APPENDIX I

GROUNDWATER LABORATORY ANALYTICAL REPORTS

APPENDIX J

STATE AND FEDERAL LISTED THREATENED AND ENDANGERED SPECIES

North Florida Field Office

Manatee County Federally Listed Species

MANATEE COUNTY

93 Total Elements Found
Last Updated: April 2005

Fish

EXPLANATION

Scientific Name	Common Name	Global Rank	State Rank	Federal Status	State Status
Microphis brachyurus	Opossum Pipefish	G4G5	S2	SC	N
Rivulus marmoratus	Mangrove Rivulus	G3	S3	C	LS

Amphibians

EXPLANATION

Scientific Name	Common Name	Global

Crotalus adamanteus	Eastern Diamondback Rattlesnake	G4	S3	N	N
Dermochelys coriacea	Leatherback	G2	S2	LE	LE
Drymarchon couperi					

Athene cunicularia floridana	Florida Burrowing Owl	G4T3	S3	N	LS
Buteo brachyurus					
			S2	LT	LT
Charadrius alexa5949 0.9059 rg 43.5 633.758					

( ) Tj ET q 451.5 6

Falco r

Falco sparverius paulus	Southeastern American Kestrel	G5T4	S3	N	LT
Fregata magnificens	Magnificent Frigatebird	G5	S1	N	N

Rynchops niger	Black Skimmer	G5	S3	N	LS
Sterna antillarum					
	Caspian Tern	G5	S2	N	N
Sterna maxima	Royal Tern	G5	S3	N	N
Sterna sandvicensis	Sandwich Tern	G5	S2	N	N

Scientific Name	Common Name	Global Rank	State Rank	Federal Status	State Status
Acrostichum aureum					

FNAI STATE RANK DEFINITIONS

S1 = Critically imperiled in Florida because of extreme rarity (5 or fewer occurrences or less than 1000 individuals) or because of extreme vulnerability to extinction due to some natural or man-made factor.

S2 = Imperiled in Florida because of rarity (6 to 20 occurrences or less than 3000 individuals) or because of vulnerability to extinction due to some natural or man-made factor.

S3 = Either very rare and local in Florida (21-100 occurrences or less than 10,000 individuals) or found locally in a restricted range or vulnerable to extinction from other factors.

S4 = Apparently secure in Florida (may be rare in parts of range).

S5 = Demonstrably secure in Florida.

SH = Of historical occurrence in Florida, possibly extirpated, but may be rediscovered (e.g., ivory-billed woodpecker).

SX = Believed to be extirpated throughout Florida.

SU = Unrankable; due to a lack of information no rank or range can be assigned.

SNA = State ranking is not applicable because the element is not a suitable target for conservation (e.g. a hybrid species).

SNR = Element not yet ranked (temporary).

STATE LEGAL STATUS

Provided by FNAI for information only.
For official definitions and lists of protected species, consult the relevant federal agency.

Animals: Definitions derived from "Florida's Endangered Species and Species of Special Concern, Official Lists" published by Florida Fish and Wildlife Conservation Commission, 1 August 1997, and subsequent updates.

LE Endangered: species, subspecies, or isolated population so few or depleted in number or so restricted in range that it is in imminent danger of extinction.
LT Threatened: species, subspecies, or isolated population facing a very high risk of extinction in the future.

LS

APPENDIX K

TOXICITY PROFILES AND CHEMICAL DATA SHEETS

Toxicity Summary for COPPER

Prepared by: Rosmarie A. Faust, Ph.D., Chemical Hazard Evaluation and Communication Group, Biomedical and Environmental Information Analysis Section, Health and Safety Research Division, *, Oak Ridge, Tennessee.

Prepared for: Oak Ridge Reservation Environmental Restoration Program.

*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under Contract No. DE-AC05-84OR21400.

