Health Consultation
Initial Draft

National Oil Company

Plant City, Hillsborough County, Florida

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Division of Disease Control and Health Protection
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U. S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry
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Foreword

The Florida Department of Health (the Department) evaluates the public health risk of hazardous waste sites through a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ASTDR) in Atlanta, Georgia. This is a state report, meaning the Department health professionals reviewed it. The Department also prepared this report using the same guidelines and equations we use for U.S. Environmental Protection Agency (EPA) sites that ATSDR reviews by mandate. This health consultation is part of an effort to evaluate health effects associated with soil and groundwater from the National Oil Company site in Plant City, Florida. The Department evaluates site-related public health issues through the following processes:

**Evaluating exposure:** The Department scientists review available information about environmental conditions at the site. The first task is to find out how much contamination is present, where it is on the site, and how human exposures might occur. The EPA provided the data for this assessment.

**Evaluating health effects:** If evidence is found that exposures to hazardous substances are occurring or might occur, the Department scientists next determine whether that exposure could be harmful to human health. The Department focuses on potential health effects for the community as a whole. The Department bases our conclusions and recommendations on current scientific information.

**Developing recommendations:** The Department lists its conclusions regarding any potential health threat posed by groundwater, air, and soil. The Department then offers recommendations for reducing or eliminating human exposure. The role of the Department in dealing with hazardous waste sites is primarily advisory. Our public health assessments will typically recommend actions for other agencies including the EPA and the Florida Department of Environmental Protection (DEP). If a health threat is actual or imminent, the Department will issue a public health advisory warning people of the danger and will work with the regulatory agencies to resolve the problem.

**Soliciting community input:** The evaluation process is interactive. The Department starts by soliciting and evaluating information from various government agencies, individuals, or organizations responsible for cleaning up the site, and those living in communities near the site. The Department will share any conclusions about the site with the groups and organizations providing the information, and asks for feedback from the public.

*If you have questions or comments about this report, please write to*

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Division of Disease Control and Health Protection  
Bureau of Environmental Health  
4052 Bald Cypress Way, Bin # A-08  
Tallahassee, FL 32399-1720  
*Or, call (850) 245-4240 or toll-free in Florida: 1-877-798-2772*
INTRODUCTION

At the former National Oil Company (NOC) site, the Florida Department of Health’s (the Department) top priority is to ensure the public has the best information to safeguard their health.

The former NOC site is at 402 Michigan Avenue in Plant City, Hillsborough County, Florida. The NOC recycled petroleum products for an unknown period. Operations at the NOC ceased in 1980. In 1987, the U.S. Environmental Protection Agency (EPA) found soil and groundwater contamination. Since then, neither EPA, nor the state has done more testing or cleaned the soil and groundwater.

The Department reached the following five conclusions:

CONCLUSION #1 Incidental ingestion (swallowing) of lead in surface soils at the playground on the site is not likely to harm the health of children. Soil testing on the playground, however, was inadequate to determine the levels of contaminants other than lead.

BASIS FOR DECISIONS #1 Lead in the on-site surface soils is not likely to result in a blood lead level in children above 5 micrograms per deciliter (µg/dL). The Centers for Disease Control and Prevention (CDC) recommend public health actions for blood lead levels above 5 µg/dL.

CONCLUSION #2 Incidental ingestion of lead in surface soils other than the playground may harm the health of future resident children.

BASIS FOR DECISIONS #2 Lead in the on-site surface soils (1,300 mg/kg) could result in a blood lead level in children above 5 µg/dL. The CDC recommends public health actions for blood lead levels above 5 µg/dL.

CONCLUSION #3 Previous investigations collected too few samples to adequately characterize the extent of soil and groundwater contamination.

BASIS FOR DECISIONS #3 Many areas on the site, to include the current playground, were not adequately sampled during previous investigations.

CONCLUSION #4 Drinking on-site groundwater may harm the health of future residents.

BASIS FOR DECISIONS #4 Drinking on-site groundwater contaminated with vinyl chloride could cause an elevated cancer risk.

NEXT STEPS The property owner should not install drinking water wells on the site.
CONCLUSION #5  Incidental ingestion of contaminants in on-site surface soils is not likely to harm workers’ health.

BASIS FOR DECISIONS #5 Pollutants in the on-site surface soils are below levels likely to harm workers’ health. Incidental ingestion would result in, at most, a low increased cancer risk for workers.

CONCLUSION #6 Incidental ingestion of contaminants in on-site surface soils is not likely to harm trespassers’ health.

BASIS FOR DECISIONS #6 Contaminants in on-site surface soil are below levels likely to harm the health of trespassers.

FOR MORE INFORMATION If you have concerns about your health or the health of your children, you should contact your health care provider. You may also call the Department toll-free at 877-798-2772 and ask for information about the National Oil Company site.
Background and Statement of Issues
The purpose of this health consultation report is to assess the public health threat from toxic chemicals in soil and groundwater at the former National Oil Company site (NOC). The Florida Department of Health (the Department) initiated this assessment. The site is at 402 Michigan Avenue, Plant City, Hillsborough County, Florida, 33566 (Figure 1).

The former NOC site is now divided into two parcels, owned by different parties. The western portion is fenced and contains the former NOC building. The eastern portion is vacant, unfenced, and used for parking. On May 26, 2016, Department personnel observed a fenced playground area on the eastern portion (Figure 2). Nearby workers report that children use this playground two or three times a week. In October 2016, the playground was tested for lead and the blood lead level was tested in the children using the playground. Lead tests results were below those that EPA recommends taking action.

NOC recycled petroleum products for an unknown period. They transferred oils and related petroleum products between tanker trucks. NOC used an unlined sump, about 31 feet long, 25 feet wide, and 2 feet deep, to collect spills and overfills. Stormwater runoff from this sump area carried oils and grease northeast to an unlined drainage ditch along East Herring Street. From there, an underground stormwater drain carried oil and grease one block east and then one block south to a drainage canal (Figure 3). Operations at NOC ceased in 1980. Subsequently, wastes in the sump were removed and disposed of at Sidney Mine Landfill in Brandon, Florida [NUS 1991, Tetra Tech 2006].

This assessment estimates the health risk for individuals exposed to the highest measured level of contamination. At this site it is uncertain if anyone contacted contamination in soil or groundwater where consultants collected samples. Therefore, the health risk for workers, trespassers, and children is most likely less than the health risks estimated in this report. Those without exposure have no health risk from this site.

Site Description
The former NOC site is in a mixed residential, commercial, and industrial district of Plant City. The site covers approximately 0.5 acre. The NOC site is bordered to the north by East Herring Street, beyond which lies Jackson Elementary School and residential properties. The site is bordered to the east by North Illinois Street, to the south by First & Fresh Convenience Store and offices, and to the west by North Michigan Street. During a site visit on May 26, 2016, the Department observed a small playground area near the center of the southern site border (Figure 2).

Demographics
The Department examines demographic and land use data to identify sensitive populations, such as young children, the elderly, and women of childbearing age, to determine whether these sensitive populations are exposed to any potential health risks. Demographics also provide details on population mobility and residential history in a particular area. This information helps the Department evaluate how long residents might have been exposed to contaminants.
Approximately 8,600 people live within one mile of the NOC site. Sixty-four percent (64%) are white, 28% are African-American, and 5% are other. Eighteen percent (18%) are less than 18 years old. Approximately twenty-one percent (21%) are women of child-bearing age (15-44 years old). Forty-two percent (42%) have a high school diploma or less and 16% have at least two years of college. Seventy-five percent (75%) speak only English and 69% have a household income of less than $50,000 a year [EPA 2010a].

**Land Use**

Industrial/commercial properties are east and southwest of the site. Jackson Elementary School is north of the site. Most of the other nearby properties are residential.

**Community Health Concerns**

The Department is unaware of any community health concerns regarding this site. The Florida Department of Health in Hillsborough and the EPA did not report any community health concerns regarding this site.

**Discussion**

**Environmental Data**

In February 1990, consultants for the EPA collected 5 surface soil samples (0 to 0.5 feet below ground surface), 3 off-site sediment samples, and 3 groundwater samples (Figure 3). They analyzed all samples for inorganic and organic constituents, including pesticides and polychlorinated biphenyls (PCBs) (Table 4) [NUS1991].

EPA collected too few samples to adequately characterize the extent of soil and groundwater contamination. They did not collect any surface soil samples from the area now used as a playground. The Department needs this information to determine the health risk to children playing on the site.

On September 28, 2016, a Florida-licensed professional geologist collected 4 soil samples just under a surface layer of mulch within the chain-link fence enclosed playground at the site. The consultant collected the samples from the four corners of the playground area. A laboratory analyzed the samples for total lead.

This assessment addresses surface soil samples (0 to 6 inches deep) and does not include deeper samples. Individuals are less likely to contact soil samples deeper than 6 inches.

**Pathway Analyses**

Chemical contamination in the environment can harm your health but only if you have contact with those contaminants (exposure). Without contact or exposure, there is no harm to health. If there is contact or exposure, how much of the contaminants you contact (concentration), how often you contact them (frequency), for how long you contact them (duration), and the danger level of the contaminant (toxicity) all determine the risk of harm.

Knowing or estimating the frequency with which people could have contact with hazardous substances is essential to assessing the public health importance of these contaminants. The
method for assessing whether a health hazard exists to people is to determine whether there is a completed exposure pathway from a contaminant source to a receptor population and whether exposures to contamination are high enough to be of health concern.

An exposure pathway is a series of steps starting with the release of a contaminant in environmental media and ending at the interface with the human body. A completed exposure pathway consists of five elements:

1. A source of contamination like a hazardous waste site
2. An environmental medium like air, water or soil that can hold or move the contamination
3. A point where people come into contact with a contaminated medium like water at the tap or soil in the yard
4. An exposure route like ingesting (contaminated soil or water) or breathing (contaminated air)
5. A population who could be exposed to contamination like nearby residents

Generally, the ATSDR/the Department consider three exposure categories: 1) completed exposure pathways; that is, all five elements of a pathway are present; 2) potential exposure pathways; that is, one or more of the elements may not be present, but information is insufficient to eliminate or exclude the element; and 3) eliminated exposure pathways; that is, a receptor population does not come into contact with contaminated media. Exposure pathways are used to evaluate specific ways in which people were, are, or will be exposed to environmental contamination.

**Completed exposure pathways**

The Department evaluated three completed human exposure pathways: incidental ingestion of on-site soil by a worker, trespasser, and child (at playground). Waste oil disposed of at the NOC site is the source. On-site surface soil (0 to 0.5 feet deep) is the medium and point of exposure. Incidental ingestion, incidentally swallowing very small amounts of soil, is the exposure route. On-site workers, trespassers, and children at the playground are the exposed populations. These exposures happened in the past, are happening now, and will likely happen in the future (Table 1).

