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TASK 4 - Endangerment -  
Assessment

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TASK 4 REPORT

ENDANGERMENT ASSESSMENT  
FOR THE  
LOCKPORT COAL TAR SITE

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## TASK 4 - ENDANGERMENT ASSESSMENT

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### INTRODUCTION

Endangerment assessment combines site evaluation, chemical fate and transport evaluation, basic toxicology and risk assessment, into a description and quantification of actual and potential hazards associated with the site. The objective of an endangerment assessment is to characterize potential hazards to assess the level to which site clean up is required.

The endangerment assessment process consists of the following steps:

- o source characterization
- o environmental fate of contaminants
- o exposure pathway characterization
- o receptor characterization
- o exposure assessment
- o risk assessment

### SOURCE CHARACTERIZATION

The former coal gasification site located in Lockport, New York has been investigated in detail during the past two years. Data from these studies suggest that coal tar exists in the surface soils at the site, and that a plume of coal tar derivatives and solute exists within the local ground water. The specific findings of these site studies have been reported in a series of reports (Task 1 through Task 3 and Task 7).

The chemical and physical properties of coal tar compounds govern the release of various derivatives and solute into the local environment. This release into the

air, ground water, surface water and potential migration within site soils contribute to the endangerment assessment discussed here.

The field studies conducted during the past two years have characterized the type and concentration of contaminants present both on and off site. Contamination was found to occur in two media: soil and ground water. Ambient air monitoring at the site using field survey instruments did not detect any air quality impacts. Coal tar contamination in site soils was confirmed, with the highest concentrations found in soils in test trenches that intercepted several wooden sump pits.

Principal compounds found representing coal tar derivatives and solute in soils were polynuclear aromatic hydrocarbons (PAH), volatile aromatic hydrocarbons and phenols. The major component of the PAH's found in this area was naphthalene at concentrations of up to 29,000 ppm or approximately 3% naphthalene. Other major constituents include anthracene at 13,000 ppm and flouranthene at 5300 ppm. Volatile aromatic compounds in soil in the sump pit area included up to 155 ppm toluene and 128 ppm benzene.

Ground-water contamination resulting from the site is confused by the apparent contributions of gasoline derivatives from an off-site source and the presence of fuel oils at other locations. It is apparent, however, that a ground-water contaminant plume has resulted from the presence of coal tar wastes. This plume is centered at the northwest corner of the NYSEG substation. Concentrations near the center of the plume for polynuclear aromatic hydrocarbons range up to 17 ppm. Approximately 400,000 sq. ft of area is included within a plume interpreted on the basis of total PAH concentrations exceeding 0.1 ppm.

#### ENVIRONMENTAL FATE OF CONTAMINANTS

The fate of a chemical compound in the environment is a function of the behavior of the compound under conditions associated with natural surroundings.

Environmental fate is usually described in terms of the mobility and persistence of a compound, which can be deduced from its physical and chemical properties.

A summary of relevant physical/chemical characteristics is presented in Table 1 for representative chemicals from the major classes of contaminants observed at the site - polynuclear aromatic hydrocarbons (PAHs), volatile aromatics, and phenols. A summary of data describing half-lives of these compounds for various environmental fate processes is presented in Table 2. Significance of these data are discussed below.

#### Polynuclear Aromatic Hydrocarbons (PAHs)

Available data for a variety of PAH compounds are included in Tables 1 and 2. Properties for this class of compounds generally correlate with molecular weight, or number of rings in the compound structure. Solubilities range from 34.4 mg/l for the 2-ring naphthalene to less than 0.01 mg/l for 4 and 5 ring compounds (such as chrysene and benzo(a)pyrene). Estimated sediment/water partition coefficients similarly vary from about 100 for naphthalene to 10,000 to 100,000 for representatives of the 4 and 5 ring compounds respectively. These values indicate a strong tendency for the higher molecular weight PAH compounds to adsorb to sediments and suspended particulate matter. In natural waters, most PAH compounds will occur bound to solids rather than in the dissolved aqueous phase. This trend can be expected to continue for higher molecular weight PAH compounds, although available data are sparse. This low solubility limits the ground-water mobility of heavier PAH compounds, with perhaps the only exception being that of relatively soluble naphthalene.

These compounds are not prone to rapid volatilization. Vapor pressures are relatively low for PAH compounds. The largest reported vapor pressure is for naphthalene (0.05 mm Hg), decreasing to less than  $10^{-6}$  mm Hg for 4 ring and heavier compounds.

The low solubility and high organic/water partition coefficient ( $K_{oc}$ ) values for the PAH compounds are reflected in high bioconcentration factors (see Table 1). These factors reflect the tendency for PAH compounds to be taken up in organic material (including biota) in preference to water. Bioconcentration factors (BCF) represent the ratio of steady-state-tissue concentrations to water concentrations of a given chemical. Despite the relatively high estimated BCF for PAH compounds, these compounds show little tendency to accumulate in fatty tissues of fish or man (USEPA, 1980). This appears to be due to the fact that PAHs are rapidly metabolized and excreted. Therefore, while significant bioconcentration can occur, tissue PAH levels will decrease after exposure ends. Considering the tendency of PAH compounds to adsorb to sediments, bioconcentration in fish is likely to be most significant for species of bottom feeders resident in areas where accumulation of PAHs in sediment may have occurred.

In developing ambient water quality criteria for PAH compounds, a bioconcentration factor of 30 for PAH in fish, is recommended by EPA (USEPA, 1980). This value reflects the tendency of PAHs to be metabolized and excreted, as well as their tendency to bioconcentrate.

