

## **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 immersed 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.

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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

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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:

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- (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.

<b>Activity</b>	<b>Anticipated or Actual Start</b>	<b>Anticipated Completion</b>
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<sup>3</sup>. 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-cd)pyrene, 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 (4.80), 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):

<b>Lead</b>	<b>Arsenic</b>	<b>Cadmium</b>	<b>Chromium (IV)</b>	<b>Aroclor</b>	<b>Antimony</b>
<i>Neurological</i> <b>Hematological</b> <b>Cardiovascular</b> Renal Testicular	<i>Dermal lesions</i> <b>Cardiovascular</b> <b>Hematological</b> Renal <b>Neurological</b> <b>Cancer</b>	<i>Renal (proteinuria)</i> Cardiovascular Hematological Hepatic Neurological Testicular	Hematological Hepatic Renal Neurological Testicular	<i>Immunological</i> <i>Neurological</i> Cancer Dermal Hepatic Hematological	<i>Lifespan</i>

<sup>a</sup>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:

<b>Health Effect</b>	<b>Impact of interaction</b>
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  $1 \times 10^{-6}$  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
<b>Paint Sludge</b>						
Bis(2-ethylhexyl) phthalate	0.00048	0.02	0.024	480.7	NA <sup>1</sup>	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 <sup>2</sup>	59	10	5.9		NA	NA
<b>Soil</b>						
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
<b>Sediment</b>						
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.)**

<b>Contaminant</b>	<b>Child Exposure Dose (mg/kg/day)</b>	<b>Health Guideline CV (mg/kg/day)</b>	<b>Hazard Quotient</b>	<b>HI</b>	<b>NOAEL (mg/kg/day)</b>	<b>Dose/NOAEL</b>
<b>Surface Water</b>						
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

<sup>1</sup>Not available; <sup>2</sup>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
<b>Paint Sludge</b>						
Bis(2-ethylhexyl) phthalate	5.54 x10 <sup>-5</sup>	0.02	0.0027	57.37	NA <sup>1</sup>	NA
Aroclor 1248/1254	6.88 x10 <sup>-7</sup>	0.00002	0.034		0.005	0.00013
Antimony	2.16 x10 <sup>-2</sup>	0.0004	54		0.003	7.2
Arsenic	1.99 x10 <sup>-6</sup>	0.0003	0.0066		0.0008	0.0024
Cadmium	4.83 x10 <sup>-6</sup>	0.0002	0.024		0.005	0.001
Chromium	7.53 x10 <sup>-4</sup>	0.003	0.25		2.5	0.0003
Copper	2.59 x10 <sup>-4</sup>	0.04	0.0064		NA	NA
Lead <sup>2</sup>	30.5	10	3.05		NA	NA
<b>Soil</b>						
Benzene	1.56 x10 <sup>-5</sup>	0.004	0.004	0.12		
Benzo[a]pyrene	6.4 x10 <sup>-8</sup>					
Arsenic	9.3 x10 <sup>-7</sup>	0.0003	0.003			
Lead	6 x10 <sup>-5</sup>					
Thallium	8.72 x10 <sup>-6</sup>	0.00007	0.12			
<b>Sediment</b>						
Benzo[a]pyrene	2.08 x10 <sup>-7</sup>	NA	NA	0.076		
Arsenic	4.19 x10 <sup>-6</sup>	0.0003	0.014			
Thallium	4.34 x10 <sup>-6</sup>	0.00007	0.062			

**Table E2: (Cont'd.)**

<b>Contaminant</b>	<b>Adult Exposure Dose (mg/kg/day)</b>	<b>Health Guideline CV (mg/kg/day)</b>	<b>Hazard Quotient</b>	<b>HI</b>	<b>NOAEL (mg/kg/day)</b>	<b>Dose/NOAEL</b>
<b>Surface Water</b>						
Benzene	4.0 x10 <sup>-5</sup>	0.004	0.01	1.57	NA	NA
1,2-Dichloropropane	3.4 x10 <sup>-4</sup>	0.09	0.0037		NA	NA
Arsenic	4.7 x10 <sup>-4</sup>	0.0003	1.56		0.009	0.05
Lead	3.0 x10 <sup>-3</sup>	NA	NA		NA	NA
Mercury	7.0 x10 <sup>-5</sup>	NA	NA		NA	NA