Copper occurs naturally in elemental form and as a component of many minerals. Because of its high electrical and thermal conductivity, it is widely used in the manufacture of electrical equipment. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH, 1986). Copper can be absorbed by the oral,

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Toxicity Summary for LEAD

December 1994

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Prepared for OAK RIDGE RESERVATION ENVIRONMENTAL RESTORATION PROGRAM.

*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under contract No. DE-AC05-84OR21400.

Lead occurs naturally as a sulfide in galena. It is a soft, bluish-white, silvery gray, malleable metal with a melting point of 327.5C. Elemental lead reacts with hot boiling acids and is attacked by pure water. The solubility of lead salts in water varies from insoluble to soluble depending on the type of salt (IARC, 1980; Goyer, 1988; Budavari et al., 1989).

Although similar effects occur in adults and children, children are more sensitive to lead exposure than are adults. Irreversible brain damage occurs at blood lead levels greater than or equal to 100 ug/dL in adults and at 80-100 ug/dL in children; death can occur at the same blood levels in

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745 21.75 0j 3.75 0 TD -0.0175 Tc 0.307 Tw (35.) Tj 16.5 0 TD 0 Tc 0.2895 Tw ( ) Tj -327.7S8 (Stol Tcf Tw (83/028

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Toxicity Summary for 1,1,2,2-TETRACHLOROETHANE

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Prepared for OAK RIDGE RESERVATION ENVIRONMENTAL RESTORATION PROGRAM

*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under Contract No. DE-AC05-84OR21400

1,1,2,2-Tetrachloroethane (CAS No. 79-34-5) is a two-carbon chain molecule with two chlorine atoms on each carbon atom. Uses of 1,1,2,2-tetrachloroethane have been as a chemical intermediate, industrial solvent, and extractant. 1,1,2,2-Tetrachloroethane was found on at least 278 of the hazardous waste sites on the United States Environmental Protection Agency's National Priorities List. Chemical degradation occurs by the loss of chlorine atoms, and the half-life of 1,1,2,2-

1977). Rats had a body weight loss of 38% for the males and 24% for the females after 6 weeks of 178 mg/kg/day but apparently recovered by the end of the 78-week treatment regiment (NCI 1978). At the 280 mg/kg/day dosage, rats died after 70 weeks. At the end of the 78 weeks of 284 mg/kg/day, male mice died of tubular nephrosis and female mice demonstrated hydronephrosis (NCI 1978). [These NCI (1978)dosages were time-weighted averages of the different doses given.]

Lethal exposure concentrations and exposure times for rats were approximately 1000 ppm after 4 to 6 hours (Carpenter et al. 1949, Deguchi 1972, Schmidt et al. 1980b, Smyth et al. 1969) and 5100;ppm after 30 minutes (Price et al. 1978). One of 10 rats exposed to 6300 ppm for 30 minutes exhibited myocardial damage (Price et al. 1978). Mice exposed to 600 ppm for 3 hours developed fatty changes in the liver (Tomokuni 1969, 1970; Hayrack et al. 1962). Exposure of rats to 130 ppm for 15 weeks resulted in increased liver weights, granulation and vacuolization of the liver, and liver hyperplasia (Truffert et al. 1977). Rabbits exposed to 15 ppm for 7 to 11 months exhibited signs of liver degeneration (Navrotskiy et al. 1971). One monkey exposed to a time-weighted average of 1974;ppm for 2 hours/day, 6 days/week for 9 months (no control) had transient diarrhea, anorexia, centrilobular vacuolization, and fatty degeneration of the liver (Hayrack et al. 1962).

The dermal LD_{50} value in rabbits was determined to be 6.36 g/kg (Smyth et al. 1969). Thickening of the cellular nucleus and pseudoeosinophilic infiltration was observed after dermal application of 514 mg/cm^2 for 16 hours on guinea pigs (Kronevi et al. 1981).

