Public Health Assessment for

RSR CORPORATION
DALLAS, DALLAS COUNTY, TEXAS
CERCLIS NO. TXD079348397
AUGUST 16, 1995

U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

77458

77458

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.

Agency for Toxic Substances and Disease Registry.....David Satcher, M.D., Ph.D., Administrator
Barry L. Johnson, Ph.D., Assistant Administrator

Division of Health Assessment and Consultation.....Robert C. Williams, P.E., DEE, Director
Juan J. Reyes, Deputy Director

Exposure Investigations and Consultations Branch.....Edward J. Skowronski, Acting Chief

Federal Facilities Assessment Branch.....Sandra G. Isaacs, Acting Chief

Petitions Response Branch.....Cynthia M. Harris, Ph.D., Chief

Superfund Site Assessment Branch.....Sharon Williams-Fleetwood, Ph.D., Chief

Program Evaluation, Records, and Information Services Branch.....Max M. Howie, Jr., Chief

Use of trade names is for identification only and does not constitute endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

Additional copies of this report are available from:
National Technical Information Service, Springfield, Virginia
(703) 487-4650

PUBLIC HEALTH ASSESSMENT

RSR CORPORATION

DALLAS, DALLAS COUNTY, TEXAS

CERCLIS NO. TXD079348397

Prepared by

The Texas Department of Health
Under Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

FOREWORD

The Agency for Toxic Substances and Disease Registry, ATSDR, is an agency of the U.S. Public Health Service. It 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. (The legal definition of a health assessment is included on the inside front cover.) 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 cooperataive agreements.

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 then evaluate whether or not there will be any harmful effects from these exposures. The report focuses on public health, or the health impact on the community as a whole, rather than on individual risks. Again, ATSDR generally makes use of existing scientific information, which can include the results of medical, toxicologic and epidemiologic studies and the data collected in disease registries. 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 research studies are needed.

Conclusions: The report presents conclusions about the level of health threat, if any, posed by a site and recommends ways to stop or reduce exposure in its public health action plan. 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.

Interactive Process: The health assessment is an interactive process. ATSDR solicits and evaluates information from numerous city, state and federal agencies, the companies responsible for cleaning up the site, and the community. It then shares its conclusions with them. Agencies are asked to respond to an early version of the report to make sure that the data they have provided is accurate and current. When infomed of ATSDR's conclusions and recommendations, sometimes the agencies will begin to act on them before the final release of the report.

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.

TABLE OF CONTENTS

LIST OF TABLES ii

SUMMARY 1

BACKGROUND 4
- Site Description and History 4
- Site Visit 8
- Demographics, Land Use, and Natural Resources Use 10
- Health Outcome Data 12

COMMUNITY HEALTH CONCERNS 16

ENVIRONMENTAL CONTAMINATION AND OTHER HAZARDS 17
- On-site Contamination 18
- Off-site Contamination 25
- Quality Assurance and Quality Control 28
- Physical and Other Hazards 28

PATHWAYS ANALYSES 28
- Completed Exposure Pathways 29
- Potential Exposure Pathways 30

PUBLIC HEALTH IMPLICATIONS 31
- Toxicologic Evaluation 31
- Health Outcome Data Evaluation 41
- Community Health Concerns Evaluation 44

CONCLUSIONS 48

RECOMMENDATIONS 49

PREPARERS OF THE REPORT 51

CERTIFICATION 52

REFERENCES 53

APPENDICES 59
- Appendix A - Figures A-1
- Appendix B - Public Comments and Responses B-1

LIST OF TABLES

TABLE 1 - 1982 Blood Lead Levels of Preschool Children in the West Dallas Area Lead Assessment Study
TABLE 2 - Summary of Blood Lead Levels By Age Group City of Dallas Walk-in Clinics Between July 1992 and December 1993
TABLE 3 - Blood Lead Levels City of Dallas Walk-in Clinics Age 0 to 6.99 Years July 1992 - December 1993
TABLE 4 - Summary of Blood Lead Levels of Dallas Children Tested in EPSDT Program January 1993 - June 1993
TABLE 5 - West Dallas Childhood Lead Exposure Study, 1993
TABLE 6 - Health Assessment Comparison Values For Contaminants of Concern
TABLE 7 - Air Lead Values Measured by EPA Monitors April - July 1982
TABLE 8 - Contaminant Concentrations in Soil 1993 EPA Database
TABLE 9 - Contaminant Concentrations in Soil Samples Collected Where Battery Chips/Slag Were Observed
TABLE 10 - Follow-up Environmental Sampling in Homes of Individuals With Elevated Blood Lead Levels Identified at City of Dallas Walk-in Clinics, 1991 through 1993
TABLE 11 - In-home Environmental Sampling Data for Lead Content from 1993 Biological Indicators of Exposure Study
TABLE 12 - Chemical Releases to Air and Land Reported in Toxic Release Inventory
TABLE 13 - Exposure Pathway Elements
TABLE 14 - Comparison of 1982 and 1993 Blood Lead Data for Randomly-Selected West Dallas Children
TABLE 15 - Comparison of Recent Blood Lead Data from West Dallas and Other Areas of Dallas

SUMMARY

The RSR Corporation National Priority List (NPL) site, also known as West Dallas Lead, includes the facility formerly associated with the RSR Corporation Lead Smelter and the surrounding 13.6 square miles of residential, commercial, and industrial property of West Dallas, Dallas County, Texas that were contaminated with lead from RSR. This contamination resulted from historical air emissions from the RSR lead smelter, the use of battery chips and slag as residential fill, and the disposal of battery chips and slag in landfills and dumps.

The U.S. Environmental Protection Agency (EPA) is removing environmental contamination associated with the RSR smelter in five separate operable units, the first of which (Operable Unit 1) addresses the single-family residential properties that were contaminated by materials from the smelter. This report focuses on the current public health significance of Operable Unit 1. This area of the site formerly constituted a public health hazard since residents were exposed to significant levels of site contaminants in the past. More recent environmental sampling data and blood lead data indicate that residents currently are not being exposed to contaminants from the site at levels that would be expected to cause adverse health effects; therefore, the single-family residential area of the site currently constitutes no apparent public health hazard. As information about the remaining operable units -- the area with multi-family housing units, dumpsites/slag disposal areas, and the smelter facility site -- becomes available, the remaining public health implications of site contamination will be evaluated.

Air sampling data collected at the fenceline of RSR and in the residential areas near the smelter indicate that inhalation of contaminated air was a significant route of exposure in the past. Persons who were exposed to lead from the site in the past include residents and workers who inhaled contaminated air when the smelter was active and who ingested contaminated soil and dust prior to remedial activities. Exposures to lead-contaminated air were probably greatest prior to 1968 when air emissions from the smelter were largely uncontrolled. Due to the limited availability of ambient air data collected in the residential area when the smelter was active, we were unable to quantify past inhalation exposures. Air emissions from the smelter were reduced in the early 1980s and were eliminated as a source of contamination in 1984 when the smelter closed.

Environmental sampling data indicate that soil was contaminated with arsenic and cadmium in addition to lead. We were unable to determine from available data whether arsenic and cadmium were associated with battery chips, stack emissions, or both. If arsenic and cadmium were associated with stack emissions, past exposures to these contaminants could have occurred through inhalation as well as ingestion of contaminated soil and dust. Due to limited data, we were unable to quantitatively evaluate past or present exposure to these contaminants.

Pre-remedial soil lead data were unavailable in a format suitable for exposure quantification; however, in 1982 the average blood lead level of 227 randomly-selected children living near the RSR Smelter was 20.1 μg/dL, indicating that significant exposure to lead was occurring. The likelihood of human exposure to site contaminants was decreased through remedial

efforts in 1984 and 1985 that included removal of soil with lead levels above 1,000 ppm. The potential for human exposure was further reduced by clean-up activities initiated in 1991 to identify and remove soil in residential areas with lead concentrations greater than 500 ppm, arsenic concentrations greater than 20 ppm, and cadmium concentrations greater than 30 ppm.

In 1993, when this clean-up effort was nearing completion, the City of Dallas and the Agency for Toxic Substances and Disease Registry (ATSDR) conducted a lead exposure study in which the blood lead levels of randomly-selected West Dallas children were determined. The average blood lead level of all 305 children tested was 5.5 μg/dL. The average blood lead of the 53 children who lived nearest the smelter was 7.0 μg/dL. These data suggest that exposure to lead in West Dallas has decreased significantly since 1982. The study identified 26 West Dallas children (8.5% of the 305 children tested) with elevated blood lead levels ($\geq$ 10 μg/dL). Ten of the children with elevated blood lead levels lived in the area closest to the smelter (18.9% of the 53 children from this area who were tested). The study, however, did not find a relationship between blood lead concentrations and lead in residential soil and home dust.

Some areas within the site boundaries still have not been fully characterized; these areas include the unoccupied Dallas Housing Authority (DHA) property, slag piles/disposal areas, the smelter facility, and other RSR/Murmur property. Although trespassers could be exposed to contaminants, current human exposure in these areas is unlikely since public access is minimal. We were unable to evaluate the public health significance of these unremediated areas and the residential properties where owners refused access to EPA for remediation activities because environmental sampling data were not available.

Residents of West Dallas have raised several questions about the potential for adverse health effects among individuals who were exposed to high levels of lead as children, the health effects of exposure to lead on adults, and the safety of lead cleanup levels for soil. Detailed answers to these questions are contained in the Public Health Implications section of this document.

The Texas Department of Health (TDH) has recommended continuing measures initiated by the EPA and the City of Dallas to protect public health in West Dallas, including limiting public access to areas of the site which have not yet been remediated, further evaluating the sources of exposure among the 10 children identified in Area 1 with elevated blood lead levels, and providing ongoing blood lead testing for residents of West Dallas. TDH also has recommended evaluating the potential public health implications of the site as additional sampling data become available for review.

This report has been reviewed by ATSDR's Health Activities Review Panel (HARP) for determination of appropriate follow-up health activities. HARP recommended that TDH and ATSDR work with the City of Dallas Department of Health and Human Services to provide community education with specific attention to answering the community's health concerns. Although exposure to lead has occurred in the past, HARP did not recommend additional follow-up activities at this time because contaminants from the RSR site do not now pose a significant threat to health.

While the health assessment process deals with scientifically verifiable findings, all phases of the process involve a degree of uncertainty. The degree of uncertainty depends upon the completeness of site data, the accuracy of assumptions that simplify and approximate site conditions and site exposures, the completeness of toxicity data for the contaminants of concern, and the professional judgement used in developing and evaluating various parameters. Data gaps are bridged by using a variety of health protective assumptions. All assumptions that we used in developing this document weigh heavily toward the protection of public health.

BACKGROUND

In cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR), the Texas Department of Health (TDH) has evaluated the public health significance of this site. More specifically, ATSDR and TDH have determined whether adverse health effects can result from exposures to contaminants at this site and recommended actions to reduce or prevent possible health effects. ATSDR, a federal agency within the Department of Health and Human Services, is authorized by the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) to conduct health assessments at hazardous waste sites.

A.Site Description and History

The RSR NPL site, also known as West Dallas Lead, includes the former RSR (West Dallas) Lead Smelter and approximately 13.6 square miles of surrounding commercial, industrial, and residential property in West Dallas, Dallas County, Texas. The RSR site (also referred to as the study area) is bounded to the north by the Trinity River, to the east by Fort Worth Avenue, to the south by Davis Street, and to the west by Texas local highway Loop 12 (Walton Walker Boulevard). See Figure 1.

RSR was a secondary smelter and lead recovery facility that operated at the intersection of Singleton Boulevard and Westmoreland Road from 1936 until 1984. The facility was originally located just outside the Dallas city limits until 1952 when the city of Dallas annexed the area. In the subsequent 15 years, residences, schools, a Boys Club, and a major Dallas Housing Authority project were constructed nearby [1]. In 1971, RSR acquired Murph Metals, Inc., the previous owner of the smelter, and established it as an operating subsidiary. Murph Metals and its predecessors had operated the smelter continuously from 1936 until 1971 under the names Southern Lead and Southern Smelter [2].

Contamination at the RSR NPL site has resulted from historical air emissions from the RSR Lead Smelter, the use of battery chips (pieces of automotive battery casings generated from recycling operations at the smelter) and slag as residential fill, and the disposal of battery chips and slag in landfills and dumps. Contaminants associated with the site include lead, arsenic, and cadmium [3]. Lead has been identified in the soil on residential properties north and northeast of the smelter in a pattern suggesting that it was deposited through air emissions from the RSR smelter. Contaminants on residential properties throughout the community have been associated with battery chips and other materials from the smelter. According to residents, people used battery chips from the smelter as fill and paving material at a time when people had little awareness of the potential dangers that might be associated with these materials. These practices took place over a period of approximately 30 years, ending in the 1970s. The RSR properties and three landfills where RSR disposed of battery chips, slag, and smelter waste have not yet been fully characterized, but also contain site contaminants (Figure 2).

Principal activities associated with air pollution from the smelter included the preparation of scrap lead batteries for smelting, reverberatory and cupola smelting processes, hard and soft lead refining processes, alloying, and fabrication. According to available records, emissions from the smelter were uncontrolled prior to 1968 when a lead control ordinance was enacted by the City of Dallas. After RSR acquired the smelter in 1971, violations of the city ordinance began to occur almost immediately [1].

Numerous enforcement actions and compliance efforts were initiated in the early 1970s. In 1975, RSR upgraded the capture efficiency of emissions control devices under a State Court order. The smelter operated inconsistently throughout the late 1970s under the constraints of the control strategy imposed by the court. In 1979 and 1980, RSR completely revamped the smelter control system in preparation for enforcement of the United States Environmental Protection Agency's (EPA's) new national lead standard. In the early 1980s, however, the Texas Air Control Board [TACB, which is now a part of the Texas Natural Resource Conservation Commission (TNRCC)] found that the smelter was in violation of the national ambient air standard despite the recent implementation of controls. In the same period, blood screening of children indicated that excessive exposure to lead was occurring [4].

In May 1983, the City of Dallas and the State of Texas filed suit against RSR and Murph Metals, Inc. under the Texas Clean Air Act and city ordinances alleging damage from air emissions of lead. The 95th District Court ordered RSR to remove and replace contaminated soil on approximately forty acres of land within one-fourth mile of the smelter, to sod or plant grass on bare spots within one-half mile of the smelter, and to implement a comprehensive remedial plan. The order also required continued blood lead screening of children in the area and installation of adequate air pollution control equipment on the smelter stack [5].

In the same period, the EPA pursued an administrative order under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA or Superfund) and the Federal Trade Commission ordered that RSR divest itself of the Dallas smelter. In February 1984, RSR sold the facility to the Murmur Corporation and the court subsequently held Murmur liable for air pollution control costs [1].

Murmur assumed occupancy, began to install necessary controls, and ultimately received TACB approval to begin operations. In September 1984, however, the Dallas Board of Adjustment denied Murmur's application for an operating permit on the basis that the smelter was a non-conforming use of the land and that cessation of operations in February had terminated its "grandfathered" status. After legal appeals, Murmur was permanently enjoined from operating the smelter in February 1985. There have been no lead emissions from the RSR stack since February 24, 1984 when RSR ceased operation of the smelter [1].

Remedial work was initiated in 1984 under the direction of a court-appointed Special Master. A private contractor removed soil with lead levels of more than 1,000 parts per million (ppm) (a level considered protective of public health at that time) from residential areas,

public play areas, day care centers, and private gardens within a one-half mile radius of the smelter. Most soil removed from the area was taken to a dedicated landfill in southeast Dallas county, near Wilmer, Texas. The small amount of soil that exceeded the extraction procedure toxicity (EPT) standard was manifested to a commercial hazardous waste disposal site near Robstown, Texas. Soil was replaced with clean fill [1,6].

Areas in which contaminated soil was not removed, such as the former smelter facility site and uninhabited Dallas Housing Authority (DHA) buildings, were fenced to restrict public access. Groundcovers were planted and water credits were provided to residents to support sod watering and growth. Unpaved driveways and alleys were paved to reduce dust emissions. Remedial work was completed in 1985 [5,6].

A medical monitoring and public health program was initiated in 1984 by the City of Dallas. The program offered free blood lead screenings at six-month intervals for children and pregnant women living within one-half mile of the smelter [6]. The City continues to offer free-of-charge blood lead screening to all Dallas residents.

Problems reemerged at the site in July 1991 when the Texas Water Commission (TWC) [which is now part of the Texas Natural Resource Conservation Commission (TNRCC)] began receiving complaints from residents around the RSR Smelter. TWC soil samples showed elevated concentrations of lead, arsenic, and cadmium [3].

TWC requested that the EPA become involved at the site to review the activities of the 1980s. As of December 1991, most soil samples contained less than 250 ppm lead, but approximately 16 percent of samples contained levels between 500 and 1,000 ppm. The highest contaminant concentrations were found in areas of West Dallas where contaminated soil, slag, and battery chips had been improperly disposed or used as residential fill [7,8].

EPA began a second removal effort in West Dallas in 1991, which involved the cleanup of contaminated residential areas. EPA proposed the RSR Corporation site to the National Priorities List (NPL) of Superfund sites May 10, 1993. EPA has prioritized the West Dallas cleanup into five operable units which are being addressed separately (Figure 2) [2,9]. Operable Unit 1 consists of all single-family residential properties that were contaminated by materials from the RSR smelter facility. Contamination in this area resulted from air deposition from the smelter smoke stacks and the use of battery chips and slag as yard and driveway fill material. Removal efforts for Operable Unit 1 were divided into two phases.

During Phase 1, EPA excavated soil from 206 residential lots that were subject to air deposition. Work began in October 1991, but due to legal challenges regarding soil disposal arrangements, full-scale removal activities were delayed until June of 1992. Phase 1 work was completed in June of 1994. Phase 2 removal activities have focused on 194 residential properties where battery chips or slag have been identified or where soil lead levels are over 500 ppm, arsenic concentrations are over 20 ppm, or cadmium concentrations are over 30 ppm. Phase 2 removal was completed in June of 1994. Soils deemed non-hazardous under

federal law were trucked to the Republic Waste Industries facility in Avalon, Texas, while materials deemed hazardous were trucked to a hazardous waste disposal facility in Peoria, Illinois [8,9].

Operable Unit 2 includes the multi-family housing units owned by the Dallas Housing Authority. Most of the buildings in this area have been unoccupied in recent years, with the exception of buildings on the eastern portion of DHA property that were renovated in 1988 and 1989. Properties with the highest levels of contamination were fenced to restrict access. Contamination of this area is associated primarily with air deposition of lead emissions from the smelter. EPA and DHA formalized an agreement for DHA to conduct studies for this site and conduct a cleanup comparable to EPA's Phase 1 removal action. Contamination removal activities began in early 1995. Final remedial action decisions are currently being issued for both Operable Units 1 and 2.

Operable Unit 3 includes three properties/dump sites where slag and/or battery chips may have been disposed. The properties are in three locations on the southern and western perimeters of the site. EPA describes the properties as Sites 1, 2, and 3: Site 1 is in the 1000 block of North Westmoreland Avenue; Site 3, also referred to as the Walton Walker Property is near Loop 12 (Walton Walker Boulevard) and Singleton Boulevard; and Site 4 is at the end of Claibourne Street. Two of these properties were formerly City landfills. EPA began field activities and an investigation of these areas in October 1994 [2,8,9].

Operable Unit 4 refers to the secondary lead smelter facility at 2820 North Westmoreland Avenue. Operable Unit 5 refers to other properties south of Singleton Boulevard and west of Westmoreland Avenue that are associated with the smelter, including a disassembled battery wrecking facility, a closed solid waste surface impoundment, and other potential slag/battery chip disposal areas. In November 1993, the potentially responsible parties declined the opportunity to conduct site studies at these locations. EPA recently conducted field activities and Remedial Investigation/Feasibility Studies (RI/FSs) in Operable Units 4 and 5. The RI/FS reports were released for public review in the Spring of 1995.

A Biologic Indicators of Exposure Study was initiated in Fall 1993 by ATSDR and the City of Dallas, along with assistance from EPA, to determine the public health significance of the RSR Corporation Smelter facility. This study includes a random sampling of more than 500 homes in the West Dallas area and a northern Oak Cliff neighborhood, which serves as a baseline comparison area. EPA conducted a home sampling study in October and November 1993. The City of Dallas Health Department workers took blood samples from 386 children six years of age and under and administered questionnaires to identify other sources of exposure to lead [8]. Results of this lead exposure study indicate that few West Dallas children currently have elevated blood levels; 26 of 222 children tested in the study area, including 10 of 53 children in Area 1, had blood lead levels greater than or equal to 10 μg/dL. Additional information about this study is provided in this report and detailed analyses of the results is presented in a separate report by the participating agencies.

