (EPA, 1986)]. The environmental implications of these properties are that PCBs in

aquatic systems tend to quickly and firmly sorb to both dissolved and particulate organic matter. Regardless of the phase of the organic matter to which PCBs have sorbed, it is rendered biologically unavailable. This tendency to sorb to organic matter results in the settlement of most of the PCBs in a bioneutral state within the sediments. The compound is released into the interstitial pore water of the sediment at a rate directly proportional to the product of its K_{ow} and the fraction of organic carbon (F_{oc}) in the sediment until kinetic equilibrium is achieved. The fraction of PCBs in this interstitial pore water is bioavailable and potentially harmful to benthic organisms which are in contact with the sediment. As organisms pass waters contaminated with PCBs through their respiratory and digestive systems, the PCB mixture [especially the penta and hexachlorobiphenyl fractions] (Verscheuren, 1983)] is absorbed by lipid molecules and tissues. At this first level of biological incorporation of PCBs, tissue concentrations of the compound can be hundreds or hundreds of thousands of times greater than the concentrations in the ambient water (Eisler, 1986; Verscheuren, 1983). This increase in chemical concentration relative to the environmental media is referred to as bioconcentration.

Higher trophic level predators, feeding on prey that have bioconcentrated the compound, may be exposed to potentially harmful levels of PCBs. These predators may concentrate PCBs from contaminated water, as well as from their contaminated prey, resulting in *a* yet higher concentration. The combined uptake of a contaminant directly from a contaminated medium and via food ingestion is referred to as bioaccumulation.

The toxicity of PCBs varies greatly between isomers, generally increasing with increasing chlorination (Eisler, 1988). Toxicity also varies significantly between organisms. PCBs can increase the toxicity of other environmental contaminants (Bills *et al.,* 1977; Rhodes *et al.,* 1985). The AWQC for PCBs is 0.014 ppb and incorporates assumptions regarding the bioconcentration potential of these compounds.

The acute LC50 values for PCBs range from 10 ppb for the scud *Gammarusfasciatus* to 400 ppb for the damselfly *Ischnura verticalis* (EPA, 1980). The chronic lethal toxicity of Aroclor 1254 varies from 2.1 ppb in *Daphnia magna* to 0.8 ppb in the midge *Tantytarsus dissimilis* (EPA, 1980).

Due to the paucity or lack of toxicological data for some PCB mixtures, it is assumed that effects resulting from exposure to a specific Aroclor are representative of effects that may be produced by the other Aroclors (ATSDR, 1989). Although mink are not expected to be present in the OU2 exposure areas, the toxicity of PCBs to mink *(Mustela visori)* has received extensive scientific attention, perhaps due to the mink's perceived sensitivity to PCBs and its widespread distribution. In a review of PCB hazards to fish and wildlife, Eisler (1986) reported that minks which received Aroclor 1254 in dietary supplements of 2 mg/kg for eight months or 5 mg/kg for four months suffered a high death rate of their kits, while dietary levels of 1 mg/kg did not effect reproduction. Diets containing 50 ppm (2.5 mg/kg body weight/day) Aroclor 1254 caused adverse developmental effects in rats (Collins *et al.,* 1980).

Dietary concentrations of 20 ppm caused reduced litter sizes in first and second generation rat pups (ATSDR, 1989). The US Fish and Wildlife Service proposed a safe dietary intake of PCBs for mammals (based on mink) of less than 0.640 mg/kg fresh weight-diet (Eisler, 1986).

PCBs can disrupt normal patterns of growth, reproduction, metabolism, and behavior (Eisler, 1986). Dietary concentrations of 10 mg/kg Aroclor 1254 reduced the reproductive success of Ringed turtle-doves and Mourning doves *(Zanaida macroura carolinensis),* while 5 mg/kg impaired the reproductive success of chickens (Eisler, 1986). The US Fish and Wildlife Service proposed a safe dietary intake of PCBs for birds of less than 3.0 mg/kg fresh weightdiet (Eisler, 1986).

5.00 PESTICIDES

There is extensive research regarding pesticide toxicity to receptors in aquatic ecosystems. The following discussion focuses on DDT, chlordane, and endosulfan toxicity to aquatic receptors from Power et al, 1989.

DDT

DDT persists in the environment for a long period of time, and is able to accumulate within the aquatic food web. DDT enters organisms at different life stages through several routes including prey species and water intake both orally and via absorption through the skin. A number a factors can affect the accumulation of DDT in aquatic receptors including the stage of development, length of exposure, and previous exposure.

The mechanisms of toxicity of DDT are mainly due the physiological responses; however, behavioral abnormality can also be a significant factor (Power et al, 1989). Exposure of *R. temporaria* tadpoles to DDT resulted in abnormalities in glandular development in the external skin of the snout, and hyperactive behavior. The combination of the toxic effects caused the loss of the upper mandible, which resulted in a blunt snout and brain deformity.

In an acute toxicity study of DDT to amphibians, adult frogs were injected with 150 mg/kg of DDT, which resulted in 100% mortality. Mortality was also observed after injections of 10 mg/kg. However, in field application of DDT to ponds, no amphibian death resulted from exposure to 0.11 kg/ha, but 80% mortality resulted from 1.0 kg/ha. Acute exposure can lead to build up of residue in the blood and subsequent build up in the nervous system. An acute dose of DDT to tadpoles resulted in hyperactivity in response to a tissue build up of 2 ppm. Long-term effects of DDT exposure are mainly behavioral irregularities. Tadpoles and small frogs exposed to 0.1 ppb showed hyperactive behavior 5 to 8 days after exposure, and tissues of these receptors contained up to 2 to 5 ppm

Chlordane

In long-term toxic effect studies of chlordane, exposure to 0.5 ppm resulted in 40% death of frogs after 30 days. Effects such as neuromuscular changes, excessive thrashing, and tremors were observed. However, a low dose, 0.11 kg/ha, applied in the field, did not result in mortality.

Endosulfan

In a static bioassay with endosulfan, frogs were more sensitive to endosulfan exposure compared to damselfly nymphs and juvenile catfish. LC50 values were reported to be 2.1 ppb at 24 hr, 2.0 ppb at 48 hr, and 1.8 ppb at 96 hr. In field application of 0.014 kg/ha of endosulfan, no mortality resulted in adult frogs, however, fish kills in shallow water were observed.

6.00 METALS

6.10 METALS IN SURFACE WATER

The measured concentrations of total metals in sediments or surface water do not directly reflect the toxicity or the bioavailability of the metals. Site-specific environmental factors strongly influence the toxicity and bioavailability of metals. Such factors include ionic strength, pH, reduction-oxidation potential (Eh), water hardness, sediment particle size, total organic carbon, dissolved organic carbon, and suspended particulate matter. Toxicity and bioavailability are also influenced by the species of metal present and the synergistic or antagonistic effects that may be associated with exposure to multiple contaminants. For instance, a mixture of arsenic, cadmium, chromium, copper, mercury, lead, nickel, and zinc, when combined at individual concentrations deemed protective (Dutch water quality criteria) was severely toxic to *Daphnia magna* and caused 50% mortality in Rainbow trout *(Salmo gairdneri)* (Enserink *et al.,* 1991). Even a reduction to one/fifth of the water quality criteria caused a 10% decrease in *D. magna* populations (Enserink *et al.,* 1991). The following paragraphs briefly discuss some of the influences of environmental variables on the toxicity and bioavailability of metals to aquatic/semi-aquatic organisms, avian and mammalian receptors, and plants.

In natural surface waters, 30-80 percent of the copper, nickel, and zinc, and 90-95 percent of the lead may be in a particulate phase, greatly reducing toxicity and bioavailability (EPA, 1992). Both particulate and dissolved phases are detected in measurements of total metal concentrations. Because most AWQC and other laboratory toxicity tests are conducted using

metal salts which quickly dissolve in water, comparison of total metal concentrations to benchmarks developed using dissolved metals is inherently conservative and may result in overestimating the toxicity (EPA, 1992).

The speciation and solubility of metals in natural surface waters is dependent upon pH and ionic activity. Dissolved metals complex with dissolved inorganic ligands such as SO_4^2 and F " and organic ligands including humic and fulvic acids. This complexation and change in metal speciation is largely controlled by pH and the presence of organic ligands, and greatly reduces their toxicity (Freda, 1991).

The reduction of dissolved phase metals in water and sediments by complexation and immobilization directly influence toxicity to aquatic and semi-aquatic organisms by reducing the exposure point concentration. The potential for toxic effects on higher trophic level organisms may also be indirectly reduced because less dissolved phase metal is incorporated into the tissues of food organisms. The influences of complexation and immobilization on metal toxicity to plants is less certain, because many plants directly alter the Eh and pH of the rhizosphere (Crowder, 1991). Additionally, some plants release carriers or solubilizing agents from their roots, some of which accelerate metal uptake (Crowder, 1991).

6.20 METALS IN SEDIMENT

Most metals retained as COPEC were retained because they were present in sediment above screening level benchmark. The processes that alter a metals bioavailability and toxicity for dissolved metals in surface water are discussed above. These processes also occur often to a much greater degree, in sediments. Therefore, bulk metals concentrations in sediment are not well correlated with toxicity.

Metals in the aquatic environment partition between media such as soil and water, water and biota, or sediment and biota (Menzie *et al,* 1991). Partitioning of metals in sediments is affected by sediment Eh, pH, sulfide concentrations, organic content, and metal solubility products (Menzie *et al,* 1991). Because metal toxicity occurs predominantly in the dissolved phase, complexation and immobilization of metals in sediments can significantly reduce their toxicity by limiting the amount of dissolved metal in the sediment pore water to which organisms are exposed

Arsenic

Arsenic was selected as a COPEC in sediment from Sedimentation Ponds 2 & 3, Upper Simmons Reservoir, Lower Simmons Reservoir, and Almy Reservoir.

The toxicity of arsenic depends on the valence or oxidation state of the arsenic (-3, +3, or +5), as well as on the physical and chemical properties of the compound in which it occurs. Trivalent (As+3) compounds such as arsenic trioxide (As2O3), arsenic trisulfide (As2S3), and sodium arsenite (NaAsO2), are generally more toxic than pentavalent (As+5) compounds such as arsenic pentoxide (As2O5), sodium arsenate (Na2HAsO4), and calcium arsenate (Ca3(AsO4)2). The relative toxicity of the trivalent and pentavalent forms may also be affected by factors such as the water solubility of the compound. The more water soluble arsenic compounds are generally more toxic and more likely to have systemic effects in ecological receptors (http://risk.lsd.ornl.gov/tox/profiles/arsenic.htm#t2).

Eisler (1988) summarized chronic toxicological effects of arsenic on aquatic invertebrates, including benthic species. For *Gammarus pseudolimnaeus,* this report cited a 28-day LC20 for trivalent arsenic of 0.088 mg/1, and a 28-day LC-100 of 0.96 mg/1. Eisler (1988) cited a G. pseudolimnaeus LC-20 for As⁺⁵ of 0.97 mg/l. For the snail, Helisoma campanulata, Eisler (1988) reported an As⁺³ 28-day LC-10 of 0.96 mg/l, and for As⁺⁵ a 28-day LC-0 of 0.97 mg/l. These data suggests that arsenic in sediment has low bioavailability and low toxicity, and that adverse effects to sediment invertebrates would not be expected.

Cadmium

Cadmium was considered as a COPEC in sediment from the Upper Simmons Reservoir, Lower Simmons Reservoir, Almy Reservoir, and Almy Watershed.

The toxicity of cadmium to organisms that are exposed to contaminated surface water and sediments was evaluated by reviewing toxicological literature from 1967 to 1996. The results of this review are discussed below.

The chronic U.S. EPA AWQC for cadmium is 0.38 ppb, based on a water hardness of 25 mg/l as $CaCO_3$ (EPA, 1985). Free cadmium ions (Cd^{2+}) are believed to be the bioavailable species in the dissolved phase and accumulate in microorganisms, plant and animal tissue (Wren *et al.,* 1991). Cadmium is preferentially associated with the colloidal and particulate size fractions (Wren *et al.,* 1991).

Freshwater invertebrate BCFs for cadmium in water, measured over 52 weeks, ranged from 164 *(Pytiscidae* sp.) to 2200 *(Chironomidae)* (Eisler, 1985). BCFs for fish ranged from 33 *(Salmo gairdneri* over 10 weeks) to 7440 *(Gambusia affinis* over 26 weeks) (Eisler, 1985). The freshwater algae, *Chlorella vulgaris,* had a BCF of 2550 over 1.4 weeks of exposure (Eisler, 1985).

The toxicity of cadmium to aquatic invertebrates has received extensive scientific study (for example, see review by Sheedy *et* a/., 1991). Toxic responses to chronic cadmium exposure occurred at levels as low as 0.2 ppb in *Daphnia pulex* (Wren *et al.,* 1991). Some common benthic invertebrate families exhibit marked tolerance to cadmium relative to other taxa, especially stoneflies *(Plecopterd),* caddisflies *(Trichoptera),* mayflies *(Ephemeroptera),* and crayfish (Wren *et al.,* 1991).

Little is known about the toxicity of cadmium to amphibian species. Cadmium was toxic to the larvae of the frog, *Rana temporaria,* at a concentration off 4 ppb (Freda, 1991).

The toxicity of cadmium to fish is also well documented in Sheedy *et al.* (1991). Bluegill sunfish *(Lepomis macrochirus),* a species which may inhabit Cedar Swamp Brook, exposed to 1 ppb cadmium for 48 hours showed enzymatic impairment (Sheedy *et al.,* 1991), while 80 ppb caused physical malformations (Eisler, 1985).

Mammals are relatively resistant to cadmium (Eisler, 1985). A LOAEL dose for systemic effects of 1.2 mg/kg/day was identified using rats exposed to cadmium via drinking water (ATSDR, 1989). A NOAEL for developmental effects in rats exposed by gavage was reported at 0.04 mg/kg body weight/day (ATSDR, 1989). Most acute oral LD50 values for cadmium chloride and cadmium oxide range from 50 to 300 mg/kg (ATSDR, 1989). The U.S. Fish and Wildlife Service recommends that dietary concentrations above 0.100 mg/kg be viewed with caution (Eisler, 1985).

Sublethal effects of cadmium exposure in birds include growth retardation, anemia, and testicular damage (Eisler, 1985). Dietary levels of cadmium of 200 mg/kg (dry weight) over 90 days caused a decline in egg production in mallards (White *et al.,* 1978). Dietary levels of cadmium of 48 mg/kg (dry weight) caused a decline in egg production in chickens (Leach *et al.,* 1979). Drinking water concentrations of 0.600 ppm caused cardiovascular disease in pigeons (Eisler, 1985). The U.S. Fish and Wildlife Service (Eisler, 1985) suggests that wildlife dietary levels exceeding 100 ug cadmium/kg diet (fresh weight) on a sustained basis should be viewed with caution.

In general, submergent and floating-leaved plant species accumulate higher levels of cadmium than emergent species, and concentrations are usually higher in roots than in shoots (Crowder, 1991). The bioaccumulation factor for millfoil *(Myriophyllum)* was as high as 10,000 (Hutchinson, 1979). Symptoms of cadmium toxicity to wetland plants include reduced growth, with chlorosis and necrosis (Hutchinson, 1979). Toxic thresholds for cadmium are extremely variable between species. For instance, *Iris pseudoacorus* was not harmed by exposure to 5.0 mg/1 cadmium (Barboliani *et al.,* 1986), while 2.0 ug/1 cadmium reduced the growth rate of the freshwater algae *Asterionellaformosa* (Eisler, 1985).

Copper

Copper was selected as a contaminant of potential ecological concern in sediments from Sedimentation Ponds 2 & 3 and Stream Channels, Upper Simmons Reservoir, Lower Simmons Reservoir, Almy Reservoir, and Almy Watershed. Total and dissolved copper in surface water of Almy Reservoir, and dissolved copper in Almy Watershed and Upper Simmons Reservoir are also considered to be contaminants of potential ecological concern.

The toxicity of copper to organisms which are exposed to contaminated surface water, and sediments was evaluated, by reviewing toxicological literature from 1967 to 1999. The results of this review are discussed below.

The chronic EPA AWQC for copper is 3.62 ug/1, based on a water hardness of 25 mg/1 as CaCOS (EPA, 1984). The toxicity of copper to aquatic animals is reduced hi the presence of humic acids and selenium (EPA, 1984). Examples of chronic lethal thresholds for some taxa which may be present in the exposure areas include: amphipods *(Gammarus pseudolimnaeus) =* 6.066 ug/1 (hardness = 45); caddisflies *(Clistornia magnified) -* 10.39 ug/1 (hardness = 26); snails *(Physa integra) =* 10.88 ug/1 (hardness = 35 - 55); bluegill sunfish *(Lepomis macrochirus)* = 28.98 (hardness = 45) (EPA, 1984). Midge *(Chironomidae)* emergence was impaired following 32 weeks of exposure to 30 ug/1 (EPA, 1984). Changes in the number of species groups in aquatic insect communities were noted at copper (as copper sulfate) concentrations between 10.7 and 12 ug/1 (Clements, *et al.,* 1988; Clements, *et al,* 1990).

Very little information was available on the ecotoxicological properties of copper. In a 50-week study of the effects of dietary copper on mink, increased mortality of kits was reported at a concentration of 3.2 mg/kg/day, while a NOAEL of 12.9 mg/kg/day was identified for reproductive harm (Aulerich, *et al.*, 1982). Rats exposed to copper in drinking water exhibited hepatic impairment at a dose of 7.9 mg/kg body weight/day (ATSDR, 1989).

Copper concentrations from 1 to 8,000 ug/1 have been shown to inhibit the growth of various plant species (EPA, 1984). The population growth of freshwater algae was reduced following chronic exposure to copper (as copper sulfate) at concentrations between 20 to 40 ug/1 (Winner *et al.,* 1990).

Chromium

Maximum concentrations of chromium in sediments from Sedimentation Ponds 2 $\&$ 3 and Stream Channels, Upper Simmons Reservoir, and Lower Simmons Reservoir exceeded the chromium sediment quality benchmark, and thus copper was considered to be COPEC in sediments from these exposure areas. Neither dissolved nor total chromium in water exceeded water quality benchmarks in any of the exposure areas.

The toxicity of chromium to organisms which are exposed to contaminated surface —wwater, sediments, and wetland soils was evaluated by reviewing lexicological literature from 1967 to 1996. Thorough reviews of ecotoxicological literature pertaining to chromium are presented in Eisler (1986) and Sheedy *et al.* (1991). Pertinent data presented in these reviews are discussed below.

Hexavalent chromium is more bioavailable and toxic compared to trivalent chromium. However, chromium can convert from Cr^{6} to Cr^{3} (and vice versa) under appropriate natural conditions, which significantly lowers toxicity (US EPA, 1985). Water hardness, pH, humic acids and temperature have also been shown to influence chromium toxicity (for example; EPA, 1985; Joshi *et al.,* 1992; Stackhouse *et al,* 1989). It was found that hexavalent chromium was more toxic to the frog, *Rana cyanophlyctis,* at higher temperatures, and/or low pH and hardness (Joshi *et al.,* 1992). Developmental impairment to

tadpoles of the frog, *Rana tigrina,* was recorded at hexavalent chromium concentrations as low as 2 ppm (Abbase *et al.,* 1984).

Both trivalent and hexavalent chromium adversely effected rabbit blood and serum chemistry and caused significant morphological changes in the liver at 1.7 mg/kg body weight/day for 6 weeks (Eisler, 1986). This dose contrasts with a reported NOAEL of 1,468 mg/kg body weight/day for rat survival, body weight, blood and urine clinical chemistry values, and gross and microscopic appearance of organs and tissues (Ivankovic *et al.,* 1975). A NOAEL for hexavalent chromium in drinking water of 2.4 mg/kg body weight/day was reported for systemic/target toxicity in rats (MacKenzie *et al.,* 1958).

Only two reports of chromium toxicity to avian receptors were available. A 5 month NOAEL of 50 mg/kg-diet for black duck survival, reproduction and blood chemistry was reported in Eisler (1986). A 7-day NOAEL of 100 mg/kg-diet was reported for black duck (ducklings) behavior in Eisler (1986).

Eisler (1986) reports that "plants with elevated Cr residues show no toxic effects, although concentrations in excess of 1 ppm in the aqueous medium may inhibit germination of the seed and growth of roots and shoots". Complete elimination of three and six algal species was observed at 0.8 ppm and 8.0 ppm, respectively, after 12 days of exposure (Singh *et al.,* 1991).

Lead

Lead in sediments from Almy Reservoir, and dissolved lead in surface water from Almy Watershed were considered to be potential contaminants of ecological concern.

The toxicity of lead to organisms, which are exposed to contaminated surface water, sediments, and wetland soils, was evaluated by reviewing toxicological literature from 1967 to 1999. The results of this review are discussed below.

The toxicity of lead in water is greatly influenced by several environmental factors. The chronic AWQC for lead (0.54 ppb based on a hardness of 25 mg/1 as CaCO3) is dependent upon site-specific water hardness. However, the water hardness from Almy Watershed was below the minimum allowable concentration of 25 mg/l; therefore, the 0.54 ppb benchmark was used for this exposure area. Lead toxicity is greater in low pH systems than in neutral or basic systems (Starodub, *et al.,* 1987; Buckler, *et al.,* 1987). However, the presence of humic acids have been shown to reduce the toxicity of lead in aquatic systems (Shanmukhappa, *et al.,* 1990). Additionally, organic compounds of lead, such as teraethyllead and tertramethyllead, are more toxic and have a greater tendency for bioaccumulation than inorganic forms (Eisler, 1988), for which body burdens tend to decrease with increasing trophic levels (Wren *et al.,* 1991). In combination, these factors reduce the certainty of applying a single protective benchmark to sites with elevated lead concentrations. In addition, the responses of test organisms to lead exposure vary greatly.

Water concentrations as low as 750 ppb have been shown to cause sublethal lead toxicosis in tadpoles of Green frog *(Rana clamitans)* (Taylor, *et al.* 1990) and concentrations as low as 500 ppb brought about behavioral changes in Bullfrog *(Rana catesbeiand)* tadpoles (Steele, *et al.,* 1989). The 30-day LC-50 value for the Leopard frog *(Rana pipiens)* has been reported at 105 ppm (Eisler, 1988). Concentrations in water of 1 ppb caused reproductive impairment in the freshwater invertebrate *Daphnia magna* (Eisler, 1988). For the Zebra mussel, the NOEL was measured at 116 ppb, and the EC-50 concentration was 370 ppb (Bleeker, *et aL,* 1992). Mortality rates of *Lymnaea palustris,* a freshwater snail, increased with exposure of 19 ppb lead. The acute (48 hour) LC-50 for *Chironomus tentans* was 2.68 ppm (Oladimeji, *et al.,* 1989).

The effects of lead poisoning, or plumbism, in mammals are similar to those documented for humans and include impairment of the central nervous system, the gastrointestinal tract, and the muscular and hematopoietic systems (Eisler, 1988). The following generalizations can be made regarding lead toxicity to animals: there are significant differences in the lead sensitivity of different species; organic lead compounds (tetramethyllead and tetraethyllead) are much more toxic than inorganic lead; and younger developmental stages are more sensitive than adults (Eisler, 1988). Because species-specific toxicological information for lead was not available for mammalian receptors expected to utilize the OU2 exposure areas, the following discussion focuses on reproductive, developmental, and lethal effects to similar species, primarily rodents, obtained from studies reported in Eisler (1988) and ATSDR (1988).

The acute effects of dietary lead on rats *(Rattus* sp.) are well documented. The single oral dose LD-50s for tetramethyllead and tetraethyllead were 108 mg/kg body weight and 12 mg/kg body weight, respectively (Branica *et al.,* 1980). In mice *(Mus* sp.) 2.2 mg tetraethyllead/kg body weight/day reduced the frequency of pregnancy (Clark, 1979). Increased locomotor activity was measured in rats fed 25 mg/kg dietary lead for 3 weeks (Nriagu, 1978). Testicular damage to rats was recorded at a dietary concentration (in a 30 day test) of 0.29/mg/kg body weight/day of lead acetate (in drinking water), while irregular estrous cycles were recorded at 0.014 mg/kg body weight/day (Grant *et al.* 1980). Rats exposed to 25 mg/kg body weight/day of lead acetate in food for two years had statistically increased incidences of kidney tumors (Azar *et al.,* 1973), illustrating the differences in assimilation efficiency between drinking water and food exposure. The lowest oral dose which caused death (LD_{LO}) to guinea pigs was 313 mg/kg body weight (Sax, 1984).

The U.S. Fish and Wildlife Service (Eisler, 1988) has proposed a protective dietary lead criterion (based on irreversible inhibition of ALAD activity in bone marrow and red blood cells in mice) of ≤ 0.05 mg/kg body weight daily. Eisler (1988) also recommends a protective lead drinking water concentration (based on domestic livestock) of <0.100 ppm.

Lead poisoning in birds has been relatively well documented due to the high incidence of avian lead poisoning caused by ingestion of shotgun pellets (Eisler, 1988). However, most of the toxicological data reported in the literature are based on single oral doses of shot pellets. Waterfowl, which have ingested toxic levels of lead, may exhibit nervous system damage, muscular paralysis, liver and kidney damage, and other impairment. Death follows exposure by an average of 2 to 3 weeks (Eisler, 1988).

Tundra swans which spent only a few weeks during migration at a lead contaminated wetland were shown to accumulate lethal concentrations of lead from ingestion of sediment that contained up to 8,700 ppm of lead and plants that contained up to 400 ppm of lead (Blus, *et al.,* 1991). All European starlings *(Styrnus vulgaris)* administered 28 mg/kg body weight/day tetraethyllead or tetramethyllead died within 6 days, while a dose of 2.8 mg/kg body weight/day did not cause death over a period of 11 days, but caused reduced food consumption and/or hyperactivity. Diets containing 1,850 mg/kg lead (as lead acetate) for 4 weeks suppressed growth rates by 47 percent. Diets containing 10 mg/kg of metallic lead powder caused no measurable effects to American kestrels *(Falco sparverius)* over 5 months. Mallards *(Anas platyrhynchos)* fed diets containing 25 mg/kg lead nitrate for 12 weeks experienced decreases in blood ALAD (aminolerulinic acid dehydrase). Ringed turtle doves (*Streptopelia risoria*) exposed to 0.100 ppm Pb 2^+ in drinking water for 2 weeks before pairing and throughout a breeding cycle exhibited reduced testes weight and sperm counts, possibly influencing reproductive fitness (Eisler, 1988). The U.S. Fish and Wildlife Service (Eisler, 1988) has not proposed protective dietary lead criterion for birds.

Lead inhibits plant growth, reduces photosynthesis and reduces mitosis and water absorption (Eisler, 1988). Generally, submergent species are found to have the highest lead concentrations (Crowder, 1991). Lead levels of approximately 500 mg/kg in soil reduced pollen germination by greater than 90 percent in two weed species (Eisler, 1988). Normal germination rates were observed at soil lead levels of 46 mg/kg but other adverse effects were observed at lead levels of 12 mg/kg to 312 mg/kg soil (Eisler, 1988). Some algae accumulate lead from water and there is evidence which suggest that ingestion of such algae may be an important exposure route for aquatic invertebrates (Crowder, 1991).

Manganese

Manganese in sediments from Upper Simmons Reservoir, Lower Simmons Reservoir, Almy Reservoir, and Almy Watershed, and total and dissolved manganese in surface water from the Upper Simmons Reservoir were all identified as contaminants and media of potential ecological concern.

Manganese is acutely toxic at high concentrations in water compared other metals such as Cu or Cd. Stubblefield et al (1997) listed a number of LC50 values of manganese adjusted to a hardness of 50 mg/1 for various aquatic species including: rainbow trout, 3.68 mg/1; Fathead minnow, 7.96 mg/1; *Daphnia magna,* 10.55 mg/1, and *Chironimus tentans,* 207.83 mg/1.

Manganese toxicity is influenced by water hardness. Early life stage toxicity tests were conducted on fertilized eggs and larvae/fry of brown trout *(Salmo trutta).* Brown trout embryos were insensitive to manganese exposure, and there were limited effects of exposure on hatch rate. However, effects on growth were observed and were indicative of manganese

toxicity; therefore, IC25 (the inhibition concentration estimated to cause a 25% reduction in survival or growth of exposed fish compared to control) values were calculated based on the combined effects of these parameters. Manganese toxicity appeared to decrease with increasing water hardness with regard to the IC25 values which were determined to be 4.67, 5.59, and 8.68 mg/1 for 30-, 150-, and 450- mg/1 hardness tests. In a review by Stubblefield et al. of chronic toxicity studies on preexposed trout, exposure of sublethal concentrations of manganese was reported to result in some degree of tolerance among brown trout.

Mercury

The toxicity of mercury to ecological receptors has been extensively reviewed by Eisler (1987), Scheuhammer (1991), Wren *et al.* (1991), and USEPA (1984). The following sections will summarize the findings of these reviews, as well as other current literature pertaining to the ecotoxicology of mercury.

