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DOE/OR/21950-003

BASELINE RISK ASSESSMENT FOR THE TONAWANDA SITE

TONAWANDA, NEW YORK

AUGUST 1993

U.S. Department of Energy
Oak Ridge Operations Office
Formerly Utilized Sites Remedial Action Program

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prepared by
U.S. Department of Energy, Oak Ridge Operations Office, Formerly Utilized Sites Remedial Action Program

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LIST OF ACRONYMS

ACGIH	American Conference of Governmental Industrial Hygienists
AEC	Atomic Energy Commission
ALARA	as low as reasonable achievable
ARARs	applicable or relevant and appropriate requirements
ATVs	all terrain vehicles
BEIR	biological effects of ionizing radiation
BCF	bioconcentration factor
BCI	Beak Consultants, Inc.
BNAE	base/neutral, and acid extractable
BNI	Bechtel National, Incorporated
BRA	baseline risk assessment
CDI	chronic daily intake
CEDE	committed effective dose equivalent
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended
CF	conversion factor
COC	contaminants of concern
DCF	dose conversion factor
DOE	U.S. Department of Energy
ED	exposure duration
EF	exposure frequency
EFH	Exposure Factors Handbook
EPA	U.S. Environmental Protection Agency
ERA	ecological risk assessment
ET	exposure time
ETF	environmental transport factor
EQ	ecological quotient
FS	Feasibility Study
FUSRAP	Formerly Utilized Sites Remedial Action Program
FWS	U.S. Fish and Wildlife Service
HEAST	Health Effects Assessment Summary Tables
HI	hazard index
HQ	hazard quotient
ICRP	International Commission on Radiation Protection
IG	ingestion rate
IRIS	Integrated Risk Information System
LET	linear energy transfer
MED	Manhattan Engineer District
NAS	National Academy of Sciences
NCRP	National Council on Radiation Protection

LIST OF ACRONYMS (continued)

NEPA	National Environmental Policy Act
NESHAPs	National Emission Standards for Hazardous Air Pollutants
NOAEL	no observed adverse effect levels
NPL	National Priorities List
NYSDEC	New York State Department of Environmental Conservation
ORAU	Oak Ridge Associated Universities
ORNL	Oak Ridge National Laboratory
OSWER	Office of Solid Waste and Emergency Response
PAHs	polycyclic aromatic hydrocarbons
PCBs	polychlorinated biphenyls
P-S	Performance Standards Use
QA/QC	quality assurance/quality control
RAGS	Risk Assessment Guidance of Superfund
RCRA	Resource Conservation and Recovery Act
RESRAD	RESidual RADioactivity computer code
RfC	reference concentration
RfD	reference dose
RI	Remedial Investigation
RI/FS	Remedial Investigation/Feasibility Study
RI/FS-EIS	Remedial Investigation/Feasibility Study-Environmental Impact Statement
RME	reasonable maximum exposure
ROD	Record of Decision
SAIC	Science Applications International Corporation
SARA	Superfund Amendments and Reauthorization Act
SHRTSC-ECAO	Superfund Health Risk Technical Support Center-Environmental Criteria and Assessment Office
SF	slope factor
SVOC	semivolatile organic compound
TLV-STEL	toxic limit value - short-term exposure limit
TLV-TWA	toxic limit value - time-weighted average
TMA/E	Thermo Analytical/Eberline
UCL	upper confidence limit
UL_{95}	95 percent upper confidence limit
UNSCEAR	United Nations Scientific Committee on the Effects of Atomic Radiation
VOCs	volatile organic compounds
WL	working level
WLM	working-level month
WQCAW	Water Quality Criteria, Aquatic
XQ	exposure quotient

Also included is the square area outside the eastern property boundary, the non-functional fuel transfer station, the drainage ditch between Ashland 1 and Seaway properties, and the area where the ditch enters the underground conduit at Seaway.

Subarea B includes all of the raised berms built when oil storage tanks were located on the property. Included are the areas within the berms where surface drainage and berm erosion accumulate.

Chemical Sources

Chemical soil data were limited and, therefore, were combined for characterization of Ashland 1 chemical contamination.

1.2.1.3 Ashland 2

Radiological Sources

Ashland 2 radionuclide concentrations were evaluated using the comparison of contaminants to background and guideline concentrations as completed for the Linde and Ashland 1 properties. This evaluation, as well as consideration of site surface features and drainage patterns, were used in subarea delineation at Ashland 2. Figure 1-7 shows the subareas at Ashland 2 which are described below.

Subarea A includes the drainage path on the Niagara Mohawk property southwest of Ashland 2 where drainage from the Seaway property occurs. This subarea includes the western portion of the onsite wetlands, drainage from the underground conduit from Seaway, property surrounding the elevated fill, and the elevated fill area. Subarea B is the area designated as Ashland 2 South in the RI (BNI 1992).

Chemical Sources

Soil chemical data were combined for the entire Ashland 2 property.

1.2.1.4 Seaway

Radiological Sources

The area at Seaway where uranium processing residues were disposed is a small portion (approximately 13 acres) of the Seaway property. The disposal area at Seaway was divided into four subareas in the RI (BNI 1992), A through D. Note that the subareas of Seaway delineated for the BRA are not the same as those used to delineate contaminated areas at Seaway in the RI. Area D soil data were included with Ashland 1 radionuclide data because the contamination extended from the Ashland 1 property onto the Seaway property. Radionuclide concentrations

in the Seaway areas were reevaluated for purposes of the risk assessment as they were for Linde, Ashland 1, and Ashland 2. Figure 1-8 shows the Seaway subareas which are described below.

Subarea A includes the outer boundary area of Seaway contamination where radionuclide concentrations are near the surface. This area corresponds with the area designated as area A in the RI (BNI 1992).

Subarea B is the remaining area covered by 40 feet of fill according to the RI (BNI 1992). Due to the depth of cover material, no complete exposure pathways exist for contamination in Subarea B.

As described in Section 2.3, surface water and sediment data from Ashland 1, Ashland 2, and Seaway were combined and aggregated as single units (e.g., Tonawanda surface water and Tonawanda sediment).

1.2.2 General Site Description - History

Linde

Linde is an approximately 55-ha (135-acre) operating industrial plant presently owned by Union Carbide Industrial Gases (Figure 1-3). Portions of this site were previously owned by the Town of Tonawanda, Excelsior Steel Ball Company, Metropolitan Commercial Corporation, and the Pullman Trolley Land Company. Buildings on the site are currently being used as offices, research laboratories, fabrication facilities, and warehouse storage areas. The entire site is fenced, with the entrance to the plant continuously guarded by security personnel. The Linde property is bounded on the north and south by other industry and small businesses, on the east by the Consolidated Rail Corporation (Conrail) railroad tracks, and on the west by a public park that was part of the former Sheridan Park Golf Course, now owned by Linde.

The Linde Air Products Corporation (Linde), formerly a subsidiary of Union Carbide Corporation, contracted with MED to separate uranium from uranium ore from 1942 to 1946. Wastes generated from the separation process conducted at the Linde property were disposed at Ashland 1. In later activities at Ashland 1, part of the contaminated material was transported to Seaway and Ashland 2. Five Linde buildings were involved in MED activities: Building 14, which was built by Union Carbide in the mid-1930s, and Buildings 30, 31, 37, and 38 which were built by MED on land owned by Union Carbide. Ownership of Buildings 30, 31, 37, and 38 was transferred to Linde when the MED contract was terminated. Building 90 was constructed after uranium processing operations ceased. The following are historical descriptions of the various buildings:

- Building 14: used for laboratory and pilot plant studies for uranium separation in early part of MED operations; currently being used for offices, research laboratories, and fabrication facilities.

- Building 30: used as primary process building for uranium processing during MED operations; some processing of metallic nickel with nitric acid to produce nickel salt; currently being used as a shipping and receiving warehouse.

- Building 31: used in uranium separation process, primarily fluorination, during MED operations; currently being used for maintenance and offices.

- Building 37: used in uranium separation process during MED operations; demolished in 1981.

- Building 38: used in uranium separation process, primarily fluorination, during MED operations; currently part of the building is being used as a storage area.

- Building 90: built in an area where tailings accumulated during MED operations; tailings were removed from the site when operations ceased in 1948; before construction, soil contaminated with low-level radioactivity was removed from the construction area (currently stored in a pile west of the building).

A three-phase process was used to separate uranium from the uranium ores and tailings. Phase 1, conducted in Building 30, consisted of separating U_3O_8 (uranium oxide) from the feedstock materials by a series of process steps consisting of acid digestion, precipitation, and filtration.

The primary waste resulting from Phase 1 was a solid, gelatinous filter cake consisting of impurities remaining after filtration of the uranium carbonate solutions. Approximately 7,250 metric tonnes (8,000 tons) of filter cake from the Phase 1 processing of domestic tailings were disposed at Ashland 1. These residues contained approximately 0.54 percent uranium, which corresponds to 26.5 Curies (Ci) of natural uranium. Residue from the African ore was shipped to the former Lake Ontario Ordnance Works in Lewiston, New York, where it was isolated and stored in a secure area. Evaluation of waste material at this site is not included in this BRA.

Liquid effluent from filtration of the sodium diuranate cake was initially discharged to the sanitary sewer system; by April 1944, approximately 100 x 10^6 L (26 x 10^6 gal), had been discharged. In June 1944, the process was slightly modified, resulting in a liquid with pH exceeding the then applicable limits for disposal in the sanitary sewer. It was determined that injection wells would be used for disposal of the liquid wastes whenever possible. Seven onsite injection wells were then used to dispose of liquid waste: one group of three wells east of Building 14 and another group of four near Buildings 30 and 38 (Figure 1-3). The disposal wells ranged from 28 to 46 m (90 to 150 ft) deep; some were drilled 9 to 12 m (30 to 40 ft) into bedrock (Aerospace 1981). When the injection wells became blocked, the effluent was discharged into a storm sewer that drained into a ditch north and outside of the fenced areas of the plant. This ditch eventually drained into Twomile Creek. The injection wells were later backfilled with debris. Extensive portions of the original sanitary and storm sewer systems, including the ditch leading to Twomile Creek, have also been abandoned, removed, or filled in.

Furthermore, the sewage systems have undergone periodic cleaning since 1946 (ORAU 1981). It is estimated that slightly more than 210 x 10^6 L (55 x 10^6 gal) of effluent were discharged into the seven disposal wells and 212 x 10^6 L (56 x 10^6 gal) were discharged into Twomile Creek via the storm sewer (BNI 1992). Historical records indicate that Ra-226 and uranium were the principal radioactive materials in the liquid effluent (Aerospace 1981).

The average concentration of uranium oxide in the liquid effluent was 0.026 g/L (Aerospace 1981). This concentration would indicate that 9,600 kg (21,000 lb) of uranium (6.5 Ci) were released to the sanitary sewer, and that 5,600 kg (12,320 lb) of uranium (3.8 Ci) were released into the storm sewer leading to Twomile Creek; and 5,400 kg (11,900 lb) of uranium (3.7 Ci) were injected into the onsite wells. Based on the total amount of effluent estimated to have been released, the amount of Ra-226 released into the sanitary sewer was approximately 2.6 Ci. The amount released into the storm drain was approximately 5.5 Ci, and the amount injected into the wells was approximately 5.5 Ci. These are conservative estimates based on the total amount of liquid effluent discharged from both the domestic ores (low in radium) and the African ores (high in radium). Tests performed by the University of Rochester indicate that the total amount of Ra-226 disposed with the liquid effluent could be as low as approximately 0.6 Ci (Aerospace 1981).

Renovation of the facility over the years prompted some consolidation of contaminated materials. Before Building 90 was constructed, soil contaminated during MED operations was removed from the construction area and placed in two windrows along the north and east fences and in one pile on the north portion of the property. All three piles of contaminated soil were subsequently consolidated into one uncovered pile west of Building 90. Recent construction activity occurring in the northwest area of the site has also disturbed soils at the site. However, all data were collected before the recent construction; therefore, this BRA does not include consideration of the recent construction activities at the site.

Ashland 1

Ashland 1 is located in an industrialized area, and has been owned and used as part of an oil refinery by Ashland Oil Company since 1960. The 4.4-ha (10.8-acre) site is bounded on the east by a strip of land owned by Penn Central Transportation Company, and land along the northern and western boundaries is owned by Ashland Oil Company (Figure 1-4). The land at the southern end of the property is owned by Niagara Mohawk Power Corporation. A non-operating Iroquois Gas Company receiving and metering station is located in the southeast corner of the site. Ashland 1 is southwest of Ashland 2 and Seaway, and the property is divided into three sections by berms.

Currently, land adjacent to Ashland 1 is used for dismantling the vacant Ashland Oil Refinery. There is a 25-cm (10-in.) water line that runs from the refinery along the western side of the property and a 0.3-m (12-in.) gas line in an easement that runs along the eastern border. At one time, two large petroleum product storage tanks and process piping were located within a bermed area on the site. Ashland Oil disassembled and removed the tanks in 1989. The

bermed area was equipped with a sump pump system to pump runoff into an open ditch and ultimately to a culvert beneath Seaway that empties into Rattlesnake Creek and then into Twomile Creek. Presently, surface runoff from Ashland 1 is controlled by the berms and several small pipes lead from the inside of the bermed area and drain surface runoff to the open ditch. The flow from this ditch is directed into the concrete culvert that passes beneath Seaway.

From 1944 to 1946, Ashland 1 served as a disposal site for approximately 7,250 metric tonnes (8,000 tons) of ore refinery residues generated at Linde. Following a radiological survey in 1958 by the Environmental Measurements Laboratory, AEC released the Ashland 1 property for use without removal of the residues. In 1960, the land was transferred to Ashland Oil Company. In 1974, Ashland Oil constructed two bermed petroleum product storage tanks and a drainage ditch on the Ashland 1 property. Most of the soil that was removed in construction of the storage tanks and the drainage ditch was disposed on the Seaway and Ashland 2 properties. The soil that was not transported offsite was used to construct the berms around the storage tanks which were removed in 1989.

As observed in an October 1991 visit, a portion of Ashland 1 is presently utilized as a drum storage area for unknown materials belonging to Ashland Oil Company. No data are available concerning this drum storage area.

Ashland 2

Ashland 2 is a roughly rectangular tract of land, approximately 47 ha (115 acres), located northeast of Ashland 1 and Seaway (Figure 1-4). It is owned by Ashland Oil and is bounded by commercial property owned by Niagara Mohawk Power Corporation, Benson Development Company, and G. K. Hambleton. The property is vacant and overgrown with weeds, bushes, and grass; currently, no commercial operations are being conducted. Land uses near the property are the same as those of Ashland 1. Utilities in the area include high-voltage transmission lines which run parallel to the property to the southwest.

A portion of Ashland 2 is contaminated with residues relocated from processing operations conducted at the Linde facility. The residues were initially disposed at Ashland 1 and were later moved to Ashland 2, where they were placed in a fill area near the Ashland Oil Company's industrial landfill that was formerly active on the property.

Drainage from Ashland 1 is carried to Ashland 2 via a reinforced concrete pipe that traverses the Seaway property. A series of open drainage channels and Rattlesnake Creek convey surface water runoff from Ashland 2, with eventual discharge to Twomile Creek. These drainage channels begin to the southeast of the property, near Seaway, and drain north. Rattlesnake Creek carries the outflow from the conduit that drains Ashland 1 and the surrounding area.

From 1957 to 1982, Ashland Oil used a northern portion of Ashland 2 as a landfill for disposal of general plant refuse and chemical and industrial by-products (BNI 1992). The

industrial wastes were estimated to be composed of five tons per year of phosphoric acid polymerization catalyst sludge, 72 tons per year of lime slurry sludge, and 50 tons per year of spent clay. Ashland Oil closed the landfill in 1982 and covered it with clayey soil (Engineering Science 1986). From 1974 to 1982, Ashland Oil transported an unknown quantity of the radioactive residues from Ashland 1 to an area adjoining the Ashland 2 landfill area.

Seaway

The Seaway Industrial Park (Figure 1-4) is an operating sanitary landfill approximately 38 ha (93 acres) in size, which Seaway Industrial Park Development Company has owned since 1964. Seaway Industrial Park has been used as a landfill for 50 to 60 years. It is presently operated by Browning-Ferris Industries. Seaway Industrial Park Development Company was formerly North Waterway Company, which owned the site before 1964. Ownership before North Waterway Company is unknown (FBDU 1981b). Solid waste landfilled at Seaway primarily derives from municipal, commercial, industrial, and construction sources. Two buildings on the site serve as check-in and weigh-in stations for trucks entering the property and are located on the northwest corner. There are no permanent buildings on this site.

The Seaway property is bounded by Ashland Oil to the south; River Road to the northwest; Murphy Trucking, Inc. to the north; Leffler Auto Parts to the east; and property owned by Niagara Mohawk Power Corporation to the northeast. The Seaway site is accessible from River Road on the northwest side. However, access is controlled by a guard gate, and the site is bordered with an approximately six-foot wire fence. A 1-m (3-ft) diameter, reinforced concrete drainage conduit transects the property and passes beneath the landfill. This pipe carries stormwater from Ashland 1 to Ashland 2. The condition of the pipe is unknown.

In 1974, approximately 4,588 m^3 (6,000 yd^3) of residue, composed of low-grade uranium ore tailings, were excavated by Ashland Oil from Ashland 1 and placed in Areas A, B, and C (Figure 1-2) of the landfill. The total volume of contamination in or near Areas A, B, and C is estimated at 37,800 m^3 (49,000 yd^3) (FBDU 1981b). Area A is approximately 4 ha (10 acres), and Areas B and C combined are approximately 1.4 ha (3.5 acres).

Since 1974, portions of the residues have been buried under refuse and fill material. A fourth area, Area D, is 46 m by 46 m (150 ft by 150 ft) and is located along the boundary with Ashland 1 on the southeastern border of the Seaway property. This fourth area may have been formed when residues were accidentally spread across the Ashland 1 property line during the soil moving operations at Ashland 1 (BNI 1992).

1.2.3 Summary of Site Contamination

Numerous investigations have been performed on the Tonawanda site. Information on the most recent sampling and analyses is presented in the RI Report (BNI 1992). Surface soils, subsurface soils, groundwater, surface water, and sediments have all been sampled and analyzed

for radiological and chemical contamination. Structures also have been sampled and analyzed for radionuclides.

DOE is the lead agency for remedial actions at the Tonawanda site. Under FUSRAP, DOE assumes responsibility for:

- managing radioactive contamination that is related to MED processing at the Linde plant, including contamination that has spread to Ashland 1 and 2, Seaway, or other properties; and

- managing any chemical contamination at the Tonawanda site that is mixed with radioactive contamination or that resulted from activities conducted for MED.

During the RI phase of the CERCLA-NEPA process, multi-media samples were collected for radiological and chemical analyses from a number of locations throughout the Tonawanda site and its vicinity, from areas later determined contaminated by MED-related wastes, and also from areas not impacted by MED-related sources. Data used in the BRA include results of sitewide sampling and, therefore, the assessment includes, in part, risks in site areas not impacted by MED-related sources. DOE has no authority for identification or cleanup in areas not impacted by MED-related sources. The presentation of risks in this report should not be interpreted as indicating DOE responsibility for remediation of site areas not impacted by MED-related sources.

1.3 SCOPE OF THE BRA

This BRA includes the determination of most likely (i.e., average) and reasonable maximum individual human risks potentially resulting from exposure to contaminants at each property unit, as well as an estimate of human population risk and an ecological risk assessment (ERA). The approach used for the Tonawanda BRA is based on EPA Risk Assessment Guidance for Superfund [RAGS (EPA 1989a, 1989b)], and ERA guidance (EPA 1989c, 1991b).

The scope of the study is to use site-specific data to isolate localized areas of elevated contaminant concentrations and evaluate risk. The data in the RI report and those found in previous reports were used to identify and screen potential COCs for risk evaluation. The evaluation focused primarily on the comparison of site concentrations to background and evaluation of sample quantitation limits, as well as consideration of detection frequency. Exposure point concentrations were estimated for the COCs, and were used to estimate potential exposure and lifetime risk to humans of developing cancer for various combinations of locations, receptors, and exposure scenarios. These are compared with DOE and EPA guidelines for exposure and risk. The determination of final cleanup criteria is beyond the scope of the BRA and will be addressed in the FS-EIS, Proposed Plan, and ROD for the site.

1.3.1 Time Period

Because DOE is responsible for the cleanup of this site and is committed to pursuing a timely response, the time period considered as the hypothetical future in this assessment of risks for the no-action alternative is the immediate future (i.e., the next 150 years). Thus, further dispersal of contaminants that would occur over very long time periods has not been considered in the BRA. Current contaminant concentrations in the environmental media identified for this site are assumed for each scenario evaluated, including the future scenarios. The RESidual RADioactivity (RESRAD) computer code used to estimate radiation exposure dose in this analysis can estimate doses at specified future times. However, the ability of this or any currently available model to accurately predict contaminant fate and transport and resultant exposure and risk at distant future times is highly uncertain. The estimated doses for each future scenario were conservatively assumed to be the maximum annual doses up to 1,500 years in the future. For the radiological COCs at least, this assumption yields the maximum estimates of dose and risk.

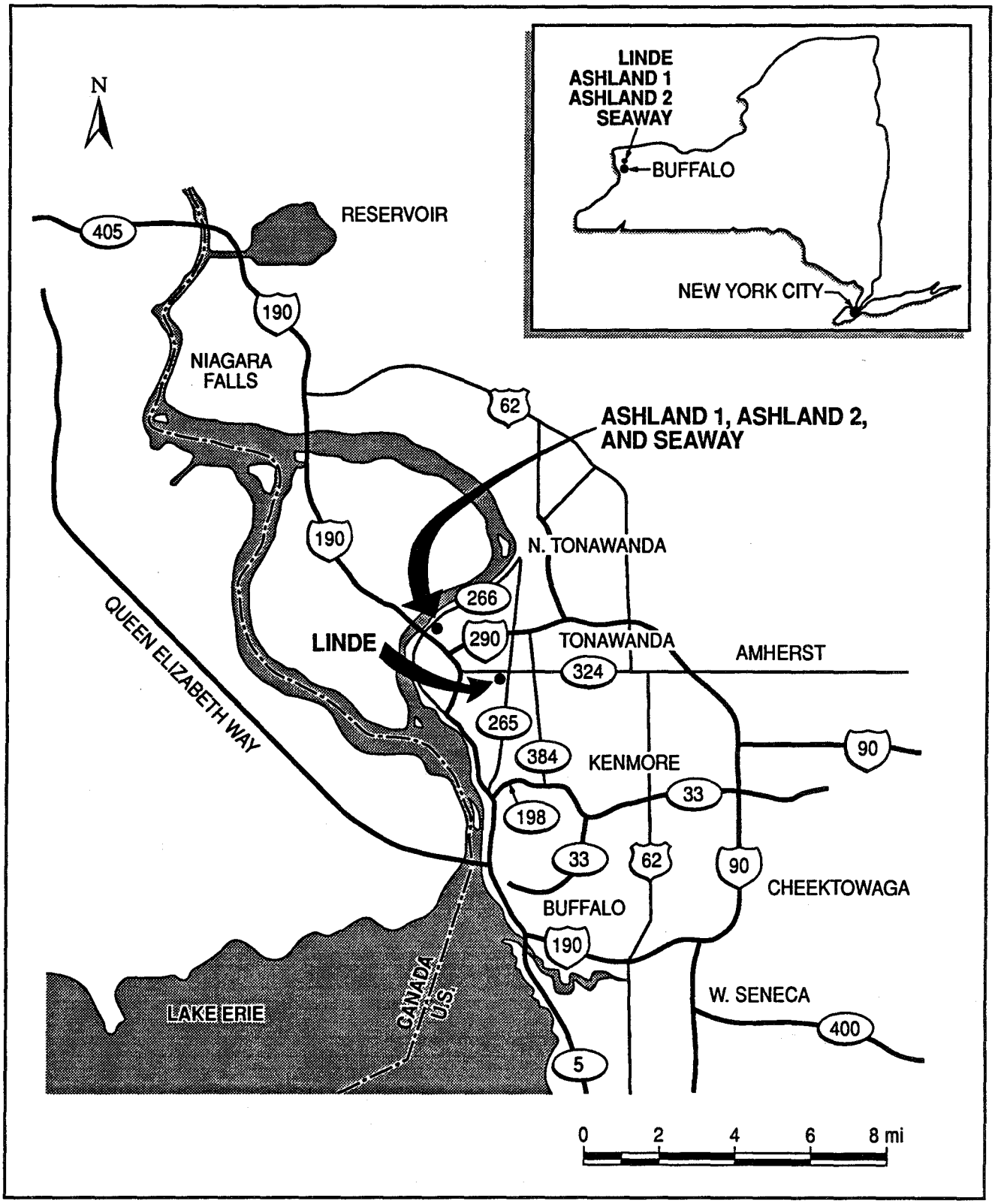
1.3.2 Institutional Control and Exposure Scenarios

Under the CERCLA process, a BRA typically considers impacts that could occur if remedial action were not performed at a site. It assesses impacts under both current conditions, which can include institutional controls, and projected future conditions, assuming no institutional controls. Under the NEPA process, the impact assessment for the no-action alternative typically addresses the status quo at the site, which includes the retention of existing institutional controls (e.g., access restrictions and monitoring) up to the next 100 years. The exposure assessments presented in Section 3 of this document address, in detail, the potential receptors and locations selected to assess baseline impacts for the Tonawanda site.

1.4 REPORT ORGANIZATION

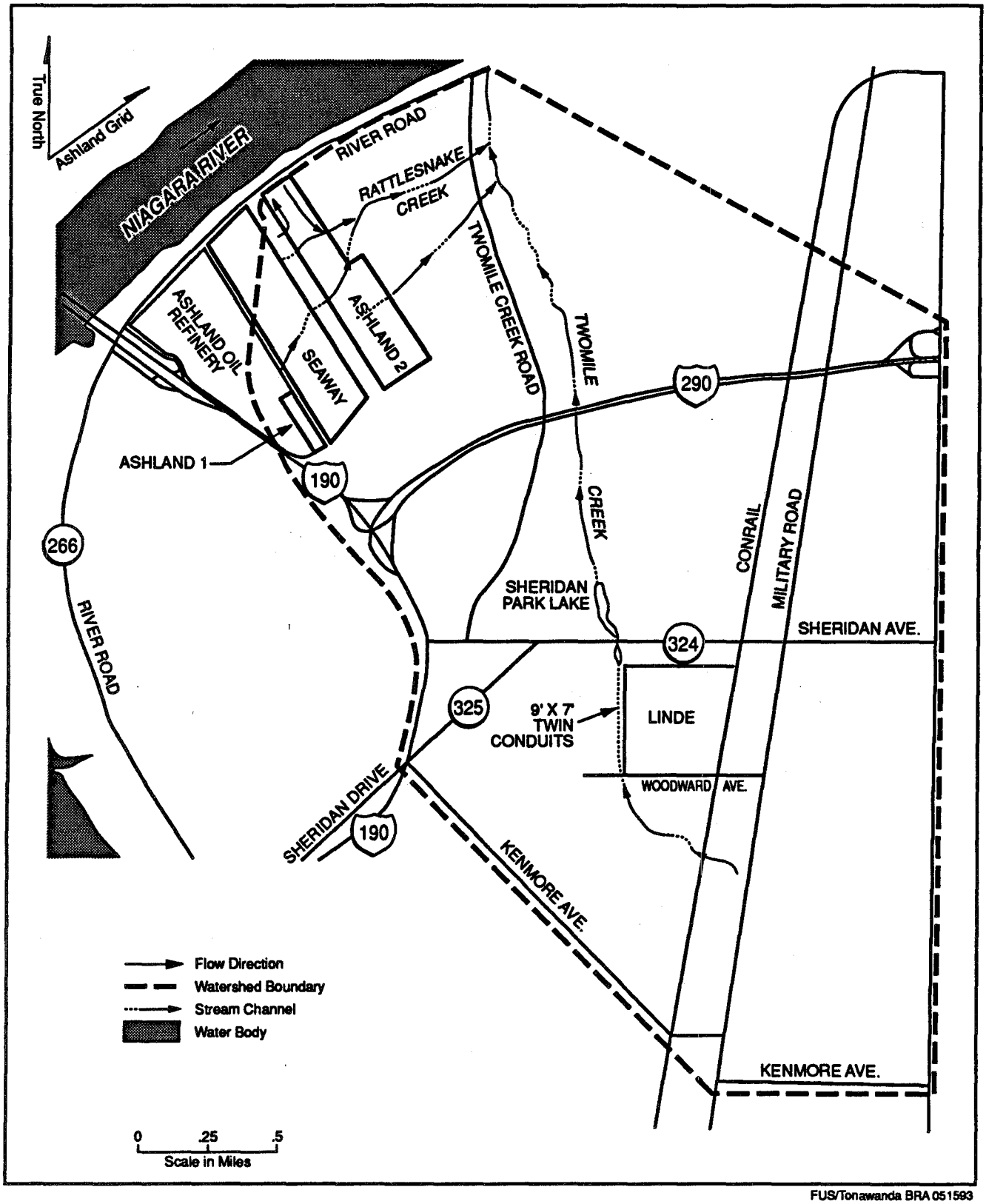
This report is organized according to the suggested EPA RAGS outline (EPA 1989a) with minor modifications to address NEPA considerations and DOE programmatic guidance. Section 2, Identification of Contaminants of Concern, reviews existing radiological and chemical data collected from the site and surrounding area, and identifies the data and COCs used in this risk assessment. Section 3, Exposure Assessment, provides a brief description of those physical features of the Tonawanda site that affect the risk assessment, especially in terms of fate and transport of hazardous substances present at the site. Also, Section 3 summarizes site characteristics pertinent to the exposure assessment, develops exposure point concentrations, identifies potentially exposed populations, and defines primary exposure pathways. Exposure point concentrations are estimated for each selected exposure pathway and subarea. Section 4, Toxicity Assessment, discusses human health effects of each category of COC, and presents quantitative toxicity values for those contaminants. Section 5, Risk Characterization, presents estimates of incremental risk from each selected COC and exposure pathway on a subarea-by-subarea basis to each receptor identified in Section 3. Section 6, Ecological Assessment,

presents a framework for evaluating potential effects on biota from the contamination at the Tonawanda site.



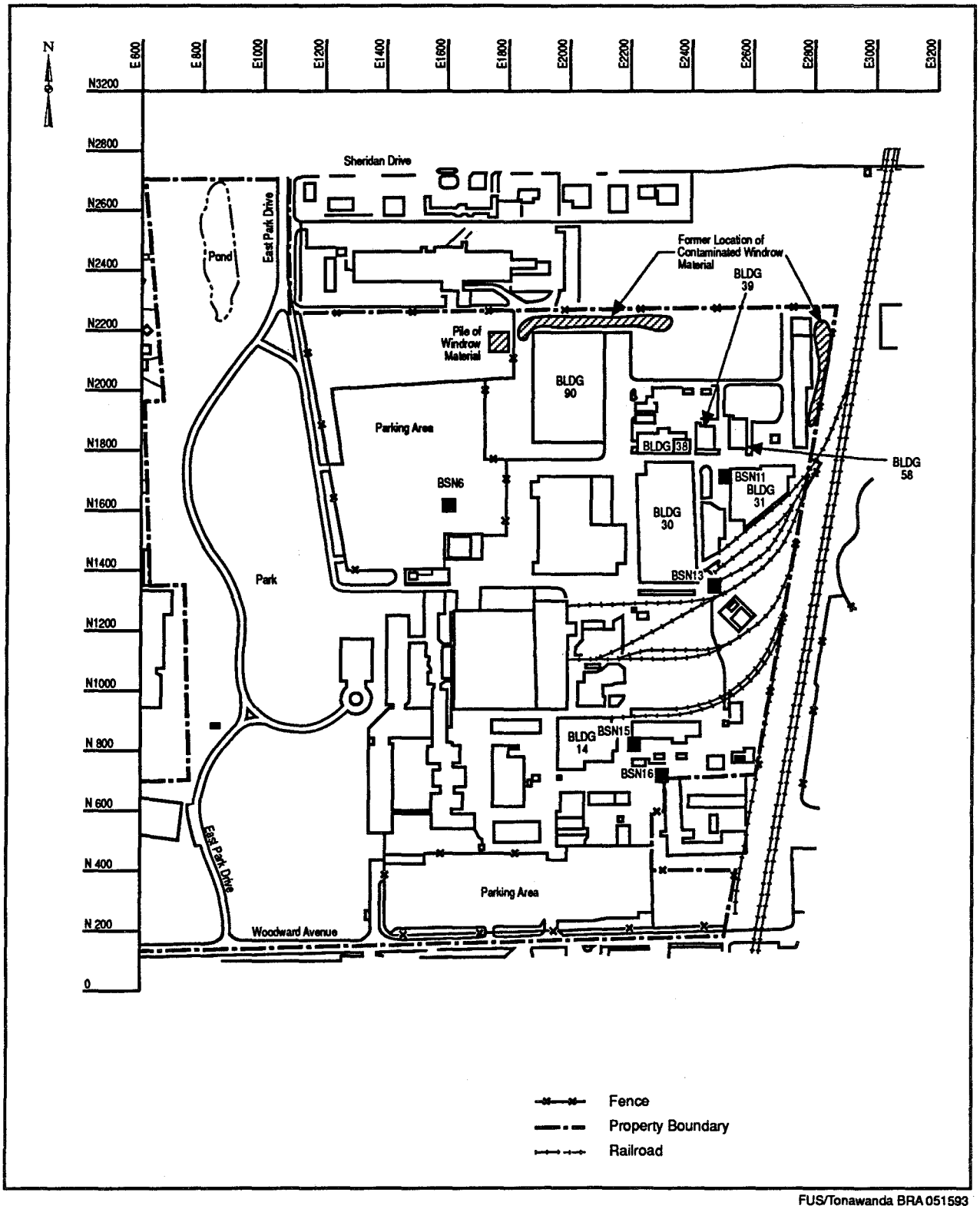
FUS/Tonawanda BRA 051593

Figure 1-1. Regional Setting of the Linde, Ashland 1, Ashland 2, and Seaway Properties, Tonawanda, NY



FUS/Tonawanda BRA 051593

Figure 1-2. Locations of the Linde, Ashland 1, Ashland 2, and Seaway Properties, Tonawanda, NY



FUS/Tonawanda BRA 051593

Figure 1-3. Site Map of Linde Property, Tonawanda, NY

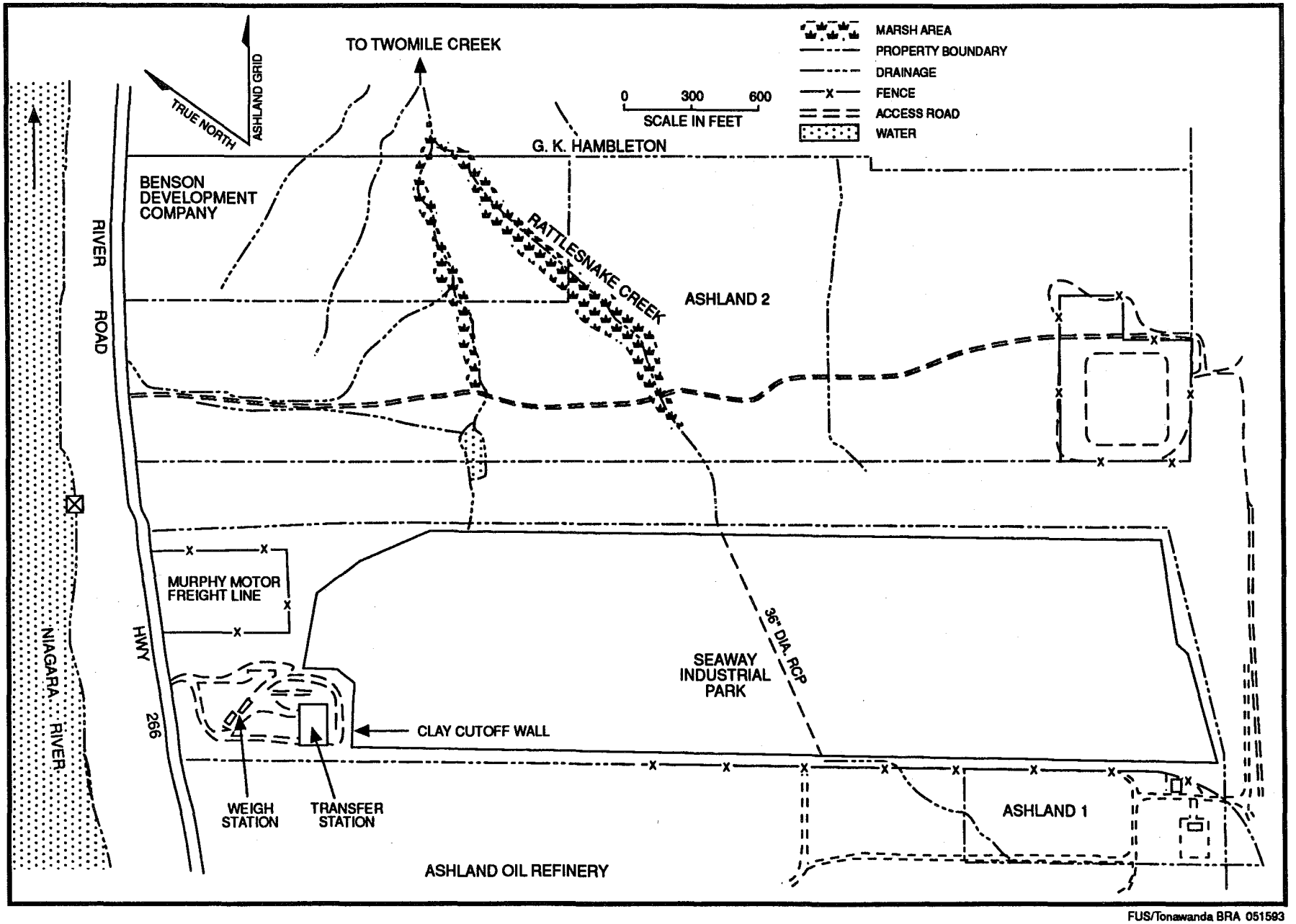


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Figure 1-4. Site Map of Ashland 1, Ashland 2, and Seaway Properties, Tonawanda, NY.

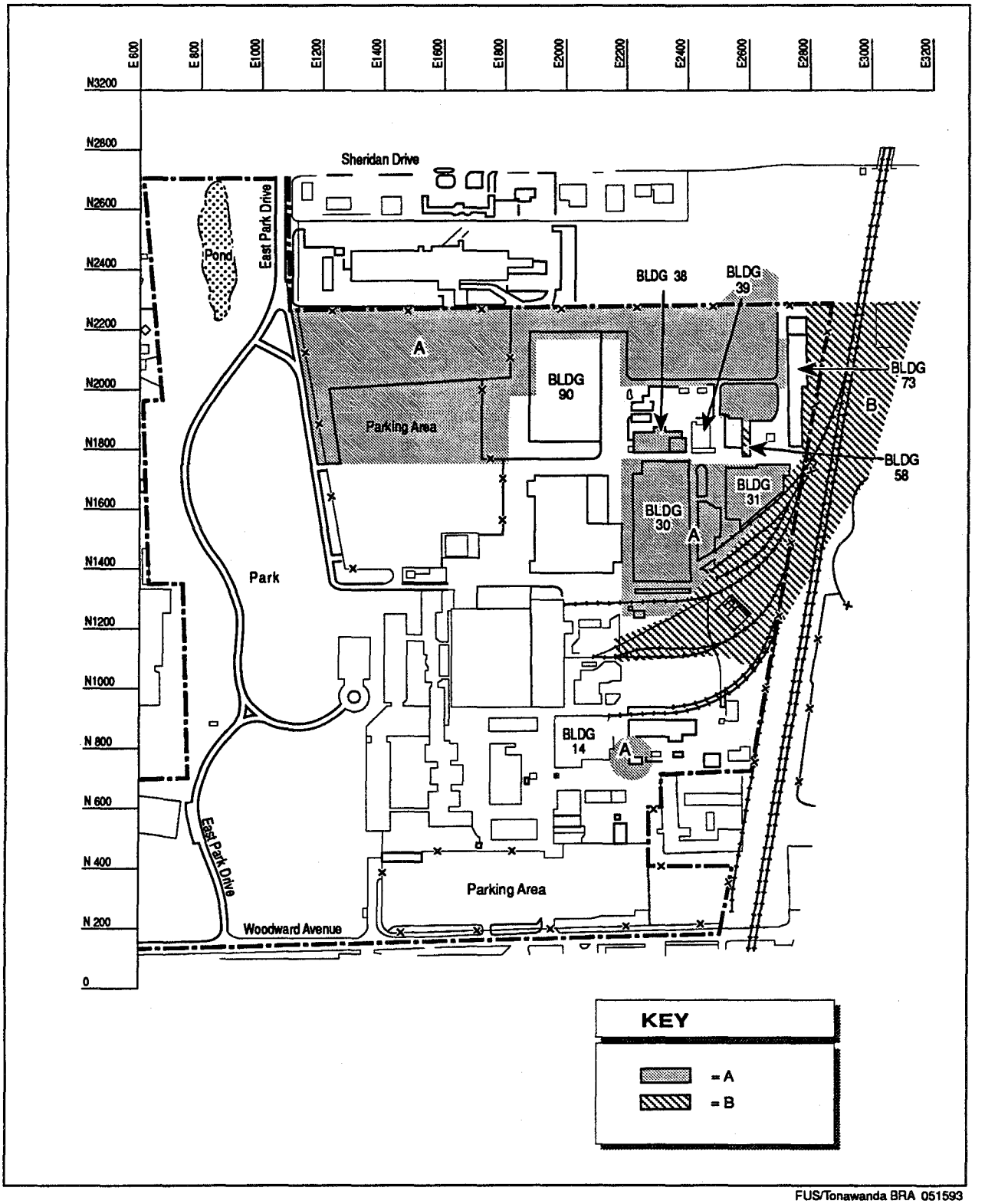

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Figure 1-5. Linde Subareas

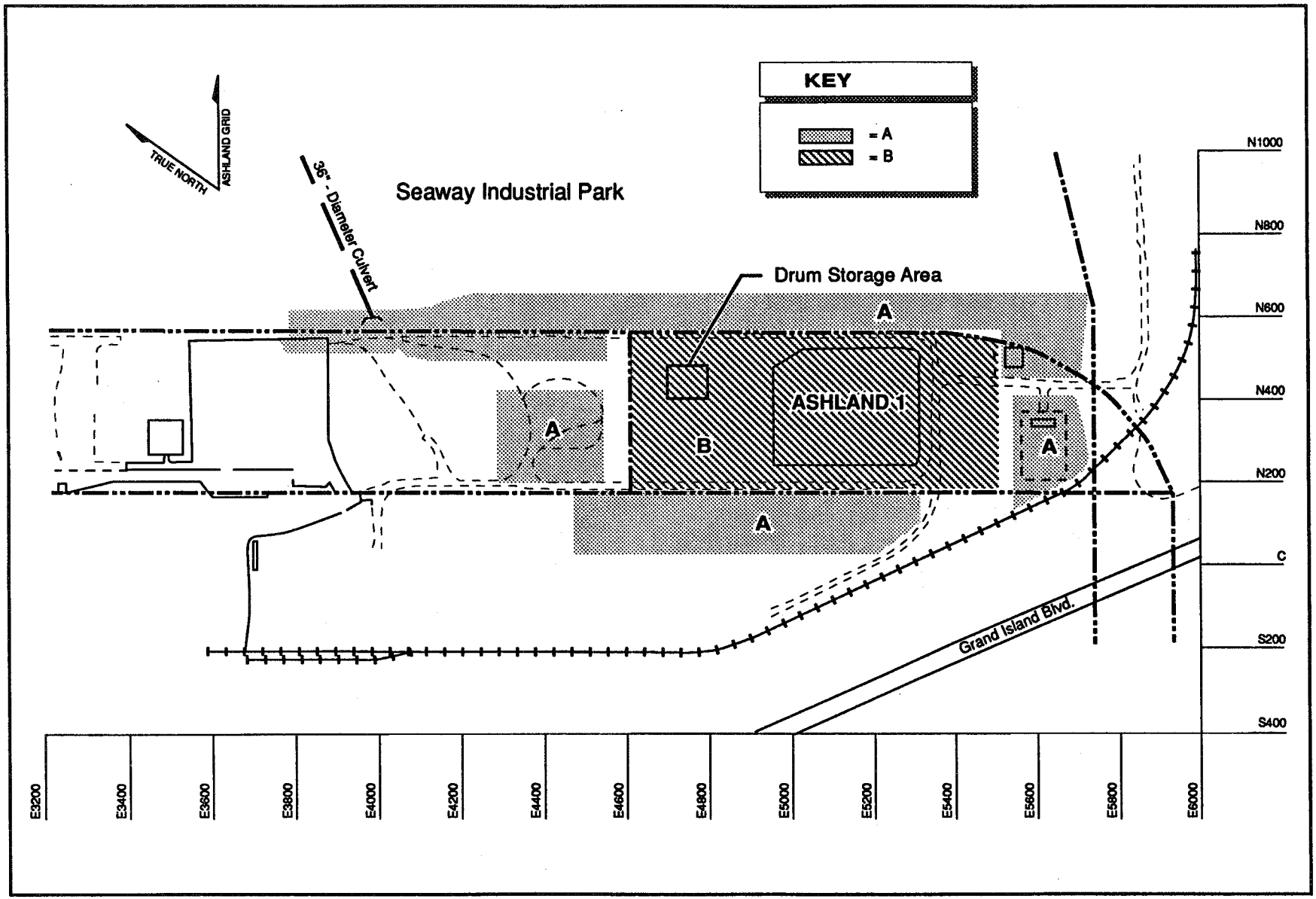

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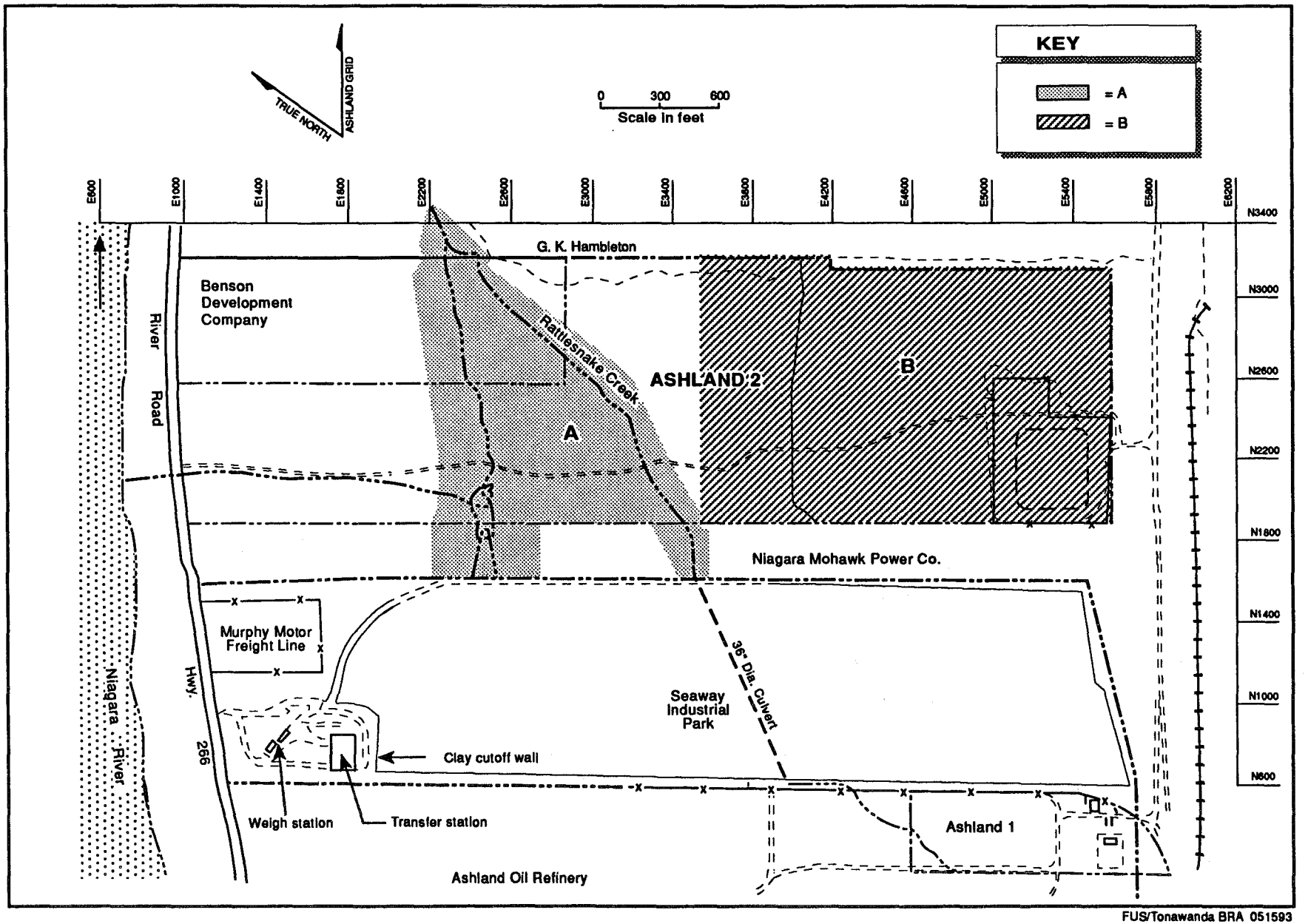
Figure 1-6. Ashland 1 Subareas



FUS/Tonawanda BRA 051593

Figure 1-7. Ashland 2 Subareas

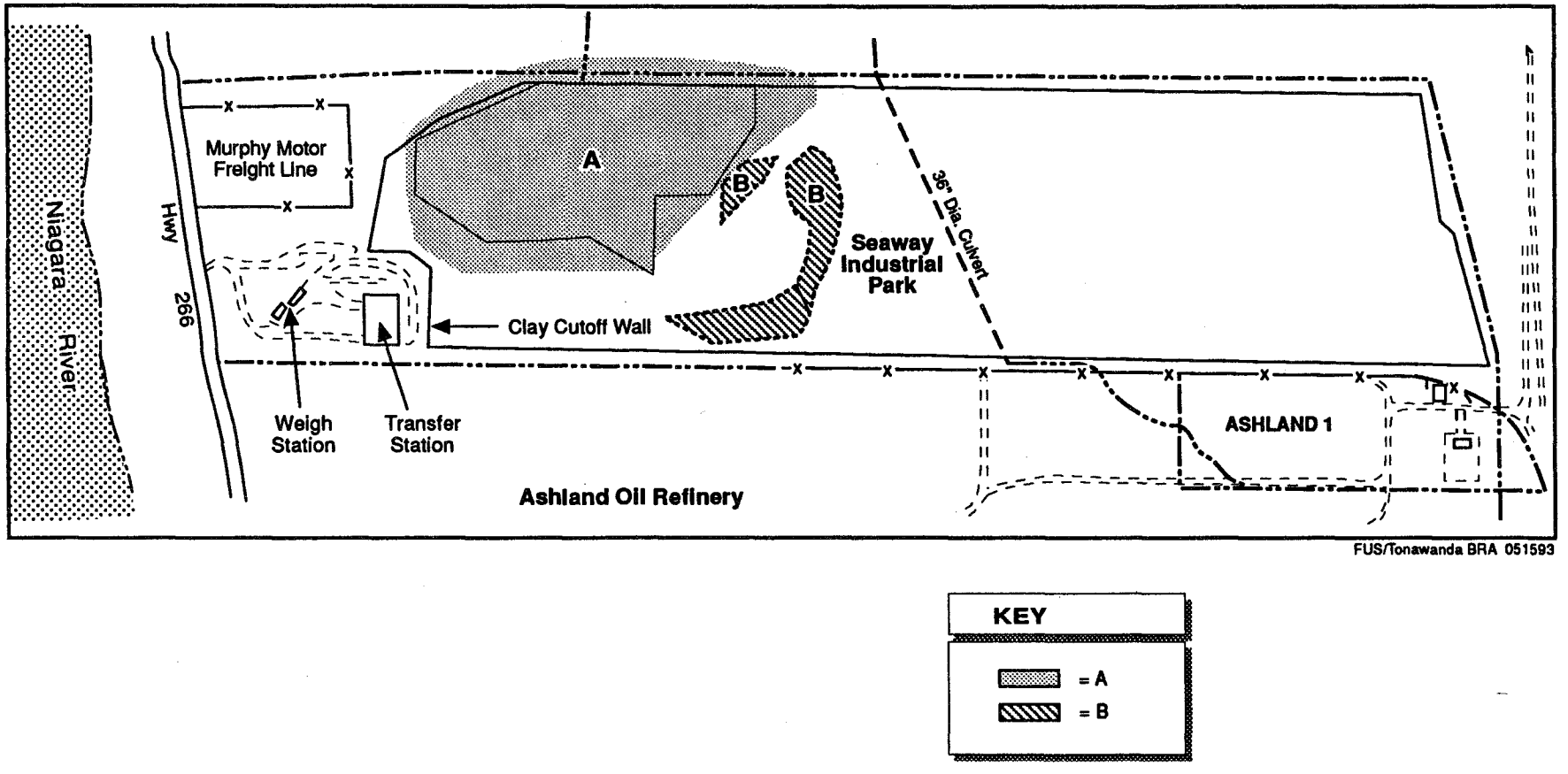


Figure 1-8. Seaway Subareas

2. IDENTIFICATION OF CONTAMINANTS OF CONCERN

2.1 SOURCES, TYPES, AND DISTRIBUTION OF CONTAMINANTS

The purpose of this section is to describe the process of identifying COCs and to present a summary of those selected for modeling exposure and risk. An important part of the process is evaluating not only the types and sources of contamination, but sampling, analysis, and modeling procedures used to estimate contaminant concentrations and distribution relative to the properties, media, and receptors that comprise the Tonawanda site.

Portions of the four properties that comprise the Tonawanda site (Linde, Ashland 1, Ashland 2, and Seaway) are contaminated with radionuclides and metals that originated from uranium ore processing at Linde. In addition, organic contamination has been detected at the Tonawanda site. The source of organic contamination is not considered MED-related (BNI 1992). The geographic distribution of properties and the history of several characterization studies have produced two distinct data collections, one for radiological and one for chemical contaminants, which are reflected in the organization and presentation of data results.

2.1.1 Radiological Contaminants

Radiological contaminants known or suspected to be present at the Tonawanda site properties resulted from uranium ore processing operations conducted at Linde. Radionuclides from the U-238, U-235, and Th-232 decay chains have been identified in the RI (BNI (1992).

2.1.2 Chemical Contaminants

Chemical contamination sources are described in the RI report (BNI 1992). The chemical contaminants include inorganic constituents present in the filter cake, effluents, fly ash and slag associated with the uranium ore extraction process. Numerous organic chemicals were detected at the Tonawanda site, including polycyclic aromatic hydrocarbons (PAHs), VOCs, and other semi-volatile organic compounds (SVOCs).

Organic contamination is not attributed to MED-related activities (BNI 1992). However, in this BRA, all chemical contaminants detected at the Tonawanda site are evaluated as potential COCs regardless of whether they are within the definition of FUSRAP wastes.

2.1.3 Assumptions

The procedures for selecting radiological and chemical COCs are described in the following sections and the results are presented in tabular format. The assumptions used to evaluate radiological and chemical data and to identify chemical COCs are discussed in Sections 2.2 and 2.3 and summarized in Appendix B.

2.2 RADIOLOGICAL DATA EVALUATION

Radiological contamination data have been collected in many survey and characterization studies conducted since 1978. These are discussed in the RI report (BNI 1992). Aerial mapping and ground surveys located properties with elevated radiation levels. These surveys were followed with more intensive near-surface gamma radiation surveys to more accurately define areas of radioactivity. Soil samples collected in areas of elevated radiation levels were analyzed using gamma spectroscopy to determine Th-232, Ra-226, and U-238 concentrations.

The radiological data used in this BRA were taken from the RI report for the Tonawanda properties (BNI 1992) and from radiological surveys conducted by Oak Ridge National Laboratory (ORNL) for DOE in 1978 at the Linde, Ashland 1, and Seaway sites (ORNL 1978a, b, and c) and by Ford, Bacon, and Davis, Utah, Inc. (FBDU) for DOE at the Seaway and Linde properties in 1981 (FBDU 1981a and b).

The validation and verification of RI report data are addressed in the RI report review process; therefore, the data are assumed to be acceptable for this evaluation. The historical data were collected under several quality assurance programs by various contractors over many years. Direct validation and verification of all these data may be impossible. Thus, the historical data are used where RI report data are unavailable and to supplement the RI report data.

The goal of the data evaluation is to identify a set of radiological COCs that are likely site-related and then select those COCs that are valid to use in the quantitative risk characterization. Radiological sample analyses for the RI were performed by Thermo Analytical/Eberline, (TMA/E) in accordance with approved protocols. The detailed analytical results are contained in appendices to the RI report (BNI 1992). Data quality objectives and Quality Assurance/Quality Control (QA/QC) procedures are discussed in Appendix D of the RI.

2.2.1 Rationale and Criteria for Selection of COCs

Samples from the following media were evaluated for potential radiological COCs; surface and subsurface soils; groundwater; surface water, and sediment from the drainage ditches. Sediment data and surface water data were aggregated across all drainages.

The radionuclides in the U-238, Th-232, and U-235 decay series (Figures 2-1, 2-2, and 2-3) that are expected to significantly contribute to site risk can be identified in a preliminary screening. A source term analysis indicates that the radiological hazards of the various radionuclides in the U-238 decay series can be determined from the activity concentrations of U-238, Th-230, and Ra-226. Activities of radionuclides from U-238 through U-234 can be assumed to be in secular equilibrium, because Th-234 and protactinium (Pa)-234 have short half-lives. Also, the activity of each individual radionuclide from Ra-226 through polonium (Po)-210 is assumed to be equal to that of Ra-226. The activity of U-235 (and progeny) was assumed to be equal to five percent of the U-238 activity, based on the typical natural abundance ratios of

these isotopes. The activities of the radionuclides in the Th-232 decay series, from Th-232 through Po-212 and thallium (Tl)-208, are assumed to be in secular equilibrium.

Mean contaminant concentrations were determined using detected results or the value of the quantitation limit, when results were reported as less than that value. Ubiquitous, naturally occurring radionuclides such as potassium (K)-40 were not considered in this BRA.

Statistical analysis was performed on the aggregated data sets to identify mean concentrations for each contaminant in these media. Radionuclides were selected as potential COCs if the mean detected concentrations exceeded twice the arithmetic mean background concentration for that radionuclide in a specific medium. For completeness, all radionuclides in the decay series of a given potential radiological COC were considered in the risk assessment.

2.2.2 Background

Background samples for each medium were used to identify naturally-occurring levels of radionuclides not affected by onsite sources. Radiological data were compared to arithmetic mean background levels to select the subset of radiological COCs appropriate for quantitative risk assessment, as described below.

2.2.2.1 Soil

Background levels of radionuclides in soil in the vicinity of the Tonawanda site were determined by sampling at locations that were considered to be undisturbed and within reasonable proximity of the site. Locations in the uncontaminated portion of Ashland 2 South were selected as representative of typical background radionuclide levels. A summary of the results is presented in Table 2-1.

2.2.2.2 Groundwater

Background levels of radionuclides in the groundwater in the vicinity of the Tonawanda site was determined by sampling well B29W05D, upgradient from the Linde property. A summary of the results is presented in Table 2-1.

2.2.2.3 Surface Water

A surface water sample was collected at location number 113, upstream of Ashland 1, Ashland 2, and Seaway. Results are presented in Table 2-1.

2.2.2.4 Sediment

The stormwater drainage system at Linde is covered and/or underground. The site contains large paved or covered areas, precluding significant erosion and sediment transport. The drainage system at Ashland 1, Ashland 2, and Seaway was considered as a single unit.

Three sampling locations upstream of these properties were sampled to determine arithmetic mean background concentrations. A summary of the results is presented in Table 2-1.

2.2.3 Soil

More than 1,600 surface and subsurface soil samples were collected from the Tonawanda site. Radiological analysis indicates widespread contamination of Ra-226, Th-232, and U-238 (BNI 1992). Table 2-2 summarizes the analytical results.

The sampling regime focused on the relatively long-lived isotopes. The progeny, as shown on Table 2-3, are assumed to be in equilibrium with their respective parent nuclides. Radionuclides in soils were considered on a sitewide basis rather than as property-specific contaminants. Therefore, some radionuclides are included as COCs in soils on properties or subareas where concentrations did not exceed twice background. This conservative methodology reduces complexity while addressing sitewide conditions.

2.2.4 Groundwater

Groundwater analysis data for radionuclides for sampling conducted in 1990 were used to screen potential COCs.

2.2.5 Surface Water

Surface water samples were collected from nine locations as shown on Table 2-2. Radiological analyses indicate that radionuclides are found at above background concentrations in the groundwater.

2.2.6 Sediment

Sediment samples were collected from 32 locations on the Ashland 1, Ashland 2, and Seaway properties. Results are summarized on Table 2-2.

2.2.7 Radiological COCs

The final list of radiological COCs for soil includes Ra-226, Th-230, U-238 and their associated decay products. Thorium-230, Th-232, Ra-226, and U-238 were identified as radiological COCs in surface water. Uranium-238 was the only radiological COC identified in groundwater. Thorium-230 and U-238 were identified as radiological COCs in sediment. Although not considered a COC during the sitewide screening, the Th-232 and U-235 series were included in the risk assessment.

2.3 CHEMICAL DATA EVALUATION

The chemical data evaluated are those reported in the RI report for the Tonawanda site (BNI 1992). The validation and verification of RI analytical data were performed as part of the RI data review process; therefore the data are considered acceptable for this evaluation. Samples from the following media were evaluated for chemicals of concern: surficial soil horizon (0 to 2 ft depth), surface water and sediment.

The chemical data are organized according to property and medium. Surface soil data were available for the Linde, Ashland 1 and Ashland 2 operable properties. There were no chemical data available for Seaway. As an operating municipal landfill, Seaway is likely to contain a wide variety of chemical contaminants. Isolation of FUSRAP-derived chemical contamination is not practicable. The uncertainty associated with this data gap is discussed in Section 5.

Surface water and sediment data are evaluated for Ashland 1, Ashland 2, and Seaway as single units. This decision was made based on limited data in some areas and on the uniform drainage pattern over most of the site (BNI 1992). A depiction of the site drainage is found on Figure 2-5. The Linde surface water and sediment evaluation was not addressed in the BRA because there was no complete onsite exposure pathway present due to the fact that stormwater drainage system is covered and/or underground (BNI 1992). The generally level topography, coupled with the large paved or covered areas precludes significant erosion and sediment transport. See Section 3.2 for a more complete discussion of exposure pathways.

The groundwater in the area is drawn from the Camillus Shale. This formation is the most productive bedrock aquifer in the region, but it also contains the poorest water quality (La Sala 1968). Because of the high levels of total dissolved solids, sulfates and chlorides, the water from this formation is considered nonpotable without extensive, costly treatment (BNI 1992). Therefore, the groundwater was not evaluated due to the lack of a complete exposure pathway.

Chemical sample analyses for the RI report were performed by Roy F. Weston, Inc., analytical laboratories, in accordance with approved protocols. The detailed analytical results are contained in Appendix E to the RI report (BNI 1992).

The QA/QC Evaluation process is discussed in Appendix D of the RI. A detailed discussion of the QA/QC procedures is presented in the *Quality Assurance Project Plan for the Remedial Investigation/Feasibility Study-Environmental Impact Statement for the Tonawanda Site* (BNI 1991b).

After samples were analyzed, results were reviewed for precision, accuracy, completeness, and representativeness. Upon successful completion of the QA/QC process, data were included in the overall site database. QC samples were used to assess data quality for precision and accuracy and to document that sampling and analysis procedures did not introduce variables that would render the data questionable. QC samples included field blanks and

duplicates, method blanks and spikes, matrix spikes and duplicates, laboratory duplicates, and standard reference materials. The guidance documents used in the assessment and qualification of chemical data are the *Laboratory Data Validation: Functional Guidelines For Evaluating Organic Analysis (EPA 1988a) and the Laboratory Data Validation: Functional Guidelines For Evaluating Inorganic Analyses* (EPA 1988b).

2.3.1 Rationale and Criteria for Selection of COCs

Chemical data were aggregated by property units and medium for evaluation of COCs. These aggregates were:

Medium	Property
Soil	1. Linde
	2. Ashland 1
	3. Ashland 2

In addition to these formally designated properties, the following additional subarea was considered for aggregating chemical COCs:

Surface Water and Sediment	4. Ashland 1, Ashland 2, and Seaway

Only surficial soil (0-2 ft) was evaluated for the first three properties because the limited erosion is not expected to expose significant areas of the subsurface soils. Also, repeated, prolonged contact with subsurface soils by any of the credible receptors discussed in Section 3 is unlikely. Surface water and sediment data were aggregated as single units. Statistical analysis was performed on the aggregated data sets to determine mean concentrations for each contaminant in these media (see Section 3.4 and Appendix C).

Chemicals in the RI database were evaluated in accordance with EPA data validation guidance in *Risk Assessment Guidance for Superfund, Volume I* (EPA 1989a). Background samples for soil were used to identify naturally occurring levels of chemicals and ambient concentrations attributable to onsite sources.

Data presented in the RI Report were evaluated on the basis of quality, with respect to sample quantitation limits, laboratory qualifiers and codes, and blanks. Data selected to use in the BRA include unqualified data, those data with qualifiers that indicate uncertainties in concentrations but not in compound identification, and those data detected at levels significantly elevated above concentrations detected in associated sample blanks. Organic contaminant data selected include those with no qualifiers and those designated with = (no data qualifier required), J (estimated value, but below method detection limit), B (detected in associated laboratory blank), or E (beyond calibration limits of instrument and subsequently analyzed). Inorganic contaminant data selected include those with no qualifier and those designated with =, J, B (reported value less than Contract Required Detection Limit but greater than or equal

to Instrument Detection Limit and detected in associated laboratory blank), BJ (both B and J), or E (estimated value). All data with any other qualifier or combination of qualifiers were excluded from the BRA database. Chemical data were evaluated according to the following criteria to select the subset of COCs appropriate for quantitative risk assessment:

- Comparison to Background - an inorganic chemical was eliminated if the mean concentration of the sample population was less than twice the mean background concentration; the statistical basis for detected sample population distributions (i.e., normal or lognormal) can be found in Section 3.4;

- Comparison to Quantitation Limits - inorganic and organic chemicals were eliminated if the chemical was detected only once or twice in a medium and if detected concentrations were less than contract required quantitation limits; and

- Frequency of Detection - when there were twenty or more samples, chemicals that were detected at a frequency of five percent or less were eliminated.

2.3.2 Background

2.3.2.1 Soils

It is important to establish soil chemical constituent concentrations that represent background conditions at the Tonawanda site. For the RI, BNI determined site-specific background levels of radionuclides and metals instead of using the area-wide background values reported earlier. These site-specific background values are based on concentrations in samples collected from Ashland 2 South (the southern portion of the Ashland 2 property); samples were analyzed for metals. The background sampling locations chosen were selected for the following reasons:

(1) The sampling locations are representative of the natural Tonawanda site soils because the samples were collected from undisturbed soils at Ashland 2 South, where the depth to undisturbed soil generally is between 0 and 0.15 m (0 and 0.5 ft).

(2) The sampling locations are removed from the area of radioactive contamination to the northwest and the Ashland tank and possible area of chemical contamination to the southeast.

(3) The locations are not in the stormwater-runoff flowpath of any areas of contamination.

Table 2-4 summarizes the background soil concentrations.

VOC analyses were not performed on the background soil samples because VOCs are not considered to be naturally occurring, and it is anticipated that their background concentrations are non-existent. Complete analytical results are reported in Appendix E of the RI report (BNI 1992).

2.3.2.2 Surface Water and Sediment

Surface water and sediment samples were collected from two locations upgradient of the Ashland 1 property. Figure 2-4 depicts the locations of GS-5 and AS-8, the upgradient samples. Because the surface water samples showed elevated levels of contaminants not found in downgradient samples, they were determined to be unsuitable as a background for the site; therefore, no background comparison could be performed for the surface water.

The upgradient sediment samples also exhibited elevated levels of contaminants; however, the lower value of the two upgradient samples was used for the background concentration.

2.3.3 Soil

Soils were evaluated for three properties as described in Section 2.3.1.

2.3.3.1 Linde

General site characterization activities were conducted at Linde from October 1988 through March 1989 to investigate two potential contaminant sources: (1) contaminated soil outside the buildings and beneath Building 30, and (2) portions of Buildings 14, 30, 31, and 38 to confirm previous survey results.

Supplemental investigations were conducted from November 1990 through May 1991 to investigate four potential contaminant sources.

A total of 13 surface soil samples were collected from the four areas and analyzed for metals. Twenty samples were analyzed for base/neutral and acid extractables (BNAEs) and one for VOCs (BNI 1992).

Twenty-three metals were detected in the surficial soil horizon (Table 2-5). Aluminum, barium, boron, calcium, chromium, copper, iron, lead, magnesium, manganese, and zinc were detected in all 13 samples. Three VOCs and 19 BNAEs were detected in the surficial soil horizon (Table 2-6).

Thirteen inorganics were eliminated as COCs because the mean concentration was less than twice the mean background. Fifteen VOCs and two BNAEs were detected less than three times and at concentrations below contract required limits, or the chemical was not detected (Table 2-7).

2.3.3.2 Ashland 1

First phase site characterization activities were conducted at Ashland 1 from October 1988 through March 1989 to determine whether MED-related filter cake material was still present and to determine the material's migration potential to the groundwater and surface water/sediment regime.

Second phase selective investigations were conducted from November 1990 through May 1991 to further refine the boundaries of radioactive contamination in the soil and determine the potential for RCRA-hazardous waste in the soil.

A total of 12 surface soil samples were collected and analyzed for metals, 31 samples were analyzed for VOCs, 11 for BNAEs, and 12 for pesticides/PCBs (BNI 1992).

Twenty-four metals were detected in all of the surficial soil horizon samples (0-2 ft). Aluminum, barium, calcium, chromium, copper, iron, lead, magnesium, manganese, nickel and thallium were detected in all of the samples (Table 2-8). Additionally, two VOCs, 19 BNAEs, and one pesticide were detected in the surficial soil zone (Table 2-9).

Eighteen metals were eliminated as COCs from Ashland 1 surface soil because the mean concentration was less than twice the mean background concentration. Six BNAEs were detected less than three times and were present at a concentration below contract required limits, or the chemical was not detected (Table 2-7).

2.3.3.3 Ashland 2

Site characterization activities were conducted at Ashland 2 during the same time frame as those conducted at Ashland 1.

Eighteen surface soil samples were collected and analyzed for metals, two for VOCs, 11 for BNAEs, and 11 for pesticides/PCBs.

Twenty-two metals were detected in the surficial soil horizon with aluminum, barium, calcium, chromium, copper, iron, lead, magnesium, manganese, nickel, potassium, vanadium, and zinc detected in all of the samples (Table 2-10). Additionally, six VOCs and seven BNAEs were detected in the surface soils (Table 2-11).

Thirteen metals were eliminated as COCs from Ashland 2 surficial soils because the mean was less than two times the mean background concentration. Two VOCs and four BNAEs were detected less than three times and were present at concentrations below the contract required limits, or the chemical was not detected (Table 2-11).

2.3.4 Surface Water and Sediment

The surface water and sediment evaluations were performed on data aggregated as single units, encompassing: Ashland 1, Ashland 2, and Seaway. Samples GS-4 and GS-6 were eliminated from the evaluation due to the fact that they were oil refinery indicator samples (Figure 2-4). Also eliminated from the evaluation were samples SP-10, SP-11, and LOC 3 due to their remote proximity.

2.3.4.1 Surface Water

Fifteen surface water samples were collected and analyzed for metals, twelve for BNAEs, and fourteen for VOCs. Results from the inorganic analyses are summarized on Table 2-12, organic results are summarized on Table 2-13. Four VOCs and fourteen BNAEs were eliminated because they were detected less than three times at concentrations below contract required limits. The containment screening process is summarized on Table 2-14. No contaminants were eliminated by the background comparison screen since there was no valid background sample.

2.3.4.2 Sediment

The sediment sampling program encompassed 15 samples. Twenty metals were detected, with aluminum, calcium, chromium, iron, manganese, selenium, and zinc found in each sample. Results are summarized on Table 2-15. Four metals remained as COCs in sediment: aluminum, calcium, magnesium, and vanadium. The contaminant screening process is summarized on Table 2-16.

2.3.5 Contaminants of Concern

COCs detected at the Tonawanda site were screened according to the EPA guidance for data evaluation (EPA 1989a). Uranium was retained as a COC for the purpose of assessing potential risk from nonradiological effects. Toxicity information for these COCs was then reviewed to ascertain the availability of chemical-specific toxicity data. Appropriate toxicity data values were not available for several contaminants, thereby precluding their inclusion in the quantitative risk assessment. These COCs are denoted by an asterisk (*) in Tables 2-5 through 2-15. The final list of COCs retained for risk assessment in all media is summarized in Table 2-17. The COCs for soil include 4 metals, 5 VOCs, and 19 BNAEs. The COCs for surface water include 25 metals, 3 VOCs, and 7 BNAEs. The COCs for sediments include four metals. BNAEs were the predominant COCs in soils, while metals were prevalent COCs in surface water and sediment.

2.4 SUMMARY OF COCs

2.4.1 Radiological Contaminants

The potential radiological COCs for the Tonawanda site were screened according to EPA guidance (EPA 1989a) to identify a list of COCs for the quantitative risk assessment. Radiological data were aggregated by medium before screening. The screening rationale and criteria are discussed in Section 2.2.1. The final list of COCs for the risk assessment is comprised of those radionuclides (Table 2-3) that remained after application of the screening criteria.

2.4.2 Chemical COCs

The final list of COCs for the risk assessment is comprised of those chemicals that remained after application of the screening criteria discussed in Section 2.3.1, and for which appropriate toxicity factors were available. In addition, uranium was retained as a COC for evaluation of risk from nonradiological effects.

