RISK ASSESSMENT
FOR
JACKSONVILLE LANDFILL SITE
JACKSONVILLE, ARKANSAS

April 1990

DOCUMENT CONTROL NO. 09-RI1-RA-147

PREPARED BY

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REM VI
U.S. EPA CONTRACT NO. 68-01-7448
WORK ASSIGNMENT NO. 09-6LB6

PERFORMANCE OF REMEDIAL RESPONSE ACTIVITIES
AT UNCONTROLLED HAZARDOUS WASTE SITES

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1.0 EXECUTIVE SUMMARY

A remedial investigation (RI) was conducted at the Jacksonville Landfill, Jacksonville, Arkansas. Field studies took place from November, 1988, to May, 1989, and an RI report was subsequently prepared. In conjunction with the RI, a risk assessment (RA) was performed based upon the results of the investigation. This report documents the methodology, results and conclusions of the risk assessment. The methodology followed is based on the procedures contained in the EPA Superfund Public Health Evaluation Manual (1986) and the Superfund Exposure Assessment Manual (1988).

Using the above procedures, the health risks identified at the Jacksonville Landfill were determined based on the distribution and extent of chemical contamination, the potential for contaminant transport, opportunities for exposure and toxicity of the contaminants.

Risk assessment findings at the landfill indicate a potential health risk is predominantly associated with direct contact with or accidental ingestion of contaminated soil at the drum site ("hot spot") on the landfill. Specifically, the drum site contaminants cause excess carcinogenic health risks and noncarcinogenic health hazards. The excess carcinogenic risks calculated for 2,3,7,8-TCDD (dioxin) concentrations at the drum site ("hot spot") range from 6.47×10^{-4} for plausible exposure to 1.56×10^{-2} for worst possible exposure (based on the maximum concentration). "Plausible" or most likely exposure results are derived using arithmetic and geometric means of laboratory chemical analyses of field samples. Worst possible exposure values were calculated using the highest value for the laboratory chemical analyses of these samples. Total lifetime risk calculated for the drum site, including the dioxins,

furans and other carcinogens, is 1.48×10^{-3} for plausible exposure and 1.61×10^{-2} for worst possible exposure. These risks are limited to an extremely small area on the landfill property.

Analytical results indicate that 2,3,7,8-TCDD, as well as 2,4,5-T or 2,4,5-TP herbicide contamination is not present in any off-site residential areas above analytical detection limits. These detection limits ranged from 0.006 to 0.08 ηg/g. A hypothetical calculation was made to determine what additional risks would occur if dioxin existed below detection limits. Assuming that the concentrations in the residential areas average one-half of the detection limit, risks were calculated for an off-site 2 to 6 year old child and an adult gardener. Risks were 2.39×10^{-6} for a child and 2.71×10^{-6} for an adult gardener for plausible exposure from 2,3,7,8-TCDD. The 2 to 6 year old age group was chosen for detailed calculation because their exposure rate and physical characteristics (e.g., low body weight) represent a worst case situation for children. It should be stressed that off-site risks for 2,3,7,8-TCDD and the family of dioxins (including 2,3,7,8-TCDD) and furans are only hypothetical values since no 2,3,7,8-TCDD; 2,4,5-T or 2,4,5-TP was ever detected at these off-site residential backyards.

Total risk from all detected carcinogenic contaminants calculated for the off-site residential area is 9.65×10^{-5} for plausible exposure and 1.91×10^{-4} for worst possible exposure. The major contributor to this risk is arsenic in off-site areas. Risks calculated were 9.64×10^{-5} for plausible exposure and 1.91×10^{-4} for worst possible exposure to arsenic alone. These off-site risks are for a 2 to 6 year-old child. For an adult off-site gardener, the risk for arsenic alone is 8.61×10^{-6} and 1.70×10^{-5} for the worst case. Arsenic is fairly widely distributed on the landfill and off-site residential backyards. This substance, a common

component of soil, is known to exist at higher background concentrations in Central Arkansas than throughout the contiguous United States.

Arithmetic and geometric mean soil TCDD concentrations in soils at the landfill hot spot exceed the 1 ng/g limit recommended by the Centers for Disease Control (CDC) for TCDD in residential soil. At the hot spot, the 2,3,7,8-TCDD equivalent geometric and arithmetic mean concentrations due to all dioxins and furans, including 2,3,7,8-TCDD, were 18.0 ng/g and 38.7 ng/g, respectively.

The noncarcinogenic health hazards at the landfill drum site are related to the herbicides present in the drums. Hazard indices (HIs) calculated for 2,4,5-T and 2,4,5-TP at the drum site were large and range from 88.5 and 3.45, respectively, for plausible exposure, to 468 and 41.8, respectively, for worst possible exposure. Hazard indices greater than 1.00 indicate that chronic toxicity may occur in an exposed individual, for example, a teenager coming into contact with the drum contents or surrounding soils.

Hazard indices for off-site herbicide exposure were insignificant, as concentrations were nondetectable.

In conclusion, this risk assessment indicates that dioxin, herbicide and arsenic concentrations exceed criteria for excess lifetime cancer risks and/or health hazards. The later chemical is most probably a natural constituent of local soils. Plausible routes of exposure and a likely exposed population have been defined. Therefore, a potential health hazard exists.

2.0 INTRODUCTION

This document presents the risk assessment for the Jacksonville Landfill, Jacksonville, Arkansas. It outlines the type and degree of hazards posed by chemical contaminants, the extent to which a particular group of individuals have been or may be exposed to the chemicals, and the current or potential future health risk that exists at the Jacksonville Landfill. The assessment also serves as a baseline evaluation of the site under a "no-action" remedial alternative (i.e., in the absence of any remediation and assuming nonrestricted future site use). It will provide a basis for assessing remedial alternatives to be considered in the Feasibility Study.

This assessment has been conducted using conservative assumptions according to the general guidelines outlined in the EPA Superfund Public Health Evaluation Manual (1986) and the EPA Superfund Exposure Assessment Manual (1988) with consideration of changes contained in the December 1989 draft revisions to this guidance. It is based primarily on data gathered during the remedial investigation (RI), completed in 1989, that accompanies this report. The purpose of using conservative assumptions is to compensate for uncertainty and to protect the general population, even its most sensitive members, from increased risk of disease.

The steps involved in this assessment include identification of the extent of contamination in various media (hazard assessment) and comparison to standards, criteria and guidelines, called ARARs (applicable or relevant and appropriate requirements). This is presented in Section 3.0. Potential migration pathways for these chemicals are then assessed based on fate and transport properties of the chemicals and the characteristics of the site. In Section 4.0, potential human receptors for contaminants released through

migration pathways are then identified and dose rates of the chemicals in the receptors are calculated under current conditions (exposure assessment). In Section 5.0 the dose rates, together with their corresponding unit cancer risk values, are used to calculate the potential risks associated with the site in the absence of remediation (risk characterization). Refer to Section 5.2 for a further discussion of unit cancer risk (q^*) values. The excess cancer risks calculated are then compared to acceptable EPA risk levels to determine if the risk at the site falls within these levels. Noncarcinogenic health hazards are evaluated by comparing calculated dose rates to the respective reference dose (RfD) for the contaminant.

2.1 SITE LOCATION

The Jacksonville Landfill site is located in Lonoke County, outside the city limits of Jacksonville, Arkansas (Figures 2-1 and 2-2). It is situated south of Graham Road, one-tenth mile east of the Pulaski/Lonoke County Line. Land records at the Lonoke County Court House describe the eighty-acre plot of land as the west half of the northwest quarter of Section 27, Township 3 North, Range 10 West. The site is approximately 12 miles northeast of Little Rock, Arkansas.

2.2 SITE DESCRIPTION AND HISTORY

The property was purchased by the City of Jacksonville in June 1960. The site has been referred to as the Jacksonville Landfill, Graham Road Landfill, Graham Road Site, Graham Road Dump and Jacksonville City Dump.

Approximately 40 acres were used for landfilling; this portion of the property was fenced in 1986 to prevent unauthorized entry.

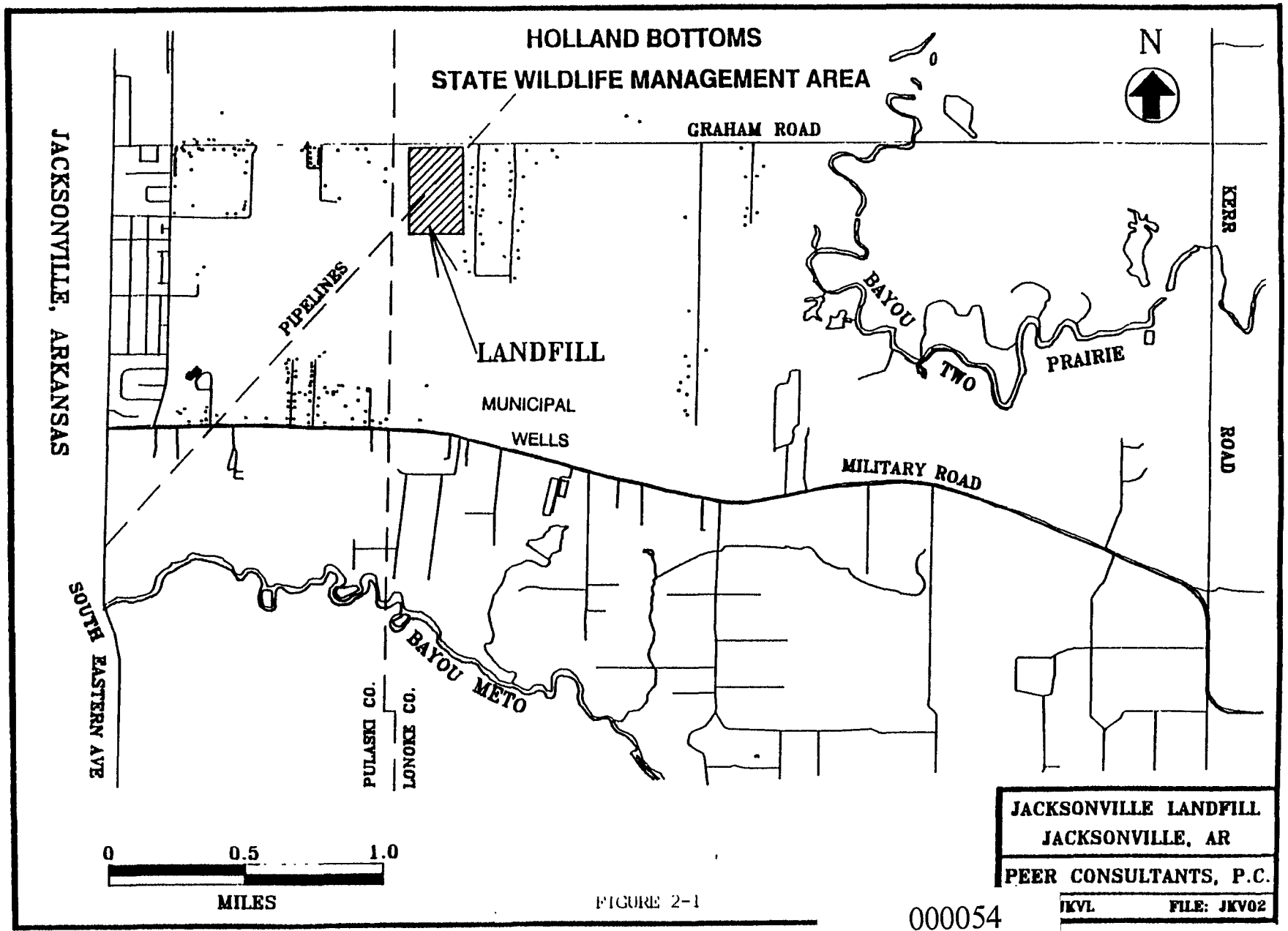
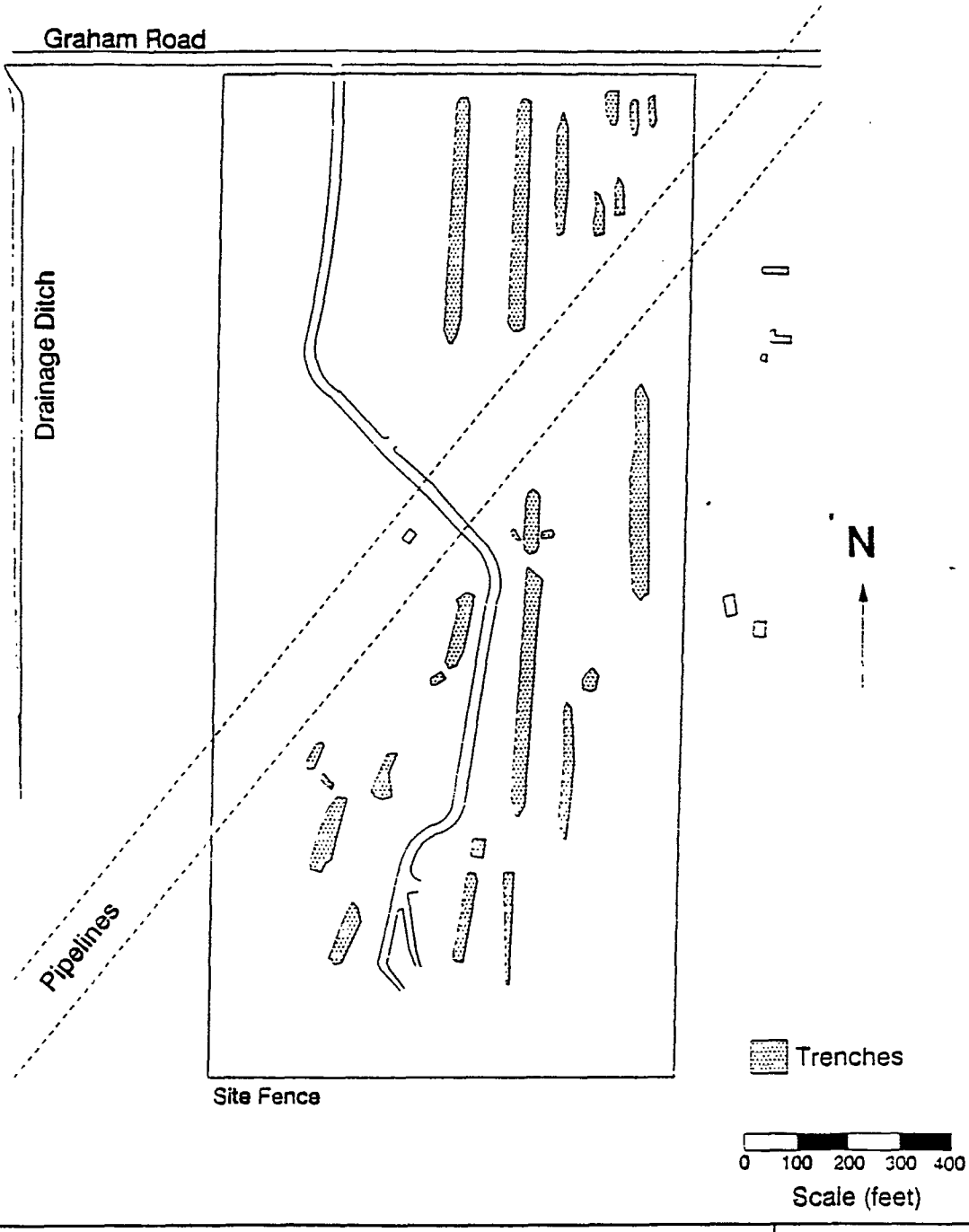

HOLLAND BOTTOMS
STATE WILDLIFE MANAGEMENT AREA
N
GRAHAM ROAD
JACKSONVILLE, ARKANSAS
PIPELINES
LANDFILL
MUNICIPAL
WELLS
BAYOU
TWO
PRAIRIE
KERR
ROAD
MILITARY ROAD
SOUTH EASTERN AVE
BAYOU METO
PULASKI CO.
LONOKE CO.
0
0.5
1.0
MILES
JACKSONVILLE LANDFILL
JACKSONVILLE, AR
PEER CONSULTANTS, P.C.
JKVL
FILE: JKV02

FIGURE 2-1



SITE FEATURES MAP

JACKSONVILLE LANDFILL, JACKSONVILLE, AR

FIG. 2-2

Records indicate that openburning and trenching with bucket and dragline were the waste handling methods used until 1969. After 1970, trenching was the sole method used on-site. The landfill was closed in July 1973 when the Arkansas Department of Pollution Control and Ecology (ADPCE) refused to grant a landfill permit because of the high water table and poor drainage in the area.

Wastes appear to have been disposed of in approximately 19 excavated trenches, and in several long surface piles, accompanied by open dumping in numerous areas around the site. After waste disposal, the trenches and some of the larger surface piles were covered with a layer of soil. After the landfill was closed, local residents continued to use the site as an open dump until the site was fenced.

An underground gas transmission pipeline, operated by Texas Eastern, runs northeast-southwest through the landfill property. The only structure on-site is a gasoline valve and pumping station. An access road used by landfill operations equipment runs roughly north-south through the fenced area of the site. The only routine maintenance performed on-site is mowing of the pipeline easement by Texas Eastern. The remainder of the fenced area is overgrown with brush and partially wooded. On the eastern side of the landfill there is a subdivision with approximately 30 houses, some of which have backyards which adjoin the site.

In a report on a private investigation conducted for the EPA during a Potentially Responsible Party Search, it was stated that municipal wastes from the City of Jacksonville as well as chemical wastes were disposed of at the Jacksonville Landfill. There apparently were no written records maintained by the commercial or residential users which identify quantities or types of wastes disposed of at the site.

The Jacksonville Landfill was proposed for inclusion on the EPA National Priorities List (NPL) of uncontrolled hazardous waste sites on January 22, 1987. It was added to the NPL on July 22, 1987. The NPL score for this site was 29.64.

Previous Investigations

The Jacksonville Landfill was identified on May 17, 1983, through a citizen's complaint to EPA regarding the possible disposal of hazardous waste at the site. In July 1983, a Preliminary Assessment/Site Investigation was conducted at the site by Ecology and Environment. One off-site soil sample was collected across Graham Road from the site entrance. Laboratory analysis of this sample detected low levels of six organic compounds. Compounds which were identified to be above laboratory detection limits were: methylene chloride (102 ppb), methyl benzene (150 ppb) and tetrachloroethane (740 ppb).

A Technical Assistance Team (TAT) from Weston-SPER inspected the site on May 23, 1985, for photo documentation and to assess the site for access. An Ecology and Environment field investigation team (FIT) performed site investigations in 1984 and 1985. A FIT report was prepared in August, 1985, outlining the soil, residential well, surface water, sediment and air sampling results. A follow-up report was prepared in November, 1986, outlining additional air sampling results.

The initial FIT investigation report (February 1984) recommended that additional soil, water and sediment sampling be performed to more accurately characterize contamination at the site. The November 1986 FIT report recommended that no additional air sampling be conducted at the site. Review of the air sampling data by the Agency for Toxic Substances and Disease Registry

(ATSDR) concluded that "based on the data provided and the sampling conditions reported, airborne volatile organic compounds do not represent a public health problem on-site and do not appear to be contributing to off-site exposure."

Sampling results from the previous site investigation are summarized in Appendix A of the accompanying Remedial Investigation (RI) Report prepared by PEER Consultants, P.C. and Resource Applications, Inc.

3.0 HAZARD ASSESSMENT

Hazard Assessment is the process of determining whether exposure to an agent can cause an increase in the incidence of a health condition. The hazard assessment is a two-step process consisting of a toxicological evaluation and a dose-response evaluation.

3.1 TOXICOLOGICAL EVALUATION

Chemicals of potential concern at the site include: dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin = 2,3,7,8-TCDD); some congeners and isomers of dioxins and furans; trichlorophenoxyacetic acid (2,4,5-T); 2,4,5-trichlorophenoxypropionic acid (2,4,5-TP = Silvex); 2,4-dichlorophenol; arsenic; benzene; 1,4-dichlorobenzene; 1,1-dichloroethene; methylene chloride; 2,4,6-trichlorophenol; polycyclic aromatic hydrocarbons (PAHs); bis-(2-ethylhexyl)phthalate; 4,4'-DDT; beryllium; manganese; aluminum; barium; chromium; copper and mercury.

Methylene chloride and phthalate esters (as well as acetone, 2 butanone and toluene) are generally considered as lab contaminants. The general rule for these contaminants is to disregard them if they are less than 10 times the maximum concentration detected in any laboratory blank (U.S. EPA, 1989). If they are found to be greater than 10 times the maximum concentration, they may be actual contaminants at the site. In this case, the methylene chloride geometric mean for soil samples at the hot spot was 1.87×10^3 ng/g compared to approximate values of 4-6 ng/g for the blanks. For the bis-(2-ethylhexyl) phthalate esters, the geometric mean was 6.39×10^2 ng/g on the landfill excluding the hot spot and 4.80×10^1 ng/g in the offsite residential area compared to 4-6 ng/g for the blanks. The

monitored concentrations are therefore 1-3 orders of magnitude higher than the blanks and they cannot be completely dismissed as lab contaminants. However, the increased risk that the methylene chloride poses is only 2.34×10^{-7} for the hot spot. The increased risk for the bis-(2-ethylhexyl) phthalate is 1.48×10^{-7} for the landfill excluding the hot spot and 5.62×10^{-8} for the offsite residential area. Because of these extremely low risks, the inclusion of the common lab contaminants in the total excess lifetime cancer risk calculations would have a minor effect. For example, at the offsite residential areas, the total cancer risk would only increase from 9.65×10^{-5} to 9.66×10^{-5}. For this reason, the lab contaminants are not included in the risk tables throughout this report.

Additional chemicals have been detected and reported in the Remedial Investigation Report but have not been assessed for potential risk. They were eliminated from the risk assessment because of quantitation limits, qualifiers, blank contamination and background level comparisons. By focusing on contaminants presenting the highest risk, complex data reduction and analyses that can obscure the true hazards can be avoided.

Toxicological evaluations characterizing the inherent toxicity of chemicals detected at the site have been performed and summarized in the form of toxicology profiles which are included in Appendix H. They have been prepared by evaluating a scientific body of literature available through the Environmental Protection Agency databases and other scientific sources. The profiles provide a weight-of-evidence for the human carcinogenic potential of certain contaminants. Weight of evidence is the extent to which the available biomedical data support the hypothesis that a substance causes cancer in humans.

As shown in the toxicity profiles in Appendix H, the chemicals detected at Jacksonville Landfill can, at high concentrations, produce mild, acute effects such as headache or more chronic effects (from long-term exposure) such as kidney or liver failure. Where appropriate, routes of exposure and human or animal studies are specified in the toxicology profiles. These chemicals can produce their effects after a certain threshold concentration in the body is reached. There are other chemicals detected during the RI that have nonthreshold health effects, such as carcinogenicity, teratogenicity and mutagenicity. For the latter chemicals, even a small concentration is associated with some risk. It is advisable to keep the exposure to the latter chemicals at or near zero. Another group of environmental effects, for example, to aquatic biota, have been reported in the literature for certain chemicals. In summary, the chemicals detected on and around the landfill have been reported in the literature to be associated with threshold and nonthreshold effects in animals and/or humans.

The major potential health risk at the site results from carcinogenic effects from exposure to human or animal carcinogens. Compounds known or suspected of being carcinogens and having risks calculated to be in excess of 10^{-7} include 2,3,7,8-TCDD; some congeners and isomers of dioxin and furan; 1,1-dichloroethene; 1,4-dichlorobenzene; benzene; arsenic; tetrachloroethene; methylene chloride; 2,4,6-trichlorophenol; selected polycyclic aromatic hydrocarbons (PAHs); 4,4'-DDT and chromium.

These compounds will be addressed in this assessment and an excess cancer risk calculated for them.

3.2 DOSE-RESPONSE EVALUATION

An important part of a risk assessment is to establish the relationship between the dose of the chemical and the response. This dose-response relationship describes the concentration at which adverse health effects may appear in the general population. Some standards, criteria and guidelines have been established for certain chemicals that quantitatively delineate the concentration at which adverse health effects are likely to occur. The standards and criteria are generally referred to as ARARs, i.e., applicable or relevant and appropriate requirements.

Comparisons of monitored data to standards, criteria and guidelines, that include those which qualify as ARARs, are shown in Table 9 in Section 5.0. A brief background on ARARs is also provided below.

The comparison of environmental concentrations to ARARs is required under the Superfund Amendments and Reauthorization Act (SARA) of 1986. It states that "remedial actions . . . under this Act shall attain a degree of cleanup of hazardous substances, pollutants and contaminants released into the environment and of control of further release at a minimum which assures protection of human health and the environment. Such remedial actions shall be relevant and appropriate under the circumstances presented by the release or threatened release of such substance, pollutant or contaminant." Standards and criteria used for comparison in this risk assessment are primarily derived from the Clean Water Act and the Safe Drinking Water Act (USEPA, 1988). For purposes of this risk assessment, comparisons are made to ambient water quality criteria (AWQC), maximum contaminant levels (MCLs), maximum contaminant level goals (MCLGs), state environmental standards and

health advisories (HAs) and secondary maximum contaminant levels (SMCLs). A discussion of each follows:

Maximum Contaminant Levels (MCLs) represent enforceable drinking water standards and are set as close to MCLGs as technologically or economically feasible. The MCL takes into consideration analytical methodology, treatment technology and costs, economic impact, and regulatory impact. For substances other than human or probable human carcinogens, the MCL generally equals the MCLG. For human or probable human carcinogens, MCLs will generally fall in the 10^{-4} to 10^{-6} hypothetical excess lifetime cancer risk range. The MCLGs and MCLs are proposed and promulgated simultaneously.

MCLs are based on the allowable lifetime exposure to the contaminant for a 70 kg adult who is presumed to consume 2 liters of water per day (U.S. EPA, 1988).

A Maximum Contaminant Level Goal (MCLG) is a nonenforceable health goal. It is set at a level at which no known or anticipated adverse human health effect would occur and which allows an adequate margin of safety. For chemicals not classified as human or probable human carcinogens, the MCLG is derived from the reference dose (RfD). For contaminants classified as human or probable human carcinogens, the MCLG is set at zero. For these substances, carcinogenicity is considered the most sensitive endpoint of toxicity. In addition, carcinogenicity is assumed not to exhibit a threshold of toxicity. Thus, for these substances, the EPA sets a health goal of zero. In May, 1989, EPA proposed MCLGs for 38 inorganic and organic contaminants. These proposed MCLGs will be final in December 1990.

EPA has also set Ambient Water Quality Criteria (AWQC). These criteria are estimates of the ambient surface water concentrations that will not result in adverse health effects in humans. In the case of suspect or proven carcinogens, concentrations associated with a range of incremental cancer risks are provided to supplement a criterion of zero. The EPA criteria are nonenforceable guidelines, which many states have used in the development of enforceable ambient water quality standards.

For most chemicals, EPA Water Quality Criteria to protect human health are available for two different exposure pathways. One criterion is based on lifetime

ingestion of both drinking water and aquatic organisms, and the other is based on lifetime ingestion of aquatic organisms alone (Integrated Risk Information System, IRIS, 1988 and 1989).

The Health Advisory (HA) Program, sponsored by the EPA Office of Drinking Water (ODW), provides information on the health effects, analytical methodology and treatment technology that are useful in dealing with the contamination of drinking water. Health Advisories (HAs) describe nonregulatory concentrations of drinking water contaminants at which adverse health effects would not be anticipated to occur over specific exposure durations. They are quoted for acute, chronic and lifetime exposure. Health Advisories contain a margin of safety to protect sensitive members of the population.

Health Advisories serve as informal technical guidance to assist federal, state and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable federal standards.

Secondary Maximum Contaminant Levels (SMCLs) are set for contaminants in drinking water that may adversely affect the odor or appearance of the water and consequently may cause a substantial number of persons to discontinue its use or otherwise adversely affect public welfare. (Federal Register, Vol. 54, No. 97). SMCLs are not federally enforceable but instead offer additional guidance based on odor, aesthetics and appearance. SMCLs were established in 1979 for 12 contaminants (Federal Register, Vol. 44), and in 1986 for fluoride (Federal Register, Vol. 51).

ARARs do not currently exist for most contaminants in soil and sediments or for many contaminants in groundwater, surface water or air. Contaminant concentrations that would result in excess risk can be calculated, however, using site-specific information for exposure duration, establishing exposure scenarios and knowing chemical characteristics (reference doses and unit cancer risk factors). In addition, concentrations of compounds found in soils and sediments can be compared to background concentrations to determine if the concentrations detected in the area under investigation exceed those which should be "normally present" in

the area. The Centers for Disease Control (CDC), has recommended that TCDD concentrations not exceed 1 ppb in residential surface soils (U.S. EPA, 1986). CDC's recommendation was made for a residential setting, where continual contact with soils would occur over a 70-year lifetime from infancy to old-age. CDC also recommended this level for channel sediments and flood plain soils that are subject to erosion and transport processes. They further recommended that TCDD concentrations not exceed 5-7 ppb in surface soils and sediments where the general public may have infrequent contact and also determined that subsurface soils containing concentrations less than 10 ppb would not pose a health hazard if covered with 12 inches of clean soil.

For purposes of this report, groundwater contaminant concentrations are compared to the MCLs, MCLGs and Health Advisories (HAs) which are included in Appendix G. Surface water concentrations can be compared to both State Arkansas Surface Water Standards listed in Table G-2 and to AWQC, both found in Appendix G. Results of this comparison are presented under the appropriate source headings in the next section.

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4.0 EXPOSURE ASSESSMENT

4.1 INTRODUCTION

Exposure assessment is the process of measuring or estimating the intensity, frequency and duration of human exposure to an agent currently present in the environment or of estimating hypothetical exposure. This section identifies actual or potential routes of exposure, characterizes the population exposed and evaluates the degree or magnitude of exposure.

To determine if exposure might occur, the human and environmental activity patterns near the site, and the most likely pathways of chemical release and transport were defined. A complete exposure pathway has four necessary components: (1) a source of chemical release to the environment; (2) a route of contaminant transport through an environmental medium; (3) an exposure or contact point; and (4) the presence of a human or environmental receptor at the exposure or contact point. These components are addressed in the following sections.

4.2 SOURCES OF CONTAMINATION AND ROUTES OF TRANSPORT

The site includes approximately 40 acres at Jacksonville Landfill. Historical records indicate hazardous wastes were placed in burn areas and later in unlined trenches from 1960 to 1973. Local residents also dumped household wastes into the landfill. At various times, standing water exists in the trenches. The site was closed in 1973 because of a high water table and poor drainage in the area. Eighteen drums are visible at an area located in the south central part of the landfill. They are not labelled and are very corroded. Parts of the drums are missing and the contents

are exposed. Some of the wastes in on-site drums were found to contain dioxin.

4.2.1 SOURCES

Previous investigators under contract to EPA identified certain contaminants at the landfill. They reported in a document (memorandum from Gene A. McDonald, FIT, Ecology and Environment, to Region VI, dated August 29, 1985) that drums on the landfill contained chlorinated phenoxy acetic acid and ester isomers, for example 2,4-D and 2,4,5-T, other organics, 2,3,7,8-TCDD and nickel and chromium. 4,4-DDT was also detected on- and off-site. Inorganics, such as barium, calcium, iron, magnesium, manganese, potassium and zinc were detected at higher concentrations in downstream than in upstream samples. Phenol and 4-methyl phenol were also detected in a downstream sample. Unknown organics were detected in three of the intermediate drainage path soil samples, in two samples collected on the east side of the site and in a downstream sample. Heptachlor epoxide was detected in one residential yard. Three residential wells showed trace hydrocarbon or octochloro dioxin contamination. Three City of Jacksonville wells contained low levels (1 ppt) of several chlorofurans and dioxins.

Sampling and analysis of soil, groundwater and surface water during the remedial investigation conducted for this risk assessment indicated dioxin, furan and herbicide contaminants in and around exposed drums; manganese found in landfill trench water samples in excess of AWQC for protection of human health; landfill and off-site residential backyard soil samples with arsenic in excess of background soil levels; and groundwater in some of the shallow monitoring wells with concentrations of the following contaminants in excess of ARARs: 1,1-dichloroethene, benzene,

chlorobenzene, aluminum, barium, chromium, manganese and selenium. Groundwater in one residential well was contaminated with traces of benzene and mercury in excess of ARARs. Several residential wells were additionally contaminated with manganese and aluminum that were in excess of SMCLs. The occurrence and distribution of these contaminants are described in other areas of the report.

4.2.2 ROUTES OF TRANSPORT

There are several contaminant transport pathways to human and environmental receptors that have been identified. These include the following:

- Direct contact with contaminated surface soil onsite and/or offsite. Direct contact includes dermal exposure as well as accidental ingestion. Teenagers may play onsite and take contaminated waste elsewhere from the landfill. Pet animals that wander onsite may play with children offsite and transfer contamination to them. On-site visitors could also accidentally transfer contaminants off-site.

- Contaminant transport via the movement of groundwater under the site. Contaminants could be transported by the soil column to the groundwater. If the shallow aquifer were used by local residents as a potable water source, an exposure could occur. Presently, nearby wells are not used for a drinking water supply. There is also a possibility of direct contact exposure, accidental ingestion, or inhalation associated with the use of groundwater for nonpotable purposes such as lawn watering, car washing or industrial processes.

- Contaminated soils and sediment transport via surface water runoff. Surface water runoff could transport contaminated soil or sediments from the landfill to off-site residential backyards or to off-site drainage ditches. Chemical analyses suggest this has not been an important pathway for dioxin and herbicide migration from the site.

Airborne transport of contaminated dusts or evaporation of volatile organic compounds from contaminated surface soil to ambient air are considered minor routes of contaminant migration. Ethylbenzene and xylenes were detected in air samples collected from around the site (FIT team memorandum, dated August 29, 1985). However, they were not found in high concentrations. Further, no volatile compounds were detected in high enough concentrations to register on field survey instruments (flame and photo ionization detectors) during the subsequent remedial investigation. This lack of airborne hazards is likely due to the vegetative cover over most areas of the site that tends to prevent suspension or emission of particulate contaminants. An additional factor inhibiting airborne emissions is the relatively high moisture of the soils due to precipitation and drainage.

The most important pathways have been selected for further analysis in this document. They include both on and off-site direct contact with contaminated soils. These pathways are not only the most probable but, due to measured concentrations and likely frequency of exposure, produce the highest exposure dose rates.

4.3 IDENTIFICATION OF RECEPTORS AND ROUTES OF EXPOSURE

4.3.1 RECEPTORS

Human and environmental receptors that may potentially be exposed to hazardous constituents associated with the site have been identified. These include:

Local populations -- people residing near the site boundaries or site visitors/trespassers.

Environmental receptors -- wildlife and domestic animals that come into contact with the area.

The site is located within a residential area. Within a one-half mile radius of the Jacksonville Landfill, there are approximately 51 single family homes. The one-half mile radius was chosen because of the population distribution, that is, there is a relatively large population density within a one-half mile radius, followed by a more sparsely populated area. Assuming an average of three to four people per home, approximately 153 to 204 people live within a one-half mile radius of the site. The nearest eight residential dwellings are located approximately 30 yards east of the fence outside of the landfill.

There are no businesses or commercial areas located within one and one-half miles. However, there is a school within three quarters of a mile. The types of receptors are not expected to change within the forseeable future. This is because no new businesses, commercial areas or schools are expected to relocate within the immediate area. Part of the landfill is located within a flood plain and development would be costly. The rest of the

landfill is located within a predominantly agricultural area. Because of these factors, commercial development is not a likely event. Within the next 50 years, the number of residences immediately adjacent to the landfill could increase (personal communication, City Engineer, Jacksonville, Arkansas).

Over the next 40 years, the numbers of people in the entire City of Jacksonville are expected to increase relatively slowly compared to more densely populated regions in the United States. The Metroplan Council of Governments for the Little Rock/North Little Rock Metropolitan Statistical Area has estimated the population of the City of Jacksonville to be 53,000 in the year 2020 and 62,540 in the year 2030. Taken together, within the next 50 years, the types of receptors are expected to remain relatively stable, with the number of people in the immediate off-site area of the landfill increasing slightly.

Vegetation is lush throughout the landfill with the exception of the drainage ditches, trenches, and the drum area. Previous reports stated that vegetation had died 10 feet outward from the drainage ditch banks and 30 feet or more around the drums (memorandum from Gene A. McDonald, FIT, Ecology and Environment, Region VI, dated August 29, 1985). However, during a recent site inspection, no stressed vegetation was observed beyond the drainage ditch banks and only 5 feet or less of stressed vegetation was observed outside individual drums. There are footpaths throughout the area, most leading from the homes on the east side. The large embankments of fill material have bicycle tracks as do the impoundments. It appears that children played there quite extensively. During a FIT Team Sampling inspection in 1985, a blue pickup truck was observed dumping some household waste

and the driver seemed to be scavenging through the other trash as the FIT Team left the site (memorandum from Gene A. McDonald, FIT, Ecology and Environment, Region VI, dated August 29, 1985).

Investigations indicate there are no sensitive or endangered species or critical habitats located within or around the landfill. Animals most likely impacted are common wildlife, such as squirrels, rabbits, birds and deer, and domestic animals that were observed on-site during the remedial investigation.

4.3.2 ROUTES OF EXPOSURE

The mode of exposure influences risks to receptors. Modes of exposure usually include ingestion, inhalation and direct contact. Ingestion may take the form of direct exposure through drinking contaminated water or eating contaminated food or may involve indirect routes such as the use of contaminated water for food preparation. Direct inhalation exposure may result from breathing air that has become contaminated through volatilization of contaminated water. As stated previously, direct inhalation of airborne contaminants is not a problem because of the absence of significant air emissions from the site. Dermal exposure may result from direct contact with contaminated water, soil or other material. The following is a media-by-media discussion of the major potential routes of exposure to hazardous constituents associated with the site. This section also identifies populations most likely at risk via each potential exposure pathway.

4.3.2.1 Groundwater

Groundwater from both the monitoring and residential wells was analyzed for possible migration of contaminants from the soil to the water. Calculations of the risk and hazard indices were

performed on the contaminants in these wells to quantify carcinogenic and noncarcinogenic effects. For monitoring wells, calculations were performed to indicate possible risks if the shallow groundwater were ever consumed in the future. For residential wells, calculations were made on the pollutants for the worst possible case, that is, if it were ever ingested by humans at the monitored pollutant concentrations. A well inventory performed by RI field team members conducted during the RI indicated that most of the residential wells were decommissioned; only one well immediately adjacent to the site is used as a nonpotable source for lawn watering and car washing. The residents adjacent to the landfill currently are on a municipal water system. Hypothetical dose rates were calculated for pollutants in the monitoring wells (Table 8A) and for residential wells (Table 8B). These can be compared to RfDs indicated in the respective tables.

4.3.2.2 Surface Soils and Sediments

Sampling and analysis of on-site and off-site surface soils and sediments indicate the presence of various organic and inorganic compounds. Tables 3 through 6 summarize the occurrence and distribution of surface soil contaminants. Compounds of concern include dioxin and congeners of dioxin; furans; herbicides; methylene chloride; 2,4,6-trichlorophenol; polycyclic aromatic hydrocarbons (PAHs); bis-(2 ethylhexyl) phthalate; arsenic and 4,4′-DDT. The significance of the detected methylene chloride and phthalate lab contaminants has been previously discussed in paragraph 3.1. In addition, the inorganic elements barium, beryllium, cadmium, chromium, manganese, mercury, nickel, silver, zinc and lead were detected in concentrations above background. Lead is of particular concern because of its high concentration (geometric mean of 0.47 mg/g and a range of 0.1 to 1.5 mg/g) and

reports that it is a probable human carcinogen. Actual quantitation of the lead cannot be performed in this risk assessment because no carcinogen potency factor (q^*) is yet available (IRIS, 1989). Further, EPA has not set a standard for lead in soil. However, an interim soil cleanup level for total lead was set at 0.5 to 1.0 mg/g for protection of health from direct contact in residential settings (U.S. EPA, 1989c). The geometric mean concentration did not exceed the interim levels, but 7 samples on the landfill excluding the hot spot did exceed 0.5 mg/g, the low end of the soil cleanup level. However, because of the existing fence, there is no direct residential contact by young children or pregnant women, the two groups for whom the levels were designed to protect.