Mercury occurs in the aquatic environment in different forms which may readily be transformed by chemical and biological processes from forms with relatively low toxicity to others with very high toxicity (Wren *et al.,* 1991). Depending on the pH, redox potential, and the type of ligands present, mercury may be present as elemental mercury $[Hg(0)]$, mercurous mercury [Hg(I)], or as mercuric ions [Hg(II)] (USEPA, 1984; Wren *et al.,* 1991; Eisler, 1987). Under natural conditions elemental and mercurous mercury are oxidized to Hg(II), which can be converted by biological and chemical processes to methylmercury (CH_3Hg^T) . Methylmercury is the most toxic and hazardous form of mercury in the environment, largely because it readily penetrates biological membranes and is lipophilic (fat soluble). Methylmercury is subject to bioconcentration, bioaccumulation, and biomagnification.

Uncontaminated surface waters generally contain <5 ng/liter mercury (Gilmour *et al.* 1991). Methylmercury can represent up to 25 percent of total mercury in aerobic fresh waters, and up to 58 percent of total mercury in anoxic fresh waters (Gilmour *et al.,* 1991). In lake sediments methylmercury has been reported as high as 37 percent of total mercury (Gilmour *et al.,* 1991). Soils and wetlands retain large percentages of mercury due to the fact that inorganic mercury [Hg(II)] in atmospheric deposition is extremely reactive and tends to bind strongly with soils and vegetative matter.

Aquatic organisms can be impacted by mercury via direct toxicity, or accumulation from water, sediments, and food. Methylated forms of mercury predominate in the tissues of aquatic organisms. Between 85 and 95+ percent of the mercury in fish tissues is methylmercury (Wren *et al.,* 1991; Porcella, 1994), while methylmercury accounts for 60 percent or less of the total mercury in aquatic invertebrates (Wren *et al.,* 1991). BCFs from water to invertebrates ranged from 75 for water boatmen to 29,000 for damselfly nymphs (Wren *et al.,* 1991). Benthic forms of aquatic invertebrates generally exhibit higher body burdens than those in the water column and predatory organisms tend to accumulate higher concentrations than herbivores or detrivores (Persaud *et al.,* 1987; Wren *et al.,* 1991). Both organic and inorganic mercury associated with food items which are not assimilated is eliminated (Eisler, 1987). Thus, most inorganic mercury which has been absorbed by aquatic

organisms via water or food is excreted or eliminated in a matter of days or weeks (Weiner, 1987; Phillips and Gregory, 1979), while methylmercury is assimilated and bound to protein throughout the bodies of aquatic animals, becoming especially concentrated in fatty tissues such as the liver and kidney.

Because it becomes tightly bound to animal protein, methylmercury is eliminated very slowly, with retention times estimated at months to years (Tollefson and Cordle, 1986). Because methylmercury assimilation is often faster than elimination, methylmercury may accumulate and build up to high concentrations in aquatic organisms, especially long-lived biota such as large piscivorous fish. Aquatic and semi-aquatic prey organisms which concentrate or accumulate mercury may present substantial risk of harm to birds, wildlife, and humans.

The lowest reported methylmercury concentration reported to elicit impairment to invertebrates with chronic exposure was <0.04 ppb (reproductive impairment in *Daphnia magna* (Biesinger *et al.,* 1982). The lowest concentration reported to cause impairment to invertebrates with acute exposure was 0.02 ppb (four-day LC50 for *Faxonella clypeatd)* (Wren *et al.,* 1991). Mercury toxicity to invertebrates varies considerably between species. For example, the LC50 for *Chironomus* sp. (midge) has been reported as 20 ppb, while damselfly and caddisfly larvae had a reported LC50 of 1200 ppb (Rehwoldt *et al.,* 1973).

Information regarding the toxicity of mercury to amphibians is extremely limited. The sole report of a chronic effect for an amphibian species was a study in which metamorphosis was prevented in the Leopard frog *(Rana pipiens)* after 4 months of exposure to 1 ppb methylmercury (Eisler, 1987). Unlike invertebrates and fish, amphibians are not believed to be an important link in methylmercury bioaccumulation in food chains (Scheuhammer, 1991).

A lethal chronic mercuric mercury concentration for Fathead minnows *(Pimephales promelas)* of <0.26 ppb has been reported (EPA, 1984). The lethal chronic concentration of methylmercury to Brook trout *(Salvelinus fontinalis)* has been reported at 0.5193 ppb (EPA, 1984). Chronic exposure to 1.8 ppb methylmercury caused impaired spermatogenesis in male guppies *(Poecilia reticulatd).* Enzymatic changes in bluegill fish *(Lepomis macrochirus*) have been reported after acute exposure to 3.4×10^{-12} molar (M) methylmercury (Hossain *et al.,* 1986).

From a toxicological perspective, dietary methylmercury is a better indicator of potential health risks than is the total mercury concentration (Scheuhammer, 1991). The feeding habits of species determine relative risks to methylmercury exposure. Species which feed on aquatic organisms are at higher risk than those that are associated with terrestrial food chains and carnivorous species are at higher risk than herbivorous or detrivorous species.

Reproductive and developmental harm has been reported at dietary methylmercury concentrations as low as 0.05 mg/kg-body weight/day in a study which examined the occurrence of fetal eye anomalies in rats (ATSDR, 1989). This dosage was also identified as a NOAEL in studies of male mink (Wobeser, 1976). Death occurred in sensitive species of mammals at methylmercury concentrations of 0.1 to 0.5 mg/kg body weight (1.0 - 5.0 mg/kg) diet) (Eisler, 1987). Inorganic mercury impaired the renal function of rats at 1.27 mg/kgbody weight/day (ATSDR, 1989). Inorganic mercury administered in drinking water for 530 days at 2.2 mg/kg body weight/day caused reduced body weight and water intake in mice, while the same effect was elicited by organic mercury administered in drinking water for 18 months at 0.80 mg/kg body weight/day (ATSDR, 1989). The U.S. Fish and Wildlife Service (Eisler, 1987) has proposed a protective total mercury criterion for small mammals of <1,100 ug/kg diet (fresh weight) or <250 ug/kg body weight/day.

The tissue-mercury concentrations associated with neurological impairment and death in birds are often similar despite differences in species, body size, dietary mercury concentration, or duration of exposure (Scheuhammer, 1991). The dietary concentrations of methylmercury that are required to elicit reproductive impairment in birds are about 20 percent of those required to produce overt toxicity (Scheuhammer, 1991).

A study in which three generations of mallard were fed a 0.5 mg/kg-dry weight diet of methylmercury (0.01 mg/kg-fresh weight diet), resulted in reduced egg production and hatching (Eisler, 1987). Dietary concentrations of methylmercury as low as 0.3 mg/kg (wet weight) decreased egg laying and territory use in the Common loon *(Gavia immer)* and 0.4 mg/kg severely effected territory use and egg laying (Scheuhammer, 1991). The U.S. Fish and Wildlife Service (Eisler, 1987) has proposed a protective criterion for birds of 50 to <100 ug/kg diet (fresh weight) or <640 ug/kg body weight/day.

The amount of information available on the toxicity and bioaccumulation potential for mercury in aquatic plants is meager. Toxic effects include reduced growth rates, discoloration, necrosis of floating leaves, and death of roots (Crowder, 1991). Sedges and water lilies may move mercury up to the leaves (Siegel *et al.,* 1987), while many other species concentrate mercury in their roots or rhizomes (Crowder, 1991). Maury *et al.* (1988) reported that methylmercury sediment concentrations as low as 0.12 ppb impair the growth of Elodea densa. The ratio of methylmercury to total mercury in the presence of contaminated sediments was reported as >20 (Ribeyre *et al.,* 1991).

Silver

The toxicological literature contains abundant references to the toxicity of silver to aquatic organisms, yet very little information is available on the toxicity of silver to semiaquatic, terrestrial, or avian receptors. The following sections summarize the results of our literature search.

Mechanisms of metal toxicity include blocking of essential functional groups of proteins or enzymes, displacing essential metal ions in proteins or enzymes, and modifying the site of biological activity in proteins or enzymes (Connell and Miller, 1984). Silver is among the most toxic of all metals, causing the inactivation of enzymes critical to biological functions by one or more of the above mechanisms. Many silver compounds have been used

in medicine as germicides and antiseptics due to their toxic effects on bacteria and other microbes.

The chronic U.S. EPA AWQC for silver is 0.12 ppb (EPA, 1980). The estimated maximum acceptable toxicant concentration for silver nitrate, based on Fathead minnow *(Pimephales promelas)* survival, is between 0.37 and 0.65 ppb (Holcombe *et al.,* 1983). The chronic no-effect concentration of silver (as silver nitrate) for rainbow trout *(Salmo gairdneri)* was between 0.09 and 0.17 ppb (Davies *et al.,* 1978).

Brachiopods, mollusks, and arthropods (especially crustaceans) accumulate silver in the heptopancreas and nephridial organs (EPA, 1980). BCFs for mollusks/sediment ranged from 0.02 to 6.14 (EPA, 1980).

The toxicity of silver compounds to mammals can be classified as moderate (EPA, 1980). The chronic oral LD_{90-100} for silver sulfadiazine in mice was >1050 mg. (EPA, 1980). Administration of 18.1 mg silver/kg/day (in water) for 125 days did not elicit a response in mice (Rungby *et al.,* 1984). Administration of 362.4 mg silver/kg/day (in water) caused the death of 25 percent of test rats (Walker, 1971).

Silver concentrations in plants tend to be highest in seeds, nuts, and fruits compared to other parts (EPA, 1980). No other information was available on silver toxicity or bioaccumulation in plants.

Zinc

The toxicity of zinc to organisms which are exposed to contaminated surface water, sediments, and wetland soils was evaluated by reviewing toxicological literature from 1967 to 1996. Thorough reviews of ecotoxicological literature pertaining to zinc are presented in Eisler (1993) and Sheedy *et al.* (1991). Pertinent data presented in these reviews are discussed below.

The acute and chronic EPA AWQC for zinc are 36.15 and 32.75 ug/1, respectively, based on a water hardness of 25 mg/l as $CaCO₃$ (EPA, 1980). Many factors influence the relative toxicity of zinc to aquatic and semi-aquatic organisms. For instance the toxicity of zinc is higher to embryos and juveniles than to adults, to starved animals, at elevated temperatures, at low dissolved oxygen concentrations and in the presence of cadmium and mercury (Eisler, 1993). The lethal limit for tadpoles of the toad, *Bufo boreas,* has been reported to lie between 100 and 500 ug/1 (Porter *et al.,* 1976). Fifty percent of Narrowmouthed toad *(Gastrophryne carolinensis)* embryos exposed to 10 ug/1 zinc were dead or deformed within 7 days (Eisler, 1993). Acute LC50 (96 hour) values for freshwater invertebrates were between 32 and 40,930 ug/1 (Eisler, 1993). The 10-day LC50 for midge larvae, *(Tanytarsus dissimilis),* a taxa which may be present at the site, was 37 ug/1 (Eisler, 1993). Concentrations as low as 76 ug/1 inhibited reproduction in bluegills *(Lepomis macrochirus) (Eisler,* 1993).

BCFs for aquatic insects range from 107 to 1,130 and range from 51 to 432 in fish (Eisler, 1993).

Zinc is relatively non-toxic in mammals (Eisler, 1993). The reproductive organs of adult male rats were damaged by exposure to 500 mg/kg diet for 3 weeks or longer (Eisler, 1993). The laboratory white rat can tolerate a dietary concentration of 320 mg/kg body weight, while 640 mg/kg body weight is considered harmful (Llobet *et al.,* 1988).

The growth and reproduction of terrestrial invertebrates may be impaired by soil concentrations as low as 470 mg/kg and soil concentrations of 1,600 mg/kg zinc have been shown to reduce natural populations of soil invertebrates (Eisler, 1993).

Eisler (1993) recommended that bird diets should contain less than 178 mg/kg zinc (dry weight) to prevent marginal sublethal effects, and less than 2,000 mg/kg zinc (dry weight) to prevent the death of chicks.

Sensitive terrestrial plants die when zinc concentrations in soil exceed 100 mg/kg (Eisler, 1993). The sensitivity of aquatic plants is extremely variable. As little as 19 ug/1 inhibits the growth of some algae, while some tolerant strains can live in waters containing 3 g/1 zinc (Eisler, 1993).

7.00 AMMONIA

Ammonia is commonly found in natural waters primarily due to the normal breakdown of proteins; however, at high concentrations, ammonia can be toxic to aquatic receptors. The following paragraphs summarize ammonia toxicity to a number of aquatic receptors reported by the National Research Council Subcommittee on Ammonia (1979).

Only unionized ammonia is considered toxic, and several factors can affect ammonia toxicity, including the pH of water, temperature, dissolved oxygen concentrations, salinity, and the presence of other contaminants. For example, in a study on the effects of DO on ammonia toxicity, decreases in dissolved oxygen concentrations lead to higher ammonia toxicity to rainbow trout. Increases of carbon dioxide up to 30 ppm lead to reduced ammonia toxicity. Mixtures of ammonia with other contaminants such as phenol, zinc sulfate, or copper sulfate, resulted in additive toxicity. In a different study, the mixture of ammonia and hydrocyanic acid was found to be more toxic compared to the toxicity of the individual substance.

A 24 hr LC50 value of 0.5 mg/1 of ammonia for rainbow trout was reported fairly consistently in various studies; however, lower threshold values, such as LC50 of 0.2 mg/1 have been reported in other studies. In an ammonia toxicity study designed to simulate natural conditions, ammonia concentrations that were fluctuated from 0.5 to 1.5 times the LC50 on a two hour cycle lead to a higher increase in trout mortality compared to exposure to a constant concentration.

Exposure of rainbow trout to sublethal concentrations of ammonia lead to an increased rate of detoxification via induction of urination. However, in studies of sublethal effects on other fish species, chronic exposures lead to toxic effects. Damaged skin, gills, and intestines, and disruption to the circulatory system including hemorrhage and congestion were observed in carp due to exposure of a sublethal concentration of ammonia. Exposures to chronic concentrations were found to result in more harmful effects than effects resulting from short-term exposure. Hyperplasia of gill tissue in salmon was reported to have resulted from chronic exposure of 0.002 mg/1 for a period of six weeks. Reduced growth rate and reduced physical stamina was also attributed to chronic exposure.

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APPENDIX G

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JUNE 1998 CHRONIC TOXICITY TEST REPORTS

NEW ENGLAND BIOASSAY, INC.

CHRONIC TOXICITY TO THE DAPHNID, Ceriodaphnia dubia, AND THE AMPHIPOD, Hvalella azteca, OF FRESH WATER AND SEDIMENT SAMPLES COLLECTED IN THE VICINITY OF THE CENTRAL LANDFILL IN JOHNSTON, RI ON 27 AND 28 MAY 1998 BY GZA GEOENVIRONMENTAL, INC.

'30 June 1998

Performed For:

GZA GeoEnvironmental, Inc. 320 Needham Street Newton Upper Falls, MA 02164

Performed by:

New England Bioassay, Inc. 77 Batson Drive Manchester, Connecticut 06040

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SUMMARY

*** Survival of daphnids in the five site water samples was compared against survival in the concurrently run control groups (Fisher's exact test); in addition, daphnid reproduction was compared against reproduction in the concurrently run control group (ANOVA and Dunnett's multiple comparison test).

b Survival of amphipods in the five site sediments were compared against a survival criterion of 80% at 14 days; in addition, amphipod survival and growth were compared among the five site sediments with the control group excluded (ANOVA and Tukey's multiple comparison test).

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TABLE OF CONTENTS

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TABLE OF CONTENTS **(Continued)**

LIST OF APPENDICES Page

APPENDIX A

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APPENDIX B

APPENDIX C

LIST OF TABLES

CHRONIC TOXICITY TO THE DAPHNID, Ceriodaphnia dubia, AND THE AMPHIPOD, **Hvalella azteca.** OF FRESH WATER AND **SEDIMENT SAMPLES COLLECTED IN THE VICINITY OF THE CENTRAL LANDFILL IN JOHNSTON, RI ON 27 AND 28 MAY 1998 BY 6ZA GEOENVIRONMENTAL, INC.**

30 June 1998

INTRODUCTION

This report contains results of chronic toxicity tests performed using grab samples of potentially-contaminated fresh water and sediment collected on 27 and 28 May 1998 by GZA GeoEnvironmental staff from a freshwater site in the vicinity of the Central Landfill in Johnston, RI. Chronic tests with the water samples were conducted by exposing the freshwater daphnid, Ceriodaphnia dubia, to each of the five site water samples for 7 days in a static-renewal test system; chronic tests with the whole-sediment samples were conducted by exposing the freshwater amphipod, Hyalella azteca, to each of the five sediment samples for 14 days in a flow-through test system. All work reported here was performed at New England Bioassay (NEB) located in Manchester, CT.

MATERIALS AND METHODS

Sample Collection and Handling

Grab samples of potentially-contaminated fresh water and sediment were collected on 27 and 28 May 1998 by GZA personnel (Table 1). Grab samples of water and sediment were picked up by a NEB courier on the same days as sample collection. Copies of chain of custody forms are provided in Appendix A. Upon receipt at NEB, the water and sediment samples were logged into the laboratory and assigned unique identification numbers (Table 1) . Standard wet chemistry analyses [pH, dissolved oxygen, specific conductivity, total residual chlorine (TRC), hardness, and alkalinity] were performed on the freshwater samples when they were received (Table 2).

NEW ENGLAND BIOASSAY. INC.

Sample Description	Sample Date \sim	Sample Type	NEB ID Nos.
	WATER SAMPLES		
GZA No. SW98-50	05/27/98	Grab	98-1803
GZA No. SW98-51	05/27/98	Grab	$98 - 1804$
GZA No. SW98-52	05/27/98	Grab	98-1805
GZA No. SW98-53	05/28/98	Grab	98-1809
GZA No. SW98-54	05/28/98	Grab $\mathcal{R}^{\bullet}_{\mathcal{R}}$	98-1810
		$\mathcal{A}^{\mathcal{A}}$	
	SEDIMENT SAMPLES		
GZA No. SED98-50	05/27/98	Grab	98-1806
GZA No. SED98-51	05/27/98	Grab	98-1807
GZA No. SED98-52	05/27/98	Grab	98-1808
GZA No. SED98-53	05/28/98	Grab	98-1811
GZA No. SED98-54	05/28/98	Grab	98-1812

TABLE 2. WET CHEMISTRY RESULTS FOR FRESH WATER SAMPLES

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TRC readings above 0.1 mg/L may be due to positive interference with the colorimetric DPD method used; no dechlorination with sodium thiosulfate was performed.

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Chronic toxicity tests were initiated with the water samples on the same days that the samples were received. The remainder of each water sample was stored in a cold room $(4^{\circ} \pm 2^{\circ}C)$ in the darkduring testing; an aliquot of each sample was removed each day from the cold room, warmed to the test temperature, and then used for the test-solution renewals.

Sediment samples (two 1-gallon plastic containers per sediment) were stored immediately after receipt in the dark in a cold room $(4^{\circ} \pm 2^{\circ}C)$ until testing was initiated. On the morning of 2 June 1998, the five sediment samples were removed from the cold room and any large stones and sticks were removed; the sediment samples from both containers were composited and then manually stirred to ensure homogeneity. After homogenization, 200 g of the wet sediment was then measured into a 1000-mL Mason jar; overlying water was then added to each replicate beaker. After settling overnight, chambers containing the sediment and overlying water were ready for the introduction of the test organisms on 3 June. All five sediments were of a black muck-silt consistency with a mild hydrogen sulfide odor; SED98-51 contained some sand. The SED98-053 sample contained many hair-like fibers; the SED98-054 sample contained a large amount of organic matter, mostly grass.

In addition to testing the five site sediment samples, an additional artificial sediment was evaluated as a quality-control check to determine the adequacy of the test system. The artificial sediment sample was prepared by NEB on 1 June 1998- from a recipe described in EPA guidance manual (EPA, 1994b; pages 24-25); the composition of the artificial sediment was 78.5% sand, 16.6% silt/clay, and 5% peat moss. After preparation, the artificial sediment was handled similarly to the site sediments.

Test Organisms

Ceriodaphnia dubia

Ceriodaphnia dubia used in the chronic toxicity tests were obtained from NEB in-house cultures; daphnids were cultured in laboratory-prepared fresh water under controlled conditions (temperature 25° \pm 2°C; photoperiod 16 h light and 8 h dark). $C.$ dubia were individually cultured in 30-mL plastic cups (one C. dubia per cup) containing 15 mL of laboratory water. Each culture chamber received 50 μ L of a yeast/alfalfa/Tetramin (YAT) food suspension (EPA, 1994a) and 150 μ L of the green alga, Selenastrum capricornutum, when cultures were changed. Survival and reproduction of culture animals were checked each time culture water was changed (on a daily basis after production of a first brood of young). After 14 days, adults were discarded and new cultures were started. All young were removed from culture chambers 24 h before starting a test to ensure that only ceriodaphnids \leq 24 h old would be available to start tests.

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Hyalella azteca

Hyalella azteca Saussure (Crustacea, Amphipoda) is one of the recommended test species for sediment tests because of its ease of culturing, relatively short generation time, relatively large size as juveniles, and ease of handling the immature stages. This species is widely distributed throughout the U.S. in permanent lakes, ponds and streams; H. azteca is an epibenthic detritivore and will burrow in the sediment surface. Its feeding habits include both filter feeding and ingesting sediment. Amphipods required for testing (480 animals for sediment tests, 40 animals for initial lengths and weights, and 120 animals for reference toxicant tests) were obtained from a commercial supplier.

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Aquatic Biosystems (ABS; Fort Collins, CO) maintains known-age cultures. ABS shipped approximately 640 juvenile amphipods (7- to 13-days old) to NEB on 1 June 1998 by overnight courier. NEBreceived the animals in good condition on 2 June 1998. The amphipods were transferred to a 50:50 mix of laboratory water and shipment water. On the morning of 3 June, the amphipods were sorted, by length (approximately 2 mm as estimated with a millimeter ruler) and 480 amphipods were used to initiate the chronic sediment tests. At test initiation, four subsamples of 10 amphipods each were measured for total length (mean size: 1.8 mm/amphipod) and then oven dried and weighed (mean dry weight: 0.076 mg/amphipod) . The remaining amphipods were used in a reference toxicant test with potassium chloride.

Test Water

The control water for the C . dubia toxicity tests was laboratory fresh water (SRCF nominal hardness and alkalinity: 48 mg/L and 31 to 35 mg/L as $CaCO₃$, respectively). The overlying water used in the whole-sediment chronic toxicity tests with $H.$ azteca was also prepared in the laboratory (MHRCF nominal hardness and alkalinity: 92-98 mg/L and 60-65 mg/L as $CaCO₃$, respectively). The laboratory waters for the chronic tests were prepared based on instructions cited in the EPA chronic testing guidance manual (1994a). The base water used in preparing the SRCF and MHRCF was deionized water from a Millipore Milli-Q® water system; reagent grade salts were added in the appropriate amounts to carboys containing deionized water and mixed. After preparation, each batch of water was aerated at room temperature and then used in testing.

Test Systems

C. dubia Static-Renewal Chronic Surface Water Toxicitv Tests $\overline{}$

The C. dubia chronic toxicity test procedures are based on recommendations in the EPA guidance document (1994a) titled "Methods for Estimating the Chronic Toxicity of Effluents and Receiving Waters to Freshwater Organisms." For chronic testing, young C. dubia (< 24-h old at test initiation) were continuously exposed for 7 days under static-renewal conditions to the undiluted site'water samples. One water sample was collected from each site; therefore, all test solution renewals were performed with the initial water sample. C. dubia were individually exposed in 30-mL plastic cups containing 15 mL of site water or control water with 10 replicate beakers per water sample. Test beakers were maintained under the specified conditions (mean and individual test temperatures 25° \pm 1°C; photoperiod 16 h light and 8 h dark). Surviving Ceriodaphnia were transferred daily with a large-bore pipette to newly prepared solutions containing food.

Temperature, dissolved oxygen, pH, and specific conductivity' were measured daily on composite samples of newly prepared solutions. Temperature, dissolved oxygen, and pH were measured in one replicate of the 24-h-old test solutions for each sample. Observations on the number of live and dead (or immobilized) animals were made daily. Reproduction was monitored daily by counting number of live and dead young per female when adults were transferred to new solutions. Young were discarded after counting.

H. azteca Flow-through Chronic Whole-Sediment Toxicity Tests

The amphipod chronic toxicity test procedures are based on recommendations in the EPA guidance document (1994b) titled "Methods for Measuring Toxicity and Bioaccumulation of Sediment associated Contaminants with Freshwater Invertebrates."

6

Per client request, the test duration was extended from 10 days to 14 days and water-renewals were changed from a static/staticrenewal system to a flow-through system. Immature amphipods (approximately 2 mm in size at test initiation) were continuously exposed for 14 days under flow-through conditions to sediment samples from each of the five sites. An artificial sediment was also evaluated. Laboratory prepared water (nominal hardness: 92 to 98 mg/L as $CaCO₃$) was used as overlying water for the tests.

Immature amphipods were exposed in groups of 10 animals in l-quart glass Mason jars containing 0.2 kg of sediment and approximately 500 mL of laboratory-prepared water, with eight replicate beakers per concentration (80 animals per sediment). Each test chamber contained a 7/8-inch hole in the side of the chamber covered with 800 μ m Nitex mesh at the 750-mL mark. Laboratory water was continuously added to each test chamber using an Ismatic peristaltic pump (Model 7338-20) at a rate of approximately 42 to 60 mL per hour (two chamber volumes per day) ; excess water was removed through the overflow hole.

Test animals were fed 4.3 mL of a mixture of YAT per beaker per day. After adding amphipods to the test chambers, the chambers were loosely covered to reduce evaporation. Because the amphipods were fed daily during the test, test chambers were gently aerated (single bubble aeration) during testing to maintain adequate dissolved oxygen levels (> 30% saturation) in the overlying water. Test chambers were placed in a water bath in a controlled environment room under the test conditions (temperature 23° \pm 2°C; photoperiod 16 h light and 8 h dark).

Temperature, dissolved oxygen, pH, and conductivity were measured daily in one replicate per concentration. Hardness and alkalinity were measured on each batch of laboratory water before use; in addition, hardness and alkalinity were measured on a sample of overlying water from one replicate in each set of sediment chambers at the end of the test. Ammonia levels were measured in the overlying water at the end of the test. Observations on number of live and dead animals were made when chronic tests were terminated (14 days).

 $\overline{7}$

At the end of 14 days when tests were terminated, overlying water and sediment in the test chambers were poured through a series of stainless steel sieves to collect the amphipods. After most of the overlying water was removed from the test beakers, the remaining overlying water was poured through #35 (500 μ m) and #50 (300 *^m)* stainless steel sieves. If the amphipods were still in the sediment, the top layer of the sediment was rinsed with a squirt bottle containing deionized water and poured through the sieve. The amphipods were usually large enough to be trapped by the larger sieve.

After sieving, amphipod survival counts were performed. All live amphipods within a replicate were placed in pre-weighed drying pans and kept in a drying oven overnight. (100° \pm 5°C). The following day, the weigh pans containing the dried amphipods were reweighed as a group using a 5-place balance. Total dry amphipod weight per replicate was then divided by the number of amphipods weighed to obtain an average dry amphipod weight per replicate.

Reference Toxicant Tests

Acute reference toxicant tests were conducted with in-house cultures of C. dubia (sodium chloride; NaCl) and with purchased stocks of \underline{H} . azteca (potassium chloride; KCl). The 48-h data from the NaCl reference toxicant test was used to calculate an LC_{50} for C. dubia; the NaCl reference toxicant test consisted of five nominal concentrations $(0.3, 0.5, 1.0, 2.0, \text{ and } 3.0 \text{ g/L NaCl})$ and a dilution-water control. The 96-h data from the KC1 reference toxicant test was used to calculate an LC_{50} for $H.$ azteca; the KCl reference toxicant test consisted of five nominal concentrations (0.0625, 0.125, 0.25, 0.5, and 1.0 g/L KC1) and a dilution-water control. Laboratory-prepared fresh water [nominal hardness values of 48 mg/L for C . dubia (SRCF) and 92 mg/L as CaCO, for H. azteca (MHRCF)] was used as dilution and control water for the reference toxicant tests.

8

Statistical Analysis

Chronic toxicity data from the C. dubia static-renewal tests were used to determine if the five site waters exhibited significant chronic effects when compared with laboratory-water control animals. C. dubia survival data were analyzed by using Fisher's exact test comparing survival of organisms in the site waters with survival in the laboratory-water control. A parametric ANOVA and Dunnett's multiple comparison test (if the ANOVA was significant, P < 0.05) were used for comparing C. dubia reproduction in the test concentrations with that in the control water.

Chronic toxicity data from the amphipod flow-through tests were used to determine if the sediment samples exhibited significant chronic effects. Because of the poor survival of the amphipods in the artificial laboratory sediment, chronic effects on amphipod survival in the five site sediments were compared against a survival criterion of 80% at 14 days.

In addition, amphipod survival and growth were compared among the five site sediments with the control group excluded (ANOVA and Tukey's multiple comparison test); Tukey's multiple comparisons test allows comparisons of either survival or growth among all five site sediment samples excluding the control group. Printouts for statistical analyses of chronic toxicity test data for C . dubia and H. azteca are provided in.Appendices A and B, respectively. Copies of the raw data sheets and statistical summary printouts for the C. dubia and H. azteca reference acute toxicity tests are located in Appendix C.

