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QUALITATIVE RISK ASSESSMENT

BUFFALO OUTER HARBOR SITE

CITY OF BUFFALO
ERIE COUNTY, NEW YORK

915026

Prepared For:

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File No. 0291037.02

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SECTION 1

INTRODUCTION

1.1 PROJECT BACKGROUND

The 113-acre Buffalo Outer Harbor Site is the result of filling activities which occurred over the past 100 years. Fill materials, including incinerator ash, casting sands, blast furnace slag, dredged lake spoils, and miscellaneous construction and demolition debris were disposed in the area, forming the land mass which is known as the Buffalo Outer Harbor Site. The property has been utilized most recently as a transfer station for dry bulk materials such as gypsum, sand, salt, iron pellets, coke, and possibly coal. The only current occupant at the site is the Allen Boat Company, which operates a boat yard in the center of the property. Currently, the remainder of the site is vacant.

A Phase I Remedial Investigation was performed to determine the nature, extent, and source(s) of contamination at the site. A total of 112 surface soil samples, 110 subsurface soil samples, 30 groundwater samples, 16 sediment samples, and 6 surface water samples were analyzed.

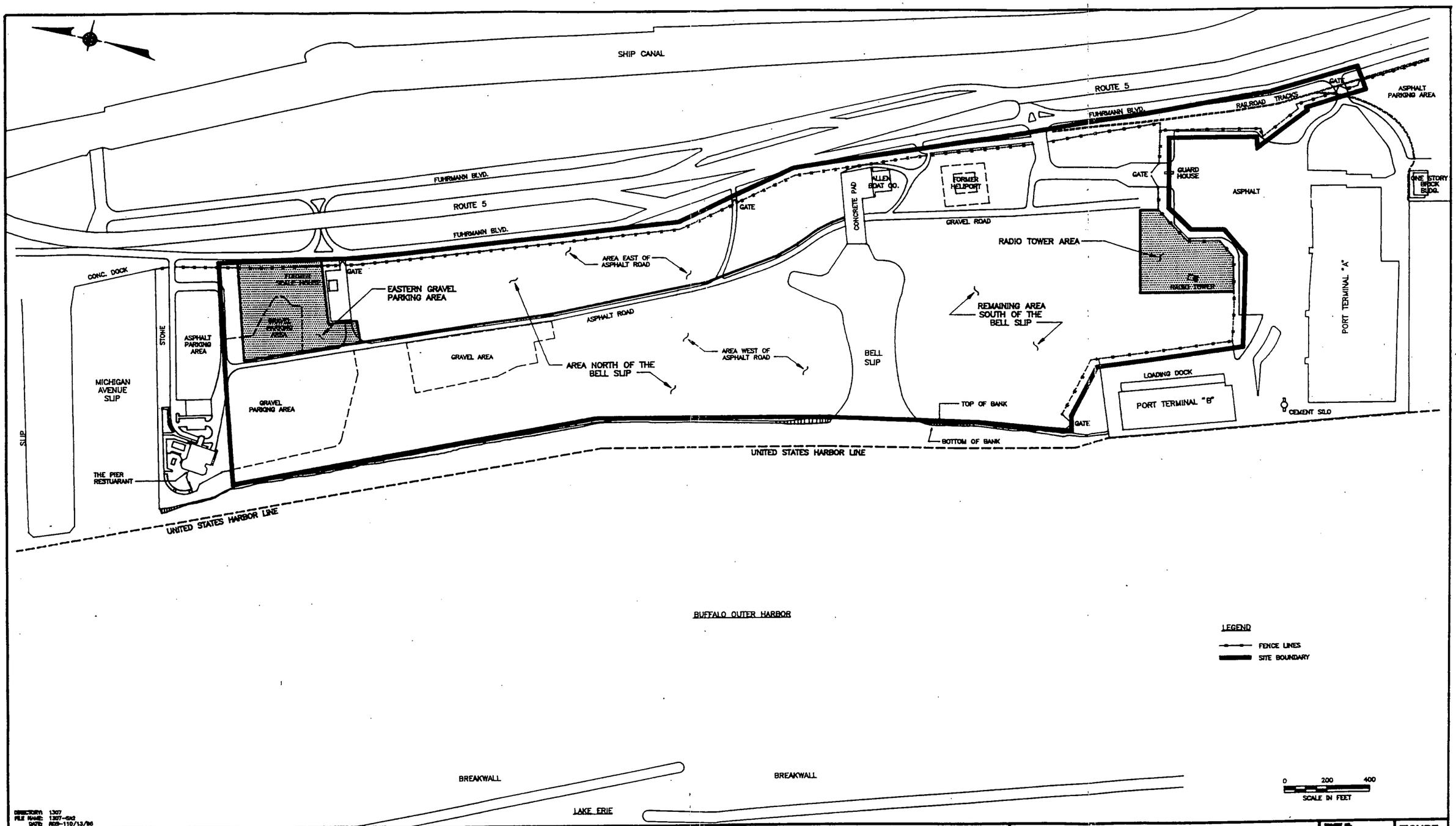
Subsequent to the Phase I investigation, additional analyses were conducted in selected areas of the site to further define the extent of contamination. This Phase II investigation consisted of ten additional surface soil analyses in the Eastern Portion of the Gravel Parking Area, installation of four additional groundwater monitoring wells in the Radio Tower Area, collection of groundwater samples from 11 existing wells and the four new wells, and collection of 12 subsurface soil boring samples. Results from the investigations have been published in the draft document entitled "Phase I/Phase II Remedial Investigation Report for the Buffalo Outer Harbor Site" (RI) prepared by Dvirka and Bartilucci Consulting Engineers.

The New York State Department of Environmental Conservation requires that a qualitative risk assessment be prepared in conjunction with the Remedial Investigation to determine the risk to human health and the environment from site contamination and to determine the need for remedial action at the site. This document constitutes the qualitative risk assessment prepared for the Buffalo Outer Harbor Site.

1.2 RISK ASSESSMENT METHODOLOGY

The goals of the qualitative health risk and environmental assessment are to:

- Provide a qualitative analysis of human health risks under the current site conditions;
- Identify the qualitative potential impacts to flora and fauna posed by existing contamination at the site; and
- Provide a basis for determining what chemical concentrations can remain on site while providing adequate protection of human health and the environment.



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NO.	DATE	REVISION

DVIRKA AND BARTILUCCI
CONSULTING ENGINEERS

BUFFALO OUTER HARBOR
BUFFALO, NEW YORK

SITE AREAS

PROJECT NO. 1307
DATE MARCH 1988
SCALE 1"=200'

FIGURE 1

Because the nature of contamination at the site varies based on the fill material that was placed in different areas, each of the five areas designated in the RI will be evaluated individually in terms of the human health and environmental risks. The five site areas are as follows:

- Radio Tower Area;
- Area South of the Bell Slip (excluding Radio Tower Area);
- Area East of the Asphalt Road;
- Area West of the Asphalt Road; and
- Eastern Portion of the Gravel Parking Area.

The location of each of the five areas is indicated on the site map in Figure 1.

For each of these areas, the human health and the environmental assessments will:

- Identify contaminants of concern;
- Define migration pathways;
- Identify routes of exposure and potential receptors; and
- Provide a qualitative assessment of risk.

The qualitative risk assessment serves as a basis for the Feasibility Study which will evaluate alternatives for the remedial approach to be taken at the site.

SECTION 2

IDENTIFICATION OF CHEMICALS OF CONCERN

2.1 CRITERIA USED TO IDENTIFY CHEMICALS OF CONCERN

Determination of chemicals of concern for each medium was based on analysis performed during the Buffalo Outer Harbor Phase I and Phase II investigations as follows:

- **Surface Soil.** A total of 112 Phase I on-site composite surface soil samples were analyzed. Ten additional surface soil samples from the Eastern Portion of the Gravel Parking Area were collected during Phase II to further define the extent of surface soil contamination in this area.

- **Shallow Subsurface Soil.** A total of 53 shallow subsurface composite soil samples (collected from 0 to 8 feet) were collected during installation of piezometers and construction of soil borings and monitoring well boreholes during Phase I. Four additional grab samples were collected at 0 to 2 feet during construction of new monitoring wells in Phase II. In addition, two borings were installed in the vicinity of well GW3A and grab samples were collected at 5 to 7 feet for total lead and TCLP parameters analysis.

- **Deep Subsurface Soil.** A total of 57 deep subsurface soil grab samples (depths between 8 and 26 feet) were collected during installation of piezometers and construction of soil borings and monitoring well boreholes during Phase I. Four additional grab samples were collected from deep subsurface soil during construction of new monitoring wells in Phase II. Two borings were installed in the vicinity of well GW9 to collect subsurface soil samples for total lead and TCLP parameters.

- **Groundwater.** Samples were collected from 29 monitoring wells and one piezometer during Phase I. During Phase II, four groundwater monitoring wells were installed in the Radio Tower area to provide additional information regarding groundwater quality and flow direction. Groundwater samples were collected from 11 existing wells and the four newly installed wells to further evaluate on-site groundwater quality. Duplicate analyses were performed for 4 samples.

- **Surface Water.** Six samples were collected from the Michigan Avenue Slip, the Bell Slip, and the Outer Harbor during Phase I.

- **Sediment.** Sixteen sediment samples were collected from the Michigan Avenue Slip, the Bell Slip, and the Outer Harbor during Phase I.

- **Air.** Ten ambient air samples were collected during the installation of biased soil borings in the Radio Tower Area. Compounds were analyzed for VOCs and nitrobenzene.

As part of the RI, screening levels were established for chemicals detected in soil, water, and sediment samples to characterize the nature and extent of contamination and to identify chemicals of concern. Screening level values are presented in Section 4 of the RI. Screening levels for chemical concentrations in each medium were established as follows:

- **Soil.** Screening levels were established based on applicable guidance documents, primarily the New York State Department of Environmental Conservation (NYSDEC) Technical and Administrative Guidance Memorandum (TAGM): Determination of Soil Cleanup Objectives and Cleanup levels (January 1994). Additional sources of information included screening and cleanup levels utilized for various sites located in New York State with similar contaminants of concern, discussions with NYSDEC and New York State Department of Health, as well as screening and cleanup levels developed and used by other states, including Massachusetts and New Jersey. Off-site samples for surface soil collected near the Buffalo Outer Harbor Site also were considered in determining screening levels.

- **Groundwater.** Screening levels were obtained from the NYSDEC Technical and Operational Guidance Series (TOGS) - Ambient Water Quality Standards and Guidance Values (October 1993).

- **Surface Water.** Surface water analytical results were compared to Class B Surface Water Standards for the propagation of fish, or wildlife consumption of fish (Type A). As stated in the RI, chemical concentrations detected in the surface water were not excessively elevated and do not appear to be attributable to site contamination. Therefore, surface water is not considered in the risk assessment.

- **Sediment.** Screening levels based on the NYSDEC Division of Fish and Wildlife Technical Guidance for Screening Contaminated Sediment (November 1993) were used to determine chemicals of concern in sediment.

- **Air.** Occupational Safety and Health Administration (OSHA) time weighted average limits for air contaminants were compared to analytical results for air samples. As stated in the RI, none of the compounds detected exceeded OSHA limits. However, because excavation at the site could expose volatile compounds including benzene, nitrobenzene, 4-chloroaniline, and cyanide, air is considered a potential media of concern for these compounds.

Chemicals that exceeded these screening levels were identified as potential chemicals of concern for the purposes of the risk assessment. The chemicals of concern discussed in this risk assessment are present in one or more media including surface soil, subsurface soil, sediment, or groundwater. These chemicals include:

- PCBs
- Lead
- Antimony
- Arsenic
- Benzene
- CaPAHs
- Nitrobenzene
- 4-Chloroaniline
- Cyanide

When several chemicals with similar properties and toxicity were detected above screening levels in a specific site area (for example, VOCs in the Radio Tower Area), the chemical within that class that appears to present the greatest hazard in terms of prevalence, toxicity, and/or mobility was selected as the indicator chemical for that class of compounds for further consideration in the risk assessment.

Chemicals that exceeded screening levels, but which are not addressed individually in the risk assessment, are as follows:

- Trichloroethene concentrations slightly exceeded screening levels in two of 30 Phase I groundwater samples. Trichloroethene was detected in one surface water sample (the RI does not specify surface water standards). Singular exceedances of chloromethane and chloroform screening levels also occurred in groundwater samples. Since these compounds were not detected in other groundwater, surface water, sediment, or soil samples, they were excluded from further consideration.

- Toluene and chlorobenzene exceeded screening levels in one groundwater sample. These compounds were not detected in sediment samples, and toluene was measured below the detection limit (at an estimated quantity) in one surface water sample. Soil screening levels for these compounds were not provided in the RI; however, the screening level for total VOCs was exceeded in two soil borings which showed high concentrations of toluene and chlorobenzene. These two compounds were eliminated from further discussion because benzene, which exceeded screening levels in the same groundwater sample that contained toluene and chlorobenzene, was selected as the indicator chemical for further consideration in this risk assessment. Benzene also was detected in the soil borings which exceeded the total VOCs screening level.

- Pesticides including Dieldrin, Endrin, beta BHC, Heptachlor epoxide, 4,4-DDD, and/or 4,4-DDT were detected at very low levels (less than 70 ppt) in at most two groundwater samples (not necessarily the same samples). When the same pesticide was detected in two wells, one well was not directly down-gradient from the other or in the same site area. The

groundwater screening level for these pesticides is non-detection. No pesticides were detected in surface water samples. Dieldrin, Endrin, 4,4-DDD, and 4,4-DDT were detected in sediment samples, but did not exceed screening levels (note the RI does not provide screening levels for Dieldrin and 4,4-DDD). Endosulfan I, Endosulfan II, alpha-Chlordane, and gamma-Chlordane exceeded screening levels in several sediment samples, but the RI states that the presence of PCBs in the samples may have affected these results (in the sample analysis, peaks for the PCB pattern fell within the retention time window for some pesticide compounds). Pesticides were not considered further in this risk assessment because the low concentrations and frequency of detection do not represent a significant risk to human health and the environment.

- Naphthalene, 1,2-Dichlorobenzene, 1,3-Dichlorobenzene, and 1,4-Dichlorobenzene are semi-volatile organic compounds (SVOC) which exceeded screening levels in only one groundwater sample (in the Radio Tower area) in both the Phase I and Phase II investigations. These compounds also were detected at high levels in soil samples collected from the Radio Tower area. 4-Chloroaniline (an SVOC detected in the groundwater sample) and nitrobenzene (an SVOC detected in the soil samples) were selected as the related indicator chemicals for consideration in the risk assessment.

- Phenol exceeded NYSDEC sediment criteria in four sediment samples. Two groundwater samples, located in the southeast corner of the site near the Port Terminals and asphalt parking area, slightly exceeded phenol screening levels. Phenol was detected in 2 of 122 surface soil samples and 8 of 122 subsurface soil samples (RI does not provide soil screening levels for phenol).

  Phenol is highly water soluble, and biodegrades quickly in water and in soil under both aerobic and anaerobic soil conditions. Phenol may persist for longer periods of time if a large amount is released at one time, or if it is constantly released to the environment from a source. Because phenol was not detected in groundwater or soil samples along the shoreline where phenol was present in the sediment, the site does not appear to be a source of phenol in the sediment. In addition, soil and groundwater concentrations of phenol at the site do not indicate that a significant point source exists on the site. For these reasons, phenol was not considered further in this risk assessment.

- Barium concentrations in groundwater exceeded screening levels in only two wells sampled during Phase I, and slightly exceeded screening levels in a different well during Phase II analysis. Barium toxicity is low compared to other metals discussed as indicator chemicals in this risk assessment. Therefore, barium was excluded from further consideration.

- Screening levels for cadmium and chromium were exceeded in at most four surface soil samples and at most six subsurface soil samples overall in the Phase I and Phase II investigations. Samples in which the chemicals were detected were not focused in one area. Lead or antimony, discussed as indicator chemicals in this risk assessment, were present at concentrations

above screening levels in almost all samples where cadmium and/or chromium exceeded screening levels. In cases where no indicator metals were detected in the same sample (one surface soil and one subsurface soil sample for cadmium, two surface soil samples for chromium), concentrations for these two chemicals were only slightly above screening levels. These chemicals did not exceed screening levels in any groundwater, surface water, or sediment samples. Therefore, these metals were removed from consideration in this risk assessment.

- Screening levels for nickel were exceeded in three surface soil samples and 14 subsurface soil samples (all from Phase I). One sediment sample also exceeded screening levels. Samples in which nickel was detected were not focused in one area. Lead or arsenic, discussed as indicator chemicals in this risk assessment, were present at concentrations above screening levels in almost all samples where nickel exceeded screening levels. In cases where no indicator metals were detected in the same sample (one surface soil, one subsurface soil, and one sediment sample), nickel concentrations were only slightly above screening levels. Nickel did not exceed screening levels in any groundwater or surface water samples. Therefore, nickel was removed from consideration in this risk assessment.

- Screening levels for copper were exceeded in six surface soil samples and 22 subsurface soil samples overall in Phase I and Phase II investigations. One sediment sample also exceeded screening levels. Samples in which copper was detected were not focused in one area. Lead or arsenic, discussed as indicator chemicals in this risk assessment, were present at concentrations above screening levels in almost all samples where copper exceeded screening levels. In cases where no indicator metals were detected in the same sample (three surface soil and four subsurface soil samples), copper concentrations were not significantly elevated. Copper did not exceed screening levels in any groundwater or surface water samples. Copper is an essential element and does not present a significant risk to human health or the environment at concentrations observed at the Buffalo Outer Harbor. Therefore, copper was removed from consideration in this risk assessment.

- Thallium concentrations were slightly above screening levels in six Phase II groundwater samples. Duplicate analyses were performed for two of these samples, and thallium was not detected in the duplicates. During Phase I, thallium did not exceed groundwater screening levels and was not detected in any soil or surface water samples. Thus, thallium was excluded from this risk assessment.

- Zinc concentrations exceeded screening levels in one Phase I groundwater sample and a different Phase II groundwater sample. A total of 18 surface soil and 28 subsurface soil samples exceeded screening levels overall in the Phase I and Phase II investigations. Zinc concentrations in two of 16 sediment samples exceeded the severe effect level indicated in the NYSDEC sediment criteria document. Zinc concentrations did not exceed screening

levels in any surface water samples. Samples in which zinc was detected were not focused in one area.

Lead or arsenic, discussed as indicator chemicals in this risk assessment, were present at concentrations above screening levels in almost all samples where zinc exceeded screening levels. In cases where no indicator metals were detected in the same sample (eight surface soil, three subsurface soil, and one sediment sample), zinc concentrations were not significantly elevated. Because zinc toxicity is minor and requires exposure to significant amounts of the material, zinc was excluded from further consideration in this risk assessment. However, since both sediment samples of concern were taken from the Bell Slip area, the Allen Boat Company operations may be a source of zinc in the sediments and surface soil.

2.2 BRIEF TOXICITY INFORMATION FOR CHEMICALS OF CONCERN

Information pertaining to the chemical use, health effects, and environmental fate was obtained from the Hazardous Substance Data Bank (HSDB) database at the National Institute of Health (NIH) during May and June, 1995. Additional toxicity information was obtained from Toxicological Profiles published for the Agency for Toxic Substances and Disease Registry, and Casarett and Doull's Toxicology (Amdur, Doull, and Klaassen, 1991).

2.2.1 PCBs

Polychlorinated biphenyls (PCBs) are very stable, environmentally persistent compounds. The United States has discontinued production of PCBs, but PCBs formerly were used in electrical capacitors and transformers, plasticizers, and in paper manufacturing.

If released to soil, PCBs adsorb tightly to soil, with adsorption generally increasing with the degree of chlorination of the PCB. PCB leaching into groundwater is low. PCBs are very lipid soluble, and as a result, have the potential to accumulate and bioconcentrate in the fat tissues of organisms.

Effects of PCB exposure include liver damage, immunosuppression, and skin reactions (chloracne). PCBs are classified as a probable human carcinogen (Class B2) based on animal evidence. PCBs have been shown to interfere with reproduction in wildlife, especially birds.

2.2.2 Lead

Lead is a highly stable metal used in the manufacture of batteries, pigments and ceramics, ammunition, and solder. Lead additives in gasoline have been another major source of lead in the environment.

Lead in soil is relatively immobile; it binds tightly to soils and may be converted to insoluble sulfate, sulfide, oxide, and phosphate salts. Lead in water is frequently in the form of suspended solids.

Most lead poisoning occurs gradually over a period of time from the accumulation of lead from sources such as lead paint or industrial dusts. In young children, developmental defects, including learning disabilities, lowered IQ, and behavioral abnormalities, can occur. At high levels, lead can cause encephalopathy with imminent risk of death, permanent mental retardation, and motor deficits.

Chronic exposure to lead can cause decreased fertility, renal dysfunction, and anemia. Lead is classified as a probable human carcinogen (Class B2) based on animal evidence.

Lead is a common contaminant for vegetation through deposition on the plant surface. Plants can absorb and translocate available lead from nutrient solution, and to a lesser degree from soil, but not in significant quantities. Birds and mammals are susceptible to lead poisoning. Affected animals have shown neurological effects, weight loss, and decreased reproductivity.

2.2.3 Antimony

Antimony is used in alloys with metals for electrical and other uses, in fire retardant compounds, and as a chemical intermediate for ceramic and glass additives.

Toxic effects from antimony exposure include myocardial changes, gastrointestinal symptoms, weight loss, and skin eruptions known as antimony spots. Some evidence of reproductive toxicity also exists. This compound is not classifiable as to its human carcinogenicity.

2.2.4 Arsenic

Arsenic is used in metal alloys and in the manufacture of electrical devices and glass. It is a stable metal, but is found in many different compounds. Elemental arsenic is virtually insoluble in water or body fluids. However, different compounds of arsenic vary in terms of environmental fate and transport. Depending on physical and chemical characteristics of the soil and groundwater, soluble forms of arsenic in groundwater may transform into other forms which adsorb to particles. Arsenic in soil is generally insoluble.

Arsenic may be absorbed by ingestion, inhalation, and permeation of skin or mucous membranes. Acute exposure to arsenic may be fatal; the primary target organs are the gastrointestinal tract, the heart, brain, and kidneys. Chronic exposure to arsenic may cause neurological damage, liver injury, and irritation of the throat and respiratory tract. Arsenic is classified as a human carcinogen based on epidemiological studies. However, it has been difficult to confirm this relationship with laboratory animals.

2.2.5 Benzene

Benzene is a volatile organic compound found in gasoline and used in the manufacture of medicinal chemicals, dyes, organic compounds, linoleum, and lacquers. Its use as a solvent is now discouraged.

Rapid volatilization of benzene occurs in surface soil or water. Benzene is highly mobile in soil and may leach into groundwater. Benzene is relatively soluble in water.

Benzene toxicity may occur through inhalation or dermal exposure, and to a lesser extent through ingestion. Acute toxic effects of benzene include depression of the central nervous system and burning and blistering of the skin. Chronic exposure may lead to blood disorders including aplastic anemia and leukemia, and respiratory problems. Benzene is classified as a human carcinogen based on epidemiological studies.

2.2.6 CaPAHs

Carcinogenic Polycyclic Aromatic Hydrocarbons (CaPAHs) are semi-volatile organic compounds that have little commercial use or application in the United States, but rather are products of incomplete combustion. They are commonly found in coal and petroleum tars, asphalt, carbon black, and cigarette smoke, as well as smoked and charcoal-broiled foods. CaPAHs adsorb strongly to sediments and particulate matter, and are generally found in this form. These compounds do not leach readily, and if released in water, will adsorb to sediments.

Exposure to these chemicals occurs through inhalation or ingestion. Acute toxicity from these chemicals generally does not occur. Chronic exposure can cause a variety of health effects including eye irritation, respiratory problems, immunosuppression, and precancerous lesions ("coal tar warts"). CaPAHs are considered probable human carcinogens based on epidemiological and animal studies. Chronic exposure has been correlated to increased incidences of cancers of the skin, bladder, lung, and gastrointestinal tract.

2.2.7 Nitrobenzene

This semi-volatile organic compound is used for the manufacture of aniline, in soaps and shoe polishes, and in the production of pesticides, rubber chemicals, and pharmaceuticals. Nitrobenzene is water soluble, and is moderately adsorbed to soil. No significant evidence of biomagnification in organisms has been observed.

Nitrobenzene is classified as a poison, and is toxic by all routes of exposure (dermal, ingestion, and inhalation). The most common effect is methemoglobinemia, which prevents oxygen exchange in the blood. Repeated exposure may cause liver impairment. Teratogenic effects and reproductive toxicity have been observed in laboratory experiments. This compound is not classifiable as to its human carcinogenicity.

2.2.8 4-Chloroaniline

4-Chloroaniline is a semi-volatile organic compound used in the production of dyes, pharmaceuticals, and agricultural chemicals. In the environment, this compound binds tightly to organic soil. Mineralization of the compound also occurs, releasing carbon dioxide as a result.

Like nitrobenzene, 4-Chloroaniline is absorbed rapidly by all routes of exposure, and induces methemoglobinemia. This compound is classified as a probable human carcinogen based on evidence from animal toxicity studies.

2.2.9 Cyanide

Cyanide is a common element of rat and pest poisons, metal polishes, and photographic solutions. Cyanide in the form of alkali metal salts is very soluble in water.

Cyanide is a chemical asphyxiant which inhibits the uptake of oxygen in the cells and ultimately causes respiratory failure. It is absorbed readily via all routes of exposure, and can cause rapid death. This compound is not classifiable as to its human carcinogenicity.

SECTION 3

QUALITATIVE RISK ASSESSMENT

This risk assessment qualitatively evaluates chemicals of concern and the affected media with respect to potential exposure pathways and receptors. For the site as a whole, potential pathways for human and wildlife exposure include ingestion directly from soil or through contaminated biota (for example fish containing PCBs), inhalation of dust, and dermal contact with contaminated soil and fugitive dusts. Potential human receptors include on-site workers (Allen Boat Company), individuals accessing the site for fishing, recreation, or other purposes, remedial and construction workers, and off-site individuals in the vicinity of the site (The Pier Restaurant, Port Terminals A and B). Potential wildlife receptors include waterfowl and mammals which live or feed on site, fish, and other aquatic organisms.

Based on this information, the following assumptions were made in the qualitative exposure pathway analyses:

- Areas of debris piles and contaminated soil on the site may be subject to direct contact by individuals or wildlife on site.
- Areas of debris piles and contaminated soil on the site are potentially subject to wind scour, and particulate-bound contaminants may be released to the air during windy conditions.
- Groundwater is in contact with waste and contaminated soil, and dissolved contaminants may be released to the groundwater.
- Surface runoff carrying contaminated soil may migrate off site.
- Excavation may expose waste and/or contaminated soils.

3.1 EXPOSURE AND PATHWAY OVERVIEW FOR THE SITE

To qualitatively assess the exposures related to the site, the various exposure scenarios can be classified in terms of the following general release mechanisms:

- Volatilization;
- Wind erosion (dust);
- Direct contact;
- Overland runoff; and
- Infiltration to groundwater.

Direct exposures to the chemicals of concern from the above four release mechanisms can occur in a number of ways:

- Ingestion of contaminated soil containing dissolved or particulate-bound contaminants;

- Inhalation of vapors or airborne particulate-bound contaminants;
- Dermal absorption of contaminants via direct contact with vapors, waste, contaminated soil, and groundwater; and
- Direct contact of contaminated runoff.

Potential exposure pathways are examined for functionality and completeness as follows:

- Functional Exposure Pathway - A functional pathway requires that a waste source, release mechanism, and transport mechanism be present. If any one or some of these components is missing, the pathway is considered nonfunctional. Functional pathways for this site are listed in Table 3-1.
- Complete Pathway - A complete pathway requires that a functional pathway exists, as well as potential receptors to the exposure, and an exposure/uptake route. If one or more of these components is missing, an exposure is incomplete and poses a low risk.

3.2 EXPOSURE AND PATHWAY ASSESSMENT FOR THE FIVE SITE AREAS

Currently, access to the Buffalo Outer Harbor Site is unrestricted. Humans access the site for fishing, mainly in the Bell Slip area, and for scavenging from the construction and demolition wastes that have been disposed at the site. Employees and customers of the Allen Boat Company also enter the site. Activity at the site increases during the summer. Individuals using watercrafts in the Outer Harbor area may access the site. Parts of the site are used for parking for water activities, for overflow parking at The Pier Restaurant, and for an annual summer festival just north of the site.

The number of individuals exposed and frequency and duration of exposure could increase under various future use plans. Potential future site uses include residential, park/recreational (including a beach area), or commercial/industrial.

Wildlife, including native and migratory birds and waterfowl; mammals, including muskrats, raccoons, and voles; and some reptiles and amphibians have been observed or noted by sign on the site.

The following sections summarize the chemicals of concern in each medium for each site area, and assess the completeness of the exposure pathways for the contaminants of concern in each of the site areas based on the potential receptors and exposure routes.

Table 3.1

SUMMARY OF POTENTIAL EXPOSURE ROUTES FOR THE BUFFALO OUTER HARBOR SITE

Source Media	Chemicals of Concern	Potential Exposure	Route of Migration	Potential Receptors
Surface Soil (0 to 0.5 feet)	PCBs Lead Arsenic CaPAHs	Dermal contact, ingestion and inhalation of fugitive dust emissions	Dust and surface water runoff, direct contact by humans or animals	on-site and off-site
Shallow Subsurface Soil (0.5 to 8 feet)	Lead Antimony Arsenic CaPAHs	Dermal contact, ingestion and inhalation during construction	Leaching to groundwater, direct contact by humans or burrowing animals, dust generated during construction	on-site
Deep Subsurface Soil (8 to 22 feet)	Lead Antimony Arsenic CaPAHs Benzene Nitrobenzene	Dermal contact, ingestion and inhalation during construction	Leaching to groundwater, direct contact with exposed soil or inhalation of vapors or dust generated during construction activities involving deep excavation	on-site
Ground-water	Lead Antimony Arsenic CaPAHs Benzene Cyanide 4-Chloroaniline	Dermal contact and ingestion	Direct contact by animals burrowing through the unstabilized shoreline area, discharge to Bell Slip and Buffalo Outer Harbor	on-site and off-site
Sediments	Lead CaPAHs	Dermal contact and ingestion, and indirect uptake through ingestion of contaminated biota	Direct contact, ingestion of contaminated biota	on-site and off-site

3.2.1 Radio Tower Area

Chemicals of concern which exceeded screening levels in samples from the Radio Tower Area are summarized in Table 3.2. Surface soils in the Radio Tower Area contain PCBs and lead at concentrations above screening levels. In the shallow subsurface soils, lead, antimony, and CaPAHs exceeded screening levels.

Lead, antimony, and/or nitrobenzene were detected above screening levels in several of the 15 grab samples collected from the deeper subsurface soils. Nitrobenzene also was detected above maximum levels established by the United States Environmental Protection Agency (USEPA) in a TCLP analysis performed on a sample from this area. Note multiple grab samples were collected from different depths in two soil borings to assess the vertical extent of contamination. Soil boring results indicate that nitrobenzene contamination is localized at depths of 10 to 20 feet. Note benzene was detected in one of four Phase I soil samples and one of two Phase II soil samples analyzed for VOCs (no screening levels were specified in the RI). Its presence corresponds to the detection of elevated benzene levels in the groundwater in this area.

In the Radio Tower Area, one well was sampled during Phase I and five wells were sampled during Phase II. One well showed significant groundwater contamination during both Phase I and Phase II; chemicals of concern which exceeded screening levels in this well included antimony, benzene, and 4-chloroaniline. In Phase II samples, one other well showed benzene above screening levels, and one well showed lead above screening levels.

Table 3.2

CHEMICALS OF CONCERN IN THE RADIO TOWER AREA

Media	Total Samples	Chemicals of Concern	Screening Level (SL)	Samples Above SL	Concentration Range
Surface Soil	8	PCBs	1 mg/kg	4	1.5 to 8.2 mg/kg
		Lead	500 mg/kg	4	512 to 777 mg/kg
Shallow Subsurface Soil	9 (composite, 0.5 - 8 feet)	Lead	500 mg/kg	1	615 mg/kg
		Antimony	20 mg/kg	1	29.4 mg/kg
	4 (grab, 0.5 - 2 feet)	Lead	500 mg/kg	1	1,170 mg/kg
		CaPAHs	10 mg/kg	2	10.5 & 112 mg/kg
Deep Subsurface Soil	15	Lead	500 mg/kg	2	1,060 & 1,720 mg/kg
		Antimony	20 mg/kg	6*	27.2 to 5,470 mg/kg
		Nitrobenzene	1 mg/kg	5*	4.1 to 13,000 mg/kg
		CaPAHs	10 mg/kg	1	16 mg/kg
		Benzene	none	2**	12 and 5,000 ug/kg
		4-Chloroaniline	none	4**	320 to 49,000 ug/kg
Groundwater	1 (Phase I)	Antimony	3 ug/l	1	244 ug/l
		Benzene	0.7 ug/l	1	15 ug/l
		4-Chloroaniline	5 ug/l	1	2,400 ug/l
	5 (Phase II)	Lead	25 ug/l	1	28.4 ug/l
		Antimony	3 ug/l	1	189 ug/l
		Benzene	0.7 ug/l	2	2 & 8 ug/l
		4-Chloroaniline	5 ug/l	1	1,400 ug/l

* In two cases, samples were from different depths of the same soil boring.
** Number of samples in which chemical was detected (no screening levels specified in the RI).

Surface Soil--

Human access to this area of the site probably occurs infrequently because of the tall vegetation and difficulty in accessing the shoreline for fishing. However, this vegetation provides a favorable habitat for native and migratory birds and waterfowl; mammals including rabbits, raccoons, and voles; and some reptiles and amphibians.

Although the area contains significant cover including high grassland, shrub, and climax vegetation, some areas contain exposed debris piles and soils. Dusts from these exposed areas may be disturbed via wind action and vehicular traffic, providing the potential for inhalation and ingestion of these materials by individuals and wildlife on site and in the neighboring off-site terminal areas. Direct contact exposure also can occur by dermal absorption or ingestion of contaminated surface soils by individuals or wildlife on site.

Therefore, both the air pathway and the direct contact pathway are considered functional and complete for existing site conditions in the Radio Tower Area.

Future use of the site for residential, recreational, or commercial/industrial purposes is likely to increase the number of individuals accessing this area of the site. If the surface soils remain exposed, the inhalation, ingestion, and direct contact exposure pathways will be functional and complete, with an increased number of individuals at risk of exposure. A future use plan which involves removal of affected soil and replacement with clean soil, or covering surface soils in this site area (for example a parking lot), would render the exposure pathways incomplete.

Shallow Subsurface Soil--

Burrowing animals at the site which may contact shallow subsurface soil include rabbits, voles, muskrats, and some reptiles and amphibians. Muskrat dens can be as deep as five feet below the ground surface, but are generally within 50 feet of the shoreline. Because animals on site could burrow to the depths where elevated levels of chemicals of concern were detected, the direct contact pathway for wildlife exposure to shallow subsurface soil in the Radio Tower Area is considered functional and complete.

Because subsurface soil contaminants in the Radio Tower Area currently are not exposed, the air pathway and the direct contact pathway for human exposure to subsurface soil can be considered functional and complete only if construction or excavation activities that expose contaminated soils are performed at the site. Such exposures can be controlled through informed, limited access to the area and compliance with OSHA personal protection requirements.

The likelihood of the shallow subsurface soils becoming exposed may increase under future use scenarios. Under a residential, commercial, or industrial use scenario, occupants may expose contaminated soil as a result of planting, building, or other excavation projects, causing the air and/or direct contact pathways for human exposure to become complete. Again, a future use scenario which removes or decreases accessibility to subsurface soil would decrease significantly the risk of exposure.

Deep Subsurface Soil--

Because subsurface soil contaminants in the Radio Tower Area currently are not exposed, the air pathway and the direct contact pathway for human exposure to subsurface soil can be considered functional and complete only if construction or excavation activities that expose contaminated soils are performed at the site. Excavation also can expose volatile compounds (benzene, nitrobenzene, and 4-chloroaniline) detected in the subsurface soil in the Radio Tower Area, rendering the inhalation and direct contact pathway complete for exposure to vapors. Such exposures can be controlled through informed, limited access to the area and compliance with OSHA personal protection requirements.

Groundwater--

Water level measurements indicate that groundwater flows generally to the east from this area, and the chemicals of concern are below screening levels in downgradient wells. For the groundwater pathway to be considered complete, a receptor and exposure route must

be present. The absence of these chemicals in other downgradient wells sampled indicates that the groundwater contamination is due to fill materials in the immediate area. There is no known use of private wells for potable water in this vicinity. As discussed above regarding subsurface soils, animal habitats at the site are generally above the water table. Therefore, the groundwater pathway is not considered complete for humans or wildlife in the Radio Tower Area under current site conditions.

As long as groundwater is not extracted or exposed for any purpose (e.g. constructing a well or unlined pond), the pathway for human exposure to chemicals of concern in the groundwater or through volatilization from groundwater is not likely to become complete under future use scenarios.

3.2.2 Remaining Area South of the Bell Slip

Chemicals of concern which exceeded screening levels in samples from the Remaining Area South of the Bell Slip are summarized in Table 3.3. Chemicals of concern present in the surface soil samples from this area included lead, arsenic, and CaPAHs. The highest concentration for total CaPAHs in surface soil at the site was measured in the area of the Allen Boat Company. Both surface soil samples which exceeded arsenic screening levels were collected along the rail tracks at the extreme southern end of the site. Lead, Arsenic, and CaPAHs also exceeded screening levels in shallow and deep subsurface samples from this site area. Antimony exceeded screening levels in one deep subsurface sample.

Ten wells and one piezometer were sampled in the Area South of the Bell Slip during the Phase I investigation; only one well in this site area was sampled during the Phase II investigation. During the Phase I investigation, arsenic exceeded screening levels slightly in one shallow well sample, and concentrations were well above screening levels in samples from two intermediate depth wells. These wells are on the western side of the site near the Outer Harbor. The well sampled during Phase II was not one of the wells in which arsenic screening levels were exceeded during Phase I.

Table 3.3

CHEMICALS OF CONCERN IN THE REMAINING AREA SOUTH OF THE BELL SLIP

Media	Total Samples	Chemicals of Concern	Screening Level (SL)	Samples Above SL	Concentration Range
Surface Soil	43	Lead	500 mg/kg	4	514 to 834 mg/kg
		Arsenic	20 mg/kg	2	24.9 & 1,301 mg/kg
		CaPAHs	10 mg/kg	11	10.4 to 80.3 mg/kg
Shallow Subsurface Soil	12 (composite, 0.5 - 8 feet)	Lead	500 mg/kg	2	1,250 & 1,260 mg/kg
		Arsenic	20 mg/kg	1	35.7 mg/kg
		CaPAHs	10 mg/kg	4	10.5 to 16 mg/kg
Deep Subsurface Soil	13	Lead	500 mg/kg	2	580 and 2,200 mg/kg
		Antimony	20 mg/kg	1	74.4 mg/kg
		Arsenic	20 mg/kg	1	21.4 mg/kg
		CaPAHs	10 mg/kg	4	22.5 to 30.8 mg/kg
Groundwater	12	Arsenic	25 ug/l	3	27.4 to 95 ug/l

Surface Soil--

Much of this area of the site is vegetated with high grassland, but rubble and debris piles were distributed over almost half of the area in the section closer to the Bell Slip. The frequency of human exposure is expected to be higher in this area (when compared to other areas of the site) due to the nearby location of the Allen Boat Company and the access to the Bell Slip for fishing.

The potential exists for inhalation and ingestion of contaminants that become airborne through wind action and vehicular traffic. Direct contact with surface soils by on-site individuals and wildlife is also possible. Therefore, both the air pathway and the direct contact pathway are considered functional and complete for existing site conditions in the Area South of the Bell Slip.

Future use of the site for residential, recreational, or commercial/industrial purposes is likely to increase the number of individuals accessing this area of the site. If the surface soils remain exposed, the inhalation, ingestion, and direct contact exposure pathways will be functional and complete, with an increased number of individuals at risk of exposure. A future use plan which involves removal of affected soil and replacement with clean soil, or covering surface soils in this site area (for example a parking lot), would render the exposure pathways incomplete.

Shallow Subsurface Soil--

Because chemicals of concern found in the shallow subsurface soil are not exposed, the air pathway and the direct contact pathway for human exposure are not considered complete under current site conditions. Pathways may become complete if construction or excavation activities that expose contaminated soils are performed at the site. Such exposures can be controlled through informed, limited access to the area and compliance with OSHA personal protection requirements.

Most of the Outer Harbor shoreline is stabilized with rip/rap, but the Bell Slip area consists of unstabilized shoreline with emergent vegetation in some areas. The unstabilized shoreline with emergent vegetation provides a favorable habitat for muskrats to establish burrows. Elevated CaPAHs have been detected in the shallow subsurface in this area. Although no muskrat dens have been documented in the Bell Slip area, muskrats have been observed in the area, and the conditions are such that the exposure pathway is complete.

The likelihood of the shallow subsurface soils becoming exposed may increase under future use scenarios. Under a residential, commercial, or industrial use scenario, occupants may expose contaminated soil as a result of planting, building, or other projects involving excavation, causing the air and/or direct contact pathways for human exposure to become complete. Again, a future use scenario which removes or decreases accessibility to subsurface soil would decrease significantly the risk of exposure.

Deep Subsurface Soil--

Chemicals of concern in the deep subsurface soil in this area were detected at depths of 10 to 24 feet and are not exposed. Therefore, the air pathway and the direct contact

pathway for human exposure are not considered complete under current site conditions. Pathways may become complete if construction or excavation activities that expose contaminated soils are performed at the site. Such exposures can be controlled through informed, limited access to the area and compliance with OSHA personal protection requirements.

Groundwater--

The groundwater pathway is not considered complete for human exposure in the Area South of the Bell Slip under current site conditions because no known potable water supply wells exist in the vicinity of the site. The exposure pathway for wildlife is also incomplete due to the depth at which the groundwater contamination was detected.

Groundwater contour maps presented in the RI indicate that groundwater flows toward the Outer Harbor from the three wells in this site area which exceeded arsenic screening levels. Arsenic-containing groundwater could flow through the native overburden soil and discharge to the Outer Harbor through the bottom sediment. However, arsenic was not detected in surface water samples in this area, and arsenic concentrations in sediment samples in this area were below the severe effect level and below concentrations measured in the background sediment sample. For these reasons, the potential future exposure pathway by migration of groundwater to surface water is considered incomplete.

As long as groundwater is not extracted or exposed for any purpose (e.g. constructing a well or unlined pond), the groundwater pathway for human exposure is not likely to become complete under future use scenarios.

3.2.3 Area East of the Asphalt Road

Table 3.4 summarizes the chemicals of concern which exceeded screening levels in the Area East of the Asphalt Road. Only one category of chemical of concern (CaPAHs) was found above screening levels in one surface soil sample, taken in the vicinity of the former scalehouse near Fuhrmann Boulevard.

Ten of the Phase I and both Phase II shallow subsurface samples were collected in the vicinity of monitoring well No. 3 (GW3) to investigate elevated levels of PAHs and lead detected during earlier investigations. One Phase I sample collected near GW3 showed multiple chemicals of concern, with concentrations above screening levels for CaPAHs, antimony, arsenic, and lead. In both Phase II samples, lead concentrations exceeded screening levels, and lead exceeded maximum allowable levels in the TCLP analysis for one of these samples (screening level is 5 mg/l, concentration of 8.4 mg/l).

Concentrations were well above screening levels in multiple deep subsurface soil samples in the area of GW3. CaPAH concentrations were over 30 mg/kg in two samples; antimony concentrations were over 200 mg/kg in three samples; and lead concentrations were over 1100 mg/kg in five samples.

Chemicals of concern that exceeded groundwater screening levels in this site area were detected in one well (GW3A) screened in the shallow aquifer. Lead and six CaPAH compounds were detected above screening levels in this well during both Phase I and Phase II analyses.

Table 3.4

CHEMICALS OF CONCERN IN THE AREA EAST OF THE ASPHALT ROAD

Media	Total Samples	Chemicals of Concern	Screening Level (SL)	Samples Above SL	Concentration Range
Surface Soil	23	CaPAHs	10 mg/kg	1	18.3 mg/kg
Shallow Subsurface Soil (near GW3)	10 (composite, 0.5 - 8 feet)	Lead	500 mg/kg	2	504 & 700 mg/kg
		Antimony	20 mg/kg	2	20.1 & 37 mg/kg
		Arsenic	20 mg/kg	1	20.9 mg/kg
		CaPAHs	10 mg/kg	1	16.6 mg/kg
	2 (grab, 5 - 7 feet)	Lead	500 mg/kg	2	781 & 931 mg/kg
Shallow Subsurface Soil (away from GW3)	3 (composite, 0.5 - 8 feet)	Lead	500 mg/kg	2	644 & 1,200 mg/kg
		Arsenic	20 mg/kg	2	22.8 & 33.9 mg/kg
Deep Subsurface Soil (near GW3)	10	Lead	500 mg/kg	8	638 to 2,800 mg/kg
		Antimony	20 mg/kg	5	21.5 to 1,170 mg/kg
		Arsenic	20 mg/kg	2	22.5 & 24.3 mg/kg
		CaPAHs	10 mg/kg	3	19.1 to 37.6 mg/kg
Deep Subsurface Soil (away from GW3)	5	Lead	500 mg/kg	2	909 & 4,860 mg/kg
		Antimony	20 mg/kg	1	34.9 mg/kg
		Arsenic	20 mg/kg	3	20.9 to 412 mg/kg
Groundwater	5 (Phase I)	Lead	25 ug/l	1	170 ug/l
		CaPAHs*	0.002 ug/l	1	13 ug/l
		Benzo(a)pyrene	Nondetect	1	3 ug/l
		Indeno(1,2,3-cd)pyrene	0.002 ug/l	1	0.6 ug/l
	2 (Phase II)	Lead	25 ug/l	1	410 ug/l
		CaPAHs*	0.002 ug/l	1	13 ug/l
		Benzo(a)pyrene	Nondetect	1	4 ug/l
		Indeno(1,2,3-cd)pyrene	0.002 ug/l	1	2 ug/l

* The screening level of 0.002 ug/l pertains to the sum of concentrations for only the following CaPAHs: Benzo(a)anthracene, Chrysene, Benzo(b)flouranthene, and Benzo(k)flouranthene.

Surface Soil--

This area of the site is covered with well-tended low grassland, with little or no exposed soil or debris. Wind erosion causing airborne particles is negligible under current site conditions. Direct contact with the surface soil is possible for on-site individuals who access the area or maintain the grounds (e.g. through mowing or planting). This area is used for parking or recreation during the summer months; however, the extent of this use is unknown. Based on available information, the direct contact pathway for human exposure is considered complete. Wildlife exposure through direct contact also is considered complete, since the grassland provides a favorable habitat and feeding area for voles, rabbits and birds. However, health risks are low due to the relatively low

concentration and limited extent of chemicals of concern in the area, and the low potential for direct human contact.

Future use of the site for residential, recreational, commercial, or industrial purposes is likely to increase the number of individuals accessing this area of the site. If the surface soils remain exposed, the inhalation, ingestion, and direct contact exposure pathways will be functional and complete, with an increased number of individuals at risk of exposure. A future use plan which involves removal of affected soil and replacement with clean soil, or covering surface soils in this site area (for example a parking lot), would render the exposure pathways incomplete.

Shallow Subsurface Soil--

Chemicals of concern in the subsurface soil in this area are not exposed. Therefore, the air pathway and the direct contact pathway for human exposure to subsurface soils are not considered complete under current site conditions. Pathways may become complete if construction or excavation activities that expose contaminated soils are performed at the site. Such exposures can be controlled through informed, limited access to the area and compliance with OSHA personal protection requirements.

The direct contact exposure pathway for wildlife is considered complete, since the area provides a favorable habitat and feeding area for burrowing animals such as voles and rabbits.

The likelihood of the shallow subsurface soils becoming exposed may increase under future use scenarios. Under a residential, commercial, or industrial use scenario, occupants may expose contaminated soil as a result of planting, building, or other projects involving excavation, causing the air and/or direct contact pathways for human exposure to become complete. Again, a future use scenario which removes or decreases accessibility to subsurface soil would decrease significantly the risk of exposure.

Deep Subsurface Soil--

Chemicals of concern in the subsurface soil in this area are not exposed. Therefore, the air pathway and the direct contact pathway for human and wildlife exposure to subsurface soils are not considered complete under current site conditions. Pathways may become complete if construction or excavation activities that expose contaminated soils are performed at the site. Such exposures can be controlled through informed, limited access to the area and compliance with OSHA personal protection requirements.

Groundwater--

The groundwater pathway is not considered complete in the Area East of the Asphalt Road under current site conditions because no known groundwater wells exist in this vicinity, and the habitat in this area is not suitable for burrowing animals which may come in contact with groundwater.

