Occidental Chemical Corporation

Olin

Baseline Risk Assessments

Final Report

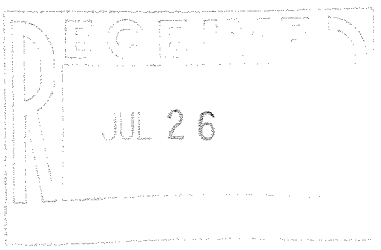
102nd Street Landfill Site
Niagara Falls, New York

July 1990

SIRRINE ENVIRONMENTAL CONSULTANTS

OLIN CORPORATION

OCCIDENTAL CHEMICAL CORPORATION



BASELINE RISK ASSESSMENTS

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SIRRINE ENVIRONMENTAL CONSULTANTS
GREENVILLE, SOUTH CAROLINA

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EXECUTIVE SUMMARY

These baseline risk assessments relate to the 102nd Street Site (Site) in Niagara Falls, New York and were prepared for Occidental Chemical Corporation (OCC) and Olin Corporation (Olin), the current Site owners. Oversight was provided by the United States Environmental Protection Agency (EPA) and the State of New York (State). The Site was used as a disposal site for industrial wastes by both companies or their predecessors. The Site is currently ranked 901 out of 989 sites on the National Priorities List.

The baseline risk assessments were performed to evaluate potential risks to human health and the environment. They are based in part on the information presented in the FS Work Plan prepared by the EPA and the State of New York (December 6, 1988), the Site Remedial Investigation (RI) report prepared for OCC/Olin by Conestoga-Rovers & Associates and Woodward-Clyde Consultants (July 1990), the Feasibility Study prepared for OCC/Olin by Sirrine Environmental Consultants (July 1990), and previous studies undertaken by OCC/Olin.

The public health assessment (PHA) evaluated the exposure routes of drinking water from the Niagara River, swimming in the River, fish consumption, and contact with off-site soils. Direct exposure to materials within the fenced and covered landfill and to groundwater were not evaluated because they do not represent a complete pathway for human exposure under current (baseline) conditions. The PHA determined that neither the individual exposure routes nor the cumulative effects of the Site present any significant risks to human health under current conditions.

The environmental endangerment assessment (EEA) evaluated the exposure pathways involving contact with sediments and surface water containing site-related chemicals and any resultant bioaccumulation effects. Potential exposures to terrestrial organisms were not considered significant because of the small populations in the area of the Site, the existing

cover on the landfill, and the limited extent of the shoreline area. The EEA determined that a limited area of Niagara River sediments posed a potential risk to infaunal organisms, although this risk was not considered significant because of the limited area of impact. Chemical concentrations in the embayment pose no significant risk to the survival or propagation of invertebrates or fish or to animals feeding on aquatic organisms.

1.0 RISK ASSESSMENT FRAMEWORK

The baseline public health and environmental risk assessments have common elements in the hazard identification process. The potential hazards of chemicals detected at the 102nd Street Landfill Site, therefore, are addressed for both human health and environmental concerns in the following section. Potential public health exposures and risks are treated in Section 3.0, and Section 4.0 addresses these elements for environmental concerns. Section 5.0 concludes the baseline risk assessment process with a discussion of all significant Site risks.

Both risk assessments are based upon the data for the SSIs compiled in the Remedial Investigation Draft Final Report (draft RI; Occidental Chemical Corporation and Olin Chemical Group, November 1988) and additional sampling data gathered in response to a request by the regulatory agencies to provide supplemental data on a separate list of assessment chemicals. The basis for selection of SSIs is described in Section 2.2.1 of the Feasibility Study, 102nd Street Landfill Site (FS; Sirrine Environmental Consultants, July 1990). These chemicals were consolidated to form a single list which includes the "chemicals of concern" appearing as Table 2 in the 102nd Street Feasibility Study Work Plan (U.S. EPA and State of New York, December 6, 1988) and pentachlorobenzene. This consolidated list is shown below:

Identified Chemicals

1,1 Dichloroethylene
Trichloroethylene
Benzene
Toluene
Monochlorobenzene
2-Monochlorotoluene
4-Monochlorotoluene
1,2-Dichlorobenzene
1,4-Dichlorobenzene
1,2,3-Trichlorobenzene
1,2,4-Trichlorobenzene
1,2,3,4-Tetrachlorobenzene
Benzo(a) anthracene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
2-Chloronaphthalene
Perchloropentacyclodecane (Mirex)
PCBs as Arochlor 1248
2,3,7,8-TCDD
Other TCDDs
1,2,3,7,8-PeCDD
Other PeCDDs
1,2,3,4,7,8-HxCDD
1,2,3,6,7,8-HxCDD

1,2,4,5-Tetrachlorobenzene
Pentachlorobenzene
Hexachlorobenzene
alpha-Hexachlorocyclohexane
beta-Hexachlorocyclohexane
gamma-Hexachlorocyclohexane
delta-Hexachlorocyclohexane
2,5-Dichloroaniline
3,4-Dichloroaniline
Phenol
2-Chlorophenol
4-Chlorophenol
2,4-Dichlorophenol
2,5-Dichlorophenol
2,4,5-Trichlorophenol
2,4,6-Trichlorophenol
Pentachlorophenol
4-chloro-3-methylphenol
2,4-Dimethylphenol
2-Chlorobenzoic Acid
3-Chlorobenzoic Acid
4-Chlorobenzoic Acid

1,2,3,7,8,9-HxCDD
Other HxCDDs
1,2,3,4,6,7,8-HpCDD
Other HpCDDs
OCDD
2,3,7,8-TCDF
Other TCDFs
1,2,3,7,8-PeCDF
2,3,4,7,8-PeCDF
Other PeCDFs
1,2,3,4,7,8-HxCDF
1,2,3,6,7,8-HxCDF
2,3,4,6,7,8-HxCDF
1,2,3,7,8,9-HxCDF
Other HxCDFs
1,2,3,4,6,7,8-HpCDF
1,2,3,4,7,8,9-HpCDF
Other HpCDFs
OCDF
Arsenic
Cadmium
Mercury
Phosphorus

2.0 HAZARD IDENTIFICATION

2.1 Potential Risk Assessment Chemicals

The primary agency guidance used in this risk assessment was the Superfund Public Health Evaluation Manual (SPHEM, USEPA, 1986) published October 1986. This guidance includes the selection of risk assessment indicator chemicals to identify a manageable list of chemicals that collectively characterize the potential risks associated with a site. The Human Health Environmental Manual published by U.S. EPA in December 1989 (USEPA, 1989c) updated the human health risk assessment guidance. This risk assessment, completed in initial draft by June 1989, is consistent with the guidance.

The hazard parameters of toxicity, mobility, persistence and concentration in various environmental media were considered, following in general the guidelines developed in the

Superfund Public Health Evaluation Manual, to develop a final list of risk assessment indicator chemicals. A SPHEM procedure had been previously used in the RI only to confirm that the selection of SSI's included the primary chemicals of toxicological concern. The present more detailed application confirms the results obtained in the initial RI report.

2.2 Selection of Representative Chemical Concentrations

The 69 chemicals listed in Table 1, including the 16 congeners and homologues of 2,3,7,8 tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) as well as the four categories of non-2378-substituted congeners, are presented with their respective ranges and representative concentrations in various media. The organic carbon partition coefficient (K_{oc}) is also listed for all organics for which this parameter was available in the literature. In general, the sources for the K_{oc} values shown in the table were the RI, the SPHEM or the Hazardous Substance Data Bank (HSDB) in the National Library of Medicine's MEDLARS data base system, in that order of precedence. The ten lowest K_{oc} values (indicative of potentially higher mobility in groundwater) are identified by an L next to their respective values, and the ten highest K_{oc} values (indicative of potentially higher absorption potential to soils and sediments, and bioaccumulation tendency) are identified by an H.

The sources for all representative concentrations for the various media are given in the references at the bottom of Table 1. All sampling data were critically reviewed and the sources selected are those that best describe the Site for the occurrence of a particular chemical or group of chemicals.

Groundwater and Bulkhead Seeps

The groundwater representative concentrations of 28 chemicals (the "SSI" list from the Remedial Investigation) were derived from the means for each chemical from all samples taken from 10 fill and 11 alluvium wells compiled in Tables 11.16 and 11.17 of the RI. All

entries and calculations presented in these tables are described in the RI and are included in the risk assessment by reference.

The data provided both ranges of concentrations and the most representative site-wide concentrations for application to chronic exposure scenarios. Since the risk assessment treats the long-term risks from the entire 102nd Street Site, no attempt was made to consider effects of exposure to smaller site units and any exposure was assumed to include all representative concentrations for each source medium.

The arithmetic mean of means presented in the RI was taken and the procedure used in the RI was followed in deriving representative ground water concentrations. That is, if a chemical was detected at least once in a well, all other sampling events for which that particular chemical was not detected above the quantitation level were assigned a value of one-half the quantitation level and the minimum concentration was set at one-half the quantitation level. If a chemical was not detected for all analyses of all sampling events for a given well, a value of zero was assigned for minimum, most likely and maximum concentrations. It is only in this latter case that a value of zero was used in the calculation of representative concentrations.

Since some groundwater samples indicated a complex mixture of chlorinated dibenzo-p-dioxins (CDDs) and chlorinated dibenzofurans (CDFs), the appropriate representative concentration for this group was derived in accordance with the procedure recommended by the CDD/CDF Technical Panel of the Risk Assessment Forum (Bellin and Barnes, 1987). This involved the application of toxicity equivalence factors that, when multiplied by the concentration of the respective CDD or CDF, expresses that congener in terms of an equivalent concentration of 2378-TCDD based upon structure-activity relationships. The sum of all 2378-TCDD equivalents of CDD/CDF representative concentrations provides a single representative concentration for all CDDs/CDFs detected in given site groundwater samples.

The potential for seepage of chemicals from the bulkhead into the river was evaluated as part of the groundwater pathway. Direct contact with bulkhead seeps or associated surface soils near the bulkhead is considered to be infrequent, so that this potential exposure is not considered to be significant. Therefore this exposure pathway was not developed further.

On-site Soils and Storm Sewer Fill

Sampling data for fill material and soil contaminants on site exist for 2378-TCDD and mercury. These data, given in the soil column on Table 1, are from analyses of on-site soils reported in Table 1.5 (2,3,7,8-TCDD) and Figure 7.4 (Hg) of the RI as referenced in the footnotes of Table 1. For the mercury data all "nondetects" were included at one-half the quantification level. Other than these data for mercury and 2,3,7,8-TCDD, no analysis of on-site surface soils was available. One method by which on-site soil chemical concentrations could be estimated for other chemicals is by analysis of waste survey records. However, the great uncertainty involved in extrapolations from waste surveys (see Table 1) precludes their use in arriving at on-site soil concentrations for other chemicals.

Soil samples also were taken along the storm sewer crossing the Olin property, but these data are considered only representative of the bedding material near the storm sewer and not representative of Site conditions as a whole. Waste disposal summaries describe the chemical contents of fill materials at the Site (Appendix A, FS).

The RI stated that: "The site was covered with topsoil which currently supports a vegetative cover essentially across the entire site" (Section 1.2.2.2) and that the "...full extent of the waste has been covered with imported soil since the early 1970's" (Section 7.4.2). This was confirmed by recent on-site inspections. On-site soils, therefore, pose no significant exposure risk to human health or the environment from direct contact. Likewise, the potential for airborne transport of particulates/vapors is considered minimal under present

conditions because of the imported soil, vegetation cover and the fact that the site is snow covered or wet and less susceptible to airborne transport for six months of the year.

The RI concluded that "the possibility of HNAPL migration offshore in the fill is considered minimal..." and "...the extent of HNAPL migration in the Alluvium beneath the River is considered minimal" (Section 9.5.2). Since there was no defined exposure route, NAPL was not considered further in the risk assessments. NAPL is an indirect exposure route as a source of organic SSI in groundwater. This indirect route was therefore evaluated under the groundwater migration pathway.

Off-site Soils

Off-site soil samples were analyzed for site-related chemicals but these data are not applicable for estimating representative soil concentrations within the fenced landfill area and are not included in this analysis. The chemicals detected off-site are scored separately in Section 3.1 for their respective hazards to off-site exposures.

102nd Street Storm Sewer

The remedial investigation concluded that the storm sewer "is a minor pathway of chemical migration from the Site to the environment" (Section 11.5). Potential risks to human health and the environment would therefore be governed by the overall groundwater discharge, which was evaluated in the baseline risk assessments. The storm sewer was recently resampled and the results are presented in the draft final RI (February 1990). These sampling data are used to evaluate the storm sewer as a separate source of chemicals from the groundwater efflux.

Niagara Sediments

Analyses were performed for a total of 18 chemicals in sediment samples taken from and beyond the embayment of the Niagara River immediately off the Occidental and Olin properties. Although the presence and distribution of these chemicals in the sediment are not uniform and mechanisms of transport vary, particularly between the organics and inorganics, a single line can be drawn beyond which there is no sample evidence for transport from the site of any of the 18 analytes. To derive a representative concentration for the bay sediments, it thus is appropriate to exclude stations from beyond this "clean line" from any averaging of sample results. Background sediment mercury levels lead to an agreed-upon survey level of 200 ug/kg to define potential site-related detects. The survey level for each organic was 100 ug/kg. SPHEM guidance suggests separating the effects of site-related chemicals from those attributable to background concentrations. In order to limit the inclusion of background chemical levels not related to the site, any chemical not detected in a given sample collected during the RI at or above its respective survey level was treated as zero for that sample rather than including that chemical at one-half its background concentration. Background levels were not entirely eliminated since values above the survey levels were not adjusted for background. Following this approach, all sediment sample data were averaged for all stations occurring between the shoreline and the "clean line" established at the furthest extent of detection for site-related contaminants to establish representative concentrations for this medium.

2.3 Scoring of Potential Risk Assessment Chemicals

Of the original list of chemicals, eight were not included in the indicator chemical scoring because they were either not detected in a given medium during the RI or implicated in a significant exposure scenario. These included 1,1-dichloroethylene, 3,4-dichloroaniline, pentachlorophenol, 2,4-dimethylphenol, benzo(a)anthracene, benzo(b)fluoranthene,

benzo(k)fluoranthene and perchloropentacyclodecane (Mirex). With the elimination of these chemicals and the consolidation of the CDDs/CDFs, the list was reduced to 36 chemicals.

The respective toxicity information used for scoring the 36 remaining chemicals is given in Table 2. Data generally were taken from the SPHEM although the toxicological class and EPA category designations were checked against current entries in the EPA Integrated Risk Information System (IRIS). Where toxicity constants were not given in the SPHEM, values from similar chemicals were used if appropriate structure-activity information supported this approach.

Tentative ranks were developed separately for carcinogenic and noncarcinogenic effects in Tables 3 and 4, respectively. Indicator chemical scoring was based entirely upon the product of the groundwater concentrations and water toxicity constants for the respective chemicals, since this data set was the only one complete enough to allow a comparative scoring. CT values for soil and sediment concentrations were included, however, to permit comparisons among chemicals identified in these media for the final selection of indicator chemicals. The tentative ranks are based upon the respective indicator score (IS) values for carcinogens and noncarcinogens with a rank of one representing the highest score for severity of effects in each category.

The chemicals receiving the top 10 rankings for both categories were considered as candidate indicator chemicals in the final selection process. These chemicals are grouped below in order of the ranked severity of their hazard potential within their respective category of effects.

Rank	Carcinogenic Effects	Noncarcinogenic Effects
1	Arsenic	Arsenic
2	alpha-HCCH	Benzene
3	Benzene	Monochlorobenzene
4	PCBs	Mercury
5	gamma-HCCH	1,2,4-Trichlorobenzene
6	Hexachlorobenzene	1,2,3,4-Tetrachlorobenzene
7	beta-HCCH	1,2,3-Trichlorobenzene
8	2,3,7,8-TCCD	Cadmium
9	Trichloroethylene	1,4-Dichlorobenzene
10	2,4,6-Trichlorophenol	4-Chlorophenol

In addition to the scored chemicals from which these 18 candidate indicator chemicals were selected, eight noncarcinogens could not be scored by the method described above due to insufficient data available to establish a comparative toxicity constant. These chemicals include:

delta - HCCH,
2,5 - dichloroaniline,
4-chlor-3-methylphenol,
2-chlorobenzoic acid,
3-chlorobenzoic acid,
4-chlorobenzoic acid,
2-chloronaphthalene, and
phosphorus.

In order to assign relative ranks for these non-scored chemicals an attempt was made to compare them with the scored chemicals by using the standard toxicological criteria given

in Table 5. As shown in this table, standard toxicological endpoints have not been developed for many of the detected chemicals. In order to obtain a toxicity value relevant to human health assessment for which data would be available for at least most of these chemicals, LD50 values were selected.

A relationship between LD50 data and ADIs developed through traditional procedures has been demonstrated in the literature and provides support for their use in a qualitative toxicological scoring process. The work of Layton et al. (1987) shows that an LD50 value estimated from the use of accepted test procedures can be used to compare noncarcinogenic toxicity with less uncertainty than is inherent in the structure activity approach. Layton et al. used log probability plots of the ratios of chronic no observed effect levels (NOELs) and acceptable daily intakes (ADIs) to oral LD50 values (data were derived from the World Health Organization and the Food and Agriculture Organization joint expert panel to establish ADIs for pesticides) to illustrate the application of LD50 values to estimate chronic noncarcinogenic endpoints. The ADIs selected were commonly based on toxicity studies using enzyme inhibition as a measure of toxicity; in 95% of the compounds, this resulted in lower ADIs than would be estimated from studies that address chronic toxic responses directly.

LD50 values (median lethal dose) were obtained from mouse and rat bioassays of toxicants administered by oral and intraperitoneal routes. The data source used was the most current Registry of Toxic Effects of Chemical Substances (RTECS) entries from the MEDLARS database system. These LD50 values are presented in Table 6 for the 10 highest ranked carcinogens and noncarcinogens and the eight non-scored chemicals. Using all appropriate comparative data available, a qualitative judgement was made for each non-scored chemical and relative ranks were determined. This decision process is discussed below for each of the subject chemicals.

delta-Hexachlorocyclohexane - Delta-HCCH was compared with the other HCCH isomers to assure that the highest hazard potential for this group was represented in the final selection of risk assessment indicator chemicals. The groundwater concentration for delta-HCCH is similar to the alpha isomer, but significantly greater than either the beta or gamma isomers. The relevant LD50 value (rat oral) for delta-HCCH is most similar to comparable values for the beta isomer but is one to two orders of magnitude greater (i.e., indicating lower toxicity) than all oral and intraperitoneal mouse and rat LD50s for both alpha and gamma isomers. Because of its low relative toxicity and the additional fact that this is the only isomer of hexachlorocyclohexane that is not classified as a carcinogen, delta-HCCH was not selected as an indicator chemical for risk assessment.

2,5-Dichloroaniline (25-DCA) - The groundwater concentration of 25-DCA is most similar to the respective site concentrations of 2,3,4-trichlorobenzene (234-TCB) and 1,2,3,4-tetrachlorobenzene (1234-TECB). All three chemicals are classified as noncarcinogens. The only relevant LD50 value for 25-DCA (rat oral) is slightly higher than the comparable value for 1234-TECB and an order of magnitude higher than both rat oral and mouse oral LD50s for 124-TCB. 124-TCB and 1234-TECB have tentative indicator score ranks of 5 and 6, respectively. On the basis of the reported LD50s, this rank order is correct for these two chemicals and 25-DCA should be ranked slightly higher (i.e., higher ranking indicating a lesser hazard).

1,2,3-Trichlorobenzene (123-TCB) - is also classified as a noncarcinogen and has an IS rank of 7. The groundwater concentration of 123-TCB is lower but in the same order of magnitude as the other three chemicals. Comparing rat oral (25-DCA) with rat intraperitoneal (123-TCB) LD50s, it appears that 25-DCA is slightly less toxic. Considering, however, 25-DCA's greater groundwater concentration on site (by a factor of 4.5) it should be ranked at least equal to 123-TCB, or with a relative rank of 7. On the basis of this comparison, 25-DCA is included as a candidate indicator chemical.

4-Chloro-3-methylphenol (CMP) - The only other site chemical scored for noncarcinogenic effects that has been detected at a similar groundwater concentration to CMP is hexachlorobenzene (HCB). A rat oral LD50 value given for CMP of 1,830 mg/kg is most comparable to a mouse oral LD50 of 4,000 mg/kg for HCB. The IS rank for HCB noncarcinogenic effects is 19. The next higher concern for noncarcinogenic effects is 2,4,5 trichlorophenol (245-TCP; IS rank = 18). However, 245-TCP is an order of magnitude more concentrated in site groundwater. On the basis of comparable LD50s and water concentrations, CMP should be ranked similar to the noncarcinogenic effects of HCB and, therefore, has not been selected as an indicator chemical.

2-Chlorobenzoic acid (2-CBA) - 2-CBA is selected as a site surrogate for 3-CBA and 4-CBA since it has a higher mean concentration and a broader distribution on site than the other CBA isomers, as would be expected due to its greater solubility. 2-CBA is less toxic than 3-CBA by a factor of about 3 (rat intraperitoneal LD50s), but 2-CBA is an order of magnitude more concentrated in site groundwater. The toxicity of 2-CBA and 4-CBA is similar.

The only other classified noncarcinogen with a site groundwater concentration closely comparable with 2-CBA is 1,4-dichlorobenzene (14-DCB). Since comparable LC50s (rat intraperitoneal) are also very similar for these chemicals, the IS rank of 9 for 14-DCB is taken to apply as well to 2-CBA. In consideration of this relative rank and the surrogate status of 2-CBA, it is included as a candidate risk assessment indicator chemical.

2-Chloronaphthalene (2-CNPH) - 2-CNPH has the lowest reported groundwater concentration of any detected chemical on the original Table 2 list of "chemicals of concern." Although it has a similar reported toxicity to that of 4-chloro-3-methylphenol (rat oral LD50s), 2-CNPH is less concentrated in groundwater by a factor of 2.2. Since this indicates that 2-CNPH is of lower concern than CMP, which already has been rejected as a possible candidate, 2-CNPH will not be included as a risk assessment indicator chemical.

Phosphorus - Although phosphorus in the elemental form is particularly toxic and bioaccumulative, the fact that elemental phosphorus is essentially insoluble in water, and the fact that phosphorus-containing wastes were buried below the water table due to the high reactivity of elemental phosphorus in air (draft RI, p. 1.9), practically preclude reasonable pathways for either human or environmental exposure. The principal problem with soluble forms, particularly phosphates, is not directly related to human health. Because of its role as an essential nutrient, phosphate is a major contributor to the eutrophication process; however, there is no evidence of any significant eutrophication in the Niagara River. The data reported as P in the RI document represents analyses of PO_4 (phosphates) and not elemental phosphorus. Based upon these considerations, phosphorus is not included as a risk assessment indicator chemical.

2.4 Selection of Human Health Assessment Chemicals

The selection process, using the Superfund Public Health Evaluation Manual approach and assessment of certain non-scored chemicals, identified a total of 20 candidate indicator chemicals based upon criteria relevant to human health. These chemicals are listed in Table 7 along with their respective IS ranking and several factors indicative of exposure potentials to facilitate the final selection of indicator chemicals for human health assessment.

The top five ranked chemicals for both carcinogenic and noncarcinogenic effects were selected as final indicator chemicals for human health assessment.

Although 2378-TCDD is present in site media at very low concentrations, it has a very high potency for carcinogenicity as well as high noncarcinogenic toxicity, and potentials for exposure exist through inhalation and fish consumption. Because of these considerations and the high public concern for chlorinated dioxins and furans, 2378-TCDD has been selected as a final indicator chemical.

Cadmium, although present at low levels, was also selected as a final indicator chemical. The primary reason for inclusion is based upon a relatively high potential for carcinogenicity and the need to evaluate the potential for exposure through particulate inhalation.

2-Chlorobenzoic acid was included in the list of final indicators partly due to its assigned status as a surrogate for its other congeners present in site media, but also due to its high potential for mobility through groundwater as shown by its high solubility and low K_{oc} value.

2,4,6-Trichlorophenol has been selected as a final indicator as the most appropriate representative of chlorophenols to assess potential risks from this group. Although it is less concentrated in groundwater than 4-chlorophenol, it has a high potential for mobility through groundwater as shown by its low K_{oc} value, and has a higher measured bioconcentration factor. The noncarcinogenic toxicity of 2,4,6-trichlorophenol is comparable to that of 4-chlorophenol as shown by LD50 values, and it is, in addition, a probable carcinogen of moderate potential.

Hexachlorobenzene has been included in the list of final indicators because it has a relatively high carcinogenicity potential with a medium hazard rank and a high bioconcentration potential.

Dichloroaniline has a relatively high concentration in site groundwater despite the fact that it appears to have a high K_{oc} value and is only slightly soluble in water. Although there is no datum for its concentration in the landfill, the above considerations indicate a relatively high overall site presence. Since dichloroaniline also appears to be similar in noncarcinogenic toxicity to at least five of the other selected indicator chemicals, it also has been added to the list of final indicator chemicals.

Of the remaining candidate indicator chemicals on Table 7, five isomers were represented by appropriate indicator isomers of their respective groups and therefore not included in the final list of risk assessment chemicals.

These chemicals were:

1,4-dichlorobenzene
1,2,3-trichlorobenzene
1,2,3,4-tetrachlorobenzene
beta hexachlorocyclohaxane
4-chlorophenol

This leaves only trichloroethylene, which was not selected due to its relatively low concentration in site groundwater, a low cancer hazard ranking, and a relatively low concern for water and fish consumption.

The final indicator chemicals selected to provide focus on the chemicals of greatest concern for the human health assessment are the following:

FINAL HUMAN HEALTH ASSESSMENT CHEMICALS

Benzene
Monochlorobenzene
1,2,4-Trichlorobenzene
Hexachlorobenzene
alpha-Hexachlorocyclohexane
gamma-Hexachlorocyclohexane
2,5-Dichloroaniline
2,4,6-Trichlorophenol
2-Chlorobenzoic acid

PCBs

2,3,7,8-Tetrachlorodibenzo-p-dioxin

Arsenic

Cadmium

Mercury

2.5 Selection of Environmental Endangerment Assessment Chemicals

"Chemicals of concern" identified through the scoring process discussed in Section 2.1 were evaluated independently to identify a reasonable list of chemicals that would collectively characterize the potential environmental risk to the biota living in or near the 102nd Street Landfill Site environs. The evaluation was carried out using the Quotient Method (Barnthouse and Suter, 1986). This method compares an expected environmental concentration (EEC) with an appropriate toxicological benchmark concentration (BC), calculated as the ratio EEC/BC. This method was used as a screening mechanism to define EECs that are likely to be of concern. Concern levels were identified as follows:

quotient ≤ 0.1	no concern
$0.1 <$ quotient < 10	possible concern
quotient > 10	probable concern

Acute and chronic toxicological endpoints for selected species are shown in Tables 8 and 9, respectively. These were the LC50 for acute effects and the NOEC for chronic effects, as available in the literature for the various species considered. The LOEC and MATC values in Table 9 were retained to provide supplemental information on the toxicity of environmental endangerment chemicals. In addition to the EEC/BC quotients for respective chemicals, the bioconcentration factors (BCFs) also were considered in the final selection of "chemicals of concern" for the environmental endangerment assessment (Table 10).

