Public Health Assessment for

HIPPS ROAD LANDFILL
JACKSONVILLE, DUVAL COUNTY, FLORIDA
CERCLIS NO. FLD980709802
MAY 23, 1995

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
Agency for Toxic Substances and Disease Registry

PUBLIC HEALTH ASSESSMENT

HIPPS ROAD LANDFILL

JACKSONVILLE, DUVAL COUNTY, FLORIDA

CERCLIS NO. FLD980709802

Prepared by

The Florida Department of Health and Rehabilitative Services
Under Cooperative Agreement With the
Agency for Toxic Substances and Disease Registry

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6), and in accordance with our implementing regulations 42 C.F.R. Part 90). In preparing this document ATSDR has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 30 day public comment period. Subsequent to the public comment period, ATSDR addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes the public health assessment process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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FOREWARD

The Agency for Toxic Substances and Disease Registry, ATSDR, is an agency of the U.S. Public Health Service. It was established by Congress in 1980 under the Comprehensive Environmental Response, Compensation, and Liability Act, also known as the *Superfund* law. This law set up a fund to identify and clean up our country's hazardous waste sites. The Environmental Protection Agency, EPA, and the individual states regulate the investigation and clean up of the sites.

Since 1986, ATSDR has been required by law to conduct a public health assessment at each of the sites on the EPA National Priorities List. The aim of these evaluations is to find out if people are being exposed to hazardous substances and, if so, whether that exposure is harmful and should be stopped or reduced. (The legal definition of a health assessment is included on the inside front cover.) If appropriate, ATSDR also conducts public health assessments when petitioned by concerned individuals. Public health assessments are carried out by environmental and health scientists from ATSDR and from the states with which ATSDR has cooperataive agreements.

Exposure: As the first step in the evaluation, ATSDR scientists review environmental data to see how much contamination is at a site, where it is, and how people might come into contact with it. Generally, ATSDR does not collect its own environmental sampling data but reviews information provided by EPA, other government agencies, businesses, and the public. When there is not enough environmental information available, the report will indicate what further sampling data is needed.

Health Effects: If the review of the environmental data shows that people have or could come into contact with hazardous substances, ATSDR scientists then evaluate whether or not there will be any harmful effects from these exposures. The report focuses on public health, or the health impact on the community as a whole, rather than on individual risks. Again, ATSDR generally makes use of existing scientific information, which can include the results of medical, toxicologic and epidemiologic studies and the data collected in disease registries. The science of environmental health is still developing, and sometimes scientific information on the health effects of certain substances is not available. When this is so, the report will suggest what further research studies are needed.

Conclusions: The report presents conclusions about the level of health threat, if any, posed by a site and recommends ways to stop or reduce exposure in its public health action plan. ATSDR is primarily an advisory agency, so usually these reports

identify what actions are appropriate to be undertaken by EPA, other responsible parties, or the research or education divisions of ATSDR. However, if there is an urgent health threat, ATSDR can issue a public health advisory warning people of the danger. ATSDR can also authorize health education or pilot studies of health effects, full-scale epidemiology studies, disease registries, surveillance studies or research on specific hazardous substances.

Interactive Process: The health assessment is an interactive process. ATSDR solicits and evaluates information from numerous city, state and federal agencies, the companies responsible for cleaning up the site, and the community. It then shares its conclusions with them. Agencies are asked to respond to an early version of the report to make sure that the data they have provided is accurate and current. When infomed of ATSDR's conclusions and recommendations, sometimes the agencies will begin to act on them before the final release of the report.

Community: ATSDR also needs to learn what people in the area know about the site and what concerns they may have about its impact on their health. Consequently, throughout the evaluation process, ATSDR actively gathers information and comments from the people who live or work near a site, including residents of the area, civic leaders, health professionals and community groups. To ensure that the report responds to the community's health concerns, an early version is also distributed to the public for their comments. All the comments received from the public are responded to in the final version of the report.

Comments: If, after reading this report, you have questions or comments, we encourage you to send them to us.

Letters should be addressed as follows:

Attention: Chief, Program Evaluation, Records, and Information Services Branch, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road (E-56), Atlanta, GA 30333.

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SUMMARY

The seven acre Hipps Road Landfill is in the Jacksonville Heights area of Jacksonville, Duval County, Florida. From 1967 to 1970, several hauling companies reportedly disposed of many substances at the site from two nearby naval air stations. These substances included but were not limited to: airplane parts, electric cable, paints, various solvents, grease, oils, and possibly plating and medical wastes. The property owner then covered the landfill and subdivided it for residential lots. Six homes on the site as well as other nearby homes used private wells as their drinking water source, and many homes continue to use private wells for drinking and household water uses today. In 1983, the Duval County Public Health Unit discovered contamination in nearby private wells and EPA added the site to the Superfund National Priority List. From 1988 to 1990, contractors for Waste Control of Florida, a potentially responsible party, purchased and demolished the six on-site houses and constructed a landfill cover. In the 1990 amended Record of Decision, EPA selected extraction and air stripping for groundwater clean up. ATSDR published the first public health assessment for this site in 1986. Because of a citizen's request and congressional directives, the Florida Department of Health and Rehabilitative Services began a new public health assessment in 1993.

Over 200 nearby residents have expressed site-related health concerns. In general, nearby residents are concerned that exposure to site-related contaminants via ingestion of contaminated groundwater and other exposure routes has seriously affected their health. Residents are also concerned that the groundwater treatment system will again expose them to site-related contaminants and cause additional illnesses.

Over 130 contaminants have been detected in various environmental media near the site. During our initial analysis, we eliminated 90 of these chemicals from further evaluation because they were either found in concentrations below standard comparison values or did not have sufficient toxicological information for further evaluation. We categorized the remaining 43 contaminants into two broad groups, 8 contaminants with drinking water standards and 35 contaminants of concern. Evaluating the 35 contaminants of concern is the focus of this health assessment.

The 35 contaminants of concern were detected in one or more of the following on- and off-site media: subsurface soil, sediment, surface water, groundwater, and air. Most of these media did not have adequate numbers of samples or adequate numbers of analyses for these contaminants. Two media, surface soil (0-3 inches deep) and biota (plants and animals), did not have any sample analyses. This lack of data contributes uncertainty to our toxicological evaluation and a more complete evaluation of exposure pathways.

Based on existing environmental data, the completed exposure pathways were incidental ingestion and skin absorption of contaminants in subsurface soils and sediments; ingestion, skin absorption, and vapor inhalation of contaminants in surface water and groundwater; and inhalation of contaminants from the air stripping tower. The potential exposure pathways

include incidental ingestion and skin absorption of contaminants in surface soil; inhalation of contaminants in ambient air; and ingestion of contaminants in biota (plants and animals).

After further data analyses for the 35 contaminants of concern, we subdivided this group into categories, 15 minimal risk contaminants and the following 20 possible risk contaminants: arsenic, barium, benzene, bromodichloromethane, cadmium, chromium(VI), cresol (total), 1,1-dichloroethane, 1,2-dichloroethane, di(2-ethylhexyl)phthalate, hexachloroethane, lead, manganese, methylene chloride, n-nitrosodiphenylamine, PCBs (total), 1,1,2,2-tetrachloroethane, tetrachloroethene, trichloroethene, and vinyl chloride. Even though toxicological data were available for these 20 contaminants, the literature was sometimes insufficient to draw conclusions about all potential routes of exposure, particularly skin absorption.

Nevertheless, in the past, nearby residents were exposed to arsenic, cadmium, lead, and tetrachloroethene in the environment at doses associated with (noncancer) illnesses in human and animal studies. Furthermore, residents were exposed to arsenic, benzene, 1,2-dichloroethane, di(2-ethylhexyl)phthalate, methylene chloride, PCBs, and vinyl chloride in the environment at levels that could increase their cancer risk. Presently, all nearby residents drinking from private well water might be exposed to low levels of solvents and metals.

Based on these findings, we classified this site as a public health hazard and made several recommendations. To cease or reduce exposure, we recommend performing a well survey to determine which residents living near groundwater contamination are still using their well water, connecting homes with contaminated wells to public supplies, and maintaining the demister on the air stripper as long as the air stripper is used. To complete site-related characterization, we recommend conducting further analyses of off-site surface soils, sediments, and surface and flood waters; and conducting a groundwater characterization study in directions other than northeast of the site. To detect further contaminant migration, we recommend continuing groundwater monitoring in the area, including analyzing influent to the air stripper. To increase public awareness, we recommend implementing an education program for nearby residents, local physicians, and other health care professionals concerning the possible health effects from exposure to contaminants found around the site. Finally, we recommend conducting a health study of nearby residents to determine if adverse health effects have occurred from exposure to contaminants found on or near the site.

BACKGROUND

The Florida Department of Health and Rehabilitative Services (FHRS), in cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR), will evaluate the public health significance of the Hipps Road Landfill site. Specifically, FHRS will determine whether health effects are possible and will recommend actions to reduce or prevent them. ATSDR, located in Atlanta, Georgia, is a federal agency within the U.S. Department of Health and Human Services and is authorized by the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA, also known as "Superfund") to conduct public health assessments at hazardous waste sites.

There are uncertainties inherent in the public health assessment process. In general, these uncertainties fall into four categories: 1) the uncertainty of science in general (that is, science is never 100% certain), 2) the inexactness of the health assessment process, 3) the incompleteness of the information collected thus far, and 4) differences in opinion as to the implications of the information (NJDEP 1990). In general, scientists and public health officials incorporate uncertainties into health assessments by using worst-case assumptions when estimating or interpreting health risks, and by using wide safety margins when setting health-related threshold values. Because of these actions, health assessments tend to err on the side of protecting public health. In accordance with this practice, the assumptions, interpretations, and recommendations we make throughout this public health assessment tend to err in the direction of protecting public health.

A. Site Description and History

The Hipps Road Landfill is in the Jacksonville Heights area of Jacksonville, Duval County, Florida (Figures 1 and 2, Appendix A). The landfill is southeast of the intersection of Hipps Road and Exline Road in the middle of this residential community (Figures 3 and 4, Appendix A). Two small grocery stores, a plant nursery, and a church are located near the site; there are no other commercial or public facilities within the immediate area. Similarly, there are no National Priorities List (NPL), Resource Conservation and Recovery Act, or other industrial facilities within one-half mile of the site. The U.S. Environmental Protection Agency's (EPA) project manager for the Hipps Road Landfill is Patsy Goldberg; her phone number is (404) 347-2643. The Florida Department of Environmental Protection's (formerly known as FDER, the Florida Department of Environmental Regulation) project manager is George Linder; his phone number is (904) 488-0190.

Prior to 1967, the site was a freshwater cypress swamp. Late in 1967, the property owner began using the site as a landfill to eliminate its swampy conditions. Eventually, the landfill covered 6.8 acres and was approximately 25-30 feet deep. During the landfill's operation, several hauling companies reportedly brought many waste materials to the site from two nearby naval air stations. These materials included but were not limited to: lumber, airplane parts, wire, electrical cable, ordnance shells, paint, paint strippers, other solvents, grease, oils, and

possibly plating and medical wastes. Septic tank trucks also may have dumped wastes of unknown composition at the site (FDER 1983d, 1983e). Landfill operations continued until January 1970. The property owner then covered the landfill with a thin layer of soil, subdivided the property, and sold the lots to homeowners. Eventually, six families built homes on, or adjacent to, this landfill. Private wells provided potable water for these homes; one private well actually penetrated the landfill. Other homes in the area also used private wells as their drinking water source. Residents report some homes close to the contaminant plume continue to use private wells for drinking water and other household uses (FDER 1984b; Hipps Road residents, pers. comm.).

From 1967 - 1989, site access was unrestricted. Area residents scavenged materials from the landfill, and children used the site as a play area. In addition, neighborhood children swam and fished in ponds adjacent to the landfill (FHRS 1993b; Hipps Road residents, pers. comm.).

Environmental problems began in 1968, when an area resident reported the presence of a brown sludge, that smelled like airplane stripper, covering one of the ponds adjacent to the landfill. There was a fish kill in this pond in 1968, and in an adjacent pond in 1971 (FDER 1983d). Sporadic complaints about potable water quality in the Hipps Road area began in 1972 (Keneagy 1991). In 1981, taste and odor complaints led to hydrocarbon and pesticide testing for two private wells near the landfill. No contamination was found (FDER 1984a; FHRS 1981a, 1981b). Following another taste and odor complaint in February 1983, the Duval County Public Health Unit (CPHU) found vinyl chloride, methylene chloride, and toluene in an off-site well. By April 1, 1983, the Duval CPHU had sampled ten more wells in the Hipps Road area and found volatile organic compounds (VOCs; hereafter called "solvent") in five wells northeast of the landfill. The four homes with contaminated wells were placed on bottled water. The fifth home had only a trace of the solvent, methylene chloride. Other wells were scheduled for testing (FDER 1983a).

In response to the groundwater contamination at Hipps Road, FDER, the U.S. Geological Survey, Duval CPHU, Jacksonville's Bio-Environmental Services Division (BESD), and the St. Johns River Water Management District formed a multi-agency committee to evaluate the type and extent of contamination (FDER 1983g). Study results indicated a plume of solvents had moved at least 1000 feet northeast of the site. Some of the local wells also contained heavy metals (FDER 1984b). By mid-October 1983, the City of Jacksonville extended public water lines into the neighborhood; however, only those residents who were willing and could afford to pay a "tap in" fee, as well as the monthly water charges, were hooked to city water (FHRS 1983b). In November 1983, the Mayor of Jacksonville declared a water pollution emergency area within a zone delineated around the landfill (City of Jacksonville 1983). All residents within the emergency area were urged, but could not be required, to cease using their private wells and to hook up to city water. The City stopped providing bottled water in December 1983 since the public supply was available to all residences in the plume area (BESD 1983b, 1984). By November 1984, only 44 of 131 residences had hooked up to the public water supply (FDER 1984b, 1993). In January 1985, EPA used CERCLA funds to connect the remaining residents within the contaminated area, who gave permission, to city water (FDER 1993).

In September 1983, EPA assigned the Hipps Road Landfill a MITRE score of 31.94 and placed it on the NPL (EPA 1986d). EPA took the lead role for conducting the remedial investigation/feasibility study (RI/FS) in February 1984 (FDER 1993). The 1985 remedial investigation found a variety of metals and organic compounds within the landfill in quantities exceeding federal Clean Water Act water quality criteria and at least one contaminant exceeding cancer-based criteria for drinking water. Contaminants in the landfill were likely to migrate downward into the lower water table zone and horizontally to the northeast, the direction of groundwater flow. The remedial investigation concluded the landfill was the primary source of nearby groundwater contamination, and confirmed the contaminant plume was approximately 1000 feet northeast of the site. The remedial investigation also concluded that unknown pollution sources, in addition to the landfill, were contributing to the low level groundwater, surface water, and sediment contamination found in the area (EPA 1985a). In May 1986, EPA completed the draft RI/FS, and held a public meeting to discuss the RI/FS results and site cleanup alternatives (FDER 1993). EPA recommended capping and fencing the site, and continuing groundwater monitoring (Burr 1986). Also in May 1986, ATSDR published their public health assessment for the site. The public health assessment concluded that by providing an alternate supply of potable water, EPA had eliminated the only significant route of human exposure creating a public health threat. ATSDR concurred with EPA's cleanup plans by recommending the site be properly closed and the groundwater monitored (ATSDR 1986). Because of strong public opposition to this cleanup proposal, EPA decided to investigate other cleanup alternatives. EPA completed the revised FS in July 1986. The September 1986 Record of Decision (ROD) formalized EPA and FDER's alternative cleanup agreement which included: 1) capping the site, 2) monitoring the groundwater, 3) instituting exposure controls (for example, fencing the site, plugging existing private wells, banning new well drilling in the area, etc.), 4) extracting contaminated groundwater and treating it at the local sewage treatment plant, and 5) beginning site operations and maintenance activities (EPA 1988; FDER 1993).

During the site investigation, EPA identified the U.S. Navy and Waste Control of Florida (WCF) as the primary responsible parties for the Hipps Road Landfill. WCF originally committed to closing the landfill, relocating residents within the cleanup area, conducting groundwater studies, and designing the groundwater recovery system. The Navy, still not admitting liability for site contamination, entered into a separate agreement with WCF, agreeing to fund half of the cleanup activities' cost and provide project oversight (EPA 1991, FDER 1993).

In August 1987, WCF's contractor submitted a satisfactory remedial design work plan to EPA. In January 1989, a partial Consent Decree formalizing WCF's role in the site cleanup was entered by the U.S. District Court in Jacksonville. In this decree, the responsible parties agreed to undertake landfill closure, but did not agree to clean up the groundwater (FDER 1993). To make room for the landfill cover and other cleanup structures, WCF purchased and demolished or moved the homes of the six families located within the site cleanup area (EPA 1989; Keneagy 1987). Landfill cover construction began in May 1989 and finished in September 1990. In March 1990, the responsible parties filed a brief in U.S. District Court stating that groundwater cleanup at the Hipps Road Landfill was not necessary. At the same time, the

responsible parties also submitted an alternate groundwater cleanup proposal to air strip contaminated water and return the treated water to the ground via retention ponds. The legal attempts to eliminate groundwater cleanup from the ROD failed, but EPA accepted the alternative cleanup plan (FDER 1993). At the July 1990 public meeting, many residents opposed the air-stripping proposal for two reasons: 1) the air stripper would merely turn water pollution into air pollution that would re-expose residents who had already been exposed to contaminated drinking water, and 2) EPA had not adequately considered the cleanup of all harmful contaminants in the groundwater, especially the heavy metals (BESD 1990; EPA 1990c). EPA did not concur with the residents' arguments, and in October 1990 signed an amended ROD approving the air-stripping plan. In November 1990, EPA approved the revised the groundwater treatment and design plan (Figure 5, Appendix A). The responsible parties signed a new Consent Decree in August 1991, and it was entered by the U.S. District Court in December 1991.

Preconstruction activities for the groundwater treatment system began in December 1991. In June 1992, EPA held a public meeting to present the groundwater treatment system construction plan. In January 1993, the responsible parties attempted to gain access to local properties for well installation (FDER 1993). Because of difficulties in gaining access to private properties, the responsible parties' contractor installed the needed piezometers and extraction wells along the street right-of-way in front of homes in the contaminated area. In July 1993, they finished constructing the retention ponds (Hipps Road residents, pers. comm.) and began air-stripper operation on September 2, 1993. In response to a FHRS request, EPA agreed to shut down the air stripper after two weeks of operation to allow air sample analysis and data evaluation to ensure protection of public health in the stripper's vicinity (EPA 1993b; FHRS 1993a). Air-stripper operation stopped on September 18, 1993 (Golder Associates 1993a). FHRS' health consultation for the air stripper found the individual contaminant concentrations reaching the nearest residence were unlikely to cause any health effects. Interactive effects among contaminants and combined effects from past exposures were not evaluated in the health consultation (FHRS 1994a). Air stripping resumed in March 1994 (WMF 1994).

Community concern about the Hipps Road Landfill has been and continues to be high. After discovery of the groundwater contamination, fears about health and property values prompted nearby residents to organize a citizens' group known as the Jacksonville Citizens Against Contaminated Water (JCACW). Nearly one-third of the families in the Hipps Road area joined this group to voice their concerns. In June 1983, two JCACW members testified before the U.S. House of Representatives' Environment, Energy, and Natural Resources Subcommittee. They described problems with local government supervision of the landfill, involvement of the U.S. Navy, and community health problems allegedly caused by ingestion of contaminated groundwater coming from the landfill (EPA 1985a). Community health concerns still exist and range from nonspecific symptoms such as headache and dizziness to birth defects, cancer, and other diseases (FHRS 1993b). Community leaders also believe residents are not adequately informed of site-associated activities or the true health hazards posed by the site (EPA 1985a; Hipps Road residents, pers. comm.). In June 1991, at least 50 residents joined a second citizens' group, the Misinformed, Uninformed Concerned Citizens to gain more information

about the landfill, the extent of contamination in the private wells, and the chemicals associated with the site (Keneagy 1991; Hipps Road residents, pers. comm). Later, the Hipps Road Landfill Coalition, Inc., including both citizens' groups, formed and was awarded a $50,000 EPA grant to hire their own technical adviser to review and interpret site-related documents (Hipps Road residents, pers. comm.; Nyenhuis 1993).

In February 1987, 172 Hipps Road residents filed suit against the U.S. Navy, WCF, and three other waste disposal companies in U.S. District court, seeking $463 million for the physical, financial, and emotional losses of homeowners. Of these litigants, only 11 plaintiffs (3 families) had their cases heard in court (Hipps Road residents, pers. comm.; Keneagy 1987, 1991). In December 1991, WCF reached an out-of-court settlement with the original 1987 litigants; settlement terms were undisclosed (Florida Times Union 1991). In January 1992, a U.S. District Court judge found the U.S. Navy and WCF liable for the Hipps Road contamination (Keneagy 1992c). In March 1992, the judge ruled only one plaintiff could recover damages, on the basis his symptoms stopped shortly after he stopped drinking the water. The judge also ruled all plaintiffs were exposed to toxic levels of cancer-causing agents, and were entitled to receive medical monitoring to be paid for by the government (Keneagy 1992b, Marshall 1992). In November 1992, 150 residents returned to court to obtain a medical monitoring program (Keneagy 1992a). To date, the award disbursement to the one plaintiff and the initiation of a medical monitoring program are still pending (Hipps Road residents, pers. comm.). In February 1993, more than 200 residents filed claims against the U.S. Navy, seeking $150 million in damages, for drinking contaminated water in the Hipps Road area (Avery 1993). In July 1993, 23 more lawsuits were filed, claiming toxic wastes from the landfill caused illnesses in the neighborhood (Pinkham and Nyenhuis, 1993). These cases are currently pending. In March 1994, the family of one resident who died of cancer filed suit against the U.S. Navy, seeking compensation for medical expenses, lost wages, and pain and suffering. The judge's decision in this case is still pending (Nyenhuis 1994).

B. Site Visits

Ms. Carolyn Voyles and Mr. Randy Merchant, FHRS, visited the site on June 30 and August 24, 1993; and on May 23, August 31, and November 18, 1994. Each site visit consisted of a windshield survey of the site and surrounding neighborhood to observe current site conditions and verify file information.

A chain-link fence topped with barbed wire surrounds the site, and warning signs are posted on the fence at regular intervals. The landfill itself occupies the western half of the site and has a grass-covered cap that is 5-6 feet above ground level. Monitor wells are visible along the northern and western boundaries of the site. The southern boundary of the site cannot be seen very well from the road because it borders private property.

Off site, there are storm water swales immediately outside of the northern and western fence boundaries. After heavy rains, water collects in these ditches and percolates into the ground. A small cypress pond, approximately ¼ acre in size and located just east of the northeastern side

of the site, is part of a second storm water collection system. Storm water runoff from the site flows into this cypress pond, between two homes, and underneath Camfield Road into a storm water conduit. There is a third storm water runoff pathway northeast of the site. This pathway begins in a vacant lot north of the intersection of Hipps and Bunion Roads, and continues north to Mile Branch Creek (also known as the unnamed tributary to the Ortega River). It is unclear how much of the site's storm water runoff enters this third pathway.

The residential neighborhood around the site is well-established, and there is no new construction in the immediate neighborhood except for that related to site cleanup. Area homes are mostly single-wide trailers and small block houses. Many homes have vegetable gardens, and a few have livestock (horses, fowl, cows). The closest residence to the fenced boundary is approximately 50 feet east of the northeast corner of the site; another residence is less than 100 feet south of the southwestern fenced boundary. There are no schools or special facilities (for example, day care, nursing homes, hospitals) within a one mile radius of the site.

During the June 30, 1993 visit, two retention ponds were being excavated on the eastern side of the site as part of the air-stripping system (Figure 5, Appendix A). Temporary buildings (trailers) and other construction-related equipment were visible on the northeastern part of the site. The groundwater extraction system for the air stripper had been installed along Camfield and Paul Howard Roads, but the air-stripper tower was not yet built. By the August 24, 1993 visit, the air stripper system's construction had been completed. During both site visits, the gate to the site was closed and locked.

At the time of the May 23, 1994 visit, tall plastic sheets, extending several feet upwards from the ground along the fence, had been placed along the site's eastern boundary. These sheets appeared to channel rain water from the site into the off-site cypress pond, and were bulging with dirt in some places. It is not known how effective these structures are in containing dirt on site or channeling storm water into the pond. There was no obvious evidence of contamination in the off-site storm water collection systems. During the May visit, the gate to the site was closed and locked. Nevertheless, FHRS staff saw a large hole in the site's fence along Exline Road, permitting site access. Outside of the fence along Hipps Road, the City of Jacksonville had installed a municipal tap to provide public water temporarily to area residents. Nearby, the City had posted large signs describing their plans for extending public water to all area residents. Finally, HRS staff walked for a short distance along the banks of Mile Branch Creek to observe its depth and flow rate. Near Shindler Road, the creek appeared slow moving with depths ranging from a couple of inches to four or five feet.

During the August 31, 1994 site visit, the tall plastic sheets containing dirt along the eastern fence had been removed, but shorter plastic sheeting remained. New fencing had been placed around the southern periphery of the off-site cypress pond; the northern shore was difficult to see from the road, and FHRS staff could not tell if it had also been fenced off. The hole in the fence along Exline Road still existed.

During the November 18, 1994 site visit, the short plastic sheets still lined the eastern fence boundary. In some places, there were large gaps between the sheets. In other places, the sheeting had been pressed to the ground. Water was being pumped from the back pond (closest to the air stripper) to the front pond, and from the front pond onto the front of the site, reportedly because of a filtration problem in the back pond. The water in the swales on Exline Road was dark and clear with no odor. The hole in the fence along Exline Road still existed. From the back yard of a home on Camfield Road, a hole in the side of the air stripper was visible.

Either before or after each site visit, Ms. Voyles and Mr. Merchant met with community leaders to discuss the public health assessment process, verify site history, and gather community health concerns. During these meetings, residents reported:

- Prior to the posting of warning signs, local residents routinely walked on the site. After the site was fenced, vandals occasionally gained site access. In addition, a few residents occasionally enter the site through the hole in the fence to observe cleanup activities, exercise their dogs, etc.

- Prior to 1985, neighborhood children attended schools 6-7 miles away. Currently, the closest school is approximately 1 mile away.

- Storm water runs off the site to the north and northeast through the series of swales, ditches, and creeks. Children frequently play in the storm water ponds, and used to swim in the cypress pond close to the site.

- Neighborhood children swam and fished in the on-site ponds before cleanup activities began. Currently, residents swim and fish in Mile Branch Creek, the local receiving body of water for storm water runoff.

- Neighborhood children hunt and eat squirrel, doves, and other birds that may have contact with the site.

- Even though public water lines will eventually be available to residents in the Hipps Road area, homeowners will still have to pay to tap in and many residents cannot afford the connection fees. Cost was also a problem in 1983, when public supply lines were extended to homes within the water pollution emergency area, and only 44 of 131 homes connected to city water.

- Residents report repeated trouble with the on-site retention ponds overflowing and flood waters pouring into adjacent yards east of the site. Such flooding reportedly has occurred several times during the fall of 1994.

On February 3, 1994, Mr. Merchant, Ms. Winter, and Ms. Lanzon, FHRS, held an additional meeting with community leaders to discuss the status of the public health assessment, FHRS'

preliminary evaluation of the air stripper, and the availability of environmental medicine training for local doctors. This latter issue is of particular interest to nearby residents because they believe local health care professionals are not adequately trained to consider the effects of environmental exposures when treating the residents' illnesses.

On September 1, 1994, Dr. Isabel Stabile, M.D., PhD., an Associate Research Scientist at Florida State University's Center for Biomedical and Toxicological Research and Hazardous Waste Management, gave a seminar entitled "Hipps Road Landfill: Clinical Environmental Issues" to approximately 25 health care providers at the St. Vincent's Medical Center in Jacksonville, Florida. This presentation was paid for under an existing cooperative agreement between FHRS and ATSDR's Division of Health Studies. Doctors attending the seminar not only learned about possible health effects related to the site, but also received Continuing Medical Education credit for their attendance. Nearby residents have requested another seminar be given to doctors at the Orange Park Hospital in the near future.

C. Demographics, Land Use, and Natural Resource Use

Demographics

There are approximately 150 homes in the residential neighborhood surrounding the Hipps Road Landfill, and 190 residences located within one mile topographically down slope and hydraulically down gradient of the site (EPA 1992b, Golder Associates 1992a). Extrapolating from 1990 census data, there are an estimated 400-500 people currently living in the Hipps Road area. The racial makeup of the census tract containing the site is 88% White, 8% Black, and 3% Hispanic. The median age for the tract is 28 years, and 21% of the tract residents are children between the ages 0-9 years. The median family income for the census tract is $37,045 (BOC 1992).

Land Use

The land use within one mile of the site is mostly residential with a few commercial facilities. A church, a plant nursery, and two small grocery stores (one of which sells gasoline) are the only public and commercial facilities located within the immediate neighborhood of the site. New residential developments are located 1+ miles south and southeast of the site. The closest school is 1.6 miles south of the site. There are no day care facilities, hospitals, nursing homes, or recreational areas within one mile of the site.

Natural Resource Use

Groundwater in the site area occurs in a surficial aquifer underlain by the Floridan aquifer (Figure 6). In the vicinity of the site, the surficial aquifer is a four-layered system consisting of (in descending order): the Sand aquifer, the semi-confining Clay Marl unit, the Limestone aquifer, and the Lower Marl unit. The Sand aquifer and Limestone aquifer are the main water-bearing units in this system. The surficial aquifer is approximately 200 feet thick and is

underlain by the Hawthorn Group, a confining aquiclude approximately 300 feet thick. The Floridan aquifer underlies the Hawthorn Group (EPA 1986d; Golder Associates 1992a).

The Sand aquifer is of primary interest to this public health assessment, as it contains both the contaminant plume and most drinking water wells for local residents. Water is usually reached between 2-5 feet below ground surface in this unit. Near the landfill, groundwater flows downward at an average linear vertical flow velocity of 0.007-0.012 feet per day. Groundwater flows outward from the landfill to the north and east toward the Ortega River system at an average linear horizontal flow velocity of 0.02-0.10 feet per day (EPA 1986d).

Before the site was fenced, neighborhood children swam and fished in two ponds near the landfill. These ponds have since been destroyed by cleanup activities. Currently, area residents fish and swim in Mile Branch Creek, a tributary of the Ortega River ½ mile north of the site (Hipps Road residents, pers. comm.). 1986 groundwater modeling data indicate plume contaminants could enter this creek by 1993, with maximum contaminant concentrations entering the creek by 2008. These models estimate the maximum concentrations entering the creek will be less than 0.07 μg/l (micrograms per liter), using assumptions that do not consider biodegradation, adsorption, or volatilization of groundwater contaminants, nor contaminant dilution in the surface water (EPA 1986d).

Many area homes have small vegetable gardens, and neighborhood children hunt and eat small game that may have contact with the site.

D. Health Outcome Data

FHRS epidemiologists attempted to evaluate the cancer rates in the 32222 zip code encompassing the site. This evaluation considered all cancer data contained in the Florida Cancer Data System (FCDS), a FHRS program operated by the University of Miami School of Medicine that covers all cancers reported in Florida between 1981 and 1987. In addition, in response to the residents' request, FHRS epidemiologists reviewed the 1991 epidemiology report prepared for the Hipps Road residents' lawsuit against the U.S. Navy (Paigen 1991). We discuss the results of these reviews in the Public Health Implications, Health Outcome Data Evaluation section.

COMMUNITY HEALTH CONCERNS

Over 200 nearby residents have expressed site-related health concerns. In general, nearby residents are concerned that exposure to site-related contaminants via ingestion of contaminated groundwater and other exposure routes has seriously affected their health. Residents are also concerned that the groundwater cleanup system will again expose them to site-related contaminants and cause additional illnesses. In addition, residents are concerned that testing of environmental media has been inadequate to identify all of the contaminants, that their illnesses are undiagnosed due to a lack of environmental medicine training for local

physicians, and that governmental officials have minimized the extent of contamination and severity of their illnesses.

We compiled the following health concerns from newspaper articles, EPA reports, transcripts of public meetings, court records, and our June 30, 1993, August 24, 1993, February 3, 1994 and May 23, 1994 meetings with community leaders:

Circulatory System Complaints:

1. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused heart and circulatory problems in the community, including: blood clots, anemia, blood disorders, blood poisoning, phlebitis, heart murmurs (including mitral valve prolapse in males), arrhythmias, palpitations, cardiomyopathy, pericarditis, atherosclerosis, high blood pressure, angina, other chest pain, aortic aneurysms, heart attacks, and stroke.

2. Nearby residents are concerned that some children in the community have never had normal blood counts (that is, red cell to white cell ratio) and many residents have chronically high white cell counts.

3. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused leukemia in the community.

4. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused some residents to die from heart disease.

Digestive System Complaints:

5. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused digestive problems in the community, including: oral ulcers, submaxillary gland problems, stomach pain, stomach ulcers (in children 10+ years and older, and in adults), reflux problems, herniated bowels, appendicitis, gas pains, pancreas attacks, gallbladder attacks, gallstones, gallbladder removal, chronic disaccharidase deficiencies, gastroenteritis (including gastritis, enteritis, and gastrointestinitis), hepatitis (in both children and adults), other liver dysfunction (including jaundice and enlarged liver), colitis, diverticulitis, spastic colon, proctitis, constipation, acute and chronic diarrhea, and rectal bleeding.

6. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air during pregnancy caused a child to be born with decayed teeth (decayed in the sack) and another child to be born with a navel hernia.

7. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused esophageal, stomach, liver, and colon cancer in the community.

Endocrine System Complaints:

8. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused endocrine system problems in the community, including: hypoglycemia, diabetes, and thyroid trouble.

Excretory System Complaints:

9. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused excretory problems in the community, including: bladder infections, kidney infections, urinary tract infections, hematuria, cystitis, urethritis, ureteral reflux, incontinence, bladder suspension, kidney stones, and kidney disease (requiring kidney removal).

10. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air during pregnancy caused a child to be born without any kidneys and another child to be born with severe kidney disease that required kidney removal shortly after birth.

11. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused bladder and kidney cancer in the community.

12. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused residents to die from kidney disease.

Hypersensitivity Complaints:

13. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused them to become hypersensitive to the presence of chemicals. Several residents report they can smell chemicals in the air even when nonresidents cannot smell them, and other residents report they cannot tolerate any chemical smell (for example, while using oven cleaners or going into hardware stores where chemicals are present). One resident reports experiencing a runny nose, a feeling of bulging eyes, and an almost emotional response to immediately get away from the chemicals when encountering a chemical smell.

Immune System Complaints:

14. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused immune system problems in the community, including: swelling of lymph nodes, mononucleosis, and lupus.

15. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air caused residents to die from lymphoma.

Learning Disabilities:

16. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused community children to have learning disabilities, low IQ (intelligence quotient) scores, memory problems, and behavioral problems in school.

Mental Health Complaints:

17. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused depression, panic attacks, nervous breakdowns, psychosis (including schizophrenia), and attempted suicides in the community.

Nervous System Complaints:

18. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused nervous disorders in the community, including tremors or trembling; pain, numbness, tingling, or loss of feeling in hands, feet, arms, or around the lips; weakness in hands and dropping things; ringing in ears; neuralgia; ganglion cysts; Parkinson's disease; and meningitis.

19. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air during pregnancy caused a child to be born with 1/3 of the brain missing, and another child to be born with only half of the brain developed and cerebral palsy.

20. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air caused a resident to die from a brain tumor.

21. Nearby residents are concerned the neurotoxin tri-ortho-cresyl phosphate has not been analyzed for but was present in the groundwater. Residents are concerned exposure to this substance may have adversely affected their health.

Reproductive System Complaints:

22. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused female reproductive system problems in the community, including: abnormal female breast development, abnormal female reproductive organ development, ovarian adhesions and cysts, vaginal cysts, reproductive organ tumors, pelvic inflammatory disease, heavy menstrual bleeding (requiring a visit to a doctor), difficulty in conceiving, postpartum difficulty, persistent lactation, hysterectomy (in both young and older women), abnormal vaginal bleeding after a hysterectomy, abnormal or precancerous cervical cells (in teenagers and older women, requiring rechecks every 3-6 months), oophorectomy, and breast cysts.

23. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused male reproductive system problems in the community, including: lumps or swelling in the groin (in children), epididymitis (in children), other swelling or pain in the testes (in children and adults), and difficulty in conceiving.

24. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air during pregnancy caused a child to be born with a twisted testicle.

25. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air during pregnancy has caused miscarriages (including loss due to blighted ovum and nonimmune fetal hydrops), premature birth, and delayed birth in the community.

26. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused breast, cervical, ovarian, and prostate cancer in the community.

Respiratory System Complaints:

27. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused respiratory problems in the community, including: sinus problems, allergy problems, asthma, bronchitis, pleurisy, pneumonia, shortness of breath, dyspnea, and infant apnea.

28. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air during pregnancy caused children to be born with rib cage/chest deformities and hyaline membrane disease.

29. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused lung cancer in the community.

30. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused some residents to die from lung disease, including lung cancer.

Skeletal/Muscular and Other Connective Tissue Complaints:

31. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused skeletal/muscular problems and connective tissue disorders, including: rhabdomyolysis, weak joints and bones, arthritis, cold gout, bursitis, bone deterioration, disc problems (in adults, 20+ years and older), chondromalacia patella, hip infections, vertebral spurs, Schmoral's nodes on lumbar vertebrae, and back pain.

32. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air during pregnancy caused children in the community to be born with turned feet, twisted legs, crooked spines, and a cleft palate.

Skin Complaints:

33. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused: abnormally dry skin, unexplained hand and foot rashes, itching skin, various benign skin tumors, actinic keratosis, lichen planus, warts, hives, and Herpes viral infections in children.

34. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air during pregnancy caused a child to be born with skin cancer and other children to be born with benign skin tumors.

35. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused melanoma, basal cell carcinoma, and other skin cancers in the community.

Visual Complaints:

36. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused visual problems in the community, including: cataracts, blurred vision, eye irritation, burning eyes while showering, and other visual disturbances.

Nonspecific Illness and Unexplained Death Complaints:

37. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused the following nonspecific illnesses and complaints in the community: dental problems (including loose teeth), nose bleeds, muscle spasms, equilibrium loss (including dizziness, loss of balance, vertigo, Meniere's syndrome, lightheadedness, unsteadiness), clumsiness, falling, difficulty walking, swelling (including edema, angioedema, and swelling in limbs), nausea, vomiting, dehydration, acute and chronic headaches, migraines, fatigue, lethargy, fainting (including syncope and black outs), seizures, fevers, frequent flus and colds, chronic sore throats, chronic ear infections, premature hair loss, chronic insomnia and other sleep disturbances, anxiety, nervousness, irritability, memory problems, and difficulty in healing after surgery.

38. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused the following illnesses and complaints in the community: vocal cord nodules, fluid in ears (infant), and adenocarcinoma.

39. Nearby residents are concerned that exposure to contaminated groundwater, surface water, sediment, soil, and air has caused several unexplained deaths in the community. A child died shortly after birth from unknown causes. In addition, residents report there are several other cases in which the causes of death could not be found, even after autopsies were performed on the deceased.

Health of Pets:

40. Nearby residents report that pets in the community have had the following health problems: dogs have had cancer, kidney problems, and birth defects; one dog developed an abnormally swollen head; thoroughbred horses have gone crazy, necessitating their removal from racing; one horse died from kidney problems after drinking from surface waters close to the site; and several cats, monkeys, ferrets, chickens, and pet birds bave died unexpectedly.

Other (Nonhealth) Concerns:

41. Nearby residents are concerned about declining values of property near the site. Some residents report they cannot sell their homes because of their proximity to the site. These residents also believe even if they were able to sell their homes, they would not have enough money to move out of the neighborhood because of the money they've lost from declining property values. At least one resident has rental property in the area and reports a loss of income because of the difficulty in finding tenants to live near the site.

42. Nearby residents are concerned that government officials responsible for investigating or cleaning up the site have not considered or do not have all of the data generated about the site, particularly the data values showing the greatest contamination. Some residents believe officials have played down the examination of site-related illnesses and will continue to do so in the future.

43. Nearby residents are concerned that site-related contamination has not been fully identified or delineated. Some residents point out the lack of sampling data for some exposure points, such as volatilization of solvents from nearby surface waters, landfill and yard soils, and while showering. Others think the extent of groundwater contamination has not been properly delineated, and the contaminant plume is actually much larger in all directions than government officials currently believe or publicly state.

ENVIRONMENTAL CONTAMINATION AND OTHER HAZARDS

In this section, we review the environmental data collected at the site, evaluate sampling adequacy, select contaminants of concern, and list the maximum concentration and detection frequency for the contaminants of concern in the various media (that is, water, soil, and air). We select contaminants of concern based on the following factors:

1. Concentrations of contaminants on and off site. Although background concentrations are useful in determining if contaminants are site-related, contaminants are only eliminated from further consideration if both the background and on-site concentrations are below standard comparison values. This is necessary to assess the public health risk to all contaminants detected, whether site-related or not.

2. Field data quality, laboratory data quality, and sample design.

3. Community health concerns.

4. Comparison of maximum on- and off-site concentrations with published ATSDR standard comparison values. ATSDR's published standard comparison values are media-specific concentrations used to select contaminants for further evaluation. They are not used to predict health effects or to set clean-up levels. Contaminants with media concentrations above an ATSDR standard comparison value do not necessarily represent a health threat, but are selected for further evaluation. Contaminants with media concentrations below an ATSDR standard comparison value are unlikely to be associated with illness and are not evaluated further.

5. Contaminants without ATSDR standard comparison values, but which have toxicological information published in documents called ATSDR toxicological

profiles. These profiles are chemical-specific and contain a variety of toxicological information found in the scientific literature.

We used the following ATSDR standard comparison values (ATSDR 1993a), in order of priority, to select contaminants of concern:

1. EMEG--Environmental Media Evaluation Guide--derived from ATSDR's Minimal Risk Level (MRL) using standard exposure assumptions, such as ingestion of two liters of water per day and body weight of 70 kg for adults. MRLs are an estimate of daily human exposure to a chemical likely to be without an appreciable risk of noncancerous illnesses.

2. CREG--Cancer Risk Evaluation Guide--calculated from EPA's cancer slope factors, is the contaminant concentration that is estimated to result in no more than one excess cancer per one million persons exposed over a lifetime.

3. RMEG--Reference Dose Media Evaluation Guide--derived from EPA's Reference Dose (RfD) using standard exposure assumptions. RfDs are an estimate of daily human exposure to a chemical likely to be without an appreciable risk of noncancerous illnesses.

4. LTHA--Lifetime Health Advisory for Drinking Water--EPA's estimate of the concentration of a contaminant in drinking water at which illnesses are not expected to occur over a lifetime of exposure. LTHAs provide a safety margin to protect sensitive members of the population.

Because of the community's concern about the health effects of all contaminants, especially cancer-causing agents, we used the lowest value for either the EMEG or CREG when selecting contaminants of concern. This ensured the selection of the maximum number of contaminants for further evaluation.