As long as groundwater is not extracted or exposed for any purpose (e.g. constructing a well or unlined pond), the groundwater pathway for human exposure is not likely to become complete under future use scenarios.

3.2.4 Area West of the Asphalt Road

Table 3.5 summarizes the chemicals of concern which exceeded screening levels in the Area West of the Asphalt Road. Surface soils appear to be uncontaminated in this site area except in isolated samples collected adjacent to a large gravel area, which showed lead and CaPAHs above screening levels.

A total of 19 shallow subsurface samples were collected in the Area West of the Asphalt Road (all Phase I samples). Six of these samples were collected in the vicinity of monitoring well No. 9 (GW9) to investigate elevated levels of lead detected in earlier investigations. No chemicals of concern were detected in any of the shallow subsurface samples taken near GW9. Lead and total CaPAHs exceeded screening levels in a few shallow subsurface samples located away from GW9.

Eighteen deep subsurface soil samples were collected during Phase I, and two were collected during Phase II. In the area of GW9, three of the Phase I samples contained chemicals of concern; the chemicals which exceeded screening levels included total CaPAHs, arsenic, and lead. Lead is the only chemical of concern which was analyzed in the two Phase II samples; lead exceeded screening levels in both samples. TCLP constituents did not exceed maximum allowable levels for either of these Phase II samples. Several samples collected away from GW9 contained lead, antimony, arsenic, and/or CaPAHs at concentrations above screening levels.

Twelve samples were collected from wells in the Area West of the Asphalt Road during Phase I, and six samples were collected during Phase II. In Phase I samples, Antimony exceeded screening levels in all three wells within the Monitoring Well 8 cluster near the shoreline. Antimony exceeded the screening level in the intermediate depth well in this cluster during Phase II. Cyanide exceeded the screening level in monitoring well No. 13 during Phase I, and in two wells further North along the shoreline during Phase II; all three are shallow wells.

Table 3.5

CHEMICALS OF CONCERN IN THE AREA WEST OF THE ASPHALT ROAD

Media	Total Samples	Chemicals of Concern	Screening Level (SL)	Samples Above SL	Concentration Range
Surface Soil	34	Lead	500 mg/kg	2	554 & 815 mg/kg
		CaPAHs	10 mg/kg	3	10.8 to 18 mg/kg
Shallow Subsurface Soil (near GW9)	6 (composite, 0.5 - 8 feet)	none			
Shallow Subsurface Soil (away from GW9)	13 (composite, 0.5 - 8 feet)	Lead	500 mg/kg	1	1,160 mg/kg
		CaPAHs	10 mg/kg	3	12.2 to 18.4 mg/kg
Deep Subsurface Soil (near GW9)	8	Lead	500 mg/kg	5	876 to 1,630 mg/kg
		Arsenic	20 mg/kg	3	24.2 to 45.6 mg/kg
		CaPAHs	10 mg/kg	3	25.6 to 38.2 mg/kg
Deep Subsurface Soil (away from GW9)	12	Lead	500 mg/kg	3	1,040 to 2,850 mg/kg
		Antimony	20 mg/kg	2	28.1 & 89.1 mg/kg
		Arsenic	20 mg/kg	3	24.4 to 57.8 mg/kg
		CaPAHs	10 mg/kg	2	12.2 & 21.3 mg/kg
Groundwater	12 (Phase I)	Antimony	3 ug/l	3	77.1 to 124 ug/l
		Cyanide	100 ug/l	1	259 ug/l
	6 (Phase II)	Antimony	3 ug/l	1	26.7 ug/l
		Cyanide	100 ug/l	2	715 & 892 ug/l

Surface Soil--

The section of this site area near the Bell Slip to the south is well vegetated with low and high grasses and some trees, and provides a favorable habitat for a variety of animals on site. Little surface soil contamination was detected in this part of the site. The northern part of this site area contains some exposed debris piles and sparsely vegetated areas, as well as large gravel areas. Wind erosion and vehicular traffic may cause air transport of soil and dust particles in these areas, and potential inhalation and/or ingestion of these particles by on-site and off-site individuals and wildlife provides a complete pathway. A complete pathway also exists for direct soil contact by on-site individuals who access this area of the site for parking or recreation, and by wildlife foraging or living in this area.

Future use of the site for residential, recreational, commercial, or industrial purposes is likely to increase the number of individuals accessing this area of the site. If the surface soils remain exposed, the inhalation, ingestion, and direct contact exposure pathways will be functional and complete, with an increased number of individuals at risk of exposure. A future use plan which involves removal of the affected soil and replacement with clean soil, or covering surface soils in this site area (for example a parking lot), would render the exposure pathways incomplete.

Shallow Subsurface Soil--

Chemicals of concern in the subsurface soil in this area were detected at depths of 0.5 to 8 feet and are not exposed. Therefore, the air pathway and the direct contact pathway for subsurface soil exposure are not considered complete under current site conditions. Pathways may become complete if construction or excavation activities that expose contaminated soils are performed at the site. Such exposures can be controlled through informed, limited access to the area and compliance with OSHA personal protection requirements.

As discussed regarding subsurface soils in the Area South of the Bell Slip, the area around the Bell Slip is a favorable muskrat habitat. No subsurface sampling was performed in the immediate area on the north side of the Bell Slip except at the edge of the harbor, where the shoreline is stabilized with rip/rap (where elevated CaPAHs were detected in shallower subsurface soil). This exposure pathway can be considered complete, but actual risks are unknown due to the limited sampling along the Bell Slip. The direct contact exposure pathway for other wildlife also is considered complete, since the area provides a favorable habitat and feeding area for burrowing animals such as voles and rabbits.

The likelihood of the shallow subsurface soils becoming exposed may increase under future use scenarios. Under a residential, commercial, or industrial use scenario, occupants may expose contaminated soil as a result of planting, building, or other excavation projects, causing the air and/or direct contact pathways for human exposure to become complete. Again, a future use scenario which removes or decreases accessibility to subsurface soil would decrease significantly the risk of exposure.

Deep Subsurface Soil--

Chemicals of concern in the subsurface soil in this area were detected at depths of 8 to 27 feet and are not exposed. Therefore, the air pathway and the direct contact pathway for subsurface soil exposure are not considered complete under current site conditions. Pathways may become complete if construction or excavation activities that expose contaminated soils are performed at the site. Such exposures can be controlled through informed, limited access to the area and compliance with OSHA personal protection requirements.

Groundwater--

The groundwater pathway is not considered complete in the Area South of the Bell Slip under current site conditions because no known potable water supply wells exist in the vicinity of the site. The wildlife exposure pathway also is considered incomplete because rip/rap along the shoreline prevents wildlife from burrowing into the area along the shoreline where chemicals of concern were detected.

As indicated in the RI, shallow groundwater flows toward the Outer Harbor/Bell Slip from wells along the shoreline in this site area. Groundwater containing antimony and cyanide could discharge to surface water in this area. However, antimony and cyanide were undetected in sediment and surface water samples from this area of the Outer Harbor. Therefore, this potential exposure pathway also is considered incomplete.

As long as groundwater is not extracted or exposed for any purpose (e.g. constructing a well or unlined pond), the pathway for human exposure to chemicals of concern in the groundwater or through volatilization of cyanide from groundwater is not likely to become complete under future use scenarios.

3.2.5 Eastern Portion of the Gravel Parking Area

As shown in Table 3.6, only surface soil and groundwater samples were collected from the Eastern Portion of the Gravel Parking Area. Four surface soil samples were collected during Phase I sampling, and ten soil samples were collected during Phase II. PCBs, lead, and CaPAHs exceeded screening levels in multiple surface soil samples. Lead concentrations also exceeded the screening level in the groundwater sample during both Phases of sampling, and chrysene exceeded the screening level during Phase II sampling.

Table 3.6

CHEMICALS OF CONCERN IN THE EASTERN PORTION OF THE GRAVEL PARKING LOT

Media	Total Samples	Chemicals of Concern	Screening Level (SL)	Samples Above SL	Concentration Range
Surface Soil	14	PCBs	1 mg/kg	2	4.9 & 12 mg/kg
		Lead	500 mg/kg	6*	505 to 14,000 mg/kg
		Antimony	20 mg/kg	1	24.4 mg/kg
		Arsenic	20 mg/kg	1	21.3 mg/kg
		CaPAHs	10 mg/kg	3	10.2 to 14.8 mg/kg
Groundwater	1 (Phase I)	Lead	25 ug/l	1	45.4 ug/l
	1 (Phase II)	Lead	25 ug/l	1	500 ug/l
		Chrysene**	0.002 ug/l	1	0.6 ug/l

* Lead concentrations ranged from 505 to 2,040 mg/kg for five samples; the sixth sample, with a concentration of 14,000 mg/kg, was taken from an area where the soil was visibly stained and open oil containers had been discarded.

** The screening level of 0.002 ug/l pertains to the sum of concentrations for only the following CaPAHs: Benzo(a)anthracene, Chrysene, Benzo(b)flouranthene, and Benzo(k)flouranthene.

Surface Soil--

This area of the site is similar to the adjacent area West of the Asphalt Road, with some low grassland as well as a large gravel area. Wind erosion and vehicular traffic can cause air transport of soil and dust particles in this area, and the potential inhalation and/or ingestion of these particles by on-site and off-site individuals and wildlife provides a complete pathway. A complete pathway also exists for direct soil contact by on-site individuals who access this area of the site for parking or recreation.

Future use of the site for residential, recreational, commercial, or industrial purposes is likely to increase the number of individuals accessing this area of the site. If the surface soils remain exposed, the inhalation, ingestion, and direct contact exposure pathways will be functional and complete, with an increased number of individuals at risk of exposure. A future use plan which involves removal of the affected soil and replacement with clean

soil, or covering surface soils in this site area (for example a parking lot), would render the exposure pathways incomplete.

Groundwater--

The groundwater pathway is not considered complete in the Eastern Portion of the Gravel Parking Area under current site conditions because no known potable water supply wells exist in the vicinity of the site, and the habitat in this area is not suitable for burrowing animals which may come in contact with groundwater. Moreover, groundwater elevation measurements in wells GW1 and PZ9 suggest that the depth to groundwater is probably over 10 feet, below the habitat of burrowing animals.

As long as groundwater is not extracted or exposed for any purpose (e.g. constructing a well or unlined pond), the groundwater pathway for human exposure is not likely to become complete under future use scenarios.

3.2.6 Sediment

Table 3.7 summarizes the chemicals of concern detected in the 16 sediment samples collected from the slips and off-shore areas of the Buffalo Outer Harbor Site during Phase I. Lead concentrations exceeded the severe effect level (SEL) in a sample near the Allen Boat Company and in another sample off-shore at the south end of the site.

NYSDEC has not defined screening criteria for total CaPAHs in sediment. However, in one sample from the Bell Slip area near the Allen Boat Company, total CaPAHs detected were almost 15 ug/kg, over twice the concentration detected in the background sediment sample from the Outer Harbor. CaPAHs are prevalent in surface soils near the Bell Slip, and CaPAHs adsorbed to soil particles contained in surface runoff may have been transported to the sediments.

Table 3.7

CHEMICALS OF CONCERN IN SEDIMENT

Media	Total Samples	Chemicals of Concern	Screening Level (SL)	Above SL	Concentration Range
Sediment	16	Lead	110 mg/kg	2	326 & 339 mg/kg
		CaPAHs	none	3**	4.8 to 14.7 mg/kg

** Number of samples in which chemical was detected (no screening levels specified in the RI).

Surface water depth in the Bell Slip Area is relatively shallow. At the location where CaPAHs and lead were detected in sediment, water depth recorded during the sampling event was seven feet. Swimming and boating in the area may disturb the sediment, causing chemicals adsorbed to sediment to become suspended in the water column.

Lead and CaPAHs in the sediments provide a complete exposure pathway for humans swimming or boating in the area who ingest water containing suspended sediments. The exposure pathway is also complete for wildlife which ingest suspended particles and feed from sediments. Since bioconcentration of CaPAHs in fish may occur, and fishing has been observed in the area, a complete exposure pathway also exists for humans through ingestion of contaminated fish.

SECTION 4

SUMMARY AND CONCLUSIONS

The objective of the qualitative risk assessment is to identify potential human and wildlife exposure pathways which may be of concern at the Buffalo Outer Harbor Site. Each exposure pathway was evaluated for functionality and completeness. Functional pathways were identified based on the potential existence of a physical mechanism by which contaminants can be transported into the environment. From among those pathways considered functional, complete pathways were identified on the basis of a potential receptor population and a potential exposure and uptake mechanism. For each site area, a summary table is presented below which lists the waste sources present in the area, as well as the exposure pathways, potential receptors, and pathway status.

Based on the criteria used to identify contaminants of concern which may pose health risks and the analysis of functional and complete pathways at the site, a number of areas are of concern. These areas are discussed below.

4.1 RADIO TOWER AREA

A summary of the exposure pathways for the existing site conditions in the Radio Tower Area is presented in Table 4.1.

Current levels and extent of PCBs and lead in surface soil pose a health risk to humans and wildlife on site, and to a lesser degree off site. Although current access to this area by humans is relatively infrequent, human access may increase under future use scenarios for the site. These soils should be considered for remediation.

Under the current site conditions, chemicals of concern detected in the subsurface soil and groundwater pose a low risk to humans or wildlife due to low potential for exposure. Metals detected in the subsurface soil have little mobility. However, nitrobenzene was detected in high concentrations and was shown to be leachable. Benzene and 4-Chloroaniline were detected at high concentrations in groundwater. Although no current, known uses of groundwater exist in the site area, remediation of the contaminated soils in the Radio Tower Area should be considered to remediate this source of groundwater contamination.

If future land use considerations will increase human utilization of other areas of the site, access to this area may be restricted, or containment or treatment measures may be undertaken to limit potential exposure. In addition, before commencing any excavation or other activities which may cause exposure to subsurface soil or groundwater from this area, involved parties should be informed of the appropriate safety precautions and personal protection requirements.

Table 4.1

SUMMARY OF EXPOSURE PATHWAYS FOR THE RADIO TOWER AREA

Waste Source	Release Mechanism	Transport Mechanism	Exposure/ Uptake	Potential Receptors	Status*	Rationale
Contaminants include: *PCBs* - surface soils. *Lead* - surface soils, shallow and deep subsurface soils. *Antimony* - shallow and deep subsurface soils. *CaPAHs* - shallow subsurface soils. *Nitrobenzene* - deep subsurface soil. *Benzene* - deep subsurface soil, groundwater. *4-Chloroaniline* - deep subsurface soil, groundwater.	Volatilization	Air	Inhalation, dermal absorption	Humans	Incomplete	Excavation of contaminated soils may result in release of vapors from volatile chemicals of concern.
				Wildlife	Incomplete	
	Wind erosion (dust)	Air	Inhalation	Humans	Complete	Exposed debris piles and surface soil may result in airborne-particulate bound contaminants
				Wildlife	Complete	
	Direct contact	Surface soil	Dermal absorption, ingestion	Humans	Complete	Access to the site is not restricted. Debris piles and surface soil are exposed. Animals and humans may contact contaminated soil on-site.
				Wildlife	Complete	
	Direct contact	Shallow subsurface soil	Dermal absorption, ingestion	Humans	Incomplete	Burrowing animals may contact contaminated materials. Subsurface soil contact by humans could occur during construction or as a result of erosion.
				Wildlife	Complete	
	Direct contact	Deep subsurface soil	Dermal absorption, ingestion	Humans	Incomplete	Subsurface soil contact by humans and wildlife could occur during construction.
				Wildlife	Incomplete	
	Direct leaching to groundwater from soils	Groundwater	Ingestion, inhalation, and dermal absorption	Humans	Incomplete	No known potable water supply wells exist in the vicinity of the site. Animal habitats are above the water table in this area.
				Wildlife	Incomplete	

* Based on current use of the area.

Table 4.3

SUMMARY OF EXPOSURE PATHWAYS FOR AREA EAST OF ASPHALT ROAD

Waste Source	Release Mechanism	Transport Mechanism	Exposure/ Uptake	Potential Receptors	Status*	Rationale
Contaminants include: *Lead* - shallow and deep subsurface soils, groundwater. *Antimony* - shallow and deep subsurface soils. *Arsenic* - shallow and deep subsurface soils. *CePAHs* - surface soils, shallow and deep subsurface soils, groundwater.	Wind erosion (dust)	Air	Inhalation	Humans	Complete	Exposed debris piles and surface soil may result in airborne-particulate bound contaminants
				Wildlife	Complete	
	Direct contact	Surface soil	Dermal absorption, ingestion	Humans	Complete	Access to the site is not restricted. Debris piles and surface soil are exposed. Animals and humans may contact contaminated soil on-site.
				Wildlife	Complete	
	Direct contact	Shallow subsurface soil	Dermal absorption, ingestion	Humans	Incomplete	Burrowing animals may contact contaminated materials. Subsurface soil contact by humans would occur during construction or as a result of erosion events.
				Wildlife	Complete	
	Direct contact	Deep subsurface soil	Dermal absorption, ingestion	Humans	Incomplete	Subsurface soil contact by humans and wildlife could occur during construction.
				Wildlife	Incomplete	
	Direct leaching to groundwater from soils	Groundwater	Ingestion, inhalation, and dermal absorption	Humans	Incomplete	No known potable water supply wells exist in the vicinity of the site. Animal habitats are above the water table in this area.
				Wildlife	Incomplete	

* Based on current use of the area.

4.2 REMAINING AREA SOUTH OF THE BELL SLIP

A summary of the exposure pathways for the existing site conditions in the Remaining Area South of the Bell Slip is presented in Table 4.2.

Table 4.2

SUMMARY OF EXPOSURE PATHWAYS FOR REMAINING AREA SOUTH OF THE BELL SLIP

Waste Source	Release Mechanism	Transport Mechanism	Exposure/ Uptake	Potential Receptors	Status*	Rationale
Contaminants include: *Lead* - surface soils, shallow and deep subsurface soils. *Antimony* - deep subsurface soils. *Arsenic* - surface soils, shallow and deep subsurface soils, groundwater. *CePAHs* - surface soils, shallow and deep subsurface soils.	Wind erosion (dust)	Air	Inhalation	Humans	Complete	Exposed debris piles and surface soil may result in airborne-particulate bound contaminants
				Wildlife	Complete	
	Direct contact	Surface soil	Dermal absorption, ingestion	Humans	Complete	Access to the site is not restricted. Debris piles and surface soil are exposed. Animals and humans may contact contaminated soil on-site.
				Wildlife	Complete	
	Direct contact	Shallow subsurface soil	Dermal absorption, ingestion	Humans	Incomplete	Burrowing animals may contact contaminated materials. Subsurface soil contact by humans would occur during construction or as a result of erosion events.
				Wildlife	Complete	
	Direct contact	Deep subsurface soil	Dermal absorption, ingestion	Humans	Incomplete	Subsurface soil contact by humans and wildlife could occur during construction.
				Wildlife	Incomplete	
	Direct leaching to groundwater from soils	Groundwater	Ingestion, inhalation, and dermal absorption	Humans	Incomplete	No known potable water supply wells exist in the vicinity of the site. Animal habitats are above the level at which groundwater contamination was observed in this area.
				Wildlife	Incomplete	

* Based on current use of the area.

Small sections of elevated CaPAH concentrations in surface soil are distributed throughout the Area South of the Bell Slip. Due to the carcinogenicity of these compounds and the frequency of human exposure through the Allen Boat Company or for recreational use of the Bell Slip, these soils should be considered for remediation. Soils in a different section of the Remaining Area South of the Bell Slip which contained elevated arsenic concentrations also should be considered for remediation due to their proximity to Fuhrmann Boulevard and the high potential for human exposure.

Under the current site conditions of limited potential for human or wildlife exposure to subsurface soils or groundwater, chemicals detected in these media do not warrant remediation. Again, excavation or construction activities in this area should be conducted using appropriate precautions to limit exposure to the chemicals of concern.

4.3 AREAS EAST AND WEST OF THE ASPHALT ROAD

A summary of the exposure pathways for the existing site conditions for the Area East of the Asphalt Road is presented in Table 4.3. The corresponding summary for the Area West of the Asphalt Road is presented in Table 4.4

Four localized areas of elevated CaPAH concentrations in surface soil were found in the Areas East and West of the Asphalt Road. Due to the carcinogenicity of these compounds and the frequency of human exposure for recreational use, these soils should be considered for remediation. CaPAHs were detected in several shallow subsurface samples, but these locations do not appear to correlate with CaPAHs found in surface soils.

Under the current site conditions of limited potential for human or wildlife exposure to subsurface soils or groundwater, chemicals detected in these media do not warrant remediation. Again, excavation or construction activities in this area should be conducted using appropriate precautions to limit exposure to the chemicals of concern.

Table 4.4

SUMMARY OF EXPOSURE PATHWAYS FOR AREA WEST OF ASPHALT ROAD

Waste Source	Release Mechanism	Transport Mechanism	Exposure/ Uptake	Potential Receptors	Status*	Rationale
Contaminants include: *Lead* - surface soils, shallow and deep subsurface soils. *Antimony* - deep subsurface soils, groundwater. *Arsenic* - deep subsurface soils. *CaPAHs* - surface soils, shallow and deep subsurface soils. *Cyanide* - groundwater.	Wind erosion (dust)	Air	Inhalation	Humans	Complete	Exposed debris piles and surface soil may result in airborne-particulate bound contaminants
				Wildlife	Complete	
	Direct contact	Surface soil	Dermal absorption, ingestion	Humans	Complete	Access to the site is not restricted. Debris piles and surface soil are exposed. Animals and humans may contact contaminated soil on-site.
				Wildlife	Complete	
	Direct contact	Shallow subsurface soil	Dermal absorption, ingestion	Humans	Incomplete	Burrowing animals may contact contaminated materials. Subsurface soil contact by humans would occur during construction or as a result of erosion events.
				Wildlife	Complete	
	Direct contact	Deep subsurface soil	Dermal absorption, ingestion	Humans	Incomplete	Subsurface soil contact by humans and wildlife could occur during construction.
				Wildlife	Incomplete	
	Volatilization from groundwater	Groundwater	Inhalation, dermal absorption	Humans	Incomplete	No known potable water supply wells exist in the vicinity of the site.
				Wildlife	Incomplete	
	Direct leaching to groundwater from soils	Groundwater	Ingestion, inhalation, and dermal absorption	Humans	Incomplete	No known potable water supply wells exist in the vicinity of the site. Rip/rap along the shoreline prevents wildlife from burrowing into the area along the shoreline in which groundwater contamination was observed in this area.
				Wildlife	Incomplete	

* Based on current use of the area.

4.4 EASTERN PORTION OF THE GRAVEL PARKING AREA

Exposure pathways for the Eastern Portion of the Gravel Parking Area are summarized in Table 4.5. PCBs, lead, arsenic, and CaPAHs were detected at elevated levels in these surface soils. The potential for human exposure to these chemicals is high under current and proposed future use scenarios. Therefore, measures should be taken to remediate the surface soil in this small area.

Table 4.5

SUMMARY OF EXPOSURE PATHWAYS FOR EASTERN PORTION OF THE GRAVEL PARKING AREA

Waste Source	Release Mechanism	Transport Mechanism	Exposure/ Uptake	Potential Receptors	Status*	Rationale
Contaminants include: *PCBs* - surface soil. *Lead* - surface soil, groundwater. *Antimony* - surface soil. *Arsenic* - surface soil. *CaPAHs* - surface soil, groundwater.	Wind erosion (dust)	Air	Inhalation	Humans	Complete	Exposed debris piles and surface soil may result in airborne-particulate bound contaminants
				Wildlife	Complete	
	Direct contact	Surface soil	Dermal absorption, ingestion	Humans	Complete	Access to the site is not restricted. Debris piles and surface soil are exposed. Animals and humans may contact contaminated soil on-site.
				Wildlife	Complete	
	Direct leaching to groundwater from soils	Groundwater	Ingestion, inhalation, and dermal absorption	Humans	Incomplete	No known potable water supply wells exist in the vicinity of the site. Animal habitats are above the water table in this area.
				Wildlife	Incomplete	

* Based on current use of the area.

4.5 SEDIMENTS

Table 4.6 provides a summary of pathways for exposure to sediments. The potential exposure to lead and CaPAHs in sediments in the Bell Slip area may present a health risk to humans and wildlife. The prevalence of these chemicals in other areas of the Bell Slip and on the shoreline is unknown due to limited sampling. Since dermal exposure as well as ingestion and inhalation of lead and CaPAHs may produce toxic effects, recreational uses (e.g., beach and swimming area) in the Bell Slip area are not recommended. Remediation of site surface soils containing lead and CaPAHs may remove the source of these chemicals in sediments. Because sediments containing chemicals of concern were found in the Bell Slip area, the Allen Boat Company operations may be a source of these chemicals.

Table 4.6

SUMMARY OF EXPOSURE PATHWAYS FOR SEDIMENTS

Waste Source	Release Mechanism	Transport Mechanism	Exposure/ Uptake	Potential Receptors	Status*	Rationale
Contaminants include: *Lead* *CaPAHs*	Indirect uptake	Contaminated biota	Ingestion	Humans	Complete	Bioconcentration of CaPAHs in fish may occur. Fishing occurs in the area.
				Wildlife	Complete	
	Direct contact	Sediments	Dermal absorption, ingestion	Humans	Complete	Access to the Bell Slip area is not restricted. Animals and humans may contact contaminated sediment.
				Wildlife	Complete	

* Based on current use of the area.

APPENDIX A

TOXICITY PROFILES FOR CHEMICALS OF CONCERN

POLYCHLORINATED BIPHENYLS

RN - 1336-36-3
SY - Chlorinated diphenyl
SY - PCB
SY - 1,1'-Biphenyl, chloro derivs
SY - Caswell no 672A
SY - EPA pesticide chemical code 017801
SY - Clophen
SY - Kanechlor
SY - Aroclor
SY - Fenclor
SY - Chlorextol
SY - Inerteen
SY - Monter
SY - Phenoclor
SY - Pyralene
SY - Pyranol
MP - 340 to 375 deg C [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 2249]
DEN - 1.44 at 30 deg C [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 2249]
OCPP - Solubility in water is extrememly low; soluble in oils and organic solvents. [IARC MONOGRAPHS. 1972-PRESENT V18 49 (1978)]
OCPP - With the exception of Aroclors 1221 and 1268, Aroclors do not crystallize upon heating or cooling, but at a specific temperature, defined as a "pour point", change into a resinous state. [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.A-3 (1980) EPA 440/5-80-068]
SSL - ... PCB's are chemically very inert and are stable to conditions of hydrolysis and oxidation in industrial use. [IARC MONOGRAPHS. 1972-PRESENT V18 52 (1978)]
CAREV- CLASSIFICATION: B2; probable human carcinogen. BASIS FOR CLASSIFICATION: Hepatocellular carcinomas in three strains of rats and two strains of mice and inadequate yet suggestive evidence of excess risk of liver cancer in humans by ingestion and inhalation or dermal contact. HUMAN CARCINOGENICITY DATA: Inadequate. ANIMAL CARCINOGENICITY DATA: Sufficient. [U.S. Environmental Protection Agency's Integrated Risk Information System (IRIS) on Polychlorinated biphenyls (PCBs) (1336-36-3) from the National Library of Medicine's TOXNET System, November 1, 1994]

o EMCE - CLINICAL EFFECTS :

SUMMARY

- o PCBs have low acute toxicity, but because they accumulate in the environment and in animal and human tissues, the potential for chronic or delayed toxicity is significant.

HEENT

- o PCBs are mildly irritating to eyes and skin. Possible symptoms include facial edema, eye discharge, swollen eyelids, conjuctival hyperemia, and visual and hearing disturbances.

CARDIOVASCULAR

- o Increases in diastolic and systolic blood pressure are possible.

NEUROLOGIC

- o Weakness and numbness of the extremities has been reported.
- o Neurobehavioral and psychomotor impairment have been seen after occupational and in utero exposure.

GASTROINTESTINAL

- o GI upset and diarrhea have been seen.

HEPATIC

- o Liver damage is a consistent finding in animal studies. Clinical hepatitis was seen in the Yusho epidemic. Consistent documentation of elevated hepatic enzymes after chronic exposure has been difficult to demonstrate.

METABOLISM

- o PCBs are potent enzymes inducers, more potent than phenobarbital.

DERMATOLOGIC

- o Chloracne is a specific skin reaction associated with cyclic halogenated compounds.

ENDOCRINE

- o Asymptomatic hyperthyroxinemia has been reported.

PREGNANCY/BREAST MILK

- o Some evidence of teratogenicity does exist.

CARCINOGENICITY

- o PCBs are clearly carcinogens in animals, producing liver tumors in rats. Although human studies have not been conclusive, PCBs should be considered potential human carcinogens.

o EMTOX- RANGE OF TOXICITY :

- o Toxicity of the compounds may vary by impurities present. Polychlorinated dibenzofurans and other highly toxic compounds have been found as contaminants. Symptoms are expected after a minimal oral intake of 500 mg. In the Yusho experience, the average Japanese intake was 72.4 mcg/day.

HTOX - Deaths that occurred up to 5 1/2 yr after first exposure to PCB's ... were reported. Nine (41%) of 22 deaths were due to malignant neoplasms. Three of the tumors occurred in the stomach, one in the liver, two in the lung and one in the breast, and two were malignant lymphomas. (Kuratsune, 1976; Omae, 1975) [IARC MONOGRAPHS. 1972-PRESENT V18 82 (1978)]

HTOX - A significant correlation was found between plasma levels of polychlorinated biphenyls(PCBs) in mothers occupationally exposed to these cmpd and the PCB milk levels. It has been observed that if these mothers nursed their babies for more than three months, the PCB levels in the infants exceeded that of their mothers. These cmpd were subsequently retained in the children for many years. ... [ENCYC OCCUPAT HEALTH & SAFETY 1983 , p. 1755]

HTOX - /RESPONSES TO PCBS/: ACNE; HYPERPIGMENTATION OF SKIN; HYPERACTIVE MEIBOMIAN GLANDS; CONJUNCTIVITIS; EDEMA OF EYELIDS; SUBCUTANEOUS EDEMA; KERATIN CYSTS IN HAIR FOLLICLES; HYPERPLASIA OF HAIR FOLLICLE EPITHELIUM; HEPATIC HYPERTROPHY; DECR NUMBER OF RED BLOOD CELLS; DECR HEMOGLOBIN; SERUM HYPERLIPIDEMIA; LEUCOCYTOSIS. [IARC MONOGRAPHS. 1972-PRESENT V18 70 (1978)]

HTOX - A leaking heat exchanger in a chemical plant discharged polychlorinated biphenyl (PCB) vapors. No employees worked routinely at the point of leakage, but breathing zone levels in work areas were found to be 0.1 mg/cu m. The period of exposure was 19 months. Seven of 14 exposed workers developed mild to moderate chloracne after exposure durations of 5-14 months. Liver function tests showed normal serum bilirubin, 24 and 48 hr cephalin flocculations, thymol turbidities, and serum alkaline phosphatase activities in six of the seven workers, but borderline increases in cephalin flocculation and thymol turbidity in the seventh. After 13 months, the thymol turbidity but not the cephalin flocculation had improved. [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.C-56 (1980) EPA 440/5-80-068]

HTOX - An analysis of the health effects of polychlorinated biphenyls (PCB) on eight laboratory workers involved in testing dielectric fluids was made. ... The workers, all males 25 to 49 yr of age, had been employed 2.5 to 18 yrs. Breathing zone, point source, and general work area samples were collected on three separate occasions. The ranges were: breathing zone, 0.014 to 0.073 mg/cu m; point source (near an oven), 0.042 to 0.264 mg/cu m; and room area, 0.013 to 0.15 mg/cu m. Blood concns were 36 to 286 ppb which is substantially above the range in several studies of general populations. Workers complained of dry, sore throat (6/8), skin rash (3/8), gastrointestinal disturbances (3/8), and eye irritation and headache (2/8). Examination disclosed one patient with skin rash, two with nasal irritation, one showing rales, and four with high blood pressure, but no abnormalities in liver function. [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.C-55 (1980) EPA 440/5-80-068]

HTOX - Irregular menstrual cycles, early abortions and the birth of small, hyperpigmented and hyperkeratotic infants have been observed. [IARC MONOGRAPHS. 1972-PRESENT V18 37 (1978)]

HTOX - Spirometric findings in a retrospective cohort of 136 capacitor workers with occupational exposure to polychlorinated biphenyls (PCBs) during active use (1976) and after the PCB ban (1979 and 1983) are reported. Quantitative exposure levels are not known. Subjects were categorized as having high, medium, or low exposure depending primarily on the extent of dermal contact. Mean 1979 serum PCB levels were elevated 35 to 40 times the normal level. Duration of employment ranged from 1-35 yr. Obstructive impairment was consistently found in 15% of the workers in 1976 and 1979. A history of respiratory illness and reduced FEV 1/FVC was correlated in a dose-response fashion with PCB exposure category and serum PCB levels in females in 1976 but the association disappeared in 1979. It is not clear whether the association held when controled for

smoking. There was no association between PCB exposure and abnormal pulmonary function tests in males. [Lawton R et al; J Occup Med 28 (6): 453-6 (1986)]

HTOX - PCBS ARE LIVER TOXINS & CAUSE CHLORACNE & POSSIBLY PERIPHERAL NEUROPATHY IN MAN. [NRC. DRINKING WATER & HEALTH 1977 , p. 757]

HTOX - The first documentation of human effects as a result of ingestion of PCBs was derived from the Japanese poisoning incident that occurred in 1968. The victims suffered an acute toxicosis from consuming rice oil contaminated with an industrial oil, Kanechlor-400, consisting of a mixture of polychlorinated biphenyls (PCB), polychlorinated dibenzofurans (PCDF), and polychlorinated quinones (PCQ). The average total amount of PCBs consumed was estimated to be approximately 2 g, with approximately 0.5 g being the least total amount consumed by an affected group of some 325 people at the time. ... The most notable symptoms of Yusho among 189 patients included dark brown pigmentation of nails and skin, follicular accentuation, acneform eruptions, increased eye discharge, increased sweating at the palms and feeling of weakness. ... [Kuratsune M et al; Environ Health Perspect 1: 119-28 (1972) as cited in USEPA; Drinking Water Qual Crit Doc: Polychlorinated Biphenyls (PCBs) ECAO-CIN-414 p.VI-15 (1987)]

HTOX - A mass outbreak of a peculiar skin disease /including pigmentation and acne from eruptions/ was recorded in Taichung and Changwa in Central Taiwan. The cause of the disease was later identified to be the ingestion of rice bran oil contaminated with polychlorinated biphenyls (PCBs), and there were > 1900 victims. Blood PCB levels of 66 affected persons ranged from 11-720 ppb (mean 49 ppb) at approx 9-12 months after consumption of the PCB-contaminated oil. [Chen PH et al; Bull Environ Contam Toxicol 25: 325-9 (1980) as cited in USEPA; Drinking Water Qual Crit Doc: Polychlorinated Biphenyls (PCBs) ECAO-CIN-414 p. VI-14 (1987)]

HTOX - Polychlorinated biphenyl blood residues were measured in 29 infertile males and in 14 matched control subjects at a hospital in Jerusalem, Israel. The patients' ages ranged from 25 to 45 years. The patients exhibited one or more impaired semen characteristics such as decreased spermatozoa count, lower sperm motility, or a greater proportion of morphologically abnormal spermatozoa. The control group, matched by age and smoking habits, consisted of randomly selected patients with minor illnesses. Each of them had at least one child not older than two years of age. None of the subjects had a history of occupational exposure to organochlorine compounds. The polychlorinated biphenyl levels were measured by GC-ECD. The mean concentration of total polychlorinated biphenyls in the infertile patients was 11.21 +/- 13.48 ng/g blood serum (range 0 to 64.2 ng/g). The control subjects had a mean concentration of 7.94 +/- 14.69 ng/g (range 0 to 47.3 ng/g). [Pines A et al; Arch Environ Contam Toxicol 16: 587-597 (1987)]

HTOX - A retrospective cohort mortality study of workers exposed to polychlorinated biphenyl (PCBs) in two plants manufacturing electrical capacitors was reported in 1981. The study was conducted primarily to examine the risk of cancer mortality associated with exposure to PCBs: due to the availability of animal data, liver cancer was the disease of most interest. Because of the small number of deaths and a relatively short observation period the study was inconclusive. Therefore, the study was updated by adding 7 yr of observation increasing the number of deaths in the study cohort from 163 to 295. Mortality from all causes was found to be lower than expected (295 observed versus 318 expected deaths) as well as mortality from all cancers (62 observed versus 80 expected deaths). A statistically significant excess in deaths was observed in the category that includes cancer of the liver (primary and unspecified), gall bladder, and biliary tract (5 observed versus 1.9 expected; $p < 0.05$). Most of this excess was observed in women employed in one plant. [Brown DP; Arch Environ Health 42 (6): 333-9 (1987)]

HTOX - The possibility of polychlorinated biphenyl-induced porphyria after transplacental exposure was investigated using children born to mothers exposed to contaminated rice oil in central Taiwan in 1979. The exposure was to a mixture of thermally degraded polychlorinated biphenyls, polychlorinated quaterphenyls, & polychlorinated dibenzofurans, which had become mixed with the oil during processing. Women who became pregnant had children with high perinatal mortality and a dysmorphic syndrome. Seventy four controls and 12 siblings of the exposed children were included in the study. Four of the transplacentally exposed children, 2 controls and 1 sib had a type B hepatic porphyria; total porphyrin excretion was elevated in the exposed children as a group (95 ug/l, exposed; 81 ug/l, control); and 8 of the 75 exposed children and 2 controls had total urinary porphyrin concentrations of >200 ug/l. The children did not appear to have symptoms directly attributable to porphyria, but a mild disturbance in porphyrin metabolism appeared to be related to their intrauterine exposure. [Gladen BC et al; Arch Environ Health 43 (1): 54-8 (1988)]

HTOX - People occupationally exposed to PCB's have relatively high PCB residue levels. [ENCYC OCCUPAT HEALTH & SAFETY 1983 , p. 1753]

HTOX - The Carcinogen Assessment Group (CAG), Office of Health and Environmental Assessment in EPA'S Research and Development Office, has prepared a list of chemical substances for which substantial or strong evidence exists showing that exposure to these chemicals, under certain conditions, causes cancer in humans, or can cause cancer in animal species which in turn, makes them potentially carcinogenic in humans. Substances are placed on the CAG list only if they have been demonstrated to induce malignant tumors in one or more animal species or to induce benign tumors that are generally recognized as early stages of malignancies, and/or if positive epidemiologic studies indicated they were carcinogenic. Polychlorinated biphenyls are on that list. [USEPA/CAG; The Carcinogen Assessment Group's List of Carcinogens (7/14/80)]

HTOX - Digestive symptoms such as abdominal pain, anorexia, nausea, vomiting, jaundice, with rare cases of coma and death, may occur. At autopsy, acute yellow atrophy of the liver was found in lethal cases. ... Neurological symptoms such as headache, dizziness, depression, nervousness ... and other symptoms such as fatigue, loss of weight, loss of libido and muscle and joint pains were found in various percentages of exposed people. ... By the study of PCB-associated diseases in the general population, pathological pregnancies (toxemia of pregnancy, abortions, stillbirths, underweight births, etc) were frequently associated with increased PCB serum levels. ... [ENCYC OCCUPAT HEALTH & SAFETY 1983 , p. 1753]

HTOX - Mother's milk contaminated with PCB's appears to be a source of exposure for infants. Developmental abnormalities have been observed in PCB-intoxicated infants. Premature eruption of teeth was observed ... and larger frontal and occipital fontanelles, exophthalmos and the maintenance of an abnormally wide sagittal suture were observed. ... [IARC MONOGRAPHS. 1972-PRESENT V18 82 (1978)]

HTOX - Skin and mucous membrane changes; swelling of the eyelids, burning of the eye, and excessive eye discharge, burning sensation and edema of face and hands, simple erythematous eruptions with pruritus, acute eczematous contact dermatitis, chloracne, hyperpigmentation of skin and mucous membranes, discoloration of finger nails and thickening of the skin were reported. [ENCYC OCCUPAT HEALTH & SAFETY 1983 , p. 1754]

HTOX - CATEGORIES OF KNOWN OR SUSPECTED ORG CHEM CARCINOGENS FOUND IN DRINKING WATER: HIGHEST OBSERVED CONCN IN FINISHED WATER: 3 UG/L; UPPER 95% CONFIDENCE ESTIMATE OF LIFETIME CANCER RISK: 3.1X10-6 UG/L. [NRC. DRINKING WATER & HEALTH 1977 , p. 794]

HTOX - Dental records were studied and dental exams given to children living in Taiwan transplacentally exposed to polychlorinated biphenyls (PCB) (Yu-Cheng babies) as confirmed by epidemiological studies in the early 1980s. Nine school aged Yu-Cheng males and 9 females were compared to an unexposed reference group of 26 males and 18 females on the prevalence of missing permanent teeth germ while taking congenital factors into account. Among 9 transplacental Yu-Cheng girls, 4 were missing permanent teeth germ due to congenital factors. Among the 18 girls in the reference group, none were missing permanent teeth germ due to congenital factors. Among 9 transplacental Yu-Cheng boys, 1 was missing permanent teeth germ due to congenital factors. Among the 26 boys in the reference group, 1 was missing permanent teet germ due to congenital factors. [Lan SJ et al; Bull Environ Contam Toxicol 42 (6): 931-4 (1989)] **QC REVIEWED**

HTOX - A job exposure matrix was developed linking the work tasks in the Swedish National Census of population 1960 to exposure to 50 single agents or groups of substances, including polychlorinated biphenyls. All 1,905,660 men (ages 20-64 yr) in 1960, reporting themselves as gainfully employed in the Census, were observed for the occurrence of urothelial cancer during the 1961-1979 period by linkage to the National Swedish Cancer Registry. Only subjects in 1 work task, electricians in electric power stations, were assigned exposure to polychlorinated biphenyls with a moderate predictive value. The relative risk (with 95% confidence interval) for this group was 1.3 (1.0-1.8) for urinary bladder cancer. [Steineck G et al; Am J Ind Med 16 (2): 209-24 (1989)] **QC REVIEWED**

HTOX - In Taiwan in 1979, rice oil contaminated with polychlorinated biphenyls (PCBs) and polychlorinated dibenzofurans was ingested by approx 2000 people. Blood samples were taken from 36 women who were potentially exposed, and 24 non-exposed women (controls). The frequency of sister chromatid exchanges in lymphocytes from their heparinized whole blood was assessed after culturing cells in the presence or absence of 40 uM alpha-naphthoflavone for 72 hr. There was no

significant difference in baseline sister chromatid exchanges for PCB exposed compared to the control group (7.29 vs 7.61). In contrast, addition of alpha-naphthoflavone resulted in a dramatic induction of sister chromatid exchange frequencies in PCB exposed lymphocytes ($p < 0.01$). PCB exposed frequencies increased to 10.75, while those of the unexposed group only increased to 8.85. [Thompson C et al; Chemosphere 18 (1-6): 687-94 (1989)] **QC REVIEWED**

NTOX - PCB ADMIN HAS BEEN FOUND TO RESULT IN INCREASED SYNTHESIS, HEPATIC CONTENT & EXCRETION OF PORPHYRINS IN RATS, QUAILS & CHICKENS; & THIS HAS BEEN ASSOCIATED WITH AN INCR IN LIVER MITOCHONDRIAL GAMMA-AMINOLEVULINIC ACID SYNTHETASE. [IARC MONOGRAPHS. 1972-PRESENT V18 74 (1978)]

NTOX - The carcinogenicity of polychlorinated biphenyls was shown in animal, experimentally exposed. Benign and malignant liver cell tumors, lymphomas and leukemias, and carcinomas of the gastrointestinal tract were obtained. [ENCYC OCCUPAT HEALTH & SAFETY 1983 , p. 1754]

NTOX - EXPOSURE OF RATS, RABBITS, MONKEYS, CHICKS AND RAINBOW TROUT TO PCBS RESULTED IN INCREASED ACTIVITY OF ... URIDINE DIPHOSPHOGLUCURONOSYLTRANSFERASE & NITROREDUCTASE, OR A SIGNIFICANT INCR IN THE LEVEL OF CYTOCHROME P450. [IARC MONOGRAPHS. 1972-PRESENT V18 74 (1978)]

NTOX - EGGSHELL THINNING, LOSS OF REPRODUCTIVE ABILITY, OR BOTH HAVE BEEN ATTRIBUTED ... TO PCBS /IN BIRDS/. [HAYES. TOX OF PESTICIDES 1975 , p. 498]

NTOX - ISOMERICALLY PURE PCBS WERE TESTED AS INDUCERS OF HEPATIC DRUG-METABOLIZING ENZYMES IN THE RAT. THE CHLORINATED BIPHENYL ISOMERS CAN BE CATEGORIZED INTO 2 DISTINCT GROUPS OF INDUCERS, WHILE COMMERCIAL PCB MIXT HAVE CHARACTERISTICS OF BOTH GROUPS. BIPHENYLS CHLORINATED SYMMETRICALLY IN BOTH THE META AND PARA POSITIONS INCREASE THE FORMATION OF CYTOCHROME P448, BUT DECREASE THE AMINOPYRINE N-DEMETHYLASE ACTIVITY. BIPHENYL ISOMERS CHLORINATED IN BOTH THE PARA AND ORTHO POSITIONS INDUCE THE FORMATION OF CYTOCHROME P450 AND N-DEMETHYLASE ACTIVITY. ISOMERS WHICH ARE CHLORINATED IN ONLY 1 RING, OR ARE CHLORINATED IN BOTH RINGS BUT NOT IN THE PARA POSITIONS, HAVE VERY LITTLE ACTIVITY AS INDUCERS OF LIVER ENZYMES. [GOLDSTEIN JA ET AL; CHEM-BIOL INTERACT VOL 17 (1): 69-87 (1977)]

NTOX - The most consistent pathological changes occurring in mammals after polychlorinated biphenyls (PCB) exposure are in the liver. In rats, rabbits, and guinea pigs ... fatty deposits after acute injections and similar changes in rabbits and guinea pigs after dermal application /were observed/. In feeding experiments, marked fatty metamorphosis was noted in guinea pig liver with intracellular hyaline bodies observed in rats. Less striking changes were noted in the kidneys, lung, adrenals, and heart of guinea pigs. Rats exposed repeatedly to dietary PCBs showed increased liver weights. [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.C-37 (1980) EPA 440/5-80-068]

NTOX - Hepatic microsomal activity was elevated by single large doses of Aroclor 1242. Monkeys given 300 ppm for 90 days developed alopecia, chloracne, subcutaneous edema, liver hypertrophy, and hypertrophy and hyperplasia of the gastric mucosa. [GOSSELIN. CTCP 5TH ED. 1984 II-171]

NTOX - Animal studies have shown that polychlorinated biphenyls (PCBs) can cross the placental barrier and are excreted in the mother's milk. [IARC MONOGRAPHS. 1972-PRESENT V18 37 (1978)]

NTOX - GROUPS OF 10 MALE & 10 FEMALE 3-4-WEEK-OLD SHERMAN RATS WERE FED 0, 20, 100, 500 OR 1000 MG AROCLOR 1260/KG DIET ... SEVERAL ... GIVEN THE TWO HIGHEST DOSE LEVELS DIED BEFORE 6 MO. ... LESIONS ... DESCRIBED AS ADENOFIBROSIS OF LIVER OCCURRED IN 2 MALES FED 1000 MG/KG & IN 1, 1 AND 4 FEMALES FED 100, 500, AND 1000 MG/KG ... (KIMBROUGH ET AL, 1972) [IARC MONOGRAPHS. 1972-PRESENT V18 67 (1978)]

NTOX - Certain substitution patterns are believed to influence the biological activities of chlorobiphenyls. The presence of two adjacent carbon atoms without chlorine substitution in one or both rings is believed to facilitate metabolism because it permits the formation of arene oxide intermediates. Essentially all chlorobiphenyls with five or fewer chlorine atoms have at least one pair of adjacent unsubstituted carbon atoms because of the rarity of 3,5-substitution in the natural mixtures. ... Chlorobiphenyls with three or four chlorine atoms in the ortho-positions (2- and 6-) are more easily metabolized by humans than those with only one or two ortho- chlorines. ... Chlorobiphenyl isomers with chlorine substitutions in both the 4- and 4'- positions tend to be biologically active and well retained in tissues. [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.A-9-11 (1980) EPA 440/5-80-068]

