AN EVALUATION OF LEAD HAZARDS IN PRE-1978 RESIDENTAL HOUSING WITHIN CLARK COUNTY, NEVADA, USA

by

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Bachelor of Arts San Francisco State University 2003

A thesis submitted in partial fulfillment of the requirements for the

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ABSTRACT

An Evaluation of Lead Hazards in Pre-1978 Residential Housing Within Clark County, Nevada, USA

by

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Dr. Shawn L. Gerstenberger, Examination Committee Chair Professor of Environmental and Occupational Health University of Nevada, Las Vegas

Lead poisoning remains a public health concern due to leads persistence in the environment from anthropogenic uses. Initial efforts to address the impacts of lead on vulnerable communities have included secondary prevention measures which typically occur after a child has been poisoned. However, in recent years there has been a pragmatic shift toward primary prevention efforts.

This study evaluates lead hazards identified through primary prevention activities in residential housing within Clark County, Nevada, USA. It is the first study to systematically address and evaluate pre-1978 housing in Southern Nevada. Risk assessments were conducted in 81 dwellings built prior to 1979 classified as a mobile, single family or multi family unit. Risk assessments included the evaluation of lead hazards using a Niton X-ray Fluorescence analyzer and collection of dust and soil samples.

Of the 81 homes inspected 60 (74%) of the homes had lead-based hazards from traditional (paint, dust, soil) and non-traditional (tile) sources. Evaluation of lead-based

paint hazards suggest that hazards within Clark County follow national trends, specifically that lead is found more frequently in older housing units. An evaluation of age of the home as a marker for dust and soil hazards indicated no statistically significant trend which could conclude that year of construction is not associated with such hazards. Data does suggest that non-traditional lead-based hazards such as tile are more commonly found in Southern Nevada Homes.

Understanding housing characteristics, such as age of home, and their association to paint, dust and soil hazards can be used to develop targeted screening and education efforts that actively evaluate housing with the greatest risk of exposing humans to lead. Further, the identification of non-traditional lead hazards as a common source of exposure for Nevada residents indicates the need for prevention and educational efforts that address reducing risks associated with such hazards.

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CHAPTER 1

INTRODUCTION

Childhood lead poisoning continues to be a considerable public health concern. According to the Centers for Disease Control and Prevention(CDC), 8.9 percent of preschoolers are affected, making lead poisoning the foremost preventable childhood disease (Farr, 1996). The National Health and Nutrition Examination Surveys (NHANES) reports that the average blood lead level (BLL) in the late 1970's, for children ages one to five years was 14.9 microgram per deciliter (ug/dL), with over 13 million children affected. As of 2000, the average BLL for children ages 1-5 had decreased to 2.2 ug/dL impacting over 400,000 children (NHANES, 2009). The NHANES data displays a significant decrease in the number of lead poisoned children within the United States and an overall decrease in blood lead concentrations (Table 1).

Federal, state and local regulations have played a significant role in reducing childhood lead poisoning by regulating the use of lead in specific products, such as paint and gasoline (Kemper, Cohn, Fant, Dombkowski, & Hudson, 2005). Nevertheless, the potential for childhood exposure to lead remains high, particularly due to the stability of lead in the environment, usage of lead in numerous industrial applications and widespread usage of lead-based paint in older housing. Recent studies have associated lower blood lead concentrations (<10 ug/dL) with negative health outcomes. In a study conducted by Lanphear (2007), BLLs once thought to pose little to no risk have shown to be risk factors for reading problems, intellectual delays, school failure, attention deficit-hyperactivity disorder and antisocial behavior (Lanphear, 2007).

In attempts to mitigate effects of childhood lead poisoning many efforts have been initiated among schools of public health, public health departments, and health care professionals comprising a secondary prevention initiative (Satcher, 1997). The main focus of these efforts is to "prevent" lead toxicity by screening children and identifying those who have blood lead levels ≥ 10 ug/dL (Lanphear, Hornung, & Ho, 2005). Although a secondary prevention approach is critical in addressing immediate health concerns of affected communities it fails to address the root cause of the disease; the sources of lead exposure. Incorporating housing-based screenings to identify hazards can enhance our efforts in reducing lead poisoning (Lanphear, Hornung, & Ho, 2005). As a formidable goal housing-based primary prevention efforts must embody "prevention" initiatives that conduct screenings to identify hazards, prior to a child being poisoned, before tenancy into a new home, and after remediation (Lanphear, 2008).

Year	Geometric Mean ¹	Prevalence ³ of	Estimated Number
	BLLs (95% CI ²⁾	BLLs >=10 μ g/dL ⁴	of Children with
		(95% CI)	BLLs >= $10\mu g/dL$
			(95% CI)
1976 - 1980	14.9	88.2%	13,500,000
	(14.1 - 15.8)	(83.8 - 92.6)	(12,800,000 -
			14,100,000)
1988 - 1991	3.6	8.6% ⁵	1,700,000
	(3.3-4.0)	(4.8-12.4%)	(960,000-2,477,000)
1991 – 1994	2.7	4.4%	890,000
	(2.5-3.0)	(2.9-6.6%)	(590,000-1,330,000)
1999 - 2000	2.2	2.2%	434,000 ⁶
	(2.0-2.5)	(1.0-4.3%)	(189,000-846,000)

Table 1. NHANES blood lead level measurements in children aged 1-5 years of age

Source: (NHANES, 2009)

The University of Nevada Las Vegas School of Community Health Sciences and The Childhood Lead Poisoning Prevention Program (CLPPP) coordinated through the Southern Nevada Health District, have incorporated primary and secondary prevention approaches to address childhood lead poisoning in Clark County. However, little is known about hazards within the housing stock. It is estimated that 94,000 homes within Clark County where built prior to 1978 (SNHD, 2007). Most of the homes serve as permanent places of residence for Hispanics, African-Americans, and lower socioeconomic status subgroups (Housing-Needs Assessment 2005-2009 Consolidated Plan, 2004).

While a great deal of knowledge has propelled lead prevention for the last 40 years, the majority of efforts have focused on secondary prevention avenues, initiated after a child has been lead poisoned. This project focuses on housing-based primary prevention efforts conducted through residential risk assessments on homes constructed prior to 1979. The study will be the first to systematically evaluate, address and determine the extent of lead hazards in Southern Nevada housing.

Children who have been lead poisoned can suffer from a vast number of health effects. A growing body of research has identified key areas of concern: 1) health effects identified below our current level of concern of 10 ug/dL (Canfield, Henderson, & Cory-Slechta, 2003; Jusko, Henderson, Lanphear, Cory-Slechta, Parsons, & Canfield, 2008; Lanphear, Dietrich, Auinger, & Cox, 2000; Rogan & Ware, 2003; Schwartz, 1994), 2) the importance of primary prevention efforts in circumventing lead poisoning in children (Brown, 2008; Farr, 1996; Needleman, 1998; Lanphear, 2005; Lanphear, Hornung, & Ho, 2005), 3) additional routes of exposure through non-traditional sources (Gorospe & Gerstenberger, 2008), 4) disproportionate amount of children affected from low socioeconomic and minority status (Kemper, Cohn, Fant, Dombkowski, & Hudson, 2005; Lanphear, Weitzman, & Elberly, 1996) and 5) homes built prior to 1979 who have hazards that have not been remediated (Jacobs et al., 2002).

A majority of lead-based hazards from traditional (ie. paint, dust, soil) and nontraditional sources (ie. tile) are a result of exposures that come directly from the environment in which the child lives. Thus, the home environment becomes a key determinate of health and avenue to prevent lead toxicity in children. Low screening rates within the county and the lack of lead-based housing data warrant the need to evaluate lead-based hazards in Southern Nevada. Evaluation of exposure sources would allow public health officials to effectively target and educate high-risk communities.

Specific aims of this project are: 1) to establish housing-based primary prevention efforts that target at risk populations within Clark County, 2) to identify traditional leadbased paint hazards (paint, dust, soil) that are prevalent within the county and 3) to identify the prevalence of non-traditional lead hazards (tile, porcelain tubs).

One of the fundamental keys to housing-based primary prevention is to implement screening of high-risk, older housing units to identify lead hazards; and should function as the core of lead poisoning prevention programs in order to circumvent lead poisoning in children (Committee on Enviornmental Health, 2005; Farr, 1996; Lanphear, 2005; Needleman, 1998; Phelps, Ryan, Quinn, Malon, & Scott, 2004). Farr (1996) elaborates that prevention efforts thwart painful medical interventions such as chelation therapy, reduce health-care costs, and advances our national educational goal of sending children to school "ready to learn" (Farr, 1996).

CHAPTER 2

REVIEW OF RELATED LITERATURE

Physical Properties

Lead is found within the periodic table under the elemental symbol Pb. Its atomic number is 82 and has an atomic weight of 207.2 (McMurry & Fay, 2001). Physical properties that make the use of lead, as a abundant as it is include, its low melting point of 327°C and high boiling point of 1749°C. Lead has many isotopic forms, of which five occur naturally. Three of the stable isotopes, lead-206,-207, and -208 are the end products of the radioactive decay of uranium, actinium and thorium (Lansdown & Yule, 1986). Uranium and thorium are abundantly found in the earth's crust while actinium is formed as part of the decay process of uranium. Uranium can undergo a series of alpha and beta decays to produce stable lead isotopes -206 and -207 (Figure 1)



Figure 1. The decay of uranium-238 to stable lead-206

Source: (Burns, 2002)

However, unlike other metals, lead is not found in the environment in its metallic state and is strictly obtained from ores, galena (PbS), found in the earth's crust. Lead ores are mined throughout the world. Lead properties, such as corrosion resistance, density, and low melting point, make it a familiar metal in pipes, solder, weights and storage batteries (ATSDR, 2007).

Health Outcomes

Lead poisoning can cause a variety of health problems. The effects of lead toxicity can be observed in various systems within the body including the neurological system, the reproductive system, the cardiovascular system, and the kidneys (Jacobs, 1996; Myers, Davidson, Weitzman, & Lanpher, 1997). Lead interferes with blood cell formation, resulting in anemia. Lead can cause kidney damage, sterility, miscarriage and birth defects (Nadakavukaren, 2006). Depending on the degree of exposure, symptoms of lead poisoning can be hyperirritability, poor memory, or sluggishness at lower concentrations and mental retardation, epileptic convulsions, coma, and death at higher concentrations (Nadakavukaren, 2006).

Younger children are especially susceptible to the toxic effects of lead because their blood-brain barrier and skeletal systems are still developing (Gracia & Sondgrass, 2007; Goldstein, 1992; Goyer, 1990). Those at highest risk for developing adverse health effects due to lead exposure are children, between one and four years of age, exhibiting pica type behavior (ingestion of nonfood substances) and hand-to-mouth activity (Heneman & Zidenberg-Cherr, 2006; Mielke & Reagan, 1998). Absorption of lead has been found to be five to ten times greater in children than in adults (Alexander, 1974; Gracia & Sondgrass, 2007; James, Hilburn, & Blair, 1985; Ziegler, Edwards, Jensen,

Mahaffey, & Fomon, 1978). In addition, to young children, pregnant women, and those who are malnourished can absorb 40-70 percent of ingested lead (Gracia & Sondgrass, 2007).