Three processes are known to contribute to the removal of PAHs from aquatic systems - photolysis, volatilization, and biodegradation. PAH compounds are strong UV absorbers, and degrade in hours in the presence of UV light. Despite the relatively low vapor pressures, volatilization half-lives on the order of days have been estimated for several PAHs from shallow surface waters.

Reported biodegradation rates for PAHs are highly variable. Rates depend on environmental conditions and the acclimation of microbes to PAH. Reported biodegradation half-lives in sediments are 5 hours for naphthalene, 280 hours for anthracene, 7000 hours for benzo(a)anthracene, and 21,000 hours for benzo(a)pyrene, (USEPA, 1979). Rates are 10 to 400 times slower in pristine sediments than oil-contaminated sediments where microbes are acclimated to PAH (USEPA, 1979). Canal sediments likely represent an environment where this acclimation has already



occurred, and therefore biodegradation of PAH's in the sediments is likely to be taking place.

PAH compounds containing 4 or more rings (e.g., benzo(a)anthracene, chrysene, pyrene, benzo(a)pyrene, benzo(b) fluoranthene) are considered resistant to biodegradation. Experience with landfarming of heavy petroleum residues indicates that significant biodegradation of aromatics occurs only under aerobic conditions in well-aerated, fertilized soils.

PAH compounds in subsurface soil and ground water are quite persistent. The potential removal processes for PAHs - volatilization, photolysis and biodegradation - does not occur at significant rates under typical subsurface conditions.

In summary, PAH compounds will generally be sorbed to soils, sediments, and suspended particulate matter. PAHs may be removed from shallow surface waters by volatilization and photolysis. Significant biodegradation of the lighter PAH compounds may occur in surface soil and sediments. Bioconcentration of PAHs can be significant, but can be metabolized and excreted thus limiting bioaccumulation. The lighter (2 and 3 ring) PAH compounds, especially naphthalene, potentially have high mobility in the aqueous phase; heavier PAH compounds have extremely low mobility in ground water. PAH compounds are quite persistent in subsurface soils and ground water.

#### Volatile Aromatics

Volatile aromatics are represented by benzene, toluene, and xylene. These compounds are slightly soluble in water, with solubilities ranging from 180 mg/l for xylene to 1780 mg/l for benzene. In general, solubility decreases with increasing molecular weight. Environmentally significant concentrations can occur for these substances in true solution. They are considered relatively mobile in both surface water and ground water. Estimated sediment/water partition coefficients for volatile aromatics in sediment containing a nominal 5% organic carbon range from 9.2 for

benzene to 37.6 for xylene. These values are relatively low for organic compounds. They indicate only a slight tendency for these compounds to accumulate in sediments or to bioaccumulate.

The volatile aromatics are volatilized from soils or surface waters in contact with the atmosphere. Vapor pressures range from about 6 mm Hg for xylene to 95.2 mm Hg for benzene. Half-lives for volatilization of benzene and toluene have been estimated on the order of 5 hours in surface water of one meter depth. The volatile aromatics are subject to microbial degradation, particularly under aerobic conditions in surface waters and aerated soils. A first-order biodegradation rate constant of  $0.11 \text{ day}^{-1}$  for benzene in aquatic systems has been reported. This corresponds to a half-life for biodegradation of 6.3 days. Benzene and toluene are considered relatively degradable (Lyman, et al, 1982).

In summary, volatile aromatics may be removed from shallow surface waters by volatilization in a matter of hours to days. They are unlikely to accumulate in sediments or to bioaccumulate. They may biodegrade in the presence of acclimated aerobic microbes. The volatile aromatics are relatively mobile in ground water due to their solubility. They are relatively persistent in ground water since the significant removal processes of volatilization and biodegradation are inefficient in the ground-water medium.

### Phenols

Properties of three representative phenolic compounds - phenol, m-cresol, and 2,4-xylene, are included in Table 1 and 2. As a group, the lower molecular weight phenolics are relatively soluble, with solubilities ranging from 17,000 mg/l for 2,4-xylene to 93,000 mg/l for phenol. They have correspondingly low K<sub>oc</sub> values, sediment/water partition coefficients, and bioconcentration factors. They do not accumulate significantly in sediments or organisms, and are relatively mobile in the aqueous phase of surface and ground waters.

Phenols are biodegraded by a variety of mixed microbial populations in soils and surface waters. Biodegradation of phenols in ground waters is limited by the ability of the ground water to support microbial populations. A paucity of quantitative data exist concerning other removal processes.

In summary, the low molecular weight phenolic substances are mobile in surface and ground waters. They are unlikely to be adsorbed by soils and are unlikely to bioaccumulate significantly. Biodegradation is probably the major removal process for phenols, where conditions support appropriate microbial populations. Such conditions may have evolved at the site given the duration of the presence of phenols. This summary of behavioral characteristics applies to the compounds identified. Higher molecular weight phenolics, also may be present in "total phenol" analyses reported at the site. Behavior characteristics of the higher molecular weight compounds may differ somewhat from the typical representative phenolics discussed above.

#### EXPOSURE PATHWAY CHARACTERIZATION

Routes of exposure are defined by evaluating possible migration pathways in terms of population and adjacent environments that may be affected. Pathways are characterized through an assessment of the physical, topographical, geological, hydrological and meteorological features of the site and its environs.

Figure 1 shows the potential exposure pathways at the Lockport site. Each of these pathways is discussed below.

Air - Available ambient air monitoring data do not indicate significant airborne vapor emissions from the site. All air measurements taken during field investigations at the site showed no detectable concentrations of contaminants.

Soil - Direct contact with waste materials is not a significant potential route of exposure to the public because the site is secured.