**Potential exposure pathways**

The Department evaluated two potential human exposure pathways on the NOC site: private drinking water wells and on-site soil ingestion (Table 2).

For the private drinking water well pathway, the source is waste oil disposal. Ground water is the media and future on-site wells would be the point of exposure. Ingestion would be the exposure route. Future on-site residents would be the exposed population.

For the on-site soil ingestion pathway, the source is waste oil disposal. On-site surface soil is the medium and point of exposure. Ingestion would be the route of exposure. Future on-site residents would be the exposed population.

**Eliminated exposure pathways**

The Department concludes that incidental ingestion of on- or off-site sub-surface soil and use of water from municipal wells are eliminated exposure pathways (Table 3). The Department is
unaware of any exposure to subsurface soils (deeper than 0.5 feet) or sediments on or near the site. No excavations or other activities that might regularly expose people to subsurface soil or sediments are currently on or near the site. Drinking and showering with water from nearby municipal wells are also eliminated exposure pathways. Nearby residents receive municipal water that is routinely tested and is safe to drink. The Department did not locate any private wells within a 1-mile radius of the site [the Department data search 2016].

**Identifying Contaminants of Concern**

The Department compares the maximum concentrations of contaminants found at a site to ATSDR and other comparison values. Comparison values are specific for the medium contaminated (soil, water, air, etc.). The Department screens the environmental data using these comparison values:

- ATSDR Cancer Risk Evaluation Guide (CREG)
- ATSDR Environmental Media Evaluation Guides (EMEGs)
- ATSDR Reference Media Evaluation Guides (RMEGs)
- ATSDR Minimal Risk Level (MRL)
- Florida DEP Soil Cleanup Target Levels (SCTLs)
- EPA Maximum Contaminant Levels (MCLs)
- EPA Lifetime Health Advisory (LTHA)
- EPA Reference Concentration for Chronic Inhalation Exposure (RfC)
- Other guidelines

When determining which comparison value to use, the Department follows ATSDR’s general hierarchy and uses professional judgment.

The Department selects for further evaluation contaminants with maximum concentrations above a comparison value. Comparison values, however, are not thresholds of toxicity. The Department and ATSDR do not use them to predict health effects or to establish clean-up levels. A concentration above a comparison value does not necessarily mean harm will occur. It does indicate, however, the need for further evaluation.

Maximum contaminant concentrations below comparison values are not likely to cause illness and the Department /ATSDR does not evaluate them further.

By comparing the highest measured concentrations in soil and groundwater to ATSDR and EPA screening guidelines, the Department selected aluminum, antimony, arsenic, benzene, benzo(a)pyrene toxic equivalents (BaP TEQ) as a measurement for Polycyclic Aromatic Hydrocarbons (PAHs), cadmium, lead, polychlorinated biphenyls (PCBs) -1254 and 1260, trichloroethylene (TCE), vanadium, and vinyl chloride as contaminants of concern.

Selection of these contaminants does not necessarily mean there is a public health risk. Rather, the Department selected these contaminants for closer scrutiny. Concentrations of other contaminants are below screening guidelines and are not likely to cause illness. The Department /ATSDR does not evaluate these contaminants further.
**Aluminum**
Aluminum is the most abundant metal in the earth's crust and is always found combined with other elements such as oxygen, silicon, and fluorine. Aluminum is obtained from aluminum-containing minerals. Small amounts of aluminum can be found dissolved in water.

Aluminum metal is light in weight and silvery-white in appearance. Aluminum is used for beverage cans, pots and pans, airplanes, siding and roofing, and foil. Alloys are stronger and harder mixtures of aluminum and other metals. Aluminum compounds have many different uses, for example, as alums in water-treatment and alumina in abrasives and furnace linings. Consumer products such as antacids, astringents, buffered aspirin, food additives, cosmetics, and antiperspirants also contain aluminum compounds [ATSDR 2008a].

The most sensitive target of aluminum toxicity is the nervous system. Impaired performance on neurobehavioral tests of motor function, sensory function, and cognitive function have been observed in animals. Neurobehavioral alterations have been observed following exposure of adult or weanling animals and in animals exposed during gestation and/or lactation [ATSDR 2011].

**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. Little antimony is currently mined in the United States. It is brought into this country from other countries for processing. However, there are companies in the United States that produce antimony as a by-product of smelting lead and other metals.

Because antimony is found naturally in the environment, the general population is exposed to low levels of it every day, primarily in food, drinking water, and air. It may be found in air near industries that process or release it, such as smelters, coal-fired plants, and refuse incinerators.

Exposure to antimony occurs in the workplace or from skin contact with soil at hazardous waste sites. Breathing high levels of antimony for a long time can irritate the eyes and lungs, and can cause problems with the lungs, heart, and stomach [ATSDR 1995].

**Arsenic**
Arsenic is a naturally occurring metal widely distributed in soil. Scientists usually find it combined with oxygen, chlorine, and sulfur. Most arsenic compounds have no smell or special taste [ATSDR 2007a].

Arsenic, like most metals, is not well absorbed through the skin. If you get arsenic-contaminated soil on your skin, only a small amount will go through your skin into your body, so skin contact is usually not a health risk [ATSDR 2007a].

Ingesting very high levels of arsenic can result in death. Exposure to lower levels 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.

Several studies have shown that ingestion of inorganic arsenic can increase the risk of skin cancer and cancer in the liver, bladder, and lungs. Inhalation of inorganic arsenic can cause increased risk of lung cancer. The U.S. Department of Health and Human Services (DHHS) and the EPA have determined that inorganic arsenic is a known human carcinogen. The International Agency for Research on Cancer (IARC) has determined that inorganic arsenic is carcinogenic to humans.

There is some evidence that long-term exposure to arsenic in children may result in lower IQ scores. There is also some evidence that exposure to arsenic in the womb and early childhood may increase mortality in young adults [ATSDR 2007a].

State and federal environmental agencies base their arsenic cleanup standards on workplace studies and laboratory animal studies. Because of uncertainties in these studies, their cleanup standards include large safety factors to ensure public health.

**Benzene**

Benzene is a colorless liquid with a sweet odor. It evaporates into the air very quickly and dissolves slightly in water. It is highly flammable and is formed from both natural processes and human activities.

Some industries use benzene to make other chemicals which are used to make plastics, resins, and nylon and other synthetic fibers. Benzene is also used to make some types of rubbers, lubricants, dyes, detergents, drugs, and pesticides. Natural sources of benzene include emissions from volcanoes and forest fires. In addition, benzene is a natural part of crude oil, gasoline, and cigarette smoke.

Benzene is a widely used chemical formed from both natural processes and human activities. Breathing benzene can cause drowsiness, dizziness, and unconsciousness; long-term benzene exposure causes effects on the bone marrow and can cause anemia and leukemia [ATSDR 2007b].

**Cadmium**

Cadmium is a natural element in the earth’s crust. It is usually found as a mineral combined with other elements such as oxygen (cadmium oxide), chlorine (cadmium chloride), or sulfur (cadmium sulfate, cadmium sulfide).

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 cadmium happens mostly in the workplace where cadmium products are made. The general population is exposed from breathing cigarette smoke or eating cadmium contaminated foods. Cadmium damages the kidneys, lungs, and bones [ATSDR 2008b].
**Lead**

Lead is a naturally-occurring bluish-gray metal found in small amounts in the soil. Lead exists in all parts of our environment. Much of it comes from human activities including burning fossil fuels, mining, and manufacturing. Because of health concerns, lead from paints, ceramic products, caulking, and pipe solder has been dramatically reduced in recent years. In 1996, the government banned the use of lead as an additive to gasoline in the United States.

Adults and children may be exposed to lead by hand-to-mouth contact after exposure to lead-containing soil or dust. Most exposure to lead comes from accidental ingestion rather than dermal exposure. Health scientists have long recognized environmental exposure to lead as a public health problem particularly among children. Health scientists have shown excessive concentrations of lead in soil to increase blood lead levels in young children [ATSDR 2007c].

Lead, like most metals, is not well absorbed through the skin. Soil that contains lead may get on your skin, but only a small portion of the lead will pass through your skin and enter your blood. The only kinds of lead compounds that easily penetrate the skin are the additives in leaded gasoline, which is no longer sold to the general public. Therefore, the general public is not likely to encounter lead that can enter through the skin [ATSDR 2007b].

Exposure to lead can happen from breathing workplace air or dust, eating contaminated foods, or drinking contaminated water. Children can be exposed from eating lead-based paint chips or playing in contaminated soil. Lead can damage the nervous system, kidneys, and reproductive system. Signs and symptoms associated with lead toxicity include decreased learning capacity and memory, lowered intelligence quotient (IQ), speech and hearing impairments, fatigue and lethargy.

Protecting children from exposure to lead is important to lifelong good health. No safe blood lead level in children has been identified. Even low levels of lead in blood have been shown to affect IQ, ability to pay attention, and academic achievement. And effects of lead exposure cannot be corrected. The goal is to prevent lead exposure to children before they are harmed. There are many ways parents can reduce a child’s exposure to lead. The most important is stopping children from coming into contact with lead [CDC 2012].

The Department used EPA’s Integrated Exposure Uptake Biokinetic (IEUBK) model to estimate the possible blood lead levels of children exposed daily to surface soil [EPA 2013]. It is important to note that there are uncertainties and limitations in the IEUBK model. One limitation is the inability to decrease the exposure frequency from 365 days per year. Another limitation is that it can only be used to calculate blood lead levels for children.

**Polycyclic Aromatic Hydrocarbons (PAHs) as BaP TEQ**

PAHs are a group of over 100 different chemicals 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.

To evaluate toxicity, ATSDR relates the toxicities of the carcinogenic PAH family members to the toxicity of BaP. They estimate carcinogenic activity relative to BaP as the toxic equivalency factor, or TEF (Appendix C). To determine the PAH toxicity equivalent (TEQ), multiply the concentration of each carcinogenic PAH (other than BaP) by its TEF and then add these to the
BaP concentration. ATSDR considers the PAH TEQ concentration the most valid measure of cancer-producing potency of a complex mixture of PAH compounds.

Animal studies have 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 DHHS has determined that some PAHs may reasonably be expected to be carcinogens [ATSDR 1995b]. Because health scientists believe PAHs may cause cancer through a mutagenic mode, ATSDR and the Department use age-dependent adjustment factors to estimate the increased cancer risk.

**Polychlorinated Biphenyls (PCBs)**

PCBs are a mixture of individual chemicals no longer produced in the United States, but still found in the environment. Manufacturers have used PCBs as coolants and lubricants in transformers, capacitors, and other electrical equipment because they do not burn easily and are good insulators. PCBs have no known smell or taste. Aroclor is the U.S. trade name for many commercial PCB mixtures.

Health effects associated with exposure to PCBs include acne-like skin conditions in adults and neurobehavioral and immunological changes in children. The DHHS concludes that PCBs may reasonably be anticipated to be carcinogens. The EPA and the IARC have determined that PCBs are probably carcinogenic to humans [ATSDR 2000].

