

# Public Health Assessment for

BROWN'S DUMP  
JACKSONVILLE, DUVAL COUNTY, FLORIDA  
CERCLIS NO. FLD980847016  
JUNE 26, 2000

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



**PUBLIC HEALTH ASSESSMENT**

**BROWN'S DUMP**

**JACKSONVILLE, DUVAL COUNTY, FLORIDA**

**CERCLIS NO. FLD980847016**

**Prepared by:**

**Florida Department of Health  
Bureau of Environmental Toxicology  
Under Cooperative Agreement With the  
Agency for Toxic Substances and Disease Registry**

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

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

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

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## FOREWORD

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

Since 1986, ATSDR has been required by law to conduct a public health assessment at each of the sites on the EPA National Priorities List. The aim of these evaluations is to find out if people are being exposed to hazardous substances and, if so, whether that exposure is harmful and should be stopped or reduced. If appropriate, ATSDR also conducts public health assessments when petitioned by concerned individuals. Public health assessments are carried out by environmental and health scientists from ATSDR and from the states with which ATSDR has cooperative agreements. The public health assessment program allows the scientists flexibility in the format or structure of their response to the public health issues at hazardous waste sites. For example, a public health assessment could be one document or it could be a compilation of several health consultations - the structure may vary from site to site. Nevertheless, the public health assessment process is not considered complete until the public health issues at the site are addressed.

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

**Health Effects:** If the review of the environmental data shows that people have or could come into contact with hazardous substances, ATSDR scientists evaluate whether or not these contacts may result in harmful effects. ATSDR recognizes that children, because of their play activities and their growing bodies, may be more vulnerable to these effects. As a policy, unless data are available to suggest otherwise, ATSDR considers children to be more sensitive and vulnerable to hazardous substances. Thus, the health impact to the children is considered first when evaluating the health threat to a community. The health impacts to other high risk groups within the community (such as the elderly, chronically ill, and people engaging in high risk practices) also receive special attention during the evaluation.

ATSDR uses existing scientific information, which can include the results of medical, toxicologic and epidemiologic studies and the data collected in disease registries, to determine the health effects that may result from exposures. The science of environmental health is still developing, and sometimes scientific information on the health effects of certain substances is not available. When this is so, the report will suggest what further public health actions are needed.

**Conclusions:** The report presents conclusions about the public health threat, if any, posed by a site. When health threats have been determined for high risk groups (such as children, elderly, chronically ill, and people engaging in high risk practices), they will be summarized in the conclusion section of the report. Ways to stop or reduce exposure will then be recommended in the public health action plan.

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

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

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

Letters should be addressed as follows:

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

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## Summary

The Brown's Dump site at 4330 Pearce Street encompasses approximately 50 acres in Duval County, Florida. From 1949 to 1955, the city of Jacksonville disposed of municipal solid waste and municipal incinerator ash at the site. In 1955, the city built the Mary McLeod Bethune Elementary School on part of the site. Houses and an electrical substation occupy the rest of the site. The surface soil is contaminated with metals and organic chemicals. The sediments and water in Moncrief Creek are contaminated. Groundwater is also contaminated with metals.

The Duval County Health Department requested a review of environmental data in the Environmental Protection Agency's March 1998 expanded site investigation report. In this public health assessment, we evaluate the potential for health effects from exposure to contaminated surface soil, sediments, surface water, and groundwater.

Currently, this site is a public health hazard because potential incidental ingestion of lead-contaminated surface soil inside the Environmental Protection Agency's Emergency Response/Removal Branch (ER/RB) fencing could result in illness. Fencing around the site is currently intact. In the past, the fencing around this area has not been consistently maintained. If access is not restricted and the fencing is not constantly maintained, the site will be a public health hazard. Soil in the elementary school playground area is currently not a health threat. If, in the future, the surface soil erodes or is disturbed, the playground could become a health threat. Contaminated surface soils in the residential areas are a potential threat. Sampling of residential soils; however, has been inadequate. Contaminated surface water and sediments in Moncrief Creek are not a public health threat. Groundwater is not a completed exposure pathway and therefore not a public health threat.

We recommend that the city of Jacksonville continue to ensure that nearby residents and school officials are aware of contamination within the area enclosed by the ER/RB fencing and that they know how to minimize exposures. We recommend that the city restrict access to the area inside the ER/RB fencing to limit exposure to lead-contaminated surface soil. We recommend that the city check the fences around the site monthly and repair damage. We recommend the city post warning signs on the fence. We also recommend periodic sampling of soil in the playground. We recommend additional sampling in the residential area, Moncrief Creek and groundwater to determine the extent of contamination. If land use changes or if additional environmental sampling data becomes available, we will reevaluate the potential public health threat.

## Background

### A. Site Description and History

In April 1998, the Duval County Health Department (CHD) requested assistance from the Bureau of Environmental Toxicology of the Florida Department of Health (FDOH). The Duval CHD requested assistance reviewing the analyses of surface soil and water collected in July 1977 by Tetra Tech EM, Inc., for the U.S. Environmental Protection Agency (EPA) Expanded Site Inspection (ESI) Report at the Brown's Dump Site in Jacksonville. The ESI report was completed on March 6, 1998.

In this public health assessment report, we evaluated the public health threat based on the surface soil and water samples collected in July 1997, reported in the 1998 ESI. In a previous health consultation report (February 1997), we assessed the public health threat based on the environmental data available at that time (FDOH 1997).

The Brown's Dump site encompasses approximately 50 acres in Duval County, Florida (Figure 1, 2, and 3). From 1949 to 1955, the city of Jacksonville disposed of municipal solid waste and municipal incinerator ash at the site. In 1955, the Duval County School Board (DCSB), formerly the County Board of Public Instruction, built the Mary McLeod Bethune Elementary School on 14 acres in the central portion of the site. The Jacksonville Electric Authority electrical substation occupies about 2 acres in the northeastern portion of the site. The site includes residential areas such as the Bessie Circle apartments to the west of the elementary school and the Moncrief Village and Palm Terrace apartment complexes in the northern part of the site. Moncrief Creek runs through the northwestern part of the site (EMCON 1995).

Between 1994 and 1996, the Duval County Health Department and EPA sampled surface soil and ash samples for lead. Lead was typically in the 1000 - 2000 parts per million (ppm) range for these samples. The areas around Moncrief Creek had the highest concentrations of lead in the soil (more than 5,000 ppm). The highest levels found and the percentage of samples greater than 500 ppm are in Table 1.

FDOH evaluated this data in a February 1997 health consultation. We concluded the site was a public health hazard because children could incidentally ingest lead at high concentrations in the soil. Blood lead data, however, indicated children had either not ingested contaminated soil in the few months prior to the blood lead testing event, that children did not come into contact with the lead contaminated soil a few months prior to the blood lead testing event, or that the lead in the soil was not bioavailable (bioavailability of lead in soil is virtually impossible to determine because it is dependent on a multitude of interacting factors). Because other organic chemicals, such as polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans

(PCDFs), are typically found in incinerator ash, we recommended additional soil testing. We also recommended restricting site access (FDOH 1997).

In late 1995 and early 1996, the city placed six inches of clean soil around the basketball court and playground area, at the gate along the western property line near the Bessie Circle apartments, and on the western entrance to the courtyard between the southern two elementary school buildings (Figure 2). The DCSB repaired the fence along the elementary school's western property line. They installed a fence around the parking lot in the front of the elementary school to control pedestrian access during and after school hours (Young, February 1996).

In July 1997, EPA Emergency Response and Removal Branch used an x-ray analysis machine to test the soils for lead. They found a maximum of 1,266 parts lead per million parts soil (ppm) in the residential area. Quality assurance and quality control of the data was not presented in the report (EPA 1997a).

Also in July 1997 the EPA and contractors collected surface soil samples from residential yards, the school playground, and from school property behind the Emergency Response and Removal Branch (ER/RB) fencing (See Figure 3 for location of the ER/RB fence). EPA analyzed surface soil for organic chemicals typically found in incinerator ash (PAHs, PCBs, PCDDs, and PCDFs), as well as inorganic and organic chemicals. They collected sediment and surface water samples from Moncrief Creek and groundwater samples. They analyzed the samples for inorganic and organic chemicals. The maximum amount of lead found in the residential area was 1900 ppm (EPA 1998b). We used this information to assess the health threat at the Brown's Dump site. The results of EPA's sampling are in Tables 2-5.

## **B. Site Visit**

On December 3, 1996, Randy Merchant and Julie Smith of the FDOH, Bureau of Environmental Toxicology and Mary Nogas and Richard Rachal of the Florida Department of Environmental Protection visited the site. The northern portion of the school property was fenced off; however, the fence gates were open. There was a cut in the fence at the Bessie Circle West cul-de-sac that allowed access to the site.

On July 16, 1998, David Kincaid of the Duval CHD visited the site. He reported that a fence surrounding the northern portion of the elementary school property had an opening at the fence at Bessie Circle West cul-de-sac. He observed that parts of the fence were in disrepair (see Figure 3).

On December 10, 1998, Ms. Bland of the FDOH Bureau of Environmental Toxicology visited the site. She observed a new, intact six-foot chain link fence at the end of

Bessie Circle West. She also noticed two openings along the fence around the school playground: one on 33<sup>rd</sup> Street and one on Bessie Circle East. She noted that the 4-foot high fence between Bessie Circle West and Moncrief Creek was pushed down to about 2.5 feet (See Figure 3).

On January 6, 1999, Randy Merchant and Lu Grimm of the FDOH Bureau of Environmental Toxicology visited the site. They observed that the gate dividing the school playground from the contaminated area was so loosely chained that people could easily slip through the gate. They also observed that a tree which had fallen on the fence at the end of Bessie Circle West allowed access to the contaminated area.

On March 25, 1999, Julie Smith and Beth Copeland of the Florida Department of Health, Bureau of Environmental Toxicology, visited the site. They observed the gate leading from the school playground to the contaminated area was wide open. They observed children playing along the fence line and around the open gate. On the morning of March 25, the fence at the end of Bessie Circle was down; a truck had flattened it level with the ground. In the afternoon, workers came to fix the fence at the end of Bessie Circle. Workers said they were told to check the fence once a year. Nash Road, on the other side of Moncrief Creek, was freshly paved.