Ingesting lead poses the greatest hazard, especially for young children. In adults only 10 percent of lead swallowed passes from the intestine into the bloodstream. In children it is found that 40 percent of lead ingested by preschoolers remains within their bodies, making children the highest risk group for lead poisoning (Nadakavukaren, 2006). Once lead enters into the body it competes with calcium used in the bone. This competitive behavior results in high concentrations of lead in growing bones. In children, this affects fast growing bones of the body such as, the distal femur, both ends of the tibia, and distal radii (Khan, Munir, Turnbull, & MacDonald, 2009).

Adverse consequences of low-level lead exposure include intellectual impairments, behavioral problems, and delinquency which can persist into adolescence (Lanphear, Hornung, & Ho, 2005). In a study conducted by Lanphear, Dietrich, Auinger, & Cox (2000) researchers found that lead exposure affected reading score performance and their results suggest that cognitive deficits are associated with blood lead concentrations lower than 5ug/dL (Lanphear, Dietrich, Auinger, & Cox, 2000). Jusko et al. (2008) found that childhood blood lead concentrations are inversely related to IQ scores. They observed this relationship, regardless of whether lead exposure was measured by lifetime and infancy average measures, maximal (peak) exposure, or the same day the IQ test was administered (Jusko, Henderson, Lanphear, Cory-Slechta, Parsons, & Canfield, 2008). The study further demonstrated that intellectual functioning of children is impaired at BLLs below the CDC's level of action of 10 ug/dL. Miranda et

al. (2007) found that blood lead levels in early childhood are related to educational achievement in early elementary school as measured by performance on end-of-grade testing (Miranda, Kim, Galeano, Paul, Hull, & Morgan, 2007).

Lead has also been associated with health deficits resulting from pre-natal exposure. Early exposure to lead may interfere with synaptogenesis, disrupt apoptosis, lower levels of serotonin and increase levels of D-aminolevulinic acid, which may antagonize gamma-Aminobutyric acid (GABA) inhibition (Dietrich, Ris, Succop, Berger, & Bornschein, 2001). Dietrich, Ris, Succop, Berger, & Bornschein (2001) report a significant relationship between low level prenatal lead exposure and behavioral problems in adolescents (Dietrich, Ris, Succop, Berger, & Bornschein, 2001). Opler et al. (2004) suggest that prenatal exposure to lead and/or elevated δ -aminolevulinic acid, may extend into later life and must be further investigated as risk factors for adult psychiatric diseases (Opler et al., 2004).

In a prospective birth cohort study conducted by Wright et al. (2008), researchers following 250 individuals measuring prenatal and postnatal blood concentrations found that childhood blood lead concentrations were predictors for adult arrest (Wright et al., 2008). In a case control study conducted by Needleman, McFarland, Ness, Fienberg & Tobin (2002) researchers found that in addition to leads direct action on the brain and impulse control, lead exposure can increase risk for delinquency through a separate, indirect route; impaired cognitive function and classroom performance (Needleman, McFarland, Ness, Fienberg, & Tobin, 2002).

Braun et al. (2006) concluded that exposure to both prenatal tobacco and environmental lead are risk factors for attention deficit hyperactivity disorder (ADHD) in

U.S. children (Braun, Kahn, Froehlich, Aulinger, & Lanphear, 2006). Another study suggests that environmental exposure to lead may delay growth and pubertal development in girls (Selevan, Rice, Hogan, Euling, Pfahles-Hutchens, & Bethel, 2003).

Health outcomes resulting from lead exposure are vast and wide. Screening and diagnosis add to the complexities of identifying lead poisoning cases. Most children who suffer from the effects of lead poisoning will have subclinical disease (ie, the presence of disease without symptoms) (Markowitz, 2000). Thus, many children experiencing lead poisoning may be overlooked due to a lack of physical symptoms. Such perils emphasize that a more effective intervention for lead toxicity is early identification and removal of the lead source, in order to prevent exposure (Gracia & Sondgrass, 2007).

Sources of Exposure

Lead has been mined and used by mankind for 6,000 years, and the history of lead poisoning is nearly 2,500 years old (Hernberg, 2000). Physical and chemical properties of lead have contributed to its vast anthropogenic uses. Throughout antiquity and into the preindustrial period lead was widely used both for industrial, domestic and medicinal purposes (Hernberg, 2000). However, the industrial period marked a critical time in which, the use of lead increased significantly. As lead products became prevalent in all aspects of modern life, such as ornamental use, home use, and industrial use, sources of exposure also expanded. During the Industrial Revolution, the greater number of people employed in factories and the increased use of lead in industrial processes, led to an increase in lead poisoning from occupational exposure (Lansdown & Yule, 1986). This would explain the increase in lead poisoning cases in adults. However, it was not until the late 1800's when the first published reports described afflicitons of children experiencing

lead colic. The twentieth century would not only come with dynamic social and economic changes, but it would also bring a greater understanding of the many routes of lead exposure, and consequently, its impacts on health.

Lead in Paint

The Environmental Protection Agency (EPA) banned lead from consumer paint use in the U.S. in 1978, by enacting the Toxic Substance Control Act (TSCA). However, its long term use as an additive to paint to provide color and durability has resulted in a vast number of children who have been poisoned by lead. Although the residential use of lead-based paint is no longer available, lead poisoning cases still remain a public health concern. Studies indicate that 25 percent of all U.S. housing has significant lead-based hazards (Rothweiler, Cabb, & Gerstenberger, 2007). Deteriorating paint found in peeling, chipping, chalking and/or flaking condition can put young children at risk. High impact surface areas and areas damaged by structural problems are of particular concern. Thus, implementing proper precautions becomes an area of priority to reduce lead-based paint contamination of dust or soil, through deterioration or disruption during maintenance, repainting, remodeling, demolition, or paint removal (Jacobs et al., 2002).

Lead in Soil

One route of exposure to lead is from soil. The presence of lead in soil likely resulted from deterioration of products containing lead, specifically paint and gasoline. Lead exhibits a clear industrial legacy, with millions of tons of anthropogenic lead used in lead-based paint and leaded gasoline still largely present in the surface (Figure 2) (Mielke, Gonzales, Powell, & Mielke, 2008). Leads stability and propensity to travel long distances, contribute to its ability to cause environmental contamination. Its binding

ability to soil particles, contributes to soil contamination observed in the 21st century, commonly termed "legacy soil". Soil acts as a reservoir of lead dust and becomes an environmental pathway or carrier of lead from sources to humans (Mielke, 1994). The use of lead, as an additive to paint has posed a significant hazard. During the natural aging process, the paint in homes built prior to the ban of lead-based paint in 1978, is no longer stable and has significantly contributed to soil contamination.

However, spatial factors play an important role in concentrations of lead found in soil such as, traffic volume and proximity to buildings and highways. Studies conducted by Button (2008) & Motto, Daines, Childo, & Motto (1970) found that soil contamination is highest in areas that are in closest proximity to buildings that contain lead-based paint and/or are in proximity to highly trafficked highways.



Figure 2. Lead usage in paint and gasoline measured in metric tons

Source: (Laidlaw & Filippelli, 2008)

Lead in Air

The major sources of lead emissions have historically been motor vehicles (such as cars and trucks) and industrial sources (Hilton, 1998; Romieu, Lacasana, McConnell, 1997; Tong, Von Schimding, & Prapamontol, 2000). The use of leaded gasoline has contributed to high rates of poisoned children. However, EPA regulations have significantly contributed to the reduction of lead particles released into the air via motor vehicles (McCarthy, 2008). Subsequently, after regulations banned the use of lead in gasoline, childhood lead poisoning cases decreased (Mushak & Crocetti, 1990; Pirkle, Kaufmann, Brody, Hickman, Gunter, & Paschal, 1998).





Source: (EPA, 2008)

The major risk of exposure to lead particulates in the air result from, occupational exposure and proximity to lead smelters (Petrosyan, Orlova, Dunlap, Babayan, Farfel, & Braun, 2004). Lead emissions within Nevada and Clark County are highest in non-road equipment (i.e., used for construction or agriculture) and industrial processes (Figure 3

and 4). Inhalation of lead, through the respiratory system, allows for an immediate and wide distribution and absorption into the body.



Figure 4: Lead emissions by source sector in Clark County in 2002

Lead in Water

Sources of lead in water are derived from the manufactured use of lead in the production of pipes, solders and materials used for plumbing. Homes built prior to 1986, before the Federal Lead Contamination Control Act was passed, are at greatest risk for drinking water contamination where lead can leach from the solders or pipes, into the water supply (Berkowitz, 1995). Increased velocity, hot temperatures, soft water (low mineral content), aged piping, and acidity create conditions favorable for leaching of lead (Bryant, 2004).

Source: (EPA, 2008)

Lead in Non-Traditional Sources

The single largest use of lead, accounting for over 70% of the total U.S. consumption of the metal, is for lead storage batteries. Other lead-containing products include ammunition, brass, coverings for power and communication cables, glass TV tubes, solder and pigments (Nadakavukaren, 2006). Not surprisingly, lead is now found throughout the environment – in soils, water, air, and food (Nadakavukaren, 2006). Studies continue to report that the influx of atypical sources, such as unregulated imports (ceramic cookware), ethnic remedies and accessories, candy, chocolate and poorly controlled nutritional supplements and other apparently harmless products such as toys, continue to perpetuate the movement of environmental lead into situations of potential human exposure (Gorospe & Gerstenberger, 2008; Heneman & Zidenberg-Cherr, 2006). The vast number of products containing some degree of lead content adds to the complexities in identifying sources of lead poisoning. In an evaluation of lead hazards found in homes within the U.S., Jacobs et al. (2002), reports that half of XRF measurements above 1.0 mg/cm^2 in newer units were on painted tile or stone substrates (Jacobs et al., 2002).

Regulations

Federal, state, and local agencies establish regulations and policies to protect adults and children from adverse effects of lead poisoning (Rothweiler, Cabb, & Gerstenberger, 2007). In 1971, the Lead-Based Paint Poisoning Prevention Act (LBPPPA), was enacted to prohibit lead-based paint in residential structures constructed or rehabilitated by the Federal government or with Federal assistance (HUD, 2004). Although this addressed some immediate concerns to reducing exposures for vulnerable

populations, an issue remained with homes that did not satisfy the category of Federal housing. Non-federally subsidized housing remained a source of exposure to millions of children. In 1978, the Consumer Product Safety Commission (CPSC) banned the residential use of lead-based paint containing greater than or equal to 0.06 percent or 600 parts per million (ppm) of lead and disallowed the use of lead-based paint in all residential housing (HUD, 2004). Continued efforts such as the Residential Lead-Based Paint Hazard Reduction Act of 1992 (P.L. 102-550), known as Title X, adopted the concept of primary prevention and established a strategic approach for allocating initial resources to the worst lead hazards (Farr, 1996).