The major potential routes of human exposure to contaminated soil and sediments are dermal contact and accidental ingestion by anyone who frequents the on-site drum area.

No quantitative data are available on the size of the population potentially exposed to on-site hazardous constituents via direct contact. Access to the site is now restricted with a fence and gate, but previous report(s) suggest that the landfill was not always secure between 1973 to 1985. Scavengers and/or dumpers have been observed on-site as the FIT team left the site on April 4, 1985 (memorandum from Gene A. McDonald, FIT, Ecology and Environment, Region VI, dated August 29, 1985).

Recently, bicycle tracks and/or teenagers riding bikes have been observed playing on-site during RI field studies. Some of the barbed wire fencing is low enough with wide enough gaps to allow access by trespassers. Receptors entering the site boundaries via this route are at risk. Receptors most likely to come into direct contact with surface soils are local teenage and adult residents.

Direct exposure with on-site surface soils and sediments is therefore a likely exposure scenario.

Off-site surface soil is also of concern. Residential areas are juxtaposed to the landfill. Young children can play in the soil and adults can garden in residential backyards next to the landfill and accidentally or willfully ingest the soil. The potential exists for exposure via this route. Therefore, this route is considered a likely off-site exposure scenario.

Wildlife and domestic animals may also be exposed to on-site or off-site contaminated soils and sediments. Domestic animals may carry contaminated sediments or soils to local residences and potentially affect human occupants. Wildlife could eat contaminated soil and the local population could eat contaminated wildlife (rabbits), potentially being exposed to contaminants via ingestion. Exposure to animals is, however, estimated to be sporadic and of limited duration.

4.3.2.3 Surface Water

Human or environmental exposure to surface water contaminants may occur in the unnamed pond adjacent to the landfill, a drainage ditch at the entrance to Holland Bottoms Wildlife Management Area, in landfill trench waters, and other areas adjacent to the Jacksonville Landfill. Manganese levels in excess of AWQC for protection of human health have been detected in the trench waters, in adjacent drainage ditches and surrounding areas. Beryllium in excess of AWQC for protection of human health has been detected in the background water sample, the Bayou Two Prairie and the unnamed pond near the landfill. People can gain access to the landfill, wade in the trench and accidentally ingest the contaminated water.

However, their exposure would probably be sporadic and of short duration.

Environmental receptors may also be exposed to hazardous constituents in surface waters. Wild and domestic animals may frequent potentially impacted surface waters to wade or bathe and ingest the water. As with humans, their exposure would be sporadic and of short duration.

4.3.2.4 Ambient Air

Under present site conditions, inhalation of airborne contaminated dusts and/or inhalation of volatilized surface soil contaminants are considered to be very minor routes of human or environmental exposure. No dioxins or furans were measured in the air samples during previous investigations. Dioxin and phenoxy herbicides adsorb strongly to the soil and local conditions preclude suspension of soil particulates. The only organics detected in air samples at the landfill were ethylbenzene and xylenes (FIT team document dated September 13, 1986). All airborne contaminants detected were at very low concentrations.

The potential for emissions is low because surface soils are not appreciably contaminated with volatile organic contaminants and the vegetative cover over most areas of the site and other factors limit the emission of airborne nonvolatile particulates. Because of these factors, no definitive air sampling was conducted during RI studies; no quantitative estimates of dose rates and risks from the inhalation route of exposure were performed for this report. However, air monitoring was conducted during the RI as part of the Health and Safety Program using nonspecific field survey instruments (flame and photo ionization detectors). No volatile compounds were detected in high enough concentrations to register

on these survey meters, further validating the assumption of insignificant risk via the airborne route.

4.3.2.5 Summary

The preceding discussion identified human and environmental exposure pathways of concern. In summary, the potential routes of exposure to local and/or distant populations requiring further evaluation include the following:

- Direct contact with contaminated surface soil on-site and/or soils and sediments adjacent to the site that may have been contaminated by surface water runoff or erosion processes. Direct contact includes dermal exposure as well as accidental ingestion.

- Accidental ingestion of surface waters in the drainage ditch at the entrance to Holland Bottoms Wildlife Management Area; landfill trench waters; areas around, and drainage ditches adjacent to, the Jacksonville Landfill and an unnamed pond near the landfill.

- Direct contact exposure, accidental ingestion or inhalation associated with the use of groundwater for nonpotable purposes such as lawn watering, and car washing. This only applies to one residential well used as a nonpotable source. Deliberate ingestion of contaminated groundwater is not presently expected to be a route of exposure because it is not used as a potable source.

4.4 CHARACTERIZATION OF EXPOSURE

The previous section identified human receptors and exposure pathways of concern. The final step in conducting an exposure assessment requires a quantitative determination of the contaminant dose rates received by those receptors. This section provides route-specific estimates of the total amount of each contaminant to which a receptor is exposed, or potentially exposed, on a daily basis. Dose rate estimates are calculated for compounds detected in environmental media using dose-response relationships [Acceptable Daily Intakes (ADIs) or reference doses (RfDs) or Unit Cancer Risk Slope Factors (UCRs or q^*)] presented in other sections, that have been established in recognized databases such as the Integrated Risk Information system (IRIS).

A dose rate is defined as the amount of a compound (mg) absorbed by a receptor on a daily basis per kilogram of body weight. Dose rates can be calculated for lifetime or less than lifetime exposures as follows:

$$\text{Dose Rate} = \frac{\text{Concentration in an environmental medium} \times \text{Contact rate} \times \text{exposure duration} \times \text{absorbed fraction}}{\text{body weight}}$$

See Appendix C and F for units.

4.4.1 GROUNDWATER ROUTE

The Quaternary/Eocene alluvial aquifer is the most important water bearing unit in the Jacksonville Landfill area, providing water for agricultural, domestic and municipal uses. However, the section underlying the Jacksonville Landfill Site actually consists of deep sands and clays of the Eocene Series, and shallow silts and

clays, which represent Quaternary deposits. The boundary between the Eocene Series and the Quaternary deposits is difficult to define. The Paleocene Midway Foundation serves as the bottom confining unit for the Eocene/Quaternary section in the vicinity of the landfill.

At the Jacksonville Landfill Site, there is a clay and/or silty clay layer at the surface, a clay layer at a the depth of about 25 feet, and a clay layer at the Midway formation below the underlying aquifer. The clay layer at 25 feet acts as an aquitard and separates the two aquifers. The top aquifer is composed of clayey silt and fine to medium grained sand. The lower aquifer is composed of both fine to medium sand and clayey silt. Groundwater flows generally towards the east-southeast.

4.4.1.1 Ingestion

There is no current hazard associated with shallow groundwater ingestion because none is currently consumed by local residents. One residential well located adjacent to the landfill is currently used for nonpotable purposes, i.e., watering yards and providing drinking water for animals.

If the water were consumed at the detected concentration of contaminants, the maximum potential exposures associated with long-term ingestion of contaminated groundwater in the monitoring and residential wells would be as listed in Tables 1A and 1B, respectively. Monitoring wells (Table 1A) that were installed during this investigation indicate the extent of any groundwater contamination within the landfill and upgradient areas. One of the residential wells that was sampled (Table 1B), as previously stated, is used as a nonpotable water supply by a local resident.

TABLE 1A

DOSE RATE ESTIMATES
INGESTION OF CONTAMINATED GROUNDWATER - MONITORING WELLS

GROUNDWATER ROUTE		
	JK GW-02	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Xylene	8	2.29×10^{-4}
Selenium	2.5	7.14×10^{-5}

GROUNDWATER ROUTE		
	JK GW-03	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Manganese	1,820	5.2×10^{-2}

GROUNDWATER ROUTE		
	JK GW-04	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	990	2.83×10^{-2}
Carcinogens		
1,1-dichloroethene	11	3.14×10^{-4}
1,4-dichlorobenzene	58	1.66×10^{-3}

(Sample calculations are provided in Appendix F)

TABLE 1A (Continued)

DOSE RATE ESTIMATES
INGESTION OF CONTAMINATED GROUNDWATER - MONITORING WELLS

GROUNDWATER ROUTE		
	JK GW-05	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	940	2.69×10^{-2}
barium	964	2.75×10^{-2}
manganese	4,170	1.19×10^{-1}
selenium	17	4.86×10^{-4}
Carcinogens		
Benzene	15	4.29×10^{-4}

GROUNDWATER ROUTE		
	JK GW-06	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Cadmium	4	1.14×10^{-4}
manganese	643	1.84×10^{-2}
selenium	18	5.14×10^{-4}

GROUNDWATER ROUTE		
	JK GW-07	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	38	1.09×10^{-3}

(Sample calculations are provided in Appendix F)

TABLE 1A (Continued)

DOSE RATE ESTIMATES
INGESTION OF CONTAMINATED GROUNDWATER - MONITORING WELLS

GROUNDWATER ROUTE		
	JK GW-09	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	8	2.29×10^{-4}
benzoic acid	6	1.71×10^{-4}
barium	1,090	3.11×10^{-2}

GROUNDWATER ROUTE		
	JK GW-10	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Xylene	7	2.00×10^{-4}

GROUNDWATER ROUTE		
	JK GW-12	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	11	3.14×10^{-4}
benzoic acid	30	8.57×10^{-4}
phenol	18	5.14×10^{-4}
selenium	2	5.71×10^{-5}
Carcinogens		
Chromium (VI)	197	5.63×10^{-3}

(Sample calculations are provided in Appendix F)

TABLE 1A (Continued)

DOSE RATE ESTIMATES
INGESTION OF CONTAMINATED GROUNDWATER - MONITORING WELLS

GROUNDWATER ROUTE		
	JK GW-13	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	5	1.43×10^{-4}
barium	1,110	3.14×10^{-2}

(Sample calculations are provided in Appendix F)

TABLE 1B

DOSE RATE ESTIMATES
INGESTION OF CONTAMINATED GROUNDWATER - RESIDENTIAL WELLS

GROUNDWATER ROUTE		
	JK RW-01*	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Antimony	33.5	9.57×10^{-4}
Silver	15.1	4.31×10^{-4}

GROUNDWATER ROUTE		
	JK RW-02	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	18	5.14×10^{-4}
Antimony	31.7	9.06×10^{-4}
Mercury	8.2	2.34×10^{-4}
Carcinogen		
Benzene	6	1.71×10^{-4}

(Sample calculations are provided in Appendix F)

* Residential Well RW-01 used for watering yards and providing drinking water for animals.

It provides an indication of the closest downgradient groundwater that could be accessible to a local resident. The tables indicate the concentration of quantifiable contaminants in ug/l and the dose rates in mg/kg-day for each individual well. These calculations are only hypothetical calculations if residents were to ingest the water having the reported contaminant concentration. Dose rate estimates are provided for all compounds using RfDs or q^*s currently available. The criteria for a compound to be included in the tables is that the chemical have a q^* or an RfD to allow quantitation. For some chemicals there are no RfD's or q^*s published. However, none of this latter group was found in significant concentrations and none exceeded any ARARs. The estimated dose rates assume a 70-kg person ingests 2 liters of water per day. Sample calculations are provided in Appendix F.

Monitoring wells (GW-01, GW-10) and one of the residential wells (RW-02) had common lab contaminants in them, including toluene (28 ug/l) and acetone (1200 ug/l and 19 ug/l), respectively. The dose rates for ingestion calculated from the concentrations are 8.00×10^{-4} mg/kg-day for toluene and 3.43×10^{-2} mg/kg-day for acetone. The general rule for these contaminants is to disregard them if they are less than 10 times the maximum amount detected in any laboratory blank (U.S. EPA, 1989d). If they are greater than 10 times the maximum value, they may be actual contaminants at the site. In this case, all the blank values were undetectable. The monitored concentrations are, therefore, greater than ten times the blanks and they cannot be completely dismissed as lab contaminants. However, the dose rates calculated from the concentrations did not exceed the oral RfDs and therefore no chronic effects are expected from ingesting the toluene and acetone at the monitored concentrations in the groundwater.

In addition, aluminum and copper were detected in the monitoring wells. However, they were also found in the same concentration range in the upgradient wells, and therefore cannot be attributed to site contamination.

4.4.1.2 Inhalation -

As with ingestion, this is only a hypothetical calculation as there is no reported deliberate human ingestion nor inhalation of shallow groundwater based on results of the house-to-house survey conducted during the remedial investigation.

Domestic use of contaminated groundwater can contribute to the total dose rate a receptor may receive. Inhalation of volatile organic contaminants during showering can constitute a significant, quantifiable route of human exposure. For inorganics, the inhalation route is considered negligible because they do not volatilize.

To estimate a dose rate or daily exposure associated with inhalation, several assumptions are made (Andelman, 1985; U.S. EPA, 1985b and 1989a). These include the following:

- 190 liters of water are used during showering;
- 50% of the contaminant volatilizes to the air;
- 0.33 hours/day are spent in the bathroom;
- 0.6 m^3/hr. are inhaled.
- The estimated volume of a bathroom is 12 m^3.
- 100% of the compound is absorbed upon entering the lungs;
- The weight of an adult male is 70 kg.

Using these assumptions, dose rates associated with inhalation during showering were calculated for the volatile organic compounds

detected in groundwater monitoring and residential well samples. The results are shown in Table 2. Example calculations are provided in Appendix F.

Monitoring wells (GW-01, GW-10) and the residential well (RW-02) had common lab contaminants in them, including toluene (28 ug/l) and acetone (1200 ug/l and 19 ug/l), respectively. The dose rates for inhalation calculated from the concentrations are 5.94 x 10^{-4} mg/kg-day for toluene and 2.55 x 10^{-2} and 4.03 x 10^{-4} mg/kg-day for acetone. The general rule for these contaminants is to disregard them if they are less than 10 times the maximum amount detected in any laboratory blank (U.S. EPA, 1989d). If they are greater than 10 times the maximum value, they may be actual contaminants at the site. In this case, all the blank values were undetectable. The monitored concentrations are, therefore, greater than ten times the blanks and they cannot be completely dismissed as lab contaminants. No inhalation RfDs are available in the IRIS (1989) for these common lab contaminants. However, toxic effects from the toluene and acetone due to inhalation are not expected. This is because the monitored concentrations were found to be orders of magnitude below the OSHA permissible exposure limits (PELs), and American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit values (TLVs) (U.S. Department of Labor, 1989 and U.S. Department of Health and Human Services, 1987).

4.4.1.3 Nonpotable Uses

No quantitative data are available to estimate exposures associated with dermal contact, inhalation or ingestion of contaminated groundwater during activities such as car washing or lawn watering. The degree of exposure depends on human activity patterns and factors such as the frequency and duration of

TABLE 2A

DOSE RATE ESTIMATES
INHALATION OF GROUNDWATER DURING SHOWERING - MONITORING WELLS

GROUNDWATER ROUTE		
	JK GW-02	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Xylene	8	1.70×10^{-4}

GROUNDWATER ROUTE		
	JK GW-04	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	990	2.10×10^{-2}
Carcinogens		
1,1-dichloroethene	11	2.33×10^{-4}
1,4-dichlorobenzene	58	1.23×10^{-3}

GROUNDWATER ROUTE		
	JK GW-05	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	940	1.99×10^{-2}
Carcinogens		
benzene	15	3.18×10^{-4}

[Sample calculations are provided in Appendix F]

TABLE 2A (Continued)

DOSE RATE ESTIMATES
INHALATION OF GROUNDWATER DURING SHOWERING - MONITORING WELLS

GROUNDWATER ROUTE		
	JK GW-07	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	38	8.06×10^{-4}

GROUNDWATER ROUTE		
	JK GW-09	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	8	1.7×10^{-4}
benzoic acid	6	1.27×10^{-4}

GROUNDWATER ROUTE		
	JK GW-10	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Xylene	7	1.49×10^{-4}

[Sample calculations are provided in Appendix F]

TABLE 2A (Continued)

DOSE RATE ESTIMATES
INHALATION OF GROUNDWATER DURING SHOWERING - MONITORING WELLS

GROUNDWATER ROUTE		
	JK GW-12	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	11	2.33×10^{-4}
benzoic acid	30	6.36×10^{-4}
phenol	18	3.82×10^{-4}

GROUNDWATER ROUTE		
	JK GW-13	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	5	1.06×10^{-4}

[Sample calculations are provided in Appendix F]

TABLE 2B

DOSE RATE ESTIMATES
INHALATION OF GROUNDWATER DURING SHOWERING - RESIDENTIAL WELL

GROUNDWATER ROUTE		
	JK RW-02	
Compound	Concentration, ug/l	Dose Rate, mg/kg-day
Noncarcinogens		
Chlorobenzene	18	3.82×10^{-4}
Carcinogens		
benzene	6	1.27×10^{-4}

[Sample calculations are provided in Appendix F]

exposure. However, it is unlikely that these pathways are significant routes of human exposure to site-associated hazardous contaminants since exposure is apt to be of short duration and intermittent. Further, none of the detected contaminants display acute toxicity at the measured concentrations.

4.4.2 SOIL ROUTE

Dermal Contact and Accidental Ingestion

Dermal contact or accidental ingestion of contaminated soil can be significant routes of human exposure to site-associated contaminants. The degree of exposure is dependent upon human activity patterns on or near the site and factors such as the amount of skin area exposed, duration of contact, absorption and soil conditions.

Tables 3, 4, 5 and 6 list the dose rates associated with direct contact to on-site and off-site residential surface soil contaminants. Tables 3A, B and C summarize dose rates due to noncarcinogens in soil while Tables 4A, B and C summarize carcinogenic doses. Table 5 lists estimated 2,3,7,8-TCDD dose rates to individuals onsite and hypothetical doses for offsite exposure. No 2,3,7,8-TCDD was measured offsite so, as a worst case, the hypothetical calculation was made assuming concentrations were at one-half the laboratory analytical detection limit. Similar calculations are shown in Table 6 for all dioxins (including 2,3,7,8-TCDD) and furans. These worst case, hypothetical calculations were included because of the sensitivity of the area residents to dioxin contamination issues and hence the need to explore even highly unlikely possibilities. Dose rates for soils were calculated for the following three areas:

TABLE 3A

SUMMARY OF ESTIMATED DOSE RATES FROM SOIL - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE - HOT SPOT (DRUM SAMPLES)			
		DOSE RATE = mg/kg-DAY		
	CONCENTRATION, ng/g	ACCIDENTAL INGESTION	DERMAL CONTACT	TOTAL
2,4,5-T:	Average: 1.18×10^{7}	1.20×10^{-2} (2.86×10^{-2})**	1.96×10^{0} (4.65×10^{0})	1.97×10^{0} (4.68×10^{0})
	Geometric Mean: 5.29×10^{6}	5.40×10^{-3} (2.86×10^{-2})	8.80×10^{-1} (4.65×10^{0})	8.85×10^{-1} (4.68×10^{0})
2,4,5-TP:	Average: 3.72×10^{5}	3.79×10^{-4} (2.04×10^{-3})	6.17×10^{-2} (3.32×10^{-1})	6.21×10^{-2} (3.34×10^{-1})
	Geometric Mean: 1.65×10^{5}	1.69×10^{-4} (2.04×10^{-3})	2.74×10^{-2} (3.32×10^{-1})	2.76×10^{-2} (3.34×10^{-1})
2,4-dichlorophenol	Average: 5.56×10^{4}	5.71×10^{-5} (1.73×10^{-4})	9.51×10^{-4} (2.89×10^{-3})	1.01×10^{-3} (3.06×10^{-3})
	Geometric Mean: 2.49×10^{4}	2.54×10^{-5} (1.73×10^{-4})	4.24×10^{-4} (2.89×10^{-3})	4.49×10^{-4} (3.06×10^{-3})
Tetrachloroethene	Average: 8.0×10^{0}	8.16×10^{-9} (1.02×10^{-8})	1.36×10^{-7} (1.7×10^{-7})	1.44×10^{-7} (1.8×10^{-7})
	Geometric Mean: 7.8×10^{0}	7.91×10^{-9} (1.02×10^{-8})	1.29×10^{-7} (1.7×10^{-7})	1.37×10^{-7} (1.8×10^{-7})
2,4,5-Trichloro-phenol	Average: 1.17×10^{6}	1.19×10^{-3} (3.06×10^{-3})	1.98×10^{-2} (5.1×10^{-2})	2.10×10^{-2} (5.41×10^{-2})
	Geometric Mean: 4.96×10^{5}	5.06×10^{-4} (3.06×10^{-3})	8.43×10^{-3} (5.1×10^{-2})	8.93×10^{-3} (5.41×10^{-2})

** The numbers outside parentheses are most plausible case; the numbers inside parentheses are worst case.
Averages and geometric means are defined in Glossary.

TABLE 3A (Continued)

SUMMARY OF ESTIMATED DOSE RATES FROM SOIL - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE - HOT SPOT (DRUM SAMPLES)			
		DOSE RATE = mg/kg-DAY		
	CONCENTRATION, ng/g	ACCIDENTAL INGESTION	DERMAL CONTACT	TOTAL
1,2,4-Trichloro-benzene	Average: 2.40×10^{3}	2.44×10^{-6} (7.04×10^{-6})**	4.07×10^{-5} (1.17×10^{-4})	4.32×10^{-5} (1.24×10^{-4})
	Geometric Mean: 5.24×10^{2}	5.34×10^{-7} (7.04×10^{-6})	8.91×10^{-6} (1.17×10^{-4})	9.44×10^{-6} (1.24×10^{-4})
Naphthalene	Average: 1.10×10^{4}	1.12×10^{-5} (1.12×10^{-5})	1.87×10^{-4} (1.87×10^{-4})	1.98×10^{-4} (1.98×10^{-4})
	Geometric Mean: 1.10×10^{4}	1.12×10^{-5} (1.12×10^{-5})	1.87×10^{-4} (1.8×10^{-4})	1.98×10^{-4} (1.98×10^{-4})
2-Chlorophenol	Average: 4.95×10^{2}	5.05×10^{-7} (6.83×10^{-7})	8.42×10^{-6} (1.14×10^{-5})	8.93×10^{-6} (1.21×10^{-5})
	Geometric Mean: 4.63×10^{2}	4.73×10^{-7} (6.83×10^{-7})	7.87×10^{-6} (1.14×10^{-5})	8.34×10^{-6} (1.21×10^{-5})
Benzoic Acid	Average: 3.53×10^{3}	3.60×10^{-6} (9.89×10^{-6})	6.00×10^{-5} (1.65×10^{-4})	6.36×10^{-5} (1.75×10^{-4})
	Geometric Mean: 1.09×10^{3}	1.11×10^{-6} (9.89×10^{-6})	1.85×10^{-5} (1.65×10^{-4})	1.96×10^{-5} (1.75×10^{-4})
Phenol	Average: 2.40×10^{2}	2.45×10^{-7} (2.45×10^{-7})	4.08×10^{-6} (4.08×10^{-6})	4.33×10^{-6} (4.33×10^{-6})
	Geometric Mean: 2.40×10^{2}	2.45×10^{-7} (2.45×10^{-7})	4.08×10^{-6} (4.08×10^{-6})	4.33×10^{-6} (4.33×10^{-6})

** The numbers outside parentheses are most plausible case; the numbers inside parentheses are worst case.

TABLE 3B

SUMMARY OF ESTIMATED DOSE RATES FROM SOIL - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE - LANDFILL EXCLUDING HOT SPOT (DRUM SAMPLES)			
		DOSE RATE - mg/kg-DAY		
	CONCENTRATION, ng/g	ACCIDENTAL INGESTION	DERMAL CONTACT	TOTAL
2,4,5-T:	Average: 4.50×10^{3}	4.59×10^{-6} (4.59×10^{-6})**	7.48×10^{-4} (7.48×10^{-4})	7.52×10^{-4} (7.52×10^{-4})
	Geometric Mean: 4.50×10^{3}	4.59×10^{-6} (4.59×10^{-6})	7.48×10^{-4} (7.48×10^{-4})	7.52×10^{-4} (7.52×10^{-4})
Barium	Average: 6.90×10^{2}	7.04×10^{-4} (8.40×10^{-4})	---	7.04×10^{-4} (8.40×10^{-4})
	Geometric Mean: 6.76×10^{2}	6.90×10^{-4} (8.40×10^{-4})	---	6.90×10^{-4} (8.40×10^{-4})
Beryllium	Average: $8.80 \times 10^{+2}$	8.98×10^{-7} (1.63×10^{-6})	---	8.98×10^{-7} (1.63×10^{-6})
	Geometric Mean: $8.30 \times 10^{+2}$	8.47×10^{-7} (1.63×10^{-6})	---	8.47×10^{-7} (1.63×10^{-6})
Cadmium	Average: 8.03×10^{3}	8.19×10^{-5} (1.94×10^{-5})	---	8.19×10^{-5} (1.94×10^{-5})
	Geometric Mean: 4.96×10^{3}	5.06×10^{-6} (1.94×10^{-5})	---	5.06×10^{-6} (1.94×10^{-5})
Chromium, total	Average: 1.47×10^{5}	1.50×10^{-4} (4.95×10^{-4})	---	1.50×10^{-4} (4.95×10^{-4})
	Geometric Mean: 1.01×10^{5}	1.03×10^{-4} (4.95×10^{-4})	---	1.03×10^{-4} (4.95×10^{-4})
Manganese	Average: 2.55×10^{6}	2.60×10^{-3} (4.89×10^{-3})	---	2.60×10^{-3} (4.89×10^{-3})
	Geometric Mean 2.39×10^{6}	2.43×10^{-3} (4.89×10^{-3})	---	2.43×10^{-3} (4.89×10^{-3})

** The numbers outside parentheses are most plausible case; the numbers inside parentheses are worst case.

TABLE 3B (Continued)

SUMMARY OF ESTIMATED DOSE RATES FROM SOIL - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE - LANDFILL EXCLUDING HOT SPOT (DRUM SAMPLES)			
	CONCENTRATION, ng/g	DOSE RATE = mg/kg-DAY		
		ACCIDENTAL INGESTION	DERMAL CONTACT	TOTAL
Mercury	Average: $8 \times 10^{+2}$	8.16×10^{-7} (8.16×10^{-7})	---	8.16×10^{-7} (8.16×10^{-7})
	Geometric Mean: $8 \times 10^{+2}$	8.16×10^{-7} (8.16×10^{-7})	---	8.16×10^{-7} (8.16×10^{-7})
Nickel	Average: 3.59×10^{4}	3.66×10^{-5} (1.33×10^{-4})	---	3.66×10^{-5} (1.33×10^{-4})
	Geometric Mean: 2.74×10^{4}	2.79×10^{-5} (1.33×10^{-4})	---	2.79×10^{-5} (1.33×10^{-4})
Silver	Average: 4.64×10^{3}	4.73×10^{-6} (1.94×10^{-5})	---	4.73×10^{-6} (1.94×10^{-5})
	Geometric Mean: 3.59×10^{3}	3.66×10^{-6} (1.94×10^{-5})	---	3.66×10^{-6} (1.94×10^{-5})
Zinc	Average: 9.75×10^{5}	9.95×10^{-4} (1.10×10^{-2})	---	9.95×10^{-4} (1.10×10^{-2})
	Geometric Mean: 3.74×10^{5}	3.81×10^{-4} (1.10×10^{-2})	---	3.81×10^{-4} (1.10×10^{-2})

** The numbers outside parentheses are most plausible case; the numbers inside parentheses are worst case.

TABLE 3C

SUMMARY OF ESTIMATED DOSE RATES FROM SOIL - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE - OFF-SITE (OS) RESIDENTIAL AREA			
		DOSE RATE = mg/kg-DAY		
	CONCENTRATION, ng/g	ACCIDENTAL INGESTION	DERMAL CONTACT	TOTAL
4,4'-DDT	Average: 4.40×10^{1}	3.52×10^{-6} (3.52×10^{-6})**	1.71×10^{-5} (1.71×10^{-5})	2.06×10^{-5} (2.06×10^{-5})
	Geometric Mean: 4.40×10^{1}	3.52×10^{-6} (3.52×10^{-6})	1.71×10^{-5} (1.71×10^{-5})	2.06×10^{-5} (2.06×10^{-5})
Beryllium	Average: $6.40 \times 10^{+2}$	5.12×10^{-5} (8.00×10^{-5})	---	5.12×10^{-5} (8.00×10^{-5})
	Geometric Mean: $6.10 \times 10^{+2}$	4.88×10^{-5} (8.00×10^{-5})	---	4.88×10^{-5} (8.00×10^{-5})

** The numbers outside parentheses are most plausible case; the numbers inside parentheses are worst case.

TABLE 4A

SUMMARY OF ESTIMATED DOSE RATES FROM SOIL - CARCINOGENS

COMPOUND	SOIL ROUTE - HOT SPOT (DRUM SAMPLES)			
	CONCENTRATION, ng/g	DOSE RATE = mg/kg-DAY		
		ACCIDENTAL INGESTION	DERMAL CONTACT	TOTAL
2,3,7,8,-TCDD	Average: 3.34×10^{1}	8.85×10^{-10} (5.04×10^{-9})**	1.66×10^{-8} (9.47×10^{-8})	1.75×10^{-8} (9.97×10^{-8})
	Geometric Mean: 7.91×10^{0}	2.10×10^{-10} (5.04×10^{-9})	3.94×10^{-9} (9.47×10^{-8})	4.15×10^{-9} (9.97×10^{-8})
Equivalent Dioxins and Furans	Average: 3.87×10^{1}	1.03×10^{-9} (5.20×10^{-9})	1.93×10^{-8} (9.77×10^{-8})	2.03×10^{-8} (1.03×10^{-7})
	Geometric Mean: 1.80×10^{1}	4.79×10^{-10} (5.20×10^{-9})	8.99×10^{-9} (9.77×10^{-8})	9.47×10^{-9} (1.03×10^{-7})
Tetrachloroethene	Average: 8.0×10^{0}	8.16×10^{-10} (1.02×10^{-9})	1.33×10^{-7} (1.66×10^{-7})	1.39×10^{-7} (1.67×10^{-7})
	Geometric Mean: 7.75×10^{0}	7.91×10^{-10} (1.02×10^{-9})	1.29×10^{-7} (1.66×10^{-7})	1.29×10^{-7} (1.67×10^{-7})
2,4,6-Trichloro-phenol	Average: 3.80×10^{3}	3.88×10^{-7} (3.88×10^{-7})	6.32×10^{-5} (6.32×10^{-5})	6.36×10^{-5} (6.36×10^{-5})
	Geometric Mean: 3.80×10^{3}	3.88×10^{-7} (3.88×10^{-7})	6.32×10^{-5} (6.32×10^{-5})	6.36×10^{-5} (6.36×10^{-5})

** The numbers outside parentheses are the most plausible case; the numbers inside parentheses are the worst case.

NOTE: The Dioxin/Furan calculations were based on 1989 toxicity equivalence and proportionality factors (see Appendix B). If the 1987 toxicity equivalence factors were used, the average concentration would be 34.81 ng/g and the plausible case dose rate would be 1.83×10^{-8}; the worst case dose rate would be 1.03×10^{-7}.

TABLE 4B

SUMMARY OF ESTIMATED DOSE RATES FROM SOIL - CARCINOGENS

COMPOUND	SOIL ROUTE - LANDFILL EXCLUDING HOT SPOT			
	CONCENTRATION, ng/g	DOSE RATE = mg/kg-DAY		
		ACCIDENTAL INGESTION	DERMAL CONTACT	TOTAL
2,3,7,8-TCDD	Average: 5.1×10^{-1}	1.36×10^{-11} (5.57×10^{-10})**	2.55×10^{-10} (1.05×10^{-8})	2.68×10^{-10} (1.11×10^{-8})
	Geometric Mean: 3.0×10^{-2}	7.87×10^{-13} (5.57×10^{-10})	1.48×10^{-11} (1.05×10^{-8})	1.82×10^{-11} (1.11×10^{-8})
Equivalent Dioxins and Furans	Average: 8.7×10^{-1}	2.30×10^{-11} (7.06×10^{-10})	4.32×10^{-10} (1.33×10^{-8})	4.55×10^{-10} (1.40×10^{-8})
	Geometric Mean: 4.0×10^{-2}	1.19×10^{-12} (7.06×10^{-10})	2.23×10^{-11} (1.33×10^{-8})	2.35×10^{-11} (1.40×10^{-8})
Arsenic	Average: 1.11×10^{4}	1.13×10^{-6} (2.95×10^{-6})	---	1.13×10^{-6} (2.95×10^{-6})
	Geometric Mean: 9.84×10^{3}	1.00×10^{-6} (2.95×10^{-6})	---	1.00×10^{-6} (2.95×10^{-6})

** The numbers outside parentheses are the most plausible; the numbers inside parentheses are the worst case.

NOTE: Carcinogenic PAH were found in one sample. For that location, the additional dose rate would be 8.02×10^{-6} mg/kg-day.

The Dioxin/Furan calculations were based on 1989 toxicity equivalence and proportionality factors (see Appendix B). If the 1987 toxicity equivalence factors were used, the average concentration would be 0.53 ng/g and the plausible case dose rate would be 2.80×10^{-10}; the worst case dose rate would be 1.17×10^{-8}.

TABLE 4C

SUMMARY OF ESTIMATED DOSE RATES FROM SOIL - CARCINOGENS

COMPOUND	SOIL ROUTE - OFF-SITE (OS) RESIDENTIAL AREA			
	CONCENTRATION, ng/g	DOSE RATE = mg/kg-DAY		
		ACCIDENTAL INGESTION	DERMAL CONTACT	TOTAL
4,4'-DDT	Average: 4.4×10^{1}	2.24×10^{-9} (2.24×10^{-9})	3.66×10^{-7} (3.66×10^{-7})	3.68×10^{-7} (3.68×10^{-7})
	Geometric Mean: 4.4×10^{1}	2.24×10^{-9} (2.24×10^{-9})	3.66×10^{-7} (3.66×10^{-7})	3.68×10^{-7} (3.68×10^{-7})
Arsenic	Average: 1.02×10^{4}	5.83×10^{-5} (1.09×10^{-4})	---	5.83×10^{-5} (1.09×10^{-4})
	Geometric Mean: 9.64×10^{3}	5.5×10^{-5} (1.09×10^{-4})	---	5.5×10^{-5} (1.09×10^{-4})

** The numbers outside parentheses are the most plausible; the numbers inside parentheses are the worst case.

NOTE: Carcinogenic PAH were found in one sample. For that location, the additional dose rate would be 4.12×10^{-5}.
Doses quoted for a 2-6 year-old child.

TABLE 5

ACTUAL AND HYPOTHETICAL DOSE RATES FROM SOIL DUE TO 2,3,7,8-TCDD

EXPOSURE SCENARIO	CONCENTRATION, ng/g	SOIL ROUTE - DOSE RATES, mg/kg-day		
		INGESTION	DERMAL	TOTAL
Child in Off-Site Backyard***	Average: 7.10×10^{-3}	1.05×10^{-11} (2.15×10^{-11})**	5.90×10^{-12} (1.20×10^{-11})	1.64×10^{-11} (3.35×10^{-11})
	Geometric Mean: 6.60×10^{-3}	9.81×10^{-12} (2.15×10^{-11})	5.48×10^{-12} (1.20×10^{-11})	1.53×10^{-11} (3.35×10^{-11})
Adult Gardening in Off-Site Backyard***	Average: 7.10×10^{-3}	9.40×10^{-13} (1.92×10^{-12})	1.77×10^{-11} (3.61×10^{-11})	1.86×10^{-11} (3.80×10^{-11})
	Geometric Mean: 6.60×10^{-3}	8.74×10^{-13} (1.92×10^{-12})	1.65×10^{-11} (3.61×10^{-11})	1.74×10^{-11} (3.80×10^{-11})
Teenager (near hot spot)	Average: 3.34×10^{1}	8.85×10^{-10} (5.04×10^{-9})	1.66×10^{-8} (9.47×10^{-8})	1.75×10^{-8} (9.97×10^{-8})
	Geometric Mean: 7.91×10^{0}	2.10×10^{-10} (5.04×10^{-9})	3.94×10^{-9} (9.47×10^{-8})	4.15×10^{-9} (9.97×10^{-8})
Teenager (elsewhere on landfill)	Average: 5.1×10^{-1}	1.36×10^{-11} (5.57×10^{-10})	2.55×10^{-10} (1.05×10^{-8})	2.69×10^{-10} (1.11×10^{-8})
	Geometric Mean: 3.0×10^{-2}	7.87×10^{-13} (5.57×10^{-10})	1.48×10^{-11} (1.05×10^{-8})	1.56×10^{-11} (1.11×10^{-8})

** The numbers outside parentheses are the most plausible case; the numbers inside parentheses are the worst case.

*** All values are hypothetical, based on one-half of the analytical detection limits. No 2,3,7,8-TCDD were actually found off-site.

TABLE 6

ACTUAL AND HYPOTHETICAL DOSE RATES FROM SOIL DUE TO DIOXINS AND FURANS

EXPOSURE SCENARIO	CONCENTRATION, ng/g	SOIL ROUTE - DOSE RATES, mg/kg-day		
		INGESTION	DERMAL	TOTAL
Child in Off-Site Backyard***	Average: 1.0×10^{-2}	1.53×10^{-11} (3.03×10^{-11})	8.60×10^{-12} (1.70×10^{-11})	2.39×10^{-11} (4.73×10^{-11})
	Geometric Mean: 1.0×10^{-2}	1.41×10^{-11} (3.03×10^{-11})	7.89×10^{-12} (1.70×10^{-11})	2.20×10^{-11} (4.73×10^{-11})
Adult Gardening in Off-Site Backyard***	Average: 1.0×10^{-2}	1.37×10^{-12} (2.71×10^{-12})	2.57×10^{-11} (5.09×10^{-11})	2.71×10^{-11} (5.36×10^{-11})
	Geometric Mean: 1.0×10^{-2}	1.26×10^{-12} (2.71×10^{-12})	2.37×10^{-11} (5.09×10^{-11})	2.50×10^{-11} (5.36×10^{-11})
Teenager (near hot spot)	Average: 3.87×10^{1}	1.03×10^{-9} (5.20×10^{-9})	1.93×10^{-8} (9.77×10^{-8})	2.03×10^{-8} (1.03×10^{-7})
	Geometric Mean: 1.80×10^{1}	4.79×10^{-10} (5.20×10^{-9})	8.99×10^{-9} (9.77×10^{-8})	9.47×10^{-9} (1.03×10^{-7})
Teenager (elsewhere on landfill)	Average: 8.7×10^{-1}	2.30×10^{-11} (7.06×10^{-10})	4.32×10^{-10} (1.33×10^{-8})	4.55×10^{-10} (1.40×10^{-8})
	Geometric Mean: 4.0×10^{-2}	1.19×10^{-12} (7.06×10^{-10})	2.23×10^{-11} (1.33×10^{-8})	2.35×10^{-11} (1.40×10^{-8})

** The numbers outside parentheses are the most plausible case; the numbers inside parentheses are the worst case.

*** All values are hypothetical, based on one-half of the analytical detection limits. No 2,3,7,8-TCDD was actually found off-site.

