NEW JERSEY DEPARTMENT of HEALTH and SENIOR SERVICES

Public Health Assessment

Ringwood Mines/Landfill Site
Ringwood
Passaic County

Public Comment Release

Public Comment Period:
May 1 - July 3, 2006

Prepared Under a Cooperative Agreement with the:

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

All comments must be submitted in writing to:

Health Assessment Project Manager
NJ Department of Health and Senior Services
Public Health Services Branch
Consumer and Environmental Health Services
PO Box 369
Trenton, NJ 08625-0369

THE ATSDR HEALTH ASSESSMENT: A NOTE OF EXPLANATION

Section 104 (i) (6) (F) of the Comprehensive Response, Compensation, and Liability Act of 1980 (CERCLA), as amended, states "...the term 'health assessment' shall include preliminary assessments of potential risks to human health posed by individual sites and facilities, based on such factors as the nature and extent of contamination, the existence of potential pathways of human exposure (including ground or surface water contamination, air emissions, and food chain contamination), the size and potential susceptibility of the community within the likely pathways of exposure, the comparison of expected human exposure levels to the short-term and long-term health effects associated with identified hazardous substances and any available recommended exposure or tolerance limits for such hazardous substances, and the comparison of existing morbidity and mortality data on diseases that may be associated with the observed levels of exposure. The Administrator of ATSDR shall use appropriate data, risk assessments, risk evaluations, and studies available from the Administrator of EPA."

In accordance with the CERCLA section cited, this Health Assessment has been conducted using available data. Additional Health Assessments may be conducted for this site as more information becomes available.

The conclusions and recommendations presented in this Health Assessment are the result of site specific analyses and are not to be cited or quoted for other evaluations or Health Assessments.

Public Health Assessment

Ringwood Mines/Landfill Site

Borough of Ringwood, Passaic County, New Jersey

USEPA Facility ID: NJD980529739

Prepared by:

New Jersey Department of Health and Senior Services
Public Health Services Branch
Consumer and Environmental Health Services
Hazardous Site Health Evaluation Program

Under a Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

Table of Contents

Page Number

Summary…….…….. 1

Statement of Issues…….…… 3

Background …….……. 3
- Site Description…….……..... 3
- Ramapough Mountain Indians…….…… 4
- Ringwood Mines…….……..... 4
- Waste Disposal at the Site…….…….. 5
- Site Investigation and Remediation…….…… 5
- Environmental Monitoring Program…….……... 6
- Current Site Remedial Activities…….…… 7
- Site Activities by the ATSDR and NJDHSS…….……..... 7

Community Concerns……..... 9

Environmental Contamination…….. 10
- Environmental Guideline Comparison……. 11
- Site Conditions……..... 11
- Pre Remedial Investigation ……... ……. 12
- Remedial Investigation: Site Contamination…….……. 13
  - *Drum Content*……..... 14
  - *Surface Soil* ……..... 14
  - *Paint Sludge*……..... 14
  - *Soil*……..... 15
  - *Sediment*……..... 15
  - *Surface Water from Brooks* ……..... 15
  - *Surface Water from Springs/Seeps*……..... 16
  - *Groundwater*……..... 16
  - *Potable Wells*……..... 16

Remedial Actions Summary……. 16
- Environmental Monitoring Program (Post 1987/1988 Remediation)……..…… 17
  - *Potable Wells*……..... 17
  - *Monitoring Wells*…… 17

Surface water……..... 18
Public Supply Water……... 18
Residential Soil……... 18

Contaminants of Concern Summary …… 19
Pre 1987/1988 Remediation…..…….…….... 19
Post 1987/1988 Remediation…..……..……. 20

Discussions …….. 21
Exposure Pathway Evaluation……. 21
Completed Exposure Pathways…… 22
Incidental Ingestion - Paint Sludge, Soil, Sediment…… 22
Dermal Contact - Paint Sludge, Soil, Sediment……. 22
Ingestion - Surface Water …….. 23
Potential Exposure Pathways…….. 23
Inhalation - Ambient Air……. 23
Ingestion - Biota…….. 23
Ingestion - Groundwater (Off site Potable Wells)…….. 24

Public Health Implications…… 24
Non-Cancer Health Effects…….. 24
Ingestion - Contaminants in Sludge, Soil, and Sediment…….. 25
Ingestion - Surface Water (Springs/Seeps, Brooks)…… 30
Cancer Health Effects……..... 33
Ingestion - Sludge, Soil and Sediment…….……... 33
Ingestion - Surface Water (Springs/Seeps, Brooks)……..……. 35
Assessment of Joint Action of Chemical Mixtures……. 35

Child Health Considerations……. 36

Health Outcome Data…… 36
Childhood Lead Exposure…… 37
Cancer Incidence……. 38
Evaluation of Other Community Health Concerns…….. 39

Conclusions…….. 42

Recommendations……. 44

Public Health Action Plan (PHAP) …….. 45
Actions Undertaken…….. 45

Actions Planned……... 45

Prepares of Report…… 47

References……. 48

Tables
Figures
Photographs

Appendix A: February 24, 2004 RNAA Statement
Appendix B: NJDHSS Public Health Response Plan (PHRP)
Appendix C: September 23, 2004 RNAA Response to PHRP
Appendix D: Toxicologic Characteristics
Appendix E: Assessment of Joint Toxic Action of Chemical Mixtures
Appendix F: Cancer Incidence Analysis
Appendix G: Summary of ATSDR Conclusion Categories
Appendix H: ATSDR Glossary of Terms

Summary

The Ringwood Mines/Landfill site is located in Ringwood Borough, Passaic County, New Jersey. Between 1965 and 1972, wastes (e.g., car parts, paint sludge, solvents) from the Ford Motor Company's Mahwah, Bergen County, New Jersey assembly plant were dumped at the site. Based on an evaluation of hazards associated with site-related contamination, the site was added to the National Priorities List on September 1, 1983. Subsequent to investigation and cleanup under United States Environmental Protection Agency and New Jersey Department of Environmental Protection oversight, the site was deleted from the National Priorities List on November 2, 1994, however, the site has been proposed to be restored to the National Priorities List on April 19, 2006. Further investigations have determined that paint sludge remains widespread at the site and that multiple media (soil, sediment, ground and surface water) have been adversely impacted.

In September 2003, the Native American community residing on the site expressed health concerns allegedly related to widespread paint sludge contamination remaining at the site and requested assistance from the Agency for Toxic Substances and Disease Registry. Through a cooperative agreement with the Agency for Toxic Substances and Disease Registry, the New Jersey Department of Health and Senior Services prepared a public health assessment for the Ringwood Mines/Landfill site. Environmental contamination detected at the site and associated exposure pathways were evaluated. Contaminants of concern identified for the site were benzene, 1,2-dichloropropane, methylene chloride, pentachlorophenol, Aroclors, bis(2-ethylhexyl)phthalate, benzo[a]pyrene, antimony, arsenic, cadmium, chromium, copper, lead, mercury and thallium. It was determined that completed exposure pathways via the ingestion of contaminated surface water and the incidental ingestion of contaminated paint sludge, soil, and sediment existed in the past. Although exposures have been interrupted to a certain extent, contaminated paint sludge deposits and contaminated soil and sediment remain at the site. Potential pathways were also identified and included past inhalation of ambient air and past and current ingestion of biota and groundwater from off-site potable wells.

Past exposures associated with antimony and lead (in paint sludge), arsenic (in surface water), and lead (in soil and surface water) may have resulted in non-cancer adverse health effects in children and adults. Potential health hazard due to additive or interactive effects of chemical mixtures may be greater than estimated by the endpoint-specific hazard index, particularly for neurological effects associated with co-exposure to lead and arsenic. Lifetime excess cancer risks associated with the ingestion of paint sludge, surface soil, and sediment were estimated to be very low when compared to the New Jersey background cancer risk. Based on the maximum and mean arsenic concentrations detected in surface water, lifetime excess cancer risks were estimated to be approximately five and two excess cancer cases per 10,000 individuals, respectively.

Paint sludge is the likely source of most of the lead, as well as the antimony at the site. Paint from pre-1978 housing may also contribute to lead in the environment.

Arsenic, however, may be naturally occurring in the area. Based on health risks posed by exposures to lead and antimony, the site posed a ***Public Health Hazard*** in the past. Since there may be on-going exposure from paint sludge and soil at levels of health concern, the site currently poses a ***Public Health Hazard***.

Childhood blood lead data were evaluated for the Ringwood Mines/Landfill site. Results showed both a higher proportion of children with elevated blood lead levels and a slightly higher average childhood blood lead level in the focus area closest to the Ringwood Mines/Landfill site compared to the rest of Ringwood Borough. Although there are multiple sources of lead in a child's environment (such as peeling lead-based paint in homes), lead-containing paint sludge may have contributed to these differences in blood lead levels.

An analysis of cancer incidence for the period 1979 through 2002 in the Ringwood Mines/Landfill area indicated that overall cancer incidence was not elevated. However, lung cancer incidence was statistically elevated in males in the area closest to the Ringwood Mines/Landfill site. Information on smoking history, the most important risk factor for lung cancer, was not available. Since lung cancer incidence was not elevated in females, there is little evidence that cancer incidence has been affected by site-related contamination.

Other health concerns that residents believe are related to exposures to the Ringwood Mines/Landfill site contamination include respiratory diseases, reproductive and developmental effects, neurological disorders, heart disease, skin rashes and eye irritation, anemia, and diabetes. Many of the community's concerns are consistent with health effects of lead and arsenic exposures reported in the scientific literature; however, these health outcomes may also be caused by other environmental and non-environmental risk factors.

Recommendations for the site include the remediation of paint sludge and associated soil and groundwater contamination, characterization of potential biota contamination, further assessment of background concentrations of arsenic and other site-related contaminants, and an exposure investigation of the community living on the Ringwood Mines/Landfill site. The NJDHSS and ATSDR also recommend concurrent testing of environmental media such as indoor dust and soils close to homes.

The NJDHSS and ATSDR will begin planning for implementation of an Exposure Investigation to determine the extent of exposure to heavy metals from environmental media contaminated by paint sludge. An exposure investigation should include biological testing of adults and children for exposure to lead, antimony, and arsenic. Plans for an exposure investigation should be developed in conjunction with community members. The NJDHSS and ATSDR will also work with the USEPA and NJDEP to coordinate potential environmental testing that would be conducted in association with biological monitoring.

Statement of Issues

In September 2003, the Agency for Toxic Substances and Disease Registry (ATSDR) received a letter from attorney Stephen Sheller of Sheller, Ludwig and Badey, P.C., on behalf of a Native American community residing on the Ringwood Mines/Landfill site, Ringwood Borough, Passaic County, New Jersey. Mr. Sheller requested that the ATSDR provide assistance in determining whether past and current exposures to hazardous substances disposed at the site presented a public health hazard. The ATSDR considered Mr. Sheller's letter a petition and approved the request.

The Ringwood Mines/Landfill site contains abandoned magnetite mines which were in operation from the mid 1700s through the early 1900s. Between 1965 and 1972, wastes (e.g., car parts, paint sludge, solvents) from the Ford Motor Company's Mahwah Bergen County, New Jersey assembly plant were dumped at the site on the ground, in open pits, and in mine shafts. Based on an evaluation of hazards associated with site-related contamination, the site was added to the National Priorities List (NPL) on September 1, 1983. Subsequent to investigation and cleanup under U.S. Environmental Protection Agency (USEPA) and New Jersey Department of Environmental Protection (NJDEP) oversight, the site was deleted from the NPL on November 2, 1994. Further investigations have determined that paint sludge remains widespread at the site and that multiple media (soil, sediment, ground and surface water) have been adversely impacted.

Through a cooperative agreement with the ATSDR, the New Jersey Department of Health and Senior Services (NJDHSS) prepared the following public health assessment for the Ringwood Mines/Landfill site. The goal of this public health assessment was to examine environmental contamination at the Ringwood Mines/Landfill site, evaluate available health outcome data, and address community health concerns. The report provides conclusions, recommendations, and an action plan designed to protect public health.

Background

Site Description

The Ringwood Mines/Landfill site is located in the Borough of Ringwood, Passaic County, New Jersey (see Figure 1). The site is approximately 500 acres and approximately one half mile wide by one and one half miles long. The site is characterized by a variety of features including abandoned mine shafts and surface pits, an inactive landfill, an industrial refuse disposal area, small surface dumps, a municipal recycling area, a municipal garage, and 48 residences. The terrain is forested, with open areas.

Ramapough Mountain Indians

There are approximately 900 people living within one mile of the site (see Figure 2). About 200 of these individuals are Ramapough Mountain Indians living at 48 residences on the site. The Ramapough Mountain Indians are descendants of the Lenape Indians. Many tribal members live around the Ramapo Mountains (a range of the Appalachian Mountains) of northern New Jersey and southern New York. Although not currently a federally recognized Native American tribe, the Ramapough Mountain Indians have been recognized by the state of New Jersey.

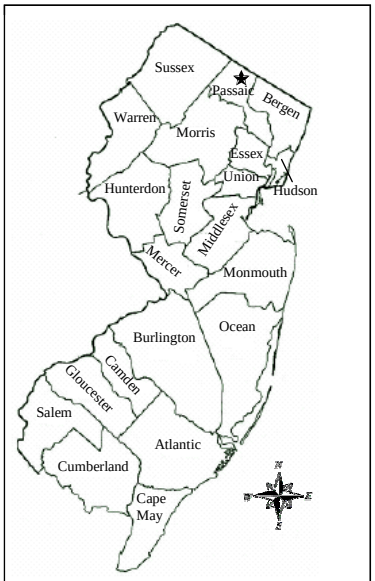


Figure 1: Location of Ringwood Mines/Landfill Site

Health and economic challenges in this tribal community were documented over 30 years ago. A news article which referred to a William Paterson College health survey of Ringwood Mines area residents conducted in the early 1970s described "the cycle of poverty, poor nutrition, inadequate education, and lower standard of health" among the individuals interviewed by nursing students (Kupferstein 1973)[1]; another article reported on the "very poor" health conditions and substandard housing existing in the Ringwood Mines area (West Milford Argus Today 1975).

Ringwood Mines

The Ringwood Mines comprise a group of open pits and shafts that were mined from the mid 1700s to the early 1900s. Some of the ore deposits were well known before the American Revolution, and the older openings were reportedly made before 1760 (Pustay and Shea 1992). The principal product of the mines during the years of operation was magnetite ore, which was processed on-site and shipped to local iron foundries. Five of the pits and shafts which comprise the Ringwood Mines are the Peters, St. George, Miller, Keeler, and Cannon mines. To illustrate, the Cannon Mine was a large, open pit, measuring approximately 140 by 180 feet and 200 feet deep. In 1880, the total yield of the entire Ringwood Mines was estimated at 896,000 tons of ore. Active mining activities ceased around 1931.

In 1941, the Ringwood Mines property was purchased by the federal government and subsequently leased to the Alan Wood Steel Company. The intent was to restart mining activities to support the World War II effort. Wartime production needs did not develop, and the mines remained inactive. In July 1958, the property was sold at a government auction to the Pittsburgh Pacific Company, a Minnesota-based mining company. It is believed that Pittsburgh Pacific Company did not engage in active mining activity at the Ringwood Mines. Use of the site between 1956 and 1965 is not well

[1]Attempts by the NJDHSS to obtain the report were unsuccessful.

documented. In April 1965, the New Jersey Bureau of Mine Safety inspected the Ringwood Mines. Refuse, including municipal wastes, was present in open pits and mine shafts (YE[2]ARS, Inc 1983). In an annual report of the Ringwood Borough Planning Board, it was stated that 31 of the total 33 mine shafts were sealed[2] under the supervision of the New Jersey State Department of Mines following the removal of over 500 abandoned vehicles (Ringwood Borough Planning Board 1965).

Waste Disposal at the Site

In 1965, the Ringwood Realty Corporation, a wholly-owned subsidiary of the Ford Motor Company, bought the property and subsequently began dumping wastes (e.g., car parts, paint sludge, solvents) from Ford's Mahwah, Bergen County, New Jersey assembly plant. Some wastes were deposited on the ground in natural depressions and in man-made pits associated with abandoned mine shafts or other mining activities. There are conflicting reports about the time frame for the disposal of wastes at the Ringwood Mines/Landfill site[3] (YE[2]ARS 1983; Muszynski 1993; USEPA 2004; Latham-Watkins 2005).

Of the approximately 900 acres purchased by Ringwood Realty Corporation from the Pittsburgh Pacific Company in 1965, only 150 acres in the vicinity of Peters Mine (a.k.a. O'Connor Refuse Disposal Area) was permitted for dumping (YE[2]ARS 1983). In 1965, Ringwood Realty Corporation began selling portions of the property to Jersey Central Power & Light and High Point Homes. In 1970, Ringwood Realty Corporation donated 290 acres to the Ringwood Solid Waste Management Authority (RSWMA). The RWSMA operated a municipal landfill on a portion of this property from 1972 until it was ordered closed by the NJDEP in 1976. In 1973, Ringwood Realty Corporation donated 150 acres, including the O'Connor Refuse Disposal area, to the NJDEP and the affordable housing authority, Housing Operation With Training Opportunity, Incorporated (HOWTO Inc.).

Site Investigation and Remediation

In 1976, the NJDEP sampled surface water from the vicinity of the site and detected contaminated leachate emanating from the landfill. The landfill was subsequently closed. Between November 1979 and April 1980, the NJDEP and the USEPA conducted preliminary assessments of the site, and in 1982, groundwater sampling of the Peters Mine shaft was conducted. Results indicated contamination with benzene, ethylbenzene, xylene, chloroethane, and bis(2-ethylhexyl)phthalate; samples obtained from the Peters Mine Brook showed heavy metal contamination (nickel,

[2] "Seal" does not necessarily imply permanent closure, as with, say, a concrete cap poured over installed supports. A steel fence (to keep away children, etc.) or rocks and trees could be claimed as a "seal". Liquids or other materials may be poured or pushed through many of these "seals." (H. Black, New Jersey Department of Labor; R. Dalton, NJDEP, personal communications, March 2006.)

[3] 1963 - 1974 (YE[2]ARS 1983); 1967 - mid 1970s (Muszynski 1993); 1967 - 1974 (USEPA 2004); 1967 - 1971 (Latham-Watkins 2005)

cadmium, tin, chromium), some of which may have been naturally occurring. The site was added to the NPL on September 1, 1983.

On July 1, 1987, the USEPA issued a Unilateral Order to the Ford Motor Company which required that paint sludge with high heavy metal content be excavated and disposed of at a hazardous waste landfill. Subsequently, a September 29, 1988 Record of Decision (ROD) was issued for the site. Since the selected site remedy resulted in hazardous substances remaining on-site above the health-based levels, a review was required to be conducted within five years after the commencement of remedial action and every five years thereafter to ensure that the remedy continued to provide adequate protection of human health and the environment. Also required was an operation and maintenance program consisting of the sampling of selected on- and off-site groundwater monitoring wells semi-annually for the first five years, then for another 25 years if deemed necessary.

Approximately 7,000 cubic yards of paint sludge were removed from the site in 1987 and 1988, and remediated areas were backfilled with clean soil. Groundwater, surface water, soil, and sediment samples were collected. Post-remedial sampling (Woodward-Clyde 1988) indicated continued elevation of certain contaminants, including lead, in some soils. Following the removal of the paint sludge, risks to human health and the environment associated with the site were evaluated by an environmental consulting firm retained by the Ford Motor Company (Environ 1988; Woodward-Clyde 1990). The evaluations concluded that the presence of metals[4] in environmental media at the site presented the most significant public health and environmental risk, albeit these metals occur naturally and could be the result of past mining activities or natural weathering processes. The reports further stated that there was no evidence that metal concentrations detected in site soil, sediment, and surface water were significantly higher than those measured elsewhere in the New Jersey Highlands.

In 1990 and 1991, an additional 600 cubic yards of paint sludge as well as about 54 drums containing various wastes were removed from the site. No paint sludge samples were collected; however, drum contents were analyzed. In 1995, a resident contacted the USEPA regarding the discovery of paint sludge on his property, and five cubic yards were removed (Geraghty & Miller 1996). In 1997 and 1998, additional paint sludge was identified during a USEPA site visit. One hundred cubic yards of paint sludge were removed and post-excavation soil samples were collected.

Environmental Monitoring Program

At the direction of the USEPA, Ford initiated a five-year Environmental Monitoring Program (EMP) in the fall of 1989, which continued through 1995. Ford sampled area potable and groundwater monitoring wells to determine contaminant concentrations in the upper aquifer. Surface water sampling was discontinued in 1990,

[4]Arsenic, barium, cadmium, chromium, copper, lead, manganese, mercury, nickel, selenium, thallium, and zinc.

when sampling and analysis showed no contamination above surface water quality criteria.

In 1998, the USEPA directed Ford to conduct two additional rounds of groundwater sampling in select wells because the data showed elevated levels of lead and arsenic in four on-site monitoring wells. Beginning in August 1999, Ford conducted several sampling rounds of surface water and groundwater monitoring wells; results indicated that except for one elevated level of arsenic, lead and arsenic levels had decreased and were below health-based standards. In June 1998, the USEPA collected surface water samples in response to citizen concerns regarding discolored surface water. The discoloration was later determined to be associated with iron bacterial growth.

Throughout the remedial investigation and EMP activities, the NJDEP reviewed and commented on reports submitted to the USEPA by Ford's environmental consultant. In a 1998 review of the EMP, the NJDEP stated that it remained unclear as to whether exceedances of lead and other metals detected in monitoring wells were due to natural conditions versus former paint sludge disposal areas. Based on this uncertainty, the NJDEP rejected a No Further Action request as the contaminant source had not been adequately demonstrated by Ford (NJDEP 1998). Subsequent to reported fish deaths at the site, the NJDEP contacted appropriate wildlife officials to conduct oversight in determining further evidence of fish deaths for at least six months (Zalaskus 2000). In 2002, the USEPA, in concurrence with the NJDEP, determined that the EMP was complete at the site.

Current Site Remedial Activities

In 2004, and with the oversight from the USEPA, NJDEP, and U.S. Army Corps of Engineers, the Ford Motor Company initiated a comprehensive program to address concerns about the adequacy of past remedial activities implemented at the Ringwood Mines/Landfill site. Between January and December 2005, 13,156 tons of paint sludge and associated soil were excavated and removed from the site. Post-remedial soil samples in the vicinity of the excavations showed remaining areas of lead contamination (ARCADIS 2006). Additional areas of paint sludge have been identified and will be remediated. Drum remnants identified in the Peters Mine Area will also be addressed.

In October 2005, the NJDEP negotiated access agreements for three on-site residential properties. Paint sludge from three properties was investigated and remediated (NJDEP 2005). Edison Wetlands Association also collected sludge and post-excavation soil samples from these areas (Chapin Engineering 2005).

Site Activities by the ATSDR and NJDHSS

In 1989, the ATSDR prepared a public health assessment for the Ringwood Mines/Landfill site. The report concluded that the site posed a *"potential public health concern"* due to risks of exposures through the incidental ingestion of soil.

Recommendations included the limiting of access to contaminated areas and the performance of a detailed well inventory.

In 1994, in preparation for the site being deleted from the NPL, a Site Review and Update (SRU) for the Ringwood Mines/Landfill site was prepared (ATSDR 1994a). The purpose of this report was to perform a review of current site conditions and recommend further actions for ATSDR to take at the site. The 1994 report concluded that there were no completed human exposure pathways associated with the site. The report stated, however, that if new information became available indicating that exposures to hazardous materials may be occurring, additional actions would be taken.

Following ATSDR approval of Mr. Sheller's petition, representatives of the NJDHSS, ATSDR, and Ringwood Borough Health Department conducted a site visit of the Ringwood Mines/Landfill site on October 28, 2003. NJDHSS representatives were Christa Fontecchio, Somia Aluwalia, Tariq Ahmed, Steven Miller, and Julie Petix; Leah Escobar represented the ATSDR. Litter and trash were observed on the site (see Photograph 1), and a guardrail had been installed near the site entrance reportedly to keep out illegal dumpers. Two local residents present at the time of the site visit reported past and/or current use of the site for recreational activities (e.g., fishing, hunting, dirt riding, mountain biking, swimming, and ice-skating on an on-site pond known as "the pool") (see Photographs 2 and 3). They also reported that in the 1960s through the early 1980s, on-site residences did not have indoor plumbing or electricity. There were no potable wells on the site. Using buckets, water for all domestic household use was obtained from an on-site spring.

On April 14, 2004, a second site visit was conducted. Participants included representatives of the NJDHSS, ATSDR, NJDEP, USEPA, Ringwood Borough (Mayor, Deputy Mayor, Health Officer), an aide to Senator Frank Lautenberg, local media, community members (including attorneys representing the community), and the Passaic County Department of Health. The site visit began with a meeting at the Church of the Good Shepherd located in Ringwood Borough near the site. An environmental consultant for the community's law firm described concerns about the adequacy of the USEPA-supervised cleanup of the site prior to and after deletion from the NPL. Film footage believed to be taken by a local resident sometime in the 1960s was shown. In the film, children could be seen playing in an area where waste materials were being dumped and moved around with a backhoe. The film also showed fires in the mine shafts. According to local residents, the fires would burn for weeks and emit black smoke that would sicken residents. The USEPA spoke briefly about recent environmental sampling events at the site.

After the meeting, a three-hour tour of the site was conducted. Hardened paint sludge was observed in several areas throughout the site (see Photograph 4). A 55-gallon metal drum and a drum lid were also spotted in one area of the site (see Photographs 5 and 6). The site visit ended with a visit to "Sludge Hill" where the USEPA had conducted a major removal action in 1998 (see Photographs 7 and 8). Along with an occasional 55-gallon drum, some small pieces of sludge were seen on the slope of the

hill. Large piles of garbage and old tires were observed near residences and particularly in the vicinity of the hill. Ammunition casings were also observed at the top of the hill.

On February 24, 2004, the NJDHSS and ATSDR sponsored two availability sessions (afternoon and evening) at the Ringwood Borough municipal building. The purpose of the sessions was to provide an opportunity for residents to meet one-on-one with NJDHSS and ATSDR staff to discuss personal health concerns suspected to be associated with site-related contamination. In concurrence with the decision of the Ringwood Neighborhood Action Association (RNAA) President, Mr. Wayne Mann, about 60 community members chose to attend only the evening session. Mr. Mann read a prepared statement expressing concerns pertaining to the presence of paint sludge at the site (see Appendix A).

On September 23, 2004, NJDHSS and ATSDR staff attended a meeting with the Ringwood Mines area residents to discuss a draft Public Health Response Plan (PHRP) proposed by the NJDHSS (see Appendix B). The meeting was attended by approximately 20 residents and began with a statement read by RNAA President Mr. Mann (see Appendix C). Essentially, Mr. Mann stated that the draft PHRP did not adequately address the full range of community health concerns expressed by residents.

On June 15, 2005, the NJDHSS and ATSDR arranged a public meeting to discuss the progress on the public health assessment being prepared for the Ringwood Mines/Landfill site. During the meeting, past and current exposure pathways were discussed and methods and preliminary results of health outcome data analysis (cancer incidence, childhood blood lead) were presented. Feedback from meeting participants, particularly as related to historic exposure pathways, was solicited and encouraged.

On October 27, 29, and November 5, 2005, the NJDHSS sponsored free medical screenings for Ringwood Mines area residents. Medical professionals affiliated with the North Hudson Community Action Corporation mobile facility provided age-appropriate health screening evaluations to both children and adults. Although not part of the public health assessment for the Ringwood Mines/Landfill site, the screenings were conducted to ensure that basic health care services were available to the community.

Community Concerns

Community exposure and health concerns have been expressed through written communications from legal counsel, prepared statements by the RNAA, and by residents during site visits and community meetings. The inadequacy of past cleanups at the site, and resultant exposure to toxic chemicals, is of foremost importance. In order to emphasize the extent of the problem, one individual brought chunks of hardened paint sludge to the February 2004 meeting which had been collected near a residence. He described a volatile organic smell and skin irritation of his hands from picking up the paint sludge. Residents expressed concern that the extent of past dumping was not fully appreciated. One resident reported observing an average of 12 trucks with a carrying

capacity of 20 cubic yards dumping Ford waste on the site five days per week, and approximately three to four truckloads of waste were dumped on the site on weekends.

Community members expressed concerns that the PHRP drafted by the NJDHSS in response to the petition would not fully address the range of community concerns about the site. The scope of planned health outcome data reviews (using existing surveillance data assembled by the NJDHSS) was viewed as inadequate in showing the overall health impact experienced by the community. In early 2005, the RNAA proposed an Environmental Health Intervention Program (EHIP) which would include full participation of the community, in conjunction with the NJDHSS and ATSDR, in the investigation of the extent and causes of health problems experienced by community members (RNAA 2005). The EHIP also included components to document the history and culture of the Ramapough Mountain Indians.

On November 15, 2004, the RNAA petitioned the New Jersey Environmental Justice Task Force to obtain Environmental Justice designation for the Ringwood Mines/Landfill site. A description of resident health concerns allegedly related to the dumping of wastes by the Ford Motor Company at the site was provided in the petition letter.

Health concerns that community members feel are related to exposures associated with the Ringwood Mines/Landfill site contamination include: cancer (ovarian, cervical, leukemia, breast, lung, Ewing sarcoma, colon), respiratory disease (asthma, emphysema), reproductive and developmental effects (female reproductive disorders, miscarriages, birth defects, learning disabilities, behavioral problems), neurological disorders, heart disease, skin rashes, eye irritation, anemia, diabetes, and shorter lifespan.

A series of articles in the Bergen Record extensively documented the history of the site and the scope of the community's concerns about environmental exposure and health (Bergen Record 2005).

Environmental Contamination

An evaluation of site-related environmental contamination consists of a two-tiered approach: 1) a screening analysis; and 2) a more in-depth analysis to determine public health implications of site-specific exposures. First, maximum concentrations of detected substances are compared to media-specific environmental guideline comparison values (CVs). If concentrations exceed the environmental guideline CV, these substances, referred to as Contaminants of Concern (COC), are selected for further evaluation. Contaminant levels above environmental guideline CVs do not mean that adverse health effects are likely, but that a health guideline comparison is necessary to evaluate site-specific exposures. Once exposure doses are estimated, they are compared with health guideline CVs to determine the likelihood of adverse health effects.

Environmental Guideline Comparison

There are a number of CVs available for the screening environmental contaminants to identify COCs. These include ATSDR Environmental Media Evaluation Guides (EMEGs) and Reference Media Evaluation Guides (RMEGs). EMEGs are estimated contaminant concentrations that are not expected to result in adverse noncarcinogenic health effects. RMEGs represent the concentration in water or soil at which daily human exposure is unlikely to result in adverse noncarcinogenic effects. If the substance is a known or a probable carcinogen, ATSDR's Cancer Risk Evaluation Guides (CREGs) were also considered as comparison values. CREGs are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million (10^{-6}) persons exposed during their lifetime (70 years). In the absence of an ATSDR CV, other comparison values may be used to evaluate contaminant levels in environmental media. These include New Jersey Maximum Contaminant Levels (NJMCLs) for drinking water, and USEPA Region 3 Risk-Based Concentrations (RBCs). RBCs are contaminant concentrations corresponding to a fixed level of risk (i.e., a hazard quotient[5] of 1, or lifetime excess cancer risk of one in one million, whichever results in a lower contaminant concentration) in water, air, biota, and soil. For soils and sediments, other CVs include the New Jersey Residential and Non-Residential Direct Contact Soil Cleanup Criteria (RDSCC, NRDSCC). Based primarily on human health impacts, these criteria may also take into account natural background concentrations, analytical detection limits, and ecological effects.

Substances exceeding applicable environmental guideline CVs were identified as COCs and evaluated further to determine whether these contaminants pose a health threat to exposed or potentially exposed receptor populations. In instances where an environmental guideline CV was unavailable, the substance was retained for further evaluation. There are exceptions, however. For example, some naturally occurring substances such as sodium, calcium, potassium, and magnesium are typically not harmful under most environmental exposure scenarios and may not necessarily be retained for further analysis.

Site Conditions

The Ringwood Mines/Landfill site is located at the southeastern extension of the New Jersey Highlands Physiographic Province (Woodward-Clyde 1988). The terrain is mountainous with peaks up to 900 feet above sea level. Bedrock at the site consists primarily of Precambrian gneiss. The topographic low areas throughout the site consist of overburden material including weathered bedrock, excavated rock, mine tailings, refuse, and fill soil. Three perennial surface water bodies drain the site: Mine Brook, Peters Mine Brook, and Park Brook (see Figure 3). Surface water flowing from the site ultimately discharges to the Wanaque Reservoir located approximately one mile south of the site. Park Brook flows into Ringwood Creek approximately one mile upstream of its confluence with the Wanaque Reservoir. Along the southern site boundary, Peters Mine

[5]The ratio of estimated site-specific exposure to a single chemical in a particular medium from a site over a specified period to the estimated daily exposure level, at which no adverse health effects are likely to occur.

Brook joins Mine Brook to flow into Ringwood Creek upstream of the Wanaque Reservoir. The intake for the North Jersey District Water Supply Commission water treatment plant, which supplies drinking water to more than two million people, is located approximately eight miles downstream of the Ringwood Mines/Landfill site at the southern end of the Wanaque Reservoir (NJDWSC 2005).

Regional groundwater flow has not been evaluated, although it is known that groundwater flow through a fracture network is strongly influenced by the orientation and geometry of bedrock fractures. At the site, there is an upper aquifer (consisting of overburden and shallow bedrock) and a lower aquifer (deep bedrock). Groundwater in the upper aquifer ranges from a few feet to approximately 60 feet below ground surface. Flow generally follows the topography, recharging surface water bodies that discharge into the Wanaque Reservoir (see Figure 4). The direction of groundwater flow in the lower aquifer is uncertain, although it is believed to consist of three components: shallow flow to local streams; intermediate flow to regional streams; and deep flow towards the ocean. The upper and lower aquifers interconnect throughout the area, but the flow between the aquifers is limited by poor vertical permeability (Woodward-Clyde 1988).

Pre Remedial Investigation

A Remedial Action Master Plan for the Ringwood Mines/Landfill site was prepared based on information obtained from the USEPA, NJDEP, New Jersey Geological Survey, New Jersey Bureau of Mine Safety, and the Ringwood Borough Planning Board (YE^2ARS 1983).

Results of analyses for volatile organic compounds (VOCs), metals, and cyanide in Mine Brook surface water and municipal landfill leachate obtained between July 1974 and April 1975 indicated concentrations of cadmium, copper, iron, and manganese above drinking water standards. Although the maximum concentration of lead detected (24 ppb) was below the 50 ppb standard applicable at that time, it is above the current action level of 15 ppb. Groundwater samples collected (three rounds of sampling conducted; some parameters were not analyzed) from the Peters Mine shaft were analyzed for a number of contaminants including VOCs, metals, and pesticides (YE^2ARS 1983). Pesticides and polychlorinated biphenyls (PCBs) were not detected; concentrations of VOCs and metals are provided in the table below. The concentrations of benzene, bis(2-ethylhexyl)phthalate, chloroethane, methylene chloride, iron, lead, and beryllium were above the corresponding environmental guideline CV. A high concentration of iron (32,000 micrograms per liter (μg/L)) exceeded the secondary NJMCL of 300 μg/L, which is based on aesthetic (color or taste) rather than health effects.

VOCs and Metals Results Peters Mine Shaft Groundwater, October 1980		
Contaminant	**Concentration (µg/L)**	**Environmental Guideline CV (µg/L)**
Volatile Organic Compounds		
Benzene	**19**	**1 (NJMCL)**
Bis(2-ethylhexyl)phthalate	**304**	**4.8 (RBC)**
Chloroethane	**150**	**3.6 (RBC)**
1,1-Dichloroethane	10.2	50 (NJMCL)
Ethylbenzene	95	700 (NJMCL)
Methylene Chloride	**4**	**3 (NJMCL)**
Xylenes	150	1,000 (NJMCL)
Metals		
Beryllium	**7.8**	**4 (NJMCL)**
Chromium (IV)	7[1]	100 (NJMCL)
Copper	15	1,300 (AL[2])
Lead	**70[1]**	**15 (AL)**
Zinc	61	3,000 (RMEG)

[1]approximate value; [2]Action Level

Bold font indicates environmental guideline CV was exceeded

In 1982, samples collected from the Ringwood Water Department water supply wells (i.e., the Mine Supply spring and the Windbeam municipal supply well) were analyzed for standard drinking water parameters (YE[2]ARS 1983). No contaminants were reported in the Mine Supply sample. Contaminants detected in the Windbeam municipal supply well and the corresponding NJMCLs are provided in the following table; concentrations of all parameters were below the NJMCLs.