<sup>1</sup>Not available; <sup>2</sup>Based on blood lead levels in µg/dL

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

<b>Child</b>									
	<b>Exposure Dose (mg/kg/day)</b>	<b>Neuro- logical</b>	<b>Dermal</b>	<b>Renal</b>	<b>Cardio- vascular</b>	<b>Hemato- logical</b>	<b>Testicular</b>	<b>Hepatic</b>	<b>Immunological</b>
Lead	59 <sup>1</sup>	5.9	NA <sup>2</sup>	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 x10 <sup>-5</sup>	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 x10 <sup>-6</sup>	0.2	NA	NA	NA	0.01	NA	0.06	0.3
<b>Hazard Index =</b>		<b>10.3</b>	<b>1.25</b>	<b>2.62</b>	<b>9.24</b>	<b>9.83</b>	<b>2.81</b>	<b>0.06</b>	<b>0.3</b>
<b>Adult</b>									
Lead	30.5 <sup>1</sup>	3.05	NA	0.9	3.05	3.05	0.76	NA	NA
Arsenic	4.70 x10 <sup>-4</sup>	1.57	0.59	0.01	1.57	0.78	NA	NA	NA
Cadmium	4.83 x10 <sup>-6</sup>	0.02	NA	0.02	0.00	0.01	0.00	NA	NA
Chromium (VI)	7.53 x10 <sup>-4</sup>	0.08	NA	0.08	NA	0.25	0.15	NA	NA
Aroclor	6.88 x10 <sup>-7</sup>	0.02	NA	NA	NA	0.00	NA	0.007	0.03
<b>Hazard Index =</b>		<b>4.74</b>	<b>0.6</b>	<b>1</b>	<b>4.62</b>	<b>4.09</b>	<b>0.9</b>	<b>0.007</b>	<b>0.03</b>

<sup>1</sup>Blood lead levels in µg/dL; <sup>2</sup>Not available

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

<b>Neurological Toxicity</b>					
		On Toxicity of			
		Lead	Arsenic	Cadmium	Chromium(VI)
Effect of	Lead		>IIB (+0.23)	? (0)	? (0)
	Arsenic	>IIB (+0.50)		? (0)	<IIC2ii (-0.06)
	Cadmium	>IIC (+0.10)	? (0)		? (0)
	Chromium(VI)	? (0)	>IIC (=0.10)	? (0)	
<b>Dermal Toxicity</b>					
Effect of	Lead		? (0)	NA	NA
	Arsenic	NA		NA	NA
	Cadmium	NA	? (0)		NA
	Chromium(VI)	NA	>IIC(+0.10)	NA	
<b>Renal Toxicity</b>					
Effect of	Lead		<IIB (-0.23)	=IIAi (0)	? (0)
	Arsenic	<IIB (-0.23)		? (0)	<IIB2ii (-0.14)
	Cadmium	<IIA (-0.71)	=IIB (0)		? (0)
	Chromium(VI)	? (0)	<IIB2ii (-0.14)	? (0)	
<b>Cardiovascular Toxicity</b>					
Effect of	Lead		? (0)	=IIIA (0)	NA
	Arsenic	? (0)		? (0)	NA
	Cadmium	=IIIA (0)	? (0)		NA
	Chromium(VI)	? (0)	>IIC (+0.10)	? (0)	
<b>Hematological Toxicity</b>					
Effect of	Lead		<IIB (-0.23)	=IIC (0)	? (0)
	Arsenic	<IIB (-0.23)		<IIB (-0.23)	<IIC2ii (-0.06)
	Cadmium	<IIB (-0.23)	<IIB (-0.23)		? (0)
	Chromium(VI)	? (0)	>IIC (+0.10)	? (0)	
<b>Testicular Toxicity</b>					
Effect of	Lead		NA	>IIA (+0.71)	? (0)
	Arsenic	? (0)		<III2Bii (-0.14)	<IIC2ii (-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).