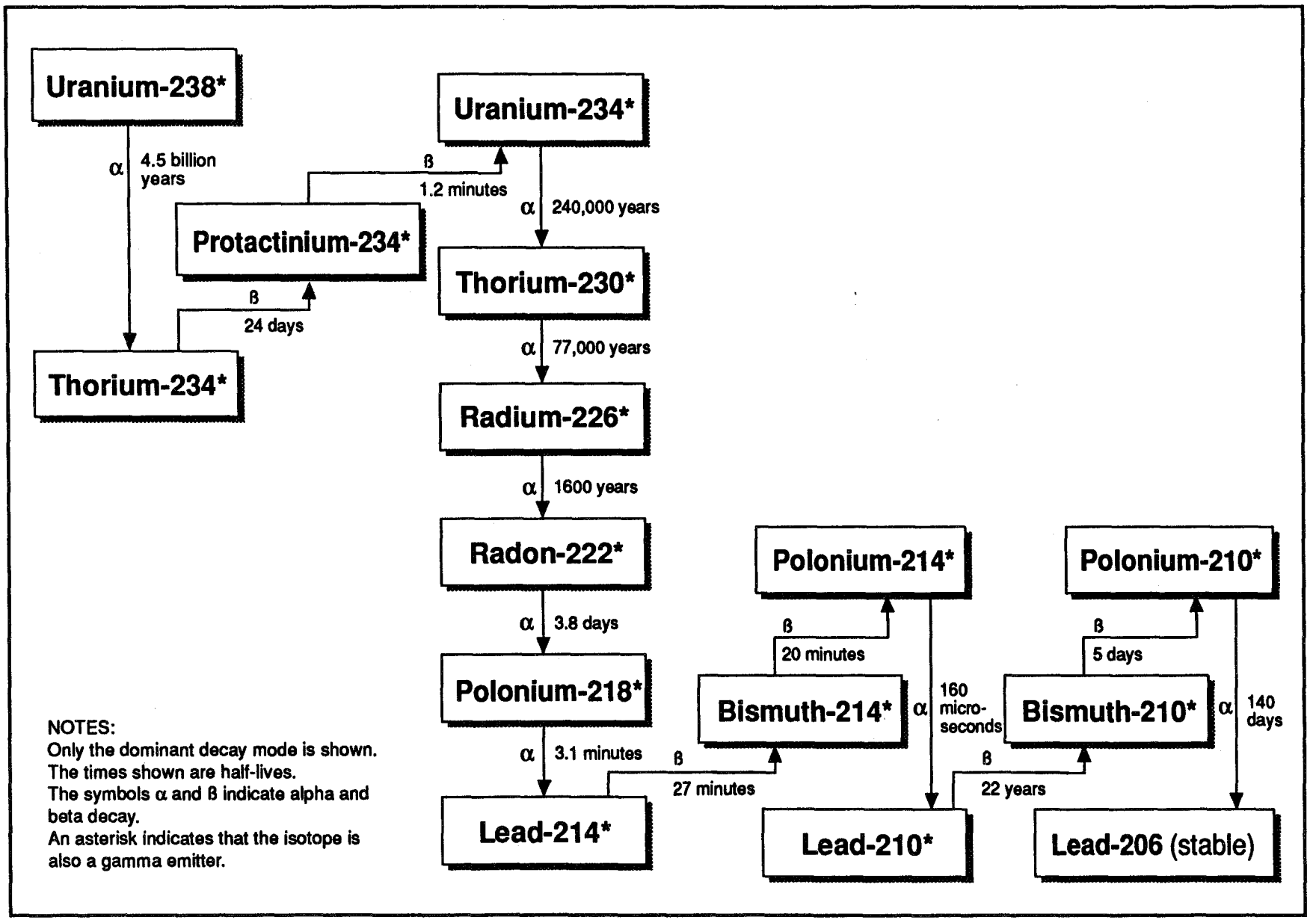
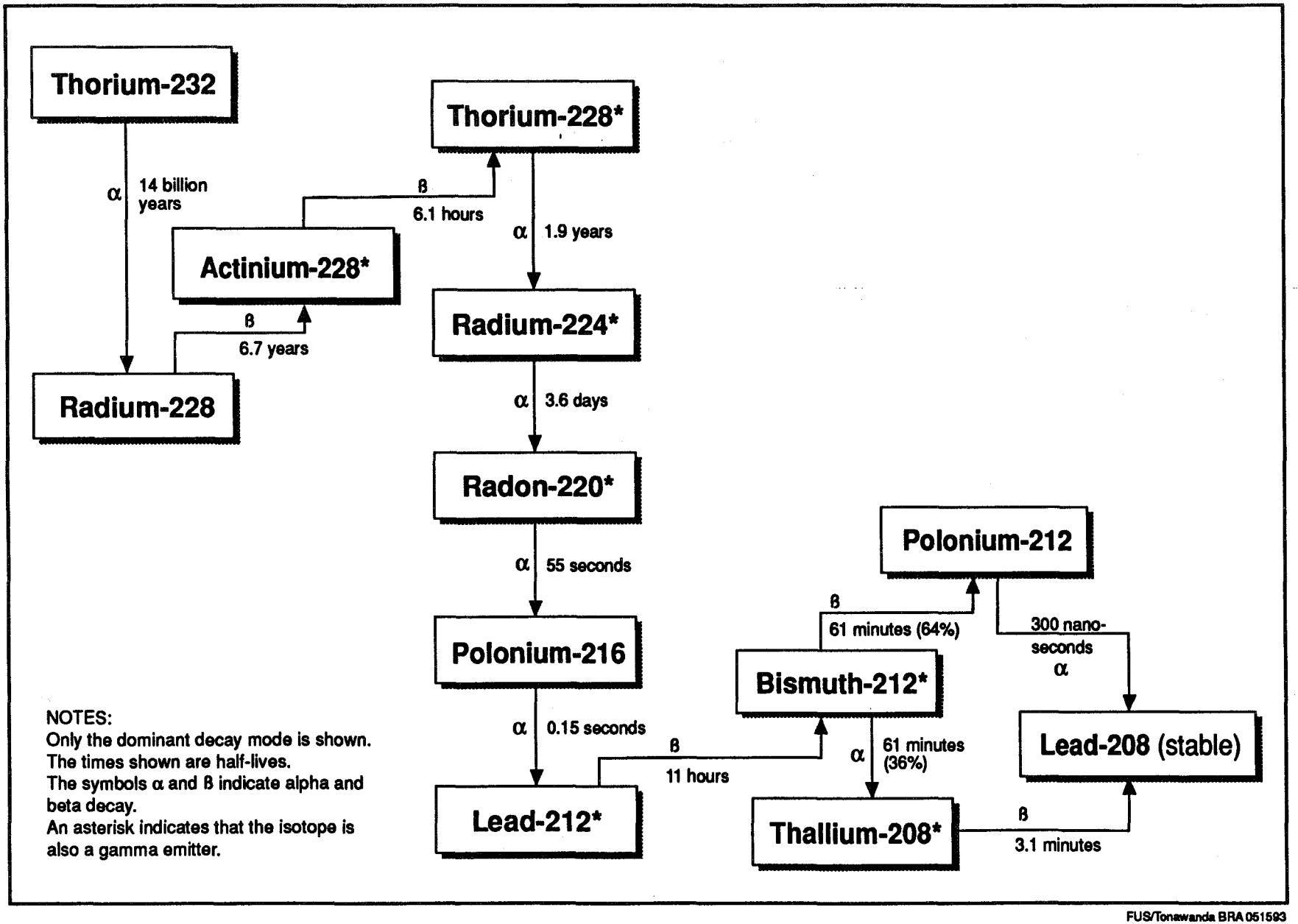
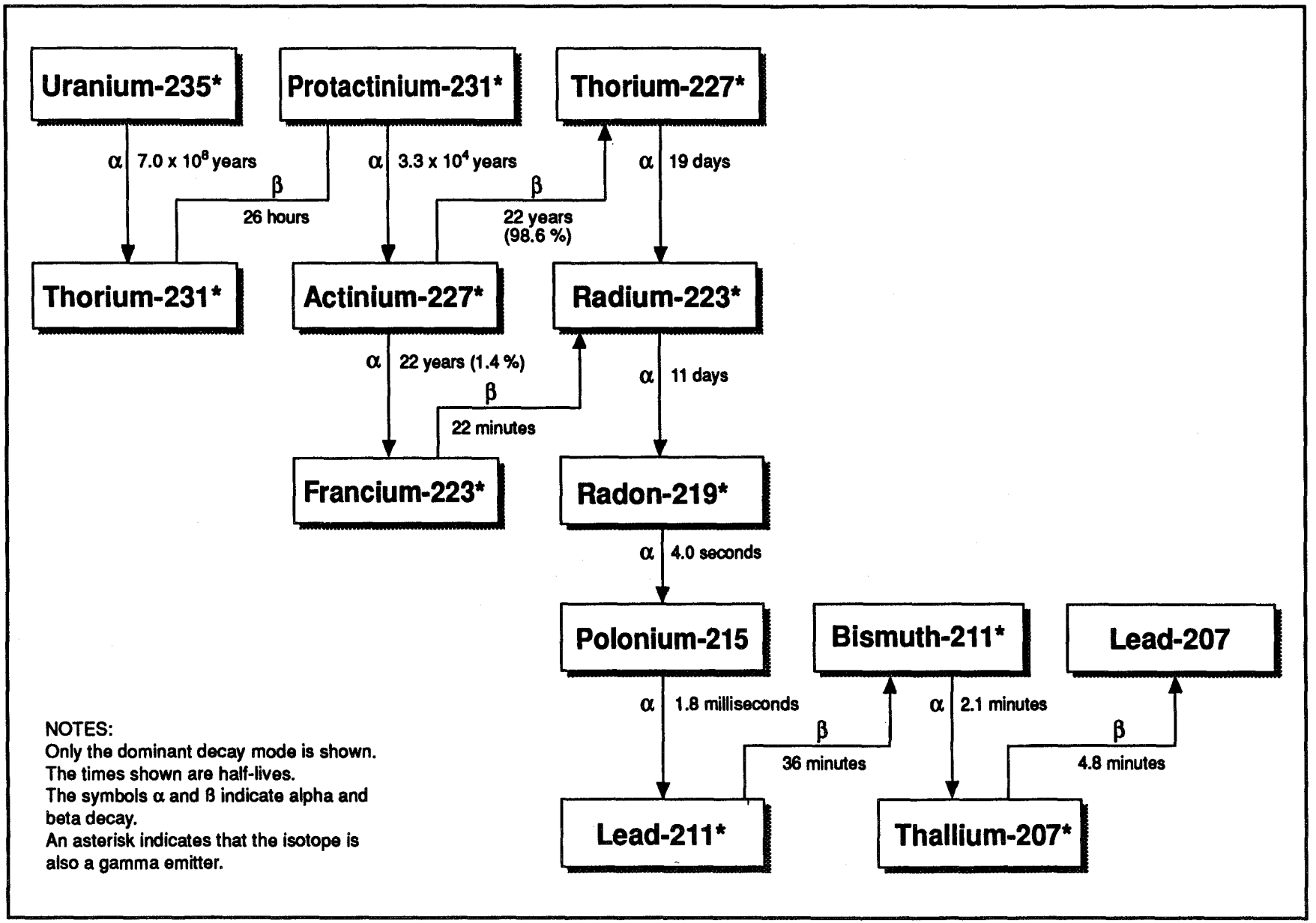


Figure 2-1. Uranium-238 Radioactive Decay Series



FUS/Tonawanda BRA 051593

Figure 2-2. Thorium-232 Radioactive Decay Series



FUS/Tonawanda BRA 051593

Figure 2-3. Uranium-235 Radioactive Decay Series

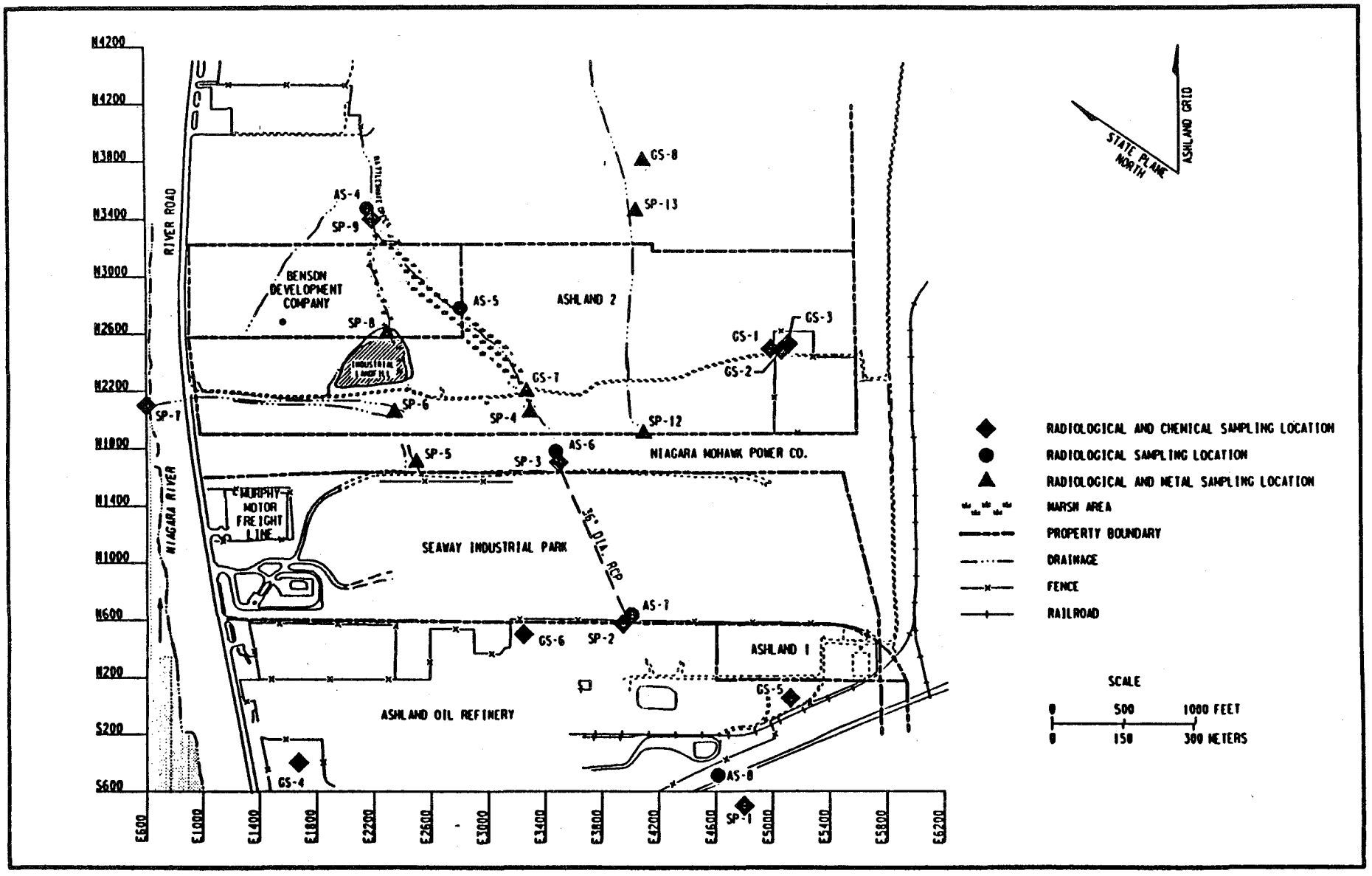


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Figure 2-4. Surface Water & Sediment Characterization Samplings Locations at Ashland 1 and Ashland 2

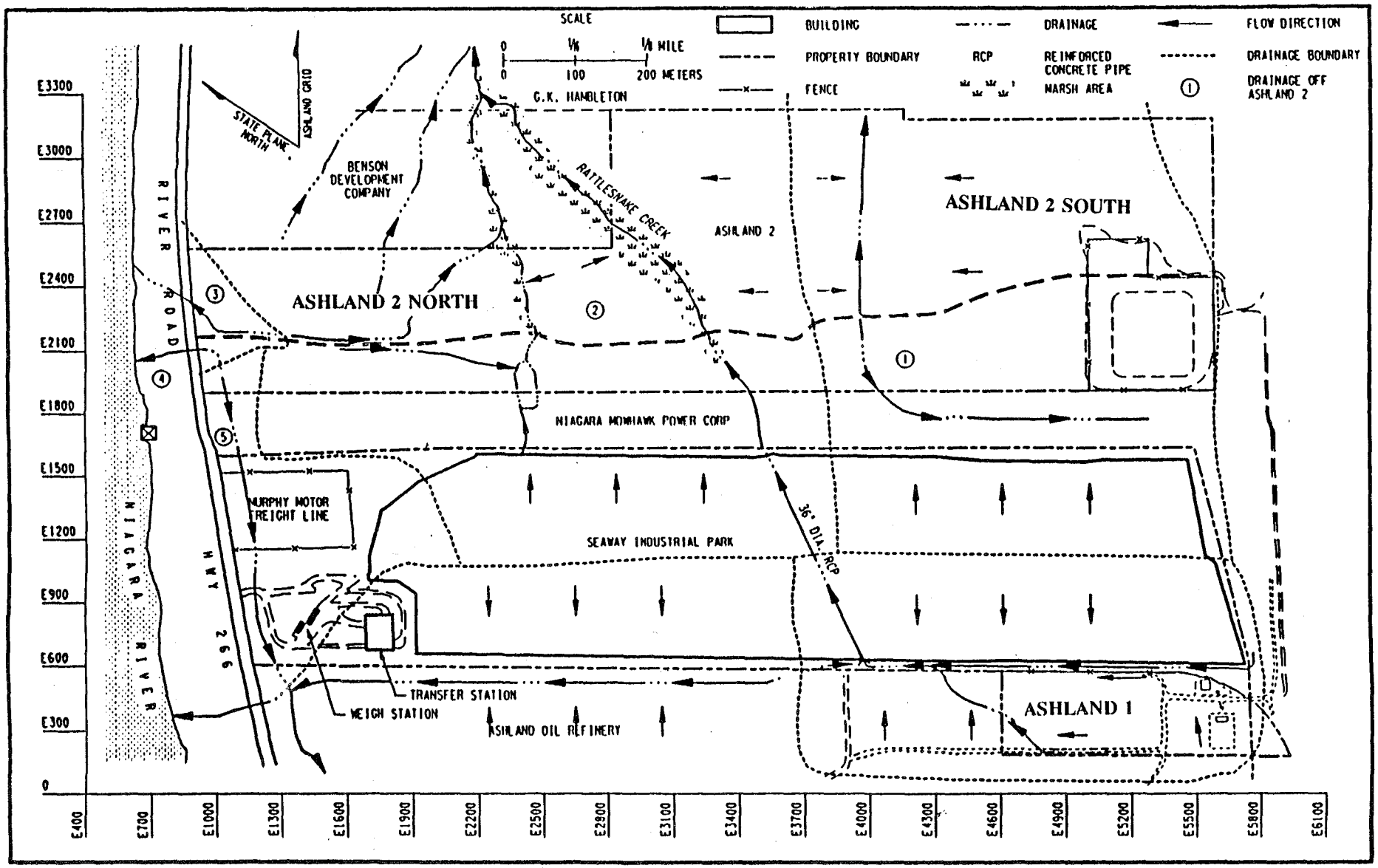

Figure 2-5. Drainage at Ashland 1, Ashland 2 and Seaway

Table 2-1. Analytical Results for Background Radionuclide Concentration

Arithmetic Mean Concentration					
Medium	**Units**	**U-238**	**Ra-226**	**Th-230**	**Th-232**
Soil	pCi/g	3.1	1.1	1.4	1.2
Groundwater	pCi/l	0.3	2.8	0.2	0.2
Surface Water	pCi/l	1.1	0.3	0.1	0.1
Sediment	pCi/g	1.8	0.8	1.1	1.4

Source: BNI 1992

Table 2-2. Screening of Source Area Contaminants for Tonawanda, NY Site

Nuclide	Detections	Matrix	Maximum Concentration (pCi/g)	Mean Concentration (pCi/g)	Background Concentration (pCi/g)	COC
Ra-226	1176	subsurface soil	750	6.0	1.1	YES
Ra-226	449	surface soil	813	9.8	1.1	YES
Th-230	483	subsurface soil	4400	68.0	1.4	YES
Th-230	262	surface soil	2200	58.3	1.4	YES
Th-232	764	subsurface soil	20	1.4	1.2	NO
Th-232	308	surface soil	27	1.4	1.2	NO
U-238	983	subsurface soil	4300	33.0	3.1	YES
U-238	374	surface soil	12000	74.0	3.1	YES
Ra-226	32	sediment	3.3	.12	0.8	NO
Th-230	32	sediment		5.5	1.4	YES
Th-232	25	sediment	1.9	1.1	1.1	NO
U-238	32	sediment	24	4.1	1.8	YES
Nuclide	**Detections**	**Matrix**	**(pCi/l)**	**(pCi/l)**	**(pCi/l)**	**COC**
Ra-226	11	surface water	3.3	0.9	0.3	YES
Th-230	11	surface water	13	1.4	0.1	YES
Th-232	6	surface water	1.4	0.3	0.1	YES
U-238	11	surface water	470	75	1.1	YES
Ra-226	8	groundwater	7.4	1.9	2.8	NO
Th-230	7	groundwater	0.6	0.3	0.2	NO
Th-232	7	groundwater	0.5	0.2	0.2	NO
U-238	8	groundwater	3.8	1.0	0.3	YES

Table 2-3. Groupings of Radionuclides

Parent Radionuclide	Principal Radionuclide	Associated Decay Products[a]
Thorium-232		
	Thorium-232	
	Radium-228 + progeny	Actinium-228
	Thorium-228 + progeny	Radium-224, Radon-220, Polonium-216, Lead-212, Bismuth-212, Polonium-212, Thallium-208
Uranium-238		
	Uranium-238 + progeny	Thorium-234, Protactinium-234
	Uranium-234	
	Thorium-230	
	Radium-226 + progeny	Radon-222, Polonium-218, Lead-214, Bismuth-214, Polonium-214, Lead-210, Bismuth-210, Polonium-210
Uranium-235		
	Uranium-235 + progeny	Thorium-231
	Protactinium-231	
	Actinium-227 + progeny	Thorium-227, Radium-223, Radon-219, Polonium-215, Lead-211, Bismuth-211, Thallium-207

[a] Assumed to be in secular equilibrium with the principal radionuclides.
Source: Gilbert et al. 1989

Table 2-4. Background Concentrations of Metals in Soil, Ashland 2 South and Tonawanda Area

Analyte	Ashland 2 South[a]			Erie County[b]	Rural Value[c]	Urban Value[d]
	Range	Mean	Std. Dev.			
Metals (ppm):						
Aluminum	9280 - 18600	13975.56	3324.21	30000	18300	10500
Antimony	6.6 - 10	8.24	1.21	ND	<60	<20
Arsenic	16.5 - 25.1	20.47	2.95	13	3.2	7
Barium	80.2 - 165	112.81	27.77	500	49.1	246
Beryllium	0.83 - 1.4	1.08	0.18	ND	<0.8	0.7
Boron	16.5 - 25.1	20.47	2.95	70	NA	NA
Cadmium	0.83 - 1.3	1.03	0.16	ND	<4	2.3
Calcium	1490 - 66100	32876.67	26798.81	2800	2520	26800
Chromium	17.2 - 27.4	21.48	3.73	30	12.5	31
Cobalt	8.3 - 12.5	10.53	1.36	15	<4	7.8
Copper	14 - 25.8	18.28	3.73	20	15.3	65
Iron	16400 - 31500	23700	4816.12	30000	15300	24700
Lead	24.1 - 48.4	36	9.78	30	41.4	557
Magnesium	3020 - 18400	10421.11	5807.47	7000	1840	11300
Manganese	224 - 1060	542.56	251.93	300	107	489
Molybdenum	16.5 - 25.1	20.47	2.95	3	<20	<8
Nickel	18 - 29	22.52	4.14	15	14.1	26
Potassium	1050 - 2710	1625.56	627.26	16600	5330	923
Selenium	96.4 - 192	149.16	33.36	0.4	<0.5	NA
Silver	1.7 - 5.3	2.91	1.33	ND	<8	<4
Sodium	826 - 1250	1023	146.25	7000	<400	<400
Thallium	34.3 - 68.3	48.36	10.71	ND	<10	<10
Vanadium	19.5 - 31.8	24.51	3.58	70	22.9	39
Zinc	66.1 - 102	84.77	15.36	63	73	<20

Source: BNI 1992

[a] Samples taken from boreholes 55G030, 55G0031, 55G034, 55G039, and 55G045. Boring logs indicate that all samples from these boreholes had similar texture.

[b] USGS 1981

[c] Samples taken in West Seneca, New York, approximately 15 miles southeast of Tonawanda, on Reserve Road (New York Department of Health data; considered background for hazardous waste site evaluations).

[d] Samples from Buffalo residential area (New York Department of Environmental Conservation Kingsley Park Investigation).

ND = Not Detected.

Table 2-5. Potential Inorganic Contaminants of Concern in the Linde Site Surficial Soils (0 - 2 ft.)

Contaminant	Frequency of Detection	Range of Detected Concentrations (mg/kg)	Mean Detected Concentration (mg/kg)	Mean Background Concentration (mg/kg)
Metals				
aluminum	13 / 13	7380 - 30800	19536.92	13975.56
antimony	2 / 13	13.2 - 91.5	33.75	8.24
* arsenic	12 / 13	55.2 - 207	106.45	20.47
* barium	13 / 13	96.2 - 499	256.32	112.81
* beryllium	11 / 13	1.3 - 6.3	4.19	1.08
* boron	13 / 13	29.1 - 94.1	52.06	20.47
* cadmium	4 / 13	1.2 - 6	0.97	1.03
* calcium	13 / 13	24700 - 198000	114361.54	32876.67
chromium	13 / 13	9.5 - 45.3	16.66	21.48
cobalt	1 / 13	68.8	67.80	10.53
* copper	13 / 13	13 - 1080	77.55	18.28
iron	13 / 13	5140 - 27200	13063.85	23700.00
* lead	13 / 13	29.2 - 1120	71.18	36.00
magnesium	13 / 13	4810 - 36300	12344.27	10421.11
* manganese	13 / 13	570 - 3070	1670.69	542.56
* nickel	11 / 13	12.7 - 265	34.07	22.52
potassium	8 / 13	1190 - 2540	1575.00	1625.56
selenium	12 / 13	33.4 - 216	104.35	149.16
silver	5 / 13	2.7 - 5	2.88	2.91
sodium	7 / 13	949 - 3240	1559.38	1023.00
thallium	3 / 13	28.8 - 80.8	40.45	48.36
vanadium	10 / 13	13.8 - 437	28.04	24.51
zinc	13 / 13	22.7 - 634	87.12	84.77

* Contaminant of Concern

Note: Chemicals analytically evaluated and not detected are not shown. See Table 2-7

Table 2-6. Potential Organic Contaminants of Concern in the Linde Site Surficial Soils (0-2 ft.)

Contaminant	Frequency of Detection	Range of Detected Concentrations (µg/kg)
BNAEs		
* 2-methylnaphthalene	4 / 19	60 - 830
* acenaphthene	3 / 20	120 - 820
* anthracene	6 / 20	74 - 870
* benzo(a)anthracene	8 / 20	150 - 3100
* benzo(a)pyrene	7 / 20	99 - 3000
* benzo(b)fluoranthene	8 / 20	89 - 3200
* benzo(g,h,i)perylene	6 / 20	85 - 2200
* benzo(k)fluoranthene	8 / 20	83 - 3100
* bis(2-ethylhexyl)phthalate	6 / 20	190 - 750
* chrysene	8 / 20	180 - 3900
* di-n-butylphthalate	6 / 20	54 - 280
* dibenzo(a,h)anthracene	2 / 20	380 - 420
* dibenzofuran	4 / 19	56 - 640
* fluoranthene	8 / 20	310 - 7000
* fluorene	4 / 20	94 - 660
* indeno(1,2,3-cd)pyrene	4 / 20	93 - 2100
* naphthalene	5 / 19	58 - 960
* phenanthrene	8 / 20	130 - 4700
* pyrene	8 / 20	240 - 6200
VOCs		
* acetone	1 / 1	66
* methylene chloride	1 / 1	11
* toluene	1 / 1	5.6

* Contaminant of Concern

Note: Chemicals analytically evaluated and not detected are not shown. See Table 2-7.

Table 2-7. Potential Contaminants of Concern In Soil Eliminated from the Risk Assessment

Contaminant	Screening Rationale
LINDE	
Metals	
aluminum	1
antimony	1
chromium	1
cobalt	1
iron	1
magnesium	1
potassium	1
selenium	1
silver	1
sodium	1
thallium	1
vanadium	1
zinc	1
BNAEs	
diethylphthalate	2
dimethylphthalate	2
VOCs	
acrolein	2
acrylonitrile	2
benzene	2
bromodichloromethane	2
bromoform	2
bromomethane	2
carbon disulfide	2
carbon tetrachloride	2
chlorobenzene	2
chloroethane	2
chloroform	2
chloromethane	2
cis-1,3-dichloropropene	2
dibromochloromethane	2
ethylbenzene	2

Table 2-7. (continued)

Contaminant	Screening Rationale
ASHLAND 1	
Metals	
aluminum	1
antimony	1
arsenic	1
barium	1
beryllium	1
boron	1
cadmium	1
chromium	1
cobalt	1
iron	1
manganese	1
molybdenum	1
nickel	1
potassium	1
selenium	1
silver	1
sodium	1
thallium	1
BNAEs	
acenaphthene	2
anthracene	2
benzoic acid	2
dibenzofuran	2
diethylphthalate	2
indeno(1,2,3-cd)pyrene	2

Table 2-7. (continued)

Contaminant	Screening Rationale
ASHLAND 2	
Metals	
aluminum	1
barium	1
boron	1
calcium	1
chromium	1
iron	1
manganese	1
molybdenum	1
potassium	1
selenium	1
silver	1
sodium	1
thallium	1
BNAEs	
benzo(a)anthracene	2
benzo(a)pyrene	2
bis(2-ethylhexyl)phthalate	2
fluorene	2
VOCs	
benzene	2
toluene	2

Screening Rationale Key

1 - Mean of detected concentrations was less than twice the mean background concentration.

2 - Chemical detected less than three times and the detected concentrations were below the sample quantitation limit (SQL), or the chemical was not detected.

Table 2-8. Potential Inorganic Contaminants of Concern in the Ashland 1 Site Surficial Soils (0-2 ft.)

Contaminant	Frequency of Detection	Range of Detected Concentrations (mg/kg)	Mean Detected Concentration (mg/kg)	Mean Background Concentration (mg/kg)
Metals				
aluminum	12 / 12	4480 - 43200	13198.63	13975.56
antimony	2 / 12	8.6 - 9.8	8.18	8.24
arsenic	3 / 12	66.8 - 122	85.82	20.47
barium	12 / 12	43.1 - 230	116.45	112.81
beryllium	6 / 12	0.99 - 6.7	0.86	1.08
boron	9 / 12	20.3 - 52.8	33.31	20.47
cadmium	1 / 12	31.9	30.90	1.03
* calcium	12 / 12	2650 - 181000	66093.33	32876.67
chromium	12 / 12	10.5 - 109	22.98	21.48
cobalt	7 / 12	10 - 39.5	16.51	10.53
* copper	12 / 12	13.5 - 1870	38.70	18.28
iron	12 / 12	20.6 - 28000	16823.38	23700.00
* lead	12 / 12	23.7 - 21100	137.19	36.00
* magnesium	12 / 12	3400 - 79100	15762.28	10421.11
manganese	12 / 12	233 - 2030	803.42	542.56
molybdenum	5 / 12	19.5 - 69.9	27.90	20.47
nickel	12 / 12	12.8 - 96.2	28.00	22.52
potassium	6 / 12	647 - 3000	1584.06	1625.56
selenium	10 / 12	25.3 - 172	98.18	149.16
silver	1 / 12	6.5	5.50	2.91
sodium	3 / 12	1240 - 1750	1481.01	1023.00
thallium	12 / 12	24.5 - 65.4	43.89	48.36
* vanadium	11 / 12	11.9 - 1070	42.61	24.51
* zinc	11 / 12	9.1 - 1060	97.55	84.77

* Contaminant of Concern

Note: Chemicals analytically evaluated and not detected are not shown. See Table 2-7.

Table 2-9. Potential Organic Contaminants of Concern in the Ashland 1 Site Surficial Soils (0-2 ft.)

Contaminant	Frequency of Detection	Range of Detected Concentrations (mg/kg)
Pesticides		
delta-BHC	1 / 12	200
BNAEs		
* 2-methylnaphthalene	3 / 19	110 - 120
acenaphthene	1 / 22	150
anthracene	1 / 22	54
* benzo(a)anthracene	6 / 22	60 - 780
* benzo(a)pyrene	4 / 22	140 - 820
* benzo(b)fluoranthene	4 / 22	150 - 990
* benzo(g,h,i)perylene	3 / 22	200 - 960
* benzo(k)fluoranthene	3 / 22	120 - 920
benzoic acid	1 / 19	700
* bis(2-ethylhexyl)phthalate	6 / 22	110 - 640
* chrysene	6 / 22	83 - 1100
* di-n-butylphthalate	5 / 22	85 - 260
dibenzofuran	1 / 19	92
diethylphthalate	1 / 22	68
* fluoranthene	6 / 22	110 - 1300
indeno(1,2,3-cd)pyrene	1 / 22	210
* naphthalene	4 / 22	50 - 120
* phenanthrene	5 / 22	55 - 760
* pyrene	6 / 22	78 - 1600
VOCs		
* methylene chloride	1 / 31	4.8
* toluene	4 / 31	1.1 - 54

* Contaminants of Concern

Note: Chemicals analytically evaluated and not detected are not shown. See Table 2-7.

Table 2-10. Potential Inorganic Contaminants of Concern in the Ashland 2 Site Surficial Soils (0 - 2 ft.)

Contaminant	Frequency of Detection	Range of Detected Concentrations (mg/kg)	Mean Detected Concentration (mg/kg)	Mean Background Concentration (mg/kg)
Metals				
aluminum	9 / 9	12600 - 45900	19754.19	13975.56
barium	9 / 9	87.9 - 167	126.03	112.81
* beryllium	7 / 9	0.97 - 6.5	0.99	1.08
boron	1 / 9	21.4	21.40	20.47
* cadmium	4 / 9	0.94 - 34.3	7.94	1.03
calcium	9 / 9	3330 - 115000	50631.11	32876.67
chromium	9 / 9	21 - 60.4	29.36	21.48
* cobalt	5 / 9	12.4 - 83.5	24.05	10.53
* copper	9 / 9	14.4 - 1360	86.22	18.28
iron	9 / 9	21700 - 40200	26018.79	23700.00
* lead	9 / 9	33.3 - 354	80.48	36.00
magnesium	9 / 9	5490 - 23200	13912.22	10421.11
manganese	9 / 9	492 - 2480	895.89	542.56
molybdenum	1 / 9	17.2	17.20	20.47
* nickel	9 / 9	21.4 - 134	36.44	22.52
potassium	9 / 9	1600 - 2490	1938.89	1625.56
selenium	8 / 9	75.4 - 239	138.10	149.16
silver	8 / 9	2.4 - 11.8	3.23	2.91
sodium	3 / 9	1780 - 8840	3386.32	1023.00
thallium	8 / 9	26.1 - 68.1	50.06	48.36
* vanadium	9 / 9	25.3 - 748	73.52	24.51
* zinc	9 / 9	82.3 - 1900	193.91	84.77

* Contaminant of Concern

Note: Chemicals analytically evaluated and not detected are not shown. See Table 2-7.

Table 2-11. Potential Organic Contaminants of Concern in the Ashland 2 Site Surficial Soils (0 - 2 ft.)

Contaminant	Frequency of Detection	Range of Detected Concentrations (µg/kg)
BNAEs		
benzo(a)anthracene	2 / 11	350 - 2700
benzo(a)pyrene	1 / 11	3400
bis(2-ethylhexyl)phthalate	2 / 11	240 - 450
* di-n-butylphthalate	3 / 11	76 - 100
* fluoranthene	4 / 11	160 - 4900
* phenanthrene	4 / 11	160 - 4000
* pyrene	4 / 11	160 - 4000
VOCs		
* acetone	1 / 2	170
benzene	1 / 2	3.7
* chlorobenzene	1 / 2	17
* methylene chloride	1 / 2	24
toluene	1 / 2	5.1
* xylenes (total)	1 / 2	44

* Contaminant of Concern

Note: Chemicals analytically evaluated and not detected are not shown. See Table 2-7.

Table 2-12. Potential Inorganic Contaminants of Concern in Tonawanda Surface Water

Contaminant	Frequency of Detection	Range of Detected Concentrations (µg/l)	Mean Detected Concentration (µg/l)	Mean Background Concentration (µg/l)
Metals				
* aluminum	14 / 15	100.0 - 151,000	7,738.66	NA
* antimony	3 / 15	20-103	31.97	NA
* arsenic	1 / 15	50-259	143.93	NA
* barium	5 / 15	100-1640	250.10	NA
* beryllium	1 / 15	2.5-19.2	3.29	NA
* boron	14 / 15	50-18,300	3351.27	NA
* cadmium	1 / 15	2.5-22.6	3.40	NA
* calcium	15 / 15	35,600-903,000	180,667.00	NA
* chromium	4 / 15	5-469	23.70	NA
* cobalt	1 / 15	25-134	30.72	NA
* copper	4 / 15	12.5-964	55.56	NA
* iron	15 / 15	126-222,000	29,633.00	NA
* lead	3 / 15	45-2700	128.95	NA
* magnesium	15 / 15	7,930-237,000	55,235.00	NA
* manganese	15 / 15	40.5-6,620	1676.82	NA
* mercury	1 / 6	0.1-0.53	0.17	NA
* molybdenum	2 / 15	50-217	68.19	NA
* nickel	5 / 15	20-805	57.46	NA
* potassium	9 / 15	2,500-66,900	16,764.00	NA
* selenium	2 / 15	50-914	162.40	NA
* silver	1 / 15	5-17.2	5.71	NA
* sodium	15 / 15	5,380-361,000	80,427.00	NA
* thallium	2 / 15	50-775	198.23	NA
* vanadium	5 / 15	25-1190	76.75	NA
* zinc	14 / 15	10-5380	635.59	NA

* Contaminants of Concern
NA - Data Not Available

Note: Chemicals analytically evaluated and not detected are not shown. See Table 2-14.

Table 2-13. Potential Organic Contaminants of Concern in Tonawanda Surface Water

	Contaminant	Frequency of Detection	Range of Detected Concentrations ug/L
	BNAEs		
	2-methylnaphthalene	2 / 12	1.6 - 5.0
*	2,4-dimethylphenol	1 / 12	12
*	4-methylphenol	2 / 12	5 - 35
	benzo(a)anthracene	1 / 12	4.6
*	benzo(b)fluoranthene	1 / 12	4
	benzo(k)fluoranthene	1 / 12	3.2
	benzo(g,h,i)perylene	1 / 12	3.9
	benzo(a)pyrene	1 / 12	4.3
	benzoic acid	2 / 12	13 - 29
	benzyl alcohol	1 / 12	3
*	bis(2-ethylhexyl)phthalate	6 / 12	1.1 - 82
	chrysene	1 / 12	4.4
	di-n-butylphthalate	6 / 12	1.1 - 3.1
	diethylphthalate	1 / 12	3
*	fluoranthene	1 / 12	12
	fluorene	1 / 12	5
	indeno(1,2,3-cd)pyrene	1 / 12	4.3
	naphthalene	1 / 12	2
	phenanthrene	3 / 12	1 - 6
*	phenol	2 / 12	5 - 33
*	pyrene	2 / 12	1.1 - 11
	VOCs		
	1,1-dichloroethane	1 / 14	1
	1,1,1-trichloroethane	2 / 14	0.5 - 2
*	1,2-dichloroethane	1 / 14	3.5
*	1,2-dichloropropane	1 / 14	1.3
*	2-butanone	2 / 5	5 - 94
	4-methyl-2-pentanone	1 / 5	4
*	acetone	5 / 5	15 - 99
*	benzene	3 / 14	0.5 - 110
*	bromodichloromethane	2 / 14	0.5 - 2.7
*	bromoform	1 / 14	5.2
*	dibromochloromethane	1 / 14	2
	ethylbenzene	2 / 14	1
*	methylene chloride	5 / 14	2 - 29
*	toluene	5 / 14	0.5 - 6
*	trans-1,2-dichloroethene	1 / 9	1.9
*	trans-1,3-dichloropropene	1 / 14	2.2
*	xylenes (total)	3 / 14	0.5 - 16

* Contaminant of Concern

Note: Chemicals analytically evaluated and not detected are not shown. See Table 2-14.

Table 2-14. Potential Organic Contaminants of Concern in Surface Water Eliminated from the Risk Assessment

	Contaminant	Screening Rationale
	BNAEs	
	2-methylnaphthalene	2
*	2,4-dimethylphenol	
*	4-methylphenol	
	benzo(a)anthracene	2
*	benzo(b)fluoranthene	
	benzo(k)fluoranthene	2
	benzo(g,h,i)perylene	2
	benzo(a)pyrene	2
	benzoic acid	2
	benzyl alcohol	2
*	bis(2-ethylhexyl)phthalate	
	chrysene	2
	di-n-butylphthalate	2
	diethylphthalate	2
*	fluoranthene	
	fluorene	2
	indeno(1,2,3-cd)pyrene	2
	naphthalene	2
	phenanthrene	2
*	phenol	
*	pyrene	
	VOCs	
	1,1-dichloroethane	2
	1,1,1-trichloroethane	2
*	1,2-dichloroethane	
*	1,2-dichloropropane	
*	2-butanone	2
	4-methyl-2-pentanone	2
*	acetone	
*	benzene	
*	bromodichloromethane	
*	bromoform	
*	dibromochloromethane	
	ethylbenzene	2
*	methylene chloride	
*	toluene	
*	trans-1,2-dichloroethene	
*	trans-1,3-dichloropropene	
*	xylenes (total)	

* Contaminant of concern

1 – Mean of detected concentrations was less than twice the mean background concentration.

2 – Chemical detected less than three times and detected concentrations were less than sample quantitation limit (SQL) or the chemical was not detected.

3 – Chemical detected at a frequency five percent or less at a sample size of 20 or greater.

Table 2-15. Potential Inorganic Contaminants of Concern in Tonawanda Sediment

	Contaminant	Frequency of Detection	Range of Detected Concentrations (mg/kg)	Mean Detected Concentration (mg/kg)	Mean Background Concentration (mg/kg)
	Metals				
*	aluminium	15 / 15	1590-38,000	13400	3,320
	antimony	4 / 15	3.8-27.7	9.96	22.30
	arsenic	0 / 15	9.50	16.29	36.90
	barium	14 / 15	46.5-349	169.00	151
	beryllium	2 / 15	0.5-1.4	0.91	1.80
	boron	10 / 15	9.9-73.6	37.50	36.90
	cadmium	2 / 15	0.47-2.3	0.96	1.80
*	calcium	15 / 15	30,300-71,700	38160.00	12,900
	chromium	15 / 15	16.3-63.9	31.05	19
	cobalt	3 / 15	4.75-18.2	9.67	18.40
	copper	14 / 15	5.8-124	55.23	42.70
	iron	15 / 15	4,980-65,100	26014.00	26,900
	lead	14 / 15	28.55-211	91.28	90.60
*	magnesium	14 / 15	11,600-20.200	9069.00	2,870
	manganese	15 / 15	83-1,800	828.00	430
	molybdenum	0 / 15	9.5	16.29	36.90
	nickel	14 / 15	9.3-85.5	41.34	28.40
	potassium	5 / 15	476-1,810	1090.00	1840.00
	selenium	15 / 15	56.6-533	201.19	113
	silver	0 / 15	0.95	1.63	3.70
	sodium	0 / 15	476.00	813.98	1840.00
	thallium	14 / 15	23.3-115	51.29	61.10
*	vanadium	14 / 15	11.6-149	52.18	24.6
	zinc	15 / 15	79.4-1,260	301.66	152

* = Contaminant of Concern
NA - Data Not Available

Note: Chemicals analytically evaluated and not detected are not shown. See Table 2-16

Table 2-16. Potential Contaminants of Concern in Sediment Eliminated from the Risk Assessment Ashland 1

Contaminant	Screening Rationale
antimony	1
arsenic	2
barium	1
beryllium	1
boron	1
cadmium	1
chromium	1
cobalt	1
copper	1
iron	1
lead	1
manganese	1
molybdenum	2
nickel	1
potassium	1
selenium	1
silver	2
sodium	2
thallium	1
zinc	1

1 – Mean of detected concentrations was less than twice the mean background concentration.
2 – Chemical detected less than three times and detected concentrations were less than sample quantitation limit (SQL) or the chemical was not detected.
3 – Chemical detected at a frequency five percent or less at a sample size of 20 or greater.

Table 2-17. Summary of Contaminants of Concern (in all media) Retained for Risk Assessment

Contaminant of Concern	Surface Soils			Surface Water	Sediment
	Linde	Ashland 1	Ashland 2	Tonawanda	Tonawanda
Metals:					
aluminum				X	X
antimony				X	
arsenic	X			X	
barium	X			X	
beryllium	X		X	X	
boron	X			X	
cadmium	X		X	X	
calcium	X	X		X	X
chromium	X			X	
cobalt			X	X	
copper	X	X	X	X	
iron				X	
lead	X	X	X	X	
magnesium	X	X		X	X
manganese	X			X	
mercury				X	
molybdenum				X	
nickel	X		X	X	
potassium				X	
selenium				X	
silver				X	
sodium				X	
thallium				X	
uranium	X	X	X	X	X
vanadium		X	X	X	X
zinc		X	X	X	

Table 2-17. (continued)

Contaminant of Concern	Surface Soils			Surface Water	Sediment
	Linde	Ashland 1	Ashland 2	Tonawanda	Tonawanda
BNAEs:					
2-methylnaphthalene	X	X			
4-methylphenol				X	
2,4-dimethylphenol				X	
acenaphthene	X				
anthracene	X				
benzo(a)anthracene	X	X			
benzo(a)pyrene	X	X			
benzo(b)fluoranthene	X	X		X	
benzo(g,h,i)perylene	X	X			
benzo(k)fluoranthene	X	X			
bis(2-ethylhexyl)phthalate	X	X		X	
chrysene	X	X			
di-n-butylphthalate	X	X	X		
dibenzo(a,h)anthracene	X				
dibenzofuran	X				
fluoranthene	X	X	X	X	
fluorene	X				
indenol(1,2,3-cd)pyrene	X				
napthalene	X	X			
phenanthrene	X	X	X		
phenol				X	
pyrene	X	X	X	X	

Table 2-17. (continued)

Contaminant of Concern	Surface Soils			Surface Water	Sediment
	Linde	Ashland 1	Ashland 2	Tonawanda	Tonawanda
VOCs:					
2-butanone				X	
1,2-dichloroethane				X	
1,2-dichloropropane				X	
acetone	X		X	X	
benzene				X	
bromodichloromethane				X	
bromoform				X	
dibromochloromethane				X	
chlorobenzene			X		
methylene chloride	X	X	X	X	
toluene	X	X		X	
trans-1,2-dichloroethene				X	
trans-1,3-dichloropropene				X	
xylenes			X	X	

3. EXPOSURE ASSESSMENT

This section addresses the environmental fate and transport of the COCs identified in Section 2 and the potential pathways by which human populations (e.g., transient visitors and workers) could be exposed to radiological and chemical contaminants at the Tonawanda site. As explained below (Section 3.2), there are no current and future residential exposure pathways. Exposure estimates are provided for the Linde facility, the Ashland 1 and Ashland 2 properties, and the Seaway property. These are the property units used in the RI report (BNI 1992); the properties have been divided further into subareas for risk assessment purposes as described in Chapter 1. Contaminant concentrations were determined by sampling and analysis, radiation survey measurements, and/or modeling. The data are summarized in this section and Appendix C of the BRA and presented as the mean and RME (defined as the 95th percentile upper confidence limit on the mean, UL_{95}) for each of the subareas. In identifying primary pathways of exposure at each location, current and plausible future land uses of the properties and surrounding areas are considered. This section describes exposure scenarios, develops information on exposure pathways, estimates the concentration of the radionuclides and chemical contaminants of potential concern at points of human exposure, and determines receptor intakes (doses). Mean and RME estimates are presented for radiation dose and chemical intakes within each scenario. The uncertainties of the exposure assessment are discussed in Section 5.

3.1 CHARACTERIZATION OF EXPOSURE SETTING

The exposure setting for the Tonawanda site is described briefly in terms of both the natural environment and local land use and demography. The setting is described in more detail in the RI report (BNI 1992). The purpose of the following discussion is to provide information pertinent to the identification of exposure pathways and estimation of exposure rates for receptors who could be exposed to contaminants.

3.1.1 Environmental Setting

Site conditions that may affect fate and transport of contaminants and resultant potential for human exposure include physical surficial features, climate, ecological resources, geology, surface water and groundwater, and soil type, including erosion potential. These site conditions are discussed in the following sections.

3.1.1.1 Topography

The Tonawanda site is located in the eastern lake section of the central lowland physiographic province (Fenneman 1946). The characteristic landscape of this section consists of dissected and glaciated lowlands and escarpments. The specific physical surface features of the Tonawanda properties are described in the following subsections. Information was derived from an October 1991 site visit and the RI (BNI 1992).

Linde

The Linde property is generally flat due to onsite grading of the surface soil. The property contains office buildings, fabrication facilities, warehouse storage areas, material laydown areas, parking lots, and railroad tracks. The property is underlain by a series of utility tunnels that interconnect some of the main buildings and a network of storm and sanitary sewers (Figure 3-1). The parking lot in the northwest corner of the property is paved. Areas around the buildings are mostly paved concrete. Several railroad spurs extend onto the property from the Conrail trackage located outside the site's eastern boundary. The soil in the area of the railroad tracks is hard, packed gravel. The soil along the fence which borders the site boundary is vegetated with native grasses. A blast wall, located east of Building 58 (Figure 1-3), consists of piled soil next to the building, and is held in place with wooden planks. The soil here is vegetated with native grasses.

Contaminated soil was removed from the Building 90 area before the building's construction. The soil was placed in two windrows, one between Buildings 73 and 73B and the east property boundary and the other north of Building 90 along the north property boundary (Figure 1-3). Soil removed from the Building 90 area was also placed in a third pile on the northern portion of the property. The three piles were subsequently consolidated into one uncovered pile west of Building 90. A contaminated pile of waste material was also historically located north of Building 38 (FBDU 1981a).

Stormwater drains into the open-grate storm sewers on the property and flows in underground piping to the west and south (Figure 3-1). Flow to the west discharges to an underground twin cell conduit along the western boundary of the property which carries the flow of Twomile Creek. This twin-cell conduit discharges back to Twomile Creek through gates located on the downstream side of the dam that forms Sheridan Park Lake. Runoff from the extreme southern portion of the site drains into a storm sewer in the center of Woodward Avenue. This storm sewer also joins the Twomile Creek twin-cell conduit. Surface water hydrology is discussed in detail in the RI report (BNI 1992).

Ashland 1

Two large petroleum product storage tanks were formerly located at Ashland 1. The tanks were removed in 1989. Construction of the tanks involved excavation and removal of approximately 4,598 m^3 (6,000 yd^3) of contaminated material. Some remaining contaminated soil was used to build earthen berms which surrounded the storage tanks. The berms still exist at the site and divide the property into thirds. Native grasses, weeds, and shrubs make up the site vegetation. However, the inner bermed areas and the inner area of the northern part of the site are mostly bare soil.

A dirt access road runs along the eastern and western property boundaries. The road crosses the southern portion of the site and curves northward along the east boundary. Small pipes under the east access road allow surface drainage from within the bermed area to drain

into the main ditch along the east property boundary (Figure 3-2). This ditch, which is overgrown with cattails and weeds, flows north to an opening on the adjacent Seaway property where an underground concrete conduit carries the flow beneath Seaway to Ashland 2.

An electrical substation is located in the southwestern corner of the property. This small building and land surrounding it are fenced with an approximately six-foot high chain link fence. A gas company receiving and metering station is located in the southeast corner of the property. The station has had no other uses. The area around these buildings is vegetated with native grasses.

Ashland 2

Ashland 2 is a large tract of land separated from the Seaway property by a strip of land owned by the Niagara Mohawk Power Corporation (Figure 1-4). The property is vegetated with native grasses and shrubs, and the surface topography is crosscut with several drainage pathways including federally-designated wetlands. Drainage is received from Ashland 1 via the underground Seaway culvert which exits Seaway at the Niagara Mohawk property and flows across the Niagara Mohawk property to Ashland 2 (Figure 1-4).

Areas of the Ashland 2 property were used in the past as an industrial landfill by the Ashland Oil Company (BNI 1992).

Seaway

The original surface topography at the Seaway site has been significantly altered due to the extensive landfill operations conducted there. The site is terraced with steep slopes extending to a height of approximately 36 m (120 ft) above the surrounding area. Areas where the radiologically contaminated residues were disposed have been covered with refuse and fill material up to a height of 12 m (40 ft). The site is vegetated with native grasses.

A 1-m (3-ft) diameter reinforced concrete pipe transects the property and passes beneath the landfill (Figure 1-4). The pipe carries stormwater from the ditch at Ashland 1 to Ashland 2. The condition of the pipe is not known (BNI 1992).

3.1.1.2 Climate

The climate of New York is generally the humid, continental type that prevails in the northeastern United States. The monthly normal temperature range for the Tonawanda area (Buffalo, New York) is -4.4 to 21.7°C (24 to 71°F), with a mean annual temperature of 8.9°C (48°F). Mean annual precipitation is 96 cm (37.5 in.) and is fairly evenly distributed throughout the year. Winds in the area blow predominantly from the southwest or west-southwest, across Lake Erie (FBDU 1981b). The average monthly wind speed ranges from 15.9 to 23 km/h (9.9 to 14.3 mph), with an annual average wind speed of 19 km/h (12 mph). These data, from the

Buffalo International Airport located about 14 km (8 mi) southeast of the site, are considered representative of the project area.

3.1.1.3 Ecological Resources

Linde, Ashland 1, Ashland 2, and Seaway lie between the Northern Hardwoods Forest section of the Laurentian Mixed Forest and the Beech-Maple Forest section of the Eastern Deciduous Forest (BNI 1992). The typical native trees that inhabit the area are aspen, fire cherry, hawthorn, maple, and beech. Although hemlock and white pine trees were once quite abundant on these sites, they have been eliminated from much of the area. Oak trees are also quite common to the area. Most of the natural vegetation remains only in small woodlands or undrained areas (Galvin 1979). There is little or no actual forest habitat present in the immediate vicinity of the four Tonawanda properties.

The official wetlands map for Erie County shows four wetland areas in the Tonawanda area that are under the jurisdiction of New York State Department of Environmental Conservation (NYSDEC); none of these areas are associated with any of the subject properties (BNI 1988a). However, the federal wetlands map shows one wetland on Ashland 2, one along the northeast boundary of Linde, and one on Linde where a stream previously existed (BNI 1992).

Sheridan Park Lake, an urban fishery in the Tonawanda area that is stocked annually by NYSDEC, receives approximately 2,000 adult calico bass. The fish are harvested each year from Chautaugua Lake (Erie and Niagara Counties Regional Planning Board 1978; BNI 1988b).

The only federally-listed species that could come into contact with the Tonawanda site are the bald eagle (*Haliaeetus leucocephalus*) and the osprey (*Pandion haliaetus*). However, it is expected they pass over the area only as occasional transients (Gill 1989). There are no habitats critical for their survival in the vicinity (DeGraaf and Rudis 1988).

Linde

There are no known rare or endangered plant or animal species on or near Linde (BNI 1992). There is, however, an undeveloped area north of the property that consists of wetland areas that contain a variety of plant and animal species. This property is federally classified as a wetland. There were some trees placed on the Linde property during landscaping efforts. These include eastern cottonwood (*Populus deltoides*), American sycamore, white ash, northern red oak, and shagbark hickory. The natural plant succession and the original habitat have been disrupted or destroyed due to the fact that Linde and its vicinity are part of an industrial complex. Years of continuous industrial activity have left only marginal plant communities (FBDU 1981a). The property supports no fixed wildlife habitat. The animal life present is best described as transitory and typical of an urban industrial setting (BNI 1992). Only the cosmopolitan species of birds (i.e., pigeons and sparrows) and small mammals are seen. Some

rodents such as squirrels and mice may be present, especially around the periphery of the plant area (FBDU 1981a).

Ashland 1

Vegetation on Ashland 1 is scarce, as a result of industrial activity, and the only flora found on the site are shrubs and grasses. Wildlife activity is limited to cosmopolitan species that are typical in an urban industrial setting: pigeon, mourning dove, killdeer, European starling, common grackle, American robin, house mouse, Norway rat (*Rattius norvegicus*), eastern cottontail rabbit, and eastern gray squirrel (BNI 1992). There are no known federally- or state-listed threatened or endangered species found on or near the site. However, osprey (*Pandion haliaetus*) and bald eagles are occasionally seen. There are no documented nesting areas on or near the property, which has no features that tend to attract either of these species or support their habitats (FBDU 1981b).

Ashland 2

Ashland 2 is generally considered a brushland, with large marshy areas (wetlands) transecting the area. Much of the area is covered with a mixture of grasses, forbs, shrubs, and smaller trees and varies from areas with essentially no vegetation to areas with fairly dense stands of woody shrubs and trees (BNI 1992).

Because fewer available habitats are disturbed and/or landscaped on Ashland 2 than on either Linde or Ashland 1, the area may be expected to support a relatively diverse population of animals, including those cosmopolitan species described for Ashland 1 as well as a number of waterfowl species, fox, raccoon (*Procyon lotor*), skunk (*Mustelidae* spp.), opossum (*Didelphis marsupialis*), and deer (*Cervidae* spp.)(BNI 1992). Similarly to Ashland 1, bald eagles and osprey are known to migrate through the area, but the property does not contain nesting habitats that would attract these protected species (FBDU 1981b).

Seaway

Seaway consists of sparse vegetation, primarily shrubs and grasses. This is due to the activities associated with the landfill operations. Other vegetation at the site includes field daisies, milkweeds (*Asclepias* spp.), vetch (*Vicia* spp.), foxtail grasses (*Setaria* spp.), yellow and red clovers (*Melilotus officanalis, Trifolium pratense*), dock sorrels (*Rumex* spp.), and cattails (*Typha* spp.) (BNI 1992). The site's present condition, location, or operation have not impacted the current floral species. Any potential impact from inadequate vegetation would be from erosion (BNI 1992).

Due to the landfill operations and the industrial activities at Seaway and its vicinity, the wildlife activity is restricted. Natural wildlife habitats have observably been altered or eliminated. Seagulls flock to the site to scavenge waste. Transitory faunal species that may occupy the site include seagulls, crows, and rodents, especially rats and mice (FBDU 1981b).

There are no known federally- or state-listed endangered, threatened, or sensitive animal or plant species found on or near the Seaway landfill.

3.1.1.4 Geology

The regional geology of the Tonawanda area can be described as a series of marine sedimentary rocks separated by infrequent nondepositional and erosional unconformities. These rocks are bounded below by crystalline basement and above by unconsolidated glacial and alluvial sediments. A southward dipping monocline is the only tectonic feature in the area (BNI 1992).

All of the Tonawanda properties are in the Niagara Frontier area of the Central Lowlands Physiographic Province. The ground surface elevation is 180 m (590 ft) to 183 m (600 ft) at all sites. The elevation varies less than 3 m (10 ft) at any property (BNI 1992).

3.1.1.5 Surface Water and Groundwater

This section provides an overview of surface water and groundwater hydrology at the Tonawanda properties. Additional details are provided in the RI report (BNI 1992).

Receiving Waters

The Niagara River is the major receiving water to which the Tonawanda properties drain via Rattlesnake Creek and Twomile Creek (Figure 3-3). The Tonawanda site is located along the upper reach of the river, specifically along the Tonawanda Channel.

Rattlesnake Creek is a natural channel formed from surface drainage received from Ashland 1 and Seaway. Drainage from Ashland 1 travels under the Seaway property through an underground concrete conduit and exits at the Niagara Mohawk property line. Rattlesnake Creek receives this drainage, crosses the Niagara Mohawk property, and then the Ashland 2 property. The 2,320-m (7,600-ft) channel drains 139 ha (340 acres) before joining Twomile Creek (Figure 3-4). Twomile Creek flows into the Niagara River approximately 305 m (1,000 ft) downstream of the confluence with Rattlesnake Creek (BNI 1992). The Rattlesnake Creek channel is approximately 3 m (10 ft) wide and 1 m (3 ft) deep at bank-full capacity, and it has a 1.1 percent slope on the Ashland 2 property. The channel and creek floodplain are vegetated with a thick growth of cattails and bulrushes, which limits flow velocities. The floodplain is approximately 30 m (100 ft) wide on Ashland 2. Three small drainage ditches join Rattlesnake Creek after it crosses Ashland 2. The creek then travels approximately 975 m (3,200 ft) before its confluence with Twomile Creek (BNI 1992).

Twomile Creek originates south of the Linde property in a natural channel (Figure 3-3). The creek flow consists of groundwater discharge and stormwater runoff. The creek enters a two-channel underground culvert and flows north, where the two pipes empty into two 2.7 m x 2.1 m (9 ft x 7 ft) box culvert conduits which run side-by-side toward the north. These

conduits serve as the outlet to the municipal storm sewer draining the eastern half of the Town of Tonawanda and the Village of Kenmore. Runoff from Linde enters the conduits before they discharge to Twomile Creek through two large flow-control gates on the downstream face of the concrete dam impounding Sheridan Park Lake. The gates are pressure operated, releasing storm flow when necessary. When enough stormwater backs up and the gates are opened, the onslaught of water flushes out accumulated sediments in the conduits. Sediments are then deposited in the natural stream channel downstream.

Twomile Creek and its tributaries are classified by NYSDEC as Class B: "primary contact recreation and any other uses, except as a source of water supply for drinking, culinary, or food processing purposes." Class B waters are protected under the New York State Environmental Conservation Law, Article 15; thus, certain activities in the waters or along the banks require state permits (BNI 1988b).

Linde Surface Drainage

Site runoff at Linde collects in the facility's storm sewer system which drains through seven outfalls to the underground conduits carrying Twomile Creek (Figure 3-1). Outfalls 1 and 2 drain stormwater runoff from the southern end of the site. Outfall 3 carries runoff from a small area in front of the main office building. The fourth outfall drains the middle portion of the property, including runoff from the Building 14 area where several injection wells were historically located. Outfall 5 collects runoff from a very small area in the western part of the site. Outfall 6 receives runoff from most of the northern portion of the site, including drainage from the areas around Buildings 30, 31, 38, and 58. Shallow groundwater from agricultural tile beneath the gravel-packed parking areas is also collected by Outfall 6. The seventh outfall collects runoff from the extreme northern section of Linde, including the Building 90 area. This drainage area also includes some underground agricultural tiles for shallow groundwater collection. The surface runoff from the northwest corner of the plant area is collected by a ditch located just outside the Linde fence and conveyed by a 76-cm (30-in.) culvert to the Twomile Creek twin conduit.

Erosion is not evident at the Linde site. Most of the surface area is paved and covered by buildings. Erosion at Linde and the other three Tonawanda properties was evaluated in the RI Report for the Tonawanda site (BNI 1992).

Ashland 1 Surface Drainage

The Ashland 1 site is flat except where berms were created to surround storage tanks historically located on the property. An approximately three-acre area is enclosed by the berms. Water from precipitation collects within the bermed area and infiltrates into the soil, evaporates, or flows to the east drainage ditch via small pipes which extend through the berm and under the access road to the ditch. The portion of the site southeast of the bermed area is flat and covered with grass except for the dirt access road and electrical substation area. Drainage from this area

is directed toward the ditch running along the east boundary, between Ashland 1 and Seaway (Figure 3-3).

The western section of Ashland 1 is low-lying and vegetated with tall grass and bushes. Runoff from this area flows into the main ditch along the Seaway boundary via a small ditch running west which flows through a 30-cm (12-in.) steel pipe and then into the main ditch (Figure 3-3). The main ditch flows northwest into a low marshy area where the 1-m (3-ft) underground conduit opening exists that carries Ashland 1 drainage under Seaway.

Ashland 2 Surface Drainage

Storm runoff leaves the Ashland 2 property by drainage channels that exist on the property (Figure 3-3). The southeastern portion of the property drains to a small 1-m (3-ft) wide ditch running northeast toward Twomile Creek. The ditch carries surface drainage from nearly 38 percent of the property's total area (BNI 1992). It travels under Twomile Creek Road through a 76-cm (30-in.) culvert and empties into Twomile Creek approximately 6 m (20 ft) below the Fletcher Street bridge over Twomile Creek (BNI 1992).

Rattlesnake Creek is the main channel which drains Ashland 2. Approximately 59 percent of Ashland 2 overland runoff empties into Rattlesnake Creek (BNI 1992). The Ashland 1 drainage, which is carried under Seaway and exits Seaway at the Niagara Mohawk property, makes up part of the Rattlesnake Creek flow. A second channel which drains the western portion of the property joins Rattlesnake Creek just across the Benson Development Company property line (Figure 3-4). Runoff from Seaway is collected in this channel. Two other ditches draining the northern and southern sides of the property's access road flow into this ditch before it empties into Rattlesnake Creek. Two separate channels drain small areas in the extreme western portion of the property: one on the north side of the access road, and one on the southern side. These channels are directed under River Road and empty into the Niagara River.

The Ashland 2 property is covered with grass and thick bushes which act to impede surface erosion. Soils were disturbed in the past when the Linde residues were disposed there, during operation of the Ashland Oil Company industrial landfill and during construction of a large berm that surrounded a petroleum storage tank in the southeast corner of the property. Some erosion likely occurred when soils were disturbed. Present erosion is limited due to the thick ground cover.

Seaway Surface Drainage

The Seaway property (Figure 3-4) consists of a long, narrow rectangular landfill pile with side slopes of approximately 30 percent (BNI 1992). The ridge of the pile is at the center of the property, resulting in half the surface runoff flowing southwest toward the Ashland refinery property and half flowing northeast onto Ashland 2. Runoff to the southwest is directed to the drainage ditch along the Seaway/Ashland 1 boundary. Most runoff from the northeastern slope

is directed onto Ashland 2 as overland flow into the existing channels at Ashland 2. The southeast runoff enters the small drainage ditch in the southeast portion of Ashland 2 that eventually empties into Twomile Creek. The middle portion of Seaway drains into Rattlesnake Creek. The northwestern area, which includes the area where residues were deposited, drains to the drainage ditch on the southern side of the Ashland 2 access road that is directed under River Road and empties into the Niagara River (Figure 3-4).

Engineering controls are implemented in the landfill design to prevent surface erosion of the landfill property at Seaway. These include seeding with native grasses and terracing of the steep slopes. The areas where residues were deposited are vegetated with thick grass.

Groundwater

Groundwater in the Tonawanda area is considered to be in three general zones. The first zone is the unconsolidated glacial till and glaciolacustrine clay. The second is a series of soluble limestones and dolostones that surround the third, the Camillus Shale. The Camillus Shale is considered to be the most important unit and is defined as a single aquifer because of its extremely high conductivity (BNI 1992).

Groundwater at each of the Tonawanda properties exists in perched zones within the glacial overburden and fill, in a shallow water table within the glacial material, and in the bedrock and its upper surface. The glacial material is considered to be an aquitard and produces low yields (BNI 1992).

The bedrock directly below the Tonawanda site is the Camillus Shale. It is the most significant aquifer in the area. The shale and its contact with the unconsolidated material produce high yields of water, but are confined from the surface by the glacial overburden. This water is poor quality and not considered potable (BNI 1992).

3.1.1.6 Soils

At the Linde property, the natural soils appear to be covered with a fill layer ranging in thickness from 0 to 5.1 m (0 to 17 ft). This fill, as noted in borehole logs, contains substantial quantities of slag and fly ash that were apparently brought onsite from local sources for grading purposes (BNI 1992). Approximately half of the Linde plant area is covered by impervious surfaces such as buildings, paved areas, and sidewalks. Because Linde is an industrial site with a significant portion of the surface area paved and covered by buildings, little erosion is evident (BNI 1992).

The surface soils at Ashland 2 are mainly silt loam (BNI 1992). Although the soils at Ashland 1 are classed as "urban land," they are assumed to be similar to soils at Ashland 2 (SCS 1986).

The Seaway property is an active landfill. The surface of the landfilled area is steep. Most of the surface is bare and both sheet and rill erosion occur on the slopes. Grass covers the base of the pile and the areas that are already capped (BNI 1992).

The estimated soil loss due to erosion from radioactively contaminated areas of the Tonawanda site is (BNI 1992):

Property	Soil Loss (ton/yr)
Ashland 1	0.002
Seaway	0.06
Ashland 2	0.0005
Linde	0.02

3.1.2 Land Use and Demography

The following sections describe the current and potential land use and population distribution in the site vicinity to provide a basis for estimating potential exposure scenarios.

3.1.2.1 Current and Future Land Use

The four properties comprising the Tonawanda site are located in the Town of Tonawanda, Erie County, New York. The properties are situated in a mostly industrial area. The properties lie in areas zoned "Performance Standards Use" (P-S), according to the Town of Tonawanda zoning map (Town of Tonawanda 1990). The Tonawanda Town Code defines the purpose of the Performance Standards Use District as follows: "The purpose of this district is to encourage and allow the most appropriate use of the land available now as well as approaching future commercial and industrial uses unhampered by restrictive categorizing, thus extending the desirability of flexible zoning, subject to change with changing condition."

Linde

Present land use at the Linde site is strictly industrial. The Linde Gas Products Company, Inc. operates an industrial gas production facility on the property. The west side of the site, where the main office building is located, includes a portion of the former Sheridan Park Golf Course, which Linde purchased from the Town of Tonawanda.

The area near the Linde site is used for a mixture of industrial, commercial, recreational, public, and residential purposes. There is a public park west of the site which Linde owns, and beyond the park is a residential area. An elementary school is located at the southern end of the park and beyond that are buildings associated with the local recreation and highway departments.

The areas east and north of the site across Military Road and Sheridan Drive, respectively, are also residential. The Kenmore Sister of Mercy Hospital is approximately one-half mile from the site. In summary, there are six schools, a hospital, two recreational areas, two community buildings, and a senior citizens center within one mile of the Linde site. Future land use of the Linde property will likely remain industrial.

Ashland 1, Ashland 2, and Seaway

The Ashland properties and Seaway property are located in an industrial area along River Road. The Ashland 1 property is part of the defunct Ashland Oil Refinery. The property is not used by Ashland except for temporary storage of drums containing unknown materials. The adjacent refinery is inactive except for present dismantling of refinery equipment.

The Seaway property is an operating sanitary landfill. Municipal, industrial, and construction solid wastes are currently disposed of in the landfill. Hazardous wastes, liquids, sewage sludges, insecticides, whole tires, trees, and explosives are excluded from disposal at the Seaway landfill (FBDU 1981b). The site has been used to dispose of waste since 1930.

Ashland 2 is an open area and is not used for any purpose at the present time. Past use includes an industrial landfill which the Ashland Oil Company utilized on the property, Linde residue disposal, and petroleum storage in a bermed tank formerly located in the southwest corner of the property. Trespassers have been sighted in the past at Ashland 2, using the site for recreational purposes such as driving all terrain vehicles (ATVs).

The area in the vicinity of the Ashland and Seaway properties is used for a mixture of industrial, commercial, public, and residential purposes (FBDU 1981b; site visit 1991). Open brushland lying northeast of the property is posted as being for sale for commercial purposes. However, the property contains potential wetlands which would limit its use. A waterfront park runs along the Niagara River within one quarter mile of the properties and marina/boat launching areas are within three fourths of a mile of the properties. The border of the Town of Tonawanda, the only residential area near the property, is approximately one-half mile east of Ashland 2. Homes are situated along the east side of Twomile Creek, east of Twomile Creek Road. A small public park and one residence are located on the west side of the road. The Town of Tonawanda Sewage Treatment Plant is on Twomile Creek Road within one mile of the properties. Homes are also located across the Niagara River on Grand Island within three fourths of a mile of the properties.

Future land use in the area of the Seaway and Ashland properties will likely remain commercial/industrial based on the present zoning. However, a rewrite draft master plan for the area indicates potential for additional commercial, residential, and recreational development in the vicinity of these properties (Town of Tonawanda, 1992).

3.1.2.2 Relative Locations of Population with Respect to the Site

The Town of Tonawanda includes the Village of Kenmore. Both communities cover a total of 5,128 ha (19.8 mi^2). The Town of Tonawanda has a population of 91,270; while Erie County, on the whole, has a population of nearly 970,000 (U.S. Department of Commerce 1991). Erie County includes the City of Buffalo and encompasses 274,021.8 ha (1,058 mi^2). The projected populations for 1995 and 2005 in Erie County are 985,770 and 986,600, respectively.

Linde

The FBDU report estimated that 18,669 people live within one mile of the property, and 2,631 live within 0.4 km (0.25 mi) of Linde (FBDU 1981a). Of the total living within a one mile radius of the property, 67 percent are Town of Tonawanda residents, 31 percent are Kenmore residents to the southeast, and one percent are Buffalo residents living to the south-southwest.

Approximately 1,700 people are employed on the Linde property (FBDU 1981a). Current work activity within specific buildings could not be determined. Approximately 16,830 people work within one mile of the property (FBDU 1981a).

Ashland 1, Ashland 2, and Seaway

The Ashland properties are included in the Seaway demographics discussion due to the lack of data on the properties and the close proximity of both properties to Seaway. It is estimated that 1,282 people live within 1.6 km (1 mi) of the Seaway property. There are no residents within 0.8 km (0.5 mi) of Seaway. The closest residents live at Grand Island, across the Niagara River. Two hundred of these live between one half and three fourths of a mile of the property (FBDU 1981b). Of the total population residing within one mile of the property, only 3.5 percent are Tonawanda residents, mainly to the southwest of the property. Thirty-seven percent are Grand Island residents living in the northwest quadrant, and 59.5 percent are residents of the Town of Tonawanda to the east of the property. Approximately 7,150 people work within one mile of the Ashland and Seaway properties (FBDU 1981b).

3.2 EXPOSURE SCENARIO DESCRIPTIONS AND ASSUMPTIONS

In this BRA, two time-sequenced exposure scenarios are considered:

- current use - land use remains as it is now, and
- future use - land use in some areas changes to a reasonable maximum condition, based on estimates of future land use, and annual radiological exposure is calculated based on the one-year period in which maximum exposure occurs.

3.2.1 Current Use Scenarios

Receptors at Linde consist of employees. Other potential receptors include transients, who may be visitors, customers, trespassers, or temporary or contractor personnel. Because the exposure frequency and duration for a transient at Linde is assumed to be a small fraction of the frequency and duration for an employee, the transient scenario is not considered further. An excavation worker scenario was evaluated during screening and was found to have lower risk than was calculated for employees because of the shorter exposure duration and therefore is also not considered further. The employee receptor will provide the most conservative estimate of dose and intake at Linde. Employees are assumed not to consume water from the site, because the groundwater aquifer is non-potable.

Ashland 1 is a fenced property with no active use. The only current receptors are transient adults who would visit the site for business purposes. Adult transients are not assumed to come in contact with surface waters and sediments at the site.

Ashland 2 is a partially fenced property with no active use. The property is near a residential area, and trespassers have been sighted in contaminated areas. The current use scenario assumes that access is gained to the area by an older child by foot, bypassing the partial fence and locked gate at the entrance. This older child is assumed to play frequently at the site during the summer and occasionally during the school year. An additional scenario describing an older child wading and being exposed to surface water and sediment in an area such as the confluence of Rattlesnake Creek and Twomile Creek is included.

Seaway Industrial Park is an operating sanitary landfill which is entirely fenced and which has controlled access. Contaminated areas are either buried deep in the landfill with no pathway to potential receptors or are access restricted. Current use receptors are assumed to be transients at the property for business purposes.

3.2.2 Future Use Scenarios

Linde is an operating industrial facility and is expected to remain so in the future. Employees are considered as the receptor for future exposure at the Linde property.

Ashland 1 and Ashland 2 are considered to change from inactive industrial facilities to ones which are actively operating, and employees are considered as the receptors for exposure at those sites.

Although Seaway is an active landfill which has been operating for over 40 years, this area is included in plans for a future waterfront park (Town of Tonawanda 1992). Therefore, the future receptor is conservatively assumed to be an older child transient who plays frequently at the site.

3.3 IDENTIFICATION OF EXPOSURE PATHWAYS

A complete exposure pathway consists of at least the following four elements: (1) a source and mechanism of contaminant release to the environment (with receiving media); (2) environmental transport medium (fate and transport) for the released contaminants; (3) a point of human contact with the contaminated medium (exposure point); and (4) a route of human receptor exposure (exposure route) at the exposure point. An integration of sources, releases, fate and transport mechanisms, exposure points, and exposure routes are evaluated for complete exposure pathways. If any of these elements is missing, the pathway is incomplete and will not be considered further in the risk evaluation.

Conceptual site models were developed for Linde, Ashland 1, Ashland 2, and Seaway to illustrate the potential exposure pathways. Figures 3-5 and 3-6 are schematic diagrams depicting the pathways. In the conceptual site model diagrams, completed exposure pathways are indicated by shaded blocks. A completed pathway exists when a receptor potentially could be exposed to a contaminated source by one of the exposure routes. Incomplete pathways occur when any of the pathway components are missing or when features such as engineering controls or access restrictions are in place to prevent release and migration of, or contact with, contaminants. Blocks with no shading indicate incomplete pathways.

3.3.1 Contaminant Sources and Release Mechanisms

The principal contaminant source is contaminated soils. A smaller amount of contamination is present in surface water and sediment. Groundwater is not considered a contaminant source of concern in this BRA, because due to high dissolved solids, sulfates, and chloride levels it is not of drinking water quality. Release mechanisms include human activity that can mobilize contaminants or incur direct contact with contaminants, external gamma irradiation from contaminated soil and materials, emanation of radon and/or volatilization of chemicals into the atmosphere, wind dispersal of fugitive dust, erosion, surface runoff over contaminated soil following precipitation, leaching from contaminated surface and subsurface soil to groundwater, transport from contaminated groundwater to surface water or sediment, and plant or animal uptake.

3.3.1.1 Radiological Contaminant Sources

The primary source for radiological contaminants at the Tonawanda properties is soil which was contaminated from uranium ore processing activities at Linde from 1942 to 1946 and disposed at Linde and Ashland 1. Periodic disturbances of contaminated soil occurred at Ashland 1 which further distributed the soil contamination to Seaway and Ashland 2. Liquid effluent releases also occurred at the Linde property, which contaminated subsurface soils in the area of the injection wells.

3.3.1.2 Chemical Contaminant Sources

The chemical contaminant source areas are soils at Linde and Ashland 1 which were contaminated due to disposal of chemicals used in MED-related activities. These contaminants were distributed to Ashland 2 and Seaway with the movement of contaminated soil. Additional sources of chemical contaminated soils include disposal of industrial wastes at Ashland 2 which are not MED-related (BNI 1992).

3.3.1.3 Release Mechanisms For Radiological and Chemical Contaminants

Contamination may be released from contaminant sources in a number of ways, including:

- human activity such as excavation of soils, repair or demolition of structures, and management of stored waste;
- emission of radioactive gases or chemical vapors that escape the soil into the airborne environment where they or their progeny are inhaled by humans or deposited on the soil surface, plants, or structures;
- fugitive dust resulting from resuspension of particulate material from soil surfaces, where it is inhaled by humans or deposited on the soil surface, plants, or structures;
- erosion and surface runoff, which may carry contaminants to sedimentation points or to surface water or groundwater;
- leaching of material from subsurface and surface soil, which may transfer contaminants to the groundwater; and
- contaminant uptake from soil by plants growing in contaminated soil, with subsequent ingestion.

3.3.2 Fate and Transport Mechanisms

Following release from sources, contaminants may migrate in environmental media through several transport mechanisms. However, because of the site-specific factors, certain potential release mechanisms and receiving media do not play a significant role in contaminant fate and transport and resulting human exposure at the Tonawanda site. For example, because the groundwater aquifer is not of drinking water quality, migration of contaminants through groundwater is not considered an important release mechanism. Similarly, due to the urban and industrial nature of the site, uptake of contaminants by biota is not currently an important release mechanism.

The environmental pathways considered most important for potential human exposures to site contaminants under current conditions include:

- external gamma radiation from radioactively contaminated soils and materials,
- emanation of radon gas from radium-contaminated soils and groundwater and volatilization of chemicals from contaminated soils,
- resuspension and airborne dispersal of particulates,
- direct contact with contaminated soil and materials, and
- direct contact with contaminated surface water and sediment.

3.3.3 Exposure Points and Exposure Routes

In the assessment of human health risk, exposure points are locations where human receptors can come in contact with contaminants. Exposure route refers to the process by which the human receptor comes in contact with the contaminant at the exposure point. The exposure routes that exist at the Tonawanda site are:

- dermal contact occurring when contaminated soils, sediments, structural materials, or stored waste are handled, or when contaminated surface water is contacted;
- inhalation of radon and radon progeny or resuspended particulates containing radiological or chemical COCs;
- direct ingestion of soils;
- ingestion of surface water or sediment; and
- direct gamma radiation from soils and contaminated structures.

A quantitative assessment of dermal absorption of all contaminants from the Tonawanda soil was not performed because of the uncertainties in toxicological data for assessing dermal absorption for the Tonawanda site soil, surface water, and sediment COCs. EPA's *Dermal Exposure Assessment: Principals and Applications* (EPA 1992a) states that "very little chemical-specific data are available, especially for soils, and the predictive techniques have not been well validated." The report does identify experimentally derived values of percutaneous absorption from soil for nine chemicals, only one of them a metal (cadmium). Because of the uncertainties in assessing dermal absorption of the COCs from soil, a quantitative assessment of this exposure route is only performed for cadmium.

3.3.4 Exposure Pathways

Conceptual site models of potential radiological or chemical exposure pathways were developed for the Tonawanda properties and are shown in Figures 3-5 and 3-6. Inhalation of contaminated particulates and incidental soil ingestion are complete pathways for all receptors and scenarios. Inhalation of radon and direct radiation are complete in radiological exposure pathways. Pathways involving inhalation of radionuclide particulates for employees and residents at nearby properties were evaluated as part of population dose. Potential dermal exposure is a complete chemical exposure pathway, but is only quantitatively evaluated for cadmium in this risk assessment because of inadequacies in available methodologies and chemical-specific absorption data for the other COCs.

Complete groundwater pathways do not occur in current or future scenarios because groundwater is not potable. Employees at the properties are assumed to not have contact with the surface water and sediment because there is minimal access to those media in work areas. Ingestion of surface water and sediment by a transient older child wading and playing in the confluence of Rattlesnake and Twomile Creeks is considered a complete pathway.

3.4 EXPOSURE POINT CONCENTRATIONS

Statistical analysis was performed on the radiological data sets by subareas, and the chemical data sets by soil horizon, to determine the normality of the data set. If the analyte passed the Shapiro-Wilk (W) statistical test for normalcy (SAS 1990), the untransformed data were used to calculate the mean and UL_{95} concentrations. Otherwise the measured results were assumed to be log normally distributed and were transformed before use.

Exposure point concentrations of contaminants must be determined for quantitative health risk assessment. This may be accomplished by analyzing samples collected from locations where human receptors may come in contact with the contaminants or by onsite measurements with radiation detection instruments. When laboratory analysis or onsite measurement data are not available, exposure point concentrations may be estimated using a variety of modeling techniques.

Because of the uncertainty associated with any estimate of exposure concentration, the upper confidence limit on the arithmetic average (i.e, UL_{95}) is used as the RME exposure point concentration (EPA 1989a). This concentration does not necessarily reflect the maximum concentration that could be contacted at any one time, but it is regarded as a reasonable estimate of the maximum concentration likely to be contacted over time (EPA 1989a). In cases where the UL_{95} exceeds the maximum measured concentration, the maximum measured concentration is used as a proxy concentration for the RME estimate in accordance with EPA guidance (EPA 1989a).

Average scenarios are computed using mean values of exposure point concentrations and average values for assumptions. The RME scenarios are computed using upper 95-percent confidence level values for exposure point concentrations and a combination of average and reasonable maximum values for assumptions (see Appendix B).