EPA has used the results of the West Dallas Lead Study as part of its risk assessment process to determine the necessary actions for making the residential areas of the RSR Corporation Site safe for long-term exposure.

B. Site Visit

TDH and ATSDR staff members visited the neighborhoods encompassed within the boundaries of the Superfund "site" numerous times. They attended community meetings and public availability sessions in the area on at least seven occasions. John Villanacci, Ph.D. and Kathryn Evans, M.P.H. of the Texas Department of Health, and Jennifer Lyke of ATSDR conducted a "site visit" on September 21, 1993 to examine the physical characteristics and layout of the area in a comprehensive manner and to learn more about EPA's remedial efforts. The site visit included a drive through the smelter facility and most of the surrounding neighborhoods and commercial areas, and a walk through one of the three slag disposal areas. The EPA Project Manager guided the site visit and was available to answer questions. Approximately two and one-half hours were spent on this tour.

The RSR Smelter is at the intersection of Singleton Boulevard and Westmoreland Road in West Dallas. The smelter is bordered to the south by the Missouri Pacific Railroad tracks, north by the Boys and Girls Club, north and northwest by the Lake West Housing Project (which is partially occupied), and further north and northwest by single-family residential housing.

The smelter was inaccessible to the general public. A brick fence topped with barbed wire surrounds the facility and there was a 24-hour guard at the entrance on Westmoreland Road. Buildings formerly associated with the smelter were being used to stockpile contaminated soil before transporting it to disposal facilities. EPA field offices were located in a mobile office on the smelter site.

Dilapidated facilities on the site of the former smelter may present physical hazards, but the area is well-restricted from the public to prevent accidents and injury. Other areas of the RSR Corporation site which have not yet been fully characterized may present similar hazards. We did not observe any apparent potential causes of avoidable physical hazard.

The area defined as the RSR NPL site surrounds the smelter and is used for commercial, industrial, and residential purposes. At its widest points, the site is approximately six miles from east to west and approximately three miles from north to south. The site's boundaries are roughly defined by Canada Drive on the north and east, Interstate 30 and Fort Worth Avenue on the south, and Texas local highway Loop 12 (Walton Walker Boulevard), the West Fork Trinity River, and mud flats on the west (Figure 2).

Numerous businesses and industrial sites are east and west of the smelter along Singleton Boulevard, including automobile repair shops, tire repair and sales shops, and GAF, a building materials manufacturing plant. Residences and several schools also are on Singleton Boulevard. There is a mixture of commercial, light industrial, and residential use along the

busier streets through the area. More densely populated residential neighborhoods are immediately northwest, north, and northeast of the smelter across Singleton Boulevard as well as southwest of the smelter off Singleton Boulevard and south of the smelter off Westmoreland Road.

The residences closest to the smelter are directly across Singleton Boulevard to the north. Many of the buildings in this area, which are part of the Lake West Housing Project, were unoccupied and boarded up; the yards surrounding most of the vacant buildings were enclosed with a six-foot chainlink fence. Apartment buildings north of Bickers Street were apparently occupied. These areas were included in the 1984/1985 remediation activities.

We drove through residential neighborhoods east of the smelter on both the north and south sides of Singleton Boulevard. These areas include primarily low socio-economic, single family dwellings. Many of the residents in these neighborhoods are Hispanic and African American.

Within the residential areas, we saw a church parking lot and several yards and driveways that were being remediated or that had been recently remediated. Yards had been seeded with grass and driveways covered with new gravel. We also saw two driveways in which battery chips had been used for fill or gravel that had not yet been remediated.

According to the EPA On-site Director, three "slag piles" where large quantities of slag material may have been dumped are located on the site: 1) at the end of Claibourne Road near a residential neighborhood on the northwest side of the site; 2) in an inaccessible area between the railroad track and Walton Walker Boulevard on the southwestern edge of the site; and 3) in an industrial area between Westmoreland Road and Cement Plant Road near the central southern edge of the site (Figure 2).

We walked around the slag pile at the end of Claibourne Road, off Singleton Boulevard. The area where slag had been dumped was fenced, but one side of the six-foot chainlink fence had been crushed, making the area easily accessible. A sign stating "Warning - contamination area, by TWC" was on the ground by the opening in the fence.

There was a dirt road within the fence leading across the dumping area to the Trinity River. The EPA Project Manager said that people have knocked the fence down in the past with four-wheel drive vehicles in order to gain access to the road through the site which leads to the Trinity River bottoms.

Pieces of slag were visible along the roadside, but most of the area within the fence was covered with tall grass. We saw several beer bottles and a few shotgun shells along the road within the fenced area, but no evidence that young children had been playing in the area. We saw no evidence of trespassing within the fenced area other than the trash near the road. This slag pile area is approximately two blocks from Jaycee/Zaragoza Park, which has well maintained playground facilities for children.

We drove by the slag pile near Westmoreland Road, south of the smelter. The immediate surrounding area was predominantly commercial and light industrial, and there was a mobile home/recreational vehicle park just south of the slag pile. The fence on the perimeter of the slag area along Westmoreland Road was approximately six feet tall and in good repair. The area was covered with dense vegetation and was apparently difficult to access; however, we were able to see only the perimeter of the slag pile area adjacent to Westmoreland Road. We did not see the slag pile area at the western edge of the site, south of Interstate 30. The project manager said that this area was less easily accessible than the other slag piles.

The EPA Project Manager explained that the site has been divided into five operable units so that clean-up activities can take place in order of priority. Current clean-up efforts began in 1991 and are part of Operable Unit 1. Operable Units 1 and 2 include residential areas off Singleton Boulevard immediately north, northeast, and northwest of the smelter. Operable Unit 3 includes the three slag piles near the southern and western perimeters of the site. Operable Units 4 and 5 include the smelter and additional land owned by RSR and the Murmur corporation. Clean-up efforts will address the remaining operable units when EPA completes the clean-up of residential and public use areas (Figure 2).

On September 22, 1993, Health Department staff met with EPA, ATSDR, the City of Dallas Department of Health and Human Services, and private contractors involved with the 1993 West Dallas Blood and Environmental Lead Study. This study was designed to assess the extent and impact of current lead contamination in West Dallas. Study participants would include children under age six in approximately 500 randomly-selected households in West Dallas and a comparison area in northern Oak Cliff neighborhood. A pilot study testing the questionnaire, blood lead sampling techniques, and environmental sampling techniques that would be used in the study had just been completed; results were discussed in the meeting. The group estimated that questionnaires would be administered and samples would be collected in Fall 1993 and that analyses would be completed in Winter and Spring 1994. (A report of this study was released by the participating agencies in November 1994 and is entitled "Biologic Indicators of Exposure to Lead, RSR Smelter Site, Dallas, Texas).

C. Demographics, Land Use, and Natural Resources Use

Demographics

According to 1990 census data, the total population of the census tracts in which the site lies (43, 101.01, 101.02, 102, 103, 104, 105, and 106) is 27,349 persons, living in 6,615 housing units [10]. Some of the residences in census tract 43 may not be within the site's boundaries, but all the other census tracts apparently match the site's boundaries exactly.

As of 1990, approximately 27 percent of the primary householders in West Dallas had lived in the same house since 1969 or earlier; 43 percent had lived in the same house since 1979 or earlier. In 1990, 55 percent of the population over age five had lived in the same house for five or more years. Approximately ten percent of the total population are younger than five years of age and approximately eight percent are older than 65 years of age.

The 1990 census reports that approximately 43 percent of the people in this area are African American, 18 percent are white, 1 percent are Asian, and 38 percent are of "other races." Approximately 52 percent of the total census population are of Hispanic origin. Fifty percent of people over age five reported speaking a language other than English at home; most of these individuals (97%) reported speaking Spanish at home. Twenty-five percent of the total population over five reported that they do not speak English "very well."

The unemployment rate for persons ages 16 years and older was 16.4%. The median family income ranged from $4,999 in census tract 103 to $19,886 in census tract 43. In West Dallas in 1989, the income of approximately 46 percent of families with children under five years of age was below the federally-defined poverty level. Approximately 29% of persons ages 25 and over graduated from high school and just less than one percent graduated from college.

Land Use

The RSR Corporation NPL site covers approximately 13.6 square miles of West Dallas. The predominant land uses within the boundaries of the site are residential, commercial, and light industrial. Most commercial and industrial activities occur along Singleton Boulevard, Westmoreland Road, and other major streets. Businesses include Lilly Industrial Coatings, Dallas Woodcraft, Inc., Blanks Engraving Company, Comet Steel Inc., GAF Building Materials, QDT Ltd. boiler plate manufacturers, a cement plant, and numerous tire repair and automotive repair shops [11]. The Boys and Girls Club is immediately north of the site. A church and a school lie east of the site. Two other schools, several churches, day care centers, and parks are located within the site's boundaries.

The most densely populated residential areas are in the central portion of the site north of Singleton Boulevard and at both the western and eastern ends of the site on the north side of I-30. Most of the residences are low-income, single-family dwellings. A large part of the residential area in the northern central portion of the site is owned by the Dallas Housing Authority (DHA) and includes numerous multi-unit buildings that make up the Lake West Housing Project. Many of these apartments, which are in the high lead deposition area have been unoccupied for several years and have been demolished recently as part of the operable Unit 2 removal actions.

The Missouri Pacific Railroad (Amtrak) crosses the site from east to west on the south side of Singleton Boulevard. The Atchison, Topeka & Santa Fe Railway runs from south to north through the central portion of the site and has several short extensions connecting it with the Missouri Pacific.

Natural Resource Use

The Woodbine, Paluxy, and Travis Peak aquifers underlie the West Dallas area. The Vulnerability Assessment Program of TNRCC's Water Utilities Division maintains a database

which includes city wells, community-use wells and some residential wells throughout Texas. This database documents three public water supply wells screened in the aquifers underlying the RSR site [12]. These wells, which belong to the City of Grand Prairie, the City of Irving, and the Gifford Hill American Company, are located just west and northwest of the study area. The wells range in depth from 2,001 to 2,163 feet. The TNRCC database does not include any residential wells in the West Dallas area, but lists approximately 25 additional wells within the study area that are owned by industries and the City of Dallas. These wells range in depth from 443 to 2,689 feet [13].

Since the 1950s, West Dallas has used a municipal water supply that is collected from surface water bodies outside the area. Most of the water supplied to West Dallas is drawn from lakes Grapevine and Lewisville [14].

The boundary of the site is formed by the Trinity River on the east, north, and part of the west side of West Dallas. This river contains water intermittently throughout the year, depending on rainfall. We were unable to determine whether the river is used for fishing. The climate of Dallas County is moderate to hot with average temperatures ranging from 36 to 95 degrees. The average monthly rainfall ranges from under two inches in July to almost five inches in May, with a yearly average rainfall of 35.9 inches [15].

The general surface water runoff flow pattern in the area is dominated by constructed features such as ditches, culverts, and drainageways. Surface water generally flows toward the Trinity River and other small bodies of surface water [16]. Fish Trap Lake is three-fourths of a mile northeast of the RSR Smelter facility and is the largest of approximately six small bodies of surface water contained within the site. The Trinity River eventually flows through Lake Livingston and into Galveston Bay.

Health Outcome Data

We obtained blood lead data collected in the West Dallas area by numerous agencies over the past twelve years. These data are from a variety of sources including the report of the 1982 West Dallas Area Lead Assessment Study, the City of Dallas Health Department Walk-in Clinic database, the TDH Early Periodic Screening, Detection, and Treatment (EPSDT) Program database, and the 1994 City of Dallas/ATSDR Biologic Indicators of Exposure to Lead Study. Although these data are best classified as exposure assessment information rather than health outcome data, the relationship between blood lead levels and potential adverse health outcomes is well documented. We are presenting these data here to provide a frame of reference for subsequent discussions of health outcomes. A complete discussion of these data and their relevance to the site will be presented in the Health Outcome Data Evaluation section of this report.

Report of the 1982 Dallas Area Lead Assessment Study

The earliest blood lead data we were able to obtain were reported for the 1982 Dallas Area Lead Assessment Study [17]. This study was conducted by the EPA, the CDC, and the City of Dallas to evaluate the extent and potential effect of lead contamination in West Dallas. The Lead Assessment Study provided blood lead testing of 496 randomly-selected preschool children living within one mile of the RSR smelter. The results were evaluated based on CDC's definition of blood lead toxicity at that time (blood lead level ≥ 30 μg/dL and erythrocyte protoporphryn (EP) ≥ 50 μg/dL); the number of children with blood lead levels greater than or equal to 10 μg/dL was not available in this report. Table 1 presents blood lead test results as they were available in the 1983 report.

TABLE 1 - 1982 Blood Lead Levels of Preschool Children in the West Dallas Area Lead Assessment Study [17]

Distance from RSR Smelter	Number of Children Tested	Mean Blood Lead Level	Approximate Percent of Children with Blood Lead Level ≥ 30 μg/dL and EP ≥ 50 μg/dL	Approximate Percent of Children with Blood Lead Level ≥ 30 μg/dL
< 1/2 mile	269	20.1 μg/dL	5 %	10 %
1/2 - 1 mile	227	15.8 μg/dL	0 %	1 %

EP = erythrocyte protoporphryn.

City of Dallas Walk-in Clinics

The City of Dallas Health Department established a medical monitoring program after the 1984 and 1985 remedial efforts in West Dallas. Voluntary blood lead screening has been offered to all Dallas county residents continuously since that time. Summary data were available for two periods of testing conducted between the years 1991 and 1993.

During the first period of testing (July through December of 1991). 110 West Dallas children under the age of six were tested in City of Dallas walk-in clinics. Thirty-three (30%) had elevated blood lead levels (≥10 μg/dL). During the same period, 52 children who lived in other areas of Dallas county were tested at walk-in clinics. Seven (13.5%) of these children had elevated blood lead levels [18].

During the second period of testing (July 1992 through December 1993), 4,475 individuals from different age groups were tested at City of Dallas walk-in clinics (Table 2) [19]. Among those tested, 1,138 were children ages six and under from the West Dallas area. The mean blood lead level of these West Dallas children was 6.2 μg/dL. During the same period, 330 children who lived in other areas of Dallas county were tested. The mean blood lead level of these children was 7.0 μg/dL. Among the West Dallas children, 158 (13.9%) had elevated blood lead levels (≥ 10 μg/dL) [20]. Among the children from other areas of Dallas county, 59 (17.9%) had elevated blood lead levels (Table 3).

Also during this period, 39 pregnant women were tested -- 19 from West Dallas and 20 from other parts of Dallas county. The mean blood lead level of the West Dallas women was 3.5 μg/dL (levels ranged from 1.0 μg/dL to 8.0 μg/dL). The mean blood lead level of pregnant women from other areas of Dallas county was 3.1 μg/dL (levels ranged from 1.0 μg/dL to 5.0 μg/dL) [20]

TABLE 2 - Summary of Blood Lead Levels By Age Group City of Dallas Walk-in Clinics Between July 1992 and December 1993 [19]

Age and Residence of Individuals Tested	Individuals	Mean (μg/dL)	Min (μg/dL)	Max (μg/dL)
Ages 0 - 6				
West Dallas Residents	1,138	6.2	0.5	28.0
Other Dallas Residents	330	7.0	0.0	32.0
Ages 7 - 12				
West Dallas Residents	444	4.9	0.5	29.0
Other Dallas Residents	178	4.6	1.0	14.0
Ages 13 and Above				
West Dallas Residents	965	4.4	0.0	37.0
Other Dallas Residents	1,367	4.6	0.0	26.0
Total - All Ages				
West Dallas Residents	2,969	5.2	0.0	29.0
Other Dallas Residents	1,506	5.0	0.0	37.0

* Totals include pregnant women and individuals whose ages were not provided; therefore, totals do not equal sum of tests listed by age.

TABLE 3 - Blood Lead Levels City of Dallas Walk-in Clinics Age 0 to 6.99 Years July 1992 - December 1993 [20]

Location of Residence	Elevated Tests		Range of Blood Lead Levels						# Tests
	(≥ 10)μ/dL		10-14 μ/dL		15-19 μ/dL		>20 μ/dL		Total
	#	%	#	%	#	%	#	%	#
West Dallas	158	13.88	126	11.07	24	2.11	8	.70	1,138
Dallas, Excluding RSR Area	59	17.88	37	11.21	12	3.64	10	3.03	330
Dallas, Total	217	14.78	163	11.10	36	2.45	18	1.23	1,468

Early Periodic Screening, Detection, and Treatment Program, January to June 1993

The Texas Department of Health maintains a database of blood lead testing data from the Early Periodic Screening, Detection, and Treatment (EPSDT) Program. The EPSDT Program enrolls Medicaid-eligible children for routine medical testing which includes periodic blood lead screening. Table 4 summarizes blood lead levels detected among Dallas children tested in the program between January and June of 1993 [21]. A small number of tests may have been duplicated for some children since children with elevated finger stick tests were referred for a repeat venous puncture sample. The results therefore are presented as the number of tests and not the number of children tested. Of the 1,126 tests run on children from the West Dallas area, 125 (11.1%) were elevated ($\geq$ 10 μg/dL). Of the 5,361 tests run on children from other areas of Dallas, 486 (9.1%) were elevated.

TABLE 4 - Summary of Blood Lead Levels of Dallas Children Tested in EPSDT Program January 1993 - June 1993 [21]

Residence of Child by Zip Code	Number of Tests[1]	Number of Blood Lead Levels $\geq$ 10 μg/dL	Percent of Blood Lead Levels $\geq$ 10 μg/dL
75208[2]	279	37	13.26%
75212	847	88	10.39%
West Dallas Total	1,126	125	11.10%
Dallas, Excluding West Dallas	5,361	486	9.07%
Dallas Total	6,487	611	9.42%

[1] Some of these tests are finger stick tests, rather than venous punctures, which may falsely register elevated blood leads due to contaminated samples.

[2] Majority of neighborhoods nearest RSR lie within this zip code.

The 1993 ATSDR/City of Dallas Biologic Indicators of Exposure to Lead Study

In 1993, ATSDR and the City of Dallas, with assistance from EPA, conducted a lead exposure study to determine the impact of lead from the RSR smelter facility on public health [22]. In August and September of 1993, the City of Dallas Health Department collected blood samples from 305 randomly-selected children in West Dallas and 81 children in the northern Oak Cliff neighborhood, which serves as a baseline comparison area for the study. The West Dallas study area was divided into subareas according to source and level of lead contamination in the area: Area 1 includes all homes in the area previously designated by the EPA as the high dispersion area; Areas 2 and 3 include the homes in the areas designated as the eastern and western low dispersion areas, respectively (Figure 3); and Area 4 includes homes located throughout West Dallas where battery chips or slag were found during the 1993 Texas Water Commission (now TNRCC) survey. The average blood lead level of all 305 children in the study area was 5.5 μg/dL; the average blood lead level of the 53 children who lived nearest the smelter was 7.0 μg/dL. The study identified a total of 26 West Dallas children (8.5% of the 305 children tested)

with elevated blood lead levels (≥ 10 μg/dL). Ten of the children with elevated blood lead levels lived Area 1, representing 18.9% of the 53 children from this area who were tested (Table 5).

TABLE 5 - West Dallas Childhood Lead Exposure Study, 1993 [22]

Area of Residence	Households	Individuals	Blood Lead Level (μg/dL)			Elevated Blood Lead Levels (≥10μg/dL)					
			Mean	Min	Max	10 to <15μg/dL		≥15μg/dL		Total ≥10μg/dL	
						#	%	#	%	#	%
Area 1	32	53	7.0	0.8	18.4	6	11.3	4	7.5	10	18.9
Area 2	67	79	5.7	1.5	14.8	9	11.4	0	0.0	9	11.4
Area 3	78	103	4.9	1.5	12.6	3	2.9	0	0.0	3	2.9
Area 4	45	70	5.1	2.2	13.8	4	5.7	0	0.0	4	5.7
Study Area Total	222	305	5.5	0.8	18.4	22	7.2	4	1.3	26	8.5
Comparison Area	60	81	5.4	0.7	19.0	3	3.7	1	1.2	4	4.9
Total	282	386	5.5	0.7	19.0	25	6.5	5	1.3	30	7.8

Area 1 - Includes all homes designated by the EPA as the high air dispersion area
Area 2 - Includes all homes in low air dispersion area east of Area 1.
Area 3 - Includes all homes in low air dispersion area west of Area 1.
Area 4 - Includes all homes in West Dallas where battery chips and/or slag were found during TWC survey.
Comparison area - The North Oak Cliff area, consisting of all homes in census tracts 48.0 and 54.0.

