

Health Consultation

LINCOLN PARK COMPLEX
DURRS NEIGHBORHOOD (OFF-SITE) SOIL

FT. LAUDERDALE, BROWARD COUNTY, FLORIDA

EPA FACILITY ID: FLN000407550

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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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Prepared By:

Florida Department of Health
Bureau of Community Environmental Health
Under a Cooperative Agreement with the
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

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Summary

In this report, the Florida Department of Health reviewed 2004 and 2006 soil test results from the Durrs neighborhood at the request of the Florida Department of Environmental Protection. This neighborhood surrounds the 16.5-acre Lincoln Park Complex in Ft. Lauderdale, Florida. Previously, Florida DOH reviewed blood-lead test results and 2004 soil and groundwater test results.

The City of Ft. Lauderdale completely fenced the former incinerator area, cleaned contaminated soil on the “One-stop” property, and capped contaminated soil on the Lincoln Park playground. As a result, there are no current exposure pathways to soil contaminants on the Lincoln Park Complex site proper, as opposed to the potential for exposures to soil chemicals offsite in Durrs neighborhood yards and road right-of-ways, and other non-residential properties.

Chemicals found in off-site soil could have originated on the Lincoln Park Complex site as incinerator ash, wastewater treatment plant sludge, or from wastewater treatment plant flooding. Primary or secondary sources of chemicals measured in off-site soil could be residues from gasoline or diesel fuel combustion, asphalt roads or roofing materials, and residues from residential burning.

Although offsite soil testing is limited, the locations and levels of the chemicals measured seem to indicate incinerator ash may have been locally used as fill, because the highest arsenic and lead levels were measured in subsurface soil. In contrast, the highest dioxin and polychlorinated aromatic hydrocarbon (PAH) levels were measured in surface soil. The occurrence of the highest dioxin levels near the site and in surficial soil may indicate dioxins moved off the site as residues when the wastewater treatment plant flooded. PAH levels may not be related to the site; their levels tended to be only slightly elevated and were highest near roads in surface soil, additionally the highest PAH levels were not measured nearest the site.

The extent of soil contamination in the Durrs neighborhood needs to be adequately determined. Residents who dig (or have dug) in their yards may bring (or may have brought) **subsurface** incinerator residues to the surface where they are more likely to be contacted. Once the areas and depths of soil contamination are known, measures can be taken to prevent contact with soils having elevated chemical contaminant levels.

Although elevated chemical levels have not been measured in the surface soils of properties that are currently residential, persons walking in bare feet or sandals could contact contaminated **surface** soil on vacant properties or on rights-of ways near the Lincoln Park Complex that have elevated contamination, especially children that might then accidentally eat (ingest) the contacted soil. Depending on where soil contaminants are measured and at what levels, removal operations, deed restrictions, and/or engineering controls can be used to prevent future chemical exposures, for workers on City property and areas accessing utility lines, and for residents.

Until the full extent of soil contamination is determined, The Florida DOH recommends area residents and workers should avoid contact with soil that contains ash, glass, or metal pieces that might allow them to accidentally eat (ingest) soil and inhale dust. In addition as a precaution, residents should:

- use safe gardening practices (Appendix C); and
- only grow edible fruits and vegetables using raised beds with clean soil or compost.

Purpose

The Florida Department of Health (DOH) evaluates the public health significance of environmental contamination through a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR) in Atlanta, Georgia. In March 2005, the Florida DOH reported the results of blood lead testing of children attending the former Lincoln Park School at the Lincoln Park Complex in Ft. Lauderdale. In October 2005, Florida DOH assessed the public health threat from soil and groundwater in the Durrs neighborhood surrounding the Lincoln Park Complex. At the request of the Florida Department of Environmental Protection (DEP), Florida DOH reviewed the additional 2006 soil test results together with the 2004 Durrs neighborhood soil testing results in this report.

Background

The 16.5-acre Lincoln Park Complex is in a commercial and light industrial area north of Sistrunk Boulevard between NW 18th Avenue and Interstate 95, in Ft. Lauderdale (Broward County), Florida (Figure 1). The complex includes the former City of Ft. Lauderdale (City) municipal incinerator and later a waste water treatment plant, the former Lincoln Park School (now the One-stop Shop for City of Ft. Lauderdale permits), and the Lincoln Park playground. Remediation of the playground and One-stop properties and the fencing of the incinerator propriety prevent exposure to the on-site soil contamination.

At a November 2003 public meeting, the Florida Department of Environmental Protection (DEP) shared the results of environmental testing in the Lincoln Park Complex with nearby residents. Florida DEP found layers of ash in soil deeper than one to two feet. Residents living north of the complex reported finding similar buried debris in their yards. In response, Florida DEP tested 30 surface soil (0-3") and 10 subsurface residential soil samples north of the complex in July 2004.

In a March 2005 health consultation report, Florida DOH found the blood lead levels in 40 children and three young adults attending or playing at the former Lincoln Park School were below the CDC guidelines for intervention for lead-poisoned individuals (ATSDR 2005d).

In an October 2005 public health assessment report, Florida DOH reviewed all of the 2004 soil and groundwater test data for the Lincoln Park Complex (ATSDR 2005a). We found the site posed "no apparent public health hazard," based on the information available at that time. We also found the health threat from past exposures was "indeterminate." In the public health assessment, Florida DOH addressed arsenic, copper, dioxins and polycyclic aromatic hydrocarbons (PAHs) in on-site surface soil (0-6"); and arsenic, copper, lead, and PAHs in on-site subsurface soil (3-24"). We concluded that recent exposures to surface soil are unlikely to have caused non-cancer illness. We also found "no apparent" increased theoretical cancer risk using the highest levels of all the chemicals measured in surface soil on the site.

In 2005, the City of Ft. Lauderdale secured grant funding for additional off-site soil testing. In February and March 2006, the City funded Florida DEP's contractor to test 25 more surface and subsurface soil samples in the surrounding Durrs neighborhood. In June 2006, the Florida DEP asked the Florida DOH to evaluate these soil test results. In this health consultation, we evaluate these results in combination with the 2004 soil test results.

In 2000, about 19,643 persons lived within a 1-mile radius of the site. Approximately 39% were 19 years of age or less. Approximately 88% were black, 8% were white, and less than 3% were

Latino/Hispanic, American Indian/Alaska Native, Asian/Pacific Islander, and all other racial/ethnic groups made up about 1% of the population (US Census Bureau 2000).

Community Health Concerns

Some residents in the Durrs neighborhood north of the Lincoln Park complex are concerned about the potential for contaminants in debris and waste materials they found buried in their yards.

Discussion

In this report, Florida DOH evaluates 2004 and 2006 test results from Durrs neighborhood soil. Florida DOH estimated soil ingestion and dust inhalation levels based on studies that measured people's actual exposure levels via soil dust and ingestion using a computer program (Risk Assistant 1.1). We are not aware of any other completed exposure pathways. Site-related contamination has not been traced to off-site surface water.

Although the City's contractor recently identified limited groundwater contamination in a monitoring well on the southeastern part of the site, municipal water is currently used by homes and businesses in the area, and drinking water wells are not likely to have been a past exposure pathway. However, some residents currently use irrigation wells. Although site-related contamination has not been measured in off-site groundwater, irrigation wells may be shallow and could pull in surface water. Because surface water may contain bacteria, DOH recommends people should not drink water from shallow irrigation wells or use it for bathing, cleaning food contact surfaces (like grills, dishes, or grilling utensils), or for rinsing food.

In July 2004, Florida DEP's contractor collected surface soil samples at 30 locations and subsurface soil samples at 10 of those locations. They analyzed all the surface samples for polycyclic aromatic hydrocarbons (PAHs), metals and dioxins. They analyzed all the subsurface samples for PAHs and metals and analyzed five samples for dioxins. They collected twenty-four **surface** soil samples on residential properties, half on City-owned, vacant or right-of-way properties which we designate "other" properties. They collected 4 **subsurface** soil samples on residential properties, and 6 on "other" properties.

In February and March 2006, Florida DEP's contractor collected **surface** and **subsurface** soil samples at 25 locations. They analyzed all the surface samples for PAHs and metals, and 16 of the 25 samples for dioxins. They analyzed all the subsurface samples for PAHs and metals, and 15 of the 25 samples for dioxins. They collected nineteen **surface** and **subsurface** soil samples on residential properties, and 6 on "other" properties.

Levels of arsenic, dioxins, lead, and PAHs in some Durrs neighborhood soil samples were above ATSDR screening values. Arsenic, dioxins, lead and PAHs are chemicals that could be associated with incinerator ash. Use of chlorine in wastewater treatment can also produce dioxins. We discuss the public health threat of these contaminants below. The highest soil concentration of barium is below the ATSDR screening values and is thus not likely to cause illness.

We separated the soil data into yards currently being used as residences and "other" areas (vacant lots, City property, and rights-of-way). In general, residential yards had lower contaminant levels than "other" areas. Surface soil (0-3 inches deep) in residential yards had lower levels of arsenic, lead, and dioxins than "other" areas. Subsurface soil (3-24 inches deep)

in residential yards had lower levels of dioxins and lead than “other” areas. The highest level of arsenic, however, was in subsurface soils in a residential yard.

Figures 2 through 5 (Appendix A) show the soil sample locations where chemicals were measured above the ATSDR 1 in 1 million additional cancer risk screening guideline (CREGs)[†]. Florida DEP found arsenic, dioxins, and lead immediately north and east of the site with higher concentrations in subsurface than surface soil. Conversely, they found PAHs scattered throughout the area with higher concentrations in surface than subsurface soil. The distribution and depth of PAHs in soil suggest they may be associated with road asphalt, roofing tar or shingles, and vehicle emissions rather than incinerator ash. PAHs also tended to have less elevated levels, relative to their screening values.

Distribution of Arsenic and Dioxin in Durrs Neighborhood Surface/Subsurface Soil

Surface soil (0-3”)	Arsenic and dioxins >1 in 1 million cancer-risk soil value 38% (21/55)
Subsurface soil (3-24”)	Arsenic and dioxins >1 in 1 million cancer-risk soil value 49% (17/35)

Although contractors for Florida DEP have tested subsurface soil in 35 locations, the full extent of soil contamination in the Durrs neighborhood north and east of the site has still not been determined. Current data suggests that soil contamination may extend further to the north and east of the Lincoln Park Complex. Additional soil testing between NW 7th St and NW 7th Place, and one block east of NW 18th Place between NW 6th Place and NW 7th Place (Figure 6) is necessary to assess the public health risk fully. Until the full extent of soil contamination is determined, residents living north and east of the Lincoln Park Complex (Figure 6) should avoid contact with soil that contains ash, glass, or metal pieces. Residents in this area that have ash, glass, or metal pieces in their soil should use the safe gardening practices outlined in Appendix C. Residents with ash, glass, or metal pieces in their soil should only grow edible fruits and vegetables using raised beds with clean soil or compost.