Over 130 contaminants have been detected in various environmental media near the site (Table 1, Appendix B). Using the methodology described above, we eliminated 28 chemicals detected in various media at concentrations below their standard comparison values from further consideration (Table 2, Appendix B). Sixty-three other chemicals had no standard comparison values, and the human health data were insufficient to determine their public health significance, requiring us to eliminate these contaminants from further consideration as well (Table 3, Appendix B). We divided the remaining contaminants into two broad categories: contaminants with drinking water standards and contaminants of concern. We evaluated these categories separately. pH and the eight contaminants in the contaminants with drinking water standards category include inorganic chemicals found at the site that have primary or secondary drinking water standards established in Florida but do not have published comparison values. We discuss our findings for contaminants in this category in

the Public Health Implications section below. We classified the remaining 35 contaminants as contaminants of concern. These contaminants are:

Arsenic	Cyanide	Methylene Chloride
Barium	DDT	Naphthalene
Benzene	1,4-Dichlorobenzene	Nickel
Beryllium	1,1-Dichloroethane	n-Nitrosodiphenylamine
Bromodichloromethane	1,2-Dichloroethane	PCBs (total)
Cadmium	1,2-Dichloropropane	Selenium
Chlorobenzene	Di(2-ethylhexyl)phthalate	1,1,2,2-Tetrachloroethane
Chlorodibromomethane	1,2-Diphenylhydrazine	Tetrachloroethene
Chloroform	Hexachloroethane	Tin
Chromium(VI)	Lead	Trichloroethene
Cobalt	Manganese	Vinyl Chloride
Cresol (total)	Mercury	

We summarize the environmental sampling data for the contaminants of concern in Tables 4 through 13, Appendix B. In nearly all cases, the laboratory analyses did not specify the valence of the chromium detected. Since hexavalent chromium (that is, chromium(VI)) is the most toxic form of the metal, we assumed all chromium detected to be hexavalent chromium. Similarly, some laboratory analyses did not specify the isometric form of dichlorobenzene. When this specification was not made, we assumed the dichlorobenzene detected to be the para isomer (that is, 1,4-dichlorobenzene, also known as p-dichlorobenzene), the most toxic form. These assumptions are the most protective of public health. In addition, because the sampling around the site focused on the seven PCB mixtures that together comprised 98% of the PCBs sold in the United States (ATSDR 1993q), this health assessment focuses on these same seven mixtures. These PCB mixtures contain: Aroclor-1016, -1221, -1232, -1242, -1248, -1254, and -1260.

ATSDR standard comparison values are used only to select contaminants of concern for further consideration. Identification of a contaminant of concern in this section does not necessarily mean that exposure will be associated with illnesses. Identification serves to narrow the focus of the public health assessment to those contaminants most important to public health. When we selected a contaminant of concern in one medium, we also reported that contaminant in all other media. We evaluate the contaminants of concern in subsequent sections and determine whether exposure has public health significance.

To identify industrial facilities that could contribute to the contamination near this site, we searched the 1987, 1988, 1989 and 1990 EPA Toxic Chemical Release Inventory (TRI) data bases. EPA developed TRI from the chemical release information (air, water, and soil) provided by certain industries. The Hipps Road Landfill site is in the 32222 zip code area. Our TRI search of this zip code revealed no industries reporting releases of chemicals found at levels of concern at this site.

In this assessment, we discuss the contamination that exists on the site first and separately from the contamination that occurs off the site.

A. On-site Contamination

For the purposes of this evaluation, we defined "on-site" as the area within the fenced boundaries of the Hipps Road Landfill at present (Figure 7, Appendix A). This area includes not only the landfill, but also the area used by the groundwater treatment system.

We compiled data in this subsection from the following sources: BESD samples (BESD 1983a); Duval CPHU samples (FHRS 1981b, 1983c); EPA's 1984 site screening study, 1985 draft RI/FS work plan, and 1985 RI field work (EPA 1985a, 1985c, 1986d); CompuChem's private well samples (CompuChem 1988); Disposal Safety's groundwater contamination assessment (Disposal Safety 1990); Golder Associates' groundwater recovery system design report (Golder Associates 1990). We reviewed several other reports containing on-site sample data results (EPA 1985b, 1986a, 1986b, 1986c; ATSDR 1986) but determined these data had already been presented in and counted from earlier reports.

In counting the number of analyses for a contaminant, we used raw data whenever these data were available to us. In some cases, such as in our review of the remedial investigation data, we could only count the results presented in the report's summary data tables because the raw data were not available from EPA or other governmental agencies. Therefore, we acknowledge the total number of samples for some contaminants is likely greater than the number we show in our data tables, and some contaminants may have been analyzed for that are not identified in the summary reports we examined. When we were able to identify duplicate samples among the many reports we reviewed, we counted these samples only once.

Overall, we found the number of on-site samples and analyses too few to fully characterize the nature and extent of soil or water contamination at the site. Our having only summary data to review from some sources may have contributed to our finding small sample numbers for some contaminants. In addition, Disposal Safety's review of the 1985 RI solvent analyses found holding times were greatly exceeded, indicating that concentrations for these compounds may be underestimated (Disposal Safety 1990). Finally, most on-site samples were collected only one time from any given sample point; consequently, we do not have the data to examine how contaminant concentrations might have changed over time. These deficiencies precluded our determining if individual contaminants of concern were site-related or not.

On-site Surface Soil (0-3 inches deep)

There is no record of surface soil samples (0-3 inches deep) being collected on site. Although EPA collected soil samples at three locations on site, they did not specify sample depths (EPA 1985c). We consider the results from these EPA soil samples under the

subsurface soil category. The RI report mentions collection of two on-site surface soil samples for dioxin analysis, but does not give the sample depths or present the analytical results (EPA 1986d); therefore, we do not use these data in our analysis. The lack of surface soil samples is a significant data gap for this public health assessment because there were homes on site, and family members played or gardened in on-site soils. Without sample data, we cannot evaluate the potential health effects from the resident's exposure to on-site surface soils. Nevertheless, there is no need to collect on-site surface soil samples in the future because the landfill is capped, the on-site homes are gone, and site access is restricted.

On-site Subsurface Soil (deeper than 3 inches)

Between 1984 and 1985, EPA collected on-site subsurface soil samples at 20 locations in the landfill (Figure 8, Appendix A). In 1984, EPA collected soil samples (unspecified depth) at three locations on the eastern side of the landfill. The EPA report identified two of the samples as composites, but did not identify the sample type for the third (EPA 1985c). In 1985, EPA collected subsurface soil samples from 17 boreholes in the landfill. EPA took these composite samples at depths ranging from 15 - 26.5 feet, and used the sample results to identify the chemical waste composition and distribution within the landfill (EPA 1986d). In our analysis, we used raw data from EPA's site screening report and summary data from the RI.

During the RI, EPA collected one soil sample for background data. They collected this sample while drilling a temporary well less than ¼ mile from the landfill boundary (EPA 1986d). Because this sample was taken within the area we judged likely to be affected by the site, we did not consider this sample point representative of background conditions. Consequently, we did not use data from this point for background information in our analysis.

Sixteen contaminants of concern were detected in on-site subsurface soils (Table 4, Appendix B). Four of these (arsenic, cadmium, di(2-ethylhexyl)phthalate, and 1,2-diphenylhydrazine) were found in concentrations above their respective comparison values for soil, and four others (chromium, 1,4-dichlorobenzene, lead, and nickel) are known or suspected cancer-causing agents. Six contaminants of concern (benzene, chlorobenzene, cyanide, manganese, mercury, and methylene chloride) were found in concentrations below their respective comparison values. The other two detected contaminants of concern (naphthalene and tin) did not have comparison values for soil. Eighteen contaminants of concern were not detected, and one contaminant of concern was not analyzed for in the on-site subsurface soils.

For the purposes of this public health assessment, there were not enough samples taken or analyses done to fully characterize on-site subsurface soil quality. In a couple of cases, such as for arsenic and lead, there were enough analyses to characterize on-site subsurface soil contamination by these substances. However, for most compounds, there were too few

analyses to fully characterize on-site subsurface soil contamination. Nevertheless, there is no need to collect more on-site subsurface soil samples as long as the site remains undisturbed, the fence and cap continue to be maintained, and the on- and off-site groundwater continue to be monitored. If any of these conditions are not met or if there are plans to develop the site in the future, a comprehensive study of the landfill's contents will need to be conducted.

On-site Sediment

Between 1983 and 1985, various parties collected on-site sediment samples in at least 4 pond or ditch locations (Figure 9, Appendix A). In 1983, BESD sampled one on-site pond at the request of a private citizen. The BESD data did not include a map of the sample location, and we could not determine which on-site pond was sampled. (This sample location is not included in Figure 9, Appendix A.) BESD did not identify the collected sample as grab or core (BESD 1983a). During the 1984 site screening study, EPA collected one sediment sample from an on-site pond 75 feet east of the landfill, and another sediment sample from a drainage ditch immediately south of the landfill. EPA did not indicate if the samples taken were grab or core (EPA 1985c). During the 1985 RI fieldwork, EPA collected grab samples from two ponds each 75 feet east of the landfill for inclusion in the remedial investigation. The two ponds EPA sampled during the RI were different from the pond sampled during the site screening study (EPA 1986d). In our analysis, we used raw data from BESD and EPA's site screening report, and summary data from the RI.

EPA was the only investigator to collect a background sediment sample. During the RI, they collected this sample from an intermittent stream approximately 10,000 feet south of the site (EPA 1986d). The acceptability of this sample as representative of true background conditions is questionable for several reasons. First, the sample point is nearly two miles south of the site and not in the proper location (that is, not upgradient) to determine if the site is having an effect on pond sediments. Second, on-site ponds are groundwater-fed, not stream-fed. Third, a road crosses the background stream and is the likely source of lead found in the background sediment. Fourth, at least six of the contaminants found in the background sediment have not been found in the area around the site. For these reasons, we do not consider CDM's background sample adequate, and we do not include these data in our analysis.

Ten contaminants of concern were detected in on-site sediments (Table 5, Appendix B). Two of these (arsenic and PCBs) were found in concentrations above their respective comparison values for soil, and two others (chromium and lead) are known or suspected cancer-causing agents. Four contaminants of concern (cyanide, 1,2-diphenylhydrazine, manganese, and mercury) were found in concentrations below their respective comparison values. The other two detected contaminants of concern (cobalt and cresol) did not have comparison values for sediment. Twenty-five contaminants of concern were analyzed for but not detected in the on-site sediments.

For the purposes of this public health assessment, site investigators did not collect enough samples to fully characterize on-site sediment quality. Nevertheless, site cleanup activities have eliminated the on-site ponds and ditches, and no more samples can be collected.

On-site Surface Water

Between 1983 and 1985, various parties collected on-site surface water samples in at least 4 pond or ditch locations (Figure 10, Appendix A). In 1983, BESD sampled one on-site pond at the request of a private citizen. The BESD data did not include a map of the sample location, and we could not determine which on-site pond was sampled (BESD 1983a). (This sample location was not included in Figure 10, Appendix A.) During the 1984 site screening study, EPA collected one surface water sample from an on-site pond 75 feet east of the landfill, and another surface water sample from a drainage ditch immediately south of the landfill (EPA 1985c). During the 1985 RI fieldwork, EPA collected surface water samples from two ponds each 75 feet east of the landfill for inclusion in the remedial investigation. These two ponds were different from the pond EPA sampled during the site screening study (EPA 1986d). In our analysis, we used raw data from BESD and the EPA site screening report, and summary data from the RI.

EPA was the only investigator to collect a background surface water sample. They collected this sample from an intermittent stream approximately 10,000 feet south of the site (EPA 1986d). We do not consider this sample representative of true background conditions because the sample point is too far from the site and in an unsuitable location, the background stream does not feed any of the ponds, and outside sources seem to be contributing contaminants to the background stream.

Four contaminants of concern detected were detected in on-site surface waters (Table 6, Appendix B). One of these (DDT) was found in a concentration above its comparison value for water, and two others (manganese and mercury) were found in concentrations below their respective comparison values. The other detected contaminants of concern (tin) did not have a comparison value for water. Thirty-one contaminants of concern were analyzed for but not detected in the on-site surface water.

For the purposes of this public health assessment, site investigators did not collect enough samples to fully characterize on-site surface water quality. Nevertheless, site cleanup activities have eliminated the on-site ponds and ditches, and no more samples can be collected.

On-site Shallow Groundwater - Boreholes and Monitor Wells

Between 1983 and 1989, many parties sampled on-site shallow groundwater in boreholes or monitor wells to determine contaminant identity and migration. Over the years, site investigators sampled 21 on-site sample points multiple times (Figure 11, Appendix A). Four reports presented these data (EPA 1985a, 1986d; Disposal Safety 1990; Golder

Associates 1990). In our analysis, we used raw data from the Golder Associates report and summary data from the draft RI, final RI, and Disposal Safety reports.

Of the four reports, only the remedial investigation presented background sample data information. This report identified one monitor well as a background sample point, but did not identify which private wells were considered background sample points (EPA 1986d). For our analysis, we did not consider the selected monitor well an adequate background sample because it was less than ¼ mile from the landfill boundary and within the area we judged likely to be affected by the site. We could not evaluate the adequacy of the referenced background private well sample points because we did not know their locations. In order to examine background shallow groundwater quality, we selected 11 private wells within a one-mile radius around the site and used their data to assess background conditions. We had well depth information for 2 of the 11 wells, confirming they were in the shallow aquifer. We assumed the other 9 wells to also be in the shallow aquifer because this is common for private wells in the area (FDER 1983a, 1983c). The 11 background wells were located at homes southeast of the site (Hilma, Worthington, and Shindler Roads), south of the site (Brett Forest Drive and Brett Forest Court), west of the site (Old Middleburg Road), and north of the site across Mile Branch Creek (Marlee and Shindler Roads). FHRS sampled these private wells between 1990 and 1993. We considered these background sample results to also apply to earlier sample results because of the slow movement of groundwater in the area. The analytical results showed both lead and manganese in the background shallow groundwater. Only one of the eleven background wells had detectable levels of lead. Without more information about this well, we cannot determine if this result represents background contamination or is an artifact of well construction or water storage in this particular well before sampling. All background wells had detectable levels of manganese in low concentrations, indicating this metal naturally exists in the shallow groundwater around the site. One background well, the same well having detectable lead, had a manganese concentration above the comparison value.

Twenty-five contaminants of concern were detected in on-site shallow groundwater (Table 7, Appendix B). In comparing sample data to background data, the maximum values for both lead and manganese were significantly higher than background concentrations; 19 other contaminants (arsenic, barium, benzene, cadmium, chlorobenzene, chloroform, chromium, 1,4-dichlorobenzene, 1,1-dichloroethane, 1,2-dichloroethane, di(2-ethylhexyl)phthalate, mercury, methylene chloride, naphthalene, nickel, n-nitrosodiphenylamine, PCBs, selenium, and vinyl chloride) were also above background levels. Four contaminants (beryllium, cobalt, cresol, and cyanide) did not have background data for comparison.

Of the 25 contaminants of concern detected, 14 (arsenic, barium, benzene, beryllium, chlorobenzene, cyanide, 1,2-dichloroethane, di(2-ethylhexyl)phthalate, manganese, mercury, n-nitrosodiphenylamine, PCBs, selenium, and vinyl chloride) had maximum concentrations above their respective comparison values for water. Six more chemicals (cadmium, chromium, 1,4-dichlorobenzene, 1,1-dichloroethane, lead, and nickel) are known or suspected cancer-causing agents. One monitor well contained chloroform in trace amounts.

The study presenting this datum did not report the detection limit for chloroform (EPA 1985a); without this information, we cannot determine if chloroform exceeds the 6 µg/l (micrograms per liter) comparison value. Two contaminants of concern (methylene chloride and naphthalene) were found in concentrations below their comparison values for this media. The remaining two detected contaminants of concern (cobalt and cresol) did not have comparison values for water. Ten contaminants of concern were analyzed for but not detected in the on-site shallow groundwater.

For the purposes of this public health assessment, it is equivocal if the sample results adequately characterize on-site shallow groundwater quality. In some cases, such as for arsenic and benzene, there were enough analyses to characterize on-site shallow groundwater contamination by these substances. In other cases, such as for 1,2-diphenylhydrazine and hexachloroethane, there were too few analyses to fully characterize on-site shallow groundwater contamination by these compounds. Nevertheless, EPA should continue monitoring on-site groundwater to ensure changes in the composition or migration of the contaminant plume are discovered as quickly as possible.

On-site Shallow Groundwater - Private Wells

Between 1981 and 1985, FHRS and EPA sampled six on-site private wells to determine the extent of groundwater contamination and the threat to public health. In 1988, CompuChem sampled these private wells. In our analysis, we used raw data from FHRS and CompuChem (FHRS 1981b, 1983c; CompuChem 1988), and summary data from the RI and Disposal Safety reports (EPA 1986D; Disposal Safety 1990). We used data from 11 off-site private wells as background information for our analysis.

Ten contaminants of concern were detected in on-site private wells (Table 8, Appendix B). In comparing sample data to background data, the maximum value for lead was significantly higher than its background concentration. The maximum concentration for manganese was within background range. Seven other detected contaminants (arsenic, barium, cadmium, chloroform, mercury, methylene chloride, and nickel) were also above background levels, and one contaminant (tin) did not have background data for comparison.

Of the ten contaminants of concern detected, two (arsenic and methylene chloride) was found in concentrations above their respective comparison values for water, and three others (cadmium, lead, and nickel) are suspected cancer-causing agents. Manganese, a naturally occurring element in the area, was found in concentrations below its comparison value, as were barium, chloroform, and mercury. The other detected contaminant of concern (tin) did not have a comparison value for water. Sixteen contaminants of concern were not detected, and the remaining 9 contaminants of concern were not analyzed for in the on-site private wells.

For the purposes of this public health assessment, investigators did not collect enough samples to fully characterize on-site private well water quality. Nevertheless, site cleanup

activities have eliminated the on-site homes and their private wells, and no more samples can be collected.

On-site Air

There is no record of air samples being collected on site prior to August 1993. Although EPA collected air samples during the 1985 RI fieldwork, these were general monitoring measurements from an HNu meter, an OVA meter, an explosimeter, and a dust monitor (EPA 1986d). These instruments yield general quantitative results applicable to monitoring site safety conditions; they do not yield the qualitative and precise quantitative data needed for a public health assessment. Therefore, we did not use these data to evaluate environmental contamination at the site. The lack of air samples from the past is a significant data gap for this public health assessment because there were homes on site, and nearby residents played or scavenged in the landfill. Without past sample data, we cannot fully evaluate the potential health effects from the resident's exposure to on-site air.

Presently, there is an air stripper on site to remove solvents from the groundwater. In August and September 1993, Golder Associates performed a trial run to evaluate the stripper's performance, and found the stripper was removing solvents from the groundwater and expelling them into the on-site air (Golder Associates 1993a). Because site access is restricted, nearby residents are not likely to be exposed to on-site air contaminants. We evaluate the movement of this contaminated air off site and the need for additional samples in our discussion of off-site air contamination.

On-site Biota

There is no record of biotic samples being collected on site. The lack of biotic samples is a significant data gap for this public health assessment because area residents ate vegetables from their gardens and fish from on-site ponds. Without sample data, we cannot evaluate the potential health effects from the resident's exposure to on-site biota. Nevertheless, there is no need to collect on-site biotic samples in the future because the these food sources no longer exist.

B. Off-site Contamination

For the purposes of this evaluation, we defined "off-site" as the area outside the fenced boundaries of the Hipps Road Landfill but within the area we consider likely to be affected by the site. We defined the affected area as: the area within approximately ¼ mile of the landfill's perimeter, the area directly north of the site to Mile Branch Creek and northeast of the site to Shindler Road, and the area southwest of the site along Exline Road (Figure 12, Appendix A). In considering private well data from this area, we used data from homes on both sides of a street, even if only one side of the street was within the ¼-mile boundary. Our definition of the affected area included groundwater contamination likely to result from the known contaminant plume northeast of the site, the mounding of water when the landfill

was uncapped, and possible groundwater flow southwest from the site. Our definition encompassed an area larger than the federal cleanup area. By incorporating areas around the site where groundwater flow is likely as well as where it is known, we were better assured of identifying the maximum values of potentially site-related chemicals for use in our analysis.

We compiled data in this subsection from the following sources: FDER samples (FDER 1983f); BESD samples (BESD 1983a); Duval CPHU samples (FHRS 1981b, 1983c, 1984, 1990, 1991, 1992, 1993d); EPA's 1984 site screening study, 1985 draft RI/FS work plan, and 1985 RI field work (EPA 1985a, 1985c, 1986d); Disposal Safety's groundwater contamination assessment (Disposal Safety 1990); and Golder Associates' groundwater recovery system design report, baseline groundwater sampling study, and air stripper report, (Golder Associates 1990, 1992, 1993). We reviewed several other reports containing off-site sample data results (EPA 1985b, 1986a, 1986b, 1986c; ATSDR 1986) but determined these data had already been presented in and counted from earlier reports.

In counting the number of analyses for a contaminant, we used raw data whenever these data were available to us. In some cases, such as in our review of the remedial investigation data, we could only count the results presented in the report's summary data tables because the raw data were not available from EPA or other governmental agencies. Therefore, we acknowledge the total number of samples for some contaminants is likely greater than the number we show in our data tables, and some contaminants may have been analyzed for that aren't identified in the summary reports we examined. In several reports, sample locations were not precisely identified. When an incomplete sample point description contained enough information to judge it to be within the affected area, we included the sample in our off-site analysis; when a sample description did not contain enough information to judge its approximate location, we excluded the sample point from our analysis. Finally, when we were able to identify duplicate samples among the many reports we reviewed, we counted these samples only once.

In comparing soil and water data values on and off site, we noticed some contaminants of concern were found in higher concentrations off site than on site for the same media. Similarly, the RI asserted cadmium, lead, and chloroform were not site-related for various reasons (EPA 1986d). Because on-site contamination was insufficiently characterized, we could not determine if these irregularities represented contamination from other sources or were statistical artifacts of the unequal sampling among these locations.

Off-site Surface Soil (0-3 inches deep)

There is no record of surface soil samples (0-3 inches deep) being collected off site. The lack of surface soil samples is a significant data gap for this public health assessment because there are homes off site, and family members continue to play or garden in off-site surface soils. Without sample data, we cannot evaluate the potential health effects from the resident's exposure to off-site surface soils. Therefore, we recommend EPA collect one surface soil sample (0-3 inches deep) from the part of each private yard, bordering the

southern eastern, northern, and western site boundaries, that is most likely to have received surface soils blown off site. We recommend these soils be analyzed for inorganics, pesticides, base neutrals, and acid extractables including: arsenic, barium, beryllium, cadmium, chromium, cobalt, cresol, DDT, di(2-ethylhexyl)phthalate, 1,2-diphenylhydrazine, hexachloroethane, lead, manganese, mercury, naphthalene, nickel, n-nitrosodiphenylamine, PCBs, selenium, and tin.

Off-site Subsurface Soil (deeper than 3 inches)

In 1985, EPA collected off-site subsurface soil samples at seven locations around the landfill to determine the nature and extent of off-site subsurface soil contamination. EPA collected these samples from the two water-bearing units of the surficial aquifer while drilling off-site temporary monitor wells. EPA took seven subsurface soil samples from the Sand aquifer at depths ranging from 29 - 60.5 feet, and five subsurface soil samples from the Limestone aquifer at depths ranging from 69 - 130 feet (EPA 1986d). Although site investigators collected subsurface soil samples from areas most likely to be contaminated by groundwater (deep in the Sand aquifer and in the top of the Limestone aquifer), residents are not likely to be exposed to these soils. Consequently, subsurface soils that residents might come in contact with through gardening or digging activities are uncharacterized.

For the purposes of this public health assessment, site investigators did not collect enough samples or perform all of the analyses needed to adequately characterize off-site subsurface soil quality that residents were exposed to. Nevertheless, we do not expect solvents which might be transported to these soils to remain because of the high volatility and water solubility of these compounds. Likewise, we do not expect substances that adsorb to soils or have a low water solubility to have a transport mechanism allowing them to permeate subsurface soils below a couple of inches. Therefore, we do not recommend EPA collect off-site subsurface soil samples.

Off-site Sediment

Between 1983 and 1985, BESD and EPA collected off-site sediment samples in at least four pond, ditch, or creek locations (Figure 13, Appendix A). In 1983, BESD sampled one pond, presumed to be off-site, at the request of a private citizen. The BESD data did not include a map of the sample location, and we could not determine where the sampled pond was located. (This sample location is not included in Figure 13, Appendix A.) BESD did not identify the collected sample as grab or core (BESD 1983a). In 1985, EPA collected grab samples from a pond 300 feet east of the site, another pond 1,900 feet north of the site, a creek (Mile Branch Creek) 4,000 feet northeast of the site, and a storm water ditch 1,000 feet south of the site. EPA used these samples to determine the nature and extent of contamination off site. EPA also collected a sediment sample from an intermittent stream 10,000 feet south of the site for background information (EPA 1986d), but we did not use these data in our analysis because they did not represent background conditions. In our analysis, we used raw data from BESD and summary data from the RI.

Five contaminants of concern were detected in off-site sediments (Table 9, Appendix B). One of these (PCBs) was found in a concentration above its comparison value for soil, and two others (chromium and lead) are known or suspected cancer-causing agents. One contaminant of concern (barium) was found in a concentration below its comparison value. The other detected contaminant of concern (cresol) did not have a comparison value for sediment. Four contaminants of concern were not detected, and the remaining 26 contaminants of concern were not analyzed for in the off-site sediments.

For the purposes of this public health assessment, site investigators did not collect enough samples or perform all of the analyses needed to adequately characterize off-site sediment quality. Without adequate sample data, we cannot fully evaluate the potential health effects from the resident's exposure to off-site sediment. Therefore, we recommend EPA collect one sediment sample every 150 feet for the first 500 feet of every storm water drainage system leaving the site. These systems include: the storm water swales along the sites northern and western borders, the cypress pond along the site's eastern boundary, and any other storm water conduits leaving the site. In addition, we recommend collection of two sediment samples from the ground depression immediately northeast of the intersection of Hipps and Bunion Roads. We recommend these sediments be analyzed for inorganics, pesticides, base neutrals, and acid extractables including: arsenic, barium, beryllium, cadmium, chromium, cobalt, cresol, DDT, di(2-ethylhexyl)phthalate, 1,2-diphenylhydrazine, hexachloroethane, lead, manganese, mercury, naphthalene, nickel, n-nitrosodiphenylamine, PCBs, selenium, and tin.

Off-site Surface Water

Between 1983 and 1985, BESD and EPA collected off-site surface water samples in at least four pond, ditch, or creek locations (Figure 14, Appendix A). In 1983, BESD sampled one pond, presumed to be off-site, at the request of a private citizen. The BESD data did not include a map of the sample location, and we could not determine which off-site pond was sampled (BESD 1983a). (This sample location is not included in Figure 14, Appendix A.) In 1985, EPA collected surface water samples from a pond 300 feet east of the site, another pond 1,900 feet north of the site, a creek (Mile Branch Creek) 4,000 feet northeast of the site, and a storm water ditch 1,000 feet south of the site. EPA used these samples to determine the nature and extent of contamination off site. EPA also collected a surface water sample from an intermittent stream 10,000 feet south of the site for background information (EPA 1986d), but we did not use these data in our analysis because they did not represent background conditions. In our analysis, we used raw data from BESD and summary data from the RI.

Five contaminants of concern were detected in off-site surface waters (Table 10, Appendix B). One of these (PCBs) was found in a concentration above its comparison value for water, and two others (chromium and lead) are known or suspected cancer-causing agents. One contaminant of concern (manganese) was found in a concentration below its comparison value. The other detected contaminant of concern (cobalt) did not have a comparison value

for water. Four contaminants of concern were not detected, and the remaining 26 contaminants of concern were not analyzed for in the off-site surface waters.

For the purposes of this public health assessment, site investigators did not collect enough samples or perform all of the analyses needed to adequately characterize off-site surface water quality. Without adequate sample data, we cannot evaluate the potential health effects from the resident's exposure to off-site surface waters. Presently, there are two issues concerning storm water runoff. First, contaminants may be entering the cypress pond east of the site through groundwater recharge from the site. Neighborhood children play in the storm water ditches draining this pond. We recommend 1 surface water sample be collected from the cypress pond to determine if solvents (VOCs) are present in this water body. pH should be measured on all surface water or groundwater samples collected, and additional analyses for metals should be conducted if the pH is low. Second, residents also report the retention ponds for the air stripper have overflowed several times with runoff entering the residential yards east of the site (Hipps Road residents, pers. comm.). For example, after heavy rains in October 1992, the retention ponds reportedly overflowed and flooded the off-site cypress pond. One resident, living on property east of and contiguous to the site, noticed oily-looking fluids overflowing from the landfill onto his property (Norman 1994). The dirt bulging in the storm water control structures and the gaps in the plastic sheeting along the site's eastern boundary, seen during our May 23 and November 18, 1994 site visits, suggest retention pond overflow and storm water runoff may still be a problem at the site. In addition, site overflow going into the cypress pond east of the site subsequently flows into to storm water ditches that children play in. To determine if contaminants are present in storm water leaving the site, we recommend flood water be sampled in the cypress pond and in the site's perimeter ditches at 50 foot intervals within 12 hours of the next reported flooding event. In the cypress pond, at least one sample should be collected at the surface to capture oils or other substances less dense than water, and at least one other sample should be collected from below the pond's surface. Flood water samples should be measured for pH and analyzed for inorganics, pesticides, purgeables, base neutrals, acid extractables, and any contaminant of concern not covered by this list. Additional analyses for metals should be conducted if the pH is low. Furthermore, if the landfill's cap is somehow breached in the future, then a comprehensive study of the landfill's off-site surface waters will need to be conducted.

Off-site Shallow Groundwater - Monitor Wells

Between 1983 and 1993, many parties sampled off-site shallow groundwater in monitor wells to determine contaminant identity and migration, and to identify the contaminant plume boundaries for cleanup. Over the years, site investigators sampled 35 off-site sample locations multiple times at varying depths (Figure 15, Appendix A). Several reports and individual sample analyses contained these data (Disposal Safety 1990; EPA 1985a, 1986d; FDER 1983f; Golder Associates 1990, 1992, 1993a). In our analysis, we used raw data from three sources (FDER 1983f; Golder Associates 1990, 1993a) and summary data from

the other four. We used data from 11 off-site private wells as background information for our analysis.

Twenty-seven contaminants of concern were detected in off-site shallow groundwater (Table 11, Appendix B). In comparing sample data to background data, the maximum values for both lead and manganese were significantly higher than background concentrations. Twenty-two other detected contaminants (arsenic, barium, benzene, cadmium, chlorobenzene, chlorodibromomethane, chloroform, chromium, 1,4-dichlorobenzene, 1,1-dichloroethane, 1,2-dichloroethane, di(2-ethylhexyl)phthalate, mercury, methylene chloride, naphthalene, nickel, n-nitrosodiphenylamine, selenium, 1,1,2,2-tetrachloroethane, tetrachloroethene, trichloroethene, and vinyl chloride) were also above background levels. The three remaining detected contaminants (beryllium, cobalt, and cresol) did not have background values for comparison.

Of the 27 contaminants of concern detected, 16 (arsenic, benzene, beryllium, cadmium, chlorodibromomethane, chloroform, 1,2-dichloroethane, di(2-ethylhexyl)phthalate, manganese, methylene chloride, naphthalene, n-nitrosodiphenylamine, 1,1,2,2-tetrachloroethane, tetrachloroethene, trichloroethene, and vinyl chloride) had maximum concentrations above their respective comparison values for water. Five more chemicals (chromium, 1,4-dichlorobenzene, 1,1-dichloroethane, lead, and nickel) are known or suspected cancer-causing agents. Four contaminants of concern (barium, chlorobenzene, mercury, and selenium) were found in concentrations below their respective comparison values. The other two detected contaminants of concern (cobalt and cresol) did not have comparison values for water. The remaining eight contaminants of concern were not detected in the off-site shallow groundwater.

In 1991, Disposal Safety tested the water at one monitor well in the vicinity of the groundwater contaminant plume for the presence of the prescription drug components pentobarbital, meprobamate, and phensuxamide. Pentobarbital was detected in the one groundwater sample taken at a concentration of 1 μg/l. Attempts to develop methods to extract the other two compounds in sufficient quantities failed (Disposal Safety 1991). These results suggest medical wastes may have been disposed of in the landfill. If medical wastes were disposed of at the site, low-level radioactive wastes may also be present in the fill material.

For the purposes of this public health assessment, sample results adequately characterize off-site shallow groundwater quality in the vicinity of the plume northeast of the site, with the exception of radionuclides. EPA should continue monitoring off-site groundwater in this area to ensure changes in the composition or migration of the contaminant plume are discovered as quickly as possible. In addition, EPA has not fully investigated groundwater movement in directions other than to the northeast of the landfill nor the possibility of off-site plumes in these directions. In fact, the remedial investigation's groundwater contaminant transport analysis diagrams show a groundwater flow gradient east and southeast of the site, in addition to the somewhat steeper gradient northeast (EPA 1986d). Moreover, groundwater

movement in the area west of the site has been in question since FDER's initial investigation in 1983 (FDER 1983c), and remained undefined after the RI's groundwater contaminant transport analysis (EPA 1986d). Therefore, EPA should initially investigate groundwater movement within ½-mile around the eastern, southern, and western site boundaries and delineate the extent of groundwater contamination in this area. If contaminant plumes are found in these directions, the scope of this groundwater investigation will likely need expansion. All ground water analyses should include metals, other inorganics, purgeables, base neutrals, acid extractables, and radionuclides.

Off-site Shallow Groundwater - Private Wells

Between 1981 and 1993, various parties sampled approximately 90 off-site private wells to determine the extent of groundwater contamination and the threat to public health. In our analysis, we used raw data from FHRS (FHRS 1981b, 1983c, 1984, 1990, 1991, 1992, 1993d) and EPA's site screening study (EPA 1985c), and summary data from the RI and Disposal Safety reports (Disposal Safety 1990; EPA 1986d). For background information, we used data from 11 off-site private wells outside of the area of concern.

Twenty contaminants of concern were detected in off-site private wells (Table 12, Appendix B). In comparing sample data to background data, the maximum values for both lead and manganese were significantly higher than background concentrations. Seventeen other detected contaminants (barium, benzene, bromodichloromethane, chlorodibromomethane, chloroform, 1,4-dichlorobenzene, 1,1-dichloroethane, 1,2-dichloroethane, 1,2-dichloropropane, di(2-ethylhexyl)phthalate, hexachloroethane, mercury, methylene chloride, nickel, tetrachloroethene, trichloroethene, and vinyl chloride) were also above background levels. The final detected contaminant (cresol) did not have a background value for comparison.

Of the 20 contaminants of concern detected, 12 (benzene, bromodichloromethane, chlorodibromomethane, chloroform, 1,2-dichloroethane, di(2-ethylhexyl)phthalate, hexachloroethane, manganese, methylene chloride, tetrachloroethene, trichloroethene, and vinyl chloride) had maximum concentrations above their respective comparison values for water. Five more chemicals (1,4-dichlorobenzene, 1,1-dichloroethane, 1,2-dichloropropane, lead, and nickel) are suspected cancer-causing agents. Two contaminants of concern (barium and mercury) were found in concentrations below their respective comparison values. The other detected contaminant of concern (cresol) did not have a comparison value for water. Fourteen contaminants of concern were not detected in the off-site private wells, and one contaminant of concern was not analyzed for in any private well samples.

An examination of private well sampling frequencies shows very few private wells were sampled between 1981-1985, even fewer were sampled between 1986-1989, and many were sampled between 1990-1993. Considering that drinking water complaints reportedly began as early as 1972 (Keneagy 1991), there was a time period of approximately 18 years during which private well water quality was virtually uncharacterized. Many of the highest contaminant concentrations were measured during the 1983-1985 time period. We cannot

know if these values were the peak exposure concentrations. Therefore, for the purposes of this public health assessment, investigators did not collect enough samples to adequately characterize private well water quality prior to 1990. However, investigators collected enough samples to characterize off-site private well water quality from 1990-1993 for most contaminants, with the notable exceptions of beryllium, cresol, cyanide, DDT, and tin. Nevertheless, FHRS should continue analyzing private well samples near the site to ensure any new wells contaminated by plume migration are discovered as quickly as possible. Radionuclides should be added to the list of contaminants currently analyzed for. If further groundwater studies show contamination in other areas around the landfill, private wells in these areas will need to be sampled. Concerned citizens close to known or suspected groundwater contamination areas should limit their exposure by connecting their homes to the public water supply where it is available, using public water for all home uses including car washing and irrigation, and properly plugging their private wells so that they are not available for future use by any party.

In addition to the private well sampling described above, FHRS also sampled private wells outside of the area of concern, in the area we called "off-site". We reviewed approximately 100 sample results collected between 1990-1993, and located within a 1-mile radius of the landfill boundaries. Between 1992-1993, there were nine isolated spots of mostly low-level groundwater contamination. Contaminants found in these locations were: "other volatiles" at two homes east of and about ½ mile from the site (Hipps Road); chloroform, p-cymene, di-n-butylphthalate, lead, mercury, styrene, or 1,2,4-trimethylbenzene at four homes east-southeast of and about ½ mile from the site Shindler Road); chloroform, bromodichloromethane, and chlorodibromomethane at one home southeast of and about 1 mile from the site (Walden Road); di(2-ethylhexyl)phthalate or lead at three homes south-southeast of and about ½ mile from the site (Bunion Drive); lead at one home south of and about ½ mile from the site (Taylorfield Road); lead or "other volatiles" at three homes south of and about 1 mile from the site (Brett Forest Drive and Brett Forest Court); barium, benzene, 1,1-dichloroethane, 1,1-dichloroethene, xylenes, or heptachlor at three homes southwest of and about ½ mile from the site (Sun Lane); ethylbenzene and "other volatiles" at one home west of and about ½ mile from the site (Hipps Road); and chromium or lead at two homes northwest of and about ½ mile from the site (Loves Drive). Heptachlor and styrene were new contaminants; heptachlor was found in concentrations below its comparison value (CREG), but styrene, a suspected cancer-causing agent, did not have a CREG value for comparison. Nearly all of the other contaminants were found in concentrations smaller than the maximum values already described in this assessment; however, one home had a new maximum value for lead, and another home had new maximum values for chloroform, bromodichloromethane, and chlorodibromomethane.

Nearby residents are concerned these latter results indicate the landfill's groundwater contaminant plume is much larger than currently believed. They contend the apparent random distribution of the contaminants simply could reflect the randomness of the private well sampling in the area. This is one possible explanation. Alternatively, because these contaminants are found in many household products (such as cosmetics, cleaners, paints,

varnishes, paint and varnish removers, plumbing products, and pesticides), the contamination could result from residents' washing product residues into household septic systems or dumping unwanted products on the ground. We do not have enough information to determine which of these possibilities is most likely. Nevertheless, these results support our recommendation that a more comprehensive examination of groundwater flow and contamination is needed in the eastern, southern and western directions from the site.

Finally, the shallow depths and slow movement of Mile Branch Creek, observed during our May 1994 visit, suggest groundwater contaminants not captured by the air stripper treatment system might flow under this creek rather than be discharged into it, as suggested by the RI (EPA 1986d). Therefore, we recommend Duval CPHU periodically test the private wells of homes north of the creek for contaminants of concern and radionuclides.

Off-site Air

There is no record of air samples being collected off site. Although EPA collected air samples during the 1985 RI fieldwork, these were general monitoring measurements from an HNu meter, an OVA meter, an explosimeter, and a dust monitor (EPA 1986d). These instruments yield general quantitative results applicable to monitoring site safety conditions; they do not yield the qualitative and precise quantitative data needed for a public health assessment. Therefore, we did not use these data to evaluate environmental contamination off site. The lack of air samples from the past is a significant data gap for this public health assessment because of the residents' potential exposure to airborne contaminants in and around their homes. To estimate past exposure to volatile contaminants, we used the computer software Risk*Assistant™ (1993) to predict airborne concentrations of solvents volatilized from groundwater, based on their maximum concentrations in groundwater.

To estimate present and future exposure to airborne contaminants from the air stripper, we used a different model to predict off-site solvent concentrations from on-site air measurements taken during the air stripper's 1993 trial run and performance test. During this trial run, Golder sampled on-site air at three locations to ensure the newly installed air stripper was working as predicted and was not posing a public health threat (Figure 16, Appendix A). The sample points were at the top of the air stripper tower, 1,000 feet northeast of the stripper tower along the eastern fenced boundary, and 1,300 feet northwest of the stripper tower along the northern fenced boundary. For the first five days of the air stripper's operation, Golder collected 24-hour composite samples at all three sample locations. For the last 16 days of the trial run, Golder collected 24-hour composite samples only at the top of the air-stripper tower (Golder Associates 1993a). For our analysis, we used raw data from the trial run.

Prior to air stripper operation, Golder colleeted two composite samples (an eight hour and a twenty-four hour) at the northernmost sample location along Hipps Road. In addition, we consider the August 26 air samples taken from the two locations north of the tower to be background because the wind was predominately from the northeast on that day and blew the

stripper emissions away from these sample points. The analytical results showed methylene chloride in the background air. This methylene chloride could be coming from the landfill itself or from solvent uses in the surrounding area.

Because it is unrealistic to assume nearby residents will breathe air in concentrations found at the top of the air stripper, we examined the data from the closest sample point. However, this sample point was 1,000 feet northeast of the tower, and the closest resident lives approximately 300 feet east of the tower. To predict air concentrations for this resident, we contacted the Florida Department of Environmental Protection's (formerly known as FDER) Air Modeling and Assessment Section in Tallahassee. They used a model called "Screen 2" to predict the dilution with distance from the air stripper (EPA 1992a). This model predicted the highest concentrations likely at various distances assuming worst case weather conditions of a gentle breeze with little dispersion. To check the accuracy of the model, we compared the predicted dilution to the actual concentrations measured at the two northernmost sample points on August 25, a day when the wind was predominately from the south. In general, the dilution predicted by the model is consistent with actual measured concentrations; therefore, we used the predicted concentrations at 300 feet for our analysis.

Nine contaminants of concern were detected at the top of the tower and are predicted to be present in off-site air (Table 13, Appendix B) at the closest residence. One of these (1,2-dichloroethane) is likely to be found in a concentration above its comparison value for air. Two more detected contaminants of concern (1,1-dichloroethane and vinyl chloride) are known or suspected cancer-causing agents. Four contaminants of concern (benzene, methylene chloride, tetrachloroethene, and trichloroethene) are likely to be in concentrations below their respective comparison values. The remaining two detected contaminants of concern (chlorobenzene and 1,2-dichloropropane) did not have comparison values for air. Four contaminants of concern were not detected at the top of the tower, and the remaining 22 contaminants of concern were not analyzed for in the on-site air.

For the purposes of this public health assessment, investigators collected enough samples to fully characterize on-site air quality resulting from the air stripper. Because the air stripper is an on-going pollution source in a residential community, we recommend EPA collect and analyze the water influent to the air stripper at least monthly for the first three months of operation, and at least every three months for the duration of operation. These samples should be analyzed for all of the volatile organic compounds already detected in the groundwater at this site, as well all of the volatile organic compounds already detected in the air from the air stripper. If any influent sample result exceeds the maximum detected concentration measured during the air stripper's trial run, we recommend re-evaluation of the influent data to determine if adverse health effects are likely.

Nearby residents are concerned metals in the groundwater may be emitted as aerosols from the air stripper. Golder reports they have placed a demister screen on the top of the air stripper to intercept any aerosols formed during the air-stripping process. Droplets formed

on the demister drop back into the air stripper and are not released into the air (FHRS 1994b).

Off-site Biota

There is no record of biotic samples being collected off site. The lack of biotic samples is a significant data gap for this public health assessment because area residents ate vegetables from their gardens. Presently, there is no need to collect off-site biotic samples because the off-site contaminants presently detected in groundwater and air are not expected to significantly accumulate in plants. However, if the off-site samples we have recommended above reveal the presence of contaminants that bioaccumulate, we may request biotic sampling.

C. Quality Assurance and Quality Control

We requested a data review summary from EPA, but were told one does not exist for this site (Patsy Goldberg, pers. comm.). Although the remedial investigation contained a quality assurance/quality control (QA/QC) assessment asserting the RI data could be used with confidence (EPA 1986d), Disposal Safety's review of the solvent analyses found excessive holding times (exceeded by 70-110 days), suggesting that concentrations for these compounds may be underestimated (Disposal Safety 1990). Because we do not have the raw data and sample sheets to review, we cannot evaluate this assertion. Nevertheless, we used these data in our analyses because they are the only data we have for many contaminants measured during this time period. The 1988 CompuChem QA data showed methylene chloride was present in the blank. Because the methylene chloride concentration in the blank was close to detected well sample concentrations, we did not consider this compound to be detected in wells of this sample set. The quality assurance data we reviewed from the Golder reports (Golder Associates 1990, 1993a) indicated those data were reliable. We did not have QA/QC data for Golder's baseline groundwater sampling study (Golder Associates 1992), but we assumed these data were valid because the results were cousistent with other data we had about the detected contaminants. Most of the FHRS private well data had information on trip blanks accompanying them, indicating these data were also reliable (FHRS 1981b, 1983c, 1984, 1990, 1991, 1992, 1993d). In cases where we did not have QA/QC information, we assumed these data were valid, since the environmental samples were collected and analyzed by governmental agencies or their contractors. In preparing this public health assessment, we relied on the information provided by these agencies or contractors and assumed that site investigators followed adequate quality assurance and quality control measures followed in regard to chain-of-custody, laboratory procedures, and data reporting, with the noted question about the RI data. The validity of the analysis and conclusions drawn for this public health assessment are determined by the completeness and reliability of the referenced information.

