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DRAFT FINAL BASELINE RISK ASSESSMENT FOR FOCUSED FEASIBILITY STUDY NAS
FORT WORTH TX
1/1/2001
HYDROGEOLOGIC



**NAVAL AIR STATION
FORT WORTH JRB
CARSWELL FIELD
TEXAS**

**ADMINISTRATIVE RECORD
COVER SHEET**

AR File Number 661



**DRAFT FINAL
BASELINE RISK ASSESSMENT
FOR THE FOCUSED FEASIBILITY STUDY
FORMER CARSWELL AIR FORCE BASE, TEXAS**

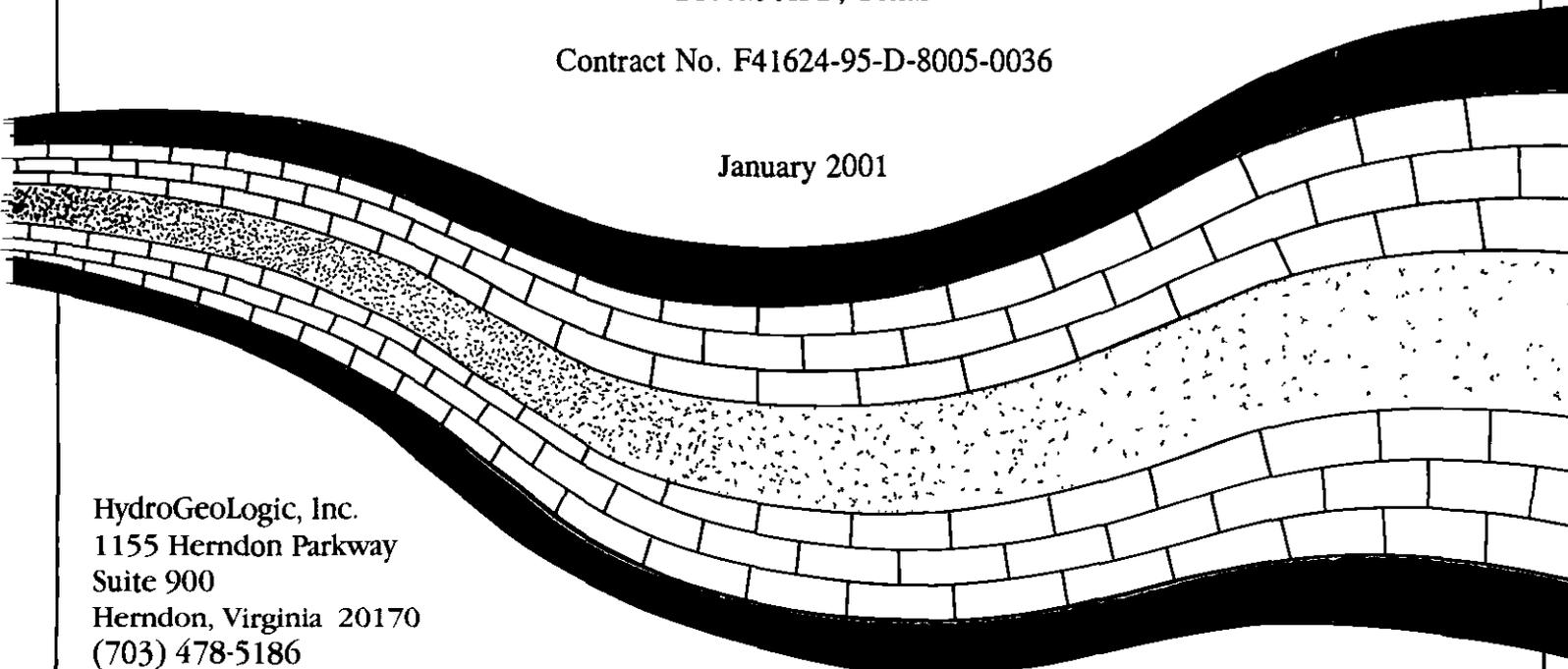


Prepared for

Air Force Center for Environmental Excellence
Brooks AFB, Texas

Contract No. F41624-95-D-8005-0036

January 2001

A decorative graphic consisting of several wavy, horizontal bands. The top band is solid black. Below it are several bands with a brick-like pattern. The bottom-most band is filled with a stippled or dotted texture.

HydroGeoLogic, Inc.
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January 2001

**ECOLOGICAL RISK ASSESSMENT
TIER 1: EXCLUSION CRITERIA CHECKLIST**

Figure : 30 TAC §350.77(b)

TIER 1: Exclusion Criteria Checklist

This exclusion criteria checklist is intended to aid the person and the TNRCC in determining whether or not further ecological evaluation is necessary at an affected property where a response action is being pursued under the Texas Risk Reduction Program (TRRP) Exclusion criteria refer to those conditions at an affected property which preclude the need for a formal ecological risk assessment (ERA) because there are incomplete or insignificant ecological exposure pathways due to the nature of the affected property setting and/or the condition of the affected property media. This checklist (and/or a Tier 2 or 3 ERA or the equivalent) must be completed by the person for all affected property subject to the TRRP. The person should be familiar with the affected property but need not be a professional scientist in order to respond, although some questions will likely require contacting a wildlife management agency (i.e., Texas Parks and Wildlife Department or U.S. Fish and Wildlife Service). The checklist is designed for general applicability to all affected property; however, there may be unusual circumstances which require professional judgement in order to determine the need for further ecological evaluation (e.g., cave-dwelling receptors). In these cases, the person is strongly encouraged to contact TNRCC before proceeding.

Besides some preliminary information, the checklist consists of three major parts, each of which must be completed unless otherwise instructed. PART I requests affected property identification and background information. PART II contains the actual exclusion criteria and supportive information. PART III is a qualitative summary statement and a certification of the information provided by the person. Answers should reflect existing conditions and should not consider future remedial actions at the affected property. Completion of the checklist should lead to a logical conclusion as to whether further evaluation is warranted. Definitions of terms used in the checklist have been provided and users are strongly encouraged to familiarize themselves with these definitions before beginning the checklist.

Name of Facility:

Air Force Plant 4

Affected Property Location.

Former Carswell Air Force Base / Golf Course Area

Mailing Address:

ASC/ENVR, BLDG. 8
Attn: George Walters
1801 Tenth St, Suite 2

TNRCC Case Tracking #s:

None

Solid Waste Registration #s:

65004

Voluntary Cleanup Program #:

None

EPA I.D. #s:

Carswell – TX0571924042 and TPDES0118257

Figure: 30 TAC §350.77(b) continued

Definitions¹

Affected property - The entire area (i.e., on-site and off-site; including all environmental media) which contains releases of chemicals of concern at concentrations equal to or greater than the assessment level applicable for residential land use and groundwater classification.

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Chemical of concern - Any chemical that has the potential to adversely affect ecological or human receptors due to its concentration, distribution, and mode of toxicity. Depending on the program area, chemicals of concern may include the following: solid waste, industrial solid waste, municipal solid waste, and hazardous waste as defined in Texas Health and Safety Code, §361.003, as amended; hazardous constituents as listed in 40 Code of Federal Regulations Part 261, Appendix VIII, as amended; constituents on the groundwater monitoring list in 40 Code of Federal Regulations Part 264, Appendix IX, as amended; constituents as listed in 40 CFR Part 258 Appendices I and II, as amended; pollutant as defined in Texas Water Code, §26.001, as amended, hazardous substance as defined in Texas Health and Safety Code, §361.003, as amended, and the Texas Water Code §26.263, as amended; regulated substance as defined in Texas Water Code §26.342, as amended and §334.2 of this title (relating to Definitions), as amended; petroleum product as defined in Texas Water Code §26.342, as amended and §334.122(b)(12) of this title (relating to Definitions for ASTs), as amended, other substances as defined in Texas Water Code §26.039(a), as amended; and daughter products of the aforementioned constituents.

Community - An assemblage of plant and animal populations occupying the same habitat in which the various species interact via spatial and trophic relationships (e.g., a desert community or a pond community).

Complete exposure pathway - An exposure pathway where a human or ecological receptor is exposed to a chemical of concern via an exposure route (e.g., incidental soil ingestion, inhalation of volatiles and particulates, consumption of prey, etc).

De minimus - The description of an area of affected property comprised of one acre or less where the ecological risk is considered to be insignificant because of the small extent of contamination, the absence of protected species, the availability of similar unimpacted habitat nearby, and the lack of adjacent sensitive environmental areas.

¹These definitions were taken from 30 TAC §350.4 and may have both ecological and human health applications. For the purposes of this checklist, it is understood that only the ecological applications are of concern.

Figure: 30 TAC §350.77(b) continued

Ecological protective concentration level - The concentration of a chemical of concern at the point of exposure within an exposure medium (e.g., soil, sediment, groundwater, or surface water) which is determined in accordance with §350.77(c) or (d) of this title (relating to Ecological Risk Assessment and Development of Ecological Protective Concentration Levels) to be protective for ecological receptors. These concentration levels are primarily intended to be protective for more mobile or wide-ranging ecological receptors and, where appropriate, benthic invertebrate communities within the waters in the state. These concentration levels are not intended to be directly protective of receptors with limited mobility or range (e.g., plants, soil invertebrates, and small rodents), particularly those residing within active areas of a facility, unless these receptors are threatened/endangered species or unless impacts to these receptors result in disruption of the ecosystem or other unacceptable consequences for the more mobile or wide-ranging receptors (e.g., impacts to an off-site grassland habitat eliminate rodents which causes a desirable owl population to leave the area)

Ecological risk assessment - The process that evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors; however, as used in this context, only chemical stressors (i.e., COCs) are evaluated.

Environmental medium - A material found in the natural environment such as soil (including non-waste fill materials), groundwater, air, surface water, and sediments, or a mixture of such materials with liquids, sludges, gases, or solids, including hazardous waste which is inseparable by simple mechanical removal processes, and is made up primarily of natural environmental material.

Exclusion criteria - Those conditions at an affected property which preclude the need to establish a protective concentration level for an ecological exposure pathway because the exposure pathway between the chemical of concern and the ecological receptors is not complete or is insignificant.

Exposure medium - The environmental medium or biologic tissue in which or by which exposure to chemicals of concern by ecological or human receptors occurs.

Facility - The installation associated with the affected property where the release of chemicals of concern occurred.

Functioning cap - A low permeability layer or other approved cover meeting its design specifications to minimize water infiltration and chemical of concern migration, and prevent ecological or human receptor exposure to chemicals of concern, and whose design requirements are routinely maintained.

Landscaped area - An area of ornamental, or introduced, or commercially installed, or manicured vegetation which is routinely maintained.

Off-site property (off-site) - All environmental media which is outside of the legal boundaries of the on-site property.

On-site property (on-site) - All environmental media within the legal boundaries of a property owned or leased by a person who has filed a self-implementation notice or a response action plan for that property or who has become subject to such action through one of the agency's program areas for that property.

Figure: 30 TAC §350.77(b) continued

Physical barrier - Any structure or system, natural or manmade, that prevents exposure or prevents migration of chemicals of concern to the points of exposure.

Point of exposure - The location within an environmental medium where a receptor will be assumed to have a reasonable potential to come into contact with chemicals of concern. The point of exposure may be a discrete point, plane, or an area within or beyond some location.

Protective concentration level - The concentration of a chemical of concern which can remain within the source medium and not result in levels which exceed the applicable human health risk-based exposure limit or ecological protective concentration level at the point of exposure for that exposure pathway.

Release - Any spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing into the environment, with the exception of:

(A) A release that results in an exposure to a person solely within a workplace, concerning a claim that the person may assert against the person's employer;

(B) An emission from the engine exhaust of a motor vehicle, rolling stock, aircraft, vessel, or pipeline pumping station engine;

(C) A release of source, by-product, or special nuclear material from a nuclear incident, as those terms are defined by the Atomic Energy Act of 1954, as amended (42 U.S.C. §2011 et seq.), if the release is subject to requirements concerning financial protection established by the Nuclear Regulatory Commission under §170 of that Act;

(D) For the purposes of the environmental response law §104, as amended, or other response action, a release of source, by-product, or special nuclear material from a processing site designated under §102(a)(1) or §302(a) of the Uranium Mill Tailings Radiation Control Act of 1978 (42 U.S.C. §7912 and §7942), as amended, and

(E) The normal application of fertilizer.

Sediment - Non-suspended particulate material lying below surface waters such as bays, the ocean, rivers, streams, lakes, ponds, or other similar surface water body (including intermittent streams). Dredged sediments which have been removed from below surface water bodies and placed on land shall be considered soils.

Sensitive environmental areas - Areas that provide unique and often protected habitat for wildlife species. These areas are typically used during critical life stages such as breeding, hatching, rearing of young, and overwintering. Examples include critical habitat for threatened and endangered species, wilderness areas, parks, and wildlife refuges.

Source medium - An environmental medium containing chemicals of concern which must be removed, decontaminated and/or controlled in order to protect human health and the environment. The source medium may be the exposure medium for some exposure pathways.

Stressor - Any physical, chemical, or biological entity that can induce an adverse response; however, as used in this context, only chemical entities apply.

Figure: 30 TAC §350.77(b) continued

Subsurface soil - For human health exposure pathways, the portion of the soil zone between the base of surface soil and the top of the groundwater-bearing unit(s). For ecological exposure pathways, the portion of the soil zone between 0.5 feet and 5 feet in depth.

Surface cover - A layer of artificially placed utility material (e.g., shell, gravel).

Surface soil - For human health exposure pathways, the soil zone extending from ground surface to 15 feet in depth for residential land use and from ground surface to 5 feet in depth for commercial/industrial land use; or to the top of the uppermost groundwater-bearing unit or bedrock, whichever is less in depth. For ecological exposure pathways, the soil zone extending from ground surface to 0.5 feet in depth.

Surface water - Any water meeting the definition of surface water in the state as defined in §307.3 of this title (relating to Abbreviations and Definitions), as amended.

Figure: 30 TAC §350.77(b) continued

PART I. Affected Property Identification and Background Information

- 1) Provide a description of the specific area of the response action and the nature of the release. Include estimated acreage of the affected property and the facility property, and a description of the type of facility and/or operation associated with the affected property. Also describe the location of the affected property with respect to the facility property boundaries and public roadways.

Air Force Plant 4

Air Force Plant (AFP) 4 became operational in 1942 when Consolidated Aircraft began manufacturing the B-24 bomber for national defense during World War II. In 1953, General Dynamics took over operation of the manufacturing facility. Since 1953, AFP 4 has produced B-36, B-58, F-111 aircraft. The plant currently produces F-16 aircraft. In addition to F-16 aircraft, AFP 4 produces spare parts, radar units, and missile components. On March 1, 1993, Lockheed, Forth Worth Company, took over operations of AFP 4 as a successor to General Dynamics. AFP 4 currently occupies 602 acres.

Manufacturing operations at AFP 4 have resulted in the generation of various hazardous wastes that include waste oils, fuels, spent solvents, paint residues, and spent process chemicals. Throughout most of the plant's history, waste oil, solvents, and fuels were disposed at on-site landfills or were burned during fire training exercises. Chemical wastes were initially discharged to the sanitary sewer system and treated by the City of Fort Worth's treatment system. In the 1970's, chemical process wastes were treated on site at a newly constructed chemical waste treatment system prior to being discharged to the sanitary sewer system. Currently, on site burning of waste has been discontinued while waste oils and solvents are disposed through a contractor. Chemical wastes continue to be treated on site. AFP 4 was placed on the National Priority List (NPL) in August 1990 because of a large release of trichloroethene (TCE) arising from past disposal practices at AFP 4. While the source areas are currently being remediated, the dissolved TCE plume appears to have migrated toward the east of APF 4 and extends under NAS Fort Worth JRB and the Former Carswell AFB/Base Realignment and Closure (BRAC) area. The plume is referred to as the southern lobe, and is migrating in a southeast direction.

NAS Fort Worth JRB

The NAS Fort Worth JRB started as a modest dirt runway built to service the aircraft manufacturing plant formerly located at AFP 4's current location. In August 1942, the base was opened as Tarrant Field Airdrome and was used to train pilots to fly B-24 bombers. In May 1943, the field was re-designed as Fort Worth Army Air Field. It was renamed Carswell Air Force Base in 1948, and the 7th Bomber Wing became the base host unit. The Strategic Air Command (SAC) mission remained at Carswell AFB until 1992, when the Air Combat Command assumed control of the base upon de-establishment of SAC. In October 1994, the U.S. Navy assumed responsibility for much of the facility, and its name was changed from Carswell AFB to NAS Fort Worth JRB. The principal activities on the base have been maintaining and servicing bombers, fuel tankers, and fighter jet aircraft.

Major industrial operations that have been performed at the NAS Fort Worth JRB include the following: maintenance of jet engines, aerospace ground equipment, fuel systems, weapons systems, pneudraulic systems and general and special purpose vehicles; aircraft corrosion control; and non-destructive inspection activities. Most liquid wastes that have been generated by industrial operations can be characterized as waste oils, recoverable fuels, spent solvent, and spent cleaners. Several landfills exist just up gradient of the BRAC area, with one landfill (SWMU 22) on the western portion of the BRAC property. Two areas of concern (AOC) exist within the BRAC area; they are the AOC 9, the Golf Course Maintenance Yard, and AOC 16, the Family Camp.

In 1991, the Corps of Engineers performed excavation activities at Waste Burial Area 7 (WP-07), SWMU 24, to remove a total of 34 drums, of which 9 were partially full, and 25 were empty. TCE and perchloroethylene (PCE)

were the primary constituents contained within the drums. These drums contributed to the southern lobe TCE plume contamination. As part of an RFI at SWMU 24, an electromagnetic survey was performed on May 2000, for the purpose of confirming drum removal activities performed by the Corps of Engineers. In July, 2000 IT Corporation began excavation activities to investigate twelve geophysical anomalies. A total of 16 metal 55-gallon drums were encountered. Of the 16 drums, 12 were empty, compressed, or A total of 21 metal 55-gallon drums were encountered between two areas. Of the 21 drums, 17 were empty, compressed, or corroded, and contained no liquids. Also discovered were lengths of pipe, a tire iron, and metal post. Three of the drums were still in tact and partially full with an unknown liquid. Analytical results from characterization sampling will be addressed under a separate and pending (December 2000) project report by IT Corporation, but preliminary results indicate that the drums contain at least a fraction of TCE. A fourth in tact drum contained a blue, wet, powdery substance. Analytical results from characterization sampling of this unknown powdery substance will also be addressed in the IT report on excavation activities. Although analytical results from excavation activities are not available for this Internal Draft Risk Assessment, it is expected that the analytical results will be available and incorporated in the Final Risk Assessment.

The resulting southern lobe TCE plume originating from AFP 4 and possibly other NAS Fort Worth source areas covers approximately 453 acres, 75 of which are on the BRAC property. The down gradient extent (TCE at 5 µg/L) of the plume is within 6 feet of the federal property boundary in WHGLRW015. An off-site well has been installed and analytical results are pending. Two additional offsite wells WHGLRW016 and WHGLRW017 (approximately 20 feet from the boundary show no detectable concentrations of TCE).

Attach available USGS topographic maps and/or aerial or other affected property photographs to this form to depict the affected property and surrounding area. Indicate attachments:

- Topo map
- Aerial photo
- Other

2) Identify environmental media known or suspected to contain chemicals of concern (COCs) at the present time. Check all that apply:

- | | | |
|---|---|-----------------------------|
| Known/Suspected COC Location | Based on sampling data? | |
| <input type="checkbox"/> Soil ≤ 5 ft below ground surface | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| <input type="checkbox"/> Soil >5 ft below ground surface | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| <input checked="" type="checkbox"/> Groundwater | <input checked="" type="checkbox"/> Yes | <input type="checkbox"/> No |
| <input checked="" type="checkbox"/> Surface Water/Sediments | <input checked="" type="checkbox"/> Yes | <input type="checkbox"/> No |

Explain (previously submitted information may be referenced):

Detected chemicals in groundwater, surface water and sediment are identified in Tables 6-1, 6-3, and 6-4, respectively.

Figure: 30 TAC §350.77(b) continued

- 3) Provide the information below for the nearest surface water body which has become or has the potential to become impacted from migrating COCs via surface water runoff, air deposition, groundwater seepage, etc. Exclude wastewater treatment facilities and stormwater conveyances/impoundments authorized by permit. Also exclude conveyances, decorative ponds, and those portions of process facilities which are:
- Not in contact with surface waters in the State or other surface waters which are ultimately in contact with surface waters in the State, and
 - Not consistently or routinely utilized as valuable habitat for natural communities including birds, mammals, reptiles, etc.

The nearest surface water body is 0 _____ feet/miles from the affected property and is named Farmers Branch Creek. The water body is best described as a:

- X freshwater stream: _____ perennial (has water all year)
 _____ intermittent (dries up completely for at least 1 week a year)
X intermittent with perennial pools
- freshwater swamp/marsh/wetland
 saltwater or brackish marsh/swamp/wetland
 reservoir, lake, or pond; approximate surface acres:
 drainage ditch
 tidal stream bay estuary
 other, specify

Is the water body listed as a State classified segment in Appendix C of the current Texas Surface Water Quality Standards; §§307.1 - 307.10?

Yes Segment # _____ Use Classification:

X No

If the water body is not a State classified segment, identify the first downstream classified segment.

Name: West Fork of the Trinity Below Lake Worth

Segment #: 0806

Use Classification: Contact recreation, high aquatic life use, public water supply

As necessary, provide further description of surface waters in the vicinity of the affected property:

Figure: 30 TAC §350.77(b) continued

PART II. Exclusion Criteria and Supportive Information**Subpart A. Surface Water/Sediment Exposure**

1) Regarding the affected property where a response action is being pursued under the TRRP, have COCs migrated and resulted in a release or imminent threat of release to either surface waters or to their associated sediments via surface water runoff, air deposition, groundwater seepage, etc.? Exclude wastewater treatment facilities and stormwater conveyances/impoundments authorized by permit. Also exclude conveyances, decorative ponds, and those portions of process facilities which are:

- a. Not in contact with surface waters in the State or other surface waters which are ultimately in contact with surface waters in the State; and
- b. Not consistently or routinely utilized as valuable habitat for natural communities including birds, mammals, reptiles, etc.

X Yes No

Explain:

Measured concentrations of volatile and semivolatile chemicals (see Tables 6-3 and 6-4) have been detected in surface water and sediment samples.

If the answer is Yes to Subpart A above, the affected property does not meet the exclusion criteria. However, complete the remainder of Part II to determine if there is a complete and/or significant soil exposure pathway, then complete PART III - Qualitative Summary and Certification. If the answer is No, go to Subpart B.

Soil is not included under this remedial investigation.

Subpart B. Affected Property Setting

In answering "Yes" to the following question, it is understood that the affected property is not attractive to wildlife or livestock, including threatened or endangered species (i.e., the affected property does not serve as valuable habitat, foraging area, or refuge for ecological communities). (May require consultation with wildlife management agencies.)

1) Is the affected property wholly contained within contiguous land characterized by: pavement, buildings, landscaped area, functioning cap, roadways, equipment storage area, manufacturing or process area, other surface cover or structure, or otherwise disturbed ground?

X Yes No

Explain:

Figure: 30 TAC §350.77(b) continued

If the answer to Subpart B above is Yes, the affected property meets the exclusion criteria, assuming the answer to Subpart A was No. Skip Subparts C and D and complete PART III - Qualitative Summary and Certification. If the answer to Subpart B above is No, go to Subpart C.

Subpart C. Soil Exposure

- 1) Are COCs which are in the soil of the affected property solely below the first 5 feet beneath ground surface or does the affected property have a physical barrier present to prevent exposure of receptors to COCs in surface soil?

Yes No

Explain:

Soil is not included under this remedial investigation.

If the answer to Subpart C above is Yes, the affected property meets the exclusion criteria, assuming the answer to Subpart A was No. Skip Subpart D and complete PART III - Qualitative Summary and Certification. If the answer to Subpart C above is No, proceed to Subpart D.

Subpart D. *De Minimus* Land Area

In answering "Yes" to the question below, it is understood that all of the following conditions apply:

- ❖ The affected property is not known to serve as habitat, foraging area, or refuge to threatened/endangered or otherwise protected species. (Will likely require consultation with wildlife management agencies.)
- ❖ Similar but unimpacted habitat exists within a half-mile radius.
- ❖ The affected property is not known to be located within one-quarter mile of sensitive environmental areas (e.g , rookeries, wildlife management areas, preserves). (Will likely require consultation with wildlife management agencies.)
- ❖ There is no reason to suspect that the COCs associated with the affected property will migrate such that the affected property will become larger than one acre.

- 1) Using human health protective concentration levels as a basis to determine the extent of the COCs, does the affected property consist of one acre or less and does it meet all of the conditions above?

Yes No

Explain how conditions are met/not met:

The surface water body is contained within a golf course area that is highly maintained and does not serve as a viable habitat for threatened/endangered or otherwise protected species.

Figure: 30 TAC §350.77(b) continued

If the answer to Subpart D above is Yes, then no further ecological evaluation is needed at this affected property, assuming the answer to Subpart A was No. Complete PART III - Qualitative Summary and Certification. If the answer to Subpart D above is No, proceed to Tier 2 or 3 or comparable ERA.

PART III. Qualitative Summary and Certification (Complete in all cases.)

Attach a brief statement (not to exceed 1 page) summarizing the information you have provided in this form. This summary should include sufficient information to verify that the affected property meets or does not meet the exclusion criteria. The person should make the initial decision regarding the need for further ecological evaluation (i.e., Tier 2 or 3) based upon the results of this checklist. After review, TNRCC will make a final determination on the need for further assessment. **Note that the person has the continuing obligation to re-enter the ERA process if changing circumstances result in the affected property not meeting the Tier 1 exclusion criteria.**

Completed by: Deborah L. McKean, Ph D (Typed/Printed Name)

Senior Toxicologist, IT Corporation (Title)

November 20, 2000 (Date)

I believe that the information submitted is true, accurate, and complete, to the best of my knowledge.

_____ (Typed/Printed Name of Person)

_____ (Title of Person)

_____ (Signature of Person)

_____ (Date Signed)

HydroGeoLogic, Inc.—NAS Fort Worth JRB, Texas

Figure A.1

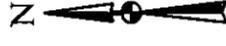
Topographic/Aerial Map
Golf Course/BRAC Area



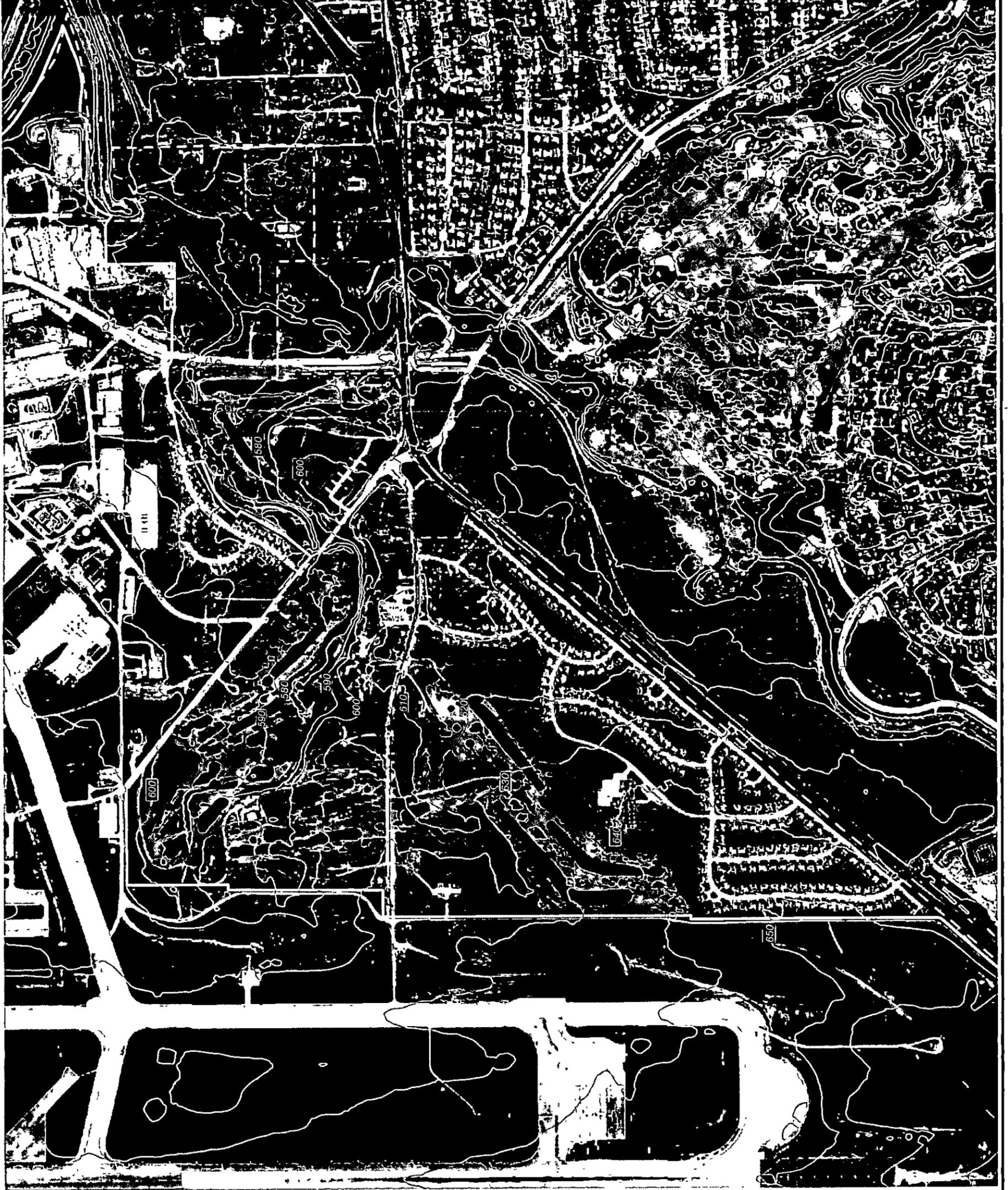
U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- 10' Contours



Project AFC001-36CA
 Filename X:\AFC001\36ca\Report\Golf_Course.apr
 Created 11/17/00 ASP
 Revised 12/04/00 cf
 Map Source HydroGeoLogic, Inc.—GIS Database



**ECOLOGICAL RISK ASSESSMENT
TIER 1: EXCLUSION CRITERIA CHECKLIST**

Figure : 30 TAC §350.77(b)

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ASC/ENVR, BLDG. 8
Attn: George Walters
1801 Tenth St, Suite 2

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65004

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Chemical of concern - Any chemical that has the potential to adversely affect ecological or human receptors due to its concentration, distribution, and mode of toxicity. Depending on the program area, chemicals of concern may include the following: solid waste, industrial solid waste, municipal solid waste, and hazardous waste as defined in Texas Health and Safety Code, §361.003, as amended; hazardous constituents as listed in 40 Code of Federal Regulations Part 261, Appendix VIII, as amended; constituents on the groundwater monitoring list in 40 Code of Federal Regulations Part 264, Appendix IX, as amended; constituents as listed in 40 CFR Part 258 Appendices I and II, as amended; pollutant as defined in Texas Water Code, §26.001, as amended; hazardous substance as defined in Texas Health and Safety Code, §361.003, as amended, and the Texas Water Code §26.263, as amended; regulated substance as defined in Texas Water Code §26.342, as amended and §334.2 of this title (relating to Definitions), as amended; petroleum product as defined in Texas Water Code §26.342, as amended and §334.122(b)(12) of this title (relating to Definitions for ASTs), as amended; other substances as defined in Texas Water Code §26.039(a), as amended; and daughter products of the aforementioned constituents.

Community - An assemblage of plant and animal populations occupying the same habitat in which the various species interact via spatial and trophic relationships (e.g., a desert community or a pond community).

Complete exposure pathway - An exposure pathway where a human or ecological receptor is exposed to a chemical of concern via an exposure route (e.g., incidental soil ingestion, inhalation of volatiles and particulates, consumption of prey, etc).

De minimus - The description of an area of affected property comprised of one acre or less where the ecological risk is considered to be insignificant because of the small extent of contamination, the absence of protected species, the availability of similar unimpacted habitat nearby, and the lack of adjacent sensitive environmental areas.

¹These definitions were taken from 30 TAC §350.4 and may have both ecological and human health applications. For the purposes of this checklist, it is understood that only the ecological applications are of concern.

Figure: 30 TAC §350.77(b) continued

Ecological protective concentration level - The concentration of a chemical of concern at the point of exposure within an exposure medium (e.g., soil, sediment, groundwater, or surface water) which is determined in accordance with §350.77(c) or (d) of this title (relating to Ecological Risk Assessment and Development of Ecological Protective Concentration Levels) to be protective for ecological receptors. These concentration levels are primarily intended to be protective for more mobile or wide-ranging ecological receptors and, where appropriate, benthic invertebrate communities within the waters in the state. These concentration levels are not intended to be directly protective of receptors with limited mobility or range (e.g., plants, soil invertebrates, and small rodents), particularly those residing within active areas of a facility, unless these receptors are threatened/endangered species or unless impacts to these receptors result in disruption of the ecosystem or other unacceptable consequences for the more mobile or wide-ranging receptors (e.g., impacts to an off-site grassland habitat eliminate rodents which causes a desirable owl population to leave the area).

Ecological risk assessment - The process that evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors; however, as used in this context, only chemical stressors (i.e., COCs) are evaluated.

Environmental medium - A material found in the natural environment such as soil (including non-waste fill materials), groundwater, air, surface water, and sediments, or a mixture of such materials with liquids, sludges, gases, or solids, including hazardous waste which is inseparable by simple mechanical removal processes, and is made up primarily of natural environmental material.

Exclusion criteria - Those conditions at an affected property which preclude the need to establish a protective concentration level for an ecological exposure pathway because the exposure pathway between the chemical of concern and the ecological receptors is not complete or is insignificant.

Exposure medium - The environmental medium or biologic tissue in which or by which exposure to chemicals of concern by ecological or human receptors occurs.

Facility - The installation associated with the affected property where the release of chemicals of concern occurred

Functioning cap - A low permeability layer or other approved cover meeting its design specifications to minimize water infiltration and chemical of concern migration, and prevent ecological or human receptor exposure to chemicals of concern, and whose design requirements are routinely maintained.

Landscaped area - An area of ornamental, or introduced, or commercially installed, or manicured vegetation which is routinely maintained.

Off-site property (off-site) - All environmental media which is outside of the legal boundaries of the on-site property.

On-site property (on-site) - All environmental media within the legal boundaries of a property owned or leased by a person who has filed a self-implementation notice or a response action plan for that property or who has become subject to such action through one of the agency's program areas for that property.

Figure: 30 TAC §350.77(b) continued

Physical barrier - Any structure or system, natural or manmade, that prevents exposure or prevents migration of chemicals of concern to the points of exposure.

Point of exposure - The location within an environmental medium where a receptor will be assumed to have a reasonable potential to come into contact with chemicals of concern. The point of exposure may be a discrete point, plane, or an area within or beyond some location.

Protective concentration level - The concentration of a chemical of concern which can remain within the source medium and not result in levels which exceed the applicable human health risk-based exposure limit or ecological protective concentration level at the point of exposure for that exposure pathway.

Release - Any spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing into the environment, with the exception of:

(A) A release that results in an exposure to a person solely within a workplace, concerning a claim that the person may assert against the person's employer;

(B) An emission from the engine exhaust of a motor vehicle, rolling stock, aircraft, vessel, or pipeline pumping station engine;

(C) A release of source, by-product, or special nuclear material from a nuclear incident, as those terms are defined by the Atomic Energy Act of 1954, as amended (42 U.S.C. §2011 et seq.), if the release is subject to requirements concerning financial protection established by the Nuclear Regulatory Commission under §170 of that Act;

(D) For the purposes of the environmental response law §104, as amended, or other response action, a release of source, by-product, or special nuclear material from a processing site designated under §102(a)(1) or §302(a) of the Uranium Mill Tailings Radiation Control Act of 1978 (42 U.S.C. §7912 and §7942), as amended; and

(E) The normal application of fertilizer.

Sediment - Non-suspended particulate material lying below surface waters such as bays, the ocean, rivers, streams, lakes, ponds, or other similar surface water body (including intermittent streams). Dredged sediments which have been removed from below surface water bodies and placed on land shall be considered soils.

Sensitive environmental areas - Areas that provide unique and often protected habitat for wildlife species. These areas are typically used during critical life stages such as breeding, hatching, rearing of young, and overwintering. Examples include critical habitat for threatened and endangered species, wilderness areas, parks, and wildlife refuges.

Source medium - An environmental medium containing chemicals of concern which must be removed, decontaminated and/or controlled in order to protect human health and the environment. The source medium may be the exposure medium for some exposure pathways.

Stressor - Any physical, chemical, or biological entity that can induce an adverse response, however, as used in this context, only chemical entities apply.

Figure. 30 TAC §350 77(b) continued

Subsurface soil - For human health exposure pathways, the portion of the soil zone between the base of surface soil and the top of the groundwater-bearing unit(s). For ecological exposure pathways, the portion of the soil zone between 0.5 feet and 5 feet in depth.

Surface cover - A layer of artificially placed utility material (e.g., shell, gravel).

Surface soil - For human health exposure pathways, the soil zone extending from ground surface to 15 feet in depth for residential land use and from ground surface to 5 feet in depth for commercial/industrial land use; or to the top of the uppermost groundwater-bearing unit or bedrock, whichever is less in depth. For ecological exposure pathways, the soil zone extending from ground surface to 0.5 feet in depth.

Surface water - Any water meeting the definition of surface water in the state as defined in §307.3 of this title (relating to Abbreviations and Definitions), as amended.

Figure: 30 TAC §350.77(b) continued

PART I. Affected Property Identification and Background Information

- 1) Provide a description of the specific area of the response action and the nature of the release. Include estimated acreage of the affected property and the facility property, and a description of the type of facility and/or operation associated with the affected property. Also describe the location of the affected property with respect to the facility property boundaries and public roadways

Air Force Plant 4

Air Force Plant (AFP) 4 became operational in 1942 when Consolidated Aircraft began manufacturing the B-24 bomber for national defense during World War II. In 1953, General Dynamics took over operation of the manufacturing facility. Since 1953, AFP 4 has produced B-36, B-58, F-111 aircraft. The plant currently produces F-16 aircraft. In addition to F-16 aircraft, AFP 4 produces spare parts, radar units, and missile components. On March 1, 1993, Lockheed, Forth Worth Company, took over operations of AFP 4 as a successor to General Dynamics. AFP 4 currently occupies 602 acres.

Manufacturing operations at AFP 4 have resulted in the generation of various hazardous wastes that include waste oils, fuels, spent solvents, paint residues, and spent process chemicals. Throughout most of the plant's history, waste oil, solvents, and fuels were disposed at on-site landfills or were burned during fire training exercises. Chemical wastes were initially discharged to the sanitary sewer system and treated by the City of Fort Worth's treatment system. In the 1970's, chemical process wastes were treated on site at a newly constructed chemical waste treatment system prior to being discharged to the sanitary sewer system. Currently, on site burning of waste has been discontinued while waste oils and solvents are disposed through a contractor. Chemical wastes continue to be treated on site. AFP 4 was placed on the National Priority List (NPL) in August 1990 because of a large release of trichloroethene (TCE) arising from past disposal practices at AFP 4. While the source areas are currently being remediated, the dissolved TCE plume appears to have migrated toward the east of APF 4 and extends under NAS Fort Worth JRB and the Former Carswell AFB/Base Realignment and Closure (BRAC) area. The plume is referred to as the southern lobe, and is migrating in a southeast direction.

NAS Fort Worth JRB

The NAS Fort Worth JRB started as a modest dirt runway built to service the aircraft manufacturing plant formerly located at AFP 4's current location. In August 1942, the base was opened as Tarrant Field Airdrome and was used to train pilots to fly B-24 bombers. In May 1943, the field was re-designed as Fort Worth Army Air Field. It was renamed Carswell Air Force Base in 1948, and the 7th Bomber Wing became the base host unit. The Strategic Air Command (SAC) mission remained at Carswell AFB until 1992, when the Air Combat Command assumed control of the base upon de-establishment of SAC. In October 1994, the U.S. Navy assumed responsibility for much of the facility, and its name was changed from Carswell AFB to NAS Fort Worth JRB. The principal activities on the base have been maintaining and servicing bombers, fuel tankers, and fighter jet aircraft.

Major industrial operations that have been performed at the NAS Fort Worth JRB include the following: maintenance of jet engines, aerospace ground equipment, fuel systems, weapons systems, pneudraulic systems and general and special purpose vehicles; aircraft corrosion control; and non-destructive inspection activities. Most liquid wastes that have been generated by industrial operations can be characterized as waste oils, recoverable fuels, spent solvent, and spent cleaners. Several landfills exist just up gradient of the BRAC area, with one landfill (SWMU 22) on the western portion of the BRAC property. Two areas of concern (AOC) exist within the BRAC area; they are the AOC 9, the Golf Course Maintenance Yard, and AOC 16, the Family Camp.

In 1991, the Corps of Engineers performed excavation activities at Waste Burial Area 7 (WP-07), SWMU 24, to remove a total of 34 drums, of which 9 were partially full, and 25 were empty. TCE and perchloroethylene (PCE)

were the primary constituents contained within the drums. These drums contributed to the southern lobe TCE plume contamination. As part of an RFI at SWMU 24, an electromagnetic survey was performed on May 2000, for the purpose of confirming drum removal activities performed by the Corps of Engineers. In July, 2000 IT Corporation began excavation activities to investigate twelve geophysical anomalies. A total of 16 metal 55-gallon drums were encountered. Of the 16 drums, 12 were empty, compressed, or A total of 21 metal 55-gallon drums were encountered between two areas. Of the 21 drums, 17 were empty, compressed, or corroded, and contained no liquids. Also discovered were lengths of pipe, a tire iron, and metal post. Three of the drums were still in tact and partially full with an unknown liquid. Analytical results from characterization sampling will be addressed under a separate and pending (December 2000) project report by IT Corporation, but preliminary results indicate that the drums contain at least a fraction of TCE. A fourth in tact drum contained a blue, wet, powdery substance. Analytical results from characterization sampling of this unknown powdery substance will also be addressed in the IT report on excavation activities. Although analytical results from excavation activities are not available for this Internal Draft Risk Assessment, it is expected that the analytical results will be available and incorporated in the Final Risk Assessment.

The resulting southern lobe TCE plume originating from AFP 4 and possibly other NAS Fort Worth source areas covers approximately 453 acres, 75 of which are on the BRAC property. The down gradient extent (TCE at 5 µg/L) of the plume is within 6 feet of the federal property boundary in WHGLRW015. An off-site well has been installed and analytical results are pending. Two additional offsite wells WHGLRW016 and WHGLRW017 (approximately 20 feet from the boundary show no detectable concentrations of TCE).

Attach available USGS topographic maps and/or aerial or other affected property photographs to this form to depict the affected property and surrounding area. Indicate attachments:

- Topo map Aerial photo Other

2) Identify environmental media known or suspected to contain chemicals of concern (COCs) at the present time. Check all that apply:

- | | | |
|---|---|-----------------------------|
| Known/Suspected COC Location | Based on sampling data? | |
| <input type="checkbox"/> Soil ≤ 5 ft below ground surface | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| <input type="checkbox"/> Soil >5 ft below ground surface | <input type="checkbox"/> Yes | <input type="checkbox"/> No |
| <input checked="" type="checkbox"/> Groundwater | <input checked="" type="checkbox"/> Yes | <input type="checkbox"/> No |
| <input checked="" type="checkbox"/> Surface Water/Sediments | <input checked="" type="checkbox"/> Yes | <input type="checkbox"/> No |

Explain (previously submitted information may be referenced):

Detected chemicals in groundwater, surface water and sediment are identified in Tables 6-1, 6-3, and 6-4, respectively.

Figure 30 TAC §350.77(b) continued

- 3) Provide the information below for the nearest surface water body which has become or has the potential to become impacted from migrating COCs via surface water runoff, air deposition, groundwater seepage, etc. Exclude wastewater treatment facilities and stormwater conveyances/impoundments authorized by permit. Also exclude conveyances, decorative ponds, and those portions of process facilities which are.
- a. Not in contact with surface waters in the State or other surface waters which are ultimately in contact with surface waters in the State; and
 - b. Not consistently or routinely utilized as valuable habitat for natural communities including birds, mammals, reptiles, etc.

The nearest surface water body is 0 feet/miles from the affected property and is named Farmers Branch Creek. The water body is best described as a:

- X freshwater stream: perennial (has water all year)
 intermittent (dries up completely for at least 1 week a year)
 X intermittent with perennial pools
- freshwater swamp/marsh/wetland
 saltwater or brackish marsh/swamp/wetland
 reservoir, lake, or pond; approximate surface acres:
 drainage ditch
 tidal stream bay estuary
 other; specify

Is the water body listed as a State classified segment in Appendix C of the current Texas Surface Water Quality Standards; §§307.1 - 307.10?

Yes Segment # Use Classification:

X No

If the water body is not a State classified segment, identify the first downstream classified segment.

Name: West Fork of the Trinity Below Lake Worth

Segment #: 0806

Use Classification: Contact recreation, high aquatic life use, public water supply

As necessary, provide further description of surface waters in the vicinity of the affected property:

Figure: 30 TAC §350.77(b) continued

PART II. Exclusion Criteria and Supportive Information**Subpart A. Surface Water/Sediment Exposure**

1) Regarding the affected property where a response action is being pursued under the TRRP, have COCs migrated and resulted in a release or imminent threat of release to either surface waters or to their associated sediments via surface water runoff, air deposition, groundwater seepage, etc.? Exclude wastewater treatment facilities and stormwater conveyances/impoundments authorized by permit. Also exclude conveyances, decorative ponds, and those portions of process facilities which are:

- a. Not in contact with surface waters in the State or other surface waters which are ultimately in contact with surface waters in the State; and
- b. Not consistently or routinely utilized as valuable habitat for natural communities including birds, mammals, reptiles, etc.

X Yes No

Explain:

Measured concentrations of volatile and semivolatile chemicals (see Tables 6-3 and 6-4) have been detected in surface water and sediment samples.

If the answer is Yes to Subpart A above, the affected property does not meet the exclusion criteria. However, complete the remainder of Part II to determine if there is a complete and/or significant soil exposure pathway, then complete PART III - Qualitative Summary and Certification. If the answer is No, go to Subpart B.

Soil is not included under this remedial investigation.

Subpart B. Affected Property Setting

In answering "Yes" to the following question, it is understood that the affected property is not attractive to wildlife or livestock, including threatened or endangered species (i.e., the affected property does not serve as valuable habitat, foraging area, or refuge for ecological communities). (May require consultation with wildlife management agencies.)

1) Is the affected property wholly contained within contiguous land characterized by: pavement, buildings, landscaped area, functioning cap, roadways, equipment storage area, manufacturing or process area, other surface cover or structure, or otherwise disturbed ground?

X Yes No

Explain:

Figure: 30 TAC §350.77(b) continued

If the answer to Subpart B above is Yes, the affected property meets the exclusion criteria, assuming the answer to Subpart A was No. Skip Subparts C and D and complete PART III - Qualitative Summary and Certification. If the answer to Subpart B above is No, go to Subpart C

Subpart C. Soil Exposure

- 1) Are COCs which are in the soil of the affected property solely below the first 5 feet beneath ground surface or does the affected property have a physical barrier present to prevent exposure of receptors to COCs in surface soil?

Yes No

Explain:

Soil is not included under this remedial investigation.

If the answer to Subpart C above is Yes, the affected property meets the exclusion criteria, assuming the answer to Subpart A was No. Skip Subpart D and complete PART III - Qualitative Summary and Certification. If the answer to Subpart C above is No, proceed to Subpart D

Subpart D. *De Minimus* Land Area

In answering "Yes" to the question below, it is understood that all of the following conditions apply:

- ❖ The affected property is not known to serve as habitat, foraging area, or refuge to threatened/endangered or otherwise protected species. (Will likely require consultation with wildlife management agencies.)
- ❖ Similar but unimpacted habitat exists within a half-mile radius.
- ❖ The affected property is not known to be located within one-quarter mile of sensitive environmental areas (e.g., rookeries, wildlife management areas, preserves) (Will likely require consultation with wildlife management agencies.)
- ❖ There is no reason to suspect that the COCs associated with the affected property will migrate such that the affected property will become larger than one acre.

- 1) Using human health protective concentration levels as a basis to determine the extent of the COCs, does the affected property consist of one acre or less and does it meet all of the conditions above?

Yes No

Explain how conditions are met/not met:

The surface water body is contained within a golf course area that is highly maintained and does not serve as a viable habitat for threatened/endangered or otherwise protected species.

Figure: 30 TAC §350.77(b) continued

If the answer to Subpart D above is Yes, then no further ecological evaluation is needed at this affected property, assuming the answer to Subpart A was No. Complete PART III - Qualitative Summary and Certification. If the answer to Subpart D above is No, proceed to Tier 2 or 3 or comparable ERA.

PART III. Qualitative Summary and Certification (Complete in all cases.)

Attach a brief statement (not to exceed 1 page) summarizing the information you have provided in this form. This summary should include sufficient information to verify that the affected property meets or does not meet the exclusion criteria. The person should make the initial decision regarding the need for further ecological evaluation (i.e., Tier 2 or 3) based upon the results of this checklist. After review, TNRCC will make a final determination on the need for further assessment. **Note that the person has the continuing obligation to re-enter the ERA process if changing circumstances result in the affected property not meeting the Tier 1 exclusion criteria.**

Completed by, Deborah L. McKean, Ph.D. (Typed/Printed Name)
Senior Toxicologist, IT Corporation (Title)
November 20, 2000 (Date)

I believe that the information submitted is true, accurate, and complete, to the best of my knowledge.

_____ (Typed/Printed Name of Person)
 _____ (Title of Person)
 _____ (Signature of Person)
 _____ (Date Signed)

HydroGeologic, Inc.—NAS Fort Worth JRB, Texas

Figure A.1

Topographic/Aerial Map
Golf Course/BRAC Area



U.S. Air Force Center for
Environmental Excellence

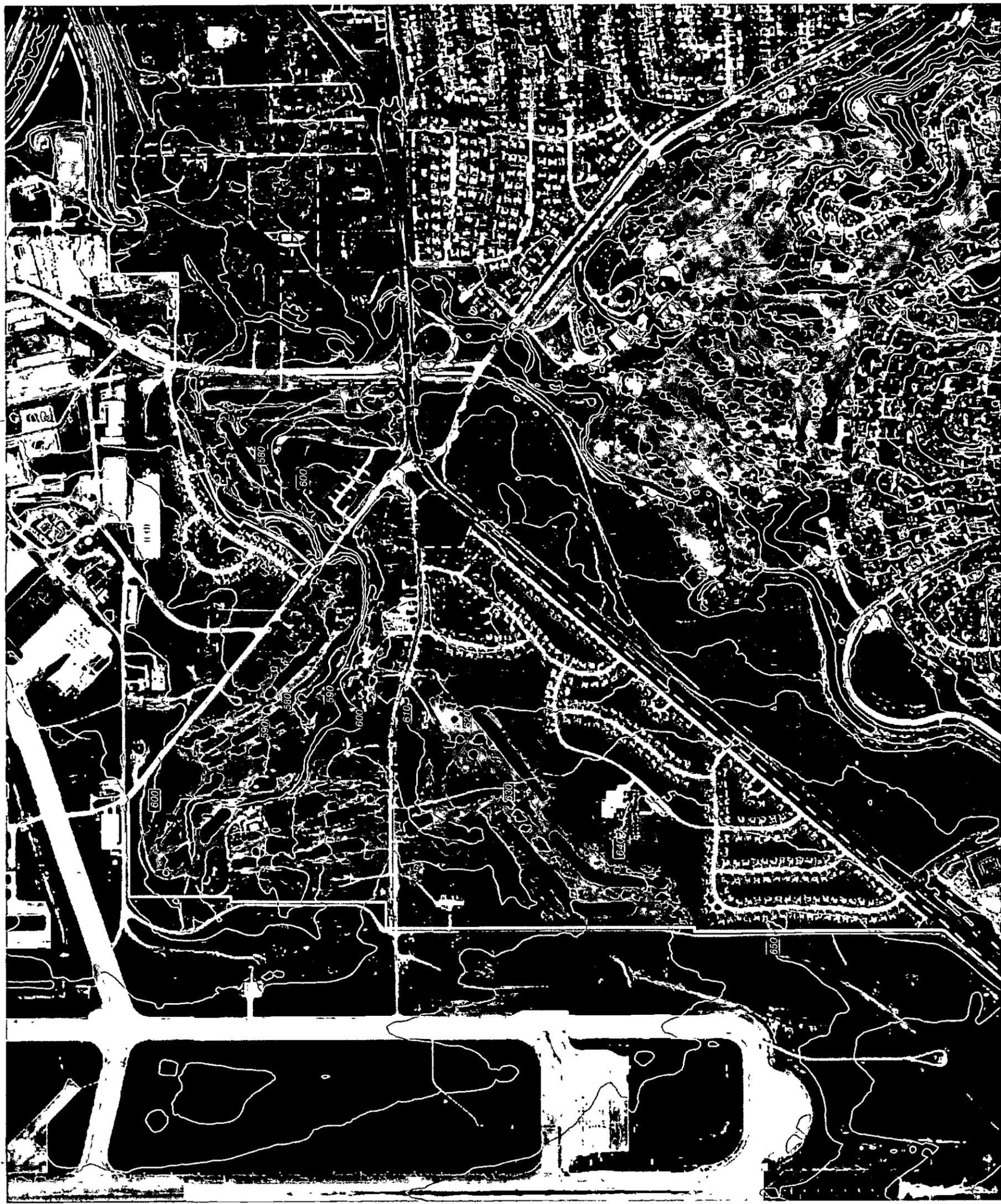
Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- 10' Contours



SCALE IN FEET

Project AFC001-36CA
Filename X:\AFC001\36ca\Report\Golf_Course.apr
Created 11/17/00 ASP
Revised 12/04/00 of
Map Source HydroGeologic, Inc.—GIS Database



**RESPONSES TO COMMENTS
INTERNAL DRAFT
BASELINE RISK ASSESSMENT
FOR THE FOCUSED FEASIBILITY STUDY
FORMER CARSWELL AIR FORCE BASE, TEXAS
DECEMBER 1, 2000**

Introduction:

A technical review was conducted for the subject document submitted by HydroGeoLogic, Inc. The objective of reviewing this report is to assess whether the report satisfies the AFCEE and the Texas Natural Resource Conservation Commission (TNRCC) requirements and format.

Responses to Unitec's Comments

General Comments:

The document as a whole satisfies AFCEE and/or the TNRCC requirements and format. However, the Contractor should re-evaluate all calculations in the report and address the comments below, as the next version of the document is prepared.

Comment 1 *The Contractor should present a rationale and/or justification paragraph explaining why soil, as a media pathway, was eliminated from evaluation.*

Response **In this section of the Base, there are no soil source units. Additional text will be added to Section 2.0: "...receptors may be impacted by groundwater, surface water and/or sediment. These media may have been impacted by up gradient source areas. Soil is not included in this risk assessment because no soil source areas have been identified in this section of the Base."**

Comment 2 *The Contractor should provide a rationale/justification why exposure routes identified in section 6.2.2 (Exposure Pathways) are different from the actual pathways that were evaluated in section 6.9 (Results of the Human Risk Assessment). For example, under the construction worker scenario, it is stated that the exposure routes include: ingestion of groundwater; inhalation of volatiles from groundwater, surface water,*

and sediment; dermal contact with chemicals in the groundwater; incidental ingestion of surface water and sediment; and inhalation of vapors in basements from groundwater contaminants. Yet in the calculations and presentation of risk and hazard indices, the exposure routes presented above were not evaluated. The Contractor should revisit this paragraph and tailor the exposure routes to match calculated exposure routes for each identified subpopulation at the base.

Response

Upon review of Section 2.2 and the presentation of the risk assessment results in Section 4.0, some errors in presentation have been noted that make it appear that all identified exposure pathways were not evaluated. The resident and construction worker are evaluated for groundwater exposure pathways while the recreational user, trespasser and maintenance worker are evaluated for surface water and sediment pathways. Therefore, bullet lists in Section 2.2 will be modified as indicated below:

“Exposure routes for the resident and construction worker include:

- Ingestion of groundwater
- Inhalation of volatiles from groundwater, ~~surface water, and sediment~~
- Dermal contact with chemicals in the groundwater
- ~~Incidental ingestion of surface water and sediment~~
- Inhalation of vapors in basements from groundwater contaminants

Exposure routes for the recreational user, trespasser, and maintenance worker include:

- Incidental ingestion of surface water and sediment
- Dermal contact with chemicals in the surface water and sediment
- ~~Inhalation of volatiles from groundwater, surface water, and sediment~~
- Limited ingestion of fish”

Comment 3

It is recommended that the Contractor use the TNRCC’s soil screening benchmarks, Table 3-4 in the Guidance For Conducting Ecological Risk Assessment Under The Texas Risk Reduction Program to screen sediments COPCs instead of EPA Region 6 PRGs for residential soil (Table 6.20 in the report captioned, “Selection of Contaminants of Potential Concern, Sediment”).

Response The selection of COPCs in the reference table is for human health receptors. Although the use of soil screening criteria is more conservative than necessary, it is a more appropriate screen than the use of ecological endpoints. The selection of COPCs for ecological receptors is a comparison to background and detection frequency only as stated in Section 6.6.1.

Comment 4 *The Contractor should re-examine equations presented on pages -9 through 6-12.*

The intake equation for groundwater ingestion has an extra input parameter (fraction ingested from contaminated source). Delete this term from the equation.

The intake equation for dermal contact, dimensionally, does not yield mg/kg-day as illustrated in the report. A term in the equation is missing. Include this term in the equation.

The D_{event} equation for inorganics is missing from the report. How was the intake dose calculated for inorganics that were identified as chemicals of potential concern? The D_{event} for $ET > t^$ is missing some terms. These terms should be in the denominator for ET in the brackets.*

All calculations that used these equations should be recalculated using the correct equations. Dermal exposure equations should be taken from USEPA document, Dermal Exposure Assessment: Principles and Applications (EPA/600/8-91/001B).

It is stated on page 6-12 that chemical-specific ABSs are presented in the risk assessment spreadsheet in Appendix A. Appendix A in the report does not contain ABS values for chemicals of concern; rather it contains toxicity profiles of chemicals.

It is stated in the report that Johnson and Ettinger Model (EPA, 1989b) was used to derive inhalation screening criteria for detected groundwater constituents and a comparison of screening criteria to detected groundwater constituents determines the need for a more quantitative evaluation of this pathway. Was the Johnson and Ettinger Model simulated? If the model was simulated, the Contractor should present the input and output parameters for model runs. If the pre-calculated model values were used for comparisons, provide the model outputs that were used. Is it the model for Tier 1 or Tier 2? We have looked at the

values that are presented in Table 6.2 and they are different from pre-calculated values for Tier 1 and Tier 2 values.

Response

The parameter “fraction ingested” is intended to provide flexibility for those instances when a receptor is exposed to multiple water sources. Since the parameter value is 1.0 in this risk assessment as shown in Table 6, implying that 100 percent of the drinking water in from one source. No change is necessary.

The equation for dermal contact is merely an algebraic simplification of the equation presented in Dermal Exposure Assessment: Principles and Applications (EPA/600/8-91/001B) and yields the same results as the equation presented in this guidance document. For clarity, however, we have included the complete form of the equations.

Appendix A presents both Toxicity Profiles and spreadsheets. However, the absorbance values can be found in Tables 9 and 11. The sentence in Section 2.6.5 on page 2-11 will be modified to read: “Chemical-specific ABS are presented in Tables 9 and 11.”

The input parameters and results of the Johnson and Ettinger Model will be included in the next draft of the document.

Comment 5

A table that contains the chemical and physical values for chemicals of concern should be presented in the report. Present these values to support statements made in the report.

Responses

A table of the chemical and physical parameters used in the risk calculations will be included in Appendix A in the next draft of the document.

Comment 6

Under the conventional way of presenting baseline risk assessment (as presented in the report), the physiological parameters used in the manipulations of the EPA’s standard equations had an age-adjusted weight value of 59 kilograms for the resident. The PRG for Region 6 and the TNRCC’s equations uses a weight value of 70 kilograms. Note that the body weight is a denominator term. Proportionally, any risk and hazard index calculated in the report were overestimated by 70 kilograms divided by 59 kilograms.

It is always prudent to err on the side that produces more risk using conservative input parameters as presented in the report. However, the

overly conservative nature of the risk calculation renders the results of the risk assessment unrealistic and creates the appearance of increased risk unnecessarily.

Response **During the review of the Risk Assessment Assumptions Document which presented all of the risk methodology and parameter values, Region 6 and TNRCC reviewers requested that the age-adjusted resident with a body weight of 59 Kg be used to evaluate carcinogenic groundwater COPCs while the child resident with a body weight of 15 Kg be used to evaluate noncarcinogenic COPCs. Therefore, the use of 59 Kg for the age-adjusted resident reflects the preference of the Agency reviewers.**

Comment 7 *Under the unconventional way of presenting risk (risk isopleths), generation of risk contour maps, the Contractor should re-evaluate and elaborate on the following:*

Under paragraph 6.8.3 captioned, "Development of Risk Maps," the example states, if a plume of TCE is found in one area at concentrations that range from 1.6×10^{-3} mg/l to 1.6×10^{-1} mg/l, a corresponding risk map will describe the TCE as a risk plume ranging in cancer risk from approximately 1×10^{-6} to 1×10^{-4} for residential receptors. This is accomplished by calculating a unit risk value (risk per mg/l) for each COPC and multiplying that value by every concentration at each point in a concentration plume map for the same COPC." Note the following:

Present and provide equations that were used to generate risk for each point of reference for both the resident and the construction worker risk isopleths.

Were a risk of 1×10^{-6} and a hazard quotient of 1 assigned to 1 mg/l concentration for each COPC? Are these risks per unit mg/l cumulatively summed across exposure routes for the subpopulations? If this is the case (using either Region 6 or TNRCC PRGs) then for any COC with PRGs less than 1, risk and hazard index are overestimated. Vice-versa, any chemical with a PRG more than 1, has an underestimated risk and hazard index.

Elaborate on how the contours were generated. How were the contours lines for 10^{-4} demarcated from contours lines for 10^{-5} ? If the lines were generated by extrapolation, a sample equation should be presented in the report.

- Response** The discussion that follows has also been added to the text under Section 3.3. The calculations requested are presented in Appendix A in spreadsheets entitled “Unit Risk” and “Unit Hazard”. As you will see from the spreadsheets, we do not assign a risk of 1×10^{-6} to a concentration of 1mg/L. Rather, risk and hazard are calculated for each COPC when concentration is fixed at 1 mg/L. This results in the values presented in Tables 13 (Unit Risk Values for Carcinogenic Groundwater COPCs) and 14 (Unit Hazard Values for Noncarcinogenic Groundwater COPCs). Using these “Unit Risk”(risk per mg/L) and “Unit Hazard”(hazard per mg/L) values one can calculate risk and hazard for any groundwater concentration. These values are then used to create the risk and hazard isopleth maps. For example, the unit risk value for vinyl chloride is multiplied by every groundwater concentration measured for vinyl chloride in the study area. The resultant values represent cancer risk (for a particular receptor) at each well where vinyl chloride was detected. These risks are then contoured resulting in a risk isopleth map for vinyl chloride.
- Comment 8** The Contractor should include a discussion of site *characterization and the identification of chemicals of potential ecological concern (essential nutrients, criteria values, frequency of detection and background screening) in the report. This discussion will give a reader an insight into the habitats and ecological resources on and around the site, as well as the nature and extent of chemical contamination of the site.*
- Response** The data requested for chemicals of potential ecological concern are already presented in the document. Tables 3 through 5 present both frequency of detection and background screening (see table footnotes for nutrient information). Tables 19 and 20 also present screening information of the ecological benchmarks.
- Comment 9** *A table showing assessment and measurement endpoints for ecological risk assessment should be presented in the report.*
- Response** A reference has been added in Section 6.2 to assessment and measurement endpoints which are presented as screening criteria in Tables 19 and 20 and toxicity values in Table 21 and 22.
- Comment 10** *The Contractor should provide rationale/justification for not evaluating some ecological receptors. Assessment endpoints, like herbaceous*

vegetation and vertebrates, where eliminated from evaluation without justification.

Response **Since soil is not included in this risk assessment, an evaluation of herbaceous vegetation and ingestion of herbaceous vegetation by vertebrates would not be warranted. Additional text will be added to Section 6.1 that provides this justification: “As stated in Section 2.0, soil is not included in this risk assessment. Therefore, terrestrial plants will not be addressed in this ecological risk assessment.”**

Comment 11 *How were the receptor profiles to represent the site selected? An elaborate discussion should be presented to justify the reason why only the deer mouse and quail were selected for the site.*

Response **As indicated in the second paragraph of 6.3, the deer mouse was selected for the following reasons: 1) the deer mouse has a limited range and will not go far afield, therefore exposure time and pathways will be maximized; 2) there is sufficient toxicological and exposure information available in the literature for comparative and interpretive purposes; and 3) all of the selected species are likely to occur after site remediation (if risk management decisions require it). Additional text will be added to the end of the second paragraph in Section 6.3: “The mouse and quail have been chosen merely as representative species that could be found at the site and become exposed to surface water and sediment. Since much of this area is a golf course (and is expected to remain a golf course), it is not available to a variety of species.”**

Comment 12 *The fonts for tables in the report should be increased. The size of the table fonts should be the same as the text size.*

Response **The fonts will be increased.**

Comment 13 *Tables Appendix. Any column that contains calculations, sample equations, and calculations should be presented at the bottom of the table. If any of the equations are derived from other equations, the methodology and logic that were used to arrive at the final forms should be presented. The appropriate derivations should be performed in a manner that is technically defensible and sufficiently conservative to allow for variation in site conditions, and they should be presented in a fashion that streamlines their review.*

Response All equations used in the calculation of intake, risk, hazard and ecological quotients are presented in Section 2.6 in the methods section..

Specific Comments:

Comment 1 *Page 6-1, Section 1.0. Second paragraph, 7th sentence, eliminate the phrase, “across the BRAC property boundary.”*

Response This sentence reflects requested language from AFCEE. No changes are required.

Comment 2 *Page 6-1, Section 1.0. Reword paragraph three to reflect the fact that exposure point concentration is the chemical concentration at the point of human exposure and that the concentration may be based on sampling data at the exposure point or estimated from a contaminant fate and transport model. Monitoring data generally provide the best estimate of current conditions and models may be necessary to estimate exposure point concentrations where: exposure points are spatially separated from monitoring points, where temporal distribution of data is lacking, and where monitoring data are restricted by the limit of quantification (US EPA, 1989a).*

Response Rather than adding this text to Section 1.0, it is suggested that the above text be added to the second paragraph in Section 6.5 (Exposure Point Concentrations): “ The concentration may be based on sampling data at the exposure point or estimated from a contaminant fate and transport model. Monitoring data generally provide the best estimate of current conditions and models may be necessary to estimate exposure point concentrations where: exposure points are spatially separated from monitoring points, where temporal distribution of data is lacking, and where monitoring data are restricted by the limit of quantification (US EPA, 1989a). Measured groundwater concentrations were used to evaluate current conditions within the aquifers underlying the BRAC property that is being considered for public transfer.”

Comment 3 *Page 6-1, Section 1.0. Revise the first sentence to “For ground water as a media pathway, contour maps were generated to represent risk for the entire BRAC property.”*

Response **The first sentence in the fourth paragraph will be amended as requested.**

Comment 4 *Page 6-1, Section 6.2.1. In the sentence leading to ecological receptors, it states that ground water as a media pathway was not evaluated. Provide rationale/justification why groundwater, as a media pathway, was eliminated*

Response **The sentence will be modified as follows: “...surface water and sediment, but since ecological receptors are not directly exposed to groundwater, groundwater exposures are not included~~-do not include groundwater.~~”**

Comment 5 *Page 6-4, Section 6.2.2. The Contractor should provide rationale/justification for including the exposure pathway routes that were chosen under this subsection. Also, rationale/justification should be given for exposure pathway routes that were eliminated from evaluation under this section.*

Response **Please see response to General Comment 2.**

Comment 6 *Page 6-4, Section 2.2. Third bullet at the bottom of the page states that, “direct contact with the surface water and sediment (plants and aquatic organisms) is an open exposure route for ecological receptors.” Nowhere in the report are plants as an ecological receptor mentioned again or evaluated.*

Last bullet reads, “Ingestion of prey that may bioaccumulate (or bioconcentrate) contaminants.” Please reconcile this statement with your calculation of ecological risk.

Response **The bullet at the bottom of page 2-3 will be modified to read: “Direct contact with the surface water and sediment (aquatic organisms).” The last bullet was originally included in the risk assessment assumptions document before the COPCs were known. Since there are no bioaccumulative compounds detected in surface water (mercury was detected, but below detection limits. Therefore, its relevance as a COPC is uncertain.) or sediment the last bullet will be removed.**

Comment 7 *Page 6-4, Section 6.4. This should read “Chemicals of Concern”. From this page beyond, all COPCs should be changed to COCs. Any chemical encountered during corrective action activities is a chemical of potential concern. After the chemical goes through the elimination process and is retained for risk evaluation it then becomes a chemical of concern (COC).*

Response *As Risk Assessment Guidance for Superfund (EPA540/1-89/002) states: “Chemicals remaining in the quantitative risk assessment based upon this evaluation are referred to in this guidance as ‘chemicals of potential concern.’ “ Therefore, the selection process described in Section 2.4 defines the list of COPCs that remain in the quantitative risk assessment. The risk characterization and uncertainty assessment defines which of the COPCs are truly COCs. Therefore, no changes are necessary.*

Comment 8 *Page 6-6, Section 6.4.2. Second paragraph. It is stated that the Johnson & Ettinger Model was used to derive inhalation screening criteria for detected groundwater constituents. Provide the input and output parameters for this model. Re-simulated results using default values (whether it is Tier I or Tier II) gives different values than the ones presented in the table.*

Response *As stated in response to General Comment 4, the input parameters will be provided in Appendix A of the next draft of the document.*

Comment 9 *Page 6-6, Section 6.4.3. Last sentence. It is stated “Exceptions are made for Class A carcinogens which remain on the COPC list.” Was this exception made for other COCs at the base? Elaborate on this in next sub section, Selection of COPC (COC) Results.*

Response *This is a conservative measure provided discussed in Risk Assessment Guidance for Superfund (EPA540/1-89/002) (page 5-21). It merely permits the inclusion of Class A carcinogens when detected infrequently. Therefore, no changes are necessary.*

Comment 10 *Page 6-7, Section 6.4.4. In the 6th paragraph it is stated that the risk of potential vapor intrusion will be discussed in the risk characterization (Section 3.0). Yet, there is no discussion of such in Section 3.0.*

Response Vapor intrusion is discussed on page 6-16, the Results of the Risk Characterization. Therefore, the sentence on page 6-7 will be changed to read: “The risk of potential vapor intrusion will be discussed in Section 4.0 (Results of the Human Health Risk Assessment).”

Comment 11 *Page 6-7, Section 6.5. Section 6.5, Exposure Point Concentration. Please revise to make the section more specific to this risk assessment. Although Table 1 contains a column that shows statistically manipulated 95th UCL of some chemicals, the maximum detected concentrations were used in most of the evaluations in the report.*

Response A comparison is made between the maximum detected concentration and the calculated UCL. If the maximum concentration is less than the calculated UCL, the maximum concentration is chosen as the exposure point concentration. It is true, that for many COPCs in surface water and sediment in this risk assessment, the maximum concentration is chosen as the exposure point concentration. However, since some instances remain where the UCL is chosen, the text should remain that describes its calculation and use. For clarity, a footnote will be added to Table 1 (Groundwater COPCs) that states All detected concentrations at all monitoring well locations are used as exposure point concentrations in the risk assessment.

Comment 12 *Page 6-12, Section 6.6.1. It is recommended that presentation of the D_{event} equation and the intake equation be reversed in order, for easy cross-reference.*

Response It would seem more appropriate to present the variable, and then define the calculation of the variable. Again, if *Risk Assessment Guidance for Superfund* (Exhibits 6-11 through 6-20) is used as a guide, the parameter “AT”, averaging time, is presented as an equation variable and then the equation used to calculate the parameter follows. Therefore, no changes are necessary.

Comment 13 *Page 6-12, Section 6.6.5. The last sentence after the equation states that chemical-specific ABS are presented in the risk assessment spreadsheet in Appendix A. Appendix A contains toxicity profiles of COCs and not ABS of COCs.*

- Response** Please see response to General Comment 4 which states that the sentence in Section 2.6.5 on page 2-11 will be modified to read: “Chemical-specific ABS are presented in Tables 9 and 11.”
- Comment 14** *Page 6-13, Section 6.8. In the fifth and seventh sentences, change the word “projected” to “calculated.”*
- Response** The change will be made as requested.
- Comment 15** *Page 6-14, Section 6.8.1 and 6.8.2. It is recommended that presentation of cancer risk, hazard quotient and total cancer risk and hazard index equations be reversed in order, for easy cross-reference.*
- Response** Please see response to Specific Comment 12.
- Comment 16** *Page 6.15, Section 6.8.3. First paragraph. As stated in the general comments, equations and written explanations should be provided to illustrate how the risk isopleths were generated. Third sentence. Change “form” to “from.”*
- Response** Please see response to General Comment 7. “Form” will be changed to “from” in the third line of Section 3.3. A search of “form” was also performed and additional changes were made where necessary.
- Comment 17** *Page 6-15, Section 6.9. In the last paragraph using the age-adjusted resident parameters in the standard EPA intake equations to calculate intake dose is too stringent. That is why half the detection limit of arsenic (0.25 mg/l) is yielding a risk in excess of 1×10^4 . Revise this statement so that it is in sync with new calculated values*
- Response** In the next draft of the risk assessment, risk and hazard isopleth maps (and the text describing their development) will be modified. Wells where COPCs are not detected will show no risk or hazard.
- Comment 18** *Page 6-16, Section 6.9. Provide an explanation why, for groundwater as a media pathway, only the adult resident was evaluated for carcinogenic chemicals and for non-carcinogens, only the child resident was evaluated. Again, present equations and written explanations to illustrate how hazard index for each point of reference was calculated.*

- Response** **The following text was added as the second sentence under adult residents Adult residents provide the most conservative receptor for the evaluation of carcinogenic effects from groundwater exposures. The rationale is already provided in the text. Under the subheading for “Child Resident” the text states that child residents provide a more conservative receptor for the evaluation of noncarcinogenic effects from groundwater exposures. The original intent was to limit the number of maps produced by presenting one residential exposure scenario. The Agencies requested that the more conservative residential receptor be presented for carcinogenic and noncarcinogenic endpoints. Therefore, they requested that adult residents be chosen to represent carcinogenic effects while child residents be chosen to represent noncarcinogenic effects.**
- Comment 19** *Page 6-17, Section 6.9. Risk and hazard quotients should be presented in one significant figure. Change all hazard indexes on this page to scientific figures.*
- Response** **According to EPA’s newest Risk Assessment Guidance for RAGS Part D, risk and hazard is presented in two significant figures. Therefore, no changes are necessary, unless otherwise instructed.**
- Comment 20** *Page 6-22, Section 6.11. Second paragraph, 2nd sentence, insert “and/or physical” after the word chemical.*
- Response** **The change will be made as requested.**
- Comment 21** *Page 6-24, Section 6.11.3. First paragraph, last sentence, provide reference table at the end of the sentence.*
- Response** **The reference (EPA, 2000b) will be added to the text.**
- Comment 22** *Page 6-24,, Section 6.11.3. Receptor Profiles. An elaborate explanation should be provided as to why only the Deer Mouse and Quail were the only chosen receptor profiles to represent the Base.*
- Response** **Please see response to General Comment 11.**

Comment 23 *Page 6-26, Section 6.11.5.1. Reconcile the units given on page 6-26 for NIR with units presented in Table 6.18. If NIR is the normalized water ingestion rate, fraction of body weight consumed as water per unit time, then the units presented in the Table are the correct units.*

The units presented in the Table, if inserted in the equation as presented do not yield a unit of mg/kg-day for the average daily dose of surface water.

The conversion factor in the equation has units of volume over weight (L/kg). Present the value of this conversion factor and its source.

Response **The inconsistency is corrected merely with the understanding that the density of water is 1 g/ml. Therefore, the units in Table 18 for water intake rate will be changed to g/g-day.**

Comment 24 *Page 6-28, Section 6.11.6.2. Re-evaluate Tables 6.21 and 6.22, because one of the equations used to calculate intake dose is wrong. After ecological hazard quotients are re-calculated with correct equations, the whole paragraph should be re-written.*

Response **Please see response to specific comment 23.**

Comment 25 *The fourth sentence in the first paragraph states that water consumption rates were estimated by allometric equations based on body weight when empirical data were not available. These equations were not used in this baseline risk assessment report; therefore, it is suggested that this sentence be rephrased or deleted from the paragraph.*

Response **The sentence will be deleted.**

Comment 26 *Page 6-29, Section 6.12. This section needs to be re-written to reflect new risk and hazard indexes after correct equations are used to re-calculate risk and hazard indexes for the base. A recommendation section should follow the risk assessment conclusion section.*

Response **Since the risk and hazard estimates are correct, this section does not require any modification. See response to previous comments. A summary section has already been included, however,**

recommendations will be made as part of the risk management decision after the feasibility study has been completed.

Comment 27 *Page 6-31, References. It is recommended that the Contractor separate citation No.4 from citation No.5. Also, citation No. 7 should be separated from No.8.*

Response **The references will be modified as requested.**

Comment 28 *Table. Exposure route of this exposure pathway does not include ground water ingestion, so why are the COCs being screened against toxicity/concentration based on EPA Region 6 PRGs for tap water? COCs for this exposure pathway should not be screened. All volatile and semi-volatile compounds should be carried through risk and hazard calculations.*

Response **The following discussion has been added as the last paragraph in section 2.4.2. It should also be noted that, since the selection of surface water COCs is for human receptors, and the exposure pathways for surface water are consistent with groundwater exposure pathways, tap water PRGs provide a conservative toxicity/concentration screen. Screening against tap water PRGs are further justified since the screening is merely intended to remove those chemicals that would not contribute significantly to risk or hazard. The screen is applied equally to metals, volatiles and semivolatiles. However, all detected volatiles and semivolatiles are carried through the ecological risk assessment.**

Comment 29 *Table 6.9. Under this table it is stated that the equation used to derive the adjusted dermal RfD is presented in the text. Nowhere in the document is the equation presented.*

Response **The footnote 2 in Table 9 will be modified to state: “Dermal RfD = Oral RfD x Oral Adjustment Factor”.**

Comment 30 *Table 6.12. Provide source of adjustment values. Assign units to the adjustment values and present a sample equation indicating how they are being applied to arrive at the values for Inhalation Cancer Slope Factors.*

Response Footnotes will be added to Tables, 9, 10, 11 and 12 that states: “Inhalation Slope Factor = Unit Risk x Adjustment Factor”.

Comment 31 *Tables 6.13 and 6.14. Present sample equations for each column. The equations should be presented in a step-wise fashion.*

Response A footnote will be added to both tables indicating that equations used to calculate unit risk and unit hazard values are presented in Section 2.6 and spreadsheets providing chemical-specific parameter values are provided in Appendix A.

Responses to Doris A. Anders’ (AFCEE) Comments

General Comments:

Overall, this document is excellent; it is clearly and concisely written, and incorporates all the necessary elements in sufficient detail. I have no suggestions for changes in the text.

As Lynn Morgan, Don Ficklen, and I have discussed, the risk isopleth maps should show “0” for NDs.

Specific Comments:

Comment 1 *Page. 6-3, Section 6.2.1, bullet at bottom of page: Good, logical 'current and future conditions' Recreational User description;*

Response **Comment noted.**

Comment 2 *Page. 6-4, Section. 6.2.1, 'Trespasser' bullet: There is also a 'professional golf ball retriever' -- a person who waits until the course is closed and then goes diving to recover golf balls in the water hazard, which he/she sells to driving ranges, etc. Would we also want to include this category in the Trespasser scenario? The dermal exposure to both water and sediments would increase our risk calculations. I would suggest that we wait and see if any of the regulators suggest such a scenario; if not, then we don't include.*

Response **Comment noted.**

Comment 3 Page. 6-15 - 6-18, Section. 6.9: Concur with results of the HHRA; well done! Should we have to include the 'golf ball retriever' scenario, the Trespasser (and Surface Water, Sediment) risk would increase fairly substantially.

Response Comment noted.

Comment 4 *Good write-up on eco risk.*

Response Comment noted.

Comment 5 *Page. 6-30, Sec. 6.12 last 2 paragraphs: Concur with conclusion that remedial action for GW is warranted; no remedial action for surface water and sediment.*

Response Comment noted.

Responses to Gregory Harvey's Comments

General Comments:

Comment 1 *I am unaware of any routine uses of arsenic in the building of aircraft or the operation of a flightline. I found it interesting that arsenic was a major risk driver here in light of the fact that the former Carswell Air Force Base was at one time a cotton plantation. According to Ronald Eisler of the USGS Biological Resource Division agricultural applications of arsenic provide the largest anthropogenic source to the environment. Another potential source of arsenic is in the use of crab grass killers. A recent article in Environmental Science and Technology 1 Sept, 2000 page 376 states the use of arsenical herbicides has proven to be problematic in Denver, Colorado. Since this site has been a golf course for decades has anyone ruled out the use of these agents here being used to control crabgrass or other weeds? We need to know the background levels for arsenic and lead are for this area. Lack of this background information is a major data gap in addressing uncertainties within this risk assessment. Has anyone contacted the National Resource Conservation Service (former Soil Conservation folks) or the USGS to obtain this info?*

Response **The arsenic maps presented in the latest version of the risk assessment showed arsenic as one of the major risk drivers. In creating the maps, however, if arsenic was not detected a value of ½ the detection was used to calculate risk. This approach was problematic in that ½ the detection limit lead to relatively high risk values. Therefore, based on frequency of detections, the arsenic maps have been redone and if arsenic is not detected a zero risk value is shown.**

Comment 2 *The statement on page 115 in Appendix A concerning the lack of phytotoxicity data on trichloroethylene in the literature is problematic. The Carswell Golf Course site has been extensively studied for the last five years by the USAF, USFS, USGS, and EPA with regard to the phytoremediation of TCE and it's degradation products. Results from numerous studies conducted at this site have been presented and published in peer reviewed journals. TCE and it's degradation products do not phytoaccumulate and it appears that most plants have the enzymatic capability of degrading TCE and it's daughter products. If you would like copies of these studies please contact me.*

Response **The paragraph on page B-118 has been reworded to the following:**

Data on measured concentrations of trichloroethene in plants have been reported in grain-based foods, which range from 0.77 to 2.7 g/kg (Heikes and Hopper, 1986, as cited in Howard, 1990).

Please also consider this response a request to obtain copies of the relevant reports that you mention.

Comment 3 *It should also be stressed somewhere in this risk assessment that TCE, DCE, and Vinyl Chloride do not bioaccumulate in terrestrial or aquatic animals. The bioavailability of compounds that do bioaccumulate should also be addressed since many of these compounds have been there for decades.*

Response **The following sentence has been added to the end of the first bullet in Section 7.0.**

It should also be noted that none of these volatile organics are known to bioaccumulate in terrestrial or aquatic animals.

Specific Comments

Comment 1 *Page 6-1, last paragraph, third sentence. Recommend changing wording to state that this risk characterization is new or innovative. Current statement that this approach is not conventional may be problematic with regulators.*

Response **Wording has been changed to innovative.**

Comment 2 *Page 6-3, Section 6.2.1. State where these deep wells are and in what aquifer. Expand discussion of potential future use of groundwater at all depths stating that it is unlikely with two uncontaminated lower aquifers and Lake Worth nearby that the contaminated shallow alluvial aquifer will ever be used as a source of drinking water.*

Response **The following text will be added to Section 2.1:**

“It should be pointed out that it is unlikely with two prolific lower aquifers (Paluxy and Glen Rose) and Lake Worth nearby that the contaminated shallow alluvial aquifer will ever be used as a source of drinking water.

A water well survey was purchased to identify all existing deep water wells. The coordinates given in the survey do not appear to be correct. A field survey or literature search will be performed to include this information in the Final Report.

Comment 3 *Page 6-3,4. State while limited fishing in Farmer’s Branch Creek is possible it is highly unlikely considering Lake Worth is so close by. I have yet to see anyone fishing in this creek over the past 8 years.*

Response **Statement has been incorporated as requested.**

Comment 4 *Page 6-4 Section 6.2.2 Exposure Pathways. Are there even any basements in this area? Most homes and buildings have been built on concrete slabs due to the out cropping of bedrock.*

Response **Comment noted. The basement scenario was performed as a request from several previous reviewers, in order to ensure that potential future exposure pathways are considered.**

Comment 5 *Page 6-23. We should state that the biggest risk to aquatic vertebrates is when Farmer's Branch Creek dries up like it has during the recent record droughts that North Central Texas has been experiencing.*

Response **Statement has been incorporated as requested.**

Comment 6 *Page 6-29, 6.12. We need to qualify statement concerning potential future residential cancer with groundwater exposures i.e. reminding the reader/ reviewer that this is only possible if the shallow contaminated aquifer not currently in use is used as a source of drinking water.*

Response **Statement has been incorporated as requested.**

Comment 7 *Appendix A, page 17. Update discussion of arsenic and it's source and uses to reflect comments made above with regard to agriculture and weed killers.*

Response **The following text has been added to the very beginning of the arsenic discussion:**

Most arsenic enters water supplies either from natural deposits in the earth or from industrial and agricultural pollution. Arsenic is a natural element of the earth's crust. It is used in industry and agriculture, and for other purposes. It also is a byproduct of copper smelting, mining and coal burning. U.S. industries release thousands of pounds of arsenic into the environment every year.

Comment 8 *Appendix A, page 44. Reword statement concerning erythropoiesis in the first paragraph. Erythropoiesis is the formation of red blood cells. Vertebrates not forming red blood cells are suffering from aplastic anemia or pure red cell aplasia both conditions are fatal if not corrected. I never thought I would see Quebec Beer Drinker's Cardiomyopathy mentioned again after graduate school but it appears I am wrong. Cobalt was used to enhance the quality of the beer foam head however with less than optimum results.*

Response **In the first sentence on the page "aplastic anemia" has been substituted for "erythropoiesis".**



DEPARTMENT OF THE AIR FORCE
HEADQUARTERS AIR FORCE CENTER FOR ENVIRONMENTAL EXCELLENCE
BROOKS AIR FORCE BASE TEXAS

661 51

10 January 2001

MEMORANDUM FOR RUBEN MOYA (EPA REGION 6)

FROM: Mr. Don Ficklen
HQ AFCEE/ERD
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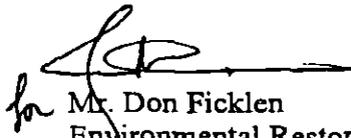
SUBJECT: Draft Final Baseline Risk Assessment
Former Carswell AFB

Dear Mr. Moya,

One copy of the Draft Final Baseline Risk Assessment is attached for your review. Please feel free to distribute the report to any of the EPA risk assessors who have been involved in the development of the report (Cheryl Overstreet and/ or Jon Rauscher) I assume that you will be submitting one set of comments collectively from Gary Miller, the above mentioned risk assessors, and yourself. These comments, along with the comments from the TNRCC reviewers can be either submitted in writing, or if you prefer, a conference call can be held among all the involved parties to answer any questions or comments.

Should you have any questions regarding this report, please contact me at (210) 536-5290.

Sincerely,



for Mr. Don Ficklen
Environmental Restoration Team Chief
AFCEE/ERD



Printed on Recycled Paper

cc:

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LIST OF ACRONYMS AND ABBREVIATIONS

AFB	Air Force Base
BRAC	Base Realignment and Closure
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
<i>cis</i> -1,2-DCE	<i>cis</i> -1,2-dichloroethene
cm	centimeters
COPCs	chemicals of potential concern
COPEC	constituents of potential ecological concern
CSM	conceptual site model
1,4- DCB	1,4-dichlorobenzene
1,1-DCE	1,1-dichloroethene
EPA	Environmental Protection Agency
ERA	ecological risk assessment
EQs	ecological hazard quotients
g	gram
H ₀	null hypothesis
HI	Hazard Index
HQ	Hazard Quotient
J	qualified as estimated data
JRB	Joint Reserve Base
kg	kilograms
L	Liter
LD ₅₀	lethal dose for 50 %
LOAEL	lowest observed adverse effect level
m	meters
mg	milligrams
NAS	Naval Air Station
NOAEL	no observed adverse effect level
PCB	polychlorinated biphenyl
PCE	tetrachloroethene

LIST OF ACRONYMS AND ABBREVIATIONS (continued)

PCLs	protective concentration levels
R	classified as rejected data
RfDs	reference doses
RME	reasonable maximum exposure
SQL	sample quantitation limit
SFs	slope factors
TCE	trichloroethylene
TNRCC	Texas Natural Resource Conservation Commission
U	qualified below detection limits
μ g	micrograms
UCL	Upper Confidence Level
UJ	qualified as estimated data

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1.0 INTRODUCTION

This risk assessment provides an evaluation of federal property located adjacent to the Naval Air Station (NAS) Fort Worth Joint Reserve Base (JRB) on Former Carswell Air Force Base (AFB) property. This property is approximately 300 acres, and it includes the Carswell Golf Course. The property is being evaluated for transfer under the Base Realignment and Closure (BRAC) program. From this point forward in the document, the area will be referred to as the “BRAC property”. This document summarizes the approach used to perform a human health and ecological risk assessment for the BRAC property. The risk assessment has been conducted to support the Focused Feasibility Study.

Investigations of contaminant source areas at NAS Fort Worth JRB revealed the presence of groundwater contaminants in varying concentrations throughout the area. These contaminants, primarily volatile organic compounds (i.e., predominantly trichloroethylene (TCE) and its degradation products) occur as definable plumes. Because of movement of groundwater, and the physio-chemical properties of the individual contaminants, contaminants may be transported from one source area through others, commingling contaminants and finally moving into remote portions of the BRAC property or across the BRAC property boundary. This risk assessment examines the potential for risks posed to human health and the environment by exposure to the contaminants in groundwater, surface water and sediment. The risk assessment incorporates previous groundwater, surface water, and sediment characterization efforts to allow for the development, evaluation, and selection of appropriate remedial actions for the BRAC property.

Human health risk from exposure to contaminated groundwater is evaluated quantitatively. Traditionally, the groundwater exposure point concentration is estimated as the maximum concentration for any constituent. However, in situations involving large areas and multiple chemicals of concern, the maximum detected constituent concentration associated with one chemical may be at a different location from the maximum detected constituent concentration associated with a second chemical. Assuming equivalent exposure for both chemicals to any receptor would, therefore, be inaccurate and overly conservative. Since current and future exposures to groundwater occur at particular locations, it would be helpful to estimate risk at all possible locations within the BRAC property based on land use scenarios. For these purposes, a risk assessment that evaluates risk at multiple locations for multiple contaminants of concern is more realistic.