**Trichloroethylene (TCE)**

TCE is a colorless, volatile liquid. Liquid trichloroethylene evaporates quickly into the air. It is nonflammable and has a sweet odor.

The two major uses of TCE are as a solvent to remove grease from metal parts and as a chemical that is used to make other chemicals, especially the refrigerant, HFC-134a.

TCE is a solvent for cleaning metal parts. Breathing very high concentrations of trichloroethylene can cause dizziness, headaches, sleepiness, incoordination, confusion, nausea, unconsciousness, and even death. The EPA and the IARC classify trichloroethylene as a human carcinogen [ATSDR 2015].

**Vanadium**

Vanadium is an element that occurs in nature as a white-to-gray metal compound and forms crystals. Pure vanadium has no smell. It usually combines with other elements such as oxygen, sodium, sulfur, or chloride. Geologist have found vanadium and vanadium compounds in the earth's crust and in rocks, some iron ores, and crude petroleum deposits.

Vanadium is in rust-resistant, spring, and high-speed tool steels. Manufacturers use vanadium pentoxide as a catalyst in ceramics and in the production of superconductive magnets.

Everyone is exposed to low levels of vanadium in air, water, and food; however, most people are exposed mainly from food. Breathing high levels of vanadium pentoxide may cause lung damage. Ingesting vanadium can cause nausea and vomiting. In animals, ingesting vanadium can cause decreased red blood cells and increased blood pressure [ATSDR 2012a].
Vinyl Chloride

Vinyl chloride is a colorless gas. It burns easily and it is not stable at high temperatures. It has a mild, sweet odor. It is a manufactured substance that does not occur naturally. It is formed when other substances such as trichloroethane, trichloroethylene, and tetrachloroethylene break down. Vinyl chloride is used to make polyvinyl chloride (PVC). PVC is used to make a variety of plastic products, including pipes, wire and cable coatings, and packaging materials.

Vinyl chloride is also known as chloroethene, chloroethylene, and ethylene monochloride.

Exposure to vinyl chloride occurs mainly in the workplace. Breathing high levels of vinyl chloride for short periods can cause dizziness, sleepiness, unconsciousness, and at extremely high levels can cause death. Breathing vinyl chloride for long periods can result in permanent liver damage, immune reactions, nerve damage, and liver cancer.

Public Health Implications

Health scientists look at what chemicals are present and in what amounts. They compare those amounts to health guidelines. These guidelines are set far below known or suspected levels associated with health effects. The Department uses guidelines developed to protect children. If chemicals are not present at levels high enough to harm children, they would not likely harm adults.

This public health assessment also considers health concerns of nearby residents and explores possible associations with site-related contaminants. This assessment requires the use of assumptions and judgments, and relies on incomplete data. These factors contribute to uncertainty in evaluating the health threat. Assumptions and judgments in the assessment of the site’s impact on public health err on the side of protecting public health and may overestimate the risk.

The Department provides site-specific public health recommendations on the basis of toxicological literature, levels of environmental contaminants, evaluation of potential exposure pathways, duration of exposure, and characteristics of the exposed population. Whether a person will be harmed depends on the type and amount of contaminant, how they are exposed, how long they are exposed, how much contaminant is absorbed, genetics, and individual lifestyles.

After identifying contaminants of concern, the Department evaluates exposures by estimating daily doses for children and adults. Kamrin [1988] explains the concept of dose as follows:

“…all chemicals, no matter what their characteristics, are toxic in large enough quantities. Thus, the amount of a chemical a person is exposed to is crucial in deciding the extent of toxicity that will occur. In attempting to place an exact number on the amount of a particular compound that is harmful, scientists recognize they must consider the size of an organism. It is unlikely, for example, that the same amount of a particular chemical that will cause toxic effects in a 1-pound rat will also cause toxicity in a 1-ton elephant.

Thus, instead of using the amount that is administered or to which an organism is exposed, it is more realistic to use the amount per weight of the organism. Thus, 1 ounce
administered to a 1-pound rat is equivalent to 2,000 ounces to a 2,000-pound (1-ton) elephant. In each case, the amount per weight is the same; 1 ounce for each pound of animal.”

This amount per weight is the dose. Toxicology uses dose to compare toxicity of different chemicals in different animals. The Department uses the units of milligrams (mg) of contaminant per kilogram (kg) of body weight per day (mg/kg/day) to express doses in this assessment. A milligram is 1/1,000 of a gram (3-4 grains of rice weigh approximately 100 mg); a kilogram is approximately 2 pounds.

To calculate the daily doses of each contaminant, the Department uses standard factors for dose calculation [ATSDR 2005; EPA 1997]. The Department assumes that people are exposed daily to the maximum concentration measured and makes the health protective assumption that 100% of the ingested chemical is absorbed into the body. The percent actually absorbed into the body is likely less.

Noncarcinogens - For an assessment of the noncancer health risk, the Department and ATSDR use the following formula to estimate a dose:

\[
D = \frac{(C \times IR \times EF \times CF)}{BW}
\]

D = exposure dose (milligrams per kilogram per day or mg/kg/day)
C = contaminant concentration (milligrams per kilogram or mg/kg)
IR = intake rate of contaminated sediment (milligrams per day or mg/day)
EF = exposure factor (unitless)
CF = conversion factor (10^-6 kilograms per milligram or kg/mg)
BW = body weight (kilograms or kg)

\[
EF = \frac{F \times ED}{AT}
\]

EF = exposure factor (unitless)
F = frequency of exposure (days/year)
ED = exposure duration (years)
AT = averaging time (days) (ED x 365 days/year for noncarcinogens; 70 years x 365 days/year for carcinogens)

ATSDR groups health effects by duration of exposure. Acute exposures are those with duration of 14 days or less; intermediate exposures are those with duration of 15 – 364 days; and chronic exposures are those that occur for 365 days or more (or an equivalent period for animal exposures). ATSDR Toxicological Profiles also provide information on the environmental transport and regulatory status of contaminants.

The Department compares contaminant air concentrations directly to air comparison values and other doses reported in the toxicological literature for inhalation exposures. Children’s doses are generally higher than adults are because their ingestion rates of soil and water, and inhalation of air compared with their low body weights exceed those of adults. For non-cancer illnesses, the Department first estimates the health risk by comparing the exposure dose for children to chemical-specific minimal risk levels (MRLs).
MRLs are health guidelines that establish exposure levels many times lower than levels where scientists observed no effects in animals or human studies. ATSDR designed the MRL to protect the most sensitive, vulnerable individuals in a population. The MRL is an exposure level below which non-cancerous harmful effects are unlikely, even after daily exposure over a lifetime. Although ATSDR considers concentrations at or below the relevant comparison value reasonably safe, exceeding a comparison value does not imply adverse health effects are likely. If contaminant doses/concentrations are above comparison values, the Department further analyzes exposure variables (for example, duration and frequency), toxicology of the contaminants, past epidemiology studies, and the weight of evidence for health effects. The Department uses chronic MRLs where possible because exposures are usually longer than a year. If chronic MRLs are not available, the Department uses intermediate length MRLs [ATSDR 2005].

The Department and ATSDR use the following equation to estimate increased cancer risk:

\[
\text{Risk} = D \times SF
\]

- \( \text{Risk} \) = Cancer risk
- \( D \) = Age specific non-cancer dose (mg/kg/day)
- \( SF \) = Slope factor (mg/kg/day)^{-1}

If the chemical increases the cancer risk due to early life exposure, the Department and ATSDR use the following equation to estimate increased cancer risk:

\[
\text{Risk} = D \times SF \times \text{ADAF}
\]

- \( D \) = Age specific exposure dose (mg/kg/day)
- \( SF \) = Slope factor (mg/kg/day)^{-1}
- \( \text{ADAF} \) = Age Dependent Adjustment Factor

This is a conservative estimate of the increased cancer risk. The actual increased cancer risk is likely lower. Because of large uncertainties in the way scientists estimate cancer risks, the actual risk may be as low as zero. The Department usually estimates the cancer risk for lifetime (78 years) exposure. Studies of animals exposed over their entire lifetime are the basis for calculating cancer slope factors. Usually, researchers know little about the cancer risk in animals from less than lifetime exposures. Therefore, the Department also uses lifetime exposure to estimate the cancer risk in people. If there is no cancer slope (potency) factor, the Department /ATSDR cannot quantify the risk.

**Completed Human Exposure Pathways**

**On-Site Surface Soil – Worker Exposure**

The Department assumed soil intake (incidental ingestion) of 100 milligrams (mg)/day by an adult worker (outdoor with low soil contact) weighing 80 kilograms (kg) (approximately 176 pounds), exposed 5 times per week for 25 years.
The Department evaluates the health risk based on the highest levels from five on-site soil samples and three off-site sediment samples. Eight samples are, however, too few to adequately characterize the extent of soil contamination. This is a data gap. Additional testing may identify areas with higher levels and thus a higher health risk.

**Antimony**
The Department estimated exposure using the maximum on-site soil concentration of 100 milligrams per kilogram (mg/kg).

**Noncancer illnesses**
A worker who incidentally swallows very small amounts of surface soil from the site with the highest antimony levels is unlikely to develop noncancer illnesses. ATSDR has not established MRLs for antimony [ATSDR 1995a]. However, the maximum dose \(8.9 \times 10^{-5} \text{mg/kg/day}\) is hundreds of times less than the oral no adverse effect level (NOAEL) of \(2.6 \times 10^{-1} \text{mg/kg/day}\) and thus unlikely to cause noncancer illnesses (Table 5).

**Cancer**
It is not known whether antimony will cause cancer in people. The DHHS, the IARC, and the EPA have not classified antimony as to its human carcinogenicity [ATSDR 1995a].

**Polycyclic Aromatic Hydrocarbons (PAHs) - Benzo(a)pyrene (BaP) TEQ**
The Department estimated exposure using the maximum on-site soil concentration of 1.97 mg/kg.

**Noncancer illnesses**
A worker who incidentally swallows very small amounts of surface soil with the highest noncarcinogenic PAH levels is unlikely to develop noncancer illnesses. The Department estimated exposure using the maximum soil concentration for each of the noncarcinogenic PAHs (1-methylnaphthalene, 2-methylnaphthalene, acenaphthene, anthracene, fluoranthene, fluorene, naphthalene, and pyrene). The Department compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. All maximum concentrations were below the EPA noncarcinogenic screening levels.

**Cancer**
Workers who incidentally ingest (swallow very small amounts of) surface soil with the highest BaP TEQ levels over a 25-year period are at a “low” increased estimated risk of cancer. Multiplying the maximum BaP TEQ dose \(5.6 \times 10^{-7} \text{mg/kg/day}\) by the EPA cancer slope factor \((7.3 \text{mg/kg/day}^{-1})\) results in an increased estimated cancer risk of \(4 \text{in} 1,000,000 \left(4 \times 10^{-6}\right)\) (Table 6).