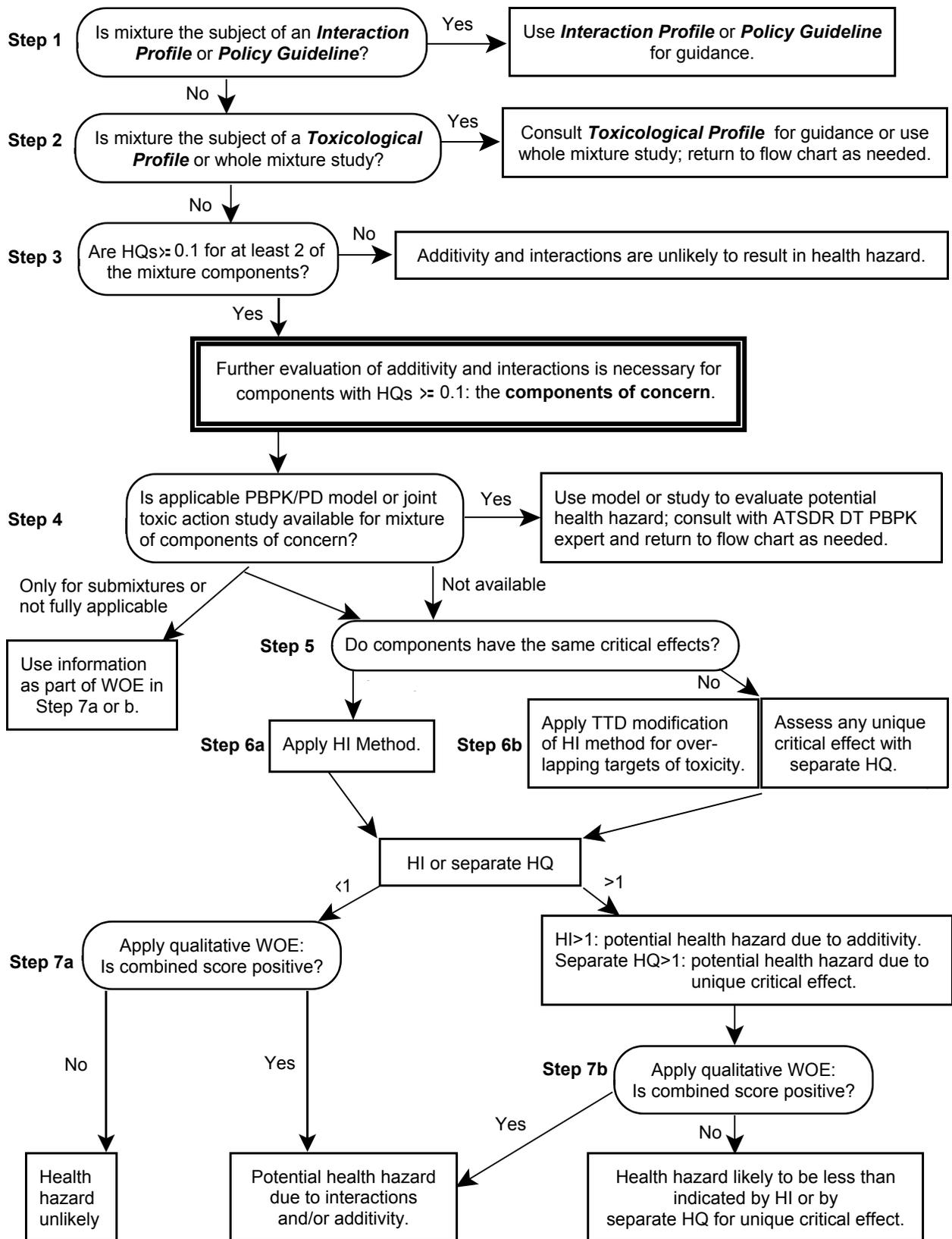


Figure E1: Exposure-Based Assessment of Joint Toxic Action of Chemical Mixtures: Non-Cancer Effects