Paths through the contaminated area lead to Moncrief Creek, an area attractive to trespassers. There is evidence trespassers have used the paths through highly contaminated areas in the past. There is a well worn path along Moncrief Creek behind the school, and it is littered with trash and a basketball was in the creek. There is a path leading from contaminated area behind the school in the direction of Moncrief Creek.

### **C. Demographics, Land Use, and Natural Resource Use**

According to the 1990 Census, approximately 3,930 people (6% white, 90% black, 1.5% Hispanic, and 2.5% other) live within one-half mile of the site. About 16% of the population is under the age of 9 and 18% of the population is over the age of 65. About 48% of the population over age 25 graduated from high school. About 37% have less than a 9<sup>th</sup>-grade education. The median family income is about \$17,814. Eighty-five percent of the housing units are occupied (Bureau of Census, 1990).

The area around the site is residential. The Mary McLeod Bethune Elementary School is on the site and seven child care centers are within one-half mile of the site (Bureau of Census, 1990).

## D. Health Outcome Data

Between May 24 and June 5, 1995, the Duval CHD conducted free lead screenings for prekindergarten and kindergarten children in the area. They screened a total of 203 children (194 children confirmed for the 1997 Health Consultation) in the area: from the elementary school; children living in the Bessie Circle, Moncrief Village, and Palm Terrace apartment complexes, and children from a nearby daycare (Goldhagen 1997). They used the capillary method for blood level testing (FDOH 1997). We discuss the results of this screening in the *Public Health Implications, Health Outcome Data Evaluation* section.

## Community Health Concerns

In May 1999, the FDOH held a public availability session to collect health concerns from nearby residents. In July 1999, FDOH attended a community meeting to collect resident's concerns and report on our findings in the public health assessment. In this section, we identify the community health concerns. We address each community health concern presented in the *Public Health Implications* section, *D. Community Health Concerns*.

What chemicals were found?

What is the effect of lead on plants in the Dodge/Nash area?

Will anyone do more testing?

What are the boundaries of contamination?

How were carcinogens addressed in assessment?

What about kids over 6 and adults?

Twenty three of my neighbors have died. Is this related to the site?

The statement that higher levels of chemicals are fenced off so that people cannot touch them is misleading, creating a false sense of security.

There are other hazardous substances found on the school property above health action levels (arsenic, PCB's, dioxin).

There is no established safe level of lead exposure for children 6 and under.

Residents say children travel through the open gate on school grounds.

Why does FDOH say they do not think the site made people sick in the past?

Why were the children not tested for other chemicals besides lead?

Could the children whose blood leads were normal in 1995 have been exposed to lead before or after the 3-4 months of exposure and could they have lead in their brain, kidneys, liver or bones?

Why does the report not mention the extent of the contamination in the residential area north of the CSX railroad tracks?

Why did the City place 6 inches of soil on playground?

- Why are the lead levels getting higher as time goes by?
- What were the findings of the EPA 1998 sampling?
- Why doesn't a concentration above ATSDR comparison value represent a health threat?
- Will more sampling give more information about the contaminants, for example aluminum?
- Where are the N qualifiers in this report?
- Why do we say there is not an increased risk of cancer to arsenic when we know arsenic causes cancer?
- Why do we say that people have been exposed to copper but we do not expect people to be at an increased risk of illness from incidentally swallowing copper-contaminated soil?
- Why do we say that swimming in Moncrief Creek is not a health hazard?

### **Environmental Contamination and Other Hazards**

In this section, we review the environmental data collected at the site in July 1997, evaluate sampling adequacy, and identify contaminants with the greatest potential of harming health (contaminants of concern). Every natural or manmade contaminant cannot be tested at once due to limited resources and time. We must identify those contaminants that possibly cause health problems in humans. We select contaminants of concern based on the following factors:

*1. Concentrations of contaminants on and off the site.*

We compare maximum concentrations at the site with published ATSDR standard comparison (screening) values. Since there are typically voluminous quantities of environmental sampling data, ATSDR's published standard comparison values help us narrow the list of contaminants so we can concentrate on the ones that have the potential to cause health problems. They are not used to predict health effects or to select clean-up levels. These screening values are based on doses that are not likely to cause illness. They are derived to protect the most sensitive members of the population (e.g., children), and are not cut-off levels, but rather screening values. They are very conservative concentration values designed to protect the public.

Contaminants with media concentrations above an ATSDR standard comparison (screening) value do not necessarily represent a health threat, but are selected for further evaluation. For further evaluation, we estimate a dose based on site specific information and the level of a contaminant in media and compare it to health guidelines called Minimal Risk Levels (MRLs). See *Health Guidelines, Public Health Implications* section for a description of the MRL.

We eliminated contaminants that are essential human nutrients present at low concentrations. Examples of these chemicals are iron, calcium, potassium and sodium.

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

Before using environmental data to reach conclusions in the public health assessment, the data must be analyzed as to its quality and to assure standard operating procedures were followed during collection. The quality of the data is discussed in this section, *Environmental Contamination and Other Hazards, Section B. Quality Assurance and Quality Control.*

*3. Community health concerns.*

Community concerns are a main part of the public health assessment. They expand our knowledge of past and present activities and issues at the site. Community health concerns are discussed in the *Public Health Implications* section, *D. Community Health Concerns.*

*4. Completed and potential exposure pathways.*

Contaminants can only harm people if they contact the contaminants. Pathways are discussed under the *Pathway Analysis* section.

*5. Toxicological Information.*

If a contaminant is above the screening level, we estimate the dose (the amount people might take into their bodies) based on concentrations of contaminants in the soil, sediments and surface water and compare them with health guidelines called MRLs or toxicological information including information from ATSDR toxicological profiles. These profiles are chemical specific and summarize toxicological information found in the scientific and medical literature. From this information, we can determine if there is a possibility the contaminant may cause illness. The toxicological information is discussed in the *Public Health Implications* section, *Toxicological Evaluation.*

We used the following ATSDR standard comparison values (ATSDR 1998a), in order of priority, to select contaminants of concern.

1. EMEG—'Environmental Media Evaluation Guide'- are not site-specific and are not predictive of health effects. They are only used to select contaminants for further evaluation. They are based on levels unlikely to cause illness. They are derived to protect the most sensitive members of the population and are not cut-off levels but rather screening levels. Contaminants below screening values are unlikely to pose a health threat

and not evaluated further. Contaminants above the screening value are evaluated further by estimating a dose and comparing the dose to health guidelines, the Minimal Risk Level (MRLs). See *Health Guidelines, Public Health Implications* section for a description of the MRL.

2. CREG--'Cancer Risk Evaluation Guide'--ATSDR calculated CREGs from the EPA's cancer potency factors, a contaminant concentration estimated to result in no more than one excess case of cancer per million persons exposed over a lifetime.
3. RMEG--'Reference Dose Media Evaluation Guide'--ATSDR derived RMEGs from the EPA's reference dose (RfD) value, using standard exposure assumptions. RfDs estimate the maximum amount of a contaminant that a person could be exposed to without increasing the risk of noncancer illness.

Identifying a contaminant of concern does not necessarily mean that exposure to a contaminant will be associated with illnesses. Identification serves to narrow the focus of the public health assessment to those contaminants most important to public health. We evaluate the contaminants of concern in subsequent sections and decide whether exposure has public health significance.

#### **A. On-site Contamination**

For the purpose of this public health assessment, we define "on-site" as the area of possible contamination as shown in Figure 2. For the 1998 ESI, EPA contractors collected three surface soil samples from the Mary McLeod Bethune Elementary School property behind ER/RB fencing, four samples from the playground, and seven samples near the street of six homes in the nearby neighborhood (See Figure 2). EPA contractors also collected one sample in front of the school property on 33rd Street and one background sample outside of the contaminated area (EPA 1998b).

All surface soil samples were collected from a depth of 0 - 3" below the surface. EPA analyzed the samples for metals, pesticides, PCBs, cyanide, volatile organic chemicals, extractable organic chemicals, and dioxins (EPA 1998b). While no area of the school property or the immediately surrounding neighborhood was free of contamination, EPA detected relatively lower levels in areas surrounding the school buildings and playground (Tetra Tech 1997).

EPA detected metals and organic chemicals in the surface soil. Forty percent (6 out of 15 soil samples) contained lead at concentrations greater than 400 ppm, the EPA's *proposed* health action level in 1998 (EPA 1998a). Three of the seven residential yards contained soil lead at concentrations greater than 400 ppm. All

soil samples in the playground area contained lead concentrations less than 400 ppm; however, the EPA detected other metals and organic chemicals including PAHs in the playground soil samples. Results of sampling for the EPA's ESI are in Table 2 in the Appendix.

EPA collected four sediment samples from Moncrief Creek, which is used for recreation. They found elevated levels of mercury and lead as well as other inorganic compounds (EPA 1998b). Results are in Table 3 in the Appendix.

EPA collected four surface water samples from Moncrief Creek. The water samples are contaminated with arsenic, lead, zinc, and other inorganic compounds (EPA 1998b). Results are in Table 4 in the Appendix.

EPA collected four groundwater samples. Three groundwater samples were collected from behind the ER/RB fencing and one from in front of the playground on 33rd Street West. They detected elevated levels of inorganic and organic contaminants (EPA 1998b). Results are in Table 5 in the Appendix.

## **B. Quality Assurance and Quality Control**

In preparing this public health assessment, we relied on the existing environmental data. We used laboratory-reported data. EPA contractors who collect and analyze environmental data must follow EPA-approved protocol and follow standard operating procedures. They must show adequate quality assurance and quality control measures concerning chain-of-custody, laboratory procedures, and data reporting. For example, a contract laboratory must show they calibrate the laboratory equipment before and after analysis. They analyze blank and spiked samples and perform checks on the laboratory equipment during the analysis. In addition, EPA employees observe the contractors at work. The completeness and reliability of the referenced information determine the validity of the analyses and conclusions drawn for this public health assessment.