Soil-Groundwater - A well survey performed indicated that no wells were being used for drinking within a 1.5-mile radius of the site. Migration of the contaminations has not affected the Lockport water supply mains in the vicinity of the site because the contamination is located well below the elevation of these mains. Vertical migration of the contaminants into deeper aquifers is retarded by the low vertical permeabilities of the Rochester shale and hence is considered unlikely.

Soil-Groundwater - Surface Water - The primary potential pathway of public and environmental exposure to contaminants is through ground water/subsurface transport and ultimately entering a surface water body (i.e., Erie Barge Canal). Contaminants originating from on-site soils percolate downward through vertical fractures in the bedrock entering the ground-water table. The contaminants continue downward until they reach the Rochester shale. The very high ratio of horizontal to vertical permeability of the Rochester shale changes the direction of transport in this material so that the contaminants begin to move laterally from the site. The only identified discharge point to a surface water body is at the Erie Barge Canal. Once in the canal, PAH compounds tend to adsorb onto particulates and eventually settle out of the water column and enter the sediments. Phenols and volatile aromatics entering the canal are likely to be transported in the aqueous phase, and are subject to volatilization and biodegradation in the canal. These compounds are likely to be removed from the water column in times on the order of days by these processes. The potential receptors are as follows:

Ingestion of Contaminated Surface Water. This does not pose a risk because water in this section of the canal is not used as a drinking water supply. The intake for the local emergency water supply is approximately one mile up stream of the point of entry of site-derived contaminants.

Fish Uptake of Contaminants. Fish in the contaminated portion of the canal can ingest and concentrate PAH's, both from the canal water and

the sediments (for bottom feeders). Volatile aromatics and phenols are unlikely to accumulate in fish tissue.

Ingestion of Fish. Residents who catch and eat fish from the contaminated portion of the canal would increase their intake of PAH's by that amount held or bioconcentrated in fish tissue.

Direct Contact with Contaminated Sediments. The canal is not used for swimming or other water contact recreation. Hence, exposure is limited to times when the canal is drained of water, thus exposing potentially contaminated sediments. Because of the difficult terrain, it is unlikely that children would play in the area or that local residents would enter the drained canal. Therefore, direct contact is unlikely to represent a significant route of exposure.

## RECEPTOR CHARACTERIZATION

Exposure pathways may lead to sensitive receptors that could be adversely impacted by the sites contaminants. Potential receptors include the public or ecological resources.

Based upon the pathway analysis, the two receptors potentially affected by risks associated with the Lockport site are fish in the canal and residents who catch and eat fish from the canal. These receptors are further characterized below.

Fish - Fish sampling programs conducted by the NYSDEC (NYSDEC, 1985a, b) in Tonawanda Creek and the western portion of the Erie Barge Canal indicate that the following fish are commonly found in these waters:

Brown Bullhead  
Goldfish  
Smallmouth Bass  
White Sucker

Northern Pike  
Pumpkinseed  
Rock Bass

The white sucker is primarily a bottom feeder; the other species feed upon insects, crustaceans, molluscs and small fish. Given the drained condition of the canal during about one-half of the year, the food supply for non-bottom feeders is likely limited to insects and small fish. No studies have been performed to determine the home range of fish in the canal; in general, however, these species tend to remain in a relatively limited area except in spawning season, when fish tend to travel greater distances in search of suitable spawning habitat.

Fishing in the Barge Canal. According to a recent NYSDEC survey of angler use associated with the locks and lift bridges of the Barge Canal (NYSDEC, 1984a), the canal is a sport fishing resource. Most of the data used in the report was collected by lock operators. Even though data for Locks 34/35 in Lockport indicate that access for shorefishing is only "fair" because of the steep terrain and high bonks in the vicinity of these locks, this area still ranks 20th out of a total of 50 locks in fishing effort, with 2421 angler trips from May through October, 1982. This usage level is close to the mean level of fishing pressure for the entire canal system. Residents who eat fish caught in the contaminated portion of the barge canal are potential receptors of contaminants observed in surface water samples taken from the barge canal.

#### EXPOSURE ASSESSMENT

The objective of exposure assessment is to evaluate the extent and duration of any exposure to the site contaminants by human or ecological resources. Insufficient aquatic toxicological data exists for individual PAHs to adequately assess the potential toxic effects on fish. Criteria for acute aquatic toxicity have been developed for acenaphthene, fluoranthene, and naphthalene, and a chronic toxicity criterion has been developed for naphthalene. However, no data are available to

develop criteria for aquatic toxicity of a total PAH as a class of compounds. None of the canal water samples showed concentrations exceeding the criteria levels listed in Table 3 for criteria contaminants. The levels of other contaminants observed in canal waters are low, and as previously identified, their half-lives are short in surface water environments. Hence, no further consideration of the fish population will be made with respect to direct effects.

Exposure to contaminants by humans is possible through the food chain. Residents who include in their diet fish caught in the contaminated portion of the Barge Canal potentially increase their intake of PAHs. An estimate of this exposure has been made based upon the available canal water analyses.

No significant exposure is anticipated for volatile aromatics or phenolic substances. As shown in Table 4, observed concentrations of total volatile aromatics in canal water is low, averaging less than 0.001 mg/l at all locations. Since the canal adjacent to and downstream of the site does not represent a drinking water supply, and volatile aromatics (e.g., benzene) are not expected to bioaccumulate significantly in fish, no significant exposure is expected for these compounds.

Average concentrations of total phenolic substances ranged from 0.002 mg/l (CSL-4) to 0.023 mg/l (CSL-2) as shown on Table 5. The relatively high means reported for CSL-2 and CSL-3 were due almost entirely to data obtained during the October 19-21, 1983 sampling event. The cause of these high values is not known. All other reported values were at or below 0.005 mg/l. Since there is no potential risk of exposure to a drinking water supply, and since phenols do not significantly bioaccumulate in fish, no significant human health exposure is anticipated for phenols in the canal in the vicinity of the site.

