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What Influences Do Age, Gender and Race/ Ethnicity Have on the Overall Health Consequences of Excess Lead In Children?

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WHAT INFLUENCES DO AGE, GENDER AND RACE/ETHNICITY HAVE
ON THE OVERALL HEALTH CONSEQUENCES OF EXCESS LEAD IN CHILDREN?

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A Research Paper
Submitted in Partial Fulfillment of the Requirements for the
Master of Science

Department of Animal Science, Food and Nutrition
in the Graduate School
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RESEARCH PAPER APPROVAL

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A Research Paper Submitted in Partial
Fulfillment of the Requirements
For the Degree of
Master of Science
In the field of Food and Nutrition

Approved by:

Dr. William Banz, Chair

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TITLE: WHAT INFLUENCES DO AGE, GENDER AND RACE/ETHNICITY HAVE ON THE OVERALL HEALTH CONSEQUENCES OF EXCESS LEAD IN CHILDREN?

MAJOR PROFESSOR: Dr. William Banz

After Flint, Michigan governmental authorities introduced a more corrosive water source into an already aging water system without adequate corrosion control, the percentage of children with elevated blood lead levels increased, particularly in socioeconomically disadvantaged neighborhoods. The main objective of this research paper is to clarify influences of age, gender and race, ethnicity have on the overall health consequences of excess lead in children. There are studies that focus on the short and long term effects of neurological, hypertension, renal, and developmental on children. However, since specific demographic evidence is still limited, future research is still needed to implement a system that will adequately help the children in Flint.

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CHAPTER 1

INTRODUCTION

Everyone needs water to survive, so what about the people of Flint, Michigan? A struggling, majority-black city has found itself in turmoil of late because of the city's cost-saving decision recently to switch its water supply from Detroit's cleaner system, to the Flint River. After months of disregarding the warning signs that surfaced about the dilemma with the water in Flint, city, state and federal officials in Michigan are now scrambling to respond to the public health emergency because it found that high levels of lead seeped from pipes, into the water supply. It wasn't much of a surprise that soon afterward, a group of physicians documented a spike in the blood lead levels of local children. The objective of this research paper is to discuss the impact of the lead on children's health.

Research questions:

- What is lead poisoning?
- What are the long and short-term effects of lead poisoning in children?
- What influences do age, gender and race/ethnicity have on the overall health consequences of excess lead in children?

Background

The city of Flint, Michigan is located about 60 miles northwest of Detroit, the region's major urban center, in Genesee County. The Flint River was very popular during the 1800s because it was used as a means of commerce. The brisk, lucrative trading of essential items such as lumber and fur eventually lead to the production of horseless carriages, which ultimately gave birth to what is now known as the American automotive industry. This progressive industry brought newfound wealth to the area by laying the groundwork for companies such as the Buick Motor Company in 1903, and General Motors (GM) in 1908 (Scorsone, Bateson, 2011).

Innovative advancements to working conditions and wages made Flint the optimal place to live and work. The burgeoning wealth created well-respected communities and educational systems. Between 1932 and 1962, the population of Flint increased substantially, chiefly owing to African American immigration. This swelling immigrant population simultaneously produced a considerable expansion of African American neighborhoods. The general population increased as a result of the expansion of heavy industry—mainly automobile manufacturing (Lewis, 1965). In the 1970s, there was a 17.4% decline in the population, however. The falling employment rates at the top automotive companies began to have a substantial impact on Flint. The fiscal stress felt from the plummeting industry decentralized the metropolitan areas.

Today, an analysis of the demographics of Flint can provide a better a perspective of how to answer its unique long-term and current challenges with lead poisoning. The U.S. Census Bureau reports that there have been double-digit declines in the city of Flint within the years 2000 to 2010, totaling 18% of the population of 102,434 (Scorsone, Bateson, 2011). The most recent update to these figures shows that from 2010 to 2014, the population was reduced to 99,002 which is a percentage change of -3.3%.