NTOX - In addition to the inhibition of tumor induction by some chemicals, PCBs were also shown to inhibit the growth of experimental tumors in rats. Sprague-Dawley rats were innoculated with Walker 256 carcinosarcoma cells and the effects of PCBs determined. Both dietary and injected aroclor 1254 reduced the size of solid tumors and increased animal life span. [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.C-75 (1980) EPA 440/5-80-068] NTOX - The time course of induction and inhibition of several enzymes in the liver of male C57BL/6 and ddy mice fed a diet containing Kanechlor-500 (500 ppm) was examined. Controls were maintained without treatment. Four animals/group were killed at 1, 2, 3, 6, and 10 weeks, following a 24 hr fast. In treated C57BL/6 and ddY mice there was an increase in the microsomal p450 level at 1 week. In treated C57BL/6 mice, the activity of mitochondrial delta-aminolevulinic acid synthetase (ALA-S) gradually increased for 2 wk (5.7 times the control value) and then rose rapidly for 3 wk (20 times the control value). These changes were accompanied by the rapid development of porphyria (characterized by increased excretion and hepatic accumulation of uroporphyrin). The activity of uroporphyrinogen decarboxylase (URO-D) was depressed approximately 40% within the first wk and 80% within 3 wk. In treated ddY mice there was a moderate increase of ALA-S (8 times the control at 3 wk); URO-D activity was unaffected for 3 wk and a significant decrease (p value not given) was observed at wk 6. Activities of ALA-S and URO-D in both strains of control mice were constant during the study. The control level (time 0) of URO-D in ddY mice was significantly higher ($p < 0.01$) than that of C57BL/6 mice, whereas control ALA-S activities were similar in the 2 strains. In C57BL/6 mice, the hepatic uroporphyrin level was elevated during the first week of exposure. By the third week, the liver porphyrin level was 2100 times that of the controls. Porphyrin did not accumulate in treated ddy mice at week 3, was slightly increased at week 6, and remained constant at week 10. There were no increases in uroporphyrin levels in untreated mice. /Kanechlor-500/ [Seki Y et al; Toxicol Appl Pharmacol 90 (1): 116-25 (1987)] **QC REVIEWED**

NTOX - Groups of eight female Sprague-Dawley rats were fed 50 ppm Aroclor 1242 or Aroclor 1254 in their normal diets for seven months. One group of animals served as controls. Because (86)Rb mimics K+ in membrane transport, it was used to assess the amount of K+ uptake in erythroid cells. In a culture medium depleted of K+, the uptake of (86)Rb by erythroid cells was significantly lower in the Aroclor 1254-treated group (7.78%) ($p < 0.05$) compared with the control group (21.9%). Uptake of (86)Rb in the Aroclor 1242-treated group was not significantly lower than that of controls. In a sodium-depleted culture medium, erythroid cells from only the Aroclor 1254-treated animals showed a minimal, but significant reduction in (86)Rb uptake (1.16%) ($p < 0.05$) compared with the control group (3.91%). The difference in 86(Rb) uptake between the Na+ and K+-depleted culture media was attributed to the relative saturation of the K+ transport system. When erythroi cells were challenged with ouabain in order to suppress Na+, K+, and ATPase activity, (86)Rb uptake was depressed in all but the Aroclor 1254 group (this group had already been maximally depressed). [Byrne JJ, Sepkovic DW; Arch Environ Contam Toxicol 16: 573-7 (1987)] **QC REVIEWED**

NTOX - Juvenile rainbow trout (Salmo gairdneri) were fed diets of coho salmon containing polychlorinated biphenyls (PCBs) naturally bioaccumulated from Lake Michigan, Lake Ontario, or the Pacific Ocean for a 20 wk period. Gas chromatographic analysis indicated that the bioaccumulated PCB levels in rainbow trout were similar to the levels in coho salmon used as dietary supplements. Following dietary exposure to control chow or coho salmon from the Pacific Ocean, the rainbow trout contained low PCB levels, whereas trout which were fed Lake Michigan salmon and Lake Ontario salmon contained logarithmically elevated levels of PCBs. The effect on natural resistance was assessed by challenge with a titrated dose of Vibrio anguillarum (VA-58). The ability to mount a protective immune response in trout exposed for a 20-wk period to control or Lake Ontario coho salmon diets was determined by immunization with a VA-58 bacteria followed by challenge with virulent VA-58. A high level of protective immunity was demonstrated in all dietary groups suggesting that the parameters of host resistance of rainbow trout were not compromised following dietary exposure to Great Lakes coho salmon. [Cleland GB et al; Aquatic Toxicol 13 (4): 281-290 (1989)] **QC

NTOX - Eggs of three seabird species, double-crested cormorant (Phalacrocorax auritus), Leach's storm-petrel (Oceanodroma leucorhoa), and Atlantic puffin (Fratercula artica) were collected at 4-yr intervals from 1968 to 1984, from colonies (4 to 10 eggs/colony) in eastern Canada (A: Great Island, Newfoundland; B: Kent Island C: Manawagonish Island and D: Machias Seal Island, Bay of Fundy; and E: Ile-aux-Pommes, St. Lawrence River) and analyzed for organochlorines. PCBs

declined significantly in all species from the Bay of Fundy, and from site A, but not significantly at site E. Over the entire period, PCB residues were highest in the cormorant. [Pearce PA et al; Environ Pollut 56 (3): 217-35 (1989)] **QC REVIEWED**

NTOX - Channel catfish were obtained from Devil's Swamp (exposed fish), a river basin in southern LA heavily impacted by industrial complexes and hazardous waste sites. Reference fish were taken from LA State University's experimental Ben Hur aquaculture facility. The hepatic microsomal mediated O-dealkylation of various substituted alkoxyresorufins by the catfish were compared and correlated with polychlorinated biphenyl (PCB) contents from fat tissues of these fish. The most abundant PCB congeners found were pentachlorinated PCB (3900 ppb) followed by hexachlorinated (2800 ppb), tetrachlorinated (2400 ppb), heptachlorinated (540 ppb), octachlorinated (230 ppb), trichlorinated (130 ppb) and dichlorinated (40 ppb) biphenyls. The monochlorinated, nonochlorinated and decachlorinated biphenyls were represented in trace concn. Fatty tissue from reference fish were essentially devoid of PCB congeners. Specific activities and turnover numbers of 7-methoxyresorufin, 7-ethoxyresorufin, 7-pentoxyresorufin and benzyloxyresorufin O-dealkylases in Devil's Swamp microsomes of both male and female fish were significantly elevated relative to that of reference fish. When expressed per mg of protein, the degree of induction of these activities was notably higher in female catfish than in male catfish. [Winston GW et al; J Environ Sci Health (B) 24 (3): 277-89 (1989)] **QC REVIEWED**

NTOX - To study the chronic effects of contaminated sediments on mortality, growth, gonad production and bioaccumulation in the urchin, Lytechinus pictus, urchins were exposed to 3 of the most contaminated sediment types in southern California and to a control sediment in the laboratory for 60 days in flow through experiments. Initial concentrations of polychlorinated biphenyls in the sediments used were very high (1118-3484 ng/g vs < 59 ng/g dry wt in the control), as were those of other contaminants. The sediments caused significant mortality and reduction of growth. Both male and female gonad production was also significantly decreased. Gonads accumulated up to 7.4 ppm polychlorinated biphenyls during the 60 day exposure period, but their concentrations of Cd, Cu and Zn were greatly reduced. [Thompson BE et al; Environ Toxicol Chem 8 (7): 629-37 (1989)] **QC REVIEWED**

NTOX - In order to investigate interspecific responses to pollutants, physiological and biochemical parameters were studied in 2 sp of Gobiidae under both natural and experimental conditions. In addition to higher mixed function oxidase activity, Gobius niger, collected in a heavily polluted port, had higher polychlorinated biphenyls residues (0.45 ug/g dry wt vs 0.23 ug/g dry wt) than another sp, Zosterisessor ophiocephalus, collected from a relatively clean lagoon. After 20 days of acclimatization to clean water, the 2 sp exhibited practically identical levels of mixed function oxidase activity and polychlorinated biphenyls residues. Subsequent exposure to polychlorinated biphenyls (Arochlor 1260) at 10 ug/l resulted in considerably higher mixed function oxidase activity in G niger than in Z ophiocephalus and the polychlorinated biphenyls residue level was 4 times higher in G niger than in Z ophiocephalus after 20 days of exposure. [Fossi C et al; Ecotoxicol Environ Safety 18 (1): 11-4 (1989)] **QC REVIEWED**

NTOX - Two groups of 12 female common seals (Phoca vitulina) were fed fish having high levels of polychlorinated biphenyl (PCB) contamination from the Wadden Sea (Group 1), or fish having low PCB contamination from the northeast Atlantic (Group 2) for almost 2 yr. Seals in Group 1 had a drastic reduction in plasma retinol concn as compared to those in Group 2 (30 to 55% reduction). The PCB-induced reduction in plasma retinol levels disappeared when seals were subsequently fed low-PCB Atlantic Ocean fish for 6 mo. Significant reductions of total and free thyroxine and triiodothyronine were also observed in Group 1. [Brouwer A et al; Aquatic Toxicol 15 (1): 99-106 (1989)] **QC REVIEWED**

NTOX - Regeneration experiments on the liver of flounder (Platichthys flesus L.) were performed to identify the role of diverse pollutants (chlorinated hydrocarbons including polychlorinated biphenyls) in the pathogenesis of the liver abnormalities observed during a 3-yr multidisciplinary survey in the Elbe estuary (Federal Republic of Germany). Flounder kept under contaminant-free conditions and fed ad libitum with uncontaminated food indicated initial and complete liver regeneration in 50% of the individuals after 20 days, and in 70% after 40 days. Signs of regeneration, diagnosed at the light and electron microscope level were accompanied by a significant decr in the concn of chlorinated hydrocarbons in the liver. Livers of flounder without regenerative signs maintained their high levels of contaminants. The ultrastructural findings indicated that transfer of Elbe flounder into a contaminant-free environment induced incr activity of biotransformation and detoxification in the

hepatocytes (tubular smooth endoplasmic reticulum, lysosomes). [Kohler A; Aquat Toxicol 14 (3): 203-32 (1989)]

NTOX - Rhesus monkeys exposed to polychlorinated biphenyl mixtures during gestation and lactation were tested on two-choice discrimination-reversal learning (DR). In the first experiment, offspring of mothers fed 1.0 ppm Aroclor 1248 (avg exposure 20.7 + or - 3.1 mo), and offspring born 1.5 yr after maternal exposure to 2.5 ppm Aroclor 1248 ended (avg exposure 18.2 + or - 1.7 mo) did not differ from controls on spatial, color or shape discrimination-reversal problems. In the second experiment, offspring of mothers fed 0.25 or 1.0 ppm Aroclor 1016 (avg exposure of 21.8 + or - 2.2 mo) and offspring born 3 yr after maternal exposure to 2.5 ppm Aroclor 1248 ended were tested on the same spatial, color and shape problems, but a spatial problem with color and shape as irrelevant cues was inserted after the initial spatial problem. Performance of the high dose Aroclor 1016 offspring was impaired on the initial spatial problem, and facilitated on the shape problem. Performance of the Aroclor 1248 postexposure offspring was facilitated on the shape problem. [Schantz SL et al; Neurotoxicol Teratol 11 (3): 243-50 (1989)] **QC REVIEWED**

ADE - Polychlorinated biphenyls (PCBs) are readily absorbed through the gut, respiratory system and skin. /PCBs/ may initially concentrate in the liver, blood, and muscle mass, but long-term storage in mammals is primarily in adipose tissue and skin. ... [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.C-31 (1980) EPA 440/5-80-068]

ADE - PCBs which are readily metabolized are also rapidly excreted in the urine and bile. Excretion in urine is most prominent for the least chlorinated, while bile becomes the more significant route of excretion for more highly chlorinated isomers. Those isomers which are most refractory to metabolism accumulate for increasing periods of time in fatty tissues. Highly chlorinated isomers are accumulated almost indefinitely. [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.C-32 (1980) EPA 440/5-80-068]

ADE - The uptake of polychlorinated biphenyl (PCB) congeners was investigated in leaf composites and final fruits of four crop species at two alluvial mud sites (a control plot in the flood plain of the Normanskill, NY, and an experimental plot on Patroon Island, Albany, NY). Soy beans and corn were grown in the control plot. Soy beans, string beans, and pinto beans were grown in the experimental plot along with corn transplanted with soil confined in plastic bags from the control site. The three major constituents in each plant species were the same (2-mono-, 2,4'-di-, and 2,2'-dichlorobiphenyls), but thereafter the rank of the remaining major congeners varied according to species. Mean total PCB concentrations in leaves in ppb were 12-14 at the control site and 12-15 at the experimental site. However, samplings in late August showed relatively high levels of 30-50 ppb. Corn plants, tended to depurate PCBs over time. No trends for pinto beans or string beans were discernible. PCB levels (ppb) in fruits were as follows: corn kernels, 0.13 at both sites; corn husk, 3.06 at control site and 8.03 at experimental site; corn silk, 4.76 at control site; soybean beans, 0.55 at experimental site; soybean pods, 7.4 at experimental site. [Shane LA, Bush B; Ecotoxicol Environ Safety 17 (1): 38-46 (1989)]

ENVS - Current evidence suggests that the major source of PCB release to the environment is an environmental cycling process of PCBs previously introduced into the environment; this cycling process involves volatilization from ground surfaces (water, soil) into the atmosphere with subsequent removal from the atmosphere via wet/dry deposition and then revolatilization. PCBs are also currently released to the environment from landfills containing PCB waste materials and products, incineration of municipal refuse and sewage sludge, and improper (or illegal) disposal of PCB materials, such as waste transformer fluid, to open areas. PCBs are mixtures of different congeners of chlorobiphenyl and the relative importance of the environmental fate mechanisms generally depends on the degree of chlorination. In general, the persistence of PCBs increases with an increase in the degree of chlorination. Mono-, di- and trichlorinated biphenyls (Aroclor 1221 and 1232) biodegrade relatively rapidly, tetrachlorinated biphenyls (Aroclors 1016 and 1242) biodegrade slowly, and higher chlorinated biphenyls (Aroclors 1248, 1254, and 1260) are resistant to biodegradation. Although biodegradation of higher chlorinated congeners may occur very slowly on an environmental basis, no other degradation mechanisms have been shown to be important in natural water and soil systems; therefore, biodegradation may be the ultimate degradation process in water and soil. If released to soil, PCBs experience tight adsorption with adsorption generally increasing with the degree of chlorination of the PCB. PCBs will generally not leach significantly in aqueous soil systems; the higher chlorinated congeners will have a lower tendency to leach than the lower chlorinated congeners. In the presence of organic solvents PCBs may leach quite rapidly

through soil. Vapor loss of PCBs from soil surfaces appears to be an important fate mechanism with the rate of volatilization decreasing with increasing chlorination. Although the volatilization rate may be low, the total loss by volatilization over time may be significant because of the persistence and stability of PCBs. Enrichment of the low Cl PCBs occurs in the vapor phase relative to the original Aroclor; the residue will be enriched in the PCBs containing high Cl content. If released to water, adsorption to sediment and suspended matter will be an important fate process; PCB concentrations in sediment and suspended matter have been shown to be greater than in the associated water column. Although adsorption can immobilize PCBs (especially the higher chlorinated congeners) for relatively long periods of time, eventual resolution into the water column has been shown to occur. The PCB composition in the water will be enriched in the lower chlorinated PCBs because of their greater water solubility, and the least water soluble PCBs (highest Cl content) will remain adsorbed. In the absence of adsorption, PCBs volatilize relatively rapidly from water. However, strong PCB adsorption to sediment significantly competes with volatilization, with the higher chlorinated PCBs having longer half-lives than the lower chlorinated PCBs. Although the resulting volatilization rate may be low, the total loss by volatilization over time may be significant because of the persistence and stability of the PCBs. PCBs have been shown to bioconcentrate significantly in aquatic organisms. If released to the atmosphere, PCBs will primarily exist in the vapor-phase; the tendency to become associated with the particulate-phase will increase as the degree of chlorination of the PCB increases. The dominant atmospheric transformation process is probably the vapor-phase reaction with hydroxyl radicals which has estimated half-lives ranging from 12.9 days for monochlorobiphenyl to 1.31 years for heptachlorobiphenyl. Physical removal of PCBs from the atmosphere, which is very important environmentally, is accomplished by wet and dry deposition. The major PCB exposure routes to humans are through food and drinking water, and by inhalation of contaminated air. (SRC) [CITATION]

ARTS - Current sources of PCB release to the environment include releases from landfills containing PCB waste materials and products, incineration of municipal refuse and sewage sludge, and improper (or illegal) disposal of PCB materials, such as waste transformer fluid, to open areas(1,2). Monitoring of atmospheric emissions of PCBs from landfills and incinerator stacks has indicated that emission rates from these sources are small compared to the 900,000 kg/yr of PCBs estimated to cycle through the atmosphere over the USA annually(2). Atmospheric washout and fallout have been identified as non-point sources of PCB exposure to the environment(1,3,4). Although additional research is required for a definitive answer, current evidence suggests that the major source of PCB release to the environment is an environmental cycling process of PCBs previously introduced into the environment; this cycling process involves volatilization from ground surfaces (water, soil) into the atmosphere with subsequent removal from the atmosphere via wet/dry deposition and then revolatilization(2,3,4,SRC). During all these cycles, the PCB composition changes in each phase relative to the original Aroclor so that specific Aroclor contributions may become impossible to assign. Specific congerner PCB analysis must be performed on environmental media to obtain the total extent of PCB pollution(5). [(1) Weant GE, McCormick GS; Nonindustrial Sources of Potential Toxic Substances and Their Applicability to Source Apportionment Methods. p.36, 86 (1984) USEPA-450/4-84-003 NTIS PB 84-231232 (2) Murphy TJ et al; Environ Sci Technol 19: 942-6 (1985) (3) Swackhamer DL, Armstrong DE; Environ Sci Technol 20: 879-83 (1986) (4) Larrson P; Nature 317: 347-9 (1985) (5) USEPA; Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs) ECAO-CIN-414 p. IV-30 (1987)] ARTS - In preparation of remedial action plans for the St. Clair, Detroit, and St. Mary's rivers, a planning-level methodology for evaluation of pollutant loadings from urban nonpoint sources was developed and applied in 3 Canadian cities: Sarnia, Sault Ste. Marie, and Windsor. This methodology uses computed annual volumes of runoff and mean constituent concn, estimated from field sampling, to produce estimates of annual pollutant loadings. The mean stormwater concn and point source equivalent concn (ug/l) for polychlorinated biphenyls (PCBs) were 0.179 and 0.179 for Sarnia, 0.0269 and 0 for Sault Ste. Marie, and 0.0888 and 0.641 for Windsor, respectively. [Marsalek J, Ng HYF; J Great Lakes Res 15 (3): 444-51 (1989)]

FATE - ... Percentages of chlorine /in mixtures/ changes with time and location as the mixtures are transported through the environment. [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.B-2 (1980) EPA 440/5-80-068]

FATE - TERRESTRIAL FATE: PCBs are mixtures of different congeners of chlorobiphenyl and the relative

importance of the environmental fate mechanism generally depends on the degree of chlorination(1). In general, the persistence of PCBs increases with an increase in the degree of chlorination. Mono-, di- and trichlorinated biphenyls (Aroclors 1221 and 1232) biodegrade relatively rapidly, tetrachlorinated biphenyls (Aroclors 1016 and 1242) biodegrade slowly, and higher chlorinated biphenyls (Aroclors 1248, 1254 and 1260) are resistant to biodegradation. The position of chlorination in the isomeric classes also determines the extent of biodegradation. Although biodegradation of higher chlorinated congeners may occur very slowly on an environmental basis, no other degradation mechanisms have been shown to be important in soil systems; therefore, biodegradation may be the ultimate degradation process in soil. PCBs have been shown to be tightly adsorbed in soil with adsorption generally increasing with the degree of chlorination. Although the monochlorobiphenyls may have some low mobility in soil, PCBs will generally not leach significantly in most aqueous soil systems. The higher chlorinated congeners will have a lower tendency to leach than the lower chlorinated congeners. In the presence of organic solvents, which may be possible at waste sites, PCBs may leach quite rapidly through soil. Vapor loss of PCBs from soil surfaces appears to be an important mechanism with the rate of volatilization decreasing with increasing chlorination. Although the volatilization rate may be low, the total loss by volatilization over time may be significant because of the persistence and stability of PCBs(2). [(1) USEPA; Drinking Water Criteria Document for Polychlorianted Biphenyls (PCBs) p. II 1-30 (1987) USEPA ECAO-Cln-414 (2) Sklarew DS, Girvin DC; Rev Environ Contam Toxicol 98: 1-41 (1987)]

FATE - AQUATIC FATE: PCBs are mixtures of different congeners of chlorobiphenyl and the relative importance of the environmental fate mechanism generally depends on the degree of chlorination(1). In general, the persistence of PCBs increases with an increase in the degree of chlorination. Mono-, di- and trichlorinated biphenyls (Aroclors 1221, and 1232) biodegrade relatively rapidly, tetrachlorinated biphenyls (Aroclors 1016 and 1242) biodegrade slowly, and higher chlorinated biphenyls (Aroclors 1248, 1254, and 1260) are resistant to biodegradation. The position of chlorination in the isomeric classes also determines the extent of biodegradation. Although biodegradation of higher chlorinated congeners may occur very slowly on an environmental basis, no other degradation mechanisms have been shown to be important in environmental aquatic systems; therefore, biodegradation may be the ultimate degradation process in natural water. In water, adsorption to sediments and organic matter is a major fate process for the PCBs(1,2). Experimental and monitoring data have shown that PCB concentrations are higher in sediment and suspended matter than in the associated water column. The lower chlorinated PCBs will sorb less strongly than the higher chlorinated PCBs. Although adsorption can immobilize PCBs for relatively long periods of time in the aquatic environment, resolution into the water column has been shown to occur on an environmental level suggesting that the substantial quantities of PCBs contained in aquatic sediments can act as an environmental sink for environmental redistribution of PCBs(3,4). Volatilization of dissolved PCBs is an important aquatic process. A study conducted on Lake Michigan has indicated that volatilization may be the major removal mechanism of PCBs from lakes(3). Strong PCB adsorption to sediment significantly decreases the rate of volatilization, with higher chlorinated PBCs having longer half-lives than the lower chlorinated PCBs(1). Although the volatilization rate may be low, the total loss by volatilization over time may be significant because of the persistence and stability of the PCBs. Aquatic hydrolysis and oxidation are not important processes with respect to the PCBs. PCBs have been shown to bioconcentrate significantly in aquatic organisms(SRC). [(1) USEPA; Drinking Water Criteria Document of Polychlorinated Biphenyls (PCBs) p. II 1-30 (1987) USEPA ECAO-CIN-414 (2) Callahan MA et al; Water-Related Environmental Fate of 129 Priority Pollutants Chapter 36 EPA-440/4-79-029a (1979) (3) Swackhamer DL, Armstrong DE; Environ Sci Technol 20: 879-93 (1986) (4) Baker JE et al; Environ Sci Technol 19: 854-61 (1985)]

FATE - ATMOSPHERIC FATE: The vapor pressures of the PCBs indicate that they should exist primarily in the vapor-phase in the atmosphere; monitoring data have shown that between 87 and 100% of the PCBs in air are in the vapor-phase(1). The vapor pressure of PCBs generally decreases with an increase in the degree of chlorination; therefore, the higher chlorinated PCBs are more likely to be associated with the particulate-adsorption-phase in air than are the lower chlorinated PCBs. Physical removal of PCBs in the atmosphere is accomplished by wet and dry deposition(1); dry deposition occurs only for PCBs associated in the particulate phase. The PCB concn of rain anywhere in the world may typically range between 1 and 250 ng/l which is an indication of the

importance of wet deposition(1). The vapor phase reaction of PCBs with hydroxyl radicals (which are photochemically formed by sunlight) may be the dominant transformation process in the atmosphere. The estimated half-life for this reaction in a typical atmosphere ranges from 12.9 days for monochlorobiphenyl to 1.31 years for heptachlorobiphenyl; the half-life increases with an increase in the degree of chlorination. [(1) Eisenreich SJ et al; Environ Sci Technol 15: 30-8 (1981)]

BIOD - Results of a static-culture flask-screening procedure utilizing biological oxygen demand dilution water, settled domestic wastewater inoculum, 5-10 ppm test compound and 28 days of incubation of the individual Aroclors were as follows (% biodegraded): Aroclors 1221 and 1232 (100%), Aroclor 1016 (13-48%), Aroclor 1242 (0-66%), Aroclors 1248, 1254, and 1260 (0%)(1). Reviews of available PCB biodegradation data have reported that mono-, di-, and tri-isomers (Aroclors 1221 and 1232) biodegrade relatively rapidly, tetra-isomers (Aroclors 1016 and 1242) biodegrade slowly and higher PCBs (Aroclor 1248, 1254,and 1260) are resistant to biodegradation(2,3). [(1) Tabak HH et al; J Water Pollut Contr Fed 53: 1503-18 (1981) (2) USEPA; Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs) p.II-29 (1987) USEPA ECAO-CIN-414 (3) Leifer A et al; Environmental Transport and Transformation of Polychlorinated Biphenyls (1983) EPA-560/5-83-025, NTIS PB84-142579]

BIOD - Measured as a decr in congener concn, clear evidence for anaerobic biodegradation of ambient PCBs was found in untreated Hudson River sediments incubated in the laboratory under a nitrogen atmosphere. Clay encapsulated dredged sediment samples were also taken from the Moreau site. The Moreau sediment contained total PCB concn of 936 + or - 15 mg/kg dry wt, while the river sediment contained 707 + or - 14 mg/kg dry wt. About 53% of the total PCBs (375 mg/kg sediment dry wt), mainly mono to pentachlorobiphenyls, was degraded in the biphenyl amended Hudson River sediments and 30% (281 mg/kg sediment) in biphenyl amended Moreau sediments after 7 mo, with the spectrum of congeners degraded much broader in Hudson River than Moreau sediments. Biphenyl amendment enhanced the disappearance of highly chlorinated congeners. Inoculation with the mixed culture showed positive results in Moreau sediments but not in Hudson River sediments. Regardless of treatments, no biodegradation occurred in a carbon dioxide/hydrogen atmosphere. Moreau sediments incubated in situ (from November to June) showed little change in congener concn in all treatments. [Rhee GY et al; Water Res 23 (8): 957-64 (1989)]

BIOC - polychlorinated biphenyls (PCBs) are highly lipophilic and bioconcentrate in tissue from concentrations in water ... [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.B-1 (1980) EPA 440/5-80-068]

KOC - MICROBIAL DEGRADATION & VOLATILITY OF POLYCHLORINATED BIPHENYLS (PCBS) WERE DETERMINED. ADSORPTION WAS CORRELATED TO THE TOTAL CHLORINE CONTENT & SURFACE AREA OF EARTH MATERIAL (SOIL, CLAY, SAND). PCBS WERE IMMOBILE WHEN LEACHED WITH WATER OR LANDFILL LEACHATE BUT WERE INTENSELY MOBILE WHEN LEACHED WITH ORGANIC SOLVENTS. HIGHER CHLORINATED ISOMERS ARE LESS MOBILE & MORE PERSISTENT IN THE ENVIRONMENT THAN LOWER CHLORINATED ISOMERS. [GRIFFIN RA, CHIAN ES K; US ENVIRON PROT AGENCY, OFF RES DEV, (REP); EPA-600/2-80-027: 101 PAGES (1980)] KOC - PCB mobility in aqueous soil-sediment systems has reported experimental Koc values ranging from 510 to 13,300,000 for a variety of Aroclors and PCB congeners; reported Koc values were mostly above 5000(1). Reviews of the PCB mobility literature have found that adsorption of PCBs to soil and sediment generally increases with an increase in the degree of chlorination(2,3). Using soil TLC, column leaching and five different soils, PCBs were found to be generally immobile when leached with water or aqueous landfills leachate, but highly mobile when leached with organic solvents(4). [(1) Sklarew DS, Girvin DC; Rev Environ Contam Toxicol 98: 1-42 (1987) (2) Leifer A et al; p.4-1 to 4-21 Environmental Transport and Transformation of Polychlorinated Biphenyls EPA-560/5-83-025, NTIS PB84-142579 (1983) (3) USEPA; Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs) p.II-27 (1987) USEPA ECAO-CIN-414 (4) Griffin RA, Chou SFJ; Wat Sci Tech 13: 1153-63 (1981)]

VWS - SOIL: Vapor loss of aroclor 1254 from clay soil was negligible over a 4 week period, but vapor loss from sand varied from 10-75% over the same period with evaporation of the lower chlorinated cogeners occurring at the faster rates(1). Vapor loss of Aroclor 1254 from three different soils was observed to be 40-50% over 2-4 months(2). Hexachloro- and heptachlorobiphenyls volatilized more slowly from sand than pentachlorbiphenyl which volatilized at only half the rate of tetrachlorobiphenyls(2). [(1) Haque R et al; Environ Sci Technol 8: 139-42 (1974) (2) Pal D et al;

NAME - CALCIUM CYANIDE

HTOX - SYMPTOMATOLOGY: 1. Massive doses may produce, without warning, sudden loss of consciousness and prompt death from respiratory arrest. With smaller but still lethal doses, the illness may be prolonged for 1 or more hours. 2. Upon ingestion, a bitter, acrid, burning taste is sometimes noted, followed by a feeling of constriction or numbness in the throat. Salivation, nausea and vomiting are not unusual ... 3. Anxiety, confusion, vertigo, giddiness, and often a sensation of stiffness in the lower jaw. 4. Hyperpnea and dyspnea. Respirations become very rapid and then slow and irregular. Inspiration is characteristically short while expiration is greatly prolonged. 5. The odor of bitter almonds may be noted on the breath or vomitus ... 6. In the early phases of poisoning, an increase in vasoconstrictor tone causes a rise in blood pressure and reflex slowing of the heart rate. Thereafter, the pulse becomes rapid, weak, and sometimes irregular. ... A bright pink coloration of the skin due to high concentrations of oxyhemoglobin in the venous return may be confused with that of carbon monoxide poisoning. /Cyanide/ [GOSSELIN. CTCP 5TH ED. 1984 III-126]

HTOX - SYMPTOMATOLOGY: 7. Unconsciousness, followed promptly by violent convulsions, epileptiform, or tonic, sometimes localized but usually generalized. Opisthotonos and trismus may develop. Involuntary micturition and defecation occur. 8. Paralysis follows the convulsive stage. The skin is covered with sweat. The eyeballs protrude, and the pupils are dilated and unreactive. The mouth is covered with foam, which is sometimes bloodstained. ... The skin color may be brick red. Cyanosis is not prominent in spite of weak and irregular gasping. In the unconscious patient, bradycardia and the absence of cyanosis may be key diagnostic signs. 9. Death from respiratory arrest. As long as the heart beat continues, prompt and vigorous treatment offers some promise of survival. /Cyanide/ [GOSSELIN. CTCP 5TH ED. 1984 III-127] **PEER

HTOX - MOST SPECIFIC PATHOLOGICAL FINDING IN ACUTE CASES /OF CYANIDE POISONING/ IS BRIGHT RED COLOR OF VENOUS BLOOD. THIS IS STRIKING, VISIBLE EVIDENCE OF INABILITY OF TISSUE CELLS TO UTILIZE OXYGEN ... VENOUS BLOOD IS ONLY ABOUT 1 VOL % LOWER IN OXYGEN CONTENT THAN ARTERIAL BLOOD ... /CYANIDES/ [PATTY. INDUS HYG & TOX 3RD ED VOL2A,2B,2C 1981-82 , p. 4847]

HTOX - WORKERS IN ELECTROPLATING INDUST HAVE SHOWN DERMATITIS TO BE A PROBLEM. ALSO REPORTED WERE ITCHING, SCARLET RASH, PAPULES ... IRRITATION OF NOSE, LEADING TO OBSTRUCTION, BLEEDING, SLOUGHS AND IN SOME CASES PERFORATION OF SEPTUM. /CYANIDES/ [ENCYC OCCUPAT HEALTH & SAFETY 1983 , p. 575]

HTOX - ... IT IS POSSIBLE FOR CYANIDE TO CAUSE BLINDNESS & TO DAMAGE OPTIC NERVES & RETINA. /CYANIDE ION/ [GRANT. TOX OF THE EYE 1974 , p. 334]

HTOX - A STUDY WAS UNDERTAKEN TO ASSESS THE HEALTH STATUS OF WORKERS EXPOSED TO CYANIDE FUMES & AEROSOLS IN A FACTORY. CYANIDE LEVELS WERE MEASURED IN THE WORK ENVIRONMENT & IN BLOOD & URINE. SMOKERS HAD HIGHER CONCENTRATIONS THAN NON-SMOKERS. THE HIGHEST LEVELS WERE 0.8 & 0.2 MG/CU M IN BREATHING ZONE & GENERAL WORKROOM ATMOSPHERE, RESPECTIVELY. THE WORKERS COMPLAINED OF TYPICAL CYANIDE POISONING IN SPITE OF THE LOW CONCN. ... MAC VALUES OF 5 MG/CU M (IN TERMS OF CN) SHOULD BE REVIEWED IN INDIA. /CYANIDES/ [CHANDRA H ET AL; J ANAL TOXICOL 4 (4): 161-65 (1980)]

HTOX - Cyanides are absorbed from the skin & mucosal surfaces and are ... dangerous when inhaled because toxic amt are ... absorbed through bronchial mucosa & alveoli. Symptoms, which /may/ occur ... are giddness, headache, palpitation, dyspnea, & unconsciousness. There may be some evidence of local irritation from the salts & nausea & vomiting. ... Central nervous depression. ... Early electrocardiographic changes may include atrial fibrillation, ectopic

ventricular beats, and abnormal QRS complex with T wave originating high on the R wave. Sinus bradycardia is a common presenting sign. As cyanide levels in the blood rise, ataxia develops & is followed by coma, convulsions, & death. /Cyanides/ [HADDAD. CLIN MGT POISON & DRUG OVERDOSE 1983 , p. 745]

HTOX - Signs & symptoms of acute cyanide poisoning reflect cellular hypoxia & are often nonspecific. Onset of symptoms depends on dose, route, & duration of exposure. Inhalation produces ... flushing, headache, tachypnea, & dizziness ... irregular stridulous breathing, coma, seizure, & death ... /Cyanide/ [ELLENHORN. MEDICAL TOXICOLOGY 1988 , p. 831]

HTOX - WHEN ABSORBED, /CYANIDE/ ... REACTS READILY WITH ... CYTOCHROME OXIDASE IN MITOCHONDRIA; CELLULAR RESPIRATION IS THUS INHIBITED & CYTOTOXIC HYPOXIA RESULTS. ... RESPIRATION IS /INITIALLY/ STIMULATED ... A TRANSIENT STAGE OF CNS STIMULATION WITH HYPERPNEA AND HEADACHE IS OBSERVED; FINALLY THERE ARE HYPOXIC CONVULSIONS AND DEATH DUE TO RESPIRATORY ARREST. /CYANIDE/ [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1642]

HTOX - ... ENLARGED THYROID GLANDS /WERE REPORTED/ IN WORKERS EXPOSED TO CYANIDE SALTS IN HEAT TREATMENT OF METALS. IT WAS SUGGESTED THAT ABSORPTION OF CYANIDE DUST & HYDROGEN CYANIDE PRODUCED BY HYDROLYSIS OF CYANIDE SALTS, WAS FOLLOWED BY METABOLISM TO THIOCYANATE, & THAT FAILURE TO ELIMINATE THIS ... CAUSED GOITROGENIC EFFECT. /CYANIDE SALTS/ [ACGIH. DOCUMENTATION OF TLVS 5TH ED 1986 , p. 153]

HTOX - USE OF CALCIUM CYANIDE SHOULD BE RESTRICTED TO PERSONS WHO UNDERSTAND ITS DANGERS & PROPER HANDLING /CYANIDE GAS IS LIBERATED ON CONTACT WITH MOISTURE/. GAS IS EXTREMELY TOXIC BY INHALATION & SKIN ABSORPTION. [FARM CHEMICALS HANDBK 1987 C-47]

HTOX - VOLATILE CYANIDES /SRP: AND ALL AIRBORNE CYANIDE SALTS/ RESEMBLE HYDROCYANIC ACID PHYSIOLOGICALLY, INHIBITING TISSUE OXIDN & CAUSING DEATH THROUGH ASPHYXIA. CYANOGEN IS PROBABLY AS TOXIC AS HYDROCYANIC ACID ... /CYANIDES/ [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 822]

NTOX - IN EXPTL ANIMALS, DEMONSTRATION OF EFFECTS OF CYANIDE POISONING ON RETINA & OPTIC NERVE HAS BEEN SUCCESSFUL PRINCIPALLY WITH ACUTE SEVERE, NEAR-LETHAL OR LETHAL POISONINGS. /CYANIDES/ [GRANT. TOX OF THE EYE 1974 , p. 334]

NTOX - IN RABBITS, AFTER SUBLETHAL DOSES OF CYANIDE, CHANGES IN ELECTRORETINOGRAM HAVE BEEN OBSERVED. /CYANIDES/ [GRANT. TOX OF THE EYE 1986 , p. 287]

NTOX - IN THE CASE OF HYDROCYANIC ACID AND CYANIDES /IN VERY HIGH DOSES/, DEATH USUALLY OCCURS /IN ANIMALS/ WITHIN A FEW SECONDS: THERE MAY BE CONVULSIONS, PARALYSIS, STUPOR, & CESSATION OF RESPIRATION BEFORE THAT OF HEARTBEATS. /CYANIDES/ [CLARKE. VET TOX 1981 , p. 176]

NTOX - Except for the more sensitive invertebrate species, such as Daphnia pulex and Gammarus pseudolimnaeus, invertebrate species are usually more tolerant of cyanide than are freshwater fish species, which have most acute values clustered between 50 to 200 ug/l. A long-term survival and two life cycle test with fish gave chronic values of 7.9, 14, and 16 ug/l, respectively, with Gammarus pseudolimnaeus being comparable to fish in sensitivity and isopods being considerably more tolerant. /Free cyanide: HCN and CN-/ [USEPA; Ambient Water Quality Criteria Doc: Cyanides p.B-6 (1980) EPA 440/5-80-037]

NTOX - ... /THERE IS A/ COMBINED EFFECT OF PULMONARY EDEMA AND THE INTERFERENCE OF CELLULAR METABOLISM BY THE CYANIDE RADICAL. /CYANIDE ION/ [PATTY. INDUS HYG & TOX 3RD ED VOL2A,2B,2C 1981-82 , p. 4861]

NTOX - IF ... ANIMALS ... HAVE EATEN CYANOGENIC PLANTS, CLINICAL SIGNS MAY VARY FROM MILD TACHYPNEA & APPARENT ANXIETY TO SEVERE PANTING, GASPING, & BEHAVIORAL ALARM. OTHER SIGNS INCL SALIVATION, MUSCLE TREMORS, LACRIMATION, URINATION & DEFECATION, SEVERE COLIC, EMESIS, PROSTRATION, ... CLONIC CONVULSIONS, MYDRIASIS, & RAPID DEATH. ... MUCOUS MEMBRANES ARE ... PINK & BLOOD IS CHERRY RED & MAY NOT CLOT. RED COLOR IS DUE TO HYPEROXYGENATION THAT OCCURS WHILE THE ANIMAL IS DYING. THERE MAY BE AGONAL HEMORRHAGES ON HEART. GI TRACT & LUNG MAY HAVE CONGESTION &

PETECHIAL HEMORRHAGES. /CYANOGENIC PLANTS/ [BOOTH. VET PHARM THERAP 5TH ED 1982 , p. 961]

NAME - SODIUM CYANIDE

RTEX - Toxic by skin absorption through open wounds, by ingestion, and by inhalation of hydrogen cyanide from the decomposition of the material. [AAR. EMERGENCY HANDLING HAZ MAT SURFACE TRANS 1987 , p. 628]

RTEX - /IN ELECTROPLATING/ ... SODIUM BATH CONTAINS SODIUM CYANIDE ... ... [BROWNING. TOX INDUS METALS 2ND ED 1969 , p. 164]

RTEX - ... SYMPTOMS OF CHRONIC DISEASE ... REPORTED IN ELECTROPLATERS & SILVER POLISHERS AFTER SEVERAL YEARS OF EXPOSURE. /CYANIDES/ [ENCYC OCCUPAT HEALTH & SAFETY 1983 , p. 575]

RTEX - Dermatitis in workers chronically exposed TO CYANIDE SOLN. ELECTROPLATERS SUFFER FROM SUCH IRRITATION. /CYANIDE SOLN/ [HAMILTON. INDUS TOX 3RD ED 1974 , p. 224]

FATE - Aquatic Fate: The alkali metal salts are very soluble in water, and as a result, they readily dissociate into their respective anions and cations upon release to water. The resulting cyanide ion may then form hydrogen cyanide or react with various metals present in natural water. If the cyanide ion is present in excess, complex metallocyanides may form; however, if metals are prevalent, simple metal cyanides may form. [DHHS/ATSDR; Toxicological Profile for Cyanide (Draft) p.76 (1/88)]

Res Rev 74: 45-98 (1980)]

SEDS - Polychlorinated biphenyls (PCBs) were among anthropogenic organic contaminants measured in 33 sediment samples collected in the Trenton Channel of the Detroit River, connecting Lake St. Clair and Lake Erie. Analysis was by electron capture, negative ionization gas chromatographic mass spectrometry. Total PCB (sum of Cl3 to Cl10 homologs) concentrations ranged from none detected to 13,000 ng/g dry weight of sediment at Station 77 (near Monguagon Creek and the Federal Marine Terminal) and 14,000 ng/g near Elizabeth Park. The PCBs were skewed toward the higher chlorine homologs (Cl8 to Cl10). Sediment samples collected at the shore of Fighting Island and on the southern shore of Gross Ile showed <100 ng/g of PCBs. [Furlong ET et al; J Great Lakes Res 14 (4): 489-501 (1989)]

SEDS - The concentrations of polychlorinated biphenyl congeners were determined in Lake Ontario sediments. Surficial sediments had a reasonably uniform contaminant distribution throughout the sedimentation basins, with no strong plumes to sources. Mean concentrations of polychlorinated biphenyls (ng/g) in the basins of Lake Ontario were 510 + or - 160 in Niagara, 690 + or - 220 in Mississauga, 630 + or - 340 in Rochester, and 200 + or - 150 in Kingston. Sediment samples outside the sedimentation basins displayed very low contaminant concentrations, averaging of 4 ppb. Sediment trap studies showed that a considerable amount of sediment resuspension occurs in the lake, especially when it is unstratified during the winter. Sediment core studies showed peak discharges of the contaminants occurred in the late 1960s, in good agreement with production and usage history. [Oliver BG et al; Environ Sci Technol 23 (2): 200-8 (1989)]

SEDS - Using gas chromatography with a (63)Ni electron capture detector on a capillary column, polychlorinated biphenyls were analyzed in surface soil samples (0.5 cm) collected from 49 different locations (remote, rural and urban) in Wales in order to define the background levels of contamination. The polychlorinated biphenyl concentrations ranged from < 0.2 to 12.2 ug/kg of soil, with mean and median of 3.1 and 2.5 ug/kg, respectively. The higher levels were found in soil samples collected from the industrial south east of Wales. Soil properties, such as organic matter or clay content, were not found to correlate with the polychlorinated biphenyl content of the soils. [Jones KC; Chemosphere 18 (7-8): 1665-72 (1989)]

RTEX - ... Environmental contamination may be a significant source of human exposure. Likely routes of exposure for the general population are water and particularly food, while inhalation and dermal contact are likely to be more significant routes in occupational exposure. [USEPA; Ambient Water Quality Criteria Doc: Polychlorinated Biphenyls p.C-3 (1980) EPA 440/5-80-068]

RTEX - The PCB major exposure routes to humans are through food and drinking water, and by inhalation of contaminated air(1). Dermal exposure is important for workers involved with handling PCB-containing electrical equipment, spills or waste-site materials and for swimmers in polluted water(1). Exposure through consumption of contaminated fish may be especially important. Large exposure may be characteristic of PCB fires(1). [(1) USEPA; Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs) p.IV-36 (1987) USEPA ECAO-CIN-414]

RTEX - Serum samples from 285 4-yr-old Michigan children were evaluated for levels of 11 environmental contaminants. Polychlorinated biphenyls (PCBs) were found in half the samples tested. Nursing (mothers' milk) was the principal source of these exposures. Congener-specific analysis documented the presence of at least one highly toxic PCB congener, 2,3',4,4',5-pentachlorobiphenyl. Mothers were exposed to PCBs through their consumption of Lake Michigan sport fish or farm products (milk or meat) contaminated with PCBs as ascertained by the Michigan Department of Public Health. [Jacobson JL et al; Am J Public Health 79 (10): 1401-4 (1989)]

LEAD

BP - 1740 DEG C [MERCK INDEX. 10TH ED 1983 , p. 776]
MP - 327.4 DEG C [MERCK INDEX. 10TH ED 1983 , p. 776]
MW - 207.20 [MERCK INDEX. 10TH ED 1983 , p. 776]
SOL - INSOL IN HOT OR COLD WATER; SOL IN NITRIC ACID, HOT CONCN SULFURIC ACID [WEAST. HDBK CHEM & PHYS 68TH ED 1987-1988. B-99]
CAREV- CLASSIFICATION: B2; probable human carcinogen. BASIS FOR CLASSIFICATION: Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statisticlly significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short term studies show that lead affects gene expression. Human evidence is inadequate. HUMAN CARCINOGENICITY DATA: Inadequate. ANIMAL CARCINOGENICITY DATA: Sufficient. [U.S. Environmental Protection Agency's Integrated Risk Information System (IRIS) on Lead and compounds (inorganic) (7439-92-1) from the National Library of Medicine's TOXNET System, August 29, 1994]
CAREV- Notice of Intended Changes (1993-1994): A3. A3 = Animal carcinogen. /Lead, elemental, and inorganic cmpd, as Pb/ [ACGIH. TLV'S CHEM SUBSTS & PHYSICAL AGENTS & BIOLOGICAL EXP INDICES 1994-1995 , p. 37]
o EMCE - CLINICAL EFFECTS :

Signs and symptoms of lead poisoning are referable to the nervous, hematologic, renal, gastrointestinal, and cardiac systems (Cullen et al, 1983). There may be no unique signs or symptoms to suggest lead poisoning as the diagnosis. Serious injury can occur when neither signs nor symptoms have been present. Case detection is best done by screening populations at risk, which include operatives in lead industries, their children, and children living in homes built before 1980 where lead-containing paint may be the source. Universal screening of children beginning at 6 months is recommended by the CDC.

Most lead poisoning is slow in onset and results from gradual accumulation of lead from sources of low solubility such as lead paint or industrial dusts. The ingestion of rapidly absorbed salts causes an acute syndrome of hepatic injury and hemolysis as well as chronic exposure effects.

VITAL SIGNS

- o Chronic lead exposure may cause hypertension.

CARDIOVASCULAR

- o Chronic exposure in adults leads to renal hypertension and secondary cardiac effects.

NEUROLOGIC

In young children, developmental defects, including learning disabilities, lowered IQ, and behavioral abnormalities, can occur without symptoms. At higher levels of exposure, non-specific signs occur. At high levels, encephalopathy with imminent risk of death, permanent mental retardation, and motor deficits.

GASTROINTESTINAL

Anorexia, constipation, and diarrhea may be present due to direct effects or the result of altered CNS control.

HEPATIC

Liver injury occurs from poisoning with readily available lead salts but rarely with chronic inorganic lead exposure.

GENITOURINARY

In acutely ill patients, proteinuria, glucosuria, and aminoaciduria may occur. Chronic exposure leads to tubular damage, azotemia, and gout.

Decreased sperm count in lead exposed males has been described.

METABOLISM - Hyperuricemia may be seen after chronic lead poisoning.

HEMATOLOGIC

Lead interrupts several steps in heme synthesis resulting in anemia. Assay of substrates of blocked enzymes is used in diagnosis. With some lead salts, hemolysis is possible. RBCs show endoplasmic clumping known as stippling.

MUSCULOSKELETAL

Lead is deposited in teeth and bones. In children, lead deposition can be seen as an opacity in the metaphyseal plate.

OTHER

Lead sulfide precipitates in gum margins causing a blue-black line. Present in older children and adults; rarely in children <5 years as the sulfide comes from bacteria rarely found in the mouths of young children.

o EMLAB- LABORATORY :

Elevation of blood lead level is essential to the diagnosis of childhood and industrial cases. Children with a blood lead level of 45 or greater require medical treatment. Children with blood lead levels between 25 and 44 mcg/dL may need evaluation with a CaNa2EDTA challenge test and children with levels of 10 to 24 mcg/dL require medical monitoring and environmental evaluation.