The contribution of PAH's to the canal from the site was estimated for purposes of this analysis. The flux of PAH's from ground water into the canal was computed using Darcy's Law as follows:

$$Q = A \times I \times K \times C$$

- where
- C is the concentration of PAH in groundwater
  - A is the area through which water flows into the canal
  - I is the local hydraulic gradient
  - K is the permeability of material through which groundwater flows

The data used to calculate the flux and the results in terms of minimum, maximum and most probable values, are shown in Table 6. In computing the area of infiltration into the canal, the entire length and depth of canal adjacent to the observed plume was considered. Concentrations of inflow were estimated assuming, the entire length to be bounded by the 0.01 mg/l contour line (845 ft. in length). The depth of water in the canal was taken as 12 feet. Permeabilities for maximum and minimum cases were estimated from slug test data. The most probable values used represent judgemental estimates based on experience. Hydraulic conductivities were computed at the south end of the site as 0.09 ft/ft, near W. Genesee Avenue as 0.125 ft/ft and at the north end of the site as 0.086 ft/ft. These values are assumed to be representative of the entire site area, and average about 0.1 ft/ft. At the canal in the vicinity of the site, PAH concentrations in ground water samples ranged from approximately .01 mg/l to 6.2 mg/l. A weighted average of the concentrations along the canal yields a concentration of about 2.2 mg/l of PAH.

As illustrated in Table 6, under worst case assumptions, the site is expected to contribute up to 28 lbs/day of total priority pollutant PAH to the canal water. Worst case assumptions are considered unrealistic, but do represent an upper bound estimate, beyond which no consideration should be given. Based upon average flow in the canal during summer months of 1100 cfs, this quantity of total PAH entering the canal would result in an average increase in total priority pollutant PAH concentration in the canal water of about 0.005 ppm. It should be noted however, that with "most probable" assumptions, the calculated contribution of total priority pollutant PAH to the canal water is only  $1.26 \times 10^{-3}$  pounds per day.



Table 7 summarizes priority pollutant PAH data for canal water samples in the vicinity of the site collected during September and October 1984. For stations CSC-1, immediately upstream of the site, and CSC-2 adjacent to the site, the average total priority pollutant PAH content of all samples reported was about 1.1 mg/l. These results indicate that the current contribution of the site contaminants to PAH levels in the canal water via ground water discharge is small - less than 1% of the total observed priority pollutant PAH levels in the vicinity of the site. This suggests that additional sources of PAH to the canal exist. Based on relatively high levels of contaminants observed upstream of the site an upstream source (or sources) is suggested. Upstream sources of oil or tar compounds, boat traffic, leaking underground tanks or sediment reservoirs of PAH compounds all represent possible explanations of observed canal contamination.

The public health risk associated with PAH compounds is generally attributed to the carcinogenic properties of a number of PAH compounds. However, it should be recognized that not all PAH compounds are carcinogenic. Table 8 summarizes information reported on the potential carcinogenic properties of the priority pollutant PAH compounds reported in studies at the site. Based upon this information, the following PAH compounds have been identified as carcinogenic PAH for the purposes of this risk analysis:

- o Benzo (a) anthracene;
- o Benzo (a) pyrene;
- o Benzo (b or k) fluoranthene;
- o Chrysene; and
- o Indeno (1,2,3,-c,d) pyrene or dibenzo (a, h) anthracene.

In selecting this group, it was conservatively assumed that chrysene is a carcinogenic PAH, although it appears to be weakly carcinogenic relative to the other four members of this group. In addition for PAH isomers which cannot be distinguished by GC analysis (e.g., benzo (b) fluoranthene/benzo (k) fluoroanthene and ideno (1,2,3,-

c,d) pyrene/dibenzo (a,h) anthracene), it was conservatively assumed that concentrations reported for an isomeric pair were considered as carcinogenic PAH if either member of the pair is carcinogenic. This leads to a worst case estimate of true concentrations, but no better estimate can be readily supported.

Since the canal water downstream of the site is not used as a drinking water supply, the primary potential route of ingestion of PAH from the site will be ingestion of contaminated fish from the canal.

In order to evaluate the potential contribution from site contaminants to risk associated with PAH in the canal water, it was conservatively assumed that the site contributed a total of 28 lbs/day of total priority pollutant PAH to the canal water, resulting in an increased concentration of about 0.005 mg/l, as discussed above. Since the primary risk from PAH compounds is due to their carcinogenic potential, the carcinogenic fraction of total PAH entering the canal via seepage of ground water was determined. Well MW-8, adjacent to the canal, represents the primary source of estimated total priority pollutant PAH to the canal. Table 9 presents the ratio of carcinogenic PAH to total priority pollutant PAH for this well during five reported sampling events (WCC, 1985). The fraction of carcinogenic PAH ranged from 0% to 17.9%, with an average of about 10%. The ratio of carcinogenic to total PAH of 10% is consistent with data reported by USEPA (1980). Potential exposure to carcinogenic PAH from ground water seepage from the site is therefore assumed to represent 10% of total PAH.