Army workers exposed to 1,1,2,2-tetrachloroethane vapor in a clothing processing plant had a very slight increase in death due to genital cancers, leukemia, or other lymphomas than workers not employed in a clothing plant (Norman et al. 1981). Male and female mice orally administered 142 and 284 mg/kg/day for 78 weeks had an increase in hepatocellular carcinomas (NCI 1978). Based on these results, 1,1,2,2-tetrachloroethane has been classified as Group C, possible human carcinogen (IRIS 2005).

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Toxicity Summary for ARSENIC

Prepared by: Dennis M. Opresko, Ph.D., Chemical Hazard Evaluation and Communication Group, Biomedical and Environmental Information Analysis Section, Health and Safety Research

hyperkeratosis and bile duct enlargement with hyperplasia, focal necrosis, and fibrosis (Baroni et al., 1963; Byron et al., 1967). Reduction in litter size, high male/female birth ratios, and fetotoxicity without significant fetal abnormalities occur following oral exposures (Schroeder and Mitchener, 1971; Hood et al., 1977; Baxley et al., 1981); however, parenteral dosing has resulted in exencephaly, encephaloceles, skeletal defects, and urogenital system abnormalities (Ferm and Carpenter, 1968; Hood and Bishop, 1972; Beaudoin, 1974; Burk and Beandoin, 1977).

Acute inhalation exposures to inorganic arsenic can damage mucous membranes, cause rhinitis, pharyngitis and laryngitis, and result in nasal septum perforation (U.S. EPA, 1984). Chronic inhalation exposures, as occurring in the workplace, can lead to rhino-pharyno-laryngitis, tracheobronchitis, (Lundgren, 1954); dermatitis, hyperpigmentation, and hyperkeratosis (Perry et al., 1948; Pinto and McGill, 1955); leukopenia (Kyle and Pease, 1965; Hine et al., 1977); peripheral nerve dysfunction as indicated by abnormal nerve conduction velocities (Feldman et al., 1979; Blom et al., 1985; Landau et al., 1977); and peripheral vascular disorders as indicated by Raynaud's syndrome and increased vasospastic reactivity in fingers exposed to low temperatures (Lagerkvist et al., 1986). Higher rates of cardiovascular disease have also been reported in some arsenic-

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Although U.S. EPA's Science Advisory Board recommended a weight-of-evidence classification of C-B2 continuum (C = possible human carcinogen; B2 = probable human carcinogen), the agency has not adopted a current position on the weight-of-evidence classification (IRIS, 2005).

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Toxicity Summary for TRICHLOROETHENE

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NTP (National Toxicology Program). 1988. Toxicology and Carcinogenesis Studies of Trichloroethylene (CAS No. 79-01-6) in Four Strains of Rats (ACI, August, Marshall, Osborne-Mendel) (Gavage Studies). U.S. Department of Health and Human Services, National Institutes of Health, Bethesda, MD. NTP TR 273.

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Toxicity Summary for 1,1-DICHLOROETHANE

Prepared by: Dennis M. Opresko, Ph.D., Chemical Hazard Evaluation Group, Biomedical and Environmental Information Analysis Section, Health Sciences Research Division, *

Prepared for: Oak Ridge Reservation Environmental Restoration Program.

*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under Contract No. DE-AC05-84OR21400.

1,1-Dichloroethane is used primarily as an intermediate in manufacturing vinyl chloride and 1,1,1-trichloroe1ATSDR, 1990) Program32 -D

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generally negative results (NTP, 1982; Quast et al., 1983; Maltoni et al., 1984, 1985). In one inhalation study (Maltoni et al., 1985), statistically significant increases in renal adenocarcinomas were noted in male Swiss mice exposed to 25 ppm for 12 months. Also observed were statistically significant increases in mammary gland carcinomas in females and lung tumors in both sexes. Results of other inhalation studies with rats, mice, and hamsters have been negative (Hong et al., 1981; Maltoni et al., 1984; Quast et al., 1986).