Table 1: The U.S. Census Bureau et al. (2016):
Flint's Population Based Upon Race, 2010

African American	56.6%
Caucasian	37.4%
Hispanic or Latino	3.9%
American Indian and Alaska Native	0.5%
Asian	0.5%
Native Hawaiian and Other Pacific Islander	X

Table 1 breaks down Flint's population by race, for 2010. The distribution of population by race is shown in the chart. The 2010 Census report counts and related change from the 2000 Census have the Black/African American population at 56.6% (an increase of 3.3%), while Caucasians is 37.4% (a decrease of 4%) . (Table 1; US Census Bureau. 2016).

In 1986, lead plumbing material was banned from being used, but in older cities such as Flint, lead-based service lines still existed without the proper updates. Thus, in April of 2014 when Flint governing officials switched the city's water supply from Detroit's Lake Huron to the Flint River, lead began to percolate from the pipes directly into the citizens' drinking water. The main reasons why lead began to develop from the switch are that Detroit's water harbored very low corrosivity for lead, as indicated by low chloride, low chloride-to-sulfate mass ratio and the presence of an orthophosphate corrosion inhibitor. By contrast, Flint River water had high chloride, high chloride-to-sulfate mass ratio and no corrosion inhibitors (Hanna, Lachance, Sadler and Schnepf, 2016). It is estimated that 10% to 80% of Flint's service lines contain lead (Hanna, Lachance, Sadler and Schnepf, 2016).

Lead is known to be a potent neurotoxin that significantly impacts children by specifically altering their developmental and biological processes. The effects of lead poisoning are usually seen in the altering of a child's performance, intellect or even just impacting basic life achievements. The societal cost to eradicate the lead poisoning epidemic within low income communities such Flint would be in the billions of dollars, and for this reason many minority children will, and are suffering.

Table 2: The U.S. Census Bureau et al. (2016):
Flint's Population Based Upon Age and Sex, 2010

Persons under 18 years	27.3%
Persons under 5 years	8.0%
Persons 65 years and over	10.7%
Female persons	52.0%

Table 2 above breaks down Flint's population by age and sex for the year 2010. This chart shows that 27.3% of people are under 18, and that there are 52.0% women in Flint. The impact the population has the potential to be severe.

The lead found in drinking water will soon disproportionately affect the children and expectant mothers of Flint. Research shows that orally, water soluble lead is absorbed by children at the rate of 40% to 50%, and adults at the rate of 3% to 10%. For every child 1 to 5 years of age, and for every 1-ppb

increase of lead in the water, there will be a 35% increase of blood lead. Each time tap water at 10 ppb is used with drinking formula for infants, there will be a 25% chance that that child will experience a BLL above the Centers for Disease Control and Prevention (CDC) elevated blood lead level (EBLL) of 5 micrograms per deciliter (mg/dL) (Hanna, Lachance, Sadler and Schnepf, 2016).

With these statistics, we see that formula alone will account for more than 85% of the lead exposure among infants. It is clear that lead exposure is inevitable, metamorphic and costly, proving that primary prevention is necessary.

Heavy metal has always been toxic to human beings. Lead has been a heavily studied metal because of its toxicity to the developing brain and nervous system. When exposed to lead during intrauterine periods, it can have detrimental effects beginning at birth. During the first few years of life, lead poisoning can develop. It is common for prenatal exposure with mercury, whereas lead poisoning is more common during the first few years of life. By providing information, including a synopsis of lead's toxicity and sources of exposure, health professionals can better inform the public of the adverse effects of lead, and to guide primary prevention tactic.