HTOX - TOXIC BY INGESTION & INHALATION OF DUST OR FUME. [SAX. HAWLEY'S CONDENSED CHEM DICT 11TH ED 1987 , p. 687]

HTOX - ONE OF 2 /EPIDEMIOLOGICAL/ STUDIES ON METALLIC LEAD WORKERS SHOWED NO EXCESS OF CANCER DEATHS. THE OTHER SHOWED A SLIGHT (ALTHOUGH SIGNIFICANT) EXCESS OF DEATHS DUE TO CANCERS OF DIGESTIVE SYSTEM & RESP SYSTEM AMONG SMELTER WORKERS BUT NOT AMONG WORKERS IN LEAD-ACID battery FACTORY. AS 60% OF MEMBERS OF SMELTER WORKERS COHORT WERE HIRED AFTER 1950, FURTHER FOLLOW-UP OF THIS COHORT IS WARRANTED, IN ORDER TO DETERMINE MORE RELIABLY IF THERE IS AN EXCESS RISK. [IARC MONOGRAPHS. 1972-PRESENT V23 387 (1980)]

HTOX - In a cross-sectional study, the neurobehavioral effects of low-level lead exposure were evaluated in a group of 59 lead workers compared with 59 matched controls. The groups were not significantly different in age, education level, sleep pattern, or use of alcohol. The mean blood lead level in the exposed group was 2.37 umol/l (50 ug/100 ml) which was similar to the previous three years (2.36, 2.36, and 2.32 umol/l, respectively). The mean duration of exposure was 8109 hrs. Visual sensory function was affected, and perhaps as a consequence sustained attention and psychomotor tasks were performed more slowly by the lead exposed group. Cognitive functions were also impaired, with sensory store and short term memories, and learning abilities all showing deficits in lead workers. Multiple linear regression analysis relating to lead workers test performance and their lead exposure showed that performance on the sensory store memory test alone was significantly related to exposure. [Williamson AM, Teo RKC; Br J Ind Med 43: 374-80 (1986)]

HTOX - Battery workers (N= 18), who were exposed to high airborne lead levels, were compared/ with cement workers (N= 18), who were exposed to ambient lead levels. Blood lead urinary lead, semen lead, and zinc protoporphyrin concentrations were markedly elevated ($p< 0.001$) in battery workers. Battery workers had a significantly shifted ($p< 0.025$) frequency distribution of sperm count (median count, 45 vs 73x10x6 cells/cc, respectively). These results suggest a direct toxic effect of increased lead absorption on sperm production or transport in man. [Assennato G et al; Arch Environ Health 42 (2): 124 (1987)]

HTOX - Acute lead poisoning is relatively infrequent and occurs from ingestion of acid soluble lead compounds or inhalation of lead vapors. Local actions in the mouth produce marked astringency, thirst, and a metallic taste. Nausea, abdominal pain, and vomiting ensue. The vomitus may be milky from the presence of lead chloride. Although the abdominal pain is severe, it is unlike that of chronic poisoning. Stools may be black from lead sulfide, and there may be diarrhea or constipation. If large amounts of lead are absorbed rapidly, a shock syndrome may develop secondary to massive gastrointestinal loss of fluid. Acute central nervous system symptoms incl paresthesia, pain, and muscle weakness. An acute hemolytic crisis sometimes occurs and causes severe anemia and hemoglobinuria. The kidneys are damaged, and oliguria and urinary changes are evident. Death may occur in 1 or 2 days. If the patient survives the acute episode, characteristic signs & symptoms of chronic lead poisoning are likely to appear. [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1607]

HTOX - Signs & symptoms of chronic lead poisoning (plumbism) can be divided into 6 categories: gastrointestinal, neuromuscular, CNS, hematological, renal, & other. They may occur separately or in combination. The neuromuscular & CNS syndromes usually result from intense exposure, while the abdominal syndrome is a more common manifestation of a very slowly & insidiously developing intoxication. In the United States, the CNS syndrome is usually more common among children, while the GI syndrome is more prevelant in adults. /Inorganic lead/ [GOODMAN. PHARM

BASIS THERAP 7TH ED 1985 , p. 1607]

HTOX - ... /IN CHRONIC LEAD POISONING/ THE ABDOMINAL SYNDROME OFTEN BEGINS WITH VAGUE SYMPTOMS, SUCH AS ANOREXIA, MUSCLE DISCOMFORT, MALAISE, AND HEADACHE. CONSTIPATION IS USUALLY AN EARLY SIGN, ESP IN ADULTS, BUT DIARRHEA OCCASIONALLY OCCURS. A PERSISTENT METALLIC TASTE APPEARS EARLY IN THE COURSE OF THE SYNDROME. AS INTOXICATION ADVANCES, ANOREXIA AND CONSTIPATION BECOME MORE MARKED. INTESTINAL SPASM, WHICH CAUSES SEVERE ABDOMINAL PAIN, OR LEAD COLIC, IS THE MOST DISTRESSING FEATURE OF THE ADVANCED ABDOMINAL SYNDROME. The attacks are generally EXCRUCIATING. THE ABDOMINAL MUSCLES BECOME RIGID, & TENDERNESS IS ESP MANIFESTED IN THE REGION OF THE UMBILICUS. /INORGANIC LEAD/ [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1607]

HTOX - NEUROMUSCULAR SYNDROME OR LEAD PALSY IS NOW RARE IN THE United States. IT IS A MANIFESTATION OF ADVANCED SUBACUTE POISONING. MUSCLE WEAKNESS & EASY FATIGUE OCCUR LONG BEFORE ACTUAL PARALYSIS AND MAY BE THE ONLY SYMPTOMS. WEAKNESS OR PALSY MAY NOT BECOME EVIDENT UNTIL after EXTENDED MUSCLE ACTIVITY. THE MUSCLE GROUPS INVOLVED ARE USUALLY THE MOST ACTIVE ONES (EXTENSORS OF THE FOREARM, WRIST, & FINGERS & EXTRAOCULAR MUSCLES), & THE PALSY OFTEN OCCURS ONLY ON THE DOMINANT SIDE. WRIST DROP &, TO A LESSER EXTENT, FOOT DROP WITH APPROPRIATE HISTORY OF EXPOSURE HAVE BEEN CONSIDERED ALMOST PATHOGNOMONIC FOR LEAD POISONING. THERE IS USUALLY NO SENSORY INVOLVEMENT. DEGENERATIVE CHANGES IN MOTONEURONS & THEIR AXONS HAVE BEEN DESCRIBED. /INORGANIC LEAD/ [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1607]

HTOX - EXPOSURE TO LEAD OCCASIONALLY PRODUCES ... PROGRESSIVE MENTAL DETERIORATION IN CHILDREN. THE HISTORY OF THESE CHILDREN INDICATES NORMAL DEVELOPMENT DURING THE FIRST 12-18 MONTHS OF LIFE OR LONGER, FOLLOWED BY A STEADY LOSS OF MOTOR SKILLS & SPEECH. THEY MAY HAVE SEVERE HYPERKINETIC AND AGGRESSIVE BEHAVIOR DISORDERS & A POORLY CONTROLLED CONVULSIVE DISORDER. THE LACK OF SENSORY PERCEPTION SEVERELY IMPAIRS LEARNING. CONCN OF LEAD IN BLOOD EXCEED 60 UG/DL OF WHOLE BLOOD, & X-RAY MAY SHOW HEAVY, MULTIPLE BANDS OF INCR DENSITY IN THE GROWING BONES. ... AN INCREASED INCIDENCE OF HYPERKINETIC BEHAVIOR & A STATISTICALLY SIGNIFICANT, ALTHOUGH MODEST, DECREASE IN IQ HAVE BEEN SHOWN IN CHILDREN WITH BLOOD LEAD CONCN OF 30-50 UG/DL. /INORGANIC LEAD/ [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1608]

HTOX - Renal toxicity occurs in two forms: a reversible renal tubular disorder (usually seen after acute exposure of children to lead) and an irreversible interstitial nephropathy (more commonly observed in chronic industrial lead exposure). Clinically, a Fanconi like syndrome seen ... (proteinuria, hematuria, and casts in the urine; gylcosuria and aminoaciduria). In some patients, hyperuricemia may be associated with renal insufficiency. Histologically, lead nephropathy is revealed by a characteristic nuclear inclusion body, composed of a lead protein complex; this appears early & resolves after chelation therapy. Such inclusion bodies have been reported in the urine of workers exposed to lead in an industrial setting. /Inorganic lead/ [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1609]

HTOX - Other SIGNS & SYMPTOMS OF PLUMBISM ARE ASHEN COLOR OF FACE & PALLOR of LIPS; RETINAL STIPPLING; APPEARANCE OF "PREMATURE AGING," WITH STOOPED POSTURE, POOR MUSCLE TONE, & EMACIATION; & BLACK OR GRAYISH SO CALLED LEAD LINE ALONG THE GINGIVAL MARGIN. ... THE CARCINOGENICITY OF LEAD IN MAN IS NOT WELL ESTABLISHED, BUT IT HAS BEEN SUGGESTED, & SEVERAL CASE REPORTS OF RENAL ADENOCARCINOMA IN LEAD WORKERS HAVE BEEN PUBLISHED. /INORGANIC LEAD/ [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1609]

HTOX - The reproductive ability of men was ... shown to be adversely affected by moderate absorption of lead. Concn of lead in blood greater than 52

ug/100 ml were associated with a high frequency of altered spermatogenesis. Disorders of sexual dynamics were evident with blood lead values greater than 41 ug/100 ml. Among the workers with the highest concn of lead in blood (mean 74.50 + or - 26 ug/100 ml), 75% were judged to be hypofertile, 50% being even infertile. It was not possible, however, to demonstrate a reliable association between lead absorption in these men and the number of normal pregnancies per couple, or the frequency of miscarriages, ectopic pregnancies, or premature births. Nevertheless, these results were interpreted to indicate that lead clearly has a direct toxic action on the male gonads at relatively low levels of absorption. /Inorganic lead/ [Lancranjan I et al; Arch Environ Health 30: 396-401 (1975) as cited in NIOSH; Criteria Document: Inorganic Lead p.XI-52 (1978) DHEW Pub. NIOSH 78-158]

HTOX - ON BASIS OF CLINICAL OBSERVATION ... DISTURBANCES /OCCUR IN/ VISUAL CORTEX & SUPRAGENICULATE PATHWAY; OPTIC NERVE ... RETROBULBAR & BULBAR; RETINA; INTRAOCULAR MUSCLE; LENS; & EXTRAOCULAR MUSCLE. INDIVIDUAL PORTION OF VISUAL SYSTEM MAY BE AFFECTED ... MORE /OFTEN/ SEVERAL PORTIONS ... /LEAD CMPD/ [GRANT. TOX OF THE EYE 1974 , p. 621]

HTOX - Although per day children ingest less lead (Pb) in their diets and inhale less Pb than do adults, on a dose per body weight basis children may have 2-3 times the exposure. Furthermore children because of their poor oral hygiene are more likely than adults to absorb Pb from extraneous sources: ingestion of foreign objects, dust, paint chips, inhalation of resuspended settled dusts. Preliminary data indicate that children absorb 50% of ingested Pb, ie at a rate 5 times greater than adults. Animal studies support the hypothesis that the young retain more of a Pb dose than do adults and that this is reflected in soft tissue Pb levels. The portions of the Pb body burden found in soft tissues of children and adults are 27.5% and 5% respectively. Hence, a higher fraction of a child's body burden of Pb is available to produce toxic effects in soft tissues. [USEPA; Ambient Water Quality Criteria Doc: Lead p.C1-9 (1984) EPA 440/5-84-027]

HTOX - SRP: THE TOXICITY INFORMATION IN THIS RECORD CANNOT, IN MANY INSTANCES, BE ACCURATELY ASCRIBED TO METALLIC LEAD. MANY CITATIONS MAY BE BASED ON STUDIES USING LEAD SALTS. [CITATION]

HTOX - Lead is poisonous in all forms. ... Systemic lead poisoning can result from the inhalation of airborne lead particulate matter or fumes, or from the ingestion of lead /in its ionic form from/ water, food, etc. ... The ingestion of metallic lead may lead to an acute attack after a long (45 days in one case) asymptomatic period. Thus, the acute & chronic syndromes may merge ... . [GOSSELIN. CTCP 5TH ED. 1984 III-226]

HTOX - SYMPTOMATOLOGY: B. Lead encephalopathy in chronic lead poisoning. 1) Headache and insomnia. 2) Persistent vomiting, which is sometimes projectile. A typical lead colic may or may not be present. 3) Visual disturbances, choked optic disks. 4) Irritability, restlessness, delirium, hallucinations. 5) Convulsions and coma. 6) The intracranial pressure is characteristically high. The cerebrospinal fluid is generally unremarkable except for and elevation of total protein. 7) Death from exhaustion and respiratory failure. The mortality rate is high; Recovery is slow and frequently incomplete. /Lead cmpd/ [GOSSELIN. CTCP 5TH ED. 1984 III-233]

NTOX - In waterfowl, lead shot ingestion causes anorexia, loss of wt, weakness, lethargy, diarrhea, coma, & quiet death. ... Progressive tachycardia /has been observed/ in geese as the birds became more & more ill. Some geese developed ECG abnormalities. [BOOTH. VET PHARM THERAP 5TH ED 1982 , p. 1008]

NTOX - In geese chronically poisoned with lead shot, microscopic degenerative

lesions appear in the myocardium before any ECG abnormalities are manifested. Lesions include upper GI impaction with plant materials, emaciation, distended gallbladder, flabby hemorrhagic heart, discolored friable liver, & enteritis. Lesions in mallard ducks given lead shot include destruction of proventricular epithelium, bone medullary osteocytes, & pectoral muscle cells. Renal proximal tubules contain intranuclear inclusions. Mallard ducks also may develop encephalopathy & peripheral neuropathy. [BOOTH. VET PHARM THERAP 5TH ED 1982 , p. 1009]

NTOX - Metallic lead in the form of weights or foil can cause poisoning, as also can shot; duck frequently dredge up shot from the mud at the bottom of ponds. Lead shot in muscle is usually encapsulated, & systemic poisoning from it is unlikely. Nevertheless, lead shot dropped by anglers on river banks is a serious cause of poisoning in swans. ... Vegetation in the neighborhood of a smelter engaged in melting down old battery plates had a lead content of up to 3200 ppm. ... Vegetation near a busy highway may contain 500 ppm of lead due to contamination by exhaust fumes. It should be noted that in these cases the lead is only a surface contaminant; significant quantities of the element cannot be taken up by vegetation from lead-bearing soil, although the latter has itself caused poisoning in small animals. [CLARKE. VET TOX 1981 , p. 55]

NTOX - Birds (fowls, ducks, geese & pigeons) are all susceptible to lead poisoning. They show anorexia & ataxia, followed by excitement & loss of condition. Egg production, fertility, & hatchability decrease; & mortality may be high. [CLARKE. VET TOX 1981 , p. 57]

NTOX - It is doubtful whether the term "toxic dose" has any real meaning when when it is applied to a substance such as /elemental/ lead /in its ionic form/, as it is affected by so many different factors. Among these are environment, nutrition, disease & age, the last of these being considerably more important, as young animals are considerably more sensitive than old ones. There is also a seasonal variation. Over half the cases of lead poisoning in cattle in Scotland occur in the spring. Similar effects have been noted for dogs. ... Even more important is the fact that lead is both cumulative & ubiquitous. All living creatures are continually absorbing it, & the "toxic" dose is only the amount necessary to bridge the gap between this normal intake & a potentially dangerous level. ... Three or four lead shots will kill a duck, 10 a goose. [CLARKE. VET TOX 1981 , p. 56]

NTOX - The acute oral lethal single dose of lead in ... calves /is/ 50-600 mg/kg as lead or lead salts. ... Solid lead is not as toxic as /the/ more soluble salts, which are more readily absorbed. [BOOTH. VET PHARM THERAP 5TH ED 1982 , p. 1005]

NTOX - Eleven pregnant squirrel monkeys were perorally exposed to lead during the latter two-thirds of pregnancy (mean blood lead 0.54 ug/ml (2.61 umol/l), range 0.39-0.82 ug/ml (1.88-3.96 umol/l), at a dosing regime producing no maternal toxic symptoms. Lesions similar to lead encephalopathy and growth retardation of the fetal cerebrum were seen in some of the offspring, as well as neurological and behavioral symptoms at adult age. Cerebral lead levels in offspring (an abortion, stillborns, a sacrificed full-term fetus, and a neonatal death) were between 0.1-0.7 ug/g. Pre- and perinatal mortality, and prematurity, was increased, and the size of the offspring at birth was reduced. The head circumference tended to be reduced postnatally. [Logdberg MD et al; Scand J Work Environ Health 13: 135-45 (1987)]

NTOX - Behavorial aberrations are manifested, beginning with apparent anxiety or apprehension & proceeding to such things as hyperexcitability, bellowing or other vocalization, rolling of eyes & apparent fear or terror, possible belligerence, pressing of the head against a wall or

post, attempts to climb the wall, sudden jumping into the air, & frenzied or manical behavior. One fascinating aspect of this category is the effect of small amounts of lead in exptl animals. Lead can disrupt conditioned (learned) behavior in adult rats, rabbits & sheep. It can also disrupt learning & memory in young & adult rats & in lambs born of lead treated ewes. /Lead and inorganic lead compounds/ [BOOTH. VET PHARM THERAP 5TH ED 1982 , p. 1008]

NTOX - Levels of lead above the allowable drinking water standard of 50 ug/l are toxic, or result in morphological changes in aquatic vertebrates. ... Embryo and fingerling stages of fish are more susceptible to lead poisoning than adults. ... Fish are more susceptible to poisoning in soft water than in hard water. /Lead and inorganic lead comopunds/ [USEPA; The Health and Environmental Impacts of Lead: p.152 (1979) EPA 560/2-79-001]

NTOX - Animal studies indicate that relatively high levels of lead exposure interfere with resistance to infectious disease. [Gainer JH; Environ Health Perspect Exp 7: 113-9 (1974) as cited in USEPA; Ambient Water Quality Criteria Doc: Lead p.C-71 (1980) EPA 440/5-80-057]

ENVS - Lead is the fifth most important metal commercially in the United States and it may enter the environment during its mining, ore processing, smelting, refining use, recycling or disposal. Generally the initial means of entry is via the atmosphere. Lead may also enter the atmosphere from the weathering of soil and volcanos, but these sources are minor compared with anthropogenic ones. Generally the form of lead that enters the atmosphere is not determined. However metallic lead may be released from smelting and refining plants. If released or deposited on soil, lead will be retained in the upper 2-5 cm of soil, especially soils with at least 5% organic matter or a pH 5 or above. Leaching is not important under normal conditions although there is some evidence to suggest that Pb is taken up by some plants. Generally, the uptake of Pb from soil into plants is not significant. It is expected to slowly undergo speciation to the more insoluble sulfate, sulfide, oxide, and phosphate salts. Lead enters water from atmospheric fallout, runoff or wastewater; little is transferred from natural ores. Lead is a stable metal and adherent films of protective insoluble salts form that protect the metal from further corrosion. That which dissolves tends to form ligands. Lead is effectively removed from the water column to the sediment by adsorption to organic matter and clay minerals, precipitation as insoluble salt (the carbonate or sulfate, sulfide), and reaction with hydrous iron and manganese oxide. Under most circumstances, adsorption predominates. Lead does not appear to bioconcentrate significantly in fish but does in some shellfish such as mussels. When released to the atmosphere, lead will generally be in dust or adsorbed to particulate matter and subject to gravitational settling and be transformed to the oxide and carbonate. General lead exposure occurs from ambient air especially in areas with high automotive traffic and sites near industrial sources. However, the highest intake is from food and water. Concentrations in food may be elevated due to surface contamination of fresh fruits and vegetables. Food in soldered-tin cans may contain particularly high levels of lead. Elevated levels of lead in drinking water usually result from distribution systems containing lead pipe. (SRC) [CITATION]

USE- In the USA in 1984, 71.7% of the 1.2 million metric tons of lead metal consumed was used for batteries, 6.5% was used as an intermediate for gasoline antiknock additives, 6.4% for pigments and ceramics, 4.0% for ammunition, 2.0% for solder, 1.0% for cable covering, 0.3% for caulking, 2.3% for pipe and sheet, 0.18% for type metal, 0.6% for brass and bronze, 0.4% for bearings, 4.6% for miscellaneous uses(1). Estimates of lead dispersal into the environment indicate that the atmosphere is the major initial recipient(1). [(1) USEPA; Air Quality Criteria for Lead USEPA-600-8-83-028bF (1986) (2) USEPA; Industrial Process Profiles for Environmental Use. Chpt 27. USEPA-600/2-80-168 (1980) (3) Howe HE; Kirk-Othmer Encycl Chem Tech 3rd ed. NY, NY: Wiley 14: 98-139 (1981)]

FATE - TERRESTRIAL FATE: Lead is a extremely stable metal, although it dissolves in acid. While some corrosion may be expected in soil, generally an inert coat of an insoluble salt will form and limit further corrosion. It is expected to convert to more insoluble forms

such as PbSO4, Pb3(PO4)2, PbS, and PbO(1). It also forms complexes with organic matter and clay minerals that limits its mobility. Concentrations of Pb in soil solution reach a minimum between pH 5 and 6 and increase below pH 4 to 5 and above pH 6 to 7, because metal-organic complexes form in this pH range(2). Only a small fraction of lead in lead contaminated soil appears to be in water-soluble form, 0.2-1% according to one report, although the fraction soluble in EDTA ranged from 43-67% of the total lead in soil(3). The EDTA extractable material should include largely the chelated metal ions held in the soil organic and organomineral complexes although there is some evidence that the metal adsorbed on colloidal surfaces such as insoluble inorganic compounds may also be extracted(3). When 500 ppm Pb was added to soil as the soluble chloride, it became insoluble within an hour of contact with the soil, this insolubility lasted the duration of a 42-day experiment. After 42 days 0.7 and 70% were in the water-soluble and EDTA-soluble fractions, respectively(3). The percentage of lead in the water-soluble and EDTA-extractable fraction was similar when the insoluble PbO oxide was added to soil rather than the soluble chloride(3). [(1) USEPA; Health Effects Assessment for Lead. USEPA 540/1-86-055 (1984) (2) Bruemmer GW et al; Z Pflanzenernaehr Bodenk 149: 382-98 (1986) (3) Khan DH, Frankland B; Environ Pollut (B) 6: 15-31 (1983)]

FATE - AQUATIC FATE: If released into water, metallic lead will simply sink into the sediment. Surface layers of insoluble salts may form and protect the surface from further corrosion. In the dissolved state, it will form ligands, the dominant ones varying with pH(2). In freshwater systems, the most important ligands are HCO3, CO3, OH and (OH)2, whereas in seawater they are Cl, CO3, (OH), (OH)2, Cl2, and Cl3(2). A characteristic of lead is its tendency to form compounds of low solubility with the major anions of natural water. As a result natural concentrations of lead in lead-ore deposits do not move appreciably in ground or surface water but rather any dissolved lead will tend to combine with carbonate or sulfate ions to form insoluble lead carbonates or sulfates or be absorbed by ferric hydroxide(1). [(1) USEPA; Air Quality Criteria for Lead; pp. 6-1 to 6-28 USEPA-600/8-77-017 (1977) (2) Long DT, Angino EE; Geochim Cosmochim Acta 41: 1183-91 (1977)]

FATE - AQUATIC FATE: Precipitation has been shown to be important(2), at relatively high pH. The amount of lead that can remain in solution in water is a function of the pH of the water and the dissolved salt content which is about 30 ug/l in hard water (pH > 5.4) and about 500 ug/l in soft water (pH <5.4)(1). Much of the lead carried by river water is in the form of suspended solids. One study of the distribution of lead between filtrate and solids in stream water from urban and rural areas reported the ratio of lead in suspended solids to that in filtrate varied from 4% in rural areas to 27% in urban areas(1). [(1) USEPA; Air Quality Criteria for Lead; pp. 6-1 to 6-28 USEPA-600/8-77-017 (1977) (2) Callahan MA et al; Water-related Environmental Fate of 129 Priority Pollutants Vol 1. USEPA-440/4-79-029a 13-1 to 13-19 (1979)]

FATE - AQUATIC FATE: Sorption also appears to be an important process in removing lead from both fresh and estuarine natural waters into sediment(1). The amount adsorbed depends on parameters such as the availability of ligands, pH, redox conditions, salinity, iron concentration, composition of dissolved particulate matter and sediment, and lead concentration(1). Lead is adsorbed by polar particulate matter as is evidenced by its dominance in sediment of

specific gravity 2.0-2.9, where the clay fraction is found. It is almost absent from less dense sediment, characterized by organic matter not active in complex formation or denser fractions, characterized by precipitation(1). Another study showed that the organic content of the bottom mud was the most significant factor affecting adsorptivity(2). [(1) Callahan MA et al; Water-related Environmental Fate of 129 Priority Pollutants Vol 1. USEPA-440/4-79-029a p. 13-1 to 13-19 (1979) (2) Tada F, Suzuki S; Water Res 16: 1489-94 (1982)]

FATE - ATMOSPHERIC FATE: Lead released to the atmosphere will be in particulate matter and be subject to washout and gravitational settling. Transformations in the atmosphere to the carbonate and oxide may be expected. Estimates of the annual net Pb deposition to the tropical North Pacific Ocean is 2.0 and 5.0 ng/sq cm, respectively and is predominately from noncrustal sources(2). Recycled sea spray represents a significant but variable component of the deposition. The average annual scavenging ratio (concn in precipitation (mg/l) to air concn (ug/cu m)) for Pb is 0.18X10+6, the lowest value of the 7 trace metals studied(1). The mean ratio of wet to dry deposition of lead in southern, central and northern Ontario is 1.63, 1.99, and 2.50, respectively(1). [(1) Chan WH et al; Water Air Soil Pollut 29: 373-89 (1986) (2) Arimoto R et al; J Geophys Res 90: 2391-408 (1985)]

PLNT - Natural constituent in all plants with normal concentration in leaves and twigs of woody plants 2.5 ppm, pasture grass 1.0 ppm; cereals 0.1-1.0 ppm(1). Some trees have the capacity to accumulate large amounts of lead from contaminated soil - the tips of larch, pine, and fir contained 100 ppm lead when grown in lead mining areas with soil concentration appreciablly different from the usual concentration (80,000 ppm) in most soils. However, in most cases this indicates that there is no significant bioconcentration of lead from soil into plants(1). Lead on leafy parts of plants result from deposition of Pb from air(1). An experiment performed with hydroponically grown corn showed that lead was taken up and precipitates in the cell walls(3). Aquatic plants from the Chesapeake Bay region 2.2-18.9 ppm dry weight(4). Aquatic bryophytes from the Pb/Ag mining district of mid-Wales contained 34-49,400 ppm dry weight which correlated well with the concn of dissolved Pb in the streams(2). [(1) IARC; Monograph Some Metal and Metallic Compounds 23: 325-415 (1980) (2) James KC et al; Water Air Soil Pollut 24: 329-38 (1985) (3) USEPA; Air Quality Criteria for Lead; p. 6-1 to 6-28 USEPA-600/8-77-017 (1977) (4) DiGiulio RT, Scanlon PF; Sci Tot Environ 41: 259-74 (1985)]

ANML - Principal source of exposure to ducks and waterfowl is from lead shot which is ingested by the birds in search of gravel. Livers of 28 species of birds with no known lead exposure 0.3-7 ppm(2). Small mammals within 10 m of road 2.6-15.2 ppm with the contents higher along high traffic road and among insectivores(3); urban biota 11-367 ppm, rural biota 4.7-16 ppm(4). Riparian wildlife from sites in the active New Lead Belt mining district of southeastern Missouri (species - geometric mean in ppm wet weight (range in ppm)): Bullfrog carcasses - 1.22-1.47 (not detected - 7.40), Northern water snake carcasses 0.15-1.21 (not detected - 3.90), Rough-winged swallow carcasses - 0.23-2.39 (not detected - 61.2), Muskrat livers - 0.07-0.26 (not detected - 0.53)(1). [(1) Niethammer KR et al; Arch Environ Contam Toxicol 14: 213-23 (1985) IARC; Monograph Some Metals and Metallic Compounds 23: 325-415 (1980) (3) National Science Foundation; Lead in the Environment; Boggers WR ed NSF/RA-770214 (1977) (4) International Register of Potentially Toxic Chemicals; p. 171-77 United Nations Environment Program Geneva Switzerland (1979)]

FDWS - EPA 15 ug/l (Action Level) [USEPA. SUM STATE FED DRINK WATER Stds Guide 1993]

ANTIMONY

SY - ANTIMONY BLACK
SY - ANTIMONY, REGULUS
SY - ANTYMON (POLISH)
SY - CI 77050
SY - REGULUS OF ANTIMONY
SY - STIBIUM
MF - Sb
MMFG - The antimony content of commercial ores ranges from 5-60%, and determines the method of recovery. In general, the lowest grades of sulfide ores, 5-25% antimony, are volatized as oxides; 25-40% antimony ores are smelted in a blast furnace; and 45-60% antimony ores are liquated or treated by iron precipitation. The blast furnace is generally used for mixed sulfide and oxide ores, and for oxidized ores containing up to about 40% antimony; direct reduction is used for rich oxide ores, and complex ores are treated by leaching and electrolysis. [KIRK-OTHMER. ENCYC CHEM TECH 3RD ED 1978-PRESENT 3(78) 98]
USE - MANUFACTURE OF WHITE METAL, TYPE, BULLETS, BEARING METAL; IN FIREWORKS, THERMOELECTRIC PILES; COATING METAL, BLACKENING IRON [MERCK INDEX. 10TH ED 1983 , p. 102]
USE - High purity antimony (>99.999%) has a limited but important application in the manufacture of semiconductor devices. When alloyed with elements of Group III-A, the III-V compounds are formed; these have important applications as infrared devices, diodes, and Hall effect devices. [KIRK-OTHMER. ENCYC CHEM TECH 3RD ED 1978-PRESENT 3(78) 103]
USE - PRINCIPALLY IN ALLOYS WITH LEAD FOR STORAGE BATTERY GRIDS; IN ALLOYS WITH OTHER METALS FOR ELECTRICAL & OTHER USES; CHEM INTERMED FOR OTHER ANTIMONY AGENTS, FIRE RETARDANT CMPD, CERAMIC & GLASS ADDITIVES, PAINT PIGMENTS, RUBBER VULCANIZATION AGENTS [SRI]
CPAT - (PRIMARY ANTIMONY): 44% WAS USED IN TRANSPORTATION APPLICATIONS, MOSTLY STORAGE BATTERY GRIDS; 15% AS A CHEM INTERMED FOR FIRE-RETARDANT CMPD; 9% AS A CHEM INTERMED FOR RUBBER VULCANIZATION AGENTS; 6% AS A CHEM INTERMED FOR CERAMIC & GLASS ADDITIVES; 17% AS A CHEM INTERMED FOR other ANTIMONY CMPD; 5% IN ALLOYS FOR MACHINERY; & 4% IN MISC USES (1974) [SRI]
CPAT - FLAME RETARDANTS, 60%; TRANSPORTATION INCLUDING BATTERIES, 10%; CERAMICS & GLASS, 10%; CHEMICALS, 10%; AND OTHER, 10% (1985) [BUREAU OF MINES. MINERAL COMMODITY SUMMARIES 1986 p.10]
BP - 1635 DEG C [MERCK INDEX. 10TH ED 1983 , p. 102]
MP - 630 DEG C [MERCK INDEX. 10TH ED 1983 , p. 102]
SOL - INSOL IN HOT & COLD WATER [WEAST. HANDBK CHEM & PHYSICS 69TH ED 1988-1989 B-72]
VAP - 1 MM HG AT 886 DEG C [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 300]
DOT - Health Hazards: Poisonous if swallowed. Inhalation of dust poisonous. Fire may produce irritating or poisonous gases. Runoff from fire control or dilution water may cause pollution. /Antimony, powder/ [DOT. EMERGENCY RESPONSE GUIDEBOOK 1987 G-53]
TOXC - MODERATELY DANGEROUS; WHEN HEATED ... IT EMITS TOXIC FUMES. [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 300]
REAC - ... ON CONTACT WITH ACID, IT EMITS TOXIC FUMES OF ANTIMONY HYDRIDE. [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 300]
REAC - ANTIMONY IS SPONTANEOUSLY FLAMMABLE IN FLUORINE, CHLORINE, OR BROMINE. WITH IODINE, REACTION PRODUCES HEAT, WHICH CAN CAUSE FLAME OR EVEN EXPLOSION IF QUANTITIES ARE GREAT ENOUGH. [NFPA. FIRE PROTECT GUIDE HAZARD MATLS 9TH ED 1986 491M-25]
REAC - AVOID CONDITIONS IN WHICH NASCENT HYDROGEN WILL REACT WITH ANTIMONY TO FORM STIBINE (SBH3) WHICH IS EXTREMELY TOXIC. ... [MERCK INDEX. 10TH ED 1983 , p. 102]
o EMCE - CLINICAL EFFECTS :

- Metal fume fever is an illness produced by inhaling freshly produced metal oxides. These oxides are produced by heating various metals including cadmium, zinc, magnesium, copper, antimony, nickel, cobalt, manganese, tin, lead, beryllium, silver, chromium, aluminum, selenium, iron, and arsenic. The two most common agents involved are zinc and copper.
- Flushing, dry throat, blurred vision, and stiff neck have been reported. A sweet or metallic taste is also frequently seen.

CARDIOVASCULAR

- Myocardial injury was reported in one case.

RESPIRATORY

- Shortness of breath, chest tightness, rales in the midzone and basal regions, a non-productive cough, dyspnea, expiratory wheeze, and diffuse rales have all been reported. Sequelae and lung lesions are uncommon.

NEUROLOGIC

- Headache, myalgias, weakness, and paresthesias in both feet have been reported.

GASTROINTESTINAL

- Various non-specific GI effects may be manifested including anorexia, constipation or diarrhea, nausea, vomiting, and abdominal pain.

TEMPERATURE REGULATION

- Fever is common and is usually mild, but has reached temperatures of 104 degrees F. Initially, there is often chills and shivering.

HEMATOLOGIC

- Leukocytosis is almost always present.

DERMATOLOGIC

- Hives may occur.

MUSCULOSKELETAL

- Muscle injury may be seen.

IMMUNOLOGIC

- Anaphylactoid reactions with urticaria and angioedema may be noted.

OTHER

- Lactic dehydrogenase is often elevated, especially the pulmonary fraction.
- BLOOD: Elevated pulmonary isoenzyme for lactic acid dehydrogenase and elevated zinc levels have occurred after exposure to zinc oxide fumes. Elevated blood copper levels have also been reported.
- In the majority of cases, little treatment is needed beyond supportive care. Bedrest, analgesics, and antipyretics are administered to ease a patient through the one to two days of symptoms. Unless the base metal and its coatings are known, a heavy metal screen may be warranted in order to prevent or treat acute toxicity from a more toxic metal moiety.
- No serious ocular toxicity has been reported from metal fume fever.
- Acute antimony poisoning in animals is characterized by marked weight loss, hair loss, dry scaly skin, eosinophilia and myocardial failure. Exposure to antimony salts may result in irritation of skin and mucous membranes.
- Acute oral exposures are unusual since there are few oral preparations. The limited use of antimony compounds for treatment of schistosomes also tends to make the exposure unlikely in developed countries. Most exposures are of a chronic nature and related to occupational exposures. Tartar emetic is still used to treat Schistosomiasis japonicum, but is used IV because of its oral toxicity.

HEENT

- Conjunctivitis may be seen after exposure to antimony salts as may pharyngitis, laryngitis, rhinitis and nose bleed.
- Nasal perforation from occupational exposure has been reported.

CARDIOVASCULAR

- Animals have shown myocardial failure after being fatally poisoned by antimony. The sulfide salts are much more prone to produce this effect.
- Thrombophlebitis is common after IV use, but has even been reported when poisoning is by the oral route.

RESPIRATORY

- o COUGH: Antimony salts are known to be mucous membrane irritants, and may cause pulmonary irritation. Metal fume fever has also been reported with this agent.

GASTROINTESTINAL

- o Vomiting, diarrhea and stomatitis have been reported after exposure to antimony and its salts.

HEMATOLOGIC

- o EOSINOPHILIA: Has been seen in acutely and fatally poisoned animals. There are reports of leukopenia occurring in the treamtent of Leishmaniasis with antimony compounds.

DERMATOLOGIC

- o Hair loss, dry skin and antimony spots have all been seen after exposure to antimony. Dermatitis is rare following exposure to metallic antimony.

OTHER

- o Some antimony salts may cause liver toxicity. Monitor liver enzymes if patients have had significant exposures.

RANGE OF TOXICITY

- o An estimated toxic amount of the organic antimony tartar emetic (non elemental antimony) is 0.1 to 1 grams.
- o Workers exposed to air having about 3 mg of antimony per cubic meter showed urinary valves ranging from 0.8 to 9.6 mg/L.
- o The TLV-TWA is 0.5 mg/m(3).

HTOX - Chronic effects due to antimony are alterations of the ECG, especially T-wave abnormalities, myocardial changes, pneumoconiosis, but also pneumonitis, tracheitis, laryngitis, bronchitis, pustular skin eruptions called antimony spots, and contact allergy to the metal. [SEILER. HDBK TOXICITY INORGANIC CPDS 1988 , p. 71]

HTOX - ANTIMONY DUST IN FOUNDRY WAS IMPORTANT CAUSE OF GASTROINTESTINAL SYMPTOMS ... /IN/ EXPOSED WORKERS. ... DYSPNEA, HEADACHE, VOMITING, CONJUNCTIVITIS ... BLOODY PURULENT DISCHARGE FROM NOSE ... . [HAMILTON. INDUS TOX 3RD ED 1974 , p. 27]

HTOX - Oral. ... Seventy people who drank lemonade from preparations left overnight in white enamelware buckets (the enamel contained 2.88% antimony trioxide) became ill. Antimony trioxide had been leached from the enamel by the acidic lemonade. Fifty-six people were hospitalized, suffering from burning stomach pains, colic, nausea and vomiting; most recovered within 3 hours. Analysis found that the lemonade contained 0.013% antimony. Each person ingesting 300 ml lemonade would have received 36 mg antimony, which is similar to an emetic dose listed in the British Pharmacopoeia. [Dunn JJ; Analyst 53: 532-33 (1928) and Monier-Williams GW; Report on Public Health and Medical Subjects No. 73 Ministry of Health, London (1934) as cited in USEPA; Health Effects Assessment for Antimony and Compounds p.7 (1987) EPA 600/8-88/018]

HTOX - Women working in an antimony metallurgical plant were compared to a similar group of women not exposed to antimony. The exposed group had higher incidences of spontaneous abortions (12.5 vs 4.1%), premature births (3.4 vs 1.2%), and gynecological problems (77.5 vs 56%). the gynecological problems included menstrual cycle disorders (61.2 vs 35.7%), inflammatory disease (30.4 vs 55.3%) and other reproductive problems (8.4% of exposed women). These women were exposed to unspecified amounts of antimony trioxide, metallic antimony, and antimony pentasulfides. Children born to these exposed women weighed significantly less after 1 year when compared with children born to nonexposed women; there were no statistically significant differences in weights at birth of the two sets of children. [Belyaeva AP; Gig Truda Prof Zabole 11:32 (1964) as cited in Health Effects Assessment for Antimony and Compounds p.13-14 (1987) EPA 600/8-88/018]

HTOX - BECAUSE OF ASSOCIATION WITH LEAD AND ARSENIC IN INDUSTRY, IT IS OFTEN DIFFICULT TO ASSESS THE TOXICITY OF ANTIMONY ... . /ANTIMONY CMPD/ [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 301]

HTOX - It is known that some antimony miners have developed a disabling form of silicosis but owning to the fact that antimony is not permanently retained in the lung but is constantly

excreted in small amounts in the urine and feces, it is generally believed that this is a benign condition. [BROWNING. TOX INDUS METALS 2ND ED 1969 , p. 32]

HTOX - ANTIMONY AND ITS COMPOUNDS REPORTED TO CAUSE DERMATITIS, KERATITIS, CONJUNCTIVITIS, & NASAL SEPTAL ULCERATION BY CONTACT, FUMES OR DUST. /ANTIMONY AND ANTIMONY CMPD/ [MERCK INDEX. 10TH ED 1983 , p. 102]

NTOX - MINIMUM LETHAL IP DOSE OF ANTIMONY METAL ... FOR RATS ... 10 MG ... /100 G BODY WT/. ANIMALS DYING A FEW DAYS AFTER INJECTION SHOWED DYSPNEA, LOSS OF WT, GENERAL WEAKNESS, LOSS OF HAIR, AND EVIDENCE OF MYOCARDIAL INSUFFICIENCY. THE MOST MARKED PORT-MORTEM FINDINGS WERE CARDIAC LESIONS--MYOCARDIAL CONGESTION AND DILATATION OF RIGHT HEART. ... DEATH WAS ATTRIBUTED TO MYOCARDIAL EDEMA WITH MARKED HYPEREMIA AND CAPILLARY ENGORGEMENT. LIVERS ALSO SHOWED CONGESTION WITH SOME DEGENERATION AND POLYMORPHONUCLEAR INFILTRATION. ... MODERATE HYPERPLASIA OF THE SPLEEN, WITH SOME EOSINOPHILIA /WAS ALSO PRODUCED/. [BROWNING. TOX INDUS METALS 2ND ED 1969 , p. 29]

NTOX - ANTIMONY IS MUTAGENIC IN BACTERIA OR PHAGE. IT WAS SHOWN TO INDUCE CHROMOSOMAL ABERRATIONS OR ABNORMAL CELL DIVISIONS IN ANIMAL OR PLANT CELLS. [FLESSEL CP; ADV EXP MED BIOL 91: 117-28 (1977)]

NTOX - ... ABORTIONS WERE FREQUENT IN RABBITS FED DOSES OF METALLIC ANTIMONY OF 5 TO 55 MG EVERY OTHER DAY FOR 30, 60, AND 90 DAYS ... . [PATTY. INDUS HYG & TOX 3RD ED VOL2A,2B,2C 1981-82 , p. 1513]

NTOX - ... A METALLIC ANTIMONY (LESS THAN OR EQUAL TO 5 UM DIAMETER) /ADMIN/ BY INHALATION AT DOSE OF 50 MG/KG TO 30 FEMALE RATS. ONLY 15 RATS BECAME PREGNANT, SOME ONLY AFTER MULTIPLE MATINGS. THESE RATS PRODUCED FEWER OFFSPRING (5.4 VERSUS 7.8) THAN CONTROLS. [NRC. DRINKING WATER & HEALTH VOL3 1980 , p. 80]

NTOX - Signs of acute antimony poisoning /in rats/ were marked weight loss, loss of hair, and dry, scaly appearance of the skin. Eosinophilia /was/ the hematologic finding. Pathological findings consisted of acute congestion of the heart, liver, & kidneys. Death was from myocardial failure. This effect on heart muscle was felt ... to be diagnostic of antimony poisoning. Tissue analyses indicated very little storage of antimony. /Antimony and antimony cmpd/ [PATTY. INDUS HYG & TOX 3RD ED VOL2A,2B,2C 1981-82 , p. 1510]

NTOX - Three groups of 8 month old Wistar derived rats (90 males and 90 females per group) were exposed by inhalation to either antimony trioxide (time-weighted average (TWA) 45 mg/cu m), antimony ore concentrate (TWA 36 + 40 mg/cu m), or filtered air (controls) for 7 hr/day, 5 day/wk, for up to 52 wk and sacrificed 20 wk after terminating exposures. The concentraion of antimony (Sb) in the lung of male rats (38,300 ug Sb/g) exposed to antimony trioxide was significantly greater than that in female rats (25,000 ug/g) exposed to antimony trioxide. The lung of both male and female rats exposed to antimony trioxide contained significantly more Sb than the lungs of males and females exposed to Sb ore (approx 5 times greater). The most significant findings were the presence of lung neoplasms in 27% of females exposed to antimony trioxide and 25% of females exposed to Sb ore concentrate. None of the male rats in any group or the female controls developed lung neoplasms. There were no significant differences in incidence of cancer of other organs between exposed and control rats. [Groth DH et al; J Toxicol Environ Health 18: 607-26 (1986)]

ADE - ANTIMONY MAY ENTER BODY ... THROUGH LUNG. FROM LUNG ... FREE ANTIMONY, IS ABSORBED & TAKEN UP BY BLOOD & TISSUES. /ANTIMONY AND CMPD/ [ENCYC OCCUPAT HEALTH & SAFETY 1983 , p. 177]

ADE - Environment and nutrition influence antimony concentrations of human tissues, causing large differences among individuals. Extremely high levels were found in the lung and bone tissues but not in liver or kidney of deceased antimony smelter workers. The lung contain the highest antimony levels of human tissues. The distribution of antimony

is not homogeneous within organs or tissues. ... Great variations of antimony content were found in normal human platelets, serum albumin, and different tissues. /Antimony and cmpd/ [SEILER. HDBK TOXICITY INORGANIC CPDS 1988 , p. 71]

ADE - A STUDY OF THE RETENTION PATTERNS OF LABELED ANTIMONY IN MICE FOLLOWING INHALATION OF PARTICLES FORMED AT DIFFERENT TEMP WAS CONDUCTED. THE LOWER TEMP AEROSOL WAS MORE SOL & LEFT THE LUNG RAPIDLY, LOCALIZING IN THE SKELETON. THE 2 AEROSOLS PRODUCED AT HIGHER TEMP RESULTED IN (124)ANTIMONY REMAINING IN THE LUNG FOR EXTENDED PERIODS. [THOMAS RG ET AL; PROC SOC EXP BIOL MED 144 (2): 544-50 (1973)]

ADE - A STUDY OF ACCUMULATION OF ANTIMONY IN BLOOD & CERTAIN ORGANS OF RATS WITH A GRAFTED SARCOMA-45 TUMOR WAS CONDUCTED. AT 1 & 2 DAYS POST-ADMINISTRATION METALLIC ANTIMONY (6 IP DOSES OF 50 MG/KG EACH) WAS PRESENT IN HIGHER CONCN IN BLOOD OF SARCOMA 45-BEARING RATS THAN IN THE BLOOD OF NORMAL RATS. ANTIMONY DID NOT SELECTIVELY ACCUMULATE IN THE TUMOR TISSUE. HIGHEST LEVELS WERE OBSERVED IN MUSCLES, LUNG, & SKIN OF TUMOR-BEARING RATS & IN LUNG & SKIN OF NORMAL RATS. [TRIFONOVA NF; VOP KLIN EKSP ONKOL 8: 283-5 (1972)]

ADE - Tissue concn of antimony in lung, liver, and kidney tissue from a group of deceased smelter workers from northern Sweden were compared with those of a group of persons without occupational exposure from a nearby area. Antimony concn of lung tissue from exposed workers were 12 fold higher than those of referents ($p < 0.001$). For lung tissue there was no tendency towards decr antimony concn with time (up to 20 yr) after the cessation of exposure. There was no significant difference between the antimony concn of the lung tissue from workers who had died of lung cancer and those of persons who died of other malignancies, cardiovascular disease or other causes. This finding did not rule out the possibility of a role for antimony in the etiology of lung cancer among smelter workers since multiple factors may have been operating. [Gerhardsson L et al; Scand J Work Environ Health 8 (3): 201-8 (1982)]

FDWS - EPA 6 ug/l [USEPA. SUM STATE FED DRINK WATER STDS GUIDE 1993]

WQCHU- Water and Fish Consumption: 1.46E+2 ug/L
Fish Consumption Only: 4.5E+4 ug/L

Discussion -- The WQC of 1.46E+2 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 4.5E+4 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

WQCAQ-
Freshwater:	Acute --	8.8E+1 ug/L
	Chronic --	3.0E+1 ug/L
Marine:	Acute --	1.5E+3 ug/L
	Chronic --	5.0E+2 ug/L

MCLG - 0.003 mg/L (Proposed, 1990)

Discussion -- EPA is proposing to regulate antimony based on its potential adverse effects (decreased longevity and altered blood cholesterol and glucose) reported in a lifetime oral exposure study in rats. The MCLG is based upon a DWEL of 0.015 mg/L and an assumed drinking water contribution of 20 percent.

MCL - 0.01 mg/L (Proposed, 1990)

Discussion -- EPA proposes an MCL of 0.01 mg/L based upon a PQL of 10x the MDL. EPA also proposes as an alternative option an MCL of 0.005 based on a PQL of 5x the MDL.