For groundwater as a media pathway, contour maps were generated to represent risk for the entire BRAC property. These maps represent total incremental cancer risk and noncancer hazard for all chemicals of potential concern (COPCs) as a function of contaminant concentration and location (i.e., risk isopleths). This approach to risk characterization is innovative. In most risk assessments, risk is presented for a discrete area in a tabular format. This approach, however, does not present the spatial distribution of risk on a continuous basis. Instead, statistical methods are used to develop conservative risk numbers that are representative of a large discrete area. The risk characterization approach provides a

mechanism for presenting quantitative estimates of carcinogenic risk and noncarcinogenic hazard in a fashion that can be easily communicated to all stakeholders and allows the spatial distribution of risk to be presented at every location within the BRAC property.

Surface water and sediment constituent concentrations are also evaluated to assess potential human health and ecological risk using more traditional methods. Human health risk is evaluated through the estimation of average surface water and sediment concentrations from which, numerical risk and hazard estimates are derived. For ecological risk, surface water and sediment constituent concentrations are evaluated by a tiered approach (Texas Natural Resource Conservation Commission [TNRCC], 2000). The need for a more rigorous ecological evaluation will be based on the results of the initial evaluation.

The risk assessment is intended to reflect appropriate guidance provided by U.S. Environmental Protection Agency (EPA) (1989a, 1995c, and 1998a) for human health risk assessment and guidance provided by TNRCC (2000) for ecological risk assessment. EPA's Part D risk assessment guidance (1998a) provides standardized tables that present data and calculated values used in the risk assessment. Part D guidance is used to present the majority of the risk assessment. However, since the groundwater risk characterization takes the form of risk isopleth maps rather than single numerical estimates of risk, the groundwater risk characterization does not specifically conform to Part D risk characterization formats.

The risk assessment consists of the following elements:

- Conceptual Site Model (CSM) for both human and ecological health;
- Data Compilation and Evaluation describing methodologies used to summarize data used in this evaluation;
- Summary of COPCs;
- Exposure Assessment which includes a summary of the unit risk values used in the risk characterization;
- Toxicity Assessment used to evaluate carcinogenic risk and noncarcinogenic hazard from groundwater, surface water and sediment exposures. The Toxicity Assessment includes both carcinogenic and noncarcinogenic toxicity values as well as toxicity profiles for potential human health and ecological receptors; and
- Risk Characterization and an Evaluation of Uncertainties in the exposure, toxicity, and risk estimates.

2.0 CONCEPTUAL SITE MODEL

The conceptual site model for the risk assessment is developed to provide the basis for identifying and evaluating the potential risks to human health in the baseline risk assessment. The conceptual model facilitates consistent and comprehensive evaluation of risks by creating a framework for identifying the paths by which potential human and ecological receptors may be impacted by groundwater, surface water, and/or sediment. The elements necessary to construct a complete exposure pathway and develop the conceptual model include:

- Land use scenarios and potential populations of concern
- COPCs and their sources
- Release mechanisms
- Transport pathways
- Exposure pathway scenarios
- Potential receptors (both current and future)

2.1 LAND USE SCENARIOS AND POTENTIAL POPULATIONS OF CONCERN

Land use in the BRAC property ranges from industrial to residential. Although current groundwater supplies in the vicinity of the NAS Fort Worth JRB originate from deep wells, this risk assessment addresses potential future use of groundwater at all depths beneath the site. It should be pointed out that it is unlikely with two prolific lower aquifers (Paluxy and Glen Rose) and Lake Worth nearby that the contaminated shallow alluvial aquifer will ever be used as a source of drinking water.

Also included is an exposure scenario that evaluates current conditions where shallow groundwater is not available for residential use and the only potential exposure to contaminated groundwater would be during construction activities. All receptors are evaluated for the reasonable maximum exposure (RME).

The potential human groundwater receptor exposure scenarios include:

- Resident - This exposure assumes that adults and children reside within the BRAC property and that these receptors obtain all household water from on-site supply wells.
- Construction Worker - This exposure assumes that a construction worker is exposed through dermal contact, inhalation of volatiles, and incidental ingestion while engaged in construction activities in the BRAC property.

The potential human surface water and sediment receptor exposure scenarios include:

- Recreational User - This exposure assumes that adults frequent the BRAC property and occasionally come in contact with surface water and sediment. Since a portion of the property will remain a golf course, a typical exposure

would be a frequent golfer retrieving golf balls. The stream (Farmer's Branch) in this area is ephemeral and does not provide a habitat that supports sport fishing. As a conservative measure, however, surface water will be evaluated assuming some limited fishing may be possible, although it is highly unlikely considering the close proximity of Lake Worth.

- Trespasser – This receptor is a young adult that visits the area intermittently. This receptor is exposed to surface water and sediment while exploring and playing in the surface water bodies.
- Site Maintenance Worker - This receptor is an adult that works as a groundskeeper and occasionally performs maintenance activities in the surface water bodies and becomes exposed to surface water and sediment.

The receptor exposure scenarios included in the ecological risk assessment include only exposures to surface water and sediment, but since ecological receptors are not directly exposed to groundwater, groundwater receptors are not included.

- Ecological Receptors – Ecological receptors include hydric and aquatic organisms, plants, and wildlife that live in or use the habitat provided in the BRAC property.

2.2 EXPOSURE PATHWAYS

Receptors may be impacted by groundwater, surface water and/or sediment. These media may have been impacted by up gradient source areas. Soil is not included in this risk assessment because no soil source areas have been identified in this section of the Base. Exposure pathways relevant to human and ecological exposures to groundwater, surface water and sediment are listed below.

Exposure pathways relevant to human and ecological exposures to groundwater, surface water and sediment are listed below.

Exposure routes for the resident and construction worker include:

- Ingestion of groundwater
- Inhalation of volatiles from groundwater
- Dermal contact with chemicals in the groundwater
- Inhalation of vapors in basements from groundwater contaminants

Exposure routes for the recreational user, trespasser, and maintenance worker include:

- Incidental ingestion of surface water and sediment
- Dermal contact with chemicals in the surface water and sediment
- Limited ingestion of fish

Exposure routes for ecological receptors:

- Direct contact with the surface water and sediment (aquatic organisms)
- Ingestion of food from the surface water and sediment
- Ingestion of prey that may bioaccumulate (or bioconcentrate) contaminants
- Ingestion of surface water

2.3 DATA COMPILATION AND EVALUATION

Historical groundwater, surface water and sediment data were compiled and summarized from previous investigations. Groundwater quality data collected during the Data Gaps Investigation (HGL, 2000), conducted as part of the FFS, were integrated with historical data from July 1997 through April 2000 to develop the COPC list. The full data set was used to provide the most conservative list of COPCs. Risk contour maps were developed using 1999 data only (to represent the current risks at the site) as described below. Surface water and sediment data were statistically summarized to derive exposure point concentrations used in both the human health and ecological risk assessment.

Only data validated to EPA Level III were used in this risk assessment. Data may be classified as rejected (R), qualified as estimated (J or UJ), or qualified below detection limits (U). Rejected data was not in the risk assessment. J-qualified data represent estimated values, but are treated in the same manner as unqualified data and will be included in the exposure estimates. Methods used to include U-qualified data are discussed in Section 2.5.

2.4 CHEMICALS OF POTENTIAL CONCERN

The process for selecting COPCs for groundwater, surface water and sediment in the BRAC property is defined below. The selection process for COPCs for the human health risk assessment includes a comparison to background, a risk-based concentration screen and an evaluation of frequency of detection. The selection process for COPCs for the ecological risk assessment includes only a comparison to background and an evaluation of frequency of detection.

2.4.1 Comparison of Site-Related Data to Background Data

The initial selection of inorganic constituents for evaluation in the risk assessment is based on a statistical comparison of site-related data to background data. A statistical representation of background concentrations is calculated for all inorganic constituents (see Section 2.5 that describes statistical methods for the derivation of the 95% Upper Confidence Level [UCL] which will be used to describe the representative concentration of background constituents). The initial list of COPCs is based on a comparison of detected analyte concentrations to representative background concentrations. Inorganic constituents are considered to be similar to background concentrations if the UCL concentration of the detected site constituent is less than or equal to the background UCL for the selected inorganic constituent. Those inorganic compounds that are within background levels are eliminated as COPCs.

2.4.2 Risk-Based Concentration Screen

After screening out chemicals that are not COPCs on the basis of background comparisons, the remaining chemicals are screened against risk-based concentrations. The purpose of this screening is to make the human health baseline risk assessment process more efficient by focusing on the dominant chemicals and routes of exposure at the earliest feasible stage.

The risk-based concentration screen includes the following steps:

- The maximum concentration is identified for each chemical detected in each medium.
- The maximum concentration is compared to the Region 6 Media-Specific Screening Criteria (EPA, 2000).
- If a specific chemical exceeds the risk-based concentration for that medium, the chemical is retained for the risk assessment for all routes of exposure involving that medium.
- If a specific chemical does not exceed its risk-based concentration for any medium, the chemical is eliminated from the COPC list.

In addition to the concentration/toxicity screen described above, additional screens are applied to the groundwater data to evaluate the potential for significant vapor intrusion to future residential basements. The Johnson and Ettinger Model (EPA, 1989b) was used to derive inhalation screening criteria for detected groundwater constituents. Input parameters for the Johnson and Ettinger Model are presented as Appendix A. A comparison of screening criteria to detected groundwater constituents determines the need for a more quantitative evaluation of this pathway.

In addition, surface-water constituent concentrations are compared to TNRCC screening criteria for non-sustainable fisheries (TNRCC, 2000). This comparison was used to determine the need for a more quantitative evaluation of this pathway. It should also be noted that, since the selection of surface water COPCs is for human receptors, and the exposure pathways for surface water are consistent with groundwater exposure pathways, tap water PRGs provide a conservative toxicity/concentration screen. Screening against tap water PRGs are further justified since the screening is merely intended to remove those chemicals that would not contribute significantly to risk or hazard. The screen is applied equally to metals, volatiles and semivolatiles. However, all detected volatiles and semivolatiles are carried through the ecological risk assessment.

2.4.3 Detection Frequency

In accordance with EPA guidance (EPA, 1989a), consideration of detection frequency was applied in the selection of COPCs. Chemicals that are detected infrequently (i.e., in less than 5 percent of 20 or more samples) at less than five times the reporting limit were eliminated from the COPC list. Exceptions are made for Class A carcinogens which remain on the COPC list.

2.4.4 Selection of COPC Results

Table 1 presents the COPCs for groundwater; these chemicals include inorganics; arsenic and chromium, and volatile organics; 1,1-dichloroethene (1,1-DCE), 1,4-dichlorobenzene (1,4-DCB), benzene, chloroform, cis-1,2-dichloroethene (cis-1,2-DCE), tetrachloroethene (PCE), trichloroethene (TCE) and vinyl chloride; a semivolatile organic; bis(2-ethylhexyl)phthalate; and an herbicide; 000-triethylphosphorothioate.

The groundwater COPCs were further evaluated to determine if any present a potential to contribute to vapor intrusion to future residential basements. Table 2 presents a comparison of screening criteria for vapor intrusion calculated using the Johnson and Ettinger Model (EPA, 1989b). Only TCE and vinyl chloride present such a potential. The risk of potential vapor intrusion will be discussed in Section 4.0 (Results of the Human Health Risk Assessment).

Surface water COPCs are presented in Table 3. Surface water COPCs for the human health risk assessment include inorganics; aluminum, antimony, cobalt, copper, iron, lead, magnesium, manganese, vanadium, and zinc; and volatile organics; cis, 1-2, DCE, TCE, and vinyl chloride; and the semivolatile, bis(2-ethylhexyl)phthalate. Surface water COPCs were also evaluated for their potential to cause adverse effects if this water body is evaluated a non-sustainable fishery (TNRCC, 2000). Table 4 presents a comparison of surface water COPCs to those criteria. None of the surface water COPC concentrations exceed these criteria. This pathway will, therefore, not be evaluated further.

Sediment COPCs are presented in Table 5. Sediment COPCs for the human health risk assessment include inorganics; antimony, arsenic, barium, iron, magnesium, manganese,

nickel, vanadium and zinc; and semivolatile organics; benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, chrysene, and indeno(1,2,3-cd)pyrene.

2.5 EXPOSURE POINT CONCENTRATIONS

The exposure point concentration is the concentration of a COPC in an exposure medium that may be contacted by a real or hypothetical receptor. Determination of the exposure point concentration depends on factors such as:

- Availability of data
- Amount of data suitable for statistical analysis
- Location of the potential receptor

The concentration may be based on sampling data at the exposure point or estimated from a contaminant fate and transport model. Monitoring data generally provide the best estimate of current conditions and models may be necessary to estimate exposure point concentrations where: exposure points are spatially separated from monitoring points, where temporal distribution of data is lacking, and where monitoring data are restricted by the limit of quantification (US EPA, 1989a). Measured groundwater concentrations were used to evaluate current conditions within the aquifers underlying the BRAC property that is being considered for public transfer.

Historical surface water and sediment data were statistically evaluated to determine conservative constituent concentrations used in the risk assessment. In Superfund risk assessments, the concentration term in the intake equation is an estimate of the arithmetic average concentration for a contaminant based on a set of site sampling results (EPA 1989a and 1992d). Because of the uncertainty associated with estimating the true average concentration at a site, the UCL of the arithmetic mean will be used in the risk assessment if sufficient data are available. If the data are limited, the maximum detected concentration will be used as the exposure point concentration. The UCL provides reasonable confidence that the true site average will not be underestimated.

The EPA has determined that most large environmental contaminant data sets from soil sampling are lognormally distributed rather than normally distributed (EPA, 1992d).

The W test developed by Shapiro and Wilk (Gilbert, 1987; Equations 12.3 and 12.4) are used to determine whether or not a data set has been drawn from a population that is normally distributed.

The equation used to calculate the UCL for the lognormal distribution is shown below:

$$UCL = e^{\bar{x} + 0.5 \left(s^2 \right) + sH / \sqrt{n - 1}}$$

where:

- UCL = 95 percent upper confidence limit
- e = constant (base of the natural log, equal to 2.718)
- \bar{x} = arithmetic mean of transformed data
- s = standard deviation of the transformed data
- H = H-statistic (Gilbert, 1987)
- n = number of samples

The equation used to calculate the UCL for the normal distribution is:

$$UCL = \bar{x} + t(s/\sqrt{n})$$

where:

- UCL = 95 percent upper confidence limit
- \bar{x} = arithmetic mean of the untransformed data
- s = standard deviation of the untransformed data
- t = Student-t statistic (Gilbert, 1987)
- n = number of samples

In many cases, analytes are below the applicable detection limit in each sample. Non-detected results (U-qualified) are reported as less than the sample quantitation limit (SQL). The chemical may be present at the concentration just below the reported quantitation limit, or it may not be present in the sample at all. For media in which a chemical has been otherwise detected, non-detected results for that chemical will be treated statistically as one-half the SQL as a proxy concentration. This standard conservative approach is used to determine the concentrations most representative of potential exposures.

The statistical methods described in this section are parametric procedures and are intended for use in cases where the percentage of non-detects in a particular data set is less than 50 percent. In the event that the percentage of non-detects for a particular chemical is greater than 50 percent, non-parametric procedures will be applied as appropriate. Procedures for evaluating and applying non-parametric statistics are described in the guidance document *Statistical Analysis of Ground-Water Monitoring Data at RCRA Facilities, Addendum to Interim Final Guidance* (EPA, 1992a).

2.6 HUMAN INTAKE ASSUMPTIONS AND EXPOSURE QUANTIFICATION

This section describes methods that are used for quantifying chronic exposures for exposure pathways identified in the conceptual model. Exposures are determined to characterize the RME, the maximum exposure reasonably expected to occur at the site (EPA, 1989a). If the RME concentration is determined to be below the appropriate threshold, then it is likely that all other lesser exposure concentrations at the site will also be below levels of concern. Exposure parameters that will be used to estimate the RME are provided in Table 6 for groundwater exposure pathways and in Tables 7 and 8 for surface water and sediment, respectively.

2.6.1 Groundwater and Surface Water Ingestion

A receptor can ingest water by drinking it or through using household water for cooking. An estimate of intake from ingesting water is calculated as follows (EPA, 1989a):

$$I_w = \frac{C_w \cdot IR \cdot FI \cdot ED \cdot EF}{BW \cdot AT}$$

where:

I_w	=	intake of contaminant from drinking water (mg/kg/day)
C_w	=	concentration of contaminant in water (mg/L)
IR	=	ingestion rate (L/day)
FI	=	fraction ingested from contaminated source (unitless)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT	=	averaging time (days); for noncarcinogens, AT equals [(ED)(365 days/year)]; for chemical carcinogens, AT equals [(70 years)(365 days/year)]

2.6.2 Dermal Contact with Water

The estimate of intake of contaminants in water via absorption through the skin is determined using the concentration of a chemical in the water source evaluated. Evaluation of the dermal absorption pathway is performed for residents and construction workers exposed to groundwater and trespassers, recreational users and maintenance workers exposed to surface water using EPA default exposure parameters. The amount of a chemical taken into the body upon exposure via dermal contact is referred to as an absorbed dose. The absorbed dose is calculated using the dermal guidance contained in EPA 1989a, 1991b, and 1992b:

$$I_w = \frac{D_{event} \cdot SA \cdot EF \cdot ED}{BW \cdot AT}$$

where:

I_w	=	intake through skin from showering or wading (mg/kg/day)
D_{event}	=	absorbed dose per event (mg/cm ² -event)
SA	=	skin surface area (cm ²)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT	=	averaging time (days); for noncarcinogens, AT equals [(ED)(365 days/year)]; for chemical carcinogens, AT equals [(70 years)(365 days/year)]

D_{event} for inorganics can be calculated as:

$$DA_{event} = K_p^w C_w t_{event}$$

where:

DA_{event} = Dose absorbed per unit area per event (mg/cm²-event)

K_p^w = Permeability coefficient from water (cm/hr)

C_w = Concentration of chemical in water (mg/cm³)

t_{event} = duration of event (hr/event)

D_{event} for organics can be calculated as:

$$\text{If } t_{event} < t^*, \text{ then: } DA_{event} = 2 K_p C_v \sqrt{\frac{6\tau t_{event}}{\pi}}$$

or

$$\text{If } t_{event} > t^*, \text{ then: } DA_{event} = K_p C_v \left[\frac{t_{event}}{1+B} + 2\tau \left(\frac{1+3B}{1+B} \right) \right]$$

where:

C_w = concentration of constituent in water (mg/L)

K_p = permeability constant (cm/hour)

τ = lag time (hour)

B = partitioning coefficient (unitless)

ET = exposure time (hours)

π = Pi (3.14)

t^* = time to equilibrium conditions (hours)

2.6.3 Inhalation of Volatiles Released from Groundwater

The amount of a chemical taken into the body via exposure to volatilization of chemicals is evaluated using the concentration of a chemical in the water source (EPA, 1991a). Intake from the volatilization of chemicals in household water is calculated using the Andelman model (EPA, 1991a):

$$I_w = \frac{C_w \cdot K \cdot IR_i \cdot EF \cdot ED}{BW \cdot AT}$$

where:

I_w	=	intake of volatile in water from inhalation (mg/kg/day)
C_w	=	concentration of contaminant in water (mg/L)
K	=	volatilization factor (0.5 L/m ³)
IR	=	inhalation rate (m ³ /day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT	=	averaging time (days); for noncarcinogens, AT equals [(ED)(365 days/year)]; for chemical carcinogens, AT equals [(70 years)(365 days/year)]

This exposure pathway will only be evaluated for organic chemicals with a Henry's Law constant greater than 1×10^{-5} and with a molecular weight of 200 g/mole or less (EPA, 1991a).

2.6.4 Incidental Ingestion of Sediment

The estimation of intake of contaminants in sediment is determined using the concentration in sediment at the location of interest (EPA, 1989b).

$$I_s = \frac{C_s \cdot IR \cdot CF \cdot FI \cdot EF \cdot ED}{BW \cdot AT}$$

where:

I_s	=	intake from sediment (mg/kg-day)
C_s	=	concentration of contaminant in sediment (mg/kg)
IR	=	ingestion rate (g/day)
CF	=	conversion factor (10 ⁻³ kg/g)
FI	=	fraction ingested from contaminated source (unitless)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT	=	averaging time (days); for noncarcinogens, AT equals [(ED)(365 days/year)]; for chemical carcinogens, AT equals [(70 years)(365 days/year)]

2.6.5 Dermal Contact with Sediment

The estimation of intake of organic contaminants in sediment via absorption through the skin is determined using the concentration in sediment at the location evaluated (EPA, 1991b).

$$AB_s = \frac{C_s \cdot CF \cdot SA \cdot AF \cdot ABS \cdot EF \cdot ET \cdot ED}{BW \cdot AT \cdot TC}$$

where:

AB _s	=	amount of constituent absorbed during contact with sediment (mg/kg-day)
C _s	=	concentration of constituent in sediment (mg/kg)
SA	=	skin surface area available for contact (cm ² /event)
AF	=	skin adherence factor (mg/cm ²)
ABS	=	absorption factor (unitless)
CF	=	conversion factor (10 ⁻⁶ kg/mg)
EF	=	exposure frequency (events/year)
ET	=	event time (hours/day)
TC	=	time conversion (24 hours/day)
ED	=	exposure duration (years)
BW	=	body weight (kg)
AT	=	averaging time (days); for noncarcinogens, AT equals [(ED)(365 days/year)]; for chemical carcinogens, AT equals [(70 years)(365 days/year)]

Chemical-specific ABS are presented in Tables 9 and 11.

2.7 TOXICITY ASSESSMENT

The toxicity assessment describes appropriate toxicity values that are used to generate estimates of potential health risks associated with chemical exposure. This is accomplished by identifying appropriate sources of toxicity values and reviewing available information to identify the most appropriate values to use in the assessment. In addition, the toxicity assessment provides the basis for developing summaries of the potential toxicity of the COPCs for inclusion in the risk assessment. This is accomplished by reviewing available information on the toxicity of the COPCs and summarizing the factors pertinent to the exposures being assessed.

Toxicity values used in the risk assessment are provided by the EPA (2000). The data used by the EPA to guide the derivation of cancer slope factors (SFs) for carcinogenic effects and reference doses (RfDs) for noncarcinogenic effects may include epidemiological studies, long-term animal bioassays, short-term test, and comparisons of molecular structure. Data from these sources are reviewed to determine whether a chemical is likely to be toxic to humans. Because of the lack of available human studies, however, the majority of toxicity data used to derive SFs and RfDs come from animal studies.

The most appropriate animal model, i.e., the species biologically most similar to the human, is identified in the development of the RfD. In the absence of sufficient data to identify the most appropriate animal model, the most sensitive animal species is chosen. The RfD is generally derived from the most comprehensive toxicology study that characterizes the dose-response relationship for the critical effect of the chemical. Preference is given to studies using the exposure route of concern. In the absence of such data, however, an RfD for one route of exposure may be extrapolated from study data that was generated using a different route of

exposure. Uncertainty factors are applied to the highest no observed adverse effect level (NOAEL) to adjust for inter- and intraspecies variation, deficiencies in the toxicological database, and use of short-term rather than long-term animal studies.

SFs are classified in different groups according to the amount of evidence available that points to the chemicals carcinogenicity. Weight-of-evidence Group A (human carcinogen) or Group B (probable human carcinogen) chemicals are generally derived from cancer studies that adequately identify positive results, identify the target organ in the test animal, and characterize the dose-response relationship. SFs for Group C (possible human carcinogen) chemicals are derived when data are sufficient, but are not derived for Group D (not classified) or E (evidence of noncarcinogenicity) chemicals.

The toxicity assessment includes a list of toxicity values for both carcinogenic and noncarcinogenic effects and toxicity profiles that summarize the data used to derive the toxicity values. Toxicity values are presented in Tables 9 through 12. Toxicity profiles are provided in Appendix B.

3.0 RISK CHARACTERIZATION

The purpose of the risk characterization step is to integrate the exposure and toxicity assessments to generate quantitative expressions of cancer risk and noncancer hazard. The risk characterization is performed in accordance with EPA risk assessment guidelines (EPA, 1989a). To characterize potential noncarcinogenic effects, comparisons are made between calculated intakes of chemicals and toxicity values. To characterize potential carcinogenic effects, probabilities that an individual will develop cancer over a lifetime of exposure are estimated from calculated intakes and chemical-specific dose-response information.

Risk characterization serves as the bridge between risk assessment and risk management and is, therefore, a key step in the ultimate site decision-making process. This step summarizes risk assessment information for the risk manager to consider with other factors important for decision-making such as economics, technical feasibility, and regulatory context. The following sections provide separate discussions for carcinogenic and noncarcinogenic effects because the methodology differs for these two modes of chemical toxicity. In addition to providing methods for calculating risk estimates, this section provides information for the interpretation of results with regard to the uncertainty associated with the estimates (EPA, 1989a).

3.1 CARCINOGENIC RISK ESTIMATES

Cancer risk will be compared to a target risk range of 1×10^{-6} to 1×10^{-4} . Total cancer risk from all exposures can be summed:

$$\text{Total Cancer Risk} = \Sigma \text{Cancer Risk}_i$$

where:

$$\begin{aligned} \text{Total Cancer Risk} &= \text{Total lifetime cancer risk from exposures to all chemicals} \\ &\quad \text{(unitless)} \\ \text{Cancer Risk}_i &= \text{Lifetime cancer risk from exposures to chemical contaminant } i \\ &\quad (i=1 \dots n) \text{ (unitless)} \end{aligned}$$

Cancer risk from exposures to chemical contaminants can be estimated using the equation:

$$\text{Cancer Risk}_i = I_i \cdot SF_i$$

where:

$$\begin{aligned} \text{Cancer Risk}_i &= \text{lifetime cancer risk (unitless) from chemical contaminant } i (i=1 \dots n) \\ I_i &= \text{total daily intake of contaminant } i (i=1 \dots n) \text{ from indirect exposures} \\ &\quad \text{(mg/kg/day)} \\ SF_i &= \text{slope factor } ([\text{mg/kg/day}]^{-1}) \text{ for chemical contaminant } i (i=1 \dots n) \end{aligned}$$

3.2 NONCANCER HAZARD ESTIMATES

The hazard index (HI) is used to evaluate noncancer risk for any given target organ. The target HI is 1. The Hazard Quotient (HQ) is used to evaluate noncancer toxicity of individual chemical contaminants. The HQ represents the ratio of the dose received by the exposed individual to the dose that is associated with no adverse effects, i.e. the threshold or reference dose. HQs that affect the same target organ (i.e., liver, kidney, etc.) are summed to obtain a HI for an individual target organ. The HI can be estimated using the equation:

$$HI = \sum HQ_i$$

where:

- HI = hazard index (unitless)
 HQ_{*i*} = hazard quotient for chemical *i* (*i*=1...*n*) (unitless)

The HQ for exposures to chemical contaminants which have noncancer health effects can be estimated using the equation below:

$$HQ_i = \frac{I_i}{RfD_i}$$

where:

- HQ_{*i*} = hazard quotient for chemical *i* (*i*=1...*n*) (unitless)
 I_{*i*} = total daily intake from exposures to chemical contaminant *i* (*i*=1...*n*) (mg/kg/day)
 RfD_{*i*} = reference dose for chemical *i* (*i*=1...*n*) (mg/kg/day)

3.3 DEVELOPMENT OF RISK MAPS

In order to generate risk maps (i.e., risk isopleth maps), it is necessary to estimate risk for every location on the site map. This can be accomplished by calculating a unit risk value (risk per mg/L) for each COPC and multiplying that value by every concentration value at each point in a concentration plume map for the same COPC (see spreadsheets “Unit Risk” and “Unit Hazard” in Appendix C for calculations). Note that in the spreadsheets (Appendix C), a value of 1×10^{-6} is not assigned to a concentration of 1 mg/L. Rather, risk and hazard are calculated for each COPC when a concentration is fixed at 1 mg/L. This results in the values presented in Tables 13 (Unit Risk Values for Carcinogenic Groundwater COPCs) and 6.14 (Unit Hazard Values for Noncarcinogenic Groundwater COPCs). Using these “Unit Risks” (risk per mg/L) and “Unit Hazard” (hazard per mg/L) values, risk and hazard can be calculated for any groundwater concentration. These values are then used to create the risk and hazard isopleth maps. Where the COPC was not detected, risk and HQ values were assigned a value of zero. For example, if a plume of TCE is found in one area at concentrations that range from 1.6×10^{-3} mg/L to 1.6×10^{-1} mg/L, a corresponding risk map will describe the TCE

as a risk plume ranging in cancer risk from approximately 1×10^{-6} to 1×10^{-4} for residential receptors.

These risk estimates are contoured (i.e., extrapolated) in the same manner as the concentration contours. A similar procedure is followed for noncarcinogens using unit HQ values. Unit risk and unit hazard values used to derive risk and hazard maps for the age-adjusted resident, the child resident and the construction worker are presented in Table 13.

Contaminant Risk Maps were created for each of the following risk scenarios:

- 1) Cancer/Resident
- 2) Noncancer/Resident
- 3) Cancer/Construction Worker
- 4) Noncancer/Construction Worker

In addition to selected COPC-specific risk maps, total risk maps combining cancer risk for all COPCs, and total hazard maps combining noncancer hazard for all noncarcinogenic COPCs were prepared for each exposure scenario.

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4.0 RESULTS OF THE HUMAN HEALTH RISK ASSESSMENT

Results of the human health risk assessment are grouped according to receptors. Risk and hazard estimates for each receptor are discussed according to relevant media. The figures referenced throughout this section display the hazard quotients and risks for each COPC. It should be noted that many of the COPCs exceeding risk and hazard quotients are calculated from data that was non-detect. These non-detects are noted on the figures and should be considered when assessing true risks at the site.

4.1 ADULT RESIDENT

The age-adjusted resident was chosen as the representative residential receptor for carcinogenic COPCs. Adult residents provide the most conservative receptor for the evaluation of carcinogenic effects from groundwater exposures. This receptor is intended to evaluate exposures for the entire lifetime of a resident living at the site. Figures 1 through 7 illustrate the risk isopleths for individual groundwater carcinogenic COPCs. Risk in excess of 1×10^{-4} is seen in the northwest corner of the site for risk drivers, TCE (Figure 2), vinyl chloride (Figure 3) and 1,1-DCE (Figure 4).

An isopleth map of cumulative risk from all organic COPCs is provided in Figure 39. Cumulative risk is found to be between 1×10^{-6} and 1×10^{-5} throughout the residential areas along the southeast site border. Risk is estimated to be between 1×10^{-4} and 1×10^{-3} in the northwest area that includes the golf course.

The only two COPCs found to exceed criteria for potential vapor intrusion to basements were TCE and vinyl chloride (Section 2.4.4, Table 2). The portions of the TCE plume that intersect residential areas in the greater portion of the south-west residential area represent risk less than 1×10^{-6} which corresponds to a TCE concentration less than $2.5 \mu\text{g/L}$ [$(1 \times 10^{-6}/\text{unit risk}) = \text{concentration at } 1 \times 10^{-6} \text{ risk}$]. The TCE screening concentration for vapor intrusion into basements is $2200 \mu\text{g/L}$ (Table 2). There is a three order of magnitude difference between the TCE vapor intrusion screening criterion and the TCE groundwater concentrations in the residential area. Likewise, the vapor intrusion screening criterion for vinyl chloride is $12 \mu\text{g/L}$. The vinyl chloride groundwater concentration in the residential area (Figure 3) is approximately $0.5 \mu\text{g/L}$. Both TCE and vinyl chloride groundwater concentrations in the residential area are less than their vapor intrusion screening criteria.

4.2 CHILD RESIDENT

Child residents provide a more conservative receptor for the evaluation of noncarcinogenic effects from groundwater exposures. Figures 17 through 24 illustrate the hazard isopleths for individual groundwater noncarcinogenic COPCs. Hazard in excess of the target of 1 is seen for PCE (Figure 17), TCE (Figure 18), cis-1,2-DCE (Figure 19), and chloroform (Figure 23). Cumulative noncancer hazard for all organic COPCs is illustrated in Figure 38. The plume area associated with residential areas (the southeast side of the site, is associated with

cumulative hazard less than 1. However, cumulative noncancer hazard for the residential receptor is greater than 1 in the north-west corner associated with the golf course.

4.3 CONSTRUCTION WORKER

Exposure to groundwater was also evaluated for the potential future construction worker. This receptor would be the only receptor exposed to groundwater if institutional controls were in place to restrict use of groundwater for residential use. Figures 9 through 15 illustrate the risk isopleths for individual groundwater carcinogenic COPCs. There are no individual COPCs that result in cancer risk to the construction worker in excess 1×10^{-4} . In addition, cumulative risk to all organic COPCs (Figure 37) is less than 1×10^{-4} .

Figures 27 through 35 illustrate the noncarcinogenic hazard isopleths for individual groundwater noncarcinogenic COPCs. Noncarcinogenic COPCs associated with potential hazard in excess of the target of 1 for the construction worker are TCE (Figure 28), cis-1,2-DCE (Figure 29) and vinyl chloride (Figure 30). Cumulative hazard in excess of 1 for the construction worker (Figure 40) is found throughout the north-west portion of the site in areas associated with the golf course.

4.4 TRESPASSER

A summary of the cancer risk and hazard (non-cancer) estimates associated with trespasser exposures to surface water and sediment is presented in Table 15.

4.4.1 Surface Water

At 1.5×10^{-8} , the cancer risk for the RME trespasser exposed to surface water falls below the EPA point of departure of 1×10^{-6} . The noncancer hazard for exposures to surface water (0.0025) is below the limit of 1.

4.4.2 Sediment

The cancer risk for the RME trespasser (2.5×10^{-8}) exposed to sediment is below the EPA point of departure of 1×10^{-6} . The noncancer hazard for exposure to sediment (0.0027) is below the limit of 1.

4.4.3 Cumulative Across All Media

The cumulative cancer risk for the RME trespasser exposed to surface water and sediment is 4.1×10^{-8} , which is below the departure point of 1×10^{-6} . The cumulative noncancer hazard for the RME trespasser exposure to surface water and sediment is 0.0053, which is below the limit of 1.

4.5 MAINTENANCE WORKER

A summary of the cancer risk and hazard (non-cancer) estimates associated with maintenance worker exposures to surface water and sediment is presented in Table 16.

4.5.1 Surface Water

At 5.5×10^{-8} , the cancer risk for the RME trespasser exposed to surface water falls below the EPA point of departure of 1×10^{-6} . The noncancer hazard for exposures to surface water (0.0022) is below the limit of 1.

4.5.2 Sediment

The cancer risk for the RME trespasser (3.9×10^{-8}) exposed to sediment is below the EPA point of departure of 1×10^{-6} . The noncancer hazard for exposure to sediment (0.0009) is below the limit of 1.

4.5.3 Cumulative Across All Media

The cumulative cancer risk for the RME trespasser exposed to surface water and sediment is 9.4×10^{-8} , which is below the departure point of 1×10^{-6} . The cumulative noncancer hazard for the RME trespasser exposure to surface water and sediment is 0.0032, which is below the limit of 1.

4.5.4 Recreational User

A summary of the cancer risk and hazard (non-cancer) estimates associated with trespasser exposures to surface water and sediment is presented in Table 17.

4.5.5 Surface Water

At 4.7×10^{-8} , the cancer risk for the RME trespasser exposed to surface water falls below the EPA point of departure of 1×10^{-6} . The noncancer hazard for exposures to surface water (0.0045) is below the limit of 1.

4.5.6 Sediment

The cancer risk for the RME trespasser (3.3×10^{-8}) exposed to sediment is below the EPA point of departure of 1×10^{-6} . The noncancer hazard for exposure to sediment (0.0019) is below the limit of 1.

4.5.7 Cumulative Across All Media

The cumulative cancer risk for the RME trespasser exposed to surface water and sediment is 8.0×10^{-8} , which is below the departure point of 1×10^{-6} . The cumulative noncancer hazard for the RME trespasser exposure to surface water and sediment is 0.0064, which is below the limit of 1.

5.0 UNCERTAINTY ASSESSMENT

Calculated risk estimates are subject to varying degrees of uncertainty from a variety of sources. Areas of uncertainty in a risk assessment can be categorized as: generic or methodological and site-specific. Methodological uncertainties are those that are inherent to the methods or procedures used for risk assessments (e.g., policy decisions made to reflect EPA's desire to err on the side of conservatism). Site-specific areas of uncertainty are those characteristics of the site or the investigation of the site that could result in overestimates or underestimates of risk. The most significant sources of uncertainty in the risk assessment is itemized and evaluated qualitatively for their potential to contribute to either the over- or underestimation of risk. Specific areas of uncertainty are discussed in following sections.

5.1 METHODOLOGICAL UNCERTAINTY

There are four major areas of methodological uncertainty: uncertainty in the estimation of contaminant concentration, uncertainty in the estimation of exposure, and uncertainty in the estimation of toxicity, and uncertainty in the estimation of risk.

5.2 CONTAMINANT CONCENTRATION

It is not possible to completely characterize the nature and extent of contamination at any site. In selecting COPCs, and in estimating concentrations, uncertainties arise from limits on the number and locations of environmental samples that can be collected to characterize a site and from eliminating constituents that are infrequently detected. These limitations may tend to over- or underestimate risk. The use of the 95 percent UCL of average contaminant concentrations or maximum detected concentrations tend toward a conservative, health-protective bias. However, when evaluating constituents with low detection frequencies, the use of the maximum detected concentration, in some instances, over estimates average exposures by an order of magnitude or more. Since exposures to any medium can be more accurately reflected by evaluating media concentrations over some area rather than by a single point, exposure estimates using maximum detected values over estimate the exposure for most exposed individuals.

5.3 EXPOSURE ASSESSMENT

Standard assumptions for population characteristics, such as body weight or life expectancy, and exposure characteristics, such as frequency, duration, amount of intake or contact may not represent actual exposure conditions. Standard exposure assumptions are used to characterize residential groundwater exposures. The assumption that a population receives all of their liquid intake from one source is generally recognized as an overestimation of exposure. In addition, exposure is estimated over the lifetime of the resident, in the case of the age-adjusted resident used to estimate carcinogenic risk from groundwater exposure, and to the entire childhood time, in the case of the child resident used to estimate noncancer risk from groundwater exposure. This attempt to average exposure becomes necessary in the interest of

simplifying the risk assessment. The alternative would be to calculate estimates for every year of a receptor's life, which would still be fraught with inaccuracies. This is not only true for the residential receptors. Assumptions made to characterize exposures for each receptor, whether construction worker, trespasser, or recreational user are assumptions, based on best professional judgement. The reader must recognize that all exposure estimates are just that, estimates, and not measurements of actual exposure.

5.4 TOXICITY ASSESSMENT

The principal uncertainties associated with the toxicity assessment are:

- Extrapolation of toxic effects observed at the high doses necessary to conduct animal studies to effects that might occur at much lower, "real-world" doses; and
- Extrapolation from toxic effects in animals to toxic effects in man.

For noncancer effects, these uncertainties are given numerical value by using an uncertainty factor, which is actually a product of as many as five separate factors, each intended to account for one type of uncertainty (EPA, 1998). For cancer effects, the uncertainty is addressed by estimating the 95 percent upper bound on the slope of the dose-response curve (EPA, 1998). Utilizing the guidance of the EPA will minimize uncertainties by using EPA-derived toxicity values (EPA, 1998 and 1997a) to evaluate the risks posed by constituents. The basis of EPA policy in the derivation of toxicity values is to err on the side of conservatism, which may tend to overestimate risk. However, uncertainties associated with the lack of published toxicity data on many constituents would tend to balance any overestimation of risk by tending to underestimate risk from these constituents.

5.5 RISK CHARACTERIZATION

Risk is assumed to be additive for chemicals with similar sites of toxicological action. In the event that any combinations of these chemicals result in multiplicative effects, risk may be underestimated. Furthermore, many assumptions made in the application of SFs and RfD's are uncertain. For example, the estimate of dermal risk and hazard are based on extrapolations from oral doses since data are lacking for dermal exposures. Although current EPA methodology typically leads to conservative estimates, the magnitude of the error associated with these extrapolations is unknown.

5.6 SITE-SPECIFIC UNCERTAINTIES

Site-specific uncertainties can be categorized into two major areas: analytical methodology and background. Each of these areas will be discussed in the context of the impact on risk assessment.

- Analytical Methodology: Some uncertainty may be introduced by combining the data sets from multiple investigations because there are differences in the compounds that have been analyzed, methods used to collect the samples and differences in the laboratory analytical procedures.
- Background: Background was evaluated for inorganics only. EPA recognizes that some organic constituents are found as anthropogenic background constituents. These chemicals are present on a site as a function of human activity. For example, polyaromatic hydrocarbons, byproducts of combustion of fossil fuel, may be found in sediment due to runoff of industrialized areas. In this risk assessment, only inorganic chemicals were included in the background comparison. Since anthropogenic organic chemicals were not evaluated as background constituents, risk may be overestimated.

A risk assessment of a site is ultimately an integrated evaluation of historical, chemical, analytical, environmental, demographic, and toxicological data that are as site-specific as possible. To minimize the possibility of underestimating risk, each step was biased toward health-protective estimations. Because each step builds on the previous one, this biased approach compensates for risk assessment uncertainties that underestimate true risk. In addition, these calculations do not represent currently existing or expected future exposure or health risks. Rather, they are estimates of potential risk only if all of the conservative exposure assumptions are realized. This risk assessment does not represent a worst-case scenario, therefore, the potential for underestimating some risks to some receptors, however unlikely, does exist.

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6.0 ECOLOGICAL RISK ASSESSMENT

An ecological risk assessment (ERA) is a process that can be used to estimate the risk or probability of adverse effects to biota. Estimates of risk to biota based on this ERA can be used to determine if risks are acceptable or if further assessment is necessary.

Ecological risk assessment is a qualitative and/or quantitative appraisal of the actual or potential effects of chemical or physical stressors on plants and animals other than people and domesticated species. The objective of this ecological risk assessment is to determine whether or not there are any potential adverse ecological effects that may be caused by exposure to potential contaminants in surface water and sediment at the BRAC property. The primary objective of the ERA is to determine whether unacceptable adverse risks are posed to ecological receptors as a result of the hazardous substance releases. This objective is met by characterizing the ecological communities in the vicinity of the surface water body, determining the particular hazardous substances associated with the surface water body, identifying pathways for receptor exposure, and determining the extent to which response actions are necessary.

The State of Texas has recently published ecological risk assessment guidance (TNRCC, 2000a). This guidance applies to sites regulated within the Texas Natural Resource Conservation Commission' (TNRCC) Remediation Division. Although this site is regulated under CERCLA, and since this guidance mirrors the EPA's Ecological Risk Assessment Guidance for Superfund (EPA, 1997c), and the Tri-Service Procedural Guidelines for Ecological Risk Assessments (Wentzel, et al., 1996), the TNRCC guidance will be used as the primary guidance document used in performing this ecological risk assessment.

The TNRCC ecological risk assessment methodology is a tiered approach to assessing ecological risk. Tier 1 is an exclusion criteria checklist. If the site does not meet the exclusion criteria, a Tier 2, screening-level ecological risk assessment, will be conducted. The Tier 2 assessment includes:

- 1) A comparison of detected constituent concentrations for non-bioaccumulative COPCs to established ecological benchmarks.
- 2) Identify communities and major feeding guilds and their representative species which are supported by habitats at the site.
- 3) Develop a conceptual model which depicts the movement of COPCs through media to communities and the feeding guides.
- 4) Discuss COPC fate and transport and toxicological profiles.
- 5) Prepare a list of input data which includes values from the literature (e.g., exposure factors, intake equations, no observed adverse effect level (NOAEL) and lowest observed adverse effect level (LOAEL) values, references) and reasonably conservative exposure assumptions, and then calculate the total exposure to selected ecological receptors from each COPC not eliminated according to item number 1.

- 6) Utilize an ecological hazard quotient methodology to compare exposures to NOAELs in order to eliminate COPCs that pose no unacceptable risk (i.e., NOAEL hazard quotient 1). If all COPCs are eliminated at this point, the ecological risk assessment process ends. Otherwise, the process continues.
- 7) Less conservative assumptions for exposure may be applied and the hazard quotients re-calculated. If all COPCs are eliminated at this point, the ecological risk assessment process ends. Otherwise, the process continues.
- 8) Develop an uncertainty analysis that discusses the major areas of uncertainty associated with the screening level ecological risk assessment. If all COPCs are eliminated at this point, the ecological risk assessment process ends. Otherwise, the process continues.
- 9) Calculated medium-specific protective concentration levels (PCLs) bounded by NOAELs and LOAELs for those COPCs which are not eliminated as a result of the hazard quotient exercises or the uncertainty analysis.
- 10) Make recommendations for managing ecological risk at the site based on final PCLs. Recommendations can also be made for proceeding with a Tier 3 evaluation.

6.1 PROBLEM FORMULATION

This section presents the problem formulation that establishes the goals, breadth, and focus of the ERA through an evaluation of Constituents of Potential Ecological Concern (COPEC), a characterization of the ecological communities, a selection of assessment and measurement endpoints, an identification of ecological receptors, and a presentation of an ecological conceptual site model. As stated in Section 2.0, soil is not included in this risk assessment. Therefore, terrestrial plants will not be addressed in this ecological risk assessment.

6.2 SELECTION OF ASSESSMENT AND MEASUREMENT ENDPOINTS

The protection of ecological resources, such as habitats and species of plants and animals, is a principal motivation for conducting an ERA. Key aspects of ecological protection are presented as policy goals. These are general goals established by legislation or agency policy that are based on societal concern for the protection of certain environmental resources. For example, environmental protection is mandated by a variety of legislation and government agency policies (e.g., CERCLA, National Environmental Policy Act). Other legislation includes the Endangered Species Act 16 U.S.C. 1531-1544 (1993, as amended) and the Migratory Bird Treaty Act 16 U.S.C. 703-711 (1993, as amended). To determine whether these protection goals are met at the site, assessment and measurement endpoints have been formulated to define the specific ecological values to be protected and to define the degree to which each may be protected.

An ecological endpoint is a characteristic of an ecological component that may be affected by exposure to a chemical and/or physical stressor. Assessment endpoints represent environmental values to be protected and generally refer to characteristics of populations and

ecosystems (Suter, 1993). Unlike the human health risk assessment process, which focuses on individual receptors, the ERA focuses on populations or groups of interbreeding nonhuman, nondomesticated receptors. In the ERA process, the risks to individuals are assessed only if they are protected under the Endangered Species Act, as well as species that are candidates for protection and those considered rare.

Given the diversity of the biological world and the multiple values placed on it by society, there is no universally applicable list of assessment endpoints. Suggested criteria that were considered in selecting assessment endpoints suitable for this ecological risk assessment are: (1) ecological relevance, (2) susceptibility to the contaminant(s), (3) accessibility to prediction and/or measurement, (4) societal relevance, and (5) definable in clear, operational terms (Suter, 1993). Assessment and measurement endpoints are presented as screening criteria in Tables 19 and 20 and toxicity values in Table 21 and 22.

6.2.1 Assessment Endpoints

The assessment endpoints for Former Carswell AFB are stated as the protection of long-term survival and reproductive capabilities for small omnivorous mammals, omnivorous birds, benthic invertebrates, and aquatic vertebrates (fin fish). The corresponding null hypothesis (H_0) for each of the assessment endpoints is stated as: the presence of site contaminants within surface water and sediment will have no effect on the survival or reproductive capabilities of small omnivorous mammals, omnivorous birds, benthic invertebrates, and aquatic vertebrates.

Assessment receptor species were selected based on the likelihood of finding the species at the Former Carswell AFB. Historical information, and the availability of toxicological data were used to select receptor species. Generic terrestrial vertebrates, benthic invertebrates, and aquatic vertebrates were used to represent receptors.

6.2.2 Measurement Endpoints

Measurement endpoints are defined as a measurable ecological characteristic that is related to the valued characteristic chosen as the assessment endpoint (USEPA, 1992). Measurement endpoints are frequently numerical expressions of observations (*e.g.* toxicity test results or community diversity indices) that can be compared statistically to detect adverse responses to a site contaminant. Examples of typical measurement endpoints include mortality, growth or reproduction in toxicity tests; individual abundance; species diversity; and the presence or absence of indicator data in field surveys of existing impacts (USEPA, 1994).

For this assessment measurable responses to stressors include lowest observed adverse effect levels (LOAEL), and no observed adverse effect levels (NOAEL) (for terrestrial and avian species), and screening criteria (for benthic and aquatic species). The most appropriate measurement endpoints were chosen based on exposure pathways as well as ecotoxicity of the contaminant.

6.3 IDENTIFICATION OF REPRESENTATIVE ECOLOGICAL RECEPTORS

This section presents the selection and rationale for representative terrestrial and aquatic ecological receptors at the site.

6.3.1 Terrestrial

Indicator species represent two classes of vertebrate wildlife (mammals and birds). The species selected include the deer mouse (*Peromyscus maniculatus*) (small, omnivorous mammal), and the quail (*Colinus virginianus*) (small, omnivorous bird).

The deer mouse has a limited home range which makes them particularly vulnerable (i.e., conservative) to exposure to site contaminants. The selected terrestrial receptor species have a potential high abundance and wide distribution at the site and sufficient toxicological information is available in the literature for comparative and interpretive purposes. In addition, all of the selected species are likely to occur after site remediation (if risk management decisions require it), and all are important to the stability of the local ecological food chain and biotic community. Finally, all the selected species have readily available exposure data, as summarized in the *Wildlife Exposure Factors Handbook* (USEPA, 1993). The mouse and quail have been chosen merely as representative species that could be found at the site and become exposed to surface water and sediment. Since much of this area is a golf course (and is expected to remain a golf course), it is not available to a variety of species.

6.3.2 Aquatic

Exposure to aquatic organisms within the water bodies is assumed to occur via direct exposure to contaminants in the water and via ingestion of benthic invertebrates and pelagic prey exposed to contaminants in surface water and sediment. Potential effects to fish, macroinvertebrates, and phytoplankton (algae) were assessed using available surface water and sediment quality criteria for the protection of aquatic life. Adverse effects to aquatic species are evaluated through comparisons with surface water and sediment screening criteria (EPA Region IV values) (EPA, 2000b).

6.3.3 Receptor Profiles

This section presents brief receptor profiles for the representative receptors selected for the site.

6.3.4 Deer Mouse (*Peromyscus maniculatus*)

This medium-sized mouse is found in the eastern United States from the Hudson Bay to Pennsylvania, the southern Appalachians, central Arkansas and central Texas. In the west it is found from Mexico to the south Yukon and Northwest Territories (Whitaker, 1995). Deer mice habitat includes nearly every dry land habitat within its range, including forest,

grasslands, or a mixture of the two (Burt and Grossenheider, 1980). Nocturnal and active year-round, these mice construct nests in the ground, trees, stumps, and buildings (Burt and Grossenheider, 1980). Omnivorous, the deer mouse feeds on nuts and seeds (e.g., jewel weed and black cherry pits), fruits, beetles, caterpillars, and other insects. Their home range is 0.5 to 3 acres (Burt and Grossenheider, 1980). Density of populations is 4 to 12 mice per acre, and average life span is two years in the wild (Burt and Grossenheider, 1980). The breeding season is from February to November, depending on latitude. Three to five young are born in each of two to four litters per year (Burt and Grossenheider, 1980). They are greyish to reddish-brown with a white belly, with a distinctly short-haired, bicolor tail (Whitaker, 1995). Weight range is 14.8 (USEPA, 1993) to 33 grams (Whitaker, 1995).

6.3.5 Quail (*Colinus virginianus*)

Quail are ground-dwelling birds with short, heavy bills adapted for foraging on the ground for seeds and insects. Most species inhabit brush, abandoned fields, and open woodlands; some inhabit parklands. They are poor flyers that seldom leave the ground and do not migrate. They range from southeastern Wyoming, east to southern Minnesota and across to southern Main, south through the central and eastern United States to eastern New Mexico in the west and to Florida in the east. Quail forage during the day, primarily on the ground or in a light litter layer less than 5 cm deep. Seeds from weeds, woody plants, and grasses comprise the majority of the adult quail's diet. In some areas, quail can acquire their daily water needs from dew, succulent plants, and insects; in more arid areas, however, quail need surface water for drinking. In breeding season, the quail's home range includes foraging areas, cover, and the nest site and may encompass several hectares (EPA, 1993).

6.4 EXPOSURE PATHWAYS

Exposure pathways evaluated in this ERA include sediment and surface water.

6.4.1 Surface Water Exposure Pathway

Surface water represents a potential transport medium for the COPECs. Potential sources for contaminated surface water for this assessment includes seepage of groundwater. Potential receptors of contaminated surface water include terrestrial and aquatic fauna and aquatic flora. Exposure routes for contaminated surface water include ingestion by terrestrial fauna, and uptake and absorption by aquatic flora and fauna. Consumption of bioaccumulated contaminants constitutes a potential indirect exposure pathway for faunal receptors. Chemical bioavailability of some metals and other chemicals is controlled by water hardness, pH, and total suspended solids. It should also be kept in mind, however, that the greatest risk to aquatic vertebrates is when Farmer's Branch Creek becomes dry like it has during the recent record droughts that North Central Texas has been experiencing.

6.4.2 Sediment Exposure Pathway

Sediment consists of materials precipitated or settled out of suspension in surface water. Potential contaminant sources for sediment in this assessment includes seepage from groundwater. The release mechanisms include surface water runoff, groundwater discharge, and airborne deposition. Potential receptors of chemicals in contaminated sediment include aquatic flora and fauna. Direct exposure routes for contaminated sediment include uptake by aquatic flora and ingestion by aquatic fauna. Indirect exposure pathways from sediment include consumption of bioaccumulated contaminants by consumers in the food chain. Chemical bioavailability of many nonpolar organic compounds, including PCBs and pesticides, decreases with increasing concentrations of total organic carbon in sediment; however, these compounds can still bioaccumulate up the food chain (Landrum and Robbins, 1990). Neither PCBs nor pesticides have been detected as COPECs in this assessment.

6.4.3 Groundwater Exposure Pathway

Groundwater represents a potential transport medium for COPECs. Potential contaminant sources for groundwater include contaminated soil, and buried or stored waste. The release mechanism for contaminants into groundwater is direct transfer of contaminants from waste materials to water as water passes through the materials.

Groundwater itself is not an exposure point. Contaminant transport along the shallow groundwater pathway may be an exposure route to aquatic life, wetlands, and some wildlife where the groundwater discharges to surface water. The potential impact of groundwater to surface water has been examined through direct sampling and evaluation of surface water.

6.5 EXPOSURE ESTIMATE AND RISK CALCULATION

Risk is estimated by comparing reasonable maximum exposure levels (i.e., 95% UCL) with the screening-level ecotoxicity values derived in the ecological effects evaluation. The risk evaluation includes:

1. A description of complete exposure routes.
2. Ecological hazard quotients developed for each constituent of potential concern in each media for each potentially exposed representative species.
3. Discussion of uncertainties and overall confidence in the ERA.

Methods for quantitation of intake for each species of concern and each media were developed by EPA (1993). Intake is estimated for sediment and surface water and compared with NOAELs and LOAELs described above.

6.5.1 Estimation of Sediment Intake

The equation used to estimate sediment intake by terrestrial and avian species is described below (EPA, 1993):

$$ADD_s = (C_k \cdot FS \cdot IR \cdot FR_k) / BW$$

where:

ADD _s	=	average daily dose from sediment (mg/kg-day)
C _k	=	average contaminant concentration in sediment in the k th foraging area (mg/kg)
FS	=	fraction of sediment in diet (unitless)
IR	=	food ingestion rate on a dry-weight basis (kg/day)
FR _k	=	fraction of total food intake from the k th foraging area (unitless)
BW	=	body weight (kg)

Input parameters for each species of concern are provided in Table 18. Estimates of foraging area are based on comparisons of IRP site area with known foraging area data for the species under evaluation.

6.5.2 Estimation of Surface Water Intake

The equation used to estimate surface water intake is described below (EPA, 1993):

$$ADD_w = C \cdot CF \cdot NIR$$

where:

ADD _w	=	average daily dose of surface water (mg/kg-day)
C	=	average contaminant concentration in surface water body (mg/L)
CF	=	conversion factor (L/kg)
NIR	=	normalized water ingestion rate; fraction of body weight consumed as water per unit time (g/g-day)

Input parameters for each species of concern are provided in Table 18.

6.5.3 Risk Calculation

Ecological hazard quotients (EQs) are developed for each constituent of potential concern in each media for each potentially exposed representative species. The EQ is expressed as the ratio of a potential exposure or dose to a toxicity value (EPA, 1994):

$$EQ = \frac{ADD}{TL} \text{ or } \frac{EEC}{Benchmark}$$

where:

EQ	=	ecological hazard quotient (unitless)
ADD	=	average daily dose (mg/kg-day)

TL	=	toxicity level; either a NOAEL or extrapolate NOAEL based on a LOAEL (mg/kg-day)
EEC	=	estimated environmental concentration (mg/kg or mg/L)
Benchmark	=	media concentration associated with minimal adverse effects to the species of concern (mg/kg) or (mg/L)

EQs for exposures to terrestrial and avian species are developed using intake values developed using equations in Sections 6.5.1 and 6.5.2. Sediment and surface water exposures to aquatic and benthic species are evaluated by comparisons of sediment and surface water constituent concentrations with ecotoxicologically-based benchmarks developed by EPA (1996b) (Tables 19 and 20). EQs for exposures to surface water and sediment in the mouse and quail are presented in Table 21 and 22.

The intent of the ERA is to evaluate population effects rather than effects to the individual. NOAELs are benchmarks which evaluate effects to all individuals within the exposed population compared with LD₅₀'s or LD₅'s which evaluate population benchmarks. Since NOAELs are the chosen benchmarks for this evaluation, an EQ of 1 will be evaluated as the target EQ. An EQ greater than 1 will be interpreted as a level at which adverse ecological effects may occur to the population. An EQ less than 1 will be associated with less likelihood of adverse ecological effects. Risk management decisions should take into account the magnitude of the EQ when determining the need for remediation. There is no consensus regarding the issue of summation across pollutants in the calculation of EQs. Since there is little data concerning mechanism of action or target organ toxicity for species other than mammalian species, contaminant-specific EQs will not be summed.

6.6 RESULTS OF THE ECOLOGICAL RISK ASSESSMENT

As indicated above, the ERA has been developed using a tiered approach. Tier 1 involves a criteria exclusion checklist. This Tier 1 form is found in Appendix D. The results of this exclusion checklist indicate that a Tier II evaluation is necessary. The Tier 2 evaluation included a comparison of detected constituent concentrations in surface water and sediment to benchmark criteria and a calculation of EQs for site-specific receptors. The results of the Tier 2 evaluations are provided below.

6.6.1 Comparisons with Surface Water and Sediment Benchmarks

Tables 19 and 20 summarize the comparisons of surface water and sediment benchmarks with COPCs. The COPCs included in the ecological risk assessment are those detected chemicals that exceed background and have been detected at a frequency greater than five percent.

Surface water COPCs found to exceed surface water benchmarks are inorganics beryllium, copper, lead, mercury, selenium, silver and zinc; and the semivolatile organic, bis(2-ethylhexyl)phthalate (Table 19). Detections of beryllium, copper, and mercury that exceed criteria were below detection limits and are, therefore, associated with some level of

uncertainty. Bis(2-ethylhexyl)phthalate, as a component of many plastics, is found ubiquitously in the environment. Its presence in surface water at Carswell may not be associated with site-specific activities.

Sediment COPCs found to exceed sediment benchmarks include inorganics, arsenic, nickel, and zinc; and polyaromatic hydrocarbons, benzo(a)anthracene and indeno(1,2,3-cd)pyrene (Table 20). Polyaromatic hydrocarbons are byproducts of fossil fuel combustion and are, therefore, typically associated with anthropogenic activities rather than from site-related contamination.

6.6.2 Estimates of Ecological Risk

Tables 21 and 22 summarize the calculated EQs for the mouse and quail, respectively. The only EQ found to exceed unity is aluminum. The maximum detected concentration of aluminum in surface water is 32 mg/L, which resulted in an EQ for the mouse of 2.8 (Table 21). All other EQs for all detected constituents in surface water and sediment for both the mouse and quail are less than 1. This implies that there is little potential for adverse health effects in these species associated with their exposures to surface water and sediment.

6.6.3 Uncertainties Associated with the Ecological Risk Assessment

A wide variety of factors contribute to the uncertainty associated with this ecological risk assessment. These factors are related to the exposure assessment, characterization of ecological effects, and the characterization of risk. The quantitative modeling of exposures to wildlife receptors incorporates a large number of parameters which are highly stochastic in nature or for which very limited quantitative information is available in the literature. In general, the values used in the exposure models were selected to result in a conservative estimation of risk. That is, the values for uncertain or stochastic parameters were generally biased toward those that would more likely overestimate the actual exposure rather than underestimate it.

The COPC concentrations used in all exposure models were the 95 percent UCL or maximum measured concentrations, thereby allowing for the overestimation of the probable concentration at this point. Further, this concentration was assumed to be uniform throughout the receptor's home range, allowing for the probable overestimation of exposure to the receptor species. The expected result of these factors is an overestimation of exposure and a conservative estimation of risk estimated by either HQs or by comparison with the screening values.

Wildlife exposure factors included body weight, daily food consumption, and dietary composition. In general, these were selected as average or mid-range values, to model exposure to an "average" individual of the modeled species. Body weights were taken as averages or the midpoint of ranges and the food. Because most animals feed opportunistically, dietary composition is also highly variable between individuals. The dietary compositions selected for the key receptor species were generalized from published literature,

which will lead to the overestimation of exposure to some individuals and the underestimation of others.

Exposure pathways were limited to ingestion. Although the exclusion of inhalation and dermal contact may result in an underestimation of exposure, this is probably compensated by conservatism in the dietary exposure modeling.

The use of NOAELs is conservative and may over estimate the hazards that will actually occur. The wildlife NOAELs are extrapolated from test species that are different from the target wildlife receptor species. When the test species was in a different class (e.g., a mammal species compared with a bird species), no extrapolation was performed as the target class may be either more or less sensitive to the chemical than the test species class. This results in a toxicity benchmark data gap for several of the avian COPCs.

The lack of toxicity data for a number of COPCs may result in the underestimation of receptor hazards, however, these constituents are not believed to be overly toxic to the selected receptors and it is unlikely hazard indices and overall ERA conclusions would change significantly if toxicity data were included for these COPCs.

In conclusion, many factors contribute to the uncertainty associated with these predicted risk results. Several of the factors can be ascribed to either leading to probable overestimation of risk or underestimations. It is expected that, in this ecological risk assessment, most factors were overestimated.

7.0 RISK ASSESSMENT CONCLUSIONS

Results of media with carcinogenic or noncarcinogenic constituents contributing to human health risk and hazard above the target risk range include:

- Groundwater – Potential future residential cancer risk associated with groundwater exposures in excess of 1×10^{-4} risk is estimated for TCE (Figure 2), vinyl chloride (Figure 3) and 1,1-DCE (Figure 4). Noncancer hazard associated with potential future residential exposures is in excess of the target of 1 for PCE (Figure 17), TCE (Figure 18), cis-1,2-DCE (Figure 19) and chloroform (Figure 23). Evaluation of site-specific screening criteria for the potential intrusion of volatile organics into residential basements indicate that this pathway is not associated with risk greater than 1×10^{-6} . It should also be noted that none of these volatile organics are known to bioaccumulate in terrestrial or aquatic animals.

Exposures to groundwater under the scenario of institutional controls to prevent residential use of groundwater was evaluated using the construction worker as the only potential receptor. Cumulative risk to organic COPCs for this receptor was less than 1×10^{-6} . However, noncancer hazard in excess of the target of 1 was estimated for TCE (Figure 28), cis-1,2-DCE (Figure 29) and vinyl chloride (Figure 40). Furthermore, it should be kept in mind, that groundwater exposures are only possible if the shallow contaminated aquifer, which is not currently in use, is used as a source of drinking water.

- Surface Water – All cancer risks and noncancer hazards are below EPA limits for surface water (Tables 15 through 17).
- Sediment – All cancer risks and noncancer hazards are below EPA limits for sediment (Tables 15 through 17).

Results of media with ecological risk about target include:

- Surface Water – Surface water COPCs found to exceed surface water benchmarks are inorganics beryllium, copper, lead, mercury, selenium, silver and zinc; and the semivolatile organic, bis(2-ethylhexyl)phthalate (Table 19). Detections of beryllium, copper, and mercury that exceed criteria were below detection limits and are, therefore, associated with some level of uncertainty. Bis(2-ethylhexyl)phthalate, as a component of many plastics, is found ubiquitously in the environment. Its presence in surface water at Carswell may not be associated with site-specific activities. The only detected constituent found to be associated with an EQ greater than 1 was aluminum for exposures in the mouse.

- Sediment – Sediment COPCs found to exceed sediment benchmarks include inorganics, nickel, and zinc; and polyaromatic hydrocarbons, benzo(a)anthracene and indeno(1,2,3-cd)pyrene (Table 20). Polyaromatic hydrocarbons are byproducts of fossil fuel combustion and are, therefore, typically associated with anthropogenic activities rather than from site-related contamination. No EQs associated with sediment exposures to either the mouse or quail were greater than 1.

Groundwater risk was estimated to be in excess of risk-based targets for both potential future residents and construction workers. Those COPCs that contribute to the potential for adverse health effects include 1,1-DCE, cis-1,2-DCE, PCE, TCE, and vinyl chloride. Since adverse health effects are estimated under both the residential scenario and the scenario of institutional controls, remedial action is warranted.

No human health effects have been estimated for exposures to surface water and sediment. In addition, although some exceedances have been noted for ecological screening criteria, with the exception of aluminum in surface water, EQs for surface water and sediment exposures were less than 1. Therefore, remedial action for surface water and sediment is not warranted.

8.0 REFERENCES

Burt, W.H. and R.P. Grossenheider, 1980, A Field Guide to Mammals, Peterson Field Guide Series, Houghton Mifflin, Boston.

Gilbert, R.O., 1987, Statistical Methods for Environmental Pollution Monitoring. Van Nostrand Reinhold, New York, NY.

Landrum, P.F. and J.A. Robbins, 1990, "Bioavailability of Sediment-Associated Contaminants to Benthic Invertebrates," in Sediment: Chemistry and Toxicity of In-Place Pollutants, R. Baudo, J.P. Giesy and Muntau Eds, Chelsea, Michigan: Lewis, 1990, pp. 237-263.

Suter, II, G.W., 1993, Ecological Risk Assessment, Lewis Publishers, Chelsea, Michigan.

Texas Natural Resource Conservation Commission (TNRCC), 2000, Ecological Risk Assessment and Development of Ecological Protective Concentration Levels. (Chapter 350 - Texas Risk Reduction Program - §350.77)

U.S. Environmental Protection Agency (EPA), 2000, Region 6 Media-Specific Screening Criteria Tables.

U.S. Environmental Protection Agency (EPA), 2000b, Region 4 Ecological Screening Criteria Tables.

U.S. Environmental Protection Agency (EPA), 1998a, Risk Assessment Guidance for Superfund: Volume 1 - Human Health Evaluation Manual (Part D, Standardized Planning, Reporting, and Review of Superfund Risk Assessments). Solid Waste and Emergency Response, Washington, DC. OSWER 9285.7-01D.

U.S. Environmental Protection Agency (EPA), 1998b, Risk Assessment Guidance for Superfund: Volume 1 - Human Health Evaluation Manual, Supplemental Guidance, Dermal Risk Assessment. NCEA-W-0364. External Review Draft.

U.S. Environmental Protection Agency (EPA), 1997a, Exposure Factors Handbook. Office of Health and Environmental Assessment, Washington, DC. PB 98-124217.

U.S. Environmental Protection Agency (EPA) 1997c, Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments. Solid Waste and Emergency Response. EPA 540/R-97/006.

U.S. Environmental Protection Agency (EPA) 1995a, Review of a Methodology for Establishing Risk-Based Soil Remediation Goals for Commercial Areas of the California Gulch Site. Technical Review Workgroup for Lead, October 26, 1995.

U.S. Environmental Protection Agency (EPA), 1995b, Region III Technical Guidance Manual: Assessing Dermal Exposure from Soil. Office of Superfund Programs, Hazardous Waste Management Division. EPA/903/K-95/003.

U.S. Environmental Protection Agency (EPA), 1995c, Supplemental Region VI Risk Assessment Guidance. Dallas, TX.

U.S. Environmental Protection Agency (EPA), 1994a, Uptake/Biokinetic Model, Version 0.99. Office of Health and Environmental Assessment, Cincinnati, OH.

U.S. Environmental Protection Agency (EPA), 1994b, Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. Office of Solid Waste and Emergency Response, Washington, DC. (OSWER Directive #9355.4-12) EPA/540/F-94/043.

U.S. Environmental Protection Agency (EPA), 1994c, Ecological Risk Assessment for Superfund: Process for Designing and Conducting Ecological Risk Assessment, EPA Environmental Response Team, Edison, NJ.

U.S. Environmental Protection Agency (EPA), 1993, Wildlife Exposure Factors Handbook, Office of Research and Development, Washington, DC EPA/600/R-93/187

U.S. Environmental Protection Agency (EPA), 1992a, Statistical Analysis of Ground-Water Monitoring Data at RCRA Facilities, Addendum to Interim Final Guidance, Office of Solid Waste Management Division, Washington, D.C.

U.S. Environmental Protection Agency (EPA), 1992b, Dermal Exposure Assessment: Principles and Applications. Office of Research and Development, Washington, DC. EPA/600/8-91/011B.

U.S. Environmental Protection Agency (EPA), 1992c, Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual, Supplemental Guidance, Dermal Risk Assessment, Interim Guidance. Office of Emergency and Remedial Response, Washington, DC. August 18.

U.S. Environmental Protection Agency (EPA), 1992d, Supplemental Guidance to RAGS: Calculating the Concentration Term. Office of Emergency and Remedial Response, Washington, DC. Publication #9285.7-081.

U.S. Environmental Protection Agency (EPA), 1991a, Risk Assessment Guidance for Superfund, Vol. 1: Human Health Evaluation Manual, Part B, Development of Risk-Based Preliminary Remediation Goals. Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1991b, Risk Assessment Guidance for Superfund, Vol. 1: Human Health Evaluation Manual, Supplemental Guidance: Dermal Risk Assessment Interim Guidance. Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1991c, Risk Assessment Guidance for Superfund, Vol. 1: Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors. Office of Emergency and Remedial Response, Washington, DC. OSWER Directive 9285.6-03.

U.S. Environmental Protection Agency (EPA), 1990, National Oil and Hazardous Substances Pollution Contingency Plan, Final Rule. 40 CFR Part 300, March 8.

U.S. Environmental Protection Agency (EPA), 1989a, Risk Assessment Guidance for Superfund, Vol. 1: Human Health Evaluation Manual, Part A. Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-89/002.

U.S. Environmental Protection Agency (EPA), 1989b, Exposure Factors Handbook. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-89/043.

Wentsel, R. S., T.W. LaPoint, M. Simini, R.T. Checkai, D. Ludwig, and L.W. Brewer, 1996, Tri-Service Procedural Guidelines for Ecological Risk Assessment, Volume 1, prepared by the U.S. Army Edgewood Research, Development, and Engineering Center, Aberdeen Proving Ground, Maryland.

Whitaker, Jr., J.O. 1995, The Audubon Society Field Guide to North American Mammals, Alfred A. Knopf, Inc. New York.

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TAB

TABLES

Table 1
 Selection of Contaminants of Potential Concern, Groundwater
 Former Carswell AFB, Texas
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Chemical	Frequency of Detection	Range of values, mg/L Detected / Nondetected	Statistical Distribution	95% UCL mg/L	Background Criteria mg/L	Screening Criteria mg/L	Exclusion Rationale	COPC?	Representative Concentration mg/L
Inorganics									
Aluminum	12/17(71)	6.5E-02 - 6.8E+00	L	1.9E+00	1.3E+00	3.7E+01	C	N	--
Antimony	2/28(7)	2.9E-03 - 3.8E-03	NP	2.9E-03	NA	1.5E-02	C	N	--
Arsenic	2/28(7)	3.0E-03 - 1.5E-02	NP	3.0E-03	NA	4.5E-05	--	Y	1.5E-02
Barium	27/27(100)	7.8E-02 - 2.1E-01	L	1.4E-01	5.9E-01	2.6E+00	A,C	N	--
Beryllium	2/28(7)	1.0E-03 - 1.1E-03	NP	1.0E-03	3.0E-04	7.3E-02	C	N	--
Calcium	17/17(100)	1.1E+02 - 2.0E+02	L	1.6E+02	2.7E+02	NA	A,D	N	--
Chromium, Total	6/28(21)	8.9E-04 - 1.2E-02	NP	1.2E-02	6.0E-03	NA	--	Y	1.2E-02
Cobalt	2/28(7)	3.8E-03 - 8.2E-03	NP	3.8E-03	NA	2.2E+00	C	N	--
Copper	5/28(18)	1.0E-03 - 7.3E-03	NP	1.0E-03	2.8E-03	1.4E+00	C	N	--
Iron	12/17(71)	5.9E-03 - 7.2E+00	L	6.0E+00	2.2E+01	1.1E+01	C	N	--
Lead	1/28(4)	2.0E-03	NP	2.0E-03	NA	1.5E-02	B,C	N	--
Magnesium	17/17(100)	4.4E+00 - 1.1E+01	N	9.2E+00	3.8E+01	NA	A	N	--
Manganese	16/17(94)	2.5E-03 - 4.3E-01	L	8.1E-01	1.7E+00	1.7E+00	C	N	--
Molybdenum	4/15(27)	2.0E-03 - 2.3E-03	NP	2.3E-03	1.8E-01	1.8E-01	A,C	N	--
Nickel	2/28(7)	5.3E-03 - 1.7E-02	NP	5.3E-03	7.3E-01	7.3E-01	A,C	N	--
Potassium	17/17(100)	1.2E+00 - 4.3E+00	L	2.9E+00	1.5E+01	NA	A,D	N	--
Selenium	1/28(4)	2.1E-03	NP	2.1E-03	7.7E-03	1.8E-01	A,B,C	N	--
Silver	1/28(4)	5.4E-04	NP	5.4E-04	2.0E-04	1.8E-01	B,C	N	--
Sodium	17/17(100)	1.3E+01 - 4.2E+01	N	3.2E+01	1.7E+02	NA	A,D	N	--
Vanadium	9/28(32)	9.0E-04 - 1.5E-02	NP	9.0E-04	1.2E-02	2.6E-01	C	N	--
Zinc	13/28(46)	3.2E-03 - 3.0E-02	NP	1.7E-02	1.2E-01	1.1E+01	A,C	N	--
Volatile Organic Compounds									
1,1-Dichloroethane	13/88(15)	4.0E-04 - 2.0E-03	NP	4.0E-04	NA	8.1E-01	C	N	--
1,1-Dichloroethene	23/88(26)	5.0E-04 - 1.1E-02	NP	5.0E-04	NA	4.6E-05	--	Y	1.1E-02
1,2-Dichlorobenzene	4/79(5)	4.0E-04 - 9.0E-04	NP	4.0E-04	NA	3.7E-01	C	N	--
1,2-Dichloropropane	1/88(1)	9.0E-04	NP	9.0E-04	NA	1.6E-04	B	N	--
1,4-Dichlorobenzene	18/79(23)	4.0E-04 - 1.2E-02	NP	4.0E-04	NA	4.7E-04	--	Y	1.2E-02
Benzene	9/89(10)	2.0E-04 - 9.0E-04	NP	2.0E-04	NA	4.2E-04	--	Y	9.0E-04
Chlorobenzene	16/88(18)	3.0E-04 - 4.0E-03	NP	3.0E-04	NA	3.9E-02	C	N	--
Chloroform	7/88(8)	3.0E-04 - 8.0E-04	NP	3.0E-04	NA	1.6E-04	--	Y	8.0E-04
Chloromethane	1/85(1)	3.0E-03	NP	3.0E-03	NA	1.5E-03	B	N	--
Cis-1,2-Dichloroethylene	60/88(68)	8.0E-04 - 5.1E-01	U	1.7E-02	NA	6.1E-02	--	Y	5.1E-01
Dichlorodifluoromethane	2/73(3)	1.0E-03 - 2.0E-03	NP	1.0E-03	NA	3.9E-01	B,C	N	--
Methyl Isobutyl Ketone (4-methyl-2-pentanone)	1/20(5)	5.0E-04	NP	5.0E-04	NA	1.6E-01	C	N	--
Methylene Chloride	1/76(1)	3.0E-04	NP	3.0E-04	NA	4.3E-03	B,C	N	--
Naphthalene	2/70(3)	2.0E-03 - 2.0E-03	NP	2.0E-03	NA	6.2E-03	B,C	N	--
p-Cymene (p-Isopropyltoluene)	1/79(1)	5.0E-04	NP	5.0E-04	NA	NA	B	N	--
Styrene	1/88(1)	4.0E-04	NP	4.0E-04	NA	1.6E+00	B,C	N	--
Tetrachloroethylene (PCE)	9/87(10)	3.0E-03 - 1.5E-02	NP	3.0E-03	NA	1.1E-03	--	Y	1.5E-02
Trans-1,2-Dichloroethene	40/86(47)	7.0E-04 - 6.5E-02	NP	1.1E-03	NA	1.2E-01	C	N	--
Trichloroethylene (TCE)	61/89(69)	8.0E-04 - 3.3E+00	U	5.6E-02	NA	1.6E-03	--	Y	3.3E+00
Vinyl Chloride	26/86(30)	1.0E-03 - 1.0E-01	NP	1.0E-03	NA	2.0E-05	--	Y	1.0E-01

Table 1
 Selection of Contaminants of Potential Concern, Groundwater
 Former Carswell AFB, Texas
 Page 2 of 2

Chemical	Frequency of Detection	Range of values, mg/L	Statistical Distribution	95% UCL, mg/L	Background mg/L	Screening Criteria mg/L	Exclusion Rationale	COPC?	Representative Concentration mg/L
Semi-Volatile Organic Compounds									
Bis(2-ethylhexyl) phthalate	4/19(21)	2.0E-03 - 2.9E-02	NP	2.9E-02	NA	4.8E-03	--	Y	2.9E-02
Pesticides									
No detected values									
Herbicides									
O,O-Dimethyl Phosphorothioate	12/17(71)	1.1E-03 - 1.3E-02	N	7.8E-03	NA	NA	--	Y	1.3E-02
Dioxins									
No detected values									

* Most recent groundwater data from monitoring wells
 b Statistical Distribution N = Normal distribution, L = Lognormal distribution, U = Undetermined distribution, NP = Nonparametric distribution for data sets with greater than 50% nondetects, NA = distribution not determined if sample size is less than 5
 c 95% Upper Confidence Limit calculated for the indicated distribution NA = sample size is less than 5 and distribution is not calculated
 d Background concentrations for inorganic constituents are from low-stress groundwater samples Sept. 1998
 e Toxicity/concentration screen based on USEPA Region VI PRGs for tap water
 f Rationale for exclusion of chemical as a contaminant of potential concern (COPC)
 A = within background concentration
 B = detection frequency less than 5%
 C = maximum detection is less than screening criteria
 D = essential nutrient
 g N = Chemical is not chosen as a COPC, Y = Chemical is chosen as COPC
 h All detected concentrations at all monitoring well locations are used as exposure point concentrations in the risk assessment.

Table 2
 Selection of Contaminants of Potential Concern Effecting Indoor Air Groundwater
 Former Carswell AFB, Texas
 Page 1 of 1

Chemical	Frequency of Detection	Range of values, mg/L	Statistical Distribution ^b	95% UCL ^c , mg/L	Background ^d , mg/L	Screening Criteria ^e , mg/L	Exclusion Rationale ^f	COPC?
		Detected	Nondetected					
Inorganics								
Arsenic	2/28(7)	3.0E-03 - 1.5E-02	5.0E-03 - 6.0E-01	NP	3.0E-03	6.7E-03	NA	E
Volatile Organic Compounds								
1,1-Dichloroethene	25/92(27)	5.0E-04 - 1.1E-02	5.0E-04 - 2.4E-01	NP	5.0E-04	NA	2.5E-02	C
1,4-Dichlorobenzene	18/82(22)	4.0E-04 - 1.2E-02	3.0E-04 - 2.0E-01	NP	4.0E-04	NA	7.4E+01	C
Benzene	9/93(10)	2.0E-04 - 9.0E-04	4.0E-04 - 2.0E-01	NP	2.0E-04	NA	7.9E-01	C
Chloroform	9/92(10)	3.0E-04 - 8.0E-04	3.0E-04 - 2.0E-01	NP	3.0E-04	NA	4.0E-01	C
Cis-1,2-Dichloroethylene	64/92(70)	8.0E-04 - 7.5E-01	5.0E-04 - 1.2E-03	U	2.1E-02	NA	1.3E+02	C
Tetrachloroethylene(PCE)	10/91(11)	2.0E-03 - 1.5E-02	5.0E-04 - 2.8E-01	NP	2.0E-03	NA	4.1E+00	C
Trichloroethylene (TCE)	65/93(70)	8.0E-04 - 3.3E+00	5.0E-04 - 1.0E-03	U	9.8E-02	NA	2.2E+00	Y
Vinyl Chloride	28/92(30)	1.0E-03 - 1.0E-01	5.0E-04 - 2.2E-01	NP	1.0E-03	NA	1.2E-02	Y
Semi-Volatile Organic Compounds								
Bis(2-ethylhexyl) phthalate	4/19(21)	2.0E-03 - 2.9E-02	1.0E-02 - 1.1E-02	NP	2.9E-02	NA	3.4E-01	C
Pesticides								
No detected values								
Herbicides								
O,O,O-Triethyl Phosphorothioate	12/17(71)	1.1E-03 - 1.3E-02	2.0E-03 - 2.0E-03	N	7.8E-03	NA	NA	E
Dioxins								
No detected values								

^a Data from most recent groundwater monitoring data. Chemicals listed represent those chosen as COPCs
^b Statistical Distribution N = Normal distribution; L = Lognormal distribution; U = Undetermined distribution; NP = Nonparametric distribution for data sets with greater than 50% nondetects; NA = distribution not determined if sample size is less than 5.
^c 95% Upper Confidence Limit calculated for the indicated distribution NA = sample size is less than 5 and distribution is not calculated
^d Background concentrations for inorganic constituents are from low-stress groundwater samples, Sept, 1998.
^e Groundwater concentrations protective of indoor air concentrations calculated using the Johnson and Eitenger Model (EPA, 1998)
^f Rationale for exclusion of chemical as a contaminant of potential concern (COPC)
 A = within background concentration.
 B = detection frequency less than 5%.
 C = maximum detection is less than screening criteria
 D = essential nutrient
 E = Non-volatile
^g N = Chemical is not chosen as a COPC; Y = Chemical is chosen as COPC

Table 3
 Selection of Contaminants of Potential Concern, Surface Water
 Former Carswell AFB, Texas
 Page 1 of 2

Chemical	Frequency of Detection	Range of values, mg/L	Detected	Not Detected	Statistical Distribution	95% UCL	Background	Screening Criteria mg/L	Exclusion Rationale	COPC?	Representative Concentration mg/L
Inorganics											
Aluminum	8/11(73)	1.1E-01 - 3.2E+01	5.0E-01 - 5.0E-01	U	3.2E+01	2.7E-01	NA	NA	--	Y	3.2E+01
Antimony	3/11(27)	3.8E-03 - 7.1E-03	5.0E-03 - 5.0E-03	NP	7.1E-03	3.0E-03	6.0E-03	6.0E-03	--	Y	7.1E-03
Arsenic	1/11(9)	3.3E-02	5.0E-03 - 5.0E-03	NP	3.3E-02	NA	5.0E-02	5.0E-02	C	N	--
Barium	11/11(100)	7.5E-02 - 5.7E-01	NA	U	5.7E-01	1.5E-01	2.0E+00	2.0E+00	C	N	--
Beryllium	1/11(9)	1.6E-03	3.0E-03 - 3.0E-03	NA	NA	NA	4.0E-03	4.0E-03	C	N	--
Calcium	11/11(100)	6.5E+01 - 4.1E+02	NA	L	1.9E+02	1.3E+02	NA	NA	D	N	--
Cobalt	1/11(9)	2.0E-02	7.0E-02 - 7.0E-02	NP	2.0E-02	NA	NA	NA	--	Y	2.0E-02
Copper	3/11(27)	3.3E-03 - 3.2E-02	6.0E-02 - 6.0E-02	NP	3.2E-02	1.0E-02	NA	NA	--	Y	3.2E-02
Iron	11/11(100)	5.7E-02 - 4.6E+01	NA	U	4.6E+01	9.2E-01	NA	NA	--	Y	4.6E+01
Lead	9/11(82)	2.0E-03 - 6.3E-02	5.0E-03 - 5.0E-03	U	6.3E-02	NA	1.5E-02	1.5E-02	--	Y	6.3E-02
Magnesium	11/11(100)	5.4E+00 - 2.1E+01	NA	U	2.1E+01	9.4E+00	NA	NA	--	Y	2.1E+01
Manganese	11/11(100)	5.0E-03 - 3.9E+00	NA	U	3.9E+00	4.2E-01	NA	NA	--	Y	3.9E+00
Mercury	4/11(36)	5.7E-05 - 2.1E-04	1.0E-03 - 1.0E-03	NP	2.1E-04	1.0E-04	2.0E-03	2.0E-03	C	N	--
Nickel	1/11(9)	2.8E-02	1.5E-01 - 1.5E-01	NP	2.8E-02	1.8E-02	1.0E-01	1.0E-01	C	N	--
Potassium	11/11(100)	9.7E-01 - 1.2E+01	NA	U	1.2E+01	6.4E+00	NA	NA	D	N	--
Selenium	1/11(9)	6.8E-03	5.0E-03 - 5.0E-03	NP	6.8E-03	2.5E-03	5.0E-02	5.0E-02	C	N	--
Silver	1/11(9)	7.8E-04	2.0E-04 - 2.0E-04	NP	7.8E-04	3.0E-04	1.8E-01	1.8E-01	C	N	--
Vanadium	4/11(36)	2.4E-03 - 5.8E-02	8.0E-02 - 8.0E-02	NP	5.8E-02	1.6E-02	NA	NA	--	Y	5.8E-02
Zinc	3/11(27)	3.0E-02 - 4.2E-01	2.0E-02 - 2.0E-02	NP	4.2E-01	1.2E-02	NA	NA	--	Y	4.2E-01
Volatile Organic Compounds											
1,1-Dichloroethane	1/11(9)	4.7E-03	4.0E-04 - 8.0E-03	NP	4.7E-03	NA	3.7E+00	3.7E+00	C	N	--
1,1-Dichloroethene	1/11(9)	3.1E-04	1.2E-03 - 2.4E-02	NP	3.1E-04	NA	7.0E-03	7.0E-03	C	N	--
Cis-1,2-Dichloroethylene	11/11(100)	3.2E-04 - 6.4E-02	NA	L	2.6E-01	NA	7.0E-03	7.0E-03	--	Y	6.4E-02
Tetrachloroethylene	1/11(9)	1.1E-03	1.4E-03 - 2.8E-02	NP	1.1E-03	NA	5.0E-03	5.0E-03	C	N	--
Trichloroethylene	10/11(91)	3.5E-04 - 2.0E-01	1.0E-03	L	6.7E+00	NA	5.0E-03	5.0E-03	--	Y	2.0E-01
Vinyl Chloride	1/11(9)	7.3E-03	1.1E-03 - 2.2E-02	NP	7.3E-03	NA	2.0E-03	2.0E-03	--	Y	7.3E-03

Table 3
 Selection of Contaminants of Potential Concern, Surface Water
 Former Carswell AFB, Texas
 Page 2 of 2

Chemical	Frequency of Detection	Range of values, mg/L		Statistical Distribution	95% UCL, mg/L	Background, mg/L	Screening Criteria, mg/L	Exclusion Rationale	COPC?	Representative Concentration, mg/L
		Detected	Nondetected							
Semi-Volatile Organic Compounds										
Bis(2-ethylhexyl) phthalate	3/11(27)	4.4E-03 - 1.2E-02	1.0E-02 - 1.2E-02	NP	1.2E-02	NA	6.1E-03	-	Y	1.2E-02
Diethyl phthalate	1/11(9)	1.5E-03	1.0E-02 - 1.2E-02	NP	1.5E-03	NA	2.9E+01	C	N	-
Di-n-butyl phthalate	2/11(18)	2.1E-03 - 5.8E-03	1.0E-02 - 1.1E-02	NP	5.8E-03	NA	3.7E+00	C	N	-

* Statistical Distribution: N = Normal distribution; L = Lognormal distribution; U = Undetermined distribution; NP = Nonparametric distribution for data sets with greater than 50% nondetects; NA = distribution not determined if sample size is less than 5.
 † 95% Upper Confidence Limit calculated for the indicated distribution. NA = sample size is less than 5 and distribution is not calculated.
 ‡ Background concentrations for inorganic constituents.
 § Toxicity/concentration screen based on EPA Region VI PRGs for tap water (EPA, 2000).
 ¶ Rationale for exclusion of chemical as a contaminant of potential concern (COPC):
 A = within background concentration
 B = detection frequency less than 5%.
 C = maximum detection is less than screening criteria
 D = essential nutrient.
 † N = Chemical is not chosen as a COPC, Y = Chemical is chosen as COPC.
 ‡ Concentration used in risk assessment equals the maximum value.