NOTE: The dioxin/furan calculations were based on 1989 toxicity equivalence and proportionality factors. If the 1987 toxicity equivalence factors were used, the average concentations and dose rates for plausible and worst cases would be as follows:

EXPOSURE SCENARIO	CONCENTRATION	TOTAL DOSE RATE
Child in Off-Site Backyard***	8.92×10^{-3}	2.07×10^{-11} (3.34×10^{-11})
Adult Gardening in Off-Site Backyard***	8.92×10^{-3}	2.34×10^{-11} (3.78×10^{-11})
Teenager (Near Hot Spot)	3.48×10^{1}	1.83×10^{-8} (1.03×10^{-7})
Teenager (Elsewhere on Landfill)	5.33×10^{-1}	2.80×10^{-10} (1.17×10^{-8})

(a) the on-site drum area ("hot spot");

(b) the landfill excluding the on-site drum area (landfill excluding "hot spot"); and

(c) the off-site (OS) residential areas near the landfill where soils and residential wells were sampled.

Sample calculations for dose rates, including case-specific assumptions, are provided in Appendix C.

Assumptions used to estimate the "plausible maximum" exposure associated with dermal contact include:

- The amount of soil in contact with the skin is 2.77 mg/cm^2-day for clay soil (U.S. EPA, 1988). The choice of data for clay-like soil is based on actual field classification of soil types by geologists during the remedial investigation.

- Unless otherwise known, one hundred percent of a compound is assumed to be absorbed through the skin (McLaughlin, 1984). For dioxin, three percent of the compound is absorbed through the skin (Schaum, 1984). Ten percent of the pesticides are absorbed through the skin (McLaughlin, 1984). Negligible dermal absorption is assumed for inorganics (personal communication, Schaum, EPA, Washington, DC, August 22, 1989).

- Assumed body weights are:

  Adult - 70 kg
  Teenager - 49 kg
  6-12 year old child - 30 kg
  2-6 year old child - 10 kg

- An expected lifetime is 70 years.

Assumptions used to estimate the "plausible maximum" exposure associated with accidental ingestion of contaminated soils include:

- Exposure durations are 1,825 days for a 2-6 year old child; 1440 days for a 6-12 year old grade schooler; and 2,555 days for a teenager; and 18,250 days for an adult (Schaum, 1984). These exposure duration assumptions are based on a knowledge of site conditions derived from personal observations, discussion with RI field team investigators and the rather temperate climate in Arkansas. That is, it was assumed that a teenager could, conservatively, frequent the landfill for eight months out of the year.

- 0.8 g/day of soil is ingested by a 2-6 year old child; 0.05 g/day of soil is ingested by an adult or teenager (Schaum, 1984). 0.1 g/day is ingested by a 6-12 year old grade schooler (U.S. EPA, 1989b). 0.8 g/day is considered to be an upper bound. Recent guidance recommended 0.2 g/day for a child of 1-6 years and 0.1 g/day for adults as soil ingestion rates (U.S. EPA, 1989b). The soil ingestion rates used in the analyses were tailored to site conditions and scenarios.

- Unless otherwise known, one hundred percent of a compound is assumed to be absorbed through the gastrointestinal

tract (personal communication, John Schaum, EPA, Washington, DC, August 22, 1989). For dioxin, assume twenty-six percent is absorbed through the gastrointestinal tract (Schaum, 1984).

- o Body weights and expected lifetime are as shown above for dermal contact.

Dose rates were calculated using the arithmetic mean (average), geometric mean and maximum concentrations to provide a range of exposure estimates. Geometric means were used to provide a better representation than arithmetic means for soil data that is skewed. The geometric mean is the "n" root of the product of "n" numbers. For skewed distributions (i.e., data with a few outlier values that are higher than most other recorded values), an arithmetic meancalculation is disproportionately affected by the outliers and a higher "average" is calculated. Geometric means are not as strongly influenced by outliers and, therefore, more accurately indicate the most common value (e.g., the most likely concentration encountered). All detected compounds having RfDs or q*s were considered in evaluating the degree of contamination for on-site and off-site exposures.

For 2,3,7,8-TCDD and congeners and isomers of dioxins and furans, dose rates were calculated for a child, an adult and a teenager using three scenarios to determine if any particular receptor group might be subject to increased exposure (Tables 5 and 6) and risk. Dose rates were calculated for congeners and isomers of dioxins and furans using the toxicity equivalent factors (TEFs) and methodology, as outlined in Appendix B. Also, for other compounds, dose rates, risks and hazard indices were quantified for the adult, teenager and child; results were quoted in the tables for those having increased exposure and risk.

Dose rates and risks were not presented for a 6-12 year old grade schooler because their total dose rates and risks would always be significantly lower than those quoted for a 2-6 year old child. For example, the accidental ingestion dose rate would decrease by 3.3% and the dermal exposure dose rate would decrease by 38% compared to the 2-6 year old child. This reduction primarily occurs because there is a reduction in ingestion rate and exposure duration and an increase in body weight.

4.4.3 SURFACE WATER ROUTE

Local residents may be exposed to hazardous constituents via direct contact with contaminated surface waters. Comparisons of inorganic contaminant concentrations in the surface waters to AWQC for protection of human health are presented in Table 7. Inorganic contaminant concentrations for manganese and beryllium in an off-site drainage ditch, the landfill trenches and unnamed pond exceed AWQC for protection of human health (due to ingestion of water or fish). A complete table of ARARs is in Appendix G.

The inorganics in the surface water should not present a dermal hazard because these inorganic contaminants would not penetrate the skin nor could they cause defatting. Further, accidental ingestion would not present a hazard because the low concentrations of the inorganics are not acutely toxic and continuous ingestion of these water sources is a very unlikely scenario. There are no organic compounds that exceed ARARs, such as AWQC, and health advisories in the ambient surface waters.

Ingestion of fish should not be a problem because there are no known fish in the off-site ditches and on-site trenches. There is, however, concern for human receptors who eat fish from the

unnamed pond because of the presence of certain inorganics which exceed ambient water quality criteria (see Table 7). The unnamed pond is in the residential area and people could conceivably consume the fish.

TABLE 7

SURFACE WATER - AWQC EXCEEDANCES

JACKSONVILLE LANDFILL

Compound	Measured Concentration (µg/l)	AWQC (µg/l)		
		Ingestion of Biota* Only	Ingestion of Water** Only	Ingestion of Water & Fish*
Mn	336 (a)	---	10	0.144
	109 (b)	---	10	0.144
	193 - 1,880 (c)	---	10	0.144
	128 - 395 (d)	---	10	0.144
Be	0.2 (a)	0.117	0.0039	0.0068
	0.1 - 0.2 (d)	0.117	0.0039	0.0068

NOTES: (a) Bayou Two Prairie water (background sample).
(b) Water from drainage ditch at entrance to Holland Bottoms Wildlife Management Area.
(c) Water from landfill trenches JK-TW-01 through JK-TW-15.
(d) Off-site surface water samples from drainage ditches adjacent to the landfill and an unnamed pond.

Sources: * IRIS, 1988 and 1989.
** PhRED, 1988. For Be, AWQC value for ingestion of water and fish is given as 3.7×10^{-3} µg/l.

As Table 7 shows, beryllium and manganese are the two contaminants which are in excess of AWQC in the unnamed pond. Beryllium is of some concern because it has been shown to be carcinogenic in animals, and has been classified as a probable human carcinogen (IRIS, 1989). In the United States, concentrations of beryllium in the drinking water range from 0.01 to 1.22 ug/l, with a mean value of 0.19 ug/l (Kopp and Kroner, 1967). The measured concentrations on or near the

Jacksonville landfill surface water sources are within this range. Manganese standards are primarily based on the undesirable taste and discoloration of water, not on health effects. As with beryllium, the measured manganese concentrations on or near the landfill are within the 0.3 to 3230 ug/l (mean of 59 ug/l) range found in the United States (Kopp and Kroner, 1967).

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5.0 RISK CHARACTERIZATION

Risk characterization is the process of estimating the incidence of health effects under various conditions of human exposure as described in the Chapter 4 exposure assessment. It is performed by combining the exposure and dose-response assessments. Toxicity information and exposure estimates are combined to formulate conclusions regarding the health risks from the site. Quantitative risk estimates give an indication of the potential magnitude of adverse health effects resulting from exposure to toxic substances.

Contaminants are separated for quantitation purposes into those that can cause carcinogenic and noncarcinogenic effects. An excess lifetime cancer risk is calculated for exposure to carcinogens because of the prevailing scientific theory that even a small amount of a carcinogen can evoke changes in a cell or cells, leading to cancer over the 70-year lifetime of that individual. Many scientists believe there is no threshold for a carcinogen or safe exposure level that is without some effect. For this report, calculations are provided for excess cancer risks associated with given concentrations of the carcinogens. For noncarcinogenic effects, there is a reserve capacity or a threshold that must be exceeded before certain adverse effects are observed. The cell may have, for instance, metabolizing enzymes that modify the contaminant and allow small amounts of the contaminant to be tolerated by the organism. For noncarcinogenic effects, it is appropriate to calculate hazard indices. Both of these effects will be addressed in the following sections.

5.1 NONCARCINOGENIC EFFECTS

Qualitative and quantitative evaluations are usually performed for noncarcinogens. They are conducted in the following general manner:

- A comparison of observed or estimated environmental concentrations is made to relevant standards, criteria or guidelines presented in the ARARs table (Appendix G).

- A comparison of estimated dose rates calculated in the previous section is made to the Acceptable Daily Intake (ADI) or Reference Dose (RfD). A summary of the ADIs or RfDs for noncarcinogens is presented in Appendix G. Tables 8 and 10 compare the RfDs to the measured dose rates for each of the chemicals in the groundwater and soil, respectively. RfDs may be compared to monitoring well contaminant concentrations in Table 8A and to residential contaminant concentrations in Table 8B. Chronic RfDs were used in these calculations in lieu of subchronic RfDs because it was assumed realistic exposures were likely to occur for many years rather than for a short-term exposure duration.

For noncarcinogens, a comparison is made between acceptable dose rates, called acceptable daily intakes (ADI) or reference doses (RfD), and the monitored concentrations (after conversion to equivalent dose rates). This comparison is made by calculating hazard indices (HI) for each chemical having an ADI or RfD, as follows:

HI = hazard index

$$HI = \frac{E}{AL}$$

where E = Expected exposure, mg/kg - day

AL = Acceptable level = RfD or ADI, mg/kg - day

The monitored concentration is converted to a dose rate, i.e., mg/kg-day, and used as the numerator in calculating HI. If the hazard index is greater than 1, there is a potential health risk. For chemical mixtures, the total hazard index is calculated, as follows:

$$HI\ Total = \sum \frac{E}{AL}$$

Using this equation, the HI total may exceed 1, even if no single chemical exceeds its reference dose. In these cases, toxicity data is reviewed, and the chemicals are segregated in the mixture according to similar target organs or physiological effects. New hazard indices can be calculated for each group having similar effects; values that exceed 1 indicate a potential health risk.

A discussion of noncarcinogenic effects at the Jacksonville Landfill associated with exposure routes of concern follows.

5.1.1 GROUNDWATER ROUTE

5.1.1.1 Ingestion

A dose estimate was calculated for ingestion of groundwater based on analytical results of monitoring and residential wells. This groundwater is not consumed by residents and these

calculations are therefore only hypothetical. However, they indicate potential future risk if either the residential or monitoring wells were used or groundwater contaminants migrate downgradient to a well using the aquifer for potable water consumption.

None of the estimated dose rates calculated from organic analyses of monitoring and residential wells exceed RfDs for ingestion and, therefore, all HIs are less than 1.0 for organics. Refer to Table 8 for dose rate estimates and RfDs used to calculate HIs. For inorganics, HIs exceed 1.0 for chromium (HI = 1.13 for monitoring well JK-GW-12) and antimony (HI = 2.39 for residential well JK-RW-01 and 2.26 for residential well JK-RW-02).

Acute and subchronic toxic effects associated with ingestion of contaminated groundwater in the monitoring and residential well samples can be estimated by comparing the observed concentrations with maximum contaminant levels (MCLs) and health advisories (HA) (Table 9A). Monitored concentrations for the wells are shown in Tables 9A and 9B if that concentration exceeded an allowable concentration. Background and blank comparisons are also presented in the tables. They indicate that, for some inorganics such as aluminum, manganese, and mercury, measured concentrations are in the same order of magnitude as the respective background concentration.

In the monitoring wells, for organics: chlorobenzene exceeded the proposed MCLG and MCL (100 ug/l) and the lifetime HA (300 ug/l); 1,1-dichloroethene exceeded the MCL and lifetime HA both 7 ug/l); benzene exceeded the MCL (5 ug/l). For inorganics: barium exceeded the MCL (1,000 ug/l), proposed MCL and MCLG (5,000 ug/l) and HA (1500 ug/l); total chromium exceeded the MCL (50 ug/l) and proposed MCLG (100 ug/l); manganese exceeded the SMCL (50

ug/l); selenium exceeded the MCL (10 ug/l) and propoesd MCLG (50 ug/l) (Table 9A). Aluminum exceeded the SMCL (50 ug/l). SMCLs are established for aesthetic and similar reasons; therefore, they do not imply that a health risk exists.

In the residential wells, benzene exceeded the MCL (5 ug/l). For inorganics: aluminum exceeded the SMCL (50 ug/l); manganese exceeded the SMCL (50 ug/l); mercury exceeded the MCL (2.0 ug/l) and MCLG (3.0 ug/l) (Table 9B).

Based on these comparisons, chronic toxic effects may result from long-term repeated ingestion of groundwater in the contaminated monitoring and residential wells at the detected pollutant concentrations if they were ever used as potable water sources.

TABLE 8A

SUMMARY OF ESTIMATED DOSE RATES FROM GROUNDWATER - NONCARCINOGENS

GROUNDWATER ROUTE - MONITORING WELL												
INGESTION (DOSE RATE = mg/kg/day)												
Compound	JK GW-09	JK GW-13	JK GW-04	JK GW-05	JK GW-07	JK GW-10	JK GW-12	JK GW-01	JK GW-02	JK GW-06	JK GW-03	RfD
chlorobenzene	2.29×10^{-4} @ 8 ug/l	1.43×10^{-4} @ 5 ug/l	2.83×10^{-2} @ 990 ug/l	2.69×10^{-2} @ 940 ug/l	1.09×10^{-3} @ 38 ug/l	---	3.14×10^{-4} @ 11 ug/l	---	---	---	---	3.0×10^{-2}
benzoic acid	1.71×10^{-4} @ 6 ug/l	---	---	---	---	---	8.57×10^{-4} @ 30 ug/l	---	---	---	---	4.0×10^{0}
xylene	---	---	---	---	---	2.00×10^{-4} @ 7 ug/l	---	---	2.29×10^{-4} @ 8 ug/l	---	---	2.0×10^{0}
phenol	---	--	---	---	---	---	5.14×10^{-4} @ 18 ug/l	---	---	---	---	6.0×10^{-1}
barium	3.11×10^{-2} @ 1,090 ug/l	3.14×10^{-2} @ 1,110 ug/l	---	2.75×10^{-2} @ 964 ug/l	---	---	---	---	---	---	---	5×10^{-2}
cadmium	---	---	---	---	---	---	---	---	---	1.14×10^{-4} @ 4 ug/l	---	5×10^{-4} (water)
chromium	---	---	---	---	---	---	5.63×10^{-3} @ 197 ug/l	---	---	---	---	5×10^{-3}
manganese	---	---	---	1.19×10^{-1} @ 4,170 ug/l	---	---	---	---	---	1.84×10^{-2} @ 643 ug/l	5.2×10^{-2} @ 1,820 ug/l	2×10^{-1}
selenium	---	---	---	4.86×10^{-4} @ 17 ug/l	---	---	5.71×10^{-5} @ 2 ug/l	---	7.14×10^{-5} @ 2.5 ug/l	5.14×10^{-4} @ 18 ug/l	---	3×10^{-3}

Values shown are calculated dose rates for the concentration of contaminant found in each well. The corresponding RfD is shown in the far right hand column.

TABLE 8A (Continued)

SUMMARY OF ESTIMATED DOSE RATES FROM GROUNDWATER - NONCARCINOGENS

GROUNDWATER ROUTE - MONITORING WELL										
INHALATION (DOSE RATE = mg/kg-day)										
Compound	JK GW-09	JK GW-13	JK GW-04	JK GW-05	JK GW-07	JK GW-10	JK GW-12	JK GW-01	JK GW-02	RfD
chlorobenzene	1.70×10^{-4} @ 8 ug/l	1.06×10^{-4} @ 5 ug/l	2.10×10^{-2} @ 990 ug/l	1.99×10^{-2} @ 940 ug/l	8.06×10^{-4} @ 38 ug/l	---	2.33×10^{-4} @ 11 ug/l	---	---	5.0×10^{-3}
benzoic acid	1.27×10^{-4} @ 6 ug/l	---	---	---	---		6.36×10^{-4} @ 30 ug/l	---	---	---
Xylene	---	---	---	---	---	1.49×10^{-4} @ 7 ug/l	---	---	1.70×10^{-4} @ 8 ug/l	---
phenol	---	---	---	---	---	---	3.82×10^{-4} @ 18 ug/l	---		--

Values shown are calculated dose rates for the concentration of contaminant found in each well. The corresponding RfD is shown in the far right hand column.

TABLE 8B

SUMMARY OF ESTIMATED DOSE RATES FROM GROUNDWATER - NONCARCINOGENS

Groundwater Route - Residential Well (mg/kg-day)					
	Ingestion			Inhalation	
Compound	JK RW-02	JK-RW-01	RfD (mg/kg-day)	JK RW-02	RfD (mg/kg-day)
Chlorobenzene	5.14×10^{-4} @ 18 ug/l	---	3.0×10^{-2}	3.82×10^{-4} @ 18 ug/l	5.0×10^{-3}
Antimony	9.06×10^{-4} @ 31.7 ug/l	9.57×10^{-4} @ 33.5 ug/l	4×10^{-4}	---	---
Mercury	2.34×10^{-4} @ 8.2 ug/l	---	3×10^{-4}	---	---
Silver	---	4.31×10^{-4} @ 15.1 ug/l	3×10^{-3}	---	---

SOURCE: IRIS (= Integrated Risk Information System), 1988 and 1989.

Values shown are calculated dose rates for ingestion and inhalation of contaminants found in each residential well sampled. Corresponding RfD's are shown to the right.

TABLE 9A

GROUNDWATER - MONITORING WELLS
COMPARISON OF MEASURED CHEMICAL CONCENTRATIONS TO BACKGROUND LEVELS
AND APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs)

Compound	Location	Measured Concentration (ug/l)*	Background Concentration (ug/l)**		Allowable Concentration (ug/l)					
			Average	Highest	MCL	PMCL	MCLG	PMCLG	SMCL	HA
Chlorobenzene	JK-GW-04 JK-GW-05	990 940	undetectable	undetectable		100 100		100 100		300 lifetime 300 lifetime
1,1-Dichloroethene	JK-GW-04	11	undetectable	undetectable	7		7			7 lifetime
Benzene	JK-GW-05	15	undetectable	undetectable	5					
Aluminum	JK-GW-01 through JK-GW-13	1,030 - 12,600	8,195	9,370					50	
Barium	JK-GW-09 JK-GW-13	1,090 1,110	130	155	1,000 1,000	5,000 5,000		5,000 5,000		1,500 child and adult, acute and lifetime
Chromium, Total	JK-GW-12	197	21	24	50	100		100		
Manganese	JK-GW-01 through JK-GW-13, excluding JK-GW-08 & 12	76 - 4,170	125	138					50	
Selenium	JK-GW-05 JK-GW-06	17 18	---	---	10 10	50 50		50 50		

NOTES: Secondary Maximum Contaminant Levels (SMCL) "apply to any contaminant in drinking water that may adversely affect the odor or appearance of such water and consequently may cause a substantial number of persons served by public water systems providing such water to discontinue its use, or that may otherwise adversely affect public welfare."

SOURCES: Federal Register, Vol. 54, No. 97, Monday, May 22, 1989, p. 22,064.
IRIS (= Integrated Risk Information System), 1988 and 1989, IRIS Database, EPA, Washington, DC.
PhRED (= Public Health Evaluation Database), 1988, PhRED Database, EPA, Washington, DC

* Measured concentration represents the monitored concentration for the monitoring well specified or the range of monitored concentrations for the wells, if more than one well is concerned.

** For organic samples, field and lab method blanks were used for comparison with monitored well samples. Lab method blanks were undetectable for organics. For inorganic samples, groundwater monitoring wells, RR-GW-01 and 02, were used as background.

TABLE 9B

GROUNDWATER - RESIDENTIAL WELLS
COMPARISON OF MEASURED CHEMICAL CONCENTRATIONS TO BACKGROUND LEVELS AND APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs)

Compound	Location	Measured Concentration (ug/l)*	Background Concentration (ug/l)**			Allowable Concentration (ug/l)					
			Average	Geometric Mean	Highest	MCL	PMCL	MCLG	PMCLG	SMCL	MA
Benzene	JK-RW-02	6	undetectable	undetectable	undetectable	5					
Aluminum	JK-RW-01	441	346	25	943					50	
	JK-RW-03	943								50	
	JK-RW-04	524								50	
	JK-RW-05	18,400								50	
Manganese	JK-RW-02	181	119	81	184					50	
	JK-RW-03	95.2									
	JK-RW-04	340									
	JK-RW-05	522									
Mercury	JK-RW-02	8.2	2	2	8	2		3			

NOTES: Secondary Maximum Contaminant Levels (SMCL) "apply to any contaminant in drinking water that may adversely affect the odor or appearance of such water and consequently may cause a substantial number of persons served by public water systems providing such water to discontinue its use, or that may otherwise adversely affect public welfare."

SOURCES: Federal Register, Vol. 54, No. 97, Monday, May 22, 1989, p. 22,064.
IRIS (= Integrated Risk Information System), 1988 and 1989, IRIS Database, EPA, Washington, DC
PhRED (= Public Health Evaluation Database), 1988, PhRED Database, EPA, Washington, DC

* Measured concentration represents the monitored concentration for the residential well specified.

** For organic samples, lab method blanks were used for comparison with monitored well samples. Lab method blanks were undetectable for organics. For inorganic samples, residential wells, RW-01 and 03, were used as background.

5.1.1.2 Inhalation

Hazard indices due to inhalation were calculated using the monitored concentrations and RfDs presented in Table 8. Assumptions used for the calculations are shown in Appendix F. As with ingestion, these are only hypothetical since residents do not currently drink the water.

Hazard indices calculated for inhalation of chlorobenzene in monitoring wells JK-GW-04 and JK-GW-05 are 4.2 and 3.98, respectively. These hazard indices indicate that chronic exposure would be of concern if the volatilized water contaminants were inhaled. Since all other HIs for chlorobenzene in the remaining monitoring and residential wells are less than 1.0, noncarcinogenic effects are not expected to be of concern from the other wells.

Most of the other compounds detected in the monitoring wells do not have RfDs for inhalation. Some are inorganics and therefore they present no inhalation hazard due to lack of volatility. Organic compounds without RfDs were compared to OSHA permissible exposure limits (PELs), and American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit values (TLVs) (U.S. Department of Labor, 1989 and U.S. Department of Health and Human Services, 1987). All the monitored contaminants were found to be orders of magnitude below these standards, even assuming complete volatilization. Therefore, toxic effects from the noncarcinogenic chemicals listed in Table 8A due to inhalation are not expected.

5.1.1.3 Nonpotable Uses

There are no standards or criteria to evaluate the potential for health impacts associated with exposure to contaminated groundwater during nonpotable water-using activities. Inhalation

is an exposure pathway of possible concern under certain circumstances. Depending on the frequency and duration of exposure, frequent inhalation of volatilized contaminants from contaminated groundwater during car washing, lawn watering, etc., could potentially be associated with adverse chronic health impacts. This could occur for certain organic contaminants, such as chlorobenzene that exceeded the RfD for inhalation in two monitoring wells (Table 8A). However, car washing and lawn watering, the two most probable nonpotable uses, take place in unconfined spaces and infrequent intervals. Therefore, toxic concentrations from the pollutants detected in the wells would not occur as they do in a confined shower stall and adverse health effects would not be expected.

5.1.2 SOIL ROUTE

To evaluate the potential for chronic toxic effects associated with dermal exposure or accidental ingestion of contaminated surface soils, estimated exposure levels calculated in a previous section can be compared to RfDs. Table 10 shows this comparison for three different locations. These are the hot spot (drum area), landfill excluding hot spot, and off-site residential area. Offsite hazard indices for some chemicals for the plausible maximal exposure are greater than equivalent hazard indices onsite, in certain instances. This may be explained by the extremely variable nature of soil contaminants that produce correspondingly varying HIs.

Table 10 shows that all estimated exposure levels are below RfDs, except for 2,4,5-T; 2,4,5-TP; and 2,4-Dichlorophenol at the drum sample location. The hazard indices are 88.5 for 2,4,5-T (468: worst case); 3.45 for 2,4,5-TP (41.8: worst case); and 0.15 for 2,4-dichlorophenol (1.02: worst case). Based on this

comparison, only exposure via direct contact with the contents or soil near the drums is likely to be associated with systemic health effects. If the additive effects of these chemicals are considered, the total health hazard would be even greater. Hazard indices were summed for the soil route to account for the mixture of contaminants in the drums. The HI aggregates obtained by summing the individual HIs were 92.19 for the drum samples, 0.075 for the landfill excluding the hot spot and 0.041 for the off-site residential area. These summations confirm that adverse health effects are expected in the hot spot area due to chronic exposure to the drum samples from the herbicides, 2,4,5-T and 2,4,5-TP, since the HI aggregate is greater than 1 and that no adverse health effects are expected offsite due to the mixture of noncarcinogens in the residential areas.

TABLE 10A

SUMMARY OF HAZARD INDICES - SOILS - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE			
	"HOT SPOT" (DRUM SAMPLES)			
	CONCENTRATION, ng/g	TOTAL DOSE RATE (mg/kg-day)	HAZARD INDEX	RfD, mg/kg-day
2,4,5-T:	Average: 1.18×10^{7}	1.97×10^{0} (4.68×10^{0})**	1.97×10^{2}* (4.68×10^{2})*	1×10^{-2}
	Geometric Mean: 5.29×10^{6}	8.85×10^{-1} (4.68×10^{0})	8.85×10^{1}* (4.68×10^{2})*	
2,4,5-TP:	Average: 3.72×10^{5}	6.21×10^{-2} (3.34×10^{-1})	7.77×10^{0}* (4.18×10^{1})*	8×10^{-3}
	Geometric Mean: 1.65×10^{5}	2.76×10^{-2} (3.34×10^{-1})	3.45×10^{0}* (4.18×10^{1})*	
2,4-Dichlorophenol	Average: 5.56×10^{4}	1.01×10^{-3} (3.06×10^{-3})	3.40×10^{-1} (1.02×10^{0})*	3×10^{-3}
	Geometric Mean: 2.49×10^{4}	4.49×10^{-4} (3.06×10^{-3})	1.50×10^{-1} (1.02×10^{0})*	
tetrachloroethene	Average: 8.0×10^{0}	1.44×10^{-7} (1.8×10^{-7})	1.44×10^{-5} (1.8×10^{-5})	1×10^{-2}
	Geometric Mean: 7.8×10^{0}	1.37×10^{-7} (1.8×10^{-7})	1.37×10^{-5} (1.8×10^{-5})	
2,4,5-Trichloro-phenol	Average: 1.17×10^{6}	2.10×10^{-2} (5.41×10^{-2})	2.1×10^{-1} (5.4×10^{-1})	1×10^{-1}
	Geometric Mean: 4.96×10^{5}	8.93×10^{-3} (5.41×10^{-2})	9.0×10^{-2} (5.4×10^{-1})	
1,2,4-Trichloro-benzene	Average: 2.40×10^{3}	4.32×10^{-5} (1.24×10^{-4})	2.16×10^{-3} (6.22×10^{-3})	2×10^{-2}
	Geometric Mean: 5.24×10^{2}	9.44×10^{-6} (1.24×10^{-4})	4.72×10^{-4} (6.22×10^{-3})	

* Exposure > RfD

** The numbers outside parenthesis are most plausible case; the numbers inside parentheses are worst case.

TABLE 10A (Continued)

SUMMARY OF HAZARD INDICES - SOILS - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE			
	"HOT SPOT" (DRUM SAMPLES)			
	CONCENTRATION, ng/g	TOTAL DOSE RATE (mg/kg-day)	HAZARD INDEX	RfD, mg/kg-day
Naphthalene	Average: 1.10×10^{4}	1.98×10^{-4} (1.98×10^{-4})**	4.96×10^{-4} (4.96×10^{-4})	4×10^{-1}
	Geometric Mean: 1.10×10^{4}	1.98×10^{-4} (1.98×10^{-4})	4.96×10^{-4} (4.96×10^{-4})	
2-Chlorophenol	Average: 4.95×10^{2}	8.93×10^{-6} (1.21×10^{-5})	1.79×10^{-3} (2.42×10^{-3})	5×10^{-3}
	Geometric Mean: 4.63×10^{2}	8.34×10^{-6} (1.21×10^{-5})	1.67×10^{-3} (2.42×10^{-3})	
Benzoic Acid	Average: 3.53×10^{3}	6.36×10^{-5} (1.75×10^{-4})	1.59×10^{-5} (4.37×10^{-5})	4×10^{0}
	Geometric Mean: 1.09×10^{3}	1.96×10^{-5} (1.75×10^{-4})	4.91×10^{-6} (4.37×10^{-5})	
Phenol	Average: 2.40×10^{2}	4.33×10^{-6} (4.33×10^{-6})	7.21×10^{-6} (7.21×10^{-6})	6×10^{-1}
	Geometric Mean: 2.40×10^{2}	4.33×10^{-6} (4.33×10^{-6})	7.21×10^{-6} (7.21×10^{-6})	

* Exposure > RfD

** The numbers outside parenthesis are most plausible case; the numbers inside parentheses are worst case.

TABLE 10B

SUMMARY OF HAZARD INDICES - SOILS - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE			
	LANDFILL EXCLUDING "HOT SPOT"			
	CONCENTRATION, ng/g	TOTAL DOSE RATE (mg/kg-day)	HAZARD INDEX	RfD, mg/kg-day
2,4,5-T:	Average 4.50×10^{3} Geometric Mean 4.50×10^{3}	7.52×10^{-4} (7.52×10^{-4})** 7.52×10^{-4} (7.52×10^{-4})	7.52×10^{-2} (7.52×10^{-2}) 7.52×10^{-2} (7.52×10^{-2})	1×10^{-2}
barium	Average: 6.90×10^{2} Geometric Mean: 6.70×10^{2}	7.04×10^{-4} (8.40×10^{-4}) 6.90×10^{-4} (8.40×10^{-4})	1.41×10^{-2} (1.68×10^{-2}) 1.38×10^{-2} (1.68×10^{-2})	5×10^{-2}
beryllium	Average: $8.80 \times 10^{+2}$ Geometric Mean: $8.30 \times 10^{+2}$	8.98×10^{-7} (1.63×10^{-6}) 8.47×10^{-7} (1.63×10^{-6})	1.80×10^{-4} (3.27×10^{-4}) 1.69×10^{-4} (3.27×10^{-4})	5×10^{-3}
cadmium	Average: 8.03×10^{3} Geometric Mean: 4.96×10^{3}	8.19×10^{-6} (1.94×10^{-5}) 5.06×10^{-6} (1.94×10^{-5})	8.19×10^{-3} (1.94×10^{-2}) 5.06×10^{-3} (1.94×10^{-2})	1×10^{-3}
chromium, total	Average: 1.47×10^{5} Geometric Mean: 1.01×10^{5}	1.50×10^{-4} (4.95×10^{-4}) 1.03×10^{-4} (4.95×10^{-4})	2.99×10^{-2} (9.90×10^{-2}) 2.05×10^{-2} (9.90×10^{-2})	5×10^{-3}
manganese	Average: 2.55×10^{6} Geometric Mean: 2.39×10^{6}	2.60×10^{-3} (4.89×10^{-3}) 2.43×10^{-3} (4.89×10^{-3})	1.30×10^{-2} (2.44×10^{-2}) 1.22×10^{-2} (2.44×10^{-2})	2×10^{-1}
mercury	Average: $8 \times 10^{+2}$ Geometric Mean: $8 \times 10^{+2}$	8.16×10^{-7} (8.16×10^{-7}) 8.16×10^{-7} (8.16×10^{-7})	2.72×10^{-3} (2.72×10^{-3}) 2.72×10^{-3} (2.72×10^{-3})	3×10^{-4}

* Exposure > RfD

** The numbers outside parenthesis are most plausible case; the numbers inside parentheses are worst case.

TABLE 10B (Continued)

SUMMARY OF HAZARD INDICES - SOILS - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE			
	LANDFILL EXCLUDING "HOT SPOT"			
	CONCENTRATION, ng/g	TOTAL DOSE RATE (mg/kg-day)	HAZARD INDEX	RfD, mg/kg-day
nickel	Average: 3.59×10^{4}	3.66×10^{-5} (1.33×10^{-4})	1.83×10^{-3} (6.63×10^{-3})	2×10^{-2}
	Geometric Mean: 2.74×10^{4}	2.79×10^{-5} (1.33×10^{-4})	1.40×10^{-3} (6.63×10^{-3})	
silver	Average: 4.64×10^{3}	4.73×10^{-6} (1.94×10^{-5})	1.58×10^{-3} (6.46×10^{-3})	3×10^{-3}
	Geometric Mean: 3.59×10^{3}	3.66×10^{-6} (1.94×10^{-5})	1.22×10^{-3} (6.46×10^{-3})	
zinc	Average: 9.75×10^{5}	9.95×10^{-4} (1.10×10^{-2})	4.98×10^{-3} (5.51×10^{-2})	2×10^{-1}
	Geometric Mean: 3.74×10^{5}	3.81×10^{-4} (1.10×10^{-2})	1.91×10^{-3} (5.51×10^{-2})	

* Exposure > RfD

** The numbers outside parenthesis are most plausible case; the numbers inside parentheses are worst case.

TABLE 10C

SUMMARY OF HAZARD INDICES - SOILS - NONCARCINOGENIC EFFECTS

COMPOUND	SOIL ROUTE			
	OFF-SITE (OS) RESIDENTIAL AREA			
	CONCENTRATION, ng/g	TOTAL DOSE RATE (mg/kg-day)	HAZARD INDEX	RfD, mg/kg-day
4,4'-DDT	Average: 4.40×10^{1}	2.06×10^{-5} (2.06×10^{-5})**	4.12×10^{-2} (4.12×10^{-2})	5×10^{-4}
	Geometric Mean: 4.40×10^{1}	2.06×10^{-5} (2.06×10^{-5})	4.12×10^{-2} (4.12×10^{-2})	
beryllium	Average: $6.40 \times 10^{+2}$	5.12×10^{-5} (8.00×10^{-5})	1.02×10^{-2} (1.60×10^{-2})	5×10^{-3}
	Geometric Mean: $6.10 \times 10^{+2}$	4.88×10^{-5} (8.00×10^{-5})	9.76×10^{-3} (1.60×10^{-2})	

** The numbers outside parentheses are most plausible case; the numbers inside parentheses are worst case.

5.1.3 SURFACE WATER ROUTE

The presence of several inorganic contaminants detected in surface water samples is of concern. Inorganic contaminants in surface waters that exceed AWQC for the protection of human health include beryllium and manganese (see Table 7).

Inorganic contaminant concentrations in excess of AWQC have been detected in off-site surface water samples, in all trench water samples, in drainage ditches adjacent to the Jacksonville Landfill, in a drainage ditch at the entrance to Holland Bottoms Wildlife Management Area (a background sample), and in a small unnamed pond in the residential area between two houses adjacent to the landfill. Beryllium (0.2 ug/l in a drainage ditch adjacent to the landfill and 0.1 ug/l in the unnamed pond) exceeded AWQC for ingestion of biota (0.117 ug/l), ingestion of water (0.0039 ug/l) and ingestion of water and fish (0.0068 ug/l). Manganese (109 ug/l in the drainage ditch at the entrance to Holland Bottoms, 193 ug/l to 1,880 ug/l in all 15 trenches numbered JK-TW-01 through and including JK-TW-15 and 128 to 395 ug/l in drainage ditches adjacent to the Jacksonville Landfill and the unnamed pond) exceeded AWQC for ingestion of water (10 ug/l) and ingestion of water and fish (0.144 ug/l). There are no Arkansas surface water ARARs applicable to the major contaminants found in the surface water, such as herbicides, dioxins and furans and the above-named inorganics. Surface water samples did not exceed Arkansas surface water ARARs for concentrations of other detected inorganic contaminants, such as selenium and silver.

Residents have been observed fishing in the small pond adjacent to the landfill. Therefore, the previously cited AWQC for biota (fish) are applicable.

Based on these results, chronic toxic effects to humans are possible from manganese and beryllium if humans were to eat the fish in the unnamed pond on a continuous basis. This is, in reality, very unlikely. Continuous consumption of water from the pond, trenches or ditches, also an unlikely event, could also produce adverse effects. Even if local residents attempted to drink these surface waters, the bitter taste imparted by manganese would tend to make the water unpalatable.

5.2 CARCINOGENIC RISKS

Risks are estimated as a probability or a range of probabilities that a specific adverse effect will occur under conditions of exposure of the human population at risk. Risk is calculated using a "carcinogenic potency factor" [also known as q^* or unit cancer risk, (UCR)]. By definition, the q^* is the excess cancer risk due to a continuous lifetime exposure to one unit of carcinogenic concentration. Graphically, the q^* is the slope of the dose-response curve at low doses. Excess cancer risk is the added risk to that portion of the population in excess of the background tumor rate. It is the risk attributable to site-related chemicals.

Excess cancer risk at low doses is calculated, as follows:

$$\text{Excess Cancer Risk} = P = q^* \times d$$

where q^* = carcinogenic potency factor, $(\text{mg/kg-day})^{-1}$

d = dose rate = exposure level, mg/kg-day
= daily dose of a compound averaged over an individual's lifetime and body weight

P = Excess cancer risk, unitless

This equation is an approximation of the dose-response curve and is valid only for q^* x d values less than 0.1 (1×10^{-1}).

For an excess cancer risk of 1×10^{-6}, there is one chance in a million (1/1,000,000) that the exposed person will develop cancer.

For exposure to multiple chemicals, total excess cancer risk for the mixture is calculated by summing the individual excess cancer risks for each chemical to obtain the total excess risk for the site, as follows:

$P_{total} = \Sigma\ P_1 + P_2 + P_3 \ldots P_n$

P_1 = excess cancer risk for Chemical 1.

P_2 = excess cancer risk for Chemical 2.

P_3 = excess cancer risk for Chemical 3.

P_n = excess cancer risk for Chemical n.

P_{total} = excess cancer risk for chemical mixture.

Refer to Appendices C and F for sample calculations for obtaining the dose rates in mg/kg-day. Total excess cancer risks between 10^{-6} to 10^{-4} serve as action levels for EPA.

5.2.1 GROUNDWATER ROUTE

Local residents do not use the groundwater for drinking but obtain potable water from the City of Jacksonville. Cancer risks were calculated for ingestion of groundwater based on analytical results of monitoring and residential wells. These calculations are therefore hypothetical. However, they indicate potential future risk if either the residential wells were used or the groundwater contaminants were to migrate. Hypothetical risks associated with ingestion and inhalation are presented in Tables 11A and 11B. Sample calculations are provided in Appendix F.