Radiological Data

For the Tonawanda site, laboratory analysis data are available for the radionuclides Th-232, Th-230, Ra-226, and U-238 in surface and subsurface soil samples in most of the areas. Soil radiological data were used to estimate exposure point concentrations for the following pathways: incidental ingestion of soil, inhalation of radon, inhalation of particulates, and direct gamma exposure. Radionuclide concentrations in surface water and sediment from all sites were aggregated and used to calculate ingestion by an older child playing in a hypothetical area of the creek.

Chemical Data

Chemical intake estimates are based on EPA methodology presented in RAGS (EPA 1989a) and related guidance (EPA 1991d). Estimated chemical-specific intakes for each exposure pathway being quantitatively evaluated in this BRA are presented in Appendix C.

Intakes were calculated for soil ingestion and inhalation of soil particulates for current and future employees at Linde, future employees at Ashland 1 and 2, current transients at Ashland 1 and 2, and current and future transients at Seaway. Surface water and sediment intakes were estimated for future transients (children) playing in a hypothetical portion of the confluence of Rattlesnake Creek and Twomile Creek. As stated earlier, receptors have no access to surface water and sediment at Linde and Seaway. Section 3.2 discusses exposure pathways selected for quantitative assessment.

3.4.1 Soil Analysis and Calculated Contaminant Concentrations

Radiological Data

Radiological data indicate that contamination with Th-230, Th-232, U-238, Ra-226 is widespread in soil at most site properties. Available characterization results and information about the uranium extraction process and the characteristics of the uranium ore processed were used in conjunction with results from the source term analysis to estimate soil exposure point concentrations of all radionuclides in these decay series.

Radionuclide concentrations in surface and subsurface soils are presented in Tables 3-1A through 3-1D. Surface soils are generally defined here as the initial 0.6 m (2 ft) from the surface, whereas subsurface soils are generally greater than 0.6 m (2 ft) in depth. Depths are based on the need to ensure an adequate number of samples in surface and subsurface soil for meaningful statistical analyses. To estimate radionuclide concentrations for each area, the

arithmetic mean and the UL_{95} values for the radionuclide concentrations at each area were calculated (i.e., Ra-226, Th-232, and U-238). Sample results reported as below the sample quantitation limit (nondetects) were considered in this analysis as present at the quantitation limit as a proxy concentration (EPA 1989a). Because it is assumed that radionuclides are present in secular equilibrium, values were derived from the concentrations of these measured radionuclides for all other radionuclides in the decay series of interest.

Chemical Data

Chemical concentrations in soils at the Tonawanda sites are presented in Appendix C. The arithmetic mean concentration and the UL_{95} concentration value for each data set are used as the soil exposure point concentrations to calculate average and RME intakes, respectively. Sample results reported as below the sample quantitation limit (nondetects) were included in this analysis at one-half the quantitation limit as a proxy concentration (EPA 1989a).

3.4.1.1 Exposure Point Concentrations for Incidental Soil Ingestion

Radionuclide concentrations and chemical concentrations in surface soils were used to calculate radiation doses and chemical intakes from incidental soil ingestion. This soil depth assumes limited intrusion (up to 2 ft) by current and future receptors into areas where contamination also is found below the ground surface.

3.4.1.2 Exposure Point Concentrations for External Gamma Radiation

No direct gamma exposure rate measurements were available for site properties. RESRAD 4.6 was used to estimate direct exposure based on soil contamination levels. Exposure from external gamma irradiation is mainly from the top 10 cm (4 in.) of soil due to gamma ray attenuation by the overlying soil at deeper depths. Therefore, the radiological concentrations from the 0 to 0.6 m (0 to 2 ft) soil layer were used to calculate external gamma exposure. The RESRAD model incorporates an assumed erosion rate that exposes the subsurface soil over time. The dose from external irradiation reported for the future use scenarios is the higher of the present or future conditions.

Details of external gamma dose calculations are provided in Gilbert et al. (1989) and are summarized as follows:

$$D_i = C_{soil,i} \times ETF \times DCF_i$$

where:

D_i = exposure rate from radionuclide i (mrem/yr);

$C_{soil,i}$ = Mean or UL_{95} soil concentration of radionuclide i (pCi/g);

ETF = environmental transport factor (g/cm^3) (accounts for density of soil material, thickness of contaminated zone and cover, occupancy factor, shielding factor, shape factor, area factor, and depth factor); and

DCF_i = external gamma dose conversion factor for radionuclide i, $(mrem/yr)/(pCi/cm^3)$.

Environmental transport factors provided in Gilbert et al. (1989) were used. it was assumed that the indoor external gamma exposure rate was reduced by 20 percent due to shielding afforded by structural materials (EPA 1989b).

3.4.1.3 Exposure Point Concentrations for Radon

No radon measurements were available for site properties. Radon concentrations in outdoor or indoor air, as appropriate for the various scenarios, were estimated for the other properties from the concentrations of Ra-226 in soil using RESRAD 4.6. Thoron (Rn-220) emanation from soils was not considered because, due to its relatively short half-life compared to Rn-222, most thoron would decay to nongaseous daughter nuclides before reaching the ground surface.

3.4.1.4 Exposure Point Concentrations for the Inhalation of Particulates

Radiological Data

Air concentrations of resuspended particles of radionuclide COCs were derived from surface soil concentrations for the areas comprising the Tonawanda site. An ambient airborne dust loading of about 0.10 mg (100 μg) of total particulates per cubic meter of air on the average and about 0.20 mg (200 μg) of total particulates per cubic meter of air for the RME (Gilbert 1983; Paustenbach 1989) has been assumed. Approximately 50 percent of the dust loading originates from soil or similar material (Trijois et al. 1980). For the Tonawanda site, contaminated soil is assumed to be the only source for that portion of the airborne particulates. Therefore, 50 percent of the airborne dust is assumed to have originated from the contaminated soil.

The respirable portion of the total particulate concentration is used as the exposure point concentration for all calculations involving the inhalation of particulates. For this assessment, 30 percent is used as a conservative estimate (Paustenbach 1989).

The contaminant concentration in air (pCi/m_3) is estimated for each radionuclide COC as follows:

$$C_{air,i} = C_{soil,i} \times Dust_{air} \times 10^{-3} g/mg \times 0.3 \times 0.5$$

where:

$C_{soil,i}$	=	mean or UL_{95} soil concentration of radionuclide i (pCi/g)
$Dust_{air}$	=	the average or UL_{95} dust concentration in air (0.1 mg/m^3 = ave; 0.2 mg/m^3 = UL_{95})(Gilbert 1983; Paustenbach 1989)
10^{-3}g/mg	=	conversion factor
0.3	=	30% respirable portion of dust concentration in air (Paustenbach 1989)
0.5	=	50% of total dust concentration in air originates from contaminated soil (Trijois et al. 1980).

As with the incidental soil ingestion pathway of exposure, all particulate inhalation exposure scenarios use surface soil data. Where the UL_{95} estimate exceeds the maximum measured soil concentration for the given data set, the maximum concentration is used as the soil concentration in the $C_{air,RME}$ calculation. The exposure point concentrations for particulates in air are shown in Appendix C.

The Clean Air Act 1988 Assessment Package Computer Modeling Program (CAP 88-PC) was used to calculate dose of airborne radionuclide particulates to the population within an 80 km (50 mi) radius of the site (Parks 1991).

Chemical Data

Concentrations of chemical COCs in airborne particulates were derived from surficial soil concentrations for the Tonawanda site. The methodology used is equivalent to that presented in the previous section for radionuclides. The equations and assumptions are the same with the exception that soil concentrations are given in mg/kg and a 10^{-6} conversion factor is required.

3.4.2 Surface Water and Sediments

Because there is a possibility that children may wade and play in accessible portions of Rattlesnake or Twomile Creek, the inadvertent ingestion exposure pathway is included in this BRA.

Radiological Data

Measured surface water and sediment contamination concentrations (assuming a constant concentration over time) are used as the exposure point concentrations for inadvertent ingestion of radiological COCs by older children wading in the brook. The exposure point concentrations for sediment are presented in Tables 3-1A and 3-1C.

Chemical Data

Measured surface water and sediment contaminant concentrations (assuming a constant concentration over time) are used as the exposure point concentrations for inadvertent ingestion of COCs by children while wading and splashing in the stream. These exposure point concentrations are presented in Appendix C.

3.5 ESTIMATION OF CONTAMINANT DOSE AND INTAKE

The estimates of contaminant dose and intake are based on the COC information presented in Section 2, the exposure point concentrations discussed in Section 3.4, the assumptions given in Appendix B, and chemical intake calculation methodology presented in RAGS (EPA 1989a).

Two point estimates are presented for chemical intakes within each scenario, as recommended by recent EPA guidance (EPA 1992f). The mean estimate represents the most likely intake or dose received by the hypothetical receptor, assuming mean values from the distributions of each exposure parameter. The RME assumes that the value for one or two most sensitive parameters within each pathway are the RME levels (UL_{95}) of the distribution for that parameter. A listing of the values and the sources for the average and RME parameters is given in Appendix B.

Radiological Data

Dose conversion factors assumed in the RESRAD modeling are presented in Appendix B. Dose, as reported here, represents the committed effected dose equivalent (CEDE) from an annual exposure dose estimated for a receptor. Total radiation exposures for each location, scenario, and receptor are presented in Table 3-2. A tabulation of incremental contributions to each total dose from each relevant pathway (e.g., soil ingestion, water ingestion, particulate inhalation, direct radiation, and radon inhalation) is presented in Appendix A.

The radiation doses were estimated for current and future use scenarios using the RESRAD computer code (Version 4.6) for the following pathways:

- inhalation of radioactively contaminated particles;

- incidental ingestion of soil;
- direct external exposure; and
- indoor and outdoor radon exposure.

All of the previously mentioned pathways were considered for employee and transient scenarios. Children wading in the local brook were considered to only ingest sediment and surface water.

Chemical Data

Chemical intake estimates are based on EPA methodology presented in RAGS (EPA 1989a) and related guidance (EPA 1991d). Estimated chemical-specific intakes for each primary exposure pathway quantitatively evaluated in the assessment is presented in Appendix C.

Intakes were calculated for soil ingestion and inhalation of soil particulates for current and future employees at Linde, future employees at Ashland 1 and 2, and current transients at Ashland 1 and 2. No chemical characterization data were available to evaluate exposure to chemical COCs at Seaway. Surface water and sediment intakes were estimated for children playing in accessible portions of a local creek. As stated earlier, receptors have no access to surface water and sediment at Linde and Seaway. Section 3.2 discusses exposure pathways selected for quantitative assessment.

Population Dose

Exposure to the general population within 80 km (50 mi) of the Tonawanda site to airborne radioparticulates which might originate at the site was modeled using CAP88-PC (Parks 1991). This model incorporates area-specific population and wind data to estimate dispersal of airborne contaminants. EPA emission factors (EPA 1985) were used to estimate the amount of contaminated dust released as a result of erosion. Wind data from the Buffalo, NY airport was used in the modeling.

3.5.1 Scenario-Specific Assumptions and Intake Parameters

The assumptions used to estimate radiological and chemical intakes for the receptors described in Section 3.3 are discussed in Sections 3.5.1.1 through 3.5.1.3 and Appendix B. Values assumed for scenario parameters, and the guidance on which they are based, are presented in Table B-1.

3.5.1.1 Exposure Time, Exposure Frequency, and Exposure Duration

Exposure time (ET), frequency, and duration determine the total time a receptor is exposed to the contaminant source. ET is the number of hours per day that a receptor is present

at a specific exposure point. Exposure frequency (EF) is the number of days per year that the exposure occurs, and exposure duration (ED) is the total number of years over which exposure occurs.

The employee scenario at Linde is divided into indoor and outdoor exposures. For the current employee at Linde, it is assumed that 0.2 to one hour per day is spent outdoors onsite (ET = 0.2-1.0). Employees are assumed to spend seven hours per day indoors working in contaminated areas. Adding the outdoor and indoor average and RME exposures may give a realistic presentation of the range of actual employee exposures that could be expected. The Linde employee is assumed to work onsite 250 days a year (EF) for 30 years (ED) in the RME and for 22 years (ED) in the average exposure site specific values. While much higher than the national average, the ED at Linde is based on current employee retention patterns. Future employees at Ashland 1 and Ashland 2 are assumed to have an ED of 25 years in the RME and seven years in the average exposure (EPA 1989b). Seven years is used as the average time spent at one job based on the ratio of 30 years as the RME and a 50th percentile of nine years at one residence as the average (EPA 1989b). No indoor chemical exposure is considered for the employee risk.

Because Ashland 1 is not an active industrial site and Seaway is an operating landfill without a permanent structure on or near any contamination, receptors in the current scenario are adult transients who might occasionally visit the site to make deliveries, monitor the site, or dump a truckload at the landfill. The average transient is represented to spend 15 minutes per day for a total of 100 days per year. The RME transient spends one hour per day, 250 days per year in contaminated areas of the site.

Ashland 2 is only partially fenced and is accessible on foot, so the receptors in the current scenario are represented as older (35 kg [77 lbs]) children who play at the site. Future land use at Seaway is assumed to be recreational with an older child as a receptor. The average child is assumed to play occasionally at the site (one-half hour per day, two days a week, 25 weeks a year). The RME child plays frequently at the site (two hours a day, five days a week) during the 10 week summer vacation and half an hour a day, two days a week during the remainder of the year.

Surface water and sediment ingestion intake estimates for exposure to older children wading in a local stream which could be the confluence of Rattlesnake and Twomile Creeks assume the same exposure frequency as RAGS suggests for swimming, seven events (d/yr) (EPA 1989a), one h/event. Because the receptor is a child, a six-year exposure time was used.

3.5.1.2 Inhalation

An inhalation rate of 15 m^3/day for average exposures and 20 m^3/day for RME exposures was used in the BRA for assessment of current and future employee scenarios (EPA 1991d). Inhalation rates are based on a combination of light and moderate activity.

The inhalation rate for the transient scenario was adjusted to account for greater activity and higher inhalation rates while outdoors. An inhalation rate of 1.0 m^3/h was used for the average and 1.4 m^3/h was used for the RME. These rates are based on a combination of light and moderate activity and age-adjusted inhalation rates (EPA 1989a).

3.5.1.3 Ingestion Rates

Incidental soil ingestion rates are based on recent EPA guidance (EPA 1991d). The RME soil ingestion rate recommended for employees is 50 mg/day (EPA 1991d). Transient soil ingestion doses for adults and older children were based on average and RME ingestion rates of 50 mg/day and 100 mg/day, respectively (EPA 1991d).

The RME surface water ingestion rate used for chemical and radiological intake estimates for the local child wading scenario is 0.05 L/day [incidental ingestion rate while swimming (EPA 1989b)]. The sediment ingestion rate used is the same as the child soil ingestion rate.

3.5.1.4 Body Weight

The standard assumption for adult body weight is 70 kg (155 lb) (EPA 1989b). A body weight of 70 kg is used for all employee scenarios and for the adult transient. A body weight of 35 kg (77 lb) was assumed for child transients (EPA 1989a).

3.5.1.5 Exposed Skin Surface Area

Adults were assumed to have 5,300 cm^2 of exposed skin; children were assumed to have 3,700 cm^2 exposed (EPA 1992a). Children who are wading or swimming were assumed to have 12,000 cm^2 exposed which is 100 percent of the surface area of the 50th percentile of a 10-year-old child (EPA 1989a, EPA 1992a).

3.5.2 Exposure Calculation Equations for Ingestion of Soil

3.5.2.1 Radiological Dose From Soil Ingestion

Doses associated with the intake of radioactive contaminants resulting from incidental ingestion of surface soil were calculated using RESRAD (Gilbert at al. 1989) as follows:

$$D_i = C_{soil,i} \times IR_s \times EF \times DCF_i \times CF_m$$

where:

D_i = dose from radionuclide i (mrem/yr);

$C_{soil,i}$ = soil concentration of radionuclide i (pCi/g); (arithmetic mean for average exposure and UL_{95} for RME)

IR_S = soil ingestion rate (mg/day)(from EPA 1991b);

EF = exposure frequency (day/yr);

DCF_i = ingestion dose conversion factor for radionuclide i (mrem/pCi); and

CF_m = conversion factor, 10^{-3} g/mg.

3.5.2.2 Chemical Intake Due to Soil Ingestion

The equation used to calculate chemical soil intake for soil ingestion was obtained from RAGS (EPA 1989a) as follows:

$$Intake(mg/kg\text{-}day) = \frac{C_s \times IR_s \times CF \times FI \times EF \times ED}{BW \times AT}$$

where:

C_s = chemical concentration in soil (mg/kg)

IR_s = ingestion rate

CF = conversion factor (10^{-6} kg/mg)

FI = fraction ingested from contaminated source (unitless): assumed that all of the ingested soil is from the contaminated source area for each scenario, therefore, FI = 1;

EF = exposure frequency (day/yr): receptor/scenario-specific time spent outdoors;

ED = exposure duration (yr) receptor/scenario specific;

BW = body weight (kg); and

AT = averaging time (days).

Receptor-specific variable values are contained in Appendix B (Table B-1).

3.5.3 Equations for Exposure to Water

3.5.3.1 Ingestion of Contaminants from Surface Water

Radiological Data

The doses associated with intake of radiological contaminants resulting from incidental ingestion of surface water while wading or swimming were calculated using RESRAD 4.6 as follows:

$$D_i = C_{sw,i} \times IR_w \times EF \times DCF_i$$

where:

D_i = dose from radionuclide i (mrem/yr),

$C_{sw,i}$ = concentration of radionuclide i in water (pCi/L),

IR_w = water ingestion rate (L/d),

EF = exposure frequency (d/yr), and

DCF_i = ingestion dose conversion factor for radionuclide i (mrem/pCi).

Chemical Data

The following equation was obtained from RAGS (EPA 1989a) for incidental ingestion of surface water while swimming and is used for the estimated chemical intake calculations for the current and future child transients playing and wading in the accessible portions of Rattlesnake Creek:

$$Intake(mg/kg-d) = \frac{C_{sw} \times CR \times EF \times ED \times ET}{BW \times AT}$$

where:

C_{sw} = chemical concentration in water (mg/L): chemical-specific arithmetic mean for average exposure and UL_{95} for RME;

CR = contact rate (0.05 L/h);

EF = exposure frequency (7 events/yr) ;

ED = exposure duration (6 yr);

ET = exposure time (1 h/event);

BW = body weight (35 kg); and

AT = averaging time (days).

The contaminant intake calculation for ingestion of sediment during the surface water scenario is the same as that for soil as recommended by EPA (EPA 1989a). Variable values for these two equations are contained in Appendix B (Table B-1). Intake estimates for surface water and sediment ingestion are presented in Appendix C.

3.5.4 Equations for Exposure to Contaminants in Air

3.5.4.1 Inhalation of Radon

The doses resulting from inhalation of Rn-222 and its short-lived decay products were based on the exposure point concentrations in both indoor and outdoor air (Section 3.3.1.3), using the following method:

$$E_{Rn} = E_{RnI} + E_{RnO}$$

$$E_{RnI} = C_{Rn\text{-}I} \times ECF_{CI} \times \frac{1\,WL}{100\,pCi/L}\,RT_I \times 12\ months/yr \times NWCF$$

$$E_{RnO} = C_{Rn\text{-}O} \times ECF_O \times \frac{1\,WL}{100\,pCi/L} \times RT_O \times 12\ months/yr \times NWCF$$

where:

E_{Rn} = radon exposure in working level months (WLM)

E_{RnI} & E_{RnO} = exposure for indoor and outdoor exposure, respectively (in WLM)

$C_{Rn\text{-}I}$ & $C_{Rn\text{-}O}$ = radon concentration for indoor and outdoor, respectively ($_p$Ci/L)

ECF_I & ECF_O = equilibrium factors for indoor and outdoor air (.45 and .1, respectively)

RT_I & RT_O = fraction of the year spent indoors and outdoors at the location, respectively

NWCF = correction factor to WLM (A WLM is defined in terms of 170 hours of exposure per month. The average month contains 730 hours, yielding a correction factor of 4.3)

WL = working level

These doses are not true radiation doses but are actually exposures expressed in WLM. The WLM unit was used because the risk of inhalation of radon decay products is typically expressed in this unit (1 WLM is approximately equivalent to 1,000 mrem) (ICRP 1985). The estimated doses associated with the inhalation of Rn-222 decay products are presented in Appendix A.

3.5.4.2 Inhalation of Particulates

Radiological Data

Radiation dose estimates for the inhalation pathway were calculated using the exposure point concentrations in air discussed in Section 3.4.1.4. Dust concentrations for indoor exposure were assumed to be 40 percent of those outdoors (Alzona et al. 1979).

The RESRAD code was used to calculate the radiological dose from the inhalation of airborne radioparticulates. The dose calculation is detailed in Gilbert et al. (1989) and summarized as follows:

$$D_i = C_{air,i} \times FA \times EF \times IR \times DCF_i \times CF_T$$

where:

D_i = dose from radionuclide i (mrem/yr);

$C_{air,i}$ = air concentration of radionuclide i ($_p$Ci/m^3) which is based on the soil concentration (Section 3.4.1.4);

FA = area factor, dimensionless (represents the fraction of airborne dust that is contaminated);

EF = exposure frequency (day/yr);

IR = inhalation rate (m^3/h);

DCF_i = inhalation dose conversion factor for radionuclide i (mrem/pCi); and

CF_T = conversion factor (24 h/day).

The estimated doses for the identified receptors resulting from the inhalation of airborne radioactive particulates are presented in Appendix A.

Chemical Data

The following equation was obtained from RAGS (EPA 1989a) for the calculation of chemical intake from inhalation of airborne particulates. All exposure is assumed to occur outdoors.

$$Intake\ (mg/kg\text{-}day) = \frac{C_{air} \times IR_a \times ET \times EF \times ED}{BW \times AT}$$

where:

C_{air} = contaminant concentration air (mg/m^3);

IR_a = recommended EPA inhalation rate (m^3/day);

ET = exposure time (h/day) receptor-specific time spent outdoors;

EF = exposure frequency (day/yr) receptor-specific (EPA 1991b);

ED = exposure duration (yr) receptor-specific (EPA 1989a);

BW = body weight (kg); and

AT = averaging time (days).

Variable values used to calculate chemical intakes are contained in Appendix B (Table B-1). Estimates of inhalation intakes for chemical COCs are presented in Appendix C.

3.5.5 Equations for Dermal Exposure to Soil and Surface Water

Dermal exposure to cadmium was calculated using the following method:

$$DAD\ (mg/kg\text{-}d) = \frac{DA_{event} \times EF \times ED \times SA}{BW \times AT}$$

where:

DAD = dermally absorbed dose (mg/kg-day);

DA_{event} = absorbed dose per event (mg/cm^2 - event);

SA = skin surface area available for contact (cm^2);

EF = exposure frequency (d/yr);

ED = exposure duration (6 yr);

BW = body weight (kg); and

AT = averaging time.

$$DA_{event}\ (mg/cm^2\text{-}event) = C_{soil} \times AF \times ABS$$

where:

DA_{event} = absorbed dose per event (mg/cm^2 - event);

C_{soil} = contaminant concentration in soil (mg/kg) $\times$ (10^{-6} kg/mg);

AF = adherence factor (mg/cm^2 - event); and

ABS = absorption fraction.

3.5.6 Summary of Radiological Exposure Estimates

3.5.6.1 Maximally Exposed Individuals

Estimates of the committed effective dose equivalent from a one-year exposure to radiological COCs at the Tonawanda site are given in Table 3-2. In compiling this tabulation, contributions to dose were calculated for soil ingestion, inhalation of resuspended particulates, inhalation of radon daughters, and direct external gamma radiation. The incremental dose components are provided in Appendix A.

The annual doses for the Tonawanda site also are presented in coded maps for all scenarios and receptors in Figures 3-7 through 3-20. The graphical presentation of total dose for the Tonawanda properties includes five ranges; <10 mrem/yr, 10 to 25 mrem/yr, 25 to 100 mrem/yr, 100 to 500 mrem/yr, and >500 mrem/yr. The 10 mrem/yr threshold is selected to provide an order of magnitude reduction of the primary public dose limit of 100 mrem/yr to account for potential multiple exposures; a 10 mrem/yr limit is also imposed by the EPA National Emission Standards for Hazardous Air Pollutant (NESHAPs) for doses from airborne radioactive material and is the threshold for reporting per DOE Order 5400.5 The 25 mrem/yr breakpoint is specified in 40 CFR 192.41(d) for maximum whole body dose to the public from

thorium ore processing operations. The 100 mrem/yr limit is the primary DOE dose limit to the public from all sources of radiation, as described in DOE Order 5400.5, Chapter II. The 500 mrem/yr upper breakpoint is the maximum annual dose limit to the public (for a single year only) allowed by DOE Order 5400.5.

3.5.6.2 Average Population Dose

The population dose from the airborne dispersion of radioactive particulates to a radial distance of 80 km (50 mi) was estimated. The collective dose, for a population of three million in the area considered, is 1 person-rem/yr, or an average population dose of 3×10^{-7} mrem/yr per person. This compares with the annual background dose from all sources and exposure routes of approximately 300 mrem/yr.

3.5.7 Summary of Chemical Intake Estimates

Estimated intakes of chemical COCs are summarized in Appendix C. Estimated intakes for incidental soil ingestion and inhalation of soil particulates were calculated for the current and future employee at Linde, future employees at Ashland 1 and Ashland 2, and current and future transients at Ashland 1 and Ashland 2 and current transient at Seaway. Average exposure intakes and RME intakes were calculated using arithmetic mean and UL_{95} soil and concentrations, respectively, to develop the exposure point concentrations for all scenarios. Inhalation intakes included the assumptions for airborne contaminated particulate dust concentrations discussed in Section 3.3.1. Dermal contact with soil and surface water was assessed for cadmium. Section 3.2 discusses exposure pathways selected for quantitative assessment.

Average and RME surface water and sediment intakes were estimated for the current and future transient child wading and playing in a local creek.

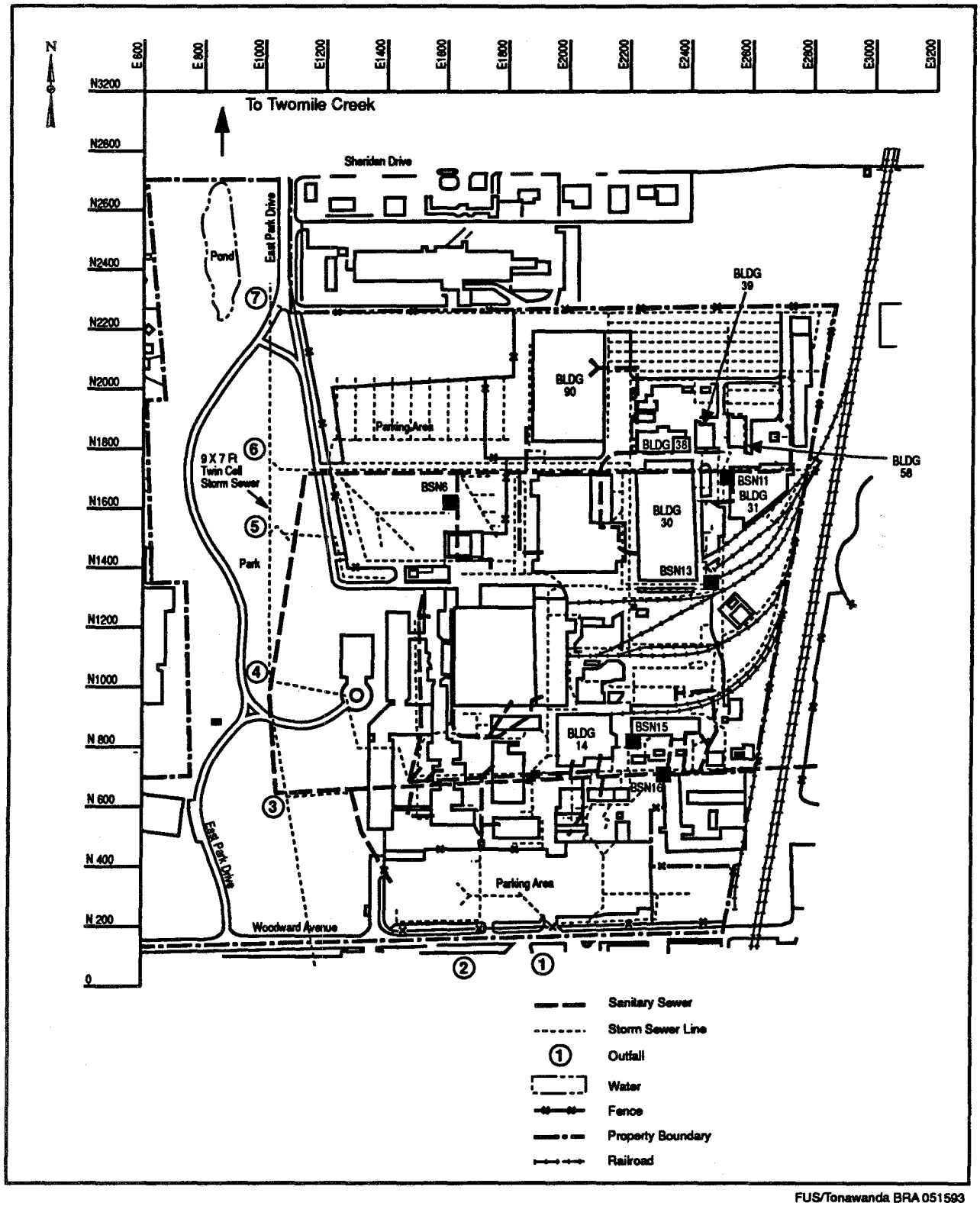

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Figure 3-1. Locations of Storm and Sanitary Sewers at Linde

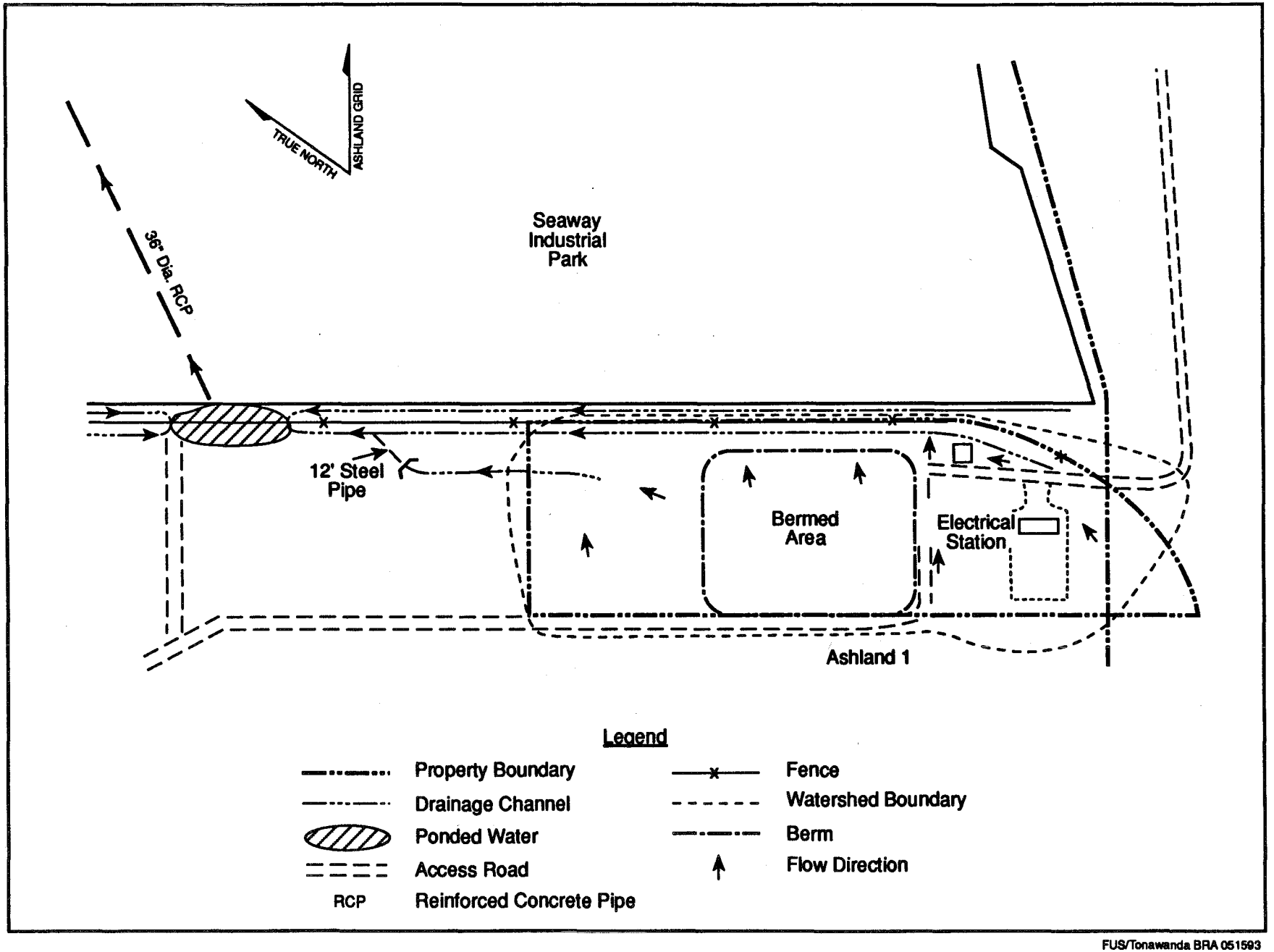

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Figure 3-2. Ashland 1 Site Map

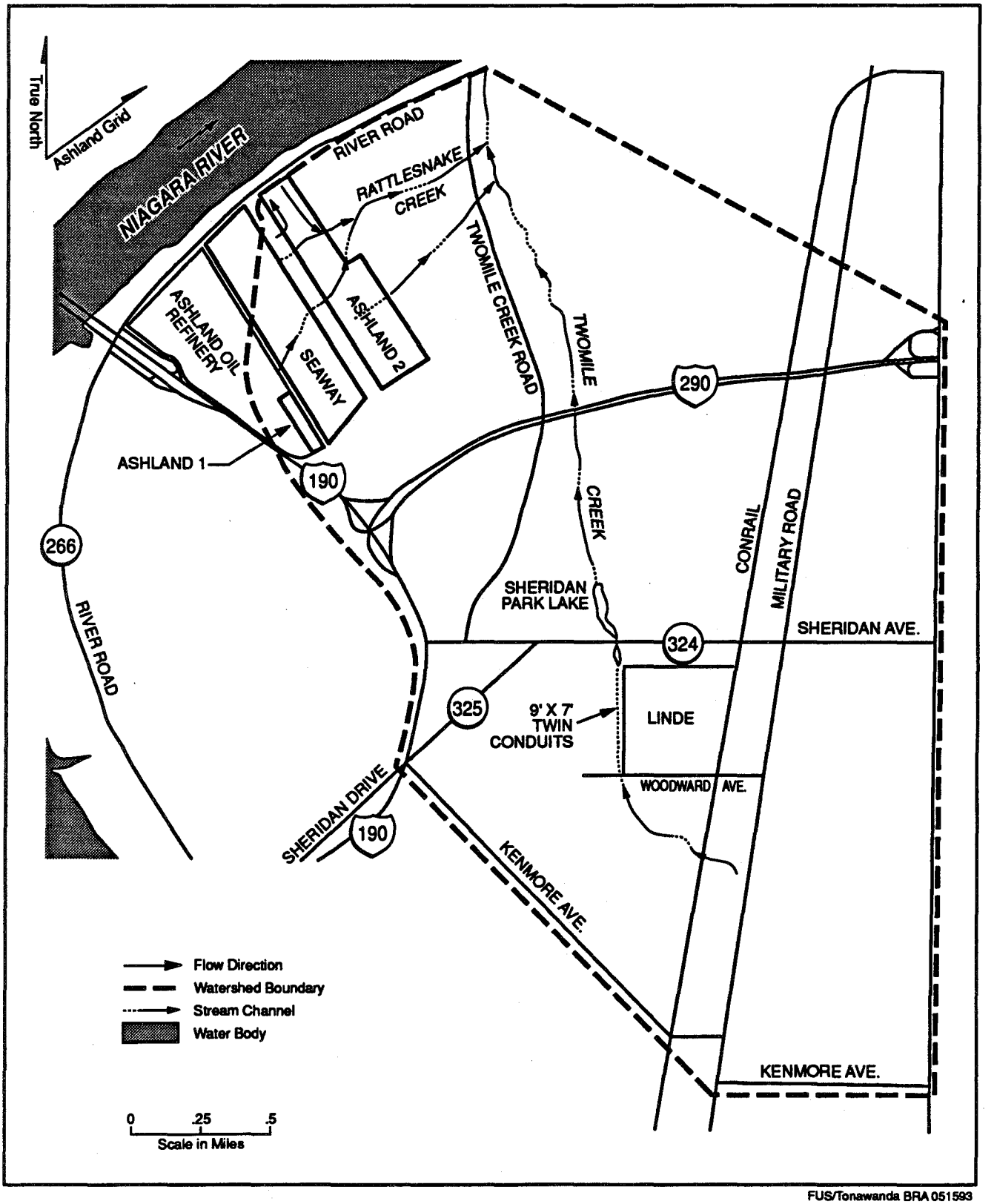

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Figure 3-3. Receiving Waters for the Tonawanda Site

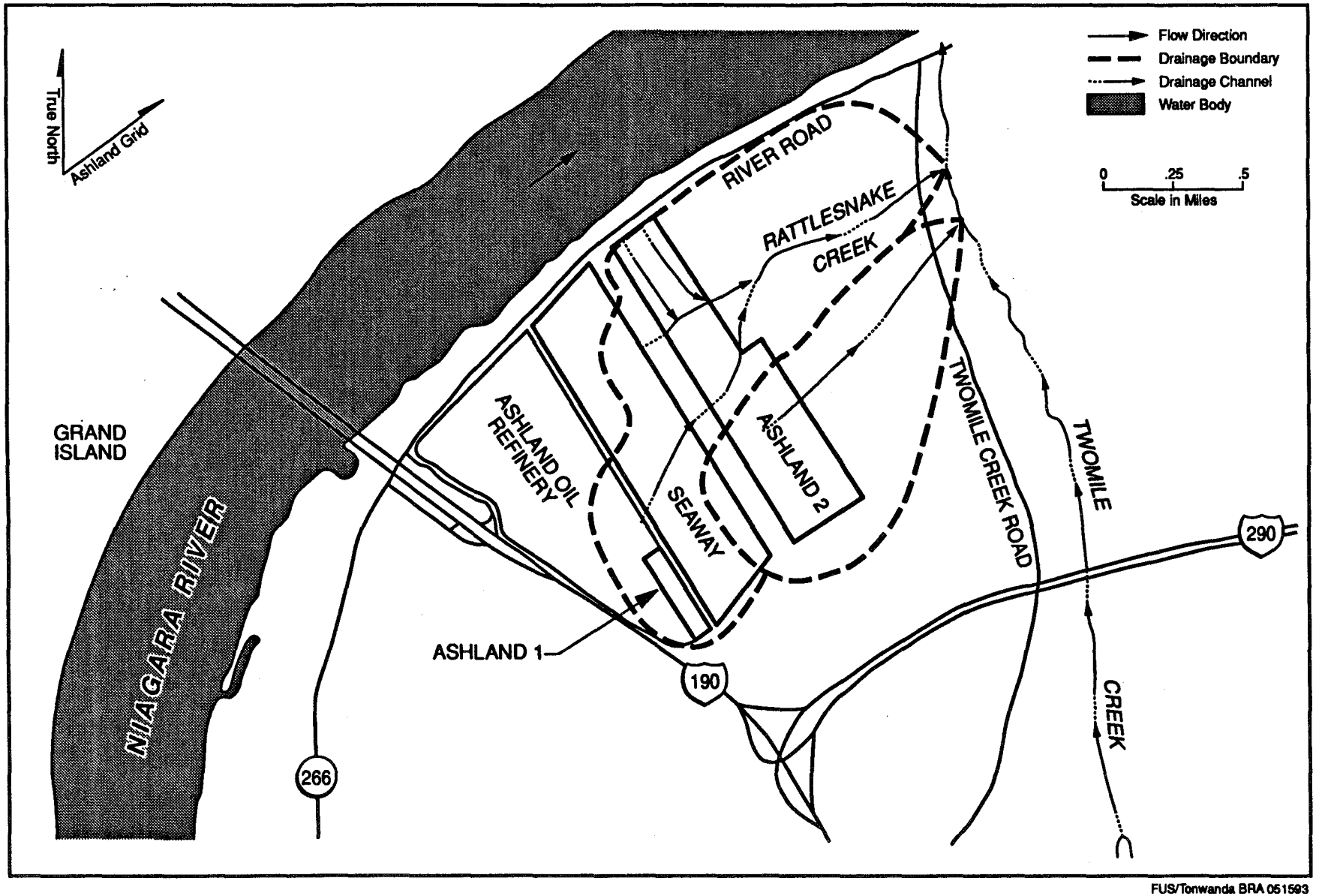

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Figure 3-4. Rattlesnake Creek Drainage Area

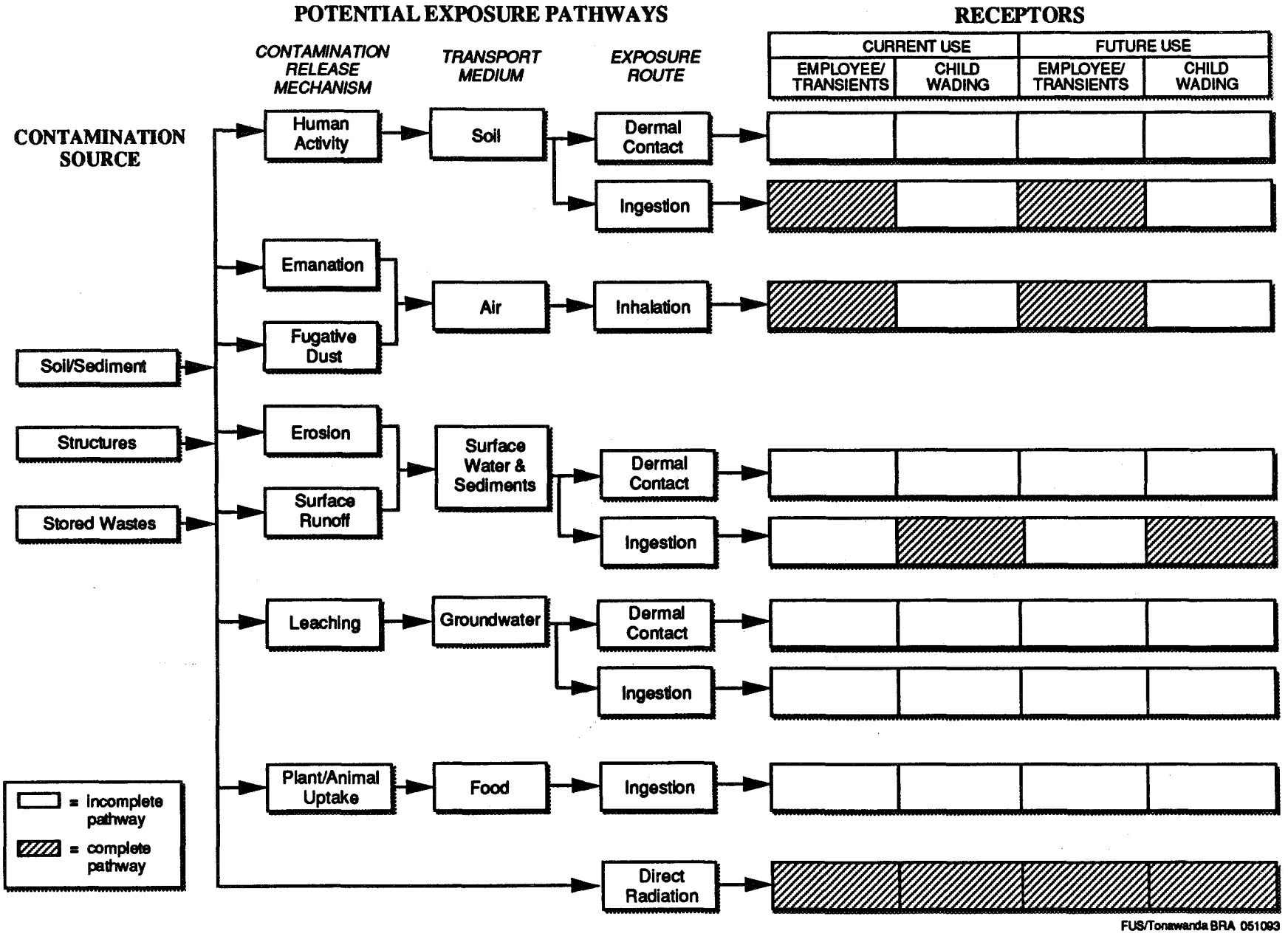

Figure 3-5. Conceptual Site Model for Exposure Pathways for Radiologically Contaminated Soil and Sediments

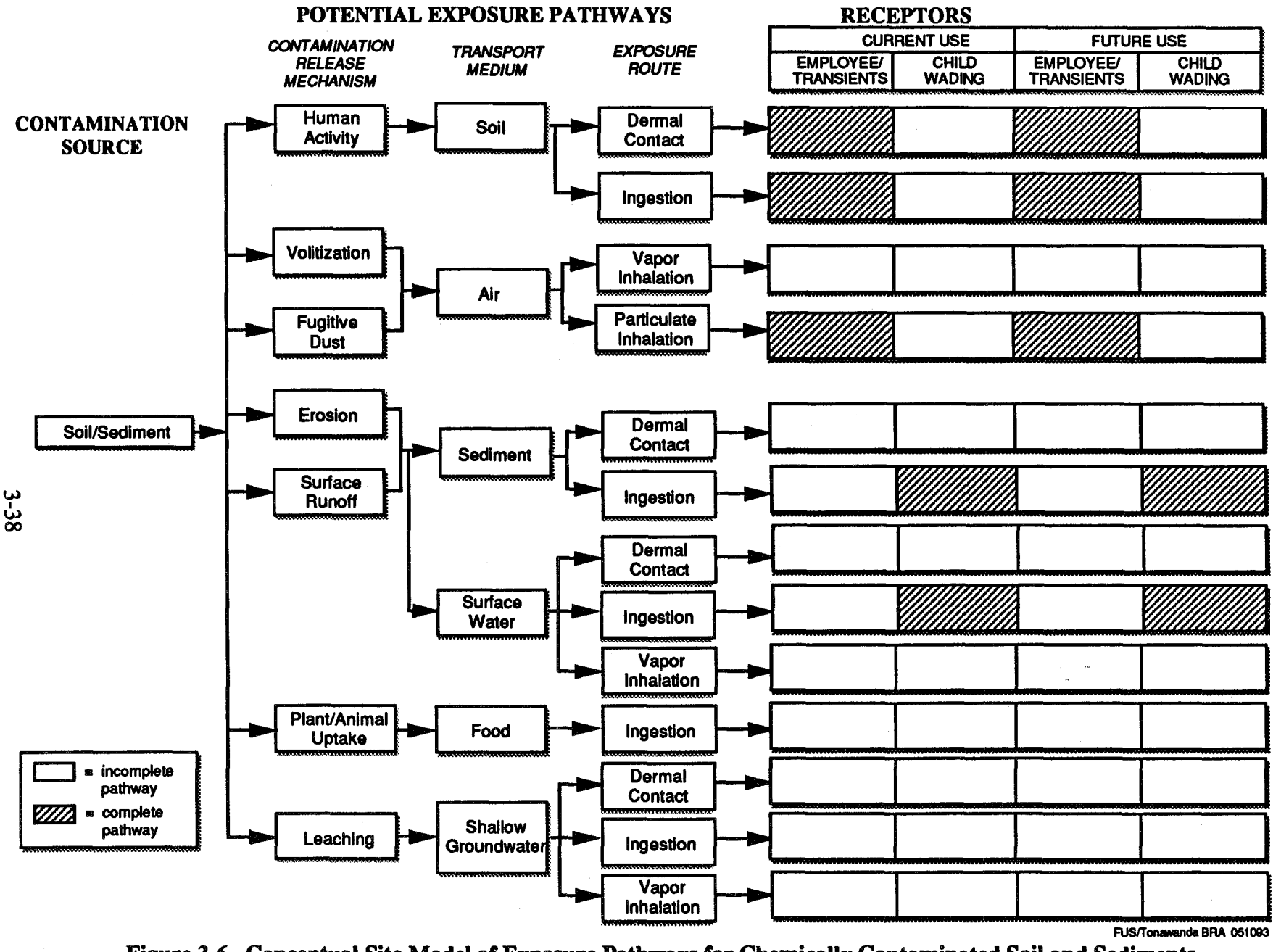

Figure 3-6. Conceptual Site Model of Exposure Pathways for Chemically Contaminated Soil and Sediments

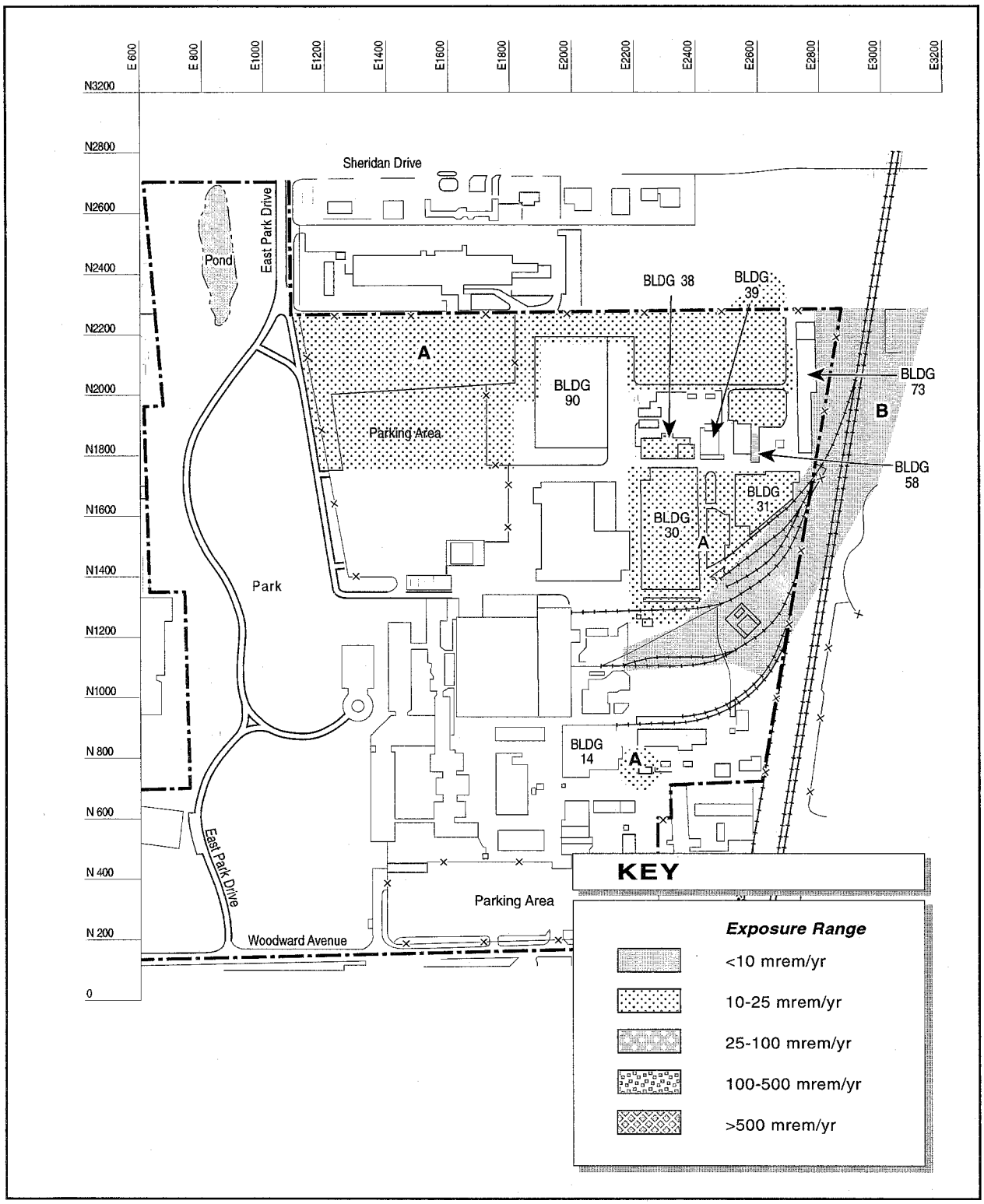

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Figure 3-7. Mean Radiological Exposure in the Current and Future Use Scenarios at the Linde Property

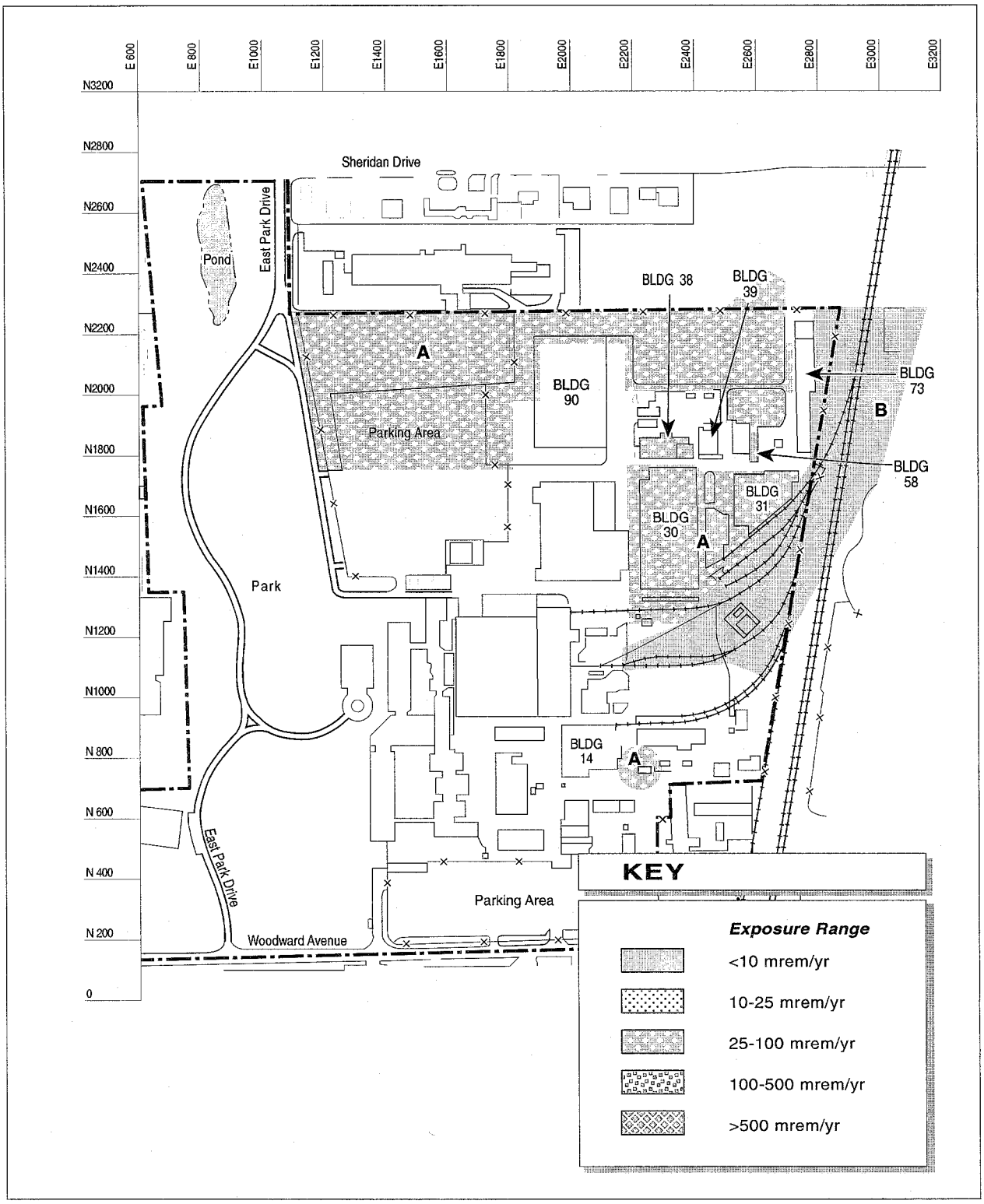

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Figure 3-8. RME Radiological Exposure in the Current and Future Use Scenarios at the Linde Property

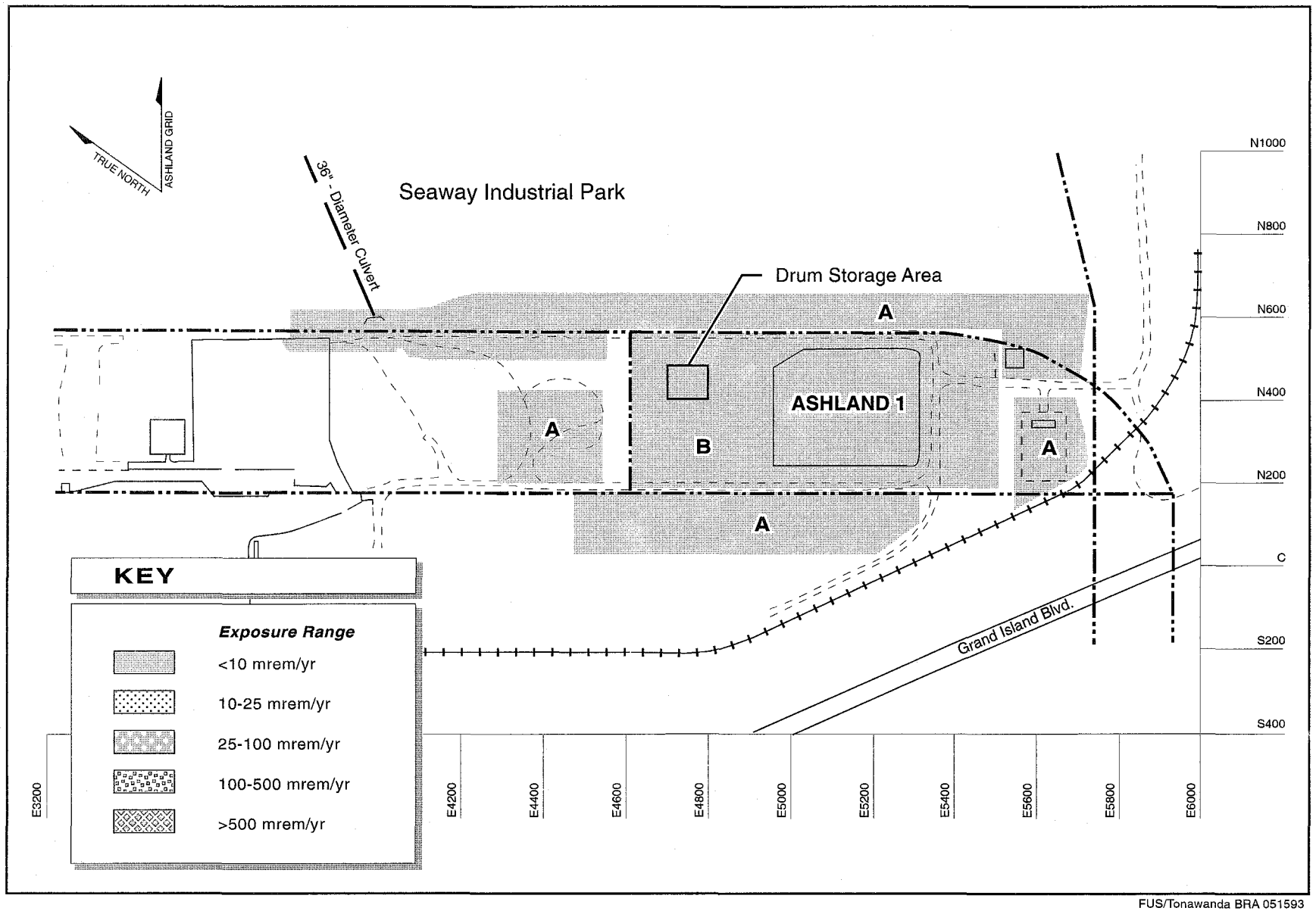

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Figure 3-9. Mean Annual Radiological Exposure in the Current Use Scenario at the Ashland 1 Property

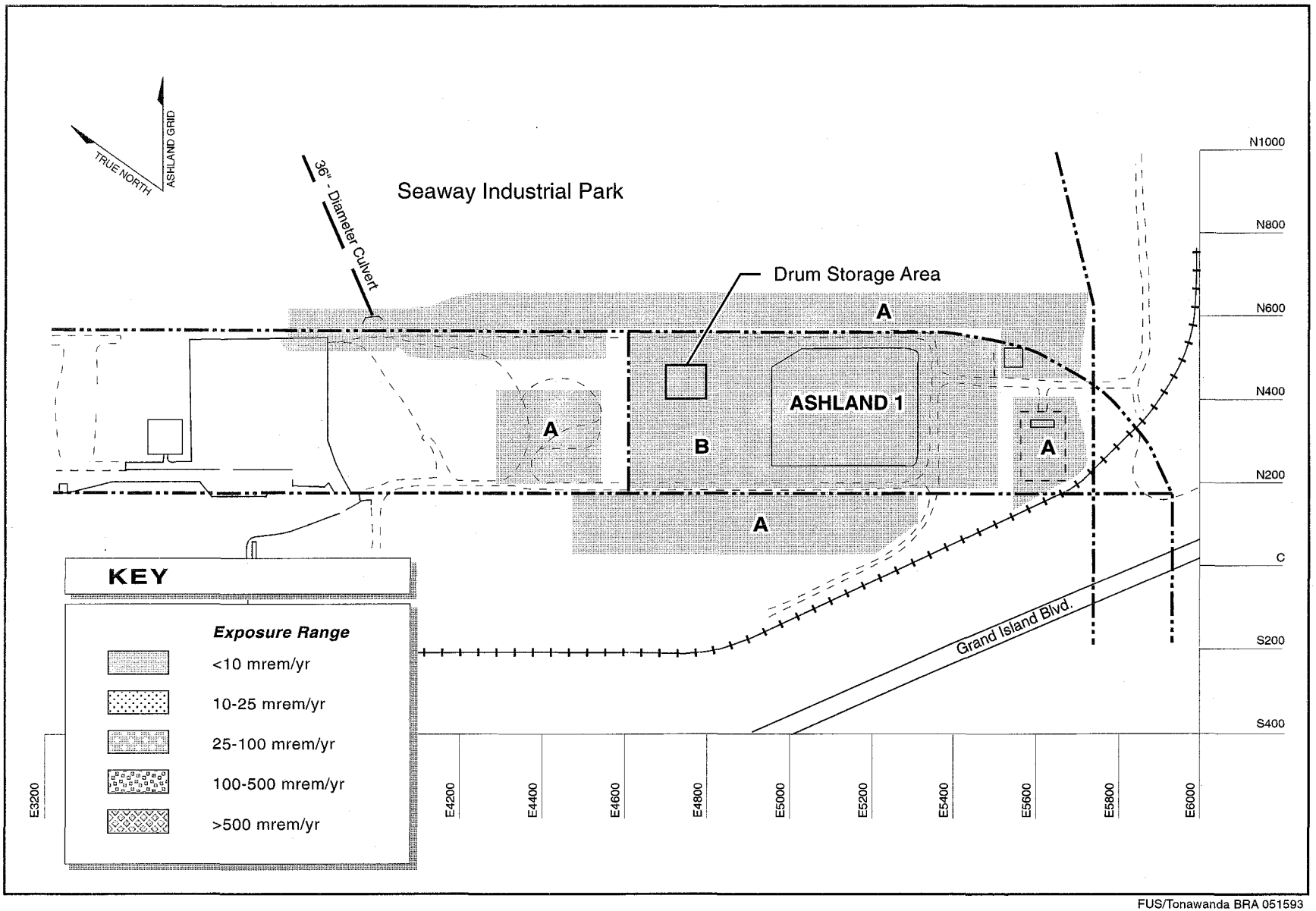

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Figure 3-10. RME Annual Radiological Exposure in the Current Use Scenario at the Ashland 1 Property

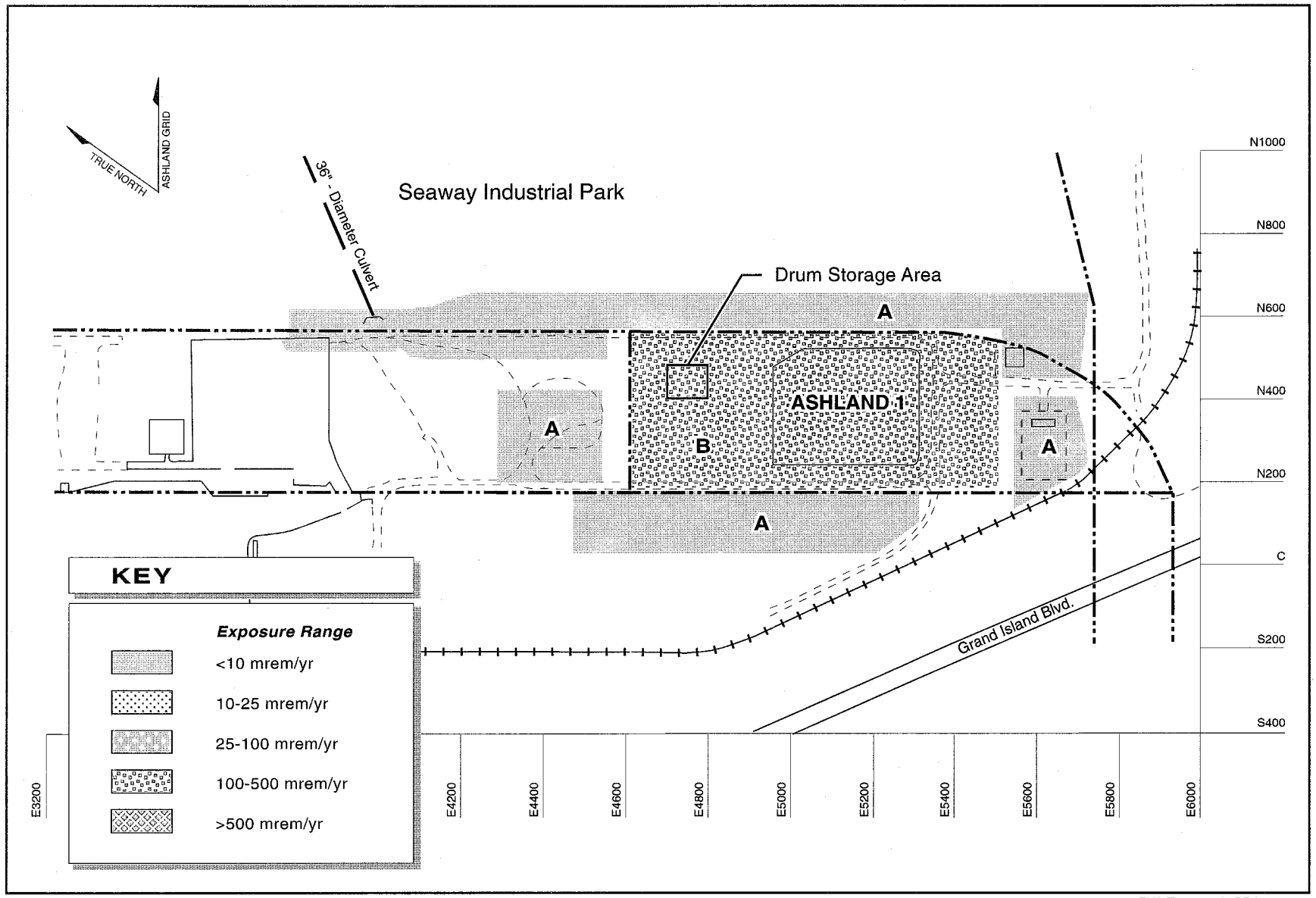

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Figure 3-11. Mean Annual Radiological Exposure in the Future Use Scenario at the Ashland 1 Property

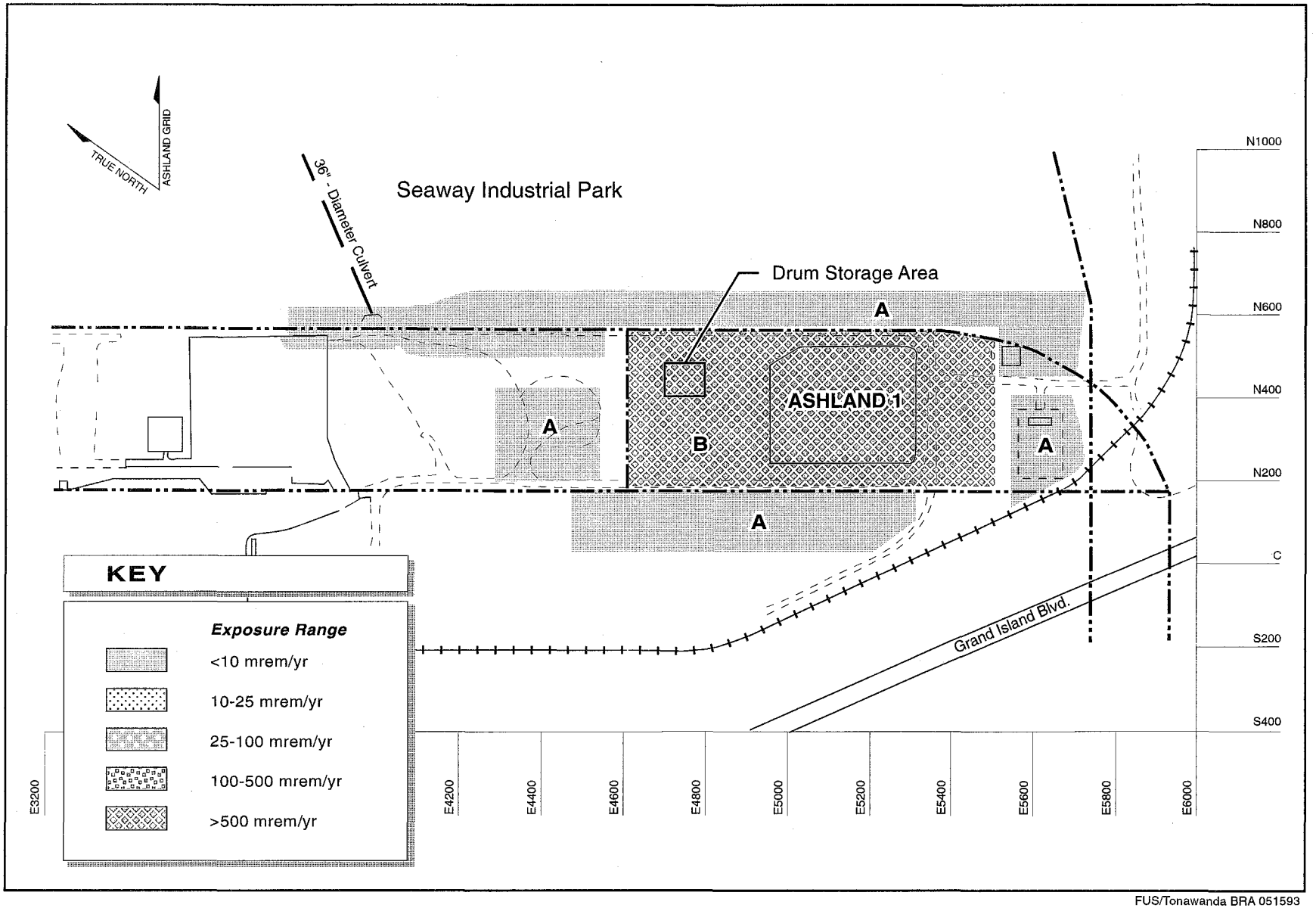

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Figure 3-12. RME Annual Radiological Exposure in the Future Use Scenario at the Ashland 1 Property

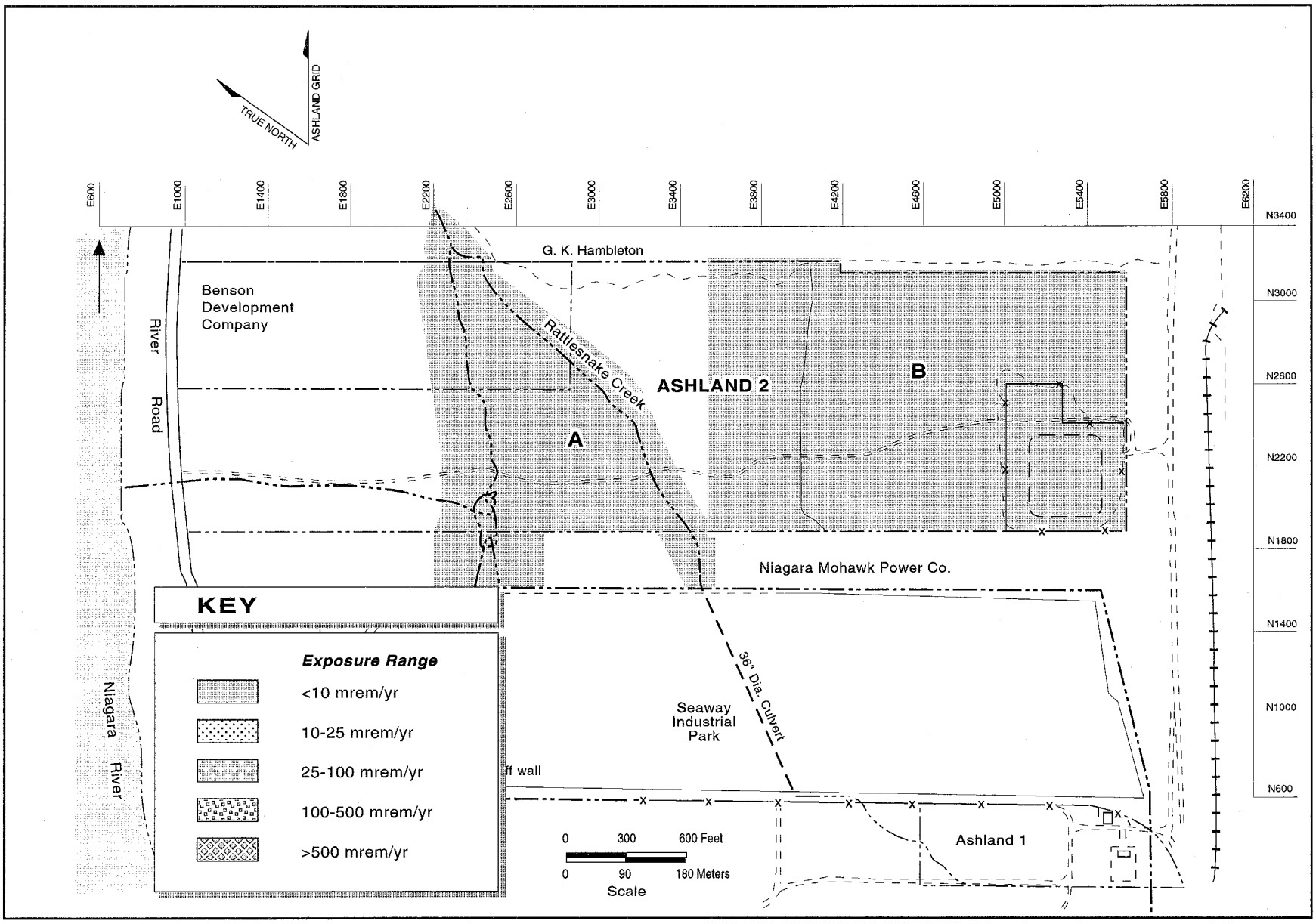

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Figure 3-13. Mean Annual Radiological Exposure in the Current Use Scenario at the Ashland 2 Property

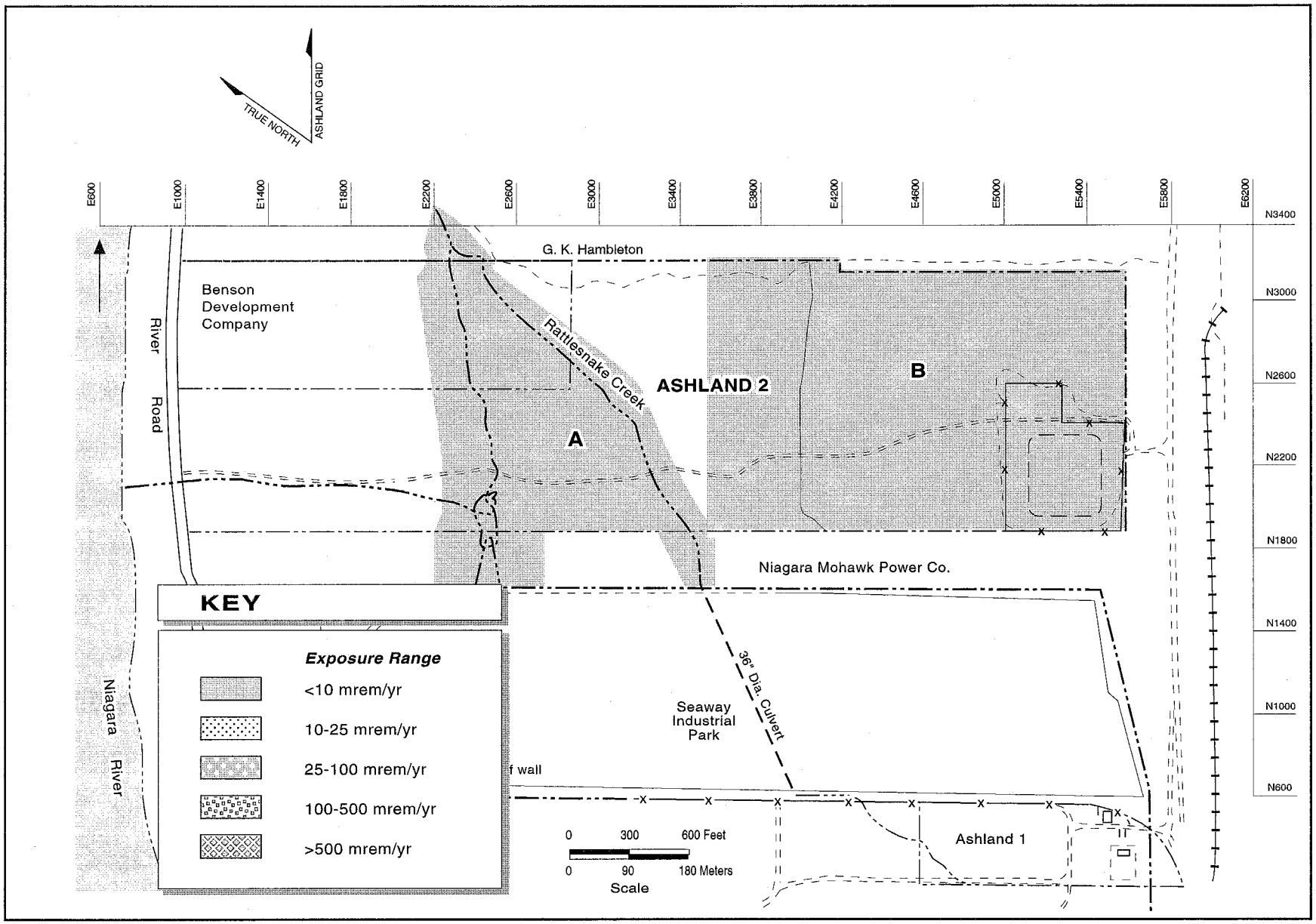

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Figure 3-14. RME Annual Radiological Exposure in the Current Use Scenario at the Ashland 2 Property