Table 4
Selection of Contaminants of Potential Concern for Non-Sustainable Fisheries, Surface Water
Former Carswell AFB, Texas

Chemical	Frequency of Detection ^a	Range of values, mg/L		Statistical Distribution ^a	95% UCL ^b mg/L ^c	Background ^d mg/L ^e	Screening Criteria mg/L ^d	Exclusion Rationale ^f	COPC?
		Detected	Nondetected						
Inorganics									
Aluminum	8/11(73)	1.1E-01 - 3.2E+01	5.0E-01 - 5.0E-01	U	3.2E+01	2.7E-01	NA	E	N
Antimony	3/11(27)	3.8E-03 - 7.1E-03	5.0E-03 - 5.0E-03	NP	7.1E-03	3.0E-03	NA	E	N
Cobalt	1/11(9)	2.0E-02	7.0E-02 - 7.0E-02	NP	2.0E-02	NA	NA	E	N
Copper	3/11(27)	3.3E-03 - 3.2E-02	6.0E-02 - 6.0E-02	NP	3.2E-02	1.0E-02	NA	E	N
Iron	11/11(100)	5.7E-02 - 4.6E+01	NA	U	4.6E+01	9.2E-01	NA	E	N
Lead	9/11(82)	2.0E-03 - 6.3E-02	5.0E-03 - 5.0E-03	U	6.3E-02	NA	1.7E-01	C	N
Magnesium	11/11(100)	5.4E+00 - 2.1E+01	NA	U	2.1E+01	9.4E+00	NA	E	N
Manganese	11/11(100)	5.0E-03 - 3.9E+00	NA	U	3.9E+00	4.2E-01	NA	E	N
Vanadium	4/11(36)	2.4E-03 - 5.8E-02	8.0E-02 - 8.0E-02	NP	5.8E-02	1.6E-02	NA	E	N
Zinc	3/11(27)	3.0E-02 - 4.2E-01	2.0E-02 - 2.0E-02	NP	4.2E-01	1.2E-02	NA	E	N
Volatile Organic Compounds									
Cis-1,2-Dichloroethylene	11/11(100)	3.2E-04 - 6.4E-02	NA	L	2.6E-01	NA	7.0E-03	E	N
Trichloroethylene	10/11(91)	3.5E-04 - 2.0E-01	1.0E-03	L	6.7E+00	NA	4.1E+00	C	N
Vinyl Chloride	1/11(9)	7.3E-03	1.1E-03 - 2.2E-02	NP	7.3E-03	NA	2.8E+00	C	N
Semi-Volatile Organic Compounds									
Bis(2-ethylhexyl) phthalate	3/11(27)	4.4E-03 - 1.2E-02	1.0E-02 - 1.2E-02	NP	1.2E-02	NA	6.1E-03	E	N

^a Statistical Distribution: N = Normal distribution, L = Lognormal distribution; U = Undetermined distribution; NP = Nonparametric distribution for data sets with greater than 50% NA = distribution not determined if sample size is less than 5.
^b 95% Upper Confidence Limit calculated for the indicated distribution. NA = sample size is less than 5 and distribution is not calculated.
^c Background concentrations for inorganic constituents.
^d Toxicity/concentration screen based on criteria for the protection of non-sustainable fisheries (TNRCC, 2000).
^e Rationale for exclusion of chemical as a contaminant of potential concern (COPC):
 A = within background concentration.
 B = detection frequency less than 5%.
 C = maximum detection is less than screening criteria.
 D = essential nutrient.
 E = no criteria available
^f N = Chemical is not chosen as a COPC; Y = Chemical is chosen as COPC

Table 5
 Selection of Contaminants of Potential Concern, Sediment
 Former Carswell AFB, Texas
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Chemical	Frequency of Detection	Range of values Detected	Non Detected	Statistical Distribution	95% UCL mg/kg	Background mg/kg	Screening Criteria mg/kg	Exclusion Rationale	COPC?	Representative Concentration mg/kg
Inorganics										
Aluminum	12/12(100)	8.9E+02 - 1.3E+04	NA	L	7.5E+03	2.0E+04	NA	A	N	--
Antimony	10/12(83)	1.7E-01 - 6.5E+00	6.1E-01 - 1.1E+00	U	6.5E+00	3.3E-01	6.0E-01	--	Y	6.5E+00
Arsenic	12/12(100)	5.5E+00 - 3.7E+01	NA	L	1.6E+01	8.0E+00	5.0E+00	--	Y	1.6E+01
Barium	12/12(100)	1.2E+01 - 7.9E+02	NA	L	1.0E+03	1.4E+02	2.0E+02	--	Y	7.9E+02
Beryllium	12/12(100)	1.6E-01 - 5.5E-01	NA	L	4.4E-01	7.2E-01	4.0E-01	A	N	--
Cadmium	9/12(75)	7.8E-02 - 5.9E-01	5.1E+00 - 5.5E+00	L	3.0E+00	1.8E+00	5.0E-01	A	N	--
Calcium	12/12(100)	2.6E+04 - 3.6E+05	NA	N	2.4E+05	3.6E+05	NA	A,D	N	--
Cobalt	12/12(100)	1.4E+00 - 2.2E+01	NA	L	1.3E+01	4.8E+01	NA	A	N	--
Copper	12/12(100)	1.9E+00 - 8.6E+00	NA	L	6.0E+00	1.7E+01	NA	A	N	--
Iron	12/12(100)	5.7E+02 - 2.5E+04	NA	N	1.4E+04	1.0E+04	NA	--	Y	1.4E+04
Lead	12/12(100)	5.2E+00 - 7.8E+01	NA	L	4.2E+01	8.8E+01	1.5E+00	A	N	--
Magnesium	12/12(100)	1.0E+03 - 3.1E+03	NA	N	2.5E+03	2.6E+03	NA	--	Y	2.5E+03
Manganese	12/12(100)	1.7E+02 - 2.1E+03	NA	L	1.6E+03	3.5E+02	NA	--	Y	1.6E+03
Mercury	12/12(100)	1.2E-02 - 1.2E-01	NA	U	1.2E-01	3.6E-02	2.0E-01	C	N	--
Nickel	12/12(100)	2.7E+00 - 2.1E+01	NA	L	1.3E+01	1.6E+00	1.0E+01	--	Y	1.3E+01
Potassium	12/12(100)	1.3E+02 - 1.5E+03	NA	L	1.1E+03	5.0E+03	NA	A,D	N	--
Sodium	12/12(100)	8.8E+01 - 2.7E+02	NA	N	2.1E+02	6.1E+00	NA	D	N	--
Vanadium	12/12(100)	1.4E+01 - 1.1E+02	NA	L	4.9E+01	3.2E+01	NA	--	Y	4.9E+01
Zinc	8/12(67)	1.3E+01 - 1.6E+02	2.2E+01 - 4.2E+01	U	1.6E+02	8.1E+01	NA	--	Y	1.6E+02
Volatile Organic Compounds										
Cis-1,2-Dichloroethylene	5/12(42)	2.7E-03 - 1.2E-02	6.7E-03 - 1.2E-02	NP	1.2E-02	NA	7.0E-02	C	N	--
Toluene	1/1(9)	1.4E-02	5.9E-03 - 9.7E-03	NP	1.4E-02	NA	1.0E+02	C	N	--
Trichloroethylene	7/12(58)	2.1E-03 - 5.6E-02	1.1E-02 - 1.9E-02	L	2.4E-02	NA	5.0E-01	C	N	--

Table 5
 Selection of Contaminants of Potential Concern, Sediment
 Former Carswell AFB, Texas
 Page 2 of 2

Chemical	Frequency of Detection		Range of values, mg/kg		Statistical Distribution ^a	95% UCL ^b , mg/kg	Background, mg/kg ^c	Screening Criteria mg/kg ^d	Exclusion Rationale ^e	COPC? ^f	Representative Concentration mg/kg ^g
	Detected	Nondetected	Detected	Nondetected							
Semi-Volatile Organic Compounds											
Benzofluoranthene	1/12(8)	2 5E-01	7 2E-01 - 9 2E-01		NP	2 5E-01	NA	NA	--	Y	2 5E-01
Benzofluoranthene	1/12(8)	4 2E-01	7 2E-01 - 9 2E-01		NP	4 2E-01	NA	NA	--	Y	4 2E-01
Benzofluoranthene	1/12(8)	3 5E-01	7 2E-01 - 9 2E-01		NP	3 5E-01	NA	NA	--	Y	3 5E-01
Benzofluoranthene	1/12(8)	3 6E-01	7 2E-01 - 9 2E-01		NP	3 6E-01	NA	NA	--	Y	3 6E-01
Chrysene	1/12(8)	4 2E-01	7 2E-01 - 9 2E-01		NP	4 2E-01	NA	NA	--	Y	4 2E-01
Fluoranthene	1/12(8)	8 7E-01	7 2E-01 - 9 2E-01		NP	8 7E-01	NA	1 5E+02	C	N	--
Indeno(1,2,3-cd)pyrene	1/12(8)	3 0E-01	7 2E-01 - 9 2E-01		NP	3 0E-01	NA	NA	--	Y	3 0E-01
Pyrene	1/12(8)	6 2E-01	7 2E-01 - 9 2E-01		NP	6 2E-01	NA	1 1E+02	C	N	--

^a Statistical Distribution: N = Normal distribution, L = Lognormal distribution, U = Undetermined distribution, NP = Nonparametric distribution for data sets with greater than 50% nondetects.

NA = distribution not determined if sample size is less than 5

^b 95% Upper Confidence Limit calculated for the indicated distribution. NA = sample size is less than 5 and distribution is not calculated

^c Background concentrations for inorganic constituents

^d Toxicity/concentration screen, based on EPA Region VI PRGs for residential soil (EPA, 2000)

^e Rationale for exclusion of chemical as a contaminant of potential concern (COPC)

A = within background concentration

B = detection frequency less than 5%

C = maximum detection is less than screening criteria

D = essential nutrient.

^f N = Chemical is not chosen as a COPC; Y = Chemical is chosen as COPC.

^g Concentration used in risk assessment equal to 95% UCL or maximum value, if maximum value is less than UCL or if no UCL is calculated.

Table 6
Parameters Used to Estimate Potential Exposures
For Groundwater Receptors^{a,b}

Pathway Parameter	Age-Adj. Resident	Child Resident	Construction Worker
Ingestion of Groundwater			
IR (L/day)	18 ^d	1	0.1 ^c
FI (unitless)	1.0	1.0	1.0
EF (days/year)	350	350	250
ED (years)	30 ^d	6	1 ^c
BW (kg)	59	15	70
AT-Noncancer (days)	10950 ^e	2190 ^e	250 ^e
AT-Cancer (days)	25550 ^f	25550 ^f	25550 ^f
Inhalation of Volatiles from Household Uses of Groundwater			
IR (m ³ /day)	15	10	15
EF (days/year)	350	350	250
ED (years)	30 ^d	6	1 ^c
BW (kg)	59	15	70
AT-Noncancer (days)	10950 ^e	2190 ^e	365 ^e
AT-Cancer (days)	25550 ^f	25550 ^f	25550 ^f
Dermal Contact with Groundwater			
SA (cm ²)	20090 ^g	5000	2200 ^g
EF (days/year)	350	350	250
ED (years)	30 ^d	6	1 ^c
BW (kg)	59	15	70
AT-Noncancer (days)	10950 ^e	2190 ^e	250 ^e
AT-Cancer (days)	25550 ^f	25550 ^f	25550 ^f
Kp (cm/hour)	Csv ^h	Csv ^h	csv ^h
B (unitless)	Csv ^h	Csv ^h	csv ^h
ET (hours)	0.2	0.2	4 ^c
t* (hours)	Csv ^h	Csv ^h	Csv ^h

- ^a Parameter values are intended to characterize the reasonable maximum exposure. The age-adjusted resident is used to evaluate carcinogenic groundwater constituents and the child resident is used to evaluate noncarcinogenic groundwater constituents.
- ^b Parameter values obtained from EPA (1991), unless otherwise noted.
- ^c Best professional judgment.
- ^d EPA (1997a and 1999). "Resident" is a time-weighted-average adult and child resident. Exposure parameters for the resident are calculated based on default values for the adult and child.
- ^e Calculated as the product of ED (years) x 365 days/year.
- ^f Calculated as the product of 70 years (assumed lifetime) x 365 days/year.
- ^g EPA (1997a). Surface area for the resident includes the entire body surface area. Surface area for the construction worker includes hands and feet.
- ^h Chemical specific value.

Table 7
Parameters Used to Estimate Potential Exposures
For Surface Water Receptors^{a,b}

Pathway Parameter	Trespasser	Maintenance Worker	Recreational User
<i>Incidental Ingestion of Surface Water</i>			
Ingestion Rate (L/day)	0.005 ^d	0.005 ^d	0.005 ^d
Exposure Frequency (days/year)	12 ^d	12 ^d	24 ^d
Exposure Duration (years)	6 ^d	24 ^d	10 ^d
Body Weight (kg)	56	70	70
Averaging Time-Noncancer (days)	2,190 ^e	8,760 ^e	3,650 ^e
Averaging Time-Cancer (days)	25,550 ^f	25,550 ^f	25,550 ^f
<i>Dermal Exposures to Surface Water</i>			
Skin Surface Area (cm ²)	980 ^g	1,120 ^g	1,120 ^g
Exposure Frequency (days/year)	12 ^d	12 ^d	24 ^d
Exposure Duration (years)	6 ^d	24 ^d	10 ^d
Body Weight (kg)	56	70	70
Averaging Time-Noncancer (days)	2,190 ^e	8,760 ^e	3,650 ^e
Averaging Time-Cancer (days)	25,550 ^f	25,550 ^f	25,550 ^f

^a Parameter values are intended to characterize the RME.

^b Parameter values obtained from EPA (1991c), unless otherwise noted.

^c EPA (1989a).

^d Best professional judgment.

Ingestion rate estimated as 1/10th the volume of incidental ingestion while wading.

Exposure Frequency: Assumes that the recreational user will visit the site 2 days of every month; trespasser will visit once a month; and maintenance worker will work in the water bodies once a month.

Exposure Duration: Assumes that the recreational user will visit the site for 10 years; the trespasser will visit during the 6 years between age 13 and 18; and the maintenance worker will work for a traditional 24 year working age.

^e Calculated as the product of ED (years) x 365 days/year.

^f Calculated as the product of 70 years (assumed lifetime) x 365 days/year.

^g Based on the surface area of adult hands for the maintenance worker and recreational user and teenage hands and feet for the trespasser.

Table 8
Parameters Used to Estimate Potential Exposure
For Sediment Receptors

Pathway Parameter	Trespasser	Maintenance Worker	Recreational User
Incidental Ingestion of Sediment			
Ingestion Rate (mg/day)	5 ^a	5 ^a	5 ^a
Fraction Ingested (unitless)	1 ^a	1 ^a	1 ^a
Sediment Exposure Frequency (day/yr)	12 ^a	12 ^a	24 ^a
Exposure Duration (years)	6 ^a	24 ^a	10 ^a
Body Weight (kg)	56	70	70
Averaging Time-Noncancer (days)	2,190 ^b	8,760 ^b	3,650 ^b
Averaging Time-Cancer (days)	25,550 ^c	25,550 ^c	25,550 ^c
Dermal Exposures to Sediment			
Skin Surface Area (cm ²)	980 ^d	1,120 ^d	1,120 ^d
Skin Adherence Factor (mg/cm ²)	0.3 ^e	0.08 ^e	0.08 ^e
Absorption Factor (unitless)	Chemical-specific ^f	Chemical-specific ^f	Chemical-specific ^f
Exposure Frequency (days/year)	12 ^a	12 ^a	24 ^a
Exposure Duration (years)	6 ^a	24 ^a	10 ^a
Body Weight (kg)	56	70	70 ^d
Averaging Time-Noncancer (days)	2,190 ^b	8,760 ^b	3,650 ^b
Averaging Time-Cancer (days)	25,550 ^c	25,550 ^c	25,550 ^c

^a Best professional judgment.

Ingestion rate is 1/10th of the adult soil ingestion rate.

Fraction Ingested: For RME, it is assumed that 100 percent of the sediment ingested on days that the site is visited.

Exposure Frequency: Assumes the recreational user visits the site two days each month; trespasser will visit once a month; and maintenance worker will work in the water body once a month.

Exposure Duration: Assumes the recreational user visits the site for 10 years; trespasser will visit during the 6 years between age 13 and 18; and the maintenance worker will work for a traditional 24 year working age.

^b Calculated as the product of ED (years) x 365 days/year.

^c Calculated as the product of 70 years (assumed lifetime) x 365 days/year.

^d EPA (1997a). Surface area based on adult hand surface area for recreational user and maintenance worker and teenage hand for trespasser.

^e EPA, 1998b Adherence factor for trespasser based on child default value; value for maintenance worker and recreational user is based on adult default value.

^f EPA, 1998b

Table 9
Non-Cancer Toxicity Data — Oral/Dermal

Chemical of Potential Concern	Chronic/Subchronic	Oral RfD Value	Oral RfD Units	Oral to Dermal Adjustment Factor (1)	Adjusted Dermal RfD (2)	Units	Primary Target Organ	Combined Uncertainty Modifying Factors	Sources of RfD Target Organ	Dates of RfD Target Organ (3) (MM/DD/YYYY)
Inorganics										
Aluminum	Chronic	1E+00	mg/kg-day	27%	2.7E-01	mg/kg-day	Developmental Neurotoxicity	100	NCEA	8/28/96
Antimony	Chronic	4E-04	mg/kg-day	15%	6.0E-05	mg/kg-day	Blood Chemistry	1,000	IRIS	10/19/99 2/1/91
Arsenic	Chronic	3E-04	mg/kg-day	100%	3.0E-04	mg/kg-day	Skin, Vascular Effects	3	IRIS	10/19/99 2/1/93
Barium	Chronic	7E-02	mg/kg-day	7%	4.9E-03	mg/kg-day	Kidney	100	IRIS	10/19/99 3/30/98
Chromium (VI)	Chronic	3E-03	mg/kg-day	2%	6.0E-05	mg/kg-day	NOAEL	900	IRIS	10/19/99 9/3/98
Cobalt	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Copper	Chronic	4E-02	mg/kg-day	57%	2.3E-02	mg/kg-day	<Body Weight	N/A	NCEA	N/A
Iron	Chronic	3E-01	mg/kg-day	15%	4.5E-02	mg/kg-day	GI Irritation	1	NCEA	7/23/96
Lead	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Magnesium	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Manganese	Chronic	5E-02	mg/kg-day	100%	4.7E-02	mg/kg-day	CNS	3	IRIS	10/19/99 5/1/96
Nickel	Chronic	2E-02	mg/kg-day	10%	2.0E-03	mg/kg-day	<Body Weight	300	IRIS	10/19/99 12/1/96
Vanadium	Chronic	7E-03	mg/kg-day	3%	2.1E-04	mg/kg-day	NOAEL	100	HEAST	1997
Zinc	Chronic	3E-01	mg/kg-day	100%	3.0E-01	mg/kg-day	Blood Chemistry	10	IRIS	10/19/99 10/1/92
VOCs										
1,1-Dichloroethene	Chronic	9E-03	mg/kg-day	100%	9.0E-03	mg/kg-day	Liver	1,000	IRIS	11/21/00 4/1/89
1,4-dichlorobenzene	Chronic	2E-01	mg/kg-day	100%	2.3E-01	mg/kg-day	---	---	EPA 2000	2000
Benzene	Chronic	3E-03	mg/kg-day	100%	3.0E-03	mg/kg-day	Blood, Immune System	3,000	NCEA	10/19/99 7/2/96
Chloroform	Chronic	1E-02	mg/kg-day	100%	1.0E-02	mg/kg-day	Liver	1,000	IRIS	10/19/99 9/1/92
cis-1,2-Dichloroethene	Chronic	1E-02	mg/kg-day	100%	1.0E-02	mg/kg-day	Blood Chemistry	3,000	HEAST	1997
Tetrachloroethene	Chronic	1E-02	mg/kg-day	100%	1.0E-02	mg/kg-day	Liver, >Body Weight	1,000	IRIS	10/19/99 3/1/88
Trichloroethene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Vinyl chloride	Chronic	3E-03	mg/kg-day	100%	3.0E-03	mg/kg-day	Liver	30	IRIS	11/10/00 8/7/00
SVOCs										
Benzofluoranthracene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(a)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(b)fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Benzo(g,h,i)fluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Bis(2-ethylhexyl)phthalate	Chronic	2E-02	mg/kg-day	100%	2.0E-02	mg/kg-day	Liver	1,000	IRIS	11/21/00 1/31/87
Chrysene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Indeno(1,2,3-cd)pyrene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Herbicides										
O,O,O-Trimethyl phosphorothioate	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

N/A = Not Available
 IRIS = Integrated Risk Information System
 HEAST = Health Effects Assessment Summary Tables
 NCEA = National Center for Environmental Assessment
 EPA 2000 = EPA Region VI RBC table
 (1) Refer to RAGS, Part A
 (2) Dermal RfD = Oral RfD x Oral Adjustment Factor
 (3) For IRIS values, the date IRIS was searched and the date of the most recent review are provided
 For HEAST values, the date of HEAST is provided
 For NCEA values, the date of the article provided by NCEA is provided
 (4) Inhalation Slope Factor = Unit Risk x Adjustment Factor

Table 10
Non-Cancer Toxicity Data -- Inhalation

Chemical of Potential Concern	Chronic/ Subchronic	Value Inhalation RfC	Units	Adjusted Inhalation RfD (1)	Units	Primary Target Organ	Combined Uncertainty/ Modifying Factors	Sources of RfC/RfD Target Organ	Dates (2) (MM/DD/YY)
Inorganics									
Arsenic	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Chromium VI	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
VOCs									
1,1-Dichloroethene	---	---	---	9 0E-03	mg/kg-day	---	---	EPA 2000	2000
1,4-dichlorobenzene	---	---	---	2 3E-01	mg/kg-day	---	---	EPA 2000	2000
Benzene	Chronic	6E-03	mg/m ³	1 7E-03	mg/kg-day	Blood Chemistry	1,000	NCEA	7/2/96
Chloroform	Chronic	3E-04	mg/m ³	8 6E-05	mg/kg-day	N/A	N/A	NCEA	No Date
cis-1,2-Dichloroethene	---	---	---	1 0E-02	mg/kg-day	---	---	EPA 2000	2000
Tetrachloroethene	Chronic	5E-01	mg/m ³	1 1E-01	mg/kg-day	N/A	N/A	NCEA	No Date
Trichloroethene	---	---	---	6 0E-03	mg/kg-day	---	---	EPA 2000	2000
Vinyl Chloride	Chronic	1 E-01	mg/m ³	2 9E-02	mg/kg-day	Liver	30	IRIS	8/7/00
SVOCs									
Bis(3-ethylhexyl)phthalate	---	---	---	9 0E-03	mg/kg-day	---	---	EPA 2000	2000
Herbicides									
O,O-Dimethyl phosphorothioate	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

N/A = Not Available
 IRIS = Integrated Risk Information System
 NCEA = National Center for Environmental Assessment
 EPA 2000 = EPA Region VI RfC table
 (1) The adjusted inhalation RfD was derived from the RfC value assuming a 70 kg adult inhales 20 m³/day as follows RfD = RfC * (20 m³/day / 70 kg)
 (2) For HEAST values, the date of HEAST is provided
 For NCEA values, the date of the article provided by NCEA is provided
 (3) Inhalation Slope Factor = Unit Risk x Adjustment Factor

Table 11
Cancer Toxicity Data -- Oral/Dermal

Chemical of Potential Concern	Oral Cancer Slope Factor	Oral to Dermal Adjustment Factor	Adjusted Dermal Cancer Slope Factor (1)	Units	Weight of Evidence Cancer Guideline Description	Source	Date (2) (MM/DD/YY)
Inorganics							
Aluminum	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Antimony	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Arsenic	1.5E+00	100%	1.5E+00	(mg/kg-day)	A	IRIS	10/19/99 4/10/98
Barium	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Chromium (VI)	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Cobalt	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Copper	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Iron	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Lead	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Magnesium	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Manganese	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Nickel	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Vanadium	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Zinc	N/A	N/A	N/A	N/A	N/A	N/A	N/A
VOCs							
1,1-Dichloroethene	6.0E-01	100%	6.0E-01	(mg/kg-day)	C	IRIS	11/21/00 2/1/98
1,4-Dichlorobenzene	2.4E-02	100%	2.4E-02	(mg/kg-day)	C	HEAST	1997
Benzene	2.9E-02	100%	2.9E-02	(mg/kg-day)	A	IRIS	10/19/99 2/1/94
Chloroform	6.1E-03	100%	6.1E-03	(mg/kg-day)	B2	IRIS	10/19/99 3/1/91
cis-1,2-Dichloroethene	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Tetrachloroethene	5.2E-02	100%	5.2E-02	(mg/kg-day)	---	NCEA	No Date
Trichloroethene	1.1E-02	100%	1.1E-02	(mg/kg-day)	---	NCEA	No Date
Vinyl chloride	1.4E+00	100%	1.4E+00	(mg/kg-day)	A	IRIS	11/10/00 8/7/00
SVOCs							
Benzofuran	7.3E-01	100%	N/A	(mg/kg-day)	B2	IRIS	10/19/99 3/1/94
Benzofluoranthene	7.3E-01	100%	N/A	(mg/kg-day)	B2	IRIS	10/19/99 11/1/94
Benzofluoranthene	7.3E-01	100%	N/A	(mg/kg-day)	B2	IRIS	10/19/99 3/1/94
Benzofluoranthene	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Bis(2-ethylhexyl)phthalate	1.4E-02	100%	1.4E-02	(mg/kg-day)	B2	IRIS	11/21/00 2/1/93
Chrysene	7.3E-01	100%	N/A	(mg/kg-day)	B2	IRIS	11/21/00 3/1/94
Indeno(1,2,3-cd)pyrene	7.3E-01	100%	N/A	(mg/kg-day)	B2	IRIS	10/19/99 3/1/94
Herbicides							
O,O,O-Trimethyl phosphorothioate	N/A	N/A	N/A	N/A	N/A	N/A	N/A

N/A = Not Available
 IRIS = Integrated Risk Information System
 HEAST = Health Effects Assessment Summary Tables
 NCEA = National Center for Environmental Assessment
 (1) The equation for deriving the adjusted dermal cancer slope factors are presented in the text
 (2) For IRIS values, the date IRIS was searched and the date of the most recent review are provided
 For HEAST values, the date of HEAST is provided
 For NCEA values, the date of the article provided by NCEA is provided
 (3) Inhalation Slope Factor = Unit Risk x Adjustment Factor
 EPA Group
 A - Human carcinogen
 B1 - Probable human carcinogen - indicates that limited human data are available
 B2 - Probable human carcinogen - indicates sufficient evidence in animals and

Table 12
Cancer Toxicity Data -- Inhalation

Chemical Of Potential Concern	Unit Risk	Units	Adjustment	Inhalation Cancer Slope Factor	Units	Weight of Evidence/ Cancer Guideline Description	Source	Date (1) (MM/DD/YY)
Inorganics								
Arsenic	4.3E-03	(ug/m ³)-1	3500	1.5E+01	(mg/kg-day) ⁻¹	A	IRIS	11/10/00:4/10/98
Chromium	1.2E-02	(ug/m ³)-1	3500	2.9E+02	(mg/kg-day) ⁻¹	A	IRIS	10/21/99:9/3/98
VOCs								
1,1-Dichloroethene	5.00E-05	(ug/m ³) ⁻¹	3500	2E-08	(mg/kg-day) ⁻¹	C	IRIS	11/21/00:2/1/98
1,4-dichlorobenzene	---	---	---	2.4E-02	(mg/kg-day) ⁻¹	A	EPA 2000	2000
Benzene	7.80E-06	(ug/m ³) ⁻¹	3500	2.7E-02	(mg/kg-day) ⁻¹	A	IRIS	10/21/99:10/16/98
Chloroform	2.30E-05	(ug/m ³) ⁻¹	3500	8.1E-02	(mg/kg-day) ⁻¹	B2	IRIS	10/21/99:3/1/91
cs-1,2-Dichloroethene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
trans-1,2-Dichloroethene	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Tetrachloroethene	5.80E-07	(ug/m ³) ⁻¹	3500	2.0E-03	(mg/kg-day) ⁻¹	---	NCEA	No Date
Trichloroethene	1.70E-06	(ug/m ³) ⁻¹	3500	6.0E-03	(mg/kg-day) ⁻¹	---	NCEA	No Date
Vinyl Chloride	---	---	---	3.1E-02	(mg/kg-day) ⁻¹	A	EPA 2000	2000
SVOCs								
Bis(3-ethylhexyl)phthalate	---	---	---	1.4E-02	(mg/kg-day) ⁻¹	---	EPA 2000	2000
Herbicides								
O,O,O-Triethyl phosphorothioate	NA	NA	NA	NA	NA	NA	NA	NA

IRIS = Integrated Risk Information System
 HEAST = Health Effects Assessment Summary Tables
 NCEA = National Center for Environmental Assessment
 EPA 2000 = EPA Region VI RBC table.

(1) For IRIS values, the date IRIS was searched and the date of the most recent review are provided
 For HEAST values, the date of HEAST is provided
 For NCEA values, the date of the article provided by NCEA is provided.

(2) Inhalation Slope Factor = Unit Risk x Adjustment Factor.

- EPA Group:
- A - Human carcinogen
 - B1 - Probable human carcinogen - indicates that limited human data are available
 - B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans
 - C - Possible human carcinogen
 - D - Not classifiable as a human carcinogen
 - E - Evidence of noncarcinogenicity

Table 13
Unit Risk Values for Carcinogenic Groundwater COPCs^a
Former Carswell AFB, TX

Chemicals of Concern	Receptors	
	Age-Adjusted Adult Residents	Construction Workers
<i>Inorganics</i>		
Arsenic	1.9e-2	6.2e-4
Chromium VI	-- ^b	--
<i>Volatile Organics</i>		
Benzene	1.8e-3	2.9e-5
Chloroform	4.3e-3	8.5e-5
1,4-Dichlorobenzene	1.6e-3	2.6e-5
1,1-Dichloroethene	1.8e-2	2.1e-4
Cis-1,2-Dichloroethene	--	--
Tetrachloroethene	1.2e-3	6.8e-6
Trichloroethene	4.7e-4	6.7e-6
Vinyl chloride	2.0e-2	6.6e-5
<i>Semivolatile Organics</i>		
Bis(2-ethylhexyl)phthalate	5.4e-4	6.7e-4

^a values represent risk per mg/L.

^b -- = not a carcinogenic COPC.

(1) Equations used to calculate Unit Risk and Unit Hazard values are presented in Section 2.6, and spreadsheets providing chemical-specific parameter values in Appendix C.

Table 14
Unit Hazard Values for Noncarcinogenic Groundwater COPCs^a
Former Carswell AFB, TX

Chemicals of Concern	Receptors	
	Child Residents	Construction Workers
<i>Inorganics</i>		
Arsenic	2.7e2	1.7e1
Chromium VI	2.6e1	2.6e0
<i>Volatile Organics</i>		
Benzene	2.2e2	6.4e1
Chloroform	3.7e3	1.2e3
1,4-Dichlorobenzene	3.7e0	5.4e-1
1,1-Dichloroethene	4.4e1	1.2e1
Cis-1,2-Dichloroethene	3.9e1	1.1e1
Tetrachloroethene	1.7e1	1.9e0
Trichloroethene	6.7e1	1.9e1
Vinyl chloride	3.4e1	3.8e1
<i>Semivolatile Organics</i>		
Bis(2-ethylhexyl)phthalate	1.5e1	1.4e0

^a values represent hazard per mg/L.

(1) Equations used to calculate Unit Risk and Unit Hazard values are presented in Section 2.6, and spreadsheets providing chemical-specific parameter values in Appendix C.

Table 15
Summary of Receptor Risks and Hazards for COPCs
Reasonable Maximum Exposure
Former Carswell AFB, Texas

Scenario Timeline Receptor Population Receptor Age	Exposure Medium	Exposure Point	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			Exposure Routes Total		
			Ingestion	Inhalation	Dermal	Ingestion	Inhalation	Dermal			
Site-Wide	Surface Water	Site-Wide	Be(2-ethylhexyl)phthalate	4 2E-11	3 5E-09	3 5E-09	1 8E-06	---	1 4E-04	1 4E-04	
			cs-1,2-Dichloroethene	5 5E-10	---	---	1 9E-05	---	3 4E-05	5 3E-05	
			Trichloroethene	2 6E-09	5 2E-09	5 2E-09	---	---	1 5E-05	2 2E-05	
			Vinyl Chloride	---	---	---	9 4E-05	---	1 8E-05	1 1E-04	
			Aluminum	---	---	---	5 2E-05	---	6 8E-05	1 2E-04	
			Antimony	---	---	---	---	---	---	---	
			Cobalt	---	---	---	2 5E-06	---	5 0E-07	3 0E-06	
			Copper	---	---	---	4 8E-04	---	8 8E-05	5 4E-04	
			Iron	---	---	---	---	---	---	---	
			Lead	---	---	---	---	---	---	---	
			Manganese	---	---	---	---	---	---	---	
			Manganese	---	---	---	2 4E-04	---	8 0E-04	1 0E-03	
			Vanadium	---	---	---	2 4E-05	---	4 8E-04	5 0E-04	
			Zinc	---	---	---	4 1E-06	---	8 1E-07	4 8E-06	
			(Total)	3 2E-09	1 2E-08	1 5E-08	0 0009	---	0 0018	0 0025	
Site-Wide	Sediment	Site-Wide	Benzo(a)anthracene	4 6E-11	3 5E-10	3 5E-10	---	---	---	---	
			Benzo(b)pyrene	7 7E-10	5 8E-09	5 8E-09	---	---	---	---	
			Benzo(k)fluoranthene	6 4E-11	4 9E-10	4 9E-10	---	---	---	---	
			Benzo(g,h,i)perylene	---	---	---	---	---	---	---	
			Chrysene	7 7E-13	5 9E-12	5 9E-12	---	---	---	---	
			Indeno(1,2,3-cd)pyrene	5 5E-11	4 2E-10	4 2E-10	---	---	---	---	
			Antimony	---	---	---	---	---	---	---	
			Arsenic	---	---	---	---	---	---	---	
			Barium	6 0E-09	1 1E-08	1 1E-08	4 8E-05	---	1 8E-04	2 4E-04	
			Iron	---	---	---	1 8E-04	---	2 8E-04	4 4E-04	
			Magnesium	---	---	---	3 3E-05	---	2 8E-04	3 1E-04	
			Manganese	---	---	---	1 4E-04	---	8 1E-05	2 2E-04	
			Nickel	---	---	---	---	---	---	---	
			Vanadium	---	---	---	1 0E-04	---	9 6E-04	1 1E-03	
			Zinc	---	---	---	1 9E-06	---	2 8E-05	3 0E-05	
(Total)	6 9E-09	1 8E-08	2 5E-08	0 0005	---	0 0022	0 0027				
Total Risk Across All Media and All Exposure Routes			Total Risk Across [Surface Water]			Total Hazard Index Across All Media and All Exposure Routes			Total [Liver] HI = 0 00016		
			1 5E-08						Total [Blood Chemistry] HI = 0 0004		
			2 5E-08						Total [Kidney] HI = 0 0003		
			4 1E-08						Total [Developmental Neurotoxicity] HI = 0 0001		
									Total [<Body Weight] HI = 0 000003		
									Total [GI Irritation] HI = 0 0008		
									Total [Skin, Vascular Effects] HI = 0 0004		
									Total [Organ Weight] HI = 0 000003		

N/A = Not Applicable
 GI = Gastrointestinal
 CNS = Central Nervous System
 NOAEL = No Observed Adverse Effect Level

Table 16
Summary of Receptor Risks and Hazards for COPCs
Reasonable Maximum Exposure
Former Carswell AFB, Texas

Scenario Timeframe Receptor Population Receptor Age	Current/Future Exposure Point Adult	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient						
		Chemical	Ingestion	Inhalation	Dermal	Exposure Routes Total	Chemical	Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Site-Wide	Bis(2-ethylhexyl)phthalate	1.4E-10	---	1.3E-08	1.9E-08	---	---	1.4E-06	---	1.3E-04	1.3E-04
		cis-1,2-Dichloroethene	---	---	---	---	---	---	1.5E-05	---	3.1E-05	4.6E-05
		Trichloroethene	1.8E-09	---	1.3E-08	1.9E-08	---	---	---	---	---	---
		Vinyl Chloride	8.2E-09	---	---	---	---	---	5.7E-06	---	1.3E-05	1.9E-05
		Aluminum	---	---	---	---	---	---	7.9E-05	---	1.7E-05	9.2E-05
		Antimony	---	---	---	---	---	---	4.2E-05	---	6.2E-05	1.0E-04
		Cobalt	---	---	---	---	---	---	---	---	---	---
		Copper	---	---	---	---	---	---	2.0E-06	---	4.5E-07	2.5E-06
		Iron	---	---	---	---	---	---	3.8E-04	---	8.1E-05	4.4E-04
		Lead	---	---	---	---	---	---	---	---	---	---
		Manganese	---	---	---	---	---	---	---	---	---	---
		Vanadium	---	---	---	---	---	---	1.9E-04	---	7.3E-04	9.2E-04
		Zinc	---	---	---	---	---	---	NOAEL	---	4.4E-04	4.6E-04
(Total)	1.0E-08	---	4.5E-08	---	5.5E-08	---	---	0.0007	---	0.0015	0.0022	
Sediment	Site-Wide	Benzo(a)anthracene	1.5E-10	---	3.4E-10	4.9E-10	---	---	---	---	---	---
		Benzo(a)pyrene	2.5E-09	---	5.8E-09	8.3E-09	---	---	---	---	---	---
		Benzo(b)fluoranthene	2.1E-10	---	4.8E-10	6.9E-10	---	---	---	---	---	---
		Benzo(g,h,i)perylene	---	---	---	---	---	---	---	---	---	---
		Chrysene	2.5E-12	---	5.8E-12	8.3E-12	---	---	---	---	---	---
		Indeno(1,2,3-cd)pyrene	1.8E-10	---	4.1E-10	5.9E-10	---	---	---	---	---	---
		Antimony	---	---	---	---	---	---	---	---	---	---
		Arsenic	1.9E-08	---	1.0E-08	2.9E-08	---	---	---	3.8E-05	4.6E-05	8.4E-05
		Barium	---	---	---	---	---	---	---	1.3E-04	6.7E-05	2.0E-04
		Iron	---	---	---	---	---	---	---	2.7E-05	6.8E-05	9.5E-05
		Magnesium	---	---	---	---	---	---	---	1.1E-04	2.0E-05	1.3E-04
		Manganese	---	---	---	---	---	---	---	---	---	---
		Nickel	---	---	---	---	---	---	---	8.0E-05	2.4E-04	3.2E-04
Vanadium	---	---	---	---	---	---	---	1.5E-06	6.8E-06	8.3E-06		
Zinc	---	---	---	---	---	---	---	1.3E-05	9.8E-05	1.1E-04		
(Total)	2.2E-08	---	1.7E-08	---	3.9E-08	---	---	0.0004	---	0.0005	0.0009	
Total Risk Across [Surface Water]		Total Risk Across [Sediment]				Total Hazard Index Across All Media and All Exposure Routes						
		Total Risk Across [Sediment]				Total [Liver] HI =						
		Total Risk Across [Sediment]				Total [Blood Chemistry] HI =						
		Total Risk Across [Sediment]				Total [Kidney] HI =						
		Total Risk Across [Sediment]				Total [Developmental Neurotoxicity] HI =						
		Total Risk Across [Sediment]				Total [Skin, Vascular Effects] HI =						
		Total Risk Across [Sediment]				Total [Organ Weight] HI =						

N/A = Not Applicable
 GI = Gastrointestinal
 CNS = Central Nervous System
 NOAEL = No Observed Adverse Effect Level

Table 17
Summary of Receptor Risks and Hazards for COPCs
Reasonable Maximum Exposure
Former Carswell AFB, Texas

Scenario / Receptor Population / Receptor Age	Exposure Medium	Exposure Point	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			Exposure Routes Total
			Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ	Ingestion	
Current/Future Recreational User	Surface Water	Site-Wide	(Total)			(Total)			0.0045
			8.9E-09	3.9E-08	4.7E-08	2.8E-06	3.0E-05	2.6E-04	
Sediment	Sediment	Site-Wide	(Total)			(Total)			0.0030
			1.2E-10	2.9E-10	4.1E-10	2.8E-06	3.0E-05	2.6E-04	
Sediment	Sediment	Site-Wide	(Total)			(Total)			0.0014
			1.2E-10	2.9E-10	4.1E-10	2.8E-06	3.0E-05	2.6E-04	
Sediment	Sediment	Site-Wide	(Total)			(Total)			0.0011
			1.2E-10	2.9E-10	4.1E-10	2.8E-06	3.0E-05	2.6E-04	
Sediment	Sediment	Site-Wide	(Total)			(Total)			0.0008
			1.2E-10	2.9E-10	4.1E-10	2.8E-06	3.0E-05	2.6E-04	
Sediment	Sediment	Site-Wide	(Total)			(Total)			0.0003
			1.2E-10	2.9E-10	4.1E-10	2.8E-06	3.0E-05	2.6E-04	
Sediment	Sediment	Site-Wide	(Total)			(Total)			0.0002
			1.2E-10	2.9E-10	4.1E-10	2.8E-06	3.0E-05	2.6E-04	
Sediment	Sediment	Site-Wide	(Total)			(Total)			0.0001
			1.2E-10	2.9E-10	4.1E-10	2.8E-06	3.0E-05	2.6E-04	
Sediment	Sediment	Site-Wide	(Total)			(Total)			0.00005
			1.2E-10	2.9E-10	4.1E-10	2.8E-06	3.0E-05	2.6E-04	
Sediment	Sediment	Site-Wide	(Total)			(Total)			0.00002
			1.2E-10	2.9E-10	4.1E-10	2.8E-06	3.0E-05	2.6E-04	

Total [Liver] HI =	0.00030
Total [Blood Chemistry] HI =	0.0005
Total [Kidney] HI =	0.0002
Total [Developmental Neurotoxicity] HI =	0.0002
Total [GI Ingestion] HI =	0.00005
Total [GI Inhalation] HI =	0.0011
Total [Skin, Vascular Effects] HI =	0.0004
Total [Organ Weight] HI =	0.00002

Total Hazard Index Across All Media and All Exposure Routes

Total Risk Across (Surface Water)

Total Risk Across (Sediment)

Total Risk Across All Media and All Exposure Routes

N/A = Not Applicable
 GI = Gastrointestinal
 CNS = Central Nervous System
 NOAEL = No Observed Adverse Effect Level

Table 18
Species-Specific Exposure Parameters^a
Former Carswell AFB, TX

Parameter	Mouse	Quail
Body Weight (g) - BW	22	190
Fraction of Soil in the Diet (%) - FS ^b	2	9.3
Food Intake Rate (kg/day) - IR	0.0044	0.017
Fraction of food from foraging area (%) - FR	100	100
Water Intake Rate (g/g-day) - NIR	0.19	0.10

a Wildlife Exposure Factors Handbook (EPA, 1993)

b Total fraction of soil in diet is partitioned into soil and sediment fractions. The total ingested fraction is assumed to be 50% for soil and 50% for sediment.

Table 19
 Comparison of Contaminants of Potential Concern with Surface Water Benchmarks, Surface Water
 Former Carswell AFB, Texas
 Page 1 of 2

Chemical	Frequency of Detection	Range of values, mg/L Detected	Range of values, mg/L Not Detected	Statistical Distribution	95% UCL mg/L	Background mg/L	Screening Criteria mg/L	Exclusion Rationale	Exceeds Criteria?
Inorganics									
Aluminum	8/11(73)	1.1E-01 - 3.2E+01	5.0E-01 - 5.0E-01	U	3.2E+01	2.7E-01	NA	E	N
Antimony	3/11(27)	3.8E-03 - 7.1E-03	5.0E-03 - 5.0E-03	NP	7.1E-03	3.0E-03	1.6E-01	C	N
Arsenic	1/11(9)	3.3E-02	5.0E-03 - 5.0E-03	NP	3.3E-02	NA	1.9E-01	C	N
Barium	11/11(100)	7.5E-02 - 5.7E-01	NA	U	5.7E-01	1.5E-01	NA	E	N
Beryllium	1/11(9)	1.6E-03	3.0E-03 - 3.0E-03	NA	NA	NA	5.3E-04	-	Y
Calcium	11/11(100)	6.5E+01 - 4.1E+02	NA	L	1.9E+02	1.3E+02	NA	D	N
Cobalt	1/11(9)	2.0E-02	7.0E-02 - 7.0E-02	NP	2.0E-02	NA	NA	E	N
Copper	3/11(27)	3.3E-03 - 3.2E-02	6.0E-02 - 6.0E-02	NP	3.2E-02	1.0E-02	6.5E-03	-	Y
Iron	11/11(100)	5.7E-02 - 4.6E+01	NA	U	4.6E+01	9.2E-01	NA	E	N
Lead	9/11(82)	2.0E-03 - 6.3E-02	5.0E-03 - 5.0E-03	U	6.3E-02	NA	1.3E-03	-	Y
Magnesium	11/11(100)	5.4E+00 - 2.1E+01	NA	U	2.1E+01	9.4E+00	NA	E	N
Manganese	11/11(100)	5.0E-03 - 3.9E+00	NA	U	3.9E+00	4.2E-01	NA	E	N
Mercury	4/11(36)	5.7E-05 - 2.1E-04	1.0E-03 - 1.0E-03	NP	2.1E-04	1.0E-04	1.2E-05	-	Y
Nickel	1/11(9)	2.8E-02	1.5E-01 - 1.5E-01	NP	2.8E-02	1.8E-02	8.7E-02	C	N
Potassium	11/11(100)	9.7E-01 - 1.2E+01	NA	U	1.2E+01	6.4E+00	NA	D	N
Selenium	1/11(9)	6.8E-03	5.0E-03 - 5.0E-03	NP	6.8E-03	2.5E-03	5.0E-03	-	Y
Silver	1/11(9)	7.8E-04	2.0E-04 - 2.0E-04	NP	7.8E-04	3.0E-04	1.2E-05	-	Y
Vanadium	4/11(36)	2.4E-03 - 5.8E-02	8.0E-02 - 8.0E-02	NP	5.8E-02	1.6E-02	NA	E	N
Zinc	3/11(27)	3.0E-02 - 4.2E-01	2.0E-02 - 2.0E-02	NP	4.2E-01	1.2E-02	5.8E-02	-	Y
Volatile Organic Compounds									
1,1-Dichloroethane	1/11(9)	4.7E-03	4.0E-04 - 8.0E-03	NP	4.7E-03	NA	NA	E	N
1,1-Dichloroethene	1/11(9)	3.1E-04	1.2E-03 - 2.4E-02	NP	3.1E-04	NA	3.0E-01	C	N
Cis-1,2-Dichloroethylene	11/11(100)	3.2E-04 - 6.4E-02	NA	L	2.6E-01	NA	NA	E	N
Tetrachloroethylene	1/11(9)	1.1E-03	1.4E-03 - 2.8E-02	NP	1.1E-03	NA	8.4E-02	C	N
Trichloroethylene	10/11(91)	3.5E-04 - 2.0E-01	1.0E-03	L	6.7E+00	NA	NA	E	N
Vinyl Chloride	1/11(9)	7.3E-03	1.1E-03 - 2.2E-02	NP	7.3E-03	NA	NA	E	N

Table 19
Comparison of Contaminants of Potential Concern with Surface Water Benchmarks, Surface Water
Former Carswell AFB, Texas
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Chemical	Frequency of Detection	Range of values, mg/L		95% UCL mg/L ^b	Background mg/L ^c	Screening Criteria mg/L ^d	Exclusion Rationale ^e	Exceeds Criteria? ^f
		Detected	Nondetected					
Semi-Volatile Organic Compounds								
Bis(2-ethylhexyl) phthalate	3/11(27)	4.4E-03 - 1.2E-02	1.0E-02 - 1.2E-02	1.2E-02	NA	3.0E-04	--	Y
Diethyl phthalate	1/1(9)	1.5E-03	1.0E-02 - 1.2E-02	1.5E-03	NA	5.2E-01	C	N
Di-n-butyl phthalate	2/11(18)	2.1E-03 - 5.8E-03	1.0E-02 - 1.1E-02	5.8E-03	NA	9.4E-03	C	N

^a Statistical Distribution: N = Normal distribution; L = Lognormal distribution, U = Undetermined distribution; NP = Nonparametric distribution for data sets with greater than 50% NA = distribution not determined if sample size is less than 5.
^b 95% Upper Confidence Limit calculated for the indicated distribution. NA = sample size is less than 5 and distribution is not calculated.
^c Background concentrations for inorganic constituents.
^d Toxicity/concentration screen based on EPA Region IV Freshwater Surface Water Screening Values (EPA, 2000b).
^e Rationale for exclusion of chemical as a contaminant of potential concern (COPC):
 A = within background concentration.
 B = detection frequency less than 5%.
 C = maximum detection is less than screening criteria
 D = essential nutrient.
 E = no criteria
^f N = Chemical does not exceed criteria; Y = Chemical exceeds criteria.

Table 20
 Comparison of Contaminants of Potential Concern with Ecological Sediment Screening Criteria, Sediment
 Former Carswell AFB, Texas
 Page 1 of 2

Chemical	Frequency of Detection	Range of values, mg/kg	Detected	NonDetected	Statistical Distribution	95% UCL mg/kg	Background mg/kg	Screening Criteria mg/kg	Exclusion Rationale	Exceeds Criteria?
INORGANICS										
ALUMINUM	12/12(100)	8.9E+02 - 1.3E+04		NA	L	7.5E+03	2.0E+04	NA	A	N
ANTIMONY	10/12(83)	1.7E-01 - 6.5E+00		6.1E-01 - 1.1E+00	U	6.5E+00	3.3E-01	1.2E+01	C	N
ARSENIC	12/12(100)	5.5E+00 - 3.7E+01		NA	L	1.6E+01	8.0E+00	7.2E+00	--	Y
BARIUM	12/12(100)	1.2E+01 - 7.9E+02		NA	L	1.0E+03	1.4E+02	NA	E	N
BERYLLIUM	12/12(100)	1.6E-01 - 5.5E-01		NA	L	4.4E-01	7.2E-01	NA	A	N
CADMIUM	9/12(75)	7.8E-02 - 5.9E-01		5.1E+00 - 5.5E+00	L	3.0E+00	1.8E+00	1.0E+00	A,C	N
CALCIUM	12/12(100)	2.6E+04 - 3.6E+05		NA	N	2.4E+05	3.6E+05	NA	A,D	N
COBALT	12/12(100)	1.4E+00 - 2.2E+01		NA	L	1.3E+01	4.8E+01	NA	A	N
COPPER	12/12(100)	1.9E+00 - 8.6E+00		NA	L	6.0E+00	1.7E+01	1.9E+01	A,C	N
IRON	12/12(100)	5.7E+02 - 2.5E+04		NA	L	1.4E+04	1.0E+04	NA	E	N
LEAD	12/12(100)	5.2E+00 - 7.8E+01		NA	L	4.2E+01	8.8E+01	3.0E+01	A	N
MAGNESIUM	12/12(100)	1.0E+03 - 3.1E+03		NA	N	2.5E+03	2.6E+03	NA	E	N
MANGANESE	12/12(100)	1.7E+02 - 2.1E+03		NA	L	1.6E+03	3.5E+02	NA	E	N
MERCURY	12/12(100)	1.2E-02 - 1.2E-01		NA	U	1.2E-01	3.6E-02	1.3E-01	C	N
NICKEL	12/12(100)	2.7E+00 - 2.1E+01		NA	L	1.3E+01	1.6E+00	1.6E+01	--	Y
POTASSIUM	12/12(100)	1.3E+02 - 1.5E+03		NA	L	1.1E+03	5.0E+03	NA	A,D	N
Sodium	12/12(100)	8.8E+01 - 2.7E+02		NA	N	2.1E+02	6.1E+00	NA	D	N
Vanadium	12/12(100)	1.4E+01 - 1.1E+02		NA	L	4.9E+01	3.2E+01	NA	E	N
Zinc	8/12(67)	1.3E+01 - 1.6E+02		2.2E+01 - 4.2E+01	U	1.6E+02	8.1E+01	1.2E+02	--	Y
Voatile Organic Compounds										
Cis-1,2-Dichloroethylene	5/12(42)	2.7E-03 - 1.2E-02		6.7E-03 - 1.2E-02	NP	1.2E-02	NA	NA	E	N
Toluene	1/11(9)	1.4E-02		5.9E-03 - 9.7E-03	NP	1.4E-02	NA	NA	E	N
Trichloroethylene	7/12(58)	2.1E-03 - 5.6E-02		1.1E-02 - 1.9E-02	L	2.4E-02	NA	NA	E	N

Table 20
 Comparison of Contaminants of Potential Concern with Ecological Sediment Screening Criteria, Sediment
 Former Carswell AFB, Texas
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Chemical	Frequency of Detection	Range of values, mg/kg		Statistical Distribution	95% UCL, mg/kg ^b	Background, mg/kg ^c	Screening Criteria, mg/kg ^d	Exclusion Rationale ^e	Exceeds Criteria?
		Detected	Non-detected						
Semi-Volatile Organic Compounds									
Benzo(a)anthracene	1/12(8)	2.5E-01	7.2E-01 - 9.2E-01	NP	2.5E-01	NA	3.3E-01	C	N
Benzo(a)pyrene	1/12(8)	4.2E-01	7.2E-01 - 9.2E-01	NP	4.2E-01	NA	3.3E-01	--	Y
Benzo(b)fluoranthene	1/12(8)	3.5E-01	7.2E-01 - 9.2E-01	NP	3.5E-01	NA	3.3E-01	--	Y
Benzo(g,h,i)perylene	1/12(8)	3.6E-01	7.2E-01 - 9.2E-01	NP	3.6E-01	NA	3.3E-01	--	Y
Chrysene	1/12(8)	4.2E-01	7.2E-01 - 9.2E-01	NP	4.2E-01	NA	3.3E-01	--	Y
Fluoranthene	1/12(8)	8.7E-01	7.2E-01 - 9.2E-01	NP	8.7E-01	NA	3.3E-01	--	Y
Indeno(1,2,3-cd)pyrene	1/12(8)	3.0E-01	7.2E-01 - 9.2E-01	NP	3.0E-01	NA	3.3E-01	C	N
Pyrene	1/12(8)	6.2E-01	7.2E-01 - 9.2E-01	NP	6.2E-01	NA	3.3E-01	--	Y

^a Statistical Distribution: N = Normal distribution; L = Lognormal distribution, U = Undetermined distribution; NP = Nonparametric distribution for data sets with greater than 50% n.c.
 NA = distribution not determined if sample size is less than 5.
^b 95% Upper Confidence Limit calculated for the indicated distribution. NA = sample size is less than 5 and distribution is not calculated.
^c Background concentrations for inorganic constituents.
^d Toxicity/concentration screen based on EPA Region IV Sediment Screening Values for Hazardous Waste Sites (EPA, 2000)
^e Rationale for exclusion of chemical as a contaminant of potential concern (COPC):
 A = within background concentration
 B = detection frequency less than 5%
 C = maximum detection is less than screening criteria.
 D = essential nutrient.
 E = no criteria
 Y = Chemical does not exceed criteria; Y = Chemical exceeds criteria.

Table 21
 Ecological Hazard Quotients (EQs) for the Mouse Exposed to Surface Water and Sediment
 Former Carswell AFB, TX

Chemical	Sed. Con. (mg/kg)	Sediment Dose (mg/kg/day)	SW Conc. (mg/L)	SW Dose (mg/kg/day)	Toxicity Value (mg/kg/day)	Total Dose (mg/kg/day)	EQ (unitless)
Inorganics							
Aluminum	0.0E+00	0.0E+00	3.2E+01	6.1E+00	2.1E+00	6.1E+00	2.8E+00
Antimony	6.5E+00	1.3E-02	7.1E-03	1.3E-03	1.4E-01	1.4E-02	1.0E-01
Arsenic	1.6E+01	3.2E-02	3.3E-02	6.3E-03	1.4E-01	3.8E-02	2.7E-01
Barium	7.9E+01	1.6E-01	5.7E-01	1.1E-01	1.4E+01	2.7E-01	2.0E-02
Beryllium	0.0E+00	0.0E+00	1.6E-03	3.0E-04	1.7E+00	3.0E-04	1.8E-04
Cobalt	1.3E+01	2.6E-02	2.0E-02	3.8E-03	NA	3.0E-02	NA
Copper	0.0E+00	0.0E+00	3.2E-02	6.1E-03	4.1E+01	6.1E-03	1.5E-04
Iron	1.4E+04	2.8E+01	4.6E+01	8.7E+00	NA	3.7E+01	NA
Lead	0.0E+00	0.0E+00	6.3E-02	1.2E-02	2.0E+01	1.2E-02	6.0E-04
Magnesium	2.5E+03	5.0E+00	2.1E+01	4.0E+00	NA	9.0E+00	NA
Manganese	1.6E+03	3.2E+00	3.9E+00	7.4E-01	2.2E+02	3.9E+00	1.8E-02
Mercury	1.2E+01	2.4E-04	2.1E-04	4.0E-05	1.6E-02	2.8E-04	1.7E-02
Nickel	1.3E+01	2.6E-02	2.8E-02	5.3E-03	1.0E+02	3.1E-02	3.1E-04
Selenium	0.0E+00	0.0E+00	6.8E-03	1.3E-03	8.3E-02	1.3E-03	1.6E-02
Silver	0.0E+00	0.0E+00	7.8E-04	1.5E-04	1.4E-03	1.5E-04	1.1E-01
Vanadium	4.9E+01	9.8E-02	5.8E-02	1.1E-02	4.7E-01	1.1E-01	2.3E-01
Zinc	1.6E+02	3.2E-01	4.2E-01	8.0E-02	4.0E+02	4.0E-01	1.0E-03
Volatile Organics							
1,1-Dichloroethane	0.0E+00	0.0E+00	4.7E-03	8.9E-04	5.0E+01	8.9E-04	1.8E-05
1,1-Dichloroethene	0.0E+00	0.0E+00	3.1E-04	5.9E-05	3.0E+01	5.9E-05	2.0E-06
Cis-1,2-Dichloroethene	1.2E-02	2.4E-05	6.4E-02	1.2E-02	5.0E+01	1.2E-02	2.4E-04
Tetrachloroethylene	0.0E+00	0.0E+00	1.1E-03	2.1E-04	NA	2.1E-04	NA
Toluene	1.4E-02	2.8E-05	0.0E+00	0.0E+00	2.9E+01	2.8E-05	9.7E-07
Trichloroethene	2.4E-02	4.8E-05	2.0E-01	3.8E-02	7.0E-01	3.8E-02	5.4E-02
Vinyl Chloride	0.0E+00	0.0E+00	7.3E-03	1.4E-03	4.2E-01	1.4E-03	3.3E-03
Semivolatile Organics							
Benzo(a)anthracene	2.5E-01	5.0E-04	0.0E+00	0.0E+00	1.1E+00	5.0E-04	4.5E-04
Benzo(a)pyrene	4.2E-01	8.4E-04	0.0E+00	0.0E+00	1.1E+00	8.4E-04	7.6E-04
Benzo(b)fluoranthene	3.5E-01	7.0E-04	0.0E+00	0.0E+00	1.1E+00	7.0E-04	6.4E-04
Benzo(g,h,i)perylene	3.6E-01	7.2E-04	0.0E+00	0.0E+00	1.1E+00	7.2E-04	6.5E-04
Bis(2-ethylhexyl)phthalate	0.0E+00	0.0E+00	1.2E-02	2.3E-03	2.0E+01	2.3E-03	1.1E-04
Chrysene	4.2E-01	8.4E-04	0.0E+00	0.0E+00	1.1E+00	8.4E-04	7.6E-04
Di-n-butylphthalate	0.0E+00	0.0E+00	5.8E-03	1.1E-03	6.1E+02	1.1E-03	1.8E-06
Diethyl phthalate	0.0E+00	0.0E+00	1.5E-03	2.9E-04	5.5E+02	2.9E-04	5.2E-07
Fluoranthene	8.7E-01	1.7E-03	0.0E+00	0.0E+00	1.3E+02	1.7E-03	1.4E-05
Indeno(1,23-cd)pyrene	3.0E-01	6.0E-04	0.0E+00	0.0E+00	1.1E+00	6.0E-04	5.5E-04
Pyrene	6.2E-01	1.2E-03	0.0E+00	0.0E+00	7.5E+01	1.2E-03	1.7E-05

Table 22
 Ecological Hazard Quotients (EQs) for the Quail Exposed to Surface Water and Sediment
 Former Carswell AFB, TX

Chemical	Sed. Conc. (mg/kg)	Sediment Dose (mg/kg day)	SW Conc. (mg/L)	SW Dose (mg/kg day)	Toxicity Value (mg/kg day)	Total Dose (mg/kg day)	EQ (unitless)
Inorganics							
Aluminum	0.0E+00	0.0E+00	3.2E+01	3.2E+00	1.0E+02	3.2E+00	3.1E-02
Antimony	6.5E+00	2.7E-02	7.1E-03	7.1E-04	NA	2.8E-02	NA
Arsenic	1.6E+01	6.7E-02	3.3E-02	3.3E-03	8.8E+00	7.0E-02	8.0E-03
Barium	7.9E+01	3.3E-01	5.7E-01	5.7E-02	1.8E+01	3.9E-01	2.2E-02
Beryllium	0.0E+00	0.0E+00	1.6E-03	1.6E-04	NA	1.6E-04	NA
Cobalt	1.3E+01	5.4E-02	2.0E-02	2.0E-03	NA	5.6E-02	NA
Copper	0.0E+00	0.0E+00	3.2E-02	3.2E-03	4.7E+01	3.2E-03	6.8E-05
Iron	1.4E+04	5.8E+01	4.6E+01	4.6E+00	NA	6.3E+01	NA
Lead	0.0E+00	0.0E+00	6.3E-02	6.3E-03	3.4E+00	6.3E-03	1.9E-03
Magnesium	2.5E+03	1.0E+01	2.1E+01	2.1E+00	NA	1.3E+01	NA
Manganese	1.6E+03	6.7E+00	3.9E+00	3.9E-01	9.8E+02	7.0E+00	7.2E-03
Mercury	1.2E-01	5.0E-04	2.1E-04	2.1E-05	1.1E-02	5.2E-04	4.7E-02
Nickel	1.3E+01	5.4E-02	2.8E-02	2.8E-03	1.2E+02	5.7E-02	4.7E-04
Selenium	0.0E+00	0.0E+00	6.8E-03	6.8E-04	8.5E-01	6.8E-04	8.0E-04
Silver	0.0E+00	0.0E+00	7.8E-04	7.8E-05	NA	7.8E-05	NA
Vanadium	4.9E+01	2.0E-01	5.8E-02	5.8E-03	2.0E+01	2.1E-01	1.0E-02
Zinc	1.6E+02	6.7E-01	4.2E-01	4.2E-02	5.1E+00	7.1E-01	1.4E-01
Volatile Organics							
1,1-Dichloroethane	0.0E+00	0.0E+00	4.7E-03	4.7E-04	1.7E+01	4.7E-04	2.7E-05
1,1-Dichloroethene	0.0E+00	0.0E+00	3.1E-04	3.1E-05	NA	3.1E-05	NA
Cis-1,2-Dichloroethene	1.2E-02	5.0E-05	6.4E-02	6.4E-03	NA	6.4E-03	NA
Tetrachloroethylene	0.0E+00	0.0E+00	1.1E-03	1.1E-04	NA	1.1E-04	NA
Toluene	1.4E-02	5.8E-05	0.0E+00	0.0E+00	NA	5.8E-05	NA
Trichloroethene	2.4E-02	1.0E-04	2.0E-01	2.0E-02	NA	2.0E-02	NA
Vinyl Chloride	0.0E+00	0.0E+00	7.3E-03	7.3E-04	NA	7.3E-04	NA
Semivolatile Organics							
Benzo(a)anthracene	2.5E-01	1.0E-03	0.0E+00	0.0E+00	NA	1.0E-03	NA
Benzo(a)pyrene	4.2E-01	1.7E-03	0.0E+00	0.0E+00	NA	1.7E-03	NA
Benzo(b)fluoranthene	3.5E-01	1.5E-03	0.0E+00	0.0E+00	NA	1.5E-03	NA
Benzo(g,h,i)perylene	3.6E-01	1.5E-03	0.0E+00	0.0E+00	NA	1.5E-03	NA
Bis(2-ethylhexyl)phthalate	0.0E+00	0.0E+00	1.2E-02	1.2E-03	1.1E+00	1.2E-03	1.1E-03
Chrysene	4.2E-01	1.7E-03	0.0E+00	0.0E+00	NA	1.7E-03	NA
Di-n-butylphthalate	0.0E+00	0.0E+00	0.0E+00	0.0E+00	1.0E-01	0.0E+00	-
Diethyl phthalate	0.0E+00	0.0E+00	5.8E-03	5.8E-04	NA	5.8E-04	NA
Fluoranthene	8.7E-01	3.6E-03	0.0E+00	0.0E+00	NA	3.6E-03	NA
Indeno(123-cd)pyrene	3.0E-01	1.2E-03	0.0E+00	0.0E+00	NA	1.2E-03	NA
Pyrene	6.2E-01	2.6E-03	0.0E+00	0.0E+00	NA	2.6E-03	NA

TAB

FIGURES

HydroGeologic, Inc.—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

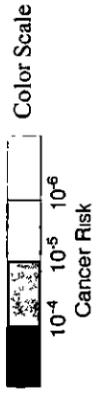
Figure 4 1,1-Dichloroethene Carcinogenic Risk Isopleth For Residents



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ◆ Monitoring Wells
- 5 40E-05 Risk
- 9 Highest Concentration (µg/L) of Constituent in 1999

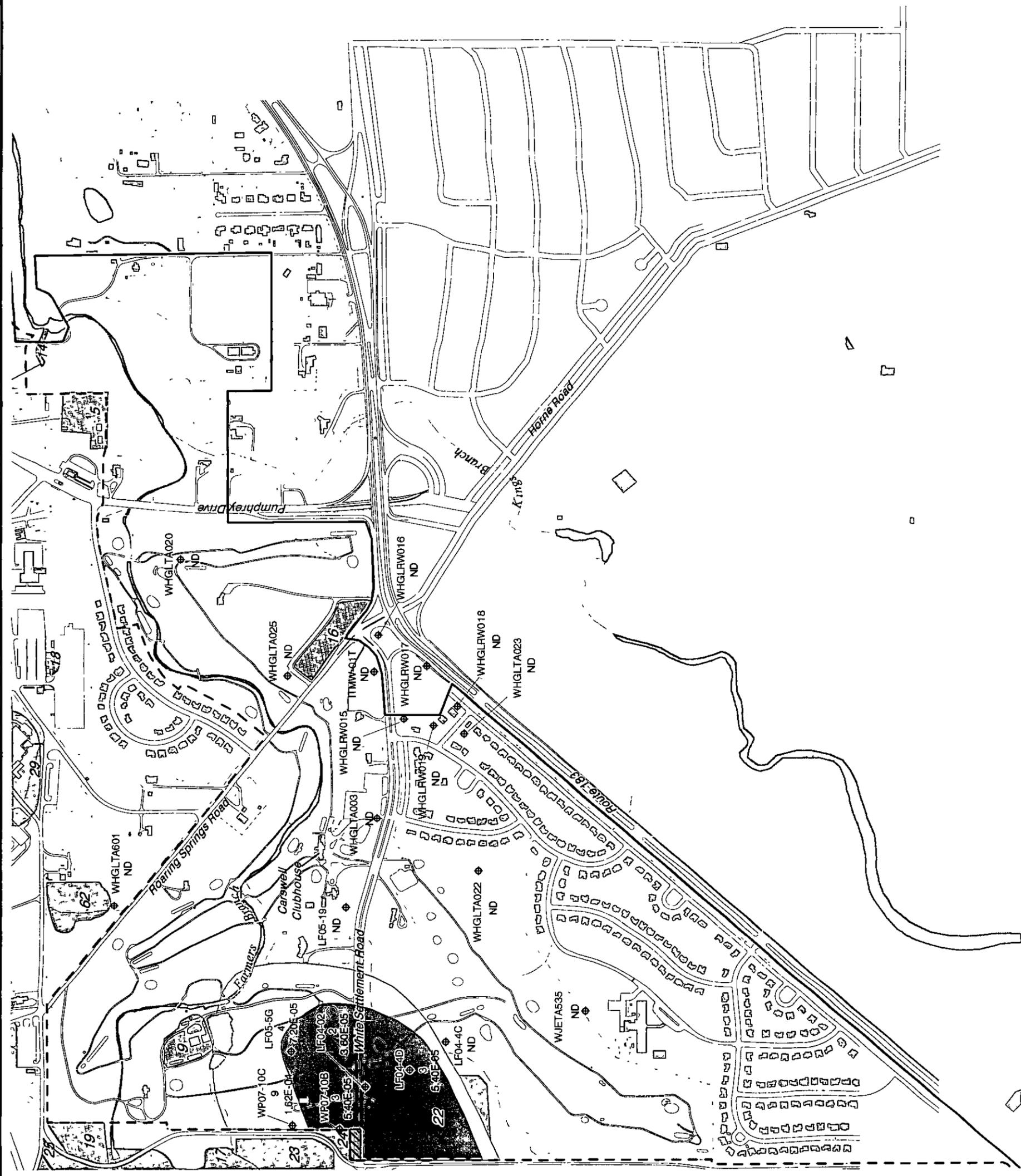
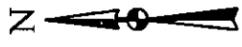


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No. 2
- 62 Landfill No 6



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Revised 12/29/00 jb
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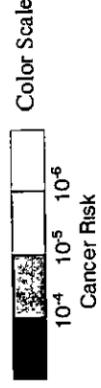
HydroGeologic, Inc.—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

Figure 5 1,4-Dichlorobenzene Carcinogenic Risk Isoleth For Residents



Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 1 60E-06 Risk
- 12 Highest Concentration (µg/L) of Constituent in 1999

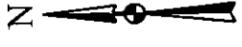


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

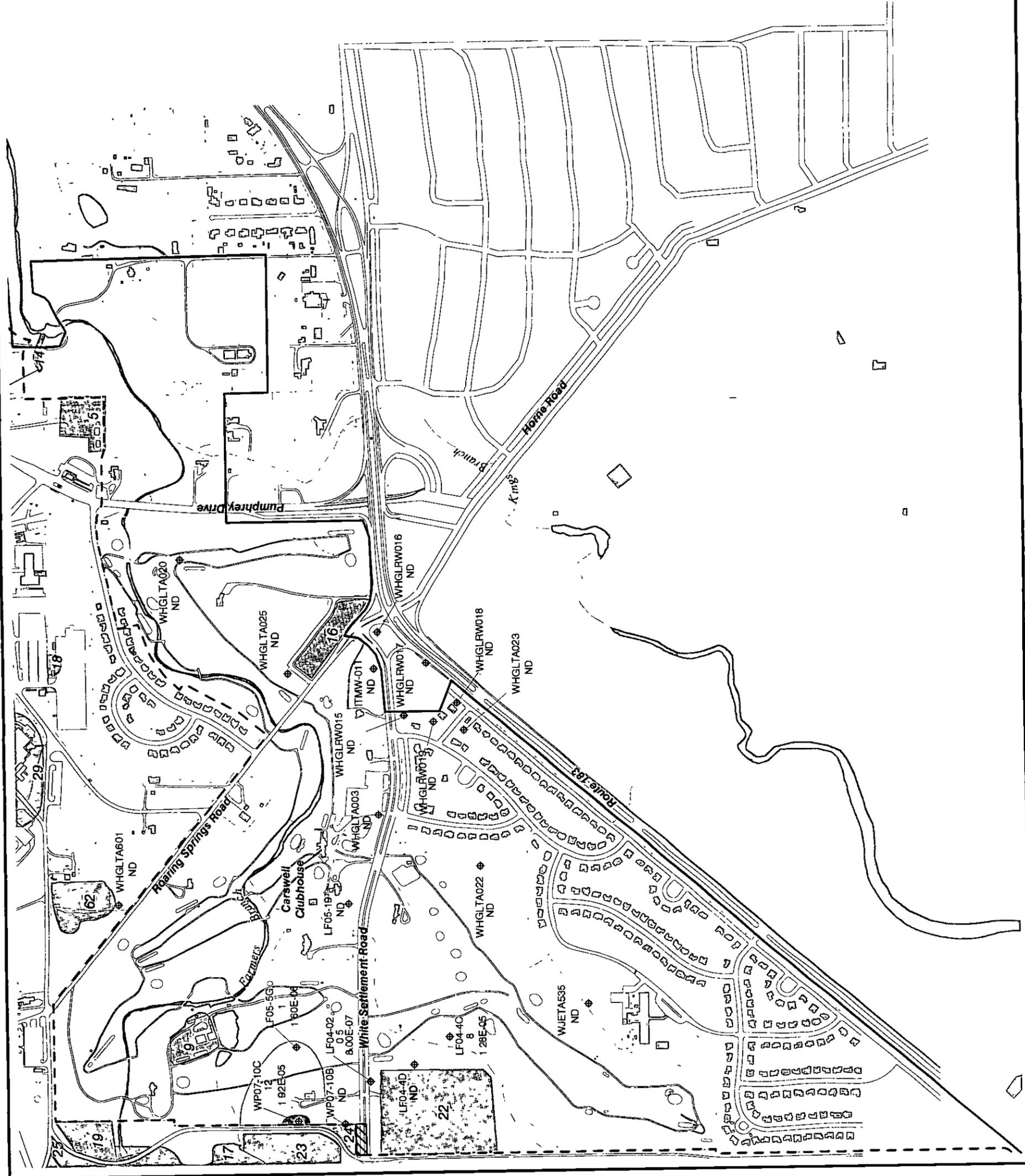
Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No. 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



SCALE IN FEET

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HydroGeoLogic, Inc —Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

Figure 6 Chloroform Carcinogenic Risk Isoleth For Residents



Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 3.01E-06 Risk
- 0.5 Highest Concentration (µg/L) of Constituent in 1999

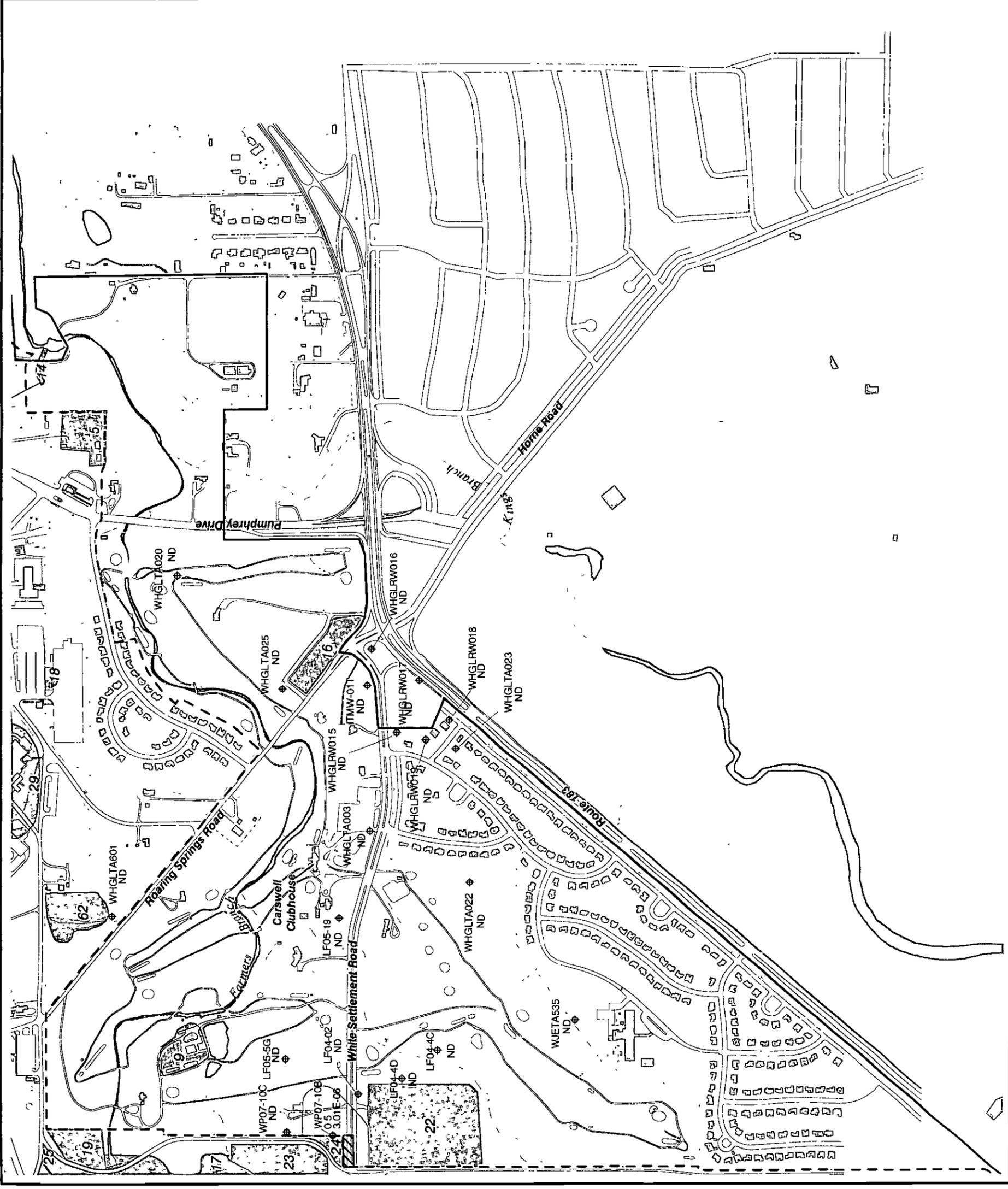
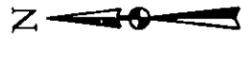


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No. 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No. 8
- 29 Landfill No 2
- 62 Landfill No 6



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Former Carswell AFB, Fort Worth, Texas

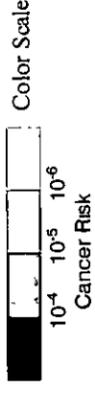
Figure 7 Arsenic Carcinogenic Risk Isopleth For Residents



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- ND Highest Concentration (µg/L) of Constituent in 1999



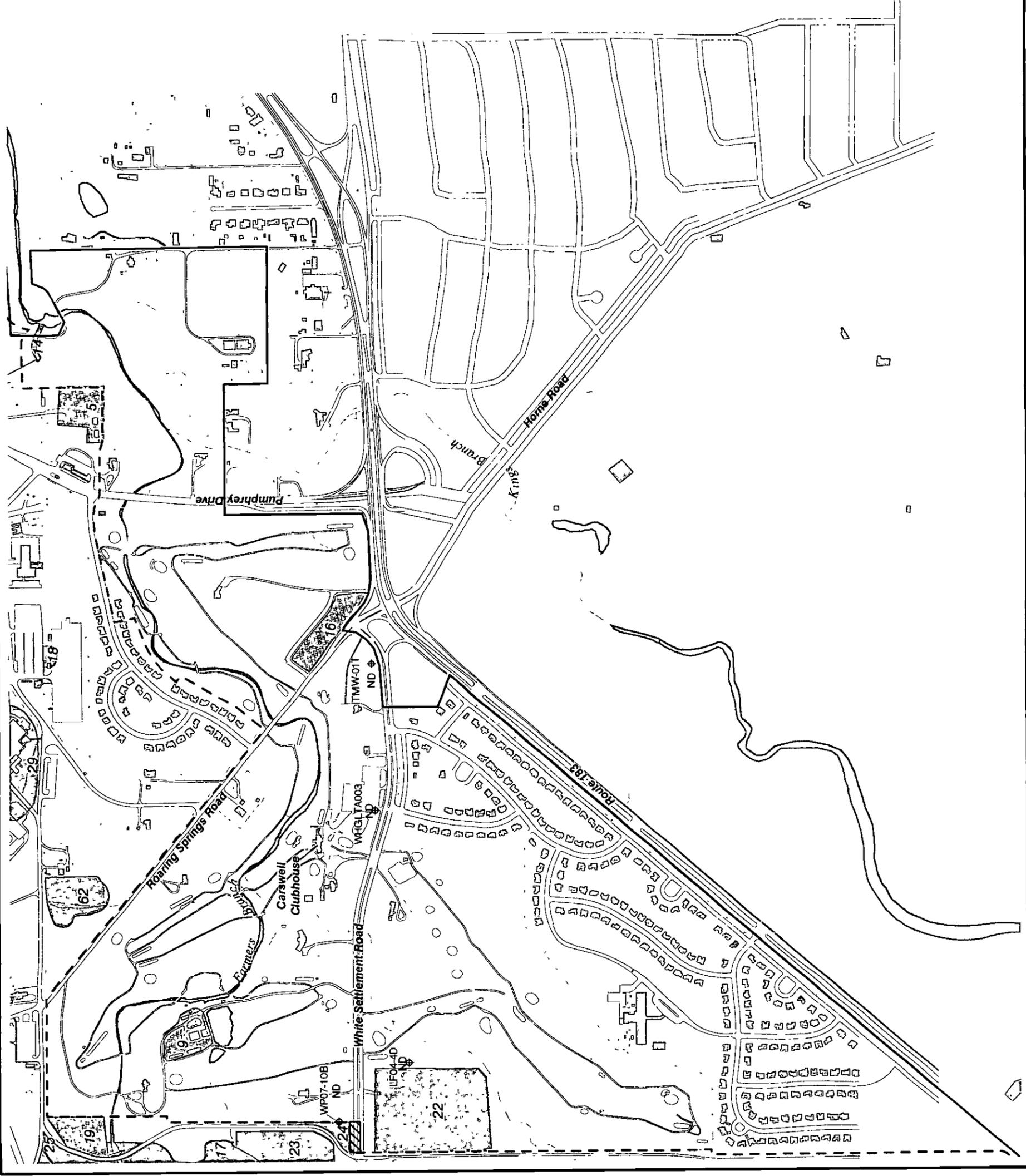
Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area



Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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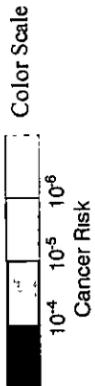
HydroGeoLogic, Inc.—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

Figure 8 Bis (2-ethylhexyl) phthalate Carcinogenic Risk Isopleth For Residents



Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ◆ Monitoring Wells
- 157E-05 Risk
- 29 Highest Concentration (µg/L) of Constituent in 1999

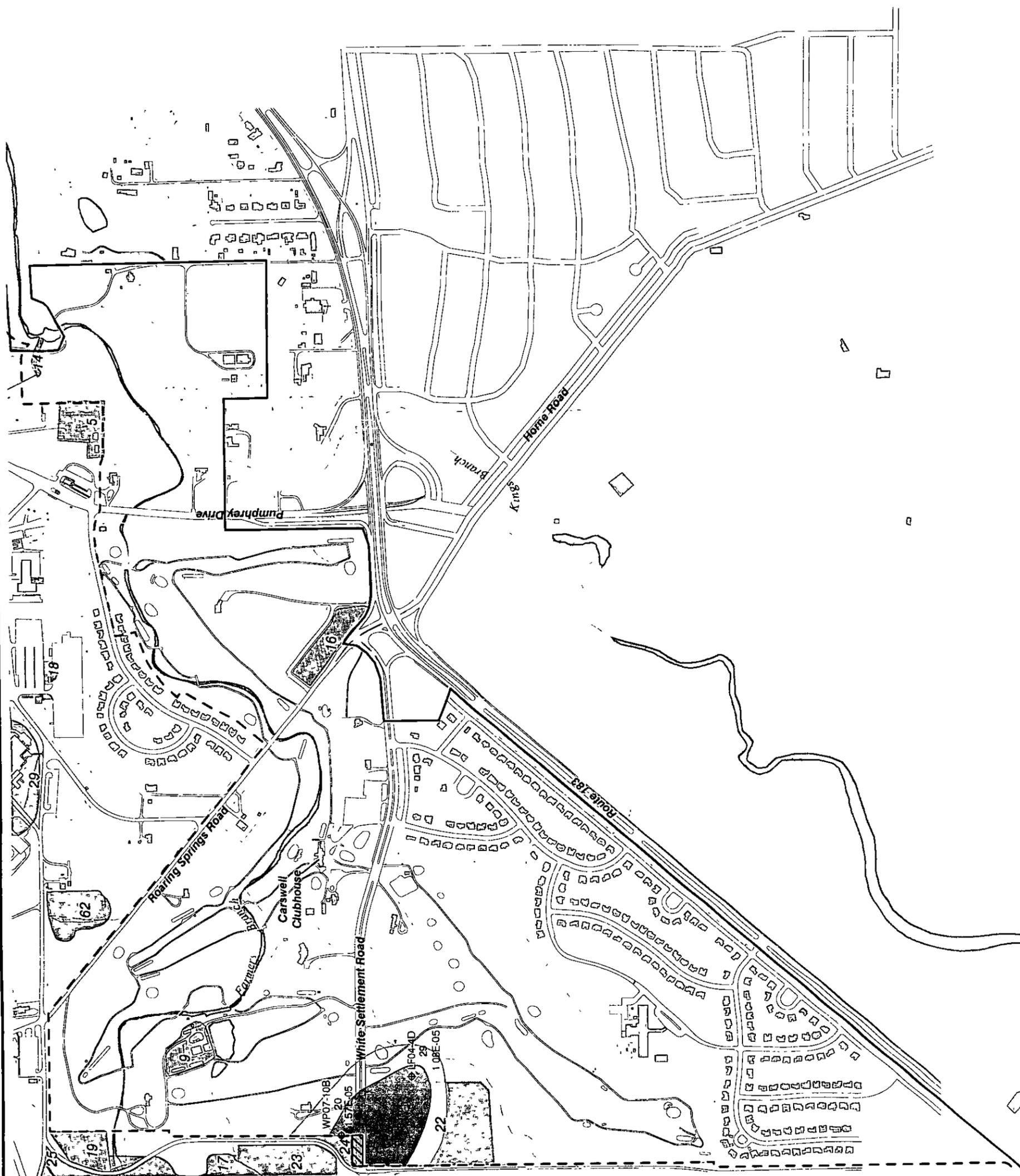


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No. 7
- 22 Landfill No. 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No. 8
- 29 Landfill No. 2
- 62 Landfill No. 6



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HydroGeologic, Inc.—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

Figure 14 Chloroform Carcinogenic Risk Isopleth For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ◆ Monitoring Wells
- 5 95E-08
- 0.5

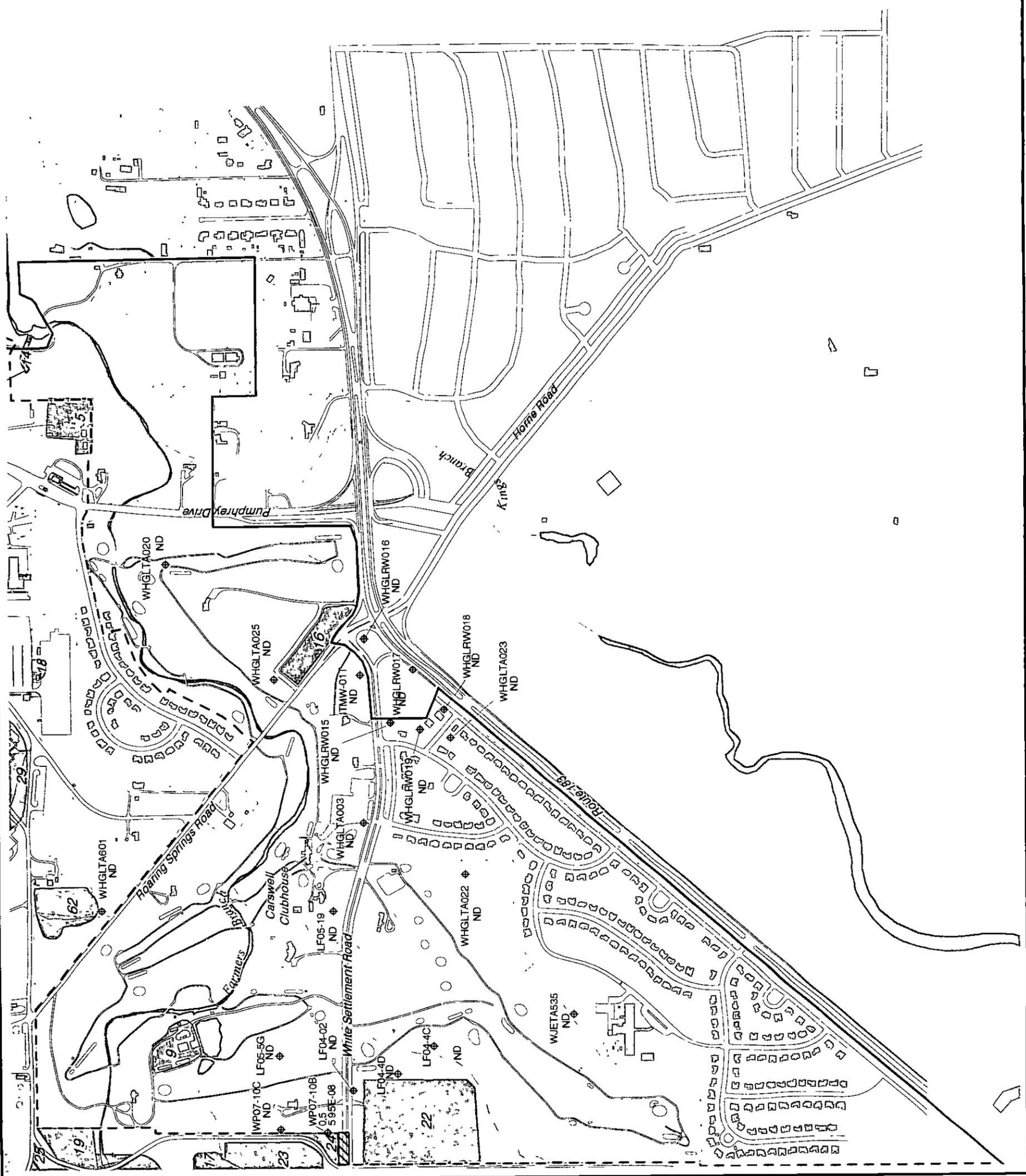


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No. 7
- 22 Landfill No. 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No. 8
- 29 Landfill No. 2
- 62 Landfill No. 6



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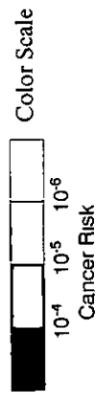
Figure 15
Arsenic
Carcinogenic Risk Isopleth
For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- ND Highest Concentration (µg/L) of Constituent in 1999

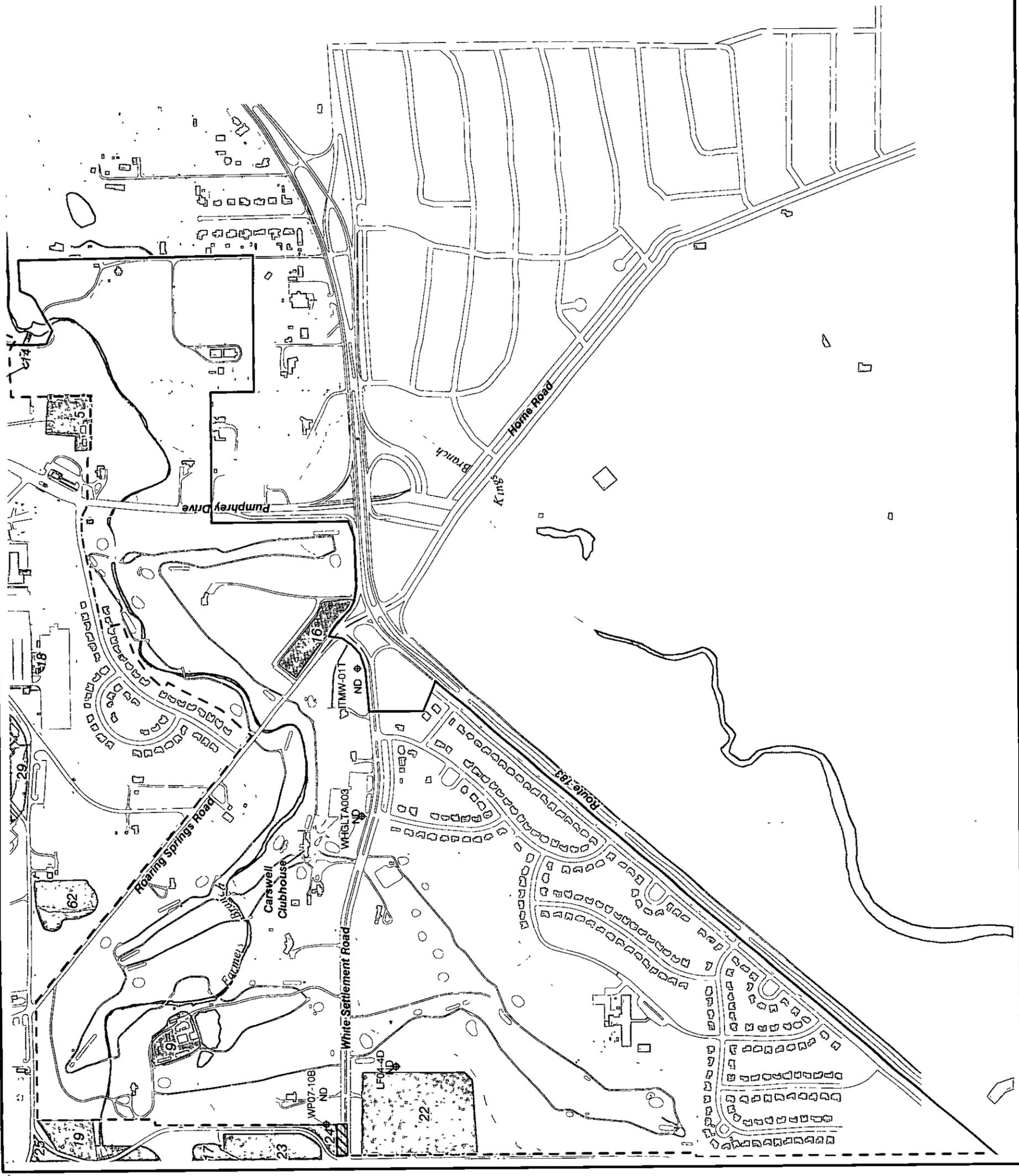


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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Former Carswell AFB, Fort Worth, Texas