To estimate exposure to carcinogenic PAH from the site, it was assumed that the average bioconcentration factor (BCF) for PAH in fish is 30, and the average per capita consumption of fish is 6.5 g/day, consistent with USEPA (1980). Potential exposure to carcinogenic PAH is calculated as follows:

$$\begin{aligned} &0.005 \text{ ppm total PAH (from site)} \times 10\% \text{ (fraction carcinogenic PAH)} \times 30 \text{ (BCF)} \\ &= 0.015 \text{ ppm (ug/g) average fish tissue concentration of carcinogenic PAH.} \end{aligned}$$

Conservatively assuming all fish consumed ore from the barge canal in the vicinity of the site, the daily carcinogenic PAH dose is:

$$0.015 \text{ ug/g} \times 6.5 \text{ g/day} = 0.0975 \text{ ug/day carcinogenic PAH.}$$

Since the canal flows only 6 months per year, the annual average daily dose would be about 1/2 this value, or about 0.05 ug/day carcinogenic PAH. Note that this dose is the portion potentially contributed to the canal by ground water seepage from the site.

This contribution is small compared to total PAH observed in the canal water. Using analagous methods the carcinogenic PAH dosage associated with canal water in the vicinity of the site (from sources other than groundwater seepage from the site) has been estimated as follows.

Average total priority pollutant PAH in the canal water near the site is 1.1 mg/l. During the two sampling events reported, carcinogenic PAH represented approximately 90% of the total priority pollutant PAH reported. This value is anomalously high, and may indicate a mix of PAH in canal water different from that observed in ground water migrating from the site.

Potential exposure to carcinogenic PAH from canal water is calculated:

$$1.1 \text{ ppm (total PAH)} \times 90\% \text{ (fraction carcinogenic PAH)} \times 30 \text{ (BCF)} = 29.7 \text{ ppm (ug/g) average fish tissue concentration of carcinogenic PAH.}$$

$$29.7 \text{ ug/g} \times 6.5 \text{ g/day} = 193 \text{ ug/day carcinogenic PAH.}$$

Assuming this exposure occurs 6 months per year, based upon canal flow, the annual average potential carcinogenic PAH dose from ingestion of fish from the canal would be 96.5 ug/day.

The calculated "worst case" exposure to carcinogenic PAH resulting from ground water seepage from the site (0.05 ug/day) is less than 0.1% of the total estimated carcinogenic PAH dose resulting from ingestion of fish taken from the canal. This "worst case" estimated dose of PAH can be compared to overall estimated human exposure to PAH from all media summarized by USEPA (1980). As shown in Table 10, overall exposure to total PAH is in the range of 1.6 to 16 ug/day, with carcinogenic PAH representing greater than 10% of the total PAH exposure. Using "worst case" assumptions the contribution of site contaminants to canal water is calculated to result in less than about 1% of total PAH exposure for those consuming fish from the canal.

## RISK ASSESSMENT

After the routes of exposure are identified and evaluated, a risk assessment is conducted to further characterize those routes of exposure that contribute significant threats to public health.

The following potential concerns are raised with respect to the Lockport Cool Tar Site:

- o Risk of toxic effects on fish
- o Risk of contaminating a deep aquifer beneath the site
- o Risk of ingestion of PAH's via consumption of fish taken from the canal

Each of these concerns is discussed in the following paragraphs.

### Aquifer Contamination

The hydrogeological testing program conducted at the site supports the conclusion that the coal tar constituents have migrated off-site. The lateral extent of the contaminant plume has been documented. The vertical extent has not been confirmed via testing. The observed change in lateral migration of observed

contaminants upon reaching the Rochester Shale, and the high ratio of horizontal to vertical permeability within that unit suggests that deeper migration of contaminants is unlikely to be a significant problem.

#### Toxic Effects to Fish

As stated above, insufficient published data is available to assess the aquatic toxicological effects of total PAH's on fish in the canal. All canal water samples were below aquatic toxicity criteria levels for the three PAH compounds for which available published data were sufficient to support development of water quality criteria for protection of aquatic life.

#### Ingestion of PAH through Fish Consumption

There are no specific U.S. standards or limitations concerning ingestion of PAH in drinking water or food based upon toxicological properties of PAH. The World Health Organization has developed a recommended drinking water standard of 0.2 ug/l for the sum of six indicator PAH compounds (fluoranthene, benzo (a) pyrene, benzo (g,h,i) perylene, benzo (b) fluoranthene, benzo (k) fluoranthene, and indeno (1,2,3,-c,d) pyrene). These compounds were selected for monitoring PAH levels, rather than for specific toxicological properties (USEPA, 1980), and the list includes both carcinogenic and non-carcinogenic PAH compounds. As such, this standard cannot be used to estimate allowable dosage of carcinogenic PAH.

The USEPA (1980) has developed risk estimates for drinking water containing PAH compounds based upon the carcinogenic properties of benzo (a) pyrene, one of the more potent carcinogens in the PAH group. Lifetime cancer risks estimates for drinking water were developed.

Concentrations of benzo (a) pyrene in drinking water of 28 ng/l, 2.8 ng/l, and 0.28 ng/l (1 ng/l = 0.001 ug/l) were associated with lifetime cancer risks of  $10^{-5}$ ,  $10^{-6}$ , and  $10^{-7}$ .

6 and  $10^{-7}$  respectively, using a linear multi-stage model. EPA applied the risk estimated for benzo (a) pyrene to carcinogenic PAH as a class of compounds.

Assuming an average consumption of drinking water of 2 liters/day, ingested carcinogenic PAH dose associated with lifetime cancer risks can be calculated. For example, for the  $10^{-5}$  lifetime risk, the estimated carcinogenic PAH dose is:

$$0.028 \text{ ug/l} \times 2 \text{ liters/day} = 0.056 \text{ ug/day.}$$

In this model, cancer risk is linear with dosage. Risk increases an order of magnitude for each order of magnitude increase in dosage.

The potential exposure to carcinogenic PAH through ingestion of fish was estimated to be 0.05 ug/day. Based upon USEPA risk estimates for carcinogenic PAH, this exposure is estimated to result in a lifetime cancer risk of slightly less than 1 in 100,000 (or  $10^{-5}$ ) for persons eating fish removed from the canal. This risk estimate is conservative, since it is based upon maximum projected contribution of PAH from the site to the canal, and the assumption that all fish consumption is from the canal.

