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Survey of Health Effects among Residents Adjacent to a National Priority List Site in Ssouthwest Virginia.

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Survey of Health Effects among Residents Adjacent to a National Priority List Site in Southwest Virginia

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ABSTRACT

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The Saltville Waste Disposal Site is a National Priority List site used during the 77- year operation (1895–1972) of an electrolytic chlorine and caustic soda plant. A cross-sectional study was conducted to determine disease prevalence among Saltville Medical Center (SMC) patients. Diseases associated with chronic exposures to mercury, arsenic, and lead were reviewed in patient records and these data were analyzed. Ratios of illness observed at the SMC were compared to health studies of similar environmental exposures and disease prevalence among residents of neighboring communities. Prevalence ratios were calculated for residents and non-residents of Saltville who were SMC patients. Saltville residence accounted for a higher risk of developing the targeted diseases (odds ratio=1.68, 95% confidence interval (1.54, 1.82)). Increased risk was among patients aged 31-45 years, with a history of smoking, and family history of the same disease.

DEDICATION

I dedicate my thesis research to my grandmother, Annette Jordan Pettigrew. She strongly respected and encouraged the pursuit of an academic career and a professional career that would help everybody.

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CHAPTER 1

INTRODUCTION

The Saltville Waste Disposal Site (SWDS) is a National Priority List (NPL) site in Saltville, Virginia (VA). The SWDS is next to the North Fork of the Holston River (NFHR) in Smyth County, Virginia between Saltville, VA and the Allison Gap community (Figure 1). It covers approximately 120 acres of land. Pollutants of concern in the SWDS include mercury, arsenic, lead, and cadmium. An electrolytic chlorine and caustic soda plant operated on the site from 1895 until 1972. Wastewater generated by the plant was disposed of into a waste pond (WP 5). Elemental mercury was a primary waste component in the wastewater. Before regulation in 1970, approximately 100 pounds of elemental mercury were disposed of daily in WP5 and the NFHR (ATSDR 1987). Another waste pond (WP 6) received ammonia-soda waste slurry and received waste if WP 5 overflowed (ATSDR 1987). WP 5 and WP 6 are collectively the SWDS. WP 6 contains buried equipment cleaned about one year after the plant closed. The water used to clean the equipment percolated into the soil of the chlorine plant site. The Olin-Mathieson Corporation, the Water Quality Control Boards of Virginia and Tennessee, and the United States Environmental Protection Agency (U.S. EPA) performed subsequent investigations on the SWDS. These agencies report significant mercury contamination in fish, groundwater, surface water, soils, and sediments (Table 1). The groundwater monitored by Olin-Mathieson in 1984 flows under the SWDS south-southeast toward the NFHR. The sediment of the NFHR sampled at six monitoring stations at the SWDS and a periodic intervals to 75 miles downstream. Mercury concentrations in NFHR sediments, periphyton, and snail tissue are lower in upstream

samples than in samples of the chlor-alkali plant and downstream from the plant (Dye, unpublished data).

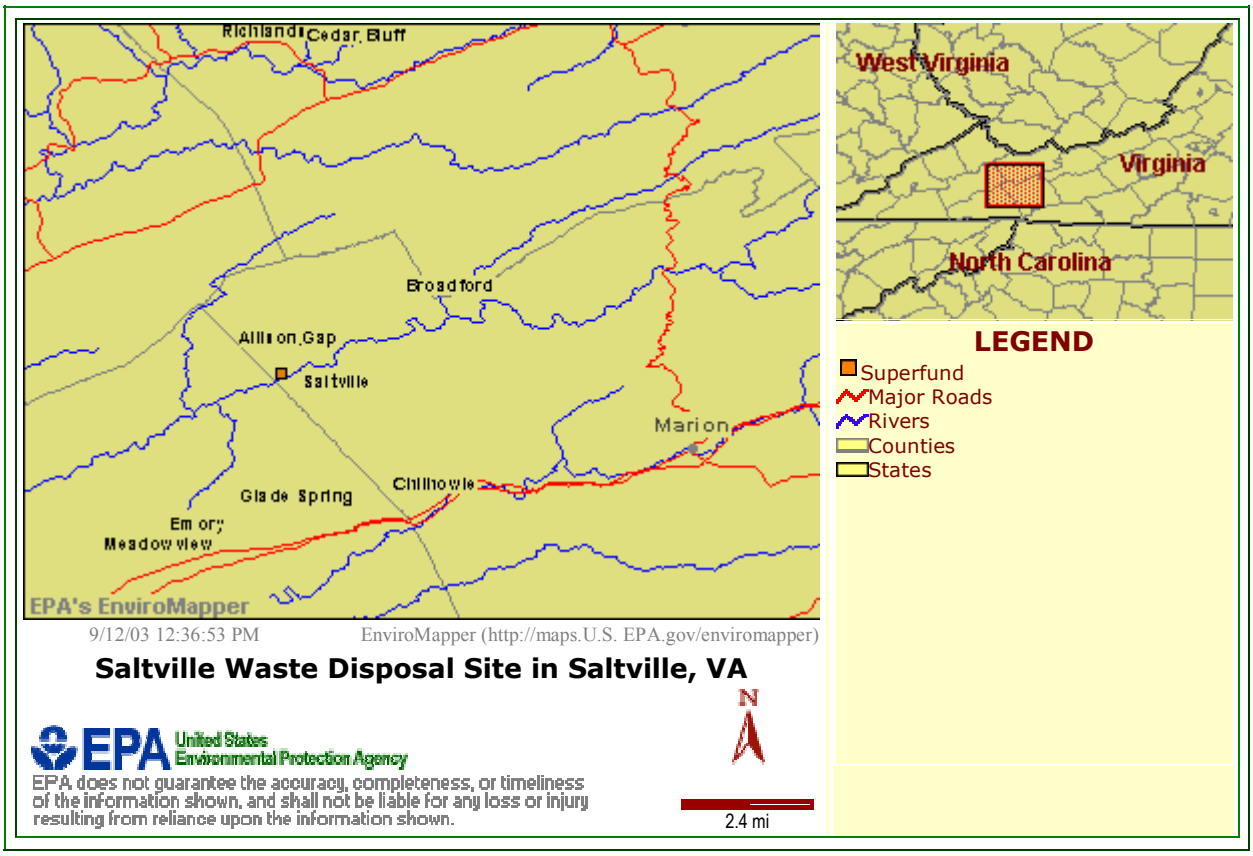

Figure1. Saltville Waste Disposal Site in Saltville, Virginia (www.U.S. EPA.gov/enviromapper 2003)

Table 1 Investigations Conducted on Environmental Media in Relation to the Saltville Waste Disposal Ponds (ATSDR 1987)

Agency	Year	Environmental Media (Site)	Contaminant	Contaminant Concentration
Olin-Mathieson Corporation (PRP)	---	Soil (Chlor-alkali plant)	Mercury	Up to 1,882 ppm
Olin-Mathieson Corporation (PRP)	1984	Groundwater (Beneath SWDS)	Mercury	5 – 439 ppb
---	1970-1981	Surface water	Mercury	<1 ppb (total mercury)*
Virginia State Water Control Board	1970-1981; 1983-1984	Sediment (NFHR)	Mercury	0.5 – 3.95 ppm**
Tennessee Water Quality Control Board	1980	Fish Flesh (75 miles downstream of SWDS)	Mercury	40 % with mercury concentrations above 1 ppm ***in 4 of 6 monitoring stations
Virginia State Water Quality Control Board	July 1981 – October 1985	Water (Outflow from WP 5 to NFHR)	Mercury	Non-detectable to 246 ppb

Virginia set concentrations of 0.05 ppb total mercury and 0.012 ppb methyl mercury as acceptable.
****Acceptable levels are less than or equal to 0.5 ppm.***
*****FDA has assigned 1 ppm as the human health risk action level in fish.***

Mercury, arsenic, and lead are contaminants of concern (COCs) in the Saltville Waste Disposal Ponds, with mercury as the major COC (ATSDR 1987, 1992). The U.S. EPA collected two dust samples from the basement and the first floor of the chlor-alkali plant and found elevated levels of mercury, arsenic, and lead (Table 2) (ATSDR 1994).

Several remediation activities release these metals to the environment (Table 3). These remediation activities and natural environmental releases of these metals may lead to human exposure and resulting health effects through exposure pathways (Table 4) (ATSDR 1987). These exposure pathways pertain to only mercury, but arsenic and lead are also COCs available for human exposure. There are identified exposure pathways for humans, but no investigations assess whether Saltville residents have diseases from the long-term release of contaminants from the (SWDS). Selected health effects manifested in Saltville residents characterized human exposure to the SWDS. A review of the literature was the basis for the selected diseases characteristic of chronic exposure to heavy metals.

Table 2 Heavy Metal Concentrations at the Inactive Power Plant and the SWDS (ATSDR 1994)

Metal	Concentration (Location)
Mercury	16 ppm (basement) 5.6 ppm (first floor)
Arsenic	None detected (basement) 120 ppm (first floor)
Lead	1,100 ppm (basement) 8,800 ppm (first floor)

Table 3 Remediation Activities Related to the Saltville Waste Disposal Ponds (ATSDR 1987, U.S. EPA 2000)

Agency	Year	Remediation Activity
Olin-Mathieson Corporation	1978	Complete drainage of WP 5 and WP 6
Olin-Mathieson Corporation	1982	Dredging of mercury contaminated sediments of NFHR according to special order issued by Virginia State Water Control Board
Olin-Mathieson Corporation	1983	Construction of surface water diversion ditches around WP 5
Olin-Mathieson Corporation	1987	Implementation of groundwater treatment plant
Olin-Mathieson Corporation	1995	Modification of groundwater treatment plant; long-term monitoring; cap 75 acres
Olin-Mathieson Corporation	Present	Destruction of homes in close vicinity of SWDS

Table 4 Pathways of Human Exposure to the SWDS in Saltville, Virginia

Media/Matrix	Human Exposure Pathway
Contaminated fish	Ingestion
Contaminated Groundwater	Ingestion
Livestock grazing on contaminated crops	Ingestion
Vegetables grown in contaminated soil	Ingestion
Other aquatic life that bioaccumulates contaminants from sediments	Ingestion
Airborne contaminants	Inhalation/Ingestion
Contaminated surface water, soils, and sediments	Direct dermal contact

A cross-sectional study was conducted to assess the human health risk posed by release of mercury, arsenic, and lead to the environment. The first null hypothesis was

that Saltville Medical Center (SMC) patients do not have diseases characteristic of chronic heavy metal exposure. The second null hypothesis was that the odds of developing a disease characteristic of chronic exposure to the selected heavy metals do not differ between people living in Saltville, VA and not living in Saltville, VA.

Objectives

The first objective was to determine if SMC patients had diseases consistent with exposure to mercury, arsenic, and lead. The second objective was to determine if there was a significant difference between prevalence ratios in Saltville Medical Center patients residing in Saltville and patients not residing in Saltville.

Site Description

Saltville, Virginia is located in Smyth and Washington counties. The city has a population of 2,204 (United States Census Bureau 2000). The median family (n=655) income for Saltville is $36,394. The majority (67%) of the Saltville population aged 25 years or more are high school graduates or higher and 7.3 % have a bachelor's degree or more. The top three job types held by Saltville residents, aged 16 years and older, are production, transportation, and material moving (38.2%), management and professional (20.4%), and sales and office (20.1%) (U.S. Census 2000).

Owners occupy most housing units (70.2%). The median year in which these structures were built is 1957. Most Saltville housing units are in Smyth County (89.2%) (U.S. Census 2000). There are 52 homes located between the former site of the chlor-alkali plant and WP5. Three homes are south of the outfall from WP5 and two homes are southwest of the plant site across the NFHR (ATSDR 1987). The Perryville community includes homes moved to WP1 from an area considered "too close" to the active chlor-

alkali plant (ATSDR 1992). The wind direction for Saltville is northwest during winter months and southeast in summer months (Rule and Iwashchenko 1998). The SMC serves people living in Saltville and other Southwest Virginia cities (i.e., Damascus and Glade Springs).

Benefits Expected

This study was undertaken because of the potential for an increased risk of adverse health effects in this community. Further surveillance of disease prevalence in populations exposed to living near hazardous waste sites is needed.

Study Limitations

No tests were conducted to quantify the metal body burden from specific exposure for individuals (i.e. measurements of COCs in hair and blood). Another limitation was that the purpose of compiling medical records was not to specifically investigate exposure to heavy metals.