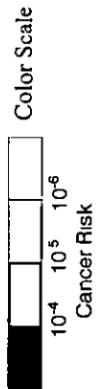
Figure 16
Bis (2-ethylhexyl) phthalate
Carcinogenic Risk Isopleth
For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- - - - - NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 1 94E-05 Risk
- 29 Highest Concentration (µg/L) of Constituent in 1999

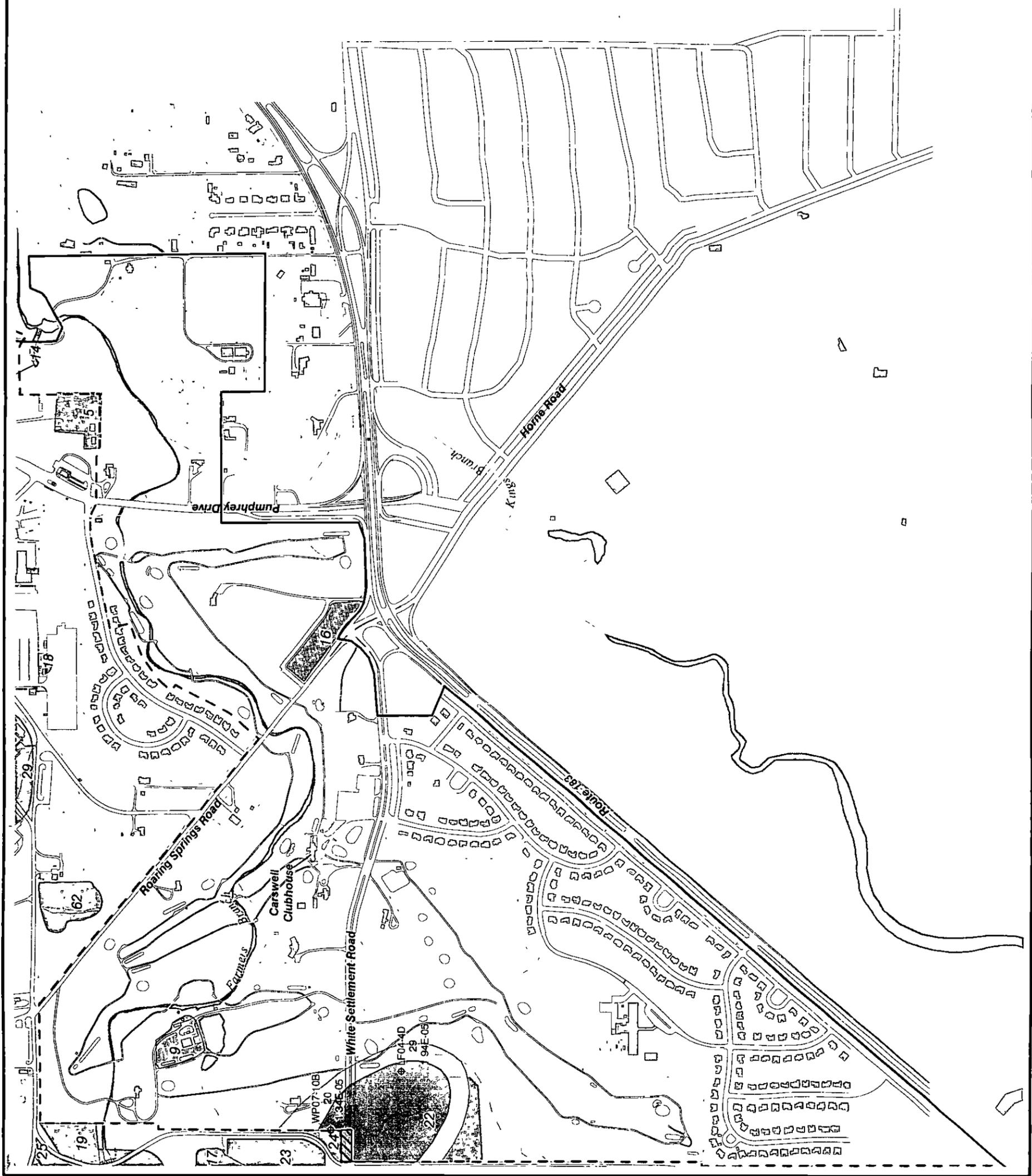
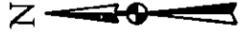


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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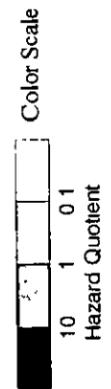
HydroGeologic, Inc — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

Figure 19 Cis-1,2-Dichloroethene Non-Carcinogenic Risk Isopleth For Residents



Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 20 Hazard Quotient
- 510 Highest Concentration (µg/L) of Constituent in 1999

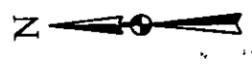


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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Former Carswell AFB, Fort Worth, Texas

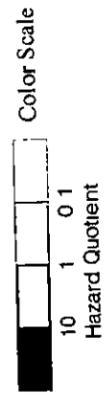
Figure 22 1,4-Dichlorobenzene Non-Carcinogenic Risk Isopleth For Residents



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 0.04 Hazard Quotient
- 12 Highest Concentration (µg/L) of Constituent in 1999

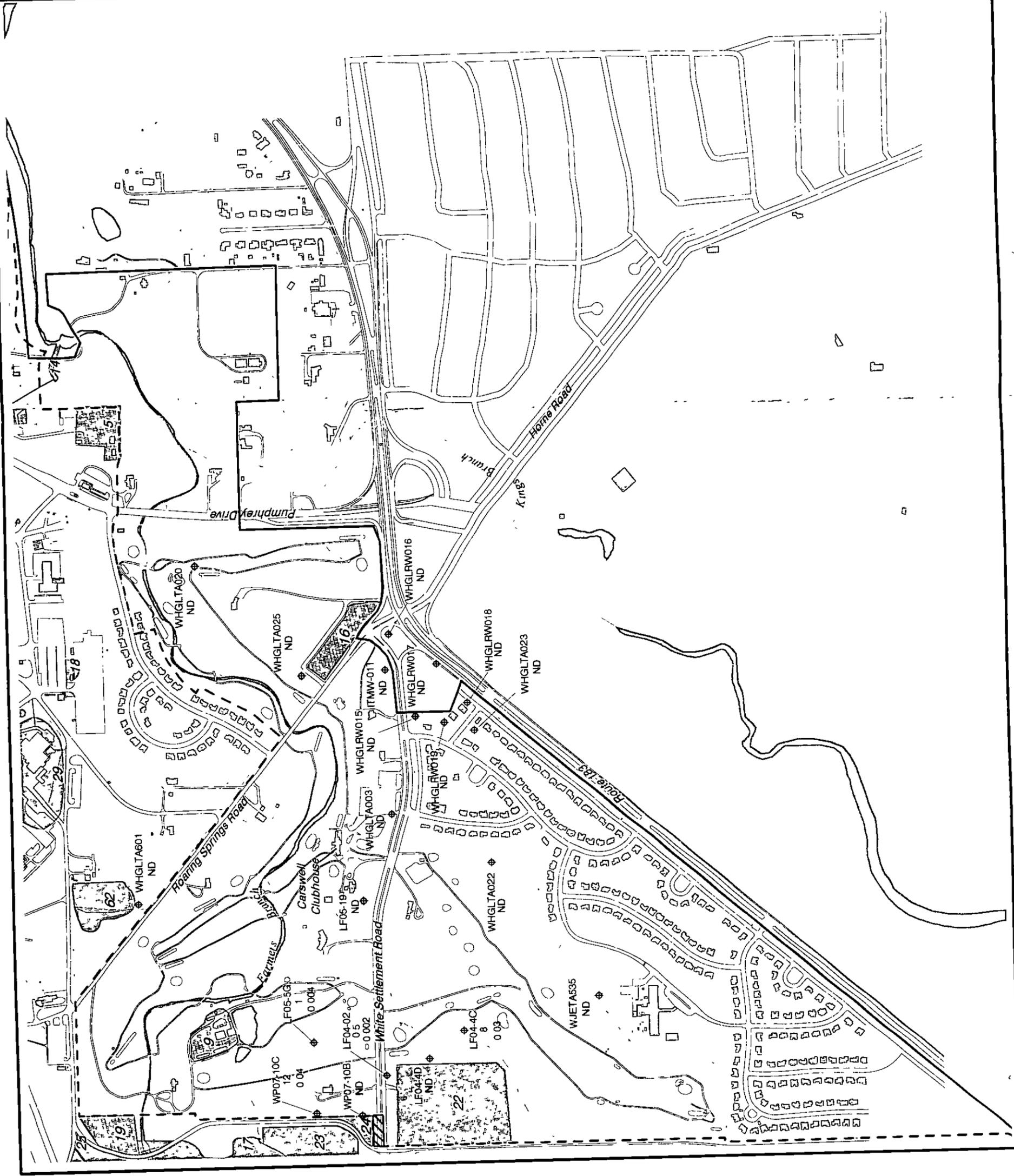


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No. 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No. 8
- 29 Landfill No. 2
- 62 Landfill No. 6



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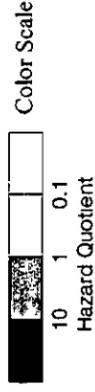
Figure 23 Chloroform Non-Carcinogenic Risk Isoleth For Residents



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ◆ Monitoring Wells
- 3 Hazard Quotient
- 0.5 Highest Concentration (µg/L) of Constituent in 1999



Area of Concern

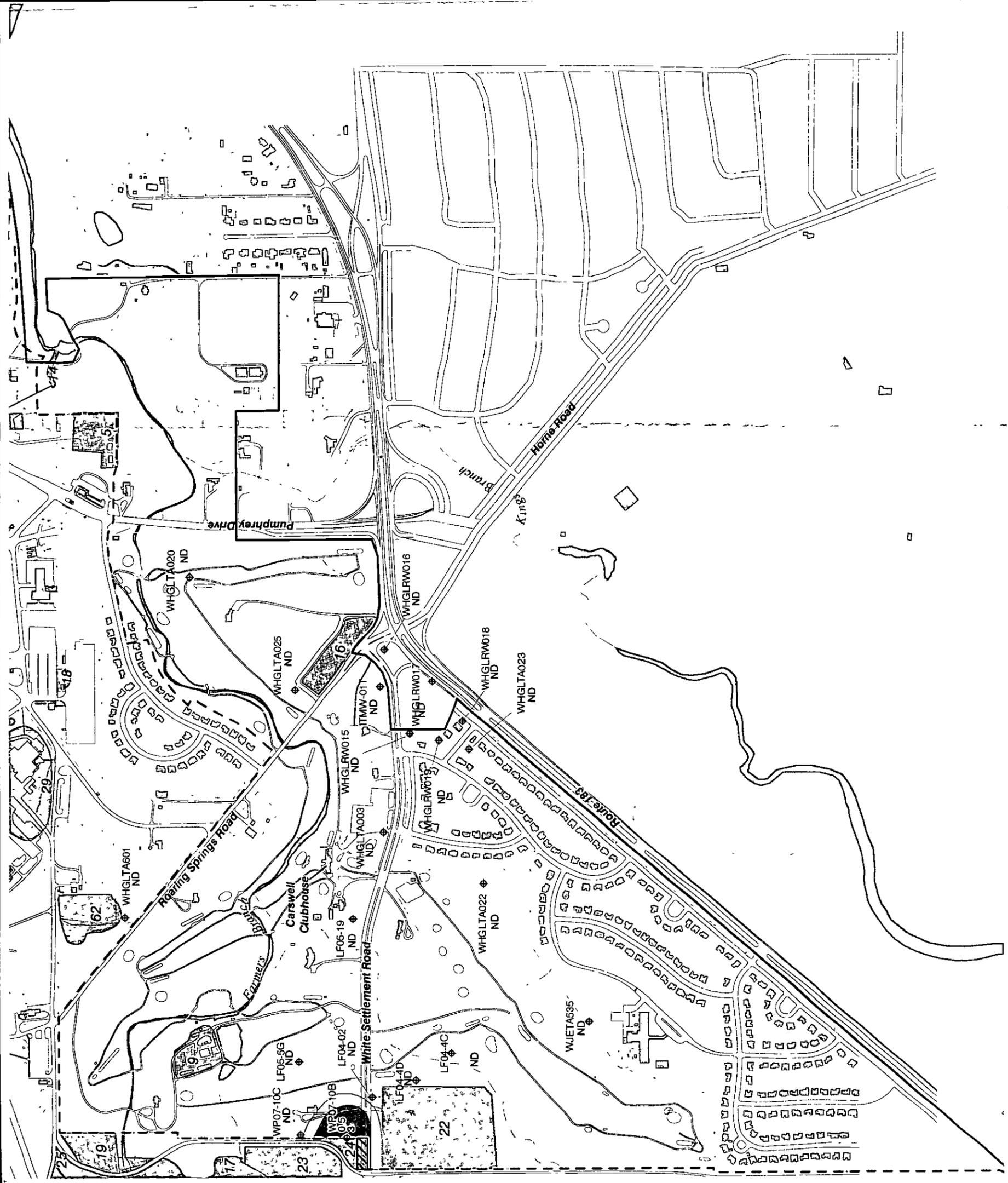
- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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Project AFC001-36CB
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Revised 12/29/00 cf
Map Source HGL ArcView GIS Database 2000



HydroGeologic, Inc. — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

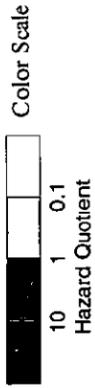
Figure 24 Arsenic Non-Carcinogenic Risk Isopleth For Residents



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 6.45 E-07 Hazard Quotient
- ND Highest Concentration (µg/L) of Constituent in 1999



Area of Concern

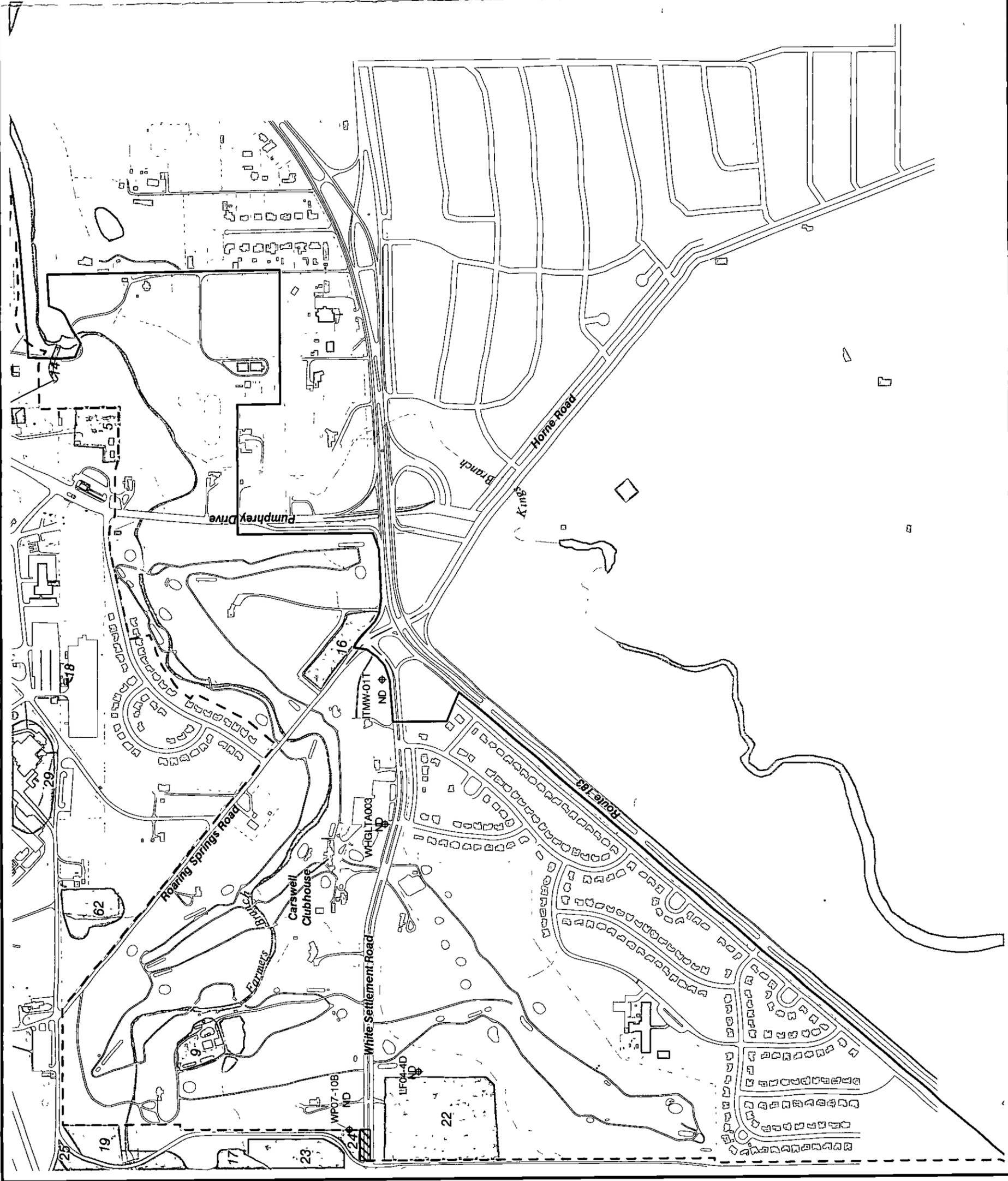
- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No. 7
- 22 Landfill No. 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No. 8
- 29 Landfill No. 2
- 62 Landfill No. 6



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Revised: 12/28/00 jb
Map Source HGL ArcView GIS Database 2000



HydroGeologic, Inc. — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

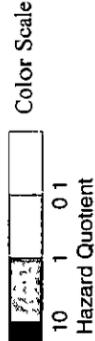
Figure 25 Chromium Non-Carcinogenic Risk Isoleth For Residents



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 0.26 Hazard Quotient
- 10 Highest Concentration (µg/L) of Constituent in 1999



Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

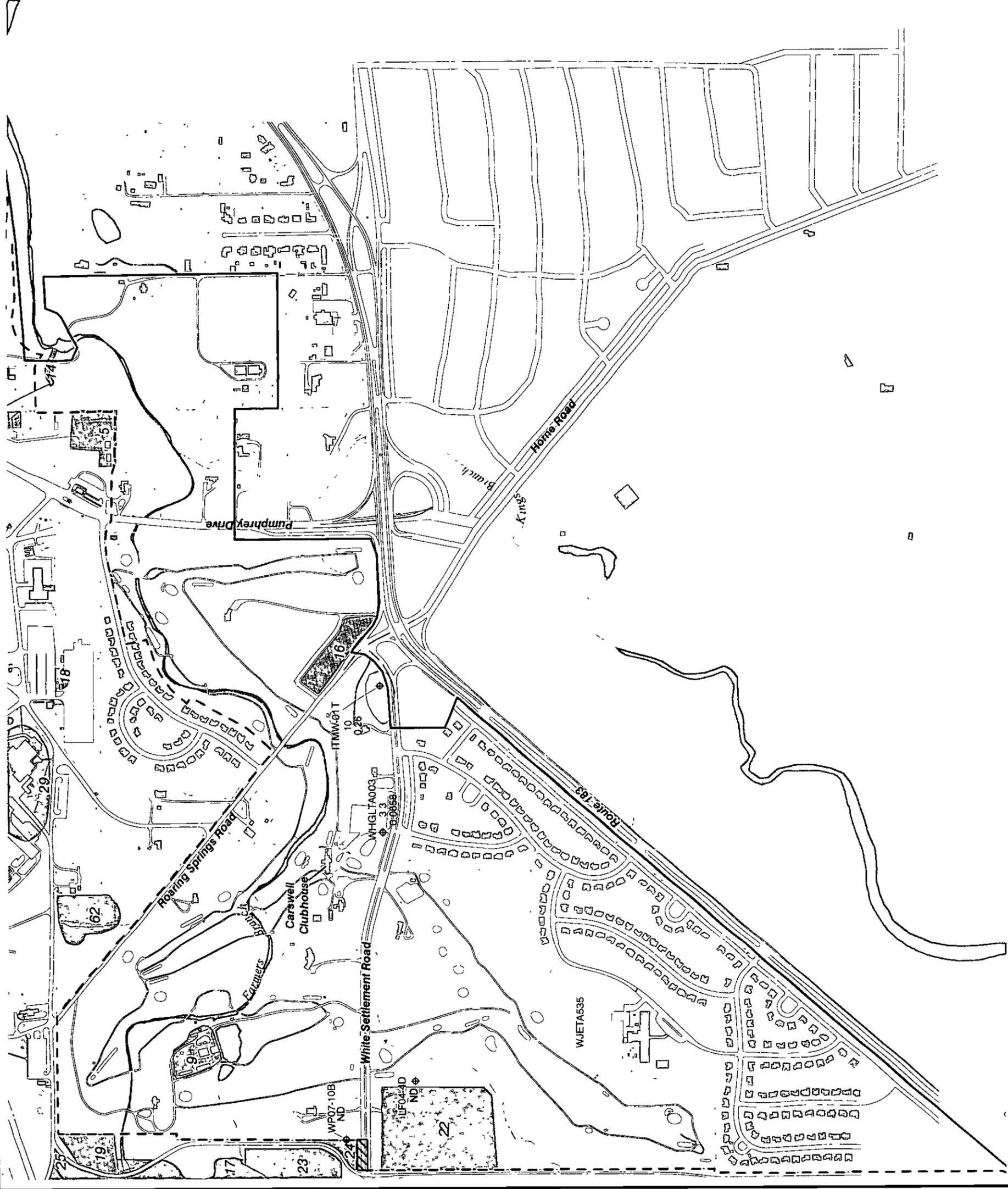


Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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Revised 12/28/00 JB
Map Source HGL ArcView GIS Database 2000



HydroGeologic, Inc — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

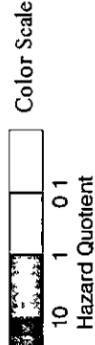
Figure 26 Bis (2-ethylhexyl) phthalate Non-Carcinogenic Risk Isoleth For Residents



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 0.4 Hazard Quotient
- 29 Highest Concentration (µg/L) of Constituent in 1999

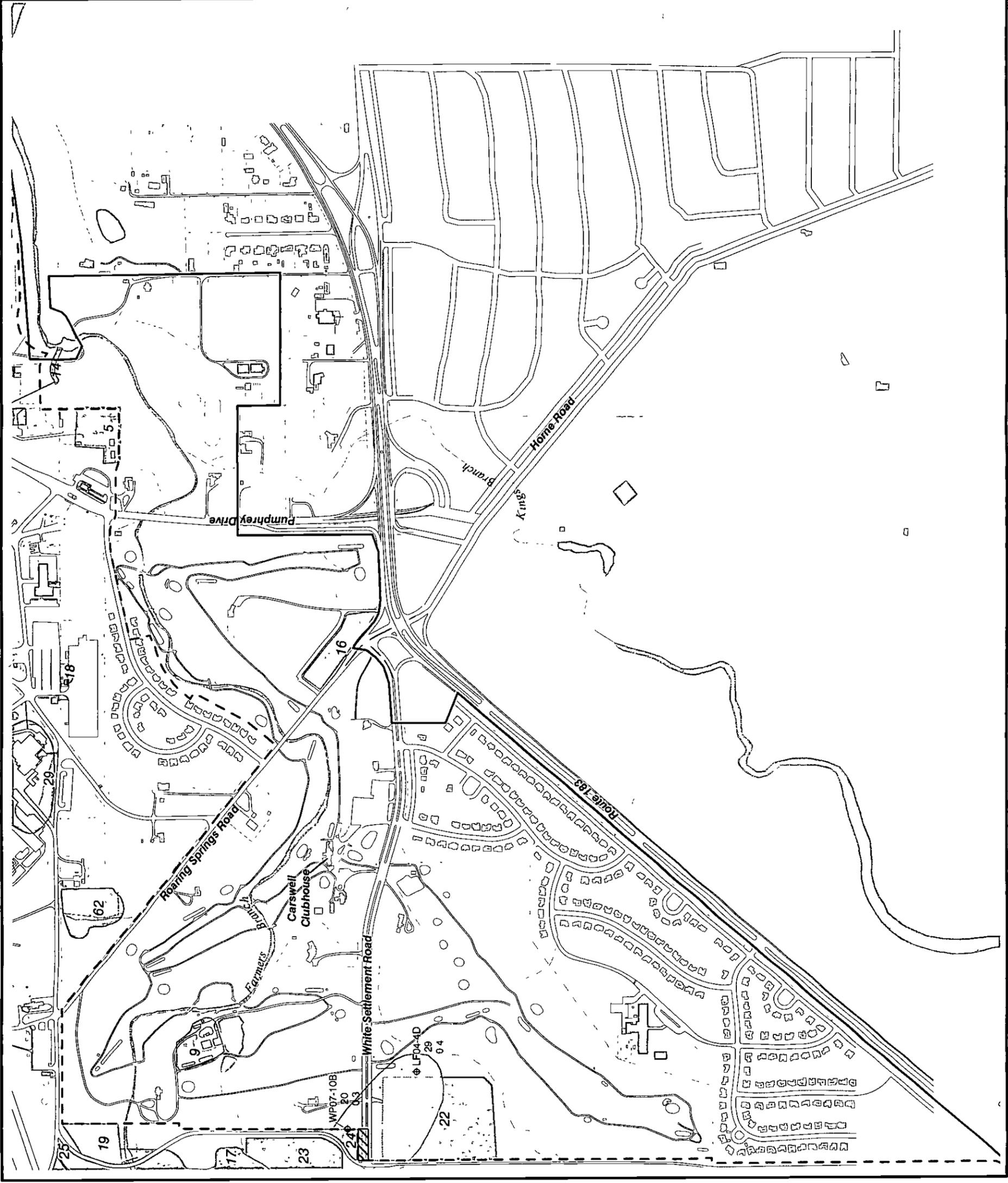


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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Revised 12/28/00 jb
Map Source HGL ArcView GIS Database
2000



HydroGeologic, Inc — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

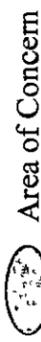
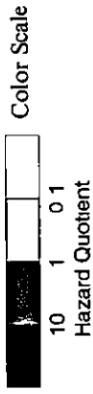
Figure 28 Trichloroethene Non-Carcinogenic Risk Isopleth For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 63 Hazard Quotient
- 3300 Highest Concentration (µg/L) of Constituent in 1999



Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

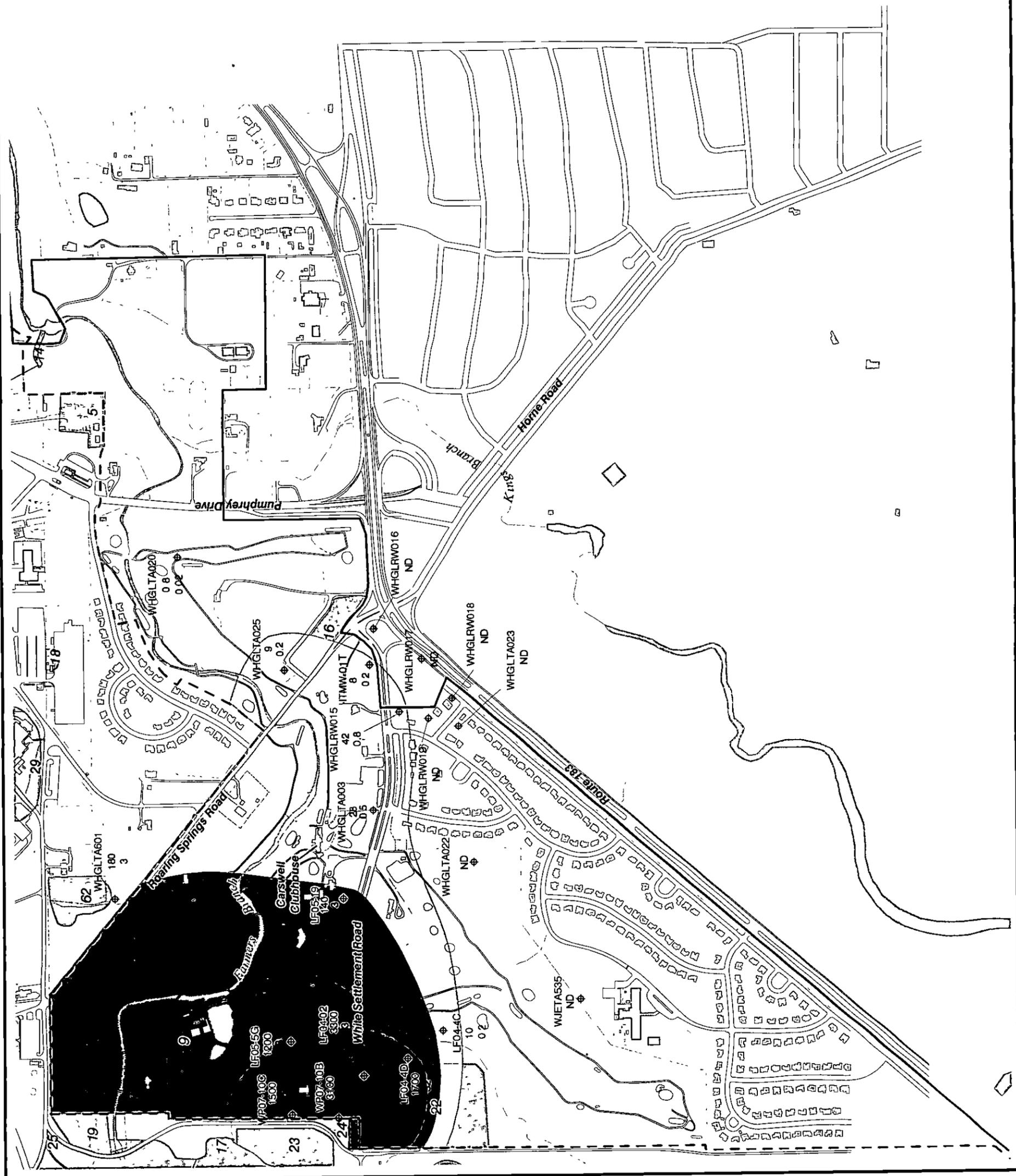


Solid Waste Management Unit

- 17 Landfill No. 7
- 22 Landfill No. 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No. 8
- 29 Landfill No. 2
- 62 Landfill No. 6



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Project AFC001-36CB
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Revised 12/29/00 cf
Map Source HGL Arc View GIS Database 2000



HydroGeoLogic, Inc.—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

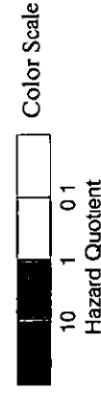
Figure 32 1,4-Dichlorobenzene Non-Carcinogenic Risk Isopleth For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- - - - - NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 0.006 Hazard Quotient
- 12 Highest Concentration (µg/L) of Constituent in 1999



Area of Concern

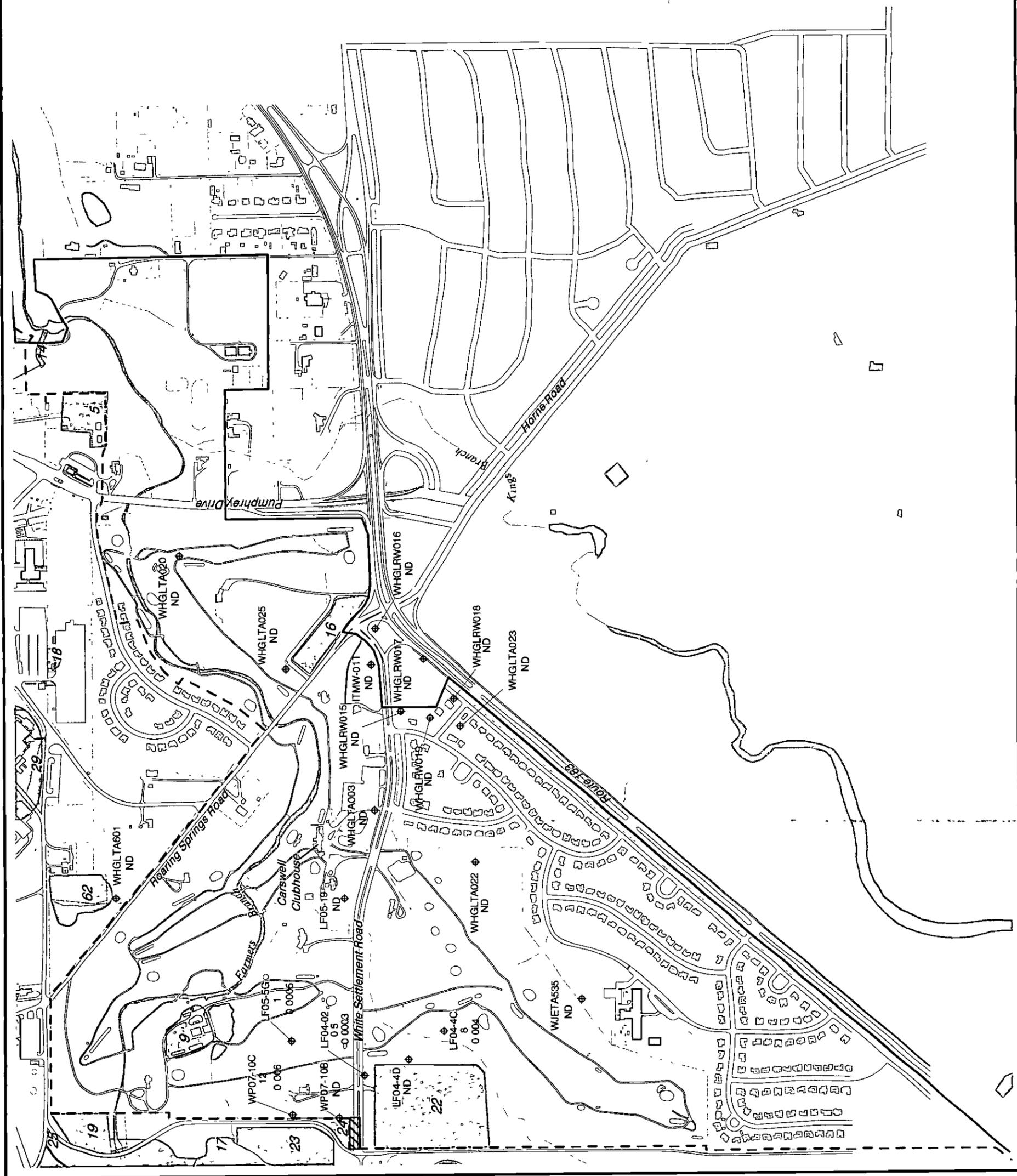
- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No. 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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HydroGeologic, Inc — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

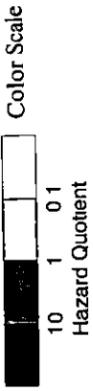
Figure 33 Chloroform Non-Carcinogenic Risk Isoopleth For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- - - - - NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ◆ Monitoring Wells
- 0.8 Hazard Quotient
- 0.5 Highest Concentration (µg/L) of Constituent in 1999

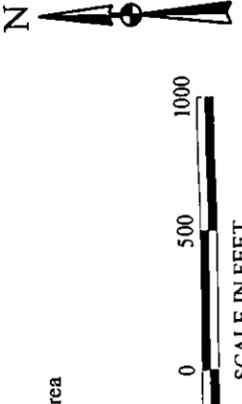


Area of Concern

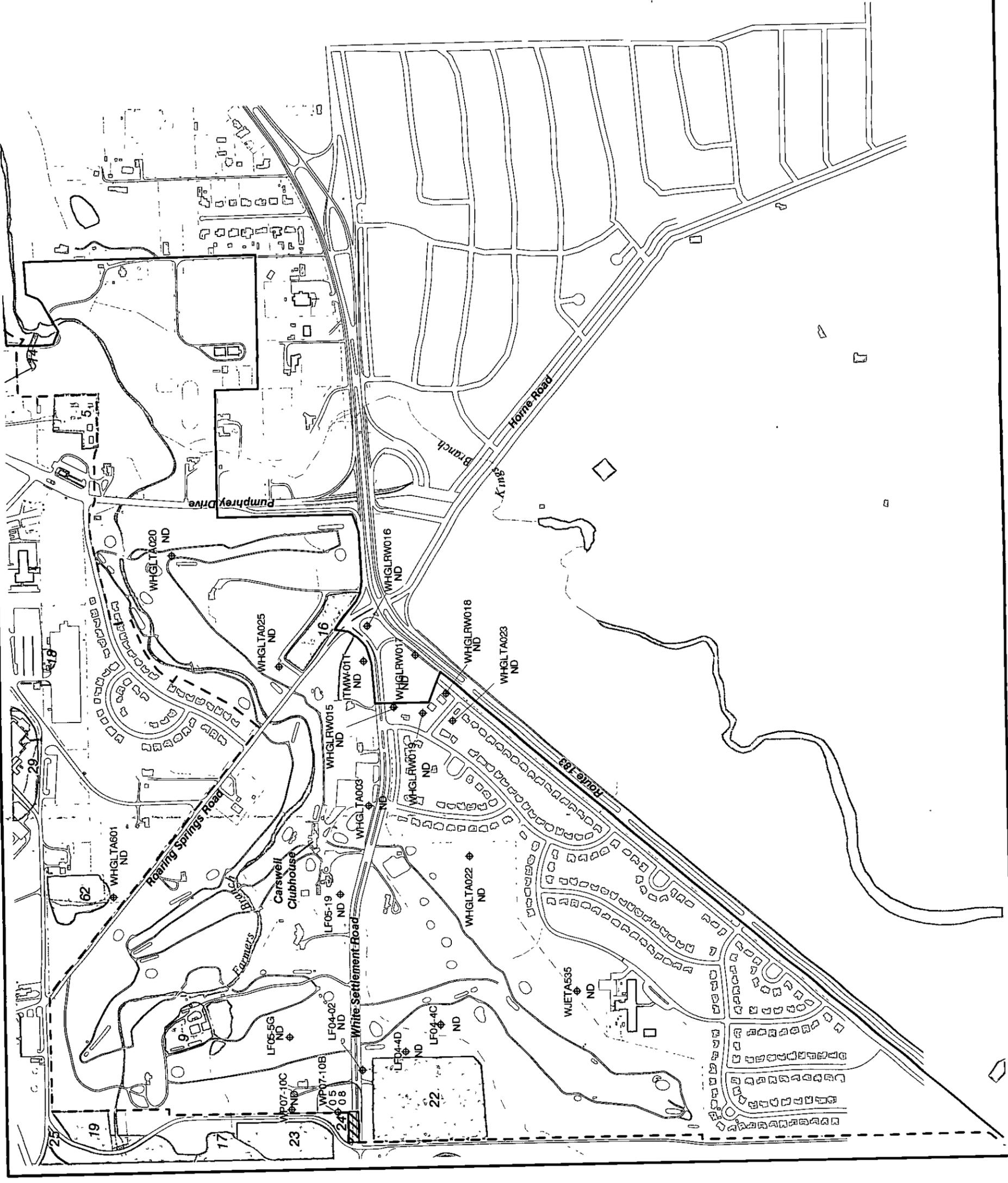
- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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Project AFC001-36CB
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Revised 12/29/00 cf
Map Source HGL ArcView GIS Database 2000



HydroGeologic, Inc. — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

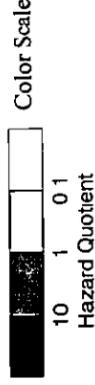
Figure 34
Arsenic
Non-Carcinogenic Risk Isopleth
For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 6 45 E-07 Hazard Quotient
- ND Highest Concentration (µg/L) of Constituent in 1999

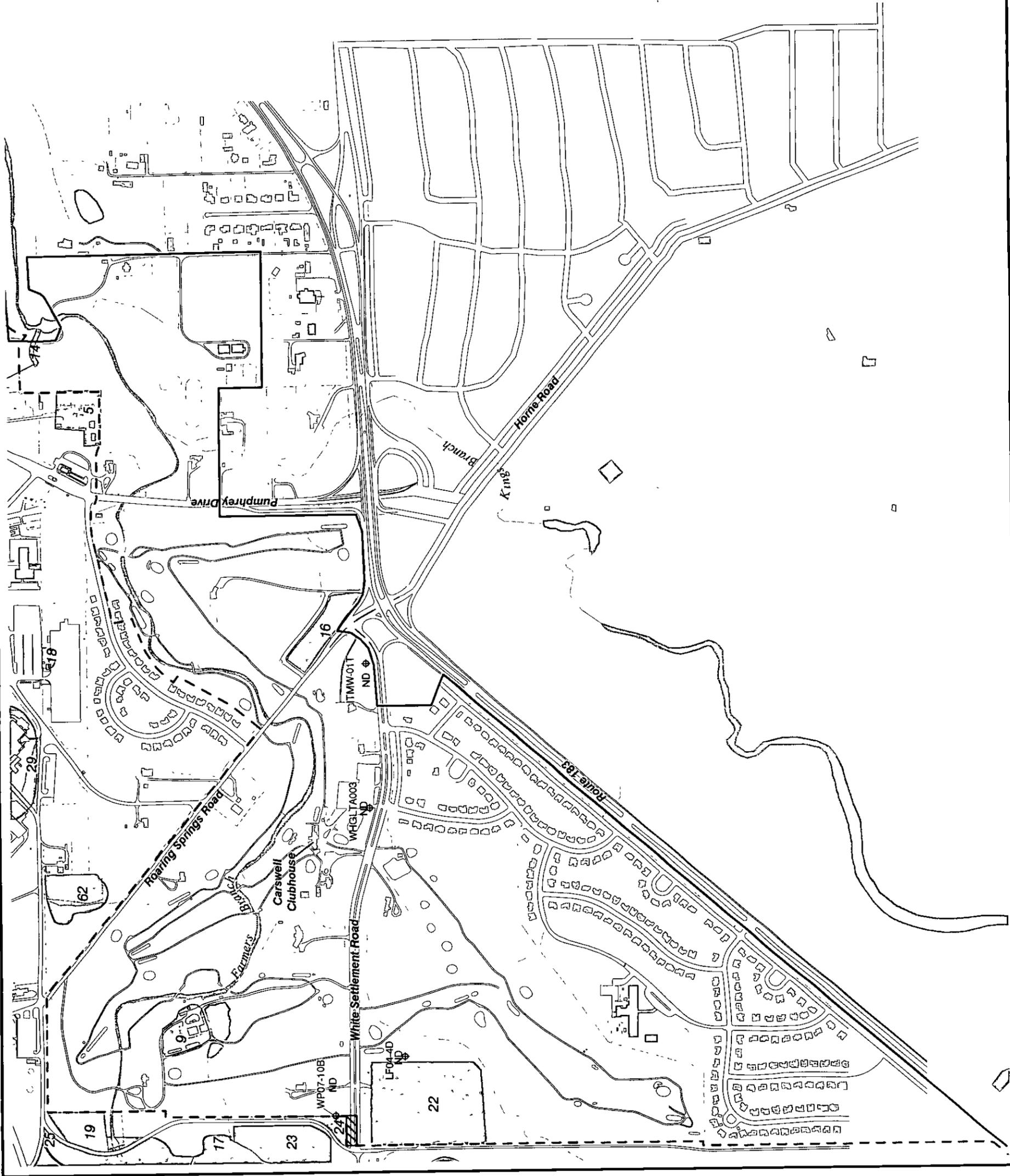


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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HydroGeologic, Inc.—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

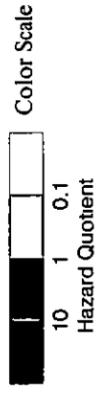
Figure 35 Chromium Non-Carcinogenic Risk Isoleth For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 0.03 Hazard Quotient
- 10 Highest Concentration (µg/L) of Constituent in 1999

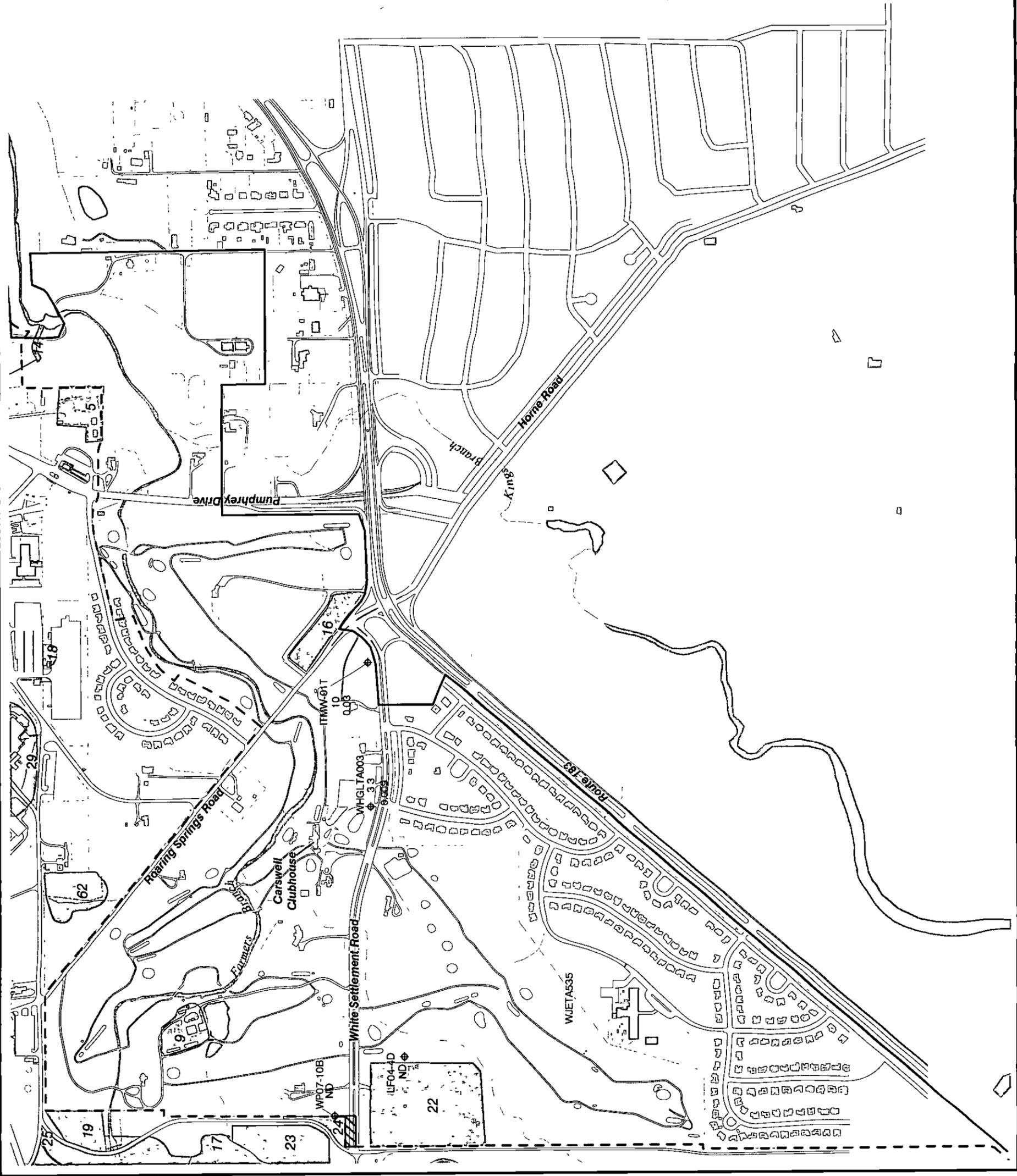


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No. 7
- 22 Landfill No. 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No. 8
- 29 Landfill No. 2
- 62 Landfill No. 6



HydroGeologic, Inc.—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

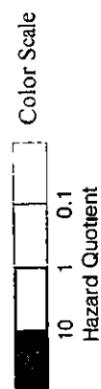
Figure 36
Bis (2-ethylhexyl) phthalate
Non-Carcinogenic Risk Isoleth
For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 0.0406 Hazard Quotient
- 29 Highest Concentration (µg/L) of Constituent in 1999



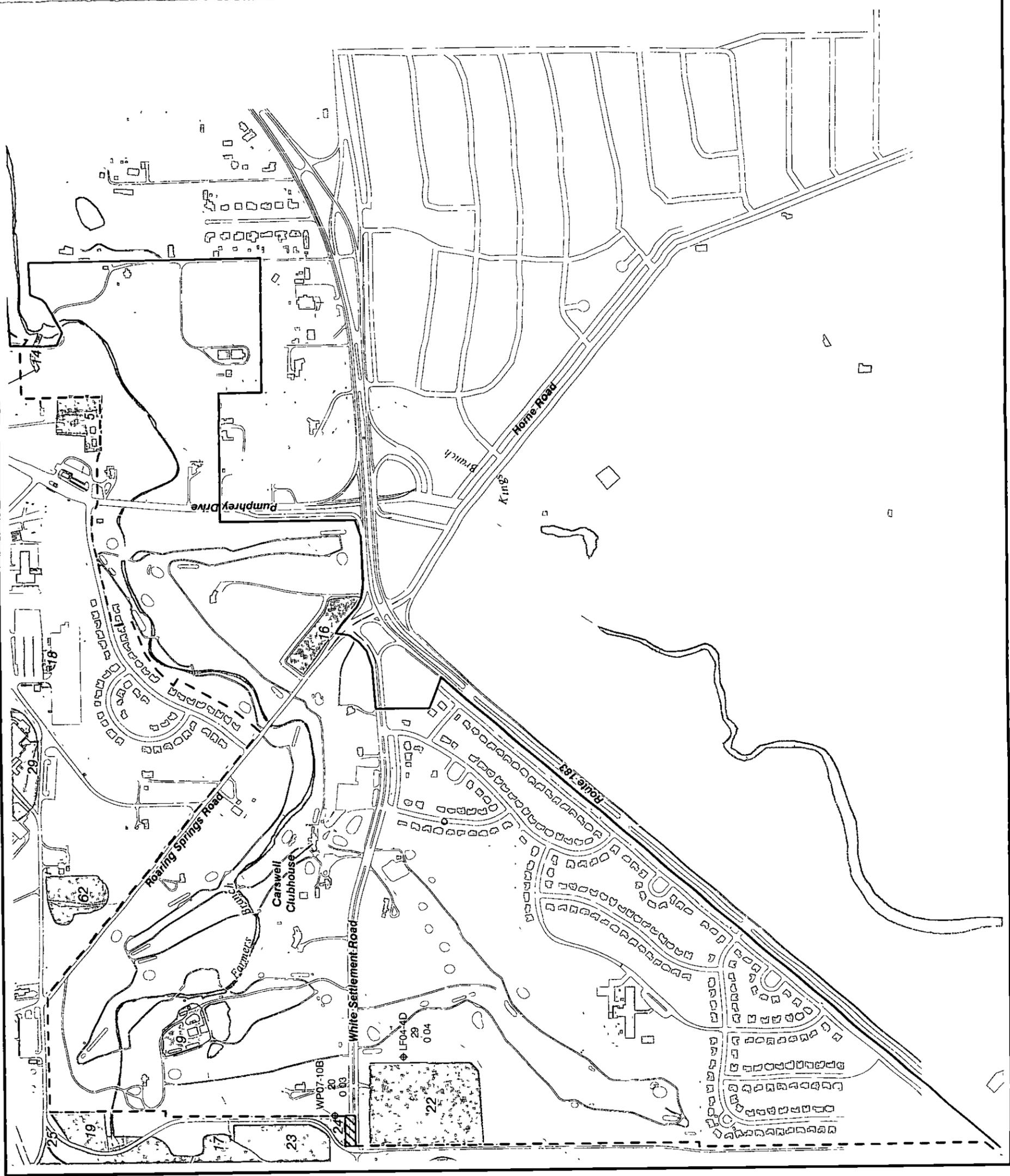
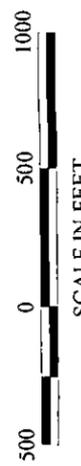
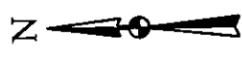
Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area



Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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HydroGeologic, Inc.—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

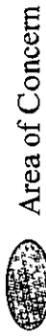
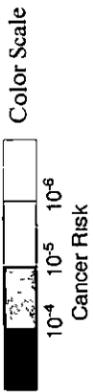
Figure 37 Cumulative Organic Carcinogenic Risk Isoleth For Construction Workers



U.S. Air Force Center for
Environmental Excellence

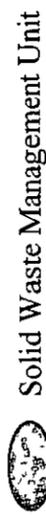
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- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ◆ Monitoring Wells
- 5.39E-07 Risk



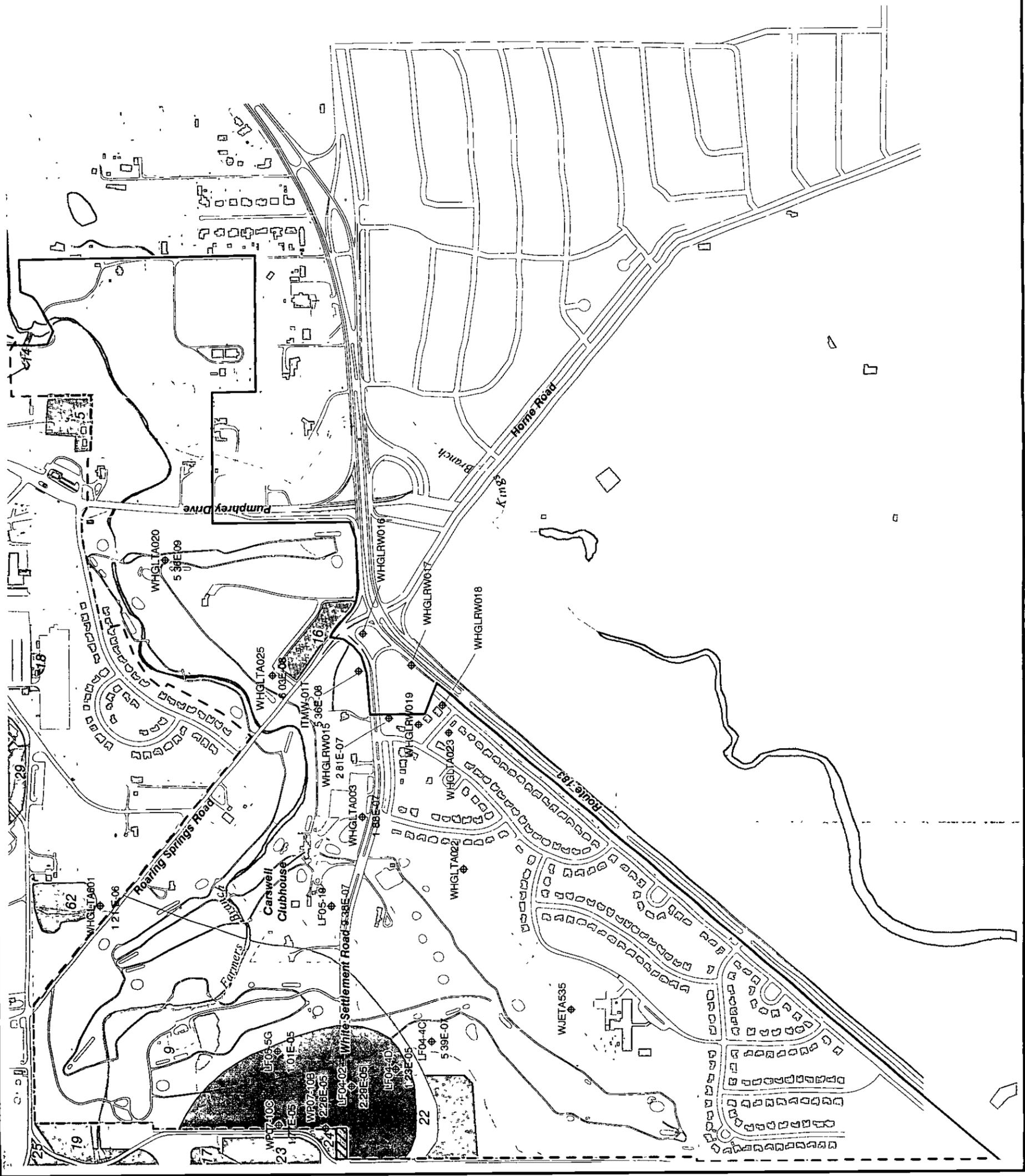
Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area



Solid Waste Management Unit

- 17 Landfill No. 7
- 22 Landfill No. 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No. 8
- 29 Landfill No. 2
- 62 Landfill No. 6



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HydroGeologic, Inc. — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

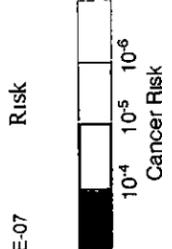
Figure 39 Cumulative Organic Carcinogenic Risk Isopleth For Residents



U.S. Air Force Center for
Environmental Excellence

Legend

- - - - - NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 3.76E-07 Risk

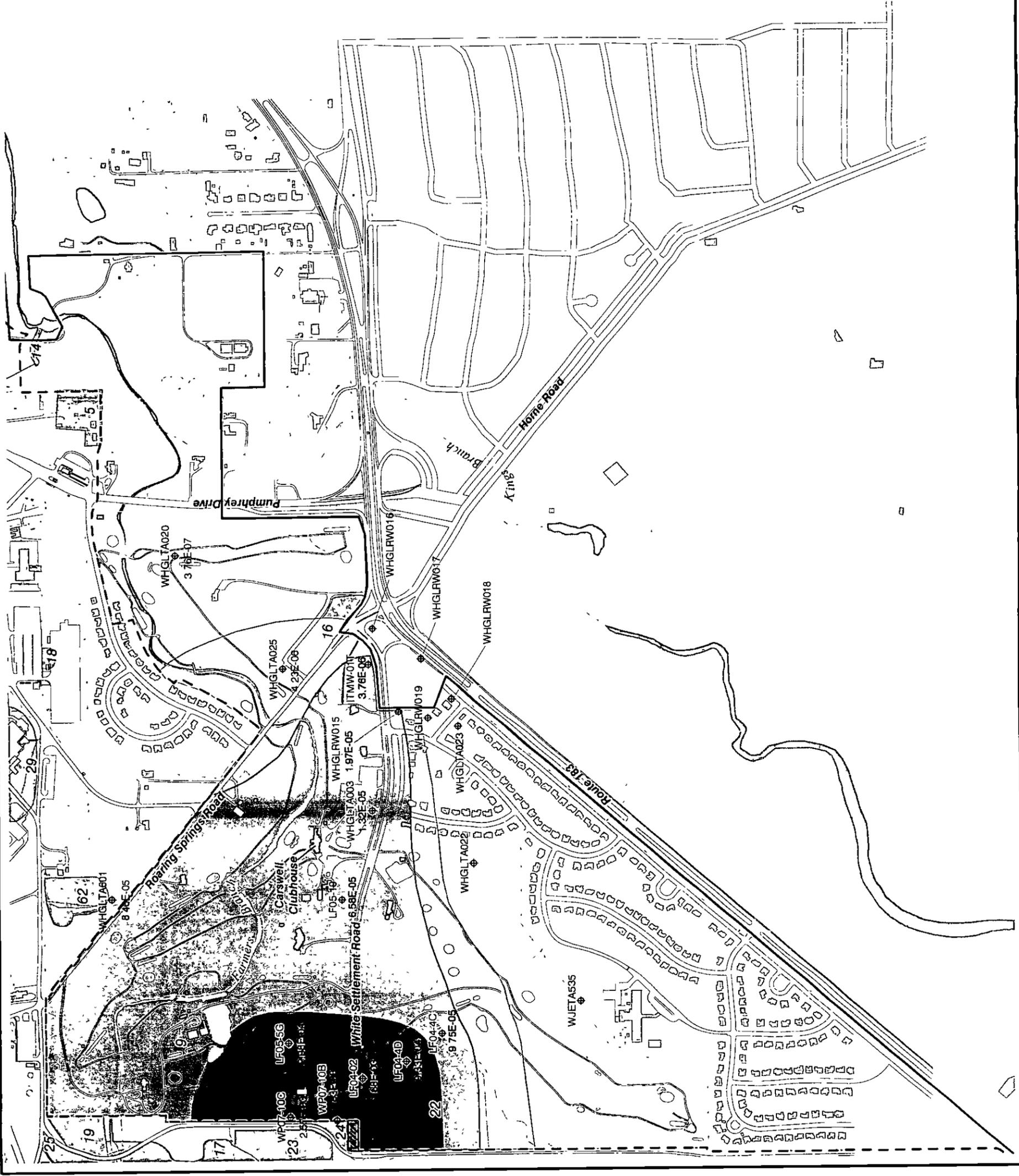


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No. 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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 Project AFC001-36CB
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 Revised 12/28/00 jb
 Map Source HGL ArcView GIS Database 2000



Figure 40 Cumulative Organic Non-Carcinogenic Risk Isopleth For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells
- 0.634 Hazard Quotient

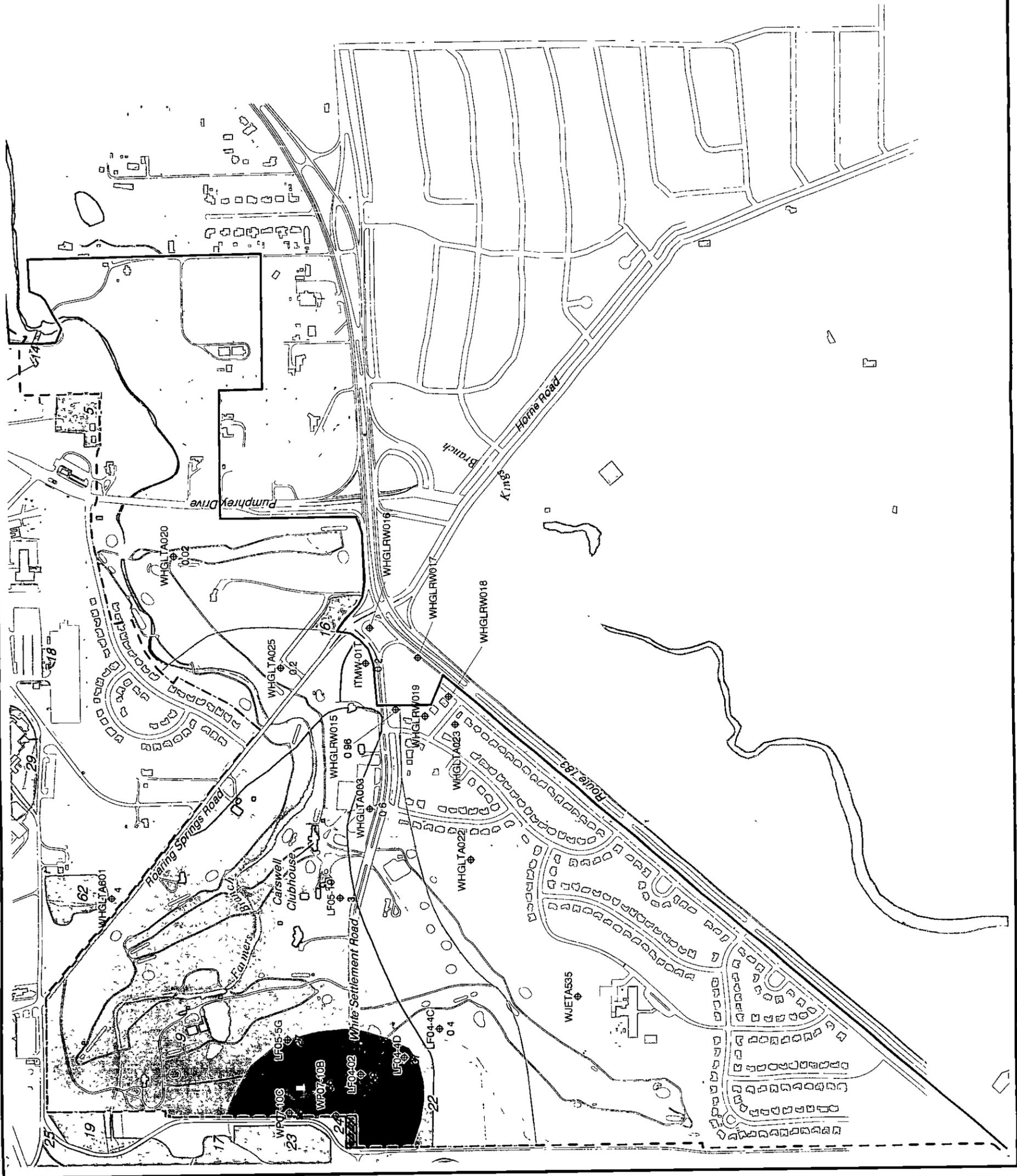


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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HydroGeologic, Inc—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

Figure 41 Cumulative Metals Carcinogenic Risk Isoleth For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

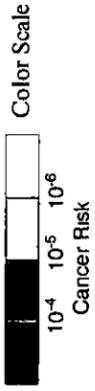
--- NAS Fort Worth JRB (Carswell Field)

— Former Carswell Air Force Base

▨ Groundwater Treatment System

⊕ Monitoring Wells

6 45 E-07 Risk (Note: No risk based on 1999 data.)

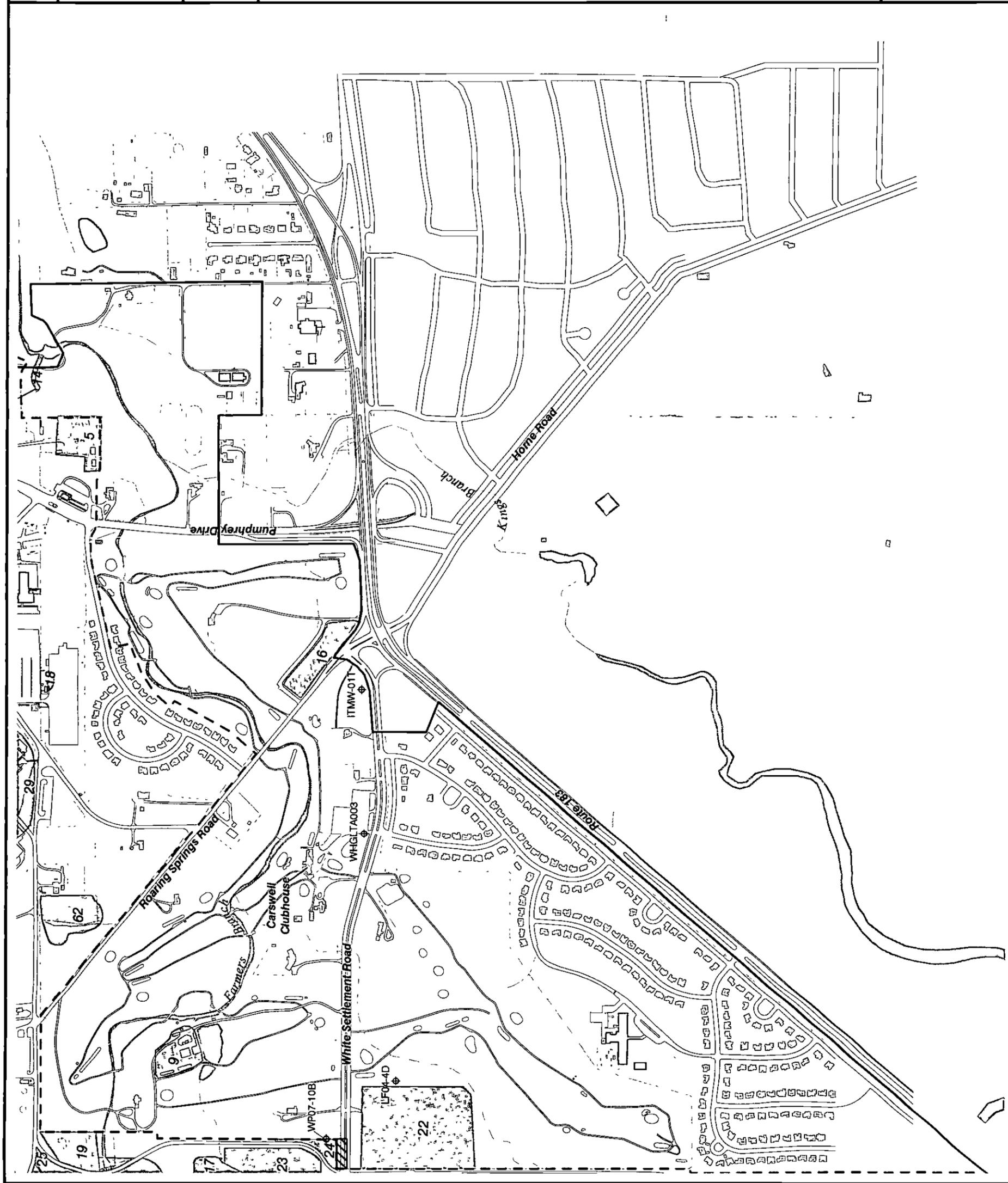


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



HydroGeologic, Inc.—Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

Figure 42 Cumulative Metals Carcinogenic Risk Isopleth For Residents

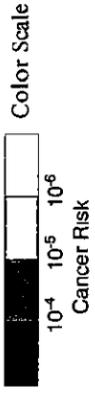


U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ⊕ Monitoring Wells

6 45 E-07 Risk (Note: No risk based on 1999 data.)



Area of Concern

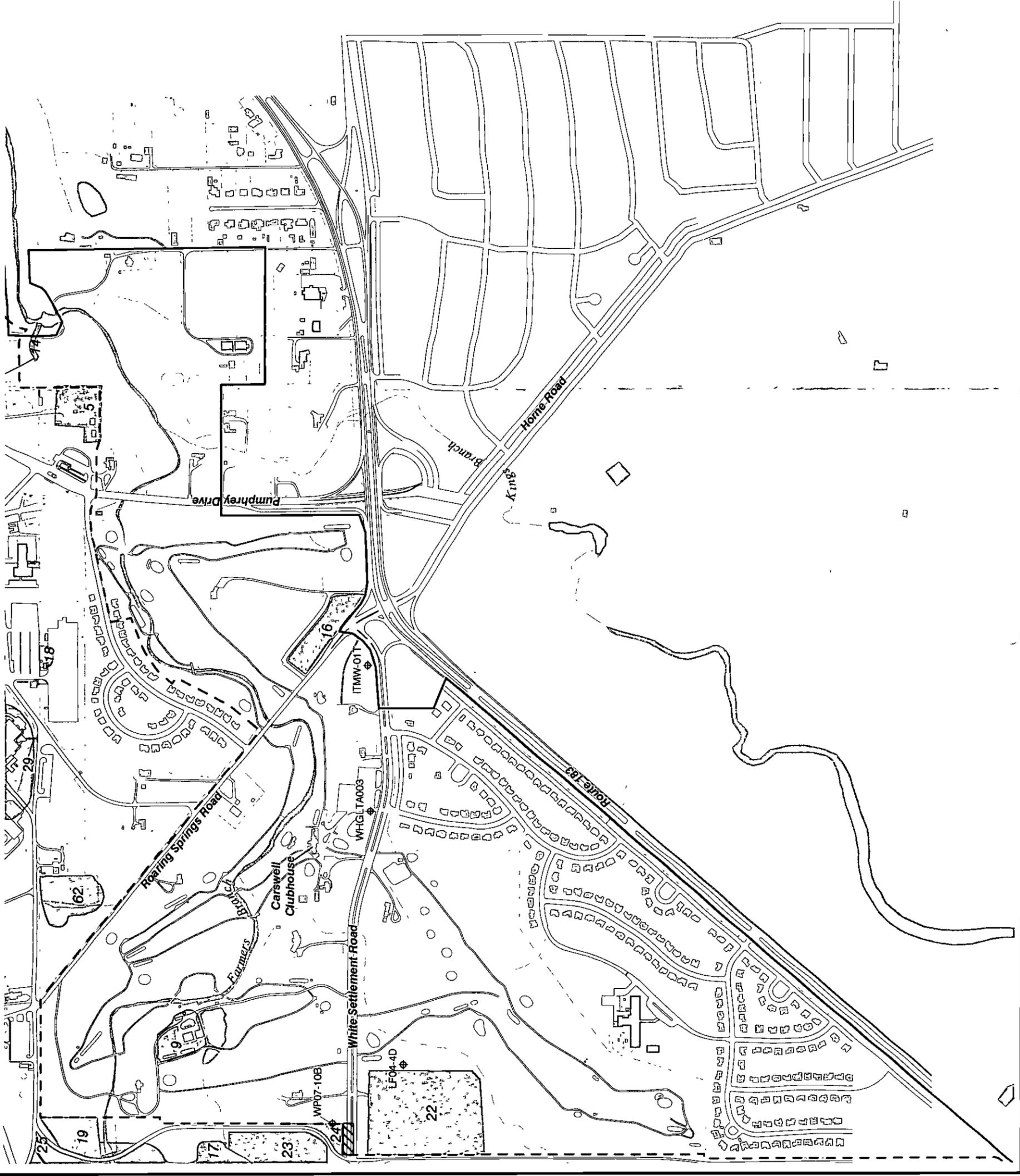
- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



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Map Source: HGL ArcView GIS Database 2000



HydroGeoLogic, Inc. — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

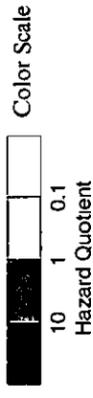
Figure 43 Cumulative Metals Non-Carcinogenic Risk Isoleth For Residents



U.S. Air Force Center for
Environmental Excellence

Legend

- - - - - NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ◆ Monitoring Wells
- 0.3 Hazard Quotient

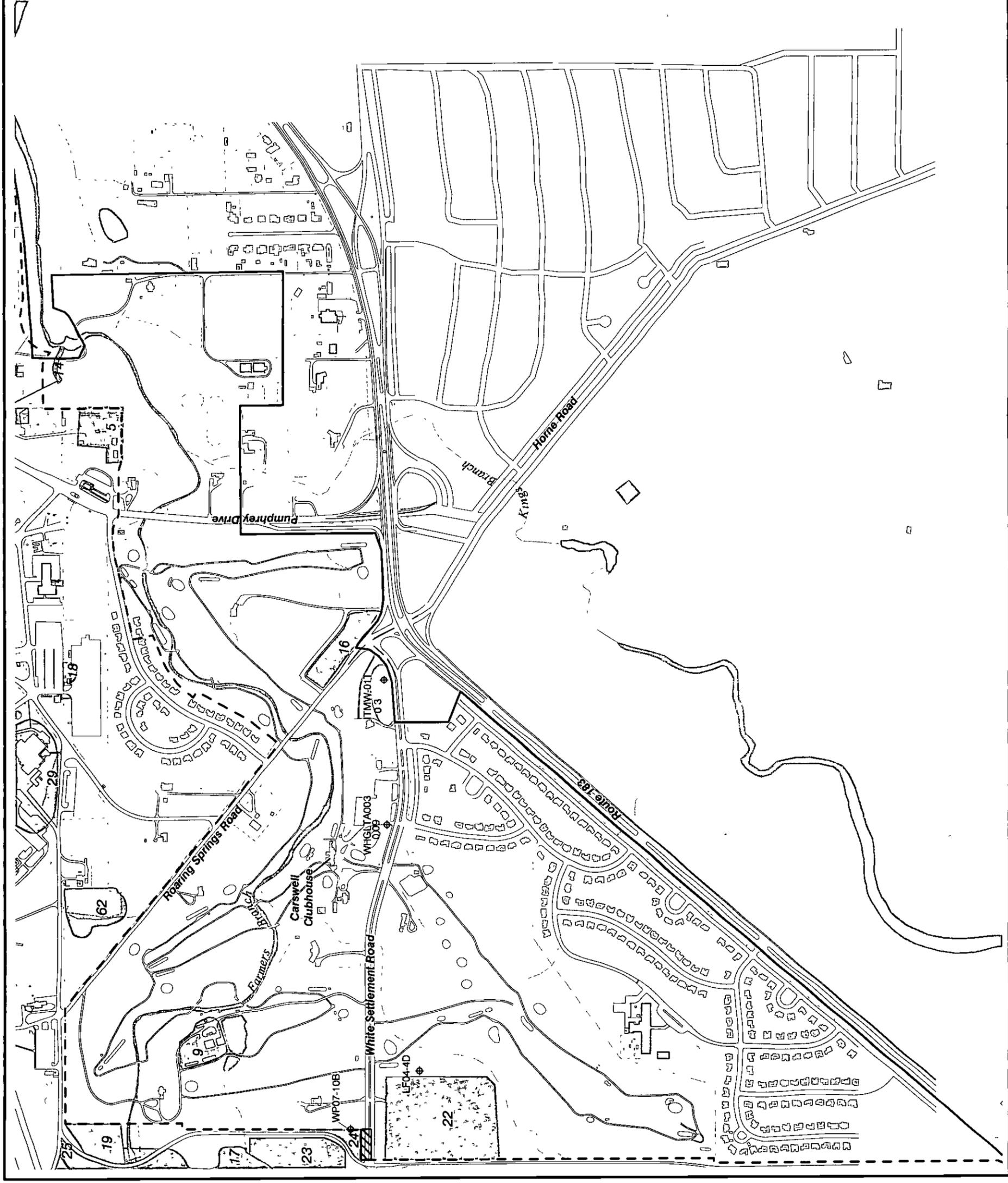
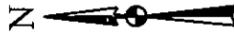


Area of Concern

- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No. 7
- 22 Landfill No. 4
- 23 Landfill No. 5
- 24 Waste Burial Area
- 25 Landfill No. 8
- 29 Landfill No. 2
- 62 Landfill No. 6



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Created 11/28/00 cfarmer
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HydroGeologic, Inc — Baseline Risk Assessment
Former Carswell AFB, Fort Worth, Texas

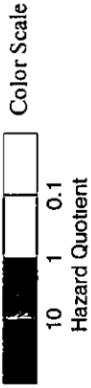
Figure 44 Cumulative Metals Non-Carcinogenic Risk Isopleth For Construction Workers



U.S. Air Force Center for
Environmental Excellence

Legend

- NAS Fort Worth JRB (Carswell Field)
- Former Carswell Air Force Base
- ▨ Groundwater Treatment System
- ◆ Monitoring Wells
- 0.03 Hazard Quotient



Area of Concern

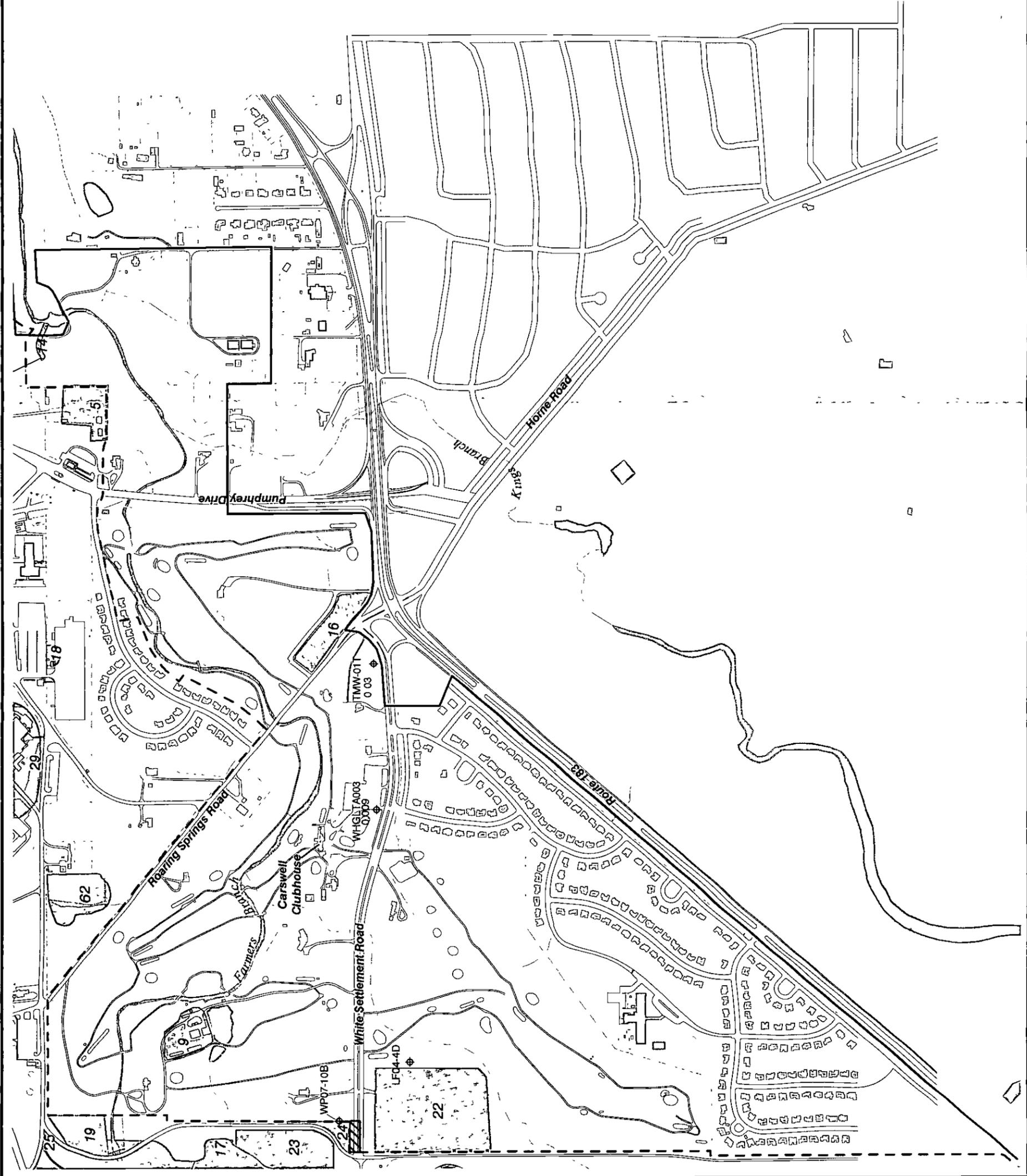
- 5 Grounds Maintenance Yard
- 9 Golf Course Maintenance Yard
- 13 O/W Separator
- 14 Unnamed Stream
- 16 Family Camp
- 18 Suspected Former Fire Training Area
- 19 Suspected Former Fire Training Area

Solid Waste Management Unit

- 17 Landfill No 7
- 22 Landfill No 4
- 23 Landfill No 5
- 24 Waste Burial Area
- 25 Landfill No 8
- 29 Landfill No 2
- 62 Landfill No 6



Filename X:\afco01\366cal\Report\metals_feasibility_study.apr
Project AFC001-36CB
Created 11/28/00 cfarmer
Revised 12/29/00 cf
Map Source HGL ArcView GIS Database 2000



TAB

APPENDIX A

Appendix A

Johnson and Ettinger Model Input Parameters

Appendix A
DATA ENTRY SHEET

VERSION 1.2
September, 1998

CALCULATE RISK-BASED GROUNDWATER CONCENTRATION (enter "X" in "YES" box)

YES X OR

CALCULATE INCREMENTAL RISKS FROM ACTUAL GROUNDWATER CONCENTRATION (enter "X" in "YES" box and initial groundwater conc below)

YES

ENTER Initial groundwater conc., C_w ($\mu\text{g/L}$)

79016 3300

Chemical
Trichloroethylene

ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER
Chemical CAS No (numbers only, no dashes)	Initial groundwater conc., C_w ($\mu\text{g/L}$)	Depth of enclosed space floor, L_f (cm)	Depth below grade to bottom of soil, L_{WT} (cm)	Depth below grade to water table, L_{WT} (cm)	Totals must add up to value of L_{WT} (cell D28)	Soil stratum directly above water table, (Enter A, B, or C)	Soil stratum directly above water table, (Enter A, B, or C)	Soil stratum directly above water table, (Enter A, B, or C)	User-defined stratum A soil vapor permeability, k_v (cm^2)
79016	3300	200	595	334	61	334	334	334	
ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER
Average groundwater temperature, T_g ($^{\circ}\text{C}$)	Depth of enclosed space floor, L_f (cm)	Depth below grade to bottom of soil, L_{WT} (cm)	Depth below grade to water table, L_{WT} (cm)	Thickness of soil stratum A, (Enter value or 0) (cm)	Thickness of soil stratum B, (Enter value or 0) (cm)	Thickness of soil stratum C, (Enter value or 0) (cm)	Soil stratum directly above water table, (Enter A, B, or C)	Soil stratum directly above water table, (Enter A, B, or C)	Soil stratum A SCS soil type (used to estimate soil vapor permeability)
10	200	595	334	61	334	334	334	334	CL

ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER
Stratum A bulk density, P_b^A (g/cm^3)	Stratum A soil dry soil total porosity, n^A (unitless)	Stratum A soil water-filled porosity, θ_w^A (cm^3/cm^3)	Stratum A soil dry soil total porosity, n^B (unitless)	Stratum B soil water-filled porosity, θ_w^B (cm^3/cm^3)	Stratum B soil dry soil total porosity, n^C (unitless)	Stratum C soil water-filled porosity, θ_w^C (cm^3/cm^3)	Stratum C soil dry soil total porosity, n^C (unitless)	Stratum C soil dry soil total porosity, n^C (unitless)	Stratum C soil water-filled porosity, θ_w^C (cm^3/cm^3)	Stratum C soil water-filled porosity, θ_w^C (cm^3/cm^3)	Stratum C soil water-filled porosity, θ_w^C (cm^3/cm^3)
1.325	0.5	0.425	0.4	0.34	0.4	0.34	0.4	0.43	0.43	0.43	0.255

ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER
Enclosed space thickness, L_{enc} (cm)	Soil-bldg pressure differential, ΔP (g/cm^2)	Enclosed space floor length, L_g (cm)	Enclosed space floor width, W_g (cm)	Enclosed space height, H_g (cm)	Enclosed space width, W (cm)	Floor-wall seam crack width, w (cm)	Indoor air exchange rate, ER (1/h)				
15	40	861	861	488	0.1	0.45	0.45	0.45	0.45	0.45	0.45

ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER	ENTER
Averaging time for carcinogens, AT_c (yrs)	Averaging time for noncarcinogens, AT_{nc} (yrs)	Exposure duration, ED (yrs)	Exposure frequency, EF (d/yr)	Target risk for carcinogens, TR (unitless)	Target risk for noncarcinogens, TR (unitless)	Target hazard quotient for carcinogens, THQ (unitless)	Target hazard quotient for noncarcinogens, THQ (unitless)	Target hazard quotient for carcinogens, THQ (unitless)	Target hazard quotient for noncarcinogens, THQ (unitless)
30	30	350	350	1.0E-06	1.0E-06	1	1	1	1

Used to calculate risk-based groundwater concentration

Appendix A
CHEMICAL PROPERTIES SHEET

Diffusivity in air, D_a (cm^2/s)	Henry's law constant at reference temperature, H ($\text{atm}\cdot\text{m}^3/\text{mol}$)	Henry's law constant reference temperature, T_R ($^\circ\text{C}$)	Enthalpy of vaporization at the normal boiling point, $\Delta H_{v,b}$ (cal/mol)	Normal boiling point, T_b ($^\circ\text{K}$)	Critical temperature, T_c ($^\circ\text{K}$)	Organic carbon partition coefficient, K_{oc} (cm^3/g)	Pure component water solubility, S (mg/L)	Unit risk factor, URF ($\mu\text{g}/\text{m}^3$) ⁻¹	Reference conc., RfC (mg/m^3)	
7 90E-02	9 10E-06	1 03E-02	25	7.505	360 36	544 20	1 66E+02	1.10E+03	1 7E-06	0 0E+00

Appendix A
INTERMEDIATE CALCULATIONS SHEET

Exposure duration, τ (sec)	Source-building separation, L_I (cm)	Stratum A soil air-filled porosity, θ_A^A (cm^3/cm^3)	Stratum B soil air-filled porosity, θ_B^B (cm^3/cm^3)	Stratum C soil air-filled porosity, θ_C^C (cm^3/cm^3)	Stratum A effective total fluid saturation, S_{ie} (cm^3/cm^3)	Stratum A soil intrinsic permeability, k_i (cm^2)	Stratum A soil relative air permeability, k_{rg} (cm^2)	Stratum A soil effective vapor permeability, k_v (cm^2)	Thickness of capillary zone, L_{cz} (cm)	Total porosity in capillary zone, n_{cz} (cm^3/cm^3)	Air-filled porosity in capillary zone, $\theta_{w,cz}$ (cm^3/cm^3)	Water-filled porosity in capillary zone, $\theta_{w,cz}$ (cm^3/cm^3)	Floor-wall seam perimeter, X_{crack} (cm)
9.46E+08	395	0.075	0.060	0.175	0.815	9.64E-10	0.332	3.20E-10	30.00	0.43	0.084	0.346	3.844

Bldg ventilation rate, $Q_{building}$ (cm^3/s)	Area of enclosed space below grade, A_B (cm^2)	Crack-to-total area ratio, η (unitless)	Crack depth below grade, Z_{crack} (cm)	Enthalpy of vaporization at ave groundwater temperature, $\Delta H_{v,ts}$ (cal/mol)	Henry's law constant at ave groundwater temperature, H'_{ts} (unitless)	Henry's law constant at ave groundwater temperature, H'_{ts} (unitless)	Vapor viscosity at ave soil temperature, μ_{ts} (g/cm-s)	Stratum A effective diffusion coefficient, D_{eff}^A (cm^2/s)	Stratum B effective diffusion coefficient, D_{eff}^B (cm^2/s)	Stratum C effective diffusion coefficient, D_{eff}^C (cm^2/s)	Capillary zone effective diffusion coefficient, D_{eff}^{cz} (cm^2/s)	Total overall effective diffusion coefficient, D_{eff}^{total} (cm^2/s)	Diffusion path length, L_d (cm)
5.63E+04	1.69E+06	2.27E-04	200	8,557	4.79E-03	2.06E-01	1.75E-04	6.69E-05	4.97E-05	1.29E-03	1.19E-04	2.30E-04	395

Convection path length, L_p (cm)	Source vapor conc, C_{source} ($\mu\text{g}/\text{m}^3$)	Crack radius, r_{crack} (cm)	Average vapor flow rate into bldg, Q_{soil} (cm^3/s)	Crack effective diffusion coefficient, D_{crack} (cm^2/s)	Area of crack, A_{crack} (cm^2)	Exponent of foundation Pecliet number, exp(Pe')	Infinite source indoor attenuation coefficient, α (unitless)	Unit nsk factor, URF ($\mu\text{g}/\text{m}^3$)	Reference conc, RFC (mg/m^3)	
200	2.06E+02	0.10	2.12E-01	6.69E-05	3.84E+02	6.23E+53	3.10E-06	6.40E-04	1.7E-06	NA