Other political efforts in the U.S. focused on reducing lead exposure in drinking water, such as the amendments to the Safe Drinking Water Act, signed into law in December 1986. These amendments banned the use of solders and flux containing more than 0.2 percent lead (solders and flux were typically composed of 40 percent to 50 percent lead) and restricted the allowable amount of lead to less than 8 percent in any brass or other material intended to be in contact with water (Maas, Patch, Morgan, & Pandolfo, 2005). The EPA's action level is 15 parts per billion (ppb) for public water suppliers, schools and nonresidential building (Bryant, 2004; EPA, 1994).

Following various epidemiological and surveillance studies examining lead poisoning, the CDC, repeatedly lowered its definition of an elevated blood lead concentration. The current CDC blood lead level of concern is 10 ug/dL (Rogan & Ware, 2003). Figure 5. Illustrates the decrease in the blood lead concentration of concern over the years. Recommended levels of action guide public health agencies in addressing childhood and adult poisoning cases.



Figure 5. CDC-recommended level of action for blood lead concentrations in children

Source: (CDC, Case studies in environmental medicine lead toxicity, 2007)

A variety of laws and federal agencies have served to regulate the use of lead. In turn, modifications of how lead is used have acted as a way to mitigate environmental exposures to lead and reduce lead poisoning cases in children and adults

Prevention

Efforts to reduce lead poisoning in children typically entail a secondary prevention component. After a child has been identified through a blood test as poisoned (> 10 ug/dL), action may be taken to investigate the home environment in order to identify and address lead hazards (Farr, 1996). Unfortunately, this strategy fails to prevent the adverse consequences of lead exposure since the child with an elevated blood lead level is used as a trigger to control lead hazards (Lanphear, Hornung, & Ho, 2005).

In a retrospective study conducted by Kemper, Cohn, Fant, Dombkowski & Hudson (2005) it was found that almost half of the children with elevated blood lead levels did not complete follow-up testing although they had at least one medical encounter within six months of the initial elevated blood lead level. The study found a failure to conduct follow-up testing disproportionately impacts: nonwhite children, those living in urban areas or in communities with a high risk of lead exposure, and those living in area's with the greatest number of elevated screening blood lead levels (Kemper, Cohn, Fant, Dombkowski, & Hudson, 2005). An implication of the Kemper et al. study is that secondary prevention efforts can fail to protect children from adverse consequences of lead toxicity (Lanphear, 2005; Lanphear, 1998).

Although the number of housing units with lead paint hazards has decreased dramatically since 1990, an estimated 24 million housing units still contain this poisonous material (Jacobs et al., 2002). Thus, the most important factor in controlling lead poisoning is reducing the child's exposure to lead (Zierold, Havtena, & Anderson, 2007).

A primary prevention approach emphasizes identifying and correcting lead hazards in housing before a child is poisoned, thus shifting the focus from the child to the environmental exposure sources (Farr, 1996). Studies have shown that timely remediation of lead hazards in homes of children with elevated BLLs has a protective effect on decreasing lead levels in the blood (Leighton, Klitzman, Sedlar, Mattle, & Cohen, 2003; Staes, Matte, Gilbert, Flanders, & Binder, 1994; Taha, Kanarek, Schultz, & Murphy, 1999; Zierold, Havtena, & Anderson, 2007). Thus, identification of a lead source and prevention of child exposures is critical (Committee on Environmental Health, 1993; Needleman, 1998).

The shift towards primary prevention has experienced many hurdles, mainly due to the high cost of abatement, the scarcity of adequately trained lead-abatement

professionals, and the absence (until 1995) of Federal guidelines for implementing less costly methods of leaded paint hazard containment (Pirkle, Kaufmann, Brody, Hickman, Gunter, & Paschal, 1998). Thus residential lead-based paint-abatement efforts have primarly focused on homes of children with an elevated BLL (i.e., secondary prevention), rather than on homes that have the potential to expose a child to lead (i.e., primary prevention).

In the movement towards primary prevention it is critical to screen high-risk, older housing units to reduce lead hazards that can led to a child being lead poisoned. In addition, primary prevention components which encompass screening housing before a family moves in and after lead remediation activities are critical in maintaining a safe home (Lanphear, 2005). Inspection of painted surfaces will not only reveal whether the paint poses an immediate hazard (eg, peeling, flaking, or chalking), but allow the opportunity to educate families on potential hazards such as currently intact lead-based paint surfaces (Markowitz, 2000). Studies have found that low cost procedures such as enclosing window wells with aluminum and wet-scrapping, as well as repainting walls with latex paint, have decreased BLLs as much as 22 percent (Taha, Kanarek, Schultz, & Murphy, 1999; Zierold, Havtena, & Anderson, 2007).

A pragmatic shift towards primary prevention can reduce exposures and reduce adverse toxicological outcomes resulting from environmental hazards such as lead. Screening and follow-up testing of high-risk children will remain an important part of lead poisoning prevention programs, but as we move towards primary prevention efforts, screening should serve as a safety net, not the focus (Lanphear, 2005).

Lead Poisoning as a Health Disparity

Lead poisoning afflicts children across all socioeconomic strata and in all regions of the country (Farr, 1996). In some cities, especially those in the Northeast and Midwest, the prevalence of children with blood lead levels exceeding 10 ug/dL is considerably higher than in the rest of the U.S. (Brown, Shenassa, Matte, & Catlin, 1997; Lanphear, Auinger, & Schaeffer, 1998; Lanphear, Hornung, & Ho, 2005; Sargent, Brown, Freeman, Bailey, Goodman, & Freeman, 1995). However, because lead hazards are most severe in older, deteriorating housing, people of color and the inner-city poor are disproportionately affected (Farr, 1996).

A study conducted by Lanphear, Weitzman & Eberly (1996), researchers found that housing conditions and exposure to lead-contaminated house dust appear to be major contributors to the racial disparity in children's blood lead levels (Lanphear, Weitzman, & Elberly, 1996). A study conducted by Sargent et al. (1995), identified correlations between socio-demographics and housing conditions. The project took place in the state of Massachusetts, where universal lead screening was required for children from 9 months to 4 years of age. The study results showed that children living in communities with high rates of poverty, single-parent families, and pre-1950s housing and low rates of home ownership were seven to ten times more likely to have lead poisoning (Sargent, Brown, Freeman, Bailey, Goodman, & Freeman, 1995).

Blood lead surveillance data reported to the CDC in 2001 confirmed that Hispanic children have a higher prevalence of elevated BLLs than do children from the general population (Vallejos, Strack, & Aronson, 2006). In addition to exposure to lead in paint, there are at least three other sources of lead exposure that are more common among

families of Mexican origin than among other groups: ceramic cooking pots with lead glaze, some imported candies from Mexico, and certain home remedies used for the treatment of stomach ailments (Vallejos, Strack, & Aronson, 2006). A variety of cultural and socio economic factors can put minority populations at greater risk for lead poisoning. Thus, continued outreach with minority communities, coupled with grassroots involvement to both identify the problem and its solutions, is critical for closing the health disparities gap (Vallejos, Strack, & Aronson, 2006).

Summary

The toxicological impact of lead on health has been well documented. Physical and chemical properties that make lead a durable and inexpensive metal have resulted in its widespread usage in products from centuries ago to the present day. Consequently, sources of exposure have expanded beyond what is traditionally expected (i.e., paint, soil, dust, water) to include a diversified group of manufactured products that contain lead (i.e., tile, mini-blinds).

National data identifies specific housing characteristics such as age of the home to determine high risk groups for lead exposure. However, there is no published data on lead hazards and housing characteristics for Nevada. Based on the literature it is clear that housing-based primary prevention is a critical component in meeting Healthy People 2010 goals to eliminate childhood lead poisoning. This study will be the first study to evaluate the following items in Clark County, Nevada: the proportion of traditional and non-traditional hazards found in pre-1978 housing and the relationship between age of the home and traditional hazards such as paint, dust and soil.

CHAPTER 3

QUESTIONS, OBJECTIVES, AND HYPOTHESES

Questions

- Are lead hazards found in pre-1978 homes in Southern Nevada a result of traditional or non-traditional sources?
- Does the age of the home determine the presence or absence of lead-based paint hazards?
- Will the age of the home be related to lead dust concentrations?
- Is the age of the home related to soil concentrations of lead?

Objectives

- To determine if the most common lead hazards found in a pre-1978 home risk assessments are traditional lead-based paint hazards or non-traditional hazards.
- To determine if the age of home correlates with the presence or absence of leadbased paint.
- To determine if dust and soil are sources of lead hazards.

Hypotheses

Lead Hazards of Painted & Non-Painted Surfaces

• The most common lead hazard found in pre-1978 homes in Clark County, Nevada will be from nontraditional sources, specifically non-painted surfaces.

Painted surfaces, a common traditional source of lead, are defined as painted wood, drywall, stucco and siding. Non-painted surfaces, also known as non-traditional sources of lead, are identified as tile, porcelain, vinyl, plastic, ceramic and metal. A binomial test will be used to evaluate the probability of traditional and non-traditional lead sources found during a risk assessment.

Age of Home & Lead-Based Paint Hazards

• Older homes are more likely to contain lead-based paint hazards.

Lead-based paint hazards are identified using a Niton XRF. Values must reach or exceed EPA standard of 1 mg/cm².

Lead- based paint hazards will be dichotomized into absence and presence of lead paint hazards. A histogram will be used to detect to break years of construction into decades. A Point Biserial Correlation and likelihood ratio test will be utilized to determine the association between year of construction and the absence and presence of hazards with a significance of $\alpha = 0.05$.

Age of the Home & Lead Dust Concentrations

• Dust samples collected from older homes will have a higher concentration of lead upon collection.

Dust samples are collected from surfaces that have exceeded 1 mg/cm² by XRF analysis. Homes will be grouped by year of construction based on trends identified by a histogram. A one-way ANOVA will be used to determine if there is a difference between mean dust concentrations across percentiles of year of construction. If ANOVA results are significant a post hoc test will be utilized to determine which group differs.

Age of the Home & Lead Soil Concentrations

• Older homes will have higher concentrations of lead in the soil.

Soil samples are collected from bare soil surrounding the home. Age of homes will be broken down into quartiles based the number of homes inspected across years of construction. A Pearson's r correlation will be used to determine the direction of the relationship between year of construction and soil concentration. A one way ANOVA will be utilized to determine if there is a difference between soil concentrations across years of construction with a significance of $\alpha = 0.05$.

CHAPTER 4

METHODOLOGY

Participants/Recruitment

Participants were recruited through non-systematic recruiting strategies. The majority of referrals were obtained from the Clark County Childhood Lead Poisoning Prevention Program (CLPPP). In addition, collaborative efforts with local agencies, such as, Rebuilding Together provided additional volunteers. Various media efforts through television, newspaper, brochures and flyers were utilized to recruit participants. Eightyone owners/renters from the Southern Nevada area, with residences built prior to 1979 participated in the study. Participants represent an opportunistic sample since all volunteered to participate in the study. All dwellings included in the study were constructed prior to 1979. The age of construction was verified through the Clark County Assessor Office website, whenever possible. However, some homes located in rural communities or on tribal lands were not available for verification through the Clark County Assessor Office.