TABLE 11A

SUMMARY OF ESTIMATED EXCESS LIFETIME CANCER RISKS* - GROUNDWATER

	GROUNDWATER ROUTE Monitoring Well			
	Ingestion			
Compound	JK-GW-03	JK-GW-04	JK-GW-05	JK-GW-12
1,1-dichloroethene	---	1.89×10^{-4}	---	---
1,4-dichlorobenzene	---	3.98×10^{-5}	---	---
benzene	---	---	1.24×10^{-5}	---
arsenic	7.35×10^{-4}	---	8.00×10^{-4}	1.95×10^{-4}
Total Risk	7.35×10^{-4}	2.29×10^{-4}	8.12×10^{-4}	1.95×10^{-4}

	GROUNDWATER ROUTE Monitoring Well	
	Inhalation	
Compound	JK-GW-04	JK-GW-05
1,1-dichloroethene	2.80×10^{-4}	---
1,4-dichlorobenzene	---	---
benzene	---	9.23×10^{-6}
arsenic	---	---
Total Risk	2.80×10^{-4}	9.23×10^{-6}

	GROUNDWATER ROUTE Monitoring Well			
	Inhalation and Ingestion			
Compound	JK-GW-03	JK-GW-04	JK-GW-05	JK-GW-12
1,1-dichloroethene	---	4.69×10^{-4}	---	---
1,4-dichlorobenzene	---	3.98×10^{-5}	---	---
benzene	---	---	2.16×10^{-5}	---
arsenic	7.35×10^{-4}	---	8.00×10^{-4}	1.95×10^{-4}
Total Risk	7.35×10^{-4}	5.09×10^{-4}	8.22×10^{-4}	1.95×10^{-4}

* All values are for excess lifetime cancer risk. For example, 8.00×10^{-4} excess cancer risk for arsenic means 8.00 excess lifetime cancer risks per 10,000 people.

TABLE 11B

SUMMARY OF ESTIMATED EXCESS LIFETIME CANCER RISKS - GROUNDWATER

GROUNDWATER ROUTE Residential Well			
	Ingestion	Inhalation	Total
Compound	JK-RW-02	JK-RW-02	JW-RW-02
benzene	4.97×10^{-6}	3.69×10^{-6}	8.66×10^{-6}
Total Risk	4.97×10^{-6}	3.69×10^{-6}	8.66×10^{-6}

5.2.1.1 Monitoring Wells

The hypothetical risk from contaminated groundwater, if residents were to ingest and inhale water from the monitoring wells, is summarized below:

On-Site Monitoring Well Groundwater Risks

	JK-GW-03	JK-GW-04	JK-GW-05	JK-GW-12
Ingestion of Contaminated Ground Water	7.35×10^{-4}	2.29×10^{-4}	8.12×10^{-4}	1.95×10^{-4}
Inhalation of Contaminated Groundwater During Showering	---	2.80×10^{-4}	9.23×10^{-6}	---
TOTAL RISK	7.35×10^{-4}	5.09×10^{-4}	8.21×10^{-4}	1.95×10^{-4}

This table presents the total excess quantifiable lifetime cancer risk due to measured trace carcinogens in the groundwater if it were ever ingested and inhaled. Since the residents do not drink this groundwater, the risks only represent a future possibility, not a current actuality.

5.2.1.2 Residential Wells

As with the monitoring wells, residential well water is not consumed by people. The total hypothetical range of lifetime carcinogenic risks associated with both ingestion and inhalation of contaminants detected in residential well (JK-RW-02) would be:

Off-Site Residential Well Groundwater Risks

	JK-RW-02
Ingestion of Contaminated Drinking Water	4.97×10^{-6}
Inhalation of Contaminated Groundwater During Showering	3.69×10^{-6}
TOTAL RISK	8.66×10^{-6}

Local residents reported that this well may be used for watering yards and providing drinking water for animals. There are no quantitative data to estimate risk associated with exposure during nonpotable uses of contaminated groundwater, but as discussed earlier, the hazards to health caused by nonpotable use are extremely minor.

Comparing onsite to offsite groundwater risks, a two order of magnitude difference is observed. On-site well water consumption would cause between 1.95 and 7.35 x 10^{-4} excess cancers (about 2 to 7 excess cancers per 10,000 population), whereas consumption of the offsite water would cause 8.66 x 10^{-6} cancers (about 9 excess cancers per 1,000,000 population).

5.2.2 SOIL ROUTE

Measured soil concentrations of 2,3,7,8-TCDD [7.91 ppb (= ng/g) using geometric mean and 33.36 ppb (= ng/g) using arithmetic mean in drum samples, Table 12] exceed 1 ppb set by CDC for residential soils that are subject to erosion and transport processes and 5-7 ppb in soils where the general public may have infrequent contact.

Excess lifetime cancer risk and dose rates for each chemical in the soil are listed in Table 12, along with the q^* values obtained in Appendix G. Sample calculations for calculating dose rates are provided in Appendix C. The individual excess lifetime cancer risks are summed to determine the total excess cancer risk for: the area where the drums of chemicals are located on the landfill ("hot spot"); the landfill excluding the "hot spot"; and the off-site residential backyards next to the landfill, where the soils were sampled and a residential well was monitored.

See Table 12 for the total excess lifetime cancer risks for each of these three areas. Risk addition assumes that individual intakes are small, there are no synergistic or antagonistic chemical interaction(s), individuals will be exposed to all contaminants detected and all of the compounds induce carcinogenic effects in humans (U.S. EPA, 1986).

The information contained in Table 12 was taken from Tables 13 and 14. Tables 13 and 14 indicate the excess lifetime cancer risks for three different scenarios calculated to determine if any particular age group is at increased risk due (1) to 2,3,7,8-TCDD and (2) to total dioxins and total furans, including the 2,3,7,8-TCDD. The scenarios are for (1) a child aged 2-6 years old who plays in a residential backyard, (2) an adult who gardens in a residential backyard near the landfill and (3) a teenager who plays near the drums or anywhere else on the landfill. Results indicate the teenager who plays near the drums is at increased risk with an excess cancer risk for 2,3,7,8-TCDD of 6.69×10^{-4} and a "worst case" risk using the highest detected concentration of 1.60×10^{-2}. The above calculations assume continuous, daily exposure. Total excess cancer risks between 10^{-6} and 10^{-4} serve as action levels for EPA (U.S. EPA, 1988).

TABLE 12A

SUMMARY OF ESTIMATED EXCESS LIFETIME CANCER RISKS - SOILS - CARCINOGENS

COMPOUND	SOIL ROUTE - "HOT SPOT" (DRUM SAMPLES)			
	CONCENTRATION, ng/g	TOTAL DOSE RATE (mg/kg-day)	q^*, $(mg/kg\text{-}day)^{-1}$	RISK
2,3,7,8,-TCDD	Average: 3.33×10^{1}	1.75×10^{-8} (9.97×10^{-8})**	1.56×10^{5}	2.72×10^{-3}* (1.56×10^{-2})*
	Geometric Mean: 7.91×10^{0}	4.15×10^{-9} (9.97×10^{-8})	1.56×10^{5}	6.47×10^{-4}* (1.56×10^{-2})*
Dioxins and Furans	Average: 3.87×10^{1}	2.03×10^{-8} (1.03×10^{-7})	1.56×10^{5}	3.17×10^{-3}* (1.61×10^{-2})*
	Geometric Mean: 1.80×10^{1}	9.47×10^{-9} (1.03×10^{-7})	1.56×10^{5}	1.48×10^{-3}* (1.61×10^{-2})*
Tetrachloroethene	Average: 8.00×10^{0}	1.39×10^{-7} (1.67×10^{-7})	5.1×10^{-2}	7.09×10^{-9} (8.52×10^{-9})
	Geometric Mean: 7.75×10^{0}	1.29×10^{-7} (1.67×10^{-7})	5.1×10^{-2}	6.60×10^{-9} (8.52×10^{-9})
2,4,6-Trichloro-phenol	Average: 3.80×10^{3}	6.36×10^{-5} (6.36×10^{-5})	2.0×10^{-2}	1.27×10^{-6} (1.27×10^{-6})
	Geometric Mean: 3.80×10^{3}	6.36×10^{-5} (6.36×10^{-5})	2.0×10^{-2}	1.27×10^{-6} (1.27×10^{-6})
TOTAL RISK, using Geometric Mean:				1.48×10^{-3}* (1.61×10^{-2})*

* Risk in excess of 10^{-4}

** The numbers outside parentheses are the most plausible case; the numbers inside parentheses are the worst case.

NOTE: "Dioxins and Furans" includes 2,3,7,8-TCDD.

The Dioxin/Furan calculations were based on 1989 toxicity equivalence and proportionality factors (see Appendix B). If the 1987 toxicity equivalence factors were used, the average concentration would be 34.81 ng/g and the plausible case risk would be 2.85×10^{-3}; the worst case risk would be 1.60×10^{-2}.

TABLE 12B

SUMMARY OF ESTIMATED EXCESS LIFETIME CANCER RISKS - SOILS - CARCINOGENS

COMPOUND	SOIL ROUTE - LANDFILL EXCLUDING "HOT SPOT"			
	CONCENTRATION, ng/g	TOTAL DOSE RATE (mg/kg-day)	q^*, $(mg/kg\text{-}day)^{-1}$	RISK
2,3,7,8-TCDD	Average: 5.1×10^{-1}	2.68×10^{-10} (1.11×10^{-8})**	1.56×10^{5}	4.18×10^{-5} (1.73×10^{-3})*
	Geometric Mean: 3.0×10^{-2}	1.82×10^{-11} (1.11×10^{-8})	1.56×10^{5}	2.83×10^{-6} (1.73×10^{-3})*
Dioxins and Furans	Average: 8.7×10^{-1}	4.55×10^{-10} (1.40×10^{-8})	1.56×10^{5}	7.10×10^{-5} (2.18×10^{-3})*
	Geometric Mean: 4.0×10^{-2}	2.35×10^{-11} (1.40×10^{-8})	1.56×10^{5}	3.66×10^{-6} (2.18×10^{-3})*
Arsenic	Average: 1.11×10^{4}	1.13×10^{-6} (2.95×10^{-6})	1.75×10^{0}	1.98×10^{-6} (5.16×10^{-6})
	Geometric Mean: 9.84×10^{3}	1.00×10^{-6} (2.95×10^{-6})	1.75×10^{0}	1.75×10^{-6} (5.16×10^{-6})
TOTAL RISK, using Geometric Mean:				5.41×10^{-6} (2.19×10^{-3})*

* Risk in excess of 10^{-4}

** The numbers outside parentheses are the most plausible case; the numbers inside parentheses are the worst case.

NOTE: Carcinogenic PAH were found in one sample; the calculated risk for that location is 9.22×10^{-5} and the total risk would be 9.62×10^{-5}.

"Dioxins and Furans" includes 2,3,7,8-TCDD.

The Dioxin/Furan calculations were based on 1989 toxicity equivalence and proportionality factors (see Appendix B). If the 1987 toxicity equivalence factors were used, the average concentration would be 0.5 ng/g and the plausible case risk would be 4.37×10^{-5}; the worst case risk would be 1.82×10^{-3}.

TABLE 12C

SUMMARY OF ESTIMATED EXCESS LIFETIME CANCER RISKS - SOILS - CARCINOGENS

COMPOUND	Soil Route - Off-Site (OS) Residential Area			
	CONCENTRATION, ng/g	TOTAL DOSE RATE (mg/kg-day)	q^*, $(mg/kg\text{-}day)^{-1}$	RISK
4,4'-DDT	Average: 4.40×10^{1}	3.68×10^{-7} (3.68×10^{-7})	3.4×10^{-1}	1.25×10^{-7} (1.25×10^{-7})
	Geometric Mean: 4.40×10^{1}	3.68×10^{-7} (3.68×10^{-7})	3.4×10^{-1}	1.25×10^{-7} (1.25×10^{-7})
Arsenic***	Average: 10.2×10^{3}	5.83×10^{-5} (1.09×10^{-4})	1.75×10^{0}	1.02×10^{-4}* (1.91×10^{-4})*
	Geometric Mean: 9.64×10^{3}	5.5×10^{-5} (1.09×10^{-4})	1.75×10^{0}	9.64×10^{-5} (1.91×10^{-4})*
TOTAL RISK, using Geometric Mean:				9.65×10^{-5} (1.91×10^{-4})*

* Risk in excess of 10^{-4}

** The numbers outside parentheses are the most plausible case; the numbers inside parentheses are the worst case.

*** The calculation does not reflect an increased cancer risk due to arsenic since the downgradient arsenic concentraiton equaled the upgradient concentration. The numbers reflect the background risk due to the naturally occurring concentrations of this inorganic substance.

NOTE: Carcinogenic PAH were found in one sample; the calculated risk for that one location is 4.74×10^{-4} and the total risk would be 5.71×10^{-4}.

For arsenic, risk quoted off-site is for a 2-6 year old child. For an adult off-site gardener, the risk is 9.10×10^{-6} for the arithmetic mean and 8.61×10^{-6} for the geometric mean, and 1.70×10^{-5} for the worst case.

"Dioxins and Furans" includes 2,3,7,8-TCDD.

TABLE 13

SUMMARY OF ESTIMATED EXCESS LIFETIME CANCER RISKS - SOILS: 2,3,7,8-TCDD

EXPOSURE SCENARIO	CONCENTRATION, ng/g	SOIL ROUTE	
		TOTAL DOSE RATE (mg/kg-day)	RISK
Child in Off-Site Backyard***	Average: 7.10×10^{-3}	1.64×10^{-11} (3.35×10^{-11})**	2.56×10^{-6} (5.23×10^{-6})
	Geometric Mean: 6.60×10^{-3}	1.53×10^{-11} (3.35×10^{-11})	2.39×10^{-6} (5.23×10^{-6})
Adult Gardening in Off-Site Backyard***	Average: 7.10×10^{-3}	1.86×10^{-11} (3.80×10^{-11})	2.90×10^{-6} (5.93×10^{-6})
	Geometric Mean: 6.60×10^{-3}	1.74×10^{-11} (3.80×10^{-11})	2.71×10^{-6} (5.93×10^{-6})
Teenager (near hot spot)	Average: 3.34×10^{1}	1.75×10^{-8} (9.97×10^{-8})	2.72×10^{-3}*(1.56×10^{-2})*
	Geometric Mean: 7.91×10^{0}	4.15×10^{-9} (9.97×10^{-8})	6.47×10^{-4}*(1.56×10^{-2})*
Teenager (elsewhere on landfill)	Average: 5.1×10^{-1}	2.69×10^{-10} (1.11×10^{-8})	4.20×10^{-5} (1.73×10^{-3})*
	Geometric Mean: 3.0×10^{-2}	1.56×10^{-11} (1.11×10^{-8})	2.43×10^{-6} (1.73×10^{-3})*

* Risks in excess of 10^{-4}

** The numbers outside parentheses are the most plausible case; the numbers inside parentheses are the worst case.

*** The values are hypothetical, based on one-half of the analytical detection limits. No 2,3,7,8-TCDD was actually found off-site.

TABLE 14

SUMMARY OF ESTIMATED EXCESS LIFETIME CANCER RISKS - SOILS: DIOXINS AND FURANS

EXPOSURE SCENARIO	CONCENTRATION, ng/g	SOIL ROUTE	
		TOTAL DOSE RATE (mg/kg-day)	RISK
Child in Off-Site Backyard***	Average: 1.0×10^{-2}	2.39×10^{-11} (4.73×10^{-11})	3.73×10^{-6} (7.38×10^{-6})
	Geometric Mean: 1.0×10^{-2}	2.20×10^{-11} (4.73×10^{-11})	3.43×10^{-6} (7.38×10^{-6})
Adult Gardening in Off-Site Backyard***	Average 1.0×10^{-2}	2.71×10^{-11} (5.36×10^{-11})	4.22×10^{-6} (8.36×10^{-6})
	Geometric Mean: 1.0×10^{-2}	2.50×10^{-11} (5.36×10^{-11})	3.89×10^{-6} (8.36×10^{-6})
Teenager (near hot spot)	Average: 3.87×10^{1}	2.03×10^{-8} (1.03×10^{-7})	3.17×10^{-3}*(1.61×10^{-2})*
	Geometric Mean: 1.80×10^{1}	9.47×10^{-9} (1.03×10^{-7})	1.48×10^{-3}*(1.61×10^{-2})*
Teenager (elsewhere on landfill)	Average: 8.7×10^{-1}	4.55×10^{-10} (1.40×10^{-8})	7.10×10^{-5} (2.18×10^{-3})*
	Geometric Mean: 4.0×10^{-2}	2.35×10^{-11} (1.40×10^{-8})	3.66×10^{-6} (2.18×10^{-3})*

* Risk in excess of 10^{-4}

** The numbers outside parentheses are the most plausible case; the numbers inside parentheses are the worst case.

*** These values are hypothetical, based on one-half of the analytical detection limits. No 2,3,7,8-TCDD was actually found off-site.

NOTE: The Dioxin/Furan calculations were based on 1989 toxicity equivalence and proportionality factors. If the 1987 toxicity equivalence factors were used, the average concentrations and risks for plausible and worse cases are as follows:

EXPOSURE SCENARIO	CONCENTRATION	RISK
Child in Off-Site Backyard***	8.92×10^{-3}	3.23×10^{-6} (5.21×10^{-6})
Adult Gardening in Off-Site Backyard***	8.92×10^{-3}	3.65×10^{-6} (5.90×10^{-6})
Teenager (Near Hot Spot)	3.48×10^{1}	2.85×10^{-3}* (1.60×10^{-2})*
Teenager (Elsewhere on Landfill)	5.33×10^{-1}	4.37×10^{-5} (1.82×10^{-3})*

The total estimated excess lifetime cancer risks associated with dermal contact and accidental ingestion for on-site and off-site surface and drum contents due to the presence of carcinogens are shown below:

SOIL RISKS

On-Site Soils		Off-Site Residential Areas
"Hot Spot"	Landfill Excluding "Hot Spot"	
1.48×10^{-3} (1.61×10^{-2})	5.41×10^{-6} (2.19×10^{-3})	9.65×10^{-5} (1.91×10^{-4})

The numbers outside the parentheses are the most plausible case; the numbers inside parentheses are the worst case.

The estimates represent the risks associated with exposure using more conservative estimates in the calculations. Most of the risk at the drum site is attributable to 2,3,7,8-TCDD, the contaminant of primary concern. The risk for a teenager near the drum site due to the presence of 2,3,7,8-TCDD alone was estimated to be 6.47×10^{-4} (Table 13). There are also important contributions to the risk from the drum contents and soils due to 2,4,6-trichlorophenol. The risk from the landfill excluding the drum samples (landfill excluding "hot spot") is due to the presence of 2,3,7,8-TCDD and arsenic. Though there were only a few samples on the landfill excluding the drum site with a concentration greater than 1 ηg/g (= 1 ppb) (maximum 21.0 ηg/g), the risk to a teenager on the landfill excluding the hot spot, due to 2,3,7,8-TCDD, was estimated to be 2.83×10^{-6}.

Most of the risk in off-site residential areas is attributable to arsenic, with minor additional risk posed by a single sample containing PAHs and a single sample containing 4,4'-DDT. Risk calculated for a 2-6 year old child off-site was 9.64×10^{-5} for

arsenic alone; total off-site risk was estimated at 9.65×10^{-5}. However, since the arsenic concentrations producing this risk are similar to background concentrations, the risk is primarily due to a naturally occurring inorganic substance. In off-site residential areas, analytical results indicate that 2,3,7,8-TCDD was not present in any samples above the analytical detection limits. These detection limits ranged from 0.006 to 0.08 ηg/g. Assuming that concentrations in the backyard were one-half the detection limit, the hypothetical risk calculated for a 2-6 year old child was 2.39×10^{-6} and 2.71×10^{-6} for an adult gardener.

Risks calculated for off-site residential areas appear to be greater than risks on the landfill. However, this is due to differences in the exposed populations. The on-site calculations were based on exposure for teenagers having a 49 kg body weight. Off-site exposures were based on exposure for a child having a body weight of 10 kg. This and other assumptions resulted in higher off-site dose rates and risks for the child.

Offsite risks were also calculated for older children. For a 6-12 year old grade schooler offsite, the risk due to 2,3,7,8-TCDD alone would be 3.78×10^{-7}; that is, 15.8% less than the risk quoted for 2-6 year old child. The reduction in risk primarily occurs because there is a reduction in the assumed ingestion rate and exposure duration, and an increase in body weight. Specifically, the assumption for these two age groups are (U.S. EPA, 1989a and U.S. EPA, 1989b):

	Age Group	
	2-6 year old child	6-12 year old grade schooler
Soil Ingestion Rate	0.8 g/day	0.1 g/day
Body Weight	10 kg	30 kg
Exposure Duration	1825 days	1440 days
Exposed Surface Area	1400 cm^2	2022 cm^2

The overall net effect of all of the above changes is a 3.3% reduction in the accidental ingestion dose rate and a 38% reduction in dermal exposure dose rate producing a net 15.8% decrease in the total dose rate and risk. Similar reduced risks would occur in a grade schooler for exposure to other detected compounds.

5.3 SUMMARY

Based on excess lifetime cancer risk calculations for contaminated soils, the drum site ("hot spot") presents a significant health hazard due to dioxins and furans and 2,3,7,8-TCDD in particular. The hot spot also presents a noncarcinogenic hazard due to the presence of 2,4,5-T; 2,4,5-TP and 2,4-dichlorophenol in the drums which is expected to be associated with chronic adverse health effects. Off-site cancer risks are elevated due to the presence of arsenic. The highest potential exposure is to small children aged 2-6. However, arsenic is a naturally occurring component of soils.

REFERENCES

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6.0 UNCERTAINTIES IN THE RISK ASSESSMENT

This section discusses a number of uncertainties inherent in this risk assessment that should be taken into consideration.

For instance, all exposures are calculated based on the assumption that chemical concentrations are constant with time. The assumption is accurate on a more short-term basis, but most chemical concentrations change as time passes due to natural processes. This will have potential implications over the duration of exposure. For example, concentrations of the chemicals of potential concern in the soil could be reduced by erosion and by the leaching of contaminants into the groundwater. In this case, the exposure estimates in the assessment would be an over estimation of the true exposure in the soil. Conversely, chemicals that leach from the soil into the groundwater could result in increased groundwater concentrations and exposure from ingestion. Chemicals in the groundwater may contaminate residential drinking water wells that currently appear to be relatively uncontaminated. The landfill is also subject to periodic flooding; and the contaminated soil may be subject to surficial movement. Natural degradation processes may also serve to reduce contaminant concentrations. Given these uncertainties, it is difficult to precisely predict future exposure concentrations and resultant risks.

Uncertainties regarding the toxicity information for humans, fauna and flora add additional degrees of uncertainty to the final estimates of risk. The toxicity information is mainly derived from animal studies that may, or may not, be an accurate reflection of the human response to a certain chemical. For instance, there are differences in absorption in the gastrointestinal tract, alveoli and respiratory tract, and skin between animals and humans that

would affect the dose of a chemical introduced in the body. There are also assumptions made regarding human behavior patterns that may or may not adequately describe human activity patterns at the site. For instance, some children may eat more soil than others; some teenagers may frequent the landfill more than others.

Assumptions are made regarding standard values used by the U.S. EPA (U.S. EPA, 1988b; Schaum, 1984; U.S. EPA, 1989; personal written communication from Dr. Pi-yun Tsai and Sarah Levinson, U.S. EPA, Region I, memorandum dated December 21, 1988). For instance, the U.S. EPA (1988b) adopted Calabrese (et al., 1987) values for typical soil ingestion rates for children by age group. These average values may, or may not, be accurate for the situation at hand. Values for average body weight and average lifetime used in the assessment are based on national averages; these values may or may not be representative of certain individuals or local populations. Dioxin exposure factors were used in this risk assessment to calculate the dose rate, mg/kg-day for furans, congeners and isomers of dioxin. This was done because exposure factors for the latter compounds are not available (U.S. EPA, 1988a). The dose rates and risks for congeners and isomers of dioxins and furans, therefore, may not be entirely accurate. Computation of the dose rate and risk using the congeners and isomers of dioxin and furans increased the risk only slightly. For PAHs, the q^* derived from animal studies using benzo(a)pyrene was used for all PAHs suspected of being carcinogenic; this will tend to overestimate the risk for PAHs.

There is no q^* in the IRIS (1989) currently available for lead, a probable human carcinogen, that is present in the site soils. This means there may be an underestimation of the total risk in the soils.

There is also measurement error in the exposure factors listed in the literature; that is, there is uncertainty that arises from random and systematic error in the measurement technique. There is also sampling error or uncertainty that arises from the actual population being sampled. Professional judgment must be exercised because data gaps must be filled based on engineering and scientific assumptions. Making professional judgments introduces some variability and subjectivity into the assessment.

Finally, there are limitations associated with the chemical database. There are constraints imposed by the chemical analytical procedures such as the lack of specificity of the data as related to contaminant detections at or near the detection limit and the inability to determine the specific valence state of some inorganic contaminants (e.g., for chromium). Chromium was quantitated as if all the chromium present was both Cr III and Cr VI; this introduces uncertainty in the assessment and must be taken into consideration when evaluating it.

Conservative assumptions were used in this analysis to compensate for these uncertainties so that the assessment would be less likely to underestimate the risks and hazards.

REFERENCES

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U.S. EPA. 1988b. Superfund Exposure Assessment Manual. Prepared by the U.S. Environmental Protection Agency, Office of Remedial Response, Washington, D.C. EPA-540/1-88/001, OSWER Directive 9285.1-1.

U.S. EPA. 1989. Exposure Factors Handbook, Final Report. Prepared by the U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, D.C. EPA-600/8-89/043.

7.0 CONCLUSIONS

The major health risks identified at the Jacksonville Landfill are based on the distribution and extent of chemical contamination, the potential for contaminant transport, opportunities for exposure, and the toxicity of the contaminants.

- A major potential health risk at the landfill is associated with the drum contents ("hot spot"). Potential routes of exposure to local populations include direct contact with contaminated surface soil on-site via dermal absorption and accidental ingestion of contaminated soil by people who frequent the landfill, such as teenagers. Chronic and carcinogenic health risks exist due to these potential exposure routes. The chemicals of concern include dioxins and furans, including 2,3,7,8-TCDD; 2,4,5-TP; and 2,4-dichlorophenol.

  There is less potential for excess lifetime carcinogenic health risks from contaminants distributed elsewhere on the landfill, and in off-site residential backyards. The carcinogens of concern on the landfill outside the drum area include dioxins and furans, including 2,3,7,8-TCDD; 4,4'-DDT, arsenic and carcinogenic PAH. In off-site residential backyards, the carcinogens of concern include arsenic; 4,4'-DDT and carcinogenic PAH. However, the major contributor of the off-site risk, arsenic, is due to naturally occurring concentrations presenting background risk to the population as a whole.

  Phthalates and methylene chloride found in soil samples cannot be entirely dismissed as common lab contaminants since concentrations exceeded the lab blank concentration

by greater than 10-fold. However, the additional risk they could contribute is extremely small.

Future transport of the chemicals contained in the drums is possible since the contents are exposed to the air. Dioxins adsorb to the soil and are transported by soil erosion or by surface water runoff of contaminated soils. In the past, people who might frequent the site such as teenagers had access to the drum area because the landfill was not always secure -- openings existed in the fence surrounding the landfill. The contents of the deteriorated drums that are visible on the ground surface represent the principal health hazard at the landfill.

- Ingestion and inhalation of contaminated groundwater at the monitored concentrations would be associated with noncarcinogenic and carcinogenic health risks if the water were consumed. The monitoring wells (JK-GW-03, 04, 05 and 12) contained 1,1-dichloroethene; 1,4-dichlorobenzene; benzene, arsenic and chromium. Many of the groundwater monitoring wells also had inorganics in excess of ARARs. However, residents currently drink municipal water, not residential well water, and any risks due to this route of exposure are only theoretical.

- Another possible mode of human exposure is via ingestion in the food chain; i.e., the consumption of aquatic biota taken from the unnamed pond adjacent to the landfill. Toxic effects to humans may occur if humans continuously ingest the surface water and/or eat the fish in the unnamed pond. ARARs for manganese and beryllium are

exceeded. Continuous exposure is, however, very unlikely.

- Ingestion of surface water from landfill trenches and drainage ditches near the landfill could present hazards to those who are exposed to them. This may occur when children or teenagers play around the landfill water sources. All monitored trench water samples exceeded ARARs for manganese. The effects observed in those who accidentally or willfully ingest the trench surface waters will depend on how much water is ingested and for how long. However, since manganese imparts an objectionable taste and color to water, frequent consumption, and hence a chronic health hazard, is extremely unlikely.

The risks presented herein are overestimates because conservative assumptions for the exposure duration and soil ingestion rates were used in the calculations. For instance, it was assumed teenagers would frequent the landfill daily for eight months per year during their teenage years, which is not very likely. However, overestimating the risk provides managers with the worst possible case as the basis for the remedial action decisionmaking process.

APPENDIX A

DATA EVALUATION

APPENDIX A
DATA EVALUATION

Data were summarized for this risk assessment by calculating the geometric and arithmetic means and using the maximum concentration ("worst case"). Geometric means were calculated since environmental media contamination is frequently log-normally distributed (Dean, 1981).

The geometric means were calculated using the following equation (Rosner, 1986):

$$\text{Geometric Mean} = \sqrt[n]{(x_1)\ (x_2)\ \ldots\ (x_n)}$$

$$= 10^{\frac{(\log x_1 + \log x_2 + \ldots + \log x_n)}{n}}$$

$$= e^{\frac{(\ln x_1 + \ln x_2 \ldots \ln x_n)}{n}}$$

The following criteria and guidelines were followed in evaluating chemical data:

- Sample concentration levels were compared to controls, including background, field, equipment, lab method and trip blanks. Professional judgment was used in the evaluation. Only concentrations of chemicals believed to be sufficiently above "control" samples were used in the analysis. If a common laboratory contaminant (acetone, methylene chloride, toluene, 2-butanone, or the phthalate esters) was detected in a sample, the level detected was used as the sample concentration only if the level exceeded the concentration in the field, equipment,

lab method or trip blank by a factor of ten. Otherwise the sample was considered not detectable for that compound. For organic and inorganic chemicals other than the common laboratory contaminants, the concentration in the sample was used only if it exceeded the concentration in the field, trip, equipment or lab method blank by a factor of five. Otherwise the sample was considered not detectable for that compound (USEPA, 1988).

- Concentrations reported for duplicate samples for a given sampling point were used in the analysis. Averages and geometric means were calculated for each chemical using the duplicates.

- Several sample levels were flagged with a J, indicating that these chemicals were detected but that the reported levels were estimated. These estimated results add an additional degree of uncertainty to the concentration levels (i.e., may overestimate or underestimate actual values); however, they have been taken at face value in this assessment. If one of these values significantly contributed to the estimated risk for a particular exposure scenario, the uncertainty was noted.

- To calculate the geometric mean for media in which a chemical was nondetectable, nondetects were included in the mean by using one-half of the sample analytical detection limit. This was especially important for dioxins and furans where many monitored concentrations were nondetectable.

- Some samples were flagged with an R, indicating that the data are unreliable because of quality control problems.

The R flag indicates uncertainty in both the identity of the compound and its measured concentration. R-flagged values were not used in the risk assessment.

- Some samples were flagged with an E, indicating the data exceeds the calibration range and that the sample is present, at least, for the minimum value given. The duplicate for the sample having the E was used in calculating geometric means or averages.

REFERENCES

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ROSNER, B. 1986. Fundamentals of Biostatistics. Duxbury Press, Boston, Massachusetts.

U.S. EPA, June 1988. Draft Final Endangerment Assessment Report, Old Springfield Landfill, Town of Springfield, Windsor County, VT, p. 2-3.

APPENDIX B

2,3,7,8-TCDD EQUIVALENTS IN THE RISK ASSESSMENT

APPENDIX B

2,3,7,8-TCDD EQUIVALENTS IN THE RISK ASSESSMENT

Data for congeners and isomers and furans were quantitated using the 2,3,7,8-TCDD toxicity equivalence factor (TEF, U.S. EPA, 1987); the international toxicity equivalence factor (I-TEF, U.S. EPA, 1989); and the proportionality factor for PCB fire soot (P.F., U.S. EPA, 1989). Both the EPA-TEF/87 and I-TEF/89 approaches were used to determine which produced the higher values. In most cases, the I-TEF/89 factors resulted in a higher 2,3,7,8-TCDD equivalent concentration and these higher values were then used in risk calculations presented throughout the main body of this report. The following list shows the TEFs used in this risk assessment:

CDFs/CDDs	EPA-TEF/87	I-TEF/89	Proportionality Factor (PF)
TCDFs	0.001		1
2,3,7,8-TCDF	0.1	0.1	0.03
Other		0	0.97
PeCDFs	0.001		1
1,2,3,7,8-PeCDF		0.05	0.035
2,3,4,7,8-PeCDF		0.5	0.035
Other		0	0.930
HxCDFs	0.0001		1
2,3,7,8-HxCDF	0.01	0.1	0.25
Other		0	0.75
HpCDFs	0.00001		1
2,3,7,8-HpCDF	0.001	0.01	0.5
Other		0	0.5
OCDF	0	0.001	1
TCDDs	0.01		1
2,3,7,8-TCDD	1	1	0.05
Other		0	0.95
PeCDDs	0.005		1
2,3,7,8-PeCDD	0.5	0.5	0.07
Other		0	0.93
HxCDDs	0.0004		1
2,3,7,8-HxCDD	0.04	0.1	0.3
Other		0	0.7
HpCDDs	0.00001		1
2,3,7,8-HpCDD	0.001	0.01	0.5
Other		0	0.5
OCDD	0	0.001	1

TCDD Equivalency calculations used the following process:

1. Analytical determination of the CDDs and CDFs in the environmental sample.

2. For EPA-TEF/87, multiplication of congener concentration in the environmental sample by the equivalency factor from the accompanying list to express the concentration in terms of 2,3,7,8-TCDD equivalents.

3. For I-TEF/89, multiplication of congener concentration in the environmental sample by the proportionality factor (PF) to obtain the proportional concentration; then multiplication of the proportional concentration by the I-TEF/89 equivalency factor to obtain the 2,3,7,8-TCDD equivalents. For 2,3,7,8-TCDF and 2,3,7,8-TCDD, corrections were not made using the PF because the actual total and 2,3,7,8-TCDD/TCDF concentrations were known.

4. Summation of the products in Step 2 or 3 to obtain the <u>total</u> 2,3,7,8-TCDD equivalents in the sample.

5. Summation of the human exposure of the mixture samples in question, expressed in terms of 2,3,7,8-TCDD equivalents.

6. Combination of exposure from Step 5 with toxicity information on 2,3,7,8-TCDD (usually carcinogenicity and/or reproductive effects) to estimate risks associated with the mixture.

A sample calculation using actual data from this study and the EPA-TEF/87 follows.

Compound	Measured Concentration (ng/g)	EPA-TEF/87	2,3,7,8-TCDD Equivalents
TCDFs	180.0	0.001	1.80×10^{-1}
PeCDFs	130.00	0.001	1.30×10^{-1}
HxCDFs	0.85*	0.0001	8.50×10^{-5}
HpCDFs	0.44*	0.00001	4.35×10^{-6}
OCDF	0.85*	0	0
2,3,7,8-TCDF	31.00	0.1	3.10×10^{0}
TCDDs	210.00	0.01	2.10×10^{0}
PeCDDs	2.40*	0.005	1.18×10^{-2}
HxCDDs	1.00*	0.0004	4.00×10^{-4}
HpCDDs	0.50*	0.00001	5.00×10^{-6}
OCDD	1.10*	0	0
2,3,7,8-TCDD	190.00	1.0	1.90×10^{2}
			Σ 2,3,7,8-TCDD equivalents: 195.52

NOTE: Wherever the measured concentration is nondetectable, one-half the detection limits were used to compute the 2,3,7,8-TCDD equivalents.

* This value was arrived at by using one-half the detection limit.

Using EPA-TEF/87 TCDD equivalents (i.e., 195.52 ng/g) rather than the concentration of 2,3,7,8-TCDD (i.e., 190 ng/g) increases the estimated concentration slightly. Thus, the risk increases. Compare risks for 2,3,7,8-TCDD in Table 12 to risks for total dioxins and furans. Using I-TEF/89 for the same sample, the 2,3,7,8-TCDD equivalent is 196 ng/g. See the example that follows:

Chemical Name	I-TEF/89	PF	Measured Concentration (ng/g)	Proportional Concentration	2,3,7,8-TCDD Equivalents (Equivalent Concentration)
Total TCDFs			180		
2,3,7,8-TCDF	0.1		31	31.000	3.10E+00
Other	0		149	149.000	0.00E+00
Total PeCDFs			130		
1,2,3,7,8-PeCDF	0.05	0.035		4.550	2.28E-01
2,3,4,7,8-PeCDF	0.5	0.035		4.550	2.28E+00
Other	0	0.93		120.900	0.00E+00
HxCDFs			0.85*		
2,3,7,8-HpCDF	0.1	0.25		0.213	2.13E-02
Other	0	0.75		0.638	0.00E+00
HpCDFs			0.435*		
2,3,7,8-HpCDF	0.01	0.5		0.218	2.17E-03
Other	0	0.5		0.218	0.00E+00
OCDF	0.001	1	0.85*	0.850	8.50E-04
TCDDs			210		
2,3,7,8-TCDD	1		190	190.000	1.90E+02
Other	0		20	20.000	0.00E+00
PeCDDs			2.35*		
2,3,7,8-PeCDD	0.5	0.07		0.165	8.23E-02
Other	0	0.93		2.186	0.00E+00
HxCDDs			1*		
2,3,7,8-HxCDD	0.1	0.3		0.300	3.00E-02
Other	0	0.7		0.700	0.00E+00
HpCDDs			0.5*		
2,3,7,8-HpCDD	0.01	0.5		0.250	2.50E-03
Other	0	0.5		0.250	0.00E+00
OCDD	0.001	1	1.1*	1.100	1.10E-03
				Σ 2,3,7,8-TCDD equivalents:	196

NOTE: The measured concentration is nondetectable, one-half the detection limits were used to compute the 2,3,7,8-TCDD equivalents.

* This value was arrived at by using one-half the detection limit.

Using the recently revised approach to calculate 2,3,7,8-TCDD equivalents and the International Toxicity Equivalency Factor (I-TEF, U.S. EPA, 1989), generally speaking, the TEF increases, or

remains constant for the 2,3,7,8 congeners and is reduced to zero for others. For the Jacksonville calculations, the 2,3,7,8-TCDD equivalents increased proportionally, but the risk changed only slightly because I-TEFs/89 are still 1 to 3 orders of magnitude below the 2,3,7,8-TCDD TEF; and for this site, 2,3,7,8-TCDD is the dominant concentration (i.e., of the 196 ng/g equivalent concentration shown previously, 190 ng/g is due to 2,3,7,8-TCDD itself).

REFERENCE

U.S. EPA. 1987. Interim Procedures for Estimating Risks Associated With Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and Dibenzofurans (CCDs and CDFs), U.S. EPA. Risk Assessment Forum, EPA/625/3-87/012.

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APPENDIX C
ESTIMATION OF DOSE RATE, mg/kg-day

APPENDIX C

ESTIMATION OF EXPOSURE POINT CONCENTRATIONS FOR JACKSONVILLE LANDFILL

C.1. Direct Contact with Soil by Children

Dose rate estimates are made in this report for children, teenagers and adults. Children are included in the calculations because they are at increased risk from cancer because they have a smaller body weight and eat more dirt than adults and teenagers. This gives them a higher dose rate and risk than for some other members of the population. Estimating risks from dioxin for children would be important because dioxin is very toxic and children are at increased risk compared to adults and teenagers because children ingest more soil than adults.

The equations and scenarios presented herein are specifically outlined for dioxin. However, where appropriate and where data exists, they were used for other chemicals, making minor changes in the equations.