Results of Ringwood Borough Water Department Water Supply Sampling, June 1982		
Contaminant	**Windbeam Municipal Supply Well (µg/L)**	**NJMCL (µg/L)**
Chromium	1	100
Fluoride	50	4,000
Lead	10	15 (AL[1])
Nitrate - N	750	10,000

[1]Action Level

Remedial Investigation: Site Contamination

Subsequent to the site being added to the NPL, a Remedial Investigation (RI) was conducted to determine the nature and extent of site contamination (Woodward-Clyde 1988). Test pit locations were reportedly selected based on site reconnaissance, literature

review, terrain conductivity, and resistivity surveys. During test pit excavation, waste materials (e.g., garbage, construction material and debris, rubber hoses) were encountered. Samples of paint sludge, soil (fill and indigenous), contents of 55-gallon drums, and surface and groundwater were collected and analyzed for metals, VOCs, semi-volatile organic chemicals (SVOCs including PCBs), total petroleum hydrocarbons (TPH), and cyanide.

Drum Content

Drums disposed of at the site contained waste oil, sludge, brake fluid, antifreeze, "Speedy Dry", gloves, rags, and cloths. Laboratory analysis of drum content was conducted in June and September of 1990; results indicated the presence of VOCs, PCBs (Aroclor[6] 1254 and 1262), and metals (see Table 1) (A. Robinson, ARCADIS, personal communication, 2005).

Surface Soil[7]

As presented in Figure 3, four primary areas of surficial paint sludge contamination were identified:

- Peters Mine Area, a.k.a. O'Connor Disposal Area;
- St. George Pit/Miller-Keeler Pit Area;
- Cannon Mine Area; and
- Borough Landfill Area.

Test pits were dug in each area and soil samples were obtained. Surface soil from test pit 3 (TP-3) indicated the presence of VOCs, with benzene above its environmental guideline CV (Table 2). Low levels of barium were reported for test pits 3 and 12; lead was also detected in test pit 3.

Paint Sludge

Paint sludge from each of the four primary paint sludge areas was sampled in March and April 1987 and analyzed to determine waste disposal classification. The sludge was classified as "EP toxic[8] for lead", excavated, and disposed off-site, and the areas were backfilled with fill soil. Ten surficial paint sludge samples collected from the four primary paint sludge disposal areas were analyzed for VOCs, SVOCs, pesticides, PCBs, and metals (A. Robinson, ARCADIS, personal communication, 2004). The range and mean of contaminant concentrations detected are provided in Table 3. Levels of PCBs, (Aroclor 1248 and 1254), other SVOCs (bis(2-ethylhexyl)phthalate), and metals (antimony, arsenic, cadmium, chromium, copper, lead) were present above their

[6]Commercial mixtures of PCBs.
[7]Specific soil depths unavailable.
[8]A test defined by the USEPA to check a substance for the presence of arsenic, barium, cadmium, chromium, lead, mercury, selenium, or silver for hazardous waste classification.

corresponding environmental guideline CV. Antimony and lead comprised nearly 5% and over 6% of the sludge material, respectively.

It should be noted that from the time that paint sludge was disposed of at the site until the time of sampling, the paint sludge had been subjected to various degrees of physical, chemical, and biological degradation over a period spanning 20 years. As such, contaminant concentrations reported in Table 3, particularly for VOCs, SVOCs, and PCBs, may not represent conditions close to the time of sludge disposal.

Soil

Pre-remediation surface soil sampling conducted at the site was limited to test pits TP-3 and TP-12 (see Table 2). However, apparent natural soil in proximity to the excavated paint sludge was collected from each of the four primary paint sludge areas and analyzed for VOCs, SVOCs, PCBs, metals, and cyanide. The range and mean of contaminant concentrations detected are provided in Table 4. Maximum concentrations of benzo[a]pyrene, arsenic, lead, and thallium exceeded their corresponding environmental guideline CVs.

Sediment

Sixteen sediment samples were collected (July 1984 and March 1988) from the Mine, Peters Mine, and Park Brooks during two sampling rounds. Samples were analyzed for VOCs, SVOCs, pesticides, PCBs, and metals. Concentrations of benzo[a]pyrene, arsenic, iron, manganese, and thallium exceeded their respective environmental guideline CVs (see Table 5). Arsenic was detected in 14 of 16 samples; the maximum concentration was 31.4 mg/kg. Polycyclic aromatic hydrocarbons (PAHs), including benzo[a]pyrene, were detected in less than half of the samples at concentrations less than 1.0 mg/kg. Pesticides and PCBs were not detected in the samples analyzed. The presence of iron and manganese were attributable to natural sources and eliminated from further consideration. Although arsenic is known to occur naturally in the Ringwood Mines/Landfill area (NJGS 2005), the source of arsenic detected in the sediment could not be determined.

Surface Water from Brooks

Between July 1984 and March 1988, surface water samples were collected from the Mine, Peters Mine, and Park Brooks. Samples were analyzed for metals, VOCs, SVOCs, PCBs, pesticides, and other drinking water parameters. Arsenic was detected in one of 20 samples at a concentration of 40 micrograms of arsenic per liter of water (μg/L), above its environmental guideline CV (see Table 6). It should be noted that arsenic occurs naturally in the groundwater in that area (NJGS 2005). PCBs were not detected in any samples.

Surface Water from Springs/Seeps

Twenty samples from seeps/springs, collected during two sampling rounds (July 1984 and March 1988) were analyzed for VOCs, SVOCs, PCBs, and metals. Concentrations of benzene, 1,2-dichloropropane, arsenic, lead, and mercury exceeded their respective environmental guideline CV (Table 7). Benzene was detected in two of 10 samples at a maximum concentration of 2 μg/L; 1,2- dichloropropane was detected in one sample at a concentration of 12 μg/L. The maximum arsenic concentration was 21 μg/L. Lead was detected in two samples with a maximum concentration of 120 μg/L. Mercury was detected in six samples with a maximum concentration of 8.7 μg/L.

Maximum concentrations of iron and manganese detected in springs/seeps water were above their respective environmental guideline CV. Since iron and manganese are considered to be naturally occurring metals, they were not retained for further evaluation.

Groundwater

Between July 1984 and March 1988, 45 groundwater samples were collected (August and September 1984, June 1986 and March 1988) from 15 on-site monitoring wells during three sampling rounds; one of these samples was from a Peters Mine air shaft. Monitoring well depths ranged from 14 - 543 feet below ground surface. Samples were analyzed for metals, VOCs, SVOCs, and other drinking water parameters; results are presented in Table 8. The concentrations of benzene, methylene chloride, bis(2-ethylhexyl)phthalate, pentachlorophenol, arsenic, cadmium, lead, and thallium exceeded their respective environmental guideline CV. Though infrequently detected, the maximum concentration of cadmium was 93,000 μg/L.

Maximum concentrations of iron and manganese detected in groundwater were above their respective environmental guideline CV. As mentioned earlier, since iron and manganese are considered to be naturally occurring metals, they were not retained for further evaluation.

Potable Wells

There were no known on-site private potable wells.

Remedial Action Summary

As discussed above, remedial actions for the site consisted of the removal of paint sludge and soil contaminated by paint sludge, institutional controls (e.g., controls on the drilling of groundwater wells and/or deed restrictions) and implementation of an EMP. In 1987 and 1988, Ford removed approximately 7,000 cubic yards of paint sludge and associated soil from four areas of the site. The EMP was designed to monitor long-term on- and off-site groundwater and surface water quality to ensure the future protection of public health and the environment. After the implementation of the removal action, soil erosion and earthwork activities uncovered remnants of paint sludge at the site. In 1990,

54 waste containing drums were discovered and were disposed off-site. In 1995, five cubic yards of surficial paint sludge and soil were removed from the site. In December 1997 and January 1998, an additional 30 cubic yards of paint sludge was discovered and disposed off-site. Workplans have been developed for further site investigation and removal of additional paint sludge from the site (ARCADIS 2004; USEPA 2004) including residential properties (J. Seebode, NJDEP, personal communication, 2005).

Environmental Monitoring Program (Post 1987/1988 Remediation)

Groundwater from on-site monitoring wells and off-site potable wells were sampled during the EMP (1989 -1995). Nine off-site potable wells, eight on-site monitoring wells, and tributaries to the Wanaque Reservoir were sampled (see Figure 5). Surface water sampling was discontinued in 1990 when analytical results showed no contamination above the NJDEP surface water quality criteria (Geraghty & Miller 1998).

Potable Wells

Off-site potable wells are located on Margaret King Avenue (see Figure 5); the closest one is approximately 3,000 feet southwest from the intersection of Peters Mine Road and Margaret King Avenue. These wells supply residences and commercial/light industrial facilities with potable water (ARCADIS 1999). Samples from potable wells were collected during the EMP and analyzed for metals, VOCs, and cyanide. The maximum concentration of tetrachloroethene, antimony, beryllium, iron, lead, manganese, and silver detected in the potable wells exceeded their respective environmental guideline CV (see Table 9). The presence of iron and manganese may be attributable to natural sources and were eliminated from further consideration.

Monitoring Wells

Groundwater samples were collected from the on-site monitoring wells located in the northern part of the site and analyzed for VOCs, metals, and cyanide (see Figure 5). The maximum concentration of benzene, chloroethane, 1,1,2,2-tetrachloroethane, aluminum, antimony, arsenic, beryllium, cadmium, chromium, cobalt, iron, lead, manganese, mercury, nickel, thallium, and vanadium detected in the groundwater exceeded their respective environmental guideline CV (see Table 10). The presence of iron and manganese may be attributable to natural sources and were eliminated from further consideration.

In 1998, two additional sampling rounds were performed in select monitoring wells; an elevated arsenic level was detected in one of the wells (ARCADIS 2001). Subsequent sampling showed that lead and arsenic levels had decreased and were below health-based standards, except for one elevated level of arsenic.

Surface water

In March 1998, two surface water samples were collected by the USEPA in response to community concerns regarding areas of standing, discolored water. The samples were collected (from a ponded seep area located north of the end of Peters Mine Road and beneath a culvert along Peters Mine Road south of the municipal recycling area) and analyzed for metals and VOCs (USEPA 1998). Results indicated the presence of VOCs (chloroethane, 1,1-dichloroethane, naphthalene, acetone, and N-nitrosodiphenylamine) and metals (iron, manganese, and zinc). The concentration of all VOCs and zinc were below their respective NJMCLs. Iron and manganese were present above their secondary NJMCLs.

In April 2000, USEPA requested the sampling of Park Brook which runs adjacent to the O'Connor Disposal Area (ARCADIS 2001). Three surface water samples were collected, one upstream, one downstream, and one adjacent to the O'Connor Disposal Area. The samples were analyzed for metals, VOCs, and SVOCs. Concentrations of metals detected in the adjacent and downstream samples did not exceed NJDEP surface water quality criteria.

In response to the discovery of paint sludge by residents, the USEPA collected two surface water samples (from the entrance to an abandoned mining structure and from runoff along the west side of Cannon Mine Road) and one soil sample (from material located in monitoring well OB-8) in May 2004 (USEPA 2004; J. Gowers, USEPA Region II, personal communication, 2006). The aqueous sample results were compared to the New Jersey Groundwater Quality Standards and the National Primary Drinking Water Regulations; iron and manganese detected in one of the samples exceeded NJMCLs. Contaminants concentrations detected in the soil sample were below RDCSCCs.

Public Supply Water

Sampling data available for public supply springs (including cistern number 10) which supplied the upper Ringwood area were reviewed (Edward Haack, Borough of Ringwood, personal communication, 2003). The data included 11 sampling events between May 1988 and December 1997. The cistern number 10 was in use until 2000. Concentration of lead detected in cistern water was 2 μg/L; lead levels at other supplies were non-detect. Three VOCs (bromodichloromethane, chloroform, dibromochloromethane) were also detected. These VOCs are disinfection byproducts associated with water chlorination and are unrelated to the site.

Residential Soil

In November 2005, the NJDEP collected a limited number of surface soil samples from an unpaved driveway, front lawn, and side and backyard of three residential properties and one municipal property located on the Ringwood Mines/Landfill site (NJDEP 2005). The samples were analyzed for VOCs, SVOCs, and lead. Results

indicated the presence of VOCs (ethylbenzene, toluene, xylenes, chlorobenzene, trichloroethene) and lead. SVOC data from all four properties and lead data from one residential property were rejected due to laboratory calibration problems. The maximum VOC concentrations detected were below environmental guideline CVs. Both the maximum and mean lead concentrations detected in the surface soil of Residence 1 exceeded the RDCSCC of 400 mg/kg:

Results of Residential Surface Soil Sampling for Lead (mg/kg)			
Concentration	**Residence 1**	**Residence 2**	**Residence 3**
Maximum	3,857	68.5	Rejected
Mean	634	43.17	

Paint sludge and contaminated soil at the three residential properties were excavated and disposed off-site.

The Edison Wetlands Association also collected sludge and soil samples from these areas (Chapin Engineering 2005). Sludge sample results indicated the presence of antimony, arsenic and lead above their respective environmental guideline CVs, and that lead was leachable from the paint sludge. The post-excavation soil samples were "split samples" (with NJDEP) collected from the bottom of excavations. These results were comparable to those reported by the NJDEP.

Contaminants of Concern: Summary

Pre 1987/1988 Remediation

Paint Sludge, Soil, and Sediment - The maximum concentrations of contaminants detected in paint sludge, soil, and sediment, along with appropriate environmental guideline CVs, are presented in Tables 3 - 5. The following contaminants exceeded their corresponding CV, and as such, are designated as COCs:

COCs			
	Paint Sludge	**Soil[1]**	**Sediment**
VOCs	-	Benzene[2]	-
SVOCs	Aroclor 1248 and 1254, Bis(2-ethylhexyl)phthalate	Benzo[a]pyrene	Benzo[a]pyrene
Metals	Antimony, Arsenic, Cadmium, Chromium, Copper, Lead	Arsenic, Lead, Thallium	Arsenic, Thallium

[1]Post-remediation soil; [2]Pre-remediation test pit sample (see Table 2)

A brief discussion of the toxicologic characteristics of these COCs is presented in Appendix D.

Surface Water (Springs/Seeps, Brooks) - Maximum contaminant concentrations detected in surface water along with the respective environmental guideline CVs are presented in Tables 6 and 7. The following contaminants exceeded their CV, and as such, are selected as COCs:

Surface Water COCs	
VOCs	Benzene, 1,2-Dichloropropane
Metals	Arsenic, Lead, Mercury

A brief discussion of the toxicologic characteristics of these COCs is presented in Appendix D.

Groundwater - The maximum contaminant concentrations detected in groundwater, along with the respective environmental guideline CVs, are presented in Table 8. The following contaminants exceeded their CVs, and as such, are selected as the COCs:

Groundwater COCs	
VOCs	Benzene, Methylene Chloride, Pentachlorophenol
SVOCs	Bis(2-ethylhexyl)phthalate
Metals	Arsenic, Cadmium, Lead, Thallium

A brief discussion of the toxicologic characteristics of these COCs is presented in Appendix D.

Post 1987/1988 Remediation

Groundwater – The maximum contaminant concentrations detected in groundwater, along with their respective environmental guideline CVs, are presented in Table 9. The following contaminants exceeded their CVs, and as such, are selected as the COCs:

Groundwater COCs	
VOCs	Benzene, Chloroethane, 1,1,2,2-Tetrachloroethane
Metals	Aluminum, Antimony, Arsenic, Beryllium, Cadmium, Chromium, Cobalt, Lead, Mercury, Nickel, Thallium, Vanadium

A brief discussion of the toxicologic characteristics of these COCs is presented in Appendix D.

Off-site Potable Wells - The maximum concentrations of contaminants detected in off-site potable wells along with appropriate environmental guideline CVs are presented

in Table 10. The following contaminants in the potable wells exceeded their corresponding CVs, and as such, are selected as the COCs for the site:

COCs in the Off-site Potable Wells	
VOCs	Tetrachloroethene
Metals	Antimony, Beryllium, Lead, Silver

A brief discussion of the toxicologic characteristics of these COCs is presented in Appendix D.

On-site Residential Soil - As discussed earlier, the maximum concentrations of VOCs detected in three on-site residential properties did not exceed their corresponding CVs. Lead was identified as the COC for these properties.

Discussion

Since the presence of contaminated environmental medium does not necessarily mean that there are exposures, the next step in the public health assessment process is to determine whether there is a completed exposure pathway from a contaminant source to a receptor population.

Exposure Pathway Evaluation

An exposure pathway is a series of steps starting with the release of a contaminant to an environmental medium, movement of the contaminant, and ending at the interface with the human body. A completed exposure pathway consists of five elements:

1. source(s) of contamination;
2. environmental media and transport mechanisms;
3. point of exposure;
4. route of exposure; and
5. receptor population.

Generally, the ATSDR categorizes exposure pathways as follows: 1) *completed* exposure pathways, that is, all five elements of a pathway are present; 2) *potential* exposure pathways, that is, one or more of the elements may not be present, but information is insufficient to eliminate or exclude the element; and 3) *eliminated* exposure pathways, that is, one or more of the elements is absent. Exposure pathways are used to evaluate specific ways in which people were, are, or will be exposed to environmental contamination in the past, present, and future. Completed and potential pathways may be *interrupted* by remedial or public health interventions that disrupt the pathway. Information provided by Ringwood Mines area residents regarding circumstances of exposure to environmental contaminants was taken into consideration in evaluating exposure pathways for the Ringwood Mines/Landfill site.

Site exposures reported by residents included using on-site spring water for all domestic household use until the early 1980s, consuming vegetables from residential gardens, riding bicycles through the paint sludge and playing on Sludge Hill as children, inhaling smoke from on-site fires which occurred during the 1960s, and consuming fish and game which foraged on-site (although residents do not consume fish and game to the extent that they had in the past).

Completed Exposure Pathways

Incidental Ingestion - Paint Sludge, Soil, Sediment

Paint sludge disposal areas were located in close proximity to residences and in other areas that were easily accessible to residents (including children) and others. Children and adults reportedly accessed the contaminated areas for recreational activities (e.g., dirt riding, swimming, hiking), scavenging (auto parts, scrap metal, salvaged food dumped at the site by a local supermarket), and for subsistence fishing and hunting. At the time of disposal, the paint sludge was described by residents as a semi-soft material. Over time, the surficial paint sludge slowly solidified; it is assumed that the surface solidification took place in weeks to months. Direct exposure to fresh paint sludge during the years 1965 - 1972 was assumed to have occurred through incidental ingestion.

Due to the weathering and leaching of paint sludge, contaminants have migrated into on-site soils and sediments resulting in exposures via the incidental ingestion pathway. Four primary paint sludge areas were remediated in 1987/1988 serving to interrupt this exposure pathway to some degree. Actions to remove paint sludge deposits during the 1990s, and the November 2005 removal by NJDEP of paint sludge at three residential properties, have also served to interrupt exposures. However, paint sludge deposits and contaminated soils presently remain in scattered areas at the site, accessible to residents and others, and the site is not yet fully characterized (see Figure 6). Exposures to this contamination may have begun in 1965 when the dumping of Ford Motor Company wastes began.

Dermal Contact - Paint Sludge, Soil, Sediment, Surface Water

Dermal contact with paint sludge and contaminated soil and sediment was possible during household and recreational activities. The extent of dermal absorption of contaminants depends on the area and duration of contact, chemical and physical attraction between the contaminant and the media (loosely or tightly bound), and the ability of the contaminant to penetrate the skin. Although the potential for exposure by dermal absorption of chemicals exists, ATSDR generally considers dermal exposure to be a minor contributor to the overall exposure dose relative to contributions from ingestion and inhalation for most exposure scenarios (ATSDR 2005). However, direct dermal contact with certain contaminants (e.g., chromium, which was found in paint sludge) may elicit dermal reactions based on chemical reactivity or allergic sensitivity (Stern et al. 1993; Bagdon and Hazen 1991).

Ingestion - Surface Water

Public water was not available to all Ringwood Mines/Landfill site residents until the 1980s. Before that time, community members used buckets to collect surface water (seeps/springs, brooks) for domestic uses such as drinking and cooking. Therefore, contaminant exposures through ingestion of surface water are assumed to have occurred for about 20 years (1965 through mid 1980s). Incidental ingestion of surface waters during recreational activities may also have occurred.

A summary of completed exposure pathways identified for the site is presented in Table 11.

Potential Exposure Pathways

Inhalation - Ambient Air

Ringwood Mines area residents and others may have been exposed to organic vapors from the paint sludge as volatile chemicals off-gassed into the ambient air. Although organic vapor was not detected above background levels during site survey, drilling or excavation of test pits, and paint sludge removal activities (Woodward-Clyde 1988), it should be noted that these activities were conducted about 15 to 20 years after the paint sludge dumping. Odors were noticed during recent paint sludge delineation and remediation activities (A. Robinson, ARCADIS, personal communication, 2005) indicating that organic vapors may still be present within the sludge. Since no data are available to evaluate exposures, this exposure pathway is considered potential.

Another potential ambient air exposure pathway is associated with the mine shaft fires reported at the site. Exposure to combustion products from burning waste material associated with paint sludge may have occurred during these episodes. Residents reported being sickened by smoke from these fires. However, no air monitoring data are available to evaluate this exposure pathway.

Ingestion - Biota

Biota (e.g., fish, small game, deer, plants) living or foraging in the Ringwood Mines/Landfill site may have been exposed to contaminants in paint sludge, soil, and sediment. Contaminants may accumulate in the tissue, fat, and bone of animals, and some plants grown in contaminated soil may absorb these chemicals. For example, root crops (such as carrots, beets and potatoes) can take up arsenic and lead contamination in their roots. Lead is also found in the edible portions of leafy vegetables and herbs, as a result of uptake through the roots or deposition on the plant surfaces (ATSDR 1999a). Ringwood Mines area residents who stated that they fished and hunted the site for subsistence may have been exposed to site-related contaminants. However, no data are available to evaluate this exposure pathway.

Ingestion - Groundwater (Off site Potable Wells)

Although the EMP was discontinued in 1995, on-site groundwater remains contaminated. A number of metals (e.g., antimony, beryllium, lead) were detected above their respective CVs in on-site groundwater monitoring wells and off-site potable wells (see Tables 9 and 10). At present, there is insufficient information regarding groundwater flow and the source of off-site potable well contamination to evaluate this exposure pathway.

A summary of potential exposure pathways identified for the site is presented in Table 11.

Public Health Implications

Once it has been determined that individuals have or are likely to come in contact with site-related contaminants (i.e., a completed exposure pathway), the next step in the public health assessment process is the calculation of site-specific exposure doses. This is called a health guideline comparison which involves looking more closely at site-specific exposure conditions, the estimation of exposure doses, and the evaluation with health guideline comparison values (CVs). Health guideline CVs are based on data drawn from the epidemiologic and toxicologic literature and often include uncertainty or safety factors to ensure that they are amply protective of human health.

Completed human exposure pathways associated with the Ringwood Mines/Landfill site include the incidental ingestion of paint sludge, soils, and sediments, the ingestion of surface water from springs/seeps and brooks, and dermal exposure to sludge, soil, sediment, and surface water. Since there is insufficient information available on the nature and magnitude of potential exposures associated with the inhalation of ambient air, ingestion of biota, and the ingestion of water from off-site potable wells, an evaluation with health guideline CVs could not be conducted.

Non-Cancer Health Effects

To assess non-cancer health effects, ATSDR has developed Minimal Risk Levels (MRLs) for contaminants that are commonly found at hazardous waste sites. An MRL is an estimate of the daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of adverse, non-cancer health effects. MRLs are developed for a route of exposure, i.e., ingestion or inhalation, over a specified time period, e.g., acute (less than 14 days); intermediate (15-364 days); and chronic (365 days or more). MRLs are usually extrapolated doses from observed effect levels in animal toxicological studies or occupational studies, and are adjusted by a series of uncertainty (or safety) factors or through the use of statistical models. In toxicological literature, observed effect levels include:

- no-observed-adverse-effect level (NOAEL); and
- lowest-observed-adverse-effect level (LOAEL).

A NOAEL is the highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or in experimental animals. A LOAEL is the lowest dose of a substance that has been reported to cause harmful (adverse) health effects in people or in experimental animals. In order to provide additional perspective on the potential for adverse health effects, calculated exposure doses may also be compared to the NOAEL or LOAEL. As the exposure dose increases beyond the MRL to the level of the NOAEL and/or LOAEL, the likelihood of adverse health effects increases.

To ensure that MRLs are sufficiently protective, the extrapolated values can be several hundred times lower than the observed effect levels in studies of people or experimental animals. When MRLs for specific contaminants are unavailable, other health based comparison values such as the USEPA's Reference Dose (RfD) are used. The RfD is an estimate of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime of exposure.

Ingestion - Sludge, Soil, and Sediment

Non-cancer health effects associated with the selected COCs (see Tables 3, 4, and 5) were assessed by comparing child and adult exposure doses with health guideline CVs. Contaminant exposure doses were calculated using the following formula:

$$\textit{Exposure Dose (mg/kg/day)} = \frac{C \times IR \times EF}{BW}$$

where, mg/kg/day = milligrams of contaminant per kilogram of body weight per day;

C = concentration of contaminant (mg/kg);
IR = soil ingestion rate (kg/day);
EF = exposure factor representing the site-specific exposure scenario; and,
BW = body weight (kg).

Since available data represent a snapshot in time, it is not possible to definitively determine the level or duration of individual resident exposure. However, given that the potential for exposure persisted with no or limited interruption (i.e., paint sludge remedial actions), it is assumed that the exposure duration is seven years (i.e., 1965 - 1972) for paint sludge (semi-soft sludge) and 40 years (i.e., 1965 - 2005) for soil (including solidified sludge) and sediment. It is further assumed that on average, exposures were intermittent (three days per week, nine months per year). The following assumptions were used to calculate site-specific exposure doses for children and adults:

Exposure Scenario Assumptions[1]					
Media	**Receptor Population**	**Ingestion Rate (mg/day)**	**No. of Days of Exposure Per Year**	**Years Exposed**	**Body Weight (kg)**
Paint Sludge	Child	200	108 days (3 days per week, 9 months per year)	7	16
	Adult	100			70
Soil	Child	200		10 (child) 40 (adult)	16
	Adult	100			70
Sediment	Child	200		10 (child) 40 (adult)	16
	Adult	100			70

[1]USEPA 1991; USEPA 1997; NJDEP 2004; ATSDR 2005

Paint Sludge. Maximum chronic exposure doses calculated for children and adults for bis(2-ethylhexyl)phthalate, arsenic, cadmium, and copper were lower than their corresponding health guideline CV and, therefore, are unlikely to cause adverse non-cancer health effects (see Table 12). The USEPA Region 3 RfD for chronic Aroclor 1248 exposure was unavailable; however, the RfD for Aroclor 1254 is 0.00002 mg/kg/day. Using the sum of Aroclor 1248 and 1254, the estimated exposure dose (i.e., 0.000011 mg/kg/day) was lower than the RfD. As such, non-cancer health effects associated with ingestion of PCBs in paint sludge are not expected.

Calculated child and adult exposure doses for antimony and chromium exceeded their respective health guideline CV (see Table 12). As such, the potential exists for non-cancer adverse health effects; a brief evaluation of the non-cancer health implications is presented below. Although an RfD is unavailable for lead, it has also been evaluated for possible non-cancer adverse health effects.

Antimony - Ingesting large doses of antimony can cause vomiting. Long-term chronic animal studies have also reported liver damage and blood changes (ATSDR 1992). Although information on the toxic effects of chronic oral exposure to antimony is limited, antimony appears to affect heart muscle, the gastrointestinal tract, and the nervous system. The chronic oral RfD for antimony (0.0004 mg/kg/day) is based on reduced longevity, blood glucose, and altered cholesterol levels of a group of male and female rats in an oral bioassay study. A LOAEL of 0.35 mg/kg/day and an uncertainty factor of 1,000 were used to calculate the oral RfD. Based on the maximum concentration of antimony detected in the paint sludge, the exposure dose calculated for children (1.85 mg/kg/day) exceeded the LOAEL whereas the adult exposure dose (0.21 mg/kg/day) was lower than the LOAEL by a factor of 1.7 (see Table 12). Based on the mean concentration of antimony detected in the paint sludge, child and adult exposure doses (0.19 and 0.021 mg/kg/day) were lower than the LOAEL by a factor of 1.8 and 17, respectively. Based on the dose being near the level that showed effect in animal studies, there was a potential for non-cancer adverse health effects in children and adults from

incidental ingestion of antimony in paint sludge. No health guideline CVs are available to evaluate potential acute and intermediate duration exposures.

Chromium – Chromium may occur in several forms; in nature, chromium (III) is much more common than the more toxic chromium (VI) (USEPA 1994a; NJDEP 1998). Chromium measured in the paint sludge was reported as total chromium. Since the form of chromium in soil is a function of source materials and environmental conditions, to be conservative, the total chromium was assumed to be in the more toxic chromium (VI) form. It should be noted, however, that this assumption may result in an overestimation of exposure dose and potential for health effects.

The chronic oral RfD for chromium (VI) of 0.003 mg/kg/day is based on reduced water consumption in a group of male and female rats (USEPA 2005). An uncertainty factor of 900 and a NOAEL (i.e., the dose that showed no effect in animal studies) of 2.5 mg/kg/day were used to calculate the oral RfD. Based on the maximum and mean concentration of chromium detected in the paint sludge, the child exposure doses (i.e., 0.009 mg/kg/day and 0.0066 mg/kg/day) were 277 and 378 times lower than the NOAEL, respectively (see Table 12). Based on the fact that RfD is based on NOAEL and all chromium detected was assumed to be in the chromium (VI) form, non-cancer adverse health effects for exposures by ingestion to chromium detected in sludge is low.

Lead - The maximum and mean lead concentration detected in paint sludge was 310,000 mg/kg and 64,880 mg/kg, respectively (see Table 12). The maximum concentration was about 775 times higher than the RDCSCC (400 mg/kg). No MRL or RfD is available for lead (ATSDR 1999a). Health effects associated with lead exposure, particularly changes in children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold (i.e., no NOAEL or LOAEL is available). Accumulation of lead in the body can cause damage to the nervous and gastrointestinal systems, kidneys, and red blood cells. Children, infants, and fetuses are the most sensitive populations to lead exposures. Lead may cause learning difficulties and stunted growth, and may endanger fetal development.

Lead exposures associated with the intermittent recreational use of paint sludge contaminated areas at the Ringwood Mines Landfill site were evaluated using the USEPA's integrated exposure uptake biokinetic (IEUBK) model (USEPA 1994b). The IEUBK model estimates a plausible distribution of blood lead levels centered on the geometric mean blood lead levels from available exposure information. Blood lead levels are indicators of recent exposure, and are also the most widely used index of internal lead body burdens associated with potential health effects. The model also calculates the probability (or P_{10}) that children's blood lead levels will exceed a level of concern. Health effects of concern have been determined to be associated with childhood blood lead levels at 10 micrograms of lead per deciliter of blood (or μg/dL) or less (USEPA 1986, 1990; CDC 1991). In using the IUEBK model, the USEPA recommends that the lead concentration in site soil does not result in a 5% probability of exceeding a blood lead concentration of 10 μg/dL (USEPA 1994c). The average lead level in paint sludge (64,880 mg/kg; see Table 12) was used as an input value to calculate expected

children's blood lead levels due to incidental ingestion of paint sludge during the time frame of 1965 - 1972. The assumptions for the recreational exposure scenario for children aged six to 84 months are as follows:

1. Children were exposed to paint sludge containing lead each time the site was visited. The site visit frequency was three days per week over nine months of the year; exposure during the remaining days of the week was at the residence.
2. Model default values were used for all other variables (USEPA 2002) including residential soil and dust.

The predicted geometric mean blood lead levels and the probability of blood lead levels exceeding 10 µg/dL (P_{10}) for children are shown in the following table:

Exposure Scenario		
Age (months)	**Three Site Visits Per Week[1]**	
	Blood Lead Level[2] (µg/dL)	**P_{10} (%)[3]**
6 -12	52	99.97
12 - 24	59	99.99
24 - 36	57	99.98
36 - 48	56	99.98
48 - 60	50	99.97
60 - 72	45	99.93
72 - 84	41	99.88

[1]background soil lead concentration = 200 ppm; weighted paint sludge lead concentration (64,880 ppm x 3/7) + (200 ppm x 4/7) = 27,920 ppm (USEPA 2003a); [2]Geometric mean lead levels in blood; [3]probability of blood lead level > 10 µg/dL

For the incidental paint sludge ingestion exposure scenario, the model predicted that the blood lead levels for children ages 6 - 84 months were four to six times higher than the level of concern (10 µg/dL). In addition, the probabilities of blood lead levels exceeding 10 µg/dL for children ages 6 - 84 months was near 100 percent. Therefore, for children exposed to paint sludge contaminated areas at the Ringwood Mines/Landfill site in the period 1965 - 1972, the predicted blood lead levels could have been extremely high. An adult blood lead model estimated a geometric mean blood lead level of 42 µg/dL (USEPA 2003b).

It is important to note that the IEUBK model should not be relied upon to accurately predict blood lead levels above 30 µg/dL since the model was not empirically validated. Additionally, the model should not be used for exposure periods of less than three months, or in which a higher exposure occurs less than once per week or varies irregularly.

Soil. Since several paint sludge contaminated areas remain and are currently being delineated and remediated, exposure to soil contaminants was assumed to be 40 years (1965 - 2005). The maximum chronic exposure dose calculated for children and adult for benzene, arsenic, and thallium are lower than their corresponding health

guideline CVs, and, therefore, are unlikely to cause non-cancer adverse health effects (see Table 13).

Benzo[a]pyrene - Benzo[a]pyrene, was also detected in the soil. Benzo[a]pyrene is one of a group of compounds called polycyclic aromatic hydrocarbons (PAHs). PAHs are formed as a result of incomplete combustion of organic materials. Many industrial products contain PAHs, including coal tar, roofing tar, and creosote. Additionally, the burning of rubber tires can generate PAHs. No acute or chronic MRL have been derived for Benzo[a]pyrene because no adequate human or animal dose-response data are available that identify threshold levels for appropriate non-cancer health effects. However, intermediate duration oral MRLs of 0.4 mg/kg/day have been derived for fluoranthene and fluorene; both were based on LOAELs of 125 mg/kg/day for increased relative liver weight in male mice (ATSDR 1999b). Based on the maximum concentration of Benzo[a]pyrene detected in soil, the estimated child and adult dose of 7.23×10^{-7} and 8.27×10^{-8} mg/kg/day, respectively are several orders of magnitude lower than the most conservative MRL of 0.4 mg/kg/day for any of the PAHs (see Table 13). Therefore, it is unlikely that non-cancer adverse health effects would occur in children or adults. This determination takes into account that PAHs have similar physical, chemical, and toxicological characteristics.

Lead - The maximum concentration of lead detected in non-residential site soils (1,300 mg/kg) was about three times higher than the RDCSCC, however, the mean concentration (129.6 mg/kg) was lower than the RDCSCC. Health effects associated with lead exposures were presented earlier in this section.

Residential Soil. Lead contamination above the RDCSCC was detected in residential properties located on the Ringwood Mines/Landfill site.

Lead - The maximum and mean concentrations of lead detected in Residence 1 (3,857 and 634 mg/kg) exceeded the RDCSCC. As discussed earlier, the IEUBK model may be used to evaluate the residential soil exposure pathway. The assumptions for the residential exposure scenario for children ages 6 - 84 months are:

- Children were exposed to residential lead contaminated soil and dust, and,
- Model default values were used for all other variables.

The predicted geometric mean blood lead levels and the probability of blood lead levels exceeding 10 µg/dL (P_{10}) for children are shown below:

Age (months)	Exposure Scenario			
	Maximum Lead Concentration (3,857 mg/kg)		Mean Lead Concentration (634 mg/kg)	
	Blood Lead Level[1] (µg/dL)	P_{10} (%)[2]	Blood Lead Level (µg/dL)	P_{10} (%)
6 -12	24	97	7.5	28
12 - 24	27	98	8.6	38
24 - 36	26	98	8.1	33
36 - 48	26	98	7.7	29
48 - 60	23	96	6.4	18
60 - 72	20	93	5.5	10
72 - 84	18	89	4.9	6

[1]Geometric Mean lead levels in blood; [2]probability of blood lead level > 10 µg/dL

For residential exposures to maximum lead soil concentration detected in Residence 1, the model predicted that the blood lead levels for the ages 6 - 84 months were considerably elevated above 10 µg/dL. In addition, the probabilities of blood lead levels exceeding 10 µg/dL for children ages 6 - 84 months was from 89 to 97 percent. For residential exposures to mean concentration, the predicted blood lead levels for the ages 6 - 84 months were below 10 µg/dL. However, the probabilities of blood lead levels exceeding 10 µg/dL for children ages 6 - 84 months was from 6 to 38 percent.