COMMUNITY HEALTH CONCERNS

We contacted the Community Outreach Officer (a contractor hired by EPA to serve as a community liaison), the EPA Community Relations Coordinator, and the City of Dallas Health Department, and talked with individual residents to determine current community health concerns. We also attended two EPA-sponsored public availability sessions at the West Dallas Multi-Purpose Center, reviewed the October 1993 Resolution by members of the TNRCC's West Dallas Citizen's Advisory Committee, and reviewed newspaper coverage about the site. The following health-related concerns were identified:

1. Why is so much attention still focused on children?

2. Should we be concerned about the residents who grew up in West Dallas and were exposed to lead all of their lives? Are they likely to develop illnesses resulting from their exposure to lead?

3. Should individuals with high blood lead levels be tracked for follow-up treatment?

4. Can residents be provided with free medical help for pollution-related illnesses?

5. Could exposure to lead in the environment during pregnancy cause learning disabilities and hearing problems for the unborn child?

6. Is EPA's 500 ppm clean-up level adequate to protect public health?

ENVIRONMENTAL CONTAMINATION AND OTHER HAZARDS

This section lists contaminants of concern at the RSR Corporation site. These contaminants were selected by comparing contaminant concentrations to health assessment comparison (HAC) values for non-carcinogenic endpoints and carcinogenic endpoints. HAC values are media specific contaminant concentrations that are used to screen contaminants for further evaluation.

The list below summarizes acronyms and symbols used to discuss contaminants of concern:

* HAC = Health Assessment Comparison Value
* MRL = Minimal Risk Level
* CREG = Carcinogenic Risk Evaluation Guide
* RfD = Reference Dose
* EMEG = Environmental Media Evaluation Guide
* ppm = parts per million
* mg/Kg = milligrams per kilogram (equal to ppm)
* $\mu g/m^3$ = micrograms per cubic meter of air
* $\mu g/dL$ = micrograms per deciliter

HAC values include non-cancer comparison values and carcinogenic risk evaluation guides (CREGs). Non-cancer comparison values are HAC values based on the Agency for Toxic Substances and Disease Registry's (ATSDR's) minimal risk levels (MRLs), EPA's reference doses (RfDs), or other non-carcinogenic health-based values. CREGs are HAC values based on EPA's chemical specific cancer slope factors and an estimated excess lifetime cancer risk of one-in-one-million persons.

Exceeding a HAC value does not imply that a contaminant represents a public health threat but suggests that the contaminant warrants further consideration. Subsequent sections of this assessment will evaluate whether exposure to the contaminants at this site has public health significance.

Although the data presented in this section were collected from areas outside the boundaries of the RSR Smelter facility site, for the purposes of this health assessment the term "on-site" refers to all areas within the defined 13.6 square-mile NPL site.

Sampling and analyses conducted at the RSR Corporation site primarily have evaluated the presence of lead, cadmium, and arsenic in soil. HAC values for these contaminants in soil are listed in Table 6.

TABLE 6 - Health Assessment Comparison Values For Contaminants of Concern in Soil

Contaminant	HAC Value (ppm)		Non-cancer HAC Value Source
	Non-cancer	CREG[1]	
Lead	500	NA[2]	EPA/CDC
Cadmium	40	NA	$EMEG_{Child}$
Arsenic	20	0.4	$EMEG_{Child}$

[1] CREG value is based on EPA's chemical specific cancer slope factor.
[2] NA = Not available.
[3] EMEG = Environmental media evaluation guide; based on ATSDR's chronic oral minimal risk level (MRL) of 0.0007 mg/kg/day for cadmium and 0.0003 mg/kg/day for arsenic.

A. On-site Contamination

Air

Historical ambient air monitoring data in the residential areas near the smelter are sparse. A well-documented history of the smelter's operations, however, indicates that the quantity of lead emitted from the smelter was highest prior to 1968, when emissions were largely uncontrolled. Results of fenceline air monitoring by the City of Dallas indicate that emissions continued to be quite high throughout the 1970s, even after primary point source controls and initial sanitary emission controls had been implemented [4].

A special enforcement study was conducted during a 30-day period in March 1973 by the City in preparation for its 1974 lawsuit against the smelter. This study, which was implemented after the smelter had installed initial emission controls, recorded that the downwind lead concentration at the fenceline of RSR averaged 44.6 $\mu g/m^3$ and the upwind fenceline concentration averaged 20.9 $\mu g/m^3$. (According to descriptions of the sampling locations, the "upwind" measurement actually was collected between portions of the smelter complex and therefore reflects contaminant levels associated with the smelter). The City of Dallas standard for lead in air was 5 $\mu g/m^3$ at that time [4].

The first adequate monitoring facilities were placed in the community near the smelter in 1982 when the EPA installed three 24-hour ambient air monitors. The results of air

monitoring for the second quarter of 1982 are presented in Table 7 below. This monitoring was conducted prior to the closure of the RSR smelter facility and after the smelter had implemented the most comprehensive emission controls in its history of operation. Two of the monitors measured lead levels in the air above 1.5 $\mu g/m^3$, the National Ambient Air Quality Standard (NAAQS) for lead. These monitors were located 50 and 500 yard north of the smelter. Wind direction at the time of monitoring was not provided, but the prevailing wind direction at this site is north-northeast [4].

Since the smelter's closure in 1984, air samples taken before, during, and after remedial activities have never detected levels of lead above NAAQS for high volume sampling (1.5 $\mu g/m^3$) [23].

TABLE 7 - Air Lead Values Measured by EPA Monitors April - July 1982 [4,17]

Monitor/ Distance from Smelter	Monitor A 50 yards North	Monitor B 500 yards North	Monitor C 150 yards Southwest	Monitor D 1 mile East	NAAQS
Value	2.97 $\mu g/m^3$	1.57 $\mu g/m^3$	1.13 $\mu g/m^3$	0.26 $\mu g/m^3$	1.50 $\mu g/m^3$

NAAQS = National Ambient AIr Quality Standard.

Surface Soil

Soil samples have been collected during various periods over the past 12 years. We reviewed samples collected before, during, and after both soil removal actions which were initiated in 1984 and in 1993. A summary of data associated with these removal efforts are presented below in chronological order.

The first soil samples were collected at this site in 1982 as part of the Dallas Area Lead Assessment Study. Sampling included soil grid sampling (750-feet grids) within a one-mile radius of the RSR Smelter. Analysis of the grid sampling produced isopleths (lines on the map showing the pattern of lead contamination in soil) consistent with the predominant wind direction from the RSR smelter. The isopleths are plotted on the map in Figure 4. The pattern of soil lead concentrations observed surrounding the smelter reflects the pattern of lead deposited from the smelter through the air. Lead concentrations in soil were highest near the RSR Smelter facility (up to 3,000 ppm) and decreased with increasing distance from the smelter (200 ppm at approximately one-half mile). The mean lead concentration of soil in residential yards within one-half mile of the smelter was 826 ppm; the mean soil lead concentration in yards located between one-half mile and one mile of the smelter was 192 ppm. In 1982, the overall mean soil lead concentration of all yards sampled within one mile of the smelter was 489 ppm [17]. Lead levels were higher north and northeast of the smelter, the direction of prevailing winds [6].

In 1984 and 1985, soil with lead levels of more than 1,000 ppm was removed from high use areas within one-half mile of the smelter. Soil was removed to a depth of six inches, replaced with seven inches of clean fill, and resodded. In public play areas and day care centers, contaminated soil was removed to a depth of 12 inches and replaced with washed sand. In gardens within a one-half mile radius of the smelter, 12 inches of soil were removed and replaced with clean fill. A vegetative barrier was established in less contaminated areas within a one-half mile radius of the smelter where soil was not removed [1,6]. Soil sampling data characterizing the site after removal were not available for review.

In 1991, TWC analyzed battery chips, slag material, and contaminated soil discovered by West Dallas residents. The chips and slag material contained lead levels as high as 64,000 ppm, arsenic in excess of 2,000 ppm, and cadmium above 100 ppm [24]. Between August and December 1991, EPA collected and analyzed approximately 1,200 additional soil samples. The majority of these samples contained less than 250 ppm of lead; lead levels ranged from levels below the detection limit up to 128,000 ppm. Approximately 16 percent of the samples contained 500 to 1,500 ppm of lead [7].

In 1992 and 1993, in an attempt to identify all properties containing battery chips and slag, TWC surveyed more than 6,800 properties within the 13.6 square-mile study area. Battery chips were identified on approximately 366 properties, including 282 residential properties, 53 vacant lots, 17 commercial properties, and 14 residential properties where the owner declined to sign a survey form [25].

Table 8 is a compilation of the results of soil sampling conducted by EPA from August 1991 through April 1993 and by TWC from September 8 through October 28, 1992 and February 23 through March 16, 1993. Sample concentrations represent soil contamination resulting from air deposition as well as the use of contaminated soil, slag, and battery chips as fill [26]. Only a small percentage of samples were highly elevated and many were below detection limits, as evidenced by means which are low compared to the maximum concentration levels. For arsenic, the highest detected concentration was six times higher than the next highest measured concentration.

TABLE 8 - Contaminant Concentrations in Soil 1993 EPA Database[1] [26]

Contaminant	Number of Samples	Range[2] (ppm)	Detection Limits (ppm)	Mean[3] (ppm)
Lead	2,323	BDL-128,000	6 and 7	740.18
Cadmium	2,261	BDL-200	2	4.71
Arsenic	2,275	BDL-2,880	6 and 7	14.62

[1] Compilation of data from pre-excavation soil analysis performed by EPA from August 1991 through April 1993 and the Neighborhood Survey and Sampling performed by TWC from September 8 through October 28, 1992 and February 23 through March 16, 1993.

[2] BDL = Below Detection Limit.

[3] Samples containing contaminants below detection limits were included in the calculation of mean by using the value of the larger detection limit.

Between June 1992 and June 1993, approximately 206 properties with soil lead levels greater than 500 ppm were remediated. This removal action included properties within the high air deposition area and all properties identified as high risk areas for children. In June 1993, EPA began to remediate an additional 194 properties where battery chips and slag had been identified. Soil with lead levels over 500 ppm, arsenic levels over 20 ppm, and cadmium levels over 30 ppm was removed and replaced with clean fill [27].

Sampling collected during removal efforts indicated that lead exceeded action levels on most properties, but that arsenic was detected above its action level on only a few lots. All properties with elevated arsenic levels also had elevated lead levels; therefore, arsenic was removed during the excavation of soil to address elevated lead levels. A cadmium level above the 30 ppm action level was found on only two properties where elevated lead was not also found [26,28].

Table 9 is based on a subset of samples included in the compilation of data presented in Table 8. Table 9 includes results of 663 samples collected by an EPA contractor between September 1, 1992 and April 30, 1993 [29]. These samples were collected where surveyors observed signs of battery chips and/or slag and in the yards of residents who requested sampling. This table reports the range of lead, arsenic, and cadmium concentrations observed in the samples collected.

ATSDR provided a health consultation to EPA for one West Dallas property where cadmium levels were found in concentrations ranging from 2.0 to 57.5 ppm [30].

Surface Water and Sediment

The City of Dallas found no lead contamination in two surface water samples from Fish Trap Lake, which is three-fourths of a mile northeast of the RSR Smelter [14,18]. According to a 1992 ATSDR Public Health Consultation, a West Dallas resident stated that samples he had taken from Fish Trap Lake have shown elevated lead levels, but the citizen's data were not available for review. Additional surface water and sediment sampling data were not available for this site.

TABLE 9 - Contaminant Concentrations in Soil Samples Collected Where Battery Chips/Slag Were Observed[1] [29]			
Contaminant	Concentration Range (ppm)	Number of Samples Within Concentration Range	Percentage of Samples Within Concentration Range
Lead	0-249	456	68.8%
	250-499	87	13.1%
	500-749	31	4.7%
	750-999	14	2.1%
	1,000-1,499	18	2.7%
	1,500-1,999	8	1.2%
	2,000-5,000	18	2.7%
	5,000+	31	4.7%
Arsenic	0-2.49	32	4.8%
	2.5-4.9	213	32.1%
	5.0-7.49	227	34.2%
	7.5-9.9	74	11.2%
	10-14.9	74	11.2%
	15-19.9	19	2.9%
	20+	24	3.6%
Cadmium	0-0.99	476	71.8%
	1.0-1.9	108	16.3%
	2.0-4.9	51	7.7%
	5.0-9.9	14	2.1%
	10.0-29.9	10	1.5%
	30+	4	0.6%

[1] Samples Collected between September 1, 1992 and April 30, 1993 by EPA Contract Laboratory in areas with visible contamination and where residents requested sampling.

In-Home Environmental Sampling

Walk-in Clinic Follow-up Data

In-home environmental sampling, including soil, dust, and water sampling, is offered to all individuals with elevated blood lead levels identified at City of Dallas Walk-in Clinics (children with $\geq$ 15 μg/dL and adults with levels $\geq$ 25 μg/dL). We reviewed data collected between 1991 and 1993 when 21 such follow-ups were conducted. There is no consistent relationship between any of the measured parameters and observed blood lead levels; however, paint, which is commonly the most significant source of childhood residential lead exposure, was tested for lead in only three of the homes. Data are presented in Table 10.

Biologic Indicators of Exposure Study Data

The most recent in-home sampling conducted in West Dallas was collected as part of the 1993 Biological Indicators of Exposure Study. This study provided more comprehensive environmental data than the Walk-in Clinic data and included soil, dust, water, and indoor and outdoor paint sampling at the homes of the 305 West Dallas study participants. Environmental samples also were collected in the homes of 81 children from a comparison community. A summary of the environmental sampling results are provided in Table 11.

As part of this study, 374 composite soil samples were collected from yards. The average concentration of lead in soil ranged from 70 ppm (in the "eastern low air dispersion area") up to 148 ppm (in the comparison community).

In West Dallas, 576 dust samples were collected from children's bedrooms and the entryways of homes; in the comparison community, 323 dust samples were collected from children's bedrooms and the entryways of homes. The average concentration of lead in dust samples collected in children's bedrooms ranged from 80 ppm (in the area of West Dallas where chips and slag had been identified) up to 146 ppm (in the area nearest the site). The average concentration of lead in dust collected from the entryways of homes ranged from 81 ppm (in the chips and slag area) to 205 ppm (in the comparison community).

Approximately 386 indoor paint samples were collected. Approximately 9.6% of homes had contaminated paint on at least one indoor surface and the percentage of homes with contaminated paint ranged from 5.7% (in the homes where battery chips were used as yard fill) up to 16.1% (in the comparison community). Exterior paint samples also were collected at 386 homes; 35.5% had lead-contaminated paint on exterior surfaces, with a range from 19.4% (in the western low air dispersion area) up to 45.7% (in the comparison area).

The mean lead concentration from the 382 first-drawn cold water samples collected from the kitchen faucets of homes in the study was 3.5 mg/L. The lead concentrations in water ranged from 2.71 mg/L (in the high air dispersion area) up to 3.73 mg/L (in the eastern low air dispersion area).

All of the environmental sampling results were compared with the blood lead levels of children living in the homes. The study did not find a statistically significant relationship between blood lead levels and any of the factors that were analyzed.

TABLE 10 - Follow-up Environmental Sampling in Homes of Individuals With Elevated Blood Lead Levels Identified at City of Dallas Walk-in Clinics, 1991 through 1993 [31]

Date Sampled	Year Born	Blood Lead Level (µg/dL)	Soil in Yard (mg/kg)		Dust (µg/sq ft)		Paint Chips (mg/kg)		Water (mg/L)
			Front	Back	Floor	Window Sill	Indoor	Outdoor	Tap
			>500[1]		>200	>500	>5,000		>0.015
08-26-91	86	15	39	51	10	<MDL	NS	NS	<MDL
09-18-91	89	21	253	95	NS	<MDL	NS	NS	<MDL
09-18-91	89	21	253	95	NS	<MDL	NS	NS	<MDL
09-18-91	84	21	253	95	<MDL	NS	NS	NS	<MDL
10-10-91	89	16	37	34	<MDL	NS	NS	NS	0.005
11-04-91	88	24	88	NS	10	10	NS	NS	0.006
11-07-91	91	15	22	91	<MDL	<MDL	304	NS	<MDL
11-07-91	62	28	54	121	<MDL	**3,800**	3	NS	0.003
02-26-92	90	15	44	24	<MDL	<MDL	NS	NS	0.005
02-26-92	88	19	44	24	<MDL	<MDL	NS	NS	0.005
08-17-92	91	15	53	60	<MDL	1.5	NS	NS	0.005
09-16-92	89	19	49	19	7	12	NS	NS	0.005
09-16-92	86	27	60	40	9	8	NS	NS	0.005
04-23-93	92	16	75	160	12	112	NS	NS	0.005
04-30-93	93	20	385	NS	321	NS	NS	NS	NS
06-25-93	92	15	78	NS	9	42	NS	**17,900**	<MDL
08-12-93	90	17	50	37	47	64	NS	NS	<0.005
08-16-93	90	19	44	31	16	31	NS	NS	<0.005
09-02-93	89	25	143	343	<4	20.4	NS	NS	NS
09-02-93	92	16	88.8	62.3	106	51.6	NS	NS	NS
09-02-93	92	18	40	102	30	14.8	NS	NS	NS

[1] Numbers in shaded row indicate general guides of comparison used by the City of Dallas to identify levels of contamination that may be a cause for concern. Bolded numbers indicate sample results exceeding these guides.
<MDL = less than the minimum detection limit.
NS = not sampled.

TABLE 11 - In-home Environmental Sampling Data for Lead Content from 1993 Biological Indicators of Exposure Study [22]						
Type of Sample	Area 1	Area 2	Area 3	Area 4	Comparison Area	Total
Composite Soil (average concentration)	126 ppm	119 ppm	70 ppm	142 ppm	148 ppm	116 ppm
Dust in child's bedroom (average concentration)	147 ppm	84 ppm	97 ppm	80 ppm	117 ppm	99 ppm
Dust in entryway (average concentration)	139 ppm	179 ppm	85 ppm	81 ppm	206 ppm	136 ppm
Indoor paint (% homes with lead paint on at least one surface)	7.6%	12.7%	5.8%	5.7%	16.1%	9.6%
Outdoor paint (% homes with lead paint on at least one surface)	37.7%	37.8%	19.4%	42.9%	45.7%	35.5%
Water from kitchen faucet (average concentration)	2.71 ppm	3.73 ppm	3.70 ppm	3.47 ppm	3.70 ppm	3.53 ppm

Other Sources of Contamination on Site

The Toxic Chemical Release Inventory (TRI) is an EPA database that documents contaminants released by industrial facilities into the environment. TRI data is self-reported and only certain types of facilities are required to report. Table 12 lists all chemical releases reported to EPA in the West Dallas area between 1987 and 1991.

According to TRI data, the Murmur Corporation released 500 pounds of lead between 1987 and 1991. These emissions were reported by Murmur in association with its active reclamation and recycling operation at the site; however, Murmur reported these releases to EPA under the category of environmental losses between 11 and 500 pounds per year and the actual amount that was released may be much less than 500 pounds. According to EPA, in 1993 the company estimated that less than 100 pounds of lead per year were emitted to the environment (air, water, soil, etc.) from its processes and the site does not create detectable levels of lead air pollution. Air pollution monitors across the street from Murmur have not detected lead levels in air above the National Ambient Air Quality Standard since 1984 when the smelter closed. We will thoroughly evaluate the levels of lead contamination remaining on the smelter property when we address Operable Unit 4, which is currently being assessed and cleaned up by EPA.

B. Off-site Contamination

Off-site sampling data were not available at this site. The boundaries of the 13.6 square mile site were drawn to include all areas potentially effected by the RSR Smelter.