In each of the preceding On- and Off-site Contamination subsections, we evaluated the adequacy of the data to estimate exposures. We assumed that estimated data (J) and

presumptive data (N) were valid. This second assumption errs on the side of public health by assuming that a contaminant exists when actually it may not exist.

D. Physical and Other Hazards

In 1988, Golder conducted a methane gas survey at the site to assess the fire/explosion hazard at the site and to determine the need for a gas venting system in the landfill's cap. The survey did not find any methane being generated at the site. Golder Associates concluded if methane were to be generated by the site in the future, the gas could escape through the soil cover; therefore, it would not accumulate under the cover or move laterally underground (Golder Associates 1989a). Consequently, we expect the fire/explosion hazard from methane gas at the site to be negligible.

During our site visits, drowning seemed to be a potential physical hazard if someone were to accidentally fall into the retention ponds. The ponds are about 4½ feet deep, and the slope of the sides is 2:1 (Golder Associates 1989b) which should enable adults and older children to easily climb out. However, children less than five feet tall might drown if the pond were full. The potential for this problem currently exists, since the site's fence on the western boundary (Exline Road) has a large hole through it, enabling trespassers to enter the site.

PATHWAYS ANALYSES

To determine if nearby residents are exposed to contaminants migrating from the site, we evaluated the environmental and human components of exposure pathways. Exposure pathways consist of five elements: a source of contamination, transport through an environmental medium, a point of exposure, a route of human exposure, and an exposed population.

An exposure pathway can be eliminated if at least one of the five elements is missing and will never be present. We categorize exposure pathways that are not eliminated as either completed or potential. For completed pathways, all five elements exist and exposure to a contaminant has occurred, is occurring, or will occur. For potential pathways, at least one of the five elements is missing, but could exist. For potential pathways, exposure to a contaminant could have occurred, could be occurring, or could occur in the future.

In this analysis, "on-site" is defined as the area within the fenced boundaries of tbe Hipps Road Landfill (Figure 7, Appendix A), and "off-site" is defined as the area outside of the fenced boundaries and within the area of we judged likely to be affected by the site (Figure 12, Appendix A). Much of the exposure information comes from interviews with nearby residents (FHRS 1993b, 1993c; Hipps Road residents, pers. comm.).

A. Completed Exposure Pathways

For a summary of the completed exposure pathways at this site, refer to Table 14, Appendix B.

Subsurface Soil Pathway

In the past, adult residents had contact with on-site subsurface soils (that is, soils more than three inches deep) as they scavenged materials out of the landfill. Neighborhood children had contact with on-site subsurface soils as they scavenged materials from and dug forts into the fill material while playing at the site. Exposure to subsurface soil contaminants occurred via skin absorption and incidental ingestion. Since the landfill is now capped and site access is restricted, nearby residents are not likely to be exposed to on-site subsurface soil contaminants in the present or future.

Sediment Pathway

In the past, neighborhood children played in the sediments of dried storm water swales and on-site ponds. Exposure to sediment contaminants occurred via skin absorption and incidental ingestion. Because site access is restricted and cleanup activities eliminated the ponds and storm water swales on site, neighborhood children are not likely to be exposed to on-site sediment contaminants in the present or future. However, neighborhood children are likely to be exposed to sediment contaminants from past storm water run off in off-site swales and ditches in both the present and future.

Surface Water Pathway

In the past, neighborhood children played in storm water swales and swam in on- and off-site ponds. Nearby residents also swam in Mile Branch Creek. Exposure to surface water contaminants occurred via skin absorption and incidental ingestion. Because site access is restricted and cleanup activities eliminated the ponds and storm water swales on site, neighborhood children are not likely to be exposed to on-site surface water contaminants in the present or future.

Shallow Groundwater Pathway

Prior to 1987, on-site and off-site homes drew potable water from private wells in the contaminated Sand aquifer. Exposure to shallow groundwater contaminants occurred via ingestion, as well as skin absorption and inhalation of solvents. Because on-site homes were vacated and demolished as a part of cleanup activities, residents will not be exposed to on-site shallow groundwater in the present or future. In contrast, many off-site homes in or near the contaminant plume still use private well water. Exposure to off-site shallow groundwater contaminants will continue in the present and future for residents using private well water close to the plume.

Air (Tower Effluent) Pathway

As a part of site cleanup activities, the responsible parties have installed an air-stripping tower to remove solvents from the groundwater. Solvents will be emitted in tower effluent and are likely to be blown off-site in both the present and future. Exposure to off-site air contaminants is likely via inhalation.

B. Potential Exposure Pathways

We categorize the following exposure pathways as potential because there are no environmental data measuring contaminant types or amounts. Without these data, we cannot fully evaluate the contribution of each potential pathway to the residents' total exposure. For a summary of the potential exposure pathways at this site, refer to Table 15, Appendix B.

Surface Soil Pathway

In the past, residents living on and off site may have been exposed to surface soil contaminants (that is, 0-3 inches deep) in their yards both by playing and gardening in the soil. In addition, adults and children visiting the landfill to scavenge or to play may have had contact with landfill surface soil contaminants. Exposure to surface soil contaminants may have occurred via skin absorption, incidental ingestion, and inhalation of dust. Since the landfill is now capped and site access is restricted, nearby residents are not likely to be exposed to on-site surface soils in the present or future. However, residents may be exposed to off-site surface soil contaminants in the present and future as they continue to play and work in their yards.

Surface Water Pathway

In the present and future, neighborhood children are expected to continue to play and swim in off-site storm water ditches, potentially exposing them to site-related contaminants via skin absorption and incidental ingestion. Nearby residents may also be exposed to any contaminants entering Mile Branch Creek from the groundwater contaminant plume northeast of the site.

Air (Odor) Pathway

In the past and present, nearby residents complained about landfill odors and worried about their possible exposure to airborne contaminants. Exposure to air contaminants may have occurred via inhalation and skin absorption. Since site access is now restricted, nearby residents are not likely to be exposed to on-site air contaminants in the present or future. However, nearby residents may be exposed to off-site air contaminants in the present and future as the wind blows air off site.

Biota Pathway

In the past, neighborhood children fished in the ponds adjacent to the landfill, and ate squirrels and other small game from the site. Furthermore, nearby residents ate vegetables from their gardens. Exposure to contaminants in biota may have occurred via ingestion of plant and animal tissue. Because the on-site ponds and gardens are now gone, nearby residents will not be exposed to on-site fish or garden vegetables. However, nearby residents may be exposed to contaminants in biota in the present and future by eating vegetables or small game from the site. In addition, nearby residents may be exposed to contaminants in biota in the present and future by eating fish from Mile Branch Creek, if plume contaminants reach sediments in this water body.

C. Eliminated Pathways

We did not evaluate a pathway for deep groundwater (that is, the Floridan aquifer) because we do not know of any exposure points to this media or have any sampling data from this aquifer.

We do not believe a pathway for off-site subsurface soil exposure exists. Contaminated groundwater from the site flows mostly downward before moving horizontally (EPA 1986d). Contaminants have been found in the Sand and Limestone aquifers at depths unlikely to be accessed by residents. Residents are concerned local flooding will bring these contaminants to the shallow soils where they can be exposed through gardening or other digging activities. Yet, when flooding occurs, we do not expect any solvents to remain in these soils because of the high volatility and water solubility of these compounds. Likewise, we do not expect substances that adsorb to soils or have a low water solubility to have a transport mechanism allowing them to permeate subsurface soils at shallow depths. Similarly, we expect any contaminants attached to on-site soil particles and later blown off site to remain within the surface soil layer.

PUBLIC HEALTH IMPLICATIONS

In this section, we discuss the risk of illness and possible health effects for persons exposed to specific contaminants, evaluate state and local health databases, and address specific community health concerns.

Risk of Illness

In this health assessment, the risk of illness is the chance that exposure to a hazardous contaminant is associated with a harmful health effect or illness. The risk of illness is not a measure of cause and effect; only an in-depth health study may identify a cause and effect relationship. Instead, we use the risk of illness to indicate whether or not a follow-up health

study is needed, and to provide possible associations to be addressed in a follow-up health study if the study is needed.

In general, the greater the exposure to a hazardous contaminant, the greater the risk of illness. However, the risk of illness is also determined by the amount of a substance that is required to harm a person's health. In theory, everyone who is exposed to a hazardous contaminant above a minimum level has an increased risk of illness, but only in unusual circumstances do many people actually become ill. Individual risks of illness usually are measured and reported as an expression of chance. Consequently, scientists discuss the likelihood of becoming ill, and may express the chance of becoming ill as a fraction. For example, in the 1930's and 1940's, some workers exposed to very high levels of asbestos in asbestos factories had an estimated cancer risk of one chance in one hundred (1/100). However, the estimated cancer risk from exposure to the lower levels of asbestos in air outside of these plants was one chance in ten thousand (1 in 10,000). Sometimes, scientists compare the severity of different risks by looking at the expected occurrences of an illness for the total exposed population. For example, in 100,000 workers exposed to high levels of asbestos in the 1930's and 1940's, scientists would expect to see 1,000 (= 100,000 x 1/100) extra cancer cases. If 100,000 people were exposed only to the low levels of asbestos, scientists would expect to see 10 (= 100,000 x 1/10,000) extra cases of cancer (EPA 1990b).

Information from human studies provides the strongest evidence that exposure to a hazardous contaminant is related to a particular illness. Some of this evidence comes from doctors reporting unusual incidences of a specific illness in exposed individuals. More formal studies compare illnesses in people with different levels of exposure. However, human information is very limited for most hazardous contaminants, and scientists frequently must depend upon data from animal studies. Animal studies are used to estimate risk of illness in humans because hazardous contaminants that are associated with harmful health effects in humans often also are associated with harmful health effects in other animal species. There are limits to relying only on animal studies, however. For example, scientists have found some hazardous contaminants are associated with cancer in mammals, but lack evidence of a similar association in humans. In addition, human and animals have differing abilities to protect themselves against low levels of contaminants. Furthermore, most animal studies test the possible health effects of high exposure levels only. Consequently, the possible effects of a hazardous contaminant on humans is uncertain when there is information only from animal experiments (EPA 1990b).

Dose-Response and Threshold Concepts

The focus of toxicological studies in humans or animals is identification of the relationship between exposure to different doses of a specific contaminant and the chance of having a health effect from each exposure level. This dose-response relationship provides a mathematical formula or graph that is used to estimate a person's risk of illness. The actual shape of the dose-response curve requires scientific knowledge of how a hazardous substance

affects different cells in the human body. There is one important difference between the dose-response curves used to estimate the risk of noncancer illnesses and those used to estimate the risk of cancer: the existence of a threshold dose. The threshold dose is the highest exposure dose at which there is no risk of illness. The dose-response curves for noncancer illnesses include a threshold dose that is greater than zero. Scientists include a threshold dose in these models because of the observation that the human body is capable of adjusting to varying amounts of other types of cell damage without showing signs of illness. The threshold dose differs for different contaminants and different exposure routes, and is estimated from information gathered in human and animal studies. In contrast, the dose-response curves used to estimate the risk of cancer assume there is no threshold dose (or, the cancer threshold dose is zero). This assumes a single cancer cell may be sufficient to cause a clinical case of cancer (EPA 1990b). This assumption is very conservative, and many scientists believe a threshold dose greater than zero also exists for the development of cancer.

Uncertainty in Health Assessments

All health assessments require the use of assumptions, judgements, and incomplete data to varying degrees. These contribute to the uncertainty of the final risk estimates. Some of the more important sources of uncertainty in this public health assessment include environmental sampling and analysis, exposure parameter estimation, use of modeled data, and present toxicological knowledge. These uncertainties may cause risk to be overestimated or underestimated to different extents (EPA 1993a). As a result of the uncertainties described below, this public health assessment should not be construed as representing an absolute estimate of risk to persons potentially exposed to chemicals at or near the Hipps Road Landfill Site.

Environmental chemistry analysis errors can arise from random errors in the sampling and analytical processes, resulting in either an over- or under-estimation of risk. These errors can be controlled to some extent by increasing the number of samples collected and analyses performed, and by sampling the same locations over several different time periods. These actions tend to make uncertainty contributed from random sampling errors small (EPA 1993a). However, only a small number of samples were collected for some contaminants, and many sample locations were not sampled more than once. The limited data from these areas may not be representative of the presence or concentrations of contaminants across the entire area. Consequently, the risk of illness for these contaminants may be over- or under-estimated.

There are two areas of uncertainty related to exposure parameter estimation. The first is related to exposure point concentration estimation. The second is related to the parameter values used to estimate chemical exposures (EPA 1993a). In this assessment we used maximum detected concentrations as the exposure point concentration. We believe using the maximum measured value to be appropriate because we cannot be certain what the peak contaminant concentrations are, and we cannot statistically predict peak values because the sample numbers and distribution are unsuitable for this type of analysis. Nevertheless, this

assumption introduces uncertainty into the health assessment that may over- or under-estimate the actual risk of illness. When selecting parameter values to estimate exposure dose, we used default assumptions and values within the ranges recommended by ATSDR or EPA. These default assumptions and values are designed to be conservative and may contribute to the over-estimation of risk of illness. Similarly, we assumed exposures took place from the time the landfill opened and that exposure occurred on a regular basis for each selected pathway. Both of these assumptions are likely to contribute to the over-estimation of risk of illness

For some of the identified data gaps we used modeled data to obtain exposure dose estimates. In particular, we used modeled data to estimate past contaminant concentrations in air and in some foods. ATSDR does not support using modeled data for evaluating possible health effects; rather, they recommend these data be used only to support a need for more sampling. Nevertheless, we believe we are justified in using modeled data in this public health assessment for two reasons. First, the maximum groundwater concentrations in the past generally are much higher than those in the present, and no amount of present-day sampling will yield past concentration data (unless other models are used). Second, nearby residents are greatly concerned their exposure from solvent volatilization will be ignored; they have specifically asked us to address this concern in the public health assessment to account for their total probable exposure to these contaminants. Still, using modeled data introduces uncertainties into the exposure dose estimates that may over- or under-estimate the actual risk of illness.

There are also data gaps and uncertainties in the design, extrapolation, and interpretation of toxicological experimental studies (EPA 1993a). Data gaps contribute uncertainty because information is either not available or must be addressed qualitatively. For example, possible health effects related to skin absorption represents a data gap for most contaminants in this public health assessment. Moreover, the available information on the interaction among chemicals found at the site, when present, is qualitative (that is, a description instead of a number) and cannot be applied mathematically to the dose estimates. These kinds of data gaps may tend to underestimate the actual risk of illness. In addition, there are great uncertainties in extrapolating from high to low doses, and from animal to human populations. Extrapolating from animals to humans is uncertain because of the differences in the uptake, metabolism, distribution, and body organ susceptibility between different species. Human populations are also variable because of differences in genetic constitution, diet, home and occupational environment, activity patterns, and other factors. These uncertainties can result in an over- or under-estimation of risk of illness. Finally, there are great uncertainties in extrapolating from high to low doses, and controversy in interpreting these results. Because the models used to estimate dose-response relationships in experimental studies are conservative, the risk estimates resulting from these models tend to be over-estimated. Currently, there is much debate in the scientific community as to how much the actual risks are over-estimated and what the risk estimates really mean.

A. Toxicological Evaluation

Introduction

In this subsection, we discuss exposure levels and possible health effects that might occur in people exposed to the 35 contaminants of concern at the site. To evaluate exposure, we estimated the daily dose of each contaminant of concern found at the site. Kamrin (1988) explains a dose in this manner:

> "...all chemicals, no matter what their characteristics, are toxic in large enough quantities. Thus the amount of a chemical a person is exposed to is crucial in determining the extent of toxicity that will occur. In attempting to place an exact number on the amount of a particular compound that is harmful, scientists recognize that the size of an organism has to be taken into account. It is unlikely, for example, that the same amount of a particular chemical that will cause toxic effects in a 1-pound rat will also cause toxicity in a 1-ton elephant.
>
> Thus instead of using the amount that is administered or to which an organism is exposed, it is more realistic to use the amount per weight of organism. Thus it could be said that an amount of 1 ounce administered to a 1-pound rat is equivalent to 2000 ounces to a 2000-pound (1-ton) elephant. In each case, the amount per weight is the same: 1 ounce for each pound of animal.
>
> This amount per weight is known as the dose. It is used to determine the amount of drug to prescribe to patients of differing weights and is used in toxicology to compare the toxicity of different chemicals in different animals."

In expressing the daily dose, we used the units of milligrams of contaminant per kilogram of body weight per day (mg/kg/day).

To calculate the daily dose of each contaminant, we used standard assumptions about body weight, ingestion and inhalation rates, exposure time length, and other factors needed for dose calculation (Tables 16-19, Appendix B). The standard values and dose-related equations we used originated from ATSDR and EPA guidance manuals (ATSDR 1992a, 1993a; EPA 1990a). In calculating the dose, we assumed residents were exposed to the maximum concentration measured for each contaminant in each medium (Tables 4-13, Appendix B). To calculate daily doses, we used the computer software, Risk*Assistant™ (1993). Using this software enabled us to estimate doses from skin contact, and gave us modeled dose estimates for other potential routes of exposure we would not have been able to evaluate otherwise including inhalation of shower vapors and ambient air, ingestion of fish, and ingestion of homegrown vegetables. Still, we did not have models available to evaluate potential inhalation of vapors from the fill material; skin absorption from contact with organic materials in soil, especially contact with the fill material at the site; or skin absorption of contaminants from household uses of water, other than showering.

Because some body functions work differently in adults and children, we estimated contaminant doses for three hypothetical individuals: a young child, an average child, and an adult. We defined a young child as a child from 0-6 years of age who exhibited pica behavior, the abnormal ingestion of large amounts of non-food substances including soil. Although all children inadvertently ingest soil as a part of normal mouthing behavior, this activity usually stops around 18 months of age. Pica behavior is rare. However, when it occurs, pica behavior is usually established by 18 months of age and may persist until a child is six years old (EPA 1990a). In terms of exposure, pica children are likely to ingest abnormally large amounts of soil, making their daily dose of a soil-borne contaminant much higher than that of other children or adults. We defined an average child by using mid-range values for all parameters for children between 0-18 years of age. We assumed average children did not exhibit pica behavior. To estimate contaminant exposure during swimming, we assumed swimming in area ponds and creeks began at 6 years of age, and average children had more opportunities to swim than adults. For adults, we assumed exposure to contaminants took place from 1967-1993, unless we knew a specific exposure pathway (such as swimming in on-site ponds) ceased to exist beforehand. For all individuals, we assumed exposure to air-stripper contaminants will last for ten years, twice the proposed length of operation, in case operation of this device continues longer than expected.

For each of the three hypothetical individuals, we estimated human exposure from incidental ingestion of contaminated soil and sediment, incidental ingestion of contaminated surface water during swimming, ingestion of contaminated groundwater used for domestic purposes, skin absorption of contaminants while swimming or showering, and inhalation of contaminants from the air stripper. Because there are no existing data on contaminant exposure from eating locally harvested food, inhaling vapors while showering, or breathing air inside and outside the home, we used Risk*Assistant's model data to evaluate the residents' exposure from these pathways.

In some cases, contaminants were found in monitor wells but not private wells. When this occurred, we considered the exposure pathway likely to be complete for three reasons. First, there were not enough samples to adequately characterize shallow groundwater in on-site private wells; consequently, we could not eliminate the possibility that a substance found in a bore hole or on-site monitor well might also be in an on-site private well. Second, off-site private well water quality was largely unknown prior to 1990, and we could not identify all contaminants nearby residents were exposed to during the time when few samples were taken. Third, both on- and off-site monitor wells and private wells drew water from the same aquifer, and in some cases the monitor wells are in residential yards. Therefore, it seemed likely that a substance detected in a monitor well but not in a private well was a sampling artifact. As a result, we used the highest contaminant concentration found in either monitoring or private wells to predict possible health effects from groundwater exposure.

To evaluate possible noncancerous health effects at these doses, we compared the calculated dose to contaminant-specific MRLs or RfDs, when they existed, for each type of exposure route (inhalation, ingestion, and skin contact) and length of exposure (chronic - greater than

364 days of exposure, intermediate - 15 to 364 days of exposure, and acute - less than 15 days of exposure). An MRL is an estimate of the daily dose of a contaminant below which non-cancer illnesses are unlikely to occur. ATSDR develops MRLs from scientific studies found in the toxicological literature, and publishes them in a series of chemical-specific documents called toxicological profiles. These documents contain not only MRLs, but also information on possible health effects, environmental transport, human exposure, and regulatory status of contaminants. EPA publishes similar minimal risk doses, called RfDs, below which non-cancer illnesses are unlikely to occur. In evaluating the dose data for contaminants at this site, we used the MRL for comparison when both an MRL and a RfD were available. In some cases, there are no MRLs or RFDs for comparison. In these cases, we compared the estimated doses we calculated to doses in published human or animal studies in order to estimate possible health effects. Our conclusions from these comparisons are judgements based on: what we know about the quality of the study, natural disease rates in the test organisms, and how close our estimated doses are to published experimental doses. These judgements always contain some uncertainty because of natural variation within human and animal populations, and because of species differences among humans and animals. Humans and animal differences are particularly important because a given test animal species may be either more or less sensitive to a particular contaminant than humans, and often the direction of this sensitivity difference is unknown.

To evaluate possible cancerous health effects, we used standard equations to calculate an individual's additional risk of developing cancer over a lifetime after exposure to a potentially cancer-causing contaminant. This calculated probability is known as the cancer risk, the number of excess cancer cases that could develop per unit of population if the exposure assumptions are met for a specific contaminant. Usually, an excess cancer risk of 1 in 10,000 to 1 in 1,000,000 is considered a negligible increase in cancer risk. There are three things to consider when evaluating cancer risk. First, when examining the numeric cancer risk value, it is important to recognize there is a background cancer rate of around 25% in the United States (ATSDR 1993b). This means that in a group of a million people, 250,000 people can be expected to develop cancer in their lifetime without exposure to contaminants at a particular site. Within the negligible cancer risk range of 1 in 1,000,000 to 1 in 10,000 excess cancer cases for a specific contaminant, 250,001 - 250,100 people in this same group might develop cancer in their lifetime if they are exposed to that contaminant at the specified dose and exposure period. Because these cancer risk calculations are made for a lifetime, and because some cancers don't develop until many years after exposure, we do not calculate a separate cancer risk for children. Second, when interpreting the associated cancer information, it is important to note whether or not the associated cancers have been looked for and found to occur in humans. This is because a given test animal species can be more or less likely to develop cancer than humans. When only animal studies of cancer are available, we present the suggestive evidence from the animal studies, but cannot necessarily conclude human exposure will be linked to cancer. Third, there is much scientific controversy about the validity of adding cancer risks from different exposure routes together. Some scientists believe exposure to a cancer-causing chemical via multiple pathways seems likely to increase the overall cancer risk. Other scientists believe cancer risks can be added

only if the cancer-causing agent affects the same cell type within the same organ, and works through the same cellular mechanism within the common cell type. In this document, we support the principle that a common mechanism is required. Often, cellular mechanisms of action are not known; in these cases, the suitability of adding estimated cancer risks together cannot be determined. In this subsection, we present the estimated cancer risks from different exposure pathways separately. In the Community Health Concerns Evaluation subsection, we discuss additive cancer risks, when appropriate.

After examining the dose-related calculations for the 35 contaminants of concern and making the appropriate comparisons, we divided the contaminants among two categories: a minimal risk category and a possible risk category.

The minimal risk category identifies those contaminants whose dose-related value is very close to or below the applicable MRL, RfD, or within the negligible cancer risk range for a medium (soil, water, or air); or significantly below exposure levels associated with noncancer illnesses in a medium; or both. In defining "close to" values, we included contaminant doses that slightly exceeded a health value in this group for three reasons. First, the estimated dose values are not known with great precision due to the uncertainty inherent in exposure parameter estimation. Second, the conservative assumptions behind our calculations are likely to cause us to overestimate contaminant doses, and consequently to overestimate the public health risk. Third, our evaluation of the toxicological literature used to estimate the RfDs or MRLs for these specific contaminants supports this categorization. Therefore, we consider the actual risk of becoming ill from exposure to these contaminants to be minimal. The 15 minimal risk contaminants are:

Minimal Risk Contaminants

Beryllium	Cyanide	Mercury
Chlorobenzene	DDT	Naphthalene
Chlorodibromomethane	1,4-Dichlorobenzene	Nickel
Chloroform	1,2-Dichloropropane	Selenium
Cobalt	1,2-Diphenylhydrazine	Tin

The possible risk category includes those contaminants whose dose-related value is significantly greater than either the MRL or RfD, or is greater than the negligible cancer risk range in at least one medium, or has too few studies for evaluation. The 20 possible risk contaminants are:

Possible Risk Contaminants

Arsenic	1,1-Dichloroethane	n-Nitrosodiphenylamine
Barium	1,2-Dichloroethane	PCBs (total)
Benzene	Di(2-ethylhexyl)phthalate	1,1,2,2-Tetrachloroethane
Bromodichloromethane	Hexachloroethane	Tetrachloroethene
Cadmium	Lead	Trichloroethene
Chromium(VI)	Manganese	Vinyl Chloride
Cresol	Methylene Chloride	

It is important to understand that contaminants in the possible risk category are not necessarily threats to public health; they are simply selected for further evaluation. For some contaminants in this category, there simply isn't enough reliable toxicological data to fully evaluate the potential health effects from exposure.

In addition to evaluating contaminants in the latter two categories, we also examined data for contaminants we had earlier classified as contaminants with drinking water standards. The chemicals in this category include pH and inorganic chemicals with established drinking water standards but without other comparison values. In our evaluation, we compared the maximum groundwater measurements for these substances with Florida's primary and secondary drinking water MCL values. Dose calculations were not necessary. The substances and measurements in this category are:

Contaminants with Drinking Water Standards

Aluminum	Iron	pH
Copper	Nitrate	Sodium
Fluoride	Nitrite	Sulfate

Below, we discuss the concentration or estimated doses of contaminants in each category, the known interactive effects, and the possible health effects from exposure. When we have enough information, we evaluate past, present, and future exposures individually. In our evaluation, past exposure includes data collected before May 1993, present exposure includes data collected from May-October 1993, and future exposure includes the time period after October 1993.

When providing information on how close our estimated doses were to experimental doses reported in toxicological studies, we used several different terms to indicate the degree of closeness (smaller or larger) to an observed experimental dose. "Similar to" means an estimated dose value is very close to the lower values in a range of comparison doses, usually within a fraction less than one (for example, 3.4 compared to 4.0). "Slightly" means an estimated dose value is a little farther away than a fraction from the lower values of the

comparison doses, such as by a factor of two or three (for example, two or three times smaller than the comparison doses). "Somewhat" means an estimated dose value is still farther away from the lower values of the comparison doses, usually by a factor close to ten (for example, nine or ten times smaller than the comparison doses). "Much" means an estimated dose value is greater than a factor of ten from the lower values of the comparison doses (for example 30 or 100 times smaller than the comparison doses). As a dose value becomes much smaller than the lower values of the comparison doses, the uncertainty of an association between the estimated dose and possible health effects increases.

Contaminants with Drinking Water Standards

Some inorganic contaminants that do not have toxicological comparison values but do have primary drinking water standards (including sodium, fluoride, sulfate, nitrate, and nitrite) were found in the shallow groundwater surrounding the site. None of these contaminants were found in concentrations exceeding their respective maximum contaminant levels. Therefore, we do not expect illnesses to result from ingestion of these substances.

The shallow groundwater concentrations of aluminum, copper, and iron, as well as the lower range for pH violate Florida's secondary drinking water standards (Table 20, Appendix B). A comparison of the range of values between the on-site boreholes and monitoring wells and the off-site private wells suggests the landfill is a possible source of these contaminants. Secondary drinking water standards are established to provide consumers with water that is aesthetically pleasing in taste, smell, and appearance. The contaminants violating secondary drinking water standards in the Hipps Road area will not cause illnesses; however, these contaminants are found in concentrations which can contribute to the taste and odor problems reported by residents.

Minimal Risk Contaminants

Based on our comparison of the doses of these contaminants to studies in the toxicological literature, we do not believe exposure to any contaminant in this category is likely to be associated with illnesses. Nevertheless, it is important to consider the interactive effects these contaminants might have with each other, with other contaminants found at the site, and with chemicals from other common sources such as cigarette smoking, alcohol consumption, or food nutrients when this information is known. Frequently, interactive effects are grouped into one of four categories: additive effects, synergistic effects, potentiation, and antagonistic effects. Additive effects occur when the combined effects of two contaminants equals the sum of their individual effects; thus neither contaminant enhances or diminishes the effect the other (for example, 2 + 3 = 5). Additive effects are the most common interactive effects. Synergistic effects occur when the combined effects of two contaminants are much greater than the sum of their individual effects; thus each contaminant amplifies the effects of other (for example, 2 + 2 = 20). Potentiation occurs when one contaminant does not have a toxic effect on a certain body organ or system; however, when combined with another contaminant, it makes the latter much more toxic (for

example, 0 + 2 = 10). Antagonistic effects occur when the combined effects of two contaminants are less than the sum of their individual effects; the effects are reduced by one contaminant interfering with the other, or both contaminants interfering with each other (for example, 4 + 6 = 8; 4 + (-4) = 0; 4 + 0 = 1) (Amdur et al. 1991).

ATSDR or EPA has published information on all 15 of the minimal risk contaminants found at the Hipps Road Landfill site:

1. Beryllium - Beryllium is a hard, grayish element that occurs as a chemical component of certain rocks, coal and oil, soil, and volcanic dust. Beryllium is used to make making electrical and electronic parts, machinery, molds for plastics, nuclear weapons and reactors, aircraft, space vehicles, x-ray machines, and mirrors. Ingesting beryllium usually does not harm health because very little enters the body from the digestive system. Most of the small amount of beryllium that does enter the bloodstream is carried to the kidneys where it leaves the body within a few days through urination. Animal studies suggest beryllium exposure is not likely to affect reproduction. It is not known if beryllium ingestion affects the development of unborn babies. The potential interactive effects between beryllium and other substances found at the site are unknown. Beryllium ingestion is not known to be associated with cancer in humans or animals (ATSDR 1993f).

   In the past, nearby residents were exposed to beryllium through ingestion of groundwater. Present-day analyses indicate beryllium may no longer be present in well water. The past beryllium ingestion doses we estimated for all age groups are slightly smaller than EPA's RfD (IRIS 1994), indicatmg noncancer illnesses are unlikely to be associated with this exposure.

2. Chlorobenzene - Chlorobenzene is a colorless liquid with an almond-like odor. It is manufactured as a solvent and is used in the production of other chemicals. Chlorobenzene can enter the body through ingestion, inhalation, and skin absorption. Once in the body, most chlorobenzene is expelled by exhaling and urinating. Harm to human health from ingesting chlorobenzene has not been established. In animals, exposure to high levels of chlorobenzene is associated with adverse effects on the brain, liver, and kidneys. Other animal studies suggest chlorobenzene exposure does not adversely affect reproduction or the development of unborn babies. The potential interactive effects between chlorobenzene and other substances found at the site are not known (ATSDR 1990b). Chlorobenzene is not classified as a potential cancer-causing agent (ATSDR 1993a).

   In the past, nearby residents were exposed to chlorobenzene through incidental ingestion of on-site subsurface soils and ingestion of groundwater. Present-day analyses indicate chlorobenzene may no longer be present in well water. The past chlorobenzene ingestion doses we estimated for all age groups are similar to or somewhat smaller than EPA's RfD (IRIS 1994), and the modeled chlorobenzene

inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the animal studies we reviewed (ATSDR 1990b). These results indicate noncancer illnesses are unlikely to be associated with ingestion or inhalation exposure. There are no human or animal studies of the potential health effects from skin absorption of chlorobenzene (ATSDR 1990b); therefore, we cannot evaluate any potential association between this exposure route and noncancer illnesses.

3. Chlorodibromomethane - Chlorodibromomethane is a colorless liquid with a sweetish odor. In the past, chlorodibromomethane was used to make other chemicals such as fire extinguisher fluids, spray can propellants, refrigerator fluid, and pesticides. Today, it is produced only in small quantities for use in laboratories. Chlorodibromomethane is also formed as an unwanted byproduct of chlorinating drinking water. Once chlorodibromomethane enters the body, it is quickly removed by exhalation. Human and animal studies indicate ingestion of large amounts of chlorodibromomethane can affect the brain, liver, and kidneys. Exposure to low levels of this compound does not seem to be associated with serious effects on these organs. Animal studies suggest chlorodibromomethane is not likely to adversely affect reproduction or the development of unborn babies at typical human exposure levels. Studies of chlorodibromomethane's interactive effects show acetone, and possibly other ketones, potentiate the toxic effects chlorodibromomethane has on the liver. Animal studies indicate long-term intake may be associated with cancer (ATSDR 1990a).

   In the past and present, nearby residents were and are exposed to chlorodibromomethane through use of their well water. In addition, nearby residents may have been coexposed to acetone and chlorodibromomethane in their drinking water in the past, and may be exposed to acetone in the air and chlorodibromomethane in their drinking water in the future. Still, the past chlorodibromomethane ingestion doses we estimated for all age groups are much smaller than EPA's RfD (IRIS 1994), and the modeled chlorodibromomethane inhalation doses we estimated for all age groups are much smaller than ATSDR's chronic MRL (ATSDR 1990a). These results indicate noncancer illnesses are unlikely to be associated with ingestion or inhalation exposure, even if interactions with acetone occur. There are no human or animal studies of the potential health effects from skin absorption of chlorodibromomethane (ATSDR 1990a); therefore, we cannot evaluate any potential association between this exposure route and noncancer illnesses. We estimated the increased cancer risk from chlorodibromomethane exposure to be negligible.

4. Chloroform - Chloroform is a colorless liquid with a pleasant odor and a slightly sweet taste. In the past, hospitals used chloroform as an anesthetic. Today, it is used to make other chemicals and is an unwanted byproduct of chlorinating drinking water. Chloroform is found in air from all areas of the United States, and in nearly all

drinking water supplies. Once inside the body, travels to body organs and can collect in body fat. Some of the chloroform in the body is exhaled, and the rest is broken down into other chemicals. Some of these breakdown products are excreted, and others can attach to chemicals inside cells where they may cause harmful effects to the liver and kidneys. In humans, ingestion or inhalation of high doses of chloroform can have adverse effects on the brain, liver, and kidneys. Ingestion or inhalation of small doses of chloroform can be associated with harm to the liver and kidneys. Animal studies indicate inhaling moderate amounts of chloroform may adversely affect reproduction and may be associated with birth defects in rats and mice. Similar reproductive and developmental effects have not been associated with chloroform ingestion in animals (ATSDR 1993h). Several animal studies indicate chloroform may interact with other chemicals inside the body. In rats, exposure to ketones and ethanol (drinking alcohol) can increase chloroform's toxic effects on the liver and kidneys. Similarly, experiments with rat liver cells suggest cadmium and chloroform may potentiated the toxic effects of each other on these cells (ATSDR 1989b, 1993h). Human studies indicate ingesting chlorinated drinking water, which contains chloroform and other chlorination by-products, may be linked with colon and urinary bladder cancer. Animal studies indicate ingestion of small amounts of chloroform for long time periods is associated with liver and kidney cancer, but it is not known if chloroform exposure is associated with these same cancers in humans (ATSDR 1993h).

In the past, nearby residents were exposed to chloroform through household uses of well water. Present-day analyses indicate chloroform may no longer be present in well water, and chloroform was not detected in the air stripper's influent or effluent (Golder Associates 1993a). The past chloroform ingestion doses we estimated for all age groups are much smaller than EPA's RfD for this contaminant (IRIS 1994), and the modeled past chloroform inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the human and animal studies we reviewed (ATSDR 1993h). These results indicate noncancer illnesses are unlikely to be associated with ingestion or inhalation exposure even if coexposure with ethanol, ketones, or cadmium occurred. There are no human or animal studies of the potential internal health effects from skin absorption of low doses of chloroform (ATSDR 1993h); therefore, we cannot evaluate any potential association between this exposure route and noncancer illnesses. We estimated the increased cancer risk from chloroform exposure to be negligible.

5. Cobalt - Cobalt is an element that naturally occurs in rocks, soil, surface water, groundwater, plants, and animals. It is used to make alloys and colored pigments, and as a drier for paint and porcelain enameling. Ingestion is the most likely route for cobalt exposure. Small amounts of cobalt are found in tea, coffee, many fruits and vegetables, and some fish. Some of the cobalt entering the body quickly leaves in the feces; the rest is absorbed into the blood where it travels throughout the body, particularly to the liver, kidneys, and bones. This cobalt leaves the body slowly,

mainly through urination. Cobalt has both beneficial and harmful effects on human health. Vitamin B_{12} is a cobalt-containing compound essential for good health. Cobalt is also used as a treatment of anemia (a decrease in the number of red blood cells) because it causes red blood cells to be produced. However, too much cobalt may have harmful health effects. In some people, treatment with cobalt has been associated with adverse effects on the thyroid gland. In addition, some people can develop dermatitis after skin exposure to cobalt-containing compounds. It is not known if cobalt ingestion adversely affects human reproduction. Animal studies suggest high levels of cobalt may be associated with health effects in unborn babies; however, birth defects have not been seen in human babies whose mothers took cobalt for anemia during pregnancy. In other animal studies, cobalt ingestion is associated with adverse effects on the blood, liver, kidneys, and heart. When cobalt is administered in conjunction with the anti-tumor antibiotic bleomycin, these compounds interact to amplify each other's anti-tumor effects. In addition, there is some evidence that people with nickel sensitization may develop an allergy to cobalt under some circumstances. Cobalt ingestion is not known to be associated with cancer in humans or animals (ATSDR 1992c).

In the past, nearby residents were exposed to cobalt through incidental ingestion of on-site sediment and off-site surface water, through ingestion of groundwater. There are no present-day analyses of cobalt, and it is not known if exposure to this substance is continuing. The cobalt ingestion doses we estimated for all age groups are much smaller than the levels associated with noncancer illnesses in the human and animal studies we reviewed (ATSDR 1992c).

6. Cyanide - Cyanides are a group of compounds naturally produced by certain bacteria, fungi, and algae; they are naturally found in a number of foods (for example, cassava roots, lima beans, and almonds). Most cyanides in the soil and water, however, come from industrial sources. Cyanides are also found in vehicle exhaust. Once in the body, cyanide can quickly enter the bloodstream. The health effects of cyanide depend on the chemical form it is in. Inhaling or ingesting large amounts of cyanide harms the brain, heart, and lungs, and can result in coma or death. Nevertheless, small amounts of some cyanide compounds are always present in the body. Animal studies suggest cyanide ingestion does not adversely affect reproduction, but may affect the development of unborn babies. In the body, some of the cyanide is changed to a harmless chemical that is excreted in the urine, some interacts with a different body chemical to form vitamin B_{12}, and some is converted to carbon dioxide and exhaled. Most of the ingested cyanide will leave the body within 24 hours after exposure. One study found synergism between potassium cyanide and vitamin C in guinea pigs, resulting in increased tremors, muscle incoordination, and muscle twitches in these animals. Antagonists stabilizing cyanide into nonharmful compounds include sodium nitrite, amyl nitrite, hydroxylamine, and cobalt containing compounds (ATSDR 1993c). Cyanide is not classified as a potential cancer-causing agent (ATSDR 1993a).

In the past, nearby residents were exposed to cyanide through incidental ingestion of on-site subsurface soils, on-site sediments, and ingestion of groundwater. In addition, nearby residents may have been coexposed to cyanide and dietary vitamin C, and to cyanide and nitrates and cobalt in their drinking water in the past. There are no present-day analyses of cyanide, and it is not known if exposure to this substance is continuing. The degree of interaction of ingested vitamin C with ingested cyanide is unknown, but such interactions might occur in humans. Ingestion of nitrates and cobalt with cyanide could somewhat lessen the health effects from cyanide exposure. The past cyanide ingestion doses we estimated for all age groups are similar to or somewhat smaller than EPA's RfD (IRIS 1994), indicating noncancer illnesses are unlikely to be associated with this exposure.

7. DDT - DDT, a widely used pesticide in the past, is sometimes found in food. It is also found at many waste sites, and releases from these sites can be additional sources of human exposure. DDT does not enter the body through the skin easily. Once in the body, DDT can be broken down and excreted in the urine. Nevertheless, DDT is readily stored in body fat, where levels may increase if exposure continues, or decrease slowly over time if exposure decreases. Short-term exposure to high DDT doses is associated primarily with effects on the nervous system. Long-term exposure to low doses is associated with temporary changes in liver enzyme levels. Although there is no indication DDT adversely affects human reproduction, animal studies suggest DDT ingestion may affect the development of unborn babies. DDT seems to have broad interactive effects by changing the effects of other chemicals. DDT reportedly promotes the tumor-forming effects of some cancer-causing agents, but inhibits the tumor-forming effects of other cancer-causing agents. Similarly, some pharmaceutical drugs prevent DDT's toxic effects on the nervous system, while other drugs enhance DDT's toxicity to the nervous system. The potential interactive effects between DDT and other substances found at the site are unknown. In some animal studies, DDT ingestion has been associated with liver cancer. It is not known if DDT ingestion is associated with cancer in humans (ATSDR 1992e).

   In the past, nearby residents were exposed to DDT through incidental ingestion while swimming in on-site ponds. Because these ponds no longer exist, exposure via this route has stopped. The past DDT ingestion doses we estimated for all age groups are much smaller than EPA's RfD (IRIS 1994), indicating noncancer illnesses are unlikely to be associated with this exposure. We estimated the increased cancer risk from DDT ingestion to be negligible.

8. 1,4-Dichlorobenzene - At room temperature, 1,4-dichlorobenzene is a white solid with the strong odor of mothballs. 1,4-Dichlorobenzene is produced by chemical industries to make mothballs, deodorant blocks, and resins. Most people are exposed to 1,4-dichlorobenzene from breathing household products containing this compound such as mothballs and toilet deodorizer blocks. Because 1,4-dichlorobenzene is sometimes used to control odor in animal stalls, it can be found pork, chicken, and eggs. It may

also be found in fish and human breast milk. After exposure, most 1,4-dichlorobenzene enters the bloodstream. Almost all 1,4-dichlorobenzene entering the body is broken down into the chemical 2,5-dichlorophenol. It is not known if this breakdown product is more or less harmful than 1,4-dichlorobenzene itself. Almost all of 1,4-dichlorobenzene leaves the body within a week through urination. Tiny amounts remain in body fat, and may stay there for a long time (ATSDR 1993j). There is no evidence moderate use of household products containing 1,4-dichlorobenzene is associated with illnesses. There are cases of people eating sweet-tasting 1,4-dichlorobenzene products and subsequently experiencing skin blotches and blood illnesses, such as anemia. In animal studies, breathing or eating 1,4-dichlorobenzene can be associated with illnesses of the liver, kidneys, and blood. Concentrations of 1,4-dichlorobenzene typically found around hazardous waste sites are not likely to adversely affect human reproduction or the development of unborn babies. The potential interactive effects between 1,4-dichlorobenzene and other substances found at the site are unknown. Studies of rats and mice suggest life-long ingestion of 1,4-dichlorobenzene may be associated with in higher incidences of cancer, but these studies are not conclusive (ATSDR 1993j).