Public Health Implications

In this health consultation, we evaluate the risk of illness from exposure to soil in the surrounding Durrs neighborhood via incidental ingestion or inhalation of air-borne dust. We use a computer program (Risk Assistant 1.1) to estimate soil ingestion and dust inhalation levels.

Florida DOH evaluates chemical exposures by estimating daily doses for children and adults (Tables 5a, 5b, 6a, and 6b). A dose is an amount of chemical per body weight per day. Florida DOH compares estimated doses to amounts having known health effects from animal studies or from human medical reports (Tables 8 & 9). We use the units of milligrams (mg) of contaminant per kilogram (kg) of body weight per day (mg/kg/day). A milligram is 1/1,000 of a gram (a gram weighs about as much as a small raisin or paper clip); a kilogram is approximately 2 pounds.

[†] **CREG:** ATSDR’s Cancer Risk Evaluation Guide. A concentration in air, water, or soil (or other environmental media), which is derived from EPA’s cancer slope factor and carcinogenic risk of 1x10⁻⁶ for oral exposure. It is the concentration that would be expected to cause no more than one excess cancer in a million persons exposed over a lifetime.

The following sections describe the relationships of the doses we calculated for the highest measured soil concentrations to health effects at the lowest doses known from human medical reports or animal studies. For each chemical, we considered:

- Child and adult exposures
- Inhalation and ingestion exposures
- Residential and “other” location exposures
- Surface and subsurface soil exposures

Compared to ingestion, the estimated dose for inhalation was insignificant. This means that, if the ingestion and inhalation doses are added together, the inhalation doses insignificantly increase the entire exposure dose. Therefore, we only considered the ingestion doses for exposure. **Because we considered so many exposure scenarios (Tables 8 and 9), we compare only the highest estimated doses with the lowest doses known to cause illness in the following sections. Similarly, for cancer causing chemicals we discuss only the largest theoretical increased cancer risk.** We assumed residents are exposed to subsurface soil (3-24 inches deep) although routine contact with subsurface soil is unlikely.

Arsenic

Numerous medical studies document adverse health effects of long-term ingestion of arsenic-contaminated water. We compare the highest estimated daily soil dose for Durrs neighborhood children and adults to the results of these studies. Recent animal studies indicate that mammals absorb between a quarter and a third of the arsenic that they ingest with their food, which may mean that soil ingestion results in a lower absorbed dose of arsenic than would result from arsenic-contaminated water (DEP 2005).

The highest estimated arsenic dose was for a child’s daily ingestion of residential subsurface soil. This dose, 0.0007 mg/kg/day, is 1/3 the dose associated with cerebrovascular disease and interruption of the blood supply to the brain (resulting in brain damage). This dose is also about 1/3 the dose associated with adverse skin effects known from contaminated drinking water. While recent studies (Wasserman et al. 2004) have shown children’s mental deficits beginning at this dose, daily exposure to subsurface soil is unlikely and all the other estimated exposure levels are lower. The highest surface soil dose (for a non-residential property) was about half the subsurface dose; for residential property, the highest measured level was 10 times lower than the highest-measured subsurface dose.

Based on limited off-site sampling, it appears that if incinerator ash was used as fill, it may generally still be buried; therefore the assumptions we made when calculating doses for daily, long-term (chronic) exposures probably **are not being met with subsurface soil**. Florida DOH’s primary motivation for estimating the likelihood of adverse health effects for chronic exposures at the estimated dose levels was to demonstrate the need for determining where offsite contaminants occur so that appropriate measures are taken to prevent potential future exposures, including in areas with contaminated subsurface soil. Depending on where soil contaminants are measured and at what levels, removal operations, deed restrictions, and/or engineering controls can be used to prevent future chemical exposures, for workers on City property and for residents.

Although chronic exposures to subsurface soils are currently unlikely, the theoretical increased cancer risk for an adult’s daily exposure to arsenic in residential subsurface soil is 5 in 100,000,

between “low” and “no apparent increased risk”. Different cancers are associated with different arsenic doses. From lowest to highest dose, chronic arsenic exposures in people have been linked to lung cancer, basal and squamous cell skin cancers, liver cancer (haemangioendothelioma), urinary tract cancers (bladder, kidney, ureter and all urethral cancers), and intraepidermal cancers. Intraepidermal is the name for the early pre-invasive form of squamous cell skin cancer (ATSDR 2006).

Dioxin

Dioxins are a family of chemicals with similar structures and differing toxicities. The toxicities of each congener (chemical family member) are related to the most well studied member (2,3,7,8-dibenzo-p-dioxin) and they are added together to give a toxicity equivalent (TEQ) for the entire family. Dioxins can be produced when materials containing chlorine such as plastics are burned. Primary and secondary treatment of sewage and wastewater as well as the use of oxidation ponds also creates dioxins. Municipal garbage incineration, burning of yard trash and wastewater treatment may have contributed to the dioxin found in soil near the Lincoln Park Complex.

The highest dioxin TEQ dose was estimated for children via surface soil ingestion at one of the “other” properties. Because this dose was 40 times lower than the dose associated with altered developmental, social, and reproductive effects in a long-term study of rhesus monkeys fed dioxins in their food, and children’s daily exposures to non-residential property soils are less likely to occur daily, non-cancer health effects from the measured dioxin levels are unlikely. DEP’s contractors measured dioxins at lower levels in surface soil at residential properties and in subsurface soils, see Tables 8 and 9, Appendix B.

While daily long-term exposures might be unlikely to surface soil dioxins on “other” properties, the theoretical increased cancer risk for an adult’s daily exposure is 2 in 100,000, or “no apparent increased risk”. For specific information addressing dioxin and cancer links, see Tables 8 and 9.

Lead

For lead, estimated blood levels more accurately predict health effects than traditional dose estimates. We used a simple model (ATSDR 2006) to estimate blood lead levels from exposure to the highest levels of lead in Durrs neighborhood soil (Table 1). This model takes into account children and adults’ exposures to lead from sources other than soil. Using this model, DOH assumed people might be exposed to lead-contaminated soil 8 hours per day. While this might be an exceptional assumption for other parts of the United States, it might be reasonable for persons not working outside the home, given the enjoyable climate and weather in Ft. Lauderdale. Table 7 (Appendix B) lists other model assumptions.

Table 1. Modeled (Estimated) Blood Lead Levels

Exposure	Child [†] Blood Lead (µg/dl)	Adult [†] Blood Lead (µg/dl)
Residential surface soil (maximum = 290 mg/kg)	1.5 – 3.3	0.8 – 2.9
“Other” surface soil (maximum = 330 mg/kg)	1.5 – 3.6	0.9 – 3.2
Residential subsurface soil (maximum = 870 mg/kg)	2.6 – 7.2	2.0 – 6.8
“Other” subsurface soil (maximum = 4,500 mg/kg)	9.8 – 31.1	9.2 – 30.7

mg/kg = milligrams per kilogram or parts per million (ppm)

µg/dl = micrograms per deciliter

Current Centers for Disease Control Guidelines call for intervention when people’s blood lead levels (BLLs) are above 10 µg/dl. Intervention measures include determining where the person is contacting lead in their environments and abating that source. The BLLs FDOH modeled from the highest residential surface (290 ppm) and subsurface (870 ppm) soil lead levels are below this intervention BLL. Only the non-residential subsurface soil contained lead (4,500 ppm) at a level that might increase children’s or adult’s BLLs over this guideline; however, since this soil is non-residential and subsurface, **chronic exposures are currently unlikely.**

We discuss the potential for adverse health effects from chronic exposures to soil *the model shows might be below CDC’s intervention levels*, because:

- the adverse health effects documented for low blood-lead levels include conditions that could exist for other reasons in the community, therefore chronic exposures to soil with lead might add to the causes of these conditions,
- off-site soil testing is incomplete, and
- we saw two gardens when we took residents’ soil test results door-to-door.

Chronic exposures to the highest lead level measured in residential surface soil (290 mg/kg) might result in BLLs between 1.5 and 2.9 µg/dl. In children, very low BLLs decreased the activity of an enzyme (ALAD) necessary for heme synthesis (no blood lead threshold level, Roels & Lauwerys 1987). Decreased heme synthesis can lead to anemia. Neurological and immunological effects occur in children at relatively low levels of exposure (1-17 µg/dl, Altmann et al. 1998; and 1-45 µg/dl Winneke et al. 1994). A BLL average of 2.3 µg/dl increased blood pressure in adults (Den Hond et al. 2002).

Chronic exposures to the highest lead level measured in residential subsurface soil (870 mg/kg) may *currently* be much less likely than exposures to surface soil; however, if these soils were ever dug up, chronic exposures to them could have greater health impact because the lead level is higher. If excavated, chronic exposures to soil with the highest lead level might result in BLLs between 2.6 and 6.8 µg/dl. In children, BLLs of:

- 5.4 µg/dl (Chiodo et al. 2001) were linked with decrements in attention, executive function, and visual-motor integration;
- 7.7 µg/dl (Canfield et al. 2003) were linked with decline of 7.4 IQ points, and

[†] Most of the modeled levels are below Centers for Disease Control (CDC) guidelines of 10 µg/ dl for intervention when patients’ actual blood lead levels are tested, adverse health effects are documented at blood lead levels below this intervention guideline level.

- 7-80 µg/dl (Angle and McIntire 1978; Angle et al. 1982) were linked with decreases in enzymes involved in cellular-level metabolism.

In adults, BLLs of:

- 3.3 µg/dl (mean, Muntner et al. 2003) were linked to decreased kidney function,
- 4.5 µg/dl (mean, Gennart et al. 1992) were linked to impaired cognitive performance (measurable, objective mental processes),
- ≥ 5.1 µg/dl (Torres-Sanchez et al. 1999) were linked to increased pre-term births to exposed mothers.

These modeled levels for residential subsurface soil do not represent levels people are likely to be exposed to currently, especially on a daily basis. Rather, they represent the potential for exposure, potential that may not be recognized by the community, and potential that may become more likely if residents dig on their property. For example, if residents plant shrubs and trees this might result in greater exposure potentials later, if contaminated buried materials are brought to the surface. The higher exposure levels that might come from currently buried materials adds urgency to the need to characterize offsite areas with incinerator ash and the need for implementation of mechanisms to prevent future exposures.

Currently, daily exposures to subsurface soil (the highest measured lead level was 4,500 ppm) on vacant properties, City-owned properties, or road right-of-ways, **probably would be less likely than the levels we modeled**^Ω and would probably be work-related. However, if these materials were ever excavated, chronic exposures could result in the highest modeled BLLs (9.8-30.7 µg/dl). As with residential subsurface soil, we modeled potential BLLs for chronic exposures to show that the areas with contaminated soils need to be identified (and addressed), to anticipate and prevent potential future exposures. In children, in addition to the results described above for lower BLLs, BLLs of:

- 11.9 µg/dl (Bhattacharya et al. 1993) were linked with postural disequilibrium (dizziness when standing);
- 12-120 µg/dl (Mahaffey et al. 1982, Rosen et al. 1989) were linked with decreased Vitamin D metabolism, and
- 12-17 µg/dl (Bornschein et al. 1989, McMichael et al. 1996, Moore et al. 1982, Ward et al. 1987, Wibberly et al. 1977) were linked reduced birth weight and/or reduced gestational age, and increased incidence of stillbirth and neonatal death.