Direct contact with contaminated soil could occur when children play in contaminated soil in their backyard. Conservative estimates for the dose rates are used in the calculations. That is, if there is a range for an exposure factor in a numerator, then the larger number is put in the equation to compute the dose rate. If the exposure factor is in the denominator, then the smaller factor is used.

Table C-1 presents assumptions used to assess these potential exposures. Direct contact is considered for the average exposure (most probable) and plausible maximum exposure (realistic worst

case). Calculations for average and plausible maximum exposure cases were used in the analysis so decisionmakers could evaluate the full range of risk in the analysis and make careful judgments in selecting remediation. The use of two exposure scenarios is especially important for soil contaminants that vary widely in concentration.

Only one set of exposure assumptions for both the average exposure and plausible maximum exposure were used. Therefore, the only differences between the two cases is due to the concentration term.

Based on the ages of children currently living near the landfill, the exposure period is for children between 2 and 6 years old, and the exposure duration is 1,825 days (5 years). Because of the rather temperate climate in Arkansas, it is assumed children can play outdoors every day of the year. During the year 1987, there were only about 60 days when the ambient temperature was at, or less than, 32°F (National Climatic Data Center, Ashville, NC, 704-259-0871, personal communication from Mr. Al Chen and Annual 1987 Summary).

Average and plausible maximum incidental soil ingestion rates for a child 2-6 years old used are 0.1 g/day to 0.2 g/day and 0.8 g/day, respectively (U.S. EPA, 1989a). Recent guidance recommended 0.2 g/day for a child of 1-6 years (U.S. EPA, 1989b). 0.8 g/day for a child is used in this assessment to be conservative. The dioxin adsorbs strongly to the soil and is thus less available to be absorbed through the gastrointestinal tract (20-26% of dioxin is absorbed; Poiger & Schlatter, 1980, as cited in Schaum, 1984).

Between 0.07% to 3% (Schaum, 1984) of the dioxin is assumed to be absorbed dermally. This value was extrapolated from animals

to humans and introduces uncertainty in the risk assessment due to differences between humans and animals in skin properties. Using 3% is a conservative estimate in the risk assessment.

Dioxin is absorbed through the skin, through inhalation of vapors and ingestion of contaminated soils, water and organisms. (Sittig, M., 1980). For purposes of these calculations and from the monitored environmental data available, dose rates (mg/kg-day) for dioxin are calculated using the ingestion and dermal absorption equations for dioxin-contaminated soil.

Using the assumptions previously described, a dose rate (mg/kg-day) can be calculated for oral and dermal exposure from dioxin-contaminated soil in the 2-6 year old child who plays in a residential backyard near the landfill (see the following calculations).

Risks are calculated for contaminated soil by calculating actual dose rates of a particular chemical using various routes of chemical exposure. The equations used to calculate the actual dose rates are of the following general form:

$$\text{Dose Rate} = \frac{\text{Exposure} \times \text{Frequency of Contact}}{\text{Body Mass}}$$

$$= \frac{\text{mg}}{\text{kg-day}}$$

There are various forms of this general equation in the following pages; these equations estimate the dose rate of a chemical using various exposure routes, such as dermal and oral ingestion (U.S. EPA, 1988a).

TABLE C-1

ASSUMPTIONS FOR USE IN THE RISK ASSESSMENT FOR DIRECT CONTACT WITH SOIL (ACCIDENTAL INGESTION AND DERMAL EXPOSURE) BY CHILDREN (2-6 YEARS OLD) AT JACKSONVILLE LANDFILL

Parameter	Average Exposure	Plausible Maximum Exposure
Concentration of chemical in soil or Waste	Average or geometric mean: ηg/g	Maximum: ηg/g
Amount of soil ingested/ day for 2-6 year old child	0.8 g/day	0.8 g/day
Exposure duration	1,825 days	1,825 days
Absorption of dioxin from gastrointestinal tract	0.26	0.26
Weight of a 2-6 year old child	10 Kg	10 Kg
An expected lifetime	70 years	70 years
Number of days/year	365 days	365 days
Amount of soil in contact with the skin	2.77 mg/cm^2 - day	2.77 mg/cm^2 - day
Skin surface area exposed	1,400 cm^2	1,400 cm^2
Absorption of dioxin through the skin	0.03	0.03

EXAMPLE OF DOSE RATE CALCULATIONS

Accidental Ingestion of Contaminated Surface Soil or Wastes by a child aged 2-6 years.

Assumptions:

- Concentration of chemical in soil or waste ($\eta g/g$)
- 0.1 g/day to 0.2 g/day is the average soil ingestion in the population of young children, under age 7 (Binder, *et al*, 1986) and Clausing, *et al* (1987), as cited in U.S. EPA (1989a). 0.8 g/day of soil is the upper range ingestion estimate among children with a higher tendency to ingest soil materials.
- An exposure duration ranges from 1,235 to 1,825 days (Schaum, 1984).
- Absorption of the compound through the gastrointestinal tract is 0.2 to 0.26 (Poiger & Schlatter, 1980, as cited in Schaum, 1984).
- The weight of a child (2-6 years old) is 10 or 24 kg (Schaum, 1984).
- An expected lifetime is 70 years.
- Chemical is a known or suspected carcinogen for less-than-lifetime exposure and is associated with lifetime risk.

Average (Most Probable) Exposure:

Dose Rate (mg/kg-day) =

$$\frac{\eta g}{g} \times \frac{0.8 g}{day} \times \frac{mg}{10^3 \mu g} \times \frac{1 \mu g}{10^3 \eta g} \times 0.26 \times \frac{1}{10 kg} \times \frac{1825 \text{ days}}{70 \text{ years}} \times \frac{1}{\frac{365 \text{ days}}{\text{year}}}$$

$$= \eta g/g \times 1.49 \times 10^{-9}$$

Maximum Exposure:

Dose Rate (mg/kg-day) =

$$\frac{\eta g}{g} \times \frac{0.8 g}{day} \times \frac{mg}{10^3 \mu g} \times \frac{1 \mu g}{10^3 \eta g} \times 0.26 \times \frac{1}{10 kg} \times \frac{1825 \text{ days}}{70 \text{ years}} \times \frac{1}{\frac{365 \text{ days}}{\text{year}}}$$

$$= \eta g/g \times 1.49 \times 10^{-9}$$

Dermal Exposure to Contaminated Surface Soil or Wastes by a Child Aged 2-6 Years.

Assumptions:

- Concentration of chemical in soil (ηg/g).
- Amount of soil in contact with skin (2.77 mg/cm^2-day for clay soil; U.S. EPA, 1988b).
- Skin surface area exposed (980 - 1400 cm^2) (Schaum, 1984).
- Absorption of the dioxin compound through the skin is 0.07 - 3% (Schaum, 1984).
- The weight of a child (2-6 years old) is 10 or 24 kg (Schaum, 1984).
- An expected lifetime is 70 years.
- Total duration of exposure is 1,235 days to 1,825 days (5 years).
- Chemical is a known or suspected carcinogen for which less-than-lifetime exposure is associated with lifetime risk.

Average (Most Probable) Exposure:

Dose Rate (mg/kg - day) =

$$\frac{\eta g}{g} \times \frac{2.77\ mg}{cm^2\text{-day}} \times \frac{1400\ cm^2}{1} \times \frac{g}{10^9\ \eta g} \times \frac{1}{10\ kg} \times 0.03 \times \frac{1825\ days}{70\ years} \times \frac{1}{\frac{365\ days}{year}}$$

$$= \eta g/g \times 8.31 \times 10^{-10}$$

Maximum Exposure:

Dose Rate (mg/kg - day) =

$$\frac{\eta g}{g} \times \frac{2.77\ mg}{cm^2\text{-day}} \times \frac{1400\ cm^2}{1} \times \frac{g}{10^9\ \eta g} \times \frac{1}{10\ kg} \times 0.03 \times \frac{1825\ days}{70\ years} \times \frac{1}{\frac{365\ days}{year}}$$

$$= \eta g/g \times 8.31 \times 10^{-10}$$

The total dose rate for dioxin from dermal and oral ingestion from the soil is the sum of the individual dose rates derived from oral and dermal absorption. For this assessment, the total dose rate of the dioxin is approximated by the dioxin absorbed through the skin and ingested orally. There is an inhalation route of exposure for dioxin that is not addressed in this report because no dioxins or furans were detected in air samples from previous studies (memorandum from Gene A. McDonald, FIT, Ecology and Environment, Region VI, dated September 10, 1985).

C.2 Direct Contact with Soil by a Teenager

Direct contact with dioxin-contaminated soil could occur in teenagers (aged 13-19 years) from outdoor activities, such as riding bikes through the landfill. Bike-riding trespassers have been observed on the landfill (personal communication, members of the RI field team). Table C-3 presents the assumptions used in calculating the dose rate (mg/kg - day) for these routes of exposure by the teenager.

The teenager differs from the adult used in the previous scenario by the exposure duration. The exposure duration for the teenager is 2,555 days. Here, it is assumed that the teenager can be on the landfill riding his/her bike every day during a seven-year period. The body weight for a teenager, aged 13-17 years, is between 49-70 kg (Schaum, 1984). Other factors for dioxin bioavailability previously used for adults were used in these equations for the teenager.

TABLE C-3

ASSUMPTIONS FOR USE IN RISK ASSESSMENT FOR DIRECT CONTACT WITH SOIL (ACCIDENTAL INGESTION AND DERMAL EXPOSURE) BY TEENAGERS (13-19 YEARS OLD) AT JACKSONVILLE LANDFILL

Parameter	Average Exposure	Plausible Maximum Exposure
Concentration of chemical in soil or waste	Average or geometric mean: ng/g	Maximum: ng/g
Amount of soil ingested/ day for 13-19 year old	0.05 g/day	0.05 g/day
Exposure duration	2,555 days	2,555 days
Absorption of dioxin from gastrointestinal tract	0.26	0.26
Weight of a 13-19 year old teenager	49 kg	49 kg
An expected lifetime	70 years	70 years
Number of days/year	365 days	365 days
Amount of soil in contact with the skin	2.77 mg/cm^2 - day	2.77 mg/cm^2 - day
Skin surface area exposed	2,940 cm^2	2,940 cm^2
Absorption of dioxin through the skin	0.03	0.03

Dose rates (mg/kg - day) are calculated and included on the following pages.

Accidental Ingestion of Contaminated Surface Soil or Wastes by a teenager aged 13-19 years who rides his/her bike through the landfill.

Assumptions:

- Concentration of chemical in soil or waste (ηg/g).
- 0.05 g/day of soil are ingested by older children and adults (personal written communication from U.S. EPA, Region I, Pi-yun Tsai, Ph.D., and Sarah Levinson, 12/21/88).
- An exposure duration ranges from 1,729 days - 2,555 days.
- Absorption of the compound through the gastrointestinal tract is 0.2 to 0.26 (Poiger and Schlatter, 1980, as cited in Schaum, 1984).
- The weight of a teenager (13-19 years) is 49 to 70 kg (Schaum, 1984).
- An expected lifetime is 70 years.
- Chemical is a known or suspected carcinogen for which less-than-lifetime exposure and is associated with lifetime risk.

Average (Most Probable) Exposure:

Dose Rate (mg/kg - day) =

$$= \frac{\eta g}{g} \times \frac{0.05\ g}{day} \times \frac{mg}{10^3\ \mu g} \times \frac{\mu g}{10^3\ \eta g} \times 0.26 \times \frac{1}{49\ kg} \times \frac{2555\ days}{70\ years} \times \frac{1}{\frac{365\ days}{year}}$$

$$= \eta g/g \times 2.65 \times 10^{-11}$$

Maximum Exposure:

Dose Rate (mg/kg - day) =

$$= \frac{\eta g}{g} \times \frac{0.05\ g}{day} \times \frac{mg}{10^3\ \mu g} \times \frac{\mu g}{10^3\ \eta g} \times 0.26 \times \frac{1}{49\ kg} \times \frac{2555\ days}{70\ years} \times \frac{1}{\frac{365\ days}{year}}$$

$$= \eta g/g \times 2.65 \times 10^{-11}$$

Dermal Exposure to Contaminated Surface Soil or Wastes:

Assumptions:

- Concentration of chemical in soil or waste (ηg/g).
- Amount of soil in contact with skin (2.77 mg/cm^2 - day for clay soil; U.S. EPA, 1988b).
- Skin surface area exposed (2,940 cm^2) (Schaum, 1984).
- Absorption of the compound through the skin is 0.07% - 3% (Schaum, 1984).
- The weight of a teenager (aged 13-19 years) is 49 to 70 kg.
- An expected lifetime is 70 years.
- Total duration of exposure is 1,729 days - 2,555 days.
- Chemical is a known or suspected carcinogen for which less-than-lifetime exposure is associated with lifetime risk.

Average (Most Probable) Exposure:

Dose Rate (mg/kg - day) =

$$= \frac{\eta g}{g} \times \frac{2.77\ mg}{cm^2\text{-day}} \times \frac{2940\ cm^2}{1} \times \frac{g}{10^9\ \eta g} \times \frac{1}{49\ kg} \times 0.03 \times \frac{2555\ days}{70\ years} \times \frac{1}{\frac{365\ days}{year}}$$

$$= \eta g/g \times 4.99 \times 10^{-10}$$

Maximum Exposure:

Dose Rate (mg/kg - day) =

$$= \frac{\eta g}{g} \times \frac{2.77\ mg}{cm^2\text{-day}} \times \frac{2940\ cm^2}{1} \times \frac{g}{10^9\ \eta g} \times \frac{1}{49\ kg} \times 0.03 \times \frac{2555\ days}{70\ years} \times \frac{1}{\frac{365\ days}{year}}$$

$$= \eta g/g \times 4.99 \times 10^{-10}$$

C.3 Direct Contact with Soil by an Adult

Direct contact with dioxin-contaminated soil could occur in adults from outdoor activities such as gardening. Adults may be exposed to dioxin while gardening from dermal absorption and accidentally ingesting contaminated soil by inadvertently eating before washing their hands. Table C-2 presents the assumptions used in calculating the dose rate (mg/kg-day) for these routes of exposure by the adult.

TABLE C-2

ASSUMPTIONS FOR USE IN THE RISK ASSESSMENT FOR DIRECT CONTACT WITH SOIL (ACCIDENTAL INGESTION AND DERMAL EXPOSURE) BY ADULTS (20-70 YEARS) AT JACKSONVILLE LANDFILL

Parameter	Average Exposure	Plausible Maximum Exposure
Concentration of chemical in soil or waste	Average or geometric mean: ηg/g	Maximum: ηg/g
Amount of soil ingested for adult aged 20-70 years	0.05 g/day	0.05 g/day
Exposure duration	18,250 days	18,250 days
Absorption of dioxin from gastrointestinal tract	0.26	0.26
Weight of an adult aged 20-70 Years	70 kg	70 kg
An expected lifetime	70 years	70 years
Number of days/year	365 days	365 days
Amount of soil in contact with the skin	2.77 mg/cm^2 - day	2.77 mg/cm^2 - day
Skin surface area exposed	2940 cm^2	2940 cm^2
Absorption of dioxin through the skin	0.03	0.03

Average (most probable) and plausible maximum cases were developed using geometric mean or average concentrations of dioxin in residential backyards near the landfill.

Exposure occurs throughout life, from age 20 to 70 years, for an exposure duration of 12,350 - 18,250 days. The climate in Arkansas is temperate and it was assumed, at the maximum, adults could be outdoors every day of the year. The amount of soil ingested/day was assumed to be 0.05 g/day (personal written communication, EPA, Region I, Pi-yun Tsai, Ph.D., and Sarah Levinson, dated 12/21/88). However, soil ingestion rates for adults have not been experimentally determined as they have been for children. Recent guidance recommended 0.1 g/day for an adult (U.S. EPA, 1989b). Soil ingestion rates used in the analyses were tailored to site conditions and scenarios. The exposed skin surface area has been estimated to be 2940 cm^2 for an adult wearing short-sleeved shirts, open-necked shirts, pants, shoes and no gloves or hats (Poiger and Schlatter, 1980, as cited in Schaum, 1984). Other factors for dioxin bioavailability previously cited for children were used for adult exposure.

Dose rates (mg/kg - day) are calculated and included on the following pages.

Accidental Ingestion of Contaminated Surface Soil or Wastes by an Adult Aged 20 - 70 years who gardens in his/her backyard next to the landfill.

Assumptions:

- Concentration of chemical in soil or waste (ηg/g).
- 0.05 g of soil are ingested per day (g/day) (personal written communication, EPA, Region I, Pi-yun Tsai, Ph.D., and Sarah Levinson, 12/21/88).
- An exposure duration ranges from 12,350 days - 18,250 days.
- Absorption of the compound through the gastrointestinal tract is 0.2 to 0.26 (Poiger and Schlatter, 1980, as cited in Schaum, 1984).
- The weight of an adult, aged 20-70 years, = 70 kg (Schaum, 1984).
- An expected lifetime is 70 years.
- Chemical is a known or suspected carcinogen for which less-than-lifetime exposure is associated with lifetime risk.

Average (Most Probable) Exposure:

Dose Rate (mg/kg - day) =

$$\frac{ng}{g} \times \frac{0.05\ g}{day} \times \frac{mg}{10^3\ \mu g} \times \frac{1\ \mu g}{10^3\ ng} \times 0.26 \times \frac{1}{70\ kg} \times \frac{18{,}250\ days}{70\ years} \times \frac{1}{\frac{365\ days}{year}}$$

$= ng/g \times 1.33 \times 10^{-10}$

Maximum Exposure:

Dose Rate (mg/kg-day) =

$$\frac{ng}{g} \times \frac{0.05\ g}{day} \times \frac{mg}{10^3\ \mu g} \times \frac{1\ \mu g}{10^3\ ng} \times 0.26 \times \frac{1}{70\ kg} \times \frac{18{,}250\ days}{70\ years} \times \frac{1}{\frac{365\ days}{year}}$$

$= ng/g \times 1.33 \times 10^{-10}$

Dermal Exposure to Contaminated Surface Soil or Wastes:

Assumptions:

- Concentration of chemical in soil or waste (ηg/g).
- Amount of soil in contact with skin (2.77 mg/cm^2 - day for clay soil; U.S. EPA, 1988b).
- Skin surface area exposed (2,940 cm^2) (Schaum, 1984).
- Absorption of the compound through the skin is 0.07% to 3% (Schaum, 1984).
- The weight of an adult aged 20-70 years is 70 kg.
- An expected lifetime is 70 kg.
- Total duration of exposure is 12,350 days to 18,250 days (50 years).
- Chemical is a known or suspected carcinogen for which less-than-lifetime exposure is associated with lifetime risk.

Maximum (Most Probable) Exposure:

Dose Rate (mg/kg - day) =

$$\frac{\eta g}{g} \times \frac{2.77\ mg}{cm^2\text{-day}} \times \frac{2940\ cm^2}{1} \times \frac{g}{10^9\ \eta g} \times \frac{1}{70\ kg} \times \frac{0.03}{1} \times \frac{18{,}250\ days}{70\ years} \times \frac{1}{\frac{365\ days}{year}}$$

$$= \eta g/g \times 2.49 \times 10^{-9}$$

Maximum Exposure:

Dose Rate (mg/kg - day) =

$$\frac{\eta g}{g} \times \frac{2.77\ mg}{cm^2\text{-day}} \times \frac{2940\ cm^2}{1} \times \frac{g}{10^9\ \eta g} \times \frac{1}{70\ kg} \times \frac{0.03}{1} \times \frac{18{,}250\ days}{70\ years} \times \frac{1}{\frac{365\ da}{year}}$$

$$= \eta g/g \times 2.49 \times 10^{-9}$$

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APPENDIX D

ACRONYMS AND ABBREVIATIONS

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ACRONYMS AND ABBREVIATIONS

ACGIH	American Conference of Governmental Industrial Hygienists
ADI	Acceptable Daily Intake
ARAR	Applicable or Relevant and Appropriate Requirement
AWQC	Ambient Water Quality Criteria (also called, Water Quality Criteria)
bw	body weight
CAG	Carcinogen Assessment Group, U.S. EPA
CAS	Chemical Abstracts Service
cc	cubic centimeters
CDC	Centers for Disease Control
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act of 1980
CFR	Code of Federal Regulations
CNS	central nervous system
CRAVE	Carcinogen Risk Assessment Verification Endeavor
cu.m, M^3	cubic meter
CWA	Clean Water Act
DHEW	U.S. Department of Health, Education, and Welfare (now U.S. Department of Health and Human Services)
DNA	deoxyribonucleic acid
DOT	U.S. Department of Transportation
DW	drinking water
E	exponent (e.g., 1.5E-6 = 1.5 x 10 to the power of -6 = 0.0000015)
EEG	electroencephalogram
EKG	electrocardiogram
EPA	U.S. Environmental Protection Agency
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FOI	Freedom of Information
FR	Federal Register

FWS	U.S. Fish and Wildlife Service
g	grams
GI	gastrointestinal
HA	Health Advisory
HCT	hematocrit
HEEP	Health and Environmental Effects Profile
Hgb	Hemoglobin
HHS	U.S. Department of Health and Human Services
HI	Hazard Index
HSDB	Hazardous Substance Data Base
IARC	International Agency for Research on Cancer
ICR	Institute of Cancer Research
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
ITII	International Technical Information Institute
kg	kilogram
L	liter
LC50	Lethal Concentration 50; concentration lethal to 50% of the animals
LD50	Lethal Dose 50; dose lethal to 50% of the animals
LEL	lower explosive limit
LOAEL	Lowest-Observed-Adverse-Effect Level
m	meter
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
MED	minimum effective dose
MF	Modifying Factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
mm Hg	millimeters of mercury; a measure of pressure

MOS	Margin of Safety
MTD	maximum tolerated dose
NAAQS	National Ambient Air Quality Standards
NAS	National Academy of Sciences
NESHAP	National Emission Standards for Hazardous Air Pollutants
ng	nanogram
NIH	National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
NLM	National Library of Medicine
NOAEL	No-Observed-Adverse-Effect Level
NOEL	No-Observed-Effect Level
NRC	National Research Council
NTIS	National Technical Information Service
NTP	National Toxicology Program
OAQPS	Office of Air Quality Planning and Standards, U.S. EPA
ODW	Office of Drinking Water, U.S. EPA
OHEA	Office of Health and Environmental Assessment, U.S. EPA
OHM/TADS	Oil and Hazardous Materials Technical Assistance Data Systems
OPP	Office of Pesticide Programs, U.S. EPA
OPTS	Office of Pesticides and Toxic Substances, U.S. EPA
ORD	Office of Research and Development, U.S. EPA
OSHA	U.S. Occupational Safety and Health Administration
OSWER	Office of Solid Waste and Emergency Response, U.S. EPA
OTS	Office of Toxic Substances, U.S. EPA
OWRS	Office of Water Regulations and Standards, U.S. EPA
P	probit dose extrapolation model
PEL	Permissible Exposure Limit
PHS	U.S. Public Health Service
p.o.	per os (by mouth)
ppb	parts per billion
ppm	parts per million
ppt	parts per trillion

q*	cancer potency factor (also called unit cancer risk or "slope factor")
RA	Risk Assessment
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RfD	Reference Dose
RfDi	Inhalation Reference Dose
RfDo	Oral Reference Dose
RM	Risk Management
RTECS	Registry of Toxic Effects of Chemical Substances
SAB	Science Advisory Board
SAP	serum alkaline phosphatase
SARA	Superfund Amendments and Reauthorization Act of 1986
sc	subcutaneous
SDWA	Safe Drinking Water Act
SF	Safety Factor
SGOT	serum glutamic-oxaloacetic transaminase
SGPT	serum glutamic-pyruvic transaminase
STEL	short-term exposure limit
TDB	Toxicology Data Base (renamed "Hazardous Substance Data Base")
TLV	Threshold Limit Value
TSCA	Toxic Substances Control Act
TWA	time-weighted average
UCL	upper confidence limit
UCR	unit cancer risk (also called q* or "slope factor")
UEL	upper explosive limit
UF	Uncertainty Factor
ug	microgram
ug/cu.m, ug/m^3	micrograms per cubic meter
ug/L	micrograms per liter
umol	micromoles

VOC	volatile organic compound
v/v	volume for volume
WBC	white blood cell(s)
WQC	Water Quality Criteria

APPENDIX E

GLOSSARY

APPENDIX E

GLOSSARY OF, RISK ASSESSMENT-RELATED TERMS

Acceptable Daily Intake -- An estimate of the daily exposure dose that is likely to be without deleterious effect, even if continued exposure occurs over a lifetime.

Acute exposure -- One dose or multiple doses occurring within a short time (24 hours or less).

Acute hazard or toxicity -- see Health Hazard.

Arithmetic Mean -- The sum of all the measurements in a data set divided by the number of measurements in the data set.

Attributable Risk -- The difference between risk of exhibiting a certain adverse effect in the presence of a toxic substance and that risk in the absence of the substance.

Average - see "Arithmetic Mean"

Benign -- Not malignant; remaining localized.

Bioassay -- The determination of the potency or concentration of a test substance by noting its effects in live animals or in isolated organ preparations, as compared with the effect of a standard preparation.

Bioavailability -- The degree to which a drug or other substance becomes available to the target tissue after administration or exposure.

Carcinogen -- An agent capable of inducing a carcinogenic response.

Carcinogenesis -- The origin or production of cancer, very likely a series of steps. The carcinogenic event so modifies the genome and/or other molecular control mechanisms in the target cells that these can give rise to a population of altered cells.

Case-control study -- An epidemiologic study that looks back in time at the exposure history of individuals who have the health effect (cases) and at a group who do not (controls), to ascertain whether they differ in proportion exposed to the chemical under investigation.

Chronic effect -- An effect that is manifest after some time has elapsed from initial exposure. See also Health Hazard.

Chronic exposure -- Multiple exposures occurring over an extended period of time, or a significant fraction of the animal's or the individual's lifetime.

Chronic hazard or toxicity -- see Health Hazard.

Chronic study -- A toxicity study designed to measure the (toxic),effects of chronic exposure to a chemical.

Cohort study -- An epidemiologic study that observes subjects in differently exposed groups and compares the incidence of symptoms. Although ordinarily prospective in nature, such a study is sometimes carried out retrospectively, using historical data.

Control group -- A group of subjects observed in the absence of agent exposure or, in the instance of a case/control study, in the absence of an adverse response.

Developmental toxicity -- The study of adverse effects on the developing organism (including death, structural abnormality, altered growth, or functional deficiency) resulting from exposure prior to conception (in either parent), during prenatal development, or postnatally up to the time of sexual maturation.

Dose -- The amount of a substance available for interaction with metabolic processes of an organism following exposure and absorption into the organism. It may appear in scientific literature as mg/kg. The amount of a substance crossing the exchange boundaries of skin, lungs, or digestive tract is termed **absorbed dose**, while the amount available for interaction by any particular organ or cell is termed the **delivered dose** for that organ or cell. Theoretically, the sum of the delivered doses plus the metabolic transformations should equal absorbed dose. (The terms **administered dose** and **applied dose** refer to amounts of a substance made available for absorption, and therefore are measures of exposure rather than dose. As such, these terms, sometimes found in the literature, are somewhat confusing and should be avoided if possible by exposure assessors).

Dose-response assessment -- A quantitative relationship is derived between the dose, or more generally the human exposure, and the probability of induction of a carcinogenic effect.

Dose-response relationship -- A relationship between the amount of an agent (either administered, absorbed, or believed to be effective) and changes in certain aspects of the biological system (usually toxic effects), apparently in response to that

agent.

Endpoint -- A response measure in a toxicity study.

Excess lifetime risk -- The additional or extra risk incurred over the lifetime of an individual by exposure to a toxic substance.

Exposure -- Contact of an organism with a chemical, physical, or biological agent. Exposure is quantified as the amount of the agent available at the exchange boundaries of the organism (e.g., skin, lungs, digestive tract) and available for absorption.

Exposure assessment -- An evaluation is made of the human exposure to the agent. Exposure assessments identify the exposed population, describe its composition and size, and present the type, magnitude, frequency, and duration of exposure.

Exposure Pathway -- The course a chemical or pollutant takes from the source to the organism exposed.

Exposure Scenario -- A set of assumptions about how exposure takes place (including assumptions/conditions concerning sources, exposure pathways, concentrations of pollutants, individual or population habits and characteristics), which aid the exposure assessor in evaluating, estimating, or quantifying exposures.

Extra or Excess risk -- The added risk to that portion of the population that is not included in measurement of background tumor rate; ER(d) = [P(d) - P(0)]/[1-P(0)].

Extrapolation -- An estimation of a numerical value of an empirical (measured) function at a point outside the range of data which were used to calibrate the function. The quantitative risk estimates for carcinogens are generally low-dose extrapolations based on observations made at higher doses. Generally one has a measured dose and measured effect.

Gamma multi-hit model -- A dose-response model of the form

$$P(d) = \text{Integral from 0 to d of } ([a^{**}k][s^{**}(k-1)][\exp(-as)]/G(u))ds$$

where: $G(u) = \text{integral from 0 to infinity of } [s^{**}(u-1)][\exp(-s)]ds$

$P(d)$ = the probability of cancer from a dose rate d

k = the number of hits necessary to induce the tumor

a = a constant

when: k = 1, see the one-hit model.

Geometric Mean -- The nth root of the product of n values.

Guidelines -- Principles and procedures to set basic requirements for general limits of acceptability for assessments.

Guidelines for Carcinogen Risk Assessment -- U.S. EPA guidelines intended to guide Agency evaluation of suspect carcinogens in line with statutory policies and procedures. See FR 33992-34003, September 24, 1986.

Guidelines for Exposure Assessment -- U.S. EPA guidelines intended to guide Agency analysis of exposure assessment data in line with statutory policies and procedures. See 51 FR 34042-34054, September 24, 1986.

Guidelines for Health Assessment of Suspect Developmental Toxicants -- U.S. EPA guidelines intended to guide Agency analysis of developmental toxicity data in line with statutory policies and procedures. See 51 FR 34028-34040, September 24, 1986.

Guidelines for the Health Risk Assessment of Chemical Mixtures -- U.S. EPA guidelines intended to guide Agency analysis of information relating to health effects data on chemical mixtures in line with statutory policies and procedures. See 51 FR 34014-34025, September 24, 1986.

Guidelines for Mutagenicity Risk Assessment -- U.S. EPA guidelines intended to guide Agency analysis of mutagenicity data as related to heritable mutagenic risks, in line with statutory policies and procedures. See 51 FR 34006-34012, September 24, 1986.

Hazard assessment -- A determination is made as to whether human exposure to the agent in question has the potential to increase the incidence of cancer.

Health Advisory -- An estimate of acceptable drinking water levels for a chemical substance based on health effects information; a Health Advisory is not a legally enforceable federal standard, but serves as technical guidance to assist federal, state, and local officials.

Health hazard (types of) --

1. Acute toxicity -- The older term used to describe immediate toxicity. Its former use was associated with toxic effects that were severe (e.g., mortality) in contrast to the term "subacute toxicity" that was

associated with toxic effects that were less severe. The term "acute toxicity" is often confused with that of acute exposure.

2. Allergic reaction -- Adverse reaction to a chemical resulting from previous sensitization to that chemical or to a structurally similar one.

3. Chronic toxicity -- The older term used to describe delayed toxicity. However, the term "chronic toxicity" also refers to effects that persist over a long period of time whether or not they occur immediately or are delayed. The term "chronic toxicity" is often confused with that of chronic exposure.

4. Idiosyncratic reaction -- A genetically determined abnormal reactivity to a chemical.

5. Immediate versus delayed toxicity -- Immediate effects occur or develop rapidly after a single administration of a substance, while delayed effects are those that occur after the lapse of some time. These effects have also been referred to as acute and chronic, respectively.

6. Reversible versus irreversible toxicity -- Reversible toxic effects are those that can be repaired, usually by a specific tissue's ability to regenerate or mend itself after chemical exposure, while irreversible toxic effects are those that cannot be repaired.

7. Local versus systemic toxicity -- Local effects refer to those that occur at the site of first contact between the biological system and the toxicant; systemic effects are those that are elicited after absorption and distribution of the toxicant from its entry point to a distant site.

Incidence -- The number of new cases of a disease within a specified period of time.

Initiation -- The ability of an agent to induce a change in a tissue which leads to the induction of tumors after a second agent, called a promoter, is administered to the tissue repeatedly. See also Promoter.

Latency period -- The time between the initial induction of a health effect and the manifestation (or detection) of the health effect; crudely estimated as the time (or some fraction of the time) from first exposure to detection of the effect.

Limited evidence -- According to the U.S. EPA's Guidelines for

Carcinogen Risk Assessment, limited evidence is a collection of facts and accepted scientific inferences which suggests that the agent may be causing an effect, but this suggestion is not strong enough to be considered established fact.

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Linearized multistage procedure -- The modified form of the multistage model (see Multistage Model). The constant q_1 is forced to be positive (>0) in the estimation algorithm and is also the slope of the dose-response curve at low doses. The upper confidence limit of q_1 (called q_1^*) is called the slope factor.

Logit model -- A dose-response model of the form

$$P(d) = 1/[1 + \exp -(a + b \log d)]$$

where P(d) is the probability of toxic effects from a continuous dose rate d, and a and b are constants.

Lowest-observed-adverse-effect level (LOAEL) -- The lowest exposure level at which there are statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group.

Lowest-effect level (LEL) -- Same as LOAEL.

Malignant -- Tending to become progressively worse and to result in death if not treated; having the properties of anaplasia, invasiveness, and metastasis.

Metastasis -- The transfer of disease from one organ or part to another not directly connected with it; adj., metastatic.

Model -- A mathematical function with parameters which can be adjusted so that the function closely describes a set of empirical data. A "mathematical" or "mechanistic" model is usually based on biological or physical mechanisms, and has model parameters that have real world interpretation. In contrast, "statistical" or "empirical" models are curve-fitting to data where the math function used is selected for its numerical properties. Extrapolation from mechanistic models (e.g., pharmacokinetic equations) usually carries higher confidence than extrapolation using empirical models (e.g., logit).

Modifying factor (MF) -- An uncertainty factor that is greater than zero and less than or equal to 10; the magnitude of the MF depends upon the professional assessment of scientific uncertainties of the study and database not explicitly treated with the standard uncertainty factors (e.g., the completeness of the overall data base and the number of species tested); the default

value for the MF is 1.

Multistage model -- A dose-response model often expressed in the form

$$P(d) = 1 - \exp\{-[p(0) + q(1)d + q(2)d^{**}2 + \ldots + q(k)d^{**}k]\}$$

where P(d) is the probability of cancer from a continuous dose rate d, the q(0) to q(k) are the constants, and k is the number of dose groups (or, if less, k is the number of biological stages believed to be required in the carcinogenesis process). Under the multistage model, it is assumed that cancer is initiated by cell mutations in a finite series of steps. A one-stage model is equivalent to a one-hit model.

No data -- according to the U.S. EPA Guidelines for Carcinogen Risk Assessment, "no data" describes a category of human and animal evidence in which no studies are available to permit one to draw conclusions as to the induction of a carcinogenic effect.

No evidence of carcinogenicity -- According to the U.S. EPA Guidelines for Carcinogen Risk Assessment, a situation in which there is no increased incidence of neoplasms in at least two well-designed and well-conducted animal studies of adequate power and dose in different species.

No-observed-adverse-effect level (NOAEL) -- An exposure level at which there are no statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered as adverse, nor precursors to adverse effects. In an experiment with several NOAELs, the regulatory focus is primarily on the highest one, leading to the common usage of the term NOAEL as the highest exposure without adverse effect.

No-observed-effect level (NOEL) -- An exposure level at which there are no statistically or biologically significant increases in the frequency or severity of any effect between the exposed population and its appropriate control.

One-hit model -- A dose-response model of the form

$$P(d) = a - \exp(-bd)$$

where P(d) is the probability of cancer from a continuous dose rate d, and b is a constant. The one-hit model is based on the concept that a tumor can be induced after a single susceptible target or receptor has been exposed to a single effective dose unit of a substance.

Organoleptic -- Affecting or involving a sense organ as of

taste, smell, or sight.

Pharmacokinetics -- The study of the time course of absorption, distribution, metabolism, and excretion of a foreign substance (e.g., a drug or pollutant) in an organism's body.

Promoter -- In studies of skin cancer in mice, an agent that results in an increase in cancer induction when administered after the animal has been exposed to an initiator, which is generally given at a dose that would not result in tumor induction if given alone. A cocarcinogen differs from a promoter in that it is administered at the same time as the initiator. Cocarcinogens and promoters do not usually induce tumors when administered separately. Complete carcinogens act as both initiator and promoter. Some known promoters also have weak tumorigenic activity, and some also are initiators. Carcinogens may act as promoters in some tissue sites and as initiators in others.

Prospective study -- A study in which subjects are followed forward in time from initiation of the study. This is often called a longitudinal or cohort study.

q_1^* -- Upper bound on the slope of the low-dose linearized multistage procedure.

Reference Dose (RfD) -- An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime.

Risk -- The probability of deleterious health or environmental effects.

Risk -- The probability of injury, disease, or death under specific circumstances. In quantitative terms, risk is expressed in values ranging from zero (representing the certainty that harm will not occur) to one (representing the certainty that harm will occur). The following are examples showing the manner in which risk is expressed: E-4 = a risk of 1/10,000; E-5 = a risk of 1/100,000; E-6 = a risk of 1/1,000,000. Similarly, 1.3E-3 = a risk of 1.3/1000 = 1/770; 8E-3 = a risk of 8/1000 = 1/125; and 1.2E-5 = a risk of 1.2/100,000 = 1/83,333.

Risk assessment -- The determination of the kind and degree of hazard posed by an agent, the extent to which a particular group of people has been or may be exposed to the agent, and the present or potential health risk that exists due to the agent.

Risk characterization -- The exposure and dose-response assessments are combined to produce a quantitative risk estimate

and in which the strengths and weaknesses, major assumptions, judgments, and estimates of uncertainties are discussed.

Risk management -- A decision making process that entails considerations of political, social, economic, and engineering information with risk-related information to develop, analyze, and compare regulatory options and to select the appropriate regulatory response to a potential chronic health hazard.

Safety Factor -- See Uncertainty Factor.

Short-term exposure -- Multiple or continuous exposures occurring over a week or so.

Slope Factor -- The slope of the dose-response curve in the low-dose region. When low-dose linearity cannot be assumed, the slope factor is the slope of the straight line from 0 dose (and 0 excess risk) to the dose at 1% excess risk. An upper bound on this slope is usually used instead of the slope itself. The units of the slope factor are usually expressed as 1/(mg/kg-day).

Subchronic exposure -- Multiple or continuous exposures occurring usually over 3 months.

Subchronic study -- A toxicity study designed to measure effects from subchronic exposure to a chemical.

Sufficient evidence -- According to the U.S. EPA's Guidelines for Carcinogen Risk Assessment, sufficient evidence is a collection of facts and scientific references that are definitive enough to establish that the adverse effect is caused by the agent in question.

Superfund -- Federal authority, established by the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) in 1980, to respond directly to releases or threatened releases of hazardous substances that may endanger health or welfare.

Systemic effects -- Systemic effects are those that require absorption and distribution of the toxicant to a site distant from its entry point, at which point effects are produced. Most chemicals that produce systemic toxicity do not cause a similar degree of toxicity in all organs, but usually demonstrate major toxicity to one or two organs. These are referred to as the target organs of toxicity for that chemical.

Systemic toxicity -- See Systemic effects.

Target organ of toxicity -- See Systemic effects.