CHAPTER 2

LITERATURE REVIEW

Environmental Fate and Transport of Mercury, Arsenic, and Lead

ATSDR (1987) identified human exposure pathways from transport of chemicals from the SWDS to the surrounding environment (Table 4). The waste constituents were disposed using land application and eventually contaminated groundwater (Table 1). The hydrologic cycle, wind current, and volatilization from remediation activities transport the metals to the sediment of the NFHR, air, surface water, and biota (Table 3) (ATSDR 1992, 1993, 1999, 1987). These metals are in different oxidation states and chemical forms depending on the environmental media and conditions.

Mercury

Mercury exists in soils in the mercuric (Hg^{+2}) and mercurous (Hg^{+1}) states (ATSDR 1993). Soil organic matter and mineral colloids have an affinity for mercury (II) and form complexes with inorganic mercury (II) compounds (U.S. EPA 1997). These complexes limit mercury mobility in soil. Some mercury (II) can be absorbed onto dissolvable organic ligands or reduced to elemental mercury by humic substances and photolysis (U.S. EPA 1997). Photolysis of organic mercury compounds is the primary transformation for mercury compounds in the atmosphere, where metallic mercury is the most reduced form and readily vaporizes (ATSDR 1993).

Elemental mercury in sediments saturates surface waters (ATSDR 1993). Santschi et al. (1999) found mercury in sediment layers closest to a chlor-alkali facility and assume it will remain there unless disturbed. Balogh et al. (1997) support this assumption in their report of a high correlation ($r = 0.98$, $r^2 = 0.96$) between total mercury

concentrations and sediment mercury concentrations. Particulate mercury concentrations are similar to total mercury concentrations in surface sediments of Lake Michigan (Mason and Sullivan 1997). Methyl mercury and total mercury concentrations in biota increase as trophic level increases (Mason and Sullivan 1997).

Mercuric mercury (Hg^{2+}) is the dominant form in surface waters (ATSDR 1993). Aerobic and anaerobic biotransformations are very important in environmental fate of mercury in fresh and marine surface waters (ATSDR 1993). Biotransformation of mercury involves conversion of organic and inorganic forms where demethylation and methylation occur, respectively. Inorganic mercury is an important source of methyl mercury in anaerobic settings where sulfate-reducing bacteria methylate inorganic mercury (Morel et al. 1998). According to the Agency for Toxic Substances and Disease Registry (ATSDR) (1993), methyl mercury is the most common organic form with concentrations in carnivorous fish 10,000 to 100,000 times the concentration in surrounding waters. This is significant for biomagnification in aquatic biota consumed by humans or other consumers in the food chain. Methyl mercury rapidly enters the aquatic food chain because it is highly soluble and mobile (ATSDR 1993). Mercury concentrations in yellow perch from the Adirondack lakes increase as length, weight, and age of the perch increase (Driscoll et al. 1994). The elevation of mercury concentrations in fish also correlates with increasing mercury concentrations and with decreasing pH in the lakes. Mercury concentrations in plants and accumulation in livestock are small (U.S. EPA 1997).

Arsenic

Most arsenic in the environment is in soil or rock (ATSDR 1992). Arsenic is widely distributed in nature in the combined state including minerals as sulfides, oxides, arsenites, and arsenates (Yan-Chu 1994). Arsenic occurs mainly as an inorganic species but is bound to organic material in soils where microorganisms convert them to organic forms (Yan-Chu 1994). Organic arsenic as dimethylarsinic acid (DMAA) is volatile and is possibly dominant in all soils (Bhumbla and Keefer 1994). Arsenate (As (V)) is the dominant form in aerobic soils. Arsenite (As (III)) is the dominant form in moderately reduced soils. Arsine, methylated arsenic, and elemental arsenic are the dominant forms in highly reduced soils (ATSDR 1992). Phosphate from fertilizers or disposed wastes inhibits arsenic movement in soils (Bhumbla and Keefer 1994). Arsenic is toxic to plants but is chemically and behaviorally similar to phosphorus, a plant nutrient (Bhumbla and Keefer 1994). Arsenic accumulates in plants grown on soils contaminated by arsenic pesticides. Most arsenic remains in plant roots because it does not transport to shoots (Bhumbla and Keefer 1994).

The transport and partitioning of arsenic in water is dependent upon its chemical form and interactions with other chemicals (ATSDR 1992). Arsenic bound to bicarbonate or ferric ions leaches more efficiently by groundwater than by deionized water in Marshall Sandstone (Kim et al. 2000). Organic arsenic concentration is at least ten times greater than inorganic arsenic in most fresh and marine waters. Because of this, most aquatic organisms accumulate very low concentrations of inorganic arsenic (Phillips 1994). Methylation detoxifies inorganic arsenic as it moves through the food chain

(Phillips 1994). Organic arsenic in aquatic plants, mollusks, crustaceans, and fish is not biomagnified.

Arsenic incorporates into sediments by co-precipitation as manganese and iron oxyhydrides (Bhumbla and Keefer 1994, Mok and Wai 1994). Adsorbed arsenicals are difficult to remove and leachability is limited (Mok and Wai 1994). Their mobility depends on the rate and volume of water passing through sediments (Mok and Wai 1994). Toxicity from arsenic is higher in reduced sediments than in oxidized sediments because As (III) is more toxic than As (V) and organic arsenic compounds (Bhumbla and Keefer 1994). Methylation in sediments produces the less toxic compounds, monomethylarsonic acid (MMAA) and DMAA (Mok and Wai 1994). Chemical or biological conversion of arsenic species releases sediment-bound arsenic into water (ATSDR 1992). Arsenic released into the atmosphere is mainly as arsenic trioxide (AsO_3) and less as volatile organic compounds (VOCs) (ATSDR 1992).

Lead

Lead in the air is in vapor (primarily from tetraalkyllead in gasoline) and particulate phases (Davidson and Rabinowitz 1992). Emissions from mines and smelters release lead particulates mainly as lead sulfate ($PbSO_4$) and lead carbonate ($PbCO_3$) (ATSDR 1999). Airborne alkyl lead species decompose to inorganic lead by successive loss of alkyl groups from photolysis or by reacting with the hydroxyl radical (Davidson and Rabinowitz 1992).

Natural mineral forms of lead are a minor part of U.S. soils (ATSDR 1999). Wet and dry deposition from the atmosphere and surface water add more lead. Photolysis of organic lead compounds forms $PbSO_4$ and $PbCO_3$ in soil (ATSDR 1999).

Transformation of lead into precipitates and complexes depends on the soil type (ATSDR 1999). Lead has a high binding capacity to organic matter in soil and enters surface waters from erosion and other modes of physical transport (i.e., soil turnover by worms and landscaping) (ATSDR 1999, Davidson and Rabinowitz 1994).

The amount of lead in surface waters depends on pH, partial pressure of carbon dioxide, dissolved salt content, and water temperature (ATSDR 1999). Lead and the major anions in natural water form low soluble compounds (ATSDR 1999). Because hard drinking water is more acidic than soft drinking water, it contains higher levels of lead (Needleman 1992). Photolysis degrades water insoluble tetraalkyl lead compounds to inorganic lead (ATSDR 1999). Evaporation causes the loss of the more volatile compounds. Triethyl and trimethyl lead are more water soluble and susceptible to photolysis than tetraalkyl lead. Organic lead compounds are more toxic than inorganic lead compounds and bioconcentrate in aquatic organisms (ATSDR 1999). Sediments contain higher concentrations of lead than surrounding surface waters (ATSDR 1999) and the source is mostly anthropogenic.

Organic lead compounds become soluble, leach out, or enter plants at a pH range of 4 to 6 (U.S. EPA 1986a as cited by ATSDR 1999). Lead is taken up in edible plants from the soil through the roots, through leaves, translocation in the plant, and by particulate matter deposits on the surface; biomagnification has not been seen (ATSDR 1999).

Diseases Associated with Chronic Exposure to Mercury, Arsenic, and Lead

ATSDR Toxicological Profiles of Mercury, Arsenic, and Lead

Human exposure to metals is routine. This exposure causes adverse human health effects in settings such as close residential proximity to the SWDS. Multiple contaminants, other exposure sources, symptoms (nonspecific and non-reported), lifestyle factors, demographics, and latency periods of illness confound hazardous waste site exposure as an etiologic agent. The ATSDR addresses the ambiguity of diseases caused by exposure from hazardous waste sites in toxicological profiles of mercury, arsenic, and lead (ATSDR 1992, 1993, 1999). The ATSDR describes chronic human diseases from exposure to these chemicals through reviews of toxicological and epidemiological studies. The studies are different in design, in test population, and in sources of exposure but the effects are similar. This literature review cites not every health effect in the toxicological profiles because some are common among all three metals or non-specific (i.e., gastrointestinal irritation).

The mercury profile consists mainly of case-control and cross-sectional studies conducted in occupational settings (ATSDR 1993). Chronic exposure to metallic, organic, and inorganic mercury affects the cardiovascular system, central nervous system, peripheral nervous system, and the musculoskeletal system (ATSDR 1993). Irritability, tremors, and parathesia are the effects most unique to chronic mercury exposure. Some reported symptoms are not limited to exposure to one form of mercury or one exposure route. The overlap of illness from chronic exposure to different mercury forms is due to metabolic conversion in the body (Lu 1996). For example, metallic mercury oxidizes to inorganic divalent mercury in the hydrogen peroxidase-catalase pathway (ATSDR 1993).

Diseases contracted by humans chronically exposed to only inorganic arsenic are in the toxicological profile (ATSDR 1993). The information on development of human diseases following arsenic exposure is limited (ATSDR 1993). Secondary effects from cardiovascular or vascular injury occur in the liver, respiratory system, and kidneys (Table 5).

Information about dose-response relationships of blood lead levels (BLLs) versus adverse health outcomes (Table 6) is in the lead toxicological profile (ATSDR 1999). The routes of exposure are inhalation in occupational settings and ingestion in non-occupational settings.

Table 5 Symptoms and diseases related to Chronic Exposure to Arsenic (ATSDR 1993)

Body System	Effect (route of exposure)
Cardiovascular	• Cardiac arrhythmias (oral, inhalation) • Blackfoot disease (oral, inhalation) • Raynaud's disease (oral, inhalation) • Cyanosis of phalanges (oral, inhalation) • Thickening and occlusion of blood vessels (oral, inhalation)
Dermal	• Hyperpigmentation, hypopigmentation (oral, inhalation [rare]) • Skin cancer (oral) • Dermal sensitization (dermal) • Hyperkeratotic warts (oral, inhalation [rare])
Respiratory	• Laryngitis (inhalation) • Bronchitis (inhalation) • Rhinitis (inhalation) • Increased lung cancer incidence (inhalation)

Table 6 Symptoms and diseases related to Exposure to Lead (ATSDR 1999)

Body System (# of reviewed studies)	Effect
Cardiovascular (8)	• Increased blood pressure • Increased systolic pressure and diastolic pressure with every doubling in BLL • Increase in systolic blood pressure and increase in diastolic blood pressure as BLL increased from 10 to 30 μg/L
Hematological (14)	• Decrease in hemoglobin and pyrimidine-5-nucleotidase • Anemia
Renal (5)	• Renal impairment with gout or hypertension

U.S. EPA Integrated Risk Information System (IRIS) Documents for Mercury, Arsenic, and Lead

The U.S. EPA publishes data from studies in the Integrated Risk Information System (IRIS) database. These data, in part, helped researchers establish the No Observed Adverse Effect Level (NOAEL) and the Lowest Observed Adverse Effect Level (LOAEL) of a chemical. This database was useful in learning what diseases develop from chronic exposure to low concentrations of mercury, arsenic, and lead. There were no studies described in IRIS about the health effects from chronic oral exposure to mercury. The referenced studies were about chronic inhalation in occupational settings (U.S. EPA 1998). The occupational settings included chlor-alkali production, dentistry, and fluorescent tube manufacturing. The LOAEL for occupational

inhalation exposure to mercury is 0.025 mg/m^3 (U.S. EPA 1998). Hand tremors and increased memory disturbances occurred at this concentration (U.S. EPA 1998).