To put this into context, the American Cancer Society estimates that one out of every three Americans (or 333,333 in 1,000,000) will be diagnosed with some form of cancer in their lifetime. Adding the estimated increased cancer risk from exposure to PAHs in the surface soil at the NOC site would increase the cancer incidence from 333,333 in 1,000,000 to 333,337 in 1,000,000.
**Lead**
The Department estimated exposure using the maximum on-site soil concentration of 1,300 mg/kg.

**Noncancer illnesses**
Estimated blood lead levels more accurately predict health effects than traditional dose estimates. Using EPA’s Adult Lead Model, the Department estimates that exposure to the highest concentration of lead in surface soil on the site (1,300 mg/kg) would result in approximately 2.9 to 3.4 micrograms of lead per deciliter blood (µg/dL) in workers [EPA 2009]. In general, adults with blood lead levels less than 5 µg/dL are not likely to suffer any noncancer illness [ATSDR 2007c].

**Cancer**
The DHHS has determined that lead is reasonably anticipated to be a human carcinogen based on limited evidence from studies in humans and sufficient evidence from animal studies. EPA has determined that lead is a probable human carcinogen. The IARC has determined that inorganic lead is probably carcinogenic to humans [ATSDR 2007c].

EPA has not established a cancer slope factor for lead. Therefore, the Department was unable to calculate a lifetime increased cancer risk.

**Polychlorinated Biphenyls (PCBs)**
The Department estimated adult worker exposure using a maximum on-site soil concentration for PCBs of 8.2 mg/kg. This amount is the total for the groups of PCBs Aroclor 1254 (6.3 mg/kg) and Aroclor 1260 (1.9 mg/kg).

**Noncancer illnesses**
A maintenance worker who incidentally ingests very small amounts of surface soil from the site with the highest PCBs levels is unlikely to develop noncancer illnesses. The maximum adult PCBs dose (7.3 x 10^{-6} mg/kg/day) is less than ATSDR’s chronic MRL (2 x 10^{-5} mg/kg/day) [ATSDR 2000] and thus unlikely to cause noncancer illnesses (Table 5).

**Cancer**
Workers who incidentally ingest surface soil with the highest PCBs levels at the site over a 25-year period are at a “low” increased estimated risk of cancer (Table 6). Multiplying the maximum PCB dose (2.3 x 10^{-6} mg/kg/day) by the EPA cancer slope factor (2.0 mg/kg/day^{-1}) results in an increased estimated cancer risk of approximately 5 in a million (0.000005 or 5 x 10^{-6}).

**On-Site Surface Soil – Child Playground Exposure**
The Department assumes a child 2 to 6 years of age weighing 17.4 kg (approximately 38 pounds) incidentally swallows 100 mg of surface soil per day, 3 times per week for 4 years.

The Department evaluates the health risk to children based on the highest levels from four surface soil samples collected from the on-site playground September 2016. The samples from
the playground area were only tested for lead. In October 2016, the blood lead levels were tested for children using the playground. The results were below the EPA action level of 5 micrograms of lead per deciliter blood (µg/dL). The lack of testing for other site-related contaminants is a data gap. Additional testing in the playground area may identify other contaminants.

**Lead**
The Department estimated child playground exposure using a maximum soil concentration for lead of 150 mg/kg.

**Noncancer illnesses**
Estimated blood lead levels more accurately predict health effects than traditional dose estimates [ATSDR 2007c]. Using EPA’s IEUBK model, the Department estimates that exposure to the highest concentration of lead in surface soil on the site (150 mg/kg) would result in an approximate 1.0 to 2.1 µg/dL (Table 7). Although CDC has not identified a safe blood lead level, they recommend public health actions above 5 µg/dL. A child who incidentally ingests very small amounts of surface soil with the highest lead levels is unlikely to develop noncancer illnesses.

**Cancer**
The DHHS has determined that lead is reasonably anticipated to be a human carcinogen based on limited evidence from studies in humans and sufficient evidence from animal studies. EPA has determined that lead is a probable human carcinogen. The IARC has determined that inorganic lead is probably carcinogenic to humans [ATSDR 2007c].

EPA has not established a cancer slope factor for lead. Therefore, the Department was unable to calculate a lifetime increased cancer risk.

**On-Site Surface Soil – Trespasser Exposure**
The Department assumes a trespasser weighing 55 kg (approximately 121 pounds) incidentally swallows 100 mg of surface soil per day 2 times per week for 10 years.

The Department evaluates the health risk based on the highest levels from five on-site soil samples and three off-site sediment samples. Eight samples are, however, too few to adequately characterize the extent of soil contamination. This a data gap. Additional testing may identify areas with higher levels and thus a higher health risk.

**Antimony**
The Department estimated trespasser exposure using a maximum on-site soil concentration for antimony of 100 mg/kg.

**Noncancer illnesses**
A trespasser who incidentally ingests very small amounts of surface soil from the site with the highest antimony levels is unlikely to develop noncancer illnesses. The ATSDR has not established MRLs for antimony [ATSDR 1995a]. However, the maximum antimony noncancer dose (4.9 x 10^{-5} mg/kg/day) is thousands of times less than the oral no adverse effect level (NOAEL) of 2.6 x 10^{-1} mg/kg/day and thus unlikely to cause noncancer illnesses (Table 5).
Cancer
It is not known whether antimony will cause cancer in people. The DHHS, the IARC, and the EPA have not classified antimony as to its human carcinogenicity [ATSDR 1995a].

PAHs - Benzo(a)pyrene (BaP) as a TEQ
The Department estimated trespasser exposure using a maximum on-site soil concentration for BaP of 1.97 mg/kg.

Noncancer illnesses
The Department estimated exposure using the maximum soil concentration for each of the noncarcinogenic PAHs (1-methylnaphthalene, 2-methylnaphthalene, acenaphthene, anthracene, fluoranthene, fluorene, naphthalene, and pyrene). The Department compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. A trespasser who incidentally ingests very small amounts of surface soil with the highest noncarcinogenic PAH levels is unlikely to develop noncancer illnesses. The Department did not calculate doses for the noncarcinogenic PAHs since all maximum concentrations were below the EPA noncarcinogenic screening levels.

Cancer
Trespassers who incidentally ingest (swallow very small amounts of) surface soil with the highest BaP TEQ levels at the site over a 10-year period are at a “low” increased estimated risk of cancer (Table 5). Multiplying the maximum BaP TEQ dose (1.2 x 10^{-7} mg/kg/day) by the EPA cancer slope factor (7.3 mg/kg/day^{-1}) results in an increased estimated cancer risk of 9 in 10,000,000 (9 x 10^{-7}) (Table 8).

To put this into context, the American Cancer Society estimates that one out of every three Americans (or 3,333,333 in 10,000,000) will be diagnosed with some form of cancer in their lifetime. Adding the estimated increased cancer risk from exposure to PAHs in the surface soil at the NOC site would increase the cancer incidence from 3,333,333 in 10,000,000 to 3,333,342 in 10,000,000.

Lead
The Department estimated trespasser exposure using a maximum on-site soil concentration for lead of 1,300 mg/kg.

Noncancer illnesses
Estimated blood lead levels more accurately predict health effects than traditional dose estimates [ATSDR 2007a]. Using EPA’s Adult Lead Model, the Department estimates that exposure to the highest concentration of lead in surface soil on the site (1,300 mg/kg) would result in 1.9 to 2.4 micrograms of lead per deciliter blood (µg/dL) in trespassers [EPA 2009]. In general, adults with blood lead levels less than 5 µg/dL are not likely to suffer any noncancer illness [ATSDR 2007c].

Cancer
The DHHS has determined that lead is reasonably anticipated to be a human carcinogen based on limited evidence from studies in humans and sufficient evidence from animal studies. EPA has
determined that lead is a probable human carcinogen. The IARC has determined that inorganic lead is probably carcinogenic to humans [ATSDR 2007c].

EPA has not established a cancer slope factor for lead. Therefore, the Department was unable to calculate a lifetime increased cancer risk.

**Polychlorinated Biphenyls (PCBs)**

The Department estimated trespasser exposure using a maximum on-site soil concentration for PCBs of 8.2 mg/kg. This amount is the total for the group of PCBs Aroclor 1254 (6.3 mg/kg) and Aroclor 1260 (1.9 mg/kg).

**Noncancer illnesses**

A trespasser who incidentally ingests very small amounts of surface soil from the site with the highest PCBs levels is unlikely to develop noncancer illnesses. The maximum trespasser PCBs dose (4.0 x 10^{-6} mg/kg/day) is less than ATSDR’s chronic MRL (2 x 10^{-5} mg/kg/day) [ATSDR 2000] and thus unlikely to cause noncancer illnesses (Table 5).

**Cancer**

Trespassers who incidentally ingest surface soil with the highest PCBs levels at the NOC site over a 10-year period are at a “low” increased estimated risk of cancer (Table 8). Multiplying the maximum PCB dose (5.1 x 10^{-7} mg/kg/day) by the EPA cancer slope factor (2.0 mg/kg/day^{-1}) results in an increased estimated cancer risk of approximately 1 in a million (0.0000001 or 1 x 10^{-6}).

To put this into context, the American Cancer Society estimates that one out of every three Americans (or 333,333 in 1,000,000) will be diagnosed with some form of cancer in their lifetime. Adding the estimated increased cancer risk from exposure to PCBs in the surface soil at the NOC site would increase the cancer incidence from 333,333 in 1,000,000 to 333,334 in 1,000,000.

**Potential Human Exposure Pathways**

**On-Site Surface Soil – Residential Exposure**

The Department assumes a future on-site adult resident weighing 80 kg (approximately 176 pounds) incidentally swallows 100 mg of surface soil every day for 33 years. The Department took into account the mutagenic effects of PAHs for children 6 weeks to 16 years of age when estimating the cancer risks for residential exposure. The Department calculated exposure risks using the maximum concentration for each contaminant above the screening level. Increased cancer risk at maximum exposure levels were low (10^{-5} to 10^{-6}). Combined cancer risks for residential exposure were also low.

The Department evaluates the future health risk based on the highest levels from five on-site soil samples and three off-site sediment samples. Eight samples are insufficient to adequately characterize the extent of soil contamination. This a data gap. Additional testing may identify areas with higher levels and thus a higher health risk.
**Antimony**

The Department estimated future residential exposure using a maximum on-site soil concentration for antimony of 100 mg/kg.

**Noncancer illnesses**

A future resident who incidentally ingests very small amounts of surface soil from the site with the highest antimony levels is unlikely to develop noncancer illnesses. ATSDR has not established MRLs for antimony [ATSDR 1995a]. However, the maximum residential antimony noncancer dose (6.3 x 10^{-5} mg/kg/day) is thousands of times less than the oral no adverse effect level (NOAEL) of 2.6 x 10^{-1} mg/kg/day and thus unlikely to cause noncancer illnesses (Table 5).