Appendix A
RESULTS SHEET

RISK-BASED GROUNDWATER CONCENTRATION CALCULATIONS

Indoor exposure groundwater carcinogen conc. (µg/L)	Indoor exposure groundwater conc. noncarcinogen (µg/L)	Risk-based indoor exposure groundwater conc. (µg/L)	Pure component water solubility, S (µg/L)	Final indoor exposure groundwater conc. (µg/L)
2.24E+03	NA	2.24E+03	1.10E+06	2.24E+03

INCREMENTAL RISK CALCULATIONS

Incremental risk from vapor intrusion to indoor air, carcinogen (unitless)	Hazard quotient from vapor intrusion to indoor air, noncarcinogen (unitless)
NA	NA

ERROR SUMMARY BELOW: (DO NOT USE RESULTS IF ERRORS ARE PRESENT)

Appendix A
VLOOKUP TABLES

SCS Soil Type	K _s (cm/h)	α (1/cm)	N (unitless)	M (unitless)	D _w (cm ² /s)	D _a (cm ² /s)	Diffusivity in air, D _a (cm ² /s)	Diffusivity in water, D _w (cm ² /s)	Pure component water solubility, S (mg/L)	Henry's law constant, H ¹ (unitless)	Henry's law constant at reference temperature, H (atm-m ³ /mol)	Henry's law reference temperature, T _R (°C)	Normal boiling point, T _b (°K)	Critical temperature, T _c (°K)	Enthalpy of vaporization at the normal boiling point, ΔH _{v,b} (cal/mol)	Unit risk factor, URF (μg/m ³) ⁻¹	Reference conc., RfC (mg/m ³)
C	0.20	0.008	1.09	0.083	0.38	0.068	0.0092	0.016	0.085	0.016	0.016	25	533.15	720.75	11,000	8.7E-05	0.0E+00
CL	0.26	0.019	1.31	0.237	0.41	0.085	0.016	0.016	0.085	0.016	0.016	25	715.90	969.27	15,000	0.0E+00	0.0E+00
L	1.04	0.036	1.56	0.359	0.43	0.078	0.020	0.020	0.078	0.020	0.020	25	605.28	827.85	15,000	0.0E+00	7.0E-03
LS	14.59	0.124	2.28	0.561	0.41	0.057	0.040	0.040	0.057	0.040	0.040	25	743.24	890.41	18,000	2.1E-03	0.0E+00
S	29.70	0.145	2.68	0.627	0.43	0.045	0.044	0.044	0.045	0.044	0.044	25	349.90	558.80	7,127	1.5E-05	0.0E+00
SC	0.12	0.027	1.23	0.187	0.38	0.100	0.025	0.025	0.100	0.025	0.025	25	708.15	1004.79	15,000	2.1E-04	0.0E+00
SCL	1.31	0.059	1.48	0.324	0.39	0.100	0.029	0.029	0.100	0.029	0.029	25	624.24	885.73	13,000	3.7E-04	0.0E+00
SI	0.25	0.018	1.37	0.270	0.46	0.034	0.046	0.046	0.034	0.046	0.046	25	596.55	839.36	13,000	3.7E-04	0.0E+00
SIC	0.02	0.005	1.09	0.083	0.28	0.070	0.0039	0.0039	0.070	0.0039	0.0039	25	613.32	842.25	13,000	4.6E-03	0.0E+00
SICL	0.07	0.010	1.23	0.187	0.43	0.089	0.0058	0.0058	0.089	0.0058	0.0058	25	720.00	751.00	10,000	0.0E+00	1.4E+01
SIL	0.45	0.020	1.41	0.291	0.45	0.067	0.011	0.011	0.067	0.011	0.011	25	334.32	536.40	6,988	2.3E-05	0.0E+00
SL	4.42	0.075	1.89	0.471	0.41	0.085	0.030	0.030	0.085	0.030	0.030	25	458.00	685.00	9,510	4.0E-06	0.0E+00

Chemical Properties Lookup Table

CAS No	Chemical	Organic carbon partition coefficient, K _{oc} (cm ³ /g)	Diffusivity in air, D _a (cm ² /s)	Diffusivity in water, D _w (cm ² /s)	Pure component water solubility, S (mg/L)	Henry's law constant, H ¹ (unitless)	Henry's law constant at reference temperature, H (atm-m ³ /mol)	Henry's law reference temperature, T _R (°C)	Normal boiling point, T _b (°K)	Critical temperature, T _c (°K)	Enthalpy of vaporization at the normal boiling point, ΔH _{v,b} (cal/mol)	Unit risk factor, URF (μg/m ³) ⁻¹	Reference conc., RfC (mg/m ³)
50293	DDT	2.69E+06	1.37E-02	4.95E-06	2.50E-02	3.32E-04	8.10E-06	25	533.15	720.75	11,000	8.7E-05	0.0E+00
50328	Benz(a)pyrene	1.02E+06	4.30E-02	9.00E-06	1.62E-02	4.63E-05	1.19E-06	25	715.90	969.27	15,000	2.1E-03	0.0E+00
51285	2,4-Dinitrophenol	1.00E-02	2.73E-02	8.08E-06	2.78E+03	1.83E-05	4.44E-07	25	605.28	827.85	15,000	0.0E+00	7.0E-03
53703	Dibenz(a,h)anthracene	3.80E+06	2.02E-02	5.19E-06	2.49E-03	6.03E-07	1.47E-08	25	743.24	890.41	18,000	2.1E-03	0.0E+00
56235	Carbon tetrachloride	1.74E+02	7.80E-02	8.80E-06	7.93E+02	1.25E+00	3.05E-02	25	349.90	558.80	7,127	1.5E-05	0.0E+00
56553	Benz(a)anthracene	3.98E+05	5.10E-02	9.00E-06	9.40E-03	1.37E-04	3.34E-06	25	708.15	1004.79	15,000	2.1E-04	0.0E+00
57749	Chlordane	1.20E+05	1.18E-02	4.37E-08	5.60E-02	1.99E-03	4.85E-05	25	624.24	885.73	13,000	3.7E-04	0.0E+00
58889	gamma-HCH (Lindane)	1.07E+03	1.42E-02	7.34E-08	6.80E+00	9.94E-04	1.40E-05	25	596.55	839.36	13,000	3.7E-04	0.0E+00
60571	Dieldrin	2.14E+04	1.25E-02	4.74E-08	1.95E-01	6.19E-04	1.51E-05	25	613.32	842.25	13,000	4.6E-03	0.0E+00
65850	Benzoic Acid	8.00E-01	5.36E-02	7.97E-06	3.50E+03	6.31E-05	1.54E-06	25	720.00	751.00	10,000	0.0E+00	1.4E+01
67641	Acetone	5.75E-01	1.24E-01	1.14E-05	1.00E+06	1.59E-05	3.89E-05	25	329.20	508.10	6,855	0.0E+00	3.5E-01
67663	Chloroform	3.88E+01	1.04E-01	1.09E-05	7.82E+03	1.50E-01	3.68E-03	25	334.32	536.40	6,988	2.3E-05	0.0E+00
67721	Hexachloroethane	1.78E+03	2.50E-03	8.80E-06	5.00E+01	1.59E-01	3.89E-05	25	458.00	685.00	9,510	4.0E-06	0.0E+00
71363	Butanol	6.82E+00	8.00E-02	9.30E-06	7.40E+04	3.81E-04	8.80E-06	25	390.88	563.00	10,346	0.0E+00	3.5E-01
71432	Benzene	5.89E+01	8.80E-02	9.80E-06	1.75E+03	2.28E-01	5.56E-03	25	353.24	562.18	7,342	8.3E-06	0.0E+00
71556	1,1,1-Trichloroethane	1.10E+02	7.80E-02	8.80E-06	1.33E+03	7.05E-01	1.72E-02	25	347.24	545.00	7,136	0.0E+00	1.0E+00
72208	Endrin	1.23E+04	1.25E-02	4.74E-06	2.50E-01	3.08E-04	7.51E-06	25	718.15	886.20	12,000	0.0E+00	1.1E-03
72435	Methoxychlor	8.77E+04	1.56E-02	4.48E-06	4.48E-02	6.48E-04	1.58E-05	25	651.02	848.49	14,000	0.0E+00	1.8E-02
72548	DDD	1.00E+06	1.69E-02	4.78E-06	9.00E-02	1.64E-04	4.00E-06	25	639.80	863.77	14,000	6.9E-05	0.0E+00
72559	DDE	4.47E+06	1.44E-02	5.87E-06	1.20E-01	8.61E-04	2.10E-05	25	636.44	860.38	13,000	9.7E-05	0.0E+00
74839	Methyl bromide	1.05E+01	7.28E-02	1.21E-05	1.52E+04	2.58E-01	2.76E-01	25	276.71	467.00	5,714	0.0E+00	5.0E-03
75014	Vinyl chloride (chloroethene)	1.86E+01	1.06E-01	1.23E-06	2.78E+03	1.11E+00	2.71E-02	25	259.25	432.00	5,250	8.4E-05	0.0E+00
75092	Methylene chloride	1.17E+01	1.01E-01	1.17E-05	1.30E+04	8.95E-02	2.18E-03	25	313.00	510.00	6,706	4.7E-07	3.0E+00
75150	Carbon disulfide	4.71E+01	1.04E-01	1.00E-05	1.19E+03	1.24E+00	3.02E-02	25	319.00	552.00	6,391	0.0E+00	7.0E-01
75252	Bromoform	8.71E+01	1.48E-02	1.03E-05	3.10E+03	2.19E-02	5.34E-04	25	422.35	686.00	9,479	1.1E-06	0.0E+00
75274	Bromodichloromethane	5.50E+01	2.98E-02	1.06E-05	8.74E+03	6.56E-02	1.60E-03	25	363.15	585.85	7,000	1.8E-05	0.0E+00
75343	1,1-Dichloroethane	3.16E+01	7.42E-02	1.05E-05	5.06E+03	2.30E-01	5.61E-03	25	330.55	523.00	6,885	0.0E+00	5.0E-01
75354	1,1-Dichloroethylene	5.89E+01	9.00E-02	1.04E-05	2.25E+03	1.07E+00	2.61E-02	25	304.75	576.05	6,247	5.0E-05	0.0E+00
76448	Heptachlor	1.41E+06	1.12E-02	5.69E-06	1.80E+01	4.47E-02	1.09E-03	25	603.69	846.31	13,000	1.3E-03	0.0E+00
77474	Hexachlorocyclopentadiene	2.00E+05	1.61E-02	7.21E-06	1.80E+00	1.11E+00	2.71E-02	25	512.15	746.00	10,931	0.0E+00	7.0E-05
78591	Isophorone	4.69E+01	6.23E-02	8.76E-06	1.20E+04	2.72E-04	6.63E-06	25	489.35	715.00	10,271	2.7E-07	0.0E+00
78875	1,2-Dichloropropane	4.37E+01	7.82E-02	8.73E-06	2.80E+03	1.15E-01	2.80E-03	25	369.52	572.00	7,590	0.0E+00	4.0E-03
79005	1,1,2-Trichloroethane	5.01E+01	7.80E-02	8.80E-06	4.42E+03	3.74E-02	9.12E-04	25	366.15	602.00	8,322	1.6E-05	0.0E+00
79016	Trichloroethylene	1.66E+02	7.90E-02	9.10E-06	1.10E+03	4.22E-01	1.03E-02	25	380.36	544.20	7,505	1.7E-06	0.0E+00
79345	1,1,2,2-Tetrachloroethane	9.33E+03	4.10E-02	7.90E-06	2.97E+00	1.41E-02	3.44E-04	25	419.60	661.15	8,996	5.8E-05	0.0E+00
83328	Acenaphthene	7.08E+03	4.21E-02	7.69E-06	4.24E+00	6.36E-03	1.55E-04	25	550.54	803.15	12,155	0.0E+00	2.1E-01
84862	Diethylphthalate	2.88E+02	2.56E-02	6.35E-06	1.08E+03	1.85E-05	4.51E-07	25	567.15	757.00	13,733	0.0E+00	2.8E+00
84742	Di-n-butyl phthalate	3.98E+04	4.38E-02	7.86E-06	1.12E+01	3.85E-08	9.39E-10	25	613.15	798.67	14,751	0.0E+00	3.5E-01
85687	Butyl benzyl phthalate	5.75E+04	1.74E-02	4.83E-06	2.69E+00	5.17E-05	1.26E-06	25	660.60	839.68	13,000	0.0E+00	7.0E-01

Appendix A
VLOOKUP TABLES

86306 N-Nitrosodiphenylamine	1 29E+03	3 12E-02	6 35E-06	3 51E+01	2 05E-04	5 00E-06	632 28	890 45	13 000	1 4E-06	0 0E+00
86737 Fluorene	1 38E+04	3 62E-02	7 88E-06	1 98E+00	2 81E-03	6 37E-05	570 44	870 00	12 668	0 0E+00	1 4E-01
87449 Hexachole	3 99E+03	3 90E-02	7 03E-06	7 48E+00	6 26E-07	1 53E-08	627 87	899 00	13 977	5 7E-06	0 0E+00
87683 Hexachloro-1,3-butadiene	5 37E+04	5 81E-02	6 16E-06	3 29E+00	3 34E-01	8 15E-03	486 15	738 00	10 206	2 2E-05	0 0E+00
87865 Pentachlorophenol	5 92E+02	5 80E-02	6 10E-06	1 95E+03	1 00E-06	2 44E-08	582 15	813 20	14 000	3 4E-05	0 0E+00
88062 2,4,6-Trichlorophenol	3 81E+02	3 18E-02	6 25E-06	8 00E+02	3 19E-04	7 78E-06	519 15	748 03	12 000	3 1E-06	0 0E+00
91203 Naphthalene	2 00E+03	5 90E-02	7 50E-06	3 10E+01	1 98E-02	4 83E-04	491 14	748 40	10 373	0 0E+00	1 4E-01
91941 3,3-Dichlorobenzidine	7 24E+02	1 94E-02	6 74E-06	3 11E+00	1 64E-07	4 00E-09	560 26	754 03	13 000	1 3E-04	0 0E+00
95476 o-Xylene	3 62E+02	8 70E-02	1 00E-05	1 78E+02	2 13E-01	5 20E-03	417 60	630 30	8 661	0 0E+00	7 0E+00
95487 2-Methylphenol (o-cresol)	9 12E+01	7 40E-02	8 30E-06	2 80E+04	4 92E-05	1 20E-06	464 19	697 60	10 800	0 0E+00	1 8E-01
95501 1,2-Dichlorobenzene	6 17E+02	6 90E-02	1 56E+02	1 56E+02	7 79E-02	1 90E-03	453 57	705 00	9 700	0 0E+00	2 0E-01
95578 2-Chlorophenol	3 88E+02	5 01E-02	9 46E-06	2 20E+04	1 60E-02	3 90E-04	447 53	675 00	8 572	0 0E+00	1 8E-02
95954 2,4,5-Trichlorophenol	1 60E+03	2 91E-02	7 03E-06	1 20E+03	1 78E-04	4 34E-08	528 15	719 03	13 000	0 0E+00	3 5E-01
98953 Nitrobenzene	6 48E+01	7 60E-02	8 60E-06	2 98E+03	9 84E-04	2 40E-05	483 95	719 00	10 568	0 0E+00	2 0E-03
100414 Ethylbenzene	3 83E+02	7 50E-02	7 80E-06	1 69E+02	3 23E-01	7 88E-03	409 34	617 20	8 501	0 0E+00	1 0E+00
100425 Styrene	7 76E+02	7 10E-02	8 00E-06	3 10E+02	1 13E-01	2 78E-03	418 31	636 00	8 737	0 0E+00	1 0E+00
105679 2,4-Dimethylphenol	2 09E+02	5 84E-02	8 69E-06	7 87E+03	8 20E-05	2 00E-06	484 13	707 60	11 329	0 0E+00	7 0E-02
106423 p-Xylene	3 89E+02	7 69E-02	8 44E-06	1 85E+02	3 14E-01	7 66E-03	411 52	616 20	8 525	0 0E+00	7 0E+00
106487 1,4-Dichlorobenzene	6 17E+02	6 90E-02	7 80E-06	7 38E+01	9 86E-02	2 43E-03	447 21	684 75	9 271	0 0E+00	9 0E-01
106478 p-Chloroaniline	6 61E+01	4 63E-02	1 01E-05	5 30E+03	1 36E-05	3 32E-07	503 85	754 00	11 689	0 0E+00	1 4E-02
107062 1,2-Dichloroethane	1 74E+01	1 04E-01	9 80E-06	8 52E+03	4 01E-02	9 78E-04	358 65	561 00	7 643	2 8E-05	0 0E+00
108054 Vinyl acetate	5 25E+00	8 50E-02	9 20E-06	2 00E+04	2 10E-02	5 12E-04	345 65	519 13	7 800	0 0E+00	2 0E-01
108383 m-Xylene	4 07E+02	7 00E-02	7 80E-06	1 81E+02	3 01E-01	7 34E-03	412 27	617 05	6 523	0 0E+00	7 0E+00
108683 Toluene	1 82E+02	8 70E-02	8 60E-06	5 28E+02	2 72E-01	6 63E-03	383 78	591 79	7 930	0 0E+00	4 0E-01
108807 Chlorobenzene	2 88E+01	8 20E-02	9 10E-06	8 28E+04	1 63E-05	3 98E-07	404 67	632 40	8 410	0 0E+00	2 0E-02
111444 Bis(2-chloroethyl)ether	1 55E+01	6 92E-02	7 53E-06	1 72E+04	7 38E-04	1 80E-06	455 02	694 20	10 920	0 0E+00	2 1E+00
115297 Endosulfan	1 14E+03	1 15E-02	4 55E-06	5 10E-01	4 59E-04	1 12E-05	451 15	659 79	9 000	3 3E-04	0 0E+00
117177 Bis(2-ethylhexyl)phthalate	2 15E+07	3 51E-02	3 66E-06	3 40E-01	4 18E-06	1 02E-07	674 43	942 00	14 000	0 0E+00	2 1E-02
117840 Di-n-octyl phthalate	9 32E+07	1 51E-02	3 58E-06	2 00E-02	2 74E-03	6 68E-05	704 09	862 22	15 999	4 0E-06	0 0E+00
118741 Hexachlorobenzene	5 50E+04	4 2E-02	5 91E-06	6 20E+00	5 41E-02	1 32E-03	562 55	825 00	14 447	4 8E-04	0 0E+00
120127 Anthracene	2 85E+04	3 24E-02	7 74E-06	4 34E-02	2 67E-03	6 51E-05	615 18	873 00	13 121	0 0E+00	1 1E+00
120821 1,2,4-Trichlorobenzene	1 78E+03	3 00E-02	8 23E-06	3 00E+02	5 82E-02	1 42E-03	486 15	725 00	10 471	0 0E+00	2 0E-01
120932 2,4-Dichlorophenol	1 47E+02	3 48E-02	8 77E-06	4 50E+03	1 30E-04	3 17E-06	482 15	708 17	11 000	0 0E+00	1 1E-02
121142 2,4-Dinitrotoluene	9 55E+01	2 03E-01	7 06E-06	2 70E+02	3 80E-06	9 27E-08	590 00	814 00	13 467	1 9E-04	0 0E+00
124481 Chlorodibromomethane	6 31E+01	1 86E-02	1 05E-05	2 60E+03	3 21E-02	7 83E-04	416 14	678 20	8 000	2 4E-05	0 0E+00
127184 Tetrachloroethylene	1 55E+02	7 20E-02	8 20E-06	2 00E+02	7 54E-01	1 84E-02	394 40	620 20	8 288	5 8E-07	0 0E+00
128000 Pyrene	1 05E+05	2 72E-02	7 24E-06	1 35E-01	4 51E-04	1 10E-05	667 95	936 00	14 370	0 0E+00	1 1E-01
156592 cis-1,2-Dichloroethylene	3 55E+01	7 36E-02	1 13E-05	3 50E+03	3 85E-01	4 07E-03	333 65	544 00	7 192	0 0E+00	3 5E-02
156605 trans-1,2-Dichloroethylene	5 25E+01	7 07E-02	1 18E-05	6 30E+03	3 85E-01	4 07E-03	328 85	516 50	6 717	0 0E+00	7 0E-02
193395 Indeno(1,2,3-cd)pyrene	3 47E+06	1 90E-02	5 86E-06	2 20E-05	6 56E-05	1 60E-06	809 15	1078 24	17 000	2 1E-04	0 0E+00
205992 Benzobifluoranthene	1 23E+06	2 28E-02	5 56E-06	1 50E-03	1 11E-04	9 46E-05	715 90	969 27	15 000	2 1E-04	0 0E+00
206440 Fluoranthene	1 07E+05	3 02E-02	6 35E-06	2 06E-01	6 60E-04	1 61E-05	655 95	905 00	13 615	0 0E+00	1 4E-01
207069 Benzokifluoranthene	1 23E+06	2 28E-02	5 56E-06	8 00E-04	3 40E-05	8 29E-07	753 15	1019 70	16 000	2 1E-05	0 0E+00
218019 Chrysene	3 88E+05	2 48E-02	6 21E-06	1 50E-03	3 88E-03	9 46E-05	714 15	979 00	16 455	4 9E-03	0 0E+00
309002 Aldrin	2 45E+06	1 32E-02	4 86E-06	1 80E-01	6 97E-03	1 70E-04	603 01	839 37	13 000	1 8E-03	0 0E+00
318948 alpha-HCH (alpha-BHC)	1 26E+03	1 42E-02	7 34E-06	2 00E+00	4 35E-04	1 06E-05	598 55	839 36	13 000	1 8E-03	0 0E+00
318957 beta-HCH (beta-BHC)	1 26E+03	1 42E-02	7 34E-06	2 40E-01	3 05E-05	7 44E-07	581 15	839 36	13 000	3 7E-05	2 0E-02
542758 1,3-Dichloropropane	4 57E+01	6 26E-02	1 00E-05	2 80E+03	7 26E-01	1 77E-02	381 15	587 38	7 000	3 7E-05	2 0E-02
606202 2,6-Dinitrotoluene	6 92E+01	3 27E-02	7 26E-06	1 82E+02	3 06E-05	7 46E-07	558 00	770 00	12 938	1 9E-04	0 0E+00
621847 N-Nitrosodi-n-propylamine	2 40E+01	5 45E-02	8 17E-06	9 88E+03	9 23E-05	2 25E-06	509 00	746 87	11 000	2 0E-03	0 0E+00
1024573 Heptachlor epoxide	8 32E+04	1 32E-02	4 32E-06	2 00E-01	3 90E-04	9 51E-06	848 76	13 000	13 000	2 6E-03	0 0E+00
7499376 Mercury (elemental)	5 20E+01	3 07E-02	6 30E-06	5 62E-02	4 67E-01	1 14E-02	628 88	1750 00	14 127	0 0E+00	3 0E-04
8001352 Toxaphene	2 57E+05	1 18E-02	4 34E-06	7 40E-01	2 46E-04	6 00E-06	657 15	873 31	14 000	3 2E-04	0 0E+00
1108625 Aroclor 1260 (PCB-1260)	2 90E+05	1 38E-02	4 32E-06	8 00E-02	1 89E-01	4 60E-03	402 50	539 37	19 000	1 0E-04	0 0E+00
11087891 Aroclor 1254 (PCB-1254)	2 00E+05	1 56E-02	5 00E-06	5 70E-02	8 20E-02	2 00E-03	377 50	512 27	19 000	1 0E-04	0 0E+00
12674112 Aroclor 1016 (PCB-1016)	3 30E+04	2 22E-02	5 42E-06	4 20E-01	1 19E-02	2 80E-04	340 50	475 22	18 000	1 0E-04	0 0E+00
53489219 Aroclor 1242 (PCB-1242)	3 50E+04	2 14E-02	5 31E-06	3 40E-01	2 13E-02	5 20E-04	345 50	482 20	16 000	1 0E-04	0 0E+00

TAB

APPENDIX B

Appendix B

Toxicity Profiles for Carcinogenic and Noncarcinogenic COPCs

ACETONE

Human Health Effects

Exposure to acetone may occur through inhalation, ingestion, or dermal exposure. Studies of workers exposed to acetone revealed irritation of the ocular and respiratory tract mucosa, and at high concentrations, central nervous system (CNS) effects (American Conference of Governmental Industrial Hygienists [ACGIH], 1991). Rats exposed by inhalation to high concentrations exhibited narcosis and slight decreases in organ and body weight, compared with controls, but no clinical pathological or histopathological evidence of organ damage. Inhalation reference concentration (RfC) values were not located for acetone. Oral toxicity data are limited to a comprehensive 90-day gavage study in rats, in which 100 mg/kg/day was a no observed effect level (NOEL) and 500 mg/kg/day was the lowest observed adverse effect level (LOAEL) associated with increased liver and kidney weight and tubular nephropathy (EPA, 1996). A verified reference dose (RfD) for chronic oral exposure of 0.1 mg/kg/day was derived by applying an uncertainty factor of 1,000 to the NOEL of 100 mg/kg/day. The EPA (1995) presented a provisional subchronic oral RfD of 1 mg/kg/day, based on the same NOEL and an uncertainty factor of 100. The target organs for inhalation exposure to acetone are the CNS and the respiratory and ocular mucosa. Target organs for oral exposure are the liver and the kidney. There is no evidence to suggest that acetone is carcinogenic or mutagenic and the EPA has classified acetone in Group D (EPA, 1996).

Ecological Effects

Acetone is widely used as a chemical intermediate and solvent. It is also released from volcanoes and forest fires and it a metabolic product released by plants and animals (National Library of Medicine [NLM], 1993). Acetone in soil is expected to volatilize and leach into the groundwater (NLM, 1993). It may also be degraded by microorganisms (NLM, 1993).

Acetone has been detected as a natural volatile metabolite in onions, apples, grapes, cauliflower, tomatoes, morning glory, and wild mustard (Graedel et al., 1986 as cited in NLM, 1993). It has also been identified as a volatile component of baked potatoes,

roasted filbert nuts, and in dried legumes (NLM, 1993). Information on the phytotoxicity of acetone is very limited.

Because acetone is highly volatile, it can easily be inhaled (International Labour Office [ILO], 1983 as cited in NLM, 1993). It may also be absorbed through the skin (ILO, 1983 as cited in NLM, 1993). Elimination of acetone and its metabolites occur primarily via the lungs or in urine (ILO, 1983 as cited in NLM, 1993). Acetone has been measured in insects as a naturally occurring volatile metabolite (Graedel et al., 1986 as cited in NLM, 1993). It has also been identified as a component of human breath (Conkle et al., 1975 as cited in Howard, 1990). Concentrations of acetone in wild birds and mammals could not be located in the literature.

Specific data on the toxicity of acetone to wildlife do not exist. Exposure of mammals to acetone can induce a depression of the central nervous system, loss of corneal reflexes (Clayton and Clayton, 1982 as cited in NLM, 1993). Oral LD₅₀ values for rats, mice, and rabbits exposed to acetone are 5.8 g/kg, 3 g/kg, and 5.34 g/kg, respectively (Registry of Toxic Effects of Chemical Substances [RTECS], 1993). Dermal LD₅₀ values for rabbits and guinea pigs exposed to acetone are 20 g/kg and >7.407 g/kg, respectively (RTECS, 1993). An inhalation LC₅₀ value of 50.1 g/m³/8hr has been determined for rats exposed to acetone (RTECS, 1993). Adverse impacts on fertility have been reported in male rats exposed to 273 g/kg acetone for 13 weeks prior to mating (RTECS, 1993). Wildlife NOAELs for acetone based on extrapolations from laboratory rat studies are 20 mg/kg for the white-footed mouse, 16.8 mg/kg/d for the meadow vole, and 5.3 mg/kg/d for the red fox (Opresko et al., 1996).

Acetone in aquatic environments is expected to biodegrade and volatilize (NLM, 1993). Adsorption to sediment and bioconcentration in biota are not expected to be significant (NLM, 1993). A bioconcentration factor of 0.69 has been reported for adult haddock exposed to acetone (Lyman et al., 1982 as cited in NLM, 1993).

Data on the toxicity of acetone to freshwater biota are limited. Federal Water Quality Criteria does not exist for the protection of freshwater aquatic life from exposure to acetone (EPA, 1986). The Ohio EPA Warmwater Habitat Water Quality Criteria for the exposure to acetone is 78 mg/L. Suter et al. (1992), however, recommend acute

and chronic advisory values of 31,000 $\mu\text{g/L}$ and 770 $\mu\text{g/L}$, respectively for the protection of freshwater biota. Lowest chronic toxicity values of acetone to fish and daphnids are estimated as 507,640 $\mu\text{g/L}$ and 1,560 $\mu\text{g/L}$, respectively (Suter and Tsao, 1996). The test EC_{20} for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than a 20% reduction in either the weight of young fish per initial female fish in a life cycle or partial life cycle test or the weight of young per egg in an early life stage test (Suter and Tsao, 1996). The value for acetone has been estimated to be 161,867 $\mu\text{g/L}$ (Suter and Tsao, 1996).

REFERENCES

American Conference of Government Industrial Hygienists (ACGIH), 1991, Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed., Cincinnati, OH.

Clayton, G. D. and F. E. Clayton, 1982, Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology, 3rd edition, John Wiley and Sons, New York, New York.

Conkle, J. P., et al., 1975, Archives Environmental Health, 30:290-295.

Graedel, T. E., et al., 1986, Atmospheric Chemical Compounds, Academy Press, New York, New York.

National Library of Medicine (NLM), 1993, Hazardous Substance Data Bank, produced by Micromedex, Inc.

Howard, P. H. (ed.), 1990, Handbook of Environmental Fate and Exposure Data for Organic Chemicals, Vol. 2, Solvents, Lewis Publishers, Chelsea, Michigan.

International Labour Office (ILO), 1983, Encyclopedia of Occupational Health and Safety, Vol. I and II, International Labour Office, Geneva, Switzerland.

Lyman, W. J., et al., 1982, Handbook of Chemical Property Estimation Methods, McGraw-Hill, New York, New York.

Opresko, D. M., B. E. Sample, and G. W. Suter II, 1996, Toxicological Benchmarks for Wildlife: 1996 Revision, Environmental Sciences Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee. ORNL/ES/ER/TM-86/R3.

Registry of Toxic Effects of Chemical Substances (RTECS), 1993, Produced by Micromedex, Inc.

Suter, G. W. II, M. A. Futrell, and G. A. Kerchner, 1992, Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota on the Oak Ridge Reservation, Oak Ridge, Tennessee, Oak Ridge National Laboratory, Oak Ridge, Tennessee. ORNL/ER-139.

Suter, G. W. II and C. L. Tsao, 1996, Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision, Oak Ridge National Laboratory, Oak Ridge, Tennessee. ORNL/ES/ER/TM-96/R2.

US Environmental Protection Agency (EPA), 1986, Quality Criteria for Water 1986, Office of Water Regulations and Standards, USEPA, Washington, DC. EPA 40/5-86-001.

U.S. Environmental Protection Agency (EPA), 1995, Health Effects Assessment Summary Tables, Annual Update FY1992, Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996, Integrated Risk Information System (IRIS), Computer Database, EPA, Washington, DC.

ALUMINUM

Human Health Effects

Aluminum has long been regarded as a non-toxic metal primarily because of its very low absorption from the gastrointestinal (GI) tract. While it is true that aluminum is not absorbed to a great extent, there may be significant differences in absorption and bioavailability depending on its speciation (EPA, 1987).

The greatest health concern regarding aluminum is its effect on the neurological system. Some association has been implied between aluminum and Alzheimer's Disease. However, there is no evidence for a causative role for aluminum in the development of Alzheimer's Disease. A second target organ for aluminum in both humans and laboratory animals is bone. Several studies have shown that aluminum exposure may cause osteomalacia. Osteomalacia has been documented in humans exposed to aluminum in dialysis fluids. Several researchers have shown a positive correlation between the level of exposure to aluminum, the amount of aluminum present in the bone tissue, and the severity of the disease. Aluminum can also produce adverse hematological effects in both humans and laboratory animals. Dialysis patients who were exposed to high levels of aluminum tended to develop microcytic hypochromic anemia, the mechanism for which is not clear. In a study by Touam et al. (1983) using uremic and normal rats it was determined that when treated with aluminum both types of rats developed microcytic anemia where uremic rats not treated with aluminum displayed normocytic anemia (EPA, 1987).

Although there is sufficient data to demonstrate that aluminum is absorbed from the gastrointestinal tract, the bioavailability and mechanism of absorption are not known (Wilhelm, et al. 1990). Aluminum absorption is dependent on the chemical form, pH of the intestine, concentration of aluminum, and dietary factors. Aluminum absorption can range from 0.27 to 2.18% (Yokel and McNamara, 1988). Aluminum compounds ranked in order of increasing absorption are aluminum borate, aluminum glycinate, aluminum hydroxide, aluminum chloride, sucralfate, aluminum lactate, aluminum nitrate, and aluminum citrate. Dietary factors such as phosphate, citrate, and fluoride with which aluminum can complex influence absorption.

In a subchronic and reproductive toxicity study, Ondreicka et al. (1966) exposed groups of 10 male and female Dobra Voda mice (number of animals per sex not reported) to 0 or 19.3 mg Al/kg/day as aluminum chloride in drinking water for 180-390 days. The diet contained 160 to 180 ppm aluminum (20.8 mg/kg/day, using a food factor of 0.13 kg diet/kg body weight/day, [EPA, 1986]). The total aluminum intake was, therefore, 40.1 mg/kg/day. The F0 group produced 3 litters, the F1a group produced 2 litters. The weanlings were exposed to aluminum in the drinking water starting at 4 weeks of age. In the treated F0 group, no effect on body weight gain was observed; significant decreases ($p < 0.001$) in body weight gain were observed in the treated F1b, F1c, F2a, and F2b groups. No effects on erythrocyte count, hemoglobin levels, or histopathology of the liver, spleen, and kidneys were observed in the F0 or F2 generations. No significant differences were seen in the number of litters or offspring between the exposed and control groups. This study identifies a lowest-observed-adverse-effect-level (LOAEL) of 40.1 mg/kg/day.

Groups of 16 pregnant Swiss-Webster mice were fed a diet containing 25, 500, or 1000 mg Al/kg diet as aluminum lactate throughout gestation and lactation (Donald et al., 1989). Animals fed the 25 mg/kg diet serve as the control groups. After weaning, the young rats were fed the control diet for 2 weeks. The authors calculated that the maternal doses at the beginning of gestation were 5 (control group), 100, and 200 mg/kg/day; at the end of lactation, the doses were 10.5 (control group), 210, and 420 mg/kg/day. No effects on maternal mortality, body weight, food intake, clinical signs, or neurobehavioral performance were observed. Gestation length was statistically ($p < 0.028$) altered in rats in the low and high dose groups. No effects were noted in litter size, sex ratio, birth weight, body length, postnatal mortality, or the ability to perform the righting reflex at birth. In preweanling neurobehavioral development testing, significant alternations ($p < 0.007$) were observed in offspring of rats exposed to the highest concentration. In post-weanling neurobehavioral testing, alterations were observed in the low and high dose groups. Since Muller et al. (1990) determined that developmental toxicity of aluminum occurs during the early part of gestation, the doses at the beginning of gestation are used to define a LOAEL of 100 mg/kg/day.

The most sensitive endpoints of toxicity following oral exposure to aluminum appear to be decreased body weight gain and neurotoxicity. The Ondreicka et al. (1966) study

identified the lowest LOAEL (40.1 mg/kg/day) for decreased body weight gain. This study is inadequate for use as a basis for an RfD due to its small sample size and poor reporting of study details. A LOAEL of 100 mg/kg/day for minimal neurotoxicity in the offspring of mice exposed to aluminum lactate in the diet during gestation and lactation was identified by Donald et al. (1989). The RfD for aluminum and soluble aluminum compounds can be based on this LOAEL of 100 mg Al/kg/day. Application of an uncertainty factor of 100 (3 for use of a minimal LOAEL, 10 for interspecies extrapolation, and 3 to protect sensitive individuals) results in a RfD of 1 mg/kg/day.

Ecological Effects

Aluminum appears to be essential for the growth of some plant species (Kabata-Pendias and Pendias, 1992). Concentrations in the foliage of crop plants are usually less than 300 milligrams (mg) per kilogram (kg) (dry weight) (Bollard, 1983). Higher concentrations of aluminum are usually detected in older rather than younger leaves (Bollard, 1983). Some species of plants, such as the cranberry (*Vaccinium macrocarpon*), are able to tolerate high concentrations of aluminum (Medappa and Dana, 1968, as cited in Foy, 1974). Generally, acid-soil plants (calcifuges) are more tolerant to aluminum than calcareous-soil plants (calcicoles) (Clymo, 1962; Grime and Hodgson, 1969, as cited in Foy, 1974). Because flower color in *Hydrangea macrophylla* is related to aluminum concentrations (blue flowers contain higher concentrations than pink flowers) (Asen et al., 1963, as cited in Foy, 1974), *Hydrandea* can serve as useful indicators of soluble aluminum concentrations in soil.

Difference in the toxicity of aluminum to plants is closely linked to the differential uptake and transport of calcium (Foy, 1974). Interactions of aluminum with potassium, silicon, and organic acids have also been reported (Foy, 1974). According to Foy (1974), aluminum toxicity in plants usually does not occur in soils with pH values above 5.5. Toxicity is, however, common and adverse at soil pH values below 5.0 (Foy, 1974). The addition of nitrogenous fertilizers to soil increases the toxicity of aluminum to plants by displacing exchangeable aluminum into soil solution and lowering soil pH (Foy, 1974). Concentrations of silver in leaf tissue that are excessive or toxic to various plant species with the exclusion of very sensitive and highly tolerant species, range from 5 to 10 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Tissue aluminum concentrations that may result in a 10 percent reduction in crop yield

range from 40 to 280 mg/kg (dry weight) (Macnicol and Beckett, 1985). A soil concentration of 50 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for aluminum phytotoxicity. Signs of aluminum toxicity in plants include overall stunting of growth, the presence of dark green leaves, purpling of stems, death of leaf tips, and coralloid and damaged root systems (Kabata-Pendias and Pendias, 1992).

Aluminum is not an essential element for animal growth and development. Limited data exist on the concentrations and effects of aluminum on wildlife. Most absorbed aluminum is eliminated through the kidney (Kovalchik et al., 1978, as cited in NLM, 1996).

Data do not exist on the effects of aluminum on wild mammals. Laboratory studies have shown inhalation of aluminum dust to induce infections and diseases of the lung (Browning, 1969, as cited in NLM, 1996). A derived chronic no observable adverse effect level (NOAEL) of 0.043 mg/kg per day (/d) has been reported for laboratory rats exposed to aluminum (EPA, 1996). Based on laboratory data on aluminum toxicity in laboratory mice, Opresko et al. (1994) estimated chronic oral NOAELs to be 2.138 mg/kg/d for the white-footed mouse and 0.369 mg/kg/d for the red fox. The drinking water NOAELs for these species were estimated to be 7.127 and 4.374 mg/L, respectively.

There is a greater amount of information on the toxicity of aluminum to birds than on the toxicity of aluminum to mammals. Dietary ingestion of aluminum at concentrations of approximately 1,400 mg/kg produced declines in inorganic phosphorus levels in blood and resulted in the development of severe rickets in chickens (Browning, 1969, as cited in NLM, 1996). No adverse effects were observed in black ducks (*Anas rubripes*) fed diets containing 1,000 mg/kg aluminum as aluminum sulfate over a period of 12 days (Sparling, 1990). Diets with low calcium and phosphorus concentrations adversely affected the response of the ducks to aluminum (Sparling, 1990). Reduced consumption of diets containing 5,000 mg/kg aluminum has also been observed (Sparling, 1990). An estimated acute LD₅₀ (lethal dose that will result in 50 percent deaths in the test population) of 111 mg/kg is reported for exposure of birds to aluminum (Schafer et al., 1983). Based on avian test data, extrapolated NOAELs for

chronic exposure of avian species to aluminum sulfate are 45.17 mg/kg/d for the great blue heron and 57.9 mg/kg/d for the red-tailed hawk (Opresko et al., 1994). Drinking water NOAELs for these species were 1.02 and 1.018 g/L, respectively.

Bioconcentration of aluminum has been reported for several freshwater species. A bioconcentration factor for daphnids exposed to aluminum is 574 (Cowgill and Burns, 1975, as cited in Wren and Stephenson, 1991). Crayfish have been reported to have a bioconcentration factor for aluminum of 1305 (Malley et al., 1987, as cited in Wren and Stephenson, 1991). Forester (1980, as cited in Havlik and Marking, 1987) reported aluminum concentrations in the mollusc *Anodonta grandis* collected from acid-stressed lakes to be as high as 1,500 mg/kg.

Federal Water Quality Criteria exist for the protection of freshwater aquatic life from exposure to aluminum phosphide (EPA, 1996). The values for acute and chronic exposure to aluminum are 750 and 87 micrograms (μg) per liter (L), respectively (EPA, 1996). Ohio Warmwater Habitat Water Quality Criteria do not exist for aluminum. The lowest chronic values of aluminum reported in the literature for fish and *Daphnia* are 3,290 and 1,900 $\mu\text{g/L}$, respectively (Suter and Mabrey, 1994). The test EC_{20} (the concentration that will result in a specified effect on 20 percent of the test population) for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). The value for aluminum is 4,700 $\mu\text{g/L}$ (Suter and Mabrey, 1994). A similar value can be determined for daphnids, which reflects the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC_{20} benchmark for exposure of daphnids to aluminum is 540 $\mu\text{g/L}$ (Suter and Mabrey, 1994).

REFERENCES

- Asen, S., N. W. Stuart, and E. L. Cox, 1963, "Sepal Color of *Hydrangea macrophylla* as Influenced by the Source of Nitrogen Available to Plants," *Proceedings of the American Society of Horticultural Science*, Vol. 82, pp. 504-507.
- Bollard, E. G., 1983, "Involvement of Unusual Elements in Plant Growth and Nutrition," in *Inorganic Plant Nutrition*, A. Lauchli and R. L. Bielecki (eds.), Springer-Verlag, Berlin, Germany, pp. 795-743.
- Browning, E., 1969, *Toxicity of Industrial Metals*, 2nd ed., Appleton-Century-Crofts, New York, New York.
- Clymo, R. S., 1962, "An Experimental Approach to Part of the Calcicole Problem," *Journal of Ecology*, Vol. 50, pp. 707-731.
- Cowgill, U. M., and C. W. Burns, 1975, "Differences in Chemical Composition Between Two Species of *Daphnia* and Some Freshwater Algae Cultured in the Laboratory," *Limnology and Oceanography*, Vol. 20, pp. 1005-1011.
- Donald, J.M., Golum, M.S., Gershwin, M.E., and Keen, C.L., 1989, Neurobehavioral effects in offspring of mice given excess aluminum in diet during gestation and lactation. *Neurotoxicol. Teratol.* 11: 345-351.
- Forester, A. J., 1980, "Monitoring the Bioavailability of Toxic Metals in Acid-Stressed Lakes Using Pelecypod Molluscs (Clams, Mussels)," *Proceedings of the University of Montana Annual Conference on Trace Substances Environmental Health*, Vol. 14, pp. 142-147.
- Foy, C. D., 1974, "Effects of Aluminum on Plant Growth," in *The Plant Root and Its Environment*, E. W. Carson (ed.), University Press of Virginia, Charlottesville, Virginia, pp. 601-642.
- Havlik, M. E., and L. L. Marking, 1987, "Effects of Contaminants on Naiad Mollusks (*Unionidae*): a Review," *U.S. Fish & Wildlife Service Resource Publication*, Vol. 164, Washington, D.C.
- Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.
- Kovalchik, M. T., et al., 1978, *Journal of Laboratory and Clinical Medicine*, Vol. 92, pp. 712.

Medappa, K. C., and M. N. Dana, 1968, "Influence of pH, Calcium, Iron and Aluminum on the Uptake of Radiophosphorus by Cranberry Plants," *Soil Sciences American Proceedings* Vol. 32, pp. 381-383.

Macnicol, R. D., and P. H. T. Beckett, 1985, "Critical Tissue Concentrations of Potentially Toxic Elements," *Plant and Soil*, Vol. 85, pp. 107-129.

Maier, K. J., C. G. Foe, and A. W. Knight, 1993, "Comparative Toxicity of Selenate, Selenite, Seleno-DL-methionine and Seleno-DL-cystine to *Daphnia magna*," *Environmental Toxicology and Chemistry*, Vol. 12, pp. 755-763.

Malley, D. F., P. S. Chang, C. M. Moore, and S. G. Lawrence, 1987, "Changes in Aluminum Content of Tissues of Crayfish Held in the Laboratory and in Experimental Field Enclosures," G. H. Green and K. L. Woodward, eds., *Canadian Technical Report on Fish and Aquatic Sciences*, No. 1480, 330 pp.

Muller, G., Bernuzzi, V., Desor, D., Hutin, M-F., Burnel, D., and Lehr, P. R., 1990, Developmental alterations in offspring of female rats orally intoxicated by aluminum lactate at different gestation periods. *Teratol.* 38: 253-261.

National Library of Medicine (NLM), 1996, "Hazardous Substance Data Bank," produced by Micromedex, Inc.

Ondreicka, R., Ginter, E., and Kortus, J., 1966, Chronic toxicity of aluminum in rats and mice and its effects on phosphorus metabolism. *Brit. J. Industr. Med.* 23: 305-312.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Schafer, E. W., Jr., W. A. Bowles, Jr., and J. Hurlbut, 1983, "The Acute Toxicity, Repellency, and Hazard Potential of 998 Chemicals to One or More Species of Wild and Domestic Birds," *Archives of Environmental Contamination and Toxicology*, Vol. 12, pp. 355-382.

Sparling, D. W., 1990, "Conditioned Aversion of Aluminum Sulfate in Black Ducks," *Environmental Toxicology and Chemistry*, Vol. 9, pp. 479-483.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Touam, M., Marinez, F., Lacour, B., Bourdon, R., Zingraff, J., DiGiulio, S., and Druke, T., 1983, Aluminum-induced, reversible microcytic anemia in chronic renal failure: Clinical and experimental studies. *Clin. Nephrol.* 19: 295-298.

U.S. Environmental Protection Agency (EPA), 1987, Health Effects Assessment for Aluminum. Office of Health and Environmental Assessment, Cincinnati, OH.

U.S. Environmental Protection Agency (EPA), 1996, "Integrated Risk Information System," on-line database, maintained by the U.S. Environmental Protection Agency.

Wilhelm, M., Jager, D.E., and Ohnesorge, F.K., 1990, Aluminum toxicokinetics. *Pharmacol. Toxicol.* 60: 4-9.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Wren, C. D., and G. L. Stephenson, 1991, "The Effect of Acidification on the Accumulation and Toxicity of Metals to Freshwater Invertebrates," *Environmental Pollution*, Vol. 7, pp. 205-241.

Yokel, R.A., and McNamara, P.J., 1988, Influence of renal impairment, chemical form and serum protein binding on intravenous and oral aluminum kinetics in the rabbit. *Toxicol. Appl. Pharmacol.* 95: 32-43.

ANTIMONY

Human Health Effects

Antimony exists in the tri- and pentavalent states (Budavari, 1989). The pharmacokinetics of antimony appear to be strongly valence- and species-dependent. Elinder and Friberg (1986) estimated GI absorption to be at least 15 percent in mice given a single oral dose of labeled trivalent antimony potassium tartrate. This estimate was based on the recovery of labeled antimony in urine and tissues. Actual absorption may have been considerably higher, because GI excretion starts immediately after absorption following an oral dose. The 15 percent absorption efficiency is considered sufficiently conservative and well documented for use in estimating a dermal RfD from the oral RfD.

Although quantitative data were not provided, Elinder and Friberg (1986) stated that the pulmonary absorption of inhaled trivalent antimony is substantial.

Patterns of tissue distribution of absorbed antimony appear to be largely species-dependent. In humans injected with labeled sodium antimony dimercaptosuccinate, highest amounts of antimony are located in the liver, thyroid, and heart (Elinder and Friberg, 1986). Smelter workers exposed to inhaled antimony compounds retain antimony in their lungs for several years. Single or repeated injections of trivalent or pentavalent antimony in monkeys, dogs, and mice result in highest levels in the kidney, liver, and thyroid. Rats appear to retain higher levels in the blood than do other laboratory animals. In rats, trivalent antimony is retained principally in the erythrocytes (at least 95 percent), but pentavalent antimony is retained principally in the plasma (about 90 percent).

In humans, pentavalent antimony appears to be cleared from the body more efficiently than trivalent antimony (Elinder and Friberg, 1986). Urinary excretion predominates over fecal excretion for both penta- and trivalent antimony, but particularly for pentavalent antimony. In rats and hamsters, urinary excretion predominates for pentavalent antimony and fecal excretion predominates for trivalent antimony.

Chronic effects from occupational exposure include irritation of the respiratory tract, pneumoconiosis, pustular eruptions of the skin called "antimony spots," allergic contact dermatitis, and cardiac effects, including abnormalities of the electrocardiograph (ECG) and myocardial changes (Elinder and Friberg, 1986). Cardiac effects were also observed in rats and rabbits exposed by inhalation for 6 weeks and in animals (dogs, and possibly other species) treated by intravenous injection. Inhalation RfC or RfD values were not located. The heart, respiratory tract, and skin are the principal target organs for antimony.

Data were not located regarding the carcinogenicity of antimony to humans. Antimony fed to rats did not produce an excess of tumors (Goyer, 1991), but a high frequency of lung tumors was observed in rats exposed by inhalation to antimony trioxide for 1 year (Elinder and Friberg, 1986). The EPA (1995) classifies antimony a cancer weight-of-evidence Group D substance. Quantitative cancer risks are not estimated for Group D substances.

Ecological Effects

Aquatic organisms do not bioaccumulate antimony to an appreciable degree. Antimony uptake by plants in contaminated soils has been reported as minimal, and is probably restricted to the soluble or exchangeable species of antimony (Ainsworth, 1988).

Effects from antimony exposure on benthic community composition have been detected at levels between 3.2 and 150 mg/kg (Long and Morgan, 1990). Data on antimony suggest an effects range-low (ER-L) of 2 mg/kg, and an effects range-medium (ER-M) of 25 mg/kg.

REFERENCES

Ainsworth, N., 1988, Distribution and biological effects of antimony in contaminated grassland, Dissertation.

Budavari, S., ed., 1989, The Merck Index, An Encyclopedia of Chemicals, Drugs, and Biologicals., Eleventh Edition, Merck and Co., Inc., Rahway, NJ.

Elinder, C. G. and L. Friberg, 1986, Antimony, in Handbook on the Toxicology of Metals, L. Friberg, G. F. Nordberg, and V. B. Vouk, eds., 2nd ed., Vol. 2: Specific Metals, Elsevier Science Publishers B.V., New York, NY, pp. 26-42.

Goyer, R. A., 1991, Toxic Effects of Metals, Casarett and Doull's Toxicology, the Basic Science of Poisons, M. O. Amdur, J. Doull, and C. D. Klaassen, eds., 4th ed., Pergamon Press, New York.

Long, E. R. And L. G. Morgan, 1990, The Potential for Biological Effects of Sediment-sorbed Contaminants Tested in the National Status and Trends Program, National Oceanic and Atmospheric Administration Technical Memorandum NOSOMA 52, NOAA, Seattle, Washington.

U.S. Environmental Protection Agency (EPA), 1995, Health Effects Assessment Summary Tables. Annual Update FY 1995, including Supplements. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

ARSENIC

Human Health Effects

Most arsenic enters water supplies either from natural deposits in the earth or from industrial and agricultural pollution. Arsenic is a natural element of the earth's crust. It is used in industry and agriculture, and for other purposes. It also is a byproduct of copper smelting, mining and coal burning. U.S. industries release thousands of pounds of arsenic into the environment every year.

Arsenic occurs in compounds in the trivalent and pentavalent forms (Budavari, 1989). The extent of the GI absorption of arsenic depends on the particular arsenic compound ingested. Several studies with humans and laboratory animals indicate that the GI absorption of dissolved trivalent or pentavalent arsenic exceeds 90 percent (Ishinishi, et al., 1986). Hamsters appear to have somewhat lower (50 to 75 percent) GI absorption of soluble arsenic compounds (ATSDR, 1990). Organic arsenic compounds, such as occur in seafoods, are also readily absorbed (70 to 99.7 percent). The GI absorption of less soluble compounds (e.g., arsenic trioxide) is determined by particle size and pH of the gastric juice. An estimate of 80 percent GI absorption efficiency is considered to be sufficiently conservative and well documented for use in estimating a dermal RfD and cancer slope factor from the respective oral values.

The extent of absorption of arsenic from the lungs depends on the solubility of the inhaled compound and particle size (ATSDR, 1990; Ishinishi, et al., 1986). In a study with arsenite in cigarettes and with arsenic aerosols in lung cancer patients, deposition was estimated at approximately 40 percent, and 75 to 85 percent of the deposited arsenic was absorbed from the lungs within 4 days.

The occurrence of systemic toxic effects following dermal exposure to arsenic acid or arsenic trichloride (Ishinishi, et al., 1986) indicates qualitatively that dermal absorption of some arsenic compounds occurs.

In most animals, all but a small fraction of systemic arsenic is rapidly cleared from the blood and other tissues (ATSDR, 1990). Residual arsenic is located in tissues (liver, kidney, spleen, heart, skin, hair, epithelium of the upper GI tract) containing a high

concentration of sulfhydryl groups, to which arsenic preferentially binds (Ishinishi, et al., 1986). In rats, more than in the other laboratory animals and in humans, arsenic binds to the erythrocytes with high affinity and clearance from the blood is slow (ATSDR, 1990).

Arsenic is extensively metabolized, principally in the liver, in humans and animals (ATSDR, 1990). Metabolism involves methylation of trivalent arsenic (arsenite) to dimethylarsinic acid, or, to a lesser extent, to monomethylarsonic acid. Both methylation products, as well as inorganic arsenic, are excreted principally and rapidly through the urine.

A lethal dose of arsenic trioxide in humans is 70 to 180 mg (approximately 50 to 140 mg arsenic) (Ishinishi, et al., 1986). Acute oral exposure of humans to high doses of arsenic produces liver swelling, skin lesions, disturbed heart function, and neurological effects. The only noncancer effects in humans clearly attributable to chronic oral exposure to arsenic are dermal hyperpigmentation and keratosis, as revealed by studies of several hundred Chinese exposed to naturally occurring arsenic in well water (EPA, 1996). Similar effects were observed in persons exposed to high levels of arsenic in water in Utah and the northern part of Mexico. Occupational (predominantly inhalation) exposure is also associated with neurological deficits, anemia, and cardiovascular effects (Ishinishi, et al., 1986), but concomitant exposure to other chemicals cannot be ruled out. The principal target organ for arsenic appears to be the skin. The nervous system and cardiovascular systems appear to be significant target organs for acute exposure to higher levels. Inorganic arsenic may be an essential nutrient, exerting beneficial effects on growth, health, and feed conversion efficiency (Underwood, 1977).

Inorganic arsenic is clearly a carcinogen in humans. Inhalation exposure is associated with increased risk of lung cancer in persons employed as smelter workers, in arsenical pesticide applicators, and in a population residing near a pesticide manufacturing plant (EPA, 1996). Oral exposure to high levels in wellwater is associated with increased risk of skin cancer (Tseng, 1977; EPA 1996). Extensive animal testing with various forms of arsenic given by many routes of exposure to several species; however, has not demonstrated the carcinogenicity of arsenic (International Agency for Research on

Cancer [IARC], 1987). EPA (1996) classifies inorganic arsenic in cancer weight-of-evidence Group A (human carcinogen) based on the incidence of skin cancer in the Tseng (1977) study. EPA (1996) notes that the uncertainties associated with the oral unit risk are considerably less than those for most carcinogens.

Ecological Effects

The National Academy of Sciences (1977) reports background arsenic concentrations in terrestrial plants as ranging from 0.01 to 5 mg/kg (dry weight). On a fresh-weight basis, concentrations in terrestrial flora are usually less than 1 mg/kg (Eisler, 1988). Plants growing near smelters generally contain higher concentrations than those grown in uncontaminated areas. Clover tends to contain higher concentrations of arsenic than grasses collected from the same area (Jones and Hatch, 1945). Natural variations among plants, plant species, available soil arsenic, and growing conditions are all responsible for differences in reported arsenic concentrations in plants. Generally, roots of a plant contain higher concentrations of arsenic than leaves. Mushrooms are relatively good accumulators of arsenic. The toxicity of arsenic to plants may differ due to different soil conditions. Tissue concentrations of iron, aluminum, organic matter, phosphate, and soil pH may have an effect on the availability of arsenic to the plant. Various chemical forms of arsenic have different phytotoxicities. In general, arsenates are less toxic to plants than arsenites. Concentrations of arsenic in leaf tissue that are excessive or toxic to various plant species, with the exclusion of very sensitive and highly tolerant species, range from 5 to 20 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Concentrations from 1 to 20 mg/kg (dry weight) are expected to result in a 10 percent loss in crop yield (Macnicol and Beckett, 1985). A soil concentration of 10 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for phytotoxicity in soils. In the past, organoarsenical herbicides were used to inhibit the growth of weedy plants. General symptoms of arsenic toxicity in plants include the presence of red-brown necrotic spots on old leaves, yellowing or browning of roots, depressed tillering, wilting of new leaves, and root discoloration (Kabata-Pendias and Pendias, 1992).

Background concentrations of arsenic in terrestrial biota are usually less than 1 mg/kg (wet weight) (Eisler, 1988). Elfving et al. (1979) collected several species of small mammals from an uncontaminated site in New York and found whole-body arsenic

concentrations to range from the limit of detection to 0.8 mg/kg (dry weight). Arsenic tissue concentrations in mammals are highest in kidney and liver tissues (Gregus and Klaassen, 1986). Arsenic is not biomagnified through food chains.

In general, inorganic arsenic compounds are more toxic than organic arsenic compounds, and trivalent forms of arsenic are more toxic than pentavalent forms. Reported cases of arsenic poisoning in wildlife species are either due to acute or subacute exposures. Incidents of chronic arsenic poisoning are rarely encountered in wildlife (Eisler, 1988).

Adverse effects of arsenic in mammals have been noted at a single oral dose of 2.5 to 33 mg/kg body weight (Eisler, 1988). Chronic oral doses of arsenic of 1 to 10 mg/kg body weight has produced adverse effects in laboratory mammals, as have diets containing arsenic at concentrations of 5 to 50 mg/kg (Eisler, 1988). The oral LD₅₀ for laboratory rats exposed to arsenic is 763 mg/kg (RTECS, 1996). Based on laboratory data on arsenic toxicity (as arsenite) in laboratory mice, Opresko et al. (1994) estimated chronic oral NOAELs to be less than 0.140 mg/kg/d for the white-footed mouse and 0.024 mg/kg/d for the red fox. The NOAELs for arsenic consumed in drinking water have been estimated to be 0.465 and 0.286 mg/L, respectively (Opresko et al., 1994). This assumes no ingestion of arsenic in the diet. Arsenic metabolism and toxicity vary greatly between species. Arsenic concentrations of greater than 10 mg/kg (wet weight) in tissue are usually indicative of arsenic poisoning (Goede, 1985). Concentrations of arsenic in hair of greater than 5 mg/kg have been reported in cases of chronic poisoning and 10 to 30 mg/kg in cases of acute poisoning (Buck, 1978). Detoxification and excretion of arsenic are relatively rapid processes, making the probability of chronic arsenic poisoning from the continuous ingestion of small amounts of arsenic a rare event (Eisler, 1988). General signs of arsenic poisoning include intense abdominal pain, staggering gait, extreme weakness, trembling, salivation, vomiting, diarrhea, prostration, collapse, and death (Eisler, 1988). Arsenic poisoning in mammals may involve the respiratory, gastrointestinal, cardiovascular, and hematopoietic systems. Adverse effects of arsenic toxicity can be reversible; however, they can also lead to cancer and death. Teratogenic and mutagenic effects can also occur as a result of exposure to arsenic.

Studies with mallards have shown the acute oral LD₅₀ for sodium arsenate to be 323 mg/kg body weight (NAS, 1977). The LD₅₀ for California quail exposed to a single oral dose of sodium arsenite is 47.6 mg/kg body weight (Hudson et al., 1984). A dose of 500 mg sodium arsenate/kg diet is fatal to 50 percent of the mallards in the test group within a period of 32 days (NAS, 1977). Based on avian toxicity data for mallards exposed to sodium arsenite (5.135 mg/kg/d), chronic oral NOAELs for the great blue heron and red-tailed hawk have been estimated by Opresko et al. (1994) as 3.85 and 4.94 mg/kg/d, respectively. Drinking water NOAELs for these species were estimated to be 86.9 mg/L for the mallard, 87.0 mg/L for the heron, and 86.9 mg/L for the hawk. Birds poisoned by inorganic trivalent arsenite usually show signs of muscular incoordination, such as slowness, jerkiness, fluffed feather, drooped eyelids, huddled position, immobility, seizures, and unkempt appearance (Eisler, 1988).

Several factors can modify the toxicity of arsenic in freshwater environments. These include abiotic factors such as temperature, pH, organic content, phosphate concentration, suspended solids, the speciation of arsenic, and the concentration of other inorganic elements in the water column (Eisler, 1988). Inorganic forms of arsenic are more toxic to aquatic biota than organic forms of arsenic. Early life stages appear to be the most sensitive to arsenic concentrations. Although arsenic is bioconcentrated by aquatic organisms, there is no evidence to support biomagnification in aquatic food chains (Eisler, 1988). The bioconcentration factor for arsenic in fish and invertebrates is approximately 17 (EPA, 1980, as cited in Eisler, 1988).

The United States Environmental Protection Agency's (EPA) National Ambient Water Quality Criteria for arsenic in freshwater is 360 µg/L for acute exposure and 190 µg/L for chronic exposure of aquatic life to arsenic III (based on a water hardness of 100 mg/L) (EPA, 1996). The Ohio Warmwater Habitat Water Quality Criteria has been set at 0.19 mg/L for arsenic. The lowest chronic values of arsenic III reported in the literature for fish and *Daphnia* are 2,962 and 914.1 µg/L, respectively (Suter and Mabrey, 1994). The test EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter

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and Mabrey, 1994). The value for arsenic III is 2,130 $\mu\text{g/L}$ for fish and 633 $\mu\text{g/L}$ for daphnids (Suter and Mabrey, 1994).

REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR), 1990, Toxicological Profile for Arsenic, U.S. Public Health Service, Atlanta, GA.
- Buck, W. B., 1978, "Toxicity of Inorganic and Aliphatic Organic Arsenicals," in *Toxicity of Heavy Metals in the Environment, Part I*, F. W. Oehme, ed., Dekker, New York, New York, pp. 357-374.
- Budavari, S., ed., 1989, The Merck Index, An Encyclopedia of Chemicals, Drugs, and Biologicals., Eleventh Edition, Merck and Co., Inc., Rahway, NJ.
- Eisler, R., 1988, "Arsenic Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review," U.S. Fish and Wildlife Service Contaminant Hazard Review, *Report No. 12*, U.S. Department of the Interior, Washington, D.C.
- Elfving, D. C., R. A. Stehn, I. S. Pakkala, and D. J. Lisk, 1979, "Arsenic Content of Small Mammals Indigenous to Old Orchard Soils," *Bulletin of Environmental Contamination and Toxicology*, Vol. 21, pp. 62-64.
- Goede, A. A., 1985, "Mercury, Selenium, Arsenic and Zinc in Waders from the Dutch Wadden Sea," *Environmental Pollution*, Vol. 37A, pp. 287-309.
- Gregus, Z., and C. D. Klaassen, 1986, "Disposition of Metals in Rats: A Comparative Study of Fecal, Urinary, and Biliary Excretion and Tissue Distribution of Eighteen Metals," *Toxicology and Applied Pharmacology*, Vol. 85, pp. 24-38.
- Hudson, R. H., R. K. Tucker, and M. Haegele, 1984, "Handbook of Toxicity of Pesticides to Wildlife," *U.S. Fish Wildlife Service Resource Publication*, Vol. 153, U.S. Fish and Wildlife Service, Washington, D.C., 90 pp.
- International Agency for Research on Cancer (IARC), 1987, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Vol. 1 to 42. Preamble. Supplement 7. World Health Organization: Lyon, France, pp. 17-34.
- Ishinishi, N., K. Tsuchiya, M. Vahter, and B. A. Fowler, 1986, Arsenic, in Handbook on the Toxicology of Metals, L. Friberg, G. F. Nordberg, and V. B. Vouk, eds., 2nd ed., Vol. 2: Specific Metals, Elsevier Science Publishers B.V., New York, NY, pp. 43-83.
- Jones, J. H., and M. B. Hatch, 1945, "Spray Residues and Crop Assimilation of Arsenic and Lead," *Soil Sciences*, Vol. 60, pp. 277-288.

Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.

Macnicol, R. D., and P. H. T. Beckett, 1985, "Critical Tissue Concentrations of Potentially Toxic Elements," *Plant and Soil*, Vol. 85, pp. 107-129.

Maier, K. J., C. G. Foe, and A. W. Knight, 1993, "Comparative Toxicity of Selenate, Selenite, Seleno-DL-methionine and Seleno-DL-cystine to *Daphnia magna*," *Environmental Toxicology and Chemistry*, Vol. 12, pp. 755-763.

National Academy of Sciences (NAS), 1977, "Arsenic," Medical and Biological Effects of Environmental Pollutants Series, National Academy of Sciences, Washington, D.C., 332 pp.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Registry of Toxic Effects of Chemical Substances (RTECS), 1996, produced by Micromedex.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Tseng, W. P., 1977, Effects and Dose-Response Relationships of Skin Cancer and Blackfoot Disease with Arsenic, *Environmental Health Perspectives*, Vol. 19, pp. 109-119.

Underwood, E. J., 1977, *Trace Elements in Human and Animal Nutrition*, Fourth Edition. New York: Academic Press.

U.S. Environmental Protection Agency (EPA), 1996, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1980, "Ambient Water Quality Criteria for Arsenic," *Report 440/5-80-021*, U.S. Environmental Protection Agency, Washington, D.C.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

BARIUM

Human Health Effects

Barium is a naturally occurring alkaline earth metal that comprises approximately 0.04 percent of the earth's crust (Reeves, 1986). Acute oral toxicity was manifested by GI upset, altered cardiac performance, and transient hypertension, convulsions, and muscular paralysis. Repeated oral exposures were associated with hypertension. Occupational exposure to insoluble barium sulfate induced benign pneumoconiosis (ACGIH, 1991). The EPA (1996a) presented a verified chronic oral RfD of 0.07 mg/kg/day, based on an NOAEL of 0.21 mg/kg/day in a ten-week study in humans exposed to barium in drinking water and an uncertainty factor of 3. The EPA (1996a) presented the same value as a provisional RfD for subchronic oral exposure. A provisional chronic inhalation Reference Concentration (RfC) of 0.0005 mg/m³ and a provisional subchronic inhalation RfC of 0.005 were based on an NOEL for fetotoxicity in a four-month intermittent-exposure inhalation study in rats (EPA, 1996a). Uncertainty factors of 1000 and 100 were used for the chronic and subchronic RfC values, respectively. The chronic and subchronic inhalation RfD values are equivalent to 0.0001 and 0.002 mg/kg/day, assuming a human inhalation rate of 20 m³/day and body weight of 70 kg. Barium is principally a muscle toxin. Its targets are the GI system, skeletal muscle, the cardiovascular system, and the fetus.

The EPA (1995) classifies barium as a cancer weight-of-evidence Group D substance (not classifiable as to carcinogenicity in humans). Cancer risk is not estimated for Group D substances.

Ecological Effects

Although commonly detected in plants, barium is not an essential element for plant growth. Background concentrations of barium in various food and feed plants are reported to range from 1 to 198 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Concentrations are often highest in the leaves of cereals and legumes and lowest in grains and fruits (Kabata-Pendias and Pendias, 1992). Barium concentrations in excess of 10,000 mg/kg (dry weight) have been measured in some trees, shrubs, and in Brazil nuts (Shacklette et al., 1978, as cited in Kabata-Pendias and Pendias, 1992). The availability of barium to plants is greatly influenced by the pH of the soil, with barium

more available under acidic soil conditions (Kabata-Pendias and Pendias, 1992). In addition, antagonistic relationships have been observed between barium and calcium, magnesium, and sulfur in soil and within plants (Kabata-Pendias and Pendias, 1992).

The concentration of barium in leaf tissue that has been reported as excessive or toxic to various plant species, with the exclusion of very sensitive and highly tolerant species, is 500 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 500 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for barium phytotoxicity.

There is some controversy over whether barium is an essential element for animals. Underwood (1971, as cited in Hammond and Beliles, 1980) observed improper growth in rats and guinea pigs reared on barium-free diets. Barium acts much like calcium and, as such, is found to accumulate in bone (Luckey et al., 1975). Background concentrations of barium in wildlife were not found in the literature.

Soluble forms of barium have been found to be much more toxic than insoluble forms following oral exposure (Hammond and Beliles, 1980). Insoluble forms of barium, however, can be hazardous if inhaled (Hammond and Beliles, 1980). Based on laboratory rat toxicity data for barium chloride (estimated NOAEL of 5.06 mg/kg/d), extrapolated NOAELs for chronic oral exposure of various mammalian wildlife species to barium range from 1.015 mg/kg/d to 19.32 mg/kg/d (Opresko et al., 1994). Calculated chronic drinking-water NOAELs for wildlife are 45.2 mg/L for the white-footed mouse, 37.5 mg/L for the cottontail rabbit, and 27.7 mg/L for the red fox (Opresko et al., 1994). Similar values have been estimated by Opresko et al. (1994) for birds orally exposed to barium hydroxide. Based on a NOAEL of 20.86 mg/kg/d for a chicken, oral NOAELs for the great blue heron and red-tailed hawk were calculated to be 7.8 and 10.0 mg/kg/d, respectively. The drinking water NOAEL for these birds was 176 mg/L.

Background concentrations of barium in freshwater aquatic organisms could not be located in the literature. The USEPA benchmark for barium is 0.0039 (EPA, 1996b). No Ohio Warmwater Habitat Water Quality Criteria exist for the protection of freshwater biota from elevated barium concentrations. Suter and Mabrey (1994),

however, have estimated acute and chronic advisory levels for barium to be 69.1 and 3.8 $\mu\text{g/L}$, respectively. Most waters are believed to contain sufficient concentrations of sulfate or carbonate to precipitate the barium in the water column as an insoluble nontoxic compound (EPA, 1986).

REFERENCES

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed., ACGIH, Cincinnati, OH.

Hammond, P. B., and R. P. Beliles, 1980, "Metals," in J. Doull, C. D. Klaassen, and M. O. Amdur (eds.), *Casarett and Doull's Toxicology: The Basic Science of Poisons*, 2nd ed., Macmillan Publishing Co., Inc., New York, New York, pp. 409-4671.

Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.

Luckey, T. D., B. Venugopal, and D. Hutchenson, 1975, *Heavy Metal Toxicity, Safety and Hormology*, Georg Thieme Publishers, Stuttgart, Germany.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Reeves, A. L., 1986, Barium, *Handbook on the Toxicology of Metals*, Friberg, L., G. F. Nordberg, and V. B. Vouk, eds., Vol. II, Elsevier Science Publishers B. V., New York, pp. 84-94.

Shacklette, H. T., J. A. Erdman, and T. F. Harms, 1978, "Trace Elements in Plant Foodstuffs," in *Toxicity of Heavy Metals in the Environment, Part I*, F. W. Oehme (ed.), Marcel Dekker, New York, New York.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Underwood, W. J., 1971, *Trace Elements in Human and Animal Nutrition*, Academic Press, Inc., New York, New York.

U.S. Environmental Protection Agency (EPA), 1995. Health Effects Assessment Summary Tables. Annual Update FY 1995, including Supplements. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996a, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996b, Ecotox Thresholds, EPA 540/F-95/038.

U.S. Environmental Protection Agency (EPA), 1986, "Quality Criteria for Water, 1986," *EPA 40/5-86-001*, Office of Water Regulations and Standards, U.S. Environmental Protection Agency, Washington, DC.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

BENZENE

Human Health Effects

Benzene, the simplest of the aromatic hydrocarbons, is a by-product of the petroleum and coke oven industries. It has been widely used in the chemical and drug industries and as a solvent for paints, resins, lacquers, and plastics. Benzene is part of gasoline and a by-product of partial combustion such as with vehicle exhausts and cigarette smoke. Its toxicity has been reviewed by EPA (1980, 1984, and 1989), Mehlman (1983), Sandmeyer (1981), ATSDR (1987), and NLM (1996).

The most significant environmental fate process for benzene is volatilization for either water or soil into air. Within the air medium, the dominant fate process is oxidation via the hydroxyl radical. The half-life for benzene biodegradation depends upon temperature, substrate concentration, oxygen availability and organism acclimation. Half-lives for benzene biodegradation in ground water range from 10 days to 2 years. The EPA has estimated a half-life in soil for benzene of 5 to 16 days based upon the unacclimated aerobic biodegradation half-life.

Exposure to benzene can occur by ingestion, inhalation, and by dermal absorption. Due to its volatility, the major route of entry of benzene is by vapor inhalation. Respiratory uptake is approximately 50 percent. Dermal absorption in humans, which is approximately 1 percent, is a function of concentration and contact time with the skin. Much of the absorbed benzene is deposited in the body fat and fatty tissues. Small amounts of benzene are exhaled unchanged, but most is metabolized in the liver followed by excretion by the kidney.

Following an acute exposure, benzene toxicity appears to be due primarily to its effect on the central nervous system. Recovery from an acute exposure depends on the initial severity of the exposure. Symptoms may last for two to three weeks. The important toxic manifestations related to chronic, low dose benzene exposure are effects to the blood forming tissues, although central nervous system effects and gastrointestinal effects are also seen. There is evidence of a progression in severity of effects in these hematopoietic tissues, ranging from pancytopenia to aplastic anemia and possibly to myelogenous leukemia. Benzene is classified by the EPA as a Group A, known human,

carcinogen. Immunologic effects have also been related to exposure to benzene. In humans, depression of antibody producing cells (B-cells) and cells that mediate cellular immune function (T-cells) has been reported. In animal experiments, benzene has been shown to be embryotoxic and fetotoxic, although these effects were observed at doses that were also maternally toxic. Studies on the reproductive effects of benzene have been inconclusive.

Benzene toxicity may be altered by simultaneous exposure to some other solvents (e.g., xylene or toluene). These other aromatic solvents are metabolized by many of the same hepatic enzyme systems that metabolize benzene. Since the metabolites of benzene are suspected of inducing bone marrow toxicity, inhibition of benzene metabolism by toluene may increase the toxicity of benzene.

Only limited aquatic studies are available for benzene. In acute studies, it is lethal to various species at doses ranging for 5,000 µg/L to over 200,000 µg/L. No useful chronic studies were located.

Ecological Effects

Both natural and artificial sources of benzene exist. Natural sources include volcanoes, crude oil, forest fires, and volatile plant components (IARC, 1982, and Graedel, 1978, as cited in NLM, 1996). Artificial sources are related to benzene's use in gasoline and as a solvent (NLM, 1996). Benzene at the surface of soils is expected to rapidly volatilize (NLM, 1996). The compound will be mobile in soil and is expected to leach into groundwater (NLM, 1996). Biodegradation of benzene in soil may occur (NLM, 1996). Benzene in aquatic environments is expected to volatilize rapidly from the water surface (NLM, 1996). Adsorption to sediment, hydrolysis, and bioconcentration are not expected to be significant (NLM, 1996). There is limited evidence that supports biodegradation of benzene in freshwater environments (NLM, 1996).

Information on the concentration of benzene in wild flora or phytotoxicity data on benzene could not be found in the literature.

Benzene is readily absorbed by the lung and the GI tract and accumulates mainly in fat, with lower concentrations in bone marrow, brain, heart, kidney, lung, and muscle

(IARC, 1974; Goodman and Gilman, 1970; and Clayton and Clayton, 1982, as cited in NLM, 1996). Benzene is eliminated in its original form in expired air following exposure to the compound (IARC, 1974, as cited in NLM, 1996).

Specific data on the toxicity of benzene to wildlife do not exist. Toxicity to benzene is often associated with adverse effects on the heart (Clayton and Clayton, 1982, as cited in NLM, 1996) and nervous system (Patty, 1963, as cited in NLM, 1996). Toxicity to benzene is largely attributed to one or more metabolites of benzene (IARC, 1982, as cited in NLM, 1996). Benzene is considered a potent bone marrow toxin in animals (Lewis et al., 1988, as cited in NLM, 1996). Oral LD₅₀ value for rats and mice exposed to benzene are 930 and 4,700 mg/kg, respectively (RTECS, 1996). Lethal concentrations that will result in death of 50 percent of the test population (LC₅₀) following exposure via inhalation of benzene are 10,000 ppm over a seven hour period (ppm/7 hr) for the rat and 9,980 ppm for the mouse (RTECS, 1996). Dermal LD₅₀ value for rabbits and guinea pigs exposed to benzene are greater than 18.263 g/kg for both species (RTECS, 1996). The lowest published lethal oral dose of benzene for dogs is 2 g/kg (RTECS, 1996). The lowest published lethal inhalation concentration of benzene to rabbits, dogs, and cats are 45,000 ppm/30 minutes (min); 146,000 mg/m³; and 170,000 mg/m³, respectively (RTECS, 1996). Benzene can be phytotoxic at inhalation concentrations as low as 50 ppm/24 hrs (RTECS, 1996). Teratogenic effects have been reported in the offspring of pregnant mice exposed to benzene at an inhalation concentration as low as 20 ppm/6 hr during the sixth to fifteenth day of pregnancy (RTECS, 1996). Benzene has been shown to be genotoxic, mutagenic, and carcinogenic to rodents (RTECS, 1996).

Wildlife NOAELs for benzene based on extrapolations from laboratory rat studies are 29.2 mg/kg for the white-footed mouse, 7.8 mg/kg/d for the cottontail rabbit, and 5.05 mg/kg/d for the red fox (Opresko et al., 1994). Calculated chronic drinking-water NOAELs for mammalian wildlife exposed to benzene in drinking water only range from 33.4 to 260 mg/L (Opresko et al., 1994).

Data on concentrations of benzene in aquatic organisms could not be found in the literature. A bioconcentration factor of 4.3 has, however, been reported for goldfish exposed to benzene (Ogata et al., 1984, as cited in NLM, 1996).

Data on the toxicity benzene to freshwater biota are limited primarily to fish studies. Ninety-six hour LC_{50} values for bass (*Morone saxatilis*), crab larvae (*Cancer magister*), and grass shrimp (*Palaemonetes pugio*) exposed to benzene are 5.8 to 10.9 mg/L, 220 mg/L, 1,108 mg/L, and 27 mg/L, respectively (Verschueren, 1983, as cited in NLM, 1996). Federal Water Quality Criteria does not exist for the protection of freshwater aquatic life from exposure to benzene (EPA, 1996). The lowest effect level listed by EPA (1996) for acute exposure is 5,300 $\mu\text{g/L}$. Suter and Mabrey (1994), however, recommend acute and chronic advisory values of 815 and 45.5 $\mu\text{g/L}$, respectively for the protection of freshwater biota. Lowest chronic toxicity values of benzene to fish and daphnids are 8,250 $\mu\text{g/L}$ and greater than 98,000 $\mu\text{g/L}$, respectively (Suter and Mabrey, 1994). The test EC_{20} for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than a 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early lifestage test (Suter and Mabrey, 1994). The value for benzene is 21 $\mu\text{g/L}$ (Suter and Mabrey, 1994).

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR), 1987, Draft Toxicological Profile for Benzene, U.S. Public Health Service, Atlanta, GA.

Clayton, G. D. and F. E. Clayton (eds.), 1982, *Patty's Industrial Hygiene and Toxicology*, Vols. 2A, 2B, 2C, 3rd ed., John Wiley and Sons, New York, New York.

Goodman L. S., and A. Gilman, 1970, *The Pharmacological Basis of Therapeutics*.

Graedel, T. E., 1978, *Chemical Compounds in Atoms*, Academic Press, New York, New York.

International Agency for Research on Cancer (IARC), 1972 through 1986, *Monograph on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, World Health Organization, International Agency for Research on Cancer, Geneva, Switzerland.

Lewis, J. G., et al., 1988, *Toxicology Applied Pharmacology*, Vol. 92, pp. 246-254.

Mehlman, M.A. (Editor), 1983, Carcinogenicity and Toxicity for Benzene, in *Advances in Modern Environmental Toxicology*, Volume IV.

National Library of Medicine (NLM), 1996, Hazardous Substances Databank File, Toxicology Information Network (TOXNET).

Ogata, M., et al., 1984, *Bulletin of Environmental Contamination and Toxicology*, Vol. 33, pp. 561-567.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Patty, F. (ed.), 1963, *Industrial Hygiene and Toxicology: Volume II: Toxicology*, 2nd ed., Interscience Publishers, New York, New York.

Registry of Toxic Effects of Chemical Substances (RTECS), 1996, produced by Micromedex.

Sandmeyer, E.E., 1981, Aromatic Hydrocarbons, in *Patty's Industrial Hygiene and Toxicology*, George D. Clayton and Florence E. Clayton (Editors), Third Revised Edition, Volume 2B. New York, John Wiley & Sons.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

U.S. Environmental Protection Agency (EPA), 1996, "Integrated Risk Information System," on-line database, maintained by the U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency (EPA), 1989, Updated Health Effects Assessment for Benzene. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-89/086.

U.S. Environmental Protection Agency (EPA), 1984, Health Effects Assessment for Benzene. Office of Health and Environmental Assessment, Washington, DC. ECAO-CIN-H016.

U.S. Environmental Protection Agency (EPA), 1980, An Exposure and Risk Assessment for Benzene. Office of Health and Environmental Assessment, Washington, DC. EPA/440/4-85/006.

Verschueren, K., 1983, *Handbook of Environmental Data of Organic Chemicals*, 2nd ed., Van Nostrand Reinhold Co., New York, New York, 1080 pp.

BIS(2-ETHYLHEXYL)PHTHALATE; (DI[2-ETHYLHEXYL]PHTHALATE)

Human Health Effects

The acute oral toxicity of bis(2-ethylhexyl)phthalate is very low; oral LD_{50/30} (lethal dose to 50 percent of population within 30 days without medical treatment) values in rats and mice were 33,800 and 26,300 mg/kg, respectively (ACGIH, 1991). Repeated high-dose oral exposures were associated with decreased growth, altered organ weights, testicular degeneration, and developmental effects. The EPA, (1996a) presented a verified chronic oral RfD of 0.02 mg/kg/day based on an LOAEL for increased relative liver weight in guinea pigs and an uncertainty factor of 1000. The EPA (1995) adopted the chronic oral RfD as the provisional subchronic oral RfD. The principal target organs for the toxicity of bis(2-ethylhexyl)phthalate are the liver and testis.

The EPA (1996a) classifies bis (2-ethylhexyl)phthalate in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate human cancer data (one limited occupational study) and sufficient cancer data in laboratory animals. An oral slope factor of 0.014 per mg/kg/day was based on the increased incidence of liver tumors in a dietary study in male mice.

Ecological Effects

Bis(2-ethylhexyl)phthalate is highly lipid soluble and tends to partition into the lipid compartments of animals and plants. However, with the exception of a few aquatic crustacea and midge larvae, most organisms metabolize bis(2-ethylhexyl)phthalate at a rate sufficient to offset the tendency for bioconcentration (ATSDR, 1988). The Ohio EPA Warmwater Habitat Water Quality Criteria for bis(2-ethylhexyl)phthalate is set at 0.0084 mg/L; the USEPA benchmark is 0.32 mg/L (EPA, 1996b)

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR), 1988, Draft Toxicological Profile for Bis(2-ethylhexyl)phthalate.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed., ACGIH, Cincinnati, OH.

U.S. Environmental Protection Agency (EPA), 1995, Health Effects Assessment Summary Tables. Annual Update FY 1995, including Supplements. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996a, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996b, Ecotox Thresholds, EPA 540/F-95/038.

CHLOROMETHANE (METHYL CHLORIDE)

Chloromethane is a colorless gas at room temperature (ACGIH, 1991). It is used as a methylating agent in the synthesis of a wide variety of organic compounds, pesticides, pharmaceuticals and quarternary drugs, and as a blowing agent for some polystyrene and polyurethane foams.

Data regarding the gastrointestinal (GI) absorption of chloromethane were not located in the available literature. A compilation of data for 19 organic compounds of various chemical classes and molecular weights indicates that GI absorption ranges from approximately 50 percent to virtually complete, with an arithmetic mean of 90 percent (Jones and Owen, 1989). The value of 90 percent (0.9) appears to be adequately conservative for low molecular weight compounds and is adopted as the gastrointestinal absorption factor (GAF) for this evaluation.

The acute toxicity of oral treatment with chloromethane is relatively low, as suggested by an LD₅₀ of 1.8 g/kg in rats (Lewis, 1992); the signs preceding death were not reported. Data regarding the effects of subchronic or chronic oral exposure to chloromethane were not located in the available literature. The data are inadequate for derivation of a reference dose (RfD) for chronic oral exposure.

A four-hour inhalation LC₅₀ of 5300 mg/m³ was reported in rats, but the signs preceding death were not reported (Lewis, 1992). A four-hour LC₅₀ in an unreported species was accompanied by histological evidence of injury to the brain, lungs, kidneys and liver (ACGIH, 1991). Humans (occupational exposure) and laboratory animals (repeated exposure) exhibit neurological signs when acutely exposed to high levels (ACGIH, 1991; Lewis, 1992). Neurological damage is observed upon histological evaluation. If death does not occur quickly, histological damage is observed in the liver and kidneys. Occupational exposure to routine workplace levels is associated with ocular changes, neurological effects, GI effects and, occasionally, liver and kidney effects (ACGIH, 1991). Chronic intermittent exposure of rats and mice in a 2-year carcinogenicity experiment showed that both species develop liver and kidney degeneration and mice develop cerebellar degeneration, splenic atrophy and functional limb muscle impairment (ACGIH, 1991). Bilateral atrophy of the seminiferous tubules

was observed in the rats. The data were insufficient for derivation of a reference concentration (RfC) for the noncancer effects of inhalation exposure.

Chloromethane is reported to be an experimental teratogen, producing cardiac malformations in mice (but not rats) exposed by inhalation during organogenesis (ACGIH, 1991).

Cancer data are limited to the observation of a statistically significant increase in total malignant and benign tumors of the kidney in male mice in the intermittent exposure inhalation study briefly described above (ACGIH, 1991). EPA (1997) classified chloromethane in cancer weight-of-evidence Group C (possible human carcinogen) on the basis of the mouse data, and derived a provisional slope factor (SF) of $6.3E-3$ per mg/kg-day for inhalation exposure. A provisional SF of $1.3E-2$ per mg/kg-day for oral exposure was derived from the inhalation data, assuming that respiratory tract absorption is approximately one-half as efficient as GI absorption.

REFERENCES

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, Documentation of the Threshold Limit Values and Biological Exposure Indices, Sixth Edition, ACGIH, Cincinnati, OH, pp. 953-957.

Jones, TD. and BA Owen, 1989, Health Risks from Mixtures of Radionuclides and Chemicals in Drinking Water, Oak Ridge National Laboratory, Oak Ridge, TN, ORNL-6533.

Lewis, R.J., Sr., 1992, Sax's Dangerous Properties of Industrial Materials, Eighth Edition, Van Nostrand Reinhold, New York, pp. 2284-2285.

U.S. Environmental Protection Agency (EPA), 1987, Health Effects Assessment for Chloromethane, Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1997, Health Effects Assessment Summary Tables, FY 1997 Update, Office of Solid Waste and Emergency Response, 9200.6-303 (97-1), EPA-540-R-97-036, NTIS No. PB97-921199.

CHLOROFORM

Human Health Effects

Oral or inhalation exposure of animals to chloroform was associated with liver and kidney damage (ACGIH, 1991; EPA, 1996). In humans, acute inhalation exposure to high levels induced narcosis, ventricular fibrillation, and death (ACGIH, 1991). Limited occupational data associated chronic exposure to chloroform with CNS depression, digestive disturbances, and enlarged livers. The EPA (1996) presented a verified chronic oral RfD of 0.01 mg/kg/day based on a LOAEL for fatty cyst formation in the livers of dogs treated orally for 7.5 years and an uncertainty factor of 1,000. The same value was presented as a provisional subchronic oral RfD (EPA, 1993). Target organs for the toxicity of chloroform include the liver and kidney for oral and inhalation exposure, and the heart and CNS for inhalation exposure.

Chloroform is classified as a cancer weight-of-evidence Group B2 compound (probable human carcinogen), based on increased incidence of several tumor types in rats and liver tumors in mice (EPA, 1996). Human carcinogenicity data are inadequate. An oral slope factor of 0.0061 per mg/kg/day was derived from the incidence of kidney tumors in rats treated with chloroform in drinking water for two years. An inhalation unit risk of 2.3×10^{-5} per $\mu\text{g}/\text{m}^3$ was based on the incidence of hepatocellular carcinomas in mice treated by gavage for 78 weeks. The inhalation unit risk is equivalent to 0.081 per mg/kg/day, assuming an inhalation rate of 20 m^3/day and a body weight of 70 kg for humans.

Ecological Effects

Chloroform, also known as trichloromethane, primarily enters the environment as an industrial solvent. It is also released as a volatile product by plants (Howard, 1990). Chloroform poorly adsorbs to soil and sediment (NLM, 1996). Near the surface of soils, chloroform is expected to evaporate (Howard, 1990). The compound may leach into groundwater. Laboratory studies indicate that biodegradation of chloroform may also occur (Howard, 1990).

According to IARC (1972), small amounts of chloroform have been detected in tomatoes and muscat grapes. Information on the phytotoxicity of chloroform is limited.

Concentrations of chloroform greater than 0.25 percent have been shown to be lethal to plant cells (Kayser et al., 1982). Toxic effects and abnormal mitosis have been noted in plant cells exposed to 0.025 percent chloroform (Kayser et al., 1982).

Information on the concentration of chloroform in wild animals is limited. According to Pearson and McConnell (1975, as cited in NLM, 1996), grey seals collected from the English coast contained 7.6 to 22 g/kg chloroform in blubber and 0 to 12 g/kg chloroform in liver. Marine and freshwater birds collected from England contained 0.7 to 65 g/kg chloroform (Pearson and McConnell, 1975, as cited in NLM, 1996). Concentrations of chloroform in terrestrial mammals could not be located in the literature.

Laboratory studies have shown ingested chloroform to be eliminated in expired air and in urine (IARC, 1979, as cited in NLM, 1996). Liver microsomal enzymes metabolize chloroform to carbon monoxide (Stevens et al., 1979, as cited in NLM, 1996). The metabolism of chloroform to phosgene in the kidney can lead to nephrotoxicity (Branchflower et al., 1984, as cited in NLM, 1996).