In each of the preceding subsections, we evaluated the adequacy of the data to estimate exposures. The laboratory uses "qualifiers" to describe the confidence they have in the accuracy of the data. Some of the lead in the soil was qualified with a "J" qualifier. We assumed that estimated data, J qualified was valid. Laboratory quality assurance procedures add "J" qualifiers to the data to indicate uncertainty in the quality of the contamination level. For every tenth sample the laboratory analyzes, they analyze a known quantity of contaminant to see what the equipment reads. If the equipment reads a higher or lower level of contaminant due to equipment variability, the previous nine samples obtain a "J" qualifier. Therefore, a "J" qualifier does *not* mean the contaminant was not there at the level indicated by the equipment, but indicates uncertainty in the equipment reading. In addition, due to past sampling events, we are certain that lead is in the soil at higher levels than identified in this current limited sampling

event.

Another qualifier the laboratory uses to describe the data are "N" qualified presumptive data. The "N" qualifier means there was uncertainty in the identity of the compound. The qualifiers are on the laboratory analysis data sheets. Lead in the area behind the school was labeled as presumptive; however, due to the history of the site, we know lead is present in the soil at these levels. Therefore, we included it in our assessment. We did not carry tentatively identified organic compounds through the quantitative assessment (EPA 1989).

### **C. Physical and Other Hazards**

A well-maintained fence surrounds the Jacksonville Electric Authority electrical substation in the northeastern part of the site. A fence surrounds the school area. Pieces of broken glass in the soil of the school yard pose a hazard.

### **Pathway Analysis**

The amount of contact people have with hazardous substances is essential to assessing the public health significance of a contaminant. Chemical contaminants in the environment have the potential to harm human health, but only if people contact those contaminants.

The way an individual contacts contaminants is called an exposure pathway. To decide whether nearby residents have contacted contaminants from the site, we looked at the human exposure pathways. An exposure pathway has five elements. The first element is contamination source, such as an industrial site. The second element is an environmental media, such as air or groundwater, which moves a contaminant from the source to a place where people can contact the contaminant. The third element is a location where people could contact the contaminated soil or groundwater, such as topsoil or drinking water wells. The fourth element is the method of exposure, such as drinking contaminated water or touching contaminated soil. The fifth element is a person or group of people who can potentially come in contact with the contamination, such as people living or working near the contaminated site. A completed exposure pathway includes all five elements.

We eliminate an exposure pathway if at least one element is missing and will never be present. For completed pathways, all five elements exist and exposure to a contaminant has occurred, is occurring, or will occur. For potential pathways, exposure to a contaminant could have occurred, could be occurring, or could occur in the future.

The public health findings for communities surrounding Brown's Dump site are based on a review of recent environmental data to identify past, present, and

future exposure pathways. In this assessment, we identified exposure pathways that are significant to public health (See Table 6 in the Appendix).

### **A. Completed Exposure Pathways**

In the past, people may have been exposed to contaminated surface soils soil behind the ER/RB fencing, in residential yards and at the Mary McLeod Bethune Elementary School playground soil. Because there was an opening in the fence at Bessie Circle leading to the area inside the ER/RB fencing, we assume that children may have been exposed to contaminated surface soil inside the ER/RB fence in the past.

Currently people are likely be exposed to contaminated surface soils in residential yards and at the playground. Children attending the Mary McLeod Bethune Elementary School are likely to contact the playground soil. Children and adults are likely to contact contaminated surface soil in the residential area.

Soil ingestion is an important exposure route for children younger than six years old. Children are likely to consume a significant amount of soil, compared with adults, from playing outdoors and hand-to-mouth activity. Although adults are less likely to eat significant amounts of soil, soil ingestion remains a potentially significant exposure source to environmental contaminants through hand-to-mouth activities. People may touch the soil, soil covered trees, branches, rocks or grass. Then when they put their hands to their mouth when smoking or eating, they incidentally eat the soil. Breathing contaminated dust from the soil is also a significant exposure route for children and adults. Pound for pound, children breath more than adults due to their smaller size.

Past and current exposure may result from contact with contaminated surface water and sediment from Moncrief Creek. Moncrief Creek borders the site on the west and north. EPA estimated the dump site and ash extends to Moncrief Creek. Local governmental officials reported that children play in the creek. It is also used for recreation. Access to the creek is not limited.

### **B. Potential Exposure Pathways**

Currently, the contaminated soil behind the ER/RB fencing is not accessible to the community because the fencing is intact. If in the future, the fences are not repaired when damaged, people could again come into contact with the contaminated soil. In the past, the fences have not been consistently maintained.

Future exposure may result from contact with contaminated surface water and sediment from Moncrief Creek.

### **C. Eliminated Exposure Pathways**

We eliminated drinking contaminated groundwater as an exposure pathway because people in the area do not use private drinking water wells (personal communication July 14, 1998, Grazyna Pawlowicz, Duval CHD).

### **Public Health Implications**

In this section we will discuss how and when people contact contaminants and the public health implications of those exposures. We estimated an exposure dose of each contaminant a child (for potential noncancer effects) and adult (for potential carcinogenic effects) might receive by coming into contact with contaminated surface soil.

For non-cancerous contaminants in surface soil, we estimated the dose that an elementary school child weighing 24 kilograms (50 pounds) would receive by incidentally eating 200 milligrams of contaminated surface soil 350 days a year for 6 years. Scientific studies reveal a reasonable maximum amount of incidental soil ingestion for children is 200 mg/day. Children could be exposed 350 days a year because they would not just contact the contamination while at school; they could also contact contaminated soil after school during play time, weekends and in their yards. We assume children are away from home for about two weeks each year. Children represent a sensitive subpopulation. Exposures that are protective of children are most likely protective of adults. For carcinogenic compounds, we estimated an exposure dose that an adult weighing 70 kilograms (150 pounds) would receive over a lifetime (estimated at 70 years) of incidentally ingesting 100 milligrams of surface soil a day, 350 days out of the year (Risk Assistant 1994).

We also estimated how much dust from contaminated soils residents might breathe. For a child, we used an inhalation value of 0.76 cubic meters an hour; this corresponds to one-third of a day at rest and the rest of the day playing (not heavy activity). For adults, we used 1.67 cubic meters per hour; this corresponds to one-third of the day at rest and the remaining day divided between light, moderate, and heavy activity (Risk Assistant 1994).

It is widely accepted that one estimate of blood lead only provides an indication of the amount of lead circulating in the blood at one point in time and may be a poor index both of overall body lead burden and of long-term exposure to lead. The health outcome data consists of blood lead levels of area children collected by the Duval CHD. Blood lead data reflects only recent exposure. The Duval CHD also taught people ways to reduce their exposure. Due to the heightened awareness of lead in the soil, residents may have temporarily changed their behavior in ways that resulted in reduced blood lead levels during the time of the blood lead sampling. Therefore, we used two mathematical models in addition to

looking at the health outcome data to assess the public health threat for lead at this site (Landsdown and Yule 1986). We used mathematical models to predict the probability of an illness as a worst case scenario - to compensate for the possibility that residents might have changed their behavior during blood lead testing.

Models incorporate information about adverse health effects we know from scientific and epidemiological studies. The Arnhem lead study showed that the most important determinant of blood lead concentrations in children were the levels of lead in soil and dust both indoors and outdoors (Brunekreff et al. 1981 in Landsdown and Yule, 1986). Numerous epidemiological studies have shown a positive correlation between lead in soil and lead in blood (Steele et al. 1990; ATSDR 1998b). The studies showed an increase of 1 to 8 ug/dL of blood lead in young children living in residential area with lead-contaminated soil for every 1000 ppm lead in soil. The variation in blood lead values is a factor of the source of lead, the type of lead, the size of lead particles, the degree of contact with contaminated areas, the concentration of lead and the behavior of children. Because of these epidemiological studies, elevated levels of lead might increase blood lead level in the future for some children living in lead-contaminated yards and playing in lead contaminated fields and creeks.

There are many ways, in addition to soil lead, that people can be exposed. Many fruits, vegetables, grains, meat and soft drinks contain lead. Drinking water may contain lead (usually less than 0.005 milligram per liter). Children living in older houses may eat lead-based paint chips from peeling surfaces. Lack of nutrients in the diet such as iron or calcium may aggravate the toxic effects of lead (ATSDR 1998b).

The models we used include health assessment (risk assessment) techniques and predicted blood lead levels based on soil lead concentrations using the EPA's integrated exposure uptake biokinetic (IEUBK) model. The IEUBK model is useful in comparing blood lead levels from toxicological studies while the health assessment technique allows us to compare exposure doses. The IEUBK model predicts blood lead levels in children less than six years of age in a residential setting. The model predicts blood lead levels for children exposed to lead in soil and other sources. Since children's behavior such as time spent outdoors and size change with age, default data used in the model changes with age. We used site-specific data for exposure resulting from incidentally ingesting soil. Children may incidentally ingest soil from mouthing objects or hands. Mouthing behavior is normal in children. For estimating ingestion of soil and dust, the model uses the value of 200 mg of outside soil per day and 100 mg of household dust per day (EPA 1990).

For contaminants in sediment, we estimated the dose that a person might receive by incidentally eating the sediment. For noncancerous compounds, we

estimated the amount a child might eat and for carcinogenic compounds, we estimated the amount an adult might eat, using the same exposure estimates that we used for contaminants in surface soil. This estimate is conservative because sediment would likely wash off during exposure leading to less sediment incidentally consumed than soil.

For noncarcinogenic contaminants in surface water, we estimated the dose an elementary child might receive from swimming or wading in the creek. Swimming can expose large areas of skin to inorganic contaminants that can be absorbed from water through the skin. We estimated that a child weighing 24 kilograms would swim 7 times per year for 2.6 hours (national average for swimming; USEPA 1989). We estimated the body surface area for children aged 6 - 9 years old and for adults (USEPA 1989).

Although dermal exposures are the most obvious exposures associated with swimming in contaminated surface water, incidental ingestion of contaminated surface water may also contribute to exposure. We estimated incidental ingestion of 50 milliliters of water per hour of swimming (USEPA 1989).