Sediment. The maximum chronic exposure dose calculated for children and adult for arsenic and thallium are lower than the corresponding health guideline CVs (see Table 14), and, therefore, are unlikely to cause non-cancer adverse health effects.

Benzo[a]pyrene - Benzo[a]pyrene was also detected in sediment. As discussed earlier, no acute or chronic MRL have been derived for Benzo[a]pyrene; however, intermediate duration oral MRLs of 0.4 mg/kg/day have been derived for fluoranthene and for fluorene (ATSDR 1995). Based on the maximum concentration of Benzo[a]pyrene detected in sediment, the estimated child and adult dose of 2.45×10^{-6} and 2.08×10^{-7} mg/kg/day, respectively are several orders of magnitude lower than the most conservative MRL of 0.4 mg/kg/day for any of the PAHs (see Table 14). Therefore, it is unlikely that non-cancer adverse health effects would occur in children or adults.

Ingestion - Surface Water (Brooks, Springs/Seeps)

The evaluation of potential non-cancer health effects for the selected COCs (see Table 6 and 7) in surface water is accomplished by estimating the amount or dose of those contaminants that an adult or child might have ingested on a daily basis. The contaminant exposure dose is calculated using the following formula:

$$\textit{Exposure Dose (mg/kg/day)} = \frac{C \times IR}{BW}$$

where, mg/kg/day = milligrams of contaminant per kilogram of body weight per day;

C = concentration of contaminant in water (milligrams per liter or mg/L);
IR = ingestion rate (liters per day or L/day); and,
BW = body weight (kg)

Based on the historical information, it was assumed that Ringwood Mines/Landfill area residents were exposed to surface water contaminants for approximately 20 years (i.e., from 1965 to mid 1980s). The following assumptions were used to estimate the site-specific exposure doses for children and adult.

Exposure Scenario Assumptions				
Water Source	**Exposed Population**	**Ingestion Rate (L/day)**	**Years Exposed**	**Body Weight (kg)**
Surface	Child	1	10 (child)	16
	Adult	2	20 (adult)	70

Based on the maximum concentrations of benzene and 1,2-dichloropropane detected, exposure doses calculated for children and adults were lower than their corresponding health guideline CV and are unlikely to cause adverse non-cancer health effects (see Table 15). Based on the maximum (40 μg/L) and mean (16.56 μg/L) arsenic concentrations detected, exposure doses for children and adults were higher than the corresponding health guideline CV (see Table 15). Although health guideline CVs are unavailable for mercury and lead, non-cancer adverse health effects are discussed below.

Arsenic - Arsenic is a naturally occurring element widely distributed in the earth's crust. The MRL for arsenic is set at a level meant to protect against non-cancer health effects, specifically dermal lesions (ATSDR 2000). Chronic exposure to low levels of inorganic arsenic can cause a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. Skin contact with inorganic arsenic may cause redness and swelling. Organic arsenic compounds are less toxic than inorganic arsenic compounds.

Based on the maximum concentration of arsenic detected in surface water, the chronic exposure dose calculated for children and adults (i.e., 0.0025 mg/kg/day and 0.0011 mg/kg/day) exceeded the ATSDR MRL of 0.0003 mg/kg/day (see Table 15). The calculated child and adult exposure doses are about 3.1 and 1.4 times higher than the NOAEL (i.e., 0.0008 mg/kg/day), respectively. Additionally, based on the mean concentration of arsenic detected (the more likely exposure scenario), the calculated chronic exposure dose for child was about 1.25 times higher than the NOAEL. As such,

there is a potential for non-cancer adverse health effects from exposures to arsenic in surface water in the period 1965 - 1985 when the water was used for potable purposes.

Mercury - Thirty percent (6/20) of the samples collected from seeps were contaminated with mercury in the Ringwood Mines/Landfill site. Since a chronic oral MRL and RfD are unavailable for mercury, the calculated exposure dose for children and adults could not be compared to a health guideline CV (see Table 15). However, an intermediate oral MRL for mercury is available (0.002 mg/kg/day) and is based on increased kidney weight of rats exposed to mercuric chloride once every five days for twenty-six weeks (ATSDR 1999c). An uncertainty factor of 100 and a NOAEL of 0.23 mg/kg/day were used to calculate the MRL. Maximum exposure doses calculated for children and adults (i.e., 0.00083 mg/kg/day and 0.00025 mg/kg/day) were about 277 and 920 times lower than the oral intermediate NOAEL, respectively. It should also be noted that the oral RfD for mercuric chloride ($HgCl_2$) and methylmercury (CH_3Hg) are 0.0003 mg/kg/day and 0.0001 mg/kg/day, respectively (USEPA 2005). As such, although the exposure to mercury may have continued for about 20 years, the likelihood of non-cancer adverse health effects in area residents is considered low.

Lead - Both the maximum and the mean concentration of lead detected in the surface water exceeded the New Jersey action level (see Table 7). As discussed earlier, the IEUBK model may be used to evaluate the surface water ingestion pathway. The assumptions for the residential exposure scenario for children ages 6 - 84 months are:

- Children were exposed to lead through potable water, and,
- Model default values were used for all other variables.

The predicted geometric mean blood lead levels and the probability of blood lead levels exceeding 10 µg/dL (P_{10}) for children are shown in the following table:

Age (months)	**Exposure Scenario**			
	Maximum concentration = 120 µg/L		**Mean concentration = 105 µg/L**	
	Blood Lead Level[1] (µg/dL)	**P_{10} (%)[2]**	**Blood Lead Level (µg/dL)**	**P_{10} (%)**
6 -12	8.4	36	7.9	30
12 – 24	11.8	64	11	58
24 – 36	11.7	63	10.8	57
36 – 48	11.5	62	10.6	55
48 – 60	11	59	10.1	51
60 – 72	10.7	58	9.8	48
72 – 84	10.1	52	9.3	44

[1]Geometric Mean lead levels in blood; [2]probability of blood lead level > 10 µg/dL

For ingestion exposures to maximum lead concentration detected in surface water, the predicted blood lead levels in children for ages 6 - 84 months were from 8.4 to 11.8 µg/dL. In addition, the probabilities of blood lead levels exceeding 10 µg/dL for children

ages 6 - 84 months was from 36 to 64 percent. For ingestion exposures to mean lead concentration detected in surface water, the predicted blood lead levels for children ages 6 - 84 months were between 7.9 and 10.8 µg/dL. However, the probabilities of blood lead levels exceeding 10 µg/dL for children ages 6 - 84 months was between 30 and 58 percent for the period 1965 - 1985 when the water was used for potable purposes.

Cancer Health Effects

The site-specific lifetime excess cancer risk (LECR) indicates the cancer potential of contaminants. LECR estimates are usually expressed in terms of excess cancer cases in an exposed population in addition to the background rate of cancer. For perspective, the lifetime risk of being diagnosed with cancer in the United States is 46 per 100 individuals for males, and 38 per 100 for females; the lifetime risk of being diagnosed with any of several common types of cancer ranges approximately between 1 in 100 and 10 in 100 (SEER 2005). Typically, health guideline CVs developed for carcinogens are based on a lifetime risk of one excess cancer case per 1,000,000 individuals. ATSDR considers estimated cancer risks of less than one additional cancer case among one million persons exposed as insignificant or no increased risk (expressed exponentially as 10^{-6}).

According to the United States Department of Health and Human Services (USDHHS), the cancer class of contaminants detected at a site is as follows:

1 = Known human carcinogen
2 = Reasonably anticipated to be a carcinogen
3 = Not classified

Ingestion - Sludge, Soil and Sediment

The cancer class of the COCs detected in the sludge, soil and sediment are given in Tables 16, 17, and 18. The tables show that bis(2-ethylhexyl)phthalate, PCBs, arsenic, cadmium, chromium in the paint sludge, benzene, benzo[a]pyrene, arsenic in the surface soil, and benzo[a]pyrene, arsenic in the sediment have the potential to cause cancer among exposed populations.

Estimated cancer exposure doses were calculated using the following formula:

$$\textit{Cancer Exposure Dose (mg/kg/day)} = \frac{C \times IR \times EF}{BW} \times \frac{ED}{AT}$$

where C = concentration of contaminant in soil (mg/kg);
IR = soil ingestion rate (kg/day);
EF = exposure factor representing the site-specific exposure scenario;
ED = exposure duration (year);
BW = body weight (kg); and,
AT = averaging time (year).

The assumptions used to calculate site-specific exposure doses were the same as described previously for non-cancer health effects. The LECR for adults was calculated by multiplying the cancer exposure dose by the cancer slope factor (CSF). The CSF is defined as the slope of the dose-response curve obtained from animal and/or human cancer studies and is expressed as the inverse of the daily exposure dose, i.e., $(mg/kg/day)^{-1}$.

Paint Sludge. Of the COCs identified in the paint sludge, arsenic is classified as a known human carcinogen, and bis(2-ethylhexyl)phthalate and Aroclors 1248 and 1254 are classified as reasonably anticipated to be carcinogens among exposed populations (see Table 16). Carcinogenicity information of chromium by oral exposure in humans is inadequate. Limited epidemiologic studies have indicated that exposure to cadmium in food or drinking water is not carcinogenic.

Based on the maximum concentration of arsenic detected in paint sludge, the LECR calculated was one in 1,000,000 to the exposed population (see Table 16). At the mean arsenic concentration (4.33 mg/kg), the more likely exposure scenario, the LECR was three in 10,000,000 to the exposed population. Overall, the LECRs associated with the contaminants indicated five in 100,000,000 to one in 1,000,000 based on the maximum and the mean concentrations, respectively.

Surface Soil. Of the COCs identified in the surface soil, benzene and arsenic are classified as known human carcinogens and benzo(a)pyrene is classified as reasonably anticipated to be a carcinogen among exposed populations (see Table 17). At the maximum concentration of contaminants in the surface soil, the LECR calculated was seven in 1,000,000 to the exposed population for arsenic (see Table 17). At the mean arsenic concentration (2.03 mg/kg), the more likely exposure scenario, the LECR was one in 1,000,000 to the exposed population.

The LECR calculated for other carcinogens (benzene, benzo[a]pyrene) were below one in 1,000,000 to the exposed population.

Sediment. Of the COCs identified in the sediment, arsenic is classified as a known human carcinogen and benzo[a]pyrene is classified as reasonably anticipated to be a carcinogen among exposed populations (see Table 18). Based on the maximum concentration of arsenic (31.4 mg/kg) detected in the sediment, the calculated LECR was one in 100,000 to the exposed population. Based on the mean concentration (9.13 mg/kg) of arsenic detected in the sediment (i.e., the more likely exposure scenario), the LECR was four in 1,000,000 to the exposed population. The LECR calculated for benzo[a]pyrene was one in 1,000,000 to the exposed population.

In summary, excess cancer risk from ingestion of paint sludge, surface soil, and sediment is estimated to be very low when compared to background cancer risk (see Figure 7).

Lead in paint sludge and surface soil. Although lead has not been classified as a carcinogen by the USDHHS[9], the carcinogenicity of inorganic lead and lead compounds have been evaluated by the USEPA (USEPA 1986, 1989). The USEPA has determined that data from human studies are inadequate for evaluating the carcinogenicity of lead, but there is sufficient data from animal studies which demonstrate that lead induces renal tumors in experimental animals. In addition, there are some animal studies which have shown evidence of tumor induction at other sites (i.e., cerebral gliomas; testicular, adrenal, prostate, pituitary, and thyroid tumors). A cancer slope factor has not been derived for inorganic lead or lead compounds, so no estimation of LECR can be made for lead exposure.

Ingestion - Surface Water (seeps/springs, brooks)

The ingestion cancer exposure doses were calculated using the following formula:

$$\textit{Cancer Exposure Dose (mg/kg/day)} = \frac{C \times IR}{BW} \times \frac{ED}{AT}$$

where, C = concentration of contaminant in water (mg/L)
IR = contact rate (L/day)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (years)

LECRs were calculated by multiplying the cancer exposure dose with the CSF. The USDHHS cancer class for the contaminants of concern in the surface water and springs is presented in Table 19.

Surface Water. Of the COCs identified in the surface water, benzene and arsenic are classified as known human carcinogens among exposed populations (see Table 19). Based on the maximum and mean concentrations of benzene detected in surface water, the calculated LECRs are nine and seven in 10,000,000, respectively. Based on the maximum concentration of arsenic detected in the surface water, the calculated LECR was five in 10,000 to the exposed population (see Table 19). Based on the mean concentration (16.56 μg/L), i.e., the more likely exposure scenario, the calculated LECR for arsenic was two in 10,000 to the exposed population (see Figure 7).

Assessment of Joint Toxic Action of Chemical Mixtures

In the Ringwood Mines/Landfill site, residents may have been exposed to a number of contaminants detected in paint sludge, soil, sediment and surface water. Exposure to multiple chemicals with similar toxicological characteristics may increase

[9]Lead and Lead Compounds are listed in the Eleventh Edition of the Report on Carcinogens as ''reasonably anticipated to be human carcinogens'' (NTP 2006)

their public health impact. The severity of the impact depends on the particular chemicals being ingested, pharmacokinetics, and toxicity in children and adults.

To assess the risk for non-cancer adverse health effects of chemical mixtures, the hazard indexes (HI) and the ratio of exposure dose to NOAEL for the contaminants was calculated (see Appendix E for details). The results indicated that potential exists for additive or interactive effects of chemical mixtures from exposures to paint sludge and surface water, particularly for neurological effects associated with co-exposure to lead and arsenic (ATSDR 2004; ATSDR 2005).

Child Health Considerations

The NJDHSS and ATSDR recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination in their environment. Children are at greater risk than adults from certain types of exposures to hazardous substances. Their lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. The developing body systems of children can sustain permanent damage if toxic exposures occur during critical growth stages. Most important, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care.

The NJDHSS and ATSDR evaluated the potential risk for children residing in the Ringwood Mines area who were exposed to site contaminants. Exposures at the site (based on lead and antimony contamination of paint sludge, arsenic contamination of surface water, and lead contamination of soil and surface water) were found to have the potential to cause non-cancer adverse health effects in children. LECRs associated with the ingestion of paint sludge, surface soil, and sediment was estimated to be very low when compared to background cancer risk. Based on the maximum and mean concentrations of arsenic detected in surface water, the calculated LECRs were estimated to be approximately five and two excess cancer cases per 10,000 individuals (including exposure as children), respectively.

Health Outcome Data

Community members have raised health concerns they feel are related to exposures associated with site contamination. Those health concerns include cancer (ovarian, cervical, leukemia, breast, lung, Ewing sarcoma, colon), respiratory disease (asthma, emphysema), reproductive and developmental effects (female reproductive disorders, miscarriages, birth defects, learning disabilities, behavioral problems), neurological disorders, heart disease, skin rashes, eye irritation, anemia, and diabetes. With the exception of cancers and birth defects, these conditions are not reportable, and documentation on the frequency of each of these conditions over time is not available in any community in New Jersey. Cancer has been a reportable disease since late 1978 and has been evaluated for this public health assessment. Birth defect data are available, but

because of the small size of the population and the rarity of the outcomes, these data have not been reviewed for this public health assessment.

Because of the potential for exposure to lead in contaminated site media, data on childhood blood lead tests were evaluated for the community. Information from the NJDHSS' Childhood Lead Poisoning Surveillance System is summarized below.

Childhood Lead Exposure

Since lead is an important contaminant associated with the paint sludge at the Ringwood Mines/Landfill site, the NJDHSS evaluated data on childhood blood lead levels. Blood lead is an excellent indicator of exposure to lead. Current state regulations, in accordance with federal Centers for Disease Control and Prevention (CDC) guidelines, require health care providers to do a blood lead test on all one and two year old children. This is the age at which lead poisoning is most damaging to the developing nervous system. State regulation requires all clinical laboratories to report the results of all blood lead tests to the NJDHSS. Prior to July 1999, only blood lead tests above 20 micrograms per deciliter (µg/dL) were reportable. While the current CDC blood lead guideline is 10 µg/dL, all blood-lead test data are reportable to the NJDHSS' Childhood Lead Poisoning Prevention Surveillance System.

Data from the Childhood Lead Poisoning Prevention Surveillance System was reviewed for the period July 1999 through October 2005 for Ringwood Borough. For the purpose of this discussion, children with multiple tests were assigned their highest blood lead level. A total of 909 Ringwood children were tested during this period.

The NJDHSS defined a "Focus Area" comprising the population in the Ringwood Mines area, to better understand the potential for exposure due to this site. The Focus Area includes children living on the following streets: Peters Mine Road, Cannon Mine Road, Horseshoe Bend Road, Van Dunk Lane, Milligan Drive, Petzold Avenue, Sloatsburg Road, Farm Road, Industrial Parkway, Boro Parkway, Chicken House Road, Manor Road, Margaret King Avenue, and Cable House Road. Of the 909 Ringwood children tested for blood lead between July 1999 and October 2005, 45 lived in the Focus Area, 861 lived in non-Focus Area locations in Ringwood, and three had insufficient address information to determine residential location.

For the non-Focus Area, seven children had a blood lead level of 10 µg/dL or higher. The rate of elevated blood lead level was 8 children per 1,000 tested. The range of blood lead levels was 1 to 26 µg/dL with a geometric average of 2.5 µg/dL (95% confidence interval: 2.4 to 2.6 µg/dL). The average age at time of the test for non-Focus Area children was 28 months, with a range of less than one month to 198 months.

For the Focus Area, two children had a blood lead level of 10 µg/dL or higher. The rate of elevated blood lead level was 44 children per 1,000 tested. The range of blood lead levels was 1 to 28 µg/dL with a geometric average of 3.7 µg/dL (95% confidence interval: 3.0 to 4.4 µg/dL). The geometric average blood lead level was

statistically significantly higher in the Focus Area children than the non-Focus Area children. The average age at time of the test for Focus Area children was 30 months, with a range of two to 113 months.

In Figure 8, childhood blood lead levels were categorized into 14 2-µg/dL intervals by Area, and displayed as a percentage for each category. While most children had a blood lead level below the 10 µg/dL level, there appears to be a slight shift to the right (higher levels) in the distribution of blood lead levels in the Focus Area children. This shift in the distribution of blood lead levels in Focus Area children could be an artifact due to the relatively small sample size, or it could indicate that these children had slightly more exposure to lead in the environment than non-Focus Area children.

The Ringwood Health Department has followed up on the two children in the Focus Area whose blood lead levels exceeded 10 µg/dL. The elevated blood lead level for one child was attributed to potential exposure to lead in paint sludge, while for the other child the likely cause of elevated blood lead was lead paint during home renovation (S. Wogish, Ringwood Borough Health Department, personal communication, 2003).

The occurrence of a child with an elevated blood lead level associated with potential exposures to contaminated soils is consistent with lead model estimates (based on limited data available for Residence 1) for average lead levels in residential soils.

Cancer Incidence

The NJDHSS and ATSDR evaluated cancer incidence in the population living near the Ringwood Mines/Landfill site (see Appendix F for a detailed report). Total cancer incidence and 13 specific cancer types were evaluated. The specific cancers types were selected because they represent cancer groupings that may be more sensitive to the effects of environmental exposure, in general. The New Jersey State Cancer Registry, a population-based cancer incidence registry covering the entire state, was used for the ascertainment of cancer cases. The study period for this investigation was January 1, 1979 through December 31, 2002. Standardized incidence ratios (SIRs) were used for the quantitative analysis of cancer incidence. The SIR compares the observed number of cases to an expected number of cases based on average state rates. Males and females, all races combined, were evaluated separately. Cancer data was evaluated for all of Ringwood and for the area of town closest to the site. As with the analysis of blood lead levels, this area is called the Focus Area for this discussion (see Appendix F Figure 1).

For Ringwood Borough as a whole, neither all cancers combined nor any of the 13 specific cancer types were elevated compared to the state. For the Focus Area, lung cancer in males was significantly higher than expected (SIR=2.8). Lung cancer in Focus Area females was slightly lower than expected (SIR=0.9). No other specific cancer types analyzed were significantly higher than expected, that is, differences from expected are within the range of variation due to chance.

Cancer is a group of more than 100 different diseases (i.e., cancer types and subtypes); each cancer type has its own set of risk factors. The multifactorial nature of cancer etiology, where a given disease may have more than one cause, complicates the evaluation of potential risk factors and specific disease outcomes. Known or probable human carcinogens were found in completed human exposure pathways at the Ringwood Mines/Landfill site. Arsenic has been identified as a possible risk factor for certain cancer types, including lung cancer (ATSDR 2000). PAHs are considered a probable human carcinogen based on animal experiments and may increase the risk of developing cancer, especially lung and skin cancers (American Cancer Society 2004 and ATSDR 1995).

While there are multiple risk factors for lung cancer, tobacco smoking is considered the most important risk factor, estimated to account for more than 85% of all lung cancer cases (National Cancer Institute, 1996). Other known risk factors for lung cancer include indoor exposure to radon and environmental tobacco smoke, occupational exposure to asbestos and other cancer-causing agents in the workplace (including radioactive ores; chemicals such as arsenic, vinyl chloride, nickel, chromates, coal products, mustard gas, and chloromethyl ethers; fuels such as gasoline; and diesel exhaust), and exposure to air pollution (American Cancer Society, 2004).

The overall cancer incidence (all cancers combined) was not elevated in the Focus Area. Lung cancer in males was significantly higher than expected while lung cancer in females was slightly lower than expected in the Focus Area. Since smoking histories are not available in the NJSCR, it is unknown what influence this important risk factor may have played. Given that lung cancer incidence in females is lower than expected, the current analysis provides little evidence that the rate of cancer incidence in the Focus Area population is due to potential exposure to Ringwood Mines contamination.

Evaluation of Other Community Health Concerns

In addition to cancers, the community has raised other health concerns they feel are related to exposures associated with site contamination: respiratory diseases (asthma, emphysema), reproductive effects (female reproductive disorders, miscarriages, birth defects), developmental effects (learning disabilities, behavioral problems), neurological disorders, heart disease, skin rashes and eye irritation, anemia, and diabetes. Community members have also expressed concerns about early mortality.

This is a diverse list of diseases and conditions, each of which may be caused by multiple non-environmental and environmental factors. There is no systematically collected surveillance data for these diseases (except for birth defects) in New Jersey, so an analysis of data cannot be conducted. As such, these community health concerns will be discussed in relation to the known or suspected toxicologic characteristics of the chemicals in completed exposure pathways that had the potential to cause non-cancer adverse health effects. The evaluation is based on the health effects reported in ATSDR's Toxicological Profiles for lead, antimony and arsenic. (Detailed discussions of general toxicologic characteristics of these chemicals are found in Appendix D.)

Although a quantitative assessment of exposure to chromium by dermal exposure was not conducted, chromium will be included in this discussion.

Lead. Lead exposure may affect many body organs and systems, causing effects in the gastrointestinal tract, hematopoietic system (blood making organs), cardiovascular system (blood pressure), central and peripheral nervous systems, kidneys, immune system, and reproductive system. Based on the exposure dose estimates for the Ringwood Mines/Landfill site discussed earlier, the highest estimated exposures would have occurred through the ingestion of paint sludge around the time of dumping. Lead in soils, particularly in residential areas, has also been a significant contributor to lead exposure; removal of paint sludge has decreased the potential for this exposure over the years.

Lead exposures are generally expressed in terms of concentration of blood lead. The concentration of blood lead reflects mainly the exposure history of the previous few months and does not necessarily reflect the cumulative exposure to lead over longer periods of time. Depending on the length of exposure, lead may accumulate in bone.

Based on presumed exposure scenarios, the child and adult blood lead level associated with ingestion of lead in paint sludge were calculated using the IEUBK and adult lead models, respectively. For adults, mean blood lead levels were estimated to reach 40 µg/dL, and for children, mean blood lead levels exceeded this level. Blood lead data are not available to determine whether the levels in children or adults reached these modeled levels in the past, in the population living near the Ringwood Mines/Landfill site. Based on the maximum and mean soil lead levels detected in residential soils, the model predicted a mean blood level up to 27 µg/dL and up to 8.6 µg/dL for children, respectively. As discussed above, additional lead exposure may have occurred during the time that surface water was used as a drinking water source.

It should be noted that the mean current blood lead level among children aged 1 - 5 years in the U.S. is approximately 2 µg/dL (CDC 2005). However, general population exposures were considerably higher in the past due to the use of tetraethyl lead in gasoline. For example, in the late-1970s, the geometric mean blood lead levels in children (1 - 5 years) in the US were 15 µg/dL (ATSDR 1999a).

For children, blood lead levels exceeding 30 µg/dL may result in delayed nerve conduction velocity. Levels above 40 µg/dL may cause depressions in hemoglobin levels, and levels above 60 µg/dL may result in gastrointestinal disturbances such as colic. Blood lead levels in children above 70 µg/dL may result in serious effects on brain function (encephalopathy). Lower levels of blood lead in children may also increase the risk of certain health effects. Blood lead levels above approximately 15 µg/dL may depress Vitamin D levels and affect red blood cell production. Even levels of 10 µg/dL or below may be associated with delays or impairments in neurodevelopment, delayed sexual maturation, and inhibition of enzymes involved in the synthesis of hemoglobin, a component of red blood cells. There is also some indication that lead exposure may heighten immune response and increase the risk of asthmatic reactions. In adults, high

levels of lead exposure (>30 to 40 µg/dL) may result in kidney effects, neurological and neurobehavioral effects, reduced fertility, altered thyroid hormones, and depressed hemoglobin.

Health effects of exposure to lead include several of the diseases and conditions of concern to the community. This is especially true of past exposures to paint sludge, but also to a lesser but still important degree, of exposure to lead in residential soils.

Antimony. No information is available regarding the chronic toxicity of antimony in humans. From experimental animal studies, target body systems and organs for long-term exposure to antimony are the blood (hematological disorders) and liver (mild hepatotoxicity) (ATSDR, 1990). In rats, long-term exposure to potassium antimony tartrate in the drinking water resulted in decreased lifespan. The LOAEL of 0.35 mg/kg/day from this study was used to calculate the chronic oral RfD of 0.0004 mg/kg/day. Mean exposure doses of antimony from ingestion of paint sludge (children, 0.19 mg/kg/day; adults 0.021 mg/kg/day) were estimated to exceed the chronic oral RfD, and were near the LOAEL. The same study showed an increase in serum cholesterol and a decrease in fasting glucose levels for rats receiving a lifetime exposure to potassium antimony tartrate (746 mg/kg/day) in drinking water. However, the biological significance of these findings in rats or humans is not certain. Since the estimated mean exposure doses from ingestion of antimony in paint sludge approached the LOAEL, it is possible that exposures to antimony in paint sludge caused an adverse health impact. However, it is not clear whether any of the health outcomes of concern to the community might be related to antimony exposure.

Arsenic. Ingestion of water from springs/seeps and brook may have resulted in long-term mean exposure to arsenic exposure doses of approximately 0.001 mg/kg/day in children and 0.0005 mg/kg in adults.

The effect of long-term oral exposure to inorganic arsenic compounds is associated with development of skin lesions. These lesions may appear at chronic exposure doses ranging from 0.002 to 0.02 mg/kg/day. Studies of chronic oral exposure to arsenic at levels ranging from 0.0004 to 0.01 mg/kg/day have not reported dermal effects. The mechanism(s) by which inorganic arsenic causes dermal effects is not well-understood.

Numerous studies of acute, high-dose exposures have reported nausea, vomiting, diarrhea, and abdominal pain, although specific dose levels associated with the onset of these symptoms have not been identified. Chronic oral exposures have been reported to result in irritant effects on gastrointestinal tissues at levels as low as 0.01 mg/kg/day. For both acute and chronic exposures, the gastrointestinal effects generally diminish or resolve with cessation of exposure.

Ingestion exposure to high levels of inorganic arsenic may result in the development of peripheral neuropathy. Reports of neurological effects at lower arsenic levels (0.004–0.006 mg/kg/day) have been inconsistent, with some human studies

reporting fatigue, headache, depression, dizziness, insomnia, nightmare, and numbness while other studies reported no neurological effects.

Relatively little information is available on effects due to direct dermal contact with inorganic arsenic compounds, but several studies indicate the chief effect is local irritation and dermatitis, with little risk of other adverse effects.

Mean arsenic exposure doses in the Ringwood Mines/Landfill area are lower than levels of arsenic exposure associated with non-cancer health effects. Therefore, it seems unlikely that exposure to arsenic is related to health outcomes of concern to the community.

Chromium. Chromium was detected in paint sludge at a mean concentration of 1,640 mg/kg, as total chromium. However, the proportion of chromium (VI), the more potent form, is not known. At soil concentrations exceeding 270 mg/kg, exposure to hexavalent chromium may cause allergic contact dermatitis. Therefore, it is possible that "skin rashes" reported to be of concern by the community may be related to past exposure to chromium in paint sludge.

Summary of Other Community Health Concerns in Relation to Site Contaminants. Respiratory diseases mentioned by the community include asthma and emphysema. While emphysema is unlikely to be related to exposure to site-related contaminants, there is some evidence that lead exposure may increase asthmatic episodes. However, there are numerous other, common triggers of asthma, and any linkage to potential site exposures would have to be determined on an individual basis. Diabetes is also unlikely to be related to site-related contaminants.

Community concerns also included reproductive and developmental effects, neurological disorders, cardiovascular disease, and anemia. Studies have shown that these health effects may be associated with exposure to lead at varying levels of chronic or acute exposure. However, all of these health outcomes may be caused by many other non-environmental (e.g., behavioral) and environmental risk factors.

Skin lesions and neurological disorders may also be associated with exposure to arsenic. However, the estimated levels of ingestion exposure to arsenic in the past do not appear to be sufficiently high to have resulted in these effects. It is possible that skin rashes reported to be of concern by the community may be related to past dermal exposure to chromium in paint sludge.

Conclusions

Disposal of paint sludge and other waste materials at the Ringwood Mines/Landfill site during the late 1960s and early 1970s resulted in the contamination of soil, sediment, and ground and surface water. Although remedial actions were taken in 1987/1988 and the site was deleted from the NPL in 1994, paint sludge and associated

soil contamination is still being found, including at on-site residential properties. At the present time, additional site characterization and remedial actions are being implemented to address the paint sludge and soil contamination.

In the past, there were completed exposure pathways to area residents via the ingestion of contaminated surface water and the incidental ingestion of contaminated paint sludge, soil, and sediment. Contaminants of concern identified for the site were Aroclors, bis(2-ethylhexyl)phthalate, antimony, arsenic, cadmium, chromium, copper and lead in paint sludge, benzene, benzo[a]pyrene, arsenic, lead and thallium in soil, benzo[a]pyrene, arsenic and thallium in sediment, benzene, 1,2-dichloropropane, arsenic, lead and mercury in surface water, and benzene, methylene chloride, bis(2-ethylhexyl)phthalate, pentachlorophenol, arsenic, cadmium, lead and thallium in groundwater. In addition, tetrachloroethene, antimony, beryllium, lead and silver detected in off-site potable wells and lead detected in residential soils exceeded their respective environmental guideline CVs.

Exposures associated with lead and antimony contamination detected in paint sludge, arsenic contamination detected in surface water, and lead contamination detected in soil and surface water were found to have the potential to cause non-cancer adverse health effects in children and adults. Potential health hazard due to additive or interactive effects of chemical mixtures may be greater than estimated by the endpoint-specific hazard index, particularly for neurological effects associated with co-exposure to lead and arsenic. Lifetime excess cancer risks associated with the ingestion of paint sludge, surface soil, and sediment were estimated to be very low when compared to background cancer risk. Based on the maximum and mean concentrations of arsenic detected in surface water, the calculated lifetime excess cancer risks were estimated to be approximately five and two excess cancer cases per 10,000 individuals, respectively.

Paint sludge is the likely source of lead and antimony at the site. Arsenic, however, may be naturally occurring in the area. Lead was detected in on-site residential soils at concentrations of health concern to children. Based on health risks posed by exposures to lead and antimony, the site posed a ***Public Health Hazard***[10] in the past. Since there may be on-going exposure from paint sludge and soil at levels of health concern, the site currently poses a ***Public Health Hazard***.

Ringwood Mines area residents and others may have been exposed to contaminated ambient air, biota, and off-site groundwater. These exposures constitute an ***Indeterminate Public Health Hazard*** as no data or insufficient data are available for evaluation.

Childhood blood lead data available from the NJDHSS Child and Adolescent Health Program were evaluated for the Ringwood Mines/Landfill area site. Data available since 1999 showed a higher proportion of children with elevated blood lead levels (>10 μg/dL) and a slightly higher average childhood blood lead level in the area closest to the Ringwood Mines/Landfill area in comparison to the rest of Ringwood

[10]A complete summary of ATSDR conclusion categories are provided in Appendix G.

Borough. Although there are multiple sources of lead in a child's environment (such as peeling lead-based paint in homes), lead containing paint sludge may have contributed to these differences in blood lead levels.

An analysis of cancer incidence in the Ringwood Mines/Landfill area was conducted. In the period 1979 - 2002, overall cancer incidence was not elevated. However, lung cancer incidence was statistically elevated in males in the area closest to the Ringwood Mines/Landfill site. It is not known whether past exposure pathways are related to this observation. Information on smoking history, the most important risk factor for lung cancer, was not available. Since lung cancer incidence was not elevated in females, there is little evidence that cancer incidence has been affected by Ringwood Mines/Landfill site contamination.

Other health concerns that residents believe are related to exposures to the Ringwood Mines/Landfill site contamination include respiratory diseases, reproductive and developmental effects, neurological disorders, heart disease, skin rashes and eye irritation, anemia, and diabetes. Many of the community's concerns are consistent with health effects of lead and arsenic exposures reported in the scientific literature; however, these health outcomes may also be caused by other environmental and non-environmental risk factors.

Recommendations

1. Efforts by the USEPA and NJDEP to fully characterize, delineate and remediate the paint sludge contamination of environmental media and residential properties should be completed as soon as feasible. Special consideration should be given to children's play areas and residential gardens.

2. The USEPA should delineate groundwater contamination and consider reinstituting an Environmental Monitoring Plan, particularly for off-site potable wells and other potential exposure points.

3. The USEPA or the NJDEP should characterize the potential contamination of local biota, particularly game consumed by Ringwood Mines/Landfill area residents.

4. The USEPA should further characterize the site to determine the background contribution to the concentration of arsenic and other COCs.

5. Because of the potential for exposure to metals from the paint sludge and contaminated soils, an exposure investigation of the population living on the Ringwood Mines/Landfill site should be conducted. This investigation should include biological testing of adults and children for current exposure to lead, antimony, and arsenic. Such testing should be undertaken at a time of year when the potential for exposure is highest, and it should be made clear that biological

testing for these metals would not be indicative of past exposure levels. The exposure investigation should also include concurrent testing of environmental media such as indoor dust and soils close to homes.

Public Health Action Plan

The purpose of a PHAP is to ensure that this public health assessment not only identifies public health hazards, but also provides a plan of action designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment. Included is a commitment on the part of ATSDR and NJDHSS to follow up on this plan to ensure that it is implemented. The public health actions to be implemented by the NJDHSS and the ATSDR are as follows:

Actions Undertaken

1. The NJDHSS and ATSDR have prepared this public health assessment in response to a petition from legal counsel representing the community.

2. The NJDHSS and ATSDR have participated in public availability sessions and meetings with local residents. ATSDR and NJDHSS met with the community to inform area residents of the preliminary results of the public health assessment and to obtain pertinent exposure-related information.

Actions Planned

1. Copies of this Public Health Assessment will be provided to concerned residents in the vicinity of the site via direct mail, the township library and the Internet.

2. Public meetings will be scheduled with area residents to discuss the findings of this report and to address any community concerns.

3. As remedial investigation data (from the residential properties) become available, the NJDHSS and ATSDR will evaluate the public health implications of contaminants detected and provide assistance to residents in reducing exposures to chemicals.

4. As a member of the New Jersey Environmental Justice Task Force, the NJDHSS will work with NJDEP and other state agencies to develop an appropriate Action Plan in cooperation with the community.

5. The NJDHSS and ATSDR will begin planning for implementation of an Exposure Investigation to determine the extent of exposure to heavy metals from environmental media contaminated by paint sludge. Plans should be developed in conjunction with community members, and may follow a phased approach as outlined in the January 2005 Environmental Health Initiative (RNAA 2005). As a

first step, the NJDHSS and ATSDR have begun outlining available biological monitoring tests, meanings and limitations of such tests, and laboratory capabilities for testing, and will provide this information to the community. The NJDHSS and ATSDR will also work with the USEPA and NJDEP to coordinate potential environmental testing that would be conducted in association with biological monitoring.