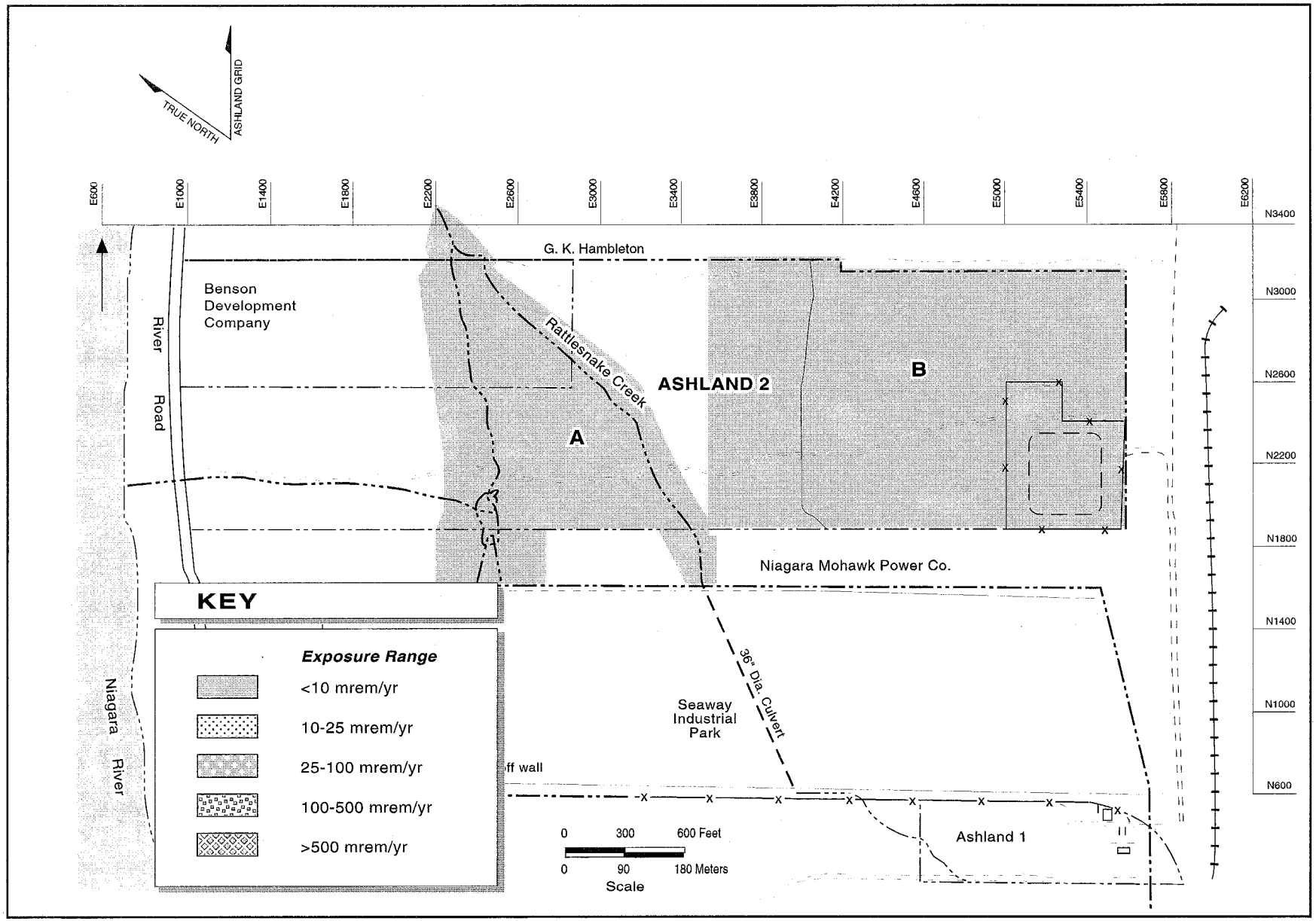

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Figure 3-15. Mean Annual Radiological Exposure in the Future Use Scenario at the Ashland 2 Property

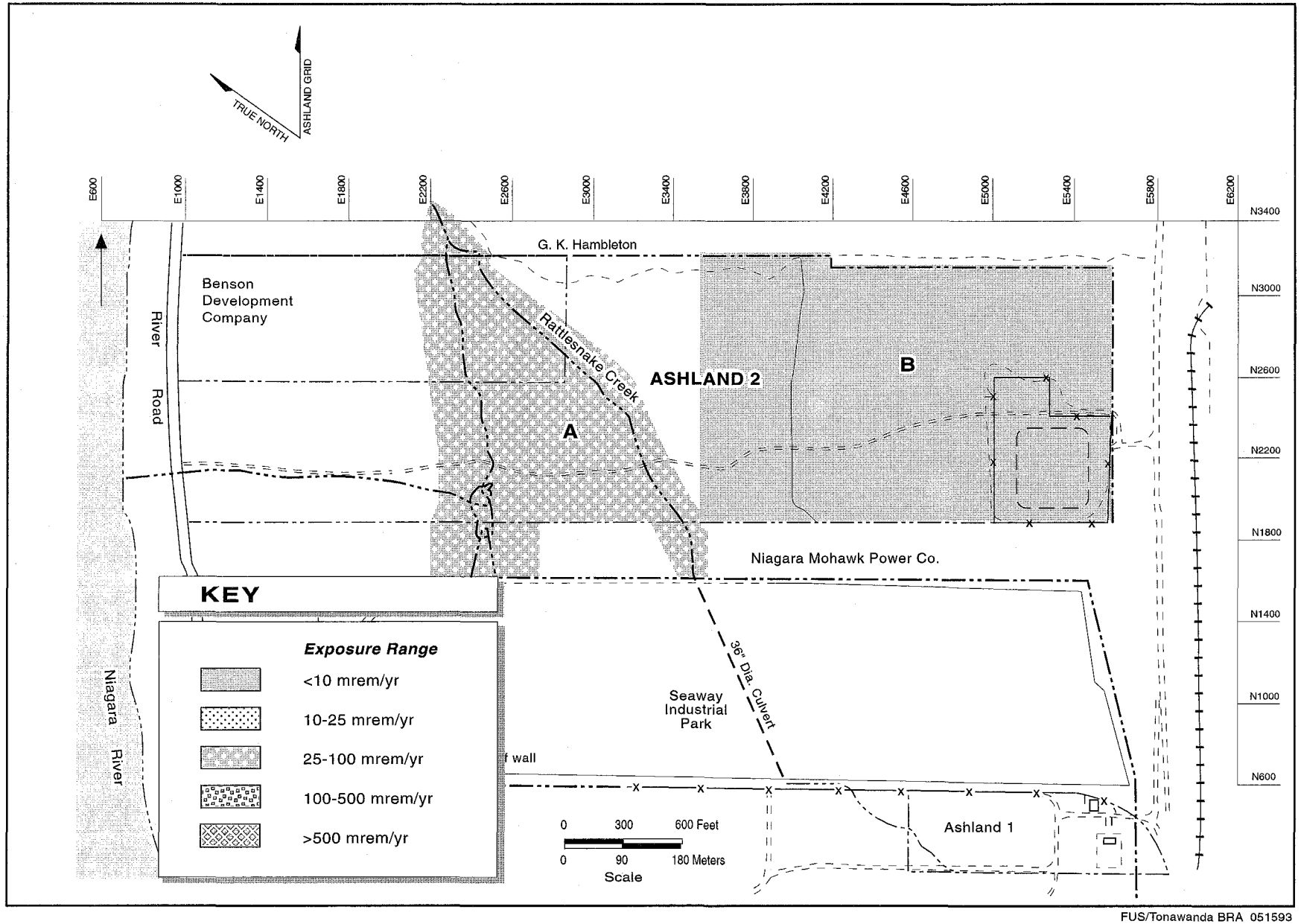

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Figure 3-16. RME Annual Radiological Exposure in the Future Use Scenario at the Ashland 2 Property

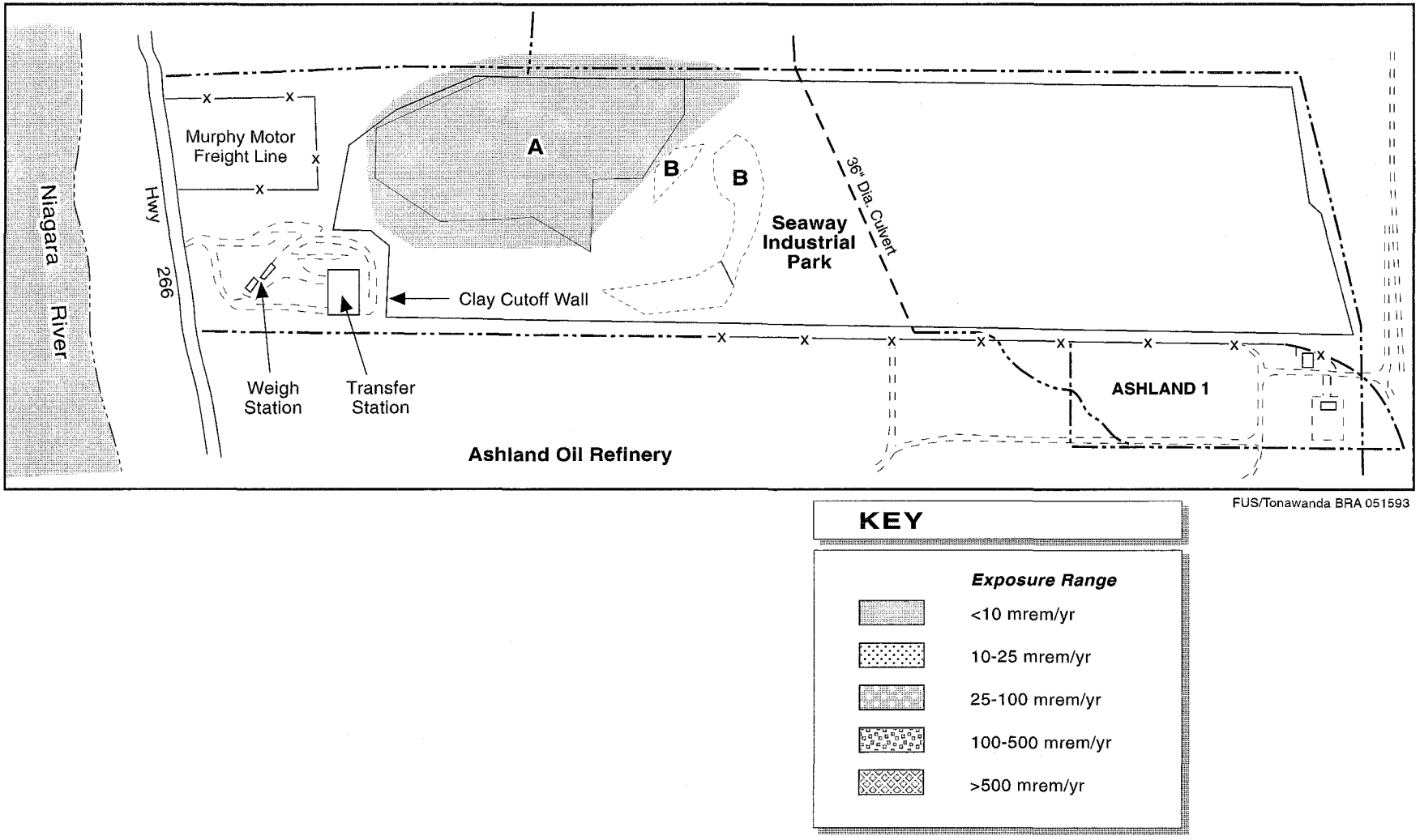


Figure 3-17. Mean Annual Radiological Exposure in the Current Use Scenario at the Seaway Property

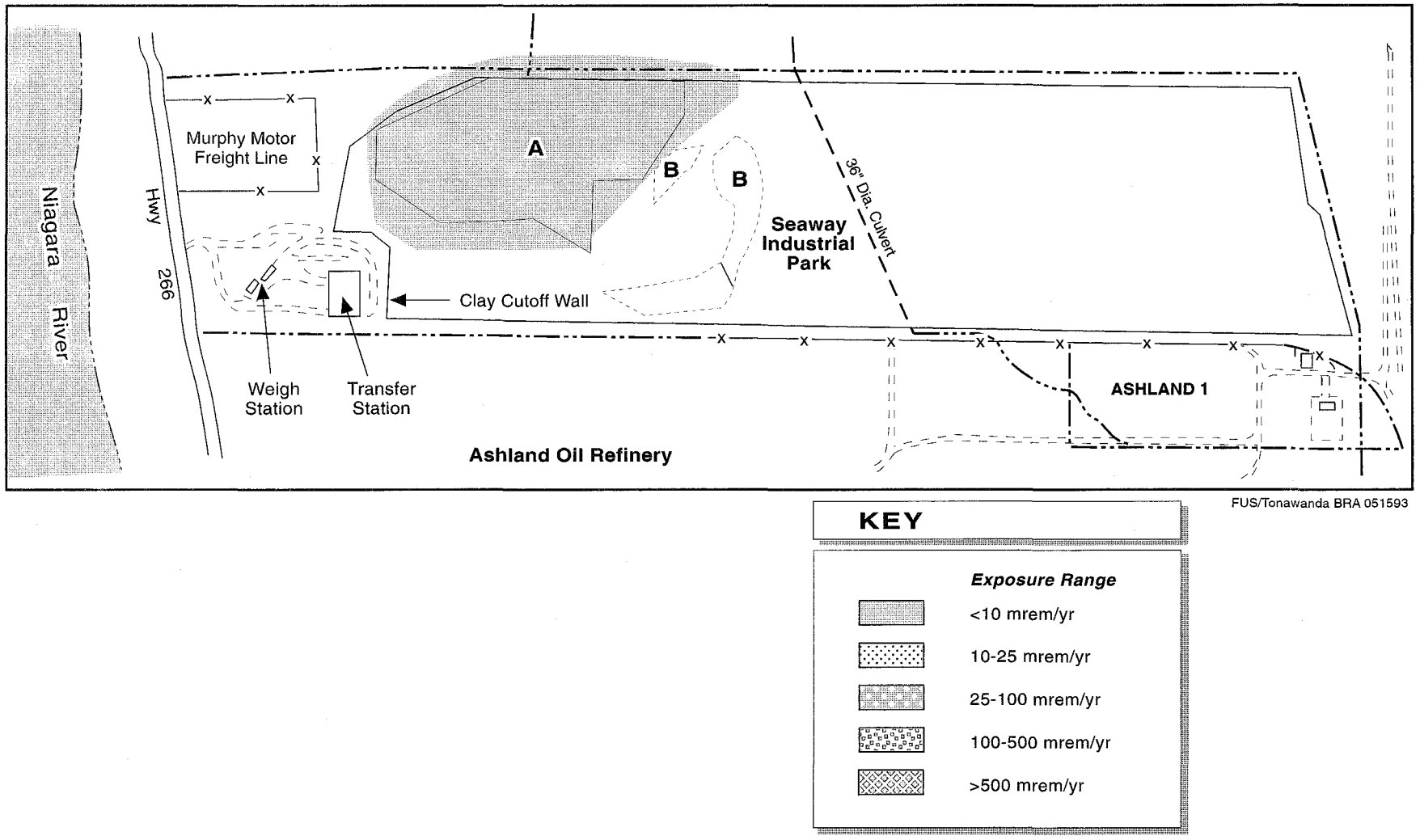


Figure 3-18. RME Annual Radiological Exposure in the Current Use Scenario at the Seaway Property

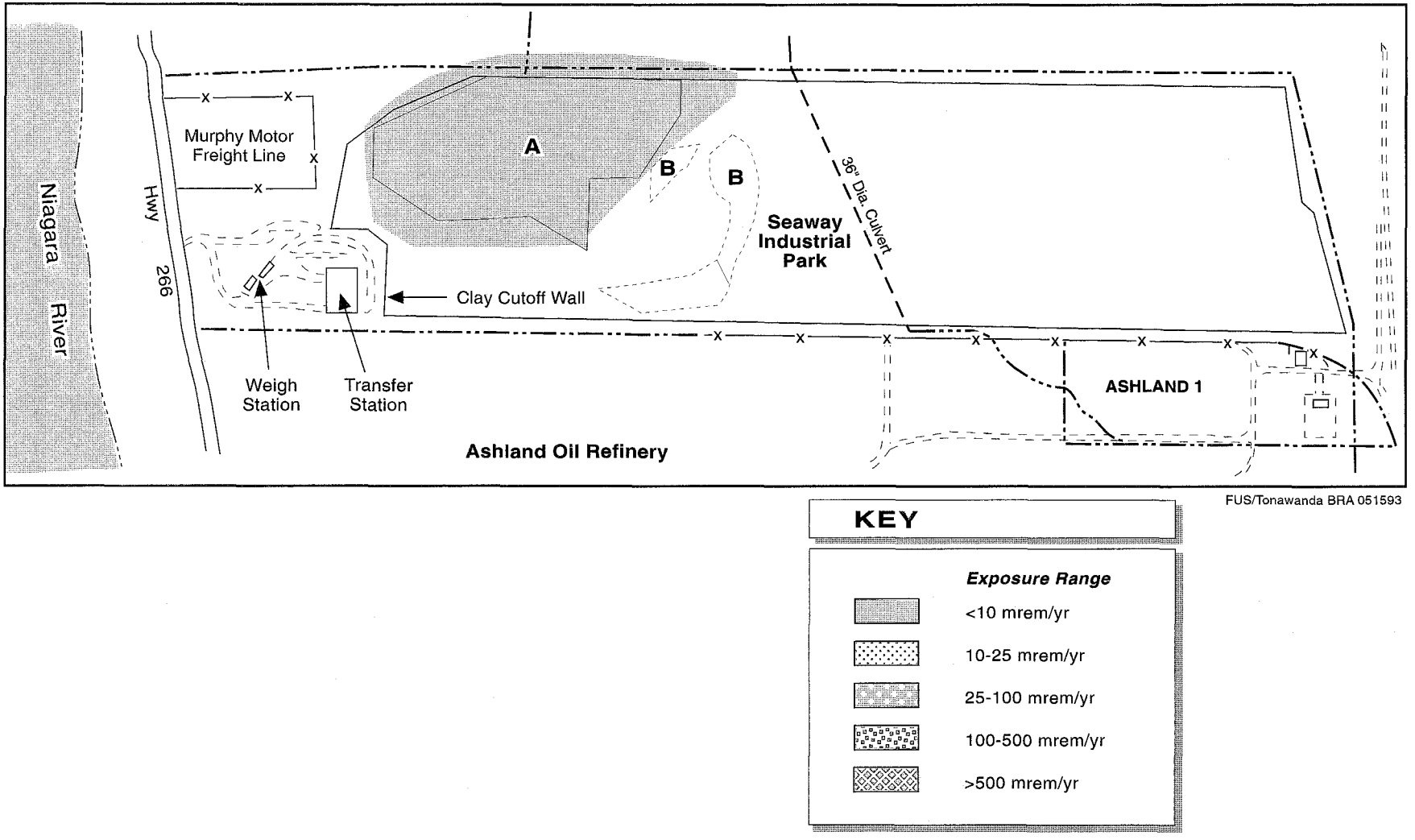


Figure 3-19. Mean Annual Radiological Exposure in the Future Use Scenario at the Seaway Property

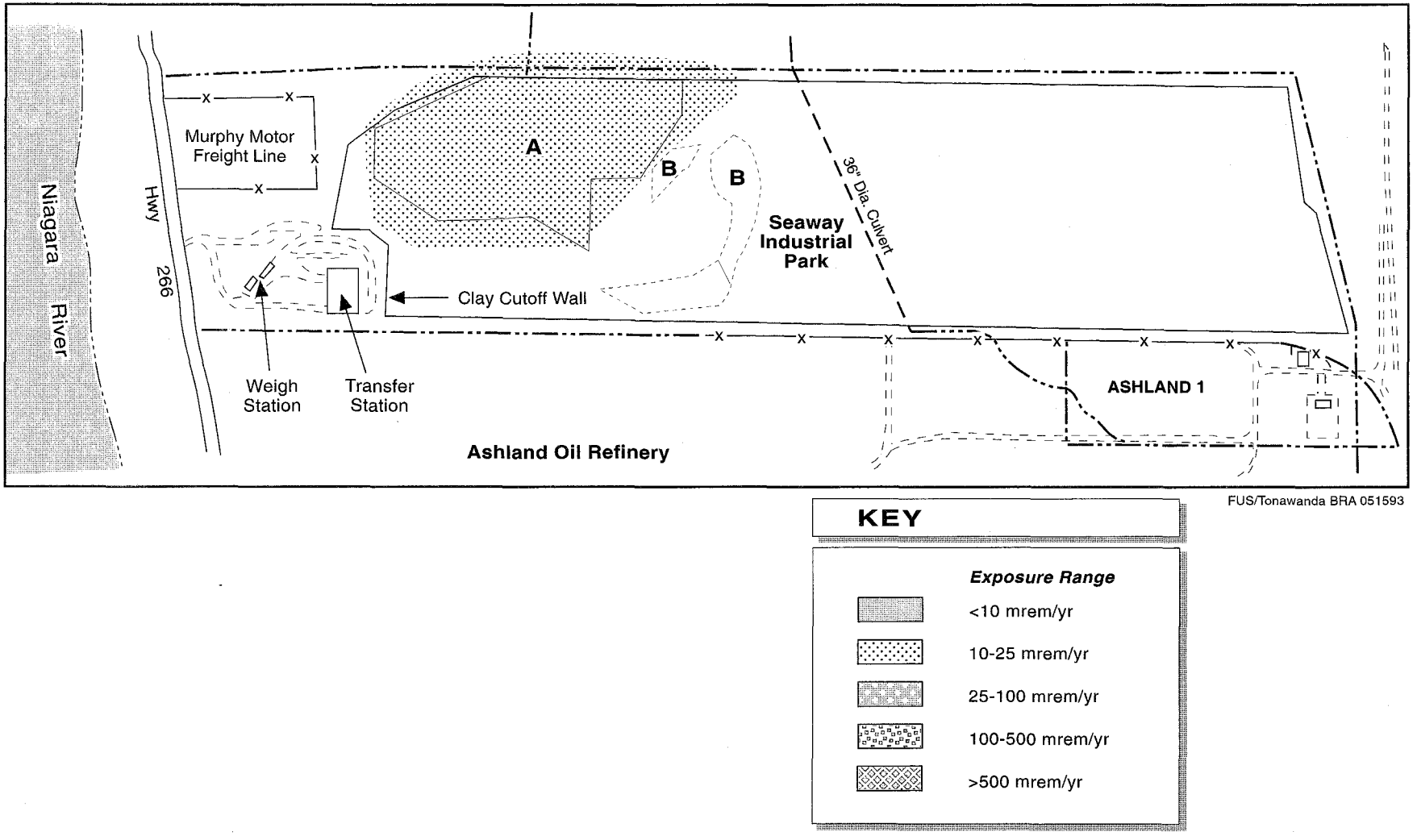


Figure 3-20. RME Annual Radiological Exposure in the Future Use Scenario at the Seaway Property

Table 3-1A. Mean Radionuclide Concentrations in Surface Soil, pCi/g

(LESS BACKGROUND)

LOCATION	PROPERTY UNITS	Th-232 +D*			Ra-226 +D		Th-230	U-238 +D		U-235 +D**		
		Th-232	Ra-228	Th-228	Ra-226	Pb-210	Th-230	U-238	U-234	U-235	Pa-231	Ac-227
LINDE	A	0.14	0.14	0.14	5.94	5.94	6.07	48.49	48.49	2.42	2.42	2.42
	B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
ASHLAND 1	A	0.00	0.00	0.00	0.57	0.57	6.45	2.61	2.61	0.13	0.13	0.13
	B	0.00	0.00	0.00	3.98	3.98	34.76	25.31	25.31	1.27	1.27	1.27
ASHLAND 2	A	0.21	0.21	0.21	2.06	2.06	18.98	7.59	7.59	0.38	0.38	0.38
	B	0.02	0.02	0.02	0	0	0.85	0.18	0.18	0.01	0.01	0.01
SEAWAY	A	0.00	0.00	0.00	3.23	3.23	1.13	3.35	3.35	0.17	0.17	0.17
	B	0.28	0.28	0.28	15.63	15.63	325.76	17.19	17.19	0.86	0.86	0.86
LOCAL CREEK	SEDIMENT	0.07	0.07	0.07	0.37	0.37	4.11	2.28	2.28	0.11	0.11	0.11
	SURFACE WATER	0.22	0.22	0.22	0.55	0.55	1.29	74.25	74.25	3.71	3.71	3.71

Shaded area indicates measured concentrations

* +D denotes that secular equilibrium was assumed to derive concentrations for associated decay products (non-shaded boxes)

** U-235 +D concentrations are 5% of U-238 value

ND = No Data

Table 3-1B. Mean Radionuclide Concentrations in Subsurface Soil, pCi/g

(LESS BACKGROUND)

LOCATION	PROPERTY UNITS	Th-232 +D*			Ra-226 +D		Th-230	U-238 +D		U-235 +D**		
		Th-232	Ra-228	Th-228	Ra-226	Pb-210	Th-230	U-238	U-234	U-235	Pa-231	Ac-227
LINDE	A	17.99	17.99	17.99	1.54	1.54	6.01	17.99	17.99	0.90	0.90	0.90
	B	6.23	6.23	6.23	0.52	0.52	1.34	6.23	6.23	0.31	0.31	0.31
ASHLAND 1	A	1.49	1.49	1.49	0.21	0.21	3.23	1.49	1.49	0.07	0.07	0.07
	B	86.36	86.36	86.36	11.51	11.51	363.81	86.36	86.36	4.32	4.32	4.32
ASHLAND 2	A	4.93	4.93	4.93	1.19	1.19	19.05	4.93	4.93	0.25	0.25	0.25
	B	0.06	0.06	0.06	0.02	0.02	ND	0.06	0.06	0.00	0.00	0.00
SEAWAY	A	4.64	4.64	4.64	2.76	2.76	26.30	4.64	4.64	0.23	0.23	0.23
	B	8.61	8.61	8.61	6.24	6.24	128.19	8.61	8.61	0.43	0.43	0.43

Shaded area indicates measured concentrations

* +D denotes that secular equilibrium was assumed to derive concentrations for associated decay products (non-shaded boxes)

** U-235 +D concentrations are 5% of U-238 value

ND = No Data

Table 3-1C. RME Radionuclide Concentrations in Surface Soil, pCi/g

(LESS BACKGROUND)

LOCATION	PROPERTY UNITS	Th-232 +D*			Ra-226 +D		Th-230	U-238 +D		U-235 +D**		
		Th-232	Ra-228	Th-228	Ra-226	Pb-210	Th-230	U-238	U-234	U-235	Pa-231	Ac-227
LINDE	A	0.26	0.26	0.26	8.34	8.34	8.96	77.44	77.44	3.87	3.87	3.87
	B	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
ASHLAND 1	A	0.06	0.06	0.06	0.79	0.79	11.72	3.94	3.94	0.20	0.20	0.20
	B	0.03	0.03	0.03	7.06	7.06	468.60	69.16	69.16	3.46	3.46	3.46
ASHLAND 2	A	0.39	0.39	0.39	3.25	3.25	54.03	12.04	12.04	0.60	0.60	0.60
	B	0.16	0.16	0.16	0.14	0.14	2.70	1.45	1.4505	0.07	0.07	0.07
SEAWAY	A	0.08	0.08	0.08	5.68	5.68	2.50	9.65	9.65	0.48	0.48	0.48
	B	0.76	0.76	0.76	22.91	22.91	878.60	25.46	25.46	1.27	1.27	1.27
LOCAL CREEK	SEDIMENT	0.32	0.32	0.32	0.54	0.54	11.66	5.19	5.19	0.26	0.26	0.26
	SURFACE WATER	0.53	0.53	0.53	0.93	0.93	3.86	143.70	143.70	7.18	7.18	7.18

Shaded area indicates measured concentrations

* +D denotes that secular equilibrium was assumed to derive concentrations for associated decay products (non-shaded boxes)

** U-235 +D concentrations are 5% of U-238 value

ND = No Data

Table 3-1D. RME Radionuclide Concentrations in Subsurface Soil, pCi/g

(LESS BACKGROUND)

LOCATION	PROPERTY UNITS	Th-232 +D*			Ra-226 +D		Th-230	U-238 +D		U-235 +D**		
		Th-232	Ra-228	Th-228	Ra-226	Pb-210	Th-230	U-238	U-234	U-235	Pa-231	Ac-227
LINDE	A	0.27	0.27	0.27	1.89	1.89	8.30	25.15	25.15	1.26	1.26	1.26
	B	0.08	0.08	0.08	0.95	0.95	3.06	12.04	12.04	0.60	0.60	0.60
ASHLAND 1	A	0.09	0.09	0.09	0.31	0.31	6.26	2.19	2.19	0.11	0.11	0.11
	B	0.36	0.36	0.36	16.66	16.66	1340.15	154.75	154.75	7.74	7.74	7.74
ASHLAND 2	A	0.14	0.14	0.14	1.50	1.50	34.62	6.17	6.17	0.31	0.31	0.31
	B	0.07	0.07	0.07	0.14	0.14	ND	0.02	0.02	0.01	0.01	0.01
SEAWAY	A	0.38	0.38	0.38	3.99	3.99	61.30	7.67	7.67	0.38	0.38	0.38
	B	0.28	0.28	0.28	10.31	10.31	407.33	13.05	13.05	0.65	0.65	0.65

Shaded area indicates measured concentrations

* +D denotes that secular equilibrium was assumed to derive concentrations for associated decay products (non-shaded boxes)

** U-235 +D concentrations are 5% of U-238 value

ND = No Data

TABLE 3-2. TOTAL EXPOSURE DOSE SUMMARY

		CURRENT USE SCENARIO (mrem/yr)			
LOCATION	SUBAREA	Employee		Transient	
		X	RME	X	RME
LINDE	A	15.38	26.98		
	B	0.58	1.23		
ASHLAND 1	A			0.02	0.43
	B			0.19	6.22
ASHLAND 2	A			0.07	3.04
	B			0.00	0.30
SEAWAY	A			0.10	2.76
	B*				
LOCAL CREEK	A			0.03	0.05
		FUTURE USE SCENARIO (mrem/yr)			
LOCATION	SUBAREA	Employee		Transient	
		X	RME	X	RME
LINDE	A	15.38	26.98		
	B	0.98	2.55		
ASHLAND 1	A	1.56	5.70		
	B	159.49	660.16		
ASHLAND 2	A	9.79	30.43		
	B	0.11	1.18		
SEAWAY	A			0.13	13.36
	B*				
LOCAL CREEK	A			0.03	0.05

X --- Mean; RME --- Reasonable Maximum Exposure
* --- No Pathways

4. TOXICITY ASSESSMENT

This section briefly summarizes the effects of ionizing radiation and chemicals on exposed populations.

4.1 RADIATION TOXICITY

The potential health effects associated with exposure to radionuclides at the Tonawanda site are due to low-level ionizing alpha, beta, and gamma radiation emitted by the members of the Th-232, U-238, and U-235 decay series (see Figures 2-1 through 2-3). Primarily, effects include an increase in the occurrence of cancer in irradiated individuals and possible genetic effects that may occur in future generations. The risk of serious genetic effects is much lower than the risk of cancer induction (EPA 1989d). Therefore, genetic effects are not the focus of this toxicity assessment, and radiological risks are evaluated only with respect to incremental cancer probabilities per EPA guidance (EPA 1989d). Non-radiological health effects of uranium are considered as appropriate in the chemical toxicity section.

Radiation-induced health effects for humans have been confirmed only at relatively high doses or high dose rates with large populations. For low doses, health effects are presumed to occur but can only be estimated statistically. Risk estimates are strictly applicable only to large populations, because the appearance of health effects after an exposure is a chance event. Predicting health effects with certainty for small populations (e.g., one person) is not possible. For purposes of radiological impact assessment, the health effects are measured by cancer incidence in the exposed population. However, risk estimates in the low-dose range are uncertain because of extrapolation from high doses and because of assumptions made on dose-effect relationships and the underlying mechanisms of carcinogenesis. Radiation effects in the exposed population cannot be readily identified since radiogenic cancers are indistinguishable from those occurring as a result of other factors. Studies of populations chronically exposed to low-level radiation, such as those residing in regions of elevated natural background, have not shown consistent evidence of an associated increase in the risk of cancer.

The exposure routes can be separated into either external or internal exposure. External exposure occurs when the radioactive material is outside the body. Internal exposure occurs when the radioactive material enters the body by routes such as inhalation or ingestion. Inhaled material can be exhaled, expelled from the lungs to be spit or swallowed and excreted, deposited in the lungs, or absorbed by the blood and relocated to systemic organs where it may be excreted over time. Some ingested material enters the blood and is either excreted in the urine or feces or relocated to other organs and excreted over time; most insoluble ingested material is not absorbed into the blood but is excreted directly in the feces. For internal exposures, alpha and beta particles are the dominant concern because their energy is absorbed in cells before the particles leave the body. Gamma rays are important primarily with respect to external exposure,

since for internal exposures they may leave the body without depositing a large fraction of their energy.

During the radioactive decay processes in the thorium, uranium, and actinium series, alpha, beta and gamma radiations are released. Each type of radiation differs in its physical properties and in its ability to induce damage to biological tissue. The BEIR IV report (NRC 1988) addresses the risk from radon and alpha radiations. Alpha particles are a hazard principally when taken into the body because, in external exposure, they are unable to penetrate the dead skin cell layer of the body before reaching living tissue. Within the body, alpha particles are the most effective of the three types of radiation in damaging cells because their energy is completely absorbed by tissue conventionally referred to as high linear energy transfer (LET). The BEIR V report (NRC 1990) addresses the risk from low linear energy transfer (LET) radiation such as gamma and beta particles. Beta particles are primarily an internal hazard. However, in cases of external skin exposure, energetic beta particles can penetrate living skin cells, representing an external hazard as well. Beta particles deposit less energy to small volumes of tissue than alpha particles and, therefore, induce much less damage than alpha particles. Gamma radiation is primarily an external hazard because it can penetrate tissue and reach internal organs without being taken into the body.

4.1.1 Radiation Toxicity Related to the Tonawanda Site

Exposure to a high dose of radiation (e.g., a thousand times the average annual background dose rate) during a short period of time (a few hours) affects all the organs and systems of the body. However, such acute exposures are not credible at the Tonawanda site. The only possible exposures at Tonawanda are chronic low-level exposures. Although lethal effects in human populations for chronic low-level exposure have never been documented, the effects have been projected from animal experiments. Studies assessing the difference between acute (short period) and chronic (long term) exposures show that radiation effects decrease dramatically as the period over which a given exposure is administered is extended (NRC 1990). Thus for sites like Tonawanda, where all exposures are longer term, it is likely that immediate effects would be observed. Rather the statistical impacts of possible increases in cancer or genetic changes are the only credible potential radiation effects (NRC 1990).

The radionuclides that occur at the Tonawanda site include Th-232, U-238, and U-235 and their progeny. The toxicity of the various radionuclides is based on:

- the types and energies of radiation they emit,
- the biological importance of the organs/tissues being irradiated,
- the radiological sensitivity of the organs/tissues being irradiated, and
- for internal exposure only, metabolic behavior in the body and biological retention characteristics in the body.

These factors were considered by the International Commission on Radiological Protection (ICRP), which established the concept of the committed effective dose equivalent (CEDE) to measure the detriment of exposure to radiation or radioactive materials. The CEDE value is calculated based on the models and criteria established by ICRP (e.g., ICRP 1977 and 1978) to allow quantification of this detriment, using all of the factors discussed above. Thus an estimate of risk from exposure to radiation or radioactive material may be made by determining the CEDE and multiplying by a dose-to-risk (e.g., cancer risk) conversion factor. The radiogenic cancer risk factor has been estimated by the National Academy of Sciences (NAS) in BEIR IV (NRC 1988) and BEIR V (NRC 1990). For detailed discussion of radiation dosimetry and toxicity, the reader is referred to publications of the ICRP (1977, 1978), NAS/NRC (1988, 1990), and UNSCEAR (1988).

4.1.2 Methods of Evaluating Radiation Toxicity

For this BRA, a risk factor of 6×10^{-7}/mrem was used to estimate the likelihood of cancer induction from radiation exposure. EPA used this risk factor to develop revisions to NESHAPs for radionuclides under Section 112 of the Clean Air Act (EPA 1989d). It is a lifetime average value and believed to be representative of conditions defined for the exposure scenarios at the Tonawanda site.

The BEIR V study (NRC 1990) also presents a detailed description of current data on the health risks associated with radiation exposure. A mortality risk factor of about 8×10^{-7}/mrem is estimated in the BEIR V report. However, not all radiation-induced cancers are fatal, (i.e., the cancer mortality rate is about 60 percent of the cancer induction rate given on average) (EPA 1989d). A cancer induction rate of about 1.3×10^{-6}/mrem for acute exposures can thus be inferred from the results presented in the BEIR V study. However, BEIR V estimates were derived primarily from data on acute exposures (a single instantaneous exposure), and the BEIR V report suggests that it is appropriate to reduce this risk by applying a dose rate effectiveness factor of two or more in cases of continuous low-level exposure. Thus, the radiation risk factor of 6×10^{-7} per mrem used in this report is consistent with the value recommended in BEIR V.

EPA also has developed guidance for radiological risk assessment that is generally consistent with existing guidance for assessing chemical carcinogenic risks, except that it consists of a two-phase (i.e., dual-endpoint) evaluation (EPA 1989d). For the first phase, radiation doses are calculated for all relevant radionuclides and pathways for the purpose of comparing CEDEs with established radiation protection standards and criteria. For the second phase, carcinogenic risks are calculated for the radionuclides of concern in a manner similar to existing methods for chemical carcinogens by using an age-averaged lifetime excess cancer incidence per unit intake (and per unit external exposure). To support this second evaluation, EPA has developed cancer incidence factors per unit intake that are analogous to the slope factors developed for chemical carcinogens. A preliminary evaluation indicates that estimates of potential health risk based on this approach would be less conservative than those presented in Appendix A (Tables A-5 to A-9).

In this BRA, the risk of cancer induction from inhalation of Rn-222 decay products has been estimated by converting Rn-222 exposure (in WLM) to mrem for CEDE. National Commission on Radiation Protection and Measurements (NCRP) report number 92 (NCRP 1987) indicates that one WLM is equal to about 14 rem. Weighting this by the 0.12 lung weighting factor (ICRP 1978) results in a CEDE of 1,000 mrem per WLM.

4.2 CHEMICAL TOXICITY

4.2.1 Chemical Contaminants of Concern

Chemical COCs in soil, surface water and sediment are identified in Section 2, Tables 2-5 through 2-11 (soils), Tables 2-12 through 2-25 (surface water, sediment). Chemical COCs are summarized by medium in Tables 2-27, 2-28. Toxicological properties of the COCs, including both carcinogenic and noncarcinogenic factors are summarized in Tables 4-1 and 4-2. The table also briefly describes routes of exposure, critical effects, and carcinogenic effects of the chemicals.

4.2.2 Methods of Evaluating Chemical Toxicity

Toxicity values utilized in the risk characterization of Tonawanda chemicals of concern are presented in Tables 4-1 and 4-2. These tables include supporting toxicological information along with source identifiers. Toxicity values utilized in Tonawanda risk calculations include the chronic reference dose (RfD) for noncarcinogenic risk and the oral slope factor (SF) for the carcinogenic risk.

The chronic RfD is defined as "an estimate of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime" (EPA 1989a). If the sum of the ratios of intake to RfD value (i.e., hazard indices) for all contaminants is less than one, this indicates that noncarcinogenic toxicity is unlikely. The SF is defined as a "plausible upper-bound estimate of the probability of a response (i.e., cancer) per unit intake of a chemical over a lifetime" (EPA 1989a). The SFs multiplied by the estimated lifetime intake levels yield lifetime cancer risk estimates. Both RfD and SF values are specific to the route of exposure (e.g., ingestion or inhalation exposure).

4.2.2.1 Chemicals for which EPA Toxicity Values are Available

The EPA Integrated Risk Information System (IRIS) database was used to provide up-to-date toxicity values to use in the Tonawanda risk calculations (EPA 1992b). When values were not available in IRIS, the 1992 EPA Health Effects Assessment Summary Tables (HEAST) were utilized (EPA 1992c). A chemical may be under review or re-examination by EPA according to IRIS and a value may be obtained from HEAST. When toxicity values were not available on IRIS or listed in HEAST, the Superfund Health Risk Technical Support Center - Environmental

Criteria and Assessment Office (SHRTSC-ECAO) was contacted (EPA 1992d). Provisional/interim values were obtained for these COCs if they were available.

EPA RfDs are available for 40 of the chemicals of concern. Oral SFs are available for 14 of the chemicals of concern. All polyaromatic hydrocarbons (PAHs) (6) used the oral SF for benzo(a)pyrene. Inhalation SFs and reference concentrations (RfCs) are available for six carcinogenic and nine noncarcinogenic chemicals of concern, respectively. As noted in Tables 4-1 and 4-2, the toxicity values for several chemical COCs have been withdrawn from IRIS or are currently under review by EPA and are not listed.

4.2.2.2 Chemicals For Which No EPA Toxicity Values Are Available

A number of the chemical COCs presently do not have RfDs for determination of potential noncarcinogenic health effects from oral and inhalation exposure. The possible impacts of the absence of the risk estimation for these contaminants is discussed in Section 5.3.

Carcinogenic effects are evaluated for these chemicals of concern in Section 5. Chemical toxicity data for the radioactive element thorium, and rare earth metals are not available in IRIS or found in HEAST. Therefore, thorium and rare earth metals were not carried through the chemical risk assessment.

As shown in Tables 4-1 and 4-2, EPA-derived RfD values incorporate uncertainty factors to account for data that were used but would not apply to chronic exposures in the most sensitive human subpopulations. In general, the use of these uncertainty factors provides confidence that exposure levels less than the RfD values are unlikely to cause toxic effects. However, the RfD values may actually be much lower than levels that will cause toxic effects in sensitive human subpopulations.

Table 4-1. Contaminants of Concern: Toxicity Values for Potential Carcinogenic Effects

Contaminants	CAS No.	Weight of Evidence Classification[a]	Slope Factor		Chemical Route	Type of Cancer/ Target Organ/Species	Source
			Oral $(mg/kg/day)^{-1}$	Inhalation $(mg/kg/day)^{-1}$			
Organics:							
Acenaphthene	83-32-9	ND	ND	ND	Oral	ND	IRIS
Acetone	67-64-1	ND	ND	ND	ND	ND	IRIS
Anthracene	120-12-7	D	ND	ND	Oral	ND	IRIS
Benzene	71-43-2	A	0.029	0.029	Inhalation/ Occupational	Leukemia/Blood/Humans	IRIS
Benzo(a)anthracene	56-55-3	B2	7.3[b]	ND	Oral	ND	IRIS
Benzo(b)fluoranthene	205-99-2	ND	ND	ND	Oral	ND	IRIS
Benzo(k)fluoranthene	207-08-9	B2	7.3[b]	ND	Oral	ND	IRIS
Benzo(g,h,i)perylene	191-24-2	D	ND	ND	Oral	ND	IRIS[*]
Benzo(a)pyrene	50-32-8	B2	7.3[b]	ND	Oral	Tumor/Forestomach /Mouse	IRIS
Bis(2-ethylhexyl)phthalate	117-81-7	B2	0.014	ND	Oral	Hepatocellular Carcinomas/Liver/Mouse	IRIS
Bromodichloromethane	75-27-4	B2	0.13	ND	Oral	Carcinomas/Liver/Mouse	IRIS
Bromoform	75-25-2	B2	0.0079	ND	Oral	Lesions/Intestine/Rat	IRIS
2-Butanone	78-93-3	ND	ND	ND	ND	ND	IRIS/HEAST
Chlorobenzene	108-90-7	D	ND	ND	ND	ND	IRIS
Chrysene	218-01-9	B2	7.3[b]	ND	Oral	ND	IRIS
Dibenzo(a,h)anthracene	53-70-3	B2	7.3[b]	ND	Oral	ND	IRIS
Dibenzofuran	132-64-9	D	ND	ND		ND	IRIS
Dibromochloromethane	124-48-1	C	0.084	ND	Oral	Carcinomas/Liver/Mouse	IRIS/HEAST

Table 4-1. (continued)

Contaminants	CAS No.	Weight of Evidence Classification[a]	Slope Factor		Chemical Route	Type of Cancer/ Target Organ/Species	Source
			Oral $(mg/kg/day)^{-1}$	Inhalation $(mg/kg/day)^{-1}$			
1,2-Dichloroethane	107-06-2	B2	0.091	ND	Oral	Carcoma/Circulatory System/Rat	IRIS/HEAST
Organics: (continued)							
T-1,2-Dichloroethene	156-60-5	ND	ND	ND	ND	ND	IRIS
1,2-Dichloropropane	78-87-5	ND	ND	ND	ND	ND	IRIS
T-1,3-Dichloropropene	542-75-6	ND	ND	ND	ND	ND	IRIS/HEAST
2,4-Dimethylphenol	ND	ND	ND	ND	ND	ND	IRIS
Di-n-butyl phthalate	84-74-2	D	ND	ND	ND	ND	IRIS
Fluoranthene	206-44-0	D	ND	ND	ND	ND	IRIS
Fluorene	86-73-7	ND	ND	ND	ND	ND	IRIS
Indeno(1,2,3-cd)pyrene	193-39-5	B2	7.3[b]	ND	ND	ND	IRIS
Methylene chloride	75-09-2	B2	0.0075	ND	Oral/ Inhalation	Carcinomas/Liver/Mouse	IRIS
2-Methylnaphthalene	91-57-6	ND	ND	ND	ND	ND	IRIS*
4-Methylphenol	106-44-5	ND	ND	ND	ND	ND	IRIS
Napthalene	91-20-3	D	ND	ND	ND	ND	IRIS
Phenanthrene	85-01-8	D	ND	ND	ND	ND	IRIS
Phenol	108-95-2	D	ND	ND	ND	ND	IRIS
Pyrene	129-00-0	D	ND	ND	Oral	ND	IRIS
Toluene	108-88-3	D	ND	ND	ND	ND	IRIS
Xylenes (total)	1330-20-7	D	ND	ND	ND	ND	IRIS

Table 4-1. (continued)

Contaminants	CAS No.	Weight of Evidence Classification[a]	Slope Factor		Chemical Route	Type of Cancer/ Target Organ/Species	Source
			Oral $(mg/kg/day)^{-1}$	Inhalation $(mg/kg/day)^{-1}$			
Inorganics:							
Aluminum	7429-90-5	ND	ND	ND	ND	ND	IRIS*/HEAST
Antimony	7440-36-0	D	ND	ND	ND	ND	IRIS/SHRTSC
Arsenic	7440-38-2	A	1.75	50	Oral/ Inhalation	Tumors/Lung/Human	IRIS
Barium	7440-39-3	ND	ND	ND	ND	ND	IRIS
Boron	7440-42-8	ND	ND	ND	ND	ND	IRIS
Beryllium	7440-41-7	B2	4.3	8.4	Oral/ Inhalation	Tumors/Lung/Human	IRIS
Cadmium	7440-42-8	B1	ND	6.1	Inhalation	Lungs, Trachea, Bronchus/Human	IRIS
Calcium	7440-70-2	ND	ND	ND	ND	ND	IRIS*
Chromium (VI)	18540-29-9	A	ND	41	Inhalation	Tumors/Lung/Human	IRIS*/HEAST
Cobalt	7440-48-4	ND	ND	ND	ND	ND	IRIS/SHRTSC
Copper	7440-50-8	D	ND	ND	ND	ND	IRIS
Lead[c]	7439-92-1	B2	ND	ND	ND	ND	IRIS
Magnesium	7439-93-2	ND	ND	ND	ND	ND	IRIS*
Manganese	7439-96-5	ND	ND	ND	ND	ND	IRIS
Mercury	7439-97-6	ND	ND	ND	ND	ND	IRIS
Molybdenum	7439-98-7	ND	ND	ND	ND	ND	IRIS
Nickel	7440-02-0	ND	ND	0.84	Inhalation	Tumors/Respiratory System/Humans	IRIS/HEAST
Potassium	7440-09-7	ND	ND	ND	ND	ND	IRIS

Table 4-1. (continued)

Contaminants	CAS No.	Weight of Evidence Classification[a]	Slope Factor		Chemical Route	Type of Cancer/ Target Organ/Species	Source
			Oral $(mg/kg/day)^{-1}$	Inhalation $(mg/kg/day)^{-1}$			
Inorganics: (continued)							
Selenium	7784-49-2	ND	ND	ND	ND	ND	IRIS
Silver	7440-22-4	ND	ND	ND	ND	ND	IRIS
Sodium	7440-23-5	ND	ND	ND	ND	ND	IRIS*/HEAST
Thallium	7440-28-0	ND	ND	ND	ND	ND	IRIS*
Vanadium	7440-62-2	ND	ND	ND	ND	ND	IRIS
Zinc	7440-66-6	D	ND	ND	ND	ND	IRIS

Source: IRIS, 1992. Integrated Risk Information System.
HEAST, 1992. Health Effects Assessment Summary Tables.
SHRTSC-ECA0, 1992. Superfund Health Risk Technical Support Center - Environmental Criteria and Assessment Office.

ND = No data.
* = not available on IRIS.
a A = Human carcinogen (sufficient evidence of carcinogenicity in humans);
B1 = Carcinogen (limited evidence of carcinogenicity in humans);
B2 = Probable human carcinogen (sufficient evidence of carcinogenicity in animals, with inadequate or lack of evidence of carcinogenicity in humans);
C = Possible human carcinogen (limited evidence of carcinogenicity in animals, and inadequate or lack of evidence of human data);
D = Not classifiable as to human carcinogenicity.
b Carcinogenicity assessment for all PAHs is based on slope factor data for benzo(a)pyrene (EPA 1989).
c A Lead Uptake/Biokinetic Model is currently under review by EPA.

Table 4-2. Contaminants of Concern: Toxicity Values for Potential Noncarcinogenic Effects

Contaminants	CAS No.	Chronic		Confidence Level	Chemical Route/ Critical Effect	Modifying Factors	Source
		Oral RfD (mg/kg/day)	Inhalation RfC (mg/m^3)[a]				
Organics:							
Acenaphthene	83-32-9	0.06	ND	Low	Hepatotoxicity	UF = 3000 MF = 1	IRIS
Acetone	67-64-1	0.1	ND	Low	Increased weight/nephrotoxicity	UF = 1000 MF = 1	IRIS/HEAST
Anthracene	120-12-7	0.3	ND	Low	No observed effect	UF = 3000 MF = 1	IRIS
Benzene	71-43-2	UR	0.0002	Medium	Inhalation/Hematological immunological effects	UF = 300	IRIS/SHRTSC
Benzo(a)anthracene	56-55-3	ND	ND	ND	ND	ND	IRIS
Benzo(b)fluoranthene	205-99-2	ND	ND	ND	ND	ND	IRIS
Benzo(k)fluoranthene	207-08-9	ND	ND	ND	ND	ND	IRIS
Benzo(g,h,i)perylene	191-24-2	ND	ND	ND	ND	ND	IRIS
Benzo(a)pyrene	50-32-8	ND	ND	ND	ND	ND	IRIS
Bis(2-ethylhexyl)phthalate	117-81-7	0.02	ND	Medium	Oral/Increased relative liver weight	UF = 1000 MF = 1	IRIS
Bromodichloromethane	75-27-4	0.02	ND	Medium	Oral/Renal cytomegaly	UF = 1000 MF = 1	IRIS
Bromoform	75-25-2	0.02	ND	Medium	Oral/Hepatic Lesions	UF = 1000 MF = 1	IRIS
2-Butanone	78-93-3	0.05	1	Low	Oral/Inhalation/Decreased fetal weight	UF = 1000 MF = 3	IRIS/HEAST

Table 4-2. (continued)

Contaminants	CAS No.	Chronic		Confidence Level	Chemical Route/ Critical Effect	Modifying Factors	Source
		Oral RfD (mg/kg/day)	Inhalation RfC (mg/m^3)[a]				
Chlorobenzene	108-90-7	0.02	UR	Medium	Oral/Histopathologic changes in liver	UF = 1000 MF = 1	IRIS
Chrysene	218-01-9	ND	ND	ND	ND	ND	IRIS
Dibenzo(a,h)anthracene	53-70-3	ND	ND	ND	ND	ND	IRIS
Dibenzofuran	132-64-9	0.004[b]	UR	Low	Oral/Renal effects	UF = 3000 MF = 1	IRIS/SHRTSC
Dichlorobromomethane	124-48-1	0.02	ND	Medium	Oral/Hepatic Lesions	UF = 1000 MF = 1	IRIS
1,2-Dichloroethane	107-06-2	ND	ND	ND	ND	ND	IRIS
T-1,2-Dichloroethene	156-60-5	0.02	ND	Low	Oral/Increased serum alkaline phosphatase	UF = 1000 MF = 1	IRIS
1,2-Dichloropropane	78-87-5	ND	0.004	ND	Inhalation/Hyperplasia of nasal mucosa	UF = 300 MF = 1	IRIS
T-1,3-Dichloropropene	542-75-6	0.0003	0.02	Low	Oral/Increased organ weights inhalation/hypertrophy, hyperplasia of the nasal respiratory epithelium	UF = 10,000, MF = 1; UF = 30, MF = 1	IRIS
2,4-Dimethylphenol	105-67-9	0.02	ND	Low	Clinical signs and hematological changes	UF = 3000 MF = 1	IRIS
Di-n-butyl phthalate	84-74-2	0.1	UR	Low	Increased mortality	UF = 1000 MF = 1	IRIS
Fluoranthene	206-44-0	0.04	ND	Low	Oral/Nephropathy, increased liver weight, hematological alterations	UF = 3000 MF = 1	IRIS

Table 4-2. (continued)

Contaminants	CAS No.	Chronic		Confidence Level	Chemical Route/ Critical Effect	Modifying Factors	Source
		Oral RfD (mg/kg/day)	Inhalation RfC (mg/m^3)[a]				
Fluorene	86-73-7	0.04	ND	Low	Oral/Decreased red blood cells	UF = 3000 MF = 1	IRIS
Indeno(1,2,3-cd)pyrene	193-39-5	ND	ND	ND	ND	ND	IRIS
Methylene chloride	75-09-2	0.06	UR	Medium	Oral/Liver toxicity	UF = 100 MF = 1	IRIS
2-Methylnaphthalene	91-57-6	ND	ND	ND	ND	ND	IRIS
4-Methylphenol	106-44-5	ND	ND	ND	ND	ND	IRIS
Naphthalene	91-20-3	0.04 (UR)	0.0013	Medium	Inhalation/Lesion in the lungs and nasal cavity	UF = 1000 MF = 1	IRIS/SHRTSC
Phenanthrene	85-01-8	ND	ND	ND	ND	ND	IRIS
Phenol	108-95-2	0.6	ND	Low	Oral/Reduced fetal body weight	UF = 100 MF = 1	IRIS
Pyrene	129-00-0	0.03	ND	Low	Oral/Kidney effects	UF = 3000 MF = 1	IRIS
Toluene	108-88-3	0.2	0.4	Medium/ Medium	Oral/Increased kidney and liver weights/neurological effects	UF = 1000 MF = 1; UF = 300 MF = 1	IRIS
Xylene (total)	1330-20-7	2.0	UR	Medium	Oral/Hyperactivity, decreased body weight, increased mortality	UR = 100 MF = 1	IRIS
Inorganics:							
Aluminum	7429-90-5	1[b]	ND	Medium	Oral/Decreased body weight, neurotoxicity	ND	IRIS/SHRTSC
Antimony	7440-36-0	0.0004	ND	Low	Oral/Longevity, blood glucose, cholesterol	UF = 1000 MF = 1	IRIS

Table 4-2. (continued)

Contaminants	CAS No.	Chronic		Confidence Level	Chemical Route/ Critical Effect	Modifying Factors	Source
		Oral RfD (mg/kg/day)	Inhalation RfC (mg/m^3)[a]				
Arsenic	7440-38-2	0.0003	ND	Medium	Oral/Hyperpigmentation, keratosis, vascular complications	UF = 3 MF = 1	IRIS
Barium	7440-39-3	0.07	UR	Medium	Oral/Increased blood pressure	UF = 3 MF = 1	IRIS
Beryllium	7440-41-7	0.005	ND	Low	No adverse effects	UF = 100 MF = 1	IRIS
Boron	7440-42-8	0.09	ND	Medium	Oral/Testicular atrophy, spermatogenic arrest	UF = 100 MF = 1	IRIS
Cadmium	7440-43-9	0.0005 (water) 0.001 (food)	UR	High	Oral/Significant Proteinuria	UF = 10 MF = 1	IRIS
Calcium	7440-70-2	ND	ND	ND	ND	ND	IRIS
Chromium (VI)	18540-29-9	0.005	UR	Low	Oral/No observed effects	UF = 500 MF = 1	IRIS
Cobalt	7440-48-4	0.96 adult[b] 0.06 child[b]	ND	ND	Oral/Respiratory effects	ND	IRIS/SHRTSC
Copper	7440-50-8	0.04[c]	ND	ND	Oral/Hepatic necrosis	ND	IRIS/SHRTSC
Iron	7439-89-6	ND	ND	ND	Oral/Hepatic effects	ND	IRIS
Lead[d]	7439-92-1	UR	ND	ND	ND	ND	IRIS
Magnesium	7439-93-2	ND	ND	ND	ND	ND	IRIS
Manganese	7439-96-5	0.1	0.004	ND	Oral, Inhalation/Respiratory and CNS effects	UF = 900	HEAST
Mercury	7439-97-6	0.0003	0.0003	ND	Oral, Inhalation/renal effects, CNS effects	UF = 1000; UF = 30	IRIS/HEAST

Table 4-2. (continued)

Contaminants	CAS No.	Chronic		Confidence Level	Chemical Route/ Critical Effect	Modifying Factors	Source
		Oral RfD (mg/kg/day)	Inhalation RfC (mg/m^3)[a]				
Molybdenum	7439-98-7	0.005	ND	ND	Oral/Pain and swelling in joints	UF = 30	IRIS/SHRTSC
Nickel	7440-02-0	0.02	0.0006	Medium	Oral/Inhalation/Decreased body and organ weights, tumors in respiratory system	UF = 300 MF = 1	IRIS/SHRTSC
Potassium	7440-09-7	ND	ND	ND	ND	ND	IRIS
Selenium	7782-49-2	0.005	ND	High	Oral/Clinical selenosis	UF = 3 MF = 1	IRIS
Silver	7440-22-4	0.005	ND	Low	Oral/Argyria	UF = 3 MF = 1	IRIS
Sodium	7440-23-5	ND	ND	ND	ND	ND	IRIS
Thallium	7440-28-0	ND	ND	ND	ND	ND	IRIS
Uranium	7440-61-6	0.003	ND	ND	Oral/Initial body weight loss/ Moderate nephrotoxicity	UF = 1000 MF = 1	IRIS
Vanadium	7440-62-2	0.007 (UR)	ND	ND	None observed	UF = 100	IRIS/HEAST
Zinc	7440-66-6	UR	ND	ND	ND	ND	IRIS

Source: IRIS, August 1992. Integrated Risk Information System.
HEAST, 1992. Health Effects Assessment Summary Tables.
SHRTSC-ECAO, 1992. Superfund Health Risk Technical Support Center - Environmental Criteria and Assessment Office.

[a] = EPA SHRTSC-ECAO does not recommend conversion to a dose equivalent due to the potential for inaccuracy.
[b] = Provisional/interim values provided by SHRTSC-ECAO.
[c] = An interim number (most conservative provided by SHRTSC (an Rfd between 0.04 and 0.07 is recommended).
[d] = A Lead Uptake/Biokinetic Model is currently under review by EPA.
NA = not available.
ND = no data.
UR = Toxicity values currently under review by EPA work group.
* = not available on IRIS.

5. RISK CHARACTERIZATION

This section presents the current and future risk projections for human receptors at the Tonawanda site. Human receptors include workers, transient adults and children, and recreational users. Radiological risks and chemical risks are estimated separately.

For the radiological assessment, carcinogenic risk is defined as the incremental lifetime probability of cancer morbidity and does not include genetic or noncarcinogenic effects. Cancer risk estimates and hazard index (HI) estimates are presented, as appropriate, for the chemical COCs where toxicity values are available. Cancer risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of pathway-specific exposure to carcinogenic contaminants.

EPA does not presently use a probabilistic approach to estimate the potential for noncarcinogenic health effects (EPA 1989a). Instead, the potential for noncarcinogenic effects is evaluated by comparing an exposure level over a specified time period (exposure duration) with a reference dose derived for a similar exposure period (EPA 1989a). This ratio of exposure is called a hazard quotient (HQ). HQs for each COC are then summed to obtain an HI for the specific pathway. An HI greater than one has been defined as the level of concern for potential adverse noncarcinogenic health effects (EPA 1989a).

5.1 RISK CHARACTERIZATION METHODOLOGY AND ASSUMPTIONS

Assumptions describing risk characterization methodology are described in the following sections. The assumptions are more completely described in Appendix B.

5.1.1 Radiological Risks

Exposures to low levels of ionizing radiation could result in cancer induction, serious genetic effects, and other detrimental health effects. The predominant health concern associated with the radioactive contaminants at the Tonawanda site is the induction of cancer. The radiological health risks presented in this BRA are limited to this concern. This approach is consistent with EPA guidance, which notes that, generally, the risk of cancer is limiting and may be used as the sole basis for assessing the radiation-related human health risks for a site contaminated with radionuclides (EPA 1989a).

Risk from exposure to radioactive contaminants was estimated following EPA (EPA 1989d), BEIR IV (NRC 1988), and BEIR V (NRC 1990) recommendations. As discussed in Section 4, for the purposes of this BRA, a population-weighted average excess risk of cancer of 6×10^{-7} per mrem was assumed. The radiation doses associated with the scenarios considered in this assessment are presented in Section 3. These doses are expressed as committed effective dose equivalent resulting from a one-year exposure, in millirem/yr, for all exposure routes. The

risk factor, the annual exposure in millirem, and the number of years of exposure are multiplied to obtain estimates of lifetime cancer morbidity risk.

EPA cancer slope factors (SFs) as presented in the 1992 HEAST tables also were used to assess radiological risk. A comparison between EPA SF methodology and the conventional approach (dose x risk) indicated a reasonable agreement between the two methods for predicted risk (Appendix A). The dose/risk method was selected for presentation in this BRA because it allows for the maximum use of site-specific exposure information, which reduces uncertainty associated with the assessment, and is consistent with the approach mandated in DOE Order 5400.5 (DOE 1990).

The radiological risks associated with exposures to contaminants at the Tonawanda site are to be considered an addition to the risks from exposure to natural sources of radiation. Radiation exposure from natural sources of radioactivity results in an annual dose of about 300 mrem/yr: 200 mrem/yr from exposure to Rn-222 and its short-lived decay products, and 100 mrem/yr from exposure to other natural and man-made sources of radiation (NCRP 1987). Using the corresponding risk factors given above, this background dose results in a lifetime risk of cancer induction of approximately 1.3 percent (1.3×10^{-2}). EPA has estimated that the individual lifetime risk of fatal cancer associated with background radiation, including radon, is 1×10^{-2} (EPA 1989a).

5.1.2 Chemical Risks and Hazard Quotients

5.1.2.1 Cancer Risks

The risk to an individual resulting from exposure to chemical carcinogens is expressed as the increased probability of a cancer occurring over the course of a lifetime. To calculate the excess cancer risk, the estimated daily intake, averaged over a lifetime, is multiplied by a chemical-specific SF. Oral and inhalation pathway-specific SFs have been derived by EPA for certain carcinogens; some carcinogens do not have an SF available or are presently under review by EPA. All SFs utilized in the risk estimate calculations were obtained from EPA's IRIS (EPA 1992b) or, where not available on IRIS, were obtained from EPA's Health Effects Assessment Summary Tables (HEAST) (EPA 1992a). If SFs were not available from IRIS or HEAST, EPA's Superfund Health Risk Technical Support Center-ECAO was contacted and interim or provisional SFs obtained for use in the risk characterization (EPA 1992d).

The SF converts estimated daily intakes averaged over a lifetime of exposure directly to the incremental risk of an individual developing cancer (EPA 1989a). The carcinogenic risk estimate is generally an upper-bound estimate because the SF is often an upper 95 percentile confidence limit of the probability of response based on experimental animal data (EPA 1989a). Thus, EPA is reasonably confident that the "true risk" will not exceed the risk estimate derived through use of the SF and is likely to be less than that predicted. (EPA 1989a). The estimation of daily intakes (averaged over a lifetime) resulting from exposure to the chemical carcinogens of concern was described in Section 3.4, and available SFs were identified in Section 4.2.

5.1.2.2 Hazard Quotients and Hazard Indexes

The potential for adverse health effects other than cancer is evaluated as the ratio of the daily intake for the exposure period over the RfD (or RfC for inhalation exposure); this ratio is the HQ. The RfD is a provisional estimate of the daily exposure to the human population, including sensitive subgroups (with uncertainty spanning perhaps an order of magnitude), without an appreciable risk of deleterious health effects during a lifetime for chronic exposure, or during a portion of a lifetime for subchronic exposure (EPA 1992b). EPA has derived RfDs and RfCs for both chronic and subchronic exposure periods. In accordance with guidance for Superfund, chronic exposures for humans range in duration from seven years to a lifetime; and subchronic human exposures range in duration from two weeks to seven years (EPA 1989a). Because the potential exposures considered in this BRA are for periods of more than seven years, only chronic RfDs and RfCs are considered. The estimated average daily intakes resulting from exposure to the chemical COCs at the site were presented in Appendix B, and the RfDs and RfCs for these contaminants were identified in Section 4.2.

The noncancer HQ assumes that there is a level of exposure (the RfD or RfC, as appropriate) below which it is unlikely for even sensitive populations to experience adverse noncarcinogenic health effects (EPA 1989a). If the intake exceeds this threshold (i.e., Intake/RfD or RfC exceeds unity or 1), there may be concern for potential noncarcinogenic effects (EPA 1989a). The greater the ratio (intake/RfD or RfC), the greater the level of concern (EPA 1989a). The HQs for each chemical addressed in the intake and exposure pathway are summed to obtain the HI, which allows assessment of the overall potential for noncarcinogenic effects (EPA 1989a). When the HI exceeds unity (1), there may be concern for potential adverse health effects. For exposure to multiple chemicals, as at Tonawanda, the summed HI which exceeds unity indicates a potential health risk, even if no single chemical exposure exceeds its RfD ($HQ < 1$).

The assumption of dose additivity is most properly applied to chemicals that induce the same effect by the same mechanism of action (EPA 1989b). When the HI exceeds unity as a result of summing several HQs, it is appropriate to segregate the chemicals by effect and by mechanism of action.

5.2 RISK ESTIMATES FOR THE TONAWANDA SITE

For clarity of presentation, the risk estimates resulting from potential radiological and chemical exposures are presented separately in the following sections. Exposure estimates are presented for each exposure scenario for the most probable exposure conditions (mean receptor) and the reasonable maximum exposure conditions (RME receptor).

5.2.1 Radiological Risk Estimates

The radiological risks for the Tonawanda site are presented in shaded maps for all scenarios and receptors in Figures 5-1 through 5-13. The risk ranges shown in the maps include the 10^{-4} to 10^{-6} target range specified by EPA as generally acceptable. Within this risk range, remedial action may be selected based on protection of human health and the environment and compliance with applicable or relevant and appropriate requirements (ARARs). Accordingly, the risk ranges presented on the maps are $\leq 10^{-6}$, 10^{-5}, 10^{-4}, 10^{-3}, and $> 10^{-3}$.

Potential risks as a result of exposure to contaminants found at the Tonawanda site were estimated for reasonable current uses and hypothetical future uses of the site properties. Radiological risk estimates are discussed in Section 5.2.1.1 for current use and in Section 5.2.1.2 for future use.

The potential receptors and routes of exposure to contamination at each subarea comprising the Tonawanda site are summarized in Section 3.2. Exposure point concentrations and doses are presented in Sections 3.4 and 3.5. The estimates of radiological risk consider exposure to contaminated soil, sediment, and indoor and outdoor air.

Contaminated soil has been identified in various areas at the Tonawanda site, as indicated by the characterization and environmental monitoring results. Air is considered because of the potential for transport of airborne radioactive particulates from contaminated soil, radon gas from radium contaminated soil, and external gamma irradiation from contaminated soil.

5.2.1.1 Current Use Scenarios

Risk estimates for potential exposure from current site use are presented in Table 5-1. The estimated radiological risks for the mean and RME exposures are within the EPA target risk range (10^{-4} to 10^{-6}) for all receptors and all scenarios. The highest estimated risks are for the hypothetical RME employee at Linde Subarea A with an estimated risk of 4×10^{-4}.

Gamma irradiation generally contributes 63 to 98 percent of the radiological risk to receptors in the different scenarios. The gamma irradiation contribution to Linde employees ranges from 45 percent of the total risk for mean at Subarea B to 88 percent for the RME conditions at Subarea A. The remainder of the employee risk is derived from the radon exposure pathway. The particulate inhalation and ingestion pathways contribute an insignificant amount to overall risk.

In the transient scenario, 97 to 76 percent of the radiological risk results from direct gamma irradiation for the mean and RME conditions, respectively.

The current use scenario estimated mean carcinogenic risk for employee receptors at the Linde property were 7×10^{-5} and 3×10^{-6} for Subareas A and B, respectively. RME risks for the same Subareas were 4×10^{-4} and 2×10^{-5}. The mean risks to transients range from 1×10^{-6}

to 5×10^{-9}. RME risks for transients range from 1×10^{-4} to 5×10^{-6}. The mean risk to a child wading in the creeks is 2×10^{-7}, the RME risk is 9×10^{-7}.

5.2.1.2 Hypothetical Future Use Scenarios

Risk estimates for potential exposure from hypothetical future property use are presented in Table 5-1. The estimated radiological risks for mean receptors at all property units except Ashland 1 Subarea B are within the EPA target risk range. RME risks at Ashland 1 Subarea B and Ashland 2 Subarea A also exceed the EPA target risk range. Dominant exposure pathway risks in the future use scenarios are similar to those in the current use scenarios in that direct gamma irradiation contributes the bulk of the risk to the receptors.

Since the majority of the estimated mean carcinogenic risk for employee receptors is attributed to direct gamma irradiation (which is estimated from surface soil concentrations and is assumed to remain unchanged in the future), the excess carcinogenic risk for the future employee scenario at Linde Subarea A is the same as that under current use. The risk for the future employee scenario at Linde Subarea B increases, but still remains within the EPA target range.

The risks to future employees on properties subject to land use changes (i.e., from vacant property to commercial use) are estimated to range from 7×10^{-4} to 4×10^{-7} (mean) and 1×10^{-2} to 2×10^{-5} (RME). The risks to the wading children are expected to remain constant.

5.2.1.3 Risk to Offsite Receptors

Risk to the population within an 80-km (50-mi) radius of the Tonawanda site was found to be insignificant when compared to the background incidental cancer rate. This population risk evaluation is intended for use in as low as reasonably achievable (ALARA) evaluations consistent with the requirements of DOE Order 5400.5 (DOE 1990) and the implementing guidance for remediation activities (Gilbert et al. 1989). This information also can be used to support the remedial action decision based on impacts to the surrounding community. DOE Order 5400.5 (DOE 1990) mandates that DOE implement the ALARA process in all activities. The ALARA process requires that after the applicable radiation protection limits (e.g., dose) are met, the dose/risk shall be further reduced as low as reasonably achievable, taking into account technical, economic, and social factors.

5.2.2 Chemical Risk and Hazard Index Estimates

Estimates of risk to site receptors resulting from exposure to chemical carcinogens are presented in Table 5-2, expressed as the increased probability of a cancer occurring over the course of a lifetime. Estimates are presented for both most probable exposure conditions (mean) and RME conditions.

Chemical-specific intakes and carcinogenic risks are tabulated in Appendix C. Risks could be estimated only for those COCs with a toxicity value currently available from IRIS (EPA 1992b) or HEAST (EPA 1992a), or from interim or provisional values available from the Superfund Health Risk Technical Support Center - ECAO and EPA Region II (EPA 1992c). The polycyclic aromatic hydrocarbons (PAHs) are grouped as a class of chemicals for consideration of carcinogenic risk (EPA 1989a). The oral SF for benzo(a)pyrene is utilized in the risk calculations for all of the carcinogenic PAHs (EPA 1989a). The class of PAHs includes acenaphthene, anthracene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)-fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, fluoranthene, fluorene, indeno(1,2,3-cd)pyrene, naphthalene, phenanthrene, and pyrene (ATSDR 1990). Total pathway and total site carcinogenic risks for both RME and average exposure are presented in Table 5-2.

The potential for adverse noncarcinogenic health effects is expressed as chemical-specific HQs, which are tabulated in Appendix C. The HQs were tabulated for all COCs where reference doses are currently available (EPA 1992b; EPA 1992a; EPA 1992c). The HQs are summed for each pathway to provide a total HI for the pathway. The pathway-specific and total pathway HIs are presented in Table 5-3.

Current receptors considered in the assessment of chemical risks at the Tonawanda site are employees at Linde, transient visitors to the Ashland 1 and Ashland 2 properties, and children playing in the creeks. No data were available for Seaway. Therefore, no risks have been projected. Future receptors include employees, transients, and children playing in the creeks. Based on available data from the RI report (BNI 1992), potential cancer risk and noncancer health hazards were estimated for reasonable current uses and hypothetical future uses (Tables 5-2 and 5-3).

5.2.2.1 Current Use Scenarios

Under the current use scenario, the mean and RME carcinogenic risks for employee receptors at Linde were 2×10^{-5} and 8×10^{-5}, respectively (Table 5-2). These risks are associated primarily with the ingestion of arsenic. The carcinogenic risks associated with the inhalation of particulates by employees at Linde were 3×10^{-8} and 7×10^{-7}, respectively. None of the estimated cancer risks exceeded the EPA target range. Other pathways of exposure at Linde were considered incomplete.

The mean carcinogenic risks for current transients, due to soil ingestion, at Ashland 1 and Ashland 2 were 2×10^{-7} and 2×10^{-7}, respectively. RME risks were 3×10^{-6} and 2×10^{-6}. The mean risks associated with the inhalation of particulates by transients at Ashland 1 and Ashland 2 were 2×10^{-12} and 1×10^{-10}, respectively. RME risks were 3×10^{-10} and 1×10^{-8}. None of the estimated cancer risks exceeded the EPA target range.

The mean risks from surface water and sediment ingestion, by children wading in a local creek, were 4×10^{-7} and 8×10^{-8}, respectively. RME risks were 8×10^{-7} and 2×10^{-7}.

The HIs for current employees and transients at Linde, Ashland 1, and Ashland 2 were all less than one (Table 5-3). Mean and RME values for surface water ingestion at the local creek were 2×10^{-2} and 7×10^{-2}, respectively. These values do not exceed unity and do not indicate a concern for potential adverse health effects.

5.2.2.2 Hypothetical Future Use Scenarios

Since land use and contaminant concentrations were assumed to remain unchanged, the carcinogenic and noncarcinogenic risks to hypothetical future employees at the Linde property and the child wading in the local creek are the same as estimated from current use scenarios. Land use at Ashland 1 and 2 is assumed to change to commercial. Carcinogenic risks to the hypothetical future employees are within the EPA target range. Noncarcinogenic risks reach a maximum of 0.1 at Ashland 2 (RME).

5.3 UNCERTAINTY RELATED TO RISK ESTIMATES

The evaluation of radiological and chemical risks to human health presented in this BRA was, by necessity, based on a number of assumptions. In addition, many uncertainties are inherent to the risk assessment process. This section provides additional discussion of the rationale for the major assumptions used in this assessment and associated uncertainties, in order to address their potential impact on the results contained herein. Uncertainties for ecological risk assessment are discussed in Section 6.4.3.

5.3.1 Uncertainty in Radiological Risk Estimates

Uncertainty is inherent in the selection of COCs for a BRA and is associated with a number of factors. First, limitations in data relative to locations and analytes sampled and to analytical considerations (e.g., laboratory procedures) may affect the contaminants identified for a site. The uncertainty associated with the site sampling data is considered to be low because the sampling plans generally targeted appropriate areas and analytes using historical information, visual observations, and both phased and biased characterization strategies. Uncertainty relative to sample analysis and data evaluation is also considered low because an extensive, site-specific quality assurance program has been implemented and is ongoing.

5.3.1.1 Identification of Contaminants of Concern

The identification of COCs for a human health evaluation relies on both information from site characterization activities and the application of a selection process. Considerable data have been collected for the site under both DOE's environmental monitoring program and the site characterization effort. The COC selection process was designed using EPA guidance to identify those contaminants that contribute most to the estimates of excess risks.

The contaminant selection screening may also introduce uncertainty. The estimated health effects could be higher if all compounds were included in the baseline assessment. To address this uncertainty, the selection process for radionuclides is designed to include all components of the measured radioactive decay series by assuming secular equilibrium. Hence, the uncertainty associated with the screening step for radiological COCs is considered to be biased towards overestimation of risk.

Soil, which is the primary focus of this assessment relative to forthcoming cleanup decisions, is considered to be fairly well characterized for identification of radiological contaminants. The radiological risks for soil were based on reported radionuclide concentrations, as provided in the RI report. Analyses were conducted for only selected radionuclides of the uranium-238 and thorium-232 decay series; no other naturally occurring, accelerator-produced, or fission product radionuclides were considered. Because samples were generally not analyzed for radionuclides other than Th-232, Th-230, Ra-226, and U-238, the conservative assumption of secular equilibrium between the radionuclides in each decay series was made. Concentrations of the associated decay products were estimated based on this assumption and included in the risk characterization. This assumption may lead to overestimation of dose and risk. The approach used in this BRA is consistent with the history of site operations (i.e., as a processing facility) and the characteristics of radionuclides in these two decay series (i.e., the half-lives of the various radionuclides). The radionuclides of concern included in this assessment are considered to represent the possible extent of onsite contamination adequately.

Because not all radionuclides were reported for each sample location, a property-wide analysis may underestimate the radiological risk from exposure to a particular region of soil. Further, the majority of the properties were bias sampled at areas of elevated gamma radiation levels. No surface samples are available for Linde Subarea B. Shielding by overlying materials may have attenuated the gamma activity, allowing oversight of subsurface deposits. The property unit-wide analysis considered direct gamma irradiation, ingestion, and inhalation exposures to all radionuclides of concern in soil. However, the predominant radiological risk associated with contaminated soil is from external gamma irradiation. Measured gamma exposure rates were used where available in this assessment. The uncertainty in the estimates of the radiological risk from soil as a result of lack of location-specific radionuclide concentrations and undetected subsurface deposits is expected to be low.

5.3.1.2 Exposure Assessment

An exposure assessment is constructed from a number of site-specific considerations, including exposure point concentrations, scenario assumptions and intake parameters, and primary exposure pathways.

Factors that can contribute to uncertainty in exposure point concentrations include data availability and data heterogeneity. Extensive data are available for radionuclide concentrations in soil, but heterogeneity in the spatial distribution of contaminants could contribute to

uncertainties when estimating appropriate exposure point concentrations. The mean and UL_{95} confidence of the mean were used for the exposure point concentrations. This spatial averaging may overestimate or underestimate exposures for a receptor who may preferentially spend time at a particular location. The majority of the individual properties were sampled using a biased methodology. Although the majority of the receptors are expected to be mobile, and the intent of the data aggregation methodology was to identify areas of similar contaminant levels and land uses, the uncertainties related to data heterogeneity in soil remain significant and may be the most important component of total uncertainty in exposure assessment.