In the past, nearby residents were exposed to 1,4-dichlorobenzene through incidental ingestion of on-site subsurface soils and household uses of groundwater. The limited number of present-day samples indicate 1,4-dichlorobenzene is no longer found in the groundwater. The 1,4-dichlorobenzene ingestion and modeled inhalation doses we estimated for all age groups are, respectively, much smaller than and similar to their corresponding intermediate MRLs, and much smaller than the levels associated with noncancer illnesses in the acute and chronic human and animal studies we reviewed. There were no skin absorption studies available for evaluation (ATSDR 1993j). There is no apparent increased risk of cancer from 1,4-dichlorobenzene ingestion. It is not known if 1,4-dichlorobenzene inhalation or skin absorption is associated with cancer in humans or animals (ATSDR 1993j).

9. 1,2-Dichloropropane - 1,2-Dichloropropane is a colorless liquid that evaporates easily at room temperature. It is now used only in research and industry. Before 1980, it was used as a soil fumigant for farming and was a component of some paint strippers, varnishes, and furniture finish removers. 1,2-Dichloropropane released into the environment usually ends up in the groundwater or air where it breaks down slowly. Once in the body, 1,2-dichloropropane quickly leaves the body through urination, defecation, and exhalation. Drinking or breathing very high levels of 1,2-dichloropropane is associated with poisoning in humans, but there are no reports of any human health effects associated with short or long term exposure to low-levels of this chemical. However, in animal studies, low-level exposure for short or long time periods is associated with liver, kidney, and respiratory damage. One animal study suggests ingestion of high amounts of 1,2-dichloropropane may be associated with harmful effects on sperm formation. Skin contact with 1,2-dichloropropane is associated with skin irritation in both humans and animals. 1,2-Dichloropropane is

not associated with birth defects in humans or animals, but studies of rats indicate delayed bone growth in unborn babies may be associated with maternal exposure to 1,2-dichloropropane during pregnancy. Animals studies indicate inhalation of 1,2-dichloropropane with tetrachloroethene can have additive effects on the liver, lung, and nervous system. Human studies of short-term ingestion or inhalation of 1,2-dichloropropane have not found an association with cancer. However, in animals, long-term ingestion of 1,2-dichloropropane may be associated with liver cancer in mice and breast cancer in female rats. The significance of the animal cancer findings to humans is not well understood (ATSDR 1989c).

In the past, nearby residents were exposed to 1,2-dichloropropane through household uses of well water. Present-day analyses indicate exposure is continuing for residents still using private well water from areas next to the groundwater contaminant plume. In addition, the air stripper's trial run demonstrated this device will successfully remove 1,2-dichloropropane from groundwater and expel it into the air. Furthermore, nearby residents may have been coexposed to tetrachloroethene and 1,2-dichloropropane in their drinking water and air in the past, and may be exposed to tetrachloroethene and 1,2-dichloroethane in the drinking water and air in the present and future. Still, the past and present 1,2-dichloropropane ingestion doses we estimated for all age groups are much smaller than ATSDR's intermediate MRL for this contaminant. Chronic ingestion studies were not available for review. The modeled past and present 1,2-dichloropropane inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the animal studies we reviewed (ATSDR 1989c). These results indicate noncancer illnesses are unlikely to be associated with ingestion or inhalation exposure even if coexposure with tetrachloroethene occurred or occurs. Nevertheless, several case studies indicate some people are allergic to 1,2-dichloropropane-containing products and may develop contact dermatitis upon skin contact with these products in sensitized individuals. It is not known at what exposure level the allergic response develops. There are no human studies of the potential internal effects from skin absorption of 1,2-dichloropropane, but the two available animal studies did not find an association between skin exposure to 1,2-dichloropropane and internal noncancer illnesses (ATSDR 1989c). We estimated the increased cancer risk from 1,2-dichloropropane ingestion to be negligible. It is not clear if 1,2-dichloropropane inhalation is associated with cancer in animals. There are no human or animal studies of the potential association between skin absorption of 1,2-dichloropropane and cancer (ATSDR 1989c).

10. 1,2-Diphenylhydrazine - 1,2-Diphenylhydrazine is a white solid that tends to stick to soil. 1,2-Diphenylhydrazine is used to make fabric dyes and to make certain medicines. Most people are exposed to 1,2-diphenylhydrazine by incidentally ingesting dirt or breathing in dust in areas where 1,2-diphenylhydrazine has been recently spilled or uncovered. Not much is known about how 1,2-diphenylhydrazine enters the body or how the body breaks down 1,2-diphenylhydrazine. Still, two of

the known breakdown products, aniline and benzidine, may contribute to 1,2-diphenylhydrazine's toxicity. Animal studies indicate at least some 1,2-diphenylhydrazine leaves the body through urination. The health effects of 1,2-diphenylhydrazine have not been studied in humans. Animals studies indicate chronic oral exposure to low doses of 1,2-diphenylhydrazine may be associated with liver damage. It is not known if exposure to 1,2-diphenylhydrazine has adverse effects on reproduction or the development of unborn babies. The potential interactive effects between 1,2-diphenylhydrazine and other substances found at the site are unknown. In addition, it is not known if 1,2-diphenylhydrazine is associated witb cancer in humans. However, in rats and mice, chronic oral exposure to 1,2-diphenylhydrazine is associated with liver and breast cancer (ATSDR 1990d).

In the past, nearby residents were exposed to 1,2-diphenylhydrazine through incidental ingestion of on-site subsurface soils and sediments. There are no present-day analyses of 1,2-diphenylhydrazine, and it is not known if exposure to this substance is continuing. The 1,2-diphenylhydrazine ingestion doses we estimated for all age groups are much smaller than the levels associated with noncancer illnesses in the animal studies we reviewed (ATSDR 1990d). There is no apparent increased risk of cancer from past 1,2-diphenylhydrazine ingestion.

11. Mercury - Mercury is a naturally occurring metal found throughout the environment as a result of normal breakdown of the earth's crust by wind and water. Mercury can occur in metallic, organic and inorganic forms. All forms of mercury are considered poisonous. Mercury has many different uses. Metallic mercury is used in thermometers, barometers, batteries, and tooth fillings. Inorganic mercury is used in electrical equipment, skin care and medicinal products, and some fungicides. Organic mercury can be found in some paints and fungicides. A natural form of organic mercury is sometimes found in fish. Mercury found in air, water, and soil is thought to be mostly in the inorganic form. Inorganic mercury can enter the body through the digestive system and subsequently reach many tissues. It can stay in the kidneys for a relatively long time. Inorganic mercury leaves the body through urination or defecation after several weeks or months. The kidneys seem to be the most sensitive target of low-level exposure to inorganic mercury. Long-term exposure to higher than normal levels of inorganic mercury may be associated with kidney and brain damage in some people. In animals, short- and long-term exposure to low inorganic mercury levels is associated with adverse kidney and brain effects, and may be associated with adverse effects on unborn babies. It is not known if ingestion of inorganic mercury adversely affects reproduction. Vitamin D, vitamin E, selenium, and copper are antagonistic to the toxic effects of mercury. In rats, pretreatment with zinc seems to be protective against inorganic mercury's effects on the kidneys. In contrast, ethanol (drinking alcohol) consumption appears to increase the toxicity of mercury. Mercury ingestion is not known to be associated with cancer in humans or animals (ATSDR 1992i).

Based on information we have about the site and the chemical analyses run, we presume the mercury detected around the site to be in the inorganic form. In the past, nearby residents were exposed to inorganic mercury through incidental ingestion of on-site subsurface soils and sediments, incidental ingestion of on-site surface water, and ingestion of groundwater. Present-day analyses indicate inorganic mercury may no longer be present in well water. The inorganic mercury ingestion doses we estimated for all age groups are somewhat smaller than and similar to ATSDR's acute and intermediate MRL's, respectively, and much smaller than the levels associated with noncancer illnesses in the chronic animal studies we reviewed. Nevertheless, several case studies indicate some people are allergic to mercury-containing products and may develop contact dermatitis, rashes or blisters upon skin contact with these products in sensitized individuals. It is not known at what exposure level the allergic response develops (ATSDR 1992i).

12. Naphthalene - Naphthalene is a white solid with a strong odor that evaporates easily. It is used to make moth repellents, deodorizing blocks, dyes, resins, leather tanning agents, and insecticides. Naphthalene enters the body by breathing air, smoking, drinking water, or touching products containing this chemical. These exposure routes include breathing in vapors or wearing clothes stored in naphthalene-containing mothballs. Once in the blood, naphthalene travels to the liver and other organs where it is changed into other chemicals, some of which can be harmful to health. Naphthalene is able to cross a pregnant woman's placenta and get into a baby's blood. Most breakdown products of naphthalene are excreted in the urine. Smaller amounts are excreted in feces, and some can be excreted in mother's milk. It may take several weeks for all traces of naphthalene to leave the body. In humans, exposure to a high amount of naphthalene can cause hemolytic anemia, a condition in which unusual numbers of red blood cells are damaged or destroyed as they move through tbe circulatory system, as well as nausea, vomiting, diarrhea, blood in the urine, and yellow-colored skin. Pregnant women who develop naphthalene-induced anemia can have anemic children. Animal studies suggest inhaling naphthalene vapors can be associated with nose and lung inflammation, and ingesting naphthalene can be associated with weight reduction in the thymus and spleen or with cataract (cloudiness) development in the eyes. Some animal studies also suggest naphthalene ingestion may adversely affect reproduction. The potential interactive effects between naphthalene and other substances found at the site are unknown (ATSDR 1993n). Naphthalene is not classified as a cancer-causing agent (ATSDR 1993a).

In the past, nearby residents were exposed to naphthalene through incidental ingestion of on-site subsurface soil and ingestion of groundwater. A limited number of present-day samples indicate naphthalene is no longer found in the groundwater. The past ingestion, inhalation, and skin absorption doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the animal studies we reviewed (ATSDR 1993n). These results indicate noncancer illnesses are unlikely to be associated with naphthalene exposure.

13. Nickel - Nickel is a hard, silvery white metal that naturally occurs in the earth's crust. Nickel is commonly mixed with other metals to make metal coins, jewelry, stainless steel, industrial valves, and heat exchangers. Nickel compounds are used in hairdressing, nickel plating, coloring ceramics, making batteries, and forming enzymes used in chemical reactions. Nickel that enters the bloodstream leaves the body in the urine. In one study, some workers who drank high amounts of nickel from a water fountain developed stomach aches, increased numbers of red blood cells, and protein in the urine. In humans, skin exposure to nickel can cause an allergic reaction characterized by skin rashes and asthma. Eating nickel can cause this skin rash to return in sensitive people. In animals, ingesting large amounts of nickel has been associated with lung disease in dogs and rats, and with adverse effects on the stomach, liver, blood, kidneys, and immune system in rats and mice. Animal data suggest nickel ingestion may have adverse effects on reproduction and the development of unborn babies. Manganese appears to interact and reduce nickel's deposition in the liver, kidney, and lung while increasing its elimination through the urine. Pretreatment with cadmium, on the other hand, appears to enhance nickel's toxic effects on the kidney and liver. In iron deficient rats, nickel enhanced the absorption of one form of iron (ferric sulfate), but not others. Nickel ingestion is not known to be associated with cancer in humans or animals (ATSDR 1993o).

In the past, nearby residents were exposed to nickel through incidental ingestion of on-site subsurface soil and ingestion of groundwater. Present-day analyses indicate nickel may no longer be present in groundwater. The past nickel ingestion doses we estimated for all age groups are similar to EPA's RfD (IRIS 1994), indicating noncancer illnesses are unlikely to be associated with this exposure. Nevertheless, several case studies indicate some people are allergic to nickel-containing products and may develop contact dermatitis upon skin contact with these products in sensitized individuals. It is not known at what exposure level the allergic response develops.

14. Selenium - Selenium is an essential nutrient found in grains, cereals, and meat. It is commonly found in drinking water and sometimes found at hazardous waste sites. Selenium can be harmful in daily levels only somewhat larger than needed for good nutrition. Selenium exposure can lead to brittle hair, deformed nails, and in extreme cases, loss of feeling and control in arms and legs. Some animal studies suggest selenium ingestion may adversely affect female fertility. Animal data associating selenium ingestion with birth defects are inconclusive. Once ingested, selenium leaves the body mostly through urination. Interactions between selenium and other metals, vitamins, and nutrients usually lead to a reduced toxicity of selenium and/or the interacting substance. Selenium reduces the toxicity of many metals including cadmium, lead, mercury, silver, and to some extent copper. Arsenic decreases the toxicity of selenium in most cases (ATSDR 1989d). Selenium is not classified as a cancer-causing agent (ATSDR 1993a).

In the past, nearby residents were exposed to selenium through ingestion of groundwater. Present-day analyses indicate selenium may no longer be present in groundwater. The past selenium ingestion doses we estimated for all age groups are similar to or much smaller than EPA's RfD (IRIS 1994), indicating noncancer illnesses are unlikely to be associated with this exposure.

15. Tin - Tin is a soft, white metal found in small amounts in the earth's crust. It is also found in food containers, plastics, and a wide variety of industrial and household products. Because tin is naturally found in soil and water, it is normally present in plants and animals. Tin is sometimes found in elevated concentrations around hazardous waste sites, and people living near these sites may be exposed to higher-than-normal levels of this metal. Most ingested tin leaves the body in the feces, and some leaves the body in the urine. Very little tin can enter the body through unbroken skin. Although inorganic tin compounds tend to leave the body quickly, very small amounts stay in some body tissues, such as the bones, for longer periods of time. Exposure to large amounts of inorganic tin compounds is associated with stomach aches, anemia (a decrease in the number of red blood cells), liver and kidney problems, and skin and eye irritation. Inorganic tin compounds are not associated with adverse reproductive effects, birth defects, or cancer. Tin can interact with other essential metals needed in the diet. In rats, iron and copper lessen tin's effects on blood hemoglobin (proteins that carry oxygen in the blood). In humans, zinc uptake seems to decrease when administered with equal amounts of tin and iron (ATSDR 1992g). Tin is not classified as a cancer-causing agent (ATSDR 1993a).

    In the past, nearby residents were exposed to tin through incidental ingestion of on-site subsurface soils, incidental ingestion of surface water while swimming in on-site ponds, and ingestion of well water. There are no present-day analyses of tin, and it is not known if exposure is continuing. The past tin ingestion doses we estimated for all age groups are much smaller than EPA's RfD (IRIS 1994), indicating noncancer illnesses are unlikely to be associated with this exposure.

Possible Risk Contaminants

Contaminants included in this category have estimated doses above the MRL, RfD, or negligible cancer risk range; have estimated doses relatively close to doses associated with health effects in humans or animals; or do not have enough information for evaluation. Being above a health value does not necessarily mean exposure to a contaminant will cause illnesses; it simply means the contaminant needs further evaluation. We perform this evaluation by comparing the doses we estimated for different age groups of residents with doses found in human or animal studies published in the toxicological literature. In examining this literature, we relied heavily on the study summaries presented in the ATSDR toxicological profiles and in EPA's IRIS (Integrated Risk Information System) database. IRIS contains toxicological information for many contaminants commonly found at hazardous waste sites.

Likely health effects are influenced not only by exposure dose (how much), but also by exposure duration (how long), and exposure route (breathing, eating and drinking, or skin contact). Once exposure occurs, a person's individual characteristics such as age, sex, diet, general health, lifestyle, chemical exposure history, and genetics also influence how the body absorbs, distributes, metabolizes, and excretes a chemical. Together these factors determine health effects exposed people might have.

Overall, the doses we estimate for some contaminants may be associated with illness, while the estimated doses for other contaminants are unlikely to be associated with illness. For many contaminants, we do not have sufficient information to fully estimate the potential health effects a particular contaminant might have.

For each of the 20 contaminants below, we present a summary of our findings, followed by more detailed information about the contaminant. This more detailed information includes a summary of the contaminant's use and likely route(s) of exposure, as well as general information on known health effects. In many cases, health effects for a specific contaminant are only associated with high exposure doses, and it can be inappropriate to assume these effects will also associated with the low exposure doses estimated for the residents. Nevertheless, we intentionally present high exposure dose information in this assessment so that the description of a contaminant's interactive effects can be better understood. Because the interactive effects information is not quantitative, we can only address these effects qualitatively in our discussion. Under site-specific health effects, we discuss the human or animal illnesses associated with exposure doses close to the doses estimated for the residents. We discuss noncancer and cancer illnesses separately. At the end of each discussion, we present information known about groups of people likely to be unusually sensitive to the contaminant.

1. Arsenic

Summary - In human studies, arsenic ingestion doses similar to past doses we estimated for young children have been associated with symptoms of digestive system irritation, mild symptoms of nerve dysfunction, various skin changes, liver enlargement, or a thickening of blood vessel walls that eventually may lead to vessel damage. The past arsenic ingestion doses we estimated for average children and adults are much smaller than the doses associated with noncancer illnesses in the human studies we reviewed. We estimate the increased cancer risk from past arsenic ingestion to be moderate.

Use and Human Exposure - Because arsenic is a natural element, low levels of this metal are commonly present in water, soil, food, and air. Commercially, arsenic is used as a wood preservative and is found component in some insecticides and weed killers. Most arsenic compounds can dissolve in water. Although arsenic is not broken down or destroyed in the environment, it can change from one form to another through chemical reactions with natural substances, including bacteria. Some fish

build up arsenic in their tissues, but most of it is in a form that is not toxic. Arsenic does not pass through the skin easily, and exposure to this element usually occurs through ingestion. Once in the body, the liver changes some of the arsenic to a less harmful form. Most of the arsenic that enters body leaves through urination within several days. However, some remains in the body for several months or longer (ATSDR 1993d).

General Health Effects - Arsenic has been known to be a human poison since ancient times. In very high doses, it can cause death. At lower doses, it can irritate the stomach, impair blood formation, cause skin changes, and affect the functioning of the heart, blood vessels, and nerves. It is not known if arsenic adversely affects reproduction or the development of unborn children. Arsenic exposure has been linked with skin cancer, and may also increase the risk of cancer in the liver, bladder, lung, and kidney. Arsenic is classified as a known cancer-causing agent in humans via ingestion (ATSDR 1993a, 1993d).

Interactions with Other Chemicals - Arsenic compounds tend to decrease the toxic effects of selenium. The interaction between arsenic and smoking has not been extensively investigated, although there seems to be a positive interaction (either additive or synergistic) between the two in increasing lung cancer risk. Similarly, experiments with hamsters suggest a positive interaction between arsenic and benzo(a)pyrene in increasing lung cancer risk (ATSDR 1993d).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to arsenic through incidental ingestion of on-site subsurface soils and sediment, and ingestion of groundwater. Present-day analyses indicate arsenic may no longer be present in well water, but we do have enough groundwater, surface soil, or sediment samples to confirm exposure has stopped. In human studies, arsenic ingestion doses similar to past doses we estimated for young children have been associated with symptoms of digestive system irritation including nausea, vomiting, diarrhea, and abdominal pain; mild symptoms of nerve dysfunction, initially appearing as numbness in the hands and feet which may later develop into a painful "pins and needles" sensation; a thickening of the skin, as well as wart or corn formation on the palms or soles; skin pigmentation changes on the face, neck, and back; tenderness or enlargement of the liver; or a thickening of blood vessel walls that can lead to vessel damage. The past arsenic ingestion doses we estimated for average children and adults are much smaller than the doses associated with noncancer illnesses in the human studies we reviewed (ATSDR 1993d).

Site-specific Cancer Risk - There is convincing evidence that arsenic ingestion can increase the risk of developing skin cancer. The most common lesions appear to develop from some of the warts and corns described above, although other sources of arsenic-induced skin cancer occur. In addition to the risk of skin cancer, there is mounting evidence that arsenic ingestion may increase the risks of several internal

cancers, including bladder, kidney, liver, and lung cancer (ATSDR 1993d). Consequently, EPA, the U.S. Department of Health and Human Services' National Toxicology Program (NTP), and the International Agency for Research on Cancer (IARC) each have classified arsenic as a known human cancer-causing agent via ingestion of drinking water; EPA and NTP each have classified arsenic as a known human cancer-causing agent via ingestion of soil (ATSDR 1993a). Based on the exposure and dose information we have, we estimate the increased cancer risk from past arsenic ingestion to be moderate at 4 in 1,000. This means the risk of developing cancer, above the background rate, could rise from 250 cases per 1,000 people to 254 cases in a 70-year lifetime.

Sensitive Populations - ATSDR's toxicological profile for arsenic did not cite any studies concerning groups of people that were unusually sensitive to arsenic. However, because methylation of arsenic in the liver is a detoxification mechanism, it seems likely that some members of the population who have a lower than normal methylating capacity may be especially susceptible to the toxic effects of arsenic (ATSDR 1993d).

2. Barium

Summary - The past barium ingestion doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in nearly all studies we reviewed. It is not clear if the barium ingestion doses we estimated for all age groups might be associated with increased blood pressure. Barium's potential to cause cancer via any exposure route is unknown.

Use and Human Exposure - Barium is a silvery-white metal that occurs in nature in many different forms or compounds. Barium and its compounds are used to make drilling muds, paints, bricks, tiles, glass, rubber, insect and rat poisons, and fuel additives. Doctors sometimes use barium compounds to perform medical tests and take x-ray photographs of the stomach and small intestine. Background levels of barium in the environment tend to be very low. Industrial operations can release barium into the air, soil, and water where they may be inhaled or ingested by people. Some foods such as Brazil nuts, seaweed, fish, and certain plants may contain high amounts of barium. Only a small amount of barium can enter the body through skin contact with barium compounds. Most of the barium that enters the body leaves within a few days in the feces and urine. The small amount of barium that stays in the body mostly goes into bones and teeth (ATSDR 1992b).

General Health Effects - The potential health effects of different barium compounds depends on how well they dissolve in water. Water-insoluble barium compounds have few health effects, but water-soluble barium compounds can cause illnesses. Most of what is known about water-soluble barium's effects in people come from studies of short-term exposure at fairly large doses. Eating or drinking very high

doses of barium compounds can cause paralysis or death. At somewhat lower doses, barium ingestion is associated with breathing difficulties, increased blood pressure, changes in heart rhythm, stomach irritation, minor changes in the blood, muscle weakness, changes in nerve reflexes, swelling of the brain, and damage to the liver, kidney, heart, and spleen. The long-term effects of barium that stays in the body are unknown. Similarly, barium's effects on reproduction or the development of unborn children are unknown, as is its potential to cause cancer (ATSDR 1992b).

Interactions with Other Chemicals - Barium may interact with potassium, magnesium, and calcium normally present in the body. In animals, potassium is a powerful antagonist of paralysis and heart effects caused by barium. In other experiments, magnesium and calcium suppress the uptake of barium in pancreas cells grown in an artificial environment. In addition, barium interacts with components found in several prescription drugs. In rats, barium increases the depressive effects drugs containing sodium pentobarbital and phenobarbital have on the heart. In mice, atropine and naloxone inhibit the lethal toxicity of barium. In rabbits, verapamil and doxepin seem to offer some protection against barium-induced heart rhythm abnormalities. The interactive effects between barium and other substances found at the site are unknown (ATSDR 1992b).

Site-specific Noncancer Health Effects - Nearby residents were exposed to barium through incidental ingestion of off-site sediments and ingestion of groundwater. Present-day analyses indicate barium may no longer be present in well water, but we do not have enough groundwater, surface soil, or sediment samples to confirm exposure has stopped. The past barium ingestion doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in nearly all studies we reviewed. In the one human study available, the barium ingestion doses we estimated for all ages were close to the "no observed adverse effects level" (NOAEL) for circulatory system effects presented in the study. However, this study is limited by a small sample size and some flaws in its methodology. There are no other human studies available for review or confirmation of the NOAEL value. Similarly, the barium ingestion doses we estimated for all age groups were much smaller than the levels associated with circulatory system effects in most animal studies we reviewed. In one chronic ingestion study of rats, however, barium doses slightly larger than the past doses we estimated for all age groups have been associated with increased blood pressure (ATSDR 1992b). It is not clear what either of these studies means for humans ingesting very low doses of barium. More studies examining the potential association between low-dose barium ingestion and circulatory system effects are needed before we can make reliable comparisons with our estimated doses.

Site-specific Cancer Risk - There are no studies of barium's likelihood to cause cancer in humans. In two animal studies, rats and mice exposed to barium in drinking water for a lifetime did not show an increased incidence of tumors. However, these studies

had design and methodology errors making them inadequate for determining if an association exists between barium ingestion and cancer (ATSDR 1992b).

Sensitive Populations - Because barium seems to be related to increased blood pressure, residents with hypertension or other heart problems could be at increased risk of becoming ill from exposure to barium. Similarly, since barium appears to interact with sodium pentobarbital and phenobarbital by enhancing these drugs' depressive effect on the heart, individuals on this medication may experience an increased risk of heart problems if exposed to barium. In addition, children may be at increased risk after exposure to barium since animal studies have shown a higher barium absorption rate in young animals than in older animals. One study indicated people who drink large quantities of milk, including children and pregnant women, may also have an increased barium absorption. Finally, people who smoke, have a history of lung disease, or take diuretics may be more susceptible to barium toxicity (ATSDR 1992b).

3. Benzene

Summary - The past and present benzene ingestion and modeled inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the human and animal studies we reviewed. It is not known if skin absorption of benzene is associated with internal health effects. We estimate the increased risk of digestive system cancer from past benzene ingestion to be low. Using modeled inhalation data, we estimate the increased risk of developing leukemia (blood cancer) from past benzene inhalation to be moderate if actual exposure conditions are close to the estimated conditions used in the model. We estimate the increased cancer risk from present-day benzene ingestion and inhalation to be negligible. There is not have enough information to estimate the increased cancer risk from past or present skin exposure to benzene.

Use and Human Exposure - Benzene is a colorless, flammable liquid with a sweet odor. Most benzene is made from petroleum sources, although small amounts occur naturally. Benzene is used to make other chemicals, rubber, lubricants, dyes, glues, paints, furniture wax, detergents, drugs, and pesticides. Most people are exposed to a small amount of benzene daily, mainly through breathing in tobacco smoke, gas station vapors, motor vehicle exhaust, industrial emissions, and household vapors from benzene-containing products. Benzene dissolves easily in water and leakage from gas stations, landfills, or hazardous waste sites containing benzene can contaminate well water. People using benzene-contaminated tap water can be exposed to benzene through drinking the water, eating foods prepared with the water, or breathing in benzene while bathing or cooking. A small amount of benzene can enter the body through skin contact with benzene-containing compounds. At high levels of air-borne benzene, about half of the inhaled benzene is subsequently exhaled, and the rest goes into the bloodstream. Ingested benzene also goes into the bloodstream. Once in the bloodstream, benzene travels throughout the body and can be temporarily

stored in bone marrow and fat. The bone marrow and liver change benzene into breakdown products. Most benzene breakdown products leave the body within two days, but some remain in the body longer. Several of benzene's harmful effects are believed to be caused by these breakdown products in the body (ATSDR 1993e).

General Health Effects - The potential health effects of benzene depend upon the exposure amount and length. Most information on the health effects of long-term benzene exposure are from studies of industrial workers exposed to very high levels in air. Exposure to high benzene levels in air can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. In most cases, these symptoms stop once exposure ends and the individual breathes fresh air. However, people who breathe benzene for a long period of time may experience harmful effects in the tissues that form blood cells. These effects can disrupt normal blood production and cause a decrease in important blood components. Eventually, this disruption may lead to anemia (a decrease in the number of red blood cells) or excessive bleeding (caused by a decrease in the number of clotting components in blood). Exposure to benzene in air can also harm the immune system and has been linked with damage to chromosomes, the parts of cells responsible for hereditary characteristics. Exposure to air-borne benzene may also harm the reproductive organs and, in animals, has been associated with adverse effects on unborn babies. Long-term exposure to relatively high benzene in the air levels has been linked to leukemia (blood cancer). Eating or drinking high amounts of benzene can cause vomiting, stomach irritation, dizziness, sleepiness, convulsions, rapid heart rate, coma, and death. The health effects of ingesting lower levels of benzene are unknown. Benzene irritates the skin and can cause redness and sores. Benzene is classified as a known cancer-causing agent in humans via ingestion and inhalation (ATSDR 1993a, 1993e).

Interactions with Other Chemicals - Benzene metabolism is complex, and agents that alter benzene metabolism may also alter benzene toxicity. In animals, ethanol (drinking alcohol) enhances both the metabolism and toxicity of benzene, particularly the toxic effects on blood. Likewise, benzene can interfere with ethanol metabolism, thereby increasing ethanol-induced effects on the brain. In rats and mice, treatment with the prescription drugs containing phenobarbital before exposure to very high levels of benzene in air increases their benzene metabolism. In contrast, experiments with rat cells grown in an artificial environment and pretreated with phenobarbital show no metabolic effects at lower benzene levels. Coadministration of toluene with benzene inhibits the metabolism of benzene in rats, but pretreatment with phenobarbital alleviates toluene's suppressive effect on benzene (ATSDR 1993e).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to benzene through incidental ingestion of on-site subsurface soils and household uses of well water. Present-day groundwater analyses indicate exposure is continuing for residents still using private well water. In addition, the air stripper's trial run demonstrated this device will successfully remove benzene from groundwater and

expel it into the air. We used modeled data to estimate past inhalation exposure to benzene volatilized in the shower and present in ambient air. The past and present benzene ingestion and modeled inhalation doses we estimated for all age groups are much smaller than the levels associated with noncancer illness in the human and animal studies we reviewed. Benzene is a known skin irritant, but there are no studies of benzene's effects on internal body systems resulting from skin absorption (ATSDR 1993e).

Site-specific Cancer Risk - There are no human studies examining benzene's cancer-causing potential from chronic oral exposure. However, animal studies indicate chronic oral exposure at high doses can cause cancer in various parts of the digestive system, particularly the mouth (ATSDR 1993e). EPA, NTP, and IARC each have classified benzene as a known human cancer-causing agent via ingestion (ATSDR 1993a). Based on the exposure and dose estimates we have, we estimate the increased cancer risk from past benzene ingestion to be low at 1 in 10,000. This means the risk of developing cancer, above the background rate, could rise from 2,500 cases per 10,000 people to 2,501 cases in a 70-year lifetime. We estimate the present-day increased cancer risk from oral exposure to be negligible.

In addition, EPA, NTP, and IARC each have classified benzene as a known cancer-causing agent via inhalation (ATSDR 1993a). Chronic inhalation of benzene can cause leukemia in humans (ATSDR 1993e). Since we did not have actual measurements of past benzene concentrations in air, we used known groundwater concentrations to estimate the cancer risk from inhaling benzene vapors in the shower and in the ambient air inside and outside the home. Based on the modeled exposure and dose information we have, we estimate the increased cancer risk from past benzene inhalation to be moderate at 12 in 10,000. This means the risk of developing cancer, above the background rate, could rise from 2,500 cases per 10,000 people to 2,512 cases in a 70-year lifetime if tbe actual inhalation exposure conditions were similar to that predicted by the model. We estimate the present-day increased cancer risk from benzene inhalation to be negligible.

There are no human studies of benzene's cancer-causing potential from skin exposure. One animal study found benzene did not induce skin tumors in mice after intermediate or chronic exposure lengths, but the study's authors concluded mouse skin may not be the best study system for this experiment. Therefore, it is not known if skin exposure to benzene is associated with cancer (ATSDR 1993e).

Sensitive Populations - People who drink alcohol and are exposed to benzene may be more susceptible to the benzene's toxic effects on blood. In addition, individuals with viral hepatitis may have accelerated occurrences of aplastic anemia (low red blood cell count). Similarly, individuals with thalassemia (abnormal blood hemoglobin) may experience an increase in the harmful effects of benzene on the blood. Finally, children and unborn babies may also be more susceptible to benzene's harmful effects

because their blood cell populations are growing, and rapidly dividing cells are at greater risk than slowly dividing cells (ATSDR 1993e).

4. Bromodichloromethane

Summary - Past and present bromodichloromethane ingestion is unlikely to be associated with noncancer illnesses. There is not enough toxicological information to determine if noncancer illnesses have been associated with bromodichloromethane inhalation or skin absorption doses similar to those we estimated for all age groups. We estimate the increased cancer risk from past or present bromodichloromethane ingestion to be negligible. There is not enough toxicological information to determine if past or present inhalation or skin absorption could be associated with cancer.

Use and Human Exposure - Bromodichloromethane is a colorless liquid produced for laboratory use and chemical manufacturing. Most bromodichloromethane found in the environment is formed as an unwanted byproduct of chlorinating drinking water. Bromodichloromethane in water or air slowly breaks down into other substances. Most people are exposed to bromodichloromethane through drinking chlorinated water or swimming in chlorinated swimming pools. Small amounts can occur in foods made with chlorinated water such as ice cream and soft drinks. Almost all ingested bromodichloromethane moves from the digestive system into the blood. Because bromodichloromethane evaporates easily, people can inhale its vapors from household water, swimming pools, and hazardous waste sites. In addition, bromodichloromethane can cross the skin and people may be exposed while showering, swimming, or touching soil containing this compound. Once in the body, most bromodichloromethane rapidly leaves the body through exhalation, but small amounts are excreted in urine and feces. Little builds up in the body (ATSDR 1989a).

General Health Effects - In animal studies, eating or drinking large amounts of this compound is associated with injury to the liver and kidneys, and with adverse affects on the brain leading to incoordination and sleepiness. In addition, there is some animal evidence high doses of bromodichloromethane may adversely affect unborn babies (ATSDR 1989a). Bromodichloromethane is classified as a suspected cancer-causing agent in humans via ingestion and inhalation (ATSDR 1993a).

Interactions with Other Chemicals - In rats, pretreatment with oral doses of acetone dramatically increases ingested bromodichloromethane's toxic effects on the liver and kidneys (ATSDR 1989a).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to bromodichloromethane through household uses of well water. Present-day groundwater analyses indicate exposure is continuing for residents still using private well water. In addition, nearby residents may have been coexposed to acetone and

bromodichloromethane in their drinking water in the past, and may be exposed to acetone in the air and bromodichloromethane in the drinking water in the present and future. Still, the past and present bromodichloromethane ingestion doses we estimated for all age groups are much smaller than EPA's RfD (IRIS 1994), indicating noncancer illnesses are unlikely to be associated with this exposure, even if interactions with acetone occur. There are no human or animal studies of the health effects from bromodichloromethane inhalation or skin absorption (ATSDR 1989a).

Site-specific Cancer Risk - There are no human studies specifically examining bromodichloromethane's cancer-causing potential from chronic exposure. However, animal studies indicate chronic oral exposure to high doses of bromodichloromethane is associated with increased incidences of liver and kidney tumors (ATSDR 1989a). EPA and NTP each classified bromodichloromethane as a suspected cancer-causing agent in humans via ingestion (ATSDR 1993a). Based on the exposure and dose information we have, we estimate the increased cancer risk from past or present bromodichloromethane ingestion to be negligible. NTP has also classified bromodichloromethane as a suspected cancer-causing agent via inhalation. However, there are no studies examining the potential association between bromodichloromethane inhalation or skin absorption and cancer (ATSDR 1989a).

Sensitive Populations - ATSDR's toxicological profile for bromodichloromethane did not cite any studies concerning groups of people that were unusually sensitive to this compound. However, because bromodichloromethane exposure is associated with adverse effects on the kidneys and liver, people with pre-existing kidney or liver disease may be unusually sensitive to this compound (ATSDR 1989a).

5. Cadmium

Summary - Cadmium ingestion doses much smaller than the past doses we estimated for all age groups have been associated with excreting abnormal amounts of protein in the urine, a symptom suggestive of mild kidney tubule dysfunction, in the human studies we reviewed. Kidney tubule dysfunction can result in a secondary loss of calcium which, in turn, may be associated with a variety of bone disorders. In a few animal studies, cadmium ingestion doses similar to past doses we estimated for all age groups have been associated with high blood pressure, but is not clear if cadmium exposure affects human blood pressure. In one animal study, a cadmium ingestion dose similar to the past dose we estimated for adults has been associated with effects on the nervous system of unborn baby rats. However, it is uncertain if maternal cadmium exposure has effects on unborn human babies. It is not known if oral exposure to cadmium causes cancer.

Use and Human Exposure - Cadmium is a soft, silvery white metal that occurs naturally in the earth's crust. It is found in all soils and rocks, including coal and mineral fertilizers, and is commonly present as small particles in air. Cadmium is

found in many industrial and consumer product uses, including batteries, pigments, metal coatings, and plastics. People can breathe in air-borne cadmium from industrial sources, the burning of coal or household wastes, and smoking tobacco. Workers who solder or weld metal may also be exposed to air-borne cadmium. People may also be exposed to cadmium through ingestion. Cadmium can enter drinking water supplies from disposal of household or industrial wastewater, use of fertilizers, or leaks from hazardous waste sites containing cadmium. Cadmium is commonly found in food, and most people ingest small amounts of cadmium daily from the things they eat. Very little cadmium enters the body through the skin. The body quickly absorbs about 25% of inhaled cadmium and 5% of ingested cadmium. Once in the body, cadmium stays in the liver and kidneys. Most of this cadmium is in a form that is not harmful, but too much cadmium can overload the kidneys' storage system and harm human health. Cadmium slowly leaves the body through urine and feces (ATSDR 1993g).

General Health Effects - Cadmium is not known to have any beneficial health effects. Breathing very high levels of cadmium can cause severe lung damage and death. Breathing lower levels of cadmium for years can cause kidney disease, lung damage, and fragile bones. Workers who inhale cadmium over a long period of time have an increased risk of getting lung cancer. Eating very high cadmium levels severely irritates the stomach. Eating lower levels for a long time period is associated with kidney damage and fragile bones. It is not known if breathing or eating cadmium adversely affects reproduction or the development of unborn children; however, cadmium exposure is associated with these effects in laboratory rats. Similarly, it is not known if eating or breathing cadmium harms the liver, heart, nervous system, or immune system of humans. Cadmium is classified as a suspected cancer-causing agent in humans via inhalation. (ATSDR 1993a, 1993g).

Interactions with Other Chemicals - Oral cadmium toxicity can be influenced by a wide variety of substances. In humans, dietary deficiencies of calcium, protein, and vitamin D likely account for cadmium's effects on bone. Similarly, iron deficiency increases cadmium's absorption from the digestive system. In quail, cadmium toxicity is intensified by zinc, copper, iron, calcium, and protein deficiencies. In other animal experiments, dietary calcium deficiencies aggravate cadmium's toxic effects on the immune system and on fetuses. In rats, coexposure of cadmium and ethanol (drinking alcohol) in a liquid diet produces liver damage at doses that are not toxic by themselves. Simultaneous administration of garlic decreases cadmium's toxic effects on rat kidneys, but pretreatment with drugs containing acetaminophen increases rat sensitivities to these effects. Coadministration of cadmium and lead in rat diets has an additive effect on reducing body weight, but an antagonistic effect on nervous system toxicity. Coexposure with selenium reduced cadmium's effects on mouse bone marrow (ATSDR 1993g).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to cadmium through incidental ingestion of on-site subsurface soils and ingestion of groundwater. The limited number of present-day samples indicate cadmium is no longer found in the groundwater, but we do not have enough groundwater, surface soil, or sediment samples to confirm exposure has stopped. In one human study, cadmium ingestion doses much smaller than the past doses we estimated for all age groups have been associated with excess protein in the urine, a symptom suggestive of mild kidney tubule dysfunction. Findings in animal studies support the probable existence of an association between cadmium exposure and the functioning of the kidney tubules. In addition, human and animal data indicate excess protein in the urine can develop only after a specific threshold of cadmium in the kidney is exceeded. Because small amounts of cadmium are normally present in the American diet and in tobacco smoke, people may not have a large margin of safety with respect to cadmium intake from other sources. Two studies indicate having excess protein in the urine may not decrease when cadmium exposure stops; rather, kidney tubule dysfunction and decreased filtration may continue to increase in severity. Moreover, there is some evidence cadmium exposure may affect kidney vitamin D metabolism with subsequent disturbances in calcium balance and bone density. Increased calcium excretion may increase the risk of osteoporosis, particularly in post-menopausal women. In addition, bone disorders such as osteomalacia (softening of the bones) and spontaneous bone fracture have been observed in some humans chronically exposed to unspecified amounts of cadmium in their diets (ATSDR 1993g).

In a few animal studies, cadmium ingestion doses similar to past doses we estimated for all age groups have been associated with high blood pressure. However, cadmium's potential toxic effects on the human circulatory system are not clear, and, after several human studies, it is still unknown if cadmium exposure adversely affects human blood pressure. In addition, there is evidence cadmium exposure can affect the development of unborn babies of animals. In one animal study, a cadmium ingestion dose similar to the past dose we estimated for adults has been associated with pregnant rats having babies with reduced body movement ability and impaired balance. Cadmium's potential effects on the development of human babies is uncertain (ATSDR 1993g).

Site-specific Cancer Risk - Although there is strong evidence that breathing cadmium dust for prolonged time periods can cause lung cancer in rats, the human evidence of cadmium's cancer-causing potential from ingestion is more limited. Neither human nor animal data provide sufficient evidence to determine if cadmium ingestion is associated with cancer (ATSDR 1993g).

Sensitive Populations - ATSDR's toxicological profile for cadmium did not cite any studies concerning groups of people that were unusually sensitive to this element. Still, based on what is known about cadmium toxicity, people with depleted stores of calcium, iron, or other dietary components are likely to have an increased cadmium

absorption from the digestive system. Likewise, people with kidney damage are likely to experience toxic effects on the kidneys at lower exposures than the general population (ATSDR 1993g).

6. Chromium(VI)

Summary - Direct skin contact with chromium containing compounds can elicit an allergic response in humans, characterized by eczema and dermatitis. Chromium ingestion doses similar to past doses we estimated for all age groups have been associated with a worsening of chromium-induced dermatitis in sensitized individuals in a couple of the human studies we reviewed. There is not enough information to estimate an increased cancer risk from chromium ingestion.

Use and Human Exposure - Chromium is a naturally occurring element found in rocks, soil, plants, and animals. It is present in the environment in several different forms. Chromium(III) occurs naturally in many fresh vegetables, fruits, meat, yeast, and grain. It is an essential nutrient required by the body for the metabolism of sugars, fats, and proteins. Chromium (III) is also used as brick lining for high temperature industrial furnaces. Chromium(0) and chromium(VI) are usually produced by industrial processes. Chromium(0) is used for making steel and other alloys. Both chromium(III) and chromium(VI) are used for plating, manufacturing dyes and pigments, tanning leather, and preserving wood. Smaller amounts are found in drilling muds, rust and corrosion inhibitors, textiles, and copy machine toner. People can breathe in air-borne chromium from industrial sources and tobacco. However, most people are exposed to chromium in their diets. Chromium is not only found in many fresh foods, but is also present in steel and can leach out of stainless steel cans containing acidic foods. Chromium can enter drinking water supplies from hazardous waste sites containing chromium. Very little chromium enters the body through the skin. Inhaled chromium particles are either coughed up and swallowed or slowly absorbed from the lungs into the bloodstream. Only a small amount of ingested chromium is absorbed from the intestines into the bloodstream; most leaves the body through the feces. The small amount of chromium that does enter the bloodstream is distributed throughout the body where it is used to carry out essential functions. Chromium then passes through the kidneys and is eliminated in the urine within a few days (ATSDR 1993i).

General Health Effects - Breathing in large amounts of chromium can irritate the nose, and chronic exposure to very high amounts of chromium has been associated with lung cancer. Breathing in small amounts of chromium(VI) for short or long time periods does not seem to be associated with harmful effects. Ingesting small amounts of chromium(III) is essential for good nutrition, and ingesting small amounts of chromium(VI) does not seem to be harmful; however, ingesting large amount of either chromium(III) or chromium(VI) may cause health problems. Swallowing large amounts of chromium(VI) may cause upset stomachs and ulcers, convulsions, and

kidney or liver damage. Some people are very sensitive to chromium(III) or chromium(VI), and may develop an allergic reaction characterized by redness and swelling of the skin. Not much is known about the health effects of chromium(0). There is no reliable information about chromium's effects on reproduction or unborn babies. Chromium is classified as a known cancer-causing agent in humans via ingestion and inhalation. (ATSDR 1993a, 1993i).