In adults, BLLs of:

- > 10 µg/dl (mean, Muntner et al. 2003) were linked to increased incidence of miscarriages and stillbirths, and
- 36 µg/dl (mean, Chia et al. 1992) were linked with postural disequilibrium (dizziness when standing).

Blood lead levels above 10 µg/dl have not been confirmed for residents near the Lincoln Park Complex site. The Broward County Health Department offered blood-lead testing in April 2004 to anyone who was concerned they might have had exposure to lead-contaminated soil associated with the Lincoln Park Complex site. The County Health Department publicized this free testing (for children six years of age and younger, living in the 33311 zip code) through a press release to major and community media outlets. Broward County Health Department staff provided the testing at the Sunrise Health Center—Edgar P. Mills Multipurpose Center at 900

^Ω Our model assumed daily exposure, for eight hours a day, a scenario unlikely with subsurface soil, but deliberately done to show why future exposures to this buried soil should be prevented.

NW 31st Avenue in Fort Lauderdale. Testing was available on Wednesday April 7, 2004 from 8:00 a.m. to 11:00 a.m. and Thursday, April 8, 2004 from 4:00 p.m. at 7:00 p.m. Approximately 50 persons were tested, including some adults, but none had blood lead level greater than or equal to 10 µg/dl.

The Environmental Protection Agency (EPA) considers lead to be a probable human carcinogen. While worker studies have shown limited associations between elemental lead exposure and lung, stomach, kidney, and glioma (brain and spinal cord) cancers in humans, a dose-response relationship has not been established and a cancer slope factor has not been calculated. Therefore, we were unable to calculate a lifetime excess cancer risks these estimated lead exposures.

Polycyclic Aromatic Hydrocarbons (PAHs)

Like dioxins, PAHs are a family of chlorinated compounds formed when organic chemicals (garbage, coal, oil, gasoline, wood, tobacco, and charbroiled meat) are burned. They are also found in asphalt, crude oil, coal, coal tar pitch, creosote, and roofing tar. To evaluate toxicity, we relate the toxicities of the carcinogenic PAH family members to the toxicity of benzo(a)pyrene, and then add them together for the PAH toxicity equivalent (TEQ).

The location of PAHs in surface soil (Figure 3) suggests they might not be exclusively site related. PAHs in soil may be more related to the proximity of asphalt and vehicle emissions from roads. Only 3 surface soil PAH TEQs were greater than 10 times the ATSDR 1 in 1 million increased cancer risk screening value. One soil sample appeared to contain roofing tar or asphalt. While 25 of 45 surface soil samples contained PAHs above the screening value, only 7 of 35 **subsurface** samples contained elevated PAHs. These 7 are located near roads rather than near the site (Figure 5).

The theoretical increased cancer risk for residential children's and adults' exposures to subsurface soil with the highest PAH TEQ level is 2 in 100,000, or "no apparent" increased risk, all other measured levels are lower and are listed in Tables 8 and 9.

Exposures to Mixtures

Dioxins (Schantz et al. 1992), arsenic (Wasserman et al. 2004) and lead (Chiodo et al. 2004) have been linked with developmental decrements at or near the lowest levels of exposure having reported health effects. Although the off-site sampling has been limited:

- 1 surface and 3 subsurface soil samples contained dioxin sufficient to produce children's doses above the minimum risk level calculated from a study showing adverse developmental effects in monkeys,
- 1 subsurface soil sample contained arsenic at a level that causes developmental and cognitive effects in children drinking arsenic-contaminated water, and
- 2 surface and 6 subsurface soil samples contained lead at levels that could affect children's and adults' cognitive processes.

Daily, long-term exposures to more than one of these chemicals in soil could have additive effects. Complete characterization of off-site soil contamination will assure that the future measures taken adequately safeguard children from possible developmental decrements.

Cancers from Mixtures - Although the highest individual chemical levels were not measured in samples from the same locations, we added the cancer risks for residential yards and “other” properties. While the theoretical increased cancer risk is higher for subsurface soil, people are not likely to have daily exposure to it. The theoretical increased cancer risk from exposure to the mixture to chemicals found in residential yards is between “no apparent increase” (1 in 100,000) and “low increased risk” (1 in 10,000). Arsenic, dioxin, lead, and PAHs have been linked to lung cancer. Arsenic dioxin and PAHs have been linked to skin cancer. In additions, arsenic has been linked to liver, gastrointestinal, and urinary tract cancers (stomach, intestines, kidney, and bladder). PAHs have been linked to bladder and other cancers. Lead has been linked to kidney, stomach, brain and spinal cord cancers. Dioxin has been linked with sort-tissue sarcoma, non-Hodgkin’s lymphomas, liver and thyroid cancers.

Increased Theoretical Cancer Risk from Chemical Mixture

	Residential Yards	“Other” Properties
• Surface Soil	“no apparent increase” ^Y 3 in 100,000 ^Y	“low increased risk” 5 in 100,000
• Subsurface Soil	“low increased risk” ^Y 7 in 100,000 ^Y	“low increased risk” 5 in 100,000

Quality Assurance and Quality Control

The completeness and reliability of the referenced environmental data determine the validity of the analyses and conclusions drawn for this health consultation. Florida DOH used existing environmental data. We assume these data are valid: Florida DEP’s contractor and the laboratory they used have approved comprehensive quality assurance project plans.

Child Health Considerations

ATSDR and FDOH recognize the unique vulnerabilities of infants and children demand special attention (ATSDR 2005b). Children are at a greater risk than are adults for some hazardous substance exposures. Because children are smaller than adults are, their exposures can result in higher doses of chemical per body weight. If toxic exposures occur during critical growth stages, the developing body systems of children can sustain permanent damage. Probably most important, however, is that children depend on adults for risk identification and risk management, hygiene awareness, and access to medical care. Thus, adults should be aware of public health risks in their community so they can guide their children accordingly. In recognition of these concerns, ATSDR developed the chemical screening values for children’s exposures that FDOH used in preparing this report to address site-specific child health considerations.

Susceptible populations have different or enhanced responses to toxic chemicals than most people. Reasons include genetic makeup, age, health, nutritional status, and exposure to other toxic substances (like cigarette smoke or alcohol). These factors may limit a susceptible person’s ability to detoxify or excrete harmful chemicals or may increase the effects of damage to their organs or systems. For example, persons with the ALAD2 gene may be genetically susceptible to

^Y Total does not include the cancer risk from PAHs which may not be site related.

^Y We did not use the cancer risk for the highest PAH value measured at one residence, because it was 2 in 10,000 and the other highest PAH values gave an increased risk of 2 in 1 million. FDOH asked FDEP about this PAH anomaly and they thought there might have been asphalt in this sample.

meningioma. Persons with the ALAD2 gene tend to have higher lead concentrations in their blood, and lead has been shown to increase the risk of brain cancer, particularly meningioma (Ranjaraman et al. 2005).

Conclusions

1. Based on the limited available data, the public health hazard category for surface soil contaminants on residential properties is “No Apparent Public Health Hazard” although daily exposures lasting for periods longer than a year (chronic exposures) to the highest levels of lead measured in surface soil might adversely affect sensitive subpopulations with high blood pressure and anemia. Arsenic, lead, and dioxin levels measured in residential surface soil might adversely affect sensitive subpopulations such as children with developmental disabilities.

Some of the levels of contaminants in residential surface and subsurface soil exceed the Florida residential Soil Target Cleanup Levels. The theoretical increased cancer risk from long-term exposures to a mixture of the highest measured levels of contaminants in residential surface soil is “low” to “no apparent.”[†]

Arsenic and lead levels are generally higher in subsurface soils than in surface soils on residential properties, indicating incinerator ash may have been used as fill. Digging into subsurface soils on residential properties could increase residents’ exposure potentials especially if buried wastes are brought to the surface.

2. Some off-site soil on non-residential properties that has been tested would be a “public health hazard” if people had daily, long-term exposures to it.

Modeling indicates daily exposures to off-site subsurface soils with the highest lead levels could result in blood lead levels above the Centers of Disease Control’s action level (for intervention) of 10 µg/L. While the model assumptions we made (daily, long-term exposures, lasting 8 hours a day) are unlikely to be met for buried soils that are not currently on residential property, these levels indicate **a potential** for excess lead exposure. The community may not recognize this potential, and workers who may replace or repair utility lines or cables buried in road right-of-ways, or may carry out construction on vacant properties, similarly may not recognize their potential for exposure.

3. Based on the distribution and the measured levels, polycyclic aromatic hydrocarbons (PAHs) in Durrs neighborhood soil do not appear to be related to the Lincoln Park Complex site.

Recommendations

1. Collect additional surface and subsurface soil samples from the Durrs neighborhood north and east of the Lincoln Park Complex (Figure 6). Analyze for arsenic, lead, and dioxins to characterize the extent and levels of contamination.

[†] Between 1 in 100,000 and 1 in 10,000 risk of an increased cancer case over the expected numbers of cases of all types of cancers combined.

2. Take measures to prevent future contamination exposures once the levels and extent of contamination are known.
3. Until the full extent of soil contamination is determined, avoid contact with soil in the untested areas north and east of the Lincoln Park Complex that contains ash, glass, or metal pieces (Figure 6). Residents in this untested area that have ash, glass, or metal pieces in their soil should use the safe gardening practices outlined in Appendix C. Residents with ash, glass, or metal pieces in their soil should only grow edible fruits and vegetables using raised beds with clean soil or compost. DEP has advised the residents whose yards have been tested and are known to have elevated levels of contaminants to follow these same recommendations.

Public Health Action Plan

Florida DOH will continue to work with DEP to inform and educate Durrs neighborhood residents.