Procedure

Risk assessments were conducted according to protocols set forth by Environmental Protection Agency (EPA), Department of Housing and Urban Development (HUD), and the American Society for Testing and Materials (ASTM) (Jacobs, 2004). Specific protocols for the collection of paint, dust, soil, water and other items were closely followed and are described in more detail below. In addition to dust, soil and water collection, investigators administered a questionnaire (Appendix 1) at each home to determine potential sources of exposure related to remodeling, repainting and/or repair. A Buildings and Conditions Form (Appendix 2) was completed for each home, to evaluate the overall condition of the home and to identify any areas of the home that can impact health (ie. moisture, mold, excess dust). A map of the home was drawn to identify all major areas of the dwelling and to notate where samples were obtained for future reference. Any material found in the home is considered a hazard if it exceeds the established standards listed in Table 2.

Paint Sampling

Interior and exterior painted surfaces were tested using a Niton XLp 300A/700A Series X-Ray Fluorescence Analyzers (XRF) which contains a ¹⁰⁹cadmium radioactive isotope source. The XRF is able to identify the presence of lead in paint. The cadmium

Soil	Play area	400 ppm
	Other	1200 ppm
	Must abate	5000 ppm
Water	Municipal – motionless for	15 ppb
	at least 6 hrs.	
Dust	Floor	40 ug/ft^2
	Window sills	250 ug/ft^2
	Window trough	400 ug/ft^2
	Concrete	800 ug/ft^2
Paint	XRF	$1.0 \text{ mg/cm}^2 = 5000 \text{ppm}$
		=.5%
	New paint	600ppm=.06%
Air	Permissible Exposure Level	30 ug/m^3
Blood Lead Levels	Children	10 ug/dL
(CDC level of concern)	Adult	25 ug/dL
	Occupational	40 ug/dL

Table 2. Standards used to establish residential lead hazards during risk assessments

Source : (Rothweiler, Cabb, & Gerstenberger, 2007)
source, within the XRF, emits gamma rays (Niton, 2009). When gamma rays come into contact with lead on painted surfaces, electrons within lead are excited (Figure 6). The electrons are ejected, thus emitting its own x-ray with an energy frequency that is unique to lead (Bilets, 2006). This energy is then measured by the XRF instrument and the concentration of lead in the paint sample can be determined.

In accordance, with EPA protocols all rooms in the interior of the home are tested. In addition, exterior surfaces of the home are analyzed for lead-based paint. Photographs are taken of all items that exceed the EPA standard of 1.0 mg/cm² for lead-based paint. A paint sampling log (Appendix 3) is completed for each assessment. The log includes; case number, address, date of investigation, sample number, substrate, component, location, condition, XRF reading, and any additional notes.



Fig. 6: Niton XRF excitation model of lead detection

Source: (Niton, 2009)

Dust Sampling

Prior to collection of dust samples, areas of the home to be sampled are identified based on positive (>1 mg/cm²) XRF readings and high friction and impact surfaces. Dust samples are collected according to EPA, HUD and ASTM guidelines (Jacobs, 2004). Risk assessors wear disposable gloves during each dust sample collection and new gloves are used for each sample gathered. No other surfaces are touched during the collection of samples. Areas to be sampled are measured using a template with an area of 0.25 ft² for window sills and 0.5 ft² for all other samples.

Once the area to sample is measured, a sterile dust wipe is removed from a sealed package. The wipe is opened, and placed in the palm of the hand grasping the wipe firmly between the thumb and the palm. Three different passes are taken within the measured area (Figure 7).

	Figure 7: Step	by step	process used	l to collect	dust sample
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First pass "S" shaped motion	Second pass Top-bottom motion	Third pass Perimeter motion
\rightarrow		$ \longrightarrow \longrightarrow \longrightarrow \longrightarrow \downarrow $
\leftarrow		$ \uparrow \qquad \downarrow$
\rightarrow	\downarrow \downarrow	$\begin{vmatrix} \uparrow \\ \downarrow \\$
~~		$ \uparrow \qquad \qquad \downarrow \\ \uparrow \leftarrow \leftarrow \leftarrow \leftarrow \downarrow$

The first pass takes a side to side "S" like motion wiping the interior of the marked off area. The wipe is then folded in half with the collected sample inside. The second pass, is

then taken with a top to bottom "S" like motion. The wipe is then folded one last time and the inside perimeter is wiped. The dust sample is then placed into a corresponding marked test tube. Dust samples are analyzed by a NLLAP laboratory for lead concentration using National Institute for Occupational Safety and Health (NIOSH) 9100/7082 analytical methods and flame atomic absorption spectroscopy (FAAS) techniques (CDC, 2003). A dust sampling log (Appendix 4) is completed for each assessment. The log includes; sample number, case number, date of collection, area of surface wipe, location of where wipes were taken, and description of component wiped. Soil Sampling

Soil samples are collected from homes in which bare soil is visible at the time of investigation. Specifically, samples are taken from the front yard, back yard or any identified children's play areas. Risk assessors wear disposable gloves when taking samples. A coring device with a T handle, which is cleaned in between each collection, is used in order to core into the ground about 0.5 inches into the soil. Depending on the area of the bare soil a composite of three to six samples is taken per area. Soil samples are sent to an NLLAP laboratory to be analyzed for lead concentrations using EPA 3050B/7420 analytical methods and graphite furnace atomic absorption spectroscopy (GFAAS) techniques (CDC, 2003). A soil sampling log (Appendix 5) is completed for each assessment. The log includes, sample number, case number, date of collection, and number of composites taken.

Water Sampling

Water samples were collected from homes in which owners/renters indicated a possible presence of lead based pipes in the home. A one liter water collection container

was left at each home where sampling was determined necessary. Homeowners/renters were instructed to collect a cold water sample first thing in the morning, before any of the plumbing had been used. Thus, collection of water is a "first draw" (Appendix 6) sample following a six to eight hour stand time. The risk assessor returned the next day to retrieve the sample. Water samples are analyzed by a NLLAP laboratory for lead concentration using SM 3113B analytical methods and GFAAS techniques (CDC, 2003). Questionnaire

A questionnaire (Appendix 1) was completed at each risk assessment. The questionnaire gathered information to identify potential sources of exposure, due to remodeling and repainting in the home or nearby homes. Information gathered also identified important items such as; year of construction, proximity to lead industries, to roadway, freeway, elevated highway or other transportation structures (must be within two blocks of home), and the use of blinds and shutters in the home.

Risk Assessment Report

Once all samples collected were analyzed, the risk assessor wrote a report identifying the actual and potential lead hazards found in the home. Actual hazards were determined based on the condition of the component identified with lead-based content that exceeded EPA/HUD guidelines. Typically, actual hazards were found in dwellings with cracking, chalking, chipping paint and overall poor conditions. Potential hazards found in the home were identified as components that posed no immediate threat (found intact or otherwise in good condition) at time of investigation, but contained lead above established standards (Table 2). All actual and potential hazards were photographed at the time of the investigation and were included in the reports with their respective location.

The risk assessment report contained recommendations on how to minimize exposure to lead hazards including, information on interim controls, abatement and/or remediation. Completed reports and cover letters were sent to the SNHD and to the owner/renter. Data Analysis

Data analysis was conducted using SPSS for Windows® Version 17.0. The following tests were performed: Binomial test, likelihood ratio test and a one way ANOVA, as detailed in the hypothesis section.

Quality Control Measures

Various measures were taken throughout the study for quality control purposes. The Niton XRF is calibrated before and after each investigation. Three calibration readings are taken and verified to be within 0.8 to 1.2 mg/cm². In addition, ten repeat readings are taken within the home at the end of sampling, at approximately the same location, to ensure accuracy of reading taken by the equipment. A dust wipe blank is collected each time dust samples are obtained to ensure wipes meet ASTM standards and cross-contamination has not occurred. Further NLLAP certified laboratories follow standard quality assurance and quality control protocols.

CHAPTER 5

RESULTS

A total of 94 lead risk assessments were scheduled throughout the study period. Lead risk assessments not meeting the qualification of a single family or multi-family dwelling or mobile home were excluded from analysis, leaving a total of 81 units.

		Total Sample (n=81)		
Age of primary resident				
Mean/SD		57.38/18.21		
Range		22-88		
Mode/Median		70/64		
Total family members				
living in home				
Mean/SD		3.12/1.83		
Range		1-9		
Mode/Median	2/3			
	#	%		
Gender				
Male	24	29.6		
Female	51	63.0		
Ethnicity				
Caucasian	26	32.1		
African American	26	32.1		
Hispanic/Latino	17	17		
Asian	1	1		
Native American	1	1		
Other	4 4			
Families with 1 + child				
under 6	22	27.1		

Table 3. Characteristics of participating residents

Note: Questionnaire was not available at the start of the study thus gender and ethnicity do not total 100%. Demographic data available for 75 of the 81 homes. The primary resident of the home granted access and completed a risk assessment questionnaire. The mean age of the primary resident was 57 years of age with a standard deviation (SD) of 18 years (Table 3). The total number of family members living in the home ranged from one to nine members, with a mean of 3.12 and SD of 1.83. Twenty-seven percent of the homes where a lead risk assessment was conducted had one or more child under the age of six. Fifty one percent of the primary residents were female. The majority of participants identified themselves as Caucasian or African American (32% respectively) followed by the next highest ethnic category being Hispanic/Latino (17%).

		Total Sample: n=81
	#	%
Residence type		
Single family unit	73	90.2
Multi family unit	4	4.9
Mobile home	4	4.9
Year of Construction		
<= 1950	6	7.4
1951-1960	9	11.1
1961-1970	41	50.6
>= 1971	25	30.9
Geographic Areas		
North Las Vegas	29	35.8
City of Las Vegas	24	29.6
Unincorporated	23	28.4
Henderson	3	3.7
Boulder City	2	2.5

Table 4. Characteristics of homes investigated in Clark County, Nevada

Lead risk assessments were conducted in mobile homes and single family and multi-family units (Table 4) constructed prior to 1979. Year of construction for the

sample ranged from 1939 to 1978. Corresponding breakdowns by decade are reported in Table 4. The majority of risk assessments were investigated in North Las Vegas (35.8%), City of Las Vegas (29.6%), and unincorporated locales of Las Vegas (28.4%). The remaining investigations were completed in Henderson and Boulder City.

Lead Hazards: Painted & Non-painted Surfaces

A total of 8,289 XRF readings were collected, accounting for 1,771 non-painted surfaces and 6,518 painted surfaces. Values below EPA/HUD standards and values used for quality control where excluded from analysis. Painted surfaces and non-painted surfaces with lead content at or above 1 mg/cm² were found in 60(74%) of the 81 homes where lead risk assessments were completed. Final count obtained was 222 lead positive readings for non-painted surfaces and 170 for painted surfaces (Table 5). A total of 49 surfaces where identified and categorized into painted and non-painted surfaces. A frequency distribution of lead hazards is found in Table 6.