Threshold -- The dose or exposure below which a significant adverse effect is not expected. Carcinogens are thought to be non-threshold chemicals, to which no exposure can be presumed to be without some risk of adverse effect.

Threshold Limit Values (TLVs) -- Recommended guidelines for occupational exposure to airborne contaminants published by the American Conference of Governmental Industrial Hygienists (ACGIH). The TLVs represent the average concentration (in mg/cu.m) for an 8-hour workday and a 40-hour work week to which nearly all workers may be repeatedly exposed, day after day, without adverse effect.

Tumor progression -- The sequence of changes in which a tumor develops from a microscopic lesion to a malignant stage.

Uncertainty Factor -- One of several, generally 10-fold factors, used in operationally deriving the Reference Dose (RfD) from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population; (2) the uncertainty in extrapolating animal data to the case of humans; (3) the uncertainty in extrapolating from data obtained in a study that is of less-than-lifetime exposure; and (4) the uncertainty in using LOAEL data rather than NOAEL data.

Unit Risk -- The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 ug/L in water, or 1 ug/cu.m in air.

Upper bound -- An estimate of the plausible upper limit to the true value of the quantity. This is usually not a statistical confidence limit.

Weibull model -- A dose-response model of the form

$$P(d) = 1 - \exp\,[-b(d^{**}m)]$$

where P(d) is the probability of cancer due to continuous dose rate d, and b and m are constants.

Weight-of-evidence of carcinogenicity -- The extent to which the available biomedical data support the hypothesis that a substance causes cancer in humans.

APPENDIX F

SAMPLE CALCULATIONS

APPENDIX F

SAMPLE CALCULATIONS

F.1. Dose Estimates

A dose rate is defined as the amount of a compound (mg) absorbed by a receptor on a daily basis per kilogram of body weight. Doses can be calculated for lifetime or less than lifetime exposures. A dose can be estimated as follows:

$$\text{Dose} = \frac{\text{Concentration in environmental medium} \times \text{contact rate} \times \text{exposure duration} \times \text{absorbed fraction}}{\text{Body weight}} = \frac{\text{mg}}{\text{kg day}}$$

Carcinogenic risk can be calculated as follows:

$$\text{Risk} = (q^*)\ (\text{dose})$$

where q^* = unit cancer risk slope factors, $(\text{mg/kg-day})^{-1}$

Sample calculations and site-specific assumptions used to estimate dose are provided in the following sections.

F.1.1 Ingestion of Drinking Water

Assumptions used to estimate the dose associated with long-term ingestion of contaminated drinking water include:

- A receptor ingests 2 liters of water/day.
- An average man weighs 70 kg.

- o 100% of the compound is absorbed in the gastrointestinal tract.

A dose rate associated with ingestion of the maximum concentration of benzene (15 ug/l in JK-GW-05) can be estimated as follows:

$$\text{Dose Rate}_{\text{ingestion}} = \frac{15 \text{ ug/l} \times 2 \text{ l/day} \times 1 \text{ mg}/10^{+3} \text{ ug}}{70 \text{ kg}}$$

$$= 15 \text{ ug/l} \times 2.86 \times 10^{-5}$$

$$\text{Dose Rate}_{\text{ingestion}} = 4.29 \times 10^{-4} \text{ mg/kg-day}$$

$$\text{Risk}_{\text{ingestion}} = (q^*) \text{ (dose rate)}_{\text{ingestion}}$$

where $q^* = 2.9 \times 10^{-2}$ $(\text{mg/kg-day})^{-1}$ (Table G-1)

$$\text{Risk}_{\text{ingestion}} = (2.9 \times 10^{-2})(\text{mg/kg-day})^{-1}(4.29 \times 10^{-4})(\text{mg/kg-day})$$

$$\text{Risk}_{\text{ingestion}} = 1.24 \times 10^{-5} \text{ (Table 10)}$$

F.1.2 Inhalation During Showering

Assumptions used to estimate the dose associated during showering include:

- o 180 liters of water are used during showering (U.S. EPA, 1985).
- o 50% of the compound volatilizes to the air (Andelman, 1985).
- o 0.33 hr/day are spent in the bathroom (U.S. EPA, 1985).
- o 0.6 m^3/hr are inhaled (U.S. EPA, 1989).

- The estimated dimension of a bathroom is 12 m^3 (U.S. EPA, 1985).

- 100% of a compound is absorbed upon entering the lungs.

- The weight of an adult is 70 kg.

A dose associated with inhalation of benzene (15 ug/l in JK-GW-05) during showering can be calculated, as follows:

$$180\ l \times 0.5 \times 15\ ug/l = 1.35 \times 10^{+3}\ ug$$

$$\frac{1.35 \times 10^{3}\ ug}{12 m^3} = 1.13 \times 10^{2}\ ug/m^3$$

$$\text{Dose Rate}_{inhalation} = \frac{(1.13 \times 10^{2}\ \frac{ug}{m^3}) \times \frac{0.6 m^3}{hr} \times (0.33\ \frac{hr}{day}) \times \frac{1\ mg}{10^{3} ug}}{70\ kg}$$

$$= 1.13 \times 10^{2} \times 2.83 \times 10^{-6}$$

$$\text{Dose Rate}_{inhalation} = 3.18 \times 10^{-4}\ mg/kg\text{-}day$$

Simplification of the above equations, the Dose Rate inhalation can be calculated, as follows:

$$\text{Dose Rate}_{inhalation} = \frac{(180)(0.5)(\text{Concentration})(0.6)(0.33)}{(12)(10^{3})(70)}$$

$$= \text{Concentration}\ (2.12 \times 10^{-5})$$

$$= 15\ (2.12 \times 10^{-5})$$

$$\text{Dose Rate}_{inhalation} = 3.18 \times 10^{-4}\ mg/kg\text{-}day$$

$$\text{Risk}_{inhalation} = (q^*)(\text{dose})$$

$$q^* = 2.9 \times 10^{-2}\ (mg/kg\text{-}day)^{-1} \quad \text{(Table G-1)}$$

$$\text{Risk}_{inhalation} = (2.9 \times 10^{-2})(mg/kg\text{-}day)^{-1}(3.18 \times 10^{-4})(mg/kg\text{-}day)$$

$$\text{Risk}_{inhalation} = 9.23 \times 10^{-6} \quad \text{(Table 10)}$$

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APPENDIX G

CONTAMINANTS AND ARARs LISTINGS

TABLE G-1
ARARs TABLE
HUMAN HEALTH EFFECTS
JACKSONVILLE LANDFILL

Compound	q^* $(mg/kg\text{-}day)^{-1}$	Maximum Contaminant Level (ug/l)	Ambient Water Quality Criteria			Health Advisory (ug/l)	RfD mg/kg/day	Other
			Ingestion of Biota	Ingestion of Water	Ingestion of Water & Fish			
Acenaphthene (83329)	---	---	---	---	---	---	---	
Acetone (67641)	---	---	---	---	---	---	1×10^{-1} (O)	
Aluminum	---	5×10^{1} (SMCLS)	---	---	---	---	---	
Anthracene (120127)	---	---	---	3.1×10^{-3} ug/l	2.8×10^{-3} ug/l	---	---	
Antimony (7440-36-0)	---	---	$4.5 \times 10^{+4}$ ug/l	1.46×10^{2} ug/l	$1.46 \times 10^{+2}$ ug/l	---	4×10^{-4} (O)	
Arsenic (7440-38-2)	1.75×10^{0} (O) 5.0×10^{1} $(mg/kg\text{-}day)^{-1}$;(I) 4.3×10^{-3} $(ug/m^3)^{-1}$;(I)	5.0×10^{1} (MCL) 5.0×10^{1} (MCLG)	1.75×10^{-2} ug/l	2.2×10^{-3} ug/l	2.2×10^{-3} ug/l	---	---	
Barium (7440-39-3)	---	1.0×10^{3} (MCL) 5.0×10^{3} (PMCL) 5.0×10^{3} (PMCLG)	---	---	---	1 day, child: 1.5×10^{3} 10 day, child: 1.5×10^{3} Longer term, child: 1.5×10^{3} Longer term, adult: 1.5×10^{3} Lifetime, adult: 1.5×10^{3}	5×10^{-2};(O) 1×10^{-4} mg/kg -day;(I) 5×10^{-4} mg/m^3;(I)	
Benzene (71432)	2.9×10^{-2} (O,I)	5	$4.0 \times 10^{+1}$ ug/l	---	6.6×10^{-1} ug/l	10 day: 235	---	
Benzo(a)anthracene (56553)	---	---	---	3.1×10^{-3} ug/l	2.8×10^{-3} ug/l	---	---	

TABLE G-1 (Continued)
ARARs TABLE
HUMAN HEALTH EFFECTS
JACKSONVILLE LANDFILL

Compound	q^* $(mg/kg\text{-}day)^{-1}$	Maximum Contaminant Level (ug/l)	Ambient Water Quality Criteria			Health Advisory (ug/l)	RfD mg/kg/day	Other
			Ingestion of Biota	Ingestion of Water	Ingestion of Water & Fish			
Benzo(b)fluoranthene (205992)	---	---	---	3.1×10^{-3} ug/l	2.8×10^{-3} ug/l	---	---	
Benzo(k)fluoranthene (207089)	---	---	---	3.1×10^{-3} ug/l	2.8×10^{-3} ug/l	---	---	
Benzoic acid (65850)	---	---	---	---	---	---	4×10^{0} (O)	
Benzo(ghi)perylene (191242)	---	---	---	3.1×10^{-3} ug/l	2.8×10^{-3} ug/l	---	---	
Benzo(a)pyrene	---	---	---	3.1×10^{-3} ug/l	2.8×10^{-3} ug/l	---	---	
Beryllium (7440-41-7)	8.4×10^{0} $(mg/kg\text{-}day)^{-1}$;(I) 2.4×10^{-3} $(ug/m^3)^{-1}$;(I)	---	1.17×10^{-1}	3.9×10^{-3} ug/l[a]	6.8×10^{-3} ug/l	---	5×10^{-3} (O)	
Bis-(2-ethyl hexyl) phthalate (=DEHP) (117817)	1.4×10^{-2};(O)	---	5×10^{4} ug/l	2.1×10^{4} ug/l	1.5×10^{4} ug/l	---	2×10^{-2} (O)	
Bromomethane (74839)	---	---	$1.6 \times 10^{+1}$ ug/l	---	1.9×10^{-1} ug/l	Longer term, child: 1.4×10^{2} Longer term, adult: 4.9×10^{2} Lifetime, adult: 1×10^{1}	1.4×10^{-3}	
2 Butanone (= methylethylketone) (78933)	---	---	---	---	---	---	5×10^{-2} (O) 9×10^{-2} mg/kg-day;(I) 3×10^{-1} mg/m^3;(I)	

TABLE G-1 (Continued)
ARARs TABLE
HUMAN HEALTH EFFECTS
JACKSONVILLE LANDFILL

Compound	q^* $(mg/kg\text{-}day)^{-1}$	Maximum Contaminant Level (ug/l)	Ambient Water Quality Criteria			Health Advisory (ug/l)	RfD mg/kg/day	Other
			Ingestion of Biota	Ingestion of Water	Ingestion of Water & Fish			
Cadmium (7440-43-9)	6.1×10^{0} $(mg/kg\text{-}day)^{-1}$;(I) 1.8×10^{-3} $(ug/m^3)^{-1}$;(I)	1.0×10^{1} (MCL) 5.0×10^{0} (MCLG)	---	1.0×10^{1} ug/l	1.0×10^{1} ug/l	1 day, child: 4.3×10^{1} 10 day, child: 4.3×10^{1} Longer term, child: 5×10^{0} Longer term, adult: 2.0×10^{1} Lifetime, adult: 5.0×10^{0}	1×10^{-3} (food) 5×10^{-4} (water)	
Chlorobenzene (108907)	---	1×10^{2} (PMCL) 1×10^{2} (PMCLG)	---	4.88×10^{2} ug/l	4.88×10^{2} ug/l	1 day, child: 4.3×10^{3} 10 day, child: 4.3×10^{3} Longer term, child: 4.3×10^{3} Longer term, adult: 1.5×10^{4} Lifetime, adult: 3.0×10^{2}	3×10^{-2};(O) 5×10^{-3} mg/kg day;(I) 0.02 mg/m^3;(I)	
Chlorophenol (95578)	---	---	---	0.1 ug/l (organoleptic)	0.1 ug/l (organoleptic)	---	5×10^{-3};(O)	
Chrysene (218019)	---	---	---	---	---	---	---	
Copper (7440-50-8)	---	1.3×10^{3} (MCL) 1.3×10^{3} (MCLG) 1.0×10^{3} (SMCL)	---	1.0×10^{3} ug/l (organoleptic)	1.0×10^{3} ug/l (organoleptic)	---	1.3 mg/l;(O)	
Chromium VI (7440-47-3)	4.1×10^{1};(I)	5.0×10^{1} (MCL)+ 1.0×10^{2} (PMCL) 1.0×10^{2} (PMCLG)	---	---	$5.0 \times 10^{+1}$ ug/l	1 day, child: 1.4×10^{3}+ 10 day, child: 1.4×10^{3}+ Longer term, child: 2.4×10^{2}+ Longer term, adult: 8.4×10^{2}+ Lifetime, adult: 1.2×10^{2}+	5×10^{-3};(O)	
Chromium III (16065-83-1)	---	5.0×10^{1} (MCL)+ 1.0×10^{2} (PMCL) 1.2×10^{2} (MCLG)+	3.43×10^{6} ug/l	1.79×10^{5}	1.7×10^{5} ug/l		1×10^{0};(O)	

+ Based on total chromium (III and IV)

TABLE G-1 (Continued)
ARARs TABLE
HUMAN HEALTH EFFECTS
JACKSONVILLE LANDFILL

Compound	q^* $(mg/kg\text{-}day)^{-1}$	Maximum Contaminant Level (ug/l)	Ambient Water Quality Criteria			Health Advisory (ug/l)	RfD mg/kg/day	Other
			Ingestion of Biota	Ingestion of Water	Ingestion of Water & Fish			
Cyanide (57-12-5)	---	---	---	2×10^5	2.0×10^2 ug/l	10 day, child: 2.20×10^2 Longer term, child: 2.20×10^2 Longer term, adult: 7.70×10^2 Lifetime, adult: 1.54×10^2	2×10^{-2};(O)	
4,4'-DDD (p,p'-Dichlorodiphenyldichloroethane) (72548)	2.4×10^{-1};(O)	---	---	---	---	---	---	
4,4'-DDE (p,p'-Dichlorodiphenyldichloroethylene) (72559)	3.4×10^{-1};(O)	---	---	---	---	---	---	
4,4'-DDT (p,p'-Dichlorodiphenyltrichloroethane) (50293)	3.4×10^{-1};(O,I)	---	2.4×10^{-5} ug/l	1.2×10^{-3} ug/l	2.4×10^{-5} ug/l	---	5×10^{-4}	
Di-n-butylphthalate (84742)	---	---	1.5×10^5 ug/l	4.4×10^4 ug/l	3.4×10^4 ug/l	---	1×10^{-1};(O)	
1,3-Dichlorobenzene (541731)	---	---	---	4.7×10^2 ug/l	4.0×10^2 ug/l	1 day, child: 9.0×10^3 10 day, child: 9.0×10^3 Longer term, child: 8.9×10^3 Longer term, adult: 3.1×10^4 Lifetime, adult: 6.2×10^2	---	

TABLE G-1 (Continued)
ARARs TABLE
HUMAN HEALTH EFFECTS
JACKSONVILLE LANDFILL

Compound	q^* $(mg/kg\text{-}day)^{-1}$	Maximum Contaminant Level (ug/l)	Ambient Water Quality Criteria			Health Advisory (ug/l)	RfD mg/kg/day	Other
			Ingestion of Biota	Ingestion of Water	Ingestion of Water & Fish			
1,4-Dichlorobenzene (106467)	2.4×10^{-2};(O)	$7.5 \times 10^{+2}$	---	4.7	4.0×10^{2} ug/l	1 day, child: 1.1×10^{4} 10 day, child: 1.1×10^{4} Longer term, child: 1.1×10^{4} Longer term, adult: 3.8×10^{4} Lifetime, adult: 7.5×10^{1}	---;(O) 7×10^{-1};(I)	
1,1-Dichloroethene (75344)	6.0×10^{-1};(O) 1.2×10^{0} ;(I)	7:MCL 7:MCLG	$1.85 \times 10^{+0}$ ug/l	3.3×10^{4} ug/l	3.3×10^{-2} ug/l	1 day, child: 2.0×10^{3} 10 day, child: 1.0×10^{3} Longer term, child: 1.0×10^{3} Longer term, adult: 3.5×10^{3} Lifetime, adult: 7	9×10^{-3};(O)	
2,4-Dichlorophenol (120832)	---	---	$3.09 \times 10^{+3}$ ug/l	$3.09 \times 10^{+3}$ ug/l	3.09×10^{3} ug/l	---	3×10^{-3};(O)	
Di-n-octylphthalate (117840)	---	---	---	---	---	---	---	
2,4-Dichlorophenoxy-acetic acid (2,4-D) (94757)	---	7×10^{1}(PMCL) 7×10^{1}(PMCLG)	---	---	---	1 day child: 1.1×10^{3} 10 day child: 3.0×10^{2} Longer term, child: 1.0×10^{2} Longer term, adult: 3.5×10^{2} Lifetime, adult: 7.0×10^{1}	---	
Dieldrin (60571)	$1.6 \times 10^{+1}$;(O,I)	---	---	1.1×10^{-3} ug/l	7.1×10^{-5} ug/l	1 day, child: 0.5 ug/l 10 day, child: 0.5 ug/l Longer term, child: 0.5 ug/l Longer term, adult: 1.8 ug/l	5×10^{-5}	

TABLE G-1 (Continued)
ARARs TABLE
HUMAN HEALTH EFFECTS
JACKSONVILLE LANDFILL

Compound	q^* $(mg/kg\text{-}day)^{-1}$	Maximum Contaminant Level (ug/l)	Ambient Water Quality Criteria			Health Advisory (ug/l)	RfD mg/kg/day	Other
			Ingestion of Biota	Ingestion of Water	Ingestion of Water & Fish			
Diethylphthalate (84662)	---	---	---	4.3×10^{5} ug/l	3.5×10^{5} ug/l	---	8×10^{-1};(O)	
Fluoranthene (206440)	---	---	---	1.9×10^{2} ug/l	4.2×10^{1} ug/l	---	---	
Ideno(1,2,3-CD)pyrene (193395)	---	---	---	---	---	---	---	
Lead (7439-92-1)	---	5.0×10^{1} (MCL) 2.0×10^{1} (MCLG)	---	$5.0 \times 10^{+1}$ ug/l	$5.0 \times 10^{+1}$ ug/l	---	---	
Manganese (7439-96-5)	---	5×10^{1} (SMCL)	---	1.0×10^{1} ug/l	1.44×10^{-1} ug/l	---	2×10^{-1};(O) 3×10^{-4} mg/kg-day;(I) 1×10^{-3} mg/m^3;(I)	
Mercury (7439-97-6)	---	2.0×10^{0} (MCL) 3.0×10^{0} (MCLG)	---	1.0×10^{1} ug/l	1.44×10^{-1} ug/l	1 day, child: 1.58×10^{0} 10 day, child: 1.58×10^{0} Longer term, child: 1.58×10^{0} Longer term, adult: 5.5×10^{0} Lifetime, adult: 1.1×10^{0}	3×10^{-4};(O)	
Methylene chloride (75092)	7.5×10^{-3};(O) 1.4×10^{-2};(I)	---	1.57×10^{1} ug/l	---	1.9×10^{-1} ug/l	1 day, child: 1.33×10^{4} 10 day, child: 1.5×10^{3} Longer term, child: 5×10^{2} Longer term, adult: 1.75×10^{3}	6×10^{-2};(O)	

TABLE G-1 (Continued)
ARARs TABLE
HUMAN HEALTH EFFECTS
JACKSONVILLE LANDFILL

Compound	q^* $(mg/kg\text{-}day)^{-1}$	Maximum Contaminant Level (ug/l)	Ambient Water Quality Criteria			Health Advisory (ug/l)	RfD mg/kg/day	Other
			Ingestion of Biota	Ingestion of Water	Ingestion of Water & Fish			
Naphthalene (91203)	---	---	---	---	---	1 day, child: 5.3×10^3 10 day, child: 5.3×10^3 Longer term, child: 5.3×10^3 Longer term, adult: 1.9×10^4	4×10^{-1};(O)	
Nickel (7440-02-0)	---	---	---	1.34×10^1 ug/l	1.34×10^1 ug/l	1 day, child: 1.0×10^3 10 day, child: 1.0×10^3 Longer term, child: 1.0×10^2 Longer term, adult: 3.5×10^2 Lifetime, adult: 1.5×10^2	2×10^{-2};(O)	
Phenanthrene (85018)	---	---	---	3.1×10^{-3} ug/l	2.8×10^{-3} ug/l	---	---	
Phenol (108952)	---	---	---	---	3.0×10^2 ug/l	---	6×10^{-1};(O)	
Polycyclic Aromatic Hydrocarbon (PAH) (59)	1.15×10^1;(O) 6.1×10^0;(I)	---	---	2.8×10^4 ug/l	---	---	---	
Pyrene (129000)	---	---	---	---	---	---	---	
Selenium (7782-49-2)	---	1.0×10^1 (MCL) 5.0×10^1 (PMCL) 5.0×10^1 (PMCLG)	---	1.0×10^1 ug/l	1.0×10^1 ug/l	---	3×10^{-3};(O) 1×10^{-3} mg/kg·day;(I) 4×10^{-3} mg/m^3;(I)	
Silver (7440-22-4)	---	5.0×10^1 (MCL)	---	$5.0 \times 10^{+1}$	$5.0 \times 10^{+1}$ ug/l	---	3×10^{-3};(O)	

TABLE G-1 (Continued)
ARARs TABLE
HUMAN HEALTH EFFECTS
JACKSONVILLE LANDFILL

Compound	q^* $(mg/kg\text{-}day)^{-1}$	Maximum Contaminant Level (ug/l)	Ambient Water Quality Criteria			Health Advisory (ug/l)	RfD mg/kg/day	Other
			Ingestion of Biota	Ingestion of Water	Ingestion of Water & Fish			
2,3,7,8-TCDD (Dioxin) (1746016)	1.56×10^{5};(O)	---	---	1.8×10^{-7} ug/l	1.3×10^{-8} ug/l	1 day, child: 1×10^{-3} 1 day, adult: 1×10^{-4} Longer term, child: 1×10^{-5} Longer term, adult: 3.5×10^{-5}	---	
Tetrachloroethene (127184)	5.1×10^{-2};(O) 3.3×10^{-3};(I)	O:MCLG	8.9×10^{0} ug/l	8.8×10^{-1} ug/l	$8. \times 10^{-1}$ ug/l	1 day, child: 2×10^{3} 10 day, child: 2×10^{3} Longer term, child: 1.4×10^{3} Longer term, adult: 5.0×10^{3} Lifetime: 1.0×10^{1}	1×10^{-2};(O)	
Thallium (7440-28-0)	---	---	---	1.78×10^{1} ug/l	1.3×10^{1} ug/l	---	7×10^{-5};(O)	
2,4,5-Trichlorophenol (95954)	---	---	---	2.6×10^{3} ug/l	1×10^{-0} ug/l	---	1×10^{-1};(O)	
1,2,4-Trichlorobenzene (120821)	---	---	---	---	---	---	2×10^{-2};(O) 3×10^{-3} mg/kg-day;(I) 9×10^{-3} mg/m^3;(I)	
2,4,6-Trichlorophenol (88062)	2×10^{-2};(O,I)	---	3.6×10^{0} ug/l	---	1.2×10^{0} ug/l	---	---	
2,4,5-Trichlorophenoxy-acetic acid (2,4,5-T) (93765)	---	---	---	---	---	1 day, child: 8×10^{2} ug/l 10 day, adult: 8×10^{2} ug/l Longer term, 10 day: 3×10^{2} ug/l Longer term, adult: 1.1×10^{3} ug/l Lifetime: 2.1×10^{1} ug/l	1×10^{-2}	

TABLE G-1 (Continued)
ARARs TABLE
HUMAN HEALTH EFFECTS
JACKSONVILLE LANDFILL

Compound	q^* $(mg/kg\text{-}day)^{-1}$	Maximum Contaminant Level (ug/l)	Ambient Water Quality Criteria			Health Advisory (ug/l)	RfD mg/kg/day	Other
			Ingestion of Biota	Ingestion of Water	Ingestion of Water & Fish			
2,4,5-Trichlorophenoxy-propionic acid (2,4,5-TP) (93721)	---	1×10^1 (MCL) 5×10^1 (PMCL) 5×10^1 (PMCLG)	---	---	---	1 day, child: 2×10^2 ug/l 10 day, child: 2×10^2 ug/l Lifetime, adult: 5.2×10^1 ug/l	8×10^{-3}	
Toluene (108883)	---	2×10^3(MCLG) 2×10^3(PMCL)	4.2×10^5 ug/l	1.5×10^4 ug/l	1.43×10^4 ug/l	1 day, child: 2.2×10^4 10 day, child: 3.5×10^3 Longer term, child: 3.5×10^3 Longer term, adult: 1.2×10^4 Lifetime: 2.4×10^3	3×10^{-1};(O) 1×10^0 mg/kg-day;(I) 5×10^0 mg/m^3;(I)	
Vinyl chloride (75014)	2.3×10^0;(O) 2.95×10^{-1};(I)	2:MCL 0:MCLG	---	2.0 ug/l	2.0 ug/l	1 day, child: 2.6×10^3 10 day, child: 2.6×10^3 Longer term, child: 1.3×10^1 Longer term, adult: 4.6×10^1	---	
Xylene (1330207)	---	4.4×10^2(MCLG)	---	---	---	1 day, child: 1.2×10^4 10 day, child: 7.8×10^3 Longer term, child: 7.8×10^3 Longer term, adult: 2.7×10^4 Lifetime: 4×10^2	2×10^0;(O) 4×10^{-1} mg/kg-day;(I) 1×10^0 mg/m^3;(I)	
Zinc (7440-66-6)	---	---	---	5.0×10^3 ug/l (organoleptic)	5.0×10^3 ug/l (organoleptic)	---	2×10^{-1};(O)	

NOTES:

(a) The q^* and RfD are calculated based on studies of oral ("O") exposure in animals, except for those indicated by "I" (animal inhalation).
(b) Recommended Maximum Contaminant Level (RMCL).

(c) Proposed Maximum Contaminant Level (PMCL).
(d) Maximum Contaminant Level (MCL).
(e) Maximum Contaminant Level Goal (MCLG).
(f) Secondary Maximum Contaminant Level (SMCLs) "apply to any contaminant in drinking water that may adversely affect the odor or appearance of such water and consequently may cause a substantial number of persons served by public water systems providing such water to discontinue its use, or that may otherwise adversely affect public welfare."
(g) The Ambient Water Quality Criteria (AWQC) for the maximum protection of human health is zero. Because zero may not be attainable, the values tabulated correspond to a 10^{-6} carcinogenic risk.
* For Be, AWQC value for ingestion of water and fish is given as 3.7×10^{-3} µg/l in PhRED, 1988.
\- Signifies criterion is not available or not applicable.

SOURCES:

IRIS (= Integrated Risk Information System), 1988 and 1989, IRIS Database, EPA, Washington, DC.
PhRED (= Public Health Risk Evaluation Database), 1988, PhRED Database, EPA, Washington, DC.
Federal Register, Vol 54, No. 97, Monday, May 22, 1989, p 22,064.
U.S. EPA, 1989, Health Effects Assessment Summary Tables, 2nd Quarter, FY 89, Prepared by Environmental Criteria and Assessment Office, OSWER (OS-230), ORD (RD-689), OERR 9200.6-303-(89-2).
U.S. EPA, 1984, Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAHs), Office of Research and Development, Office of Health and Environmental Assessment, PB86-134244, EPA/540/1-86/013.

TABLE G-2

STATE ARARs for
SURFACE WATER CONCENTRATIONS AT JACKSONVILLE LANDFILL
(All Concentrations, ug/l)

	ARAR[1]	
Chemical	Chronic Toxicity (24-hr avg-ug/l)	Acute Toxicity (never to exceed-ug/l)
PCBs	0.014	2.0
Aldrin	---	3.0
Dieldrin	0.0019	2.5
DDT (& metabolites)	0.0010	1.1
Endrin	0.0023	0.18
Toxaphene	0.0002	0.73
Chlordane	0.0043	2.4
Endosulfan	0.056	0.22
Heptachlor	0.0038	0.52
Hexachlorocyclohexane[2]	0.080	2.0
Pentachlorophenol	e[1.005(pH)-5.290]	e[1.005(pH)-4.830]
Chlorpyrifos	0.041	0.083
Selenium	35.00	260.00
Silver	0.12	e(1.72[ln(hardness)]-6.52)

(1) The State of Arkansas Surface Water Standards
(2) Total of all isomers

TABLE G-3
CONTAMINANTS FOUND AT
JACKSONVILLE LANDFILL

Carcinogens	Noncarcinogens
1,1-dichloroethene	chlorobenzene
1,4-dichlorobenzene	benzoic acid
benzene	barium
cadmium	manganese
chromium VI	selenium
methylene chloride	xylene
bis-(2-ethylhexyl) phthalate	toluene
beryllium	phenol
4,4'-DDT	chromium III
2,3,7,8-TCDD	acetone
dioxins and furans	antimony
tetrachloroethene	mercury
2,4,6-trichlorophenol	silver
arsenic	2,4,5-trichlorophenoxy-acetic acid (= 2,4,5-T)
lead	2,4,5-trichlorophenoxy-propionic acid (=2,4,5-TP)
	2,4-dichlorophenol
	2,4,5-trichlorophenol
	1,2,4-trichlorobenzene
	naphthalene
	2-chlorophenol
	nickel
	zinc
	aluminum
	copper

APPENDIX H

TOXICOLOGY PROFILES

APPENDIX H

TOXICOLOGY PROFILES

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Anthracene

Anthracene exists as colorless crystals; it has a molecular weight of 178.24 and a molecular formula of $C_{14}H_{10}$. It is soluble in a variety of organic solvents, including ethanol, methanol, benzene, toluene, and carbon disulfide (Windholz, 1983), but it is almost insoluble in water (Pearlman, et al., 1984). This compound is susceptible to oxidation by ozone, peroxides and other oxidants (NAS, 1972). The commercial production of anthrancene in the U.S. is believed to have been stopped in 1982 (IARC, 1983), but it is still imported into this country (USITC, 1984).

The fate and transport of anthracene in aquatic media is important; it's been estimated that greater than 95% of environmental anthracene will reside in the aquatic compartment. Anthracene is ubiquitous in the aquatic environment. The fate and transport of anthracene in surface waters will depend on the nature of the water. In most waters, the loss of anthracene is mainly due to photolysis and biodegradation (Mackay, et al., 1985); however, in a very shallow, fast-flowing clear water, volatilization and photolysis will play dominant roles in determining the fate of anthracene (Southworth, 1979). In deep, slow-flowing and muddy waters, microbial degradation and adsorption may account for the major losses of anthracene from water (Southworth, 1979). Therefore, the half-life of anthracene in natural surface waters depends on the nature of the water bodies. In very shallow, fast-flowing and clear water, its half-life may be about 1-hour (Southworth, 1979; Herbes, et al, 1980). On the other hand, the half-life may be as high as 29 days in a deep eutrophic pond (Zepp, 1980).

The atmospheric half-life of anthracene may vary from hours to days (Atkinson, 1985; Korfmacher, et al., 1980; Behymer and Hites, 1985; Lunde and Bjoerseth, 1977).

The fate and transport of anthracene in soils is not well studied. Both biodegradation and abiotic processes will degrade anthracene in soils (Bossert, et al., 1984). The half-life of anthracene in soil may be about 1 month (Bossert, et al, 1984). Anthracene may not leach from most soils because of its high Koc value which means anthracene will adsorb strongly to soil; however, it may leach through soils that have attained the breakthrough capacity for anthracene sorption (Piet and Morra, 1979).

There is relatively little information concerning the toxicity of anthracene to aquatic organisms. Acutely toxic concentrations range from 1.9 ug/l for Daphnia pulex (Allred and Giesy, 1985; Oris, et al., 1984) to 3030 ug/l for Daphnia magna (Bobra, et al, 1983). Some of this variability may be explained by the fact that anthracene toxicity is affected by lighting conditions, with increased toxicity under natural sunlight and ultraviolet radiation rather than fluorescent lights (Allred and Giesy, 1985; Bowling, et al., 1983; Kagan, et al., 1985).

Pertinent data regarding chronic or subchronic toxic effects, teratogenicity or other reproductive effects of anthracene could not be located in the available literature (U.S. EPA, 1987).

Anthracene has been classified as a Group D, not classified, chemical for carcinogenicity by the U.S. EPA (1987). This is because the carcinogenicity data taken together and available for humans and animals are inadequate. Administration of diets that supplied anthracene for 78 weeks and then observed for life produced tumors in 2/28 rats (a liver sarcoma and a uterine adenocarcinoma) (Schmahl, 1955). However, a control group was not used and the tumors were not ascribed to treatment. A single intrapulmonary injection of anthracene did not induce local neoplastic responses in rats after 4-55 weeks of observation (Stanton, et al., 1972).

Twice or thrice weekly skin applications of anthracene for life did not produce local tumors in mice (Bachmann, et al., 1937; Wynder and Hoffmann, 1959; Miescher, 1942), but contradictory results were obtained when anthracene was applied to mouse skin with concurrent or subsequent ultraviolet irradiation (Heller, 1950; Forbes, et al., 1976). Mouse skin initiation-promotion assays using croton oil (Salaman and Roe, 1956) or TPA (Scribner, 1973) as the promoter did not indicate a tumor initiating effect for anthracene. Weekly subcutaneous injections of anthracene starting at 6 weeks given for life to rats, (Pollia, 1941; Schmahl, 1955; Boyland and Burrows, 1935), weekly intraperitoneal injections for 33 weeks to rats (Schmahl, 1955) or brain or eye implants of anthracene in rabbits for 4.5 years (Russell, 1947) did not produce local tumors. However, these findings should be regarded as inconclusive because of the inadequacies in experimental design.

Anthracene has been tested in numerous mutagenicity and other short-term assays with primarily negative results (IARC, 1983; Langenbach, et al., 1983; Lubet, et al., 1983; Ved Brat, et al., 1983; Mamber, et al., 1984; Quillardet, et al., 1985). In vivo and in vitro tests included DNA damage, mutation, cytogenicity and transformation assays in bacterial, yeast and mammalian systems.

Exposure criteria and TLVs have been developed for polycyclic aromatic hydrocarbons (PAH) as a class, as well as for several individual polycyclic aromatic hydrocarbons. OSHA (1985) has set an 8-hour TWA concentration limit of 0.2 mg/m^3 for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, benzo(a)pyrene, phenanthrene, acridine, chrysene, pyrene). NIOSH (1977) recommended a concentration limit for coal tar, coal tar pitch, creosote and mixtures of the substances of 0.1 mg/m^3 of the cyclohexane-extractable fraction of the sample, determined as a 10-

hour TWA. The U.S. EPA (1980) has recommended a concentration limit of 28 ng/l for the sum of all carcinogenic polycyclic aromatic hydrocarbons in ambient water. Daily consumption of water containing 28 ng/l of the carcinogenic aromatic hydrocarbon, benzo(a)pyrene, over an entire lifetime is estimated to keep the lifetime risk of cancer development below one chance in 100,000.

Data were insufficient to derive an ADI (also known as RfD), q_1^*, RQ or F factor, and thus, a hazard ranking is precluded (U.S. EPA, 1987).

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Arsenic (As):

Arsenic, atomic weight 74.92, is an element showing metallic as well as nonmetallic properties. Inorganic compounds of every oxidation state and numerous metalloorganic compounds with C-As, bonds are known. The major stable valences of arsenic are 3-, 3+ and 5+. All soluble compounds of arsenic are considered poisonous to humans (Seiler and Sigel, 1988).

Up to the early 1940s, arsenic compounds were used to exterminate the vine louse in wine-growing regions and as a general poison against rodents. To a limited extent, arsenic compounds are still used to defoliate cotton fields to render possible a mechanized harvest. They are furthermore applied as fungicides, insecticides and for timber preservation (Woolson, E. A., 1975). Copper arsenite (Scheele green) and copper arsenite acetate (Schweinfurt Green) have been used as pigments whereas lead arsenate served as an insecticide and fungicide.

Arsenic is ubiquitous, it is found in various concentrations in soil, atmosphere, minerals, sea- and freshwater and is present in various biological substrates. Certain bacteria and other microbiological systems are able to methylate inorganic arsenic compounds by different mechanisms (Braman, R., 1975; R.S. Braman, 1977; G. R. Sandberg, 1975; and D. L. Johnson and R. M. Burke, 1978). Arsenic can enter the soil from wet and dry precipitation of atmospheric arsenic, from runoff of surface waters and from disposal of arsenic-containing waters. The fate of arsenic in soil is inadequately studied. However, the fate may be dependent on the nature of soil. The factors that may significantly determine the fate of soil arsenic are organic matter content, clay content and microbial activity capable of metabolizing arsenic. Soil containing high levels of sorptive materials, such as clay or

organic matter, are likely to retard the leachability of arsenic in soils. However, arsenic may leach into groundwater from soils with low sorptive capacity. Indirect evidence suggests that leaching of arsenic from soils into groundwater may be quite common (Page, 1981).

Arsenic can enter aquatic media through atmospheric wet and dry deposition (Boyle and Jonasson, 1973), through runoff from soils and through industrial discharge into surface waters. The BCFs for arsenic in aquatic organisms have been determined by a few investigators and have been found to vary from 333-6000 (Callahan, et al., 1979).

Absorption of arsenic from the GI tract is predominantly governed by the solubility of the specific compound administered. Coulson, et al., (1935) reported that solutions of either trivalent or pentavalent soluble inorganic arsenic compounds were almost completely absorbed from the GI tracts of rats. Coulson, et al. (1935) and Ray-Bettley and O'Shea (1975) estimated that >95% of the inorganic arsenic that man consumes is absorbed.

Absorption of arsenic from the respiratory tract is governed by the specific chemical compound and, in the case of aerosols or dusts, the particle size.

The amount of arsenic that results in intoxication varies in warm-blooded organisms to a considerable degree. The overt symptoms of arsenic intoxification are dependent on the amount and kind of arsenic compound (Geldmacher, M., et al, 1975; V. E. Schmidt, 1983; E. A. Woolson, 1975; and O. Oster and W. Prellwitz, 1985). Also, the mode of application has a special importance. There is also a continuous transition between the different symptoms of arsenic poisoning.

In acute intoxification, two forms of poisoning are evident, including: the paralytic and gastrointestinal forms (Seiler, H. G., and H. Sigel, 1988). The paralytic form will be observed if large doses of arsenic are absorbed quickly. Within 1-2 hour a severe shock develops by a general paralysis of the capillaries, accompanied by acute excitability of the brain, often with signs of delirium. Death occurs by general paralysis. In the gastrointestinal form of arsenic poisoning abdominal symptoms are dominant: nausea, headache, intense pain, vomiting, and diarrhea, that are caused by paralysis of the central mechanism of the capillary control in the intestinal tract. A capillary transudation of plasma occurs that forms vesicles under the gastrointestinal mucosa and there also develops a statis in the tissues of liver, kidneys, and spleen. The vesicles rupture and the result is a sloughing of the epithelium. These injuries result also in a subsequent decrease in blood volume. Blood pressure falls to shock levels. This in turn results in disturbed heart action and in failure of the vital function of the brain, resulting in death due to enormous loss of water, kidney failure and anuria.