Specific data on the toxicity of chloroform to wildlife do not exist. Exposure to chloroform via inhalation, ingestion, or dermal contact can induce toxic responses in mammals. Chloroform is a hepatotoxic compound (NLM, 1996). Acute toxicity to chloroform in experimental animals is species-, strain-, gender-, and age-dependent (Kayser et al., 1982, as cited in NLM, 1996). Oral LD₅₀ values for rats, mice, and rabbits exposed to chloroform are 908, 36, and greater than 20 mg/kg, respectively (RTECS, 1996). The dermal LD₅₀ value for rabbits exposed to chloroform is greater than 20 g/kg (RTECS, 1996). An inhalation LC₅₀ value of 47.702 g/m³/4 hr has been determined for rats exposed to chloroform (RTECS, 1996). Adverse impacts on fertility and fetotoxicity and teratogenicity have been reported in rats exposed chloroform at 30 ppm/7 hr during the sixth to fifteenth day of pregnancy (RTECS, 1996). An estimated NOAEL value for white-footed mice exposed to chloroform was 37.4 mg/kg/d, based on a laboratory rat study where the approximate NOAEL was 15 mg/kg/d (Opresko et al., 1994). Based on this same data, estimated oral NOAELs for chloroform were predicted to be 10.0 mg/kg/d for the cottontail rabbit and 6.5 mg/kg/d for the red fox (Opresko et al., 1994). Calculated chronic drinking-water

NOAELs for the white-footed mouse, cottontail rabbit, and red fox were 125, 123, and 76.5 mg/L, respectively (Opresko et al., 1994).

There is little tendency for chloroform to bioconcentrate in fish (Barrows et al., 1980, as cited in NLM, 1996). A bioconcentration factor of 6, however, has been reported for bluegill sunfish exposed to chloroform for a 14-day period (EPA, 1980, as cited in NLM, 1996).

A limited amount of data exist on the toxicity of chloroform to freshwater biota. Federal Water Quality Criteria do not exist for the protection of freshwater aquatic life from exposure to acetone (EPA, 1996). According to the EPA (EPA, 1996), however, acute toxicity has been noted in freshwater species at chloroform concentrations as low as 28,900 $\mu\text{g/L}$, and chronic toxicity may occur at concentrations as low as 1,240 $\mu\text{g/L}$. Suter and Mabrey (1994) has recommended acute and chronic advisory values of 3,360 and 188 $\mu\text{g/L}$, respectively, for the protection of freshwater biota. Lowest chronic toxicity values of chloroform to fish and daphnids are estimated as 1,240 and 4,483 $\mu\text{g/L}$, respectively (Suter and Mabrey, 1994). The test EC_{20} for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than a 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). The value for chloroform has been estimated to be 8,400 $\mu\text{g/L}$ (Suter and Mabrey, 1994).

REFERENCES

American Conference of Government Industrial Hygienists (ACGIH), 1991, Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed., Cincinnati, OH.

Barrows, M. E., et al., 1980, *Dynamic Exposure Hazard Assessment of Toxic Chemicals*, Ann Arbor Press, Ann Arbor, Michigan.

Branchflower, R. V., et al., 1984, *Toxicology and Applied Pharmacology*, Vol. 72, pp. 159-168.

Howard, P. H. (ed.), 1990, *Handbook of Environmental Fate and Exposure Data for Organic Chemicals, Vol. II, Solvents*, Lewis Publishers, Chelsea, Michigan.

International Agency for Research on Cancer (IARC), 1972 through 1986, *Monograph on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, World Health Organization, International Agency for Research on Cancer, Geneva, Switzerland.

Kayser, R., D. Sterling, and D. Viviani (eds.), 1982, *Intermedia Priority Pollutant Guidance Documents*, U.S. Environmental Protection Agency, Washington, D.C.

National Library of Medicine (NLM), 1996, "Hazardous Substance Data Bank," produced by Micromedex, Inc.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Pearson, C. R., and G. McConnell, 1975, *Proceedings of the Royal Society of London*, Series B, Vol. 189, pp. 305-332.

Registry of Toxic Effects of Chemical Substances (RTECS), 1996, produced by Micromedex.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

U.S. Environmental Protection Agency (EPA), 1996, *Integrated Risk Information System (IRIS)*, Computer Database, EPA, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1993, *Selecting Exposure Routes and Contaminants of Concern by Risk-Based Screening, Region III Technical Guidance Manual*.

U.S. Environmental Protection Agency (EPA), 1980, "Ambient Water Quality Criteria Document: Chloroform," *EPA 440/5-80-033*, U.S. Environmental Protection Agency, Washington, D.C.

COBALT

Human Health Effects

Acute high oral or parenteral doses of cobalt in humans or animals induced myocardial degeneration often leading to mortality, aplastic anemia, enlarged thyroid, and, in animals, renal tubular degeneration (Elinder and Friberg, 1986). Chronic ingestion from the consumption of beer containing high concentrations of cobalt was associated with "beer-drinkers cardiomyopathy," which includes polycythemia and goiter, as well as marked myocardial degeneration and mortality. The therapeutic use of 0.16 to 0.32 mg cobalt/kg/day in anemic, anephric dialysis patients for 12 to 32 weeks induced a significant, but reversible, rise in blood hemoglobin concentration (EPA, 1992).

Occupational (inhalation and dermal) exposure was associated with allergic dermatitis, chronic interstitial pneumonitis, reversibly impaired lung function, occupational asthma, and myocardial effects (ACGIH, 1991). Cobalt was determined to be the etiologic factor in hard metal disease, the syndrome of respiratory symptoms, and pneumoconiosis associated with inhalation exposure to dusts containing tungsten carbide with cobalt powder as a binder (Elinder and Friberg, 1986). The lowest occupational air concentration of cobalt associated with hard metal disease was 0.003 mg cobalt/m³ (Sprince et al. 1988). It should be noted that the workers were also exposed to tungsten and sometimes to titanium, tantalum, and niobium (Elinder and Friberg, 1986). Similar lung effects were seen in animals exposed to cobalt by inhalation.

The developmental toxicity of cobalt was tested in rodents treated orally with cobalt chloride (EPA, 1992). Maternal effects (unspecified) were reported in rats treated with 5.4 to 21.8 mg cobalt/kg/day from gestation day 14 through lactation day 21. Effects on the offspring included stunted growth at 5.4 mg cobalt/kg/day and reduced survival at 21.8 mg cobalt/kg/day. In rats treated with 6.2, 12.4, or 24.8 mg cobalt/kg/day on gestation days 6 through 15, maternal effects included reduced food consumption and body weight gain and altered hematologic parameters, although it is unclear at what dose level(s) these effects occurred. There were no effects on fetal survival, although a nonsignificant increase in fetal stunting was observed in rats treated with 12.4 mg

cobalt/kg/day. Mice treated with 81.7 mg cobalt/kg/day had reduced maternal weight gain, but no fetal effects.

Several studies reported testicular degeneration and atrophy in rats treated with cobalt chloride in the diet or drinking water at concentrations equivalent to doses of 5.7 to 30.2 mg cobalt/kg/day (EPA, 1992).

Cobalt is nutritionally essential as a cofactor in cyanocobalamin (vitamin B12) (EPA, 1992). Cobalt is universally present in the diet. Average daily adult dietary intakes of cobalt range from 0.16 to 0.58 mg/day (0.002 to 0.008 mg/kg/day, assuming adults weight 70 kg) (Tipton et al., 1966). In 9- to 12-year-old children, dietary intakes of cobalt range from 0.3 to 1.77 mg/day (Murthy et al., 1971; National Research Council, 1989). Assuming an average weight for children in this age range of 28 kg (National Research Council, 1989), the dietary intakes are equivalent to 0.01 to 0.06 mg/kg/day.

The EPA (1992) concluded that the oral toxicity data were insufficient for derivation of an oral RfD for cobalt. The relatively well characterized dietary intake data, however, can provide useful guidance. The EPA (1992) noted that the upper range of dietary intake for children, 0.06 mg/kg/day, was below the level associated with enhanced erythropoiesis in anephric patients. Therefore, the upper range of dietary intake, 0.06 mg cobalt/kg/day, can be considered a guidance level for the oral intake of cobalt and can be used in place of an oral RfD in CERCLA and RCRA baseline risk assessments.

The EPA (1990) derived an interim inhalation RfC from the LOAEL of 0.003 mg cobalt/m³ associated with hard metal disease in occupationally exposed humans (Sprince et al., 1988). Correcting for intermittent occupational exposure (10 m³ of air inhaled per work day/20 m³ of air inhaled per day x 5 work days per week/7 days per week) yielded an adjusted LOAEL of 0.001 mg/m³. Application of an uncertainty factor of 1000 resulted in an interim chronic inhalation RfC of 1x10⁻⁶ mg/m³. Assuming humans inhale 20 m³ of air/day and weight 70 kg, the RfC is equivalent to 2.9x10⁻⁷ mg/kg/day, rounded to 3x10⁻⁷ mg/kg/day.

Important target organs in orally exposed humans are the heart, erythrocyte, and thyroid. Target organs for occupational exposure are the skin, lungs, and heart.

Data regarding the carcinogenicity of cobalt were not located.

Ecological Effects

Although cobalt is essential to some blue-green algae, fungi, and microorganisms, it is not essential for the growth of higher plants (Kabata-Pendias and Pendias, 1992; Vanderploeg et al., 1975). Background concentrations of cobalt in immature grasses and clovers collected in the United States averaged 0.08 mg/kg (dry weight) and 0.19 mg/kg (dry weight), respectively (Kabata-Pendias and Pendias, 1992). Plants that grow on serpentine soils or in soils naturally high in cobalt usually contain higher concentrations of the element (Kabata-Pendias and Pendias, 1992). Cobalt accumulator plants include species from the families Cruciferae, Caryophyllaceae, Violaceae, Leguminosae, Boraginaceae, Myrtaceae, and Nyssaceae (Kabata-Pendias and Pendias, 1992). Some of these plants are known to contain concentrations of cobalt as high as 4,000 mg/kg (dry weight) without apparent harm to the plant (Brooks, 1977). Several abiotic factors govern the availability of cobalt to plants. Soil factors include organic matter and clay content, pH, leachability, and concentration of manganese and iron oxides (Kabata-Pendias and Pendias, 1992). Uptake of cobalt can occur via the roots or leaves of a plant.

Concentrations of cobalt in leaf tissue that are excessive or toxic to various plant species, with the exclusion of very sensitive and highly tolerant species, range from 15 to 50 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Concentrations of cobalt in plant tissue that could result in a 10 percent reduction in crop yield range from 20 to 40 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 20 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for cobalt phytotoxicity. General symptoms of cobalt toxicity in plants include interveinal chlorosis in new leaves followed by induced iron chlorosis and white leaf margins and damaged root tips (Kabata-Pendias and Pendias, 1992).

Cobalt is a component of vitamin B₁₂ and therefore, is an essential micronutrient for animal growth. Ruminants require a minimum of between 0.08 to 0.1 mg/kg (dry weight) in their diet to prevent cobalt deficiency (Kabata-Pendias and Pendias, 1992). Background concentrations of cobalt in mammals and birds are usually less than 0.75 mg/kg (wet weight) and less than 0.3 mg/kg (wet weight), respectively (Jenkins,

1980). According to Talmage and Walton (1991), cobalt concentrations in the livers and kidneys of various species of small mammals were generally less than 5 mg/kg (dry weight). Highest concentrations of cobalt in the body occur in kidney and liver tissues (Gregus and Klaassen, 1986). Urine is the predominant route of excretion for cobalt (Gregus and Klaassen, 1986).

Cobalt toxicity may occur following exposure via ingestion or inhalation. Skin and eye lesions have also been associated with exposure to cobalt (Hammond and Beliles, 1980). Juvenile rats were found to tolerate an acute dose of 1,250 mg of cobalt in a single dietary dose, whereas a daily dose of 30 mg of cobalt metal over a period of one month was found to be fatal (Venugopal and Luckey, 1978). Changes in thyroid function were noted in rats exposed to aerosols of cobalt metal at 0.5 mg/m³ (Popov et al., 1977, as cited in NLM, 1996). No adverse effects were found in chickens and sheep given dosages of cobalt under 50 mg/kg diet or under 2 mg/kg of body weight (NRC, 1977, as cited in NLM, 1996).

Cobalt concentrations in tadpoles (*Rana catesbeiana* and *Rana clamitans*) collected from uncontaminated sites were reported to contain less than 2 mg/kg cobalt (dry weight) (Hall and Mulhern, 1984). A mixed group of adult frogs and toads collected from the same site averaged 2.1 mg/kg cobalt (dry weight) (Hall and Mulhern, 1984). Background concentrations of cobalt in the muscle of freshwater fish are generally less than 0.1 mg/kg (wet weight) (Vanderploeg et al., 1975). Increased dissolved organic matter concentrations in eutrophic waters are believed to keep cobalt in solution (Vanderploeg et al., 1975). Soluble cobalt in eutrophic lakes is generally less available than soluble cobalt in oligotrophic or mesotrophic lakes (Vanderploeg et al., 1975). This is supported by the fact that lower bioconcentration factors have been reported for fish in eutrophic environments (Vanderploeg et al., 1975). Cobalt bioconcentration factors for whole fish average 43.8 in eutrophic waters and 439 in mesotrophic waters (based on filtered water samples, Vanderploeg et al., 1975). Cobalt bioconcentration factors for submerged and floating vascular aquatic plants in eutrophic water have been reported as 2,000 and 400, respectively (Vanderploeg et al., 1975). Vanderploeg et al. (1975) recommend the use of 400 and 10,000 as bioconcentration factors for cobalt in eutrophic and in either mesotrophic or oligotrophic waters, respectively. Copepods have been reported to have a bioconcentration of 700 for cobalt (Vanderploeg et al.,

1975). Cobalt bioaccumulation factors tend to decrease with increasing trophic level (Vanderploeg et al., 1975).

The USEPA benchmark for cobalt is 0.003 mg/L (EPA, 1996). No Ohio EPA Warmwater Habitat Water Quality Criteria exists for cobalt. Suter and Mabrey (1994), however, have estimated acute and chronic advisory levels for cobalt to be 195 and 3.06 $\mu\text{g/L}$, respectively. The lowest chronic values of cobalt reported in the literature for fish and *Daphnia* are 290 and 5.1 $\mu\text{g/L}$, respectively (Suter and Mabrey, 1994). The test EC_{20} for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration of cobalt causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). The value for cobalt is 810 $\mu\text{g/L}$ (Suter and Mabrey, 1994). A similar value can be determined for daphnids, which reflects the highest tested concentration of cobalt causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC_{20} benchmark for daphnids is less than 4.4 $\mu\text{g/L}$ (Suter and Mabrey, 1994).

REFERENCES

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed., ACGIH, Cincinnati, OH.

Brooks, R. R., 1977, "Copper and Cobalt Uptake by *Hausmaniastrum* Species," *Plant and Soil*, Vol. 48, p. 545.

Elinder, C.G., and L. Friberg, 1986, Cobalt, Handbook on the Toxicology of Metals, L. Friberg, G. F. Nordberg, and V. B. Vouk, eds., Vol. II, Elsevier Science Publishers B. V., New York, pp. 211-232.

Gregus, Z., and C. D. Klaassen, 1986, "Disposition of Metals in Rats: A Comparative Study of Fecal, Urinary, and Biliary Excretion and Tissue Distribution of Eighteen Metals," *Toxicology and Applied Pharmacology*, Vol. 85, pp. 24-38.

Hall, R. J., and B. M. Mulhern, 1984, "Are Anuran Amphibians Heavy Metal Accumulators?," in R. A. Seigel, L. E. Hunt, J. L. Knight, L. Malaret and N. L. Zuschlag (eds.), *Vertebrate Ecology and Systematics—A Tribute to Henry S. Fitch*, Museum of Natural History, University of Kansas, Lawrence, Kansas, pp. 123-133.

Hammond, P. B., and R. P. Beliles, 1980, "Metals," in J. Doull, C. D. Klaassen, and M. O. Amdur (eds.), *Casarett and Doull's Toxicology: The Basic Science of Poisons*, 2nd ed., Macmillan Publishing Co., Inc., New York, New York, pp. 409-4671.

Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.

Murthy, G. K., U. Rhea, and J. T. Peeler, 1971, Levels of Antimony, Cadmium, Chromium, Cobalt, Manganese, and Zinc in Institutional Total Diets, *Environmental Science and Technology*, Vol. 5, pp. 436-442, (Cited in EPA 1992).

National Library of Medicine (NLM), 1996, "Hazardous Substance Data Bank," produced by Micromedex, Inc.

National Research Council (NRC), 1989, Recommended Dietary Allowances, 10th Edition. Washington, DC: National Academy Press.

National Research Council (NRC), 1977, "Drinking Water and Health Volume 1," National Academy Press, Washington, D.C., 248 pp.

Popov, L. N., et al., 1977, *Gig Sanit* 42, pp. 12-15.

Sprince, N. L., L. C. Oliver, E. A. Eisen, et al., 1988, Cobalt Exposure and Lung Disease in Tungsten Carbide Production: A Cross-sectional Study of Current Workers, *American Review of Respiratory Diseases*, Vol. 138, pp. 1220-1226, (Cited in EPA 1990).

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Talmage, S. S., and B. T. Walton, 1991, "Small Mammals as Monitors of Environmental Contaminants," *Reviews in Environmental Contamination and Toxicology*, Vol. 119, pp. 47-145.

Tipton, I. H., P. L. Stewart, and P. G. Martin, 1966, "Trace Elements in Diets and Excreta," *Health Physics*, Vol. 12, pp. 1683-1689, (Cited in EPA 1992).

U.S. Environmental Protection Agency (EPA), 1990, Memorandum from Pei-Fung Hurst, ECAO, Cincinnati, OH to R. Riccio, EPA Region III, Philadelphia, PA, Subject: Toxicity of Cobalt (Halby Chemical/Wilmington, Delaware), dated October 9, 1990.

U.S. Environmental Protection Agency (EPA), 1992, Memorandum from D. L. Forman, U.S. EPA Region III, Philadelphia, PA, Subject: Cobalt Toxicity, dated March 12, 1992.

U.S. Nuclear Regulatory Commission, 1996, Ecotox Thresholds, EPA 540/F-95/038.

Vanderploeg, H. A., D. C. Parzyck, W. H. Wilcox, J. R. Kercher, and S. V. Kaye, 1975, "Bioaccumulation Factors for Radionuclides in Freshwater Biota," *ORNL-5002*, Oak Ridge National Laboratories, Oak Ridge, Tennessee.

Venugopal, B., and T. D. Luckey, 1978, *Metal Toxicity in Mammals*, Vol. 2, Plenum Press, New York, New York, 289 pp.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

COPPER

Human Health Effects

Copper is a nutritionally essential element that functions as a cofactor in several enzyme systems (Aaseth and Norseth, 1986). Acute exposure to large oral doses of copper salts was associated with GI disturbances, hemolysis, and liver and kidney lesions. Chronic oral toxicity in humans has not been reported. Chronic oral exposure of animals was associated with an iron-deficiency type of anemia, hemolysis, and lesions in the liver and kidneys. Occupational exposure may induce metal fume fever, and, in cases of chronic exposure to high levels, hemolysis and anemia (ACGIH, 1991). Neither oral nor inhalation RfD or RfC values were located for copper. The target organs for copper are the erythrocyte, liver, and kidney, and, for inhalation exposure, the lung.

Copper is classified in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) (EPA, 1995). Quantitative risk estimates are not derived for Group D chemicals.

Ecological Effects

Copper is an essential nutrient for the growth of plants. Background concentrations of copper in grasses and clovers collected in the United States averaged 9.6 mg/kg (dry weight) and 16.2 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Some plants are able to accumulate and tolerate elevated levels of copper within their tissues. The shoots and roots of the coffee plant (*Coffea arabica*) may reach 4,186 mg/kg (dry weight) (Lepp and Dickinson, 1987, as cited in Kabata-Pendias and Pendias, 1992). Copper is one of the least mobile heavy metals in soil, and its availability to plants is highly dependent on the molecular weight of soluble copper complexes (Kabata-Pendias and Pendias, 1992). Copper-containing fungicides and bactericides are used in the control of some crop pests.

According to Rhodes et al. (1989), copper concentrations in plant tissues do not serve as conclusive evidence of copper toxicity in species of plants such as tomatoes, because some species are able to tolerate higher concentrations of copper than others. The pH of the soil may also influence the availability and toxicity of copper in soils to plants (Rhodes et al., 1989). In a study with tomato plants, Rhodes et al. (1989) found a

reduction in plant growth when plants were grown in soils containing greater than 150 mg/kg of copper at a pH of less than 6.5. At pH values of greater than 6.5, soil copper concentrations of greater than 330 mg/kg were required to reduce plant growth. Concentrations of copper in leaf tissue that are excessive or toxic to various plant species, with the exclusion of very sensitive and highly tolerant species, range from 20 to 100 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Concentrations of copper in plant tissues that are expected to result in a 10 percent loss in crop yield range from 10 to 30 mg/kg (dry weight) (Macnicol and Beckett, 1985). A soil concentration of 100 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for copper phytotoxicity in soils. General symptoms of copper toxicity in plants include the presence of dark green leaves followed by induced iron chlorosis; thick, short, or barbed-wire roots; and depressed tillering (Kabata-Pendias and Pendias, 1992).

Copper is an essential element for hemoglobin synthesis and oxidative enzymes in animals. Copper concentrations in small mammals collected from various uncontaminated sites ranged from 8.3 to 13.4 mg/kg (whole-body concentrations) (Talmage and Walton, 1991). Concentrations of copper in liver and kidney tissue of voles (*Microtus agrestis*) collected from uncontaminated grasslands were found to average 16 mg/kg (dry weight) and 21 mg/kg (dry weight), respectively (Beardsley et al., 1978). Highest concentrations of copper tend to be in hair, followed in decreasing concentration by liver, kidney, and whole body (Hunter and Johnson, 1982). Animals that reside near mining and refinery operations and sewage-treated areas usually contain elevated concentrations of copper. Among the small mammals collected, Hunter and Johnson (1982) found shrews (*Sorex araneus*) to contain the highest concentrations of copper. Mice were found to contain the lowest copper concentrations. Copper concentrations in the muscle tissue of migratory blue-winged teal (*Anas discors*) were found to average 6.57 mg/kg (dry weight) in autumn and 4.96 mg/kg (dry weight) in spring (Warren et al., 1990).

Based on toxicity data specific to the mink, extrapolated NOAELs for chronic exposure of various mammalian wildlife species to copper sulfate are 41.3 mg/kg/d for the white-footed mouse, 11.0 mg/kg/d for the cottontail rabbit, and 7.13 mg/kg/d for the red fox (Opresko et al., 1994). Examples of calculated chronic drinking-water NOAELs for

mammalian wildlife are 138 mg/L for the white-footed mouse, 114 mg/L for the cottontail rabbit, and 84.4 mg/L for the red fox (Opresko et al., 1994). Symptoms of acute copper poisoning in mammals include vomiting, hypotension, melena, coma, jaundice, and death (Hammond and Beliles, 1980). Selenium can act as an antidote for copper poisoning (Hammond and Beliles, 1980).

Based on toxicity test data specific to the chicken, extrapolated NOAELs for chronic exposure of avian species to copper oxide are 20.2 mg/kg/d for the great blue heron and 25.9 mg/kg/d for the red-tailed hawk (Opresko et al., 1994). The calculated drinking-water NOAEL for these birds consuming copper oxide exclusively through drinking water is 457 mg/L (Opresko et al., 1994).

Concentrations of copper in freshwater fish collected from 112 monitoring stations in the United States from 1978 to 1981 ranged from 0.25 to 38.75 mg/kg (wet weight), with an average of 0.68 mg/kg (wet weight) during 1980 to 1981 (Lowe et al., 1985). Because shellfish contain copper proteins in their blood that act as oxygen carriers, molluscs often contain higher concentrations of copper than other aquatic species (Hammond and Beliles, 1980). Copper concentrations in *Sphagnum* moss collected from northern Canadian freshwaters ranged from 13 to 15 mg/kg (Glooschenko and Capobiano, 1978, as cited in Leland et al., 1979). Adult toads of certain species can accumulate high concentrations of copper in their livers without apparent adverse effects (Hall and Mulhern, 1984). This is believed to be related to the ability of liver lysosomes, present within these toads, to sequester copper, thereby preventing the copper from damaging liver cells (Goldfischer et al., 1970). The bioconcentration factor for freshwater aquatic invertebrates exposed to copper is 1,000 (Bodek et al., 1988).

Fish appear relatively sensitive to copper. This is attributed to the absorption of copper across the gills (Hammond and Beliles, 1980). Laboratory studies have shown an increase in the bioavailability and toxicity of copper at low pH (Stokes et al., 1985; Schubauer-Berigan et al., 1993; Cusimano et al., 1986). Federal Water Quality Criteria for the Protection of Aquatic Life from acute and chronic exposure to copper in freshwater systems are 18 and 12 µg/L, respectively (EPA, 1996a). These criteria are based on a water hardness of 100 mg/L. Because the toxicity of copper to aquatic

organisms is affected by water hardness, all water quality criteria must be adjusted with site-specific water hardness data. The USEPA ecotox threshold benchmark for copper is 0.011 mg/L (EPA, 1996b). The Ohio Warmwater Habitat Water Quality Criteria for copper is set at 0.0062 mg/L. The lowest chronic values of copper reported in the literature for fish and *Daphnia* are 3.8 and 0.23 $\mu\text{g/L}$, respectively (Suter and Mabrey, 1994). The test EC_{20} for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). The value for copper is 5 $\mu\text{g/L}$ (Suter and Mabrey, 1994). A similar value can be determined for daphnids, which reflects the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC_{20} benchmark for daphnids is 0.205 $\mu\text{g/L}$ (Suter and Mabrey, 1994).

REFERENCES

Aaseth, J., and T. Norseth, 1986, Copper, Handbook on the Toxicology of Metals, Vol. II, L. Friberg, G. F. Nordberg, and V. B. Vouk, eds., Elsevier Science Publishers B. V., New York, pp. 233-254.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed., ACGIH, Cincinnati, OH.

Beardsley, A., M. J. Vagg, P. H. T. Beckett, and B. F. Sansom, 1978, "Use of the Field Vole (*M. agrestis*) for Monitoring Potentially Harmful Elements in the Environment," *Environmental Pollution*, Vol. 16, pp. 65-71.

Bodek, I., W. J. Lyman, W. F. Reehl, and D. H. Rosenblatt (eds.), 1988, *Environmental Inorganic Chemistry: Properties, Processes, and Estimation Methods*, Pergamon Press, New York, New York.

Cusimano, R. F., D. F. Brakke, and G. A. Chapman, 1986, "Effects of pH on the Toxicities of Cadmium, Copper and Zinc to Steelhead Trout (*Salmo gairdneri*)," *Canadian Journal of Fish and Aquatic Sciences*, Vol. 43, Canada, pp. 1497-1503.

Glooschenko, W. A., and J. H. Capobianco, 1978, "Metal Content of *Sphagnum* Mosses from Two Canadian Bog Ecosystems," *Water, Air and Soil Pollution*, Vol. 10, pp. 215.

Goldfischer, S., B. Schiller, and I. Sternwieg, 1970, "Copper in Hepatocyte Lysosomes of the Toad *Bufo marinus* L.," *Nature*, Vol. 228, pp. 172-173.

Hall, R. J., and B. M. Mulhern, 1984, "Are Anuran Amphibians Heavy Metal Accumulators?," in R. A. Seigel, L. E. Hunt, J. L. Knight, L. Malaret and N. L. Zuschlag (eds.), *Vertebrate Ecology and Systematics—A Tribute to Henry S. Fitch*, Museum of Natural History, University of Kansas, Lawrence, Kansas, pp. 123-133.

Hammond, P. B., and R. P. Beliles, 1980, "Metals," in J. Doull, C. D. Klaassen, and M. O. Amdur (eds.), *Casarett and Doull's Toxicology: The Basic Science of Poisons*, 2nd ed., Macmillan Publishing Co., Inc., New York, New York, pp. 409-4671.

Hunter, B. A., and M. S. Johnson, 1982, "Food Chain Relationship of Copper and Cadmium in Contaminated Grassland Ecosystems," *Oikos*, Vol. 38, pp. 108-177.

Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.

Leland, H. V., S. N. Luoma, and J. M. Fielden, 1979, "Bioaccumulation and Toxicity of Heavy Metals and Related Trace Elements," *Journal of Water Protection, Control Federation*, Vol. 51, pp. 1592-1616.

Lepp, N. W. and N. M. Dickinson, 1987, "Partitioning and Transport of Copper in Various Components of Kenyan *Coffea arabica* Stands," in *Pollution Transport and Fate in Ecosystems*, P. J. Coughtrey, M. H. Martin, and M. H. Unsworth (eds.), Blackwell Scientific Publications, Oxford, England, 289 pp.

Lowe, T. P., T. W. May, W. G. Brumbaugh, and D. A. Kane, 1985, "National Contaminant Biomonitoring Program: Concentrations of Seven Elements in Freshwater Fish, 1978-1981," *Archives of Environmental Contamination and Toxicology*, Vol. 14, pp. 363-388.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Rhodes, F. M., S. M. Olson, and A. Manning, 1989, "Copper Toxicity in Tomato Plants," *Journal of Environmental Quality*, Vol. 18, pp., 195-197.

Schubauer-Berigan, M. K., J. R. Dierkes, P. D. Monson, and G. T. Ankley, 1993, "pH-dependent Toxicity of Cd, Cu, Ni, Pb and Zn to *Ceriodaphnia dubia*, *Pimephales promelas*, *Hyaella azteca* and *Lumbriculus variegatus*," *Environmental Contamination and Toxicology*, Vol. 12, pp. 1261-1266.

Stokes, P. M., R. C. Bailey, and G. R. Groulx, 1985, "Effects of Acidification On Metal Availability to Aquatic Biota, with Special Reference to Filamentous Algae," *Environmental Health Perspectives*, Vol. 63, pp. 79-87.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Talmage, S. S., and B. T. Walton, 1991, "Small Mammals as Monitors of Environmental Contaminants," *Reviews in Environmental Contamination and Toxicology*, Vol. 119, pp. 47-145.

U.S. Environmental Protection Agency (EPA), 1996a, "Integrated Risk Information System," on-line database, maintained by the U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency (EPA), 1996b, Ecotox Threshold, EPA 540/F-95/038.

U.S. Environmental Protection Agency (EPA), 1995, Health Effects Assessment Summary Tables. Annual Update FY 1995, including Supplements. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

Warren, R. J., B. M. Wallace, and P. B. Bush, 1990, "Trace Elements in Migrating Blue-winged Teal: Seasonal-, Sex- and Age-Class Variations," *Environmental Contamination and Toxicology*, Vol. 9, pp. 521-528.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

1,2-DICHLOROETHANE

Human Health Effects

Oral or inhalation exposure to humans or laboratory animals to 1,2-dichloroethane induced liver and kidney effects (ACGIH, 1991). Inhalation exposures also induced pulmonary congestion or edema, and, in humans, CNS depression. Neither oral nor inhalation RfD or RfC values were located. The target organs for 1,2-dichloroethane toxicity are the liver, kidney, lung, and CNS.

EPA classifies 1,2-dichloroethane as a cancer weight-of-evidence Group B2 compound (probable human carcinogen), based on the induction of several tumor types in rats and mice treated by gavage, and on the induction of benign lung papillomas in mice after dermal application (EPA, 1996). The EPA (1995) presented a slope factor for oral exposure of $0.091(\text{mg/kg/day})^{-1}$, and a unit risk for inhalation exposure of $2.6 \times 10^{-6} (\mu\text{g/m}^3)^{-1}$, based on the incidence of vascular system hemangiosarcomas in male rats in the gavage study. The inhalation unit risk is equivalent to $0.091 (\text{mg/kg/day})^{-1}$, assuming humans inhale 20 m^3 of air/day and weigh 70 kg.

Ecological Effects

When released into the environment, 1,2-dichloroethane is rapidly volatilized (ATSDR, 1997). Based on a biconcentration factor of 2 (Banerjee and Baughman, 1991, as cited in ATSDR, 1997) it is not expected to biconcentrate in fish or aquatic organism or bioaccumulate in the food chain (Farrington, 1991 as cited in ATSDR, 1997).

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR), 1997, ATSDR's Toxicological Profiles on CD-Rom, CRC Press, Inc.

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed., ACGIH, Cincinnati, OH.

Banerjee S., Baughman G.L. 1991. Bioconcentration factors and lipid solubility. Environ Sci Technol 25:536-539.

Farrington, J.W. 1991. Biogeochemical processes governing exposure of organic pollutant compounds in aquatic organisms. Environ Health Perspect 90:75-84.

U.S. Environmental Protection Agency (EPA), 1995, Health Effects Assessment Summary Tables. Annual Update FY 1995, including Supplements. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

1,2-DICHLOROETHYLENE

Human Health Effects

1,2-Dichloroethylene (1,2-DCE) exists as one of two isomers: cis-1,2-dichloroethylene and trans-1,2-dichloroethylene (according to the spatial orientation of the chlorine substitutes on the carbon chain). It is used directly in the synthesis of other chlorinated solvents, as a low temperature extraction solvent, as a solvent for organic synthesis and as a solvent for specialty applications. The limited toxicity data for 1,2-DCE has been reviewed by ATSDR (1989), NLM (1996), Torkelson and Rowe (1981) and EPA (1984a, 1984b and 1991).

Limited pharmacokinetic data indicate that 1,2-DCE is absorbed readily from the respiratory tract. Because it is a neutral, low molecular weight, lipophilic substance, it is expected to be readily absorbed by oral and dermal routes of exposure. 1,2-DCE is metabolized by hepatic cytochrome P-450. Data could not be located regarding the excretion of 1,2-DCE following oral, inhalation or dermal exposure.

Limited information is available regarding the toxicity of 1,2-DCE. The majority of available studies were done on the trans-isomer or on mixtures of the cis- and trans-isomers. Biochemical studies in rats with sublethal oral does suggest that the cis- and trans-isomer shows some evidence of hepatotoxicity and nephrotoxicity. Studies on the mixed isomers indicate effects in the central nervous system, liver and kidney. Because of the lack of data regarding carcinogenicity in humans and animals, 1,2-DCE is not classifiable as to carcinogenicity.

Ecological Effects

The dominant fate of 1,2-DCE in surface water is expected to be rapid volatilization to the atmosphere. The half-life for the volatilization for a model river is approximately 3 hours. Microbial degradation of 1,2-DCE under aerobic conditions can occur, but at a slow rate. The EPA has estimated a ground water half-life of 8 weeks to 95 months. It is not expected to significantly bioconcentrate in fish and aquatic organisms, nor is it expected to absorb to sediment and suspended organic matter. Limited data are available on the fate of cis-1,2-DCE in soil. It is expected to display high mobility in soil and leach into ground water. The EPA has estimated a soil half-life of 4 weeks to

6 months. Experimental results indicate that microbial degradation of cis-1,2-DCE in soil may occur with certain organisms if secondary nutritional sources are available. In soil and groundwater, cis-1,2-DCE gives the products chloroethane and vinyl chloride and trans-1,2-DCE gives vinyl chloride, exclusively.

1,2-DCE had LC₅₀ values of 218 mg/L in water fleas, *Daphnia magna*, exposed for 48 hours and 135 mg/L in bluegill sunfish, *Lepomis macrochirus*, exposed for 96 hours.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR), 1989, Draft Toxicological Profile for 1,2-Dichloroethene, U.S. Public Health Service, Atlanta, GA.

National Library of Medicine (NLM), 1996, Hazardous Substances Databank, Toxicology Information Network (TOXNET).

Torkelson, T.R. and V.K. Rowe, 1981, Halogenated Aliphatic Hydrocarbons Containing Chlorine, Bromine, and Iodine, in Patty's Industrial Hygiene and Toxicology, George D. Clayton and Florence E. Clayton (Editors), Third Revised Edition, Volume 2B. New York, John Wiley & Sons.

U.S. Environmental Protection Agency (EPA), 1984a, Health Effects Assessment for cis-1,2-Dichloroethylene. Office of Health and Environmental Assessment, Washington, DC. ECAO-CIN-H015.

U.S. Environmental Protection Agency (EPA), 1984b, Health Effects Assessment for 1,2-t-Dichloroethylene. Office of Health and Environmental Assessment, Washington, DC. ECAO-CIN-H041.

U.S. Environmental Protection Agency (EPA), 1991, Health and Environmental Effects Document for 1,2-Dichloroethylene (Mixed Isomers). ECAO-CIN-G099.

ETHYLBENZENE

Human Health Effects

Ethylbenzene is used as a solvent and as a chemical intermediate in the production of styrene and other compounds. It is found in gasoline and similar petroleum distillates. Ethylbenzene is soluble in most organic solvents and only slightly soluble in water. The limited data on ethylbenzene are reviewed in Sandmeyer (1981), EPA (1985), and NLM (1996).

In the atmosphere, ethylbenzene degrades rapidly. The half-life for this process ranges from a few hours to 2 days. The major mechanisms of loss from ambient water are volatilization and biodegradation; half-lives range from several hours to approximately two weeks. Ethylbenzene has a moderate tendency to be absorbed by soil. Fate processes in soil include evaporation, leaching to groundwater and biodegradation in the presence of acclimated microbes. The half-life of ethylbenzene in soil has been estimated to be in the range of 3 to 10 days based on the unacclimated aqueous aerobic half-life of ethylbenzene. The EPA has estimated the half-life of ethylbenzene in ground water to be in a range of 6 to 228 days.

Ethylbenzene is well absorbed from the lung and gastrointestinal tract, but poorly absorbed through the skin. It is distributed throughout the body but, due to its lipophilic nature, will accumulate primarily in adipose tissue. Small amounts are exhaled unchanged, but most is metabolized in the liver, primarily by oxidation of the side-chain, and excreted in the urine. Metabolic pathways vary considerably among species; in humans, mandelic acid (2-phenyl-2-hydroxyacetic acid), and phenylglyoxylic acid (2-phenyl-2-ketoacetic acid) are the major urinary metabolites.

Acute effects from oral and inhalation exposures target the central nervous system and lungs. Following inhalation pulmonary irritation and nervous system depression leading to a dose-related anesthetic effect is experienced. Repeated doses have been reported to cause a number of lung, nervous system, bone marrow, and hepatic lesions in workers. The most common complaint was inflammation of the respiratory tract. In addition, renal effects are observed in chronically exposed animals. Limited reproductive studies describe testicular degeneration in rabbits and monkeys.

Ethylbenzene has been shown to be embryotoxic and teratogenic. Observed effects include retarded skeletal development, reduced weight gain, and increased incidence of extra ribs and sacral displacement with abnormal development. The EPA classifies ethylbenzene in Group D, not classified as to carcinogenicity due to a lack of data.

Ecological Effects

Ethylbenzene is a product of biomass combustion and a component of crude oil (Graedel, 1986, and Nunes and Benville, 1979, as cited in NLM, 1996). It is also used in the manufacture of styrene and is used as a solvent (EPA, 1980, as cited in NLM, 1996). When released onto soil, part of the ethylbenzene will evaporate, and some may adsorb to soil (NLM, 1996). Biodegradation is not considered a significant removal process (NLM, 1996). Both evaporation and biodegradation are involved in the removal of ethylbenzene from aquatic environments (NLM, 1996). Ethylbenzene can adsorb to sediment (NLM, 1996).

Ethylbenzene has been detected in roasted filbert nuts (EPA, 1980, as cited in NLM, 1996) and dried legumes (Lovegren et al., 1979). Concentrations in legumes ranged from not detected to 11 g/kg in beans and averaged 13 g/kg in split peas and 5 g/kg in lentils (Lovegren et al., 1979, as cited in NLM, 1996). Information on the phytotoxicity of ethylbenzene could not be found.

Absorption of ethylbenzene is primarily by inhalation (Patty, 1963, as cited in NLM, 1996). Most of the inhaled compound is excreted in urine as metabolites (Patty, 1963, as cited in NLM, 1996). Absorption through the skin is slow (Clayton and Clayton, 1982, as cited in NLM, 1996). Ethylbenzene can be transported across the placenta (Clayton and Clayton, 1982, as cited in NLM, 1996). Concentrations of ethylbenzene in wild birds and mammals could not be located in the literature.

Specific data on the toxicity of ethylbenzene to wildlife do not exist. Exposure to high concentrations of ethylbenzene via ingestion, inhalation, or dermal absorption can cause depression of the central nervous system in animals (Friberg et al., 1986, as cited in NLM, 1996). Concentrations of ethylbenzene greater than 2 mg/L have been reported to be acutely toxic in laboratory animals (ILO, 1983, as cited in NLM, 1996). The oral LD₅₀ value for rats exposed to ethylbenzene is 3.5 g/kg (RTECS, 1996). The dermal

LD₅₀ value for mice exposed to ethylbenzene is 17.8 g/kg (RTECS, 1996). Lowest published lethal inhalation doses or concentrations of ethylbenzene in rats, mice, and guinea pigs are 4,000 ppm/4 hrs; 50 g/m³/2 hrs; and 10,000 ppm, respectively (RTECS, 1996). No adverse effects were reported in guinea pigs, rabbits, or monkeys exposed to ethylbenzene at 400 to 2,200 ppm, 7 to 8 hours per day, 5 days/week, for up to 6 months (Patty, 1963, as cited in NLM, 1996). Adverse impacts on fertility have been reported in female rats exposed to 97 ppm ethylbenzene for 7 hours for 15 days prior to mating (RTECS, 1996). An inhalation dose of 600 mg/m³ over a 24-hour period during the seventh to fifteenth day of pregnancy was also shown to adversely affect fertility in female rats (RTECS, 1996). Ethylbenzene has also been shown to be phytotoxic and teratogenic (RTECS, 1996).

Limited data exist on the concentration of ethylbenzene in aquatic biota. Bioconcentration of ethylbenzene by fish is expected to be insignificant (NLM, 1996). Experimentally determined bioconcentration factors for ethylbenzene in goldfish and clams are 79 and 4.68, respectively (Ogata et al., 1984, and Nunes and Benville, 1979, as cited in NLM, 1996). Oysters collected from Lake Pontchartrain, Louisiana, contained an average ethylbenzene concentration of 8 g/kg (Ferrario et al., 1985). Clams from the same site did not contain measurable amounts of ethylbenzene (Ferrario et al., 1985).

Data on the toxicity of ethylbenzene to freshwater biota are limited. Federal Water Quality Criteria do not exist for the protection of freshwater aquatic life from exposure to ethylbenzene (EPA, 1996). A value of 32,000 µg/L is, however, listed by EPA (1996) as the lowest effect level in freshwater environments. Suter and Mabrey (1994), however, recommend acute and chronic advisory values of 6,970 and 389 µg/L, respectively, for the protection of freshwater biota. Laboratory-determined 96-hour LC₅₀ values for bluegill sunfish (*Lepomis macrochirus*), fathead minnows (*Pimephales promelas*), and sheepshead minnows (*Cyprinodon variegatus*) are 32 mg/L (Pickering and Henderson, 1966, as cited in NLM, 1996), 12.1 to 48.5 mg/L (Pickering and Henderson, 1966, and Greiger et al., 1986, as cited in NLM, 1996), and 275 mg/L (EPA, 1978, as cited in NLM, 1996). Lowest chronic toxicity values of ethylbenzene to fish and daphnids are greater than 440 and 12,922 µg/L (estimated), respectively (Suter and Mabrey, 1994).

REFERENCES

Clayton, G. D. and F. E. Clayton (eds.), 1982, *Patty's Industrial Hygiene and Toxicology*, Vols. 2A, 2B, 2C, 3rd ed., John Wiley and Sons, New York, New York.

Ferrario, J. B., et al., 1985, *Bulletin of Environmental Contamination and Toxicology*, Vol. 34, pp. 246-255.

Friberg, L., G. F. Nordberg, E. Kessler, and V. B. Vouk (eds.), 1986, *Handbook of the Toxicology of Metals*, 2nd ed., Vol. II, Elsevier Science Publishers B.V., Amsterdam, Holland, 130 pp.

Graedel, T. E., et al., 1986, *Atmospheric Chemical Compounds*, Academy Press, New York, New York.

Greiger, D. L., S. H. Poirier, L. T. Brooke, and D. J. Call (eds.), 1986, *Acute Toxicities of Organic Chemicals to Fathead Minnows (*Pimephales promelas*)*, Vol. III, University of Wisconsin-Superior, Superior, Wisconsin, 189 pp.

International Labour Office (ILO), 1983, *Encyclopedia of Occupational Health and Safety*, Vols. I and II, International Labour Office, Geneva, Switzerland.

Lovegren, N. V., et al., 1979, *Journal of Agriculture, Food, and Chemistry*, Vol. 27, pp. 851-853.

National Library of Medicine (NLM), 1996, Hazardous Substances Data File, Toxicology Information Network (TOXNET).

Nunes, P., and P. E. Benville, 1979, *Bulletin of Environmental Contamination and Toxicology*, Vol. 21, pp. 71-74.

Ogata, M., et al., 1984, *Bulletin of Environmental Contamination and Toxicology*, Vol. 33, pp. 561-567.

Patty, F. (ed.), 1963, *Industrial Hygiene and Toxicology: Volume II: Toxicology*, 2nd ed., Interscience Publishers, New York, New York.

Pickering, O. H., and C. Henderson, 1966, *Journal of Water Pollution Control Federation* Vol. 38, pp. 1419.

Registry of Toxic Effects of Chemical Substances (RTECS), 1996, produced by Micromedex.

Sandmeyer, E.E., 1981, Aromatic Hydrocarbons. In Patty's Industrial Hygiene and Toxicology, George D. Clayton and Florence E. Clayton (Editors), Third Revised Edition, Volume 2B. New York, John Wiley & Sons.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

U.S. Environmental Protection Agency (EPA), 1996, "Integrated Risk Information System," on-line database, maintained by the U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency (EPA), 1985, Drinking Water Criteria Document for Ethylbenzene, Final Draft. Office of Health and Environmental Assessment, Washington, D.C., EPA/600/X-84/163-1.

U.S. Environmental Protection Agency (EPA), 1980, "Investigations of Selected Environmental Contaminants: Styrene, Ethylbenzene and Related Compounds," *EPA 560/11-80-018*, U.S. Environmental Protection Agency, Washington, D.C.

U.S. Environmental Protection Agency (EPA), 1978, "In-Depth Studies on Health and Environmental Impacts of Selected Water Pollutants," *EPA No. 68-01-4646*, U.S. Environmental Protection Agency. Washington, D.C.

IRON

Human Health Effects

Iron is a necessary component of many proteins, including hemoglobin, myoglobin, and enzymes such as the catalases, the cytochromes and peroxidases (Spivey Fox and Rader, 1988). Its biological role derives from its ability to undergo reversible oxidation-reduction reactions. About 67% of the 3-5 g of iron in the body is bound to hemoglobin, »10% is bound to myoglobin and various enzymes and the remainder is bound to ferritin and hemosiderin, which are storage proteins for iron (Goyer, 1991).

Iron is an essential trace element (NRC, 1989). The current RDAs are: 6 mg/day for infants 0-0.5 years of age, 10 mg/day for infants 0.5-1 years, for children 1-10 years, for adult males 19-50 years, and for adults of either sex greater than 51 years, 12 mg/day for males 11-18 years, 15 mg/day for females 11-50 years, and 30 mg/day for pregnant women (NRC, 1989). The primary physiological concern regarding iron is iron deficiency anemia, which may result from inadequate intake or excessive blood loss (Finch, 1980). Average daily intakes for eight age-sex groups in the U.S. for 1982-1989, based on a survey of core foods in the U.S. food supply, ranged from 8.9-15.1 mg/day (71-162% of the RDA), but the distributions about the averages were not reported (Pennington and Young, 1991).

Iron homeostasis of the body is controlled by regulating the active transport mechanisms involved in GI absorption (Goyer, 1991; Knoebel, 1971; Spivey Fox and Rader, 1988). Generally, »2-15% of dietary iron is absorbed. GI absorption of iron increases when body stores, i.e., when intestinal mucosal stores of ferritin iron, are low, and decreases when body stores are ample. In cases of iron-deficiency anemia, as much as 40% of the iron in nutritional supplements may be absorbed (Finch, 1980). Other factors that influence GI uptake of iron are age, the chemical form of iron in the diet (the ferrous form is more readily absorbed), and other dietary factors. High dietary levels of phosphate, cobalt, copper or zinc depress GI uptake. Ascorbic acid and other organic acids increase GI absorption of iron.

When luxury amounts of iron are available, hepatic ferritin formation is increased (Goyer, 1991). In the case of frank iron overload, ferritin is converted to hemosiderin,

which is a more stable and less available storage form. Both ferritin and hemosiderin protect the cells from the toxicity of excess iron by maintaining the iron in a bound form.

Both acute and chronic toxicity syndromes occur from the ingestion of excess iron (Goyer, 1991). The acute form usually involves the accidental ingestion of iron-containing medicines, often candy-coated tablets, by children 1-5 years old. Ingestion of greater than 500 mg of iron (2,500 mg ferrous sulfate) leads to vomiting, severe ulceration of the GI tract, metabolic acidosis and shock, liver damage, and blood coagulation defects (Finch, 1980). Sequelae may include renal failure and liver cirrhosis. Doses of 1,000-2,000 mg may cause death.

Chronic iron toxicosis, known as hemochromatosis, may result from a congenital defect that increases iron absorption from the gut (Goyer, 1991). High dietary concentrations, or excess ingestion of tonics or medicines containing iron, may contribute to iron overload, but the dose required to induce disease was not reported. The disease is characterized by hemosiderin deposits in soft tissues, which may interfere with liver function, induce diabetes mellitus or other endocrinologic dysfunction, or damage the heart. At the cellular level, lipid peroxidation is increased, which results in damage to the membranes of intracellular organelles. The usual oral dose of iron to treat iron deficiency or blood-loss anemia, 200 mg, was associated with a low level of disturbances of the GI tract, including nausea, upper abdominal pain, and constipation or diarrhea, but was not associated with iron overload (Finch, 1980).

There are no verified or provisional toxicity values or primary (health-based) drinking water quality criteria for iron (EPA, 1996a). WHO (1984) recommended a drinking water quality guideline of 0.3 mg/L to prevent precipitation of ferric hydroxide, which settles out as a rust-colored silt.

It would be inappropriate to develop a health-based toxicity value from the usual 200 mg/day oral dose of iron used to treat iron-deficiency anemia, because the patients for whom this dose is intended represent a subpopulation with altered health state and iron homeostasis. Although excessive iron intake can induce hemochromatosis, the intake required to induce disease was not quantified (Goyer, 1991). Underwood (1977),

however, reported that daily doses of 25-75 mg, or even much greater, have been consumed without harmful effects.

A chronic oral RfD may be derived for iron from the dose of 75 mg/day, the high end of the range of daily intake consumed without adverse effect (Underwood, 1977), which is a NOAEL for normal humans. An uncertainty factor of 1, applied to the NOAEL of 75 mg/day, yields an RfD of 75 mg/day, or 1 mg/kg-day. The uncertainty factor of 1 is appropriate for a NOAEL in normal humans. Presumably, the most sensitive subpopulation consists of individuals with congenital hemochromatosis. No uncertainty factor is applied to protect these individuals, however, because they represent a group with altered physiological state who would suffer disease from daily intakes in the range of normal dietary amounts. Application of an uncertainty factor of 10 to provide protection for sensitive individuals would result in an RfD below the current RDA.

Ecological Effects

Iron is a metal in Group VIII of the periodic table (Budavari, 1989; Sax and Lewis, 1987). It is the second most abundant metal, comprising about 5% of the earth's crust. Chemically, iron is a strong reducing agent. Because of its reactivity, it occurs primarily as oxides in ores. In water, it generally occurs in the divalent or trivalent state (WHO, 1984). Fully aerated water generally contained <0.5 mg iron/L (van der Leeden et al., 1991). Ground water with a pH <8.0 may contain 10 mg/L, or rarely as much as 50 mg/L. Acid water from thermal springs, mine wastes or industrial wastes may contain >6,000 mg/L. Concentrations in finished public water supplies in the 100 largest cities in the U.S. ranged from 0.002-1.7 mg/L, with a median of 0.043 mg/L. The use of iron or steel distribution pipes, and the use of iron salts in the production of potable water may contribute to iron in drinking water (WHO, 1984).

The USEPA benchmark and the Ohio EPA Warmwater Habitat Water Quality Criteria for iron is 1.0 mg/L (EPA, 1996b).

REFERENCES

Budavari, S., ed., 1989, The Merck Index, An Encyclopedia of Chemicals, Drugs, and Biologicals., Eleventh Edition, Merck and Co., Inc., Rahway, NJ.

Finch, C.A., 1980, Drugs Effective in Iron-Deficiency Anemia and Other Hypochromic Anemias. In: Gilman, A.G., L.S. Goodman and A. Gilman, Eds. Goodman and Gilman's The Pharmacological Basis of Therapeutics. Sixth Edition. New York: Macmillan Publishing Co. pp. 1315-1330.

Goyer, R. A., 1991, Toxic Effects of Metals, Casarett and Doull's Toxicology, the Basic Science of Poisons, M. O. Amdur, J. Doull, and C. D. Klaassen, eds., 4th ed., Pergamon Press, New York.

Knoebel, L.K, 1971, Secretion and Action of Digestive Juices: Absorption. In: Selkurt, E.E., Ed. Physiology. Third Edition. Boston: Little, Brown and Co. pp. 599-634.

National Research Council (NRC), 1989, Recommended Dietary Allowances, 10th Edition. Washington, DC: National Academy Press.

Pennington, J.A.T. and B.E. Young, 1991, Total diet study nutritional elements, 1982-1989. J. Am. Dietetic Assoc. 91: 179-183NN.

Sax, N. I., and R. J. Lewis, Sr., eds., 1987, Hawley's Condensed Chemical Dictionary, 11th ed., Van Nostrand Reinhold Co., New York.

Spivey Fox, M.R. and J.I Rader, 1988, Iron. In: Seiler, H.G. and H. Sigel, Eds. Handbook on Toxicity of Inorganic Compounds. New York: Marcel Dekker, Inc. pp. 345-354.

Underwood, E.J.; 1977, Trace Elements in Human and Animal Nutrition. Fourth Edition. New York: Academic Press.

U.S. Environmental Protection Agency (EPA), 1996a, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996b, Ecotox Thresholds, EPA 540/F-95/038.

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van der Leeden, F., F.L. Troise and D.K. Todd., Eds., 1991, The Water Encyclopedia. Second Edition. Chelsea, MI: Lewis Publishers, Inc. pp. 422, 445.

World Health Organization (WHO), 1984, Guidelines for Drinking Water Quality. Volume 2. Health Criteria and Other Supporting Information. WHO, Geneva.

LEAD

Human Health Effects

Studies in humans indicate that an average of 10 percent of ingested lead is absorbed, but estimates as high as 40 percent were obtained in some individuals (Tsuchiya, 1986). Nutritional factors have a profound effect on GI absorption efficiency. Children absorb ingested lead more efficiently than adults; absorption efficiencies up to 53 percent were recorded for children three months to eight years of age. Similar results were obtained for laboratory animals; absorption efficiencies of 5 to 10 percent were obtained for adults and 50 percent were obtained for young animals. The deposition rate of inhaled lead averages approximately 30 to 50 percent, depending on particle size, with as much as 60 percent deposition of very small particles ($0.03 \mu\text{m}$) near highways. All lead deposited in the lungs is eventually absorbed.

Approximately 95 percent of the lead in the blood is located in the erythrocytes (EPA, 1990). Lead in the plasma exchanges with several body compartments, including the internal organs, bone, and several excretory pathways. In humans, lead concentrations in bone increase with age (Tsuchiya, 1986). About 90 percent of the body burden of lead is located in the skeleton. Neonatal blood concentrations are about 85 percent of maternal concentrations (EPA, 1990). Excretion of absorbed lead is principally through the urine, although GI secretion, biliary excretion, and loss through hair, nails, and sweat are also significant.

The noncancer toxicity of lead to humans has been well characterized through decades of medical observation and scientific research (EPA, 1996a). The principal effects of acute oral exposure are colic with diffuse paroxysmal abdominal pain (probably due to vagal irritation), anemia, and, in severe cases, acute encephalopathy, particularly in children (Tsuchiya, 1986). The primary effects of long-term exposures are neurological and hematological. Limited occupational data indicate that long-term exposure to lead may induce kidney damage. The principal target organs of lead toxicity are the erythrocyte and the nervous system. Some of the effects on the blood, particularly changes in levels of certain blood enzymes, and subtle neurobehavioral changes in children, appear to occur at levels so low as to be considered nonthreshold effects.

EPA (1996a) presents no inhalation RfC for lead, but referred to the National Ambient Air Quality Standard (NAAQS) for lead, which could be used in lieu of an inhalation RfC. The NAAQSs are based solely on human health considerations and are designed to protect the most sensitive subgroup of the human population. The NAAQS for lead is $1.5 \mu\text{g}/\text{m}^3$, averaged quarterly (EPA, 1996a). The NAAQS is equivalent to $0.00043 \text{ mg}/\text{kg}/\text{day}$, assuming a body weight of 70 kg and an inhalation rate of $20 \text{ m}^3/\text{day}$.

The EPA (1990) determined that it is inappropriate to derive an RfD for oral exposure to lead for several reasons. First, the use of an RfD assumes that a threshold for toxicity exists, below which adverse effects are not expected to occur; however, the most sensitive effects of lead exposure, impaired neurobehavioral development in children and altered blood enzyme levels associated with anemia, may occur at blood lead concentrations so low as to be considered practically nonthreshold in nature. Second, RfD values are specific for the route of exposure for which they are derived. Lead, however, is ubiquitous, so that exposure occurs from virtually all media and by all pathways simultaneously, making it practically impossible to quantify the contribution to blood lead from any one route of exposure. Finally, the dose-response relationships common to many toxicants, and upon which derivation of an RfD is based, do not hold true for lead. This is because the fate of lead within the body depends, in part, on the amount and rate of previous exposures, the age of the recipient, and the rate of exposure. There is, however, a reasonably good correlation between blood lead concentration and effect. Therefore, blood lead concentration is the appropriate parameter on which to base the regulation of lead.

The EPA's Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children, version 0.99d, is an iterative set of equations that estimate blood lead concentrations in children aged 0 to 7 years (EPA, 1994a). The biokinetic part of the model describes the movement of lead between the plasma and several body compartments and estimates the resultant blood concentration. The rate of the movement of lead between the plasma and each compartment is a function of the transition or residence time (i.e., the mean time for lead to leave the plasma and enter a given compartment, or the mean residence time for lead in that compartment). Compartments modeled include the erythrocytes, liver, kidneys, all the other soft tissue of the body, cortical bone, and trabecular bone. Excretory pathways and their rates are

also modeled. These include the mean time for excretion from the plasma to the urine, from the liver to the bile, and from the other soft tissues to the hair, skin, sweat, etc. The model permits the user to adjust the transition and residence times. EPA guidance (EPA, 1994b) establishes a screening level of 400 ppm for lead in soil at Superfund sites. This concentration is considered by EPA to be protective for direct contact with lead-contaminated soil in a residential setting.

EPA (1996a) classifies lead in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate evidence of cancer in humans and sufficient animals evidence. The human data consist of several epidemiological occupational studies that yielded confusing results. All of the studies lacked quantitative exposure data and failed to control for smoking and concomitant exposure to other possibly carcinogenic metals. Rat and mouse bioassays showed statistically significant increases in renal tumors following dietary and subcutaneous exposure to several soluble lead salts. Various lead compounds were observed to induce chromosomal alterations in vivo and in vitro, sister chromatid exchange in exposed workers, and cell transformation in Syrian hamster embryo cells; to enhance simian adenovirus induction; and to alter molecular processes that regulate gene expression. EPA (1996a) declined to estimate risk for oral exposure to lead because many actors (e.g., age, general health, nutritional status, existing body burden and duration of exposure) influence the bioavailability of ingested lead, introducing a great deal of uncertainty into any estimate of risk.

Ecological Effects

Although lead is not an essential nutrient for plant growth, it is detected in plant tissues due to the prevalence of lead in the environment. The bioavailability of lead in soil to plants is limited. It may, however, be enhanced by a reduction in soil pH, a reduction in the content of organic matter and inorganic colloids in the soil, a reduction in iron oxide and phosphorus content, and by increased amounts of lead in soils (NRCC, 1973). Plants can absorb lead from soil and air. Aerial deposition of lead can also contribute significantly to the concentration of lead in aboveground plant parts. Lead is believed to be the metal of least bioavailability and the most highly accumulated metal in root tissues (Kabata-Pendias and Pendias, 1992). Mean background concentrations of lead in grasses and clovers have been reported to range from 2.1 mg/kg (dry weight)

to 2.5 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Older plant parts contain higher concentrations of lead than younger parts (Bunzl and Kracke, 1984).

Adverse effects of lead on terrestrial plants occur only at total concentrations of several hundred mg/kg of soil (Eisler, 1988). This is explained by the fact that, in most cases, lead is tightly bound to soils, and substantial amounts must accumulate before it can affect the growth of higher plants (Boggess, 1977). Some species of plants have the ability to tolerate or adapt to high concentrations of lead. Grass shoots growing near a lead smelter were reported by de Konig (1974) to contain lead concentrations ranging from 229 to 2,714 mg/kg (dry weight). Concentrations in leaf tissue that are excessive or toxic to various plant species range from 30 to 300 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 50 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for phytotoxicity in soils. General symptoms of lead toxicity include the wilting of older leaves; the presence of dark green leaves; stunted leaf growth; and the presence of brown, short roots (Kabata-Pendias and Pendias, 1992).

As with plants, lead is not considered an essential nutrient for mammalian or avian life. It is commonly detected in wildlife with background concentrations of lead in whole bodies of small mammals collected from several locations reported to range from 1 to 7 mg/kg (dry weight) (Eisler, 1988). The highest concentrations of lead reported in wild species were in animals from urban areas with heavy vehicular traffic or areas near lead smelters or mines (Eisler, 1988). Ingestion of spent lead shot has resulted in elevated lead levels in waterfowl. Ingestion is the major route of exposure for wildlife. Lead tends to accumulate in bone, hair, teeth, and feathers. Biomagnification of lead is negligible.

In general, organic lead compounds are more toxic than the inorganic forms. Trialkyl lead salts are 10 to 100 times more toxic to birds than are the inorganic salts (Forsyth et al., 1985). It has been shown in mammals that tetramethyl lead is approximately seven times more toxic than tetraethyl lead, both of which induce toxic effects earlier than inorganic lead compounds (Eisler, 1988). As in humans, immature organisms are more sensitive to lead than adult organisms (Eisler, 1988).

Concentrations of lead that are toxic to sensitive mammalian species have been summarized by Eisler (1988). Individual survival was reported as being reduced at acute oral doses of lead as low as 5 mg/kg body weight in rats, at a chronic dose of 0.3 mg/kg body weight in dogs, and at a dietary level of 1.7 mg/kg body weight in the horse (Eisler, 1988). The no-effect level of lead intake for sheep is about 0.1 mg/kg/d (Booth and McDonald, 1982, as cited in NLM, 1996). Examples of extrapolated NOAELs for chronic exposure of various mammalian wildlife species to lead acetate are 19.9 mg/kg/d for the white-footed mouse, 5.33 mg/kg/d for the cottontail rabbit, and 3.44 mg/kg/d for the red fox (Opresko et al., 1994). Drinking water NOAELs for various mammalian wildlife were reported to range from 22.8 to 178 mg/L (Opresko et al., 1994). Symptoms of lead poisoning in mammals are diverse and depend on the form of lead ingested, the concentration, and on the species and its age. These symptoms may include reproductive impairment, decreased body weight, vomiting, uncoordinated body movements, visual impairment, reduced life span, renal disorders, and abnormal social behavior (Eisler, 1988). Lead can cross the placenta and result in stillbirths and skeletal deformities (Eisler, 1988).

Toxicity of lead to birds is dependent upon the form of lead, the route of exposure and exposure duration, and the species and age of the bird. Hatchlings of chickens, Japanese quail, mallards, and pheasants are relatively tolerant to moderate lead exposure (Eisler, 1988). Chickens fed diets containing lead in the form of lead acetate at 1,850 mg/kg for four weeks did not experience death or severe clinical hematological effects (Franson and Custer, 1982). Growth rate, however, was suppressed by 47 percent. No effect on growth or survival was reported in juvenile American kestrels exposed to dietary lead levels of 500 and 2,000 mg/kg, respectively (Hoffman et al., 1985a; Hoffman et al., 1985b). LD₅₀ values for birds given a single oral dose of tetraethyllead were 107 mg/kg body weight for the mallard and 24.6 mg/kg body weight for the Japanese quail (Hudson et al., 1984). No adverse effects were observed in birds fed diets containing lead at 100 mg/kg in the form of lead nitrate over a 12-week period (Finley et al., 1976). Based on toxicity data specific to American kestrels exposed orally to metallic lead, Opresko et al. (1994) estimated NOAELs for the great blue heron and red-tailed hawk to be 1.47 and 1.89 mg/kg/d, respectively. The drinking water NOAEL for these birds was estimated as 33 mg/L. Lead-poisoned birds

may exhibit external symptoms such as loss of appetite, lethargy, emaciation, tremors, drooping wings, green liquid feces, and abnormal motor skills (Eisler, 1988).

Highest recorded concentrations of lead in freshwater biota have been in areas located near industrialized and urban areas, from ponds containing large quantities of lead shot, in ponds near lead mines (Eisler, 1988). Lead concentrations are usually highest in algae, benthic invertebrates, and shellfish. Bioconcentration factors for freshwater biota are discussed in Eisler (1988). High bioconcentration factors for aquatic biota such as algae are attributed in part to the adsorption of lead onto the surface of the organism (Demayo et al., 1982). The bioconcentration factor for lead in aquatic insects is approximately 500 (EPA, 1985, as cited in Eisler, 1988). Sediments serve as sinks for lead. As a result, submergent aquatic plants and benthic invertebrates may be exposed to higher lead concentrations than organisms in the water column. No significant biomagnification of lead occurs in aquatic ecosystems (Bogges, 1977). Background concentrations of lead in fish tend to be less than 1 mg/kg (dry weight) (Eisler, 1988).

The EPA's National Ambient Water Quality Criteria for lead in freshwater is 82 $\mu\text{g/L}$ for acute exposure and 3.2 $\mu\text{g/L}$ for chronic exposure of aquatic life to lead (based on a water hardness of 100 mg/L) (EPA, 1996a). The USEPA ecotox threshold for lead is 0.0025 mg/L (EPA, 1996b). The Ohio Warmwater Habitat Water Quality Criteria for lead is set at 0.00285. Because the toxicity of lead to aquatic organisms is affected by water hardness, all water-quality criteria must be adjusted for with site-specific water hardness data. The lowest chronic values of lead reported in the literature for fish and *Daphnia* are 18.88 and 12.26 $\mu\text{g/L}$, respectively (Suter and Mabrey, 1994). The test EC_{20} for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). This value is 22 $\mu\text{g/L}$ for lead (Suter and Mabrey, 1994). The 30-day LC_{50} value for adult leopard frogs (*Rana pipiens*) exposed to lead is 105 mg/L, with deaths noted at a concentration of 25 mg/L (EPA, 1985). Delays in metamorphosis times have been reported in tadpoles (*Rana utricularia*) exposed to lead concentrations of 0.5 mg/L (Yeung, 1978).

In general, dissolved lead is more toxic than total lead, and organic forms of lead are more toxic than inorganic forms. Soluble lead in the water column becomes less bioavailable as water hardness increases. The toxicity of lead to fish may also be influenced by calcium concentrations in the environment (Varanasi and Gmur, 1978). Chronic exposure of fish to lead may result in signs of lead poisoning, such as spinal curvature, anemia, darkening of the dorsal tail region, destruction of spinal neurons, difficulties in swimming, growth inhibition, changes in blood chemistry, retarded sexual development, and death (Eisler, 1988). It has been reported that freshwater isopods may develop a tolerance to lead (Fraser, 1980).

REFERENCES

Boggess, W. R. (ed.), 1977, "Lead in the Environment," *National Science Foundation Report NSF/RA-770214*, National Science Foundation, Washington, D.C., 272 pp.

Booth, N. H., and L. E. McDonald (eds.), 1982, *Veterinary Pharmacology and Therapeutics*, 5th ed., Iowa State University Press, Ames, Iowa.

Bunzl, K. and W. Kracke, 1984, "Distribution of Pb-210, Po-210, Stable Lead and Fallout Cs-137 in Soil, Plants and Moorland Sheep of a Heath," *Science of the Total Environment*, Vol. 39, pp. 143-159.

de Konig, H. W., 1974, "Lead and Cadmium Contamination in the Area Immediately Surrounding a Lead Smelter," *Water, Air and Soil Pollution*, Vol. 3, p. 63.

Demayo, A., M. C. Taylor, K. W. Taylor, and P. V. Hodson, 1982, "Toxic Effects of Lead and Lead Compounds on Human Health, Aquatic Life, Wildlife, Plants, and Livestock," *CRC Critical Reviews in Environmental Control*, Vol. 12, pp. 257-305.

Eisler, R., 1988a, "Arsenic Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review," U.S. Fish and Wildlife Service Contaminant Hazard Review, *Report No. 12*, U.S. Department of the Interior, Washington, D.C.

Finley, M. T., M. P. Dieter, and L. N. Locke, 1976, "Sublethal Effects of Chronic Lead Ingestion In Mallard Ducks," *Journal of Toxicology and Environmental Health*, Vol. 1, pp. 929-937.

Forsyth, D. S., W. D. Marshall, and M. C. Collete, 1985, "Interaction of Alkyllead Salts with Avian Eggs," *Journal of Environmental Science and Health*, Vol. 20A, pp. 177-191.

Franson, J. C., and T. W. Custer, 1982, "Toxicity of Dietary Lead in Young Cockerels," *Veterinary and Human Toxicology*, Vol. 24, pp. 421-423.

Fraser, J., 1980, "Acclimation to Lead in the Freshwater Isopod *Asellus aquaticus*," *Oecologia*, Vol. 45, pp. 419-420.

Hoffman, D. J., J. C. Franson, O. H. Pattee, C. M. Bunck, and A. Anderson, 1985a, "Survival, Growth, and Accumulation of Ingested Lead in Nestling American Kestrels (*Falco sparverius*)," *Archives of Environmental Contamination and Toxicology*, Vol. 14, pp. 89-94.

Hoffman, D. J., J. C. Franson, O. H. Pattee, C. M. Bunck, and H. C. Murray, 1985b, "Biochemical and Hematological Effects of Lead Ingestion in Nestling American

Kestrel (*Falco sparverius*)," *Comparative Biochemical Physiology*, Vol. 80C, pp. 431-439.

Hudson, R. H., R. K. Tucker, and M. Haegele, 1984, "Handbook of Toxicity of Pesticides to Wildlife," *U.S. Fish Wildlife Service Resource Publication*, Vol. 153, U.S. Fish and Wildlife Service, Washington, D.C., 90 pp.

Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.

National Library of Medicine (NLM), 1996, "Hazardous Substance Data Bank," produced by Micromedex, Inc.

National Research Council Canada (NRCC), 1973, "Lead in the Canadian Environment," *Publ. BY73-7(ES)*, National Research Council, Canada, 116 pp.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/RI*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/RI*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Tsuchiya, K., 1986, Lead, *Handbook on the Toxicology of Metals*, Friberg, L., G. F. Nordberg, and V. B. Vouk, eds., Vol. II, 2nd ed., Elsevier Science Publishers B. V., New York, pp. 298-353.

U.S. Environmental Protection Agency (EPA), 1996a, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996b, Ecotox Thresholds, EPA 540/F-95/038.

U.S. Environmental Protection Agency (EPA), 1994a, Uptake/Biokinetic Model, Version 0.99. Office of Health and Environmental Assessment, Cincinnati, OH.

U.S. Environmental Protection Agency (EPA), 1994b, Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. Office of Solid Waste and Emergency Response, Washington, DC. (OSWER Directive #9355.4-12) EPA/540/F-94/043.

U.S. Environmental Protection Agency (EPA), 1990, Technical Support Document for Lead, Prepared by the Chemical Hazard Assessment Division, Syracuse Research Corporation, under contract to the Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. Environmental Protection Agency (EPA), 1985, "Ambient Water Quality Criteria for Lead-1984," *Report 440/5-80-057*, U.S. Environmental Protection Agency, Washington, D.C., 151 pp.

Varanasi, U., and D. J. Gmur, 1978, "Influence of Water-Borne and Dietary Calcium on Uptake and Retention of Lead by Coho Salmon (*Oncorhynchus kisutch*)," *Toxicology and Applied Pharmacology*, Vol. 46, pp. 65-75.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Yeung, G. L., 1978, "The Influence of Lead, an Environmental Pollutant on Metamorphosis of *Rana utricularia* (Amphibia: Ranidae)," *Arkansas Academy of Sciences Proceedings*, Vol. 32, Arkansas, pp. 83-86.

MAGNESIUM

Human Health Effects

Magnesium is an alkaline earth metal in Group IIA of the periodic table (Sax and Lewis, 1987). It comprises about 2.1% of the earth's crust by weight, and occurs in nature in the combined form, particularly in magnesite, dolomite, sea water and brines (Budavari, 1989). Concentrations in natural water were several hundred mg/L in some western U.S. streams (van der Leeden et al., 1991). Ocean water contains >1,000 mg/L, and brines may contain up to 57,000 mg/L. Concentrations in finished public water supplies in the 100 largest cities in the U.S. ranged from 0-120 mg/L, with a median of 6.25 mg/L.

Magnesium is a nutritionally essential element required for many enzyme systems, particularly those involved in phosphate transfer, and for proper functioning of the neuromuscular and cardiovascular systems (Mudge, 1980; Selkurt, 1971). The average adult body contains approximately 2000 mEq (24 g), 50% of which is located in the skeleton. The average U.S. adult ingests 20-40 mEq (240-490 mg)/day, and absorbs about one third of the ingested amount, probably by an active transport system identical with or closely related to that which effects absorption of calcium. The extent of absorption appears to increase in cases of increased requirement (Birch, 1988). The current RDAs are 40-60 mg/day for infants up to one year of age, 80-170 mg/day for children 1-10 years, 270-400 mg/day for children 11-18 years, and 280-350 mg/day for adults (NRC, 1989).

Magnesium salts are used as saline cathartics and gastric antacids (Birch, 1988; Fingle, 1980; Harvey, 1980). When used as cathartics, the usual doses are 15 g of magnesium sulfate (approximately 3 g magnesium) or 40-160 mEq (0.5-2 g) magnesium from magnesium hydroxide (milk of magnesia).

Hypermagnesemia may result from consumption of very large quantities of magnesium by persons with renal failure (Birch, 1988). Ingestion of large doses of magnesium usually induces vomiting in humans, which limits the toxic hazard. Symptoms of hypermagnesemia include muscle weakness, hypotension, electrocardiographic changes, sedation, confusion, and possibly loss of deep tendon reflexes and respiratory arrest.

When used as a laxative, as little as 5 g magnesium sulfate (1 g magnesium) can induce a significant laxative effect (Fingle, 1980). Prolonged use of magnesium hydroxide as an antacid may rarely cause fecal stones composed of magnesium carbonate and magnesium hydroxide (Harvey, 1980).

There are no verified or provisional toxicity values or water quality criteria for magnesium (EPA, 1996a). Intake of a single 1 g dose of magnesium may induce a significant laxative effect (Mudge, 1980). Prolonged high intake may induce hypermagnesemia, but only in persons in whom excretion of magnesium is compromised because of kidney damage, or may induce the formation of fecal stones (Harvey, 1980; Mudge, 1980), but the dose associated with these effects is not known.

In the absence of quantitative chronic toxicity data, the high end of the range of normal dietary values, 490 mg/day (Mudge, 1980), may be considered a NOAEL for normal humans and selected as the basis for an RfD for chronic oral exposure to magnesium. Application of an uncertainty factor of 1 yields an RfD of 490 mg/day, or 7 mg/kg-day. The uncertainty factor of 1 is appropriate for a NOAEL in normal humans. Presumably, the most sensitive subpopulation consists of individuals with renal failure. No uncertainty factor is applied to protect these individuals, however, because application of an uncertainty factor of 10 would yield an RfD below the RDA for magnesium.

Ecological Effects

There are no USEPA benchmarks (EPA, 1996b) or USEPA Warmwater Habitat Water Quality Criteria for the protection of freshwater biota against exposure to magnesium.

REFERENCES

Birch, N.J, 1988, In: Seiler, H.G. and H. Sigel, Eds. Handbook on Toxicity of Inorganic Compounds. New York: Marcel Dekker, Inc.

Budavari, S., ed., 1989, The Merck Index, An Encyclopedia of Chemicals, Drugs, and Biologicals., Eleventh Edition, Merck and Co., Inc., Rahway, NJ.

Fingle, E, 1980, Laxatives and Cathartics. In: Gilman, A.G., L.S. Goodman and A. Gilman, Eds. Goodman and Gilman's The Pharmacological Basis of Therapeutics. Sixth Edition. New York: Macmillan Publishing Co. pp. 1002-1012.

Harvey, S.C, 1980, Gastric Antacids and Digestants. In: Gilman, A.G., L.S. Goodman and A. Gilman, Eds. Goodman and Gilman's The Pharmacological Basis of Therapeutics. Sixth Edition. New York: Macmillan Publishing Co. pp.988-1001.

Mudge, G.H, 1980, In: Gilman, A.G., L.S. Goodman and A. Gilman, Eds. Goodman and Gilman's The Pharmacological Basis of Therapeutics. Sixth Edition. New York: Macmillan Publishing Co.

National Research Council (NRC), 1989, Recommended Dietary Allowances, 10th Edition. Washington, DC: National Academy Press.

Sax, N. I., and R. J. Lewis, Sr., eds., 1987, Hawley's Condensed Chemical Dictionary, 11th ed., Van Nostrand Reinhold Co., New York.

U.S. Environmental Protection Agency (EPA), 1996a, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996b, Ecotox Thresholds, EPA 540/F-95/038.

van der Leeden, F., F.L. Troise and D.K. Todd., Eds., 1991, The Water Encyclopedia. Second Edition. Chelsea, MI: Lewis Publishers, Inc. pp. 422, 445.

MANGANESE

Human Health Effects

Manganese is a nutritionally essential element (Saric, 1986). Its absorption from the GI tract is homeostatically controlled. Absorption of manganese from the GI tract of healthy humans was measured at 3 percent of a single 200 mg oral dose. Human epidemiological data suggest that manganese in drinking water is somewhat more bioavailable than manganese in the diet (EPA, 1996a). In humans suffering from manganese toxicity or anemia, GI absorption was measured at 4 and 7.5 percent, respectively. The 3 percent GI absorption estimate is considered sufficiently conservative and well documented to use in estimating a dermal RfD from an oral RfD. Sufficient data were not located for estimating respiratory tract or dermal uptake of manganese.

Distribution of absorbed manganese is first to the liver, and then to other tissues (Saric, 1986). Although no tissue accumulates large amounts, highest concentrations of manganese in humans are located in the liver, kidney, endocrine glands, and the intestines. The principal route of excretion is through the feces, in part due to biliary and pancreatic secretion. Urinary excretion and loss through sweat, hair, and lactation also occur.

Humans exposed to approximately 0.8 mg manganese per kg-day in drinking water exhibited lethargy, mental disturbances (1/16 committed suicide), and other neurologic effects. The elderly appear to be more sensitive than children. Oral treatment of laboratory rodents induces biochemical changes in the brain, but rodents do not exhibit the neurological signs exhibited by humans. Occupational exposure to high concentrations in air induces a generally typical spectrum of neurological effects, and increased incidence of pneumonia (ACGIH, 1991).

EPA (1996a) derived separate verified RfD values for chronic oral exposure to manganese in drinking water and in the diet, reflecting the presumption of greater bioavailability of manganese from drinking water. The chronic oral RfD for ingestion of manganese in drinking water is 0.005 mg/kg-day, based on an NOAEL of 0.005 mg/kg-day and an LOAEL of 0.06 mg/kg-day associated with neurological impairment

in a human epidemiology study. The elderly appeared to be more severely affected than children or younger adults. An uncertainty factor of 1 was used. A chronic oral RfD of 0.14 mg/kg-day was based on studies of dietary intake in humans. The intake of 0.14 mg/kg-day was considered an NOAEL; an uncertainty factor of 1 was used. EPA (1996) presents a verified chronic inhalation RfC of 0.0004 mg/m³ based on an LOAEL for respiratory symptoms and psychomotor disturbances in occupationally exposed humans and an uncertainty factor of 300. The inhalation RfC is equivalent to 0.00011 mg/kg-day, assuming humans inhale 20 m³ of air/day and weight 70 kg. The central nervous system (CNS) and respiratory tract are target organs of inhalation exposure to manganese.

EPA (1996a) classifies manganese in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans). Quantitative cancer risk estimates are not derived for Group D chemicals.

Ecological Effects

Manganese is an essential element for plant growth. Uptake of manganese may occur via root or foliar uptake (Kabata-Pendias and Pendias, 1992). Background concentrations of manganese in immature grasses collected in the United States are reported to range from 20 to 665 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). The concentration of manganese in plants is dependent upon plant and soil characteristics. Plants grown on flooded or acid soils tend to contain higher concentrations of manganese than plants grown in other, uncontaminated soil types (Kabata-Pendias and Pendias, 1992). In addition, concentrations of manganese in plants are positively correlated with soil organic matter (Kabata-Pendias and Pendias, 1992). Biological and geochemical interactions of manganese with other metals can also alter the amount of available manganese (Kabata-Pendias and Pendias, 1992).

Concentrations of manganese in leaf tissue that are excessive or toxic to various plant species, with the exclusion of very sensitive and highly tolerant species, range from 400 to 1,000 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 500 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for manganese phytotoxicity. General symptoms of manganese toxicity in plants include the presence of chlorosis and necrotic lesions on old leaves, blackish-

brown or red necrotic spots, dried leaf tips, and stunted root and plant growth (Kabata-Pendias and Pendias, 1992).

Manganese is an essential nutrient that is homeostatically regulated in vertebrates (Schroeder et al., 1966, as cited in Vanderploeg et al., 1975). Data on background concentrations of manganese in mammalian and avian wildlife are limited. Beardsley et al. (1978) reported kidney and liver tissues of field voles (*Microtus agrestis*) collected from a reference location to contain mean manganese concentrations of 6 and 8 mg/kg (dry weight), respectively. Liver and kidney tissues generally contain the highest concentrations of manganese in the body (Gregus and Klaassen, 1986). Manganese in the body is primarily excreted in feces (Gregus and Klaassen, 1986).

Divalent manganese is more toxic than trivalent manganese in mammals. Exposure to manganese dust via inhalation is usually of greater toxicological concern than ingestion of manganese (Hammond and Beliles, 1980). Based on an oral NOAEL of 88 mg/kg/d for rats exposed to manganese oxide, extrapolated NOAELs for chronic oral exposure of various mammalian wildlife species to manganese were estimated to be 219 mg/kg/d for the white-footed mouse, 58.6 mg/kg/d for the cottontail rabbit, and 37.9 mg/kg/d for the red fox (Opresko et al., 1994). Calculated chronic drinking-water NOAELs for wildlife are 731 mg/L for the white-footed mouse, 606 mg/L for the cottontail rabbit, and 449 mg/L for the red fox (Opresko et al., 1994). Laboratory studies with rats have found no hematologic, behavioral, or histologic effects in animals exposed to manganese dioxide at concentrations of 47 mg/cubic meter (m³) for five hours a day, five days a week for 100 days (Martone, 1964, as cited in Hammond and Beliles, 1980). Concentrations of manganese in the brain of the rats did, however, increase by fourfold.

As mentioned earlier, manganese is a required nutrient for plant and animal life. Manganese concentrations in most invertebrates are homeostatically controlled (Schroeder et al., 1966, as cited in Vanderploeg et al., 1975). Concentrations of manganese in Sphagnum mosses collected from northern Canada were reported to range from 39 to 389 mg/kg (Glooschenko and Capobianco, 1978, as cited in Leland et al., 1979). Bioconcentration factors for freshwater macrophytes have been reported to range from 190 to approximately 25,000 (Vanderploeg et al., 1975). With regard to

freshwater fish, concentrations of manganese in fish muscle are generally less than 0.5 mg/kg and range from 3 to 10 mg/kg in whole fish (Vanderploeg et al., 1975). Bioconcentration factors from water to whole fish range from 40 to 2,300 (Vanderploeg et al., 1975). Manganese bioconcentration factors for molluscs are relatively high. Vanderploeg et al. (1975) suggest a factor of 10,000 to be used for snail shells and whole snails and a factor of 2,000 for soft tissue of snails. A bioconcentration factor of 10,000 was also suggested for crustaceans (Vanderploeg et al., 1975).

The USEPA benchmark for manganese is set at 0.80 mg/L (EPA, 1996b). No Ohio EPA Warmwater Habitat Water Quality Criteria has been set for manganese. Suter and Mabrey (1994), however, have estimated acute and chronic advisory levels for manganese to be 1,470 and 80.3 $\mu\text{g/L}$, respectively. The lowest chronic values of manganese reported in the literature for fish and *Daphnia* are 1,770 $\mu\text{g/L}$ and less than 1,100 $\mu\text{g/L}$, respectively (Suter and Mabrey, 1994). The test EC_{20} for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). The values for manganese is 1,270 $\mu\text{g/L}$ (Suter and Mabrey, 1994). A similar value can be determined for daphnids, which reflects the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC_{20} benchmark for daphnids is less than 1,100 $\mu\text{g/L}$ (Suter and Mabrey, 1994).

REFERENCES

American Conference of Governmental Industrial Hygienists (ACGIH), 1991, Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed., ACGIH, Cincinnati, OH.

Glooschenko, W. A., and J. H. Capobianco, 1978, "Metal Content of *Sphagnum* Mosses from Two Canadian Bog Ecosystems," *Water, Air and Soil Pollution*, Vol. 10, pp. 215.

Gregus, Z., and C. D. Klaassen, 1986, "Disposition of Metals in Rats: A Comparative Study of Fecal, Urinary, and Biliary Excretion and Tissue Distribution of Eighteen Metals," *Toxicology and Applied Pharmacology*, Vol. 85, pp. 24-38.

Hammond, P. B., and R. P. Beliles, 1980, "Metals," in J. Doull, C. D. Klaassen, and M. O. Amdur (eds.), *Casarett and Doull's Toxicology: The Basic Science of Poisons*, 2nd ed., Macmillan Publishing Co., Inc., New York, New York, pp. 409-4671.

Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.

Leland, H. V., S. N. Luoma, and J. M. Fielden, 1979, "Bioaccumulation and Toxicity of Heavy Metals and Related Trace Elements," *Journal of Water Protection, Control Federation*, Vol. 51, pp. 1592-1616.

Martone, M. T., 1964, "A Study of the Effects of Chronic Inhalation of Manganese Dioxide in Pigeons and Rats," M.S. Thesis, University of Rochester, Rochester, New York.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Saric, M., 1986, Manganese, in *Handbook on the Toxicology of Metals*, L. Friberg, G. F. Nordberg, and V. B. Vouk, eds., 2nd ed., Vol. 2: Specific Metals, Elsevier Science Publishers B.V., New York, NY, pp. 354-386.

Schroeder, H. A., J. J. Balassa, and I. H. Tipton, 1966, "Essential Trace Metals in Man: Manganese," *Journal of Chronic Diseases*, Vol. 19, pp. 545-571.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

U.S. Environmental Protection Agency (EPA), 1996a, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996b, Ecotox Thresholds, EPA 540/F-95/038.

Vanderploeg, H. A., D. C. Parzyck, W. H. Wilcox, J. R. Kercher, and S. V. Kaye, 1975, "Bioaccumulation Factors for Radionuclides in Freshwater Biota," *ORNL-5002*, Oak Ridge National Laboratories, Oak Ridge, Tennessee.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

NICKEL

Human Health Effects

In a subchronic gavage study with nickel chloride in water, clinical signs of toxicity in rats included lethargy, ataxia, irregular breathing, reduced body temperature, salivation, and discolored extremities (EPA, 1996). Inhalation exposure was associated with asthma and pulmonary fibrosis in welders using nickel alloys (ACGIH, 1986). Lung effects were observed in laboratory animals exposed by inhalation. The EPA (1996) presented a verified RfD of 0.02 mg/kg/day for chronic oral exposure to nickel, based on an NOAEL for decreased organ and body weights in a two-year dietary study with nickel sulfate in rats and an uncertainty factor of 300. The EPA (1995) presented the same value as a provisional subchronic oral RfD. The CNS appears to be the target organ for the oral toxicity of nickel. The lung is clearly the target organ for inhalation exposure.

Occupational exposure to nickel was associated with increased risk of nasal, laryngeal and lung cancer (ATSDR, 1988). Inhalation exposure of rats to nickel subsulfide increased the incidence of lung tumors. The EPA (1996) presents a cancer weight-of-evidence Group A classification (human carcinogen) for nickel, and presents an inhalation unit risk of $0.00024 (\mu\text{g}/\text{m}^3)^{-1}$ for nickel refinery dust. The unit risk is equivalent to $0.84 (\text{mg}/\text{kg}/\text{day})^{-1}$, assuming humans inhale 20 m^3 of air/day and weigh 70 kg. The quantitative estimate was derived from the human occupational studies.

Ecological Effects

Nickel is not believed to be an essential element for plant growth; however, beneficial effects of nickel have been reported on the growth of legumes (Kabata-Pendias and Pendias, 1992). Background concentrations of nickel in grasses and clovers collected in the United States averaged 0.13 and 1.5 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Grains contain relatively high concentrations of nickel ranging from 0.10 to 1.2 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). The concentration of nickel in plants is positively correlated with nickel concentrations in soil (Kabata-Pendias and Pendias, 1992). Soil pH can have an effect on the availability of nickel to plants, where increasing the pH from 4.5 to 6.5 decreased the nickel concentration in oat grains by a factor of approximately 8 (Berrow and Burridge, 1981, as cited in

Kabata-Pendias and Pendias, 1992). Some plants, such as some alyssums (*Berteroa* sp.), are able to accumulate and tolerate elevated levels of nickel within their tissues. Elevated concentrations of nickel can be found in plants growing on or near sewage sludge and in areas where nickel occurs as an airborne pollutant. Adsorbed nickel can be washed off the leaves rather easily (Ashton, 1972).

Concentrations of nickel in leaf tissue that are excessive or toxic to various plant species, with the exclusion of very sensitive and highly tolerant species, range from 10 to 100 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Concentrations of nickel in plant tissues that are expected to result in a 10 percent loss in crop yield range from 10 to 30 mg/kg (dry weight) (Macnicol and Beckett, 1985). A soil concentration of 30 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for nickel phytotoxicity. General symptoms of nickel toxicity in plants include the presence of interveinal chlorosis in new leaves, gray-green leaves, and brown and stunted root and plant growth (Kabata-Pendias and Pendias, 1992). The uptake of nutrients and minerals, especially iron, can be substantially reduced as a consequence of nickel toxicity in plants (Kabata-Pendias and Pendias, 1992).