Bioconcentration data were preferentially selected on the basis of measurements in lipid when specified in the data source. BCFs based on whole body analyses were used when

values on a lipid basis were not available. When no bioconcentration data were found, BCF was calculated from the relationship log BCF = 0.79 K_{ow} - 0.40 (U.S. EPA, 1988a) for respective species. For aquatic species, BCF values greater than 300 are generally considered significant. Those chemicals that pose a possible concern for bioconcentration (BCF > 300, log BCF > 2.48) were included in the list of environmental endangerment assessment chemicals.

Expected environmental concentrations (EECs) for the various chemicals (Table 11) were calculated for sediment pore water and Niagara River water as described in Section 2.3 of the FS. The pore water or sediment/water equilibrium concentration was calculated for those chemicals for which sediment concentrations were reported in the draft RI. Sediment concentration (maximum reported value in Appendix C of the RI) and appropriate partition coefficients (Koc) were used to derive estimated pore water concentrations. Estimated maximum and representative sediment pore water concentrations are presented in Section 2.3, Tables 2.6 and 2.7 of the FS. Surface water concentrations were estimated for the embayment adjacent the landfill based upon waste loadings presented in the RI and Chapter 2 of the FS. Concentrations for the river were generated by a mixing cell model using the river flow between the landfill property lines and Grand Island and the waste loadings as inputs. The flow for the embayment and river, assumptions and characteristics used to derive surface water concentrations are described in the FS, Section 2.3.1.

Screening of the environmental endangerment chemicals of concern was performed for daphnids, fathead minnows and trout. These species were selected as indicator species because they have been determined by EPA to be sensitive indicators for use in toxicity testing. As such, there is a sizeable data base on the toxicity of many chemicals to these particular species. The daphnids were specifically chosen to represent aquatic invertebrate populations. Fathead minnows and trout were selected to represent forage and sport fish, respectively.

The EEC selected was that for surface water in the embayment because these values represented one hundred times higher concentration than those values for the river and, as opposed to sediment pore water EECs, more accurately corresponded to the toxicological benchmarks available from the literature for the indicator species. Both acute and chronic BCs were used in the screening to account for both acute and chronic effects. The BCs for metals were chosen for hard water only (100-200 mg/L hardness as $CaCO_3$). The chronic BCs were for partial or full life cycle tests when available, although BCs for some chemicals and indicator species were from early life stage tests. When chronic BCs were not available they were calculated from acute LC50 values for a specific indicator species. Slooff et al. (1986) have shown an acute/chronic toxicity relationship, determined as log NOEC = -1.28 + 0.95 log LC50, with a 95 percent uncertainty factor of 25.6. Estimated NOECs based on this relationship compared with actual test data from the literature as reported in Tables 8 and 9 showed that this was an acceptable procedure for estimating NOECs when values could not be obtained from the literature. An assessment factor of 0.1 was applied to the lowest NOEC chronic benchmark and a factor of 0.01 to the lowest LC50 acute benchmark for any species (U.S. EPA, 1984b). These factors were applied to reflect the degree of extrapolation from available toxicity data to data appropriate to assessing effects in natural populations.

Results of the screening showed that three of the environmental endangerment chemicals of greatest concern had a quotient (obtained from the EEC/BC ratio) of greater than 10, indicating probable concern. They are benzene, 1,2,4-trichlorobenzene, and gamma-hexachlorocyclohexane. Eight chemicals had calculated quotients above 0.1 to 10, indicating possible concern: monochlorobenzene, 1,2,3-trichlorobenzene, 1,2,3,4-tetrachlorobenzene, 2,5-dichloroaniline, 4-chlorophenol, PCBs, cadmium, and mercury. Based upon these results, for surface water, estimated sediment pore water concentrations appear to be high enough to pose potential risk of acute or chronic effects to organisms living on or in the sediment in the embayment adjacent the landfill. Potential risks from these chemicals including bioconcentration considerations will be addressed in the risk

characterization section. Based on this assessment, the final environmental assessment chemicals are the following:

FINAL ENVIRONMENTAL ASSESSMENT CHEMICALS

Benzene
Monochlorobenzene
1,2,3-Trichlorobenzene
1,2,4-Trichlorobenzene
1,2,3,4-Tetrachlorobenzene
Hexachlorobenzene
gamma-Hexachlorocyclohexane
2,5-Dichloroaniline
4-Chlorophenol
PCBs
2,3,7,8-Tetrachlorodibenzo-p-dioxin
Cadmium
Mercury

2.6 Summary of Risk Assessment Chemicals

The 20 candidate indicator chemicals listed in Table 7 have been evaluated above for both human health and environmental concerns. Selection has been made based on both human health and environmental concerns. The final indicator chemicals predominately represent chemicals of human health concern. These risk assessment chemicals are listed below. The area(s) of primary concern upon which each selection was predicated is also indicated (PC = potential carcinogen; NC = noncarcinogen), as well as significant bioconcentration potentials (BIOC). Three additional chemicals pose a potential environmental risk to aquatic organisms. These are 1,2,3-trichlorobenzene, 1,2,3,4-tetrachlorobenzene and 4-chlorophenol.

RISK ASSESSMENT CHEMICALS

Benzene	-	PC, NC
Monochlorobenzene	-	NC, BIOC
1,2,4-Trichlorobenzene	-	NC, BIOC
Hexachlorobenzene	-	PC, BIOC
alpha - Hexachlorocyclohexane	-	PC
gamma-Hexachlorocyclohexane	-	PC
2,5-Dichloroaniline	-	NC
2,4,6-Trichlorophenol	-	PC
2-Chlorobenzoic acid	-	NC
PCBs	-	PC, BIOC
2,3,7,8-Tetrachlorodibenzo-p-dioxin	-	PC, BIOC
Arsenic	-	PC, NC
Cadmium	-	PC, NC
Mercury	-	NC, BIOC

2.7 Toxicity Profiles for Risk Assessment Chemicals

Toxicity profiles for each of the risk assessment indicator chemicals identified in Section 2.4 are given in Appendix A with selected data given to characterize briefly their toxicity and behavior in biological and ecological systems. Where appropriate, such as in cases of limited data or when an indicator chemical serves as the predominate member of a group of related chemicals, characteristics of other isomers or homologues may be discussed. Relevant chemical and physical data have been presented in Tables 1 and 7, and toxicological data are presented in Tables 5, 6 and 8 through 10. These data are referred to in the toxicity profiles. Additional data are referenced directly or drawn from peer reviewed entries in the Hazardous Substances Data Bank (NLM, 1989).

3.0 Public Health

3.1 Exposure Assessment

3.1.1 Identification of Exposure Routes

As stated in the RI (Section 1.2.2.2), the large majority of the 102nd Street Landfill site has been covered with approximately one foot of clean soil and has been completely grassed to control surface erosion. Visual inspection confirmed that the site presently supports a vegetative cover. Erosion potential is further reduced by the presence of a dike along the entire river frontage of the property. This present condition limits the potential of any atmospheric transport of chemicals or direct contact. Therefore, unless site soils are physically disturbed as in source potential remedial strategies, no determination of potential risk through this route is warranted at this time because of an incomplete exposure pathway.

The presence of site-related chemicals in groundwater is of concern only with respect to mass efflux to the Niagara River. Since net groundwater flow from the site is in the direction of the river, and all residential water use in the surrounding area comes from municipal sources, groundwater access for drinking or other purposes is not a complete pathway and will not be considered in this assessment.

Concern for ingestion of water from the Niagara River with respect to the concentrations calculated from groundwater efflux and storm sewer infiltration (Tables 2.8 and 2.9 of the FS, respectively) will be addressed with respect to the municipal water treatment plant intake approximately 2.5 miles down river and accidental ingestion during swimming events.

Potential exposure due to ingestion of fish containing site-related chemicals is the only food-chain route to human receptors that will be considered, since no agricultural activity is likely

for the immediate area, no irrigation or atmospheric pathway exists, and fisheries for other aquatic species have not been identified.

The baseline public health risk assessment, therefore, will address primarily those potential risks associated with the following possible routes for human exposure:

- ingestion of drinking water from the municipal water treatment plant distribution system;
- absorption due to dermal, ingestion and inhalation routes during swimming events;
- intake via fish consumption from sport and commercial fishing;
- contact with off-site soils containing chemicals potentially derived from the 102 Street Site.

Potential exposure to bulkhead seepage and NAPL is evaluated as part of the groundwater efflux from the site as discussed in the FS, Section 3.2.2. The full extent of waste materials has been covered with imported soil since the early 1970s. The Site currently supports a vegetative cover essentially across the entire Site and is fenced along the whole land-exposed perimeter thereby limiting incidental exposure to on-site soils. All of the surficial soil samples taken within the landfill for dioxin were below detection limits. For these reasons, on-site fill materials do not represent a complete exposure pathway from direct contact or airborne vapors or particulates under current conditions and will not be evaluated under the baseline risk assessment. Although the grassed cover over the contaminated fill of the site properties preclude direct exposures from soil contact at this time, an assessment of potential risk may be developed as needed later in the FS for the evaluation of remedial activities that could disturb the existing cover.

3.1.2 Identification of Potential Human Receptors

Drinking Water Ingestion

The population receiving service directly from distribution from the water treatment plant would be exposed to any residual concentrations of site-related chemicals not removed by the treatment process.

Swimming Event Considerations

Neither estimated chemical concentrations for the Niagara River nor those for the restricted embayment next to the landfill were sufficiently high to trigger concern for acute or chronic toxicity effects on sensitive aquatic species (Section 2.3). Based upon these findings, only the higher set of concentrations calculated for the embayment area warrant performing a risk characterization of exposures due to recreational contact. The population subject to possible swimming contact with water containing the estimated embayment contaminant concentrations (Tables 2.8 and 2.9, FS) is relatively small. The only substantial population in the immediate area is the residents of Cayuga Island located immediately across the Little Niagara River from Griffon Park. The current population of Cayuga Island is 1,228 (Department of Economic Development, Niagara Falls, personal communication, June 13, 1989). Griffon Park, adjacent the site to the west, has been closed to the public since 1986 except for boat launching facilities. It is likely that some recreational boaters would enter the water and only a very small percentage of Cayuga Island residents, since no public beach facilities exist in the area (City of Niagara Falls Chamber of Commerce, personal communication, June 12, 1989).

Fish Consumption

Fish consumption rates vary substantially among subpopulation groups according to age, race, religion and regional factors. 82 Percent of U.S. households eat seafood or fish, based on an analysis of National Marine Fisheries Service data (Longwoods Research Group, 1984). Since consumption of locally caught fish would be regionally restricted, an

estimate of the fish consuming public who might occasionally eat locally-caught fish as part of their diet is taken to include 82% of the combined populations of Niagara Falls including the population of Cayuga Island and the Town of Wheatfield, or approximately 66,000 people.

To provide a conservative estimate of exposure through fish consumption, the sportfishing sales population, including residents of Cayuga Island, was chosen as the exposed population.

Off-Site Soils Contact

The area of off-site soils containing apparently site-related chemicals is restricted to the vicinity of the property boundary, sampling transects FG through L (Figure 2.3, FS), an area that would be accessible to the public walking adjacent to the site along Buffalo Avenue. The population likely to be in this area and that may come into contact with these soils would be persons who habitually walk through this area and is therefore considered to be low. Current occupancy in this area is restricted to one residence to the east of the site property and one residence to the north of the site and south of the LaSalle Expressway. The property to the north is zoned C-1 (retail business), while the property to the east is zoned R-2 (one- and two-family residential). Variability in the composition and chemistry of off-site soils is discussed in the following section and in Section 2.2.2.2 of the FS.

3.1.3 Exposure Point Concentrations

Drinking Water Ingestion

The chemical concentrations in drinking water derived from the municipal water treatment plant intake are taken to be those estimated for the cross section of the Niagara River adjacent to the site and are given in Tables 2.8 and 2.9 of the FS. Chemical concentrations were derived through the use of maximum chemical flux rates as discussed in the FS,

Section 2.3. For the purposes of this risk assessment, no credit is taken for losses that would occur in the river itself or in purification processes in the water treatment plant.

Swimming Event Considerations

Exposures incurred during swimming events would be assumed to be subject to those chemical concentrations given in the FS, Tables 2.8 and 2.9, for embayment surface waters. Derivation of these chemical concentrations is discussed in Section 2.3 of the FS.

Fish Consumption

Individuals consuming fish from the Niagara River may be exposed to concentrations of chemicals in the edible portions of fish tissue. These concentrations are estimated for each chemical from the product of the concentration of that chemical in the Niagara River (Tables 2.8 and 2.9, FS) and the bioconcentration factor (BCF) unique to that chemical (Table 5). Since bioconcentration factors are often derived as averages of tissue loadings, and the highest concentrations are often present in tissues not ordinarily consumed by humans, use of the published BCF values probably represents an overestimate of actual exposure.

Off-Site Soils Contact

Off-site soil samples were taken along primary sampling vectors leading away from the site boundaries along the west, north and east sides. The remedial investigation has indicated that the data obtained may be influenced by high background levels of chemicals, particularly in the case of mercury. In order to derive a representative concentration of site-related chemicals, the significant vectors of probable transport from the site were first identified, and then the two highest concentrations were averaged from those sample groups that showed progressively decreasing concentrations with distance from the site along these vectors. The selection of vectors showing clear indication of decreasing chemical concentration away from the site permitted focus on the highest levels of site-related chemicals. With the exception of mercury, the highest concentrations of all

chemicals assumed to be site-related that were scored as significant site chemicals occurred at sample locations near the site boundary along vectors I and J to the north of the site.

Interim corrective measures have been taken for the purpose of preventing inadvertent contact with soils containing 2,3,7,8-tetrachlorodibenzo-p-dioxin. 2378-TCDD was detected above the action level of 1 ug/kg in only two samples (5.2 and 2.2 μg/kg at vectors I and J, respectively). Other isomers of dioxin also were present in Griffon Park samples. These two areas, coincident with the highest concentrations of all other site-related chemicals of human-health concern except mercury, were covered with clean fill and gravel, thus limiting exposure potentials under baseline conditions. The locations of higher concentrations observed for mercury are not consistent with the migration trends observed for all of the remaining site-related chemicals. The RI (Section 7.4.2) stated that the dispersal of mercury and organic SSI off-site were not well-correlated. While the single highest mercury concentration in soil was found on-site, the remaining on-site concentrations were within the range of mercury concentrations found off-site. The RI concluded that the Site is only one possible source of mercury. Other sources for mercury in the area of highest off-site soil concentrations is believed to be related to off-site disposal and handling of wastes by others, as stated in the RI document.

Therefore, representative concentrations of site-related indicator chemicals contained in off-site soils were developed from concentrations detected in accessible areas along the significant vectors of probable transport as described above. This area extends along Buffalo Avenue from just west of the boundary of Griffon Park at about 96th Street to just west of the western boundary of the Olin Corporation property at about 99th Street. All detects within this area were averaged for the organic indicator chemicals except for three sample locations that showed significantly increasing concentrations of chemicals with distance from the site. To derive a representative concentration for mercury, only those samples that coincided with sample locations showing significant site-related concentrations

for all other indicator chemicals were averaged. The final representative concentrations for indicator chemicals found in off-site soils, along with the number of detects included in the calculations, are given in Table 13.

3.1.4 Development of Exposure Profiles

A conservative approach per SPHEM (1986) guidance was used to develop the following exposure profiles. Common parameter values for chronic human exposure are given in Table 14, and specific exposure parameters applicable to swimming events are given in Table 15. Exposure profiles for the indicator chemicals identified in Section 2.4 are developed separately for the three chemical release sources: ground water efflux, storm sewer infiltration, and off-site soil.

Ground Water Efflux

All of the indicator chemicals were detected in the ground water. Exposure point concentrations for the following exposure profiles are based on efflux of ground water from the site into the Niagara River and the embayment.

Benzene

Drinking Water Pathway

Exposure due to the drinking water pathway is calculated by:

$$\text{Daily Intake (mg/kg/day) from drinking water ingestion} = \frac{C_w \times R_i}{W_a}$$

where

C_w = concentration of chemical in water (mg/l)
R_i = rate of ingestion (l/day)
W_a = adult weight (kg).

Thus, the benzene intake from water taken from the Niagara River due to efflux from the 102nd Street Site is given by:

$$\text{Intake from Drinking Water Ingestion} = \frac{5.13 \times E\text{-}6 \text{ mg/l} \times 2 \text{ l}}{70 \text{ kg}}$$

$$= 1.47 \times 10^{-7} \text{ mg/kg/day.}$$

Swimming Absorption Pathway

Since the concentrations in the water of the off-site embayment are very low, no significant inhalation pathway exists, and, therefore, only accidental ingestion and dermal absorption will be considered potential intake routes. Swimming is assumed to occur 25 times per year, or 0.0685 times per day, for a total of 65 hours per year.

Accidental ingestion while swimming can be calculated according to the following equation:

$$\text{Daily Intake (mg/kg/day) from Accidental Ingestion} = \frac{C_w \times R_i \times D_h \times f_d}{W_a}$$

where

C_w = concentration of chemical in water (mg/l)
R_i = rate of ingestion (l/hr)
D_h = duration of exposure (hours/event)
f_d = daily frequency (events/day)
W_a = adult weight (kg).

Thus, average daily intake of benzene due to accidental ingestion while swimming near the 102 Street Landfill is calculated by:

$$\text{Intake from Accidental Ingestion} = \frac{5.13\text{ E-4 mg/l} \times 0.05\text{ l/hr} \times 2.6\text{ hr} \times 0.0685\text{ events/day}}{70\text{ kg}}$$

$$= 1.90 \times 10^{-8}\text{ mg/kg/day.}$$

Dermal absorption while swimming can be calculated according to the following equation:

$$\text{Daily Intake (mg/kg/day) from Dermal Absorption} = \frac{C_w \times K_p \times D_h \times f_d \times S}{W_a}$$

where

C_w = concentration of chemical in water (mg/l)
K_p = chemical-specific dermal permeability constant (l/cm^2-hr).
D_h = duration of exposure (hrs/event)
f_d = daily frequency (events/day)
S = skin surface area exposed (sq cm).

In this application, it is assumed that the chemicals are carried through the skin as a solute in water, and that the permeability rate for water, 8.0×10^{-7} l/cm^2-hr, is the rate controlling parameter (S.T. Hwang, personal communication, February 9, 1989).

Thus, average daily intake of benzene due to dermal absorption while swimming near the 102 Street Landfill is calculated by:

$$\text{Intake from Dermal Absorption} = \frac{1.53\text{ E-4 mg/l} \times 8.0\text{ E-7 l/cm}^2\text{-hr} \times 2.6\text{ hr} \times 0.0685\text{ events/day} \times 18{,}150\text{ cm}^2}{70\text{ kg}}$$

$= 6.53 \times 10^{-8}$ mg/kg/day, and the total daily intake due to swimming is

8.43×10^{-8} mg/kg/day.

Fish Consumption Pathway

The tissue load for site-related chemicals in fish may be reasonably assumed to be derived from river water concentrations calculated for the cross section of the Niagara River opposite the 102nd Street Landfill site (Table 11). The bioconcentration factors used for tissue load calculations are those listed in Table 5. Exposure to site-related chemicals via fish consumption is based on the sportfishing subpopulation to provide a conservative estimate of potential intake. Fish consumption, based on Rodricks (1985), is assumed to be 14 grams per day for the average sport fisherman in the Great Lakes area. The most recent angler survey conducted by the NYSDEC Bureau of Fisheries shows that sport fishermen utilizing the Lake Erie-Niagara River-Lake Ontario system expend only 7% fishing effort on the entire Upper Niagara River and shore and pier fishing represents only 44% of this effort (NYSDEC, 1984). A reasonable case assumption was made that 10% of the average 14 g/day consumption was derived from the local site-related source.

From these data, chemical intake from ingestion of fish, assuming 100% absorption from the gastrointestinal tract, can be calculated by:

$$\text{Daily Intake (mg/kg/day) from Fish Consumption} = \frac{C_w \times BCF \times R_i}{W_a}$$

where

C_w = concentration of chemical in water (mg/l)
BCF = bioconcentration factor (l/kg)
R_i = rate of ingestion (kg/day)
W_a = adult body weight (kg).

Thus the average daily intake of benzene derived from the 102nd Street Landfill through the food chain pathway is calculated by:

$$\text{Intake from Fish Consumption} = \frac{5.13\text{ E-6 mg/l} \times 5.2\text{ l/kg} \times 14\text{ E-4 kg/day}}{70\text{ kg}}$$

$$= 5.34 \times 10^{-10}\text{ mg/kg/day}$$

Thus, from the sum of the daily intakes through all exposure routes, the total chronic daily intake of benzene derived from the 102nd Street site is estimated to be 2.32×10^{-7} mg/kg/day.

Exposures profiles for the remaining chemicals are derived following the procedures described above. The remaining ground water efflux exposure profiles are presented in Appendix B.

Storm Sewer Infiltration

Exposure profiles are developed for each indicator chemical detected in the storm sewer bedding. Exposure point concentrations for the river and the embayment are based on storm sewer infiltration discharge from the site. These exposure profiles, derived following the same procedure illustrated for benzene (above), are presented in Appendix C. Exposure point concentrations can be found in the FS (Section 2.3).

Off-Site Soils

Additional exposures must be evaluated for individuals who may come into contact with off-site soil chemicals. It is expected that the population of potentially exposed individuals, those persons habitually walking adjacent to the site along Buffalo Avenue, would be small and that chronic exposure over a lifetime would be unlikely. Nevertheless, for the indicator chemicals that were detected in accessible areas and that are assumed to be of site origin (Table 13), the following calculations estimate exposure potential for incidental ingestion, dermal absorption and particulate inhalation.

Exposure due to vaporization of site-related chemicals in off-site soils is considered to be minimal. Since the primary area of soils containing site-related chemicals, between Buffalo Avenue and the 102nd Street site, is only about 30 feet wide, there is considered to be no possibility for a residence to be built on this site in the future. For this reason, the average daily exposures will be estimated based on an assumption of two visits of one hour duration per week to account for casual visits or walks through the area. These estimates are considered to represent a reasonably conservative scenario. A child, if allowed to play next to Buffalo Road, might spend one hour in the area of maximum contamination. If this time (2 hours per week) was spent by adults while walking, say, to a grocery, it would translate to about 24 one-way walks per week, assuming 5 minutes to pass the site. This accounts for the assumption that two visits of one hour duration per week would account for casual visits or walks through the area. The approach of assessing exposure to maximally exposed individuals, those most likely to have direct contact with the area of highest concentrations of off-site soil chemicals, is considered to be sufficiently protective for individuals living in the residences or occupying the commercial facility described in Section 3.1.2. Note that conservatism is built in because no change of residence is considered over the lifetime of the exposed individual, although the EPA Exposure Factors Handbook estimates 9 years as the average length of residence and 30 years as the reasonable upper bound of residence time.

1,2,4-Trichlorobenzene

Some skin penetration may be expected due to contact with 1,2,4-trichlorobenzene. Estimation of intake due to skin absorption of site-related chemicals from soil is based upon a model developed by Hawley (1985) as elaborated by Rosenblatt and Spinney (1986). The calculation of average daily intake is based upon a 10 kg child (a conservative estimate for average daily exposure over a lifetime) and that the chemical contents of only 38.6 mg of soil would be absorbed in a day (assuming that as much as 5,100 mg/M^2 might be loaded on the skin, that a child's exposed skin area is 0.21 M^2, that 24% of a pure compound is absorbed by the skin in a 24-hour period, and that only 15% of that amount

would be absorbed from the soil). A 10 kg child is used for this scenario to be consistent with the skin absorption model developed by Hawley, whereas the 15 kg child's weight was used for the soil ingestion scenario for consistency with EPA's model for soil ingestion.

In the case of skin absorption, it was assumed that the soil would not be immediately washed from the skin after the direct contact period. This would allow continued absorption from residues remaining on the body. Therefore, the previous estimate of exposure duration used for the soil ingestion scenario (an average lifetime value of 0.286 hours per day) is doubled to provide a safety factor for the fact that soil residues may not be immediately washed from the skin.

Thus, the average daily intake due to skin penetration in this scenario is calculated by,

$$\text{Daily Intake (mg/kg/day) from Skin Absorption} = \frac{C_s \times A_s \times D}{W_c}$$

where

C_s = concentration of chemical in soil (mg/kg)

A_s = absorption of soil by child (kg/hr)

D = duration of exposure (hrs/day)

W_c = weight of child (kg)

Thus, for 1,2,4-Trichlorobenzene,

$$\text{Intake from Skin Absorption} = \frac{0.141 \times 1.608E\text{-}6 \times 0.571}{10}$$

$$= 0.141 \times 9.182E\text{-}8$$

$$= 1.30 \times 10^{-8}$$

Soil ingestion is calculated by considering the respective ingestion rates for children of 1 to 6 years and adults (Table 14) and deriving proportional intake estimates representative of a chronic lifetime exposure. For a child, the proportional exposure is 6 of 70 years or 0.086; for an adult the factor is based on 64 years of exposure over a 70 year lifetime or 0.914. The average exposure due to incidental soil ingestion is calculated by:

$$\text{Daily Intake (mg/kg/day) from Soil Ingestion} = \frac{(C_s \times R_{ic} \times D \times 0.086)}{W_c} + \frac{(C_s \times R_{ia} \times D \times 0.914)}{W_a}$$

where

C_s = concentration of chemical in soil (mg/kg)
D = duration of exposure (hours/day)
R_{ic} = ingestion rate of child (kg/hr)
W_c = weight of child (kg)
R_{ia} = ingestion rate of adult (kg/hr)
W_a = weight of adult (kg).

Thus,

$$\text{Intake from Soil Ingestion} = \frac{0.141 \times 8.33\text{ E-}6 \times 0.286 \times 0.086}{15} + \frac{0.141 \times 4.17\text{ E-}6 \times 0.286 \times 0.914}{70}$$

$$= (0.141 \times 1.366\text{ E-}8) + (0.141 \times 1.557\text{ E-}8)$$

$$= 4.12 \times 10^{-9}\text{ mg/kg/day}$$

The calculation for dust inhalation is based upon breathing rate and the concentration of particulates in the air. The latter value is taken from total suspended particulate matter measurements averaged over a period of three years for a monitoring station 500 m from an inactive landfill, the Hyde Park Landfill, immediately to the east of the 102nd Street site (Hawley, 1985). These data were selected because of the proximity of the air monitoring station to the 102nd Street site. The airborne particulate concentration assumed is considered conservative because the Hyde Park is surrounded by heavy industries that

would emit particulate loads, whereas the 102nd Street Site with no heavy industry nearby and wind direction typically from the river would be expected to exhibit lower airborne particulate levels. Taking the conservative assumption that the total atmospheric loading of dust at breathing levels derives from the area of highest site-related chemical concentrations for off-site soils,

$$\text{Daily Intake (mg/kg/day) from particulate inhalation} = \frac{C_s \times C_a \times R_b \times D}{W_a \times 1E+6}$$

where

C_s = concentration of chemical in soil (mg/kg)

C_a = dust concentration in air (mg/m^3)

R_b = breathing rate of adult (m^3/hr)

D = duration of exposure (hours/day)

W_a = weight of adult (kg).

thus,

$$\text{Intake from Particulate Inhalation} = \frac{0.141 \times 0.07 \times 0.77 \times 0.286}{70 \times 1E6}$$

$$= 0.141 \times 2.202E\text{-}10$$

$$= 3.11 \times 10^{-11} \text{ mg/kg/day}$$

Thus, the total chronic intake from 1,2,4-trichlorobenzene derived from off-site soil concentrations is estimated to be 1.72×10^{-8} mg/kg/day.

The remaining exposure profiles are presented in Appendix D.