To evaluate our estimated dose of each contaminant, we compared our exposure dose with ATSDR and EPA health guidelines. These health guidelines help us screen the contaminants that require further investigation. Guidelines alone, however, cannot determine a particular contaminant's potential health threat. If exposure dose estimates were less than the health guideline, we did not evaluate the contaminant further. If exposure dose estimates exceeded the health guideline or if no health guideline existed, we then compared exposure estimates with doses in human or animal studies and described the results.

#### Health Guidelines

For noncancerous contaminants, we compared our exposure estimate with health guidelines such as ATSDR's minimal risk level (MRLs) and EPA's reference doses (RfDs). RfDs and MRLs estimate the level of contamination that a person could be exposed to at the maximum duration and frequency without increasing the risk of noncancerous illness. MRLs are ten to one-thousand times lower than the lowest levels causing the most sensitive endpoint (illness) in the most sensitive animal species study. MRLs are also screening values. We used a dose estimate based on the maximum level of contamination to compare to an intermediate duration MRL and a dose estimate based on a measure of central tendency to compare to chronic duration MRLs. We used long-term exposure MRLs before intermediate duration MRLs, if both existed. We used intermediate duration MRLs before short-term exposure MRLs, if both existed, because our exposure estimates are based on a longer term exposure.

For cancerous contaminants, we compared our exposure estimates with EPA's cancer potency factors. We used a potency factor to estimate an upper-bound

probability of an individual developing cancer from a lifetime of exposure to a particular level of a potential carcinogen (ATSDR 1992a). These estimates are based on the assumption that there is no safe level of exposure to a carcinogenic contaminant so instead of looking for a level that will not cause harm, we look for the risk of harm. Each exposure carries some degree of risk, no matter how small. To err on the side of safety, these estimates may overestimate the risk associated with cancer. When examining cancer risks, it is important to recognize the background cancer rate in the United States is about 25% or 250 in 1,000 (ATSDR 1993a).

## **A. Toxicological Evaluation**

Very little is known about the toxicity of dibenzofuran and carbazole. Because so little is known about this toxicity, we are unable to assess the public health threat from exposure to these contaminants at this site.

We do not expect any adverse effects from exposure to contaminated sediment. We do not expect any adverse effects from exposure to soil or surface water contaminated with aluminum, cobalt, antimony, mercury, silver, bis(2-ethylhexyl)phthalate, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), toxicity equivalency to dioxin and dioxin-like compounds (TEQ), dieldrin, barium, chromium, manganese, magnesium or zinc. Our estimated dose from all of these contaminants was below our minimal risk level (MRL) or EPA's reference dose and cancer potency factors. If a MRL or RfD did not exist for a contaminant, our estimated dose was far below the level associated with adverse effects in humans or animals (ATSDR 1989, ATSDR 1990b, ATSDR 1992b, ATSDR 1992c, ATSDR 1992d, ATSDR 1993c, ATSDR 1993d, ATSDR 1993e, ATSDR 1994, ATSDR 1995, ATSDR 1997a, ATSDR 1997b, ATSDR 1998d, ATSDR 1998e).

In the following section, we discuss contaminants at concentrations above health guidelines (MRLs).

### **Lead**

Lead is a naturally occurring bluish-gray metal. Natural and manufactured substances contain lead, and it is also found in incinerator ash. Certain people have a higher risk of illness from lead exposure. Children are more sensitive than adults because of their smaller size because their nervous systems are developing. The fetus is susceptible to toxic effects from lead crossing the placental barrier. Women exposed to lead before pregnancy store lead in their bones. The stress of pregnancy may cause lead to move from bone to the fetus.

There does not appear to be a threshold for the effects of lead (ATSDR 1998b).

Exposure to lead causes a wide range of effects. The level of lead in blood is a good measure of recent exposure to lead. A level greater than 10 micrograms of lead per deciliter of blood ( $\mu\text{g}/\text{dL}$ ) indicates that excessive exposure may be occurring; however, there does not seem to be a threshold level (a blood lead level that is safe) (CDC 1991 in ATSDR 1998b). Some studies show adverse health effects for children with blood lead levels between 10-15  $\mu\text{g}/\text{dL}$ . These health effects include behavior problems, hearing deficits and intelligence problems such as reduced number skills and reading skills (Schwartz and Otto 1991 and Silva et al. 1988 in ATSDR 1998b). Blood lead levels of 40-60  $\mu\text{g}/\text{dL}$  are markedly elevated in children and scientists and doctors generally accept adverse neurological effects (lower intelligence) can occur (ATSDR 1998b).

The highest levels of lead found in the residential soil was 1900 parts per million (ppm). Results of the IEUBK model are based on exposure that might occur, not on exposure that has actually occurred. The model predicts that exposure to lead at the maximum surface soil level found in the residential area would result in a blood lead about 18  $\mu\text{g}/\text{dL}$ . Based on our dose estimate of children who incidentally swallow lead-contaminated residential surface soil, a child's exposure dose is three times higher than the level causing adverse blood enzyme effects (decreased ALAD activity and increased red blood cell porphyrin) in people (ATSDR 1998b). Our dose estimate is very conservative. We used the maximum amount of lead found in the residential area and the maximum number of times a child could contact the lead. It is unlikely that people would come into contact with that level of lead in the soil every day, 350 days per year for six years. Our estimates are for children six and under, the most sensitive population.

Results of the IEUBK model predict that exposure to lead at the maximum surface soil level found on the school property inside the ER/RB fencing (9,100 ppm) would result in a blood lead level of 74  $\mu\text{g}/\text{dL}$  suggesting lead-poisoning (EPA 1990). Based on our dose estimate, children who incidentally swallow lead-contaminated surface soil inside the ER/RB fencing may be at risk for some adverse health effects. Our estimated exposure dose for a child is 14 times higher than the level causing adverse blood enzyme effects (decreased ALAD activity and increased red blood cell porphyrin) in people (ATSDR 1998b).

We do not expect illness from breathing lead-contaminated dust from contaminated soil in the fenced area. The lead children could breathe from dust is 13 to 63 times lower than the lowest level shown to cause illness in people (ATSDR 1998b).

EPA concluded that the human data are inadequate to refute or confirm the potential carcinogenicity of lead exposure. Nonetheless, EPA concluded that the animal data are sufficient to indicate that lead and lead compounds, particularly soluble lead salts, are carcinogenic to animals, and they classified lead as a probable human carcinogen (ATSDR 1998b). EPA does not have a cancer potency factor to use to compare the carcinogenic effects of lead. There is not enough information available to assess the carcinogenic potential of lead.

## **Arsenic**

Arsenic is a naturally occurring gray, metal-like material naturally found in rocks. Smelters are used to heat the rocks and separate the arsenic for use as a wood preservative, insecticide, or weed killer (ATSDR 1993b). Arsenic is also found in incinerator ash. People may have been exposed to arsenic in the surface soil behind the ER/RB fencing and surface water.

Our estimated exposure dose for a child swallowing arsenic-contaminated surface soil at the site is slightly higher than the MRL. The MRL is based on a dose that did not cause illness in people. We searched the toxicological literature for levels causing illness in people. Our estimated dose is 52 times lower than the lowest exposure dose causing illness in people (ATSDR 1993b). Therefore, we do not expect any illness from people incidentally swallowing arsenic-contaminated surface soil.

Our estimated exposure dose for a child breathing dust from arsenic-contaminated surface soil is thirty thousand times lower than the level causing illness in people (ATSDR 1993b). Therefore, we do not expect any illness from people incidentally breathing arsenic-contaminated dust.

The exposure dose we calculated for a child swallowing arsenic-contaminated surface water is below the MRL. We do not expect any illness from people incidentally swallowing arsenic-contaminated surface water.

Scientific studies show clear evidence that arsenic exposure increases the risk of cancer. Inhalation exposure increases the risk of lung cancer, and oral exposure increases the risk of skin cancer (ATSDR 1993b). Instead of looking for a level that will not cause illness, we assume that people will be harmed and look for the risk of harm. We used the EPA's cancer potency factors for arsenic to assess the risk of harm at this site. Our best estimate of an adult's exposure from swallowing arsenic-contaminated surface soil or surface water or breathing arsenic-contaminated dust is unlikely to result in an increased risk of cancer in this population.

## **Copper**

Copper is a naturally occurring reddish metal, but many copper compounds are blue-green. Copper is used for pennies, electrical wiring, and pipes. It is found in incinerator ash. Small amounts of copper in the diet are necessary for good health (ATSDR 1990a). People may have been exposed to excess copper from residential and playground soil and from surface soil behind the ER/RB fencing.

We do not expect people who incidentally swallow copper-contaminated surface soil behind the ER/RB fencing to be at increased risk of illness. From the levels found in the soil, we estimated a dose, the amount of copper a person might ingest. The level we estimated a child might ingest is slightly lower than the level that caused vomiting, nausea, respiratory edema, liver problems, and heart problems in two sensitive people. However, people who are sensitive or who are exposed to additional sources of copper may experience illness (ATSDR 1990a).

We do not expect any illness from incidentally swallowing copper-contaminated surface soil or breathing copper-contaminated dust from the playground or residential areas. A child's exposure dose from breathing copper-contaminated dust is at least 300 times lower than the level causing adverse effects in animals (ATSDR 1990a).

Copper has not been shown to cause cancer in humans by any exposure route (ATSDR 1990a).

### **B. Children's Health**

Before birth, children are forming the body organs that need to last a lifetime. Exposures during development may lead to serious injury or illness. Injury during certain periods of growth and development may lead to malformation of organs (teratogenesis), disruption of function, and premature death. Exposure of the mother leads to exposure of the fetus because some contaminants cross the placental barrier (ATSDR 1998c).

Small children may have greater exposures to environmental contaminants than adults. Pound for pound of body weight, children drink more water, eat more food, and breathe more air than adults. For example, children in the first 6 months of life drink 7 times as much water per pound as the average adult living in the United States. Children's exposure to contaminants in the environment is also greater because they put things into their mouths and play close to the

ground, increasing their exposure to contaminants in dust and soil. Their hands, toys, and other items may have soil and dust containing contaminants from paint, gasoline, vehicle emissions, and industrial sources. In addition, children may accidentally or deliberately enter restricted locations. The obvious implication for environmental health is that contaminants present much greater doses to children than to adults (ATSDR 1998c).