**Cancer**

It is not known whether antimony will cause cancer in people. The DHHS, the IARC, and the EPA have not classified antimony as to its human carcinogenicity [ATSDR 1995a].

**PAHs - Benzo(a)pyrene (BaP) as a TEQ**

The Department estimated future residential exposure using a maximum on-site soil concentration for BaP of 1.97 mg/kg.

**Noncancer illnesses**

The Department estimated exposure using the maximum soil concentration for each of the noncarcinogenic PAHs (1-methylnaphthalene, 2- methylnaphthalene, acenaphthene, anthracene, fluoranthene, fluorene, naphthalene, and pyrene). The Department compared the maximum concentration against the EPA noncarcinogenic screening levels using a noncancer hazard index of 0.1. A future on-site resident who incidentally ingests very small amounts of surface soil with the highest noncarcinogenic PAH levels is unlikely to develop noncancer illnesses. The Department did not calculate doses for the noncarcinogenic PAHs since all maximum concentrations were below the EPA noncarcinogenic screening levels.

**Cancer**

Future on-site residents who incidentally ingest surface soil with the highest BaP TEQ levels at the site over a 33-year period are at a “low” increased estimated risk of cancer (Table 5). Multiplying the maximum BaP TEQ residential dose (5.2 x 10^{-7} mg/kg/day) by the EPA cancer slope factor (7.3 mg/kg/day^{-1}) results in an increased estimated cancer risk of 1 in 100,000 (1 x 10^{-5}) (Table 9).

To put this into context, the American Cancer Society estimates that one out of every three Americans (or 33,333 in 100,000) will be diagnosed with some form of cancer in their lifetime. Adding the estimated increased cancer risk from exposure to PAHs in the surface soil at the NOC site would increase the cancer incidence from 33,333 in 100,000 to 33,334 in 100,000.

**Lead**

The Department estimated future residential exposure using a maximum on-site soil concentration for lead of 1,300 mg/kg.
Noncancer illnesses
Estimated blood lead levels more accurately predict health effects than traditional dose estimates [ATSDR 2007c]. Using EPA’s IEUBK model, the Department estimates that future residential child exposure to the highest concentration of lead in surface soil on the site (1,300 mg/kg) would result in an approximate 4.8 to 7.5 micrograms of lead per deciliter blood (µg/dL) (Table 7). Although CDC has not identified a safe blood lead level, they recommend public health actions above 5 µg/dL. ATSDR lists impaired development of the nervous system, delayed sexual maturation, and neurobehavioral effects as possible adverse health effects in children with blood lead levels less than 10 µg/dL [CDC 2016].

Cancer
The DHHS has determined that lead is reasonably anticipated to be a human carcinogen based on limited evidence from studies in humans and sufficient evidence from animal studies. EPA has determined that lead is a probable human carcinogen. The IARC has determined that inorganic lead is probably carcinogenic to humans [ATSDR 2007c].

EPA has not established a cancer slope factor for lead. Therefore, the Department was unable to calculate a lifetime increased cancer risk.

Polychlorinated Biphenyls (PCBs)
The Department estimated future residential exposure using a maximum on-site soil concentration for PCBs of 8.2 mg/kg. This amount is the total for PCB groups Aroclor 1254 (6.3 mg/kg) and Aroclor 1260 (1.9 mg/kg).

Noncancer illnesses
A future resident who incidentally ingests very small amounts of surface soil from the site with the highest PCBs levels is unlikely to develop noncancer illnesses. The maximum residential PCBs dose (5.1 x 10^{-6} mg/kg/day) is less than ATSDR’s chronic MRL (2 x 10^{-5} mg/kg/day) [ATSDR 2000] and thus unlikely to cause noncancer illnesses (Table 5).

Cancer
Future residents who incidentally ingest surface soil with the highest PCBs levels at the NOC site over a 33-year period are at a “low” increased estimated risk of cancer (Table 9). Multiplying the maximum PCB dose (2.2 x 10^{-6} mg/kg/day) by the EPA cancer slope factor (2.0 mg/kg/day⁻¹) results in an increased estimated cancer risk of approximately 2 in a 100,000 (0.00002 or 2 x 10^{-5}).

To put this into context, the American Cancer Society estimates that one out of every three Americans (or 33,333 in 100,000) will be diagnosed with some form of cancer in their lifetime. Adding the estimated increased cancer risk from exposure to PCBs in the surface soil at the NOC site would increase the cancer incidence from 33,333 in 100,000 to 33,335 in 100,000.

On-Site Groundwater – Residential Exposure
The Department assumes a future on-site adult resident weighing 80 kg (approximately 176 pounds) drinks 1.23 liters of contaminated groundwater every day for 33 years. The Department
took into account any mutagenic effects for children 6 weeks to 16 years of age when estimating the cancer risks for residential exposure. The Department calculated exposure risks using the maximum concentration for each contaminant above the screening level (Table 10). Maximum exposure risks were low ($10^{-4}$ to $10^{-6}$). Combined cancer risks for residential exposure are also low.

The Department evaluates the health risk based on the highest levels from three groundwater samples collected and analyzed 26 years ago. Additional testing may identify groundwater with higher levels. Conversely, additional testing may show that contaminant levels declined over time. Three groundwater samples are inadequate to determine the extent of groundwater contamination. This is a data gap.

### Aluminum

The Department estimated future residential exposure using a maximum on-site groundwater concentration for aluminum of 100 milligrams per liter (mg/L).

#### Noncancer illnesses

A future resident who incidentally ingests groundwater from the site with the highest aluminum levels is unlikely to develop noncancer illnesses. The estimated annual arsenic dose ($1.5 \text{ mg/kg/day}$) for a resident is slightly greater than ATSDR’s chronic MRL ($1.0 \text{ mg/kg/day}$) [ATSDR 2008a]. However, the maximum residential aluminum noncancer dose is 17 times less than the oral no adverse effect level (NOAEL) of 26 mg/kg/day and thus unlikely to cause noncancer illnesses (Table 11).

#### Cancer

Aluminum has not been shown to cause cancer in animals and has not been tested in humans.

### Arsenic

The Department estimated future residential exposure using a maximum on-site groundwater concentration for arsenic of 0.02 mg/L (estimated) and a relative bioavailability factor of 60% [EPA 2015].

#### Noncancer illnesses

Future adult residents who drink groundwater from the site with the highest arsenic levels are unlikely to develop noncancer illnesses. The estimated annual arsenic dose ($2.0 \times 10^{-4} \text{ mg/kg/day}$) for an adult resident is less than ATSDR’s chronic MRL ($3.0 \times 10^{-4} \text{ mg/kg/day}$). Children less than 2 years of age have estimated dose range ($3.5 \text{ to } 8.5 \times 10^{-4} \text{ mg/kg/day}$) above the MRL. The dose for children less than 2 would fall between the NOAEL of $4.0 \times 10^{-4} \text{ mg/kg/day}$ and the lowest observed adverse effect level (LOAEL) of $2.2 \times 10^{-2} \text{ mg/kg/day}$ (Table 11). Since the highest child dose is only two times more than the NOAEL but 25 times less than the LOAEL, noncancer illnesses are unlikely [ATSDR 2007a].

#### Cancer

A future resident who drinks groundwater with the highest arsenic levels from the site is at a “low” increased risk of cancer. Multiplying the maximum arsenic dose ($1.4 \times 10^{-4} \text{ mg/kg/day}$) by the EPA cancer slope factor ($1.5 \text{ mg/kg/day}^{-1}$) results in an increased estimated cancer risk of 3 in 100,000 (0.00003 or $3 \times 10^{-5}$) (Table 12).
**Benzene**
The Department estimated future residential exposure using a maximum on-site groundwater concentration for benzene of 0.012 mg/L.

**Noncancer illnesses**
Future residents who drink groundwater from the site with the highest benzene levels are unlikely to develop noncancer illnesses. The estimated annual benzene dose for adults (1.8 x 10^{-4} mg/kg/day) is less than ATSDR’s chronic MRL (5.0 mg/kg/day x 10^{-4}). For children less than 1 year of age the estimated dose (7.8 x 10^{-4} mg/kg/day) is slightly above the MRL but over 370 times less than the LOAEL of 0.29 mg/kg/day (Table 11) [ATSDR 2007b].

**Cancer**
A future resident who drinks groundwater with the highest benzene levels from the site is at a “low” increased risk of cancer. Multiplying the maximum benzene dose (7.8 x 10^{-5} mg/kg/day) by the EPA cancer slope factor (0.055 mg/kg/day^{-1}) results in an increased estimated cancer risk of 5 in 1,000,000 (0.000005 or 5 x 10^{-6}) (Table 12).

**Cadmium**
The Department estimated future residential exposure using a maximum on-site groundwater concentration for cadmium of 0.049 mg/L.

**Noncancer illnesses**
Future residents who drink groundwater from the site with the highest cadmium levels are unlikely to develop noncancer illnesses. The estimated annual cadmium dose (7.5 x 10^{-4} mg/kg/day) is greater than ATSDR’s chronic MRL (1.0 x 10^{-4} mg/kg/day) but 10 times less than the NOAEL of 7.8 x 10^{-3} mg/kg/day (Table 10) [ATSDR 2012b].

**Cancer**
Although cadmium can cause cancer when inhaled, neither human nor animal studies provide sufficient evidence to determine whether it causes cancer when ingested.

**Lead**
The Department estimated future residential exposure using a maximum on-site groundwater concentration for lead of 0.049 mg/L.

**Noncancer illnesses**
A future resident who drinks groundwater from the site with the highest lead levels has a risk to develop noncancer illnesses such as impaired development of the nervous system, delayed sexual maturation, and neurobehavioral effects. Children could also be at risk of reduced mental and physical growth. There are no established oral MRL, NOAEL, or LOAELs for lead [ATSDR 2007c]. However, the Florida DEP has established a maximum contaminant level (MCL) of 0.015 mg/L for lead [DEP 2012] (Table 11).
Cancer
EPA has not established a cancer slope factor for lead with which to quantify an increased cancer risk. Therefore, the Department was unable to calculate an increased cancer risk for lead exposure.

Trichloroethylene (TCE)
The Department estimated future residential exposure using a maximum on-site groundwater concentration for TCE of 0.012 mg/L.

Noncancer illnesses
Future residents who ingest groundwater from the site with the highest TCE levels are unlikely to develop noncancer illnesses. The estimated annual adult TCE dose ($1.8 \times 10^{-4}$ mg/kg/day) is less than ATSDR’s chronic MRL ($5.0 \times 10^{-4}$ mg/kg/day). Although the estimated dose for children less than 1 year of age ($7.8 \times 10^{-4}$ mg/kg/day) is slightly above the MRL, it is significantly less than the NOAEL of 50 mg/kg/day (Table 11) [ATSDR 2014].