In the absence of measurements needed to assess the inhalation pathway at the site, air particulate concentrations have been modeled to estimate exposure point concentrations. Although greater uncertainty is associated with the exposures calculated for this pathway, inhalation is generally a minor contributor to radiological risks associated with the site. Therefore, the effect of this uncertainty on the exposure assessment is considered to be small.

Radon exposure also can be an important contributor to total risk. The use of measured radon concentrations at the Tonawanda site would have eliminated most of the uncertainty associated with those estimates. Radon measurements were not available. Radon concentrations were modeled based on soil radionuclide concentrations. The use of modeled values is a source of uncertainty associated with risk estimates. Although the uncertainty of the projected radon based risk is high, the relative contribution from radon is relatively small. Hence, the uncertainty introduced by the lack of measured radon values is considered to be relatively small.

The method for addressing nondetects (less than values) also affects the exposure point concentrations. The inclusion of the detection limit for nondetects tends to increase the reported concentrations and resultant uncertainty. The detection limits for most analyses were low relative to background or the appropriate soil concentration guidelines. The uncertainty associated with the incorporation of non-detects is considered to be small.

Another source of uncertainty is associated with the assumptions used to identify scenarios and intake parameters for the exposure estimates. Site-specific factors were used to select the scenario assumptions such as the extent of exposure (i.e., the exposure time, frequency, and duration) and to identify potential receptors (e.g., employees and transients). These assumptions use information on current land use and reasonable projections of future land use that consider the time frame of the assessment. The uncertainty in the scenarios developed for the current conditions is low because the time period is relatively short; current land uses are expected to continue during this period.

Future site use is hypothetical but based on reasonable projections for land use within the time frame of this assessment. The uncertainty in the selected scenarios is low.

Best professional judgment was used to define the variables used to estimate mean and RMEs for the identified receptors. Intake parameters used in the exposure assessment were derived from data in the literature, including EPA guidelines. Since considerable information

is available with respect to reasonable assumptions for intake parameters (e.g., inhalation rates), the related uncertainty is expected to be low. Furthermore, uncertainties associated with selecting values from the typical ranges identified for these parameters are not expected to significantly affect potential exposure estimates.

The exposure pathways quantified in this BRA were determined on the basis of the site conceptual model and related characterization data. The uncertainty associated with selected pathways for this assessment is low because site characterization data support the conceptual model.

5.3.1.3 Toxicity Assessment

Standard dose conversion factors and risk estimates were used to estimate the carcinogenic hazards associated with radioactive contaminants. The health effects associated with radiation exposure have been studied for many years and are well known. The risk estimators used in this assessment are generally accepted by the scientific community as representing reasonable projections of the hazards associated with radiation exposure.

Human epidemiological data on carcinogenesis from exposure to ionizing radiation is more extensive than that for most chemical carcinogens. However, these data are based primarily upon studies of populations exposed to radiation doses and dose rates that are orders of magnitude higher than the levels of concern at the Tonawanda site (e.g., atomic bomb survivors, uranium mine workers, radium dial painters). Use of these data to predict excess cancer risk from low-level radiation exposure requires extrapolation based upon very uncertain dose-response assumptions. This uncertainty is evidenced by the revision in cancer risk estimates presented in the BEIR V report (NRC 1990) by a factor of 3 to 4 over those presented only 10 years earlier in the BEIR III report (NRC 1980), due primarily to additional study of the atomic bomb survivors and reassessment of the atomic bomb dosimetry. Whereas this revision would indicate higher radiological risks than previously predicted, the BEIR V report also states that "...epidemiological data cannot rigorously exclude the existence of a threshold in the millirem dose range. Thus, the possibility that there may be no risks from exposures comparable to the external natural background radiation cannot be ruled out. At such low doses and dose rates, it must be acknowledged that the lower limit of the range of uncertainty in the risk estimates extends to zero" (NRC 1990).

5.3.1.4 Risk Characterization

Most of the assumptions built into this BRA tend to overestimate potential risks, including conservative assumptions for the exposure scenarios. Therefore, actual risks are likely to be lower than those presented in this assessment. However, some of the procedures used and uncertainties inherent in the human health assessment process may tend to underestimate potential risks, including the use of standard dose conversion factors based on adult exposures for estimating radiation doses.

The radiological dose conversion factors used in this assessment are based on the ICRP reference man. The reference man is an adult male weighing 70 kg. The ICRP selected such a standardized individual for their dosimetry models because their main concern is associated with worker protection; the majority of radiation workers are adult males. Children are more susceptible to radiation exposure, and such effects are significant only for young children. Young children are not expected to be the primary receptors of the Tonawanda site. Therefore, while use of dose conversion factors derived for adult males may introduce some uncertainty into the risk assessment, this uncertainty is considered to be small.

The estimation of health effects associated with radiation doses was based on lifetime-average risk estimators for all routes of exposure. These lifetime-average risk estimators are appropriate because they reflect the likely conditions of exposure, i.e., any given age group could be exposed to the radioactive contaminants. The uncertainty associated with the risk estimates used to assess radiation toxicity in this BRA is, therefore, low.

5.3.2 Uncertainty in Chemical Risk

5.3.2.1 Identification of Contaminants of Concern

COCs for the Tonawanda properties were determined using available characterization data and a selection process recommended by EPA for human health evaluation (EPA 1989a). Little documentation exists for chemical process operations that were conducted at the Tonawanda site. Controlled and uncontrolled chemical wastes were generated after MED operations ceased in 1946. Chemical waste was disposed of onsite at Linde, but existing site sampling has not determined fully the limits of waste constituents onsite or whether contamination exists offsite (BNI 1992). The existing grid of soils samples at Linde provides an adequate determination of contamination onsite, but gaps exist as to whether contamination extends offsite. There is limited surface soil sampling for Ashland 1 and 2, and none for Seaway.

The lack of chemical sampling results for the Seaway property precludes the quantification of the resulting excess carcinogenic and noncarcinogenic risks. The transient is the current and assumed future receptor. This receptor is exposed to maximum mean risks of 2×10^{-7} and RME risks of 3×10^{-6} at the other Tonawanda properties. Since the contamination is derived from the same source, these results may serve as a proxy of the Seaway risks. The chemical risks for transients are generally an order of magnitude smaller than the radiological risks. Based on this discussion, the Seaway chemical data gap is not considered to be a large source of uncertainty.

Insufficient data exist to quantify adequately the degree of groundwater contamination at the Tonawanda site or to delineate the extent of its migration. However, this is not a significant source of uncertainty since ingestion of groundwater is not a complete pathway. There are limited data to characterize offsite surface water and sediment contamination. There are no data for surface water and sediment at Seaway.

5.3.2.2 Exposure Assessment

The identification of potential receptors was based on reasonable land uses for the current scenarios. Future site use is hypothetical and was intended to indicate a reasonable conservative use of the site properties. Site-specific receptors were identified to the extent possible and exposure parameters tailored to those receptors (e.g., Linde employee and current recreational users of Ashland 2) to reduce uncertainty in the intake calculations and to make risk estimates more realistic.

Values assumed for exposure parameters (e.g. inhalation rate and exposure frequency) used in calculations for intakes, were based primarily on EPA guidance (EPA 1990 and 1991b). These assumptions might result in underestimating or overestimating the intakes calculated for specific receptors, depending on the accuracy of the assumptions relative to actual site conditions and uses. For example, a 50 mL/day water ingestion rate and a 200 mg/day sediment ingestion rate were used for the child playing in surface water drainages. The water ingestion rate is the EPA recommended value for incidental ingestion while swimming, and the sediment ingestion rate is the default value for child soil ingestion. It was also assumed that the child would be exposed to the highest dose at the confluence of Rattlesnake and Twomile Creeks. These assumptions overestimate intake, and thus risk, for the wading scenario.

This assessment does not include a complete estimation of the exposure through the dermal pathways because of the evolving nature of EPA's policies on quantifying this pathway for the COCs included in this BRA and because of the uncertainties in the values (i.e., dermal adsorption coefficients) necessary to calculate or estimate these pathways. Cadmium was the only chemical contaminant for which dermal contact was assessed. The estimated noncarcinogenic risk from this pathway and contaminant is one to three orders of magnitude less than the risk from the soil ingestion pathway at each property.

5.3.2.3 Toxicity Assessment

Uncertainty also is inherent in the toxicity values used in characterizing the carcinogenic and noncarcinogenic risks. Such uncertainty is chemical-specific and is incorporated into the toxicity value during its development. For example, an uncertainty factor may be applied for interspecies and intrahuman variability, for extrapolation from subchronic to chronic duration of exposures, or for epidemiological data limitations. A number of identified COCs are currently under EPA review with the possibility of changed RfDs, SFs, or carcinogenic weight of evidence. Interim and provisional toxicity values were used, where available, when values could not be obtained from IRIS or HEAST.

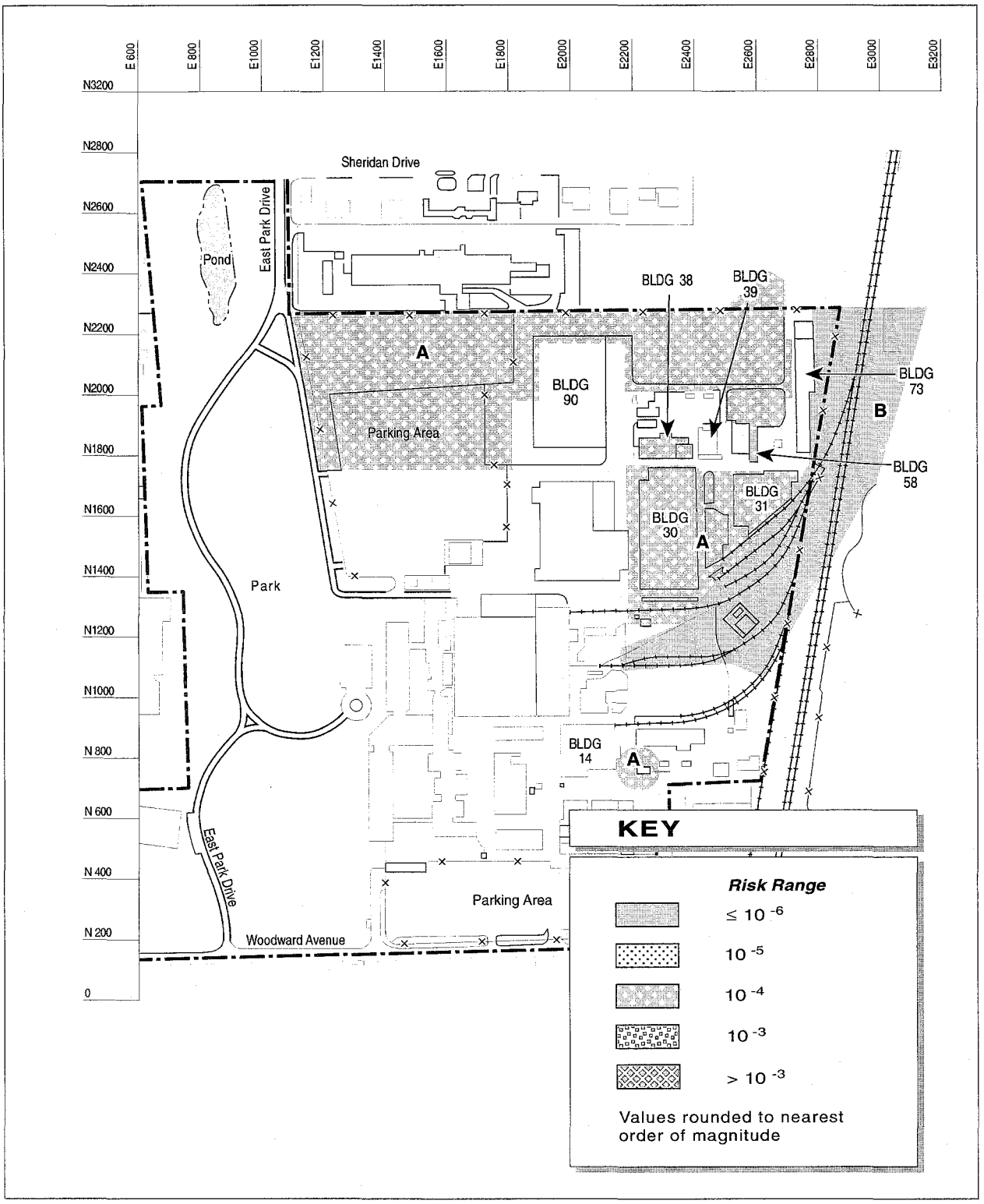
Additional uncertainty in risk estimates is introduced when all COCs do not have valid toxicity factors for use in quantitative estimates. Toxicity values could not be obtained for 14 COCs identified in Section 2, thereby precluding their inclusion in the quantitative risk assessment. Toxicity factors are not available for any of the rare earth element COCs. Although lead exposure causes significant toxic effects and lead may also be carcinogenic,

toxicity factors are not available (they are currently under EPA review). Recent draft guidance from EPA (1992e) suggests a quantitative method for estimating detrimental environmental lead levels (uptake/biokinetics model), but this method is not yet approved for use.

5.3.2.4 Risk Characterization

Some of the procedures used and uncertainties inherent in the human health assessment process may tend to underestimate potential risks. These include the lack of appropriate methodology and toxicity values to quantify chemical health effects for all identified COCs and routes of exposure. Assumptions built into this BRA tend to overestimate rather than underestimate potential risks, including conservative assumptions for the exposure scenarios. For example, contamination is assumed to remain constant over time. Fate and transport mechanisms were not considered in the exposure evaluation for chemical COCs. Actual concentrations may change over time, which would influence the intake and related risk values. Thus, actual risks are likely to be lower than those presented in this assessment.

Finally, for this assessment, it was assumed that the toxic and carcinogenic effects of the chemical COCs are additive. This assumption could result in the underestimation of risks because concurrent exposure to several contaminants might have synergistic toxic effects, (i.e., exposure to two of the metals concurrently might induce a greater toxic effect than that expected if the separate effects were simply added). Conversely, concurrent exposure to some of the metals might also mitigate the toxic effects of exposure to individual metals.



FUS/Tonawanda BRA 051593

Figure 5-1. Mean Excess Radiological Cancer Risk in the Current and Future Use Scenarios at the Linde Property

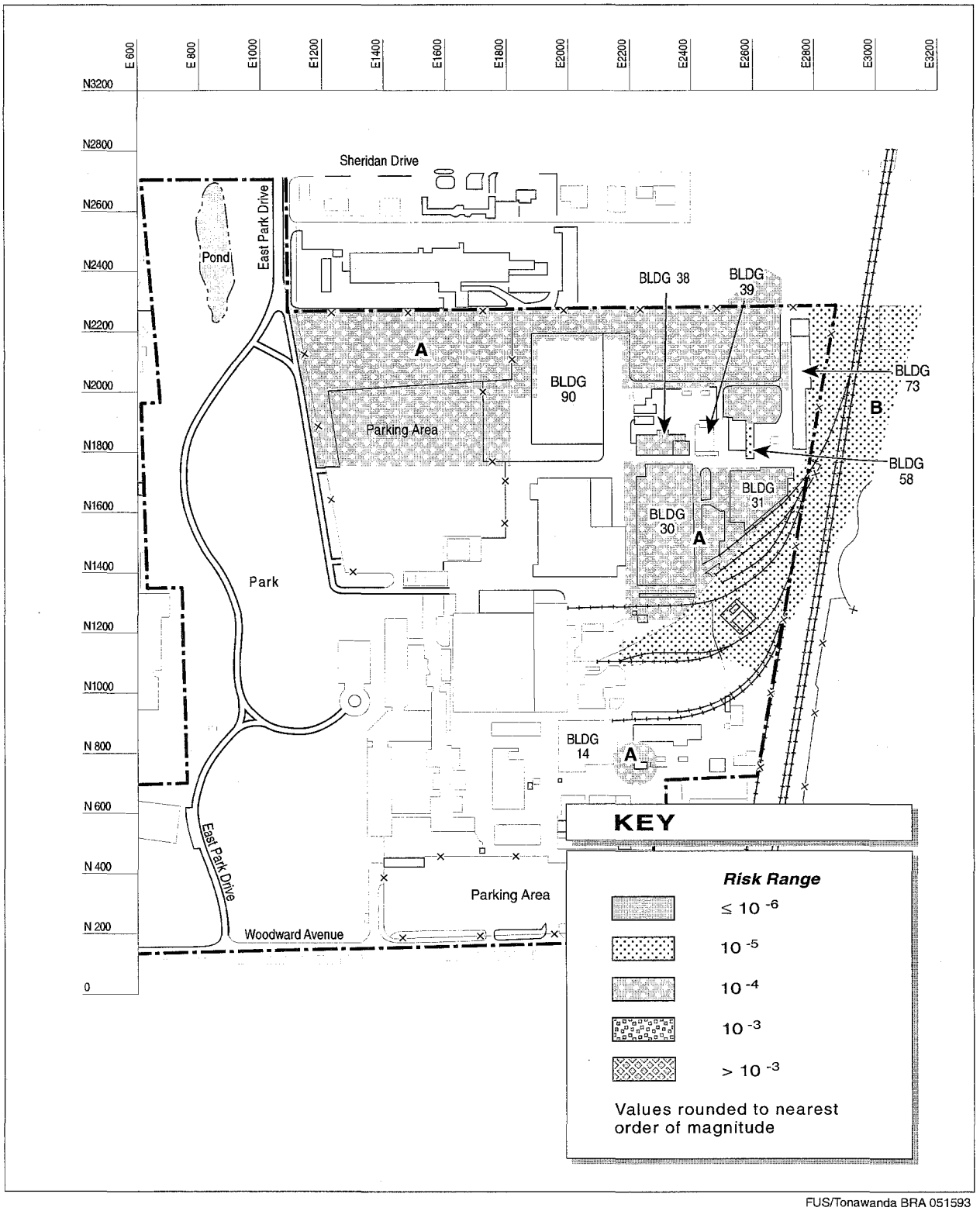


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Figure 5-2. RME Excess Radiological Cancer Risk in the Current and Future Use Scenarios at the Linde Property

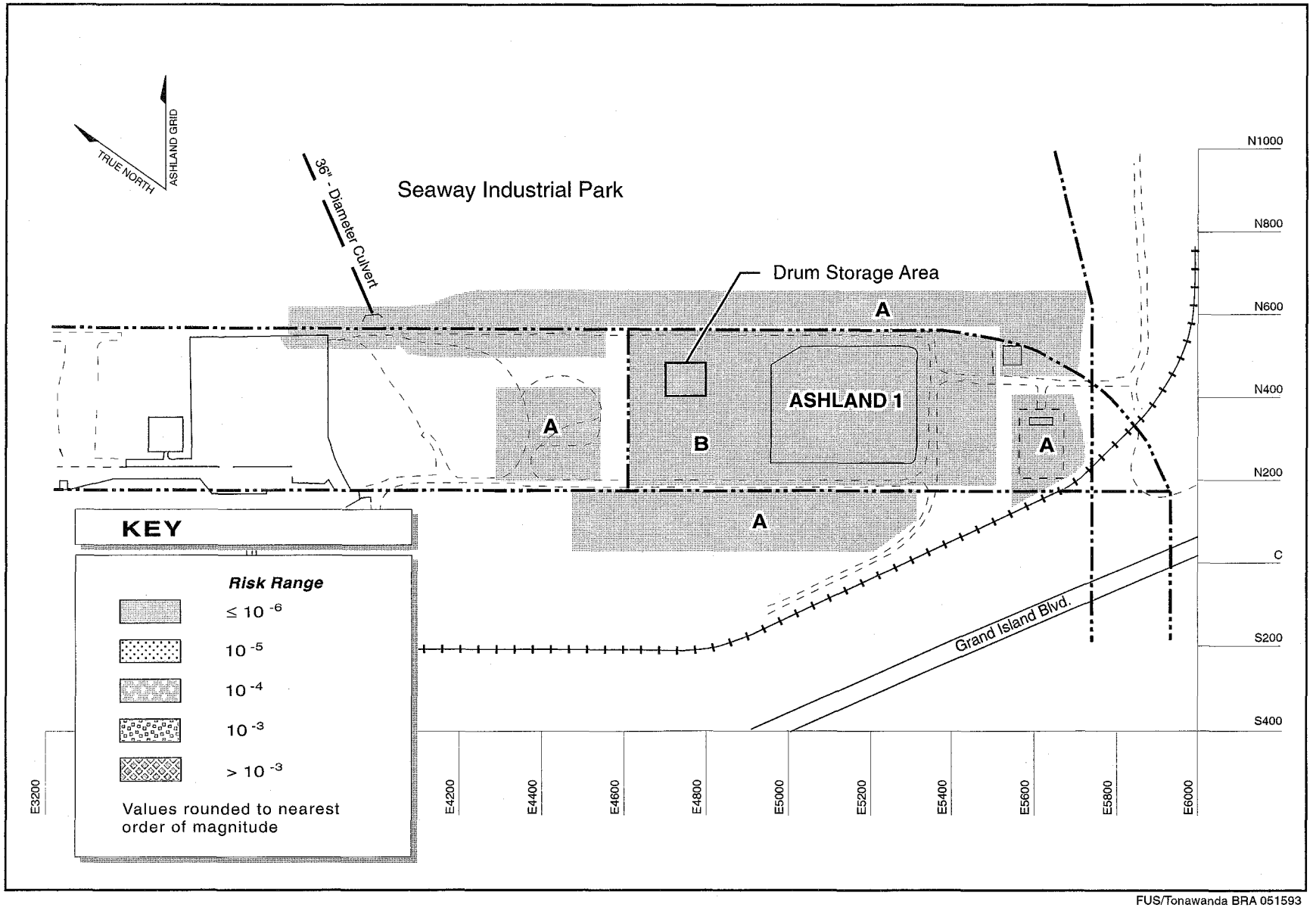

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Figure 5-3. Mean Excess Radiological Cancer Risk in the Current Use Scenario at the Ashland 1 Property

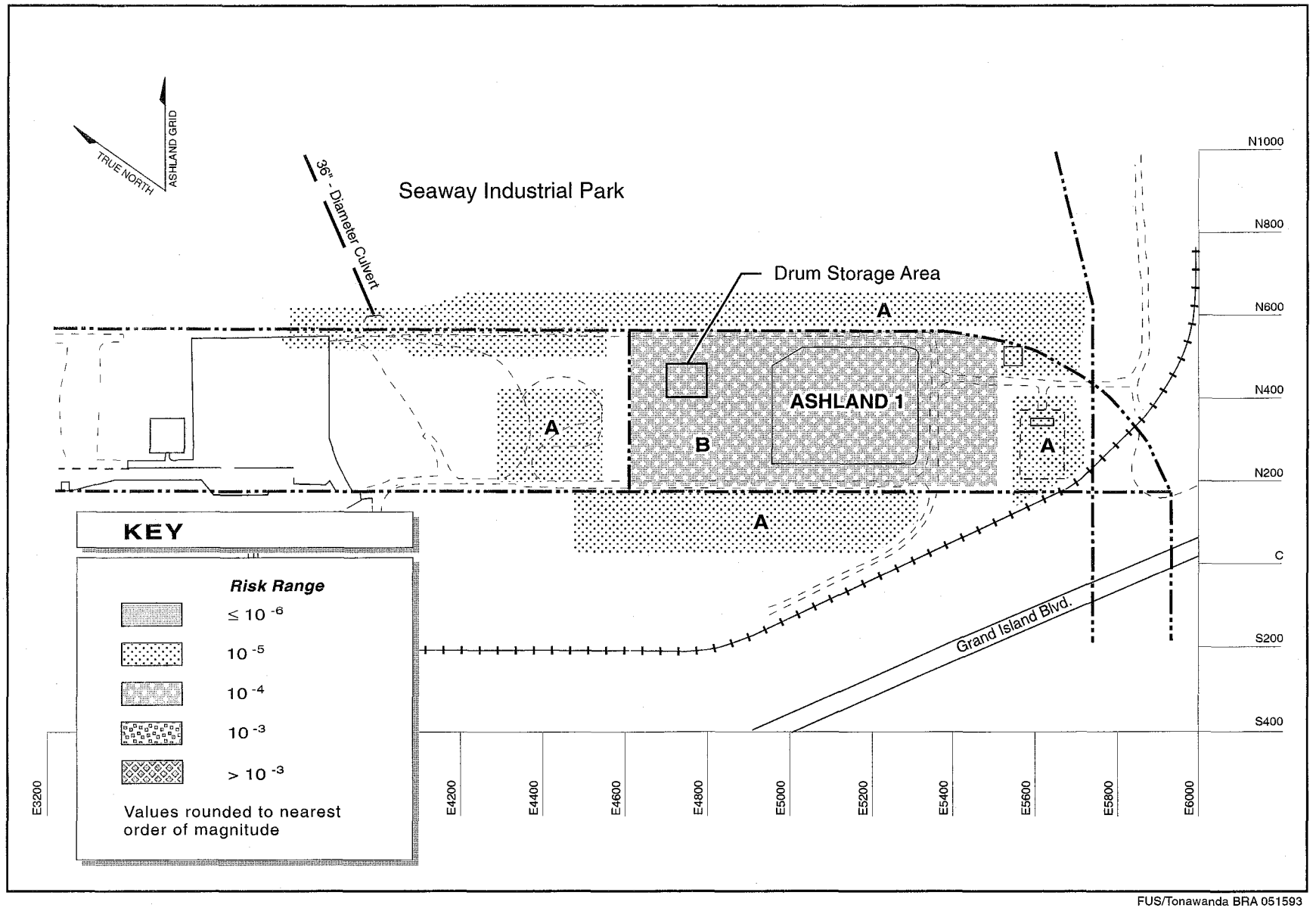

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Figure 5-4. RME Excess Radiological Cancer Risk in the Current Use Scenario at the Ashland 1 Property

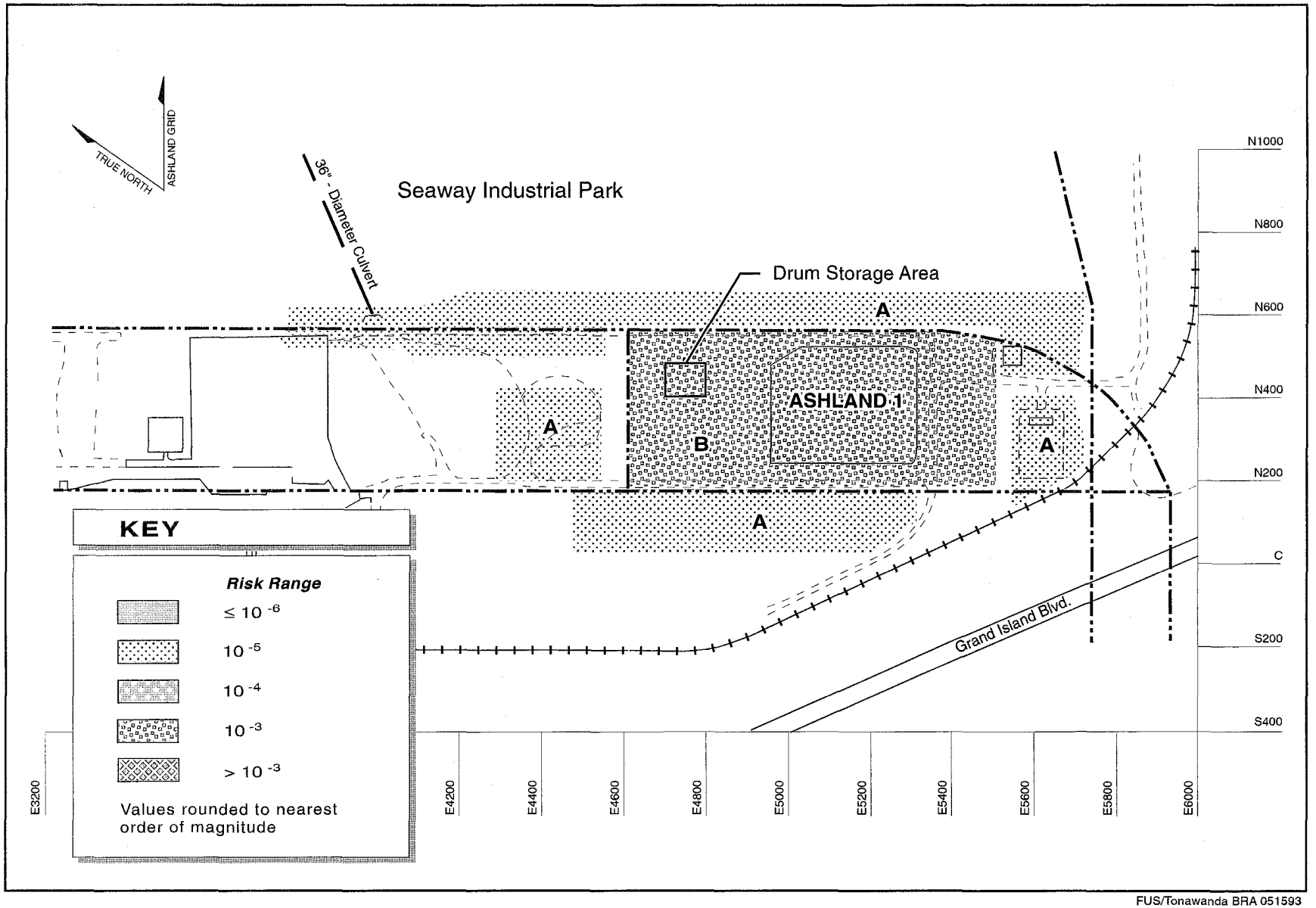

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Figure 5-5. Mean Excess Radiological Cancer Risk in the Future Use Scenario at the Ashland 1 Property

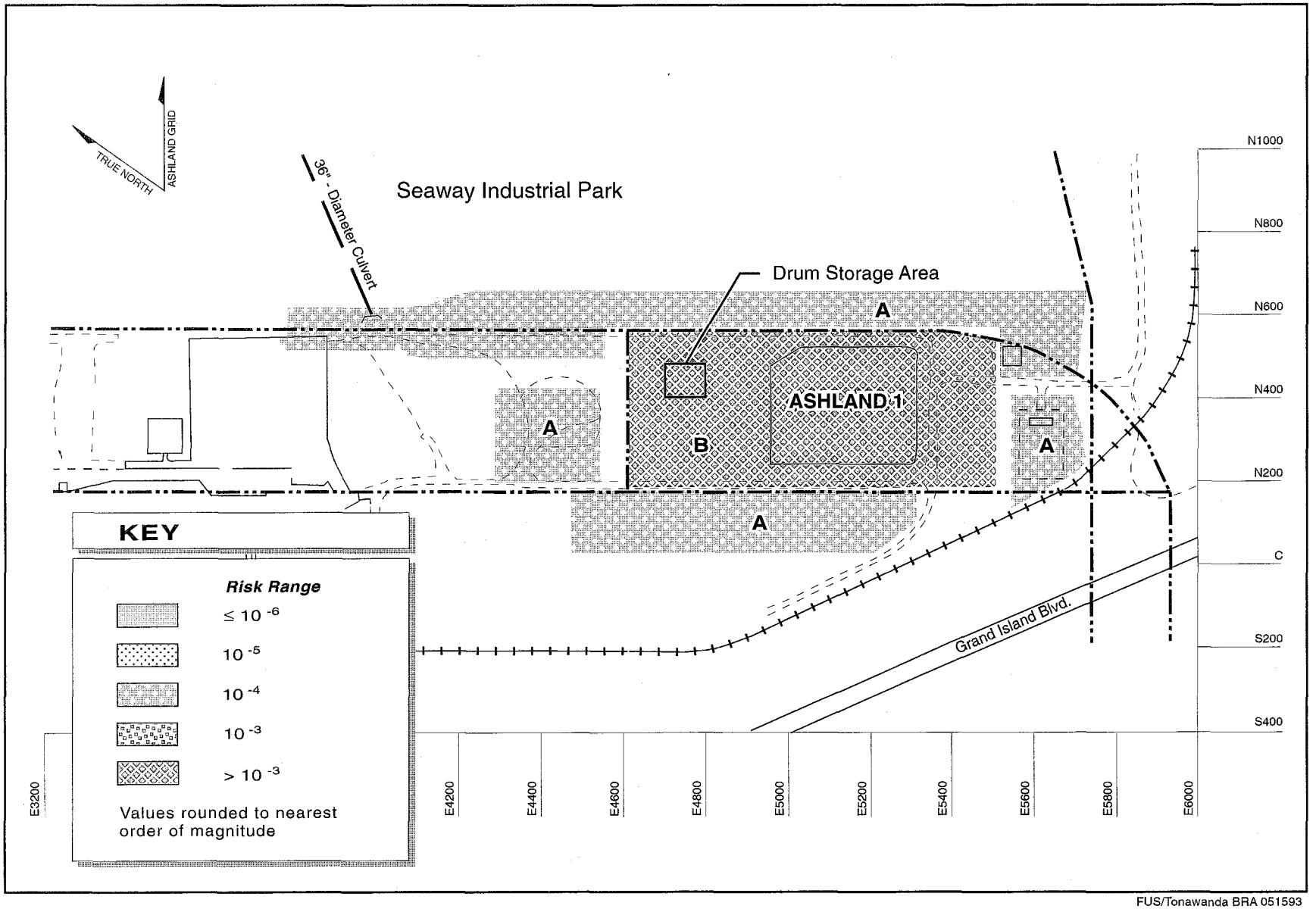

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Figure 5-6. RME Excess Radiological Cancer Risk in the Future Use Scenario at the Ashland 1 Property

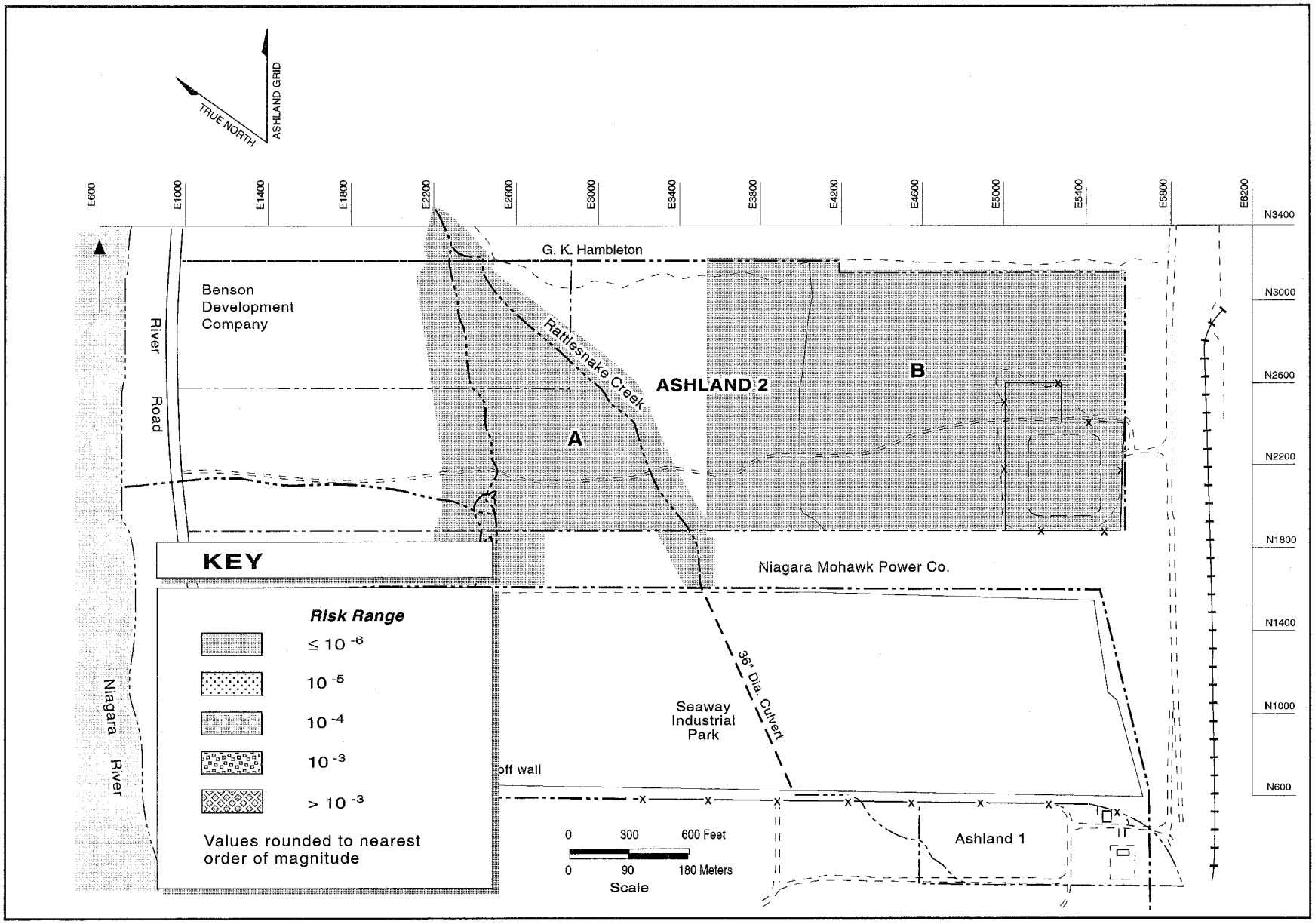

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Figure 5-7. Mean Excess Radiological Cancer Risk in the Current Use Scenario at the Ashland 2 Property

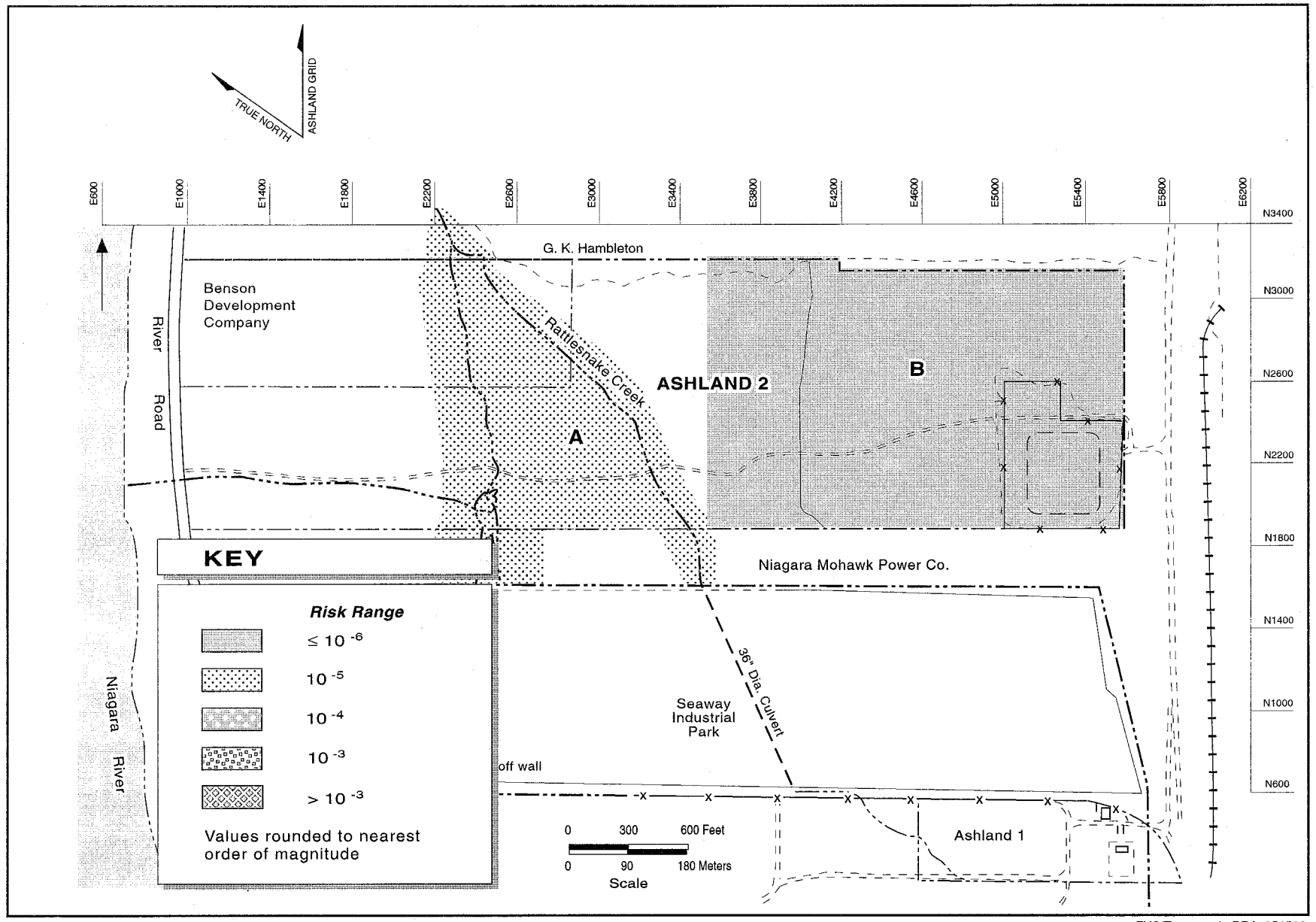

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Figure 5-8. RME Excess Radiological Cancer Risk in the Current Use Scenario at the Ashland 2 Property

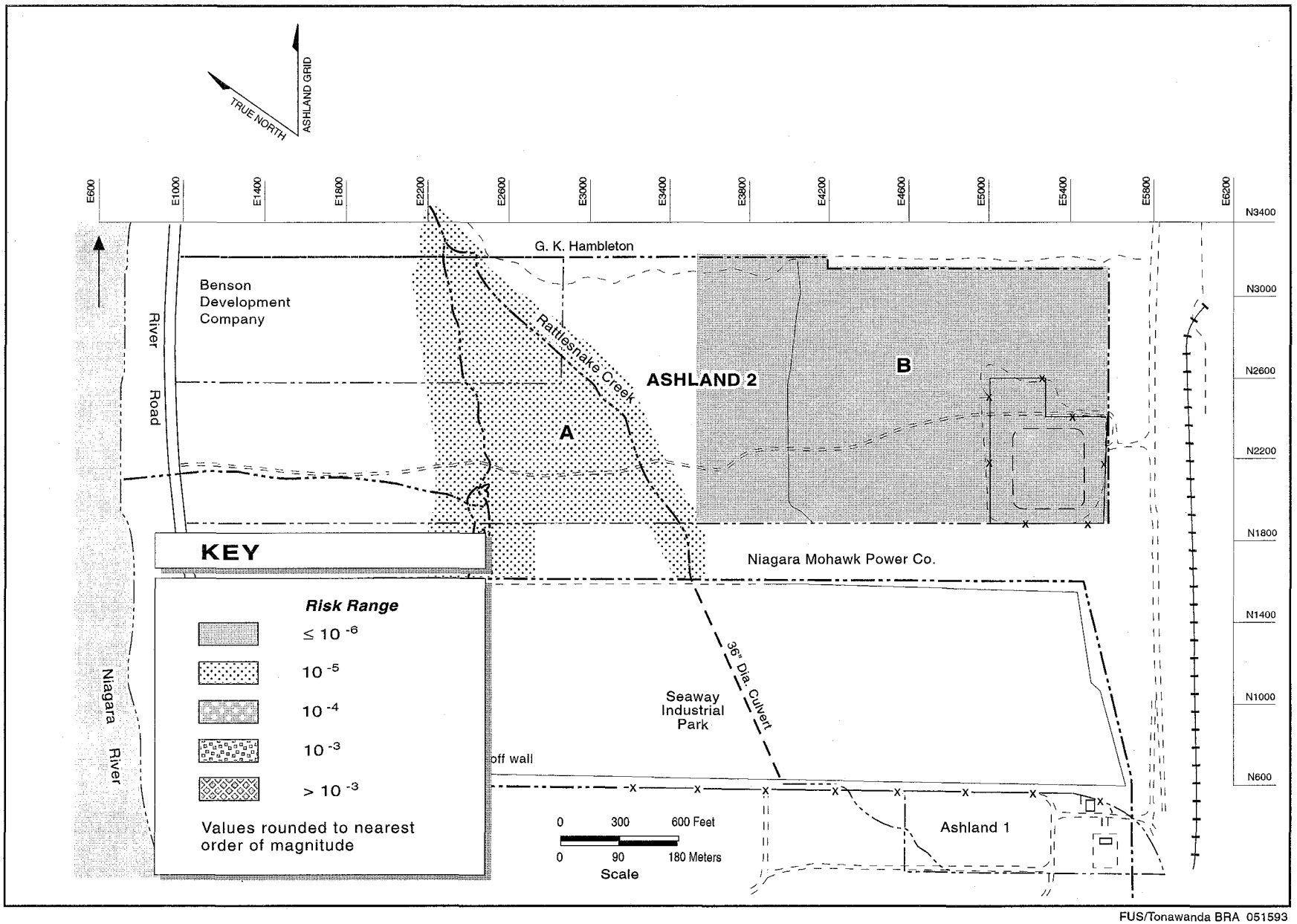

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Figure 5-9. Mean Excess Radiological Cancer Risk in the Future Use Scenario at the Ashland 2 Property

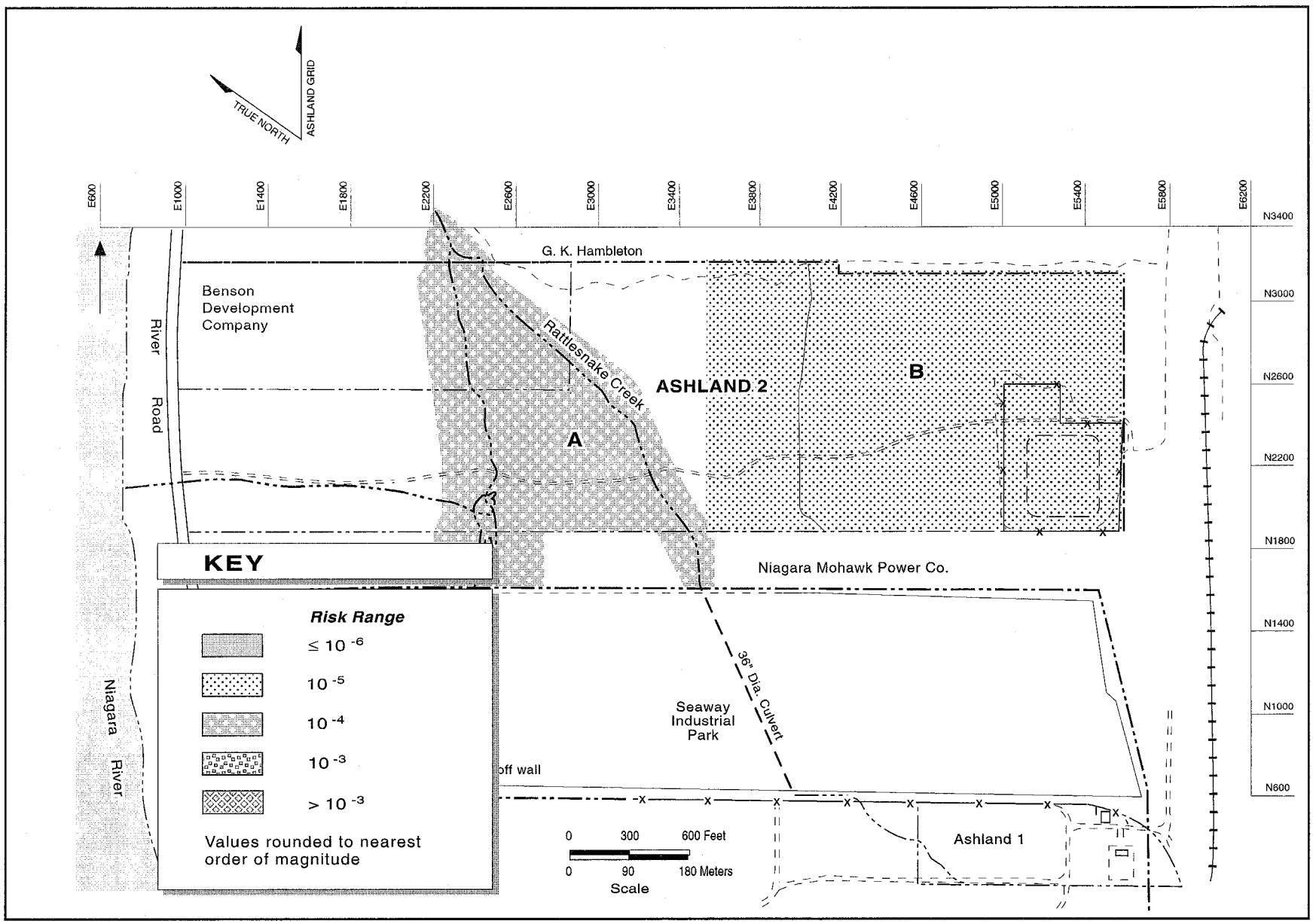

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Figure 5-10. RME Excess Radiological Cancer Risk in the Future Use Scenario at the Ashland 2 Property

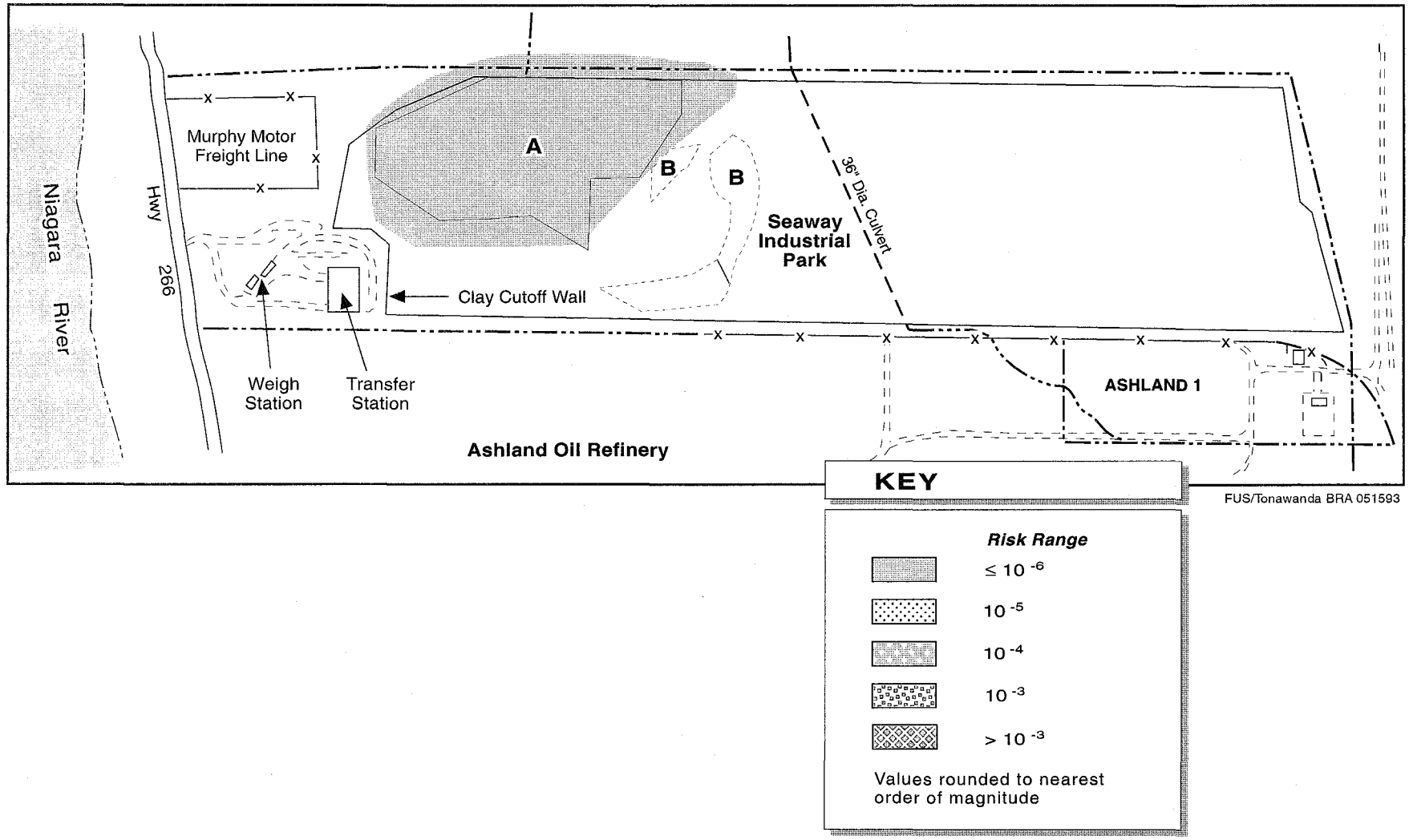

Figure 5-11. Mean Excess Radiological Cancer Risk for the Current and Future Use Scenarios at the Seaway Property

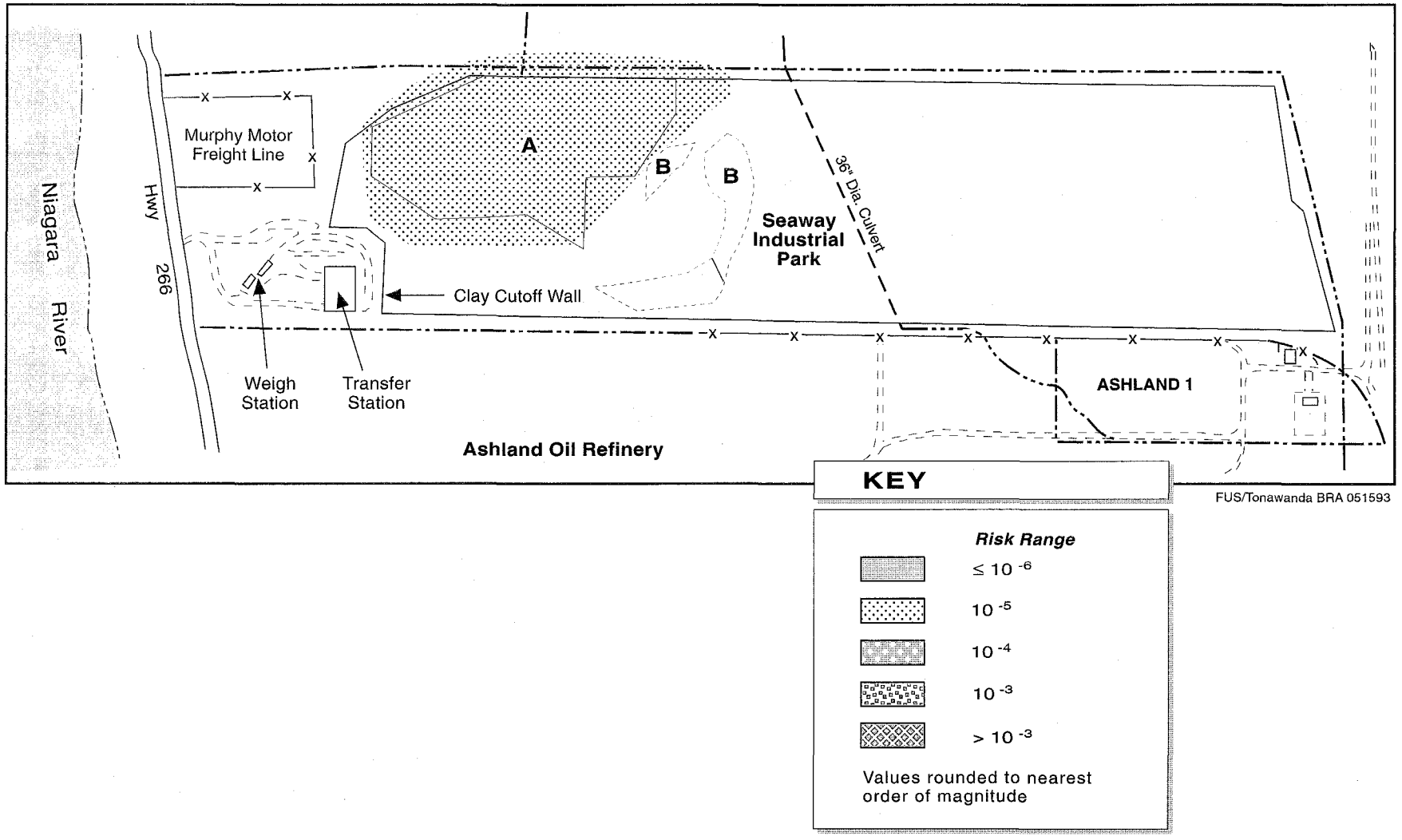

Figure 5-12. RME Excess Radiological Cancer Risk for the Current Use Scenario at the Seaway Property

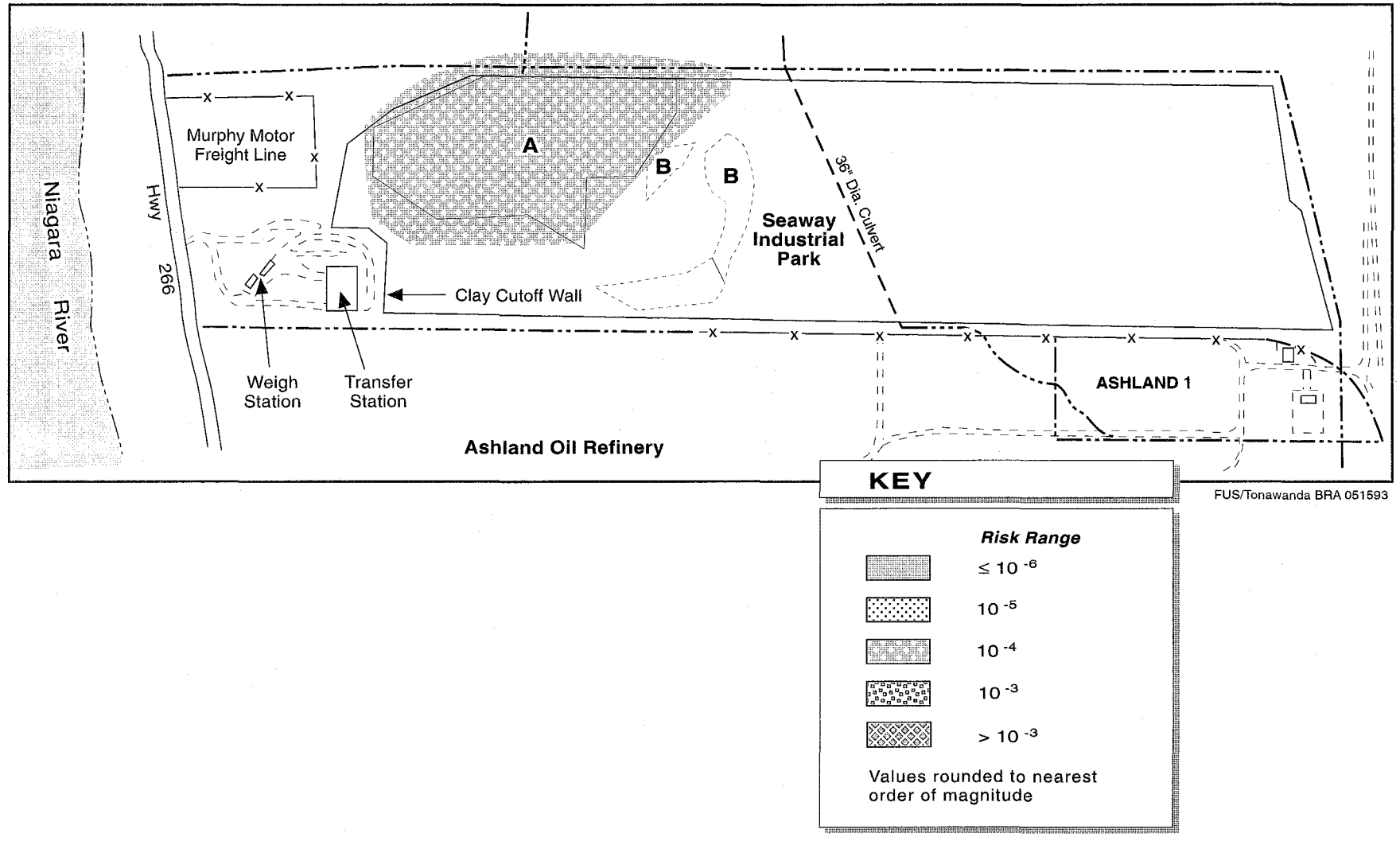

Figure 5-13. RME Excess Radiological Cancer Risk for the Future Use Scenario at the Seaway Property

TABLE 5-1. TOTAL RADIOLOGICAL RISK SUMMARY

		CURRENT USE SCENARIO (mrem/yr)			
LOCATION	SUBAREA	Employee		Transient	
		X	RME	X	RME
LINDE	A	6.5E-05	4.0E-04		
	B	2.5E-06	1.8E-05		
ASHLAND 1	A			9.5E-08	7.8E-06
	B			1.0E-06	1.1E-04
ASHLAND 2	A			3.9E-07	5.5E-05
	B			5.1E-09	5.4E-06
SEAWAY	A			5.6E-07	5.0E-05
	B				
LOCAL CREEK	A			1.5E-07	8.6E-07
		FUTURE USE SCENARIO (mrem/yr)			
LOCATION	SUBAREA	Employee		Transient	
		X	RME	X	RME
LINDE	A	6.5E-05	4.0E-04		
	B	4.1E-06	3.8E-05		
ASHLAND 1	A	6.6E-06	8.6E-05		
	B	6.7E-04	9.9E-03		
ASHLAND 2	A	4.1E-05	4.6E-04		
	B	4.4E-07	1.8E-05		
SEAWAY	A			6.8E-07	2.4E-04
	B				
LOCAL CREEK	A			1.5E-07	8.6E-07

X --- Mean

RME --- Reasonable Maximum Exposure

Table 5-2. Summary of Chemical Risk - Carcinogens

CURRENT USE SCENARIO				
Location	Employee		Transient	
	Mean	RME	Mean	RME
LINDE				
Soil ingestion	2E-5	8E-5		
Particulate inhalation	3E-8	7E-7		
ASHLAND 1				
Soil ingestion			2E-7	3E-6
Particulate inhalation			2E-12	3E-10
ASHLAND 2				
Soil ingestion			2E-7	2E-6
Particulate inhalation			1E-10	1E-8
LOCAL CREEK				
Surface water ingestion			4E-7	8E-7
Sediment ingestion			8E-8	2E-7

FUTURE USE SCENARIO				
Location	Employee		Transient	
	Mean	RME	Mean	RME
LINDE				
Soil ingestion	2E-5	8E-5		
Particulate inhalation	3E-8	7E-7		
ASHLAND 1				
Soil ingestion	3E-7	4E-6		
Particulate inhalation	1E-10	2E-9		
ASHLAND 2				
Soil ingestion	4E-7	4E-6		
Particulate inhalation	5E-9	2E-7		
LOCAL CREEK				
Surface water ingestion			4E-7	8E-7
Sediment ingestion			8E-8	2E-7

Table 5-3. Summary of Chemical Risk - Noncarcinogens

CURRENT USE SCENARIO				
Location	Employee		Transient	
	Mean	RME	Mean	RME
LINDE				
Soil ingestion	1E-1	3E-1		
Particulate inhalation	8E-6	1E-4		
Dermal contact	4E-4	6E-4		
ASHLAND 1				
Soil ingestion			5E-3	3E-2
Particulate inhalation			1E-9	3E-8
ASHLAND 2				
Soil ingestion			1E-2	2E-1
Particulate inhalation			0E-0	0E-0
Dermal contact			5E-4	5E-3
LOCAL CREEK				
Surface water ingestion			2E-2	7E-2
Sediment ingestion			2E-3	6E-3
Dermal contact			4E-7	8E-7

FUTURE USE SCENARIO				
Location	Employee		Transient	
	Mean	RME	Mean	RME
LINDE				
Soil ingestion	1E-1	3E-1		
Particulate inhalation	8E-6	1E-4		
Dermal contact	4E-4	6E-4		
ASHLAND 1				
Soil ingestion	7E-3	3E-2		
Particulate inhalation	6E-8	2E-7		
ASHLAND 2				
Soil ingestion	2E-2	1E-1		
Particulate inhalation	0E-0	0E-0		
Dermal contact	2E-3	7E-3		
LOCAL CREEK				
Surface water ingestion			2E-2	7E-2
Sediment ingestion			2E-3	6E-3
Dermal contact			4E-7	8E-7

6. ECOLOGICAL RISK ASSESSMENT

The environmental evaluation process is outlined in the *Risk Assessment Guidance for Superfund Vol. II Environmental Evaluation Manual Interim Final* (EPA 1991c, OSWER Directive 9285.7-01). The *Environmental Evaluation Manual* does not provide a step-by-step approach to risk assessment as does the *Human Health Evaluation Manual* but instead discusses an overall framework for considering environmental effects and identifies sources of pertinent information. Although environmental evaluations and human health evaluations are different processes, they share certain chemical data and information. The phrase "environmental evaluation" was patterned after "human health evaluation;" however, the term "ecological risk assessment" (ERA) has become standard and will be used throughout this ERA. A discussion of the scientific basis for assessing ecological effects is found in *Ecological Assessments of Hazardous Waste Sites: A Field and Laboratory Reference Document* (EPA/600/3-89/013, EPA 1989c).

The ERA for the Tonawanda BRA is structured according to the proposed general framework for ecological assessments in the Superfund Program (EPA 1991b, EcoUpdate No. 2). This ERA comprises four interrelated activities: problem formulation (Section 6.1), exposure assessment (Section 6.2), effects assessment (Section 6.3), and risk characterization (Section 6.4), which includes a discussion of uncertainties. Because of the qualitative nature of the characterization of habitats and biota at risk and the semiquantitative screening of contaminants, the assessment of potential impacts to wildlife from exposure to contaminants must be based largely on the toxicological effects reported in the literature for many of the contaminants of ecological concern and on expected mechanisms of transport and biological uptake. Where toxicity data were available, a semiquantitative characterization of the risk to Tonawanda biotic communities from exposure to the ecological COCs was based on the ratio of environmental concentration to toxicity threshold concentration (Barnthouse et al. 1986).

6.1 PROBLEM FORMULATION

The conceptual model of the Tonawanda site, which identifies the potential contaminants and their sources, is presented in Section 2. In this ERA, the ecological resources at the site, COCs, and exposure pathways are identified, and the nature and relative magnitude of the risk to these resources (especially animals) are characterized. This is done on a location by location and medium by medium basis.

6.1.1 Objective

The objective of this ERA is to define and evaluate the risk of adverse effects on the biotic environment from exposure to the contaminants at the Tonawanda site. A qualitative habitat characterization identifies biotic components of the ecosystem, including organisms potentially exposed to contaminants. Field measurements of contaminant concentrations and

published toxicity data for aquatic and terrestrial organisms allow a semiquantitative estimate of risk using the ratio or quotient method. This information is used to characterize the relative magnitudes of risks to ecological resources from contaminated media at the various Tonawanda properties.

6.1.2 Scope

The scope of this ERA includes both aquatic and terrestrial organisms that may be directly or indirectly exposed to contaminants associated with sources at the Tonawanda site. Identifying and assessing the risks to local biota and habitats on the Tonawanda properties exposed to site contaminants is feasible, even though environmental and toxicological data are limited. Concentration data exist for radionuclides, metals, VOCs, and SVOCs in environmental media at the Linde, Ashland 1, and Ashland 2 properties. No quantitative site-specific biological studies have been conducted. A reconnaissance study of biota was performed in 1988 (BNI 1991a). This reconnaissance was updated in July and August 1992, and the updated survey forms the basis of the habitat characterization (Section 6.1.4). The biotic diversity in the Tonawanda area is consistent with a modified urban environment, with industries, residential areas, commercial properties, and scattered wetlands and old-fields. The wildlife and habitat characteristics of these wetlands, in particular, could have ecological, recreational, and aesthetic value from a regional perspective, which is not considered explicitly in this assessment. Site contaminants that qualify as COCs for quantitative risk assessment (Section 2.3.1) are screened further for assessment as ecological COCs (Section 6.1.5). A contaminant qualifies as an ecological COC if its environmental concentration exceeds a toxicity-threshold concentration and if it meets mobility and persistence criteria. Emphasis is given to both aquatic and terrestrial organisms at the Tonawanda site. Terrestrial organisms may be exposed to contaminants in Linde, Ashland 1, and Ashland 2 soils through ingestion of soil, groundwater, or contaminated organisms. Contaminants in soil and groundwater can be released via surface water runoff to Twomile and Rattlesnake Creeks, thereby exposing aquatic communities by direct contact or indirectly by ingestion of water and sediments. The relative risks to classes of organisms exposed by different means to various contaminants at the Tonawanda properties are estimated using ratios of the environmental concentrations of contaminants (corrected or uncorrected for exposure differences) to toxicity threshold concentrations obtained from published data in AQUIRE (1992) and other environmental and toxicological databases.

6.1.3 Assumptions

The Tonawanda ERA assumes that the basic approach to ERAs described in EPA guidance (EPA 1989b; EPA 1989d) is acceptable. Habitat maps based on site visits are assumed to be sufficient to identify potentially exposed habitats and species known or likely to occur in those habitats (EPA 1991d). The Tonawanda ERA assumes that there are no threatened or endangered species on the site (USFWS 1992). This ERA assumes that a separate screening of contaminants to identify COCs based on ecological criteria is necessary because not all contaminants that pose a risk to human health are ecologically important and vice versa (EPA 1991d). Further, separate exposure, effects, and risk assessments are preferred (EPA 1991d).

Identifying ecological COCs and characterizing risks requires toxicity thresholds based on chronic exposure. U.S. government standards are the first sought and used values, followed by published "no effect" levels and then chronic toxicity values. In the absence of the above, an acute measurement is used. Deriving chronic thresholds from acute toxicity data is based on the assumption that the resulting chronic threshold will be protective of the majority of species most of the time (Michigan Water Resources Commission 1986). Thresholds derived from published "no effects" data or chronic toxicity are also assumed to be protective to most organisms most of the time but with less uncertainty than those derived from acute values. In general, it is assumed that the ecological COC screening process is conservative. Assumptions about primary exposure pathways and differences in exposure to various classes of ecological receptors are based on professional judgment.

6.1.4 Habitat Characterization

The Tonawanda site consists of four properties near Buffalo, New York: Linde, Ashland 1, Seaway, and Ashland 2 (Figure 1-2). This urban area has undergone intensive development for commercial, industrial, or residential uses. An overview of the Tonawanda site is given below (Section 6.1.4.1) and the habitats on each property are discussed separately (Section 6.1.4.3).

6.1.4.1 Habitat Overview

The Tonawanda site lies within the Beech-Maple Forest section of the Eastern Deciduous Forest division (Bailey 1980). Eyre (1980) shows the predominant forest type in this area as elm-ash-cottonwood (locally exhibited as ash-elm-maple), surrounded by a maple-beech-birch type. Black and green ash (*Fraxinus nigra* and *F. pennsylvanica*, respectively); red and silver maple (*Acer rubrum* and *A. saccharinum*, respectively); and American, rock, and slippery elm (*Ulmus americana, U. thomasii*, and *U. rubra*, respectively) are typical trees in the area. Aspen (*Populus* spp.), pin cherry (*Prunus pennsylvanicum*), hawthorn (*Crataegus* spp.), and beech (*Fagus grandifolia*) are common associates. Eastern hemlock (*Tsuga canadensis*) and white pine (*Pinus strobus*), once abundant, have been logged and eliminated from much of the area.

Little or no actual forest habitat currently occupies any of the properties or the immediate vicinity. Urban development for residential and industrial use has eliminated any undisturbed habitat. Most natural habitat types remain only as small woodlands or in poorly drained areas (Galvin 1979). Three properties (Linde, Ashland 1, and Seaway) provide minimal habitat to urban wildlife, supporting only cosmopolitan species of birds and small mammals (FBDU 1981a). The larger and less disturbed area at Ashland 2 supports a more diverse population of animals because it has several small wetlands and is unevenly covered with a mixture of grasses, forbs, shrubs, and small trees.

All four of the properties lie within the watershed of Twomile Creek, a tributary to the Niagara River. The Linde property is linked by various drainage ditches to Twomile Creek. Several small intermittent drainageways and Rattlesnake Creek, which flows into Twomile Creek

300 m (1,000 ft) above the confluence with the Niagara River (Figure 6-1) hydrologically connect Ashland 1, Seaway and Ashland 2 with Twomile Creek.

6.1.4.2 Threatened or Endangered Species

Except for occasional transient individuals, no federally-listed or proposed candidate species under the jurisdiction of the U.S. Fish and Wildlife Service (USFWS) have been sighted in the project impact area (USFWS 1992). The most likely listed species to appear on or near the site are osprey (*Pandion haliaetus*), bald eagle (*Haliaetus leucocephalus*), and peregrine falcon (*Falco peregrinus*) (FBDU 1981b, Gill 1989). The properties nearest the Niagara River are most likely to host transient individuals of these species. No listed or suspected critical habitats occur on the Tonawanda site.

A New York state-listed threatened plant species, stiff leaf goldenrod (*Solidago rigida*), occurs near the Tonawanda site. A site-specific survey performed in August 1992 by a qualified botanist determined that this species is not currently present on any of the Tonawanda properties (Cunningham 1992).

6.1.4.3 Tonawanda Area Habitat Survey

During July and August 1992, site surveys were conducted at the Tonawanda site. Qualitative surveys were conducted on foot and by automobile. This survey included all properties except Seaway, with emphasis placed on Ashland 2. During the August site visit, the properties were surveyed for threatened or endangered species (Cunningham 1992). During both focused walkovers, additional findings about existing habitats and species were recorded. These findings and other studies form the authority for the following descriptions.

Linde

Linde is located within the Town of Tonawanda. The area around Linde is a mixture of commercial, industrial, and residential properties. There is virtually no natural habitat at Linde. Years of continuous industrial activity at the Linde facility and its surroundings have left only marginal areas for natural plant communities. The entire plant area within the Linde fence is paved or occupied by buildings or gravel-covered storage areas. The Linde property is landscaped with several nearly mature eastern cottonwood (*Populus deltoides*), American sycamore (*Platanus occidentalis*), white ash (*Fraxinus americana*), northern red oak (*Quercus rubra*), and shagbark hickory (*Carya ovata*) and residential lawns with shrub plantings.

Twomile Creek used to flow on the west side of Linde outside the fence. Presently, the creek flows underground through culverts. The old channel has been filled in, graded, and is now maintained as a lawn. Downstream from Linde, Twomile Creek flows beneath Sheridan Lake Park. Very little natural habitat exists within the Tonawanda city limits. North of Interstate 290 Twomile Creek flows to the Niagara River through a corridor of native vegetation. This corridor ranges from about 15 - 150 m (50 - 500 ft) in width.

A small palustrine wetland is located outside the fence on the northeast corner of the plant. The wetland includes an emergent hydrophyte community adjacent to a small wooded area. The emergent plant community is dominated by common rush (*Phragmites australis*), broad-leaf cattail (*Typha latifolia*), and purple loosestrife (*Lythrum salicaria*). The wooded portion of the wetland is dominated by a mixture of pin oak (*Quercus palustris*) and red-osier dogwood (*Cornus stolonifera*), stiff dogwood (*C. foemina*), or silky dogwood (*C. amomum*).

No aquatic resources are present onsite at Linde. However, a small pond is located northwest of Linde and is presumably connected to Sheridan Park Lake by a culvert beneath Sheridan Drive.

In the upper part of its watershed near Linde, Twomile Creek has been channelized or flows beneath the ground through large culverts. In the lower reaches Twomile Creek is mostly a free-flowing stream. Throughout the stretch of Twomile Creek between Linde and the Niagara River, the creek exhibits evidence of degradation, such as a pervasive odor of sewage and a great deal of rubbish in and along the creek banks.

Ashland 1

The Ashland 1 property is located at the now defunct Ashland Oil Refinery (Figures 6-2, 6-3). Much of the area is covered with concrete or buildings, and is nearly devoid of vegetation because of the nearly constant disturbance from previous operations associated with the Ashland refinery. One-third of the property, bermed as a containment area for petroleum storage tanks, contains a sparse cover of shrubs and grasses. The plant species most prevalent at Ashland 1 is common rush. Industrial development and related activities have significantly altered or eliminated any natural plant communities. Wildlife is represented by birds such as rock dove (*Columba livia*), American crow (*Corvus brachyrhynchos*), unidentified gulls, mourning dove (*Zenaida macroura*), killdeer (*Charadrius vociferus*), European starling (*Sturnus vulgaris*), common grackle (*Quiscalus quiscula*), and American robin (*Turdus migratorius*), and mammals such as house mouse (*Mus musculus*), Norway rat (*Rattus norvegicus*), eastern cottontail (*Sylvilagus floridanus*), and eastern gray squirrel (*Sciurus carolinensis*). The only animals observed at Ashland 1 during the July 1992 site visit were unidentified species of seagulls.

Seaway

The Seaway property, an active solid waste disposal facility (Figure 6-4), supports sparse vegetation composed of shrubs and grasses, daisies, milkweeds, vetches, foxtail grasses, clovers, sorrels, and cattails. New York regulations require seeding with native grasses during the closure and post-closure phases of solid waste disposal to slow erosion and promote evapotranspiration (Figure 6-5). Landfill operations and nearby industrial activity limit use of the area by wildlife, although gulls and crows are present. The drainage channels at Seaway would not be expected to support any aquatic organisms beyond those ordinarily found in man-made drainage systems.

Ashland 2

Past land use at Ashland 2 has resulted in a mosaic of different habitats dominated by trees, shrubs, or herbaceous species (Figure 6-6). Wetlands dominated by shrubs and herbaceous vegetation are found in poorly drained depressions in upland areas, as well as in the swales and drainageways that make up the tributaries to Twomile Creek (BCI 1992). The nature and diversity of the plant communities at Ashland 2 provide a mixture of habitats that provide cover and food for a variety of animals. White-tail deer (*Odocoileus virginianus*), rabbits, frogs, snakes, and many unidentified species of birds were seen at the property in August 1992. During a site visit in July 1992, American goldfinches (*Carduelis tristis*), an unidentified hawk, unidentified gulls, and crows were seen. In July 1976, Roblee (1976) reported evidence that muskrat (*Odontra zibethecus*), red-winged blackbird (*Agelaius phoeniceus*), ring-necked pheasant (*Phasianus colchicus*), mallard (*Anas platyrynchos*), raccoon (*Procyon lotor*), mink (*Mustela vison*), and killdeer used the Twomile Creek wetland that includes Rattlesnake Creek. Roblee based his assessment on direct observation of animals and animal signs such as spoor or tracks. In addition to the common mammal species mentioned at Ashland 1, a number of waterfowl species, foxes (*Vulpes* spp.), striped skunk (*Mephitis mephitis*), weasels (*Mustela spp.*) and opossum (*Didelphis virginiana*) may use the property.

Six habitat types are present at Ashland 2, (1) forested wetlands, (2) forested uplands, (3) upland shrubs, (4) herbaceous wetlands, (5) herbaceous uplands, and (6) vegetated drainage swales. These habitat types were characterized by direct observations during site visits and a detailed wetland delineation report was prepared for Ashland Oil Co. by Beak Consultants, Inc. (BCI 1992). Brief narrative descriptions of each habitat type are presented below.