3.2 Risk Characterization

3.2.1 Identification/Development of Health-based Endpoints

The health-based endpoints appropriate to a determination of risk potential are the cancer potency slope and the acceptable chronic intake (AIC) values given in Table 5. For the 13 chemicals selected as indicator chemicals for risk assessment, respective endpoint values are available for all but 2,5-dichloroaniline (25-DCA) and 2-chlorobenzoic acid (2-CBA).

The most appropriate toxicity datum found for 25-DCA is an oral LD50 of 2,900 mg/kg for the rat (Table 6). Using this LD50 value, we can estimate an acceptable chronic intake after the method of Layton et al. (1987) by multiplying by a factor of 1.5×10^{-6} to obtain an AIC of 4.35×10^{-3} mg/kg/day. This relationship was derived by comparing Acceptable Daily Intakes (ADIs) developed by the World Health Organization and Food and Agriculture Organization (WHO/FAO) expert committee for 96 pesticides and associated LD50 values. The WHO/FAO ADIs were developed by the standard approach of a toxicological evaluation, the identification of an animal no effect level (NOEL), and the selection of a safety factor to extrapolate the safe intake for the animal to a safe intake for humans. An additional safety factor is implicit in the calculation since the ADIs selected were commonly based on toxicity studies using enzyme inhibition as a measure of toxicity; in 95% of the compounds, this results in lower ADIs than would be estimated from studies that address chronic toxic responses.

Similarly, the most appropriate toxicity datum found for 2-CBA is an intraperitoneal LD50 of 2,300 mg/kg for the rat (Table 6). Using the method of Layton et al. (1987), an AIC of 3.45×10^{-3} is calculated.

3.2.2 Characterization of Risk of Harm to Human Health

Noncarcinogenic Effects

Risk from individual noncarcinogenic chemicals is derived by comparing the sum of the intakes for all pathways with the acceptable chronic intake for that chemical. To assess the overall risk from multiple chemicals the hazard index (the sum of the ratio of the estimated intake to the accepted chronic intake for all indicator chemicals) is used (USEPA, 1986). A hazard index exceeding unity indicates that there may be a concern for potential health risk.

The risk characterization for noncarcinogenic indicator chemicals is summarized in Table 16. The sum of intakes for each chemical is shown to be at least three orders of magnitude below the acceptable chronic intake (AIC) level that would indicate a potential adverse effect. Furthermore, assuming additivity of effects, the sum of the ratios of the subthreshold exposures to acceptable exposures gives a hazard index of only 2.3×10^{-3}. On the basis of this characterization, it is concluded that there is no significant risk of harm to human health due to noncarcinogenic effects of chemicals from the 102nd Street Site.

Carcinogenic Effects

Table 17 summarizes the route- and chemical-specific risks for each of the eight indicator chemicals selected because of carcinogenic concerns. The combined upper bound risk for additive concerns is estimated at 1.54×10^{-5}, or a chance of 1.5 excess cancers in a population of 100,000 over a 70-year period. USEPA guidance (USEPA, 1986) requires that remedial alternatives reduce total potential carcinogenic risks to individuals to a target level of 10^{-4} to 10^{-7}. Although there is much discussion among regulatory agencies over what constitutes an unacceptable level of cancer risk, a recent analysis of 132 regulatory decisions by Federal agencies (Travis et al., 1987) indicates that a lifetime cancer risk

greater than 1 x 10^{-3} is considered to provide cause for regulatory concern under nearly all circumstances.

4.0 Environmental Endangerment

4.1 Exposure Assessment

4.1.1 Identification of Exposure Routes

Birds and small mammals have been observed on the landfill and a small number of these could potentially nest or live on the site. The landfill has been covered with clean soil material and is presently under grass cover. Birds visiting the area are likely to remain for only brief periods and exposure to soils containing site-related chemicals is not likely under present site conditions. Small animals that burrow, such as rabbits, could potentially be exposed to these soils; however, there was no evidence of extensive populations of burrowing wildlife on the site during a recent site reconnaissance. Because of the limited likelihood of significant exposure of wildlife to soils containing site-related chemicals, this exposure pathway is considered to be insignificant and will not be considered further in the endangerment assessment.

The small (approximately 0.6 acre) lowland area on the property bordering the river presents the only area for potential exposure of terrestrial organisms to site-related chemicals in soils. Because of the small size of this area, it is not considered to be a significant pathway for exposure and will not be further considered in the baseline endangerment assessment. This lowland area does not meet the State wetland criteria of 12.4 acres for size and is not considered to be of unusual local importance (Personal Communications, Mr. Richard Foley, NYSDEC - Regulatory Affairs; Mr. Jim Snyder, NYSDEC - Fish and Wildlife). The limited habitat available would preclude significant utilization of this area for nesting, foraging or other use by wildlife in the vicinity.

There is no permanent surface water on the landfill, therefore potential for significant exposure of birds, waterfowl or other wildlife to site-related chemicals via this pathway does not exist.

The most significant pathways for exposure to site-related chemicals are through contact with sediments in the river immediately adjacent to the landfill shoreline and indirectly through contact with groundwater entering the river from the site or by consumption of organisms that have accumulated residues from exposure to sediment or to groundwater leaving the site. Therefore, the endangerment assessment will focus on exposure via contact with sediment and surface water (groundwater from the site diluted by river water) and address the potential effects of bioaccumulation.

4.1.2 Identification of Potential Environmental Receptors

Based upon the exposure assessment, potential environmental receptors are considered to be the aquatic invertebrates living in or on the sediments immediately adjacent to the landfill, fish that may frequent or reside in the shallow embayment adjacent the landfill and waterfowl that may rest or forage in the waters of the embayment.

Potentially exposed invertebrates would include the infaunal invertebrates, planktonic crustaceans and algae, shellfish and crayfish. Submerged aquatic plants and associated epifauna could potentially bioaccumulate site-related chemicals although submerged vegetation was not observed in the embayment.

Important recreational fish in the area include smallmouth bass, perch, muskies and possibly walleye. Some trout and salmon could occur seasonally in the area, depending upon water temperature. Forage fish such as emerald shiners also would be present in the area.

The most notable waterfowl frequenting the area year-round is considered to be herring and ringbill gulls. There is a major concentration of diving ducks, utilizing this segment of the river during winter (October through March/April). These include the Canvasback, Goldeneye, Buffle-Head, and Greater Scaup. Surface feeding ducks present during winter are principally Mallards and Black duck. The major population of fish-eating ducks consists of Common Mergansers.

Based upon site visits, conversations with NYSDEC biologists and findings from the ecological survey of the Hooker Chemical Center (Spotila et al., 1985), the presence of threatened or endangered species is considered unlikely. Therefore, no threatened or endangered species is expected to experience any significant exposure to site-related chemicals. The Bald Eagle migrates through the area, but there is no known nesting in the area of the site and the eagles predominately utilize the river below the falls.

Risk characterization for every potentially exposed organism is impractical because of the lack of adequate, consistent toxicological data for all organisms. Sensitive indicator species, such as the invertebrate Daphnia, were therefore used as surrogate species to represent the potentially exposed populations. Rationale for the selection of these indicator species is discussed in Section 2.5.

4.1.3 Exposure Point Concentrations

The expected exposure concentrations for organisms within the sediment, the embayment and the river are shown in Table 11. None of the estimated concentrations for the risk assessment chemicals (Section 2.5) are high enough to reach concern levels for acute or chronic effects in aquatic organisms potentially exposed to waters of the embayment or river. The criteria used are NYSDEC ambient water quality standards and guidance values or the USEPA chronic criteria when NYSDEC criteria were not available. Therefore exposure due to contact is not expected to present a significant risk to these organisms.

Exposure point concentrations, for those site-related chemicals that were detected in sediments and for which environmental concentrations could be calculated, could be high enough to pose a risk to infaunal or epibenthic organisms due to chronic exposure. This potential risk will be addressed in risk characterization for those chemicals and organisms for which sufficient appropriate toxicological endpoints are available.

To characterize the risk to fish and waterfowl from the consumption of food organisms potentially containing site-related chemicals, bioaccumulation factors will be applied to expected environmental concentrations. Projected residues will then be compared with available dose-response information in order to characterize the potential risk to consumers.

4.2 Risk Characterization

4.2.1 Identification of Environmental-based Endpoints

The environmental-based endpoints selected to characterize potential risk to potentially exposed aquatic biota were the NYSDEC ambient water quality standards and guidance values or the USEPA chronic criteria when NYSDEC criteria were not available (NYSDEC, 1987). These values were chosen to characterize potential risk because they represent "safe" values for the protection of fishing and fish propagation and were promulgated for the protection of aquatic life.

The environmental-based endpoints for the selected risk assessment chemicals are contained in Table 18 along with corresponding expected environmental concentrations for surface water of the embayment and sediment pore water. These endpoints represent concentrations at which no significant chronic effects are expected to occur from exposure to the selected site-related chemicals. Published criteria levels were not obtainable for 2,5-Dichloroaniline or 2-Chlorobenzoic acid.

The risk to aquatic biota from potential bioaccumulation of selected site-related chemicals will be evaluated through the comparison of bioconcentration factors (BCFs) and persistence of respective site-related chemicals according to methodology recommended by the USEPA Office of Toxic Substances Testing Triggers Workshop (Life Systems, Inc., 1983). Based upon criteria recommended to trigger toxicological testing of chemicals under TSCA, the environmental-based endpoint will be calculated as the product of BCF x Persistence. The use of bioconcentration and persistence combined provides a better estimate of potential risk then either parameter along because there is no single specific point at which either phenomenon individually becomes a singular reliable criterion for assessing potential risk from exposure. Persistence is expressed as half-life of the chemical in days. Values for these parameters and their products are contained in Table 19. The risk criteria (R) used will be:

The upper bound of the ranges (1,200) and 14,000) represent BCFX Persistence products of 300 x 4 and 1000 x 4, respectively.

R	=	1-1,200	No Risk
R	=	1,200-14,000	Possible Risk
R	=	>14,000	Probable Risk

The potential risk to wildlife (ducks and gulls) from biomagnification of site-related chemicals through the food chain was evaluated on the basis of available fish flesh criteria for piscivorous wildlife (NYSDEC, 1987) contained in Table 19. Exceedance of these criteria suggests that the potential exists for toxic effects from consumption of fish containing site-related chemicals although the expression of toxic effects would depend upon the magnitude and duration of dose received.

4.2.2 Characterization of Risk of Harm to Environmental Populations

Estimated environmental concentrations (EECs) in the embayment water pose no significant risk to survival or propagation of invertebrates or fish residing in, frequenting or passing through the embayment. The EECs are one to several orders of magnitude below "safe" levels for ambient water quality based upon NYSDEC and USEPA criteria (Table 18). According to the endpoints from which these criteria were derived such as bioconcentration, propagation and food tainting as defined in Table 18, trichloroethylene, benzene and 2378-TCDD pose no risk for significant bioconcentration and the chlorinated phenols are below levels expected to cause aquatic food tainting.

The concentration of site-related chemicals derived from storm sewer discharge were estimated (FS, Table 2.9). Embayment concentrations from storm sewer contribution were three to five order of magnitude lower than those from groundwater efflux. Therefore the relative contribution from storm sewer discharge is no considered to represent any significant risk compared to that from ground water efflux.

EECs for sediment pore water do exceed ambient water quality criteria for those site-related chemicals for which concentrations could be estimated (Table 18). The EEC of 32 ppb for hexachlorobenzene is essentially equivalent to the USEPA criterion of 30 ppb for chronicity. The total estimated concentration of chlorinated phenols (4-CP and 246-TCP) is 40 times greater than the criterion value of 1 ppb. It should be noted that the pore water EECs were derived from maximum sediment concentrations measured and were not derived on the basis of lower estimated "representative" concentrations. The remaining pore water EECs exceeded criteria values by several orders of magnitude and therefore are considered to pose a potential risk to propagation and possibly survival to infaunal organisms. However, the extent of detected site-related chemicals was restricted to the shoreline adjacent the landfill (Figure 1). This area represents only approximately three percent of the riverbed between the landfill shoreline and Grand Island. Because of the limited extent

of this area, the overall risk to Niagara River infaunal and epibenthic populations living outside this area is not expected to be significant.

The interrelation of BCF and persistence for the selected site-related chemicals showed that 124-TCB, alpha-HCCH, and 246-TCP have a possible risk for bioaccumulation, while hexachlorobenzene, PCBs and 2378-TCDD exhibit characteristics of bioconcentration factor and chemical persistence that would place them in a probable risk category (Table 19). All three of these site-related chemicals exhibit relatively large BCFs and both hexachlorobenzene and 2378-TCDD are quite persistent in surface water (Tables 7 and 12). The relationship of a chemical's BCF and environmental persistence was developed to screen potential chemicals for further testing under TSCA and does not per se indicate that exposure to the chemical would result in significant toxic effects in the environment. As discussed above, the estimated environmental concentration for 2378-TCDD did not exceed the NYSDEC criteria level based on bioconcentration and significant risk from bioconcentration is, therefore, not expected to occur at concentrations present in the embayment.

Chemical residues in trout were estimated by multiplying expected environmental concentrations in the river by the bioconcentration factor for trout. These were compared with fish flesh criteria for piscivorous wildlife developed by the NYSDEC (December, 1987). The estimated residues for trichlorobenzene, hexachlorobenzene, hexachlorocyclohexane, PCBs and 2378-TCDD were all below the risk criteria for non-carcinogenic effects on wildlife (Table 20). Because of the similar magnitude between carcinogenic and non-carcinogenic criteria estimated residues also would be below the criteria for carcinogenic effects. Therefore the risks to wildlife utilizing food resources (fish) that reside in this section of the river appear to be limited.

4.2.3 Community Effects

The survival and propagation of planktonic, forage fish and game fish communities within this section of the river are not expected to be significantly affected by exposure to estimated baseline concentration of site-related chemicals. Because the risk of harm to infaunal organisms affects only a very restricted population, the presence of site-related chemicals in the embayment should not significantly affect the maintenance of a balanced aquatic ecosystem. At the estimated environmental concentrations used in this endangerment assessment, aquatic communities in this section of the river are not expected to be at significant risk from bioaccumulation of site-related chemicals nor are wildlife utilizing food resources (fish) that reside in this part of the river expected to be at significant non-carcinogenic risk from consumption of forage fish.

5.0 Potentially Significant Site Risks

From Table 17 it is apparent that the highest estimates of risk due to potential carcinogenic effects are associated with direct contact with off-site soils. Since there are only two occupied residences and three operating businesses in the immediate vicinity of the 102nd Street Site, the calculated cumulative risk of 1.54 excess cancers in a population of 100,000 would apply to an actual population probably not more than 25 people. This represents 0.001 excess cancer over a 70-year period among this limited population. Among the larger area population that would not come into direct contact with off-site soils, the additive cancer risk from all other routes of entry is estimated at 1.02×10^{-7}. This level of cancer risk is three orders of magnitude less than the de minimis level derived from an analysis of regulatory action (Travis et al., 1987), and one order of magnitude less than the most conservative interpretation of de minimis risk (U.S. FDA, 1986) by a United States regulatory agency.

No significant risk due to noncarcinogenic effects of site-related chemicals has been identified under the baseline conditions.

Although no immediate exposure pathway has been identified from contact with soils within the fenced landfill area, there is a long-term potential of significant risk due to erosion or future mechanical disturbances of this site. These potential risks, as well as the limited risk associated with off-site soils, will be addressed in the evaluation of remedial alternatives.

A potentially significant risk to infaunal organisms has been identified for those individuals living in sediments in the embayment area adjoining the landfill. The limits of the embayment area containing site-related chemicals is shown in Figure 1. It is probable that this area contains sediments that were physically transported from the landfill area. The need and alternatives for remediation of this area will be addressed later in this document.

No further significant risk to aquatic populations or communities, or to animals feeding on aquatic organisms, was identified.

Since no significant impact derived from swimming in the vicinity of the 102nd Street Site, from ingestion of drinking water directly from the Niagara River or from consumption of Niagara River fish, and no significant ecological impact was identified adjacent to the landfill, it follows that no significant additional impact to receptors in Lake Ontario would derive from the 102nd Street Landfill Site.

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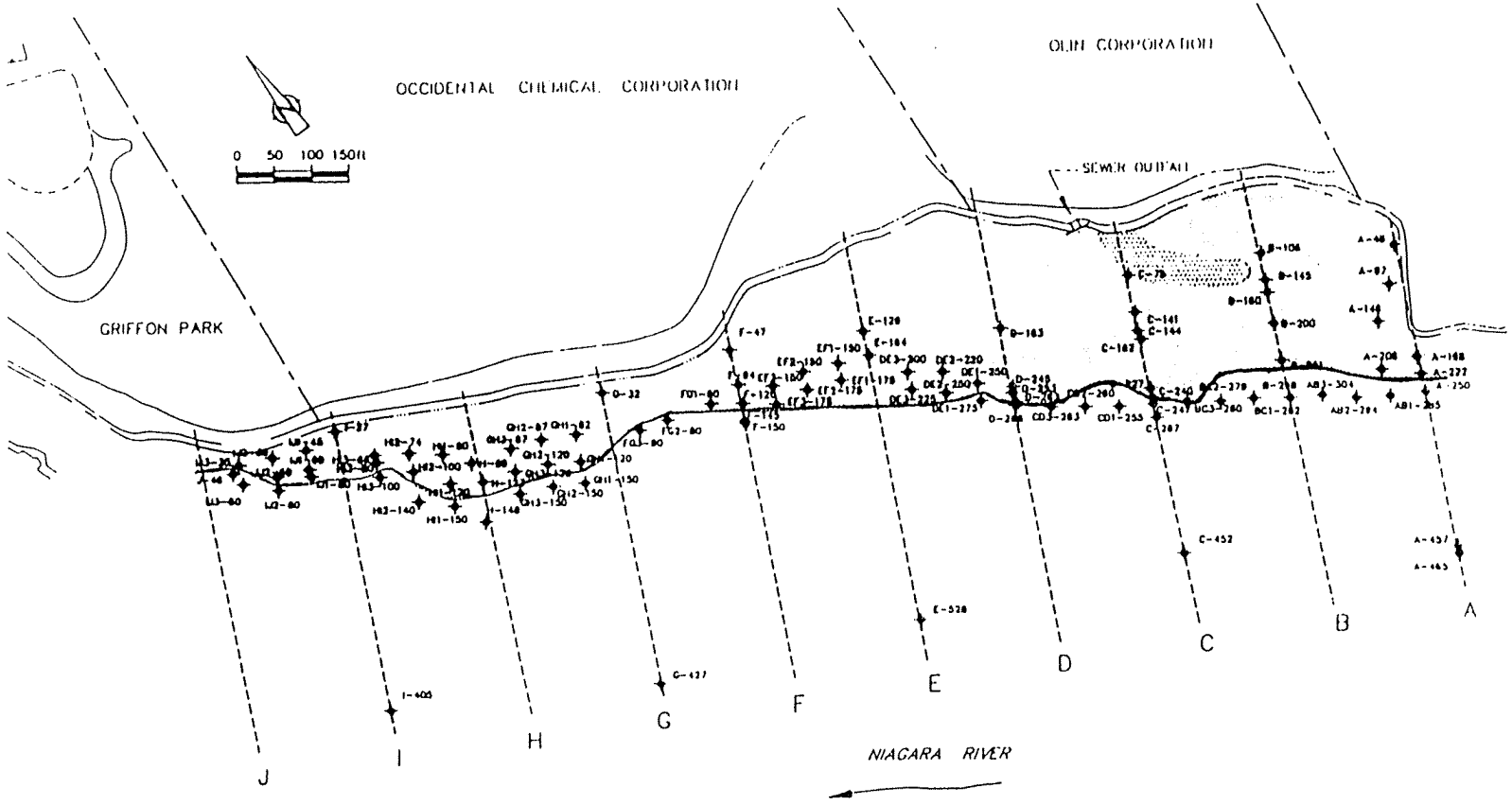

LEGEND

Approximate Location of former spit

Sediment Sample Location

A ----- Primary Sampling Vector

Probable Extent of Contamination

SOURCE: CRA/WCC 11/88

figure 8.1

SEDIMENT SAMPLE LOCATIONS
REMEDIAL INVESTIGATION
102nd Street Landfill Site

SIRRINE ENVIRONMENTAL CONSULTANTS
Greenville, South Carolina

Figure 1
Probable Extent of Site Related Chemicals in Sediments

TABLE 1
SCORING FOR INDICATOR CHEMICAL SELECTION: Koc VALUES AND CONCENTRATIONS IN VARIOUS ENVIRONMENTAL MEDIA

Chemical (CAS No.)	Koc Value	TEF (CCDs/ CDFs only)	Ground Water (mg/l) Range	Repres.	Ref	Soil (mg/kg) Range	Repres.	Ref	Sediment (mg/kg) Range	Repres.	Ref
1,1-Dichloroethylene (75-34-3)	6.50E+1 (L)		0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
Trichloroethylene (79-01-6)	1.26E+2 (L)		0 - 1.25E-1	1.16E-2	1	NA	- -		NA	- -	
Benzene (71-43-2)	8.30E+1 (L)		0 - 7.90E+1	6.05E+0	2	NA	3.91E+3	4	NA	- -	
Toluene (108-88-3)	3.00E+2 (L)		0 - 2.30E+0	1.11E-1	2	NA	- -		NA	- -	
Monochlorobenzene (108-90-7)	3.30E+2		0 1.60E+1	2.85E+0	2	NA	- -		NA	- -	
2-Monochlorotoluene (95-49-8)	3.70E+2		0 3.60E-1	1.54E-2	2	NAS	- -		0.00E+0 - 7.90E-1	1.23E-2	7
4-Monochlorotoluene (106-43-4)	9.95E+2		0 3.00E-1	1.22E-2	2	NAS	- -		0.00E+0 - 4.60E-1	7.19E-3	7
1,2-Dichlorobenzene (95-50-1)	1.70E+3		0 - 1.10E+0	1.25E-1	2	NAS	- -		0.00E+0 - 1.10E-1	1.72E-3	7
1,4-Dichlorobenzene (106-46-7)	1.70E+3		0 - 4.80E+0	4.03E-1	2	NAS	- -		0.00E+0 - 6.50E+0	2.41E-1	7
1,2,3-Trichlorobenzene (87-61-6)	9.20E+3 (H)		0 - 4.50E+0	2.14E-1	2	NAS	8.05E+2	4	0.00E+0 - 2.10E-1	3.28E-2	7
1,2,4-Trichlorobenzene (120-82-1)	9.20E+3 (H)		0 - 1.50E+1	8.25E-1	2	NAS	3.10E+3	4	0.00E+0 - 4.30E+1	6.75E-1	7
1,2,3,4-Tetrachlorobenzene (634-66-2)	1.60E+3		0 - 4.00E+1	7.62E-1	2	NAS	1.57E+4	4	0.00E+0 - 8.30E+1	1.30E+0	7
1,2,4,5-Tetrachlorobenzene (95-94-3)	1.60E+3		0 - 6.00E+0	1.27E-1	2	NAS	2.62E+3	4	0.00E+0 - 7.20E+1	1.13E+0	7
Pentachlorobenzene (608-93-5)	1.30E+4		0 - 1.80E-1	2.96E-2	1	NAS	- -		0.00E+0 - 5.40E+1	8.44E-1	7
Hexachlorobenzene (118-74-1)	3.90E+3 (H)		0 - 3.60E-1	5.00E-3	2	NAS	- -		0.00E+0 - 1.70E+0	3.21E-2	7
alpha-Hexachlorocyclohexane (319-84-6)	3.80E+3 (H)		0 - 8.40E-1	1.02E-1	2	NAS	1.35E+3	4	0.00E+0 - 2.04E+3	5.82E+1	7
beta-Hexachlorocyclohexane (319-85-7)	3.80E+3 (H)		0 - 1.40E-1	2.08E-2	2	NAS	2.74E+2	4	0.00E+0 - 3.10E+2	1.11E+1	7
gamma-Hexachlorocyclohexan (58-89-9)	1.08E+3		0 - 7.20E-1	4.57E-2	2	NAS	6.06E+2	4	0.00E+0 - 2.77E+1	4.92E-1	7
delta-Hexachlorocyclohexane (319-86-8)	6.60E+3 (H)		0 - 1.20E+0	1.27E-1	2	NAS	1.68E+3	4	0.00E+0 - 4.92E+0	1.45E-1	7
2,5-Dichloroaniline (95-82-9)	ND(H)		0 - 1.40E+1	9.52E-1	2	NA	- -		NA	- -	
3,4-Dichloroaniline (95-76-1)	HIGH		0 - 0.00E+0	0.00E+0	2	NA	- -		NA	- -	
Phenol (108-95-2)	1.42E+1 (L)		0 - 1.80E-1	1.53E-2	2	NA	- -		NA	- -	

TABLE 1
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Chemical (CAS No.)	Koc Value	TEF (CCDs/ CDFs only)	Ground Water (mg/l) Range	Repres.	Ref	Soil (mg/kg) Range	Repres.	Ref	Sediment (mg/kg) Range	Repres.	Ref
2-Chlorophenol (95-57-8)	1.86E+3 (H)		0 - 2.80E-1	2.65E-2	2	NA	- -		NA	- -	
4-Chlorophenol (106-48-9)	1.87E+2 (L)		0 - 3.90E+0	2.46E-1	2	NA	- -		NA	- -	
2,4-Dichlorophenol (120-83-2)	3.80E+2		0 - 2.10E+0	1.61E-1	2	NAS	- -		0.00E+0 - 0.00E+0	0.00E+0	7
2,5-Dichlorophenol (583-78-8)	ND		0 - 2.10E+0	1.26E-1	2	NAS	- -		0.00E+0 - 4.70E-1	7.34E-3	7
2,4,5-Trichlorophenol (95-95-4)	8.90E+1 (L)		0 - 2.90E-1	2.00E-2	2	NAS	2.56E+3	4	0.00E+0 - 3.67E-1	5.73E-3	7
2,4,6-Trichlorophenol (88-06-2)	8.90E+1 (L)		0 - 2.30E-1	1.05E-2	2	NAS	1.35E+3	4	0.00E+0 - 2.15E+0	3.40E-2	7
Pentachlorophenol (87-86-5)	5.30E+4		0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
4-Chloro-3-methylphenol (59-50-7)	5.00E+1 (L)		0 - 2.80E-2	1.33E-3	1	NA	- -		NA	- -	
2,4-Dimethylphenol (105-67-9)	4.25E+2		0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
2-Chlorobenzoic acid (118-91-2)	5.90E+1 (L)		0 - 8.60E-1	5.60E-1	2	NA	- -		NA	- -	
3-Chlorobenzoic acid (535-80-8)	3.00E+2		0 - 8.60E-1	4.31E-2	2	NA	- -		NA	- -	
4-Chlorobenzoic acid (74-11-3)	2.00E+2 (L)		0 - 2.00E+0	1.92E-1	2	NA	- -		NA	- -	
Benzo(a)anthracene (56-55-3)	1.38E+6		0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
Benzo(b)fluoranthene (205-99-2)	5.50E+5		0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
Benzo(k)fluoranthene (207-08-9)	5.50E+5		0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
2-Chloronaphthalene (91-58-7)	ND		0 - 7.50E-3	5.95E-4	1	NA	- -		NA	- -	
Mirex (2385-85-5)	2.40E+7		0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
PCBs as Aroclor 1248 (1336-36-3)	5.30E+5 (H)		0 - 1.40E-1	7.43E-3	1	NA	- -		NA	- -	
2,3,7,8-TCDD (1746-01-6)	3.30E+6 (H)		0 - 8.00E-7	6.20E-8	1	0- 1.03E-1	1.33E-2	5	NA	- -	
		1	[0 - 8.00E-7	6.20E-8]							
		Σ	[[0 - 8.00E-7	8.64E-8]]							
Other TCDDs			0 - 1.00E-6	1.05E-7	1	NA	- -		NA	- -	
		0.01	[0 - 1.00E-8	1.05E-9]							