Cancer
Future residents who ingest groundwater with the highest TCE levels from the site are at a “low” increased risk of cancer. Multiplying the maximum TCE dose ($7.8 \times 10^{-5}$ mg/kg/day) by the EPA cancer slope factor ($0.046$ mg/kg/day$^{-1}$) results in an increased estimated cancer risk of 1 in 100,000 ($0.00001$ or $1 \times 10^{-5}$) (Table 12).

Vanadium
The Department estimated future residential exposure using a maximum on-site groundwater concentration for vanadium of 0.14 mg/L.

Noncancer illnesses
Future residents who ingest groundwater from the site with the highest vanadium levels are unlikely to develop noncancer illnesses. The estimated annual vanadium dose ($2.1 \times 10^{-3}$ mg/kg/day) for a future resident is less than ATSDR’s intermediate MRL ($1.0 \times 10^{-2}$ mg/kg/day) and NOAEL ($0.7$ mg/kg/day) (Table 11) [ATSDR 2012c].

Cancer
Although breathing vanadium pentoxide causes lung cancer in mice and may cause lung cancer in people, there is not enough information to tell if vanadium in drinking water causes cancer in either animals or people [ATSDR 2012c].

Vinyl Chloride
The Department estimated future residential exposure using a maximum on-site groundwater concentration for vinyl chloride of 0.12 mg/L.

Noncancer illnesses
A resident who ingests groundwater from the site with the highest vinyl chloride levels is unlikely to develop noncancer illnesses. The estimated annual vinyl chloride dose ($1.8 \times 10^{-3}$ mg/kg/day) for a resident is less than ATSDR’s chronic MRL ($3.0 \times 10^{-3}$ mg/kg/day) but children
less than 2 years of age have estimated doses (3.2 to 7.8 x 10^{-3} mg/kg/day) above the MRL. The doses for children less than 2 are significantly less than the NOAEL of 1.7 mg/kg/day (Table 1) [ATSDR 2006].

Cancer
A resident who ingests groundwater with the highest vinyl chloride levels from the site is at an elevated increased estimated risk of cancer. Multiplying the maximum vinyl chloride dose (7.8 x 10^{-4} mg/kg/day) by the EPA cancer slope factor (0.72 mg/kg/day^{-1}) results in an increased estimated cancer risk of 6 in 10,000 (0.0006 or 6 x 10^{-4}) (Table 12).

Child Health Considerations
In communities faced with air, water, or soil contamination, the many physical differences between children and adults demand special attention. Children could be at greater risk than adults from certain kinds of exposure to hazardous substances. Children play outdoors and sometime engage in hand-to-mouth behaviors that increase their exposure potential. Children are shorter than adults; this means they breathe dust, soil and vapors close to the ground. A child’s lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. If toxic exposure levels are high enough during critical growth stages, the developing body system of children can sustain permanent damage. Finally, children are dependent on adults for access to housing, for access to medical care, and for risk identification. Thus, adults need as much information as possible to make informed decisions regarding their children’s health.

This assessment takes into account the special vulnerabilities of children. It specifically assesses the health risk for children playing in the surface soil of properties near the former NOC site.

Conclusions
Overall, the Department finds the National Oil Company site is a public health hazard. The Department concludes that:

1. Incidental ingestion (swallowing) of lead in surface soils at the playground on the site is not likely to harm the health of children. Soil testing on the playground, however, was inadequate to determine the levels of contaminants other than lead.
2. Incidental ingestion of lead in surface soils other than the playground may harm the health of future resident children. Lead in the on-site surface soils (1,300 mg/kg) could result in a blood lead level in children above 5 µg/dL. The CDC recommend public health actions for blood lead levels above 5 µg/dL.
3. Previous investigations collected too few samples to adequately characterize the extent of soil and ground water contamination.
4. Drinking on-site groundwater may harm the health of future residents. Drinking on-site groundwater contaminated with vinyl chloride could cause an elevated cancer risk.
5. Incidental ingestion of contaminants in on-site surface soils is not likely to harm workers’ health. Pollutants in the on-site surface soils are below levels likely to harm their health. Incidental ingestion would result in, at most, a low increased cancer risk for workers.
6. Incidental ingestion of contaminants in on-site surface soils is not likely to harm trespassers’ health. Contaminants in on-site surface soil are below levels likely to harm health of trespassers.

**Recommendations**

1. The playground property owner should test the surface soil for antimony, PAHs, PCBs, and other site related contaminants.
2. The site owner should clean up soil lead before any residential use.
3. The site owner should collect and test more soil and ground water samples to adequately characterize the extent of contamination.
4. The site owner should not install drinking water wells.

**Public Health Action Plan**

The Department will:

- Share this report with nearby residents via a community update.
- Solicit public comments on this draft report as well as collect any health concerns and address both in a final report.
- Consider review of new data by request.

**Report Preparation**

This report was supported in part by funds provided through a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Department of Health and Human Services (DHHS). The findings and conclusions in these reports are those of the author(s) and do not necessarily represent the views of the ATSDR or the DHHS. This document has not been revised or edited to conform to ATSDR standards.

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References


Appendices
Appendix A

Tables
### Table 1. Completed Human Exposure Pathways at the National Oil Company Site

<table>
<thead>
<tr>
<th>Completed Pathway Name</th>
<th>Source</th>
<th>Environmental Media</th>
<th>Point of Exposure</th>
<th>Route of Exposure</th>
<th>Exposed Population</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Worker on-site soil ingestion</td>
<td>Waste oil disposal</td>
<td>Surface soil</td>
<td>On-site</td>
<td>Incidental ingestion</td>
<td>Workers</td>
<td>Past, present, and future</td>
</tr>
<tr>
<td>Children on-site soil ingestion</td>
<td>Waste oil disposal</td>
<td>Surface soil</td>
<td>On-site</td>
<td>Incidental ingestion</td>
<td>Children at on-site playground</td>
<td>Past, present, and future</td>
</tr>
<tr>
<td>Trespasser on-site soil ingestion</td>
<td>Waste oil disposal</td>
<td>Surface soil</td>
<td>On-site</td>
<td>Incidental ingestion</td>
<td>Trespassers</td>
<td>Past, present, and future</td>
</tr>
</tbody>
</table>
### Table 2. Potential Human Exposure Pathways at Properties Adjacent to the National Oil Company Site

<table>
<thead>
<tr>
<th>Potential Pathway Name</th>
<th>Source</th>
<th>Environmental Media</th>
<th>Point of Exposure</th>
<th>Route of Exposure</th>
<th>Exposed Population</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Private drinking water wells</td>
<td>Waste oil disposal</td>
<td>Groundwater</td>
<td>Tap water from on-site wells</td>
<td>Ingestion</td>
<td>Residents</td>
<td>Future</td>
</tr>
<tr>
<td>On-site soil ingestion</td>
<td>Waste oil disposal</td>
<td>Surface soil</td>
<td>On-site</td>
<td>Incidental ingestion</td>
<td>Residents</td>
<td>Future</td>
</tr>
<tr>
<td>Eliminated Pathway Name</td>
<td>Source</td>
<td>Environmental Media</td>
<td>Point of Exposure</td>
<td>Route of Exposure</td>
<td>Exposed Population</td>
<td></td>
</tr>
<tr>
<td>-------------------------</td>
<td>-------------------------</td>
<td>----------------------------------</td>
<td>-------------------</td>
<td>-------------------------</td>
<td>--------------------</td>
<td></td>
</tr>
<tr>
<td>On-site subsurface soil</td>
<td>Waste oil disposal</td>
<td>Sub-surface soil and sediment</td>
<td>On-site</td>
<td>Incidental ingestion</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Off-site subsurface soil</td>
<td>Waste oil disposal</td>
<td>Sub-surface soil and sediment</td>
<td>Off-site</td>
<td>Incidental ingestion</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Drinking water from municipal wells</td>
<td>Waste oil disposal</td>
<td>Deep aquifer groundwater</td>
<td>Tap water</td>
<td>Ingestion and showering</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>
Table 4. Contaminant Concentrations in On-site Surface Soil and Off-site Sediment Samples (0 to 0.5 feet deep) at the National Oil Company Site

<table>
<thead>
<tr>
<th>Contaminants</th>
<th>Concentration Range (mg/kg)</th>
<th>Location of Maximum Concentration</th>
<th>Soil Screening Guideline (mg/kg)*</th>
<th>Source of Screening Guideline</th>
<th># of Samples Above Screening Guideline/Total # Samples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antimony</td>
<td>BDL - 100</td>
<td>NO-SS-03</td>
<td>20</td>
<td>Child RMEG</td>
<td>1/8</td>
</tr>
<tr>
<td>BaP - TEQ</td>
<td>BDL – 1.97</td>
<td>NO-SD-01</td>
<td>0.096</td>
<td>CREG</td>
<td>6/8</td>
</tr>
<tr>
<td>Lead</td>
<td>BDL – 1,300</td>
<td>NO-SS-03</td>
<td>800</td>
<td>EPA Industrial PRG</td>
<td>2/8</td>
</tr>
<tr>
<td>PCB-1254</td>
<td>BDL – 6.3</td>
<td>NO-SS-03</td>
<td>1</td>
<td>Child Chronic EMEG</td>
<td>1/8</td>
</tr>
<tr>
<td>PCB - 1260</td>
<td>BDL – 1.9</td>
<td>NO-SS-05</td>
<td>1</td>
<td>Child Chronic EMEG</td>
<td>2/8</td>
</tr>
</tbody>
</table>

Source of data [NUS 1991]

Bap-TEQ = Benzo(a)Pyrene Toxicity Equivalents
BDL = below detection limits
CREG = ATSDR cancer risk evaluation guide
EMEG = ATSDR environmental media evaluation guides
PCB = polychlorinated biphenyl
PRG = preliminary remediation goals
RMEG = ATSDR reference dose media evaluation guides
mg/kg = milligrams per kilogram

* Screening guidelines only used to select chemicals for further scrutiny, not to judge the risk of illness.
Table 5. Estimated Average* Noncancer Dose (mg/kg/day) from Incidental Ingestion of Surface Soil at the National Oil Company Site