RESULTS

C. dubia Static-Renewal Chronic Toxicitv Tests With Water Samples

Chronic toxicity tests with five water samples collected from the Central Landfill site in Johnston, RI were conducted with C. dubia (Table 3) . Analysis of daily survival data indicated that survival of C. dubia was 100% for all five sites; C. dubia survival was not significantly reduced in any of the water samples (Fisher's exact test; $P > 0.05$) when compared with laboratory-water control survival (100% survival after 7 days).

The results of 7-day survival and reproduction tests with Ceriodaphnia dubia with five surface waters provided by GZA on 27 and 28 May 1998 from the Central Landfill in Johnston, RI are summarised below. Survival of C. dubia to each of the five surface waters was 100% after a 7-day exposure. Daphnid reproduction was not significantly reduced in any of the water samples when compared with their respective controls; young production averages ranged from 24.5 to 41.8. for the five samples.

Analysis of reproductive data (Table 3) by ANOVA and Dunnett's test indicated that C. dubia reproduction was not significantly reduced (P > 0.05) in any of the site water samples when compared with reproduction in their respective laboratory-water controls. The ranges of water-quality measurements for the C. dubia tests for dissolved oxygen, temperature, pH, and specific conductivity were 7.2 to 9.3 mg/L, 24.3° to 25.5°C, 7.3 to 8.4 SU, and 180 to 730 μ mhos/cm, respectively (Table 4). (For more detail, see raw data sheets in Appendix A.)

10

TABLE 3. SURVIVAL AND REPRODUCTION OF DAPHNIDS, **Ceriodaphnia dubia,** IN **7**-DAY CHRONIC TOXICITY TESTS WITH FIVE SURFACE WATER SAMPLES COLLECTED ON 27 AND 28 MAY 1998 FROM THE CENTRAL LANDFILL SITE IN JOHNSTON, RI

		Daily Survival (%)						Total Young ^ª per female			
Test Concentration	$\mathbf{1}$	\overline{a}	3	4	5		7		N	x	CV $(*)$
Sample Date:	27 May 1998									Test Dates: 28 May - 4 June 1998	
LAB CONTROL ^b	100	100	100	100	100	100	100		10	27.5	37.5%
SW98-50 SW98-51	100 100	100 100	100 100	100 100	100 100	100 100	100 100		10 10	35.9 24.5	26.0% 19.6%
SW98-52	100	100	100	100	100	100	100		10	29.8	26.0%
Sample Date:	<u>28 May 1998</u>									Test Dates: 29 May - 5 June 1998	
LAB CONTROL ^b	100	100	100	100	100	100	100		10	43.8	8.4%
SW98-53	100	100	100	100	100	100	100		10	41.8	18.5%
SW98-54	100	100	100	100	100	100	100		10	38.9	14.0%
N: variation.										number of females at start of test; X: Mean; CV: coefficient of	
ь SRCF: soft reconstituted freshwater was used as laboratory control water.											
TABLE 4. Sample Description		WATER QUALITY MEASUREMENTS FOR Ceriodaphnia dubia TESTS DO (mg/L)			Temp. (°C)		рH (SU)		Cond. $(\mu \text{mhos/cm})$		
			Test Dates:				27 May - 4 June 1998				
LAB CONTROL	7.8 $7.5 - 8.2$			25.0 $24.4 - 25.5$			7.8 $7.3 - 8.0$		182 180-185		
SW98-50		8.1 $7.4 - 9.0$			-24.9 $24.4 - 25.3$			8.0 $7.8 - 8.1$		506 $504 - 510$	
$SW98 - 51$		8.2 $7.4 - 8.7$			24.9 $24.3 - 25.2$			8.1 $7.6 - 8.4$		726 $723 - 730$	
SW98-52	8.2 $7.4 - 8.9$			24.9 $24.3 - 25.2$			8.1 $7.6 - 8.3$		598 591-608		
							Test Dates: 28 May - 5 June 1998				
LAB CONTROL			7.7 $7.5 - 8.3$			24.9 $24.5 - 25.4$		7.8 $7.5 - 8.1$		183 180-188	

 $SW98-53$ 8.2 25.0 8.1 485

 $SW98-54$ 8.1 24.9 7.9 496

8.2 25.0 8.1 485
7.2 - 9.3 24.5 - 25.3 7.7 - 8.4 481 - 48

7.3 -8.6 24.7 -25.4 7.5 -8.2 491-503

NEW ENGLAND BIOASSAY. INC

481-488

H. azteca Flow-through Chronic Whole-Sediment Toxicity Tests

Chronic toxicity tests with five sediment samples collected by GZA on 27 and 28 May 1998 from the Central Landfill site were conducted with H. azteca during 3-18 June 1998-(Table 5). Survival of H. azteca to the five sediments was > 80% after a 14-day exposure which was higher than, the EPA-control acceptability criterion of ≥ 80 % survival at test completion for reference sediments. After a 14-day exposure, amphipods exposed to the five sediments increased in weight by an average of 3.5x (range: 3.3x to 4.Ox) when compared with initial amphipod weights at test .*f* initiation; average amphipods weights ranged from a low of 0.252 mg (SED98-50) to a high of 0.303 mg (SED98-53) compared with initial average amphipod weight of 0.076 mg (Day 0 dry weight).

Amphipod survival in the artificial sediment prepared by NEB was *i* poor (35% after 14 days) indicating that the laboratory sediment was not an acceptable substrate for use in a 14-day flow-through test with amphipods. Surviving amphipods were also about half the weights of exposed amphipods (0.124 mg/amphipod) suggesting that amphipods exposed in the artificial substrate may have "starved" because of lack of a natural food source, longer exposure period, and flow-through conditions.

Test results suggest that the five sediments were all similar in survival and weights (i.e., no statistical differences among the five samples) . The high survival (> 80% after 14 days) indicate no adverse effects on amphipod survival. Weight comparisons were made among the various site sediments using ANOVA and Tukey's multiple comparisons test; no significant differences in weights were observed when the five sites were compared against each other excluding the control group.

Ranges of water-quality measurements for overlying water in the H. azteca tests for dissolved oxygen, temperature, pH, and specific conductivity were 6.7 to 9.2 mg/L, 22.0° to 23.4° C, 7.3 to 8.1 SU, and 302 to 422 μ mhos/cm, respectively (Table 6); ammonia levels in overlying water ranged from 0.2 to 0.5 mg/L at test termination (For more detail, see raw data sheets in Appendix B.)

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Control group not included in statistical analysis. NS: Not significant (P > 0.05); *: significant (P < 0.05); Analysis of Variance (ANOVA) and Tukey's Multiple Comparisons Test. α

TABLE 6. WATER QUALITY MEASUREMENTS FOR Hyalella azteca TESTS

Reference Toxicant Tests

The NaCl 48-h LC₅₀ was estimated for C . dubia using survival data generated from an acute reference toxicant test with in-house daphnid cultures (test dates: $1-3$ June 1998). The 48-h LC_{so} for $C.$ dubia was 2.0 q/L KCl (95% confidence limits of 1.0 to 3.0 q/L NaCl; binomial method). Survival of control daphnids was 100% at test completion (48 h).

The KCl 96-h LC₅₀ was estimated for \underline{H} . azteca using survival data generated from an acute reference toxicant test with the same cohort of purchased amphipods (test dates: 5-9 June 1998). The 96-h LC₅₀ for H. azteca was 0.30.g/L KCl (95% confidence limits of 0.24 to 0.37 g/L KC1; trimmed Spearman-Karber method) . Survival of control amphipods was 95% at test completion (96 h).

The results of the acute reference toxicant tests with NaCl and KC1 indicate the health of the organisms was satisfactory. A copy of the reference toxicant data for C. dubia and H. azteca is found in Appendix C.

SUMMARY

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The results of chronic toxicity tests conducted with grab samples of water (C . dubia chronic tests) and sediment (H . azteca chronic tests) collected on 27 and 28 May 1998 from the Central Landfill in Johnston, Rhode Island by GZA GeoEnvironmental staff are summarized below. •» *•*

Summary of Results

^a Survival of daphnids in the five site water samples was compared against survival in the concurrently run control groups (Fisher's exact test);' in addition, daphnid reproduction was compared against reproduction in the concurrently run control group (ANOVA and Dunnett's multiple comparison test).

^b Survival of amphipods in the five site sediments were compared against a survival criterion of 80% at 14 days; in addition, amphipod survival and growth were compared among the five site sediments with the control group excluded (ANOVA and Tukey's multiple comparison test).

REFERENCES

U.S. EPA. 1994a. Methods for Estimating the Chronic Toxicity of Effluents and Receiving Waters to Freshwater Organisms. (Lewis, PA, Klemm, DJ, Lazorchak, JM, Norberg-King, TJ, Peltier, WH, and Heber, MA; Eds.) 3rd edition, EPA-600/4-91/002. Environmental Monitoring Systems Laboratory, Cincinnati, OH, 341 pp.

U.S. EPA. 1994b. Methods for Measuring the Toxicity and Bioaccumulation of Sediment-associated Contaminants with Freshwater Invertebrates. 1st edition, EPA 600/R-94/024, Office of Research and Development, Washington, B.C., 133 pp.

CERTIFICATION

I certify that the data presented in this report were obtained under my direction or supervision in accordance with protocols of the U.S. Environmental" Protection Agency. The information is, to the best of my knowledge and belief, true, accurate, and complete.

D. Cooney, Ph.D. oratory Director

 $130/98$

APPENDIX A

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CHAIN OF CUSTODY DOCUMENTATION CHRONIC TOXICITY DATA SHEETS AND STATISTICAL ANALYSES FOR DAPHNID, <u>Ceriodaphnia dubia</u>, TESTS WITH SITE WATER SAMPLES

NEW ENGLAND BIOASSAY, INC.
77 BATSON DRIVE
MANCHESTER, CT 06040
(860) 643-9560

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MISCELLANEOUS DATA INFORMATION

INITIAL CHEMISTRY DATA

Data Reviewed By: $\sqrt{2}$. $\frac{\partial \overline{u}u}{\partial t}$ Date Reviewed: $\frac{u}{5}/48$

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MISCELLANEOUS DATA INFORMATION

INITIAL CHEMISTRY DATA

NOTE: $NA = NOT APPLICABLE$

Data Reviewed By: R. Mulk Date Reviewed: 6/5/98

NEW ENGLAND BIOASSAY TOXICITY DATA FORM CHRONIC COVER SHEET

INVERTEBRATE

TEST SOLUTION VOLUME: 15 (mL) NO. ORGANISMS PER TEST CHAMBER: $\overline{\mathbf{1}}$ NO. ORGANISMS PER CONCENTRATION: 10 NO. ORGANISMS PER CONTROL: 10

START DATE: $\frac{\int \mu_{U} f_3.5|25|98}{\mu_{U}}$ at $\frac{1215}{\sqrt{112}}$ (hours)

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Laboratory Control Water

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Test
Day Batch Number $C985 - CO8$ (985-008 <u>985-008 .</u> C485-01 I $C185-011$ $Q8501$ $C985 - 011$ ϵ

Alkalinity
mg/l CaCO₂
3 Hardness mg/I CaCO, 31 31 37

Results of Ceriodaphnia dubia Chronic Test

NOEC: NO-OBSERVED-EFFECT-CONCENTRATION

LOEC: LOWEST-OBSERVED-EFFECT-CONCENTRATION

ANIMAL CONDITION/BEHAVIOR:

COMMENTS: REVIEWED BY: R. Taulk

DATE: $6/5/98$

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 $\frac{\text{Test No:}}{\text{Cov}} \frac{\text{S}}{\text{Cov}} = \frac{\text{S}}{\text{Cov}} \frac{\text{S}}{\text{Cov}}$

Date Reviewed: 100190

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Det Reviewed By. W. Q_0 . M_2

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GZA - Central Landfill - Test ID#98-2194 C. <u>dubia</u> 7-Day Survival
Test Dates: 28 May 1998 - 4 June 1998

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GZA - Central Landfill Test ID#98-2194 C. <u>dubia</u> 7-Day Survival
Test Dates: 28 May 1998 - 4 June 1998

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SUMMARY OF FISHER'S EXACT TESTS

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TITLE: GZA-Central Landfill Test ID#98-2194 C. dubia Repro. FILE: A:\gzacd.dat

TRANSFORM: HO TRANSFORMATION NUMBER OF GROUPS: 4

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GZA-Central Landfill Test ID#98-2194 C. dubia Repro. File: A:\gzacd.dat Transform: NO TRANSFORMATION

SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 1 of 2

GZA-Central Landfill Test ID#98-2194 C. dubia Repro. File: A:\gzacd.dat Transform: NO TRANSFORMATION

SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 2 of 2

GZA-Central Landfill Test ID#98-2194 C. dubia Repro. File: A:\gzacd.dat Transform: NO TRANSFORMATION Chi-square test for normality: actual and expected frequencies INTERVAL $\langle -1.5 \rangle$ -1.5 to $\langle -0.5 \rangle$ -0.5 to 0.5 $\langle 0.5 \rangle$ 50.5 to 1.5 $\langle 1.5 \rangle$ 9.680 15.280 EXPECTED 2.680 9.680 2.680
 3 3 9 3 OBSERVED 1 0 15 Calculated Chi-Square goodness of fit test statistic = 0.1399
Table Chi-Square value (alpha = 0.01) = 13.277 Data PASS normality test. Continue analysis. GZA-Central Landfill Test ID#98-2194 C. dubia Repro. File: A:\gzacd.dat Transform: NO TRANSFORMATION Shapiro Wilk's test for normality ~ 10 $D = 2491.500$ $W = 0.971$ Critical W (P = 0.05) (n = 40) = 0.940 Critical W (P = 0.01) (n = 40) = 0.919 Data PASS normality test at P=0.01 level. Continue analysis. GZA-Central Landfill Test ID#98-2194 C. dubia Repro. File: A:\gzacd.dat Transform: NO TRANSFORMATION Bartlett's test for homogeneity of variance Calculated B1 statistic $=$ 4.94 Table Chi-square value = 11.34 (alpha = 0.01, df = 3) Table Chi-square value = 11.34 (alpha = 0.01 , df = 3)
Table Chi-square value = 7.81 (alpha = 0.05 , df = 3) Data PASS Bl homogeneity test at 0.01 level. Continue analysis.

GZA-Central Landfill Test ID#98-2194 C. dubia Repro. File: A:\gzacd.dat Transform: NO TRANSFORMATION

ANOVA TABLE

Critical F value = 2.92 $(0.05, 3, 30)$ Since F > Critical F REJECT Ho: All equal

GZA-Central Landfill Test ID#98-2194 C. dubia Repro. File: A:\gzacd.dat Transform: NO TRANSFORMATION

GZA-Central Landfill Test ID#98-2194 C. dubia Repro. File: A:\gzacd.dat Transform: NO TRANSFORMATION

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NEW ENGLAND BIOASSAY TOXICITY DATA FORM CHRONIC COVER SHEET

TEST SOLUTION VOLUME: 15 (mL) NO. ORGANISMS PER TEST CHAMBER: \mathbf{r} NO. ORGANISMS PER CONCENTRATION: 10 NO. ORGANISMS PER CONTROL: 10

START DATE: $\overbrace{11}$, 529198 AT 1340 (hours)

END DATE: $\overline{11}$, $\frac{15}{48}$, AT 1190 (hours)

Laboratory Control Water

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Results Ceriodaphnia dubia Chronic Test ۵f 95% Conf. Limit 48 Hour 7 Day LC_{sl} NOEC: LOEC:

NOEC: NO-OBSERVED-EFFECT-CONCENTRATION

LOEC: LOWEST-OBSERVED-EFFECT-CONCENTRATION

ANIMAL CONDITION/BEHAVIOR:

COMMENTS: R. Jaule REVIEWED BY:

DATE: $6/5/98$

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Date Reviewed:

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GZA - Central Landfill - Test ID#98-2201 <u>C. dubia</u> 7-Day Survival Test Dates: 29 May 1998- 5 June 1998

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GZA - Central Landfill - Test ID#98-2201 C. dubia Repro File: A:\gza2cd.dat Transform: NO TRANSFORMATION

SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 1 of 2

GZA - Central Landfill - Test ID#98-2201 C. dubia Repro File: A:\gza2cd.dat Transform: NO' TRANSFORMATION

SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 2 of 2

 $\mathcal{L}^{\text{max}}_{\text{max}}$ and $\mathcal{L}^{\text{max}}_{\text{max}}$

GZA - Central Landfill - Test ID#98-2201 C. dubia Repro
File: A:\gza2cd.dat Transform: NO TRANSFORMATION Transform: NO TRANSFORMATION Chi-square test for normality: actual and expected frequencies INTERVAL $\left(-1.5 -1.5 \text{ to } -0.5 -0.5 \text{ to } 0.5 \right)$ $\left(-9.5 +0.5 \text{ to } 1.5 -1.5 \right)$ EXPECTED 2.010 7.260 11.460 7.260 2.010 " •—T • $OBSERVED$ 3 5 13 7 2 Calculated Chi-Square goodness of fit test statistic $=$ 1.4074 Table Chi-Square value (alpha = 0.01) = 13.277 Data PASS normality test. Continue analysis. GZA - Central Landfill - Test ID#98-2201 C. dubia Repro
File: A:\gza2cd.dat Transform: NO TRANSFORMATION Transform: NO TRANSFORMATION Shapi'ro - Wilk's test for normality $D = 926.100$ $W = 0.966$ Critical W (P = 0.05) (n = 30) = 0.927 Critical W (P = 0.01) (n = 30) = 0.900 Data PASS normality test at P=0.01 level. Continue analysis. GZA - Central Landfill - Test ID#98-2201 C. dubia Repro File: A:\gza2cd.dat Transform: NO TRANSFORMATION Bartlett's test for homogeneity of variance Calculated Bl statistic = 4.48 Table Chi-square value = 9.21 (alpha = 0.01, df = 2) Table Chi-square value = 5.99 (alpha = 0.01, df = 2).
Table Chi-square value = 5.99 (alpha = 0.05, df = 2). Data PASS Bl homogeneity test at 0.01 level. Continue analysis,

 $\label{eq:2.1} \mathcal{L}(\mathcal{L}) = \mathcal{L}(\mathcal{L}) \mathcal{L}(\mathcal{L})$

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APPENDIX B

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CHRONIC TOXICITY DATA SHEETS AND STATISTICAL ANALYSES FOR AMPHIPOD, **Hvalella azteca,** TESTS WITH WHOLE SEDIMENT SAMPLES

Client: GZA-CENTRI Landful Date: $6/2/98$ Investigator: MLalumer

Test ID #: $\frac{78 - 2238}{99 - 2238}$ Job # 198-015 $\cot \frac{498.180670}{89.1812}$

Text obystem Dexign Text Chamber confirm of 1 gr wide Mouth Maxon jay with a 7/8" hole drilled at Am 800 pm Netex the 750 rul March. Fbe Screen was glued externelly to the hole With Silicone Sealan 200.00 federat was added to each of 8 text Chawbery for each fite. 500 ml of Moderately hard beconstituted preshently was gentle a doled to each chawerer to pineme the desturbance of the sediment. The Dilution water was delivered continuously To each text chamber at a late of Two ch overy/day (1000 ML MAROF daily) by the pollowing " A finge die control vilve (Second fretten <u>lature whipper # 56001)</u> Way added to the Neck each text changer and connected to the feven text chambers of each fite with a Tuberg (Aquarium grade). Each ferres of control valuely was connected to a fingle Chairmel

Client: GZA - Centularly Test ID #: 98. $239 \rightarrow 98 - 2243$ Date: $6/2/98$ Job # $198 - 015$ Investigator: ML Jalawia $\cot \frac{1}{2} \pi / 806 \rightarrow 98 - 1812$

a puritatic pump (I swater Mode 7338-20) Utivo Silicare tubino (Cole Payne, 3/32" I.) #06411-63) Moderately Hard Reconstituted freedwater (MHRO) Was delivered to each text chawber how a 30 gallon Nalgene container vea the pump of Nato a prosecurately O. 7 ml/memete - 1.0 ml/min α Each content value was calibrated to approx 2 drops Pu 3-feconds and cheached Times clack ation way functied to turel fubb lext cha by first readers a tri I al despotable piper (Valcon Brand 7506) Currenten 1" above the fee ineor fuckice. were placed in a water both Text chawler MHRCf overflow at a temperature of 23°C. fland-pipe i the bath Way adjusted to the Height becopt the conttent temperature but hever ack flow to the text chambers. ran toc 24h Lytlem Oganism ptroducep nor to

 $Client:$ Test ID #: $\angle M$ $_{\text{Date}:}$ ω -Q& $Job \# 198-0/5$ $\text{coc}\ \#$ \mathcal{N} \mathcal{A} Investigator: Lab. Sediment Tup: Hint Silica rumbers 18, 45 and 90 were washed with water Sieura tharrigh a and 500300 and 75 micrometer sieur respectivel Cach was then fransperse tocicles sucket coop puchetasas vilted todicure Ulles R.O. Water pragnumballines weremilled and Sieved Unosign a Imm Sieve. tomoginged batch georgrol sediment was mixed vaing the following quantities: actual taiget
veight (c $\circ/_{\circ}$ (orbitionals) <u>weight ig</u> 0.6 19.2 $#18$ Silica $H45Silico.$ 8.7 278.4 228.4 $\sharp \varphi \circ \Im \mathcal{C}$ 769.2 2214.9 2214.4 326.4 10.2 320.4 Montmorilloni te Silt Kadinite clay 2048 10.4 204.8

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 $Client: \begin{array}{c} \begin{array}{c} \begin{array}{c} \begin{array}{c} \end{array} \\ \end{array} \\ \begin{array}{c} \end{array} \\ \end{array} \\ \end{array} \\ \text{Test ID } \text{\#}: \begin{array}{c} \begin{array}{c} \end{array} \\ \end{array} \\ \end{array} \end{array}$ Date: $b/1$ 9 0
Job # 198-015 Investigator: $\angle K$ COC \angle tager
weight (g) Constituent $9/2$ actual (s) 4.9 poortmass <u>156.8</u> 156.8 O *<J* $\frac{1}{2}$ tell = 32009 \mathbf{t} $\ddot{}$ $\ddot{}$

 $Client:\cancel{GZA}$ Test ID #: \mathcal{N} Date: $6/2/98$ $30b + 15 - 015$ Investigator: Maldyuall 000 + 98-1806, 98-1807 Centrel Landfill Sedement Characterigition $55098-50$ NEB $20*98-1806$ Received 412 Two 1-gal plastic Jay Aldiment was composited in a Large container Jitty Black Muck uniform consistence Wild H25 odor $SED98-S1$ NER $IDH98-1807$ Recours in 2 1-gal Plastic Jay Composited i a large container Sandy Black Muss Mill H2Soda Little organic protter unfibe

 $Client: G2A$ rest ID $\#:= \mu/A$ Date: $6/2/98$ Job # $/98-015$ J_1 J_2 J_3 J_4 J_5 J_6 J_7 J_8 J_8 <u>I federment Characterization</u> Central $55h$ 98-052 $(98 - 1808)$ <u><'</u> Recent Plastic Tare $2(9d)$ Seleim consode $41r4$ Large contain to reginal Containers \prec le *4.* <u>Suty Much.</u> Mild No Sodor. Red worm Removel أناد المصرية $551^{98 - 053}$ (98-1809 1-gel platter jay of federicin were i a lazir c Conroot the ougene jay Sediment was a futto 4 Musil *^-f*———*f—f-*——~-'————————i. rr^-——*J.*—.rr.-??.———ic——

 $Cilent: \underline{GZAI}$ Test ID #: NAI Date: 6/2/98 Job # 198-015
Investigator: Matthews coc # 98-1810 Investigator: *MULLALAL* Central SED 98-054 $(98 - 1810)$ are composited Jay fidewent June \overline{a} . the macinal tte Black Much. Jedim σ of organic Matter-Holtly Kt i L bdo $\ddot{}$ \mathbf{r}

Client: GZA- Central Lindtill Test ID #: $98 - 2238 - 98 - 22$ 1.1298 $Job \# 198.015$ Date: $\cot \frac{\pi x}{2}$ Investigator: Reconstituted Freshwater (μHdef) 11-0 prepared Reverse 1901 the following HM2 bа <u>loy</u>
0.9324gm $0a\delta v$ 0994 Wrights preasured for salts: B atchit $\#$ $.619$ $22-5$ $22.5a$ 22.5 av <u>8M 61.</u>
Boxen# n.9328 a 0.9316 I 9323ă $44\overline{)}$ **A98-mHO36** $A98 - mH35$ A_8504 14.14 Q 14. I< Hard ness: 92 mg/l as CaCO3 Frond Alkalinity: 65 mil Botch # 198-MH039 $6/11$ actual weights. N_0N_0 22.59 $14.$ 0.9325 ◠ a_{D4} 14.1

Client: GZA Central Landfill Test ID #: $98 - 2238 \rightarrow 98 - 2243$ Date: $6/13/98$ Job # $18 - 015$ Investigator: Affeluxia \cot = $\sqrt{4}$ Moderately Hard Recorditated Freshwiter Preprotect $190L$ Nille a prepared descripted water al base water with the Oldring fa added: NaHCO3 22.5 Barch# A98-MHOYO KCI $\mathcal{L}^{\text{max}}_{\text{max}}$ and $\mathcal{L}^{\text{max}}_{\text{max}}$ 0.93 $C450y.2H_2O$ 14.1 <u>Mg SO4</u> 14.1 Glilologe 1906 mieuro used to prepare MHRCF <u>Lor# A98-MHC42</u> Sants ochlid' werde (ဝှ $10a$ HCO₃ 22. Vc i 0.93 $(aSDy.2Hd)$ $|4.1$ MESO4 14.1 $\ddot{}$

HYALLELA AZTECA FEEDING JCHEDYLE

Client; (V? H____________ Test ID ft: **?g-2.?ag--> yff-?:> y** $Date:$ 0.498 Job $\frac{4}{11.98-015}$ The strategiven of the strategies of the s α . The following: uvere potatupain, when checked: $ConvOLH, q, F, e, D, C$ -50 H, 97 F $-5051H$ ぴ $-52 + 4$ -54 , H, g All valuis mus adjustichts actues of 13 sec \ddotsc \overline{a}

De : data intry eaur

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NEW ENGLAND BIOASSAY, INC. AMMONIA CALIBRATION DATA SHEET

Calibration Date: $10.24.98$

 $Technician:$ $\left| \left(\zeta \right) \right|$

Slope Check

Calibration Matrix | ISA NH3 NH3 Calc *,* t r Buffer Std Vol Conc. mV $\texttt{CAL.} \parallel \texttt{ mL } \parallel \texttt{ mL } \parallel \texttt{ ppm } \parallel \texttt{ mL } \parallel \texttt{ ppm }$ λ A | 100 | 2 | 121 | 0.10 | 0.12 hS'M.U $\overline{}$ $\begin{array}{|c|c|c|c|c|c|c|c|c|} \hline \text{B} & & \text{--} & & \text{-} & \text{121} & \text{1.00} & \text{1.32} \ \hline \end{array}$ ্ κ 55.4 $|U|$ $C \begin{array}{|c|c|c|c|c|c|c|c|} \hline \begin{array}{|c|c|c|c|c|c|c|c|} \hline \end{array} & - & - & 1210 & 1.00 & 13.2 \ \hline \end{array}$ ገ $O.$ ገ Salinity : Slope (mv/decade) - $\begin{bmatrix} 0 & \sqrt{2} \\ \sqrt{2} & \sqrt{2} \end{bmatrix}$

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Notes: <u>1 leus pumiDrani inStallen</u> $\ \lim_{\alpha \to 0}$ Balution Cl δ \bar{z} \overline{a}

NEW ENGLAND BIOASSAY, INC. AMMONIA DATA RECORD SHEET

Analytical Results

Technician:

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Notes: Samiple Valume = 50ml + 1 ml 19A. $pH\leq 2$

NEW ENGLAND BIOASSAY CHRONIC DATA SHEET Hyalella azteca Initial Length/Weight Data

Project No/28-015

 $\mathcal{L}_{\mathcal{A}}$

Length Measurement: Malaulu Date: 6/5798

NEW ENGLAND BIOASSAY TOXICITY DATA FORM CHRONIC SEDIMENT TOXICITY COVER SHEET

CLIENT:

CZA - Central Landfill

ADDRESS:

H. AZLECA TEST NO: $98 - 2238$ -PROJECT NO: 198-015 SAMPLE TYPE: WHOLE SEDIMENT

CONTACT:

V.