NAME - ARSENIC
SY - ARSENIC BLACK
SY - ARSENIC-75
SY - COLLOIDAL ARSENIC
SY - Gray arsenic
SY - Metallic arsenic
USE - ALLOYING CONSTITUENT [SRI]
USE - MFR OF CERTAIN TYPES OF GLASS; IN METALLURGY FOR HARDENING COPPER, LEAD ALLOYS [MERCK INDEX. 10TH ED 1983 , p. 116]
USE - TO MAKE GALLIUM ARSENIDE FOR DIPOLES & OTHER ELECTRONIC DEVICES; DOPING AGENT IN GERMANIUM & SILICON SOLID STATE PRODUCTS; SPECIAL SOLDERS; MEDICINE [SAX. HAWLEY'S CONDENSED CHEM DICT 11TH ED 1987 , p. 98]
USE - COMPONENT OF ALLOYS; COMPONENT OF ELECTRICAL DEVICES [SRI]
USE - MEDICATION: TO MFR ARSENICAL ORG CMPD FOR THERAPEUTIC USE [BROWNING. TOX INDUS METALS 2ND ED 1969 , p. 41]
USE - (76)As radioactive tracer in toxicology [MERCK INDEX. 10TH ED 1983 , p. 116]
USE - Used as a catalyst in the manufacture of ethylene oxide. [KIRK-OTHMER: ENCYC CHEM TECH 3RD ED 1978-PRESENT 9(80) 455]
USE - Used in semiconductor devices [IARC MONOGRAPHS. 1972-PRESENT V23 49 (1980)]
BP - NO DATA
MP - 817 DEG C @ 28 ATM [WEAST. HDBK CHEM & PHYS 68TH ED 1987-1988. B-73]
SOL - Sol in nitric acid; insol in water [WEAST. HDBK CHEM & PHYS 68TH ED 1987-1988. B-73]
SOL - INSOL IN CAUSTIC AND NONOXIDIZING ACIDS [SAX. HAWLEY'S CONDENSED CHEM DICT 11TH ED 1987 , p. 98]
o EMCE - CLINICAL EFFECTS :
ACUTE EXPOSURE

- o Acute arsenic ingestion generally produces symptoms within 30 minutes but may be delayed for several hours if ingested with food. Garlic-like odor of breath and feces may occur. Dehydration, intense thirst, vomiting, diarrhea, and fluid-electrolyte disturbances are common. Hypovolemia from capillary leaking (third-spacing of fluids) is a common early sign. Arsenic compounds are primarily absorbed through the GI tract but may be absorbed dermally or by inhalation. ACUTE inhalation exposures have resulted in irritation of the upper respiratory tract.
- o DELAYED EFFECTS: After absorption, arsenic may cause multi-organ failure by inhibiting sulfhydryl containing enzymes within cells. The primary target organs initially are the gastrointestinal tract, the heart, brain, and kidneys. Eventually the skin, bone marrow, and peripheral nervous system may be significantly damaged.
- o CHRONIC POISONING: Chronic inhalation of inorganic arsenic compounds is the most common cause of industrial poisoning. The sequence of chronic poisoning involves weakness, anorexia, and gastrointestinal complaints, followed by conjunctivitis and irritation of the throat and respiratory tract, perforation of the nasal septum, hyperkeratosis, hyperpigmentation, eczemoid and allergic dermatitis. A hoarse voice is characteristic of an arsenic worker, and a perforated nasal septum is a common result of prolonged inhalation of white arsenic dust or fume. The final phase consists of peripheral sensory neuritis of the hands and feet and sometimes motor paralysis.

VITAL SIGNS ACUTE EXPOSURE

- o Hypotension and tachycardia are common early signs.

HEENT ACUTE EXPOSURE

- o Trivalent arsenic is corrosive to the eyes, mouth, and mucous membranes. Perforation of the nasal septum can occur.

CARDIOVASCULAR ACUTE EXPOSURE

- o Hypovolemic or hemorrhagic shock, ventricular fibrillation or tachycardia, QT prolongation, and T-wave changes may be seen after acute ingestion. Myocarditis has been reported after chronic poisoning.

RESPIRATORY ACUTE EXPOSURE

- o Airway irritation may occur. Pulmonary edema and respiratory failure may develop with severe poisoning.

NEUROLOGIC ACUTE EXPOSURE

- o Altered mental status, seizures, toxic delirium, encephalopathy, and delayed peripheral neuropathy are complications of acute arsenic poisoning.

GENITOURINARY ACUTE EXPOSURE

- o Hematuria and acute tubular necrosis may occur.

FLUID-ELECTROLYTE ACUTE EXPOSURE

- o Rapid volume depletion from vomiting, diarrhea, and third spacing of fluids is common.

HEMATOLOGIC ACUTE EXPOSURE

- o Acute hemolysis, decreased hematocrit, and decreased hemoglobin may occur after acute poisoning. Pancytopenia, aplastic anemia, or leukemia may occur following chronic exposure. Bone marrow depression can occur.

DERMATOLOGIC ACUTE EXPOSURE

- o Skin findings may include hyperpigmentation, keratoses, and epidermoid carcinomas. Mee's lines of the nails are common. Trivalent arsenic compounds are corrosive to the skin. Arsenic trioxide and pentoxide are sensitizers.

MUSCULOSKELETAL ACUTE EXPOSURE

- o Muscular cramps may occur.

REPRODUCTIVE HAZARDS

- o Inorganic arsenic crosses the placenta and was reported to cause death without early chelation therapy.

GENOTOXICITY

- o Arsenic induced DNA damage in human cells.

HTOX - ... REPORTED 45 CASES OF LUNG CANCER AND 2 OF SKIN CANCER AMONG AN UNSTATED NUMBER OF WORKERS @ NI REFINERY. MATERIALS HANDLED WERE NICKEL (NI)AND COBALT (CO) ORES WITH SUBSTANTIAL ARSENIC (AS) CONTENT (RANGE 15-50%). ... AMONG PERSONNEL NOT ENGAGED IN PRODUCTION ONLY 1 CASE. OTHER AGENTS IN ENVIRONMENT INCLUDED BENZPYRENE AND SULFUR DIOXIDE (SO2). (ROCKSTROH, 1959) [IARC MONOGRAPHS. 1972-PRESENT V2 66 (1973)]

HTOX - Excess incidence of lung cancer in non-ferrous smelter workers; exposure to arsenic was considered to be a contributing factor. [Lee AM, Fraumeni JF; J Natl Cancer Inst 42 (6): 1045 (1969) as cited in USEPA; Ambient Water Quality Criteria Doc: Arsenic p.C-104 (1980) EPA 440/5-80-021]

HTOX - Although arsenic may not act as a direct agent of visceral cancer, /there have been many/ cases of secondary carcinoma of internal organs, eg colon, bladder, gallbladder, pancreas, liver, ureter, prostate, lymph nodes, and bronchi as a consequence of metastases of primary skin cancer induced by arsenic exposure (including ingestion, injection or inhalation). [Nat'l Research Council Canada; Effects of Arsenic in the Canadian Environment p.273 (1978) NRCC No.15391]

HTOX - Genitourinary effects: Hematuria, albuminuria, anuria. [ITI. TOX & HAZARD INDUS CHEM SAFETY MANUAL 1982 , p. 51]

HTOX - Arsenic associated lung cancers are usually the poorly differentiated type of epidermoid bronchogenic carcinoma. Worker groups with diagnosed lung cancer were studied in copper smelting communities in Montana. Of 25 smelter workers, 4 had well differentiated epidermoid carcinomas, 10 had poorly differentiated epidermoid carcinomas, 7 had small cell undifferentiated epidermoid carcinomas, and 3 had acinar-type adenocarcinomas. [USEPA; Health Assessment Document: Inorganic Arsenic p.7-71 (1984) EPA 600/8-83-021]

HTOX - Among smelter workers exposed to a mixture of metals including arsenic, the frequency of congenital malformations did not differ from non-exposed populations. However, mean birth wt were reported to be decr in offspring of female employees of the smelter. [FRIBERG. HDBK TOX OF METALS 2ND ED VOLS I II 1986 V1 414]

HTOX - IN INDUSTRY, ACUTE POISONING FROM SOLID ARSENIC IS RARE; SUBACUTE AND CHRONIC POISONING USUALLY ARISES FROM EXPOSURE TO ARSENIC CONTAINING DUST AND FUME. [BROWNING. TOX INDUS METALS 2ND ED 1969 , p. 43]

HTOX - ... HAS BEEN SUSPECTED OF INFLUENCING THE INCREASED RISK OF LUNG CANCER IN SMOKERS ... THIS VIEW IS NOT GENERALLY ACCEPTED. [IARC MONOGRAPHS. 1972-PRESENT V2 68 (1973)]

HTOX - TISSUE SAMPLES FROM LUNG CANCER PATIENTS WHO HAD WORKED AT A COPPER SMELTER ... SHOWED 38% OF CANCERS WERE FOUND TO BE ADENOCARCINOMAS COMPARED TO 12% ADENOCARCINOMAS AMONG CONTROL CANCER PATIENTS.

PREDOMINANCE OF ADENOCARCINOMAS WAS ASSOCIATED WITH ARSENIC EXPOSURE. [WICKS MJ ET AL; AM J IND MED 2 (1): 25-31 (1981)]

HTOX - The low toxicity of elemental arsenic is attributed to its virtual insolubility in water or in body fluids. [USEPA; Ambient Water Quality Criteria Doc: Arsenic p.A-1 (1980) EPA 440/5-80-021]

HTOX - Arsenic is capable of producing keratosis, especially on the palms and soles. [Sittig M; Handbook of Toxic and Hazardous Chemicals p.58 (1981)]

HTOX - Children /appear to be/ more sensitive to arsenic toxicity /than are adults/: a dose of 0.08 g/kg body weight resulted in arsenic intoxication in an adult, whereas a dose 80 times less produced similar toxicity in a child. [Nat'l Research Council Canada; Effects of Arsenic in the Canadian Environment p.259 (1978) NRCC No.15391]

HTOX - It was suggested that the excess cancer mortality in Butte, /Montana/ might be attributable to community air pollution arising from the sanding material, /containing arsenic,/ used on city streets during the winter. [NIOSH; Criteria Document: Inorganic Arsenic p.50 (1975) DHEW Pub. NIOSH 75-149]

HTOX - Large amounts of dolomite and bonemeal are being consumed, especially by nutrition conscious persons. The mineral content of commercial samples has been analyzed by different laboratories, and significant amounts of ... arsenic ... were detected. Physicians must consider the possibility of unrecognized self-poisoning from the consumption of such a substance, especially in the context of unexplained neurologic, gastrointestinal, cutaneous, and hematologic disorders. The use of dolomite and bonemeal by pregnant women, children with suspected milk allergy, and elderly persons requires careful evaluation. [Roberts HJ; South Med J 76 (5): 556-9 (1983)]

HTOX - MOST SERIOUS EXPOSURES TO FUMES AND DUSTS OCCUR IN CONNECTION WITH SMELTING OF COPPER, LEAD, ZINC, AND OTHER ORES, AND IN MFR AND USE OF INSECTICIDES. NON-INDUSTRIAL ABSORPTION OF ARSENIC FROM EATING SEA FOOD. SPRAYED FRUITS AND VEGETABLES, AND ... USE OF MEDICINAL ARSENICALS. ... [PATTY. INDUS HYG & TOX 3RD ED VOL2A,2B,2C 1981-82 , p. 1518]

HTOX - TOXICOLOGY RISK DURING MANUFACTURE AND PROCESSING OF AIIIBV-TYPE SEMICONDUCTORS WAS STUDIED. ARSENIC CPMD WAS ONE OF THE TOXIC PRODUCTS. [KNIZEK M; ELEKTROTECH CAS 29 (2): 152-7 (1978)]

HTOX - The burden of evidence shows arsenic to be a respiratory and dermal carcinogen. Arsenic induced cancer usually occurs at more than one site and has a long latent period, ie from 13 to 50 yr after the initial exposure. Studies indicate that a respiratory exposure of 3 ug/cu m for less than 1 yr can result in respiratory cancer. [Nat'l Research Council Canada; Effects of Arsenic in the Canadian Environment p.280 (1978) NRCC No.15391]

HTOX - Increased risk of cancer to humans 1 g/yr; chronic poisoning to humans 3.0-4.0 mg/day. [Nat'l Research Council Canada; Effects of Arsenic in the Canadian Environment p.247 (1978) NRCC No.15391]

HTOX - A patient with arsenic intoxication is reported, who presented with a variety of gastrointestinal and neurologic disturbances including unilateral facial nerve palsy and acute symptomatic pancreatitis, neither of which have been previously described as sequelae of arsenic poisoning. The patient also suffered hematologic, dermatologic, and cardiopulmonary complications. [Zaloga GP et al; Am J Med Sci 289 (5): 210-4 (1985)]

HTOX - A review of the results of chromosome studies on peripheral lymphocytes of individuals exposed to heavy metals since the 1960's. Sister chromatid exchanges (SCE) do not seem to be a substantial endpoint for assessing the action of metals on human genetic material, although significantly increased sister chromatid exchange rates have been reported in persons exposed to arsenic compounds. [Gebhart E; Carcinogen Mut Metal Cmpd p.213-25 (1985)]

HTOX - Three men with severe arsenic poisoning were hospitalized. ... Two patients had coarsely stippled red blood cells with markedly abnormal nuclei in their peripheral blood smears. Karyorrhexis or marked dyserythropoiesis in the peripheral smear in arsenic or lead poisoning, may be a unique hematologic clue. Scrutiny of the peripheral blood and/or buffy coat smear in patients with perplexing gastrointestinal or neurologic symptoms may enable earlier diagnosis and better therapy of arsenic poisoning. [Eichner ER; Am J Clin Pathol 81 (4): 533-7 (1984)]

HTOX - The utility of biochemical and immunological markers as early indicators of renal dysfunction in workers occupationally exposed to inorganic arsenic was evaluated. Urine samples were collected from 17 glassware factory workers, with a mean duration of occupational exposure to arsenic trioxide of 14.5 + 8.0 years, and 22 comparisons without known exposure to inorganic arsenic.

Urine samples were analyzed for inorganic arsenic concentrations, and for biochemical and immunological markers of renal dysfunction, urinary albumin, retinol binding protein beta2-microglobulin, and brush border antigen. The glass workers had significantly higher urinary inorganic arsenic levels than comparisons. Urinary retinol binding protein levels were significantly higher in the arsenic exposed subjects than comparisons, but there were no other significant differences between the two groups. Urinary retinol binding protein was the only indicator of renal dysfunction which was significantly elevated in the workers occupationally exposed to arsenic. [Foa V et al; Occup Environ Chem Haz 362-7 (1987)]

HTOX - Data on the respiratory cancer mortality experience of copper smelter workers occupationally exposed to arsenic were reanalyzed. Previous analysis of data on 2,802 males who worked at least one year between 1940 to 1964 at a copper smelter in Tacoma, Washington showed a two fold excess in respiratory cancer deaths. ... In the present study, the relation between air arsenic levels and respiratory cancer was reanalyzed using data on departmental air arsenic measurements from company records of the Tacoma smelter starting in 1938, and urinary arsenic measurements identified by department and worker, starting in 1948. Analysis using these new exposure estimates revealed a much stronger relation between standardized mortality ratio for respiratory cancer and time weighted air arsenic exposure. When the dose response relation was based on airborn concentration of arsenic, it was concave downward, but when it was based on urinary concentration of arsenic, the dose response relation appeared linear. Arsenic is probably a more potent carcinogen than indicated in previous reports, and these results emphasize the importance of biologic markers as opposed to external measures of exposure in risk assessments. [Enterline PE et al; Am J Epidemiology 125 (6): 929-38 (1987)]

HTOX - A large case control study was undertaken in order to examine occupational etiological factors and the possible significance of tobacco smoking in lung cancer. The final study population consisted of 589 cases, 582 dead comparisons, and 453 living comparisons. The results indicated that individuals who had been occupationally exposed to carcinogens such as arsenic, asbestos, and randon daughters (miners, copper smelters, plumbers, and electricians) for more than 15 years had a considerably increased risk of lung cancer. Workers who had been exposed to suspected carcinogens for more than 25 years (mechanics and professional drivers) were at slightly increased risk, but this disappeared after adjustment for smoking. Overall, an estimated 9 percent of lung cancers were attributable to occupation. [Damber LA, Larsson LG; Brit J Indust Med 44 (7): 446-53 (1987)]

HTOX - The proliferative response of peripheral blood lymphocytes was examined in ten workers exposed to arsenic (As) during their employment at a semiconductor manufacturing facility for at least 3 years. The level in workers' hair was 0.68 mg/kg compared to 0.05 mg/kg for the comparison group. The proliferative response peripheral blood lymphocytes was significantly higher than normal comparisons, and the average stimulation index of workers was three times the normal level. In-vitro exposure of normal lymphocytes to 10 to 100 nanograms/ml sodium arsenite as As enhanced the proliferative. At concentrations of > or = 500 ng/ml all samples showed suppression of stimulation. [Yoshida T et al; Indus Health 25 (1): 29-31 (1987)]

HTOX - A 50 year old chemical engineer, routinely screened for occupational arsenic exposure, was admitted with delirium for which no known etiology was found. Elevated levels of arsenic were found in the urine and hair. The patient received chelation treatment with British anti-Lewisite; substantial amounts of arsenic were excreted and the toxic encephalopathy improved gradually over the 8 month follow-up period. The patient was tested at 6 weeks, 4 months, and 8 months postdelirium with a battery of neuropsychological tasks. Results showed verbal learning and memory were severely impaired while general intellectual abilities and language remained unaffected. Follow up examinations with no subsequent reexposure revealed improvements in specific cognitive tasks. [Bolla-Wilson K, Bleecker ML; J Occup Med 29 (6): 500-3 (1987)]

HTOX - Arsenic induced polyneuropathy is traditionally classified as an axonal loss type, electrodiagnostically resulting in low amplitude or absent sensory and motor responses, relatively preserved proximal and distal motor conduction rates, and distal denervation. Four patients with a subacute onset of progressive polyradiculoneuropathy following high dose arsenic poisoning were studied. Three patients, showed findings suggestive of an acquired segmental demyelinating polyradiculoneuropathy. Serial testing confirmed evolution into features of a distal dying-back neuropathy. Arsenic toxicity and the resultant biochemical derangement of the peripheral nerve cell leads to subtle changes in axonal function that produce, initially, segmental demyelination and

eventually distal axonal degeneration. Acute arsenic toxicity must be suspected in patients with clinical and electrodiagnostic features supporting Guillain-Barre syndrome. [Donofrio PD et al; Muscle Nerve 10 (2): 114-20 (1987)]

HTOX - Case histories involving arsenic keratosis of the skin and arsenic associated cancer are reviewed. ... In all cases, a local keratosis condition preceded the cancer. ... [Hutchinson J; Trans Pathol Soc of London 39: 352-63 (1988)]

HTOX - The histological distribution of lung cancer was investigated in 93 men who worked at a Swedish smelter having high levels of arsenic. A comparison was made with a group of 136 lung cancer patients from the county where the smelter was located. No pronounced differences in the types of lung carcinomas between smelter workers and the reference group was found for smokers. Analyses indicated an increased proportion of adenocarcinomas among the smelter workers, confirming earlier data, but these findings were difficult to interpret. Cases among nonsmoking smelter workers showed a histological distribution similar to smokers, indicating that the work environment at the smelter and smoking had a similar influence on the lung cancer risk. [Pershagen G et al; Br J Ind Med: 44 (7): 454-8 (1987)]

NTOX - INJECTION OF SUBLETHAL CONCN OF ARSENIC INTO EGGS /CHICKEN/ PRODUCED ECTOPIC CONDITIONS, BUT NO /TERATOGENIC EFFECT/ ... [NRC. DRINKING WATER & HEALTH 1977 , p. 339]

NTOX - Mitochondria are particularly vulnerable to inorganic arsenic, swelling observed, interference with heme biosynthesis. [Fowler BA; Environ Health Perspect 19: 239 (1977) as cited in USEPA; Ambient Water Quality Criteria Doc: Arsenic p.C-41 (1980) EPA 440/5-80-021]

NTOX - Metallic arsenic in lanolin was injected into the femur marrow of 25 male rats and 6 rabbits. The doses were 0.43 mg and 0.65 mg, respectively. Only 4 rats survived 18 mo and one of these developed a spindle cell sarcoma at the site of injection. None of the rabbits showed any metaplastic reactions. No tumors were produced at the site of injection in 25 rats injected intrapleurally once a mo for 6 mo resulting in a total dose of 0.65 mg of arsenic. Similar results were obtained after nasal sinus injection of 0.65 mg of arsenic in 20 rats. [Hueper WC; JNCI 15: 113-24 (1954) as cited in WHO; Environ Health Criteria: Arsenic p.126 (1981)]

NTOX - Arsenic toxicity to microorganisms produces a decline in growth and metabolic rates. The more tolerant species can withstand arsenic levels up to 1000 ppm whereas the most sensitive organisms succumb to levels less than 375 ppm. [Nat'l Research Council Canada; Effects of Arsenic in the Canadian Environment p.153 (1978) NRCC No.15391]

NTOX - The mineralization of glucose in sediments was less at pH 4 and 5 than at pH 7, and was less at 0 deg than at 20 deg C. Most of the bacteria isolated from the sediments incubated at 0 and 20 deg C were psychrophilic and most of them were resistant to lead (Pb) and selenium (Se); a smaller proportion was resistant to mercury (Hg) and arsenic (As). Many of the bacteria were resistant to > 1 of the elements. Pb and Hg were more toxic to bacterial growth at pH 4.5 than at 7.5, but Se and As were slightly more toxic at the higher pH. [Baker MD et al; Water Res 17 (8): 925-30 (1983)]

NTOX - Autoradiographic studies on the distribution of ... arsenic (As) in the testes and epididymis of rodents. ... Arsenic accumulated in the lumen of the duct of epididymis causing long-term exposure of the semen. [Danielson BR et al; Arch Toxicol 7: 177-80 (1984)]

NTOX - The median lethal concn of arsenic for Black Sea mussel was 10 mg/l. Although, the Black Sea mussels were quite resistant to /arsenic/ considering mortality as the indicator, they were quite sensitive to sublethal concn of the toxicant as reflected by ... physiological changes (oxygen consumption respiration, trophic activity of yearlings). [Pereladov MV, Erofeeva MP; Ekol Aspekty Khim Radioakt Zagryaz Vodn Sredy p.61-4 (1983)]

NTOX - ... Oral intake of arsenic has been implicated as goiterogenic, and this possible effect has been confirmed in animals. ... [CASARETT & DOULL'S TOXICOLOGY. 2ND ED 1980 , p. 437]

NTOX - ... Inorganic arsenic poisoning in cattle is characterized by reddened abomasal or duodenal mucosa and submucosal edema and hemorrhages in the abomasum and duodenum, with resulting sloughing of the duodenal mucosa or perforation of the gut wall. The intestinal contents are fluid, foul smelling, bloody, and may contain shreds of intestinal mucosa. The liver may be soft and yellow. The lung may be edematous and congested. There may be hemorrhages on the heart, peritoneum, kidneys, and liver. In swine, there may be inflamed and edematous fauces. The edema may extend to the larynx and trachea, causing asphyxial signs and lesions. ... [BOOTH. VET PHARM THERAP 5TH ED 1982 , p. 1024]

NTOX - The genotoxic potential of 48 inorganic derivatives was studied using the bacterial colorimetric assay: the SOS Chromotest. Some of these compounds are known carcinogens (arsenic, chromium(VI), cadmium, nickel) or suspected carcinogens for human beings (mercury, lead), others are non-carcinogens. Among these 48 derivatives, only the two chromium(VI) and the tin(II) compounds gave positive results. [Olivier P, Marzin D; Mutat Res 189 (3): 263-70 (1987)]

NTOX - Two chem carcinogens (diethylstilbestrol and arsenic) induce early changes in the Syrian hamster embryo cells leading to neoplastic transformation. Both of the chem induce cell transformation in the absence of detectable gene mutations. Studies on their mechanisms of action suggest a role for gene duplication and gene amplification in carcinogenesis. [Barrett JC et al; Accomp Oncol 2 (1): 56-68 (1987)]

NTOX - ... In fowl, there may be an intense inflammation of the proventriculus and gizzard, and the horny lining of the gizzard may be sloughing away because of an underlying gelatinous exudate. The duodenal mucosa may be slightly reddened, and the liver may manifest degenerative changes. [BOOTH. VET PHARM THERAP 5TH ED 1982 , p. 1024]

FATE - Aquatic Fate: Arsenic as a free element (0-oxidation state) is rarely encountered in natural waters. Soluble inorganic arsenate (+5-oxidation state) predominates under normal conditions since it is thermodynamically more stable in water than arsenite (+3 oxidation state). [USEPA; Ambient Water Quality Criteria Doc: Arsenic p.A-1 (1980) EPA 440/5-80-021]

BIOC - Hair samples collected from common hare (Lepus europaeus), common vole (Microtus arvallis), and wood mouse (Apodemus sylvaticus) were subjected to instrumental neutron activation analysis (INAA). Up to 18 elements (arsenic, gold, bromine, cesium, cobalt, chromiuum, copper, iron, mercury, potassium, lanthanium, sodium, antimony, scandium, selenium, samarium, thorium and zinc) were detected in each hair sample. Animal hair samples from areas polluted by thermal power plants burning coal were taken and compared with hair samples from the animals living in relatively nonpolluted control areas. Animal hair samples from areas with higher levels of pollution contain usually higher concn of toxic and essential elements as As, Co, Cr, Fe, and Se. Muride rodents can be used for more detailed monitoring of environmental exposure than the hare. Moreover, the hair of the common vole shows usually highest levels of contamination as compared with the wood mouse, which could be explained by different components of feed. Animal hair was a rather sensitive indicator of environmental exposure and INAA proved to be a suitable analytical tool for this purpose. [Obrusnik I, Paukert J; J Radioanal Nucl Chem 83 (2): 397-406 (1984)]

RTEX - Arsenic may be absorbed by ingestion, inhalation, and permeation of skin or mucous membranes. [Nat'l Research Council Canada; Effects of Arsenic in the Canadian Environment p.21 (1978) NRCC No.15391]

RTEX - Occupations with potential exposure: acetylene workers, acid dippers, alloy makers, aniline color makers, aniline workers, arsine workers, Babbit metal workers, bleaching powder markers, boiler operators, brass makers, bronze makers, bronzers, cadmium workers, cattle dip workers, ceramic enamel makers, ceramic makers, copper smelters, defoliant applicators and makers, dimethyl sulfate makers, drug makers, dye makers, electrolytic copper makers, electroplaters, enamelers, etchers, farmers, ferrosilicon workers, fertilizer makers, fireworks makers, galvanizers, glass makers, gold extractors, gold refiners, hair remover makers, herbicide makers, hide preservers, hydrochloric acid workers, illuminating gas workers, insecticide makers, jewelers, lead burners, lead shot makers, lead smelters, leather workers, lime burners, metal cleaners, metal refiners, nitrocellulose makers, ore smelter workers, organic chemical synthesizers, paint makers, painters, paper makers, petroleum refinery workers, pigment makers, plastic workers, plumbers, printing ink workers, rayon makers, rodenticide makers, semiconductor compound makers, sheep dip workers, silver refiners, soda makers, solderers, submarine workers, sulfuric acid workers, taxidermists, textile printers, tinners, tree sprayers, type metal workers, water weed controllers, weed sprayers, wood preservative makers, wood preservers, and zinc chloride makers, etc. /Inorganic arsenic cmpd/ [NIOSH; Criteria Document: Inorganic Arsenic p.123 (1975) DHEW Pub. NIOSH 75-149]

RTEX - ... INDUSTRIAL AND AGRICULTURAL EXPOSURE ... THE INDIVIDUALS AT GREATEST RISK ARE SMELTER WORKERS, ALTHOUGH THERE IS SOME SUGGESTION THAT WOMEN RESIDING NEAR SUCH OPERATIONS INCUR A GREATER INCIDENCE OF RESPIRATORY CANCER. [CASARETT & DOULL'S TOXICOLOGY. 2ND ED 1980 , p. 437]

NAME - BENZENE
SY - BICARBURET OF HYDROGEN
SY - CYCLOHEXATRIENE
SY - FENZEN (CZECH)
SY - NCI-C55276
SY - Polystream
SY - (6)ANNULENE
SY - COAL NAPHTHA
SY - PHENE
SY - PHENYL HYDRIDE
SY - PYROBENZOL
SY - PYROBENZOLE
SY - AI3-00808
SY - Caswell no 077
SY - EPA pesticide chemical code 008801
SY - Benzol 90
USE - MFR MEDICINAL CHEM, DYES, ORG CMPD, ARTIFICIAL LEATHER, LINOLEUM, OIL CLOTH, VARNISHES, LACQUERS; SOLVENT FOR WAXES, RESINS, OILS /USE AS SOLVENT IS NOW DISCOURAGED/ [MERCK INDEX. 10TH ED 1983 , p. 151]
USE - Used for printing & lithography, paint, rubber, dry cleaning, adhesives & coatings, detergents [NIOSH; Criteria Document: Benzene p.20 (1974) DHEW Pub No 74-137]
USE - Extraction and rectification; preparation and use of inks in the graphic arts industries; as a thinner for paints; as a degreasing agent [Fishbein L; Potential Indust Carcins & Mutagens p.96 (1977) USEPA 560/ 5-77-005]
USE - CHEM INT FOR ETHYLBENZENE, CUMENE, CYCLOHEXANE, NITROBENZENE, MALEIC ANHYDRIDE, CHLOROBENZENES, DETERGENT ALKYLATE, ANTHRAQUINONE, BENZENE HEXACHLORIDE, BENZENE SULFONIC ACID, BIPHENYL, HYDROQUINONE, & RESORCINOL [SRI]
USE - /Benzol for/ pesticidal uses /has been/ cancelled. /It/ was in use alone or in formulations for screwworm control on animals. /It was/ an ingredient of some early grain fumigants [FARM CHEMICALS HANDBK 1987 C-35]
USE - In the tire industry (McMichael et al, 1975), & in shoe factories (Aksoy et al, 1974), benzene is used extensively. [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1638]
USE - Used primarily as a raw material in the synthesis of styrene (polystyrene plastics and synthetic rubber), phenol (phenolic resins), cyclohexane (nylon), aniline, maleic anhydride (polyester resins), alkylbenzenes (detergents), chlorobenzenes, and other products used in the production of drugs, dyes, insecticides, and plastics. [NTP; Toxicology and Carcinogenesis Studies of Benzene p.24 Report# 289 (1986) NIH Pub# 86-2545]
BP - 80.1 DEG C [MERCK INDEX. 10TH ED 1983 , p. 151]
MP - 5.5 DEG C [IARC MONOGRAPHS. 1972-PRESENT V7 203 (1974)]
SOL - 0.180 g/100 g of water at 25 deg C [KIRK-OTHMER. ENCYC CHEM TECH 3RD ED 1978-PRESENT V3(78) 746]
SOL - MISCIBLE WITH ALCOHOL, CHLOROFORM, ETHER, CARBON DISULFIDE, ACETONE, OILS, CARBON TETRACHLORIDE, & GLACIAL ACETIC ACID [MERCK INDEX. 10TH ED 1983 , p. 151]

o EMCE - CLINICAL EFFECTS :

ACUTE EXPOSURE

- o Acute benzene toxicity from inhalation or dermal exposure to the fumes results in CNS effects (prominent vascular congestion), cutaneous burns, and respiratory effects (hemorrhagic airless lungs with confluent alveolar hemorrhage and pulmonary edema). Ingestion has been associated with toxicity.
- o INHALATION: Exposure to high concentrations of the vapor (3000 ppm or higher) may cause acute poisoning, characterized by the narcotic action of benzene on the central nervous system. The anesthetic action of benzene is similar to that of other anesthetic gases, consisting of a preliminary stage of excitation followed by depression and, if exposure is continued, death through respiratory failure.
- o DERMAL: Locally, benzene has a comparatively strong irritating effect, producing erythema and burning, and, in more severe cases, edema and even blistering.

CHRONIC EXPOSURE

- o CHRONIC TOXICITY: Usually due to occupational exposure from chronic inhalation.

1. Chronic exposure has resulted in aplastic anemia, acute myeloblastic leukemia, erythroleukemia, and death. The possibility of chronic poisoning in employees of gasoline filling stations and bulk gasoline loading facilities exists due to the high concentration of benzene in gasoline.
2. In chronic poisoning the onset is slow, with the symptoms vague: fatigue, headache, dizziness, nausea and loss of appetite, loss of weight, and weakness are common complaints in early cases. Pallor, nosebleeds, bleeding gums, menorrhagia, petechiae and purpura may develop later. There is great individual variation in the signs and symptoms of chronic benzene poisoning.

CARDIOVASCULAR ACUTE EXPOSURE

- o Cardiac arrhythmias possible.

RESPIRATORY ACUTE EXPOSURE

- o Inhalation may result in bronchial irritation, cough, hoarseness, and pulmonary edema.

NEUROLOGIC ACUTE EXPOSURE

- o Euphoria, headache, giddiness, vertigo, and ataxia possible. Confusion, seizures and coma may occur following exposure to high concentrations. Fatigue, headache, anorexia and dizziness may be noted following chronic exposure to benzene. Transverse myelitis may occur.

GASTROINTESTINAL ACUTE EXPOSURE

- o A burning sensation of the oral mucous membranes, esophagus, and stomach may occur after ingestion as well as nausea, vomiting, and abdominal pain.

HEPATIC ACUTE EXPOSURE

- o Hepatic toxicity may occur.

GENITOURINARY ACUTE EXPOSURE

- o Paroxysmal nocturnal hemoglobinuria has been reported.

HEMATOLOGIC ACUTE EXPOSURE

- o Chronic exposure can result in delayed hematopoietic changes, including acute myeloblastic leukemia and aplastic anemia.

DERMATOLOGIC ACUTE EXPOSURE

- o Erythema, blistering, and dermatitis may occur.

CARCINOGENICITY

HUMAN OVERVIEW

- o Leukemia may be associated with benzene exposure.

GENOTOXICITY

- o Chromosome aberration and sister chromatid exchange frequencies have been elevated in peripheral lymphocytes of persons exposed to benzene (Tompa et al, 1994; Major et al, 1994; Turkel & Egeli, 1994).

HTOX - Benzene is irritant to skin, & by defatting the keratin layer may cause erythema, vesiculation, & dry & scaly dermatitis. [PATTY. INDUS HYG & TOX 3RD ED VOL2A,2B,2C 1981-82 , p. 3262]

HTOX - AFTER ACUTE EXPOSURE TO A LARGE AMT OF BENZENE, BY INGESTION OR BY BREATHING CONCENTRATED VAPORS, THE MAJOR TOXIC EFFECT IS ON THE CNS. SYMPTOMS FROM MILD EXPOSURE INCL DIZZINESS, WEAKNESS, EUPHORIA, HEADACHE, NAUSEA, VOMITING, TIGHTNESS IN CHEST, & STAGGERING. IF

EXPOSURE IS MORE SEVERE, SYMPTOMS PROGRESS TO BLURRED VISION, TREMORS, SHALLOW & RAPID RESP, VENTRICULAR IRREGULARITIES, PARALYSIS, & UNCONSCIOUSNESS. [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1638]

HTOX - Chronic exposure to benzene usually involves the inhalation of vapor. Signs and symptoms incl effects on CNS & the GI tract (headache, loss of appetite, drowsiness, nervousness, & pallor), but the major manifestation of toxicity is aplastic anemia. Bone marrow cells in early stages of development are most sensitive ... & arrest of maturation leads to gradual depletion of circulating cells. [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1638]

HTOX - BENZENE (BENZOL) ... HAS SPECIFIC TOXIC EFFECT ON BLOOD FORMATION, CAUSING APLASTIC ANEMIA & TENDENCY TO HEMORRHAGE. OCCASIONALLY HEMORRHAGES IN RETINA & IN CONJUNCTIVA ARE FOUND IN SYSTEMIC POISONING BY BENZENE. IN RARE INSTANCES NEURORETINAL EDEMA & PAPILLEDEMA HAVE BEEN DESCRIBED ACCOMPANYING RETINAL HEMORRHAGES. IT HAS NOT BEEN ESTABLISHED THAT BENZENE CAN INDUCE RETROBULBAR NEURITIS OR OPTIC NEURITIS ... [GRANT. TOX OF THE EYE 1986 , p. 140]

HTOX - PATHOLOGICAL FINDINGS FROM ... INHALATION INCL ACUTE GRANULAR TRACHEITIS, LARYNGITIS & BRONCHITIS, MASSIVE HEMORRHAGE OF LUNG, CONGESTIVE GASTRITIS, INFARCT OF SPLEEN, ACUTE CONGESTION OF KIDNEYS, & MARKED CEREBRAL EDEMA. [GOODMAN. PHARM BASIS THERAP 5TH ED 1975 , p. 906]

HTOX - Many acute deaths /from benzene exposure at high concn have been/ ... due to ventricular fibrillation ... /caused by exertion/ & release of epinephrine. This was probably the mechanism involved in the death of workers in tank cars which had contained benzene. Frequently, the man who went into the tank car to carry out an unconscious worker died during the effort of lifting the unconscious man up the ladder. [THIENES. CLIN TOX 5TH ED 1972 , p. 124]

HTOX - ... A large number of workers exposed to but not seriously intoxicated by benzene /were studied & results showed/ that serum complement levels, IgG, & IgA, were depressed but that IgM levels did not drop & were in fact slightly higher (Lange et al 1973; Smolik et al 1973). ... These /& other/ observations, taken together with well-known ability of benzene to depress leukocytes ... may explain why benzene-intoxicated individuals readily succumb to infection & why terminal event in severe ... toxicity is often an acute, overwhelming infection. [IARC MONOGRAPHS. 1972-PRESENT V29 116 (1982)]

HTOX - IN EXPT IN VITRO, BENZENE DID NOT CHANGE THE NUMBER OF SISTER-CHROMATID EXCHANGES OR THE NUMBER OF CHROMOSOMAL ABERRATIONS IN HUMAN LYMPHOCYTES. [GERNER-SMIDT P, FRIEDRICH U; MUTAT RES 58 (2-3): 313-6 (1978)]

HTOX - THE MUTAGENIC ACTIVITY UPON HUMAN LYMPHOCYTES WAS STUDIED AFTER ITS ADDN TO CULTURE ON THE 28TH HR OF CULTIVATION (G1-S PERIODS). CONCN OF 1, 10, 25, 50, 100, & 250 UG/ML WERE STUDIED. BENZENE IS A WEAK MUTAGEN. IT CAUSED ELONGATION OF CENTROMERE PORTIONS OF CHROMOSOMES & CHROMOSOMAL ABERRATIONS WERE MAINLY OF SINGLE & PAIRED FRAGMENT TYPE. MUTAGENIC ACTIVITY WAS ABOUT THE SAME IN THE G0 & G1-S PERIODS. [MNATSAKANOV ST, POGOSYAN AS; BIOL ZH ARM 26 (12): 38-43 (1973)] **PEER REVIEWED**

HTOX - A major concern is the relationship between chronic exposure to benzene & leukemia. Epidemiological studies have been conducted on workers in the tire industry (McMichael et al, 1975), & in the shoe factories (Aksoy et al, 1974), where benzene is used extensively. Among workers who died from exposure to benzene, death was caused by either leukemia or aplastic anemia, in approx equal proportions. [GOODMAN. PHARM BASIS THERAP 7TH ED 1985 , p. 1638]

HTOX - CHRONIC BENZENE TOXICITY IS EXPRESSED AS BONE MARROW DEPRESSION

RESULTING IN LEUCOPENIA, ANEMIA, OR THROMBOCYTOPENIA (LEUKEMOGENIC ACTION). WITH CONTINUED EXPOSURE THE DISEASE PROGRESSES TO PANCYTOPENIA RESULTING FROM BONE MARROW APLASIA. EVIDENCE HAS ACCUM IMPLICATING BENZENE IN THE ETIOLOGY OF LEUKEMIAS IN WORKERS IN INDUSTRIES WHERE BENZENE WAS HEAVILY USED. IT HAS BEEN SUGGESTED THAT LEUKEMIA IS AS FREQUENT A CAUSE OF DEATH FROM CHRONIC BENZENE EXPOSURE AS IS APLASTIC ANEMIA. [SNYDER R ET AL; LIFE SCIENCES 21 (12): 1709-22 (1977)] **PEER REVIEWED**

HTOX - MANY CASES OF ACUTE LEUKEMIA DEVELOPING AS TERMINAL STAGE OF APLASTIC ANEMIA RESULTING FROM EXPOSURE TO BENZENE MAY HAVE BEEN MISSED BECAUSE BONE MARROW PUNCTURE WAS NOT PERFORMED. BENZENE LEUKEMIA IS ACUTE STEM CELL OR MYELOBLASTIC LEUKEMIA, SOMETIMES ALEUKEMIA. THERE MAY BE A LATENT PERIOD EXTENDING OVER SEVERAL YEARS BETWEEN CESSATION OF EXPOSURE WITH MORE OR LESS PRONOUNCED ANEMIA, & THE ONSET OF LEUKEMIA. [VIGLIANI EC, FORNI A; ENVIRON RES 11 (1): 122-7 (1976)] **PEER REVIEWED**

HTOX - A dose-related increase in the number of cells with chromosomal aberrations occurred in human lymphocyte cultures treated with 4X10-5 M and 3.0X10-3 M benzene for 53 hr prior to metaphase analysis. Cells in late G2 stage were the most susceptible to the effect of benzene. [Morimoto K; Japan J Ind Health 8: 23-5 (1976)]

HTOX - Epidemiological studies (exposure to high concn is associated with hematotoxicity and acute myelocytic leukemia in humans ...) [European Chemical Industry, Ecology and Toxicology Center p.44 (1984)] **PEER REVIEWED**

HTOX - Italian shoemakers exposed to 200-500 ppm benzene in inks and glues showed an incidence of leukemia of 1 per 1,000. [Vigliani EC; Ann NY Acad Sci 271: 143 (1976)]

HTOX - Follow up study at Massachusetts rubber coating plants of 38 workers exposed over 1-24 yr at 5-50 ppm (140 ppm peak) showed no evidence of blood dyscrasias or leukemia. [Pagnotto LD et al; Am Ind Hyg Assoc J 40: 137 (1979)]

HTOX - A significantly incr frequency of chromatid and isochromatid breaks in the cultured lymphocytes of workers in chemical laboratories and in the printing industry has been reported. [Funes-Cravioto F et al; Lancet p. 322 (1977) as cited in USEPA; Ambient Water Quality Criteria for Benzene p.C-46 (1980) EPA 440/5-80-018]

HTOX - A significant incr of peripheral blood lymphocyte chromosomal aberrations in workers exposed to benzene was reported, but not in those exposed to toluene and xylene. [Vigliani EC, Forni A; J Occup Med 11 p.148 (1969) as cited in USEPA; Ambient Water Quality Criteria for Benzene p.C-46 (1980) EPA 440/5-80-018]

HTOX - A report on 52 workers exposed to benzene found chromosomal aberrations (chromosome breaks, dicentric chromosomes, translocations, and exchange figures) in peripheral lymphocytes at 2-3 times the rates found in controls. The 8 hr TWA exposure was 2-3 ppm, the average concn determined by 15 min sampling was 25 ppm, and the peak concn was 50 ppm. [USEPA; Ambient Water Quality Criteria for Benzene p.C-47 (1980) EPA 440/5-80-018]

HTOX - An epidemiological study implicating benzene as a leukemogen (acute myelocytic leukemia) followed 748 white males exposed to benzene in the manufacture of a rubber product from 1940-1949. A statistically significant ($p < \text{or} = 0.002$) excess of leukemia was found when compared against two control populations. There was a 5 fold excessive risk of all leukemias and a 10 fold excessive risk of myelocytic and monocytic leukemias combined. [Infante PF et al; Lancet p. 322 (1977) as cited in USEPA; Ambient Water Quality Criteria: Benzene p.C-58 (1980) EPA

440/5-80-018]

HTOX - A hematological investigation was carried out on 147 workers (employed for +10 years) exposed to high benzene levels (320-470 ppm). Abnormalities were noted in at least one parameter in 73%, the most common one being thrombocytopenia, which occurred in 62% followed by anemia (35%) and leucopenia (32%). Pancytopenia occurred in 21% of the workers. During the 3 months following removal from exposure, hematological parameters returned to normal in 120 workers, and one subject died. After one year, 20 of the remaining workers had only minor abnormalities, six were still off work, and one was still hospitalized. [Savilahti M; Arch Gewerbpathol Gewerbhyg 15: 147-57 (1956)]

HTOX - A retrospective mortality study of a cohort of 594 men exposed to benzene at levels ranging between 2 and 25 ppm (TWA) was carried out at the Dow Chemical Co between 1940-1973. No incr in total mortality was noted with 102 observed/128 expected (Standard Mortality Ratio (SMR) 80). A slight increase was noted in total deaths due to malignancies (30 observed/22.8 expected, SMR 132) and suicide (5 observed/3.2 expected, SMR 147) as well as deaths from leukemia (3 observed/0.8 expected) and cancers of the digestive organs and peritoneum (9 observed/6.9 expected, SMR 125). If 53 workers exposed to other chemicals are excluded from malignancies, the results would then be 24 observed/20.3 expected, SMR 108. [Ott MG et al; Arch Environ Health 33: 3-10 (1978)]

HTOX - /A subset of 292 men of the 594 in the benzene exposure of Dow cohort who were still employed in 1967/ had an examination of the health status /evaluation/ carried out between 1967-1974 and compared to a control population selected from employees not exposed to benzene, using a matched pair design (matched for age, cigarette smoking habits and length of employment). No clinically significant differences were reported although slight decr in total bilirubin levels and red blood cell counts were noted. [Towsent et al; J Occup Med 20: 543-8 (1978)]

HTOX - Thirty two patients who had recovered from a blood disease (bone marrow impairment) caused by benzene poisoning had significantly increased rates of "unstable" and "stable" chromosomes. Aberrations of chromosomes were present for several years after cessation of the exposure and after recovery from poisoning. Persistence of an increase of the "stable" changes was particularly remarkable. [Waldbott GL; Health Eff of Envir Poll p.214 (1973)]

HTOX - NUMEROUS STUDIES HAVE BEEN CARRIED OUT ON THE CHROMOSOMES OF BONE-MARROW CELLS & PERIPHERAL LYMPHOCYTES FROM PEOPLE KNOWN TO HAVE BEEN EXPOSED TO BENZENE (DEAN 1978). ... IN MANY OF THESE STUDIES, SIGNIFICANT INCR IN CHROMOSOMAL ABERRATIONS HAVE BEEN SEEN, WHICH IN SOME CASES HAVE PERSISTED FOR YEARS AFTER CESSATION OF EXPOSURE. ... BONE-MARROW CELLS & PERIPHERAL LYMPHOCYTES /HAVE BEEN EXAM/ FROM WORKERS WITH CURRENT SEVERE BLOOD DYSCRASIAS, & ... /FOLLOW-UP STUDIES HAVE BEEN DONE ON/ SEVERAL WORKERS BY REPEATED CYTOGENETIC STUDIES UP TO 12 YR AFTER RECOVERY FROM BENZENE-INDUCED PANCYTOPENIA. GROSS CHROMOSOMAL ABNORMALITIES WERE CHARACTERISTIC OF THESE CELLS; 70% OF THE BONE-MARROW CELLS & LYMPHOCYTES IN PT WITH ACUTE POISONING SHOWED KARYOTYPIC ABNORMALITIES (POLLINI & COLOMBI 1964). THE AUTHORS COULD NOT RELATE THE FREQUENCY OR TYPE OF CHROMOSOMAL ALTERATIONS TO THE SEVERITY OF BLOOD DYSCRASIA (POLLINI ET AL 1964). FIVE YR AFTER POISONING, ALL ... 5 PATIENTS STUDIED STILL SHOWED STABLE (Cs) & UNSTABLE (Cu) CHROMOSOMAL ABERRATIONS IN ... LYMPHOCYTES, ALTHOUGH ONLY 40% OF CELLS WERE NOW ABNORMAL (POLLINI ET AL 1969). BY 12 YR ... NO

CYTOGENETIC ABNORMALITIES REMAINED IN THE 4 PT STUDIED (POLLINI & BISCALDI 1977). [IARC MONOGRAPHS. 1972-PRESENT V29 118 (1982)] **PEER REVIEWED**

HTOX - METABOLIC ACTIVATION OF BENZENE BY RAT LIVER MICROSOMES & A REDUCED NADP-GENERATING SYSTEM (S-9 MIX) INDUCED SISTER CHROMATID EXCHANGES (SCE) & CELL DIVISION DELAYS IN CULTURED HUMAN LYMPHOCYTES. THERE WERE OPTIMAL CONCN OF S-9 MIX FOR THE CONVERSION OF BENZENE INTO THE ACTIVE METABOLITES THAT EXERTED THESE CYTOTOXIC EFFECTS. [MORIMOTO K; CANCER RES 43 (3): 1330-4 (1983)]

HTOX - ... INCIDENCE OF ACUTE LEUKEMIA OR 'PRELEUKEMIA' AMONG 28,500 SHOE-WORKERS IN TURKEY /WAS ESTIMATED/ ON BASIS OF CASE ASCERTAINMENT BY CONTACT WITH MEDICAL CARE. THIRTY FOUR CASES WERE IDENTIFIED. ... INCIDENCE OF ACUTE LEUKEMIA WAS SIGNIFICANTLY GREATER AMONG WORKERS CHRONICALLY EXPOSED TO BENZENE, WHICH WAS USED AS A SOLVENT BY THESE WORKERS, THAN IN THE GENERAL POPULATION. OCCUPATIONAL EXPOSURES WERE DETERMINED BY WORK HISTORIES & BY ENVIRONMENTAL MEASUREMENTS. THERE WAS SAID TO BE EXPOSURE ONLY TO BENZENE IN SMALL, POORLY VENTILATED WORK AREAS; PEAK EXPOSURES ... WERE REPORTED TO BE 210-650 PPM (670-2075 MG/CU M). DURATION ... WAS EST TO HAVE BEEN 1 TO 15 YR (MEAN 9.7 YR). ANNUAL INCIDENCE WAS EST TO BE 13/100000, GIVING APPROX RELATIVE RISK OF 2 WHEN COMPARED WITH ANNUAL EST FOR GENERAL POPULATION, 6/100000 (AKSOY ET AL 1974; AKSOY 1977). (THESE EST ARE LIMITED BY STUDY DESIGN CHARACTERISTICS & BY UNCERTAINTY ABOUT THE WAY IN WHICH CASES WERE ASCERTAINED, & HOW MANY OF THE STUDY POPULATION WERE EXPOSED & HOW MANY UNEXPOSED). [IARC MONOGRAPHS. 1972-PRESENT V29 121 (1982)] **PEER REVIEWED**