Not all acute poisonings show the exact phenotype described above. In less severe cases, nausea and diarrhea diminish after some time. In connection with polyneurotic symptoms, different skin eruptions develop that disappear after the supply of arsenic is ceased.

In chronic oral toxicity of inorganic arsenic compounds, the most common effects observed in humans were skin lesions, peripheral vascular disease and peripheral neuropathy (U.S. EPA, 1984). In experimental animals, decreased survival without apparent cause was frequently observed. The only species, other than humans, in which dermal pathologies were observed was the mouse, and these changes were relatively mild and did not include

skin cancers. Peripheral neuropathies were not observed in any experimental animals tested. Hepatic degenerative changes and renal damage were frequently observed in rats, but not in other species (Baroni, et al., 1963; Shubik, et al., 1962; Byron, et al., 1967; Schroeder, et al., 1968; Schroeder and Balassa, 1967; Kroes, et al., 1974; Zaldivar and Ghai, 1980; Borgono and Greiber, 1972; Zaldivar, 1974; Borgono, et al., 1977; Silver and Wainman, 1952).

Tseng (1977) investigated the relationship between blackfoot disease, a peripheral circulatory disease characterized by gangrene of the extremities, and the arsenic concentration in drinking water of residents of the southwest coast of Taiwan. Arsenic concentrations ranged from 0.001 to 1.82 mg/l. The overall prevalence rate for blackfoot disease was 8.9/1,000, with a positive correlation between the prevalence rate and arsenic concentration and duration of intake. This study established a NOAEL of 0.001 - 0.017 mg/l for blackfoot disease.

Chronic inhalation exposure to arsenic compounds resulted in symptoms similar to those observed following oral exposure. For example, Landau, et al. (1977) reported a direct relationship between the length and intensity of exposure of smelter workers to airborne arsenic, predominantly as arsenic trioxide, and alterations in peripheral nerve function.

In teratogenicity studies, oral doses of up to 40 mg/kg body weight/day of an arsenic compound for three consecutive days results in decreased fetal weights (Matsumoto, et al., 1973a, b). Boxley, et al. (1981) reported a single oral dose of 40 - 45 mg/kg body weight on any gestation day between days 8 - 15 will produce adverse effects in developing mice.

Numerous arsenic compounds, particularly trivalent inorganics, have been associated with lung and skin carcinomas in humans. Tseng, et al. (1968) and Tsent (1977) surveyed 40,421 residents of Taiwan who consumed artesian well water containing 0.01 - 1.8 mg arsenic/l for 45 - 60 years. There was a dose-response relationship between the prevalence of skin cancer and arsenic consumption, based on arsenic concentrations in different wells and length of exposure (age). The overall incidence of skin cancer was 10.6/1,000, with a maximum incidence of 209.6/1,000 in males over 70 years of age.

Arsenic sulfides and arsenic trioxide have also been associated with the development of malignancies in 7 patients in Singapore (Tay and Slah, 1975). These patients consumed herbal preparations containing arsenic for up to 15 years. Malignancies of the skin were reported in 6 of the 74 patients and malignancies of the visceral organs in 4 of 74.

Numerous investigators reported an association between occupational exposure to arsenic and the development of tumors, with exposure primarily by the respiratory route. Pinto, et al. (1978) found an increase in deaths from all cancers, particularly respiratory cancer, at copper smelter workers.

Animal bioassays for carcinogenicity using arsenic compounds administered orally or the inhalation route of exposure have generally produced negative results (Hueper and Payne, 1962; and Baroni, et a., 1963; Ishinishi, et al., 1976, 1977).

U.S. EPA (1984) classified arsenic as a group 1 compound because there is "sufficient evidence that inorganic arsenic compounds are skin and lung carcinogens in humans." IARC (1980)

found that "there is inadequate evidence for the carcinogenicity of arsenic compounds in animals."

ACGIH (1980) established a TWA of 0.2 mg/m^3 for arsenic and soluble arsenic compounds. OSHA established a standard of 0.01 mg/m^3 for airborne inorganic arsenic (U.S. EPA, 1980). The U.S. EPA (1980) recommended a criterion of 22 ng/l, that would result in an estimated excess cancer risk of 10^{-5}. The EPA (1980) estimated that an oral dose of 50 ng/day, corresponding to a q_1^*H of 1.4×10^{-11} $(mg/kg/day)^{-1}$), would result in an excess cancer risk of 10^{-5}. The IRIS (= Integrated Risk Information System, 1988-89) lists the q^* oral = 1.75 $(mg/kg-day)^{-1}$ and the $q^*_{inhalation} = 5.0 \times 10^1$ $mg/kg-day)^{-1}$.

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Benz(a)anthracene (Benzoanthracene)

Benz(a)anthracene has a molecular weight of 228.30 and a molecular formula of $C_{18}H_{12}$. Benz(a)anthracene exists as colorless leaflets or plates (Sax, 1984). Benz(a)anthracene is one of the family of polycyclic aromatic hydrocarbons (PAH) formed as a result of incomplete combustion of organic material. PAH, including benz(a)anthracene, are ubiquitous in the environment, being found in ambient air, food, water, soils and sediment (U.S. EPA, 1979b). The PAH class contains a number of potent carcinogens (e.g., benzo(a)pyrene), weak carcinogens (e.g., benz(a)anthracene), and cocarcinogens (e.g., fluoranthene), as well as numerous noncarcinogens (U.S. EPA, 1979b).

PAH that contain more than three rings (such as benz(a)anthracene) are relatively stable in the environment, and may be transported in air and water by adsorption to particulate matter. However, biodegradation and chemical treatment are effective in eliminating most PAH in the environment (U.S. EPA, 1980; see also U.S. EPA, 1979c).

Benz(a)anthracene apparently does not display remarkably acute toxicity; the chronic toxicity of benz(a)anthracene has not been extensively studied. The repeated injection of benz(a)anthracene in mice for 40 weeks had little apparent effect on longevity or organ weights (U.S. EPA, 1979b).

Pertinent data could not be located in the available literature concerning the possible teratogenicity of benz(a)anthracene. Other related PAH are apparently not significantly teratogenic in mammals (U.S. EPA, 1979a).

Benz(a)anthracene is recognized as a weak carcinogen in mammals (U.S. EPA, 1979a,b; U.S. EPA, 1980). It is a tumor

initiator on the skin of mice, but failed to yield significant results in the strain A mouse pulmonary tumor bioassay system. Benz(a)anthracene has shown weak mutagenic activity in several test systems, including Ames Salmonella assay, somatic cells in culture, and sister chromatid exchange in Chinese hamster cells (U.S. EPA, 1979b).

No acute toxicity or chronic toxicity data have been published for benz(a)anthracene on freshwater or marine species (U.S. EPA, 1980). The only toxicity data available for benz(a)anthracene for fish is an 87% mortality on fresh-water bluegill sunfish, Lepomis Macrochirus, exposed to 1,000 ug/l for six months (Brown, et al., 1975).

There are no established exposure criteria for benz(a)anthracene. However, PAH as a class are regulated by several authorities. The World Health Organization (WHO, 1970) has recommended that the concentration of PAH in drinking water (measured as the total of fluoranthene, benzo(g,h,i)-perylene, benzo(b)-fluoranthene, benzo(k)-fluoranthene, indeno (1,2,3-cd)-pyrene, and benzo(a)-pyrene) not to exceed 0.2 ug/l. Occupational exposure criteria have been established for coke oven emissions, coal tar products, and coal tar pitch volatiles, all of which contain large amounts of PAH, including benz(a)anthracene (U.S. EPA, 1979a).

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Benzene

Benzene is a clear, colorless liquid with a molecular weight of 78.12 and a molecular formula of C_6H_6, and is in the monocyclic aromatic hydrocarbon chemical class of compounds. Evaporation is expected to be the predominant loss mechanism from the soil surface. Considering its reasonably high water solubility and reasonably low soil-water distribution coefficient (Chiou, et al, 1983), benzene is expected to leach from soil. Both volatilization and biodegradation may account for the primary loss of benzene from soil before it has had the chance to leach appreciably from soil to groundwater (U.S. EPA, 1984).

Subchronic oral administration of benzene to rats resulted in hematopoietic effects, leucopenia and erythrocytopenia (U.S. EPA, 1984). Animals exposed to benzene vapors during inhalation experiments had leukopenia (Deichmann, et al, 1963), increased spleen and testes weights, depressed growth, unspecified histopathological changes in bone marrow, spleen, kidney and testes, as well as necrosis (Wolf, et al, 1956). Mice exposed to benzene vapors subchronically experienced 50% mortality, lymphocytopenia, anemia, reduced bone marrow, reduced spleen cellularity and reduced spleen weight.

Chronic inhalation of benzene in laboratory animals resulted in lymphocytopenia, anemia and bone marrow hypoplasia (Snyder, et al, 1978), decreased survival rates and decreased body weight gains.

Chronic inhalation of benzene vapor in humans caused pancytopenia, that is, a reduction of red and white blood cells and platelets (U.S. EPA, 1980b; IARC, 1982; ACGIH, 1980; NIOSH, 1974). Severe pancytopenia (aplastic anemia) as a result of

benzene exposure is often associated with marked reduction in bone marrow cellularity (U.S. EPA, 1980b; IARC, 1982). Occupational exposure to benzene has been linked consistently with blood dyscrasias (Greenburg, 1926; Savilahti, 1956; Vigliani & Saita, 1964). The lower limit of exposure that will result in hematologic effects in humans is not well defined, but it is thought to be <100 ppm (Hardy & Elkins, 1948; Pagnotto, et al, 1961; Pagnotto, 1972). There is some evidence for impairment of the immune system in humans chronically exposed to benzene (Lange, et al, 1973; Smolik, et al, 1973). Chronic benzene exposure in humans also produces acute myelogenous leukemia in humans (U.S. EPA, 1980 b; IARC, 1982).

Benzene inhalation in mice did not produce major malformations (teratologies) but it resulted in minor skeletal variants that are considered to be indicative of delayed development (Murray, et al, 1979). Fetal rats exposed to benzene vapors, in utero, were reported to have delayed ossification and skeletal variants or anomalies. One fetus had exencephaly, one had angulated ribs and two had out-of-sequence ossification of the forefeet and dilated brain ventricles (Kuna & Kapp, 1981).

Benzene metabolism, and therefore benzene toxicity, is altered by simultaneous exposure to some other solvents (e.g., xylene, toluene) because these aromatic solvents are oxidized by many of the same hepatic enzyme systems (Ikeda, et al, 1972; U.S. EPA, 1980b). Reported hematotoxic effects of benzene in humans may be a synergistic result of simultaneous exposure to other solvents (e.g., xylene, toluene), since benzene itself does not induce leukemia in animals (NAS, 1976; U.S. EPA, 1980b). Since benzene metabolites rather than the parent compound are suspected of inducing bone marrow toxicity, inhibition of benzene metabolism (hydroxylation) by toluene may result in increased toxic effects

of the parent compound instead of benzene metabolites (Andrews, et al, 1977; U.S. EPA, 1980b).

IARC (1982) summarized many case studies that suggest a causal relationship between benzene exposure by inhalation and leukemia in humans (Goldstein, 1977). A number of epidemiologic studies have associated occupational exposure to benzene by inhalation with an increased incidence of leukemia (Aksoy, et al, 1974; Infante, et al, 1977a & b; Ott, et al 1978; IARC, 1982; and U.S. EPA, 1978a & 1980b). Akso, et al, (1974) reported the incidence of 26 leukemia cases in 28,500 subjects who were employed in the shoe industry in Turkey for an average of 9.7 years (range 1-15 years) and having a mean age of 34.2 years. This corresponded to an annual incidence of leukemia of 13/100,000 workers, compared to an annual estimate of 6/100,000 for the general population and yields a relative risk of 2. Infante, et al (1977a, b) reported a statistically significant excess of leukemia in 748 white males exposed to benzene vapors during the manufacture of a rubber product from 1940-1949. Infante, et al (1977a) reported a 5-fold excessive risk of all leukemia and a 10-fold excessive risk of myelocytic and monocytic leukemias combined. The lag period for chronic myelocytic leukemia was 2 years from initial benzene exposure, while the lag period for acute myelocytic and monocytic leukemia was 10-21 years.

Chronic oral administration of benzene in lab animals resulted in zymbal gland and mammary gland carcinomas (Maltoni & Scarnato, 1979); inhalation of benzene vapors produced hematoporetic neoplasms in mice and myelogenous leukemia (Snyder, et al, 1980).

Benzene induced chromosomal aberrations in bone marrow cells from rabbits (Kissling & Speck, 1971), mice (Meyne & Legator, 1978, 1980) and rats (Dean, 1969; Philip & Krough Jensen, 1970; Lyapkalo,

1973; Lyon, 1976; Dobrokhotov & Enikeev, 1977; Anderson & Richardson, 1979). Significant increases in chromosomal aberrations from workers exposed to benzene have been reported; some of these persisted for years after cessation of exposure (IARC, 1982).

Benzene is assigned to Group A, a human carcinogen (U.S. EPA, 1984) from available data on benzene. The human case reports of carcinogenicity, and epidemiology studies provide sufficient evidence for the carcinogenicity of benzene to humans. Animal studies demonstrating increased incidence of zymbal and mammary gland carcinoma and hematopoietic tumors in mice provide corroborative evidence supporting a carcinogenic role for benzene.

The TLV of benzene in air is 100 ppm (Sax, 1984). Regulations for benzene have set a TWA at 10 ppm and a ceiling of 25 ppm (OSHA, 1980) for benzene; guidelines have been set by NIOSH (1980) at 1 ppm for a ceiling. Water criteria estimated by the U.S. EPA (1980b) for the consumption of benzene from water and lifetime contaminated fish have set increased risk levels of 10^{-7}, 10^{-6} and 10^{-5} for benzene concentration of 0.066, 0.66 and 6.6 ug/l, respectively. An oral carcinogenic potency factors (q_1^*) of 5.2 x 10^{-2} $(mg/kg/day)^{-1}$ was estimated for humans (U.S. EPA, 1980b). For inhalation, the carcinogenic potency factor (q_1^*) for benzene of 2.6 x 10^{-2} $(mg/kg/day)^{-1}$ was estimated (U.S. EPA, 1984).

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Benzo(a)pyrene

Benzo(a)pyrene exists as yellow crystals (Sax, 1984) with a molecular weight of 252 (Mabey, et al, 1981). Benzo(a)pyrene is in the general chemical class of compounds called polycyclic aromatic hydrocarbons (PAH). Because it has a high octanol/water partition coefficient and a low water solubility, the compound is expected to have very low mobility in soils, particularly in soils with a high content of organic matter (U.S. EPA, 1984). The bioconcentration factor is high (estimated at 28,200, U.S. EPA, 1980).

There were no apparent teratogenic, reproductive, and embryotoxic effects reported by Rigdon and Neal (1965) in male and female mice fed diets containing benzo(a)pyrene at levels of 0, 250, 500 or 1,000 mg/kg over various time spans during mating, gestation and lactation. No effect on fetal body weights was observed in mice fed benzo(a)pyrene orally (Mackenzie & Angevine, 1981). However, there was a marked and specific reduction of gonadal weight; reduced fertility and reproductive capacity were observed among the offspring. At 40 mg/kg/day, almost complete sterility was observed in both sexes of offspring.

U.S. EPA classified benzo(a)pyrene as a Group B_2 chemical, a probable human carcinogen (U.S. EPA, 1984). Benzo(a)pyrene is both a local and a systemic carcinogen, producing tumors in rats, mice, hamsters, guinea pigs, rabbits and monkeys following oral, inhalation or dermal exposure. Benzo(a)pyrene is an initiator of skin carcinogensis in mice and also produces tumors following single doses or prenatal exposure. Benzo(a)pyrene has been used extensively as a model carcinogen and as a positive control in a variety of short-term tests. IARC (1982) reported that there is sufficient evidence to indicate that benzo(a)pyrene is an animal

carcinogen, but limited evidence to show that it is a human carcinogen.

In animal studies, a dose-response relationship was noted for the incidence of stomach tumors (papillomas and carcinomas) in male and female mice fed orally with 1-250 ppm benzo(a)pyrene for up to 197 days (Neal & Rigdon, 1967). A dose-response increase in stomach tumors compared to controls were reported in animals treated with 20-250 ppm benzo(a)pyrene; lung adenoma and leukemia were noted in mice treated with 250 and 1,000 ppm benzo(a)pyrene (Rigdon & Neal, 1966, 1969).

Hamsters exposed to benzo(a)pyrene by inhalation developed tumors of the respiratory tract; those exposed to higher doses developed tumors of the respiratory and digestive tracts. No tumors were seen in control animals (Thyssen, et al, 1981).

Benzo(a)pyrene has been studied in short-term tests, yielding positive results in assays for bacterial DNA repair, bacteriophage induction and bacterial mutation, mutation in Drosophila melanogaster; DNA binding, DNA repair, SCE, chromosomal aberration, point mutation and transformation in mammalian cells in culture; and in tests in mammals in vivo, including DNA binding, SCE, chromosomal aberration, sperm abnormality and the specific locus (spot) test (IARC, 1982; de Serres & Ashby, 1981; Hollstein & McCann, 1979).

Numerous epidemiologic studies of human populations (primarily worker groups) have shown a clear association between exposure to PAH-containing materials (e.g., soots, tars, oils) and increased cancer risk (Santodonato, et al, 1981; IARC, 1973, 1983; U.S. EPA, 1981). Few case reports are available regarding the direct carcinogenic effects of benzo(a)pyrene on humans. Cottini &

Mazzone (1939) applied a 1% solution of benzo(a)pyrene in benzene to small exposed skin areas of the 26 patients. After 120 daily applications for 4 months, regressive verrucae (warts) developed in all the patients. Although reversible and apparently benign, these changes were thought to represent early stages of neoplastic proliferation. Mazzone (1939) applied a 1% solution of benzo(a)pyrene in benzene to small exposed skin areas of the 26 patients. After 120 daily applications for 4 months, regressive verrucae (warts) developed in all the patients. Although reversible and apparently benign, these changes were thought to represent early stages of neoplastic proliferation. Similar cases of epidermal changes were reported to have occurred in men accidentally exposed to benzo(a)pyrene (Rhoads, et al, 1954; Klar, 1938).

Exposure criteria and TLVs have been developed for PAH as a class and for several individual PAHs. OSHA has set an 8-hour TWA concentration limit of 0.2 mg/m^3 for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, benzo(a)pyrene, phenanthrene, acridine, chrysene, pyrene) (Code of Federal Regulations, 1981). NIOSH (1977) recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m^3 of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA. The U.S. EPA (1980) recommended a concentration limit of 28 ng/l for the sum of all carcinogenic PAHs in ambient water. This value is an environmental quality criteria for PAH recommended for ambient water to protect humans against adverse health effects. The U.S. EPA (1984) used the mouse data of Neal & Rigdon (1967) to compute a q_1^* of 11.53 $(mg/kg/day)^{-1}$ from oral administration data and a q_1^* of 6.11 $(mg/kg/day)^{-1}$ from inhalation exposure data in hamsters.

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Chromium (Chrome):

Metallic Chromium is steel grey in color, has an atomic formula of Cr and an atomic weight of 52 (Sax, 1984).

Chromium exists in nature as a mixture of 4 different oxidation states. Cr (III) plays a role in nutrition as an essential nutrient at low levels in the body and is the predominant form while hexavalent Cr (Cr VI) is known to be carcinogenic. Most of the monitoring information of the ambient environment provides information only on total elemental chromium levels. Elemental chromium does not occur naturally. There are 4 oxidation states, but only two of them, Cr (III) and Cr (VI), appear to be important, owing to their predominance and stability in the ambient environment. Solubility and reactivity of the Cr ions are affected by pH. Cr (III) is chemically basic, Cr (VI) is acidic. Cr (III) is the most stable oxidation state, is ubiquitous, forms inert hexacoordinate complexes; reacts with aqueous hydroxides to form insoluble chromium hydroxide. Cr VI is the second most stable state, occurs rarely in nature, because it is readily reduced to Cr (III) in the presence of organic matter, is quite soluble, existing in solution as a complex anion. However, in certain soils and natural waters, Cr (VI) can remain unchanged for protracted periods of time. In ambient air, Cr (III) is highly stable and Cr (VI) reacts over time to form Cr (III), therefore it is assumed that most chromium in ambient air occurs in the trivalent state (U.S. EPA, 1984). Hexavalent chromium (Cr VI) is released from cooling towers and chrome plating facilities (Towill, et al., 1978), chromic acid (CrO_3) is used in chromium plating of metal surfaces in metal finishing.

In U.S. waters, chromium concentration varies with the type of surrounding industrial sources and the type of underlying soils.

Chromium levels in soil vary with soil origin and degree of contamination from anthropogenic sources. Bioaccumulation of chromium in the soil-plant-animal system does not appear to be a significant exposure source, because chromium in food and plants is low and it does not accumulate in mammalian systems. Bioconcentration factor (BCF) is the ratio of the concentration of a chemical in aquatic species to the concentration in water in which they live.

Surface runoff, deposition from air, and release of municipal and industrial wastewaters are the sources of chromium in surface waters. Chromium may be transported in five forms in surface waters, as follows: 1) in solution and organic complexes, 2) adsorbed 3) precipitated and co-precipitated 4) in organic solids 5) in sediments (Towill, et al., 1978). The exact chemical forms of chromium in surface waters are not well defined. Although most of the soluble chromium in surface water may be present as Cr (VI) (Towill, et al., 1978), a small amount may be present as Cr (III) organic complexes (DeGroot and Allersma, 1978; Fukai, 1967).

In soil, most soil chromium is in mineral, adsorbed, or precipitated form. Chromium probably occurs as the insoluble Cr (III) oxide (Cr_2O_3 nH_2O) in soil, since the organic matter in soil is expected to reduce any soluble chromate to insoluble Cr_2O_3. Chromium in soil can be transported to the atmosphere by the way of aerosol formation (John, et al., 1973; Zoller, et al., 1974). Chromium is also transported from soil through runoff and leaching of water. Runoff could remove both chromium ions and bulk precipitates of chromium with final deposition on either a different land area or a water body. Also, flooding of soils and the subsequent anaerobic decomposition of plant matter may increase dissolution of Cr (III) oxides in the soil (Towill, et al., 1978).

Low concentrations of chromium enter the atmosphere as a result of industrial activities and soil-derived aerosols (Towill, et al., 1978). Chromium is removed from air through wet and dry deposition. The precipitated chromium from the air enters the surface water or soil. Chromium particles of aerodynamic equivalent diameter <20 um may remain airborne for long periods and may be transported to great distances from the source by wind currents and diffusion forces (Sehmel and Hodgson, 1976). Therefore, atmospheric conditions play an important role in determining the chromium concentration around emission sites.

Chromium can be rapidly absorbed in the body by oral and inhalation exposure, but only 5% is absorbed. Percutaneous absorption of chromium through unbroken skin is variable and dependent on valence as well as the specific salt. The relatively high amounts of Cr (III) that are required to cause death arise from the relative insolubility and poor intestinal absorption of most Cr (III) compounds. Unlike the trivalent compounds, those of Cr (VI) tend to cross biological membranes fairly easily, and are somewhat more readily absorbed through the gut or through the skin. The strong oxidizing power of Cr (VI) compounds explain much of their irritating and toxic properties. Both absorbed Cr (III) and Cr (VI) can be transported to a limited extent to the fetus in utero after exposure of the dams. After chromium is absorbed, it is bound by specific binding proteins for transport throughout the body, and it is transported to other organs of the body with greatest retention by the spleen, with liver and bone marrow or the lungs being a major deposition site.

Acute effects of Cr (VI) compounds include skin irritation and corrosion. Chromium may be absorbed by inhalation, cutaneously or by ingestion. Cr (III) compounds have a very low order of toxicity when administered orally; they have a low oral LD50

(Smyth, et al., 1969). Cr (VI) is more acutely toxic than Cr (III). A primary effect of acute Cr (VI) exposure is kidney failure. Kidney changes in lab animals after exposure to various chromium compounds included congestion, extravasation of red blood cells into intratubular spaces and tubular necrosis; thickening of small blood vessels, proliferation of endothelial cells, obliteration of Bowman's space and desquamation of convoluted tubular epithelium (Mathur, et al., 1977). After chromium exposure, the liver of lab animals were also affected; the livers showed congestion and dilation of central veins and sinusoids, necrosis of tissue, hemorrhage, the presence of multinucleated cells, bile duct proliferation, increased cellularity and proliferation of fibroblasts (Tandon, et al., 1978). There were also changes in the brain after acute Cr (III) exposure that included neuronal degeneration in cerebral cortex, marked chromatolysis and nuclear changes in the neurons, neuronophagia, neuroglial proliferation and meningeal congestion (Mathur, et al., 1977). Also Cr (VI) exposure resulted in brain effects, including congestion and perivascular inflitration by inflammatory cells, pyknotic changes in nuclei of cerebral cortex neurons, dissolution of Nissl's substance and neuronophagia and focal neuroglial proliferation throughout the cerebral cortex. The heart was also affected following chromium exposure, showing marked congestion and degeneration of muscle fibers. In summary, the kidney appears to be the main target for acute chromium toxicity, although hepatic, myocardial, brain effects and skin irritation were also observed.

Chromium has adversely affected fetal development and male reproduction in experimental animals (U.S. EPA, 1984). Hamsters administered chromium trioxide on day 8 of gestation had an increased incidence of cleft palates in the young when examined; however, malformations were associated with maternal toxicity (Gale and Bunch, 1979). This means that definitve conclusions between

fetal and maternal effects cannot be made with available data because the dose of the chemical was high and produced maternal weight loss. Other reproduction studies were similarly complicated and used an unnatural route of injection (i.e., IV or IP) so as to make relevance of human effects to environmental exposure uncertain.

Exposure of workers to Cr compounds resulted in nasal septum perforation (chromic acid and chromates: Vigliani and Zurlo, 1955; chromium plating plants: Bloomfield and Blum, 1928; chromate chemical plant: Mancuso, 1951). Cr compounds are responsible for a wide variety of other respiratory effects. German investigators (Alwen and Jonas, 1938; Fischer - Wasels, 1938; Koelsch, 1938; Lehmann 1932; Mancuso, 1951) reported that chronic inhalation of chromate dust caused chronic irritation of the respiratory tract and resulted in congestion and hyperemia, chronic rhinitis, congestion of the larynx, polyps of the upper respiratory tract, chronic inflammation of the lungs, emphysema, tracheitis, chronic bronchitis, chronic pharyngitis and bronchopneumonia. Researchers also reported enlargement of the hilar region of the lung, enlargement of the lymph nodes; increased peribronchial and perivascular lung markings and adhesions of the lung to the diaphragm. Another adverse effect reported for chromium compounds included chromium dermatitis (chrome leather tanning industry or chrome pigment industry). Allergic contact dermatitis may arise from either Cr (VI) or Cr (III) exposure, although Cr (VI) is responsible for most of the reported cases. Cr (VI) penetrates undamaged skin, and subsequently reduces to Cr (III) which combines with proteins or other skin components to form a whole skin allergin.

Using the IARC classifiction scheme, the level of evidence available for the combined animal and human data would place Cr

compounds into Group I, meaning there is decisive evidence for the carcinogenicity of these compounds in humans (IARC, 1980). In animals, there is some positive evidence that chromium, particularly some Cr (VI) compounds, is carcinogenic following subcutaneous injection or intrabronchial, intrapleural, intramuscular or intratracheal implantation (Steinhoff, et al., 1983). Calcium chromate is the only one that was found consistently to be carcinogenic in rats by several routes. (Hueper and Payne, 1962). Calcuim chromate, strontium chromate, zinc chromate, sodium dichromate, lead chromate, lead chromate oxide and sintered chromium trioxide have produced local sarcomas or lung tumors in rats at the site of application (Payne, 1960 a,b; Maltoni, 1974, 1976; Furst, et al., 1976; Hueper and Panye, 1959). Researchers stated that the relevance of studies using intramuscular implantation to human risk following inhalation or oral exposure to Cr compounds is not clear. Epidemiological studies of humans from chromate and chrome pigment plants have demonstrated an association of exposure to chromium compounds with respiratory cancer. Whether the association implicates hexavalent Cr (VI) alone, or trivalent Cr (III) as well, was not definitely addressed by these studies. However, the strength of a casual association between cancer and chromium is evidenced by the relatively high risk of cancer, the consistency of results by different investigators in different countries, dose-response relationships and the specificity of the tumor site (i.e., the lung). Genotoxic effects have been demonstrated primarily for Cr compounds containing the Cr (VI) species including effects such as mutagenic responses in bacterial strains (Venitt and Levy, 1974; Nishioka, 1975), morphologic changes in mammalian fetal cells (DiPaolo and Casto, 1979), cytogenic effects on mammalian bone marrow cells, increased gene conversion in yeast species (Bonatti, et al, 1976), transformation frequencies in mammalian cells (Fradkin, et al., 1975; Tsudo and Kato, 1977) and chromosomal

damage in cultures in human lymphocytes (Levis, et al., 1978; Gomez - Arroyo, 1981; Stella, et al., 1982; Littorin, et al., 1983).

For occupational exposure, ACGIH (1981) recommended a TLV for chromium metal, chromium (II), chromium (III) and chromium (soluble chromic and chromous salts) of 500 ug/m^3; ACGIH also recommended a TLV for chromium (VI) compounds (water soluble and insoluble) and chromite ore of 50 ug/m^3. NIOSH (1973 and 1975) recommended a TWA for noncarcinogenic chromium (VI)[1] of 25 ug/m^3; a TWA for chromic acid (as chromium trioxide) of 50 ug/m^3 and a carcinogenic chromium (VI) of 1 ug/m^3. Recommended standards for chromium in ambient waters have been set, as follows: 50 ug/l, of chromium (VI) in the drinking water (U.S. PHS, 1962; see U.S. EPA, 1984); 50 ug/l of total chromium in the domestic water supply (U.S. EPA, 1976, See U.S. EPA, 1984) 100 ug/l of total chromium in freshwater (aquatic life) (U.S. EPA, 1976, see U.S. EPA, 1984); 50 ug/l of chromium in community and noncommunity water systems (40 CFR 141.11).

Acceptable daily intake (ADI) for man for chromium (III) is 125 mg/day/man; the ADI for chromium (VI) is 0.175 mg/day/man. The calculated ambient water quality criteria for protection of human health (U.S. EPA, 1980) for chromium (III) is 59,000 ug/l; for chromium (VI) it is 83 ug/l. The U.S. EPA (1980) has also proposed several ambient water quality criteria for protection of aquatic life. For freshwater life, the calculated ambient water criteria to protect aquatic life for chromium (VI) is 0.29 ug/l (24-hr average) and 21 ug/l (maximum); for chromium (III), it is 44 ug/l (24-hr average: chronic value toxicity).

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[1] NIOSH listed "noncarcinogenic" chromium VI compounds as the monochromates and dichromates (bichromates) of: hydrogen, lithium, potassium, rubidium, cesuim, ammonium and chromic oxide (chromic acid anhydride).

In the air, no Federal or State (other than the State of Maine) ambient air chromium standards have been proposed (U.S. EPA, 1984). The lifetime cancer risk for air containing 1 ug/m^3 of Cr (VI) compounds is estimated to be 1.2 x 10^{-2}. This would place Cr (VI) in the first quartile of the 53 compounds evaluated by the EPA for relative potency. The carcinogen potency factor, q_1^* is 41 $(mg/kg/day)^{-1}$ (U.S. EPA, 1984).

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DDT

DDT exists as colorless crystals or white or slightly off-white powder. DDT has a molecular weight of 354.5 (USEPA, 1984), is in the pesticide class of chemical compounds and it may be odorless or have a slight aromatic odor. The half-life of DDT in is 56-110 days in lake water (Zoeteman, et al., 1980) and 3-15 years in soil (IARC, 1974). The mobility of DDT in soils is very slow (U.S. EPA, 1980). The leaching of DDT from soil is expected to be very slow, particularly in soil with high organic carbon content. Leaching of DDT from soil in groundwater at a frequency of 8-9% has been reported (Page, 1981).

DDT is readily absorbed from the intestinal tract. It may also be taken into the lung and readily absorbed if it occurs in the air in the form of an aerosol or dust. DDT is not, however, absorbed from the skin unless it is in solution. DDT acts on the CNS, but its exact mechanism has not been elucidated. Large doses of DDT also induce nausea and/or diarrhea in man. Chronically, DDT produces microscopic changes in the liver and kidneys in some experimental animals. This has not been demonstrated in man. DDT and certain of its degradation products are stored in fat; DDT stored in fat is eliminated very gradually only when further dosage is discontinued (Sax, 1984).

There have not been any reports of teratogenesis induced in experimental animals by DDT (Schmidt, 1973; Green, 1969; Ottoboni, 1969). However, DDT has produced other reproductive effects in animals, such as decreased fetal weight and fetal death (Schmidt, 1973) and decreased reproductive capacity (Keplingler, et al., 1968; McLachlan and Dixon, 1972; U.S. EPA, 1984).

DDT has been classified in Group B_2, Probable Human Carcinogen, by the U.S. EPA (1984). DDT has been associated with liver tumors in mice (Innes, et al., 1969; Tomatis, et al., 1972; Terracini, et al., 1973a, b) and rats (Rossi, et al., 1977), and lymphomas and pulmonary tumors (Tarjan and Kemeny, 1969) in mice.

There is inadequate evidence for both carcinogenicity and mutagenicity in humans,

The WHO (1971) recommended an ADI in food of 0.005 mg/kg/body weight for DDT. The U.S. EPA (1985) developed a human quantitative estimate of the carcinogenic potency, q_1^*, for orally administered DDT as 0.34 $(mg/kg/day)^{-1}$.

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Dichlorophonoxyacetic Acid (2,4-D)

Dichlorophonoxyacetic acid is a white crystalline powder, used as a herbicide, having a molecular weight of 221.04 and a molecular formula of $C_8H_6Cl_2O_3$. It is soluble in organic solvents, such as acetone, ethanol, isopropanol and benzene, toluene, xylene, ether, dioxane, heptane, carbon tetrachloride and carbon disulfide (Sax, 1984).

Acute exposure to 2,4-D by oral administration results in progressive symptoms of muscular incoordination, hindquarter paralysis, stupor, coma and death in animals (Hill and Carlisle, 1947). Oral LD_{50} values are generally in the range of 350-500 mg/kg body weight for rodents; significant differences in toxicity are not apparent between 2,4-D and its salts and esters.

Subchronic oral administration of 2,4-D in animals produced gastrointestinal disturbances and acute toxicity to hepatic tissues. Repeated subcutaneous exposure of 2,4-D caused pathologic and functional effects in the liver, kidneys, lungs, thyroid and nervous system in rats and mice (U.S. EPA, 1985).

Chronic oral administration of 2,4-D in the diet of dogs for two years did not produce adverse gross or histopathologic effects (Hansen, et al., 1971).

There are conflicting and unresolved reports of induction of lymphosarcoma in rats that were administered 2,4-D in the diet for two years (Reuber, 1979). But administration of 2,4-D or its esters by oral administration in the diet was not tumorigenic (Bionetics, 1968). Repeated dermal application of 2,4-D to mice produced skin papillomas only when treatment was preceded by application of the initiator 3-methyl cholanthrene (Archipov and Kozlova, 1974). Although increased tumor induction is suggested, the available data are insufficient to conclude that 2,4-D is carcinogenic. 2,4-D has been tested for mutagenicity in a variety of assays (e.g., plant, bacteria, yeast, fruit flies, in vitro and in vivo mammalian systems), but there is a preponderance of negative and inconsistent results in the animal assays. These

varied results may be attributed to differences in pH (U.S. EPA, 1985).

Teratogenicity testing with several species of rodents indicates that 2,4-D and several of its esters and other derivatives are embryotoxic (i.e. produce fetal malformations) or nonteratogenic. Malformations generally consisted of cleft palate and other skeletal effects (Bage, et al., 1973).

Reports of humans who acutely ingested 2,4-D solutions or were exposed to 2,4-D formulations via industrial or agricultural exposure indicate that symptoms of gastritis, vomiting, loss of consciousness, neurological signs (e.g., reflex disorders) and muscular paralysis precede death. Autopsies of fatal poisoning cases have shown widespread pathologic effects (e.g., congestion and hyperemia of most organs, and hepatic necrosis). Epidemiologic studies have associated excess incidences of cancer in humans with mild exposure to chlorophenoxy herbicides (that contained 2,3,7,8-TCDD-contaminated 2,4,5-T), but the carcinogenic effects have not been attributed to 2,4-D alone (U.S. EPA, 1985).

Dichlorophonoxyacetic acid has a threshold limit value (TLV) in air of 10 mg/m^3 (ACGIH, 1980). A lifetime adjusted acceptable daily intake (AADI) of 0.35 mg/1 is recommended by the EPA based on a subchronic rate NOAEL (i.e., no observed adverse effect level). EPA has reported a maximum safe level of 2,4-D (from all sources) of 16 ug/kg/day (U.S. EPA, 1976). The National Academy of Sciences has suggested an acceptable level in drinking water of 0.09 mg/1 (0.09 ppm) for 2,4-D in drinking water, assuming that 20% of exposure is attributable to drinking water (NAS, 1977). The maximum contaminant level (MCL) for 2,4-D is 0.100 mg/1. This standard was developed under the Safe Drinking Water Act. The MCL represents allowable lifetime exposure limits.

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Dieldrin (=HEDD)
Aldrin (=HHDN)

Dieldrin and Aldrin are white, odorless crystals. Together with heptachlor and chlordane, etc., they form the group of cyclodiene insecticides. They are a subgroup of the chlorinated cyclic hydrocarbon insecticides to which DDT and toxaphene belong, called organochlorine insecticides. They are whitish to light-brown crystals. Aldrin has an empirical formula of $C_{12}H_8Cl_6$ and a molecular weight of 365. Dieldrin has an empirical formula of $C_{12}H_8Cl_6O$ and a molecular weight of 381. Aldrin is a broad spectrum insecticide primarily used for control of a wide range of soil pests, grasshoppers and certain cotton insects. Dieldrin is a broadspectrum insecticide to control certain insects attacking principal field-, vegetable- and fruit crops. It is also used against public health pests including disease vectors and in locust and termite control. Aldrin is an intermediate in the production of Dieldrin. Aldrin is readily epoxidized to dieldrin in the liver. Vapor pressures of aldrin and dieldrin are very low and are in the range of 10^{-6} to 10^{-7} mm Hg (Jager, K., 1970).

After ingestion, aldrin and dieldrin are readily and almost completely absorbed from the gastrointestinal tract and transported from the liver to the body. The body is exposed to dieldrin and aldrin by inhalation, skin absorption and ingestion. Dieldrin and aldrin are teratogenic and affect reproduction. Single doses of dieldrin and aldrin administered orally to pregnant hamsters during fetal organ development caused a high incidence of fetal deaths, congenital anomalies and growth retardation (Ottolenghi, et al, 1974).

Chronic oral administration of aldrin or dieldrin for 2 years to rats resulted in a possible slight increase in the liver weight/body weight ratio and changes in the liver cells (Treon, et al, 1955; Fitzhugh, et al., 1964). Lab animals chronically fed dieldrin in the diet were reported to have increased irritability and an occasional convulsion, increased liver weight/body weight

ratio and increased liver weight (Walker, et al, 1968). Increased liver weights and minor liver cell changes were observed in dogs chronically fed aldrin (Treon, et al, 1955); only increased liver weights were reported in dogs chronically fed dieldrin (Wright, et al., 1968). In long-term feeding studies with lab animals, the first measurable effect in all species is an effect on the liver to stimulate the drug-metabolizing enzymes with an increase in activity of various hepatic and serum enzymes produced by the microsomal system. The effect is dose-related, reversible and is a sign of exposure and adaptation (Jager, K., 1970).