8	0 0	1	1
T 1	T 1		
Lead	Yes	No	Total
Non -Painted Surfaces	222 (3.4)	6296 (96.6)	6518
Painted Surfaces	170 (9.6)	1601 (90.4)	1771
Total	392 (4.7)	7897 (95.3)	8289

Table 5. Readings above 1 mg/cm² using an XRF for painted and non-painted surfaces

Binomial test: non-painted surfaces=56.6%, painted surfaces=43.4%; p=0.01

The most common lead hazards found in the study came from non-painted surfaces. Of the 60 homes with identified lead hazards 40 (66.7%) of the homes contained hazards

Surfaces	Hazards	Relative Frequency
Painted		
Wood	147	.3750
Drywall	18	.1582
Stucco	3	.0077
Siding	2	.0051
Non-Painted		
Tile	141	.3546
Ceramic	27	.0689
Vinyl	15	.0383
Porcelain	13	.0332
Metal	10	.0255
Plastic	4	.0026
Linoleum	3	.0077
Brick	3	.0077
Concrete	3	.0077
Laminate	1	.0026
Pyrex	1	.0026
Caulking	1	.0026
Total	392	

Table 6. Frequency distribution of painted and non-painted surfaces $> 1 \text{ mg/cm}^2$

from non-painted surfaces only and 5 (8.3%) contained hazards from lead-based painted surfaces. A total of 15 (25%) homes where identified with both non-painted and painted hazards above 1 mg/cm^2 .

A binomial test examining the proportion of painted versus non painted surfaces in the home resulted in a significant percentage of non-painted surfaces (ie, tile and ceramic products), which contained lead-based paint above EPA/HUD standards (nonpainted surfaces=56.6%, painted surfaces=43.4%; p=0.01). Therefore, there is a higher probability of finding non-painted lead surfaces in homes located in Clark County.

Age of Home and Lead-Based Paint Hazards

Year of construction was obtained for the 81 homes inspected. A visual breakdown of year of construction across the study is displayed using a histogram with an overlay of a normal distribution curve (Figure 8). The histogram was used to identify natural breaks in the data and to categorize age of home by decades as described in Table 4. Lead-based paint hazards for each home were dichotomized into two categories: presence or absence of hazards.



Figure 8. Year of construction of homes tested for lead hazards in Clark County, Nevada

Values for skewness and kurtosis were found within anticipated distributions: $-1 \le$ skew $\le +1$ and $-1 \le$ kurtosis $\le +1$. Skewness describing the symmetry of the distribution

was measured at a value of -0.924 indicating scores are clustered to the right. Kurtosis providing information about the peakedness of the distribution was found at a value of 0.912 indicating that the distribution is clustered in the centre forming a peak with long thin tails.

Year of construction and presence or absence of lead-based paint hazards was not found to be independent of one of another. A Point Bi-Serial correlation, indicates a negative correlation between the two variables, r = -0.543, n = 81, p < 0.001,

		<= 1950	1951-1960	1961-1970	>= 1971	Total
Lead-	Observed	5	4	3	1	13
Paint Hazard	Expected	1	1.4	6.6	4	13
No	Observed	1	5	38	24	68
паzаги	Expected	5	7.6	34.4	21	68
Total	Observed	6	9	41	25	81
	Expected	6	9	41	25	81

Table 7. Observed & expected frequencies for lead-based paint hazard and year of construction

Likelihood ratio test $G^2 = 8.331 \text{ p} < 0.05 \text{ for n} = 81$

therefore age of home is associated with the presence of lead-based paint hazards. Year of construction and lead-based paint hazards are significantly correlated using the likelihood ratio test, $G^2 = 8.331 \text{ p} < 0.05$, indicating the presence of lead-based paint hazards are associated with older housing (Table 7).

Age of Home and Lead Dust Concentrations

Age of the home was analyzed using periods of construction as described above. Dust samples were collected in 51 of the homes. A total of 156 samples, excluding samples utilized for quality control, were collected. The number of samples collected per home ranged from one to 12 samples with a mean of three samples per home. The number of samples collected varied based on the number hazards found in the home. Due to an unequal distribution of samples collected in each home an average dust concentration per dwelling was calculated and used for analysis after weighting for the number of samples taken in each home.

Lead dust concentrations below the limit of detection were found in 114 (73.1%) of the 156 samples collected. Half the limit of detection was substituted for any value that was found below the limit of detection for purposes of analysis. Only four of the total samples collected exceeded EPA/HUD standards.

No trend was found between year of construction and mean dust concentration using a Pearson's r correlation, r = -0.001 p > 0.05. A one-way between-groups analysis of variance was conducted to explore the impact of year of construction, measured by mean dust concentration. Year of construction was divided into categories listed in Table 8. The univariate analysis of variance indicated year of construction and dust concentration are not statistically significant, F= 0.035 p > 0.05. The mean scores between groups were small and were reflected in the effect size calculated using eta squared, at 0.002. An insignificant F test indicates that the mean dust concentrations across percentiles for year of construction are equal. Non-parametric results for a Kruskal Wallis

confirmed that no statistical difference across decades of construction exists, $x^2 = 1.20$ exact p = 0.764.

		Dust Concentration PPM
Year of Construction	# of homes	Mean ± Standard Deviation
<= 1950	6	26.41 ± 27.79
1951-1960	8	26.14 ± 37.23
1961-1970	23	30.02 ± 38.02
>= 1971	14	27.36 ± 36.92
Total	51	28.26 ± 35.56

Table 8. Mean dust concentration per decade of construction

Note: A total of 156 samples collected from 51 homes. ANOVA: F= 0.035 p >0.05

Age of Home and Lead Soil Concentrations

Age of the home and relationship to soil concentration was analyzed using periods of construction as described above. Soil samples were collected in 71 of the homes with a total of 106 samples collected. Only one sample exceeded EPA/HUD standards. The number of samples collected per home ranged from 1 to 3 samples with a mean of 1.49 samples. Due to an unequal distribution of samples collected in each home an average soil concentration was calculated and used for analysis. The average soil concentration within each decade is reported in Table 9.

The relationship between the average soil concentration and year of construction was investigated using a Pearsons r correlation which indicated no association between the two variables, r = 0.024, n = 71, p > 0.05. A one-way between-groups analysis of variance was conducted to explore the impact of year of construction, measured by mean soil concentration. Year of construction was divided into categories listed in Table 9. The univariate analysis of variance indicated year construction and soil concentrations are not statistically significant, F = 0.423 p > 0.05. An insignificant F test indicates that the mean soil concentrations across percentiles for year of construction are equal.

Table 9. Mean soil concentration per decade of construction

		Soil Concentration PPM
Year of Construction	# of homes	Mean ± Standard Deviation
<= 1950	5	80.90 ± 68.82
1951-1960	10	58.75 ± 47.91
1961-1970	35	40.65 ± 27.43
>= 1971	21	90.93 ± 300.20
Total	71	67.81 ± 164.88

Note: A total of 106 samples collected from 71 homes ANOVA: F = 0.423 p 0.737.

CHAPTER 6

DISCUSSION AND CONCLUSIONS

Lead Hazards: Painted and Non-painted Surfaces

National research indicates that common sources of lead exposure to humans, is a result of living in older homes with lead-based paint (Rothweiler, Cabb, & Gerstenberger, 2007). This study evaluated lead-based hazards within the housing stock of Clark County, Nevada. The goal of the study was to determine if hazards in residential housing are more commonly found in painted surfaces such as wood, drywall, and stucco or in non-painted surfaces such as tile. The results suggest that there is a higher probability of finding lead-based hazards in non-traditional surfaces in residential homes within Clark County.

Of the homes investigated, 76 of the dwellings had tile with lead content above 1 mg/cm². Although it is unknown whether lead is in the tile itself or a component of the glazing process when it is manufactured, the data does suggest the need to better understand tile as potential source of lead exposure. The present study agrees with much of the research contributing to our knowledge of non-traditional sources of exposure from non-painted items, but is limited in explaining whether sources of exposure can result from tiled surfaces in the home. The data suggest: 1) the need to understand tile as a source of exposure, 2) the need to provide primary education on lead tile remediation, 3) the need to develop protocols for removal and remediation of tile and 4) the need to expand protocols to identify the presence of lead in tile in post-1978 housing.

Age of Home and Lead-Based Paint Hazards

Estimates indicate that 25 percent of the nation's housing has significant leadbased paint hazards in the form of deteriorating paint (Jacobs et al., 2002). Specifically, housing constructed before 1960 has been to found to have rates five to eight times the prevalence of lead hazards compared with units built between 1960 and 1978 (Jacobs et al., 2002). While other studies indicate that homes built prior to 1950 pose the greatest risk for exposure to lead (Zierold, Havtena, & Anderson, 2007).

Nationally, lead-based paint hazards are most commonly found amongst the oldest housing segments. This study is the first of its kind to systematically evaluate housing in Nevada and determine if identified lead hazards follow national trends. In review of lead-based paint hazards found in Clark County, the data suggests homes within Southern Nevada exhibit a similar trend to nationally observed trends and reflect hazards found within the population of homes built in Las Vegas.

Statistical analysis indicates that year of construction and presence or absence of hazards was not independent. However, this could be a result of the unequal distribution of homes investigated per decade of construction. Utilizing the likelihood ratio statistic a statistically significant association between years of construction (as categorized per decade) is associated with the existence of lead-based paint hazards. Of the 81 homes inspected 13 (16.0%) of the homes had one or more lead-based paint hazards found in poor condition. While an additional 13 homes had lead hazards in good condition at the time of inspection. Of these 26 homes containing lead above acceptable standards 6 (7.4%) of them had both actual and potential hazards.

Clark County is currently the home of an estimated 1.9 million residents and, as of 2006, home to 771,000 housing units. Prior to the ban of lead-based paint (1978), Clark County had approximately 94,000 homes and a population of approximately 127,000 residents. While the number of homes built prior to 1979, is far less in comparison to cities across the nation, the data suggest that lead-based paint hazards in older housing are of concern and indicate the need to continue primary prevention efforts that educate residents about hazards associated with living in older housing.

Furthermore, from a primary prevention standpoint the presence of potential hazards (i.e. lead-based paint found intact) could necessitate the need to integrate followup visits into primary prevention programs to assist families in re-evaluating areas of the homes that contain lead. This would enable public health officials to evaluate if recommended remediation or abatements have occurred and to determine if previous areas of the home with potential hazards have deteriorated. Follow-up schedules would assist in evaluating the effectiveness in primary prevention efforts

Age of Home and Lead Dust Concentrations

An estimated 16% of all housing units nationwide (equivalent to 15.5 million units) have one or more lead dust hazards on either floors or windowsills (Jacobs et al., 2002). Dust hazards are more likely to be found in homes with deteriorating lead-based paint that can be associated with older housing units. Lead in dust is one of the primary exposures that led to lead poisoning, especially among children with extensive hand to mouth behavior.

Of the 81 homes inspected, dust samples were collected from 51 of the homes, and of the 51, four homes had lead dust hazards. In three of the homes lead-based paint

was detected, but only one of three homes had lead-based paint in deteriorating condition. One of the homes with high lead dust levels had no lead-based paint found in the home, but did have broken and deteriorating tile with a lead concentration above 1 mg/cm². In homes were paint was intact, it is possible that previous rehabilitation, maintenance, and repair could have removed lead-based paint hazards in other areas of the home, but contributed to a lead dust hazard through improper remediation (Jacobs et al., 2002). Indicating, that families maybe unaware of hazards that result from disturbing areas of the homes with lead content.