Forested Wetlands. Forested wetlands are characterized by trees tolerant of wet conditions. These wetlands occur in poorly drained depressions on higher topographic positions that have not been recently cut-over or otherwise disturbed. These wetland communities are located in the northeast corner of Ashland 2 and make up a very small portion of the property. Green ash and crack willow (*Salix fragilis*) are typical dominant species; American elm is a common associate. Shrubs such as common buttonbush (*Cephalanthus occidentalis*), silky dogwood, and crack willow are important components of this plant community. The understory comprises primarily green ash saplings and buttonbush seedlings. Herbaceous species such as broad-leaf cattail and uptight sedge (*Carex stricta*) frequently grow where openings in the canopy allow direct sunlight to reach the ground surface.

Forested Uplands. In the higher, drier uplands at Ashland 2 where recent disturbance has been minimal, trees such as chestnut oak (*Quercus prinus*) and hawthorn have become the dominant species in the overstory. Important shrubs include common buckthorn (*Rhamnus cathartica*) and stiff dogwood. The understory consists of saplings of hawthorn and American elm with buckthorn, American dewberry (*Rubus flagellaris*), and stiff dogwood seedlings. Few herbaceous species are found in the dense shade of the overstory and shrubs.

Upland Shrubs. Upland shrub communities are dominated by very dense thickets of shrubs such as dewberry and stiff dogwood (Figure 6-7). Occasionally trees such as green ash or hawthorn may be scattered throughout the shrub-dominated habitat. In areas where the shrub cover is less dense, red maple, pin oak and hawthorn saplings and hawthorn, stiff dogwood, and dewberry seedlings have become established (Figure 6-8). Herbaceous species such as strawberry (*Fragaria virginiana*), common yarrow (*Achillea millefolium*), old field cinquefoil (*Potentilla simplex*), flat-top fragrant-golden-rod (*Euthamia graminifolia*), Canada golden-rod (*Solidago canadensis*), wrinkled golden-rod (*Solidago rugosa*), tall golden-rod (*Solidago altissima*), fleabane (*Conzya canadensis*), small white aster (*Aster vimineus*), hawkweed (*Hieracium* spp.), teasel (*Dipsacus sylvestris*), purple loosestrife, Kentucky bluegrass (*Poa pratensis*), fescue grass (*Festuca* spp.), soft rush (*Juncus effusus*), and slender rush (*Juncus tenuis*) are also found growing in open areas.

Herbaceous Wetlands. Herbaceous wetlands are dominated by perennial or annual herbaceous vegetation. They are found in poorly drained depressions on higher topographic positions away from the drainage swales (Figure 6-9). Dominant herbaceous vegetation typically includes a mixture of common reed, uptight sedge, purple loosestrife, and soft rush. Other common associates in the herb layer are hop sedge (*Carex lupulina*), flat-top fragrant-golden-rod, love-vine (*Cuscuta* spp.), willow-herb (*Epilobium* spp.), South American vervain (*Verbena hastata*), Canada golden-rod, and wrinkled golden-rod. Shrubs such as stiff dogwood and red-osier dogwood and saplings of trees (e.g., green ash) are often found scattered through these communities.

Herbaceous Uplands. Herbaceous upland communities are dominated by early successional weed species typical of disturbed areas. Canada golden-rod, purple loosestrife, Kentucky bluegrass, and soft rush are dominant herbaceous species in this habitat type at Ashland 2. Strawberry, old field cinquefoil, flat-top fragrant-golden-rod, fleabane, small white aster, hawkweed, and slender rush are other associates in the herb layer. Woody species like stiff dogwood and hawthorn have become established in these herb-dominated areas.

Vegetated Drainage Swales. Vegetated drainage swales are found in poorly drained drainageways such as Rattlesnake Creek. They are dominated by nearly monotypic, dense stands of herbaceous species such as broad-leaf cattail, purple loosestrife, and common reed (Figure 6-10). Occasionally other herbaceous associates include wrinkled golden-rod, small white aster, New England aster (*Aster novae-angliae*), common boneset (*Eupatorium perfoliatum*), swamp milkweed (*Asclepias incarnata*), flat-top fragrant-golden-rod, willow-herb, and field mint (*Mentha arvensis*). Woody associates include scattered clumps of shrubs such as stiff dogwood or hawthorn saplings along the drier margins of the swales. These sites are generally too wet to support tree species.

Aquatic and Riparian Habitats

Surface water from the Tonawanda properties drains via Rattlesnake Creek and Twomile Creek to the Niagara River (Figure 6-1). At Strawberry and Grand Islands, the Niagara River

divides into two channels - the Chippawa Channel and the Tonawanda Channel. The Ashland 1 and 2 and the Seaway properties are located 150 m (500 ft) from the Tonawanda Channel of the Niagara River. The Linde property is located along the upper reach of Twomile Creek.

Rattlesnake Creek. Rattlesnake Creek is a natural channel formed from surface drainage from Ashland 1, Seaway, and Ashland 2. The 2,300-m (7,600-ft) channel drains 140 ha (340 acre) before joining Twomile Creek (Figure 2-5). Twomile Creek flows into the Niagara River approximately 300 m (1,000 ft) downstream from the confluence with Rattlesnake Creek (BNI 1992).

Drainage from Ashland 1 travels under the Seaway property through an underground concrete conduit and exits at the Niagara Mohawk property line. Rattlesnake Creek receives this drainage, crosses the Niagara Mohawk property, and then crosses the Ashland 2 property. Rattlesnake Creek receives about 60 percent of the surface runoff from Ashland 2. The creek channel is approximately 3 m (10 ft) wide and 1 m (3 ft) deep at full bank capacity, and has a slope of one percent on the Ashland 2 property. The channel and creek floodplain are vegetated with a thick growth of rushes and cattails, which limits flow velocities. The floodplain of Rattlesnake Creek is approximately 30 m (100 ft) wide at Ashland 2. From Ashland 2, the creek flows about 980 m (3,200 ft) before its confluence with Twomile Creek (Figure 2-5) (BNI 1992). A more detailed description of Rattlesnake Creek is included in Section 2 of the RI report (BNI 1992).

The drainage channels on the Ashland 1 and Seaway sites would not be expected to support any aquatic animal communities beyond those ordinarily found in manmade drainage systems. Water quality within these two areas is variable, but generally low (BNI 1988b). Flow begins within the bermed and level areas on Ashland 1. Runoff from the southwest slope of Seaway joins this flow and is conveyed by drainage ditches to the Seaway boundary, where it flows beneath the landfill through a 90-cm (36-in.) reinforced concrete pipe. Leachate infiltration into this pipe is suspected (Wehran 1979).

It is not known what type of aquatic community Rattlesnake Creek supports, if any. Fish kills in Twomile Creek have been reported and are attributed to poor water quality associated with Rattlesnake Creek and its tributaries. Leachate with a high ammonia concentration from the Seaway landfill was reported as responsible for a fish kill in Twomile Creek in 1972 (NYSDEC 1974). Water quality sampling studies indicate that water quality generally improves with distance downstream away from the Seaway and Ashland properties (Wehran 1979, Engineering-Science 1986).

Twomile Creek. Twomile Creek originates south of the Linde property in a natural channel (Figure 2-5), enters two underground culverts, and flows north through the Town of Tonawanda and the Village of Kenmore (SAIC 1992a). Runoff from Linde enters the conduits through five outfalls. The two conduits eventually discharge into the natural stream bed at the end of Sheridan Park Lake. Sheridan Lake has a surface area of about 1.2 ha (3 acres).

An aquatic survey was conducted on Sheridan Park Lake in 1980 (NYSDEC 1992a). Fish species collected included goldfish (*Carassius auratus*), bullhead catfish (*Ictalurus nebulosus*), goldfish x carp hybrid (*Cyprinus carpio*), black crappie (*Pomoxis nigromaculatus*), rock bass (*Ambloplites rupestris*), and yellow perch (*Perca flavescens*).

Twomile Creek continues northward from Sheridan Park Lake approximately 3 km (2 mi) until it empties into the Niagara River upstream from Niagara Falls. The slope of Twomile Creek is less than one percent. During periods of base flow in Twomile Creek, the water's surface width is about 6.1 m (20 ft) and the depth ranges from 0.6 to 1.2 m (2 to 4 ft). The depth increases as the creek approaches the Niagara River.

NYSDEC classifies Twomile Creek and its tributaries as Class B: "primary and secondary contact recreation and fishing. These waters shall be suitable for fish propagation and survival." Class B waters are protected under New York Environmental Conservation Law, Article 15.

No specific surveys of fish or invertebrates have been performed in Twomile Creek. However, information regarding aquatic invertebrate biota that may be considered typical of the Tonawanda site was obtained from previous aquatic surveys (NYSDEC 1992a). Survey locations and data include (1) Ransom Creek, near Clarence Center in Erie County and Tonawanda Creek, with communities typical of riffle habitats in local streams and creeks and (2) Cayuga and Bergholtz Creeks in the Niagara Falls area, with invertebrate species typical of slower-moving stream habitats. Dominant invertebrates in the Ransom Creek survey consisted of the following species: Chironomidae (midges), Trichoptera (caddisflies), Ephemeroptera (mayflies), Plecoptera (stoneflies), Coleoptera (beetles), and Oligochaeta (worms). Dominant species from the Cayuga and Bergholtz surveys were crayfish, Odonata (dragonflies), Gastropoda (snails), and hemipterans (*Belostoma* spp.)

A fish survey of Tonawanda Creek, performed in 1979 for FWS, lists 20 species including cyprinids (minnows), catostomids (suckers), ictalurids (catfish), centrarchids (sunfish), esocids (pike), and percids (perch) (COE 1981). Species from the lower section tended to be more representative of warm-water habitats. Although a smaller stream, Twomile Creek would be expected to support similar but fewer species.

6.1.4.4 Habitat Summary

The Tonawanda site comprises four properties, three of which have been highly modified such that little natural habitat remains intact. The fourth, Ashland 2, provides a diversity of naturally vegetated habitat types, including wetlands. Habitat at Linde consists primarily of landscaped areas at a highly industrialized site amid urban residential and commercial properties. Natural habitats in the vicinity of Linde are limited to the small wetland located offsite northeast of the plant. Ashland 1 is a highly industrialized site that has been heavily impacted by past operations. Virtually no natural habitat occurs at Ashland 1. The vegetation that has become

established is typical of highly disturbed areas. Seaway is an active solid waste disposal facility. It contains some native grasses, herbs and shrubs, and is visited by gulls and crows.

Ashland 2 contains a mosaic of upland and wetland ecosystems making it an attractive habitat for a variety of plants and animals, although the impacts of human activity are evident throughout the site. The primary ecological significance of habitat types at Ashland 2 is that the site represents a large (> 40 ha; > 100 acre) relatively undeveloped area surrounded by urban and industrial development that has significantly reduced natural habitat in the Tonawanda area. Aside from this, there is nothing unique about the property. Wildlife utilization at Ashland 2 is evident from direct observation, spoor, and other signs.

6.1.5 Contaminants of Ecological Concern

The ecological COCs are identified as those detected at the Linde, Ashland 1, Seaway and Ashland 2 properties with the potential to pose a hazard to the biota. Factors determining whether a contaminant qualifies as an ecological COC include: environmental concentration, frequency of occurrence, background levels, bioavailability, physical and chemical properties (e.g., solubility), potential for bioaccumulation, toxicity, and effects (EPA 1991b).

COCs at the Tonawanda properties were identified from a comparison of site and background concentrations, the frequency of occurrence, and sample quantification limits (see Section 2). Threshold concentrations for toxicity by two modes of exposure (aquatic, oral) for each potential ecological COC, and single thresholds for each of two measures of mobility (water solubility, soil sorption), and two measures of persistence (degradation half-life, bioconcentration factor) were chosen based on data in standard reference texts and compilation databases as described below. The toxicity, mobility, and persistence data for these potential COCs are given in Tables 6-1 and 6-2. The screening factors are described below.

6.1.5.1 Screening Factors

The primary screening factor for ecological COCs is whether the concentration of the contaminant at the site exceeds a threshold of toxicity. A screening process based solely on toxicity and environmental concentrations neglects the potential for contaminants to become concentrated with time in the environment or in organisms, or the possibility that a contaminant currently exceeding toxicity concentrations may not persist at high concentrations long enough to pose a continuing risk to ecological receptors. The two mobility and two persistence thresholds (and the screening rules in Section 6.1.5.2) begin to bring considerations of the physical and chemical properties of contaminants into the ecological risk assessment process (EPA 1991b). As more information is available about the behavior of contaminants in the environment and the chemical forms to which organisms are actually exposed, this screening process will embody fewer uncertainties. Until then, any contaminant not qualifying as an ecological COC because of the mobility and persistence criteria should be scrutinized carefully.

Toxicity Threshold Concentrations

Toxicity thresholds for each COC found at Tonawanda were based on toxicity data obtained from compiled toxicological databases: IRIS (EPA 1992b), HSDB (1992), AQUIRE (1992), RTECS (1992). Published toxicity data were used in the following order of preference:

- U.S. government standards;
- concentrations showing no effect;
- chronic toxicity concentrations; and
- acute toxicity concentrations.

In all cases, the appropriateness of study methods, chemical species, and test organisms relative to the Tonawanda site were considered.

The first choice for toxicity thresholds was U.S. government established standards such as EPA Water Quality Criteria, Aquatic (WQCAQ), or the No Observed Adverse Effect Levels (NOAEL) from animal studies, which were used to set Health Advisory reference doses (EPA 1992b). When these values were available, they were used to set a threshold, regardless of other data.

The lowest published concentrations showing "no effect" (e.g., NOAEL) were the first choice for toxicity thresholds when a U.S. government water quality criterion was unavailable. This is a conservative threshold for species that are equally or less sensitive than the tested species.

When "no effect" levels were unavailable, thresholds were based on the lowest published toxicity concentrations available. For aquatic organisms, toxicity is usually quantified by either the acute or chronic LC50, the concentration of toxicant in ambient water at which 50 percent of the exposed organisms die. For this ERA, acute toxicity is defined to be 96 hours or less; chronic toxicity is longer than 96 hours. Acute and chronic oral LD50s, the concentration of toxicant in the diet that causes 50 percent mortality, or similar measures of toxicity were used for elements and compounds that remain adsorbed to the soils or sediments. For some contaminants in sediments, "Effects Range-Low" (ER-L) concentrations have been identified (Long and Morgan 1991) from mostly marine and estuarine data. They are not expected to apply to oral toxicity in freshwater sediments or soils. When U.S. government standards or NOAEL values were not available, ER-Ls can, in principle, be used as the basis for setting an aquatic toxicity threshold, but ER-Ls were not used in the Tonawanda ERA.

Toxicity thresholds were set below chronic values when information on chronic toxicity was available. This approach to using chronic toxicity values is conservative but reasonable, because there is often complete uncertainty about where the threshold lies in relation to the no-effect level for the organisms actually found at the Tonawanda sites.

When chronic values were lacking, acute toxicity values were modified for use as threshold values. The acute value was divided by 45 according to the Rule of Michigan (Michigan Water Resources Commission 1986), and the toxicity threshold was set at or below the quotient. Using this rule to calculate chronic aquatic toxicity thresholds theoretically protects 95 percent or more of all fish and aquatic invertebrate families from adverse effects 80 percent of the time. Thresholds set below this quotient are expected to be even more conservative to compensate for the uncertainty injected by using a fixed reduction factor and toxicity data for organisms other than those found at the Tonawanda site. Acute oral toxicity values were also divided by 45 as a guide to establishing a chronic oral toxicity threshold.

The available toxicity data for the 62 potential ecological COCs, including government standards, are given in Table 6-1. The toxicity threshold concentrations established using these data are given in Table 6-2. Aquatic thresholds are for surface and groundwater environmental media, although groundwater was not considered in this ERA. Oral thresholds are for soil and sediment. Threshold values other than government standards reflect the paucity of directly pertinent wildlife toxicity data and uncertainty about no-effect levels for the organisms and potential COCs at Tonawanda. In all cases, the primary consideration was to choose a conservatively low threshold value to reflect these uncertainties. For example, the single datum found for the chronic oral toxicity of selenium was for laboratory mice. Accordingly, the toxicity threshold was set at 100 mg/kg, below the published TDLo of 134 mg/kg.

Mobility Thresholds

Mobility is indicated by water solubility and, for organic compounds, by soil sorption, that is, the organic carbon-water partition coefficient (K_{oc}). A threshold of 1 mg/L was chosen to represent the level of water solubility above which a potential toxicant is considered sufficiently mobile in water to present a potential hazard to aquatic organisms in surface waters or organisms exposed to groundwater. All substances identified in Table 6-1 as "insoluble" are included with those having water solubility < 1 mg/L. In addition to water solubility, a soil sorption K_{oc} of 1,000 was used as a threshold above which a contaminant would not be considered a hazard via aquatic exposure pathways. All those listed in Table 6-1 as "high" are considered to be above threshold. This threshold value was chosen following a review of the comments on soil adsorption and mobility of contaminants with varying K_{oc}s (Howard 1990). Contaminants with K_{oc}s > 1000 pose a hazard to terrestrial organisms via soil ingestion if they are above toxicity thresholds and persistence thresholds.

Persistence Thresholds

Persistence is indicated by the bioconcentration factor (BCF) and the degradation half-life of a substance in water, soils, or organisms. A half-life threshold of 14 days was selected for degradation (Gillette 1983). A half-life greater than 14 days indicates that a substance can be considered persistent at the site. Assuming no continuous source of contaminant, a half-life <14 days means that a contaminant will be reduced in concentration by eight orders of magnitude in a little more than 12 months. All substances identified in

Table 6-1 as being "persistent" are included with those having a half-life > 14 days. The BCF is the tissue concentration of a substance divided by its concentration in the environment. A threshold of 100 was chosen for BCF. A BCF above 100 indicates that a toxicant can become magnified in organisms 100 times over the source concentration and thus represents a potential persistent hazard.

6.1.5.2 Screening of Potential Ecological COCs

Not all the 62 potential ecological COCs (Table 6-1) could or needed to be screened for inclusion as ecological COCs. The three radionuclides are ecological COCs by virtue of their environmental concentrations (2 × background) and the uncertainty concerning their effects on ecological receptors. Four essential biological minerals — calcium, magnesium, potassium, and sodium — are not screened, rather they are assumed not to be ecological COCs for the Tonawanda site. These essential elements can be toxic in certain chemical forms and at very high concentrations, but government standards, or data on which to base a toxicity threshold, do not exist.

The remaining 55 potential ecological COCs are screened according to the following rules:

1. If the mean environmental concentration at the site does not exceed the toxicity-threshold concentration level, both a mobility and a persistence threshold must be exceeded for the contaminant to qualify as an ecological COC;

2. If the mean environmental concentration at the site exceeds the chronic toxicity-threshold concentration, then the contaminant's status as an ecological COC depends on whether there is a continual source of the contaminant. The subrules are as follows:

   a. If there is a continual source, the contaminant is an ecological COC.

   b. If there is no continual source, the contaminant qualifies as an ecological COC if it exceeds either the mobility or persistence threshold. A contaminant without a source that is neither mobile nor persistent is not an ecological COC, even though its concentration currently exceeds its chronic toxicity threshold.

3. In those cases where a toxicity-threshold concentration could not be established, the contaminant is defined as an ecological COC;

4. In those cases where there is no mobility or persistence data, the determination depends only on the toxicity-threshold concentration.

Rule 1 ensures that contaminants currently below toxic concentrations at the site, but with the potential to increase in concentration through their persistence and biomagnification,

are considered in the BRA. For example, some forms of lead are both soluble and persistent. Even if average concentrations of lead at Ashland 2 were below chronic toxicity levels, it could, over a sufficient period of time accumulate and concentrate in organisms (BCF > 100 in some invertebrates) to such an extent that body burdens could exceed the toxicity concentrations for predator receptors, such as raccoon. Lead qualifies as an ecological COC at one or more Tonawanda properties because its mean environmental concentration (Table 6-3) exceeds toxicity-threshold concentrations (Table 6-2). Even though their environmental concentrations were below their toxicity-threshold concentrations, three contaminants, molybdenum, chlorobenzene and xylenes, qualified as ecological COCs because of Rule 1.

Rule 2, in general, removes from the risk assessment those contaminants that are unlikely to pose a threat even though they are currently at concentrations above their chronic toxicity thresholds. Chemical contaminants that are immobile, not persistent, and for which there is not a continual source are unlikely to exceed background concentrations for an extended period of time (e.g., the several years it takes to investigate and remediate a site), because several years is generally long enough for the chemical to have significantly degraded. Rule 2a ensures against ignoring contaminants that pose a continuing hazard because they have a continual source. Contaminants that do not meet the mobility and persistence criteria, and for which there is no continual source, are unlikely to pose a continuing threat to organisms at the site or nearby, because they will be reduced by up to eight orders of magnitude in 56 weeks or less given a half-life of, at most, 14 days. No contaminant in this Tonawanda ERA was removed from further consideration as an ecological COC because it did not meet the mobility and persistence criteria (Rule 2b). Chemicals did not meet both these conditions because, generally, chemicals that are highly soluble in water are also more highly degradable and do not bioconcentrate in organisms. Chemicals that bioconcentrate greatly in organisms are generally the hydrophobic, lipophilic substances, that are immobile and persistent, especially in soils and sediments, Benzo(b)fluoranthene, for instance, has a water solubility of 0.0012 mg/L and a reported BCF of 760,000. For comparison, the solubility of benzoic acid in water is 2700 mg/L and its BCF in trout is 0.4 mg/L. Therefore, both aquatic organisms (e.g., fish) and terrestrial organisms, including animals that ingest soils (e.g., earthworms), are protected by Rule 2b, which requires a contaminant to be both immobile (i.e., low water solubility, high K_{oc}) *and* not persistent (i.e., short half-life, low BCF) before it can be removed from further consideration as an ecological COC.

Rule 3 ensures that potentially dangerous contaminants are not excluded as ecological COCs strictly for lack of toxicity data. This is a conservative assumption.

Rule 4 reflects the fact that toxicity is the primary consideration, but practicality argues against giving ecological COC status to contaminants with environmental concentrations below toxicity thresholds solely because of the lack of mobility or persistence data. This rule is conservative.

By this screening process, 33 of the 62 potential ecological COCs in Table 6-1 qualify as ecological COCs in one or more environmental media in Tonawanda remedial units. For

example, boron qualifies as an ecological COC in Ashland 2 surface waters because its mean concentration (6607 μg/l) exceeds the aquatic toxicity threshold (1,000 μg/l) and it has a high BCF (1,000). Boron does not qualify as an ecological COC in Linde soils, on the other hand, because its mean concentration (53.3 mg/kg) does not exceed the oral toxicity threshold (1,000 mg/kg). Table 6-2 indicates for each potential ecological COC whether it does (Y) or does not (N) meet each criterion for environmental media, the final determination of its status, and the properties for which it is an ecological COC.

The ecological COCs for Tonawanda are:

Radionuclides

- Radium
- Thorium
- Uranium

Metals

- Aluminum
- Barium
- Cadmium
- Copper
- Manganese
- Nickel
- Thallium
- Antimony
- Beryllium
- Chromium
- Iron
- Mercury
- Selenium
- Vanadium
- Arsenic
- Boron
- Cobalt
- Lead
- Molybdenum
- Silver
- Zinc

Organics

- Benzo(b)fluoranthene
- Chlorobenzene
- Pyrene
- Benzo(k)fluoranthene
- Dibenzofuran
- Trans-1,3-dichloropropene
- Benzo(g,h,i)perylene
- Indeno(1,2,3-cd)pyrene
- Xylenes

Each ecological COC is examined further in terms of exposure characterization (Section 6.2), ecological effects assessment (Section 6.3), and ecological risk characterization (Section 6.4).

6.2 EXPOSURE ASSESSMENT

Exposure assessment includes quantification of release, migration, and fate of contaminants, characterization of receptors, and quantification of concentrations at the point where organisms are actually exposed (EPA 1991b). Environmental concentrations of ecological COCs at the Tonawanda site are quantified in Table 6-3. This exposure assessment focuses on characterizing receptors by the different possible pathways and modes of exposure to contaminants at the Tonawanda site.

Environmental concentrations of site contaminants are adjusted for various classes of receptor species according to how their pathways and modes of exposure dilute or concentrate the contaminant. Some classes of receptors are exposed by multiple routes, and their risk will be greater than those organisms exposed to contaminants at less than the full environmental concentration. For example, carnivorous fish are exposed to contaminants in ambient water and in their prey. Burrowing rodents, on the other hand, are primarily exposed to contaminants via direct contact with and ingestion of soils, which make up only a fraction of their diet. The resulting exposure concentrations are used to characterize the risk to the nonhuman populations (Section 6.4). This approach to ecological pathway analysis recognizes the potential for contaminant residues to bioconcentrate (concentrate in aquatic organisms exposed to contaminants in ambient media), bioaccumulate (concentrate in aquatic and terrestrial organisms from dietary as well as abiotic sources), and biomagnify (systematic concentration as chemicals are passed from prey to predator), as well as the possibility that organisms are exposed to diluted environmental concentrations.

Contaminant sources at the Tonawanda site include surface soils at Linde, Ashland 1, and Ashland 2, as defined in Section 2. Eventually these contaminants might leach to groundwater. Leaching of soil contaminants may also affect surface water and sediments in Rattlesnake Creek and its tributaries on these properties. Alluvial transport of contaminated water, sediments and soil from these properties can potentially contaminate sediments and surface waters in downstream reaches of Rattlesnake and Twomile Creeks and the Niagara River (Figure 6-1). Thus, when evaluating the exposure of aquatic and terrestrial biota to ecological COCs from Tonawanda sources, water-soluble contaminants in surface waters and insoluble contaminants in soils and sediments are considered the primary sources of risks.

A pathway analysis can link contamination in the biota directly and indirectly to contaminant sources (e.g., soil, sediment, surface water), via mechanisms of release to the environment and the movement of contaminants through the ecosystem (Figure 6-11). Exposures occur over direct and indirect pathways from contaminant sources to ecological receptors. Direct exposures of an organism to a contaminant do not involve intermediary organisms; indirect exposures do. Exposures may be internal or external and passive or active with respect to the receptor. Internal exposure occurs when the contaminant directly enters into the body, usually by the ingestion of contaminated material, whereas external exposure occurs by dermal contact. External exposures are, by definition, direct. Passive exposures are unavoidable exposures; direct external exposure is usually unavoidable by those organisms living in the contaminated medium. Direct exposure is assumed when an organism lives in a contaminated medium. Internal exposures, here termed "active," can result from direct ingestion of contaminated abiotic material or indirectly from ingesting contaminated organisms. [Note that internal and external exposure pathways discussed in this context for the exposure of ecological receptors to chemicals differ from those defined previously for human exposures to radioactive contaminants (Section 4.1), which can cause "internal" exposure from outside the body]. Indirect pathways of exposure are best identified with a food web.

Figure 6-12 is a schematic representation of aquatic and terrestrial food webs that typify the Tonawanda site. Food webs generally comprise the following trophic groups:

- primary producers - green plants such as grasses, shrubs, trees, in terrestrial ecosystems, and algae, periphyton, and hydrophytes in aquatic ecosystems;

- primary consumers (herbivores) - animals that feed on plants; for example, white-tailed deer and cottontail rabbits in the terrestrial food web and ducks, fish, and certain benthic invertebrates in the aquatic food web;

- secondary consumers (omnivores/carnivores) - animals that feed on both plants and animals or feed strictly on other animals; for example, raccoons in the terrestrial food web and yellow perch and carnivorous fish in offsite aquatic food web; and

- decomposers - including certain fungi and bacteria.

Primary producers can mobilize contaminants from soils and sediments. This can occur by foliar absorption of contaminants deposited on leaf and stem surfaces, or by uptake via plant roots. Uptake of contaminants by plants could lead to subsequent exposure to herbivores and omnivores from ingestion of the contaminated vegetation. Contaminants that bioaccumulate in primary producers or their animal consumers or bioconcentrate in organisms directly exposed to contaminated media often further accumulate in secondary consumers (i.e., carnivores and omnivores). The last link in the food chain can be represented by transient secondary consumers—top predators such as the osprey (*Pandion haliaetus*) or peregrine falcon (*Falco peregrinus*). Each is listed as threatened or endangered and could occur as transients in the Tonawanda area.

Organisms at the Tonawanda site are potentially exposed to contaminants by one or more of these pathways (Figure 6-11). Internal exposure via ingestion of contaminated matter is considered here to be the primary mode of exposure to chemical contaminants for nonburrowing terrestrial animals. These will have additional but secondary exposures from direct contact with contaminated soils and surface waters, inhalation of fumes or dust and, where applicable, direct radiation by radionuclides. Subterranean organisms, e.g., rats and rabbits, will receive primary exposure by direct contact with (and inhalation of) contaminated soils. They will receive secondary exposure from ingestion of contaminated soils and groundwater. Only a few organisms of a limited number of types (e.g., fossorial rodents) are expected to reside within Linde, Ashland 1, and Ashland 2 contaminated soils. Direct external exposures are expected to be the primary mode of exposure for aquatic organisms, as opposed to trophic exposure, because of their mode of existence and because chemicals with high water solubility are not likely to have a high bioconcentration factor (Howard 1990). Aquatic organisms are expected to be exposed secondarily via ingestion of contaminated sediments and biota.

Species of aquatic and terrestrial organisms were selected from the list of those identified at the site (Section 6.1.4) to serve as proxies for the many species constituting the ecological communities at Tonawanda. The term "proxy" is used instead of "indicator" because no explicit measurement endpoint for these species has been identified for this risk assessment, and therefore there is nothing to indicate. Rather, they serve as substitutes for larger numbers of species that are potentially exposed to ecological COCs by similar modes and pathways. Additional criteria for selecting species to represent onsite communities are: (1) species closely related to toxicological test organisms; (2) economically important species (game animals, species consumed by humans); (3) abundant or common species (in their respective animal communities); (4) endangered, threatened, or listed species; and (5) key food-web species that might be sensitive to the Tonawanda ecological COCs. To identify key trophic species requires at least a quantified site-specific food web for the Tonawanda site, which is not feasible for this ERA.

Each of the three Tonawanda contaminant source media is linked by direct or indirect exposure pathway to ecological receptors (Table 6-4). Receptors are of two types: onsite and offsite. Onsite receptor species are those that utilize the Tonawanda site properties. Offsite receptors are those living outside the site boundaries but potentially exposed to contaminants via offsite movement of abiotic or biotic media; these include common and threatened and endangered species. Onsite proxy species are found at one or more of the Tonawanda properties. Proxy organisms chosen to represent the aquatic and terrestrial communities in the Tonawanda area are:

Aquatic	Terrestrial
midge larvae (onsite)	rabbit (onsite)
mallard duck (offsite)	squirrel (onsite)
carp (offsite)	rat (onsite)
yellow perch (offsite)	robin (onsite)
muskrat (offsite)	raccoon (onsite)
osprey (offsite)	

These species serve as proxies for the biotic communities in the effects assessment and risk characterization phases of the risk assessment.

The concentration of a contaminant to which ecological receptors are potentially exposed, i.e., exposure concentration, depends on the pathway and mode of exposure. Organisms exposed externally to contaminated media by direct contact are exposed to the full environmental concentration for the period of time they reside in the media. The exposure concentration for organisms that ingest contaminated media must be corrected for the fraction of their diet that is contaminated. Organisms exposed indirectly via the food web experience an environmental concentration determined by the fraction of their diet that is contaminated and the concentration of contaminant in their food, which will be a function of the

bioconcentration factor for the contaminant and organism. These internal exposures must be added to external exposures for organisms living in contaminated media. Due to the considerable uncertainty surrounding these calculated exposure concentrations, they are not used to determine ecological COCs, rather they form the basis for characterizing the risk to ecological receptors from ecological COCs at the Tonawanda properties (Section 6.4).

6.3 EFFECTS ASSESSMENT

An effects assessment quantitatively links concentrations of contaminants to adverse effects in receptors (EPA 1991b). Because no site-specific toxicological studies have been conducted, this effects assessment uses the data in Table 6-1, which were obtained from compiled databases [e.g., IRIS (EPA 1992b), HSDB 1992, AQUIRE 1992, RTECS (NIOSH 1992)]. Information on test concentrations, modes of exposure, and effects on test species that were similar to those at the Tonawanda site, e.g. rat, mouse, rabbit, was used to establish toxicity-threshold concentrations (Table 6-2). Here we describe the nature of effects on organisms of radiological and chemical contaminants.

Available data document some of the possible acute or chronic toxic effects on the nonhuman biotic receptors [e.g., minnows, squirrels, and others (or their proxies)] in the Tonawanda environment. Both terrestrial and aquatic biotic receptors are considered. Information describing chemical uptake or accumulation of radionuclides by plants and animals is limited and generally based on short-term, high-exposure laboratory experiments. Those studies may not apply to the long-term, low-level exposures presented at Tonawanda.

Chronic toxicity of contaminants at Tonawanda is the primary concern in the Tonawanda effects assessment. Many contaminants observed to date at Tonawanda, especially metals and volatile organics, are persistent in the environment because they are insoluble in water and remain as solids in soils or bioconcentrate in organisms. Although metals can occur in high concentrations in soils, most organisms do not ingest large amounts of soil and thus are unlikely to be exposed to concentrations of metals above acute toxicity thresholds. This may not be true of the volatile organics, but these are not likely to persist at or above acute concentrations over the duration of remedial activity at the site. No investigations into chronic effects on local biota as a result of exposure to wastes have been conducted at the Tonawanda site, nor have analyses been performed to determine the radionuclide or chemical contaminant concentrations in the tissues of the biota. Also, there have been no rigorous population inventory or characterization studies.

6.3.1 Radiation Toxicity

Some biological effects from radiation, such as chromosomal aberrations and organ failure, occur similarly among different species of biota. However, except for warm-blooded species, most biota are more resistant than humans to radiotoxicity effects. The National Council on Radiation Protection and Measurements (NCRP) conducted a review of available

information on the effects of ionizing radiation on aquatic biota (NCRP 1990) and concluded that no deleterious effects could be detected for radiation dose rates below 1 rad/day. Fertility and fecundity of organisms and embryonic development were found to be the most sensitive radiation response endpoints for aquatic biota, with somatic effects and mortality occurring only at much higher dose levels.

The interaction of plants with radionuclides can occur by foliar absorption of radionuclides deposited on leaf and stem surfaces or by uptake by plant roots. Information describing uptake and accumulation of radionuclides by plants is based mostly on short-term, relatively high-exposure laboratory experiments (Knight 1983) that may not be applicable to long-term, low-level exposure conditions such as those at the Tonawanda site. Of the radionuclides present in site wastes, Ra-226 appears to have the highest potential for uptake and accumulation by plants because it serves as an analog for calcium, an essential plant nutrient (Knight 1983).

Uptake of radionuclides by plants could lead to subsequent animal exposure via ingestion of contaminated vegetation. An important issue may be the potential for plants and animals to serve as vectors for the transport of radioactive contaminants from the Tonawanda site to humans or other biota. For example, burrowing animals can bring the contaminants to the surface, and animal burrows can lead to increased water infiltration. Additional modes include transport of contaminated soils brought to the surface by animals, and movement of radionuclides by predators feeding on contaminated prey (Arthur et al. 1986).

6.3.2 Chemical Toxicity

Chemicals in the ecosystem may be directly toxic to biota, or they may decrease a population's ability to survive and reproduce by decreasing reproductive rates, reducing the viability of offspring, causing alterations in behavior patterns, or increasing susceptibility to disease or predators. These disparate endpoints are characterized by different dose responses and result from different exposure pathways. Therefore, for risk characterization, it is necessary to specify what exposure pathways and endpoints are being assessed.

Toxicity of chemicals in water depends on the mode of exposure as well as the availability of the chemical to the target organism. The primary mode of exposure to aquatic organisms to dissolved contaminants, direct contact, is also the mode with greatest likely toxicity. Ingestion of contaminated water, sediments or biota will be modes of lower but additional toxicity to aquatic receptors. Aquatic toxicity can also depend on temperature, hardness of the water, and presence of other chemicals.

Toxicity of soil contaminants varies depending on the receptor species and on the attending physical and chemical factors such as pH, the presence of complexing agents, or other chemicals at the site. Some soil microorganisms live in the film of water surrounding soil particles and would be exposed by direct contact to full environmental concentrations. Others live in the air spaces where inhalation of volatile or semivolatile organic contaminants

could lead to greater toxicity than by direct contact or ingestion. Toxicity of soil contaminants to burrowing organisms could be expected to be most serious due to the multiple modes of exposure.

Plants grown in soils containing metals can accumulate higher-than-background levels of some metals. Because the ratio of plant uptake to substrate concentrations of metals is not linear, it is difficult to determine the soil concentrations of metals that are toxic to plants. Bioaccumulation is generally most significant in the roots of plants; however, several metals can be translocated to aboveground parts of the plants. Some metals (e.g., mercury) accumulate in animal tissues and can have subtle deleterious effects over long exposure times. Many of organic contaminants (e.g., PCBs) and pesticides are extremely lipophilic and can biomagnify in organisms.

6.4 RISK CHARACTERIZATION

Risk characterization compares exposures to effects (EPA 1991a). An evaluation of the risk of the ecological COCs at the Tonawanda site forms the basis of this risk characterization (1992d). No well-established methods exist for calculating the risks to ecological receptors, however, the use of quotient methods is supported by available guidance (EPA 1989b, 1991c). This ratio or "ecological quotient" (EQ) approach compares the environmental concentration of a contaminant to its toxicity threshold concentration. Any quotient greater than or equal to unity indicates that there is the potential for adverse ecological effects, and the more the ratio exceeds unity the greater the risk of potential effects. EQs were also used to characterize the relative risk to the ecological receptors, in general, from the ecological COCs at Tonawanda properties. In addition, the relative risks of ecological COCs to ecological receptors exposed via different modes and pathways are assessed using exposure quotients (XQs), the ratio of exposure concentrations (i.e., the environmental concentration corrected for exposure) to the toxicity threshold concentration.

6.4.1 Current Risks

Calculating EQs and XQs requires a toxicity threshold for each contaminant for the appropriate mode of exposure. The toxicity thresholds used in Section 6.1.5 to screen contaminants as ecological COCs in surface waters, soils, and sediments at the Tonawanda properties (Table 6-2) are also used to calculate these quotients. As described in Section 6.1.5.1, aquatic thresholds are based on WQCAQs or aquatic toxicity data (Table 6-1) and oral thresholds are based on oral toxicity data (Table 6-1).

The aquatic and oral toxicity thresholds for the identified ecological COCs are:

Radionuclides

• Radium	NA	NA
• Thorium	NA	NA
• Uranium	NA	NA

Metals

• Aluminum	NA	1000 mg/kg
• Antimony	30 μg/l	0.35 mg/kg
• Arsenic	190 μg/l	10 mg/kg
• Barium	1000 μg/l	1 mg/kg
• Beryllium	5 μg/l	0.5 mg/kg
• Boron	1000 μg/l	1000 mg/kg
• Cadmium	1 μg/l	10 mg/kg
• -Chromium (III)	120 μg/l	1000 mg/kg
-Chromium (VI)	11 μg/l	2.4 mg/kg
• Cobalt	10 μg/l	1 mg/kg
• Copper	6.5 μg/l	1 mg/kg
• Iron	NA	50 mg/kg
• Lead	3.2 μg/l	1 mg/kg
• Manganese	NA	500 mg/kg
• Mercury	0.012 μg/l	1 mg/kg
• Molybdenum	500 μg/l	50 mg/kg
• Nickel	160 μg/l	5 mg/kg
• Selenium	5 μg/l	100 mg/kg
• Silver	0.12 μg/l	1 mg/kg
• Thallium	NA	NA
• Vanadium	NA	10 mg/kg
• Zinc	110 μg/l	10 mg/kg

Organics

• Benzo(b)fluoranthene	NA	NA
• Benzo(k)fluoranthene	NA	NA
• Benzo(g,h,i)perylene	NA	NA
• Chlorobenzene	10 μg/l	14 mg/kg
• Dibenzofuran	NA	NA
• Indeno(1,2,3-cd)pyrene	NA	NA
• Pyrene	NA	75 mg/kg
• Trans-1,3-dichloropropene	NA	NA
• Xylenes	250	250 mg/kg

Table 6-5 lists the EQs for the Tonawanda ecological COCs in the various source media. These were calculated for both mean and the 95 percent upper confidence limit (UL_{95}) on the mean (Table 6-3) by dividing the concentration by the toxicity-threshold values listed above:

$$EQ = \frac{\textit{Environmental Concentration}}{\textit{Toxicity Threshold Concentration}}$$

In a few cases, an EQ could not be calculated for an ecological COC because insufficient data were available to establish a toxicity threshold. For the characterization of relative risk, the UL_{95} concentration or the maximum, whichever is less, is taken as the RME. When calculated as the ratio of the uncorrected RME to the toxicity threshold concentration, EQs represent an estimate of the risk to biota based on the RME concentration. This assumes that the environmental concentration of the ecological COC is not increased by physical or biological processes in the transport and exposure pathways.

To further characterize the relative risk to various classes of receptors, XQs were calculated for each ecological COC at each of the Tonawanda properties. Environmental concentrations at the Tonawanda site (Table 6-3) were multiplied by exposure factors (Table 6-6) to calculate hypothetical exposure concentrations for each ecological COC at each OU. Hypothetical exposure concentrations are divided by toxicity threshold concentrations to give XQs:

$$XQ = \frac{\textit{Environmental Concentration x Exposure Factor}}{\textit{Toxicity Threshold Concentration}}$$

To derive hypothetical exposure factors (Table 6-6), the following assumptions regarding chemical behavior, exposure duration, and diet were made because there have been no site-specific ecological studies. For the purposes of this assessment of relative risk to ecological receptor classes, we assume:

1) Onsite receptors exposed by direct contact to contaminants are exposed 100 percent of the time to contaminants at their measured environmental concentration (Table 6-3).

2) Offsite receptors are not exposed by direct contact.

3) Ten percent of what sediment or soil dwelling animals ingest is contaminated sediments or soils.

4) Onsite receptors obtain 100 percent of their diet from contaminated prey.

5) Offsite receptors obtain 10 percent of their diet from contaminated prey.

6) The contaminant concentration in terrestrial prey organisms is equal to the environmental concentration times a suitable bioconcentration factor (Table 6-1).

7) Biomagnification does not occur.

These assumptions, which are for the purpose of calculating XQs only, do not hold in all cases. For example, the potential for biomagnification of certain contaminants (e.g., methylmercury) could cause the relative risk to receptors exposed via trophic pathways to be underestimated under assumption 7. Mercury is an ecological COC in Ashland 2 (North)/Seaway surface water. These assumptions should suffice to calculate XQs and to distinguish different classes of onsite and offsite receptors by the relative magnitude of the risks from exposure to contaminants at the Tonawanda site.

The hypothetical exposure factors in Table 6-6, resulting from the above assumptions, are explained as follows. For aquatic and subterranean organisms, the primary exposure pathways are direct contact with and consumption of contaminated media. For nonburrowing terrestrial organisms, the primary exposure pathway is assumed to be consumption of contaminated biota. For exposure by direct contact alone, environmental concentrations are a conservative estimate of exposure concentrations. This is the case for aquatic animals that do not dwell in the sediments (e.g., most fish). Exposure concentrations for aquatic organisms that live in sediments (e.g., midge larvae), and subterranean animals in soils (e.g., rabbits) are calculated as 110 percent of the environmental concentration at their locations; 100 percent by direct exposure and an additional 10 percent by ingestion of contaminated media. To calculate the exposure concentration for receptor species exposed to ecological COCs via the trophic pathway (e.g., raccoons), the environmental concentration is multiplied by the lowest available published BCF for a possible prey organism. The lowest BCF is used to balance the neglect of factors that dilute exposure, such as the fraction of a predator's diet that is uncontaminated. Direct exposure to contaminated soil and water is not included as a secondary source of risk to nonburrowing terrestrial organisms. In the summary that follows, the characterization of risk to the different ecological receptors is based on XQs calculated by multiplying EQs by the hypothetical exposure factors in Table 6-6, as described in Section 6.4.1.

The ecological COCs having an XQ >10 for the different pathways and modes are given in Table 6-7 for the different classes of receptor organisms. In the summary that follows, the characterization of risk to the different classes of receptors is based on these modified EQs. Characterization of risks to ecological receptors in general at the different Tonawanda properties is based on EQs (Table 6-8, 6-9, and 6-10).

Overall, the heavy metals, especially lead and copper, pose the greatest risk to ecological receptors at the Ashland 1, Ashland 2 (South), and Linde properties at the Tonawanda site (Tables 6-7 and 6-8). The effects of metals, in general, and copper and lead, in particular, on organisms are well characterized (Section 6.3). With these contaminants at such high concentrations above toxic thresholds, deleterious effects on both ecosystems (e.g., community structure, primary production) and organisms (e.g. animal behaviors, reproduction)

are highly probable. Copper, lead, selenium, and silver present the greatest risk (XQ >10) to both onsite and offsite aquatic receptors (Table 6-7). Onsite aquatic receptors are also exposed to similar level of risk from mercury, chromium, cadmium, cobalt, boron, and to an unknown degree, molybdenum, pyrene, trans-1,3-dichloropropene and xylenes. Subterranean organisms exposed via direct contact with and ingestion of soils are exposed to serious risk (XQ > 10) from nine metals and seven organics (Table 6-7), because their XQs are 1.1 times as great as the calculated EQs for these contaminants. Terrestrial organisms exposed on site via trophic pathways are exposed at the same level of risk (XQs > 10) to metals -- arsenic, barium, cobalt, copper, lead, nickel, vandium and zinc -- and to an unknown degree of risk from organics -- benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, indeno(1,2,3-cd)pyrene, dibenzofuran, and xylenes (Table 6-7). The XQs for trophic exposure to lead and copper in Ashland 1 soils are high because of their potential to bioconcentrate (Table 6-1). This strongly suggests that lead and copper pose a significant risk to terrestrial organisms via the food web at Ashland 1. Major sources of risk for Tonawanda ecological receptors (Table 6-7) indicate that these ecological COCs also pose a potential risk to onsite predators at Ashland 1 even if current environmental concentrations are below toxicity levels as measured in laboratory experiments. Offsite predators are most at risk from all of these except cobalt, nickel, chlorobenzene and the xylenes. Xylenes would not be expected to bioconcentrate to 10 times the toxicity threshold, which is greater than one million times the respective environmental concentrations of xylenes.

Copper, lead, selenium and zinc pose the greatest risk (EQ >100) at Ashland 1 surface water (Table 6-8) and soils (Table 6-9). Cadmium, chromium, and cobalt and to a lesser extent, antimony, arsenic, beryllium, and nickel also present serious risks to organisms exposed to surface waters at Ashland 1 (Table 6-8). Vanadium in soils poses a lower risk (EQ < 100). The organics in soils and surface waters at Ashland 1 -- benzo(b)fluoranthane, benzo(k)fluoranthene, pyrene, and trans-1,3-dichloropropene -- pose an unknown degree of risk because of the lack of toxicity data.

In addition to copper, lead, and selenium, exposure to Ashland 2 (South) surface water presents risk from silver (EQ >100) and to lesser extent chromium (Table 6-8). The major contaminants in Ashland 2 sediments are aluminum and zinc (Table 6-10). Ashland 2 (South) soils have cobalt, vanadium, nickel, and beryllium at concentrations exceeding their soil toxicity thresholds as well as copper, lead, and zinc with EQs >100 (Table 6-9). Concentrations of chlorobenzene and xylenes in Ashland 2 (South) soils did not exceed their toxicity thresholds, but their mobility and potential to persist in the environment make them potential risks of unknown magnitude to organisms.

The ecological COCs at Ashland 2 (North)/Seaway that pose the greatest risk to ecological receptors exposed to surface waters are lead, copper, boron, and mercury, and, to a lesser extent, boron, chromium, zinc, and xylenes (Table 6-8). Aluminum, manganese and vanadium are the primary source of risk in Ashland 2 (North)/Seaway sediments (Table 6-10). No surface soil contaminant concentration data were available for Seaway soils.

Linde soils pose high levels of ecological risk from lead, copper, barium, arsenic, nickel, and an unknown level of risk from the benzofluoranthenes, indeno (1,2,3-cd) pyrene, and dibenzofuran due to their environmental concentrations and the possibility of bioconcentration in organisms (Tables 6-7 and 6-9). To a lesser extent, beryllium and manganese pose a risk at Linde. The Linde property is so highly modified with very little natural habitat that the actual risk to ecosystem structure or function is small, although the risk to individual organisms residing at or visiting the site may be large.

6.4.2 Future Risks

The risks to the biota at the Tonawanda site can be considered long-term risks. Toxicity threshold concentrations were based on subacute exposure levels. Based on their half-lives, K_{oc}, and water solubilities, the ecological COCs at the site can be expected to persist for extended periods of time. Unremediated, the many ecological COCs with EQs exceeding 10 indicate that they will likely remain above toxic concentrations for many years.

These risks to the ecological receptors at the Tonawanda site are the risks of individual contaminants. The risks from exposure to multiple contaminants depend on the interactions among them; effects could be additive, multiplicative, or mitigating. This ERA provides a foundation for an extended characterization of the risks to exposure to multiple contaminants, but such an effort cannot be conducted without additional data or evaluation of alternative assumptions.

For ecological COCs, remedial actions undertaken to protect human populations would not necessarily also protect the limited ecological resources of the Tonawanda site. A separate remedial strategy could become necessary to deal with the ecological COCs as sources of ecological risk.

6.4.3 Uncertainties in the Ecological Risk Assessment

Uncertainties in each of the four interrelated activities of the ecological risk assessment process are addressed in the following discussion. More generally, there is uncertainty about whether characterizing the risk to organisms underestimates or overestimates the risk to populations at the Tonawanda site and the ecosystems that comprise them. The issue remains unresolved, so at present, there is no alternative to organismal-based ecological risk assessment using conservative estimates of toxicity and exposure.

6.4.3.1 Uncertainties in Problem Formulation and Selection of Ecological COCs

The structure of the biotic community (i.e., the distribution and abundance of organisms) comprising the ecological receptors at the Tonawanda site was not quantified for the ERA. The lack of quantitative data introduces uncertainties concerning whether, and to what extent, the risk characterization based on proxy organisms underestimates or overestimates the risk to the remainder of the ecological community. Onsite reconnaissance establishes the

nature and quality of habitat and confirms the presence of vegetation types and of active, visible animal species. These observations justify assumptions about the presence of unobserved organisms that are essential to normal ecosystem functioning, such as soil dwelling worms and arthropods, herbivorous insects, and aquatic benthic invertebrates. It is possible that one (or more) unobserved species of organism at Tonawanda is more sensitive than those species for which toxicity data were available for use in setting toxicity thresholds. It does not necessarily follow that these organisms are at significantly greater risk of adverse ecological effects than that estimated in this ERA, because exposure concentrations could be overestimated.

Environmental concentrations of contaminants at the Tonawanda site, which are used to calculate EQs and XQs and, thus, which are critical to the characterization of ecological risk, are based on a limited number of nonrandomly located samples (Section 2). Given that assumptions on the distribution of the data are correct, there is a quantifiable degree of uncertainty about the actual spatial distribution of contaminants, that is, whether a site chosen at random would have a contaminant concentration above or below a given value. For example, the concentration in 95 out of every 100 samples will, on average, not be greater than the 95th percentile (UL_{95}) concentration. Also, because the estimated UL_{95} concentrations were used to calculate EQs and XQs, the estimates of risk from ecological COCs were conservative. Using UL_{95} concentrations decreases the likelihood of underestimating the risk posed by each ecological COC, and it increases the likelihood of overestimating the risk. If the data do not fit well the assumed distribution, the number of ecological COCs and their exposure concentrations could be overestimated or underestimated depending on how the actual data distribution differs from the assumed data distribution.

The ecological COC screening process likely overestimated the number of organic substances that pose potential risks to ecological resources at Tonawanda. While most of the inorganic contaminants and one organic contaminant were ecological COCs because their mean environmental concentrations exceeded their toxicity thresholds, six organic contaminants were ecological COCs despite low environmental concentrations (e.g., the mean concentration of benzo[b]fluoranthene in Linde soil was 0.99 mg/kg). These organic compounds were ecological COCs because there was no factual basis for choosing a toxicity threshold. Yet in most cases where a threshold existed for an organic potential ecological COC, the contaminant concentration did not exceed the threshold. Xylene's estimated mean concentration was below its toxicity threshold, but it was included as an ecological COC because of its mobility and persistence characteristics. Thus, the rules for selecting ecological COCs likely overestimate the number of ecological COCs.

6.4.3.2 Uncertainties in Ecological Exposure Assessment

Rigorous tracing of the movement of contaminants from Tonawanda source media to ecological receptors, including quantification of a site-specific food web, was not performed for this ERA. This introduces uncertainties about the actual modes and pathways of exposure for the biotic community and the actual exposure concentrations of contaminants. Exposure

concentrations can differ from measured environmental concentrations as a result of physical and chemical processes during transport from source to receptor and as a result of biomagnification through the food web. These processes could not be evaluated explicitly and quantitatively in this ERA. It is reasonable to assume that exposure to some organisms, especially top predators, would be underestimated due to neglect of biomagnification of contaminant concentrations in their prey. The exposure experienced by others would be overestimated by neglecting processes that dilute contaminants, or otherwise make them unavailable to organisms, especially those organisms exposed by direct contact or inhalation and ingestion of contaminated media.

There is little uncertainty that the modes and pathways used to characterize the exposure to ecological receptors at the Tonawanda site are most important for the large, active organisms in terrestrial and aquatic habitats. Soil-dwelling terrestrial animals may be exposed to contaminants in soil primarily by way of inhalation following volatilization, but gaseous concentrations in soil interstices, cavities, and burrows were not available. Therefore, the exposure to burrowing organisms at the Tonawanda site from direct contact with and ingestion of contaminated soil and soil interstitial water may be underestimated if gas concentrations are larger than soil concentrations, which is unlikely. The estimate of risk will also be underestimated if toxicity thresholds are lower for inhalation than they are for ingestion. Sediment contaminant concentrations likely overestimate the exposure to sediment-burrowing aquatic organisms, which may be exposed primarily by direct contact with sediment pore water. Overestimating exposure by using conservative exposure concentrations and toxicity thresholds balances the underestimating of exposure due to neglecting exposure modes and pathways of lesser importance.

Finally, some contaminants in surface water and sediments may be toxic to Tonawanda organisms at concentrations below analytical detection limits, and thus, the exposure to aquatic biota may be underestimated.

6.4.3.3 Uncertainties in Ecological Effects Assessment

There is little doubt that, for most Tonawanda organisms, the identified ecological COCs have deleterious effects at concentrations above the threshold concentrations used to screen contaminants as ecological COCs and to characterize the risks at the Tonawanda site. Toxicity thresholds were either based on concentrations reported not to have an effect on the study organism, including federal water quality criteria (WQCAQ), or were estimated conservatively. These thresholds would underestimate the risks only to organisms at Tonawanda that are considerably more sensitive than the study organisms, and overestimate the risk to organisms equally or less sensitive than the study organisms. There remains the possibility that some thresholds were set at levels at or above which some harm would occur to the study organism or to similar organisms at the Tonawanda site.

Additional uncertainty exists as to the pertinence of organismal toxicity for characterizing the risk to populations and ecosystems. It is possible that populations may

compensate for the loss of large numbers of juveniles or adults with increased survival or fecundity, and ecosystems may possess functionally redundant species that are less sensitive to contaminants. The great uncertainty as to whether ecosystems at the Tonawanda site (e.g., Twomile and Rattlesnake Creeks) possess these buffering mechanisms justifies a conservative approach to risk assessment based on organismal toxicity.

6.4.3.4 Uncertainties in Ecological Risk Characterization

In addition to the uncertainties described above, which ultimately produce the uncertainty in the assessment of current risks for the Tonawanda site, there are three additional areas of uncertainty in the risk characterization: offsite receptors, cumulative risks, and future risks.

The ERA characterizes the risk to offsite ecological receptors from onsite contaminants without benefit of contaminant tracer studies and offsite biotic and habitat surveys. Offsite receptors can be exposed to contaminants via organismal animal and physical transport processes, but evaluating the magnitude of this exposure would require additional studies. It is unlikely that offsite receptors would have lower toxicity thresholds for contaminants than the thresholds used for onsite biotic receptors. Also, there is little reason to expect that contaminants migrating offsite would be concentrated beyond measured concentrations at the Tonawanda site unless a contaminant bioconcentrates in organisms that move extensively on and off the site. In general, the risk to offsite receptors is likely to be overestimated rather than underestimated by EQs. The XQs are a means to more realistically estimate offsite risks, but the hypothetical Tonawanda exposure factors for offsite receptors (10 percent of onsite exposure) may, nevertheless, underestimate the risks. Actual offsite risks are, thus, likely to lie between those for onsite receptors based on EQs and those for offsite receptors based on XQs.

The ERA estimates the risk to ecological receptors from individual contaminants. Generally, the methods used were sufficiently conservative that individual risks are overestimated. Nevertheless, synergistic effects are possible, perhaps likely, when toxicants interact in biological systems. Deleterious effects in ecosystems (including effects on individual organisms) may cascade throughout the system and have indirect effects on the ability of a population to persist in the area even though individual organisms are not sensitive to the given contaminants in isolation. Therefore, the ecological risk characterization for the Tonawanda site may underestimate actual risks to biotic receptors from chemical mixtures.

A third area of uncertainty in the ecological risk characterization is the future risk to the environment from contamination at the Tonawanda site. The ERA characterizes the current risk based on chronic exposure to measured concentrations of toxicants with the potential to persist in the environment for extended periods of time. Nevertheless, possible mechanisms exist that could significantly increase (e.g., erosion, leaching to surface or groundwater) or decrease (e.g., enhanced microbial degradation) the risk to future nonhuman inhabitants of the Tonawanda site.

6.4.3.5 Summary

The major uncertainties in this ERA center around the estimates of the contaminant concentrations to which ecological receptors at the Tonawanda site are actually exposed (exposure concentrations) and the concentrations that present an acceptable level of risk of adverse effects to the Tonawanda organisms, their populations, and the ecosystems that comprise them (toxicity thresholds). These uncertainties arise from many sources, especially the lack of site-specific data on contaminant transport and transformation processes, organismal toxicity, animal behavior and diet, population parameters, interspecific interactions, and the lack of a fundamental understanding of how Tonawanda ecosystems respond to environmental perturbations.

6.5 SUMMARY

6.5.1 Habitats and Wildlife

The Tonawanda site is located in a highly modified urban industrial area. Three properties (Linde, Ashland 1 and Seaway) provide minimal urban wildlife habitat, supporting only cosmopolitan species of birds and small mammals (FBDU 1981a). Habitat at Linde consists primarily of landscaped areas at a highly industrialized site amidst urban residential and commercial properties. Natural habitats in the vicinity of Linde are limited to the small wetland located offsite northeast of the plant. Ashland 1 is a highly industrialized property that has been heavily impacted by past operations. Virtually no natural habitat occurs at Ashland 1. The established vegetation is typical of highly disturbed areas. Seaway is an active solid waste disposal facility. It contains some native grasses, herbs and shrubs, and is visited by gulls and crows.

The larger and less disturbed area at Ashland 2 supports a more diverse population of animals because it contains a mosaic of upland and wetland ecosystems making it an attractive habitat for a variety of plants and animals. However, the impacts of use and abuse by humans are evident throughout the site. The primary ecological significance of Ashland 2 is that it represents a large (> 40 ha; [> 100 acre]), relatively undeveloped area. This is the only unique aspect of the site. Wildlife utilization at the site is evident from direct observation, spoor, and other signs. No threatened and endangered species identified by FWS are known to inhabit the site.

The four properties lie within the watershed of Twomile Creek, a tributary to the Niagara River. The Linde property is linked by various drainage ditches to Twomile Creek. Ashland 1, Seaway, and Ashland 2 are hydrologically connected with Twomile Creek through several small intermittent drainageways and Rattlesnake Creek, which flows into Twomile Creek 300 m (1,000 ft) above the confluence with the Niagara River (Figure 6-1).

The Ashland 2 property includes portions of Rattlesnake Creek, its floodplain, and various small vegetated wetlands. If remedial activities should be considered or implemented for this area, boundaries of any actual wetlands must be delineated in accordance with Section 404 of the Clean Water Act and New York state regulations before remedial action for that wetland can take place.

Habitats and biota occurring at the Tonawanda site are believed not to be (1) unique or unusual; (2) necessary for continued propagation of key species; or (3) highly valued for economic, recreational, or aesthetic reasons. The significance of the Tonawanda site with regard to ecological resources is uncertain, and intensive field analysis for possible impacts to biota from site contaminants may not be warranted without further qualitative ecological investigations.

6.5.2 Chemicals of Ecological Concern and Risk Characterization

Sixty-two chemicals were recognized as potential ecological COCs. Most of these chemicals were found above background levels in the soils of Linde, Ashland 1 and Ashland 2 properties. Calcium, magnesium, potassium, and sodium were dropped from the risk characterization because they are essential biological minerals. There are no readily available terrestrial wildlife toxicity data for the three radionuclides at the Tonawanda sites: radium, thorium, and uranium. The risk assessment for metals, and volatile and semivolatile organic chemicals relies on aquatic and oral toxicity data for laboratory animals gathered from compendia of published studies. A discussion of uncertainties in the ERA is provided in Section 6.4.3. When the observed concentrations of ecological COCs in the environment were compared to threshold toxicity values, thirty-three of these emerged as the contaminants of ecological concern. Their EQs ranged from 1.1 to 2,153 (mean) and 1.3 to 21,100 (RME) (Table 6-5). The ecological COCs consist of three radionuclides (radium, thorium, and uranium), 21 elements (metals), seven volatile, and two semivolatile organics.

Lead and copper generally had the highest EQs in Tonawanda remedial units; their EQs exceeded 100 in Ashland 1 soils and surface waters, Ashland 2 (South) soils and Linde soils, and were between 10 and 100 in the surface waters of Ashland 2/Seaway. Selenium in surface waters at Ashland 1 and Ashland 2 (South) had an EQ in excess of 100, as did silver in Ashland 2 surface waters and vanadium and zinc in Ashland soils. Cadmium, chromium, cobalt had EQs between 10 and 100 in surface water at Ashland 1, and antimony, arsenic, beryllium and nickel between 1 and 10; EQs for pyrene and the dichloropropene in Ashland 1 surface water could not be calculated. EQs for aluminum and zinc, the only ecological COCs in Ashland 2 (South) sediments, were, respectively, 10.7 (mean) and 15.7 (RME) and 43.5 (mean) and 81.3 (RME). Aluminum, boron and mercury in surface waters at Ashland 2/Seaway had EQs between 10 and 100; those for chromium and zinc were between 1 and 10. Molybdenum and xylenes were ecological COCs in Ashland 2/Seaway surface waters because they had the potential for mobility and persistence, although their concentrations at the site do not exceed the established toxicity threshold. The primary ecological COCs in sediments of

Ashland 2/Seaway were aluminum with an EQ of 15.7 (RME) and manganese and vanadium with EQs between 1 and 10.

Chlorobenzene, cobalt and nickel in Ashland 2 soils had EQs between 10 and 100, based on RME concentrations. Chlorobenzene and xylene were ecological COCs in Ashland 2 (South) soils because of their mobility and persistence characteristics. In addition to copper and lead, Linde soil ecological COCs were arsenic, barium and nickel with EQs between 10 and 100, and beryllium and manganese with EQs between 1 and 10. The two benzofluoranthenes, dibenzofuran, and indeno (1,2,3-cd) pyrene were also ecological COCs in Linde soils, but without toxicity information, the degree of risk these four contaminants pose cannot be estimated.

Copper, lead, selenium, and silver present the greatest risk (XQ >10) to both onsite and offsite aquatic receptors (Table 6-7). Onsite receptors are also exposed to similar level of risk from mercury, chromium, cadmium, cobalt, boron, and to an unknown degree, molybdenum, pyrene, trans-1,3-dichloropropene, and xylenes. Subterranean organisms are exposed to serious risk from six metals and seven organics because their XQs are 1.1 times as great as the calculated EQs for these contaminants. Terrestrial organisms exposed onsite via trophic pathways are exposed at the same level of risk (XQs > 10) to metals -- arsenic, barium, cobalt, copper, lead, nickel, vandium, and zinc -- and to an unknown degree of risk from organics -- benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, chlorobenzene, dibenzofuran, indeno(1,2,3-cd)pyrene, and xylenes. Offsite predators are at similar risk from all of these except cobalt, nickel, chlorobenzene and the xylenes.

The numerous ecological COCs with large EQs strongly suggest that, in the absence of remediation, both onsite and offsite organisms and populations at Tonawanda properties will continue to be at serious risk of adverse effects of the sort described in Section 6.3. This is especially true of Ashland 2, where wildlife and natural habitats are more extensive due to contaminants in soils, sediments and surface waters.

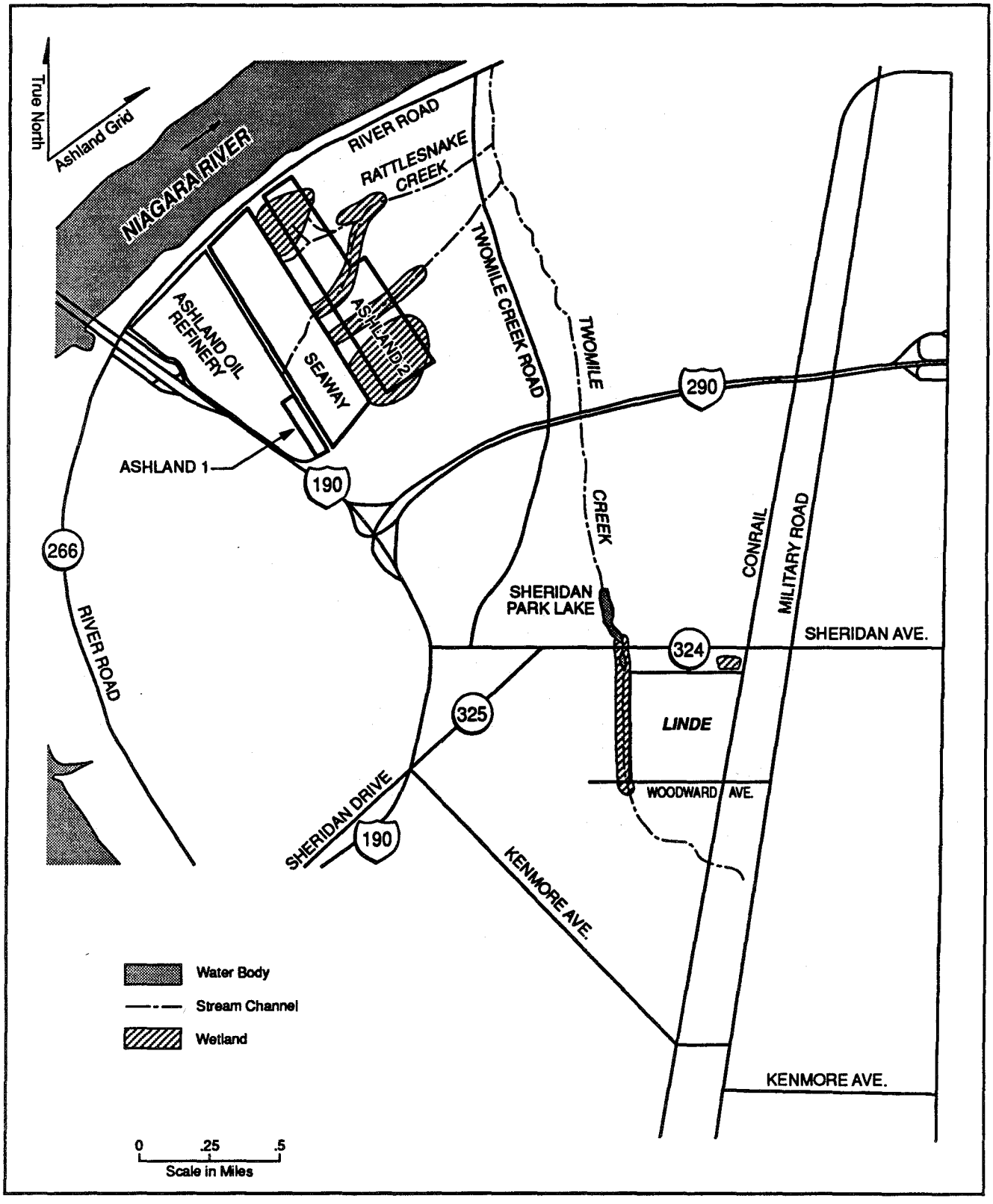

Figure 6-1. Map of Tonawanda Site Showing Remedial Units and Wetlands

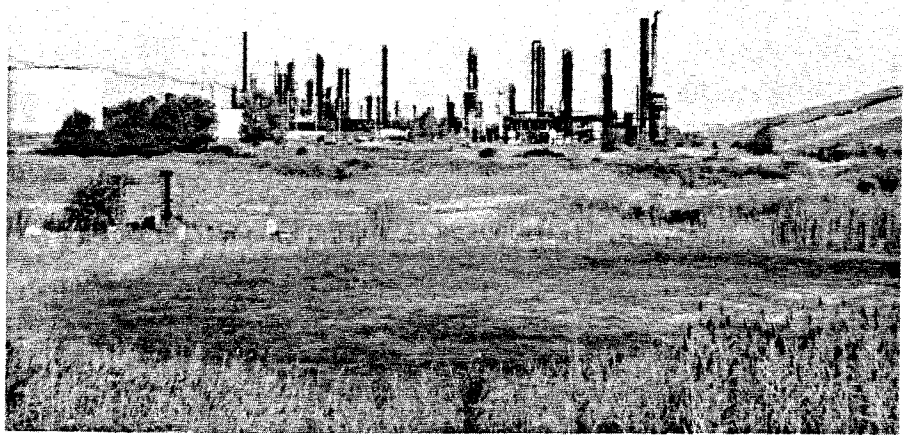

Figure 6-2. View of Ashland 1 property showing inactive oil refinery in background. Common rush, *Phragmites australis*, can be seen growing around circular denuded area in foreground from which contaminated soil was excavated. Herbaceous upland vegetation dominates the site. Seaway landfill is visible to the right.

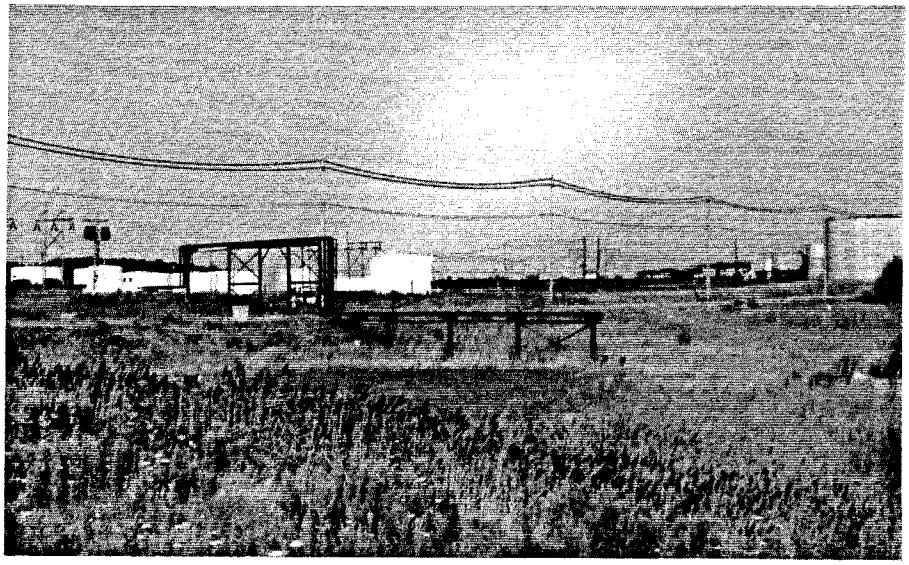

Figure 6-3. View of Ashland 1 property showing storage drums of oil refinery in background. Common rush, *Phragmites australis*, is growing on slope in foreground leading down to the bare soil where contaminated soils have been excavated.

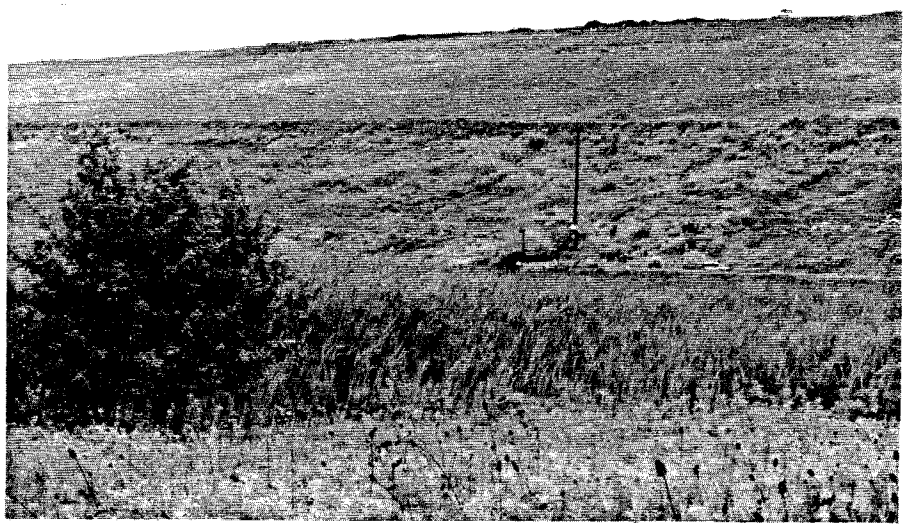

Figure 6-4. Distant view of the Seaway landfill from Ashland 2 looking across the Niagara Mohawk powerline corridor. An herbaceous upland community is in the foreground, a vegetated drainage swale (Rattlesnake Creek) in mid-frame, and Seaway in the background. Note the lack of vegetation on most of landfill's surface. Drainage pipe from Ashland 1 discharges at the base of the power pole in mid-frame at the bottom of the landfill.

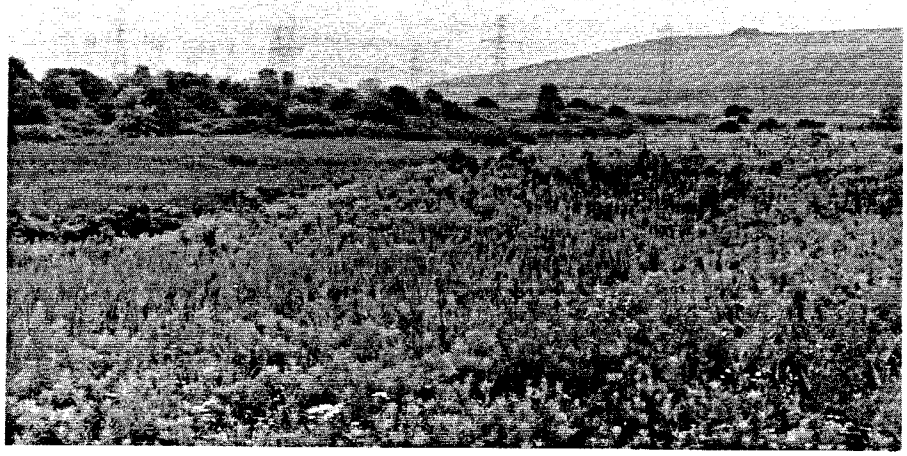

Figure 6-5. Distant view of Seaway landfill from Ashland 2 looking across herbaceous upland community in the foreground, a vegetated drainage swale (Rattlesnake Creek) in mid-frame, and shrub dominated community in the background. The Niagara Mohawk powerline corridor and the Seaway landfill are shown in the background. Note the presence of vegetation on closed portion of landfill's surface.

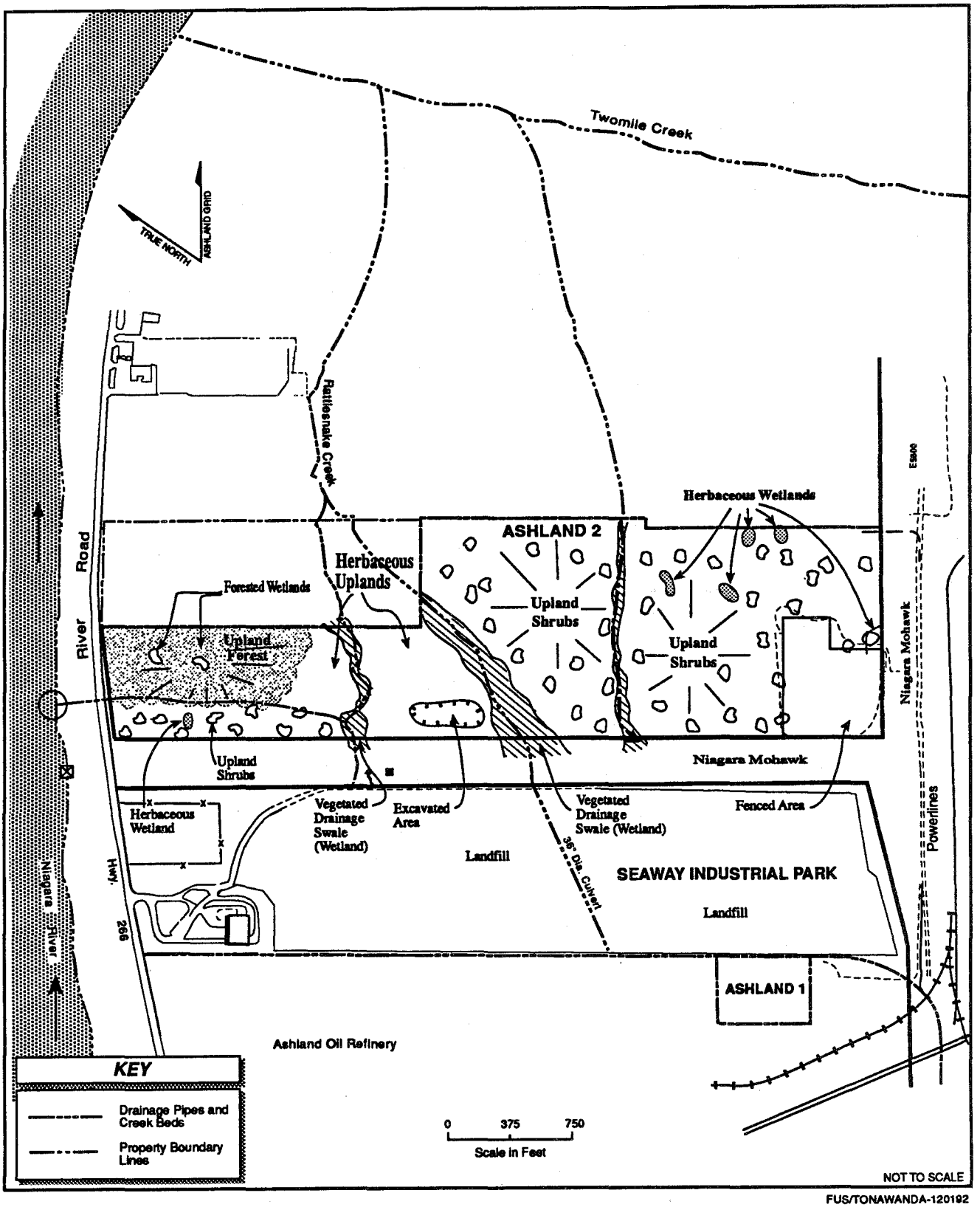

Figure 6-6. Habitat Map of Ashland 1, 2 and Seaway Properties

Figure 6-7. View of Ashland 2 showing herbaceous upland community in foreground, shrub community dominated by hawthorn (*Crataegus* spp.) and dogwood (*Cornus* spp.) in the middle, and upland forest in the background.