TABLE 12 - Chemical Releases to Air and Land Reported in Toxic Release Inventory
West Dallas 1987-1991 [11]

DATE	SOURCE OF RELEASE	CONTAMINANT	RELEASED TO	AMOUNT lbs/yr
1987	Roach Paint Co. Inc. 2121 French Settlement Road	Ethylene Glycol	Air	250
	Lilly Industrial Coatings Inc. 2518 Chalk Hill Road	Xylene (mixed isomers)	Air	1,647
	Dallas Woodcraft Inc. 2829 Sea Harbor Road	Xylene (mixed isomers) Toluene Acetone Methyl Isobutyl Ketone Methyl Ethyl Ketone	Air Air Air Air Air	390,250 71,250 23,250 37,250 10,150
	Trinity Brass Copper Co., Inc. 2920 Sylvan	Copper	Air	1,350
	Atlas Manufacturing Co. 2321 Beatrice Street	Dichloromethane Phosphoric Acid Sodium Hydroxide 1,1,1-Trichloroethane Xylene (mixed Isomers) Ammonia Formaldehyde Hydrochloric Acid Methanol	Air Air Air Air Air Air Air Air Air	20 104 80 84 10 4 0 12 0
	Blanks Engraving Co. 23443 North Beckley	Tetrachloroethylene Trichlorethylene Nitric Acid	Air Air Air	34,090 11,880 6,340
	Oak Cliff Plating 2330 North Beckley	Sulfuric Acid	Air Land	500 6,000
1988	Roach Paint Co. Inc. 2121 French Settlement Road	Ethylene Glycol	Air	250
	Lilly Industrial Coatings Inc. 2518 Chalk Hill Road	Xylene (mixed isomers) Ethylene Glycol Toluene Lead Compounds	Air Air Air Air	2,044 1,530 2,861 1,000
	Dallas Woodcraft, Inc. 2829 Sea Harbor Road	Xylene (mixed isomers) Toluene Acetone Methyl Isobutyl Keytone Methyl Ethyl Ketone	Air Air Air Air Air	640,250 63,250 8,550 47,250 7,950
	Trinity Brass Copper Co. Inc. 2920 Sylvan	Copper	Air	1,200
	Blanks Engraving Co. 2343 North Beckley	Tetrachloroethylene Trichloroethylene Nitric Acid	Air Air Air	18,579 7,840 3,005

DATE	SOURCE OF RELEASE	CONTAMINANT	RELEASED TO	AMOUNT (lbs/yr)
1988	Oak Cliff Plating 2330 North Beckley	Sulfuric Acid Cyanide compounds	Air Land Air Land	500 250 500 250
	Murmur Corporation 2823 North Westmoreland	Lead	Air	500
	Economy Forms Corporation 1915 West Commerce Street	Xylene (mixed isomers)	Air	11,196
1989	Lilly Industrial Coatings Inc. 2518 Chalk Hill Road	Xylene (mixed isomers) Barium N-Butyl Alcohol Methyl Isobutyl Ketone Lead Ethylene Glycol Toluene Ethylbenzene	Air Air Air Air Air Air Air Air	2,553 1,875 1,000 1,419 1,000 1,275 4,047 500
	Dallas Woodcraft, Inc. 2829 Sea Harbor Road	Xylene (mixed isomers) Toluene Methyl Isobutyl Ketone 1,1,1-Trichloroethane Glycol Ethers	Air Air Air Air Air	60,250 35,250 12,250 24,250 28,005
	Blanks Engraving Co. 2343 North Beckley	Tetrachloroethylene Trichloroethylene Nitric Acid	Air Air Air	15,086 11,880 16,417
	Murmur Corporation 2823 North Westmoreland	Lead	Air	500
	Economy Forms Corporation 1915 West Commerce Street	Xylene (mixed isomers)	Air	8,613
1990	Lilly Industrial Coatings Inc. 2518 Chalk Hill Road	Xylene (mixed isomers) Isopropyl Alcohol N-Butyl Ketone Methyl Isobutyl Ketone Ethylene Glycol Toluene Lead Compounds Barium Compounds Ethylbenzene	Air Air Air Air Air Air Air Air Air	2,516 750 1,351 755 255 3,095 1,554 1,362 750
	Dallas Woodcraft, Inc. 2829 Sea Harbor Road	Xylene (mixed isomers) Toluene Glycol Ethers	Air Air Air	15,250 18,250 43,005
	Blanks Engraving Company 2343 North Beckley	Tetrachloroethylene Trichloroethylene Nitric Acid	Air Air Air	20,525 9,240 13,643
	Murmur Corporation 2823 North Westmoreland	Lead	Air	500
	Comet Steel Inc. 4846 Singleton Blvd.	Toluene	Air	11,000

DATE	CONTAMINANT	SOURCE OF RELEASE	RELEASED TO	AMOUNT (lbs/yr)
1991	Dallas Woodcraft, Inc. 2829 Sea Harbor Road	Toluene Glycol Ethers	Air Air	18,005 35,005
	Blanks Engraving Company 2343 North Beckley	Trichlorethylene Nitric Acid	Air Air	15,000 2,600
	Murmur Corporation 2823 North Westmoreland	Lead	Air	500
	QDT LTD 1000 Singleton Blvd.	Freon 113 1,1,1-Trichloroethane	Air Air	13,000 13,000

C. Quality Assurance and Quality Control

The EPA has approved all quality assurance and quality control (QA/QC) criteria contained in the referenced documents. In preparing this Health Assessment, TDH staff members relied on the information provided in the referenced documents and assumed that adequate QA/QC measures were followed with regard to chain-of-custody, laboratory procedures, and data reporting. The analyses and conclusions in this Health Assessment are valid only if the referenced information is valid and complete.

D. Physical and Other Hazards

The extensive area contained within the RSR Corporation NPL site does not lend itself to an adequate characterization of existing physical hazards. The buildings and facilities formerly associated with the RSR Corporation smelter are old and in poor condition; areas of the RSR property where slag, battery chips, and other waste materials may have been disposed have not been fully characterized; and the solid waste surface impoundment associated with RSR has not yet been remediated. These remaining structures and areas could present physical hazards if the areas were accessible to the public. To our knowledge, public access to the smelter and its associated facilities is effectively prohibited.

PATHWAYS ANALYSES

We evaluated the environmental and human components required to determine the potential for human exposure to contaminants at the site. This process considers five elements of an exposure pathway: a source of contamination, transport through an environmental medium, a point of exposure, a route of exposure, and an exposed population.

Exposure pathways are categorized as completed, potential, or eliminated. For a person to be exposed to a contaminant, the exposure pathway must be completed. An exposure

pathway is considered to be completed when all five elements in the pathway are present and exposure has occurred, is occurring, or will occur in the future. An exposure pathway is considered to be a potential pathway when at least one of the five elements is missing but may be present in the future. An exposure pathway is eliminated when one or more elements are missing and will never be present to complete the pathway. Completed and potential exposure pathways at this site are presented in Table 12. Inclusion of a pathway in the table does not imply that the pathway presents a risk to public health. An evaluation of the public health significance of each pathway will be presented in the following section.

A. Completed Exposure Pathways

Air

Based on ambient air levels observed in 1982 while the smelter was operating, past exposure to lead has occurred through the inhalation of contaminated air. The pattern of soil lead concentrations that resulted from air deposition of lead suggests that exposures most likely occurred north and northwest of the smelter in the direction of prevailing winds. Individuals who were exposed to site contaminants in the air include on-site workers and people who lived, visited, worked, or attended school within approximately one-half mile of the RSR Corporation Smelter before 1984 while the smelter was operating. Approximately 10,000 people were potentially exposed through this pathway.

Although historical air sampling data were limited, exposure to lead through inhalation of contaminated air was probably higher prior to 1982 when emission controls were stepped up. Exposure to lead via this pathway was eliminated in 1984 when the smelter closed.

Soil

In the past, people living in the residential area just north and northeast of the smelter were exposed to site contaminants in soil (primarily via incidental ingestion). This area was subject to air deposition of contaminants. Residents also were exposed to high levels of contaminants in soil on properties throughout the study area where battery chips, slag, or contaminated soils were used as residential fill or disposed of improperly. The primary contaminants of concern include lead, arsenic, and cadmium.

Exposures to contaminants in soil were significantly reduced in 1984 and 1985 when all soil with lead levels above 1,000 ppm in the high deposition residential area and other high-use areas was removed. Additional remedial measures at that time, such as sodding and fencing of unremediated areas, also reduced the potential for human exposure to contaminants in soil.

The recent remedial effort, initiated in 1991, further reduced the magnitude of exposure to contaminants in soil. During these removal activities, contaminated soil has been removed from most areas of the site that are readily accessible to the public, including areas where battery chips and slag material have been identified. The ATSDR/City of Dallas exposure

study which took place in 1993 and 1994 reports a mean soil concentration of 116 ppm in West Dallas.

Soil contamination in the residential areas of West Dallas has been reduced to a level that is not expected to cause adverse health effects; however, human exposure to contaminants in soil may be occurring and may occur in the future in several areas of the site. The exposed population includes trespassers to unremediated areas of the site, residents living on several properties where property owners have refused remedial measures, and properties with soil lead levels below the 500 mg/kg action level for lead. Unremediated areas also include the slag piles/dumps which have not yet been characterized, the former smelter facility, and other RSR property.

B. Potential Exposure Pathways

Air

Ambient air monitoring data collected while the smelter was operating did not measure levels of cadmium and arsenic; we were therefore unable to determine whether this pathway was completed. Although cadmium and arsenic were detected in soil near the smelter, we could not determine whether it was deposited from the air or associated with contaminated fill and other debris.

Sediment

Sampling data were unavailable for sediment in ponds, drainage ditches, and other surface water collection areas. If runoff from contaminated areas resulted in contamination of sediments, human exposure would be possible via incidental ingestion of sediments. This exposure would be most plausible for young children playing in areas with contaminated sediment. Exposure also would be possible via consumption of contaminated fish.

Surface Water

Limited sampling by the City of Dallas found no lead contamination in surface water. One resident reported finding elevated levels of lead in Fish Trap Lake, but we were unable to obtain a copy of the sampling report for review. If surface water were contaminated, human exposure could occur via incidental ingestion while playing in the water, but this type of activity is unlikely to result in significant levels of exposure.

Pathway Name	TABLE 13 - Exposure Pathway Elements							
	Source	Media	Point of Exposure	Route of Exposure	Exposed Population	Contaminants of Concern	Time	Estimated Number Exposed
Completed Exposure Pathways								
Air	Smelter stacks	Ambient air	On site	Inhalation	Local residents On-site workers	Lead	Past	10,000
Soil	Smelter stacks Contaminated fill/ battery chips Slag piles/disposal areas	Air Soil/dust	On site	Inhalation Ingestion	Local residents, especially children On-site workers Trespassers	Lead Cadmium Arsenic	Past Present Future	1,000
Potential Exposure Pathways								
Air	Smelter	Ambient air	On site	Inhalation	Local residents On-site workers	Cadmium Arsenic	Past	10,000
Sediment	Drainage ditches, ponds	Sediment Fish	On site	Ingestion	Local residents, especially children	Lead	Present Future	500
Surface water	Drainage ditches, ponds	Water	On site	Ingestion	Local residents	Lead	Present Future	500

PUBLIC HEALTH IMPLICATIONS

A. Toxicologic Evaluation

This section discusses the possible health effects that may result from exposure to specific contaminants and the likelihood that adverse health effects will result from exposure to contaminants at levels found on this site. The section also evaluates available health data and addresses specific community health concerns.

To evaluate non-carcinogenic health effects, ATSDR has developed Minimal Risk Levels (MRLs) for contaminants commonly found at hazardous waste sites. The MRL is an estimate of daily human exposure to a contaminant below which non-cancer adverse health effects are not expected to occur during a lifetime of exposure. MRLs are developed for

each exposure, such as ingestion and inhalation, and for different lengths of exposure, such as acute (less than or equal to 14 days), intermediate (15 to 364 days), and chronic (365 days or more). EPA's Reference Doses (RfDs) or Maximum Contaminant Levels (MCLs) are used to evaluate exposures when an MRL is not available for a specific compound.

Chemical-specific cancer slope factors and cancer unit risk factors are used to evaluate carcinogenic health effects. EPA has developed these factors for contaminants commonly found at hazardous waste sites to estimate the increased chance that a person exposed to contaminants will develop cancer over the course of a lifetime.

Lead

Lead is naturally present in most soils and is widespread in the human environment as a result of industrialization. It is generally found in higher concentrations in urban environments, principally as a result of automobile emissions and the use of lead-based paint. The natural lead content of soil derived from crustal rock typically ranges from <10 to 30 parts lead per million parts soil (ppm). The concentration of lead in the top layers of soil varies widely due to deposition and accumulation of atmospheric particulates from numerous human activities associated with lead pollution, including driving automobiles. For example, the concentrations of lead in the upper layer of soil next to roadways are typically 30 to 2,000 ppm higher than natural levels, although these levels drop drastically with increasing distance from the road [32].

Because of the prevalence of lead in the environment, humans are exposed to lead through a variety of media including air, water, and soil, as well as through diet. The relative contribution of each of these sources to total lead intake varies with age and is dependent on site-specific characteristics.

Preschool-age children and fetuses are usually the most vulnerable segments of the population for exposures to lead. This increased vulnerability results from a combination of factors which include the following: 1) the developing nervous system of fetuses and neonates are more susceptible to the neurotoxic effects of lead; 2) young children are more likely to play in dirt and to place their hands and other objects in their mouths, increasing the opportunity for soil ingestion, (pica, the eating of dirt and other non-food items, also is more likely to occur in children); 3) the efficiency of lead absorption from the gastrointestinal tract is greater in children than in adults; and 4) nutritional deficiencies of iron or calcium, which are prevalent in children, may facilitate lead absorption and exacerbate the toxic effects of lead [33].

Infants often are born with lead in their bodies due to their mother's past exposure to lead. Infants and children are exposed to lead mainly through diet and ingestion of non-food materials associated with normal early hand-to-mouth behavior. The degree to which hand-to-mouth behavior contributes to blood lead levels depends on the levels of lead in house dust, soil, and paint. In the United States, leaded paint continues to cause most of the severe

lead poisoning in young children because it is the most widespread source and has the highest concentration of lead per unit of weight [33].

Most adults are exposed to lead from dietary sources. In some instances, occupational sources also are a significant source of exposure. A great deal of information on the health effects of lead has been obtained through years of medical observation and scientific research. Below is a summary of available toxicological information on lead.

Absorption, Metabolism, and Excretion

In adults, inorganic lead is readily absorbed through the lungs (approximately 40%), and less well absorbed through the gastrointestinal tract. Children absorb as much as 50% of ingested lead. Inorganic lead is not absorbed through the skin. Absorption of lead from the lungs is dependent on the size of inhaled particulates. Particles in the range of 0.5 to 5.0 microns may be deposited in the alveoli and absorbed. Larger particles are likely to be removed by ciliary action and subsequently ingested.

Lead enters the bloodstream when it is first absorbed and is almost always carried bound to the red blood cell. It is distributed throughout different compartments of the body; the highest concentrations are in bone, teeth, liver, lung, kidney, brain, and spleen. These compartments and their interrelations have been used in metabolic models to qualitatively and quantitatively describe absorption, distribution, deposition, accumulation, and excretion of lead. Although the blood contains a small portion of the total body lead burden, it is easily accessible and is the fraction that most closely correlates with recent environmental exposures. The overall half-life of lead in blood is estimated to be 36 days $\pm$ 5 days [32].

With prolonged exposure over time, most absorbed lead ends up in bone by substituting for calcium in the bone matrix. Lead in bone has been estimated to represent approximately 95% of the total body lead burden [32] and is estimated to have a half-life as long as 10 years [34]. While lead in bone is not known to cause any harmful effects, the accumulation of lead in bone can provide a source for remobilization of lead and continued toxicity after exposure has ceased.

Acute Toxicity

The most serious effects of acute high dose lead exposure is encephalopathy, characterized initially by headache and drowsiness, and in more severe cases by coma, convulsions, and death. Virtually all children who recover from acute lead encephalopathy exhibit residual reduction in intelligence and behavioral dysfunction. Acute encephalopathy is usually associated with high blood lead levels (over 150 μg/dL). Another effect of acute high dose lead exposure is the Fanconi syndrome, an acute injury to the renal tubules, characterized by spillage of glucose, protein, amino acids, and phosphates into urine.

Chronic/Subchronic Toxicity

General

Chronic exposure to lead principally affects three organ systems: the hematologic system (red blood cells and their precursors), the central and peripheral nervous system, and the kidneys. Lead also has been shown to have adverse effects on the reproductive system in both males and females. Lead is especially harmful to unborn children. Exposure to lead during pregnancy has been correlated with premature births, low birth weight infants, and spontaneous abortions. While the impact of maternal and cord blood lead levels below 10 μg/dL have not been well-defined, reduced gestational age and reduced birthweight have been associated with blood lead levels of 10 to 15 μg/dL [33]. In addition, lead has been found to lower intelligence quotient (I.Q.) scores, slow growth, and cause hearing problems in children. These adverse effects can persist and lead to decreased performance in school.

Hematologic

Anemia is the most serious effect of lead on the hematologic system. Lead-induced anemia occurs primarily by the lead-induced inhibition of several enzymes involved in the production of hemoglobin. General population studies indicate that the activity of aminolevulinic acid dehydrogenase (ALAD) a heme biosynthetic enzyme is inhibited at very low blood lead levels with no apparent threshold.

ALAD activity was inversely correlated with blood lead levels over the entire range of 3 to 34 μg/dL in urban subjects never exposed occupationally [34]. Other reports have confirmed the correlation and apparent threshold in different age groups and exposure categories (children-[35,36]; adults-[37]). Lead begins to inhibit the enzyme ferrochelatase, which catalyzes the transfer of iron from ferritin into protoporphyrin to form heme, at blood lead levels of 15 μg/dL in children and 25 to 30 μg/dL in adults. Another red blood cell enzyme, erythrocyte pyrimidine-5'-nucleotidase (EPN), is also inhibited in children at very low blood lead levels. A significant negative linear correlation between EPN and blood lead level was seen in 21 children with blood lead levels ranging from 7 to 80 μg/dL [38]. Similar findings were reported in 42 children whose blood lead levels ranged from <10 to 72 μg/dL [39].

Anemia, defined as a hematocrit of $<35\%$, has not been observed at lead levels below 20 μg/dL although many of the enzymes involved in heme biosynthesis are affected at very low blood lead levels. Heme synthesis is essential not only to hemoglobin but also to the synthesis of cytochromes necessary for all oxidative reactions. In children, exposure to lead has been shown to inhibit formation of the heme-containing protein cytochrome P-450, as reflected in decreased activity of hepatic mixed-function oxygenases [40,41]. Impairment of cytochrome P-450 activity could decrease the body's ability to detoxify other toxicants.

Central Nervous System

Chronic exposure to low lead levels has been shown to cause subtle effects on the central nervous system which manifest as deficits in intelligence, behavior, and school performance [42]. Recent information indicates that children with blood lead levels as low as 10 μg/dL can develop neurological and cognitive deficits [43]. Available evidence is not sufficient to determine whether lead-associated deficits are irreversible [44].

Peripheral Nervous System

In the peripheral nervous system, lead can cause neuropathy which primarily affects the motor nerves and appears to be axonal [45]. Lead targets motor axons and produces axonal degeneration and segmental demyelination [46]. Clinically, the neuropathy is more severe in the upper extremity; in extreme cases this demyelination can produce palsy of the wrist and ankle extensor muscles, termed "wrist or ankle drop" or "painter's palsy." While the classic wrist drop has become exceedingly rare, subclinical neuropathy has been demonstrated at blood lead levels previously considered acceptable for workers [47]. Decreases in ulnar nerve conduction velocity are seen at blood lead levels of 30 to 40 μg/dL.

Renal

Renal effects of lead have been studied extensively in humans and animals. Exposure to lead has been associated with hypertension, renal failure, and gout. Lead accumulates in the proximal tubular cells affecting urate excretion and forming densely staining intranuclear bodies. Systemic hypertension and interference with lipoprotein metabolism from lead toxicity can exacerbate chronic renal failure due to glomerular sclerosis. Exposure to high levels of lead can cause irreversible abnormalities in renal function including decreased glomerular filtration and decreased tubular concentrating ability [48].

Cardiovascular

Information from both occupational and general population studies, including the National Health and Nutrition Survey II (NHANES II), is sufficient to suggest a small but positive association between blood lead levels and increased blood pressure. A simple correlational analysis of the NHANES II data in 1988 showed statistically significant associations between blood lead levels and systolic and diastolic blood pressure for both men and women, 12 to 74 years of age [49]. Subsequent statistical analyses, controlling for a number of other potentially confounding factors, indicated significant associations between blood lead and blood pressure in men only. Additional analyses focusing on white men (40 to 59 years of age) revealed significant associations between blood lead and blood pressure, with no apparent threshold below which blood lead level was not significantly related to systolic or diastolic blood pressure across a range of 7 to 34 μg/dL [50]. Lead was a significant predictor of diastolic blood pressure greater than or equal to 90 mmHg, the criterion now used to define hypertension. A small but significant correlation between systolic blood

pressure and blood lead levels also was found in a clinical survey of 7,735 men between 40 and 49 years old [51,52].