Chemical and Physical Information

Lead is a natural metal from the Earth's crust that is heavy in weight, and bluish gray in color. Although lead comes directly from the earth, it's rarely found naturally as a metal. Lead is more commonly found integrated with other elements to form lead compounds. Lead is located on the periodic table in Group 14 (IVA). When lead is found in its natural state, it has four different blends of stable isotopes: ^{204}Pb (1.35–1.5%), ^{206}Pb (23.5–27%), ^{207}Pb (20.5–23%) and ^{208}Pb (51–53%). These isotopes are the daughter products of the natural radioactive elements ^{206}Pb from uranium, ^{207}Pb from actinium and ^{208}Pb from thorium.

Lead is not a readily bountiful element, but its ore deposits can be found all around the world, leveraged for profit. Lead is the perfect element for items such as pipes, weights and batteries because of its properties which render it long-lasting such as corrosion resistance, density and low melting points (Abadin, Ashizawa, Stevens, 2007).

There are three oxidation states of lead known as: Pb (0), the metal; Pb (II); and Pb (IV). Pb (II) is found predominantly in the environment, unlike Pb (IV) which is inorganic and made in intense, oxidized conditions which are not typically found in normal environmental conditions. Pb (0) is similar to Pd (IV), whereas it occurs in nature, but its manifestation is rare. Lead is widely used all around the world because of its low melting point and corrosion resistance—meaning it is not easily altered by ore or water. When lead is exposed to these elements, films are formed from sulfate and lead carbonates and oxides to form a protective barrier which prevents corrosion. Lead is an amphoteric element that can act as both an acid by forming plumbous and plumbic salts, and a base such as plumbites and plumbates in alkali (Abadin, Ashizawa, Stevens, 2007).

Lead poisoning

Lead is a highly toxic substance which can cause widespread health effects. Many adults can suffer from lead poisoning each year, but the more frequent malady is childhood lead poisoning. Since the onset of knowledge regarding the hazards of lead, there have been tens of millions of children who have suffered its ill health effects (Guidotti, Ragain, 2007).

Toxicity is determined by the time exposed, and the amount of lead within the tissue (Pearson, Schonfeld, 2003). There are two types of lead poisoning: acute, and chronic. Acute means that the exposure to lead was severe and intense for a short period of time. Chronic means that there was a low level of exposure for a long period of time, and this is the most common form of poisoning (Trevor, Katzung, Masters, 2007). Treatment for any form of lead exposure is based on the amount of lead in the

blood, which is called Blood Lead Levels or BLL. BLL is measured in micrograms of lead per deciliter of blood ($\mu\text{g}/\text{dL}$). One can also measure lead using urine lead levels, but this is less common. The highest concentrations in chronic lead exposure is found first in the bones, and then the kidneys. An excretion test is used to measure the urine samples collected from the patient, which provides a more precise reading of total lead.

The reference levels given by the Centers for Disease Control and Prevention (CDC) state that $>10 \mu\text{g}/\text{dL}$ is the level of concern for adverse health outcomes in children (Bellinger, (n.d.)). There is new research being done that shows that children's health is affected at much lower blood lead levels. The CDC reports that there are no safe blood levels in children because lead exposure often has no obvious symptoms or signs. Elevated blood lead levels are the root of major preventable health problems in children. Prolonged lead exposure, coupled with a high dosage heightens the likelihood of damage. Over time, complications such as behavioral problems, disabilities and mental retardation can develop. Extremely elevated levels are known to cause seizures, coma and even death (Flora, Gupta, Tiwari, 2012).

CHAPTER 2

LITERATURE REVIEW

The water crisis in Flint, Michigan has primarily focused on the long-term effects of lead exposure on children. Since it can have lifelong consequences. There is no purpose for lead in the human body, but its existence in both high and low levels can have toxic effects. Lead exposure can affect every organ system and disrupt biochemical processes within the body. There is no detailed threshold for lead poisoning developmental effects on children. The focus on lead poisoning in children tend to be on the neurological impact, but it's important to understand that childhood lead poisoning can lead to health effects later in life including hypertension ,renal, and developmental problems with offspring.