Interactions with Other Chemicals - There are a few animal studies of chromium's interaction with other chemicals in rats. One study indicates chromium potentiates mercury's toxic effects on the kidneys. Another study suggests oral ingestion of both selenium and chromium has serious adverse effects on the liver, but these effects could be due solely to chromium, a possibility the study did not investigate. Vitamin C seems to have a protective effect against skin ulcerations produced from skin exposure to a chromium-containing compound, and against toxic kidney effects produced by ingesting this same compound. Vitamin E protected against, while vitamin B_2 enhanced, chromium's toxicity to hamster cells grown in an artificial environment (ATSDR 1993i).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to chromium through incidental ingestion of on-site subsurface soils, on- and off-site sediments, off-site surface water, and ingestion of groundwater. The limited number of present-day samples indicate chromium is no longer found in the groundwater, but we do not have enough groundwater, surface soil, or sediment samples to confirm exposure has stopped. Direct skin contact with chromium-containing compounds can elicit an allergic response in humans, characterized by eczema and dermatitis in sensitized individuals. In a couple of human studies, chromium ingestion doses similar to past doses we estimated for all age groups have been associated with a worsening of chromium-induced dermatitis in sensitized individuals. The study of the interaction between chromium and selenium is inconclusive (ATSDR 1993i); therefore, we cannot evaluate selenium's potential effect on oral exposure to chromium.

Site-specific Cancer Risk - Although there is sufficient human epidemiological and animal evidence that inhalation of chromium(VI) compounds can cause lung and nasal cancer, human evidence of chromium's cancer-causing potential from ingestion is more limited. A retrospective mortality study in China indicated increased incidences of stomach and lung cancer in people living near a chromium smelting plant, but the residents were likely exposed through air, drinking water, food and soil. Therefore, it is not known if these effects could be caused by chromium(VI) ingestion alone. There is no evidence of chromium(VI) causing cancer in mice chronically exposed to chromium in drinking water or in rats chronically exposed to chromium in their food (ATSDR 1993i). Nevertheless, NTP has classified chromium(VI) as a known human cancer-causing agent via ingestion (ATSDR 1993a). However, EPA has not classified chromium(VI) as a cancer-causing agent; consequently, the agency has not derived

the toxicity values needed to estimate an increased cancer risk from chromium(VI) ingestion (ATSDR 1993a; IRIS 1994).

Sensitive Populations - Acute inhalation studies and some oral and skin absorption studies suggest female animals are more sensitive than males to the lethal effects of chromium(VI) compounds. However, it is not known if human females are more sensitive than males to chromium's toxic effects. Some individuals have less ability than others to reduce chromium(VI) to chromium(III) in their bloodstream and are more likely to become ill from chromium exposure. This ability to reduce chromium(VI) in the bloodstream may be related to vitamin C levels in plasma. Limited findings in human and animal studies suggest youths may be more susceptible to chromium's toxic effects than adults (ATSDR 1993i).

7. Cresol

Summary - The past cresol ingestion doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the animal studies we reviewed. There is not enough toxicological information to determine if noncancer illnesses have been associated with cresol inhalation or skin absorption doses similar to those we estimated for all age groups. Cresols are suspected cancer tumor promoters, but there is not enough toxicological information to determine an increased cancer risk from exposure to these compounds.

Use and Human Exposure - Pure cresols are colorless chemicals that occur in three different forms: o-cresol, m-cresol, and p-cresol. Cresols have a medicinal smell and can give water a medicinal smell and taste. Cresols are naturally present in many foods, and are also found in human and animal urine, wood and tobacco smoke, crude oil, and coal tar. In addition, cresols are used in human-made products such as wood preservatives (such as creosote and cresylic acid), disinfectants, and deodorants. Cresols do not evaporate quickly from surface waters, but can be quickly removed from rivers and lakes by bacteria. However, cresols dissolved in groundwater can persist for months without changing. People can be exposed to cresols by breathing air, drinking water and eating foods containing these compounds. The main sources of cresol in air are emissions from motor vehicles, in homes heated with coal or wood, from factories burning trash or garbage, and from industrial smokestacks. Cresols in the air quickly break down into smaller chemicals, some of which irritate the eyes. People can ingest cresols found in foods such as tomatoes, ketchup, asparagus, cheeses, butter, bacon, smoked foods, coffee, black tea, wine, whiskey, brandy, and rum. People who live near garbage dumps or hazardous waste sites may have large amounts of cresols in their water. Cresols can also be formed in the body from other compounds such as toluene and the amino acid tyrosine, a component of most proteins. Most of the cresols that enter the body are quickly changed to other substances and leave the body through urination within a day (ATSDR 1992d).

General Health Effects - Ingestion of high levels of cresol can cause a burning feeling in the throat or mouth as well as stomach pains. Skin contact with high concentrations of cresol may result in a rash, severe skin irritation, or a chemical burn. Ingestion of or skin contact with high cresol levels can result in anemia, kidney problems, unconsciousness, or even death. Long-term exposure to low doses in humans may be associated with anemia and kidney problems. In animals, low levels of cresol have been associated with loss of coordination and muscle twitching; it is not known if these effects occur in humans. Animal studies suggest cresols probably are not associated with reproductive problems or birth defects in humans. There is animal evidence that cresols may enhance the cancer-causing ability of other chemicals to produce tumors in animals, but cresols themselves have not been found to cause cancer in humans or animals. Still, cresols are classified as suspected cancer-causing agents in humans (ATSDR 1992d).

Interactions with Other Chemicals - Cresols may promote tumor development after tumor initiation by other chemicals. Although no evidence is available, it seems likely cresols could interact with phenols to affect the central nervous system, and on red blood cells to produce methemoglobinemia (the presence of an altered form of hemoglobin in the blood that cannot deliver oxygen to body tissues) (ATSDR 1992d).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to cresols through incidental ingestion of on- and off-site sediments and ingestion of groundwater. There are no present-day analyses of cresols; therefore, we do not know if exposure has stopped because we do not have groundwater, surface soil, sediment, or air samples to evaluate. The past cresol ingestion doses we estimated for all age groups are much smaller than the levels associated with noncancer illnesses in the animal studies we reviewed. Human ingestion studies were not available (ATSDR 1992d). We used modeled data to estimate past inhalation exposure to cresols volatilized in the shower and present in ambient air. However, studies of cresols' toxicity via inhalation were not sufficiently detailed to make reliable comparisons with our inhalation dose estimates. Finally, the past doses we estimated for cresol skin absorption while showering are much smaller than the doses associated with noncancer illnesses in the human and animal studies we reviewed. However, all of these studies used high cresol doses; the potential human health effects associated with chronic, low-dose skin exposure to cresol are not known (ATSDR 1992d). More studies examining the potential health effects of low-dose skin exposure to cresol are needed before we can make reliable comparisons with our estimated doses.

Site-specific Cancer Risk - There have been no human studies of cresols' ability to cause cancer in humans. In one animal study, hamsters exposed to p-cresol in their feed had an increased incidence of nontumor stomach cells, suggesting p-cresol may act as a promoter of stomach tumors in hamsters. A similar study on rats had negative results, but rats may be less sensitive to stomach tumor initiators than other animals. Cresols' tumor promotion potential has also been studied in mice. In one

study, mice were first given one skin application of a known tumor initiator, followed by application of all three forms of cresol. This resulted in increased numbers of skin growths that had the potential to develop into cancer. Still, researchers did not observe cancers developing from these growths. Based on this latter study, EPA has classified all three forms of cresol as possible human cancer-causing agents (ATSDR 1993f). However, EPA has not derived the toxicity values needed to estimate an increased cancer risk from cresol exposure (ATSDR 1993a; IRIS 1994).

Sensitive Populations - There is some evidence individuals with glucose-6-phosphate dehydrogenase deficiency may have increased susceptibility to cresols' effects on the blood. Infants may also be unusually susceptible to the effects of cresols. Furthermore, people with immune deficiencies might be unusually susceptible to the apparent cancer promotional effects of cresols. In addition, individuals with seizure disorders might be more vulnerable to cresols' effects on the central nervous system, such as convulsions and coma, than other people.

8. 1,1-Dichloroethane

Summary - The past and present 1,1-dichloroethane ingestion doses we estimated for all age groups are much smaller than the EPA's 1989 RfD. The modeled past and present 1,1-dichloroethane inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the human and animal studies we reviewed. There is not enough toxicological information to determine if noncancer illnesses have been associated with 1,1-dichloroethane skin absorption doses similar to those we estimated for all age groups. Studies pertaining to 1,1-dichloroethane's cancer-causing potential from ingestion are inconclusive. There is not enough toxicological information to determine if 1,1-dichloroethane inhalation or skin absorption could be associated with cancer.

Use and Human Exposure - 1,1-Dichloroethane is a colorless, oily liquid having an ether-like odor. It is used to make other chemicals and to dissolve other substances such as paint and varnish, and to remove grease. In the past, this chemical was used as a surgical anesthetic, but it is no longer used for this purpose. Because 1,1-dichloroethane evaporates easily into air, it is usually present in the environment as a vapor rather than a liquid. These vapors can be broken down by sunlight. Although 1,1-dichloroethane does not easily dissolve easily in water, small amounts of this compound may be found in water. In soil, 1,1-dichloroethane tends to either evaporate into the air or move into the groundwater. Most people are exposed to 1,1-dichloroethane by breathing air containing its vapors or drinking water contaminated with this compound. Inhaled or ingested I,1-dichloroethane is believed to enter the body rapidly. Animal studies indicate 1,1-dichloroethane can also enter the body through the skin. Once in the body, 1,1-dichloroethane may go to many organs. Animal experiments indicate most 1,1-dichloroethane entering the body is removed unchanged within a couple of days by exhaling. The small portion remaining in the

body is broken down, and the breakdown products quickly leave the body through exhalation and urination (ATSDR 1990c).

General Health Effects - There is no reliable information on how 1,1-dichloroethane affects human health. One study of cats found 1,1-dichloroethane is associated with kidney disease after long-term, high-dose exposure in air. Comparable effects have not been found in other animals tested, suggesting cats may be uniquely sensitive to this compound. Similarly, delayed growth in offspring of mother rats inhaling high concentrations of 1,1-dichloroethane during pregnancy; still, the study indicates humans are unlikely to experience adverse developmental effects after low-level exposure to this compound. The evidence pertaining to 1,1-dichloroethane's cancer-causing potential is inconclusive (ATSDR 1990c).

Interactions with Other Chemicals - Some evidence exists suggesting that 1,1-dichloroethane be enhanced by chlorinated hydrocarbons and acetaminophen. In addition, ethanol (drinking alcohol) increases the breakdown of 1,1-dichloroethane in the body and might affect its toxicity (ATSDR 1990c).

Site-specific Noncancer Health Effects - Nearby residents were exposed to 1,1-dichloroethane through household uses of well water. Present-day groundwater analyses indicate exposure is continuing for residents still using private well water. In addition, the air stripper's trial run demonstrated this device will successfully remove 1,1-dichloroethane from groundwater and expel it into the air. The past and present 1,1-dichloroethane ingestion doses we estimated for all age groups are much smaller than the EPA's 1989 RfD, currently under review (IRIS 1994; Risk*Assistant 1993). The modeled past and present 1,1-dichloroethane inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the human and animal studies we reviewed (ATSDR 1990c). These results indicate noncancer illnesses are unlikely to be associated with ingestion or inhalation exposure. There are no human or animal studies of the potential health effects from skin absorption of 1,1-dichloroethane (ATSDR 1990c); consequently, we cannot evaluate any potential association between this exposure route and noncancer illnesses.

Site-specific Cancer Risk - There are no human studies examining 1,1-dichloroethane's cancer-causing potential in humans. However, one animal study with rats suggests ingestion of very high doses of 1,1-dichloroethane may be associated with cancer, but this study had several flaws making the results questionable. Another study with mice and a different study with rat liver cells indicate 1,1-dichloroethane is not carcinogenic, but neither of these studies is conclusive. This limited evidence neither confirms nor dispels the cancer-causing potential of 1,1-dichloroethane. There are no studies examining the potential association between 1,1-dichloroethane inhalation or skin absorption and cancer (ATSDR 1990c).

Sensitive Populations - ATSDR's toxicological profile for 1,1-dichloroethane did not cite any studies concerning groups of people that were unusually sensitive to this compound (ATSDR 1990c).

9. 1,2-Dichloroethane

Summary - The past and present 1,2-dichloroethane ingestion doses we estimated for all age groups are much smaller than the levels associated with noncancer illnesses in the animal studies we reviewed. 1,2-Dichloroethane inhalation doses similar to modeled doses we estimated for all ages have been associated with suppressed immune response in one animal study, but similar associations have not been found in other animal species. It is not known if 1,2-dichloroethane inhalation is associated with adverse effects on the human immune system. It is not known if skin contact with 1,2-dichloroethane is associated with changes in the skin or with noncancer illnesses in humans. There is no apparent increased risk of cancer from past 1,2-dichloroethane ingestion. Using modeled inhalation data, we estimate the increased risk of developing cancer from 1,2 -dichloroethane inhalation to be low if actual exposure conditions are close to the estimated conditions used in the model. We estimate the increased cancer risk from present-day 1,2-dichloroethane ingestion and inhalation to be negligible. We did not have enough information to estimate an increased cancer risk from past or present skin exposure to 1,2-dichloroethane.

Use and Human Exposure - 1,2-Dichloroethane is a clear liquid with a sweet smell and taste that evaporates at room temperature. 1,2-Dichloroethane is used to make vinyl chloride and chemicals that dissolve grease, glue, and dirt. It is also added to gasoline to remove lead. In the past, 1,2-dichloroethane was a component of some cleaning solutions, pesticides, adhesives, paint, varnish, and finish removers. Because 1,2-dichloroethane evaporates easily into air, it is usually present in the environment as a vapor rather than a liquid. 1,2-Dichloroethane does not remain in the air for long because sunlight breaks it down. Small amounts of 1,2-dichloroethane can be found in water. In soil, 1,2-dichloroethane usually evaporates or travels downwards and enters groundwater. Small organisms in the soil and groundwater break down 1,2-dichloroethane very slowly. Most people are exposed to 1,2-dichloroethane by breathing air containing its vapors, drinking water contaminated with this compound, using old household products made with 1,2-dichloroethane, or coming in contact with gasoline or gasoline vapors. Animal studies show 1,2-dichloroethane may also enter the body through the skin. Animal experiments show 1,2-dichloroethane may go to many body organs after ingestion or inhalation. However, most 1,2-dichloroethane entering the body is removed by exhalation within two days. A small portion is broken down, and the breakdown products quickly leave the body through exhalation and urination (ATSDR 1992h).

General Health Effects - People who accidentally inhale or ingest large amounts of 1,2-dichloroethane can develop nervous system disorders and liver and kidney

disease. At very high levels, they can die of heart failure. The 1,2-dichloroethane levels causing these effects are unknown. In animals, inhalation or ingestion of large amounts of 1,2-dichloroethane is associated with nervous system disorders, kidney disease. Longer-term exposure to lower doses also is associated with kidney disease in animals. Furthermore, animal studies indicate exposure to high levels of 1,2-dichloroethane may reduce infection-fighting ability; however, there is no evidence 1,2-dichloroethane causes a similar immune reduction in humans. Animal studies indicate 1,2-dichloroethane does not affect reproduction or cause birth defects. So far, 1,2-dichloroethane has not been associated with cancer in humans. However, eating large doses of this chemical or having it applied to the skin has been linked with cancer in animals. Breathing 1,2-dichloroethane may also be linked with cancer in animals. Based on animal studies, 1,2-dichloroethane is classified as a suspected cancer-causing agent in humans via ingestion and inhalation (ATSDR 1992h, 1993a).

Interactions with Other Chemicals - There are several studies of 1,2-dichloroethane's interaction with other chemicals in animals. Prescription drugs containing phenobarbital increase the breakdown of 1,2-dichloroethane and may increase its toxicity. One study shows ethanol's (drinking alcohol) effects on metabolism depend on existing tissue concentration of ethanol at the time of 1,2-dichloroethane exposure. If tissue ethanol concentration is low, 1,2-dichloroethane break down increases; if tissue ethanol concentration is high, 1,2-dichloroethane break down decreases. These findings are important because changes in 1,2-dichloroethane breakdown rate can have broad effects on 1,2-dichloroethane toxicity. Another study showed chronic coexposure to ethanol in rats had no effect on 1,2-dichloroethane's breakdown or toxicity. Coadministration of disulfiram, a component of prescription drugs that treat alcoholism, in diet and 1,2-dichloroethane in air greatly increased 1,2-dichloroethane's liver toxicity, and increased the tissue growths in the liver, the testes, mammary glands, and skin that have the potential to cause cancer. Cotreatment with 1,2-dichloroethene had slightly greater than additive effects on the liver (ATSDR 1992h, 1993a).

Site-specific Noncancer Health Effects - Nearby residents were exposed to 1,2-dichloroethane through household uses of well water. Present-day groundwater analyses indicate exposure is continuing for residents still using private well water from areas next to the groundwater contaminant plume. In addition, the air stripper's trial run demonstrated this device will successfully remove 1,2-dichloroethane from groundwater and expel it into the air. For our health effects evaluation, we had only animal studies to review. The past and present 1,2-dichloroethane ingestion doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the animal studies we reviewed (ATSDR 1992h).

We used modeled data to estimate past inhalation exposure to 1,2-dichloroethane volatilized in the shower and present in ambient air. In one study of mice, 1,2-dichloroethane inhalation doses similar to the past doses we estimated for all age

groups have been associated with a decreased ability to fight infections after inhaling disease-causing microorganisms. This association has not been found in any of the inhalation studies with rats we examined. Human studies were not available for review (ATSDR 1992h), and it is not known if 1,2-dichloroethane is associated with adverse effects on the human immune system.

Eye contact with concentrated 1,2-dichloroethane vapors has been associated with eye irritation and clouding of the cornea in humans and animals. Similarly, direct skin contact with concentrated solutions of 1,2-dichloroethane has been associated with cellular changes in the skin of guinea pigs. Nevertheless, the potential human health effects associated with chronic, low-dose skin exposure to 1,2-dichloroethane are not known (ATSDR 1992h).

Site-specific Cancer Risk - There are no reliable human studies examining 1,2-dichloroethane's cancer-causing potential after chronic oral exposure. However, one animal study indicates chronic oral exposure at high doses may be associated with cancer in various parts of the body including the spleen, liver, pancreas, adrenal gland, stomach, breast, and lung (ATSDR 1992h). EPA, NTP, and IARC each have classified 1,2-dichloroethane as a probable human cancer-causing agent via ingestion of drinking water or soil (ATSDR 1993a). Based on the exposure and dose information we have, there is no apparent increased cancer risk from past 1,2-dichloroethane ingestion. We estimate the present-day increased cancer risk from 1,2-dichloroethane ingestion to be negligible.

Similarly, there is little evidence associating chronic inhalation of 1,2-dichloroethane with cancer in humans (ATSDR 1992h). Nevertheless, EPA, NTP, and IARC each have classified 1,2-dichloroethane as a probable human cancer-causing agent via inhalation (ATSDR 1993a) from the ingestion data (IRIS 1994). In two animal studies, chronic inhalation exposure in rats did not show cancer, but design errors in and limitations of these studies make the results indeterminate. Because we did not have actual measurements of past 1,2-dichloroethane concentrations in air, we used known groundwater concentratious to estimate the cancer risk from inhaling 1,2-dichloroethane vapors in the shower and in ambient air inside and outside the home. Based on the exposure and dose information we have from the model, we estimate the increased cancer risk from past 1,2-dichloroetbane inhalation to be low at 24 in 100,000. This means the risk of developing cancer, above the background rate, could rise from 25,000 cases per 100,000 people to 25,024 cases in a 70-year lifetime if the actual inhalation exposure was similar to that predicted by the model. We estimate the present-day increased cancer risk from 1,2-dichloroethane inhalation to be negligible.

There are no human studies examining 1,2-dichloroethane's cancer-causing potential from skin exposure. One animal study found chronic skin exposure to 1,2-dichloroethane significantly increased the incidence of benign lung tumors in mice.

This study provides supportive evidence that 1,2-dichloroethane causes cancer and can penetrate through the skin and move into the circulatory system (ATSDR 1992h). Nevertheless, it is not known if skin absorption of 1,2-dichloroethane is associated with an increased lung cancer risk in humans, and we did not have adequate information to estimate an increased cancer risk from skin exposure.

Sensitive Populations - People who drink alcohol or take prescription drugs containing phenobarbital or disulfiram may be unusually sensitive to the toxic effects of 1,2-dichloroethane. People who smoke or passively breathe cigarette smoke may be more susceptible to lung emphysema after repeated exposure to 1,2-dichloroethane than nonsmokers. In addition, people with impaired liver or immune function, or alcoholics may be unusually susceptible to the effects of 1,2-dichloroethane (ATSDR 1992h).

10. Di(2-ethylhexyl)phthalate

Summary - Past di(2-ethylhexyl)phthalate ingestion is unlikely to be associated with noncancer illnesses. There is not enough toxicological information to determine if noncancer illnesses have been associated with di(2-ethylhexyl)phthalate inhalation or skin absorption doses similar to those we estimated for all age groups. We estimate the increased cancer risk from past di(2-ethylhexyl)phthalate ingestion to be low. It is not known if di(2-ethylhexyl)phthalate inhalation or skin absorption is associated with cancer.

Use and Human Exposure - Di(2-ethylhexyl)phthalate is a colorless, odorless liquid added to many plastics to make them flexible. It is present in plastic products such as rainwear, footwear, upholstery materials, tablecloths, shower curtains, food packaging, floor tiles, toys, paints, flexible tubing, plastic bags, pesticides, and cosmetics. Di(2-ethylhexyl)phthalate is commonly present throughout the environment. It enters the environment from industrial releases and the burning of plastics. Over a long period of time, di(2-ethylhexyl)phthalate can also leach out of plastic products buried in the ground. Once in the environment, di(2-ethylhexyl)phthalate attaches strongly to soils and does not move far away from its release site. It does not break down very easily in deep soils or sediments. Small amounts of di(2-ethylhexyl)phthalate have been found in fish and other animals, and may be found in some plants. Most people are exposed to small amounts of di(2-ethylhexyl)phthalate daily in their food and drinking water. People can be exposed to elevated levels of di(2-ethylhexyl)phthalate in drinking water near landfills containing buried plastics, or in air from cities or industrial areas. People may also be exposed to this compound through medical procedures using plastic products. It is not known if di(2-ethylhexyl)phthalate can cross the skin; however, if transfer occurs, it is probably low. Once in the body, most di(2-ethylhexyl)phthalate is quickly broken down into products having toxicities similar to di(2-ethylhexyl)phthalate. These compounds travel through the blood to the liver, kidneys, and testes. Small amounts

are stored in fat or secreted in breast milk. Most of the di(2-ethylhexyl)phthalate and its breakdown products leave the body in the urine and feces within 24 hours (ATSDR 1993k).

General Health Effects - Most of what is known about di(2-ethylhexyl)phthalate comes from studies of rats and mice fed high doses of this compound. Because both rats and mice seem to be more sensitive than humans to the effects of di(2-ethylhexyl)phthalate exposure, it is difficult to predict human health effects from the animal study information. In rats and mice, short-term exposure to high levels of di(2-ethylhexyl)phthalate have been associated with problems in sperm formation. Exposure before puberty is associated with delayed sexual maturation and reversible changes in testicular structure in male rats. Nevertheless, long-term exposure to high di(2-ethylhexyl)phthalate doses is associated with decreased fertility of both male and female rats. However, these associations are not seen in nonrodent species, including monkeys and rabbits. In pregnant rats and mice, exposure to high levels of di(2-ethylhexyl)phthalate has been associated with offspring having low birth weight and survivorship, as well as malformations in the skeleton, heart, kidneys, brain, and blood vessels. It is not known if di(2-ethylhexyl)phthalate exposure is associated with similar effects in humans. In addition, long-term exposure of rats to di(2-ethylhexyl)phthalate is associated with structural and functional changes in the kidney. Such kidney changes are of special concern to humans because kidney dialysis procedures use flexible tubing containing di(2-ethylhexyl)phthalate. It is not known if di(2-ethylhexyl)phthalate causes cancer in humans; however, long-term exposure to high doses of this substance has been linked with liver cancer in rats and mice. Based on animal studies, di(2-ethylhexyl)phthalate is classified as a suspected cancer-causing agent in humans via ingestion (ATSDR 1993a, 1993k).

Interactions with Other Chemicals - There are limited data concerning di(2-ethylhexyl)phthalate's interaction with other chemicals in humans. One study suggests di(2-ethylhexyl)phthalate may interact with the β-adrenergic class of pharmaceutical drugs, although the nature of this interaction is not clear.

There are several studies of di(2-ethylhexyl)phthalate's interactive effects with other chemical compounds in rats. Di(2-ethylhexyl)phthalate seems to affect the rat liver's metabolism of ethanol (drinking alcohol), depending on the frequency of di(2-ethylhexyl)phthalate administration. A single dose of di(2-ethylhexyl)phthalate given to rats 18 hours prior to ethanol exposure seems to decrease ethanol metabolism, making ethanol's effects last longer. In contrast, when the same dose of di(2-ethylhexyl)phthalate is given to rats for seven days prior to ethanol exposure, ethanol metabolism rates increased. These differences may be related to the liver's compensation for certain di(2-ethylhexyl)phthalate breakdown products. In addition, high doses of di(2-ethylhexyl)phthalate appear to affect rat thyroid cell structure and decrease the circulating amounts of the thyroid hormone T_4. When large doses of di(2-ethylhexyl)phthalate are combined with dietary exposure to Aroclor-1254 (one of

the PCBs known to have similar effects on the thyroid), there is an apparent additive effect on the thyroid in changing the cell structure and in decreasing the circulating levels of thyroid hormones T_3 and T_4. However, at low di(2-ethylhexyl)phthalate doses combined with Aroclor-1254, these apparent additive effects are not seen. Finally, di(2-ethylhexyl)phthalate may interact with caffeine to adversely affect pregnancy and the development of unborn babies in rats. One study indicates a high dose of caffeine injected into pregnant rats with a very high dose of di(2-ethylhexyl)phthalate during the critical period of baby organ development can increase the numbers of dead or malformed offspring. All of these interactive studies with rats must be interpreted with great caution because rats are much more sensitive to the health effects of di(2-ethylhexyl)phthalate than humans. Although these studies suggest similar di(2-ethylhexyl)phthalate interactions are possible in humans, they do not indicate the likelihood of these interactions (ATSDR 1993k).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to di(2-ethylhexyl)phthalate through incidental ingestion of on-site subsurface soils and ingestion of groundwater. The limited number of present-day samples indicate di(2-ethylhexyl)phthalate is no longer found in the groundwater, but we do not have enough groundwater, surface soil, or sediment samples to confirm exposure has stopped. The past di(2-ethylhexyl)phthalate ingestion doses we estimated for all age groups are much smaller than ATSDR's acute MRL, somewhat smaller than ATSDR's intermediate MRL, and somewhat smaller than the doses associated with noncancer illnesses in the chronic animal studies we reviewed. This finding suggests noncancer illnesses are unlikely to be associated with ingestion of this contaminant. Similarly, the modeled past di(2-ethylhexyl)phthalate inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the few animal studies we reviewed. There were no human inhalation studies available for review (ATSDR 1993k), and more studies examining potential health effects from low-dose di(2-ethylhexyl)phthalate inhalation are needed before we can make reliable comparisons with our estimated doses. Similarly, there was only one skin absorption study available to review. The study results suggested di(2-ethylhexyl)phthalate may not be a skin irritant at very high doses. There were no studies of the potential internal effects from di(2-ethylhexyl)phthalate skin absorption (ATSDR 1993k). More studies examining the potential health effects of low-dose skin absorption are needed before we can make reliable comparisons with our estimated doses.

Site-specific Cancer Risk - There are no human studies concerning di(2-ethylhexyl)phthalate's ability to cause cancer. However, several chronic oral exposure studies in rats and mice indicate there may be a link between di(2-ethylhexyl)phthalate and liver cancer (ATSDR 1993k). After reviewing these studies, EPA and NTP each classified di(2-ethylhexyl)phthalate as a suspected cancer-causing agent in humans via ingestion of drinking water or soil (ATSDR 1993a). Based on the exposure and dose information we have, we estimate the increased cancer risk

from past di(2-ethylhexyl)phthalate ingestion to be low at 11 in 100,000. This means the risk of getting cancer, above the background rate, could rise from 25,000 cases per 100,000 people to 25,011 cases in a 70-year lifetime. There are no reliable studies associating di(2-ethylhexyl)phthalate inhalation or skin absorption with cancer.

Sensitive Populations - The very young and the elderly may have an increased susceptibility to the metabolic, reproductive, and nervous effects of di(2-ethylhexyl)phthalate if human response is similar to that in rats and mice. In addition, individuals with impaired liver function may be more sensitive to the effects of di(2-ethylhexyl)phthalate.

11. Hexachloroethane

Summary - Past hexachloroethane ingestion is unlikely to be associated with noncancer illnesses. There is not enough toxicological information to determine if noncancer illnesses may be associated with inhalation or skin absorption of this compound. We estimate the increased cancer risk from past hexachloroethane ingestion to be negligible. There is not enough toxicological information to determine if past hexachloroethane inhalation or skin absorption could be associated with cancer.

Use and Human Exposure - Hexachloroethane is a colorless crystal with a camphor-like odor. It is used as a solvent, as a retarding agent in fermentation, and in the synthesis of other chemicals. It is also found in pyrotechnics and smoke devices, and in explosives. People can be exposed to hexachloroethane through ingestion, inhalation, and skin absorption (Lewis 1993). It is not known how hexachloroethane is absorbed, broken down, or eliminated by the body.

General Health Effects - There are no studies of hexachloroethane's potential effects in humans. In animal studies, ingesting very high doses of this compound is associated with kidney damage, decreased litter size, and behavioral changes. In other animal studies, inhaling very large quantities of hexachloroethane is associated with behavioral changes in dogs and rats, and liver enlargement in the guinea pigs. Lower doses have not been associated with similar effects (IRIS 1994). Hexachloroethane's potential health effects via skin absorption are not known. Animal studies indicate inhalation of small amounts of hexachloroethane for long time periods is associated with liver and kidney cancer, but it is not known if hexachloroethane exposure is associated with these same cancers in humans (IRIS 1994).

Interactions with Other Chemicals - The potential interactive effects between hexachloroethane and other substances found at the site are unknown.

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to hexachloroethane through household uses of well water. There are no present-day

analyses of hexachloroethane; therefore, we do not know if exposure has stopped because we do not have groundwater, surface soil, sediment, or air samples to evaluate. The past hexachloroethane ingestion doses we estimated for all age groups are somewhat smaller than EPA's RfD indicating noncancer illnesses are unlikely to be associated with this exposure. In addition, the modeled hexachloroethane inhalation doses we estimated for all age groups are much smaller than the levels associated with noncancer illnesses in rats, dogs, or guinea pigs in the one animal study available (IRIS 1994). However, more studies examining potential health effects from low-dose hexachloroethane inhalation are needed before we can make reliable comparisons with our estimated doses. There are no available human or animal studies examining the potential health effects from skin absorption of hexachloroethane (IRIS 1994).

Site-specific Cancer Risk - There are no human studies concerning hexachloroethane's ability to cause cancer. However, one study of rats and mice indicates there may be a link between chronic ingestion of hexachloroethane and liver cancer in mice (IRIS 1994). EPA has classified hexachloroethane as a suspected cancer-causing agent in humans via ingestion (ATSDR 1993a). Based on the exposure and dose information we have, we estimate the increased cancer risk from past hexachloroethane ingestion to be negligible. In addition, EPA has also classified hexachloroethane as a suspected cancer-causing agent via inhalation, based upon the ingestion data. However, there are no human or animal studies examining the potential association between hexachloroethane inhalation or skin absorption and cancer (IRIS 1994).

Sensitive Populations - We did not find any studies or other documents identifying populations unusually sensitive to hexachloroethane.

12. Lead

Summary - Lead ingestion doses similar to past doses we estimated for all age groups have been associated with blood formation and blood pressure problems in the human and animal studies we reviewed. Young children appear to be more sensitive than adults to lead exposure. The present-day lead ingestion doses we estimated for young children are somewhat smaller than the doses associated with the blood problems in these studies. In animal studies, lead ingestion doses similar to past doses we estimated for all age groups have been associated with changes in the eye structures important in night vision, for young children have been associated with learning problems in young animals, and for adults have been associated with adverse effects on unborn baby animals and reproductive problems in adult animals. There is not enough information to estimate an increased cancer risk from lead ingestion.

Use and Human Exposure - Lead is a naturally occurring bluish-gray metal found in small quantities in the earth's crust. Most lead used by industry comes from mined ores or from recycled scrap metal. Lead is used to produce some types of batteries,

ammunition, and electronic devices. It is used as radiation shields (from x-rays, for example), and is found in sheet lead, solder, pipes, caulking, paints, ceramic glazes, and gasoline. In recent years, the amount of lead added to solder, paints, ceramic products, caulking, and gasoline has been reduced because of its harmful health effects; however, its use in ammunition and roofing has increased. Human activities, particularly the use of leaded gasoline, have spread lead to all parts of the environment (ATSDR 1993l).

People can be exposed to lead by breathing air, drinking water, eating foods, or ingesting dirt or dust containing lead. Foods such as fruits, vegetables, meats, grains, seafood, soft drinks, and wine may have lead in them. This lead can come from deposition of lead-containing dust on crops or during food processing, plant uptake of lead from soil, use of improperly glazed ceramics or leaded-crystal glassware, lead-soldered cans containing acidic foods, or lead-soldered kettles used to boil water. Communities with acidic water may have increased lead levels in water as the metal leaches out of lead pipes, lead-based solder, and brass faucets. Children can ingest lead-based paint chips. Lead enters the air from industrial releases, the weathering or burning of lead-based paints, or the burning of leaded gasoline, solid wastes, or tobacco. Consequently, tobacco smokers can be exposed to more lead than nonsmokers. Although skin contact with lead-containing dust and dirt occurs every day, not much lead passes through intact skin (ATSDR 1993l).

Most lead enters the body through ingestion. The amount of lead entering the body after ingestion depends upon when the last meal was eaten, as well as the person's age and how well the lead particles are dissolved in the stomach juices. Children tend to absorb more lead than adults, and more is absorbed from an empty stomach than from a full stomach. Frequent skin contact with lead in soil and dust can result in young children's swallowing high lead through hand-to-mouth behavior. In adults, only a small amount of lead can enter the body through intact skin if it is not washed off after skin contact. Lead can also enter the body through breathing in dust or chemicals containing lead, or through smoking tobacco products. Once in the body, lead first travels to body organs such as the liver, kidneys, lungs, brain, spleen, muscles, and heart. In adults, almost all of the lead entering the body leaves within a couple of weeks through urination or defecation. However, in children, only about a third of ingested lead leaves the body in waste. Lead that does not leave the body will, after several weeks, move to the bones and teeth where it can stay for decades. Some of the lead stored in bones and teeth may leave these tissues and reenter the blood and body organs at a later date. In adults, 94% of the total body lead is stored in bones and teeth. In children, only 73% is stored in bones and teeth; the rest is in body organs and blood.

General Health Effects - At high levels of exposure, lead can damage the brain or kidneys of adults or children. Unborn children are particularly sensitive to lead exposure during development. Exposure during pregnancy can lead to premature

birth, smaller babies, and decreased mental abilities in the infant. Young children are also more sensitive to lead exposure than are adults. Lead exposure can decrease IQ scores and reduce the growth of young children. These effects are more often seen after exposure to high lead levels rather than low lead levels. In adults, high levels of lead exposure may decrease reaction time; affect the memory; cause weakness in the fingers, wrists, or ankles; increase blood pressure in men; cause anemia; cause miscarriages; or damage the male reproductive system. It is not known if lead exposure causes cancer in humans. Some studies show rats and mice given very large doses of lead develop kidney tumors. However, the results of these animal studies are questionable because of the study methods used. Still, lead is classified as a suspected cancer-causing agent via ingestion (ATSDR 1993l).

Interactions with Other Chemicals - A number of studies of humans have found undernourished individuals are more susceptible to the effects of lead exposure because deficiencies in calcium, phosphorus, copper, iron, and zinc can increase lead absorption. Several animal studies have supported these findings by showing that sufficient dietary intake of calcium, magnesium, phosphorus, copper, iron, and zinc protects against the harmful effects of various lead compounds. A few animal studies show cadmium increases lead's toxic effects on mortality, behavior, and the male reproductive system. In addition, lead may worsen mercury's effects on the kidneys and liver. Another animal study indicates lead blocks intestinal responses to vitamin D and its by-products. In a different study, coexposure of lead and ethanol (drinking alcohol) in rats increased the rat's susceptibility to lead's toxic effects on the liver, brain, and nervous system. However, another study investigating the interactive effects of lead and ethanol during pregnancy found no interaction between these substances on reproduction or learning in rats (ATSDR 1993l).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to lead through incidental ingestion of on-site subsurface soils, incidental ingestion of on- and off-site sediments, incidental ingestion of off-site surface water, and ingestion of groundwater. Present-day groundwater analyses indicate exposure may be continuing for some residents still using private well water. In the human and animal studies we reviewed, lead ingestion doses similar to past doses we estimated for all age groups have been associated with increased blood pressure and adverse effects on blood formation that may lead to anemia and decreased blood hemoglobin formation (ATSDR 1993l). Some human evidence suggests males may be more likely to experience lead-induced blood pressure increases than females (Amdur et al. 1991; ATSDR 1993l). Lead ingestion doses somewhat larger than the present-day doses we estimated for young children have been associated with effects on blood formation and blood pressure in these studies (ATSDR 1993l).

In the animal studies we reviewed, lead ingestion doses slightly larger than the past doses we estimated for all age groups have been associated with changes in the rods of the eye (structures involved with night vision). Presumably, these changes could

lead to a decreased ability to see well at night. Visual effects associated with lead exposure have been noted in humans, but are not well documented. In addition, lead ingestion doses similar to past doses we estimated for young children have been associated with learning problems in animals. In some studies, low level lead exposure before or shortly after birth has been associated with learning impairments in young monkeys. Other studies indicated these learning problems may continue for months or years after exposure has stopped. Studies of rats also have found associations between low level lead exposure and learning impairment. In contrast, human studies of children exposed to low lead levels have mixed results. Some studies indicate low-level lead exposure may be associated with 4-5 point decreases in IQ scores, but a couple of studies show no association between learning and low-level lead exposure. Still, overall, the data suggest children are more sensitive than adults to low-level lead exposure, and animals are affected at roughly the same blood levels as humans (ATSDR 1993l).

Finally, lead ingestion doses similar to past doses we estimated for adults have been associated with adverse effects on reproduction and unborn babies in the animal studies we reviewed. In one study of female monkeys, chronic lead ingestion doses slightly larger than those estimated for adults have been associated with menstrual cycle irregularities and ovarian cyst development. Other studies of rats indicated an association between low level lead exposure before or shortly after birth and delayed sexual maturation in female offspring. In some male rats, exposure to low lead levels has been associated with decreased sperm counts, low sperm movement, increased prostate gland weight, and impotence. In humans, there are qualitative data indicating exposure to high lead levels adversely affects reproduction, but there are no data concerning the effects of low lead doses. Besides affecting reproduction, animal studies suggest low level lead exposure may be associated with blood formation problems in unborn babies, as well as the already mentioned learning behavior and sexual maturation problems after birth. There is no evidence low-level lead exposure is associated with body malformations (ATSDR 1993l).

The potential health effects of lead for adults need to be interpreted with caution. In estimating our doses, we used the maximum groundwater concentration which was found in an on-site borehole. The maximum concentrations found in on-site private wells was 10 times smaller and in off-site wells was 20 times smaller than the maximum borehole value. At either of these drinking water well concentrations, our projected blood lead concentrations for adults are below the blood lead levels associated with health effects in adults.

Site-specific Cancer Risk - There are no reliable studies available to evaluate lead's cancer-causing potential in humans. However, animal studies indicate very high doses of lead may be associated with kidney cancer (ATSDR 1993l). Based on the animal data, EPA, IARC, and NTP each have classified lead as a possible human cancer-causing agent via ingestion (ATSDR 1993a, 1993l). Nevertheless, limitations

in the animal studies do not permit derivation of the toxicity values needed to estimate an increased cancer risk due to lead ingestion (ATSDR 1993l).

Sensitive Populations - Preschool age children (under six years old), pregnant women, the elderly, smokers, alcoholics, and people with diseases affecting blood formation, nutrient uptake, and nerve or kidney function may be more susceptible to the toxic effects of lead exposure. Children are at the greatest risk for experiencing the toxic effects of lead exposure. Recent data suggest pregnant women, nursing mothers, and individuals with osteoporosis may have increased bone mobilization, resulting in increased levels of lead throughout the body. In addition, people with genetic diseases affecting the blood (such a thalassemia or sickle cell anemia), certain body enzymes, metabolic disorders (such as porphyria) may also be unusually sensitive to lead exposure.

13. Manganese

Summary - The manganese ingestion doses we estimated for adults and average children are much smaller than the doses associated with noncancer illnesses in the animal studies we reviewed. Young children may be more sensitive than adults to the effects of manganese exposure on the nervous system. Still, it is not clear if manganese ingestion doses similar to those we estimated for young children have been associated with biochemical changes in the nervous system. It is not known if manganese ingestion is associated with cancer.

Use and Human Exposure - Manganese is a naturally occurring metal found in rock and fossil fuels. Manganese compounds can exist in air as dust particles. Some manganese compounds can dissolve in water, and low levels of these compounds are normally present in lakes, streams, and the ocean. Manganese is also normally present in plants and animals. Manganese metal, once purified from mined rocks, is used to make various kinds of steel and some types of batteries. Manganese is also an ingredient in some ceramics, pesticides, fertilizers, and dietary supplements. Because manganese is commonly present in the environment, most people are exposed to small amounts of it daily in air, water, soil, and food. Food is the largest manganese source for most people. However, people living near coal- or oil-burning factories, or close to a major highway may breathe in higher than normal amounts of manganese. In addition, people living next to waste sites releasing manganese may also be exposed to unusually high amounts of this element in water or soil. Little manganese enters the body through intact skin. Inhaled manganese particles are coughed up and swallowed or slowly absorbed from the lungs into the bloodstream. Only a small amount of ingested manganese is absorbed from the intestines into the bloodstream; most leaves the body through the feces. Because manganese is a normal part of the body, the body ordinarily controls the amount of manganese that is absorbed and retained. Therefore, the total amount of manganese in the body tends to remain relatively constant, even if exposure rates are higher or lower than usual.

However, if too much manganese is taken in, the body may not be able to adjust for the added amount (ATSDR 1992f).

General Health Effects - Eating a normal diet seems to provide the required daily amount of manganese needed for good health, and there are no reported cases in humans of illnesses from eating too little manganese. However, eating too much manganese can cause serious illness. Miners and steel workers inhaling very high amounts of manganese dust for many months or years sometimes develop a disease called manganism. This disease is characterized by mental and emotional disturbances, and slow and clumsy body movements resulting from injury to the part of the brain that helps control body movements. It is not certain if eating or drinking too much manganese is associated with manganism. Impotence is also a common effect in men who breathe in very high amounts of manganese dust. Animal studies indicate too much manganese may be associated with harm to the testes. Not much is known about the effects of too much manganese in women. Animal studies suggest females may be less sensitive than males to the effects of manganese; however, this is not certain for humans. It is not known if too much manganese is associated with birth defects or cancer (ATSDR 1992f).