Lead-induced illness is a particular concern for children. Children less than 5 years old absorb lead into their body more efficiently than adults. They do not remove lead from their bodies as quickly as adults do. Children with nutritional deficiencies may absorb the lead even faster. A blood lead level of greater than 10  $\mu\text{g}/\text{dL}$  suggests that children may have swallowed or breathed higher than average levels of lead (ATSDR 1998b).

Preliminary findings from a study by the Morehouse School of Medicine show infants born to mothers with prenatal blood-lead levels between 2.5-10  $\mu\text{g}/\text{dL}$  have significant differences in motor skill development, muscle tone and hand-to-mouth activities. They have more tremors and defensive movements. Another study of Inner City Environmental Lead Exposure and Hypertension by the Charles R. Drew University of Medicine and Science in Los Angeles, California has preliminary found that lead may affect blood pressure during pregnancy (ATSDR 1999).

In addition to site-related contamination from lead, children may contact lead if they eat chips of lead-based paint or breathe lead-contaminated dust from home renovations. Lead can contaminate drinking water if the water distribution system has lead-soldered joints or connectors or other fixtures. Children may contact lead when parents take lead home on their clothing from work or from certain hobbies (such as furniture refinishing or making stained glass or pottery). Lead-soldered cans and improperly fired ceramicware can result in lead-contaminated foods (ATSDR 1998b).

An ATSDR study found exposure to lead as a child was correlated with central nervous and peripheral nervous symptoms as adults (19-20 years later). The symptoms included poor memory, difficulty reading and concentrating, depression, sleep disturbances and decreased function on neurobehavioral tests. These young adults also had difficulty conceiving children (ATSDR 1999).

### **C. Health Outcome Data Evaluation**

In 1995, the Duval CHD Childhood Lead Poisoning Prevention Program received a three-year grant award from the Centers for Disease Control and Prevention to conduct intensive clinic and field testing of children and extensive environmental

assessments. Grant activities also included educating parents and the community regarding the hazard of lead poisoning and precautionary measures for avoiding or minimizing exposure to lead (EMCON 1996).

In our 1997 health consultation report, we evaluated the blood lead data collected by the Duval CHD between May 24 and June 5, 1995. Out of a total of 194 area children, 8 (4.1%) had capillary blood lead levels greater than 10  $\mu\text{g}/\text{dL}$  (Frey 1996). More recent information reveals that 203 children were tested, with 8 having blood leads between 10-14  $\mu\text{g}/\text{dL}$  (Goldhagen 1997). Blood lead levels greater than 10  $\mu\text{g}/\text{dL}$  means children may have contacted higher than average levels of lead (ATSDR 1998b). The Duval CHD reported that the percentage of children in this area with blood lead levels greater than 10  $\mu\text{g}/\text{dL}$  (4%) was less than the county-wide percentage (9%; Frey 1996).

For our 1997 report, we obtained the addresses for 127 of the children screened, including the 8 children with blood lead levels above 10  $\mu\text{g}/\text{dL}$ . We superimposed a map of areas with high soil contamination from studies before 1997 on top of a map of the location of the residences of children with high blood leads. The location of residences of children with high blood lead levels was spread out over the neighborhood and not clustered in areas with high lead soil levels. These blood lead samples were collected from children less than 6 years of age. Children less than 6 years old are most sensitive to lead exposure and are more likely to exhibit the hand - mouth behaviors that increase the accidental ingestion of soil such as playing on the floor and mouthing toys. Further, the Duval CHD screened children in May and June. Children play outside during these months and are likely to contact the soil.

The Duval CHD has not conducted targeted screening of the Brown's Dump area since 1996. At that time, the percentage of children in the area with elevated blood lead levels was less than the countywide percentage. The Duval CHD began a door-to-door screening in June 1998 (Personal Communication, Hazel Brown, County Health Department, June 18, 1998).

The Centers for Disease Control and Prevention (CDC) does not consider children with blood lead levels less than 10  $\mu\text{g}/\text{dL}$  to be lead-poisoned. If a large portion of children have blood lead levels between 10 and 14  $\mu\text{g}/\text{dL}$ , the CDC recommends community childhood lead-poisoning prevention activities and more frequent screening (DHHS 1991). Blood lead levels actually measured in the 8 children living near the site (10-20  $\mu\text{g}/\text{dL}$ ) do not cause distinctive symptoms, but are associated with decreased intelligence and impaired neurobehavioral development. These blood lead levels can also cause decreased stature or growth, decreased hearing acuity, and decreased ability to maintain a steady posture. These blood lead levels could also interfere with vitamin D metabolism

(DHHS 1991).

While the blood lead testing did not find significant lead exposure, the results should not be used to decide that the lead contamination at the site is not a public health threat because:

- ▶ The body eliminates most of the lead in the blood in 4 to 5 months (half-life 28 - 36 days) (ATSDR 1998b). Therefore, blood measurements reflect only recent exposure, not long-term exposure.
- ▶ Following increased awareness due to publicity about the site, people may have modified their behavior and reduced their exposure such as washing children's hands after playing. If people reduced their exposure, their blood lead levels would decrease.
- ▶ The blood lead results cannot be used to decide that elevated soil lead levels are safe in the future. Blood lead levels below 10  $\mu\text{g/dL}$  do not prove that significant lead exposure did not occur in the past or may not occur in the future (EPA 1994b).

## D. Discussion of Community Health Concerns

In this section, we address each community health concern.

### What chemicals were found?

Lead levels found in studies prior to the EPA's 1998 Expanded Site Investigation Report are in Table 1. Chemicals found in the EPA's 1998 Expanded Site Investigation Report are in Tables 2-5.

### What is the effect of lead on plants in the Dodge/Nash area?

The data analyzed in this report is from the EPA's 1988 Expanded Site Investigation Report. Other data will be analyzed in future reports.

### Will anyone do more testing?

Currently the City of Jacksonville and the EPA are negotiating a plan to test more soil and water. The City is currently testing children's blood and residential soil upon request.

### What are the boundaries of contamination?

The extent of the Brown's Dump contamination is not well defined. The City and EPA are working together to find out the extent of contamination. Possible boundaries of the ash are shown in Figure 2.

### How were carcinogens addressed in assessment?

We assess carcinogens differently than non-carcinogens. Instead of looking for a level that will or will not cause illness, we assume that people may be harmed and look for the risk of harm. When assessing the amount of exposure to a carcinogen, we assume a worst case scenario. We assume people are exposed to a contaminant 350 days per year for 70 years. We use EPA's cancer potency value to estimate the risk of cancer. We use a worst case exposure scenario to obtain a risk value with a large margin of safety.

Using the highest levels of contaminants found in the soil and EPA's current cancer potency factors, we assessed the risk from arsenic, bis(2-ethyl hexyl phthalate), polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), toxicity equivalency to dioxin and dioxin-like compounds (TEQ), and dieldrin. Our best estimate of the risk of cancer from the maximum exposure to each of these contaminants could be as high as one additional case in one million people or as low as no additional cases. Since these risks are so small, we do not expect to see an increased risk of cancer from these contaminants.

There is no cancer potency factor to assess the cancer risk of lead. Scientists do

not have proof that lead causes cancer in humans. Rats and mice have developed kidney tumors when given large amounts of lead. Scientists have criticized the animal studies because of the large amounts of lead used in the studies, among other things, and they believe the results of these studies should not be used to predict the risk of lower levels of lead causing cancer in people (ATSDR 1998b).

Using the highest levels of carcinogens found in surface water, available data allow us to assess the risk of exposure to arsenic. Our estimate of a risk of cancer from arsenic in surface water could be as high as one additional case in ten million people or as low as no additional cases.

Using the highest levels of carcinogens in sediment, available data allow us to assess the risk from PAHs. We could not use an average value of PAHs in sediment because it was detected in one out of three samples. Our best estimate of a risk of cancer from PAHs in sediment could be as high as three additional cases in one-hundred-thousand people or as low as no additional cases. Since these risks are so small, we do not expect to see an increased risk of cancer in the population from these contaminants.

What about kids over 6 and adults?

The primary contaminant of concern at the site is lead. Lead, at low levels, is most harmful to children because it affects the nervous system. Children under six have nervous systems that are still developing. Lead can interfere with the development of their nervous system. Young children absorb more of the lead they ingest than do older children or adults. Children also have more hand-mouth activity than adults. This behavior can lead children to have higher exposures than adults. Therefore, if lead exposure is occurring, it will show up in children under 6 first. If we do not see illness or high blood lead levels in children under 6, then we assume older children and adults are also safe.

Twenty three of my neighbors have died. Is this related to the site?

Based on the levels of chemicals found at the site, we do not expect an increase in the death rate.

The statement that higher levels of chemicals are fenced off so that people cannot touch them is misleading, creating a false sense of security.

Chemicals in soil have the potential to cause illness but only if people contact those chemicals. A fence is a barrier that may prevent people from entering the contaminated area. A fence is only a short term solution, but it is one method that can quickly be put in place. Through the release of this report, we are trying to make people aware of the contamination so they can take precautions to

prevent exposure.

There are other hazardous substances found on the school property above health action levels (arsenic, PCB's, dioxin).

All compounds above our screening values in tables 2-5 are addressed in the Toxicological Evaluation section of this report.

Arsenic is above our comparison value (See tables 2-5). Therefore we evaluated this chemical further. We converted the levels found in soil to a dose, the amount a person would ingest from incidentally ingesting soil. To evaluate the threat from non-carcinogenic effects of arsenic, we estimated a 24 kilogram child would incidentally ingest 200 milligrams of soil per day, 350 days per year (we assume the child is away from home, for example, visiting relatives, for 15 days per year). The level a child would ingest was slightly higher than the minimal risk level (MRL). Since the MRL is based on the level that did not cause illness in people with an added margin as a safety factor, we researched the scientific literature for a level that did cause illness in people. Our estimated worst case scenario dose was 52 times lower than the level causing adverse effects in people. Therefore, we do not expect children (or adults) to become ill due to arsenic in the soil. We discussed the carcinogenic effects of arsenic in a previous question.