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Chemical (CAS No.)	Koc Value	TEF (CCDs/ CDFs only)	Ground Water (mg/l) Range	Repres.	Ref	Soil (mg/kg) Range	Repres.	Ref	Sediment (mg/kg) Range	Repres.	Ref
1,2,3,7,8-PeCDD (40321-76-4)			0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
Other PeCDD's		0.005	0 - 2.10E-6 [0 - 1.05E-8	1.33E-7 6.65E-10]	1	NA	- -		NA	- -	
1,2,3,4,7,8-HxCDD			0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
1,2,3,6,7,8-HxCDD (57653-85-7)		0.04	0 - 1.80E-6 [0 - 7.20E-8	1.48E-7 5.90E-9]	1	NA	- -		NA	- -	
1,2,3,7,8,9-HxCDD (19408-74-3)		0.04	0 - 9.00E-7 [0 - 3.60E-8	6.66E-8 2.66E-9]	1	NA	- -		NA	- -	
Other HxCDDs		0.0004	0 - 9.30E-6 [0 - 3.72E-9	8.21E-7 3.28E-10]	1	NA	- -		NA	- -	
1,2,3,4,6,7,8-HpCDD		0.001	0 - 2.36E-5 [0 - 2.36E-8	2.62E-6 2.62E-9]	1	NA	- -		NA	- -	
Other HpCDDs		0.00001	0 - 2.50E-5 [0 - 2.50E-10	1.90E-6 1.90E-11]	1	NA	- -		NA	- -	
OCDD		0	0 - 1.90E-4 [0 - 0.00E+0	1.60E-5 0.00E+0]	1	NA	- -		NA	- -	
2,3,7,8-TCDF		0.1	0 - 6.00E-7 [0 - 6.00E-8	2.86E-8 2.86E-9]	1	NA	- -		NA	- -	
Other TCDFs		0.001	0 - 1.60E-6 [0 - 1.60E-9	1.60E-7 1.60E-10]	1	NA	- -		NA	- -	
1,2,3,7,8-PeCDF			0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
2,3,4,7,8-PeCDF			0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
Other PeCDFs		0.001	0 - 2.80E-6 [0 - 2.80E-9	2.57E-7 2.57E-10]	1	NA	- -		NA	- -	
1,2,3,4,7,8-HxCDF		0.01	0 - 7.50E-6 [0 - 7.50E-8	6.66E-7 6.66E-9]	1	NA	- -		NA	- -	
1,2,3,6,7,8-HxCDF		0.01	0 - 9.00E-7 [0 - 9.00E-9	6.66E-8 6.66E-10]	1	NA	- -		NA	- -	
2,3,4,6,7,8-HxCDF			0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	

TABLE 1
SCORING FOR INDICATOR CHEMICAL SELECTION: Koc VALUES AND CONCENTRATIONS IN VARIOUS ENVIRONMENTAL MEDIA

Chemical (CAS No.)	Koc Value	TEF (CCDs/ CDFs only)	Ground Water (mg/l) Range	Ground Water (mg/l) Repres.	Ref	Soil (mg/kg) Range	Soil (mg/kg) Repres.	Ref	Sediment (mg/kg) Range	Sediment (mg/kg) Repres.	Ref
1,2,3,7,8,9-HxCDF			0 - 0.00E+0	0.00E+0	1	NA	- -		NA	- -	
Other HxCDFs		0.0001	0 - 3.10E-6 [0 - 3.10E-10	2.95E-7 2.95E-11]	1	NA	- -		NA	- -	
1,2,3,4,6,7,8-HpCDF		0.001	0 - 6.10E-6 [0 - 6.10E-9	3.05E-7 3.05E-10]	1	NA	- -		NA	- -	
1,2,3,4,7,8,9-HpCDF		0.001	0 - 3.90E-6 [0 - 3.90E-9	1.86E-7 1.86E-10]	1	NA	- -		NA	- -	
Other HpCDFs		0.00001	0 - 4.90E-6 [0 - 4.90E-11	2.57E-7 2.57E-12]	1	NA	- -		NA	- -	
OCDF	-	0	0 - 4.55E-5 [0 - 0.00E+0	3.85E-6 0.00E+0]	1	NA	- -		NA	- -	
Arsenic (7440-38-2)	-		0 - 2.30E-1	5.90E-2	3	NA	- -		NA	- -	
Cadmium (7740-43-9)	-		0 - 3.30E-2	9.00E-3	3	NA	- -		NA	- -	
Mercury (7439-97-6)	-		0 - 6.40E-2	1.50E-2	3	5.00E-2 -	4.51E+1 4.32E+0	6	0.00E+0 - 1.03E+2	2.08E+0	7
Phosphorous (12185-10-3)			0 - 2.20E-2	4.00E-3	3	NA	- -		NA	- -	

Footnotes

TEF - 2,3,7,8-TCDD Toxicity Equivalence Factors
[ ] - Concentration ranges and means in terms of 2,3,7,8-TCDD equivalents
Σ [[ ]] - Sum of TEF of each CDD/CDF isomer/congener times the concentration of the respective isomer/congener
NA - Not analyzed for in this medium
NAS - Not analyzed on site - Analysis performed on off-site soil samples
ND - No datum available
Koc Value - The 10 highest and 10 lowest Koc values among detected chemicals are indicated by H and L, respectively, 2,5 - Dichloroaniline is rated high based upon similarities with 3,4 - Dichloroaniline.

References

1. Occidental and Olin Assessment Chemical Monitoring Program, May 17, 1989, Tables 4 and 5.
2. 102 St. Landfill Site, Draft RI, Tables 11.16 and 11.17
3. Draft RI, Tables 5.1 and 5.2
4. Calculated from Olin Waste Surveys
5. Draft RI, Table 1.5
6. Draft RI, Figure 7.4
7. Draft RI, Appendix C

TABLE 2
SCORING FOR INDICATOR CHEMICAL SELECTION:
TOXICITY INFORMATION

Chemical	Toxicologic Class	Rating Value/EPA Category	wTc	sTc
Trichloroethylene	PC	B2	4.29E-3	2.14E-7
(79-01-6)	NC	5	1.05E+0	5.26E-5
Benzene	PC	A	7.71E-3	3.86E-7
(71-43-2)	NC	5	1.17E-1	5.85E-6
Toluene (108-88-3)	NC	7	5.20E-3	2.60E-7
Monochlorobenzene (108-90-7)	NC		1.43E-1	7.14E-6
2-Monochlorotoluene (95-49-8)	NC		1.43E-1	7.14E-6
4-Monochlorotoluene (106-43-4)	NC		1.43E-1	7.14E-6
1,2-Dichlorobenzene (95-50-1)	NC	4	5.19E-2	2.60E-6
1,4-Dichlorobenzene (106-46-7)	NC	4	5.19E-2	2.60E-6
1,2,3-Trichlorobenzene (87-61-6)	NC		2.14E-1	1.07E-5
1,2,4-Trichlorobenzene (120-82-1)	NC	4	2.14E-1	1.07E-5
1,2,3,4-Tetrachlorobenzene (634-66-2)	NC		9.76E-2	4.88E-6
1,2,4,5-Tetrachlorobenzene (95-94-3)	NC	1	9.76E-2	4.88E-6
Pentachlorobenzene (608-93-5)	NC	10	2.32E-2	1.16E-6

TABLE 2
SCORING FOR INDICATOR CHEMICAL SELECTION:
TOXICITY INFORMATION

Chemical	Toxicologic Class	Rating Value/EPA Category	wTc	sTc
Hexachlorobenzene	PC	B2	3.36E-1	1.68E-5
(118-74-1)	NC	10	4.00E-1	2.00E-5
alpha-Hexachlorocyclohexane (319-84-6)	PC	B2	1.56E+0	7.79E-5
beta-Hexachlorocyclohexane (319-85-7)	PC	C	4.97E-2	2.49E-6
gamma-Hexachlorocyclohexan (58-89-9)	PC	B2/C	5.23E-2	2.61E-6
delta-Hexachlorocyclohexane (319-86-8)	NC		ND	ND
2,5-Dichloroaniline (95-82-9)	NC		ND	ND
Phenol (108-95-2)	NC	3	1.00E-1	5.02E-6
2-Chlorophenol (95-57-8)	NC		8.26E-2	4.13E-6
4-Chlorophenol (106-48-9)	NC		8.26E-2	4.13E-6
2,4-Dichlorophenol (120-83-2)	NC	5	8.26E-2	4.13E-6
2,5-Dichlorophenol (583-78-8)	NC		8.26E-2	4.13E-6
2,4,5-Trichlorophenol (95-95-4)	NC	6	1.02E-1	5.10E-6
2,4,6-Trichlorophenol (88-06-2)	PC	B2	2.29E-3	1.14E-7
4-Chloro-3-methylphenol (59-50-7)	NC		ND	ND
2-Chlorobenzoic acid (118-91-2)	NC		ND	ND

TABLE 2
SCORING FOR INDICATOR CHEMICAL SELECTION:
TOXICITY INFORMATION

Chemical	Toxicologic Class	Rating Value/EPA Category	wTc	sTc
3-Chlorobenzoic acid (535-80-8)	NC		ND	ND
4-Chlorobenzoic acid (74-11-3)	NC		ND	ND
2-Chloronaphthalene (91-58-7)	NC		ND	ND
PCBs as Aroclor 1248 (1336-36-3)	PC	B2	5.71E-1	2.86E-5
2,3,7,8-TCDD (1746-01-6)	PC	B2	3.43E+3	1.71E-1
Arsenic	PC	A	4.07E+0	2.03E-4
(7440-38-2)	NC	9	1.80E+1	9.00E-4
Cadmium	PC	B1	ND	ND
(7740-43-9)	NC	10	4.45E+0	2.23E-4
Mercury (7439-97-6)	NC	7	1.84E+1	9.21E-4
Phosphorous (12185-10-3)	NC		ND	ND

Footnotes
PC - Potential carcinogen
NC - Noncarcinogen
Rating Value - For severity of noncarcinogenic effects, range 1 (low) to 10 (high)
EPA Category - Weight of evidence designation for carcinogenic effects
wTc - Toxicity constant for water
sTc - Toxicity constant for soil
ND - No datum available

TABLE 3
SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR CARCINOGENIC EFFECTS

	Ground Water		Soil		Sediment		IS Value		Tentative Rank	
Chemical	Max	Repres	Max	Repres	Max	Repres	Max	Repres	Max	Repres
Trichloroethylene (79-01-6)	5.36E-4	4.98E-5	NA	NA	NA	NA	5.36E-4	4.98E-5	9	9
Benzene (71-43-2)	6.09E-1	4.67E-2	NA	3.01E-5	NA	NA	6.09E-1	4.67E-2	3	3
Hexachlorobenzene (118-74-1)	1.21E-1	1.68E-3	NA	NA	2.86E-5	5.39E-7	1.21E-1	1.68E-3	4	6
alpha-Hexachlorocyclohexane (319-84-6)	1.31E+0	1.59E-1	NA	1.05E-7	1.59E-1	4.53E-3	1.31E+0	1.59E-1	1	2
beta-Hexachlorocyclohexane (319-85-7)	6.96E-3	1.04E-3	NA	6.82E-8	7.72E-4	3.07E-4	6.96E-3	1.04E-3	7	7
gamma-Hexachlorocyclohexan (58-89-9)	3.77E-2	2.39E-3	NA	1.58E-3	7.23E-5	1.28E-6	3.77E-2	2.39E-3	5	5
2,4,6-Trichlorophenol (88-06-2)	5.27E-4	2.40E-5	NA	2.92E-4	2.45E-7	3.88E-9	5.27E-4	2.40E-5	10	10
PCBs as Aroclor 1248 (1336-36-3)	2.28E-2	7.94E-3	NA	NA	NA	NA	2.28E-2	7.94E-3	6	4
2,3,7,8-TCDD (1746-01-6)	2.35E-3	2.37E-4	1.16E-5	3.13E-7	NA	NA	2.35E-3	2.37E-4	8	8
Arsenic (7440-38-2)	9.36E-1	2.40E-1	NA	NA	NA	NA	9.36E-1	2.40E-1	2	1

Footnotes

NA - Not analyzed for in this medium.

CT - Product of the concentration and the toxicity constant of a chemical for the respective medium and effects.

IS - Indicator score. Only groundwater CT values can be used for comparative indicator scores due to incomplete data in other media. Soil and sediment CT values are included only for reference comparisons among those chemicals analyzed for in these media.

Cadmium - Although cadmium is classified as a B1 potential carcinogen based on inhalation evidence, insufficient data exist from the 102 St. site to permit comparative scoring for this route of entry. Therefore, cadmium has been scored for noncarcinogenic effects only (see Table 4).

TABLE 4
SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR NONCARCINOGENIC EFFECTS

	Ground Water		Soil		Sediment		IS Value		Tentative Rank	
Chemical	Max	Repres	Max	Repres	Max	Repres	Max	Repres	Max	Repres
Trichloroethylene (79-01-6)	1.31E-1	1.22E-2	NA	NA	NA	NA	1.31E-1	1.22E-2	15	11
Benzene (71-43-2)	9.24E+0	7.08E-1	NA	4.57E+2	NA	NA	9.24E+0	7.08E-1	1	2
Toluene (108-88-3)	1.20E-2	5.79E-4	NA	NA	NA	NA	1.20E-2	5.79E-4	22	23
Monochlorobenzene (108-90-7)	2.29E+0	4.08E-1	NA	NA	NA	NA	2.29E+0	4.08E-1	5	3
2-Monochlorotoluene (95-49-8)	5.15E-2	2.20E-3	NA	NA	5.64E-6	8.78E-8	5.15E-2	2.20E-3	17	16
4-Monochlorotoluene (106-43-4)	4.29E-2	1.74E-3	NA	NA	3.28E-6	5.13E-8	4.29E-2	1.74E-3	18	20
1,2-Dichlorobenzene (95-50-1)	5.71E-2	6.50E-3	NA	NA	2.86E-7	4.47E-9	5.71E-2	6.50E-3	16	15
1,4-Dichlorobenzene (106-46-7)	2.49E-1	2.09E-2	NA	NA	1.69E-5	6.27E-7	2.49E-1	2.09E-2	10	9
1,2,3-Trichlorobenzene (87-61-6)	9.63E-1	4.58E-2	NA	2.09E-3	2.25E-6	3.51E-7	9.63E-1	4.58E-2	7	7
1,2,4-Trichlorobenzene (120-82-1)	3.21E+0	1.76E-1	NA	3.32E-2	4.60E-4	7.22E-6	3.21E+0	1.76E-1	4	5
1,2,3,4-Tetrachlorobenzene (634-66-2)	3.90E+0	7.44E-2	NA	7.66E-2	4.05E-4	6.34E-6	3.90E+0	7.44E-2	3	6
1,2,4,5-Tetrachlorobenzene (95-94-3)	5.86E-1	1.24E-2	NA	1.28E-2	3.51E-4	5.51E-6	5.86E-1	1.24E-2	8	13
Pentachlorobenzene	4.18E-3	6.87E-4	NA	NA	6.26E-5	9.79E-7	4.18E-3	6.87E-4	23	22
Hexachlorobenzene (118-74-1)	1.44E-1	2.00E-3	NA	NA	3.40E-5	6.42E-7	1.44E-1	2.00E-3	14	19
delta-Hexachlorocyclohexane (319-86-8)	NS	NS	NA	NS	NS	NS	NS	NS	NS	NS
2,5-Dichloroaniline	NS	NS	NA	NA	NA	NA	NS	NS	NS	NS

TABLE 4
SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR NONCARCINOGENIC EFFECTS

	Ground Water		Soil		Sediment		IS Value		Tentative Rank	
Chemical	Max	Repres	Max	Repres	Max	Repres	Max	Repres	Max	Repres
(95-82-9)										
Phenol (108-95-2)	1.80E-2	1.53E-3	NA	NA	NA	NA	1.80E-2	1.53E-3	21	21
2-Chlorophenol (95-57-8)	2.31E-2	2.19E-3	NA	NA	NA	NA	2.31E-2	2.19E-3	20	17
4-Chlorophenol (106-48-9)	3.22E-1	2.03E-2	NA	NA	NA	NA	3.22E-1	2.03E-2	9	10
2,4-Dichlorophenol (120-83-2)	1.73E-1	1.33E-2	NA	NA	0.00E+0	0.00E+0	1.73E-1	1.33E-2	11	12
2,5-Dichlorophenol (583-78-8)	1.73E-1	1.04E-2	NA	NA	1.94E-6	3.03E-8	1.73E-1	1.04E-2	11	14
2,4,5-Trichlorophenol (95-95-4)	2.96E-2	2.04E-3	NA	1.30E-2	1.87E-6	2.92E-8	2.96E-2	2.04E-3	19	18
4-Chloro-3-methylphenol (59-50-7)	NS	NS	NA	NA	NA	NA	NS	NS	NS	NS
2-Chlorobenzoic acid (118-91-2)	NS	NS	NA	NA	NA	NA	NS	NS	NS	NS
3-Chlorobenzoic acid (535-80-8)	NS	NS	NA	NA	NA	NA	NS	NS	NS	NS
4-Chlorobenzoic acid (74-11-3)	NS	NS	NA	NA	NA	NA	NS	NS	NS	NS

TABLE 4
SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR NONCARCINOGENIC EFFECTS

	Ground Water		Soil		Sediment		IS Value		Tentative Rank	
Chemical	Max	Repres	Max	Repres	Max	Repres	Max	Repres	Max	Repres
2-Chloronaphthalene (91-58-7)	NS	NS	NA	NA	NA	NA	NS	NS	NS	NS
Arsenic (7440-38-2)	4.14E+0	1.06E+0	NA	NA	NA	NA	4.14E+0	1.06E+0	2	1
Cadmium (7740-43-9)	1.47E-1	4.01E-2	NA	NA	NA	NA	1.47E-1	4.01E-2	13	8
Mercury (7439-97-6)	1.18E+0	2.76E-1	4.15E-2	3.98E-3	9.39E-2	1.92E-3	1.18E+0	2.76E-1	6	4
Phosphorous (12185-10-3)	NS	NS	NA	NA	NA	NA	NS	NS	NS	NS

Footnotes

NA - Not analyzed for in this medium.

NS - Chemical not scored by this method. Relative rank determined by comparison of toxicity and concentration data with those of scored chemicals (see text).

CT - Product of the concentration and the toxicity constant of a chemical for the respective medium and effects.

IS- Indicator score. Only groundwater CT values can be used for comparative indicator scores due to incomplete data in other media. Soil and sediment CT values are included only for reference comparisons among those chemicals analyzed for in these media.

TABLE 5
102 STREET LANDFILL DETECTED CHEMICALS
HUMAN HEALTH ASSESSMENT DATA

CHEMICAL	AIC-NONCARCINOGENIC ORAL	AIC-NONCARCINOGENIC INHAL.	POT. SLOPE-CARCINOGENIC ORAL	POT. SLOPE-CARCINOGENIC INHAL.	WOE	HAZ RANK	MCL (ug/l)	W.Q. Criteria (ug/l) WFC	W.Q. Criteria (ug/l) FC	FISH BCF (l/kg)
Trichloroethylene			1.1E-2(1)	1.3E-2(1)	B2	LOW	5	2.7	8.07E+1(d)	10.6(2)
Benzene			2.9E-2(1)	2.9E-2(1)	A	MED	5	6.6E-1	4.0E+1(d)	5.2(2)
Toluene	3E-1(1)	1.50(2)			D		2.0E+3 (a)	1.43E+4	4.24E+5	10.7(2)
Monochlorobenzene	2.70E-2(2)	5.70E-3(2)								645(4)
2-Monochlorotoluene										
4-Monochlorotoluene										
1,2-Dichlorobenzene										56(2)
1,4-Dichlorobenzene										56(2)
1,2,3-Trichlorobenzene										
1,2,4-Trichlorobenzene	2E-2(1)				D					3,200(3)
1,2,3,4-Tetrachlorobenzene										
1,2,4,5-Tetrachlorobenzene	3E-4(1)							3.8E+1	4.8E+1	1,125(2)
Pentachlorobenzene	8E-4(1)							7.4E+1	8.5E+1	2,125(2)
Hexachlorobenzene	8E-4(1)		1.69(2)		B2	MED				12,000(3)
alpha-Hexachlorocyclohexane			6.3(1)	6.3(1)	B2	MED		9.2E-3	3.1E-2(d)	605(3)
beta-Hexachlorocyclohexane			1.8(1)	1.8(1)	C	LOW		1.63E-2	5.47E-2(d)	658(3)
gamma-Hexachlorocyclohexane	3E-4(1)		1.33(2)		B2/C	MED	4	1.86E-2	6.25E-2	605(3)
delta-Hexachlorocyclohexane					D					648(3)
2,5-Dichloroaniline										72(3)
Phenol	1E-2(1)	2E-2(2)						3E+2 (b)		1.4(2)
2-Chlorophenol	5E-3(1)									
4-Chlorophenol										19(3)
2,4-Dichlorophenol	3E-1(1)							3.09E+3	3.09E+3	41(2)
2,5-Dichlorophenol										

TABLE 5
102 STREET LANDFILL DETECTED CHEMICALS
HUMAN HEALTH ASSESSMENT DATA

CHEMICAL	AIC-NONCARCINOGENIC ORAL	AIC-NONCARCINOGENIC INHAL.	POT. SLOPE-CARCINOGENIC ORAL	POT. SLOPE-CARCINOGENIC INHAL.	WOE	HAZ RANK	MCL (ug/l)	W.Q. Criteria (ug/l) WFC	W.Q. Criteria (ug/l) FC	FISH BCF (l/kg)
2,4,5-Trichlorophenol	1E-1(2)				D			2.6E+4		110(2)
2,4,6-Trichlorophenol			2E-2(1)	2E-2(1)	B2	LOW		1.2	3.6	150(2)
4-Chloro-3-methylphenol										
2-Chlorobenzoic acid										5(5)
3-Chlorobenzoic acid										
4-Chlorobenzoic acid										
2-Chloronaphthalene										
PCBs			7.7(1)		B2	MED				100,000(2)
2,3,7,8-Tetrachlorodibenzo-p-dioxin			1.56E+5(2)		B2	HIGH				128,000(6)
Arsenic		5E+1(1)	1.8(1)	5.0E+1(2)	A	HIGH	5.0E+1	2.2E-3	1.75E-2	44(2)
Cadmium	5E-4 (7)		(c)	6.1(1)	B1	MED	1.0E+1	1E+1	NA	81(2)
Mercury	3E-4 (1)	5.1E-5(2)			D					5500(2)
Phosphorous										

TABLE 5
102 STREET LANDFILL DETECTED CHEMICALS
HUMAN HEALTH ASSESSMENT DATA

CHEMICAL	AIC-NONCARCINOGENIC ORAL	AIC-NONCARCINOGENIC INHAL.	POT. SLOPE-CARCINOGENIC ORAL	POT. SLOPE-CARCINOGENIC INHAL.	WOE	HAZ RANK	MCL (ug/l)	W.Q. Criteria (ug/l) WFC	W.Q. Criteria (ug/l) FC	FISH BCF (l/kg)

REFERENCES

1. IRIS
2. SPHEM
3. Hazardous Substances Data Bank (HSDB), Toxicological Data Network, National Library of Medicine
4. U.S. EPA, 1980b
5. MacKay, D.M., 1982 Environ. Science and Technol., 16: 274-8
6. Cook, P.M., 1987
7. U.S.EPA, 1987

FOOTNOTES

(a) Proposed MCLG
(b) Based on organoleptic effects
(c) Insufficient data exist to classify carcinogenicity to humans by oral route
(d) Criteria represent a 1 in 1,000,000 lifetime cancer risk

ABBREVIATIONS

AIC	- Acceptable Chronic Intake for noncarcinogenic effects (mg/kg/day)
POT. SLOPE	-95% upper-bound slope (qi*) for carcinogenic potency 1/(mg/kg/day) based on the linearized multistage model
WOE	- Weight of Evidence for carcinogenic effects
HAZ RANK	-Hazard ranking of potential carcinogens by EPA's carcinogen Assessment Group (Environmental Monitoring & Services, Inc., Dec. 1986)
MCL	- Maximum Contaminant Level for drinking water
MCLG	- MCL Goal
W.Q. Criteria	- Ambient Water Quality Criteria for human health effects
WFC	- for water and fish consumption
FC	- for fish consumption only
BCF	- Bioconcentration Factor (from SPHEM unless otherwise indicated - see Table 10 for more detail on specific species)

TABLE 6
102 STREET LANDFILL
COMPARATIVE LD50 VALUES FOR
HIGH SCORED AND NONSCORED CHEMICALS

CHEMICAL	ORAL LD50 (MG/KG)		ITP LD50 (MG/KG)	
	MOUSE	RAT	MOUSE	RAT
Trichloroethylene	2.40E+3	N D	N D	1.28E+3
Benzene	4.70E+3	3.31E+3	3.40E+2	2.89E+0
Monochlorobenzene	2.30E+3	2.29E+3	5.15E+2	N D
1,4-Dichlorobenzene	2.95E+3	5.00E+2	2.00E+3	2.56E+3
1,2,3-Trichlorobenzene	N D	N D	1.39E+3	N D
1,2,4-Trichlorobenzene	3.00E+2	7.56E+2	1.22E+3	N D
1,2,3,4-Tetrachlorobenzene	N D	1.17E+3	N D	N D
Hexachlorobenzene	4.00E+3	N D	N D	N D
alpha-Hexachlorocyclohexane	7.80E+1	1.77E+2	N D	N D
beta-Hexachlorocyclohexane	N D	6.00E+3	N D	N D
gamma-Hexachlorocyclohexane	4.40E+1	7.60E+1	1.25E+2	3.50E+1
delta-Hexachlorocyclohexane	N D	1.00E+3	N D	N D
2,5-Dichloroaniline	N D	2.90E+3	N D	N D
4-Chlorophenol	3.67E+2	N D	N D	2.81E+2
2,4,6-Trichlorophenol	N D	8.20E+2	N D	2.76E+2
4-Chloro-3-methylphenol	N D	1.83E+2	N D	N D
2-Chlorobenzoic acid	N D	N D	N D	2.30E+3
3-Chlorobenzoic acid	N D	N D	N D	7.50E+2
4-Chlorobenzoic acid	1.17E+3	1.17E+3	N D	1.00E+3
2-Chloronaphthalene	8.86E+2	2.08E+3	N D	N D
PCBs	1.90E+3	N D	N D	N D
2,3,7,8-Tetrachlorodibenzo-p-dioxin	1.14E-1	2.00E-2	1.20E-1	6.00E-2

TABLE 6
102 STREET LANDFILL
COMPARATIVE LD50 VALUES FOR
HIGH SCORED AND NONSCORED CHEMICALS

CHEMICAL	ORAL LD50 (MG/KG)		ITP LD50 (MG/KG)	
	MOUSE	RAT	MOUSE	RAT
Arsenic	1.45E+2	7.63E+2	4.62E+1	N D
Cadmium	N D	N D	N D	N D
Mercury	N D	N D	N D	N D
Phosphorous	N D	N D	N D	N D

FOOTNOTES

ND - No datum available.
ORAL - Dose administered by oral route.
ITP - Dose administered by intraperitoneal injection.