Conception and Developmental

Starting at childbirth, lead is known to have adverse effects. Research shows that development of the fetus is at risk by blood lead crossing the placenta. This type of disruption is known to cause problems associated with the neurological development of the fetus. At this early stage of pregnancy, there isn't a blood brain barrier which means that any existing blood lead level from the mother is an indication of risk. Mothers who have had any exposure to lead are known to store it in their bones, and could possibly release it during period of calcium stress such as pregnancy, and lactation (Shannon, 2003).

Blood Lead in Soft Tissue

Blood plays a key role in the damaging effects of lead since it is used to carry a small proportion of the body's total lead by acting as a vessel for distribution for abortion throughout the body. This from of circulation allows lead to be available to other tissues, or used for excretion via the circulation process. The half-life of blood is predicted to be anywhere from 28 days (Abadin, Ashizawa, Stevens, 2007), to 36 days (Rabinowitz, Wetherill, Kopple, 1976). Blood is broken down into 99% erythrocytes, or red blood cells, and 1% blood plasma (Desilva, 1981). Lead is transferred through soft and mineralizing

tissues by blood plasma. The percentage of plasma is higher when lead concentration levels are high, therefore blood lead levels are widely used to measure lead exposure. An assay known as the erythrocyte protoporphyrin (EP) is less effective at measuring blood lead levels because of its sensitivity.

Blood circulates lead throughout the organs and tissues. According to the lead Toxicology Report, the greatest soft tissue damage is done to the liver, lungs and kidneys after acute exposure such as oral, inhalation, dermal and intravenous routes (Abadin, Ashizawa, Stevens, 2007). Lead is also distributed to the brain. The mineralizing of tissue is evidence that children retain more lead than adults. The lead is more likely to accumulate in the hippocampus portion of the brain. The half-life of lead in soft tissue is 40 days (Abadin, Ashizawa, Stevens, 2007).

Neurological

The target of lead exposure is the nervous system. The neurological effects of lead are most damaging among fetuses and young children because their systems are still in the process of development, and the blood brain barrier is incomplete. There are no safe lead levels for children. Adverse neurologic effects can start well before the CDC BLL standard of $<10 \mu\text{g/dL}$. The neurological effects of lead can be present in patients that are asymptomatic, especially with children.

Encephalopathy can become present with very high levels of lead in children, and can lead to hyperirritability, ataxia, convulsions, stupor and coma or death. The BLL vary in the case of encephalopathy, but research shows that BLL that range from $70\text{--}80 \mu\text{g/dL}$ or greater results in serious risk (Abadin, Ashizawa, Stevens, 2007). Even if symptoms of encephalopathy are not present, there is still an increased association with lasting neurologic and behavioral damage (Abadin, Ashizawa, Stevens, 2007). Lower lead exposure levels can also cause adverse neurological effects in children.

Research shows that there is a decline in intelligence quotient (IQ) performance, and other neuropsychologic defects with lead exposure. For every $10 \mu\text{g/dL}$ increase in BLL in children, there is

an IQ drop of four to seven points (Yule, Lansdown, Millar, Urbanowicz, 1981). Evidence shows that children with BLLs of 16 $\mu\text{g}/\text{dL}$ or higher have a greater likelihood of ADHD and hearing impairment. At these levels, it's also likely that children can experience disrupted balance and impaired peripheral nerve function (Abadin, Ashizawa, Stevens, 2007). These effects may rear at or below 10 $\mu\text{g}/\text{dL}$, and may not be detectable during a clinical examination. This mean that these neurological effects of lead in children can continue into adulthood. A study that focused on long-term effects of exposure to low doses of lead in children concluded that there was a correlation between lead exposure with lower classroom performance; greater absenteeism, more reading disabilities and deficits in vocabulary, fine motor skills, reaction time and hand-eye coordination in young adults more than 10 years after childhood exposure (Needleman, Schell, Bellinger, Leviton, Allred, 1990).