Preparers of Report:

Tariq Ahmed, PhD, PE, DEE
Research Scientist
New Jersey Department of Health and Senior Services

Julie R. Petix, MPH, CPM, HO
Project Manager
New Jersey Department of Health and Senior Services

Michael Berry, MPH
Research Scientist
New Jersey Department of Health and Senior Services

Patricia Haltmeier
Public Health Representative
New Jersey Department of Health and Senior Services

ATSDR Regional Representatives:

Arthur Block
Senior Regional Representative

Leah T. Escobar, R.S.
Associate Regional Representative

ATSDR Technical Project Officer:

Gregory V. Ulirsch, MS, PhD
Technical Project Officer
Superfund Site Assessment Branch
Division of Health Assessment and Consultation

Any comments or questions concerning this document should be directed to:

Hazardous Site Health Evaluation Program
Consumer and Environmental Health Services
New Jersey Department of Health and Senior Services
3635 Quakerbridge Road
P.O. Box 369
Trenton, New Jersey 08625-0369
(609) 584-5367

References

[ATSDR] Agency for Toxic Substances and Disease Registry 1989. Public Health Assessment: Ringwood Mines/Landfill National Priorities List (NPL) Site, Ringwood, Passaic County, New Jersey, 1989.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1992. Toxicological profile for Antimony. Atlanta: US Department of Health and Human Services.

[ATSDR] Agency for Toxic Substances and Disease Registry 1994. Site Review and Update: Ringwood Mines/Landfill, Ringwood, Passaic County, New Jersey, 1994.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1999a. Toxicological profile for Lead. Atlanta: US Department of Health and Human Services.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1999b. Toxicological profile for Polycyclic Aromatic Hydrocarbons (PAHs). Atlanta: US Department of Health and Human Services.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1999c. Toxicological profile for Mercury. Atlanta: US Department of Health and Human Services.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for Arsenic. Atlanta: US Department of Health and Human Services.

[ATSDR] Agency for Toxic Substances and Disease Registry 2005. Guidance Manual for the Assessment of Joint Toxic Action of Chemical Mixtures. US Department of Health and Human Services, May, 2004.

[ATSDR] Agency for Toxic Substances and Disease Registry 2005. Public Health Assessment Guidance Manual (Update), 2005.

American Cancer Society: What are the Risk Factors for Lung Cancer? Located on-line at www.cancer.org, 2004.

[ARCADIS] ARCADIS Geraghty & Miller, Inc. 1999. November 29, 1999 letter to J. Amber, Ford Motor Company from Roberta McIntyre.

[ARCADIS] ARCADIS Geraghty & Miller, Inc. 2001. Final Summary Report for Post-Environmental Monitoring Program Activities, Ringwood Mines/Landfill Site, Ringwood, New Jersey. December 2001.

[ARCADIS] ARCADIS Geraghty & Miller, Inc. 2004. Paint Sludge and Drum Remnant Removal Workplan, Ringwood Mines/Landfill site, Ringwood, New Jersey, 2004.

[ARCADIS] ARCADIS Geraghty & Miller, Inc. 2006. February 6, 2006 letter to T. Ahmed, New Jersey Department of Health and Senior Services from E. Carmen, 2006.

Bagdon RE and Hazen RE. 1991. Skin Permeation and Cutaneous Hypersensitivity as a Basis for Making Risk Assessment of Chromium as Soil Contaminant. Environ. Health Perspect. 92:111-119.

Bergen Record 2005. Accessed December 2005, at: http://www.bergenrecord.com/

Chapin Engineering 2005. Report on Cleanup of Paint Sludge at three Residential Properties in Ringwood, New Jersey. December, 2005.

[CDC] Centers for Disease Control. 1991. Preventing lead poisoning in young children. U.S. Department of Health and Human Services, October.

[CDC] Centers for Disease Control. 2005. Third National Report on Human Exposures to Environmental Chemicals. Department of Health and Human Services, July 2005.

Environ Corporation 1988. An assessment of the Human Health and Environmental Risks associated with the Ringwood Mines/Landfill site, Ringwood Borough, Passaic County, New Jersey, August 9, 1988.

Geraghty & Miller, Inc. 1998. Revised Final report: Environmental monitoring Program, Ringwood Mines/Landfill Site, Ringwood, New Jersey, June 1998.

Geraghty & Miller, Inc. 1996. January 04, 1996 letter to Mr. Jerome Amber, Ford Motor Company from Christopher J. Motta.

Kupferstein L. 1973. WPC Students Studying Ringwood Mine Area. Morning News. March 8, 1973.

Latham & Watkins 2005. August 29, 2005 letter to J. Fagliano, New Jersey Department of Health and Senior Services from David Hayes, 2005.

Muszynski, WJ. 1993. Superfund Site Close-out Report, Ringwood Mines/Landfill Site, Passaic County, New Jersey, United States Environmental Protection Agency, 1993.

National Cancer Institute: Cancer Rates and Risks. NIH Publication No. 96-691, 1996.

[NJDEP] New Jersey Department of Environmental Protection. 1998. Summary of the Basis and Background of the Soil Cleanup Criteria for Trivalent and Hexavalent Chromium. Site Remediation Program. September 18, 1998.

[NJDEP] New Jersey Department of Environmental Protection. 2004. Memorandum from Swati Toppin to Linda Cullen, 2004.

[NJDEP] New Jersey Department of Environmental Protection 2005. May 30, 2005 Memorandum to Kathleen Kunze from Linda M. Appel, 2005.

[NJDEP] New Jersey Department of Environmental Protection. 2005. Memorandum from Linda Appel to Kathleen Kunze, November 2005.

[NJDWSC] North Jersey District Water Supply Commission 2005. Accessed December 2005 at: http://www.njdwsc.com/njdw/about.htm

[NJGS] New Jersey Geological Survey. 2005. Arsenic in New Jersey Ground Water. Accessed on June 13, 2005, at: http://www.state.nj.us/dep/njgs/enviroed/infocirc/arsenic.pdf.

[NTP] National Toxicology Program 2006. 11th Report on Carcinogens. Accessed April 18, 2006, at: http://ntp.niehs.nih.gov/

Pustay, PE, Shea, TK. 1992. Abandoned Iron Mines of Passaic and Bergen Counties, Division of Public Safety and Occupational Safety and Health, Department of Labor, State of New Jersey, Trenton, New Jersey, 1992.

[RNAA] Ringwood Neighborhood Action Association 2005. Environmental Health Intervention Program to Address the Health Implications of Contamination of the Ringwood Mines/Landfill site, Ringwood, New Jersey. January 13, 2005

[SEER] Surveillance Epidemiology and End Results 2005. SEER Cancer Statistics Review, 1975 - 2002. Accessed February 3, 2006 at: http://seer.cancer.gov/csr/1975_2002/.

Stern AH, Bagdon, RE, Hazen RE, Marzulli, FN. 1993. Risk Assessment of Allergic Dermititis Potential of Environmental Exposures to Hexavalent Chromium. J. Environ. Toxicol. Health. 40:613-641, 1991.

[USEPA] United States Environmental Protection Agency 1986. Air Quality Criteria for Lead. Environmental Criteria and Assessment Office, Office of Research and Development, Research Triangle Park, N.C. EPA 600/8-83-028 a-f, June 1986.

[USEPA] United States Environmental Protection Agency 1989. Evaluation of the Potential Carcinogenicity of Lead and Lead Compounds. Office of Health and Environmental Assessment. EPA/600/8-89/045A.

[USEPA] United States Environmental Protection Agency 1991. Standard Default Factors, OSWER Directive 9285.6-03, Office of Emergency and Remedial Response, Washington, D.C.

[USEPA] United States Environmental Protection Agency 1994a. EPA Groundwater Issue: Natural Attenuation of Hexavalent Chromium in Groundwater and Soils. EPA /540/5-94/505, October 1994.

[USEPA] United States Environmental Protection Agency 1994b. Guidance Manual for the IEUBK Model for Lead in Children. Office of Solid Waste and Emergency Response. OSWER Directive #9285.7-15-1. February 1994.

[USEPA] United States Environmental Protection Agency 1994c. Memorandum: OSWER Directive: Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. OSWER Directive #9355.4-12. August 1994.

[USEPA] U.S. Environmental Protection Agency 1997. Exposure Factor Handbook (Volume I, II and III), EPA/600/P-95/002Fa, b and c, August 1997.

[USEPA] United States Environmental Protection Agency 1998. June 11, 1998 letter to Mr. Jan Barry from Monica Mertzke.

[USEPA] United States Environmental Protection Agency 2002. User's Guide for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Windows® Version – 32 Bit Version. Office of Solid Waste and Emergency Response. OSWER Directive #9285.7-42. May 2002.

[USEPA] United States Environmental Protection Agency 2003. Assessing Intermittent or Variable Exposures at Lead Sites. Office of Solid Waste and Emergency Response. OSWER Directive #9285.7-76. November 2003.

[USEPA] United States Environmental Protection Agency 2003. Recommendations of the Technical Review Workgroup for Lead for an Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil, EPA-540-R-03-001, January 2003.

[USEPA] United States Environmental Protection Agency 2004. Sampling Report for the Ringwood Mines/Landfill Site in Ringwood Borough, Passaic County, New Jersey, May 10, 2004.

[USEPA] United States Environmental Protection Agency 2005. Integrated Risk Information System. Accessed December 2005, at: http://www.epa.gov/iris/.

West Milford Argus Today 1975. Health Board Urges Mine Area State Aid. April 9, 1975.

Woodward-Clyde Consultants 1988. Final Remedial Investigation Report, Ringwood Mines/Landfill Site, Ringwood, New Jersey, September 1988.

Woodward-Clyde Consultants 1990. Geological Assessment Report: Ringwood Mines/Landfill Site, New Jersey, March 1990.

YE^{2}ARS 1983. Remedial Action Master Plan, Ringwood Mines/Landfill site, Ringwood, New Jersey, 1983.

Zalaskus G. 2000. Intradepartmental Memorandum to R. Luzecky. April 14, 2000.

Table 1: Analytical Data (June and September, 1990) Summary of Drum Contents (as obtained from Disposal Profile Sheets)

Waste Description	Unit	Waste Oil (oil & sludge)	Water & oil	Water & Antifreeze	Speedy Dry, Rags, Cloth, Gloves	Oil, Water, Antifreeze	Waste Oil
pH	---[1]	8.0	6.0	8.0	7.0	6.0	---
Chlorine, Total	% weight	0.9	6.6	0.2	< 0.1	0.2	---
Bromine, Total		< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	---
Sulfur, Total		1	< 0.1	< 0.1	0.1	< 0.1	---
Fluorine, Total		< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	---
Polyethylene glycol (Brake fluid)		ND[2]	ND	7.2	ND	10	*[3]
Hexanes (C6)		ND	ND	4.9	ND	0.3	*
Hexylene glycol		ND	ND	1	ND	ND	*
Toluene		ND	ND	0.2	ND	ND	5
1,1,1-Trichloroethane		ND	12.8	ND	ND	ND	*
Trace VOCs (< 1% each)[4]		11.9	0.2	ND	ND	ND	*
Aroclor 1254	ppm	55	25	7	10	8	139
Aroclor 1262		7	3	ND	ND	ND	---
Arsenic		---	---	---	< 5	---	---
Barium		---	---	---	400	---	---

Table 1: (Cont'd.)

Waste Description	Unit	Waste Oil (oil & sludge)	Water & oil	Water & Antifreeze	Speedy Dry, Rags, Cloth, Gloves	Oil, Water, Antifreeze	Waste Oil
Beryllium	ppm	---	---	---	2	---	---
Cadmium		---	---	---	12	---	---
Chromium		---	---	---	130	---	---
Iron		---	---	---	55,460	---	---
Lead		---	---	---	939	---	---
Mercury		---	---	---	< 5	---	---
Phosphorus		---	---	---	915	---	---
Selenium		---	---	---	< 5	---	---
Silver		---	---	---	< 5	---	---
Titanium		---	---	---	2203	---	---
Zinc		---	---	---	172	---	---

[1]Not analyzed; [2]Not detected; [3]Analytical data of VOC scan was not provided on the disposal profile sheets; [4]No single volatile organic compound (VOC) represented greater than or equal to 1% by weight of the total VOC portion of the sample

Table 2: Results of Soil samples collected (July 1984) from Test Pits at the Ringwood Mines/Landfill site

Contaminant	TP-3 (mg/kg)	TP-12 (mg/kg)	Environmental Guideline CV (mg/kg)	Selected for Further Evaluation
Volatile Organic Compounds (VOCs)				
Benzene	34	NS[1]	10 (CREG)	**Yes**
Ethylbenzene	140	NS	5,000 (RMEG)	No
Methylene Chloride	NA[2]	NS	90 (CREG)	No
Tetrachloroethylene	26	NS	500 (RMEG)	No
Toluene	510	NS	1,000 (EMEG)	No
Metals[3]				
Arsenic	NA	NA	0.5 (CREG)	No
Barium	0.7	0.2	4,000 (RMEG)	No
Cadmium	NA	NA	10 (EMEG)	No
Lead	0.56	NA	400 (RDCSCC)	No

[1]Not submitted; [2]Not available, [3]Metals were reported using EP Toxicity data, Not available

Table 3: Analytical Results of Paint Sludge samples[1] collected (March/April, 1987) from Ringwood Mines/Landfill site

Contaminant	Concentration (mg/kg)		Environmental Guideline CV (mg/kg)	COC[2]
	Range	Mean		
Volatile Organic Compounds (VOCs)				
Acetone	ND[3] - 56	24.5	100,000 (EMEG[4])	No
Benzene	ND - 1.6	1.1	10 (CREG[5])	No
Ethylbenzene	0.25 - 810	340	5,000 (RMEG[6])	No
4-Methyl-2-pentanone	ND - 39	18.7	1,000 (RDCSCC[7])	No
Methylene Chloride	ND - 50	15.6	90 (CREG)	No
Tetrachloroethene	ND - 95	57.4	500 (RMEG)	No
Toluene	0.17 - 610	276	1,000 (EMEG)	No
Trichloroethene	ND - 140	57.7	400 (EMEG)	No
m-Xylene	ND - 8,200	1,617	10,000 (EMEG)	No
o,p-Xylene	ND - 8,300	1,583	10,000 (EMEG)	No
Semi Volatile Organic Compounds (SVOCs)				
Aroclor 1248	0.077 – 1.38	0.5	0.49 (RDCSCC)	**Yes**
Aroclor 1254	0.097 – 2.2	0.9	0.49 (RDCSCC)	**Yes**
2-Chlorophenol	ND – 2	2	300 (RMEG)	No
2-Methylnaphthalene	6.2 – 140	66.8	3,000 (EMEG)	No
4-Nitrophenol	ND – 2.8	2.8	630 (RBC[8])	No
Benzyl Alcohol	ND – 21	8.5	10,000 (RDCSCC)	No
Bis(2-ethylhexyl)phthalate	9.2 – 380	120	49 (RDCSCC)	**Yes**
Butylbenzyl Phthalate	ND – 4.2	4.1	10,000 (RMEG)	No
Di-n-butyl Phthalate	ND – 6.8	3.8	5,000 (RMEG)	No
Fluoranthene	ND – 2.4	2.4	2,000 (RMEG)	No
Naphthalene	43 – 350	158	1,000 (RMEG)	No
N-Nitrosodiphenylamine	ND – 5.2	5.2	100 (CREG)	No
Phenanthrene	ND – 4.6	3.7	NA[9]	No
Phenol	ND – 7.6	4.4	20,000 (RMEG)	No
Pyrene	ND – 3	2.5	2,000 (RMEG)	No
Metals				
Antimony	160 – 460,000	47,137	20 (RMEG)	**Yes**
Arsenic	0.22 – 16	4.3	0.5 (CREG)	**Yes**
Cadmium	1.8 – 32	10.5	10 (EMEG)	**Yes**
Chromium	436 – 2,400	1,639	200 (RMEG)	**Yes**
Copper	110 – 2,100	565	1,000 (EMEG)	**Yes**
Lead	5,900 – 310,000	64,880	400 (RDCSCC)	**Yes**
Mercury	ND – 3.17	1.2	14 (RDCSCC)	No

Table 3: (Cont'd.)

Contaminant	Concentration (mg/kg)		Environmental Guideline CV (mg/kg)	COC[2]
	Range	Mean		
Metals				
Nickel	25 – 82	59	1,000 (RMEG)	No
Selenium	ND – 4.5	1.9	300 (EMEG)	No
Silver	ND – 16.1	4.6	300 (RMEG)	No
Zinc	620 – 3,500	2,437	20,000 (EMEG)	No
Other				
Cyanide	ND - 5.05	3.5	1,000 (RMEG)	No
Phenol	1.06 - 10	4.3	20,000 (RMEG)	No

[1]No. of samples taken =10; [2]Contaminant of Concern; [3]Not detected; [4]ATSDR Environmental Media Evaluation Guide; [5]ATSDR Cancer Risk Evaluation Guide; [6]ATSDR Reference Media Evaluation Guide; [7]NJDEP Residential Direct Contact Soil Cleanup Criteria; [8]EPA Region 3 Risk-Based Concentration; [9]Not available

Table 4: Post-Remedial Soil Sampling (March 1988) Results from Paint Sludge Disposal Areas

Contaminant	No. of Detection[1]	Concentration (mg/kg)		Environmental Guideline CV (mg/kg)	COC[2]
		Range	Mean		
Volatile Organic Compounds (VOCs)					
trans 1,2-Dichloroethene	1	0.003	0.003	1,000 (RMEG[3])	No
Tetrachloroethene	3	0.001- 0.002	0.002	500 (RMEG)	No
4-methyl-2-pentanone	3	0.002 - 0.033	0.012	1,000 (RDCSCC[4])	No
m-xylene	2	0.002 - 0.004	0.003	10,000 (EMEG[5])	No
o,p xylene	1	0.004	0.004	10,000 (EMEG)	No
Semi Volatile Organic Compounds (SVOCs)					
Bis[2-ethylhexyl]phthalate	9	0.042 - 5.6	0.75	49 (RDCSCC)	No
Di-n-butyl phthalate	1	0.11	0.11	5,000 (RMEG)	No
Phenanthrene	3	0.077 - 0.2	0.132	NA[6]	
Anthracene	1	0.035	0.035	20,000 (RMEG)	No
Fluoranthene	4	0.046 - 0.36	0.165	2,000 (RMEG)	No
Pyrene	4	0.046 - 0.37	0.174	2,000 (RMEG)	No
Chrysene	3	0.042 - 0.18	0.114	9 (RDCSCC)	No
Indeno[1,2,3-cd]pyrene	2	0.049 - 0.066	0.058	0.9 (RDCSCC)	No
Benzo[g,h,i]perylene	2	0.056 - 0.077	0.067	NA	No
Benzo[a]anthracene	3	0.046 - 0.17	0.115	0.9 (RDCSCC)	No
Benzo[b]flouranthene	2	0.2 - 0.32	0.26	0.9 (RDCSCC)	No
Benzo[k]fluoranthene	2	0.2 - 0.32	0.26	0.9 (RDCSCC)	No
Benzo[a]pyrene	2	0.099 - 0.18	0.14	0.1 (CREG[7])	**Yes**
Naphthalene	1	2.1	2.1	1,000 (RMEG)	No
2-methylnapthalene	1	1.1	1.1	200 (RMEG)	No
Metals					
Antimony	3	0.6 - 6.3	2.54	20 (RMEG)	No
Arsenic	12	0.95 - 9.6	2.03	0.5 (CREG)	**Yes**
Barium	12	24 - 400	75.9	4,000 (RMEG)	No
Chromium	12	9.1 - 50	23.42	200 (RMEG)	No
Copper	12	0.319 - 72	19.36	1,000 (EMEG)	No
Lead	12	3 - 1,300	129.6	400 (RDCSCC)	**Yes**
Nickel	12	0.26 - 16	8.8	1,000 (RMEG)	No
Silver	1	0.7	0.7	300 (RMEG)	No
Thallium	1	19	19	2 (RDCSCC)	**Yes**
Zinc	12	29 - 140	48.66	20,000 (EMEG)	No

Table 4: (Cont'd.)

Contaminant	No. of Detection[1]	Concentration (mg/kg)		Environmental Guideline CV (mg/kg)	COC[2]
		Range	Mean		
Other					
Cyanide	2	0.0008 - 0.0009	0.0008	1,000 (RMEG)	No

[1]No. of samples taken =12; [2]Contaminant of Concern; [3]ATSDR Reference Media Evaluation Guide; [4]NJDEP Residential Direct Contact Soil Cleanup Criteria; [5]ATSDR Environmental Media Evaluation Guide; [6]Not Available

Table 5: Concentration of Contaminants detected in the Stream Sediment collected (July 1984 and March 1988) from the Ringwood Mines/Landfill site

Contaminant	No. of Detection[1]	Concentration (mg/kg)		Environmental Guideline CV (mg/kg)	COC[2]
		Range	Mean		
Volatile Organic Compounds (VOCs)					
4-methylphenol	1	0.16	0.16	2,800 (RDCSCC[3])	No
4-nitrophenol	1	0.9	0.9	630 (RBC[4])	No
Semi Volatile Organic Compounds (SVOCs)					
Benzo[a]anthracene	2	0.062 – 0.35	0.206	0.9 (RDCSCC)	No
Benzo[k]fluoranthene	1	0.42	0.42	0.9 (RDCSCC)	No
Benzo[a]pyrene	1	0.61	0.61	0.1 (CREG[5])	**Yes**
Bis[2-ethylhexyl]phthalate	2	0.083 – 0.12	0.1015	50 (CREG)	No
Chrysene	4	0.079 – 0.41	0.2	9 (RDCSCC)	No
Di-n-octylphthalate	1	0.22	0.22	20,000 (EMEG[6])	No
Fluoranthene	3	0.11 – 0.51	0.24	20,000 (EMEG)	No
Phenanthrene	1	0.17	0.17	NA[8]	No
Pyrene	3	0.12 – 0.47	0.26	2,000 (RMEG)	No
Silvex	1	0.0075	0.0075	400 (RMEG[7])	No
Metals					
Antimony	1	2.2	2.2	20 (RMEG)	No
Arsenic	14	0.79 – 31.4	9.13	0.5 (CREG)	**Yes**
Barium	16	21 – 410	83.5	4,000 (RMEG)	No
Beryllium	9	0.46 - 2	0.92	100 (EMEG)	No
Cadmium	8	0.91 – 3.4	2.26	10 (EMEG)	No
Chromium	16	4.1 – 24	16.4	200 (RMEG)	No
Copper	16	0.163 – 39	12.37	1,000 (EMEG)	No
Iron	10	14,000 – 86,000	34,100	2,300 (RBC)	No
Lead	14	2.8 – 20	13.44	400 (RDCSCC)	No
Manganese	10	340 – 5,200	1,513	3,000 (RMEG)	No
Mercury	7	0.015 - 0.037	0.023	14 (RDCSCC)	No
Nickel	16	0.135 – 26	9.96	1,000 (RMEG)	No
Selenium	2	0.07 – 4.5	2.28	300 (EMEG)	No
Silver	9	0.5 – 0.95	0.72	300 (RMEG)	No
Thallium	10	5 – 14	9.45	2 (RDCSCC)	**Yes**
Zinc	16	17 – 130	61.25	20,000 (EMEG)	No

Table 5: (Cont'd.)

Contaminant	No. of Detection[1]	Concentration (mg/kg)		Environmental Guideline CV (mg/kg)	COC[2]
		Range	Mean		
Other					
Cyanide	3	0.29 – 0.42	0.34	1,000 RMEG)	No
Phenolics	1	0.13	0.13	20,000 (RMEG)	No

[1]No. of samples taken = 16; [2]Cotaminant of Concern; [3]NJDEP Residential Direct Contact Soil Cleanup Criteria; [4]EPA Region 3 Risk-Based Concentration; [5]ATSDR Cancer Risk Evaluation Guide; [6]ATSDR Environmental Media Evaluation Guide; [7]ATSDR Reference Media Evaluation Guide; [8]Not Available

Table 6: Results of Brook Samples collected (July 1984, April 1985 and March 1988) from the Ringwood Mines/Landfill site

Contaminant	No. of Detection[1]	Concentration (μg/L)		Environmental Guideline CV (μg/L)	COC[2]
		Range	Mean		
Semi Volatile Organic Compounds (SVOCs)					
Di-n-octyl phthalate	2	14 - 18	16	730 (RBC[3])	No
Metals					
Arsenic	1	40	40	10 (NJMCL[4])	**Yes**
Barium	1	150	150	2,000 (NJMCL)	No
Iron	20	64 – 4,200	957	11,000 (RBC)	No
Manganese	14	20 – 1,700	395	730 (RBC)	No
Zinc	14	10 - 520	133	3,000 (EMEG[5])	No
Other					
Nitrate (as N)	9	50 - 450	144	10,000	No
Phenolics	2	6 - 121	63.5	5,000 (RUL)	No

[1]No. of samples taken = 20; [2]Contaminant of Concern; [3]EPA Region 3 Risk-Based Concentration; [4]New Jersey Maximum Contaminant Level; [5]ATSDR Environmental Media Evaluation Guide

Table 7: Results of Spring/Seep samples collected (July 1984 and March 1988) from the Ringwood Mines/Landfill site

Contaminant	No. of Detection[1]	Concentration (μg/L)		Environmental Guideline CV (μg/L)	COC[2]
		Range	Mean		
Semi Volatile Organic Compounds (SVOCs)					
Benzene	2	1 – 2	1.5	1 (NJMCL[3])	**Yes**
Chlorobenzene	4	2 – 14	6.5	50 (NJMCL)	No
Chloroethane	1	2	2	3.6 (RBC[4])	No
1,2-Dichloropropane	1	12	12	5 (NJMCL)	**Yes**
Silvex	3	0.13 – 0.68	0.4	50 (NJMCL)	No
Metals					
Arsenic	6	10 – 21	12.66	5 (NJMCL)	**Yes**
Barium	10	100 – 400	210	2,000 (NJMCL)	No
Copper	1	330	330	1,300 (AL[5])	No
Iron	10	260 – 150,000	46,286	11,000 (RBC)	**Yes**
Lead	2	90 – 120	105	15 (AL)	**Yes**
Manganese	10	60 – 10,000	4,396	730 (RBC)	**Yes**
Mercury	6	0. 4 – 8.7	2.43	2 (NJMCL)	**Yes**
Zinc	8	10 – 40	23.75	5,000 (NJMCL)	No
Other					
Fluoride	7	180 – 1,230	353	4,000 (NJMCL)	No
Nitrate (as N)	8	60 – 380	164	10,000 (NJMCL)	No
Phenolics	3	11 – 14	12	3,000 (RMEG[6])	No
Sulfate	10	6.4 – 18,000	12,005	250,000 (RUL)	No

[1]No. of samples taken = 20; [2]Contaminant of Concern; [3]Maximum Contaminant Level; [4]EPA Region 3 Risk-Based Concentration; [5]Action Level; [6]ATSDR Reference Media Evaluation Guide

Table 8: Results of Groundwater samples collected (August and September 1984, June 1986 and March 1988) from the Ringwood Mines/Landfill site

Contaminant	No. of Detection[1]	Concentration (μg/L)		Environmental Guideline CV (μg/L)	COC[2]
		Range	Mean		
Volatile Organic Compounds (VOCs)					
Benzene	1	9.1	9.1	1 (NJMCL[3])	**Yes**
Methylene Chloride	1	19	19	3 (NJMCL)	**Yes**
1,1-dichloroethane	3	4 – 13	9.7	50 (NJMCL)	No
Chloroethane	2	2 – 3	2.5	3.6 (RBC[4])	No
Toluene	1	19	19	1,000 (NJMCL)	No
o,p-xylene	1	4	4	1,000 (NJMCL)	No
Ethylbenzene	1	2	2	700 (NJMCL)	No
Semi Volatile Organic Compounds (SVOCs)					
Benzoic Acid	3	2 - 11	6.3	40,000 (RMEG[5])	No
Bis(2-ethylhexyl) phthalate	3	5 - 5	5	4.8 (RBC)	**Yes**
Di-n-octyl phthalate	1	11	11	730 (RBC)	No
Pentachlorophenol	1	4	4	1 (NJMCL)	**Yes**
4-nitrophenol	1	3	3	60 (LTHA[6])	No
Metals					
Arsenic	8	2.2 - 56	16.4	5 (NJMCL)	**Yes**
Barium	8	100 - 700	247	2,000 (NJMCL)	No
Cadmium	3	20 – 93,000	41,000	5 (NJMCL)	**Yes**
Chromium	1	58	58	100 (NJMCL)	No
Copper	6	23 - 410	130	1,300 (AL[7])	No
Iron	14	160 – 33,000	9,630	11,000 (RBC)	**Yes**
Lead	4	50 - 85	65	15 (AL)	**Yes**
Manganese	13	50 – 5,400	1,316	730 (RBC)	**Yes**
Mercury	1	0.8	0.8	2 (NJMCL)	No
Nickel	1	50	50	200 (RMEG)	No
Thallium	1	100	100	2 (NJMCL)	**Yes**
Zinc	20	10 - 600	67.2	5,000 (RUL)	No

Table 8: (Cont'd.)

Contaminant	No. of Detection[1]	Concentration (μg/L)		Environmental Guideline CV (μg/L)	COC[2]
		Range	Mean		
Other					
Fluoride	11	120 – 300	173.6	2,000 (RUL)	No
Nitrate (as N)	8	50 – 1,900	546.2	10,000 (NJMCL)	No
Phenolics	3	6 – 20	11.7	3,000 (RMEG)	No
Sulfate	13	7,700 – 30,000	15,238	250,000 (RUL)	No

[1]No. of samples taken = 45; [2]Contaminant of Concern; [3]New Jersey Maximum Contaminant Level; [4]EPA Region 3 Risk-Based Concentration; [5]ATSDR Reference Media Evaluation Guide for child; [6]ATSDR Lifetime Health Advisory for Drinking water; [7]Action Level

Table 9: Results of off-site Potable Well samples collected during Environmental Monitoring Program (1989 – 95)

Contaminant	No. of Detection[1]	Concentration (µg/L)		Environmental Guideline CV (µg/L)	COC[2]
		Range	Mean		
Volatile Organic Compounds (VOCs)					
Acetone	7	20.4 - 54.5	36.54	4,000 (RMEG[3])	No
2-Butanone (MEK)	2	9 - 41	25	6,000 (RMEG)	No
Carbon Disulfide	1	1	1	1,000 (RMEG)	No
Di-n-octyl phthalate	2	8.27 - 14.1	11.2	730 (RBC)	No
Tetrachloroethene	4	1 - 2.55	1.64	1 (NJMCL)	**Yes**
1,1,1-Trichloroethane	3	1 - 2.34	1.45	30 (NJMCL)	No
Metals					
Antimony	8	1.6 - 14.9	5.45	6 (NJMCL)	**Yes**
Arsenic	3	2.3 - 8.4	5.83	5 (NJMCL)	No
Barium	95	1.2 - 59.6	11.1	2,000 (NJMCL)	No
Beryllium	30	0.06 - 26	1.6	4 (NJMCL)	**Yes**
Cadmium	2	0.52 - 2.1	1.31	5 (NJMCL)	No
Chromium	17	0.41 - 11.3	1.88	100 (NJMCL)	No
Copper	146	2 – 1,200	80.14	1,300 (AL[6])	No
Iron	120	7.4 – 38,300	470	11,000 (RBC)	**Yes**
Lead	55	0.5 - 127	12.4	15 (AL)	**Yes**
Manganese	76	0.4 - 684	13	730 (RBC)	No
Mercury	2	0.5 - 0.62	0.56	2 (NJMCL)	No
Nickel	20	0.61 - 20	4	200 (RMEG)	No
Selenium	3	3.79 - 6.65	5.5	50 (NJMCL)	No

Table 9: (Cont'd.)

Contaminant	No. of Detection[1]	Concentration (µg/L)		Environmental Guideline CV (µg/L)	COC[2]
		Range	Mean		
Metals					
Silver	7	0.15 – 5,850	2000	100 (RUL)	**Yes**
Tin	12	10.7 - 39.5	20.58	22,000 (RBC)	No
Vanadium	12	0.93 - 12	2.83	37 (RBC)	No
Zinc	137	3.9 – 1,500	188	5,000 (RUL)	No
Other					
Cyanide	1	29	29	200 (RMEG)	No

[1]No. of potable wells tested = 9; chloroform, methylene chloride and bis(2-ethylhexyl) phthalate were detected but were qualified as invalid due to their presence in the blanks; [2]Contaminant of Concern; [3]ATSDR Reference Media Evaluation Guide; [4]New Jersey Maximum Contaminant Level; [5]EPA Region 3 Risk-Based Concentration; [6]Action Level (New Jersey); [7]Recommended Upper Limit (New Jersey Secondary Standards)

Table 10: Results of Groundwater samples collected (1989 - 1995) from the Ringwood Mines/Landfill site

Contaminant	No. of Detection[1]	Concentration (µg/L)		Environmental Guideline CV (µg/L)	COC[2]
		Range	Mean		
Volatile Organic Compounds (VOCs)					
Benzene	7	1 - 9.1	3.73	1 (NJMCL[4])	**Yes**
2-Butanone (MEK)	3	4 - 37	20	6,000 (RMEG)	No
Carbon Disulfide	1	14.2	14.2	1,000 (RMEG)	No
Chloroethane	3	2 - 4	3	3.6 (RBC[5])	**Yes**
1,1-Dichloroethane	16	4 - 23	9.2	50 (NJMCL)	No
1,2-Dichloroethene	1	2	2	2 (NJMCL)	No
Di-n-octylphthalate	1	11	11	4,000 (EMEG[6])	No
Ethylbenzene	4	2 - 4	2.7	700 (NJMCL)	No
o, xylene	4	1 - 7	2.75	1,000 (NJMCL)	No
m,p xylene	4	1 - 9	4.25	1,000 (NJMCL)	No
4-Nitrophenol	1	3	3	60 (LTHA[7])	No
1,1,2,2-Tetrachloroethane	1	4	4	1 (NJMCL)	**Yes**
Toluene	5	2 - 160	37.6	1,000 (NJMCL)	No
1,1,1-Trichloroethane	2	2 - 4	3	30 (NJMCL)	No
Semi Volatile Organic Compounds (VOCs)					
Benzoic Acid	2	6 - 11	8.5	40,000 (RMEG)	No
Metals					
Aluminum	85	113 – 50,300	6,348	20,000 (RMEG)	**Yes**
Antimony	10	0.59 - 24.9	11.7	6 (NJMCL)	**Yes**
Arsenic	62	1.1 - 65	16.26	5 (NJMCL)	**Yes**
Barium	75	16 – 1,700	223	2,000 (NJMCL)	No
Beryllium	14	0.04 - 6.1	1.11	4 (NJMCL)	**Yes**
Cadmium	9	1.4 - 20	11.4	5 (NJMCL)	**Yes**
Chromium	54	2.1 - 272	26.8	100 (NJMCL)	**Yes**
Cobalt	26	0.18 - 144	30.45	100 (EMEG)	**Yes**
Copper	68	2.5 - 491	46.2	1,300 (AL[8])	No
Iron	108	160 – 376,000	30,930	11,000 (RBC)	**Yes**

Table 10: (Cont'd.)