PCBs were above our comparison value. Therefore, we evaluated this chemical further. We converted the levels found in soil to a dose, the amount a person would ingest from incidentally ingesting soil. To evaluate the threat from non-carcinogenic effects of PCBs, we estimated a 24 kilogram child would incidentally ingest 200 milligrams of soil per day, 350 days per year. Our estimated worst case scenario dose a child (or adult) would receive is lower than the Minimal Risk Level (MRL). Therefore, we do not expect noncancer illness from PCBs.

For the carcinogenic effects of PCB's, we do not assess if the compound will cause illness or not, we assume people may be harmed and look for the risk of harm. Our estimate of the risk of cancer could be as high a one additional case in ten million people or as low as no additional cases. Since these risks are so small, we do not expect to see an increased risk of cancer from exposure to PCBs.

The level of 2,3,7,8-tetrachloro dibenzo-p-dioxin (TCDD) was lower than our comparison value. Therefore, we do not expect any illness from exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin.

Since people are generally exposed to mixtures of chlorinated dibenzo-p-dioxins and other dioxin-like compounds, scientists have a special way to assess the health threat to dioxins-like compounds. 2,3,7,8-tetrachlorodibenzo-p-dioxin

(TCDD) is the most toxic and most extensively studied compound. Scientists express the toxicity of other dioxin-like compounds as a fraction of the toxicity attributed to 2,3,7,8-TCDD. Each fraction is called a Toxic Equivalent Factor (TEF). The sum of the TEFs is the Toxicity Equivalence (TEQ).

The TEQ in soil at this site is above the screening value of 2,3,7,8-TCDD but below ATSDR's Action Level. If a chemical is above an action level, public health actions such as surveillance, research additional studies or community education are conducted but at this site. However, the TEQ was below the action level. Since the estimate was above our screening value, we estimated a child's maximum exposure dose from incidentally ingesting 200 mg of soil per day, 350 days per year. Our estimated dose is below ATSDR's intermediate duration MRL (for exposure less than a year). Our estimate of a child's chronic exposure dose (using the average levels in the soils) is also below ATSDR's chronic duration MRL (for exposures longer than a year). Since the estimated dose is below the MRL, we do not expect non-cancer illness from exposure to dioxin and dioxin-related compounds in soil.

The chronic duration MRL is approximately two orders of magnitude below noncancer *and* cancer health effect levels observed in recent studies (ATSDR 1998d). However, the EPA has not developed a cancer potency factor to estimate the risk of cancer from exposure to dioxins. We are not able to assess the risk of cancer from dioxins at this time.

There is no established safe level of lead exposure for children 6 and under. There does not appear to be a threshold, or safe level of lead exposure. This site is a potential health hazard because, although there is no evidence of children that have been exposed to lead, there is the potential of exposure due to the high levels of lead.

Residents say children travel through the open gate on school grounds. The fence surrounding the school grounds is different from the ER/RB fence (See Figure 3). The levels of lead on the school grounds was below FDEP's screening value of 400 ppm. If children play in the soil behind the ER/RB fence, we estimate they might be at an increased risk of illness due to the lead in the soil.

Why does FDOH say they do not think the site made people sick in the past? The amount of contact people have with a chemical is essential in determining if the chemicals might cause illness. When scientists collect samples of the chemicals in the soil, we estimate how much of that chemical gets into the body and if it can cause illness. To estimate how much chemical gets into the body, we calculate a dose, estimating how often and how long people contact the chemical. However, it is an estimate.

A better method of assessing if a compound can cause illness is to measure the amount in the body. Since measuring the amount of a compound in the body is often unpleasant (involving taking blood or fat samples), this kind of test is usually only done if scientists suspect people contacted the chemical. Doctors may suspect people could contact the chemicals if the levels in the soil are high and if there is a way people contacted the chemical, like playing in contaminated soil.

In 1995, the Duval CHD tested children under 6 in the area and attending the elementary school. They did not find any more lead in these children's blood than in children living in other areas of Jacksonville. This suggests that children are either not contacting the lead in the soil or that the lead is not bioavailable (does not move from the stomach or intestines into the blood stream). It might also suggest children washed their hands a lot during the months before the blood lead testing. An uncertainty with blood lead testing is that it only reflects exposure in the previous 3-4 months. Since the children did not have high blood lead levels in 1995, we do not think that the site made them ill in the past.

Why were the children not tested for other chemicals besides lead?

We estimated the amount of other chemicals that children would get in their bodies based on the levels of chemicals reported by the EPA 1998 Expanded Site Investigation. These levels were below levels likely to cause illness. Therefore, biological testing for other chemicals is not warranted.

Could the children whose blood leads were normal in 1995 have been exposed to lead before or after the 3-4 months of exposure and could they have lead in their brain, kidneys, liver or bones?

Children whose blood lead levels were not elevated during the testing in 1995 could have been exposed before or after the 1995 testing. Shortly after exposure, lead moves to soft tissues (like the brain, kidneys and liver) then moves to the bones. We define the site as a potential health hazard because we are uncertain if children were exposed before or after the 1995 testing.

Why does the report not mention the extent of the contamination in the residential area north of the CSX railroad tracks?

This report assessed the health threat using data in the EPA's 1998 Expanded Site Investigation report. Data collected since then will be addressed in a future report.

Why did the City place 6 inches of soil on playground?

The Florida Department of Environmental Protection found elevated lead levels, uncovered ash and glass fragments in the playground area. The city placed clean soil in the playground area as an interim measure to safeguard the children.

Why are the lead levels getting higher as time goes by?

Lead levels would not increase as time goes by. Scientists are testing more areas and have found higher levels in 1996. The highest level the EPA found in 1998 was 9,100 ppm.

What were the findings of the EPA 1998 sampling?

This PHA is based on the EPA sampling reported in the Expanded Site Investigation, so this report presents those findings. We reviewed previous sampling in a 1997 Health Consultation.

Why doesn't a concentration above the ATSDR comparison value represent a health threat?

Since there are typically voluminous quantities of environmental sampling data, ATSDR's published standard comparison (screening) values help us narrow the list of contaminants so we can concentrate on the ones that could potentially to cause health problems. The screening values are not used for the purpose of predicting health effects or taking public action nor to select clean-up levels.

These comparison (screening) values are based on doses that will not cause illness. They are derived to protect the most sensitive members of the population (e.g., children). They are not cut-off levels, but rather screening values. They guide us in selecting the chemicals with the possible health threats.

Contaminants with concentrations above an ATSDR standard comparison value do not necessarily represent a health threat, but are selected for further evaluation. To evaluate a contaminant further, we estimate the amount a person would take into their body from contacting the contaminant. We derive a dose estimate for the most sensitive members of the population, like children. We then compare our dose estimate to a health guideline, the Minimal Risk Level (MRL). MRLs estimate the level of contamination that a person could be exposed to at the maximum length of time and frequency of exposure without increasing the risk of noncancerous illness. MRLs are ten to one thousand times lower than the lowest levels causing the most sensitive endpoint (illness) in the most sensitive animal species study. MRLs also serve as screening values.

If an estimated dose is above the MRL, we evaluated it further in the Toxicological Evaluation section of the document.

Will more sampling give more information about the contaminants, for example aluminum?

More sampling will tell us if there is aluminum in the soil. More sampling will not tell us if a contaminant causes illness in the population. ATSDR does not have a screening level for aluminum because the scientific and medical literature could not identify reliable studies to develop screening levels. This means we retain the

chemical for further evaluation. We estimated the maximum dose of aluminum people would be exposed to and compared this with toxicological, medical and scientific studies. The dose of aluminum a person would receive, based on this data, was well below the levels causing adverse effects in animals.

Where are the N qualifiers in this report?

Qualifiers are ways the laboratory describes the confidence they have in the data. They sometimes use qualifiers called "N" qualifiers. This means the laboratory has statistical uncertainty in the identity of the compound. Most of the higher levels of lead had "N" qualifiers. Normally, we would not include "N" qualified data; however, in this case we did because previous studies showed similar levels of lead in the soil.

Why do we say there is not an increased risk of cancer to arsenic when we know arsenic causes cancer?

We address carcinogens differently than noncarcinogens. Instead of looking for a level that will or will not cause illness, we assume that people may be harmed and look for the risk of harm. When assessing the amount of exposure to a carcinogen, we assume a worst case scenario. We assume people are exposed to the contaminant 350 days per year for 70 years. We use a conservative exposure scenario that are the highest possible exposures to err on the side of safety. Our best estimate of a risk of cancer from arsenic in soil and surface water could be as high as one additional case in ten million or as low as no additional cases. Since these risks are so small, we concluded we would not expect to see an increased risk of cancer from these contaminants.

Why do we say that people have been exposed to copper but we do not expect people to be at an increased risk of illness from incidentally swallowing copper-contaminated soil?

The amount of chemical a person is exposed to is essential to determining the health threat. Many things are not harmful at low doses but cause illness at high doses. For example, one or two aspirin will make you feel better, but a whole bottle will make you sick. Even though copper is in the soil, the amount we estimate would enter your body is not high enough to make you sick.

Why do we say that swimming in Moncrief Creek is not a health hazard?

We estimated the amount of chemicals in the water that would enter your body through incidental ingestion. We estimated a child (the most sensitive population) weighing 24 kilograms swim in Moncrief Creek seven times per year for 2.6 hours each time (the national average for swimming in the US; USEPA 1989). We estimated the children accidentally drink 50 milliliters each hour they swim. The doses we calculated were below the Minimal Risk Levels or well below the levels reported in the scientific literature causing illness in people.