Endocrine

Studies have shown that high lead levels in children have a strong adverse effect on Vitamin D levels. Lead prevents Vitamin D from changing into its hormonal form, known as 1, 25-dihydroxyvitamin D. This hormone is widely known in its maintenance of extracellular and intracellular calcium homeostasis. If 1, 25-dihydroxyvitamin D is diminished, it can hinder cell growth, maturation and tooth and bone development (Oussoren, Brands, Ruijter, 2011). These adverse effects are seen in children with chronic BLLs of $> 62 \mu\text{g}/\text{dL}$, thus causing nutritional deficiencies mainly with calcium, phosphorus and Vitamin D (Koo, Succop, Berger, et al). In the Rosen study, it was stated that 1, 25-dihydroxyvitamin D levels in children were reduced when BLLs were 33–55 $\mu\text{g}/\text{dL}$, levels that are comparable to children with severe renal insufficiency. Maintaining a diet that is sufficient in calcium and Vitamin D can help bone density and prevent osteoporosis (Chesney, R. W., Rosen, Hamstra, DeLuca, 1980). Lead has minor effects on thyroid function when examined in children. It was reported

that exposure and thyroxin levels have a weak negative correlation, and thus have no effect on children (Abadin, Ashizawa, Stevens, 2007).

Renal

Lead exposure and renal effects have a strong association according to major studies. Proximal tubular function experiences induced impairment in the glycosuria, hyperphosphaturmia and aminoaciduria because of high levels of lead. Though the effects show to be reversible, any type of constant exposure of toxins can lead to irreversible lead nephropathy (Abadin, Ashizawa, Stevens, 2007).

The effects of low levels as it relates to the kidneys are unknown. Most studies focus on the effects of high to high-to-moderate chronic exposures (BLL > 60 µg/dL). Increased serum creatinine and creatinine clearance levels are used for lead-based renal damage indicators at BLLs less than 60 µg/dL (Staessen, Lauwerys, Buchet, Bulpitt, Rondia, Vanrenterghem, Amery, 1992). Underlying acute lead exposure from previous years can have an effect on renal function, but with treatment within two months children can prevent the progression of chronic interstitial nephritis.

Renal disease is known to be asymptomatic, but early intervention can prevent or slow the progression of renal disease. Past lead exposure may be the culprit in kidney disease as it relates to hypertension (Abadin, Ashizawa, Stevens, 2007). Renal failure can add to the complications of hypertension, thus both conditions need to be controlled. Renal damage in general can cause diabetes mellitus, and this should be addressed in patients that have had lead exposure. Saturnine gout can develop as a result of lead, and the decreased excretion of uric acid can cause hyperuricemia. Fifty percent of patients with lead nephropathy can also get gout (Goyer, 1996). Premenopausal woman also run the risk of getting gout. Saturnine gout can make renal disease more severe (Miranda, Puig, Mateos, Ramos, Herrero, González, Vázquez, 1991).

Hematologic

Lead affects the creation of hemoglobin by altering the heme biosynthesis pathway enzymatic steps which use δ -aminolevulinic acid dehydratase and ferrochelatase activity (Bánfalvi, 2011). Ferrochelatase catalyzes iron so that it can form protoporphyrin IX. Protoporphyrin IX is known to be more sensitive to lead. When enzyme activity in the red blood cells is decreased, there is an increase of a substrate known as erythrocyte protoporphyrin (EP). Plasma δ -aminolevulinic acid, free Eps and an increase in blood can all be traced back to lead exposure (Air quality criteria for lead, 1977). The EPA suggests that a hemoglobin decrease is $50 \mu\text{g/dL}$ for adults, and $40 \mu\text{g/dL}$ for children (Air quality criteria for lead, 1977). EP is not sensitive to lower blood lead levels and sometimes isn't useful as a screening test.