Authors, Technical Advisors

Florida Department of Health Author

Connie Garrett
Bureau of Community Environmental Health
Division of Environmental Health
(850) 245-4299

Florida Department of Health Designated Reviewer

Randy Merchant
Bureau of Community Environmental Health
Division of Environmental Health
(850) 245-4249

ATSDR Reviewer

Jennifer Freed
Technical Project Officer
Division of Health Assessment and Consultation

References

- Altmann L, Sveinsson K, Kraemer U, et al. 1998. Visual functions in 6-year-old children in relation to lead and mercury levels. *Neurotoxicology and Teratology* 20(1):9-17.
- Angle CR, McIntire MS. 1978. Low-level lead and inhibition of erythrocyte pyrimidine nucleotidase. *Environ Res* 17:296-302.
- Angle CR, McIntire MS, Swanson MS, et al. 1982. Erythrocyte nucleotides in children—increased blood lead and cytidine triphosphate. *Pediatr Res* 16:331-334.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 2006. Soil Comparison Values. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Atlanta, GA.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 2006 Update. Toxicological profile for lead. Update. Atlanta: US Department of Health and Human Services.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 2005a. Public health assessment the Lincoln Park complex. Atlanta: U.S. Department of Health and Human Services.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 2005b. Public health assessment guidance manual. Atlanta: U.S. Department of Health and Human Services.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 2005d. Health Consultation, Lincoln Park Complex, Blood Lead Testing. Atlanta: U.S. Department of Health and Human Services.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 2004. Interaction profile for persistent chemicals found in fish. Atlanta: U.S. Department of Health and Human Services. February 2004.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 2002. Case study in environmental medicine. Lead toxicity. Atlanta: US Department of Health and Human Services.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for arsenic. Update. Atlanta: U.S. Department of Health and Human Services. September 2000.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 1999. Toxicological profile for lead. Update. Atlanta: US Department of Health and Human Services.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 1998a. Toxicological profile for chlorinated dibenzo-p-dioxins. Update. Atlanta: U.S. Department of Health and Human Services.
- [ATSDR] Agency for Toxic Substances and Disease Registry. 1998b. Guidance on Including Child Health Issues in Division of Health Assessment and Consultation Documents: U.S. Department of Health and Human Services. July 2, 1998
- [ATSDR] Agency for Toxic Substances and Disease Registry. 1995. Toxicological profile for polycyclic aromatic hydrocarbons. Update. Atlanta: U.S. Department of Health and Human Services. Publication No.: PB/2000/108021

Baghurst PA, Robertson EF, McMichael AJ, et al. 1987. The Port Pirie cohort study: Lead effects on pregnancy outcome and early childhood development. *Neurotoxicology* 8:395-401.

Bhattacharya A, Smelser DT, Berger O, et al. 1998. The effect of succimer therapy in lead intoxication using postural balance as a measure: A case study in a nine-year-old child. *Neurotoxicology (Little Rock)* 19(1):57-64.

Bornschein RL, Grote J, Mitchell T, et al. 1989. Effects of prenatal lead exposure on infant size at birth. In: Smith M, Grant LD, Sors A, eds. *Lead exposure and child development: An international assessment*. Lancaster, UK: Kluwer Academic Publishers.

Bureau of the Census, 2000. *LandView 5 Software on DVD, A Viewer for EPA, Census and USGS Data and Maps*. U.S. Department of Commerce.

Canfield RL, Henderson CR, Cory-Slechta DA, et al. 2003. Intellectual impairment in children with blood lead concentrations below 10 micrograms per deciliter. *N Engl J Med* 348(16):1517-1526

Chia SE, Chia HP, Ong CN, Jeyaratnam J. 1996. Cumulative concentrations of blood lead and postural stability. *Occup Environ Med* 53(4):264-268.

Chiodo LM, Jacobson SW, Jacobson JL, 2004. Neurodevelopmental effects of postnatal lead exposure at very low levels. *Neurotoxicol Teratol* 26(3):359-371

Chiou H-Y, Huang W-I, Su C-L, et al. 1997. Dose-response relationship between prevalence of cerebrovascular disease and ingested inorganic arsenic. *Stroke* 28(9):1717-1723.

Den Hond E, Nawrot T, Staessen JA. 2002. The relationship between blood pressure and blood lead in NHANES III. *J Hum Hypertens* 16:563-568.

[DEP] Florida Department of Environmental Protection. 2005. *Soil, Groundwater, and Surface Water Cleanup Target Levels (CTLs) for Chapter 62-777, Florida Administrative Code*.

Ferreccio C, Gonzalez Psych C, Milosavjlevic Stat V, et al. 1998. Lung cancer and arsenic exposure in drinking water: a case-control study in northern Chile. *Cad Saude Publica* 14 (Suppl. 3):1993-198.

Gennert J-P, Buchet J-P, Roels H, et al. 1992. Fertility of male workers exposed to cadmium, lead or manganese. *Am J Epidemiol* 135:1208-1219.

Goodfellow F J L, V S G Murray, S K Ouki, A Iversn, A Sparks, T Bartlett. 2001. Public health response to an incident of secondary chemical contamination at a beach in the United Kingdom, *Occupational Environmental Medicine*, 2001; 58:232-238 (April)

Gupta P, Banerjee DK, Bhargava SK, et al. 1993. Prevalence of impaired lung function in rubber manufacturing factory workers exposed to benzo(a)pyrene and respirable particulate matter. *Indoor Environ* 2:26-31.

Hammond PB, Bornschein RL, Succop P. 1985. Dose-effect and dose-response relationships of blood lead to erythrocytic protoporphyrin in young children. In: Bornschein RL, Rabinowitz MB, Eds. *The Second International Conference on Prospective Studies of Lead*, Cincinnati, OH: April 1984. *Environ Res* 38:187-196.

- Haque R, Mazumdar DN, Samanta S, et al. 2003. Arsenic in drinking water and skin lesions: Dose-response data from West Bengal, India. *Epidemiology* 14(2):174-182.
- Hernberg S, Nikkanen J. 1970. Enzyme inhibition by lead under normal urban conditions. *Lancet* 1:63.
- Hicks, Caldas LQA, Dare PRM, et al. 1986. Cardiotoxic and bronchoconstrictor effects of industrial metal fumes containing barium. *Archives of Toxicology, Suppl* 9. Toxic interfaces of neurons, smoke and genes, Secaucus, NJ: Springer-Verlag New York, Inc.
- Hu H. 1991. Knowledge of diagnosis and reproductive history among survivors of childhood plumbism. *Am J Public Health* 81:1070-1072.
- Ihrig MM, Shalat SL, Baynes C. 1998. A hospital-based case-control study of stillbirths and environmental exposure to arsenic using an atmospheric dispersion model linked to a geographical information system *Epidemiology* 9(3):290-294.
- Lutz, PM, Wilson TJ, Ireland J, et al. 1999. Elevated immunoglobulin E (IgE) levels in children with exposure to environmental lead. *Toxicology* 134:63-78.
- Lianfang W, Jianzhong H. 1994. Chronic arsenism from drinking water in some areas of Xinjiang, China. In: Nraigu JO ed. *Arsenic in the environment: Part II Human health and ecosystem effects*. New York, NY: John Wiley and Sons, Inc., 159-172.
- Mahaffey KR, Rosen JF, Chesney RW, et al. 1982. Association between age, blood lead concentration, and serum 1,25-dihydroxycholecalciferol levels in children. *Am J Clin Nutr* 35:1327-1331.
- McMichael AJ, Vimpani GV, Robertson EF, et al. 1986. The Port Pirie cohort study: Maternal blood lead and pregnancy outcome. *J Epidemiol Community* 40:18-25.
- Moore MR, Goldberg A, Pocock SJ, et al. 1982. Some studies of maternal and infant lead exposure in Glasgow. *Scott Med J* 27:113-122.
- Muntner P, He J, Vupputuri S, et al. 1983. Early appearance and localization of intranuclear inclusion in the segments of renal proximal tubules of rats following ingestion of lead. *Br J Exp Pathol* 64:144-155.
- Neal J, Rigdon RH. 1967. Gastric tumors in mice fed benzo[a]pyrene: A quantitative study. *Tex Rep Biol Med* 25:553-557.
- NTP 1994, Toxicology and carcinogenesis study of barium chloride dehydrate-(CAS no. 10326-27-0) in F344/N rats and B6C3F₁ mice, National Toxicology Program TR432
- Nordstrom S, Beckman L, Nordensen I. 1979. Occupational and environmental risks in and around a smelter in northern Sweden: V. Spontaneous abortion among female employees and decreased birth weight in their offspring. *Hereditas* 90:291-296.
- Perry HM Jr, Kopp SJ, Erlanger MW, et al. 1983. Cardiovascular effects of chronic barium ingestion. *Trace Subst Environ Health* 17:155-164.
- Perry HM Jr, Perry EF, Erlanger MW, et al. 1985. Barium-induced hypertension. *Adv Mod Environ Toxicol, Inorg Drinking Water Cardio Vasc Dis* 9:221-229
-

Piomelli S, Seaman C, Zullo D, et al. 1982. Threshold for lead damage to heme synthesis in urban children. *Proc Natl Acad Sci*. 7:3335-3339.

Rabinowitz MB, Levilon A, Needleman H. 1986. Occurrence of elevated protoporphyrin levels in relation to lead burden in infants. *Environ Res* 39:253-257.

Rajaraman P, Schwartz BS, Rothman N, Yaeger M, Fine HA, Shapiro WR, Seiker RG, Black PM, and Inskip PD. 2005. δ -Aminolevulinic Acid Dehydratase Polymorphism and Risk of Brain Tumors in Adults. *Environmental Health Perspectives*. Sept 2005.

Rier SE, Martin DC, Bowman RE, et al. 1993. Endometriosis in Rhesus monkeys (*Macaca mulatta*) following chronic exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Fund Appl Toxicol* 21:433-441.

Roels HA, Buchet J-P, Lauwerys R, et al. 1976. Impact of air pollution by lead on the heme biosynthetic pathway in school-age children. *Arch Environ Health* 31:310-316.

Roels HA, Lauwerys R. 1987. Evaluation of dose-effect and dose-response relationships for lead exposure in different Belgian population groups (fetus, child, adult men and women). *Trace Elements in Medicine* 4:80-87.

Rosen JF, Chesney RW, Hamstra AJ, et al. 1980. Reduction in 1,25-dihydroxyvitamin D in children with increased lead absorption. *N Engl J Med* 302:1128-1131.

Sanin LH, Gonzalez-Cossio T, Romieu I, et al. 2001. Effect of maternal lead burden on infant weight gain at one month of age among breastfed infants. *Neurotoxicol Teratol* 23:203-212.

Schantz SL, Ferguson SA, Bowman RE. 1992. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin on behavior of monkey in peer groups. *Neurotoxicol Teratol* 14:433-446.

Torres-Sanchez LE, Berkowitz G, Lopez-Carrillo L, et al. 1999. Intrauterine lead exposure and preterm birth. *Environ Res* 81:297-301.

Tseng WP, Chu HM, How SW, et al. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan, *J Nat'l Cancer Inst* 40:453-463

Van Gennip AH, 1999. Defects in metabolism of purines and pyrimidines *Ned Tijdschr Klin Chem* 1999; 24: 171-175

Ward NI, Watson R, Bruce-Smith D. 1987. Placental element levels in relation to fetal development for obstetrically normal births: A study of 37 elements: Evidence for the effects of cadmium, lead, and zinc on fetal growth and for smoking as a source of cadmium. *Int J Biosoc Res* 9:63-81.

Wasserman, GA, X Liu, F Parves, H Ahsan, P Factor-Litvak, A van Geen, V Slavkovich, NJ LoIacono, Z Cheng, I Hussain, H Momataj, and JH Graziano. September 2004. Water Arsenic Exposure and Children's Intellectual Function in Araihasar, Bangladesh. *Environmental Health Perspectives* 112(13)1329-1333.

Wibberly DG, Khera AK, Edwards JH, et al. 1977. Lead levels in human placentae from normal and malformed births. *J Med Genet* 14:339-345.