## Conclusions

1. The area inside the ER/RB fencing is a public health hazard because potential incidental ingestion of lead-contaminated surface soil inside the fence could cause illness. The fence around this area is currently intact. In the past, the fence has not been consistently maintained and access to the site has been allowed. Incidentally swallowing lead-contaminated surface soil could cause lead poisoning.
2. Currently, the surface soil in the school playground poses no apparent public health hazard. If, in the future, the playground surface soil erodes or is disturbed, the site could become a health hazard.
3. Based on limited data, the surface soil in the residential area poses a public health hazard. Even though the toxicological evaluation suggests that the dose of lead people would receive is higher than the dose that caused adverse effects, our dose estimate is overestimated to make sure sensitive people are protected. We base our estimates on the maximum amount of lead found in the residential area and assume a high percentage of the ingested lead is absorbed. There were, however, only a limited number of samples. The extent of surface soil contamination and levels in residential soil are not well defined. It is unlikely, however, that people would come into contact with that level of lead in the soil at the frequency and duration we assumed in developing the estimate. Results of the blood testing suggested children were not exposed to lead from the site. There was no apparent clustering, based on visual review of the data, between location of the houses where children with high blood lead levels resided in the spring and summer of 1995 and locations of highest lead contamination in the soil. Nonetheless, prudent public health policy dictates that we consider elevated lead concentration in surface soils as a public health threat.
4. Swimming in Moncrief Creek poses no apparent public health hazard. Even though the levels of contaminants in the creek were above comparison values, when we converted them to a dose, the amount a person would ingest, they were below levels causing illness. However, only four samples were taken and the extent of the contamination is not well defined.
5. Although groundwater is not a currently completed exposure pathway, the extent of groundwater contamination is not well defined.

### Recommendations

1. We recommend the continuation of efforts to ensure that nearby residents and school officials are aware of lead contamination behind the ER/RB fencing.
2. Access to the area inside the ER/RB fencing should be restricted to limit exposure to lead-contaminated surface soil. Fences around the site should be checked monthly and damages repaired promptly. Warning signs should be posted on the ER/RB fencing.
3. We recommend periodic sampling of the playground surface soil.
4. We recommend further sampling to determine the extent of the contamination in residential soil. Health education should be provided for nearby residents to learn ways to minimize exposures.
5. We recommend further sampling to determine the extent of contaminated soil, sediment, and surface water in Moncrief Creek.
6. We recommend further sampling to determine the extent of the groundwater contamination.

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## Appendix

Figure 1. Brown's Dump Site Jacksonville, Florida

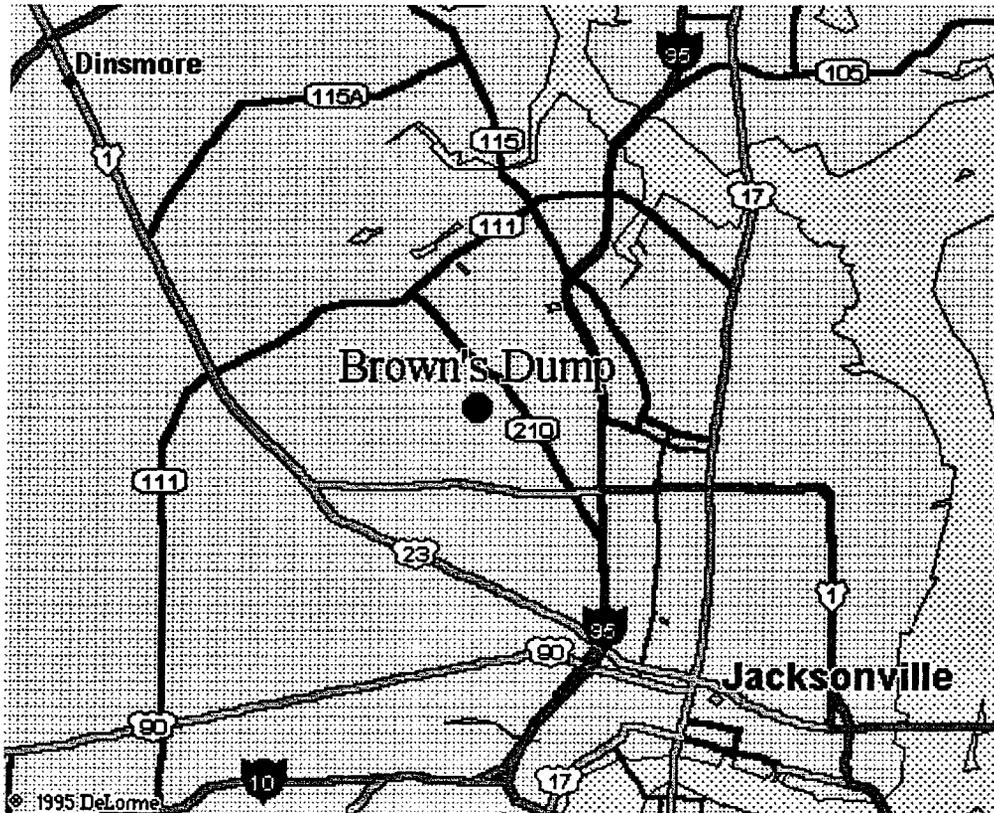
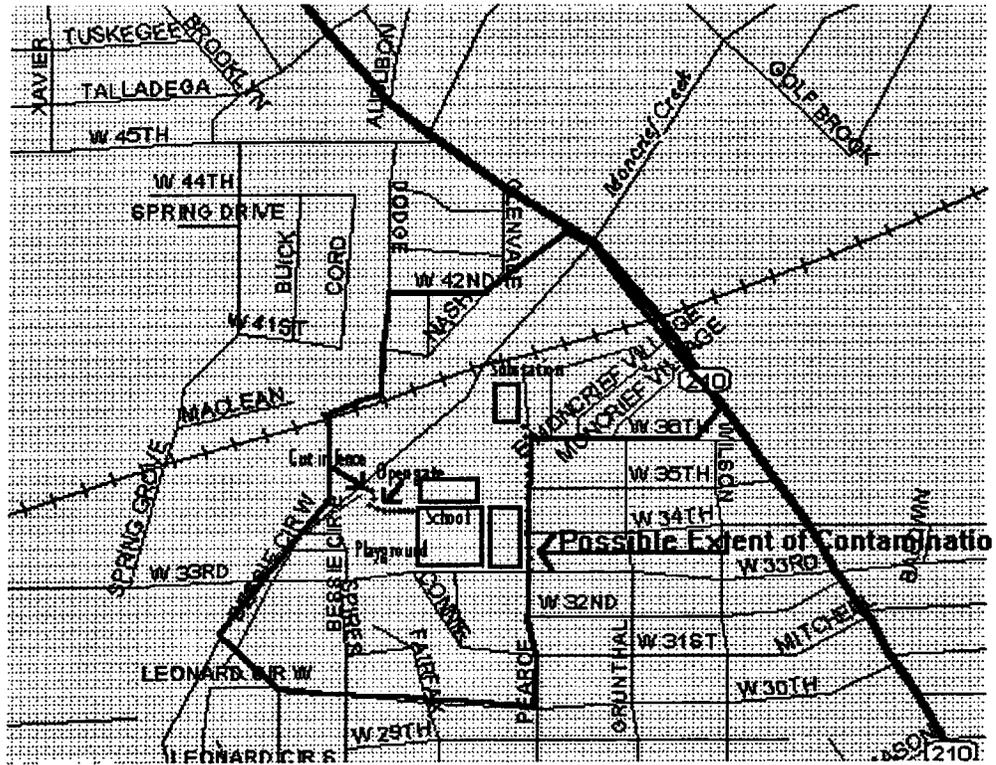
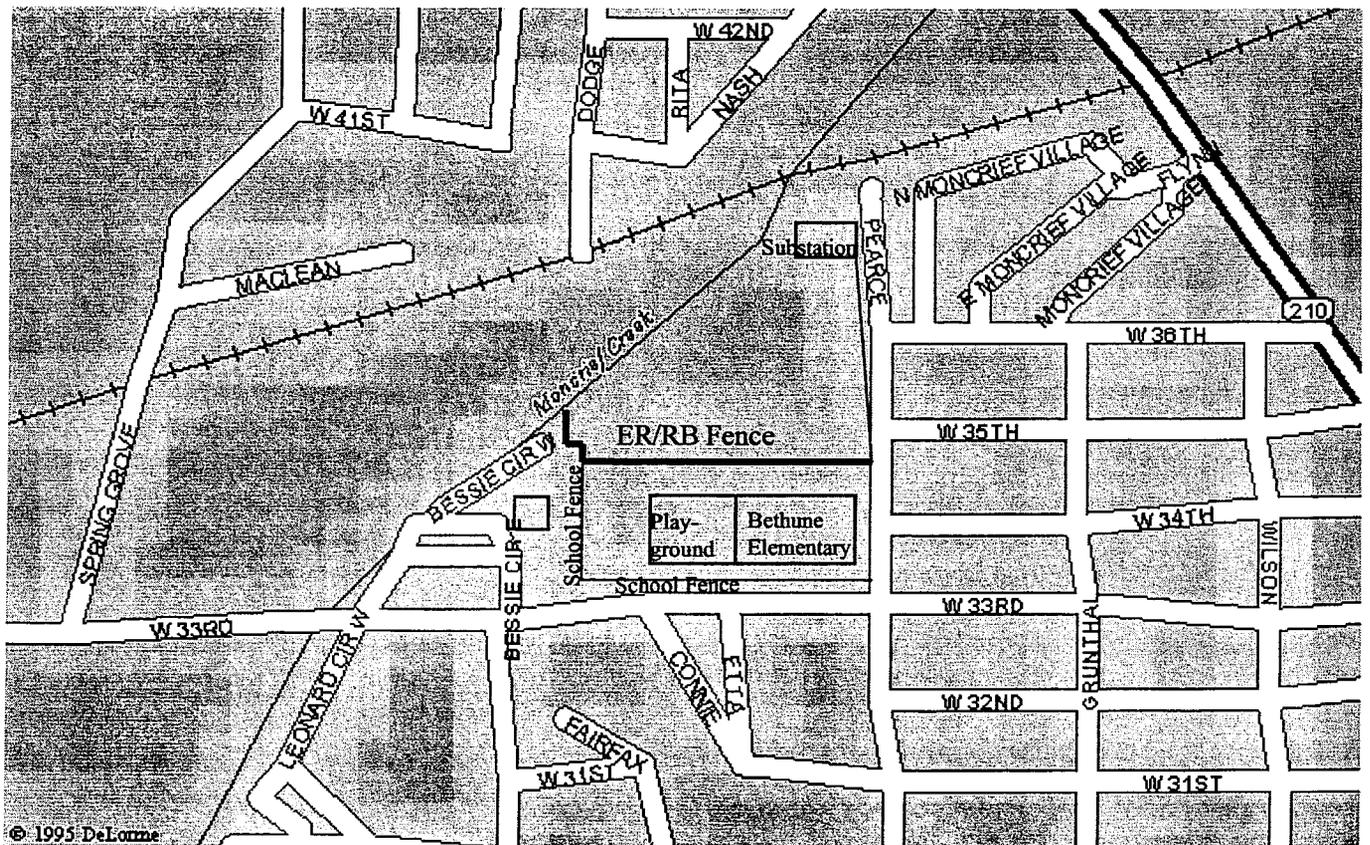