TABLE 7
SCORING FOR INDICATOR CHEMICAL SELECTION
EVALUATION OF EXPOSURE FACTORS AND FINAL CHEMICAL SELECTION

Chemical	IS Ranking PC	IS Ranking NC	Water Sol. (mg/l)	VP (mm. Hg)	MP (°C)	Koc Rank	Hc (atm-m3/mole)	Half -Life (days) GW	SW	S	A	IC
Trichloroethylene	9	11	1.10E+3	6.00E+1	-87	L	8.92E-3		1 - 90		4	
Benzene	3	2	1.78E+3	7.60E+1	5.5	L	5.35E-3		1 - 6		6	+
Monochlorobenzene	- -	3	5.00E+2	1.00E+1	-45		3.40E-3		0. 3		4	+
1,4-Dichlorobenzene	- -	9	6.90E+1	6.80E-1	53.5		1.55E-3		2 - 9		23	
1,2,3-Trichlorobenzene	- -	7	1.20E+1	2.10E-1	52.6	H						
1,2,4-Trichlorobenzene	- -	5	3.00E+1	4.60E-1	17	H	2.31E-3		1		18	+
1,2,3,4-Tetrachlorobenzene	- -	6	3.50E+0	4.00E-2	47.5							
Hexachlorobenzene	6	19	4.95E-3	1.09E-5	231	H	6.79E-4		0.3 - 300	1100 -2200	80	+
alpha-HCCH	2	- -	1.00E+1	2.50E-5	157	H	5.87E-6		>3 - 30		2	+
beta-HCCH	7	- -	5.00E+0	3.00E-7	297	H	4.47E-7					
gamma-HCCH	5	- -	7.30E+0	1.00E-4	112		3.10E-6		3 - 30		2	
2.5-Dichloroaniline	- -	[7]	slightly		50	H						+
4-Chlorophenol	- -	10	2.71E+4	1.00E-1	43.5	H						
2,4,6-Trichlorophenol	10	- -	9.00E+2	1.20E-1	68	L	1.77E-5		1 - 19	5	1	+
2-Chlorobenzoic acid	- -	[9]	2.10E+3	sublimes	142	L						+
PCBs	4	- -	3.10E-2	7.70E-5		H	1.07E-3		2 - 13		58	+
2,3,7,8-TCDD	8	- -	1.93E-5	7.40E-10	305	H	1.60E-5		365 - 730	10 ,590	0.2	+
Arsenic	1	1	insol.	0.00E+0	817		7.48E-7		persistant		5	+
Cadmium	- -	8	insol.	1.00E+0	321		5.68E-5		persistant		5	+
Mercury	- -	4	6.00E-2	2.00E-3	-38.9		1.14E-2		persistant		5	+

References:
Howard, P.H. (ed.), 1990. Handbook of Environmental Fate and Exposure Data for Organic Chemicals.
U.S. EPA, 1986. Superfund Public Health Evaluation Manual.
Verschueren, K., 1983. Handbook of Environmental Data on Organic Chemicals.

TABLE 8
102 STREET LANDFILL
ACUTE TOXICITY (LC50/EC50) CONCENTRATIONS (1) FOR SELECTED CHEMICALS
ENVIRONMENTAL ENDANGERMENT ASSESSMENT

CHEMICAL	CAS NO.	DAPHNIA	FATHEAD	TROUT	BLUEGILL	OTHER
Trichloroethylene	79-01-6	18/48-hr (5)	40.7-66.8 (2)	28.5 (15)	44.7 (2)	
Benzene	71-43-2	203-412 (5)	32.0-33.5 (5)	9.2 (8,9)	22.5-100 (5,8)	5.8-10.9 bass (2)
Monochlorobenzene	108-90-7	12.9-86/48-hr (6,13)	32 (5)	4.7 (15)	18 (5)	
1,2,3-Trichlorobenzene	87-61-6			0.71/48-hr (2)		
1,2,4-Trichlorobenzene	120-82-1	3.4-50.2/48-hr (2,10)	2.7-3.0 (10,11,15)	1.5 (15)	3.36 (2)	
1,2,3,4-Tetrachlorobenzene	634-66-2		1.1-22 (2,11)		12 (2)	14 catfish, bass (2)
Hexachlorobenzene	118-74-1		22 (8)		12 (8)	14 catfish (8)
alpha-Hexachlorocyclohexane	319-84-6					12 bass (8)
beta-Hexachlorocyclohexane	319-85-7					
gamma-Hexachlorocyclohexane	58-89-9	.46-1.5/48-hr (2,12)	0.087 (2)	.0017-.032 (2)	0.068 (2)	.01-.09 amphipod (2)
2,5-Dichloroaniline	95-82-9	.29-.44 (14)				.09 carp (2) .044-.064 catfish (2) .032 bass (2)
4-Chlorophenol	106-48-9	4.06 (2)		1.9 (15)	3.83 (2)	
2,4,6-Trichlorophenol	88-06-2	6.0 (6)	4.55-9.16 (3,4)	1.1 (16)	0.32 (7)	
2-Chlorobenzoic acid	118-91-2					
PCBs as Aroclor 1248	1336-36-3		.008-0.3 (5)	0.054 (9)	0.69 (8)	.019 sunfish (9) .002 bass (9)
2,3,7,8-TCDD	1746-01-6			.5E-6 (18)		

TABLE 8
102 STREET LANDFILL
ACUTE TOXICITY (LC50/EC50) CONCENTRATIONS (1) FOR SELECTED CHEMICALS
ENVIRONMENTAL ENDANGERMENT ASSESSMENT

CHEMICAL	CAS NO.	DAPHNIA	FATHEAD	TROUT	BLUEGILL	OTHER
Arsenic	7440-38-2	4.34 (2)	14.9-82.4 (2,5)		21.2 (5)	12.3 salmon (2)
Cadmium	7440-43-9	.033-.063 (5)	0.08-12.0 (5,17)		21.1 (5)	0.14 carp (17) 0.24 bass (17)
Mercury	7439-97-6	.005 (5)	0.15 (5)	.024-.400 (5)		0.35 catfish (2)

FOOTNOTES

1. Concentrations are in mg/l; test duration 96-hr unless noted.
2. Hazardous Substances Data Bank, Toxicological Data Network, National Library of Medicine.
3. Geiger, et. al. 1985.
4. Geiger, et. al. 1988.
5. USEPA Ambient Water Quality Criteria documents.
6. LeBlanc, 1980.
7. Buccafusco, et. al., 1981.
8. Johnson and Finley, 1980.
9. Mayer and Ellersieck, 1986.
10. Holcombe, et. al., 1987.
11. Carlson and Kosian, 1987.
12. Hermens, et. al.,1984.
13. Cowgill, et. al., 1985.
14. 3,4-DCA; Crossland and Hillaby, 1985.
15. McCarty, et. al., 1985.
16. Hattula, et. al. 1981.
17. Birge, et. al., 1985.
18. Value derived from relationship logNOEC = -1.28 + .95logLC50 (Slooff et. al., 1986).

TABLE 9
102 STREET LANDFILL
CHRONIC TOXICITY CONCENTRATIONS (ug/l) FOR SELECTED CHEMICALS
ENVIRONMENTAL ENDANGERMENT ASSESSMENT

CHEMICAL	CAS NO.	DAPHNIA	FATHEAD	TROUT	BLUEGILL	OTHER
Trichloroethylene	79-01-6	Nc 579 (9)	Nc 1636 (9)	Nc 896 (9)	Nc 1373 (9)	
Benzene	71-43-2	N > 98,000 (2) M > 98,000 (2)	Nc 1022 (9)	N 1000 (4) L 28,560 (4) M 5342 (4)	Nc 1852 (9)	
Monochlorobenzene	108-90-7	Nc 1511 (9)	Nc1000 (9)	Nc 162 (9)	Nc 579 (9)	
1,2,3-Trichlorobenzene	87-61-6			Nc 27 (9)		
1,2,4-Trichlorobenzene	120-82-1	Nc 845 (9)	N 119-507 (1,4) L 417-1001 (1,4) M 290-707 (1,4)	N 99 (4) L 159 (4) M 126 (4)	Nc 117 (9)	
1,2,3,4-Tetrachlorobenzene	634-66-2		N 237-496 (1,4) L 410-993 (1,4) M 323-712 (1,4)		Nc 394 (9)	
Hexachlorobenzene	118-74-1		Nc 700 (9)		Nc 394 (9)	
alpha-Hexachlorocyclohexane	319-84-6					Nc 394 bass (9)
beta-Hexachlorocyclohexane	319-85-7					
gamma-Hexachlorocyclohexane	58-89-9	Nc 36 (9)	N 9.1 (1) L 23.5 (1) M 9.1-34.5 (1,8)	N 8.8 (1) L 16.6 (1) M 12.1 (1)	N 9.1 (1) L 12.5 (1) M 10.7 (1)	
2,5-Dichloroaniline	95-82-9	N 10 (3) L 20 (3) M 14 (3)				Nc 3.8 carp (9)
4-Chlorophenol	106-48-9	Nc 141 (9)		N 24 (4) L 498 (4) M 111 (4)	Nc 133 (9)	
2,4,6-Trichlorophenol	88-06-2	Nc 204 (9)	Nc 231 (9)	Nc 41 (9)	Nc 13 (9)	
2-Chlorobenzoic acid	118-91-2					
PCBs as Aroclor 1248	1336-36-3	N 2.5 (2) L 7.5 (2) M 4.3 (2)	N 0.1 (1) L 0.4 (1) M 0.2-3.0 (1,8)	Nc 2.3 (9)	Nc 26 (9)	
2,3,7,8-TCDD	1746-01-6			N<.038-3 (5)		

TABLE 9
102 STREET LANDFILL
CHRONIC TOXICITY CONCENTRATIONS (ug/l) FOR SELECTED CHEMICALS
ENVIRONMENTAL ENDANGERMENT ASSESSMENT

CHEMICAL	CAS NO.	DAPHNIA	FATHEAD	TROUT	BLUEGILL	OTHER
Arsenic	7440-38-2	Nc 150 (9) M 1140 (7)	N 2130 (1) L 4300 (1) M 3026.4 (1)		Nc 676 (9)	
Cadmium	7440-43-9	N 0.16-0.21 (2) L 0.28-0.91 (2) M 0.21-2.2 (2,7)	N 37 (1,2) L 57 (1,2) M 45.9 (1,2)	N 7 (2) L 12 (2) M 9.2 (2)	N 31 (1,2) L 80 (1,2) M 49.8 (1,2)	N 12 catfish (2) L 17 (2) M 14.3 (2)
Mercury	7439-97-6	N<0.01-1.90 (2) L 0.04-3.20 (2) M 1.00-2.47 (2)	N<0.23-<0.26 (1)	Nc 8.5 (9) M 0.29-0.93 (6)		

FOOTNOTES

1. Barnthouse and Suter, 1986
2. USEDA Ambient Water Quality Criteria Documents
3. 3,4-DCA; Crossland and Hillaby, 1985
4. McCarty, et al., 1985
5. Mehrle, ef al., 1988
6. Biryl, et al., 1985
7. Spehar and Fiandt, 1986
8. McKim, 1977
9. Nc designates NOEC calculated from relationship logNOEC= -1.28 + .95logLC50 (Slooff et al., 1986)
   N=NOEC, L=LOEC, M=MATC

TABLE 10
102 STREET LANDFILL
BIOCONCENTRATION FACTORS (Log BCF) FOR SELECTED CHEMICALS
ENVIRONMENTAL ENDANGERMENT ASSESSMENT

CHEMICAL	CAS NO.	ALGAE	DAPHNIA	FATHEAD	TROUT	BLUEGILL	OTHER
Trichloroethylene	79-01-6	3.06 (4)			1.23-1.59 (1)	1.23-1.59 (1)	1.23 (2)
Benzene	71-43-2	1.47 (4)					0.72 fish, shellfish (2) 0.54 eel (1) 0.64 herring (1)
Monochlorobenzene	108-90-7						2.81 (2) 1-2 fish (1)
1,2,3-Trichlorobenzene	87-61-6						
1,2,4-Trichlorobenzene	120-82-1		2.15 (1)	3.45 (1)	3.11-3.50 (1)	2.91 (1)	2.94 fish (1)
1,2,3,4-Tetrachlorobenzene	634-66-2						
Hexachlorobenzene	118-74-1	4.3-4.4 (4)		4.2-4.8 (1,7)	3.7-4.3 (1)	3.1-4.3 (1)	4.36 (2)
alpha-Hexachlorocyclohexane	319-84-6				2.78 (1)		2.53 carp (1) 2.21 clam (1)
beta-Hexachlorocyclohexane	319-85-7						
gamma-Hexachlorocyclohexane	58-89-9	2.38 (4)			2.50 (1)		2.25 (2)
2,5-Dichloroaniline	95-82-9						
4-Chlorophenol	106-48-9						1.18 goldfish (1) 0.3-1.59 calculated (1)
2,4,6-Trichlorophenol	88-06-2	1.7 (1)					2.44 calculated (1)
2-Chlorobenzoic acid	118-91-2						
PCBs as Aroclor 1248	1336-36-3		3.58 (2)	5.44 (2)	3.48 (2)	4.72 (2)	3.26-5.27 (1)
2,3,7,8-TCDD	1746-01-6	3.6-3.95 (1)	4.3-4.4 (1)	3.9-4.2 (1)	3.2-4.59 (1,5,6)		5.11 (3)
Arsenic	7440-38-2						0.60 (2)
Cadmium	7440-43-9		2.5 (2)		0.48-2.73 (2)		
Mercury	7439-97-6	2.66-4.32 (2)		4.80 (2)	4.23-4.52 (2)	2.57-3.38 (2)	

FOOTNOTES

1. Hazardous Substances Data Bank (HSDB), Toxicological Data Network, National Library of Medici
2. USEPA Ambient Water Quality Criteria documents.
3. P.M. Cook, 1987.
4. Geyer, et. al., 1984.
5. Mehrle, et. al., 1988.
6. Branson, et al., 1985.
7. Schuytema, et. al., 1988.

TABLE 11
102 STREET LANDFILL
ESTIMATED ENVIRONMENTAL CONCENTRATIONS (ug/L) FOR SELECTED CHEMICALS
ENVIRONMENTAL ENDANGERMENT ASSESSMENT

CHEMICAL	SEDIMENT PORE WATER	SURFACE WATER-EMBAYMENT	SURFACE WATER-RIVER
Trichloroethylene	(1)	1.36E-3	1.36E-5
Benzene	(1)	1.54E+0	1.54E-2
Monochlorobenzene	(1)	4.13E-1	4.13E-3
1,2,3-Trichlorobenzene	2.7E+1	3.87E-2	3.87E-4
1,2,4-Trichlorobenzene	4.01E+2	1.60E-1	1.60E-3
1,2,3,4-Tetrachlorobenzene	3.55E+3	7.12E-2	7.12E-4
Hexachlorobenzene	3.2E+1 (3)	3.38E-4	3.38E-6
alpha-Hexachlorocyclohexane	1.34E+4 (3)	1.62E-2	1.62E-4
beta-Hexachlorocyclohexane	1.02E+3	4.07E-3	4.07E-5
gamma-Hexachlorocyclohexane	6.41E+2	1.10E-2	1.10E-4
2,5-Dichloroaniline	(1)	7.79E-3	7.79E-5
4-Chlorophenol	(1)	2.98E-2	2.98E-4
2,4,6-Trichlorophenol	4.03E+1	7.09E-4	7.09E-6
2-Chlorobenzoic acid	(1)	3.82E-3	3.82E-5
PCBs as Aroclor	(1)	6.60E-4	6.60E-6
2,3,7,8-TCDD	(1)	9.20E-9	9.20E-11
Arsenic	(2)	1.39E-3	1.39E-5
Cadmium	(2)	4.17E-4	4.17E-6
Mercury	(2)	3.57E-4	3.57E-6

FOOTNOTES

1. Not analyzed in sediments or no available Koc
2. Partition coefficients not available; strongly absorb to sediments
3. EEC exceeds water solubility limit of 6 ug/l for HCB and 1,630 ug/l for alpha HCH

TABLE 12
102 STREET LANDFILL
EEC/BC QUOTIENT FOR SELECTED CHEMICALS IN SURFACE WATER(1)
ENVIRONMENTAL ENDANGERMENT ASSESSMENT

CHEMICAL	DAPHNIA ACUTE	DAPHNIA CHRONIC	FATHEAD ACUTE	FATHEAD CHRONIC	TROUT ACUTE	TROUT CHRONIC
Trichloroethylene	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1
Benzene	0.1 < Q ≤ 10	< 0.1	0.1 < Q ≤ 10	< 0.1	> 10	< 0.1
Monochlorobenzene	0.1 < Q ≤ 10	< 0.1	0.1 < Q ≤ 10	< 0.1	0.1 < Q ≤ 10	< 0.1
1,2,3-Trichlorobenzene	(2)	(2)	(2)	(2)	0.1 < Q ≤ 10	< 0.1
1,2,4-Trichlorobenzene	0.1 < Q ≤ 10	< 0.1	0.1 < Q ≤ 10	< 0.1	> 10	< 0.1
1,2,3,4-Tetrachlorobenzene	(2)	(2)	0.1 < Q ≤ 10	< 0.1	(2)	(2)
Hexachlorobenzene	(2)	(2)	< 0.1	< 0.1	(2)	(2)
alpha-Hexachlorocyclohexane	(2)	(2)	(2)	(2)	(2)	(2)
beta-Hexachlorocyclohexane	(2)	(2)	(2)	(2)	(2)	(2)
gamma-Hexachlorocyclohexane	0.1 < Q ≤ 10	< 0.1	> 10	< 0.1	> 10	< 0.1
2,5-Dichloroaniline	0.1 < Q ≤ 10	< 0.1	(2)	(2)	(2)	(2)
4-Chlorophenol	0.1 < Q ≤ 10	< 0.1	(2)	(2)	0.1 < Q ≤ 10	< 0.1
2,4,6-Trichlorophenol	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1
2-Chlorobenzoic acid	(2)	(2)	(2)	(2)	(2)	(2)
PCBs as Aroclor 1248	(2)	< 0.1	0.1 < Q ≤ 10	< 0.1	0.1 < Q ≤ 10	< 0.1
2,3,7,8-TCDD	(2)	(2)	(2)	(2)	< 0.1	< 0.1
Arsenic	< 0.1	< 0.1	< 0.1	< 0.1	(2)	(2)
Cadmium	0.1 < Q ≤ 10	< 0.1	0.1 < Q ≤ 10	< 0.1	(2)	< 0.1
Mercury	0.1 < Q ≤ 10	0.1 < Q ≤ 10	0.1 < Q ≤ 10	< 0.1	0.1 < Q ≤ 10	< 0.1

FOOTNOTES

1. EEC = estimated environmental concentration; BC = acute or chronic benchmark toxicity concentration.
2. Toxicity data not available.

Q = EEC/BC quotient.

TABLE 13
102 STREET LANDFILL
REPRESENTATIVE INDICATOR CHEMICAL CONCENTRATIONS
FROM OFF-SITE SOIL ANALYSES

INDICATOR CHEMICAL	NUMBER OF DETECTS	REPRES. CONC. (1) (mg/kg)
1,2,4 Trichlorobenzene	3	1.41E-1
Hexachlorobenzene	7	3.38E-1
alpha-Hexachlorocyclohexane	3	2.29E-1
gamma-Hexachlorocyclohexane	2	1.09E-1
2,3,7,8 TCDD	1	8.00E-4
Mercury	7	1.53E+0

FOOTNOTES

1. Representative Concentration = average of all detects along area of significant chemical migration within vector areas FG through L, excluding samples I-3.5 and J-2.5 and two dioxin samples contained within area isolated by fence and gravel cover, and samples G-56, I-55 and J-55 which showed significantly increased concentrations away from the site.

TABLE 14

PARAMETER VALUES FOR CHRONIC HUMAN EXPOSURE

Parameter	Value	Reference
Adult body weight	70 kg	National Research Council, 1977
Adult water intake	2 l/day	National Research Council, 1977
Average fish and for sport fisherman consumption	1.4 g/day	Rodricks, 1985
Adult breathing rate	18.5 m^3/24 hr	Cleland and Kingsbury, 1977
Concentration of suspended particulate matter	0.07 mg/m^3	Hawley, 1985
Child body weight (1 to 6 yrs)	15 kg	LaGoy, 1987
Soil from which contaminants would be removed through skin absorption by child	0.0386 g/day	Rosenblatt and Spinney, 1986
Soil ingestion by 15 kg child	0.2 g/day	U.S. EPA, 1989b
Soil ingestion by adult	0.1 g/day	U.S. EPA, 1989b
De minimis risk for small populations (less than 10 million)	10^{-4}	Travis et al., 1987
Temperature	25°C	

TABLE 15

EXPOSURE PARAMETERS USED IN THE
ASSESSMENT OF RISK FROM SURFACE WATER CONTAMINANTS

Exposure	Exposure Parameters	Parameter Values for Swimming
Dermal	Duration of exposure	2.6 hrs/event (1)
	Frequency of exposure	25 events/yr (2)
	Exposed skin area	18,150 cm^2 (3)
	Body weight	70 kg (4)
Ingestion	Duration of exposure	2.6 hr/event
	Frequency of exposure	25 events/yr
	Ingestion rate	50 ml/hr (5)
	Body weight	70 kg

(1) Average value (USDOI, 1973)
(2) Upper bound limit (USDOI, 1986)
(3) Average adult whole body surface (Anderson et al., 1985)
(4) Average adult (National Research Council, 1977)
(5) Accidental ingestion while swimming (Versar, 1987)

TABLE 16
102 STREET LANDFILL
RISK CHARACTERIZATION

FOR EXPOSURES RELATED TO NONCARCINOGENIC EFFECTS

Indicator Chemical	Source/ Release	Daily Intake (mg/kg/day)	Total Daily Intake (DI) (mg/kg/day)	Acceptable Chronic Intake (AIC) (mg/kg/day)	DI/AIC
Benzene	Groundwater	2.32E-7	2.32E-7	1.43E-4	1.62E-3
Monochlorobenzene	Groundwater	7.99E-8	8.22E-8	2.70E-2	3.04E-6
	Storm Sewer	2.27E-9			
1,2,4-Trichlorobenzene	Groundwater	5.80E-8			
	Storm Sewer	3.63E-9	7.88E-8	2.00E-2	3.94E-6
	Soil	1.72E-8			
gamma-HCCH	Groundwater	2.09E-9			
	Storm Sewer	2.24E-10	1.55E-8	3.00E-4	5.17E-5
	Soil	1.32E-8			
2,5-Dichloroaniline	Groundwater	1.21E-8	1.21E-8	4.35E-3	2.78E-6
2-Chlorobenzoic acid	Groundwater	5.73E-10	5.73E-10	3.45E-3	1.66E-7
Arsenic	Groundwater	2.13E-10	2.13E-10	1.43E-3	1.49E-7
Cadmium	Groundwater	6.48E-11	6.48E-11	5.00E-4	1.30E-7
Mercury	Groundwater	1.85E-10			
	Storm Sewer	7.55E-12	1.85E-7	3.00E-4	6.17E-4
	Soil	1.85E-7			
				Hazard Index =	2.30E-3

TABLE 17
102 STREET LANDFILL RISK CHARACTERIZATION
FOR EXPOSURES RELATED TO CARCINOGENIC EFFECTS

Indicator Chemical	Exposure Route	Daily Intake (DI) (mg/kg/day)	Potency Slope (mg/kg/day)-1	Route Specific Risk	Total Chemical Specific Risk
GROUNDWATER EFFLUX					
Benzene	Drinking Water	1.47E-7	2.90E-2	4.26E-9	
	Swimming	8.43E-8	2.90E-2	2.45E-9	6.73E-9
	Fish Consumption	5.34E-10	2.90E-2	1.55E-11	
Hexachlorobenzene	Drinking Water	3.23E-11	1.69E+0	5.46E-11	
	Swimming	1.86E-11	1.69E+0	3.14E-11	5.44E-10
	Fish Consumption	2.71E-10	1.69E+0	4.58E-10	
alpha-HCCH	Drinking Water	1.54E-9	6.30E+0	9.70E-9	
	Swimming	8.86E-10	6.30E+0	5.58E-9	1.94E-8
	Fish Consumption	6.53E-10	6.30E+0	4.11E-9	
gamma-HCCH	Drinking Water	1.05E-9	1.33E+0	1.40E-9	
	Swimming	6.01E-10	1.33E+0	7.99E-10	2.79E-9
	Fish Consumption	4.43E-10	1.33E+0	5.89E-10	
2,4,6-Trichlorophenol	Drinking Water	6.74E-11	2.00E-2	1.35E-12	
	Swimming	3.87E-10	2.00E-2	7.74E-13	2.27E-12
	Fish Consumption	7.08E-12	2.00E-2	1.42E-13	
PCBs	Drinking Water	6.29E-11	7.70E+0	4.84E-10	
	Swimming	3.61E-11	7.70E+0	2.78E-10	3.47E-8
	Fish Consumption	4.40E-9	7.70E+0	3.39E-8	
2,3,7,8-TCDD	Drinking Water	8.77E-16	1.56E+5	1.37E-10	
	Swimming	5.04E-16	1.56E+5	7.86E-11	1.25E-8
	Fish Consumption	7.86E-14	1.56E+5	1.23E-8	

TABLE 17
102 STREET LANDFILL RISK CHARACTERIZATION
FOR EXPOSURES RELATED TO CARCINOGENIC EFFECTS

Indicator Chemical	Exposure Route	Daily Intake (DI) (mg/kg/day)	Potency Slope (mg/kg/day)-1	Route Specific Risk	Total Chemical Specific Risk
Arsenic	Drinking Water	1.33E-10	1.80E+1	2.39E-9	
	Swimming	7.64E-11	1.80E+1	1.38E-9	3.84E-9
	Fish Consumption	4.09E-12	1.80E+1	7.36E-11	
			TOTAL CHEMICAL SPECIFIC RISK FOR GROUNDWATER EFFLUX =		8.05E-8
STORM SEWER CONTRIBUTION					
alpha-HCCH	Drinking Water	2.55E-10	6.30E+0	1.61E-9	
	Swimming	1.47E-10	6.30E+0	9.26E-10	3.22E-9
	Fish Consumption	1.08E-10	6.30E+0	6.80E-10	
gamma-HCCH	Drinking Water	1.12E-10	1.33E+0	1.49E-10	
	Swimming	6.44E-11	1.33E+0	8.57E-11	2.98E-10
	Fish Consumption	4.74E-11	1.33E+0	6.30E-11	
PCBs	Drinking Water	2.55E-12	7.70E+0	1.96E-11	
	Swimming	1.46E-12	7.70E+0	1.12E-11	1.40E-9
	Fish Consumption	1.78E-10	7.70E+0	1.37E-9	
2,3,7,8-TCDD	Drinking Water	1.15E-15	1.56E+5	1.79E-10	
	Swimming	6.63E-16	1.56E+5	1.03E-10	1.64E-8
	Fish Consumption	1.03E-13	1.56E+5	1.61E-8	
			TOTAL CHEMICAL SPECIFIC RISK FOR STORM SEWER CONTRIBUTION =		2.13E-8