HTOX - OCCUPATIONAL EXPOSURES WERE IDENTIFIED IN ROTOGRAVURE PLANTS & SHOE FACTORIES. BENZENE CONCN NEAR ROTOGRAVURE MACHINES WERE 200-400 PPM (640-1280 MG/CU M), WITH PEAKS UP TO 1500 PPM (4800 MG/CU M); BENZENE CONCN IN AIR NEAR WORKERS HANDLING GLUE IN SHOE FACTORIES WERE 25-600 PPM (80-1920 MG/CU M), BUT WERE MOSTLY AROUND 200-500 PPM (640-1600 MG/CU M). EST LATENCY (YEARS FROM START OF EXPOSURE TO CLINICAL DIAGNOSIS OF LEUKEMIA) RANGED FROM 3-24 YR (MEDIAN, 9 YR). ... THE RELATIVE RISK OF ACUTE LEUKEMIA WAS /EST TO BE/ AT LEAST 20:1 FOR WORKERS HEAVILY EXPOSED TO BENZENE IN ROTOGRAVURE & SHOE INDUSTRIES IN THE PROVINCES STUDIED, WHEN COMPARED WITH GENERAL POPULATION (VIGLIANI 1976). (THE RELATIVE RISK IS BASED ON A NON-VALIDATED ESTIMATE). [IARC MONOGRAPHS. 1972-PRESENT V29 122 (1982)]

HTOX - A HISTORICAL COHORT MORTALITY STUDY WAS CONDUCTED OF 259 MALE EMPLOYEES OF A CHEM PLANT WHERE BENZENE HAS BEEN USED IN LARGE QUANTITIES. THE STUDY GROUP INCL ALL PERSONS WHO WERE EMPLOYED BY THE COMPANY ANY TIME BETWEEN JAN 1, 1947 & DEC 31, 1960. THE COHORT WAS FOLLOWED THROUGH DEC 31, 1977 AT WHICH TIME 58 KNOWN DEATHS WERE IDENTIFIED. THE ONLY UNUSUAL FINDING WAS FOUR DEATHS FROM LYMPHORETICULAR CANCERS WHEN 1.1 WOULD HAVE BEEN EXPECTED ON THE BASIS OF NATIONAL MORTALITY RATES. THREE OF THE DEATHS WERE DUE TO LEUKEMIA & 1 WAS CAUSED BY MULTIPLE MYELOMA. IN ADDN, 1 OF THE LEUKEMIA DEATHS HAD MULTIPLE MYELOMA LISTED ON THE DEATH CERTIFICATE. THE FINDINGS ARE CONSISTENT WITH PREVIOUS REPORTS OF LEUKEMIA FOLLOWING OCCUPATIONAL EXPOSURE TO BENZENE & RAISE THE POSSIBILITY THAT MULTIPLE MYELOMA COULD BE LINKED TO BENZENE, ALSO. [DECOUFLE P ET AL; ENVIRON RES 30 (1): 16-25 (1983)]

HTOX - HEMATOLOGIC & IMMUNOCHEMICAL INVESTIGATIONS CARRIED OUT IN 270 WORKERS WITH CHRONIC EXPOSURE TO BENZENE DEMONSTRATED CHANGES OF THE NUCLEOLOGRAM & OF THE AREA OF LYMPHOCYTE NUCLEOLI & DISORDERS OF THE HUMORAL IMMUNE RESPONSE REVEALED BY RADIAL IMMUNODIFFUSION. THE NUMERICAL RISE OF BI- & POLYNUCLEOLATED CELLS, OF CELLS WITH IRREGULAR MACRONUCLEOLI & AN ENLARGEMENT OF THE NUCLEOLAR AREA REFLECTED INCR

ENDOLYMPHOCYTIC AMT OF RNA. AN INCR CAPACITY OF IG FORMATION, PARTICULARLY OF IGM, WAS ALSO OBSERVED. [CHIRCU V ET AL; REV ROUM MED INTERNE 19 (4): 373-8 (1981)]

HTOX - SOME ASPECTS OF QUANTITATIVE CANCER RISK ESTIMATION: ... RISK IS GREATEST AMONG THOSE WITH LONGEST EXPOSURE, RELATIVE RISKS OF APPROX 2, 14 & 32 BEING OBSERVED FOR EXPOSURES OF LESS THAN 5 YR (2 CASES), 5-9 YR (2 CASES) & 10+ YR (3 CASES), RESPECTIVELY. THE RELATIVE RISK ASSOC WITH AT LEAST 5 YR OF EXPOSURE IS THUS LIKELY TO BE LOWER BOUND FOR RISK ASSOC WITH LIFETIME EXPOSURE AT SIMILAR LEVELS. FOR THOSE WITH AT LEAST 5 YR EXPOSURE, 5 CASES WERE OBSERVED COMPARED WITH AN EXPECTED NUMBER OF 0.237, GIVING A RELATIVE RISK OF 21.1. SINCE THE EXPECTED CUMULATIVE MALE ADULT LIFETIME (FROM 20 YR TO END OF LIFE, TAKEN AS AGE 75) PROBABILITY OF DYING FROM LEUKEMIA IS APPROX 7 PER 1000 IN THE GENERAL POPULATION OF THE USA, AN EXPECTED RELATIVE RISK OF 21.1 WOULD GIVE AN EXTRA (21.1-1.0)X7= 141 CASES OF LEUKEMIA PER 1000 EXPOSED POPULATION. [IARC MONOGRAPHS. 1972-PRESENT V29 395 (1982)] **PEER REVIEWED**

HTOX - The hematotoxicity of benzene is expressed primarily as a bone marrow effect leading eventually to complete destruction of myeloid and erythroid marrow components. This is manifested as a marked decrease in circulating formed elements, ie red blood cells, and platelets. The resultant aplastic anemia is a potentially fatal disorder which in its severe form has better than a fifty percent mortality rate. In both man and laboratory animals the extent of bone marrow damage appears proportional to the dose of benzene. Lesser degrees of bone marrow toxicity than aplastic anemia are more common in occupational exposure situations. Classically, the discovery of one individual with significant bone marrow toxicity has led to evaluation of the exposed work force and the finding of a wide variation in the extent of hematotoxicity. This has ranged from clinically significant pancytopenia, in which are decreases in white blood cells (leukopenia), red blood cells (anemia), and platelets (thrombocytopenia) to a situation in which only one of these is slightly below normal range. In the latter case it is of course difficult to distinguish a benzene effect from that due to the extremes of normal variation or to mild intercurrent disease. [Mehlman MA, ed; Adv Mod Environ Toxicol Vol IV: Carcinogenicity and Toxicity of Benzene p.52 (1983)]

HTOX - The type of leukemia most commonly associated with benzene is acute myelogenous leukemia and its variants, including erythroleukemia and acute myelomonocytic leukemia. Acute myelogenous leukemia is the adult form of acute leukemia and, until recent advances in chemotherapy, it was a rapidly fatal disease. The other major acute form of leukemia, acute lymphocytic leukemia, has been reported to be associated with benzene exposure but evidence of a causal association is weak. There is a somewhat stronger, although still inconclusive, association in the literature between benzene exposure and the two common forms of chronic leukemia: chronic myelogenous leukemia and chronic lymphocytic leukemia. Other hematological disorders possibly associated with benzene exposure include Hodgkin's disease, lymphocytic lymphoma, myelofibrosis and myeloid metaplasia, paroxysmal nocturnal hemoglobinuria, and multiple myeloma. [Mehlman MA, ed; Adv Mod Environ Toxicol Vol IV: Carcinogenicity and Toxicity of Benzene p.52 (1983)]

HTOX - An acute hemorrhagic pneumonitis is highly likely if ... aspirated into lung. [GOSSELIN. CTCP 5TH ED. 1984 III-398]

HTOX - Three cases of chronic leukemia were presented which had a history of chronic benzene exposure. These three patients were part of a larger

group of 58 leukemia patients with benzene exposure histories. Case 1 presented at age 43 due to cardiac complaints. The patient owned a printing shop at which he mixed pigmented dyes with solutions of toluene or methyl alcohol ketone. The individual had a practice of sniffing the solutions as control measure. The toluene solution on analysis was shown to contain 2.8% benzene 95.3% toluene. Blood and bone marrow examination revealed chronic lymphatic leukemia. Case 2 was a 51 year old man with pain in the right quadrant. This individual had owned a small plastics facility between 1955 and 1965 where he was intermittently exposed to thinners containing 27.3% benzene. Subsequent exposure included cleaning solutions without benzene. He was also diagnosed with chronic lymphatic leukemia. The third case was a 50 year old manager of a plastic facility who was diabetic for 15 years and was hospitalized due to recurrent gluteal and inguinal furunculosis during the last 3 years. He had been heavily exposed to benzene between 1957 and 1965. He admitted having removed the dirt from his hands using thinners containing benzene. Hairy cell leukemia was diagnosed. The data suggests that differences in distribution of acute or chronic leukemias in chronic benzene exposure may be related to exposure levels, mode of exposure, or exposure to benzene homologs or other chemicals. [Askoy M; Brit J Haematol 66 (2): 209-11 (1987)] **PEER REVIEWED**

HTOX - A study conducted to measure the concentration of benzene in the air and solvents at 40 small and large workplaces in Turkey where workers had contracted leukemia and lymphoma. In addition, hematological examinations were performed on the 231 workers employed at the facilities. The facilities manufactured and repaired shoes, tires, leather works, automobiles, and farm equipment. The age of the workers ranged from 14 to 57 years and the mean duration of exposure was 8.8 years (range 1 month to 40 years). Case reports were presented for five workers with 2 to 15 years of exposure who had developed acute myeloblastic leukemia, acute lymphoblastic leukemia, acute myelomonocytic leukemia, Hodgkin's disease and poorly differentiated lymphoma. Benzene concentrations in the solutions and thinners used ranged from 3 to 7.5%. The concn of benzene in air samples from the plants ranged from 0 to 110 ppm while 76.4% of solvents contained more than 1% of benzene. Hematological examinations of the workers showed that 32% of them had abnormal values. There has been a decline in the use of benzene in Turkey since an earlier study in 1972, but that the percentages of benzene in most of the materials are still above permissible limits. [Askoy M et al; Brit J Indust Med 44 (11): 785-7 (1987)]

HTOX - Benzene is widely recognized as a leukemogen, and the Occupational Safety and Health Administration is currently attempting to limit exposure to it more strictly. The proposed new regulation is a limit of an eight hr time-weighted average of 1 ppm in place of the current limit of 10 ppm. The fundamental rationale for the change is a perception that the current standard is associated with an inordinate excess of leukemia. The epidemiologic literature on benzene and leukemia supports the inference that benzene causes acute myelocytic leukemia. However, the available data are too sparse, or /have/ other limitations, to substantiate the idea that this causal association applies at low levels (ie, 1-10 ppm) of benzene. Nonetheless, under the assumption that causation does apply at such low levels, a number of researchers have perfomed risk assessments using similar data but different methodologies. The assessments that is considered acceptable suggest that, among 1,000 men exposed to benzene at 10 ppm for a

working lifetime of 30 years, there would occur about 50 excess deaths due to leukemia in addition to the baseline expectation of seven deaths. However, this estimate is speculative and whether or not enough confidence can be placed in it to justify a lower occupational benzene standard remain a decision for policy makers. [Austin H et al; Am J Epidemiol 127 (3): 419-39 (1988)]

HTOX - Results of epidemiologic studies indicating an association between solvent exposure and the development of malignancies affecting hematopoietic and lymphatic tissues are reviewed. Clinical and cytogenetic data supporting this association are discussed. A variety of malignant disorders have been associated with solvent exposure, ie acute leukemia, Hodgkin's disease (odds ratio 2.8-6.6), non-Hodgkin's lymphoma (odds ratio 3.3) and myeloma, and there are some indications that solvent exposure may be a risk factor myelofibrosis. The carcinogenic effect of benzene is epidemiologically and experimentally well documented and there are some indications that other solvents may also be hazardous. Possible mechanisms bringing about malignant transformation are discussed. The need for further epidemiologic, cytogenetic and clinical studies on the association between solvent exposure and malignant diseases is emphasized. [Brandt L; Med Oncol Tumor Pharmacother 4 (3/4): 199-205 (1987)]

HTOX - Currently the most applied technique for monitoring biological effects of exposure to genotoxic chemicals in industrial workers is the measurement of chromosome aberrations in peripheral blood lymphocytes. In the Shell petrochemical complex in the Netherlands cytogenetic monitoring studies have been carried out from 1976 till 1981 inclusive, in workers potentially exposed to a variety of genotoxic chemicals, ie vinyl chloride, ethylene oxide, benzene, epichlorohydrin, epoxy resins. Average exposure levels to these chemicals were well below the occupational exposure limits. Results of thesse studies indicate that no biologically significant increase in the frequencies of chromosome aberrations in the exposed populations occurred compared with control populations. ... Experience with this methodology has shown that the results of chromosome analyses are difficult to interpret, due to the variable and high background levels of chromosome aberrations in control populations and in individuals. It is concluded that the method is not sufficiently sensitive for routine monitoring of cytogenetic effect in workers exposed to the low levels of genotoxic compounds. [deJong G et al; Mutat Res 204 (3): 451-64 (1988)]

HTOX - The possibility of there being a link between the apparent predominance of men with specific on the job exposures to toxic materials among patients with hairy cell leukemia was explored. Of a total of 105 hairy cell leukemia patients, eight were in the medical profession (two X-ray technicians, one radiologist, two pneumologists, two orthopedists, and one internist), 21 were garage mechanics or divers of trucks or other heavy vehicles, eight worked in construction as painters, decorators or masons, three were in the printing industry as photogravure and equipment maintenance workers, ten were farmers, six were engineers and 49 held various technical or office positions. Interviews were conducted with 69 of the patients. All those in medicine had used radioscopy for periods exceeding 10 years. Exposure to petroleum derived substances was high not only among the garage mechanics and drivers, but among those 49 individuals whose occupations did not have particular exposure, but whose hobbies and paraprofessional activities involved use of benzene or other solvents. Of the 69 interviewed, 52 were able to document exposure to benzene or other solvents. [Flandrin G, Collado S; Brit J Haematol 67 (1): 119-20 (1987)]

ENVS - Benzene will enter the atmosphere primarily from fugitive emissions and exhaust connected with its use in gasoline. Another important source is emissions associated with its production and use as an industrial intermediate. In addition, there are discharges into water from industrial effluents and losses during spills. If benzene is released to soil, it will be subject to rapid volatilization near the surface and that which does not evaporate will be highly to very highly mobile in the soil and may leach to groundwater. It may be subject to biodegradation based on reported biodegradation of 24% and 47% of the initial 20 ppm benzene in a base-rich para-brownish soil in 1 and 10 weeks, respectively. It may be subject to biodegradation in shallow, aerobic groundwaters, but probably not under anaerobic conditions. If benzene is released to water, it will be subject to rapid volatilization; the half-life for evaporation in a wind-wave tank with a moderate wind speed of 7.09 m/sec was 5.23 hrs; the estimated half-life for volatilization of benzene from a model river one meter deep flowing 1 m/sec with a wind velocity of 3 m/sec is estimated to be 2.7 hrs at 20 deg C. It will not be expected to significantly adsorb to sediment, bioconcentrate in aquatic organisms or hydrolyze. It may be subject to biodegradation based on a reported biodegradation half-life of 16 days in an aerobic river die-away test. In a marine ecosystem biodegradation occurred in 2 days after an acclimation period of 2 days and 2 weeks in the summer and spring, respectively, whereas no degradation occurred in winter. According to one experiment, benzene has a half-life of 17 days due to photodegradation which could contribute to benzene's removal in situations of cold water, poor nutrients, or other conditions less conductive to microbial degradation. If benzene is released to the atmosphere, it will exist predominantly in the vapor phase. Gas-phase benzene will not be subject to direct photolysis but it will react with photochemically produced hydroxyl radicals with a half-life of 13.4 days calculated using an experimental rate constant for the reaction. The reaction time in polluted atmospheres which contain nitrogen oxides or sulfur dioxide is accelerated with the half-life being reported as 4-6 hours. Products of photooxidation include phenol, nitrophenols, nitrobenzene, formic acid, and peroxyacetyl nitrate. Benzene is fairly soluble in water and is removed from the atmosphere in rain. The primary routes of exposure are inhalation of contaminated air, especially in areas with high traffic, and in the vicinity of gasoline service stations and consumption of contaminated drinking water. (SRC) [CITATION]

FATE - TERRESTRIAL FATE: If benzene is released to soil it will be subject to rapid volatilization near the surface. That which does not evaporate will be highly to very highly mobile in soil and may leach to groundwater. The effective half-lives for volatilization without water evaporation from soil to benzene uniformly distributed to 1 and 10 cm in soil were 7.2 and 38.4 days, respectively(2). It may be subject to biodegradation based on reported biodegradation of 24% and 47% of the initial 20 ppm benzene in a based-rich para-brownish soil in 1 and 10 weeks, respectively(1). It may be subject to biodegradation in shallow, aerobic groundwaters, but probably not under anaerobic conditions. [(1) Haider K et al; Arch Microbiol 96: 183-200 (1974) (2) Jury WA et al; J Environ Qual 13: 573-9 (1984)]

FATE - AQUATIC FATE: If benzene is released to water, it will be subject to rapid volatilization; the half-life for evaporation in a wind-wave tank with a wind speed of 7.09 m/sec was 5.23 hrs(1); the estimated half-life for volatilization of benzene from a model river one meter deep flowing 1 m/sec with a wind velocity of 3 m/sec is estimated to be 2.7 hrs at 20 deg C(SRC). It will not be expected to significantly adsorb to sediment, bioconcentrate in aquatic organisms or hydrolyze. It may be subject to biodegradation based on a reported biodegradation half-life of 16 days in an aerobic river die-away test(2). In a marine ecosystem, biodegradation occurred in 2 days after an acclimation period of 2 days and 2 weeks in the summer and spring, respectively, whereas no degradation occurred in winter(3). [(1) Mackay D, Yeun ATK; Environ Sci Technol 17: 211-7 (1983) (2) Vaishnav DD, Babeu L; Bull Environ Contam Toxicol 39: 237-44 (1987) (3) Wakeman SG et al; Bull Environ Contam Toxicol 31: 582-4 (1983)]

FATE - AQUATIC FATE: Evaporation was the primary loss mechanism in winter in a mesocosm experiment which simulated a northern bay where the half-life was 13 days(1). In spring and summer the half-lives were 23 and 3.1 days, respectively(1). In these cases biodegradation plays a major role and takes about 2 days(1). However, acclimation is critical and this takes much longer in the colder water in spring(1). According to one experiment, benzene has a half-life of 17 days due to photegradation(2) which could contribute to benzene's removal. In situations of cold water, poor nutrients, or other conditions less conducive to microbial, photolysis will play a important role in degradation(SRC). [(1) Wakeham SG et al; Bull Environ Contam Toxicol 31:

582-4 (1983) (2) Hustert K et al; Chemosphere 10: 995-8 (1981)]

FATE - ATMOSPHERIC FATE: If benzene is released to the atmosphere, it will exist predominantly in the vapor phase(3). Gas-phase benzene will not be subject to direct photolysis but it will react with photochemically produced hydroxyl radicals with a half-life of 13.4 days calculated using an experimental rate constant for the reaction. The reaction time in polluted atmospheres which contain nitrogen oxides or sulfur dioxide is accelerated with the half-life being reported as 4-6 hours(2). Products of photooxidation include phenol, nitrophenols, nitrobenzene, formic acid, and peroxyacetyl nitrate. Benzene is fairly soluble in water and is removed from the atmosphere in rain(1). [(1) Kato T et al; Yokohama Kokuritsu Diagaku Kankyo Kagaku Kenkyu Senta Kiyo 6: 11-20 (1980) (2) Korte F, Klein W; Ecotox Environ Saftey 6: 311-27 (1982) (3) Eisenreich SJ et al; Environ Sci Technol 15: 30-8 (1981)]

BIOC - BCF: eels (Anguilla japonica) 3.5(1); pacific herring (Clupea harengus pallasi) 4.4(2); goldfish 4.3(3). Based on a reported log Kow of 2.13(4), a BCF of 24 was estimated(5,SRC). Based on the reported and estimated BCF, benzene will not be expected to bioconcentrate in aquatic organisms(SRC). [(1) Ogata M, Miyake Y; Water Res 12: 1041-4 (1978) (2) Korn S et al; Fish Bull Natl Marine Fish Ser 75: 633-6 (1977) (3) Ogata M et al; Bull Environ Contam Toxicol 33: 561-7 (1984) (4) Hansch C, Leo AJ; Medchem Project Issue No. 26 Claremont, CA: Pomona College (1985) (5) Lyman WJ et al; Handbook of Chem Property Estimation Methods NY: McGraw-Hill p. 5-5 (1982)]

KOC - Koc: Woodburn silt loam 31(1); 31.7-143(4); 83(8). Leaches in soil, passes through soil during bank infiltration(2,3). Based on a reported log Kow of 2.13(5), a Koc of 98 was estimated(6,SRC). Based on the reported and estimated Koc values, benzene will be expected to exhibit very high to high mobility in soil(7) and therefore may leach to groundwater(SRC). [(1) Chiou CT et al; Environ Sci Technol 17: 227-31 (1983) (2) Piet GJ, Morra CF; pp. 31-42 in Artifical Groundwater Recharge L Huisman, TI Olsthorn eds Marshfield MA; Pitman Pub (1983) (3) Green WJ et al; J Water Pollut Control Fed 53: 1347-54 (1981) (4) Sabljic A; J Agric Food Chem 32: 243-6 (1984) (5) Hansch C, Leo AJ; Medchem Project Issue No. 26 Claremont, CA: Pomona College (1985) (6) Lyman WJ et al; Handbook of Chem Property Estimation Methods NY: McGraw-Hill p. 4-9 (1982) (7) Swann RL et al; Res Rev 85: 17-28 (1983) (8) Kenaga EE; Ecotox Environ Safety 4: 26-38 (1980)]

VWS - Half-lives for evaporation of benzene from seawater in a mesocosm simulating Narragansett Bay, RI, containing the associated planktonic and microbial communities, varied with the seasons: spring (15 Apr-18 Jun) half-life 23 days, summer (19 Aug-8 Sept) 3.1 days, winter (4 Mar-4 May) 13 days(1). The effective half-lives for volatilization without water evaporation of benzene uniformly distributed at a rate of 1 kg/ha to 1 and 10 cm in soil with an organic carbon content of 1.25% were 7.2 and 38.4 days, respectively(2). The half-life for evaporation in a wind-wave tank with a wind speed of 7.09 m/sec was 5.23 hr(3). [(1) Wakeham SG et al; Environ Sci Technol 17: 611-7 (1983) (2) Jury WA et al; J Environ Qual 13: 573-9 (1984) (3) Mackay D, Yeun ATK; Environ Sci Technol 17: 211-7 (1983)]

VWS - The estimated half-life for volatilization of benzene from a river one meter deep flowing 1 m/sec with a wind velocity of 3 m/sec is estimated to be 2.7 hrs at 20 deg C(2,SRC) based on a reported Henry's Law constant of 5.3X10-3 atm-cu m/mole(1). Based on a reported vapor pressure of 95.2 mm Hg at 25 deg C(3), evaporation of benzene from surface soil and other surfaces is expected to be rapid(SRC). [(1) Hine J, Mookerjee PK; J Org Chem 40: 292-8 (1975) (2) Lyman WJ et al; Handbook of Chem Property Estimation Methods NY: McGraw-Hill pp. 15-9 to 15-31 (1982) (3) Riddick JA et al; Organic Solvents: Physical Properties and Methods of Purification. Techniques of Chemistry 4th ed. Wiley-Interscience pp. 1325 (1986)]

RTEX - Human populations are primarily exposed to benzene through inhalation of contaminated ambient air particularly in areas with heavy traffic and around filling stations. In addition, air close to manufacturing plants which produce or use benzene may contain high concentrations of

benzene(1,2). Another source of exposure from inhalation is from tobacco smoke(1). Although most public drinking water supplies are free of benzene or contain <0.3 ppb, exposure can be very high from consumption of contaminated sources drawn from wells contaminated by leaky gasoline storage tanks, landfills, etc(SRC). Although benzene has been detected in various food items, data is too scant to estimate exposure from ingestion of contaminated food(SRC). [(1) IARC; Monograph, Some Industrial Chem and Dyestuffs 29: 99-106 (1982) (2) Graedel TE; Chem Compounds in the Atmos, New York, NY Academic Press (1978)]

RTEX - Human Exposure to Atmospheric Benzene from Emission Sources: Chemical manufacturing: numbers in parenthesis are annual average concentration levels in ppb, multiply these concentrations by 10 to get 8 hr worst case levels. 7,497 (0.1-1.0), 970,000 (1.1-2.0), 453,000 (2.1-4.0), 644,000 (4.1-10.0), 319,000 (>10.0), 9,883,000 (total). Coke ovens: 15,726,000 (0.1-1.0), 521,000 (1.1-2.0), 50,000 (2.1-4.0), 2,000 (4.1-10.0), 16,299,000 (total). People using gasoline service stations: 37,000,000 (total) - 245 ppb for 1.5 hr/yr/person. People residing near gasoline service stations: 87,000,000 (0.1-1.0), 31,000,000 (1.1-2.0), 118,000,000 (total). Petroleum refineries: 6,529,000 (0.1-1.0), 64,000 (1.1-2.0), 4,000 (2.1-4.0), <500 (4.1-10.0), 6,597,000 (total). Solvent operations (rubber related industries): 208,000 (0.1-10), 5,000 (1.1-2.0), 2,000 (2.1-4.0), 215,000 (total). Storage and Distribution: very few exposures (<0.1 ppb). Urban exposure (auto emissions): 68,337,000 (0.1-1.0), 45,353,000 (1.1-2.0), 113,690,000 (total)(1). [(1) Stanford Research Institute; Human Exposure to Atmospheric Benzene. Center for Resource and Environmental Studies Report No. 30, p 3, Menlo Park, CA: SRI (1977)]

RTEX - NIOSH (NOES Survey 1981-1983) has statistically estimated that 35,577 workers are exposed to benzene in the USA(1). NIOSH (NOHS Survey 1972-1974) has statistically estimated that 1,495,706 workers are exposed to benzene in the USA(2). [(1) NIOSH; The National Occupational Exposure Survey (NOES) (1983) (2) NIOSH; The National Occupational Hazard Survey (NOHS) (1974)]

RTEX - As estimated 3 million workers may possibly be exposed to benzene. Exposure may occur during the production of benzene or during the use of substances containing the chemical as an ingredient or contaminant. ... Because of the ubiquitous nature of benzene, more than 75 percent of the population probably has been exposed to the chemcial. [DHHS/NTP; Fourth Annual Report On Carcinogens p.35 (1985) NTP 85-002] **PEER REVIEWED**

RTEX - Estimates indicate that possibly 800,000 persons may be exposed to benzene from coke oven emissions at levels greater than 0.1 ppm; 5 million persons may be exposed to benzene from petroleum refinery emissions at levels of 0.1 to 1.0 ppm. [DHHS/NTP; Fourth Annual Report On Carcinogens p.35 (1985) NTP 85-002]

CaPAHs

NAME - BENZO(A)PYRENE
SY - 3,4-BENZ(A)PYRENE
SY - BENZ(A)PYRENE
SY - BaP
SY - BP
MF - C20-H12
USE - ... used extensively as a positive control in a variety of /laboratory mutagenicity & carcinogenicity/ short-term tests. [IARC MONOGRAPHS. 1972-PRESENT V32 215 (1983)]
USE - RESEARCH CHEMICAL [SRI]
USE - NOT USED COMMERCIALLY IN USA [SRI]
BP - Boiling point: > 360 deg C at 760 mm Hg [NIOSH OSHA. OCCUPAT HEALTH GUIDE CHEM HAZARDS. 1981 , p. 3]
MP - 179-179.3 DEG C [MERCK INDEX. 10TH ED 1983 , p. 157]
SOL - 0.0038 MG (+/- 0.00031 MG) IN 1 L WATER @ 25 DEG C [MACKAY D, SHIN WY; J CHEM ENG DATA 22: 399 (1977)]
SOL - SOL IN BENZENE, TOLUENE, XYLENE; SPARINGLY SOL IN ALCOHOL, METHANOL [MERCK INDEX. 10TH ED 1983 , p. 157]
SOL - SOL IN ETHER [PATTY. INDUS HYG & TOX 3RD ED VOL2A,2B,2C 1981-82 , p. 3346]
SOL - Solubility in aqueous caffeine is higher than in water; also, native DNA has a solubilizing effect [IARC MONOGRAPHS. 1972-PRESENT V3 92 (1973)]

NAME - BENZ(A)ANTHRACENE
SY - 1,2-BENZ(A)ANTHRACENE
SY - 1,2-BENZANTHRACENE
SY - 1,2-BENZANTHRENE
SY - 1,2-BENZOANTHRACENE
SY - 2,3-BENZOPHENANTHRENE
SY - BA
SY - BENZANTHRACENE
SY - BENZANTHRENE
SY - BENZO(A)ANTHRACENE
SY - BENZO(B)PHENANTHRENE
SY - BENZOANTHRACENE
SY - TETRAPHENE
MF - C18-H12
USE - RESEARCH CHEM (NO EVIDENCE OF COMMERCIAL USE IN USA) [SRI]
BP - NO DATA
MP - 162 DEG C [WEAST. HDBK CHEM & PHYS 67TH ED 1986-87 C-105]
SOL - 0.014 mg/l in water at 25 deg C [USEPA; Ambient Water Quality Crit Doc: Polynuclear Aromatic Hydrocarbons (Draft) p.A-4 (1980)]
SOL - SOL IN ETHER, ALCOHOL, ACETONE, BENZENE [WEAST. HDBK CHEM & PHYS 67TH ED 1986-87 C-105]
SOL - SOL IN MOST ORG SOLVENTS [MERCK INDEX. 10TH ED 1983 , p. 150] **PEER
SOL - SLIGHTLY SOL IN ACETIC ACID [WEAST. HDBK CHEM & PHYS 60TH ED 1979 C-144]
o EMCE - CLINICAL EFFECTS :
  - o In general, PAHs have a low order of acute toxicity in humans.
  - o PAHs and other compounds found in COAL TAR can produce a variety of non-cancer effects with chronic exposure.

1. EYES: Photosensitivity and irritation may occur.
2. RESPIRATORY: Irritation with cough and bronchitis may be noted.
3. GASTROINTESTINAL: Leukoplakia may be seen.
4. DERMAL: "Coal tar warts" (precancerous lesions enhanced by UV light exposure), erythema, and dermal burns may develop.

5. HEPATOTOXICITY/NEPHROTOXICITY: Mild hepatotoxicity or mild nephrotoxicity have been noted.

o CANCER is the most significant PAH toxicity endpoint.

1. Increased incidences of CANCERS of the SKIN, BLADDER, LUNG, and GASTROINTESTINAL TRACT have been described in PAH-exposed workers.

HEENT

o EYE IRRITATION: Photosensitivity and irritation may occur following chronic exposure.

RESPIRATORY

o COUGH: Irritation with cough and bronchitis may be noted with chronic exposure.

GASTROINTESTINAL

o LEUKOPLAKIA: May be seen with chronic exposure.

HEPATIC

o HEPATOTOXICITY: Mild hepatotoxicity has been noted in PAH-exposed rats.

GENITOURINARY

o NEPHROTOXICITY: Mild nephrotoxicity has been noted in PAH-exposed rats.

HEMATOLOGIC

o ANEMIA: In rats chronically fed PAHs, agranulocytosis, anemia, leukopenia, and pancytopenia have been observed.

DERMATOLOGIC

o PRECANCEROUS LESIONS: "Coal tar warts" (precancerous lesions enhanced by UV light exposure), erythema, and dermal burns may develop following chronic exposure.

PREGNANCY/BREAST MILK

o Female offspring of experimental animals exposed to PAHs during pregnancy have a decrease in the number of functional oocytes, sometimes such that they are infertile.

o PAHs are lipophilic and are excreted in breast milk, allowing for secondary exposure of nursing infants, although the potential significance of such exposure has not been defined.

CARCINOGENICITY

o CANCER is the most significant PAH toxicity endpoint. CHRONIC or REPEATED EXPOSURE increases the likelihood of cancer initiation, as well as the potential for metabolism of a PAH procarcinogen to a carcinogen.

1. The first occupational cancer described was that of SCROTAL CANCER in chimney sweeps exposed to PAHs in soot and ash by Dr Percival Pott in 1775.
2. Increased incidences of CANCERS of the SKIN, BLADDER, LUNG, and GASTROINTESTINAL TRACT have been described in PAH-exposed workers.

a. OCCUPATIONS: Studies have noted increased LUNG CANCER and a SUGGESTION of increased GASTROINTESTINAL CANCER incidence in COAL CARBONIZATION, COAL GASIFICATION, and COKE OVEN WORKERS.

IMMUNOLOGIC

o IMMUNE FUNCTION: An effect of PAHs on immune function might aid in the development of neoplasms. A number of PAH compounds are immunotoxic, and some suppress selective components of the immune system.

GENOTOXICITY

o Arylamines derived from carcinogenic PAHs are mutagenically activated through S9-mediated metabolism of the related amine.

o Nitro-PAH derivatives are potent bacterial mutagens. The mutagenic activity is dependent on enzymatic reduction of the nitro group.

o In cultured human cells, benzo(a)pyrene and 7,12-demethylbenz(a)anthracene only caused a significant increase in T6 guanine-resistant mutations in the presence of cultured rat hepatocytes.

o Nitrated PAHs have caused dose-dependent cell transformations in Syrian hamster embryo cells. Metabolic reduction of the nitro- group on PAHs may be involved in their mutagenic effects.

1. Persons with a high degree of inducibility of the enzyme, aryl hydrocarbon hydroxylase, may be a high-risk population.

HTOX - PERSISTENT NODULE ... DIAGNOSED AS SQUAMOUS EPITHELIOMA DEVELOPED IN A MAN WHO HAD BEEN EXPOSED TO B(A)P FOR 3 WK WHILE HE WAS CARRYING OUT AN EXPERIMENT IN MICE (KLAR, 1938). [IARC MONOGRAPHS. 1972-PRESENT V3 115 (1973)]

HTOX - CULTURES OF HUMAN EPIDERMAL CELLS EXHIBIT TOXIC RESPONSE IN PRESENCE OF B(A)P, SUGGESTING THAT CELLS POSSESS MICROSOMAL ARYL HYDROLASE SYSTEM

WHICH IS CAPABLE OF CONVERTING THE HYDROCARBON INTO A TOXIC METABOLITE. [IARC MONOGRAPHS. 1972-PRESENT V3 114 (1973)]

HTOX - IN CELL TRANSFORMATION STUDIES, 3,4-BENZOPYRENE WAS SLIGHTLY POSITIVE IN THE ACTIVATED HUMAN WI-38 TEST & PRACTICALLY NEGATIVE IN THE NON-ACTIVATED /TEST/ ... [PATTY. INDUS HYG & TOX 3RD ED VOL2A,2B,2C 1981-82 , p. 3364]

HTOX - A 1% soln of benzo(a)pyrene in benzene was applied daily to protected & unprotected surfaces of skin of 26 patients suffering from pemphigus vulgaris, mycosis fungoides, prokeratosis, xeroderma pigmentosum, basal cell cancer, squamous cell cancer, lupus erythematosis, psoriasis, syphilis in various stages or ringworm. The period of application did not exceed 4 mo, & diameter of treated area was 2 cm. A progressive series of alterations developed in normal skin (chronically): erythema, pigmentation, desquamation, formation of verrucae, /clinically not true verrucae/ & infiltration. The manifestations regressed completely within 2 to 3 mo of cessation of treatment. Clinically, perceptible erythema occurred in only 2 patients with basal cell cancer. Pigmentation, which occurred in all patients, consisted of an increase in melanin in basal cell layer of epidermis & was more evident in exposed skin (eg, hand, face). It developed more readily in skin of senile individuals than in younger patients. Rarely, small masses of pigment granules were found in the more superficial layers. Desquamation was proportional in extent to erythema of the 1st stage. The formation of verrucae was the most constant manifestation caused by treatment. The skin of patient with xeroderma pigmentosum did not react differently ... from that of other patients (Cottini & Mazzone, 1939). [IARC MONOGRAPHS. 1972-PRESENT V32 216 (1983)]

HTOX - Human bronchial mucosa treated with benzo(a)pyrene in organ culture showed destruction of all cell types & distortion of columnar cell morphology but not of regenerative epithelium (Crocker et al, 1973). [IARC MONOGRAPHS. 1972-PRESENT V32 216 (1983)]

HTOX - The effects of B(a)P on ... human fetal lung ... /cultures have been observed to produce/ epithelial hyperplasia & inhibition of connective tissue growth (Lasnitzki, 1956). [IARC MONOGRAPHS. 1972-PRESENT V32 214 (1983)]

HTOX - ... /SIR PERVICAL POTT/ DESCRIBED SCROTAL CANCER IN CHIMNEY SWEEPS WHO HAD BEEN EXPOSED SINCE CHILDHOOD TO CONTACT WITH SOOT FROM COAL FIRES. ... FIRST INSTANCE OF OCCUPATIONAL CANCER EVER TO BE DESCRIBED. /COAL SOOT/ [ENCYC OCCUPAT HEALTH & SAFETY 1971 , p. 1344]

HTOX - A previously reported case-referent study of 85 cases of bladder cancer among aluminum smelter workers and 255 matched referents revealed an excess risk among workers exposed to coal tar pitch volatiles. For the study reported in the present investigation these data have been augmented by estimates of past workplace exposure to total tar (benzene soluble matter) and to benzo(a)pyrene (BaP). From these new data, exposure-response relationships have been estimated by maximum likelihood. A linear relationship between cumulative exposure and relative risk and a minimum latency period of ten years were assumed ... and found compatible with the data. Under these assumptions, relative risk increased for each year of exposure to benzene-soluble matter at a concentration of 1 mg/cu m by 13%, the 95% confidence interval being 5-31. The corresponding figure for BaP (as micrograms/cu m X year) was 2.3%. On the basis of these estimates, 40 years of exposure to benzene-soluble matter at the current exposure to benzene-soluble matter at the current exposure limit of 0.2 mg/cu m would lead to a relative risk of 2.4. [Armstrong BG et al; Scand J Work Environ Health 12 (5): 486-93 (1986)]

HTOX - The presence of antibodies to DNA-carcinogen adducts in human serum is demostrated for the first time and shown to provide an objective and reliable means to monitor human exposure to carcinogens and/or mutagens generally found in industrial surroundings and in the atm. Thus, serum from coke oven workers (who are exposed to substantial amt. of benzopyrene) was examined for the presence of BPDE-DNA adducts (BPDE= 7beta,8alpha-dihydroxy-(9alpha,10alpha)epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene) in the DNA of their peripheral blood lymphocytes and serum antibodies to such adducts. Enzyme-linked immunosorbent assays was used to detect the presence and specificity of the antibodies to BPDE-DNA adducts. Eleven of 41 serum samples contained antibodies to the adducts; the predominant Ig isotype of the anti-BPDE-DNA antibodies was IgG. Two of the 11 pos sera had IgM antibodies. The data clearly demonstrate that the presence of BPDE-DNA adducts and/or antibodies are indicators of exposure to benzopyrene or BPDE and results from an immune response to the adducts. [Harris C; Method and Kit for Detecting Human Exposure to Genotoxic Agents US Patent Appl# 778669 (03/14/86)]

HTOX - Workers in coke oven plants have a higher incidence of lung cancer than the general population. They are exposed to a variety of chemcials, in particular the polycyclic aromatic hydrocarbons (PAH), including benzo(a)pyrene. To evaluate the genotoxic effects of PAH exposure, air samples and urine samples were analyzed for PAH by capillary gas chromatography and high-performance liquid chromatography, respectively. Since benzo(a)pyrene is activated to 7-beta,8-alpha-dihydroxy-(9-alpha,10-alpha)-epoxy-7,8,9,10-tetrahydrobenzo(a)pyrene (BPDE) and binds to DNA adducts in lymphocyte DNA. The mean PAH exposure levels are reduced 60% when the workers wore masks during work. When compared to exposure levels, the urinary excretion of PAH was relatively low. Approximately one-third of the workers had detectable putative BPDE-DNA adducts in lymphocytes by ultrasensitive enzymatic radioimmunoassay, and 10% of the samples had emission peaks at 379 nm by synchronous fluorescence spectrophotometry. The four most positive samples were the same in both of the assays. Antibodies to an epitope(s) on BPDE-DNA were found in the sera of approximately one-third of the workers. Detection of DNA adducts and antibodies to these adducts are internal indicators of exposure to benzo(a)pyrene. [Haugen A et al; Cancer Res 46 (8): 4178-83 (1986)]

HTOX - Serum was taken from male human subjects (age 20-40 years) and the uptake of (14)C-benzo(a)pyrene (B(a)P) and subsequent extraction of bound B(a)P was determined by radio-scintillation techniques. The initial uptake velocity for B(a)P by human (60 ug/ml x hr) was similar to that of rat serum for all concentrations of B(a)P used. Maximum uptake of B(a)P was estimated at 230 ug/ml for human serum. Gel filtration of human serum containing (14)C-B(a)P revealed that 80% was associated with low-density lipopretieins (LDL), 15% with high-density lipoproteins (HDL), and 5% with albumin. B(a)P binding at concentrations up to and including 50 ug B(a)P/ml was not saturable. In human serum the highest amount of bound B(a)P was associated with LDL (44-47%) and HDL (32-35%) components. [Aaarstad K et al; Toxicology 47 (3): 235-45 (1987)] **QC REVIEWED**

HTOX - Benzo(a)pyrene constitutes ... between 1 and 20% of the total carcinogenic polycyclic aromatic hydrocarbons found in water. [Health & Welfare Canada; Polycyclic Aromatic Hydrocarbons p.1 (1979) Report No. 80-EHD-50]

HTOX - The Carcinogen Assessment Group (CAG), Office of Health and Environmental Assessment in EPA'S Research and Development Office, has

prepared a list of chemical substances for which substantial or strong evidence exists showing that exposure to these chemicals, under certain conditions, causes cancer in humans, or can cause cancer in animal species which in turn, makes them potentially carcinogenic in humans. Substances are placed on the CAG list only if they have been demonstrated to induce malignant tumors in one or more animal species or to induce benign tumors that are generally recognized as early stages of malignancies, and/or if positive epidemiologic studies indicated they were carcinogenic. Benzo(a)pyrene is on that list. [USEPA/CAG; The Carcinogen Assessment Group's List of Carcinogens (7/14/80)]

HTOX - An exptl teratogen, carcinogen, neoplastic agent by way of various routes. ... Mutagenic data are published. [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 383]

HTOX - Epidemiologic evidence suggests that workers intimately exposed to the products of combustion or distillation of bituminous coal are at an increased risk for cancers of the skin, respiratory tract, bladder, and kidney. /Coal tar pitch volatiles/ [NIOSH-OSHA. OCCUPAT HEALTH GUIDE CHEM HAZARDS. 1981 , p. 2]

NTOX - Marked differences in toxic effects of B(a)P have been reported in different strains of mice depending on ... genetic consititution. The Ah locus, which determines inducibility of aryl hydrocarbon hydroxylase, appears to be of particular importance. ... Oral admin of about 120 mg/kg body wt B(a)P per day with diet induced aplastic anemia in nonresponsive (poorly inducible) AKR/N mice (Ah(d)/Ah(d) type) & death within 4 weeks, whereas nonresponsive (markedly inducible) mice (Ah(b)/Ah(b) type) remained healthy for at least 6 months. In former, bone marrow was hypocellular with myeloid precursors & promegakaryocytes. When B(a)P was injected ip (500 mg/kg body wt) instead of being given orally (120 mg/kg body wt), survival time of nonresponsive mice (Ah(b)/Ah(b) type) was ... significantly shorter than that of responsive mice (Ah(d)/Ah(d) type) (Robinson et al, 1975). These differences may be explained in part by the greater capacitiy of bowel & liver of responsive mice to detoxify an orally admin dose of B(a)P metabolically. However, if the hydrocarbon reaches bone marrow & other distal tissues in responsive mice, it is metabolized to toxic metabolites to a greater extent (Nebert et al, 1977). [IARC MONOGRAPHS. 1972-PRESENT V32 214 (1983)]

NTOX - Ip admin of single doses of 10 mg B(a)P produced immediate & sustained reduction in growth rate of young rats (Haddow et al, 1937). [IARC MONOGRAPHS. 1972-PRESENT V32 214 (1983)]

NTOX - The effects of B(a)P on stratification of epithellium in organ cultures of rat trachea (Crocker et al, 1965), stratification of epithelium (Dirksen & Crocker, 1968) ... have been reported. Consistent findings in rat trachea were suppression of mesenchyme, stimulation of basal-cell replication & induction of metaplasia ... When B(a)P (in beeswax pellet) was implanted into isogenically transplanted rat trachea, persistent hyperplasia & metaplasia were observed (Topping et al, 1978). [IARC MONOGRAPHS. 1972-PRESENT V32 214 (1983)] **PEER REVIEWED**

NTOX - A single topical application of 0.05 ml of 1% soln of B(a)P in acetone to the interscapular area of hairless mice (hr/hr) induced an increase in the mitotic rate of epidermal cells (Elgjo, 1968). [IARC MONOGRAPHS. 1972-PRESENT V32 214 (1983)]

NTOX - NO STOMACH TUMORS WERE FOUND AT END OF 110-DAY TREATMENT WITH DIETS CONTAINING UP TO 30 PPM B(A)P. TUMOR INCIDENCES LOWER THAN 10% WERE OBSERVED IN MICE RECEIVING 40-45 PPM FOR 110 DAYS, WHEREAS MICE BEARING STOMACH TUMORS EXCEEDED 70% AMONG THOSE GIVEN 50-250 PPM B(A)P FOR

122-197 DAYS. [IARC MONOGRAPHS. 1972-PRESENT V3 103 (1973)] **PEER

NTOX - SINGLE INTRAGASTRIC ADMIN OF 0.2 MG/MOUSE IN POLYETHYLENE GLYCOL PRODUCED TOTAL OF 14 TUMORS /IN FORESTOMACH/ IN 5 /OF 11/ ANIMALS ... TUMORS APPEARED FOLLOWING SINGLE DOSES OF 0.05 & 0.012 MG/MOUSE IN 0/9 & 2/10 MICE (PEIRCE, 1961). DIET CONTAINING 250 PPM B(A)P FED FOR DIFFERENT PERIODS OF TIME PRODUCED THE FOLLOWING INCIDENCES OF TUMORS OF FORESTOMACH: 1 DAY OF FEEDING, 0%; 2-4 DAYS OF FEEDING, 10%; 5-7 DAYS OF FEEDING, 30-40%; 30 DAYS OF FEEDING, 100% (NEAL & RIGDON, 1967). IN SUBSEQUENT EXPERIMENT, DIET CONTAINING 250 PPM ... FED 140 DAYS TO MICE OF SAME STRAIN STARTING AT 18-30 DAYS OF AGE ... PRODUCED LEUKEMIAS & LUNG ADENOMAS IN ADDN TO STOMACH TUMORS (RIGEDON & NEAL, 1969). THE ABILITY OF B(A)P TO PRODUCE LUNG ADENOMAS WHEN ADMIN IN DIET WAS CONFIRMED IN ANOTHER STUDY (WATTENBERG & LEONG, 1970). [IARC MONOGRAPHS. 1972-PRESENT V3 102 (1973)]

NTOX - SINGLE ORAL ADMIN OF 100 MG B(A)P TO 50-DAY-OLD FEMALE SPRAGUE-DAWLEY RATS PRODUCED MAMMARY TUMORS IN 8 OF 9 ANIMALS (HUGGINS & YANG, 1962). IN ANOTHER STUDY WITH SPRAGUE-DAWLEY RATS OF BOTH SEXES AGED 3.5 MO AT BEGINNING OF EXPT, DAILY DOSES OF 2.5 MG B(A)P PER RAT INDUCED PAPILLOMAS IN ESOPHAGUS & FORESTOMACH IN THREE OUT OF 40 ANIMALS (GIBEL, 1964). [IARC MONOGRAPHS. 1972-PRESENT V3 103 (1973)] **PEER