Winneke G, Altmann L, Kramer U, et al. 1994. Neurobehavioral and neurophysiological observations in six-year-old children with low lead levels in East and West Germany. *Neurotoxicology* 15(3):705-713.

Wones RG, Stadler BL, Frohman LA. 1990. Lack of effect of drinking water barium on cardiovascular risk factors. *Environ Health Perspect* 85:355-359.

Appendix A—Figures

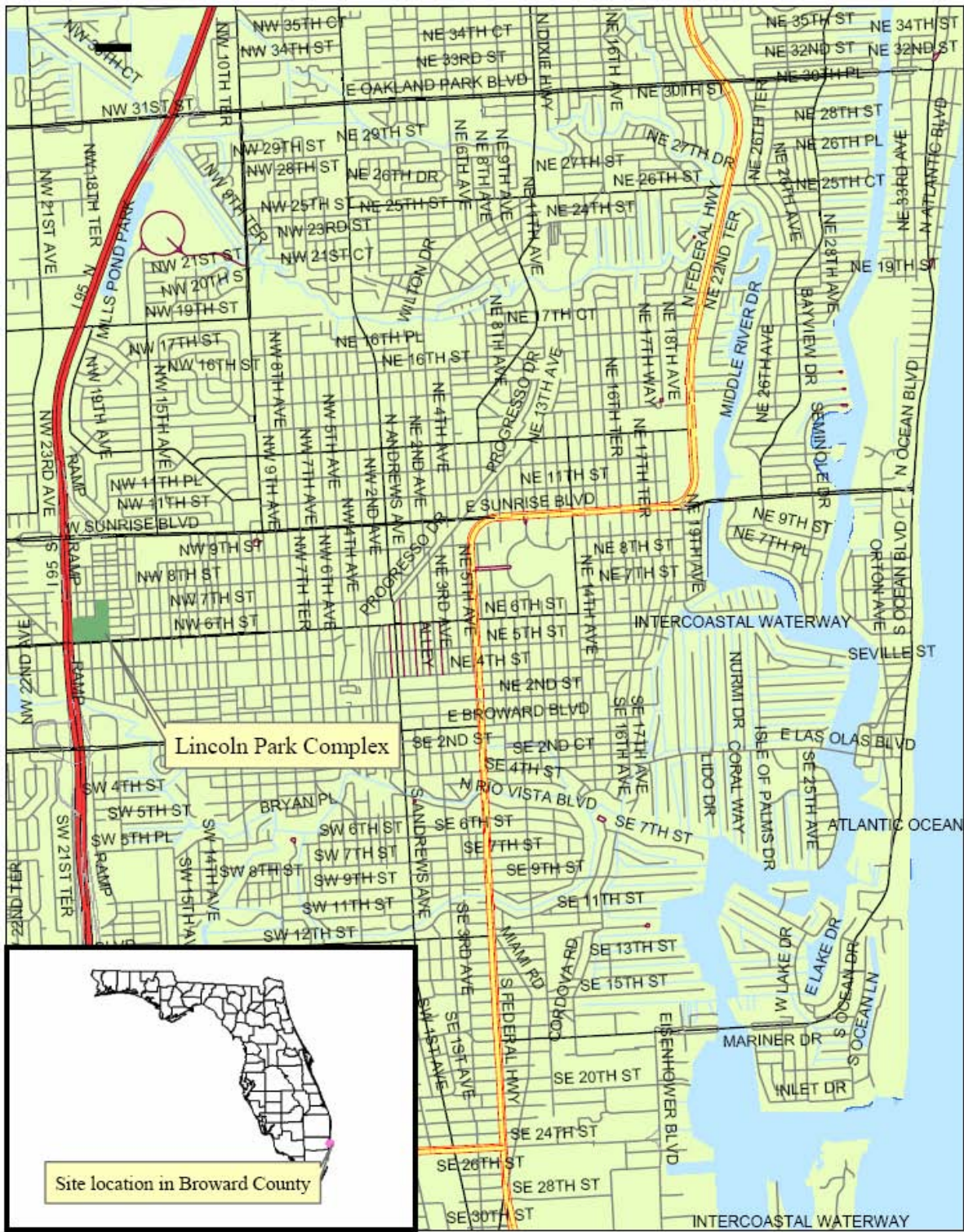


Figure 1: Location of the site in Ft. Lauderdale, Broward County

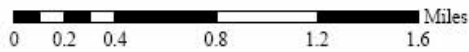




Figure 2: Levels of arsenic and dioxin in Durrs neighborhood surface soil (2004 and 2006) that could theoretically increase the risk of cancer related to arsenic or dioxin for persons having daily, long-term exposures by 1 extra case in 1 million persons, or more. ppm = parts per million ppt = parts per trillion









Figure 5: Levels of polycyclic aromatic hydrocarbons (PAHs) in Durrs neighborhood subsurface soil (2004 and 2006) that could theoretically increase the risk of cancer related to PAHs for persons having daily, long-term exposures by 1 extra case in 1 million persons, or more. ppm = parts per million





Figure 6: Areas of potential incinerator ash deposition that require more soil testing.



Appendix B—Tables

Table 2a. Completed exposure pathways

Pathway Name	Exposure Pathway Elements					Time
	Source	Environmental/ Exposure Media	Point of Exposure	Route of Exposure	Exposed Population and land use	
Contaminated off-site surface soil, dust	Residential soil and soil on other properties	Wastes, surface and subsurface soil	Off-site properties	Incidental ingestion and inhalation	Off-site residents/owners, workers	Past Current Future

Table 2b. Potential exposure pathways

Pathway Name	Exposure Pathway Elements					Time
	Source	Environmental/ Exposure Media	Point of Exposure	Route of Exposure	Exposed Population and land use	
Contaminated off-site subsurface soil, dust	Residential soil and soil on other properties	Wastes, surface and subsurface soil	Off-site properties	Incidental ingestion and inhalation	Off-site residents/owners, workers	Past Current Future

Table 2c. Incomplete exposure pathways

Pathway Name	Exposure Pathway Elements					Time
	Source	Environmental/ Exposure Media	Point of Exposure	Route of Exposure	Exposed Population and land use	
Shallow groundwater	Contaminated groundwater on the site	Shallow groundwater	Off-site irrigation well	Incidental ingestion and inhalation	Down-gradient residents	Future

Table 3. Maximum concentrations in off-site surface soil (0 to 3 inches below ground surface).

Contaminants of Concern	Screening Value (mg/kg)		Highest Soil Concentration (mg/kg)		Location of Highest Concentration		Number Soil Samples Above Screening Value	
	ATSDR: Children/adults	DEP:	residences	Other*	residences	Other*	residences	Other*
arsenic	20/200 EMEG	2.1 SCTL	6.3	30	Front yard of 2 nd res. N of former Inc. RSLPRSS-16	N boundary of former Inc. site LPRSS-9	ATSDR DEP 0/43, 12/43	ATSDR DEP 1/12, 6/12
barium	30,000 400,000 EMEG	210 SCTL	230	BSL/120	Back yard, lot N. of former inc. LPRSS-8	Right-of-way E. of former Incin. LPRSS-3	ATSDR DEP 0/43, 1/43	ATSDR DEP 0/12, 0/12
dioxin TEQ	0.00005/0.0007 EMEG	0.000007 SCTL	0.00003	0.0002	Side yard of 2 nd res. N of former Inc. RSLPRSS-19	Vacant lot N. of former Incin. LPRSS-10	ATSDR DEP 0/35, 6/35	ATSDR DEP 1/10, 5/10
lead		400 SCTL	BSL/290	BSL/330	Side yard of 2 nd res. N of former Inc. RSLPRSS-19	Right-of-way E. of former Incin. LPRSS-3	0/43	0/12
PAHs TEQ	0.1 CREG	0.1 residential SCTL	51/3	0.447	Front yard of 2 nd res. N of former LP school RS2LPSS-8	Vacant lot N. of former Incin. RS2LPSS-21	21/43	4/12

CREG—ATSDR’s Cancer Risk Evaluation Guide for 1 excess cancer case in 1 million people (ATSDR 1992a).

EMEG—Environmental Media Evaluation Guide

Inc. or Incin.—Incinerator Property

mg/kg—milligrams per kilogram

N—north

PAHs—polycyclic aromatic hydrocarbons

SCTL—FDEP’s Soil Target Cleanup Level for residential land uses.

* Other—sites include vacant lots, lots owned by the city, and road right-of-ways

Table 4. Maximum concentrations in off-site subsurface soil (3 to 24 inches below ground surface).

Contaminants of Concern	Screening Value (mg/kg)		Highest Soil Concentration (mg/kg)		Location of Highest Concentration		Number Soil Samples Above Screening Value			
	ATSDR: Children/adults	DEP:	residences	Other*	residences	Other*	residences	Other*	residences	Other*
arsenic	20/200 EMEG	2.1 SCTL	53	40	Side yard of 2 nd res. N of former Inc. RSLPRSS-19	Right-of-way E. of former Incin. LPRSSB-3	ATSDR 1/23	DEP 8/23	ATSDR 0/12	DEP 7/12
barium	30,000 400,000 EMEG	120 SCTL	520	2300	Side yard of 2 nd res. N of former Inc. RSLPRSS-19	Right-of-way E. of former Incin. LPRSSB-3	ATSDR 0/23	DEP 3/23	ATSDR 0/12	DEP 2/12
dioxin TEQ	0.00005/0.0007 EMEG	0.000007 SCTL	0.00008	0.0001	RS2LPSB-13	Vacant lot N. of former Incin. LPRSS-10	ATSDR 2/14	DEP 6/14	ATSDR 2/7	DEP 4/7
lead		400 SCTL	870	4,500	Side yard of 2 nd res. N of former Inc. RSLPRSS-19	Right-of-way E. of former Incin. LPRSSB-3	DEP	3/23		DEP 3/12
PAHs TEQ	0.1 CREG	0.1 residential SCTL	18.3/1.3	0.143	Front yard of 2 nd res. N of former LP school RS2LPSB-8	Vacant lot N. of former Incin. RS2LPSS-21		7/23		1/12

CREG—ATSDR’s Cancer Risk Evaluation Guide for 1 excess cancer case in 1 million people (ATSDR 1992a).

EMEG—Environmental Media Evaluation Guide

Inc. or Incin.—Incinerator Property

mg/kg—milligrams per kilogram

N—north

PAHs—polycyclic aromatic hydrocarbons

SCTL—FDEP’s Soil Target Cleanup Level for residential land uses.

* Other—sites include vacant lots, lots owned by the city, and road right-of-ways

Table 5a. Estimated doses from exposures to residential surface soil.

Contaminant of Concern (maximum concentration: mg/kg)	Oral MRL (mg/kg/day)	Soil/dust-Ingestion (mg/kg/day)		Inhalation MRL (mg/m ³)	Soil/dust- Inhalation (mg/m ³)
		Child	Adult		Child and Adult
arsenic (6.3)	0.0003 Chr	0.00008	0.000009	-	0.0000003
barium (230 mg/kg)	0.6 Chr	0.003	0.0003	-	0.00001
dioxin TEQ (0.00003)	0.000000001 Chr	0.0000000004	0.00000000004	-	0.000000000002
lead (290)	-	M	M	-	M
PAHs (51)/(3)	-	0.0007/0.00004	0.00007/0.0000002	-	0.000003/0.0000002

Table 5b. Estimated doses from exposures to right-of-way, city-owned or vacant lot surface soil.