TABLE 17
102 STREET LANDFILL RISK CHARACTERIZATION
FOR EXPOSURES RELATED TO CARCINOGENIC EFFECTS

Indicator Chemical	Exposure Route	Daily Intake (DI) (mg/kg/day)	Potency Slope (mg/kg/day)-1	Route Specific Risk	Total Chemical Specific Risk
OFF-SITE SOIL					
Hexachlorobenzene	Soil Ingestion	9.88E-9	1.69E+0	1.67E-8	1.68E-8
	Particulate Inhalation	7.44E-11	1.69E+0	1.26E-10	
alpha-HCCH	Skin Absorption	2.10E-8	6.30E+0	1.32E-7	
	Soil Ingestion	6.69E-9	6.30E+0	4.21E-8	1.74E-7
	Particulate Inhalation	5.04E-11	6.30E+0	3.18E-10	
gamma-HCCH	Skin Absorption	1.00E-8	1.33E+0	1.33E-8	
	Soil Ingestion	3.19E-9	1.33E+0	4.24E-9	1.76E-8
	Particulate Inhalation	2.40E-11	1.33E+0	3.19E-11	
2,3,7,8-TCDD	Skin Absorption	7.34E-11	1.56E+5	1.15E-5	
	Soil Ingestion	2.34E-11	1.56E+5	3.65E-6	1.51E-5
	Particulate Inhalation	1.76E-13	1.56E+5	2.75E-8	
				TOTAL CHEMICAL SPECIFIC RISK FOR OFF-SITE SOIL =	1.53E-5
				TOTAL UPPER BOUND RISK =	1.54E-5

TABLE 18
102 STREET LANDFILL
ENVIRONMENTAL-BASED ENDPOINTS AND EXPECTED
ENVIRONMENTAL CONCENTRATIONS (ug/l) FOR SELECTED CHEMICALS
ENVIRONMENTAL ENDANGERMENT ASSESSMENT

CHEMICAL	NYSDEC (8)	USEPA (CHRONIC)	EEC-EMBAYMENT	EEC-PORE WATER
Trichloroethylene	11 GM		1.4E-3	(4)
Benzene	6 GM		1.5E+0	(4)
Monochlorobenzene	5 SI		4.1E-1	(4)
Trichlorobenzenes	5 SIN (2)		2.0E-1	4.28E+2
1,2,3,4-Tetrachlorobenzene		50 (9)	7.1E-2	3.5E+3
Hexachlorobenzene			3.4E-4	3.2E+1
Hexachlorocyclohexanes	0.01 SH (2)		3.1E-2	1.51E+4
2,5-Dichloroaniline			7.8E-3	(4)
Chlorinated Phenols	1 SL		3.1E-2	4.0E+1
2-Chlorobenzoic acid			3.8E-3	(4)
PCBs as Aroclor	0.001 SH		6.1E-4	(4)
2,3,7,8-TCDD	0.1E-5 SM		9.2E-9	(4)
Arsenic	190 SH		1.4E-3	(6)
Cadmium	1.4 SI (1)		4.2E-4	(6)
Mercury	0.2 GH		3.6E-4	(6)

FOOTNOTES

1. Calculated as exp (0.7852 ln135)-3.490, (NYSDEC, 1987)
2. Sum of all isomers
3. Total chlorinated phenols (4-CP, 246-TCP)
4. Not analyzed in sediments or no available Koc
5. 246-TCP only
6. Partition coefficients not available
7. 1245-TCB

NOTES

G- Guidance value
S- Standard
H- USEPA published criteria
M- Bioconcentration
I- Propagation
L- Aquatic food tainting
N- Chemical and aquatic species correlation

REFERENCES

8. NYSDEC, 1987
9. U.S.EPA, 1980a

TABLE 19
102 STREET LANDFILL
RISK (1) FROM BIOACCUMULATION FROM EXPOSURE TO EMBAYMENT WATER

CHEMICAL	DAPHNIA BCF (2) x P (3)	DAPHNIA RISK	FATHEAD BCF x P	FATHEAD RISK	TROUT BCF x P	TROUT RISK	OTHER BCF x P	OTHER RISK
Trichloroethylene					1.2E+3	NO	0.7E+3(4)	No
Benzene							0.01E+3(fish, shellfish)	No
Monochlorobenzene							0.02E+3(4)	No
1,2,3-Trichlorobenzene (6)								
1,2,4,-Trichlorobenzene	1.40E+2	No	2.8E+3	Possible	2.0E+3	Possible	0.87E+3 (fish)	No
1,2,3,4-Tetrachlorobenzene (6)								
Hexachlorobenzene			4.7E+6	Possible	1.5E+6	Probable	3.4E+6(4)	Probable
alpha-Hexachlorocyclohexane					9.6E+3	Probable	3.8E+3(carp)	Possible
beta -Hexachlorocyclohexane								
gamma-Hexachlorocyclohexane (6)					5.1E+3	Possible	2.8E+3(4)	Posssible
2,5-Dichloroaniline (6)								
4-Chlorophenol								
2,4,6-Trichlorophenol							2.8E+3(5)	Possible
2-Chlorobenzoic acid								
PCBs as Aroclor	2.85E+4	Probable	2.1E+6	Probable	2.3E+4	Probable	1.5E+5 (fish)	Probable
2,3,7,8-TCDD	1.23E+7	Probable	6.1E+6	Probable	4.3E+6	Probable	7.05E+6 (fish)	Probable
Arsenic (6)								
Cadmium (6)								
Mercury (6)								

TABLE 19
102 STREET LANDFILL
RISK (1) FROM BIOACCUMULATION FROM EXPOSURE TO EMBAYMENT WATER

FOOTNOTES

1. Risk defined as R=1-1.2E+3 - No Risk
   R=1.2E+3-14E+3 - Possible Risk
   R>14E+3 - Probable Risk
2. Bioconcentration factor
3. P=Persistence, half-life in days for surface water.
4. Aquatic life
5. Calculated BCF value
6. Half-life not available

TABLE 20
102 STREET LANDFILL
RISK TO WILDLIFE FROM FOOD CHAIN BIOMAGNIFICATION

CHEMICAL	FISH FLESH CONCENTRATION (1)	SPECIES CRITERIA/RISK (1) NON-CARCINOGENIC
Trichlorobenzene	3.26 (2)	1330
Hexachlorobenzene	0.03	330
Hexachlorocyclohexane	0.13	
PCBs as Aroclor	0.01	130
2,3,7,8-TCDD	2.00E-7	3.00E-6

FOOTNOTES

1. Based on trout BCF; concentration in ppb for whole fish.
2. 124-TCB.

APPENDIX A

Toxicity Profiles for Risk Assessment Chemicals

BENZENE

Introduction

Benzene is a clear, volatile, colorless, liquid aromatic hydrocarbon. It is an intermediate in the synthesis of phenols, synthetic rubber and styrene and is also a constituent of gasoline. The low organic carbon partition coefficient high water solubility and volatile nature of benzene are indicators of environmental mobility. In soil, much of the chemical near the surface will volatilize to the atmosphere, and benzene will evaporate fairly rapidly from water.

Benzene has a half-life of 6 days in air and 1-6 days in surface water (U.S. EPA, 1986). Limited data on biodegradability in soil indicate a half-life of about 100 days, an important factor being the acclimation of soil microorganisms (NLM, 1989).

Toxicity

Human Health Effects

Benzene is readily absorbed via oral and inhalation routes, and through the skin and human placenta. Toxic effects have been attributed to combined exposure by both respiration and skin absorption. The flux of benzene through epidermis measured in vitro (i.e., passive diffusion through the stratum corneum, which is taken to be the rate-limiting step in absorption from skin penetration) from air saturated with benzene at 31°C averages 1.0 ul/cm^2/hr (Blank and McAuliffe, 1985). Benzene is a known hematotoxin and carcinogen in humans. A causal relationship has been established between exposure to benzene by inhalation and myelogenous leukemia in humans. The limit of exposure that will result in hematologic effects in humans is not well defined but is thought to be <100 ppm. There is also evidence that benzene acts as a toxicant in male reproduction and it has been shown to be a teratogen in animal models (Doull et al., 1980). In acute animal inhalation studies, adult rats and mice were more resistant to the effects of benzene than young

animals (Manyashin, et al. 1968). These effects are dependent on the respiration rate and retention of benzene.

Benzene is classified as a known human carcinogen. Studies in animals have shown that carcinogenic action is potentiated when benzene is used as a solvent or carrier (Van Duuren, et al. 1963).

Environmental Effects

Acute toxicity values for the freshwater invertebrates Daphnia magna and Daphnia pulex were determined as 380,000 and 300,000 ug/l (U.S. EPA, 1980a; Canton and Adema, 1978). 96-hr LC50 values for fish ranged from 5,300 ug/l for rainbow trout to 100,000 ug/l for bluegill (De Graeve, et al. 1980; U.S. EPA, 1980a; Johnson and Finley, 1980). Maximum acceptable toxicant concentrations that will not result in chronic toxicity have been reported to be greater than 98,000 ug/l for Daphnia and 5,342 ug/l for trout (McCarty et al., 1985; U.S. EPA, 1980a). Bioconcentration factors for fish and shellfish are reported to range from 3.5 to 5.2 and are reported as 29.5 for algae (Barnthouse and Suter, 1986; McCarty et al., 1985; U.S. EPA, 1980a).

MONOCHLOROBENZENE

Introduction

Chlorobenzene is a colorless, volatile, chlorinated, organic solvent. It is used in a number of manufacturing procedures including the manufacture of dyestuffs and many pesticides. When released into the air, almost 75 percent will degrade within 7 days (Kanno and Nojima, 1979). In water, chlorobenzene will almost completely evaporate or degrade within 2 days. Biodegradation of chlorobenzene in soils proceeds slowly but fairly rapid mineralization occurs. Since chlorobenzene is fairly volatile, much of the chemical will be lost to the atmosphere. Evaporation is considered to be the predominant loss mechanism

from the soil surface (Wilson et al., 1981). Thus, it is concluded that chlorobenzene is not persistent in the environment.

Toxicity

Human Health Effects

Chlorobenzene is absorbed by humans through the lungs, gastrointestinal tract, and skin. Workers exposed to occupational levels of chlorobenzene showed signs of central nervous system depression (sedation and narcosis) and irritation of the eyes and respiratory tract. Toxicological studies with laboratory animals indicate that this chemical is absorbed rapidly through the lungs and the gastrointestinal tract. Prolonged exposure of animals to elevated levels of chlorobenzene has resulted in liver and kidney damage. Although no human reproductive effect has been reported, decreased spermatogenesis and gonadal structural effects have occurred in dogs and rats exposed to monochlorobenzene (U.S. EPA, 1980b).

The Carcinogen Assessment Group of the U.S. Environmental Protection Agency has not classified this chemical; however, no animal data were identified that would suggest that chlorobenzene is an animal carcinogen. There is some evidence that chlorobenzene may be mutagenic (bacterial gene mutation studies), but these results have been mixed (U.S. EPA, 1980b).

Environmental Effects

Acute toxicity of chlorobenzene to daphnids was reported in the range of 12.9 to 86 mg/l (LeBlanc, 1980; Cowgill et al., 1985). McCarty et al. (1985) reported a 96-hr LC50 of 4.7 mg/L for trout and values of 32 and 18 mg/l for fathead minnows and bluegills, respectively, are reported in the USEPA Ambient Water Quality Criteria document for chlorobenzene. Bioconcentration factors of 10 to 100 times are reported for fish in NLM (1989).

1,2,4-TRICHLOROBENZENE

Introduction

1,2,4-Trichlorobenzene (124-TCB) is a colorless, aromatic liquid. Major commercial uses are as a dye carrier, a synthesis intermediate, a dielectric fluid and as a solvent. Its strong tendency to adsorb on solids accounts for low volatility from soils and turbid water. Although mobility through groundwater is expected to be minimal due its high coefficient of absorption to soils, and the fact that it will not hydrolyze under environmental conditions, 124-TCB is found at appreciable concentrations in site groundwater. 124-TCB may biodegrade slowly in soil but is not expected to biodegrade in groundwater. If released to surface wafer, its major fate pathway would be adsorption to the sediments, although evaporation may be significant if suspended sediments are low. Absorption by microorganisms and a fairly high bioconcentration potential also could affect pathway distribution. 124-TCB is expected to be relatively persistent in soils and sediments. Half-lives in rivers have been reported from 4.2 hours to 28 days. In the atmosphere, reaction with photochemically produced hydroxyl radicals results in an estimated vapor phase half-life of 18.5 days. (NLM, 1989)

Toxicity

Human Health Effects

124-TCB is absorbed from the gastrointestinal tract, intact skin and lung. Principal toxicological concerns from which oral reference doses have been determined are associated with enzyme induction at dose levels of 10 mg/kg/day and increased liver-to-body ratios effective at higher oral dose levels in rat subchronic studies. One study reported no adverse effect levels of 14.8 and 8.9 mg/kg/day, respectively, for female and male rats. 124-TCB has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential (U.S. EPA, 1989a).

Environmental Effects

Holcombe et al. (1987), Carlson and Kosian (1987) and McCarty et al. (1985) reported 96-hr LC50s in the range of 1.5 to 3.0 mg/L for fathead minnows and trout. Acute values (48-hr LC50) for Daphnia range from 3.4 to 50 mg/l (Holcombe et al., 1987; NLM, 1989). Maximum acceptable toxicant concentrations of 290 to 707 ug/L for fatheads and 126 ug/L for trout were reported by Barnthouse and Suter (1986) and McCarty et al. (1985) with respective NOECs of 119 to 507 and 99 ug/l. Bioconcentration factors for Daphnia were reported as 141 and for fish as 813 to 3,162 (NLM, 1989).

HEXACHLOROBENZENE

Introduction

Hexachlorobenzene (HCB) is a very stable, unreactive compound. At environmental temperatures it is a crystalline solid practically insoluble in water. There is no evidence that hexachlorobenzene is broken down by physical or chemical processes in the environment; however, it can be dangerous when heated to decomposition, as it emits highly toxic fumes of chlorides (Torkelson and Rowe, 1981).

Hexachlorobenzene is a byproduct of the production of fungicides to control bunt in wheat and smut fungi of other grains. Other applications include: military, porosity controller in the manufacture of electrodes, intermediate in dye manufacture and organic synthesis, and wood preservative. In recent years hexachlorobenzene has become a concern because of its widespread distribution in the environment and presence in food products used for human consumption. Aerial dispersion appears to be the major pathway for this compound entering the environment. However, HCB in soil is very persistent. Transport to groundwater will be slow and evaporation to the air, if any, will be dependent upon the organic content of the soil due to its strong adsorption characteristic. If there is release to

the atmosphere, HCB is likely to be both in the vapor phase and adsorbed to particulate matter.

Toxicity

Human Health Effects

Absorption of HCB from ingestion of water is minimal at approximately 6 percent. The absorption rate is dependent upon the vehicle by which it is administered. No useful reports of subchronic oral exposure of humans to HCB were found; however, accidental chronic ingestion of HCB occurred in Turkey as a result of HCB-treated seed grain being ground into flour and made into bread. More than 600 patients were observed during a five-year period during which time a total of approximately 3,000 people were affected. The resultant syndrome, known as PCT, is a manifestation of disturbed porphyrin metabolism and caused blistering and epidermolysis of exposed parts of the body, particularly the face and hands. Hexachlorobenzene is absorbed slowly from the G.I. tract, primarily via the lymphatic system. Oral LD50s are: cats, 1700 mg/kg; rabbits, 2600 mg/kg; rats, 3500 mg/kg/ and mice, 4000 mg/kg (Diechmann, 1981). Investigations by Kuipper-Goodman et al. (1977) determined to "no-effect" level dose to be 0.5 mg/kg/day in rats.

Few reports were located about repeated inhalation exposure to HCB. One study found HCB levels in blood ranging from 0-310 ppb in 20 spray men exposed to HCB. These individuals exhibited no signs of PCT; no correlations were observed between blood concentrations of HCB and urinary porphyrin excretion of the liver enzymes SCOT, SGPT, or of serum LDH concentration.

Reproductive effects have been reported in animal models including dose-related ovarian effects in monkeys (from 8 mg/kg/day exposure over 60 days), and fertility problems and decreased fetal viability in rats exposed to 320 and 80 ppm HCB, respectively. HCB also

has been shown to be teratogenic in rats and mice and to cause tumors in hamsters (liver, thyroid), mice (liver) and rats (liver, kidney, adrenal, parathyroid) (U.S. EPA, 1980). Although HCB has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential (U.S. EPA, 1989a), it has been reported to be carcinogenic in mice and hamsters by the International Agency for Research on Cancer (IARC, 1979).

Environmental Effects

The acute aquatic toxicity of hexachlorobenzene is difficult to determine. Tests with a midge (Tamutarsis dissimilis), rainbow trout, fathead minnows, and bluegill (USEPA, 1980c) produced LC50 values at concentrations of hexachlorobenzene above what appeared to be its solubility limit. Johnson and Finley (1980) reported 96-hr LC_{50} values of 22, 12 and 14 mg/l for fatheads, bluegills, and bass and catfish, respectively. For Daphnia magna the toxicity of chlorinated benzenes generally tended to increase as the degree of chlorination increased making hexachlorobenzene the most toxic of the chlorinated benzenes (USEPA, 1980c).

Available data indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 250 ug/l and chronic toxicity occurs at concentrations as low as 50 ug/l. Bioconcentration factors of 1,259 to 63,095 are reported for algae and fish in NLM (1989) and the USEPA Ambient Water Quality Criteria document.

ALPHA-HEXACHLOROCYCLOHEXANE
GAMMA-HEXACHLOROCYCLOHEXANE

Introduction

The persistent organochlorine insecticide, hexachlorocyclohexane (HCCH), has eight stereo isomers of which four (alpha, beta, gamma, and delta) predominate in the technical product (Deo et al., 1982). Of the isomers, only gamma-HCCH is highly insecticidal (Newell et al.,

1987). Both alpha and gamma isomers have low vapor pressure and are slightly soluble in water. Because of these properties, these isomers are relatively persistent in both surface and subsurface soils. However, experimental results show considerable variability. Gamma-HCCH is expected to leach slowly to groundwater and volatilization can be a significant factor, particularly in dry soils. One study indicated minimal leaching for alpha-HCCH with 92% of the remaining chemical found within the top 20 cm of cultivated sandy loam soil 15 years following the original application. Anaerobic degradation may be an important loss mechanism in deeper soils, however, with one study showing disappearance of 26.2% of the applied alpha isomer and 63.8% of gamma-HCCH after 3 weeks incubation. (NLM, 1989)

The controlling mechanisms for loss of gamma-HCCH from river water seem to be a combination of biodegradation and hydrolysis, which result in a half-life range of 3 to 30 days. Although volatilization may be important under some conditions, half-lives based on this mechanism are estimated at 115 to 191 days. Alpha-HCCH is considered to be more stable than gamma-HCCH and may hydrolyze more slowly due to its smaller number of axial chlorines. After 3 months in an artificial lake impoundment, it was found that 15% of the gamma isomer was converted to the alpha isomer under aerobic conditions and 90% was converted under anaerobic conditions (NLM, 1989).

If release to the atmosphere occurs, both alpha- and gamma-HCCH react with hydroxyl radicals resulting in an estimated half-life of 1.63 days (NLM, 1989).

Toxicity

Human Health Effects

Alpha- and gamma-HCCH are absorbed through all routes of entry and both have been shown to cross the placenta. Both isomers are accumulated in fatty tissue and are

converted to a variety of less toxic chlorophenolic metabolites, identified in the liver and urine. Both isomers are central nervous system stimulants and act at nerve ganglia to derange nerve impulses.

From qualitative studies, gamma-HCCH appears to be rapidly absorbed from the GI tract; absorption is enhanced in lipid vehicles. No datum regarding the absorption of gamma-HCCH via inhalation is available in the documents reviewed. Administration of 10 ppm Lindane to the diets of rats for one to two years resulted in adverse effects to them and their offspring. Body weights were decreased after treatment for five months. Administration of 100 ppm gamma-HCCH in the diet to dogs resulted in enlarged livers without histopathological change. Fifty ppm gamma-HCCH in diets have been reported as the no effect level (NOEL) in a number of animal studies. Rat and dog studies show a correlation with exposure to gamma-HCCH and reduced fertility and other maternal and paternal complications. Technical grade hexachlorocyclo-hexane used in the above studies reportedly contained 72 percent of the gamma isomer and 13.6 percent of the alpha isomer.

Toxicity to gamma-HCCH by inhalation in the workplace has been reported. Pathological changes occurred in the liver of 55 percent of 59 female and 29 male workers exposed to hexachlorocyclohexane (composition not characterized) for 11 to 23 years. Chronic pancreatitis was observed in 5 percent of the workers. No exposure datum was reported in this study.

Short-term feeding studies of alpha-; beta-; and gamma-HCCH isomers conducted by Mueller et al. (1981) concluded that beta- and gamma-HCCH may exert neurotoxic effects. When chickens were fed levels of 0.1 to 10 mg/kg gamma-HCCH, Sauter and Steele (1972) found significantly reduced hatchability. In another study, Whitehead et al. (1972) did not find reduced hatchability at 100 mg/kg dietary level, although they did note decreased egg production. The NOEL reported by Whitehead et al. (1972) was 64 mg/kg dietary level as compared to the 10 mg/kg dietary level reported by Sauter and Steele (1972).

Both alpha and gamma isomers are classified as probable human carcinogens with a medium hazard rank (Table 3.5). Alpha-HCCH produces a strong neoplastic response in mouse liver and the induction of liver tumors by alpha-HCCH is promoted by PCBs.

Environmental Effects

The acute toxicity of gamma-HCCH to Daphnia has been reported in the range of 0.46 - 1.5 mg/l (48-hr LC50; NLM, 1989; Hermens et al., 1984). 96-hr LC50 values of 0.087, 0.002 - 0.032 and 0.068 mg/l have been reported for fathead minnows, trout and bluegill, respectively (NLM, 1989). A 96-hr LC50 value of 12 mg/l was reported by Johnson and Finley (1980) for bass exposed to alpha-HCCH.

Chronic NOECs of 8.8 - 9.1 ug/l have been reported for fatheads, trout and bluegill exposed to gamma-HCCH (Barnthouse and Suter, 1986), MATCs for these species were respectively 9.1 - 34.5, 12.1 and 10.7 ug/l.

2,5-DICHLOROANILINE

Introduction

2,5-Dichloroaniline (25-DCA) occurs as a crystalline mass at environmental temperatures but is slightly soluble and has a relatively low melting point. Very little toxicological data exist for 25-DCA, since testing has not been warranted due to its very limited production and potential release to the environment. Considerable data exist for other chloroaniline isomers, however, and structural and biological similarities appear to be sufficient to draw inferences on the environmental persistence and toxicological properties of 25-DCA (U.S. EPA, 1984; 1988).

25-DCA and its isomers are used as intermediates in dye production. Although little or no use of 25-DCA occurs at the present time (U.S. EPA, 1988), the production volume of dyes

using 25-DCA was reported to be >48,000 lbs in 1980 (U.S. EPA, 1984) and the selection of intermediates is known to vary from year to year within the industry.

Occurrences of residues and persistence in soil is a concern for the category of substituted anilines and there are conflicting reports of the ability of animals, plants and microbes to metabolize and tolerate these chemicals. The potential for these substances to bioaccumulate in aquatic organisms is low, based on K_{ow} values less than 100.

Dichloroaniline isomers are adsorbed so strongly by soil organic matter that they are not extractable by solvents. Limited hydrolysis of bound DCA occurs and it is mineralized in soil only very slowly.

Occurrences of dichloroaniline in soil and water have been directly attributed to the biological degradation of the herbicide propanil.

Toxicity

Human Health Effects

The principal known health concern associated with dichloroanilines is the formation of methyl hemoglobin which reduces the oxygen carrying capacity of red blood cells resulting in anemia and other oxygen depravation effects. Methemoglobinemia was induced in rats at an intraperitoneal dose of 250 mg/kg 34-DCA (NLM, 1989). There is considerable variability to methemoglobinemia sensitivity, with humans reported to be more sensitive than rats and less sensitive than rabbits. The formation of splenic sarcomas has been confirmed from exposures to aniline and 4-chloroaniline and has raised a similar concern for other members of this category (M. McCommas, personal communication, June 9, 1989).

Environmental Effects

Crossland and Hillaby (1985) reported a 96-hr LC50 of 0.29 to 0.44 mg/l for Daphnia and NOEC, LOEC and MATC values of 0.01, 0.02 and 0.014 mg/l for Daphnia exposed to 34-DCA. Geiger et al. (1988) reported 96-hr LC50 of 7.7 mg/l for fathead minnows exposed to 34-DCA.

2,4,6-TRICHLOROPHENOL

Introduction

2,4,6-Trichlorophenol (246-TCP) occurs as colorless crystals in its pure form at environmental temperatures. Its relatively high water solubility and low coefficient for absorption to organic soils indicate a tendency for mobility in groundwater. Biodegradation in soils can be a significant loss mechanism; total degradation in as little as 3 days has been reported. Volatilization and photo-minimeralization may be significant near soil surfaces but the capacity for leaching to groundwater would be more important in controlling the fate of this compound. (NLM, 1989)

In river water and experimental pools, 246-TCP has been shown to degrade fairly rapidly with half-lives reported of 6.3 days for biodegradation, and 2.1 hours for photodegradation and 2 days for volatilization near the surface (NLM, 1989).

In the atmosphere, estimated half-lives are 17 hours for photodegradation and 2.7 days for reaction with photochemically-produced hydroxyl radicals. Deposition in snow and rainfall, as well as dry deposition, is expected to be significant. (NLM, 1989)

Toxicity

Human Health Effects

245-TCP is absorbed from the gastrointestinal tract, from contact with the skin and, to a lesser extent, from the respiratory tract. 245-TCP causes dysfunction of liver cells by interfering with the oxidative phosphorylation of mitochondria thus severely impairing cellular energy production (NLM, 1989). 245-TCP is classified as a probable human carcinogen with a low cancer hazard ranking (Table 3.5).

Environmental Effects

LeBlanc (1980) reported an LC50 of 6 mg/l for Daphnia exposed to 246-TCP. 96-hr acute toxicity ranged from 4.5 to 9.2 mg/l for fathead minnows (NLM, 1989; Geiger et al., 1988), while Hattula et al. (1981) and Buccafusco et al. reported values of 1.1 and 0.32 mg/l, respectively, for trout and bluegill. No chronic toxicity datum was available for 246-TCP. A bioconcentration factor of 50 has been reported for algae and an estimated BCF calculated from log K_{ow} was 273, indicating a low to moderate potential for bioconcentration in aquatic systems (NLM, 1989).

2-CHLOROBENZOIC ACID

Introduction

2-Chlorobenzoic acid (2-CBA) occurs at environmental temperatures as monoclinic crystals. A very high water solubility and low K_{oc} indicate a high potential for leaching from soil and transport through groundwater. No potential is indicated for vaporization. 2-CBA is used as a preservative for glues and paints and as an intermediate in the manufacture of fungicides and dyes.

Biodegradation appears to be a major mechanism for loss from environmental media. All three isomers present on site yield the respective catechols in Pseudomanas cultures. Degradation by enriched natural microbial populations from sewage treatment plant effluent completely degraded 3-chlorobenzoic acid during seven days. (NLM, 1989)

Toxicity

Human Health Effects

No reference to human toxicological effects was found for the ortho, meta or para isomers of chlorobenzoic acid. Irritation of the skin and eye of rabbits was reported after 24 hours for 500 mg and 20 mg applications of 2-CBA, respectively (NIOSH, 1982). Rat oral and intraperitoneal LD50s were reported at 6,460 mg/kg and 2,300 mg/kg, respectively (NIOSH, 1983; NLM, 1989).

Environmental Effects

No reference to environmental toxicological effects was found for the ortho, meta or para isomers of chlorobenzoic acid.