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Antimony</th>
<th>PAH as B(a)P - TEQ</th>
<th>Lead</th>
<th>PCB 1254+1260</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child Birth to &lt; 1 year</td>
<td>6.5E-04</td>
<td>1.3E-05</td>
<td>8.5E-03</td>
<td>5.3E-05</td>
</tr>
<tr>
<td>Child 1 to &lt; 2 year</td>
<td>8.8E-04</td>
<td>1.7E-05</td>
<td>1.1E-02</td>
<td>7.2E-05</td>
</tr>
<tr>
<td>Child 2 to &lt; 6 year</td>
<td>5.7E-04</td>
<td>1.1E-05</td>
<td>7.5E-03</td>
<td>4.7E-05</td>
</tr>
<tr>
<td>Child 6 to &lt; 11 year</td>
<td>3.1E-04</td>
<td>6.2E-06</td>
<td>4.1E-03</td>
<td>2.6E-05</td>
</tr>
<tr>
<td>Child 11 to &lt; 16 year</td>
<td>1.8E-04</td>
<td>3.5E-06</td>
<td>2.3E-03</td>
<td>1.4E-05</td>
</tr>
<tr>
<td>Child 16 to &lt; 21 year</td>
<td>1.4E-04</td>
<td>2.8E-06</td>
<td>1.8E-03</td>
<td>1.1E-05</td>
</tr>
<tr>
<td>Worker Scenario</td>
<td>8.9E-05</td>
<td>1.8E-06</td>
<td>1.2E-03</td>
<td>7.3E-06</td>
</tr>
<tr>
<td>Child Playground Scenario</td>
<td>2.3E-04</td>
<td>1.4E-06</td>
<td>3.0E-03</td>
<td>1.9E-05</td>
</tr>
<tr>
<td>Trespasser Scenario</td>
<td>4.9E-05</td>
<td>9.6E-07</td>
<td>6.3E-04</td>
<td>4.0E-06</td>
</tr>
<tr>
<td>Residential Scenario</td>
<td>6.3E-05</td>
<td>1.2E-06</td>
<td>8.1E-04</td>
<td>5.1E-06</td>
</tr>
<tr>
<td>ATSDR MRL</td>
<td>none</td>
<td>none</td>
<td>none</td>
<td>2.0E-05</td>
</tr>
</tbody>
</table>

Maximum Surface Soil Concentration (mg/kg)

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Concentration (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child Birth to &lt; 1 year</td>
<td>100</td>
</tr>
<tr>
<td>Child 1 to &lt; 2 year</td>
<td>1.97</td>
</tr>
<tr>
<td>Child 2 to &lt; 6 year</td>
<td>1,300</td>
</tr>
<tr>
<td>Child 6 to &lt; 11 year</td>
<td>8.2</td>
</tr>
</tbody>
</table>

Source of data [NUS 1991]

* Central tendency exposure

ATSDR = Agency for Toxic Substances and Disease Registry
PAH as B(a)P TEQ = Polycyclic Aromatic Hydrocarbon as Benzo(a)Pyrene Toxicity Equivalents
mg/kg = milligrams per kilogram
mg/kg/day = milligrams per kilogram per day
MRL = minimal risk level
PCB = polychlorinated biphenyl
Table 6. Estimated Worker Cancer Dose and Increased Risk from Incidental Ingestion of Surface Soil (0 to 0.5 feet deep) at the National Oil Company Site

<table>
<thead>
<tr>
<th>Contaminants</th>
<th>Maximum Surface Soil Concentration (mg/kg)</th>
<th>ATSDR Minimal Risk Level (mg/kg)</th>
<th>Maximum Ingestion Dose (cancer) (mg/kg/day)</th>
<th>Oral Cancer Slope Factor (mg/kg/day) (^{-1})</th>
<th>Source of Oral Cancer Slope Factor</th>
<th>Estimated Increased Cancer Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antimony</td>
<td>100</td>
<td>none</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>PAH as B(a)P - TEQ</td>
<td>1.97</td>
<td>none*</td>
<td>5.6E-07</td>
<td>7.3</td>
<td>EPA IRIS</td>
<td>4E-06</td>
</tr>
<tr>
<td>Lead</td>
<td>1,300</td>
<td>none</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>PCB - 1254 + 1260</td>
<td>8.2</td>
<td>2.0 (chronic)</td>
<td>2.3E-06</td>
<td>2.0</td>
<td>EPA IRIS</td>
<td>5E-06</td>
</tr>
</tbody>
</table>

Source of data[NUS 1991]
B(a)P TEQ = Benzo(a)Pyrene Toxicity Equivalents
EPA IRIS = U.S. Environmental Protection Agency Integrated Risk Information System [EPA 2013b]

\(mg/kg\) = milligrams per kilogram
\(mg/kg/day\) = milligrams per kilogram per day

NA = non-applicable

PCB = polychlorinated biphenyl

* = The CDC has not calculated a minimal risk level for PAHs but the maximum dose is well below the oral no adverse effect level of 1.3 \(mg/kg/day\)
Table 7. Calculated Maximum Child Blood Lead Levels (µg/dL) from Surface Soil (0 to 0.5 feet deep) at the National Oil Company Site – Child Playground and Future Residential Scenarios

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Playground Exposure Time (hours/day)</th>
<th>Playground Exposure Total Lead Uptake (µg/day)</th>
<th>Playground Exposure Calculated Blood Lead (1) (µg/dL)</th>
<th>Future Residential Exposure Time Outdoors (hours/day)</th>
<th>Future Residential Exposure Total Lead Uptake (µg/day)</th>
<th>Future Residential Exposure Calculated Blood Lead (1) (µg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5-1</td>
<td>1.0</td>
<td>12.9</td>
<td>1.8</td>
<td>1.0</td>
<td>14.9</td>
<td>7.9</td>
</tr>
<tr>
<td>1-2</td>
<td>2.0</td>
<td>19.9</td>
<td>2.1</td>
<td>2.0</td>
<td>22.9</td>
<td>9.3</td>
</tr>
<tr>
<td>2-3</td>
<td>2.0</td>
<td>20.4</td>
<td>1.9</td>
<td>3.0</td>
<td>23.6</td>
<td>8.7</td>
</tr>
<tr>
<td>3-4</td>
<td>2.0</td>
<td>20.8</td>
<td>1.8</td>
<td>4.0</td>
<td>24.1</td>
<td>8.3</td>
</tr>
<tr>
<td>4-5</td>
<td>2.0</td>
<td>16.1</td>
<td>1.4</td>
<td>4.0</td>
<td>18.9</td>
<td>6.8</td>
</tr>
<tr>
<td>5-6</td>
<td>2.0</td>
<td>14.8</td>
<td>1.2</td>
<td>4.0</td>
<td>17.3</td>
<td>5.6</td>
</tr>
<tr>
<td>6-7</td>
<td>2.0</td>
<td>14.1</td>
<td>1.0</td>
<td>4.0</td>
<td>16.6</td>
<td>4.8</td>
</tr>
</tbody>
</table>

µg/dL = micrograms per deciliter

(1) = Health scientists have not identified a safe blood level in children. CDC recommends public health actions at blood lead levels above 5 µg/dL [CDC 2016].

**Bold** numbers are those that exceed the CDC public health action blood lead level of 5 µg/dL.
Table 8. Estimated Trespasser Cancer Dose and Increased Risk from Incidental Ingestion of Surface Soil (0 to 0.5 feet deep) at the National Oil Company Site

<table>
<thead>
<tr>
<th>Contaminants</th>
<th>Maximum Concentration (mg/kg)</th>
<th>ATSDR Minimal Risk Level (mg/kg)</th>
<th>Maximum Soil Ingestion Dose (cancer) (mg/kg/day)</th>
<th>Oral Cancer Slope Factor (mg/kg/day) $^{-1}$</th>
<th>Source of Oral Cancer Slope Factor</th>
<th>Estimated Increased Cancer Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antimony</td>
<td>100</td>
<td>none *</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>PAH as B(a)P - TEQ</td>
<td>1.97</td>
<td>none *</td>
<td>1.2E-07</td>
<td>7.3</td>
<td>EPA IRIS</td>
<td>9E-07</td>
</tr>
<tr>
<td>Lead</td>
<td>1,300</td>
<td>none</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>PCB - 1254 + 1260</td>
<td>8.2</td>
<td>2.0E-05 (chronic)</td>
<td>5.1E-07</td>
<td>2.0</td>
<td>EPA IRIS</td>
<td>1E-06</td>
</tr>
</tbody>
</table>

Source of data [NUS 1991]

B(a)P TEQ = Benzo(a)Pyrene Toxicity Equivalents

EPA IRIS = U.S. Environmental Protection Agency Integrated Risk Information System [EPA 2013b]

mg/kg = milligrams per kilogram

mg/kg/day = milligrams per kilogram per day

NA = non-applicable

PCB = polychlorinated biphenyl

* = The CDC has not calculated a minimal risk level for PAHs but the maximum dose is well below the oral no adverse effect level.
Table 9. Estimated Resident Cancer Dose and Increased Risk from Incidental Ingestion of Surface Soil (0 to 0.5 feet deep) at the National Oil Company Site

<table>
<thead>
<tr>
<th>Contaminants</th>
<th>Maximum Concentration (mg/kg)</th>
<th>ATSDR Minimal Risk Level (mg/kg)</th>
<th>Maximum Soil Ingestion Dose (cancer) (mg/kg/day)</th>
<th>Oral Cancer Slope Factor (mg/kg/day)⁻¹</th>
<th>Source of Oral Cancer Slope Factor</th>
<th>Estimated Increased Cancer Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antimony</td>
<td>100</td>
<td>none (¹)</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>PAH as B(a)P - TEQ</td>
<td>1.97</td>
<td>none (¹)</td>
<td>5.2E-07</td>
<td>7.3</td>
<td>EPA IRIS</td>
<td>1E-05</td>
</tr>
<tr>
<td>Lead</td>
<td>1,300</td>
<td>none (²)</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>PCB - 1254 + 1260</td>
<td>8.2</td>
<td>2.0 (chronic)</td>
<td>2.2E-06</td>
<td>2.0</td>
<td>EPA IRIS</td>
<td>2E-05</td>
</tr>
</tbody>
</table>