NTOX - BI-WEEKLY ADMIN OF 2-5 MG B(A)P IN OIL BY STOMACH TUBE PRODUCED 5 PAPILLOMAS OF STOMACH IN 67 HAMSTERS TREATED FOR 1-5 MONTHS, 7 PAPILLOMAS & 2 CARCINOMAS IN 18 TREATED FOR 6-9 MONTHS & 5 PAPILLOMAS IN 8 TREATED FOR 10-11 MONTHS (DONTENWILL & MOHR, 1962). IN SUBSEQUENT EXPT WITH 13 HAMSTERS, A DIET CONTAINING 500 PPM B(A)P GIVEN FOR 4 DAYS/WK FOR UP TO 14 MONTHS CAUSED TOTAL OF 12 TUMORS (2 IN ESOPHAGUS, 8 IN FORESTOMACH & 2 IN INTESTINE) IN 8 HAMSTERS (CHU & MALMGREN, 1965). [IARC MONOGRAPHS. 1972-PRESENT V3 104 (1973)]

NTOX - ... DOSE-RESPONSE STUDIES, INCL A NO-EFFECT DOSE LEVEL ... INDICATE THAT THE THRESHOLD DOSE IS AFFECTED BY THE STRAIN OF MOUSE & THE SOLVENT CHOSEN. THREE WEEKLY APPLICATIONS OF B(A)P IN ACETONE TO CAF1 MICE INDUCED NO SKIN TUMORS AT CONCN OF 0.0005%, TOTAL OF 6 PAPILLOMAS & 2 CARCINOMAS AMONG 19 MICE AT CONCN OF 0.001% & INCREASING INCIDENCE OF BENIGN & MALIGNANT TUMORS WITH PROGRESSIVELY SHORTER LATENT PERIODS AT HIGHER DOSES. IN SWISS & C57BL MICE, TUMORS WERE NOT INDUCED AT CONCN OF 0.001% OR LESS, WHEREAS INCIDENCES APPROACHING 100% WERE FOUND AT 0.005% OR HIGHER CONCN (WYNDER ET AL, 1957). [IARC MONOGRAPHS. 1972-PRESENT V3 104 (1973)]

NTOX - ... /In dose-response study/ toluene was used as solvent & SWR, C3HeB & A/He mice were painted 3 times weekly with different amounts of B(a)P. In SWR & C3HeB mice the lowest effective dose was 0.38 ug B(a)P per application, with an obvious dose-response relationship above this dose for both percentage of tumor-bearing animals & the shortening of latent period; doses of 0.15 ug were ineffective. On the other hand, in A/He mice, paintings with 0.15 to 3.8 ug B(a)P were ineffective, whereas tumors were induced following doses of 19 ug or more (Poel, 1963). With acetone as solvent & using Swiss mice, borderline activity was detected following 3 times weekly painting with 0.1 to 1.0 ug B(a)P, while tumors appeared with 3 ug & above (Roe et al, 1970). [IARC MONOGRAPHS. 1972-PRESENT V3 104 (1973)]

NTOX - ... SEVEN /SKIN/ PAPILLOMAS & FOUR CARCINOMAS /WERE PRODUCED/ AMONG 15 RATS PAINTED WEEKLY WITH 0.5 TO 1% SOLN OF B(A)P IN BENZENE /IN EXPT LASTING 150 DAYS (NAKANO, 1937)/. [IARC MONOGRAPHS. 1972-PRESENT V3 106 (1973)]

NTOX - BI-WEEKLY PAINTINGS WITH A 0.01% SOLN OF B(A)P IN ACETONE FOR 40 DAYS DID NOT PRODUCE SKIN TUMORS AMONG 10 SYRIAN GOLDEN HAMSTERS. A TOTAL OF 8 APPLICATIONS OF 4 DROPS OF 0.8% SOLN IN MINERAL OIL PRODUCED 1

MELANOMA AMONG 25 SURVIVORS AT 33 WEEKS (SHUBIK ET AL, 1960). [IARC MONOGRAPHS. 1972-PRESENT V3 106 (1973)]

NTOX - TWICE-WEEKLY PAINTING WITH 0.3% SOLN OF B(A)P IN BENZENE FOR 400 DAYS PRODUCED 1 CARCINOMA & 10 PAPILLOMAS AMONG 10 RABBITS (SCHURCH & WINTERSTEIN, 1935). THIS RESULT HAS BEEN CONFIRMED REPEATEDLY ... SOME EVIDENCE OF DOSE-RESPONSE RELATIONSHIP ... /COMES/ FROM STUDY ON SMALL GROUPS OF RABBITS PAINTED 5 TIMES WEEKLY WITH CONCN OF B(A)P IN ACETONE RANGING BETWEEN 0.0001% & 0.5%: TUMORS APPEARED FOLLOWING APPLICATION OF ... 0.005% OR MORE (WYNDER ET AL, 1957). [IARC MONOGRAPHS. 1972-PRESENT V3 107 (1973)]

NTOX - ... ONE OR 5 INTRATRACHEAL ADMIN OF 100 MG B(A)P TO 8 RATS PRODUCED AT LEAST 3 LUNG TUMORS. ... 10 MONTHLY INTRATRACHEAL INJECTIONS OF EITHER 0.0005, 0.002, 0.01, 0.05, 0.25, OR 2.5 MG B(A)P MIXED WITH BLOOD SUBSTITUTE, BK-8, & INDIA INK WERE ... /OBSERVED/ FOR 2 YR. NO LUNG TUMORS WERE FOUND AT 2 LOWEST CONCN ... AT HIGHER DOSAGES PERCENTAGES OF ANIMALS DEVELOPING LUNG TUMORS WERE, RESPECTIVELY, 14%, 28%, 43%, & 80% (YANISHEVA, 1971). [IARC MONOGRAPHS. 1972-PRESENT V3 107 (1973)]

NTOX - SEVERAL DOSE-RESPONSE STUDIES ARE AVAIL, SOME ... PERMIT COMPARISON WITH OTHER CARCINOGENS TESTED UNDER SAME CONDITIONS IN SAME LAB. ... ON INDUCTION OF LOCAL SARCOMAS FOLLOWING SINGLE INJECTIONS OF B(A)P IN TRICAPRYLIN, NO TUMORS WERE FOUND FOLLOWING DOSES OF 0.031 MG OR LESS, WHEREAS 4 OF 20 C3H MICE DEVELOPED TUMORS WITH 0.062 MG, HIGHER TUMOR INCIDENCES ... FOLLOWING GREATER DOSES. THE THRESHOLDS OF CARCINOGENICITY FOR 3-METHYLCHOLANTHRENE (MC) & DIBENZ(A,H)ANTHRACENE (DB(A,H)A) IN THE SAME EXPT WERE, RESPECTIVELY, 0.0078 & 0.0019 MG. ... AVG MINIMAL LATENT PERIODS WERE 3 MONTHS FOR B(A)P, 2.5 MONTHS FOR MC & 3.7 MO FOR DB(A,H)A (BRYAN & SHIMKIN, 1943). [IARC MONOGRAPHS. 1972-PRESENT V3 109 (1973)]

NTOX - INDUCTION OF HEPATOMAS &/OR LUNG ADENOMAS & OCCASIONAL TUMORS AT OTHER SITES IN MICE OF DIFFERENT STRAINS WAS RECORDED FOLLOWING ADMIN OF B(A)P DURING FIRST DAYS OF LIFE AT DOSES OF 20-40 UG/MOUSE IN DIFFERENT SOLVENTS (PIETRA ET AL, 1961; ROE & WATERS, 1967; TOTH & SHUBIK, 1967; GRANT ET AL, 1968). [IARC MONOGRAPHS. 1972-PRESENT V3 110 (1973)]

NTOX - WEEKLY IP INJECTIONS OF 2 MG B(A)P AS COLLOIDAL SUSPENSION IN WATER TO 80 ST/A MICE INDUCED ABDOMINAL FIBROSARCOMAS IN 81% OF 40 FEMALES & 73% OF 40 MALES WITH AVERAGE LATENT PERIOD OF 33 WK (PAYNE, 1958). [IARC MONOGRAPHS. 1972-PRESENT V3 112 (1973)]

NTOX - SINGLE IP ADMIN OF 10 MG B(A)P PRODUCED 2 MAMMARY & 2 UTERINE CARCINOMAS AMONG 10 WISTAR RATS WITHIN 1 YR, WHEREAS NO MAMMARY TUMORS WERE FOUND IN CONTROLS. (PAYNE, 1958). [IARC MONOGRAPHS. 1972-PRESENT V3 112 (1973)]

NTOX - AT ORAL DOSES EST TO BE BETWEEN 6-12 MG/KG BODY WT/DAY, LEUKEMIA DEVELOPED 100 OR MORE DAYS LATER IN NONRESPONSIVE AHD/AHD HOMOZYGOUS MOUSE BUT NOT IN RESPONSIVE AHB/AHD HETEROZYGOTE. AN EXCESS OF DIETARY ALPHA-NAPHTHAFLAVONE (ANF) 20 TIMES GREATER THAN THE AMT OF B(A)P PREVENTED THESE HEMATOPOIETIC NEOPLASMS. ANF-SENSITIVE METABOLISM OF B(A)P PRESUMABLY CYTOCHROME P1-450 IN THE BONE MARROW OF AHD/AHD INDIVIDUALS MAY BE RESPONSIBLE FOR CAUSING THE LEUKEMIA. [NEBERT DW, JENSEN NM; BIOCHEM PHARMACOL 28 (1): 149-52 (1979)]

NTOX - TO IMPROVE AMES CHEM CARCINOGEN SCREENING TEST, SEVERAL SPECIES OF ANIMALS, INCL RATS, MICE, GUINEA PIGS, HAMSTERS, & RABBITS, WERE PRETREATED WITH POLYCHLORINATED BIPHENYL (PCB), 3-METHYLCHOLANTHRENE (3-MC) & PHENOBARBITAL (PB) & TESTED FOR THE EFFECTS OF THE INCREASE OF REVERTANTS COLONIES OF SALMONELLA TYPHIMURIUM TA98 & TA100. AMONG 12 STRAINS OF 5 MAMMALIAN SPECIES, S-9 FRACTION FROM PCB-TREATED HARTLEY GUINEA PIGS PROVED MORE EFFECTIVE THAN THAT FROM PCB-TREATED SD RATS IN DETECTING 3 DIFFERENT TYPES OF MUTAGENS WHICH INCLUDED B(A)P. [SUZUKI H

ET AL; J PESTIC SCI (NIHON NOYAKUGAKU KAISHI) 2 (4): 421-6 (1977)]

NTOX - ... STERILITY /WAS OBSERVED/ IN FEMALE /OFFSTRING OF/ MICE EXPOSED TO 40-160 MG/KG ON GESTATION DAYS 7-16. [SHEPARD. CAT TERATOGENIC AGENTS 4TH ED 1983 , p. 52]

NTOX - A dose of 5 mg/day SC to pregnant rats, can cause death of all fetuses (Wolfe & Bryan, 1939). Oral admin of 10 mg/kg body wt B(a)P to CD-1 mice during pregnancy resulted in marked & specific reduction of gonadal wt but had no effect on body wt of ... male or female offspring. Reduction in fertility & reproductive capacity occurred. With 40 mg/kg body wt/day ... almost complete sterility was observed in offspring of both sexes (Mackenzie & Angevine, 1981). [IARC MONOGRAPHS. 1972-PRESENT V32 214 (1983)]

NTOX - IP admin of 100-150 mg/kg body wt B(a)P to C3H/Anf mice in mid- or late pregnancy results in marked & persistent suppression of the immune system in the offspring (Urso & Gengozian, 1980). Application of B(a)P to skin of pregnant mice (strain unspecified) over 4 generations resulted in sensitization of the offspring to the effects of B(a)P, so that an increase in rate of appearance of papillomas & carcinomas was observed following skin application to offspring over that in control mice that had not been treated in utero (Andrianova, 1971). [IARC MONOGRAPHS. 1972-PRESENT V32 215 (1983)]

NTOX - THREE SC OR IP INJECTIONS TO ICR/HA MICE OF 2-4 MG B(A)P AT 11TH, 13TH & 15TH DAY OF PREGNANCY RESULTED IN INCR INCIDENCE OF LUNG ADENOMAS & INITIATION OF SKIN CARCINOGENESIS IN OFFSPRING (BULAY & WATTENBERG, 1970; BULAY, 1970). FOSTER NURSING DID NOT ALTER THESE EFFECTS (BULAY & WATTENBERG, 1971). [IARC MONOGRAPHS. 1972-PRESENT V3 113 (1973)]

NTOX - Transplacental carcinogenesis has ... been shown in rabbits (Beniashvili, 1978). [IARC MONOGRAPHS. 1972-PRESENT V32 215 (1983)]

NTOX - ... /AMONG RATS FED/ 1 MG /B(A)P/ PER G OF DIET DURING PREGNANCY ... MANY RESORPTIONS & DEAD FETUSES /WERE OBSERVED,/ BUT ONLY ONE MALFORMED FETUS /WAS NOTED/ FROM 7 LITTERS. [SHEPARD. CAT TERATOGENIC AGENTS 4TH ED 1983 , p. 52]

NTOX - OOCYTE DESTRUCTION BY POLYCYCLIC HYDROCARBONS REQUIRES DISTRIBUTION OF THE PARENT HYDROCARBON TO THE OVARY WHERE OVARIAN ENZYMES METABOLIZE THE COMPOUND TO REACTIVE INTERMEDIATES RESPONSIBLE FOR OVOTOXICITY. DESCRIPTIVE ASSAYS OF POLYCYCLIC HYDROCARBON METABOLIC ACTIVATION SUCH AS THE ARYL HYDROCARBON (BENZO(A)PYRENE) HYDROXYLASE ASSAY ARE NOT GOOD PREDICTORS OF STRAIN OR SPECIES DIFFERENCES IN SENSITIVITY TO POLYCYCLIC HYDROCARBON OVOTOXICITY. USING BENZO(A)PYRENE AS A PROBE OF OVARIAN METABOLIC PROCESSING SUGGESTS THAT THE RATE OF FORMATION OF METABOLITES ALONG THE METABOLIC PATHWAY TO THE 7,8-DIHYDRODIOL-9,10-EPOXIDE MAY BE THE APPROPRIATE MEASURE OF THE ROLE OF METABOLIC ACTIVATION IN STRAIN OR SPECIES DIFFERENCES IN SENSITIVITY TO OOCYTE DESTRUCTION IN RATS & MICE. [MATTISON DR ET AL; PROG CLIN BIOL RES 117: 191-202 (1983)]

NTOX - ... The in-utero toxicity in relation to the allelic differences at the Ah locus in mice /was studied/. A dose of 50-300 mg/kg was given ip on days 7 or 10. ... the Ah genotype of individual fetuses /was identified/ by measurement of AHH inducibility & ... /showed/ that when the mothers were Ah nonresponsive, the fetuses with Ah responsive genotype showed decreased body wt & higher resorption & malformation rates while Ah nonresponsive fetuses in the same uterus did not. The type of defect included mainly club foot, hemangioma, cleft lip, & cleft palate. All of these defects tend to be assoc with late organogenesis (Shum et al, 1979). ... using the same general protocol & 150 or 300 mg/kg on day 8 /another study/ confirmed the findings of ... toxicity (reduced fetal wt & incr resorptions) but ... not ... the same increase in malformations. ... only an increase in cervical ribs ... /which/ occurred among fetuses from Ah responsive mothers /was found/

(Hoshino et al, 1981). [SHEPARD. CAT TERATOGENIC AGENTS 4TH ED 1983 , p. 52]

NTOX - THE CARCINOGENIC POTENTIAL IN NONHUMAN PRIMATES (MONKEYS) OF A VARIETY OF CHEMICALS, DIFFERING WIDELY IN CHEMICAL STRUCTURES, ENVIRONMENTAL POLLUTANTS, MODEL RODENT CARCINOGENS, & NITROSO CMPD WAS EXAMINED. WITH THE EXCEPTION OF URETHANE, NONE OF THE MODEL RODENT CARCINOGENS WAS CARCINOGENIC IN THE OLD & NEW WORLD SPECIES OF MONKEY, ALTHOUGH BENZO(A)PYRENE PRODUCED TUMORS IN MORE PRIMITIVE PRIMATES (BUSH BABIES & TREE SHREWS), THAN IN RODENTS. [ADAMSON RH, SIEBER SM; BASIC LIFE SCI 24 (ORGAN SPECIES SPECIF CHEM CARCINOG): 129-56 (1983)]

NTOX - THE TUMORIGENICITY OF BENZO(A)PYRENE (B(A)P) APPLIED TOPICALLY AS A SKIN TUMOR INITIATOR IN SENCAR MICE & THE FORMATION OF EPIDERMAL B(A)P/DEOXYRIBONUCLEOSIDE ADDUCTS WERE COMPARED OVER A SIMILAR RANGE OF DOSES (50-1600 NMOL). THE TUMOR-INITIATING ACTIVITY OF B(A)P, ITS COVALENT BINDING TO MOUSE EPIDERMAL DNA, & THE FORMATION OF THE MAJOR HYDROCARBON/DEOXYRIBONUCLEOSIDE ADDUCT SHOWED APPROX PARALLEL DOSE-RESPONSE CURVES. THE HALF-LIFE OF THE B(A)P/DEOXYRIBONUCLEOSIDE ADDUCTS & THE TOTAL RADIOACTIVITY BOUND TO THE DNA WERE 4.5 & 5.5 DAYS, RESPECTIVELY. HOWEVER, IN SPITE OF THE LOSS OF MEASURABLE DNA-BOUND MATERIAL, THE TUMOR YIELD WAS UNCHANGED REGARDLESS OF WHETHER PROMOTION WAS BEGUN 7 OR 21 DAYS AFTER INITIATION. THUS, THERE WAS A POSSIBLE CAUSAL RELATION BETWEEN B(A)P/DEOXYRIBONUCLEOSIDE ADDUCT FORMATION & PAPILLOMA FORMATION IN MOUSE SKIN. [ASHURST SW ET AL; CANCER RES 43 (3): 1024-9 (1983)]

NTOX - In a series of soil and hydrocultures of the higher plants, tobacco, rye, and radish, as well as algae cultures of lower plants (Chlorella vulgaris, Scenedesmus obligurus, and Ankistrodesmus) /results indicate/ that certain polycyclic aromatic hydrocarbons have growth-promoting effects on plants. Further, the degree of the promoting effect corresponded to the oncogenic activity of the hydrocarbon. The six polycyclic aromatic hydrocarbons found in plants were tested one at a time or in combination. Considerable growth-promotion was noted (near to 100% in some cases) with the effectiveness of hydrocarbons ranked as follows: (1) Benzo(a)pyrene (2) Benzo(a)anthracene (3) Indeno (1,2,3-cd)pyrene, Benzo(b)fluoranthene (4) Fluoranthene (5) Benzo(ghi)perylene. [Graf W, Nowak W; Arch Hyg Bakt 150: 513-28 (1968) as cited in Health & Welfare Canada; Polycyclic Aromatic Hydrocarbons p.67 (1979) Report No. 80-EHD-50]

NTOX - Histological and skeletal examinations were performed on rainbow trout alevins reared in 0.00, 0.08, 0.21, 0.39, 1.48, 2.40, or 2.99 ng/ml aqueous benzo(a)pyrene (BaP). Nuclear pycnosis and karyorrhexis were most common in neuroectodermal and mesodermal derivatives and in liver of BaP-treated alevins. Microphthalmia was noted in 17% of the test fish and was frequently associated with a patent optic fissure. Depressed mitotic rates in the retina and brain, but not liver, were seen in alevins reared in 0.21 to 1.48 ng/ml aqueous BaP. Test alevins had a significantly higher incidence of skeletal malformations in the skull and vertebral column and abnormalities of vertebral arcualia often corresponded to areas of kyphoscoliotic flexures. The ecological significance of such morphological abnormalities would be decreased feeding and growth and inability to escape predation, leading to reduced survival. Persistent mixed function oxygenase induction in less affected larvae would lead to continuing production of cytotoxic, mutagenic, and carcinogenic BaP metabolites resulting in anemia, impaired ability to respond to environmental stress and disease, and possibly latent tumorigenesis. [Hose JE et al; Arch Environ Contam Toxicol 13 (6): 675-84 (1984)]

NTOX - The teratogenic effects of environmental levels of ... benzo(a)pyrene,

were investigated using the purple sea urchin (Strongylocentrotus purpuratus) and were related to embryonic cytotoxicity and genotoxicity as evidenced by the presence of aberrant chromosome arrangements during mitosis. Developmental abnormalities were observed in gastrulae treated with initial benzo(a)pyrene concentrations of 1-50 ng/ml relative to solvent (ethanol)-treated control embryos. However, genotoxic effects were significant at the lowest benzo(a)pyrene dose tested, 0.5 ng/ml. Micronucleus formation, a widely used test of genotoxicity in mammals was observed in embryos exposed to 1 to 50 ng/ml of benzo(a)pyrene. the results from this cytogenetic analysis demonstrated that mitotic inhibition and aberrations are more sensitive indicators of benzo(a)pyrene induced damage than are developmental effects. [Hose JE et al; Arch Environ Contam Toxicol 12 (3): 319-32 (1983)]

NTOX - Thirty-four ducks were given single intratracheal dose of 50-200 mg B(a)P in Tween 80. Survival rate was poor. One ... /duck/ developed a lung carcinoma & two had bronchial squamous metaplasia (Rigdon & Neal, 1965). [IARC MONOGRAPHS. 1972-PRESENT V3 109 (1973)]

NTOX - The growth rate of mouse ascites sarcoma cells in culture was slightly (6%) inhibited when B(a)P was added at concn of 1 umol/ml dissolved in dimethyl sulfoxide. [IARC MONOGRAPHS. 1972-PRESENT V32 214 (1983)]

NTOX - Several metabolites /of B(a)P/ can induce mutations, transform cells &/or bind to cellular macromolecules; however, only a 7,8-diol-9,10-epoxide is presently considered to be an ultimate carcinogenic metabolite. [IARC MONOGRAPHS. 1972-PRESENT V32 215 (1983)]

NTOX - B(a)P has been shown to be carcinogenic in exptl animals. ... /It/ is embryotoxic & teratogenic in mice; the inducibility of aryl hydrocarbon hydroxylase activity in dams & fetuses is an important factor in determining these effects. A reduction in fertility in ... male & female offspring was observed in mice following exposure ... in utero. B(a)P undergoes metabolism to reactive electrophiles capable of binding covalently to DNA. It was active in assays for bacterial DNA repair, bacteriophage induction & bacterial mutation; mutation in Drosophila melanogaster; DNA binding, DNA repair, sister chromatid exchange, chromosomal aberrations, point mutation & transformation in mammalian cells in culture; & in tests in mammals in vivo, including DNA binding, sister chromatid exchange, chromosomal aberration, sperm abnormality & the somatic specific locus (spot) test. There is sufficient evidence that benzo(a)pyrene is active in short-term tests. [IARC MONOGRAPHS. 1972-PRESENT V32 216 (1983)]

NTOX - Buffalo river sediment extracts contained polynuclear aromatic hydrocarbons (PAH) which caused skin darkening, hyperplasia, skin papillomas, mild coarsening and local pigmentations in the brown bullhead (Ictalurus nebulosus). Sixteen PAH were identified in the sediment extract: fluorene, phenanthrene, anthracene, fluoranthene, 2-methylphenanthrene, pyrene, 2-methylanthracene, benzanthracene, chrysene, perylene, benzo(f)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, dibenz(a,h)anthracene, benzo(g,h,i)perylene, and indeno(1,2,3-c,d)pyrene. [Black JJ; Polynucl Aromat Hydrocarbons Int Symp 7th 99-11 (1983)]

NTOX - The teratogenicity of benzo(a)pyrene (BP), after direct injection into embryonal Swiss mice /was studied/. The compounds were dissolved in acetone and trioctanoin (1:1) and injected at doses ranging from 0.4 to 16.0 nmol/embryo on days 10, 12, and 14 of deve the transplacental effects of BP given at the same gestational days and at comparable dose levels were also evaluated. The control groups received 0.5, 1.0, or 2.0 mul/embryo of vehicle on days 10, 12, or 14 of pregnancy, respectively. The fetuses were examined when they were 18 days old. On

the basis of gross external and internal malformations, the administration of BP (both transplacental intraembryonal injection) caused no significant increase of malformed fetuses in any of the developmental stages considered. [Barbieri O et al; Cancer Res 46 (1): 94-8 (1986)]

NTOX - Rats and mice were exposed to combustion gases of coal-burning furnace enriched with benzo(a)pyrene (50-90 mug/cu m) and other polycyclic aromatic hydrocarbons (PAH) 16 hr/day, 5 days/wk. After approx 22-mo exposure, the incidence of lung neoplasm was approx 10-fold above controls. /Benzo(a)pyrene/ [Heinrich U et al; Exp Pathol 29 (1): 29-34 (1986)]

NTOX - [McClellan RD; Annu Rev Pharmacol Toxicol 27: 279-300 (1987)] In animal studies, exposure to high levels of diesel exhaust particulates overwhelms the normal clearance mechanisms and results in lung burdens of diesel exhaust particulates that exceed those predicted from observations at lower exposure concentrations. A variable amount of the mass of diesel exhaust particulates is extractable with strong organic solvents. The extracted material contains more than a thousand individual compounds and is mutagenic in a number of bacterial and mammalian cell assays. Bioassay-directed chemical analysis of diesel exhaust particulates had identified several hundred compounds. Many are PAHs, some of which are considered to have human carcinogenic potential. The association of benzo(a)pyrene and nitropyrene with diesel exhaust particulates prolongs their retention in the lungs. This increased retention suggests the need to clarify the relative importance of competing mechanisms thatdetoxify particle-associated compounds and those that serve to enhance the retention of toxicologically important compounds.

ENVS - Benzo(a)pyrene's (BaP) release to the environment is quite wide spread since it is an ubiquitous product of incomplete combustion. It is largely associated with particulate matter, soils, and sediments. Although environmental concentrations are highest near sources, its presence in places distant from primary sources indicates that it is reasonably stable in the atmosphere and capable of long distance transport. When released to air it may be subject to direct photolysis, although adsorption to particulates apparently can retard this process. It may also be removed by reaction with O3 (half-life 37 min) and NO2 (half-life 7 days), and an estimated half-life for reaction with photochemically produced hydroxyl radicals is 21.49 hr. If released to water, it will adsorb very strongly to sediments and particulate matter, bioconcentrate in aquatic organisms which can not metabolize it, but will not hydrolyze. It may be subject to significant biodegradation, and direct photolysis may be important near the surface of waters; adsorption, however, may significantly retard these two processes. Evaporation may be important with a half-life of 43 days predicted for evaporation from a river 1 m deep, flowing at 1 m/sec with a wind velocity of 3 m/sec; adsorption to sediments and particulates will limit evaporation. If released to soil it will be expected to adsorb very strongly to the soil and will not be expected to appreciably leach to the groundwater, although its presence in some samples of groundwater illustrates that it can be transported there. It will not be expected to hydrolyze or significantly evaporate from soils and surfaces. It may be subject to appreciable biodegradation in soils. Human exposure will be from inhalation of contaminated air and consumption of contaminated food and water. Especially high exposure will occur through the smoking of cigarettes and the ingestion of certain foods (eg smoked and charcoal broiled meats and fish). (SRC) [CITATION]

FATE - TERRESTRIAL FATE: If benzo(a)pyrene is released to soil it will be expected to adsorb very strongly and will not be expected to leach to the groundwater; however, its presence in some groundwater samples indicates that it can be transported there by some mechanism. It will not hydrolyze, and evaporation from soils and surfaces is not expected to be significant. Biodegradation tests in soils have resulted in a wide range of reported half-lives: 2 days to 1.9 yr; based on these values and the apparent lack of a significant competing fate process, biodegradation may be an important process in soils. (SRC) [CITATION]

FATE - AQUATIC FATE: If released to water, benzo(a)pyrene (BaP) will be expected to adsorb very

strongly to sediments and particulate matter. It will not hydrolyze but will be expected to bioconcentrate in aquatic organisms that can not metabolize it. It has been shown to be susceptible to significant metabolism by microorganisms in some natural waters without use as carbon or energy source, but in most waters and in sediments it has been shown to be stable towards biodegradation. BaP will be expected to undergo significant photodegradation near the surface of waters. Evaporation may be significant with a predicted half-life of 43 days for volatilization from a river 1 m deep, flowing at 1 m/sec with a wind velocity of 3 m/sec. Adsorption to sediments and particulates may significantly retard biodegradation, photodegradation, and evaporation. (SRC) [CITATION]

FATE - ATMOSPHERIC FATE: Benzo(a)pyrene (BaP) released to the atmosphere will likely be associated with particulate matter and may be subject to moderately long transport, depending mainly on the particle size distribution and climactic conditions which will determine the rates of wet and dry deposition. Its presence in areas remote from primary sources demonstrates the potential for this long range transport as well as BaP's considerable stability in the air. A half-life of 1.4 years has been reported for removal of BaP from the gas phase by rainout and has a lifetime of 7.9 days for removal by aerosol particles(1). It may be subject to direct photodegradation but evidence suggests that this process is retarded by the material being in the adsorbed state. Half-life for reaction of a thin film of BaP with 0.19 ppm O3 is 37 min and for reaction of adsorbed BaP with NO2 is 7 days. The estimated half-life for reaction with photochemically produced hydroxyl radicals is 21.49 hr. [(1) Cupitt LT; Fate of Toxic Hazards in the Air Environment USEPA-600/3-80-084 (1980)]

BIOC - Reported BCF: Oysters (Crassostrea virginica), 3000(1); Rainbow trout, 920(2); Bluegills, 2,657(3); Daphnia magna, 1000(5); Daphnia pulex, 13,000(6). The presence of humic acid in solution have been shown to decr bioconcentration: eg, Daphnia magna, BCF 1716 (humic material (hm) 0.3 ppm dissolved organic carbon (DOC), BCF 979 (hm 1.5 ppm DOC), BCF 838 (hm 5.7 ppm DOC)(4). [(1) Verschueren K; Handbook of Environmental Data on Organic Chemicals. 2nd ed Von Nostrand Reinhold NY p.27 (1983) (2) Spehar RL et al; J Water Pollut Control Fed 52: 1703-74 (1980) (3) McCarthy JF, Jimenez BD; Environ Sci Technol 19: 1072-6 (1985) (4) Leversee GJ et al; Can J Fish Aquat Sci 40: 63-9 (1983) (5) McCarthy JF; Arch Environ Contam Toxicol 12: 559-68 (1983) (6) Biddinger GR, Gloss SP; Res Rev 91: 103-45 (1984) (7) Santodonato J et al; Health and Economic Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL: Pathotox Pub Inc. p.160-7 (1981)]

BIOC - Gillichthys mirabilis (mudsucker) exposed to benzo(a)pyrene /concn not specified/ for 96 hr exhibited a bioconcentration factor of 0.048; Oligocottus maculosus (tidepool sculpin) exposed to benzo(a)pyrene /concn not specified/ for 1 hr exhibited a bioconcentration factor of 0.13; Citharichthys stigmacus (sand dab) exposed to benzo(a)pyrene /concn not specified/ for 1 hr exhibited a bioconcentration factor of 0.02. /Edible tissue/ [Lee RG et al; Mar Biol 17: 201 (1972) as cited in USEPA; Ambient Water Quality Criteria Doc: Chloroalkyl Ethers p.B-4 (1980) EPA 440/5-80-030]

BIOC - ... Some marine organisms have no detectable aryl hydrocarbons hydroxylase enzyme systems, namely: phytoplankton, certain zooplankton, mussels (Mytilus edulis), scallops (Placopecten sp), and snails (Litternia littorea). ... Those organisms which lack a metabolic detoxification enzyme system, tend to accumulate polycyclic aromatic hydrocarbons. /Polycyclic aromatic hydrocarbons/ [Malins DC; Ann NY Acad Sci 298: 482-496 (1977) as cited in: Health and Welfare Canada; Polycyclic Aromatic Hydrocarbons p.37 (1979) Report No. 80-EHD-50]

BIOC - POLYCYCLIC AROMATIC HYDROCARBONS (PAH) WERE ANALYZED IN SURFACIAL SEDIMENTS & BENTHIC ORGANISMS IN SOUTHEASTERN LAKE ERIE, NEAR A LARGE COAL-FIRED POWER PLANT. SEDIMENT CONCN (530-770 PPB PAH) WERE RELATIVELY HOMOGENOUS THROUGHOUT MOST OF THE 150 SQUARE KM AREA, ALTHOUGH RIVER & NEARSHORE CONCENTRATIONS REACHED 4 PPM. OLIGOCHAETE WORMS DID NOT BIOCONCENTRATE (ON WET WT BASIS) ANY OF THE PAH. CHIRONOMIDE MIDGES COLLECTED 1 KM OFFSHORE EXHIBITED BIOCONCENTRATION OF 5 PAH ONE OF WHICH WAS PYRENE. FURTHER OFFSHORE, THESE APPARENT

BIOCONCENTRATIONS DISAPPEARED, WITH MIDGES AT NEAR EQUILIBRIUM WITH SEDIMENTS. /PAH/ [EADIE BJ ET AL; CHEMOSPHERE 11 (2): 185-92 (1982)]

KOC - Reported Koc: 3,950,00-5,830,000 experimental(1,2). Koc for binding to dissolved organic carbon in 3 natural waters, 18,000-52,000; Koc for binding to Aldrich humates, 890,000(3). [(1) Sims RC, Overcash MR; Res Rev 88: 1-68 (1983) (2) Smith JH et al; Environmental Pathways of Selected Chemicals in Freshwater Systems: Part II. Laboratory Studies USEPA-600/7-78-074 (1978) (3) Landrum PF et al; Environ Sci Technol 18: 187-92 (1984)]

VWS - The reported estimated theoretical maximum half-life for volatilization from a model river 1 m deep, flowing at 1 m/sec with a wind velocity of 4 m/sec is 18 days; physical factors, such as adsorption, which will slow volatilization, were not considered in this estimation and it was concluded that vaporization will be insignificant under all conditions(1). Predicted half-life for volatilization from a model river 1 m deep, flowing at 1 m/sec with a wind velocity of 3 m/sec is 43 days(3,SRC), qualified by adsorption. Volatilization half-life (hr) predicted by the one-compartment model: 140 (river), 350 (eutrophic pond), 700 (eutrophic lake), 700 (oligotrophic lake); measured half-life in a rapidly stirred aqueous solution was 22 hr; benzo(a)pyrene/O2 reaeration rate ratio, 0.0036(2). [(1) Southworth GR et al; Bull Environ Contam Toxicol 21: 507-14 (1979) (2) Smith JH et al; Environmental Pathways of Selected Chemicals in Freshwater Systems: Part II. Laboratory Studies USEPA-600/7-78-074 (1978) (3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Environmental Behavior of Organic Compounds. McGraw-Hill NY p 15-1 to 15-34 (1982)]

RTEX - Human exposure occurs mainly through the smoking of tobacco, inhalation of polluted air, and by ingestion of water contaminated by combustion effluents or ingestion of food contaminated by smoking, broiling or exposure to combustion products(1). [(1) IARC; Polynuclear aromatic compounds, Part 1. Chemicals, environmental and experimental data. 32: 211-24 (1983)]

RTEX - ... Finished waters from various treatment sites are transported to consumers through a variety of pipelines. PAH's /polynuclear aromatic hydrocarbons/ leach from the tar or asphalt linings of these pipes ... resulting in increased concn of these cmpd in water reaching the consumers. ... Cement-lined pipes produce lower PAH concn, possibly because PAH's are adsorbed from water. [NRC. DRINKING WATER & HEALTH VOL4 1982 , p. 256]

RTEX - Exposure to benzo(a)pyrene will be widespread due to its ubiquitous presence in the environment. NIOSH (NOHS survey 1972-1974) has statistically estimated that 32 workers are exposed to benzo(a)pyrene in the USA(2). Also NIOSH (NOES Survey 1981-1983) has statistically estimated that 826 workers are exposed to benzo(a)pyrene in the USA(1). Occupational exposure (ug/day): coke oven, 70-180, coal tar pitch worker, 750, airline pilots, 0.93-1.38, restaurant employee, 0.8; smoking 1 pack of cigarettes, 0.4, person living near expressway, 0.02, commuter on expressway 2 hr/day, 0.04(3). Coke plant, particulates, 8-135 mg/cu m, gaseous, 0-1.5 mg/cu m(4). [(1) NIOSH; National Occupational Exposure Survey (NOES) Sept.9 (1985) (2) RTECS; Current Awareness File (1984) (3) Santodonato J et al; Health and Ecological Assessment of Polynuclear Aromatic Hydrocarbons. Lee SD, Grant L, eds Park Forest South, IL: Pathotox Pub Inc. p 131-36 (1981) (4) Bjorseth A et al; Scand J Work Environ Health 4: 224-36 (1978)]

RTEX - Influence of occupational ... factors upon benzo(a)pyrene exposure: Coke oven workers: Top side workers: 180 ug/day, side and bench exposure. 70 ug/day; Coal tar pitch worker: 750 ug/day; Airplane pilots: Transatlantic flights: 0.93 ug/day, domestic cross country: 1.38 ug/day; Employee in restraunt: 0.8 ug/day; Person living near expressway 24 hr/day (adverse meterology). 0.02 ug/day; Commuter on an expressway 2 hr/day (adverse meterology): 0.04 ug/day. [USEPA; Health Assessment Document for Polycyclic Organic Matter p.5-2 (1979) EPA-600/9-79-008]

RTEX - POLYCYCLIC AROMATIC HYDROCARBON (PAH) CONTENT IN AIR OF 10 FERROUS & NONFERROUS FOUNDRIES WAS STUDIED. CERTAIN OCCUPATIONS REPORTED TO HAVE A HIGH RISK OF LUNG CANCER, SUCH AS MOLDERS, CASTERS & CRANE MEN, WERE ASSOCIATED WITH HIGH CONCENTRATIONS OF PAH EXPRESSED AS PERCENTAGE OF TOTAL SUSPENDED PARTICULATE. THIS RESULT WAS NOT STATISTICALLY SIGNIFICANT. [VERMA DK ET AL; ANN OCCUP HYG 25 (1): 17-26 (1982)]

NITROBENZENE
SY - NB
SY - Oil of mirbane
SY - Essence of mirbane
SY - Nitrobenzol
MF - C6-H5-N-O2
USE - MANUFACTURE OF ANILINE; SOLVENT FOR CELLULOSE ETHERS; MODIFYING ESTERIFICATION OF CELLULOSE ACETATE; INGREDIENT OF METAL POLISHES [SAX. HAWLEY'S CONDENSED CHEM DICT 11TH ED 1987 , p. 826]
USE - IN SOAPS, SHOE POLISHES, FOR REFINING LUBRICATING OILS, MFR OF PYROXYLIN CMPD [BUDAVARI. MERCK INDEX 11TH ED 1989 , p. 1042]
USE - A PRESERVATIVE IN SPRAY PAINTS, CONSTITUENT OF FLOOR POLISHES, SUBSTITUTE FOR ALMOND ESSENCE, & IN PERFUME INDUST [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 298]
USE - USED TO PRODUCE BENZIDINE AND METANILIC ACID AS WELL AS DINITROBENZENE AND DYES SUCH AS NIGROSINES AND MAGENTA [CHEMICAL PRODUCTS SYNOPSIS: NITROBENZENE, 1984]
USE - USED IN THE PRODUCTION OF ISOCYANATES, PESTICIDES, RUBBER CHEMICALS AND PHARMACEUTICALS (ACETOMINOPHEN) [CHEMICAL PRODUCTS SYNOPSIS: NITROBENZENE, 1984]
CPAT - CHEMICAL PROFILE: Nitrobenzene. Aniline, 98%; others, including N-acetyl-p-amino-phenol (acetaminophen), 2%. [Kavaler AR; Chemical Marketing Reporter 232 (5): 50 (1987)]
COFO - GREENISH-YELLOW CRYSTALS OR YELLOW, OILY LIQUID [SAX. HAWLEY'S CONDENSED CHEM DICT 11TH ED 1987 , p. 826]
ODOR - ODOR OF VOLATILE OIL ALMOND [BUDAVARI. MERCK INDEX 11TH ED 1989 , p. 1042]
ODOR - Nitrobenzene has a pungent, shoe-polish smell. [Ruth JH; Am Ind Hyg Assoc J 47: A-142-51 (1986)]
BP - 210.8 DEG C [LIDE. CRC HANDBK CHEM & PHYSICS 72TH ED 1991-1992 3-85]
MP - 5.7 DEG C [LIDE. CRC HANDBK CHEM & PHYSICS 72TH ED 1991-1992 3-85]
MW - 123.11 [BUDAVARI. MERCK INDEX 11TH ED 1989 , p. 1042]
DEN - 1.2037 @ 20 DEG C/4 DEG C [LIDE. CRC HANDBK CHEM & PHYSICS 72TH ED 1991-1992 3-85]
OWPC - log Kow= 1.85 [HANSCH. LOG P DATABASE 1984 , p. 66]
SOL - SOL IN ABOUT 500 PARTS WATER; FREELY SOL IN ALCOHOL, BENZENE, ETHER, OILS [BUDAVARI. MERCK INDEX 11TH ED 1989 , p. 1042]
SOL - SOL IN ACETATE [LIDE. CRC HANDBK CHEM & PHYSICS 72TH ED 1991-1992 3-85]
SOL - Water solubility: 1,780 ppm [Kenaga EE; Ecotoxicology and Environmental Safety 4: 26-38 (1980)]
VAP - 1 MM HG @ 44.4 DEG C [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 2010]
DOT - Health Hazards: Poisonous; may be fatal if inhaled, swallowed or absorbed through skin. Contact may cause burns to skin and eyes. Runoff from fire control or dilution water may give off poisonous gases and cause water pollution. Fire may produce irritating or poisonous gases. [DOT. EMERGENCY RESPONSE GUIDEBOOK 1990 G-55]
FPOT - /FIRE HAZARD IS/ MODERATE WHEN EXPOSED TO HEAT, FLAME, OR OXIDIZERS. [SAX. DANGER PROPS INDUS MATER. 6TH ED 1984 , p. 2010]
EXPL - Aromatic nitro compounds mixture with nitrobenzene ... are high explosives of high sensitivity & detonation velocities ... and are spark-detonable. [BRETHERICK. HDBK REACTIVE CHEM HAZARDS 1985 , p. 186]
SERI - Vapor ... is moderately irritating such that personnel will not usually tolerate moderate or high vapor concn. ... Liquid or solid ... causes smarting of skin & 1st degree burns on short exposure. ... [CHRIS. HAZARD CHEM DATA VOL. II 1984-5]
CAREV- CLASSIFICATION: D; not classifiable as to human carcinogenicity. BASIS FOR CLASSIFICATION: Based on no data concerning carcinogenicity in humans or animals. HUMAN CARCINOGENICITY DATA: None. ANIMAL

CARCINOGENICITY DATA: None. [U.S. Environmental Protection Agency's Integrated Risk Information System (IRIS) on Nitrobenzene (98-95-3) from the National Library of Medicine's TOXNET System, August 29, 1994]

EMCE - CLINICAL EFFECTS :

- o Nitrobenzene is toxic by all routes of exposure. The mean adult lethal oral dose has been estimated as about 1 to 5 grams. Symptoms may be delayed for up to 1 to 4 hours. Methemoglobinemia associated with headache, nausea, lethargy, depressed respiration, and cyanosis may be noted.
- o Because a bitter almond odor may be present, cyanide poisoning may be suspected. However, cyanide produces symptoms much more rapidly than nitrobenzene.

CARDIOVASCULAR	o	Tachycardia, hypotension, and cardiac arrhythmias may be noted.
RESPIRATORY	o	Respiratory failure may be noted.
NEUROLOGIC	o	Headache, dizziness, lethargy, and coma may be noted.
GASTROINTESTINAL	o	Nausea and vomiting may be noted. Urine and vomitus will have the odor of oil of bitter almonds.
HEMATOLOGIC	o	Methemoglobinemia associated with hypotension and cardiac arrhythmias may be noted.
DERMATOLOGIC	o	Cyanosis that does not respond to oxygen therapy will appear when the methemoglobin level is greater than 15%.

EMLAB- LABORATORY :

- o Plasma nitrobenzene levels are not clinically useful.
- o Determine methemoglobin levels in all cyanotic patients. Cyanosis generally occurs when the plasma methemoglobin levels exceed 15%.

EMTOX- RANGE OF TOXICITY :

- o At this time, there is insufficient data in the literature to assess the minimal toxic dose of nitrobenzene. The estimated mean lethal adult dose is about 1 to 5 grams. Children may be much more susceptible to nitrobenzene.