There are two types of anemia that can be induced by lead. With acute blood lead levels, people can experience hemolytic anemia. The lead interferes with heme biosynthesis, which kills red blood cells, causing an induction of anemia (Air quality criteria for lead, 1977). Anemia, as it relates to lead is microcytic, hypochromic and normocytic with correlations to reticulocytosis. Frank anemia persists when BLL are elevated for long periods of time. Neural, endocrine, renal and hepatic pathways can all be affected by the heme synthesis pathway.

Cardiovascular and Hypertension

Risk factors such as poor diet, older age, increased weight, exercise habits, alcohol intake and lead exposure can contribute to the advancement of hypertension (Wijerathne, Meier, Agampodi, Agampodi, 2015). There have been studies which have shown that the greater the lead exposure, the higher the chances of for hypertensive heart disease and cerebrovascular disease. There is a clear association between lead exposure and high blood pressure, as illustrated by Howard study it found that childhood lead poisoning heightened the risk of hypertension 50 years later (Howard, 1991). This association accrued in populations with BLLs of $>10 \mu\text{g/dL}$. It is estimated that lead accounts for 1-2%

of variance in blood pressure, and that systolic blood pressure rises 1-2mm when blood lead doubles. This is important information to note when addressing the population because the prevalence of hypertension is high, and people with a history of excessive lead exposure can suffer from its health effects.

Regulation of Lead

The confirmed dangerous effects of lead has forced law makers to push for environmental policy reform and enforcement. Over the past 50 years, there have been 80,000 new, synthetic chemicals created. Every year, 2,000 to 3,000 new chemicals are introduced to the U.S. Environmental Protection Agency (EPA) for review before manufacture. Children are particularly prone to adverse risks associated with exposure from 15,000 chemicals produced, which amount to over 10,000 pounds per year, and to the 2,800 chemicals that are over 1 million pounds per year (McPartland, Dantzker, Portier, 2015). These particular high-volume chemicals have a greater chance of contaminating our water, air, homes, crops and communities. There have been tests for potential human toxicity on only 43% of high-volume chemicals, and only 7% of these chemicals have been tested for their effects on development (McPartland, Dantzker, Portier, 2015).

Since lead has been one of the main culprits of many public health problems, various federal agencies have published or stepped in to enforce regulations that balance lead levels in various types of chemicals. Over the past 30 years, the cases of lead exposure have declined significantly. Congress has worked to develop laws that are used to regulate lead content in items such as gasoline, paint, water and other resources, all in efforts to target the problem of lead contamination.

The United States Environmental Protection Agency (EPA) is working to uphold these laws by enforcing regulations. Some of the laws issued by the EPA include the Toxic Substances Control Act (TSCA) developed in 1976, which mandates that the EPA be the lead authority when it comes to

reporting, record-keeping and testing restrictions placed upon chemical substances (McDonnery, Elroy, Schierow, 2012). Though food, drugs, cosmetics and pesticides are excluded from TSCA, it still shows its effectiveness by addressing the production, importation, use and disposal of 83,000 chemicals—including lead. Before chemicals are introduced, there is a pre-manufacture notification submitted to the EPA. The chemical is then tested and a file is created for record-keeping (McDonnery, Elroy, Schierow, 2012).

The Residential Lead-Based Paint Hazard Reduction Act of 1992 (Title X) is a national strategy that is used to eradicate lead-based paints in all housing. The EPA discovered that American homes which were built pre-1980 harbored over 3,000,000 tons of lead-based paint. This act covers all housing, including federally assisted housing, where over 3,000,000 low-income, minority children under the age of 6 reside. Making sure that contractors are licensed, abundant property is properly treated and enforcing regulation on home owners are all ways in which this act helps fight the battle against lead (Witkin, Petersen, 1998).