Mr. Tim Briggs

INVERTEBRATES:

 $\overline{1}$

PRESET BY: (TECH INIT.): MAY /KS TEST LOADED BY (TECH. INIT.) MAY XS TEST SPECIES: Hyalella^l azteca SOURCE\NEB LOT 8. HA-AB 28(6/2) TEST CONDITIONS

SEDIMENT WT: 200 + .3 q TEST SOLUTION VOLUME: 500 NO. ORGANISMS PER TEST CHAMBER: 10 NO. ORGANISMS PER CONCENTRATION: 80 NO. ORGANISMS PER CONTROL: 80

DILUTION WATER SOURCE:

Results of Hyalella azteca Chronic Test

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CHRONIC SEDIMENT DATA SHEET

Project No: 198-015

 $Date: _0.18.9$ Test Species: Hyalella azteca \mathcal{O} Total Wt: 85557 $\frac{3}{(s_4-r-r_1)}$ pate: Client: GZA - Central Landfill Tech: Tare Wt:

08582 110076 1.08314 0.00803 1.10370 **D-COORY** 7587 0.873 2.73 $\widetilde{\mathcal{C}}$ Adia \mathbf{r} \Diamond 1.08708 21313 1.271084 porcosal pesco-a OBSCXOU 0.899 chuo' 1.08388 0.320 2.69 Cv. s $\widetilde{}$ O σ Time Out 0820 1.09246 1.09654 1.09920 110172 0.00252 **OUSIT** 0.389 0.252 1.114.71 3.89 2.52 \mathbf{L} $\frac{1}{2}$ 0.00103 LO9009 110887 تيا 0.00358 LOGISCI 11185 **2.25X** 1.03 3.58 2378
10.11 t. \overline{G} \overline{J} Replicate 0.00189 -0.335 -05375 0.315 **PECC.0** 0.301 \overline{f} $26₁$ Ω ∞ $\overline{9}$ $\hat{\zeta}$ μ [1] ११ **CONCOL** 109571 0.0084 10.001 $1/1095$ 110716 $\frac{1}{1}$ $0.\overline{212}$ J
G Time In 17.80 \sum \sum \overline{O} \overline{q} $\ddot{\circ}$ $\hat{\sigma}$ **Heropin** 10.00333 0.369 **SLOTT** 97521 3.32 1.10951 $\frac{1}{2}$ 1.24 7775077 \hat{O} \mathbf{m} σ \overline{C} <u>Crecoro</u> **GEOOLO** 88101 $.08143$ C₁₈₃₄ 1034 0.213 $2\sqrt{2}$ 251 0.314 $\overline{}$ \Diamond $\tilde{\mathbf{r}}$ g Organism Weight (g) Organism Weight (g) Test ID#: $\frac{96.233}{16.2343}$ Mean Weight (mg) $\widehat{\mathbf{e}}$ Total Weight (g) $(g \tto \tmg)$ Mean Weight (mg) Conv. (g to mg) \widehat{g} $\widehat{\mathcal{Q}}$ No. Surviving Oven Temp (°C)_ No. Surviving Reviewed By: Total Weight Sample ID Tare Weight Tare Weight -53 Conv. さい

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GZA Central Landfill H. azteca Survival File: GZAHaSur.dat Transform: NO TRANSFORM

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SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 1 of 2

GZA Central Landfill H. azteca Survival
File: GZAHaSur.dat Transform: NO 7 File: GZAHaSur.dat Transform: NO TRANSFORM

SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 2 of 2

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TITLE: GZA Central Landfill H. azteca Survival

FILE: GZAHaSur.da2

TRANSFORM: NO TRANSFORM NUMBER OF GROUPS: 5

GZA Central Landfill H. azteca Survival File: GZAHaSur.da2 Transform: NO TRANSFORM

SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 1 of 2

 \sim GZA Central Landfill H. azteca Survival
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SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 2 of 2

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GZA Central Landfill H. **azteca** Survival Transform: ARC SINE(SQUARE ROOT(Y)) File: GZAHaSur.da2 Transform: ARC SINE(SQUARE ROOT(Y))

SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 1 of 2

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GZA Central Landfill H. azteca Survival File: GZAHaSur.da2 Transform: ARC SINE(SQUARE ROOT(Y))

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SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 2 of 2

GZA Central Landfill H. azteca Survival
File: GZAHaSur.da2 Transform: ARC 3 File: GZAHaSur.da2 Transform: ARC SINE(SQUARE ROOT(Y)) Chi-square test for normality: actual and expected frequencies ------------------INTERVAL \leq -1.5 -1.5 to <-0.5 -0.5 to 0.5 >0.5 to 1.5 >1.5 9.680 15.280 EXPECTED 2.680 9.680 2. 680 OBSERVED 4 9 11 16 Ω -------------*---*-Calculated Chi-Square goodness of fit test statistic = 8.7030
Table Chi-Square value (alpha = 0.01) = 13.277 Data PASS normality test. Continue analysis. GZA Central Landfill H. azteca Survival
File: GZAHaSur.da2 Transform: ARC 3 Transform: ARC SINE(SQUARE ROOT(Y)) Shapi'ro Wilk's test for normality $D = 1.165$ $W = 0.944$ $= 0.940$ Critical W ($P = 0.0$ $5)$ $(n = 40)$ $\texttt{Critical W (P = 0.01)}$ $1)$ (n = 40) $= 0.91$ Data PASS normality test at P=0.01 level. Continue analysis. GZA Central Landfill H. azteca Survival File: GZAHaSur.da2 Transform: ARC SINE(SQUARE ROOT(Y)) Bartlett's test for homogeneity of variance Calculated Bl statistic = 8.11 Table Chi-square value = 13.28 (alpha = 0.01, df = 4) Table Chi-square value = 9.49 (alpha = 0.05, df = 4) Data PASS Bl homogeneity test at 0.01 level. Continue analysis.

GZA Central Landfill H. azteca Survival
File: GZAHaSur.da2 Transform: ARC **File:** GZAHaSur.da2 Transform: ARC SINE(SQUARE ROOT(Y))

ANOVA TABLE SOURCE DF DF SS MS F Between 4 0.115 0.029 0.860 Within (Error) 35 1.165 0.033 Total 39 1.280 Critical F value = 2.69 (0.05,4,30) Since F < Critical F FAIL TO REJECT • Ho: All equal GZA Central Landfill H. azteca Survival File: GZAHaSur.da2 Transform: ARC SINE(SQUARE ROOT(Y)) \mathbf{r} TUKEY method of multiple comparisons

TITLE: GZA Central Landfill

Growth

FILE: GZAHaGro.dat TRANSFORM: NO TRANSFORM

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NUMBER OF GROUPS: 6

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GZA Central Landfill File: GZAHaGro.dat H. azteca Growth Transform: NO TRANSFORM

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SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 1 of 2

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GZA Central Landfill H. azteca Growth
File: GZAHaGro.dat Transform: NO File: GZAHaGro.dat Transform: NO TRANSFORM

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SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 2 of 2

TRANSFORM: NO TRANSFORM NUMBER OF GROUPS: 5

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GZA Central Landfill File: GZAHaGro.dat H. azteca Growth Transform: NO TRANSFORM

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SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 1 of 2

GZA Central Landfill H. <u>azteca</u> Growth File: GZAHaGro.dat Transform: NO TRANSFORM

SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 2 of 2

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GZA Central Landfill H. azteca Growth File: GZAHaGro.da2 Transform: NO TRANSFORM

SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 1 of 2

 \sim GZA Central Landfill H. azteca Growth
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SUMMARY STATISTICS ON TRANSFORMED DATA TABLE 2 of 2

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Data PASS Bl homogeneity test at 0.01 level. Continue analysis.

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 $\text{Critical F value} = 2.69 (0.05, 4, 30)$ Since F < Critical F FAIL TO REJECT .Ho: All equal

GZA Central Landfill H. azteca Growth File: GZAHaGro.da2 Transform: NO TRANSFORMATION

APPENDIX C

ACUTE TOXICITY DATA SHEETS AND STATISTICAL ANALYSES FOR REFERENCE TOXICANT TESTS WITH AMPHIPODS,H. **azteca.** (POTASSIUM CHLORIDE) AND DAPHNIDS, **C. dubia** (SODIUM CHLORIDE)

NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA FORM

COVER SHEET FOR REFERENCE TOXICANT (NaCL) LC50 TESTS

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DOCUMENTATION FORM

KIFT3 Test ID $\#:\frac{1}{2206}-7207,-2208$ $Client:$ Date: $6/198$ Job # 190-084 iMotrin Investigator: \cos # NA Preparation of 10091 L Nacl Stock Solution 50.00 Nare weighed by Mettler top loading talance asked to Jul Volumetric flank partially filled Canosis usate Drenased Kawan Udesl. strones *Nolume* unth Milli-Q and agitated unt ሥ <u>Crystals disolved. The Volumetric was again fill.</u> time as the volume decreased as the about <u>Nisolwed</u>. 2 of # Na(1) - 010 - (6/1 myation $\frac{1}{2}$.

CT-TOX: BINOMIAL, MOVING AVERAGE, PROBIT, AND SPEARMAN METHODS

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NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA FORM COVER SHEET FOR REFERENCE TOXICANT TESTS

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CT-TOX: BINOMIAL, MOVING AVERAGE, PROSIT, AND SPEARMAN METHODS

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APPENDIX H

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MAY 1993 ACUTE TOXICITY TEST REPORT

NEW ENGLAND BIOASSAY, INC.

25 May 1993

Mr. Tim Briggs GZA Environmental 320 Needham Street Newton Upper Falls, MA 02164

Dear Mr. Briggs:

ACUTE TOXICITY TEST REPORT TO GZA ENVIRONMENTAL INC. FOR SEDIMENT SAMPLES COLLECTED ON 12 MAY 1993 FROM A FRESHWATER SITE IN THE VICINITY OF THE CENTRAL LANDFILL IN JOHNSTON, RI

This report contains the results of six static-acute elutriate toxicity tests performed with the daphnid, Ceriodaphnia dubia, and the fathead minnow, Pimephales promelas. The definitive acute toxicity tests were conducted with elutriates (waterextractable phase) prepared from three sediment samples collected on 12 May 1993 by GZA Environmental staff from a freshwater site
in the vicinity of the Central Landfill in Johnston, RI. This in the vicinity of the Central Landfill in Johnston, RI. report details procedures for preparing elutriates from the sediment samples and the biological and chemical evaluations associated with performance of the acute toxicity tests with the three elutriate samples.

Sample Collection and Elutriate Preparation

On 12 May 1993, sediment samples were collected from three locations at the freshwater site by GZA staff. Sediment samples were picked up by a New England Bioassay (NEB) courier at 1520 h on 12 May 1993. Upon receipt at NEB, the sediment samples were logged into the laboratory (Table 1; Appendix A) and then stored in the dark in a cold room (4° ± 2°C); elutriate preparation was initiated on the next day. On the morning of 13 May 1993, the three sediment samples from each of the three sites were removed from the cold room and combined in individual, clean 3-L beakers; any large stones, sticks, or extraneous plant material were removed.

77 BATSON DRIVE / MANCHESTER. CONN.06040 / TEL. (203) 643 9560 / FAX. (203) 646-7169

Mr. Tim Briggs GZA Environmental 25 May 1993

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TABLE 1. DESCRIPTION OF SEDIMENT SAMPLES COLLECTED BY GZA ENVIRONMENTAL FROM A FRESHWATER SITE IN JOHNS-TON, RI FOR STATIC-ACUTE ELUTRIATE TOXICITX TESTS

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Mr. Tim Briggs GZA Environmental 3 3 25 May 1993

In addition to testing the three sediment samples from the Johnston, RI site, an additional reference sediment was evaluated as an quality-control check to determine the adequacy of the test
system and the health of the test organisms. The reference system and the health of the test organisms. sediment sample was collected by NEB on 10 May 1993 from an oligotrophic lake (Mashapaug Pond) located in Union, CT. Mashapaug Pond sediment was chosen for the reference sediment because the pond is a relatively pristine lentic system with low levels of human activity/pollution. Previous chemical characterizations performed by the Connecticut Department of Environmental Protection (CTDEP) found that Mashapaug Pond sediments were low in organic contaminants and metals. reference sediment was handled similarly to the site sediments.

Samples were manually stirred to ensure a homogeneous sediment sample. Homogenized sediment and laboratory-prepared water (hardness: 50 mg/L as CaCO₃) were combined in a 1:4 ratio (680 mL sediment: 2720 mL water; volume:volume) in 1-gallon glass widemouth specimen jars (with teflon lid inserts). For each site, two 1-gallon specimen jars were prepared in this manner.

Sediment:water mixtures were placed in foam inserts in a 55-
gallon polypropylene drum. The drum containing four glass jars (2 sediment samples) was placed on an electric drum rotator set at 12 rpm, which mixed the samples at room temperature (about 22°C) for 30 minutes in an end-over-end manner. The drum rotation was then repeated for the remaining two sediment samples.

After stirring was terminated, samples were allowed to settle for about 1 h. After settling, the supernatant (elutriate) from each jar was removed by carefully siphoning off the supernatant without disturbing the settled material. For each sediment, the supernatants collected from the two duplicate jars were combined into one sample. The supernatants were gray in color and high in suspended fine clay and silt particles. To obtain an estimate of the amount of suspended material present in the samples, aliquots of the supernatants were dried in an oven at about 100°C and weighed (Table 2); the amount of dry material in the supernatants (before filtering) averaged 14.5, 32.5, and 16.9 mg dry material/mL of liquid. Because of the high turbidity in the supernatants caused by the suspended clay and silt particles, the supernatants were filtered through a 1μ m Gelman glass-fiber filter to reduce the amount of suspended materials before use in testing.

Because the 1 *}im* filtration did not completely remove the fine silt/clay particles, an aliquot of each elutriate sample was centrifuged for 15 minutes at about 3700 revolutions per minute; the undiluted centrifuged elutriate was evaluated for acute toxicity to <u>C</u>. <u>dubia</u> during 13-15 May 1993 (Table 3). In this manner, the effects of the suspended materials on daphnid survival could be assessed.

TABLE 2. WET CHEMISTRY RESULTS FOR SEDIMENT ELUTRIATE SAMPLES

 \bullet Sample color and dry weight measurements were obtained on samples of the supernatants before filtering.

Mr. Tim Briggs GZA Environmental 25 May 1993

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TABLE 3. TEST DATES AND TEST IDENTIFICATION NUMBERS **FOR** ACUTE TOXICITT TESTS PERFORMED WITH SEDIMENT ELUTRIATE SAMPLES

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Mr. Tim Briggs .
GZA Environmental GZA Environmental 6 25 May 1993

After centrifugation, standard wet chemistry analyses [pH, dissolved oxygen, specific conductivity, hardness, and alkalinity] were performed on each sediment elutriate sample. Wet chemistry data are provided in Table 2.

The filtered elutriate samples were used in toxicity tests. Each sediment elutriate sample was serially diluted with clean laboratory-prepared water and evaluated for acute toxicity to Ceriodaphnia dubia and fathead minnows during 14-16 and 14-18 May 1993, respectively (Table 3). For the reference sediment elutriate, only the undiluted elutriate sample was evaluated for acute toxicity. Laboratory water controls were also be set up concurrently with each test to document health of test organisms. Test suspensions were not changed during the acute tests.

The basic references for the initial manipulations of the sediments for toxicity testing are the ASTM document titled "Standard Guide for Collection, Storage, Characterization, and Manipulation of Sediments for Toxicological Testing" and the National Fisheries Contaminant Research Center's Standard Operating Procedures for evaluating contaminated freshwater sediments. Additional guidance was provided by adapting the saltwater test procedures described in "Evaluation of Dredged Material Proposed for Ocean Disposal" (EPA-500/8-91/001; February 1991) and "Guidance for Performing Tests on Dredged Material to be Disposed of in Open Waters" (EPA Region I; 15 May 1989).

Test Water

The dilution/control water for the C . dubia and fathead minnow elutriate toxicity tests was dilute mineral water (DMW; nominal hardness: 50 mg/ \tilde{L} as CaCO₃). The DMW was prepared based on instructions cited in Weber et al. (1989). Base water used in preparing the DMW was deionized water from a Millipore Milli-Q® water system. To prepare the DMW, Perrier® water was added in the appropriate amount to a carboy containing deionized water and mixed. After preparation, each batch of DMW was aerated at room temperature and then used in testing. Hardness and alkalinity of DMW used as the dilution/control water for acute elutriate tests averaged 50 mg/L (as CaCO₃) and 36 mg/L (as CaCO₃), respectively.

Test Organisms

Ceriodaphnia dubia

Original stock cultures of the freshwater crustacean water flea, Ceriodaphnia dubia, used in acute elutriate toxicity tests were obtained from the CTDEP and were cultured at NEB in dilute mineral water (DMW; 12% Perrier* water) in a controlled environment chamber at the specified conditions (temperature 25° ± 2°C; photoperiod 16-h light and 8-h dark).

Mr. Tim Briggs GZA Environmental 7 7 25 May 1993

C. dubia were individually cultured in 30-mL plastic cups (1 C. dubia per cup) containing 15 mL of DMW. Each culture chamber received 50 /JL of a yeast/trout chow/Cerophyl® (YTC) food suspension (see Weber et al., 1989, for procedures for preparing the food suspension) and 150 μ L of the green alga, Selenastrum Ī capricornutum, when the cultures were changed. Survival and Ξ reproduction of culture animals were checked each time the culture water was changed (on a daily basis after production of a first brood of young). After 14 days, cultures were not used for
testing. All young were removed from culture chambers 24 h All young were removed from culture chambers 24 h before the start of a test to ensure that only ceriodaphnids \leq 24 h old would be available to start the acute tests.

Fathead Minnows (Pimephales promelas)

Immature fathead minnows (Pimephales promelas) used in the acute elutriate toxicity tests were obtained from NEB in-house cultures. The original sources of NEB brood stocks of fathead minnows were the EPA Environmental Monitoring Systems Laboratory (Newtown, OH) and Aquatic Biosystems (Fort Collins, CO). Young fathead minnows (9-days old at test initiation) were used to initiate the acute tests.

ACUTE TOXICITY TESTS

Test Systems

C. dubia Static-Acute Elutriate Toxicity Tests

Specific procedures of the C . dubia 48-h static-acute test system are described in Appendix B. These procedures are based on EPA guidelines (Weber et al., 1989; Peltier and Weber, 1985). Static-acute toxicity tests using C . dubia were initiated with each sediment elutriate. Young C . dubia (\leq 24 h old at test initiation) were continuously exposed for 48 h under static conditions to five concentrations of each sediment elutriate sample (6.25, 12.5, 25, 50, and 100% elutriate), a dilution-water control, and a procedural blank control. Mashapaug Pond sediment mixed with DMW was used as the procedural blank.

C. dubia were exposed in groups of five animals in 50-mL polypropylene beakers containing 30 mL of test solution or control water. Six replicates were used for each test concentration and control (30 animals per concentration). Test chambers were maintained under the specified test conditions (mean temperature 25° ± 1°C and individual temperature observations 25° ± 2°C; photoperiod 16-h light and 8-h dark). Per acute test protocols, organisms were not fed during the 48-h test.

Mr. Tim Briggs
GZA Environmental GZA Environmental 8 25 May 1993

Temperature, dissolved oxygen, pH, and specific conductivity were measured at test initiation in a composite sample from each test concentration and the controls before distribution to the test chambers. Temperature, dissolved oxygen, and pH were measured in one replicate at each test concentration at test completion. Hardness and alkalinity of 100% elutriate were measured at sample preparation. Hardness and alkalinity of DMW were measured at $\ddot{}$ time of preparation. Observations on the number of live and dead animals were made at 24 h and 48 h.

Fathead Minnow Static-Acute Elutriate Toxicitv Tests

Specific procedures of the fathead minnow 48-h static-acute toxicity test system are described in Appendix B and are based on EPA guidelines (Peltier and Weber, 1985; EPA Region I biomonitoring protocols, 1990). Static-acute toxicity tests using fathead minnows were conducted with each sediment
elutriate. Young fathead minnows (9-days old at test ip elutriate. Young fathead minnows (9-days old at test initiation) were continuously exposed for 96 h under static conditions to five concentrations of each elutriate sample (6.25, 12.5, 25, 50, and 100% sediment elutriate), a dilution water control, and procedural blank control. DMW was used as test dilution/control water. Mashapaug Pond sediment mixed with DMW was used as the procedural blank.

Fathead minnows were exposed in groups of 10 animals in 1000-mL Tri-pour polypropylene beakers containing 700 mL of test solution or control water. Three replicates were used for each test concentration and control (30 animals per concentration). Test beakers were maintained under the specified test conditions (mean temperature 25° ± 1°C and individual temperature observations 25° ± 2°C; photoperiod 16-h light and 8-h dark). Per acute testing protocols, organisms were not fed during the 96-h tests.

Temperature, dissolved oxygen, pH, and specific conductivity were measured in a single composite sample from each test concentration and the control before distribution to the test chambers. Temperature and pH were measured daily in one replicate at each test concentration; dissolved oxygen was measured daily in each test chamber. Hardness and alkalinity were measured on the 100% elutriate after sample preparation. Hardness and alkalinity of DMW were measured at the time of preparation. Observations on the number of live and dead animals were made daily until test completion (96 h).

Mr. Tim Briggs GZA Environmental 9

25 May 1993

Statistical Analysis

Data from the acute elutriate toxicity tests with C. dubia and fathead minnows were used to estimate daily median lethal concentrations (LC₅₀) and acute no-observed-effects concentration (A-NOEC). The LC₅₀ is the elutriate concentration that is lethal The LC₅₀ is the elutriate concentration that is lethal to 50% of the organisms within the test period. Estimates of daily LC_{50} values were obtained by using a computer program provided by the CTDEP. This program estimates an $\overline{{\rm LC}}_{50}$ by using one of four methods: binomial, moving-average, probit, or trimmed Spearman-Karber.

The method selected is based on the shape of the concentration -
effects curve and the number of concentrations with partial mortalities (mortality greater than 0% but less than 100%). The moving-average, probit, and trimmed Spearman-Karber methods both estimate the LC_{50} with 95% confidence limits. The bounds placed on the LC_{50} by using the binomial test are not 95% confidence limits, but can be used as statistically sound conservative bounds that are always above 95% when animal sample size per concentration is large $(N \ge 6)$ (Stephan, 1977). Sample size in all acute toxicity tests conducted for this study was 30 animals per concentration.

To determine the A-NOEC, C. dubia and fathead minnow survival data were analyzed by using Fisher's exact test comparing survival of organisms in the test concentrations with survival in the laboratory-water control.

RESULTS

Results of the acute toxicity tests with the three sediment elutriates are summarized in Tables 4 and 5. The filtered elutriates (1 *fm.* filtered samples) prepared from sediments collected from Site Nos. 21, 24, and 30 exhibited significant acute toxicity to <u>C. dubia</u>. Only the sediment elutriate from the Site No. 30 sample exhibited significant acute toxicity to fathead minnows (Table 4). Copies of the raw toxicity data sheets and the statistical analyses printouts are located in Appendix C.

Ceriodaphnia dubia

Survival of C . dubia at test completion (48 h) in the undiluted filtered elutriate samples was 7, 33, and 70% for sediments collected from Site Nos. 21, 24, and 30, respectively (Table 5). The 48-h LC₅₀ values for C. dubia were 40.4, 58.9, and $> 100\$ elutriate for sediments collected from Site Nos. 21, 24, and 30, respectively (Table 4).

Mr. Tim Briggs GZA Environmental 10 10 25 May 1993

TABLE 4. SUMMARY OF ACUTE TOXICITY TEST RESULTS

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Mr. Tim Briggs GZA Environmental 11 25 May 1993

TABLE 5. RESULTS OF ACUTE TOXICITY TESTS WITH ELUTRIATES PREPARED **FROM SEDIMENT SAMPLES COLLECTED BT GZA ENVIRONMENTAL OK 12 MAX 1993 FROM A FRESHWATER SITE IN THE VICINITI OF THE CENTRAL LANDFILL, IN JOHNSTON, RI**

Mr. Tim Briggs GZA Environmental 12 25 May 1993

TABLE 5. RESULTS OF ACUTE TOXICITt TESTS WITH ELUTRIATES PREPARED FROM SEDIMENT SAMPLES COLLECTED BX GZA ENVIRONMENTAL OH 12 MAT 1993 FROM A FRESHWATER SITE IN THE VICIHITX OF THE CENTRAL LANDFILL IN JOHNSTON, RI

(CONTINUED)

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TABLE 5. RESULTS OF ACUTE TOXICITI TESTS WITH ELUTRIATES PREPARED FROM SEDIMENT SAMPLES COLLECTED BT GZA ENVIRONMENTAL ON 12 MAT 1993 FROM A FRESHWATER SITE IH THE VICINITT OF THE CENTRAL LANDFILL IN JOHNSTON, RI

(CONTINUED)

 $(1\mu$ filtered) 7.4-7.7 6.0-6.8 24.2-24.5 100% (Centrifuged) 100 100 —— —— 7.8 7.0-8.0 3.6-8.1 24.6-25.9 7.3 25.6

Fathead Minnows

Mr. Tim Briggs
GZA Environmental GZA Environmental 14 25 May 1993

For Site No. 21, C . $dubia$ survival in the 6.25, 12.5, 25, and 50% elutriate was 93, 90, 77, and 43%, respectively; the A-NOEC for Site No. 21 sediment elutriate was 12.5% elutriate.

For Site No. 24, C . dubia survival in the 6.25, 12.5, 25, and 50% elutriate was 83, 83, 70, and 57%, respectively; the A-NOEC for Site No. 24 sediment elutriate was also 12.5% elutriate.

For Site No. 30, C . dubia survival in the 6.25, 12.5, 25, and 50% elutriate was 97, 73, 70, and 67%, respectively; the A-NOEC for Site No. 30 sediment elutriate was 6.25% elutriate.

All three filtered elutriates were brown in color and exhibited some turbidity (i.e., cloudy suspensions). Because C. dubia are filter feeders and are sensitive to suspended particulates in the water column, aliquots of the three sediment elutriates were centrifuged and then evaluated for acute toxicity. Survival of C. dubia in the undiluted centrifuged elutriate samples was 83, 40, and 100% for sediments collected from Site Nos. 21, 24, and 30, and 100% for sediments collected from Site Nos. 21, 24, and 30, respectively (Table 5). Thus, centrifuged elutriates prepared from sediments collected from Site Nos. 21 and 30 were less toxic than the filtered elutriates; the centrifuged elutriate from the Site No. 24 sediment was similar in toxicity to the filtered elutriate (40% and 33% survival for the centrifuged and filtered elutriates, respectively).

 C . dubia survival in the laboratory-water controls was \geq 97% for the elutriate toxicity tests. Survival in the 1 μ m filtered elutriate and the centrifuged elutriate from the reference sediment (Mashapaug Pond) was 77% and 97%, respectively (Table 6). Lower survival in the filtered elutriate may have been due to suspended particulates in the sample; centrifugation removed the particulates and eliminated the acute toxicity.

Fathead Minnows

Fathead minnow survival at test completion (96 h) in the undiluted filtered elutriate samples was $97, 93$, and 53 % for sediments collected from Site Nos. 21, 24, and 30, respectively (Table 5). collected from Site Nos. 21, 24, and 30, respectively (Table 5).
The 96-h LC_{so} values for fathead minnows were all > 100% elutriate (Table 4). Ĩ,

For Site No. 21, fathead minnow survival after 96 h in the 6.25, 12.5, 25, and 50% sediment elutriate was 97%; the A-NOEC for Site No. 21 sediment elutriate was 100% elutriate.

For Site No. 24, fathead minnow survival in the 6.25, 12.5, 25, and 50% sediment elutriate was \geq 93%; the A-NOEC for Site No. 24 sediment elutriate was also 100% elutriate.

Mr. Tim Briggs GZA Environmental 15 25 May 1993

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TABLE **6.** RESULTS OF ACUTE SCREENING TOXICITY TESTS WITH AN ELUTRIATE **PREPARED FROM A REFERENCE SEDIMENT SAMPLE COLLECTED ON 10 MAT 1993 FROM MASHAPAUG POND IN UNION, CT**

* Laboratory-water control using C. dubia was evaluated concurrently
with the 100% screening tests using the centrifuged elutriate samples (Test Dates: 13-15 May 1993).

Mr. Tim Briggs GZA Environmental 16 16 25 May 1993

For Site No. 30, fathead minnow survival in the 6.25, 12.5, 25,
and 50% elutriate was ≥ 97%; survival in the 100% sediment elutriate was only 53% at test completion. The A-NOEC for Site No. 30 sediment elutriate was 50% elutriate.

Although all three filtered elutriates were brown in color and exhibited some turbidity, the turbidity did not appear to affect the fathead minnows.

Fathead minnow survival in the laboratory-water controls was \geq 93% for the elutriate toxicity tests. Survival in the 1 μ m filtered elutriate from the reference sediment (Mashapaug Pond) was 97% (Table 6).

Summary

The elutriate test results for the sediment samples collected by GZA Environmental on 12 May 1993 can be summarized as follows:

- Site No. 21: Significant acute toxicity of filtered elutriate to **c.** dubia (48-h $LC_{50} = 40.48$ elutriate; A-NOEC = 12.5% elutriate) with 7% survival in the undiluted elutriate at test completion (survival in the centrifuged elutriate was increased to 83%). No significant acute toxicity to fathead minnows (96-h LC₅₀ > 100% elutriate; A-NOEC = 100% elutriate) with 97% survival in undiluted filtered elutriate at test completion.
- Site Ho. 24: Significant acute toxicity of filtered elutriate to $C.$ dubia (48-h LC₅₀ = 58.9% elutriate; A-NOEC = 12.5% elutriate) with 33% survival at test completion (survival elutriate) with 53% Survival at test completion (Survival
in the centrifuged elutriate was increased to only 40%). In the centrifuged clutrate was increased to only voe,
No significant acute toxicity to fathead minnows (96-h LC_{50} > 100% elutriate; A-NOEC = 100% elutriate) with 93% survival in undiluted filtered elutriate at test completion.
- Site Ho. 30: Significant acute toxicity of filtered elutriate to significant acute toxicity of fiftered endflate to
C. dubia (48-h LC₅₀ > 100% elutriate; A-NOEC = 6.25% e. **aubia** (40-n LC₅₀ > 100% eiutriate; A-NOEC = 0.25%
elutriate) with 70% survival at test completion (survival in the centrifuged elutriate was increased to 100%).
Significant acute toxicity to fathead minnows (96-h LC₅₀) > 100 % elutriate; A-NOEC = 50% elutriate) with 53% survival in undiluted filtered elutriate at test completion.