Interactions with Other Chemicals - Animal studies clearly show that intestinal absorption of manganese is inversely related to dietary iron intake. That is, high iron intake leads to decreased manganese absorption and toxicity, and low iron intake leads to increased manganese absorption and toxicity. Conversely, high dietary manganese leads to low iron absorption. Cadmium seems to have a similar inhibitory effect on the uptake of manganese. In addition, manganese appears to decrease cadmium's toxicity. There is limited animal evidence that ethanol (drinking alcohol) may increase human susceptibility to cadmium toxicity. There is also some animal evidence that chronic administration of prescription drugs containing chlorpromazine may increase manganese levels in the brain (ATSDR 1992f).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to manganese through incidental ingestion of on-site subsurface soils and sediments, incidental ingestion of on- and off-site surface water, and ingestion of groundwater. Low concentrations of manganese appear to be naturally present in the local groundwater, and present-day groundwater analyses indicate exposure is continuing for residents still using private well water. The past manganese ingestion doses we estimated for adults and average children are much smaller than the doses associated with noncancer illnesses in the animal studies we reviewed. However, in one study of newborn rats, manganese ingestion doses somewhat larger than the dose estimated for young children have been associated with biochemical changes in the nervous system. This study suggests young animals may be more susceptible than adults to manganese's toxic effects on the nervous system. In contrast, another study of rats did not find any association between such low doses of manganese and nervous system changes. Moreover, there is evidence rats may respond differently from

humans to manganese exposure, making the interpretation of the health effects in the former study uncertain. Present-day ingestion dose estimates are below levels of concern and are not likely to contribute significantly to past exposures. There are no reliable human studies available for comparison (ATSDR 1992f).

Site-specific Cancer Risk - Information on the cancer-causing potential of manganese is limited and difficult to interpret with certainty. Animal studies suggest the potential association between manganese and cancer in humans is probably small (ATSDR 1992f).

Sensitive Populations - There seems to be a wide range in individual susceptibility to nervous system effects from inhaling manganese dust. Newborn babies seem to retain a higher percentage of ingested manganese than adults, resulting in higher tissue levels of manganese, particularly in the brain. Still, it is not clear if this results in increased susceptibility to manganese-induced toxicity in infants. Elderly people may be somewhat more susceptible to manganese's toxic effects on the nervous system than the general population, perhaps because of loss of nerve cells due to aging or to accumulated damage from other environmental toxins acting on the nervous system. Because manganese is excreted through the liver, people with liver disease may have a decreased ability to handle excess manganese (ATSDR 1992f).

14. Methylene Chloride

Summary - There is not enough toxicological information to determine if noncancer illnesses may be associated with methylene chloride ingestion. The modeled past and present methylene chloride inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the human and animal studies we reviewed. Skin contact with liquid methylene chloride can cause chemical burns, but it is not known if skin absorption of methylene chloride is associated with internal health effects. We estimate the increased risk of liver cancer from past methylene chloride ingestion to be low. Using modeled inhalation data, we estimate the increased risk of developing lung or liver cancer from past methylene chloride inhalation to be moderate if actual exposure conditions are close to the estimated conditions used in the model. We estimate the increased cancer risk from present-day methylene chloride ingestion and inhalation to be negligible. We did not have enough information to estimate an increased cancer risk from past or present skin exposure to methylene chloride.

Use and Human Exposure - Methylene chloride is a human-made, colorless liquid with a sweetish odor. It is widely used as an industrial solvent and as a paint stripper. Methylene chloride is also used to manufacture photographic film, and can be found in some aerosols, pesticides, spray paints, automotive cleaners, and other household products. Because it evaporates easily, most methylene chloride enters the environment in air where it is subsequently broken down by sunlight and other air-

borne chemicals into carbon dioxide. Sometimes small amounts are found in drinking water where it is broken down by bacteria and water-borne chemicals. Because methylene chloride is so widely used, most people are exposed to this compound daily in air, water, food, or consumer products. Near hazardous waste sites, the most likely exposure route is by breathing in contaminated air. A majority of the methylene chloride that is inhaled enters the bloodstream and is quickly carried throughout the body. The methylene chloride uptake rate from the digestive system into the bloodstream is unknown, but is likely to be fast. Skin absorption is usually small. Once in the bloodstream, most of the methylene chloride goes to the liver, kidney, brain, lungs, and fatty tissue. Increased physical activity or increased body fat tends to increase the amount of methylene chloride that remains in the body. Within 40 minutes, about half of the methylene chloride leaves the blood as it is broken down into other chemicals, including carbon monoxide, a compound normally present in the body in small amounts from the periodic breakdown of blood hemoglobin. Most unchanged methylene chloride and its breakdown products leave the body by exhalation within 48 hours. Small amounts leave in the urine (ATSDR 1993m).

General Health Effects - Breathing in moderate amounts of methylene chloride for a few hours may temporarily impair hearing and vision. Inhaling larger amounts may temporarily impair reaction time, balance, and coordination. Breathing in methylene chloride for longer time periods may cause nausea, dizziness, drunkenness, and tingling or numbness in the fingers and toes. Animal studies suggest inhaling moderate amounts of methylene chloride is associated with changes in the liver and kidney. A limited number of studies have not found similar associations in humans. Animal studies also indicate exposure to high concentrations of methylene chloride vapors is associated with eye irritation and corneal changes; these effects appear to be reversible. In contrast, animal studies have not found an association between inhaling high concentrations of methylene chloride vapors and reproductive problems or birth defects. In humans, skin contact with liquid methylene chloride can cause chemical burn. Inhaling high methylene chloride concentrations has not been associated with cancer in humans; however, chronic inhalation of high methylene chloride concentrations has been associated with an increased occurrence of cancer in mice. Based on animal studies, methylene chloride is classified as a suspected cancer-causing agent in humans via ingestion and inhalation (ATSDR 1993m).

Interactions with Other Chemicals - In rats, methylene chloride can interact with carbon monoxide to produce additive increases in carboxyhemoglobin (carbon monoxide bound to blood hemoglobin) formation (ATSDR 1993m). This can decrease the blood's ability to carry oxygen and deliver oxygen to body tissues (Wilson et al. 1991). In animals, methylene chloride can interact with ethanol (drinking alcohol) to produce an additive decrease in the conduction of nerve impulses (ATSDR 1993m).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to methylene chloride through incidental ingestion of on-site subsurface soil and household uses of groundwater. Present-day groundwater analyses indicate exposure is continuing for residents still using private well water. In addition, the air stripper's trial run demonstrated this device will successfully remove methylene chloride from groundwater and expel it into the air. The past and present methylene chloride ingestion doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the few animal studies we reviewed (ATSDR 1993m). More studies examining potential health effects from low-dose methylene chloride ingestion are needed before we can make reliable comparisons with our estimated doses.

We used modeled data to estimate past inhalation exposure to methylene chloride volatilized in the shower and present in ambient air. The past and present methylene chloride inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illness in the few human studies we reviewed. However, methylene chloride inhalation doses smaller than past doses we estimated for all age groups have been associated with a reversible increase in liver fat content in studies of rats and mice. Although human study suggests the liver may not be a major target organ for methylene chloride in humans, little is known about methylene chloride's effects on the human liver or the significance of these findings. Present-day exposure doses from both the air stripper and from breathing methylene chloride vapors during household water use are below levels of concern (ATSDR 1993m).

Finally, liquid methylene chloride is a known skin irritant, and can cause chemical burns at unknown doses. There are no human or animal studies examining methylene chloride's potential effects on internal body systems from skin absorption (ATSDR 1993m).

Site-specific Cancer Risk - There are no human studies examining methylene chloride's cancer-causing potential after chronic oral exposure. However, animal studies indicate chronic oral exposure to high doses of methylene chloride is associated with liver cancer (ATSDR 1993m). EPA, NTP, and IARC each have classified methylene chloride as a suspected human cancer-causing agent via ingestion (ATSDR 1993a). Based on the exposure and dose estimates we have, we estimate adult residents' increased risk of cancer from past methylene chloride ingestion to be low at 52 in 100,000. This means the risk of getting cancer, above the background rate, could rise from 25,000 cases per 100,000 people to 250,052 cases in a 70-year lifetime. We estimate the present-day increased cancer risk from methylene chloride ingestion to be negligible.

Epidemiological studies have not identified a causal relationship between occupational exposure to airborne methylene chloride and cancer in humans. However, these studies are limited in their ability to detect small increases in cancer. Animal studies

suggest inhalation of high doses of methylene chloride is associated with lung and liver cancer in rats and mice, and is associated with an increased number of noncancerous breast tumors (in female mice and both sexes of rats) (ATSDR 1993m). EPA, NTP, and IARC each have classified methylene chloride as a suspected cancer-causing agent via inhalation (ATSDR 1993a). Since we did not have actual measurements of past methylene chloride concentrations in air, we used known groundwater concentrations to estimate the cancer risk from inhaling methylene chloride vapors in the shower and in ambient air inside and outside the home. Based on the modeled exposure and dose information we have, we estimate adult residents' increased risk of cancer from past methylene chloride inhalation to be moderate at 9 in 1,000. This means the risk of getting cancer, above the background rate, could rise from 250 cases per 1,000 people to 259 cases in a 70-year lifetime. We estimate the present-day increased cancer risk from methylene chloride inhalation to be negligible.

Finally, there are no human or animal studies of the potential association between methylene chloride skin absorption and cancer (ATSDR 1993m).

Sensitive Populations - People likely to be unusually sensitive to the effects of methylene chloride are those with pre-existing increased amounts of carboxyhemoglobin in their blood. This group includes smokers (who constantly maintain higher levels of carboxyhemoglobin in their blood) and people with cardiovascular disease (ATSDR 1993m).

15. n-Nitrosodiphenylamine

Summary - The past n-nitrosodiphenylamine ingestion doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the animal studies we reviewed. It is not known if inhalation or skin absorption of n-nitrosodiphenylamine is associated with noncancer illnesses. We estimate the increased cancer risk from past n-nitrosodiphenylamine ingestion to be negligible. There is not enough toxicological information to determine if past n-nitrosodiphenylamine inhalation or skin absorption could be associated with cancer.

Use and Human Exposure - n-Nitrosodiphenylamine is a orange-brown or yellow solid that evaporates slowly into the air, dissolves in water, and attaches to soil. There is evidence that some microorganisms may make small amounts of this compound in nature. Human-made n-nitrosodiphenylamine is used to produce rubber products and other chemicals. It enters the environment from industrial discharges or from hazardous waste site releases into air, water, or soil. Once in the environment, n-nitrosodiphenylamine breaks down into other substances. It is not known if any of these breakdown products are harmful. Most n-nitrosodiphenylamine disappears from water or soil within several weeks. Because n-nitrosodiphenylamine is not normally found in the environment, people are usually exposed to this chemical only at work or

at hazardous waste sites. Animal studies indicate n-nitrosodiphenylamine can enter the body through ingestion or skin contact. It is not known if n-nitrosodiphenylamine can enter the body through the lungs. Once in the body, animals break this chemical down into other chemicals that can harm their health. One animal study shows that n-nitrosodiphenylamine rapidly leaves the body in the urine. Some is probably excreted in the feces as well. Based on the animal data, it is likely humans have similar intake, breakdown, and elimination mechanisms (ATSDR 1993p).

General Health Effects - Very little is known about the possible health effects of n-nitrosodiphenylamine. In animals, ingesting very large amounts of this compound can cause death. In other studies, animals eating moderate to high doses of n-nitrosodiphenylamine for a long time developed swelling, changes in body weight, and bladder cancer. It is not known if this chemical has comparable effects in humans. Similarly, it is not known if n-nitrosodiphenylamine affects reproduction or causes birth defects. Based on the animal studies, n-nitrosodiphenylamine is classified as a suspected cancer-causing agent in humans via ingestion (ATSDR 1993a; IRIS 1994).

Interactions with Other Chemicals - In mice, n-nitrosodiphenylamine can interact with pentobarbital, a component of some prescription drugs, to decrease the drug's sedative effects (that is, sleeping time) (ATSDR 1993p).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to n-nitrosodiphenylamine through ingestion of groundwater. The limited number of present-day samples indicate n-nitrosodiphenylamine is no longer found in the groundwater, but we do not have enough groundwater, surface soil, or sediment samples to confirm exposure has stopped. The past n-nitrosodiphenylamine ingestion doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the animal studies we reviewed. There are no human or reliable animal studies examining the health effects associated with n-nitrosodiphenylamine inhalation or skin absorption (ATSDR 1993p). More studies examining the potential health effects from these exposure routes are needed before we can make reliable comparisons with our estimated doses.

Site-specific Cancer Risk - There are no human studies examining n-nitrosodiphenylamine's cancer-causing potential after oral exposure. However, there is a weak association between high-dose n-nitrosodiphenylamine ingestion and an increased frequency in bladder cancer in rats (ATSDR 1993p). EPA has classified n-nitrosodiphenylamine as a suspected human cancer-causing agent via ingestion (ATSDR 1993a). Based on the exposure and dose information we have, we estimate adult residents' increased risk of cancer from past exposure to n-nitrosodiphenylamine to be negligible. There are no human or reliable animal studies examining the potential association between n-nitrosodiphenylamine inhalation or skin absorption and cancer (ATSDR 1993p).

Sensitive Populations - Because of the limited toxicity data for n-nitrosodiphenylamine, it is difficult to identify persons likely to be unusually sensitive to this compound. It is possible people with bladder dysfunction or disease will be unusually sensitive to n-nitrosodiphenylamine exposure. It also seems likely n-nitrosodiphenylamine may affect the body's breakdown of some prescription drugs and ethanol (drinking alcohol) in unspecified ways (ATSDR 1993p).

16. PCBs

Summary - There is not enough toxicological information to determine if noncancer illnesses may be associated with PCB ingestion, inhalation, or skin absorption doses similar to the past doses we estimated for all age groups. We estimate the increased liver cancer risk from past PCB ingestion to be moderate. There is not enough toxicological information to determine if past PCB inhalation or skin absorption could be associated with cancer.

Use and Human Exposure - PCBs (polychlorinated biphenyls) are a group of human-made organic chemicals, consisting of over 200 individual compounds. PCBs are either oily liquids or solids that range in color from clear to light yellow. They have no smell or taste. Some PCB mixtures are called by their commercial name, Aroclor. Because they are a good insulating material and don't burn easily, PCBs were widely used as coolants and lubricants in transformers, capacitors, and other electrical equipment. In the United States, the manufacture of PCBs stopped in 1977 because of evidence that they built up in the environment and caused harmful effects. Still, consumer products made prior to October 1977, such as fluorescent lighting fixtures, electrical devices or appliances, microscope oil, and hydraulic fluids may contain PCBs. In the past, PCBs entered the air, water, and soil during their manufacture, use, and disposal. Today, PCBs enter the environment from poorly maintained hazardous waste sites containing PCBs, improper dumping of PCB wastes, leaks from electrical transformers containing PCBs, and disposal of PCB-containing consumer products into municipal landfills. In the air, PCBs can be present as solid or liquid aerosols, or as vapors. PCBs in the air can travel long distances away from their source, but eventually settle on the land or water. Only small amounts of PCBs remain dissolved in water, and most stick strongly to soils or sediments. In soils, PCBs usually don't travel downwards very deeply with rainwater. In sediments, fish may ingest PCBs, and these compounds can build up in their bodies until the PCB concentration is thousands of times larger than the concentration found in the water. The breakdown of PCBs in water and soil takes several years. In the sediments of permanent lakes and rivers, PCBs usually don't break down; instead, they are released back into the water in small amounts over time (ATSDR 1993q).

Because PCBs remain in the environment for a long time and because many pieces of electrical equipment containing PCBs are still in use, it is still possible to be exposed to these compounds. Small amounts of PCBs can be found in almost all outdoor air,

indoor air, soil surfaces, and surface waters. PCBs may also be found in fish. The concentration of PCBs in air, water soil and food have generally decreased since production stopped in 1977. Eating fish and breathing air in buildings using PCB-containing electrical equipment are the most likely sources of PCB exposure for most people. People living around hazardous waste sites containing PCBs are usually exposed by breathing air containing PCBs. Children playing at or near these sites may also be exposed by touching and eating soils containing PCBs. Infants are most likely exposed through breast milk containing PCBs (ATSDR 1993q).

Nearly all of the ingested PCBs are likely to be absorbed quickly into the bloodstream. It is not known how much or how quickly PCBs are absorbed into the body through inhalation or skin absorption. Once in the body, some PCBs are broken down into other chemicals that are eliminated in the feces within a few days. However, some PCBs and their breakdown products remain stored in body fat for months and perhaps years. Some of the stored PCBs build up in breast milk and can be passed to infants through breast-feeding. It is not known if any of the PCB breakdown products are barmful to human health (ATSDR 1993q).

General Health Effects - Human studies have shown workers exposed to relatively high concentrations of PCB vapors can develop skin irritations, such as rashes and acne, as well as nose, lung, and eye irritation. There are no studies of PCB ingestion in humans. In animal studies, ingestion of large amounts of PCBs for a short time period has been associated with mild liver damage and death in rats. In other animal experiments, ingestion of smaller amounts of PCBs over several weeks or months has been associated with many serious health effects including: liver, stomach, and thyroid gland injuries; anemia; acne; and damaged reproduction. These effects were seen in many different kinds of animals and in their offspring. PCB ingestion has not been associated with birth defects. There is limited information on health effects from skin or inhalation exposure to PCBs. In one experiment, skin exposure to moderate amounts of PCBs has been associated with liver, kidney, and skin damage in rabbits. In different experiments, breathing in large amounts of PCBs over several months has been associated with liver and kidney damage in rats and other animals. It is not known if PCBs cause cancer in humans. Experiments in rats indicate PCB exposure may be associated with liver cancer in these animals. Based on the animal data, PCBs are classified as a suspected human carcinogen (ATSDR 1993a, 1993q).

Interactions with Other Chemicals - Studies indicate PCBs can interact with pentobarbital, a component of some prescription drugs, to decrease the drug's sedative effects (that is, sleeping time). In animals, pretreatment with PCB mixtures can increase the toxic effects trichloroethene and tetrachloroethene have on the liver. In rats, pretreatment with Aroclor-1254 protected against the toxic effects on the liver due to 1,1-dichloroethene inhalation. Increased dietary vitamin C intake may have similar protective effects. In rats, coadministration of cadmium and Aroclor-1248 have additive effects on growth retardation and blood cholesterol. PCBs can interact

with cancer-causing agents in various ways, depending on the interacting chemical used. In animals, several oral exposure studies indicate PCBs may work as cancer cell promoters, and one skin absorption study suggests PCBs may have weak cancer cell initiation abilities. Other studies suggest PCBs may act as cancer inhibitors (ATSDR 1993q).

Site-specific Noncancer Health Effects - In the past, nearby residents were exposed to PCBs through incidental ingestion of on- and off-site sediments, incidental ingestion of off-site surface waters, and ingestion of groundwater. The limited number of present-day samples indicate PCBs is no longer found in the groundwater, but we do not have enough groundwater, surface soil, or sediment samples to confirm exposure has stopped. In two studies of monkeys, chronic PCB ingestion doses similar to those we estimated for all age groups have been associated with a decrease in the numbers of two kinds of antibodies. However, other immune system parameters measured in these studies were not affected, and there was no change in overall immune response (ATSDR 1993q). Nevertheless, animal studies also indicate PCB exposure at higher doses is associated with suppression of the immune system (Amdur et al. 1991; ATSDR 1993q). More studies examining potential immune system effects from low-dose PCB ingestion are needed before we can make reliable comparisons with our estimated doses.

We used modeled data to estimate past inhalation exposure to PCBs volatilized in the shower and present in ambient air. PCB inhalation doses similar to the past inhalation doses we estimated for all age groups have been associated with effects on the liver and kidney in the one animal study available. In this study, the severity of the liver effects ranged from increased numbers of storage compartments inside liver cells to fatty changes and other degenerative lesions, depending on the species of animal tested. Although epidemiologic studies of Aroclor-exposed workers indicate an association may exist between PCB exposure and increased activity of some liver enzymes, there is no conclusive evidence that PCBs are toxic to the human liver. The available study also reported an association between low-dose PCB inhalation and slight degeneration of the kidney tubules in rats, but an invalid analytical method made the PCB concentrations uncertain (ATSDR 1993q). More studies examining potential health effects from low-dose PCB inhalation are needed before we can make reliable comparisons with our estimated doses.

Finally, the doses we estimated for PCB skin absorption while showering or swimming were much smaller than the doses associated with noncancer illnesses in the few animal studies we reviewed. However, these studies did not investigate the potential health effects associated with chronic, low-dose skin exposure to PCBs. More studies are needed before we can make reliable comparisons with our estimated skin absorption doses (ATSDR 1993q).

Site-specific Cancer Risk - Studies of PCB-exposed workers provide inconclusive evidence that PCB exposure is associated with cancer in humans. However, animal studies indicate the cancer-causing potential of PCB mixtures depends on the degree of chlorination, and ingestion of PCBs that are at least 60% chlorine by weight is associated with liver cancer in rats. In addition, studies with rats and mice indicate that PCBs with lower chlorine content can act as tumor promoters once these cells have been treated with chemicals acting as tumor initiators (ATSDR 1993q). Based on the animal evidence, EPA, NTP, and IARC each have classified PCBs as a suspected cancer-causing agent in humans via ingestion (ATSDR 1993a). Based on the exposure and dose information we have, we estimate the increased cancer risk from past PCB ingestion to be moderate at 6 in 1,000. This means the risk of developing cancer, above the background rate, could rise from 250 cases per 1,000 people to 256 cases in a 70-year lifetime. There are no reliable studies examining the potential association between PCB inhalation or skin absorption and cancer (ATSDR 1993q).

Sensitive Populations - Unborn and newborn children are potentially susceptible to the health effects of PCBs because their underdeveloped enzyme systems do not eliminate chemicals from the body as easily as in adults. In addition, breast-fed infants may have additional risk because of a steroid excreted in human milk that can inhibit PCB elimination from the body. Children exposed to the antibiotic novobiocin may also be more susceptible to the health effects of PCBs because the drug may interact with the mechanism responsible for eliminating PCBs from the body. People with liver infection, dysfunction, or disease may also be more susceptible to PCB toxicity (ATSDR 1993q).

17. 1,1,2,2-Tetrachloroethane

Summary - There is not enough toxicological information to determine if noncancer illnesses may be associated with 1,1,2,2-tetrachloroethane ingestion or skin absorption doses similar to the past doses we estimated for all age groups. The past 1,1,2,2-tetrachloroethane inhalation doses we estimated for all age groups are much smaller than the doses associated with noncancer illnesses in the human and animal studies we reviewed. There is no apparent increased cancer risk from past 1,1,2,2-tetrachloroethane ingestion. There is not enough toxicological information to determine if past 1,1,2,2-tetrachloroethane inhalation or skin absorption could be associated with cancer.

Use and Human Exposure - 1,1,2,2-Tetrachloroethane is a human-made, colorless liquid with a sweet, chloroform-like odor. In the past, 1,1,2,2-tetrachloroethane was used in large quantities to produce other chemicals and as an industrial solvent. It was also used to separate other substances, to clean and degrease metals, and to manufacture paints and pesticides. In the present, 1,1,2,2-tetrachloroethane's use appears to be limited, but information about its use is not available. Most 1,1,2,2-

APPENDICES

A. Figures

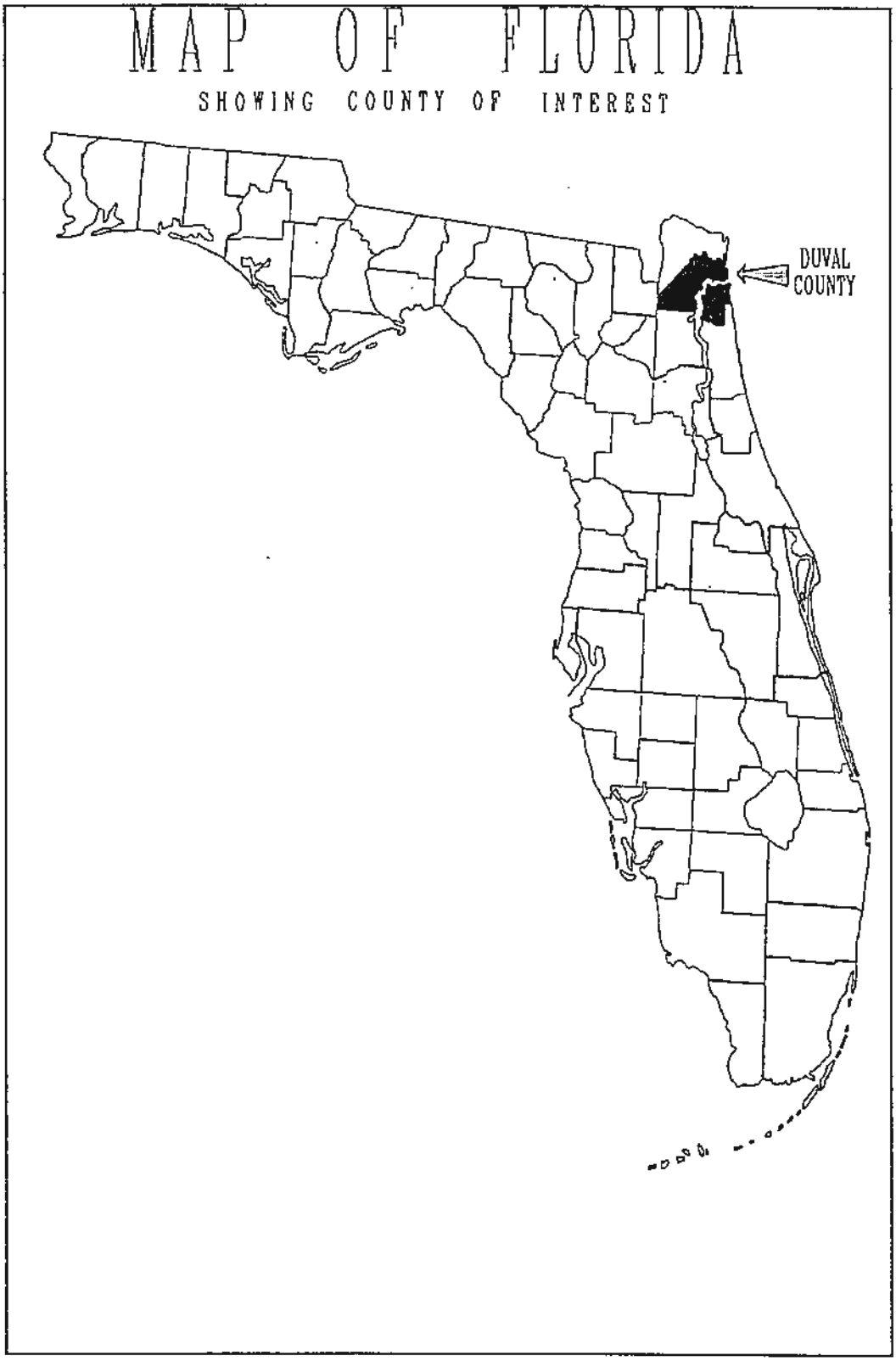

Figure 1. Location of Duval County Florida.

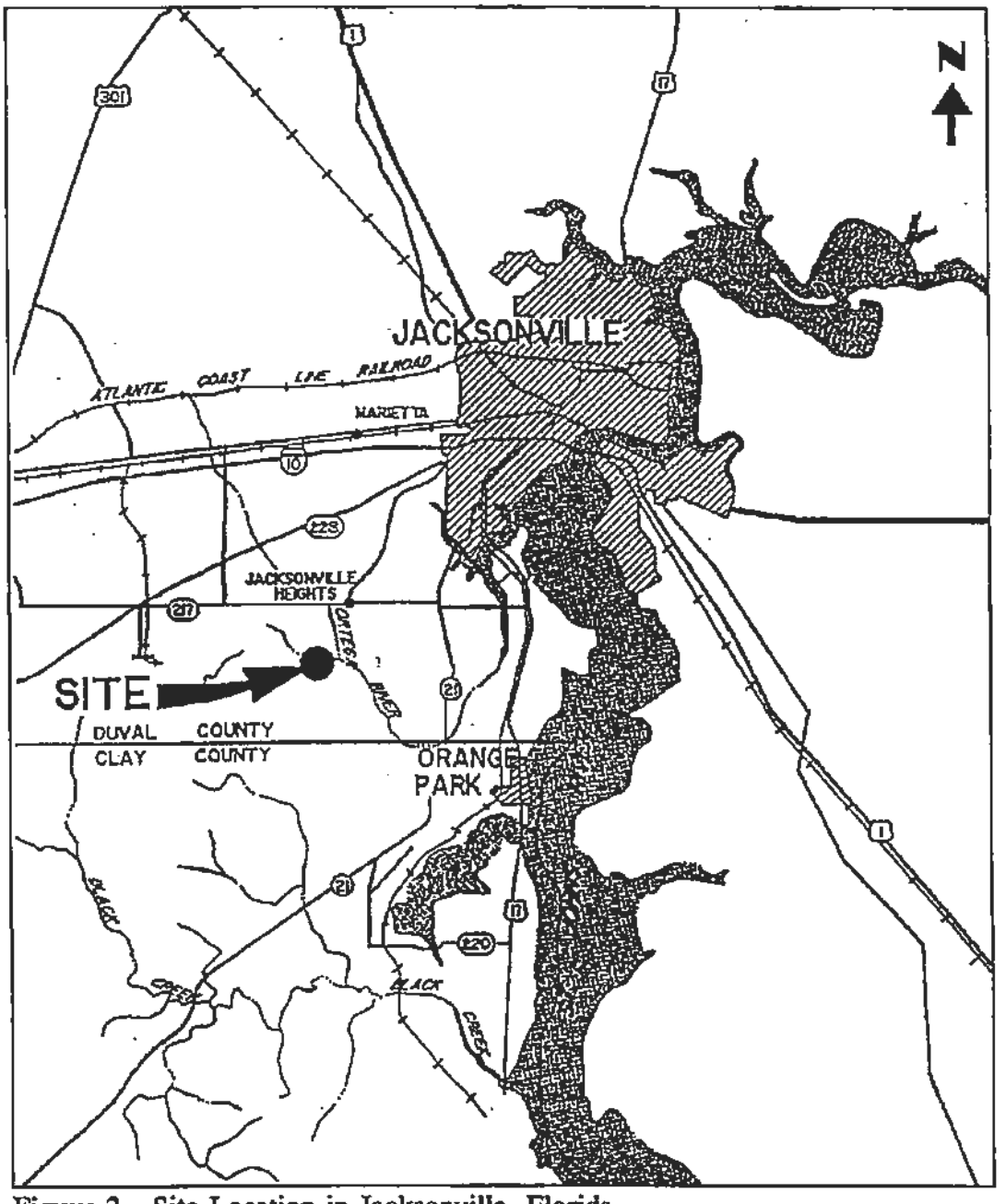

Figure 2. Site Location in Jacksonville, Florida.

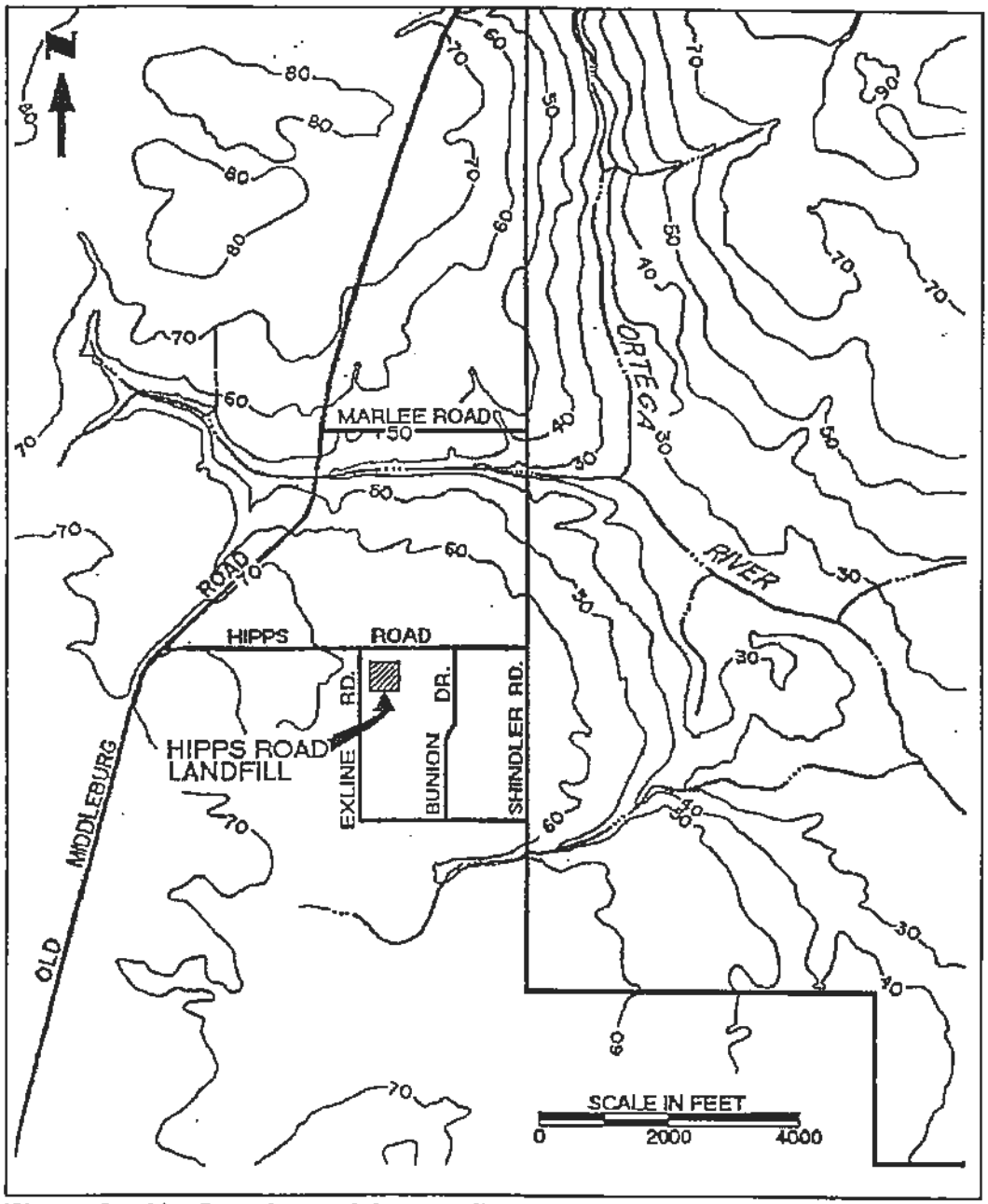

Figure 3. Site Location and Surrounding Topography.

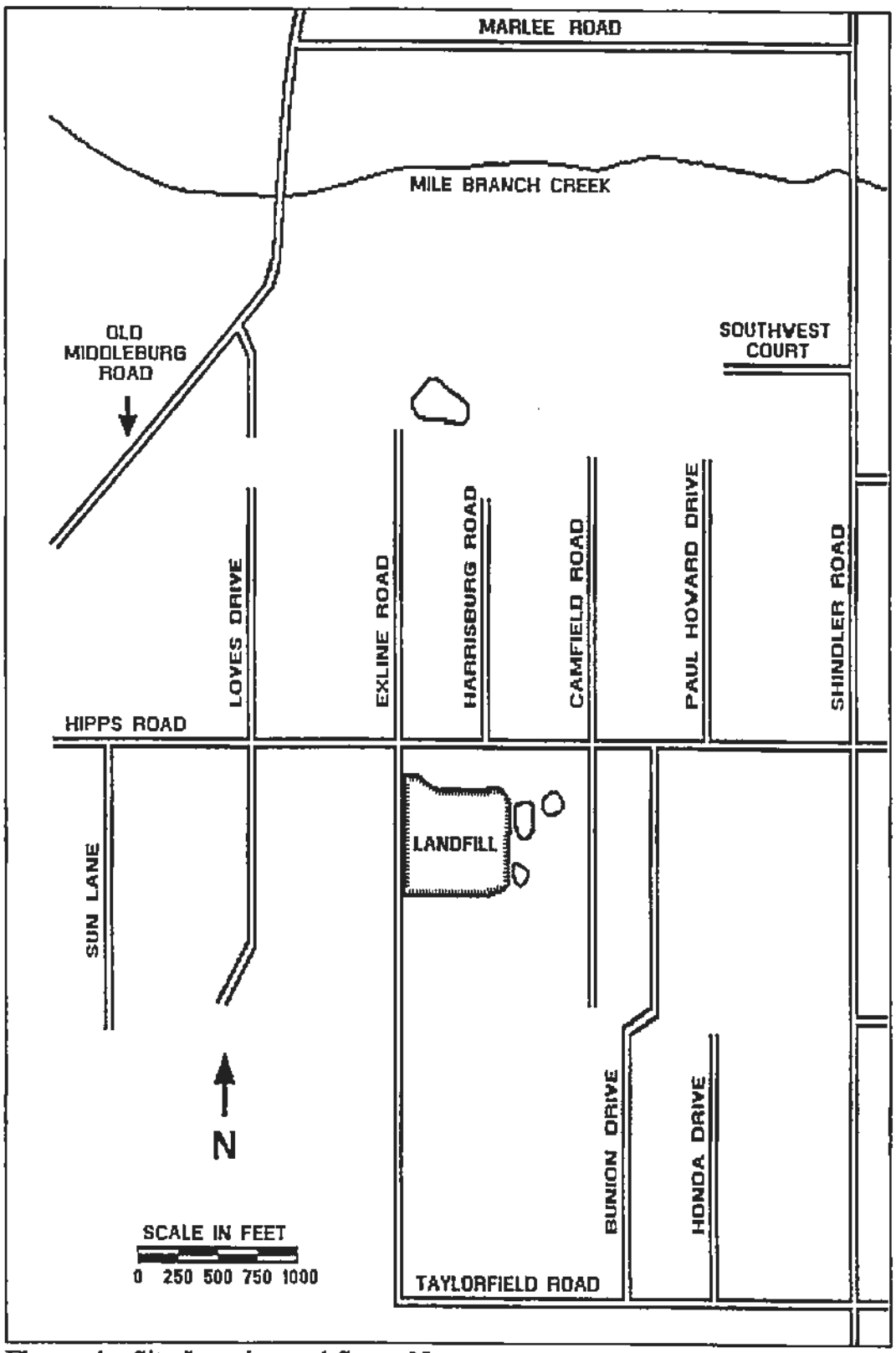

Figure 4. Site Location and Street Names.

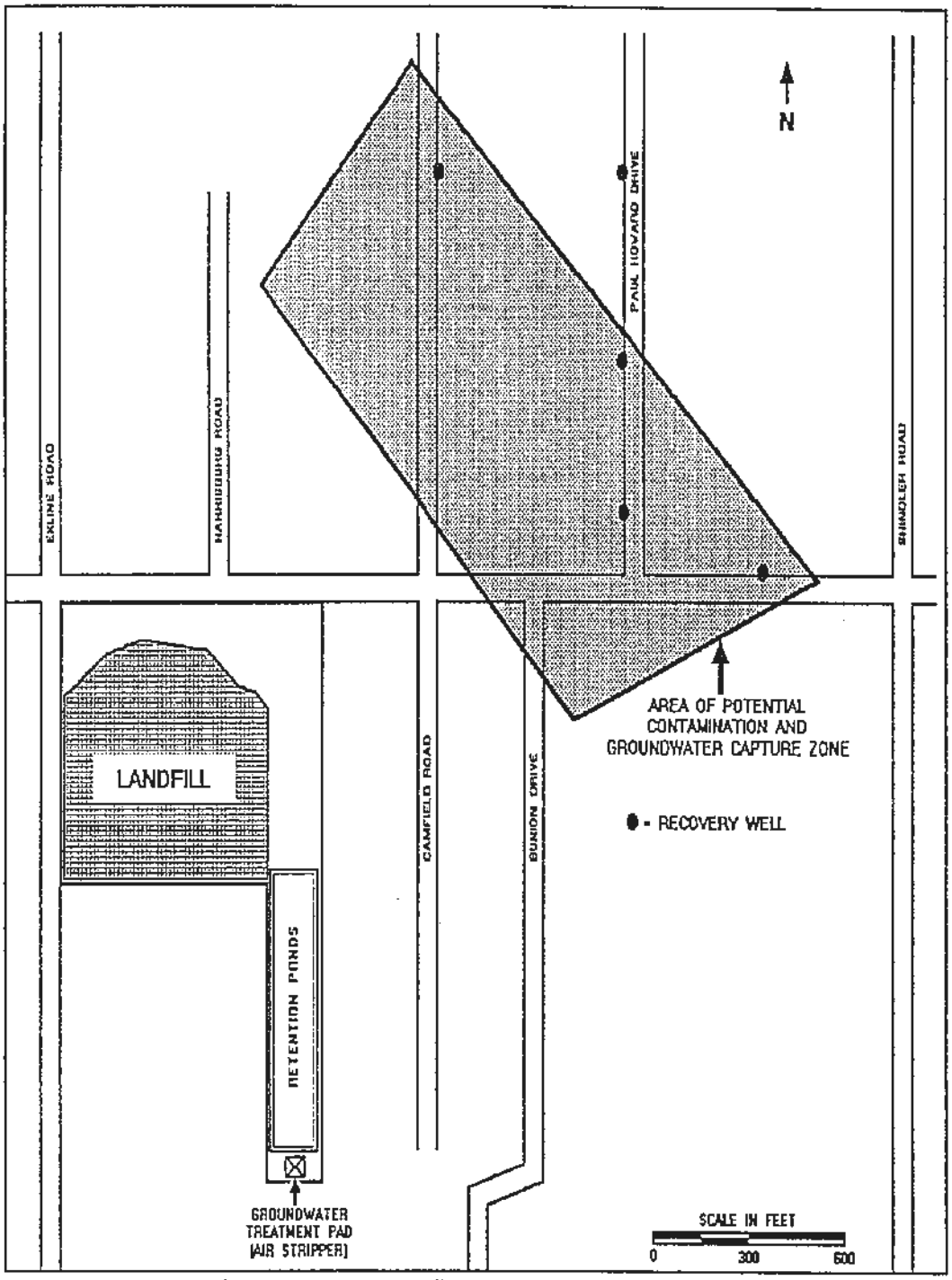

Figure 5. Groundwater Treatment System.

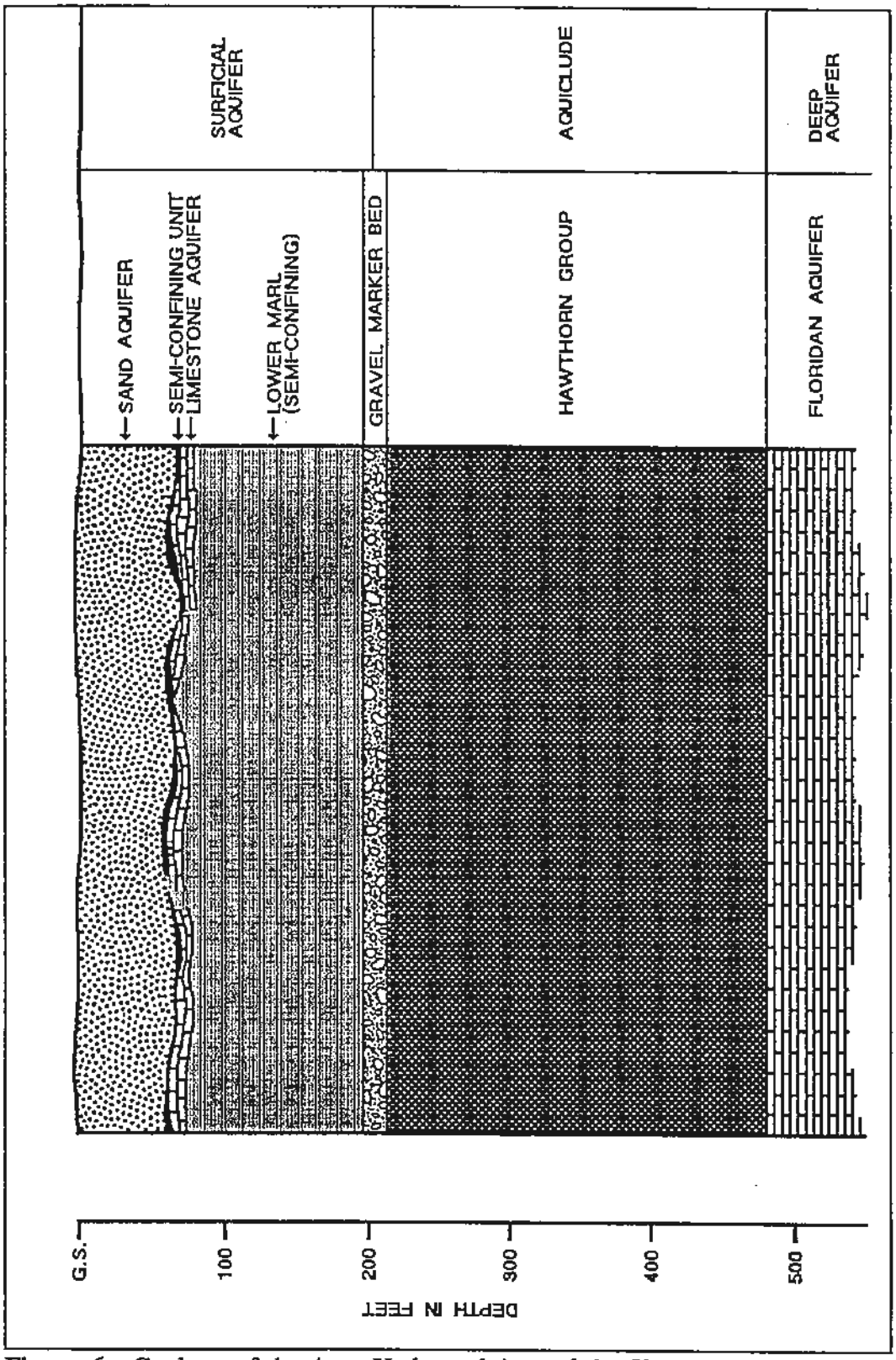

Figure 6. Geology of the Area Under and Around the Site.