Contaminant of Concern (maximum concentration mg/kg)	Oral MRL (mg/kg/day)	Soil/dust-Ingestion (mg/kg/day)		Inhalation MRL (mg/m ³)	Soil/dust- Inhalation (mg/m ³)
		Child	Adult		Child and Adult
arsenic (30)	0.0003 Chr	0.0004	0.00004	-	0.000002
barium (120 mg/kg)	0.6 Chr	0.002	0.0002	-	0.000007
dioxin TEQ (0.0002)	0.000000001 Chr	0.000000003	0.0000000003	-	0.00000000001
lead (330)	-	M	M	-	M
PAHs (0.447)	-	0.000006	0.0000006	-	0.00000002

Table 6a. Estimated doses from exposures to residential subsurface soil.

Contaminant of Concern (maximum concentration mg/kg)	Oral MRL (mg/kg/day)	Soil/dust-Ingestion (mg/kg/day)		Inhalation MRL (mg/m ³)	Soil/dust- Inhalation (mg/m ³)
		Child	Adult		Child and Adult
arsenic (53)	0.0003 Chr	0.0007	0.00008	-	0.000003
barium (520 mg/kg)	0.6 Chr	0.007	0.0007	-	0.00003
dioxin TEQ (0.00008)	0.000000001 Chr	0.000000001	0.0000000001	-	0.000000000004
lead (870)	-	M	M	-	M
PAHs (18.3)(1.3)	-	0.0002/	0.00003/	-	0.000001/0.00000007

Table 6b. Estimated doses from exposures to right-of-way, city-owned or vacant lot subsurface soil.

Contaminant of Concern (maximum concentration mg/kg)	Oral MRL (mg/kg/day)	Soil/dust-Ingestion (mg/kg/day)		Inhalation MRL (mg/m ³)	Soil/dust- Inhalation (mg/m ³)
		Child	Adult		Child and Adult
arsenic (40)	0.0003 Chr	0.0005	0.00006	-	0.000002
barium (2300 mg/kg)	0.6 Chr	0.03	0.003	-	0.0001
dioxin TEQ (0.0001)	0.000000001 Chr	0.000000001	0.0000000001	-	0.000000000006
lead (4,500)	-	M	M	-	M
PAHs (0.143)	-	0.000002	0.0000002	-	0.000000008

Explanations for abbreviations and footnotes used on Tables 5 and 6.

Acute – Acute exposure length of 0-14 days

Int – Intermediate exposure length of 15- 364 days

Chr – Chronic exposure length of more than 365 days

N.S. – Not Significant

mg/kg/day – milligram chemical per kilogram body weight per day

mg/m³ – microgram of chemical per cubic meter of air

M – values were modeled (tables precede these explanations)

MRL – Minimum Risk Level extrapolation of a No Observable Adverse Effect level in a study to exposures, calculated by dividing the study dose by safety factors.

Table 7. Estimated Blood Lead Concentrations In Children and Adults Ingesting Off-Site

(0 to 3” foot) Surface Soil and Subsurface Soil (3’-24”) (micrograms per deciliter - µg/dl)

Media	Children	Adults
Off-site residential surface soil	1.5-3.3	0.8-2.9
Off-site other surface soil	1.5-3.6	0.9-3.2
Off-site residential subsurface soil	2.6-7.2	2.0-6.8
Off-site other subsurface soil	9.8-31.1	9.2-30.7

Values used to Estimated Blood Lead Concentrations in Persons Ingesting On-Site

(0 to 2 foot) Soil (micrograms per deciliter - µg/dl)

Time 8 hrs a day for both	Values for children Slopes		Values for adults Slopes	
	Low		Low	High
0.33	2.46	0.33	1.59	3.56
0.33	2.46	0.33	1.53	3.56
0.33	0.24	0.33	0.016	0.0195
0.33	0.16	0.33	0.03	0.06
0.33	0.002	0.33	0.002	0.016
0.33	0.004	0.33	0.004	0.004

*Default Value from ATSDR 1999a, Appendix D.

These slopes were for children and adults from ATSDR 1999a, Appendix D.

ATSDR’s Regression Analysis with Multiple-uptake Parameters to Estimate Blood Lead from Environmental Exposures (ATSDR 1999a, Appendix D)

Table 8. Comparison of doses calculated from highest measured surface soil values to lowest observable adverse effect levels (LOAELs) in animal and human medical studies. Bolded doses are above LOAEL or minimum risk level (MRL).

Chemical	Soil Doses are in mg/kg/day, inh. are in mg/m ³		theoretical increased cancer risk	
	children's dose	adults' dose	children's	adults'
Arsenic (residences)	Ing 0.00008 Inh 0.000003	Ing 0.000009 Inh 0.0000003	Ing 5:1,000,000 Inh <1:1,000,000	Ing 6:1,000,000 Inh <1:1,000,000
Arsenic (other [†])	Ing 0.0004 Inh 0.000002	Ing 0.00004 Inh 0.000002	Ing 3:100,000 Inh 1:1,000,000	Ing 3:100,000 Inh 2:1,000,000
ATSDR 2000 (Update)	<p><u>Child (residential) ingestion dose</u> (0.00008) is 1/25 times the lowest observable adverse effect (LOAEL) dose (0.002, Chiou et al. 1997) associated with cerebrovascular disease and cerebral infarction (an interruption of the blood supply to any part of the brain, resulting in damaged brain tissue). Another study showed skin effects at 0.0018 (Haque et al., 2003). This child residential ingestion dose is 3.75 less than the MRL (0.0003) calculated from a no observable adverse effect level NOAEL (0.0008, Tseng et al.1968) for skin effects from long-term ingestion of arsenic in drinking water. ATSDR scientists divided this NOAEL dose (0.0008) by 3 to account for human diversity in calculating the MRL.</p> <p><u>Adult (residential) ingestion dose</u> is 8.8 times less than the (0.00008) dose referenced for children and 1/222nd the LOAEL dose, we would not expect skin or gastrointestinal health effects for most adults and children, at these exposure levels.</p> <p><u>Inhalation (residential) exposure level</u> (0.0000003) is 2,333 times less than the amount associated with increased risk of still birth in humans (0.0007, Ihrig et al., 1998, As 3⁺) and 23,333 times less than the dose causing dermatitis (0.007, Mohamed 1998, As 3⁺) in humans inhaling arsenic. Dermatitis is skin inflammation that may cause redness, pain, and occasionally itching.</p> <p><u>Child (other) ingestion dose (0.0004)</u> is 1/5th the LOAEL dose (0.002, Chiou et al. 1997) associated with cerebrovascular disease and cerebral infarction (an interruption of the blood supply to any part of the brain, resulting in damaged brain tissue). This level is 1/3 more than the MRL (0.0003) calculated from the NOAEL (0.0008, Tseng et al.1968) for adverse skin effects from long-term ingestion of arsenic in drinking water. ATSDR scientists divided this NOAEL dose (0.0008) by 3 to account for human diversity in calculating the MRL (0.0003). Sensitive children or children ingesting more than the level of soil we estimated might experience adverse skin effects.</p> <p><u>Adult (other) ingestion dose</u>, 0.00004, is 10 times less than the children's (other) dose (0.0004), we would not expect skin or gastrointestinal health effects for adults at this level of exposure.</p> <p><u>Inhalation (other) exposure level</u> (0.000002) is 350 times less than the exposure level associated with increased risk of still birth in humans (0.0007, Ihrig et al., 1998, As 3⁺) and 3,500 times less than the dose causing dermatitis (0.007, Mohamed 1998, As 3⁺) in humans inhaling arsenic. Dermatitis is skin inflammation that may cause redness, pain, and occasionally itching.</p> <p><u>Arsenic Associated cancers</u>: From lowest to highest dose cancer effect levels, chronic arsenic exposures in people have been linked to lung cancer, basal and squamous cell skin cancers, liver cancer (haemangioendothelioma), urinary tract cancers (bladder, kidney, ureter, and all urethral cancers), and intraepidermal cancers. Intraepidermal is the name for the early pre-invasive form of squamous cell skin cancer. Pre-invasive means that the cancer cells are confined to the outermost layer of skin, the epidermis. At this stage, the cancer cells are unlikely to have spread to the lymph nodes, but they can spread along the skin surface. If left untreated, these cells can develop into an invasive cancer and spread into the lymphatic system.</p>			

[†]Other properties include right-of-ways, city-owned or vacant lot surface soil.



Chemical	Soil Doses are in mg/kg/day, inh. are in mg/m ³		theoretical increased cancer risk	
	children's dose	adults' dose	children's	adults'
Barium (residences)	Ing 0.003 Inh 0.00001	Ing 0.0003 Inh 0.00001	No Slope No Unit Risk	No Slope No Unit Risk
Barium (other*)	Ing 0.002 Inh 0.000007	Ing 0.0002 Inh 0.000007	No Slope No Unit Risk	No Slope No Unit Risk
ATSDR 2006 (Update) Draft * Other properties include right-of-ways, city-owned or vacant lot surface soil.	<p><u>Child (residential) ingestion dose</u> (0.003) is 200 times less than the chronic MRL (0.6 mg/kg/day). Rats dosed (115 mg/kg/day, NTP 1994) with barium chloride in their food for 90 days showed increased kidney weight. The NOAEL for this study was 65 mg/kg/day. ATSDR authors used this NOAEL to derive the chronic MRL of 0.6 for barium exposure by dividing the NOAEL by 100 (10 to account for extrapolation from animals to humans and 10 for human variability).</p> <p><u>Adult (residential) ingestion dose</u> (0.0003) is 10 times less than the (0.003) dose referenced for children, we would not expect kidney or other health effects for most adults and children, at these exposure levels.</p> <p><u>Inhalation (other) exposure level</u> Medical case reports and animal studies are inadequate to establish the health effects of barium by inhalation. The lowest reported exposure level is 0.06 mg/m³/minute (Hicks et al. 1986). Guinea pigs exposed for an unspecified amount of time to this concentration of aerosolized barium chloride solution experienced bronchoconstriction. The estimated exposure level, (0.00001) is 6,000 times less.</p> <p><u>Child (other) ingestion dose</u> (0.002) is 300 times less than the chronic MRL (0.6 mg/kg/day). Rats dosed (115 mg/kg/day, NTP 1994) with barium chloride in their food for 90 days showed increased kidney weight. The NOAEL for this study was 65 mg/kg/day. ATSDR authors used this NOAEL to derive the chronic MRL of 0.6 for barium exposure by dividing the NOAEL by 100 (10 to account for extrapolation from animals to humans and 10 for human variability).</p> <p><u>Adult (other) ingestion dose</u> (0.0002) is 10 times less than the (0.003) dose referenced for children, we would not expect kidney or other health effects for most adults and children, at these exposure levels.</p> <p><u>Inhalation (other) exposure level</u> Medical case reports and animal studies are inadequate to establish the health effects of barium by inhalation. The lowest reported exposure level is 0.06 mg/m³/minute (Hicks et al. 1986). Guinea pigs exposed for an unspecified amount of time to this concentration of aerosolized barium chloride solution experienced bronchoconstriction. The estimated exposure level, (0.000007) is 8,571 times less.</p> <p><u>Barium associated cancers.</u> Barium has not been shown to cause cancer in people or animals.</p>			