If you have any questions concerning the elutriate toxicity test results, please contact me at (203) 643-9560.

Sincerely,

amez

ohn D. Cooney, Ph. Laboratory Director

Mr. Tim Briggs GZA Environmental 17 17 25 May 1993

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Weber, C.I., W.H. Peltier, T.J. Norberg-King, W.B. Horning, F.A. Kessler, J.R. Menkedick, T.W. Neiheisel, P.A. Lewis, D.J. Klemm, Kessler, J.R. Menkedick, T.W. Neiheisel, P.A. Lewis, D.J. Klemm, Q.H. Pickering, E.L. Robinson, J.M. Lazorchak, L.J. Wymer, and R.
W. Freyberg. 1989. Methods for estimating the chronic toxicity Q.H. Pickering, E.L. Robinson, J.M. Lazorchak, L.J. Wymer, and R. of effluents and receiving waters to freshwater organisms. EPA-
600/4-89-001. Environmental Protection Agency, Environmental Monitoring Systems Laboratory, Cincinnati, OH. 249 pp.

APPEKDIX A

CHAIN OF CUSTODY FORMS

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NEW ENGLAND BIOASSAY, INC.
77 BATSON DRIVE
MANCHESTER, CT 06040
(203) 643-9560

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TIMË TIME: B = BACTERIA BOTTLE パク・クノイ N_{ab} 6.8/11.10600 -PROJECT NUMBER: $(62A)$ C ACID H = HYDROCHLORIC ACID (HCL)
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X = OTHER/SPECIFY **TRANSFERS
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S = SODIUM HYDROXIDE (NaOH) T = SOD TYPE | $\overline{\mu}$ **RO = RUNOFF
L = LAKE/OCEAN** \overline{a} TIME 1320 TIME \dot{q} CONTAINER TYPE: P.= PLASTIC E = EPA VIAL C = CUBE
PRESERVATION CODE: I = ICED
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T = TREATMENT FACILITY NEW ENGLAND BIOASSAY, INC. NEB <u>ENVERONMENTAL</u> 77 BATSON DRIVE
MANCHESTER, CT 06040
(203) 643-9560 SOURCE
CODE $\boldsymbol{\varphi}$ β ADDITIONAL COMMENTS: SAMPLER'S SIGNATURE. SERVING Manarauc Macila PMG SEDINING METHOD OF SHIPMENT: SAMPLE
ID. $\frac{d\mathbf{y}}{d\mathbf{x}}$ SOURCE CODE:
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LF = LANDFILL PROJECT NAME: $62A$ 93-18231 $23 - 1822c$ NEB
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MISCELLANEOUS DOCUMENTATION FORM

APPENDIX B

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METHODS FOR ACUTE TOXICITY TESTING WITH FRESHWATER ORGANISMS

METHODS FOR ACUTE TOXICITY TESTING WITH FRESHWATER ORGANISMS

Unless otherwise noted, all test protocols follow those of the U.S.
Environmental Protection Agency (USEPA) as specified in <u>Methods for</u> Measuring the Acute Toxicitv of Effluents to Freshwater and Marine neasuring the Acute Toxicity of Biridents to rieshwater and Marine
<u>Organisms</u>, 3rd ed. (1985, W.H. Peltier and C.I. Weber, eds.,
EPA/600/4-85/013) and the 1 July 1990 Biomonitoring Protocols of the USEPA, Region *I.*

Sample Collection and Handling

Grab or composite samples of receiving water and effluent are collected by personnel at the client's facility, refrigerated at 4°C until pickup, and then transported to the appropriate laboratory for toxicological and chemical analysis. If the effluent contains detectable residual chlorine $(> 0.10 \text{ mg/L})$, neutralization is carried out by addition of 10 mg sodium thiosulfate per liter of effluent.

Toxicitv Testing

TEST SPECIES:

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The test organisms are the daphnids, Daphnia pulex and Ceriodaphnia dubia, and the fathead minnow, <u>Pimephales promelas</u>. All species are reared at New England Bioassay from parental stocks originally obtained from the Connecticut DEP or USEPA. The ages of the daphnids and <u>P</u>. <u>promelas</u> are < 24 h and 1 to 14 days, respectively,
at the start of testing. Fish from a single day's hatch are used whenever possible.

DILUTION WATER:

Dilution water is receiving water collected upstream of the point of discharge from the client's facility. Laboratory water prepared with either reagent-grade salts or Perrier mineral water is prepared in the New England Bioassay laboratory and used to culture the test organisms. The laboratory water may be used as the test dilution water, if requested.

WATER QUALITY DETERMINATION:

Sample temperature, pH, conductivity, hardness, alkalinity, and concentrations of dissolved oxygen and total residual chlorine (TRC) are measured upon sample receipt. Conductivity is measured with YSI Model 33 S-C-T meter; dissolved oxygen is measured with YSI Model 5IB meter; pH and temperature are measured with Beckman Model 12 pH/ISI meter; hardness is determined by the EDTA titrimetric method; alkalinity by potentiometric titration; and TRC by the DPD method.

GENERAL TEST CONDITIONS AND PROCEDURES:

The toxicity tests are static non-renewal tests, in which the test organisms are exposed to the same solution for the entire test Test conditions are summarized in Tables 1 and 2. tests are performed at a temperature of 25 ± 1°C and a photoperiod of 16 h of light and 8 h of darkness. Refrigerated solutions are brought to ambient temperature before testing. Test duration is brought to ambient temperature before testing. 48 h for both species. When the initial concentration of dissolved oxygen in the undiluted sample is less than 40% of the saturation value, samples are aerated before the organisms are introduced and subsequently during testing; otherwise no supplemental aeration is *'•* provided during testing. The organisms are not fed during testing. The daphnids test concentration are replicated 4 times with 5 animals per replicate. The fish test concentrations are replicated twice with 10 animals per replicate.

The measured effect in each test is death or immobility, evidenced by failure to respond to gentle prodding. At 24-h intervals throughout testing, survival data are collected and recorded, and dead organisms are removed from the test chambers.

REFERENCE TOXICANT

Reagent grade sodium chloride is used as a standard toxicant to authenticate the sensitivity of the laboratory stocks of daphnids Definitive tests with the reference toxicant are performed at least once per month according to standard protocols.

Specific Toxicity Tests

DEFINITIVE TEST:

In a definitive test, organisms are exposed in replicate chambers to five, dilutions of effluent using a 0.5 dilution factor (e.g. to five, dilutions of effluent using a 0.5 dilution factor (e.g. 6.25, 12.5, 25, 50, and 100), plus the permitted effluent concentration. Control organisms are exposed in replicated test chambers to dilution water(s) (0% effluent). A definitive test is used to determine the median lethal concentration (LC₅₀) of an effluent (see "Statistical Analysis of Data" below).

SCREENING ("PASS/FAIL") TEST:

In a screening test, organisms are exposed in replicated test chambers to a single concentration of either effluent or receiving
water. Control organisms are exposed in replicate chambers in Control organisms are exposed in replicate chambers in laboratory-prepared water. Survival of the test animals in the undiluted effluent or receiving water are compared with control survival data to determine toxic impacts.

Statistical Analysis of Acute Toxicity Data

All computer programs for statistical analysis of data were obtained from the Water Compliance Unit of the Connecticut DEP. Mortality data are analyzed statistically by .four different methods to determine the "median lethal concentration" (LC_{50}) and the "no observed acute effect level" (NOAEL) of the effluent. The LC_{50} is the concentration that is lethal to 50% of the organisms within the test period. The NOAEL is the highest concentration at which there is no significant difference (P > 0.05) in survival of animals in the test concentrations when compared with control survival.

The LC_{50} program estimates a LC_{50} by using one of four methods: binomial, moving-average angle, probit analysis, or trimmed Spearman-Karber. The method selected is based on the shape of the concentration - effects curve and the number of concentrations with partial mortalities (mortality greater than 0% but less than 100%). partial mortalities (mortality greater than 0% but less than 100%).
The moving-average, probit, and trimmed Spearman-Karber methods both estimate the LC_{50} with 95% confidence limits. The bounds placed on the LC_{50} using the binomial test are not 95% confidence limits, but can be used as statistically sound conservative bounds that are always above 95% when the animal sample size per concentration is large enough ($N \ge 6$) (Stephan, 1977). Sample size in all acute toxicity tests \geq 20 animals per test concentration. The value with the "best fit", i.e. that which best matches the raw data and has the narrowest 95% confidence interval (the range of values within which the true LC_{50} value could occur 95% of the time), is then selected.

For screening tests, Fisher's exact test is used to determine if there is a significant difference $(P < 0.05)$ between survival of animals exposed to either effluent or receiving water and control animals exposed to laboratory-prepared water. The statistical software program for the Fisher's exact test is TOXSTAT (Release No. 3.2) developed by Gulley et al. (1989) of the University of Wyoming (Laramie, Wyoming). This package was developed to address the statistical requirements described for analysis of C. dubia and fathead minnow acute and chronic toxicity data.

TABLE 1. EPA REGION I RECOMMENDED EFFLUENT TOXICITY TEST CONDITIONS FOR THE DAPHNIDS (Ceriodaphnia dubia and Daphnia pulex) 48 HOUR ACUTE TESTS¹

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TABLE 1. EPA REGION I RECOMMENDED EFFLUENT TOXICITY TEST CONDITIONS FOR THE DAPHNIDS *(*Ceriodaphnia dubia and Daphnia pulex) 48 HOUR ACUTE TESTS1 (CONTINUED)

Footnotes;

- 1. Adapted from EPA/600/4-85/013
- 2. Standard prepared dilution water must have hardness requirements to generally.reflect the characteristics of the receiving water.
- 3. When receiving water is used for dilution, an additional control made up of standard dilution water (0% effluent) is required).

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TABLE 2. REGION I RECOMMENDED TEST CONDITIONS FOR THE FATHEAD

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TABLE 2. REGION I RECOMMENDED TEST CONDITIONS FOR THE FATHEAD MINNOW (Pimephales promelas) 48 HOUR ACUTE TEST1

Footnotes:

- 1. Adapted from EPA/600/4-85/013. Fathead minnow test may be extended to 96 h, if required.
- $2.$ Standard dilution water must have hardness requirements to generally reflect characteristics of the receiving water.
- $3.$ When receiving water is used for dilution, an additional control made up of standard dilution water (0% effluent) is required.

APPENDIX C

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RAW ACUTE TOXICITY DATA SHEETS FOR SEDIMENT ELUTRIATES TESTS WITH Ceriodaphnia dubia AND FATHEAD MINNOWS

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NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA FORM

rest ID + 93-2043

Section

NEW ENGLAND BIOASSAY
ACUTE TOXICITY DATA

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**NEW ENGLAND BIOASSAY
ACUTE TOXICITY DATA**

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coc #93-1836

Test ID 193-2044

 $Proj$ $#$ 193-017

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Data Reviewed by: John DCevrey

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GZA ELUTRIATE SAMPLE **HO. 21** CERIODAPHKIA DUBIA TEST HO. 93-2043

- CROSSTAB / CHI-SQUARE TESTS --OBSERVED FREQUENCIES

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CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = .000, PROB.=1.0000 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = .351, PROB.= .5536

 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail = .8814, Upper Tail = .5000

————————————— CROSSTAB / CHI-SQUARE TESTS OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = .268, PROB.= .6048 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 1.071, PROB.= .3006 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail = .9438, Upper Tail = .3060

—————————————— CROSSTAB / CHI-SQUARE TESTS OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 3.606, PROB.= .0576 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 5.192, PROB.= .0227 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail = .9977, Upper Tail = .0262

GZA ELOTRIATE SAMPLE HO. 21 CERIODAPHNIA DUBIA TEST HO. 93-2043

- CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES

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CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 17.857, PROB.= 2.381E-05 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 20.317, PROB.= 6.560E-06

 $D.F. = 1$

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FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = $3.977E-06$

- CROSSTAB / CHI-SQUARE TESTS --OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 45.117, PROB.= 9.417E-11 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 48.654, PROB.= 7.866E-11

 $D.F. = 1$

 ζ FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = 7.744E-07

CT-TOX: BINOMIAL, MOVING AVERAGE, PROBIT, AND SPEARMAN METHODS

GZA ELUTRIATE SAMPLE HO. 21 FATHEAD MINNOW TEST HO. 93-2044

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GZA ELUTRIATE SAMPLE HO. 21 FATHEAD MINNOW TEST HO. 93-2044

————————————— CROSSTAB / CHI-SQUARE TESTS OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = $.517$, PROB.= .4720 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = .000, PROB.=1.0000 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail = .7542, Upper Tail = .7542

- CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = .517, PROB.= .4720 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = .000, PROB.=1.0000 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail = .7542, Upper Tail = .7542

NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA FORM COVER SHEET FOR LC50 TESTS

CLIENT: ADDRESS :

120 Needham Street Newton Upper Falls, MA 02164

CONTACT: Kr. Tim Briggs

CZA Environmental

SNOLE TYPE: Sediment Elutriate ISAmple $#24$ (1) $Finter$ DILUTION WATER SOURCE: __ MEB Artifical Freahwater

INVERTEBRATE

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- TYPE OF TEST TEST SET UP (TECH. INIT.): MS DEFINITIVE (X) SCREEN () RANGE () RENEWAL ()
- TEST SPECIES: Ceriodaphnia dubia SOURCE\NES LOT 1: NEB\ #220-236 CD-93.105-109 $ACZt \leq 24$ HOURS

TEST SOLUTION VOLUME: 30 (mL) NO. ORGANISMS PER TEST CHAMBER: 5

NO. ORGANISMS PER CONCENTRATION: -20-30

NO. ORGANISMS PER CONTROL: 44 30 START DATE: $5/14/43$ AT 1045 (hours) END DATE: $\frac{5}{12}$ $\frac{10}{12}$ $\frac{10}{2}$ at $\frac{115}{10}$ (hours)

LABORATORY WATER ARTIFICIAL FWI NEB Batch 17-062

INITIAL 100% ELUTRIATE CHEMISTRY

<u>C. dubia</u> TEST ID No: <u>73 - 2047</u>
P. promelas TEST ID No: <u>93 - 204</u>5
coc No: <u>93 - 1837</u> PROJECT NO: 193-017 Sediment GRAS SANGLE S/2/9 RDATE IATI ///O (BOURS) CONFOSITE SANFLE
COLLECTED FROM: (DATE)AT: (BOURS) τ ^{α} $(DATE)AT:$ $(2500R)$ **VERTESRATE** TTPE OF TEST TEST SET UP (TECH. INIT.): MS DEFINITIVE [X] SCREEN |] RANGE |] RENEWAL |]

EURDNESS: $\frac{U}{\frac{\log(1 + \alpha) \cos(1 + \alpha))}{\log(1 + \alpha) \cos(1 + \alpha)}}$ \mathcal{Z}_{Ω} ALKALINTTY

TECHNICIAN INITIALS:

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rest ID \$ 93-2047 coc 193-1837

NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA

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Proj # 193-017

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Data Reviewed by: Ark O Corner

Date: $5/19/93$

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**NEW ENGLAND BIOASSAY
ACUTE TOXICITY DATA**

Proj # 193-017

Data Reviewed by: John O Corre,

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Date: $5/19/97$

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CZA ELUTRIATE SAMPLE HO. 24 CERIODAPHNIA DUBIA TEST HO. 93-2047

- CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES

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CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 1.667, PROB.= .1967 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 2.963, PROB.= .0852

 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail = .9881, Upper Tail = .0973

——————————————— CROSSTAB / CHI-SQUARE TESTS OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 1.667, PROB.= .1967 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 2.963, PROB.= .0852 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail = .9881, Upper Tail = .0973

—————————————— CROSSTAB / CHI-SQUARE TESTS OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 5.880, PROB.= .0153 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 7.680, PROB.= 5.584E-03 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail = .9996, Upper Tail = $6.091E-03$

GZA ELUTRIATE SAMPLE HO. 24 CERIODRPHNIA DUBIA TEST NO. 93-2047

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 11.273 , PROB.= $7.863E-04$ CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 13.416 , PROB. = $2.495E-04$ $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = $2.155E-04$

- CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 23.736, PROB.= 1.105E-06 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 26.447, PROB.= 2.710E-07 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = $3.091E-06$

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CT-TOX: BINOMIAL, MOVING AVERAGE, PROBIT, AND SPEARMAN METHODS —— — — ^-—»••_..«_*_________**_.,»——•.—.•.•»«"»__._•-••—..«—»_______*•__...•_•_____._•__*___-— — — — .—— — — ___________ ___-_ -•____

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GZA ELUTRIATE SAMPLE HO. 24 FATHEAD MINNOW TEST HO. 93-2048

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- CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES LIVE DEAD TOTAL 30 CONTROL 30 0 30 6.25% 29 1 30 TOTAL CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = .000, PROS.=1.0000 \sim CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 1.017, PROB.= .313 $D.F. = 1$ FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = .5000 - CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES LIVE DEAD TOTAL 30 CONTROL 30 0 30
12.5% 29 1 30 12.5% 29 1 30
TOTAL 59 1 60 TOTAL CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = .000, PROB.=1.0000 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 1.017 , PROB.= .3132 $D.F. = 1$ FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = .5000 - CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES LIVE DEAD TOTAL 30 0 30 CONTROL 30 0 30
25% 28 2 30 $25%$ 28 2 30 TOTAL 58 2 60 CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = .517, PROB.= .4720 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR $=$ 2.069, PROB.= $.1503$ $D.F. = 1$ FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = .2458

GZA ELUTRIATE SAMPLE HO. 24 FATHEAD MINNOW TEST HO. 93-2048

- CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR $=$.517, PROB.= .4720 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 2.069 , PROB.= .1503

 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = .2458

NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA FORM COVER SHEET FOR LC50 TESTS

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NEW ENGLAND BIOASSAY
ACUTE TOXICITY DATA

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client: GZA Environmental

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 D^{2+a} $S^{1/9/27}$, ,
CT-TOX: BINOMIAL, MOVING AVERAGE, PROBIT, AND SPEARMAN METHODS

 MINIMUM REQUIRED TRIM IS TOO LARGE: 68.3,SO SK IS NOT CALCULABLE. SPEARMAN-KARBER

THE BINOMIAL TEST SHOWS THAT 25.00 AND +INFINITY CAN BE USED AS STATISTICALLY

SOUND CONSERVATIVE 95 PERCENT CONFIDENCE LIMITS SINCE THE ACTUAL CONFIDENCE LEVEL ASSOCIATED WITH THESE LIMITS IS 97.8613 PERCENT. THE LC50 FOR THIS DATA SET IS GREATER THAN 100.00 ---------------

THE MOVING AVERAGE METHOD CANNOT BE USED WITH THIS DATA SET BECAUSE NO SPAN WHICH PRODUCES AVERAGE ANGLES BRACKETING 45 DEGREES ALSO USES TWO PERCENT DEAD BETWEEN 0 AND 100 PERCENT.

NO CONVERGENCE IN 25 ITERATIONS. PROBIT METHOD PROBABLY CAN NOT BE USE WITH THIS SET OF DATA.

NOTE: MORTALITY PROPORTIONS WERE NOT MONOTONICALLY INCREASING. ADJUSTMENTS WERE MADE PRIOR TO SPEARMAN-KARBER ESTIMATION.

**** = LIMIT DOES NOT EXIST

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----------------- CROSSTAB / CHI-SQUARE TESTS ------------------OBSERVED FREQUENCIES

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CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = .000, PROB.=1.0000 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 1.017, PROB.= .3132 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = .5000

/ CHI—SQUARE TESTS •——— ————————————— OBSERVED FREQUENCIES

CHI-SQUARE WITH , CONTINUITY CORRECTION FACTOR = 7.067 , PROB. = $7.850E-03$ CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 9.231, PROB.= 2.380E-03 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = $2.288E-03$

— — ——————————————— «_ivj&j>j.<»uj / v*n.L— oyuAnc. OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 8.366, PROB.= 3.823E-03 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 10.588, PROB.= 1.138E-03

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 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = $9.678E-04$

CZA ELUTRIATE SAMPLE HO. 30 CERIODAPHNIA DUBIA TEST NO. 93-2045

----------------- CROSSTAB / CHI-SQUARE TESTS --------------------
OBSERVED FREQUENCIES

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CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 9,720, PROB.= 1.823E-03 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 12.000 , PROB. = $5.320E-04$ $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = 3.985E-04

- CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 8.366, PROB.= 3.823E-03 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 10.588, PROB.= 1.138E-03

 $D.F. = 1$

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FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = $9.678E-04$

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ISSTAB / CHI-SQUARE TESTS OBSERVED FREQUENCIES

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CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = .000, PROB.=1.0000 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = .351, PROB.= .5536 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail = .5000, Upper Tail = .8814

- CROSSTAB / CHI-SQUARE TESTS -OBSERVED FREQUENCIES

CHI-SQUARE WITH CONTINUITY CORRECTION FACTOR = 10.313, PROB.= 1.321E-03 CHI-SQUARE WITHOUT CONTINUITY CORRECTION FACTOR = 12.273, PROB.= 4.596E-04 $D.F. = 1$

FISHER EXACT PROBABILITY: Lower Tail =1.0000, Upper Tail = $4.549E-04$

NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA FORM COVER SHEET FOR LC50 TESTS

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Date: $5/19/93$

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Date: $5/12/23$

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NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA FORM COVER SHEET FOR LC50 TESTS

CONTACT: Mr. Tim Brigge \vec{r} 30 (centrifyed) r SANGLE TTPE: Sediment Elutriate : Sample DILUTION WATER SOURCE: __ NEB Artifical Freshwater

INVERTEBRATE

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LABORATORY WATER ARTIFICIAL PW: HES Batch & C-MY-93-063

INITIAL 100% ELUTRIATE CHEMISTRY

P. promelas TEST ID NO: $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ $\frac{1}{2}$ PROJECT NO: 193-017 Suliment /93_(DATE)ati GUAL SANGLE (EOURS) CONFOSITE SANGLE (DATZ)AT: (EOURS) \mathbf{r} (DATE)ATI $(EOURS)$ **TASESTATY** $T122$ $Q2$ $T251$ $T251$ $S27$ $Q2$ $(T2C1, 1N12, 11)$ DETINITIVE (X) SCREEN () RANGE () RENEWAL () TEST SPECISS: Fathead Kinnows (Pimephales promelas) sourcr\NES LOT #: YES\ AGE 4 DAYS $\frac{1}{2}$ solution volume: $\frac{1}{200}$ (=1) NO. ORGANISKS PRATEST CEAMBER: 10 NO. ORGANISKS PER CANTROLI 20 START DATE: AT $\overline{ }$ (hours) END DATE: λ ₇ (hours)

C. dubia TEST TO NO: $83 - 2034$

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ALXALINITY: $\frac{4}{\sqrt{3}}$ EXADNESS: $\sqrt{\frac{2}{100}}$ $\overline{c_4c_0}$ p_L) TECHNICIAN INITIALS:

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Reviewed by: John O Cerney

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Date: $5/19/93$

NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA FORM COVER SHEET FOR LC50 TESTS

SANZIE TTPE: Sedinent Elutriate (Sanple: MAShipping Cost DILUTION WATER SOURCE: MES Artifical Preshwater

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LABORATORY VATER ARTIFICIAL PH: NES Batch + $L - M \rho - P_2 - C_2/2 \delta$ $\overline{\varkappa}$ $\overline{\varkappa}$

INTTIAT, 1008 FLUTRIATE CHENISTRY

PROJECT NO: 193-017 Sediment
GUASNELL 1/30
COLLECTED ON: 5/10/93/01TEINT: 1200 (2007ES) COMPOSITE SAMPLE
COLLECTED FROM: (DATE)ATI___ $1800R5$ 701 $(DATE)AT:$ $(BOURS)$ PERTEBRATE TIPE OP TEST TEST SET UP (TICK. INIT.) 1__ DEFINITIVE XI SCREEK () RANGE () RENEVAL () TEST SPECIES: Pathead Kinnows (Pimephales promelas) source\xes for fi KEB\ $AGE +$ DAYS_ 1251 SOLUIQUA VOLUMIS: 100 (21) NO. ORGANISMS PER TEST REAMBER: 10 NO. ORGANISMS PER CONCENTRATION: 20 NO. ORGANISKS PER CONTROLA 20_o $START$ $DATE:$ \mathbf{r} (hours) END DAIE: $\overline{2}$ at $\overline{2}$

 $s.$ dubia TZST ID NO: $93-203/4$

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(bhours)

P. promelas TEST ID No: NA

EXADNESS: $\frac{1}{\frac{\log(1 + \cos \csc 0)}{\log(1 + \cos \csc 0)}}$ ALXALINITY: $\frac{473}{(29/1 \cdot 16 \cdot 5 \cdot 10^{-1})}$ \mathcal{V}_{1} TECHNICIAN INITIALS:

Reviewed by: Colo O Currey

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Date: $5/19/23$

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NEW ENGLAND BIOASSAY ACUTE TOXICITY DATA FORM COVER SHEET FOR LC50 TESTS

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P. promelas TEST ID NO: 93-203/C coc Not 93-1823 PROJECT NO: 193-017 Sedinent 1130 GRAS SAIGLE 5/10/97 (DATE)ATI/2CO (BOURS) CONZOSITE SANGLE
COLLECTED FROM: (DATE) AT: $($ KOURS) $T01$ (DATE)ATI___ $(500R)$ YERTEBRATE

<u>C. duble</u> TEST ID No: $93 - 2031B$

TTPE OF TEST TEST SET UP (TECH. INIT.): DEFINITIVE HE SCREEN D(1 RANGE [1 RENEWAL ()

TEST SPECIES: Fathead Minnows (Pinephales promelas) SOURCE\NES LOT 1: NES\ $93 - 115$ A + B $ACZ:$ 9 DXS TEST SOLUTION VOLUME: 700 (aL) NO. ORGANISMS PER TEST CEANSER: 10 NO. ORGANISMS PER CONCENTRATION: 40 30 NO. ORGANISMS PER CONTROL: $\frac{29-30}{25}$ START DATE: $S/14/93$ AT $/040$ (hours) END DATE: $5/18/93$ AT $16/5$ (hours)

EXADITESS: $\frac{5}{\frac{200}{200}}$ ACKALINITY: $\frac{93}{129/1}$ as CaCO₃)

INITIAL 100% ELUTRIATE CHEMISTRY

ARTIFICIAL PV: RES Batch + CMP 93-063

TECHNICIAN INITIALS:

RESULTS OF Pimephales promelas LC50 T2ST RESULTS OF Ceriodaphaia duble LC50 TEST LC_{50} (1) 951 CONTIDENCE LIMITS (1) **RETAÇO** LC_{50} (t) 951 CONTIDENCE LIMITS (t) **METHOD** ANOHIAL DISTRIBUTION BINOMIAL DISTRIBUTION: HOVING AVERAGE-ANGLE: HOVING AVERAGE-ANGLE: PROBIT: **PROBIT:** TRINGTO SPEARVAN KARBER: **TRINGED SPEARMAN KARBER:** OTEZR: OTHER: **NOAZL1** NOAZLI NOAEL: NO-OBSERVED-ACUTE-EFFECT LEVEL ANIMAL CONDITION/BEHAVIOR: 77% survival in 100% filted elatriate C. dubia: Fatheon Mirrory: 97% Survival in 100% Fillered clutriate

REVIEWED BY: Augh D'Cersney

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Date: $5/19/93$

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GREAT BLUE HERON FOOD WEB ASSESSMENT

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TABLE OF CONTENTS

Page

 $\bar{\nu}$

TABLE OF CONTENTS

- DATA
- TABLE 1-9 LOWER SIMMONS RESERVOIR SEDIMENT SUMMARY OF ANALYTICAL DATA
- TABLE I-10 LOWER SIMMONS RESERVOIR SURFACE WATER SUMMARY OF ANALYTICAL DATA
- TABLE 1-11 PHYSICAL AND CHEMICAL INPUT PARAMETERS FOR GOBAS BIOACCUMULATION MODEL