Figure 6-8. View of Ashland 2 showing herbaceous upland community in near foreground, a vegetated drainage swale (Rattlesnake Creek) dominated by common rush (*Phragmites australis*), a mixture of upland shrub and herbaceous communities, and upland forest in the background.

Figure 6-9. View at Ashland 2 with shrub and herbaceous upland community in the foreground, vegetated drainage swale (Rattlesnake Creek) and hawthorn and shrub dominated upland community in midframe, and forested upland in the background. Niagara Mohawk powerlines are on the horizon.

Figure 6-10. Photo of Vegetated Swale at Ashland 2

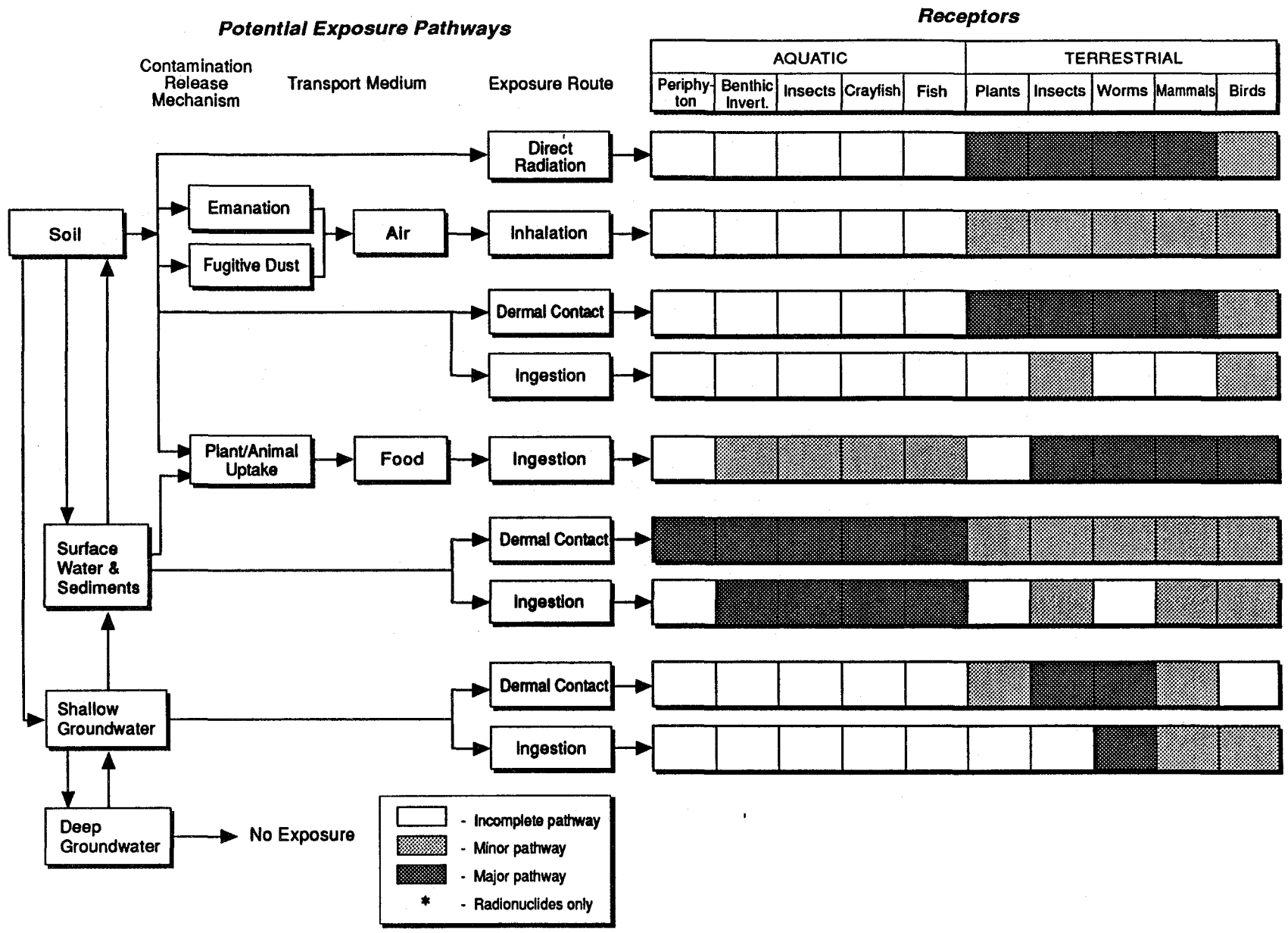

Figure 6-11. Exposure Pathways for Tonawanda Ecological Receptors

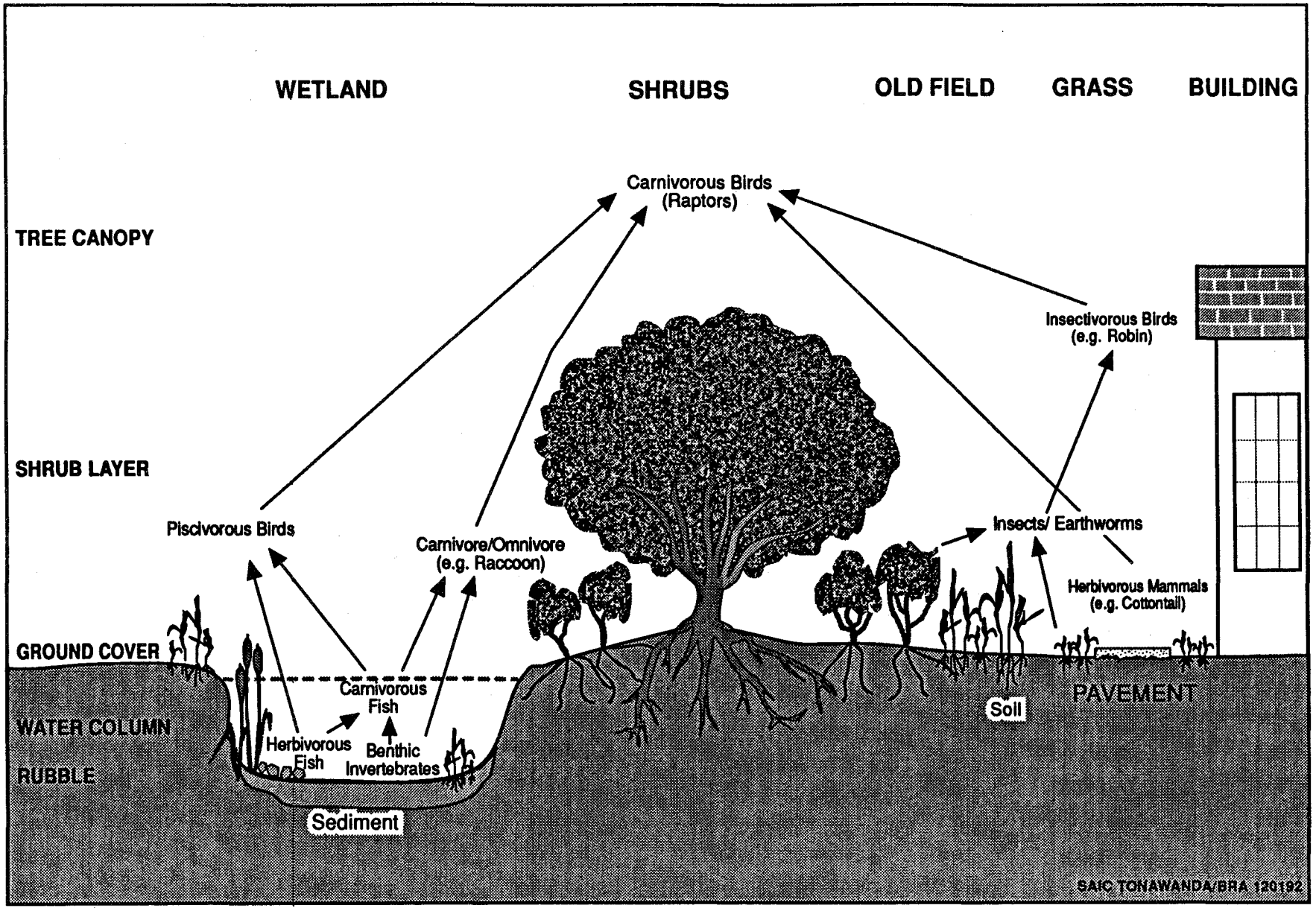

Figure 6-12. Simplified Food Web for Tonawanda Habitats

Table 6-1. Tonawanda Ecological Risk Assessment Contaminant Screening Data

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
RADIONUCLIDES								
Radium	NA	NA	NA	NA		high (mobile if soil coarse or acid)[e]	16,024[e]	NA
Thorium	NA	NA	Acute toxicity by all routes is very low.[a] 1,220 (intraperitoneal LD50, nitrate)[a]	1,000 daily (dog, 46 days, growth depression)	insoluble[e]	high[e]	1.41 x 10^{10}y[e]	2 orders of magnitude over background levels in vegetation[e]
Uranium	NA	NA	Oral toxicity of U compounds is rather low.[a]	.2 /day (dogs, 1 yr, adverse growth effects)[a] 10,000-40,000 (rat, LD50, 30 days, soluble form)[a]	insoluble[e]	high[e]	4.51 x 10^{9}y U-238[e]	1 order of magnitude over background levels in vegetation[e]
METALS								
Aluminum	NA	NA	770 (mice, LD50, $AlCl_3$)[a] 190 (guinea pigs, LDLo, $AlCl_3$)[a] 980 (mice, LD50, $Al(SO_4)$)[a]	1,400 (chickens, caused rickets)[c]		Concentration in soil in range of 150-600 g/kg.[c]	NA	NA

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Antimony	8.8E+1 ug/L (IRIS WQCAQ)	3.0E+1 ug/L (IRIS WQCAQ)	700 (rats, oral LD50)[d],	5 mg/2 days for 30-90 days, abortion[a] 0.35 mg/kg bw/day (rat, oral, LOAEL) (IRIS)	insoluble as metal oxide slightly soluble[a]	NA	NA	NA
Arsenic	190 (4d) (As III)[e] 10,000 μg/L (Black Sea mussel, LC50); quite sensitive to sublethal concentration as reflected by physiological changes.[c] 3.6E+2 ug/L (As III) (IRIS WQCAQ)	100 (fish)[e] 1.9E+2 ug/L (As III) (IRIS WQCAQ)	8-500 As_2O_3[a] 763 (rat, oral LD50)[c] 145 (mouse, oral LD50)[c] 0.58 developmental abnormalities[c]	0.6 (rat, oral TDLo, 35 wk prior to copulation, fertility effects)[c] 280 (mouse, oral, 8 weeks, mutations)[d]	insoluble as elemental As[a] As_2O_3 1.2 g/kg[a]	NA	NA	44 (fish)[e]
Barium	50,000[e]	2,000 (MCL)	5 (rabbit, $BaCl_2$)[a]	2 (rabbit)[a]	2.46 mg/L $BaSO_4$ at 25°[a]	NA	Persistent in SW[e]	4[e]

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Beryllium	1.3E+2 ug/L lowest effect concentration (IRIS WQCAQ)	5.3E+0 ug/L lowest effect concentration (IRIS WQCAQ)	30 μMol/L (mouse, DNA damage)[d]	5,000 no effect level[a] 5 mg/L (rat, oral, NOAEL) (0.54 mg/kg bw/day) (IRIS)	metal and oxides insoluble to slightly soluble[a]	NA	NA	NA
Boron	NA	NA	250 (cat, oral LD50)[c]	1,170 (rats, H_3BO_3)[a] 350 (8.8 mg/kg/day) (dog, oral, 2 yr, NOAEL) (IRIS) 1,170 (29 mg/kg/day) (dog, oral, 2 yr, LOAEL) (IRIS)	metal insoluble[a] oxides slightly soluble, 0.72 mg/L[a] 260,000 (NaBO2)	NA	NA	1,000[a]
Cadmium	3.9E+0 ug/L (1-hr average) (IRIS WQCAQ)	1.1E+0 ug/L (4-day average) (IRIS WQCAQ)	150-300 (rabbit, lethal oral dose, $CdCl_2$) 225 (rat, oral LD50)[d] 70 (rabbit, oral LDLo)[d]	21.5 (rat, oral TDLo, multi-generation fertility effects, pre-implantation mortality)[d], 448 mouse[d]	insoluble as metal[a]	NA	NA	NA

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Chromium	98 (4d) CrIII 11 (4d) CrVI (hardness of 40 mg/L)[e] 9.8E+2 ug/L CrIII (hardness dependent) (IRIS) 1.6E+1 ug/L CrVI (1-hour average) (IRIS)	100 (fish) CrVI[e] 1.2E+2 ug/L (hardness dependent) (IRIS) 1.1E+1 ug/L CrVI (4-day average) (IRIS)	1,500 (rat, oral LD50, soluble chromates)[a]	1,000 mg/day (cat, oral, no adverse effects, chromium phosphate)[a] 1,468 mg/kg/day (rat, oral, NOEL, 5% chromic oxide) (IRIS) 2.4 mg/kg/day CrVI (rat, oral, 1 yr, NOAEL) (IRIS)	insoluble[e] 1,670,000 (CrO3)	NA	NA	16 (fish); higher uptake in ultra-basic soils; to 1E+3 in shrubs; to 1E+6 in snails[e]
Cobalt	10-400 μg/L inhibit seed germination[c] 100-400 μg/L reduce plant growth[c]		1,500 (rat, metal)[a] 135 (rat, oral, CoO)[a] 6170 (rat, oral LD50)[d] 750 (rabbit, oral LDLo)[d]	30 mg/animal/d (metal)[a] 1 mg/day in water (rat, $CoCl_2$)[a]	insoluble[a] 529,000 (CoCl2)	NA	59 days[a]	low[a]

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Copper	5.4 (4d, hardness of 40 mg/L)[e] 250 (larval fathead minnow, LD50)[c] 9.2E+0 ug/L (hardness dependent) (IRIS WQCAQ)	3 (fish)[e] fathead minnow larvae 123 (28 day)[c] 6.5E+0 ug/L (hardness dependent) (IRIS WQCAQ)	140 (rat, $CuCl_2$)[a] 159 (rabbit, $CuCO_3$, $Cu(H_2O)$[a]	1.21 (rat, oral TDLo 22 wk)[d] 70 (EH-L)[h]	insoluble but pH sensitive[e] chloride and nitrate soluble, carbonate, phosphate insoluble (Lange's Handbook of Chemistry)	high in organic matrix[e]	NA	200 (fish)[e]
Iron	NA	NA	98.6 Fe powder[a] 400 (mouse, $FeCl_2$)[d] 20,000 (guinea pig, oral LD50)[d]	800 mg/d (dog, no effect level, $FeCl_2$)[a]	insoluble[a] ferric and ferrous salts soluble (Lange's)	NA	NA	NA
Lead	1.0 (4d) (hardness of 40 mg/L)[e] 8.2E+1 ug/L (1-hour average) (IRIS WQCAQ)	5,000 (fish)[e] 50 (aquatic vertebrates)[e] 3.2E+0 ug/L (4-day average) (IRIS WQCAQ)	160 (pigeon, LDLo)[d] 50-600 (acute single dose, lead salts)[c]	790 (rat, oral, multi- generation)[c] 1 (rats, no effect level)[c] 0.5 (pregnant sheep, fetal effects)[c]	0.03 (hard) 0.5 (soft)[e] insoluble[a] 2,000-10,000 mg/L soluble forms in soil[a]	high in organic or clay matrix[e]	NA	49 (fish) (log 1.4-1.7 fish; log 2.7-3.2 invertebrates)[c] 0.02 pasture herbage[c]
[Note: Biomethylation of Pb is caused by microorganisms]								

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Magnesium	NA	NA	25,000 sulfate[c] 230-280 (dogs, metal)[c]	NA	insoluble 6.2-106 mg/L[a] slightly soluble in hot water (elemental)[a] salts soluble (Lange's)	NA	NA	NA
Manganese	NA	NA	9,000 (rat, oral LD50)[d]	600 (rat, oral LD50)[c]	insoluble[a] salts soluble (Lange's)	NA	14 days	NA
Mercury	350 μg/L (catfish, LC50, 96 hr)[c] 0.5 mg/kg (mollusks, LC50, 48 hr); 0.19 mg/kg (mollusks, LC50, 96 hr)[c] 0.051 mg/kg (tadpoles, LC50, 96 hr)[c] 2.4E+0 ug/L (1-hr average) (IRIS WQCAQ)	10,000 μg/L/21 days (largemouth bass)[c] 1.2E-2 ug/L (4-day average) (IRIS WQCAQ)	110 (mouse, HgI)[a] 8 (rat, HgO)[a]	1 (no effect, methylmercury) 0.5 (rat, oral, 2 yr, renal lesions in females, phenylmercuric acetate)[a]	0.28 μMol/L H_2O at 25°[c] some salts soluble	high[a]	2-3 years[c]	1,000-1,500 (pike)[c] freshwater plants 1,000, fish 1000, invertebrates 100,000[c] 63,000 freshwater fish[c]

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Molybdenum	70,000 (fathead minnow, LC50, 96 hr)[g],	790 (trout eggs, LC50, 28d)[g]	NA	80 (rat, no effect, 12 wks)[g], 500 (rabbit, no effect, 12 wk)[g]	insoluble, 1,066 (MoO3)[a]	NA	NA	3,300 algae, 3,570 lake periphyton, 9.8 crayfish carapace[g]
Nickel	1.4E+3 ug/L (total nickel) (IRIS WQCAQ)	1.6E+2 ug/L (total nickel) (IRIS WQCAQ)	5 (guinea pig, rat, LDLo)[a]	1,000 no effect[a] 25 (rat, $NiSO_4$) 5 no effect, acetate[a] 2.5-100 (Ni, fungal inhibiter)[c]	insoluble NiO/Ni[a]	NA	NA	NA
Potassium	NA	NA	metal corrosive at low concentration[a]	NA	NA 1,111 mg/L (KOH)[a]	NA	NA	NA
Selenium	20 (IRIS WQCAQ)	5 (IRIS WQCAQ)	6,700 (rat, oral LD50)[d]	134 (mouse, oral TDLo, multi-generation)[d]	insoluble[a]	NA	NA	NA
Silver	1,100 (trout milt, enzyme effect, 0.5 h), 0.92 (RIS WQCAQ)	410 (asiatic clam) 0.12 (IRIS WQCAQ)	2,820 (rat, oral LD50, Ag_2O)[a] 50 (mouse, oral LD50, $AgNO_3$[a]	NA	insoluble	high	persistent	83 (duckweed), no magnification in algae, Daphnia, fr. mussels, fathead minnows
Sodium	NA	NA	NA	NA	insoluble as metal[a]	--	--	--

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Thallium	NA	NA	NA	NA	NA	NA	NA	NA
Vanadium	NA	NA	11 (albino mice, oxides)[a]	10 minimum toxic diet concn[c]	insoluble as metal[a]	NA	NA	NA
Zinc	49 (4d) (hardness of 40 mg/L)[e], 120 (IRIS WQCAQ)	2,000 (fish)[e], 110 (IRIS WQCAQ)	250 (guinea pig, LDLo, ZnF_2)[a] 1,190 (rat, oral LD50, zinc nitrate hexahydrate)[a]	10 (rat, oral, no effect level)[a] 50 (rat, oral, reproductive effects)[a]	insoluble as metal[a]	NA	Persistent in SW[e]	47 (fish),[e] 16,700 (oyster), 85 (clam), 500 (mussel)[c]
ORGANICS								
Acenaphthene	NA	NA	NA	175 daily (mouse, oral, NOAEL, 90 days) (IRIS) 350 daily (mouse, oral, LOAEL, 90 days, liver effects) (IRIS)	insoluble[f]	NA	NA	NA
Acetone	20,000 (Mexican axolotl, 48 hr)[c] 10,000 (Daphnia magna, TLm)[c]	NA	5,340 (rabbit, oral LD50)[d] 5,800 (rat, oral LD50)[d] 3,000 (mouse, oral LD50)[d]	NA	Completely miscible[b]	0.58 higher in clays[b]	1 day[b] 2 hr in H_2O[c]	negligible[b] 0.69 haddock[c]

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Anthracene	5,000 (trout, no effect, 24 hr)[f]	NA	NA	1,000 daily (mouse, oral, NOEL, 90 days) (IRIS)	0.075[f]	NA	NA	3,500 (mayfly, 28 hr) 760 (Daphnia)[a]
Benzo(a)anthracene	1,000 (bluegill, LC87, 6 mo)	NA	NA	180 (rat, oral, oncogenic effects)[d]	NA	1.38E+6	NA	10,109 (cladoceran, 24 hr)
Benzene	20,000 (bluegill, LC50, 1-2d), 5,000 (minnow, min. lethal, 6 hr)[f] 5,300 (IRIS WQCAQ)	NA	150-300 (rabbit, oral, LD50), 4,700 (mouse, oral, LD50), 3,400 (rat, oral, LD50)[a]	1 (rat, oral, no effect; rabbit, oral, 5 y, eye effects)[a], 930 (rat, oral, LD50)	1791[b]	<100 (est.)[b]	13-23 (winter)[b]	3.5 eel, 4.3 goldfish, 24 (est.)[b]
Benzo(b)fluoranthene	NA	NA	NA	NA	0.0012[c]	high	NA	7.6E05 (invertebrates)
Benzo(k)fluoranthene	NA	NA	NA	NA	7.6E-07[c]	high (3.2E+6)[c]	>700[c]	9.3E04 (fish, calc.)[c]
Benzo(a)pyrene	>1,000 (sandworm, LC50, 96 hr)	NA	50 mg/kg bw (rodents, oral LD50)	100.0 mg (rat, oral, no effect) 1,000 (rat, oral birth defects)[a]	NA	NA	NA	36,656 (northern pike, bile and gallbladder, 19.2 hr) 82,231 (snail, 3 days)
Benzo(g,h,i)perylene	NA	NA	NA	NA	insoluble[c]	1E06[c]	persistent[c]	(log 4-5)[c]
Bis(2-ethylhexyl) phthalate	4.0E+2 ug/L (IRIS WQCAQ)	3.6E+2 ug/L (IRIS WQCAQ)	NA	19 mg/kg bw/day (guinea pig, rat, oral, liver effects) (IRIS)	NA	NA	NA	NA

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Anthracene	5,000 (trout, no effect, 24 hr)[f]	NA	NA	1,000 daily (mouse, oral, NOEL, 90 days) (IRIS)	0.075[f]	NA	NA	3,500 (mayfly, 28 hr) 760 (Daphnia)[a]
Benzo(a)anthracene	1,000 (bluegill, LC87, 6 mo)	NA	NA	180 (rat, oral, oncogenic effects)[d]	NA	1.38E+6	NA	10,109 (cladoceran, 24 hr)
Benzene	20,000 (bluegill, LC50, 1-2d), 5,000 (minnow, min. lethal, 6 hr)[f] 5,300 (IRIS WQCAQ)	NA	150-300 (rabbit, oral, LD50), 4,700 (mouse, oral, LD50), 3,400 (rat, oral, LD50)[a]	1 (rat, oral, no effect; rabbit, oral, 5 y, eye effects)[a], 930 (rat, oral, LD50)	1791[b]	31, 98 (est.)[b]	13-23 (winter)[b]	3.5 eel, 4.3 goldfish, 24 (est.)[b]
Benzo(b)fluoranthene	NA	NA	NA	NA	0.0012[c]	high	NA	7.6E05 (invertebrates)
Benzo(k)fluoranthene	NA	NA	NA	NA	7.6E-07[c]	high (3.2E+6)[c]	>700[c]	9.3E04 (fish, calc.)[c]
Benzo(a)pyrene	>1,000 (sandworm, LC50, 96 hr)	NA	50 mg/kg bw (rodents, oral LD50)	100.0 mg (rat, oral, no effect) 1,000 (rat, oral birth defects)[a]	NA	NA	NA	36,656 (northern pike, bile and gallbladder, 19.2 hr) 82,231 (snail, 3 days)
Benzo(g,h,i)perylene	NA	NA	NA	NA	insoluble[c]	1E06[c]	persistent[c]	(log 4-5)[c]
Bis(2-ethylhexyl) phthalate	4.0E+2 ug/L (IRIS WQCAQ)	3.6E+2 ug/L (IRIS WQCAQ)	NA	19 mg/kg bw/day (guinea pig, rat, oral, liver effects) (IRIS)	NA	NA	NA	NA

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Bromodichloro-methane	11,400 (IRIS WQCAQ)	NA	916 (rat, oral, LD50), 400 (mouse, oral, LD50)[d]	17.9 (mouse gavage, LOAEL, IRIS)	NA	NA	NA	NA
Bromoform	11,400 (IRIS WQCAQ)	NA	1,147 (rat, oral, LD50), 1400 (mouse, oral, LD50)[d]	50 (rat, gavage, LOAEL,IRIS)	3,190[f]	NA	NA	NA
2-Butanone (methylethylketone)	5E06 (goldfish, LC50, 24 hr)[f]	NA	3,300 (rat, oral, single dose)[f]	NA	353,000[f]	NA	NA	NA
Chlorobenzene (phenyl chloride)	24,000 (bluegill, Tlm, 1-2 d)[f], 250 (IRIS WQCAQ LEC), 16900 (minnow, LC50, 96 hr)[c]	90 (fish larvae, LD100)[c]	2,190 (rat, oral, LD50, single dose)[f], 2,250 (rabbit, oral, LD50)[c], 2,300 (mouse, oral, LD50)[c]	14.4 (rat, rabbit, oral, no effect, 192 d), 144 (rabbit, oral, growth effects, 192 d)[f]	472[b]	83-389[b]	75[b]	447 fathead minnow[b], little or none (most fish species)[c]
Chrysene	>1,000 (sandworm, LC50, 96 hr)	NA	NA	450 (mouse, oral, cytological effects)[d]	NA	NA	NA	248-361 (pink shrimp, 28 days)
Dibenzo(a,h)-anthracene	>1,000 (sandworm, LC50, 96 hr)	NA	NA	200 (rat, oral, oncogenic effects)[d]	NA	NA	NA	NA
Dibenzofuran	NA	NA	NA	NA	0.01[c]	4,600-6,350[c]	persistent[c]	1,100 (minnow)[c]
Dibromochloro-methane	11,400 (IRIS, WQCAQ)	NA	848 (oral, rat, LD50), 800 (mouse, oral lLD50)[d]	60 (rat, gavage, NOAEL, subchronic, IRIS)	NA	NA	NA	NA

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
1,2-Dichloroethane (ethylene dichloride)	18,000 (IRIS, WQCAQ LEC)	2,000 (IRIS, WQCAQ)	680 (rat, oral, single dose)[f]	95 (rat, gavage, LD100), 38 (rat, oral, TD)[d]	8,690[f]	NA	NA	NA
1,2-dichloropropene	2,120 (IRIS WQCAQ)	NA	2,000 (guinea pig, oral, single dose)[f]	50 (mouse, gavage, NOAEL, IRIS)	2,700[f]	NA	NA	NA
2,4-dimethylphenol (2,4-xylenol)	24,000 (Daphnia, LD0)f, 13,000 (tench, TLm, 24 hr)f, 28,000 (trout embryos, TLm, 24 hr)f	NA	NA	3,200 (rat, oral, LD50), 809 (mouse, oral, LD50)[d]	NA	NA	NA	NA
Di-n-butylphthalate	9.4E+2 ug/L LEC (IRIS WQCAQ)	3.0E+0 ug/L LEC (IRIS WQCAQ)	NA	125 daily (rat, oral, 1 yr, NOAEL) (IRIS) 600 mg/kg bw/day (rat, oral, 1 yr, LOAEL) (IRIS)	13 at 25°C (IRIS)	160-6,400[b]	short lived[b]	12 (minnow), 21 (calc.)[b]
Fluoranthene	500 (sandworm, LC50, 96 hr) 3.98E+3 ug/L (IRIS WQCAQ)	NA	2,000 mg/kg bw (rodents, oral LD50)	125 daily (mouse, oral, 13 wk, NOAEL) (IRIS) 250 daily (mouse, oral, 13 wk, LOAEL) (IRIS)	NA	NA	NA	379 (rainbow trout, liver, 21 days)

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Fluorene	500 (bluegill, LC12, 30 days) 820 (rainbow trout, LC50, 96 hr) 910 (bluegill, LC50, 96 hr)	NA	NA	125 daily (mouse, oral, NOAEL, 13 wk) (IRIS) 250 daily (mouse, oral, LOAEL, 13 wk, blood effects) (IRIS)	NA	NA	NA	200-1,800 (bluegill, 30 days)
Indeno(1,2,3-cd)pyrene	NA	NA	NA	NA	0.062[c]	20,000[c]	persistent[c]	59,000[c]
Methylene Chloride	125,000 (algae)[f] 11,000 (freshwater organisms, no effect level)[c] 193,000 (fathead minnows, LC50, 96 hr)[c] 99,000 (fathead minnows, EC50, 96 hr)[c]	NA	2,000 (rabbits, LD50)[a] 1,275 (rat, oral, DNA damage)[d] 1,600 (rat, oral LD50)[d]	190 (rat, LTEL)	13,000[b] 2 g/100 mL (20,000 mg/L at 20°)[a]	48[b]	0.25[b]	5[b]
2-methylnaphthalene	2,000 (minnow, LC50)[g]	NA	1,630 (rat, oral, LD50)[d]	NA	NA	NA	NA	NA

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
4-methylphenol (p-cresol)	12,000 (Daphnia, LD0)[f], 4,000 (trout embryos, LC50, 24 hr)[f]	NA	1,800 (rat, oral, single dose)[f], 1,100 (rabbit, oral, single dose)[f]	207 (rat, oral, LD50), 344 (mouse, oral, LD50)[d]	NA	NA	NA	NA
Naphthalene	33,000 (algae, growth effects, 1 d) 150,000 (fish, Tlm, 96 hr), 1,000 (96 hr)[c], 8 (crab, TD100)[c], 2,300 (IRIS WQCAQ)	620 (IRIS WQCAQ)	NA	1,000 (rat, oral, eye degeneration)[c], 490 (rat, oral, LD50), 533 (mouse, oral, LD50), 1,200 (guinea pig, oral, LD50)[d]	30[f]	871-2,400[c]	<8[c]	131 cladoceran, 310 bluegill[g], 40-1,000[c]
Phenanthrene	600 (sandworm, LC50, 96 hr) 3.0E+1 ug/L (IRIS WQCAQ)	6.3E+0 ug/L (IRIS WQCAQ)	700 (rodents, oral LD50)[a]		insoluble[a]	NA	NA	325 (cladoceran, 24 hr)
Phenol	16,000 (Daphnia, LD0)[f], 5,000 (trout, lethal concentration, 3 hr)[f], 46,000 (goldfish, LD50, 24 hr)[f]	2,560 (IRIS WQCAQ LEC)	530 (rat, oral, single dose), 400-600 (rabbit), 100 (cat), 500 (dog)[f], 317 (rat, oral, LD50), 270 (mouse, oral, LD50)[d]	60 (IRIS NOAEL)	82,000	NA	< 8[f]	NA

Table 6-1. (continued)

Potential Ecological Contaminants of Concern	Biological Effects				Environmental Fate			
	Aquatic		Terrestrial		Mobility		Persistence	
	Acute Toxicity Level (μg/L)	Chronic Toxicity Level (μg/L)	Acute Oral Toxicity Level (mg/kg)	Chronic Oral Toxicity Level (mg/kg)	Water Solubility (mg/L)	Soil Sorption (K_{oc})	Degradation or Decay Half-Life (d)	Bioconcentration Factor (BCF)
Pyrene	NA	NA	NA	75 daily (mouse, oral, NOAEL, 13 wk) (IRIS) 125 daily (mouse, oral, LOAEL, 13 wk, kidney effects) (IRIS)	insoluble[a]	NA	NA	2,702 (cladoceran, 24 hr)
Toluene	13,000 (bluegill, LC50, 96h)[e] 7,300 (striped bass, LC50, 96 hr)[e] 17,500 (IRIS WQCAQ)	NA	636 (rat, oral LD50)[d] 3.0 mL/kg (14-day-old rat, LD50)	590 (rat, no effect)[a]	534.8 at 25°[b] insoluble[a]	37-160 (various soils)[b]	8 days[b]	11 (fish)[e]
Trans-1,3-dichloropropene	NA	NA	NA	NA	NA	NA	NA	NA
Xylene	13,000 (goldfish, LD50, 24 hr)[f]	NA	4,300 (rat, oral, LD50)[a]	250 (IRIS NOAEL)	146[b]	25.4-204[b]	persistent (anaerobic)[b]	21 eels, 62 clams, 132 fish (pred.)[b]

-- = Search not complete
NA = Search made; data not available
[a]Clayton and Clayton, 1981
[b]Howard, 1990
[c]HSDB, 1992
[d]RTECS, 1992
[e]SAIC, 1992
[f]Verschueren, 1977

Table 6-2. Tonawanda Ecological Risk Assessment Contaminant Screening

Potential Ecological COC	Environmental Media	Toxicity Threshold Concentration		Decision Criteria			Ecological COC	Remedial Unit
		aquatic µg/L	oral mg/kg	Environmental Concentration	Mobility	Persistence		
				aquatic or oral	solubility or adsorption	half-life or BCF		
RADIONUCLIDES								
Radium	surface water	NA		NA	Y	Y	Y	A1, A2(S), A2(N)/S, L
	soils and sediments		NA	NA	Y	Y	Y	A1, A2(S), A2(N)/S, L
Thorium	surface water	NA		NA	N	Y	Y	A1, A2(S), A2(N)/S, L
	soils and sediments		NA	NA	N	Y	Y	A1, A2(S), A2(N)/S, L
Uranium	surface water	NA		NA	N	Y	Y	A1, A2(S), A2(N)/S, L
	soils and sediments		NA	NA	N	Y	Y	A1, A2(S), A2(N)/S, L
METALS								
Aluminum	surface water	NA		NA	N	NA	Y	A1, A2(S), A2(N)/S
	soils and sediments		1000	Y	Y	Y	Y	A2(S), A2(N)/S
Antimony	surface water	30		Y	N	NA	Y	A1
Arsenic	surface water	190		Y	Y	N	Y	A1
	soils and sediments		10	Y	Y	N	Y	L
Barium	surface water	1000		N	Y	N	N	
	soils and sediments		1.0	Y	NA	N	Y	L
Beryllium	surface water	5		Y	N	NA	Y	A1
	soils and sediments		0.5	Y	N	NA	Y	L, A2(S)
Boron	surface water	1000		Y	N	Y	Y	A2(N)/S

Table 6-2. (continued)

Potential Ecological COC	Environmental Media	Toxicity Threshold Concentration		Decision Criteria			Ecological COC	Remedial Unit
		aquatic µg/L	oral mg/kg	Environmental Concentration	Mobility	Persistence		
				aquatic or oral	solubility or adsorption	half-life or BCF		
Cadmium	surface water	1		Y	N	NA	Y	A1
	soils and sediments		10	N	NA	NA	N	
Chromium	surface water	11 (VI), 120 (III)		Y	N	Y	Y	A1, A2(S), A2(N)/S
Cobalt	surface water	10		Y	N	Y	Y	A1
	soils and sediments			Y	N	Y	Y	A2
Copper	surface water	6.5		Y	N	Y	Y	A1, A2(S), A2(N)/S
	soils and sediments		1.0	Y	N	Y	Y	A1, A2(S), L
Iron	surface water	NA		NA	N	NA	Y	A1, A2(S), A2(N)/S
Lead	surface water	3.2		Y	N	Y	Y	A1, A2(S), A2(N)/S
	soils and sediments		1.0	Y	Y	Y	Y	A1, A2(S), L
Manganese	surface water	NA		NA	N	Y	Y	A1, A2(N)/S
	soils and sediments		500	Y	NA	Y	Y	A2(N)/S, L
Mercury	surface water	0.012		Y	N	Y	Y	A2(N)/S
Molybdenum	surface water	500		N	Y	Y	Y	A2(N)/S
Nickel	surface water	160		Y	N	NA	Y	A1
	soils and sediments		5.0	Y	N	NA	Y	A2,L
Selenium	surface water	5		Y	N	NA	Y	A1, A2(S)
Silver	surface water	0.12		Y	N	Y	Y	A2(S)

Table 6-2. (continued)

Potential Ecological COC	Environmental Media	Toxicity Threshold Concentration		Decision Criteria			Ecological COC	Remedial Unit
		aquatic µg/L	oral mg/kg	Environmental Concentration	Mobility	Persistence		
				aquatic or oral	solubility or adsorption	half-life or BCF		
Vanadium	surface water	NA		NA	N	NA	Y	A1, A2(S), A2(N)/S
	soils and sediments		10	Y	NA	NA	Y	A2(S), A2(N)/S, L
Zinc	surface water	110		Y	N	Y	Y	A2(N)/S
	soils and sediments		10	Y	NA	Y	Y	A2(S)
ORGANICS		µg/l	mg/kg					
Acenaphthene	soils and sediments		175	N	N	NA	N	
Acetone	soils and sediments		50	N	Y	N	N	
Benzo(b)fluoranthene	surface water	NA		NA	N	Y	Y	A1
	soils and sediments		NA	NA	N	Y	Y	A1, L
Benzo(k)fluoranthene	soils and sediments		NA	NA	N	Y	Y	A1, L
Benzo(g,h,i)perylene	soils and sediments		NA	NA	N	Y	Y	A1, L
Bromodichloromethane	surface water	11,000	15	N	NA	NA	N	
Bromoform	surface water	1,100	25	N	Y	NA	N	
2-butanone	surface water	100,000	50	N	Y	NA	N	
Chlorobenzene	soils and sediments	10	14	N	Y	Y	Y	A2(S), L
Chrysene	soils and sediments	10	400	N	NA	Y	N	
Dibenzo(a,h)anthracene	soils and sediments	NA	200	N	NA	NA	N	
Dibenzofuran	soils and sediments	NA	NA	NA	N	Y	Y	L
Dibromochloromethane	surface water	11,000	60	N	NA	NA	N	

Table 6-2. (continued)

Potential Ecological COC	Environmental Media	Toxicity Threshold Concentration		Decision Criteria			Ecological COC	Remedial Unit
		aquatic µg/L	oral mg/kg	Environmental Concentration	Mobility	Persistence		
				aquatic or oral	solubility or adsorption	half-life or BCF		
1,2-dichloroethane	surface water	2,000	10	N	Y	NA	N	
1,2-dichloropropene	surface water	2,120	50	N	Y	NA	N	
2,4-dimethylphenol	surface water	250		N	NA	NA	N	
Di-n-butylphthalate	soils and sediments		125	N	Y	N	N	
	surface water	3		N	Y	N	N	
Fluoranthene	soils and sediments		125	N	NA	Y	N	
	surface water	75		N			N	
Fluorene	soils and sediments	10	125	N	NA	Y	N	
Indeno(1,2,3-cd)pyrene	soils and sediments	NA	NA	NA	N	Y	Y	L
Methylene Chloride	soils and sediments		100	N	Y	N	N	
	surface water	2,000		N			N	
2-methylnaphthalene	soils and sediments	40	25	N	NA	NA	N	
4-methylphenol	surface water	75	20	N	NA	NA	N	
Naphthalene	soils and sediments	620	100	N	Y	N	N	
Phenanthrene	soils and sediments		10	N	N	Y	N	
	surface water	30		N			N	
Phenol	surface water	2,000	60	N	Y	N	N	
Pyrene	soils and sediments		75	N	N	Y	N	
	surface water	NA		NA			Y	A1

Table 6-2. (continued)

Potential Ecological COC	Environmental Media	Toxicity Threshold Concentration		Decision Criteria			Ecological COC	Remedial Unit
		aquatic µg/L	oral mg/kg	Environmental Concentration	Mobility	Persistence		
				aquatic or oral	solubility or adsorption	half-life or BCF		
Toluene	soils and sediments		100	N	Y	N	N	
	surface water	17,500		N	Y	N	N	
Trans-1,2-dichloroethene	surface water	11,600	17	N	Y	NA	N	
Trans-1,3-dichloropropene	surface water	NA	NA	NA	NA	NA	Y	A1
Xylenes (total)	soils and sediments		250		Y	Y	Y	A2(S)
	surface water	250		N			Y	A2(N)/S

Table 6-3. Tonawanda Contaminant Concentrations

Ecological COC / Remedial Unit	Surface Water (μg/L)		Sediments (mg/kg)		Surface Soils (mg/kg)	
	Mean	Upper 95	Mean	Upper 95	Mean	Upper 95
METALS						
Aluminum						
Ashland 1	38,157	151,000				
Ashland 2 (South)	3,523	9,870	10,670	15,673		
Ashland 2 (North)/Seaway	3,374	28,300	14,600	25,314		
Antimony						
Ashland 1	49.4	103				
Arsenic						
Ashland 1	252	259				
Linde					99.1	127
Barium						
Linde					256	313
Beryllium						
Ashland 1	7.00	19.6				
Ashland 2 (South)					3.06	4.76
Linde					3.63	4.62
Boron						
Ashland 2 (North)/Seaway	6,607*	11,264				
Cadmium						
Ashland 1	7.95	22.6				

Table 6-3. (continued)

Ecological COC / Remedial Unit	Surface Water (μg/L)		Sediments (mg/kg)		Surface Soils (mg/kg)	
	Mean	Upper 95	Mean	Upper 95	Mean	Upper 95
Chromium						
Ashland 1	205	469				
Ashland 2 (South)	14.6	40.6				
Ashland 2 (North)/Seaway	12.2	49.7				
Cobalt						
Ashland 1	54.1	134.0				
Ashland 2					21.6	65.1
Copper						
Ashland 1	392	964			103	447
Ashland 2 (South)	40.9	99.1			389	1360
Ashland 2 (North)/Seaway	27.8	115.2				
Linde					255	1080
Iron						
Ashland 1	56,282	222,000				
Ashland 2 (South)	39,440	137,000				
Ashland 2 (North)/Seaway	7,957	48,300				
Lead						
Ashland 1	1,018	2,700			2,153	21,100
Ashland 2 (South)	68.2	119.0			123	290
Ashland 2 (North)/Seaway	64.8	110				
Linde					127	311

Table 6-3. (continued)

Ecological COC / Remedial Unit	Surface Water (μg/L)		Sediments (mg/kg)		Surface Soils (mg/kg)	
	Mean	Upper 95	Mean	Upper 95	Mean	Upper 95
Manganese						
Ashland 1	2,420	6,620				
Ashland 2 (South)	1,829*	3,700				
Ashland 2 (North)/Seaway	775*	1,249	960	1485		
Linde					1,671	2,045
Mercury						
Ashland 2 (North)/Seaway	0.28	0.53				
Molybdenum						
Ashland 2 (North)/Seaway	94.6	213.8				
Nickel						
Ashland 1	278	805				
Ashland 2 (South)					46.9	77.8
Linde					52.3	152.8
Selenium						
Ashland 1	284	872				
Ashland 2 (South)	297	914				
Silver						
Ashland 2 (South)	8.24	17.2				
Thallium						
Ashland 1	389	775				
Ashland 2 (South)	100	239				

Table 6-3. (continued)

Ecological COC / Remedial Unit	Surface Water (μg/L)		Sediments (mg/kg)		Surface Soils (mg/kg)	
	Mean	Upper 95	Mean	Upper 95	Mean	Upper 95
Vanadium						
Ashland 1	500	1,190			80	274
Ashland 2 (South)	36.6	68.8			181	748
Ashland 2 (North)/Seaway	38.8	68.3	54.7	93.8		
Zinc						
Ashland 1					229	1060
Ashland 2 (South)			435	813	363	1234
Ashland 2 (North)/Seaway	141	614				
ORGANICS						
Benzo(b)fluoranthene						
Ashland 1	4.0	4.0			0.29	0.51
Linde					0.99	3.2
Benzo(k)fluoranthene						
Ashland 1					0.27	0.49
Linde					0.99	3.1
Benzo(g,h,i)perylene						
Ashland 1					0.30	0.50
Linde					0.90	2.2
Chlorobenzene						
Ashland 2 (South)					0.017	0.017
Dibenzofuran						
Linde					0.25	0.58

Table 6-3. (continued)

Ecological COC / Remedial Unit	Surface Water (μg/L)		Sediments (mg/kg)		Surface Soils (mg/kg)	
	Mean	Upper 95	Mean	Upper 95	Mean	Upper 95
Indeno(1,2,3-cd)pyrene						
Linde					0.79	2.1
Pyrene						
Ashland 1	7.21	11.0				
Trans-1,3-dichloropropene						
Ashland 1	2.2	2.2				
Xylenes (total)						
Ashland 2 (South)					0.044	0.044
Ashland 2 (North)/Seaway	4.2	16.0				

* Normal distribution, all others lognormal.

Table 6-4. Tonawanda Receptors and Exposure Scenarios

Source Media	Exposure Mode	Ecological Receptors	
		Onsite	Offsite
Sediment/ Surface Water	Direct contact	Midge larvae, minnows	Yellow perch
	Ingestion	NI	Carp, catfish
Soil and Groundwater	Direct contact	NI	NA
	Ingestion	Rat, earthworm	NA
Biota	Ingestion	Robin, rabbit, raccoon, mouse, seagulls	Muskrat, osprey

NA = Not applicable
NI = Not identified

Table 6-5. Ecological Quotients for Tonawanda Contaminants of Concern

Ecological COC / Remedial Unit	Surface Water		Sediments		Surface Soils	
	Mean	RME	Mean	RME	Mean	RME
METALS						
Aluminum						
Ashland 1	NA	NA				
Ashland 2 (South)	NA	NA	10.7	15.7		
Ashland 2 (North)/Seaway	NA	NA	14.6	25.3		
Antimony						
Ashland 1	1.6	3.4				
Arsenic						
Ashland 1	1.3	1.4				
Linde					10.8	12.8
Barium						
Linde					256	313
Beryllium						
Ashland 1	1.4	3.9				
Ashland 2 (South)					6.12	9.52
Linde					7.26	9.24
Boron						
Ashland 2 (North)/Seaway	6.6	11.3				
Cadmium						
Ashland 1	7.95	22.6				

Table 6-5. (continued)

Ecological COC / Remedial Unit	Surface Water		Sediments		Surface Soils	
	Mean	RME	Mean	RME	Mean	RME
Chromium						
Ashland 1	18.6 (VI) 1.7 (III)	42.6 (VI) 3.9 (III)				
Ashland 2 (South)	1.3 (VI) 0.12 (III)	3.7 (VI) 0.4 (III)				
Ashland 2 (North)/Seaway	1.1 (VI) 0.1 (III)	4.5 (VI) 0.3 (III)				
Cobalt						
Ashland 1	5.4	13.4				
Ashland 2 (South)					21.6	65.1
Copper						
Ashland 1	60.3	148.3			103	447
Ashland 2 (South)	6.3	15.2			389	1360
Ashland 2 (North)/Seaway	4.3	17.7				
Linde					255	1080
Iron						
Ashland 1	NA	NA				
Ashland 2 (South)	NA	NA				
Ashland 2 (North)/Seaway	NA	NA				

Table 6-5. (continued)

Ecological COC / Remedial Unit	Surface Water		Sediments		Surface Soils	
	Mean	RME	Mean	RME	Mean	RME
Lead						
Ashland 1	318.1	843.8			2153	21,100
Ashland 2 (South)	21.3	37.2			123	324
Ashland 2 (North)/Seaway	20.3	34.4				
Linde					127	311
Manganese						
Ashland 1	NA	NA				
Ashland 2 (South)	NA	NA				
Ashland 2 (North)/Seaway	NA	NA	1.92	2.97		
Linde					3.42	4.12
Mercury						
Ashland 2 (North)/Seaway	23.3	44.2				
Molybdenum						
Ashland 2 (North)/Seaway	0.19	0.43				
Nickel						
Ashland 1	1.7	5.0				
Ashland 2 (South)					9.4	15.6
Linde					10.5	30.6
Selenium						
Ashland 1	56.8	174.4				
Ashland 2 (South)	59.4	182.8				
Silver						
Ashland 2 (South)	68.7	143.3				

Table 6-5. (continued)

Ecological COC / Remedial Unit	Surface Water		Sediments		Surface Soils	
	Mean	RME	Mean	RME	Mean	RME
Thallium						
Ashland 1	NA	NA				
Ashland 2 (South)	NA	NA				
Vanadium						
Ashland 1	NA	NA			8.0	27.4
Ashland 2 (South)	NA	NA			18.1	74.8
Ashland 2 (North)/Seaway	NA	NA	5.5	9.4		
Zinc						
Ashland 1					22.9	106
Ashland 2 (South)			43.5	81.3	36.3	123
Ashland 2 (North)/Seaway	1.3	5.6				
ORGANICS						
Benzo(b)fluoranthene						
Ashland 1	NA	NA			NA	NA
Linde					NA	NA
Benzo(k)fluoranthene						
Ashland 1					NA	NA
Linde					NA	NA
Benzo(g,h,i)perylene						
Ashland 1					NA	NA
Linde					NA	NA

Table 6-5. (continued)

Ecological COC / Remedial Unit	Surface Water		Sediments		Surface Soils	
	Mean	RME	Mean	RME	Mean	RME
Chlorobenzene						
Ashland 2 (South)					0.001	0.001
Dibenzofuran						
Linde					NA	NA
Indeno(1,2,3-cd)pyrene						
Linde					NA	NA
Pyrene						
Ashland 1	NA	NA				
Trans-1,3-dichloropropene						
Ashland 1	NA	NA				
Xylenes (total)						
Ashland 2 (South)					1.8E-4	1.8E-4
Ashland 2 (North)/Seaway	0.017	0.064				

Table 6-6. Tonawanda Hypothetical Exposure Factors

		Source Media/Exposure Mode					
Ecological Receptors	Location	Surface Water and Sediment Direct Contact	Surface Water and Sediment Ingestion	Soil and Groundwater Direct Contact	Soil and Groundwater Ingestion	Biota Ingestion	Exposure Factor
Aquatic organisms, non-sediment dwellers	Onsite	1	0	0	0	0	1
	Offsite	0	0	0	0	0.1	0.1
Aquatic organisms, sediment dwellers	Onsite	1	0.1	0	0	0	1.1
	Offsite	0	0	0	0	0	0
Terrestrial organisms, non-soil dwellers	Onsite	0	0	0	0	1	1 x BCF
	Offsite	0	0	0	0	0.1	0.1 x BCF
Terrestrial organisms, soil dwellers	Onsite	0	0	1	0.1	0	1.1
	Offsite	0	0	0	0	0	0

BCF = Bioconcentration Factor (see Table 6-1)

Table 6-7. Major Sources* of Risk for Tonawanda Ecological Receptors

Aquatic Organisms		Terrestrial Organisms		
Onsite	Offsite	Onsite		Offsite
Direct Contact and Ingestion	Trophic	Direct Contact and Ingestion	Trophic	Trophic
Lead	Zinc	Copper	Copper	Copper
Selenium	Selenium	Lead	Lead	Lead
Copper	Copper	Barium	Barium	Barium
Silver	Silver	Arsenic	Arsenic	Arsenic
		Cobalt	Cobalt	
		Nickel	Nickel	
Mercury		Zinc	Zinc	Zinc
Chromium		Vanadium	Vanadium	Vanadium
Cadmium		Beryllium		
Cobalt				
XQs cannot be calculated for the following chemicals:				
Aluminum	Aluminum	Benzo(b)-fluoranthene	Benzo(b)-fluoranthene	Benzo(b)-fluoranthene
Molybdenum	Pyrene	Benzo(k)-fluoranthene	Benzo(k)-fluoranthene	Benzo(k)-fluoranthene
Pyrene	Trans-1,3-dichloropropene	Benzo(g,h,i)-perylene	Benzo(g,h,i)-perylene	Benzo(g,h,i)-perylene
Trans-1,3-dichloropropene	Xylenes	Dibenzofuran	Dibenzofuran	Dibenzofuran
Xylenes		Indeno(1,2,3-cd)pyrene	Indeno(1,2,3-cd)pyrene	Indeno(1,2,3-cd)pyrene
		Xylenes	Xylenes	

*XQs > 10

Table 6-8. Summary of Ecological Quotients (EQs) at Tonawanda Site: Surface Water

	Linde	Ashland 1	Ashland 2 (North)/Seaway	Ashland 2 (South)
$10^2 \leq EQ < 10^3$		copper lead selenium		selenium silver
$10^1 \leq EQ < 10^2$		cadmium chromium (VI) cobalt	boron copper lead mercury	copper lead
$10^0 \leq EQ < 10^1$		antimony arsenic beryllium chromium (III) nickel	chromium (VI) zinc	chromium (VI)
Unknown Risk	radium-226+D thorium-232+D uranium-238+D	aluminum iron manganese thallium vanadium radium-226+D thorium-232+D uranium-238+D benzo(b)fluoranthene pyrene trans-1,3-dichloropropene	aluminum iron manganese thallium vanadium radium-226+D thorium-232+D uranium-238+D	aluminum iron manganese thallium vanadium radium-226+D thorium-232+D uranium-238+D
$EQ < 1$			molybdenum xylenes (total)	

Table 6-9. Summary of Ecological Quotients (EQs) at Tonawanda Site: Surface Soils

	Linde	Ashland 1	Ashland 2 (North)/Seaway	Ashland 2 (South)
$10^4 \leq EQ < 10^5$		lead		
$10^3 \leq EQ < 10^4$	copper			copper
$10^2 \leq EQ < 10^3$	barium lead	copper zinc		lead zinc
$10^1 \leq EQ < 10^2$	arsenic nickel	vanadium		cobalt nickel vanadium
$10^0 \leq EQ < 10^1$	beryllium manganese			beryllium
Unknown Risk	radium-226+D thorium-232+D uranium-238+D benzo(b)fluoranthene benzo(k)fluoranthene benzo(g,h,i)perylene dibenzofuran indeno(1,2,3-cd)pyrene	radium-226+D thorium-232+D uranium-238+D benzo(b)fluoranthene benzo(k)fluoranthene benzo(g,h,i)perylene	radium-226+D thorium-232+D uranium-238+D	radium-226+D thorium-232+D uranium-238+D
EQ < 1				chlorobenzene xylenes (total)

Table 6-10. Summary of Ecological Quotients (EQs) at Tonawanda Site: Sediments

	Linde	Ashland 1	Ashland 2 (North)/Seaway	Ashland 2 (South)
$10^1 \leq EQ < 10^2$			aluminum	aluminum zinc
$10^0 \leq EQ < 10^1$			manganese vanadium	
Unknown Risk	radium-226+D thorium-232+D uranium-238+D	radium-226+D thorium-232+D uranium-238+D	radium-226+D thorium-232+D uranium-238+D	radium-226+D thorium-232+D uranium-238+D
$EQ < 1$				

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APPENDIX A

DOSE CALCULATION TABLES

APPENDIX A
DOSE CALCULATION TABLES

Appendix A contains tables of incremental exposure dose calculations including soil ingestion, inhalation, direct remediation, and radon for the Tonawanda, New York properties, exposure scenarios, and receptors. The results presented on these tables are discussed in Section 3. Listed below are the tables included in this Appendix.

Table A-1. Estimated Exposure Dosage - Current Employee

Table A-2. Estimated Exposure Dosage - Current Transient

Table A-3. Estimated Exposure Dosage - Future Employee

Table A-4. Estimated Exposure Dosage - Future Transient

Table A-5. Slope Factor Risk - Current Employee

Table A-6. Slope Factor Risk - Current Transient

Table A-7. Slope Factor Risk - Future Employee

Table A-8. Slope Factor Risk - Future Transient (Child)

Table A-9. Dose Risk/Slope Factor Risk

Table A-1. Estimated Exposure Dosage - Current Employee

(mrem/yr)

LOCATION	SUBAREA	DIRECT RAD.		INHALATION		IND. RADON		SOIL INGESTION		TOTAL w/o Rn		TOTAL DOSE	
		X	RME	X	RME	X	RME	X	RME	X	RME	X	RME
LINDE	A	12.839	23.875	0.1	0.5459	2.3946	2.4119	0.0476	0.152	12.987	24.573	15.381	26.985
	B	0.2626	0.6394	0	0	0.3216	0.5877	0	0	0.2626	0.6394	0.5842	1.2271
ASHLAND 1	A												
	B												
ASHLAND 2	A												
	B												
SEAWAY	A												
	B												

X --- Mean

RME --- Reasonable Maximum Exposure

Table A-2. Estimated Exposure Dosage - Current Transient

(mrem/yr)

LOCATION	SUBAREA	DIRECT RAD.		INHALATION		IND. RADON		SOIL INGESTION		WATER INGESTION		TOTAL w/o Rn		TOTAL DOSE	
		X	RME	X	RME	X	RME	X	RME	X	RME	X	RME	X	RME
LINDE	A														
	B														
ASHLAND 1	A	0.0168	0.3866	0.0006	0.0355	2.3E-05	0.0002	9.2E-05	0.011	0	0	0.0175	0.4331	0.0175	0.4332
	B	0.1871	4.734	0.0038	1.221	0.0007	0.0155	0.0006	0.2542	0	0	0.1915	6.2092	0.1923	6.2247
ASHLAND 2	A	0.0693	2.7367	0.0015	0.2589	0.0005	0.004	0.0002	0.0403	0	0	0.0709	3.0359	0.0714	3.0399
	B	0.0008	0.2749	6.3E-05	0.0189	3.4E-05	0.0019	3.2E-06	0.0027	0	0	0.0009	0.2964	0.0009	0.2983
SEAWAY	A	0.1026	2.691	0.0003	0.0259	0.0009	0.0033	0.0003	0.0383	0	0	0.1032	2.7552	0.1041	2.7585
	B														
LOCAL CREEK	A	0.0261	0.0395	0.0016	0.0084	5.1E-05	6.6E-05	5.2E-06	1.6E-05	0	0	0.0277	0.0479	0.0277	0.048

X --- Mean

RME --- Reasonable Maximum Exposure

Table A-3. Estimated Exposure Dosage - Future Employee

(mrem/yr)

LOCATION	SUBAREA	DIRECT RAD.		INHALATION		IND. RADON		SOIL INGESTION		TOTAL w/o Rn		TOTAL DOSE	
		X	RME	X	RME	X	RME	X	RME	X	RME	X	RME
LINDE	A	12.839	23.875	0.1	0.5459	2.3946	2.4119	0.0476	0.152	12.987	24.573	15.381	26.985
	B	0.7677	2.081	0.0062	0.0479	0.2082	0.4171	0.0027	0.0072	0.7765	2.1362	0.9847	2.5533
ASHLAND 1	A	1.109	5.214	0.0076	0.0564	0.4412	0.4091	0.0032	0.0223	1.1199	5.2927	1.5611	5.7018
	B	112	478.7	0.8058	10.2	46.33	169.6	0.3502	1.658	113.16	490.56	159.49	660.16
ASHLAND 2	A	6.132	26.94	0.0434	0.2851	3.592	3.089	0.0187	0.118	6.1941	27.343	9.7861	30.432
	B	0.0607	1.0444	0.0025	0.0368	0.0422	0.0959	0.0004	0.0056	0.0636	1.0869	0.1058	1.1828
SEAWAY	A												
	B												

X --- Mean

RME --- Reasonable Maximum Exposure

Table A-4. Estimated Exposure Dosage - Future Transient(Child)

(mrem/yr)

LOCATION	SUBAREA	DIRECT RAD.		INHALATION		IND. RADON		SOIL INGESTION		WATER INGESTION		TOTAL w/o Rn		TOTAL DOSE	
		X	RME	X	RME	X	RME	X	RME	X	RME	X	RME	X	RME
LINDE	A														
	B														
ASHLAND 1	A														
	B														
ASHLAND 2	A														
	B														
SEAWAY	A	0.1226	12.98	0.0016	0.2595	0.0011	0.0184	0.0002	0.1008	0	0	0.1244	13.34	0.1255	13.359
	B														
LOCAL CREEK	A	0.0261	0.0395	0.0016	0.0084	5.1E-05	6.6E-05	5.2E-06	1.6E-05	0	0	0.0277	0.0479	0.0277	0.048

X --- Mean

RME --- Reasonable Maximum Exposure

Table A-5. Slope Factor Risk - Current Employee

LOCATION	SUBAREA	DIRECT RAD.		INHALATION		IND. RADON		SOIL INGESTION		TOTAL w/o Rn		TOTAL DOSE	
		X	RME	X	RME	X	RME	X	RME	X	RME	X	RME
LINDE	A	1.0E-04	2.7E-04	6.1E-07	4.6E-06	5.3E-06	1.0E-05	9.6E-08	4.2E-07	1.1E-04	2.7E-04	1.1E-04	2.8E-04
	B	2.1E-06	7.2E-06	0.0E+00	0.0E+00	7.1E-07	2.4E-06	0.0E+00	0.0E+00	2.1E-06	7.2E-06	2.9E-06	9.5E-06
ASHLAND 1	A												
	B												
ASHLAND 2	A												
	B												
SEAWAY	A												
	B												

X --- Mean

RME --- Reasonable Maximum Exposure

Table A-6. Slope Factor Risk - Current Transient

LOCATION	SUBAREA	DIRECT RAD.		INHALATION		IND. RADON		SOIL INGESTION		WATER INGESTION		TOTAL w/o Rn		TOTAL DOSE	
		X	RME	X	RME	X	RME	X	RME	X	RME	X	RME	X	RME
LINDE	A														
	B														
ASHLAND 1	A	4.4E-08	3.6E-06	5.9E-10	1.2E-07	3.4E-10	2.1E-08	4.5E-11	1.8E-08	0.0E+00	0.0E+00	4.5E-08	3.8E-06	4.5E-08	3.8E-06
	B	4.9E-07	4.4E-05	4.5E-09	3.5E-06	1.2E-08	1.0E-06	3.3E-10	2.7E-07	0.0E+00	0.0E+00	4.9E-07	4.7E-05	5.0E-07	4.8E-05
ASHLAND 2	A	1.6E-07	6.2E-06	1.3E-09	1.9E-07	2.7E-09	6.7E-08	6.7E-11	1.5E-08	0.0E+00	0.0E+00	1.6E-07	6.4E-06	1.6E-07	6.4E-06
	B	1.9E-09	6.2E-07	4.4E-11	1.5E-08	1.1E-10	1.9E-08	6.7E-13	9.5E-10	0.0E+00	0.0E+00	1.9E-09	6.4E-07	2.0E-09	6.6E-07
SEAWAY	A	2.7E-07	2.5E-05	4.9E-10	1.6E-07	7.4E-09	2.5E-07	1.9E-10	9.2E-08	0.0E+00	0.0E+00	2.7E-07	2.6E-05	2.8E-07	2.6E-05
	B														
LOCAL CREEK	A	5.5E-08	8.2E-08	2.6E-09	1.4E-08	6.5E-10	1.2E-09	1.9E-12	5.7E-12	0.0E+00	0.0E+00	5.8E-08	9.6E-08	5.8E-08	9.7E-08

X --- Mean
RME --- Reasonable Maximum Exposure

Table A-7. Slope Factor Risk - Future Employee

LOCATION	SUBAREA	DIRECT RAD.		INHALATION		IND. RADON		SOIL INGESTION		TOTAL w/o Rn		TOTAL DOSE	
		X	RME	X	RME	X	RME	X	RME	X	RME	X	RME
LINDE	A	1.0E-04	2.7E-04	6.1E-07	4.6E-06	5.3E-06	1.0E-05	9.6E-08	4.2E-07	1.1E-04	2.7E-04	1.1E-04	2.8E-04
	B	6.3E-06	2.3E-05	2.8E-08	2.7E-07	4.6E-07	1.7E-06	4.9E-09	1.7E-08	6.3E-06	2.4E-05	6.8E-06	2.5E-05
ASHLAND 1	A	2.9E-06	6.9E-05	4.9E-09	5.4E-07	3.1E-07	1.7E-06	1.6E-09	7.9E-08	2.9E-06	7.0E-05	3.3E-06	7.2E-05
	B	3.0E-04	4.5E-03	5.3E-07	2.4E-05	3.3E-05	5.7E-04	1.8E-07	3.1E-06	3.0E-04	4.5E-03	3.3E-04	5.1E-03
ASHLAND 2	A	1.6E-05	2.5E-04	2.8E-08	7.2E-07	2.5E-06	1.2E-05	9.6E-09	2.5E-07	1.6E-05	2.6E-04	1.9E-05	2.7E-04
	B	1.6E-07	9.9E-06	2.0E-09	1.3E-07	3.3E-08	4.6E-07	1.0E-10	8.3E-09	1.6E-07	1.0E-05	1.9E-07	1.0E-05
SEAWAY	A												
	B												

X --- Mean

RME --- Reasonable Maximum Exposure

Table A-8. Slope Factor Risk - Future Transient(Child)

LOCATION	SUBAREA	DIRECT RAD.		INHALATION		IND. RADON		SOIL INGESTION		WATER INGESTION		TOTAL w/o Rn		TOTAL DOSE	
		X	RME	X	RME	X	RME	X	RME	X	RME	X	RME	X	RME
LINDE	A														
	B														
ASHLAND 1	A														
	B														
ASHLAND 2	A														
	B														
SEAWAY	A	2.8E-07	2.9E-05	8.6E-10	1.5E-07	6.2E-09	3.3E-07	9.1E-11	5.1E-08	0.0E+00	0.0E+00	2.8E-07	3.0E-05	2.9E-07	3.0E-05
	B														
LOCAL CREEK	A	5.5E-08	8.2E-08	2.6E-09	1.4E-08	6.5E-10	1.2E-09	1.9E-12	5.7E-12	0.0E+00	0.0E+00	5.8E-08	9.6E-08	5.8E-08	9.7E-08

X — Mean
RME — Reasonable Maximum Exposure

TABLE A-9. Dose Risk / Slope Factor Risk

CURRENT USE SCENARIO					
LOCATION	SUBAREA	Employee		Transient	
		X	RME	X	RME
LINDE	A	0.58	1.44		
	B	0.86	1.93		
ASHLAND 1	A			2.11	2.07
	B			2.06	2.32
ASHLAND 2	A			2.41	8.51
	B			2.48	8.16
SEAWAY	A			2.02	1.92
	B				
LOCAL CREEK	A			2.57	8.92
FUTURE USE SCENARIO					
LOCATION	SUBAREA	Employee		Transient	
		X	RME	X	RME
LINDE	A	0.58	1.44		
	B	0.61	1.51		
ASHLAND 1	A	2.01	1.19		
	B	2.04	1.94		
ASHLAND 2	A	2.19	1.71		
	B	2.28	1.69		
SEAWAY	A			2.38	8.03
	B				
LOCAL CREEK	A			2.57	8.92

X --- Mean
RME --- Reasonable Maximum Exposure

APPENDIX B

GENERAL ANALYTICAL ASSUMPTIONS

APPENDIX B
GENERAL ANALYTICAL ASSUMPTIONS

Appendix B contains tables of values assumed in the computation of doses and risks for the various scenarios. Listed below are the tables included in this appendix.

Table B-1. Values Assumed for Scenario Parameters

Table B-2. Scenario/Receptor Descriptions

Table B-3. Input Parameters for RESRAD

Table B-4. Dose Conversion Factors

Table B-1. Values Assumed for Scenario Parameters

Parameter	Unit		Employee	Transient Adult	Transient Child	Wading Child[a]	Reference
Exposure time indoors	h/d		7	NA	NA	NA	EPA 1990
Exposure time outdoors	h/d	AVE: RME:	0.2 1.0	0.25 1	0.5 2	1 1	EPA 1990
Exposure frequency (EF)	d/yr	AVE: RME:	250 250	100 100	50 130	7 7	EPA 1990 EPA 1991b
Exposure duration	yr	AVE: RME:	22[b]7 30[b]25	6 24	6 6	6 6	EPA 1989a EPA 1990
Body weight	kg		70	70	35	35	EPA 1991b
Inhalation rate	m^3/d	AVE: RME:	15 20	20 24	16 24	16 24	EPA 1990 EPA 1991b
Dust concentration in air	$\mu g/m^3$	AVE: RME:	100 200	100 200	100 200	100 200	Gilbert 1983 Paustenbach 1989
Amount of dust from contaminated soil	%		50	50	50	50	Gilbert 1983 Paustenbach 1989
Amount of respirable dust	%		30	30	30	30	Gilbert 1983 Paustenbach 1989
Amount of dust assumed to be present indoors	%		40	40	40	40	based on value in Alzona et al. 1979
Soil ingestion rate	mg/d	AVE: RME:	30 50	50 100	50 100	50 100	EPA 1989a EPA 1990 EPA 1991b
Water ingestion rate	l/d	AVE: RME:	NA	NA	NA	0.035 0.05	EPA 1989a
External gamma shielding factor			0.8	NA	NA	NA	EPA 1989a

NA = not applicable
[a] applies to a child wading in Rattlesnake Creek
[b] applies to employee at Linde

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Table B-2. Scenario/Receptor Descriptions

Employee:

CURRENT AND FUTURE:

Both the average and RME employee are estimated to spend 7 hours a day working inside the building. No drinking water is consumed from the site. Based on current work patterns, the average employee at Linde is assumed to work 22 years and the RME employee is assumed to work 30 years. Employees at the other properties are assumed to follow national trends and work an average of 7 years and an RME of 25 years at one site.

Adult Transient:

CURRENT AND FUTURE:

The adult transient is represented as someone who occasionally visits the site to make deliveries, monitor the site, or dump a truckload at the landfill. The average person spends 15 minutes per day, 2 days per week, 50 weeks per year at the site. The RME spends 1 hour per day, 250 days per year at the site.

Child Transient:

CURRENT AND FUTURE:

The average child transient is an older child who occasionally plays at the site (0.5 hr/day, 2 days/wk, 25 wk/yr). The RME is an older child who frequently plays at the site (5 day/wk [10 hr/wk] during the 10 week summer session and 2 day/wk [4 hr/wk] during the remainder of the year).

Wading Child:

CURRENT AND FUTURE:

Both the average and RME individuals are represented as an older child wading in the brook. The child is assumed to play in the brook 7 times a year for 1 hour each.

Table B-3. Input Parameters for RESRAD

Site Specific Parameters:

Area of contaminated zone (m^2)

Linde A	53,000
Linde B	2,000
Ashland 1A	40,000
Ashland 1B	32,900
Ashland 2A	116,500
Ashland 2B	285,700
Seaway A	150,000
Seaway B	28,400

Time since placement of material (yr) 0

Thickness of contaminated zone (m)	Surface	Subsurface
All properties	0.15	2.0

Cover Depth (m)	Surface	Subsurface
All properties	0	0.15

Total porosity	0.45
Saturated zone/hydraulic conductivity (m/yr)	123
Evapotranspiration coefficient	0.46
Precipitation (m/yr)	1.23
Runoff coefficinet	0.25
Unsaturated zone/hydraulic conductivity (m/yr)	9.7

Table B-4. Dose Conversion Factors

Parent Radionuclide	Inhalation mrem/pCi	Ingestion mrem/pCi	Direct (mrem/yr)/(pCi/cm^3)
Thorium-232	1.6	2.8×10^{-3}	6.04×10^{-3}
Radium-228+D	4.5×10^{-3}	1.2×10^{-3}	4.51
Thorium-228+D	3.1×10^{-1}	7.5×10^{-4}	7.36
Uranium-238+D	1.2×10^{-1}	2.5×10^{-4}	6.97×10^{-2}
Uranium-234	1.3×10^{-1}	2.6×10^{-4}	6.97×10^{-4}
Thorium-230	2.6×10^{-1}	5.3×10^{-4}	1.03×10^{-3}
Radium-226+D	7.9×10^{-3}	1.1×10^{-3}	8.56
Lead 210+D	2.1×10^{-2}	6.7×10^{-3}	2.31×10^{-3}
Uranium-235+D	1.2×10^{-1}	2.5×10^{-4}	4.9×10^{-1}
Protactinium	1.3	1.1×10^{-2}	1.21×10^{-1}
Actinium-227+D	6.7	1.5×10^{-2}	1.52

D = Daughters

Source: Yu, C. 1991

APPENDIX C

CHEMICAL CONTAMINANTS EXPOSURE AND RISK ESTIMATES

APPENDIX C

CHEMICAL CONTAMINANTS EXPOSURE AND RISK ESTIMATES

Appendix C contains tables of intake and risk estimates for chemical contaminants of concern. Results are tabulated as total cancer risk or total hazard index for carcinogens and noncarcinogens, respectively for the Tonawanda properties.