The LOAEL for arsenic was determined from a statistically significant dose-response relationship between arsenic in well water and diseases in Taiwan (U.S. EPA 2000). The high and low doses were 22 mg/kg per day and 0.3-0.4 mg/kg per day, respectively (U.S. EPA 2000). The diseases associated with chronic exposure to arsenic through ingestion are hyperpigmentation, keratosis, and vascular problems (i.e., Blackfoot disease). The estimated LOAEL is 0.17 mg/L or 0.014 mg/kg per day (U.S. EPA 2000). Diseases without statistically significant trends demonstrated biologic plausibility because they were consistent with the Taiwanese studies. Cancer of the liver, kidney, lung, bladder, and skin also occurred from arsenic ingestion (U.S. EPA 2000). There was no report of diseases caused by inhalation.

The U.S. EPA also reviewed data for lead. The agency was too uncertain to assign a LOAEL for neurobehavioral development problems (U.S. EPA 1998).

Documents Supporting ATSDR Toxicological Profiles and US U.S. EPA IRIS Reports for Mercury, Arsenic, and Lead

The exposure to chemicals in hazardous waste sites, such as the SWDS, is chronic in low concentrations (ATSDR 1992, 1993, 1999, U.S. EPA 1998, 2000). Additional literature is consistent with the ATSDR toxicological profiles and the U.S. EPA IRIS database (CDC 1989, 1990, 1991, 1995, Feng et al. 1998, Grandjean et al. 1998, Harada et al. 1998, Mathieson et al. 1999, Smith et al. 1997, Smith et al. 2000, Tondel 1999). Carcinogenic effects, bronchitis, and laryngitis were commonly reported after chronic

inhalation of arsenic (Lu 1996, Lubin et al. 2000). This research focused on non-carcinogenic diseases.

Authors report lead concentrations and associated diseases not given in the IRIS lead document. These researchers used lead concentrations in human tissues instead of exposure routes. Lead exposure was demonstrated by higher BLLs in exposed groups than in control groups with statistical significance (CDC 1997, Eidson and Tollestrup 1995, Fernandez et al. 1997, Holmes et al. 1997, Lynch et al. 2000, Murgueytio et al.1998, Rothenberg et al. 1998, 2000, and Tumpowsky et al. 2000).

Lead enters the bloodstream and distributes to the blood, soft tissue (i.e. reproductive tissue), and bone tissue. This affects the nervous system (peripheral and central), the blood cells, vitamin D and calcium metabolism, and the reproductive system (ATSDR 1997). Several researchers identified associations between levels of lead in the body and adverse health outcomes including neurological effects (Pocock et al. 1994, Soong 1999), hearing impairment (ATSDR 1995,Wu et al. 2000), and interference with heme enzyme activity causing anemia (ATSDR 1997). Hematocrit concentrations and BLLs correlate negatively (Schell et al. 2000). Hematocrit and hemoglobin concentrations correlate negatively with bone lead levels (Hu et al.1994). The interference of vitamin D metabolism from lead exposure alters cell growth and maturation, tooth development, and bone development (Kafourou et al. 1997, Moss et al. 1999). Nephropathy from chronic exposure to lead causes hypertension, gout, and renal failure (ATSDR 1997, Hu et al. 1996, Rothenberg et al. 1999, Schwartz 1992, Schwartz et al. 2000, Wedeen 1992).

Several researchers report the health effects of interest are a result of similar long-term exposures to mercury, arsenic, and lead (Table 7, Table 8, and Table 9). The design of most studies was cross-sectional like this study. There were also case reports and case–control studies of exposed individuals with health effects.

Table 7 Studies of Long-term Exposure to Mercury

Study Design	Group	Age	Gender	Race	Source of Exposure	Health Effects
Cross-sectional Ellingsen et al. (2001)	47 workers at chlor-alkali plant; 47 referents	-Case mean age=42y -Referent mean age=41.9y p=0.95	Male	N/A	Chronic exposure to mercury	Association (r=0.49,p<0.05) between past exposure to mercury and digit symbol test results (visual short-term memory, psychomotor speed, attention, and perceptual organization);
Group-control Powell (2000)	15 former chemical workers of a mercury acetate plant; 15 referents;	-Group mean age= 37.1y -Referent mean age=35.3y p=0.6	Male	Zulu	Chronic exposure to mercury	-Significant diff. between exposed and referents (p<0.0001) in delayed short-term verbal and spatial memory; Significant difference between exposed and referents (p<0.01) in screening test for attention deficits; Significant difference between exposed and referents (p<0.01) in tests of motor speed and manual dexterity; 73% body tremor, 53% memory and concentration problems in exposed subjects
Case Study Hua et al. (1995)	Employee at a lampsocket manufacturing factory; 30 referents	-Case: 29 years -Referent mean age=28.3y	Male	N/A	Chronic exposure to mercury	-Complained of tremor and unsteady gait -Neurological tests' results: Slurred speech, postural and action tremor of both hands, ataxic gait, mild constriction of visual fields -Most neurological measures were abnormal in comparison

						with test results of normal group
Case-control Kishi et al. (1993)	76 Former mercury miners; 76 controls	N/A	Male	N/A	Past chronic exposure to mercury; 18 yrs after cessation of mercury exposure	Significantly higher prevalence of hand tremors, slurred speech, and headaches in cases than controls
Cross-sectional Hassan et al. (1981)	152 employees of a chlor-alkali plant	N/A	N/A	N/A	Levels of airborne Hg vapor in the plant as high as 600μg/m^3	-37% (n=56) w/symptoms -80.3% (n=45) with tremors -80.3% (n=45) with memory and attention deficits -92.9% (n=52) with parathesia
Case report Blair et al. (1989)	Child	3 years	Male	N/A	-Previous home owner collected elemental mercury	-Hypersalivation, myalgia and tremor in the hands, myalgia and weakness in both lower extremities, diaphoresis, irritability, insomnia, and anorexia
Cross-sectional Harada et al. (1998)	191 fishermen and family living in the Minamata area	32-82 years	83 males 108 females	Japanese	Consumption of methylmercury in fish	-188 with clinical symptoms: Numbness (79.8%), forgetfulness (76.1%), pain in extremities (60.6%), motor disturbance (34%), sensory disturbance/ glove and stocking type (75%), constriction of visual field (5.9%), tremor (7.4%)
Cross-sectional McCullough et al. (2001)	High-exposure (HE) (n=13) Low-exposure (LE) (n=3)	-HE mean age=33.1y -LE mean age=29.7y	-HE group: 76.9% male -LE group: 100% men	-HE group: 92.3% white -LE group: 100% white		Tremor index for high-exposure group was 27% lower (poorer performance) than low-exposure group (p=0.04)

Cross-sectional Zavariz and Glina (1992)	71 electric lamp factory employees	N/A	N/A	N/A	-85.9% (n=61) chronically exposed, 80.3% (n=57) poor psychomotor coordination, 78.9% (n=56) neurological impairments, 71.8% (n=51) decrease in memory capacity, 66.2% (n=47) pathological findings in the clinical exam, 63.4% (n=45) psychiatric disturbances, 52.1% (n=37) poor performance in the concentration test

Table 8 Studies of Long-term Exposure to Arsenic

Study Design	Group	Age (years)	Gender	Race	Source of Exposure	Health Effects
Cross-sectional Mazumder et al. (1992)	53 members of 17 families	N/A	N/A	South Calcutta Residents	Use of water contaminated with arsenic from a Paris-green factory	20 cases of clinical signs -Pigmentation (100%) -Palmar and plantar keratosis (65%) -Hepatomegaly (65%) -Splenomegaly (35%)
Cross-sectional Tondel (1999)	1,481 Residents	>30	N/A	Bangladesh Residents	Arsenic-contaminated well water	-430 subjects with keratosis, hyperpigmentation/hypopigment-ation; Crude overall prevalence rate for skin lesions =29/100; Age-adjusted prevalence rate for males=30.1/100; females=26.5/100; $p<0.05$
Cross-sectional Cebrian et al. (1983)	Exposed rural population ; Control population	N/A	N/A	Mexican	Arsenic-contaminated drinking water -Exposed: 0.41 mg Arsenic/ L -Control: 0.007 mg Arsenic/ L	-21.6% of exposed sample had at least 1 cutaneous sign of chronic arsenic poisoning; 2.2% of control sample -RR of manifestation of chronic arsenic poisoning was 1.9 to 36 times higher in exposed sample
Cross-sectional Chakraborti et al. (2002)	8,500 residents	N/A	N/A	Murshidabad district, West Bengal, India	Arsenic-contaminated groundwater	36.5% with arsenical skin lesions

Table 9 Studies of Long-term Exposure to Lead

Study Design	Group	Age	Gender	Race	Source of Exposure	Health Effects
Cross-sectional Landrigan et al. (1976)	Children	1-9 years	Both	N/A	Residential proximity to a lead smelter	Anemia for 17% of children with BLLs >=80μg/dL
Cross-sectional Lin et al. (2002)	Clinic patients -27 cases -84 controls	- Case Mean age =53y - Control Mean age =55y -p=0.3	-Case: 75% male -Control: 77% male -p=1.0	N/A	General Environmental Chronic Exposure to Lead	Higher body lead burden in subjects with gout than subjects without gout
Cross-sectional Lin and Huang (1994)	Normal serum creatinine levels, no gout (n=10); Gout, abnormal serum creatinine levels (n=10); Abnormal serum creatinine levels, no gout (n=20)	Not given	Male	N/A	General Environmental Chronic Exposure to Lead	Significantly higher lead stores and serum urate in gout patients (group 2) with renal insufficiency than groups 1 and 3 ($p<0.05$)
Case-control Korrick et al. (1999)	Subsample of subjects from the Nurses' Health Study	Middle-aged	Female	N/A	Non-occupational exposure to low levels of lead	Increase from the 10^{th} to the 90^{th} percentile of patella lead values was associated with about 2-fold increased risk of hypertension (95% confidence interval=1.1,3.2)

Chronic Diseases and Lifestyles with Similar Symptoms as the Selected Health Effects

The diseases and symptoms selected for this study are symptoms of other chronic diseases. Memory disturbances and tremors, the symptoms related to mercury exposure, are also symptoms of Alzheimer's disease and Parkinson's disease, respectively (Backman and Herlitz 1990, Chase and Lumpe 1991, Geerlings et al. 1999, Litvan 1998, Turkington 1999, Wiggins 2001,). The symptoms related to arsenic exposure, hyperpigmentation, hypopigmentation, and keratosis, are precursors of skin cancer (Anonymous 1993, Stohrer 1991). Nephropathy causes the diseases related to lead exposure, anemia, gout, and hypertension (Astor et al. 2002, Pittman and Bross 1999, Saunders 1998). Gout and hypertension associate with obesity and diabetes mellitus (Arromdee et al. 2003, Bleicher 1992, Christ 2000, Lewis 2002,). The production of defective hemoglobin causes sickle cell anemia (Haggerty 1999). Family history of similar diseases is also associated with the selected health effects.

Tobacco use also contributes to diseases characteristic of chronic exposure to mercury, arsenic, and lead. Lead and mercury are in cigarettes (British Columbia Ministry of Health 1998). Other chemicals in cigarettes cause tremors, memory loss, skin color changes, and increased risk of cutaneous squamous cell carcinoma (British Columbia Ministry of Health 1998, De Hertog et al. 2001, Straten et al. 2001).

CHAPTER 3

MATERIALS AND METHODS

A cross-sectional study was conducted to determine the prevalence of key health effects associated with chronic exposure to mercury, arsenic, and lead. The data came from the Saltville Medical Center patient database. The East Tennessee State University Institutional Review Board (ETSU IRB) approved the study protocol.

There were 9,350 medical records at the SMC when this study began. A sample size of 190 medical records was reviewed for a 95% confidence limit (Equation 1, Appendix A) (Wang et al. 1995). The inclusion criteria were Virginia residency with a particular interest in Saltville residents and patients seen between 1991 and 2000. The data were abstracted from an admission sheet, a physical history sheet, and doctors' notes. A data collection form was used to record data (Appendix B). The chosen diseases associated with chronic exposure to the selected heavy metals are in Table 10. Chronic diseases with similar symptoms, family history of similar disease, and smoking status were recorded to analyze for their potential of having a stronger association on the likelihood of developing a disease than Saltville residence (Appendix B). Data were summarized using univariate and multivariate statistical procedures with Statistical Package for the Social Sciences version 11.0 (SPSS 11.0) (SPSS Inc., Chicago, Illinois and SPSS 1999).