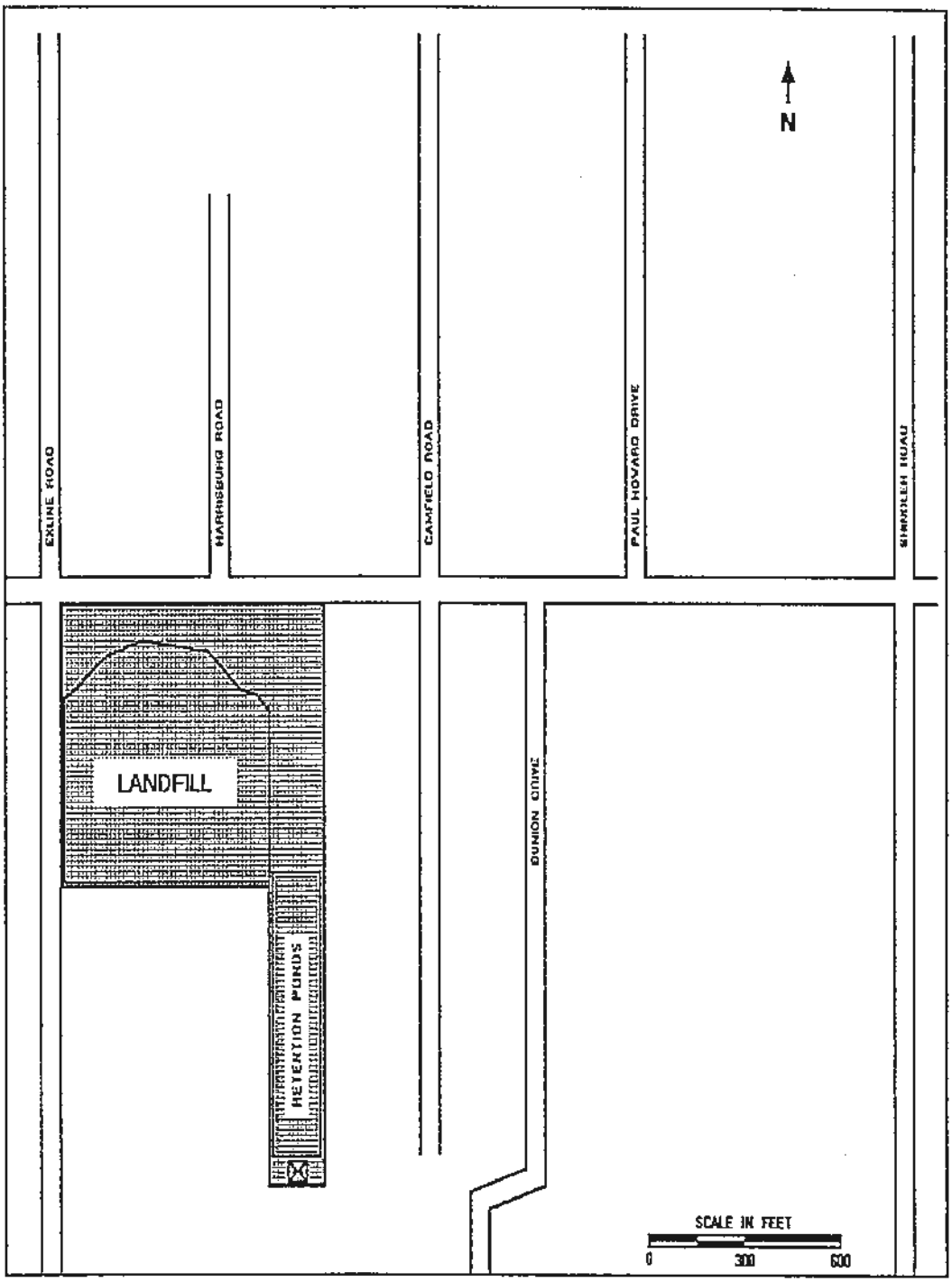

Figure 7. On-site Boundaries.

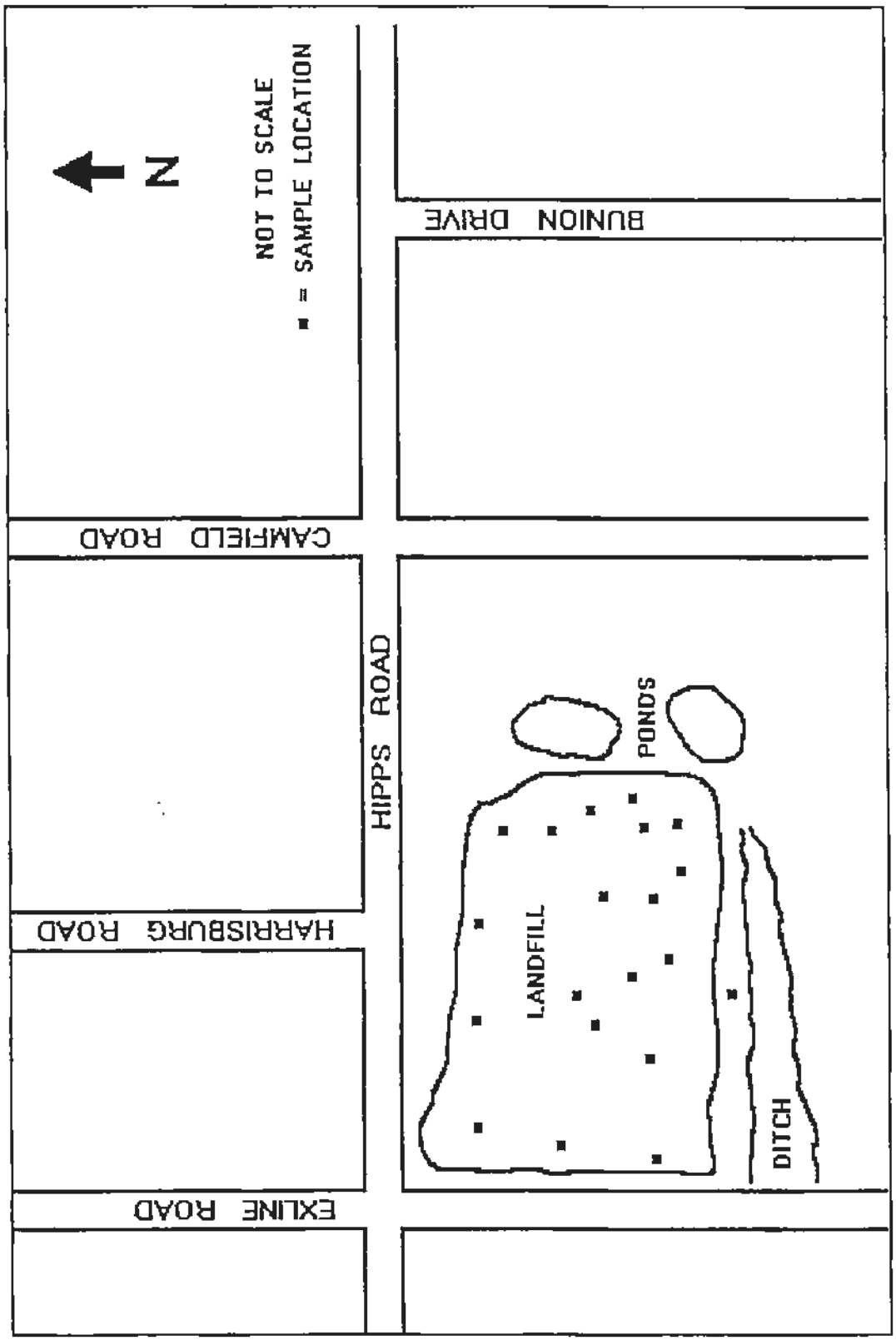

Figure 8. On-site Subsurface Soil Sample Locations.

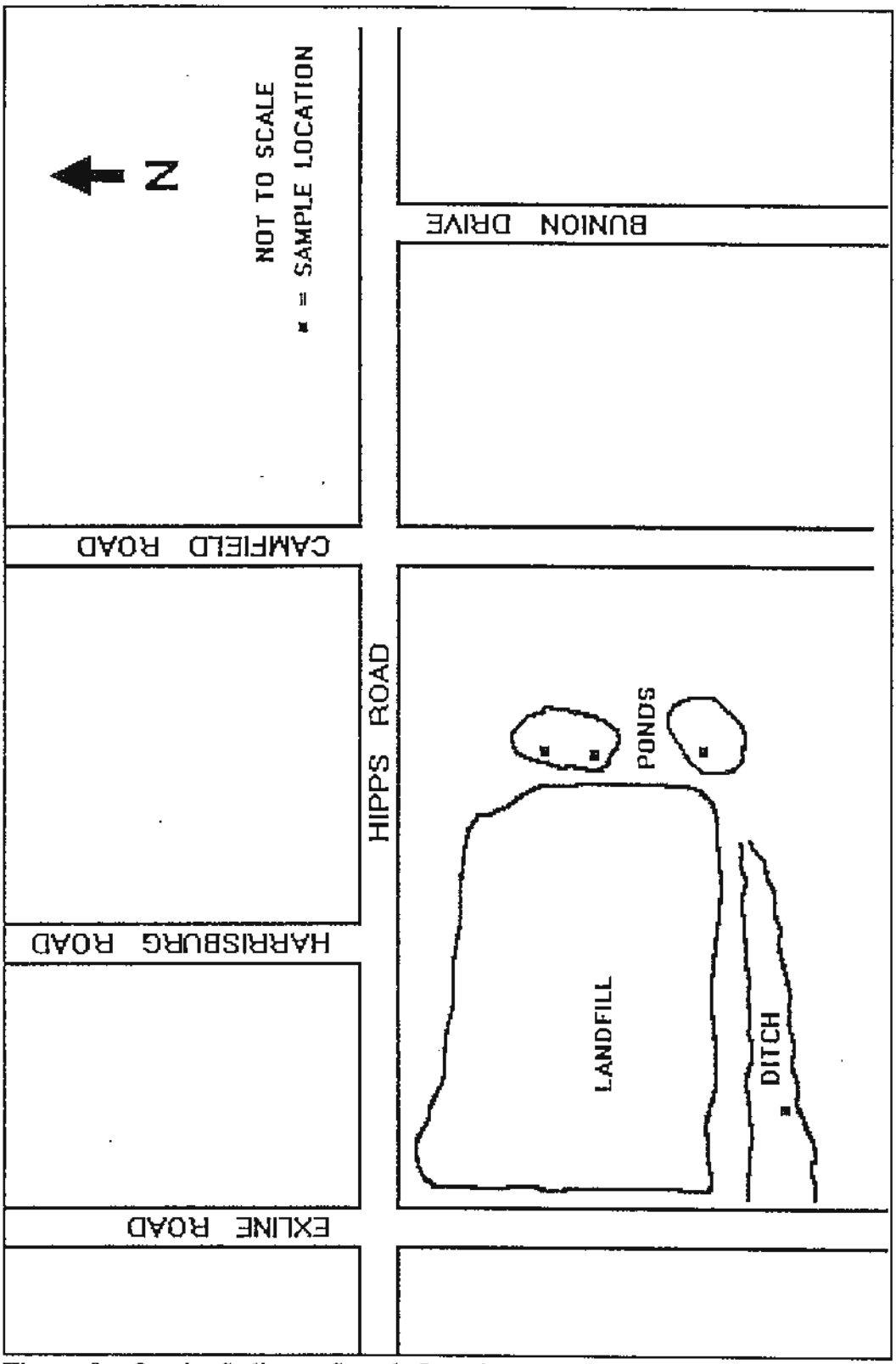

Figure 9. On-site Sediment Sample Locations.

Figure 10. On-site Surface Water Sample Locations.

Figure 11. On-site Groundwater Sample Locations.

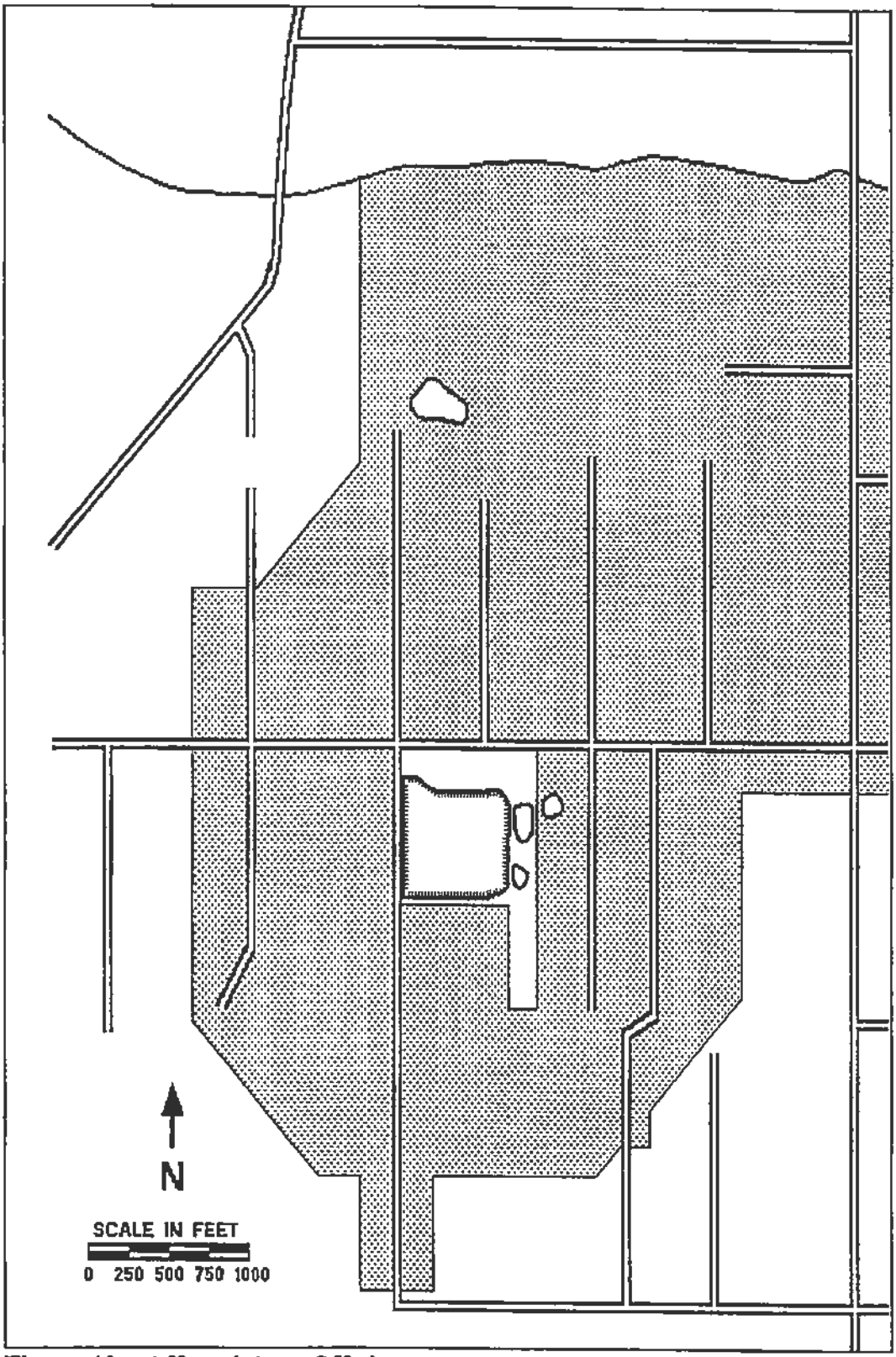

Figure 12. Affected Area Off-site.

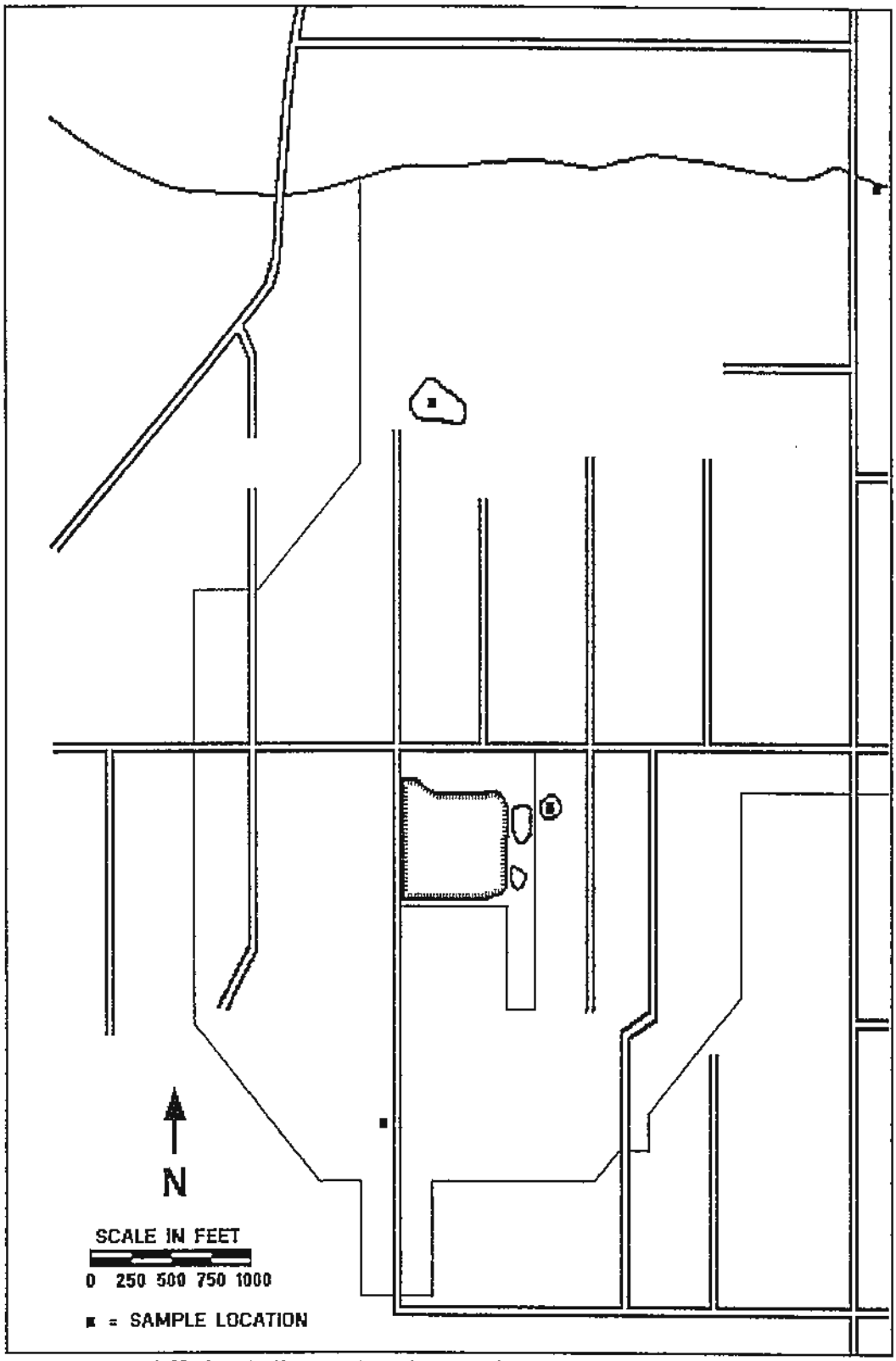

Figure 13. Off-site Sediment Sample Locations.

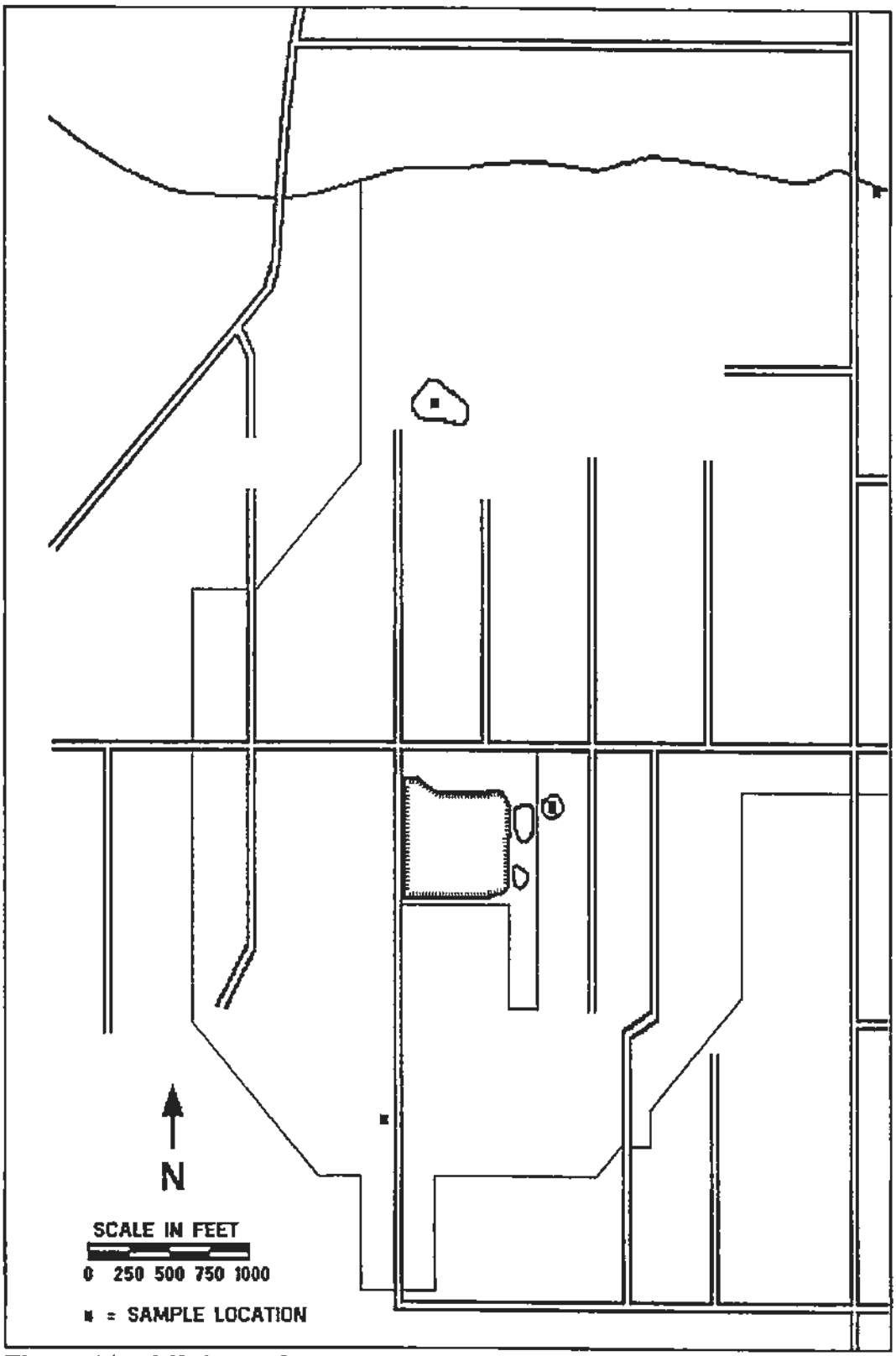

Figure 14. Off-site Surface Water Sample Locations.

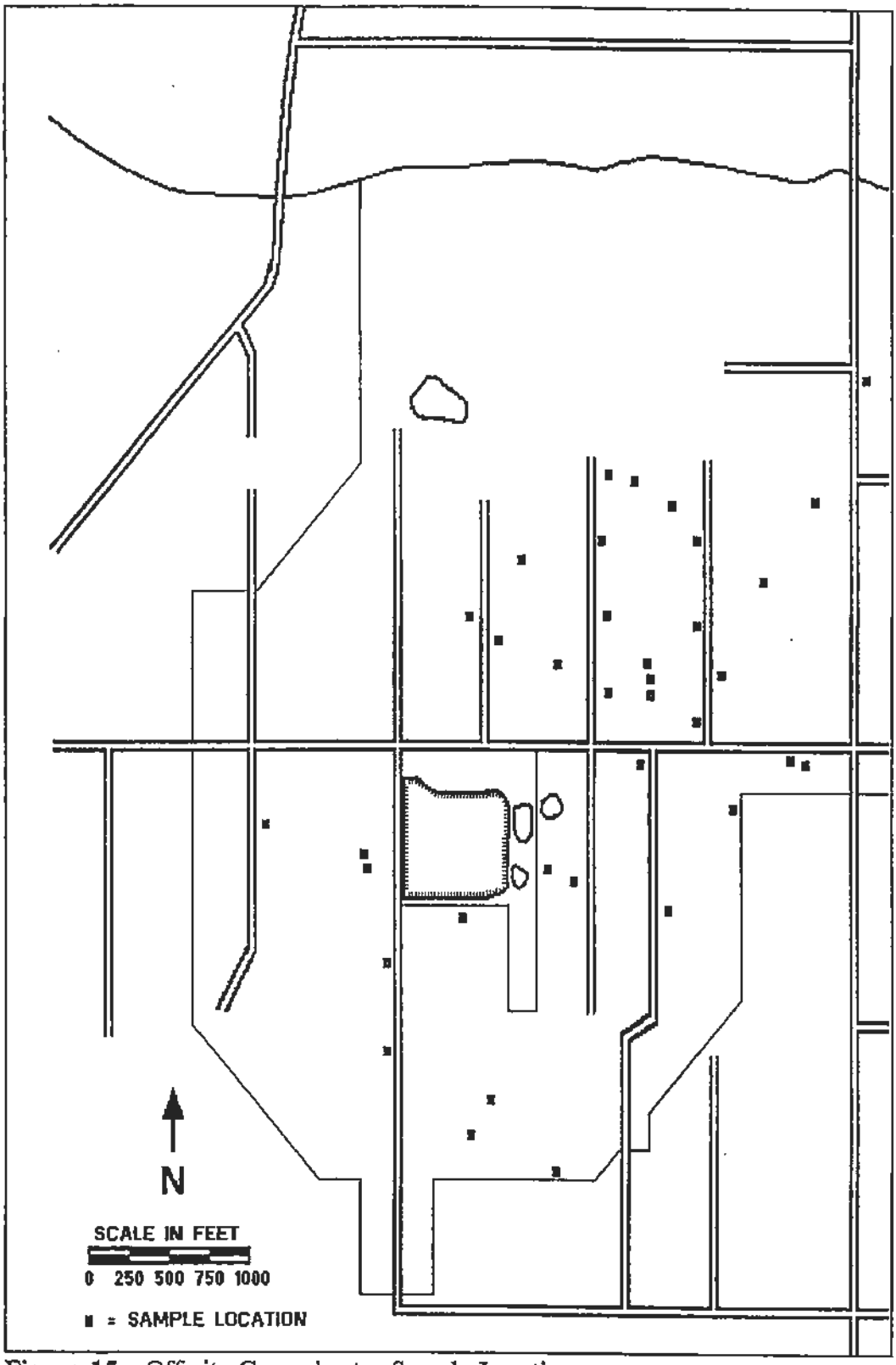

Figure 15. Off-site Groundwater Sample Locations.

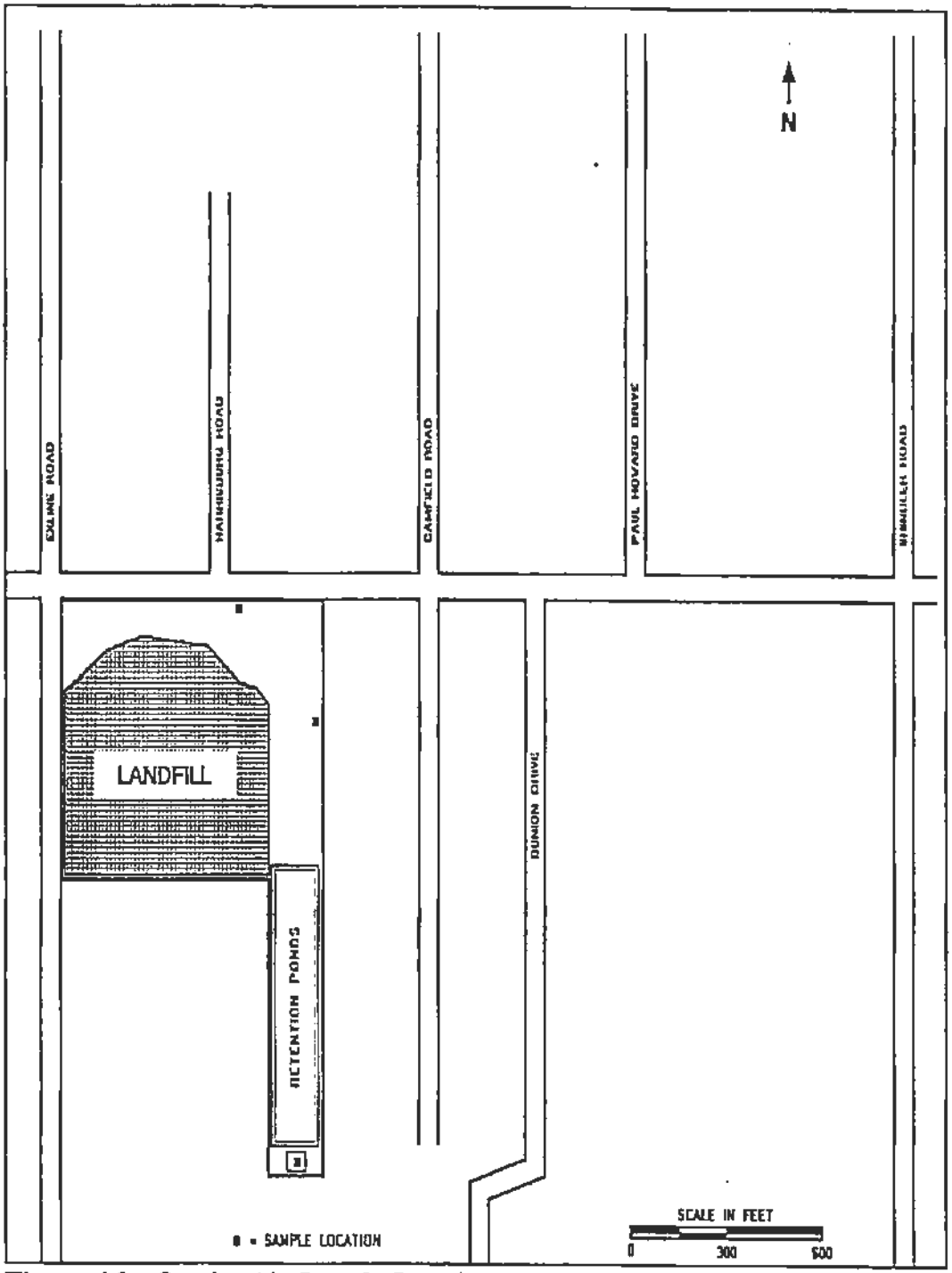

Figure 16. On-site Air Sample Locations.

B. Tables

Table 1. Detected Contaminants

Contaminant Name	
Acetone	Chlorodibromomethane
Aluminum	Chloroethanol Phosphate
Aminohexanoic Acid	Chloroform
Ammomium	1,2-Chlorotoluene
Antimony	Chromium(VI)
Arsenic	Cobalt
Barium	Copper
Benzene	Cresol
Benzeneacetic Acid	Cyanide
Benzenebutanoic Acid	Cyclohexanone
Benzenepropionic Acid	2-Cyclohexene
Benzoic Acid	p-Cymene
Benzo(g,h,i)perylene	DDT
Benzothiazolone	Di-n-Butylphthalate
Benzylbutylphthalate	1,2-Dichlorobenzene
Benzyl Alcohol	1,3-Dichlorobenzene
Beryllium	1,4-Dichlorobenzene
Biphenol	1,1-Dichloroethane
Bromochloromethane	1,2-Dichloroethane
Bromodichloromethane	1,2-Dichloroethene (total)
n-Butylbenzene	cis-1,2-Dichloroethene
C2 Alkylbenzene Butanoic Acid	trans-1,2-Dichloroethene
C2 Alkylphenol	1,2-Dichloropropane
C2 Alkylstyrene	Diethylene Glycol Diethyl Ether
C3 Alkylbenzene	Diethylene Glycol Monobutyl Ether
C3 Alkylbenzene Sulfonamide	Di(2-ethylhexyl)phthalate
C3 Alkylcyclohexane Methanol	Diethyltoluamide
C3 Alkylphenol	Dihydroindenone
C4 Alkylbenzene	Dihydromethylindole
C4 Alkylcyclohexanol	2,4-Dimethylphenol
C5 Alkylbenzamide	Di-n-octylphthalate
C5 Alkylbenzene	1,2-Diphenylhydrazine
C5 Alkylbenzene Sulfonamide	Endrin Ketone
C6 Alkylphenol	Ethylbenzene
Cadmium	Ethyl Ether
Calcium	Ethylhexanoic Acid
Carbon (organic, total)	Ethylmethylbenzene Sulfonamide
Carbon Disulfide	Fluoranthene
Chlorobenzene	Fluoride

Table 1. Detected Contaminants, continued

Contaminant Name	
Hexachloroethane	PCBs (total)
Hexadecanoic Acid	Pentaoxapentadecane
Hydrocarbons	Pentobarbital
Iron	Phenol
Lead	Phenyletbylphenol
Magnesium	Potassium
Manganese	Propanol
Mercury	Propyl Benzene
Methoxycblor	Selenium
Methylbenzene Sulfonamide	Silver
Methylene Chloride	Sodium
Methyl Ethyl Ketone	Sulfate
Methyl Indole	1,1,2,2-Tetrachloroethane
Methyl Naphthalene (total)	Tetrachloroethene
2-Methyl Naphthalene	Tetrahydrofuran
Methylnonanediol	Tin
Methylpentanediol	Toluene
4-Methyl-2-Pentanone	1,1,1-Trichloroethane
2-Methylphenol	Tri(chloroethanol)phosphate
4-Methylphenol	Trichloroethene
Methyl Styrene	Triethylene Glycol Monoethyl Ether
MTBE	Trimethylbenzene (total)
Naphthalene	Trimethylhicycloheptanone
Nickel	Vanadium
Nitrate	Vinyl Chloride
Nitrite	Xylene (total)
n-Nitrosodiphenylamine	Zinc
Oxy-bis-Ethoxythane	

Table 2. Contaminants Below Comparison Values in All Media in Which They Were Detected

Contaminant Name	
Acetone	2,4-Dimethylphenol
Antimony	Ethyl Ether
Benzoic Acid	Ethylbenzene
Bromochloromethane	Fluoranthene
Carbon Disulfide	Methoxychlor
Chloromethane	Methyl Ethyl Ketone
1,2-Chlorotoluene	2-Methylphenol
Cyclohexanone	Phenol
Di-n-Butylphthalate	Silver
1,2-Dichlorobenzene	Toluene
1,3-Dichlorobenzene	1,1,1-Trichloroethane
1,2-Dichloroethene (total)	Vanadium
cis-1,2-Dichloroethene	Xylene (total)
trans-1,2-Dichloroethene	Zinc

Table 3. Contaminants Without Comparison Values in the Media in Which They Are Detected

Contaminant Name	
Aminohexanoic Acid	Dihydroindenone
Ammomium	Dihydromethylindole
Benzeneacetic Acid	Di-n-octylphthalate
Benzenebutanoic Acid	Endrin Ketone
Benzenepropionic Acid	Ethylhexanoic Acid
Benzo(g,h,i)perylene	Ethylmethylbenzene Sulfonamide
Benzothiazolone	Hexadecanoic Acid
Benzylbutylphthalate	Hydrocarbons
Benzyl Alcohol	Magnesium
Biphenol	Methylbenzene Sulfonamide
n-Butylbenzene	Methyl Indole
C2 Alkylbenzene Butanoic Acid	Methyl Naphthalene (total)
C2 Alkylphenol	2-Methyl Naphthalene
C2 Alkylstyrene	Methylnonanediol
C3 Alkylbenzene	Methylpentanediol
C3 Alkylbenzene Sulfonamide	4-Methyl-2-Pentanone
C3 Alkylcyclohexane Methanol	4-Methylphenol
C3 Alkylphenol	Methyl Styrene
C4 Alkylbenzene	MTBE
C4 Alkylcyclohexanol	Oxy-bis-Ethoxythane
C5 Alkylbenzamide	Pentaoxapentadecane
C5 Alkylbenzene	Pentobarbital
C5 Alkylbenzene Sulfonamide	Phenylethylphenol
C6 Alkylphenol	Potassium
Calcium	Propanol
Carbon (organic, total)	Propyl Benzene
Chloroethanol Phosphate	Tetrahydrofuran
2-Cyclohexene	Tri(chloroethanol)phosphate
p-Cymene	Triethylene Glycol Monoethyl Ether
Diethylene Glycol Diethyl Ether	Trimethylbenzene (total)
Diethylene Glycol Monobutyl Ether	Trimethylbicycloheptanone
Diethyltoluamide	

Table 4. Maximum Concentrations in On-site Subsurface Soil

Contaminants of Concern	Maximum Concentration Detected (mg/kg)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range (mg/kg)	Comparison Value	
					(mg/kg)	Source
Arsenic	2.8	4/20	3/20	NA	0.4	CREG
Barium	ND	0/3	--	--	--	--
Benzene	0.005	3/10	0/10	NA	20	CREG
Beryllium	ND	0/3	--	--	--	--
Bromodichloro-methane	NA	NA	--	--	--	--
Cadmium	1.2	1/15	1/15	NA	1	EMEG
Chlorobenzene	0.11	3/10	0/10	NA	40	RMEG
Chlorodibromo-methane	ND	0/3	--	--	--	--
Chloroform	ND	0/3	--	--	--	--
Chromium(VI)	4.9	6/18	--	NA	None	Carcinogen
Cobalt	ND	0/3	--	--	--	--
Cresol	ND	0/3	--	--	--	--
Cyanide	2.2	3/7	0/7	NA	40	RMEG
DDT	ND	0/3	--	--	--	--
1,4-Dichlorobenzene	0.14	1/10	--	NA	None	Carcinogen
1,1-Dichloroethane	ND	0/3	--	--	--	--
1,2-Dichloroethane	ND	0/3	--	--	--	--
1,2-Dichloropropane	ND	0/3	--	--	--	--
Di(2-ethylhexyl) phthalate	330	3/10	1/10	NA	40	RMEG

Table 4. Maximum Concentrations in On-site Subsurface Soil, continued

Contaminants of Concern	Maximum Concentration (mg/kg)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range (mg/kg)	Comparison Value	
					(mg/kg)	Source
1,2-Diphenylhydrazine	1.0	3/3	1/3	NA	0.9	CREG
Hexachloroethane	ND	0/3	--	--	--	--
Lead	18	8/20	--	NA	None	Carcinogen
Manganese	9.3	5/10	0/10	NA	10	RMEG
Mercury	0.1	3/20	0/20	NA	4	EMEG
Methylene Chloride	0.84	4/15	0/15	NA	90	CREG
Naphthalene	0.45	2/10	--	NA	None	None
Nickel	14	2/20	--	NA	None	Carcinogen
n-Nitrosodiphenylamine	ND	0/3	--	--	--	--
PCBs (total)	ND	0/3	--	--	--	--
Selenium	ND	0/3	--	--	--	--
1,1,2,2-Tetrachloroethane	ND	0/3	--	--	--	--
Tetrachloroethene	ND	0/3	--	--	--	--
Tin	40	1/3	--	NA	None	None
Trichloroethene	ND	0/3	--	--	--	--
Vinyl Chloride	ND	0/3	--	--	--	--

mg/kg - milligrams per kilogram
NA - not analyzed
ND - not detected

Data Sources: EPA 1985c, 1986d.

Table 5. Maximum Concentrations in On-site Sediment

Contaminants of Concern	Maximum Concentration Detected (mg/kg)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range (mg/kg)	Comparison Value	
					(mg/kg)	Source
Arsenic	2.1	1/4	1/4	NA	0.4	CREG
Barium	ND	0/4	--	--	--	--
Benzene	ND	0/1	--	--	--	--
Beryllium	ND	0/2	--	--	--	--
Bromodichloro-methane	ND	0/1	--	--	--	--
Cadmium	ND	0/2	--	--	--	--
Chlorobenzene	ND	0/1	--	--	--	--
Chlorodibromo-methane	ND	0/1	--	--	--	--
Chloroform	ND	0/1	--	--	--	--
Chromium(VI)	1.4	2/4	--	NA	None	Carcinogen
Cobalt	40	1/2	--	NA	None	None
Cresol	320	1/4	--	NA	None	None
Cyanide	1.4	1/4	0/4	NA	40	RMEG
DDT	ND	0/2	--	--	--	--
1,4-Dichlorobenzene	ND	0/2	--	--	--	--
1,1-Dichloroethane	ND	0/1	--	--	--	--
1,2-Dichloroethane	ND	0/1	--	--	--	--
1,2-Dichloropropane	ND	0/1	--	--	--	--
Di(2-ethylhexyl) phthalate	ND	0/2	--	--	--	--

Table 5. Maximum Concentrations in On-site Sediment, continued

Contaminants of Concern	Maximum Concentration (mg/kg)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range (mg/kg)	Comparison Value	
					(mg/kg)	Source
1,2-Diphenylhydrazine	0.4	2/2	0/2	NA	0.9	CREG
Hexachloroethane	ND	0/2	--	--	--	--
Lead	5.8	3/4	--	NA	None	Carcinogen
Manganese	1.9	2/4	0/4	NA	10	RMEG
Mercury	0.28	2/4	0/4	NA	4	EMEG
Methylene Chloride	ND	0/1	--	--	--	--
Naphthalene	ND	0/2	--	--	--	--
Nickel	ND	0/2	--	--	--	--
n-Nitrosodiphenylamine	ND	0/2	--	--	--	--
PCBs (total)	0.02	1/3	1/3	NA	0.01	EMEG
Selenium	ND	0/2	--	--	--	--
1,1,2,2-Tetrachloroethane	ND	0/1	--	--	--	--
Tetrachloroethene	ND	0/1	--	--	--	--
Tin	ND	0/2	--	--	--	--
Trichloroethene	ND	0/3	--	--	--	--
Vinyl Chloride	ND	0/1	--	--	--	--

mg/kg - milligrams per kilogram
NA - not analyzed
ND - not detected

Data Sources: BESD 1983a; EPA 1985c, 1986d.