In review of lead-based dust hazards found in Clark County, the study suggests that lead dust concentrations are not associated with age of the home. Although homes built between 1961 – 1970 contained a higher mean concentration of lead it was not found to be statistically significant. The lack of an association between year of construction and lead dust concentration could be a result of 1) the unequal number of samples collected among each home inspected, 2) the unequal distribution of homes classified per percentile, and 3) small samples in terms of homes being inspected.

Age of Home and Lead Soil Concentrations

An estimated 5% (equivalent to ~4.9 million units) of housing units nationwide have play area soil lead concentrations \geq 400 ppm, as defined by U.S. EPA/HUD standard (Jacobs et al., 2002). Soil hazards are more likely to be found in dwellings with deteriorating lead-based paint which can result in contamination of surrounding areas of the home.

This study aimed to determine if lead contaminated soil was a prevalent hazard within the county. Samples were collected from 71 of the homes resulting in only one

with a soil hazard according to EPA/HUD standards. The home was constructed in 1974 with deteriorating lead-based paint on the exterior and interior of the home.

In review of lead-based soil hazards found in Clark County, the study suggests that lead soil concentrations are not associated with age of the home. The lack of an association between year of construction and soil concentration could be a result of a variety factors or a combination of them all: 1) small sample size, 2) housing and city wide characteristics; 3) the overall number of homes constructed in Clark County prior to the ban of leaded gasoline in 1978, and their proximity to major highways and roadways that existed at the time, and 4) temporal components unaccounted for in the statistical model could result in lower soil concentrations. Las Vegas, in comparison to cities found in the South and Midwest, has a lower prevalence of lead hazards, perhaps due to the relative young age of the city. A study conducted by Laidlaw and Filippelli (2008) indicates that high temperatures and low humidity, which decrease soil moisture during the summer months, are conditions that can resuspend soil and dust particulates into the atmosphere. The majority of home investigations conducted for the study were done from fall through the beginning of spring at a time when atmospheric conditions did not allow for capturing resuspended soil.

Research Limitations

The study was designed using a convenient, non-random sampling method that limits its ability to be representative of the general population. The sample size, n=81 and an unequal distribution across years of construction makes it difficult to explain the degree of variability among relationships as not due to mere chance.

Summary

A shift toward primary prevention initiated in Clark County has allowed us to collect baseline data on lead hazards that exist within the housing stock. This is the first dataset evaluating pre-1978 housing for lead hazards in Nevada. Consistent with US trends, it was expected to find lead-based paint hazards in older homes.

This study indicates that non-typical sources of lead exposure, such as tile, are prominent in southern Nevada, as they are found more often than traditional lead-based paint. Although sample size limitations limit the ability to generalize these findings across the U.S., products such as tile are widely distributed across the nation and are commonly found in homes regardless of geographic location. Thus, understanding tile as a source of exposure is necessary and may require the development of occupational standards to protect the health of workers.

Lead and soil dust concentration appear to be insignificant issues for Clark County residents. Allowing us to progressively work towards achieving Healthy People 2010 objectives to reduce and eliminate childhood lead poisoning. Nevertheless, lead in paint remains of significant concern specifically for our oldest housing subunits.

Primary prevention efforts across the nation remain the frontline in indentifying lead exposure sources (Levin, et al., 2008). Clark County's CLPPP and collaborative work with UNLV and other agencies must continue to target and educate people living in the oldest housing segments. Furthermore, the data suggest the need to develop a strategic plan that addresses non-traditional sources of exposure.

Recommendations for Future Research

The focus of the study was to identify lead-based paint hazards through primary prevention activities conducted through collaborative efforts with CLPPP program. Data revealed that the age of the home is a probable indicator to determine the presence of lead-based paint hazards. However, the data was limited in identifying specific populations that could be carrying a larger burden by living in older housing units. Thus, future studies in Nevada should incorporate factors to identify social determinants of health as they relate to the built environment and health.

Non-traditional hazards such as tile with lead above EPA/HUD standards are prominent in Southern Nevada homes. The need to understand lead in tile as source of exposure needs further investigation. Occupational studies among tile workers and their families evaluating corresponding blood lead levels could establish risk associated with lead tile exposure.

In order to gain a comprehensive view of lead-based hazards further housingbased evaluations are necessary. A large scale randomized study should evaluate the housing stock throughout the state. Information gathered from such a study could reveal the need to develop targeted prevention and education strategies for specific regions throughout Nevada.

In recent years there has been national transition from lead hazard control programs into developing multifaceted healthy homes initiatives that address multiple hazards simultaneously. Although addressing and identifying healthy homes issues in the home was not a primary focus of this study preliminary data collected revealed that 32% of the homes inspected had moisture problems, 35% had visible amounts of dust and 5%

had mold related issues. All which are identified asthma triggers that contribute to asthma attacks, ER visits and miss school days. Clark County health officials and educators should incorporate translational research components which can build our knowledge on multiple housing related hazards that affect health and focus on adopting best practices that address the needs of the community.

LEAD INVESTIGATION QUESTIONNAIRE

The following questionnaire was created by Shawn Gerstenberger, Anne Rothweiler, and Elena Cabb at the University of Nevada Las Vegas, Department of Environmental and Occupational Health, 4505 Maryland Parkway, Box 453063, Las Vegas, Nevada, 89154. This questionnaire is administered to parents of children with elevated blood lead levels in Nevada and has been approved by the UNLV Office for the Protection of Research

Subjects prior to use.

For additional information regarding this protocol, please call 702-895-5420. If you use materials from this questionnaire, please let us know by sending an email to shawn.gerstenberger@unly.edu.

Thank You!

Case Number:

Address:

Date of Investigation:

Purpose: To identify potential environmental sources of lead

Investigator Tasks:

- \Box Explain the reason for the visit to the parents/guardians.
- □ Explain how a child gets lead poisoning.
- \Box Explain symptoms and health effects.
- Explain the questionnaire and give a time estimate for completion.
 Inform them that we will be collecting samples and they will be notified with the results.

Explain that a Public Health Nurse will contact them for further testing and/or treatment of the affected child as well as other household members.

□ Counsel about in-place management and prevention techniques.

Questionnaire Sections:

□ Privacy Notice	.Page 2	
□ Basic Information	Page 2	
\Box Residential and Other Locations .	Page 3	
□ Hazard Control Measures	Page 3	
□Paint and Dust	Page 4	
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Soil	Page 5
Water	Page 5
Blinds & Shutters	Page 6
Previous Lead Testing	Page 7
\Box Summary	Page 8

Insert language here for privacy disclosure

Release of Information obtained from family? [] YES [] NO

Date:

BASIC INFORMATION

Last Name:		First Name:		Middle Name:
Date of Birth:	Date of Birth: Age:Y		Gen [] F	ider: Female [] Male

Primary Language:		Race:			
[] English [] Spanish	h	[] Asian	[] Black	. []] Hispanic
[] Other:		[] Native A	merican	[] Caucasi	ian
		[] Other: _			
Address:			City:		State:
Zip Code:	County	y:		Census Tract:	
Home Phone:	Work I	Phone:		Mobile Phone:	

Additional members of the household:

Age	Gender	Race
	[] Female [] Male	

RESIDENTIAL AND OTHER LOCATIONS

Describe the current t	ype of residenc	e:	
[] Single	[] Multi	[] Trailer	[] Other:
Is the current occupan	it an:		
[] Owner	[] Renter	[] Public Hou	sing (i.e. Section 8, Housing Project)

Owner or Landlord Information (if different from the occupant)

	Name:		Relationsh	ip to O	ccupant:	
	Address:			Phone	•	
Does	the family receiv	e any housing assistance	from any ag	gency?	[] YES	[] NO
If yes	, what is the Age	ncy?				
Does	the family have a	housing case manager?	[] YES	5	[] NO	
If yes	, name and phone	e of manager:				
When	n did the family m	nove into the current resid	lence?			
Appro	oximate year of c	onstruction:				
[]19	79+	[] 1950-1959		[] 192	0-1929	
[]19	70-1978	[] 1940-1949	[] Pre-	1920		
[]19] 1960-1969 [] 1930-1939 [] Don't k			i't Knov	W	
If the home was built before 1978, did the family receive disclosure of the possible						
presence of lead-based paint, or any information regarding lead-based paint and/or lead-						
based	l paint hazards in	the home? [] YES	[] NO			
Notes	8:		_			

HAZARD CONTROL MEASURES

What type(s) of floor coverings are found in the home?
[] Vinyl/Linoleum
[] Carpeting
[] Wood
[] Ceramic or Clay Tile
[] Other:_______
Are floor coverings smooth and cleanable? [] YES [] NO

Notes:

PAINT AND DUST

Has there been any recent remodeling, repainting, renovation, window replacement, sanding or scraping of painted surfaces inside or outside this residence in the last six months?

[] YES [] NO

If yes, describe the activities and duration:

Has any lead abatement work been conducted at this residence recently?

[]YES []NO

If yes, describe:

Notes:

SOIL

Is the residence near a lead industry, such as a battery plant, smelter, radiator repair shop,
or electronic/soldering facility? [] YES [] NO
Can the family smell automobile fumes from the current residence, or any residence
where they have lived in the last three years?[] YES [] NO
Is the residence located within two blocks of a major roadway, freeway, elevated
highway, or other transportation structure? [] YES [] NO
If yes, how far?
Are nearby buildings or structures being renovated, repainted, or demolished?
[]YES []NO
If yes, describe location and activities:
Is there deteriorated paint on outside fences, garages, play structures, railings, or
mailboxes? [] YES [] NO
If yes, describe:
Are there visible paint chips near the perimeter of the building, fences, garages, or play
structures?
[]YES []NO
If yes, describe the location(s):

Notes:

WAILN				
Are there lead pipes or solde	r in the residence?		[] YES	[] NO
Has anyone repaired the plur	nbing? [] YES	[] NO	С	
If yes, when?				
By whom?				
Has new plumbing been insta	alled within the las	t five year	s?[]YES	[] NO
If yes, identify the location(s):			
Did the family do any of this	work themselves?)	[] YES	[] NO
If yes, describe the work don	ie:			
8. Have the faucets been rep	placed? []	YES	[] NO	
If yes, when?				
9. Is the glazing on the batht	ubs old or deterior	ated?	[] YES	[] NO
If yes, describe:				
Notes:				
BLINDS AND SHUTTERS	5			
1. Are there any miniblinds i	n the home?	[]Y]	ES []]	NO
If yes, where are the miniblin	nds located?			
[] Living Room	[] Kitchen	[] [] [] [] [] [] [] [] [] [] [] [] [] [
		[]Di	ning Room	
[] Child's Bedroom	[] Bathroom	[] Di [] Fa	ming Room mily Room	
[] Child's Bedroom [] Parents' Bedroom [] Oth	[] Bathroom	[] Di [] Fa	mily Room	_
 Child's Bedroom Parents' Bedroom Oth Are there vertical blinds i 	[] Bathroom her: n the home? []	[]Di []Fa YES	mily Room	
 [] Child's Bedroom [] Parents' Bedroom [] Oth 2. Are there vertical blinds i a. If yes, where are the vertical 	[] Bathroom ner: n the home? [] cal blinds located?	[]Di []Fa YES	mily Room	
 [] Child's Bedroom [] Parents' Bedroom [] Oth 2. Are there vertical blinds i a. If yes, where are the vertical blinds i [] Living Room 	[] Bathroom her: n the home? [] cal blinds located? [] Kitchen	[] Di [] Fa YES [] Di	ing Room mily Room [] NO ning Room	
 [] Child's Bedroom [] Parents' Bedroom [] Oth 2. Are there vertical blinds i a. If yes, where are the vertical blinds i [] Living Room [] Child's Bedroom 	[] Bathroom ner: n the home? [] cal blinds located? [] Kitchen [] Bathroom	[] Di [] Fa YES [] Di [] Fa	ing Room mily Room [] NO ning Room mily Room	
 [] Child's Bedroom [] Parents' Bedroom [] Oth 2. Are there vertical blinds i a. If yes, where are the vertical blinds i [] Living Room [] Child's Bedroom [] Parents' Bedroom [] Oth 	[] Ritchen [] Bathroom ner: n the home? [] cal blinds located? [] Kitchen [] Bathroom ner:	[] Di [] Fa YES [] Di [] Fa	Ining Room mily Room [] NO ning Room mily Room	
 [] Child's Bedroom [] Parents' Bedroom [] Oth 2. Are there vertical blinds i a. If yes, where are the vertical blinds i [] Living Room [] Child's Bedroom [] Parents' Bedroom [] Oth 3. Are there shutters in the base 	[] Bathroom ner: n the home? [] cal blinds located? [] Kitchen [] Bathroom ner: oome? []	[] Di [] Fa YES [] Di [] Fa YES	Ining Room mily Room [] NO ning Room mily Room [] NO	
 [] Child's Bedroom [] Parents' Bedroom [] Oth 2. Are there vertical blinds i a. If yes, where are the vertical blinds i [] Living Room [] Child's Bedroom [] Parents' Bedroom [] Oth 3. Are there shutters in the h If yes, have the shutters been 	[] Ritchen [] Bathroom ner: n the home? [] cal blinds located? [] Kitchen [] Bathroom ner: nome? [] a painted or covere	[] Di [] Fa YES [] Di [] Fa YES d with an i	ning Room mily Room [] NO ning Room mily Room [] NO ndustrial cove	 rring?
 [] Child's Bedroom [] Parents' Bedroom [] Oth 2. Are there vertical blinds i a. If yes, where are the vertical blinds i a. If yes, where are the vertical blinds i [] Living Room [] Child's Bedroom [] Parents' Bedroom [] Oth 3. Are there shutters in the h If yes, have the shutters been [] YES [] NO 	[] Bathroom ner: n the home? [] cal blinds located? [] Kitchen [] Bathroom ner: nome? [] a painted or covere	[] Di [] Fa YES [] Di [] Fa YES d with an i	ning Room mily Room [] NO ning Room mily Room [] NO ndustrial cove	 :ring?
 [] Child's Bedroom [] Parents' Bedroom [] Oth 2. Are there vertical blinds i a. If yes, where are the vertical blinds i a. If yes, where are the vertical blinds i [] Living Room [] Child's Bedroom [] Child's Bedroom [] Parents' Bedroom [] Oth 3. Are there shutters in the h If yes, have the shutters been [] YES [] NO Describe the condition of the 	[] Ritchen [] Bathroom ner: n the home? [] cal blinds located? [] Kitchen [] Bathroom ner: nome? [] a painted or covere e shutters (any pain	[] Di [] Fa YES [] Di [] Fa YES d with an i t deteriora	ning Room mily Room [] NO ning Room mily Room [] NO ndustrial cove	— — >ring?

4. Notes:

PREVIOUS LEAD TESTING

1. Have any of the following been tested for lead?

HOME

HOME	
Tested for Lead? Results?	Lead Concentration?
[]YES []NO []+ []-	
When was the test performed?	Where can the test results be obtained?

LEAD-BASED PAINT OR LEAD-CONTAMINATED DUST IN THE HOME					
Tested for Lead?	Results?		Lead Concentration?		
[]YES []NO	[]+ []-				
When was the test performed?Where can the test results be obtained?					

 SOIL

 Tested for Lead?
 Results?
 Lead Concentration?

 [] YES
 [] NO
 [] +
 []

 When was the test performed?
 Where can the test results be obtained?

WATER		
Tested for Lead?	Results?	Lead Concentration?
[]YES []NO	[]+ []-	
When was the test performed?		Where can the test results be obtained?

BATHTUB			
Tested for Lead?	Results?	Lead Concentration?	
[]YES []NO	[]+ []-		
When was the test performed?		Where can the test results be obtained?	
MINIBLINDS			
Tested for Lead?	Results?	Lead Concentration?	
[]YES []NO	[]+ []-		
When was the test performed?		Where can the test results be obtained?	
VERTICAL BLINDS			
Tested for Lead?	Results?	Lead Concentration?	
[]YES []NO	[]+ []-		
When was the test performed?		Where can the test results be obtained?	
SHUTTERS			
Tested for Lead?	Results?	Lead Concentration?	
[]YES []NO	[]+ []-		
When was the test perfo	rmed?	Where can the test results be obtained?	

OTHER:				
Tested for Lead?	Results?		Lead Concentration?	
[]YES []NO	[]+ []-			
When was the test performed? Where can the test results be obtained?				

Notes:

BUILDING CONDITION FORM

- Is the unit in generally good repair or are there significant structural or moisture problems?
- Any obvious leaks?
- Do you see cracks in the walls, sagging walls, holes in the roof, water stains?
- Are there visible amounts of deteriorated paint or visible dust accumulation?
- Can you determine if the windows and doors old or relatively new?
- Are window tracks generally painted?
- Do window sills and/or troughs contain chips and dust?
- Is there any possible exterior source of lead? (For example, a garage, an old house next door with lots of peeling paint, a battery recycling shop)

PAINT LOG

PAINT						Case #:		
Address:				Date:		Page of		
Sample #	Substrate	Component	Color	Condition	Location	XRF Reading (mg/cm ²)	RESULTS	Notes
1							+ -	
2							+ -	
3								

DUST LOG

DUST						
Date:		Case M	Case Number:			
Address	:					
SAMPLE Room and # Location		Surface Type (Floor, Interior Window Sill, Window Trough, etc.)	Area (ft ²)	Actual Result (ug/ft ²)	Notes	
1						
2						

Clearance Standards:

- Interior Floors (carpeted and uncarpeted) = 40 ug/ft^2 Interior Window Sills = 250 ug/ft^2 Window Troughs = 400 ug/ft^2 •
- ٠
- •
- Exterior Concrete and Other Rough Surfaces = 800 ug/ft^2 •

SOIL LOG

SOIL						
Date:		Case Number:				
Address:						
SAMPLE #	LOCATION	Lab Result (ppm)	NOTES			
1						
2						

• Bare Play Area Soil = 400 ppm

• Bare Soil, Rest of the Yard = 1200 ppm

WATER LOG

WATER						
Date:			Case Number:			
Address:						
SAMPLE	#	LOCATION	Lab Result	NOTES		
1						
2						
3						

Procedure:

- Take 1 liter cold water
- 6-8 hour stand time before the first draw
- After running for about 1 minute, sample the water from the rest of the house
- After running for about 3 minutes, sample the city water supply

Clearance Standard:

• 15 ppb or 15 ug/L or 0.015 mg/L

BIBLIOGRAPHY

Alexander, F. (1974). The uptake of lead by children in different environments. *Environmental Health Perspective*, *7*, 155-159.

ATSDR. (2007). Toxguide for lead. ATSDR.

Berkowitz, M. (1995). Identification of lead solder and prevention of exposure to drinking water contaminated with lead from plumbing solder. *Environmental Research*, *71*, 55-59.

Bilets, S. (2006). *XRF technologies for measuring trace elements in soil and sediment*. Las Vegas: Environmental Protection Agency.

Braun, J. M., Kahn, R. S., Froehlich, T., Aulinger, P., & Lanphear, B. P. (2006). Exposure to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives*, *114* (12), 1904-1909.

Brown, M. J. (2008). Childhood lead poisoning prevention: getting the job done by 2010. *Journal of Environmental Health* , *70* (6), 56-57.

Brown, M., Shenassa, E., Matte, T., & Catlin, S. (1997). Children in Illinois with elevated blood lead levels attributed to home renovation and remodeling activities. *MMWR*, *115*, 1120-1123.

Bryant, S. (2004). Lead-contaminated drinking waters in the public schools Philadelphia. *Journal of Toxicology*, *42*, 287-294.

Burns, R. A. (2002). Fundamentals of Chemistry (4th ed.). New Jeresy: Prentice Hall.

Canfield, R. L., Henderson, C. R., & Cory-Slechta, D. A. (2003). Intellectual impairment in children with blood lead concentrations below 10 ug per deciliter. *The New England Journal of Medicine*, *348* (16), 1517-1526.

CDC. (2007). Case studies in environmental medicine lead toxicity. *Agency for Toxic Substance and Disease Registry*.

CDC. (n.d.). *NIOSH Manual of Analytical Methods 2003*. Retrieved 10 10, 2009, from http://www.cdc.gov/niosh/docs/2003-154/

Committee on Environmental Health, A. A. (1993). Lead poisoning: from screening to primary prevention. *Pediatrics*, *97*, 176-182.

Committee on Environmental Health; Lead exposure in children:prevention, detection, and management. (2005). *American Academy of Pediatrics*, *116*, 1036-1046.

Dietrich, K. N., Ris, M. D., Succop, P. A., Berger, O. G., & Bornschein, R. L. (2001). Early exposure to lead and juvenile delinquency. *Neurotoxicology and Teratology*, *23*, 511-518.

EPA. (1994). Lead in Drinking Water in Schools and Non-Residential Buildings.

EPA. (2008, 10 21). *State and County Emission Summaries*. Retrieved 2 10, 2009, from Environmental Protection Agency: http://www.epa.gov/cgi-bin/broker?_service=data&_debug=0&_program=dataprog.dw_do_all_emis.sas&pol=21 9&stfips=32

Farr, N. (1996). Childhood lead poisoning: solving a health and housing problem. *Journal of Policy Development and Research*, *2* (3), 167-181.

Gasana, J., & Chamorro, A. (2002). Environmental lead contamination in Miami innercity area. *Journal of Exposure Analysis and Environmental Epidemiology*, *12*, 265-272.

Goldstein, G. (1992). Nuerologic concepts of lead poisoning in children. *Pediatric Annals*, *21* (6), 384-388.

Gorospe, E., & Gerstenberger, S. (2008). Atypical sources of childhood lead poisoning in the United States: a systematic review from 1966-2006. *Clinical Toxicology*, *46*, 728-737.

Goyer, R. (1990). Lead toxicity: current concerns. *Environmental Health Perspective*, *89*, 91-94.

Gracia, R. C., & Sondgrass, W. R. (2007). Lead toxicity and chelation therapy. *American Journal of Health*, 64, 45-53.

Heneman, K., & Zidenberg-Cherr, S. (2006). Is lead toxicity still a risk to U.S. children. *California Agriculture*, 60 (4), 180-184.