Table 10 Selected Heavy Metals and Diseases Characteristic of Chronic Exposure

Heavy Metal	Diseases
Mercury	Tremors Memory disturbances
Arsenic	Hyperpigmentation / hypopigmentation Keratosis
Lead	Anemia Hypertension Gout

CHAPTER 4

RESULTS

Medical records were found in two formats, paper and computerized. The computerized records were not available for this study. Therefore, paper medical records (n=190) from the Saltville Medical Center were reviewed. Of the 190 records, 96 records (50.5%) were on patients from Saltville and the other 94 were records on patients residing outside the Saltville city limits. The data collection was complex because of clinical jargon (i.e. alternate names for selected diseases and abbreviations) and inconsistent organization in the records. The Ninth Revision of the International Classification of Diseases codes (ICD-9 codes) used to classify diseases was not available. The permissible error, ε=0.04, gave a small but representative sample size that allowed time to deal with these problems.

Table 11 contains demographic information on Virginia, Saltville, the comprising counties, Smyth and Washington, and the SMC sample. The sample was 56.3% (n=107) female and 41.6% (n=79) male (Table 12). The gender was not reported for 2.1% (n=4). The largest racial group represented was Caucasian (n=129, 67.9%). The race was not recorded for 58 records (30.5%). The sample of records included one (0.5%) black subject and two (1.1%) Hispanic subjects. Although concerns were raised about racial/ethnic differences in disease prevalence, records for black or Hispanic subjects were not dropped from further analyses due to the small numbers.

The mean age for all subjects in this study was 38.3 years. The U.S. Census Bureau (2000) reported age groups as 18 years and above, 60 years and above, and 65 years and above for Virginia, Saltville, and Smyth and Washington counties in Virginia

(see Table 11). Some percentages did not add up to 100% because information was not available for the parts of Saltville in Smyth and Washington counties and 54 patients in the sample did not have a physical address. Overall, 63 subjects (33.1%) in the sample were reported smokers, 31 from Saltville (Figure 2). Only 15 patients were former smokers (11 from Saltville).

Of the 190 records reviewed, 42 records cited the diagnoses or treatments of 41 individuals with one disease of interest. There was one patient with two diseases of interest. There were 37 diseases characteristic of lead exposure; 4 were characteristic of arsenic exposure; and 2 were characteristic of mercury exposure (Figures 3 and 4). There were 24 Saltville residents with a disease that may have been associated with an exposure to mercury, arsenic, or lead; 19 of the 42 subjects were diagnosed with a chronic disease that may have caused the same symptoms (Table 13, Figure 5). Therefore, only 24 subjects (12.6%) identified from the initial 190 records had unexplained symptoms. Additionally, 20 subjects (10.5%) from the sample had family members with similar conditions.

All patients with tremors (n=2) and gout (n=2) were male (Figure 7). The two patients with tremors were in the age groups, 31-45 years (n=1) and 46-60 years (n=1) (Table 14). One of the male patients with gout also had hypertension. All patients with anemia (n=3) were female. There were more females (n=3) with keratosis than males (n=1). The females with keratosis were in the age group, 46-60 years, and the male was in the age group, 76-90 years. An equal number of males and females were found to have hypertension (n=32). Most patients with hypertension were in the age group of 46-

60 years (n=18) (Table 14, Figure 8). There were 58 males, 85 females, and 4 patients of unknown gender without any of the selected diseases. Most patients with a disease did not smoke (n=23). There were 15 patients with a disease who did smoke and only 5 patients with a disease were former smokers (Figure 9).

Table 11 Demographic Variables for Virginia, Saltville, VA, Comprising Counties of Saltville, and Saltville Medical Center Sample

Location/Group	Gender		Ethnicity						Age		
	Male	Female	Caucasian	Black	American Indian and Alaska Native	Asian	Native Hawaiian and other Pacific Islander	Hispanic	18+ years	60+ years	65+ years
Virginia (Total Population =7,078,515)	49% (n=3471895)	51% (n=3606620)	72.3% (n=5,120,110)	19.6% (n=1,390,293)	0.3% (n=21,172)	3.7% (n=261,025)	0.0056% (n=3,946)	4.7% (n=329,540)	27.4% (n=1,937,086) (19 and under)	15.05% (n=1,065,502)	27.4% (n=792,333)
Saltville town, Virginia (Total Population =2,204)	47.2% (n=1,040)	52.8% (n=1,164)	98.9% (n=2,181)	0.41% (n=9)	0.18% (n=4)	0%	0%	0.45% (n=10)	77.8% (n=1,714)	22.5% (n=497)	17.2% (n=379)
Saltville (In Smyth County) (Total Population=1,939)	NA	NA	88.1% (n=1,922)	55.5% (n=5)	100% (n=4)	0%	0%	100% (n=10)	87.3% (n=1,497)	84.5% (n=420)	85.2% (n=323)
Saltville (In Washington County) (Total Population=265)	NA	NA	11.9% (n=259)	45.5% (n=4)	0%	0%	0%	0%	12.7% (n=217)	15.5% (n=77)	14.8% (n=56)
SMC Sample (n=96)	38.5% (n=37)	59.4% (n=57)	82.3% (n=79)	0%	0%	0%	0%	0.1% (n=1)	87.5% (n=84)	16.7% (n=16)	11.5% (n=11)
SMC Sample(In Smyth)	43.2% (n=16)	22.8% (n= 13)	30.4% (n=24)	0%	0%	0%	0%	NA	30.9% (n=26)	31.25% (n=5)	36.36% (n=4)
SMC Sample (in Washington)	5.4% (n=2)	8.8% (n= 5)	3.8% (n=3)	0%	0%	0%	0%	NA	5.9% (n=5)	12.5% (n=2)	9.1% (n=1)

Table 12 Demographics of the Cross-sectional Sample

Variable	Category	% of Sample (n)	% Saltville Residents (n)
Diseases	• Yes • No	• 22.1 (42) • 77.9 (148)	• 57.1 (24) • 53.4(79)
Gender	• Male • Female • Not noted	• 41.6 (79) • 56.3 (107) • 2.1 (4)	• 46.8 (37) • 53.3 (57) • 50 (2)
Race	• Caucasian • Black • Hispanic • Native American • Other • Not noted	• 68 (129) • 0.5 (1) • 1.0 (2) • 0 (0) • 0 (0) • 30.5 (58)	• 61.2 (79) • 0 (0) • 50 (1) • 0 (0) • 0 (0) • 74.1 (43)
Age (years)	• 1-15 • 16-30 • 31-45 • 46-60 • 61-75 • 76-90 • Not noted	• 7.9 (15) • 28.4 (54) • 26.3 (50) • 25.3 (48) • 6.3 (12) • 3.7 (7) • 2.1 (4)	• 46.7 (7) • 53.7 (29) • 42 (21) • 52.1 (25) • 75 (9) • 57.1 (4) • 25 (1)

Table 13 Lifestyles of the Cross-sectional Sample

Variable	Category	% of Sample (n)	% Saltville Residents (n)
Smoking Status	• Non-smoker • Former smoker • Smoker	• 59 (112) • 7.9 (15) • 33.1 (63)	• 47.3 (53) • 73.3 (11) • 50.8 (32)
Family History	• Related • Unrelated	• 10.5 (20) • 12.1 (23)	• 65(13) • 56.5 (13)
Existing Chronic Diseases	• Related • Unrelated	• 10 (19) • 12.6 (24)	• 68.4 (13) • 54.1 (13)

Table 14 The Frequency of Diseases Associated with Heavy Metal Exposure By Age Groups of the Sample

	Disease					
Age Group	Tremors	Keratosis	Anemia	Hypertension	Gout	None
1-15	0	0	0	0	0	15
16-30	0	0	1	0	0	52
31-45	1	0	2	5	0	43
46-60	1	3	0	18	1	25
61-75	0	0	0	3	1	8
76-90	0	1	0	4	0	2

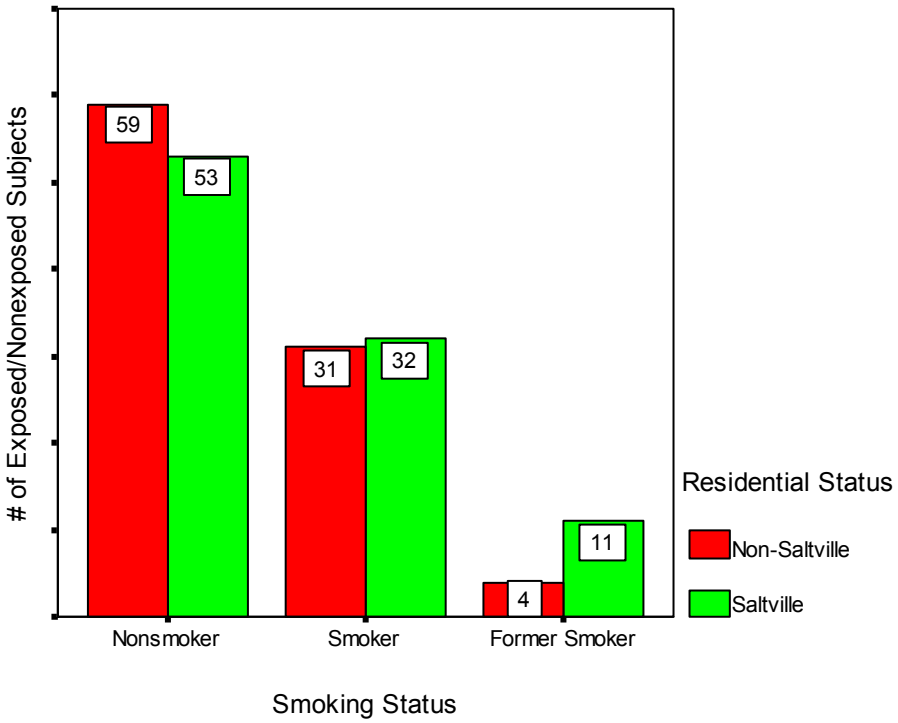

Figure 2 Number of Subjects in SMC Sample According to Smoking Status

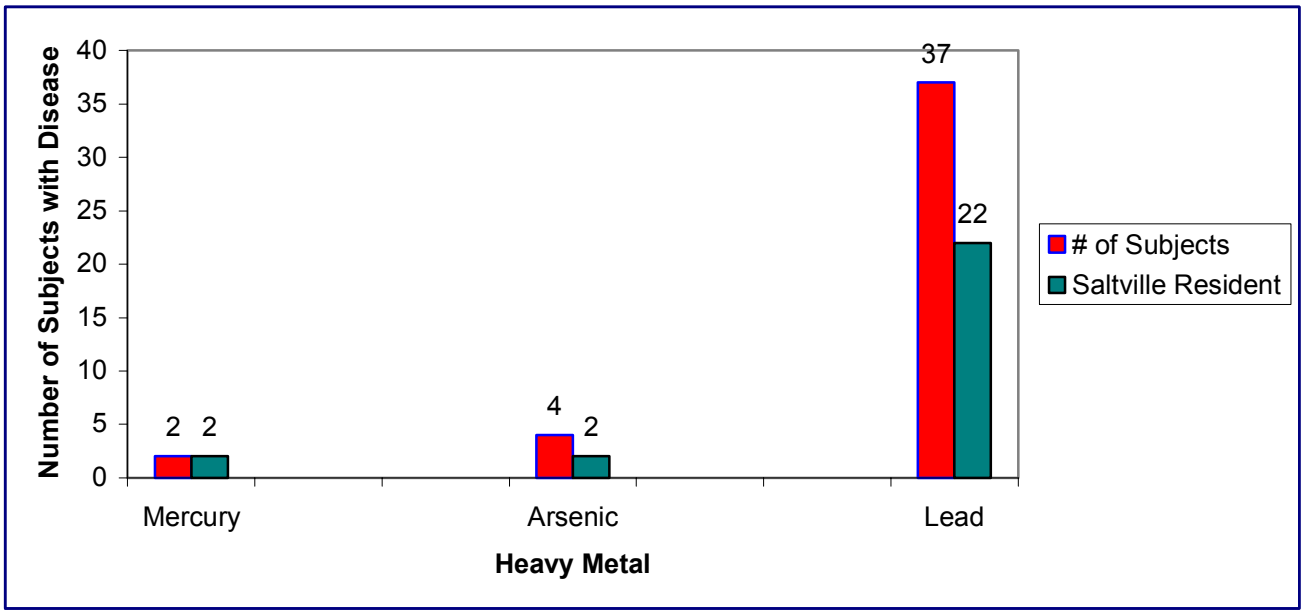

Figure 3 Residential Status of Subjects in SMC Sample with a Disease According to Heavy Metal (number above bars = frequency of subjects)