Nickel is a nonessential element for animal life. Nickel concentrations within the whole bodies of small mammals from uncontaminated sites were reported to range from 2.2 to 6.2 mg/kg (dry weight) (Talmage and Walton, 1991). Highest concentrations were measured in the deer mouse (*Peromyscus maniculatus*). Highest tissue concentrations of nickel are usually found in the liver of mammals (Schroeder et al., 1964, as cited in Jenkins, 1980). Laboratory studies have shown ingested nickel to accumulate in bone (Hammond and Beliles, 1980). Background concentrations of nickel in the feathers and eggs of birds are generally less than 0.05 mg/kg (dry weight) (Jenkins, 1980).

Because nickel is poorly absorbed by the gastrointestinal tract, ingested nickel is generally not of great toxicological concern. Inhaled nickel, however, is very toxic and has been categorized as a potent carcinogen. Based on toxicity data for rats exposed to nickel sulfate hexahydrate, extrapolated NOAELs for chronic oral exposure of various mammalian wildlife species are estimated as 99.7 mg/kg/d for the white-footed mouse, 26.6 mg/kg/d for the cottontail rabbit, and 17.2 mg/kg/d for the red fox (Opresko et al., 1994). Calculated chronic drinking-water NOAELs for mammalian wildlife are

332 mg/L for the white-footed mouse, 275 mg/L for the cottontail rabbit, and 204 mg/L for the red fox (Opresko et al., 1994).

Based on an estimated NOAEL of 77.4 mg/kg/d for mallard ducklings exposed to nickel sulphate, extrapolated NOAELs for chronic oral exposure of avian species to these compounds are 53.5 mg/kg/d for the great blue heron and 68.6 mg/kg/d for the red-tailed hawk (Opresko et al., 1994). The calculated drinking-water NOAEL for wild birds consuming either nickel sulfate only through drinking water is approximately 1,210 mg/L (Opresko et al., 1994).

Background concentrations in freshwater fish are generally less than 0.5 mg/kg (wet weight) (Jenkins, 1980). Nickel concentrations in tadpoles collected from the Patuxent Wildlife Research Center were found to average 2.7 mg/kg (dry weight) for *Rana catesbeiana* and 0.9 g/g (dry weight) for *Rana clamitans* (Hall and Mulhern, 1984). Background concentrations in adult anurans ranged between 0.9 and 2.9 mg/kg (dry weight) (Hall and Mulhern, 1984). Data do not suggest biological transformation of nickel in aquatic systems (Callahan et al., 1979).

The bioavailability and toxicity of nickel to aquatic biota is influenced by the pH of the water (Schubauer-Berigan et al., 1993). According to Schubauer-Berigan et al. (1993), toxicity of nickel to *Ceriodaphnia dubia* and *Hyalella azteca* were greatest under pH conditions of 8.3 and least toxic at a pH of 6.3. The Federal Water Quality Criteria for the Protection of Aquatic Life for acute and chronic exposure to nickel in freshwater systems are 1,400 and 160 µg/L, respectively (EPA, 1996). The Ohio EPA Warmwater Habitat Water Quality Criteria for nickel is set at 0.16 mg/L. The lowest chronic values of nickel reported in the literature for fish and *Daphnia* are less than 35 µg/L and less than 5 µg/L, respectively (Suter and Mabrey, 1994). The test EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). The value for nickel is 62 µg/L (Suter and Mabrey, 1994). A similar value can be determined for daphnids, which reflects the highest tested concentration causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a

chronic test with a daphnid species. The EC₂₀ benchmark for daphnids is 45 µg/L (Suter and Mabrey, 1994).

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR), 1988, Toxicological Profile for Nickel, U.S. Public Health Service, Atlanta, GA.

American Conference of Governmental Industrial Hygienists (ACGIH), 1986, Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed., Cincinnati, OH.

Ashton, W. M., 1972, "Nickel Pollution," *Nature*, Vol. 237, p.46.

Berrow, M. L., and J. C. Burridge, 1981, Persistence of Metals in Available Form in Sewage Sludge Treated Soils Under Field Conditions, in *Proceedings of the International Conference on Heavy Metals in the Environment, Amsterdam*, CEP Consultants Ltd., Edinburgh, United Kingdom.

Callahan, M. A., M. W. Slimak, N. W. Gabel et al., 1979, "Water-Related Environmental Fate of 129 Priority Pollutants, Vol. I," *EPA-440/4 79-029a*, U.S. Environmental Protection Agency, Washington, D.C.

Hall, R. J., and B. M. Mulhern, 1984, "Are Anuran Amphibians Heavy Metal Accumulators?," in R. A. Seigel, L. E. Hunt, J. L. Knight, L. Malaret and N. L. Zuschlag (eds.), *Vertebrate Ecology and Systematics—A Tribute to Henry S. Fitch*, Museum of Natural History, University of Kansas, Lawrence, Kansas, pp. 123-133.

Hammond, P. B., and R. P. Beliles, 1980, "Metals," in J. Doull, C. D. Klaassen, and M. O. Amdur (eds.), *Casarett and Doull's Toxicology: The Basic Science of Poisons*, 2nd ed., Macmillan Publishing Co., Inc., New York, New York, pp. 409-4671.

Jenkins, D. W., 1980, "Biological Monitoring of Toxic Trace Metals, Volume 2: Toxic Trace Metals in Plants and Animals of the World, Part I," *EPA Report 600/3-80-090*, U.S. Environmental Protection Agency, Washington, D.C., 503 pp.

Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.

Macnicol, R. D., and P. H. T. Beckett, 1985, "Critical Tissue Concentrations of Potentially Toxic Elements," *Plant and Soil*, Vol. 85, pp. 107-129.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/RI*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Schroeder, H. A., J. J. Balassa, and W. H. Vinton, 1964, "Chromium, Lead, Cadmium, Nickel and Titanium in Mice: Effects on Mortality, Tumors, and Tissue Levels," *Journal of Nutrition*, Vol. 83, pp. 239-250.

Schubauer-Berigan, M. K., J. R. Dierkes, P. D. Monson, and G. T. Ankley, 1993, "pH-dependent Toxicity of Cd, Cu, Ni, Pb and Zn to *Ceriodaphnia dubia*, *Pimephales promelas*, *Hyalella azteca* and *Lumbriculus variegatus*," *Environmental Contamination and Toxicology*, Vol. 12, pp. 1261-1266.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Talmage, S. S., and B. T. Walton, 1991, "Small Mammals as Monitors of Environmental Contaminants," *Reviews in Environmental Contamination and Toxicology*, Vol. 119, pp. 47-145.

U.S. Environmental Protection Agency (EPA), 1995, Health Effects Assessment Summary Tables. Annual Update FY 1995, including Supplements. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

POLYAROMATIC HYDROCARBONS

Human Health Effects

Several rat studies indicate that there is considerable chemical-specific variation in the pharmacokinetics of polyaromatic hydrocarbons (PAHs) (ATSDR, 1994). GI absorption is enhanced by solubilizing the chemical in a readily absorbed vehicle such as oil. Jones and Owen (1989) reported a range of 43 to 58 percent for the GI absorption of benzo(a)pyrene. The lower end of this range, 43 percent, is considered sufficiently conservative and well documented to use in estimating dermal RfDs and cancer slope factors from the respective oral values for all the EPA Group D PAHs.

The identification of metabolites of PAHs in the urine of occupationally exposed humans is semi-quantitative evidence that respiratory tract uptake occurs, although quantitative uptake data were not located (ATSDR, 1994). Studies in rats indicate that pulmonary absorption of benzo(a)pyrene is rapid. PAHs carried by insoluble particulate matter, however, would be retained in the lung longer than pure PAHs. Human and animal studies suggest that there is considerable chemical-specific variation in dermal absorption. Quantitative estimates in animals treated with radiolabeled compounds range from 33 percent for dibenzo(a,h)anthracene to 93 percent for benzo(a)pyrene.

Inhalation and oral studies in animals with radiolabeled benzo(a)pyrene indicate that distribution of absorbed material is primarily to the lipid fractions of the liver, lung, kidney, and GI tract, with redistribution to the protein fractions of these organs (ATSDR, 1994). Absorbed benzo(a)anthracene, dibenzo(a,h)anthracene, and chrysene are rapidly and widely distributed in orally treated rats. There is considerable chemical-specific variability in the distribution of the PAHs to the fetuses of pregnant rats.

Studies of the metabolism of benzo(a)pyrene provide information relevant to other PAHs, because of the structural similarities of all members of the class. Metabolism involves microsomal mixed function oxidase hydroxylation of one or more of the phenyl rings with the formation of phenols and dihydrodiols, probably via formation of arene oxide intermediates (ATSDR, 1994). The dihydrodiols may be further oxidized

to diol epoxides, which, for certain members of the class, are known to be the ultimate carcinogens (EPA, 1996a). Conjugation with glutathione or glucuronic acid and reduction to tetrahydrotetraols are important detoxification pathways.

Excretion of benzo(a)pyrene is principally through the bile, although there seems to be considerable species variation in the pattern (biliary versus urinary) and rate of excretion (ATSDR, 1994). Urinary excretion predominates slightly in rats treated dermally with anthracene.

Oral RfD values were not available for benzo(k)fluoranthene, phenanthrene, or any of the cancer weight-of-evidence Group B2 PAHs.

Mild kidney lesions appear to be the critical effects of pyrene. In mice treated by gavage for 13 weeks, 75 mg/kg/day was an NOAEL and 125 mg/kg/day was an LOAEL (EPA, 1989). Even in mice treated with 250 mg/kg/day the lesions were considered minimal to mild. The EPA (1996) verified a chronic oral RfD for pyrene of 0.03 mg/kg/day based on the NOAEL in mice and an uncertainty factor of 3,000 (10 each for inter- and intraspecies variation and to expand from subchronic to chronic exposure, and a factor of 3 to reflect gaps in the database). The EPA (1995) presented a provisional subchronic oral RfD of 0.3 mg/kg/day based on the same NOAEL and an uncertainty factor of 300. The kidney is the target organ for the toxicity of pyrene.

Benzo(a)pyrene is the most extensively studied PAH, inducing tumors in multiple tissues of virtually all laboratory species tested (ATSDR, 1994). Although epidemiology studies suggested that complex mixtures that contain PAHs (coal tar, soots, coke oven emissions, cigarette smoke) are carcinogenic to humans (EPA, 1996a), the carcinogenicity cannot be attributed to PAHs alone because of the presence of other potentially carcinogenic substances in these mixtures (ATSDR, 1994). Because of the lack of human cancer data, assignment of individual PAHs to EPA cancer weight-of-evidence groups was based largely on the results of animal studies with large doses of purified compound (EPA, 1996a). Frequently, unnatural routes of exposure, including implants of the test chemical in beeswax and trioctanoin into the lungs of rats, intratracheal instillation, and subcutaneous or intraperitoneal injection were used.

Benzo(g,h,i)perylene, phenanthrene and pyrene were classified in Group D (not classifiable as to carcinogenicity to humans), and benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene were classified in Group B2 (probable human carcinogen) (EPA, 1996a). Quantitative risk estimates are not derived for Group D compounds.

EPA (1996) verified a slope factor for oral exposure to benzo(a)pyrene of 7.3 per mg/kg-day, based on several dietary studies in mice and rats. A provisional unit risk of $0.0017 \text{ (mg/m}^3\text{)}^{-1}$ was based on respiratory tract tumors in hamsters exposed by inhalation (EPA, 1996a). The unit risk is equivalent to $6.1 \text{ (mg/kg-day)}^{-1}$, assuming an inhalation rate of $20 \text{ m}^3\text{/day}$ and a body weight of 70 kg for humans. Provisional quantitative risk estimates are available for the other PAHs in Group B2 (EPA, 1993). EPA (1980) promulgated an ambient water quality criterion for "total carcinogenic PAHs," based on an oral slope factor derived from a study with benzo(a)pyrene, as being sufficiently protective for the class. Largely because of this precedent, the quantitative risk estimates for benzo(a)pyrene are adopted for the other carcinogenic PAHs when quantitative estimates were needed.

Recent reevaluations of the carcinogenicity and mutagenicity of the Group B2 PAHs suggest that there are large differences between individual PAHs in cancer potency (Krewski, et al., 1989). Based on the available cancer and mutagenicity data, and assuming that there is a constant relative potency between different carcinogens across different bioassay systems and that the PAHs under consideration have similar dose-response curves, Thorslund and Charnley (1988) derived relative potency values for several PAHs. A more recent Toxicity Equivalency Function (TEF) scheme for the Group B2 PAHs was based only on the induction of lung epidermoid carcinomas in female Osborne-Mendel rats in the lung-implantation experiments (Clement International, 1990). Provisional TEFs for the determination of oral and inhalation slope factors are provided by EPA (1993).

Although the EPA has not verified slope factors for Group B2 PAHs other than benzo(a)pyrene, the slope factors based on TEFs represent reasonable estimates based on the data available. The relative potency approach employed here meets criteria considered to be desirable for this type of analysis (Lewtas, 1988). For example, the

chemicals compared have similar chemical structures and would be expected to have similar pharmacokinetic fate in mammalian systems. In addition, the available data suggest that the Group B2 PAHs have a similar mechanism of action, inducing frameshift mutations in *Salmonella* and tumor initiation in the mouse skin painting assay. Similar noncancer effects (minor changes in the blood, liver, kidneys) of the Group D PAHs support the hypothesis of a common mechanism of toxicity. Finally, the same endpoints of toxicity, i.e., potency in various cancer assays, and related data, were used to derive the relative potency values (Krewski, et al., 1989).

Ecological Effects

Polycyclic aromatic hydrocarbons (PAH) comprise a group of compounds containing two or more fused benzene rings. Although thousands of different PAHs are known to exist, 13 are of great environmental concern. These include acenaphthalene, anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, fluoranthene, indeno(1,2,3-cd)pyrene, naphthalene, phenanthrene, and pyrene. PAHs are ubiquitous in nature, occurring from both natural and anthropogenic sources. PAHs present in surface waters are expected to undergo hydrolysis. In general, these compounds have low water solubilities and therefore partition into sediments (Sims and Overcash, 1983).

Some PAHs are synthesized by plants at very low concentrations (Sims and Overcash, 1983). Background concentrations of specific PAH compounds usually range from 22 to 88 g/kg (dry weight) in tree leaves, 48 to 66 g/kg (dry weight) in cereal crop plants, 0.05 to 50 g/kg (dry weight) in leafy vegetables, 0.01 to 6 g/kg (dry weight) in underground vegetables, and 0.02 to 0.04 g/kg (dry weight) in fruits (Sims and Overcash, 1983). In general, PAH concentrations are usually greater in aboveground plant parts than in belowground parts and are greater on plant surfaces than within internal tissues (Edwards, 1983, as cited in Eisler, 1987). Lower-molecular-weight PAHs are taken up from soil by plants more readily than higher-molecular-weight PAHs (Eisler, 1987). Plant-to-soil concentration ratios for total PAHs have been reported to range from 0.001 to 0.183 (Wang and Meresz, 1981, as cited in Edwards, 1989). According to Edwards (1983, as cited in Talmage and Walton, 1990), plant-to-soil concentration ratios for benzo(a)pyrene are usually low, ranging from 0.0001 to 0.33. Atmospheric deposition is believed to be the usual source of PAHs in plants, not

uptake from soil (Sims and Overcash, 1983). The waxy surface of some plant leaves and fruits can concentrate PAHs through surface adsorption (EPA, 1980, as cited in NLM, 1996). Mosses have been recommended as good indicators of regional PAH air pollution (Herrmann and Hubner, 1984, as cited in Eisler, 1987). Some species of bacteria and fungi can degrade specific PAH compounds (Eisler, 1987; Sims and Overcash, 1983).

Limited data exist on the phytotoxicity of PAHs to plants. Benzo(b)fluoranthene concentrations of 6,254 g/kg in soil were reported to reduce stem growth in wheat but did not affect rye plants (Sims and Overcash, 1983). Dry-leaf mass was slightly reduced, and total dry yield was reduced by 11 percent in the wheat plants exposed to the elevated benzo(b)fluoranthene concentration. Benzo(a)pyrene and benzo(b)fluoranthene soil concentrations of up to 18,000 g/kg do not appear to be severely toxic to higher plants (Sims and Overcash, 1983). There is some evidence that low concentrations of some PAHs may actually stimulate plant growth (Edwards, 1989).

Concentrations of PAH compounds in wild mammals and birds could not be found in the open literature. Exposure to PAHs can occur via inhalation, ingestion, or dermal exposure. Most of the PAHs taken in the body are not accumulated but are oxidized, and the metabolites are excreted (Sittig, 1985, as cited in NLM, 1996). In fact, most PAH compounds are detoxified and excreted from the body (Doull et al., 1986, as cited in NLM, 1996). PAHs are metabolized in vertebrates by a group of enzymes known as mixed-function oxidase in the liver. Some of the intermediate metabolites have been identified as mutagenic, carcinogenic, and teratogenic (Sims and Overcash, 1983).

In most cases, tissue damage from exposure to PAH compounds usually occurs at dose levels that would be expected to induce carcinomas (Eisler, 1987). The toxic response to a PAH compound is a function of the specific compound, the dose, and the route of exposure. Unsubstituted aromatic PAHs with less than four condensed rings have not been shown to be tumorigenic (Eisler, 1987). Many PAHs with from four to six rings are carcinogenic (Eisler, 1987). Compounds such as 7,12-dimethylbenz(a)anthracene and benzo(a)pyrene can induce skin tumors following dermal exposure (Weisburger and Williams, 1980). One isomer of the benzo(a)pyrene metabolite 7,8-dihydrodiol 9,10-

epoxide is a very potent carcinogen to newborn mice (Slaga et al., 1978, as cited in Eisler, 1987). Some PAH compounds may act in a synergistic or cocarcinogenic manner when combined (Eisler, 1987).

Studies have not been conducted on the toxicity of PAH compounds to wildlife. A few laboratory studies on rodents have revealed acute oral toxicities of PAHs are greatest for benzo(a)pyrene, followed in decreasing order of toxicity by phenanthrene, naphthalene, and fluoranthene (Sims and Overcash, 1983). LD₅₀ values range from 50 mg/kg body weight to 2,000 mg/kg body weight (Sims and Overcash, 1983). Chronic oral doses that result in the production of cancer are lowest for 7,12-dimethylbenz(a)anthracene at a dose of 4.0×10^{-5} to 2.5×10^{-4} mg/kg body weight (Eisler, 1987). Benzo(a)pyrene concentrations of 0.002 mg/kg body weight and anthracene concentrations of 3,300 mg/kg body weight will also result in cancer following chronic oral exposure to the specific compound (Eisler, 1987). Based on an estimated laboratory mouse oral NOAEL of 1.0 mg/kg/d for benzo(a)pyrene, Opresko et al. (1994) estimated wildlife NOAELs for benzo(a)pyrene of 1.11 mg/kg/d for the white-footed mouse, 0.296 mg/kg/d for the cottontail rabbit, and 0.191 mg/kg/d for the red fox. Calculated chronic NOAELs for mammalian wildlife exposed to benzo(a)pyrene in drinking water only range from 1.91 to 7.76 mg/L (Opresko et al., 1994).

Bioconcentration factors have been reported for aquatic biota exposed to PAHs under laboratory conditions (Eisler, 1987). Bioconcentration factors for *Daphnia* exposed to specific PAH compounds for a period of at least 24 hours range from 131 for naphthalene to 134,248 for benzo(a)pyrene (Eisler, 1987). Water to liver bioconcentration factors for freshwater fish exposed to benzo(a)pyrene for a minimum of eight days range from 182 for rainbow trout (*Salmo gairdneri*) to 1,375 for Northern pike (*Esox lucius*) (Eisler, 1987). There is little evidence for bioaccumulation and biomagnification of PAHs in the aquatic environment (Eisler, 1987).

The toxicity of PAH compounds to fish is also related to the solubility of the compound in water. Toxicity to aquatic biota also increases as the molecular weight of the PAH compound and the degree of alkyl substitutions on the aromatic ring increase (Eisler, 1987). The toxicity of PAHs to aquatic organisms is very species-specific and related

to the organisms' ability to metabolize and excrete the compound (Eisler, 1987). Because many species of fish are able to metabolize benzo(a)pyrene to reactive intermediates that have mutagenic and carcinogenic properties, the presence of tumors in fish from PAH-contaminated environments is often related to exposure to PAHs (Eisler, 1987). Other toxic responses that have been noted in aquatic biota exposed to PAHs include inhibited reproduction in daphnids, delayed emergence of larval midges, decreased respiration and heart rate in mussels, inhibition of photosynthesis in algae and aquatic macrophytes, and liver enlargement in fish (Eisler, 1987).

A few Federal Water Quality Criteria exist for the protection of freshwater aquatic life. These are an acute value of 3,980 $\mu\text{g/L}$ for fluoroanthene, an acute value of 30 $\mu\text{g/L}$ and chronic value of 6.3 $\mu\text{g/L}$ for phenanthrene, and an acute value of 2,300 $\mu\text{g/L}$ and a chronic value of 620 $\mu\text{g/L}$ for naphthalene (EPA, 1996a). The OEPA Warmwater Habitat Water Quality Criteria for PAHs range from 0.00031 to 0.0089. US EPA benchmarks have been set for some PAHs: benzo(a)pyrene (1.4×10^{-5} mg/L), fluoranthene (0.00081 mg/L), and phenanthrene (0.0063 mg/L) (EPA, 1996b). Suter and Mabrey (1994), however, have derived acute and chronic advisory values for freshwater biota exposed to PAHs. These are presented in Table D-2. Also presented in Table D-2 are the lowest chronic values of specific PAHs reported in the literature for fish and daphnids (Suter and Mabrey, 1994). The test EC_{20} for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration of a specific PAH causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). A similar value can be determined for daphnids, which reflects the highest tested concentration of a PAH causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species (Suter and Mabrey, 1994) (Table below).

**Benchmarks Screening Values for Freshwater Biota
Exposed to Polycyclic Aromatic Hydrocarbons
Where No Federal Criteria Exist^a**

PAH ^b	Advisory Value (µg/L)		Lowest Chronic Value (µg/L)		Lowest Test EC ₂₀ Value (µg/L)	
	Acute	Chronic	Fish	Daphnids	Fish	Daphnids
Acenaphthene	—	—	74	6,646	< 197	—
Anthracene	0.024	0.0013	0.09	< 2.1	0.35	> 8.2
Benzo(a)anthracene	0.49	0.027	—	0.65	—	—
Benzo(a)pyrene	0.24	0.014	—	0.30	> 2.99	—

^aBenchmark screening values obtained from Suter and Mabrey, 1994.

^bPAH = Polycyclic aromatic hydrocarbons.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR), 1994, Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft for Public Comment, U.S. Public Health Service, Atlanta, GA.

Clement International, 1990, Development of Relative Potency Estimates for PAHs and Hydrocarbon Combustion Product Fractions Compared to Benzo[a]Pyrene and Their Use in Carcinogen Risk Assessments, prepared for the EPA.

Doull, J., C. D. Klaassen, and M. D. Amdur (eds.), 1986, *Casarett and Doull's Toxicology*, 3rd ed., Macmillan Co. Inc., New, York, New York.

Edwards, N. T., 1989, "Fate and Effects of PAHs in the Terrestrial Environment—An Overview," (Abstract, 89-88.4), The 82nd Air & Waste Management Association Meeting, Anaheim, California.

Edwards, N. T., 1983, "Polycyclic Aromatic Hydrocarbons (PAHs) in the Terrestrial Environment—A Review," *Journal of Environmental Quality*, Vol. 12, pp. 427-441.

Eisler, R., 1987, Polycyclic aromatic hydrocarbons hazards to fish, wildlife, and invertebrates: A synoptic review, Laurel, MD.

Herrmann, R. A., and D. Hubner, 1984, "Concentrations of Micropollutants (PAH, Chlorinated Hydrocarbons and Trace Metals) in the Moss *Hypnum cupressiforme* in and Around a Small Industrial Town in Southern Finland," *Annals Botanica Fennici*, Vol. 21, pp. 337-342.

Jones, T. D. and B. A. Owen, 1989, Health Risks from Mixtures of Radionuclides and Chemicals in Drinking Water, Oak Ridge National Laboratory, Oak Ridge, TN, ORNL-6533.

Krewski, D., T. Thorslund, and J. Withey, 1989, Carcinogenic Risk Assessment of Complex Mixtures, *Toxicol. Ind. Health*, Vol. 5, pp. 851-867.

Lewtas, J., 1988, Genotoxicity of Complex Mixtures: Strategies for the Identification and Comparative Assessment of Airborne Mutagens and Carcinogens from Combustion Sources, *Fund. Appl. Toxicol.*, Vol. 10, pp. 571-589.

National Library of Medicine (NLM), 1996, "Hazardous Substance Data Bank," produced by Micromedex, Inc.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Sims, R. C., and M. R. Overcash, 1983, "Fate of Polynuclear Aromatic Compounds (PNAs) in Soil-Plant Systems," *Residue Reviews*, Vol. 88, pp. 1-67.

Sittig, M., 1985, *Handbook of Toxic and Hazardous Chemicals and Carcinogens*, 2nd ed., Noyes Data Corp., Park Ridge, New Jersey.

Slaga, T. J., W. M. Bracken, A. Viaje, D. L. Berry, S. M. Fischer, D. R. Miller, W. Levin, A. H. Conney, H. Yagi, and D. M. Jerina, 1978, "Tumor Initiating and Promoting Activities of Various Benzo(a)pyrene Metabolites in Mouse Skin," pp. 371-382 in P. W. Jones and R. I. Freudenthal (eds.), *Carcinogenesis—A Comprehensive Survey, Vol. 3, Polynuclear Aromatic Hydrocarbons: Second International Symposium on Analysis, Chemistry, and Biology*, Raven Press, New York, New York.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Talmage, S. S., and B. T. Walton, 1990, "Comparative Evaluation of Several Small Mammal Species as Monitors of Heavy Metals, Radionuclides, and Selected Organic Compounds in the Environment," *ORNL/TM-11605*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Thorslund, T., and G. Charnley, 1988, *Comparative Potency Approach for Estimating the Cancer Risk Associated with Exposure to Mixtures of Polycyclic Aromatic Hydrocarbons*, ICF-Clement Associates, Washington, D.C.

U.S. Environmental Protection Agency (EPA), 1980, *Identification and Listing of Hazardous Waste Under RCRA, Subtitle C, Section 3001, Health and Environmental Effects Profiles*, Office of Solid Waste, Washington, DC, NTIS No. PB81 190019.

U.S. Environmental Protection Agency (EPA), 1989, *Mouse oral subchronic toxicity of pyrene*, Study conducted by Toxicity Research Laboratories, Muskegon, MI for the Office of Solid Waste, Washington, DC. (Cited in EPA, 1992).

U.S. Environmental Protection Agency (EPA), 1993, *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons*. Office of Research and Development, Washington, DC. EPA/600/R-93/089.

U.S. Environmental Protection Agency (EPA), 1995, Health Effects Assessment Summary Tables, Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996a, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996b, Ecotox Thresholds, EPA 540/F-95/038.

Weisburger, J. H., and G. M. Williams, 1980, "Chemical Carcinogenesis," in J. Doull, C. D. Klaassen, and M. O. Amdur, *Casarett and Doull's Toxicology, the Basic Science of Poisons*, Macmillan Publishing Co., Inc., New York, New York, pp. 84-138.

TETRACHLOROETHENE

Human Health Effects

Occupational (inhalation and dermal) exposure to tetrachloroethene was associated with neurologic effects, beginning with incoordination and progressing to dizziness, headache, vertigo, and unconsciousness (ACGIH, 1986). The EPA (1996) presented a verified chronic oral RfD for tetrachloroethene of 0.01 mg/kg/day based on an NOAEL for liver toxicity in mice in a subchronic gavage study, and on an NOEL for depressed body weight gain in rats in a subchronic drinking water study. An uncertainty factor of 1000 was used. The EPA (1995) presented a provisional subchronic oral RfD of 0.1 mg/kg/day based on the same NOEL and an uncertainty factor of 100. The CNS is the principal target organ for inhalation exposure and the liver is the principal target organ for oral exposure to tetrachloroethene.

Inhalation exposure to tetrachloroethene induced mononuclear cell leukemia in rats, and inhalation or oral exposure induced hepatocellular carcinomas in mice (ATSDR, 1987). Occupational exposure data do not suggest a carcinogenic role for tetrachloroethene in humans (ACGIH, 1986). Interpretation of the data regarding the carcinogenicity of tetrachloroethene is controversial, and the EPA (1996) has not adopted a final position on the cancer weight-of-evidence classification or quantitative risk estimates for tetrachloroethene.

Ecological Effects

Tetrachloroethylene, also referred to as 1,1,2,2-tetrachloroethylene, is not a naturally occurring compound (Howard, 1990). Most of the tetrachloroethylene produced is used in the dry-cleaning industry and in the cleaning and degreasing of metals (Howard, 1990). Tetrachloroethylene in soil is subject to evaporation and to leaching into groundwater (Howard, 1990). Biodegradation may be an important removal process in anaerobic soils (NLM, 1996). Tetrachloroethylene in aquatic systems is primarily lost through evaporation (Wakeham et al., 1983, as cited in NLM, 1996). Adsorption to sediment is not expected to be significant (NLM, 1996).

Information on concentrations of tetrachloroethylene in plants and phytotoxicity data could not be found in the literature.

Tetrachloroethylene is readily absorbed through the lung and to a much lesser degree through skin and the GI tract (Arena and Drew, 1986, as cited in NLM, 1996). Ingested tetrachloroethylene is largely exhaled, with a small fraction of metabolized components excreted in urine (Parke, 1968, as cited in NLM, 1996). Metabolism of the compound is relatively slow (Ikeda and Ohtsuji, 1972, as cited in Cornish, 1980). Tetrachloroethylene tends to accumulate in adipose tissues (Ellenhorn and Barceloux, 1988, as cited in NLM, 1996). Concentrations of tetrachloroethylene in wild birds and mammals could not be located in the literature.

Specific data on the toxicity of tetrachloroethylene to wildlife do not exist. Tetrachloroethylene is a central nervous system depressant (NLM, 1996). Hepatic and renal disorders have also been associated with exposure to tetrachloroethylene (NLM, 1996). The lowest published lethal dose or concentration following oral exposure of animals to tetrachloroethylene are 5 g/kg for the rabbit and 4 g/kg for the dog and cat (RTECS, 1996). Oral LD₅₀ values for rats and mice exposed to tetrachloroethylene are 2.629 and 8.1 g/kg (RTECS, 1996). Inhalation LC₅₀ values of 34.2 g/m³/8 hr and 5,200 ppm/4 hr have been determined for rats and mice, respectively, exposed to tetrachloroethylene (RTECS, 1996). Adverse impacts on fertility have been reported in male mice exposed to 500 ppm/7 hr tetrachloroethylene for five days prior to mating (RTECS, 1996). Postimplantation mortality was elevated in female rats exposed to the compound at 300 ppm/7 hr from day 6 to 15 of pregnancy (RTECS, 1996). Exposure to tetrachloroethylene is also toxic to the fetus at a concentration as low as 300 ppm/7 hr (female mice exposed from day 6 to 15 of pregnancy) (RTECS, 1996). Teratogenic effects have also been associated with tetrachloroethylene (RTECS, 1996). An oral no observed effect level (NOEL) of 14 mg/kg/day has been reported for laboratory rats exposed to tetrachloroethylene (EPA, 1996). Wildlife NOAELs for tetrachloroethylene, based on extrapolations from laboratory mouse studies, are 1.55 mg/kg for the white-footed mouse, 0.41 mg/kg/d for the cottontail rabbit, and 0.27 mg/kg/d for the red fox (Opresko et al., 1994). Calculated chronic drinking-water NOAELs for mammalian wildlife range from 1.78 to 13.8 mg/L (Opresko et al., 1994). Tetrachloroethylene has been shown to be genotoxic and carcinogenic (RTECS, 1996). Signs of tetrachloroethylene poisoning in rodents include dizziness, incoordination, and unconsciousness (NLM, 1996).

Tetrachloroethylene in aquatic environments is not expected to biodegrade or bioconcentrate in aquatic biota (NLM, 1996). Bioconcentration factors that have been reported for fathead minnows (*Pimephales promelas*) and bluegill sunfish (*Lepomis macrochirus*) exposed to tetrachloroethylene are 38.9 and 49, respectively (Neely et al., 1974, and Barrows et al., 1980, as cited in NLM, 1996). Carp and eels collected from the Delaware River were reported to contain 77 and 250 g/kg tetrachloroethylene, respectively (Dickson and Riley, 1976, as cited in NLM, 1996).

Data on the toxicity of tetrachloroethylene to freshwater biota are limited. Tetrachloroethylene was found to increase the relative abundance and decrease species diversity in phytoplankton communities initially exposed to 1.2 mg/L (0.1 mg/L after five days) (Lay et al., 1984, as cited in NLM, 1996). Lethal effects occurred in the *Daphnia* population in this study (Lay et al., 1984, as cited in NLM, 1996). Examples of 96-hour LC₅₀ values for freshwater fish exposed to tetrachloroethylene are 18.4 to 21.4 mg/L for fathead minnows, 13 mg/L for bluegill sunfish, and 5 mg/L for rainbow trout (*Salmo gairdneri*) (Verschuere, 1983; Buccafusco et al., 1981; and Shubat et al., 1982, as cited in NLM, 1996).

Federal Water Quality Criteria do not exist for the protection of freshwater aquatic life from exposure to tetrachloroethylene (EPA, 1996). Lowest effect levels listed by EPA (1996) following acute and chronic exposure in freshwater systems are 5,280 and 840 µg/L, respectively. Suter and Mabrey (1994) recommend acute and chronic advisory values of 998 and 12.5 µg/L, respectively, for the protection of freshwater biota. Lowest chronic toxicity values of tetrachloroethylene to fish and daphnids are 840 and 750 µg/L, respectively (Suter and Mabrey, 1994). The test EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than a 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). The value for tetrachloroethylene has been estimated to be 500 µg/L (Suter and Mabrey, 1994). A similar value can be determined for daphnids, which reflects the highest tested concentration of tetrachloroethylene that will cause less than a 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid

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species (Suter and Mabrey, 1994). The EC₅₀ benchmark for daphnids is 510 µg/L (Suter and Mabrey, 1994).

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR), 1987, Toxicological Profile for Tetrachloroethylene. Draft. U.S. Public Health Service, Atlanta, GA.

American Conference of Governmental Industrial Hygienists (ACGIH), 1986, Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed., Cincinnati, OH.

Arena, J. M., and R. H. Drew (eds.), 1986, *Poisoning—Toxicology, Symptoms, Treatments*, 5th ed., Charles C. Thomas Publisher, Springfield, Illinois, 257 pp.

Barrows, M. E., et al., 1980, *Dynamic Exposure Hazard Assessment of Toxic Chemicals*, Ann Arbor Press, Ann Arbor, Michigan.

Buccafusco, R. J., et al., 1981, *Bulletin of Environmental Contamination and Toxicology*, Vol. 26, pp. 446.

Cornish, H. H., 1980, "Solvents and Vapors," in *Casarett and Doull's Toxicology: The Basic Science of Poisons*, pp. 469-496, J. Doull, C. D. Klaassen, and M. O. Amdur (eds.), Macmillan Publishing Co., Inc., New York, New York, 778 pp.

Dickson, A. G., and J. P. Riley, 1976, *Marine Pollution Bulletin*, Vol. 7, pp. 167-169.

Ellenhorn, M. J., and D. G. Barceloux, 1988, *Medical Toxicology—Diagnosis and Treatment of Human Poisoning*, Elsevier Science Publishing Co., Inc., New York, 986 pp.

Howard, P. H., 1991, *Handbook of Environmental Fate and Exposure Data for Organic Chemicals, Volume III: Pesticides*, Lewis Publishers, Chelsea, Michigan, 684 pp.

Ikeda, M. and H. Ohtsuji, 1972, "A Comparative Study on the Excretion of Fuji-Ware Reaction-Positive Substances in the Urine of Humans and Rodents Given Trichloro-

Lay, J. P., et al., 1984, *Archives of Environmental Contamination and Toxicology*, Vol. 13, pp. 135-142.

National Library of Medicine (NLM), 1996, "Hazardous Substance Data Bank," produced by Micromedex, Inc.

Neely, W. B., et al., 1974, *Environmental Science and Technology*, Vol. 8, pp. 1113-1115.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Registry of Toxic Effects of Chemical Substances (RTECS), 1996, produced by Micromedex.

Shubat, P. J., et al., 1982, *Bulletin of Environmental Contamination and Toxicology*, Vol. 28, pp. 7-10.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

U.S. Environmental Protection Agency (EPA), 1995, Health Effects Assessment Summary Tables. Annual Update FY 1995, including Supplements. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1996, Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Washington, DC.

Verschueren, K., 1983, *Handbook of Environmental Data of Organic Chemicals*, 2nd ed., Van Nostrand Reinhold Co., New York, New York, 1080 pp.

Wakeham, S. G., et al., 1983, *Environmental Science and Technology*, Vol. 17, pp. 611-617.

TRICHLOROETHYLENE

Human Health Effects

Trichloroethylene (TCE) is a colorless, highly volatile liquid. It is used primarily as a dry cleaning and metal degreasing agent and to a lesser extent as a solvent in adhesives and paints. Information on TCE has been reviewed by Torkelson and Rowe (1981), EPA (1980, 1983 and 1984), Hermens and others (1984), and NLM (1996).

Due to TCE's high vapor pressure and low partition coefficients, volatilization from soils is the primary fate process. Biodegradation is a slower process; its products include dichloroethylene and vinyl chloride. The half-life in soil has been measured to be 300 days. TCE is highly mobile in soil and will leach into ground-water where it is relatively persistent. Although persistent, degradation may occur to cis- and trans-1,2-dichloroethylene. Under anaerobic conditions TCE can biodegrade to 1,1-dichloroethylene and cis- and trans-1,2-dichloroethylene.

TCE is absorbed by all major routes of entry. Absorption following inhalation is estimated at from 36 to 75 percent. TCE is assumed to be readily absorbed by ingestion due to its lipophilicity and nonpolarity. Dermal absorption is thought to be slow. TCE will attain an equilibrium with the brain, heart, kidneys and liver at a faster rate than adipose tissue. But, repeated exposures can cause accumulation of TCE in adipose tissue. The compound is metabolized in the liver to a variety of metabolites, at least some of which are responsible for much of trichloroethylene's toxicity. Metabolites are excreted primarily in the urine. TCE interacts with a number of other chemicals, including ethanol, generally increasing the severity of effects of both compounds.

Acute exposures cause central nervous system depression and irritation of mucous membranes. TCE was once used as a surgical anesthetic, but this practice has been abandoned because of side effects, such as cardiac arrhythmias and sensitization to epinephrine-induced arrhythmia, and liver failure, both sometimes fatal. Chronic dosing produces liver and kidney lesions as well as a peripheral neuritis. In the evaluation of TCE's carcinogenicity, it has been placed in Group B2, probable human carcinogen, based on sufficient evidence of carcinogenicity in animals for the oral and

inhalation routes of exposure. Exposure to TCE has been associated with developmental effects in animals. A significant increase in litter resorptions, reduction in fetal body weight, and various skeletal ossification abnormalities were observed. Studies on reproduction have reported increases in sperm abnormalities. Data regarding the genotoxicity of TCE are inconclusive.

Ecological Effects

Trichloroethene, or trichloroethylene, is not known to occur as a natural product (Howard, 1990). This chlorinated organic is primarily used for the vapor degreasing of metals (Howard, 1990). Photooxidation is the primary mode by which trichloroethene is removed from the atmosphere (NLM, 1996). The compound is fairly stable in soil; however, it can leach into groundwater (Howard, 1990). The primary removal process in aquatic systems is evaporation (Howard, 1990). Biodegradation is expected to be significant only under anaerobic conditions (NLM, 1996).

Data on measured concentrations of trichloroethene in plants have been reported in grain-based foods, which ranged from 0.77 to 2.7 g/kg (Heikes and Hopper, 1986, as cited in Howard, 1990). Phytotoxicity data on trichloroethene could not be found in the literature.

Concentrations of trichloroethene in wildlife are not reported in the literature.

Specific data on the toxicity of trichloroethene to wildlife do not exist. Trichloroethane is a hepatotoxin and central nervous system toxin (NLM, 1996). The oral LD₅₀ value for mice exposed to trichloroethene is 2.402 g/kg (RTECS, 1996). The inhalation LC₅₀ value for mice exposed to trichloroethene is 8,450 ppm/4 hr (RTECS, 1996). The LD₅₀ for dermal exposure of rabbits to trichloroethene is greater than 20 g/kg (RTECS, 1996). Fetotoxic and teratogenic effects have been reported in the offspring of pregnant rats exposed to trichloroethene (RTECS, 1996). Symptoms of chronic poisoning in dogs from inhalation of trichloroethene include lethargy, anorexia, nausea, vomiting, and weight loss (ACGIH, 1971). There is a limited amount of evidence that suggests trichloroethene is carcinogenic in mammals (RTECS, 1996).

Wildlife NOAELs for trichloroethene based on extrapolations from laboratory mouse studies with an estimated NOAEL of 0.7 mg/kg/d are 0.78 mg/kg for the white-footed mouse, 0.21 mg/kg/d for the cottontail rabbit, and 0.13 mg/kg/d for the red fox (Opresko et al., 1994). Calculated chronic drinking-water NOAELs for mammalian wildlife range from 0.89 to 6.91 mg/L (Opresko et al., 1994).

Very little data exist on concentrations of trichloroethene in aquatic biota. Concentrations of the compound in bivalve molluscs collected from Lake Pontchartrain contained average concentrations of less than 5.7 g/kg (Ferrario et al., 1985, as cited in Howard, 1990). Bioconcentration factors of between 17 and 39 have been reported for bluegill sunfish and rainbow trout exposed to trichloroethene (Barrows et al., 1980, and Lyman, 1981, respectively, as cited in Howard, 1990).

Data on the toxicity of trichloroethene to freshwater biota are limited. LC₅₀ values for sheepshead minnows (*Pimephales promelas*) and bluegill sunfish exposed to trichloroethene for 96 hours are 20 and 44.7 mg/L (Borthwick, 1977, and EPA, 1978, respectively, as cited in NLM, 1996). Federal Water Quality Criteria do not exist for the protection of freshwater aquatic life from exposure to trichloroethene (EPA, 1996). Acute and chronic lowest effect levels have, however, been listed by EPA (1996) as 45,000 µg/L and 21,900 µg/L, respectively. Suter and Mabrey (1994) recommend acute and chronic advisory values of 4,350 and 465 µg/L, respectively, for the protection of freshwater biota. Lowest chronic toxicity values of trichloroethene to fish and daphnids are 14,867 and 7,257 µg/L (estimated), respectively (Suter and Mabrey, 1994). The test EC₂₀ for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than a 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). The value for trichloroethene has been determined to be 5,758 µg/L (Suter and Mabrey, 1994).

REFERENCES

American Conference of Governmental Industrial Hygienists (ACGIH), 1971, *Documentation of the Threshold Limit Values for Substances in Workroom Air*, 3rd ed., American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio.

Barrows, M. E., et al., 1980, *Dynamic Exposure Hazard Assessment of Toxic Chemicals*, Ann Arbor Press, Ann Arbor, Michigan.

Borthwick, P. W., 1977, "Results of Toxicity Tests with Fish and Macroinvertebrates," U.S. Environmental Protection Agency, Environmental Research Laboratory, Washington, D.C.

Ferrario, J. B., et al., 1985, *Bulletin of Environmental Contamination and Toxicology*, Vol. 34, pp. 246-255.

Heikes, D. L., and M. L. Hopper, 1986, *Journal of the Association of Official Analytical Chemistry*, Vol. 70, pp. 215-226.

Hermens, J. and others, 1984, Quantitative Structure-Activity Relationships and Toxicity Studies of Mixtures of Chemicals with Anaesthetic Potency: Acute Lethal and Sublethal Toxicity of *Daphnia magna*, *Aquatic Toxicology*, 5, 143-154.

Howard, P. H. (ed.), 1990, *Handbook of Environmental Fate and Exposure Data for Organic Chemicals, Vol. II, Solvents*, Lewis Publishers, Chelsea, Michigan.

National Library of Medicine (NLM), 1996, Hazardous Substances Databank, Toxicology Information Network (TOXNET).

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/RI*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Registry of Toxic Effects of Chemical Substances (RTECS), 1996, produced by Micromedex.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/RI*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Torkelson, T.R. and V.K. Rowe, 1981, Halogenated Aliphatic Hydrocarbons Containing Chlorine, Bromine, and Iodine, in Patty's Industrial Hygiene and Toxicology, George D. Clayton and Florence E. Clayton (Editors), Third Revised Edition, Volume 2B. New York, John Wiley & Sons.

U.S. Environmental Protection Agency (EPA), 1996, "Integrated Risk Information System," on-line database, maintained by the U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency (EPA), 1984, Health Effects Assessment for Trichloroethylene. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-H046.

U.S. Environmental Protection Agency (EPA), 1983, Health Assessment Documents for Trichloroethylene. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-82/066B.

U.S. Environmental Protection Agency (EPA), 1980, Ambient Water Quality Criteria for Trichloroethylene. Office of Water Regulations and Standards, Washington, D.C. EPA/440/5-80/073.

U.S. Environmental Protection Agency (EPA), 1978, "In-Depth Studies on Health and Environmental Impacts of Selected Water Pollutants," EPA No. 68-01-4646, U.S. Environmental Protection Agency. Washington, D.C.

VANADIUM

Human Health Effects

The GI absorption of ingested vanadium is very low. A study in humans reported absorption of a very soluble compound, oxytartarovanadate, to be 0.1 to 1 percent (Lagerkvist, et al., 1986). Uptake from the diet was estimated to be not greater than 1 percent. Uptake of vanadium from vanadium pentoxide was 2.6 percent of the administered dose in rats. In the absence of better quantified absorption data, the EPA (1989) default of 5 percent is used to derive a dermal RfD from an oral RfD.

The extent of absorption of vanadium from the respiratory tract depends on particle size and solubility of the vanadium compound (Lagerkvist, et al., 1986). Although not precisely quantified, the respiratory tract absorption of soluble vanadium compounds was estimated at 25 percent (species not reported). Occupationally exposed workers excrete more vanadium in their urine than do controls. In rats, rapid uptake followed the intratracheal instillation of several vanadium compounds. For example, more than one-half of an intratracheal dose of vanadyl trichloride was absorbed from the lungs within 1 day; 3 percent of the dose remained in the lungs 63 days after treatment.

In laboratory animals, absorbed vanadium is distributed principally to bone, kidney, liver, and spleen (Lagerkvist, et al., 1986). In humans and laboratory animals, systemic vanadium is excreted principally in the urine.

The oral toxicity of vanadium and compounds to humans is very low (Lagerkvist et al, 1986), probably because little vanadium is absorbed from the GI tract. Effects in humans exposed by inhalation include upper and lower respiratory tract irritation. A chronic oral RfD of 0.007 mg/kg-day was derived from an NOEL in rats in a lifetime drinking water study with vanadyl sulfate and an uncertainty factor of 100 (EPA, 1995). A target organ could not be identified for oral exposure. The respiratory tract is the target organ for inhalation exposure.

Vanadium is classified in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) (EPA, 1995). Quantitative risk estimates are not derived for Group D chemicals.

Ecological Effects

There is some controversy over whether vanadium is an essential element for plants (Kabata-Pendias and Pendias, 1992; Lauchli and Bielecki, 1983). It appears to be required by some algal species and may be required by nitrogen-fixing bacteria (Kabata-Pendias and Pendias, 1992). Mean background concentrations of vanadium in plants are 1.6 mg/kg for angiosperms, 0.69 mg/kg for gymnosperms, and 0.67 mg/kg for fungi (dry weight) (Waters, 1977). Vanadium concentrations in mosses and lichens are often higher than in vascular plants (Jenkins, 1980), averaging about 2.3 mg/kg (dry weight) (Waters, 1977). These plants appear to be good monitors of aerial vanadium pollution (Kabata-Pendias and Pendias, 1992). The fly agaric mushroom (*Amanita muscaria*) is considered a vanadium accumulator species (Bertrand, 1950, as cited in Waters, 1977). Concentrations of up to 345 mg/kg (dry weight) have been measured in mushrooms of this species collected from an uncontaminated area with soil vanadium concentrations of 6.7 mg/kg (dry weight) (Lepp et al., 1987, as cited in Kabata-Pendias and Pendias, 1992). The availability of vanadium to plants is highly dependent on soil pH (Kabata-Pendias and Pendias, 1992). Elevated levels of vanadium in soils can also reduce the uptake of manganese, copper, calcium, and phosphorus (NRCC, 1980, as cited in NLM, 1996).

Concentrations of vanadium in leaf tissue that are excessive or toxic to various plant species, with the exclusion of very sensitive and highly tolerant species, range from 5 to 10 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). A soil concentration of 2 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for vanadium phytotoxicity.

Vanadium has been shown to be essential in the diets of chicks and rats (Waters, 1977). Wildlife generally have higher tissue concentrations of vanadium than do humans (Waters, 1977). Background concentrations of vanadium in the kidneys and livers of wild mammals are reported to range from 0 to 2.07 mg/kg (wet weight) and from 0 to 0.94 mg/kg (wet weight) (Schroeder, 1970, as cited in Waters, 1977). Liver and skeletal tissues usually contain the highest concentrations of vanadium (Bertrand, 1950, as cited in Waters, 1977), although fat may also serve as a storage tissue (Hammond and Beliles, 1980).

Toxic responses to vanadium can occur following ingestion or inhalation. The toxicity of vanadium increases with increasing valence, with pentavalent vanadium as the most toxic form (NRC, 1977, as cited in NLM, 1996). Laboratory studies have shown rats exposed to ammonium metavanadate in drinking water at concentrations of 23 to 29 mg/kg body weight for a period of 2 to 8 weeks to experience loss of appetite and thirst, diarrhea, and subsequent weight loss (Zaporowska and Wasilewski, 1989, as cited in NLM, 1996). Oral administration of 0.2 mg/mL vanadate over a four-day period was reported to reduce blood glucose levels and hypoglycemia was not observed in the rats for at least three weeks (Meyerovitch et al., 1987, as cited in NLM, 1996). The no adverse effects were observed in rats continuously exposed to vanadium pentoxide at a concentration of 0.002 mg/m³ for 70 days (Pazynich, 1966, as cited in Waters, 1977). In another study, a subchronic NOAEL of 17.9 parts per million (ppm) vanadium pentoxide was reported for rats (EPA, 1996). Based on exposure of rats to sodium metavanadate, Opresko et al. (1994) estimated oral NOAELs for the white-footed mouse, cottontail rabbit, and red fox to be 0.47, 0.13, and 0.08 mg/kg/d, respectively. Drinking water NOAELs ranged from 0.54 to 2.45 mg/kg/d for various mammals (Opresko et al., 1994). Likewise, oral NOAELs were estimated for wild birds exposed to vanadyl sulfate based on an estimated NOAEL for the mallard of 11.4 mg/kg/d (Opresko et al., 1994). NOAELs for the great blue heron and red-tailed hawk were approximated at 9.0 and 11.5 mg/kg/d, respectively. The drinking water NOAEL for these species is 203 mg/L. Signs of acute toxicity in animals include alterations in nervous system responses, gastrointestinal distress, hemorrhaging, paralysis, convulsions, and respiratory depression (Hammond and Beliles, 1980).

Background concentrations of vanadium in freshwater fish are usually less than 2.5 mg/kg (wet weight) (Jenkins, 1980). A bioconcentration factor of 3,000 has been listed for aquatic invertebrates exposed to vanadium (Neumann, 1976).

The US EPA benchmark for vanadium is 0.019 mg/L (EPA, 1996); however, there is not Ohio EPA Warmwater Habitat Water Quality Criteria. Suter and Mabrey (1994) have estimated acute and chronic advisory levels for vanadium to be 28.4 and 19.1 µg/L, respectively. The lowest chronic values of vanadium reported in the literature for fish and *Daphnia* are 80 µg/L and greater than 940 µg/L, respectively (Suter and Mabrey, 1994). The test EC₂₀ for fish can be used as a benchmark

indicative of production within a population. It is the highest tested concentration of vanadium causing less than 20 percent reduction in either the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). The value for vanadium is 41 $\mu\text{g/L}$ (Suter and Mabrey, 1994). A similar value can be determined for daphnids, which reflects the highest tested concentration of vanadium causing less than 20 percent reduction in the product of growth, fecundity, and survivorship in a chronic test with a daphnid species. The EC_{20} benchmark for daphnids is 430 $\mu\text{g/L}$ (Suter and Mabrey, 1994).

REFERENCES

- Bertrand, D., 1950, *Bulletin American Museum of Natural History*, Vol. 94, p. 403.
- Hammond, P. B., and R. P. Beliles, 1980, "Metals," in J. Doull, C. D. Klaassen, and M. O. Amdur (eds.), *Casarett and Doull's Toxicology: The Basic Science of Poisons*, 2nd ed., Macmillan Publishing Co., Inc., New York, New York, pp. 409-4671.
- Jenkins, D. W., 1980, "Biological Monitoring of Toxic Trace Metals, Volume 2: Toxic Trace Metals in Plants and Animals of the World, Part I," *EPA Report 600/3-80-090*, U.S. Environmental Protection Agency, Washington, D.C., 503 pp.
- Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.
- Lagerkvist, B., G. F. Nordberg, and V. Vouk, 1986, Vanadium, in *Handbook on the Toxicology of Metals*, L. Friberg, G. F. Nordberg, and V. B. Vouk, eds., 2nd ed., Vol. 2: Specific Metals, Elsevier Science Publishers B.V., New York, NY, pp. 638-663.
- Lauchli, A., and R. L. Bielecki, 1983, *Inorganic Plant Nutrition*, Springer-Verlag, Berlin.
- Lepp, N. W., C. S. Harrison, and B. G. Morrell, 1987, "A Role of *Amanita muscaria* L. in the Circulation of Cadmium and Vanadium in a Nonpolluted Woodland," *Environment Geochemistry and Health*, Vol. 9, pp. 61.
- Meyerovitch, J., et al., 1987, *Journal of Biological Chemistry*, Vol. 262, pp. 6658-6662.
- National Library of Medicine (NLM), 1996, "Hazardous Substance Data Bank," produced by Micromedex, Inc.
- National Research Council (NRC), 1977, "Drinking Water and Health Volume 1," National Academy Press, Washington, D.C., 248 pp.
- National Research Council Canada (NRCC), 1980, "Effects of Vanadium in the Canadian Environment," No. 18132, National Research Council, Canada.
- Neumann, G., 1976, "Concentration Factors for Stable Metals and Radionuclides in Fish, Mussels and Crustaceans—A Literature Survey," National Swedish Environmental Protection Board, Sweden.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Pazynich, V. M., 1966, *Gig. Sanit (Moscow)* (English ed.), Vol. 31, pp. 6.

Pearson, C. R., and G. McConnell, 1975, *Proceedings of the Royal Society of London*, Series B, Vol. 189, pp. 305-332.

Schroeder, H. A., 1970, "Vanadium," *Air Quality Monographs*, Monograph No. 70-13, American Petroleum Institute, Washington, D.C.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

U.S. Environmental Protection Agency (EPA), 1996, *Ecotox Thresholds*, EPA 540/F-95/038.

U.S. Environmental Protection Agency (EPA), 1995, *Health Effects Assessment Summary Tables. Annual Update FY 1995, including Supplements*. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1989, *Risk Assessment Guidance for Superfund, Vol. 1: Human Health Evaluation Manual, Part A*. Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-89/002.

Waters, M. D., 1977, "Toxicology of Vanadium," in *Advances in Modern Toxicology, Vol. 2: Toxicology of Trace Elements*, R. A. Goyer and M. A. Mehlman (eds.), John Wiley & Sons, New York, New York, pp. 147-189.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Zaporowska, H., and W. Wasilewski, 1989, *Comparative Biochemistry and Physiology*, (C) 93, pp. 175-180.

VINYL CHLORIDE

Human Health Effects

Vinyl chloride is a volatile, colorless gas which is soluble both in water and organic solvents. It was of little toxicological interest until 1974, when it was first associated with human carcinogenic effects. The available toxicological information has been summarized by Torkelson and Rowe (1981), EPA (1984), Williams and Weisburger (1986), NLM (1996) and ATSDR (1988)

In air, the primary fate mechanism is photochemical breakdown, with an estimated half-life of 1.2 to 1.8 days. Vinyl chloride in water and surface soil quickly volatilizes and does not redeposit. It does not adsorb strongly to soil or sediment and is highly mobile in soil and can leach into groundwater. Once vinyl chloride reaches ground water, the degradation is relatively slow. The EPA has estimated a half-life range of eight weeks to 96 months for vinyl chloride in ground water.

The absorption rate of vinyl chloride by inhalation is 42 percent. It is virtually completely absorbed following ingestion, but dermal absorption is negligible. Vinyl chloride is concentrated in the liver and kidney. Vinyl chloride is oxidized in the liver to an epoxide and other reactive intermediates, which react further. These intermediates are generally believed to be the active chemical species for the specific toxic effects of vinyl chloride. Excretion is primarily in the urine as conjugates of metabolites with sulfur-containing compounds. Very small amounts are exhaled unchanged.

Vinyl chloride exhibits both acute and chronic effects. Large single doses of vinyl chloride produce central nervous system depression. Early studies of its anesthetic potential found cardiac and circulatory disturbances. Repeated low doses in workers produce a syndrome called "vinyl chloride disease" which is characterized by acroosteolysis, also known as Raynaud's phenomenon, (scleroderma-like skin changes and x-ray evidence of bone destruction of the distal finger bones); lung toxicity; thrombocytopenia; and liver toxicity. Chromosomal abnormalities are reported in workers, also. Liver toxicity appears at the lowest doses.

Several human epidemiologic studies have found evidence of the carcinogenicity of this compound by inhalation exposure, including tumors in the liver, central nervous system, digestive tract, respiratory tract, and lymph and hematopoietic system. The EPA has, therefore, classified vinyl chloride as a Group A, known human carcinogen. There have been reports of reproductive toxicity in exposed workers, but no adverse effects have been seen in animal studies except at high doses that produce non-specific toxic effects. Epidemiological studies in the neighborhoods of vinyl chloride plants have been inconclusive.

Ecological Effects

There are no data on the aquatic toxicity of vinyl chloride. Its high volatility, low aquatic bioconcentration factor, and low half-life in water, greatly decreases the possibility of any adverse effects.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR), 1988, Draft Toxicological Profile for Vinyl Chloride, U.S. Public Health Service, Atlanta, GA.

National Library of Medicine (NLM), 1996, Hazardous Substances Databank File, Toxicology Information Network (TOXNET).

Torkelson, T.R. and V.K. Rowe, 1981, Halogenated Aliphatic Hydrocarbons Containing Chlorine, Bromine, and Iodine, in Patty's Industrial Hygiene and Toxicology, George D. Clayton and Florence E. Clayton (Editors), Third Revised Edition, Volume 2B. New York, John Wiley & Sons.

U.S. Environmental Protection Agency (EPA), 1984, Health Effects Assessment for Vinyl Chloride. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-H036.

Williams, G.M. and J.H. Weisburger, 1986, Chemical Carcinogens, in Casarett and Doull's Toxicology. C.D. Klaassen, M.O. Amdur, and J. Doull (Editors), Third Edition, New York, Macmillan.

ZINC

Human Health Effects

Zinc is a nutritionally required trace element. Estimates of the efficiency of GI absorption of zinc in animals range from < 10 to 90 percent (Elinder, 1986). Estimates in normal humans range from approximately 20 to 77 percent (Elinder, 1986; Goyer, 1991). The net absorption of zinc appears to be homeostatically controlled, but it is unclear whether GI absorption, intestinal secretion, or both are regulated. Distribution of absorbed zinc is primarily to the liver (Goyer, 1991), with subsequent redistribution to bone, muscle, and kidney (Elinder, 1986). Highest tissue concentrations are found in the prostate. Excretion appears to be principally through the feces, in part from biliary secretion, but the relative importance of fecal and urinary excretion is species-dependent. The half-life of zinc absorbed from the GI tracts of humans in normal zinc homeostasis is approximately 162 to 500 days.

Humans exposed to high concentrations of aerosols of zinc compounds may experience severe pulmonary damage and death (Elinder, 1986). The usual occupational exposure is to freshly formed fumes of zinc, which can induce a reversible syndrome known as metal fume fever. Orally, zinc exhibits a low order of acute toxicity. Animals dosed with 100 times dietary requirement showed no evidence of toxicity (Goyer, 1991). In humans, acute poisoning from foods or beverages prepared in galvanized containers is characterized by GI upset (Elinder, 1986). Chronic oral toxicity in animals is associated with poor growth, GI inflammation, arthritis, lameness, and a microcytic, hypochromic anemia (Elinder, 1986), possibly secondary to copper deficiency (Underwood, 1977). The EPA (1996) presented a verified RfD of 0.3 mg/kg/day for chronic oral exposure to zinc, based on anemia in humans.

The EPA (1995) classifies zinc in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) based on inadequate evidence for carcinogenicity in humans and animals. The human data consist largely of occupational exposure studies not designed to detect a carcinogenic response, and of reports that prostatic zinc concentrations were lower in cancerous than in noncancerous tissue. The animal data consist of several dietary, drinking water, and zinc injection studies, none of which provided convincing data for a carcinogenic response.

Ecological Effects

Background concentrations of zinc in terrestrial plants range from 25 to 150 mg/kg (dry weight) (NAS, 1979). The deficiency content of zinc in plants is between 10 and 20 ppm (dry weight) (Kabata-Pendias and Pendias, 1992). Fungi tend to contain higher concentrations of zinc than lichens, mosses, and vascular plants (Kabata-Pendias and Pendias, 1992). Roots often contain the highest concentrations of zinc (Kabata-Pendias and Pendias, 1992).

Certain species of plants, particularly those from the families Caryophyllaceae, Cyperaceae, and Plumbaginaceae, and some tree species are extremely tolerant to elevated zinc concentration and thereby serve as good indicators of zinc-contaminated environments (Kabata-Pendias and Pendias, 1992). Concentrations of zinc in these plants may reach 1 percent (dry weight) in the plant. Concentrations in leaf tissue that are excessive or toxic to various plant species, with the exclusion of very sensitive and highly tolerant species, range from 100 to 400 mg/kg (dry weight) (Kabata-Pendias and Pendias, 1992). Concentrations of 100 to 500 mg/kg (dry weight) are expected to result in a 10 percent loss in crop yield (Kabata-Pendias and Pendias, 1992). A soil concentration of 50 mg/kg (dry weight) has been proposed by Will and Suter (1994) as a benchmark screening value for phytotoxicity. General symptoms of zinc toxicity in plants include the presence of chlorotic and necrotic leaf tips, interveinal chlorosis in new leaves, retarded growth of the entire plant, and injured roots that resemble barbed wire (Kabata-Pendias and Pendias, 1992).

Zinc is an essential trace element for animal life. Background concentrations of zinc in mammals and birds are usually less than 210 mg/kg (dry weight). The concentration of zinc in an animal can be influenced by the animal's age, gender, and season. Elevated levels of zinc have been measured in birds and mammals collected near zinc smelters (Beyer, 1988, as cited in Eisler, 1993; Beyer et al., 1985).

Animals are quite tolerant to high concentrations of zinc in the diet. Levels 100 times that required in the diet usually do not cause detectable symptoms of toxicosis (NAS, 1979). Examples of extrapolated NOAELs for chronic exposure of various mammalian wildlife species to zinc oxide based on an estimated rat NOAEL of 160 mg/kg/d are 399 mg/kg/d for the white-footed mouse, 107 mg/kg/d cottontail rabbit, and

68.9 mg/kg/d for the red fox (Opresko et al., 1994). Drinking-water NOAELs for these species are 1,329, 1,102, and 816 mg/L, respectively (Opresko et al., 1994). Adverse effects were noted in laboratory mice and rats exposed to zinc in drinking water at concentrations of 300 mg/L (chronic exposure) and 800 mg/L (acute exposure), respectively (USPHS, 1989). Guinea pigs (*Cavia* spp.) experienced difficulty in breathing after exposed to zinc concentrations of 0.8 mg/m³ for one hour (USPHS, 1989). Symptoms of zinc poisoning in mammals include lameness, acute diarrhea, and vomiting (Eisler, 1993).

With reference to birds, dietary zinc concentrations of greater than 2,000 mg/kg diet are known to result in reduced growth of domestic poultry and wild birds (Eisler, 1993). Reduced survival has been documented at zinc concentrations greater than 3,000 mg/kg diet or at a single oral dose of greater than 742 mg/kg body weight (Eisler, 1993). Examples of extrapolated NOAELs for chronic exposure of various avian wildlife species to zinc carbonate (based on an estimated NOAEL of 3 mg/kg/L for a mallard) are 2.25 mg/kg/d for the great blue heron and 2.89 mg/kg/d for the red-tailed hawk (Opresko et al., 1994). A value of 51 mg/L has been calculated as the NOAEL for chronic exposure of birds to zinc carbonate in drinking water (Opresko et al., 1994). Diarrhea and leg paralysis have been noted in mallards exposed to toxic concentrations of zinc (Gasaway and Buss, 1972).

Background concentrations of zinc in fish tissue are usually less than 700 mg/kg (dry weight). Zinc concentrations are often higher in fish collected near urban areas (Peterson et al., 1989). Concentrations of zinc in aquatic vertebrates can be modified by diet, age, and the reproductive state of the animal (Eisler, 1993). Molluscs tend to bioconcentrate zinc. Molluscs, crustaceans, and aquatic annelids have the ability to store zinc within their bodies. Bioconcentration factors for zinc range from 107 to 1,130 for freshwater insects and from 51 to 432 for freshwater fish (EPA, 1980, as cited in Eisler, 1993). The half-life of zinc in whole mosquitofish (*Gambusia affinis*) has been estimated to be 215 days (Newman and Mitz, 1988).

The bioavailability and toxicity of zinc to aquatic organisms is greatest under the conditions of low pH, low alkalinity, low dissolved oxygen, and elevated temperatures (Weatherley et al., 1980). Decreased water hardness can also increase the toxicity of

zinc to aquatic biota. Freshwater insects and crustaceans are, in general, relatively tolerant to zinc (Eisler, 1993). Elevated levels of zinc have been shown to be teratogenic to frogs and fish (Eisler, 1993).

The EPA's National Ambient Water Quality Criteria for zinc in freshwater is 120 $\mu\text{g/L}$ for acute exposure and 110 $\mu\text{g/L}$ for chronic exposure of aquatic life to zinc (based on a water hardness of 100 mg/L) (EPA, 1996). Because the toxicity of zinc to aquatic organisms is affected by water hardness, all water-quality criteria must be adjusted with site-specific water hardness data. The Ohio EPA Warmwater Habitat Water Quality Criteria for zinc is 0.059 mg/L . The lowest chronic values of zinc reported in the literature for fish and *Daphnia* are 36.41 and 46.73 $\mu\text{g/L}$, respectively (Suter and Mabrey, 1994). The test EC_{20} for fish can be used as a benchmark indicative of production within a population. It is the highest tested concentration causing less than 20 percent reduction in the weight of young fish per initial female fish in a life cycle or partial life-cycle test or the weight of young per egg in an early life-stage test (Suter and Mabrey, 1994). This value is 47 $\mu\text{g/L}$ for zinc (Suter and Mabrey, 1994).

REFERENCES

Beyer, W. N., 1988, "Damage to the Forest Ecosystem on Blue Mountain from Zinc Smelting," in D. D. Hemphill, ed. *Trace Substances in Environment Health-XXII: A Symposium*, University of Missouri Press, Columbia, pp. 249-262.

Beyer, W. N., O. H. Pattee, L. Sileo, D. J. Hoffman, and B. M. Mulhern, 1985, "Metal Contamination in Wildlife Living Near Two Zinc Smelters," *Environmental Pollution Vol. 38A*, pp. 63-86.

Eisler, R., 1993, "Zinc Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review," U.S. Fish and Wildlife Service Contaminant Hazard Review, *Report No. 26*, U.S. Department of the Interior, Washington, D.C.

Elinder, C. G., 1986, Zinc, *Handbook on the Toxicology of Metals*, L. Friberg, G. F. Nordberg, and V. B. Vouk, eds., Vol. II, 2nd ed., Elsevier Science Publishers B.V., New York, pp. 664-679.

Gasaway, W. C., and I. O. Buss, 1972, "Zinc Toxicity in the Mallard Duck," *Journal of Wildlife Management*, Vol. 36, pp. 1107-1117.

Goyer, R. A., 1991, Toxic Effects of Metals, Casarett and Doull's Toxicology, the Basic Science of Poisons, M. O. Amdur, J. Doull, and C. D. Klaassen, eds., 4th ed., Pergamon Press, New York.

Kabata-Pendias, A., and H. Pendias, 1992, *Trace Elements in Soils and Plants*, 2nd ed., CRC Press, Boca Raton, Florida, 365 pp.

National Academy of Sciences (NAS), 1979, *Zinc*, University Press, Baltimore, Maryland, 471 pp.

Newman, M. C., and S. V. Mitz, 1988, "Size Dependence of Zinc Elimination and Uptake from Water by Mosquitofish (*Gambusia affinis*)," (Baird and Girard), *Aquatic Toxicology*, Vol. 12, pp. 17-31.

Opresko, D. M., B. E. Sample, and G. W. Suter, 1994, "Toxicological Benchmarks for Wildlife: 1994 Revision," *ES/ER/TM-86/RI*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Peterson, R. H., A. Sreedharan, and S. Ray, 1989, "Accumulation of Trace Metals in Three Species of Fish from Lakes in New Brunswick and Nova Scotia (Canada): Influence of pH and Other Chemical Parameters," *Water Pollution Research Journal of Canada*, Vol. 24, Canada, pp. 101-117.

Suter, G. W., II, and J. B. Mabrey, 1994, "Toxicological Benchmarks for Screening of Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision," *ES/ER/TM-96/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Underwood, E. J., 1977, *Trace Elements in Human and Animal Nutrition*. Fourth Edition. New York: Academic Press.

U.S. Environmental Protection Agency (EPA), 1996, *Integrated Risk Information System (IRIS)*. Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1995, *Health Effects Assessment Summary Tables. Annual Update FY 1995, including Supplements*. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA), 1980, "Ambient Water Quality Criteria for Zinc," *Report 440/5-87-003*, U.S. Environmental Protection Agency, Washington, D.C., 207 pp.

U.S. Public Health Service (USPHS), 1989, "Toxicological Profile for Zinc," U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia, 121 pp.

Weatherley, A. H., P. S. Lake, and S. C. Rogers, 1980, "Zinc Pollution and the Ecology of the Freshwater Environment," in J. O. Nriagu, ed., *Zinc in the Environment. Part I, Ecological Cycling*, John Wiley, New York, New York, pp. 337-417.

Will, M. E., and G. W. Suter II, 1994, "Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1994 Revision," *ES/ER/TM-85/R1*, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

TAB

APPENDIX C

Appendix C

Risk and Hazard Calculations

Appendix C-1

Unit Risk and Unit Hazard Calculations

Table C.1
 Unit Risk and Unit Hazard Calculations
 RME Construction Worker Noncancer Unit Risk from Exposure to Unit Concentrations of COPCs (mg/L) in Groundwater

Chemical name	Lifetme (HQ) (unitless)	IPRGs Based on All Pathways (mg/L)	Depletion Coefficients				Physical Data				Basic Information				G/Abs Factor (unitless)	Bio-transfer Coefficients (a)	Soil Forage B/Grass (unitless)
			Chem. Half-life (yr)	Chem. Degradation (1/h)	Soil Leach (cm ² /g)	Soil Leach (1/h)	Henry's Law Const. (atm-m ³ /mol)	Molec. Weight (g/m)	Permeab. Constant (cm/h)	Lag Time (h)	Time to Equilib. (h)	Partic. Coeff. (unitless)	Time to Equilib. (h)	Time to Equilib. (h)			
VOLATILE ORGANIC CHEMICALS																	
Acetone	15E-2	7 E-5	10E-3	2 7E-3	1 7E-3	2 1E-5	58 08	5 68E-4	2 0E-1	4 7E-1	5 8E-5	1 0E+0	1 0E+0	1 0E+0	2 27E+0		
Carbon tetrachloride	6 8E+0	1 E-7	1 0E-3	3 8E+0	5 1E-5	3 0E-2	154 00	2 20E-2	7 6E-1	1 1E+2	6 7E-2	1 0E+0	1 0E+0	1 0E+0	1 20E+0		
Chloroform	2 8E-1	4 E-6	1 6E-4	1 8E+0	1 0E-4	3 4E-3	119 38	8 92E-3	4 7E-1	1 1E+0	9 3E-3	1 0E+0	1 0E+0	1 0E+0	2 27E+0		
Cis-1,2-Dichloroethene	2 2E-1	5 E-6	4 0E-5	5 9E-1	2 8E-4	7 6E-3	96 94	5 30E-3	3 4E-1	8 4E-1	1 2E-1	1 0E+0	1 0E+0	1 0E+0	2 27E+0		
1,1-Dichloroethane	4 2E-1	2 E-6	1 6E-4	5 8E-1	2 9E-4	3 4E-2	97 00	1 60E-2	3 4E-1	1 4E+0	3 1E-2	1 0E+0	1 0E+0	1 0E+0	2 40E+0		
1,2-Dichloroethene (Total)	2 4E-1	4 E-6	4 0E-5	5 9E-1	2 8E-4	7 6E-3	96 94	5 30E-3	3 4E-1	8 4E-1	1 2E-1	1 0E+0	1 0E+0	1 0E+0	2 27E+0		
1,2-Dichloroethene, Trans-	1 1E-1	9 E-6	4 0E-5	5 9E-1	2 8E-4	6 6E-3	96 94	5 49E-3	3 4E-1	8 2E-1	3 0E-3	1 0E+0	1 0E+0	1 0E+0	2 27E+0		
Benzene			4 0E-5	2 6E+0	7 3E-5	5 6E-3	78 11	2 10E-2	2 6E-1	6 3E-1	1 3E-2	1 0E+0	1 0E+0	1 0E+0	2 27E+0		
Ethylbenzene	4 9E-1	2 E-6	1 3E-4	2 7E+1	7 3E-6	8 5E-3	106 17	7 40E-2	3 9E-1	1 3E+0	1 4E-1	1 0E+0	1 0E+0	1 0E+0	5 88E-1		
2-Hexanone				1 2E-1	8 5E-4		100 16	4 45E-3	3 6E-1	8 6E-1	2 4E-3	1 0E+0	1 0E+0	1 0E+0	5 88E-1		
Methyl ethyl ketone	3 7E-1	3 E-6	1 0E-4	1 1E-2	1 6E-3	2 7E-5	72 00	5 00E-3	2 4E-1	5 8E-1	1 9E-4	1 0E+0	1 0E+0	1 0E+0	2 27E+0		
4-Methyl-2-Pentanone				1 0E-3	3 8E-1	4 0E-4	100 16	4 67E-4	3 6E-1	8 6E-1	1 0E-4	1 0E+0	1 0E+0	1 0E+0	5 88E-1		
Methylene Chloride	1 6E-1	6 E-6	1 0E-3	8 5E-2	1 0E-3	3 2E-3	84 93	4 46E-3	2 9E-1	6 9E-1	1 8E-3	1 0E+0	1 0E+0	1 0E+0	5 88E-1		
M.P. Xylene	6 3E-3	2 E-4	7 9E-5	2 4E+0	7 9E-5	7 2E-3	106 16	8 00E-2	3 9E-1	1 4E+0	1 6E-1	1 0E+0	1 0E+0	1 0E+0	6 77E-1		
1,1,1-Trichloroethane	4 9E-1	2 E-6	1 3E-5	5 7E+0	2 6E-5	1 7E-1	133 00	1 70E-2	5 7E-1	1 4E+0	3 1E-2	1 0E+0	1 0E+0	1 0E+0	1 40E+0		
Tetrachloroethene	9 3E-1	1 E-6	1 8E-5	6 5E+0	3 0E-5	1 8E-2	165 83	4 80E-2	9 0E-1	4 3E+0	2 5E-1	1 0E+0	1 0E+0	1 0E+0	4 20E-1		
Toluene	9 8E-1	1 E-6	1 4E-4	9 4E+0	2 1E-5	6 6E-3	92 14	4 50E-2	3 2E-1	7 7E-1	5 4E-2	1 0E+0	1 0E+0	1 0E+0	1 02E-1		
Total Xylenes	5 1E-3	2 E-4	7 9E-5	2 1E+1	9 4E-6	7 0E-3	106 16	6 18E-2	3 9E-1	1 0E+0	1 1E-1	1 0E+0	1 0E+0	1 0E+0	6 77E-1		
Trichloroethene			1 8E-5	6 5E+0	3 0E-5	1 0E-2	131 39	1 60E-2	5 5E-1	1 3E+0	2 5E-2	1 0E+0	1 0E+0	1 0E+0	1 59E+0		
Vinyl chloride			1 0E-5	7 6E+0	2 6E-5	1 1E-2	62 50	7 30E-3	2 1E-1	5 5E-1	2 3E-3	1 0E+0	1 0E+0	1 0E+0	6 34E+0		

661 325

Table C.1 (continued)
 Unit Risk and Unit Hazard Calculations
 RME Construction Worker Noncancer Unit Risk from Exposure to Unit Concentrations of COPCs (mg/L) in Groundwater

Chemical name	Basic Information			Direct Exposure Pathways						
	Oral RFD (kg-d/mg)	Dermal RFD (kg-d/mg)	Inhalation RFD (mg/L)	Water Conc (mg/L)	Amount Ingested (mg/kg-d)	Lifetime HQ (unitless)	Amount Absorbed (mg/kg-d)	Lifetime HQ (unitless)	Amount Inhaled (mg/kg-d)	Lifetime HQ (unitless)
VOLATILE ORGANIC CHEMICALS										
Acetone	1.0E-1	1.00E-1		1.0E+00	1.4E-3	1.4E-2	7.9E-5	7.9E-4	1.1E-1	
Carbon tetrachloride	7.0E-4	7.00E-4		1.0E+00	1.4E-3	2.0E+0	3.3E-3	4.8E+0	1.1E-1	
Chloroform	1.0E-2	1.00E-2		1.0E+00	1.4E-3	1.4E-1	1.4E-3	1.4E-1	1.1E-1	
Cis-1,2-Dichloroethene	1.0E-2	1.00E-2		1.0E+00	1.4E-3	1.4E-1	7.3E-4	7.3E-2	1.1E-1	
1,2-Dichloroethane				1.0E+00	1.4E-3		7.4E-4		1.1E-1	
1,1-Dichloroethene	9.0E-3	9.00E-3		1.0E+00	1.4E-3	1.6E-1	2.3E-3	2.6E-1	1.1E-1	
1,2-Dichloroethene (Total)	9.0E-3	9.00E-3		1.0E+00	1.4E-3	1.6E-1	7.3E-4	8.1E-2	1.1E-1	
1,2-Dichloroethene, Trans-	2.0E-2	2.00E-2		1.0E+00	1.4E-3	7.1E-2	8.1E-4	4.0E-2	1.1E-1	
Benzene				1.0E+00	1.4E-3		3.0E-3		1.1E-1	
Ethylbenzene	1.0E-1	1.00E-1	2.9E-1	1.0E+00	1.4E-3	1.4E-2	1.0E-2	1.0E-1	1.1E-1	3.7E-1
2-Hexanone				1.0E+00	1.4E-3		6.6E-4			
Methyl ethyl ketone	6.0E-1	6.00E-1	2.9E-1	1.0E+00	1.4E-3	2.4E-3	7.0E-4	1.2E-3	1.1E-1	3.7E-1
4-Methyl-2-Pentanone				1.0E+00	1.4E-3		6.9E-5			
Methylene Chloride	6.0E-2	6.00E-2	8.6E-1	1.0E+00	1.4E-3	2.4E-2	6.4E-4	1.1E-2	1.1E-1	1.2E-1
M,P Xylene	2.0E+0	2.00E+0		1.0E+00	1.4E-3	7.1E-4	1.1E-2	5.6E-3	1.1E-1	
1,1,1-Trichloroethane	3.5E-2	3.50E-2	2.9E-1	1.0E+00	1.4E-3	4.1E-2	2.7E-3	7.8E-2	1.1E-1	3.7E-1
Tetrachloroethene	1.0E-2	1.00E-2		1.0E+00	1.4E-3	1.4E-1	7.9E-3	7.9E-1	1.1E-1	
Toluene	2.0E-1	2.00E-1	1.1E-1	1.0E+00	1.4E-3	7.1E-3	6.4E-3	3.2E-2	1.1E-1	9.4E-1
Total Xylenes	2.0E+0	2.00E+0		1.0E+00	1.4E-3	7.1E-4	8.8E-3	4.4E-3	1.1E-1	
Trichloroethene				1.0E+00	1.4E-3		2.5E-3		1.1E-1	
Vinyl chloride				1.0E+00	1.4E-3		1.0E-3		1.1E-1	

Table C.2
Unit Risk and Hazard Calculations
RME Resident Adult Cancer Unit Risk From Unit Concentrations (mg/L) of COPCs in Groundwater

Chemical Name	Lifeline Risks (ILCR)	PRGs Based On/All Pathways (mg/L)	Depletion/Coefficients			Physical Data		Dermal Uptake Data			Bio-transfer Coefficients (a)			
			Chem. Degradation Half-life (yr)	Decay (1/h)	Soil Leaching K _d (cm ² /g)	Henry's Law Constant (atm-m ³ /mol)	Molec. Weight (g/M)	Permeability Constant K _p (cm/h)	Lag Time (TAO) (unitless)	Time to Equilib (hr)	Partic. Coeff. B (unitless)	GI Abs. Factor (unitless)	Soil Crop Bt Crop (unitless)	
VOLATILE ORGANIC CHEMICALS														
Acetone				1.0E-3	2.7E-3	1.7E-3	2.1E-5	58.08	5.99E-4	2.0E-1	4.7E-1	5.8E-5	1.0E+0	2.27E+0
Carbon tetrachloride	4.8E-3	2 E-4		1.0E-3	3.8E+0	5.1E-5	3.0E-2	154.00	2.20E-2	7.6E-1	1.8E+0	6.7E-2	1.0E+0	1.20E+0
Chloroform	8.3E-5	1 E-2		1.6E-4	1.8E+0	1.0E-4	3.4E-3	119.38	8.92E-3	4.7E-1	1.1E+0	9.3E-3	1.0E+0	2.27E+0
Cis-1,2-Dichloroethene				1.0E-4	5.9E-1	2.8E-4	7.6E-3	96.94	5.30E-3	3.4E-1	8.2E-1	1.2E-1	1.0E+0	2.27E+0
1,2-Dichloroethane	5.9E-3	2 E-4		4.0E-5	2.4E+0	8.0E-5	9.8E-4	99.00	5.30E-3	3.5E-1	8.4E-1	1.2E-1	1.0E+0	2.40E+0
1,1-Dichloroethene	1.8E-2	6 E-5		1.6E-4	5.8E-1	2.9E-4	3.4E-2	97.00	1.60E-2	3.4E-1	1.4E+0	3.1E-2	1.0E+0	2.27E+0
1,2-Dichloroethene, Trans-					5.9E-1	2.8E-4	6.6E-3	96.94	5.49E-3	3.4E-1	8.2E-1	3.0E-3	1.0E+0	2.27E+0
Benzene	1.9E-3	5 E-4		4.0E-5	2.6E+0	7.3E-5	5.6E-3	78.11	2.10E-3	2.6E-1	6.3E-1	1.3E-2	1.0E+0	2.27E+0
Ethylbenzene				1.3E-4	2.7E+1	7.3E-6	8.5E-3	106.17	7.40E-3	3.9E-1	1.3E+0	1.4E-1	1.0E+0	5.88E-1
2-Hexanone					1.2E-1	8.5E-4		100.16	4.45E-3	3.6E-1	8.6E-1	2.4E-3	1.0E+0	2.27E+0
Methyl ethyl ketone				1.0E-4	1.1E-2	1.6E-3	2.7E-5	72.00	5.00E-3	2.4E-1	5.8E-1	1.9E-4	1.0E+0	2.27E+0
4-Methyl-2-Pentanone				1.0E-3	3.8E-1	4.0E-4		100.16	4.67E-4	3.6E-1	8.6E-1	1.0E-4	1.0E+0	2.27E+0
Methylene Chloride	1.8E-4	6 E-3		1.0E-3	8.5E-2	1.0E-3	3.2E-3	84.93	4.46E-3	2.9E-1	6.9E-1	1.8E-3	1.0E+0	2.27E+0
M.P. Xylene				7.9E-5	2.4E+0	7.9E-5	7.2E-3	106.16	8.00E-2	3.9E-1	1.4E+0	1.6E-1	1.0E+0	6.77E-1
1,1,1-Trichloroethane				1.3E-5	5.7E+0	2.6E-5	1.7E-2	133.00	1.70E-2	5.7E-1	1.4E+0	3.1E-2	1.0E+0	1.45E+0
Tetrachloroethene	1.2E-3	9 E-4		1.8E-5	6.5E+0	3.0E-5	1.8E-2	165.83	4.80E-2	9.0E-1	4.3E+0	2.5E-1	1.0E+0	4.20E-1
Toluene				1.4E-4	9.4E+0	2.1E-5	6.6E-3	92.14	4.50E-2	3.2E-1	7.7E-1	5.4E-1	1.0E+0	1.02E+0
Total Xylenes				7.9E-5	2.1E+1	9.4E-6	7.0E-3	106.16	6.18E-2	3.9E-1	1.0E+0	1.1E-1	1.0E+0	6.77E-1
Trichloroethene	4.7E-4	2 E-3		1.8E-5	6.5E+0	3.0E-5	1.0E-2	131.39	1.60E-2	5.5E-1	1.3E+0	2.5E-2	1.0E+0	1.59E+0
Vinyl chloride	4.1E-2	2 E-5		1.0E-5	7.6E+0	2.6E-5	1.1E-2	62.50	7.30E-3	2.1E-1	5.1E-1	2.3E-3	1.0E+0	6.34E+0

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Table C.2 (continued)
 Unit Risk and Hazard Calculations
 RME Resident Adult Cancer Unit Risk From Unit Concentrations (mg/L) of COPCs in Groundwater

Chemical Name	Basic Information			Direct Exposure Pathways			Household Water Use			Volatile Inhalation		
	Cancer Potency Factors		Water Conc (mg/L)	Ingestion		Dermal Absorption		Inhaled		Lifetime Risks (ILCR)		Lifetime Risks (ILCR)
	Oral Slope Factor	Dermal Slope Factor		Amount Ingested (mg/kg-d)	Lifetime Risks (ILCR)	Amount Absorbed (mg/kg-d)	Lifetime Risks (ILCR)	Amount Inhaled (mg/kg-d)	Lifetime Risks (ILCR)			
VOLATILE ORGANIC CHEMICALS												
Acetone			1.0E+00	1.3E-2		4.4E-5		5.2E-2				
Carbon tetrachloride	1.30E-1	1.30E-1	1.0E+00	1.3E-2	1.6E-3	3.3E-3	4.3E-4	5.2E-2	4.3E-4	5.2E-2	2.8E-3	
Chloroform	6.10E-3	6.10E-3	1.0E+00	1.3E-2	7.6E-5	1.1E-3	6.5E-6	5.2E-2	6.5E-6	5.2E-2		
Cis-1,2-Dichloroethene			1.0E+00	1.3E-2		5.4E-4		5.2E-2				
1,2-Dichloroethane	9.10E-2	9.10E-2	1.0E+00	1.3E-2	1.1E-3	5.4E-4	4.9E-5	5.2E-2	4.9E-5	5.2E-2	4.8E-3	
1,1-Dichloroethene	6.00E-1	6.00E-1	1.0E+00	1.3E-2	7.5E-3	1.6E-3	9.7E-4	5.2E-2	9.7E-4	5.2E-2	9.4E-3	
1,2-Dichloroethene, Trans-			1.0E+00	1.3E-2		5.5E-4		5.2E-2				
Benzene	2.90E-2	2.90E-2	1.0E+00	1.3E-2	3.6E-4	1.9E-4	5.4E-6	5.2E-2	5.4E-6	5.2E-2	1.5E-3	
Ethylbenzene			1.0E+00	1.3E-2		8.0E-4		5.2E-2				
2-Hexanone			1.0E+00	1.3E-2		4.6E-4		5.2E-2				
Methyl ethyl ketone			1.0E+00	1.3E-2		4.2E-4		5.2E-2				
4-Methyl-2-Pentanone			1.0E+00	1.3E-2		4.8E-5		5.2E-2				
Methylene Chloride	7.50E-3	7.50E-3	1.0E+00	1.3E-2	9.4E-5	4.2E-4	3.1E-6	5.2E-2	3.1E-6	5.2E-2	8.4E-5	
M,P Xylene			1.0E+00	1.3E-2		8.6E-3		5.2E-2				
1,1,1-Trichloroethane			1.0E+00	1.3E-2		2.2E-3		5.2E-2				
Tetrachloroethene	5.20E-2	5.20E-2	1.0E+00	1.3E-2	6.5E-4	7.9E-3	4.1E-4	5.2E-2	4.1E-4	5.2E-2	1.0E-4	
Toluene			1.0E+00	1.3E-2		4.4E-3		5.2E-2				
Total Xylenes			1.0E+00	1.3E-2		6.7E-3		5.2E-2				
Trichloroethene	1.10E-2	1.10E-2	1.0E+00	1.3E-2	1.4E-4	2.1E-3	2.3E-5	5.2E-2	2.3E-5	5.2E-2	3.1E-4	
Vinyl chloride	1.90E+0	1.90E+0	1.0E+00	1.3E-2	2.4E-2	5.8E-4	1.1E-3	5.2E-2	1.1E-3	5.2E-2	1.6E-2	

Table C.3

Unit Risk and Hazard Calculations
RME Construction Worker Cancer Unit Risk From Unit Concentrations (mg/L) of COPCs in Groundwater