The projected risk associated with contribution of PAH from ground water seepage is extremely small (less than 0.1%) relative to observed concentrations of carcinogenic PAH in canal water apparently resulting from other sources. The estimated exposure to carcinogenic PAH from unknown sources via ingestion of fish from the canal was estimated to be 96.5 ug/day. Based upon USEPA risk estimates for carcinogenic PAH, this exposure is associated with lifetime cancer risk of greater than 1 in 100 for persons eating fish taken from contaminated canal water. This risk assessment is conservative since it assumes all fish consumed is taken from the canal, and is based upon risk assessment for a potent carcinogen, benzo (a) pyrene, rather than an average risk for the carcinogenic PAH observed in the canal water near the site. Nonetheless, the available data indicate a significant potential risk associated with ingestion of fish taken from the canal in the vicinity of the site.

This analysis, based on available data, and the assumptions inherent in such calculations, indicates the presence of a potential public health risk associated with the fish consumption pathway. This risk results from contaminant levels observed in the canal waters. The contribution to this risk imposed by ground water migration of coal tar contamination from the site however, is small. An upper bound estimate of contaminant load and conservative estimates of inflows suggest a contribution of less than 0.1% of observed contaminants considered. The "most probable" projections of PAH loadings from the site to the canal are approximately 3 orders of magnitude lower than "maximum projections. Therefore, "most probable" projections indicate that the site contribution to public health risk associated with PAH in the canal is probably less than 0.0001%.

Available data show significant concentrations of PAH's upstream of the seeps linked to the site contaminants, indicating the presence of additional sources of these contaminants in the Lockport area. Several other potential PAH sources were identified in this study, including a service station, fuel storage facility and use of the canal by motorized boats. Given the available data, it is not considered meaningful to refine estimates of the actual contribution to total canal water contamination resulting from coal tar derivatives. Current estimates, using upper bound values for inflow to the canal which are deemed unrealistically high, indicate a contribution to the observed level of contamination that is virtually negligible. Realistic estimates of contaminant load and flux would likely result in contamination contributions of an order of magnitude (or more) less than those considered here.

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**Tables**

TABLE I

## CHEMICAL PROPERTIES AFFECTING CHEMICAL FATE IN THE ENVIRONMENT

Class/ Compound	Solubility (mg/l)	Vapor Pressure (mm Hg)	Calculated Organic/ Water Partition Coefficient Koc (1)	Estimated Sediment/ Water Partition Coefficient (2)	Bio- concentration Factor (4,5)
<u>Polynuclear Aromatic Hydrocarbons</u>					
o naphthalene	34.4	0.05	2,000	100	100(3)
o anthracene	0.073	0.0002	68,000	3,400	478(4)
o phenanthrene	1.29	0.0007	14,000	700	486(4)
o fluorene	1.98	.001 - .01	11,000	550	282(4)
o benzo (a) anthracene	0.014	5 x 10 <sup>-9</sup>	188,000	9,400	4,602(4)
o pyrene	0.14	7 x 10 <sup>-7</sup>	51,000	2,500	--
o chrysene	0.002	10 <sup>-11</sup> - 10 <sup>-6</sup>	540,000	27,000	4,620(4)
o benzo (a) pyrene	0.0038	5 x 10 <sup>-9</sup>	400,000	20,000	11,000(4)
<u>Volatile Aromatics</u>					
o benzene	1780	95.2	185	9.2	low(4)
o toluene	535	28.7	387	19.3	low(4)
o xylene	180	6.0	752	37.6	low(4)
<u>Phenols</u>					
o phenol	93,000	0.53	24.2	1.2	low(5)
o m-cresol	24,000	0.04	54.1	2.7	--(3)
o 2,4-xyleneol	17,000	0.062	69.6	3.5	--(3)

(1) Calculated by method of Karickhoff, et al, 1979, reported in Lyman, et al (1982).

(2) Calculated at nominal 5% organic carbon content

(3) Undetermined but probably low

(4) USEPA (1980)

(5) USEPA (1979)

**TABLE 2**  
**ESTIMATED HALF-LIVES FOR**  
**VARIOUS ENVIRONMENTAL FATE PROCESSES**  
**IN AQUATIC SYSTEMS**

<u>Class Compound</u>	<u>PROCESS / HALF-LIFE</u>					<u>Bio-degradation</u>
	<u>Photolysis</u>	<u>Oxidation</u>	<u>Hydrolysis</u>	<u>Volatilization</u>		
<u>PAHs</u>						
o naphthalene	---	---	---	---	---	1 day
o fluorene	---	---	---	---	---	---
o anthracene	35 min	1600 days	---	18-300 hrs	---	11.3 hr
o phenanthrene	---	8 x 10 <sup>6</sup> days	---	---	---	---
o benzo (a) anthracene	10-50 hrs	38 hrs	---	90 hrs	---	---
o pyrene	---	1,000 days	---	---	---	---
o chrysene	---	---	---	---	---	---
o benzo (a) pyrene	1-2 hrs	96 hrs	---	22 hrs	---	large
<u>Volatile Aromatics</u>						
o benzene	---	2-50 hrs	---	4.8 hrs	---	slow
o toluene	---	15 hrs	---	5.2 hrs	---	---
o xylene	---	---	---	---	---	---
<u>Phenols</u>						
o phenol	---	---	---	---	---	probably fast
o cresol	---	---	---	---	---	---
o 2,4-xylenol	---	---	---	---	---	---

--- = suspected to be very slow

Source: USEPA (1979)

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TABLE 3  
PRIORITY POLLUTANT WATER QUALITY CRITERIA  
FOR FRESHWATER FISH - PAH COMPOUNDS

	<u>Acute Toxicity</u>	<u>Chemical Toxicity</u>
Acenaphthene	1.7 mg/l	No Data
Flouranthene	3.9 mg/l	No Data
Naphthalene	2.3 mg/l	0.62 mg/l
Total PAH's	No Data	No Data

Source: Federal Register, Vol. 45, No. 231, November 28, 1980.