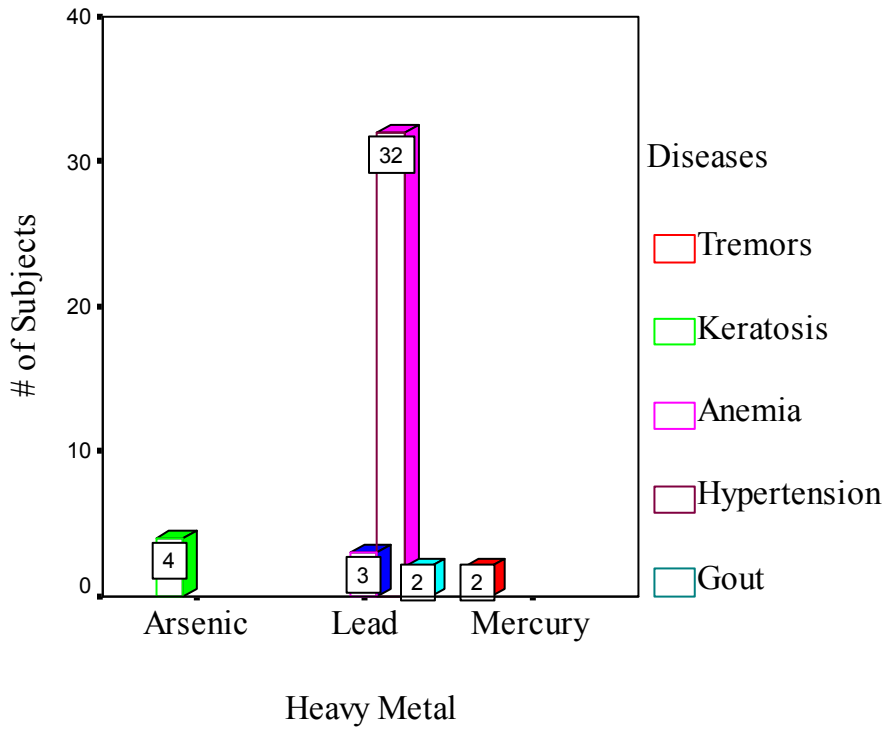

Figure 4 Diseases among SMC Sample Members According to Heavy Metal

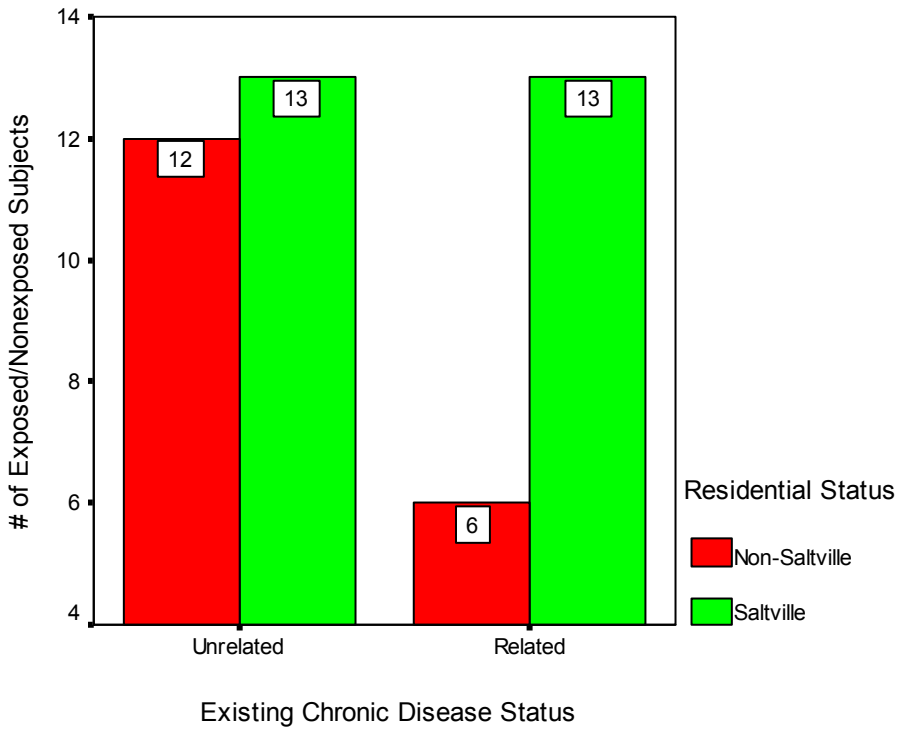

Figure 5 Number of Subjects in SMC Sample with Existing Chronic Diseases (related/unrelated) according to Residential (Exposure) Status

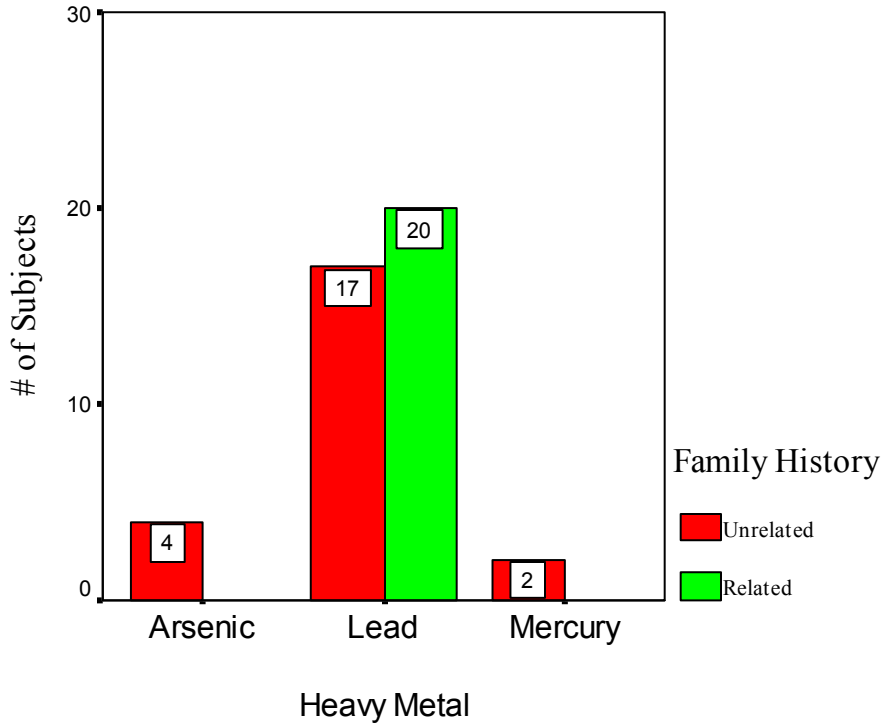

Figure 6 Frequency of Related/Unrelated Family History of Disease among the Subjects in SMC Sample with Diseases

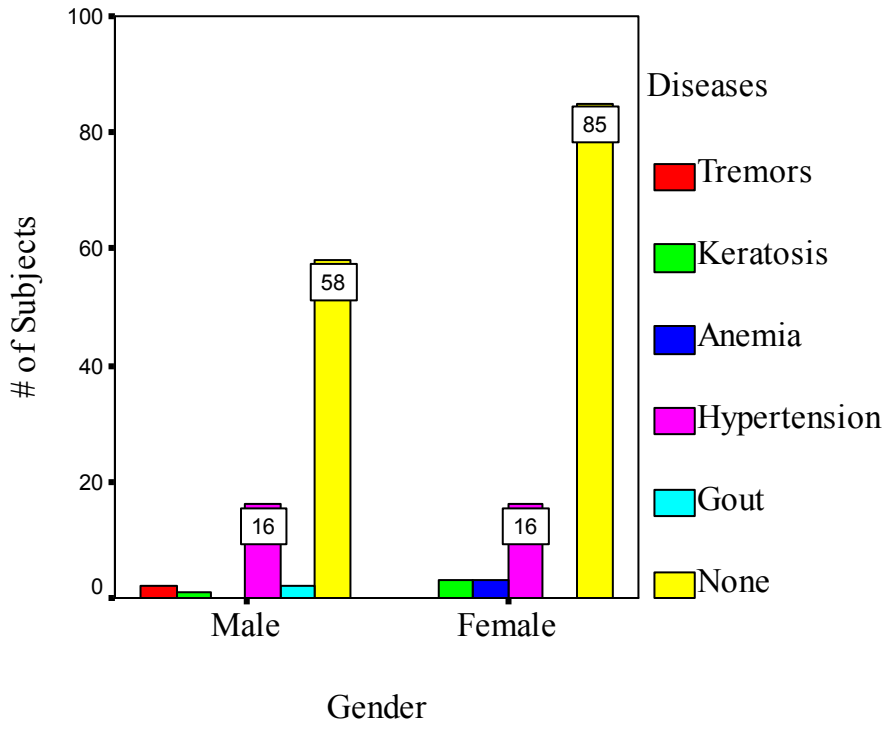

Figure 7 Diseases Associated with Chronic Heavy Metal Exposure According to Genders of the SMC Sample

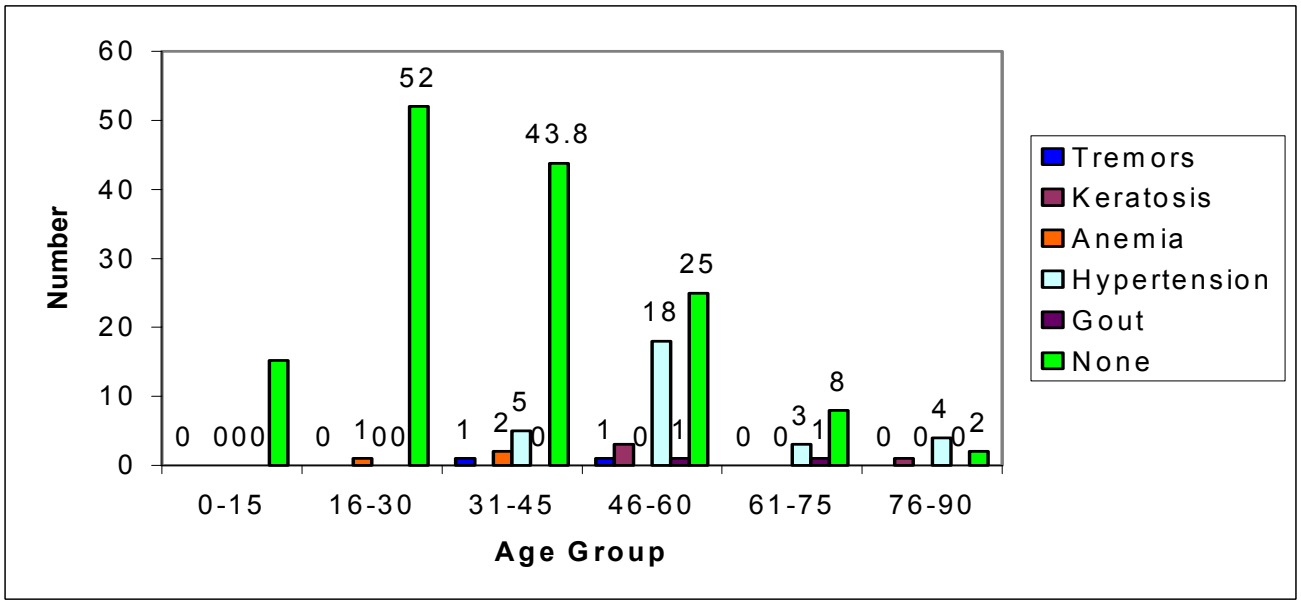

Figure 8 Diseases Associated with Chronic Exposure to Heavy Metals According to Age Groups of the SMC Sample