Source of data [NUS 1991]
B(a)P TEQ = Benzo(a)Pyrene Toxicity Equivalents
EPA IRIS = U.S. Environmental Protection Agency Integrated Risk Information System [EPA 2013b]
mg/kg = milligrams per kilogram
mg/kg/day = milligrams per kilogram per day
NA = non-applicable
PCB = polychlorinated biphenyl
(¹) = The CDC has not calculated a minimal risk level for PAHs but the maximum dose is well below the oral no adverse effect level of 1.3 mg/kg/day
(²) = Minimal risk levels for lead have not been established but the CDC considers blood lead levels in children above 5µg/dL to be elevated
<table>
<thead>
<tr>
<th>Contaminants</th>
<th>Concentration Range (mg/L)</th>
<th>Location of Maximum Concentration</th>
<th>Screening Guideline (mg/L)*</th>
<th>Source of Screening Guideline</th>
<th># of Samples Above Screening Guideline/Total # Samples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aluminum</td>
<td>23 – 100</td>
<td>NO-TW-02</td>
<td>10</td>
<td>Child Chronic EMEG</td>
<td>3/3</td>
</tr>
<tr>
<td>Arsenic</td>
<td>BDL – 0.022 J</td>
<td>NO-TW-03</td>
<td>0.000023</td>
<td>CREG</td>
<td>1/3</td>
</tr>
<tr>
<td>Benzene</td>
<td>BDL – 0.110</td>
<td>NO-TW-03</td>
<td>0.00064</td>
<td>CREG</td>
<td>1/3</td>
</tr>
<tr>
<td>Cadmium</td>
<td>BDL – 0.09(1) (0.049(2))</td>
<td>NO-TW-01(1) (02(2))</td>
<td>0.001</td>
<td>Child Chronic EMEG</td>
<td>3/3</td>
</tr>
<tr>
<td>Lead</td>
<td>0.018 -0.049</td>
<td>NO-TW-02</td>
<td>0.015</td>
<td>MCL Action Level</td>
<td>3/3</td>
</tr>
<tr>
<td>Trichloroethylene</td>
<td>BDL – 0.012</td>
<td>NO-TW-03</td>
<td>0.00076</td>
<td>CREG</td>
<td>1/3</td>
</tr>
<tr>
<td>Vanadium</td>
<td>BDL – 0.15(1) (0.14(2))</td>
<td>NO-TW-01(1) (02(2))</td>
<td>0.1</td>
<td>Child Intermediate EMEG</td>
<td>2/3</td>
</tr>
<tr>
<td>Vinyl Chloride</td>
<td>BDL – 0.12</td>
<td>NO-TW-03</td>
<td>0.000025</td>
<td>CREG</td>
<td>1/3</td>
</tr>
</tbody>
</table>

Source of data [NUS 1991]
BDL = below detection limits
CREG = ATSDR cancer risk evaluation guide
J = estimated value
mg/L = milligrams per liter
(1) = off-site background sample
(2) = maximum on-site sample
* Screening guidelines only used to select chemicals for further scrutiny, not to judge the risk of illness.
<table>
<thead>
<tr>
<th>Contaminants</th>
<th>Aluminum</th>
<th>Arsenic(^{(1)})</th>
<th>Benzene</th>
<th>Cadmium</th>
<th>Lead</th>
<th>Trichloroethylene</th>
<th>Vanadium</th>
<th>Vinyl Chloride</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child Birth to &lt; 1 year</td>
<td>6.46</td>
<td>8.4E-04</td>
<td>7.8E-04</td>
<td>3.2E-03</td>
<td>3.2E-03</td>
<td>7.8E-04</td>
<td>9.0E-03</td>
<td>7.8E-03</td>
</tr>
<tr>
<td>Child 1 to &lt; 2 year</td>
<td>2.70</td>
<td>3.5E-04</td>
<td>3.2E-04</td>
<td>1.3E-03</td>
<td>1.3E-03</td>
<td>3.2E-04</td>
<td>3.8E-03</td>
<td>3.2E-03</td>
</tr>
<tr>
<td>Child 2 to &lt; 6 year</td>
<td>2.16</td>
<td>2.8E-04</td>
<td>2.6E-04</td>
<td>1.1E-03</td>
<td>1.1E-03</td>
<td>2.6E-04</td>
<td>3.0E-03</td>
<td>2.6E-03</td>
</tr>
<tr>
<td>Child 6 to &lt; 11 year</td>
<td>1.61</td>
<td>2.1E-04</td>
<td>1.9E-04</td>
<td>7.9E-04</td>
<td>7.9E-04</td>
<td>1.9E-04</td>
<td>2.2E-03</td>
<td>1.9E-03</td>
</tr>
<tr>
<td>Child 11 to &lt;16 year</td>
<td>1.12</td>
<td>1.5E-04</td>
<td>1.3E-04</td>
<td>5.5E-04</td>
<td>5.5E-04</td>
<td>1.3E-04</td>
<td>1.6E-03</td>
<td>1.3E-03</td>
</tr>
<tr>
<td>Child 16 to &lt;21 year</td>
<td>1.08</td>
<td>1.4E-04</td>
<td>1.3E-04</td>
<td>5.3E-04</td>
<td>5.3E-04</td>
<td>1.3E-04</td>
<td>1.5E-03</td>
<td>1.3E-03</td>
</tr>
<tr>
<td>Adults ≥ 21 year</td>
<td>1.53</td>
<td>2.0E-04</td>
<td>1.8E-04</td>
<td>7.5E-04</td>
<td>7.5E-04</td>
<td>1.8E-04</td>
<td>2.1E-03</td>
<td>1.8E-03</td>
</tr>
<tr>
<td><strong>ATSDR MRL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(chronic)</td>
<td>1.0</td>
<td>3.0E-04</td>
<td>5.0E-04</td>
<td>1.0E-04</td>
<td>none(^{(1)})</td>
<td>5.0E-04 (chronic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(chronic)</td>
<td>(chronic)</td>
<td>(chronic)</td>
<td>(chronic)</td>
<td>(chronic)</td>
<td>(MCL = 0.015 mg/L)</td>
<td>(chronic)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(chronic)</td>
<td>none(^{(1)})</td>
<td>7.8E-03 (2)</td>
<td>none (^{(2)})</td>
<td>50(^{(2)})</td>
<td>7.0E-01(2)</td>
<td>1.7(^{(2)})</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Maximum Concentration (mg/L)</strong></td>
<td>100</td>
<td>.022 J</td>
<td>0.012</td>
<td>0.049</td>
<td>0.049</td>
<td>0.012</td>
<td>0.14</td>
<td>0.12</td>
</tr>
</tbody>
</table>

Source of data [NUS 1991]
ATSDR = Agency for Toxic Substances and Disease Registry
mg/L = milligrams per liter
mg/kg/day = milligrams per kilogram per day
MRL = minimal risk level
MCL = maximum contaminant level
\(^{(1)}\) = Minimal risk levels for lead have not been established. The DEP has established a MCL of 0.015 mg/L for lead in drinking water [DEP 2012].
\(^{(2)}\) = off-site background sample
\(^{(3)}\) = maximum on-site sample
Table 12. Estimated Cancer Dose and Increased Risk from Ingestion of Groundwater at the National Oil Company Site

<table>
<thead>
<tr>
<th>Contaminants</th>
<th>Maximum Concentration (mg/L)</th>
<th>ATSDR Minimal Risk Level (mg/L)</th>
<th>Maximum Groundwater Ingestion Dose (cancer) (mg/kg/day)</th>
<th>Oral Cancer Slope Factor (mg/kg/day)</th>
<th>Source of Oral Cancer Slope Factor</th>
<th>Estimated Increased Cancer Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aluminum</td>
<td>100</td>
<td>1 (Chronic)</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA*</td>
</tr>
<tr>
<td>Arsenic (1)</td>
<td>.022 J</td>
<td>0.0003 (Chronic)</td>
<td>1.4 E-04</td>
<td>1.5</td>
<td>EPA IRIS</td>
<td>3 E-05</td>
</tr>
<tr>
<td>Benzene</td>
<td>0.012</td>
<td>0.0005 (Chronic)</td>
<td>7.8 E-05</td>
<td>0.055</td>
<td>EPA IRIS</td>
<td>5 E-06</td>
</tr>
<tr>
<td>Cadmium</td>
<td>0.049</td>
<td>0.0001 (Chronic)</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA*</td>
</tr>
<tr>
<td>Lead</td>
<td>0.049</td>
<td>none** (MCL = 0.015 mg/L)</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA*</td>
</tr>
<tr>
<td>Trichloroethylene</td>
<td>0.012</td>
<td>0.0005 (Chronic)</td>
<td>7.8 E-05</td>
<td>0.046</td>
<td>EPA IRIS</td>
<td>1 E-05</td>
</tr>
<tr>
<td>Vanadium</td>
<td>0.14</td>
<td>0.01 (Intermediate)</td>
<td>NA</td>
<td>none</td>
<td>NA</td>
<td>NA*</td>
</tr>
<tr>
<td>Vinyl Chloride</td>
<td>0.12</td>
<td>0.003 (Chronic)</td>
<td>7.8 E-04</td>
<td>0.72</td>
<td>EPA IRIS</td>
<td>6 E-04</td>
</tr>
</tbody>
</table>

Source of data [NUS 1991]

EPA IRIS = U.S. Environmental Protection Agency Integrated Risk Information System [EPA 2013b]

J = estimated value

mg/kg = milligrams per kilogram
mg/kg/day = milligrams per kilogram per day
mg/L = milligrams per liter

NA = non-applicable

* = an estimated cancer risk cannot be calculated if the cancer slope factor has not been established

** = Minimal risk levels for lead have not been established but the CDC considers blood lead levels in children above 5µg/dL to be elevated
Appendix B

Figures
Figure 1. National Oil Company Site Location
Figure 2. National Oil Company Site and Surrounding Properties
Figure 3. National Oil Company Sample Locations
Appendix C

PAH Toxicity Equivalency Factors
# PAH Toxicity Equivalency Factors (TEF)

<table>
<thead>
<tr>
<th>Compound</th>
<th>TEF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acenaphthene</td>
<td>0.001</td>
</tr>
<tr>
<td>Acenaphthylene</td>
<td>0.001</td>
</tr>
<tr>
<td>Anthracene</td>
<td>0.01</td>
</tr>
<tr>
<td>Benzo(a)anthracene</td>
<td>0.1</td>
</tr>
<tr>
<td>Benzo(a)pyrene</td>
<td>1</td>
</tr>
<tr>
<td>Benzo(b)fluoranthene</td>
<td>0.1</td>
</tr>
<tr>
<td>Benzo(k)fluoranthene</td>
<td>0.1</td>
</tr>
<tr>
<td>Benzo(g,h,i)perylene</td>
<td>0.01</td>
</tr>
<tr>
<td>Chrysene</td>
<td>0.01</td>
</tr>
<tr>
<td>Dibenz(a,h)anthracene</td>
<td>5</td>
</tr>
<tr>
<td>Fluoranthene</td>
<td>0.001</td>
</tr>
<tr>
<td>Fluorene</td>
<td>0.001</td>
</tr>
<tr>
<td>Indeno(1,2,3-cd)pyrene</td>
<td>0.1</td>
</tr>
<tr>
<td>Phenanthrene</td>
<td>0.001</td>
</tr>
<tr>
<td>Pyrene</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: Data from Toxicological Profile for Polycyclic Aromatic Hydrocarbons [ATSDR 1995]
Glossary

Absorption
The process of taking in. For a person or 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).

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

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

Cancer risk
A theoretical risk of 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.

Chronic
Occurring over a long time (more than 1 year) (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).

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).
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.

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).

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.

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.

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 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.

**Groundwater**
Water beneath the earth’s surface in the spaces between soil particles and between rock surfaces (compare with surface water).

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

**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.

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

**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).

**mg/kg**
Milligram per kilogram.
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.

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.

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).

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

Receptor population
People who could come into contact with hazardous substances (see exposure pathway).

Registry
A systematic collection of information on persons exposed to a specific substance or having specific diseases.

Risk
The probability that something will cause injury or harm.

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).
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.

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.

Substance
A chemical.

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

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.

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