Figure 2. Brown's Dump Site Boundary: Possible Extent of Contamination



Source: FDOH, 1997

Figure 3. Brown's Dump Site



**Table 1**  
**Summary of previous soil lead concentrations evaluated in FDOH's 1997 health consultation**

<b>Date</b>	<b>Sampling Agency</b>	<b>Number of Samples</b>	<b>Maximum (ppm)</b>	<b>Percentage greater than 500 ppm*</b>	<b>EPA Recommendations (EPA 1994a)</b>
1/94-4/94	Duval Health Department: playground samples (Clark 1995)	4	910	50	Change use patterns and create barriers in all contaminated areas
2/95	Kiber: surface soil (Kiber 1995)	9	2,600	11	Change use patterns and create barriers in all contaminated areas
4/95	Duval Health Department (Arms 1995)	40	7,000	18	Reduce levels in soil when levels are above 5000 ppm
11/95	EMCON CAR: within the ash (EMCON 1995)	18	7,460	88	Reduce levels in soil when levels are above 5000 ppm
12/95	EMCON: within ash (Young, February 1996)	13	9,110	61	Reduce levels in soil when levels are above 5000 ppm
4/96	EMCON: within ash (Young, June 1996)	19	78,800	42	Reduce levels in soil when levels are above 5000 ppm

ppm = parts of lead per million parts of soil  
 \* 500 ppm = EPA health action level in 1997

**Table 2**  
**Maximum contaminant concentrations in surface soil (0-3" deep) above ATSDR screening levels of concern to the community (EPA 1997)**

Contaminant	Maximum Residences <sup>4</sup> (mg/kg)	Maximum Playground <sup>4</sup> (mg/kg)	Maximum behind ER/RB fencing <sup>4</sup> (mg/kg)	Screening Values (mg/kg)	Sources of Screening Values
Aluminum	3300	2100	5500	72000	FDEP
Antimony	32	3.3	21	20	RMEG child
Arsenic	11	5.1	35	20	EMEG child
Bis(2-ethylhexyl) phthalate	0.67	1.2	0.5	76	FDEP
Carbazol	0.05	0.81	0.11	53	FDEP
Cobalt	5	2.1	14	4700	FDEP
Copper	240	120	4100	110	FDEP
Dibenzofuran	-	0.32	-	280	FDEP
Dieldrin	0.009	0.005	0.059	0.04	CREG
2,3,7,8-Tetra-chlorodibenzo-p-dioxin (TCDD)	0.000007	-	-	0.00005	EMEG
Lead	1900	380	9100	400	FDEP
Magnesium	1100	220	4900	none	-
Mercury	0.41	0.22	5.6	3.4	FDEP
PAHs (Sum) <sup>2</sup>	5.2	29.6	9.4	0.1	CREG for B(a)p
PCB 1260	0.8	0.35	1.4	0.4	CREG
Silver	2.7	1.1	4.6	390	FDEP
TEQ (Reported in EPA 1998) <sup>3</sup>	0.00021	0.000017	0.00016	0.00005	EMEG for 2,3,7,8-TCDD

**Screening Values: ATSDR EMEGs (environmental media evaluation guidelines), RMEGs (reference dose media evaluation guide) and CREGs (Cancer risk evaluation guide for a one in a million excess cancer risk) are not site-specific and are not predictive of health effects. They are only used to select contaminants for further evaluation. They are based on levels unlikely to cause illness. They are derived to protect the most sensitive members of the population and are not cut-off levels but rather screening levels. Contaminants below screening values are unlikely to pose health threat and not evaluated further. Contaminants above the screening value are evaluated further by estimating a dose and comparing the dose to health guidelines.**

<sup>1</sup> Florida Department of Environmental Protection, soil cleanup target levels. For information only.

<sup>2</sup> The PAHs in soil include benzo(a)pyrene, benzo(b and/or k)fluoranthene, benzo(ghi)perylene, chrysene, phenanthrene, benzo(a)anthracene dibenzo(ah)anthracene, indeno(cd)pyrene, anthracene, fluoranthene, and pyrene. We used the screening value for benz(a)pyrene because screening values for other PAHs don't exist.

<sup>3</sup> TEQ: Toxicity Equivalent Value: Calculated by multiplying the exposure level of a particular dioxin-like compound by its toxicity equivalency factor. The TEQ includes dioxins, furans, and some dioxin-like PCBs.

<sup>4</sup> Reported in EPA 1998

Notes: The beryllium detection limit is greater than ATSDR's CREG, so there is no way to tell if beryllium is in the soil at a level greater than the screening value.

mg/kg: milligram contaminant per kilogram soil

- No data

**Table 3**  
**Maximum contaminant concentrations in Moncrief Creek sediment above ATSDR screening values**

Contaminant	Maximum <sup>2</sup> (mg/kg)	Screening Value (mg/kg)	Source of Screening Value
Aluminum	3300	72000	FDEP <sup>1</sup>
Mercury	0.62	3.4	FDEP
Carbazole	100	53	FDEP
Cobalt	4.1	4700	FDEP
Copper	190	110	FDEP
Lead	760	400	FDEP
PAHs (Sum)*	8.1	0.1	CREG B(a)p

Screening Values: ATSDR EMEGs (environmental media evaluation guidelines), RMEGs (reference dose media evaluation guide) and CREGs (Cancer risk evaluation guide for a one in a million excess cancer risk) are not site-specific and are not predictive of health effects. They are only used to select contaminants for further evaluation. Contaminants below screening values are unlikely to pose health threat and contaminant are not evaluated further. Contaminants above screening value are evaluated further.

<sup>1</sup> Florida Department of Environmental Protection, soil cleanup target levels. For information only. Screening levels used for sediments: ATSDR EMEGs, RMEGs, and CREGs

<sup>2</sup> Reported in EPA 1998

**Table 4**  
**Maximum contaminant concentrations in Moncrief Creek surface water above ATSDR screening values or of Concern to the Community**

Contaminant	Maximum <sup>3</sup> (mg/L)	Screening Value (mg/L <sup>2</sup> )	Source
Aluminum	0.07	0.013	FDEP <sup>1</sup>
Arsenic	0.012	0.003	EMEG Child
Barium	0.05	0.07	RMEG Child
Chromium	0.004	0.1	LTHA
Lead	0.004	none	none
Manganese	0.027	0.05	RMEG Child
Zinc	0.1	3.0	EMEG Child

Screening Values: ATSDR EMEGs (environmental media evaluation guidelines), RMEGs (reference dose media evaluation guide) and LTHA: Lifetime Health Advisory for drinking water (EPA) are not site-specific and are not predictive of health effects. They are only used to select contaminants for further evaluation. Contaminants below screening values are unlikely to pose health threat and contaminant are not evaluated further. Contaminants above screening value or of community concern are evaluated further.

<sup>1</sup> Florida Department of Environmental Protection, soil cleanup target levels. For information only. Screening levels used for surface water: ATSDR EMEGs, RMEGs, LTHA, and CREGs

<sup>2</sup>mg/L: milligrams contaminant per liter of water

<sup>3</sup> Reported in EPA 1997b

**Table 5**  
**Maximum contaminant concentrations in groundwater**

Contaminant	Maximum <sup>2</sup> (mg/L)	Screening Value (mg/L)	Source of Screening Value
Arsenic	0.02	0.01	EMEG
Barium	0.23	0.7	RMEG
Cadmium	0.005	0.007	EMEG
Cobalt	0.007	0.42	FDEP <sup>1</sup>
Copper	0.032	1.3	MCLG
Lead	0.073	0.015	FDEP
Manganese	2.1	0.05	RMEG
Nickel	0.019	0.2	RMEG
Zinc	0.91	3.0	EMEG

<sup>1</sup> Florida Department of Environmental Protection, groundwater cleanup target levels. For information only. Screening levels used for groundwater: ATSDR EMEG & RMEGs, CREGs and MCLG - Maximum Contaminant Level Goal

<sup>2</sup> Reported in EPA 1997b

**Table 6**

Pathway Name	Completed Exposure Pathway Elements					Time
	Source	Environmental Media	Point of Exposure	Route of Exposure	Exposed Population	
Residential soil	Brown's Dump	soil	surface soil	ingestion inhalation	3,903 children & adults	past, current
Playground soil	Brown's Dump	soil	surface soil	ingestion inhalation	3,903 children & adults	past, current
Moncrief Creek surface water	Brown's Dump	surface water	Moncrief creek	ingestion	3,903 children & adults	past, current
Moncrief Creek sediments	Brown's Dump	sediment	Moncrief creek	ingestion	3,903 children & adults	past, current

**CERTIFICATION**

The Brown's Dump Public Health Assessment was prepared by the Florida Department of Health, Bureau of Environmental Toxicology, under a cooperative agreement with the Agency for Toxic Substances and Disease Registry. It is in accordance with approved methodology and procedures existing at the time the health consultation was begun.



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



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Richard Gillig

Branch Chief, SPS, SSAB, DHAC, ATSDR