Contaminant	No. of Detection[1]	Concentration (µg/L)		Environmental Guideline CV (µg/L)	COC[2]
		Range	Mean		
Metals					
Lead	53	0.61 - 117	23.13	15 (AL)	**Yes**
Manganese	102	12 – 20,200	2,328	730 (RBC)	**Yes**
Mercury	3	0.37 - 2.51	1.23	2 (NJMCL)	**Yes**
Nickel	40	0.96 - 320	35.15	200 (RMEG)	**Yes**
Selenium	81	3.1 - 4.45	3.78	50 (NJMCL)	No
Silver	3	5 - 10	8.33	50 (RMEG)	No
Thallium	6	0.19 - 100	21	2 (NJMCL)	**Yes**
Tin	12	10.7 – 45.6	41.16	22,000 (RBC)	No
Vanadium	33	1.3 - 274	58	37 (RBC)	**Yes**
Zinc	99	4.8 - 785	69	3,000 (EMEG)	No
Cyanide	3	11.4 - 25	18.8	200 (NJMCL)	No

[1]Number of wells tested = 8; acetone, methylene chloride and bis(2-ethylhexyl)phthalate were detected but were qualified as invalid due to their presence in the blanks; [2]Contaminant of Concern; [3]ATSDR Reference Media Evaluation Guide; [4]New Jersey Maximum Contaminant Level; [5]EPA Region 3 Risk-Based Concentration; [6]ATSDR Environmental Media Evaluation Guide; [7]Lifetime Health Advisory for drinking water (EPA); [8]Action Level (New Jersey); [9]Recommended Upper Limit, New Jersey Secondary Drinking Water Standard

Table 11: Summary of Exposure Pathways

Medium	Point of Exposure	Exposure Route	Exposed Population	Exposure Pathway Classification		
				Past	Present	Future
Paint sludge	Site (including residential properties)	Ingestion, skin	Residents, hunters, recreators	Completed	Completed	Completed
Surface soils	Site, including residential properties	Ingestion, skin	Residents, hunters, recreators	Completed	Completed	Completed
Sediments	Site	Ingestion, skin	Residents, hunters, recreators	Completed	Completed	Completed
Surface water	Site (Springs/Seeps, Brooks)	Ingestion, inhalation, skin	Residents	Completed	Eliminated	Eliminated
Groundwater	Site	Ingestion, inhalation, skin	Residents	Eliminated	Eliminated	Eliminated
	Off-site (Potable wells)			Potential	Potential	Potential
Ambient air	Site (including residential properties)	Inhalation	Residents, hunters, recreators	Potential	Potential	Potential
Biota	Site	Ingestion	Residents, hunters, recreators	Potential	Potential	Potential

Table 12: Comparison of Calculated Exposure Dose Based on Maximum and Mean Concentration of Contaminants Detected in the Paint Sludge with Non-Cancer Health Guideline CV at the Ringwood Mine/Landfill site

Contaminant	Concentration (mg/kg)		Exposure Dose[1] (mg/kg/day)				Health Guideline CV (mg/kg/day)			Non Cancer Effects
	Max.	Mean	Maximum		Mean		ATSDR MRL[2]	RfD[3]	Reg 3 RfD[4]	
			Child	Adult	Child	Adult				
Semi Volatile Organic Compounds (SVOCs)										
Bis(2-ethylhexyl) phthalate	380	120	1.53×10^{-3}	1.74×10^{-4}	4.85×10^{-4}	5.54×10^{-5}	NA[5]	NA	0.02	No
Aroclor 1248	1.38	0.56	1.45×10^{-5}	1.65×10^{-6}	6.02×10^{-6}	6.88×10^{-7}	NA	0.00002	NA	No
Aroclor 1254	2.22	0.93								
Metals										
Antimony	460,000	47,137	1.85	2.11×10^{-1}	1.89×10^{-1}	2.16×10^{-2}	NA	0.0004	NA	**Yes**
Arsenic	16	4.33	6.43×10^{-5}	7.35×10^{-6}	1.74×10^{-5}	1.99×10^{-6}	0.0003	NA	NA	No
Cadmium	32	10.51	1.29×10^{-4}	1.47×10^{-5}	4.22×10^{-5}	4.83×10^{-6}	0.0002	NA	NA	No
Chromium[6]	2,400	1,639	9.64×10^{-3}	1.10×10^{-3}	6.59×10^{-3}	7.53×10^{-4}	NA	0.003	NA	**Yes**
Copper	2,100	565	8.44×10^{-3}	9.64×10^{-4}	2.27×10^{-3}	2.59×10^{-4}	NA	NA	0.04	No
Lead	310,000	64,880	1.25	1.42×10^{-1}	2.61×10^{-1}	2.98×10^{-2}	NA	NA	NA	**Yes**

[1]Adult Exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 7 year exposure duration, Child Exposure scenario: 3 days/week, 9 month/year, 200 mg/day ingestion rate, 16 kg body weight and 7 year exposure duration; [2]Minimal Risk Level; [3]EPA Reference Dose ; [4]EPA Region 3 Reference Dose; [5]Not Available; [6]as Chromium (VI)

Table 13: Comparison of Calculated Exposure Dose Based on Maximum and Mean Concentration of Contaminants Detected in the soil with Non-Cancer Health Guideline CV at the Ringwood Mine/Landfill site

Contaminant	Concentration (mg/kg)		Exposure Dose[1] (mg/kg/day)				Health Guideline CV (mg/kg/day)			Non Cancer Effects
	Max.	Mean	Maximum		Mean		ATSDR MRL[2]	RfD[3]	Reg 3 RfD[4]	
			Child	Adult	Child	Adult				
Volatile Organic Compound (VOCs)										
Benzene	34	34	1.37×10^{-4}	1.56×10^{-5}	1.37×10^{-4}	1.56×10^{-5}	NA[5]	0.004	0.004	No
Semi Volatile Organic Compound (SVOCs)										
Benzo[a]pyrene	0.18	0.14	7.23×10^{-7}	8.27×10^{-8}	5.6×10^{-7}	6.4×10^{-8}	NA	NA	NA	No
Metals										
Arsenic	9.6	2.03	3.85×10^{-5}	4.4×10^{-6}	8.16×10^{-6}	9.3×10^{-7}	0.0003	NA	NA	No
Lead	1,300	129.6	5.22×10^{-3}	6×10^{-4}	5.21×10^{-4}	6×10^{-5}	NA	NA	NA	**Yes**
Thallium	19	19	7.63×10^{-5}	8.72×10^{-6}	7.63×10^{-5}	8.72×10^{-6}	NA	NA	0.00007	No

[1]Adult Exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 40 year exposure duration, Child Exposure scenario: 3 days/week, 9 month/year, 200 mg/day ingestion rate, 16 kg body weight and 10 year exposure duration; [2]Minimal Risk Level; [3]EPA Reference Dose; [4]EPA Region 3 Reference Dose; [5]Not available

Table 14: Comparison of Calculated Exposure Dose Based on Maximum and Mean Concentration of Contaminants Detected in the sediment with Non-Cancer Health Guideline Values at the Ringwood Mines/Landfill site

Contaminant	Concentration (mg/kg)		Exposure Dose[1] (mg/kg/day)				Health Guideline CV (mg/kg/day)			Non Cancer Effects
	Max.	Mean	Maximum		Mean		ATSDR MRL[2]	RfD[3]	Reg 3 RfD[4]	
			Child	Adult	Child	Adult				
Semi Volatile Organic Compounds										
Benzo[a]pyrene	0.61	0.61	2.45×10^{-6}	2.08×10^{-7}	2.45×10^{-6}	2.08×10^{-7}	NA[5]	NA	NA	No
Metals										
Arsenic	31.4	9.13	1.26×10^{-4}	1.44×10^{-5}	3.67×10^{-5}	4.19×10^{-6}	0.0003	NA	NA	No
Thallium	14	9.45	5.63×10^{-5}	6.43×10^{-6}	3.8×10^{-5}	4.34×10^{-6}	NA	NA	0.00007	No

[1]Adult Exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 40 year exposure duration, Child Exposure scenario: 3 days/week, 9 month/year, 200 mg/day ingestion rate, 16 kg body weight and 10 year exposure duration; [2]Minimal Risk Level; [3]EPA Reference Dose; [4]EPA Region 3 Reference Dose; [5]Not Available

Table 15: Comparison of Calculated Exposure Dose Based on Maximum and Mean Concentration of Contaminants Detected in the Surface Water (Springs/Seeps, Brooks) with Non-Cancer Health Guideline Values at the Ringwood Mine/Landfill site

Contaminant	Concentration (μg/L)		Exposure Dose[1] (mg/kg/day)				Health Guideline CV (mg/kg/day)			Non Cancer Effects
	Max.	Mean	Maximum		Mean		ATSDR MRL[2]	RfD[3]	Reg 3 RfD[4]	
			Child	Adult	Child	Adult				
Volatile Organic Compounds (VOCs)										
Benzene	2	1.5	0.00013	0.00006	0.00009	0.00004	NA[5]	0.004	NA	No
1,2-Dichloropropane	12	12	0.00075	0.00034	0.00075	0.00034	0.09	NA	NA	No
Metals										
Arsenic	40	16.56	0.0025	0.0011	0.001	0.00047	0.0003	NA	NA	**Yes**
Lead	120	105	0.0075	0.0034	0.0065	0.003	NA	NA	NA	**Yes**
Mercury	8.7	2	0.00054	0.00025	0.00015	0.00007	NA	NA	NA	**Yes**

[1]Adult Exposure scenario: 3 days/week, 9 month/year, 2 L/day ingestion rate, 70 kg body weight and 20 year exposure duration, Child Exposure scenario: 3 days/week, 9 month/year, 1 L/day ingestion rate, 16 kg body weight and 10 year exposure duration; [2]Minimal Risk Level; [3]EPA Reference Dose; [4]EPA Region 3 Reference Dose; [5]Not Applicable

Table 16: Calculated Lifetime Excess Cancer Risk (LECR) based on the Maximum Concentration of Contaminants detected in the paint sludge

Contaminant	Concentration (mg/kg)		Cancer Exposure Dose[1] (mg/kg/day)		DHHS Cancer Class[2]	CSF[3]	LECR[4]	
	Maximum	Mean	Maximum	Mean			Max	Mean
Semi Volatile Organic Compounds (SVOCs)								
Bis(2-ethylhexyl) Phthalate	380	120	1.74×10^{-5}	5.54×10^{-6}	NA[5]	0.014	2.44×10^{-7}	7.75×10^{-8}
Aroclor 1248	1.38	0.565	6.34×10^{-8}	2.6×10^{-8}	2	2	1.27×10^{-7}	5.2×10^{-8}
Aroclor 1254	2.219	0.932	1.02×10^{-7}	4.28×10^{-8}	2	2	2.04×10^{-7}	8.6×10^{-8}
Metals								
Antimony	460,000	47,137	2.1×10^{-2}	2.16×10^{-3}	3	NA	NA	NA
Arsenic	16	4.33	7.35×10^{-7}	2×10^{-7}	1	1.5	1.1×10^{-6}	3×10^{-7}
Cadmium	32	10.51	1.47×10^{-6}	4.83×10^{-7}	1[6]	NA	NA	NA
Chromium	2,400	1,639	1.1×10^{-4}	7.53×10^{-5}	1[7]	NA	NA	NA
Copper	2,100	565	9.64×10^{-5}	2.6×10^{-5}	3	NA	NA	NA
Lead	310,000	64,880	1.42×10^{-2}	3×10^{-4}	3	NA	NA	NA

[1]Exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 7 year exposure duration; [2]Department of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified; [3]Cancer Slope Factor; [4]Lifetime Excess Cancer Risk; [5]Not available; [6]Information on the carcinogenicity of chromium by oral exposure in humans was unavailable; [7]Limited epidemiologic studies have indicated that exposure to cadmium in food or drinking water is not carcinogenic

Table 17: Calculated Lifetime Excess Cancer Risk (LECR) based on the Maximum and Mean Concentration of Contaminants detected in the soil

Contaminant	Concentration (mg/kg)		Cancer Exposure Dose[1] (mg/kg/day)		DHHS Cancer Class[2]	CSF[3]	LECR[4]	
	Maximum	Mean	Maximum	Mean			Max	Mean
Volatile Organic Compounds (VOCs)								
Benzene	34	34	1.56×10^{-5}	1.56×10^{-5}	1	0.055	8.6×10^{-7}	8.6×10^{-7}
Semi Volatile Organic Compounds (SVOCs)								
Benzo[a]pyrene	0.18	0.14	8.27×10^{-8}	6.43×10^{-8}	2	7.3	6×10^{-7}	4.7×10^{-7}
Metals								
Arsenic	9.6	2.03	4.4×10^{-6}	9.32×10^{-7}	1	1.5	6.6×10^{-6}	1.4×10^{-6}
Lead	1,300	129.6	6×10^{-4}	5.95×10^{-5}	3	NA[5]	NA	NA
Thallium	19	19	8.72×10^{-6}	8.72×10^{-6}	3	NA	NA	NA

[1]Exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 7 year exposure duration; [2]Department of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified; [3]Cancer Slope Factor; [4]Lifetime Excess Cancer Risk; [5]Not Applicable

Table 18: Calculated Lifetime Excess Cancer Risk (LECR) based on Maximum and Mean Concentration of Contaminants detected in the Sediment at the Ringwood Mines/Landfill Site

Contaminant	Concentration (mg/kg)		Cancer Exposure Dose[1] (mg/kg/day)		DHHS Cancer Class[2]	CSF[3]	LECR[4]	
	Maximum	Mean	Maximum	Mean			Max	Mean
Semi Volatile Organic Compounds (SVOCs)								
Benzo[a]pyrene	0.61	0.61	1.6×10^{-7}	1.6×10^{-7}	2	7.3	1.17×10^{-6}	1.17×10^{-6}
Metals								
Arsenic	31.4	9.13	8.24×10^{-6}	2.4×10^{-6}	1	1.5	1.24×10^{-5}	3.6×10^{-6}
Thallium	14	9.45	3.67×10^{-6}	2.48×10^{-6}	3	NA	NA	NA

[1]Exposure scenario: 3 days/week, 9 month/year, 100 mg/day ingestion rate, 70 kg body weight and 7 year exposure duration; [2]Department of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified; [3]Cancer Slope Factor; [4]Lifetime Excess Cancer Risk; [5]Not available

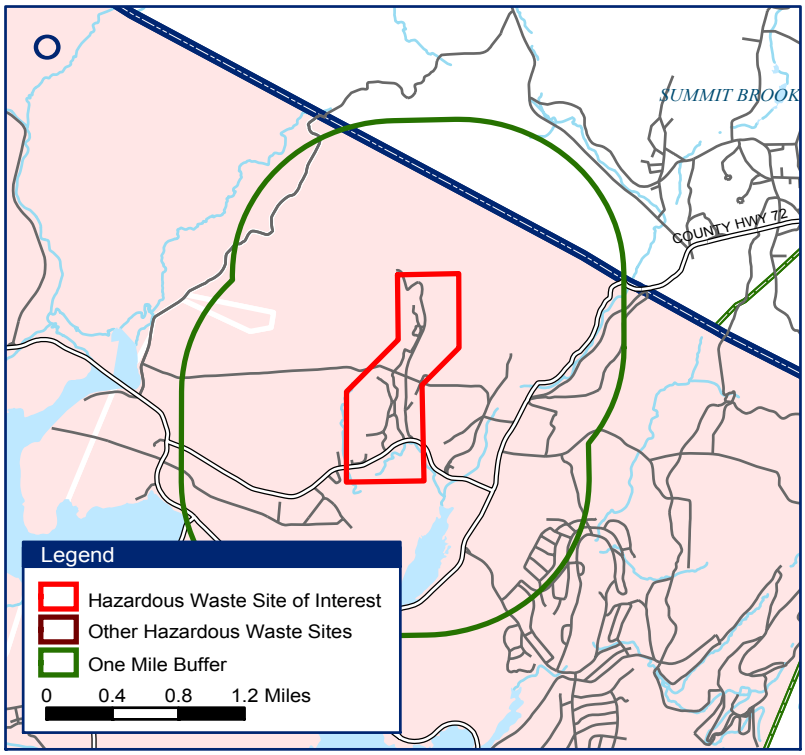
Table 19: Calculated Lifetime Excess Cancer Risk (LECR) based on Maximum Concentration of Contaminants detected in the Surface Water (Springs/Seeps, Brooks)

Contaminant	Concentration (µg/L)		Cancer Exposure Dose (mg/kg/day)[1]		DHHS Cancer Class[2]	CSF[3]	LECR[4]	
	Maximum	Mean	Maximum	Mean			Max	Mean
Volatile Organic Compounds (VOCs)								
Benzene	2	1.5	0.00002	0.00001	1	0.055	9×10^{-7}	6.73×10^{-7}
1,2-Dichloropropane	12	12	0.00114	0.00034	3	NA[5]	NA	NA
Metals								
Arsenic	40	16.56	0.00033	0.00014	1	1.5	4.9×10^{-4}	2.03×10^{-4}
Lead	120	105	0.00098	0.00086	3	NA	NA	NA
Mercury	8.7	2	0.00007	0.00002	3	NA	NA	NA

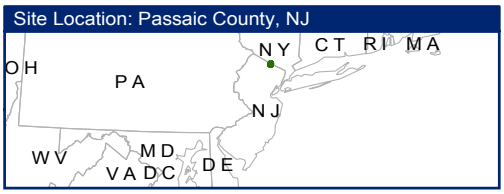
[1]Exposure scenario: 2 L/day ingestion rate, 70 kg body weight and 20 year exposure duration; [2]Department of Health and Human Services Cancer Class: 1 = known human carcinogen; 2 = reasonably anticipated to be a carcinogen; 3 = not classified; [3]Cancer Slope Factor; [4]Lifetime Excess Cancer Risk; [5]Not available

Ringwood Mines/Landfill
Ringwood Borough, NJ
EPA Facility ID: NJD980529739

ATSDR AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY | CDC



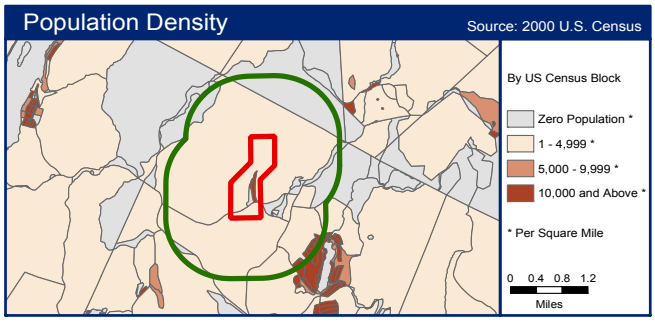
Base Map Source: Geographic Data Technology (DYNAMAP 2000), August 2002
Site Boundary Data Source: ATSDR Public Health GIS Program, August 2002
Coordinate System (All Panels): NAD 1983 State Plane New York East FIPS 3101 Feet

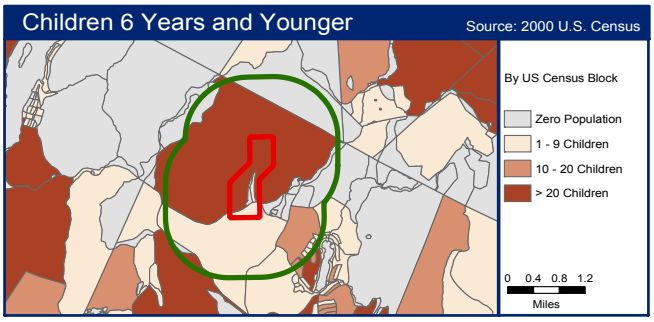


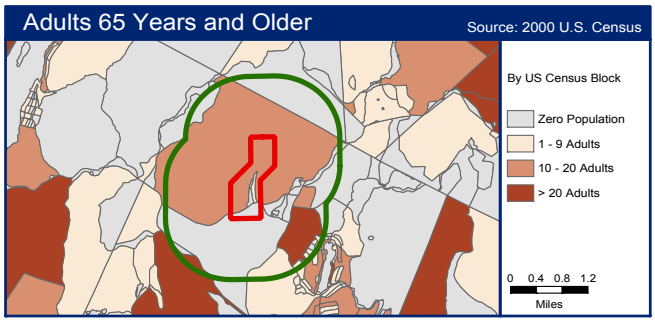
Demographic Statistics
Within One Mile of Site*

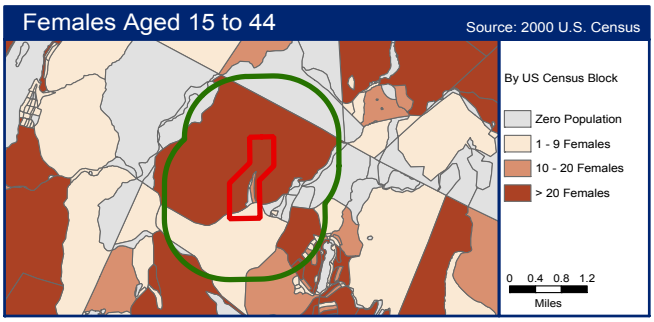
Total Population	866
White Alone	553
Black Alone	88
Am. Indian & Alaska Native Alone	149
Asian Alone	11
Native Hawaiian & Other Pacific Islander Alone	0
Some Other Race Alone	18
Two or More Races	47
Hispanic or Latino**	68
Children Aged 6 and Younger	87
Adults Aged 65 and Older	103
Females Aged 15 to 44	168
Total Housing Units	209

Demographics Statistics Source: 2000 U.S. Census
* Calculated using an area-proportion spatial analysis technique
** People who identify their origin as Hispanic or Latino may be of any race.









GENERATED: 12-03-2003

Figure 2: Ringwood Mines/Landfill Demographic Information

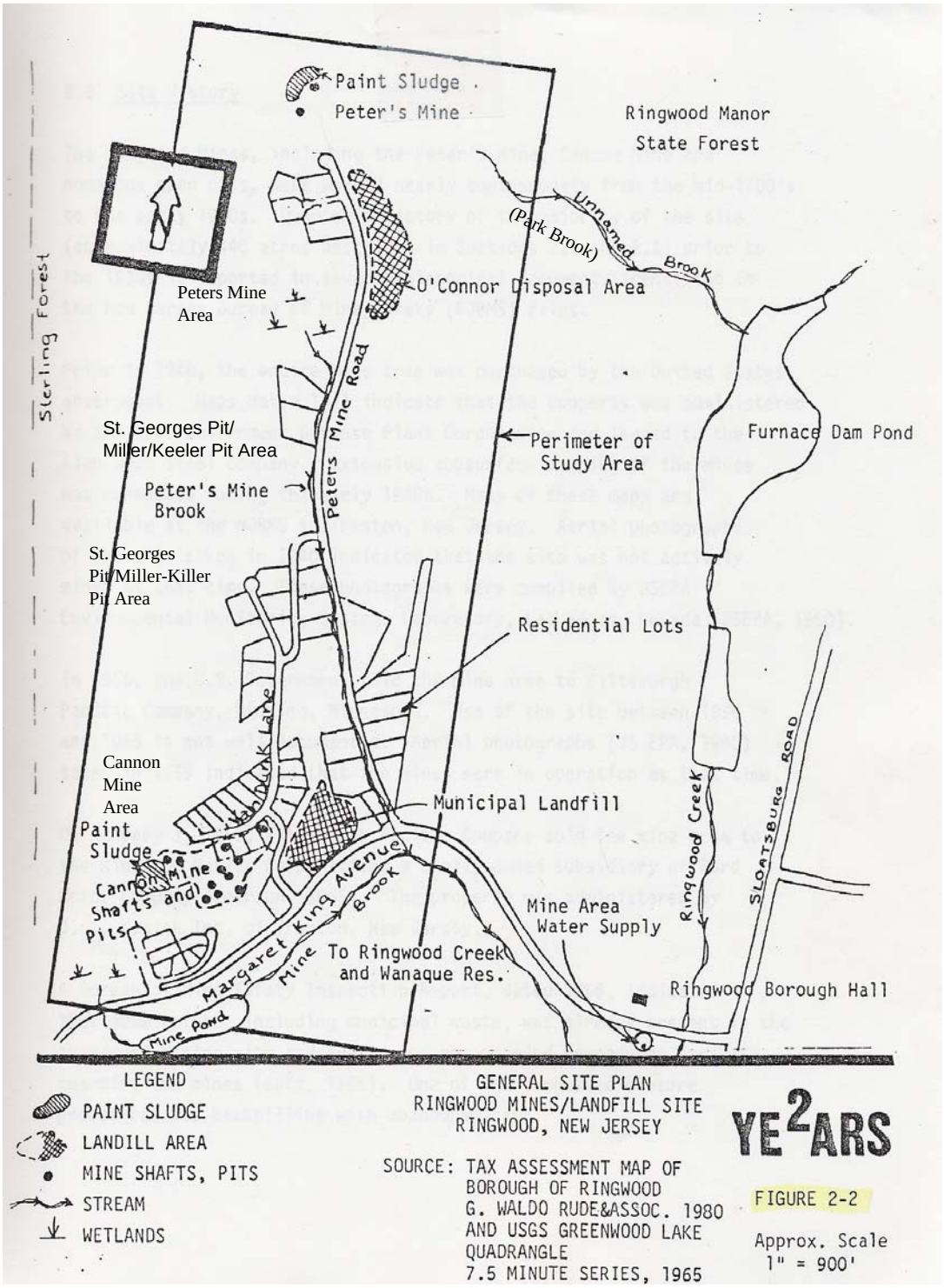
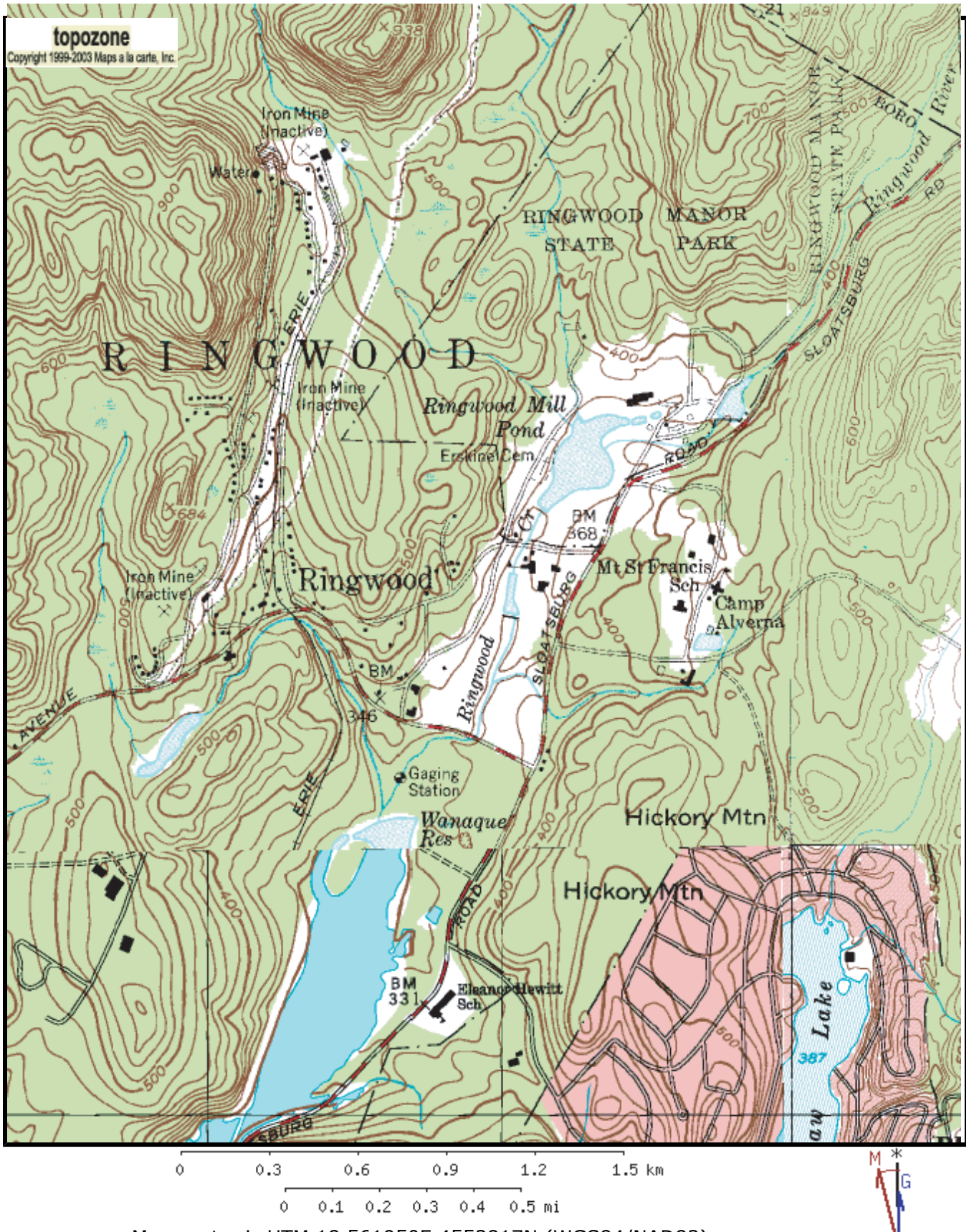


Figure 3: General Site Plan, Ringwood Mines/Landfill site



Map center is UTM 18 561950E 4553817N (WGS84/NAD83)
Greenwood Lake quadrangle
Projection is UTM Zone 18 NAD83 Datum

Figure 4: Topographic features of Ringwood Mines/Landfill stie and adjacent areas

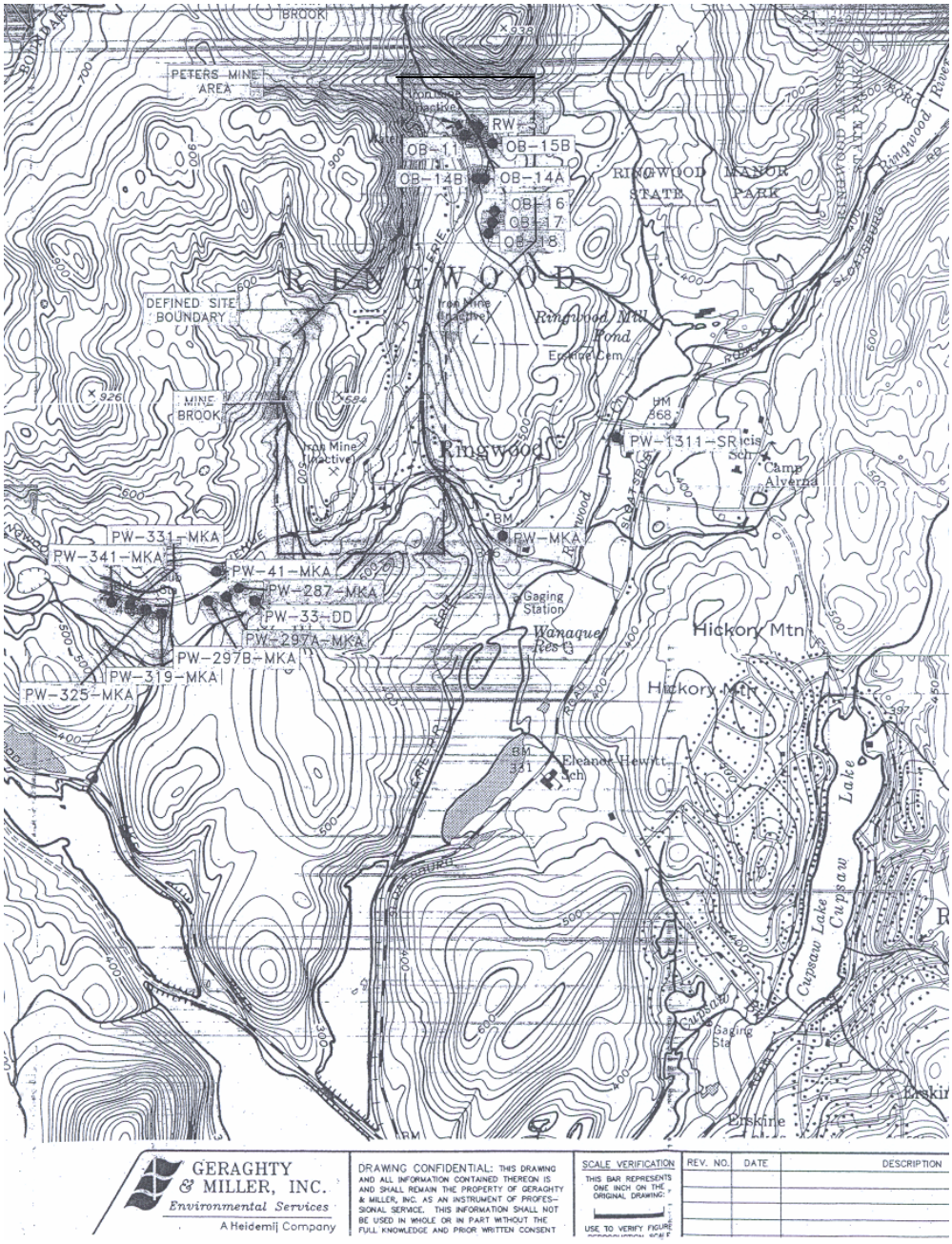


Figure 5: Location of Potables and Monitoring Wells sampled during Environmental Monitoring Program

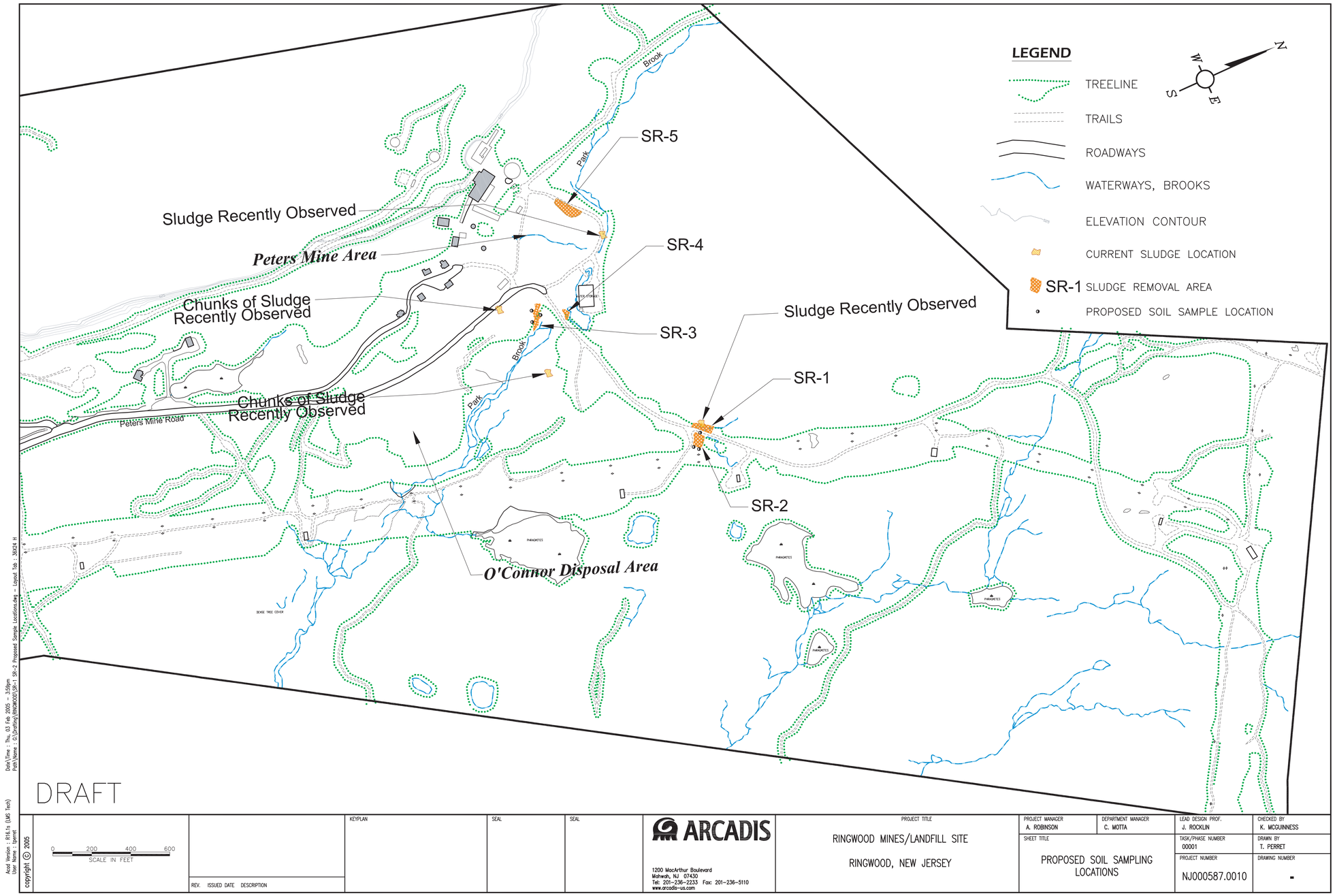


Figure 6: Proposed Soil Sampling Locations and Sludge Removal Areas

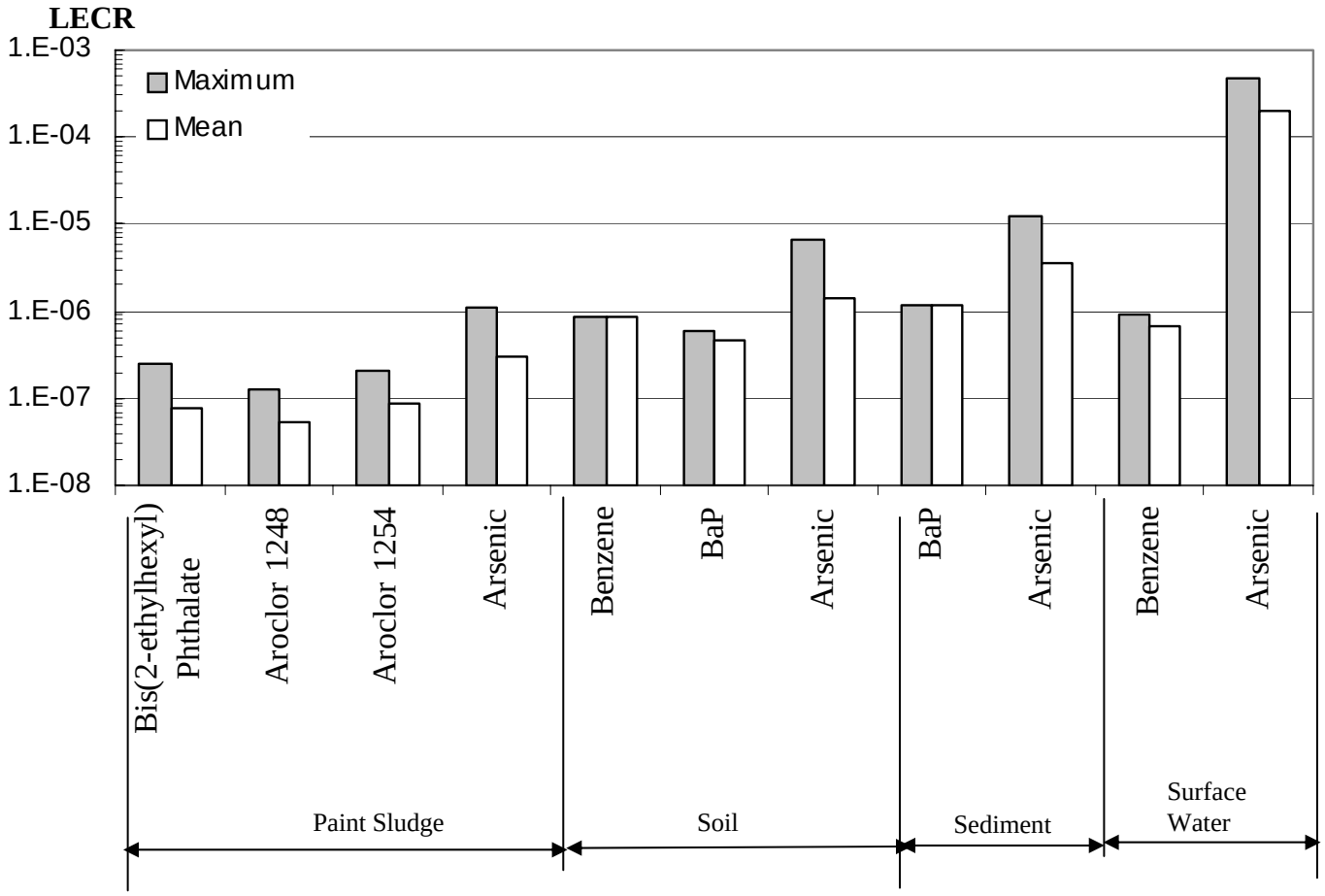


Figure 7: Calculated LECR from Exposures to Contaminants detected in paint sludge, Soil, Sediment and Surface Water