Dieldrin causes liver cancer in mice. The hepatocarcinogenicity of dieldrin in mice has been confirmed in several experiments; in some cases, the liver cell tumors metastasized (IARC, 1974).

In workers/chronically exposed to aldrin and dieldrin in a manufacturing plant, a slow accumulation of the insecticide in the blood and adipose tissue occurs if the daily intake exceeds the daily excretion. If toxic levels are reached, then the following signs may appear: headaches, lassitude, fatigue, loss of appetite, weight loss, insomnia, frequent nightmares, inability to concentrate, loss of memory, hyperirritability, hyperexcitability, paresthesia, myoclonias, black-outs, and prodomi epileptiform convulsions. There was also a few adaptive responses of liver cells found in workers in an aldrin and dieldrin production plant (Jager, K., 1970). Occupational exposure to aldrin and dieldrin for periods up to 15 years, at times, even at toxic doses, did not have any persistent adverse effect on the health of these workers, as far as could be demonstrated in all parameters used. In cases of intoxication, all signs and symptoms of aldrin or dieldrin exposure were fully reversible within weeks.

The threshold limit value (TLV) in air of aldrin and dieldrin is 0.25 mg/m^3. TLV refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed, day after day, without adverse

effect. However, because of wide variation in individual susceptibility, exposure of an occasional individual at or even below the threshold limit may not prevent discomfort, aggravation of a pre-existing condition, or occupational illness (Threshold Limit Values of Air-borne Contaminants, 1969). The safety threshold levels of dieldrin concentration in the blood of humans that has not ever produced any signs or symptoms of intoxication is 0.20 ug/ml (Jager, K., 1970).

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Phthalate Esters:

Diethylphthalate (=DEP)

Di-n-butylphthalate (=DBP)

Di-n-octylphthalate (=DOP)

Di(2-ethylhexyl) phthalate [DEHP, Bis-(2-ethylhexyl) phthalate]

Di(2-ethylhexyl) phthalate (DEHP) has a molecular formula of $C_{24}H_{38}O_4$ and a molecular weight of 390. Diethylphthalate (DEP) has a molecular formula of $C_{12}H_{14}O_4$ and a molecular weight of 222. Di-n-butylphthalate (DBP) has a molecular formula of $C_{16}H_{22}O_4$ and a molecular weight of 278. Di-n-octylphthalate (DOP) has a molecular formula of $C_{24}H_{38}O_4$ and a molecular weight of 390. DEHP is the main plasticizer for polyvinyl chloride. The other phthalate esters are used as plasticizers depending on the use of the product. Plasticizers allow different polymers to assume different shapes.

DEHP and most phthalate esters are absorbed readily in most laboratory species; they can gain access to the systemic circulation and tissues by inhalation and dermal absorption. DEHP, DBP and other diesters of o-phthalic acid, have a low order or acute toxicity when given orally and dermally in laboratory animals (Woodward, K., 1988).

DEHP produced adverse effects on the reproductive performance of zebra fish and guppies at high levels (Mayer, F. and Sanders, H. O., 1973). Long-term exposure to DEHP had little effect on rainbow trout, brook trout or fathead minnows (Mayer, F., et al., 1977). DBP affects fish embryo survival, hatchability and larval survival in fathead minnow (McCarthy, J. and Whitmore, D., 1985). There is a suggestion in the literature that DBP may cause thinning of the bird eggshell (Peakall, D., 1974). Phthalates readily accumulate in fish, but the body levels rapidly decline when animals are removed from the environment containing esters (Mayer, F., 1976).

Subchronic studies of lab animals using high doses of DEHP showed a reduction in body weight gain and polycystic kidneys.

(Nikonorov, et al., 1973; Gray, et al., 1977; Oishi, S. and Hiraga, K., 1975; Smyth, et al., 1951; Ota, et al., 1973 and 1974). The reduced body weight may be due to reduced food intake either because of the introduction of huge amounts of the esters into the stomachs of the animals or because of reduced palatability of the food (Woodward, Kevin, 1988). Subchronic oral administration of DBP produced reductions in the body weight gain, splenomegaly, increased relative kidney weights and cystic kidneys (Yamada, A., 1974; Nikonorov, et al., 1973; Radeva, M. and Dinoyeva, S. 1966; Ota, et al., 1973 and 1974). Inhalation exposure to DBP produced reduced body weights and increased relative organ weights (Kawano, M., 1980).

Teratogenic and reproductive effects have been reported for phthalate esters. In lab animals, DEHP administered in the diet, resulted in embryolethality (death of the fetus) and a 30% incidence of anomalies in fetuses that included open-eye, encephalocoele, spina bifida, tail malformation and imperforate anus (Hamano, Y., et al., 1977). In another study, DEHP was administered in the diet to dams and resulted in maternal toxicity, fetotoxicity and teratogenic effects; the major abnormalities were exencephaly, open-eye, tail constructions; aortic-arch malformations, aortic anomalies, pulmonary vessel anomalies, fused or branched ribs, fused or misaligned centra and misaligned sternebrae (Wolkowski-Tyl, et al., 1983). In another study, oral DEHP given to pregnant lab animals resulted in a dose-dependent reduction in maternal body weight, a dose-dependent increase in resorptions (fetal death) and a significant incidence of fetal exencephaly, spina bifida, tail malformations, gastroschisis and club foot (Shiota, K., et al., 1980; Shiota, K. and Nishimura, H., 1982).

DEP produced reproductive and teratogenic effects in lab animals. DBP increased preimplantation losses (Aldyreva, et al., 1975). DBP fed orally in lab animals also produced external anomalies, including open-eye, encephalocoele, cleft palate and

spina bifida (Hamano, et al., 1977). There are other reproductive effects reported for phthalate esters. DEHP produced seminiferous tubular atrophy when given orally to male rats (Oishi, S., 1984). DEHP and DBP esters produced signs of testicular atrophy in lab animals according to many reports (Sjoberg, P., et al., 1982; Gray, et al., 1977; Lake, B., et al., 1976; Foster, P., et al., 1982). There is a loss of testicular, accessory organ weight, and advanced germinal cells; only Sertoli cells and primary spermatocytes remain in tubules (Sjoberg, P., et al., 1982). However, DEP and DOP failed to produce testicular atrophy in rats (Sjoberg, P., et al., 1982; Gray, et al., 1977; NTP, 1982; RoLoff, et al., 1983; Mann, et al., 1985; Vallee, B., 1983).

DEHP administered orally in the diet to rats for 2 years resulted in an increased incidence of neoplastic nodules in the liver and hepatocellular carcinoma (NTP, 1982; Kluwe, et al., 1982; Huff, et al., 1982). The incidences of both lesions and combined incidences show clear, dose-related trends. In mice, DEHP administered in the diet orally also resulted in hepatocellular carcinoma and adenoma. Pulmonary metatases were frequently evident in the lungs (Northrup, et al., 1982). Results of the work indicate DEHP is hepatocarcinogenic in rodents when given at high dietary levels over 2 years; it is also a promoter of hepatocarcinogenesis, but not of skin carcinogenesis (Ward, et al., 1983). In genotoxicity tests designed to demonstrate mutagenic potential or to serve as a prescreen for carcinogenicity, DEHP and DBP have given negative results (Woodward, K., 1988). DEHP was found to be negative for point mutations using the Ames test and strains of Salmonella typhimurium (Rabenold, C. and Brusick, D., 1982; Zeiger, E., et al., 1982; Kirby, P., et al., 1983 and Simmon, et al., 1977). DEHP was also found to be negative in results using E.coli reversion assay (Yoshikawa, et al., 1983 and Tomita, et al., 1982). Negative results were obtained for in vitro tests using DEHP for the ability to induce point mutations in mammalian cells (Kirby, P., et al., 1983) and for cell transformation assays

(Rundell, J. and Brusick, D., 1982 and Rundell, J. and Brusick, D., 1982). DBP gave negative results for point mutations in S. typhimurium in histidine reversion assays (Zeiger, E., et al., 1982, and Kozumbo, W., 1982). DOP gave negative results in the Ames test for point mutations (Zeiger, E., et al., 1982).

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Manganese (Mn):

Manganese is a reddish-grey or silvery, brittle metal having an atomic weight of 54.9 (SAX, I., 1984) and belonging to the first transition series of the periodic table.

Mn can exist in 11 oxidation states from -3 to +7; the +4 valence is the predominant natural form manganese dioxide, MnO_2. Mn is widely distributed in the earth's crust and is the 12th most abundant element. Uses of manganese include metallurgical purposes, dry cell batteries, glass, inks, ceramics, paints, welding rods, chemical oxidizing agents, rubber and wood preservatives (Seiler, H. & H. Sigel, 1988).

The principal sources of Mn in the atmosphere are natural processes including continental dust, volcanic gas and dust and forest fires. The main anthropogenic sources of Mn are industrial emissions and combustion of fossil fuels (Lantzy and MacKenzie, 1979). Mn aerosol may be removed from the air environment through dry fallout or wet precipitation. It has been estimated that the atmospheric residence time for Mn due to such physical removal processes is about 7 days (Cupitt, 1980).

The fate of Mn in aquatic systems may be determined by its ability to undergo chemical and microbiological reactions. In most natural aquatic systems, Mn is expected to be present predominantly in the suspended particulates and sediments as MnO_2 and/or Mn_3O_4. A small amount of Mn may remain as soluble Mn^{+2}. Mn may persist in aquatic systems for a long period. By analogy with aquatic iron (U.S. EPA, 1981), the residence time of aquatic Mn may be a few hundred years.

The bioconcentration factor for Mn in a species of edible fish (Striped Bass) has been reported to be <10 (U.S. EPA, 1982). Significant bioaccumulation of Mn may not occur with organisms of higher tropic level.

Both chemical and microbiological interactions may cause speciation of Mn in soils. Both soil pH and oxidation-reduction

potential of soil may influence the speciation process. It has been suggested that in acid water-logged soils, Mn passes freely into solution and may leach into groundwater. Mn can be readily leached from waste burial sites and from other natural soils into groundwater (U.S. EPA, 1982).

High levels of Mn in the diet in subchronic animal studies have been associated with depressed reproductive performance (Gray and Laskey, 1980). Excess Mn in the diet of animals depressed hemoglobin formation (Matrone, et al., 1959), interfered with hemoglobin regeneration (Hartman, et al., 1955) and was associated with decreased blood pressure and elevated serotonin blood levels (Kimura, et al., 1978). When excess Mn was put in the drinking water of lab animals, there were ultrastructural changes in the liver. In monkey inhalation studies, animals exposed to Mn for 5 months had mild tremors of fingers, decreased pinch force and reduced dexterity of upper limbs that were considered to be evidence of neurological damage analogous to humans suffering from chronic Mn toxicity (Nishiyama, et al., 1975).

Chronic oral exposure of humans to Mn in the drinking water resulted in extra-pyramidal dysfunction symptoms such as lethargy, increased muscle tone and spasms, tremors and mental disturbances in 16 people. Elderly people seemed to be most severely affected and children were least affected. Autopsy of one case showed atrophy of the globus palladum, disappearance of its neurons, moderate congestion of the brain, spinal cord and meninges and meningeal edema (Kawamura, et al., 1941). Kawamura reported the case of water consumption from wells contaminated by Mn from dry cell batteries buried nearby. After the outbreak of chronic Mn intoxication, water from the wells was tested and found to contain 14.3 mg/l of Mn. Over a period of 6 weeks, the concentration was reported to decrease to 4.2 mg/l.

Chronic inhalation exposure of humans to Mn can be described in 3 phases (Cotzias, 1962). The first phase of manganism begins

insidiously with anorexia, asthenia, abnormal psychotic behavior and occasional criminal acts. Severe somnolence followed by insomnia are noted. Headache and leukocytopenia occur. The second phase initiates the onset of extrapyramidal disease, clumsy articulation often resulting in muteness. A mask-like face and general clumsiness and lack of skilled movements are characteristic. The third phase is characterized by severe rigidity, and the limbs manifest a "cogwheel" phenomenon. Tremors occur that become exacerbated by emotion, stress, fatigue and trauma. Indifference, interrupted by laughing or crying spells; autonomic dysfunction, manifested by excessive salivation or sweating, occur. Levels of Mn as low as 0.30 mg/m^3 (ferromanganese plant, Saric, et al., 1977), 0.44 mg/m^3 (welding fumes, Chandra, et al., 1981) and 0.5 mg/m^3 (manganese mine, Schuler, et al., 1957) have been associated with neurological evidence of manganism.

Oral exposure of humans to Mn has been associated with impotency (Penalver, 1955; Rodier, 1955; Mena, et al., 1967). In animals fed dietary Mn chronically, testicular and sex accessory organ weights were significantly decreased compared to control mice.

Using the criteria for evaluating the overall weight of evidence for carcinogenicity in humans proposed by the Carcinogen Assessment Group of the U.S. EPA (Federal Register, 1984), Mn is best designated a Group D, not classified, substance (U.S. EPA, 1984). Furst (1978); Stoner, et al., (1976); Di Paolo (1964) and Sunderman (1974, 1976) administered manganese compounds to lab animals and were unable to significantly increase the incidence of neoplasia.

For occupational exposure the ACGIH (1980) set the ceiling limit for manganese dust at 5 mg/m^3, based on reports of no cases of manganism reported in 25 ore-handlers exposed to MnO_2 dust concentrations of 1 - 5 mg/m^3.

No carcinogen potency factor, q^*, was derived for oral and inhalation exposure for Mn since there was no significant increase in the incidence of cancer associated with Mn administration (U.S. EPA, 1984).

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Methylene Chloride (Methane dichloride, dichloromethane)

Methylene chloride is a colorless, volatile liquid having a molecular formula of CH_2Cl_2 and a molecular weight of 84.93 (Sax, 1984). It is a member of the halogenated aliphatic hydrocarbon (purgeable halocarbon) chemical class of compounds. Evaporation is expected to be the predominant loss mechanism from the soil surface. In subsurface soil, biodegradation of a chlorinated aliphatic hydrocarbon such as methylene chloride may be slow (Wilson, et al, 1983). Therefore, in subsurface soil, the nondegraded methylene chloride is expected to leach into groundwater.

Subchronic inhalation studies in lab animals exposed to methylene chloride demonstrated liver and kidney damage positive staining of the liver for fat, elevated carboxyhemoglobin levels, narcosis, pronounced lethargy, reduced food consumption and weight, and high rates of mortality.

In humans, chronic inhalation of methylene chloride resulted in somnolence, lassitude, anorexia and mild lightheadedness, disturbed CNS function and depression (NAS, 1978). In chronic inhalation exposure of animals to methylene chloride, Burek, et al (1980, 1984) and Dow Chemical Company (1980) found rats suffered from a disease believed to be sialodacryoadenitis (a transient viral involvement of the salivary glands), increased liver weights, histopathologic alterations of the liver, an increased incidence of hepatocellular vacuolization indicative of fatty degeneration, multinucleated hepatocytes (a spontaneous geriatric change in female rats), a significant increase in the number of foci of altered hepatocytes, hepatocellular necrosis and coagulation necrosis.

Methylene chloride is classified as a Group B_2, probable human carcinogen, by the U.S. EPA (1984, 1985). Pertinent data regarding carcinogenicity in humans associated with methylene chloride could not be located in the available literature (U.S. EPA, 1984).

NTP (1985) performed a cancer bioassay and found the inhalation of methylene chloride vapors was associated with an increased incidence of benign mammary gland neoplasms and primarily fibroadenomas in rats, a significant increase in hepatocellular neoplastic nodules and hepatocellular carcinomas in female rats, a statistically significant increase of mononuclear cell leukemias in female rats, a significant increase in mesotheliomas (primarily in the tunica vaginalis) in male rats, and a significant increase in adrenal pheochromocytomas and interstititial cell tumors in male rats and combined incidence of pituitary gland adenomas and carcinomas in male and female rats (U.S. EPA, 1985, 1984). In mice, the NTP (1985) study also demonstrated a significant increase in alveolar/bronchiolar adenoma and/or carcinoma in both sexes of mice and a significant increase in combined incidence of hepatocellular adenoma and hepatocellular carcinoma (U.S. EPA, 1985). NTP (1985) concluded that there was some evidence supporting carcinogenicity for methylene chloride for male rats as shown by increased incidence of benign neoplasms of the mammary gland; there was clear evidence of the carcinogenicity of methylene chloride for female rats as shown by an increased incidence of benign neoplasms of the mammary gland; there was clear evidence for the carcinogenicity in male and female mice as shown by an increased incidence of lung and liver tumors. Methylene chloride has been shown to be mutagenic to Salmonella typhimurium (Simmon, et al, 1977). Thilager and Kumaroo (1983) observed extensive chromosomal aberrations in cultured Chinese hamster ovarian cells exposed to methylene chloride.

The ACGIH (1981) set the TLV at 100 ppm (360 mg/m^3) for methylene chloride. The U.S. EPA (1980) set the ambient water quality criterion at 6 ug/l on the basis of carcinogenicity to methylene chloride. Acceptable intake subchronic (AIS) and acceptable intake chronic (AIC) have not been calculated for methylene chloride since this compound was shown to be carcinogenic in lab animals. The carcinogenic potency factor (q_1^*, U.S. EPA, 1985) for methylene chloride was calculated from data for the NTP (1985) study, as follows:

Human Carcinogenic Potency Factor, q_1^*:

Inhalation:	Male Rat	0.793×10^{-3}	$(mg/kg/day)^{-1}$
	Female Rat	2.43×10^{-3}	$(mg/kg/day)^{-1}$
	Male Mouse	7.05×10^{-3}	$(mg/kg/day)^{-1}$
	Female Mouse	14.3×10^{-3}	$(mg/kg/day)^{-1}$
Oral:	Male Mice	2.6×10^{-3}	$(mg/kg/day)^{-1}$

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Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic Aromatic hydrocarbons (PAHs) are a class of compounds that are formed during the incomplete combustion or pyrolysis of organic materials containing carbon and hydrogen. Several hundred different PAH have been identified from combustion and pyrolysis sources (Grimmer, 1983). In this discussion, only a few PAHs compounds (containing 2-6 aromatic rings) that occur most frequently in the environment (Grimmer, 1983) and also appear on the U.S. EPA's list of priority pollutants will be considered.

The majority of naphthalene, phenanthrene, anthracene, fluoranthene and pyrene should exist in the vapor phase in the atmosphere, according to the theoretical predictions of Cupitt (1980) and the experimental work of Yamasaki, et al (1982). On the other hand, benz(a)anthracene, chrysene, benzo(a)pyrene (BaP) and benzo(ghi)perlene should exist predominantly in the particulate sorbed phase in the atmosphere. The removal of PAHs from the atmosphere can occur through photochemical reactions, chemical reactions (principally with OH radicals, ozone and NO_x) and physical removal mechanisms (wet and dry deposition). The PAHs that exist predominantly in the vapor phase in the atmosphere (e.g., naphthalene, phenanthrene, anthracene, fluoranthene and pyrene) are likely to be removed primarily through direct or indirect photochemical reactions (Atkinson, et al, 1984; NAS, 1983; Mabey, et al, 1981). The primary removal mechanism for benz(a)anthracene and BaP from the atmosphere is likely to be ozonolysis reactions (NAS, 1983). The reactivities of the particulate sorbed portions of the PAHs are strongly dependent on the materials on which these compounds are sorbed (Korfmacher, et al, 1980). This increased stability of particulate-sorbed PAHs may permit these compounds to participate in long distance transport (U.S. EPA, 1984).

The three likely mechanisms that may be responsible for the removal of PAHs from aquatic media are volatilization, photochemical reactions and microbial degradation. With the exception of naphthalene and other PAHs that have relatively high vapor pressures, volatilization is not likely to be a significant removal mechanism. In the case of naphthalene, both volatilization and adsorption may be quite competitive, with the dominant process being dictated by the aquatic conditions. High stream and wind velocities could enhance volatilization, while high organic carbon content could facilitate sedimentation and the subsequent microbial degradation of particle-sorbed naphthalene (U.S. EPA, 1984).

The predominant mechanism that is likely to dictate the fate of most PAHs in aquatic media is sorption onto particulate matter and subsequent sedimentation and microbial degradation (U.S. EPA, 1984).

The predominant mechanism for the removal of PAHs from soils is likely to be microbial degradation. Considering the soil sorption coefficient (Kenaga & Goring, 1980) and water solubilities, these compounds are not expected to have high mobility in soils. Therefore, significant leaching of these compounds into groundwater is not expected, particularly from soils with higher organic carbon content (U.S. EPA, 1984).

Subchronic oral administration of acenaphthylene (or acenaphthene) to rats resulted in considerable body weight loss, unspecified changes in the peripheral blood pattern, changes in renal function, and increased serum aminotransferase activities, morphologic kidney and liver damage, changes consistent with mild bronchitis and localized inflammation of the peribronchial tissue (Knoblock, et al, 1969). Subchronic inhalation of acenaphthylene resulted in chronic nonspecific pneumonia in lab rats (Reshetyuk, et al, 1970); the abstract did not, however, provide details

concerning controls or experimental protocol (U.S. EPA, 1980c).

Of 7 pregnant BaP-treated rats, only 1 dam carried viable fetuses to term, delivering 4 pups on the 23rd day of pregnancy. Two of the 4 pups were stillborn, one of which was grossly malformed; another pup died of starvation 3 days after birth, since the dam did not show any signs of lactation. At autopsy, 4 dead fetuses were found in the right uterine horn of a second dam (Rigdon & Rennels, 1964). In another teratogenicity and reproduction study in mice, Rigdon & Neal (1965) administered diets containing BaP and found no apparent reproductive teratogenic or fetotoxic effects in lab animals. Mackenzie & Angevine (1981) observed a specific reduction of gonadal weight, reduced fertility and reproductive capacity among offspring and almost complete sterility of offspring in the high dose group only of mice fed BaP orally during pregnancy.

U.S. EPA (1980c) described synergistic and antagonistic interactions among different PAHs and between PAHs and non-PAH chemicals. This is based on the metabolism of PAHs by the microsomal mixed function oxidase enzyme system and competition between the PAHs and any other potential substrate. Metabolism of PAHs by the microsomal mixed function oxidase enzyme system yields several types of reactive and potentially carcinogenic intermediates. Chemicals that induce or inhibit this enzyme system alter the patterns of PAH metabolism and, hence, alter their toxic and carcinogenic properties (U.S. EPA, 1984).

IARC (1983) has evaluated selected PAHs based on the overall weight of evidence of carcinogenicity to humans. These classifications range from Group 2A (BaP) and 2B meaning that the compound is probably carcinogenic in humans to Group 3 which indicates that there is only limited animal evidence or a paucity of evidence such that the data base is inadequate to assess the

human carcinogenic potential. Some of these classifications are based on routes of exposure other than oral and inhalation. As a class, PAH-containing soots, tars and oils are most appropriately classified as Group 1 (IARC, 1983).

The U.S. EPA (1984) has classified these chemicals in Group A, a human carcinogen (sufficient evidence from epidemiologic studies exists to support a causal association between exposure and cancer).

IARC has judged the following specific PAHs to be probably carcinogenic in humans, because there is sufficient animal evidence and/or limited human evidence. The U.S. EPA (1984) has placed the following chemicals in Group B_1 (Probable Human Carcinogens: Limited evidence of carcinogenicity in humans from epidemiological studies) or Group B_2 (Probable Human Carcinogens: Sufficient evidence of carcinogenicity in animals, inadequate evidence of carcinogenicity in humans), depending on the quality of the evidence:

1. benz(a)anthracene
2. benzo(b)fluoranthene
3. benzo(j)fluoranthene
4. benzo(k)fluoranthene
5. benzo(a)pyrene
6. dibenz(a,h)acridine
7. dibenz(a,j)acridine
8. dibenz(a,h)anthracene
9. 7H-dibenzo(c,g)carbazole
10. dibenzo(a,e)pyrene
11. dibenzo(a,h)pyrene
12. dibenzo(a,i)pyrene
13. dibenzo(a,l)pyrene
14. indeno(1,2,3-cd)pyrene

Also, the following compounds have limited animal evidence for carcinogenicity; however, the evidence according to IARC is inadequate for making a definitive statement about the human carcinogenic potential. The following compounds have been placed in Group C, Possible Human Carcinogen, category:

1. anthanthrene
2. benz(c)acridine
3. carbazole
4. chrysene
5. cyclopenta(c,d)pyrene
6. dibenz(a,c)anthracene
7. dibenz(a,j)anthracene
8. dibenzo(a,e)fluoranthene
9. 2- and 3-methylfluoranthenes

The carcinogenicity of PAHs have been extensively tested by application to the skin of mice, and have been the subject of only limited investigation by other routes of administration. U.S. EPA (1983a, b, c, d and e) summarized the studies discussed below. More complete reviews of the carcinogenicity bioassays of PAHs are presented by IARC (1973, 1983), U.S. EPA (1980a, b, c, 1981) and Santodonato, et al (1981).

In human studies, Cottini & Mazzone (1939) applied BaP in benzene to small areas of exposed and unexposed skin of patients for 4 months. Regressive verrucae (warts) developed in all the patients within 4 months. Although these changes were thought to represent early stages of neoplastic proliferation. Similar cases of epidermal changes were reported by Rhoads, et al (1954) and Klar (1938) in men accidentally exposed to BaP. Numerous epidemiologic studies of human populations (primarily worker groups) have shown a clear association between exposure to PAH-containing mixtures

(soots, tars, oils, etc.) and increased cancer risk (Santodonato, et al, 1981; IARC, 1973, 1983; U.S. EPA, 1981).

In animals, the carcinogenic properties of certain PAH compounds have been studied in animals for more than 50 yrs. The predominance of testing has been done with oral, inhalation exposures, mouse skin assays, implantations and subcutaneous injections. Benzo(a)pyrene administered orally in the diet to mice resulted in increased incidence of papillomas and carcinomas (stomach tumors: Neal and Rigdon, 1967) as well as, lung adenoma and leukemia (Rigdon and Neal, 1966, 1969). Incidence of lung adenomas and liver hepatomas was elevated in animals given BaP by gavage (Klein, 1963).

In animals, dibenz(a,h)anthracene administered orally in the water to lab animals showed carcinogenic effets, that is, animals developed pulmonary adenomatosis, carcinoma of the lung and hemangioendotheliomas and mammary carcinomas (Snell and Stewart, 1962, 1963).

Inhalation of BaP in hamsters resulted in tumors of the nasal cavity, larynx, trachea and pharynx, tumors of the respiratory tract and upper digestive tract (Thyssen, et al., 1981). Intratracheal administration of BaP resulted in an increased incidence of respiratory tract neoplasms in hamsters (Ketkar, et al., 1978; Feron and Kruysse, 1978). There was a dose-response relationship for some of the dose groups, followed by a latency period. The respiratory neoplasms included respiratory tract carcinoma, adenoma and papilloma (Ketkar, et al., 1978).

PAH-containing mixtures have been found to show carcinogenic activity in mouse skin assays including: crude coal tar, blast furnance tar, soot extracts, oil shale extracts, cigarette smoke condensates, petroleum pitch and automobile exhaust (CRC, 1983;

IARC, 1973). PAH-containing mixtures have been linked to increased incidence of cancer in exposed humans. Exposure of chimney sweeps to soot and coal tar has been associated with increased scrotal cancer (U.S. EPA, 1984). Increased skin cancer has been reported in workers in the coal tar and pitch industry (IARC, 1973). Coal tars have been shown to be carcinogenic in animals following skin painting or subcutaneous injection (IARC, 1973).

Short-term genotoxicity tests have been performed with PAHs (see Table 1). Many of the PAHs that have shown positive results in one or more *in vitro* genotoxicity screening tests have given negative results in animal bioassays (Santodonato, et al., 1981; IARC, 1973, 1983; U.S. EPA, 1981).

Exposure criteria and TLVs have been developed for PAHs as a class, as well as for several individual PAHs. The U.S. OSHA set an 8-hour TWA concentration limit of 0.2 mg/m^3 for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, BaP, phenanthrene, acridine, chrysene, pyrene) (Code of Federal Regulations, 1981). NIOSH (1977) recommends a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances at 0.1 mg/m^3 of the cyclohexane-extractable fraction of the sample determined as a 10-hour TWA. The U.S. EPA (1980c) recommended a concentration limit of 28 ug/l for the sum of all carcinogenic PAHs in ambient water. Environmental quality criteria specify concentration limits intended to protect humans against adverse health effects. This value is based on a mathematical extrapolation of the results from studies with mice treated orally with BaP, and acknowledges the conservative assumption that all carcinogenic PAHs are equal in potency to BaP. Daily consumption of water containing 28 ug/l of carcinogenic PAHs over an entire lifetime is estimated, on the basis of the animal bioassay data, to keep the lifetime risk of cancer development below one chance in 100,000. The U.S. EPA has not recommended an ambient water

quality criterion for noncarcinogenic PAHs as a class. The U.S. EPA (1980b) has recommended, however, an ambient water quality criterion for fluoranthene of 42 ug/l, which is based on chronic toxicity tests in mice that received fluoranthene by repeated application to the skin. An ambient water quality criterion of 0.02 ug/l for acenaphthene has been recommended by the U.S. EPA (1980a) on the basis of organoleptic properties.

Acceptable intake subchronic (AIS) and acceptable intake chronic (AIC) have not been calculated for PAHs. Carcinogenic potency factors (q_1*) have been calculated for PAHs based on the estimate of the unit risk from a single PAH compound, BaP (U.S. EPA, 1984). Carcinogenic potency factors, q_1*, have been calculated for PAH, as follows:

	Human q_1*
Oral:	11.53 $(mg/kg/day)^{-1}$
Inhalation:	6.11 $(mg/kg/day)^{-1}$

TABLE 1
GENOTOXICITY OF SELECTED PAH[a]

PAH	Positive Result in at Least One Genotoxicity Assay
Anthanthrene	+
Anthracene	+[b]
Benz(c)acridine	+
Benz(a)anthracene	+[b]
Benzo(b)fluoranthene	+[b]
Benzo(b)fluorene	+
Benzo(g,h,i)perylene	+
Benzo(a)pyrene	+[b]
Benzo(e)pyrene	+
Carbazole	-
Chrysene	+[b]
Coronene	+
Cyclopenta(c,d)pyrene	+
Dibenz(a,h)acridine	+
Dibenz(a,j)acridine	+
Dibenz(a,c)anthracene	+
Dibenz(a,h)anthracene	+[b]
7H-Dibenzo(c,g)carbazole	-
Dibenz(a,h)pyrene	+
Dibenzo(a,i)pyrene	+
1,4-Dimethylphenanthrene	+
Fluoranthene	+
Fluorene	+[c]
1-Methylphenanthrene	+
Perylene	+
Phenanthrene	+
Pyrene	+
Triphenylene	+

[a]Source: Adapted from IARC, 1983.

[b]Positive for Carcinogenicity in at Least One Animal Bioassay.

[c]Negative for Carcinogenicity in rats Fed Fluorene in the Diet.

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2,3,7,8-Tetrachlorodibenzo-P-Dioxin (2,3,7,8-TCDD)

2,3,7,8-TCDD exists as colorless needles with a molecular weight of 321.96 and a molecular formula of $C_{12}H_4Cl_4O_2$ (Sax, 1984). The half-life of 2,3,7,8-TCDD in water is 1-2 years and 10-12 years in soil (U.S. EPA, 1984c). Based on available data (U.S. EPA, 1984a), the possibility of vertical movement of 2,3,7,8-TCDD in soil is negligible under most conditions. Leaching of 2,3,7,8-TCDD from soil is possible under special conditions: for example, from sandy soils (U.S. EPA, 1984a).

At high oral and dermal doses, dioxin caused death in rats by hepatic cell necrosis. Death could follow a lethal dose by weeks. Acute and subacute exposure lead to hepatic necrosis, thymic atrophy, hemorrhage, lymphoid depletion and chloracne (Sax, 1984).

In chronic animal studies, liver toxicity was reported after oral exposure to dioxin in rats (Goldstein, et al., 1982; NTP, 1980) and in mice (NTP, 1980; Toth, et al., 1978, 1979). Dermatitis and amyloidosis of the kidney, spleen and liver were reported in mice exposed to dioxin orally (Toth, et al., 1978 and 1979). Humans exposed to dioxin in the environment and workplace reported increased occurrences of chloracne, peripheral neuropathy, fatigue, eye irritation, headache, possibly birth defects and tumors (U.S. EPA, 1984a,b, 1985).

Teratogenesis has been reported in the literature in animals exposed to dioxin (Smith, et al., 1976). Cleft palate has been induced in mice exposed to dioxin orally (Neubert and Dillman, 1972). Birth defects in humans have been reported in areas where dioxin was accidentally released or dioxin had been sprayed (U.S. EPA, 1979; Hanify, et al., 1981; Field and Kerr, 1979; U.S. EPA, 1984c).

Dioxin has been classified in Group B_2 Probable Human Carcinogen, by the U.S. EPA (1984c). This is because the evidence for carcinogenicity to humans was judged "inadequate"; the evidence for carcinogenicity to animals was judged "sufficient" and the evidence for activity in short-term tests to be "inadequate".

Several investigators reported the development of tumors, mostly soft tissue sarcomas, lymphomas and stomach carcinomas in people exposed to dioxin (Holden, 1979; Cook, et al., 1980; Moses and Selikoff, 1981; U.S. EPA, 1984c). The data are suggestive; however, it is not possible to link human exposure to dioxin alone with induction of tumors. In animals, there was a significant increase in tumor incidence, especially hepatocellular carcinoma in rats exposed to dioxin (Kociba, et al., 1978) and hepatocarcinoma, neoplastic nodules, adenomas, fibrosarcomas, histocyte lymphoma, and carcinoma in mice exposed to dioxin (NTP, 1980).

The National Academy of Science Committee on Drinking Water and Health (NAS, 1977) proposed an ADI of 10^{-9} ug dioxin/kg of body weight/day. EPA proposed a criteria of 1.3×10^{-9} to 1.3×10^{-7} ug dioxin/1 in ambient waters (U.S. EPA, 1984b). A carcinogenic potency for oral exposure to humans (q_1^*) was estimated to be 1.56×10^5 $(mg/kg/day)^{-1}$ by the U.S. EPA (1984c).

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Trichlorophenoxyacetic Acid (2,4,5-T)

Trichlorophenoxyacetic acid (2,4,5-T) is a white crystalline solid or a light tan solid having a molecular weight of 255.48 and a molecular formula of $C_8H_5ClO_3$. It is an herbicide (Sax, 1984).

Trichlorophenoxyacetic acid is a highly toxic chlorinated phenoxyacid herbicide that is rapidly excreted after ingestion. It is readily absorbed via inhalation and ingestion and slowly via skin. Signs of intoxication include weakness, lethargy, anorexia, diarrhea, ventribular fibrillation and/or cardiac arrest and death (Sax, 1984).

Occupational exposure of humans to 2,4,5-T (along with other chemicals such as 2,4-D and triphenols and 2,3,7,8-TCDD) resulted in reduced nerve conduction velocities (Singer, et al, 1982). Case-control epidemiological studies of populations in Scandinavian countries exposed to the phenoxy herbicides (as well as other chemicals and contaminants) indicated excess risk to the development of soft-tissue sarcomas and malignant lymphomas (Axelson and Sundell, 1974; Axelson et al., 1979; Hardell, 1977; Hardell and Sandstrom, 1979; Eriksson et al., 1981; Hardell et al., 1981; Riihimaki et al., 1978). Reanalysis of these results indicated a causal relationship existed between phenoxyherbicide exposure and sarcoma risk (U.S. EPA, 1980, 1985).

In the U.S., cases of soft tissue sarcoma in workers exposed to industrial and agricultural chemicals including 2,4,5-T and 2,4,5-TP have been reported (Cook, 1981; Moses and Selikoff, 1981; Honcher and Halperin, 1981). However, it is uncertain whether the sarcomas were caused by any specific herbicide due to the exposure to multiple chemicals.

A positive correlation 2,4,5-T usage with increased rates of spontaneous abortion and birth defects have been reported for human populations in Oregon (U.S. EPA, 1979), New Zealand (Hanify, et al., 1981) and Australia (Field and Kerr, 1979). However, it is uncertain whether the reproductive effects were caused by any specific herbicide due to the possible contamination of 2,4,5-T

with 2,3,7,8-TCDD and other impurities. There have also been reported cases of a lack of correlation of reproductive and teratogenic effects with phenoxy herbicide exposure in human populations in Arkansas (Nelson, et al., 1979), Hungary (Thomas, 1980), New Zealand (Dept. of Health, New Zealand, 1980; McQueen, et al., 1977; Smith et al., 1982) and Australia (Alfred, 1978).

The threshhold limit value (TLV) for 2,4,5-T in air is 10 mg/m^3 (Sax, 1984).

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2(2,4,5-Trichlorophenoxy)propionic acid (= 2,4,5-TP, Silvex)

2(2,4,5-Trichlorophenoxy) propionic acid exists in crystalline form with a molecular weight of 269.53. It is a plant hormone used to control woody plants on uncropped land (Merck Index, 1983). Commercial preparations contain 0.1 PPM or less of TCDD (NRC, 1977).

Silvex may be released into the environment during spraying operations on land or in water using herbicide formulations containing it. It may also be released as a result of hydrolysis of esters of Silvex. When released on land, it strongly adsorbs to soils and biodegrades (average half-lives ranging from 12-17 days). 2,4,5-Trichlorophenol has been reported to be a product of biodegradation. It is not expected to leach, hydrolyze or evaporate. It may be lost due to runoff that may be significant from treated fields. If released to water, Silvex will biodegrade slowly and strongly adsorb to sediment, where slow biodegradation will occur. It will not hydrolyze appreciably or bioconcentrate but may be subject to photooxidation near the surface of waters. Silvex may be released to air during spraying operations but not as a result of evaporation due to its low vapor pressure. Silvex will not appreciably hydrolyze or bioconcentrate in aquatic organisms [National Library of Medicine, Toxnet System, Hazardous Substances Databank (HSDB), 1989, Bethesda, MD].

In acute animal studies with mallard ducks, Silvex produced ataxia, abnormal position of wings, abnormally pointed tail, abnormal walking, and minor tremors [National Library of Medicine, Toxnet System, Hazardous Substances Databank (HSDB), 1989, Bethesda, MD]. In chronic animal studies with beagles, Silvex produced liver pathology after 1 year of exposure (NRC, 1977).

In reproductive studies using lab animals given Silvex, there were few maternal deaths, rats lost their hair (alopecia), had vaginal bleeding and there was decreased pup weight in offspring (embryotoxicity). At higher doses, however, Silvex produced cleft palates (7%) in mice (NRC, 1977). There were also malformed fetuses, including cleft palates, and fetal mortality in mice fed Silvex (Courtney, K.D., 1977).

It is irritating to the eyes, skin and mucous membranes (Merck Index, 1983). There is fatigue, weakness, anorexia, nausea, vomiting, diarrhea, lethargy progressing to coma and constricted pupils followed by flaccid paralysis, convulsions and progressive decline in blood pressure, coma and ultimate death. Chronic exposure may lead to CNS defects in control of motor function [National Library of Medicine, Toxnet System, Hazardous Substances Databank (HSDB), 1989, Bethesda, MD]. Seven men and one woman given oral doses of 1 mg/kg Silvex had no adverse effects (Sauerhoff, M. W., et al., 1977).

The NOEL was judged to be 0.9 mg/kg/day for male dogs and 2.6 mg/kg/day for female dogs (Milby, T.H., et al, 1981). The maximum contaminant level is 0.01 mg/l [40 CFR 141.12 (7/1/83)].

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