Hernberg, S. (2000). Lead poisoning in a historical perspective. *American Journal of Industrial Medicine*, *38*, 244-254.

Hilton, F. H. (1998). Factoring the environmental kuznets curve: evidence from automotive lead emissions. *Journal of Environmental Economics and Management*, *35*, 126-141.

(2004). *Housing-Needs Assessment 2005-2009 Consolidated Plan.* Clark County, Nevada Department of Finance, Community Resources Management Housing.

HUD. (2004, 6 18). U.S. Department of Housing and Urban Development. Retrieved 4 11, 2009, from History of Lead Based Paint Legislation:

http://www.hud.gov/offices/cpd/affordablehousing/training/web/leadsafe/ruleoverview/le gislativehistory.pdf

Jacobs, D. E. (2004). *Guidelines for the Evaluation and Control of Lead-based hazards in housing*. Dane Publishing Company.

Jacobs, D. E., Clickner, R. P., Zhou, J. Y., Viet, S. M., Marker, D. A., Rogers, J. W., et al. (2002). The prevalence of lead-based paint hazards in U.S. Housing. *Environmental Health Perspectives*, *110* (10), 599-606.

Jacobs, D. (1996). The health effects of lead on the human body. *Lead Perspectives*, 10-32.

James, H., Hilburn, M., & Blair, J. (1985). Effects of meals and meal times on uptake of lead from the gastrointestinal tract in humans. *Human Toxicology*, *4*, 401-107.

Jusko, T. A., Henderson, C. R., Lanphear, B. P., Cory-Slechta, D., Parsons, P. J., & Canfield, R. L. (2008). Blood lead concentrations <10 ug/dL and child intelligence at 6 years of age. *Environmental Health Perspectives*, *16* (2), 243-248.

Kemper, A. R., Cohn, L. M., Fant, K. E., Dombkowski, K., & Hudson, S. R. (2005). Follow-up testing among children with elevated screening blood level. *Journal of the American Medical Association*, 293 (18), 2232-2237.

Khan, A. N., Munir, U., Turnbull, I., & MacDonald, S. (2009). *Lead Poisoning*. eMedicine.

Laidlaw, M. A., & Filippelli, G. M. (2008). Resuspension of urban soils as a persistent source of lead poisoning in children: a review and new directions. *Applied GeoChemistry*, *23*, 2021-2039.

Lanphear, B. (2008). Childhood lead poisoning prevention. *Journal of American Medical Association*, 293 (18), 2274-2276.

Lanphear, B. P. (2005). Childhood lead poisoning prevention: too little, too late. *Journal of the American Medical Association*, 293 (18), 2274-2276.

Lanphear, B. P. (1998). The paradox of lead poisoning prevention. *Science*, 281, 1617-1618.

Lanphear, B. P., Auinger, P., & Schaeffer, S. (1998). Community characteristics associated with elevated blood lead levels in children. *Pediatrics*, *101*, 264-271.

Lanphear, B. P., Dietrich, K., Auinger, P., & Cox, C. (2000). Cognitive deficits associated with blood lead concentrations <10 ug/dL in US children and adolescents. *Public Health Reports*, *115*, 521-529.

Lanphear, B. P., Weitzman, M., & Elberly, S. (1996). Racial differences in urban children's environmental exposures to lead. *American Journal of Public Health*, *86* (10), 1460-1463.

Lanphear, B. (2007). The conquest of lead poisoning: a pyrrhic victory. *Environmental Health Perspectives*, *115*, 484-485.

Lanphear, B., Hornung, R., & Ho, M. (2005). Screening housing to prevent lead toxicity in children. *Public Health Reports*, *120*, 305-310.

Lansdown, R., & Yule, W. (1986). *Lead toxicity: history and environmental impact*. Baltimore: The Johns Hopkins University Press.

Leighton, J., Klitzman, S., Sedlar, S., Mattle, T., & Cohen, N. (2003). The effect of leadbased paint hazard remediation on blood lead levels of lead poisoned children in New York City. *Environmental Research*, *92*, 182-190.

Levin, R., Brown, M., Kashtock, M., Jacobs, D., Whelan, E., Rodman, J., et al. (2008). Lead exposures in U.S. children, 2008: implications for prevention. *Environmental Health Perspectives*, *116* (10), 1285-1293.

Maas, R. P., Patch, S. C., Morgan, D. M., & Pandolfo, T. J. (2005). Reducing lead exposure from drinking water: recent history and current status. *Public Health Report*, *120*, 316-321.

Markowitz, M. (2000). Lead Poisoning. Pediatrics in Review, 21 (10), 327-335.

McCarthy, J. E. (2008). *Revising the national ambient air quality standard for lead*. Congressional Research Service.

McMurry, J., & Fay, R. C. (2001). *Chemistry* (3rd Edition ed.). New Jersey: Prentice Hall.

Mielke, H. W. (1994). Lead in New Orleans soils: new images of an urban environment. *Environmental Geochemistry and Health* (3-4), 1573-2983.

Mielke, H. W., Gonzales, C., Powell, E., & Mielke, P. W. (2008). Urban soil-lead footprint: retrospective comparison of public and private properties in New Orleans. *Environmental Geochemistry and Health*, *30* (3), 1573-2983.
Mielke, H., & Reagan, P. (1998). Soil is an important pathway of human lead exposure. *Environmental Health Perspectives*, *106* (Suppl 1), 217-219.

Miranda, M. L., Kim, D., Galeano, M. A., Paul, C. J., Hull, A. P., & Morgan, S. P. (2007). The relationship between early childhood blood lead levels and performance on end-of grade tests. *Environmental Health Perspectives*, *115* (8), 1242-1247.

Mushak, P., & Crocetti, A. F. (1990). Methods for reducing lead exposure in young children and other risk groups: an integrated summary of a report to the U.S. congress on childhood lead poisoning. *Environmental Health Perspectives*, *89*, 125-135.

Myers, G. J., Davidson, P. W., Weitzman, M., & Lanpher, B. P. (1997). Contribution of heavy metals to developmental disabilites in children. *Metal Retardation and Developmental Disabilites*, *3*, 239-245.

Nadakavukaren, A. (2006). *Our Global Environment: A Health Perspective* (6th Edition ed.). Illinois: Waveland Press.

Needleman, H. (1998). Childhood lead poisoning: the promise and abandonment of primary prevention. *American Journal of Public Health*, 88 (12), 1871-1877.

Needleman, H. L., McFarland, C., Ness, R. B., Fienberg, S. E., & Tobin, M. J. (2002). Bone lead levels in adjudicated delinquents a case control study. *Neurotoxicology and Teratology*, *24*, 711-717.

NHANES. (2009, 3 13). *National Center of Environmental Health*. Retrieved October 22, 2008, from Center of Disease Control: http://www.cdc.gov/nceh/lead/research/kidsBLL.htm#National%20surveys

Niton. (2009). *How XRF Works*. Retrieved 2 21, 2009, from Thermo Fisher Scientific's Niton XRF Analyzers: http://www.niton.com/Portable-XRF-Technology/how-xrf-works.aspx

Niton. (2009). *Lead Paint Testing*. Retrieved 2 13, 2009, from Thermo Scientific: http://www.niton.com/Lead-Paint-Testing/literature.aspx

Opler, M. G., Brown, A. S., Graziano, J., Desai, M., Zheng, W., Schaefer, C., et al. (2004). Prenatal lead exposure, aminolevulinic acid, and schizophrenia. *Environmental Health Persepctives*, *112* (5), 548-552.

Petrosyan, Orlova, Dunlap, Babayan, Farfel, & Braun, V. (2004). Lead in residential soil and dust in a mining and smelting district in northern Armenia: a pilot study. *Environmental Research* (94), 297-308.

Phelps, A., Ryan, D., Quinn, E., Malon, J., & Scott, R. (2004). *Lead-safe housing policy guidance*. Washington: Alliance for Healthy Homes.

Pirkle, J. L., Kaufmann, R. B., Brody, D. J., Hickman, T., Gunter, E. W., & Paschal, D. C. (1998). Exposure of the U.S. population to lead, 1991-1994. *Environmental Health Perspectives*, *106*.

Rogan, W. J., & Ware, J. H. (2003). Exposure to lead in children - how low is low enough? *The New England Journal of Medicine*, 348 (16), 1515-1516.

Romieu, I., Lacasana, M., McConnell, R., & Organization, L. R.-A. (1997). Lead exposure in Latin America and the Caribbean. *Environmental Health Perspectives*, *105* (4), 398-405.

Rothweiler, A. M., Cabb, E. E., & Gerstenberger, S. L. (2007). The status of childhood lead poisoning and prevention in Nevada, USA. *The Scientific World Journal*, *7*, 479-492.

Sargent, J., Brown, M., Freeman, J., Bailey, A., Goodman, D., & Freeman, D. (1995). Childhood lead poisoning in Massachusetts communities: its association with sociodemographic and housing characteristics. *American Journal of Public Health*, *85*, 528-534.

Satcher, D. (1997). *Screening young children for lead poisoning: guidance for state and local public health officials*. Atlanta: Centers for Disease Control and Prevention.

Schwartz, J. (1994). Low-level lead exposure and childrens IQ: a meta-analysis and search for a threshold. *Environmental Research* , *65*, 42-55.

Selevan, S. G., Rice, D. C., Hogan, K. A., Euling, S. Y., Pfahles-Hutchens, A., & Bethel, J. (2003). Blood lead concentration and delayed puberty in girls. *The New England Journal of Medicine*, *348* (16), 1517-1536.

SNHD. (2007). *Childhood lead poisoning prevention program: lead poisoning elimination plan.* Las Vegas: Southern Nevada Health District.

Staes, C., Matte, T., Gilbert, C. C., Flanders, D., & Binder, S. (1994). Retrospective study of the impact of lead-based paint hazard remediation on children's blood lead levels in St. Louis, Missouri. *American Journal of Epidemiology*, *136*, 106-1026.

Taha, T., Kanarek, M. S., Schultz, B. D., & Murphy, A. (1999). Low-cost household paint abatement to reduce children's blood lead levels. *Environmental Research*, *81*, 334-338.

Tong, S., Von Schimding, Y. E., & Prapamontol, T. (2000). Environmental lead exposure: a public health problem of global dimensions. *World Health Organization*, *78*, 1068-1077.

Vallejos, Q., Strack, R. W., & Aronson, R. E. (2006). Identifying culturally appropriate strategies for educating a mexican immigrant community about lead poisoning prevention. *Family & Community Health*, *29* (2), 143-152.

Wright, J. P., Dietrich, K. N., Ris, M. D., Hornung, R. W., Wessel, S. D., Lanphear, B. P., et al. (2008). Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. *PLoS Medicine*, *5* (5), 732-740.

Ziegler, E. E., Edwards, B. B., Jensen, R. L., Mahaffey, K. R., & Fomon, S. J. (1978). Absorption and retention of lead by infants. *Pediatric Research*, 1974, 29-34.

Zierold, K. M., Havtena, J., & Anderson, H. (2007). Exposure to lead and length of time needed to make homes lead-safe for young children. *American Journal of Public Health*, *97* (2), 267-268.

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