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TABLE 4  
RESULTS OF SURFACE WATER ANALYSES FOR TOTAL  
VOLATILE AROMATICS<sup>(1)</sup>

Date	Location							
	<u>CSL-1(s)</u>	<u>CSL-1(d)</u>	<u>CSL-2(s)</u>	<u>CSL-2(d)</u>	<u>CLS-3(s)</u>	<u>CSL-3(d)</u>	<u>CSL-4(s)</u>	<u>CSL-4(d)</u>
Oct. 19-21, 1983	ND		ND		ND			
Nov. 11, 1983	ND		ND		ND			
Nov. 18, 1983	ND		ND		ND			
Sept. 17, 1984	0.0027	ND	ND	ND	0.0019	ND	ND	ND
Oct. 9, 1984	ND	ND	ND	ND	ND	ND	ND	ND
<u>Average<sup>(2)</sup></u>	0.0005	ND	ND	ND	0.0004	ND	ND	ND
<u>Average by Station<sup>(2)</sup></u>	0.0004		ND		0.0003		ND	

(1) The only volatile aromatic detected in any sample was benzene.

(2) ND counted as zero in averaging data. Blank indicates no data.

(3) Values reported in mg/l.

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**TABLE 5**  
**RESULTS OF SURFACE WATER ANALYSES FOR TOTAL PHENOLS**

<u>Date</u>	<u>Location</u>							
	<u>CSL-1(s)</u>	<u>CSL-1(d)</u>	<u>CSL-2(s)</u>	<u>CSL-2(d)</u>	<u>CLS-3(s)</u>	<u>CSL-3(d)</u>	<u>CSL-4(s)</u>	<u>CSL-4(d)</u>
Oct. 19-21, 1983	.005		0.157		0.10			
Nov. 11, 1983	ND		ND		ND			
Nov. 18, 1983	ND		ND		ND			
Sept. 17, 1984	0.005	0.006	ND	0.005	ND	0.005	0.005	0.005
Oct. 9, 1984	ND	ND	ND	ND	ND	ND	ND	ND
<u>Average(2)</u>	0.002	0.003	0.031	0.002	0.02	0.002	0.002	
<u>Average Average for station (2)</u>	0.0023		0.023		0.015		0.002	

(1) Values reported in mg/l.

(2) ND counted as zero in averaging data. Blank Indicates no data.

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**TABLE 6**  
**PAH FLOW INTO ERIE BARGE CANAL**  
**FROM THE LOCKPORT COAL TAR SITE**

	Area of Canal Wall Through which PAH Contaminated Ground Water Flows (ft <sup>2</sup> )	Hydraulic Gradient from Site Towards Canal (ft/ft)	Permeability of Material through Which Ground Flows (ft/sec)	Flow Rate of Ground Water Into Canal (ft <sup>3</sup> /sec)	Concentration of PAH's in Ground Water (PPM)	Flux of PAH Contaminants Into Canal (lb/day)
Minimum	10,740	0.085	$8.2 \times 10^{-8}$	$7.5 \times 10^{-5}$	0.1	$4.04 \times 10^{-6}$
Maximum	10,740	0.125	$6.2 \times 10^{-4}$	0.83	6.25	28
Most Probable	10,740	0.1	$10^{-7}$	$1.07 \times 10^{-4}$	2.2	$1.26 \times 10^{-3}$

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TABLE 7

RESULTS OF SURFACE WATER ANALYSES FOR SELECTED  
POLYNUCLEAR AROMATIC HYDROCARBON COMPOUNDS(1)

Date	Location							
	CSL-1(s) (upstream)	CSL-1(d)	CSL-2(s)	CSL-2(d)	CLS-3(s)	CSL-3(d)	CSL-4(s) (upstream)	CSL-4(d)
Oct. 19-21, 1983	ND		ND		ND			
Nov. 11, 1983	0.058		0.45		ND			
Nov. 18, 1983	0.005		0.026		0.03			
Sept. 17, 1984	0.210	3.86	4.11	0.283	0.209	0.186	ND	ND
Oct. 9, 1984	0.021	0.076	0.199	0.065	ND	0.050	ND	ND
Average(2)	0.059	1.97	0.876	0.174	.084	0.118	ND	ND
Average station including shallow and deep	0.604		0.675		0.068		ND	

ND = Not detected

(1) Values reported as sum of benzo (a) anthracene, benzofluoranthene, benzo (a) pyrene, chrysene, fluoranthene, methylbenz (a) anthracene, and pyrene, Units are mg/l.

(2) ND counted as zero in averaging data. Blank indicates no data.