Chemical	Soil Doses are in mg/kg/day, inh. are in mg/m ³		theoretical increased cancer risk	
	children's dose	adults' dose	children's	adults'
Dioxin TEQ (residences)	Ing 0.0000000004 Inh 0.000000000002	Ing 0.00000000004 Inh 0.000000000002	Ing 3:1,000,000	Ing 3:1,000,000
Dioxin TEQ (other [*])	Ing 0.000000003 Inh 0.00000000001	Ing 0.0000000003 Inh 0.00000000001	Ing 2:100,000	Ing 2:100,000
ATSDR 1998a (Update)	<p><u>Child res. ingestion dose</u> (0.0000000004) is 1/300th the dose (0.00000012, Rier et al., 1993) associated with reproductive effects (moderate endometriosis) and altered social behavior in a dioxin rhesus monkey study. This dose is also 2.5 time less than the MRL of 0.000000001 calculated from the 0.00000012 dose (Schantz et al., 1992) at which rhesus monkeys showed adverse neurobehavioral and developmental effects. ATSDR authors divided this dose by an uncertainty factor of 90, 3 for use of a minimal LOAEL, 3 for extrapolation from animals to humans and 10 for human variability. The results of other oral animal studies suggest that the effects that occur at the lowest levels of dioxin doses are immune and endocrine, in addition to developmental effects. People's ingestion exposures are mainly known from low levels of food contamination.</p> <p><u>Adult res. ingestion dose</u> (0.0000000004) is 10 times less than the residential children's dose. We would not expect health effects for most adults and children at these residential exposure levels.</p> <p><u>Child other ingestion dose</u> (0.0000000003) is 1/40th the dose (0.00000012) associated with reproductive effects (moderate endometriosis) and altered social behavior in a dioxin rhesus monkey study. However, this dose is 3 times greater than the MRL. While this level might indicate an increased risk for children exposed daily, such exposures to the "other" properties might not occur daily.</p> <p><u>Adult other ingestion dose</u> (0.0000000003) is 400 times less than the (0.00000012) LOAEL health effects described above for children, it is unlikely adults would experience health effects from exposures at these levels.</p> <p><u>Inhalation</u> of dioxins has not been studied in animals. People's occupational and accidental exposures to dioxin involve primarily inhalation and dermal exposure, but health effects are known primarily from associations with the levels stored in fat. The lowest levels of exposure are associated with hormone changes that can result in changes in sex ratios in children (more females are born). Higher levels are associated with immunosuppression, changes in the liver, abnormal glucose tolerance, and increased risk of diabetes. The highest exposure levels are associated with nervous system effects, chloracne, respiratory effects, and increased risk of cancer.</p> <p><u>Cancers</u> Statistically significant increases in risks for all cancers were found in workers highly exposed to dioxins with longer latency periods. Although the estimated Standardized Mortality Ratios (a ratio that is a direct comparison with a standard) are low, they are consistent across studies with the highest dioxin exposures. The evidence linking peoples' doses with site-specific cancers is weaker, with some data suggesting a possible relationship between soft-tissue sarcoma, non-Hodgkin's lymphoma, or respiratory cancers including lung cancer. Animal studies have also shown associations with liver, thyroid and skin cancer.</p>			

* Other properties include right-of-ways, city-owned or vacant lot surface soil.



Chemical	Soil Doses are in mg/kg/day, inh. are in mg/m ³		theoretical increased cancer risk	
	children's dose	adults' dose	children's	adults'
PAHs TEQ (residences)	Ing 0.0007/0.00004 Inh 0.000003/0.0000002	Ing 0.00007/0.0000002 Inh 0.000003/0.0000002	Ing 2: 10,000/ 1:100,000 Inh 4: 10,000/ 9: 1,000,000	Ing 2: 10,000/ 1:100,000 Inh 8:100,000/ 1: 100,000
PAHs TEQ (other [†])	Ing 0.000006 Inh 0.00000002	Ing 0.0000006 Inh 0.00000002	Ing 2:1,000,000 Inh <1:1,000,000	Ing 2:1,000,000 Inh <1:1,000,000
ATSDR 1995 (Update)	<p><u>Child res. ingestion dose</u> (0.0007) is 3,714 times less than the dose (2.6, Neal and Rigdon, 1967) associated with stomach cancer in mice exposed to benzo[a]pyrene ad lib in food for 30 to 197 days (non-cancer illnesses are all associated with much higher doses).</p> <p><u>Adult res. ingestion dose</u> (0.000007) is 37,142 times less than the (2.6) dose associated with stomach cancer in mice.</p> <p><u>Inhalation res. exposure level</u> (0.00000002) is 5,000 times less than the dose (0.0001, Gupta et al. 1993) associated with reduced lung function, abnormal chest x-ray, cough, bloody vomit, and throat and chest irritation, in persons exposed from 6 months to 6 years.</p> <p><u>Child other ingestion dose</u> (0.000006) is 433,333 times less than the dose (2.6) dose associated with stomach cancer in mice.</p> <p><u>Adult other ingestion dose</u> (0.0000006) is 4,333,333 times less than the (2.6) dose associated with stomach cancer in mice.</p> <p><u>Inhalation other exposure level</u> (0.00000002) is 5,000 times less than the dose (0.0001, Gupta et al. 1993) associated with reduced lung function, abnormal chest x-ray, cough, bloody vomit, and throat and chest irritation, in persons exposed from 6 months to 6 years.</p> <p><u>Cancer and occupational studies</u> Worker exposures to high levels of PAHs show cancers (skin, bladder, lung and gastrointestinal) are the most significant endpoints of PAH toxicity. Long-term worker PAH exposures have been linked with skin and eye irritation, photosensitivity, respiratory irritation (with cough and bronchitis), leukoplakia^Ω, precancerous skin growths enhanced by exposure to sunlight, erythema^Δ, skin burns, acneiform lesions, mild hepatotoxicity, and haematuria[€]. Also several PAH compounds are immunotoxic, and some suppress selective compounds of the immune system. Workers' dermal exposure studies indicate that although direct contact may be of concern at high exposure levels, they do not suggest that lower levels are likely to cause significant irritation (Goodfellow et al. 2001).</p>			

[†]Other properties include right-of-ways, city-owned or vacant lot surface soil.

Δ Erythema nodosum is an inflammation of subcutaneous fat tissue.

€ Hematuria is passage of blood in the urine.

Ω Leukoplakia is a common, potentially pre-cancerous disease of the mouth that involves the formation of white spots on the mucous membranes of the tongue and inside of the mouth. Despite the increased risk associated with having leukoplakia, many people with this condition never get oral cancer.

Table 9. Comparison of doses calculated from highest measured subsurface soil values to lowest observable adverse effect levels (LOAELs) in animal and human medical studies. Bolded doses are above LOAEL or minimum risk level (MRL).

Chemical	Soil Doses are in mg/kg/day		theoretical increased cancer risk	
	children's dose	adults' dose	children's	adults'
Arsenic (residences)	Ing 0.0007 Inh 0.000003	Ing 0.00008 Inh 0.000003	Ing 5:100,000 Inh 2:1,000,000	Ing 5:100,000 Inh 3:1,000,000
Arsenic (other [†])	Ing 0.0005 Inh 0.000002	Ing 0.00006 Inh 0.000002	Ing 3:100,000 Inh 2:1,000,000	Ing 4:100,000 Inh 3:1,000,000
ATSDR 2000 (Update)	<p><u>Child res. ingestion dose</u> (0.0007) is about 1/3rd the LOAEL dose (0.002, Chiou et al. 1997) associated with cerebrovascular disease and cerebral infarctions. Another study showed skin effects at 0.0018 (Haque, et al., 2003). This child residential ingestion dose is also 2.3 times greater than the MRL (0.0003) calculated from the NOAEL (0.0008) for adverse skin effects seen in another long-term study of ingestion of arsenic in drinking water. ATSDR scientists divided this NOAEL dose (0.0008, Tseng et al. 1968) by 3 to account for human diversity in calculating the MRL (0.0003). While sensitive children might experience cerebrovascular or skin effects if they had daily, long-term exposures to this arsenic-contaminated soil, such exposures might not occur daily since this soil is in the subsurface.</p> <p><u>Adult res. ingestion dose</u> is 8.75 times less than the (0.0007) dose referenced for children and 3.75 times less than the MRL. Most adults would be unlikely to experience skin or gastrointestinal health effects.</p> <p><u>Inhalation res. exposure level</u> (0.000003) is 1/233rd the amount associated with increased risk of stillbirth in humans (0.0007, Ihrig et al., 1998, As 3⁺) and 2,333 times less than the dose causing dermatitis (0.007, Mohamed 1998, As 3⁺) in humans inhaling arsenic. Dermatitis is skin inflammation that may cause redness, pain, and occasionally itching. Because the soil containing this level of arsenic is not directly at the surface, persons are less likely to have daily exposure to it; therefore, the risk of adverse health effect could be even less likely than these factors indicate.</p> <p><u>Child other ingestion dose</u> (0.0005) is 1/4th the LOAEL dose (0.002) associated with cerebrovascular disease and cerebral infarctions and skin effects at 0.0018. This dose is 1.5 times the MRL (0.0003). As with the child residential dose, while sensitive children might experience gastrointestinal or skin effects if they had daily, long-term exposures to this arsenic-contaminated soil, such exposures may not occur daily to this subsurface soil.</p> <p><u>Adult other ingestion dose</u> is 8.75 times less than the children's other dose (0.0005) dose. We would not expect skin or gastrointestinal health effects for adults at this level of exposure.</p> <p><u>Inhalation other exposure level</u> (0.000002) is 350 times less than the amount associated with increased risk of still birth in humans (0.0007) and 3,500 times less than the dose causing dermatitis (0.007) in humans inhaling arsenic. Dermatitis is skin inflammation which might have symptoms that include redness, pain, and occasionally itching.</p> <p><u>Arsenic Associated cancers</u>: From lowest to highest dose cancer effect levels, chronic arsenic exposures in people have been linked to lung cancer, basal and squamous cell skin cancers, liver cancer (haemangioendothelioma), urinary tract cancers (bladder, kidney, ureter, and all urethral cancers), and intraepidermal cancers. Intraepidermal is the name for the early pre-invasive form of squamous cell skin cancer. Pre-invasive means that the cancer cells are confined to the outermost layer of skin, the epidermis. At this stage, the cancer cells are unlikely to have spread to the lymph nodes, but they can spread along the skin surface. If left untreated, these cells can develop into an invasive cancer and spread into the lymphatic system.</p>			
[†] Other properties include right-of-ways, city-owned or vacant lot surface soil.				