Table C-1	Risk from Surface Soil Ingestion at Linde Using Mean Values Receptor: Current and Future Employee
Table C-2	Risk from Surface Soil Ingestion at Linde Using RME Values Receptor: Current and Future Employee
Table C-3	Risk from Surface Soil Particulate Inhalation at Linde Using Mean Values Receptor: Current and Future Employee
Table C-4	Risk from Surface Soil Particulate Inhalation at Linde Using RME Values Receptor: Current and Future Employee
Table C-5	Risk from Surface Soil Ingestion at Ashland 1 Using Mean Values Receptor: Current Transient (Adult)
Table C-6	Risk from Surface Soil Ingestion at Ashland 1 Using RME Values Receptor: Current Transient (Adult)
Table C-7	Risk from Surface Soil Particulate Inhalation at Ashland 1 Using Mean Values Receptor: Current Transient (Adult)
Table C-8	Risk from Surface Soil Particulate Inhalation at Ashland 1 Using RME Values Receptor: Current Transient (Adult)
Table C-9	Risk from Surface Soil Ingestion at Ashland 1 Using Mean Values Receptor: Future Employee
Table C-10	Risk from Surface Soil Ingestion at Ashland 1 Using RME Values Receptor: Future Employee
Table C-11	Risk from Surface Soil Particulate Inhalation at Ashland 1 Using Mean Values Receptor: Future Employee
Table C-12	Risk from Surface Soil Particulate Inhalation at Ashland 1 Using RME Values Receptor: Future Employee
Table C-13	Risk from Surface Soil Ingestion at Ashland 2 Using Mean Values Receptor: Current Transient (Older Child)
Table C-14	Risk from Surface Soil Ingestion at Ashland 2 Using RME Values Receptor: Current Transient (Older Child)
Table C-15	Risk from Surface Soil Particulate Inhalation at Ashland 2 Using Mean Values Receptor: Current Transient (Older Child)

Table C-16 Risk from Surface Soil Particulate Inhalation at Ashland 2 Using RME Values
Receptor: Current Transient (Older Child)

Table C-17 Risk from Surface Soil Ingestion at Ashland 2 Using Mean Values
Receptor: Future Employee

Table C-18 Risk from Surface Soil Ingestion at Ashland 2 Using RME Values
Receptor: Future Employee

Table C-19 Risk from Surface Soil Particulate Inhalation at Ashland 2 Using Mean Values
Receptor: Future Employee

Table C-20 Risk from Surface Soil Particulate Inhalation at Ashland 2 Using RME Values
Receptor: Future Employee

Table C-21 Risk from Sediment Ingestion at Local Creek Using Mean Values
Receptor: Wading Older Child

Table C-22 Risk from Sediment Ingestion at Local Creek Using RME Values
Receptor: Wading Older Child

Table C-23 Risk from Surface Water Ingestion at Local Creek Using Mean Values
Receptor: Wading Older Child

Table C-24 Risk from Surface Water Ingestion at Local Creek Using RME Values
Receptor: Wading Older Child

TABLE C-1
RISK FROM SOIL INGESTION AT LINDE
RECEPTOR: CURRENT AND FUTURE EMPLOYEE
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	1.21E+00	7.30E+00	1.12E-07	8.15E-07
BENZO(A)PYRENE	1.05E+00	7.30E+00	9.69E-08	7.07E-07
BENZO(K)FLOURANTHENE	9.86E-01	7.30E+00	9.10E-08	6.64E-07
CHRYSENE	1.40E+00	7.30E+00	1.29E-07	9.43E-07
DIBENZO(A,H)ANTHRACENE	4.20E-01	7.30E+00	3.87E-08	2.83E-07
INDENO(1,2,3-CD)PYRENE	7.91E-01	7.30E+00	7.30E-08	5.33E-07
BIS(2-ETHYLHEXYL)PHTHALATE	3.65E-01	7.00E+00	3.37E-08	2.36E-07
METHYLENE CHLORIDE	1.10E-02	7.50E-03	1.01E-09	7.61E-12
ARSENIC	9.91E+01	1.75E+00	9.14E-06	1.60E-05
BERYLLIUM	3.63E+00	4.30E+00	3.35E-07	1.44E-06
CADMIUM	0.00E+00	ND	0.00E+00	ND
			TOTAL CANCER RISK =	2.16E-05

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPHTHENE	3.31E-01	6.00E-02	9.72E-08	1.62E-06
ANTHRACENE	3.50E-01	3.00E-01	1.03E-07	3.42E-07
BENZO(B)FLUORANTHENE	9.90E-01	ND	2.91E-07	0.00E+00
BENZO(G,H,I)PERYLENE	9.04E-01	ND	2.65E-07	0.00E+00
FLUORANTHENE	2.17E+00	4.00E-02	6.37E-07	1.59E-05
FLUORENE	2.83E-01	4.00E-02	8.31E-08	2.08E-06
NAPHTHALENE	2.40E-01	4.00E-02	7.05E-08	1.76E-06
PHENANTHRENE	1.62E+00	ND	4.76E-07	0.00E+00
PYRENE	1.90E+00	3.00E-02	5.58E-07	1.86E-05
BIS(2-ETHYLHEXYL)PHTHALATE	3.65E-01	2.00E-02	1.07E-07	5.36E-06
DIBENZOFURAN	2.46E-01	4.00E-03	7.22E-08	1.81E-05
DI-N-BUTYLPHTHALATE	2.80E-01	1.00E-01	8.22E-08	8.22E-07
2-METHYLNAPHTHALENE	2.51E-01	ND	7.37E-08	0.00E+00
ACETONE	6.60E-02	1.00E-01	1.94E-08	1.94E-07
CHLOROBENZENE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	1.10E-02	6.00E-02	3.23E-09	5.38E-08
TOLUENE	5.60E-03	2.00E-01	1.64E-09	8.22E-09
XYLENES(TOTAL)	0.00E+00	2.00E+00	0.00E+00	0.00E+00
ARSENIC	9.91E+01	3.00E-04	2.91E-05	9.70E-02
BARIUM	2.56E+02	7.00E-02	7.51E-05	1.07E-03
BERYLLIUM	3.63E+00	5.00E-03	1.07E-06	2.13E-04
BORON	5.21E+01	9.00E-02	1.53E-05	1.70E-04
CADMIUM	2.08E+00	5.00E-04	6.11E-07	1.22E-03
CALCIUM	1.14E+05	ND	3.35E-02	0.00E+00
COBALT	0.00E+00	9.60E-01	0.00E+00	0.00E+00
COPPER	2.55E+02	4.00E-02	7.49E-05	1.87E-03
LEAD	1.27E+02	ND	3.73E-05	0.00E+00
MAGNESIUM	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	1.67E+03	1.00E-01	4.90E-04	4.90E-03
NICKEL	5.23E+01	2.00E-02	1.54E-05	7.68E-04
URANIUM	9.83E+01	3.00E-03	2.89E-05	9.62E-03
VANADIUM	0.00E+00	7.00E-03	0.00E+00	0.00E+00
ZINC	0.00E+00	ND	0.00E+00	0.00E+00
			HAZARD INDEX =	1.17E-01

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = UCL95 chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (250 d/yr)
ED = Exposure duration (yr)
BW = Body weight (70 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-2
RISK FROM SOIL INGESTION AT LINDE
RECEPTOR: CURRENT AND FUTURE EMPLOYEE
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	7.33E+00	7.30E+00	1.54E-06	1.12E-05
BENZO(A)PYRENE	3.00E+00	7.30E+00	6.29E-07	4.59E-06
BENZO(K)FLOURANTHENE	3.10E+00	7.30E+00	6.50E-07	4.74E-06
CHRYSENE	3.90E+00	7.30E+00	8.18E-07	5.97E-06
DIBENZO(A,H)ANTHRACENE	4.20E-01	7.30E+00	8.81E-08	6.43E-07
INDENO(1,2,3-CD)PYRENE	2.10E+00	7.30E+00	4.40E-07	3.21E-06
BIS(2-ETHYLHEXYL)PHTHALATE	6.04E-01	7.00E+00	1.27E-07	8.86E-07
METHYLENE CHLORIDE	1.10E-02	7.50E-03	2.31E-09	1.73E-11
ARSENIC	1.27E+02	1.75E+00	2.66E-05	4.66E-05
BERYLLIUM	4.62E+00	4.30E+00	9.69E-07	4.17E-06
CADMIUM	0.00E+00	ND	0.00E+00	ND
			TOTAL CANCER RISK =	8.20E-05

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPHTHENE	4.95E-01	6.00E-02	2.42E-07	4.04E-06
ANTHRACENE	8.70E-01	3.00E-01	4.26E-07	1.42E-06
BENZO(B)FLUORANTHENE	3.20E+00	ND	1.57E-06	0.00E+00
BENZO(G,H,I)PERYLENE	2.20E+00	ND	1.08E-06	0.00E+00
FLUORANTHENE	7.00E+00	4.00E-02	3.42E-06	8.56E-05
FLUORENE	5.76E-01	4.00E-02	2.82E-07	7.05E-06
NAPHTHALENE	7.50E-01	4.00E-02	3.67E-07	9.17E-06
PHENANTHRENE	4.70E+00	ND	2.30E-06	0.00E+00
PYRENE	6.20E+00	3.00E-02	3.03E-06	1.01E-04
BIS(2-ETHYLHEXYL)PHTHALATE	6.04E-01	2.00E-02	2.95E-07	1.48E-05
DIBENZOFURAN	5.83E-01	4.00E-03	2.85E-07	7.13E-05
DI-N-BUTYLPHTHALATE	2.80E-01	1.00E-01	1.37E-07	1.37E-06
2-METHYLNAPHTHALENE	6.28E-01	ND	3.07E-07	0.00E+00
ACETONE	6.60E-02	1.00E-01	3.23E-08	3.23E-07
CHLOROBENZENE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	1.10E-02	6.00E-02	5.38E-09	8.97E-08
TOLUENE	5.60E-03	2.00E-01	2.74E-09	1.37E-08
XYLENES(TOTAL)	0.00E+00	2.00E+00	0.00E+00	0.00E+00
ARSENIC	1.27E+02	3.00E-04	6.21E-05	2.07E-01
BARIUM	3.13E+02	7.00E-02	1.53E-04	2.19E-03
BERYLLIUM	4.62E+00	5.00E-03	2.26E-06	4.52E-04
BORON	6.27E+01	9.00E-02	3.07E-05	3.41E-04
CADMIUM	2.70E+00	5.00E-04	1.32E-06	2.64E-03
CALCIUM	1.40E+05	ND	6.85E-02	0.00E+00
COBALT	0.00E+00	9.60E-01	0.00E+00	0.00E+00
COPPER	1.08E+03	4.00E-02	5.28E-04	1.32E-02
LEAD	3.11E+02	ND	1.52E-04	0.00E+00
MAGNESIUM	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	2.05E+03	1.00E-01	1.00E-03	1.00E-02
NICKEL	1.53E+02	2.00E-02	7.49E-05	3.74E-03
URANIUM	1.38E+02	3.00E-03	6.74E-05	2.25E-02
VANADIUM	0.00E+00	7.00E-03	0.00E+00	0.00E+00
ZINC	0.00E+00	ND	0.00E+00	0.00E+00
			HAZARD INDEX =	2.62E-01

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = UCL95 chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (50 mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (250 d/yr)
ED = Exposure duration (30 yr)
BW = Body weight (70 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-3
RISK FROM SURFACE SOIL PARTICULATE INHALATION AT LINDE
RECEPTOR: CURRENT AND FUTURE EMPLOYEE
USING MEAN VALUES

CONTAMINANTS OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	1.21E+00	1.82E-08	ND	6.98E-12	0.00E+00
BENZO(A)PYRENE	1.05E+00	1.58E-08	6.10E+00	6.05E-12	3.69E-11
BENZO(K)FLOURANTHENE	9.86E-01	1.48E-08	ND	5.69E-12	0.00E+00
CHRYSENE	1.40E+00	2.10E-08	ND	8.07E-12	0.00E+00
DIBENZO(A,H)ANTHRACENE	4.20E-01	6.30E-09	ND	2.42E-12	0.00E+00
INDENO(1,2,3-CD)PYRENE	7.91E-01	1.19E-08	ND	4.56E-12	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	3.65E-01	5.48E-09	ND	2.10E-12	0.00E+00
METHYLENE CHLORIDE	1.10E-02	1.65E-10	ND	6.34E-14	0.00E+00
ARSENIC	9.91E+01	1.49E-06	5.00E+01	5.71E-10	2.86E-08
BERYLLIUM	3.63E+00	5.45E-08	8.40E+00	2.09E-11	1.76E-10
CADMIUM	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
				TOTAL CANCER RISK =	2.88E-08

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPHTHENE	3.31E-01	4.97E-09	ND	6.07E-12	0.00E+00
ANTHRACENE	3.50E-01	5.25E-09	ND	6.42E-12	0.00E+00
BENZO(B)FLUORANTHENE	9.90E-01	1.49E-08	ND	1.82E-11	0.00E+00
BENZO(G,H,I)PERYLENE	9.04E-01	1.36E-08	ND	1.66E-11	0.00E+00
FLUORANTHENE	2.17E+00	3.26E-08	ND	3.98E-11	0.00E+00
FLUORENE	2.83E-01	4.25E-09	ND	5.19E-12	0.00E+00
NAPHTHALENE	2.40E-01	3.60E-09	1.30E-03	4.40E-12	3.39E-09
PHENANTHRENE	1.62E+00	2.43E-08	ND	2.97E-11	0.00E+00
PYRENE	1.90E+00	2.85E-08	ND	3.49E-11	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	3.65E-01	5.48E-09	ND	6.70E-12	0.00E+00
DIBENZOFURAN	2.46E-01	3.69E-09	ND	4.51E-12	0.00E+00
DI-N-BUTYLPHTHALATE	2.80E-01	4.20E-09	ND	5.14E-12	0.00E+00
2-METHYLNAPHTHALENE	2.51E-01	3.77E-09	ND	4.60E-12	0.00E+00
ACETONE	6.60E-02	9.90E-10	ND	1.21E-12	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	1.10E-02	1.65E-10	ND	2.02E-13	0.00E+00
TOLUENE	5.60E-03	8.40E-11	ND	1.03E-13	0.00E+00
XYLENES(TOTAL)	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	9.91E+01	1.49E-06	ND	1.82E-09	0.00E+00
BARIUM	2.56E+02	3.84E-06	ND	4.70E-09	0.00E+00
CERYLLIUM	3.63E+00	5.45E-08	ND	6.66E-11	0.00E+00
BORON	5.21E+01	7.82E-07	ND	9.56E-10	0.00E+00
CADMIUM	2.08E+00	3.12E-08	ND	3.82E-11	0.00E+00
CALCIUM	1.14E+05	1.71E-03	ND	2.09E-06	0.00E+00
COBALT	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COPER	2.55E+02	3.83E-06	ND	4.68E-09	0.00E+00
LEAD	1.27E+02	1.91E-06	ND	2.33E-09	0.00E+00
MAGNESIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	1.67E+03	2.51E-05	4.00E-03	3.06E-08	7.66E-06
NICKEL	5.23E+01	7.85E-07	ND	9.60E-10	0.00E+00
URANIUM	9.83E+01	1.47E-06	ND	1.80E-09	0.00E+00
VANADIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ZINC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
				HAZARD INDEX =	7.66E-06

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = Mean chemical concentration in air (mg/m3)
IR = Inhalation rate (m3/hr)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
ET = Exposure time (hr/d)
BW = Body weight (kg)
AF = Averaging frequency (365d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-4
RISK FROM SURFACE SOIL PARTICULATE INHALATION AT LINDE
RECEPTOR: CURRENT AND FUTURE EMPLOYEE-ADULT
USING RME VALUES

CONTAMINANTS OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	7.33E+00	2.20E-07	ND	7.68E-10	0.00E+00
BENZO(A)PYRENE	3.00E+00	9.00E-08	6.10E+00	3.15E-10	1.92E-09
BENZO(K)FLOURANTHENE	3.10E+00	9.30E-08	ND	3.25E-10	0.00E+00
CHRYSENE	3.90E+00	1.17E-07	ND	4.09E-10	0.00E+00
DIBENZO(A,H)ANTHRACENE	4.20E-01	1.26E-08	ND	4.40E-11	0.00E+00
INDENO(1,2,3-CD)PYRENE	2.10E+00	6.30E-08	ND	2.20E-10	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	6.04E-01	1.81E-08	ND	6.33E-11	0.00E+00
METHYLENE CHLORIDE	1.10E-02	3.30E-10	ND	1.15E-12	0.00E+00
ARSENIC	1.27E+02	3.81E-06	5.00E+01	1.33E-08	6.66E-07
BERYLLIUM	4.62E+00	1.39E-07	8.40E+00	4.84E-10	4.07E-09
CADMIUM	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
				TOTAL CANCER RISK =	6.72E-07

NON-CARCINOGENS	SOIL CONCENTRATION mg/kg	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPHTHENE	4.95E-01	1.49E-08	ND	1.21E-10	0.00E+00
ANTHRACENE	8.70E-01	2.61E-08	ND	2.13E-10	0.00E+00
BENZO(B)FLUORANTHENE	3.20E+00	9.60E-08	ND	7.83E-10	0.00E+00
BENZO(G,H,I)PERYLENE	2.20E+00	6.60E-08	ND	5.38E-10	0.00E+00
FLUORANTHENE	7.00E+00	2.10E-07	ND	1.71E-09	0.00E+00
FLUORENE	5.76E-01	1.73E-08	ND	1.41E-10	0.00E+00
NAPHTHALENE	7.50E-01	2.25E-08	1.30E-03	1.83E-10	1.41E-07
PHENANTHRENE	4.70E+00	1.41E-07	ND	1.15E-09	0.00E+00
PYRENE	6.20E+00	1.86E-07	ND	1.52E-09	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	6.04E-01	1.81E-08	ND	1.48E-10	0.00E+00
DIBENZOFURAN	5.83E-01	1.75E-08	ND	1.43E-10	0.00E+00
DI-N-BUTYLPHTHALATE	2.80E-01	8.40E-09	ND	6.85E-11	0.00E+00
2-METHYLNAPHTHALENE	6.26E-01	1.88E-08	ND	1.53E-10	0.00E+00
ACETONE	6.60E-02	1.98E-09	ND	1.61E-11	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	1.10E-02	3.30E-10	ND	2.69E-12	0.00E+00
TOLUENE	5.60E-03	1.68E-10	ND	1.37E-12	0.00E+00
XYLENES(TOTAL)	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	1.27E+02	3.81E-06	ND	3.11E-08	0.00E+00
BARIUM	3.13E+02	9.39E-06	ND	7.66E-08	0.00E+00
CERYLLIUM	4.62E+00	1.39E-07	ND	1.13E-09	0.00E+00
BORON	6.27E+01	1.88E-06	ND	1.53E-08	0.00E+00
CADMIUM	2.70E+00	8.10E-08	ND	6.60E-10	0.00E+00
CALCIUM	1.40E+05	4.20E-03	ND	3.42E-05	0.00E+00
COBALT	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COPER	1.08E+03	3.24E-05	ND	2.64E-07	0.00E+00
LEAD	3.11E+02	9.33E-06	ND	7.61E-08	0.00E+00
MAGNESIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	2.05E+03	6.15E-05	4.00E-03	5.01E-07	1.25E-04
NICKEL	1.53E+02	4.59E-06	ND	3.74E-08	0.00E+00
URANIUM	1.38E+02	4.14E-06	ND	3.38E-08	0.00E+00
VANADIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ZINC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
				HAZARD INDEX =	1.26E-04

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = UL95 chemical concentration in soil (mg/m3)
IR = Inhalation rate (0.83 m3/hr)
EF = Exposure frequency (250 d/yr)
ED = Exposure duration (30 yr)
ET = Exposure time (1.0 hr/d)
BW = Body weight (70 kg)
AF = Averaging frequency (365d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-5
RISK FROM SOIL INGESTION AT ASHLAND 1
RECEPTOR: CURRENT TRANSIENT
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	2.20E-01	7.30E+00	3.69E-09	2.69E-08
BENZO(A)PYRENE	2.64E-01	7.30E+00	4.43E-09	3.23E-08
BENZO(K)FLOURANTHENE	2.70E-01	7.30E+00	4.53E-09	3.31E-08
BIS(2-ETHYLHEXYL)PHTHALATE	3.38E-01	7.00E+00	5.67E-09	3.97E-08
CHRYSENE	2.81E-01	7.30E+00	4.71E-09	3.44E-08
DIBENZO(A,H)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	7.50E-03	8.05E-11	6.04E-13
ARSENIC	0.00E+00	1.75E+00	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	4.30E+00	0.00E+00	0.00E+00
CADMIUM	0.00E+00	ND	0.00E+00	0.00E+00
			TOTAL CANCER RISK =	1.66E-07

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	6.00E-02	0.00E+00	0.00E+00
ACETONE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
ANTHRACENE	0.00E+00	3.00E-01	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	2.88E-01	ND	5.64E-08	0.00E+00
BENZO(G,H,I)PERYLENE	2.98E-01	ND	5.83E-08	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	3.38E-01	2.00E-02	6.61E-08	3.31E-06
CHLOROBENZENE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
DIBENZOFURAN	0.00E+00	4.00E-03	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.66E-01	1.00E-01	3.25E-08	3.25E-07
FLUORANTHENE	3.45E-01	4.00E-02	6.75E-08	1.69E-06
FLUORENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	6.00E-02	9.39E-10	1.57E-08
2-METHYL NAPTHALENE	1.20E-01	ND	2.35E-08	0.00E+00
NAPHTHALENE	1.20E-01	4.00E-02	2.35E-08	5.87E-07
PHENANTHRENE	2.53E-01	ND	4.95E-08	0.00E+00
PYRENE	3.09E-01	3.00E-02	6.05E-08	2.02E-06
TOLUENE	6.99E-03	2.00E-01	1.37E-09	6.84E-09
XYLENE (TOTAL)	0.00E+00	2.00E+00	0.00E+00	0.00E+00
ARSENIC	0.00E+00	3.00E-04	0.00E+00	0.00E+00
BARIUM	0.00E+00	7.00E-02	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	5.00E-03	0.00E+00	0.00E+00
BORON	0.00E+00	9.00E-02	0.00E+00	0.00E+00
CADMIUM	0.00E+00	5.00E-04	0.00E+00	0.00E+00
CALCIUM	6.61E+04	ND	1.29E-02	0.00E+00
COBALT	0.00E+00	9.60E-01	0.00E+00	0.00E+00
COPPER	1.03E+02	4.00E-02	2.02E-05	5.04E-04
LEAD	2.15E+03	ND	4.21E-04	0.00E+00
MAGNESIUM	2.22E+04	ND	4.34E-03	0.00E+00
MANGANESE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
NICKEL	0.00E+00	2.00E-02	0.00E+00	0.00E+00
VANADIUM	8.02E+01	7.00E-03	1.57E-05	2.24E-03
URANIUM	3.26E+01	3.00E-03	6.38E-06	2.13E-03
ZINC	2.29E+01	ND	4.48E-06	0.00E+00
			HAZARD INDEX =	4.88E-03

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = Mean chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (50mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (100d/yr)
ED = Exposure duration (6yr)
BW = Body weight (70kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-6
RISK FROM SOIL INGESTION AT ASHLAND 1
RECEPTOR: CURRENT TRANSIENT
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	5.34E-01	7.30E+00	7.17E-08	5.23E-07
BENZO(A)PYRENE	4.42E-01	7.30E+00	5.93E-08	4.33E-07
BENZO(K)FLOURANTHENE	4.87E-01	7.30E+00	6.54E-08	4.77E-07
BIS(2-ETHYLHEXYL)PHTHALATE	6.40E-01	7.00E+00	8.59E-08	6.01E-07
CHRYSENE	7.02E-01	7.30E+00	9.42E-08	6.88E-07
DIBENZO(A,H)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	7.50E-03	6.44E-10	4.83E-12
ARSENIC	0.00E+00	1.75E+00	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	4.30E+00	0.00E+00	0.00E+00
CADMIUM	0.00E+00	ND	0.00E+00	0.00E+00
			TOTAL CANCER RISK =	2.72E-06

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	6.00E-02	0.00E+00	0.00E+00
ACETONE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
ANTHRACENE	0.00E+00	3.00E-01	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	5.12E-01	ND	2.00E-07	0.00E+00
BENZO(G,H,I)PERYLENE	4.96E-01	ND	1.94E-07	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	6.40E-01	2.00E-02	2.50E-07	1.25E-05
CHLOROBENZENE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
DIBENZOFURAN	0.00E+00	4.00E-03	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	2.09E-01	1.00E-01	8.18E-08	8.18E-07
FLUORANTHENE	8.05E-01	4.00E-02	3.15E-07	7.88E-06
FLUORENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	6.00E-02	1.88E-09	3.13E-08
2-METHYL NAPTHALENE	1.20E-01	ND	4.70E-08	0.00E+00
NAPHTHALENE	1.20E-01	4.00E-02	4.70E-08	1.17E-06
PHENANTHRENE	5.82E-01	ND	2.28E-07	0.00E+00
PYRENE	9.63E-01	3.00E-02	3.77E-07	1.26E-05
TOLUENE	2.44E-02	2.00E-01	9.55E-09	4.77E-08
XYLENE (TOTAL)	0.00E+00	2.00E+00	0.00E+00	0.00E+00
ARSENIC	0.00E+00	3.00E-04	0.00E+00	0.00E+00
BARIUM	0.00E+00	7.00E-02	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	5.00E-03	0.00E+00	0.00E+00
BORON	0.00E+00	9.00E-02	0.00E+00	0.00E+00
CADMIUM	0.00E+00	5.00E-04	0.00E+00	0.00E+00
CALCIUM	9.45E+04	ND	3.70E-02	0.00E+00
COBALT	0.00E+00	9.60E-01	0.00E+00	0.00E+00
COPPER	4.47E+02	4.00E-02	1.75E-04	4.37E-03
LEAD	2.11E+04	ND	8.26E-03	0.00E+00
MAGNESIUM	4.28E+04	ND	1.68E-02	0.00E+00
MANGANESE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
NICKEL	0.00E+00	2.00E-02	0.00E+00	0.00E+00
VANADIUM	2.74E+02	7.00E-03	1.07E-04	1.53E-02
URANIUM	4.31E+01	3.00E-03	1.69E-05	5.62E-03
ZINC	1.06E+03	ND	4.15E-04	0.00E+00
			HAZARD INDEX =	2.54E-02

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = UL95 chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (100mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (100d/yr)
ED = Exposure duration (24yr)
BW = Body weight (70kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-7
RISK FROM SOIL PARTICULATE INHALATION AT ASHLAND 1
RECEPTOR: CURRENT TRANSIENT
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	2.20E-01	3.30E-09	ND	2.31E-13	0.00E+00
BENZO(A)PYRENE	2.64E-01	3.96E-09	6.10E+00	2.76E-13	1.68E-12
BENZO(K)FLOURANTHENE	2.70E-01	4.05E-09	ND	2.82E-13	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	3.38E-01	5.07E-09	ND	3.53E-13	0.00E+00
CHRYSENE	2.81E-01	4.22E-09	ND	2.93E-13	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	7.20E-11	ND	5.01E-15	0.00E+00
ARSENIC	0.00E+00	0.00E+00	5.00E+01	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00	8.40E+00	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
				TOTAL CANCER RISK =	1.68E-12

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ACETONE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	2.88E-01	4.32E-09	ND	3.51E-12	0.00E+00
BENZO(G,H,I)PERYLENE	2.98E-01	4.47E-09	ND	3.63E-12	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	3.38E-01	5.07E-09	ND	4.12E-12	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DIBENZOFURAN	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.66E-01	2.49E-09	ND	2.02E-12	0.00E+00
FLUORANTHENE	3.45E-01	5.18E-09	ND	4.20E-12	0.00E+00
FLUORENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	7.20E-11	ND	5.85E-14	0.00E+00
2-METHYL NAPTHALENE	1.20E-01	1.80E-09	ND	1.46E-12	0.00E+00
NAPHTHALENE	1.20E-01	1.80E-09	1.30E-03	1.46E-12	1.12E-09
PHENANTHRENE	2.53E-01	3.80E-09	ND	3.08E-12	0.00E+00
PYRENE	3.09E-01	4.64E-09	ND	3.76E-12	0.00E+00
TOLUENE	6.99E-03	1.05E-10	ND	8.52E-14	0.00E+00
XYLENE (TOTAL)	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BARIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BORON	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CALCIUM	6.61E+04	9.92E-04	ND	8.05E-07	0.00E+00
COBALT	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COPPER	1.03E+02	1.55E-06	ND	1.25E-09	0.00E+00
LEAD	2.15E+03	3.23E-05	ND	2.62E-08	0.00E+00
MAGNESIUM	2.22E+04	3.33E-04	ND	2.70E-07	0.00E+00
MANGANESE	0.00E+00	0.00E+00	4.00E-03	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
VANADIUM	8.02E+01	1.20E-06	ND	9.77E-10	0.00E+00
URANIUM	3.26E+01	4.89E-07	ND	3.97E-10	0.00E+00
ZINC	2.29E+01	3.44E-07	ND	2.79E-10	0.00E+00
				HAZARD INDEX =	1.12E-09

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = mean chemical concentration in air (mg/m3)
IR = Inhalation rate (m3/hr)
ET = Exposure time (hr/day)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-8
RISK FROM SOIL INHALATION AT ASHLAND 1
RECEPTOR: CURRENT TRANSIENT
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	5.34E-01	1.60E-08	ND	5.37E-11	0.00E+00
BENZO(A)PYRENE	4.42E-01	1.33E-08	6.10E+00	4.45E-11	2.71E-10
BENZO(K)FLOURANTHENE	4.87E-01	1.46E-08	ND	4.90E-11	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	6.40E-01	1.92E-08	ND	6.44E-11	0.00E+00
CHRYSENE	7.02E-01	2.11E-08	ND	7.07E-11	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	1.44E-10	ND	4.83E-13	0.00E+00
ARSENIC	0.00E+00	0.00E+00	5.00E+01	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00	8.40E+00	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
				TOTAL CANCER RISK =	2.71E-10

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ACETONE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	5.12E-01	1.54E-08	ND	1.50E-10	0.00E+00
BENZO(G,H,I)PERYLENE	4.96E-01	1.49E-08	ND	1.46E-10	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	6.40E-01	1.92E-08	ND	1.88E-10	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DIBENZOFURAN	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	2.09E-01	6.27E-09	ND	6.14E-11	0.00E+00
FLUORANTHENE	8.05E-01	2.42E-08	ND	2.36E-10	0.00E+00
FLUORENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	1.44E-10	ND	1.41E-12	0.00E+00
2-METHYL NAPTHALENE	1.20E-01	3.60E-09	ND	3.52E-11	0.00E+00
NAPHTHALENE	1.20E-01	3.60E-09	1.30E-03	3.52E-11	2.71E-08
PHENANTHRENE	5.82E-01	1.75E-08	ND	1.71E-10	0.00E+00
PYRENE	9.63E-01	2.89E-08	ND	2.83E-10	0.00E+00
TOLUENE	2.44E-02	7.32E-10	ND	7.16E-12	0.00E+00
XYLENE (TOTAL)	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BARIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BORON	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CALCIUM	9.45E+04	2.84E-03	ND	2.77E-05	0.00E+00
COBALT	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COPPER	4.47E+02	1.34E-05	ND	1.31E-07	0.00E+00
LEAD	2.11E+04	6.33E-04	ND	6.19E-06	0.00E+00
MAGNESIUM	4.28E+04	1.28E-03	ND	1.26E-05	0.00E+00
MANGANESE	0.00E+00	0.00E+00	4.00E-03	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
VANADIUM	2.74E+02	8.22E-06	ND	8.04E-08	0.00E+00
URANIUM	4.31E+01	1.29E-06	ND	1.26E-08	0.00E+00
ZINC	1.06E+03	3.18E-05	ND	3.11E-07	0.00E+00
				HAZARD INDEX =	2.71E-08

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = UL95 chemical concentration in air (mg/m3)
IR = Inhalation rate (m3/hr)
ET = Exposure time (hr/day)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-9
RISK FROM SOIL INGESTION AT ASHLAND 1
RECEPTOR FUTURE EMPLOYEE
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	2.20E-01	7.30E+00	6.46E-09	4.71E-08
BENZO(A)PYRENE	2.64E-01	7.30E+00	7.75E-09	5.66E-08
BENZO(K)FLOURANTHENE	2.70E-01	7.30E+00	7.93E-09	5.79E-08
BIS(2-ETHYLHEXYL)PHTHALATE	3.38E-01	7.00E+00	9.92E-09	6.95E-08
CHRYSENE	2.81E-01	7.30E+00	8.25E-09	6.02E-08
DIBENZO(A,H)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	7.50E-03	1.41E-10	1.06E-12
ARSENIC	0.00E+00	1.75E+00	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	4.30E+00	0.00E+00	0.00E+00
CADMIUM	0.00E+00	ND	0.00E+00	0.00E+00
			TOTAL CANCER RISK =	2.91E-07

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	6.00E-02	0.00E+00	0.00E+00
ACETONE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
ANTHRACENE	0.00E+00	3.00E-01	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	2.88E-01	ND	8.45E-08	0.00E+00
BENZO(G,H,I)PERYLENE	2.98E-01	ND	8.75E-08	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	3.38E-01	2.00E-02	9.92E-08	4.96E-06
CHLOROBENZENE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
DIBENZOFURAN	0.00E+00	4.00E-03	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.66E-01	1.00E-01	4.87E-08	4.87E-07
FLUORANTHENE	3.45E-01	4.00E-02	1.01E-07	2.53E-06
FLUORENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	6.00E-02	1.41E-09	2.35E-08
2-METHYL NAPTHALENE	1.20E-01	ND	3.52E-08	0.00E+00
NAPHTHALENE	1.20E-01	4.00E-02	3.52E-08	8.81E-07
PHENANTHRENE	2.53E-01	ND	7.43E-08	0.00E+00
PYRENE	3.09E-01	3.00E-02	9.07E-08	3.02E-06
TOLUENE	6.99E-03	2.00E-01	2.05E-09	1.03E-08
XYLENE (TOTAL)	0.00E+00	2.00E+00	0.00E+00	0.00E+00
ARSENIC	0.00E+00	3.00E-04	0.00E+00	0.00E+00
BARIUM	0.00E+00	7.00E-02	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	5.00E-03	0.00E+00	0.00E+00
BORON	0.00E+00	9.00E-02	0.00E+00	0.00E+00
CADMIUM	0.00E+00	5.00E-04	0.00E+00	0.00E+00
CALCIUM	6.61E+04	ND	1.94E-02	0.00E+00
COBALT	0.00E+00	9.30E-01	0.00E+00	0.00E+00
COPPER	1.03E+02	4.00E-02	3.02E-05	7.56E-04
LEAD	2.15E+03	ND	6.31E-04	0.00E+00
MAGNESIUM	2.22E+04	ND	6.52E-03	0.00E+00
MANGANESE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
NICKEL	0.00E+00	2.00E-02	0.00E+00	0.00E+00
VANADIUM	8.02E+01	7.00E-03	2.35E-05	3.36E-03
URANIUM	3.26E+01	3.00E-03	9.56E-06	3.19E-03
ZINC	2.29E+01	ND	6.72E-06	0.00E+00
			HAZARD INDEX =	7.32E-03

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = Mean chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (30mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (250d/yr)
ED = Exposure duration (7yr)
BW = Body weight (70 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-10
RISK FROM SOIL INGESTION AT ASHLAND 1
RECEPTOR: FUTURE EMPLOYEE
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	5.34E-01	7.30E+00	9.33E-08	6.81E-07
BENZO(A)PYRENE	4.42E-01	7.30E+00	7.72E-08	5.64E-07
BENZO(K)FLOURANTHENE	4.87E-01	7.30E+00	8.51E-08	6.21E-07
BIS(2-ETHYLHEXYL)PHTHALATE	6.40E-01	7.00E+00	1.12E-07	7.83E-07
CHRYSENE	7.02E-01	7.30E+00	1.23E-07	8.95E-07
DIBENZO(A,H)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	7.50E-03	8.39E-10	6.29E-12
ARSENIC	0.00E+00	1.75E+00	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	4.30E+00	0.00E+00	0.00E+00
CADMIUM	0.00E+00	ND	0.00E+00	0.00E+00
			TOTAL CANCER RISK =	3.54E-06

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	6.00E-02	0.00E+00	0.00E+00
ACETONE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
ANTHRACENE	0.00E+00	3.00E-01	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	5.12E-01	ND	2.50E-07	0.00E+00
BENZO(G,H,I)PERYLENE	4.96E-01	ND	2.43E-07	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	6.40E-01	2.00E-02	3.13E-07	1.57E-05
CHLOROBENZENE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
DIBENZOFURAN	0.00E+00	4.00E-03	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	2.09E-01	1.00E-01	1.02E-07	1.02E-06
FLUORANTHENE	8.05E-01	4.00E-02	3.94E-07	9.85E-06
FLUORENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	6.00E-02	2.35E-09	3.91E-08
2-METHYL NAPTHALENE	1.20E-01	ND	5.87E-08	0.00E+00
NAPHTHALENE	1.20E-01	4.00E-02	5.87E-08	1.47E-06
PHENANTHRENE	5.82E-01	ND	2.85E-07	0.00E+00
PYRENE	9.63E-01	3.00E-02	4.71E-07	1.57E-05
TOLUENE	2.44E-02	2.00E-01	1.19E-08	5.97E-08
XYLENE (TOTAL)	0.00E+00	2.00E+00	0.00E+00	0.00E+00
ARSENIC	0.00E+00	3.00E-04	0.00E+00	0.00E+00
BARIUM	0.00E+00	7.00E-02	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	5.00E-03	0.00E+00	0.00E+00
BORON	0.00E+00	9.00E-02	0.00E+00	0.00E+00
CADMIUM	0.00E+00	5.00E-04	0.00E+00	0.00E+00
CALCIUM	9.45E+04	ND	4.62E-02	0.00E+00
COBALT	0.00E+00	9.30E-01	0.00E+00	0.00E+00
COPPER	4.47E+02	4.00E-02	2.19E-04	5.47E-03
LEAD	2.11E+04	ND	1.03E-02	0.00E+00
MAGNESIUM	4.28E+04	ND	2.09E-02	0.00E+00
MANGANESE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
NICKEL	0.00E+00	2.00E-02	0.00E+00	0.00E+00
VANADIUM	2.74E+02	7.00E-03	1.34E-04	1.92E-02
URANIUM	4.31E+01	3.00E-03	2.11E-05	7.03E-03
ZINC	1.06E+03	ND	5.19E-04	0.00E+00
			HAZARD INDEX =	3.17E-02

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = UL95 chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (50 mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (250 d/yr)
ED = Exposure duration (25 yr)
BW = Body weight (70 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-11
RISK FROM SOIL PARTICULATE INHALATION AT ASHLAND 1
RECEPTOR: FUTURE EMPLOYEE
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	2.20E-01	3.30E-09	ND	1.42E-11	0.00E+00
BENZO(A)PYRENE	2.64E-01	3.96E-09	6.10E+00	1.71E-11	1.04E-10
BENZO(K)FLOURANTHENE	2.70E-01	4.05E-09	ND	1.75E-11	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	3.38E-01	5.07E-09	ND	2.19E-11	0.00E+00
CHRYSENE	2.81E-01	4.22E-09	ND	1.82E-11	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	7.20E-11	ND	3.11E-13	0.00E+00
ARSENIC	0.00E+00	0.00E+00	5.00E+01	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00	8.40E+00	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
				TOTAL CANCER RISK =	1.04E-10

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ACETONE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	2.88E-01	4.32E-09	ND	1.86E-10	0.00E+00
BENZO(G,H,I)PERYLENE	2.98E-01	4.47E-09	ND	1.93E-10	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	3.38E-01	5.07E-09	ND	2.19E-10	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DIBENZOFURAN	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.66E-01	2.49E-09	ND	1.07E-10	0.00E+00
FLUORANTHENE	3.45E-01	5.18E-09	ND	2.23E-10	0.00E+00
FLUORENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	7.20E-11	ND	3.11E-12	0.00E+00
2-METHYL NAPTHALENE	1.20E-01	1.80E-09	ND	7.77E-11	0.00E+00
NAPHTHALENE	1.20E-01	1.80E-09	1.30E-03	7.77E-11	5.97E-08
PHENANTHRENE	2.53E-01	3.80E-09	ND	1.64E-10	0.00E+00
PYRENE	3.09E-01	4.64E-09	ND	2.00E-10	0.00E+00
TOLUENE	6.99E-03	1.05E-10	ND	4.52E-12	0.00E+00
XYLENE (TOTAL)	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BARIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BORON	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CALCIUM	6.61E+04	9.92E-04	ND	4.28E-05	0.00E+00
COBALT	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COPPER	1.03E+02	1.55E-06	ND	6.67E-08	0.00E+00
LEAD	2.15E+03	3.23E-05	ND	1.39E-06	0.00E+00
MAGNESIUM	2.22E+04	3.33E-04	ND	1.44E-05	0.00E+00
MANGANESE	0.00E+00	0.00E+00	4.00E-03	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
VANADIUM	8.02E+01	1.20E-06	ND	5.19E-08	0.00E+00
URANIUM	3.26E+01	4.89E-07	ND	2.11E-08	0.00E+00
ZINC	2.29E+01	3.44E-07	ND	1.48E-08	0.00E+00
				HAZARD INDEX =	5.97E-08

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = mean chemical concentration in air (mg/m3)
IR = Inhalation rate
ET = Exposure time (hr/day)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-12
RISK FROM SOIL INHALATION AT ASHLAND 1
RECEPTOR: FUTURE EMPLOYEE
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	5.34E-01	1.60E-08	ND	3.25E-10	0.00E+00
BENZO(A)PYRENE	4.42E-01	1.33E-08	6.10E+00	2.69E-10	1.64E-09
BENZO(K)FLOURANTHENE	4.87E-01	1.46E-08	ND	2.97E-10	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALAT	6.40E-01	1.92E-08	ND	3.90E-10	0.00E+00
CHRYSENE	7.02E-01	2.11E-08	ND	4.28E-10	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	1.44E-10	ND	2.92E-12	0.00E+00
ARSENIC	0.00E+00	0.00E+00	5.00E+01	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00	8.40E+00	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
				TOTAL CANCER RISK =	1.64E-09

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ACETONE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	5.12E-01	1.54E-08	ND	8.73E-10	0.00E+00
BENZO(G,H,I)PERYLENE	4.96E-01	1.49E-08	ND	8.46E-10	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALAT	6.40E-01	1.92E-08	ND	1.09E-09	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DIBENZOFURAN	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	2.09E-01	6.27E-09	ND	3.56E-10	0.00E+00
FLUORANTHENE	8.05E-01	2.42E-08	ND	1.37E-09	0.00E+00
FLUORENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.80E-03	1.44E-10	ND	8.19E-12	0.00E+00
2-METHYL NAPTHALENE	1.20E-01	3.60E-09	ND	2.05E-10	0.00E+00
NAPHTHALENE	1.20E-01	3.60E-09	1.30E-03	2.05E-10	1.57E-07
PHENANTHRENE	5.82E-01	1.75E-08	ND	9.93E-10	0.00E+00
PYRENE	9.63E-01	2.89E-08	ND	1.64E-09	0.00E+00
TOLUENE	2.44E-02	7.32E-10	ND	4.16E-11	0.00E+00
XYLENE (TOTAL)	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BARIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BORON	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CALCIUM	9.45E+04	2.84E-03	ND	1.61E-04	0.00E+00
COBALT	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COPPER	4.47E+02	1.34E-05	ND	7.62E-07	0.00E+00
LEAD	2.11E+04	6.33E-04	ND	3.60E-05	0.00E+00
MAGNESIUM	4.28E+04	1.28E-03	ND	7.30E-05	0.00E+00
MANGANESE	0.00E+00	0.00E+00	4.00E-03	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
VANADIUM	2.74E+02	8.22E-06	ND	4.67E-07	0.00E+00
URANIUM	4.31E+01	1.29E-06	ND	7.35E-08	0.00E+00
ZINC	1.06E+03	3.18E-05	ND	1.81E-06	0.00E+00
				HAZARD INDEX =	1.57E-07

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = UL95 chemical concentration in air (mg/m3)
IR = Inhalation rate
ET = Exposure time (hr/day)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-13
RISK FROM SOIL INGESTION AT ASHLAND 2
RECEPTOR: CURRENT TRANSIENT (OLDER CHILD)
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BENZO(A)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BENZO(K)FLOURANTHENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	7.00E+00	0.00E+00	0.00E+00
CHRYSENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	7.50E-03	0.00E+00	0.00E+00
ARSENIC	0.00E+00	1.75E+00	0.00E+00	0.00E+00
BERYLLIUM	3.06E+00	4.30E+00	5.13E-08	2.21E-07
CADMIUM	9.07E+00	ND	1.52E-07	0.00E+00
			TOTAL CANCER RISK =	2.21E-07

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	6.00E-02	0.00E+00	0.00E+00
ACETONE	1.70E-01	1.00E-01	3.33E-08	3.33E-07
ANTHRACENE	0.00E+00	3.00E-01	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(G,H,I)PERYLENE	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
CHLOROBENZENE	1.70E-02	2.00E-02	3.33E-09	1.66E-07
DIBENZOFURAN	0.00E+00	4.00E-03	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.00E-01	1.00E-01	1.96E-08	1.96E-07
FLUORANTHENE	9.47E-01	4.00E-02	1.85E-07	4.63E-06
FLUORENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	2.40E-02	6.00E-02	4.70E-09	7.83E-08
2-METHYL NAPTHALENE	0.00E+00	ND	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
PHENANTHRENE	8.67E-01	ND	1.70E-07	0.00E+00
PYRENE	8.85E-01	3.00E-02	1.73E-07	5.77E-06
TOLUENE	0.00E+00	2.00E-01	0.00E+00	0.00E+00
XYLENE (TOTAL)	4.40E-02	2.00E+00	8.61E-09	4.31E-09
ARSENIC	0.00E+00	3.00E-04	0.00E+00	0.00E+00
BARIUM	0.00E+00	7.00E-02	0.00E+00	0.00E+00
BERYLLIUM	3.06E+00	5.00E-03	5.99E-07	1.20E-04
BORON	0.00E+00	9.00E-02	0.00E+00	0.00E+00
CADMIUM	9.07E+00	5.00E-04	1.77E-06	3.55E-03
CALCIUM	0.00E+00	ND	0.00E+00	0.00E+00
COBALT	2.16E+01	9.30E-01	4.23E-06	4.55E-06
COPPER	3.87E+02	4.00E-02	7.57E-05	1.89E-03
LEAD	1.23E+02	ND	2.41E-05	0.00E+00
MAGNESIUM	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
NICKEL	4.69E+01	2.00E-02	9.18E-06	4.59E-04
VANADIUM	1.81E+02	7.00E-03	3.54E-05	5.06E-03
URANIUM	2.23E+01	3.00E-03	4.37E-06	1.46E-03
ZINC	3.63E+02	ND	7.10E-05	0.00E+00
			HAZARD INDEX =	1.26E-02

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = Mean chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (50 mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (50 d/yr)
ED = Exposure duration (6 yr)
BW = Body weight (35 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-14
RISK FROM SOIL INGESTION AT ASHLAND 2
RECEPTOR: CURRENT TRANSIENT (OLDER CHILD)
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BENZO(A)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BENZO(K)FLOURANTHENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	7.00E+00	0.00E+00	0.00E+00
CHRYSENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	7.50E-03	0.00E+00	0.00E+00
ARSENIC	0.00E+00	1.75E+00	0.00E+00	0.00E+00
BERYLLIUM	4.76E+00	4.30E+00	4.15E-07	1.79E-06
CADMIUM	3.43E+01	ND	2.99E-06	0.00E+00
			TOTAL CANCER RISK =	1.79E-06

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	6.00E-02	0.00E+00	0.00E+00
ACETONE	1.70E-01	1.00E-01	1.73E-07	1.73E-06
ANTHRACENE	0.00E+00	3.00E-01	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(G,H,I)PERYLENE	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
CHLOROBENZENE	1.70E-02	2.00E-02	1.73E-08	8.65E-07
DIBENZOFURAN	0.00E+00	4.00E-03	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.00E-01	1.00E-01	1.02E-07	1.02E-06
FLUORANTHENE	4.90E+00	4.00E-02	4.99E-06	1.25E-04
FLUORENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	2.40E-02	6.00E-02	2.44E-08	4.07E-07
2-METHYL NAPTHALENE	0.00E+00	ND	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
PHENANTHRENE	4.00E+00	ND	4.07E-06	0.00E+00
PYRENE	4.00E+00	3.00E-02	4.07E-06	1.36E-04
TOLUENE	0.00E+00	2.00E-01	0.00E+00	0.00E+00
XYLENE (TOTAL)	4.40E-02	2.00E+00	4.48E-08	2.24E-08
ARSENIC	0.00E+00	3.00E-04	0.00E+00	0.00E+00
BARIUM	0.00E+00	7.00E-02	0.00E+00	0.00E+00
BERYLLIUM	4.76E+00	5.00E-03	4.84E-06	9.69E-04
BORON	0.00E+00	9.00E-02	0.00E+00	0.00E+00
CADMIUM	3.43E+01	5.00E-04	3.49E-05	6.98E-02
CALCIUM	0.00E+00	ND	0.00E+00	0.00E+00
COBALT	6.51E+01	9.30E-01	6.62E-05	7.12E-05
COPPER	1.36E+03	4.00E-02	1.38E-03	3.46E-02
LEAD	2.90E+02	ND	2.95E-04	0.00E+00
MAGNESIUM	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
NICKEL	7.78E+01	2.00E-02	7.92E-05	3.96E-03
VANADIUM	7.48E+02	7.00E-03	7.61E-04	1.09E-01
URANIUM	2.95E+01	3.00E-03	3.00E-05	1.00E-02
ZINC	1.23E+03	ND	1.25E-03	0.00E+00
			HAZARD INDEX =	2.28E-01

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = UL95 chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (100 mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (130 d/yr)
ED = Exposure duration (6 yr)
BW = Body weight (35 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-15
RISK FROM SOIL PARTICULATE INHALATION AT ASHLAND 2
RECEPTOR: CURRENT TRANSIENT (OLDER CHILD)
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(A)PYRENE	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
BENZO(K)FLOURANTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CHRYSENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	5.00E+01	0.00E+00	0.00E+00
BERYLLIUM	3.06E+00	4.59E-08	8.40E+00	5.16E-12	4.33E-11
CADMIUM	9.07E+00	1.36E-07	6.10E+00	1.53E-11	9.33E-11
				TOTAL CANCER RISK =	1.37E-10

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ACETONE	1.70E-01	2.55E-09	ND	3.34E-12	0.00E+00
ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(G,H,I)PERYLENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CHLOROBENZENE	1.70E-02	2.55E-10	ND	3.34E-13	0.00E+00
DIBENZOFURAN	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.00E-01	1.50E-09	ND	1.97E-12	0.00E+00
FLUORANTHENE	9.47E-01	1.42E-08	ND	1.86E-11	0.00E+00
FLUORENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	2.40E-02	3.60E-10	ND	4.72E-13	0.00E+00
2-METHYL NAPTHALENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	0.00E+00	1.30E-03	0.00E+00	0.00E+00
PHENANTHRENE	8.67E-01	1.30E-08	ND	1.71E-11	0.00E+00
PYRENE	8.85E-01	1.33E-08	ND	1.74E-11	0.00E+00
TOLUENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
XYLENE (TOTAL)	4.40E-02	6.60E-10	ND	8.65E-13	0.00E+00
ARSENIC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BARIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BERYLLIUM	3.06E+00	4.59E-08	ND	6.02E-11	0.00E+00
BORON	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CADMIUM	9.07E+00	1.36E-07	ND	1.78E-10	0.00E+00
CALCIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COBALT	2.16E+01	3.24E-07	ND	4.25E-10	0.00E+00
COPPER	3.87E+02	5.81E-06	ND	7.61E-09	0.00E+00
LEAD	1.23E+02	1.85E-06	ND	2.42E-09	0.00E+00
MAGNESIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	0.00E+00	0.00E+00	4.00E-03	0.00E+00	0.00E+00
NICKEL	4.69E+01	7.04E-07	ND	9.22E-10	0.00E+00
VANADIUM	1.81E+02	2.72E-06	ND	3.56E-09	0.00E+00
URANIUM	2.23E+01	3.35E-07	ND	4.39E-10	0.00E+00
ZINC	3.63E+02	5.45E-06	ND	7.14E-09	0.00E+00
				HAZARD INDEX =	0.00E+00

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = mean chemical concentration in air (mg/m3)
IR = Inhalation rate
ET = Exposure time (hr/day)
EF = Exposure frequency (50 d/yr)
ED = Exposure duration (6 yr)
BW = Body weight (35 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-16
RISK FROM SOIL INHALATION AT ASHLAND 2
RECEPTOR: CURRENT TRANSIENT (OLDER CHILD)
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(A)PYRENE	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
BENZO(K)FLOURANTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CHRYSENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	5.00E+01	0.00E+00	0.00E+00
BERYLLIUM	4.76E+00	1.43E-07	8.40E+00	2.49E-10	2.09E-09
CADMIUM	3.43E+01	1.03E-06	6.10E+00	1.80E-09	1.09E-08
				TOTAL CANCER RISK =	1.30E-08

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ACETONE	1.70E-01	5.10E-09	ND	1.04E-10	0.00E+00
ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(G,H,I)PERYLENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CHLOROBENZENE	1.70E-02	5.10E-10	ND	1.04E-11	0.00E+00
DIBENZOFURAN	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.00E-01	3.00E-09	ND	6.11E-11	0.00E+00
FLUORANTHENE	4.90E+00	1.47E-07	ND	2.99E-09	0.00E+00
FLUORENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	2.40E-02	7.20E-10	ND	1.47E-11	0.00E+00
2-METHYL NAPTHALENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	0.00E+00	1.30E-03	0.00E+00	0.00E+00
PHENANTHRENE	4.00E+00	1.20E-07	ND	2.44E-09	0.00E+00
PYRENE	4.00E+00	1.20E-07	ND	2.44E-09	0.00E+00
TOLUENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
XYLENE (TOTAL)	4.40E-02	1.32E-09	ND	2.69E-11	0.00E+00
ARSENIC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BARIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BERYLLIUM	4.76E+00	1.43E-07	ND	2.91E-09	0.00E+00
BORON	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CADMIUM	3.43E+01	1.03E-06	ND	2.09E-08	0.00E+00
CALCIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COBALT	6.51E+01	1.95E-06	ND	3.97E-08	0.00E+00
COPPER	1.36E+03	4.08E-05	ND	8.30E-07	0.00E+00
LEAD	2.90E+02	8.70E-06	ND	1.77E-07	0.00E+00
MAGNESIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	0.00E+00	0.00E+00	4.00E-03	0.00E+00	0.00E+00
NICKEL	7.78E+01	2.33E-06	ND	4.75E-08	0.00E+00
VANADIUM	7.48E+02	2.24E-05	ND	4.57E-07	0.00E+00
URANIUM	2.95E+01	8.84E-07	ND	1.80E-08	0.00E+00
ZINC	1.23E+03	3.69E-05	ND	7.51E-07	0.00E+00
				HAZARD INDEX =	0.00E+00

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = UL95 chemical concentration in air (mg/m3)
IR = Inhalation rate
ET = Exposure time (hr/day)
EF = Exposure frequency (130 d/yr)
ED = Exposure duration (6 yr)
BW = Body weight (35 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-17
RISK FROM SOIL INGESTION AT ASHLAND 2
RECEPTOR: FUTURE EMPLOYEE
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BENZO(A)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BENZO(K)FLOURANTHENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	7.00E+00	0.00E+00	0.00E+00
CHRYSENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	7.50E-03	0.00E+00	0.00E+00
ARSENIC	0.00E+00	1.75E+00	0.00E+00	0.00E+00
BERYLLIUM	3.06E+00	4.30E+00	8.98E-08	3.86E-07
CADMIUM	9.07E+00	ND	2.66E-07	0.00E+00
			TOTAL CANCER RISK =	3.86E-07

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	6.00E-02	0.00E+00	0.00E+00
ACETONE	1.70E-01	1.00E-01	4.99E-08	4.99E-07
ANTHRACENE	0.00E+00	3.00E-01	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(G,H,I)PERYLENE	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
CHLOROBENZENE	1.70E-02	2.00E-02	4.99E-09	2.50E-07
DIBENZOFURAN	0.00E+00	4.00E-03	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.00E-01	1.00E-01	2.94E-08	2.94E-07
FLUORANTHENE	9.47E-01	4.00E-02	2.78E-07	6.95E-06
FLUORENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	2.40E-02	6.00E-02	7.05E-09	1.17E-07
2-METHYL NAPTHALENE	0.00E+00	ND	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
PHENANTHRENE	8.67E-01	ND	2.55E-07	0.00E+00
PYRENE	8.85E-01	3.00E-02	2.60E-07	8.66E-06
TOLUENE	0.00E+00	2.00E-01	0.00E+00	0.00E+00
XYLENE (TOTAL)	4.40E-02	2.00E+00	1.29E-08	6.46E-09
ARSENIC	0.00E+00	3.00E-04	0.00E+00	0.00E+00
BARIUM	0.00E+00	7.00E-02	0.00E+00	0.00E+00
BERYLLIUM	3.06E+00	5.00E-03	8.98E-07	1.80E-04
BORON	0.00E+00	9.00E-02	0.00E+00	0.00E+00
CADMIUM	9.07E+00	5.00E-04	2.66E-06	5.32E-03
CALCIUM	0.00E+00	ND	0.00E+00	0.00E+00
COBALT	2.16E+01	9.30E-01	6.34E-06	6.82E-06
COPPER	3.87E+02	4.00E-02	1.14E-04	2.84E-03
LEAD	1.23E+02	ND	3.61E-05	0.00E+00
MAGNESIUM	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
NICKEL	4.69E+01	2.00E-02	1.38E-05	6.88E-04
VANADIUM	1.81E+02	7.00E-03	5.31E-05	7.59E-03
URANIUM	2.23E+01	3.00E-03	6.56E-06	2.19E-03
ZINC	3.63E+02	ND	1.07E-04	0.00E+00
			HAZARD INDEX =	1.88E-02

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = Mean chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-18
RISK FROM SOIL INGESTION AT ASHLAND 2
RECEPTOR: FUTURE EMPLOYEE
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BENZO(A)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BENZO(K)FLOURANTHENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	7.00E+00	0.00E+00	0.00E+00
CHRYSENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	7.30E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	7.50E-03	0.00E+00	0.00E+00
ARSENIC	0.00E+00	1.75E+00	0.00E+00	0.00E+00
BERYLLIUM	4.76E+00	4.30E+00	8.32E-07	3.58E-06
CADMIUM	3.43E+01	ND	5.99E-06	0.00E+00
			TOTAL CANCER RISK =	3.58E-06

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	6.00E-02	0.00E+00	0.00E+00
ACETONE	1.70E-01	1.00E-01	8.32E-08	8.32E-07
ANTHRACENE	0.00E+00	3.00E-01	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(G,H,I)PERYLENE	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	2.00E-02	0.00E+00	0.00E+00
CHLOROBENZENE	1.70E-02	2.00E-02	8.32E-09	4.16E-07
DIBENZOFURAN	0.00E+00	4.00E-03	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.00E-01	1.00E-01	4.89E-08	4.89E-07
FLUORANTHENE	4.90E+00	4.00E-02	2.40E-06	5.99E-05
FLUORENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
METHYLENE CHLORIDE	2.40E-02	6.00E-02	1.17E-08	1.96E-07
2-METHYL NAPTHALENE	0.00E+00	ND	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	4.00E-02	0.00E+00	0.00E+00
PHENANTHRENE	4.00E+00	ND	1.96E-06	0.00E+00
PYRENE	4.00E+00	3.00E-02	1.96E-06	6.52E-05
TOLUENE	0.00E+00	2.00E-01	0.00E+00	0.00E+00
XYLENE (TOTAL)	4.40E-02	2.00E+00	2.15E-08	1.08E-08
				0.00E+00
ARSENIC	0.00E+00	3.00E-04	0.00E+00	0.00E+00
BARIUM	0.00E+00	7.00E-02	0.00E+00	0.00E+00
BERYLLIUM	4.76E+00	5.00E-03	2.33E-06	4.66E-04
BORON	0.00E+00	9.00E-02	0.00E+00	0.00E+00
CADMIUM	3.43E+01	5.00E-04	1.68E-05	3.36E-02
CALCIUM	0.00E+00	ND	0.00E+00	0.00E+00
COBALT	6.51E+01	9.30E-01	3.18E-05	3.42E-05
COPPER	1.36E+03	4.00E-02	6.65E-04	1.66E-02
LEAD	2.90E+02	ND	1.42E-04	0.00E+00
MAGNESIUM	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	0.00E+00	1.00E-01	0.00E+00	0.00E+00
NICKEL	7.78E+01	2.00E-02	3.81E-05	1.90E-03
VANADIUM	7.48E+02	7.00E-03	3.66E-04	5.23E-02
URANIUM	2.95E+01	3.00E-03	1.44E-05	4.81E-03
ZINC	1.23E+03	ND	6.02E-04	0.00E+00
			HAZARD INDEX =	1.10E-01

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = UL95 chemical concentration in surface soil (mg/kg)
IR = Ingestion rate (mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-19
RISK FROM SOIL PARTICULATE INHALATION AT ASHLAND 2
RECEPTOR: FUTURE EMPLOYEE
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(A)PYRENE	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
BENZO(K)FLOURANTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CHRYSENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	5.00E+01	0.00E+00	0.00E+00
BERYLLIUM	3.06E+00	4.59E-08	8.40E+00	1.98E-10	1.66E-09
CADMIUM	9.07E+00	1.36E-07	6.10E+00	5.87E-10	3.58E-09
				TOTAL CANCER RISK =	5.24E-09

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ACETONE	1.70E-01	2.55E-09	ND	1.10E-10	0.00E+00
ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(G,H,I)PERYLENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CHLOROBENZENE	1.70E-02	2.55E-10	ND	1.10E-11	0.00E+00
DIBENZOFURAN	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.00E-01	1.50E-09	ND	6.47E-11	0.00E+00
FLUORANTHENE	9.47E-01	1.42E-08	ND	6.13E-10	0.00E+00
FLUORENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	2.40E-02	3.60E-10	ND	1.55E-11	0.00E+00
2-METHYL NAPTHALENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	0.00E+00	1.30E-03	0.00E+00	0.00E+00
PHENANTHRENE	8.67E-01	1.30E-08	ND	5.61E-10	0.00E+00
PYRENE	8.85E-01	1.33E-08	ND	5.73E-10	0.00E+00
TOLUENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
XYLENE (TOTAL)	4.40E-02	6.60E-10	ND	2.85E-11	0.00E+00
ARSENIC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BARIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BERYLLIUM	3.06E+00	4.59E-08	ND	1.98E-09	0.00E+00
BORON	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CADMIUM	9.07E+00	1.36E-07	ND	5.87E-09	0.00E+00
CALCIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COBALT	2.16E+01	3.24E-07	ND	1.40E-08	0.00E+00
COPPER	3.87E+02	5.81E-06	ND	2.50E-07	0.00E+00
LEAD	1.23E+02	1.85E-06	ND	7.96E-08	0.00E+00
MAGNESIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	0.00E+00	0.00E+00	4.00E-03	0.00E+00	0.00E+00
NICKEL	4.69E+01	7.04E-07	ND	3.04E-08	0.00E+00
VANADIUM	1.81E+02	2.72E-06	ND	1.17E-07	0.00E+00
URANIUM	2.23E+01	3.35E-07	ND	1.45E-08	0.00E+00
ZINC	3.63E+02	5.45E-06	ND	2.35E-07	0.00E+00
				HAZARD INDEX =	0.00E+00

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = mean chemical concentration in air (mg/m3)
IR = Inhalation rate
ET = Exposure time (hr/day)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-20
RISK FROM SOIL INHALATION AT ASHLAND 2
RECEPTOR FUTURE EMPLOYEE
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
BENZO(A)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(A)PYRENE	0.00E+00	0.00E+00	6.10E+00	0.00E+00	0.00E+00
BENZO(K)FLOURANTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CHRYSENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DIBENZO(A,H)ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
INDENO(1,2,3-CD)PYRENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	5.00E+01	0.00E+00	0.00E+00
BERYLLIUM	4.76E+00	1.43E-07	8.40E+00	2.90E-09	2.44E-08
CADMIUM	3.43E+01	1.03E-06	6.10E+00	2.09E-08	1.27E-07
				TOTAL CANCER RISK	1.52E-07

NON-CARCINOGENS	SOIL CONCENTRATION (mg/kg)	AIR CONCENTRATION (mg/m3)	INHALATION RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACENAPTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
ACETONE	1.70E-01	5.10E-09	ND	2.90E-10	0.00E+00
ANTHRACENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(B)FLUORANTHENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BENZO(G,H,I)PERYLENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BIS(2-ETHYLHEXYL)PHTHALATE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CHLOROBENZENE	1.70E-02	5.10E-10	ND	2.90E-11	0.00E+00
DIBENZOFURAN	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
DI-N-BUTYLPHTHALATE	1.00E-01	3.00E-09	ND	1.71E-10	0.00E+00
FLUORANTHENE	4.90E+00	1.47E-07	ND	8.36E-09	0.00E+00
FLUORENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
METHYLENE CHLORIDE	2.40E-02	7.20E-10	ND	4.09E-11	0.00E+00
2-METHYL NAPTHALENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	0.00E+00	1.30E-03	0.00E+00	0.00E+00
PHENANTHRENE	4.00E+00	1.20E-07	ND	6.82E-09	0.00E+00
PYRENE	4.00E+00	1.20E-07	ND	6.82E-09	0.00E+00
TOLUENE	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
XYLENE (TOTAL)	4.40E-02	1.32E-09	ND	7.50E-11	0.00E+00
ARSENIC	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BARIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
BERYLLIUM	4.76E+00	1.43E-07	ND	8.12E-09	0.00E+00
BORON	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
CADMIUM	3.43E+01	1.03E-06	ND	5.85E-08	0.00E+00
CALCIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
COBALT	6.51E+01	1.95E-06	ND	1.11E-07	0.00E+00
COPPER	1.36E+03	4.08E-05	ND	2.32E-06	0.00E+00
LEAD	2.90E+02	8.70E-06	ND	4.95E-07	0.00E+00
MAGNESIUM	0.00E+00	0.00E+00	ND	0.00E+00	0.00E+00
MANGANESE	0.00E+00	0.00E+00	4.00E-03	0.00E+00	0.00E+00
NICKEL	7.78E+01	2.33E-06	ND	1.33E-07	0.00E+00
VANADIUM	7.48E+02	2.24E-05	ND	1.28E-06	0.00E+00
URANIUM	2.95E+01	8.84E-07	ND	5.03E-08	0.00E+00
ZINC	1.23E+03	3.69E-05	ND	2.10E-06	0.00E+00
				HAZARD INDEX =	0.00E+00

$$\text{Intake(mg/kg-d)} = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CA = UL95 chemical concentration in air (mg/m3)
IR = Inhalation rate (m3/hr)
ET = Exposure time (hr/day)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-21
RISK FROM SEDIMENT INGESTION AT LOCAL BROOK
RECEPTOR: WADING OLDER CHILD
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SEDIMENT CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
ARSENIC	1.63E+01	1.75E+00	3.83E-08	6.69E-08
BERYLLIUM	9.10E-01	4.30E+00	2.14E-09	9.19E-09
CADMIUM	9.60E-01	ND	2.25E-09	0.00E+00
			TOTAL CANCER RISK =	7.61E-08

NON-CARCINOGENS	SEDIMENT CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ARSENIC	1.63E+01	3.00E-04	4.46E-07	1.49E-03
BARIUM	1.69E+02	7.00E-02	4.63E-06	6.61E-05
BERYLLIUM	9.10E-01	5.00E-03	2.49E-08	4.99E-06
BORON	3.75E+01	9.00E-02	1.03E-06	1.14E-05
CADMIUM	9.60E-01	5.00E-04	2.63E-08	5.26E-05
CALCIUM	3.82E+04	ND	1.05E-03	0.00E+00
COBALT	9.67E+00	9.30E-01	2.65E-07	2.85E-07
COPPER	5.52E+01	4.00E-02	1.51E-06	3.78E-05
LEAD	9.13E+01	ND	2.50E-06	0.00E+00
MAGNESIUM	9.07E+03	ND	2.48E-04	0.00E+00
MANGANESE	8.28E+02	1.00E-01	2.27E-05	2.27E-04
NICKEL	4.13E+01	2.00E-02	1.13E-06	5.66E-05
VANADIUM	5.22E+01	7.00E-03	1.43E-06	2.04E-04
ZINC	3.02E+02	ND	8.26E-06	0.00E+00
			HAZARD INDEX =	2.15E-03

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = Mean chemical concentration in sediment (mg/kg)
IR = Ingestion rate (mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (35 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-22
RISK FROM SEDIMENT INGESTION AT LOCAL CREEK
RECEPTOR: WADING OLDER CHILD
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	SEDIMENT CONCENTRATION (mg/kg)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
ARSENIC	2.07E+01	1.75E+00	9.70E-08	1.70E-07
BERYLLIUM	1.16E+00	4.30E+00	5.45E-09	2.34E-08
CADMIUM	1.31E+00	ND	6.15E-09	0.00E+00
			TOTAL CANCER RISK =	1.93E-07

NON-CARCINOGENS	SEDIMENT CONCENTRATION (mg/kg)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ARSENIC	2.07E+01	3.00E-04	1.13E-06	3.77E-03
BARIUM	2.26E+02	7.00E-02	1.24E-05	1.77E-04
BERYLLIUM	1.16E+00	5.00E-03	6.36E-08	1.27E-05
BORON	4.63E+01	9.00E-02	2.54E-06	2.82E-05
CADMIUM	1.31E+00	5.00E-04	7.18E-08	1.44E-04
CALCIUM	7.17E+04	ND	3.93E-03	0.00E+00
COBALT	1.25E+01	9.30E-01	6.85E-07	7.37E-07
COPPER	9.13E+01	4.00E-02	5.00E-06	1.25E-04
LEAD	1.25E+02	ND	6.86E-06	0.00E+00
MAGNESIUM	1.14E+04	ND	6.25E-04	0.00E+00
MANGANESE	1.06E+03	1.00E-01	5.83E-05	5.83E-04
NICKEL	5.80E+01	2.00E-02	3.18E-06	1.59E-04
VANADIUM	7.34E+01	7.00E-03	4.02E-06	5.74E-04
ZINC	5.01E+02	ND	2.75E-05	0.00E+00
			HAZARD INDEX =	5.58E-03

$$\text{Intake(mg/kg-d)} = \frac{CS \times IR \times CF \times FI \times EF \times ED}{BW \times AF \times AD}$$

CS = UL95 chemical concentration in sediment (mg/kg)
IR = Ingestion rate (mg/d)
FI = Fraction ingested from contaminated source (1)
CF = Conversion factor (10-6 kg/mg)
EF = Exposure frequency (d/yr)
ED = Exposure duration (6 yr)
BW = Body weight (35 kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-23
RISK FROM INGESTION OF SURFACE WATER AT A LOCAL CREEK
RECEPTOR:WADING OLDER CHILD
USING MEAN VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	WATER CONCENTRATION (mg/L)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
ACETONE	4.90E-02	ND	8.05E-08	ND
BENZENE	1.29E-02	2.90E-02	2.11E-08	6.13E-10
BENZO(B)FLUORANTHENE	4.00E-03	ND	6.58E-09	ND
BIS(2-ETHYLHEXYL)PHTHALATE	9.35E-03	1.40E-02	1.54E-08	2.15E-10
BROMODICHLOROMETHANE	1.80E-03	6.20E-02	2.96E-09	1.83E-10
BROMOFORM	1.87E-03	7.90E-03	3.07E-09	2.43E-11
2-BUTANONE	3.18E-02	ND	5.23E-08	ND
DIBROMOCHLOROMETHANE	1.64E-03	8.40E-02	2.70E-09	2.26E-10
1,2-DICHLOROETHANE	1.75E-03	9.10E-02	2.88E-09	ND
1,2-DICHLOROPROPANE	1.30E-03	ND	2.14E-09	ND
METHYLENE CHLORIDE	1.07E-02	7.50E-03	1.76E-08	1.32E-10
TRANS-1,2-DICHLOROETHENE	1.16E-03	ND	1.91E-09	ND
TRANS-1,2-DICHLOROPROPENE	1.66E-03	ND	2.73E-09	ND
ARSENIC	1.44E-01	1.75E+00	2.37E-07	4.14E-07
BERYLLIUM	3.29E-03	4.30E+00	5.41E-09	2.33E-08
			TOTAL CANCER RISK =	4.39E-07

NON-CARCINOGENS	WATER CONCENTRATION (mg/L)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACETONE	4.90E-02	1.00E-01	9.40E-07	9.40E-06
BENZENE	1.08E-02	UR	2.06E-07	ND
BENZO(B)FLUORANTHENE	4.00E-03	ND	7.67E-08	ND
BIS(2-ETHYLHEXYL)PHTHALATE	9.35E-03	2.00E-02	1.79E-07	8.97E-06
BROMODICHLOROMETHANE	1.80E-03	2.00E-02	3.45E-08	1.73E-06
BROMOFORM	1.87E-03	2.00E-02	3.59E-08	1.79E-06
DIBROMOCHLOROMETHANE	1.64E-03	2.00E-02	3.15E-08	ND
FLUORANTHENE	5.55E-03	4.00E-02	1.06E-07	2.66E-06
METHYLENE CHLORIDE	1.07E-02	6.00E-02	2.05E-07	3.42E-06
PHENOL	7.40E-03	6.00E-01	1.42E-07	2.37E-07
PYRENE	5.17E-03	3.00E-02	9.92E-08	3.31E-06
TOLUENE	5.21E-03	2.00E-01	9.99E-08	5.00E-07
XYLENE (TOTAL)	2.89E-03	2.00E+00	5.54E-08	2.77E-08
2-BUTANONE	3.18E-02	5.00E-02	6.10E-07	1.22E-05
1,2-DICHLOROETHANE	1.75E-03	ND	3.36E-08	ND
1,2-DICHLOROPROPANE	1.30E-03	ND	2.49E-08	ND
2,4-DIMETHYLPHENOL	5.60E-03	2.00E-02	1.07E-07	5.37E-06
4-METHYLPHENOL	7.03E-03	ND	1.35E-07	ND
TRANS-1,2-DICHLOROETHENE	1.06E-03	2.00E-02	2.03E-08	1.02E-06
TRANS-1,3-DICHLOROPROPENE	1.66E-03	3.00E-04	3.18E-08	1.06E-04
ALUMINUM	7.74E+00	1.00E+00	1.48E-04	1.48E-04
ANTIMONY	3.20E-02	4.00E-04	6.13E-07	1.53E-03
ARSENIC	1.44E-01	3.00E-04	2.76E-06	9.21E-03
BARIUM	2.50E-01	5.00E-02	4.79E-06	9.59E-05
BERYLLIUM	3.29E-03	5.00E-03	6.31E-08	1.26E-05
BORON	3.35E+00	9.00E-02	6.42E-05	7.14E-04
CADMIUM	3.40E-03	5.00E-04	6.52E-08	1.30E-04
CALCIUM	1.81E+02	ND	3.47E-03	ND
CHROMIUM	2.37E-02	5.00E-03	4.55E-07	9.09E-05
COBALT	3.07E-02	9.60E-01	5.89E-07	6.13E-07
COPPER	5.56E-02	4.00E-02	1.07E-06	2.67E-05
LEAD	1.29E-01	ND	2.47E-06	ND
MAGNESIUM	5.52E+01	ND	1.06E-03	ND
MANGANESE	1.68E+00	5.00E-03	3.22E-05	6.44E-03
MERCURY	1.70E-04	3.00E-04	3.26E-09	1.09E-05
MOLYBDENUM	7.06E-02	5.00E-03	1.35E-06	2.71E-04
NICKEL	5.75E-02	2.00E-02	1.10E-06	5.51E-05
SELENIUM	1.62E-01	5.00E-03	3.11E-06	6.21E-04
SILVER	5.71E-03	5.00E-03	1.10E-07	2.19E-05
THALLIUM	1.98E-01	ND	3.80E-06	ND
VANADIUM	7.68E-02	7.00E-03	1.47E-06	2.10E-04
ZINC	6.36E-01	ND	1.22E-05	ND
			HAZARD INDEX =	1.97E-02

$$\text{Intake(mg/kg-d)} = \frac{CW \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CW = mean chemical concentration in surfacewater (mg/L)
IR = Ingestion rate (l/hr)
ET = Exposure time (hr/day)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-24
RISK FROM INGESTION OF SURFACE WATER AT A LOCAL CREEK
RECEPTOR:WADING OLDER CHILD
USING RME VALUES

CONTAMINANT OF CONCERN

CARCINOGENS	WATER CONCENTRATION (mg/L)	ORAL SLOPE FACTOR (mg/kg/d)-1	INTAKE (mg/kg-d)	RISK
ACETONE	9.90E-02	ND	2.32E-07	ND
BENZENE	1.10E-01	2.90E-02	2.58E-07	7.49E-09
BENZO(B)FLUORANTHENE	4.00E-03	ND	9.39E-09	ND
BIS(2-ETHYLHEXYL)PHTHALATE	2.80E-02	1.40E-02	6.58E-08	9.21E-10
BROMODICHLOROMETHANE	2.44E-03	6.20E-02	5.73E-09	3.55E-10
BROMOFORM	2.66E-03	7.90E-03	6.25E-09	4.93E-11
2-BUTANONE	9.40E-02	ND	2.21E-07	ND
DIBROMOCHLOROMETHANE	2.00E-03	8.40E-02	4.70E-09	3.95E-10
1,2-DICHLOROETHANE	2.44E-03	9.10E-02	5.73E-09	ND
1,2-DICHLOROPROPANE	1.30E-03	ND	3.05E-09	ND
METHYLENE CHLORIDE	1.59E-02	7.50E-03	3.72E-08	2.79E-10
TRANS-1,2-DICHLOROETHENE	1.90E-03	ND	4.46E-09	ND
TRANS-1,3-DICHLOROPROPENE	2.20E-03	ND	5.17E-09	ND
ARSENIC	1.91E-01	1.75E+00	4.49E-07	7.85E-07
BERYLLIUM	4.47E-03	4.30E+00	1.05E-08	4.51E-08

TOTAL CANCER RISK = 8.40E-07

NON-CARCINOGENS	WATER CONCENTRATION (mg/L)	ORAL RfD (mg/kg/d)	INTAKE (mg/kg-d)	HAZARD QUOTIENT
ACETONE	9.90E-02	1.00E-01	2.71E-06	2.71E-05
BENZENE	1.10E-01	UR	3.01E-06	ND
BENZO(B)FLUORANTHENE	4.00E-03	ND	1.10E-07	ND
BIS(2-ETHYLHEXYL)PHTHALATE	2.80E-02	2.00E-02	7.67E-07	3.84E-05
BROMODICHLOROMETHANE	2.44E-03	2.00E-02	6.68E-08	3.34E-06
BROMOFORM	2.66E-03	2.00E-02	7.29E-08	3.64E-06
DIBROMOCHLOROMETHANE	2.00E-03	2.00E-02	5.48E-08	ND
FLUORANTHENE	6.43E-03	4.00E-02	1.76E-07	4.40E-06
METHYLENE CHLORIDE	1.59E-02	6.00E-02	4.35E-07	7.24E-06
PHENOL	1.09E-02	6.00E-01	2.99E-07	4.98E-07
PYRENE	6.29E-03	3.00E-02	1.72E-07	5.74E-06
TOLUENE	6.00E-03	2.00E-01	1.64E-07	8.22E-07
XYLENE (TOTAL)	8.77E-03	2.00E+00	2.40E-07	1.20E-07
2-BUTANONE	9.40E-02	5.00E-02	2.58E-06	5.15E-05
1,2-DICHLOROETHANE	2.44E-03	ND	6.68E-08	ND
1,2-DICHLOROPROPANE	1.30E-03	ND	3.56E-08	ND
2,4-DIMETHYLPHENOL	6.48E-03	2.00E-02	1.78E-07	8.88E-06
4-METHYLPHENOL	1.03E-02	ND	2.82E-07	ND
TRANS-1,2-DICHLOROETHENE	1.90E-03	2.00E-02	5.21E-08	2.60E-06
TRANS-1,3-DICHLOROPROPENE	2.20E-03	3.00E-04	6.03E-08	2.01E-04
ALUMINUM	8.17E+01	1.00E+00	2.24E-03	2.24E-03
ANTIMONY	4.08E-02	4.00E-04	1.12E-06	2.80E-03
ARSENIC	1.91E-01	3.00E-04	5.23E-06	1.74E-02
BARIUM	4.58E-01	5.00E-02	1.25E-05	2.51E-04
BERYLLIUM	4.47E-03	5.00E-03	1.22E-07	2.45E-05
BORON	5.73E+00	9.00E-02	1.57E-04	1.74E-03
CADMIUM	4.74E-02	5.00E-04	1.30E-06	2.60E-03
CALCIUM	2.78E+02	ND	7.60E-03	ND
CHROMIUM	8.66E-02	5.00E-03	2.37E-06	4.75E-04
COBALT	3.90E-02	9.60E-01	1.07E-06	1.11E-06
COPPER	1.95E-01	4.00E-02	5.35E-06	1.34E-04
LEAD	3.20E+02	ND	8.76E-03	ND
MAGNESIUM	5.56E+01	ND	1.52E-03	ND
MANGANESE	6.62E+00	5.00E-03	1.81E-04	3.63E-02
MERCURY	5.10E-04	3.00E-04	1.40E-08	4.66E-05
MOLYBDENUM	9.54E-02	5.00E-03	2.61E-06	5.23E-04
NICKEL	1.35E-01	2.00E-02	3.71E-06	1.85E-04
SELENIUM	2.97E-01	5.00E-03	8.14E-06	1.63E-03
SILVER	6.76E-03	5.00E-03	1.85E-07	3.70E-05
THALLIUM	3.97E-01	ND	1.09E-05	ND
VANADIUM	1.87E-01	7.00E-03	5.12E-06	7.32E-04
ZINC	5.38E+00	ND	1.47E-04	ND

HAZARD INDEX = 6.75E-02

$$\text{Intake(mg/kg-d)} = \frac{CW \times IR \times ET \times EF \times ED}{BW \times AF \times AD}$$

CW = mean chemical concentration in surfacewater (mg/L)
IR = Ingestion rate (l/hr)
ET = Exposure time (hr/day)
EF = Exposure frequency (d/yr)
ED = Exposure duration (yr)
BW = Body weight (kg)
AF = Averaging frequency (365 d/yr); and
AD = Averaging duration, yr (equal to ED for noncarcinogens and 70 years for carcinogens)

TABLE C-25
RISK FROM DERMAL CONTACT WITH CADMIUM IN SOIL
RECEPTORS: EMPLOYEES AND OLDER CHILD TRANSIENTS
USING MEAN AND RME VALUES

MEAN VALUES

LOCATION	RECEPTOR	CADMIUM CONCENTRATION (ppm)	DERMAL ABSORPTION/EVENT (mg/cm-event)	DERMALLY ABSORBED DOSE (mg/kg-day)	ORAL RfD (mg/kg-d)	HAZARD QUOTIENT
LINDE	EMPLOYEE	2.08E+00	4.16E-09	2.16E-07	5.00E-04	4.31E-04
ASHLAND 1	TRANSIENT ADULT	0.00E+00	0.00E+00	0.00E+00	5.00E-04	NA
ASHLAND 1	EMPLOYEE	0.00E+00	0.00E+00	0.00E+00	5.00E-04	NA
ASHLAND 2	TRANSIENT CHILD	9.07E+00	1.81E-08	2.63E-07	5.00E-04	5.25E-04
ASHLAND 2	EMPLOYEE	9.07E+00	1.81E-08	9.41E-07	5.00E-04	1.88E-03
LOCAL CREEK	WADING CHILD	3.40E+00	3.40E-11	2.24E-10	5.00E-04	4.47E-07

RME VALUES

LOCATION	RECEPTOR	CADMIUM CONCENTRATION (ppm)	DERMAL ABSORPTION/EVENT (mg/cm-event)	DERMALLY ABSORBED DOSE (mg/kg-day)	ORAL RfD (mg/kg-d)	HAZARD QUOTIENT
LINDE	EMPLOYEE	2.70E+00	5.40E-09	2.80E-07	5.00E-04	5.60E-04
ASHLAND 1	TRANSIENT ADULT	0.00E+00	0.00E+00	0.00E+00	5.00E-04	NA
ASHLAND 1	EMPLOYEE	0.00E+00	0.00E+00	0.00E+00	5.00E-04	NA
ASHLAND 2	TRANSIENT CHILD	3.43E+01	6.86E-08	2.58E-06	5.00E-04	5.17E-03
ASHLAND 2	EMPLOYEE	3.43E+01	6.86E-08	3.56E-06	5.00E-04	7.12E-03
LOCAL CREEK	WADING CHILD	6.20E+00	6.20E-11	4.08E-10	5.00E-04	8.15E-07

$$\text{DAD (mg/kg-d)} = \frac{\text{DAevent} \times \text{EF} \times \text{ED} \times \text{SA}}{\text{BW} \times \text{AT}}$$

DAD = Dermally absorbed dose (mg/kg-day)
DA event = Absorbed dose per event (mg/cm2-event)
SA = Skin surface area available for contact (cm2)
EF = Exposure frequency (d/yr)
ED = Exposure duration (6 yr)
BW = Body weight (kg)
AT = averaging time

$$\text{DAevent (mg/cm2-event)} = \text{Csoil} \times \text{AF} \times \text{ABS}$$

DA event = Absorbed dose per event (mg/cm2-event)
Csoil = Contaminant concentration in soil (mg/kg) X (10-6 kg/mg)
AF = Adherence factor (mg/cm2-event)
ABS = Absorption fraction