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TABLE 8  
 POTENTIAL CARCINOGENIC PROPERTIES OF  
 PRIORITY POLLUTANT PAH COMPOUNDS

PAH	Carcinogenic Index	
	Jerina, et.al. 1972(1)	NAS, 1972(2)
Acenaphthene	No data	No data
Acenaphthylene	No data	No data
Anthracene	--	--
Benzo(a)anthracene	+	+
Benzo(a)pyrene	++++	+++
Benzo(b)fluoranthene	No data	--
Benzo(k)fluoranthene	No data	--
Benzo(g,h,i)perylene	No data	--
Chrysene	+	+
Dibenzo(a,h)anthracene	++	+++
Fluoranthene	No data	--
Fluorene	No data	--
Indeno(1,2,3-c,d)pyrene	No data	+
Naphthalene	--	No data
Phenanthrene	No data	--
Pyrene	No data	--

Key:       - not carcinogenic  
           + uncertain or weakly carcinogenic  
           + carcinogenic  
           ++,+++,++++ strongly carcinogenic

Sources: (1) Reported in USEPA (1980)  
           (2) NAS, 1972

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TABLE 9  
CARCINOGENIC PAH AS A FRACTION OF  
TOTAL PRIORITY POLLUTANT PAH  
IN MW-8

Sampling Event	Fraction Carcinogenic PAH
November/December, 1983	9.6%
March, 1984	15%
May, 1984	0%
September, 1984	17.9%
October, 1984	5.1%
Average	9.52%

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TABLE 10  
ESTIMATE OF HUMAN EXPOSURE TO  
PAH FROM VARIOUS MEDIA

Source	Estimated Exposure		
	BaP	Carcinogenic PAH <sup>a</sup>	Total PAH
Water	0.0011 ug/day	0.0042 ug/day	0.027 ug/day
Food	0.160-1.6 ug/day		1.600-16 ug/day
Air	0.005-0.0115 ug/day	0.03-0.046 ug/day	0.164-0.251 ug/day
Total	0.166-1.6 ug/day		1.6-16 ug/day

<sup>a</sup>Total of Benzo(a)pyrene, Benzo(j)fluoranthene, and indeno(1,2,3-c,d) pyrene;  
no data are available for food.

Source: USEPA, 1980

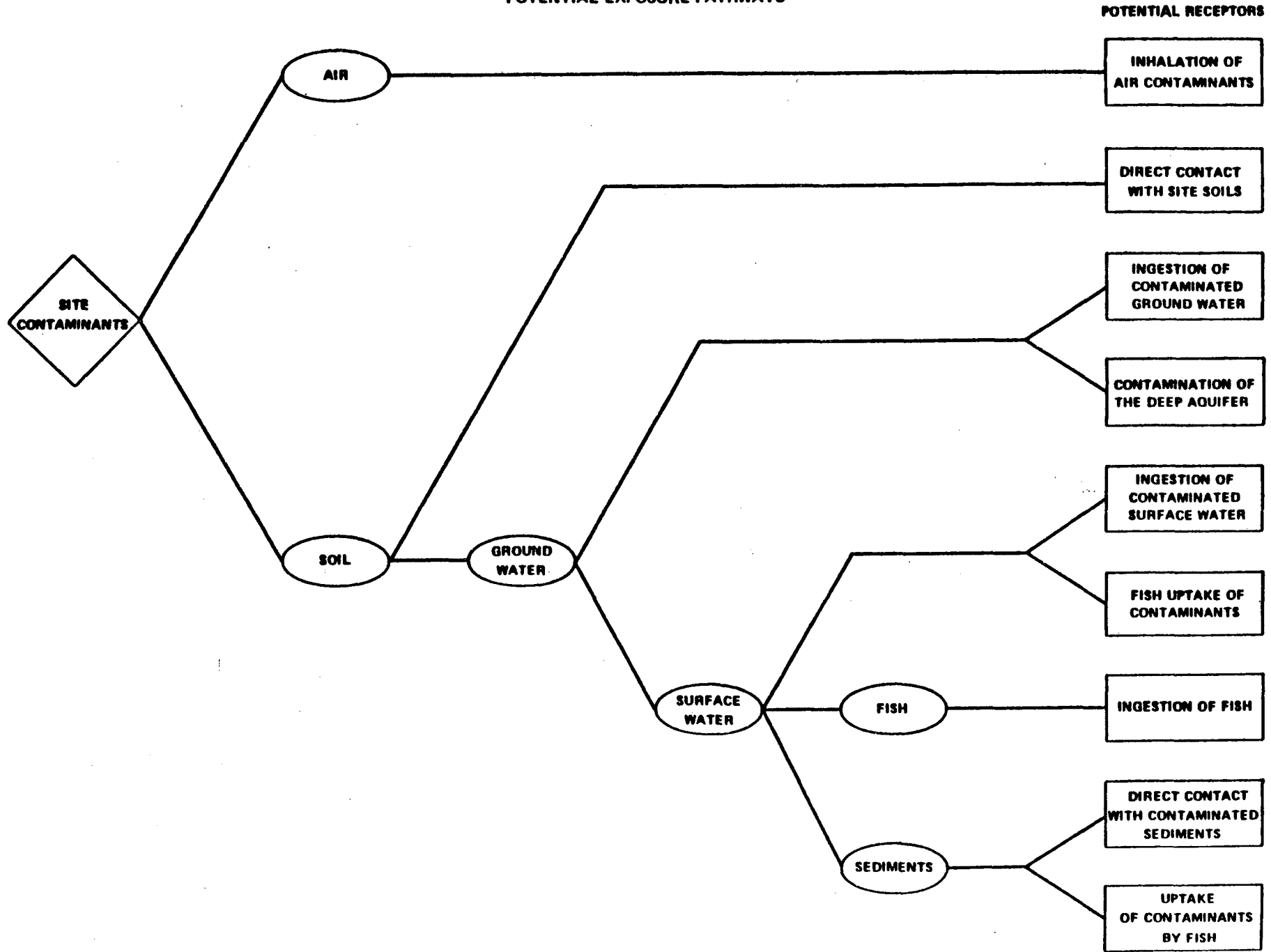
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**Figures**

**FIGURE 1  
POTENTIAL EXPOSURE PATHWAYS**



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