Chemical	Soil Doses are in mg/kg/day		theoretical increased cancer risk	
	children's dose	adults' dose	children's	adults'
Barium (residences)	Ing 0.007 Inh 0.00003	Ing 0.0007 Inh 0.00003	No Slope No Unit Risk	No Slope No Unit Risk
Barium (other [†])	Ing 0.03 Inh 0.0001	Ing 0.003 Inh 0.0001	No Slope No Unit Risk	No Slope No Unit Risk
ATSDR 2006 (Update) Draft	<p><u>Child res. ingestion dose</u> (0.007) is 86 times less than the MRL. Rats dosed (115 mg/kg/day, NTP 1994) with barium chloride in their food for 90 days showed increased kidney weight. The NOAEL for this study was 65 mg/kg/day. ATSDR authors used the NOAEL from this study to derive the chronic MRL of 0.6 for barium exposure by dividing the NOAEL by 100 (10 to account for extrapolation from animals to humans and 10 for human variability).</p> <p><u>Adult res. ingestion dose</u> (0.0007) is 10 times less than the (0.007) dose referenced for children, we would not expect kidney or other health effects for most adults and children, at these exposure levels.</p> <p><u>Child other ingestion dose</u> (0.03) is 20 times less than the MRL of 0.6.</p> <p><u>Adult other ingestion dose</u> (0.003) is 200 times less than the MRL of 0.6. We would not expect kidney or other health effects for most adults and children, at these exposure levels, nor would we expect daily exposure to the non-residential subsurface soils.</p> <p><u>Inhalation exposure level</u> Medical case reports and animal studies are inadequate to establish the health effects of barium by inhalation. The lowest reported exposure level is 0.06 mg/m³/minute. Guinea pigs exposed for an unspecified amount of time to this concentration of aerosolized barium chloride solution experienced bronchoconstriction. The estimated exposure level, (0.0001) is 600 times less.</p> <p><u>Barium associated cancers.</u> Barium has not been shown to cause cancer in people or animals.</p>			
[†] Other properties include right-of-ways, city-owned or vacant lot surface soil.				



Chemical	Soil Doses are in mg/kg/day		theoretical increased cancer risk	
	children's dose	adults' dose	children's	adults'
Dioxin TEQ (residences)	Ing 0.000000001 Inh 0.000000000004	Ing 0.0000000001 Inh 0.000000000004	Ing 8:1,000,000	Ing 8:1,000,000
Dioxin TEQ (other [†])	Ing 0.000000001 Inh 0.000000000006	Ing 0.0000000001 Inh 0.000000000006	Ing 9:1,000,000	Ing 1:100,000
ATSDR 1998a (Update)	<p><u>Child res. ingestion dose</u> (0.000000001) is 1/120th the dose (0.00000012, Rier et al., 1993) associated with reproductive effects (moderate endometriosis) and altered social behavior in a rhesus monkey dioxin ingestion study. This dose is also equal to the MRL of 0.000000001 calculated from the 0.00000012 dose (Schantz et al., 192) at which rhesus monkeys showed adverse neurobehavioral and developmental effects. ATSDR authors divided this dose by an uncertainty factor of 90, 3 for use of a minimal LOAEL, 3 for extrapolation from animals to humans and 10 for human variability. The results of other oral animal studies suggest that the effects that occur at the lowest levels of dioxin doses are immune and endocrine, in addition to developmental effects. People's ingestion exposures are mainly known from low levels of food contamination.</p> <p><u>Adult res. ingestion dose</u> (0.000000001) is 10 times less than the residential children's dose. We would not expect health effects for most adults and children at these residential exposure levels.</p> <p><u>Child other ingestion dose</u> (0.000000001) is 1/120th the dose (0.00000012) associated with reproductive effects (moderate endometriosis) and altered social behavior in a dioxin rhesus monkey study and it too equals the MRL. The results of oral animal studies suggest that the effects that occur at the lowest levels of dioxin doses are immune, endocrine, and developmental effects. People's ingestion exposures are mainly known from low levels of food contamination.</p> <p><u>Adult res. ingestion dose</u> (0.000000001) is 10 times less than the residential children's dose. We would not expect health effects for most adults and children at these exposure levels in the areas of other land use.</p> <p><u>Inhalation</u> of dioxins has not been studied in animals. People's occupational and accidental exposures to dioxin involve primarily inhalation and dermal exposure, but health effects are known primarily from associations with the levels stored in fat. The lowest levels of exposure are associated with hormone changes that can result in changes in sex ratios in children (more females are born). Higher levels are associated with immunosuppression, changes in the liver, abnormal glucose tolerance, and increased risk of diabetes. The highest exposure levels are associated with nervous system effects, chloracne, respiratory effects, and increased risk of cancer.</p> <p><u>Cancers</u> Statistically significant increases in risks for all cancers were found in workers highly exposed to dioxins with longer latency periods. Although the estimated Standardized Mortality Ratios[†] are low[†], they are consistent across studies with the highest dioxin exposures. The evidence linking peoples' doses with site-specific cancers is weaker, with some data suggesting a possible relationship between soft-tissue sarcoma, non-Hodgkin's lymphoma, or respiratory cancers including lung cancer. Animal studies have also shown associations with liver thyroid and skin cancer.</p>			
Other properties include right-of-ways, city-owned or vacant lot surface soil.				

[†] Standardized Mortality / Morbidity Ratio (SMR) is a widely used method of reporting death or disease which adjusts for differences in age and sex across regions. It is a measure of premature mortality. Instead of giving an adjusted rate, the SMR gives a ratio that is a direct comparison with a standard (e.g. the entire state).



Chemical	Soil Doses are in mg/kg/day		theoretical increased cancer risk	
	children's dose	adults' dose	children's	adults'
PAHs TEQ (residences)	Ing 0.0002/0.00004 Inh 0.000001/0.00000007	Ing 0.00003/0.000002 Inh 0.000001/0.00000007	Ing 8:100,000/ 5: 1,000,000 Inh 1:10,000/ <1:1,000,000	Ing 8:100,000/ 6: 1,000,000 Inh 9:100,000/ 4:1,000,000
PAHs TEQ (other*)	Ing 0.000002 Inh 0.000000008	Ing 0.0000002 Inh 0.000000008	Ing <1:1,000,000 Inh <1:1,000,000	Ing <1:1,000,000 Inh 2:1,000,000
ATSDR 1995 (Update) * Other properties include right-of-ways, city-owned or vacant lot surface soil.	<p>We would not expect non-cancer health effects for most adults and children at these residential or other exposure levels. <u>Child res. ingestion dose</u> (0.0002) is 13,000 times less than the dose (2.6, Neal and Rigdon, 1967) associated with stomach cancer in mice exposed to benzo[a]pyrene ad lib in food for 30 to 197 day (non-cancer illnesses are all associated with much higher doses).</p> <p><u>Adult res. ingestion dose</u> (0.00003) is 6.6 times less than the residential children's dose.</p> <p><u>Inhalation res. exposure level</u> (0.000001) is 100 times less than the dose (0.0001, Gupta et al. 1993) associated with reduced lung function, abnormal chest x-ray, cough, bloody vomit, and throat and chest irritation, in persons exposed from 6 months to 6 years.</p> <p><u>Child other ingestion dose</u> (0.000002) is 1,300,000 times less than the dose (2.6) dose associated with stomach cancer in mice.</p> <p><u>Adult other ingestion dose</u> (0.0000002) is 10 times less than the residential children's dose.</p> <p><u>Inhalation other exposure level</u> (0.000000008) is 12,500 times less than the dose (0.0001) associated with reduced lung function, abnormal chest x-ray, cough, bloody vomit, and throat and chest irritation, in persons exposed from 6 months to 6 years.</p> <p><u>Cancer and occupational studies</u> Worker exposures to high levels of PAHs show cancers (skin, bladder, lung and gastrointestinal) are the most significant endpoint of PAH toxicity. Long-term worker PAH exposures have been linked with skin and eye irritation, photosensitivity, respiratory irritation (with cough and bronchitis), leukoplakia^Ω, precancerous skin growths enhanced by exposure to sunlight, erythema^Δ, skin burns, acneiform lesions, mild hepatotoxicity, and haematuria[€]. Also several PAH compounds are immunotoxic, and some suppress selective compounds of the immune system. Workers' dermal exposure studies indicate that although direct contact may be of concern at high exposure levels, they do not suggest that lower levels are likely to cause significant irritation (Goodfellow et al. 2001).</p>			

^Ω Leukoplakia is a common, potentially pre-cancerous disease of the mouth that involves the formation of white spots on the mucous membranes of the tongue and inside of the mouth. Despite the increased risk associated with having leukoplakia, many people with this condition never get oral cancer.

^Δ Erythema nodosum is an inflammation of subcutaneous fat tissue.

[€] Hematuria is passage of blood in the urine.

Appendix C—Safe Gardening Card



Safe Gardening Tips

REMEMBER THESE FEW SIMPLE STEPS, IF YOU WANT TO BE SAFE IN THE GARDEN:

PREPARING YOUR GARDEN

- Add clean compost or soil to your garden.
- Be sure phosphate and pH levels do not fall below recommendations.
- Ask your county agriculture extension office to evaluate your soil.

WORKING IN THE GARDEN

- Be sure to wear gloves.
- Don't eat, drink or smoke while in the garden.
- Avoid dust. Use mulch and do not garden in dry soil when it is windy.
- Remove shoes before entering the house.
- Wash your hands and dirty clothing after gardening.

PREPARING FRUITS AND VEGETABLES

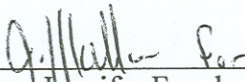
- Limit the amount of homegrown root crops you eat, especially carrots.
- Use raised beds of clean topsoil to grow root crops.
- Wash leafy vegetables growing close to the ground (like collards). Add a little vinegar to the wash water to help remove dirt.

FOR MORE INFORMATION see the Florida Department of Health website at: <http://www.myfloridahh.com/tsee/SUPERFUND/index.html>. Or call toll-free during business hours at 877-798-2772.



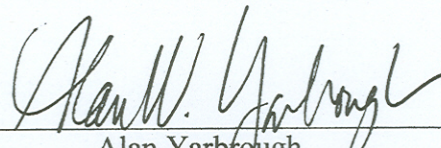
Certification

The Florida Department of Health, Bureau of Community Environmental Health prepared this Health Consultation under a cooperative agreement with the Agency for Toxic Substances and Disease Registry. Florida DOH followed approved methodologies and procedures existing at the time the health consultation was begun. The Cooperative Agreement Partner completed editorial review.



Jennifer Freed
Technical Project Officer
CAT SPAB, DHAC, ATSDR

The Division of Health Assessment and Consultation, ATSDR, reviewed this health consultation, and concurs with its findings.



Alan Yarbrough
Team Lead,
CAT, SPAB, DHAC, ATSDR