POLYCHLORINATED BIPHENYLS (including mono-chlorobiphenyls)

Introduction

The polychlorinated biphenyls (PCBs) are a class of chemical that contain a large number of congeners (groups of similar molecular composition, with two or more possible structural forms). For PCBs, 209 separate congeners are possible. The physical, chemical, and biological properties can vary among congeners. Commercially, the chemical composition of a PCB product was varied to obtain desirable properties for specific uses. Because of limitations in separation technology and analytical methods, all products consisted of

mixtures of uncertain numbers of PCB chemicals and isomers. In practice, only about one-half of the possible 209 congeners occur in commercial PCB products.

Composition of commercial PCB products were conventionally coded to indicate the percent by weight of chlorine present, e.g., Aroclors 1248 and 1260 contained 48 percent and 60 percent chlorine, respectively.

The persistence of PCBs in the environment generally increases with an increase in the degree of chlorination. Although biodegradation of the higher chlorinated congeners occurs only slowly in soil systems, it is the only degradation process shown to be important. PCBs, particularly the higher chlorinated congeners, will not leach significantly from most soils; however, in the presence of organic solvents, such as may be present at waste sites, PCBs may leach quite rapidly to groundwater. Vapor loss from soils is very slow, yet volatilization may be a significant loss mechanism over time owing to the persistence and stability of PCBs. In surface water, PCBs will tend to partition to sediments and suspended particulates. Adsorption can immobilize PCBs for relatively long periods. However, resolution of PCBs has been shown to occur, resulting in redistribution of PCBs into the environment over a long period of time from sediments initially contaminated and serving as sinks for substantial quantities of these compounds. Volatilization of dissolved PCBs may be a major removal mechanism. PCBs are highly lipophilic and bioaccumulate in tissue from concentrations in water. (NLM, 1989)

In air, PCBs exist in both the vapor phase and in association with the particulate adsorption phase. The higher chlorinated congeners will be more likely to be found adsorbed to particulates. Reaction with hydroxyl radicals may be the dominant transformation process in the atmosphere, but is active primarily on the lower chlorinated congeners associated with the vapor phase. Physical removal is accomplished by wet and dry deposition. (NLM, 1989)

Toxicity

Human Health Effects

Acute or chronic human exposure to PCBs may cause eye irritation, chloracne (acne-like eruptions of the skin), scaly skin, nervous system disorders, jaundice or atrophy of the liver, reproductive effects, liver enzyme induction, liver dysfunction, behavior deficits in offspring, and adverse developmental effects. The toxicity of PCB products appears generally to increase with increasing degree of chlorination.

Most knowledge of the human health effects of PCB exposure comes from two sources: work site exposure and a 1969 accident in Japan (the Yusho incident) where people inadvertently ingested rice oil contaminated by a PCB product (Kanechlor 400) which had leaked from a heat exchanger. Symptoms associated with this acute, relatively high exposure to PCBs included: eye irritation, headache, fatigue, chloracne, nausea and vomiting, digestive disturbances, and liver dysfunction (Kohanawa et al., 1969).

There is also evidence that excessive exposure to PCBs may adversely affect reproductive outcome. Babies born to many Japanese mothers who had consumed PCB-contaminated rice oil had discolored skin, runny eyes, and were of below average birth weight. However, in many reported cases of PCB exposure (including the Yusho incident), the PCBs were contaminated by other more toxic chemicals, leaving questions as to whether the observed health effects resulted from PCBs, from other substances, or from the combination of chemicals. A study of patients from a Yusho-type incident that occurred in Taiwan in 1979 was compared with available data on patients from the 1969 accident in Japan. The results of the comparison suggest the polychlorinated dibenzofurans (PCDFs) were mainly responsible in the pathogenesis of Yusho (Kashimoto et al., 1981).

The greatest potential PCB-related human health concern (based primarily on the results of animal studies) are from long-term, low-level exposure. There is experimental evidence

of a carcinogenic effect when the highly chlorinated PCBs are administered at high doses to laboratory animals. Recent evidence suggests some behavioral and developmental deficiencies in infants whose mothers ate PCB-contaminated fish over a period of several years (Jacobson et al., 1984).

Studies of Yusho patients in Japan and some heavily exposed workers gave equivocal evidence of increased cancer, but a NIOSH study of 2,500 exposed electrical equipment workers found no increase in cancer, cardiovascular disease or neurological manifestations (Hamilton, 1983). The PCBs are considered to be known carcinogens in rodents and are classified as probable human carcinogens (U.S. EPA, 1986).

PCBs may not be acutely toxic until the dose level reaches the mg/kg range (U.S. EPA, 1980a). Allen and Norback (1976) have characterized the histology of skin lesions associated with feeding PCBs to monkeys. Hair loss from the face, head and neck occurred within one month after exposure to diets containing 100 mg/kg of Aroclor 1248. After three months' exposure, other symptoms included anorexia, weight loss, hypoproteinemia, hypolipidemia, anemia and subcutaneous edema.

In the rat, the single oral LD50 is 1,010 mg/kg, with a LDIO of 188 mg/kg (NIOSH, 1982). Rats fed diets of Aroclor 1254 totaling 1,000 mg/kg all died in 53 days (Hudson et al., 1984). Eisler (1986a) concluded that the total (sum of exposures) rat lethal dietary level of Aroclor 1254 is from 500 to 2,000 mg/kg for 1 to 7 week exposures. Bio-test Laboratories (1970) exposed rats to a diet of 6.25 mg/kg/day (Aroclor 1254) for 2 years without significant mortality, establishing this as a NOEL for mortality. The exposure of 28 mg/kg/day Aroclor 1254 (NCI, 1978) resulted in stomach lesions and cancer in rats exposed for 2 years. Spencer (1982) however, reported reduced fetal survival from 3.14 mg/kg of Aroclor 1254 in the daily diet of female rats during 9 days of pregnancy.

Marks et al. (1981) reported that mice exposed to 3,3',4,4',5,5'-hexachlorobiphenyl in gastric does of 2 mg/kg/day had significantly more deformed offspring and fewer per litter. Mice exposed to gastric doses as low as 1 mg/kg/day showed discolored liver in Marks et al.'s (1981) research. Talcott and Koller (1983) reported higher NOEL and LOELs with Swiss-Webster mice, which appear to be PCB resistant.

Exposure of mink to doses of hexachlorobiphenyls, such as 3,3',4,4',5,5'-hexachlorobiphenyl, as low as 0.1 mg/kg produced an LD50 in 3 months and completely inhibited reproduction (Aulerich et al., 1985). No adverse reproductive effects were noted with 2,3,6-TCBP or 2,4,5-TCBP. Aulerich et al. (1985) concluded that even 0.1 mg/kg (0.0225 mg/kg/day) of 3,3',4,4',5,5'-Hexachlorobiphenyl in the diet produced a number of toxic effects. Mink are among the most sensitive species to PCBs, and are the most sensitive wildlife species tested to date (Eisler, 1986a).

The European ferret is at least three times more tolerant of PCBs than mink (Bleavings et al., 1984) even though they are closely related. Bleavins et al. (1984) found complete reproductive failure after a 4 month feeding trial on 4.8 mg/kg/day Aroclor 1254, with a LD50 estimated at 20 mg/kg/day.

Zepp and Kirkpatrick (1976) report 1 mg/kg/day as the NOEL for the cottontail rabbit, with a LD50 of about 10 mg/kg/day Aroclor 1254 for a 12-week period. Domestic rabbits (Koller and Zinke, 1983) and raccoons proved more tolerant (Montz et al., 1982).

Carcinogenicity tests with PCBs have been performed in several animal species. In the opinion of the International Agency for Research on Cancer (IARC, 1979), there is experimental evidence for carcinogenic effects of PCBs in rodents. There is also evidence that PCBs may promote cancer effects of other chemicals (NLM, 1989).

Environmental Effects

In general, acute toxicity in aquatic organisms occurs in concentrations above 2 ug/l (U.S. EPA, 1980a). Ninety-six hour LC50 values for newly hatched fathead minnows (Pimephales promelas), were 15 ug/L for Aroclor 1242 and 7.7 ug/L for Aroclor 1254. Acute values for Aroclor 1248 ranged from 8 to 300 ug/l (U.S. EPA, 1980a). Defoe et al. (1976) conducted similar flow-through acute and chronic studies with fathead minnows using Aroclor 1248 and 1260. The calculated 30-day LC50 for newly hatched fathead minnows was 4.7 ug/L for Aroclor 1248 and 3.3 ug/L for Aroclor 1260. Stalling and Mayer (1972) determined 96-hr LC50 values ranging from 1,170 to 50,000 ug/L for cutthroat trout (Salmo clarkii), using Aroclor 1221-1268. In studies using rainbow trout (Salmo gairdneri). the doses of Aroclor 1242 through 1260 required to produce acute toxicity were greater than 1500 ug/L. However, Mayer and Ellersieck (1986) reported a 96-hr LC50 value of 54 ug/l for Aroclor 1248. Fifteen-day intermittent flow bioassays carried out with bluegills (Lepomis macrochirus) using Aroclor 1242, 1248, and 1254 resulted in LC50 values of 54, 76 and 204 ug/L, respectively.

Chronic toxicity values of 2.5 (NOEC), 7.5 (LOEC) and 4.3 (MATC) ug/l have been reported for Daphnia (U.S. EPA, 1980a). Barnthouse and Suter (1986) reported an NOEC of 0.1 ug/l for fathead minnows and MATCs in the range of 0.2 to 3.0 ug/l were reported by the former authors and McKim (1977).

2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN

2,3,7,8-Tetrachlorodibenzo-p-dioxin (2378-TCDD), as the most toxic member (Bellin and Barnes, 1987) of the category of chemicals made up of the congeners of chlorinated dibenzo-p-dioxins (CDDs) and chlorinated dibenzofurans (CDFs), has been selected as a risk assessment indicator chemical in Sections 3.2.1 and 3.2.2. However, since the risk characterization will be conducted on 2378-TCDD equivalents, which is the sum of all

detected CDDs and CDFs expressed in terms of equivalent concentrations of 2378-TCDD, the following toxicity profile discusses the entire category.

Introduction

CDDs and CDFs were never produced for commercial use but inadvertently occur as impurities in the production of many other chlorohydrocarbons or are produced during combustion and are present as trace impurities in some manufacturing chemicals and industrial wastes. CDDs are environmentally stable and, in living organisms, have a tendency to accumulate in fat. Eisler (1986b) has produced a synoptic review of dioxin hazards to fish, wildlife and invertebrates, in which he notes there are 75 CDD congeners; some are extremely toxic, while others are believed to be relatively innocuous.

The CDDs and CDFs are structurally similar classes of chemicals in that each contain a large number of homologues, or similar groups of specific molecular composition.

There is a group of 75 chlorinated dibenzo-p-dioxins and 135 chlorinated dibenzofurans. However, the available evidence strongly suggests that the toxic effects of this group of substances, including carcinogenicity, result from the 2378-TCDD isomer and 2,3,7,8-tetrachlorodibenzofuran (2378-TCDF) and from the higher chlorinated derivative of these compounds containing chlorine atoms in the 2,3,7,8 positions, referred to as 2,3,7,8 congeners (e.g., 1,2,3,4,7,8-hexachlorodibenzo-p-dioxin, etc.) (Bellin and Barnes, 1985). The toxicity of these congeners relative to 2,3,7,8-tetra species has been found to decrease as additional chlorine atoms are added (Bellin and Barnes, 1985).

2378-TCDD is very persistent in soil systems as indicated by a very high K_{oc} value and very low water solubility (Tables 3.1, 3.7). The most significant removal mechanism from the soil matrix appears to be microbial degradation. Although insufficient data exist to evaluate the contribution by bacterial populations, if any, fungal degradation has been supported by the work of Bumpus et al. (1985). Since fungi require aerobic conditions in order to

degrade chemicals, this mechanism normally would be limited to the top few centimeters of soil surface. Chemical degradation via hydrolysis and oxidation is very unlikely in view of an insignificant rate of these reactions in aquatic media (U.S. EPA, 1985b). Photolytic degradation is significant under certain conditions characterized by exposure of a very thin film to direct sunlight (Young, 1983), but this has little practical application to landfill contamination. Vapor-phase diffusion would be expected to occur but this would be an extremely slow process as indicated by the very low vapor pressure of 2378-TCDD. One estimate of mass transit by this mechanism through a soil column of 25 centimeters was on the order of one million years. Loss through the leaching process would be insignificant even when compared to the very slow loss due to vapor-phase transport (U.S. EPA, 1980a, 1985a; Freeman and Schroy, 1986). However, if co-solvents are present in a landfill, the mobility of 2378-TCDD may be significantly increased.

Based on the above considerations, a conservative approach to estimating residence time of 2378-TCDD in a soil column would be to assume all losses due to fungal degradation and that the soil is well aerated and at a constant temperature favoring this process. Using the data from Bumpus et al. (1985), a rate constant can be derived corresponding to a soil half-life of about 30 years.

If released to the atmosphere, 2378-TCDD is likely to be present primarily in the vapor phase (Eitzer and Hites, 1986). If this is true, the disappearance of 2378-TCDD in the air would be controlled by vapor-phase photolysis, which would account for a half-life in the range of 2 to 6 hours (Mill et al., 1987). By comparison, removal through oxidation by hydroxyl radicals would be insignificant (Podoll et al., 1986).

No significant degradation process has been identified for 2378-TCDD in groundwater. In surface waters, 2378-TCDD present in sediments would be slowly released to the water in dissolved form or as suspended sediment. Loss from the water surface would be expected to be primarily due to volatilization, although photolysis and biodegradation may be

contributing factors. As indicated by a very high bioconcentration factor, partitioning of 2378-TCDD from sediments and water to fish and other aquatic species may be significant (e.g., Thibodeaux et al., 1986; Kuehl et al., 1987).

Toxicity

Human Health Effects

Among the 75 possible CDD congeners, toxicity is generally greatest when chlorine atoms have been substituted at four to six of the available positions in the molecule and lower when only one, two, three, or seven to eight chlorines are attached. Twenty-two TCDD isomers are possible, but the symmetrical isomer 2378-TCDD is of greatest concern. Based on animal studies, some CDDs have been found to be very toxic chemicals (IARC, 1977; U.S. EPA, 1985a). Human exposure to CDD-contaminated materials has resulted in chloracne, limited nerve damage, liver abnormalities, and psychological disorders. Based on considerations involving the chlorine substitution structure of CDDs, 23 of the 75 CDDs are believed to have higher biological activity and therefore to be potentially toxic (OMOE, 1984, pp. 1-14, 1-15). Laboratory studies have shown 2378-TCDD to be carcinogenic, embryotoxic, and teratogenic in various animal species and to affect a number of organs and systems including thyroid, liver, skin, and the immunologic system. Controlled laboratory studies have shown wide variation in the acute toxicity of 2378-TCDD across several species.

U.S. EPA, ATSDR, FDA, and the National Research Center of Canada (NRCC) have all published carcinogenic risk estimates for 2378-TCDD. The basis for these risk estimates include the 2-year rat feeding study published by Kociba et al. (1978), with an independent pathology review performed by Squires and a National Cancer Institute (NCI) study using rats and mice. A carcinogenic risk estimate for a mixture of HxCDD isomers also has been published by U.S. EPA based on an NCI study in rats and mice (U.S. EPA, 1984d).

Studies have been performed on a variety of populations and environments that have been exposed to CDDs (U.S. EPA, 1985a). Generally during these studies, only the TCDDs or 2378-TCDD were measured. These studies (Webb, 1984; Knutsen, 1984; Hoffman et al., 1986) include:

- People who have sprayed the pesticide 2,4,5-T, which contains trace amounts of CDDs;
- Veterans who were exposed to 2,4,5-T aerial spraying in Vietnam;
- Environmental and human exposure to a cloud containing CDDs released during a process upset at Seveso, Italy;
- A study of Missouri residents exposed to TCDD over long periods of time.

A study of the frequency of birth defects has been conducted in the area around Seveso, Italy. Relative risks were calculated for specific categories of birth defects and for grouped malformations. The data failed to demonstrate any increased risk of birth defects associated with 2378-TCDD (Mastroiacovo et al., 1988).

The data from the literature indicate that high (acute) exposure to TCDD can result in chloracne (a skin disease), headaches, polyarthalgia, diarrhea, epistaxis, hemorrhagic cystitis, loss of appetite, gastritis, weight loss, eye irritation, fatigue, and other physical symptoms (U.S. EPA, 1985a, pp. 8-60 to 8-65). A recent review article on the toxicity of TCDD has concluded that while the chemical is highly toxic to laboratory animals, there is no direct evidence that it has any long-term effects on humans, except chloracne. While there was suggestive evidence from two epidemiological studies of an association between high level TCDD exposure and soft tissue sarcoma, these results have not been supported by additional studies (Tschirley, 1986).

A toxicological profile report on TCDD has been published recently by the Agency of Toxic Substances and Disease Registry (ATSDR). This report stated that TCDD causes chloracne in humans. There is also suggestive evidence that, in humans, TCDD can result in liver

damage, loss of appetite, weight loss, and digestive disorders. Although never demonstrated in humans, selective animal studies have shown that TCDD produces immune system toxicity, spontaneous abortions, and birth defects, and is a demonstrated carcinogen (ATSDR, 1987).

The toxicity of the CDFs have been studied much less than that of CDDs; they are considered to be somewhat less toxic than CDDs, but have a similar array of toxic effects. Among the possible 135 different CDF congeners, the toxicities are considered to be at least an order of magnitude lower than the equivalent CDD. The LD50s for 2378-TCDF in test species were: guinea pig, 5-10 ug/kg; monkey, 1 mg/kg; rat, >1 mg/kg; and mouse, >6 mg/kg (Kociba and Cabey, 1985). CDFs were found to be teratogenic in mice, but not mutagnic to Salmonella (OMOE, 1984).

TCDD is being found more frequently than reported earlier in the environment and in humans as analytical detection limits become lower. Rappe (1984) cites studies by Ryan and Williams (1983) reporting on the levels of 2378-TCDD in autopsy samples of human adipose tissue from the Great Lakes area in Canada. Twenty-two of the 23 samples contained 2378-TCDD at levels ranging from 4.1 to 130 ppt. Additional preliminary data indicated background levels ranging from 1 to 600 ppt in the general population (Rappe, 1984; citing personal communication from Ryan, Rappe, and Nygren; see also U.S. EPA 1985a at pp. 4-31 and 4-32 and RTI, 1982).

Acute and chronic toxicity laboratory studies of 2378-TCDD in mammals and birds demonstrate the morbid toxic effects of low levels of exposure to the contaminant. TCDD causes severe liver damage in rats, mice, and rabbits, chloracne-type skin lesions in monkeys and edema formation in birds (Gilbertson 1983). The LD50 for a single oral dose for the guinea pig is 0.2 to 2.5 ug/kg, 22 to 45 ug/kg for the rat, and 1,157 to 5,051 ug/kg for the hamster (Kociba and Schwetz, 1982; McConnell et al., 1978). All species are sensitive as indicated by acute oral toxicity, but the guinea pig (oral LD50 of 0.6-2 ug/kg)

is approximately 200 times more sensitive than the mouse (Kociba, and Cabey, 1985). The range of variation of acute toxicity (up to 8,400 X) may relate to different rates of metabolism of the parent compound (Eisler, 1986b). The parent compound is considerably more toxic than the metabolites (Neal, 1985).

The main targets of TCDD appear to be the liver in rats and the thymus in rats, guinea pigs, and mice according to Gupta et al. (1973). Atrophy of the thymus is a consistent finding in mammals poisoned by 2378-TCDD, and suppression of thymus-dependent cellular immunity, particularly in young animals, may contribute to their death (Eisler, 1986b).

Chronic exposure tests with TCDD on rats (Harris et al., 1973; Kociba et al., 1977; Kociba and Schwetz, 1982) indicate the toxicity of the contaminant. Harris et al. (1973) reported 0.1 ug/kg was the NOEL in a 31 day study, but subsequent 3-generation rat tests by Kociba and Schwetz (1982) found that even this level reduced litter size at birth, increased stillborns, and reduced survival and growth in F1 and F2 generations - 0.001 ug/kg/day was selected as the NOEL. Long-term studies in rhesus monkeys (U.S. EPA, 1985) seem to indicate that even 5 ng/kg diets (0.4 ng/kg/day dose) resulted in an effect although the toxicity endpoints were bone marrow and axial lymph node deficiencies. Higher treatment levels (50 ng/kg or does of about 1.7 ng/kg/day) resulted in abortion and weight loss in the rhesus monkey (Barsotti et al., 1979 - in Eisler, 1986b) with 7- to 29-month exposures; one year exposure at dietary level of 0.5 ug/kg resulted in death of 60% of the experimental animals. Barsotti et al. (1979 - in Eisler, 1986b) reported a NOEL of 0.017 ng/kg/day, but it was two orders of magnitude below the LOEL.

The effect of 2378-TCDD on birds is characterized by marked differences in sensitivity. Hudson et al. (1984) tested bobwhite quail, mallards, and ringed turtle doves and reported LD50 single oral doses of 15 ug/kg for the bobwhite, 108 ug/kg for the mallard, and 810 ug/kg for the ringed turtle dove. All three species showed similar signs of intoxication. Domestic chickens are even more sensitive (Kociba and Schwetz, 1982; Gilbertson, 1983).

Chick edema disease developed in the domestic chickens at 1 to 10 ug/kg in the diet after 21 days (Gilbertson, 1983). These effects are similar to those noted by NRCC (Natural Resource Council of Canada, 1981 - in Eisler 1986b) for fish-eating bird populations of the Great Lakes in the 1960s and 1970s. Edema signs include pericardial, subcutaneous and peritoneal edema, also liver enlargement and frequent death (Newell et al., 1987).

Gilbertson (1983) argues that there are only a small number of chick-edema-active compounds which include a few of the chlorinated biphenyls, dibenzo-p-dioxins, dibenzofurans, azobenzenes, and azoxybenzenes. The chick-edema-active compound, 2378-TCDD, is also the most embryotoxic, teratogenic, hepatoxic, porphyrinogenic of the chemicals affecting chick embryos (IJC 1986). Herring gull chicks showed signs of edema in the 1970s and it has declined since then; concentrations in herring gull eggs have declined from about 1,000 ug/kg to less than 80 ug/kg in 1981 (Gilbertson, 1983).

2378-TCDD and a mixture of two 2378-HxCDDs have been classified as probable human carcinogens (U.S. EPA, 1986; U.S. EPA, 1989a). No supporting evidence has been found to suggest that any CDD or CDF congener is a suspect promoter of carcinogenicity initiated by other chemicals.

Environmental Effects

Acute toxicity of 2378-TCDD to trout, as derived from the NOEC value, is 5.0E-7 mg/l (Sloof et al., 1986). Chronic toxicity concentrations (NOEC) ranging from <.038 to 3μg/l for trout are reported by Mehrle et al. (1988). Bioconcentration factors for fish are reported to range from 1585 to 38,905, for Daphnia from 19,953 to 25,119, and for algae from 3981 to 8913 (Branson et al., 1985; Schuytema et al., 1988; NLM, 1989).

ARSENIC

Introduction

Arsenic is a naturally occurring element and is found in numerous soil types. Elemental arsenic is a silver-gray, cystalline material with a very high melting point, and is virtually insoluble in water (Table 3.7) and body fluids (U.S. EPA, 1976). Arsenic is used in metallurgy for the hardening of copper, lead and alloys. It is also used in the manufacture of certain types of glass and in medical applications (Merck & Co., 1968).

Arsenic can enter aquatic media through wet and dry deposition, runoff from soils, and from industrial discharge. The major source of atmospheric arsenic is coal combustion. The element enters soil from wet and dry precipitation of atmospheric arsenic, runoff of surface waters, and disposal of arsenic containing waters. Arsenic in aquatic sediments can become biologically available via methylation by bacteria (Lemo et al., 1983).

In aquatic systems, arsenic is rarely found as a free element (zero oxidation state). Soluble inorganic arsenate (+5 valance) predominates under normal conditions since it is thermodynamically more stable in water than arsenite (+3 valance). In aerobic water, arsenic III slowly oxidizes to arsenic V at neutral pH. Arsenic entering surface waters associated with particulates would be expected to remain so and to accumulate in sediments. Arsenic in solution may form complexes with low molecular weight dissolved organic material. Arsenic concentrations in fish and other aquatic organisms are reported to be low and residues are not expected to be a problem at environmental concentrations that are not directly toxic. In an arsenic-rich river a study indicated that only 3 to 4% of the annual arsenic input was bioaccumulated. (NLM, 1989)

Biodegradation may be a significant mechanism for loss of arsenic from both soils and sediments. Many organisms in these media have the ability to reduce and methylate

arsenical compounds, producing volatile arsines (arsines are a potential hazard only in enclosed spaces). (NLM, 1989)

Toxicity

Human Health Effects

Inorganic arsenic compounds are found to be almost completely absorbed by the gastrointestinal tract but absorption of other complexes of arsenic vary from 40 to 90 percent. Arsenic is poorly absorbed through the skin. Chronic intake of arsenic in humans is associated with skin disorders and peripheral circulatory disease. The concentrations of arsenic in drinking water, associated with peripheral circulatory disease ranged from 0.001 to 1.82 mg/l. Absorbed arsenic is promptly distributed in various organs and is stored in the bones, skin and keratinized tissues. It tends to accumulate in the liver, from which it is slowly released (NLM, 1989).

The reported acute toxicity of the arsenic compound As_2O_3 varies from 8 to 500 mg/kg body weight (Harrison, 1958). LD50 values range from 15.1 to 23.6 mg/kg in solution and 145.2 to 214 mg/kg dry (non-solution form) for the rat, and from 39.4 to 42.6 mg/kg for the mouse (Clayton, 1981). Marked hemorrhaging of the gastro-intestinal tract, as well as fatty degeneration of liver cells and cellular necrosis has been reported.

Arsenic has been found to be mutagenic in the fruit fly (Drosophila melanogaster) when chemically induced, and is also teratogenic (U.S. EPA, 1986). It has been found to cause hemagioendothelial tumors or angiosarcomas in the human liver (Poppes et al., 1978). Because arsenic has been implicated in the etiology of human cancer (skin), it has been classified as a known human carcinogen (U.S. EPA, 1986).

Environmental Effects

Acute toxicities for aquatic organisms tested with sodium arsenite showed LC50 values of 1,044 and 812 ug/l for the water fleas Daphnia magna and Simocephalus serrulatus, respectively. Rainbow trout (Salmo gairdneri) and bluegills (Lepomis macrochirus) had values of 13,340 and 41,760 ug/l, respectively. A 96-hr LC50 of 21,200 ug/l for bluegill is reported in the U.S. EPA Ambient Water Quality Criteria document. Sodium arsenate produced values of 7,400 ug/l for cladoceran and 10,800 ug/l for rainbow trout. Monosodium methanearsenate LC50 values ranged from 506,000 to 1,403,000 ug/l for the crayfish (Procambarus sp.) and the channel catfish (Ictalurus punctatus). Values for the fathead minnow are reported to range from 14,900 - 82,400 ug/l (NLM, 1989). Barnthouse and Suter (1986) reported chronic toxicity values of 2,130 (NOEC) and 4,300 (LOEC) ug/l for fatheads with a MATC of 3,026 ug/l.

CADMIUM

Introduction

Cadmium is a soft, ductile, silver-white metal which has an atomic weight of 112.40 and a specific gravity of 8.642. It is found in many soil profiles under natural conditions. It is readily attacked by most acids and occurs naturally as zero valence (metal and alloys) and the +2 valence (compounds) (U.S. EPA, 1980f). Commercial uses include electroplating and engraving, as a constituent of easily fusible alloys, of soft solder and of solder for aluminum, in the manufacture of cadmium-vapor lamps, photoelectric cells, Ni-Cd batteries and as an amalgam in dentistry (Merck & Co., 1968).