Carcinogenicity

Lead has not been shown to be carcinogenic in humans; however, high doses of lead have been found to produce kidney tumors in laboratory studies of rats and mice. The extremely high cumulative doses of lead used in animal studies are difficult to extrapolate to low-level exposure in humans, and do not provide a sufficient basis for quantitative risk assessment. Based on animal data, EPA currently classifies lead as a B2 carcinogen (probable human carcinogen).

Indices of Toxicity

Although no threshold level for adverse health effects has been established, evidence suggests that adverse effects occur at blood lead levels at least as low as 10 μg/dL. The Centers for Disease Control and Prevention (CDC) has determined that a blood lead level greater than or equal to 10 μg/dL in children indicates excessive lead absorption and constitutes the grounds for intervention. The 10 μg/dL level is based on observations of enzymatic abnormalities in the red blood cells at blood levels below 25 μg/dL and observations of neurologic and cognitive dysfunction in children with blood lead levels between 10 and 15 μg/dL [44].

The National Institute of Occupational Safety and Health (NIOSH) considers blood lead levels above 40 μg/dL to indicate excessive lead absorption in adults. Under regulations set forth by the OSHA, adult workers with blood lead levels above 50 μg/dL must be removed from occupational lead exposure until their blood lead levels have fallen to below 40 μg/dL. It is important to note that although this is the regulatory standard, biological effects have been reported at blood lead levels below this value [32].

Blood Lead/Soil Lead Relationship

A number of studies are available relating blood lead levels in children to levels of lead in the environment [53]. In general, blood lead levels rise 3-7 μg/dL for every 1,000 ppm increase in soil or dust lead concentration. Assuming a background blood lead level of 6.6 μg/dL, a blood lead level of 10 μg/dL would result from soil lead levels ranging from 485 ppm to 1,133 ppm. The results of several studies, however, have indicated that the increase in blood lead as a function of soil lead concentration is not linear. The rate of increase in blood lead levels at low concentrations of lead in soil is greater than at high concentrations of lead in soil. Based on data from exposure studies conducted at Helena Valley in Montana and Silver Valley in Idaho, a regression model for the correlation between blood lead levels and soil lead levels was derived [54]. This approach predicts that a soil lead concentration of 500 ppm would result in a blood lead level of 10.7 μg/dL.

A great deal of information is available on the absorption, distribution, and excretion of lead by the body. Using this information, EPA has developed an Integrated Uptake/Biokinetic Model (IU/BK Model, Version 0.5) to estimate the effects of lead intake from various sources on the potential blood lead levels in children under age seven [52]. The IU/BK model considers the monthly uptake of lead from diet, air, soil/dust, water, and paint sources, and media specific absorption factors to estimate monthly blood-lead intake. Using default parameters supplied with the model an average exposure to a soil concentration of 500 mg/kg would result in a geometric mean (average) blood lead level of 5.78 μg/dL. Using the default geometric standard deviation of 1.42, approximately 95% of the children exposed to this soil would be expected to have blood lead levels below 10 μg/dL.

A recent study was conducted to evaluate the effectiveness of removing lead-contaminated soil on reducing blood lead levels [55]. The study was conducted among children with blood lead levels ranging from 10 to 20 μg/dL, living in areas with multiple potential sources of lead exposure and high soil lead levels (the soil lead level at many residence was over 3,000 ppm). An average soil lead reduction of 1,790 ppm (down to a median level of 105 ppm) was associated with a decline in blood lead levels of only 0.8 to 1.6 μg/dL, independent of factors such as water, dust, and paint levels, children's mouthing behaviors, and other characteristics.

Based on soil lead levels measured in residential areas of West Dallas prior to the most recent remedial efforts, ingestion of contaminated soil could have resulted in elevated blood lead levels. Inhalation of contaminated air when the site was active also would have contributed significantly to elevating blood lead levels in the past. Adverse health effects from past exposures, therefore, were possible. More recent soil sampling data suggest that average soil lead levels remaining in residential areas of West Dallas are well below 500 ppm (average remaining soil lead levels range from 70 ppm to 142 ppm). Exposure to these levels of lead in soil would not be expected to result in elevated blood lead levels.

Cadmium

Small amounts of cadmium occur naturally in all soils and rocks, including coal and mineral fertilizers. In the environment, cadmium usually is not found in the metallic state, but is combined with other elements such as oxygen, chlorine, or sulfur. Most cadmium in the environment is released by human activities such as mining and smelting operations, fuel combustion, disposal of metal-containing products, and application of phosphate fertilizer or sewage sludges. Cadmium is extracted from natural materials during the production of other metals including lead, zinc, or copper [56].

Cadmium concentrations in non-polluted soil are highly variable, depending upon sources of minerals and organic materials. The mean level of cadmium in uncontaminated topsoil in the U.S. is approximately 0.25 ppm. Soil becomes contaminated primarily through the deposition of airborne cadmium, landspreading of municipal sludge, and the application of phosphate fertilizers. Cadmium enters the environment through the air as a result of burning coal and household waste, and metal mining and refining processes.

Food is the major source of human exposure to cadmium in the general population. Average cadmium levels in U.S. food range from 2 to 40 parts of cadmium per billion parts of food. Adults consume approximately 30 μg of cadmium in food each day, absorbing approximately 1 to 3 μg. Vegetables generally contain the highest levels of cadmium, particularly leafy vegetables and potatoes. Liver, kidney, and shellfish contain higher levels (up to 1 ppm) of cadmium than other meat and seafood products. For populations surrounding hazardous waste sites increased exposure can result from eating fruits and vegetables grown in cadmium-contaminated soil, ingestion of cadmium-contaminated dust on food or hands, and from the direct ingestion of soil. In highly polluted areas, the daily intake of cadmium can be as high as 400 μg per day [56].

Inhalation is another major source of cadmium exposure. Average concentrations in air range from less than 1 to 6 nanograms per cubic meter of air (ng/m^3) in rural areas and from 20 to 700 ng/m^3 in industrial areas. For the general population, cadmium intake by inhalation averages 0.02 μg/day but can be as high as 2 μg/day in highly polluted areas. Cigarette smokers absorb an additional 1 to 3 μg per day (based on smoking one pack of cigarettes per day). Populations surrounding hazardous waste sites can be exposed to higher levels of cadmium through inhalation of fugitive dust emissions from cadmium-contaminated soil.

Absorption of cadmium depends to a great extent on the route of exposure and the type of compound containing the cadmium. It has been estimated that only about 5% of cadmium ingested as cadmium salts is absorbed. The amount of cadmium an individual absorbs following ingestion also depends upon factors such as age, pregnancy history, method of ingestion (food, water, soil), body stores of iron, calcium, and zinc, and the presence of other dietary components ingested with the cadmium. Absorption of cadmium from the respiratory system is quite different. More than 90% of inhaled cadmium can be absorbed, depending on the in vivo solubility of the inhaled compound. The amount of cadmium that is absorbed is also affected by the availability of the cadmium to the lung, which is largely a function of particle size. Only 5% of particles greater than 10 microns in diameter are deposited in the lung, while up to 50% of particles less than 0.1 micron are deposited [56].

Once cadmium is absorbed, it is bound to red blood cells and serum albumin. Serum cadmium is rapidly taken up by the liver and kidney. Since the half-life of cadmium is thought to be quite long (25 to 30 years), cadmium accumulates in these tissues. Over 50% of the body burden of cadmium is found in the liver and kidney. It has been hypothesized that there is a critical concentration of cadmium in the kidney above which cadmium-induced nephropathy will occur. The placenta appears to be an effective barrier to cadmium; however, fetal exposure can occur with high maternal exposure.

The toxic effects of chronic exposure to cadmium occur primarily in the lungs and in the kidney. Effects on the lungs are associated solely with inhalation exposure, while the kidney effects may occur after oral or inhalation exposures. Chronic cadmium exposure also has been associated with cardiovascular disease and cancer of the lung, prostate, kidney, and stomach, although these associations are not well established.

The chronic pulmonary effects of cadmium exposure in humans is manifested as an obstructive lung disease. Long-term inhalation exposure to cadmium may cause decreased lung function and emphysema; Pulmonary assessment of patients with long-term inhalation exposure typically indicates a reduced vital capacity and an increased residual lung volume. The level of obstructive disease appears to be related to the duration and level of cadmium exposure [57,58]. Studies on the effects of cadmium exposure via inhalation are often complicated by such factors as cigarette smoking.

Long-term exposure to cadmium can effect the kidneys, causing proximal tubular necrosis, lesions in the renal cortex, and kidney dysfunction. Common laboratory findings include the presence of protein, amino acids, and glucose in the urine. Damage is thought to occur when the renal cortical cadmium concentration exceeds the "critical" level of 200 μg/g (wet weight) [56]. Others have proposed that for the general population, the amount of cadmium accumulated in the renal cortex should not exceed 50 μg/g, a level corresponding to a urinary excretion of 2 μg of cadmium per 24-hours [59]. Average levels in non-occupationally exposed 50 year-olds are approximately 15-30 μg/g (wet weight) [60]. Cigarette smoking can double renal cortical cadmium concentrations. Because of the high amount of cadmium ingested through diet, the margin of safety for exposure to cadmium from other sources is very small, particularly for smokers.

Studies on humans occupationally exposed to cadmium in the air report a No Observable Adverse Effects Level (NOAEL) for kidney toxicity (proteinuria) of 0.017 mg/m^3 [61]. Adjusting for continuous lifetime exposure and using an uncertainty factor of 10, ATSDR has used this NOAEL to derive an inhalation MRL of 0.0002 mg/m^3.

Studies on humans orally exposed to cadmium in cadmium-polluted areas of Japan report a NOAEL for kidney toxicity (proteinuria) of 0.0021 mg/kg/day [62]. Using an uncertainty factor of 3, ATSDR has used this NOAEL to establish a chronic oral MRL of 0.0007 mg/kg/day. The EPA used a toxicokinetic model to determine the level of chronic human oral exposure which would result the highest renal cadmium level not associated with significant proteinuria (200 μg/gram wet weight). Assuming 2.5% absorption of cadmium from food and 5% from water, the model predicts a NOAEL for chronic cadmium exposure of 0.005 and 0.1 mg/kg/day from water and food, respectively. Based on these NOAELs and an uncertainty factor of 10, the EPA calculated a chronic oral reference dose (RfD) of 0.0005 mg cadmium/kg/day for water and an equivalent RfD for cadmium in food of 0.001 mg cadmium/kg/day [63].

Substantial evidence from laboratory studies suggests that cadmium inhalation can cause lung cancer in rats, but evidence that cadmium inhalation can cause lung cancer in humans is rather weak. Evidence for evaluating potential carcinogenicity of cadmium by the oral and dermal routes in both animals and humans is insufficient. Based on extensive animal data and limited human data associating inhalation of cadmium with lung cancer, EPA classifies cadmium as a B1 carcinogen (probable human carcinogen).

In West Dallas, cadmium was detected at levels above the HAC value. Due to the cumulative nature of this toxin, however, adverse effects on the kidneys from low levels of exposure have been observed only after many years of exposure (i.e. fifty years). Based on available information, exposure to cadmium at the levels detected in West Dallas would not be expected to produce adverse health effects in most children and adults.

Arsenic

Arsenic is a naturally occurring element in the earth's crust, but is not common in the environment in elemental form. It is usually found in combination with other elements. Arsenic compounds can be classified into three main groups: 1) inorganic arsenic compounds, 2) organic arsenic compounds, and 3) arsine gas [64]. In the environment, arsenic is most often found as inorganic arsenic, which is formed when arsenic combines with other elements such as oxygen, sulfur, and chlorine. Inorganic forms of arsenic are usually more toxic than organic forms, which are produced when arsenic forms stable bonds with carbon. Inorganic compounds are important because they are the primary metabolites formed from inorganic arsenic in biological organisms. Arsine gas is the most toxic form of arsenic. In the United States, arsenic has been used in insecticides, herbicides, desiccants in the cotton industry, wood preservatives, animal feed additives, and more recently in the semiconductor industry. Environmental releases of arsenic are associated with application of pesticides, disposal of solid wastes, land applications of municipal sludge, fossil fuel combustion, and industrial processes.

Since arsenic is a natural part of the environment, people are exposed to low levels of arsenic in soil, water, food, and air. The average adult takes in approximately 50 micrograms of arsenic per day from these sources, with food as a major contributor of total intake. Based on Food and Drug Administration (FDA) Market Basket Studies, adults ingest approximately 52.6 μg arsenic per day in food. However, since organic arsenic is the predominant form of arsenic in food, the daily intake of inorganic arsenic from food is much lower (approximately 10-14 μg/day). Exposure to inorganic arsenic from other sources may range from 5 to 20 μg arsenic per day depending upon the levels of arsenic in air and water. When arsenic levels in the environment are higher than normal, greater exposure occurs. Exposure to arsenic among children living near metal smelters, particularly copper smelters, has also been reported [65].

Both inorganic and organic arsenic compounds are well absorbed. Over 90 percent of an ingested dose of inorganic trivalent or pentavalent arsenic is absorbed. Most organic and inorganic arsenic leaves the body in urine within a few days of exposure, although some remains in the body for several months or longer. The liver converts some arsenic to a less harmful organic form, but large doses can cause adverse health effects [64].

Very high doses of inorganic arsenic (above 60,000 ppb in food and water) can result in death. Lower levels (300 to 30,000 ppb in food or water) can cause stomach and intestinal irritation. Chronic exposure to arsenic through ingestion has been known to cause irritation

of the digestive tract leading to pain, nausea, vomiting, and diarrhea. The minimal dose at which these effects are usually observed in humans has ranged from 0.012 mg/kg/day to 0.05 mg/kg/day [66,67]. Other effects from swallowing arsenic include decreased production of red and white blood cells, abnormal heart function, blood vessel damage, and impaired nerve function causing a "pins and needles" sensation in the hands and feet. Sensitive individuals often begin to show one or more of the characteristic signs of arsenic poisoning at oral doses of about 0.02 mg/kg/day.

Although there is no evidence to suggest that arsenic can injure pregnant women or their fetuses, studies of animals show that doses large enough to cause illness in pregnant females may also cause low birth weight, fetal malformations, or fetal death. The single most characteristic effect of long-term oral exposure to inorganic arsenic is a pattern of skin changes that includes a darkening of the skin similar to a corn or wart on the palms, soles, and torso [64].

EPA's reference dose for arsenic of 0.0003 mg/kg/day is based on two studies in which hyperpigmentation, keratosis, and possible vascular complications were the critical effects [68,69]. The reference dose for arsenic was derived by dividing the estimated NOAEL for these studies, determined to be 0.0008 mg/kg/day, by an uncertainty factor of three to account for sensitive individuals and the lack of data on reproductive toxicity.

There is convincing evidence from a large number of epidemiologic studies and case reports that ingestion of arsenic increases the risk of developing skin cancer. The most common lesions are multiple squamous cell carcinomas which appear to develop from hyperkeratotic warts or corns. Multiple basal cell carcinomas may also occur, usually from cells not associated with hyperkeratinization. In most cases, skin cancer develops only after prolonged exposure. However, several studies have reported skin cancer in people exposed for less than one year. Liver, bladder, kidney, and lung cancer also have been associated with exposure to arsenic, but these associations are less well established [64].

In West Dallas, arsenic has been found at levels exceeding its HAC value. However, available data are insufficient to adequately assess the public health significance of this contaminant.

B. Health Outcome Data Evaluation

The following section discusses in greater detail the data presented previously in this document under the heading of Health Outcome Data.

Data indicate that blood lead levels among children in the West Dallas area have declined significantly since 1982 when the first comprehensive blood lead study was conducted in the area. In 1982, the mean blood lead level of 269 randomly-selected preschool children living within one-half mile of the RSR Smelter was 20.1 μg/dL, with ten percent of children having blood lead levels greater than or equal to 30 μg/dL. These blood lead levels were high

enough to be associated with effects on the central nervous system, which manifest as deficits in intelligence, behavior, and school performance, as well as subtle effects on the blood and the kidneys [44]. We were unable to evaluate the prevalence of these specific conditions which may have resulted from chronic exposure to lead in West Dallas because many of the effects are not readily measurable and are associated with multiple potential causes.

In contrast to historical exposure data, the 1993 lead exposure study found that the average blood lead level of 305 randomly-selected West Dallas preschool children was 5.5 μg/dL, with 26 children (8.5 percent) having blood lead levels greater than or equal to 10 μg/dL (Table 14). The **maximum** blood lead level measured in 1993 was *below* the **average** blood lead level measured in 1982. In the 1993 study, blood lead levels observed among West Dallas children differed only slightly from blood lead levels of children in the Oak Cliff neighborhood, which served as a comparison community. The mean blood lead level of children in the comparison community was 5.4 μg/dL and 4.9 percent of tests were greater than or equal to 10 μg/dL.

In the 1992 study, the highest mean blood lead level (7.0 μg/dL) and the largest proportion of children with elevated blood lead levels (10 of 53 children) in a single subarea were observed in Area 1. Although this area includes the residences nearest the smelter in the high air dispersion area, the mean soil lead level reported for this area was only 126 ppm. The study did not find a relationship between blood lead concentrations and lead concentrations in residential soil, home dust, or other environmental media. The reason for the remaining elevated blood lead levels in this area should be further explored.

The 1982 and 1993 exposure studies provide the most extensive information about lead exposure among West Dallas children since each study includes a randomly-selected study group from within the defined area of concern as well as environmental exposure data. Recent data collected at Walk-in Clinics and by the EPSDT program are more difficult to interpret since they were not collected randomly and do not include exposure data; but these data provide additional evidence that lead levels among West Dallas children have decreased significantly in recent years (Table 14).

TABLE 14 - Comparison of 1982 and 1993 Blood Lead Data for Randomly-Selected West Dallas Children [17,22]

Source of Data	Elevated Tests				Mean Blood Lead Level (μg/dL)
	≥ 10 μg/dL		≥ 30 μg/dL		
	# elevated/ # tested	percent elevated	# elevated/ # tested	percent elevated	
1982 Lead Study	na	na	26/269	10	20.1
1993 Lead Exposure Study	26/305	8.5	na	na	7.0

Blood lead data collected at Walk-in Clinics are difficult to interpret since individuals may have been tested because they suspected that exposure to lead had occurred; therefore blood lead levels reported by these clinics may not reflect blood lead levels found in the general population. Although the City of Dallas began to offer free-of-charge blood lead screening tests at voluntary walk-in clinics in 1984 for all residents of the West Dallas area, the earliest data from this program available for review were collected in 1991. Two periods of blood lead summary data were reviewed (July through December 1991 and July 1992 through December 1993). Among the 110 tests reported during the 1991 period for City of Dallas Walk-in Clinics, 33 children (30 percent) had elevated blood lead levels ($\geq$ 10 μg/dL). These tests are particularly difficult to generalize to the larger population because the total number of West Dallas residents tested (110) was small. In the 1992/93 report, 158 (13.9 percent) of the 1,138 West Dallas children ages six and under who were tested at City of Dallas walk-in clinics had elevated blood lead levels (Table 15).

TABLE 15 - Comparison of Recent Blood Lead Data from West Dallas and Other Areas of Dallas [17,22]

Source of Data	West Dallas		Other Dallas	
	Number $\geq$ 10 μg/dL per number tested	Percent $\geq$ 10 μg/dL	Number $\geq$ 10 μg/dL per number tested	Percent $\geq$ 10 μg/dL
1992-1993 Walk-in Clinic	158/1,138	13.9	59/486	17.9
1993 EPSDT Program	125/1,126	11.1	330/5,361	9.1

While these walk-in clinic data may not accurately reflect rates of elevated blood lead levels in West Dallas or in Dallas as a whole, it is unlikely that results underestimate the significance of lead as a public health problem in West Dallas compared with the rest of the city. When compared with blood lead results of residents from other parts of Dallas, the 1992/3 report indicates that the mean blood lead levels of West Dallas residents tested during this period in Walk-in clinics was comparable or slightly higher than the mean blood lead level of other Dallas residents tested during this period in every age group. These data also indicate that average blood lead levels of children tested in walk-in clinics were significantly lower than levels found through random testing in 1982.

The 1992/93 Walk-in Clinic data include the only available blood lead levels reported separately for pregnant women. These data indicate that the mean blood lead level of the 19 pregnant women residing in West Dallas area who were tested was 3.5 μg/dL. The maximum blood lead level observed among these pregnant women was 8.0 μg/dL. While the impact of maternal and cord blood lead levels below 10 μg/dL have not been well defined, known fetal effects including reduced gestational age and reduced weight at birth have not been associated with the blood lead levels observed among West Dallas women [33].

Data collected through the EPSDT program during the period between January and June

1993 also indicate that blood lead levels were not substantially different among West Dallas children and children from other parts of Dallas (Table 15). Among children living in West Dallas, approximately 11 percent had elevated blood lead levels, while approximately 9 percent of children from other parts of Dallas had elevated blood lead levels (≥ 10 μg/dL).