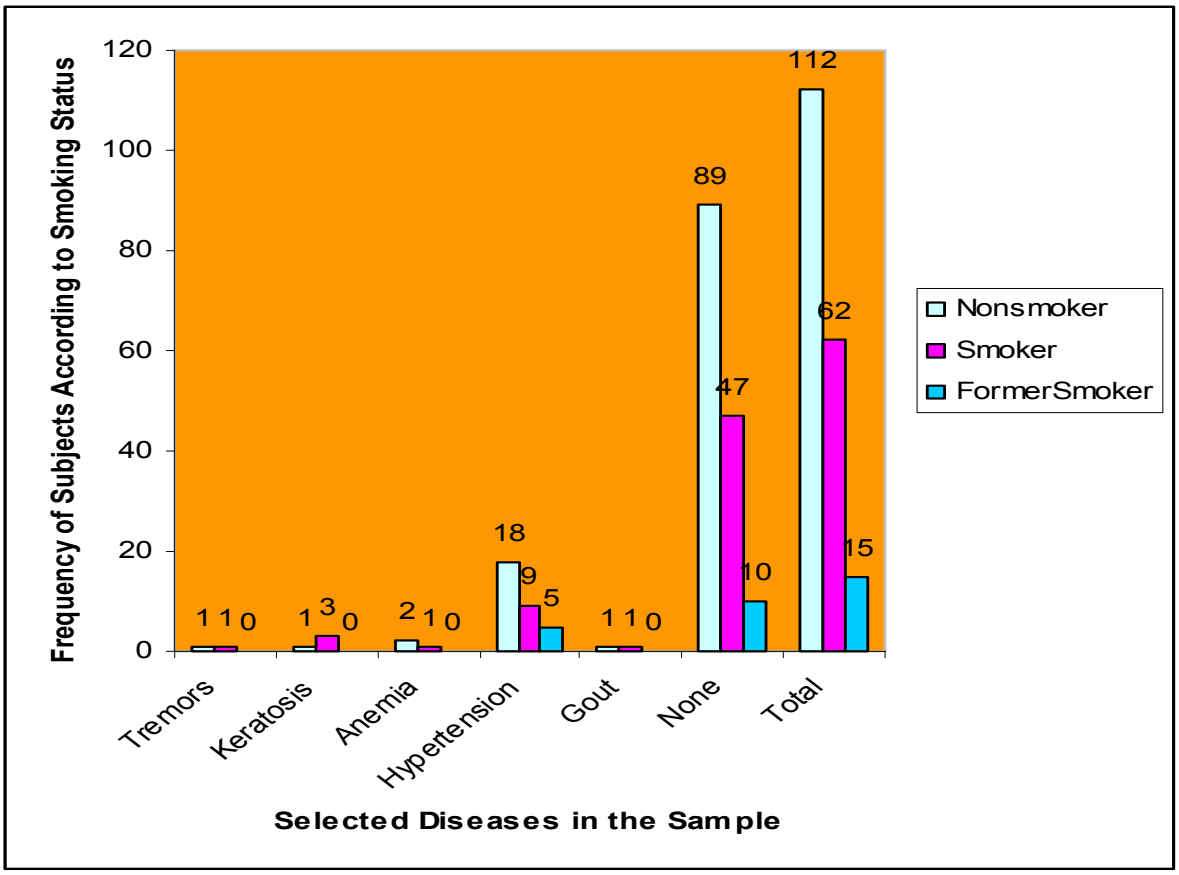

Figure 9 Smoking Status of Subjects in SMC Sample with Diseases

Prevalence ratios of the Saltville residents and non-Saltville residents were calculated to determine the odds ratio (Equation 2, Appendix A). Saltville residence served as a surrogate factor of the likelihood for exposure to the SWDS. All selected medical records were included in the odds ratio estimation. The sample was 50.5% (n=96) Saltville residents and 49.5% (n=94) non-Saltville residents. Figure 10 illustrates the cities of residence for the non-Saltville sample members. The prevalence ratio of disease (Equation 2, Appendix A) was 0.37 for Saltville residents and 0.22 for non-Saltville residents. The ratio of the prevalence ratios or odds ratio (OR) was 1.68 (Equation 3, Appendix A). The risk of developing a disease was 1.68 times higher for subjects living in Saltville, VA than the risk for those not living in Saltville, VA.

Effects of Selected Variables

Graphs illustrated the need to consider whether gender, race, age, smoking status, family history, and chronic diseases with similar symptoms had an effect on Saltville residence as a surrogate for exposure. Their effect was measured by calculating odds ratios for each (Tables 15 and 16). The odds ratios for family history and chronic diseases with similar symptoms included only subjects with diseases of interest to observe differences between Saltville residents and non-Saltville residents (Appendix C).

The odds ratio values for males ($OR_{MALE} = 1.63$) and females ($OR_{FEMALE} = 2.0$) were respectively lower and higher than the whole sample. The odds ratio for unreported races indicated an elevated risk but not as high as the OR of the entire sample.

Some groups had a higher OR than the OR_{sample}. This was true for subjects who were females, 31-45 years old, former smokers, family history of similar diseases, and chronic diseases with similar symptoms. Some groups had a lower OR than the OR_{sample}.

This was true for subjects who were males; 46-90 years old; no noted age; no noted race; non-smokers; smokers; family history of different diseases; and chronic diseases with different diseases. The odds ratio for Caucasians had a zero percent difference. This indicated that the elevation of risk was equal to the OR of the entire sample. The odds ratio value for non-smokers was very close to zero (percentage difference in $OR_{non\text{-}smoker}$ = -0.06). None of the subjects with an unknown gender had any diseases of interest. Their effect on the gender calculations is unknown.

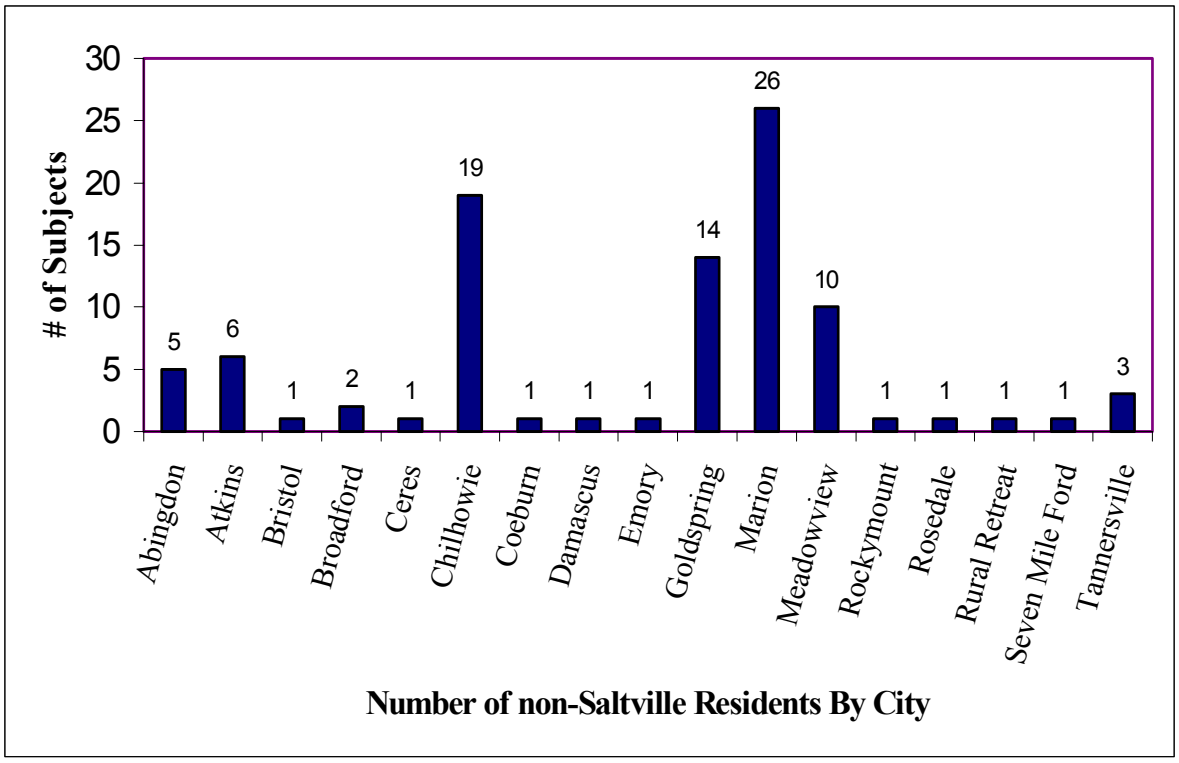

Figure 10 Virginia Cities of Residence for the Non-Saltville SMC Sample Members

Table 15 Odds Ratios of Demographic Variables

Variable	Categories	Odds ratio (OR) (95% Confidence Interval)	% Difference with OR_{Sample}
Gender	• Male • Female • Not noted	• 1.63 • 2.0 • N/A	• -.05% • +32% • --
Age	• 1-15 • 16-30 • 31-45 • 46-60 • 61-75 • 76-90 • Not noted	• N/A • N/A • 2.36 • 1.34 • 1.5 • 1.125 • 1.5	• -- • -- • +68% • -34% • -18% • -55.5% • -18%
Race	• Caucasian • Not Noted	• 1.7 (1.52,1.85) • 1.53 (1.27, 1.79)	• 0% • 17%

Table 16 Odds Ratios of Lifestyle Variables

Variable	Category	Odds Ratio (95% Confidence Interval)	% Difference with OR_{Sample}
Smoking Status	• Nonsmoker • Former smoker • Smoker	• 1.64 (1.45, 1.79) • 2.9 (2.33, 3.47) • 1.2 (0.95, 1.45)	• -.06% • +120% • -68%
Family History	• Related • Unrelated	• 1.9 (1.42, 2.38) • 1.3 (0.86, 1.74)	• 40% • -40%
Existing Chronic Disease	• Related • Unrelated	• 2.0 (1.51, 2.49) • 1.3 (0.87, 1.73)	• 50% • -40%

CHAPTER 5
DISCUSSION

The first null hypothesis was that no Saltville Medical Center patients had a disease characteristic of heavy metal exposure. This was rejected with 95% confidence because the sample contained SMC patients with diseases of interest. The second null hypothesis was that there was no difference between prevalence ratios of Saltville residents and non-Saltville residents. This was rejected with 95% confidence because the prevalence ratios yielded a 1.68 higher probability of having diseases from residing in Saltville.

Hypertension was the leading disease related to chronic exposure to lead. Some of the risk factors for hypertension are being over 60 years old, being a male, and heredity (Rizzo 1999). In this sample, the number of patients with hypertension was equal between the genders but the ratio was higher for males because more females comprised the sample (Figures 15 and 16). The majority of hypertension cases were in the older age groups (Figure 16). Gout was only present in males in this study. The contraction of gout is more common among males than females with hypertension as a risk factor (Anonymous 1992).

The effect of certain demographic factors and smoking status was measured to compare with the likelihood of having a disease from living in Saltville. Small sample size resulted in some instances. Some OR values were either unable to be calculated, were higher, or were lower than the OR_{sample}. These differences illustrated that data stratification altered sample size and produced biologically implausible results (i.e. lower risk for a smoker living in Saltville than a former smoker or non-smoker).

Another less obvious effect occurred when the sample size was calculated. The calculated sample size represented Saltville residents but the sample included patients not residing in Saltville. The representative sample size, with 95% confidence, was achieved at about 50% (n=96 Saltville subjects). A case-control study design might have been more appropriate in which 190 patients who live in Saltville would have been compared to 190 patients who did not live in Saltville. In spite of this, patients living in Saltville were 1.68 times more likely to have a disease associated with chronic exposure to the selected heavy metals. Risk is higher (OR=1.37) for central nervous system (CNS) birth defects from living within one mile of a facility that emits solvents or metals (Marshall et al. 1997).

The unexpected elevated and lowered risk values required further interpretation. The sample size and odds ratio (OR=1.64) for non-smokers were similar to the entire sample. This was also the case for Caucasians and unknown races. The sample size used to calculate the OR for former smokers was small (n=15) but resulted in a very high probability of having a disease. This suggested an interaction between tobacco use and Saltville residence. The length of cessation from use of tobacco products inversely relates to disease risk (Rea et al. 2002). The majority of the former smokers in the SMC sample had short cessation periods, thus it was plausible for their risk to be higher than the risk for current smokers. Lung cancer risk also decreases as the cessation period from smoking increases (CDC 2002).

The latency period for development of these chronic diseases may have been shorter for former smokers. The date of disease diagnosis was not in the medical record, so the latency period between Saltville residency and disease development could not be

determined. The sample size was larger for current smokers than former smokers, but the OR was only 1.2. The exposure to tobacco products was the more probable cause among smokers and former smokers. After the former smokers and current smokers were combined to form a group of "ever smokers," their odds ratio (OR=1.73) was higher than the non-smokers were. If a female smokes cigarettes then she has an 80% to 90% higher risk for death from all causes than a female who does not smoke (Centers for Disease Control and Prevention (CDC) 2002).