The Clean Air Act (CAA) of 1975 is a federally mandated law that authorizes the EPA to create Ambient Air Quality Standards (NAAQS) to protect public health and public welfare from hazardous air emissions (Behles, 2015). One of the goals of this act was to develop state implementation plans (SIPs), but many states failed to meet deadlines for their action plans that aligned with NAAQS. The main portions of the CAA which addresses hazardous air emissions is section 112. Over the years, section 122 has been revised to make more specific requirements for leakage from major groups that emit 10 tons per year or more of a hazardous air pollutants. This section has also requires the EPA to establish standards for reduction in emissions and assess the residual risks (Behles, 2015).

The Clean Water Act (CWA) of 1948 is a framework for regulating the discharge of pollutants into the water, in hopes of improving water quality. This act, previously called the Federal Water Pollution

Control Act, was reorganized and expanded in 1972 to what we know today as the “Clean Water Act” (Clayton, Gold, Gurney, Kalpin, White, 2015). The CWA allows the EPA to control and create standards for industries’ water waste. The EPA is also responsible for monitoring surface water contamination. In order for the CWA to eradicate any pollutant from any source into travelable water, a permit must be obtained. EPA's National Pollutant Discharge Elimination System (NPDES) issues the permit. Homes that are linked to municipal systems or public systems don’t require a permit, but industrial facilities do (Clayton Gold, Gurney, Kalpin, White, 2015).

The Resource Conservation and Recovery Act (RCRA) of 1984 is a mandate that gives the EPA the authority to oversee the production of hazardous waste through the process of generation, transportation, treatment, storage and disposal. This mandate also includes the management of non-hazardous waste, as well as underground storage tanks (Opp, 2011). The amendments of this act all focus upon minimizing waste, and encourage conducive release practices (Opp, 2011).

The Comprehensive Environmental Response, Compensation and Liability Act, also known as CERCLA or Superfund of 1986, is a federal funding used to clean up hazardous emergency contamination such as abandoned waste sites, or spills (Gordon, 2015). Through this act, the EPA has the capability to find and hold those accountable for hazardous accidents, and assure collaboration in cleanup efforts. When parties can’t be identified or fail to act (orphan site), the EPA accepts all responsibility for the cleanup efforts.

The EPA uses enforcement tools to obtain consent decrees for cleanup efforts. The cost of each complete cleanup is covered by the individual or company responsible (Gordon, 2015). This act covers all 50 U.S. states and territories, and is coordinated through the state waste management or environmental protection agencies. The Superfund’s main goals are to identify, monitor and respond to hazardous activities.

Cost Analysis of Lead Poisoning

Children are more vulnerable when it comes to chemicals than adults. Children's susceptibility stems from excessive exposure and high stress environments making them a target for complications during their growth and development. Any damage to a child's developing organ system can establish a disability. Creating awareness of the economic cost and prevalence of different diseases and disabilities in children can help push preventive efforts, and start the dialogue on the cost of prevention for that population.

All across the U.S., tens of millions of children have been exposed to the negative effects of lead poisoning, costing up to an alarming \$43 billion per year (Landrigan, 2002). The benefits of lead hazard prevention are inevitable because cost analysis shows that every dollar spent to reduce hazards yielded a benefit of \$17-220 (Gould, 2009). The expense that surrounds childhood lead poisoning is split into two categories: direct and indirect cost. Direct medical costs are those expenditures related to the provision of medical care to children with acute lead poisoning (Gould, 2009). These direct costs can follow children well into adulthood. For instance, exposure to lead can cause hypertension and eventually cardiovascular disease. Indirect costs are non-medical costs that depict the economic hardship resultant from the long-term effects of childhood lead poisoning in society. Indirect cost focuses on the lifelong outcomes due to a loss of intelligence, and reductions in economic productivity which result from that loss of intelligence. As a result of lack of intelligence and related behavioral changes, the indirect cost fails to take into account the increased need for special educational services, institutionalization or incarceration of people who suffered lead poisoning. Table 3 below breaks down the cost of lead poisoning as it relates to IQ, special education, health care, crime, tax revenue and return on investment.