Despite the historical reduction of blood lead levels in the West Dallas area demonstrated by all available blood lead data, a number of children continue to have elevated blood lead levels. Although these data do not indicate that elevated blood lead levels are associated with identifiable environmental factors, we cannot identify, with certainty, the source of lead exposure.

C. Community Health Concerns Evaluation

We have addressed the community concerns about health as follows.

1. Why is so much attention still focused on children?

Children in West Dallas are of particular concern because children experience many of the adverse health effects associated with lead at lower levels of exposure than adults. The increased vulnerability of children results from a combination of factors: 1) the developing nervous system is more susceptible to neurotoxic effects of lead; 2) young children are more likely than adults to experience higher exposures to lead in soil, dust, and paint because of normal early hand-to-mouth behavior (pica, the eating of dirt and other non-food material, is also more likely to occur among children); 3) children absorb lead more efficiently from the gastrointestinal tract than adults; and 4) nutritional deficiencies of iron or calcium, which are prevalent in children, may increase the amount of lead that the body absorbs through the gastrointestinal tract. At the RSR site, children also may have been exposed to higher levels of cadmium and arsenic than adults because of their frequent outdoor play and normal hand-to-mouth behavior.

2. Should we be concerned about health effects among the residents who grew up in West Dallas and were exposed to lead all of their lives? Are they likely to develop illnesses resulting from their exposure to lead?

Residents who experienced exposure to lead during childhood are not likely to develop illnesses later in life as a result of past exposure. When lead enters the human body, it travels to the soft tissues, such as the liver, kidneys, lungs, brain, spleen, muscles, and heart. In adults, about 99% of lead that is taken in leaves the body in the waste within a couple of weeks; in children, about 32% of lead is eliminated within a couple of weeks. Lead that remains in the body can be stored in the bones and teeth up to several decades, but lead stored in the body has no known health effects. The accumulation of lead in bone can provide a source for remobilization of lead to other parts of the body, but because the amount of lead remobilized is small, stored lead is unlikely to cause adverse health effects after exposure has ceased. According to available information, only individuals with very

high exposures (i.e. exposures indicated by blood lead levels over 50 μg/dL) are at risk of continued toxicity after exposure has ceased.

Blood lead levels measure lead from both remobilized lead and lead from current exposures. Measures of blood lead level are easily obtainable with a blood lead test. If current blood lead levels are not elevated (above 10 μg/dL for children and approximately 25 μg/dL for adults) adverse health effects should not be expected.

Since the past exposure levels of any given individual who grew up in West Dallas cannot be determined, it is difficult to know whether adverse health effects occurring in individuals are the result of past exposures to lead. Many of the outcomes associated with lead exposure (such as effects on kidneys, blood, and central nervous system, and particularly those associated with cognitive and physical development) that could occur at blood lead levels observed among West Dallas residents in the past are subtle and difficult to measure. These outcomes also can be associated with many factors other than lead. While exposure to the levels of lead observed in West Dallas in the past may have caused adverse health effects, residents who grew up in West Dallas are not likely to develop new illnesses resulting from their past exposure to lead.

Much of the published information about long-term effects of lead exposure has focused on people whose primary exposures occurred at work and who had chronically elevated blood lead levels in the range of 50 to 60 μg/dL or higher. These blood lead levels have been associated with a general increased risk for developing kidney problems after many years of exposure. Some laboratory evidence suggests that effects on the kidneys can occur at blood lead levels below this range, but there is no evidence that clinical illnesses or symptoms that could be detected by a physician will occur. The blood lead levels reported among West Dallas residents would not be expected to be associated with these types of problems. Studies of individuals who have experienced lead levels below approximately 20 to 30 μg/dL generally do not indicate any additional risk for developing illnesses years after exposure.

3. Should individuals with high blood lead levels be tracked for follow-up medical treatment?

Yes. Children with blood lead levels in the range of 10 to 14 μg/dL should be retested with a venous blood test to insure that the blood lead level does not increase. Adverse effects of blood lead levels in this range are subtle and are not likely to be clinically recognizable or measurable in an individual child. Although an environmental history should be taken to determine any obvious remediable source of lead, it is unlikely that there is a single predominant source of exposure for these children.

Children with blood lead levels in the 15-19 μg/dL range need more careful follow-up. This level of exposure indicates risk for decreased in IQ of up to several points and other subtle effects. Parents and health care providers should seek interventions to reduce possible blood lead exposures among children with these levels. If levels persist at or above 15 μg/dL, environmental investigation and appropriate remediation should be completed.

Children with venous blood lead levels between 20 and 69 μg/dL should have a full medical evaluation including a detailed environmental and behavioral history, physical examination, and tests for iron deficiency. The most important factor in managing childhood lead poisoning is reducing the child's exposure to lead, but some children with higher blood lead levels will benefit from chelation (treatment with oral medication that must be supervised by a physician). Health-care providers with limited experience in treating lead poisoning should consider referring children needing urgent medical follow-up (those with blood lead levels $\geq$ 45 μg/dL), to a clinic with experience managing childhood lead poisoning.

Blood lead levels greater than or equal to 70 μg/dL in children constitute a medical emergency, requiring in-patient chelation therapy. The patient preferably must be managed by someone with experience in treating children who are critically ill with lead poisoning. Medical and environmental management must begin immediately.

Medical follow-up generally is necessary for adults who experience significantly higher lead exposures than children. The CDC recommends retesting and monitoring for adults with blood lead levels of 25 μg/dL and above. The Occupational Health and Safety Administration (OSHA) recommends that adults working around potential sources of exposure to lead receive annual blood lead screening tests and that medical consultations be obtained for workers with blood lead levels above 40 μg/dL. OSHA requires medical removal for workers with blood lead levels of 50 μg/dL.

More stringent medical guidelines should apply to pregnant women because of the unique risks associated with fetal exposure to lead. Maternal blood lead levels between 10 and 15 μg/dL have been associated with fetal effects. In addition, effects on miscarriage rates and the ability to become pregnant have been detected among women with blood lead levels between 10 and 15 μg/dL.

4. Can residents be provided with free medical help for pollution-related illnesses?

Neither TDH nor ATSDR have resources to provide free medical assistance to citizens with pollution-related illnesses. If resources were available, it would be difficult to: a) diagnose many illnesses and conditions which could be related to pollutants at this site; b) determine whether the level of pollution to which an individual patient was exposed in the past was sufficient to cause illness; and 3) determine with certainty whether the diagnosed illness was caused by the specific past exposure.

Lead is the principal pollutant identified at the RSR site at sufficient levels for causing pollution-related illnesses. While severe lead exposure in children (blood lead levels $\geq$80 μg/dL) can cause coma, convulsions, and even death, the primary effects associated with lower levels of lead exposure, such as the levels observed among West Dallas residents, are much more subtle and difficult to measure. These outcomes include adverse effects on the central nervous system, kidneys, and blood. All of these effects can be associated with multiple causes and risk factors.

5. Could exposure to lead in the environment during pregnancy cause learning disabilities and hearing problems for the unborn child?

Yes. In several, but not all studies, prenatal exposures have been associated with slower sensory-motor and delayed early cognitive development. One study suggests that with low postnatal exposures and favorable socioeconomic conditions, some of these early associations may decrease as children grow older.

6. Is EPA's 500 ppm clean-up level adequate to protect public health?

Yes, while there is general agreement that children's exposure to lead should be reduced as much as possible, evidence from various sources suggests that under typical exposure scenarios, average soil lead concentrations of 500 ppm and less do not represent a threat to public health. In West Dallas, all available blood lead data suggest that the 500 ppm clean-up level has been adequate to protect public health. The recent Biological Indicators of Exposure Study in West Dallas did not find any association between blood lead levels and remaining soil lead concentrations.

It is also important to note that the clean-up level itself does not represent the *average* level of lead remaining in soil and that the average level of lead in soil is a more accurate reflection of potential exposure than the clean-up level. While the West Dallas clean-up level was 500 ppm, the most recent soil sampling results, collected in conjunction with the 1993 exposure study, indicate that average concentrations of lead remaining in soil were much lower than 500 ppm. The study reported remaining lead concentrations in residential areas for each subarea (1-4) of the study as follows: Area 1/high air dispersion area, 126 ppm; Area 2/eastern low air dispersion area, 119 ppm; Area 3/western low air dispersion area, 70 ppm; and Area 4/area with slag or battery chips, 142 ppm. The highest average soil concentration was found in the Oak Cliff comparison community (148 ppm).

CONCLUSIONS

1. Evidence indicates that people were exposed to site contaminants in the past, but available data indicate that people in the residential areas currently are not being exposed to site contaminants at levels that would be expected to cause adverse health effects.

2. The primary route of exposure associated with elevated blood lead levels among West Dallas children in the early 1980s was inhalation of lead-contaminated air emitted from the RSR smelter. Incidental ingestion of contaminated soil and dust was a secondary source of exposure. Air emissions were eliminated as a source of exposure in February 1984 when the smelter closed. The 1984/85 remedial efforts, as well as removal efforts initiated in 1993, decreased the significance of soil and dust as routes of exposure.

3. Blood lead data collected in 1982 indicate that significant exposure to lead was occurring among West Dallas children. Recent data from City of Dallas Walk-in Clinics and the EPSDT program indicate that the percentage of children with elevated blood lead levels ($\geq 10\mu g/dL$) was comparable to the percentage of children with elevated blood lead levels from other parts of Dallas. Interpreting these results with respect to the RSR smelter is difficult because these data were not collected on a randomly-selected sample and do not include exposure information. The 1993 Biologic Indicators of Exposure Study, which did include a randomly-selected group of children from the West Dallas area and also included environmental sampling and exposure data, reported that blood lead levels of children in West Dallas were similar to those observed among a comparison group of children from the Oak Cliff neighborhood. Although 10 of the 53 children from the area closest to the smelter had elevated blood lead levels, the study did not find an association between these blood lead concentrations and lead concentrations in residential soil, home dust, or other environmental media.

4. Most areas of the site that have not been remediated are not easily accessible to the general public.

5. We were unable to evaluate past exposures to arsenic and cadmium.

RECOMMENDATIONS

Cease/Reduce Exposure Recommendations

1. Continue to limit unauthorized access onto unremediated areas of the site. Repair crushed fence and place warning signs at the dump/slag disposal area on Claiborne Street (site 4).

2. Continue to provide ongoing blood lead testing for residents of West Dallas.

Site Characterization Activities

1. Characterize sediment and surface water contamination in Fish Trap Lake, drainage ditches, and other low-lying areas of the site.

2. Characterize the fate of contaminants leaving unremediated areas of the site via drainage and surface water runoff.

3. Further evaluate the potential sources of exposure to lead among the 10 children from Area 1 identified with elevated blood lead levels.

Health Activities Recommendation Panel (HARP) Recommendations

In accordance with the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980, as amended,the data and information developed in the RSR Corporation, Inc. Public Health Assessment have been evaluated by the Agency for Toxic Substances and Disease Registry's Health Activities Recommendations Panel (HARP) and TDH for appropriate follow-up activities. HARP has determined that TDH and ATSDR should work with the City of Dallas Department of Health and Human Services to provide community education with specific attention to answering the community's health concerns. Although exposure to lead has occurred in the past, based on the nature of health outcomes associated with contaminants at this site, we are unable to recommend additional follow-up activities at this time.

Public Health Action Plan

1. The City of Dallas Department of Health and Human Services is continuing to provide blood lead screening for residents.

2. The City of Dallas provided educational follow-up and counseling about the sources of lead to the families of children identified in the 1993 Biologic Indicators of Exposure to Lead Study.

3. TDH and ATSDR will work with the City of Dallas to provide community education with specific attention to answering the community's health concerns.

4. TDH and ATSDR will continue to evaluate this site as more data become available.

PREPARERS OF THE REPORT

Kathryn A. Evans, M.P.H.
Epidemiologist
Health Risk Assessment and Toxicology Program

John F. Villanacci, Ph.D.
Director
Health Risk Assessment and Toxicology Program

Nancy B. Ingram
Public Health Technician
Health Risk Assessment and Toxicology Program

Keith E. Hutchinson, M.A.
Education Specialist
Health Risk Assessment and Toxicology Program

ATSDR Regional Representative:

George Pettigrew, P.E.
Senior Regional Representative
ATSDR - Region 6

ATSDR Technical Project Officer

William Greim
Environmental Health Scientist
Division of Health Assessment and Consultation

CERTIFICATION

The RSR Corporation Public Health Assessment was prepared by the Texas Department of Health under the a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Health Assessment was initiated.

William Greim, M.S., M.P.H.
Technical Project Officer
Division of Health Assessment and Consultation (DHAC)
ATSDR

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

for Robert C. Williams, P.E., DEE
Director, DHAC, ATSDR

REFERENCES

1. BCM Memorandum from Joe G. Moore, Jr. to Debbie Straker concerning RSR Lead Smelter. November 12, 1990.

2. U.S. Environmental Protection Agency. RSR Superfund Site, Planned Home Sampling Investigations. June 1993.

3. U.S. Environmental Protection Agency. Site Assessment Report for RSR Smelter (West Dallas Lead) Site, Dallas, Dallas County, Texas. Prepared by Ecology and Environment, Inc. September 10, 1993.

4. von Lindern IH. Evaluation of Historical Lead Exposures to the Children of West Dallas, Texas. Summary Report prepared for Frederick C. Baron and Associates, Dallas, Texas. Undated.

5. U.S. Environmental Protection Agency. Administrative Order On Consent. Docket Number CERCLA VI-5-83.

6. U.S. Environmental Protection Agency. Dallas Lead - Historical Perspective. September 1991.

7. U.S. Environmental Protection Agency/Texas Water Commission. West Dallas Lead Update. December 5, 1991.

8. U.S. Environmental Protection Agency. Superfund Newsline, A U.S. EPA Region 6 Newsletter for West Dallas Residents. Winter 1993-94.

9. Texas Department of Health Record of Communication. Telephone conversation between Kathryn Evans and Carlos Sanchez, U.S. Environmental Protection Agency concerning remedial activities. November 4, 1994.

10. U.S. Census Bureau. Census of the population. U.S. Government Printing Office, Washington, D.C., 1985.

11. National Library of Medicine. Toxicology Data Network, Toxic Release Inventory 1987, 1988, 1989, 1990, 1991. Bethesda, Maryland; NLM, March 1994.

12. Texas Department of Health Record of Communication. Telephone conversation between Kathryn Evans and John Meyer, Texas Natural Resource Conservation Commission concerning water supply wells. March 31, 1994

13. Texas Water Commission. Public Water Supply Well Search Near TDH Study Area. March 31, 1994

14. Texas Department of Health Record of Communication. Telephone conversation between Kathryn Evans, Texas Department of Health, and the City of Dallas Water Quality Investigator concerning RSR West Dallas Lead. March 3, 1994.

15. The Dallas Morning News. 1992-93 Texas Almanac. Houston, Texas: Gulf Publishing Company, 1992. ISBN 0-914511-15-7.

16. Texas Department of Health Record of Communication. Telephone conversation between Kathryn Evans, Texas Department of Health, and the City of Dallas Watershed Management concerning storm water and water runoff. March 3, 1994.

17. U.S. Environmental Protection Agency. Report of the Dallas Area Lead Assessment Study. Lead Smelters Study Group, Office of Toxics Integration, February 1, 1983

18. Agency for Toxic Substances and Disease Registry (ATSDR) Memorandum from Environmental Health Scientist, Community Health Branch, to George Pettigrew, ATSDR Region 6, concerning Health Consultation: West Dallas Lead/RSR Smelter (6P08), Dallas, Texas. June 17, 1992.

19. City of Dallas Department of Health and Human Services. Facsimile communication from Douglas A. Maines to Kathryn Evans of the Texas Department of Health concerning blood lead levels. March 7, 1994.

20. City of Dallas Department of Health and Human Services. Memorandum from Douglas A. Mains to Kathryn Evans of the Texas Department of Health concerning blood lead levels. December 7, 1993.

21. Texas Department of Health. Interoffice memorandum from Jean Brender to David R. Smith concerning elevated blood lead levels for Dallas County and the City of Dallas. August 6, 1993.

22. Biologic Indicators of Exposure to Lead RSR Smelter Site, Dallas Texas. City of Dallas Department of Health and Human Services and Agency for Toxic Substances and Disease Registry. Draft for Peer Review. October 1994.

23. Texas Department of Health Record of Communication. Telephone conversation between Kathryn Evans, Texas Department of Health, and ATSDR Region 6 concerning EPA/RSR Open House. March 14, 1994.

24. U.S. Environmental Protection Agency Office of Solid Waste and Emergency Response. On Scene Intermittent Bulletin Volume 3, Number 2. Publication 9200.5-1021. Summer 1992.

25. U.S. Environmental Protection Agency. West Dallas Project Phase II Summary (7/27/92 - 4/30/93). Undated.

26. U.S. Environmental Protection Agency. Soil sampling for West Dallas database dated December 15, 1993, and accompanying memorandum of explanation. December 16, 1993.

27. U.S. Environmental Protection Agency. Superfund Newsline, A U.S. EPA Region 6 Newsletter for West Dallas Residents. Spring 1994.

28. U.S. Environmental Protection Agency. Memorandum from Kenneth Clark, On-Scene Coordinator, to Russell F. Rhoades, Environmental Services Division, concerning Request for a Twelve-Month Exemption for the Removal Action at the West Dallas (RSR) Lead Site. September 23, 1992.

29. Texas Natural Resource Conservation. West Dallas Lead Neighborhood Survey and Yard Sampling, Phase 2 Final Report. October 22, 1993.

30. Agency for Toxic Substances and Disease Registry (ATSDR) Record of Activity. Health Consultation concerning RSR Smelter Site, Dallas, Dallas County, Texas. March 9, 1994.

31. City of Dallas Health and Human Services. City of Dallas Air Pollution Control Environmental Follow Up High Blood Lead Levels. December 20, 1993.

32. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Lead. Atlanta: ATSDR, April 1993.

33. Agency for Toxic Substances and Disease Registry. The nature and extent of lead poisoning in children in the United States: a report to Congress. Atlanta: ATSDR, 1988.

34. Hernberg S, Nikkanen J, Mellin G, et al. Deltal aminolevelinic acid dehydrase as a measure of lead exposure. Archives of Environmental Health 1970; 21:140-5.

35. Chisholm JJ, Thomas DJ, Hamill TG. Erythrocyte porphobilinogen synthesis activity as an indicator of lead exposure to children. Clinical Chemistry 1985; 31:601-5.

36. Roels HA, Lauwerys R. Evolution of dose-effect and dose-response relationships for lead exposure in different Belgian population groups (fetus, child, adult men and women). Trace Elem. Med. 1987; 4:80-7.

37. Roels HA, Buchet JP. Impact of air pollution by lead on heme biosynthetic pathway in school-age children. Archives of Environmental Health 1976; 31:310-6.

38. Angle CR, McIntire MS. Low level lead and inhibition of erythrocyte pyrimidine nucleotide. Environmental Research 1978; 17:296-302.

39. Angle CR, McIntire MS, Swanson MS, et al. Erythrocyte nucleotides in children increased blood lead and cytidine triphosphate. Pediatric Res. 1982; 16:331-4.

40. Alvares AP, Kapelner S, Sassa S, et al. Drug metabolism in normal children, lead poisoned children, and normal adults. Clinical Pharmacology 1980; Ther.; 17:179-183.

41. Saenger P, Markowitz ME, Rosen JF. Depressed excretion of 6B-hydroxycontisol in lead-toxic children. Journal of Clinical Endocrinal Metabolism 1984; 58:363-7.

42. Needleman HL, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. New England Journal of Medicine 1979; 300:689-95.

43. Bellinger D, Sloman J, Leviton A, Rabinowitzm, Needleman H, Waternaux C. Low-level exposure and children's cognitive function in the preschool years. Pediatrics 1991; 87:219-27.

44. Centers for Disease Control. Preventing Lead Poisoning in Young Children, A Statement by the CDC. U.S. Department of Health and Human Services, Public Health Service, October 1991.

45. Krignam MR, Bouldin TW, Mushak P. Lead. In: Spencer PS, Schaumburg HH, eds. Experimental and clinical neurotoxicology. Baltimore: Williams and Wilkins Company 1980.

46. Landrigan P. Current issues in the epidemiology and toxicology of occupational exposure to lead. Environmental Health Perspectives 1990; 89:61-6.

47. Mantere P, Hanninen H, Hernberg S, Luukkonen R. A prospective follow-up study on psychological effects in workers exposed to low levels of lead. Scand. J. Work Environmental Health 1984; 10:43-50.