In the atmosphere, cadmium is expected to be present as dust and fumes from smelting of ores, manufacturing of metallic-alloys, reprocessing of cadmium-containing alloys, recycling of scrap steel, emissions of coal-fired power plants, and incineration of solid wastes. The principal removal mechanisms for atmospheric cadmium are wet and dry deposition (Fishbein, 1981). The predominant fate of cadmium in aquatic media is

sedimentation through binding onto clays or organic matter (U.S. EPA 1980f), and precipitation with manganese oxide, iron oxide and hydrates (NLM, 1989). Cadmium is also bound onto soil particles with increased binding as the organic matter content of soil increases (U.S. EPA, 1980f).

Transport in soils may be in the form of nitrate, chloride, carbonate complexes, hydroxide complexes, ammonia complexes, and as chelated and other organo-metallic complexes resulting from organic decay. In the aquatic environment cadmium is relatively mobile and may be transported in solution as either hydrated cations or as organic or inorganic complexes. Photolysis is not an important removal process. (NLM, 1989)

Toxicity

Human Health Effects

Several studies indicate that cadmium is poorly absorbed by the gut. In Japan, however, chronic exposure through cadmium contaminated food items caused what is known as itai-itai disease. This disease was caused by cadmium's ability to weaken bone structure. A lowest observable effect level dose is estimated to be 301 ug cadmium/kg body weight/day.

Cadmium is absorbed more efficiently by the lungs than by the gut in humans. Respiratory problems and possible renal complications are associated with chronic occupational exposure to cadmium fumes. A major nonoccupational source of respirable cadmium is cigarettes. The estimated intake from this source is 0.1 to 0.2 micrograms per cigarette. Inhalation of welding fumes from metals containing cadmium may result in "metal fume fever" an acute reaction to occupational levels of cadmium.

Cadmium also has long-term toxicity to mammals. It is particularly effective because it is not eliminated by the organism and accumulates mostly in the bones (Ramade, 1987).

There is also evidence of a correlation between cadmium poisoning and arterial hypertension (Ramade, 1987).

Cadmium has been demonstrated to be teratogenic and to reduce fertility following intravenous, intraperitoneal, and subcutaneous administration (U.S. EPA, 1980f). It appears that cadmium can be mutagenic under some conditions; however, the relationship between mutagenicity and carcinogenicity is not as well correlated for metals as for some other classes of carcinogens.

Cadmium has been classified by the U.S. EPA Carcinogen Assessment Group as a probable human carcinogen by inhalation based upon limited human exposure data. Insufficient data exist to classify cadmium as carcinogenic to humans by the oral route (U.S. EPA, 1989a).

Environmental Effects

Acute toxicity of cadmium ranged from 33-63 ug/l for Daphnia, 8-12,000 ug/l for fathead minnows and 21,00 ug/l for bluegill (USEPA, 1980a; Birge et al, 1985). Birge et al. (1985) also reported concentrations of 140 ad 240 ug/l for carp and bass, respectively, in 96-hr LC50 tests.

Bertram and Hart (1979) and Ingersoll and Winner (1982) found chronic toxicity occurring in water fleas at less than 1 to 10 ug/L, respectively. Effects on salmonids and many invertebrates have been observed at 5 ug/L or less (Ingersoll and Weiner, 1982). MATCs of 0.2 - 2.2, 46, 9.2, 50 and 14 ug/l have been reported for Daphnia, fathead minnow, trout, bluegill and catfish, respectively (U.S. EPA AWQC; Barnthouse and Suter, 1986; Spehar and Fiandt, 1986). Bioconcentration factors for cadmium in freshwater fish range from 3 for brook trout muscle (Benoit et al., 1976) to 12,400 for the whole body of mosquito fish (Giesy, et al., 1977).

MERCURY

Introduction

Mercury is classified as a heavy metal and is a silvery liquid in its elemental state. Mercury ore is ubiquitous in rock formations and is also present under natural conditions in soils. Besides a variety of inorganic compounds, mercury forms a number of organic chemicals. Organic compounds are toxicologically and environmentally significant because they can be rapidly absorbed by living organisms. Mercury is used in a number of industrial processes and in fungicides. The largest industrial use of mercury is for the manufacture of electrical apparatus. Mercury in ambient air is largely derived from electrical and chloroalkali industries and the burning of fossil fuels.

Mercury binds strongly to soils, especially to soil organic material. Elemental mercury is very immobile in soil; thus, leaching to ground water is unlikely. Organomercury develops in soil within 30 to 50 days after application in the presence of biological activity. Availability of soil mercury to plants is very low and there is a root barrier to translocation of mercury to plant tops. Volatility of elemental mercury accounts for high atmospheric concentrations, reported at 20 to 200 ug/m^3 near areas containing high soil levels of 10 mg/kg (normal atmospheric concentration = 5 ug/m^3). Mercury binds to atmospheric dust particles which are removed by wet and dry deposition. Photodegradation may be important in the removal of vapor-phase mercurial compounds. (NLM, 1989)

In aquatic systems, mercury appears to bind to dissolved matter or fine particulates and to bed sediments. Mercury in sediments can be desorbed into the water column, transported and redeposited. Methylation is likely to occur in the top two centimeters of the sediments. Virtually any mercurial compound can be microbially converted to methyl mercury. Methylation is also reported to occur among zooplankton. Transformation to volatile chemicals, such as dimethyl mercury may result in loss to the atmosphere or conversion by photolysis to methylmercury and return to the surface water. Mercury bioaccumulates

and concentrates in the food chain, which then acts as a significant transport mechanism (NLM, 1989)

Toxicity

Human Health Effects

Metallic mercury is poorly absorbed from the gastrointestinal tract. However, ingested organic mercury, especially methylmercury, is almost completely absorbed. The respiratory system is much more efficient in absorbing mercury vapor. Studies indicate that approximately 80 percent of the inhaled vapor is absorbed. Data regarding the absorption of organic mercury via the lungs have not been identified in the literature reviewed.

The toxic effects of chronic exposure to elevated levels of mercury have been well documented. Exposure to elevated organic mercury resulted in Japan after consumption of tainted seafood and in Iraq after people consumed bread made from grain treated with a mercury-containing fungicide. The major signs of toxicity were twitching in the extremities, impaired peripheral field of vision, slurred speech, and unsteadiness of gait and limb. Maximum severity of symptoms occurred several weeks after the end of exposure.

The Carcinogen Assessment Group of the U.S. Environmental Protection Agency has classified this chemical as "not classifiable as to human carcinogenicity." No human datum is available, and animal and supporting data are inadequate to classify possible human effects (U.S. EPA, 1989a).

Environmental Effects

The U.S. EPA Ambient Water Quality Criteria document reports acute 96-hr LC50 values of 5, 15 and 24-400 ug/l for Daphnia, fathead minnows and trout, respectively. A value of 350 ug/l has been reported for catfish (NLM, 1989). MATC values in the ranges of 1 - 2.47 ug/l, for Daphnia have been reported. Barnthouse and Suter (1986) reported a NOEC of <0.23 - <0.26 ug/l for fathead minnows and Biryl et al. (1985) reported a MATC of 0.29 - 0.93 ug/l for trout.

APPENDIX B

Public Health Assessment Exposure Profiles

Groundwater Efflux

Monochlorobenzene

Intake from Drinking Water Ingestion = $\frac{1.38\ E\text{-}6 \times 2}{70}$

= 3.94×10^{-8} mg/kg/day.

Intake from Dermal Absorption While Swimming = $\frac{1.38\ E\text{-}4 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$

= 5.10×10^{-9} mg/kg/day

Intake from Accidental Ingestion While Swimming = $\frac{1.38\ E\text{-}4 \times 0.05 \times 2.6 \times 0.0685}{70}$

= 1.76×10^{-8} mg/kg/day

Total Intake due to Swimming = 2.27×10^{-8} mg/kg/day

Intake from Fish Consumption = $\frac{1.38\ E\text{-}6 \times 645 \times 14\ E\text{-}4}{70}$

= 1.78×10^{-8} mg/kg/day

Sum of Intakes = 7.99×10^{-8} mg/kg/day

1,2,4-Trichlorobenzene

Intake from Drinking Water Ingestion

$$= \frac{5.32\ E\text{-}7 \times 2}{70}$$

$$= 1.53 \times 10^{-8}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{5.32\ E\text{-}5 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 1.96 \times 10^{-9}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{5.32\ E\text{-}5 \times 0.05 \times 2.6 \times 0.0685}{70}$$

$$= 6.77 \times 10^{-9}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 8.73 \times 10^{-9}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{5.32\ E\text{-}7 \times 3200 \times 14\ E\text{-}4}{70}$$

$$= 3.40 \times 10^{-8}\ mg/kg/day$$

Sum of Intakes

$$= 5.80 \times 10^{-8}\ mg/kg/day$$

Hexachlorobenzene

Intake from Drinking Water Ingestion

$$= \frac{1.13\ E\text{-}9 \times 2}{70}$$

$$= 3.23 \times 10^{-11}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{1.13\ E\text{-}7 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 4.17 \times 10^{-12}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{1.13\ E\text{-}7 \times 0.05 \times 2.6 \times 0.0685}{70}$$

$$= 1.44 \times 10^{-11}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 1.86 \times 10^{-11}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{1.13\ E\text{-}9 \times 12000 \times 14\ E\text{-}4}{70}$$

$$= 2.71 \times 10^{-10}\ mg/kg/day$$

Sum of Intakes

$$= 3.22 \times 10^{-10}\ mg/kg/day$$

alpha-Hexachlorocyclohexane

Intake from Drinking Water Ingestion

$$= \frac{5.40\ E\text{-}8 \times 2}{70}$$

$$= 1.54 \times 10^{-9}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{5.40\ E\text{-}6 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 1.99 \times 10^{-10}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{5.40\ E\text{-}6 \times 0.05 \times 2.6 \times 0.0685}{70}$$

$$= 6.87 \times 10^{-10}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 8.86 \times 10^{-10}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{5.40\ E\text{-}8 \times 605 \times 14\ E\text{-}4}{70}$$

$$= 6.53 \times 10^{-10}\ mg/kg/day$$

Sum of Intakes

$$= 3.08 \times 10^{-9}\ mg/kg/day$$

gamma-Hexachlorocyclohexane

Intake from Drinking Water Ingestion = $\frac{3.66\ E\text{-}8 \times 2}{70}$

= 1.05×10^{-9} mg/kg/day

Intake from Dermal Absorption While Swimming = $\frac{3.66\ E\text{-}6 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$

= 1.35×10^{-10} mg/kg/day

Intake from Accidental Ingestion While Swimming = $\frac{3.66\ E\text{-}6 \times 0.05 \times 2.6 \times 0.0685}{70}$

= 4.66×10^{-10} mg/kg/day

Total Intake Due to Swimming = 6.01×10^{-10} mg/kg/day

Intake from Fish Consumption = $\frac{3.66\ E\text{-}8 \times 605 \times 14\ E\text{-}4}{70}$

= 4.43×10^{-10} mg/kg/day

Sum of Intakes = 2.09×10^{-9} mg/kg/day

2,5-Dichloroaniline

Intake from Drinking Water Ingestion = $\frac{2.60\ E\text{-}7 \times 2}{70}$

= 7.43×10^{-9} mg/kg/day

Intake from Dermal Absorption While Swimming = $\frac{2.60\ E\text{-}5 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$

= 9.61×10^{-10} mg/kg/day

Intake from Accidental Ingestion While Swimming = $\frac{2.6\ E\text{-}5 \times 0.05 \times 2.6 \times 0.0685}{70}$

= 3.31×10^{-9} mg/kg/day

Total Intake Due to Swimming = 4.27×10^{-9} mg/kg/day

Intake from Fish Consumption = $\frac{2.60\ E\text{-}7 \times 72 \times 14\ E\text{-}4}{70}$

= 3.74×10^{-10} mg/kg/day

Sum of Intakes = 1.21×10^{-8} mg/kg/day

2,4,6-Trichlorophenol

Intake from Drinking Water Ingestion

$$= \frac{2.36\ E\text{-}9 \times 2}{70}$$

$$= 6.74 \times 10^{-11}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{2.36\ E\text{-}7 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 8.72 \times 10^{-12}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{2.36\ E\text{-}7 \times 0.05 \times 2.6 \times 0.0685}{70}$$

$$= 3.00 \times 10^{-11}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 3.87 \times 10^{-11}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{2.36\ E\text{-}9 \times 150 \times 14\ E\text{-}4}{70}$$

$$= 7.08 \times 10^{-12}\ mg/kg/day$$

Sum of Intakes

$$= 1.13 \times 10^{-10}\ mg/kg/day$$

2-Chlorobenzoic Acid

Intake from Drinking Water Ingestion = $\frac{1.27\ E\text{-}8 \times 2}{70}$

= 3.63×10^{-10} mg/kg/day

Intake from Dermal Absorption While Swimming = $\frac{1.27E\text{-}6 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$

= 4.69×10^{-11} mg/kg/day

Intake from Accidental Ingestion While Swimming = $\frac{1.27E\text{-}6 \times 0.05 \times 2.6 \times 0.0685}{70}$

= 1.62×10^{-10} mg/kg/day

Total Intake Due to Swimming = 2.09×10^{-10} mg/kg/day

Intake from Fish Consumption = $\frac{1.27E\text{-}8 \times 5 \times 14\ E\text{-}4}{70}$

= 1.27×10^{-12} mg/kg/day

Sum of Intakes = 5.73×10^{-10} mg/kg/day

PCBs

Intake from Drinking Water Ingestion = $\frac{2.20\ E\text{-}9 \times 2}{70}$

= 6.29×10^{-11} mg/kg/day

Intake from Dermal Absorption While Swimming = $\frac{2.20\ E\text{-}7 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$

= 8.13×10^{-12} mg/kg/day

Intake from Accidental Ingestion While Swimming = $\frac{2.20\ E\text{-}7 \times 0.05 \times 2.6 \times 0.0685}{70}$

= 2.80×10^{-11} mg/kg/day

Total Intake Due to Swimming = 3.61×10^{-11} mg/kg/day

Intake from Fish Consumption = $\frac{2.20\ E\text{-}9 \times 100{,}000 \times 14\ E\text{-}4}{70}$

= 4.40×10^{-9} mg/kg/day

Sum of Intakes = 4.50×10^{-9} mg/kg/day

2,3,7,8-Tetrachlorodibenzo-p-dioxin

Intake from Drinking Water Ingestion

$$= \frac{3.07\ E\text{-}14 \times 2}{70}$$

$$= 8.77 \times 10^{-16}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{3.07\ E\text{-}12 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 1.13 \times 10^{-16}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{3.07\ E\text{-}12 \times 0.05 \times 2.6 \times 0.0685}{70}$$

$$= 3.91 \times 10^{-16}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 5.04 \times 10^{-16}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{3.07\ E\text{-}14 \times 128{,}000 \times 14\ E\text{-}4}{70}$$

$$= 7.86 \times 10^{-14}\ mg/kg/day$$

Sum of Intakes

$$= 8.00 \times 10^{-14}\ mg/kg/day$$

Arsenic

Intake from Drinking Water Ingestion = $\frac{4.65\ E\text{-}9 \times 2}{70}$

= 1.33×10^{-10} mg/kg/day

Intake from Dermal Absorption While Swimming = $\frac{4.65\ E\text{-}7 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$

= 1.72×10^{-11} mg/kg/day

Intake from Accidental Ingestion While Swimming = $\frac{4.65E\text{-}7 \times 0.05 \times 2.6 \times 0.0685}{70}$

= 5.92×10^{-11} mg/kg/day

Total Intake Due to Swimming = 7.64×10^{-11} mg/kg/day

Intake from Fish Consumption = $\frac{4.65\ E\text{-}9 \times 44 \times 14\ E\text{-}4}{70}$

= 4.09×10^{-12} mg/kg/day

Sum of Intakes = 2.13×10^{-10} mg/kg/day

Cadmium

Intake from Drinking Water Ingestion

$$= \frac{1.39\ E\text{-}9 \times 2}{70}$$

$$= 3.97 \times 10^{-11}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{1.39\ E\text{-}7 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 5.14 \times 10^{-12}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{1.39\ E\text{-}7 \times 0.05 \times 2.6 \times 6.5\ E\text{-}4}{70}$$

$$= 1.77 \times 10^{-11}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 2.28 \times 10^{-11}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{1.39\ E\text{-}9 \times 81 \times 14\ E\text{-}4}{70}$$

$$= 2.25 \times 10^{-12}\ mg/kg/day$$

Sum of Intakes

$$= 6.48 \times 10^{-11}\ mg/kg/day$$

Mercury

Intake from Drinking Water Ingestion = $\frac{1.19\ E\text{-}9 \times 2}{70}$

= 3.40×10^{-11} mg/kg/day

Intake from Dermal Absorption While Swimming = $\frac{1.19\ E\text{-}7 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$

= 4.40×10^{-12} mg/kg/day

Intake from Accidental Ingestion While Swimming = $\frac{1.19\ E\text{-}7 \times 0.05 \times 2.6 \times 6.5\ E\text{-}4}{70}$

= 1.51×10^{-11} mg/kg/day

Total Intake Due to Swimming = 1.95×10^{-11} mg/kg/day

Intake from Fish Consumption = $\frac{1.19\ E\text{-}9 \times 5500 \times 14\ E\text{-}4}{70}$

= 1.31×10^{-10} mg/kg/day

Sum of Intakes = 1.85×10^{-10} mg/kg/day

APPENDIX C

Public Health Assessment Exposure Profiles

Storm Sewer Infiltration

Monochlorobenzene

Intake from Drinking Water Ingestion = $\frac{3.92\ E\text{-}8 \times 2}{70}$

= 1.12×10^{-9} mg/kg/day.

Intake from Dermal Absorption While Swimming = $\frac{3.92\ E\text{-}6 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$

= 1.45×10^{-10} mg/kg/day

Intake from Accidental Ingestion While Swimming = $\frac{3.92\ E\text{-}6 \times 0.05 \times 2.6 \times 0.0685}{70}$

= 4.99×10^{-10} mg/kg/day

Total Intake due to Swimming = 6.43×10^{-10} mg/kg/day

Intake from Fish Consumption = $\frac{3.92\ E\text{-}8 \times 645 \times 14\ E\text{-}4}{70}$

= 5.06×10^{-10} mg/kg/day

Sum of Intakes = 2.27×10^{-9} mg/kg/day

1,2,4-Trichlorobenzene

Intake from Drinking Water Ingestion

$$= \frac{3.33\ E\text{-}8 \times 2}{70}$$

$$= 9.51 \times 10^{-10}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{3.33\ E\text{-}6 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 1.23 \times 10^{-10}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{3.33\ E\text{-}6 \times 0.05 \times 2.6 \times 0.0685}{70}$$

$$= 4.24 \times 10^{-10}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 5.47 \times 10^{-10}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{3.33\ E\text{-}8 \times 3200 \times 14\ E\text{-}4}{70}$$

$$= 2.13 \times 10^{-9}\ mg/kg/day$$

Sum of Intakes

$$= 3.63 \times 10^{-9}\ mg/kg/day$$

alpha-Hexachlorocyclohexane

Intake from Drinking Water Ingestion

$$= \frac{8.92\text{ E-9} \times 2}{70}$$

$$= 2.55 \times 10^{-10}\ \text{mg/kg/day}$$

Intake from Dermal Absorption While Swimming

$$= \frac{8.92\text{ E-7} \times 8.0\text{ E-7} \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 3.30 \times 10^{-11}\ \text{mg/kg/day}$$

Intake from Accidental Ingestion While Swimming

$$= \frac{8.92\text{ E-7} \times 0.05 \times 2.6 \times 0.0685}{70}$$

$$= 1.14 \times 10^{-10}\ \text{mg/kg/day}$$

Total Intake Due to Swimming

$$= 1.47 \times 10^{-10}\ \text{mg/kg/day}$$

Intake from Fish Consumption

$$= \frac{8.92\text{ E-9} \times 605 \times 14\text{ E-4}}{70}$$

$$= 1.08 \times 10^{-10}\ \text{mg/kg/day}$$

Sum of Intakes

$$= 5.10 \times 10^{-10}\ \text{mg/kg/day}$$

gamma-Hexachlorocyclohexane

Intake from Drinking Water Ingestion

$$= \frac{3.92\ E\text{-}9 \times 2}{70}$$

$$= 1.12 \times 10^{-10}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{3.92\ E\text{-}7 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 1.45 \times 10^{-11}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{3.92\ E\text{-}7 \times 0.05 \times 2.6 \times 0.0685}{70}$$

$$= 4.99 \times 10^{-11}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 6.44 \times 10^{-11}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{3.92\ E\text{-}9 \times 605 \times 14\ E\text{-}4}{70}$$

$$= 4.74 \times 10^{-11}\ mg/kg/day$$

Sum of Intakes

$$= 2.24 \times 10^{-10}\ mg/kg/day$$

PCBs

Intake from Drinking Water Ingestion = $\frac{8.92\ E\text{-}11 \times 2}{70}$

= 2.55×10^{-12} mg/kg/day

Intake from Dermal Absorption While Swimming = $\frac{8.92\ E\text{-}9 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$

= 3.30×10^{-13} mg/kg/day

Intake from Accidental Ingestion While Swimming = $\frac{8.92\ E\text{-}9 \times 0.05 \times 2.6 \times 0.0685}{70}$

= 1.13×10^{-12} mg/kg/day

Total Intake Due to Swimming = 1.46×10^{-12} mg/kg/day

Intake from Fish Consumption = $\frac{8.92\ E\text{-}11 \times 100{,}000 \times 14\ E\text{-}4}{70}$

= 1.78×10^{-10} mg/kg/day

Sum of Intakes = 1.82×10^{-10} mg/kg/day

2,3,7,8-Tetrachlorodibenzo-p-dioxin

Intake from Drinking Water Ingestion

$$= \frac{4.04\ E\text{-}14 \times 2}{70}$$

$$= 1.15 \times 10^{-15}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{4.04\ E\text{-}12 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 1.49 \times 10^{-16}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{4.04\ E\text{-}12 \times 0.05 \times 2.6 \times 0.0685}{70}$$

$$= 5.14 \times 10^{-16}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 6.63 \times 10^{-16}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{4.04\ E\text{-}14 \times 128{,}000 \times 14\ E\text{-}4}{70}$$

$$= 1.03 \times 10^{-13}\ mg/kg/day$$

Sum of Intakes

$$= 1.05 \times 10^{-13}\ mg/kg/day$$

Mercury

Intake from Drinking Water Ingestion

$$= \frac{4.87\ E\text{-}11 \times 2}{70}$$

$$= 1.39 \times 10^{-12}\ mg/kg/day$$

Intake from Dermal Absorption While Swimming

$$= \frac{4.87\ E\text{-}9 \times 8.0\ E\text{-}7 \times 2.6 \times 0.0685 \times 18{,}150}{70}$$

$$= 1.80 \times 10^{-13}\ mg/kg/day$$

Intake from Accidental Ingestion While Swimming

$$= \frac{4.87\ E\text{-}9 \times 0.05 \times 2.6 \times 6.5\ E\text{-}4}{70}$$

$$= 6.20 \times 10^{-13}\ mg/kg/day$$

Total Intake Due to Swimming

$$= 8.00 \times 10^{-13}\ mg/kg/day$$

Intake from Fish Consumption

$$= \frac{4.87\ E\text{-}11 \times 5500 \times 14\ E\text{-}4}{70}$$

$$= 5.36 \times 10^{-12}\ mg/kg/day$$

Sum of Intakes

$$= 7.55 \times 10^{-12}\ mg/kg/day$$

APPENDIX D

Public Health Assessment Exposure Profiles

Off-Site Soils

Hexachlorobenzene

Hexachlorobenzene is not considered to be a skin hazard. The potential routes of entry are soil ingestion and dust inhalation. These exposures are calculated below.

Daily Intake for Soil Ingestion	=	(0.338 x 1.366 E-8) + (0.338 x 1.557 E-8)
	=	9.88×10^{-9} mg/kg/day.
Daily Intake for Particulate Inhalation	=	0.338 x 2.202 E-10
	=	7.44×10^{-11} mg/kg/day
Total Chronic Daily Intake Intake of Hexachlorobenzene derived from off-site soil concentrations.	=	9.95×10^{-9} mg/kg/day

alpha-Hexachlorocyclohexane

As in the case of 1,2,4-trichlorobenzene, alpha-HCCH is considered to be a skin hazard. Exposure to alpha-HCCH through the skin absorption, soil ingestion, and particulate inhalation routes is calculated below.

Intake from Skin Absorption	=	0.229 x 9.182 E-8
	=	2.10×10^{-8} mg/kg/day.
Intake from Soil Ingestion	=	(0.229 x 1.366 E-8) + (0.229 x 1.557 E-8)
	=	6.69×10^{-9} mg/kg/day.
Intake from Particulate Inhalation	=	0.229 x 2.202 E-10
	=	5.04×10^{-11} mg/kg/day.

Total Chronic Daily Intake of alpha-HCCH Derived from Off-site Soil Concentrations = 2.77×10^{-8} mg/kg/day.

gamma-Hexachlorocyclohexane

As in the case of alpha-HCCH, gamma-HCCH has the potential for skin absorption. Exposure to gamma-HCCH through the skin absorption, soil ingestion, and particulate inhalation routes is calculated below.

Intake from Skin Absorption = 0.109 x 9.182E-8

= 1.00×10^{-8} mg/kg/day

Intake from Soil Ingestion = (0.109 x 1.366E-8) + (0.109 x 1.557E-8)

= 3.19×10^{-9} mg/kg/day

Intake from Particulate Inhalation = 0.109 x 2.202E-10

= 2.40×10^{-11} mg/kg/day

Total Chronic Daily Intake of gamma-HCCH Derived from Off-Site Soil Concentrations = 1.32×10^{-8} mg/kg/day

2,3,7,8-Tetrachlorodibenzo-p-dioxin

2,3,7,8-TCDD is also a skin hazard. The daily intakes from skin absorption, soil ingestion and particulate inhalation are calculated below.

Intake from Skin Absorption = 8 E-4 x 9.182 E-8

= 7.34×10^{-11} mg/kg/day

Intake from Soil Ingestion	=	(8 E-4 x 1.366 E-8) + (8 E-4 x 1.557 E-8)
	=	2.34×10^{-11} mg/kg/day
Intake from Particulate Inhalation	=	8 E-4 x 2.202 E-10
	=	1.76×10^{-13} mg/kg/day
Total Chronic Daily Intake of 2,3,7,8-TCDD Derived from Off-site Soil Concentrations.	=	9.70×10^{-11} mg/kg/day

Mercury

As in the cases of alpha-HCCH and 2,3,7,8-TCDD, mercury has a potential for significant skin absorption. Exposure to mercury through the skin absorption, soil ingestion and particulate inhalation routes is calculated below. These calculations presently use concentrations determined for total mercury.

Intake from Skin Absorption	=	1.53 x 9.182 E-8
	=	1.40×10^{-7} mg/kg/day
Intake from Soil Ingestion	=	(1.53 x 1.366 E-8) + (1.53 x 1.557 E-8)
	=	4.47×10^{-8} mg/kg/day
Intake from Particulate Inhalation	=	1.53 x 2.202 E-10
	=	3.37×10^{-10} mg/kg/day
Total Chronic Daily Intake of Mercury Derived from Off-site Soil Concentrations.	=	1.85×10^{-7} mg/kg/day