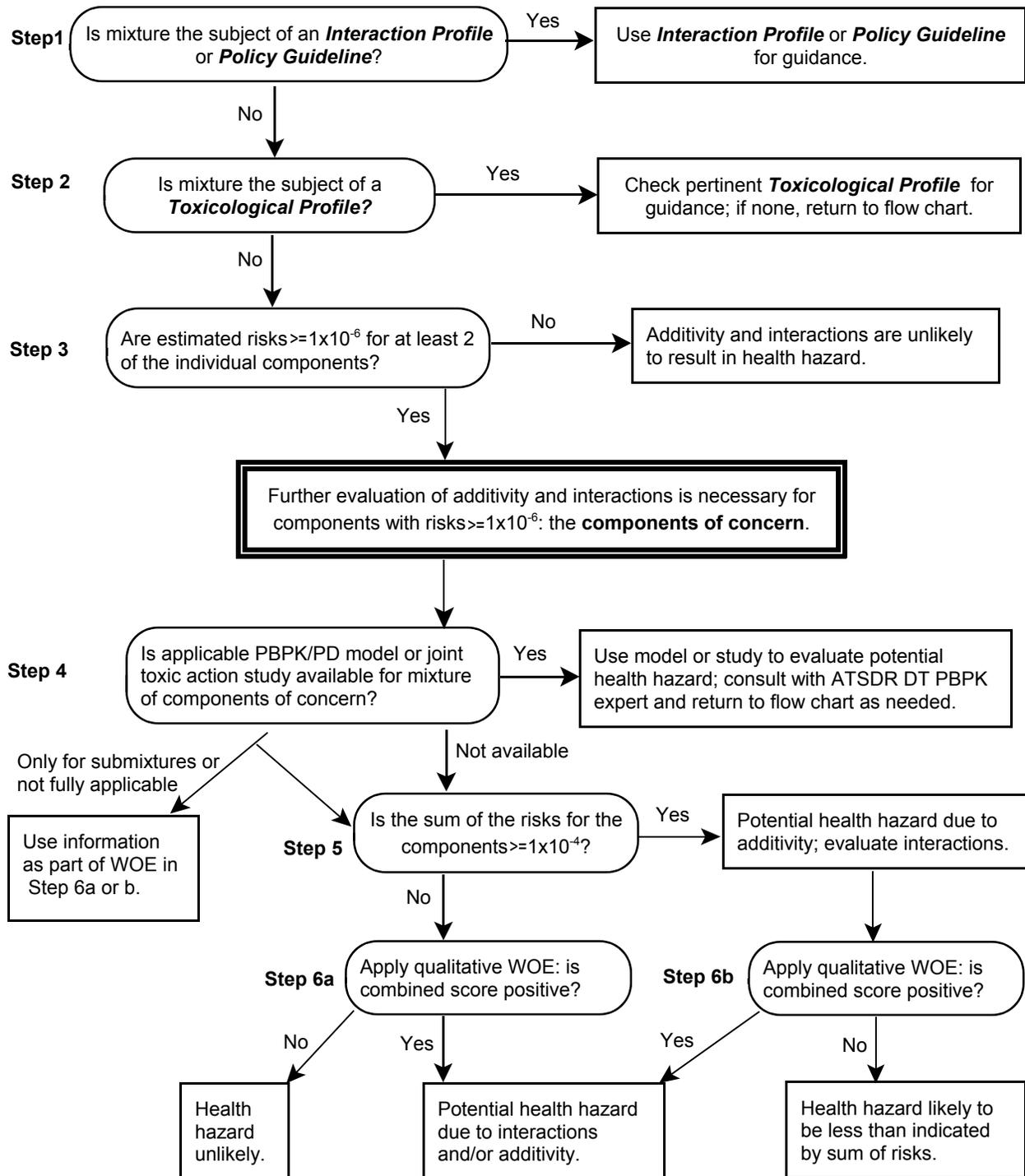


Figure E2: Exposure-Based of Joint Toxic Action of Chemical Mixtures: Cancer Effects

## **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.

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## **Appendix Tables and Figure**

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

<b>Area</b>	<b>1980</b>	<b>1990</b>	<b>2000</b>
<b>Entire Municipality</b>			
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
<b>Focus Area<sup>+</sup></b>			
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.**

<b>Demographic Characteristics</b>	<b>Entire Municipality</b>	<b>Focus Area</b>
<b>Total Cases</b>	1,003	22
<b>Sex</b>		
Male	495	13
Female	508	9
<b>Age at diagnosis</b>		
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.**

<b>Cancer Type</b>	<b>Entire Municipality</b>	<b>Focus Area</b>
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 <sup>1</sup>	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

<sup>1</sup> Note: NR= not reported because observed <5.