TABLE OF CONTENTS

- TABLE I-12 BIOLOGICAL INPUT PARAMETERS FOR GOBAS BIOACCUMULATION MODEL
- TABLE 1-13 SEDIMENTATION PONDS 2 AND 3 GOBAS BIOACCUMULATION MODEL RESULTS
- TABLE I-14 UPPER SIMMONS RESERVOIR GOBAS BIOACCUMULATION MODEL RESULTS
- TABLE I-15 UPPER SIMMONS RESERVOIR NORTH BASIN GOBAS BIOACCUMULATION MODEL RESULTS
- TABLE I-16 LOWER SIMMONS RESERVOIR GOBAS BIOACCUMULATION MODEL RESULTS
- TABLE I-17 SEDIMENTATION POND 4 GOBAS BIOACCUMULATION MODEL RESULTS
- TABLE 1-18 CALCULATION OF SEDIMENT/INVERTEBRATE BIOCONCENT-RATION FACTORS FOR HEAVY METALS
- TABLE I-19 CALCULATION OF SEDIMENT/ADPOLE BIOCONCENTRATION FACTORS FOR HEAVY METALS
- TABLE 1-20 SUMMARY OF SURFACE WATER/FISH BIOCONCENTRATION FACTORS FOR HEAVY METALS
- TABLE 1-21 CALCULATION OF SEDIMENT/FISH BIOCONCENTRATION FACTORS FOR HEAVY METALS
- TABLE 1-22 WEIGHT ASSIGNMENTS FOR SURFACE WATER BODIES BASED ON RELATIVE SURFACE AREAS
- TABLE 1-23 SITE-SPECIFIC FISH BODY BURDENS FOR ZINC
- TABLE 1-24 SUMMARY OF EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS
- TABLE 1-25 SUMMARY OF WEIGHTED EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS
- TABLE 1-26 ENTIRE CENTRAL LANDFILL DRAINAGE AREA FEEDING AREA NORMALIZED EPCs FOR THE GREAT BLUE HERON

TABLE OF CONTENTS

- TABLE 1-27 ENTIRE CENTRAL LANDFILL DRAINAGE AREA CALCULATION OF DAILY DOES FOR THE GREAT BLUE HERON
- TABLE 1-28 UPPER SIMMONS RESERVOIR CALCULATION OF DAILY DOSES FOR GREAT BLUE HERON
- TABLE 1-29 UPPER SIMMONS RESERVOIR NORTH BASIN CALCULATION OF DAILY DOSES FOR GREAT BLUE HERON
- TABLE 1-30 LOWER SIMMONS RESERVOIR CALCULATION OF DAILY DOSES FOR GREAT BLUE HERON
- TABLE 1-31 SEDIMENTATION POND 2 AND 3 CALCULATION OF DAILY DOSE FOR GREAT BLUE HERON
- TABLE 1-32 SEDIMENTATION POND 4 CALCULATION OF DAILY DOSES FOR GREAT BLUE HERON
- TABLE 1-33 ENTIRE CENTRAL LANDFILL DRAINAGE AREA GREAT BLUE HERON ESTIMATED DAILY DOSES COMPARED TO TOXICOLOGICAL REFERENCE DOSES
- TABLE 1-34 UPPER SIMMONS RESERVOIR GREAT BLUE HERON ESTIMATED DAILY DOSES COMPARED TO TOXICOLOGICAL REFERENCES DOSES
- TABLE I-35 UPPER SIMMONS NORTH BASIN GREAT BLUE HERON ESTIMATED DAILY DOSES COMPARED TO TOXICOLOGICAL REFERENCE DOSES
- TABLE 1-36 LOWER SIMMONS RESERVOIR GREAT BLUE HERON ESTIMATED DAILY DOSES COMPARED TO TOXICOLOGICAL REFERENCE **DOSES**
- TABLE 1-37 SEDIMENTATION PONDS 2 AND 3 GREAT BLUE HERON ESTIMATED DAILY DOSES COMPARED TO TOXICOLOGICAL REFERENCE DOSES
- TABLE I-38 SEDIMENTATION POND 4 GREAT BLUE HERON ESTIMATED DAILY DOSES COMPARED TO TOXICOLOGICAL REFERENCE DOSES

TABLE OF CONTENTS

- TABLE 1-39 GREAT BLUE HERON FOOD WEB ASSESSMENT SUMMARY OF TOXICITY QUOTIENTS AND TOTAL HAZARD QUOTIENTS
- TABLE 1-40 EFFECT OF TOTAL ORGANIC CARBON CONTENT IN TOTAL SUSPENDED SOLIDS ON CALCULATED TISSUE CONTAMINANT CONCENTRATIONS

LIST OF FIGURES

FIGURE 1 AQUATIC FOOD WEB CONSIDERED FOR EXPOSURE OF GREAT BLUE HERON TO CONTAMINANTS IN SURFACE WATER AND SEDIMENT

GREAT BLUE HERON FOOD WEB ASSESSMENT

1.00 INTRODUCTION

This report describes the methods used to estimate potential risks to wildlife associated with exposure to sediment and surface water contaminants within Upper and Lower Simmons Reservoirs, the Quarry Stream and Cedar Swamp Brook, and Sedimentation Ponds 2, 3 and 4 within the active portion of the Central Landfill property. The risk estimate for wildlife exposure is based on assessment of risk to the great blue heron *(Ardea herodias),* a largely piscivorous wading bird expected to utilize these exposure points as foraging habitat. An exposure model which incorporates the feeding and foraging habits of the heron was used to estimate the heron's exposure to contaminants in sediment, surface water and in representative prey organisms. Concentrations of organic contaminants in prey organisms were estimated using a widely accepted model (Gobas, 1993) which predicts the bioaccumulation of organic contaminants through an aquatic food-web. Assumptions used in this model were intended to conservatively represent the trophic relationships of aquatic species within the CLF Drainage Area (defined as Sedimentation Pond 4, Sedimentation Ponds 2&3 and Channels, the Upper Simmons Reservoir, and the Lower Simmons Reservoir). Bioconcentration factors presented in recently published regulatory and scientific literature were used to estimate concentrations of inorganic contaminants in aquatic/semiaquatic species likely to be preyed upon by the heron. The exposure levels (doses) calculated for the heron were compared to toxicological reference doses obtained from current literature to assess the potential for adverse health effects. The following sections describe the exposure model for the heron, the aquatic bioaccumulation models, input assumptions for the models, the toxicological reference doses (RfDs), and the results of this preliminary risk assessment for the great blue heron.

The selection of Contaminants of Potential Ecological Concern (COPECs) for the sub-areas within the CLF Drainage Area was based on comparisons of OU2 contaminant concentrations to background and toxicological benchmark concentrations. COPEC selection is presented in Section 9.14 of the main RI report. CLF Drainage Area COPECs include VOCs, SVOCs, PCB/Pesticides, and inorganic contaminants.

2.00 FOOD WEB EXPOSURE MODEL FOR THE GREAT BLUE HERON

The food chain model used for this evaluation estimates exposure of the indicator species to COPECs within prey organisms, due to water consumption and incidental ingestion of sediment. The model takes into consideration the daily food ingestion rate of the heron, the proportions of diet made up by different types of affected prey organisms, and the proportion of the feeding area which is comprised by the different exposure points being evaluated.

Exposure of receptors to site contaminants was estimated using the following formula:

where:

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The following sections describe assumptions made regarding great blue heron feeding habits, exposure durations, and present methods used to estimate the concentrations of contaminants in the prey organisms of the heron.

7.11 Exposure Duration and Feeding Habits

Information regarding the habitat and foraging requirements of the great blue heron were obtained from EPA (1993a&b) and from DeGraaf and Rudis (1987). The following paragraphs summarize information used to develop an exposure model for the heron.

The heron is a migratory species, and is likely to arrive in the vicinity of the Site in the spring (coincident with the ice-melt) and likely flies south by October or November (EPA, 1993a). Seasonal migration reduces the average daily exposure of the heron when factored over the course of a year or more, however, this was not taken into account in the model used for this evaluation. The reason for this is that the durations of the majority of the toxicity tests used to derive benchmark doses were less than the length of time herons are likely to spend on the site. Therefore, we assumed that if effects were to occur to the heron they would occur over the course of the breeding season when the heron (or similar bird species) may be expected to be at the Site. However, this assumption may be conservative for some contaminants; the durations of the toxicity test used to derive benchmark doses for Aroclor 1242, DDT and its degradation products, and mercury were longer than the period over which the herons are expected to be at the site each year.

The aquatic foraging habitats of the great blue heron include shallow standing and flowing waters up to 0.5 meters deep with firm substrate (EPA, 1993a). Studies which document the foraging domains of great blue herons present results in units of linear measurement (e.g., kilometers of shoreline), reflecting the dependence of the heron on shallow water. The linear extent of defended foraging territory for individual herons ranges from 0.129 km to 0.98 km (EPA, 1993b). The daily food ingestion rates for the heron has been estimated at 0.18 grams of food per gram body weight per day. The composition of the heron's diet, estimated by averaging values for habitats similar to the Site reported in EPA (1993b), includes approximately 81 percent fish, 3 percent amphibians, and 10 percent macroinvertebrates. The remainder of the heron's diet includes plants and non-aquatic animals. Fish in the heron diet are reported to be 20 cm or shorter in length. EPA (1993a) provides an estimated water ingestion rate for the heron of 0.045 grams/gram body weight per day.

An incidental sediment ingestion rate for herons was estimated based on reported ingestion rates for the semipalmated sandpiper. The semipalmated sandpiper feeds almost exclusively on mud-dwelling invertebrates. We conservatively assumed that all of the heron's macroinvertebrate and amphibian foraging activities result in incidental ingestion of sediment. The maximum reported sediment ingestion rate for the sandpiper, 30 percent (1993b), was multiplied by the fraction of the diet comprised by amphibian and macroinvertebrate portions of the heron's diet (i.e., 0.13). The resulting incidental sediment ingestion rate of 3.9 percent was used in the exposure model for the heron. This is likely conservative because macroinvertebrates ingested by herons are likely to be mainly epiphytic rather than infaunal.

2.12 Exposure Points Evaluated

Surface water bodies evaluated in this assessment included the Upper Simmons Reservoir (USR), Lower Simmons Reservoir (LSR), Sedimentation Ponds 2, 3, and 4, Cedar Swamp Brook and the Quarry Stream. These are the waterbodies to which all of the surface water from the landfill drains, and to which most of the groundwater drains. Therefore, these water bodies have the greatest potential for impacts from the landfill. These exposure points are referred to collectively as the Central Landfill Drainage Area (CLF Drainage Area). Based on their topographic positions and contaminant concentrations, data from Cedar Swamp Brook and Quarry Stream were combined with those for Sedimentation Ponds 2 and 3 (referred to as Sed Ponds 2&3 and Channels) to represent one exposure point. Because sediment containment booms are present between the northern and southern basins of the USR, the northern basin was also treated as an individual exposure point.

Several different runs of the food web model were performed. One run assumed that an individual heron feeds throughout the CLF Drainage, thus data from all the sub-areas within the CLF Drainage Area were incorporated in the body burden estimates for heron prey items. For each of the remaining food web model runs we assumed that an individual heron feeds exclusively within one of the CLF Drainage Area sub-areas, and data from only that sub-area were incorporated into the body burden estimates. Assumptions regarding the foraging area for each run of the model were as follows:

• The entire CLF Drainage Area was assumed to comprise 100% of the foraging area of the heron. Exposure Point Concentrations (EPC) within each exposure point (i.e., USR, LSR, Sed Ponds 2&3 and Channels, and Sed Pond 4) were weighted based on the size of the exposure point, relative to the entire CLF Drainage Area, to develop Feeding Area Normalized EPCs for the entire CLF Drainage Area. The surface area of water within each exposure point was used as a rough estimate of the relative amount of foraging area within that exposure point. Thus, the EPCs within exposure point X were weighted by multiplying by:

Surface Water Area at Exposure Point X Total Surface Water Area Within the CLF Drainage Area

Then, the Feeding Area Normalized EPCs were calculated by summing the weighted EPCs from each exposure point. Table 1-22 presents a summary of the weighting factors calculated for each exposure point.

- Sed Pond 4 was assumed to comprise 100 percent of the heron's foraging area.
- Sed Ponds $2\&3$ and Channels (which includes Cedar Swamp Brook and the Quarry Stream) were assumed to comprise 100 percent of the heron's foraging area.
- The Upper Simmons Reservoir was assumed to comprise 100 percent of the heron's foraging area.
- The Lower Simmons Reservoir was assumed to comprise 100 percent of the heron's foraging area.
- The North Basin of USR still contains significant amounts of potentially landfillderived sediments, which generally have higher concentrations than the naturally deposited "original" sediments exposed in the main body of USR after dredging. For this reason, and at the request of EPA, the North Basin of the USR (including Cedar Swamp Brook delta within Upper Simmons Reservoir) was assumed to comprise 100 percent of the heron's foraging area.

Within each of these different exposure points, average concentrations within surface water and sediment were used as the basis for estimating the exposure of herons to site contaminants. Maximum concentrations were not used to estimate heron exposure to COPECs because this would have produced an overly conservative assessment. The herons feeding range (approximately 0.129 to 0.98 kilometers of shoreline) is large relative to the habitat size provided by the different exposure points. Also, a fairly large numbers of surface water and sediment samples (27 surface water sampling locations, and 36 sediment samples) were used to represent the CLF Drainage area. Therefore, it is highly unlikely that any individual heron would be exposed exclusively to an area with concentrations of COPECs comparable to the maximum concentrations. In addition, because the CLF Drainage Area was broken up into several smaller areas, if there were significant areas with COPEC concentrations consistently higher than the rest of the CLF Drainage Area, these conditions would be adequately represented by the average concentration for that exposure point.

However, in order to get an idea of the magnitude of difference between risk estimates calculated using the averages, and risk estimates using the maximum COPEC concentrations, the food web model for the Upper Simmons Reservoir was rerun using the maximum concentrations. The results of the average-based and maximum-based risk estimates for the Upper Simmons Reservoir are compared below.

2.13 Exposure Assumption Summary

The following assumptions were used as input parameters for the heron food web model:

2.20 BODY BURDEN ESTIMATES FOR PREY ORGANISM

As discussed above, estimates of COPEC body burdens within heron prey organisms (and by extension, heron exposure to COPECs) were based on average COPEC concentrations in surface water and sediment of each exposure point. The use of maximum concentrations was considered to be overly conservative, however, the magnitude of difference between risk estimates based on average concentrations and those based on maximum concentrations is discussed in Section 4.00.

2.21 Gobas Model for Organic Contaminant Uptake.

In order to conservatively predict tissue concentrations of organic COPECs in aquatic organisms inhabiting the surface waters adjacent to the Site, we applied the food-web bioaccumulation model of Gobas (1993). We obtained a copy of the food-web computer model from Gobas in 1995 ("A Bioaccumulation and Trophic Transfer Model for Contaminants in Aquatic Food Webs - Version 1.00, May, 1994"). This model has been used to produce the generic Food Chain Multipliers presented in the Great Lakes Water Quality Guidance, and in EPA's "Ambient Water Quality Criteria Derivation Methodology Human Health Technical Support Document - Final Draft" (EPA, 1998). Each of these documents provides detailed descriptions of the Gobas model.

The Gobas model allows estimation of bioavailable surface water contaminant concentrations, tissue concentrations, lipid concentrations, bioconcentration factors (BCFs), bioaccumulation factors (BAFs), fugacities, and uptake/elimination factors using site-specific descriptions of sediment/water chemistry and trophic relationships. Input parameters required include:

- 1. Molecular Weight (MW) for each COPEC
- 2. Henry's Law Constant for each COPEC
- 3. Octanol-water partition coefficient (log Kow) for each COPEC
- 4. Dissociation constant (for ionizing substances only)
- 5. Concentration of suspended solids
- 6. Concentration of COPEC in suspended solids
- 7. Concentration of COPEC in sediment
- 8. Concentration of COPEC in surface water
- 9. Organic carbon content of sediment and suspended solids
- 10. Organism weight
- 11. Organism lipid content
- 12. pH
- 13. Water temperature (Celsius)
- 14. Feeding Preferences (or trophic interactions)

Figure 1 depicts trophic relationship assumptions used in the Gobas model. Tables I-1 through 1-12 list chemical, physical and biological input parameters used to run the model. Of the input parameters listed above, measured site-specific values were available for concentrations of suspended solids, concentrations of organic COPECs in sediment, concentrations of organic COPECs in surface water, organic carbon contents of sediment, and pH. We assumed that the organic carbon and organic COPEC content of suspended solids equaled that of bottom sediment. Water temperature was assumed to be 18 degrees C $(+/- 5)$ degrees). With the exception of Aroclor 1242, all physical constants for organic COPECs were obtained from EPA Guidance documents (i.e. EPA, 1986 and EPA, 1998). Whenever possible, lipid contents of species included in the food-web were obtained from EPA (1997). Other lipid content assumptions were based on values reported in current scientific literature. Reasonably conservative assumptions regarding organism feeding preferences were based on information presented in EPA (1993a&b) and in Carlander (1977).

Based on a review of limnological literature, our assumption that the total organic carbon (TOC) content of suspended solids is justifiable and conservative because the organic content of resuspended matter may exceed the organic content of bottom sediment (Kawana and Tanimoto, 1984). Other researchers (e.g. Meyers *et al.,* (1984)) have shown that the organic content of paniculate matter within the top 1 meter of surface water is largely composed of terrigenous lipid matter which is degraded slower than the lipid matter of aquatic origin which dominates the suspended solids of deeper waters. Prey items of the great blue heron are likely to spend the majority of the year in shallow water where organic carbon content of suspended solids is higher, thus, bioavailability of organic contaminants in these areas may be lower compared to deeper water. A data set for Lake Ontario summarized by Campfens and Mackay (1997) documents the organic carbon fraction of sediment as 0.02 and the organic carbon fraction of suspended particles as 0.2, supporting the conservative nature of our assumption. Because the relationship between the organic content of the suspended solids and the predicted body burdens of aquatic organisms is roughly linear, this conservative assumption may result in an approximately 3x to lOx overestimation of body burdens (see Section 6.00).

The Gobas model requires input of surface water and sediment concentrations for each contaminant being evaluated. If an organic contaminant was not detected in surface water (and therefore was not a surface water COPEC for that exposure point) we input a concentration of 1 x 10⁻⁷ mg/l, which is essentially a "zero" concentration. This value was used because, based on trial runs with DDT, 1×10^{-7} is the point at which surface water concentrations no longer have a significant influence on the estimated body burdens.

In a few cases (e.g., aldrin and DOT in the Lower Simmons Reservoir), organic contaminants were detected in surface water but not sediment. With the exception of VOCs, it is expected that contaminants found in surface water are also found in sediment, and that the reason they were not detected is likely due to small sample size or high detection limits. Therefore, in these cases, the sediment concentration was assumed to be one-half of the average sediment MDL for that data set.

2.21.1 Gobas Biological Uptake Model Results

Tables 1-13 through 1-17 present the body burdens and bioavailable surface water concentrations calculated by the model. The sections below provide a brief overview of the formulas used in the model. More detailed descriptions of these formulas are presented inGobas(1993).

Freely Dissolved Surface Water Concentrations of COPECs

The Gobas model predicts the bioavailable (or "freely dissolved") fraction of a COPEC in surface water (Bioavailable Solute Fraction [BSF]), using the formula:

$$
BSF = 1/(1 + (K_{OW} \times [OM]/dOM))
$$

where [OM] is the concentration (kg/L) of organic matter in the water, Kow is the octanol/water partitioning coefficient of the COPEC, and dOM is the density of the organic matter (kg/L). The model uses the calculated BSF to predict body burdens of organisms in the hypothetical food web.

COPRC Concentrations in Renthic Invertebrates

The Gobas model predicts COPEC concentrations in benthic invertebrates using the relationship:

$$
CB \times dL/LB = CS \times dOC/OC = KLW \times CP
$$

where CB is the chemical concentrations in the benthic invertebrate (μ g/kg wet weight), CS is the concentration in the sediments (μ g/kg dry weight), CP is the truly dissolved chemical concentration in the pore water (μ g/L water); LB is the lipid fraction of the benthic organisms (kg lipid/kg organism), dL is the density of the lipids of the benthic organisms (kg/L), OC is the organic carbon fraction of the sediments (kg organic carbon/kg sediment), dOC is the density of the organic carbon fraction of the sediments (kg/L) and KLW is the dimensionless lipid water partition coefficient. Since dL and dOC are approximately the same, CB/CS should be approximately similar for organic chemicals, namely, LB \times dOC/OC \times dL, or simply LB/OC. Thus, uptake by benthic invertebrates is seen as being primarily dependent upon the relationship between sediment organic carbon and the lipid fraction of the organism.

COPF.C Concentrations in Fish and Amphibians

The Gobas model predicts COPEC concentrations in fish using estimates of compound and species specific gill uptake rate constants, gill elimination rate constants, metabolic transformation rate constants, dietary uptake rate constants, fecal egestion rate constants, growth dilution, and feeding preferences. For the purposes of this risk assessment, we assumed that bullfrog tadpoles would exhibit the same bioaccumulation tendencies as fish. Bullfrog tadpoles are gilled and can spend as much as 3 years in the water before metamorphosing into adults (EPA, 1993a).

2.22 Inorganic Body Burden Estimates

2.22.1 Benthic Macroinvertebrate Tissue

For exposure points in which an inorganic COPEC had been detected in surface water, we estimated benthic invertebrate body burdens by multiplying the arithmetic mean total surface water concentration of the COPEC by bioconcentration factors (BCFs) for fish presented in EPA (1998b). If the COPEC was detected in sediment but not in surface water, we estimated body burdens by multiplying the arithmetic mean sediment concentration by the Invertebrate Accumulation Factors (lAFs) presented on Table 1-18. The LAFs for inorganics were calculated by dividing the COPEC-specific fish BCF from EPA (1998b) by the Kd_{bw} (bed-sediment to pore water partition coefficient) presented in EPA (1998b).

7.7.2.2 Amphibian Tissue

Body burdens for amphibians were estimated by multiplying mean total surface water concentrations by BCFs for fish presented in EPA (1998b). If an inorganic COPEC was not detected in surface water but was present in sediments, amphibian body burdens were calculated using the same method described above for invertebrates. In the case of mercury, the sediment/amphibian BCF presented on Table 1-19 was used.

2.22.3 Fish Tissue

Fish tissue samples (filets) were collected for inorganic analyses from the Upper and Lower Simmons Reservoirs by Environmental Science Services (ESS) in 1993 and 1994. Reports produced by ESS are presented in Appendix K. Fish tissue analytes consisted of cadmium, chromium, copper, lead, mercury, and zinc. With the exception of zinc and mercury, none of the other inorganic contaminants were detected. Mercury was detected in one tissue sample; 0.4 mg/kg wet weight (or about 2 mg/kg dry weight assuming a fish tissue percent solid value of 20 percent) of mercury was detected in a sample from the Upper Simmons Reservoir. Zinc was detected in all fish tissue samples; concentrations detected ranged from 4.2 to 6.0 mg/kg wet weight (about 21 to 30 mg/kg dry weight) in the Upper Simmons Reservoir, and from 2.1 to 8.6 mg/kg (about 11 to 43 percent dry weight) in the Lower Simmons. Average concentrations of zinc were 5.0 and 4.9 mg/kg wet weight (about 25 to and 24.5 mg/kg dry weight) in the Upper and Lower Simmons Reservoirs, respectively.

One potential option for estimating fish tissue concentrations for the metals analyzed but not detected by ESS was to use one-half the method detection limit as the body burden value. However, MDLs for data collected by ESS were quite high, and had we used those MDLs, this analytical artifact would have driven the results of our exposure estimate for these metals. Therefore, with the exception of zinc (which was positively detected in each fish sample), we estimated fish tissue concentrations for these, and other inorganic COPECs using BCFs obtained from EPA (1998c) (Table 1-20). The average concentration of zinc detected in fish tissue samples from the Upper Simmons was used to represent fish tissue EPCs for the Upper Simmons, and the average concentration of zinc in fish samples from the Lower Simmons was used to estimate fish tissue concentration in that waterbody. ESS reported fish tissue concentrations on an "as is" basis, which we took to mean on a wet weight basis. Therefore, these values were used as reported to estimate daily doses to the heron. Average fish tissue zinc concentrations for the Upper and Lower Simmons Reservoirs are presented in Table 23.

If an inorganic COPEC was detected in surface water, the fish tissue concentration was estimated by multiplying the arithmetic mean total surface water concentration by the published BCF. If a contaminant was detected in sediment but not in surface water, we estimated the fish tissue contaminant concentration by multiplying the average sediment concentration by the sediment to fish BCFs presented on Table 1-21, and by the fraction of solid content in fish to calculate the fish body burden on a wet weight basis. Fish species similar to species present in the reservoirs collected in a different study was measured to have approximately 20 percent solids. Thus, we used 0.2 as the fraction of solid content in fish.

3.00 REFERENCE DOSES FOR THE GREAT BLUE HERON

With the exception of the reference dose (RFD) for methoxychlor, RfDs for the great blue heron were obtained from Sample *et al.* (1996). The RFD for methoxychlor was obtained from EXTOXNET (1996). Tables 1-33 through 1-38 present RfDs for COPECs. Whenever possible, we used both No Observed Adverse Effects Levels (NOAELs) and Lowest Observed Adverse Effects Levels (LOAELs) in our risk evaluation. The following paragraphs summarize the test endpoints upon which each RFD was based.

T10 OR **fi**ANTC **fONTAMIN**AMTS

Acetone

Toxicological information regarding the effects of acetone on birds was not identified in Sample *et al.,* 1996. However, acetone toxicity data was available for rats, and thus we used the LOAEL and NOAEL reported for the rat as our benchmarks for the Great Blue Heron. Sample *et al.,* 1996 discussed a subchronic (90 days) study of the effects of acetone applied orally in three doses (100, 500, and 2,500 mg/kg/d) to rats weighing approximately 0.35 kg and consuming 0.028 kg of food per day. Doses of 500 and 2,500 mg/kg/d lead to significant kidney damage and increased kidney weights, but no differences were observed at the lowest dose. Because the exposure duration did not include the rat's reproductive lifestage, 100 and 500 mg/kg/d were considered to be subchronic doses, and were each divided by an uncertainty factor of 0.1 to estimate the chronic NOAEL and LOAEL of 10 and 50 mg/kg/d, respectively.

Chloromethane

Avian wildlife toxicity data for chloromethane (synonym: methyl chloride) was not available in Sample *et al.,* 1996. However, NOAELs and LOAELs for mammals were available, and we used the benchmarks identified in a chronic study (two years) of chloromethane toxicity to rats. Four doses of chloromethane (5.85, 50, 125, and 250 mg/kg/d) were administered orally to rats of approximately 0.35 kg in body weight. No effects were observed at the lowest dose, but changes in liver tissue were observed at 50/mg/kg/d. The chronic NOAEL and LOAEL were considered to be 5.85 and 50 mg/kg/d, respectively.

Chlorobenzene

There was no toxicological information for chlorobenzene available for avian or terrestrial wildlife in Sample *et al.,* 1996. However, toxicity studies for chlorobenzene were described in the Agency for Toxic Substances and Disease Registry (ATSDR) toxicity profile for chlorobenzene (1999). In a chronic study of rats orally exposed to chlorobenzene, altered kidney weights were observed at the dose of 120 mg/kg/d. No effects were observed at 60 mg/kg/d. These doses were the lowest LOAEL and NOAEL reported in the ATSDR profile. We divided these values by an uncertainty factor of 10 to estimate the LOAEL and NOAEL for the great blue heron.

1.2-Dichlorobenzene and 1.4-Dichlorobenzene

There was no available toxicity information about the effects of 1,2- and 1,4 dichlorobenzene on avian or terrestrial wildlife in Sample *et al.,* 1996. However, toxicological data for 1 ,4-dichlorobenzene was available in the ATSDR toxicity profile for 1 ,4-dichlorobenzene (1999). Moderate kidney damage was observed in male rats exposed to 150 mg/kg/d by gavage for 2 years in a chronic (2 years) study of the effects of 1,4 dichlorobenzene on rats. This dose was the lowest reported chronic value in the ATSDR toxicological profile. We divided this value by an uncertainty factor of 10 to estimate the LOAELs for 1,2- and 1,4-dichlorobenzene. Because a NOAEL for this particular effect was not estimated and NOAELs for other effects were higher than 150 mg/kg/d in the studies described in the ATSDR toxicological profile, we used the NOAEL reported for chlorobenzene described above.

Rutylbenzylphthalate

There were no data regarding the toxicity of butylbenzylphthalate to wildlife. However, toxicological information about the similar compound di-n-butylphthalate was identified in Sample *et al.,* 1996, and we used the identified LOAEL and NOAEL for di-nbutyl phthalate as surrogate benchmarks for butylbenzylphthalate. One dose of 10 mg of di-nbutylphthalate per kg of food administered via diet to ringed doves during a four- week period reduced eggshell thickness and water permeability of the shell. A LOAEL of 1.1 /kg/d was calculated based on the dose applied, and the body weight (0.155 kg) and food consumption rate (0.01727 kg/d) of ringed doves. Because the exposure period included the critical reproduction lifestage, this value was considered to be the chronic LOAEL, and the chronic NOAEL was estimated by multiplying the chronic LOAEL by the uncertainty factor of 0.1.

Ren7.n(a)pyrene and Other PAHs

Toxicity studies of benzo(a)pyrene, benzo(a)anthracene, and benzo(b)fluoranthene to avian wildlife were not available. However, there was information regarding the toxicity of benzo(a)pyrene to mammals in Sample *et al.,* 1996, and we used the test species LOAEL and NOAEL each divided by an uncertainty factor of 10 for the purpose of this food web. Three doses of benzo(a)pyrene (10, 40, and 160 mg/kd/d) was administered orally to mice. The dose of 10 mg/kg/d induced reduction of weight and fertility impairment among offspring. Because the exposure period included the reproduction lifestage, 10 mg/kg/d was considered to be the chronic LOAEL, and the chronic NOAEL of 1 mg/kg/d was estimated by multiplying the LOAEL by an uncertainty factor of 0.1.