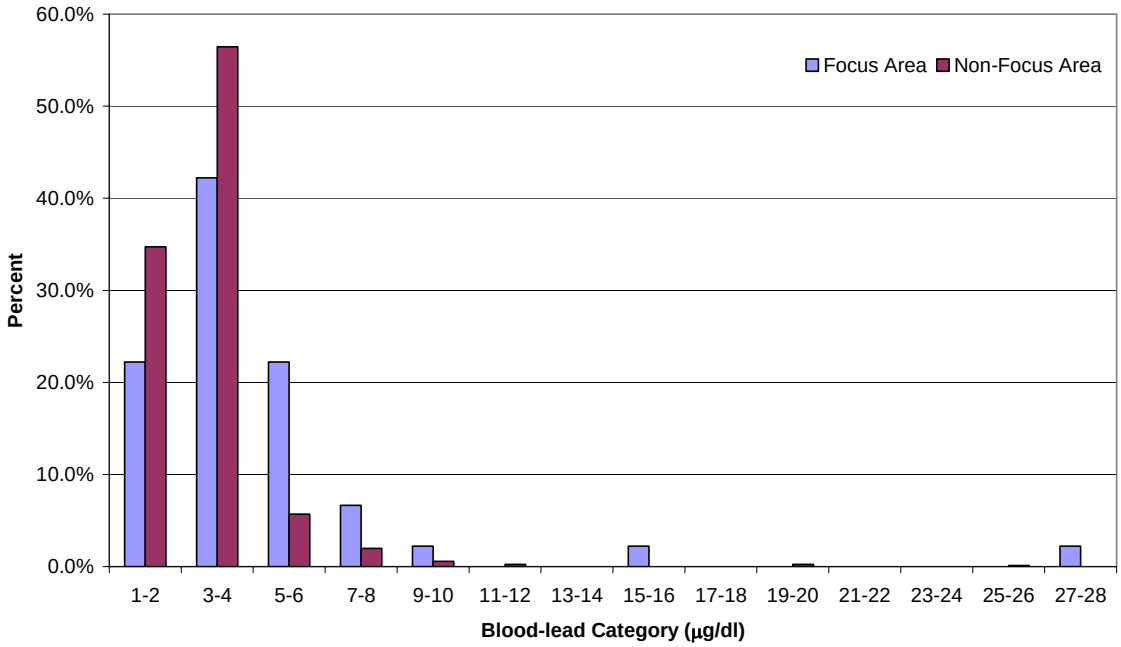


Figure 8: Blood-leads in Ringwood by Area (highest test per child only)

Photograph 1: On-site litter and trash

Photograph 2: On-site pond known as “the pool”

Photograph 3: One of the on-site brooks

Photograph 4: Hardened paint sludge

Photograph 5: On-site paint sludge and a drum lid

Photograph 6: Buried 55-gallons metal drum

Photograph 7: On-site area known as the "Sludge Hill"

Photograph 8: Area known as "Sludge Hill"

Appendix A

Ringwood Neighborhood Action Association

2A Van Dunk Lane

Ringwood, New Jersey 07456

Submission from the Ringwood Neighborhood Action Association to the New Jersey Department of Health and Senior Services concerning the Ringwood Mines/Landfill Site.

Tuesday, February 24, 2004

My name is Wayne Mann.

I am President of the Ringwood Neighborhood Action Association, and have held this position for the last three years. I am also a resident of the Ringwood Mines/Landfill Site, and have called this area my home for my entire life, as have several hundred other people. On behalf of the residents of this area, I thank the Department for its time today and for the opportunity to convey the concerns of residents.

The Ringwood Neighborhood Action Association is a not-for-profit organization that represents the interests of members of the Ringwood Mines/Landfill site community. In fulfilling my responsibilities to the community, I have become aware of concerns that residents have regarding possible health effects linked to waste dumped at the site. I have been asked by the Association and by those attending today's meeting to address these fears here today.

The Ringwood Mines/Landfill site is home to nearly 500 people. In 1983, the United States Environmental Protection Agency listed the site on the National Priorities List. EPA found the site posed an unacceptable and imminent threat to human health. Ford International Services, a subsidiary of Ford Motor Company, was identified as the potentially responsible party. As a result, U.S. EPA directed Ford to perform clean-up and monitoring efforts at the site.

The Ringwood Neighborhood Action Association has a number of concerns regarding the Ringwood Mines/Landfill site. These concerns have immerged as a result of issues raised by residents following media reports that Ford may cease its monitoring program at the site.

We have questions which need answering because, to date, we have all relied on government information that has said this site was, and is "safe" to human health. As an organization, the Ringwood Neighborhood Action Association is now trying to answer these questions, in light of increasing reports of illness among community members.

Particularly, we would like to address the issue of potential health effects caused by residents being exposed to waste at the site.

We have recently learned of the Agency for Toxic Substances and Disease Registry's 1989 conclusions regarding human health at this site: Health Assessment for Ringwood Mines/Landfill National Priorities List (NPL) Site, April 14, 1989. We have also learned of the conclusions contained in the 1994 co-operative health assessment between ATSDR and the New Jersey Department of Health: Site Review and Update - Ringwood Mines Landfill, September 8, 1994. We are confused as to the manner in which these assessments were performed, and the conclusions which were reached.

For instance, in its 1989 investigation, ATSDR expressly mentions a lack of available data concerning not only potable well locations and water consumption at the site, but also human exposure to contamination: Health Assessment, supra, pp.9-10. This information must have been important because ATSDR acknowledged not only that the "possible use of on-site contaminated ground water may pose a potential health risk", but flatly found that the site "poses a potential public health concern"

In addition to this, a June 28, 1982 Hazardous Ranking System survey of the site (at p.5) found that "potable water sourced from the site aquifer supplied private and industrial drinking water". This came on top of a November 15, 1979 EPA Hazardous Waste Site Survey Record which found (at p.4) that contamination was in the "immediate vicinity" of site residences.

What troubles this organization is that in its 1989 assessment, ATSDR concluded that there was no "indication in the information and data reviewed that human exposure is actually occurring at the present time or has occurred in the past." This is despite the contaminated drinking water, and the waste at our doorsteps.

We are, however, comforted by ATSDR's assertions in its 1989 health assessment that if data became "available suggesting that human exposure to significant levels of hazardous substance is currently occurring or has occurred in the past, ATSDR will re-evaluate this site for any indicated follow-up." We are pursuing our concerns with ATSDR.

With all due respect, we find the New Jersey Department of Health's approach to investigation of health concerns at this site to be seriously flawed. In its 1994 site review, the New Jersey Department of Health concluded that it was "unlikely that persons or children would have frequented these areas to facilitate a significant exposure", referring of course to areas containing waste. This conclusion was and is contrary to the facts.

For instance, one type of waste dumped at the site was "paint sludge", which the residents characterize as an oily paint residue with a thick consistency. Residents would not only play, walk, run and ride bicycles through the paint sludge, but salvage parts covered by it, swim

in water downstream from where it had been dumped, and fish and hunt in areas covered by it. Fires would burn regularly in areas contaminated by sludge.

The New Jersey Department of Health failed to discover that paint and solvent odors created an overwhelming stench at the site, that these odors were in our bathrooms when we bathed, and in our kitchens when we cooked. The New Jersey Department of Health failed to discover that some residents had skin rashes after bathing, and after coming into contact with paint sludge, that residents came into regular contact with soil contaminated by paint sludge, were regularly overcome by fumes coming from paint sludge, and even salvaged food dumped at the site by a local supermarket.

We respectfully disagree with the approach taken by the New Jersey Department of Health. Despite not being fully informed, the Department reached the ultimate conclusion that this site was not only safe, but posed no past threat to human health. At the very least, we ask the Department to re-evaluate its conclusions concerning past exposure.

Further, we are concerned as to the amount of contamination which remains at the Ringwood Mines/Landfill site. There are a number of areas where large amounts of paint sludge remains that is visible on the surface. Sitting on the table before me is some of the very sludge we have concerns about. We are providing this sludge to you today for your review and consideration. From what we are led to believe by the U.S. EPA, contamination remains in the upper aquifer at the site. Will this contamination cause us illness, particularly given that many of us have drunk water from natural springs at the site?

At no time has the Borough of Ringwood, the State of New Jersey, or the federal government studied the true extent of human exposure from, or the health of residents living at, the Ringwood Mines/Landfill site. We ask that the New Jersey Department of Health take this opportunity to fully explore these issues.

While the Department has taken this first important step, we feel that it must investigate the site conditions further as to past human exposure and likelihood for human injury. It has recently come to our knowledge that since dumping was discontinued, approximately seven (7) people have been linked with having leukemia, approximately sixty (60) with anemia, and we have reports of an asthma rate of over seventy (70) percent. Our women ask why they are coming down with ovarian cancer, tumors and cysts. Is this site causing us injury?

We fear that our community will not withstand the dangers presented by waste dumped at this site. The Department has failed us before, and we have every reason to believe that it will fail us again.

We leave the Department with two important questions that for the safety and health of this community require urgent responses:

(1) Is this site safe to our health? and
(2) Did this site pose a threat to our health in the past?

Thank you for your consideration and we await your response.

Sincerely,

Wayne Mann

President
Ringwood Neighborhood Action Association

Appendix B

Draft
Public Health Response Plan to Evaluate and Address the Public Health Impact of Environmental Contamination in the Ringwood Mines Area

September 2, 2004

Prepared by:
New Jersey Department of Health and Senior Services
Agency for Toxic Substances and Disease Registry

Proposed Public Health Response Plan
Ringwood Mines Site

Purpose

A Public Health Response Plan (PHRP) is a written plan that describes the scope of actions to be taken by the New Jersey Department of Health and Senior Services (NJDHSS), and the National Center for Environmental Health/Agency for Toxic Substances and Disease Registry (ATSDR) to address environmental health concerns in a community. Health agencies, regulatory agencies, and stakeholders will use the PHRP to help prioritize and evaluate the public health impact of environmental contamination. The PHRP helps facilitate increased communication and understanding between the involved agencies and community stakeholders. A PHRP is a "living" document; that is, it is updated and shared with the public as progress warrants.

A PHRP documents actions to be undertaken, which may include the following:

- identify and document community health concerns;
- assess site-related environmental contamination, document human exposures, and identify potential health implications;
- perform outreach and provide education to the impacted community to inform all stakeholders of the status and findings of the PHRP's elements.

The PHRP will also:

- establish regular communications avenues between the community and the agencies involved.
- estimate time frames for completion of each item.

This PHRP is being developed by the NJDHSS and the ATSDR in response to community concerns about health issues associated with environmental contamination in the area of the Ringwood Mines site, Ringwood, Passaic County, NJ.

Actions Planned

Identify Community Concerns

Community concerns are gathered through:

- Availability Sessions or other community-based meetings;

- a citizens focus group, as established by the community;

- petitions to the ATSDR or NJDHSS;
- the local health department;
- the US Environmental Protection Agency and/or the New Jersey Department of Environmental Protection;
- local newspapers and other media.

The Ringwood Mines area residents have expressed the following concerns about exposures and health issues associated with environmental contamination from the Ringwood Mines site:

- Exposures to paint sludge (characterized by residents as an oily paint residue with a thick consistency) occurred frequently in the past.
- Direct contact with paint sludge occurred as residents played, walked, ran, rode bicycles through, and salvaged auto parts covered by the sludge, as well as swam in water downstream from the dumping areas, and consumed wildlife that they had fished and hunted in areas contaminated by the sludge.
- Fires burned regularly at the site contaminated by sludge.
- Residents had frequent dermal contact with soil contaminated with paint sludge.
- Paint and solvent odors were overwhelming at the site, and were present in homes.
- Residents experienced skin rashes after bathing and after coming into contact with paint sludge.
- Residents did not have municipal water until 1988, and used surface and spring water for all domestic use prior to the installation of municipal water supply lines.
- Residents were regularly overcome by fumes coming from paint sludge.
- Residents consumed food products dumped at the site by a local food market.
- Since dumping ended in 1974, residents reported that approximately seven people have been linked with having leukemia and approximately 60 with anemia. The community also reports that their asthma rate is over 70 percent.
- Residents are concerned about rates of ovarian cancers, tumors and cysts, as well as heart, liver and kidney disorders.
- Residents also expressed concerns about child health, including childhood cancer, high blood pressure in children, asthma, severe skin rashes, and learning disabilities.
- Residents request that health studies, including an epidemiologic study, be conducted to determine the nature and cause of their injuries and illnesses.
- Residents want additional environmental sampling, including residential indoor air testing, to be conducted.
- Residents posed the following two questions:
  1. Is this site safe to health?
  2. Did this site pose a threat to health in the past?

Assess site-related environmental contamination, document human exposures, and identify potential health implications

The Public Health Assessment and Consultation Processes

A Public Health Assessment (PHA) is an evaluation of a contaminated site to find out if people were or are exposed to hazardous substances and, if so, whether that exposure is harmful. The PHA considers all of the ways that people may come in contact with site contaminants, either on-site or off-site. Scientists review environmental data to see how much contamination is at a site, where it is, and how people might be exposed to the contamination. A PHA will:

- identify past, present, potential future exposures through available data;
- where data is unavailable, consider if it may be obtained through other means, such as exposure investigations;
- document exposures and their potential impact to health.

If, during the process of identifying exposures, there is a biological plausibility that an exposure may lead to a specific illness or adverse health outcome, a further exploration of exposures and outcomes may be warranted.

- when exposure is likely to increase the risk for a reportable condition, or there is a source of systematically reported data, examine the sources of that data (e.g., NJ State Cancer Registry, NJ Birth Defects Registry, birth or death certificates);
- when there is no source of data, evaluate other means to obtain information;
- consider additional follow-up activities to evaluate the relationship between exposures and adverse health outcome(s).

Additionally, community concerns are incorporated into the PHA, and addressed as appropriate.

The PHA 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. Through its conclusions and recommendations, a PHA can also be used to recommend further evaluations or other actions, such as a health study or community education.

A Public Health Consultation is similar to a PHA, but usually focuses on a specific question about exposure or health.

Public Health Assessment for Ringwood Mines

The NJDHSS and the ATSDR will prepare a Public Health Assessment for the Ringwood Mines site. This PHA will incorporate the following elements:

Identify past and current exposures

The PHA will be developed for this community regarding past, present and potentially future exposures to contaminants from the Ringwood Mines site. The earlier Public Health Assessment and Site Review and Update will be re-evaluated in light of current conditions. Other community concerns that have been or are provided to the NJDHSS and ATSDR will be incorporated and addressed in the PHA.

Health outcome data review

The NJDHSS reviews disease incidence reported through existing health effects surveillance systems over time and across geographic areas. Surveillance of disease outcomes has deep historical roots, particularly for mortality due to infectious diseases. Surveillance of morbidity due to non-infectious diseases is a more recent historical development, as exemplified by the expanded development of state cancer registries and birth defects registries in the past two decades. In occupational health, surveillance of hazards and exposures has been integrated with disease surveillance for many years.

NJDHSS maintains a Childhood Lead Poisoning Surveillance System in which all clinical laboratories licensed by the state are required to report all blood lead tests performed on children. Current state regulation requires health care providers to test all one- and two-year old children for blood lead. Since 1999, results of all childhood blood lead tests were reportable, not just those considered elevated. The database records the child's name, address, birth date, and blood level as well as the medical provider and laboratory performing the test. The database contains files on more than 800,000 blood lead test results on more than 650,000 children, dating back to the mid-1970s.

The New Jersey State Cancer Registry (NJSCR) originated in October 1978 and is a statewide, population-based registry that collects summary stage of disease and vital status in accordance with all North American Association of Central Cancer Registries (NAACCR) and NCI Surveillance, Epidemiology, End Results (SEER) requirements. Since the inception of the NAACCR Gold Medal program in 1997, the NJSCR has achieved the highest standard each year and is estimated by NAACCR to have 100% complete reporting. The NJSCR has been a SEER

Registry since 2001. Approximately 43,000 new cancer cases are diagnosed annually in New Jersey and added to the registry, which contains over one million case records. Demographic data (sex, age, race, address, etc.) are collected for all cases. Cancer incidence data is currently complete through 2001.

Both childhood blood lead levels and community cancer incidence will be evaluated within the Public Health Assessment for the Ringwood Mines area as part of this PHRP.

Outreach and Education

Community Members

Throughout the process of completing the objectives of the PHRP, it is necessary that all members of the community are aware of the activities as well as background information that is important to their understanding of the activities. The NJDHSS and the ATSDR will:

- identify target audience(s), information needed, and method or methods to deliver the information;
- prepare, perform pilot tests if needed, distribute/provide, evaluate materials and other educational outreach activities.

Information will be obtained through:

- Availability Sessions or other community-based meetings to identify additional concerns of the residents, including health outcomes and exposure pathways;
- a citizens focus group, as established by the community;
- the local health department;
- the US Environmental Protection Agency and/or the New Jersey Department of Environmental Protection.

Residents living in the Ringwood Mines area were invited to participate in an Availability Session in February 2004. Residents were also invited to develop a PHRP with the NJDHSS and ASTDR.

In addition, two site visits have occurred to date (October 2003 and April 2004). The NJDHSS and ATSDR will continue to try to meet with residents to keep them informed of the progress of the investigation and obtain further concerns or comments.

Health Care Providers

It is also important that health care providers are knowledgeable about the potential for site-related exposures and the effects these exposures may have on health. The NJDHSS will establish communication with area health care providers, with emphasis on those providers identified by residents as being their source of medical care and treatment. General environmental health and site specific information will be provided through direct mailings, as well as a grand rounds or similar activity, as necessary and feasible.

Communication

The NJDHSS, ATSDR, and the community will ensure that the community and stakeholders are involved throughout the processes identified in this PHRP. This may include regular community meetings, newsletters or other written updates, or other means identified by the community that is within the capability of NJDHSS and ATSDR.

Time Line for Completion of Activities

The NJDHSS and ATSDR anticipate meeting the following schedule. It is subject to change, depending upon the availability of data, the complexity of the analyses, and need to perform additional activities that may be incorporated into this PHRP. However, these issues will be brought to the community as they are determined.

Activity	Anticipated or Actual Start	Anticipated Completion
Prepare PHA		
Identify Exposures	October 2003	October 2004
Health outcome data review:		October 2004
Childhood blood lead	May 2004	
Cancer incidence	June 2004	
Draft PHA for Public Comment	April 2004	December 2004
Finalize PHA	February 2005	March 2005
Community Outreach and Education	February 2004	March 2005
Community meetings		
Health Care Provider Outreach and Education	October 2004	March 2005

Appendix C

RINGWOOD NEIGHBORHOOD ACTION ASSOCIATION (RNAA)

STATEMENT ON NEW JERSEY DEPARTMENT OF HEALTH AND SENIOR SERVICES AND AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY'S PROPOSED PUBLIC HEALTH RESPONSE PLAN FOR THE RINGWOOD MINES SITE

September 23, 2004

Presented to:

Agency for Toxic Substances and Disease Registry, and
New Jersey Department of Health and Senior Services

The Ringwood Neighborhood Action Association (RNAA) is a community organization representing residents living on the Ringwood Mines/Landfill Site. The RNAA is actively working on behalf of residents of the mine area in an attempt to determine how the toxic waste dumped by Ford Motor Company during the 1960s and 1970s may be impacting the health of residents of our community. Towards this end, we appreciate the opportunity to meet today with the New Jersey Department of Health and Senior Services and the Agency for Toxic Substances and Disease Registry (ATSDR) to respond to the Proposed Public Health Response Plan for the Ringwood Mines Site.

The RNAA has been working with the Environmental Health Network to collect data on the health of residents and their exposure to Ford Motor Company's toxic waste. In this effort, we have conducted in-depth interviews of approximately 85% of the residents, and have completed a survey of each of these people consisting of approximately 800 questions. While we are continuing to interview residents, and to catalogue their diseases and history of exposure to Ford's toxic waste, sufficient data has been developed at this time to demonstrate that very serious health problems exist in this community. Specifically, preliminary data reveals heightened levels of the following:

Respiratory disease
Skin disease
Female reproductive disorders
Miscarriages
Birth Defects
Learning disabilities
Behavioral problems in children
Ovarian cancers and tumors

Cervical cancers and tumors
Leukemia
Colon and other cancers
Neurological disorders

Our surveys reveal further that residents for many years have experienced extensive and chronic exposure to Ford's toxic waste, and that this exposure continues.

With these findings in mind, we have reviewed the Proposed Public Health Response Plan for the Ringwood Mines Site, and have the following comments:

- Any proposed health response plan and accompanying health investigation must be geared towards helping our community understand the illnesses and deaths that we have seen, and are seeing among ourselves and our neighbors.

- The Proposed Plan in its present form does not appear adequately to explore and investigate the extent of illness and disease that we are witnessing, nor does it appear geared towards determining what is causing us to become sick.

- A fundamental problem with the Proposed Plan is that it was prepared without any opportunity for the impacted residents to be part of the drafting process. We are the major stakeholders in this process, as we are after all the ones living with-and dying from-these diseases. Any legitimate effort to investigate the health problems in our community must actively involve the residents at every stage, including drafting of a response plan.

- It appears that the Proposed Plan incorporates nothing more than a statistical analysis of previously collected data existing in the New Jersey State Cancer Registry, the New Jersey Birth Defects Registry, birth or death certificates, and the New Jersey Childhood Lead Poisoning Surveillance System. This kind of analysis WILL NOT show the overall health impact, past or present, in the Ringwood Mine community. As revealed in the government accountability report entitled, "Inconclusive by Design: Waste, Fraud and Abuse in Federal Health Research" (1991), these types of analyses have been done by health departments, using money supplied by ATSDR, in numerous communities throughout the United States with negative results and consequently NO help for impacted residents. As the Report concludes:

These intentionally inconclusive studies have been used by polluters and government officials to mislead local citizens into believing that further measures to prevent toxic exposures are unnecessary.

- The large majority of diseases that we are seeing in our community will not be caught by the proposed Response Plan. Any health assessment prepared using such incomplete data will be woefully inadequate.

We therefore propose an alternative plan, under which the Department of Health and Senior Services, ATSDR and the citizen stakeholders can work together cooperatively and effectively to

get to investigate the extent and causes of this serious health problem. Specifically, we propose the following:

- An environmental health initiative ("EHI") should immediately be developed in this community with resident stakeholders having FULL participation and partnership in this project.

- Residents will identify experts and doctors of their choice to participate in the development of this EHI.

- ATSDR funding currently earmarked for the proposed statistical analysis, plus additional funding, should be made available by ATSDR to fund EHI.

- The EHI will include investigation of ALL diseases suffered by community residents.

- The EHI also will investigate all past and present pathways of exposure to toxic waste, in an effort to identify the causes of the health crises in the Ringwood Mine area.

In closing, please let me emphasize that our concerns regarding health and safety are paramount. Our goal is to determine the extent of the current health crises and its causes, and to use the EHI to develop a response plan. We welcome the assistance of the New Jersey Department of Health and Senior Services and ATSDR in our efforts to determine why so many of us are sick and dying, and hope that both agencies will work cooperatively with residents towards this goal.

Wayne Mann
President, RNAA

Appendix D

Toxicologic Summaries

The toxicological summaries provided in this appendix are based on ATSDR's ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html). Health effects are summarized in this section for the chemicals of concern found off-site in area private wells. The health effects described in the section are typically known to occur at levels of exposure much higher than those that occur from environmental contamination. The chance that a health effect will occur is dependent on the amount, frequency and duration of exposure, and the individual susceptibility of exposed persons.

Benzene Benzene is a colorless liquid with a sweet odor. It evaporates into the air very quickly and dissolves slightly in water. It is flammable and is formed from both natural processes and human activities. Benzene is widely used in the United States; it ranks in the top 20 chemicals for production volume. Some industries use benzene to make other chemicals such as plastics, resins, and nylon and synthetic fibers. Benzene is also used to make rubber, lubricants, dyes, detergents, drugs, and pesticides. Natural sources of benzene include volcanoes and forest fires. Benzene is also a natural constituent of crude oil, gasoline, and cigarette smoke. Outdoor air contains low levels of benzene from tobacco smoke, automobile service stations, exhaust from motor vehicles, and industrial emissions. Indoor air generally contains higher levels of benzene from products such as glues, paints, furniture wax, and detergents.

Breathing very high levels of benzene can result in death, while high levels can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. Eating or drinking foods containing high levels of benzene can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, and death. The major effect of benzene from long-term (365 days or longer) exposure is on the blood. Benzene causes harmful effects on the bone marrow and can cause a decrease in red blood cells leading to anemia. It can also cause excessive bleeding and can affect the immune system, increasing the chance for infection. Some women who breathed high levels of benzene for many months had irregular menstrual periods and a decrease in the size of their ovaries. It is not known whether benzene exposure affects the developing fetus in pregnant women or fertility in men. Animal studies have shown low birth weights, delayed bone formation, and bone marrow damage when pregnant animals breathed benzene.

The USDHHS has determined that benzene is a known human carcinogen. Long-term exposure to high levels of benzene in the air can cause leukemia, cancer of the blood-forming organs.

1,2-Dichloropropane 1,2-Dichloropropane is a colorless, flammable liquid with a chloroform-like odor. It is moderately soluble in water and readily evaporates into air. It does not occur naturally in the environment. 1,2-Dichloropropane production in the United States has declined over the past 20 years. It was used in the past as a soil fumigant, chemical intermediate, and industrial solvent and was found in paint strippers, varnishes, and furniture finish removers. Most of these uses were discontinued. Today, almost all of the 1,2-dichloropropane is used as a chemical intermediate to make perchloroethylene and several other related chlorinated chemicals.

Individuals who intentionally or accidentally breathe high levels of 1,2-dichloropropane have experienced difficulty breathing, coughing, vomiting, nosebleed, fatigue, and damage to blood cells, liver, and kidneys. Ingestion of cleaning solutions containing 1,2-dichloropropane caused headaches, dizziness, nausea, liver and kidney damage, anemia, coma, and death.

Breathing low levels of 1,2-dichloropropane over short- or long-term periods causes damage to the liver, kidney, and respiratory system in animals. Breathing high levels causes death. Similar effects have been reported when animals were given 1,2-dichloropropane by mouth. Some studies indicate that ingesting 1,2-dichloropropane may cause reproductive effects. One study reported a delay in bone formation of the skull in fetal rats.

It is not known whether 1,2-dichloropropane causes cancer in people. The carcinogenicity of 1,2-dichloropropane has been evaluated in animal studies with rats and mice. Liver tumors have been observed in mice, and mammary gland tumors have been found in rats. The IARC has determined that 1,2-dichloropropane is unclassifiable as to human carcinogenicity.

Methylene Chloride Methylene chloride is a colorless liquid with a mild, sweet odor. It is used as an industrial solvent and as a paint stripper. It may also be found in some aerosol and pesticide products and is used in the manufacture of photographic film. The most likely way to be exposed to methylene chloride is by breathing contaminated air.

Breathing in large amounts of methylene chloride may cause dizziness, nausea, and tingling or numbness of fingers and toes. A person breathing smaller amounts of methylene chloride may become less attentive and less accurate in tasks requiring hand-eye coordination. We do not know if methylene chloride can affect the ability of people to have children or if it causes birth defects. Some birth defects have been seen in animals inhaling very high levels of methylene chloride.

We do not know if methylene chloride can cause cancer in humans. An increased cancer risk was seen in mice breathing large amounts of methylene chloride for a long time. The USDHHS has determined that methylene chloride can be reasonably anticipated to be a cancer-causing chemical, and the USEPA has determined that methylene chloride is a probable cancer-causing agent in humans.

Pentachlorophenol Pentachlorophenol is a manufactured chemical that does not occur naturally. Pure pentachlorophenol exists as colorless crystals. Impure pentachlorophenol (the form usually found at hazardous waste sites) is dark gray to brown and exists as dust, beads, or flakes. Pentachlorophenol was widely used as a pesticide and wood preservative. Since 1984, the purchase and use of pentachlorophenol has been restricted to certified applications (such as a wood preservative for utility poles, railroad ties, and wharf pilings) and unavailable to the general public

Occupational studies show that exposure to high levels of pentachlorophenol can cause very high fever, profuse sweating, and difficulty breathing. The body temperature can cause injury to various organs and tissues, and even death. Liver effects and damage to the immune

system have also been observed in humans exposed to high levels of pentachlorophenol for a long time. In animal studies, exposure to high doses of pentachlorophenol showed damage to the thyroid and reproductive system. Some of the harmful effects of pentachlorophenol are caused by the other chemicals present in technical grade pentachlorophenol.

Although there is sufficient evidence of carcinogenicity in animals, relevant human data is considered inadequate. Increases in liver, adrenal gland, and nasal tumors have been found in laboratory animals exposed to high doses of pentachlorophenol. The USEPA has determined that pentachlorophenol is a probable human carcinogen and the IARC considers it possibly carcinogenic to humans.

Bis(2-ethylhexyl)phthalate Bis(2-ethylhexyl)phthalate is a colorless oily liquid that is extensively used as a plasticizer in a wide variety of industrial, domestic and medical products. It is an environmental contaminant and has been detected in ground water, surface water, drinking water, air, soil, plants, fish and animals.

Animal studies have indicated that the primary target organs are the liver and kidneys; however, higher doses are reported to result in testicular effects and decreased hemoglobin and packed cell volume. The primary intracellular effects of bis(2-ethylhexyl)phthalate in the liver and kidneys are an increase in the smooth endoplasmic reticulum and a proliferation in the number and size of peroxisomes. An epidemiological study reported no toxic effects from occupational exposure to air concentrations of bis(2-ethylhexyl)phthalate up to 0.16 mg/m^3. Other studies on occupational exposures to mixtures of phthalate esters containing bis(2-ethylhexyl)phthalate have reported polyneuritis and sensory-motor polyneuropathy with decreased thrombocytes, leukocytes and hemoglobin in some exposed workers. Developmental toxicity studies with rats and mice have shown that bis(2-ethylhexyl)phthalate is fetotoxic and teratogenic when given orally during gestation. Oral exposure has also been shown to result in decreased sperm count in rats.

Bis(2-ethylhexyl)phthalate is known to induce the proliferation of peroxisomes, which has been associated with carcinogenesis. Dose-dependent, statistically-significant increases in the incidences of hepatocellular carcinomas and combined carcinomas and adenomas were seen in mice and rats exposed to bis(2-ethylhexyl)phthalate in their diet for 103 weeks. An increased incidence of neoplastic nodules and hepatocellular carcinomas was also reported in rats. The USEPA has classified antimony as a probable human carcinogen, on the basis of an increased incidence of liver tumors in rats and mice.

Polychlorinated biphenyls (PCBs) PCBs are mixtures of up to 209 individual chlorinated compounds (known as congeners). There are no known anthropogenic sources of PCBs. PCBs can exist as oily liquids, solids or vapor in air. Many commercial PCB mixtures are known by the trade name Aroclor. The majority of PCBs were used in dielectric fluids for use in transformers, capacitors, and other electrical equipment. Since PCBs build up in the environment and can cause harmful health effects, PCB production was stopped in the U.S. in 1977.

PCBs enter the environment during their manufacture, use, and disposal. PCBs can accumulate in fish and marine mammals, reaching levels that may be many thousands of times higher than in water. The most commonly observed health effects associated with exposures to large amounts of PCBs are skin conditions such as acne and rashes. Studies in exposed workers have shown changes in blood and urine that may indicate liver damage. PCB exposures in the general population are not likely to result in skin and liver effects. Most of the studies of health effects of PCBs in the general population examined children of mothers who were exposed to PCBs.

Animals administered with large PCB dose for short periods of time had mild liver damage and some died. Animals that ate smaller amounts of PCBs in food over several weeks or months developed various kinds of health effects, including anemia; acne-like skin conditions; and liver, stomach, and thyroid gland injuries. Other effects of PCBs in animals include changes in the immune system, behavioral alterations, and impaired reproduction. PCBs are not known to cause birth defects.

Few studies of workers indicate that PCBs were associated with certain kinds of cancer in humans, such as cancer of the liver and biliary tract. Rats that ate food containing high levels of PCBs for two years developed liver cancer. The Department of Health and Human Services (DHHS) has concluded that PCBs may reasonably be anticipated to be carcinogens. The EPA and the International Agency for Research on Cancer (IARC) have determined that PCBs are probably carcinogenic to humans.

Women who were exposed to relatively high levels of PCBs in the workplace or ate large amounts of fish contaminated with PCBs had babies that weighed slightly less than babies from women who did not have these exposures. Babies born to women who ate PCB-contaminated fish also showed abnormal responses in tests of infant behavior. Some of these behaviors, such as problems with motor skills and a decrease in short-term memory, lasted for several years. Other studies suggest that the immune system was affected in children born to and nursed by mothers exposed to increased levels of PCBs. There are no reports of structural birth defects caused by exposure to PCBs or of health effects of PCBs in older children. The most likely way infants will be exposed to PCBs is from breast milk. Transplacental transfers of PCBs were also reported. In most cases, the benefits of breast-feeding outweigh any risks from exposure to PCBs in mother's milk.

Polycyclic Aromatic Hydrocarbons (PAHs) Polycyclic aromatic hydrocarbons (PAHs) are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. PAHs are usually found as a mixture containing two or more of these compounds, such as soot. These include benzo(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene, benzo(g,h,i)perylene, indeno(1,2,3-cd0pyrene, phenanthrene, and naphthalene.