The small 95% confidence intervals justified the OR estimates. If the confidence intervals contained values of one or less then they were higher than those without a value of one or less (i.e. OR_{SMOKER}, $OR_{UNRELATED\ FAMILY\ HISTORY\ OF\ DISEASE}$, and $OR_{EXISTING\ CHRONIC\ DISEASE}$) (Friis and Sellers 1999).

No studies have assessed the adverse human health outcomes related to chronic exposure to presumably low levels of heavy metals in Saltville, Virginia (ATSDR 1987, 1992). The rates of stillbirth and low birth weight babies between mothers in wards near a landfill site in the United Kingdom are similar to mothers in wards not near the site (Fielder et al. 2000). Goldberg et al. (1999) compared risk of cancer development in men who lived near the Miron Quarry municipal solid waste landfill site in Quebec, Canada and men who lived in locations that are more isolated. Ratios of prevalence (odds ratios) are higher than expected for pancreatic cancer within a residential distance 1.25 kilometers (km) from the waste site; for liver cancer within 1.5 km; for kidney cancer within 2 km; and for non-Hodgkin's lymphomas within1 km (Goldberg et al. 1999).

Chapter 6

CONCLUSIONS AND RECOMMENDATIONS

Conclusions

The likelihood of having a disease associated with chronic exposure to mercury, arsenic, and lead was 1.68 times higher for Saltville residents than non-Saltville residents. Study outcomes reported in the literature review emphasized the necessity for disease surveillance of populations residing near hazardous waste sites. The epidemiological and statistical computations presented the diseases as quantifiable, adverse environmental outcomes.

The ATSDR, US U.S. EPA, and the Olin-Mathieson Corporation prove the presence of mercury in the environment. This study was performed to consider the consequential health outcomes from long-term exposure to mercury, arsenic, and lead.

Recommendations

- A mortality study of disease endpoints such as cardiovascular disease and renal failure from hypertension (Rizzo 1999) of the Saltville citizens. Death certificates and hospital records are excellent sources of data for this study design (Friis and Sellers 1999).
- Use of residential history (i.e. length of Saltville residency) and lifestyles (i.e. employed, retired, etc.) as exposure factors.
- Establishing a dose-response curve with job titles and employment history using Olin-Mathieson employee records and family interviews of former employees.
- Establishing environmental dose-response curves using environmental concentrations of all COCs disposed of in the SWDS and related diseases.

- Conduction of a probabilistic risk assessment in the Saltville community.

BIBLIOGRAPHY

Anonymous. Risk factors and incidence of gout in men. American Family Physician. 1992 v45n5.

Arromdee, E., Michet, C.J., Crowson, C.S. Incidence of Gout is on the rise. (Musculoskeletal Citations). The Journal of Musculoskeletal Medicine. 2003 v20i1.

Astor, B.C., Muntner, P., Adeera, L., Eustace, J.A., Coresh, J. Association of Kidney Function with Anemia: the Third National Health and Nutrition Examination Survey (1988-1994). Archives of Internal Medicine. 2002 v162i12.

ATSDR. 1987. Public Health Assessment for Saltville Waste Disposal Ponds.

ATSDR. 1992. Site Review and Update for Saltville Waste Disposal Ponds.

ATSDR. 1994. Health Consultation: Inactive Power Plant, Saltville Waste Disposal Site, Saltville, Virginia.

ATSDR. 1998. Health Consultation: Lagoons behind the Ice Plant (Pond 2) at Saltville Waste Disposal Ponds.

ATSDR. Case Studies in Environmental Medicine: Lead Toxicity. 1997.

ATSDR. Toxicological Profile of Arsenic. 1992.

ATSDR. Toxicological Profile of Mercury. 1993.

ATSDR. Toxicological Profile of Lead. 1999.

Backman, L. and Herlitz, A. The relationship between prior knowledge and face recognition memory in normal aging and Alzheimer's disease. Journals of Gerontology. 1990 v45 n3.

Balogh, S.B., Meyer, M.L., and Johnson, K.D. Mercury and suspended sediment loadings in the lower Minnesota River. Environmental Science and Technology. 1997 v31 n1.

Bhumbla, D.K., and Keefer, R.F. Arsenic Mobilization and Bioavailability in Soils. Nriagu, J.O. Arsenic in the Environment: Part 1: Cycling and Characterization USA: John Wiley and Sons, Inc., 1994:51-82.

Backman, L. and Herlitz, A. The relationship between prior knowledge and face recognition memory in normal aging and Alzheimer's disease. Journals of Gerontology. 1990 v45 n3.

Bleicher, S.J. Diabetes has link with many other diseases. Diabetes in the News. 1992 v11 n5.

British Columbia Ministry of Health and Ministry Responsible for Seniors. 1998 Reports on Cigarette Additives and Ingredients and Smoke Constituents. 1998.

CDC. Mercury Exposure in a Residential Community—Florida, 1994. MMWR 1995v44 n23.

CDC. Elemental Mercury Vapor Poisoning—North Carolina, 1988. MMWR 1989 v38n45.

CDC. Epidemiologic Notes and Reports Elemental Mercury Poisoning in a Household— Ohio, 1989. MMWR 1990 v39 n25.

CDC. Current Trends Acute and Chronic Poisoning from Residential Exposures to Elemental Mercury—Michigan, 1989-1990. MMWR 1991 v40 n23.

CDC. Human lead absorption-Texas. MMWR 1997 v46 n37.

CDC. Women and smoking: A report of the Surgeon General. Morbidity and Mortality Weekly Report. 2002 v51 i34.

Cebrian, M.E., Albores, A., Aguilar, M., and Blakely, E. Chronic Arsenic Poisoning in the North of Mexico. Human Toxicology. 1983 v2 i1.

Chakraborti, D., Rahman, M.M., Paul, K., Chowdhury, U.K., Sengupta, M.K., Lodh, D., Chanda, C.R., Saha, K.C., and Mukherjee, S.C. Arsenic Calamity in the Indian Subcontinent: What Lessons Have Been Learned? Talanta. 2002 v58.

Chase, D. and Lumpe, D. Not all memory loss is Alzheimer's. New Choices for the Best Years. 1991 v31n8.

Christ, P. Gout: A Complex Disorder. Ostomy Quarterly. 2000v37i3.

Davidson, C.I. and Rabinowitz, M. Lead in the Environment: From Sources to Human Receptors. Needleman, H., ed. Human Lead Exposure. Boca Raton: CRC Press, 1992: 65-88.

De Hertog, S.A.E. Tobacco and Skin Cancer. Family Practice News. 2001v31i7.

Driscoll, C.T., Cheng, Y. Schofield, C.L., Munson, R., Holsapple, J. The mercury cycle and fish in the Adirondack lakes. Environmental Science and Technology. 1994 v28 n3.

Dye, S.K. 1999. Mercury Contamination in Abiotic and Biotic Compartments in the North Fork of the Holston River, VA, TN [Thesis]: East Tennessee State University; 31p.

Eidson, M. and Tollestrup, K. Blood lead levels and remediation of an abandoned Smelter site. Journal of Environmental Health. 1995 v57 n9.

Ellingsen, D.G., Bast-Pattersen, R., Efskind, J., and Thomassen, Y. Neuropsychological Effects of Low Mercury Vapor Exposure in Chloralkali Workers. Neurotoxicology. 2001 v22.

Feng, Q., Suzuki, Y., and Hisashige, A. Hair Mercury Levels of Residents in China, Indonesia, and Japan. Archives of Environmental Health. 1998 v53 n1.

Fernandez, G.O., Martinez, R.R., Unam, T.I.F., and Palazuelos, E. High blood Lead Levels in ceramic fold art workers in Michoacan, Mexico. Archives of Environmental Health. 1997 v52 n1.

Fielder, H.M.P., Poon-King, C.M., Palmer, S.R., Moss, N., and Coleman, G. Assessment of Impact on Health of Residents Living Near the Nant-y-Gwyddon Landfill Site: Retrospective Analysis. British Medical Journal. 2000 v320 i7226.

Friis, Robert H. and Sellers, Thomas A. Epidemiology for Public Health Practice- 2nd edition. Gaithersburg, MD: Aspen Publishers, 1999.

Geerlings, M.I., Jonker, C., Bouter, L.M., Ader, H.J., Schmand, B. Association between memory complaints and incident Alzheimer's disease in elderly people with normal baseline recognition. American Journal of Psychiatry. 1999v156i4.

Goldberg, M.S., Siemiatyck, J., Dewar, R., Desy, M., and Riberdy, H. Risks of Developing Cancer Relative to Living near a Municipal Solid Waste Landfill Site in Montreal, Quebec, Canada. Archives of Environmental Health. 1999 v54 i4.

Grandjean, P., Weihe, P., White, R.F., Debes, F., Araki, S., Yokoyama, K., Murata, K., Sorensen, N., Dahl, R., and Jorgensen, P.J. Cognitive deficit in 7-year old children with prenatal exposure to Methylmercury. Neurotoxicology and Teratology. 1997 v19 n6.

Haggerty, M. Anemias. Gale Encyclopedia of Medicine: Edition 1. 1999

Harada, M., Nakanishi, J., Konuma, S., Ohno, K., Kimura, T., Yamaguchi, H., Tsuruta, K., Kizaki, T., Ookawara, T., and Ohno, H. The present mercury contents of scalp hair and clinical symptoms in inhabitants of the Minimata area. Environmental Research. 1998 v77 n2.

Hassan, A., Velasquez, E., Belmar, R., Coye, M., Drucker, E., Landrigan, P.J., Michaels,

D., and Sidel, K.B.
Holmes, S.E., Drutz, J.E., Buffone, G.J., and Rice, T.E. Blood lead levels in a Continuity Clinic population. Journal of Toxicology: Clinical Toxicology. 1997 v35 n2.

Hu, H., Watanabe, H., Payton, M., Korrick, S., and Rotnitzky, A. The relationship between bone lead and hemoglobin. The Journal of the American Medical Association. 1994 v272 n19.

Hu, H., Aro, A., Payton, M., Korrick, S., Sparrow, D., Weiss, S.T., and Rotnitzky, A. The relationship of bone and blood lead to hypertension: The Normative Aging Study. The Journal of the American Medical Association. 1996 v275 n15.

Hua, M.S., Huang, C.C., and Yang, Y.J. Chronic Elemental Mercury Intoxication: Neuropsychological Follow-up Case Study. Brain Injury. 1995 v10 n5.

Kafourou, A., Touloumi, G., Makropoulos, V., Loutradi, A., Papanagiotou, A., and Hatzakis, A. Effects of lead on the somatic. Archives of Environmental Health. 1997 v52 n5.

Kim, M., Nriagu, J., and Haack, S. Carbonate ions and arsenic dissolution by groundwater. Environmental Science and Technology. 2000 v35 i15.

Kishi, R., Doi, R., Fukuchi, Y., Satoh, H., Satoh, T., Ono, A., Moriwaka, F., Tashiro, K., Takahata, N. Subjective Symptoms and Neurobehavioral Performances of Ex-Mercury Miners at an Average of 18 years after the Cessation of Chronic Exposure to Mercury Vapor. Environmental Research. 1993 v62 i2.

Korrick, S.A., Hunter, D.J., Rotnitzky, A., Hu, H., and Speizer, F.E. Lead and Hypertension in a Sample of Middle-aged Women. American Journal of Public Health. 1999 v89 i3

Landrigan, P.J., Baker, E.L.Jr., Feldman, R.G., Cox, D.H., Eden, K.V., Orenstein, W.A., Mather, J.A., Yankel, A.J., and Von Lindern, I.H. Increased Lead Absorption with Anemia and Slowed Nerve Conduction in Children near a Lead Smelter. Journal of Pediatrics. 1976 v89 i6.

Lewis, R.O. Gout: Etiologies and treatments. (Clinical Podiatry). Podiatry Management. 2002 v21i4.

Lin, J., Tan, D., Ho, H., and Yu, C. Environmental Lead Exposure and Urate Excretion in the General Population. The American Journal of Medicine. 2002 v113.

Lin, J.L. and Huang, P.Y. Body Lead Stores and Urate Excretion in Men with Chronic Renal Disease. Journal of Rheumatology. 1994 v21 i4.

Litvan, I. Parkinsonian Features: When Are They Parkinson Disease? JAMA, Journal of the American Medical Association. 1998v280n19.