Chemical name	Lifetime Risks (ILCR)	PRGs Based on All Pathways (mg/L)	Chem. Degradation		Soil Leaching		Henry's Law		Physical Data		Basic Information		Dermal Uptake Data		Time to Equilib.		Partic. Coeffic.		Bio-transfer Coefficients (g)		
			Half-life (yr)	Decay (1/h)	Leach. (1/h)	Constant (atm-m ³ /mol)	Molec. Weight (g/m)	Permeab. Constant (cm/h)	Lag Time (h)	Time to Equilib. (h)	Partic. Coeffic. (unitless)	GI/Abs Factor (unitless)	GI/Abs Factor (unitless)	GI/Abs Factor (unitless)							
VOLATILE ORGANIC CHEMICALS																					
Acetone			1.0E-3	2.7E-3	1.7E-3	2.1E-5	58.08	5.69E-4	2.0E-1	4.7E-1	5.8E-5	1.0E+0	2.27E+0								
Carbon tetrachloride	6.2E-5	2 E-2	1.0E-3	3.8E+0	5.1E-5	3.0E-2	154.00	2.20E-2	7.6E-1	1.8E+0	6.7E-2	1.0E+0	1.20E+0								
Chloroform	1.7E-7	6 E+0	1.6E-4	1.8E+0	1.0E-4	3.4E-3	119.38	8.92E-3	4.7E-1	1.1E+0	9.3E-3	1.0E+0	2.27E+0								
Cis-1,2-Dichloroethene			1.0E-4	5.9E-1	2.8E-4	7.6E-3	96.94	5.30E-3	3.4E-1	8.2E-1	1.2E-1	1.0E+0	2.27E+0								
1,2-Dichloroethane	9.7E-5	1 E-2	4.0E-5	2.4E+0	8.0E-5	9.8E-4	99.00	5.30E-3	3.5E-1	8.4E-1	1.2E-1	1.0E+0	2.40E+0								
1,1-Dichloroethene	2.1E-4	5 E-3	1.6E-4	5.8E-1	2.9E-4	3.4E-2	97.00	1.60E-2	3.4E-1	1.4E+0	3.1E-2	1.0E+0	2.27E+0								
1,2-Dichloroethene, Trans-				5.9E-1	2.8E-4	6.6E-3	96.94	5.49E-3	3.4E-1	8.2E-1	3.0E-3	1.0E+0	2.27E+0								
Benzene	3.1E-5	3 E-2	4.0E-5	2.6E+0	7.3E-5	5.6E-3	78.11	2.10E-3	2.6E-1	6.3E-1	1.3E-2	1.0E+0	2.27E+0								
Ethylbenzene			1.3E-4	2.7E+1	7.3E-6	8.5E-3	106.17	7.40E-3	3.9E-1	1.3E+0	1.4E-1	1.0E+0	5.88E-1								
2-Hexanone				1.2E-1	8.5E-4		100.16	4.45E-3	3.6E-1	8.6E-1	2.4E-3	1.0E+0	2.27E+0								
Methyl ethyl ketone			1.0E-4	1.1E-2	1.6E-3	2.7E-5	72.00	5.00E-3	2.4E-1	5.8E-1	1.9E-4	1.0E+0	2.27E+0								
4-Methyl-2-Pentanone			1.0E-3	3.8E-1	4.0E-4		100.16	4.67E-4	3.6E-1	8.6E-1	1.0E-4	1.0E+0	2.27E+0								
Methylene Chloride	1.8E-6	5 E-1	1.0E-3	8.5E-2	1.0E-3	3.2E-3	84.93	4.46E-3	2.9E-1	6.9E-1	1.8E-3	1.0E+0	2.27E+0								
M,P Xylene			7.9E-5	2.4E+0	7.9E-5	7.2E-3	106.16	8.00E-2	3.9E-1	1.4E+0	1.6E-1	1.0E+0	6.77E-1								
1,1,1-Trichloroethane			1.3E-5	5.7E+0	2.6E-5	1.7E-2	133.00	1.70E-2	5.7E-1	1.4E+0	3.1E-2	1.0E+0	1.45E+0								
Tetrachloroethene	6.8E-6	1 E-1	1.8E-5	6.5E+0	3.0E-5	1.8E-2	165.83	4.80E-2	9.0E-1	4.3E+0	2.5E-1	1.0E+0	4.20E-1								
Toluene			1.4E-4	9.4E+0	2.1E-5	6.6E-3	92.14	4.50E-2	3.2E-1	7.7E-1	5.4E-1	1.0E+0	1.02E+0								
Total Xylenes			7.9E-5	2.1E+1	9.4E-6	7.0E-3	106.16	6.18E-2	3.9E-1	1.0E+0	1.1E-1	1.0E+0	6.77E-1								
Trichloroethene	6.7E-6	1 E-1	1.8E-5	6.5E+0	3.0E-5	1.0E-2	131.39	1.60E-2	5.5E-1	1.3E+0	2.5E-2	1.0E+0	1.59E+0								
Vinyl chloride	3.6E-4	3 E-3	1.0E-5	7.6E+0	2.6E-5	1.1E-2	62.50	7.30E-3	2.1E-1	5.1E-1	2.3E-3	1.0E+0	6.34E+0								

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Table C.3 (continued)
 Unit Risk and Hazard Calculations
 RME Construction Worker Cancer Unit Risk From Unit Concentrations (mg/L) of COPCs in Groundwater

Chemical Name	Cancer Potency Factors			Basic Information			Direct Exposure Pathways							
	Oral Slope Factor	Dermal Slope Factor (r-kg-d/mg)	Inhalation Slope Factor	Water Conc. (mg/L)	Amount Ingested (mg/kg-d)	Lifetime Risks (ILCR)	Amount Absorbed (mg/kg-d)	Lifetime Risks (ILCR)	Amount Inhaled (mg/kg-d)	Lifetime Risks (ILCR)	Amount Absorbed (mg/kg-d)	Lifetime Risks (ILCR)	Amount Inhaled (mg/kg-d)	Lifetime Risks (ILCR)
VOLATILE ORGANIC CHEMICALS														
Acetone				1.0E+00	1.4E-5		7.7E-7						1.0E-3	
Carbon tetrachloride	1.30E-1	1.30E-1	5.30E-2	1.0E+00	1.4E-5	1.8E-6	3.7E-5	4.8E-6					1.0E-3	5.6E-5
Chloroform	6.10E-3	6.10E-3		1.0E+00	1.4E-5	8.5E-8	1.3E-5	8.2E-8					1.0E-3	
Cis-1,2-Dichloroethene				1.0E+00	1.4E-5		7.2E-6						1.0E-3	
1,2-Dichloroethane	9.10E-2	9.10E-2	9.10E-2	1.0E+00	1.4E-5	1.3E-6	7.2E-6	6.6E-7					1.0E-3	9.5E-5
1,1-Dichloroethene	6.00E-1	6.00E-1	1.80E-1	1.0E+00	1.4E-5	8.4E-6	2.3E-5	1.4E-5					1.0E-3	1.9E-4
1,2-Dichloroethene, Trans-				1.0E+00	1.4E-5		7.9E-6						1.0E-3	
Benzene	2.90E-2	2.90E-2	2.90E-2	1.0E+00	1.4E-5	4.1E-7	2.9E-6	8.4E-8					1.0E-3	3.0E-5
Ethylbenzene				1.0E+00	1.4E-5		1.0E-5						1.0E-3	
2-Hexanone				1.0E+00	1.4E-5		6.5E-6						1.0E-3	
Methyl ethyl ketone				1.0E+00	1.4E-5		6.9E-6						1.0E-3	
4-Methyl-2-Pentanone				1.0E+00	1.4E-5		6.8E-7						1.0E-3	
Methylene Chloride	7.50E-3	7.50E-3	1.60E-3	1.0E+00	1.4E-5	1.0E-7	6.3E-6	4.7E-8					1.0E-3	1.7E-6
M,P Xylene				1.0E+00	1.4E-5		1.1E-4						1.0E-3	
1,1,1-Trichloroethane				1.0E+00	1.4E-5		2.7E-5						1.0E-3	
Tetrachloroethene	5.20E-2	5.20E-2	2.00E-3	1.0E+00	1.4E-5	7.3E-7	7.7E-5	4.0E-6					1.0E-3	2.1E-6
Toluene				1.0E+00	1.4E-5		5.1E-5						1.0E-3	
Total Xylenes				1.0E+00	1.4E-5		8.6E-5						1.0E-3	
Trichloroethene	1.10E-2	1.10E-2	6.00E-3	1.0E+00	1.4E-5	1.5E-7	2.5E-5	2.7E-7					1.0E-3	6.3E-6
Vinyl chloride	1.90E+0	1.90E+0	3.00E-1	1.0E+00	1.4E-5	2.7E-5	9.9E-6	1.9E-5					1.0E-3	3.1E-4

Table C.4

Unit Risk and Unit Hazard Calculations
 RME Resident Child Noncancer Unit Risk from Exposure to Unit Concentrations of COPCs (mg/L) in Groundwater

Chemical name	PRGs Based on All Pathways (mg/L)		Chem Degradation		Depletion Coefficients		Soil Leaching		Physical Data		Basic Information				Bio-transfer Coefficients (a)	
	Lifetime HQ (unitless)	Pathways (mg/L)	Half-life (yr)	Decay (1/h)	Chem Degradation (1/h)	Soil Leaching (cm ² /yr)	Henry's Law Const. (atm-m ³ /mol)	Molec. Weight (g/M)	Permeab. Constant K _p (cm/h)	Lag Time TAO (unitless)	Time to Equilib. (h)	Partic. Coeff. B (unitless)	GI/Abs. Factor GI (unitless)	Soil-Forage BT Grass (unitless)		
VOLATILE ORGANIC CHEMICALS																
Acetone	6.4E-1	2 E-6		1.0E-3	2.7E-3	1.7E-3	2.1E-5	58.08	5.69E-4	2.0E-1	4.7E-1	5.8E-5	1.0E+0	2.27E+0		
Carbon tetrachloride	1.3E+2	7 E-9		1.0E-3	3.8E+0	5.1E-5	3.0E-2	154.00	2.20E-2	7.6E-1	1.1E+2	6.7E-2	1.0E+0	1.20E+0		
Chloroform	7.4E+0	1 E-7		1.6E-4	1.8E+0	1.0E-4	3.4E-3	119.38	8.92E-3	4.7E-1	1.1E+0	9.3E-3	1.0E+0	2.27E+0		
Cis-1,2-Dichloroethene	6.9E+0	1 E-7		4.0E-5	5.9E-1	2.8E-4	7.6E-3	96.94	5.30E-3	3.4E-1	8.4E-1	1.2E-1	1.0E+0	2.27E+0		
1,2-Dichloroethane				4.0E-5	2.4E+0	8.0E-5	9.8E-4	99.00	5.30E-3	3.5E-1	8.4E-1	1.2E-1	1.0E+0	2.40E+0		
1,1-Dichloroethene	8.8E+0	1 E-7		1.6E-4	5.8E-1	2.9E-4	3.4E-2	97.00	1.60E-2	3.4E-1	1.4E+0	3.1E-2	1.0E+0	2.27E+0		
1,2-Dichloroethene (Total)	7.6E+0	1 E-7		4.0E-5	5.9E-1	2.8E-4	7.6E-3	96.94	5.30E-3	3.4E-1	8.4E-1	1.2E-1	1.0E+0	2.27E+0		
1,2-Dichloroethene, Trans-	3.5E+0	3 E-7			5.9E-1	2.8E-4	6.6E-3	96.94	5.49E-3	3.4E-1	8.2E-1	3.0E-3	1.0E+0	2.27E+0		
Benzene	2.2E+2	5 E-9		4.0E-5	2.6E+0	7.3E-5	5.6E-3	78.11	2.10E-2	2.6E-1	6.3E-1	1.3E-2	1.0E+0	2.27E+0		
Ethylbenzene	2.5E+0	4 E-7		1.3E-4	2.7E+1	7.3E-6	8.5E-3	106.17	7.40E-2	3.9E-1	1.3E+0	1.4E-1	1.0E+0	5.88E-1		
2-Hexanone					1.2E-1	8.5E-4		100.16	4.45E-3	3.6E-1	8.6E-1	2.4E-3	1.0E+0	5.88E-1		
Methyl ethyl ketone	1.2E+0	8 E-7		1.0E-4	1.1E-2	1.6E-3	2.7E-5	72.00	5.00E-3	2.4E-1	5.8E-1	1.9E-4	1.0E+0	2.27E+0		
4-Methyl-2-Pentanone				1.0E-3	3.8E-1	4.0E-4		100.16	4.67E-4	3.6E-1	8.6E-1	1.0E-4	1.0E+0	5.88E-1		
Methylene Chloride	1.5E+0	7 E-7		1.0E-3	8.5E-2	1.0E-3	3.2E-3	84.93	4.46E-3	2.9E-1	6.9E-1	1.8E-3	1.0E+0	5.88E-1		
M,P Xylene	7.2E-2	1 E-5		7.9E-5	2.4E+0	7.9E-5	7.2E-3	106.16	8.00E-2	3.9E-1	1.4E+0	1.6E-1	1.0E+0	6.77E-1		
1,1,1-Trichloroethane	3.5E+0	3 E-7		1.3E-5	5.7E+0	2.6E-5	1.7E-1	133.00	1.70E-2	5.7E-1	1.4E+0	3.1E-2	1.0E+0	1.40E+0		
Tetrachloroethene	1.4E+1	7 E-8		1.8E-5	6.5E+0	3.0E-5	1.8E-2	165.83	4.80E-2	9.0E-1	4.3E+0	2.5E-1	1.0E+0	4.20E-1		
Toluene	3.3E+0	3 E-7		1.4E-4	9.4E+0	2.1E-5	6.6E-3	92.14	4.50E-2	3.2E-1	7.7E-1	5.4E-2	1.0E+0	1.02E+0		
Total Xylenes	6.3E-2	2 E-5		7.9E-5	2.1E+1	9.4E-6	7.0E-3	106.16	6.18E-2	3.9E-1	1.0E+0	1.1E-1	1.0E+0	6.77E-1		
Trichloroethene				1.8E-5	6.5E+0	3.0E-5	1.0E-2	131.39	1.60E-2	5.5E-1	1.3E+0	2.5E-2	1.0E+0	1.59E+0		
Vinyl chloride				1.0E-5	7.6E+0	2.6E-5	1.1E-2	62.50	7.30E-3	2.1E-1	5.5E-1	2.3E-3	1.0E+0	6.34E+0		

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Table C.4 (continued)
 Unit Risk and Unit Hazard Calculations
 RME Resident Child Noncancer Unit Risk from Exposure to Unit Concentrations of COPCs (mg/L) in Groundwater

Chemical name	Basic Information			Direct Exposure Pathways						
	Reference Doses (kg-d/mg)		Water Conc. (mg/L)	Ingestion		Dermal Absorption		Household Water Use		Volatiles Inhalation
	Oral RFD	Dermal RFD		Amount Ingested (mg/kg-d)	Lifetime HQ (unitless)	Amount Absorbed (mg/kg-d)	Lifetime HQ (unitless)	Amount Inhaled (mg/kg-d)	Lifetime HQ (unitless)	
VOLATILE ORGANIC CHEMICALS										
Acetone	1.0E-1	1.00E-1	1.0E+00	6.4E-2	6.4E-1	4.0E-4	4.0E-3	3.2E-1		
Carbon tetrachloride	7.0E-4	7.00E-4	1.0E+00	6.4E-2	9.1E+1	3.0E-2	4.3E+1	3.2E-1		
Chloroform	1.0E-2	1.00E-2	1.0E+00	6.4E-2	6.4E+0	9.7E-3	9.7E-1	3.2E-1		
Cis-1,2-Dichloroethene	1.0E-2	1.00E-2	1.0E+00	6.4E-2	6.4E+0	4.9E-3	4.9E-1	3.2E-1		
1,2-Dichloroethane			1.0E+00	6.4E-2		5.0E-3		3.2E-1		
1,1-Dichloroethene	9.0E-3	9.00E-3	1.0E+00	6.4E-2	7.1E+0	1.5E-2	1.6E+0	3.2E-1		
1,2-Dichloroethene (Total)	9.0E-3	9.00E-3	1.0E+00	6.4E-2	7.1E+0	4.9E-3	5.5E-1	3.2E-1		
1,2-Dichloroethene, Trans-	2.0E-2	2.00E-2	1.0E+00	6.4E-2	3.2E+0	5.1E-3	2.5E-1	3.2E-1		
Benzene	3.0E-3	3.00E-3	1.7E-3	6.4E-2	2.1E+1	1.7E-2	5.7E+0	3.2E-1	1.9E+2	
Ethylbenzene	1.0E-1	1.00E-1	2.9E-1	6.4E-2	6.4E-1	7.3E-2	7.3E-1	3.2E-1	1.1E+0	
2-Hexanone			1.0E+00	6.4E-2		4.2E-3				
Methyl ethyl ketone	6.0E-1	6.00E-1	2.9E-1	6.4E-2	1.1E-1	3.9E-3	6.5E-3	3.2E-1	1.1E+0	
4-Methyl-2-Pentanone			1.0E+00	6.4E-2		4.4E-4				
Methylene Chloride	6.0E-2	6.00E-2	8.6E-1	6.4E-2	1.1E+0	3.8E-3	6.4E-2	3.2E-1	3.7E-1	
M,P Xylene	2.0E+0	2.00E+0	1.0E+00	6.4E-2	3.2E-2	7.9E-2	4.0E-2	3.2E-1		
1,1,1-Trichloroethane	3.5E-2	3.50E-2	2.9E-1	6.4E-2	1.8E+0	2.0E-2	5.8E-1	3.2E-1	1.1E+0	
Tetrachloroethene	1.0E-2	1.00E-2	1.0E+00	6.4E-2	6.4E+0	7.2E-2	7.2E+0	3.2E-1		
Toluene	2.0E-1	2.00E-1	1.1E-1	6.4E-2	3.2E-1	4.0E-2	2.0E-1	3.2E-1	2.8E+0	
Total Xylenes	2.0E+0	2.00E+0	1.0E+00	6.4E-2	3.2E-2	6.1E-2	3.1E-2	3.2E-1		
Trichloroethene			1.0E+00	6.4E-2		1.9E-2		3.2E-1		
Vinyl chloride			1.0E+00	6.4E-2		5.3E-3		3.2E-1		

Appendix C-2

Cancer Risk and Noncancer Hazard Risk Calculation Spreadsheets

**Table C.5
Cancer Risk and Noncancer Hazard Risk Calculation
RME Trespasser Cancer Risk from COPCs in Creek Surface Water**

Chemical name	Dermal Uptake Data		Bio-transfer Coefficients (a)		Cancer Potency Factors		Trespasser Surface Water Exposure			
	Permeability Coefficient (cm/h)	Lag Time (h)	Particulate Coeff. (unitless)	GI/Abs. Factor (unitless)	Oral Slope Factor (mg/kg-d)	Inhalation Slope Factor	Water Conc. (mg/L)	Ingestion Amount (mg/kg-d)	Dermal/Absorption Amount Absorbed (mg/kg-d)	Lifetime Risks (ILCR)
INORGANIC CHEMICALS										
Aluminum	1.00E-3			1.0E+0			3.2E+01	8.1E-6	1.6E-6	
Antimony	1.00E-3			1.5E-1			7.1E-03	1.8E-9	3.5E-10	
Cobalt	1.00E-3			5.0E-2			2.0E-02	5.0E-9	9.9E-10	
Copper	1.00E-3			1.0E+0			3.2E-02	8.1E-9	1.6E-9	
Iron	1.00E-3			1.0E+0			4.6E+01	1.2E-5	2.3E-6	
Lead	1.00E-3			1.0E+0			6.3E-02	1.6E-8	3.1E-9	
Magnesium	1.00E-3			1.0E+0			2.1E+01	5.3E-6	1.0E-6	
Manganese	1.00E-3			6.0E-2			3.9E+00	9.8E-7	1.9E-7	
Vanadium	1.00E-3			3.0E-2			5.8E-02	1.5E-8	2.9E-9	
Zinc	1.00E-3			1.0E+0			4.2E-01	1.1E-7	2.1E-8	
VOLATILE ORGANIC CHEMICALS										
Cis-1,2-Dichloroethene	5.30E-3	3.4E-1	1.2E-1	1.0E+0			6.4E-02	1.6E-8	2.9E-8	
Trichloroethene	1.60E-2	5.5E-1	2.5E-2	1.0E+0	1.10E-2	6.00E-3	2.0E-01	5.0E-8	3.2E-7	3.6E-9
Vinyl chloride	7.30E-3	2.1E-1	2.3E-3	1.0E+0	1.40E+0	3.10E-2	7.3E-03	1.8E-9	3.7E-9	5.2E-9
SEMIVOLATILE ORGANIC CHEMICALS										
Bis(2-ethylhexyl)phthalate	3.30E-2	2.1E+1	2.0E+1	1.0E+0	1.40E-2	1.40E-2	1.2E-02	3.0E-9	2.5E-7	3.5E-9
TOTALS										
Parameter										
Averaging time										
Body weight										
Exposure duration (recreational)										
Exposure fraction (recreational)										
Exposure time (recreational)										
Ingestion rate										
Skin surface area exposed										

Value Units
 25550 d/life
 56 kg
 6 yrs
 12 event/yr
 1 h/event
 0.005 L/d
 980 cm²

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Table C.6
 Cancer Risk and Noncancer Hazard Risk Calculation
 RME Trespasser Noncancer Risk from Exposure to COPCs in Creek Surface Water

Chemical name	Dermal Uptake Data		Bio-transfer Coefficients (g)		Reference Doses		Water		Trespasser Surface Water Exposure				
	Permeability Constant (cm/h)	Lag Time (h)	Time to Equilib. (h)	Partic. Coeff. (unitless)	GI Abs. Factor (unitless)	Oral RFD (kg-d/mg)	Dermal RFD (kg-d/mg)	Inhalation RFD (unitless)	Water Conc. (mg/L)	Amount Ingested (mg/kg-d)	Ingestion (L/lifetime)	Amount Absorbed (mg/kg-d)	Lifetime HQ
INORGANIC CHEMICALS													
Aluminum	1.00E-3				1.0E+0	1.0E+0	1.00E+0	1.4E-3	3.2E+01	#VALUE!		#DIV/0!	
Antimony	1.00E-3				1.5E-1	4.0E-4	6.00E-5		7.1E-03	#VALUE!		#DIV/0!	
Cobalt	1.00E-3				5.0E-2	3.7E-2	3.70E-2		2.0E-02	#VALUE!		#DIV/0!	
Copper	1.00E-3				1.0E+0	3.0E-1	3.00E-1		3.2E-02	#VALUE!		#DIV/0!	
Iron	1.00E-3				1.0E+0	3.0E-1	3.00E-1		4.6E+01	#VALUE!		#DIV/0!	
Lead	1.00E-3				1.0E+0	3.0E-1	3.00E-1		6.3E-02	#VALUE!		#DIV/0!	
Magnesium	1.00E-3				1.0E+0	3.0E-1	3.00E-1		2.1E+01	#VALUE!		#DIV/0!	
Manganese	1.00E-3				6.0E-2	4.7E-2	2.82E-3	1.4E-5	3.9E+00	#VALUE!		#DIV/0!	
Vanadium	1.00E-3				1.0E-2	7.0E-3	7.00E-5		5.8E-02	#VALUE!		#DIV/0!	
Zinc	1.00E-3				1.0E+0	3.0E-1	3.00E-1		4.2E-01	#VALUE!		#DIV/0!	
VOLATILE ORGANIC CHEMICALS													
Cis-1,2-Dichloroethene	5.30E-3	3.4E-1	8.4E-1	1.2E-1	1.0E+0	1.0E-2	1.00E-2		6.4E-02	#VALUE!		#DIV/0!	
Trichloroethene	1.60E-2	5.5E-1	1.3E+0	2.5E-2	1.0E+0	3.0E-3	3.00E-3	2.9E-2	2.0E-01	#VALUE!		#DIV/0!	
Vinyl chloride	7.30E-3	2.1E-1	5.5E-1	2.3E-3	1.0E+0	3.0E-3	3.00E-3		7.3E-03	#VALUE!		#DIV/0!	
SEMIVOLATILE ORGANIC CHEMICALS													
Bis(2-ethylhexyl)phthalate	3.30E-2	2.1E+1	1.0E+1	2.0E+1	1.0E+0	2.0E-2	2.00E-2		1.2E-02	#VALUE!		#DIV/0!	
TOTALS													

Parameter Value Units
 Averaging time 2190 d/life
 Body weight 56 Kg
 Exposure duration (recreational) 6 yrs
 Exposure fraction (recreational) 12 event/yr
 Exposure time (recreational) 1 h/event
 Ingestion rate - Human-water (while swimming) 0.005 L/d
 Skin surface area exposed 980 cm²

Table C.7
Cancer Risk and Noncancer Hazard Risk Calculation
RME Trespasser Noncancer Risk from Exposure to COPCs In Creek Sediment

Chemical Name	Concentration in sediment (CS), mg/kg		Reference Doses, mg/kg-d		Absorption factor, unitless		Dermal Absorption		Ingestion		Sum HI (unitless)
	Oral (RFDO)	Dermal (RFDD)	Oral (ABO)	Inhalation (RFDI)	Amount Absorbed (IDD), mg/kg-d	Lifetime HQ (HQB), unitless	Amount Ingested (IDIG), mg/kg-d	Lifetime HQ (HQIG), unitless			
INORGANIC CHEMICALS											
Antimony	4 0E-4	6 0E-05	0.15		0.01	1 9E-04	1 9E-08	1 9E-04	4 8E-05	2 3E-04	
Arsenic	3 0E-4	3 0E-04	1		0.03	2 8E-04	8 3E-08	2 8E-04	1 6E-04	4 3E-04	
Barium	7 0E-2	4 9E-03	0.07	1 4E-4	0.01	2 8E-04	1 4E-06	2 8E-04	3 3E-05	3 1E-04	
Iron	3 0E-1	3 0E-01	1		0.01	8 1E-05	2 4E-05	8 1E-05	1 4E-04	2 2E-04	
Magnesium	2 5E+03		1		0.01		4 3E-06				
Manganese	1 6E+03	2 8E-03	0.06	1 4E-5	0.01	9 8E-04	2 8E-06	9 8E-04	1 0E-04	1 1E-03	
Nickel	1 3E+01	8 0E-04	0.04		0.01	2 8E-05	2 2E-08	2 8E-05	1 9E-06	3 0E-05	
Vanadium	4 9E+01	2 1E-04	0.03		0.01	4 0E-04	8 5E-08	4 0E-04	2 1E-05	4 2E-04	
Zinc	1 6E+02	3 0E-01	1		0.01	9 2E-07	2 8E-07	9 2E-07	1 6E-06	2 5E-06	
VOLATILE ORGANIC CHEMICALS											
No COPCs											
SEMI-VOLATILE ORGANIC CHEMICALS											
Benzo(a)anthracene	2 5E-01		1		0.13	5 6E-09			7 3E-10		
Benzo(a)pyrene	4 2E-01		1		0.13	9 4E-09			1 2E-09		
Benzo(b)fluoranthene	3 5E-01		1		0.13	7 9E-09			1 0E-09		
Benzo(g,h,i)perylene	3 6E-01		1		0.13	8 1E-09			1 1E-09		
Chrysene	4 2E-01		1		0.13	9 4E-09			1 2E-09		
Indeno(1,2,3-cd)pyrene	3 0E-01		1		0.13	6 7E-09			8 8E-10		
Total Noncancer Hazard						2 2E-03		2 2E-03	5 0E-04	2 7E-03	

ASSUMPTIONS

Averaging Time, AT (yr)	6
Adherence Factor, AF (mg/cm ²)	0.3
Body Weight, BW (kg)	56
Contaminant Fraction, CF (unitless)	1
Conversion Factors	
Mass, MCF (kg/mg)	1 00E-06
Time, TCF (d/yr)	365
Exposure Duration	6
Dermal, EDD (yr)	NA
Inhalation from soil, EDIS (yr)	NA
Inhalation from volatiles, EDIV (yr)	NA
Ingestion, EDIG (yr)	6
Exposure Frequency, EF (d/yr)	12
Fraction Ingested, FIIG (unitless)	1
Fraction Inhaled, FIIV (unitless)	NA
Inhalation Rate, IR (m ³ /d)	NA
Ingestion Rate, IGR (mg/d)	5
Particulate Emission Factor, PEF (m ² /kg)	NA
Respirable Fraction, RF (unitless)	NA
Skin surface area, SA (cm ² /rd)	980

EQUATIONS

IDD =	$CS \cdot MCF \cdot SA \cdot AF \cdot ABS \cdot EF \cdot EDD$
	$BW \cdot AT \cdot TCF$
HQD =	$IDD / RFDD$
IDIS =	$CS \cdot IR \cdot RF \cdot CF \cdot EF \cdot EDIS$
	$BW \cdot AT \cdot TCF \cdot PEF$
HQIS =	$IDIS / RFDI$
IDIV =	$CS \cdot (1/MF) \cdot IR \cdot FIIV \cdot EF \cdot EDIV$
	$BW \cdot AT \cdot TCF$
HQIV =	$IDIV / RFDI$
IDIG =	$CS \cdot IGR \cdot FIIG \cdot MCF \cdot EF \cdot EDIG$
	$BW \cdot AT \cdot TCF$
HQIG =	$IDIG / RFDO$

Table C.8
Cancer Risk and Noncancer Hazard Risk Calculation
RME Trespasser Cancer Risk from Exposure to COPCs in Creek Sediment

Chemical Name	Concentration in sediment (CS), mg/kg	Cancer Potency Factors, 1/(mg/kg-d)			Absorption		Dermal Absorption		Ingestion		Sum
		Oral Slope Factor (SFO)	Dermal Slope Factor (SFD)	Inhalation Slope Factor (SFI)	Oral (ABO) factor, unitless	Drm (ABS)	Amount Absorbed (IDD), mg/kg-d	Lifetime Risks (CRD) (unitless)	Amount Ingested (IDIG) (mg/kg-d)	Lifetime Risks (CRIG) (unitless)	
INORGANIC CHEMICALS											
Antimony	6.5E+00	1.5E+00	1.5E+00	1.5E+01	0.15	0.01	9.6E-10	1.1E-08	1.6E-09	6.0E-09	1.7E-08
Arsenic	1.6E+01				1	0.03	7.1E-09		4.0E-09		
Barium	7.9E+02				0.07	0.01	1.2E-07		2.0E-07		
Iron	1.4E+04				1	0.01	2.1E-06		3.5E-06		
Magnesium	2.5E+03				1	0.01	3.7E-07		6.3E-07		
Manganese	1.6E+03				0.06	0.01	2.4E-07		4.0E-07		
Nickel	1.3E+01				0.04	0.01	1.9E-09		3.3E-09		
Vanadium	4.9E+01				0.03	0.01	7.2E-09		1.2E-08		
Zinc	1.6E+02				1	0.01					
VOLATILE ORGANIC CHEMICALS											
No COPCs											
SEMIVOLATILE ORGANIC CHEMICALS											
Benzof(a)anthracene	2.5E-01	7.3E-01	7.3E-01	3.1E-01	1	0.13	4.8E-10	3.5E-10	6.3E-11	4.6E-11	4.0E-10
Benzof(a)pyrene	4.2E-01	7.3E+00	7.3E+00	3.1E+00	1	0.13	8.1E-10	5.9E-09	1.1E-10	7.7E-10	6.7E-09
Benzof(b)fluoranthene	3.5E-01	7.3E-01	7.3E-01	3.1E-01	1	0.13	6.7E-10	4.9E-10	8.8E-11	6.4E-11	5.6E-10
Benzof(ghi)perylene	3.6E-01	7.3E-03	7.3E-03	3.1E-03	1	0.13	6.9E-10	5.9E-12	9.1E-11	7.7E-13	6.7E-12
Chrysene	4.2E-01	7.3E-01	7.3E-01	3.1E-01	1	0.13	8.1E-10	4.2E-10	1.1E-10	5.5E-11	4.8E-10
Indeno(1,23-cd)pyrene	3.0E-01	7.3E-01	7.3E-01	3.1E-01	1	0.13	5.8E-10	1.8E-08	7.5E-11	7.0E-09	2.5E-08
Total Cancer Risk											

ASSUMPTIONS

- Averaging Time, AT (yr) 70
- Adherence Factor, AF (mg/cm²) 0.3
- Body Weight, BW (kg) 56
- Contaminant Fraction, CF (unitless) 1
- Conversion Factors 1.00E-06
- Mass, MCF (kg/mg) 365
- Time, TCF (d/yr)
- Exposure Duration 6
- Dermal, EDD (yr) NA
- Inhalation from soil, EDIS (yr) NA
- Inhalation from volatiles, EDIV (yr) NA
- Ingestion, EDIG (yr) 6
- Exposure Frequency, EF (d/yr) 12
- Fraction Ingested, FIIG (unitless) 1
- Fraction Inhaled, FIIV (unitless) NA
- Inhalation Rate, IR (m³/d) NA
- Ingestion Rate, IGR (mg/d) 5
- Particulate Emission Factor, PEF (m²/kg) NA
- Respirable Fraction, RF (unitless) NA
- Skin surface area, SA (cm²/d) 980

EQUATIONS

- IDD = $CS * MCF * SA * AF * ABS * EF * EDD$
- $BW * AT * TCF$
- CRD = $IDD * SFD$
- IDIS = $CS * IR * RF * CF * EF * EDIS$
- $BW * AT * TCF * PEF$
- CR1 = $ID1 * SFI$
- IDIV = $CS * (1/AF) * IR * FIV * EF * EDIV$
- $BW * AT * TCF$
- CRIV = $IDIV * SFI$
- IDIG = $CS * IGR * FIIG * MCF * EF * EDIG$
- $BW * AT * TCF$

Table C.9
Cancer Risk and Noncancer Hazard Risk Calculation
RME Maintenance Worker Cancer Risk from COPCs in Creek Surface Water

Chemical name	Lifetime Risks (ILCR)	Dermal Uptake Data		Bio-transfer Coefficients (g)		Cancer Potency Factors		Ingestion		Dermal/Absorption	
		Penetration Constant K_p (cm/h)	Lag Time TAO (unitless)	Time to Equilib. (h)	Partic. Coeff. B (unitless)	GI Abs. Factor (unitless)	Oral Slope Factor (mg/kg-d)	Inhala. Slope Factor	Amount Ingested (mg/kg-d)	Lifetime Risks (ILCR)	Amount Absorbed (mg/kg-d)
INORGANIC CHEMICALS											
Aluminum		1.00E-3				1.0E+0			2.6E-5	5.8E-6	
Antimony		1.00E-3				1.5E-1			5.7E-9	1.3E-9	
Cobalt		1.00E-3				5.0E-2			1.6E-8	3.6E-9	
Copper		1.00E-3				1.0E+0			2.6E-8	5.8E-9	
Iron		1.00E-3				1.0E+0			3.7E-5	8.3E-6	
Lead		1.00E-3				1.0E+0			5.1E-8	1.1E-8	
Magnesium		1.00E-3				1.0E+0			1.7E-5	3.8E-6	
Manganese		1.00E-3				6.0E-2			3.1E-6	7.0E-7	
Vanadium		1.00E-3				3.0E-2			4.7E-8	1.0E-8	
Zinc		1.00E-3				1.0E+0			3.4E-7	7.6E-8	
VOLATILE ORGANIC CHEMICALS											
Cis-1,2-Dichloroethene		5.30E-3	3.4E-1	8.2E-1	1.2E-1	1.0E+0			5.2E-8	1.1E-7	
Trichloroethene	1.5E-8	1.60E-2	5.5E-1	1.3E+0	2.5E-2	1.0E+0	1.10E-2	6.00E-3	1.6E-7	1.2E-6	1.3E-8
Vinyl chloride	2.7E-8	7.30E-3	2.1E-1	5.1E-1	2.3E-3	1.0E+0	1.40E+0	3.10E-2	5.9E-9	1.4E-8	1.9E-8
SEMIVOLATILE ORGANIC CHEMICALS											
Bis(2-ethylhexyl)phthalate	1.3E-8	3.30E-2	2.1E+1	1.0E+1	2.0E+1	1.0E+0	1.40E-2	1.40E-2	9.7E-9	9.1E-7	1.3E-8
TOTALS	5.5E-8								1.0E-8		4.5E-8

Parameter
 Averaging time 25550 d/life
 Body weight 70 kg
 Exposure duration (recreational) 24 yrs
 Exposure fraction (recreational) 12 event/yr
 Exposure time (recreational) 1 h/event
 Ingestion rate Human-water (while swimming) 0.005 L/d
 Skin surface area exposed 1120 cm²

**Table C.10
Cancer Risk and Noncancer Hazard Risk Calculation
RME Maintenance Worker Noncancer Risk from Exposure to COPCs in Creek Surface Water**

Chemical name	Lifetime HQ (unitless)	Dermal Uptake Data		Bio-transfer Coefficients (a)		Reference Doses		Trespasser Surface Water Exposure				
		Permeability Constant (cm/h)	Lag Time (h)	Particle Coefficient (unitless)	GI Absorption Factor (unitless)	Oral RFD (kg-d/mg)	Dermal RFD (kg-d/mg)	Inhalation RFD (unitless)	Water Conc (mg/L)	Ingestion Amount (mg/kg-d)	Dermal Absorbed Amount (mg/kg-d)	Lifetime HQ (unitless)
INORGANIC CHEMICALS												
Aluminum	9.2E-5	1.00E-3		1.0E+0	1.0E+0	1.00E+0	1.4E-3	3.2E+01	7.5E-5	1.7E-5	7.5E-5	1.7E-5
Antimony	1.0E-4	1.00E-3		1.5E-1	5.0E-2	6.00E-5		7.1E-03	1.7E-8	3.7E-9	4.2E-5	6.2E-5
Cobalt	2.5E-6	1.00E-3		1.0E+0	1.0E+0	3.70E-2		2.0E-02	4.7E-8	1.1E-8	2.0E-6	4.5E-7
Copper	4.4E-4	1.00E-3		1.0E+0	1.0E+0	3.00E-1		3.2E-02	7.5E-8	1.7E-8	3.6E-4	8.1E-5
Iron		1.00E-3		1.0E+0	1.0E+0			4.6E+01	1.1E-4	2.4E-5		
Lead		1.00E-3		1.0E+0	1.0E+0			6.3E-02	1.5E-7	3.3E-8		
Magnesium		1.00E-3		1.0E+0	1.0E+0			2.1E+01	4.9E-5	1.1E-5		
Manganese	9.2E-4	1.00E-3		6.0E-2	6.0E-2	2.82E-3	1.4E-5	3.9E+00	9.2E-6	2.1E-6	1.9E-4	7.3E-4
Vanadium	4.6E-4	1.00E-3		1.0E-2	1.0E-2	7.00E-5		5.8E-02	1.4E-7	3.1E-8	1.9E-5	4.4E-4
Zinc	4.0E-6	1.00E-3		1.0E+0	1.0E+0	3.00E-1		4.2E-01	9.9E-7	2.2E-7	3.3E-6	7.4E-7
VOLATILE ORGANIC CHEMICALS												
Cis-1,2-Dichloroethene	4.6E-5	5.30E-3	3.4E-1	1.2E-1	1.0E+0	1.00E-2		6.4E-02	1.5E-7	3.1E-7	1.5E-5	3.1E-5
Trichloroethene		1.60E-2	5.5E-1	2.5E-2	1.0E+0			2.0E-01	4.7E-7	3.5E-6	5.7E-6	1.3E-5
Vinyl chloride	1.9E-5	7.30E-3	2.1E-1	2.3E-3	1.0E+0	3.00E-3	2.9E-2	7.3E-03	1.7E-8	4.0E-8		
SEMI-VOLATILE ORGANIC CHEMICALS												
Bis(2-ethylhexyl)phthalate	1.3E-4	3.30E-2	2.1E+1	2.0E+1	1.0E+0	2.00E-2		1.2E-02	2.8E-8	2.6E-6	1.4E-6	1.3E-4
TOTALS	2.2E-3										7.2E-4	1.5E-3

Value Units
 Averaging time: 8760 d/life
 Body weight: 70 Kg
 Exposure duration (recreational): 24 yrs
 Exposure fraction (recreational): 12 event/yr
 Exposure time (recreational): 1 h/event
 Ingestion rate: Human-water (while swimming): 0.005 L/d
 Skin surface area exposed: 1120 cm²

Table C.11
Cancer Risk and Noncancer Hazard Risk Calculation
RME Maintenance Worker Cancer Risk from Exposure to COPCs in Creek Sediment

Chemical Name	Concentration in sediment (CS), mg/kg	Cancer Potency Factors, 1/(mg/kg-d)			Absorption factor, unitless		Dermal Absorption		Ingestion		Sum ILCR (unitless)
		Oral (SFO) Factor	Dermal Slope Factor (SFD)	Inhalation Slope Factor (SFI)	Oral (ABO) Factor	Dermal (ABS)	Amount Absorbed (IDD), mg/kg-d	Lifetime Risks (CRD) (unitless)	Amount Ingested (IDIG), mg/kg-d	Lifetime Risks (CRIG) (unitless)	
INORGANIC CHEMICALS											
Antimony	6 5E+00	1 5E+00	1 5E+00	1 5E+01	0.15	0.01	9.4E-10	1.0E-08	5.2E-09	1.9E-08	3.0E-08
Arsenic	1 6E+01				1	0.03	6.9E-09		1.3E-08		
Barium	7 9E+02				0.07	0.01	1.1E-07		6.4E-07		
Iron	1 4E+04				1	0.01	2.0E-06		1.1E-05		
Magnesium	2 5E+03				1	0.01	3.6E-07		2.0E-06		
Manganese	1 6E+03				0.06	0.01	2.3E-07		1.3E-06		
Nickel	1 3E+01				0.04	0.01	1.9E-09		1.0E-08		
Vanadium	4 9E+01				0.03	0.01	7.1E-09		3.9E-08		
Zinc	1 6E+02				1	0.01					
VOLATILE ORGANIC CHEMICALS											
No COPCs											
SEMI-VOLATILE ORGANIC CHEMICALS											
Benz(a)anthracene	2 5E-01	7 3E-01	7 3E-01	3 1E-01	1	0.13	4.7E-10	3.4E-10	2.0E-10	1.5E-10	4.9E-10
Benz(a)pyrene	4 2E-01	7 3E+00	7 3E+00	3 1E+00	1	0.13	7.9E-10	5.8E-09	3.4E-10	2.5E-09	8.2E-09
Benz(b)fluoranthene	3 5E-01	7 3E-01	7 3E-01	3 1E-01	1	0.13	6.6E-10	4.8E-10	2.8E-10	2.1E-10	6.8E-10
Benz(ghi)perylene	3 6E-01				1	0.13	6.8E-10		2.9E-10		
Chrysene	4 2E-01	7 3E-03	7 3E-03	3 1E-03	1	0.13	7.9E-10	5.8E-12	3.4E-10	2.5E-12	8.2E-12
Indeno(1,23-cd)pyrene	3 0E-01	7 3E-01	7 3E-01	3 1E-01	1	0.13	5.6E-10	4.1E-10	2.4E-10	1.8E-10	5.9E-10
Total Cancer Risk								1.7E-08		2.2E-08	4.0E-08

EQUATIONS

$$\begin{aligned}
 \text{IDD} &= \frac{\text{CS} \cdot \text{MCF} \cdot \text{SA} \cdot \text{AF} \cdot \text{ABS} \cdot \text{EF} \cdot \text{EDD}}{\text{BW} \cdot \text{AT} \cdot \text{TCF}} \\
 \text{CRD} &= \text{IDD} \cdot \text{SFD} \\
 \text{IDIS} &= \frac{\text{CS} \cdot \text{IR} \cdot \text{RF} \cdot \text{CF} \cdot \text{EF} \cdot \text{EDIS}}{\text{BW} \cdot \text{AT} \cdot \text{TCF} \cdot \text{PEF}} \\
 \text{CRI} &= \text{IDI} \cdot \text{SFI} \\
 \text{IDIV} &= \frac{\text{CS} \cdot (\text{IAVF}) \cdot \text{IR} \cdot \text{FIIV} \cdot \text{EF} \cdot \text{EDIV}}{\text{BW} \cdot \text{AT} \cdot \text{TCF}} \\
 \text{CRIV} &= \text{IDIV} \cdot \text{SFI} \\
 \text{IDIG} &= \frac{\text{CS} \cdot \text{IGR} \cdot \text{FIIG} \cdot \text{MCF} \cdot \text{EF} \cdot \text{EDIG}}{\text{BW} \cdot \text{AT} \cdot \text{TCF}} \\
 \text{CRIG} &= \text{IDIG} \cdot \text{SFO}
 \end{aligned}$$

ASSUMPTIONS

- Averaging Time, AT (yr) 70
- Adherence Factor, AF (mg/cm²) 0.08
- Body Weight, BW (kg) 70
- Contaminant Fraction, CF (unitless) 1
- Conversion Factors
 - Mass, MCF (kg/mg) 1.00E-06
 - Time, TCF (d/yr) 365
- Exposure Duration
 - Dermal, EDD (yr) 24
 - Inhalation from soil, EDIS (yr) NA
 - Inhalation from volatiles, EDIV (yr) NA
 - Ingestion, EDIG (yr) 24
 - Exposure Frequency, EF (d/yr) 12
 - Fraction Ingested, FIIG (unitless) 1
 - Fraction Inhaled, FIIV (unitless) NA
 - Inhalation Rate, IR (m³/d) NA
 - Ingestion Rate, IGR (mg/d) 5
 - Particulate Emission Factor, PEF (m³/kg) NA
 - Respirable Fraction, RF (unitless) NA
 - Skin surface area, SA (cm²/d) 1,120

Table C.12
Cancer Risk and Non Cancer Hazard Risk Calculation
RME Maintenance Worker Noncancer Risk from Exposure to COPCs In Creek Sediment

Chemical Name	Concentration in sediment (CS), mg/kg	Reference Doses, mg/kg-d		Absorption factor, unitless		Dermal Absorption		Ingestion		Sum HI (unitless)	
		Oral (RFO), mg/kg-d	Dermal (RFDD), mg/kg-d	Inhalation (RFDI), mg/kg-d	Oral (ABO), unitless	Drm (ABS), unitless	Amount Absorbed (IDD), mg/kg-d	Lifetime HQ (HQD), unitless	Amount Ingested (IDIG), mg/kg-d		Lifetime HQ (HIQIG), unitless
INORGANIC CHEMICALS											
Antimony	6.5E+00	4.0E-4	6.0E-05		0.15	0.01	2.7E-09	4.6E-05	1.5E-08	3.8E-05	8.4E-05
Arsenic	1.6E+01	3.0E-4	3.0E-04		1	0.03	2.0E-08	6.7E-05	3.8E-08	1.3E-04	1.9E-04
Barium	7.9E+02	7.0E-2	4.9E-03	1.4E-4	0.07	0.01	3.3E-07	6.8E-05	1.9E-06	2.7E-05	9.4E-05
Iron	1.4E+04	3.0E-1	3.0E-01		1	0.01	5.9E-06	2.0E-05	3.3E-05	1.1E-04	1.3E-04
Magnesium	2.5E+03				1	0.01	1.1E-06		5.9E-06		
Manganese	1.6E+03	4.7E-2	2.8E-03	1.4E-5	0.06	0.01	6.7E-07	2.4E-04	3.8E-06	8.0E-05	3.2E-04
Nickel	1.3E+01	2.0E-2	8.0E-04		0.04	0.01	5.5E-09	6.8E-06	3.1E-08	1.5E-06	8.4E-06
Vanadium	4.9E+01	7.0E-3	2.1E-04		0.03	0.01	2.1E-08	9.8E-05	1.2E-07	1.6E-05	1.1E-04
Zinc	1.6E+02	3.0E-1	3.0E-01		1	0.01	6.7E-08	2.2E-07	3.8E-07	1.3E-06	1.5E-06
VOLATILE ORGANIC CHEMICALS											
No COPCs											
SEMIVOLATILE ORGANIC CHEMICALS											
Benzo(a)anthracene	2.5E-01				1	0.13	1.4E-09		5.9E-10		
Benzo(a)pyrene	4.2E-01				1	0.13	2.3E-09		9.9E-10		
Benzo(b)fluoranthene	3.5E-01				1	0.13	1.9E-09		8.2E-10		
Benzo(g,h,i)perylene	3.6E-01				1	0.13	2.0E-09		8.5E-10		
Chrysene	4.2E-01				1	0.13	2.3E-09		9.9E-10		
Indeno(1,2,3-cd)pyrene	3.0E-01				1	0.13	1.6E-09		7.0E-10		
Total Noncancer Hazard											
								5.4E-04		4.0E-04	9.4E-04

ASSUMPTIONS

Averaging Time, AT (yr) 24
 Adherence Factor, AF (mg/cm²) 0.08
 Body Weight, BW (kg) 70
 Contaminant Fraction, CF (unitless) 1
 Conversion Factors
 Mass, MCF (kg/mg) 1.00E-06
 Time, TCF (d/yr) 365

Exposure Duration
 Dermal, EDD (yr) 24
 Inhalation from soil, EDIS (yr) NA
 Inhalation from volatiles, EDIV (yr) NA
 Ingestion, EDIG (yr) 24
 Exposure Frequency, EF (d/yr) 12
 Fraction Ingested, FIG (unitless) 1
 Fraction Inhaled, FIIV (unitless) NA
 Inhalation Rate, IR (m³/d) NA
 Ingestion Rate, IGR (mg/d) 5
 Particulate Emission Factor, PEF (m³/kg) NA
 Respirable Fraction, RF (unitless) NA
 Skin surface area, SA (cm²/d) 1,120

EQUATIONS

IDD = $\frac{CS * MCF * SA * AF * ABS * EF * EDD}{BW * AT * TCF}$
 HQD = $\frac{IDD}{RFDD}$
 IDIS = $\frac{CS * IR * RF * CF * EF * EDIS}{BW * AT * TCF * PEF}$
 HIQIS = $\frac{IDIS}{RFDI}$
 IDIV = $\frac{CS * (1/MF) * IR * FIIV * EF * EDIV}{BW * AT * TCF}$
 HIQIV = $\frac{IDIV}{RFDI}$
 IDIG = $\frac{CS * IGR * FIG * MCF * EF * EDIG}{BW * AT * TCF}$

Table C.13
Cancer Risk and Noncancer Hazard Risk Calculation
RME Recreational User Cancer Risk from COPCs in Creek Surface Water

Chemical name	Dermal Uptake Data		Bio-transfer Coefficients (a)		Cancer Potency Factors			Ingestion		Dermal/Absorption		
	Permeability Constant K_p (cm/h)	Lag Time TAO (h)	Time to Equilib. (h)	Partic. Coeff. B (unitless)	GI/Abs Factor (unitless)	Oral Slope Factor (mg/kg-d)	Inhalation Slope Factor	Water Conc. (mg/L)	Amount Ingested (mg/kg-d)	Lifetime Risks (ILCR)	Amount Absorbed (mg/kg-d)	Lifetime Risks (ILCR)
INORGANIC CHEMICALS												
Aluminum	1.00E-3				1.0E+0			3.2E+01	2.1E-5		4.8E-6	
Antimony	1.00E-3				1.5E-1			7.1E-03	4.8E-9		1.1E-9	
Cobalt	1.00E-3				5.0E-2			2.0E-02	1.3E-8		3.0E-9	
Copper	1.00E-3				1.0E+0			3.2E-02	2.1E-8		4.8E-9	
Iron	1.00E-3				1.0E+0			4.6E+01	3.1E-5		6.9E-6	
Lead	1.00E-3				1.0E+0			6.3E-02	4.2E-8		9.5E-9	
Magnesium	1.00E-3				1.0E+0			2.1E+01	1.4E-5		3.2E-6	
Manganese	1.00E-3				6.0E-2			3.9E+00	2.6E-6		5.9E-7	
Vanadium	1.00E-3				3.0E-2			5.8E-02	3.9E-8		8.7E-9	
Zinc	1.00E-3				1.0E+0			4.2E-01	2.8E-7		6.3E-8	
VOLATILE ORGANIC CHEMICALS												
Cis-1,2-Dichloroethene	5.30E-3	3.4E-1	8.2E-1	1.2E-1	1.0E+0	1.10E-2	1.10E-2	6.4E-02	4.3E-8	8.8E-8	8.8E-8	1.1E-8
Trichloroethene	1.60E-2	5.5E-1	1.3E+0	2.5E-2	1.0E+0	1.40E+0	1.40E+0	2.0E-01	1.3E-7	9.9E-7	1.5E-9	1.1E-8
Vinyl chloride	7.30E-3	2.1E-1	5.1E-1	2.3E-3	1.0E+0	1.40E+0	1.40E+0	7.3E-03	4.9E-9	1.1E-8	6.9E-9	1.6E-8
SEMIVOLATILE ORGANIC CHEMICALS												
Bis(2-ethylhexyl)phthalate	3.30E-2	2.1E+1	1.0E+1	2.0E+1	1.0E+0	1.40E-2	1.40E-2	1.2E-02	8.1E-9	7.6E-7	1.1E-10	1.1E-8
TOTALS	4.6E-8										8.4E-9	3.7E-8

Value Units

- Averaging time: 25550 d/life
- Body weight: 70 kg
- Exposure duration (recreational): 10 yrs
- Exposure fraction (recreational): 24 event/yr
- Exposure time (recreational): 1 h/event
- Ingestion rate: Human-water (while swimming): 0.005 L/d
- Skin surface area exposed: 1120 cm²

Table C.14
Cancer Risk and Noncancer Hazard Risk Calculation
RME Recreational User Cancer Risk from COPCs in Creek Sediment

Chemical Name	Cancer Potency Factors			Absorption		Dermal Absorption		Ingestion		Sum	
	Concentration (CS) In sediment (mg/kg)	Oral Slope Factor (SFO)	Dermal Slope Factor (SFD)	Inhalation Slope Factor (SFI)	Factor, unitless		Amount Absorbed (IDD), mg/kg-d	Lifetime Risks (CRD), (unitless)	Amount Ingested (IDIG), (mg/kg-d)		Lifetime Risks (CRIG), (unitless)
					Oral (ABO)	Drm (ABS)					
INORGANIC CHEMICALS											
Antimony	6.5E+00	1.5E+00	1.5E+00	1.5E+01	0.15	0.01	7.6E-10	8.7E-09	4.4E-09	1.6E-08	2.5E-08
Arsenic	1.6E+01	1.5E+00	1.5E+00	1.5E+01	1	0.03	5.9E-09	8.7E-09	1.1E-08	1.6E-08	2.5E-08
Barium	7.9E+02	1.5E+00	1.5E+00	1.5E+01	0.07	0.01	9.5E-08	8.7E-09	5.3E-07	1.6E-08	2.5E-08
Iron	1.4E+04	1.5E+00	1.5E+00	1.5E+01	1	0.01	1.7E-06	8.7E-09	9.4E-06	1.6E-08	2.5E-08
Magnesium	2.5E+03	1.5E+00	1.5E+00	1.5E+01	1	0.01	3.0E-07	8.7E-09	1.7E-06	1.6E-08	2.5E-08
Manganese	1.6E+03	1.5E+00	1.5E+00	1.5E+01	0.06	0.01	1.9E-07	8.7E-09	1.1E-06	1.6E-08	2.5E-08
Nickel	1.3E+01	1.5E+00	1.5E+00	1.5E+01	0.04	0.01	1.6E-09	8.7E-09	8.7E-09	1.6E-08	2.5E-08
Vanadium	4.9E+01	1.5E+00	1.5E+00	1.5E+01	0.03	0.01	5.9E-09	8.7E-09	3.3E-06	1.6E-08	2.5E-08
Zinc	1.6E+02	1.5E+00	1.5E+00	1.5E+01	1	0.01	7.6E-10	8.7E-09	4.4E-09	1.6E-08	2.5E-08
VOLATILE ORGANIC CHEMICALS											
No COPCs											
SEMI-VOLATILE ORGANIC CHEMICALS											
Benzo(a)anthracene	2.5E-01	7.3E-01	7.3E-01	3.1E-01	1	0.13	3.9E-10	2.9E-10	1.7E-10	1.2E-10	4.1E-10
Benzo(a)pyrene	4.2E-01	7.3E+00	7.3E+00	3.1E+00	1	0.13	6.6E-10	4.6E-09	2.8E-10	2.1E-09	6.8E-09
Benzo(b)fluoranthene	3.5E-01	7.3E-01	7.3E-01	3.1E-01	1	0.13	5.9E-10	4.0E-10	2.3E-10	1.7E-10	5.7E-10
Benzo(g)hperylene	3.6E-01	7.3E-01	7.3E-01	3.1E-01	1	0.13	5.6E-10	4.8E-12	2.4E-10	2.1E-12	6.8E-12
Chrysene	4.2E-01	7.3E-03	7.3E-03	3.1E-03	1	0.13	6.6E-10	3.4E-10	2.8E-10	1.5E-10	4.9E-10
Indeno(1,23-cd)pyrene	3.0E-01	7.3E-01	7.3E-01	3.1E-01	1	0.13	4.7E-10	1.4E-08	2.0E-10	1.9E-08	3.3E-08
Total Cancer Risk											

EQUATIONS

$$\begin{aligned}
 \text{IDD} &= \text{CS} \cdot \text{MCF} \cdot \text{SA} \cdot \text{AF} \cdot \text{ABS} \cdot \text{EF} \cdot \text{EDD} \\
 &= \text{BW} \cdot \text{AT} \cdot \text{TCF} \\
 \text{CRD} &= \text{IDD} \cdot \text{SFD} \\
 \text{IDIS} &= \frac{\text{CS} \cdot \text{IR} \cdot \text{RF} \cdot \text{CF} \cdot \text{EF} \cdot \text{EDIS}}{\text{BW} \cdot \text{AT} \cdot \text{TCF} \cdot \text{PEF}} \\
 \text{CRI} &= \text{IDI} \cdot \text{SFI} \\
 \text{IDIV} &= \frac{\text{CS} \cdot (\text{1/VF}) \cdot \text{IR} \cdot \text{FIIV} \cdot \text{EF} \cdot \text{EDIV}}{\text{BW} \cdot \text{AT} \cdot \text{TCF}} \\
 \text{CRIV} &= \text{IDIV} \cdot \text{SFI} \\
 \text{IDIG} &= \frac{\text{CS} \cdot \text{IGR} \cdot \text{FIIG} \cdot \text{MCF} \cdot \text{EF} \cdot \text{EDIG}}{\text{BW} \cdot \text{AT} \cdot \text{TCF}} \\
 \text{CRIG} &= \text{IDIG} \cdot \text{SFO}
 \end{aligned}$$

ASSUMPTIONS

- Averaging Time, AT (yr) 70
- Adherence Factor, AF (mg/cm²) 0.08
- Body Weight, BW (kg) 70
- Contaminant Fraction, CF (unitless) 1
- Conversion Factors
- Mass, MCF (kg/mg) 1.00E-06
- Time, TCF (d/yr) 365
- Exposure Duration 10
- Dermal, EDD (yr) NA
- Inhalation from soil, EDIS (yr) NA
- Inhalation from volatiles, EDIV (yr) NA
- Ingestion, EDIG (yr) 10
- Exposure Frequency, EF (d/yr) 24
- Fraction Ingested, FIIG (unitless) 1
- Fraction inhaled, FIIV (unitless) NA
- Inhalation Rate, IR (m³/d) NA
- Ingestion Rate, IGR (mg/d) 5
- Particulate Emission Factor, PEF (m³/kg) NA
- Respirable Fraction, RF (unitless) NA
- Skin surface area, SA (cm²/d) 1,120

Table C.15
Cancer Risk and Noncancer Hazard Risk Calculation
RME Recreational User Noncancer Unit Risk from Exposure to COPCs in Creek Surface Water

Chemical name	Lifetime HQ (unitless)	Dermal Uptake Data			Reference Doses			Water		Ingestion		Dermal Absorption	
		Permeability Constant K _p (cm/h)	Lag Time TAO (unitless)	Partic. Coeff. B (unitless)	GI Abs. Factor (GI)	Oral RFD (kg-d/mg)	Inhalation RFD (unitless)	Water Conc (mg/L)	Amount Ingested (mg/kg-d)	Lifetime HQ (unitless)	Amount Absorbed (mg/kg-d)	Lifetime HQ (unitless)	
INORGANIC CHEMICALS													
Aluminum	1.8E-4	1.00E-3			1.0E+0	1.00E+0	3.2E+01	1.5E-4	1.5E-4	3.4E-5	3.4E-5	3.4E-5	
Antimony	2.1E-4	1.00E-3			1.5E-1	6.00E-5	7.1E-03	8.3E-5	8.3E-5	7.5E-9	7.5E-9	1.2E-4	
Cobalt	5.0E-6	1.00E-3			5.0E-2	3.70E-2	2.0E-02	3.70E-2	3.70E-2	2.1E-8	2.1E-8	9.1E-7	
Copper	8.8E-4	1.00E-3			1.0E+0	3.00E-1	3.2E-02	4.1E-6	4.1E-6	3.4E-8	3.4E-8	1.6E-4	
Iron		1.00E-3			1.0E+0	3.00E-1	4.6E+01	7.2E-4	7.2E-4	4.8E-5	4.8E-5		
Lead		1.00E-3			1.0E+0	3.00E-1	6.3E-02	3.9E-4	3.9E-4	6.6E-8	6.6E-8		
Magnesium	1.8E-3	1.00E-3			6.0E-2	2.82E-3	2.1E+01	1.4E-5	1.4E-5	2.2E-5	2.2E-5	1.5E-3	
Manganese	9.1E-4	1.00E-3			1.0E-2	7.00E-5	3.9E+00	3.9E-5	3.9E-5	6.1E-8	6.1E-8	8.7E-4	
Vanadium	8.0E-6	1.00E-3			1.0E+0	3.00E-1	5.8E-02	6.6E-6	6.6E-6	4.4E-7	4.4E-7	1.5E-6	
Zinc		1.00E-3			1.0E+0	3.00E-1	4.2E-01						
VOLATILE ORGANIC CHEMICALS													
Cis-1,2-Dichloroethene	9.2E-5	5.30E-3	3.4E-1	1.2E-1	1.0E+0	1.00E-2	6.4E-02	3.0E-7	3.0E-7	6.1E-7	6.1E-7	6.1E-5	
Trichloroethene	3.8E-5	1.60E-2	5.5E-1	2.5E-2	1.0E+0	3.00E-3	2.0E-01	9.4E-7	9.4E-7	6.9E-6	6.9E-6	2.7E-5	
Vinyl chloride		7.30E-3	2.1E-1	2.3E-3	1.0E+0	3.00E-3	7.3E-03	3.4E-8	3.4E-8	8.0E-8	8.0E-8		
SEMIVOLATILE ORGANIC CHEMICALS													
Bis(2-ethylhexyl)phthalate	2.7E-4	3.30E-2	2.1E+1	2.0E+1	1.0E+0	2.00E-2	1.2E-02	5.6E-8	5.6E-8	2.8E-6	2.8E-6	2.6E-4	
TOTALS	4.4E-3									1.4E-3		3.0E-3	

Parameter
 Averaging time 3650 d/life
 Body weight 70 Kg
 Exposure duration (recreational) 10 yrs
 Exposure fraction (recreational) 24 event/yr
 Exposure time (recreational) 1 h/event
 Ingestion rate: Human-water (while swimming) 0.005 L/d
 Skin surface area exposed 1120 cm²

Table C.16
Cancer Risk and Noncancer Hazard Risk Calculation
RME Recreational User Noncancer Risk from Exposure to COPCs In Creek Sediment

Chemical Name	Concentration in sediment (CS), mg/kg	Reference Doses (mg/kg-d)		Absorption factor, unitless	Dermal Absorption Amount Absorbed (IDD), mg/kg-d	Lifetme HQ (HQD), unitless	Amount Ingested (IDIG), (mg/kg-d)	Lifetme HQ (HQIG), unitless	Sum HI (unitless)
		Oral (RFDO)	Inhalation (RFDI)						
INORGANIC CHEMICALS									
Antimony	65E+00	4 0E-4	6 0E-05	0 15	5 5E-09	9 1E-05	3 1E-08	7 6E-05	1 7E-04
Arsenic	1 6E+01	3 0E-4	3 0E-04	1	4 0E-08	1 3E-04	7 5E-08	7 5E-04	3 9E-04
Barium	7 9E+02	7 0E-2	4 9E-03	0 07	6 6E-07	1 4E-04	3 7E-06	5 3E-05	1 9E-04
Iron	1 4E+04	3 0E-1	3 0E-01	1	1 2E-05	3 9E-05	6 8E-05	2 2E-04	2 6E-04
Magnesium	2 5E+03	4 7E-2	2 8E-03	1	2 1E-06	4 8E-04	1 2E-05	1 6E-04	6 4E-04
Manganese	1 6E+03	2 0E-2	8 0E-04	0 06	1 1E-08	1 4E-05	6 1E-08	3 1E-06	1 7E-05
Nickel	1 3E+01	7 0E-3	2 1E-04	0 04	4 1E-08	2 0E-04	2 3E-07	3 3E-05	2 3E-04
Vanadium	4 9E+01	3 0E-1	3 0E-01	0 03	1 3E-07	4 5E-07	7 5E-07	2 5E-06	3 0E-06
Zinc	1 6E+02			1					
VOLATILE ORGANIC CHEMICALS									
No COPCs									
SEMIVOLATILE ORGANIC CHEMICALS									
Benzo(a)anthracene	2 5E-01			1	2 7E-09		1 2E-09		
Benzo(a)pyrene	4 2E-01			1	4 6E-09		2 0E-09		
Benzo(b)fluoranthene	3 5E-01			1	3 8E-09		1 6E-09		
Benzo(g,h,i)perylene	3 6E-01			1	3 9E-09		1 7E-09		
Chrysene	4 2E-01			1	4 6E-09		2 0E-09		
Indeno(1,2,3-cd)pyrene	3 0E-01			1	3 3E-09		1 4E-09		
Total Noncancer Hazard						1 1E-03		8 0E-04	1 9E-03

ASSUMPTIONS

Averaging Time, AT (yr)	10
Adherence Factor, AF (mg/cm ²)	0 08
Body Weight, BW (kg)	70
Contaminant Fraction, CF (unitless)	1
Conversion Factors	
Mass, MCF (kg/mg)	1 00E-06
Time, TCF (d/yr)	365
Exposure Duration	
Dermal, EDD (yr)	10
Inhalation from soil, EDIS (yr)	NA
Inhalation from volatiles, EDIV (yr)	NA
Ingestion, EDIG (yr)	10
Exposure Frequency, EF (d/yr)	24
Fraction Ingested, FIG (unitless)	1
Fraction Inhaled, FIIV (unitless)	NA
Inhalation Rate, IR (m ³ /d)	NA
Ingestion Rate, IGR (mg/d)	5
Particulate Emission Factor, PEF (m ³ /kg)	NA
Respirable Fraction, RF (unitless)	NA
Skin surface area, SA (cm ² /d)	1,120

EQUATIONS

IDD =	$CS * MCF * SA * AF * ABS * EF * EDD$
HQD =	$IDD / RFDD$
IDIS =	$CS * IR * RF * CF * EF * EDIS$
HQIS =	$IDIS / RFDI$
IDIV =	$CS * (1/MF) * IR * FIIV * EF * EDIV$
HQIV =	$IDIV / RFDI$
IDIG =	$CS * IGR * FIG * MCF * EF * EDIG$
HQIG =	$IDIG / RFDO$

TAB

APPENDIX D

Appendix D

Tier 1 TNRCC Form Ecological Risk Assessment

Figure : 30 TAC §350.77(b)

TIER 1: Exclusion Criteria Checklist

This exclusion criteria checklist is intended to aid the person and the TNRCC in determining whether or not further ecological evaluation is necessary at an affected property where a response action is being pursued under the Texas Risk Reduction Program (TRRP). Exclusion criteria refer to those conditions at an affected property which preclude the need for a formal ecological risk assessment (ERA) because there are incomplete or insignificant ecological exposure pathways due to the nature of the affected property setting and/or the condition of the affected property media. This checklist (and/or a Tier 2 or 3 ERA or the equivalent) must be completed by the person for all affected property subject to the TRRP. The person should be familiar with the affected property but need not be a professional scientist in order to respond, although some questions will likely require contacting a wildlife management agency (i.e., Texas Parks and Wildlife Department or U.S. Fish and Wildlife Service). The checklist is designed for general applicability to all affected property; however, there may be unusual circumstances which require professional judgement in order to determine the need for further ecological evaluation (e.g., cave-dwelling receptors). In these cases, the person is strongly encouraged to contact TNRCC before proceeding.

Besides some preliminary information, the checklist consists of three major parts, each of which must be completed unless otherwise instructed. PART I requests affected property identification and background information. PART II contains the actual exclusion criteria and supportive information. PART III is a qualitative summary statement and a certification of the information provided by the person. Answers should reflect existing conditions and should not consider future remedial actions at the affected property. Completion of the checklist should lead to a logical conclusion as to whether further evaluation is warranted. Definitions of terms used in the checklist have been provided and users are strongly encouraged to familiarize themselves with these definitions before beginning the checklist.

Name of Facility:
Air Force Plant 4

Affected Property Location:
Former Carswell Air Force Base / Golf Course Area

Mailing Address:
ASC/ENVR, BLDG. 8
Attn: George Walters
1801 Tenth St, Suite 2

TNRCC Case Tracking #s:
None

Solid Waste Registration #s:
65004

Voluntary Cleanup Program #:
None

EPA I.D. #s:
Carswell – TX0571924042 and TPDES0118257

Figure: 30 TAC §350.77(b) continued

Definitions¹

Affected property - The entire area (i.e., on-site and off-site; including all environmental media) which contains releases of chemicals of concern at concentrations equal to or greater than the assessment level applicable for residential land use and groundwater classification.

Assessment level - A critical protective concentration level for a chemical of concern used for affected property assessments where the human health protective concentration level is established under a Tier 1 evaluation as described in §350.75(b) of this title (relating to Tiered Human Health Protective Concentration Level Evaluation), except for the protective concentration level for the soil-to-groundwater exposure pathway which may be established under Tier 1, 2, or 3 as described in §350.75(i)(7) of this title, and ecological protective concentration levels which are developed, when necessary, under Tier 2 and/or 3 in accordance with §350.77(c) and/or (d), respectively, of this title (relating to Ecological Risk Assessment and Development of Ecological Protective Concentration Levels).

Bedrock - The solid rock (i.e., consolidated, coherent, and relatively hard naturally formed material that cannot normally be excavated by manual methods alone) that underlies gravel, soil or other surficial material.

Chemical of concern - Any chemical that has the potential to adversely affect ecological or human receptors due to its concentration, distribution, and mode of toxicity. Depending on the program area, chemicals of concern may include the following: solid waste, industrial solid waste, municipal solid waste, and hazardous waste as defined in Texas Health and Safety Code, §361.003, as amended; hazardous constituents as listed in 40 Code of Federal Regulations Part 261, Appendix VIII, as amended; constituents on the groundwater monitoring list in 40 Code of Federal Regulations Part 264, Appendix IX, as amended; constituents as listed in 40 CFR Part 258 Appendices I and II, as amended; pollutant as defined in Texas Water Code, §26.001, as amended, hazardous substance as defined in Texas Health and Safety Code, §361.003, as amended, and the Texas Water Code §26.263, as amended; regulated substance as defined in Texas Water Code §26.342, as amended and §334.2 of this title (relating to Definitions), as amended; petroleum product as defined in Texas Water Code §26.342, as amended and §334.122(b)(12) of this title (relating to Definitions for ASTs), as amended; other substances as defined in Texas Water Code §26.039(a), as amended; and daughter products of the aforementioned constituents.

Community - An assemblage of plant and animal populations occupying the same habitat in which the various species interact via spatial and trophic relationships (e.g., a desert community or a pond community).

Complete exposure pathway - An exposure pathway where a human or ecological receptor is exposed to a chemical of concern via an exposure route (e.g., incidental soil ingestion, inhalation of volatiles and particulates, consumption of prey, etc).

De minimus - The description of an area of affected property comprised of one acre or less where the ecological risk is considered to be insignificant because of the small extent of contamination, the absence of protected species, the availability of similar unimpacted habitat nearby, and the lack of adjacent sensitive environmental areas.

¹These definitions were taken from 30 TAC §350.4 and may have both ecological and human health applications. For the purposes of this checklist, it is understood that only the ecological applications are of concern.

Figure: 30 TAC §350.77(b) continued

Ecological protective concentration level - The concentration of a chemical of concern at the point of exposure within an exposure medium (e.g., soil, sediment, groundwater, or surface water) which is determined in accordance with §350.77(c) or (d) of this title (relating to Ecological Risk Assessment and Development of Ecological Protective Concentration Levels) to be protective for ecological receptors. These concentration levels are primarily intended to be protective for more mobile or wide-ranging ecological receptors and, where appropriate, benthic invertebrate communities within the waters in the state. These concentration levels are not intended to be directly protective of receptors with limited mobility or range (e.g., plants, soil invertebrates, and small rodents), particularly those residing within active areas of a facility, unless these receptors are threatened/endangered species or unless impacts to these receptors result in disruption of the ecosystem or other unacceptable consequences for the more mobile or wide-ranging receptors (e.g., impacts to an off-site grassland habitat eliminate rodents which causes a desirable owl population to leave the area).

Ecological risk assessment - The process that evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors; however, as used in this context, only chemical stressors (i.e., COCs) are evaluated.

Environmental medium - A material found in the natural environment such as soil (including non-waste fill materials), groundwater, air, surface water, and sediments, or a mixture of such materials with liquids, sludges, gases, or solids, including hazardous waste which is inseparable by simple mechanical removal processes, and is made up primarily of natural environmental material.

Exclusion criteria - Those conditions at an affected property which preclude the need to establish a protective concentration level for an ecological exposure pathway because the exposure pathway between the chemical of concern and the ecological receptors is not complete or is insignificant.

Exposure medium - The environmental medium or biologic tissue in which or by which exposure to chemicals of concern by ecological or human receptors occurs.

Facility - The installation associated with the affected property where the release of chemicals of concern occurred.

Functioning cap - A low permeability layer or other approved cover meeting its design specifications to minimize water infiltration and chemical of concern migration, and prevent ecological or human receptor exposure to chemicals of concern, and whose design requirements are routinely maintained.

Landscaped area - An area of ornamental, or introduced, or commercially installed, or manicured vegetation which is routinely maintained.

Off-site property (off-site) - All environmental media which is outside of the legal boundaries of the on-site property.

On-site property (on-site) - All environmental media within the legal boundaries of a property owned or leased by a person who has filed a self-implementation notice or a response action plan for that property or who has become subject to such action through one of the agency's program areas for that property.

Figure: 30 TAC §350.77(b) continued

Physical barrier - Any structure or system, natural or manmade, that prevents exposure or prevents migration of chemicals of concern to the points of exposure.

Point of exposure - The location within an environmental medium where a receptor will be assumed to have a reasonable potential to come into contact with chemicals of concern. The point of exposure may be a discrete point, plane, or an area within or beyond some location.

Protective concentration level - The concentration of a chemical of concern which can remain within the source medium and not result in levels which exceed the applicable human health risk-based exposure limit or ecological protective concentration level at the point of exposure for that exposure pathway.

Release - Any spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing into the environment, with the exception of:

(A) A release that results in an exposure to a person solely within a workplace, concerning a claim that the person may assert against the person's employer;

(B) An emission from the engine exhaust of a motor vehicle, rolling stock, aircraft, vessel, or pipeline pumping station engine;

(C) A release of source, by-product, or special nuclear material from a nuclear incident, as those terms are defined by the Atomic Energy Act of 1954, as amended (42 U.S.C. §2011 et seq.), if the release is subject to requirements concerning financial protection established by the Nuclear Regulatory Commission under §170 of that Act;

(D) For the purposes of the environmental response law §104, as amended, or other response action, a release of source, by-product, or special nuclear material from a processing site designated under §102(a)(1) or §302(a) of the Uranium Mill Tailings Radiation Control Act of 1978 (42 U.S.C. §7912 and §7942), as amended; and

(E) The normal application of fertilizer.

Sediment - Non-suspended particulate material lying below surface waters such as bays, the ocean, rivers, streams, lakes, ponds, or other similar surface water body (including intermittent streams). Dredged sediments which have been removed from below surface water bodies and placed on land shall be considered soils.

Sensitive environmental areas - Areas that provide unique and often protected habitat for wildlife species. These areas are typically used during critical life stages such as breeding, hatching, rearing of young, and overwintering. Examples include critical habitat for threatened and endangered species, wilderness areas, parks, and wildlife refuges.

Source medium - An environmental medium containing chemicals of concern which must be removed, decontaminated and/or controlled in order to protect human health and the environment. The source medium may be the exposure medium for some exposure pathways.

Stressor - Any physical, chemical, or biological entity that can induce an adverse response; however, as used in this context, only chemical entities apply.

Figure: 30 TAC §350.77(b) continued

Subsurface soil - For human health exposure pathways, the portion of the soil zone between the base of surface soil and the top of the groundwater-bearing unit(s). For ecological exposure pathways, the portion of the soil zone between 0.5 feet and 5 feet in depth.

Surface cover - A layer of artificially placed utility material (e.g., shell, gravel).

Surface soil - For human health exposure pathways, the soil zone extending from ground surface to 15 feet in depth for residential land use and from ground surface to 5 feet in depth for commercial/industrial land use; or to the top of the uppermost groundwater-bearing unit or bedrock, whichever is less in depth. For ecological exposure pathways, the soil zone extending from ground surface to 0.5 feet in depth.

Surface water - Any water meeting the definition of surface water in the state as defined in §307.3 of this title (relating to Abbreviations and Definitions), as amended.

Figure: 30 TAC §350.77(b) continued

PART I. Affected Property Identification and Background Information

- 1) Provide a description of the specific area of the response action and the nature of the release. Include estimated acreage of the affected property and the facility property, and a description of the type of facility and/or operation associated with the affected property. Also describe the location of the affected property with respect to the facility property boundaries and public roadways.

Air Force Plant 4

Air Force Plant (AFP) 4 became operational in 1942 when Consolidated Aircraft began manufacturing the B-24 bomber for national defense during World War II. In 1953, General Dynamics took over operation of the manufacturing facility. Since 1953, AFP 4 has produced B-36, B-58, F-111 aircraft. The plant currently produces F-16 aircraft. In addition to F-16 aircraft, AFP 4 produces spare parts, radar units, and missile components. On March 1, 1993, Lockheed, Forth Worth Company, took over operations of AFP 4 as a successor to General Dynamics. AFP 4 currently occupies 602 acres.

Manufacturing operations at AFP 4 have resulted in the generation of various hazardous wastes that include waste oils, fuels, spent solvents, paint residues, and spent process chemicals. Throughout most of the plant's history, waste oil, solvents, and fuels were disposed at on-site landfills or were burned during fire training exercises. Chemical wastes were initially discharged to the sanitary sewer system and treated by the City of Fort Worth's treatment system. In the 1970's, chemical process wastes were treated on site at a newly constructed chemical waste treatment system prior to being discharged to the sanitary sewer system. Currently, on site burning of waste has been discontinued while waste oils and solvents are disposed through a contractor. Chemical wastes continue to be treated on site. AFP 4 was placed on the National Priority List (NPL) in August 1990 because of a large release of trichloroethene (TCE) arising from past disposal practices at AFP 4. While the source areas are currently being remediated, the dissolved TCE plume appears to have migrated toward the east of AFP 4 and extends under NAS Fort Worth JRB and the Former Carswell AFB/Base Realignment and Closure (BRAC) area. The plume is referred to as the southern lobe, and is migrating in a southeast direction.

NAS Fort Worth JRB

The NAS Fort Worth JRB started as a modest dirt runway built to service the aircraft manufacturing plant formerly located at AFP 4's current location. In August 1942, the base was opened as Tarrant Field Airdrome and was used to train pilots to fly B-24 bombers. In May 1943, the field was re-designed as Fort Worth Army Air Field. It was renamed Carswell Air Force Base in 1948, and the 7th Bomber Wing became the base host unit. The Strategic Air Command (SAC) mission remained at Carswell AFB until 1992, when the Air Combat Command assumed control of the base upon de-establishment of SAC. In October 1994, the U.S. Navy assumed responsibility for much of the facility, and its name was changed from Carswell AFB to NAS Fort Worth JRB. The principal activities on the base have been maintaining and servicing bombers, fuel tankers, and fighter jet aircraft.

Major industrial operations that have been performed at the NAS Fort Worth JRB include the following: maintenance of jet engines, aerospace ground equipment, fuel systems, weapons systems, pneudraulic systems and general and special purpose vehicles; aircraft corrosion control; and non-destructive inspection activities. Most liquid wastes that have been generated by industrial operations can be characterized as waste oils, recoverable fuels, spent solvent, and spent cleaners. Several landfills exist just up gradient of the BRAC area, with one landfill (SWMU 22) on the western portion of the BRAC property. Two areas of concern (AOC) exist within the BRAC area; they are the AOC 9, the Golf Course Maintenance Yard, and AOC 16, the Family Camp.

In 1991, the Corps of Engineers performed excavation activities at Waste Burial Area 7 (WP-07), SWMU 24, to remove a total of 34 drums, of which 9 were partially full, and 25 were empty. TCE and perchloroethylene (PCE)

were the primary constituents contained within the drums. These drums contributed to the southern lobe TCE plume contamination. As part of an RFI at SWMU 24, an electromagnetic survey was performed on May 2000, for the purpose of confirming drum removal activities performed by the Corps of Engineers. In July, 2000 IT Corporation began excavation activities to investigate twelve geophysical anomalies. A total of 16 metal 55-gallon drums were encountered. Of the 16 drums, 12 were empty, compressed, or A total of 21 metal 55-gallon drums were encountered between two areas. Of the 21 drums, 17 were empty, compressed, or corroded, and contained no liquids. Also discovered were lengths of pipe, a tire iron, and metal post. Three of the drums were still in tact and partially full with an unknown liquid. Analytical results from characterization sampling will be addressed under a separate and pending (December 2000) project report by IT Corporation, but preliminary results indicate that the drums contain at least a fraction of TCE. A fourth in tact drum contained a blue, wet, powdery substance. Analytical results from characterization sampling of this unknown powdery substance will also be addressed in the IT report on excavation activities. Although analytical results from excavation activities are not available for this Internal Draft Risk Assessment, it is expected that the analytical results will be available and incorporated in the Final Risk Assessment.

The resulting southern lobe TCE plume originating from AFP 4 and possibly other NAS Fort Worth source areas covers approximately 453 acres, 75 of which are on the BRAC property. The down gradient extent (TCE at 5 µg/L) of the plume is within 6 feet of the federal property boundary in WHGLRW015. An off-site well has been installed and analytical results are pending. Two additional offsite wells WHGLRW016 and WHGLRW017 (approximately 20 feet from the boundary show no detectable concentrations of TCE).

Attach available USGS topographic maps and/or aerial or other affected property photographs to this form to depict the affected property and surrounding area. Indicate attachments:

Topo map Aerial photo Other

- 2) Identify environmental media known or suspected to contain chemicals of concern (COCs) at the present time. Check all that apply:

Known/Suspected COC Location	Based on sampling data?	
<input type="checkbox"/> Soil ≤ 5 ft below ground surface	<input type="checkbox"/> Yes	<input type="checkbox"/> No
<input type="checkbox"/> Soil >5 ft below ground surface	<input type="checkbox"/> Yes	<input type="checkbox"/> No
<input checked="" type="checkbox"/> Groundwater	<input checked="" type="checkbox"/> Yes	<input type="checkbox"/> No
<input checked="" type="checkbox"/> Surface Water/Sediments	<input checked="" type="checkbox"/> Yes	<input type="checkbox"/> No

Explain (previously submitted information may be referenced):

Detected chemicals in groundwater, surface water and sediment are identified in Tables 6-1, 6-3, and 6-4, respectively.

Figure. 30 TAC §350.77(b) continued

PART II. Exclusion Criteria and Supportive Information**Subpart A. Surface Water/Sediment Exposure**

1) Regarding the affected property where a response action is being pursued under the TRRP, have COCs migrated and resulted in a release or imminent threat of release to either surface waters or to their associated sediments via surface water runoff, air deposition, groundwater seepage, etc.? Exclude wastewater treatment facilities and stormwater conveyances/impoundments authorized by permit. Also exclude conveyances, decorative ponds, and those portions of process facilities which are:

- a. Not in contact with surface waters in the State or other surface waters which are ultimately in contact with surface waters in the State; and
- b. Not consistently or routinely utilized as valuable habitat for natural communities including birds, mammals, reptiles, etc.

X Yes

 No

Explain:

Measured concentrations of volatile and semivolatile chemicals (see Tables 6-3 and 6-4) have been detected in surface water and sediment samples.

If the answer is Yes to Subpart A above, the affected property does not meet the exclusion criteria. However, complete the remainder of Part II to determine if there is a complete and/or significant soil exposure pathway, then complete PART III - Qualitative Summary and Certification. If the answer is No, go to Subpart B.

Soil is not included under this remedial investigation.

Subpart B. Affected Property Setting

In answering "Yes" to the following question, it is understood that the affected property is not attractive to wildlife or livestock, including threatened or endangered species (i.e., the affected property does not serve as valuable habitat, foraging area, or refuge for ecological communities). (May require consultation with wildlife management agencies.)

1) Is the affected property wholly contained within contiguous land characterized by: pavement, buildings, landscaped area, functioning cap, roadways, equipment storage area, manufacturing or process area, other surface cover or structure, or otherwise disturbed ground?

X Yes

 No

Explain:

Figure: 30 TAC §350.77(b) continued

If the answer to Subpart B above is Yes, the affected property meets the exclusion criteria, assuming the answer to Subpart A was No. Skip Subparts C and D and complete PART III - Qualitative Summary and Certification. If the answer to Subpart B above is No, go to Subpart C.

Subpart C. Soil Exposure

- 1) Are COCs which are in the soil of the affected property solely below the first 5 feet beneath ground surface or does the affected property have a physical barrier present to prevent exposure of receptors to COCs in surface soil?

Yes No

Explain:

Soil is not included under this remedial investigation.

If the answer to Subpart C above is Yes, the affected property meets the exclusion criteria, assuming the answer to Subpart A was No. Skip Subpart D and complete PART III - Qualitative Summary and Certification. If the answer to Subpart C above is No, proceed to Subpart D.

Subpart D. *De Minimus* Land Area

In answering "Yes" to the question below, it is understood that all of the following conditions apply:

- ❖ The affected property is not known to serve as habitat, foraging area, or refuge to threatened/endangered or otherwise protected species. (Will likely require consultation with wildlife management agencies.)
- ❖ Similar but unimpacted habitat exists within a half-mile radius.
- ❖ The affected property is not known to be located within one-quarter mile of sensitive environmental areas (e.g., rookeries, wildlife management areas, preserves) (Will likely require consultation with wildlife management agencies.)
- ❖ There is no reason to suspect that the COCs associated with the affected property will migrate such that the affected property will become larger than one acre.

- 1) Using human health protective concentration levels as a basis to determine the extent of the COCs, does the affected property consist of one acre or less and does it meet all of the conditions above?

Yes No

Explain how conditions are met/not met:

The surface water body is contained within a golf course area that is highly maintained and does not serve as a viable habitat for threatened/endangered or otherwise protected species.

Figure: 30 TAC §350.77(b) continued

If the answer to Subpart D above is Yes, then no further ecological evaluation is needed at this affected property, assuming the answer to Subpart A was No. Complete PART III - Qualitative Summary and Certification. If the answer to Subpart D above is No, proceed to Tier 2 or 3 or comparable ERA.

PART III. Qualitative Summary and Certification (Complete in all cases.)

Attach a brief statement (not to exceed 1 page) summarizing the information you have provided in this form. This summary should include sufficient information to verify that the affected property meets or does not meet the exclusion criteria. The person should make the initial decision regarding the need for further ecological evaluation (i.e., Tier 2 or 3) based upon the results of this checklist. After review, TNRCC will make a final determination on the need for further assessment. Note that the person has the continuing obligation to re-enter the ERA process if changing circumstances result in the affected property not meeting the Tier 1 exclusion criteria.

Completed by: Deborah L. McKean, Ph D. (Typed/Printed Name)Senior Toxicologist , IT Corporation (Title)November 20, 2000 (Date)

I believe that the information submitted is true, accurate, and complete, to the best of my knowledge.

_____ (Typed/Printed Name of Person)

_____ (Title of Person)

_____ (Signature of Person)

_____ (Date Signed)

HydroGeologic, Inc.—NAS Fort Worth JRB, Texas

Figure A.1

Topographic/Aerial Map
Golf Course/BRAC Area



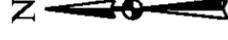
U.S. Air Force Center for
Environmental Excellence

Legend

- - - - - NAS Fort Worth JRB (Carswell Field)

———— Former Carswell Air Force Base

———— 10' Contours



SCALE IN FEET

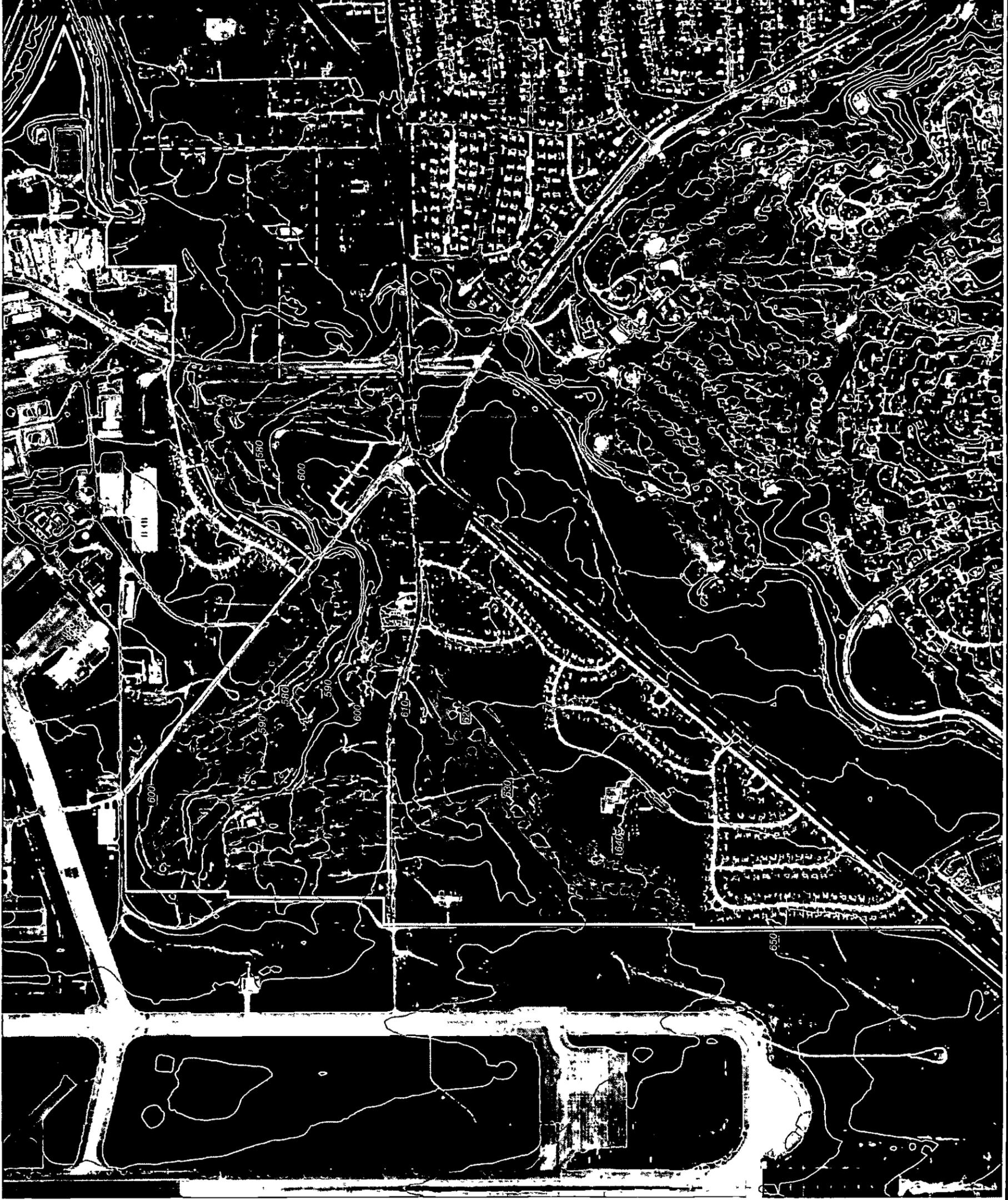
Project AFC001-36CA

Filename X:\AFC001\36ca\Report\Golf_Course.apr

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Revised 12/04/00 cf

Map Source HydroGeologic, Inc.—GIS Database



FINAL PAGE

ADMINISTRATIVE RECORD

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