Some PAHs are manufactured. These pure PAHs usually exist as colorless, white, or pale yellow-green solids. PAHs are found in coal tar, crude oil, creosote, and roofing tar, but a few are used in medicines or to make dyes, plastics, and pesticides. Mice that were fed high levels of one PAH during pregnancy had difficulty reproducing and so did their offspring. These

offspring also had higher rates of birth defects and lower body weights. It is not known whether these effects occur in people. Animal studies have also shown that PAHs can cause harmful effects on the skin, body fluids, and ability to fight disease after both short- and long-term exposure. But these effects have not been seen in people.

The USDHHS has determined that some PAHs may reasonably be expected to be carcinogens. Some people who have breathed or touched mixtures of PAHs and other chemicals for long periods of time have developed cancer. Some PAHs have caused cancer in laboratory animals when they breathed air containing them (lung cancer), ingested them in food (stomach cancer), or had them applied to their skin (skin cancer).

Antimony Antimony is a silvery-white metal that is found in the earth's crust. Antimony ores are mined and then mixed with other metals to form antimony alloys or combined with oxygen to form antimony oxide. As alloys, it is used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, and pewter. Antimony oxide is added to textiles and plastics as fire retardant. It is also used in paints, ceramics, and fireworks, and as enamels for plastics, metal, and glass.

Antimony is released to the environment from natural sources and from industry. In the air, antimony is attached to very small particles that may stay in the air for many days. Most antimony particles settle in soil, where it attaches strongly to particles that contain iron, manganese, or aluminum.

Breathing high levels for a long time can irritate eyes and lungs and can cause heart and lung problems, stomach pain, diarrhea, vomiting, and stomach ulcers. In short-term studies, animals that breathed very high levels of antimony died. Animals that breathed high levels had lung, heart, liver, and kidney damage. In long-term studies, animals that breathed very low levels of antimony had eye irritation, hair loss, lung damage, and heart problems. Problems with fertility were also noted. In animal studies, fertility problems were observed when rats breathed very high levels of antimony for a few months.

Ingesting large doses of antimony can cause vomiting. Other effects of ingesting antimony are unknown. Long-term animal studies have reported liver damage and blood changes when animals ingested antimony. Antimony can irritate the skin if it is left on it.

Lung cancer has been observed in some studies of rats that breathed high levels of antimony. No human studies are available. The USDHHS, the International Agency for Research on Cancer, and the USEPA have not classified antimony as to its human carcinogenicity.

Arsenic Arsenic is a naturally occurring element widely distributed in the earth's crust. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. Arsenic in animals and plants combines with carbon and hydrogen to form organic arsenic compounds.

Inorganic arsenic compounds are mainly used to preserve wood. Breathing high levels of inorganic arsenic can give you a sore throat or irritated lungs. Ingesting high levels of inorganic arsenic can result in death. Lower levels of arsenic can cause nausea and vomiting, decreased production of red and white blood cells, abnormal heart rhythm, damage to blood vessels, and a sensation of "pins and needles" in hands and feet.

Ingesting or breathing low levels of inorganic arsenic for a long time can cause a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. Skin contact with inorganic arsenic may cause redness and swelling.

Organic arsenic compounds are used as pesticides, primarily on cotton plants. Organic arsenic compounds are less toxic than inorganic arsenic compounds. Exposure to high levels of some organic arsenic compounds may cause similar effects as those caused by inorganic arsenic.

Several studies have shown that inorganic arsenic can increase the risk of lung cancer, skin cancer, bladder cancer, liver cancer, kidney cancer, and prostate cancer. The World Health Organization (WHO), the USDHHS, and the USEPA have determined that inorganic arsenic is a human carcinogen.

Cadmium Cadmium is a natural element in the earth's crust. All soils and rocks, including coal and mineral fertilizers, contain some cadmium. Most cadmium used in the United States is extracted during the production of other metals like zinc, lead, and copper. Cadmium does not corrode easily and has many uses, including batteries, pigments, metal coatings, and plastics. Exposure to high levels of cadmium severely damages the lungs and can cause death. Eating food or drinking water with very high levels severely irritates the stomach, leading to vomiting and diarrhea. Long-term exposure to lower levels of cadmium in air, food, or water leads to a buildup of cadmium in the kidneys and possible kidney disease. Other long-term effects are lung damage and fragile bones. Skin contact with cadmium is not known to cause health effects in humans or animals.

Chromium Chromium is a naturally occurring element found in rocks, animals, plants, soil, and in volcanic dust and gases. Chromium is present in the environment in several different forms: chromium(0), chromium(III), and chromium(VI). No taste or odor is associated with chromium compounds. The metal chromium, which is the chromium(0) form, is used for making steel. Chromium(VI) and chromium(III) are used for chrome plating, dyes and pigments, leather tanning, and wood preserving.

Chromium enters the air, water, and soil mostly in the chromium(III) and chromium(VI) forms. In air, chromium compounds are present mostly as fine dust particles which eventually settle over land and water. Chromium can strongly attach to soil and only a small amount can dissolve in water and move deeper in the soil to underground water. Fish do not accumulate much chromium from water.

Breathing high levels of chromium(VI) can cause nasal irritation, such as runny nose, nosebleeds, and ulcers and holes in the nasal septum. Ingesting large amounts of chromium(VI) can cause stomach upsets and ulcers, convulsions, kidney and liver damage, and even death. Skin

contact with certain chromium(VI) compounds can cause skin ulcers. Allergic reactions consisting of severe redness and swelling of the skin have been noted.

Several studies have shown that chromium(VI) compounds can increase the risk of lung cancer. Animal studies have also shown an increased risk of cancer. The WHO has determined that chromium(VI) is a human carcinogen. The USDHHS has determined that certain chromium(VI) compounds are known to cause cancer in humans. The USEPA has determined that chromium(VI) in air is a human carcinogen.

It is unknown whether exposure to chromium will result in birth defects or other developmental effects in people. Birth defects have been observed in animals exposed to chromium(VI). It is likely that health effects seen in children exposed to high amounts of chromium will be similar to the effects seen in adults.

Copper High levels of copper can be harmful. Breathing high levels of copper can cause irritation of nose and throat. Ingesting high levels of copper can cause nausea, vomiting, and diarrhea. Very-high doses of copper can cause damage to liver and kidneys, and can even cause death.

Exposure to high levels of copper will result in the same type of effects in children and adults. We do not know if these effects would occur at the same dose level in children and adults. Studies in animals suggest that the young children may have more severe effects than adults, but we don't know if this would also be true in humans. There are a very small percentage of infants and children who are unusually sensitive to copper.

Birth defects or other developmental effects of copper in humans are unknown. Animal studies suggest that high levels of copper may cause a decrease in fetal growth.

The most likely human exposure pathway is through drinking water, especially if the water is corrosive and copper pipes are used for plumbing. One of the most effective ways to reduce copper exposure is to let the water run for at least 15 seconds first thing in the morning before drinking or using it. This reduces the levels of copper in tap water dramatically.

Copper is found throughout the body; in hair, nails, blood, urine, and other tissues. High levels of copper in these samples can show copper exposures. However, these tests can not predict occurrence of harmful effects. Tests to measure copper levels in the body require special equipment.

Human carcinogenicity of copper is unknown. The USEPA has determined that copper is not classifiable as to human carcinogenicity.

Lead Lead is a naturally occurring metal found in small amounts in the earth's crust. Lead can be found in all parts of our environment. Much of it comes from human activities including burning fossil fuels, mining, and manufacturing. Lead has many different uses. It is used in the production of batteries, ammunition, metal products (solder and pipes), and devices to shield X-rays. Because of health concerns, lead from gasoline, paints and ceramic products,

caulking, and pipe solder has been dramatically reduced in recent years. People may be exposed to lead by eating food or drinking water that contains lead, spending time in areas where lead-based paints have been used and are deteriorating, and by working in a job or engaging in a hobby where lead is used. Small children are more likely to be exposed to lead by swallowing house dust or soil that contains lead, eating lead-based paint chips or chewing on objects painted with lead-based paint.

Lead can affect many organs and systems in the body. The most sensitive is the central nervous system, particularly in children. Lead also damages kidneys and the reproductive system. The effects are the same whether it is breathed or swallowed. At high levels, lead may decrease reaction time, cause weakness in fingers, wrists, or ankles, and possibly affect the memory. Lead may cause anemia, a disorder of the blood. It can also damage the male reproductive system. The connection between these effects and exposure to low levels of lead is uncertain.

Children are more vulnerable to lead poisoning than adults. A child who swallows large amounts of lead, for example by eating old paint chips, may develop blood anemia, severe stomachache, muscle weakness, and brain damage. A large amount of lead might get into a child's body if the child ate small pieces of old paint that contained large amounts of lead. If a child swallows smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, however, lead can affect a child's mental and physical growth. Exposure to lead is more dangerous for young children and fetuses. Fetuses can be exposed to lead through their mothers. Harmful effects include premature births, smaller babies, decreased mental ability in the infant, learning difficulties, and reduced growth in young children. These effects are more common if the mother or baby was exposed to high levels of lead.

The USDHHS has determined that two compounds of lead (lead acetate and lead phosphate) may reasonably be anticipated to be carcinogens based on studies in animals. There is inadequate evidence to clearly determine whether lead can cause cancer in people.

Mercury Mercury is a naturally occurring metal which has several forms. Metallic mercury is a shiny, silvery liquid which, when heated, can be a colorless, odorless gas. Mercury combines with other elements, such as chlorine, sulfur, or oxygen, to form inorganic mercury compounds or "salts," which are usually white powders or crystals. Mercury also combines with carbon to make organic mercury compounds. The most common one, methylmercury, is produced mainly by microscopic organisms in the water and soil. Metallic mercury is used to produce chlorine gas and caustic soda, and is also used in thermometers, dental fillings, and batteries. Mercury salts are sometimes used in skin lightening creams and as antiseptic creams and ointments. People are commonly exposed to mercury by eating fish or shellfish contaminated with methylmercury, breathing vapors in air from spills, incinerators, and industries that burn mercury-containing fuels, the release of mercury from dental work, working with mercury, or practicing rituals that include mercury.

The nervous system is very sensitive to all forms of mercury. Methylmercury and metallic mercury vapors are more harmful than other forms, because more mercury in these

forms reaches the brain. Exposure to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing fetus. Effects on brain functioning may result in irritability, shyness, tremors, changes in vision or hearing, and memory problems. Short-term exposure to high levels of metallic mercury vapors may cause effects including lung damage, nausea, vomiting, diarrhea, increases in blood pressure or heart rate, skin rashes, and eye irritation.

Young children are more sensitive to mercury than adults. Mercury in the mother's body passes to the fetus and may accumulate there. It can also pass to a nursing infant through breast milk, although the benefits of breast feeding may be greater than the possible adverse effects of mercury in breast milk.

Harmful effects due to mercury that passes from the mother to the fetus include brain damage, mental retardation, incoordination, blindness, seizures, and inability to speak. Children poisoned by mercury may develop problems with their nervous and digestive systems, and kidney damage.

There are inadequate human cancer data available for all forms of mercury. Mercuric chloride has caused increases in several types of tumors in rats and mice, and methylmercury has caused kidney tumors in male mice. The USEPA has determined that mercuric chloride and methylmercury are possible human carcinogens.

Thallium. Thallium is a bluish-white metal that is found in trace amounts in the earth's crust. It is used mostly in manufacturing electronic devices, switches, and closures, primarily for the semiconductor industry. It also has limited use in the manufacture of special glass and for certain medical procedures. Thallium enters the environment primarily from coal-burning and smelting, in which it is a trace contaminant of the raw materials. Exposure to thallium may occur through eating food contaminated with thallium, breathing workplace air in industries that use thallium, smoking cigarettes, or contact with contaminated soils, water or air.

Exposure to high levels of thallium can result in harmful health effects. A study on workers exposed on the job over several years reported nervous system effects, such as numbness of fingers and toes, from breathing thallium. Studies in people who ingested large amounts of thallium over a short time have reported vomiting, diarrhea, temporary hair loss, and effects on the nervous system, lungs, heart, liver, and kidneys. High exposures can cause death. It is not known what the reproductive effects are from breathing or ingesting low levels of thallium over a long time. Studies in rats exposed to high levels of thallium showed adverse reproductive effects, but such effects have not been seen in people. Animal data suggest that the male reproductive system may be susceptible to damage by low levels of thallium.

The USDHSS, IARC, and the USEPA have not classified thallium as to its human carcinogenicity. No studies are available in people or animals on the carcinogenic effects of breathing, ingesting, or touching thallium.

Appendix E

Assessment of Joint Toxic Action of Chemical Mixtures

Non-Cancer

In the Ringwood Mines/Landfill site, residents were exposed to contaminants detected in paint sludge, soil, sediment and surface water. Although the evaluation of health effects associated with individual chemicals for specific pathways was conducted earlier, the exposure to chemical mixtures should be considered. Exposure to multiple chemicals with similar toxicological characteristics may increase their public health impact (ATSDR 2005). The severity of the impact depends on the particular chemicals being ingested, pharmacokinetics, and toxicity in children and adults.

To evaluate the risk for non-cancer adverse health effects of chemical mixtures, a hazard index (HI) for the chemicals was calculated (ATSDR 2005). The hazard index is defined as the sum of the hazard quotients (i.e., estimated exposure dose of a chemical divided by applicable health guideline CV). If the HI is less than 1.0, it is highly unlikely that significant additive or toxic interaction would occur, so no further evaluation is necessary. If the HI is greater than 1.0, then further evaluation is necessary. For Ringwood Mines/Landfill site, based on the mean concentration of contaminants detected (the more likely scenario), the HI calculated for children for the paint sludge (480), soil (1.15) and surface water (3.33) was greater than 1.0; for adults, the HI calculated for the paint sludge (57.37) and surface water (1.57) was greater than 1.0 (see Table F1 and F2, Appendix F).

For chemical mixtures with an HI greater than 1.0, the estimated doses of the individual chemicals are compared with their NOAELs or comparable values. If the dose of one or more of the individual chemicals is within one order of magnitude of its respective NOAEL, then potential exists for additive or interactive effects. The ratio of exposure dose to NOAEL for the contaminants was calculated (see Table F1 and F2, Appendix F). Since the potential exists for additive or interactive effects of chemical mixtures from exposures to paint sludge and surface water in children and paint sludge in adults, an in-depth mixtures evaluation is required using ATSDR's *Guidance Manual for the Assessment of Joint Action of Chemical Mixtures* (2004).

The flow chart in Figure F1 gives an overview of the steps involved in the decision process for the exposure-based assessment of the potential non-cancer impact of joint toxic action (ATSDR 2004). Since toxicological profiles dealing with the mixture of chemicals detected in the paint sludge and surface water is unavailable, a component approach is employed (Step 3, Figure F1, Appendix F). The hazard quotients of Aroclor, antimony, cadmium, chromium and lead in the paint sludge and arsenic in the surface water were at least 0.1; they were selected as component of concern. Physiologically-based pharmacokinetic/pharmacodynamic (PBPK/PD) model is unavailable for the mixture (Step 4, Figure F1, Appendix F). The critical effects of the components of concern are as follows (Step 5, Figure F1, Appendix F):

Lead	Arsenic	Cadmium	Chromium (IV)	Aroclor	Antimony
Neurological **Hematological** **Cardiovascular** Renal Testicular	***Dermal lesions*** **Cardiovascular** **Hematological** Renal **Neurological** **Cancer**	***Renal (proteinuria)*** Cardiovascular Hematological Hepatic Neurological Testicular	Hematological Hepatic Renal Neurological Testicular	***Immunological*** ***Neurological*** Cancer Dermal Hepatic Hematological	***Lifespan***

[a]The basis for the MRL or health assessment approach is bolded and italicized; other sensitive effects are bolded; and less sensitive effects in common across two or more metals, or known to be affected synergistically by another metal in the mixture, are listed without bold or italics

Hazard indexes were then calculated using target organ toxicity dose (TTD) method for components with different critical effects (Step 6b, Figure F1, Appendix F). The magnitude of the hazard index shows potential neurological, dermal, renal, cardiovascular, hematological, testicular health effects in children and potential neurological, renal, cardiovascular and hematological health effects in adults due to additivity (see Table F3, Appendix F). As such, further evaluation of interaction (Step 7b, Figure F1, Appendix F) is warranted.

Binary weight of evidence (BINWOE) scores relevant to the route, duration, and endpoint for the four chemical pairs are available (ATSDR 2004); the BINWOE scores for aroclor and antimony are unavailable. The predicted direction of joint toxic action for neurological effects, an endpoint common to all four components, is greater than additive for the effect of lead on arsenic, arsenic on lead, cadmium on lead, and chromium(VI) on arsenic, and less than additive for the effect of arsenic on chromium(VI) (see Table F4, Appendix F). The remaining seven BINWOE scores were indeterminate due to a lack of toxicological and mechanistic data. Thus, the potential health hazard may be somewhat greater than estimated by the endpoint-specific hazard index for neurological effects (i.e., 10.3 for children and 4.7 for adults). The impact of interaction on potential health hazard is summarized as follows:

Health Effect	Impact of interaction
Neurological	Higher
Renal	Lower
Cardiovascular	Little Impact
Hematological	Lower
Testicular	Higher
Dermal	Indeterminate

Cancer

The flow chart in Figure F2 gives an overview of the steps involved in the decision process for the exposure-based assessment of the potential cancer impact of joint toxic action (ATSDR 2004). The cancer risk estimate for the paint sludge, soil, sediment and surface water are presented in Table 16 through 19. Since the estimated risks are not

greater or equal to 1 x10^{-6} for at least two of the individual component (Step 3, Figure F2, Appendix F), additivity or interaction are unlikely to result in health hazard.

Table E1: Multiple Chemical Exposure Analysis for Child: Sludge, Soil, Sediment and Surface Water

Contaminant	Child Exposure Dose (mg/kg/day)	Health Guideline CV (mg/kg/day)	Hazard Quotient	HI	NOAEL (mg/kg/day)	Dose/NOAEL
Paint Sludge						
Bis(2-ethylhexyl) phthalate	0.00048	0.02	0.024	480.7	NA[1]	NA
Aroclor 1248/1254	0.000006	0.00002	0.3		0.005	0.0012
Antimony	0.189	0.0004	472		0.003	63
Arsenic	0.0000174	0.0003	0.058		0.0008	0.02
Cadmium	0.000042	0.0002	0.21		0.005	0.0084
Chromium	0.0066	0.003	2.2		2.5	0.002
Copper	0.00227	0.04	0.056		NA	NA
Lead[2]	59	10	5.9		NA	NA
Soil						
Benzene	0.000137	0.004	0.034	1.15	1.2	0.0001
Benzo[a]pyrene	0.00000056	NA	NA		NA	NA
Arsenic	0.00000816	0.0003	0.027		0.009	0.0009
Lead	0.000521	NA	NA		NA	NA
Thallium	0.0000763	0.00007	1.09		0.25	0.0003
Sediment						
Benzo[a]pyrene	0.00000245	NA	NA	0.66		
Arsenic	0.0000367	0.0003	0.12			
Thallium	0.000038	0.00007	0.54			

Table E1: (Cont'd.)

Contaminant	Child Exposure Dose (mg/kg/day)	Health Guideline CV (mg/kg/day)	Hazard Quotient	HI	NOAEL (mg/kg/day)	Dose/NOAEL
Surface Water						
Benzene	0.00009	0.004	0.022	3.33	NA	NA
1,2-Dichloropropane	0.00075	0.09	0.008		NA	NA
Arsenic	0.001	0.0003	3.3		0.009	0.11
Lead	0.0065	NA	NA		NA	NA
Mercury	0.00015	NA	NA		NA	NA

[1]Not available; [2]Based on blood lead levels in μg/dL

Table E2: Multiple Chemical Exposure Analysis for Adult: Sludge, Soil, Sediment and Surface Water

Contaminant	Adult Exposure Dose (mg/kg/day)	Health Guideline CV (mg/kg/day)	Hazard Quotient	HI	NOAEL (mg/kg/day)	Dose/NOAEL
Paint Sludge						
Bis(2-ethylhexyl) phthalate	5.54×10^{-5}	0.02	0.0027	57.37	NA[1]	NA
Aroclor 1248/1254	6.88×10^{-7}	0.00002	0.034		0.005	0.00013
Antimony	2.16×10^{-2}	0.0004	54		0.003	7.2
Arsenic	1.99×10^{-6}	0.0003	0.0066		0.0008	0.0024
Cadmium	4.83×10^{-6}	0.0002	0.024		0.005	0.001
Chromium	7.53×10^{-4}	0.003	0.25		2.5	0.0003
Copper	2.59×10^{-4}	0.04	0.0064		NA	NA
Lead[2]	30.5	10	3.05		NA	NA
Soil						
Benzene	1.56×10^{-5}	0.004	0.004	0.12		
Benzo[a]pyrene	6.4×10^{-8}					
Arsenic	9.3×10^{-7}	0.0003	0.003			
Lead	6×10^{-5}					
Thallium	8.72×10^{-6}	0.00007	0.12			
Sediment						
Benzo[a]pyrene	2.08×10^{-7}	NA	NA	0.076		
Arsenic	4.19×10^{-6}	0.0003	0.014			
Thallium	4.34×10^{-6}	0.00007	0.062			

Table E2: (Cont'd.)

Contaminant	Adult Exposure Dose (mg/kg/day)	Health Guideline CV (mg/kg/day)	Hazard Quotient	HI	NOAEL (mg/kg/day)	Dose/NOAEL
Surface Water						
Benzene	4.0×10^{-5}	0.004	0.01	1.57	NA	NA
1,2-Dichloropropane	3.4×10^{-4}	0.09	0.0037		NA	NA
Arsenic	4.7×10^{-4}	0.0003	1.56		0.009	0.05
Lead	3.0×10^{-3}	NA	NA		NA	NA
Mercury	7.0×10^{-5}	NA	NA		NA	NA

[1]Not available; [2]Based on blood lead levels in μg/dL

Table E3: Target Organ Toxicity Dose modification of HI Analysis: Components with different critical effects

	Exposure Dose (mg/kg/day)	Neuro-logical	Dermal	Renal	Cardio-vascular	Hemato-logical	Testicular	Hepatic	Immunological
Child									
Lead	59[1]	5.9	NA[2]	1.74	5.9	5.9	1.48	NA	NA
Arsenic	0.001	3.33	1.25	0.01	3.33	1.67	NA	NA	NA
Cadmium	4.0×10^{-5}	0.21	NA	0.21	0.01	0.05	0.01	NA	NA
Chromium (VI)	0.0066	0.66	NA	0.66	NA	2.20	1.32	NA	NA
Aroclor	6.0×10^{-6}	0.2	NA	NA	NA	0.01	NA	0.06	0.3
Hazard Index =		**10.3**	**1.25**	**2.62**	**9.24**	**9.83**	**2.81**	**0.06**	**0.3**
Adult									
Lead	30.5[1]	3.05	NA	0.9	3.05	3.05	0.76	NA	NA
Arsenic	4.70×10^{-4}	1.57	0.59	0.01	1.57	0.78	NA	NA	NA
Cadmium	4.83×10^{-6}	0.02	NA	0.02	0.00	0.01	0.00	NA	NA
Chromium (VI)	7.53×10^{-4}	0.08	NA	0.08	NA	0.25	0.15	NA	NA
Aroclor	6.88×10^{-7}	0.02	NA	NA	NA	0.00	NA	0.007	0.03
Hazard Index =		**4.74**	**0.6**	**1**	**4.62**	**4.09**	**0.9**	**0.007**	**0.03**

[1]Blood lead levels in μg/dL; [2]Not available

Table E4: Matrix of BINWOE Determinations for Simultaneous Oral Exposure to Chemicals of Concern

		On Toxicity of			
		Lead	Arsenic	Cadmium	Chromium(VI)
Neurological Toxicity					
Effect of	Lead		**>IIIB (+0.23)**	**? (0)**	? (0)
	Arsenic	**>IIB (+0.50)**		? (0)	**<IIIC2ii (-0.06)**
	Cadmium	**>IIIC (+0.10)**	? (0)		? (0)
	Chromium(VI)	? (0)	**>IIIC (=0.10)**	? (0)	
Dermal Toxicity					
Effect of	Lead		**? (0)**	NA	NA
	Arsenic	NA		NA	NA
	Cadmium	NA	**? (0)**		NA
	Chromium(VI)	NA	**>IIIC(+0.10)**	NA	
Renal Toxicity					
Effect of	Lead		**<IIIB (-0.23)**	**=IIAii (0)**	? (0)
	Arsenic	**<IIIB (-0.23)**		**? (0)**	**<IIIB2ii (-0.14)**
	Cadmium	**<IIA (-0.71)**	**=IIB (0)**		? (0)
	Chromium(VI)	? (0)	**<IIIB2ii (-0.14)**	? (0)	
Cardiovascular Toxicity					
Effect of	Lead		**? (0)**	**=IIIA (0)**	NA
	Arsenic	**? (0)**		? (0)	NA
	Cadmium	**=IIIA (0)**	? (0)		NA
	Chromium(VI)	? (0)	**>IIIC (+0.10)**	? (0)	
Hematological Toxicity					
Effect of	Lead		**<IIIB (-0.23)**	**=IIC (0)**	? (0)
	Arsenic	**<IIIB (-0.23)**		**<IIIB (-0.23)**	**<IIIC2ii (-0.06)**
	Cadmium	**<IIIB (-0.23)**	**<IIIB (-0.23)**		? (0)
	Chromium(VI)	? (0)	**>IIIC (+0.10)**	? (0)	
Testicular Toxicity					
Effect of	Lead		NA	**>IIA (+0.71)**	? (0)
	Arsenic	? (0)		**<III2Bii (-0.14)**	**<IIIC2ii (-0.06)**
	Cadmium	**>IIA (+0.71)**	NA		? (0)
	Chromium(VI)	? (0)	NA	? (0)	

BINWOE scheme (with numerical weights in parentheses) condensed from ATSDR (2001a, 2001b):
DIRECTION: = additive (0); > greater than additive (+1): < less than additive (-1); ? indeterminate (0)
MECHANISTIC UNDERSTANDING:
I: direct and unambiguous mechanistic data to support direction of interaction (1.0);
II: mechanistic data on related compounds to infer mechanism(s) and likely direction (0.71);
III: mechanistic data do not clearly indicate direction of interaction (0.32).
TOXICOLOGIC SIGNIFICANCE:
A: direct demonstration of direction of interaction with toxicologically relevant endpoint (1.0);
B: toxicologic significance of interaction is inferred or has been demonstrated for related chemicals (0.71);
C: toxicologic significance of interaction is unclear (0.32).
MODIFYING FACTORS:
1: anticipated exposure duration and sequence (1.0);
2: different exposure duration or sequence (0.79);
a: *in vivo* data (1.0); b: *in vitro* data (0.79);
i: anticipated route of exposure (1.0); ii different route of exposure (0.79).

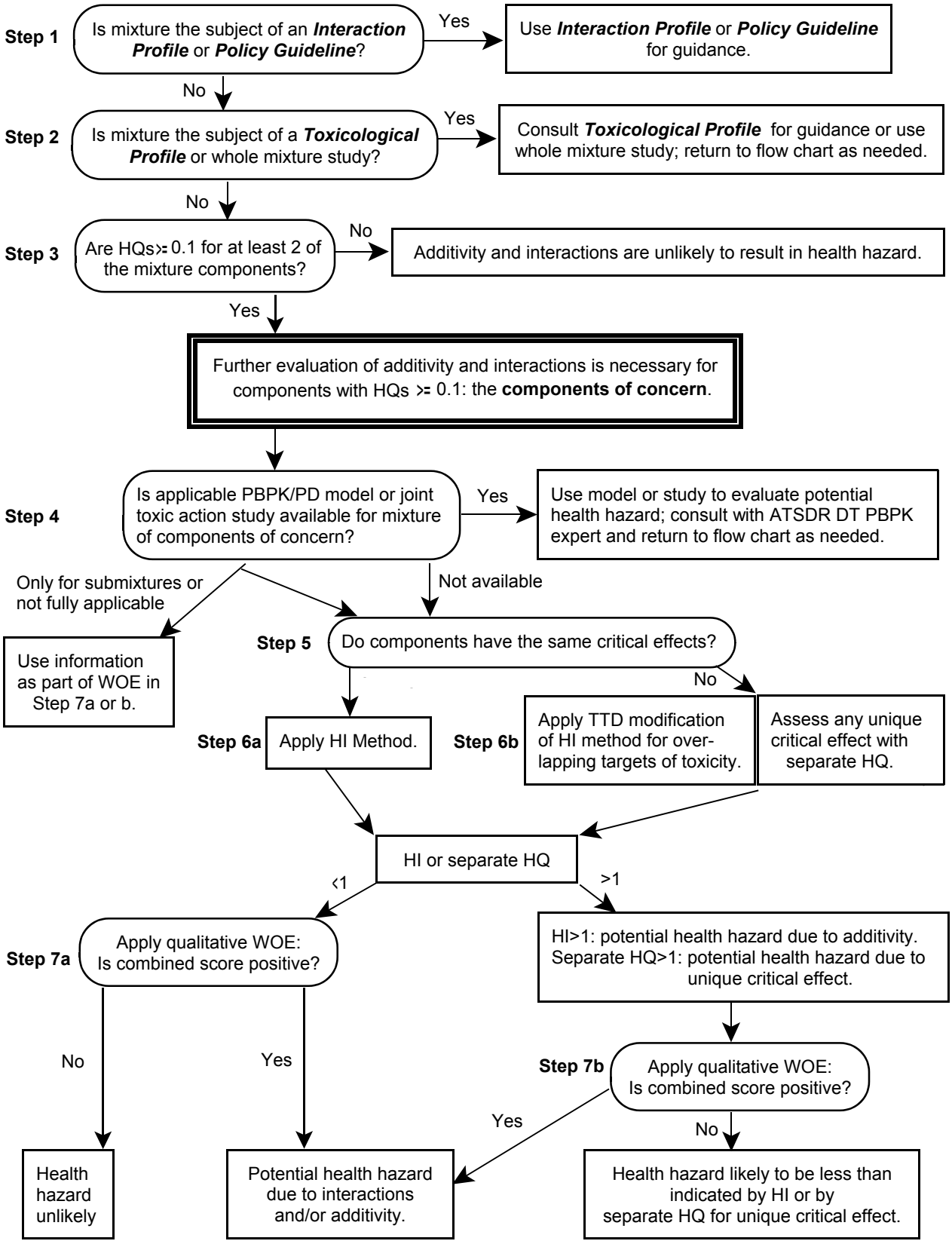


Figure E1: Exposure-Based Assessment of Joint Toxic Action of Chemical Mixtures: Non-Cancer Effects

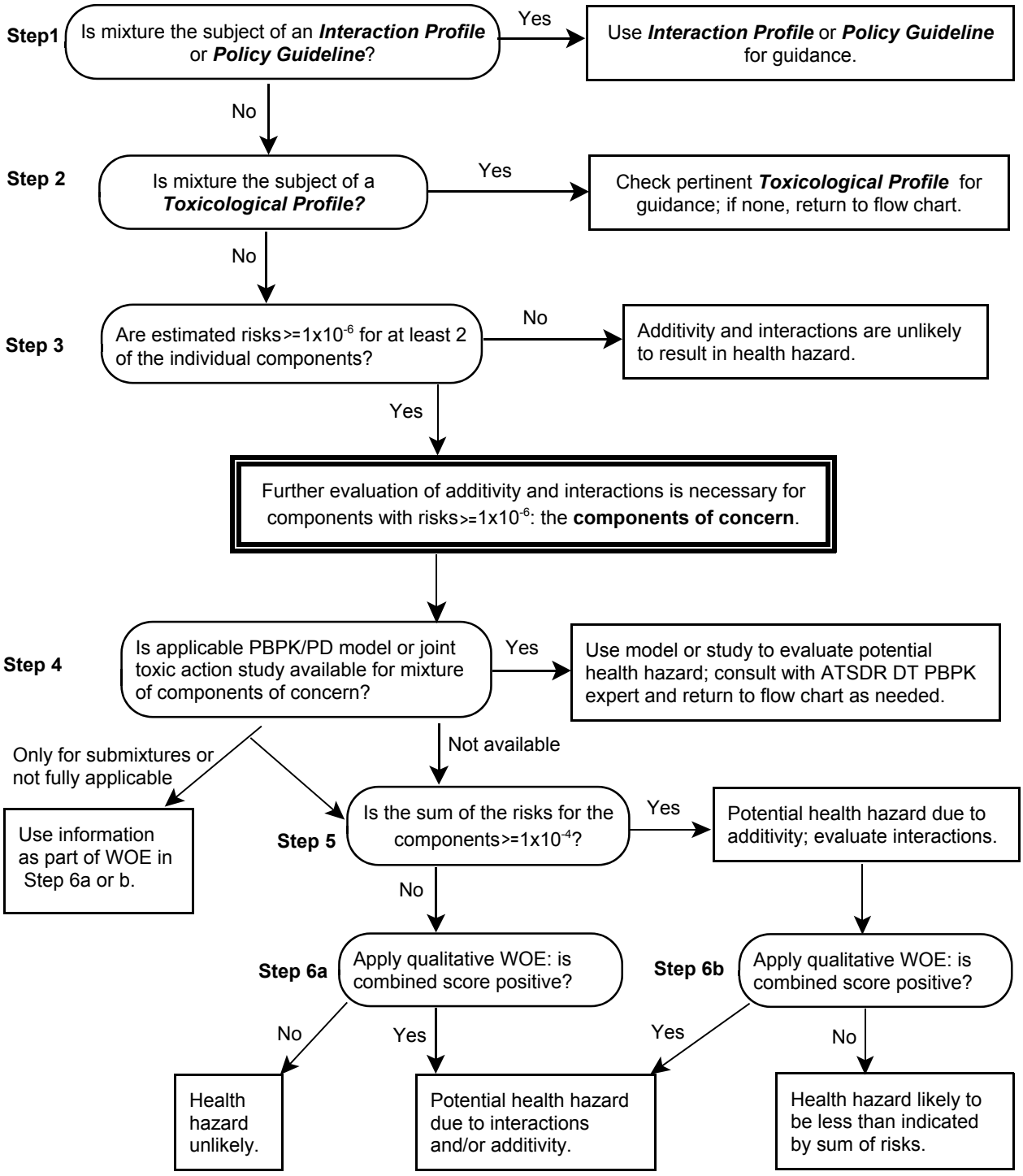


Figure E2: Exposure-Based of Joint Toxic Action of Chemical Mixtures: Cancer Effects

Appendix F

Cancer Incidence Analysis
Ringwood Mines/Landfill

Methods

Study Area and Population

The Ringwood Mines/Landfill study area for the evaluation of cancer incidence consisted of the entire population residing in the borough. In addition, a Focus Area of five census blocks (4006, 4007, 4008, 4009, and 4012) in close proximity to the contamination was evaluated separately (See Figure 1F). Enumeration of the municipal population and the Focus Area population was determined from Census Bureau data.

Cancer Case Ascertainment and Study Period

The New Jersey State Cancer Registry was used for the ascertainment of cancer cases. The cancer registry is a population-based cancer incidence registry covering the entire state of New Jersey. By law, all cases of newly diagnosed cancer are reportable to the registry except certain carcinomas of the skin. In addition, the registry has reporting agreements with the states of New York, Pennsylvania, Maryland, North Carolina, Delaware, and Florida. Information on New Jersey residents who are diagnosed in those states is supplied to the cancer registry. The registry has been in operation since October 1, 1978.

The study period for this investigation was January 1, 1979, through December 31, 2002. A "case" was defined as an individual who was diagnosed with a new primary malignant cancer during the study period while residing in Ringwood. Registry cases identified only through search of death records were excluded from this evaluation. Information on important cancer risk factors, such as genetics, personal behaviors (e.g., diet and smoking), or occupational history, is not available from the cancer registry.

Data Analysis

Analyses were completed for all malignant cancer types combined and for select cancer types for the entire borough of Ringwood. In addition to the entire borough, a portion of the city (the Focus Area) was evaluated separately. The select cancer types analyzed include: bladder, brain and central nervous system (CNS), female breast, colorectal, esophageal, pancreas, lung, leukemia, non-Hodgkin lymphoma, liver, bone, stomach, and kidney. These cancer types were evaluated because they represent cancer groupings that may be more sensitive to the effects of environmental exposures. Males and females were evaluated separately.

Standardized incidence ratios (SIRs) were used for the quantitative analysis of cancer incidence in the study areas (Kelsey, Thompson, and Evans 1986; Breslow and Day 1987). The SIR is calculated by dividing the observed number of cases (from the registry) by an expected number for the surveyed population over the time period reviewed.