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

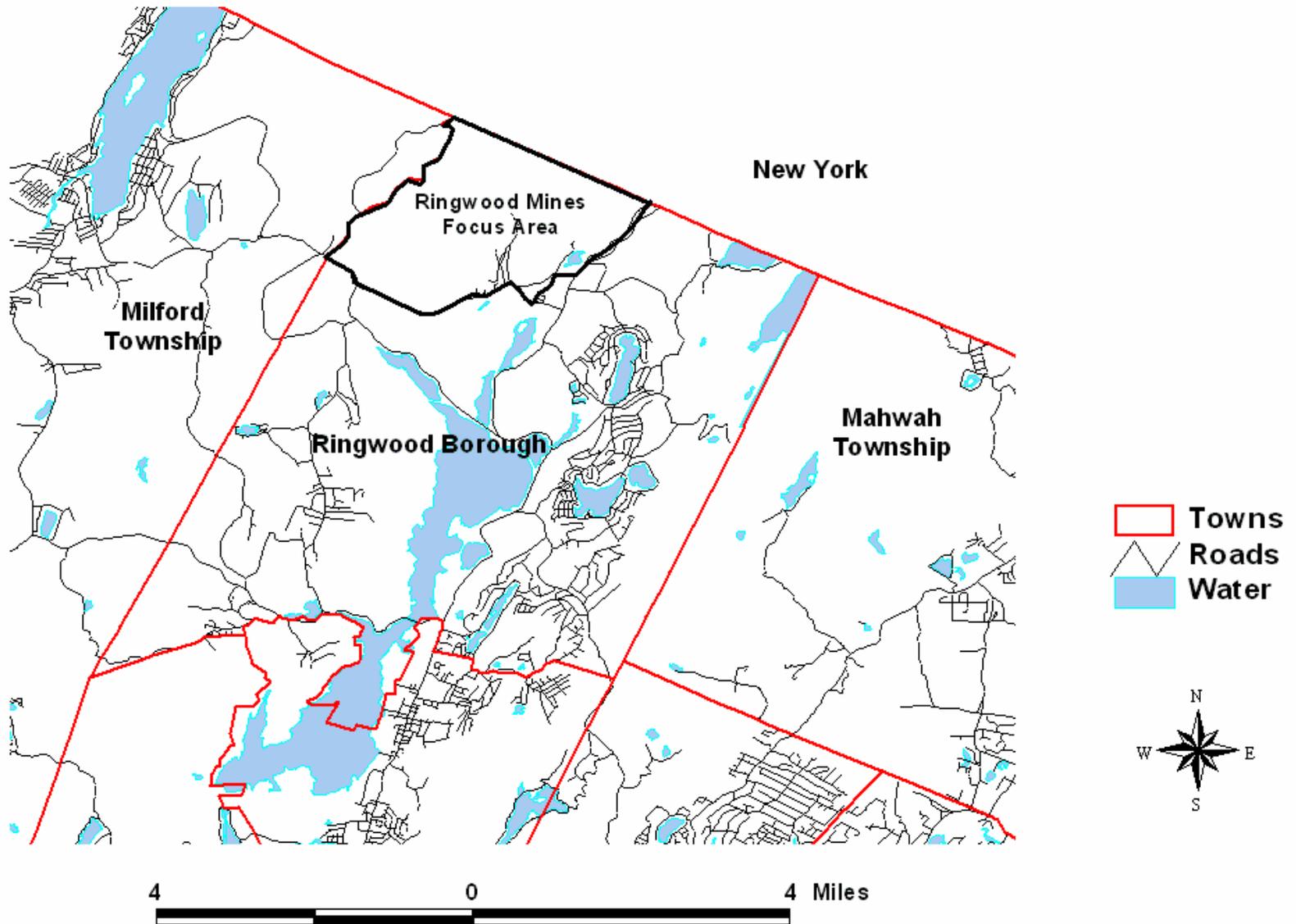
**Table 5F. Ringwood Focus Area Malignant Cancer Incidence (1979-2002), SIR Analysis by Cancer Type and Sex, All Races Combined.**

Cancer Type	Sex	Observed	Expected	SIR <sup>1</sup>	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	–

<sup>1</sup> 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 ( $t_{1/2}$ )**

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<sup>2</sup>**

Milligram per square centimeter (of a surface).

**mg/m<sup>3</sup>**

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/OCEPAt/terms/>)

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>)

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