Aldrin

Toxicological information regarding the effects of aldrin on avian wildlife were not identified. However, because aldrin quickly breaks down to dieldrin in the body and in the environment (ATSDR, 1989) we used the RfDs for dieldrin presented in Sample *et al.* (1996). Sample *et al.* summarized a study of the effects of orally administered dieldrin to the Barn owl. The single dose applied, 0.58 mg/kg, reduced eggshell thickness but did not have a significant effect on the number of eggs laid, the number of eggs hatched, the percent of eggs broken, or embryo or nestling mortality. Sample *et al.* considered this exposure level to be a No Observed Effects Level (NOAEL) and converted it to a RFD of 0.077 mg/kg/day using a literature derived body weight and food consumption rate.

Benzene Hexachloride (BHC)

Sample *et al.* (1996) reviewed the wildlife toxicity of mixed isomers of Benzene hexachloride (BHC). When exposed to 20 mg BHC/ kg food over a 90 day period, Japanese quail experienced reduced egg hatchability and egg volume. No significant effects were observed at dietary levels of 1 or 5 mg/kg. Sample *et al.* calculated a NOAEL and a Lowest Observed Effects Level (LOAEL) for birds of 0.563 mg/kg/day and 2.25 mg/kg/day from the 5 mg/kg and 20 mg/kg exposure levels, respectively. These calculations were conducted using quail body weights reported in the original study and food consumption rates estimated using an allometric equation.

Polychlorinated Biphenyls (PCBs)

Sample *et al.* (1996) reviewed the wildlife toxicity of mixed isomers of Aroclor 1242. Neither fertility or hatching success were significantly reduced when Screech owls were exposed to 3 mg/kg in diet over a period of two generations. Sample *et al.* considered this exposure level to be a NOAEL for birds and calculated a NOAEL RFD of 0.41 mg/kg/day using literature derived body weights and food consumption rates for the owl.

Sample *et al.* (1996) also summarize a 17-week study of Ring-necked pheasant exposure to Aroclor 1254 via weekly oral doses in gelatin capsules. The lowest test dose, 12.5 mg/bird/week, significantly reduced egg hatchability and was considered to be a LOAEL. Sample *et al.* (1996) calculated a LOAEL dose of 1.8 mg/kg/day using a literature derived body weight and assuming that the weekly dose could be divided by 7 to estimate a daily dose.

Because RfDs for Aroclor 1232 could not be identified, we applied the RfDs for Aroclor 1254. This should be a conservative assumption because of the higher chlorine content of Aroclor 1254 and the perceived relationship between PCB chlorine content and wildlife toxicity.

DDT and Metabolites (DDD and DDE)

Sample *et al.* (1996) summarized a study that documented the reproductive success of pelicans exposed to DDT over a 5 year period. The pelicans were exposed to DDT via consumption of anchovies, which were monitored for DDT concentrations over the course of the study. The lowest anchovy DDT concentration documented, 0.015 mg/kg (wet weight), was coincident with a reproductive success rate 30 percent below that required to maintain a stable population. Both Sample *et al.* and EPA (as cited in Sample *et al.}* have considered this exposure level to be a chronic LOAEL. Sample *et al.* estimated a chronic NOAEL by multiplying the LOAEL by an uncertainty factor of 0.1. These exposure levels were converted to NOAEL and LOAEL RfDs of 0.0028 mg/kg/day and 0.028 mg/kg/day using literature derived pelican body weights and food consumption rates. It should be noted that although DDD and DDE were not mentioned in the summary of this study provided by Sample *et al.* (1996). These compounds would be expected to occur along with DDT in the anchovy tissue, and would likely have contributed to the observed toxicity to pelicans. Therefore, the RfDs based on this study were applied to each DDT residue individually, but were also considered to be applicable to the summed total of DDT residues (DDTR).

Total Chlordanes

Sample *et al.* (1996) summarized a study that documented adverse impacts to redwinged blackbirds exposed to three dietary levels of chlordane. The study documented a 26 percent mortality in test animals over a 10 week exposure period at an exposure level of 50 mg/kg. No adverse effects were observed in test animals at an exposure level of 10 mg/kg. Sample *et al.* considered the 10 mg/kg exposure to be a NOAEL and the 50 mg/kg exposure to be a LOAEL. These values were converted to NOAEL and LOAEL RfDs of 2.14 mg/kg/day and 10.7 mg/kg/day using the body weight of the birds reported in the original study and an allometric equation to estimate a food consumption rate.

Total Endosulfans

Sample *et al.* (1996) summarized a study that failed to document adverse impacts to gray partridge exposed to three dietary levels (5, 25, and 125 ppm) of Endosulfan for 4 weeks during a life stage critical to reproduction. The maximum dose applied was considered to be
a chronic LOAEL, and was converted to a RfD of 10 mg/kg/day using the body weight of the birds reported in the original study and an allometric equation to estimate a food consumption rate.

Methoxychlor

EXTOXNET (1996) cites a study which reported that dietary levels of methoxychlor as high as about 145 mg/kg/day had no effects on reproductive function of male and female chickens over 8 to 16 weeks. We conservatively divided this value by an uncertainty factor of 10 to estimate a NOAEL of 14.5 mg/kg/day.

3.20 INORGANIC CONTAMINANTS

Aluminum

A chronic (four month critical lifestage) study of silver exposed ringed doves was described in Sample *et al.,* 1996. A single dose of 1000 ppm of aluminum was administered orally to ring doves via diet, and no effects were observed. Based on the food consumption rate (0.017 kg/d) and body weight (0.155 kg), a chronic NOAEL was estimated to be 109.7 mg/kg/d.

Arsenic

Sample *et al.* (1996) summarized a chronic (7 months) study of cowbirds exposed to arsenic in the form of copper acetoarsenite (approximately 44% arsenic). Four doses (25, 75, 225, and 675 ppm) were administered orally via the diet to cowbirds. Twenty percent mortality was observed in the cowbird group exposed to 75 ppm, and no observed effects were observed at 25 ppm. Based on body weight (0.049 kg) and food consumption rate (0.01087 kg/d), the chronic LOAEL and NOAEL were calculated at 7.38 mg/kg/d and 2.46 mg/kg/d, respectively.

Barium

Sample *et al.* (1996) described a subchronic study of 1-day old chicks exposed to eight doses (250, 500, 100, 200, 4000, 8000, 16,000, and 32,000 ppm) of barium hydroxide. Five percent mortality was observed in the group exposed to 4000 ppm, and barium exposures up to 2000 ppm lead to no significant effects. Based on body weight (0.121 kg) and food consumption rate (0.0126 kg/d), subchronic LOAEL and NOAEL were calculated at 416.53 and 208.26 mg/kg/d, respectively. Chronic LOAEL of 41.7 mg/kg/d and chronic NOAEL of 20.8 mg/kg/d were estimated by dividing the subchronic benchmarks by an uncertainty factor of 0.1.

Beryllium

Information regarding toxicity of beryllium to avian wildlife was not available in Sample *et al.*, 1996. However, beryllium toxicity to rats was described in Sample *et al.* (1996) and chronic NOAEL presented in this study were divided by an uncertainty factor of 10 to calculate this benchmark for the great blue heron. One dose of 5 ppm beryllium sulfate was administered to rats orally in water, and produced no significant effects. Because the study duration was greater than one year, this dose was considered to be the chronic NOAEL.

Cadmium

Sample *et al.* (1996) cited a chronic (90 day critical life stage) study which evaluated reproductive harm to mallard ducks exposed to cadmium chloride via diet. Body weights and food consumption rates were obtained from the original study. The LOAEL dose (210 mg/kg diet or 20.03 mg/kg/day) resulted in a significant reduction in egg-laying, while the NOAEL dose (15.2 mg/kg diet or 1.45 mg/kg/day) had no effect. Scheuhammer (1991) mentions a study that documented decreased egg production and eggshell thinning in chickens fed a diet containing 48 mg/kg cadmium.

Although the large interval between the NOAEL and LOAEL introduces some uncertainty, the study documented an ecologically relevant effect over a substantial exposure period. Based on these uncertainties, our confidence in these benchmark doses is moderate. It is reasonable to expect that exceedance of the LOAEL would elicit some effect on the local population of herons, and that doses below the NOAEL would have no effect.

Chromium

Sample *et al.* (1996) described a chronic study (ten months) of black ducks exposed to two doses (10 and 50 ppm) of trivalent copper in the diet. At 50 ppm, duckling survival was reduced, but no effects were observed at 10 ppm. Based on the body weight (1.25 kg) and food consumption rate (125 g food /day) of black ducks, and chronic LOAEL and NOAEL were calculated to be 5 mg/kg/d and Img/kg/d, respectively.

Manganese

Sample *et al.* (1996) summarized a chronic study (75 days) of manganese toxicity to 20 day old Japanese quails. One dose of 5000 ppm manganese was administered to the birds through the diet. Manganese consumption estimated at 977 mg/kg/d resulted in no significant adverse effects. Thus, this dose was considered to be the chronic NOAEL.

Mercury

For the purposes of this risk assessment, we assumed that all of the mercury ingested by the heron, including mercury associated with incidentally ingested sediment, would be in the form of methylmercury. Methylmercury is substantially more toxic and bioaccumulative than inorganic forms. Although nearly all (95-100%) of the mercury present in fish is methylmercury, obtained mostly from the diet, total concentrations of mercury in sediment, water, and biota in lower trophic levels (below fish) are not reliable predictors of methylmercury concentrations in fish (USGS, 1996). Generally the vast majority of mercury in an aquatic ecosystem is in the inorganic form (about 95 to 99%), and the vast majority of mercury in an aquatic ecosystem is found in the sediments (USGS, 1996).

Sample *et al.* (1996) summarized a study that documented reproductive impairment in mallard ducks exposed to 0.5 mg/kg methylmercury dicyandiamide in diet over three successive generations. Sample *et al.* considered this exposure level to be a LOAEL and derived a NOAEL by multiplying by an uncertainty factor of 0.1. Sample *et al.* converted these values to NOAEL and LOAEL RfDs of 0.0064 mg/kg/day and 0.064 mg/kg/day using a literature derived body weight and the food consumption rate reported in the original study.

Nickel

Sample *et al.* (1996) summarized a study that documented the effects of dietary nickel on Mallard ducklings exposed to three exposure levels for 90 days. Ducklings exposed to 1,069 mg/kg nickel experienced reduced growth and 70 percent mortality, while those exposed to 774 mg/kg did not experience adverse effects. Sample *et al.* considered the 1,069 exposure to be a LOAEL and the 774 mg/kg exposure to be a NOAEL. These values were converted to NOAEL and LOAEL RfDs of 77.4 mg/kg/day and 107 mg/kg/day using body weights reported in the original study and a literature derived food consumption rate.

Selenium

Sample *et al.* summarized several studies that documented adverse effects to avian receptors exposed to dietary selenium. We conservatively applied RfDs derived from the study that reported the lowest measured effects levels. This study reported a reduction in mallard duckling survival after exposure to 8 mg/kg dietary selenium over 100 days, with no adverse effects reported at a level of 4 mg/kg. Sample *et al.* considered 8 mg/kg to be a LOAEL and 4 mg/kg to be a NOAEL. These values were converted to NOAEL and LOAEL RfDs of 0.4 mg/kg/day and 0.8 mg/kg/day using body weights and food consumption rates reported in the original study.

Silver

There was no available information regarding silver toxicity to avian or terrestrial wildlife in Sample *et al.* (1996). However, in a review paper of silver toxicity and bioaccumulation by Ratte, 1999, silver toxicity to mammals was described. No observed adverse effects were observed to mice orally exposed to silver in the form of silver chloride at the dose of 18.1 mg/kg/d. This dose was the lowest NOAEL reported in Ratte (1999). Therefore, we divided the NOAEL for mice by an uncertainty factor of 10 to estimate to NOAEL for the great blue heron.

Thallium

Toxicological information regarding thallium exposure to avian wildlife was not available in Sample *et al.* (1996), but a subchronic (60 days) thallium exposure study to mammalian wildlife was described. One dose of 10 ppm thallium administered orally to rats resulted in reduced sperm motility. The mean daily intake of thallium was estimated to be 0.74 mg/kg/d, and this dose was considered to be the subchronic LOAEL. The chronic LOAEL of 0.074 mg/kg/d was estimated by multiplying the value by an uncertainty factor of 0.1, and the chronic NOAEL of 0.0074 mg/kg/d was estimated by multiplying the chronic LOAEL by 0.1.

Vanadium

A chronic (12 week) study of mallard ducks exposed to three doses (2.84, 10.36, 110 ppm) of vanadium in the diet was described by Sample *et al.* (1996). No adverse effects were observed at 110 ppm. Based on body weight (1.17 kg) and food consumption weight (0.121 kg/d), the chronic NOAEL was calculated to be 11.38 mg/kg/d.

Zinc

Sample *et al.* (1996) summarized a chronic study (44 weeks) of white leghorn hens exposed to three doses (20, 200, 2000 ppm) of supplemental zinc plus 28 ppm of zinc in the diet. Reduced egg hatchability was observed at 2028 ppm, but no adverse effects were observed at 48 and 228 ppm. Based on body weight and food consumption rate (1.935 kg and 123 g/d, respectively in the 228 ppm dose group, and 1.766 kg and 114 g/d, respectively in the 2028 dose group) the chronic NOAEL and LOAEL were calculated to be 14.5 mg/kg/d and 131 mg/kg/d, respectively.

4.00 RISK ESTIMATES FOR THE GREAT BLUE HERON

EPCs were converted to estimated daily doses using the exposure assumptions described in Section 2.00. Weighted EPCs are presented on Table 1-24,1-25, and 1-26. These estimated doses are presented on Tables 1-27 through 1-32. Estimated daily doses were compared to the toxicological RfDs (LOAELs and NOAELS) described above, and comparisons are presented on Tables 1-33 through 1-38. Comparisons are presented as Toxicity Quotients (TQs), which are simply the estimated dose divided by the RfD. TQs have been summed to yield Hazard Quotients (HQs); HQs were calculated separately for the organic contaminants and the inorganic contaminants, and also as a total HQ for the exposure point. Table 1-39 presents a summary of contaminants which exceeded their LOAELs or NOAELs, and presents those His which are greater than 1.

In evaluating exceedances of the RfDs, emphasis is given to exceedances of the LOAELs. LOAEL-based TQs greater than 1 indicate that the estimated dose exceeded a dose which has been shown to cause adverse effects to a test organism. Exceedance of a NOAEL, on the

other hand, indicates that the predicted exposure level is greater than the highest known "safe" level of exposure, but does not necessarily indicate that there is a significant level of risk. There are often great discrepancies and wide intervals between literature reported NOAELs and LOAELs. For instance, the LOAEL for heron exposure to cadmium (20.03 mg/kg/day) is nearly 14 times greater than the NOAEL of 1.45 mg/kg/day. Conversely, the LOAEL for heron exposure to nickel, 107 mg/kg/day, is only slightly higher than the NOAEL of 77.4 mg/kg/day. Our assessment places a higher degree of confidence in NOAELs which do not differ greatly from the original study's LOAEL.

Table 1-33 summarizes TQs for the CLF Drainage Area. Butylbenzylphthalate slightly exceeded the NOAEL, with a TQ of 1.93, but did not exceed the LOAEL. The pesticide DOT slightly exceeded the LOAEL-based RfD, with a TQ of 3.66. The LOAEL based TQ for total DDTR was 3.75. Beryllium slightly exceeded the NOAEL with a TQ of 1.33; there was no available LOAEL-based RfD for beryllium. The estimated dose of mercury slightly exceeded the NOAEL, with a TQ 4.74, but did not exceed the LOAEL. The estimated dose of thallium exceeded the LOAEL with a TQ of 7.36, and exceeded the NOAEL with a TQ of 73.6. The NOAEL and LOAEL-based total HQs were 122 and 12.4, respectively.

Table 1-34 summarizes TQs for the heron based on the assumption that the Upper Simmons Reservoir comprises the entire foraging range of a heron. None of the contaminants exceeded LOAEL RfDs. The LOAEL-based HQs for organic COPECs was less than one. Butylbenzylphthalate slightly exceeded the NOAEL-based RfD with a TQ of 3.57. Beryllium slightly exceeded its NOAEL with a TQ of 1.57. The NOAEL and LOAEL-based total HQs were 0.7 and 8.6, respectively.

To gauge the magnitude of difference the use of maximum sediment and surface water concentrations would make to the risk estimates for the heron, the heron food web model for the Upper Simmons Reservoir was re-run using maximum concentrations. In general, maximum-based TQs for individual COPECs were on the order on 50 percent to 4 times higher than the average-based TQs; the LOAEL-based and NOAEL-based total His calculated using maximums were about 2 times greater than those based on average COPEC concentrations. Use of maximum concentrations did not result in LOAEL exceedances by any of the individual COPEC, however the LOAEL-based total HI increased from 0.7 based on average concentrations to 1.8 based on maximum concentrations. Use of the maximum concentrations resulted in additional exceedances of the NOAEL by individual contaminants; these exceedances were by DDT (and DDTR), and thallium, with NOAEL-based TQs of 1.1 (2.0), and 5.8, respectively. The NOAEL-based total HQ increased from 8.6 based on average concentrations, to 21 based on maximum concentrations.

Table 1-35 summarizes TQs for the north basin of the Upper Simmons Reservoir. None of the contaminants exceeded LOAEL RfDs. NOAEL-based RfDs were slightly exceeded by butylbenzylphthalate, DDE, total DDTR, and thallium with TQs of 2.82, 1.20, 2.70, and 6.73 respectively. The total NOAEL and LOAEL-based HQs were 16.4 and 1.6, respectively.

Table 1-36 presents TQs based on the assumption that the Lower Simmons Reservoir comprises the entire foraging range of a heron. DOT, total DDTR, and thallium exceeded the LOAEL RfDs with TQs of 6.45, 6.54, and 12.8, respectively. Beryllium slightly exceeded its NOAEL with a TQ of 1.19. The NOAEL and LOAEL-based total HQs were 193 and 19.8, respectively.

Table 1-37 summarizes TQs for Sed Ponds 2&3 and Channels. Estimated doses of benzo(a)anthracene, benzo(a)pyrene, and mercury exceeded their RfDs with LOAEL TQs of 1.3, 3.1, and 4.01, respectively. The NOAEL and LOAEL-based total HQs were 90 and 8.7, respectively.

Table 1-38 summarizes TQs for Sed Pond 4. Butylbenzylphthalate was the only COPEC to exceed its RfDs: the estimated daily dose exceeded the LOAEL dose with a TQ of 1.4, and the NOAEL-TQ was 14.4.

5.00 RISK CHARACTERIZATION FOR THE GREAT BLUE HERON

The following paragraphs express the results of the food web analysis for the heron within the context of available lexicological information and contaminant distribution at the site. The intention of this section is to characterize and describe potential risks to receptors, and to provide a technical narrative supporting the risk estimates. Although sources of uncertainty in the risk estimate are mentioned throughout this risk description, more detailed discussions of uncertainties are presented in Section 6.00.

5.10 RISKS FROM DDTR

DOT and total DDTR were the only PCB and pesticide COPECs which resulted in estimated doses above their LOAELs. Estimated doses of DDT/DDTR exceeded the LOAEL for the entire CLF Drainage Area, however, this exceedance was the result of a 0.0001 mg/1 detection of DOT in a single unfiltered surface water sample (sample SW98-54) from the Lower Simmons Reservoir. Given the highly hydrophobic nature of DOT and the fact that DDT was not detected in Lower Simmons Reservoir sediment, it is likely that the detection was due to suspended matter in the sample. Although suspended solids were not detected in this sample at a MDL of 10 mg/1, if it is assumed that TSS is present at one-half the MDL the concentration of DDT associated with the suspended solids would only need to be 0.00002 mg/kg to account for the 0.0001 mg/1 detection. If this outlying surface water data point is not included in the input data set for the Gobas model, none of the sediment EPCs for DDT or DDTR are high enough to result in an exceedance of the LOAEL RfD.

Given the agricultural history of the USR and LSR watershed, and the pattern of detection for DDT, DDE, and DDD it is unlikely that DDTR present in these waterbodies resulted from Central Landfill. Although risks to the heron from ingestion of DDTR cannot be ruled out due to the single detection in sample SW98-54, it is unlikely that the landfill contributed to this risk estimate.

5.20 RISKS FROM MERCURY

The risk estimates suggest that herons may be exposed to potentially harmful dietary mercury concentrations while foraging at the sedimentation ponds, where estimated mercury doses exceeded the LOAEL RfD. As described in Section 3.20, the RfDs used for evaluation of heron exposure to mercury were based on a study in which methylmercury dicyandiamide was administered to mallard ducks, resulting in a significant reduction in reproductive success. It should be noted that mallards may possess less capability to detoxify methylmercury than other duck species, and piscivorous birds such as the heron likely possess a greater ability to detoxify methylmercury than do non-piscivorous birds like mallards (EPA, $+1997$ - Volume VI).

The Massachusetts Department of Environmental Protection (MA DEP) recently published a report documenting the sediment, water and fish tissue mercury concentrations found in 24 of the state's "least-impacted" waterbodies (MA DEP, 1997). Sediment mercury concentrations ranged from 0.029 mg/kg to 0.425 mg/kg (average $= 0.22$ mg/kg). The average fish tissue concentrations were 0.31 mg/kg (yellow perch), 0.40 mg/kg (largemouth bass), and 0.14 mg/kg (brown bullhead). The EPA Mercury Study Report to Congress (EPA, 1997 - Volume III) reported a nationwide mean bass tissue concentration of 0.38 mg/kg.

EPA (1997 - Volume VI) concluded that predatory wildlife, particularly piscivorous (fisheating) birds and mammals are potentially at risk from consumption of methylmercury in contaminated prey. The risks to wildlife were concluded to be greatest in regions receiving the highest levels of atmospheric mercury deposition, notably the Northeast.

The mean mercury concentrations detected in sediment samples for each of the exposure points evaluated in the heron risk characterization were within the range reported for relatively unimpacted waterbodies, and the fish tissue concentrations calculated for the exposure model are similar to tissue concentrations measured in fish from other freshwater lakes in the region. The risk estimate for heron exposure to mercury in fish is consistent with other risk estimates for piscivorous wildlife (e.g., EPA, 1997). Based on available information regarding regional contamination of waterbodies by atmospherically deposited mercury, it is unlikely that the risks to herons associated with consumption of mercury are related to the landfill.

5.30 RISKS FROM BUTYLBENZYLPHTHALATE, THALLIUM, BENZO(A)-ANTHRACENE, AND BENZO(A)PYRENE

Butylbenzylphthalate slightly exceeded its LOAEL in Sedimentation Pond 4 with a TQ of 1.44. However, as discussed in Section 4.0, there were no benchmarks identified for butylbenzylphthalate. Thus, we conservatively used the LOAEL-based RfD for di-n-butylyphthalate, which was the lowest reference dose for a phthalate based compound reported for avian wildlife in Sample *et al.,* 1996. Additionally, results from Sedimentation

Pond 4 were based on a single surface water sample. Because the exceedance of the LOAEL was less than a factor of two, we do not expect the potential for risk to the great blue heron in Sedimentation Pond 4.

The estimated daily dose for thallium exceeded the LOAEL in Lower Simmons Reservoir with a TQ of 12.8. However, there were no available thallium RfDs for avian wildlife. Thus, we used the LOAEL reported for the rat divided by an uncertainty factor of ten. Based on the uncertainty of this benchmark, we do not believe the exceedance of the LOAEL is indicative of potential for risk to the great blue heron in Lower Simmons Reservoir (See Section 6.00). We would not expect the actual LOAEL for avian wildlife to be comparable to the reference dose based on thallium toxicity to the physiologically dissimilar rat.

The RfDs used for both benzo(a)anthracene and benzo(a)pyrene, were based on a LOAEL for rats of lOmg/kg/day divided by an uncertainty factor of 10 to account for the extrapolation from mammals to birds. Estimated daily doses for these PAHs resulted in LOAEL TQs of just 1.3 and 3.1 in the Sed Ponds 2&3 and Stream Channels exposure area. Since the LOAEL TQs are less than 10, the use of mammal RfDs for birds presents a high degree of uncertainty, and the Sed Ponds 2&3 and Stream Channels exposure area consists of engineered waterbodies which are used to manage migration of sediments from the landfill facility, it is our opinion that these PAHs do not present a significant risk of harm to herons or similar birds which may feed within Sed Ponds 2&3 and Stream Channels.

6.00 UNCERTAINTIES

As mentioned above, we conservatively assumed that the total organic carbon (TOC) content of suspended solids (TSS) in each of the exposure points equaled the mean TOC content of the bottom sediment. Current scientific literature suggests that this assumption may underestimate the TSS TOC content by an order of magnitude (e.g., Campfens and Mackay, 1997). Table 1-40 presents an analysis of the effect of TSS TOC on body burdens predicted by the Gobas model. For this analysis, we used sediment and surface water data from the Upper Simmons Reservoir North Basin, and varied the TSS TOC input from the mean sediment fraction of 0.02065 to a TSS TOC input one order of magnitude higher (0.2065). The analysis was performed for Aroclor 1254, DDT, DDE, and ODD.

Assuming a higher TSS TOC content consistently results in significant decreases in predicted body burden concentrations and freely dissolved surface water concentrations. The decreased concentrations with increased TSS TOC are the result of greater predicted sorption to TSS. The contaminant with the highest log Kow, DDE, showed the greatest change with a change in TSS TOC. Fish tissue concentrations of DDT and its metabolites were approximately 6 to 8 times higher when the sediment TOC was used to estimate TSS TOC. The conservative uncertainty associated with this assumption may have resulted in an overestimation of potential risks to great blue heron populations.

As noted in Section 3.20, total concentrations of mercury in sediment, water, and biota in lower trophic levels (below fish) are not reliable predictors of methylmercury concentrations in fish (USGS, 1996). To reduce the possibility that prediction of tissue concentrations from sediment or water concentrations would underestimate field conditions, we made reasonably conservative assumptions regarding mercury bioconcentration. The predicted tissue concentrations of mercury were generally slightly higher than the detection limits used in the 1993 ESS study of mercury in the USR and LSR (see Appendix K), confirming that the bioconcentration calculations for mercury are unlikely to have underestimated fish tissue concentrations. However, based on available data regarding mercury distribution in the northeastern U.S., it is likely that fish tissue do contain some low level of methylmercury.

There is a high degree of uncertainty in the use of the reference dose based benchmarks for 1,2-dichlorobenzene, 1,4-dichlorobenzene, chlorobenzene, benzo(a)pyrene, benzo(a) anthracene, benzo(b)fluoranthene, beryllium, and thallium. These benchmarks reported in Sample *et al.* (1996) were derived from studies of toxicological effects to mammals using rat or mice as the test species. Interclass differences in physiological, biochemical, and behavioral factors including uptake, metabolism, and disposition would most likely result in significant differences in the toxicity of these contaminants between avian and mammalian species. Sample *et al.* (1996) stated that interclass extrapolation would carry a high degree of uncertainty, and was not performed for the purpose of deriving benchmarks for avian and mammalian wildlife receptors.

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SEDIMENTATION PONDS 2 AND 3 SEDIMENT SUMMARY OF ANALYTICAL DATA Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

 (ppm)

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SEDIMENTATION PONDS 2 AND 3 SEDIMENT SUMMARY OF ANALYTICAL DATA Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

(ppm)

Notes:

- 1. For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituents reported as non-detects (ND), and the method detection limit was used to represent the concentrations of constituents reported as "BMQL".
- 2. Analytical results were based on sediment samples: SED95-14, SED95-15, SED95-22, SED95-23, SED95-34, SED95-35, SED95-37, SED95-40, and SED95-41 collected in December 1995. SED 95-14 and SED 95-15 were collected from Cedar Swamp Brook, and SED95-22 and SED95-23 were collected from Quarry Stream.

2/9rt)1 TABLE 1-2

SEDIMENTATION PONDS 2 AND 3 SURFACE WATER SUMMARY OF ANALYTICAL DATA Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island (ppm)

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2/9/01 TABLE 1-2

SEDIMENTATION PONDS 2 AND 3 SURFACE WATER SUMMARY OF ANALYTICAL DATA Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island (ppm)

Notes:

1. For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituents reported as non-detects (ND), and the method detection limit was used to represent the concentrations of constituents reported as "BMQL".

2. Analytical results based on time averaged concentrations for surface water samples: SW95-40, SW95-41, SW95-34, SW95-35, and SW95-37.

SEDIMENTATION POND 4 SEDIMENT SUMMARY OF ANALYTICAL DATA Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

(ppm)

Notes:

1. For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituents reported as non-detects (ND), and the method detection limit was used to represent the concentrations of constituents reported as "BMQL".

2. Analytical results were based on sediment samples: SED95-24.

SEDIMENTATION POND 4 SURFACE WATER SUMMARY OF ANALYTICAL DATA Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island (ppm)

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SEDIMENTATION POND 4 SURFACE WATER SUMMARY OF ANALYTICAL DATA Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island (ppm)

Notes:

- 1. For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituents reported as non-detects (ND), and the method detection limit was used to represent the concentrations of constituents reported as "BMQL".
- 3. Analytical results based on surface water samples: SW95-24.