Lu, Frank C. Basic Toxicology: Fundamentals, Target Organs, and Risk Assessment- 3rd Edition. Washington, D.C.: Taylor and Francis, 1996.

Lubin, J.H., Pottern, L.M., Stone, B.J., and Fraumeni, J.F., Jr. Respiratory Cancer in a Sample of Copper Smelter Workers: Results from More Than 50 Years of Follow-up. American Journal of Epidemiology. 2000. v151 i6.

Lynch, R.A., Boatright, D.T., and Moss, S.K. Lead-Contaminated Imported Tamarind Candy and Children's Blood Lead Levels. Public Health Reports. 2000 v115 i6.

MapQuest, Incorporated. 2003. www.mapquest.com

Marshall, E.G., Gensburg, L.J., Deres, D.A., Geary, N.S., Cayo, M.R. Maternal Residential Exposure to Hazardous Wastes and Risk of Central Nervous System and Musculoskeletal Birth Defects. Archives of Environmental Health. 1997 v52 n6.

Mason, R.P. and Sullivan, K.A. Mercury in Lake Michigan. Environmental Science and Technology. 1997 v31 n3.

Mathiesen, T., Ellingsen, D.G., and Kjuus, H. Neuropsychological effects associated with exposure to mercury vapor among former chloralkali workers. Scandinavian Journal of Work, Environment, and Health. 1999 v25 n4.

Mazumder, D.N., Gupta, J.D., Chakraborty, A.K., Chatterjee, D.D., and Chakraborti, D. Environmental pollution and chronic arsenicosis in South Calcutta. Bulletin of The World Health Organization. 1992 v70 n4.

McCullough, J.E., Dick, R., and Rutchik, J. Chronic Mercury Exposure Examined with a Computer-Based Tremor System. Journal of Occupational and Environmental Medicine. 2001 v43 n3.

Mok, W.M. and Wai, C.M. Mobilization of Arsenic in Contaminated River Waters. Nriagu, J.O., ed. Arsenic in the Environment: Part 1: Cycling and Characterization. USA: John Wiley and Sons, Inc., 1994: 99-118.

Morel, F. M.M., Kraepiel, A. M. L., and Amyot, M. The chemical cycle and bioaccumulation of mercury. Annual Review of Ecology and Systematics. 1998 v29.

Moss, M.E., Lanphear, B.P., and Auinger, P. Association of Dental Caries and Blood Lead Levels. The Journal of the American Medical Association. 1999 v281 i24.

Murgueytio, A.M., Evans, G.R., Sterling, D.A., Clardy, S.A., Shadel, B.N., and Clements, B.W. Relationship between Lead Mining and Blood Lead Levels in Children. Archives of Environmental Health. 1998 v53 i6.

Newton, R.R. and Rudestam, K.E. Your Statistical Consultant: Answers to Your Data Analysis Questions. Thousand Oaks, CA: SAGE Publications, Inc. 1999.

Phillips, D.J.H. The Chemical Forms of Arsenic in Aquatic Organisms and Interrelationships. Nriagu, J.O., ed. Arsenic in the Environment: Part 1: Cycling and Characterization. New York: John Wiley and Sons, Inc., 1994: 263-288.

Pittman, J.R. and Bross, M.H. Diagnosis and Management of Gout. American Family Physician. 1999v59i7.

Pocock, S.J., Smith, M., and Baghurst, P. Environmental lead and children's intelligence: a systematic review of the epidemiological evidence. British Medical Journal. 1994 v309 n6963.

Powell, T.J. Chronic Neurobehavioural Effects of Mercury Poisoning on a Group of Zulu Chemical Workers. Brain Injury. 2000 v14 n9.

Rea, T.D., Heckbert, S.R., Kaplan, R.C., Smith, N.L., Lemaitre, R.N., and Psaty, B.M. Smoking status and risk for recurrent coronary events after myocardial infarction. Annals of Internal Medicine. 2002v137i6.

Rizzo, T. Hypertension. Gale Encyclopedia of Medicine, Edition 1. 1999: 1521.

Rosenblatt, J. Basic Statistical Methods and Models for the Sciences. Boca Raton: CRC Press. 2002.

Rothenberg, S.J., Manalo, M., Jiang, J., Cuellar, R., Reyes, S., Sanchez, M., Diaz, M., Khan, F., Aguilar, A., Reynoso, B., Juaregui, M., Acosta, S., and Johnson, C. Blood Lead Level and Blood Pressure During Pregnancy in South Central Los Angeles. Archives of Environmental Health. 1999 v54 i6.

Rothenberg, S.J., Schnaas, L., Perroni, E., Hernandez, R.M., and Karchmer, S. Secular trend in blood lead levels in a sample of Mexico City children. Archives of Environmental Health. 1998 v53 n3.

Rothenberg, S.J., Schnaas, L., Perroni, E., Hernandez, R.M., and Ortega, J.F. Blood Lead Secular Trend in a Sample of Children in Mexico City II: 1990-1995. Archives of Environmental Health. 2000 v55 i4.

Rule, J.H. and Iwashchenko, M.S. Mercury concentrations in soils Adjacent to a Former Chlor-Alkali Plant. Journal of Environmental Quality. 1998 v27 n1.

Santschi, P.H., Allison, M.A., Asbill, S., and Perlet, A.B. Sediment transport and Hg recovery in Lavaca Bay, as evaluated from radionuclide and Hg distributions. Environmental Science and Technology. 1999 v33 n13.

Saunders, C.S. Gout: Applying Current Knowledge. Patient Care. 1998 v32 n10.

Schell, L.M., Czerwinski, S., Stark, A.D., Parsons, P.J., Gomez, M., and Samelson, S. Variation in Blood Lead and Hematocrit Levels during Pregnancy in a Socioeconomically Disadvantaged Population. Archives of Environmental Health. 2000 v55 i2.

Schwartz, B.S., Stewart, W.F., Todd, A.C., Simon, D., and Links, J.M. Different Associations of Blood Lead, Meso 2, 3- Dimercaptosuccinic Acid (DMSA) Chelatable Lead, and Tibial Lead Levels with Blood Pressure in 543 Former Organolead Manufacturing Workers. Archives of Environmental Health. 2000 v55 i2.

Schwartz, J. Lead, Blood Pressure, and Cardiovascular Disease. Needleman, H., ed. Human Lead Exposure. Boca Raton: CRC Press, 1992: 233-242.

Smith, Allan H., Lingas, Eleana O., and Rahman, Mahfuzar. Contamination of Drinking Water by Arsenic in Bangladesh: a public health emergency. Bulletin of the World Health Organization. 2000 v78 i9.

Smith, J.C., Allen, P.V., and Burg, R. Von. Hair Methylmercury Levels in U.S. Women. Archives of Environmental Health. 1997 v52 n6.

Soong, W.T. Long-Term Effect of Increased Lead Absorption on Intelligence of Children. Archives of Environmental Health. 1999.

SPSS Version 11.0, SPSS, Inc. Chicago, IL.

SPSS Base 10.0 Applications Guide. SPSS, Inc. USA, 1999.

Stohrer, G. Arsenic: opportunity for risk assessment. Archives of Toxicology. 1991

Straten, M.V., Carrasco, D., Paterson, M.S., McCrary, M.L., Meyer, D., Tyring, S.K. Tobacco Use and Skin Disease. Southern Medical Journal. 2001v94i6.

Tondel, Martin. The Relationship of Arsenic Levels in Drinking Water and the Prevalence Rate of Skin Lesions in Bangladesh. The Journal of the American Medical Association. 1999 v282 i22.

Tumpowsky, C.M., Davis, L.K., and Rabin, R. Elevated Blood Lead Levels Among Adults in Massachusetts, 1991-1995. Public Health Reports. 2000 v115 i4.

Turkington, C.A. Tremors. Gale Encyclopedia of Medicine, Edition 1. 1999: 2908.

U.S. Census Bureau. 2000. Population Estimates for 2000.

U.S. EPA Enviromapper **http://maps.U.S. EPA.gov/enviromapper.** 2003.

U.S. EPA. IRIS Substance File of Mercury (elemental). 1998.

U.S. EPA. IRIS Substance File of Lead and compounds (inorganic). 1998.

U.S. EPA. IRIS Substance File of Arsenic (inorganic). 2000.

U.S. EPA. Mercury Study Report to Congress Volume 3: Fate and Transport of Mercury in the Environment. 1997.

U.S. EPA. 2000. General Site Information: Saltville Waste Disposal Ponds.

Wang, M.Q., Fitzhugh, E., Westerfield, R.C. Determining Sample Size for Simple-Random Surveys. Health Values. 1995 v19 i3.

Wedeen, R.P. Lead, the Kidney, and Hypertension. Needleman, H., ed. Human Lead Exposure. Boca Raton: CRC Press, 1992:169-190.

Wiggins, S.A. Parkinsonian Tremors. Clinical Reference Systems. 2001.

Wu, T., Shen, C., Lai, J., Goo, C., Ko, K., Chi, H., Chang, P., and Liou, S. Effects of Lead and Noise Exposures on Hearing Ability. Archives of Environmental Health. 2000 v55 n2.

Yan-Chu. Nriagu, J.O., ed. Arsenic in the Environment: Part 1: Cycling and Characterization. New York: Wiley, 1994.

Zavariz, C., and Glina, D.M. Clinico-neuro-psychological Evaluation of Workers Exposed to Metallic Mercury in the Electric Lamp Industry. Reviews of Saude Publica. 1992 v26 i5.

APPENDIX A

Equations

- **[Equation 1]** Sample size (n) = $2.706N\ (p\text{-}p^2) / (N\text{-}1)\varepsilon^2 + 2.706(p\text{-}p^2)$
    - N=population size (total number of medical records)= 9,350
    - p=population proportion to be estimated (assuming all Saltville residents attend the Saltville Medical Center) =2,204/9,350=0.236
    - ε=permissible error= +/- 0.04 (extent to which the results may differ if the whole population had been surveyed)
    - Sample size= $2.706 * ((9{,}350 * (0.236\text{-}0.236^2)) / (9{,}350\text{-}1) * (0.04^2) + 2.706 * (0.236\text{-}0.236^2)) = 190$

- **[Equation 2]** Prevalence rate of exposed (non-exposed) = number of exposed subjects with exposure-related disease / total number of exposed (non-exposed)
- **[Equation 3]** Odds ratio = prevalence rate of exposed/prevalence rate of non-exposed
- **[Equation 4]** Etiologic fraction = Odds ratio – 1/ Odds ratio

APPENDIX B

Data Collection Form

ADDRESS
----------DETACH HERE
ID#_____

DISTANCE FROM SWDS_____
GENDER:
MALE___
FEMALE___
AGE____

EXPOSED (SALTVILLE) _____
REFERENCE (NON-SALTVILLE) _____

RACE **Family History (list)**
CAUCASIAN____
BLACK____
HISPANIC____
NATIVE AMERICAN___
OTHER___
Occupation_____
Smoking Status_____

SYMPTOMS/DISEASES

***TREMORS____** *****HYPERTENSION _____**
***MEMORY DISTURBANCES____** *****ANEMIA____**
*****GOUT____**
****HYPER/HYPOPIGMENTATION_____**
****KERATOSIS____**

CONFOUNDING DISEASES
***PARKINSON'S DISEASE_____** *****NEPHROPATHY_____**
***ALZHEIMER'S DISEASE____** *****OBESITY_____**
****SKIN CANCER_____** *****DIABETES MELLITUS_____**
*****SICKLE CELL ANEMIA_____**

*MERCURY-RELATED SYMPTOMS
**ARSENIC-RELATED SYMPTOMS
***LEAD-RELATED SYMPTOMS

APPENDIX C

2x2 Tables

Table C.1. Family History of Disease (Related/ Unrelated)

	Disease Status	
	Yes	No
Saltville Residency		
Yes		
No		

Table C.2. Existing Chronic Diseases (Related/Unrelated)

	Disease Status	
	Yes	No
Saltville Residency		
Yes		
No		

Table C.3. Non-Smoker

	Disease Status	
	Yes	No
Saltville Residency		
Yes		
No		

Table C.4. Former Smoker

	Disease Status	
	Yes	No
Saltville Residency		
Yes		
No		

Table C.5. Current Smoker

	Disease Status	
	Yes	No
Saltville Residency		
Yes		
No		

VITA

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Natural Science, B.S., 1998
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