Based on EPA guidelines, 1,1-dichloroethylene was assigned to weight-of-evidence group C, possible human carcinogen (IRIS, 2005).

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Toxicity Summary for BENZO[A]PYRENE

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Prepared for: OAK RIDGE RES

benzo[*a*]pyrene induces skin tumors in several species, although mice appear to be the most sensitive species. Benzo[*a*]pyrene is a complete carcinogen and also an initiator of skin tumors (IARC, 1973; EPA, 1991). Benzo[*a*]pyrene has also been reported to induce tumors in animals when administered by other routes, such as intravenous, intraperitoneal, subcutaneous, intrapulmonary, and transplacental.

Based on United States Environmental Protection Agency (EPA) guidelines, benzo[*a*]pyrene was assigned to weight-of-evidence group B2, probable human carcinogen (IRIS, 2005).

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Toxicity Summary for *cis*- and *trans*-1,2-DICHLOROETHYLENE

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*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under contract No. DE-AC05-84OR21400.

1,2-Dichloroethene exists in two isomeric forms, *cis*-1,2-dichloroethene and *trans*-1,2-dichloroethene, that are colorless, volatile liquids with a slightly acrid odor. Although not used extensively in industry, 1,2-dichloroethene is used in the production of other chlorinated solvents and as a solvent for dyes, perfumes, and lacquers (Sax and Lewis 1989, Budavari et al. 1989). Humans are exposed to 1,2-dichloroethene primarily by inhalation, but exposure can also occur by oral and dermal routes.

Limited information exists on the absorption, distribution, and excretion of 1,2-dichloroethene in either humans or animals. In vitro studies have shown that the mixed function oxidases will metabolize 1,2-dichloroethene; the final metabolic products are dependent on the initial isomer of 1,2-dichloroethene (Costa and Ivanetich 1984, Henschler 1977, Liebman and Ortiz 1977).

Information on the toxicity of 1,2-dichloroethene in humans and animals is limited. Workers exposed to 1,2-dichloroethene have been reported to suffer from drowsiness, dizziness, nausea, fatigue, and eye irritation (ATSDR 1990). Acute and subchronic oral and inhalation animal studies of *trans*-1,2-dichloroethene and acute inhalation animal studies of *cis*-1,2-dichloroethene suggest that the liver is the primary target organ. The toxicity is expressed in increased activities of liver associated enzymes, fatty degeneration, and necrosis (McCauley et al. n.d., Barnes et al. 1985, Freundt et al. 1977). Secondary target organs include the central nervous system and lung.

No information was available concerning the chronic, developmental, or reproductive toxicity of *cis*-1,2-dichloroethene or *trans*-1,2-dichloroethene. No cancer bioassays or epidemiological studies were available to assess the carcinogenicity of 1,2-dichloroethene. EPA has placed both *cis*-1,2-dichloro-ethene and *trans*-1,2-dichloroethene in weight-of-evidence group D, not classifiable as to human carcinogenicity, based on the lack of human or animal carcinogenicity data and on essentially negative mutagenicity data

Freundt, K.J., G.P. Liebaldt and E. Lieberwirth. 1977. Toxicity

Toxicity Summary for DIBENZ[A,H]ANTHRACENE

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single subcutaneous injection of dibenz[*a,h*]anthracene induced local sarcomas and lung adenomas (Platt et al., 1990) and three intraperitoneal injections induced a high incidence of pulmonary tumors (Buening et al., 1979). A number of earlier studies have also demonstrated the carcinogenicity of dibenz[*a,h*]anthracene when administered by various parenteral routes in several animal species (IARC, 1973).

Based on no human data and sufficient evidence for carcinogenicity in animals, EPA has assigned dibenz[*a,h*]anthracene a weight-of-

IRIS (Integrated Risk Information System), 2005. United States National Library of Medicine, National Institute of Health, Department of Health and Human Services 8600 Rockville Pike, Bethesda, MD 20894. http://toxnet.nlm.nih.gov

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