UPPER SIMMONS RESERVOIR SEDIMENT
SUMMARY OF ANALYTICAL DATA Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

 (ppm)

 \sqrt{QA} : AJ Date: 11/18/98

TABLE 1-5

UPPER SIMMONS RESERVOIR SEDIMENT SUMMARY OF ANALYTICAL DATA Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

(ppm)

Notes:

For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituents reported as non-detects (ND), and the method detection limit was used to represent the concentrations of constituents reported as "BMQL".

Analytical results were based on samples: SED93-21-1, SED93-23-I, SED93-21-O, SED93-22-O, SED93-23-O, SED93-24-O, SED93-25-O, SED93-2 SED93-28-O, SED93-29-ORE, SED93-30-O, SED93-31-O, SED95-42, SED95-43, SED98-50, SED98-51 and SED98-52.

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TABLE 1-6

UPPER SIMMONS RESERVOIR SURFACE WATER SUMMARY OF ANALYTICAL DATA (ppm) Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

UPPER SIMMONS RESERVOIR SURFACE WATER SUMMARY OF ANALYTICAL DATA (ppm) Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Notes:

1. For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituents reported as non-detects (ND), and the method detection limit was used to represent the concentrations of constituents reported as "BMQL".

3. Analytical results based on surface water samples: SW95-04, SW95-05, SW95-06, SW95-07, SW95-08, SW95-09, SW95-42, and SW95-43 collected on December 15,1995, and October 3,1996, and SW98-50, SW98-51 and SW98-52 collected on May 27, 1998.

TABLE 1-7

NORTH BASIN SEDIMENT
SUMMARY OF ANALYTICAL DATA (ppm)
Central Landfall - OU2 Remodial Investigation
Johnston, Rhode Island

Notes:

 \pm For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituence of constituence of constituence of constituence of constitue

2 Analytical results were based on samples: SFD93-21-I, SED93-23-I, SED95-08, SED95-42, SED95-43, and SED98-51, and SED98-52

3. Multiple samples collected from the same location were averaged together to obtain an averaged concentration for that location. These
concentrations were used in the calculation of the averages presented above

4. Concentrations presented for organic contaminants (VOCs, SVOCs and Pesticides) have been normalized to the total organic carbon.
(TOC) content of the sediment sample using the following formula; Contaminant (markg)/TOC

NORTH BASIN SURFACE WATER SUMMARY OF ANALYTICAL DATA (ppm) Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

NORTH BASIN SURFACE WATER SUMMARY OF ANALYTICAL DATA (ppm) Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Notes:

1. For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituents reported as non-detects (ND), and the method detection limit was used to represent the concentrations of constituents reported as "BMQL".

3. Analytical results based on surface water samples: SW95-04, SW95-05, SW95-06, SW95-07, SW95-08, SW95-09, SW95-42, and SW95-43 SW98-50, SW98-51 and SW98-52.

File No. 31864-00 Page 1 of 1
2/9/01

TABLE 1-9

LOWER SIMMONS RESERVOIR SEDIMENT SUMMARY OF ANALYTICAL DATA (ppm) Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Notes:

1. For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituents reported as non-detects (ND), and the method detection limit was used to represent the concentrations of constituents reported as "BMQL".

2. Analytical results were based on samples: SED95-01, SED95-02, and SED95-03, SED98-53, and SED98-54.

3. Concentrations presented for organic contaminants (VOCs, SVOCs and Pesticides) have been normalized to the total organic carbon G 191864 223311864-00 LICYCLOC CONDENT of the sediment sample using the following formula; Contaminant (mg/kg)/ TOC (mg/kg) x 10⁶ (mg/kg) = G 191864 223311864-00 LICYCALCSECO_TABV230esis xisSED_all SUM

2/9/01 **Page I ofl** File No. 31864.00

TABLE 1-10

LOWER SIMMONS RESERVOR SURFACE WATER SUMMARY OF ANALYTICAL DATA (ppm) Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Notes:

1. For the purpose of calculating arithmetic mean concentrations, one-half the method detection limit was used to represent the concentrations of constituents reported as non-detects (ND), and the method detection limit was used to represent the concentrations of constituents reported as "BMQL".

2 If a location was sampled more than once, the summary statistics are based on the average concentration over time at that location.

3. Analytical results based on surface water samples: SW95-01, SW95-02, SW95-03, SW98-53, and SW98-54

TABLE 1-11

PHYSICAL AND CHEMICAL INPUT PARAMETERS FOR GOBAS BIOACCUMULATION MODEL
Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Notes.

1. EPA, 1986. Superfund Public Health Evaluation Manual. EPA/540/1-86/060. Exhibit A-1
2. ATSDR (1989), Toxicological Profile for Selected PCBs (Aroclor-1260, -1254, -1248,

2. ATSDR (1989), Toxicological Profile for Selected PCBs (Arochor-1260, -1279, -1249, -1281, -1282, -1282, -1281, and -1016).

3. EPA, July, 1998. Ambient Water Quality Criteria Derivation Methodology Human

Health - Tech

S. RPA July 1998. Human Health Risk Assessment Protocol for Hazardous Waste
Combustion Facilities - Volume Two [Peer Review Draft]. Office of Solid Waste and
Emergency Response. EPA530-D-98-001B.

6. Oak Ridge National Laboratory, (1998).

No. New Number 1 Information System contains updated toxicity values from IRIS and
HEAST (http://risk.lad.ornl.gov/kox/tox_values.html)
7. Eisler, R. 1986. Polychlorinated biphenyl hazards to fish, wildlife, and invertebra

8. Because a Henry's Law Constant could not be identified for Aroclor 1232, we applied
the constant for Aroclor 1242.

File No. 31864.00
2/9/01

TABLE I-12

BIOLOGICAL INPUT PARAMETERS FOR GOBAS BIOACCUMULATION MODEL Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

1. Gobas, F.A.P.C. (1993).

2. EPA Wildlife Exposure Factors Handbook (1993).

Department of Biological Sciences, National University of Singapore, Lower Kent Ridge Road, Science Faculty, Singapore 3. Chironomidae lipid content presented at "http://www.science.nus.edu.sg/~webdbs/fish/livefood/bloodwm.html", 117600

4. EPA Exposure Factors Handbook (August, 1997).

5. Carlander (1977); Mean of means for 102-126 mm long specimens.

6. Carlander (1977); Predicted weight of 229 mm long specimen.

7. Carlander (1977); conservative estimates based on discussions in text

8. EPA Wildlife Exposure Factors Handbook (1993), conservative estimates based on discussion in text.

File No. 31864 z33
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TABLE 1-13

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SEDIMENTATION PONDS 2 AND 3
COBAS BIOACCUMULATION MODEL RESULTS
Central Landfill - OU2 Remedial Investigation
Johnston, Rhode Island

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TABLE 1-14

File No. 31864.223
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UPPER SIMMONS RESERVOIR
COBAS BIOACCUMULATION MODEL RESULTS
Central Landiil - OU2 Remedial Investigation
Central Landiston, Rhode Island

Pite No. 31864.z23
10/27/2000

TABLE 1-14

UPPER SIMMONS RESERVOIR
GOBAS BIOACCUMULATION MODEL RESULTS
Central Landifil - OU2 Remedial Investigation
Ahmston, Rhode Island

File No. 31864.223
1027/2000

TABLE 1-14

UPPER SIMMONS RESERVOIR
GOBAS BIOACCUMULATION MODEL RESULTS
Contai Landfili - OU2 Romodial Investigation
Johnsun, Rhode Island

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File No. 31864.23

TABLE I-15

UPPER SIMMONS RESERVOIR NORTH BASIN
GOBAS BIOACCUMULATION MODEL RESULTS
Central Landfill - OU2 Remedial Investigation
Johnston, Rhode Island

File No. 31864 23
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TABLE 1-15

UPPER SIMMONS RESERVOIR NORTH BASIN
GOBAS BIOACCUMULATION MODEL RESULTS
COBAS BIOACCUMULATION MODEL RESULTS
Central Landfill - OU2 Remedial Investigation

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TABLE I-15

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UPPER SIMMONS RESERVOIR NORTH BASIN
GOBAS BIOACCUMULATION MODEL RESULTS
Central Landfill - OU2 Remedial Investigation
Johnston, Rhode Island

File No. 31864.223
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LOWER SIMMONS RESERVOIR
GOBAS BIOACCUMULATION MODEL RESULTS
Centel Landfill - OU2 Remedial Investigation
Johnson, Rhode Island **TABLE 1-16**

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File No. 31864.z23
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TABLE I-17

${\large\bf SEDIMENTATION POND 4} \\ {\large\bf GOBAS BIOACCUMULATION MODEL RESULTS}$ \circ

CALCULATION OF SEDIMENT/INVERTEBRATE BIOCONCENTRATION FACTORS FOR HEAVY METALS Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

- 1. These values are "Bed sediment-sediment pore water partition coefficients" (L water/kg bottom sediment) presented in EPA530-D-98-001B (July, 1998), based on an average sediment pH of 6.79 and linear 'interpolation of values reported in the source document.
- 2a. These values are fish/surface water bioconcentration factors presented in EPA/540/1-86/060 (October, 1986).
- 2b. These values are fish/surface water bioconcentration factors presented in EPA 530-D-98-001B, July 1998.
- 2c. If BCF not available, we assumed a BCF of 1.
- 3. These values are conservatively predicted benthic invertebrate bioconcentration factors, calculated as BCF/Kd_{bs}

TABLE I-19

CALCULATION OF SEDIMENT/TADPOLE BIOCONCENTRATION FACTORS FOR HEAVY **METALS** Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Savannah River Site data, as summarized in Burger & Snodgrass, 1998.

SUMMARY OF SURFACE WATER/FISH BIOCONCENTRATION FACTORS FOR HEAVY METALS Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

1 These values are fish/surface water bioconcentration factors presented in:

a. EPA 530-D-98-001B, July 1998.

b. EPA/540/1-86/060 (October, 1986).

2 If BCF not available, we assumed a BCF of 1.

g:\31864.z23\31864-00.1jc\calcs\eco tab\foodweb\Z2300epc.xls\fish BCF - water

CALCULATION OF SEDIMENT/FISH BIOCONCENTRATION FACTORS FOR HEAVY METALS Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Based on comparisons of median sediment and fish tissue concentrations reported in USGS Fact Sheet 105-98 9/1/1998 Organic Compounds and Trace Elements in Freshwater Streambed Sediment and Fish from the Puget Sound Basin by Dorene E. MacCoy and Robert W. Black The URL for this document is http://wwwdwatcm.wr.usgs.gov/pugt/fs. 105-98.html

WEIGHT ASSIGNMENTS FOR SURFACE WATER BODIES BASED ON RELATIVE SURFACE AREAS Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Notes:

- 1. The area reported for Sedimentation Basin No. 4 includes the approximate area of Quarry Stream.
- 2. The area reported for Sedimentation Basin No. 3 includes the approximate area of Cedar Swamp Brook.
- 3. The area reported for Sedimentation Basins Nos. 2 and 3 includes the approximate areas of Cedar Swamp Brookm and Quarry Stream.

SITE-SPECIFIC FISH BODY BURDENS FOR ZINC (mg/kg wet weight)

Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

These site specific fish body burdens for zinc are the average concentrations of zinc in fish tissue samples collected in 1993 and 1994 from Upper Simmons and Lower Simmons Reservoir (See Appendix K).

g:\31864.z23\31864-00.1jc\calcs\eco_tab\foodweb\Z2300cpc.xls\fish body burden

File No. 31864.00
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Page 1/6

TABLE I-24

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 $\begin{array}{c} \hline \end{array}$

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SUMMARY OF EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)
Central Landfil - OU2
Johnston, Rhode Island

QA: AQJ Date: 7/13/99

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SUMMARY OF EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)
Central Landfill - OU2
Johnston, Rhode Island

QA: AQJ Date: 7/13/99

File No. 31864.00
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File No. 31864.00
2/9/01
2/6 Page 3/6

TABLE 1-24

QA: AQJ Date: 7/13/99

1.69E-05

 $1.96E - 03$

DOK

 $4.50E-03$

 $\frac{1}{1}$ $\frac{1}{2}$ $\frac{1}{2}$

4.39E-04

9,20E-06

2.16E-03

File No. 31864.00
29/01
Page 4/6

TABLE 1-24

SUMMARY OF EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm) Johnston, Rhode Island Central Landfill - OU2

 $\ddot{}$ $1.71E+00⁻²$ $\overline{}$ $3.19E+00²$ 3.83E-02 2.00E-03 2.66E-02 4.43E-02 1.55E-03 1.60E+00 4.19E-02 1,36E-01 $1.27E-01$ 1.54E-01 Fish $\overline{\mathbf{c}}$ និត្ត \overline{a} \overline{a} \overline{a} $\overline{}$ Amphibians 1.71E+00 $2.00E-03$ 3.19E+00 1.55E-03 4.19E-02 1.54E-01 3.83E-02 2.66E-02 1.36E-01 4.43E-02 $1.27E-01$ 1.60E+00 **SC** និត្ត \blacksquare \sim $\ddot{ }$ $\ddot{}$ Sedimentation Pond Invertebrates 2.39E+00 2.31E+00 1.71E+00 3.19E+00 **Benthic** 4.19E-02 1.00E-02 7.47E-05 4.43E-02 1,55E-03 $1.82E + 01$ 1.54E-01 1.36E-01 **SC** និត្ត Arithmetic Mean Surface Water 3.40E-02 4.19E-02 3.50E-03 MCC
1.67E-03 8.55E-03 1,43E-02 5.80E-04 2.70E-03 L55E-03 3.83E-02 1.36E-01 χ $\overline{\mathbf{R}}$ g g Arithmetic Mean 8.40E+00 9.47E+03 $1.44E+02$ 3.20E+00 5.59E+03 $1.09E + 01$ Sediment 9.80E+01 1.00E-01 1.145+01 2.40E+01 **SC** χ **SC** និត្ត $2.65E+00^{2}$ $1.53E+00⁻²$ $1.65E+00⁻²$ $1.47E-01$ ² 3.39E+00² 2.65E-03 5.50E-02 4.91E-02 1.92E-02 6.54E-01 3.25E-02 6.30E-01 5.90E-02 1.70E-02 Fish χ **SS** w, $\ddot{ }$ Amphibians 1.65E+00 4.91E-02 1.53E+00 2.65E-03 5.50E-02 1,70E-02 $1.92E-02$ $1.475-01$ 3.25E-02 6.30E-01 6,54E-01 $2.65E+00$ 3.39E+00 5.90E-02 s sa \overline{a} \overline{z} $\ddot{ }$ $\ddot{}$ Sedimentation Ponds 2 and 3 Invertebrates $2.65E-03$ $6.74E+00$ 3.54E+00 $2.65E+00$ 1.53E+00 8.25E+00 3.25E-02 $70E+02$ 1.21E-01 2.83E-04 6.54E-01 3.20E-01 6.30E-01 Benthic 2.81E-01 es
S Arithmetic Mean Surface Water 1.53E+00 $1.175-03$ 1.20E-03 $3.27E-03$ 2.65E+00 3.00E-04 3.13E-03 2.03E-03 1.34E-03 5.50E-02 2.10E-04 2.65E-03 7.22E-02 6.30E-01 **g**
g Arithmetic Mean 1.45E+02
2.27E+00 2.42E+04 $6.73E+02$ 1.38E+04 1.44E+00 2.60E-01 3.19E+01 3.73E+01 1.74E-01 1.14E+01 2.24E+02 Sediment 2.51E+01 និត្ត Constituent Banium
Berylium
Cadmium
Chompanese
Copper
Malium
Malium
Sichendium
Sichendium
Sichendium
Sichendium Aluminum Arsenic Vetals $\sum_{i=1}^{n}$

g \31864.z23\31864.00-ljc\cales\ecotab\foodweb\Z2300epc.xls\EPC Summary - Heron

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TABLE 1-24

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SUMMARY OF EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)
Central Landfil - OU2
Johnston, Rhode Island

File No. 31864.00
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2/3 Page 6/6

TABLE I-24

SUMMARY OF EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)

Johnston, Rhode Island Central Landfill - OU2

Notes:

1. Values in italies were calculated as 0.5 * MDL.

2. These values were calculated by multiplying the measured or estimated dissolved surface water contaminant concentration by published BCFs for fish.

3. These values were calculated by multiplying the measured or sediment contaminant concentration by the IAFs presented on Table 18. 4. Tissue EPCs for organic compounds were calculated using the Gobas Food Web Model.

5. These values were calculated by multiplying the measured sediment contaminant concentration by the fish/sediment accumulation factors based on

dry weight presented on Table 15, and by the fraction of solid content of fish (0.2) to convert the fish body burden from dry weight to wet weight.

6. These values were calculated by multiplying the measured sediment contaminant concentration by the tadpole/sediment accumulation factors presented on Table 16. 7. The mean dissolve selenium concentration is presented for the Lower Simmons Reservoir because selenium was not detected in total metals analyses.

8. These values are site-specific fish body burdens based on the average wet weight concentration of zinc in fish tissue.

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TABLE I-25

SUMMARY OF WEIGHTED EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)
Central Landfil - OU2
Johnston, Rhode Island

File No 31864.00
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TABLE 1-25

SUMMARY OF WEIGHTED EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)
Central Landfil - OU2
Johnston, Rhode Island

Note:

1. Weighted EPCs were calculated by multiplying the water body-specific EPC by the ratio of the surface area of that water body to the contined surface area of all water bodies. Weighted EPCs for each water body were then

File No. 31864.00
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TABLE I-25

SUMMARY OF WEIGHTED EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)
Central Landfill - OU2
Johnston, Rhode Island

File No. 31864.00
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2990 Page 4/6

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TABLE 1-25

SUMMARY OF WEIGHTED EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)
Central Landfil - OU2
Johnston, Rhode Island

File No 31864.00
2/9/01
3/6 **Page** 5/6

TABLE 1-25

SUMMARY OF WEIGHTED EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)
Central Landfil - OU2
Johnston, Rhode Island

QA: AQJ Date: 7/13/99

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TABLE I-25

SUMMARY OF WEIGHTED EXPOSURE POINT CONCENTRATIONS FOR GREAT BLUE HERONS (ppm)
Central Landfill - OU2
Johnston, Rhode Island

File No. 31864.00
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Page 6/6

ENTIRE CENTRAL LANDFILL DRAINAGE AREA
FEEDING AREA NORMALIZED EPCs FOR THE GREAT BLUE HERON
Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Notes:

1. Results of this table are based on the assumption that the heron feeds throughout the CLF Drainage Area EPCs presented are weighted according to the size of the different exposure points within the CLF Drainage Area. Th

2. NCC = Not a Contaminant of Concern for this media.

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ENTIRE CENTRAL LANDFILL DRAINAGE AREA
CALCULATION OF DAILY DOSES FOR THE GREAT BLUE HERON
Centri Landfill - OU2 Remedial Investigation
Johnston, Riode Island

Contaminated Fraction of Fording Acro
Total Daly Water Hauke (1606-1604)
Total Daly Water Hauke (1606-1604)
Fraction Composed of Fair, and
Fraction Composed of Acro<mark>theams –</mark>
Fraction Incidentally Ingenied Sediment –
Frac

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 $\frac{1}{2} \left(\frac{1}{2} \right) \left(\frac$

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00 POLE PER

UPFER SIMMONS RESERVOIR
CALCULATION OF DAILY DOSES FOR GREAT BLUE HERON
Central Landfil - OU2 Remedial Investigation
Johnston, Rhode Island

Notes.

 $\begin{array}{c} 0 \ 16 \ 1993 \\ 0 \ 045 \ (EPA, 1993) \\ 0 \ 045 \ (EPA, 1993) \\ 0 \ 01 \ (EPA, 1993) \\ 0 \ 01 \ (EPA, 1993) \\ 0 \ 1 \ (EPA, 1993) \\ 0 \ 1 \ (EPA, 1993) \\ 0 \ 1 \end{array}$ Contamnated Fraction of Feeding Area –
Total Daily Food make (Qorgoday) =
Total Daily Food make (Qorgoday) =
Fraction Composed of Funchinan =
Fraction Gomposed of Amphithans =
Fraction Incodentally, Ingerted Sedment =
Fra

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UPPER SIMMONS RESERVOR NORTH BASIN
CALCULATION OF DAILY DOSES FOR GREAT BLUE HERON
Central Landfill - OU2 Remedial Investigation
Johnston, Rhode Island

Notes:

Comminated Fraction of Feeding Area =
Total Daily Waver Instale (g/kg-day) =
Total Daily Waver Instale (g/kg-day) =
Fraction Composed of Find
Praction Composed of Amphibrance
Fraction Incidentally Ingered Sediment =
Fract

 $\begin{array}{c} 1 \ 0 \ 18 \ \text{(FPA1 1993)} \\ 0 \ 045 \ \text{(FPA1 993)} \\ 0 \ 043 \ \text{(FPA1 993)} \\ 0 \ 18 \ \text{(FPA1 993)} \\ 0 \ 1 \ \text{(FPA1 994)} \\ 0 \ 1 \ \text{(FPA1 995)} \\ 0 \ 1 \ \text{(FPA1 995)} \end{array}$

File No. 31864.00
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LOWER SIMMONS RESERVOIR
CALCULATION OF DAILY DOSES FOR GREAT BLUE HERON
Central Landfill - OU2 Remedial Investigation
Johnston, Rhode Island

Notes:

Contaminated Fraction of Feeding Area -
Toul Daily Voact Inake (kg/kg-dey) =
Toul Daily Water Inake (kg/kg-dey) =
Fraction Composed of Fish =
Fraction Composed of Invertibulars =
Fraction Incidentally Ingested Sediment =
F

 $\begin{array}{c} 1 \\ 0.18 \text{ (EPA, 1993)} \\ 0.045 \text{ (EPA, 1993)} \\ 0.045 \text{ (EPA, 1993)} \\ 0.04 \text{ (EPA, 1993)} \\ 0.1 \text{ (EPA, 1993)} \\ 0.1 \text{ (EPA, 1993)} \\ 0.1 \text{ (EPA, 1993)} \\ 0.039 \text{ (EPA, 1993)} \end{array}$

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SEDIMENTATION POND 2 AND 3
CALCULATION OF DAILY DOSE FOR GREAT BLUE HERON
Central Landfill - OU2 Remedial Investigation
Johnston, Rhode Island

Notes:

Contaminated Fraction of Feeding Area =
Toul Daily Food Intake (kg/kg-day) =
Toul Daily Water Intake (kg/kg-day) =
Fraction Composed of Fish =
Fraction Composed of Invertebrates =
Fraction Incidentally. Ingested Sediment

 $\begin{array}{c} 1 \\ 0 \text{ is (EPA, 1993)} \\ 0.045 \text{ (EPA, 1993)} \\ 0.045 \text{ (EPA, 1993)} \\ 0.1 \text{ (EPA, 1993)} \\ 0.0 \text{ (EPA, 1993)} \\ 0.1 \text{ (EPA, 1993)} \\ 0.1 \text{ (EPA, 1993)} \end{array}$

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SEDIMENTATION POND 4
CALCULATION OF DAILY DOSES FOR GREAT BLUE HERON
Central Landfill - OU2 Remedial Investigation
Johnston, Rhode Island

Notes

 $\begin{array}{c} 1 \ 0 \ 18 \ \text{(FFA)} \ 993) \\ 0.043 \ \text{(FFA)} \ 1993) \\ 0.043 \ \text{(FFA)} \ 1993) \\ 0.01 \ \text{(FFA)} \ 993) \\ 0.1 \ \text{(FFA)} \ 993) \\ 0.1 \ \text{(FFA)} \ 993) \\ 0.1 \ \text{(FFA)} \ 993) \end{array}$

 $\ddot{}$

ENTRE CENTRAL LANDFILL DRAINAGE AREA

OREAT BLUE HERON

ESTIMATED DAILY DOSES COMPARED TO TOXICOLOGICAL REFERENCE DOSES

Central Landfill - OU2 Remedial Investigation

Johnston, Rhode Island

Johnston, Rhode Island

Notes

-
-
- 1. Except where noted, toxicological benchmark doies were obtained from Oak Ridge National Laboratory

(Sample et al., 1996).

2. The toxicity of aldrin was assumed to equal dieldrin as per ATSDR.

3. The toxicity of Arco
	-
	-
-
-
-
-
- presented in Ratte, 1999.

6. Because there were no available benchmarks for birds, we used the LOAEL and NOAEL reported for the rat

in Sample et al., 1996, divided by an uncertaintly factor of 10.

7. Because there were
-
-

UPPER SUGAONS RESERVOIR
GREAT BLUE HERON
ESTIMATED DAILY DOSES COMPARED TO TOXCOLOGICAL REPERENCE IOSES
Central Landfill - OLD Remedial Investigation
Johnston, Rhode Island

Notes

1. Except where noted, toxicological benchmark dones were obtained from Oak Ridge National Laboratory (Sample et al., 199

2. The toxicity of Aroclor 1232 was assumed to equal dielebria as per ATSDR.

3. The toxicity of A

- - -

TABLE I-X

UPPER SIMMONS RESERVOR NORTH BASIN
GREAT BLUE HERON
ESTIMATED DAILY DOSES COMPARED IN TOXICOLOGICAL REFERENCE DOSES
Control Landini - OUR Remediat Investigation
Johnnion, Rhode Island

Notes

1 Except where noted, toxicological benchmark doesn were obtained from Oak Ridge National Laboratory (Sample et a

2. The toxisty of Adviator was assumed to equal dieldran as per ATSDR

3. The toxisty of Aveclor 1232 was

LOWER SDAMONS RESERVOR
GREAT BLUE HERON
ESTIMATED DAILY DOSES COMPARED TO TOXICOLOGICAL REFERENCE DOSES
Central Landfall - OU Remedia linestigation
Manuton, Rhode Island
Manuton, Rhode Island

Notes

-
-
-
-
- 1. Except where noted, toxicological benchmark doses were obtained from Oak Ridge National Laboratory (Sample et al., 2. The toxicity of Aroclor 1252 was assumed to equal Aroclor 1254.

3. The toxicity of Aroclor 1232 was
-

TABLE L37

SEDDIMENTATION PONDS 2 AND 3
GREAT BLUE HERON
ESTIMATED DAILY DOSES COMPARED ITO TOXICOLOGICAL REFERENCE DOSES
Central Landáti - OLI2 Remedial Investigation
Johnston, Rhode Island

Notes:

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-

1. Except where noted, toxicological benchmark doses were obtained from Oak Rulge National Laboratory (Sample et a

2. The toxicity of Atocho was assumed to equal dieldrin as per ATSDR.

3. The toxicity of Atocho 1252 was

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 $\zeta_{\rm{max}}$

TABLE I-38

SEDIMENTATION POND 4 **GREAT BLUE HERON** ESTIMATED DAILY DOSES COMPARED TO TOXICOLOGICAL REFERENCE DOSES Central Landfill - OU2 Remedial Investigation Johnston, Rhode Island

Notes:

- 1. Except where noted, toxicological benchmark doses were obtained from Oak Ridge National Laboratory (Sample et al., 1996).
- 2. The toxicity of aldrin was assumed to equal dieldrin as per ATSDR
- 3. The toxicity of Aroclor 1232 was assumed to equal Aroclor 1254.
- 4. This toxicological benchmark dose was obtained from EXTOXNET, and divided by an uncertainty factor of 10.
- 5. This toxicological benchmark dose is the NOAEL reported from a chronic study of silver exposed mice, presented in Ratte, 1999.
- 6. Because there were no available benchmarks for birds, we used the LOAEL and NOAEL reported for the rat
- in Sample et al., 1996, divided by an uncertaintly factor of 10.
- 7. Because there were no available benchmarks for these PAHs we used
	- the NOAEL and LOAEL for benzo(a)pyrene reported in Sample et al., 1996.
- 8. Because there were no available benchmarks for butylbenzylphthalate we used the benchmark for Di-N-butylphthalate.

File No. 31864-00
Page 1 of 2
2/9/01

TABLE 1-39

GREAT BLUE HERON FOOD WEB ASSESSMENT
SUMMARY OF TOXICITY QUOTIENTS AND TOTAL HAZARD QUOTIENTS
Central Landfill - OU2 Remedial Investigation
Johnston, Rhode Island

Notes:

1. The LOEL and NOEL Toxicity Quotients (TQ) are the Estimated Daily Dose for the contaminant divided by the LOEL and NOEL benchmark, resepectively.

- 2. The Total Hazard Quotient (HQ) for organics is the sum of the individual toxicity quotients for organic COCs; Total Hazard Quotient for inorganics is the sum of the individual toxicity quotients for inorganic COCs; and
- 3. Only exceedances (Toxicity Quotients or Hazard Quotients that are greater than one) are presented. The Hazard Quotients may be greater than the TQs less than 1 are included in the HQs.
- 4. The Total Hazard Quotient (HQ) for organics do not include the individual toxicity quotient for Total DDTR,
- 5. Total DDTR is the sum of DDE, DDD, and DDT.

File No. 31864.00
2/9/01

TABLE 1-40

EFFECT OF TOTAL ORGANIC CARBON CONTENT IN TOTAL SUSPENDED SOLIDS
ON CALCULATED TISSUE CONTAMINANT CONCENTRATIONS
Upper Simmons Reservoir Delta
Central Landfill - OU2 Remedial Investigation
Central Landfill - OU2 Remedial I

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