48. Sullivan, JB Jr, Krieger, GR, eds. Hazardous Materials Toxicology: Clinical Principles of Environmental Health. Baltimore: Williams and Wilkins, 1992.

49. Harlan WR. The relationship of blood lead levels to blood pressure in the US population. Environmental Health Perspectives 1988; 78:9-13.

50. Pirkle JL, Schwartz J, Landis JR, et al. The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. American Journal of Epidemiology 1985; 121:246-58.

51. Pocock SJ, Shaper AG, Ashby D, et al. Blood lead concentrations, blood pressure, and renal function. British Medical Journal 1984; 289:872-4.

52. Pocock SJ, Shaper AG, Ashby D, et al. Blood lead and blood pressure in middle-aged men. In: Lekkas TD, ed. International Conference: Heavy metals in the environment, vol. 1. Athens: CEP Consultants, Ltd., 1985:303-5.

53. Agency for Toxic Substances and Disease Registry. Analysis Paper: Impact of Lead-Contaminated Soil on Public Health. U.S. Department of Health and Human Services, May 1992.

54. Schilling R, Bain RP. Prediction of children's blood lead levels on the basis of household specific lead levels. American Journal of Epidemiology 1989; 128(1):197-205.

55. Weitzman M, Aschengrau A, Bellinger D, Jones R, Hamlin JS, Beiser A. Lead-Contaminated Soil Abatement and Urban Children's Blood Lead Levels. Journal of the American Medical Association, Vol. 269, No. 13, April 7, 1993.

56. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Cadmium. Atlanta: ATSDR, April 1993.

57. Davison AG, Fayers PM, Taylor AJ, et al. Cadmium fume inhalation and emphysema. Lancet 1988; (March 26):663-667.

58. Glaser U, Kloppel H, Hochrainer D. Bioavailability indicators of inhaled cadmium compounds. Ecotoxicol Environ Safety 1986; 11:261-271.

59. Lauwerys RR, Bernard AM, Buchet JP, Roels HA. Assessment of the health impact of environmental exposure to cadmium: Contribution of the epidemiologic studies carried out in Belgium. Environmental Research 1993; 62;200-206.

60. Buchet JP, Lauwerys RR, Roels HA, Bernard AM, Braux P, Claeys F, Ducoffre G, DePlaen P, Staesser J, et. al. Renal effects of cadmium body burden of the general population. Lancet 1990; 336;699-700.

61. Jarup L, Elinder CG, Spang G. Cumulative blood-cadmium and tubular proteinuria: A dose-response relationship. International Archives of Occupational and Environmental Health 1988; 60:223-229.

62. Nogawa K, Honda R, Kido T, et al. A dose-response analysis of cadmium in the general environment with special references to total cadmium intake limit. Environmental Research 1989; 48:7-16.

63. IRIS. Integrated Risk Information System. U.S, Environmental Protection Agency, Washington, DC. October 8, 1990

64. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. Atlanta: ATSDR, October 1991.

65. Baker EL Jr, Hayes CG, Landrigan PJ, Handke JL, Leger RT, Houswarth WJ, Harrington JM. A nation\wide survey of heavy metal absorption in children living near primary copper, lead, and inc smelters. American Journal of Epidemiology 1977;106:261-273.

66. Cebrian ME, Albores A, Aguilar M, et al. Chronic arsenic poisoning in the north of Mexico. Hum Toxicol 1983; 2:121-133.

67. Huang YZ, Qian XC, Wang GQ, et al. Endemic chronic arsenicism in Xinjiang. Chin Med J (Eng) 1985; 98:219-222.

68. Tseng WP, Chu HM, How SW, et al. Prevalence of skin cancer in an epidemic area of chronic arsenicism in Taiwan. J Natl Cancer Inst 1968;40:453-463.

69. Tseng WP. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic Environ Health Perspect 1977; 19:109-119.

Other Documents Reviewed

Agency for Toxic Substances and Disease Registry. Public Health Assessment Guidance Manual. Lewis Publishers: Boca Raton, 1992.

Congress of the United States, House of Representatives, letter from Eddie Bernice Johnson to Mr. Larry Wright of the U.S. Environmental Protection Agency concerning the January 28, 1993 Texans United letter. June 4, 1993.

U.S. Environmental Protection Agency. RSR Lead Smelter Work Plan, Site Discovery, WA # 19-6JZZ. August 23, 1991.

U.S. Environmental Protection Agency. Sampling Inspection Report, March 14, 1988.

U.S. Environmental Protection Agency. Superfund Newsline, A U.S. EPA Region 6 Newsletter for West Dallas Residents. December 1993/January 1994.

APPENDICES

Appendix A - Figures

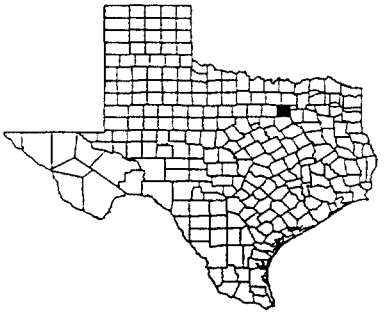
Figure 1. RSR Site Location

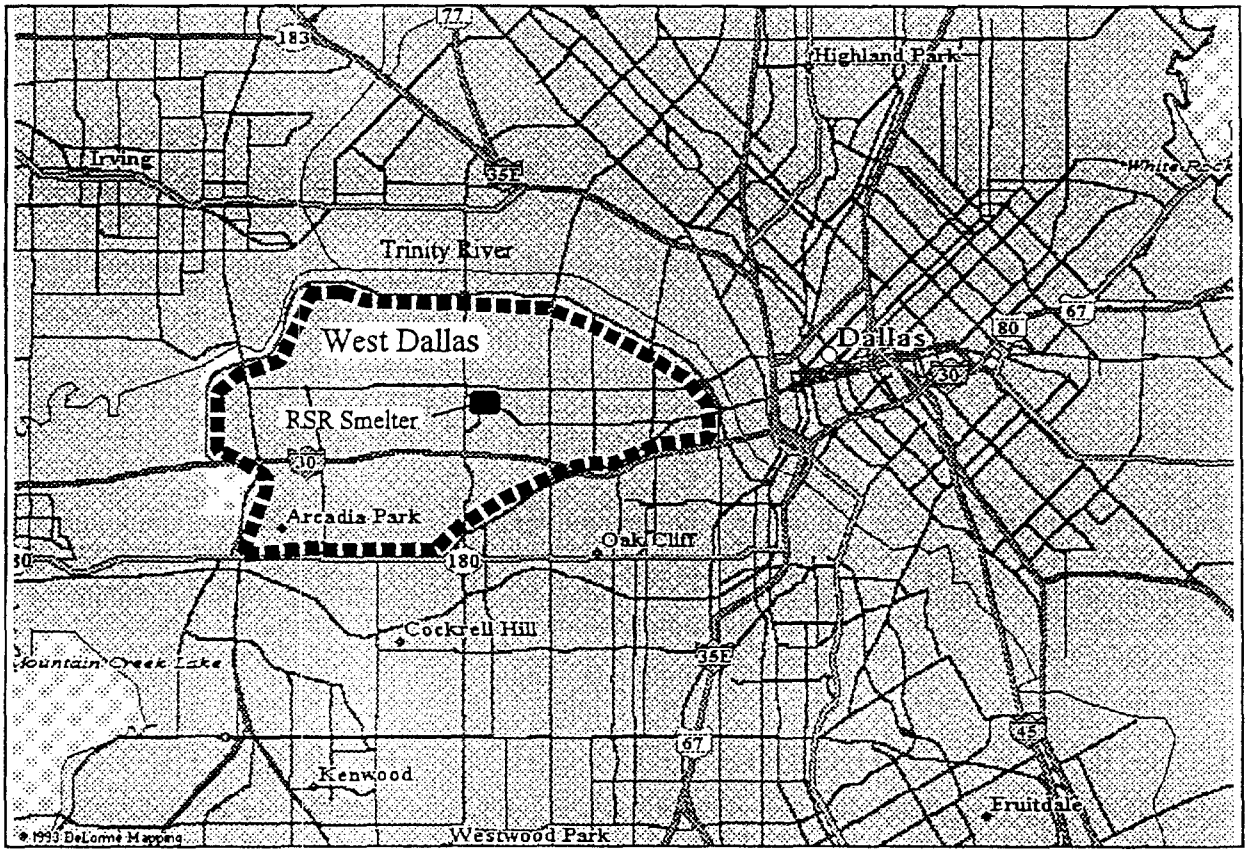
Figure 2. Detailed Site Map

Figure 3. 1993 Blood Lead Study

Figure 4. Isopleth Map Based on 1982 Soil Sampling

Figure 1. RSR Site Location





(dotted line indicates boundary of site)

Figure 2. Detailed Site Map

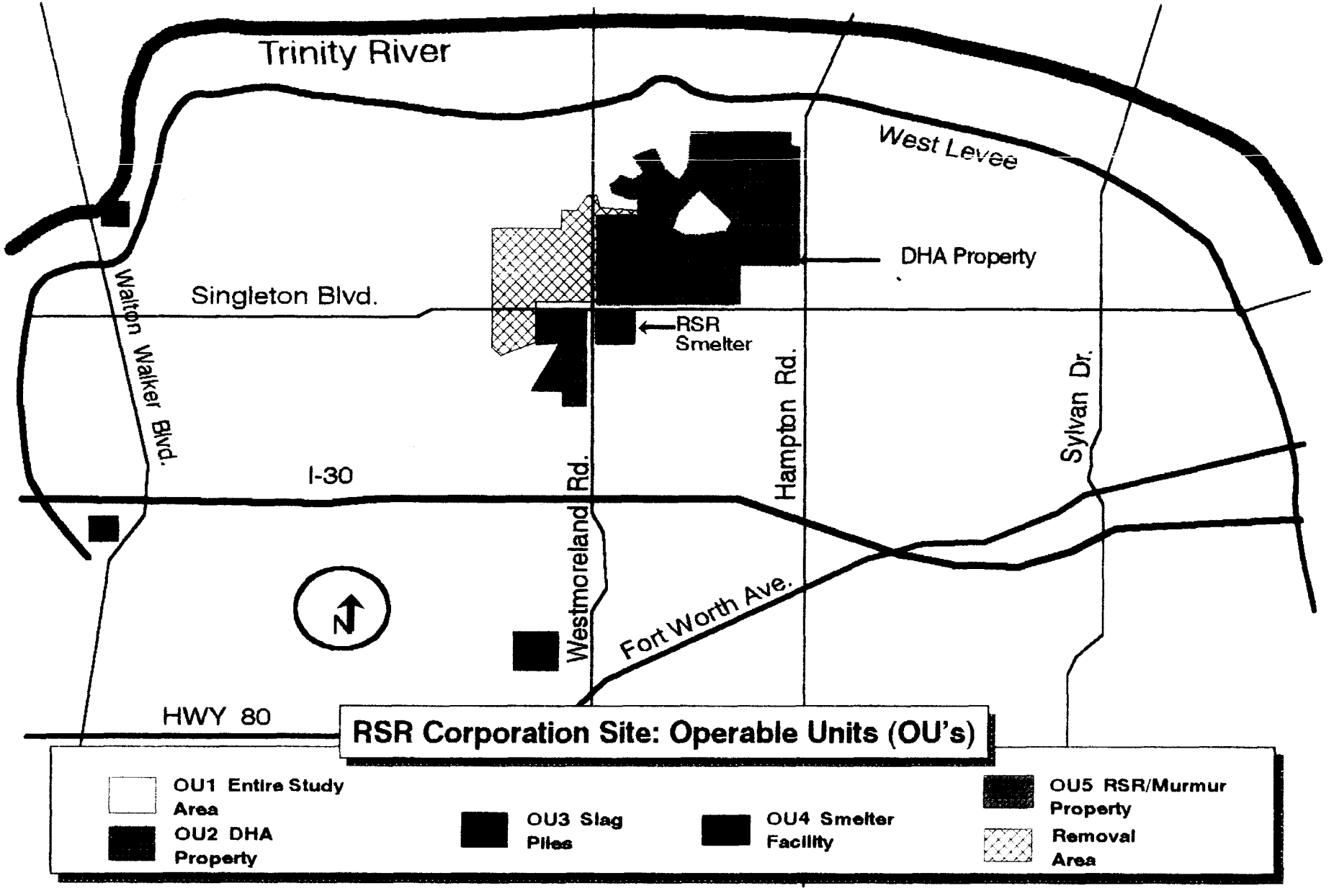


Figure 3. Blood Lead Study

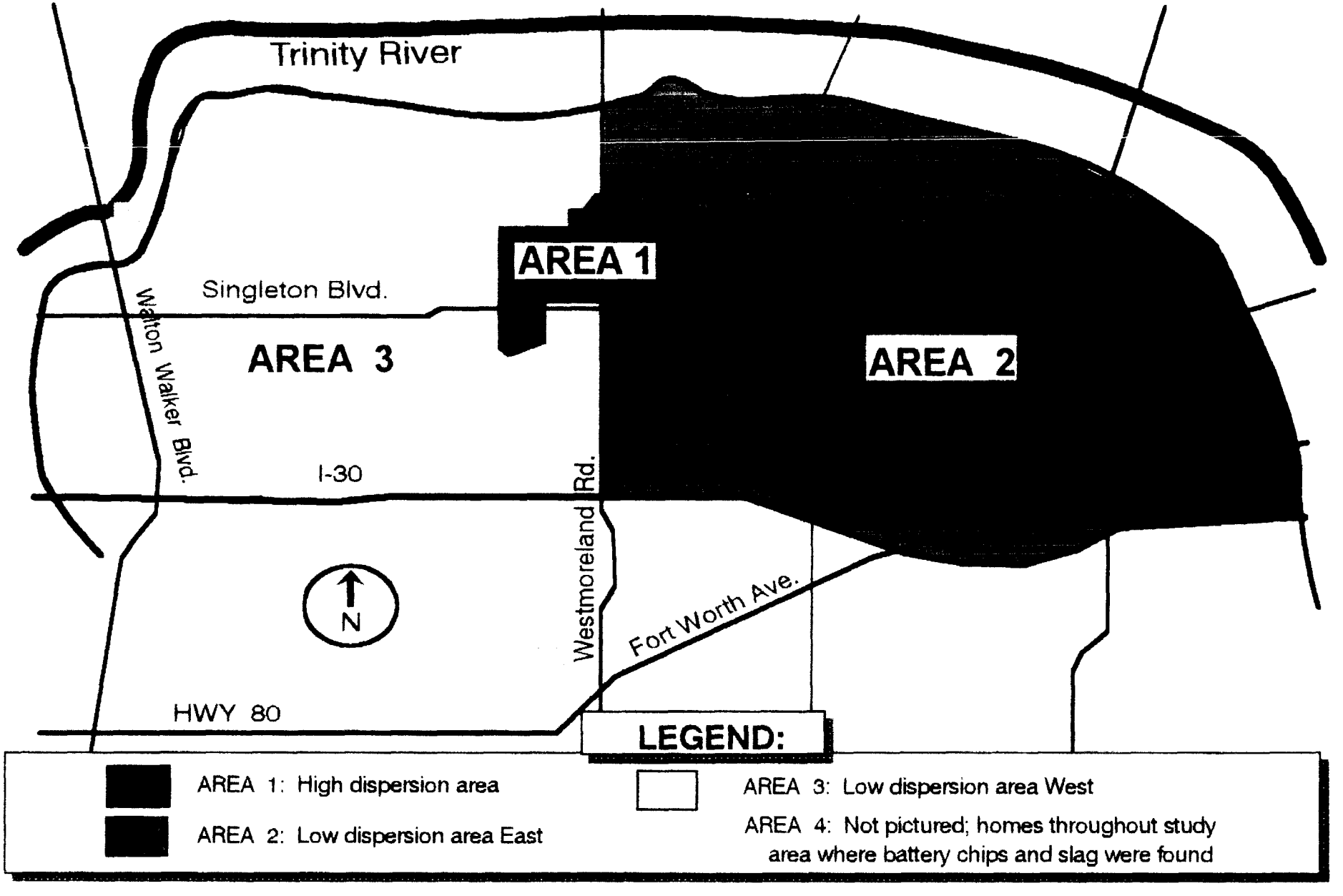
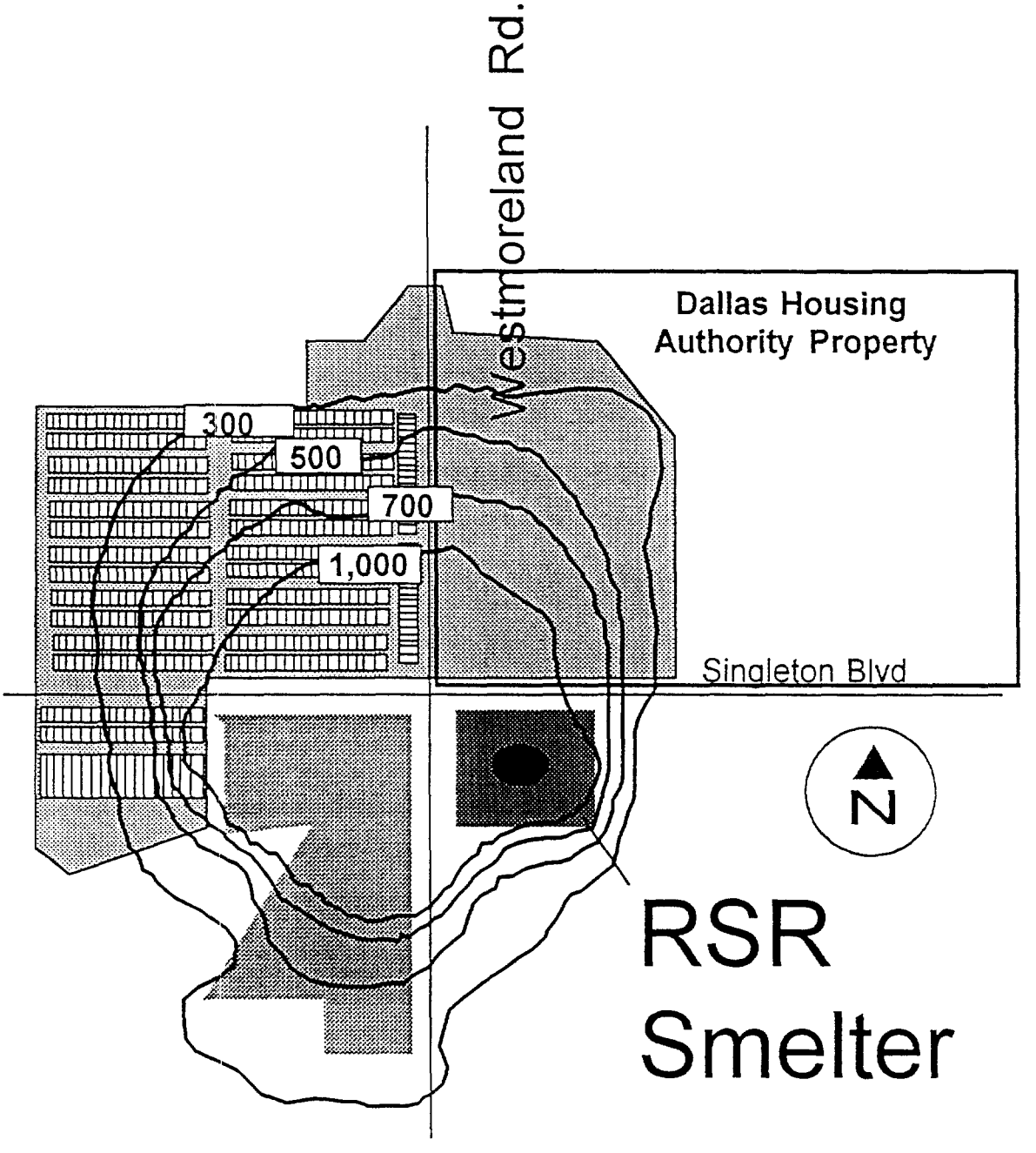


Figure 4. Isopleth Map

(Based on 1982 Soil Sampling)



Map is not to scale.
Numbers on isopleth plots (concentric circles) represent soil lead levels in parts per million.

Appendix B - Public Comments and Responses

Comments Received during the Public Comment Period
and Texas Department of Health Response

(Note to the reader: Page and paragraph numbers are quoted from the "Public Comment" document and may differ from the page and paragraph numbers of this document.)