The expected number was derived by multiplying a comparison population's age-sex-specific incidence rates and the study area age-sex-specific population figures. The comparison rates used to derive the expected number of cases were the New Jersey average annual incidence rates for 1979 to 1999. The Ringwood age-sex-specific population was determined from the 1980, 1990, and 2000 Census data, and the Focus Area age-sex-specific population was determined from the 2000 Census data (Census 1980, 1990, 2000). Eighteen age-specific population groups were used in the analysis.

Evaluation of the observed and expected numbers is accomplished by interpreting the ratio of these numbers. If the observed number of cases equals the expected number of cases, the SIR will equal one (1.0). An SIR less than one indicates that fewer cases are observed than expected. An SIR greater than one indicates that more cases than expected are observed.

Random fluctuations may account for some SIR deviations from 1.0. Statistical significance of deviations from SIR equal to 1.0 was evaluated using a 95% confidence interval (C.I.). The 95% C.I. was used to evaluate the probability that the SIR may be greater or less than 1.0 due to chance alone, and was based on the Poisson distribution (Breslow and Day 1987; Checkoway et al. 1989). If the confidence interval includes 1.0, then the estimated SIR is not considered to be statistically significantly different than 1.0.

Results

Table 1F presents the Ringwood population by age, race, and sex for the years 1980, 1990, and 2000. The borough population, all races combined, was stable from 1980 (12,625) to 1990 (12,623) and then dropped slightly in 2000 (12,396). The borough white population comprised more than 96% of the total population throughout the study period. The proportion of males in the borough was slightly higher than females. Census block population data were not available for 1980 or 1990. The Focus Area population comprised 3% of the total city population for 2000. Race in the Focus Area was proportionately different than in the 2000 Ringwood population: 13% white; 20% black; 47% American Indian and Alaska native; 14% multiple races; and 5% other or unknown race. The proportion of males in the Focus Area was similar to the total borough.

Table 2F presents the number of malignant incident cancer cases by sex and age group for Ringwood and the Focus Area. Individual races are not presented here because cancer registry race codes are not comparable to census data for race. For the town as a whole, a total of 1,003 cases were diagnosed in borough residents during the years 1979-2002. Of those cases, 22 resided in the Focus Area at the time of diagnosis and eight had insufficient address information to be geocoded to a specific area in Ringwood. Slightly more than half of the cases in the entire municipality were females. In the Focus Area, 59% of the cases were male. The proportion of cases diagnosed between 45 and 69 years of age was similar for the borough and the Focus Area. There was a lower proportion of cases in the borough (16%) diagnosed before the age of 45 than in the Focus Area. There was a higher proportion of cases 70 years of age or older in the entire municipality (31%) than in the Focus Area.

Table 3F presents cancer incidence by cancer type for all race-sex groups combined. The most frequently diagnosed cancer types for both Ringwood and the Focus Area include

colorectal, lung, breast, prostate, and bladder, representing between 54-58% of all incident cancers. The frequency of these cancer types is consistent with New Jersey statewide cancer incidence data.

Tables 4F and 5F present standardized incidence ratio (SIR) results by sex for all races combined. None of the SIRs for Ringwood (Table 4F) were statistically significantly high or statistically significantly low. In the Focus Area, one SIR, for lung cancer, was statistically significantly elevated for males (SIR=2.8; 95% CI=1.0, 6.1). Lung cancer in females was slightly lower than expected. None of the SIRs in the Focus Area were statistically significantly low.

Discussion

The purpose of this investigation was to evaluate cancer incidence in a population living relatively near to areas potentially contaminated by the Ringwood Mines/Landfill. For the entire borough of Ringwood, the occurrence of cancer (all sites combined) over the 24-year observation period was not higher than expected (based on average state rates). In the Focus Area, lung cancer in males was significantly higher than expected, while lung cancer in females was slightly lower than expected, although not statistically significant.

Cancer is a group of more than 100 different diseases (i.e., cancer types and subtypes), each with their own set of risk factors. The multifactorial nature of cancer etiology, where a given type of cancer may have more than one cause, complicates the evaluation of potential risk factors and specific disease outcomes. Contaminants at the Ringwood Mines/Landfill site include trichloroethylene (TCE), perchloroethylene (PCE), xylenes, arsenic, lead, mercury, chromium, polycyclic aromatic hydrocarbons (PAHs), and polychlorinated biphenyls (PCBs). Arsenic has been identified as a possible risk factor for certain cancer types, including lung cancer (ATSDR 2000). PAHs are considered a probable human carcinogen based on animal experiments and may increase the risk of developing cancer, especially lung and skin cancers (American Cancer Society 2004 and ATSDR 1995).

In the current analysis, the overall cancer incidence (all cancers combined) was not elevated. Lung cancer incidence was statistically significantly higher in males in the Focus Area, but not in females. Lung cancer incidence was lower than expected for males and females in Ringwood. Leukemia incidence was lower than expected for the entire borough, while there were no leukemia cases residing in the Focus Area at the time of diagnosis.

While there are multiple risk factors for lung cancer, tobacco smoking is considered the most important risk factor, estimated to account for more than 85% of all lung cancer cases (National Cancer Institute 1996). Other known risk factors for lung cancer include indoor exposure to radon and environmental tobacco smoke, occupational exposure to asbestos and other cancer-causing agents in the workplace (including radioactive ores; chemicals such as arsenic, vinyl chloride, nickel, chromates, coal products, mustard gas, and chloromethyl ethers; fuels such as gasoline; and diesel exhaust), and exposure to air pollution (American Cancer Society 2004).

A limitation of cancer studies of this type is the inability to assess past exposure levels in the population. Important information needed to assess a cause-effect relationship includes data on actual personal exposure to the contamination as well as other relevant risk factors over time; that is, who was exposed and who was not exposed and the magnitude of the exposure that did occur. Because personal exposure information does not exist, residential proximity to the contaminated site was used as a surrogate measure for potential past exposure. This was accomplished by analyzing separately the population living in the section of Ringwood closest to the location of the Ringwood Mines/Landfill. While proximity to the contamination may be a reasonable surrogate for past potential exposures, it could result in misclassifying some of the study population as exposed. Additionally, the length of residence of each case is unknown, thereby potentially adding to exposure misclassification. The consequence of exposure misclassification would be to bias the results toward not finding an association (i.e., no exposure-health outcome relationship).

Another interpretation problem is that cancer is a chronic disease that takes many years after exposure to manifest as clinical disease. The information supplied by the cancer registry provides only an address at time of diagnosis for each case. No information is available on length of time an individual may have lived at the address before diagnosis. It is possible that some cases are new, short-term residents with little or no exposure to the site. Furthermore, former residents who moved out of the study area just prior to diagnosis are not included in this analysis. Population mobility cannot be accounted for in this analysis.

Additionally, when researchers independently examine statistical associations for a large number of comparisons, it is likely that some number of statistically elevated or low SIRs will occur by chance alone. While it is possible to statistically correct for this concern, it is controversial whether such corrections are needed. In this analysis, confidence intervals are presented without adjustment for multiple comparisons.

In small populations, such as the Focus Area, the number of expected cases of all but the most common cancers is very small, generally much less than one case. Consequently, very large SIRs may result from a small number of observed cases (perhaps one or two). Because of the considerable statistical uncertainty at these low numbers of observed and expected cases, it is unlikely that statistically significant increases in the rarer cancer types will be detected in small populations.

The approach utilized for this descriptive cancer investigation was “census” based, where the entire population of Ringwood and the state of New Jersey were reviewed in order to calculate age-standardized incidence rate ratios for the study area. This “census” approach (ecologic design) is a practical surveillance or screening method for cancer incidence. Although this approach is well suited for providing a picture of cancer incidence in the specific localities, cause-effect relationships cannot be evaluated. Important information on potential risk factors (such as genetics, behaviors, environmental factors, occupation, etc.) that might explain the results, were not available for analysis using this type of study design.

Conclusion

The overall cancer incidence (all cancers combined) was not elevated in the Focus Area. Lung cancer in males was statistically significantly higher than expected while lung cancer in females was slightly lower than expected in the Focus Area. Since the prevalence of tobacco smoking is not available for these cases, it is unknown what influence this important risk factor, or other behaviors, may have played. Given that lung cancer incidence in females is lower than expected, the current analysis provides little evidence that the rate of cancer incidence in the Focus Area population is due to potential exposure to Ringwood Mines contamination.

References

American Cancer Society. Cancer facts and figures. Located on-line at www.cancer.org: 2004.

American Cancer Society. What are the Risk Factors for Lung Cancer? Located on-line at www.cancer.org: 2004.

Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Arsenic (Update). Prepared by Syracuse Research Corporation. Atlanta: 2000.

Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for polycyclic aromatic hydrocarbons. Prepared by Research Triangle Institute. Atlanta: 1995.

Breslow NE, Day NE. Statistical Methods in Cancer Research: Vol II-The Design and Analysis of Cohort Studies, E. Heseltine, ed. In IARC Scientific Publication No. 82. Lyon: International Agency for Research on Cancer; 1987.

Checkoway H, Pearce NE, Crawford-Brown DJ. Research Methods in Occupational Epidemiology, B. MacMahon, ed. In Monographs in Epidemiology and Biostatistics Vol. 13. Oxford: Oxford University Press; 1989.

Kelsey JL, Thompson WD, Evans AS. Methods in Observational Epidemiology, B. MacMahon, ed. In Monographs in Epidemiology and Biostatistics Vol. 10. Oxford: Oxford University Press; 1986.

National Cancer Institute. Cancer Rates and Risks. NIH Publication No. 96-691; 1996.

New Jersey Department of Health and Senior Services (NJDHSS). Hazardous Substance Fact Sheet – benzene. Trenton; 1994.

Census 2000. Summary file 1, New Jersey prepared by the U.S. Census Bureau, 2001.

Census 1990. Summary file 1, New Jersey prepared by the U.S. Census Bureau, 1991.

Census 1980. Summary file 1, New Jersey prepared by the U.S. Census Bureau, 1981.

Appendix Tables and Figure

Table 1F. Ringwood Population by Race and Sex, Census Bureau Data.

Area	1980	1990	2000
Entire Municipality			
Total	12,625	12,623	12,396
Sex			
Males	6,402	6,362	6,201
Females	6,223	6,261	6,195
Race*			
White	12,088	12,043	11,636
Black	252	227	199
American Indian and Alaska Native	-----	123	179
Asian or Pacific Islander	-----	176	149
Multiple Races	-----	-----	150
Other/Unknown	285	54	83
Focus Area+			
Total	-----	-----	328
Sex	-----	-----	
Males			168
Females			160
Race	-----	-----	
White			41
Black			67
American Indian and Alaska Native			154
Asian or Pacific Islander			<5
Multiple Races			47
Other/Unknown			15

* Multiple race reporting began in the 2000 census.

+ Census blocks 4006, 4007, 4008, 4009, and 4012; 1980 and 1990 population unavailable by census blocks.

Table 2F. Ringwood Malignant Cancer Incidence* (1979-2002) by Study Area, Select Demographic Characteristics.

Demographic Characteristics	Entire Municipality	Focus Area
Total Cases	1,003	22
Sex		
Male	495	13
Female	508	9
Age at diagnosis		
0 – 19	14	<5
20 – 44	142	6
45 – 69	537	12
70+	310	<5

* Data are from the New Jersey State Cancer Registry, New Jersey Department of Health and Senior Services.

Table 3F. Ringwood Malignant Cancer Incidence* (1979-2002) by Cancer Type and Study Area, All Races Combined.

Cancer Type	Entire Municipality	Focus Area
Oralpharynx	20	0
Esophagus	8	0
Stomach	15	0
Small Intestine	<5	0
Colorectal	119	<5
Liver	9	0
Pancreas	15	0
Other Digestive	12	<5
Lung	121	7
Other Respiratory	6	0
Bones and Joints	<5	<5
Soft Tissue	<5	0
Skin	55	0
Breast	165	<5
Cervix	10	<5
Uterus	40	0
Ovary	28	0
Other Female Genital	6	0
Prostate	125	<5
Other Male Genital	14	0
Bladder	54	<5
Kidney	24	0
Other Urinary	<5	0
Eye	<5	0
Brain and Central Nervous System	17	0
Endocrine	19	<5
Hodgkin Disease	11	<5
Non-Hodgkin Lymphoma	39	<5
Myeloma	7	0
Leukemia	21	0
Miscellaneous/Other	25	0
Mesothelioma	5	0

* Data are from the New Jersey State Cancer Registry, New Jersey Department of Health and Senior Services.

Table 4F. Ringwood Malignant Cancer Incidence (1979-2002), SIR Analysis by Cancer Type and Sex, All Races Combined.

Cancer Type	Sex	Observed	Expected[1]	SIR	95% CI
All Cancers Combined	Male	495	519.5	0.95	0.87 – 1.04
	Female	508	487.1	1.04	0.95 – 1.14
Bladder	Male	45	37.2	1.21	0.88 – 1.62
	Female	9	11.7	0.77	0.35 – 1.46
Brain/CNS	Male	7	9.7	0.72	0.29 – 1.49
	Female	10	7.1	1.41	0.67 – 2.59
Colorectal	Male	61	70.8	0.86	0.66 – 1.11
	Female	58	58.3	1.00	0.76 – 1.29
Esophageal	Male	5	8.6	0.58	0.19 – 1.35
	Female	<5	NR	1.19	0.24 – 3.47
Kidney	Male	12	15.5	0.78	0.40 – 1.36
	Female	12	8.2	1.47	0.76 – 2.57
Leukemia	Male	12	15.0	0.80	0.41 – 1.40
	Female	9	10.1	0.89	0.41 – 1.69
Liver	Male	5	4.4	1.13	0.36 – 2.63
	Female	<5	NR	2.36	0.63 – 6.04
NHL	Male	22	21.9	1.00	0.63 – 1.52
	Female	17	16.2	1.05	0.61 – 1.69
Stomach	Male	10	13.9	0.72	0.34 – 1.32
	Female	5	7.4	0.68	0.22 – 1.58
Lung	Male	79	89.9	0.88	0.70 – 1.10
	Female	42	49.6	0.85	0.61 – 1.14
Bone and Joint	Male	<5	NR	1.27	0.14 – 4.59
	Female	<5	NR	1.67	0.19 – 6.04
Breast	Male	<5	NR	0.75	0.01 – 4.17
	Female	164	158.7	1.03	0.88 – 1.20
Pancreas	Male	6	11.4	0.53	0.19 – 1.15
	Female	7	9.7	0.93	0.42 – 1.76

[1] Note: NR= not reported because observed <5.

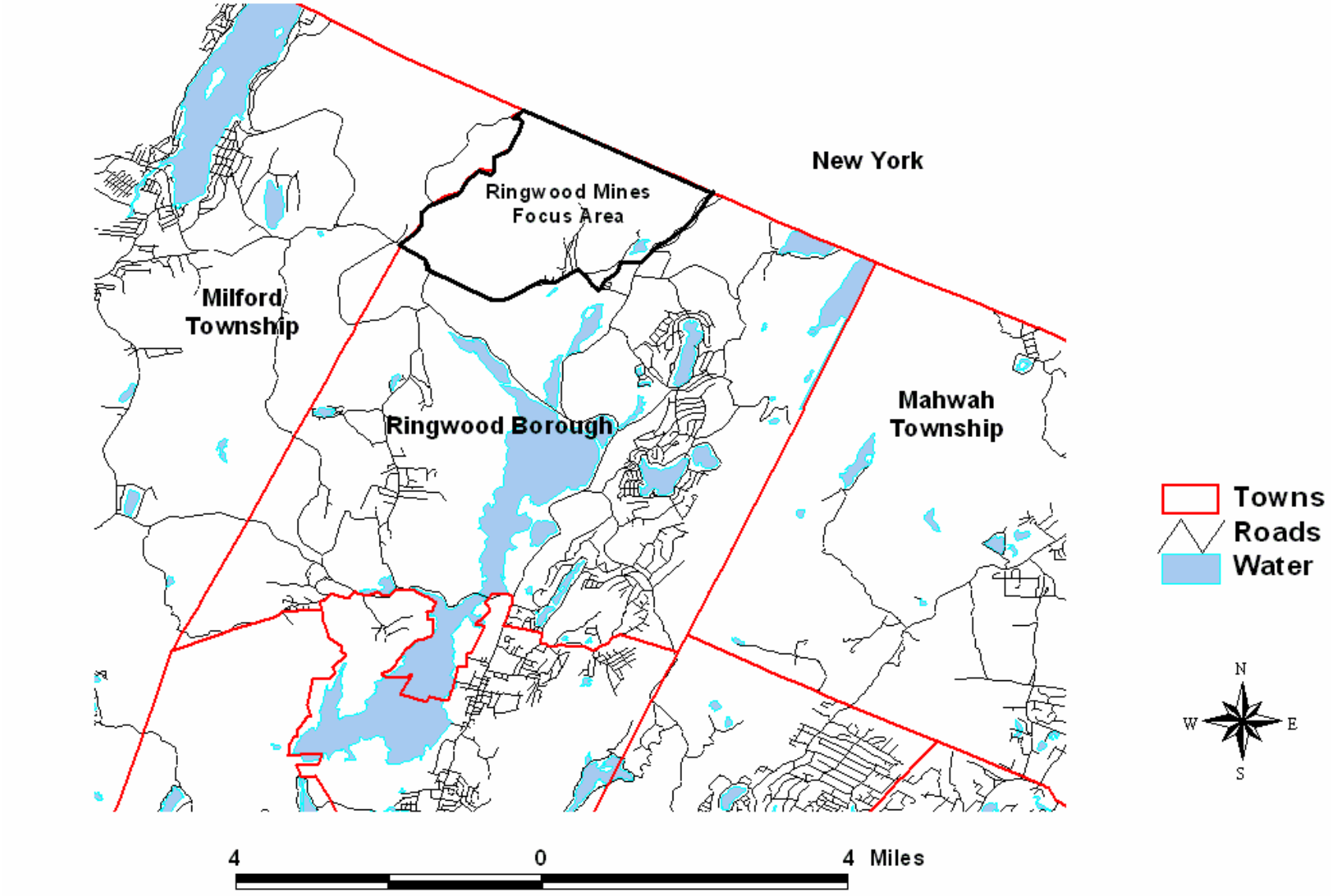
Data are from the New Jersey State Cancer Registry, New Jersey Department of Health and Senior Services.

Table 5F. Ringwood Focus Area Malignant Cancer Incidence (1979-2002), SIR Analysis by Cancer Type and Sex, All Races Combined.

Cancer Type	Sex	Observed	Expected	SIR[1]		95% CI
All Cancers Combined	Male	13	12.2	1.07		0.57 – 1.82
	Female	9	10.8	0.83		0.38 – 1.58
Bladder	Male	<5	NR	2.36		0.27 – 8.54
	Female	0	0.2	0		–
Brain/CNS	Male	0	0.2	0		–
	Female	0	0.2	0		–
Colorectal	Male	<5	NR	0.62		0.01 – 3.43
	Female	0	1.2	0		–
Esophageal	Male	0	0.2	0		–
	Female	0	0.1	0		–
Kidney	Male	0	0.4	0		–
	Female	0	0.2	0		–
Leukemia	Male	0	0.4	0		–
	Female	0	0.2	0		–
Liver	Male	0	0.1	0		–
	Female	0	0.0	0		–
NHL	Male	<5	NR	1.94		0.03 – 10.8
	Female	<5	NR	2.83		0.04 – 15.8
Stomach	Male	0	0.3	0		–
	Female	0	0.1	0		–
Lung	Male	6	2.2	2.79	*	1.02 – 6.08
	Female	<5	NR	0.89		0.01 – 4.96
Bone and Joint	Male	<5	NR	23.9		0.31 – 133
	Female	0	0.0	0		–
Breast	Male	0	0.0	0		–
	Female	<5	NR	0.56		0.06 – 2.01
Pancreas	Male	0	0.3	0		–
	Female	0	0.2	0		–

[1] Note: *= statistically high, **= statistically low, NR= not reported because observed <5.
Data are from the New Jersey State Cancer Registry, New Jersey Department of Health and Senior Services

Figure 1F: Ringwood Study Area



Appendix G

Summary of ATSDR Conclusion Categories

Category	Definition
1: Urgent Public Health Hazard	Applies to sites that have certain physical hazards or evidence of short-term (less than 1 year), site-related exposure to hazardous substances that could result in adverse health effects and require quick intervention to stop people from being exposed.
2: Public Health Hazard	Applies to sites that have certain physical hazards or evidence of chronic, site-related exposure to hazardous substances that could result in adverse health effects.
3: Indeterminate Public Health Hazard	Applies to sites where critical information is lacking (missing or has not yet been gathered) to support a judgment regarding the level of public health hazard.
4: No Apparent Public Health Hazard	Applies to sites where exposure to site-related chemicals might have occurred in the past or is still occurring, but the exposures are not at levels expected to cause adverse health effects.
5: No Public Health Hazard	Applies to sites where no exposure to site-related hazardous substances exists.

Appendix H

ATSDR Glossary of Terms

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency with headquarters in Atlanta, Georgia, and 10 regional offices in the United States. ATSDR's mission is to serve the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and diseases related to toxic substances. ATSDR is not a regulatory agency, unlike the U.S. Environmental Protection Agency (EPA), which is the federal agency that develops and enforces environmental laws to protect the environment and human health. This glossary defines words used by ATSDR in communications with the public. It is not a complete dictionary of environmental health terms. If you have questions or comments, call ATSDR's toll-free telephone number, 1-888-42-ATSDR (1-888-422-8737).

General Terms

Absorption
The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

Acute
Occurring over a short time [compare with chronic].

Acute exposure
Contact with a substance that occurs once or for only a short time (up to 14 days) [compare with intermediate duration exposure and chronic exposure].

Additive effect
A biologic response to exposure to multiple substances that equals the sum of responses of all the individual substances added together [compare with antagonistic effect and synergistic effect].

Adverse health effect
A change in body function or cell structure that might lead to disease or health problems

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

Anaerobic
Requiring the absence of oxygen [compare with aerobic].

Analyte
A substance measured in the laboratory. A chemical for which a sample (such as water, air, or blood) is tested in a laboratory. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

Analytic epidemiologic study
A study that evaluates the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Antagonistic effect
A biologic response to exposure to multiple substances that is less than would be expected if the known effects of the individual substances were added together [compare with additive effect and synergistic effect].

Background level
An average or expected amount of a substance or radioactive material in a specific environment, or typical amounts of substances that occur naturally in an environment.

Biodegradation
Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Biologic indicators of exposure study
A study that uses (a) biomedical testing or (b) the measurement of a substance [an analyte], its metabolite, or another marker of exposure in human body fluids or tissues to confirm human exposure to a hazardous substance [also see exposure investigation].

Biologic monitoring
Measuring hazardous substances in biologic materials (such as blood, hair, urine, or breath) to determine whether exposure has occurred. A blood test for lead is an example of biologic monitoring.

Biologic uptake
The transfer of substances from the environment to plants, animals, and humans.

Biomedical testing
Testing of persons to find out whether a change in a body function might have occurred because of exposure to a hazardous substance.

Biota
Plants and animals in an environment. Some of these plants and animals might be sources of food, clothing, or medicines for people.

Body burden
The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.

CAP [see Community Assistance Panel.]

Cancer
Any one of a group of diseases that occur when cells in the body become abnormal and grow or multiply out of control.

Cancer risk
A theoretical risk for getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

Carcinogen
A substance that causes cancer.

Case study
A medical or epidemiologic evaluation of one person or a small group of people to gather information about specific health conditions and past exposures.

Case-control study
A study that compares exposures of people who have a disease or condition (cases) with people who do not have the disease or condition (controls). Exposures that are more common among the cases may be considered as possible risk factors for the disease.

CAS registry number
A unique number assigned to a substance or mixture by the American Chemical Society Abstracts Service.

Central nervous system
The part of the nervous system that consists of the brain and the spinal cord.

CERCLA [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980]

Chronic
Occurring over a long time [compare with acute].

Chronic exposure
Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure]

Cluster investigation
A review of an unusual number, real or perceived, of health events (for example, reports of cancer) grouped together in time and location. Cluster investigations are designed to confirm case reports; determine whether they represent an unusual disease occurrence; and, if possible, explore possible causes and contributing environmental factors.

Community Assistance Panel (CAP)
A group of people from a community and from health and environmental agencies who work with ATSDR to resolve issues and problems related to hazardous substances in the community. CAP members work with ATSDR to gather and review community health concerns, provide information on how people might have been or might now be exposed to hazardous substances, and inform ATSDR on ways to involve the community in its activities.

Comparison value (CV)
Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

Completed exposure pathway [see exposure pathway].

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)
CERCLA, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances. This law was later amended by the Superfund Amendments and Reauthorization Act (SARA).

Concentration
The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

Contaminant
A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

Delayed health effect
A disease or an injury that happens as a result of exposures that might have occurred in the past.

Dermal
Referring to the skin. For example, dermal absorption means passing through the skin.

Dermal contact
Contact with (touching) the skin [see route of exposure].

Descriptive epidemiology
The study of the amount and distribution of a disease in a specified population by person, place, and time.

Detection limit
The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

Disease prevention
Measures used to prevent a disease or reduce its severity.

Disease registry
A system of ongoing registration of all cases of a particular disease or health condition in a defined population.

DOD
United States Department of Defense.

DOE
United States Department of Energy.

Dose (for chemicals that are not radioactive)
The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Dose (for radioactive chemicals)
The radiation dose is the amount of energy from radiation that is actually absorbed by the body. This is not the same as measurements of the amount of radiation in the environment.

Dose-response relationship
The relationship between the amount of exposure [dose] to a substance and the resulting changes in body function or health (response).

Environmental media
Soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

Environmental media and transport mechanism
Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The environmental media and transport mechanism is the second part of an exposure pathway.

EPA
United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

Epidemiology
The study of the distribution and determinants of disease or health status in a population; the study of the occurrence and causes of health effects in humans.

Exposure
Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure].

Exposure assessment
The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Exposure-dose reconstruction
A method of estimating the amount of people's past exposure to hazardous substances. Computer and approximation methods are used when past information is limited, not available, or missing.

Exposure investigation
The collection and analysis of site-specific information and biologic tests (when appropriate) to determine whether people have been exposed to hazardous substances.

Exposure pathway
The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as an abandoned business); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

Exposure registry
A system of ongoing followup of people who have had documented environmental exposures.

Feasibility study
A study by EPA to determine the best way to clean up environmental contamination. A number of factors are considered, including health risk, costs, and what methods will work well.

Geographic information system (GIS)
A mapping system that uses computers to collect, store, manipulate, analyze, and display data. For example, GIS can show the concentration of a contaminant within a community in relation to points of reference such as streets and homes.

Grand rounds
Training sessions for physicians and other health care providers about health topics.

Groundwater
Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with surface water].

Half-life ($t_{½}$)
The time it takes for half the original amount of a substance to disappear. In the environment, the half-life is the time it takes for half the original amount of a substance to disappear when it is changed to another chemical by bacteria, fungi, sunlight, or other chemical processes. In the human body, the half-life is the time it takes for half the original amount of the substance to disappear, either by being changed to another substance or by leaving the body. In the case of radioactive material, the half life is the amount of time necessary for one half the initial number of radioactive atoms to change or transform into another atom (that is normally not radioactive). After two half lives, 25% of the original number of radioactive atoms remain.

Hazard
A source of potential harm from past, current, or future exposures.

Hazardous Substance Release and Health Effects Database (HazDat)
The scientific and administrative database system developed by ATSDR to manage data collection, retrieval, and analysis of site-specific information on hazardous substances, community health concerns, and public health activities.

Hazardous waste
Potentially harmful substances that have been released or discarded into the environment.

Health consultation
A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical [compare with public health assessment].

Health education
Programs designed with a community to help it know about health risks and how to reduce these risks.

Health investigation
The collection and evaluation of information about the health of community residents. This information is used to describe or count the occurrence of a disease, symptom, or clinical measure and to evaluate the possible association between the occurrence and exposure to hazardous substances.

Health promotion
The process of enabling people to increase control over, and to improve, their health.

Health statistics review
The analysis of existing health information (i.e., from death certificates, birth defects registries, and cancer registries) to determine if there is excess disease in a specific population, geographic area, and time period. A health statistics review is a descriptive epidemiologic study.

Indeterminate public health hazard
The category used in ATSDR's public health assessment documents when a professional judgment about the level of health hazard cannot be made because information critical to such a decision is lacking.

Incidence
The number of new cases of disease in a defined population over a specific time period [contrast with prevalence].

Ingestion
The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].

Inhalation
The act of breathing. A hazardous substance can enter the body this way [see route of exposure].

Intermediate duration exposure
Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].

In vitro
In an artificial environment outside a living organism or body. For example, some toxicity testing is done on cell cultures or slices of tissue grown in the laboratory, rather than on a living animal [compare with in vivo].

In vivo
Within a living organism or body. For example, some toxicity testing is done on whole animals, such as rats or mice [compare with in vitro].

Lowest-observed-adverse-effect level (LOAEL)
The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Medical monitoring
A set of medical tests and physical exams specifically designed to evaluate whether an individual's exposure could negatively affect that person's health.

Metabolism
The conversion or breakdown of a substance from one form to another by a living organism.

Metabolite
Any product of metabolism.

mg/kg
Milligram per kilogram.

mg/cm^2
Milligram per square centimeter (of a surface).

mg/m^3
Milligram per cubic meter; a measure of the concentration of a chemical in a known volume (a cubic meter) of air, soil, or water.

Migration
Moving from one location to another.

Minimal risk level (MRL)
An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].

Morbidity
State of being ill or diseased. Morbidity is the occurrence of a disease or condition that alters health and quality of life.

Mortality
Death. Usually the cause (a specific disease, a condition, or an injury) is stated.

Mutagen
A substance that causes mutations (genetic damage).

Mutation
A change (damage) to the DNA, genes, or chromosomes of living organisms.

National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or NPL)
EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

National Toxicology Program (NTP)
Part of the Department of Health and Human Services. NTP develops and carries out tests to predict whether a chemical will cause harm to humans.

No apparent public health hazard
A category used in ATSDR's public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

No-observed-adverse-effect level (NOAEL)
The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

No public health hazard
A category used in ATSDR's public health assessment documents for sites where people have never and will never come into contact with harmful amounts of site-related substances.

NPL [see National Priorities List for Uncontrolled Hazardous Waste Sites]

Physiologically based pharmacokinetic model (PBPK model)
A computer model that describes what happens to a chemical in the body. This model describes how the chemical gets into the body, where it goes in the body, how it is changed by the body, and how it leaves the body.

Pica
A craving to eat nonfood items, such as dirt, paint chips, and clay. Some children exhibit pica-related behavior.

Plume
A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.

Point of exposure
The place where someone can come into contact with a substance present in the environment [see exposure pathway].

Population
A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

Potentially responsible party (PRP)
A company, government, or person legally responsible for cleaning up the pollution at a hazardous waste site under Superfund. There may be more than one PRP for a particular site.

ppb
Parts per billion.

ppm
Parts per million.

Prevalence
The number of existing disease cases in a defined population during a specific time period [contrast with incidence].

Prevalence survey
The measure of the current level of disease(s) or symptoms and exposures through a questionnaire that collects self-reported information from a defined population.

Prevention
Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

Public availability session
An informal, drop-by meeting at which community members can meet one-on-one with ATSDR staff members to discuss health and site-related concerns.

Public comment period
An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

Public health action
A list of steps to protect public health.

Public health advisory
A statement made by ATSDR to EPA or a state regulatory agency that a release of hazardous substances poses an immediate threat to human health. The advisory includes recommended measures to reduce exposure and reduce the threat to human health.

Public health assessment (PHA)
An ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health [compare with health consultation].

Public health hazard
A category used in ATSDR's public health assessments for sites that pose a public health hazard because of long-term exposures (greater than 1 year) to sufficiently high levels of hazardous substances or radionuclides that could result in harmful health effects.

Public health hazard categories
Public health hazard categories are statements about whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might be appropriate for each site. The five public health hazard categories are no public health hazard, no apparent public health hazard, indeterminate public health hazard, public health hazard, and urgent public health hazard.

Public health statement
The first chapter of an ATSDR toxicological profile. The public health statement is a summary written in words that are easy to understand. The public health statement explains how people might be exposed to a specific substance and describes the known health effects of that substance.

Public health surveillance
The ongoing, systematic collection, analysis, and interpretation of health data. This activity also involves timely dissemination of the data and use for public health programs.

Public meeting
A public forum with community members for communication about a site.

Radioisotope
An unstable or radioactive isotope (form) of an element that can change into another element by giving off radiation.

Radionuclide
Any radioactive isotope (form) of any element.

RCRA [see Resource Conservation and Recovery Act (1976, 1984)]

Receptor population
People who could come into contact with hazardous substances [see exposure pathway].

Reference dose (RfD)
An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

Registry
A systematic collection of information on persons exposed to a specific substance or having specific diseases [see exposure registry and disease registry].

Remedial investigation
The CERCLA process of determining the type and extent of hazardous material contamination at a site.

Resource Conservation and Recovery Act (1976, 1984) (RCRA)
This Act regulates management and disposal of hazardous wastes currently generated, treated, stored, disposed of, or distributed.

RFA
RCRA Facility Assessment. An assessment required by RCRA to identify potential and actual releases of hazardous chemicals.

RfD [see reference dose]

Risk
The probability that something will cause injury or harm.

Risk reduction
Actions that can decrease the likelihood that individuals, groups, or communities will experience disease or other health conditions.

Risk communication
The exchange of information to increase understanding of health risks.

Route of exposure
The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].

Safety factor [see uncertainty factor]

SARA [see Superfund Amendments and Reauthorization Act]

Sample
A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger population [see population]. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

Sample size
The number of units chosen from a population or an environment.

Solvent
A liquid capable of dissolving or dispersing another substance (for example, acetone or mineral spirits).

Source of contamination
The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an exposure pathway.

Special populations
People who might be more sensitive or susceptible to exposure to hazardous substances because of factors such as age, occupation, sex, or behaviors (for example, cigarette smoking). Children, pregnant women, and older people are often considered special populations.

Stakeholder
A person, group, or community who has an interest in activities at a hazardous waste site.

Statistics
A branch of mathematics that deals with collecting, reviewing, summarizing, and interpreting data or information. Statistics are used to determine whether differences between study groups are meaningful.

Substance
A chemical.

Substance-specific applied research
A program of research designed to fill important data needs for specific hazardous substances identified in ATSDR's toxicological profiles. Filling these data needs would allow more accurate assessment of human risks from specific substances contaminating the environment. This research might include human studies or laboratory experiments to determine health effects resulting from exposure to a given hazardous substance.

Superfund [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and Superfund Amendments and Reauthorization Act (SARA)

Superfund Amendments and Reauthorization Act (SARA)
In 1986, SARA amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects from substance exposures at hazardous waste sites and to perform activities including health education, health studies, surveillance, health consultations, and toxicological profiles.

Surface water
Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].

Surveillance [see public health surveillance]

Survey
A systematic collection of information or data. A survey can be conducted to collect information from a group of people or from the environment. Surveys of a group of people can be conducted by telephone, by mail, or in person. Some surveys are done by interviewing a group of people [see prevalence survey].

Synergistic effect
A biologic response to multiple substances where one substance worsens the effect of another substance. The combined effect of the substances acting together is greater than the sum of the effects of the substances acting by themselves [see additive effect and antagonistic effect].

Teratogen
A substance that causes defects in development between conception and birth. A teratogen is a substance that causes a structural or functional birth defect.

Toxic agent
Chemical or physical (for example, radiation, heat, cold, microwaves) agents that, under certain circumstances of exposure, can cause harmful effects to living organisms.

Toxicological profile
An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

Toxicology
The study of the harmful effects of substances on humans or animals.

Tumor
An abnormal mass of tissue that results from excessive cell division that is uncontrolled and progressive. Tumors perform no useful body function. Tumors can be either benign (not cancer) or malignant (cancer).

Uncertainty factor
Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a safety factor].

Urgent public health hazard
A category used in ATSDR's public health assessments for sites where short-term exposures (less than 1 year) to hazardous substances or conditions could result in harmful health effects that require rapid intervention.

Volatile organic compounds (VOCs)
Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.

Other glossaries and dictionaries:
Environmental Protection Agency (http://www.epa.gov/OCEPAterms/)

National Center for Environmental Health (CDC)
(http://www.cdc.gov/nceh/dls/report/glossary.htm)

National Library of Medicine (NIH)
(http://www.nlm.nih.gov/medlineplus/mplusdictionary.html)

For more information on the work of ATSDR, please contact:

Office of Policy and External Affairs
Agency for Toxic Substances and Disease Registry
1600 Clifton Road, N.E. (MS E-60)
Atlanta, GA 30333
Telephone: (404) 498-0080