Table 6. Maximum Concentrations in On-site Surface Water

Contaminants of Concern	Maximum Concentration Detected (µg/l)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
Arsenic	ND	0/2	--	--	--	--
Barium	ND	0/4	--	--	--	--
Benzene	ND	0/2	--	--	--	--
Beryllium	ND	0/2	--	--	--	--
Bromodichloromethane	ND	0/2	--	--	--	--
Cadmium	ND	0/2	--	--	--	--
Chlorobenzene	ND	0/2	--	--	--	--
Chlorodibromomethane	ND	0/2	--	--	--	--
Chloroform	ND	0/2	--	--	--	--
Chromium(VI)	ND	0/4	--	--	--	--
Cobalt	ND	0/4	--	--	--	--
Cresol	ND	0/2	--	--	--	--
Cyanide	ND	0/4	--	--	--	--
DDT	0.13	1/4	1/4	NA	0.1	CREG
1,4-Dichlorobenzene	ND	0/2	--	--	--	--
1,1-Dichloroethane	ND	0/2	--	--	--	--
1,2-Dichloroethane	ND	0/2	--	--	--	--
1,2-Dichloropropane	ND	0/2	--	--	--	--
Di(2-ethylhexyl) phthalate	ND	0/2	--	--	--	--

Table 6. Maximum Concentrations in On-site Surface Water, continued

Contaminants of Concern	Maximum Concentration (µg/l)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
1,2-Diphenylhydrazine	ND	0/2	--	--	--	--
Hexachloroethane	ND	0/2	--	--	--	--
Lead	ND	0/4	--	--	--	--
Manganese	41	2/4	0/4	NA	50	RMEG
Mercury	0.33	1/2	0/2	NA	2	LTHA
Methylene Chloride	ND	0/2	--	--	--	--
Naphthalene	ND	0/2	--	--	--	--
Nickel	ND	0/2	--	--	--	--
n-Nitrosodiphenylamine	ND	0/2	--	--	--	--
PCBs (total)	ND	0/3	--	--	--	--
Selenium	ND	0/2	--	--	--	--
1,1,2,2-Tetrachloroethane	ND	0/2	--	--	--	--
Tetrachloroethene	ND	0/2	--	--	--	--
Tin	39	2/2	--	NA	None	None
Trichloroethene	ND	0/2	--	--	--	--
Vinyl Chloride	ND	0/2	--	--	--	--

µg/l - micrograms per liter
NA - not analyzed
ND - not detected

Data Sources: BESD 1983a; EPA 1985c, 1986d.

Table 7. Maximum Concentrations in On-site Boreholes and Monitor Wells

Contaminants of Concern	Maximum Concentration Detected (μg/l)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range (μg/l)	Comparison Value	
					(μg/l)	Source
Arsenic	220	7/31	7/31	ND	0.02	CREG
Barium	3,900	6/9	2/9	ND	700	RMEG
Benzene	408	14/45	13/45	ND	1	CREG
Beryllium	19	2/27	2/27	NA	0.008	CREG
Bromodichloro-methane	ND	0/10	--	--	--	--
Cadmium	1400	9/33	--	ND	None	Carcinogen
Chlorobenzene	297	16/42	2/42	ND	200	RMEG
Chlorodibromo-methane	ND	0/10	--	--	--	--
Chloroform	TR	1/18	u/18	ND	6	CREG
Chromium(VI)	1,100	13/33	--	ND	None	Carcinogen
Cobalt	42	2/9	--	NA	None	None
Cresol	5.7	2/11	--	NA	None	None
Cyanide	310	1/12	1/12	NA	200	RMEG
DDT	ND	0/13	--	--	--	--
1,4-Dichlorobenzene	39.3	4/24	--	ND	None	Carcinogen
1,1-Dichloroethane	24.9	1/14	--	ND	None	Carcinogen
1,2-Dichloroethane	6	2/14	1/14	ND	0.4	CREG
1,2-Dichloropropane	ND	0/10	--	--	--	--
Di(2-ethylhexyl) phthalate	96	2/26	2/26	ND	3	CREG

Table 7. Maximum Concentrations in On-site Boreholes and Monitor Wells, continued

Contaminants of Concern	Maximum Concentration (µg/l)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
1,2-Diphenylhydrazine	ND	0/7	--	--	--	--
Hexachloroethane	ND	0/5	--	--	--	--
Lead	5,300	15/39	--	ND-5.4	None	Carcinogen
Manganese	1,400	10/24	7/24	5-58	50	RMEG
Mercury	18	6/23	2/23	ND	2	LTHA
Methylene Chloride	4.8	1/38	0/38	ND	5	CREG
Naphthalene	16	3/15	0/15	ND	20	LTHA
Nickel	370	11/32	--	ND	None	Carcinogen
n-Nitrosodiphenylamine	17	3/9	3/9	ND	7	CREG
PCBs (total)	74	3/19	3/19	ND	0.005	CREG
Selenium	27	1/16	1/16	ND	20	EMEG
1,1,2,2-Tetrachloroethane	ND	0/12	--	--	--	--
Tetrachloroethene	ND	0/29	--	--	--	--
Tin	ND	0/1	--	--	--	--
Trichloroethene	ND	0/26	--	--	--	--
Vinyl Chloride	32	1/26	1/26	ND	0.2	EMEG

µg/l - micrograms per liter
NA - not analyzed
ND - not detected
TR - trace amount
u - unknown (see text for an explanation)

Data Sources: Disposal Safety 1990; EPA 1985a, 1986d; Golder Associates 1990.

Table 8. Maximum Concentrations in On-site Private Wells

Contaminants of Concern	Maximum Concentration Detected (µg/l)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
Arsenic	1.9	2/6	2/6	ND	0.02	CREG
Barium	59	6/9	0/9	ND	700	RMEG
Benzene	ND	0/12	--	--	--	--
Beryllium	ND	0/6	--	--	--	--
Bromodichloro-methane	ND	0/14	--	--	--	--
Cadmium	21	1/9	--	ND	None	Carcinogen
Chlorobenzene	ND	0/20	--	--	--	--
Chlorodibromo-methane	ND	0/12	--	--	--	--
Chloroform	0.5	4/15	0/15	ND	6	CREG
Chromium(VI)	ND	0/6	--	--	--	--
Cobalt	NA	NA	--	--	--	--
Cresol	NA	NA	--	--	--	--
Cyanide	NA	NA	--	--	--	--
DDT	NA	NA	--	--	--	--
1,4-Dichlorobenzene	ND	0/5	--	--	--	--
1,1-Dichloroethane	ND	0/12	--	--	--	--
1,2-Dichloroethane	ND	0/15	--	--	--	--
1,2-Dichloropropane	ND	0/10	--	--	--	--
Di(2-ethylhexyl) phthalate	ND	0/3	--	--	--	--

Table 8. Maximum Concentrations in On-site Private Wells, continued

Contaminants of Concern	Maximum Concentration (µg/l)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
1,2-Diphenylhydrazine	NA	NA	--	--	--	--
Hexachloroethane	NA	NA	--	--	--	--
Lead	690	3/9	--	ND-5.4	None	Carcinogen
Manganese	34	8/9	0/9	5-58	50	RMEG
Mercury	1.13	1/6	0/6	ND	2	LTHA
Methylene Chloride	5,700	1/15	1/15	ND	5	CREG
Naphthalene	NA	NA	--	--	--	--
Nickel	17	2/9	--	ND	None	Carcinogen
n-Nitrosodiphenylamine	NA	NA	--	--	--	--
PCBs (total)	NA	NA	--	--	--	--
Selenium	ND	0/6	--	--	--	--
1,1,2,2-Tetrachloroethane	ND	0/12	--	--	--	--
Tetrachloroethene	ND	0/12	--	--	--	--
Tin	16	1/3	--	NA	None	None
Trichloroethene	ND	0/12	--	--	--	--
Vinyl Chloride	ND	0/12	--	--	--	--

µg/l - micrograms per liter
NA - not analyzed
ND - not detected

Data Sources: CompuChem 1988; Disposal Safety 1990; EPA 1986d; FHRS 1981b, 1983c.

Table 9. Maximum Concentrations in Off-site Sediment

Contaminants of Concern	Maximum Concentration Detected (mg/kg)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range (mg/kg)	Comparison Value	
					(mg/kg)	Source
Arsenic	ND	0/4	--	--	--	--
Barium	1.0	1/4	0/4	NA	100	RMEG
Benzene	NA	NA	--	--	--	--
Beryllium	NA	NA	--	--	--	--
Bromodichloromethane	NA	NA	--	--	--	--
Cadmium	NA	NA	--	--	--	--
Chlorobenzene	NA	NA	--	--	--	--
Chlorodibromomethane	NA	NA	--	--	--	--
Chloroform	NA	NA	--	--	--	--
Chromium(VI)	2.9	1/4	--	NA	None	Carcinogen
Cobalt	NA	NA	--	--	--	--
Cresol	310	1/3	--	NA	None	None
Cyanide	ND	0/4	--	--	--	--
DDT	NA	NA	--	--	--	--
1,4-Dichlorobenzene	NA	NA	--	--	--	--
1,1-Dichloroethane	NA	NA	--	--	--	--
1,2-Dichloroethane	NA	NA	--	--	--	--
1,2-Dichloropropane	NA	NA	--	--	--	--
Di(2-ethylhexyl) phthalate	NA	NA	--	--	--	--

Table 9. Maximum Concentrations in Off-site Sediment, continued

Contaminants of Concern	Maximum Concentration (mg/kg)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range (mg/kg)	Comparison Value	
					(mg/kg)	Source
1,2-Diphenylhydrazine	NA	NA	--	--	--	--
Hexachloroethane	NA	NA	--	--	--	--
Lead	120	1/4	--	NA	None	Carcinogen
Manganese	ND	0/4	--	--	--	--
Mercury	ND	0/4	--	--	--	--
Methylene Chloride	NA	NA	--	--	--	--
Naphthalene	NA	NA	--	--	--	--
Nickel	NA	NA	--	--	--	--
n-Nitrosodiphenylamine	NA	NA	--	--	--	--
PCBs (total)	0.02	1/1	1/1	NA	0.01	EMEG
Selenium	NA	NA	--	--	--	--
1,1,2,2-Tetrachloroethane	NA	NA	--	--	--	--
Tetrachloroethene	NA	NA	--	--	--	--
Tin	NA	NA	--	--	--	--
Trichloroethene	ND	0/4	--	--	--	--
Vinyl Chloride	NA	NA	--	--	--	--

mg/kg - milligrams per kilogram
NA - not analyzed
ND - not detected

Data Sources: BESD 1983a; EPA 1986d.

Table 10. Maximum Concentrations in Off-site Surface Water

Contaminants of Concern	Maximum Concentration Detected (µg/l)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
Arsenic	NA	NA	--	--	--	--
Barium	ND	0/4	--	--	--	--
Benzene	NA	NA	--	--	--	--
Beryllium	NA	NA	--	--	--	--
Bromodichloro-methane	NA	NA	--	--	--	--
Cadmium	NA	NA	--	--	--	--
Chlorobenzene	NA	NA	--	--	--	--
Chlorodibromo-methane	NA	NA	--	--	--	--
Chloroform	NA	NA	--	--	--	--
Chromium(VI)	17	2/4	--	NA	None	Carcinogen
Cobalt	4.3	1/4	--	NA	None	None
Cresol	NA	NA	--	--	--	--
Cyanide	ND	0/4	--	--	--	--
DDT	ND	0/4	--	--	--	--
1,4-Dichlorobenzene	NA	NA	--	--	--	--
1,1-Dichloroethane	NA	NA	--	--	--	--
1,2-Dichloroethane	NA	NA	--	--	--	--
1,2-Dichloropropane	NA	NA	--	--	--	--
Di(2-ethylhexyl) phthalate	NA	NA	--	--	--	--

Table 10. Maximum Concentrations in Off-site Surface Water, continued

Contaminants of Concern	Maximum Concentration (µg/l)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
1,2-Diphenylhydrazine	NA	NA	--	--	--	--
Hexachloroethane	NA	NA	--	--	--	--
Lead	31	1/4	--	NA	None	Carcinogen
Manganese	40	4/4	0/4	NA	50	RMEG
Mercury	NA	NA	--	--	--	--
Methylene Chloride	NA	NA	--	--	--	--
Naphthalene	NA	NA	--	--	--	--
Nickel	NA	NA	--	--	--	--
n-Nitrosodiphenylamine	NA	NA	--	--	--	--
PCBs (total)	0.3	1/1	1/1	NA	0.005	CREG
Selenium	NA	NA	--	--	--	--
1,1,2,2-Tetrachloroethane	NA	NA	--	--	--	--
Tetrachloroethene	NA	NA	--	--	--	--
Tin	ND	0/11	--	--	--	--
Trichloroethene	NA	NA	--	--	--	--
Vinyl Chloride	NA	NA	--	--	--	--

µg/l - micrograms/liter
NA - not analyzed
ND - not detected

Data Sources: BESD 1983a; EPA 1986d.

Table 11. Maximum Concentrations in Off-site Monitor Wells

Contaminants of Concern	Maximum Concentration Detected (μg/l)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range (μg/l)	Comparison Value	
					(μg/l)	Source
Arsenic	30	5/64	5/64	ND	0.02	CREG
Barium	110	14/20	0/20	ND	700	RMEG
Benzene	8.42	26/200	24/200	ND	1	CREG
Beryllium	25	1/35	1/35	NA	0.008	CREG
Bromodichloro-methane	ND	0/60	--	--	--	--
Cadmium	10	9/85	2/85	ND	7	EMEG
Chlorobenzene	23	30/205	0/205	ND	200	RMEG
Chlorodibromo-methane	1.5	1/72	1/72	ND	0.4	CREG
Chloroform	13	9/102	4/102	ND	6	CREG
Chromium(VI)	74	44/84	--	ND	None	Carcinogen
Cobalt	3	1/15	--	NA	None	None
Cresol	46	3/18	--	NA	None	None
Cyanide	ND	0/24	--	--	--	--
DDT	ND	0/37	--	--	--	--
1,4-Dichlorobenzene	48.5	19/165	--	ND	None	Carcinogen
1,1-Dichloroethane	43	6/99	--	ND	None	Carcinogen
1,2-Dichloroethane	33	5/99	4/99	ND	0.4	CREG
1,2-Dichloropropane	ND	0/60	--	--	--	--
Di(2-ethylhexyl) phthalate	79	8/123	8/123	ND	3	CREG

Table 11. Maximum Concentrations in Off-site Monitor Wells, continued

Contaminants of Concern	Maximum Concentration (µg/l)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
1,2-Diphenylhydrazine	ND	0/26	--	--	--	--
Hexachloroethane	ND	0/32	--	--	--	--
Lead	68	48/107	--	ND-5.4	None	Carcinogen
Manganese	230	23/60	9/60	5-58	50	RMEG
Mercury	0.27	7/56	0/56	ND	2	LTHA
Methylene Chloride	106.6	2/197	2/197	ND	5	CREG
Naphthalene	30	16/69	5/69	ND	20	LTHA
Nickel	74	32/87	--	ND	None	Carcinogen
n-Nitrosodiphenylamine	14	1/48	1/48	ND	7	CREG
PCBs (total)	ND	0/40	--	--	--	--
Selenium	15	1/37	0/37	ND	20	EMEG
1,1,2,2-Tetrachloroethane	36.8	4/78	2/78	ND	0.2	CREG
Tetrachloroethene	3	5/196	2/196	ND	0.7	CREG
Tin	ND	0/2	--	--	--	--
Trichloroethene	11	6/195	3/195	ND	3	CREG
Vinyl Chloride	73	19/195	19/195	ND	0.2	EMEG

µg/l - micrograms per liter
NA - not analyzed
ND - not detected

Data Sources: Disposal Safety 1990; EPA 1985a, 1986d; FDER 1983c; Golder Associates 1990, 1992, 1993a.

Table 12. Maximum Concentrations in Off-site Private Wells

Contaminants of Concern	Maximum Concentration Detected (µg/l)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
Arsenic	ND	0/50	--	--	--	--
Barium	35	3/64	0/65	ND	700	RMEG
Benzene	2	13/126	3/126	ND	1	CREG
Beryllium	ND	0/4	--	--	--	--
Bromodichloromethane	5.2	2/137	1/137	ND	0.6	CREG
Cadmium	ND	0/70	--	--	--	--
Chlorobenzene	ND	0/184	--	--	--	--
Chlorodibromomethane	1.7	2/118	2/118	ND	0.4	CREG
Chloroform	12	3/123	2/123	ND	6	CREG
Chromium(VI)	ND	0/58	--	--	--	--
Cobalt	NA	--	--	--	--	--
Cresol	14	2/12	--	NA	None	None
Cyanide	ND	0/2	--	--	--	--
DDT	ND	0/4	--	--	--	--
1,4-Dichlorobenzene	5	2/80	--	ND	None	Carcinogen
1,1-Dichloroethane	5	20/114	--	ND	None	Carcinogen
1,2-Dichloroethane	5.2	3/121	1/121	ND	0.4	CREG
1,2-Dichloropropane	< 5	2/99	--	ND	None	Carcinogen
Di(2-ethylhexyl) phthalate	8.3	2/51	2/51	ND	3	CREG

Table 12. Maximum Concentrations in Off-site Private Wells, continued

Contaminants of Concern	Maximum Concentration (µg/l)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range (µg/l)	Comparison Value	
					(µg/l)	Source
1,2-Diphenylhydrazine	ND	0/35	--	--	—	--
Hexachloroethane	6	1/37	1/37	ND	3	CREG
Lead	46	8/73	--	ND-5.4	None	Carcinogen
Manganese	140	65/70	6/70	5-58	50	RMEG
Mercury	0.27	1/59	0/59	ND	2	LTHA
Methylene Chloride	8	15/135	3/135	ND	5	CREG
Naphthalene	ND	0/37	--	--	--	--
Nickel	26	8/32	--	ND	None	Carcinogen
n-Nitrosodiphenylamine	ND	0/37	--	--	--	--
PCBs (total)	ND	0/37	--	--	--	--
Selenium	ND	0/59	--	--	--	--
1,1,2,2-Tetrachloroethane	ND	0/112	--	--	--	--
Tetrachloroethene	1	1/120	1/120	ND	0.7	CREG
Tin	ND	0/13	--	--	--	--
Trichloroethene	5	7/123	1/123	ND	3	CREG
Vinyl Chloride	3	27/122	22/122	ND	0.2	EMEG

µg/l - micrograms per liter
NA - not analyzed
ND - not detected

Data Sources: Disposal Safety 1990; EPA 1985c, 1986d; FHRS 1981b, 1983c, 1984, 1990, 1991, 1992, 1993d.

Table 13. Maximum Concentrations Predicted in Off-site Air

Contaminants of Concern	Maximum Concentration Predicted ($\mu g/m^3$)	Total # Detected/ Total # Samples	Total # Exceeding Comparison Value/ Total # Samples	Background Concentration Range ($\mu g/m^3$)	Comparison Value	
					($\mu g/m^3$)	Source
Arsenic	NA	NA	--	--	--	--
Barium	NA	NA	--	--	--	--
Benzene	0.07	20/31	20/31	ND	0.1	CREG
Beryllium	NA	NA	--	--	--	--
Bromodichloromethane	ND	0/31	--	--	--	--
Cadmium	NA	NA	--	--	--	--
Chlorobenzene	0.1	20/31	--	ND	None	None
Chlorodibromomethane	ND	0/31	--	--	--	--
Chloroform	ND	0/31	--	--	--	--
Chromium(VI)	NA	NA	--	--	--	--
Cobalt	NA	NA	--	--	--	--
Cresol	NA	NA	--	--	--	--
Cyanide	NA	NA	--	--	--	--
DDT	NA	NA	--	--	--	--
1,4-Dichlorobenzene	NA	NA	--	NA	--	--
1,1-Dichloroethane	0.044	18/31	--	ND	None	Carcinogen
1,2-Dichloroethane	0.05	3/31	3/31	ND	0.04	CREG
1,2-Dichloropropane	0.005	1/31	--	ND	None	None
Di(2-ethylhexyl) phthalate	NA	NA	--	--	--	--

Table 13. Maximum Concentrations Predicted in Off-site Air, continued

Contaminants of Concern	Maximum Concentration Predicted ($\mu g/m^3$)	Total # Detected/ Total # Sampled	Total # Exceeding Comparison Value/ Total # Sampled	Background Concentration Range ($\mu g/m^3$)	Comparison Value	
					($\mu g/m^3$)	Source
1,2-Diphenylhydrazine	NA	NA	--	--	--	--
Hexachloroethane	NA	NA	--	--	--	--
Lead	NA	NA	--	--	--	--
Manganese	NA	NA	--	--	--	--
Mercury	NA	NA	--	--	--	--
Methylene Chloride	0.15	26/31	22/31	ND-1.8	2	CREG
Naphthalene	NA	NA	--	--	--	--
Nickel	NA	NA	--	--	--	--
n-Nitrosodiphenylamine	NA	NA	--	--	--	--
PCBs (total)	NA	NA	--	--	--	--
Selenium	NA	NA	--	--	--	--
1,1,2,2-Tetrachloroethane	ND	0/31	--	--	--	--
Tetrachloroethene	0.007	2/31	2/31	ND	2	CREG
Tin	NA	NA	--	--	--	--
Trichloroethene	0.02	12/31	12/31	ND	0.6	CREG
Vinyl Chloride	0.16	20/31	--	ND	None	Carcinogen

$\mu g/m^3$ - micrograms per cubic meter
NA - not analyzed
ND - not detected

Data Source: Golder Associates 1993a.

Table 14. Completed Exposure Pathways

Pathway Name	Exposure Pathway Elements					Time
	Source	Environmental Media	Point of Exposure	Route of Exposure	Exposed Population	
Subsurface Soil	Hipps Road Landfill	Subsurface Soil	Fill Material	Ingestion, Skin Absorption	Nearby Residents	Past
Sediment	Hipps Road Landfill	Sediment	Ponds by the Landfill, Storm Water Swales	Ingestion, Skin Absorption	Nearby Residents	Past, Present, Future
Surface Water	Hipps Road Landfill	Surface Water	Ponds by the Landfill, Storm Water Swales	Ingestion, Skin Absorption, Inhalation of Vapors	Nearby Residents	Past
Shallow Ground-water	Hipps Road Landfill	Groundwater	Private Wells	Ingestion, Skin Absorption, Inhalation of Vapors	Nearby Residents	Past, Present, Future

Table 14. Completed Exposure Pathways, continued

Pathway Name	Exposure Pathway Elements					Time
	Source	Environmental Media	Point of Exposure	Route of Exposure	Exposed Population	
Air (Tower Effluent)	Hipps Road Landfill	Air	Ambient Air	Inhalation	Nearby Residents	Present, Future

Table 15. Potential Exposure Pathways

Pathway Name	Exposure Pathway Elements					Time
	Source	Environmental Media	Point of Exposure	Route of Exposure	Exposed Population	
Surface Soil	Hipps Road Landfill	Surface Soils	Yards, Landfill	Ingestion, Skin Absorption, Inhalation of Dust	Nearby Residents	Past, Present, Future
Surface Water	Hipps Road Landfill	Surface Water	Storm Water Swales, Creek	Ingestion, Skin Absorption	Nearby Residents	Present, Future
Air (Odor)	Hipps Road Landfill	Air	Ambient Air	Inhalation	Nearby Residents	Past, Present, Future
Biota	Hipps Road Landfill	Animal/Plant Tissue	Ponds by the Landfill, Small Game, Gardens	Ingestion	Nearby Residents	Past, Present, Future

Table 16. Parameters Used for Ingestion Dose Calculations for Hypothetical Individuals

Parameter	Hypothetical Individual		
	Adult	Average Child	Young Child
Age	18 y and older	0-18 y	0-6 y
Pica Behavior	No	No	Yes
Body Weight	70 kg	35 kg	13 kg
Lifetime Expectancy	70 y	70 y	70 y
Drinking Water Ingestion Rate	2.0 L/d	1.0 L/d	1.0 L/d
Drinking Water Ingestion Frequency	350 d/y	350 d/y	350 d/y
Contaminated Fraction of Drinking Water	1.00	1.00	1.00
Exposure Period for Drinking Water Ingestion	26 y	18 y	6 y
Homegrown Vegetables Ingestion Rate	0.20 kg/d	0.20 kg/d	0.10 kg/d
Homegrown Vegetable Ingestion Frequency	350 d/y	350 d/y	350 d/y
Contaminated Fraction of Homegrown Vegetables Due To Groundwater	0.40	0.40	0.40
Contaminated Fraction of Homegrown Vegetables Due To Soil	0.40	0.40	0.40
Exposure Period for Ingesting Homegrown Vegetables	26 y	18 y	6 y
Soil/Sediment Ingestion Rate	100 mg/d	200 mg/d	5,000 mg/d
Soil Ingestion Frequency	181-350 d/y	181-350 d/y	181-350 d/y
Sediment Ingestion Frequency	169 d/y	169 d/y	169 d/y
Contaminated Fraction of Soil/Sediment	1.00	1.00	1.00
Exposure Period for Soil/Sediment Ingestion	26 y	18 y	6 y

Table 16. Parameters Used for Ingestion Dose Calculations for Hypothetical Individuals, continued

Parameter	Hypothetical Individual		
	Adult	Average Child	Young Child
Fish Ingestion Rate	0.05 kg/d	0.03 kg/d	0.02 kg/d
Fish Ingestion Frequency	120 d/y	120 d/y	120 d/y
Contaminated Fraction of Fish	1.00	1.00	1.00
Exposure Period for Fish Ingestion	22 y	18 y	6 y
Incidental Ingestion Rate While Swimming	50 ml/event	50 ml/event	--
Swimming Event Frequency	39 events/y	78 events/y	--
Swimming Event Duration	1 h/event	1 h/event	--
Exposure Period for Swimming	22 y	12 y	--

y - year
d - day
h - hour
L - liter
kg - kilogram
mg - milligram
ml - milliliter

Table 17. Parameters Used for Inhalation Dose Calculations for Hypothetical Individuals

Parameter	Hypothetical Individual		
	Adult	Average Child	Young Child
Age	18 y and older	0-18 y	0-6 y
Body Weight	70 kg	35 kg	13 kg
Lifetime Expectancy	70 y	70 y	70 y
Inhalation Rate While Showering	0.65 m^3/h	1.00 m^3/h	0.80 m^3/h
Showering Frequency	350 d/y	350 d/y	350 d/y
Shower Length	0.25 h/d	0.5 h/d	0.5 h/d
Post-Shower Length	0.20 h/d	0.25 h/d	0.25 h/d
Exposure Period for Showering	26 y	18 y	6 y
Inhalation Rate While Inside the Home	0.71 m^3/h	0.81 m^3/h	0.60 m^3/h
Frequency Inside the Home	350 d/y	350 d/y	350 d/y
Length of Time Inside the Home	21.0 h/d	21.0 h/d	21.0 h/d
Exposure Period for Being Inside the Home	26 y	18 y	6 y
Inhalation Rate While Outside the Home	1.67 m^3/h	1.87 m^3/h	1.60 m^3/h
Frequency Outside the Home	350 d/y	350 d/y	350 d/y
Length of Time Outside the Home	3.0 h/d	3.0 h/d	3.0 h/d
Exposure Period for Being Outside the Home	26 y	18 y	6 y

y - year
d - day
h - hour
kg - kilogram
m^3 - cubic meter

Table 18. Parameters Used for Dermal Dose Calculations for Hypothetical Individuals

Parameter	Hypothetical Individual		
	Adult	Average Child	Young Child
Age	18 y and older	0-18 y	0-6 y
Body Weight	70 kg	35 kg	13 kg
Body Surface Area	19,400 cm^2	10,500 cm^2	7,280 cm^2
Lifetime Expectancy	70 y	70 y	70 y
Showering Frequency	350 d/y	350 d/y	350 d/y
Shower Length	0.25 h/d	0.5 h/d	0.5 h/d
Exposure Period for Showering	26 y	18 y	6 y
Swimming Event Frequency	39 events/y	78 events/y	--
Swimming Event Duration	1 h/event	1 h/event	--
Exposure Period for Swimming	22 y	12 y	--

y - year
d - day
h - hour
kg - kilogram
cm^2 - square centimeter

Table 19. Constants Used for Dose Calculations

Activity	Parameter	Value
Inhalation of Vapors While Showering	Bathroom Volume	9 m^3
	Flow Rate of Shower Water	600 L/h
	Fraction of Contaminant Volatilized	0.75
Inhalation of Vapors Inside the Residence	Water Flow Through the House	723 L/d
	Fraction of Contaminant Volatilized	0.50
	House Volume	177.70 m^3
	Mixing Coefficient	0.15
	Air Exchange Rate	13.7 house volumes/d
Inhalation of Vapors Outside the Residence	Flow of Irrigation Water	600 L/h
	Fraction of Contaminant Volatilized	0.50
	Length or Width of Approximate Square of Irrigated Area	10 m
	Stability Constant a	0.15
	Stability Constant b	0.75
	Near-surface Wind Speed	2.0 m/s

d - day
h - hour
s - second
m - meter
m^3 - cubic meter
L - liter

Table 20. Contaminants Violating Secondary Drinking Water Standards

Contaminant	On-site Boreholes or Monitoring Wells (mg/l)	Off-site Private Wells (mg/l)	MCL (mg/l)
Aluminum	ND-1500	ND-0.250	0.2
Copper	ND-1200	ND-1.1	1
Iron	ND-280	ND-7.92	0.3
pH	3.5-7.25	6.0-7.6	6.5-8.5

mg/l - milligrams per liter
ND - not detected

C. Acronyms

Commonly Used Acronyms

ATSDR **Agency for Toxic Substances and Disease Registry** - An organization within the federal Department of Health and Human Services that is responsible for conducting public health assessments at NPL sites. In Florida, this responsibility has been delegated to FHRS.

BESD **Bio-Environmental Services Division** - The branch of the City of Jacksonville's government that investigates pollution problems within the city limits.

CERCLA **Comprehensive Environmental Response, Compensation, and Liability Act** - A federal law passed in 1980 and amended in 1986 that created a trust fund, known as "Superfund", to investigate and clean up abandoned or uncontrolled hazardous waste sites.

CPHU **County Public Health Unit** - Often known as the county health department. The CPHU is associated with FHRS, and is responsible for investigating contamination in private drinking water wells.

CREG **Cancer Risk Evaluation Guide** - The contaminant concentration that is estimated to result in no more than one excess cancer per one million persons exposed over a lifetime.

EPA **U.S. Environmental Protection Agency** - The federal agency responsible for pollution control, including the investigation and cleanup of abandoned or uncontrolled hazardous waste sites.

FCDS **Florida Cancer Data System** - A FHRS program operated by the University of Miami School of Medicine that covers all cancers reported in Florida between 1981 and 1987.

FDER **Florida Department of Environmental Regulation** - The state agency responsible for pollution control, including the investigation and cleanup of abandoned or uncontrolled hazardous waste sites.

FHRS **Florida Department of Health and Rehabilitative Services** - The state agency responsible for investigating public health issues and running public health programs.

FS **Feasibility Study** - An EPA study that establishes cleanup criteria and identifies cleanup alternatives at a NPL site, based on the results of the remedial investigation.

JCACW	**Jacksonville Citizens Against Contaminated Water** - A group organized by residents living near site to voice community concerns to government officials.
HARP	**Health Activities Recommendation Panel** - A group within ATSDR that reviews public health assessments determines the need for specific follow-up health actions.
IARC	**International Agency for Research on Cancer** - An organization that evaluates the cancer risk from exposure to different chemicals.
IRIS	**Integrated Risk Information System** - An EPA computer database containing toxicological information. This database is updated monthly.
MRL	**Minimal Risk Level** - An estimate of the daily dose of a contaminant below which non-cancer illnesses are unlikely to occur. ATSDR develops MRL values through its research programs.
NOAEL	**No Observed Adverse Effects Level** -The highest experimental dose or exposure level at which there is no statistically or biologically significant increase in adverse effects.
NPL	**National Priorities List** - EPA's list of the most serious abandoned or uncontrolled hazardous waste sites, identified for clean up using CERCLA monies. These sites are also known as "Superfund" sites and are said to be on the "Superfund list".
NTP	**National Toxicology Program** - An organization within the federal Department of Health and Human Services that evaluates the cancer risk from exposure to different chemicals.
pH	A number indicating how acidic or caustic a substance is; the lower the pH, the more acidic the substance.
PRP	**Potentially Responsible Party** - An individual or company potentially responsible for, or contributors to, the contamination problems at a NPL site.
RfD	**Reference Dose** - An estimate of the daily dose of a contaminant below which non-cancer illnesses are unlikely to occur. EPA develops RfD values through its research programs.
RI	**Remedial Investigation** - An EPA study that identifies the nature and extent of hazardous waste contamination at a NPL site.

RI/FS	**Remedial Investigation/Feasibility Study** - The combined investigative and analytical studies that identify the nature and extent of hazardous waste contamination at a NPL site, and proposes cleanup alternatives.
ROD	**Record of Decision** - An EPA document that explains which cleanup alternative will be used at a NPL site, based on information generated during the RI/FS.
TRI	**Toxics Release Inventory** - A summary of chemical releases to the environment reported by industries to EPA.
VOCs	**Volatile Organic Compounds** - Organic chemical compounds that evaporate easily into the air. In sample data, these compounds are most often found within groups called "purgeables". In this document, VOCs are commonly referred to as "solvents".

D. Public Comments

Summary of Public Comments and Florida HRS Responses
Draft Public Health Assessment

From October 19-21, 1994, we sent 24 copies of the draft Hipps Road Landfill Public Health Assessment to community leaders, government officials, the PRP, the document repository, and a local grocery store for document access and public review. On November 17, 1994, we held a public meeting to present the findings of the draft public health assessment and to gather the public's comments on the draft document. To announce this meeting, we included a meeting announcement/health assessment fact sheet in the front of each document copy we distributed, and community leaders delivered 900 fact sheets to nearby residences. In addition, FHRS' public information staff contacted media representatives in Jacksonville and, on November 17, the Florida Times-Union (Jacksonville) published a story about the draft health assessment and announced the meeting. Approximately 80 adults attended the public meeting, 24 of whom gave comments on both health-related and nonhealth-related issues. We also solicited public comments by mail through December 16. As of December 23, we had received nine written responses, mostly from area residents. The following is a summary of the public's comments (in bold) and our responses:

1. **One resident commented that landfill operation began in 1967, not 1965. This had been established in one of the court cases involving the landfill. Another resident commented that not all of the houses on site were demolished - some were moved to other locations. Another individual recommended a few other minor, factual changes to the text.**

   Thank you for the comments; we have made appropriate corrections in the text. Because the first comment involves a date change, it also lessens the exposure period and our estimates for increased cancer risk. Therefore, we have changed the information in Tables 16-18 to reduce the number of years of potential exposure.

2. **One resident commented that children used to swim in the cypress pond, but no longer do so.**

   Thank you for this correction. We have changed the text in Conclusion #6 and reduced the recommended sampling frequency in Recommendation #6 to reflect this fact.

3. **Several residents reported health problems from present-day exposure to their private well water. These problems include: nausea, diarrhea, headaches, burning eyes while showering, cataracts, ear problems, sinus problems, thyroid disease, blood poisoning, bad nerves, liver problems, skin rashes, itching skin, allergies, back problems, difficulty healing after surgery, heart attacks, strokes, cancer, and other health problems. Many residents are worried that their children's drinking and bathing in this water will harm their health.**

We have added burning eyes while showering, itching skin, blood poisoning, and difficulty healing after surgery to the health problems listed in the text. The other health problems were already reported.

We have not been able to identify any potential health problems in children or adults from present-day exposure to the groundwater. The Duval County Public Health Unit (CPHU) periodically monitors private wells in the community to ensure the water meets primary drinking water standards.

4. **One resident questioned the safety of the air stripper. When living next to the air stripper, this individual had headaches, sinus problems, and eye irritation whenever the air stripper operated. After this person moved to a home farther away from the air stripper, these symptoms persisted but to a lesser degree. This person had not experienced these problems prior to air stripper installation, and has not experienced them since it has been down for repairs. This individual wanted HRS staff to be aware of these effects, even though the HRS analysis found the air stripper was unlikely to harm a person's health.**

Although we did not find the emissions from the air stripper were likely to harm health, we've recommended that water going into the air stripper continue to be monitored to allow us to identify potential public health problems, should they develop in the future. We have asked EPA to share with us the sample data they've gathered since the trial run. We will re-evaluate these data if any contaminant concentrations measured exceed those measured during the trial run of the air stripper.

We have added eye irritation to the health problems listed in the text. The other health problems were already reported.

5. **A few residents questioned the cancer risk evaluation in the health assessment, believing the risk estimates to be much too low.**

We, too, are concerned about the numbers of illnesses, especially cancers, in the community. When we did this assessment, we erred on the side of protecting public health when we made our estimates. We did this by evaluating only the highest contaminant concentrations for evaluation and using the maximum exposure period that was reasonable. In other words, we always erred on the side of public health and estimated the worst case exposure. The greatest increased cancer risk we estimated for any one contaminant is 1 in 1,000. Nevertheless, we do not know all there is to know about cancer-causing chemicals, nor how all of the chemicals the community was exposed to interact. This is one reason we have recommended ATSDR perform some kind of health follow-up study. Other reasons are: our analysis indicates health problems, including cancer, could occur in the community; and, there is a large number of reported illnesses.

6. **One individual strongly disagreed with the methodology of the public health assessment, stating "the overall philosophical approach and methodology presented in the Draft PHA are fundamentally flawed." In particular, this individual believed the health assessment was too conservative in using worst-case assumptions, particularly by using maximum concentrations for each chemical; contradicted EPA guidelines in its use of worst-case assumptions, which is likely to result in a substantial overestimate of a potential problem; and contradicted ATSDR guidelines in its use of screening values (EMEGs) to predict health effects.**

As explained in the text (Public Health Implications Section, Uncertainty in Health Assessments), we did not have sufficient sampling data to know if the maximum values reported were the maximum values the residents were actually exposed to, nor did we have adequate geologic and sample data to predict peak values. We decided to err on the side of protecting public health by using maximum measured values in our analysis. We cannot know if the resulting risk estimates truly are upper-bound estimates. Nevertheless, we believe the risk of illness is unlikely to be larger than the risk we have estimated, and may be smaller. Our decisions and conclusions show that a public health assessment requires the use of scientific and professional judgement; we understand readers may not agree with all of our judgements.

The confusion between EPA risk assessments and public health assessments is understandable. An EPA risk assessment is used to support the selection of cleanup activities at a Superfund site. A public health assessment is a mechanism to assess any current or future public health impacts from the release of hazardous substances into the environment, provide the community with information on the public health implications of a specific site, identify those populations for whom further health actions or studies are needed, and make recommendations for actions needed to protect public health. In the public health assessment, we acknowledge the uncertainties of our assumptions and estimates (Public Health Implications Section, Uncertainty in Health Assessments) which may lead to an under- or over-estimate of the risk of illness, largely because of gaps in the data. We cannot know the magnitude or direction of our presumed errors without evaluating the very data that are missing.

The statement about our using EMEGs (or other comparable values) as predictors of health effects is incorrect. In accordance with ATSDR guidelines and as described in the text (Environmental Contamination and Other Hazards Section), we used ATSDR's standard comparison values to select contaminants of concern for further evaluation. The individual making the comment may be confused because when we selected one contaminant of concern in one medium, we reported that contaminant in all other media. This is why some contaminants of concern are below their comparison values for a particular medium in Tables 4-12. Careful examination of these tables will show each contaminant of concern to be above its standard comparison value in at least one of these tables, or to have no standard comparison values for initial screening. In addition, all

readers should note that the draft public health assessment underwent ATSDR technical review before being released as a public draft document.

7. **One resident asked about the physician education program conducted at St. Vincent's hospital in September 1994 and wanted to know how residents could find out the names of doctors who were interested in treating people who had been exposed to chemicals.**

   Information about the physician education program can be obtained from HRS by contacting:

   Ms. Julia Winter
   HRS/HSET
   1317 Winewood Boulevard
   Tallahassee, FL 32399-0700
   (904) 488-3385

8. **Many residents had questions and comments about the proposed follow-up health study. Suggestions about the type of study residents would like to see include: a study of the incidence of learning disabilities, cancer, and other health problems reported in the community in comparison with the incidence of these problems in a nonexposed community, a biomedical study of the health of past and present community residents, and a tracking of the health problems identified within the community in the present and in the future.**

   If ATSDR accepts the recommendation for a follow-up health study, HRS and ATSDR plan to meet with residents early in 1995 to discuss the options available and the community's needs for a health study.

9. **One resident asked about testing for radiation in water and soil.**

   EPA has not sampled the groundwater or soil for radioactivity. HRS has recommended radionuclides be measured in future water samples. The Duval CPHU recently began measuring radionuclides in the private well samples they collect from the Hipps Road area. So far, those sample results have come back negative. If radionuclides are found in the groundwater in the future, HRS will evaluate the need for testing surface water and soil for radioactivity.

10. **A few residents had questions about the adequacy of past sampling around the landfill. Specifically, why was most of the sampling confined to the areas northeast of the site?**

    Because groundwater near the site generally flows to the northeast, EPA has focused their sampling and cleanup efforts northeast of the site. However, we don't believe

contaminant movement in directions west, east, or south of the site has been fully described. We also don't believe enough surface soil, surface water, or sediment samples have been collected around the site. Therefore, in the health assessment, we have recommended EPA conduct additional sampling to further investigate potential environmental contamination around the site.

11. **One resident was concerned that contaminant plume boundaries had not changed since they were first delineated years ago.**

In 1989, site contractors collected information needed to delineate the boundaries of the contaminant plume, northeast of the site, in order to design the cleanup system (Golder 1990). We do not have the hydrogeological expertise to evaluate whether or not the contaminant plume boundaries have moved significantly since these data were evaluated; this issue is best addressed by EPA.

12. **One resident questioned the purpose and effectiveness of the clay cap. Another resident was concerned the site contractor had damaged the cap by driving heavy vehicles on it.**

The clay cap has three purposes. First, because the cap covers the fill material, it prevents these materials from being blown or washed off site. Second, the cap prevents the mounding of contaminated groundwater over the fill material, which subsequently can cause contaminants to flow away from the landfill in all directions. Third, because the cap keeps rainfall out of the fill material, it prevents downward movement of contaminants into the groundwater. However, the cap will not prevent contaminants, already dissolved in the groundwater, from flowing horizontally away from the site. We do not know if the landfill cap has been damaged; it is EPA's responsibility to ensure the cap is periodically checked and remains intact.

13. **A couple of people commented on the landfill's contents. One individual requested HRS to recommend a more complete source determination be made of the landfill's contents, so that proper sampling and cleanup can ensue.**

From a public health standpoint, contaminants found on site become important only if there is a point of human exposure to them. We believe that as long as the cap remains intact, the groundwater monitoring around the site continues, and EPA collects the additional environmental samples we have recommended, we will have adequate information to assess the potential public health threat from the site. Therefore, our findings do not support an additional need for further characterization of the landfill contents at this time. In the health assessment, we have recommended future uses of the site be restricted to those compatible with the remaining contamination. If site uses were to become incompatible, in terms of potential human exposure, we would consider recommending further site characterization work be performed. For example, if a developer wanted to build homes on the site in the near future, we would likely

recommend a complete source determination be conducted for the site. We do not have the expertise to evaluate whether or not further site characterization is needed to adequately clean up the site; this issue is best addressed by EPA.

14. **One individual reported difficulty in obtaining recent site documents from EPA, and requested HRS's help in obtaining recent sampling data.**

We have requested recent sampling information from EPA and will share whatever data we obtain with the public, upon request.

15. **Several residents had questions about testing of their private well water.**

The Duval CPHU is responsible for testing private wells in the Hipps Road area. Nearby residents should call the Duval CPHU, at 630-3272, if they want to have their well water tested. Presently, there is no charge for the sampling or analyses. It takes several weeks to get the water sample results back from the laboratory in Jacksonville.

16. **Many residents had questions about when hookup to city water on their streets would occur. Others had comments about the high cost of hookup.**

The community leaders have information about the schedule for bringing city water to the Hipps Road neighborhood and the availability of financial assistance for residents unable to pay for hookup.

17. **One resident asked about current activities at the site.**

The air stripper has been shut down since September because of filtration problems in one of the holding ponds. The construction equipment and materials are on site to fix the filtration problem.