Commentor 1

Comments mentioned in the December 1994 public meeting:

1. **The Health Assessment is based on a two-hour drive through the community with no participation by local residents or their health care providers.**

ATSDR and TDH staff spent two and a half hours driving and walking through the community during a "site visit" guided by the EPA Project Manager. This site visit was conducted specifically to examine the physical layout and characteristics of the area and to learn more about EPA's remedial efforts. (Clarification of the purpose of the site visit has been added to the "Site Visit" section of the Public Health Assessment (PHA) on page 3).

Input from local residents is very important to the health assessment process. To identify community concerns at the site, TDH staff attended EPA-sponsored Open Houses and public meetings, talked with individual residents, and maintained contact with the EPA Community Relations Coordinator and the Community Outreach Officer (a contractor hired by EPA to serve as a community liaison) for the RSR Site. We also reviewed the October 1993 Resolution by members of the TNRCC's West Dallas Citizen's Advisory Committee and reviewed newspaper coverage of the site. A section of the PHA focuses specifically on community concerns.

We reviewed data collected by a number of local health care providers including blood lead data collected through the Early Periodic Screening, Diagnosis, and Testing (EPSDT) program and at City of Dallas Walk-in Clinics. A training effort for physicians was undertaken in September of 1992. Approximately 20 health care providers participated in the effort and were provided educational information relating to the chemicals of concern at the site. Six of the participants practiced in the West Dallas area.

2. **A conclusion about no risk from dust is based on only 26 samples, one of which is found to have 3,800 ppm of lead in it.**

In-home environmental sampling, including dust sampling, is offered to all individuals with elevated blood lead levels identified at City of Dallas Walk-in Clinics (children with $\geq$ 15 μg/dL and adults with levels $\geq$ 25 μg/dL). We reviewed data collected between 1991 and 1993 when 21 such followups were conducted. Dust samples were collected in the homes of these 21 individuals from either the floor or the window sill, or both. It is correct that one of the homes was found to have a lead concentration of 3,800 ppm in dust from a window sill; however, only one other home had a lead concentration in dust (321 ppm) that was above the City's screening level. These data therefore indicate that indoor dust apparently was not a primary source of exposure among these 21 individuals with elevated blood lead levels.

The most recent in-home sampling conducted in West Dallas was collected as part of the 1993 Biological Indicators of Exposure Study. This study included 386 randomly-selected children and provided much more comprehensive data than the Walk-in Clinic data. As part of this

study, **576** dust samples were collected from children's bedrooms and the entryways of homes in West Dallas. In addition, **323** dust samples were collected from children's bedrooms and the entryways of homes in a comparison community.

The average concentration of lead in dust samples collected in children's bedrooms ranged from 80 ppm (in the area of West Dallas where chips and slag had been identified) to 146 ppm (in the area nearest the site). The average concentration of lead in dust collected from the entryways of homes ranged from 81 ppm (in the chips and slag area) to 205 ppm (in the comparison community).

ATSDR analyzed the relationship between lead concentrations in dust from **each** area of the house to determined whether the lead levels were related to the concentrations of lead in children's blood. This analysis was conducted separately for each study area. Based on ATSDR's analysis, there was a (statistically significant) correlation between the concentration of lead in dust found in children's bedrooms and children's blood lead levels in both the area closest to the smelter and the "eastern low deposition area," but not in any of the other areas of the site. When the same analysis was conducted for lead in entryway dust and blood lead levels, a (statistically significant) correlation between lead in dust and blood lead levels was found among children living in the "eastern low deposition area," but not among children living in any other areas of the site.

For each home, ATSDR did an additional analysis of the **average** level of lead found in **all types** of indoor dust samples to determine whether the level correlated with the blood lead levels of children living in the home. The analysis was based on the average level of dust found in the child's bedroom, the living area, and the entryway of the house. Results indicated that there was a (statistically significant) correlation between average lead concentrations in indoor dust and the blood lead levels of children living in the homes only in the "eastern low deposition area." There was not a correlation between the average level of lead found in dust and the level of lead detected in children's blood among children who lived in any other part the West Dallas study area.

In another way of analyzing the effect of environmental exposures on blood lead levels of children, ATSDR used a statistical model (linear regression modelling) to simultaneously account for the effects of numerous variables (including environmental measurements of household dust, soil, interior and exterior paint, age of child, play habits, hygiene practices, and other variables) on blood lead levels. This type of analysis can provide the best indication of whether a single factor has an independent effect on an outcome; in other words, it gives the best indication of whether dust represents a significant exposure route. Six models were created -- one for each study area and one for all the areas combined. The models did not find a (statistically significant) relationship between the levels of interior dust found in homes and the blood lead levels observed in children in any area. According to the findings of the study, interior dust, therefore, does not represent a significant exposure pathway.

We were unable to ascertain the concentrations of lead in interior dust when the smelter was operating to determine whether indoor dust constituted a significant source of exposure in the

past. The 1982 Lead Smelters Study reports that indoor dust samples were collected, but that they were not analyzed in the report because lead levels in dust were "inexplicably low." We were unable to determine whether there was a problem with the collection or analysis of these samples or whether indoor dust samples were indeed very low at the time samples were collected in the past. One possible explanation is that at the time the samples were collected in 1982, residents had already taken measures to clean or remove contaminated dust from the areas of their homes that were sampled.

3. The Health Assessment makes no attempt to explain why the Boy's Club property is being recontaminated after supposedly being cleaned up.

TDH and ATSDR are aware of several theories regarding the re-accumulation of lead at the Boys/Girls Club property in the past. None of the theories of past contamination can be proved or disproved, but the most likely explanation is that the contamination found on the property after it had been cleaned up came from contaminated roofing material left on the ground after the building was re-roofed.

According to EPA, high levels of lead were found on Dallas Housing Authority (DHA) buildings in the vicinity of the smelter that had flat roofs. According to EPA, the lead content of roofing material on 167 buildings in the area was tested. The concentration was high in only 20 buildings, all of which were located close to the smelter and had flat roofs. EPA theorizes that since the tar-like material used on flat roofs expanded and became sticky during hot weather, it trapped lead particles that were emitted from the smelter over a period of years, prior to 1984 when stack emissions were ceased.

Air monitoring has been conducted from the roof of the Boys/Girls Club building since early in 1984 and there is no evidence that air emission of lead have contributed to the contamination of soil at the Boy's Club since the smelter stopped operating. The only time that lead concentrations in air have exceeded national regulatory standards occurred early in 1984 before the smelter stopped operating.

The DHA recently conducted sampling on the Boys/Girls Club property to determine the current levels of lead in soil. Samples collected in the grassy areas of the property did not contain any concentrations of lead that exceeded EPA's cleanup level. DHA conducted additional sampling in the right-of-way between the road and the sidewalk near Westmoreland Road and Singleton Avenue and found elevated levels of lead that were apparently associated with battery chips and slag. This material may have been used as part of the roadbase since it was found only in the right-of-way and was detected down to a depth of approximately 26 inches. Although the potential for exposure to this material is minimal under ordinary circumstances, DHA removed all of the contaminated material that was identified.

4. The Health Assessment "admits that using the EPA's cleanup guidelines, a portion of the West Dallas children will still be subject to lead levels in their blood above the danger mark of 10 deciliters."

The pathways section of the health assessment states that human exposure to contaminants in soil is occurring and may occur in the future and that the exposed population includes residents living on properties with soil lead levels below the 500 mg/kg action level for lead; this section does not evaluate the public health significance of exposure to lead in soil at these levels. The health assessment states that exposures to soil containing 500 mg/kg is not expected to produce adverse health effects.

The regression model predicting that a soil lead concentration of 500 mg/kg would result in blood lead level of 10.7 μg/dL assumes exposure to an average soil lead concentration of 500 mg/kg on a daily basis. Although soil was not removed from yards in West Dallas where randomly-selected samples were below 500 mg/kg, the *average* soil lead concentration in those yards is in all likelihood much lower than 500 mg/kg. Thus, the regression model would predict blood lead levels below 10 μg/dL. TDH and ATSDR will review available post-remedial sampling to determine if the concentration of lead remaining in the soil constitutes a threat to public health.

For the remediated yards, a clean-up standard of 500 mg/kg assures that the average concentration of lead remaining in the soil will be significantly below 500 mg/kg. Since clean fill has been used to replace the contaminated soil, the average levels to which residents will be exposed are likely well below 200 mg/kg. EPA's studies show that the average soil lead level of remaining soil in West Dallas is less than 120 ppm. In addition, EPA's sampling shows that almost 90 percent of the children found to have elevated blood lead levels live in homes with soil lead levels less than 250 ppm.

5. There is no modeling to help understand past exposures.

Our estimations of past exposures are based primarily on actual blood lead levels measured in West Dallas children in the past (particularly the 1982 Lead Study which provided the earliest comprehensive blood lead testing data). We believe that since these data reflect the levels of exposure that actually occurred among children in the neighborhoods near the smelter, they provide more reliable estimates of exposure than we could get from environmental modelling.

The blood lead testing data collected prior to 1982 indicate that exposure levels were higher prior to 1982. While these data are limited and do not provide extensive information on the extent of exposure, the findings are consistent with historical records which indicate that few emissions controls had been implemented at the smelter prior to 1982.

Other comments mentioned in a critique of the Preliminary Public Health Assessment:

- **Incomplete or Inaccurate Data**

  a) TDH did not argue against cleaning up lead-contaminated indoor dust.

  b) The Preliminary Public Health Assessment addressed areas of West Dallas presenting the most immediate potential routes for exposure and the areas for which sampling data were available. While most of the unremediated areas (DHA property, slag piles/disposal areas, and the smelter site) are not easily accessible to the general public, TDH was unable to quantitatively evaluate potential health effects associated with exposures in these areas because sampling data were not available. The Preliminary Health Risk Assessment recommended characterizing the fate of contaminants leaving unremediated areas of the site via surface water; the Public Health Action Plan recommended evaluation of other operable units of the site as additional data become available.

  c) Based on available information, TDH reported that the practice of using contaminated fill material in yards ended in the 1960s. TDH will note that improper disposal of lead waste continued into the 1970s. This discrepancy will not alter any conclusions regarding current potential exposure.

  d) The report mentions that people live just south of the slag pile area on Westmoreland Road. Although environmental sampling data from this area were not available for review, the report notes that the slag pile is not easily accessible to the public. The public health significance of this operable unit will be determined in future reports as environmental sampling data become available.

- **Contradictions in Text.**

  a) Lead is classified as a B2 carcinogen (probable human carcinogen) based on studies which found that lead produces tumors in laboratory animals. Lead has not been shown to be carcinogenic in humans.

  b) The referenced kidney problems that occurred among smelter workers years after exposure were associated with chronically elevated blood lead levels in the range of 50 to 60 μg/dL or higher. These levels are higher than those documented among West Dallas residents. Studies of individuals who have experienced lead levels below approximately 20 to 30 μg/dL generally do not indicate any additional risk for developing illnesses years after exposure.

  c) Since post-remedial sampling data and other information were not available for review, the site was classified as an "indeterminate public health hazard" in the Preliminary Public Health Assessment. The evidence available for review indicates that people were exposed to site contaminants in the past at levels that

could be associated with adverse health effects, but the available evidence does not indicate that people are currently being exposed to levels of contaminants expected to cause adverse health effects. Therefore, currently the residential area of the site constitutes "no apparent public health hazard." Additional information and sampling data from other areas of the site will be reviewed as they become available.

- **Ignores on-going sources of industrial lead emissions.**

  a) Murmur does continue to report 500 lbs. of lead per year in air emissions to EPA. These emissions are an estimate by Murmur of the emissions associated with its ongoing reclamation and recycling activities. Murmur reported these estimated releases to EPA under the category of environmental losses between 11 and 500 pounds per year although actual amounts may be much less than 500 pounds. According to EPA, in 1993 the company estimated that less than 100 pound of lead per year were emitted to the environment (air, water, soil, etc.). Air pollution monitors across the street from Murmur have not detected any lead pollution from the facility since 1984 when the smelter closed (please see answer to question 3 above).

  b) We searched the TRI database for all emissions data in the West Dallas area for the five most recent years for which data is available (1987-91). There was no information listed for Refinery Castings, Inc. We have called TRI database managers to determine why this facility was not included in the report.

  c) According to available environmental and blood lead testing data, lead emissions in West Dallas currently are not causing on-going health problems. It is important to note that we have no evidence that children in West Dallas who continue to have elevated blood lead levels are being exposed to elevated levels of lead in soil (see answer to comment 2 above).

Commentor 2

Your letter requests that the EPA and ATSDR buy out and relocate residents living in the most polluted area of West Dallas. Although ATSDR and TDH are not authorized to buy properties, they can make recommendations for reducing or eliminating human exposure. The purpose of the ATSDR's Public Health Assessments is to evaluate the human health impact of hazardous waste.

Based on the environmental data and blood lead testing data that we have reviewed for the RSR Public Health Assessment, we believe that soil removal in the residential areas has been effective at reducing human exposure to lead contamination. The greatest source of lead exposure was eliminated when the smelter stacks stopped operating in 1984 and subsequent soil cleanup efforts eliminated the primary source of lead left behind. Lead from the smelter is no longer a major source of exposure in the residential areas of West Dallas.

Blood lead testing provides the best information about actual exposures that people are currently experiencing. The most recent comprehensive testing was conducted by the City of Dallas in 1993. It showed that approximately 8.5 percent of children in West Dallas have elevated blood lead levels. This number is similar to the percentage of children in Texas as a whole who had elevated blood lead levels in 1993 (9.4 percent).

You mentioned in your letter that you are particularly concerned about the area located near the Dallas Housing Authority (DHA) property that "was granted relocation and reconstruction." Relocation of residents and reconstruction of DHA buildings was not required because of contamination. The DHA buildings are being demolished by the Department of Housing and Urban Development as part of their plans to renovate public housing.

The average blood lead level of children living in the community adjacent to DHA and immediately downwind of the RSR smelter was 7.0 μg/dL in 1993, compared with an average level of 20.1 μg/dL detected in 1982. In the early 1980s, ten percent of children had blood lead levels higher than 30 μg/dL. In contrast, today not a single child has a blood lead level this high. Only four of the 305 children from West Dallas who were tested in the recent study had blood lead levels over 15 μg/dL and none had levels higher than 18 μg/dL.

Based on the information reviewed, we concluded that the soil cleanup effort has been successful. The children who continue to have elevated blood lead levels are probably being exposed to a combination of the many sources of lead common in urban environments. Some of these sources include lead from paint, dietary sources, improperly glazed ceramics and eating utensils, and "take home" lead associated with parents' occupations and hobbies. According to EPA, the current average residential soil lead levels in West Dallas are less than 120 ppm. Ninety percent (90%) of the children with elevated lead levels already live in homes where soil lead levels are below 250 ppm. Further lowering of the soil lead levels therefore will not provide any additional benefit in lowering blood lead levels in children.

We believe that blood lead screening should be continued for West Dallas residents and that educational follow-up and counseling about the sources of lead exposure should be provided to individuals identified with elevated blood lead levels. These services are currently being provided by the City of Dallas.

The Texas Department of Health is also available to provide educational material and to answer any additional questions about health and lead contamination in West Dallas. We would be happy to attend a meeting of the Friendship Homeowners Association for Environmental Justice or to provide you with additional information.

Commentor 3

Your letter mentions that the Public Health Assessment report shows that ongoing lead emissions still exist in West Dallas and suggests that high blood lead levels among West Dallas children and high soil lead concentrations at the Boy's Club are evidence of this ongoing contamination. Below, we are providing additional information regarding these concerns.

Comment #1. ***Ongoing lead emissions still exist.***

The Murmur Corporation has an active reclamation and recycling operation at the site that does not create detectable levels of lead air pollution. Murmur reports estimates of total emissions to EPA's Toxic Release Inventory (TRI). The Public Health Assessment contains a table of industrial emissions compiled from TRI reports between 1987 and 1991. The table shows that Murmur reported releasing 500 pounds of lead per year during this period. Murmur reported these releases to EPA under the category of environmental losses between 11 and 500 pounds per year; thus, the actual amount that was released may be much less than 500 pounds.

According to EPA, in 1993 the company estimated that less than 100 pound of lead per year were emitted to the environment (air, water, soil, etc.) from its processes. Air pollution monitors across the street from Murmur have not detected lead levels in air above the National Amient Air Quality Standard since 1984 when the smelter closed (please see answer to comments #3 below). We will thoroughly evaluate the levels of lead contamination remaining on the smelter property when we address Operable Unit 4, which is currently being assessed and cleaned up by EPA.

Comment # 2. ***High blood lead levels among children in the West Dallas neighborhood provide evidence of ongoing contamination.***

We have no evidence that children in West Dallas continue to have elevated blood lead levels resulting from exposure to lead from the RSR site. Since blood lead testing provides the best information about actual exposures that people are currently experiencing, we evaluated the most recent comprehensive testing, which was conducted by the City of Dallas in 1993. It showed that approximately 8.5 percent of children in West Dallas have elevated blood lead levels (≥ 10 μg/dL). This number is similar to the percentage of children in Texas as a whole who had

elevated blood lead levels in 1993 (9.4 percent). We would expect to see more widespread occurrence of elevated blood lead levels if children were experiencing ongoing exposures to lead from the smelter.

In addition to finding fewer children with elevated blood lead levels, the recent study found that the *average* blood lead level of the children living in the community closest to the former smelter has dropped considerably since the early 1980s when emissions from the smelter were occurring. In 1993, the average blood lead level of children living in this area was 7.0 μg/dL compared with 20.0 μg/dL in 1982. Only four of the 305 children from West Dallas who were tested in the recent study had blood lead levels over 15 μg/dL and none of the children had a blood lead level higher than 18 μg/dL.

Although the recent study identified some children in West Dallas who continue to have elevated blood lead levels, the study did not find a relationship between the elevated blood lead levels and elevated soil lead levels. The children who have elevated blood lead levels most likely are being exposed to a combination of the many sources of lead common in urban environments. Some of these sources include lead from paint, dietary sources, improperly glazed ceramics and eating utensils, and "take home" lead associated with parents' occupations and hobbies.

The environmental data that we have reviewed suggest that the greatest source of lead in the residential areas was eliminated when the smelter stacks stopped operating in 1984. Subsequent soil removal efforts eliminated most of the remaining contamination that constituted an exposure hazard in the residential areas.

Comment #3. ***Recontamination is occurring at the Boys and Girls Club on Singleton Ave.***

TDH and ATSDR are aware of several theories regarding the re-accumulation of lead at the Boys/Girls Club property in the past. None of the theories of past contamination can be proved or disproved, but the most likely explanation is that the contamination found on the property after it had been cleaned up came from contaminated roofing material left on the ground after the building was re-roofed.

According to EPA, high levels of lead were found on Dallas Housing Authority (DHA) buildings in the vicinity of the smelter that had flat roofs. According to EPA, the lead content of roofing material on 167 buildings in the area was tested. The concentration was high in only 20 buildings, all of which were located close to the smelter and had flat roofs. EPA theorizes that since the tar-like material used on flat roofs expanded and became sticky during hot weather, it trapped lead particles that were emitted from the smelter over a period of years, prior to 1984 when stack emissions ceased.

Air monitoring has been conducted from the roof of the Boys/Girls Club building since early in 1984 and there is no evidence that air emission of lead have contributed to the contamination of soil at the Boys/Girls Club since the smelter stopped operating. The only time that lead concentrations in air have exceeded national ambient air quality standards occurred early in 1984 before the smelter stopped operating.

The DHA recently conducted sampling on the Boys/Girls Club property to determine the current levels of lead in soil. Samples collected in the grassy areas of the property did not contain any concentrations of lead that exceeded EPA's 500 ppm cleanup level.
DHA conducted additional sampling in the right-of-way between the road and the sidewalk near Westmoreland Road and Singleton Avenue and found elevated levels of lead that were apparently associated with battery chips and slag. This material may have been used as part of the roadbase since it was found only in the right-of-way and was detected down to a depth of approximately 26 inches. Although the potential for exposure to this material is minimal under ordinary circumstances, DHA removed all of the contaminated material that was identified.

The Public Health Assessment focuses on the public health significance of environmental exposures in the residential areas (EPA's Operable Unit 1). The other areas of the Superfund Site including the smelter facility site, the dumpsites/slag disposal areas, and the Dallas Housing Authority property are being assessed and remediated by EPA in subsequent operable units. We will evaluate the public health implications of contamination in these areas as data become available.

With regard to the residential areas, we have recommended that blood lead screening be continued for West Dallas residents and that educational follow-up and counseling about the sources of lead exposure be provided to individuals identified with elevated blood lead levels. These services are currently being provided by the City of Dallas. TDH also is working with the local health department to provide educational materials and information to health care providers and community members about lead exposure.