HTOX - IN 2 INDUSTRIAL CASES THERE WAS SLIGHT ANEMIA ... CONSIDERED TO BE OF HEMOLYTIC TYPE, WITH MARKED IRREGULARITY IN SHAPE AND SIZE OF RED CELLS & SOMETIMES AN INTENSE POLYNUCLEOSIS. [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 301]

HTOX - ... 21 INFANTS HAVE BEEN AFFECTED BY ... SKIN APPLICATION OF 'BITTER ALMOND OIL'; & ... A MIXTURE OF 2-10% NITROBENZENE AND ... COTTON SEED OIL. IN SIX CASES THE INFANTS WERE IN ... SHOCK, & SEMICOMATOSE WITH COLD EXTREMITIES AND RAPID PULSE, & OF THESE, TWO ENDED FATALLY, TERMINAL BRONCHO-PNEUMONIA HAVING DEVELOPED; THE REMAINING CASES RECOVERED, WITH ... /MINIMUM OR NO/ RESIDUAL CYANOSIS. [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 302]

HTOX - NITROBENZENE & OTHER NITRO CMPD GENERATE METHEMOGLOBIN MORE SLOWLY /THAN ANILINE, DINITROBENZENE OR NITROANILINE/, BUT CYANOSIS IS MORE PERSISTANT. ONSET OF CYANOSIS IS OFTEN FIRST NOTED AT LIPS. ... SYMPTOMS MAY BE ABSENT, ALTHOUGH, EUPHORIA, FLUSHED FACE AND HEADACHE ARE COMMON. CYANOSIS IS USUALLY DETECTABLE WHEN PROPORTION OF CONVERTED HEMOGLOBIN APPROXIMATES 15%. METHEMOGLOBIN LEVELS OF 40% MAY EXIST WITHOUT SYMPTOMS OTHER THAN A SENSE OF WELL-BEING. AT HIGHER LEVELS, ... WEAKNESS, ATAXIA, AND LIGHTHEADEDNESS OCCUR. WITH INCR CONCN OF METHEMOGLOBIN, DYSPNEA, TACHYCARDIA AND ALARMING CYANOSIS ARE NOTED. [HAMILTON. INDUS TOX 3RD ED 1974 , p. 306]

HTOX - REPEATED EXPOSURE MAY BE FOLLOWED BY LIVER IMPAIRMENT UP TO YELLOW ATROPHY, HEMOLYTIC ICTERUS AND ANEMIA OF VARYING DEGREES, WITH THE PRESENCE OF HEINZ BODIES IN THE RED CELLS. [ENCYC OCCUPAT HEALTH & SAFETY 1983 , p. 1448]

HTOX - A RARE CASE OF ACCIDENTAL POISONING BY NITROBENZENE IS DESCRIBED. THIS POISONING WAS CHARACTERIZED BY SEVERE CLINICAL PICTURE, PROTRACTED COURSE WITH RELAPSES & OCCURRENCE OF HEMATOLOGICAL & NEUROLOGICAL COMPLICATIONS. [LARENG L ET AL; EUR J TOXICOL ENVIRON HYG 7 (1): 12-6 (1974)]

HTOX - The most reliable established ocular effects are secondary to

discoloration of the blood from methemoglobinemia, & consist of brown discoloration of the vessels of the fundus & the conjunctiva. [GRANT. TOX OF THE EYE 1986 , p. 663]

HTOX - TOXIC BY ALL ROUTES INCLUDING SKIN ABSORPTION. ... SYSTEMIC EFFECTS MAY BE DELAYED A FEW HOURS. POISONING CLOSELY RESEMBLES ANILINE. MEAN LETHAL DOSE BY MOUTH PROBABLY LIES BETWEEN 1 AND 5 G. [GOSSELIN. CTCP 5TH ED. 1984 II-214]

HTOX - Highly toxic. Fatal amt may be absorbed through the skin, by inhalation, or ingestion. [NFPA. FIRE PROTECT GUIDE HAZARD MATLS 9TH ED 1986 49-67]

NTOX - ... IN WHITE RATS POISONED BY SC INJECTION OF 0.64 G/KG NITROBENZENE ... CATALASE ACTIVITY FELL CONSISTENTLY DURING A PERIOD UP TO 96 HR AFTER INITIAL DOSE TO 86.6% OF INITIAL LEVEL. ... PEROXIDASE ACTIVITY ALSO DIMINISHED. ... [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 299]

NTOX - ... IN SLOW POISONING FROM CONTINUED LOW EXPOSURES THERE MAY BE INCREASE IN NUMBER OF ERYTHROCYTES; URINE BECOMES BROWN OR DARK RED, CONTAINS BILE PIGMENTS, METHEMOGLOBIN OR HEMOGLOBIN; THERE MAY ... BE ALBUMINURIA AND POSITIVE FEHLING'S REACTION /IN RABBITS/. [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 300]

NTOX - OUTSTANDING TOXIC EFFECT ... IS ... FORMATION OF METHEMOGLOBIN, WITH RISK OF DEATH FROM RESPIRATORY FAILURE. ... [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 300]

NTOX - RESTLESSNESS, CYANOSIS OF SKIN, MUCOUS MEMBRANES; ONSET OF ... /CNS DEPRESSION/ ACCOMPANIED BY LOSS OF WEIGHT AND MARKED CYANOSIS /IN RABBITS/. METHEMOGLOBINEMIA ... /% DEPENDENT ON DOSE, IS/ GREATEST BY IP INJECTION. HEINZ BODIES /DEVELOPED/ IN 40 TO 60% OF RED CELLS. [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 301]

NTOX - IN RABBITS, GIVEN SC INJECTIONS OF 0.75 G ... HEMATOCRIT AND HEMOGLOBIN LEVELS SHOWED MARKED DECREASE ... AND TENDENCY TO SPHEROCYTOSIS; HEINZ BODIES WERE PRESENT IN ... 17 ANIMALS OUT OF 27. ... CHANGES WERE ACCOMPANIED BY ... RETICULOCYTOSIS ... [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 301]

NTOX - /IN RABBITS AFTER SC ADMIN/ BONE MARROW SHOWED CHANGES RANGING FROM HYPERPLASIA TO HYPOPLASIA AND EVEN APLASIA, WITH AN INCREASE IN MACROBLASTS AND A DECREASE IN RETICULOCYTES AND MEGAKARYOCYTES. HYPEREMIA OF ABDOMINAL CAVITY & ALL ORGANS /WERE OBSERVED/. [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 301]

NTOX - METHEMOGLOBINENIA WAS ... MAXIMUM (4%) ... 4 HR AFTER INITIAL DOSAGE FALLING GRADUALLY THEREAFTER ... SULFHEMOGLOBIN ROSE ... TO 14%. LEUCOPENIA WAS PRESENT IN SOME ANIMALS ... IN OTHERS LEUCOCYTOSIS ... WITH NEUTROPHILIA, IN MAJORITY THROMBOCYTOPENIA /DEVELOPED IN RABBITS GIVEN 0.75 G SUBCUTANEOUSLY/ [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 301]

NTOX - /CHANGES IN RABBIT ORGANS WERE/ LIVER: MACROSCOPIC APPEARANCE OF NUTMEG SIZE NODULES; MICROSCOPICALLY FATTY INFILTRATION; KIDNEYS: FATTY INFILTRATION; LUNGS: TENDENCY TO EXTRAVASATION OF BLOOD, VARYING FROM MINUTE PETECHIAE TO LARGER ECCHYMISES OR EVEN LOBULAR HEMORRHAGE IN SEVERE POISONING. [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 302]

NTOX - ... ONE RABBIT WHICH DIED AFTER RECEIVING ORALLY 200 MG/KG 30 HR LATER HAD LARGE DEPOSITS OF FAT IN TISSUES ... THIS WAS ALSO PRESENT IN GASTROINTESTINAL TRACT. [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 302]

NTOX - /NITROBENZENE WAS/ ADMIN ... IN DOSES OF 125 MG/KG/DAY SC TO PREGNANT RATS DURING PREIMPLANTATION & PLACENTATION. DELAY OF EMBRYOGENESIS, ALTERATION OF NORMAL PLACENTATION, AND ABNORMALITIES IN FETUS WERE OBSERVED. GROSS MORPHOLOGIC DEFECTS WERE SEEN IN 4 OF THE 30 FETUSES EXAMINED. [NRC. DRINKING WATER & HEALTH VOL4 1982 , p. 227]

NTOX - TESTS FOR MUTAGENIC ACTIVITY OF NITROBENZENE WERE DONE WITH RAT-LIVER S9 OR MOUSE-LIVER S9 FRACTIONS IN SALMONELLA TYPHIMURIUM STRAINS TA92, TA1535, TA100, TA1537, TA94 AND TA98. THERE WAS NO MUTAGENIC EFFECT

OBSERVED. [MIYATA R ET AL; BULL NATL INST HYG SCI (TOKYO) 0 (99): 60-5 (1981)]

NTOX - ACUTE INTOXICATION OF SC INJECTION TO YOUNG RATS 6 MO OLD AND MATURE RATS 18 MO OLD PRODUCED A HIGH MORTALITY IN THE OLDER RATS AND NONE IN THE YOUNGER ANIMALS. DEATH IN OLDER RATS WAS MAINLY DUE TO ACUTE RENAL FAILURE. [VRABIESCU N SA ET AL; AN UNIV BUCUR BIOL 30 (0): 117 (1982)]

NTOX - MALE RATS WERE GIVEN SINGLE ORAL DOSES OF NITROBENZENE (50-450 MG/KG) AND AT THE TIME OF SACRIFICE, 25 TISSUES WERE REMOVED AND EXAMINED. HEPATIC CENTROLOBULAR NECROSIS APPEARED IN RATS GIVEN VARIOUS DOSES. TESTICULAR LESIONS WERE CONFINED TO SEMINIFEROUS TUBULES AND CONSISTED OF NECROSIS OF THE PRIMARY AND SECONDARY SPERMATOCYTES WITH APPEARANCE OF MULTINUCLEATED GIANT CELLS BETWEEN 1 & 4 DAYS AFTER ADMIN OF 300 MG/KG. [BOND JA ET AL; FUNDAM APPL TOXICOL 1 (5): 389-94 (1981)]

NTOX - THE REFRACTILE HEINZ BODIES ARE OFTEN FOUND AT THE PERIPHERY OF ERYTHROCYTES, AND SOMETIMES WITHIN, AS A RESULT OF POISONING BY ... NITROBENZENE ... . [THIENES. CLIN TOX 5TH ED 1972 , p. 232]

NTOX - Toxicity threshold /as determined by/ (cell multiplication inhibition test): bacteria (Pseudomonas putida) 7 mg/l; algae (Microcystis aeruginosa) 1.9 mg/l; green algae (Scenedesmus quadricauda) 33 mg/l; protozoa (Entosiphon sulcatum) & (Uronema parduczi) 1.9 mg/l & 15 mg/l, respectively. [VERSCHUEREN.HDBK ENVIRON DATA ORG CHEM 1983 , p. 911]

NTOX - Pregnant CD (Sprague-Dawley) rats were exposed to nitrobenzene vapor at 0, 1, 10, and 40 ppm (mean analytical values of 0.0, 1.06, 9.8, and 39.4 ppm, respectively) on gestational days 6 through 15 for 6 hr/day. At sacrifice on gestation days 21, fetuses were evaluated for external, visceral, and skeletal malformations and variations. Maternal toxicity was observed: weight gain was reduced during exposure (gestation days 6-9 and 6-15) to 40 ppm, with full recovery by gestation days 21, and absolute and relative spleen weights were increased at 10 and 40 ppm. There was no effect of treatment on maternal liver, kidney, or gravid uterine weights, on pre- or postimplantation loss including resorptions or dead fetuses, on sex ratio of live fetuses, or on fetal body weights (male, female, or total) per litter. There were also no treatment-related effects on the incidence of fetal malformations or variations. In summary, during organogenesis in CD rats, there was no developmental toxicity (including teratogenicity) associated with exposure to nitrobenzene concentrations that produced some maternal toxicity (10 and 40 ppm) or that produced no observable maternal toxicity (1 ppm). [Tyl RW et al; Fund Appl Toxicol 8 (4): 482-92 (1987)]

NTOX - A two-generation reproduction study was performed by exposure of Sprague-Dawley CD rats to concentrations of 0, 1, 10, or 40 ppm of nitrobenzene (NB) vapor. No NB related effects on reproduction were observed at 1 or 10 ppm. At 40 ppm, a decrease in the fertility index of the F(0) and F(1) generations occurred, which was associated with alterations in the male reproductive organs. Specifically, weights of testes and epididymides were reduced and seminiferous tubule atrophy, spermatocyte degeneration, and the presence of giant syncytial spermatocytes were observed. The only significant finding in the litters derived from rats exposed to 40 ppm was an approximate 12% decrease in the mean body weight of F(1) pups on postnatal day 21. Survival indices were unaltered. To examine the reversibility of this selective effect on male gonads, the F(1) males from the 40 ppm group were allowed a 9 week nonexposure period and mated to naive females. An almost five-fold increase in the fertility index was observed, indicating at least partial functional reversibility upon removal from NB exposure. The numbers of giant syncytial spermatocytes and degenerated spermatocytes were greatly reduced. The results of this study support the selection of 10 ppm of nitrobenzene as the no-observable-effect level for reproduction and fertility effects in rats. [Dodd DE et al; Fund Appl Toxicol 8 (4): 493-505 (1987)]

NTOX - The neurotoxic effects of nitrobenzene were studied morphologically and biochemically in rats. Male F344 rats received a single oral dose of 550 mg/kg radiolabeled nitrobenzene. ... Nitrobenzene induced petechial hemmorrhages in the brain stem and cerebellum and malacia in the cerebellum and cerebellar peduncles within 48 hr after administration. ... Analysis of the distribution of radiolabeled nitrobenzene and HPLC analysis indicated that a very small proportion of the administered dose passed the blood brain barrier. In the brain it

was present as the parent compound, accumulated in higher concentrations in gray matter than white matter, but there was no evidence of increased accumulation of nitrobenzene in the location in which malacia occurred. [Morgan KT et al; Neurotox 6 (1): 105-116 (1985)]

NTOX - NITROBENZENE INHIBITED THE OXIDATION OF TYPE II SUBSTRATES (ANILINE AND ZOXAZOLAMINE) BY CYTOCHROME P450 DEPENDENT ENZYMES IN LIVER MICROSOMES, BUT DID NOT EFFECT THE OXIDATIVE METAB OF TYPE I CMPD (HEXOBARBITAL AND AMINOPYRENE). [STERNSON LA, GAMMANS RE; J MED CHEM 19 (1): 174-7 (1976)]

NTOX - Nineteen nitro compounds, including nitrobenzene, were evaluated for mutagenicity, using a modification of the standard Salmonella typhimurium mutagenicity assay. A preincubation protocol was used which incorporated flavin mononucleotide to facilitate nitro reduction. Nitrobenzene was found to elicit a negative response in the modified preincubation assay with flavin mononucleotide despite extensive nitro reduction that occurred in the preincubation with flavin mononucleotide. [Dellarco VL, Prival MJ; Environ Mol Mutagen 13 (2): 116-27 (1989)]

NTOX - 21 day Daphnia reproduction tests were conducted in line with the provisional procedure proposed by the Federal Environmental Agency (Umweltbundesamt, FRG), as of Jan 1, 1984. Groups of 20, 24 hr old Daphnia magna were exposed to 1.6 to 200 mg/l nitrobenzene in semi-static test vessels. Parent animals in the test and control vessels had to be pipetted 3 times/wk in freshly prepared test and control media at the corresponding concn level. The no observed effect concn (NOEC) was determined from the parameters of mortality of the parent animals, reproduction rate and appearance of the first offspring during the test period. In preliminary acute Daphnia tests, the 24 hr EC50 was 60 mg/l for nitrobenzene, the EC0 was 19 mg/l. The nominal 21 day no observed effect concn was 13 mg/l, with the most sensitive parameter being the reproductive rate. [Kuhn R et al; Water Res 23 (4): 501-10 (1989)]

NTOX - Purified Sertoli cell cultures were used to determine whether the effects of nitrobenzene in cocultures are the result of a direct effect on the Sertoli cell. Testicular cell cultures were prepared from Alpk:Ap-rats. The exposure of Sertoli cells to nitrobenzene resulted in Sertoli cell vacuolation, the exfoliation of germ cells, and dose dependent increases in lactate and pyruvate secretion in the presence or absence of germ cells. Following exposure to concentrations greater than 0.0005 molar nitrobenzene resulted in an increase in the in-vitro secretion of lactate and pyruvate by Sertoli cells in a dose dependent manner, both in the presence and absence of follicle stimulating hormone (FSH) and in the presence and absence of germ cells. Following exposure of cocultures or Sertoli cell cultures to nitrobenzene, basal inhibin secretion exhibited a reproducible alteration in response in a biphasic pattern and was taken by the authors as a confirmation of germ cell modulation of Sertoli cell inhibin secretion. The finding that these effects of nitrobenzene on inhibin secretion were not evident in follicle stimulating hormone stimulated cultures does not support the use of inhibin as a specific indicator of the toxic actions of nitrobenzene on the Sertoli cells. The authors state this to be the first study to evaluate the potential of inhibin secretion by Sertoli cells as a marker of toxicant action. [Allenby G et al; Fundam Appl Toxicol 14 (2): 364-75 (1990)]

NTXV - ... /CNS DEPRESSANT/ DOSE, BY IP INJECTION, FOR RATS, 0.18 G/KG. ... [BROWNING. TOX & METAB INDUS SOLV 1965 , p. 300]

NTXV - LD50 Rat oral 640 mg/kg [BUDAVARI. MERCK INDEX 11TH ED 1989 , p. 1042]

ETXV - TLm Fish (Vairon) 90-100 mg/l/6 hr in hard water (flow-through bioassay) [VERSCHUEREN.HDBK ENVIRON DATA ORG CHEM 1983 , p. 912]

ETXV - LC50 Daphnia magna (cladoceran) 27,000 ug/l/48 hr in a static unmeasured bioassay. /From table/ [USEPA; In-Depth Studies on Health and Environmental Impacts of Selected Water Pollutants (1978) EPA Contract No 68-01-4646, as cited in USEPA; Ambient Water Quality Criteria Doc: Nitrobenzene p.B-3 (1980) EPA 440/5-80-061]

ETXV - LC50 Lepomis macrochirus (bluegill) 42,600 ug/l/48 hr in a static unmeasured bioassay. /From table/ [USEPA; In-depth studies on health and environmental impacts of selected water pollutants. (1978) EPA Contract No. 68-01-4646 as cited in USEPA; Ambient Water Quality Criteria Doc: Chlorinated Ethanes p.B-3 (1980) EPA 440/5-80-0269]

ETXV - LC50 Pimephales promelas (fathead minnow) 163 (151-175) mg/l 24 hr, wt 160 mg,

flow-through bioassay, dissolved oxygen 7.4 (4.6-8.8) mg/l, water hardness 44.9 (42.4-46.6) mg/l as CaCO3, pH 6.9-7.7, alkalinity 42.9 (39.6-61.4) mg/l CaCO3, temp: 26.4 +/- 1.4 deg C, Purity 99% [Holcombe GW et al; Environ Pollut 35 (Series A): 367-81 (1984)]

ETXV - LC50 Pimephales promelas (fathead minnow) 117 (105-130) mg/l 96 hr, wt 160 mg, flow-through bioassay, dissolved oxygen 7.4 (4.6-8.8) mg/l, water hardness 44.9 (42.4-46.6) mg/l as CaCO3, pH 6.9-7.7, alkalinity 42.9 (39.6-61.4) mg/l CaCO3, temp: 26.4 +/- 1.4 deg C, Purity 99% [Holcombe GW et al; Environ Pollut 35 (Series A): 367-81 (1984)]

ADE - NITROBENZENE IS HIGHLY FAT-SOLUBLE AND CAN BE ABSORBED THROUGH HUMAN SKIN @ RATES AS HIGH AS 2 MG/SQ M/HR. MEDICAL LITERATURE CONTAINS MANY REPORTS OF POISONING FROM ABSORPTION OF NITROBENZENE IN SHOE DYES & LAUNDRY MARKING INK. ... CYANOSIS & POISONING OF NEWBORN WHO CAME INTO CONTACT WITH DIAPERS OR PADS CONTAINING MARKING INK WERE ALSO VERY COMMON. THIS GENERALLY OCCURRED WHEN DIAPERS OR PADS WERE FRESHLY STAMPED BY HOSPITAL LAUNDRY ... TOXICITY WAS OFTEN SEVERE IN PREMATURE INFANTS WHO WERE IN AN INCUBATOR & SURROUNDED BY FUMES ... & DYE IN CLOTH. GENERAL ABSORPTION ... IS CAUSE OF MANY OF CHRONIC & ACUTE TOXIC EFFECTS OBSERVED IN ... WORKERS. AMT OF CUTANEOUS ABSORPTION IS FUNCTION OF AMBIENT CONCN ... CLOTHING WORN, & RELATIVE HUMIDITY, WHICH INCR ABSORPTION AS IT BECOMES HIGHER. A WORKER EXPOSED TO TLV OF 5 MG/CU M COULD ABSORB UP TO A TOTAL OF 33 MG/DAY, APPROX 9 MG OF WHICH IS ABSORBED CUTANEOUSLY. [NRC. DRINKING WATER & HEALTH VOL4 1982 , p. 225]

ENVS - Nitrobenzene is produced in large quantities and may be released to the environment in emissions and wastewater during its production and use. Since 98% of nitrobenzene is used captively to produce aniline in 5 regions of the country, industrial releases will be fairly localized. Nitrobenzene is also produced by the photochemical reaction of benzene with oxides of nitrogen. This is a general source since benzene is found in petroleum products. It is difficult to estimate the ambient concentrations of nitrobenzene from this source because available air monitoring data are for areas of the country near production facilities. If released on land, nitrobenzene would leach into the soil and probably biodegrade within a few months. If released in wastewater, it should biodegrade (two experimimental values for half-lives are 1 and 3.8 days). Some volatilization would be expected, but adsorption to sediment and bioconcentration in aquatic organism should not be significant. In the atmosphere, nitrobenzene will degrade primarily by photolysis (38% degradation in 5 hr). Human exposure will be primarily occupational via inhalation of the vapor or dermal contact with the vapor or liquid. (SRC) [CITATION]

FATE - TERRESTRIAL FATE: Nitrobenzene is moderately adsorbed to soil and should leach into the ground if released on land and probably biodegrade within a few months. Nitrobenzene was completely removed from Rhine River water during soil filtration (bank or dune infiltration)(1). Generally bank filtration takes 1-12 months and dune filtration 2-3 months(1) and the soil microorganism would be well acclimated. In another study, only 60% removal was obtained during infiltration through dunes consisting of fine-grained sand mixed with clay and lens-shaped peat layers(2). [(1) Zoeteman BCJ et al; Chemosphere 9: 231-49 (1980) (2) Piet GJ et al; Proc Internat Symp The Netherlands Mar 23-27, 1981 Van Duijvenboden W et al eds. Studies in Environ Sci 17: 557-64 (1981)]

FATE - AQUATIC FATE: The half-life of nitrobenzene in the Rhine River in The Netherlands was estimated to be 1 day by measuring the concn reduction between sampling points(1). In model waste stabilization ponds that were continuously fed with a synthetic waste feedstock and detained for 12 days, 89.5% of the added nitrobenzene was degraded, 4.9% volatilized, 2.3% adsorbed to sediment, 2.3% was lost in effluent, and 1% remained in the water column(2). The biodegradation half-life in the pond was 3.8 days(SRC). [(1) Zoeteman BCJ et al; Chemosphere 9: 231-49 (1980) (2) Davis EM et al; Partitioning Of Selected Organic Pollutants In Aquatic Ecosystems pp.176-84 in Biodeterioration 5 Oxley TA & Barry S eds (1983)]

FATE - ATMOSPHERIC FATE: Nitrobenzene will degrade in the atmosphere primarily by photolysis (38% degradation in 5 hr in laboratory tests). The rate of reaction with photochemically

produced hydroxyl radicals and ozone is relatively low. Results of modeling studies and field experiments suggest that wet deposition will have little effect on nitrobenzene loss in plumes within kilometers of a source(1). [(1) Dana MT et al; Hazardous Air Pollutants EPA-600/3-84-113 (NTIS PB 85 138-626) Richland, WA: USEPA pp.106 (1985)]

ABIO - Nitrobenzene absorbs UV light to about 400 nm(1) and can therefore undergo direct photolysis. In organic solvents containing abstractable H-atoms it undergoes photoreduction when irradiated with UV light(5,6). The presence of oxygen does not appear to affect the reaction rates(5,6). In one study using a petroleum solvent and light > 290 nm, 26% degradation occurred in 5 hr(6). Azobenzene and aniline were the main products formed(6). In near-surface pure water at 40 deg N latitude, the average annual photolytic half-life is 133 days(8). This long half-life is due to low quantum yield and sunlight adsorption rate(2,8). The photolysis of humic substances in natural water gives rise to hydrated electrons that can reduce organic compounds(3). The calculated half-life of nitrobenzene in a eutrophic Swiss lake due to this reaction is 22 days(3). In the Aucilla River in northern Florida, humic substances accelerated the photolysis rate by a factor of 1.4(8). Nitrate ions in water can promote the photochemical oxidation of trace organic substances through the production of hydroxyl radicals(4). In a clear, shallow body of water, rich in nitrate (14 mg nitrate-N/l), the half-life of nitrobenzene exposed to midsummer, midday sunlight is 11 hr(4,7,SRC). Hydrolysis is not an important environmental process(1). [(1) Mabey WR et al; Aquatic Fate Process Data For Organic Priority Pollutants USEPA-440/4-81-014 (1981) (2) Wojtczak J, Maciejewski A; Poznan Tow Przyj Nauk, Pr Kom Mat-Przyr, Pr Chem 13: 117-27 (1972) (3) Zepp RG et al; Environ Sci Technol 21: 485-90 (1987) (4) Zepp RG et al; Environ Sci Technol 21: 443-50 (1987) (5) Sandus O, Slagg N; Reactions Of Aromatic Nitrocompounds I NTIS AD 753 923 Dover, NJ: Picatinny Arsenal Tech Rep 4385 (1972) (6) Barltrop JA, Bunce NJ; J Chem Soc Section C 1968: 1467-74 (1968) (7) Dorfman LM, Adams GE; Reactivity of The Hydroxyl Radical In Aqueous Solution NSRD-NBS-46 (NTIS COM-73-50623) (1973) (8) Simmons MS, Zepp RG; Water Res 20: 899-904 (1986)]

BIOC - The log BCF of nitrobenzene in golden orfe (Leuciscus idus melanotus) was <1.0 in a 3 day static test(1). In a 28 day test using fathead minnows, the log BCF was 1.18(2). Another investigator obtained a log BCF of 0.78 in fish (Poecilia reticulata)(4) and the bioconcentration test of the Japanese Ministry of International Trade and Industry report a log BCF of < 1(5). In green algae (Chlorella fusca), a log BCF of 1.38 was obtained(1). No biomagnification of nitrobenzene was observed in an aquatic ecosystem containing algae, daphnia magna, mosquito larvae, snails, and mosquito fish(3). [(1) Freitag D et al; Ecotox Environ Safety 6: 60-81 (1982) (2) Veith GD et al; J Fish Res Board Can 36: 1040-8 (1979) (3) Lu PY, Metcalf RL; Environ Health Perspect 10: 269-84 (1975) (4) Canton JH et al; Regul Toxicol Pharmacol 5: 123-31 (1985) (5) Kawasaki M; Ecotoxic Environ Safety 4: 444-54 (1980)]

KOC - The leachability of nitrobenzene was studied in three typical Norwegian soils, one which was sandy with a low organic content, and two organic soils(2). The resulting Koc and retardation factor for the sandy soil was 30.6 and 1.27, while for the two organic soils the Koc values were 42.8 and 69.6 and the retardation factors 3.36 and 5.52(2). Koc values for two Danish subsoils were 170 and 370(3). When a mixture of pollutants, including nitrobenzene, in spring water was added to a column of Lincoln fine sand over a 45 day period, the retardation factor of nitrobenzene was 1.9(1). The Koc calculated from this experiment was 200(1). For clay, 15-31 mg of nitrobenzene were retained by 1 g of sodium montmorillonite in batch experiments(4). [(1) Wilson JT et al; J Environ Qual 10: 501-6 (1981) (2) Seip HM et al; Sci Total Environ 50: 87-101 (1986) (3) Loekke H; Water Air Soil Pollut 22: 373-87 (1984) (4) Wolfe TA et al; J Water Pollut Control Fed 58: 68-76 (1986)]

WATC - DRINKING WATER: Nitrobenzene was detected, but not quantified, in finished water from the Carrollton Water Plant in Louisiana(1) and in drinking water in Cincinnati(3). In a survey of 14 treated drinking water supplies of varied sources in England, nitrobenzene was detected in one supply which came from an upland reservoir(2). [(1) USEPA; Industrial Pollution of the Lower Mississippi River in Louisiana. Technical Report. Dallas, TX: USEPA (1972) (2) Fielding M et al; Organic Micropollutants In Drinking Water TR-159 Medmenham, Eng Water Res Cent 49 pp. (1981) (3) Abrams EF et al; Springfield, VA: Versar Inc (1975)]

WATC - SURFACE WATER: Of the 836 stations reporting analysis for nitrobenzene in ambient water in EPA's STORET database, 0.4% contained detectable levels of the chemical(5). No nitrobenzene was reported in the Buffalo and Cuyahoga Rivers in the Lake Erie Basin or the St. Joseph River in the Lake Michigan basin(7). Average and maximum levels of nitrobenzene were 1.7 and 13.8 ppb in the Waal River and < 0.1 and 0.3 ppb in the Maas River, both in The Netherlands(1). Rhine River water in The Netherlands contained 0.5 ppb(3). A 2-wk composite sample taken from the Rhine River near Dusseldorf in 1984 contained a mean nitrobenzene concn of 0.42 ppb(6). Japanese river water and seawater contained 0.16-0.99 ppb of nitrobenzene(4) and it was detected, but not quantified in seawater in the Kitakyushi area of Japan(2). [(1) Meijers AP, Vanderleer RC; Water Res 10: 597-604 (1976) (2) Akiyama T et al; J UOEH 2: 285-300 (1980) (3) Zoeteman BCJ et al; Chemosphere 9: 231-49 (1980) (4) Sugiyama H et al; Eisei Kagaku 24: 11-8 (1978) (5) Staples CA et al; Environ Toxicol Chem 4: 131-42 (1985) (6) Sontheimer H et al; Sci Tot Env 47: 27-44 (1985) (7) Great Lakes Water Quality Board; An Inventory Of Chemical Subtances Identified In The Great Lakes Ecosystem Vol 1. Windsor Ontario, Canada p.195 (1983)]

SEDS - Nitrobenzene was detected at a concn of 8 ppm in soil along the Buffalo River in Buffalo, NY but not detected in three samples of bottom sediment from the river(1). None of the 349 stations reporting analysis for nitrobenzene in sediment in EPA's STORET database contained detectable levels of the chemical(2). [(1) Nelson CR, Hites RA; Environ Sci Technol 14: 1147-9 (1980) (2) Staples CA et al; Environ Toxicol Chem 4: 131-42 (1985)]

FISH - None of the 122 stations reporting analysis for nitrobenzene in fish in EPA's STORET database contained detectable levels of the chemical(1). [(1) Staples CA et al; Environ Toxicol Chem 4: 131-42 (1985)]

IDLH - 200 ppm [NIOSH. NIOSH POCKET GUIDE CHEM HAZ 1990 , p. 162]

OSHA - 8 hr Time-Weighted avg: 1 ppm (5 mg/cu m). Skin absorption designation. [29 CFR 1910.1000 (7/1/90)]

NREC - 10 hr Time-Weighted avg: 1 ppm (5 mg/cu m, skin). [NIOSH. NIOSH POCKET GUIDE CHEM HAZ 1990 , p. 162]

TLV - 8 hr Time Weighted Avg (TWA) 1 ppm, 5.0 mg/cu m, skin (1986) [ACGIH. TLV'S CHEM SUBSTS & PHYSICAL AGENTS & BIOLOGICAL EXP Indices 1994-1995 , p. 28]

4-CHLOROANILINE

SY - 1-AMINO-4-CHLOROBENZENE
SY - 4-CHLORANILIN (CZECH)
SY - 4-CHLORO-1-AMINOBENZENE
SY - 4-CHLOROBENZAMINE
SY - 4-CHLOROBENZENAMINE
SY - 4-CHLOROPHENYLAMINE
SY - ANILINE, 4-CHLORO-
SY - ANILINE, P-CHLORO-
SY - BENZENAMINE, 4-CHLORO-
SY - NCI-C02039
SY - P-AMINOCHLOROBENZENE
SY - P-CA
SY - P-CHLORANILINE
SY - P-CHLOROANILINE
SY - P-CHLOROPHENYLAMINE
USE - DYE INT, PHARMACEUTICALS, AGRICULTURAL CHEMICALS [SAX. HAWLEY'S CONDENSED CHEM DICT 11TH ED 1987 , p. 262]
USE - CHEM INT FOR DYES, EG, VAT RED 32 [SRI]
USE - CHEM INT FOR AZOIC COUPLING AGENTS 5 & 10 [SRI]
USE - CHEM INT FOR UREA HERBICIDES, EG, MONURON [SRI]
USE - CHEM INT FOR PIGMENTS, EG, PIGMENT GREEN 10 [SRI]
USE - Reacts with anhydrous hydrogen chloride and phosgene at 70-75 deg C in dioxane to produce p-chlorophenyl isocyanate, an intermediate used for the production of urea herbicides. [KIRK-OTHMER. ENCYC CHEM TECH 3RD ED 1978-PRESENT V12 319]
BP - 232 DEG C @ 760 MM HG [WEAST. HDBK CHEM & PHYS 67TH ED 1986-87 C-68]
MP - 72.5 DEG C [MERCK INDEX. 10TH ED 1983 , p. 297]
SOL - SOL IN HOT WATER; FREELY SOL IN ALCOHOL, ETHER, ACETONE, CARBON DISULFIDE [MERCK INDEX. 10TH ED 1983 , p. 297]
SOL - SOL IN ORG SOLVENTS [WEAST. HDBK CHEM & PHYS 60TH ED 1979 C-113]
SOL - Solubility in water: 3.9 g/l. [Kilzer L et al; Chemosphere 8: 751-61 (1979)]
o EMCE - CLINICAL EFFECTS :

Aniline is a skin and eye irritant and a mild dermal sensitizer. It is rapidly absorbed by all routes of exposure and induces methemoglobinemia. Symptoms of methemoglobinemia include blue skin and CNS signs of oxygen insufficiency such as headache, dizziness, weakness, lethargy, loss of coordination, coma and death. Typically a Heinz-body hemolytic crisis may follow the methemoglobinemia by 2 to 7 days. Heart, liver and kidney effects may be secondary to hemolysis.

EMTOX- RANGE OF TOXICITY :

As little as 1 gram of aniline has been fatal in a human. The mean lethal dose of aniline for humans has been estimated to be in the range of 15 to 30 grams.

HTOX - Symptomatology: 1. Lips, tongue and mucous membranes navy blue to black; skin slate gray, all without signs of cardiac or pulmonary insufficiency. 2. Severe headache, nausea, sometimes vomiting, dryness of throat. 3. Central nervous symptoms: confusion, ataxia, vertigo, tinnitus, weakness, disorientation, lethargy, drowsiness, and finally coma. Convulsions may occur but appear to be uncommon. 4. Cardiac effects: heart blocks, arrhythmias, and shock. 5. Death, although uncommon, is usually due to cardiovascular collapse and not resp paralysis. 6. Urinary signs and symptoms may incl painful micturition, hematuria, hemoglobinuria, and renal insufficiency (usually mild). 7. A late acute hemolytic episode should be anticipated at 6 to 8 days after ingestion. /Aniline/ [GOSSELIN. CTCP 5TH ED. 1984 II-197]

HTOX - MAY CAUSE METHEMOGLOBINEMIA & POSSIBLE LIVER & KIDNEY DAMAGE. /3-CHLOROANILINE/ [PATTY. INDUS HYG & TOX 3RD ED VOL2A,2B,2C 1981-82 , p. 2449]

NTOX - CHLOROANILINE WAS ADMIN IN FEED FOR 78 WK AT EITHER OF 2 CONCENTRATIONS: 500 & 250 PPM FOR RATS & 5000 & 2500 PPM FOR MICE. IN RATS & MICE, UNDER CONDITIONS OF THE BIOASSAY, SUFFICIENT EVIDENCE WAS

NOT FOUND TO ESTABLISH CARCINOGENICITY. [CARCINOG TEST PROG, BETHESDA; CARCINOGENICITY, REPORT; 97 PAGES (1979) ISS DHEW/PUB/NIH-79-1745; ORDER NO PB-295896]

NTOX - 4-CHLOROANILINE (DEGRADATION PRODUCT OF DIFLUBENZURON) HAD 96-HR MEDIAN LETHAL DOSE OF 2.4 MG/L TO BLUEGILLS & 48-HR MEDIAN IMMOBILIZATION DOSE LEVEL OF 43 MG/L FOR EARLY 4TH INSTAR MIDGE LARVAE. [JULIN AM, SANDERS HO; MOSQ NEWS 38 (2): 256-9 (1978)]

NTOX - Toxicity of p-chloroaniline was determined by the 14 day Daphnia reproduction test & the 24 hr median effective concn (EC50). EC50 values ranged from 0.008-38 ppm. p-Chloroaniline at concentrations (0.0427-1.35 ppm) which were less than the EC50 showed marked effects on the reproduction rate. [Hattori M et al; Seitai Kagaku 6 (4): 23-7 (1984)]

NTOX - P-chloroaniline was nonmutagenic in TA92, TA1535, TA100, TA1537, TA94, & TA98 strains of Salmonella typhimurium with or without metabolic activation. [Miyata R et al; Eisei Shikensho Hokoku 99: 60-5 (1981)]

NTOX - In six carcinogenicity bioassays, male and female F344 rats were fed diets containing aniline hydrochloride, p-chloroaniline, o-toluidine hydrochloride, azobenzene, daspone, (4,4'-sulfonyldianiline), or D & C red number 9; (5-chloro-2-(2-hydroxy-1-napthalenyl)azo)-4-methylbenzenesulfonic acid, barium salt). The rats, from 6 weeks to 2 years old, were given the cmpd at two dose levels, the estimated maximum tolerated dose and one-half that dose. In all six bioassays, dose dependent incidences of splenic sarcomas and fibrosis were seen, with the highest incidences in male rats. Fatty infiltration also was seen in the spleen. Sarcomas appeared to arise in the splenic red pulp or splenic capsule, usually in association with areas of parenchymal and capsular fibrosis and pigmentation. Larger tumors metastisized to the peritoneal cavity and abdominal organs. [Goodman DG et al; JNCI 73 (1): 265-73 (1984)]

NTOX - P-Chloroaniline has a lethal effect on the embryos of Xenopus laevis at 100 ppm and is teratogenic at 1 and 10 ppm. [Dumpert K; Ecotoxicol Environ Saf 13 (3): 324-38 (1987)]

ADE - TOMATO UPTAKE OF 4-CHLOROANILINE FROM SOIL INCR IN DIRECT PROPORTION WITH AMT PRESENT IN SOIL. CMPD WAS MOSTLY ROOT-LOCALIZED, BUT TRANSLOCATION TO STEM INCR WITH INCR CONCN. IN CARROTS, ROOTS ACCUM ONLY SLIGHTLY MORE CHLOROANILINE THAN GREEN PARTS. 4-CHLOROANILINE WAS ALSO TAKEN UP BY WHEAT & BARLEY. [FUCHSBICHLER G ET AL; Z PFLANZENKR PFLANZENSCHUTZ 85 (5): 298-307 (1978)]

ADE - OATS TOOK UP & TRANSLOCATED 1.4% OF SOIL-BOUND (14)C-4-CHLOROANILINE WITHIN 6 WK; UPTAKE FROM SOLN WAS 1.7-2.3%. LABELED COMPD WAS ADDED TO SOIL AT 1 PPM. THERE WERE ONLY SLIGHT DIFFERENCES IN RESULTS BETWEEN 2 SOIL TYPES (HUMUS-RICH GLEY & PARABROWN SOILS) STUDIED. [FUCHSBICHLER G ET AL; Z PFLANZENKR PFLANZENSCHUTZ 85 (7): 404-12 (1978)]

ENVS - p-Chloroaniline may be released to the environment during its production or use in the manufacture of dye intermediates, agricultural chemicals and pharmaceuticals. If released on soil, it will rapidly combine chemically with soil components and partially be mineralized by chemical and biological action. A few percent of the p-chloroaniline will volatilize from the soil. If released into water p-chloroaniline will be primarily lost due to volatilization (half-life 6.4 hr), photooxidation in surface layers (half-life 0.4 hr), and rapid chemical reactions with humic materials and clay in the water column and sediment. Degradation in air will primarily be due to reaction with hydroxyl radicals (half-life 4.6 hr), although direct photolysis is also possible. Human exposure will primarily be in the workplace from inhalation or dermal contact. (SRC) [CITATION]

FATE - The average concn of chemicals, 4-chloroaniline, hexachlorobenzene, & pentachloronitrobenzene, dosed in small experimental ponds in Southern Germany during the application period (4-6 wk) was about 50 ug/l. Residues were determined in water, sediment, & flora & fauna species up to 166 wk later. Decrease of all chemicals in water phase follows

exponential functions & can be correlated to some extent with the physicochemical properties such as volatility & vapor pressure. The residual behavior of the model compounds followed a similar pattern resulting in high initial concentrations in biota & a slow buildup & subsequent decline of concentrations in sediment. Radioactivity could be found in some fauna species & sediment 3 yr after application. Anisols & azo compounds were found to be conversion products of pentachloronitrobenzene & 4-chloroaniline. [Schauerte W et al; Ecotoxicol Environ Saf 6 (6): 560-9 (1982)]

FATE - (14)C labeled 4-chloroaniline was applied to soil in lysimeter, corresponding to 1.25 ppm to a depth of 10 cm, & barley was sown. After 20 wk, a total of 32.8% of the radiocarbon applied was recovered, in soil 32.4%, in plants 0.3% & in leaching water 0.1%. Radioactivity in soil consisted of 30.8% unextractable residues & 1.6% sol conversion products; that in plants consisted of 0.24% unextractable residues & 0.03% sol metabolites (% of applied (14)C). In the 2nd & 3rd yr after the application potatoes & carrots, respectively, were grown; total recoveries were 32.0% & 31.2% respectively. The soluble radioactive fractions in soil & plants of the first 2 yr contained 4-chloroformanilide, 4-chloroacetanilide, 4-chloronitrobenzene, 4-chloronitrosobenzene, 4,4'-dichloroazoxybenzene, and 4,4'-dichloroazobenzene. The radioactive substances unextractable in soil and those in leaching water were partially hydrolyzable and gave 4-chloroaniline. [Freitag D et al; J Agric Food Chem 32 (2): 203-7 (1984)]

FATE - TERRESTRIAL FATE: If released on soil, p-chloroaniline will bind tightly to soil although in the first few hours after a spill, a small amount will volatilize. The p-chloroaniline will undergo both biological and chemical transformation. Mineralization occurs most rapidly in the early weeks of incubation with as much as 7.5% degradation to CO2 occurring in 6 weeks and 17% occuring in 16 weeks. Most (70-90%) of the p-chloroaniline is transformed into inextractable residues and there is no significant leaching out either vertically or horizontally into surrounding layers of soil. A three year field test in which (14)C labeled p-chloroaniline was applied to 60X60X60 cm box cultivated with barley and later with potatoes and carrots under outdoor conditions resulted in approximately 30% of the applied radioactivity being retained at the application site in the upper layer of soil after the first year and 67% being lost to the atmosphere(1). Uptake by plants, migration into deeper soil layers or into leaching water was low(1). From previous experiments, it is known that the bulk of the atmospheric loss is not volatilized 4-chloroaniline or conversion products, but rather CO2 resulting from mineralization(1). The situation after the second and third year did not alter appreciably(1). No free unchanged 4-chloroaniline could be detected in either soil or plants(1). Conversion products isolated under these environmental conditions included 4-chloroformanilide, 4-chloroacetanilide, 4-chloronitrobenzene, 4-chloronitrosobenzene, and the condensation products 4,4'-dichloroazoxybenzene and 4,4'-dichloroazobenzene(1). It is hypothecized that phenolic and hydroxylamine metabolites were not identified although they were found in laboratory experiments because of their chemical instability(1). In summary, it is clear that free 4-chloroaniline is not presistent in soil; it is subject to various acylation and oxidation reactions and finally to total biodegradation and incorporation into soil and plant constituents(1,SRC). [(1) Freitag D et al; J Agric Food Chem 32: 203-7 (1984)]

FATE - AQUATIC FATE: If released into water, p-chloroaniline will volatilize (half-life 6.4 hrs in a typical river), photooxidize in surface layers

(half-life 0.4 hr), biodegrade (half-life several days in well acclimated water), and chemically bind to clays and humus in sediment and particulate matter in the water column. When (14)C labeled p-chloroaniline was added to an experimental pond, the (14)C label disappeared from the water phase in two phases with half-lives of about 3 and 11 days, respectively(1). It was assumed that the initial loss results from volatilization. After a day, a thin brown film of decomposition products was formed. The reduced loss rate after the first few days is probably a result of lower volatility of the decomposition products(1). In estuarine water, photolysis was also an important process, but no biodegradation occurred in 3 days(2). The half-life estimated by sampling at points along the Rhine River in the Netherlands is 0.3 to 3 days(3). [(1) Schauerte W et al; Ecotox Environ Safety 6: 560-9 (1982) (2) Hwang HM et al; Water Res 21: 309-16 (1978) (3) Zoetman BC et al; Chemosphere 9: 231-49 (1980)]

FATE - ATMOSPHERIC FATE: If released into air, p-chloroaniline will degrade by reacting with photochemically produced hydroxyl radicals (half-life 4.6 hr) and possibly also by photolysis in the vapor phase or while adsorbed on airborne particulate matter. The rates for vapor phase photolysis are not available. A highly soluble chemical, p-chloroaniline will probably be scavenged by rain. (SRC) [CITATION]

BIOD - The results of biodegradability screening studies for p-chloroaniline are conflicting with results ranging from no degradation to rapid degradation using soil, sewage, activated sludge and fresh water inocula. The most frequently reported results are that p-chloroaniline biodegrades rather slowly with acclimation(1-6). The reason for the conflicting results may be due to toxicity of metabolic intermediates(1), differences in concentrations and inocula used, sensitivity of p-chloroaniline to chemical oxidation, and lack of sufficient acclimation. Some results are: 28% degradation in 5 days(2), 10% and 18% degradation in 28 days(2,5), no degradation in 28-30 days(2,3), 97% degradation in 10 days including an 8 day lag period(3), 97% removal in 5 days after 20 days acclimation(4) and 46 and 100% removal in 8 and 22 days, respectively, after a 14 day acclimation period(5). Biotransformation frequently involves acylation and oxidation to phenols(14). In the one river die-away test which was found, 10 ppm p-chloroaniline degraded slowly in Nile River water over a period of 2 months but on redose increasingly larger concentrations of the chemical were degraded in shorter and shorter times so that on the 8th redose 100 ppm was degraded in a few days(7). Several studies were performed where labeled p-chloroaniline was incubated with soil(8-10). In one study, 12-17% mineralization occurred in 16 weeks with the maximum rate of degradation occurring between 1 and 3 weeks(8). 86% of the labeled residue was present as unextractable material bound to the soil and 1-4% of the residues were extractable(8). In another study 7.5% mineralization occurred in 6 weeks, with 72% and 7% unextractable and extractable residue remaining(9). In comparing fractions with autoclaved controls, it is apparent that part of the extractable fraction is being biologically mineralized(9). Biological transformation of the unextractable residue, however, also occurs(9). 8-9%, 69-73%, and 5-7% CO2, unextractable residue, and extractable residue, respectively, resulted in a third study after 16 weeks of incubation(10). It has been suggested that the binding of the chloroaniline to soil may extend its life in soil to 10 years(11). During composting with refuge, 14% of the p-chloroaniline was metabolized in 21 days(12). No mineralization occurred when p-chloroaniline was incubated under anaerobic conditions with digester

sludge for 1 month(13). [(1) Baird R, et al; J Water Pollut Control Fed 49: 1609-15 (1977) (2) Rott B et al; Chemosphere 11: 531-8 (1982) (3) Janicke W, Hilge G; Gas Wasserfach Wasser Abwasser 121: 131-5 (1980) (4) Pitter P; Water Res 10: 231-5 (1976) (5) Schmidt-Bleek F et al; Chemosphere 11: 383-415 (1982) (6) Torgeson DC; Interaction of Herbicides and Soil Microorganisms p 73 16060 DMP 03/71 (1971) (7) El-Dib MA, Aly OA; Water Res 10: 1055-9 (1976) (8) Suess A et al; Z Pflanzezernaehr Bodenkd 141: 57-66 (1978) (9) Bollag JM et al; J Agric Food Chem 26: 1302-6 (1978) (10) Fuchsbichler G et al; Z Pflanzenkr Pflanzenschutz 85: 724-34 (1978) (11) Bartha R; J Agric Food Chem 19: 385-7 (1971) (12) Korte F et al; Chemosphere 1: 79-102 (1978) (13) Shelton DR, Tiedje JM; Development of Tests for Determining Anaerobic Biodegradation Potential p 92 USEPA 560/5-81-013 (1981) (14) Freitag D et al; J Agric Food Chem 32: 203-7 (1984)]

KOC - ADSORBED 4-CHLOROANILINE DESORPTION RATES FROM DIFFERENT SOILS INTO WATER WERE 11.9% TO 68.3%; % REPLACEABLE BY 3,4-DICHLOROANILINE WAS 17.9% TO 100%, INDICATING WEAKLY SORBED FRACTION. PROLONGED SOIL RESIDENCE OF 4-CHLOROANILINE GIVES CHEMICAL BONDING, INSEPARABLE FROM SOIL. [FUCHSBICHLER G, SUESS A; CHEMOSPHERE 7 (4): 345-50 (1978)]

KOC - Adsorption of p-chloroaniline by 5 soils was studied in the lab. Adsorption decreased with the depth of the soil sample, due to decreased organic carbon. [Mueller-Wegener U; Chem Erde 41 (2): 175-81 (1982)]

WATC - SURFACE WATER: Not detected in Lakes Ontario (1 location), Erie (2 locations), Michigan (5 locations), and Superior (1 location). Rhine River (Germany) - 80 ppt, yearly average(2). Rhine River and two tributaries (The Netherlands) 130-220 ppt, average; 240-740 ppt maximum with 96-100% frequency of detection(3). Meuse River (The Netherlands) 20-30 ppt, average, 80-120 ppt maximum, 44-50% frequency of detection(3). While detected in Rhine Delta water, not found in surface waters from agricultural areas in the Netherlands(4). [(1) Great Lakes Water Quality Board; An Inventory of Chemical Sub Identified in the Great Lakes Ecosystem Vol 1 pp 195 (1983) (2) Kussmaul H; pp 265-75 in Pergamon Ser Environ Sci (1978) (3) Wegman RCC, Dekorte GAL; Water Res 15: 391-4 (1981) (4) Greve PA, Wegman RCC; Schriftenr Ver Wasser, Boden, Lufthyg Berlin-Dahlem 46: 59-80 (1975)]

SEDS - Release of tightly complexed 4-chloroaniline (4-CA) from treated soil humic acids & whole soils by pyrolysis in atmosphere of helium was studied. Pyrolysis of soil humic acids containing tightly complexed 4-CA resulted in release of approx 54% of the radioactivity with approx 5% detected via radioassay or gas-liq chromatography as intact 4-CA. Three soils of varying organic matter & clay concentrations complexed 10% of applied 4-CA; extractable radioactivity incr & tightly complexed 4-CA decreased as organic matter concn decreased & clay concn increased. The quantity of radioactivity released by pyrolysis was greatest for soils with low organic matter & high clay contents; 22-73% of the radioactivity could be released with 3-16% extractable into benzene from basified pyrolyzate, the amt depending on the nature of the soil, the type of pyrolysis probe, & the concn of the applied 4-CA. [Worobey BL, Webster GR Ba; J Agric Food Chem 30 (1): 164-9 (1982)]

SEDS - ... RESIDUAL LIFE OF CHLOROANILINES IN SOIL COULD BE AS LONG AS 10 YR. /CHLOROANILINES/ [KEARNEY. HERBICIDES 2ND ED VOLS 1,2 1975 , p. 641]

SEDS - AFTER 16-WK OF INCUBATION, 60% OR MORE OF (14)C-4-CHLOROANILINE ACTIVITY REMAINED IN SOIL UNDECOMPOSED, ADSORBED TO SOIL CONSTITUENTS, LINKED COVALENTLY TO ORG SOIL COMPONENTS, OR CONVERTED BY CONDENSATION INTO SECONDARY TRANSFORMATION PRODUCTS. [SUESS A ET AL; Z PFLANZENERNAEHR BODENKD 141 (1): 57-66 (1978)]

CREV - IARC CANCER REVIEW; ANIMAL SUFFICIENT EVIDENCE; HUMAN INADEQUATE EVIDENCE; IMEMDT 57,305,93; IARC Monogr Eval Carcinog Risk Chem Man