Table 3: Lead Poisoning Prevention Costs

IQ

<p>Children that are 6 years of age and younger can experience lower IQs due to lead exposure. Research shows that .52 IQ points can be lost for every microgram per deciliter of blood. For every IQ point lost, it is estimated that every child will earn \$16,809 less over a lifetime.</p>
<p>Health Care</p>
<p>The higher the blood lead levels, the more one will pay for prevention techniques such as therapy and doctor's visits. Effective childhood lead poisoning prevention could save around \$53 million (Kemper, 1998). Prevention of childhood lead poisoning could save the United States an estimated \$43.4 billion per year in healthcare costs (Landrigan, 2002).</p>
<p>Crime</p>
<p>Childhood lead exposure has been linked to future criminal activity—more specifically particular violent crimes which include burglary, rape and aggravated assault (Bellinger, 1994, Nevin 2008, Wright, 1998). \$1.8 billion is said to be the direct cost for violent crimes which are linked to early childhood lead poisoning (Gould, 2009).</p>
<p>Tax Revenue</p>
<p>Being that there is a loss of IQ points in children with lead poisoning, there is a likelihood of lower academic achievement which can determine the type of employment children will obtain in the future, and what they will potentially earn. Having a lower income can effect society as a whole, as there will be less money available as tax revenue. \$25-35 billion is the estimated loss in tax revenues (Gould, 2009).</p>
<p>Return on Investment</p>
<p>It has been shown that if there is an investment of \$11 billion, then the net benefit could be up to \$270 billion. In other words, for every \$1 invested, that is a \$24 return (Gould, E., 2009).</p>

CHAPTER 3

CONCLUSION

After Flint, Michigan governmental authorities introduced a more corrosive water source into an already aging water system without adequate corrosion control, the percentage of children with elevated blood lead levels increased, particularly in socioeconomically disadvantaged neighborhoods. Water is a growing source of childhood lead exposure, because of aging infrastructure.

The literature shows that lead is a harmful neurotoxin, and childhood lead poisoning has an impact on many developmental and biological processes, most notably intelligence, behavior and overall life achievement. If this childhood lead poisoning has an estimated societal costs in the billions it is economically and morally sound rationale to invest in the known mechanisms for educational outreach, identification, awareness and prevention. However, since specific demographic evidence on age, gender and race as it relates to the health consequences is still limited, future research is still needed to implement a system that will adequately target and help the children in Flint. In summary, I recommend the plan of action in Table 4 to prevent further overall childhood lead exposure by pushing leading officials to enforce existing regulations, and secure funds for lead eradication.

Table 4: Plan of Action

<p>1. Conduct an environmental investigation for children with elevated blood lead levels or a history of lead exposure:</p> <ul style="list-style-type: none"> -Inspect homes and locations of that child's activity. -Measurements of environmental lead levels, including at a minimum <ul style="list-style-type: none"> ➤ Water ➤ House dust/ Paint ➤ Soil in play areas ➤ Other media as appropriate
<p>2. Environmental interventions should ensure ongoing exposure:</p> <ul style="list-style-type: none"> -Control current hazards. -Have a prompt and appropriate measures to reduce lead exposure (e.g. Action Plan) -Have safety measures in place by trained workers (e.g. OSHA) - Have proper disposal and enclosure of the eradication of lead -Testing for clearance to reoccupy space should be done prior to opening.

<ul style="list-style-type: none">➤ Provide temporary space for occupant's to protect from further exposure. <p>-Relocate children permanently to lead-safe housing to reduce further lead exposure.</p>
<p>3. Encourage state and local governments enforce laws dealing with lead/ chemical hazards</p> <ul style="list-style-type: none">➤ Serving ordinances➤ Holding owners to the housing codes➤ Identify changes within policy and enforcement to ensure that children are protected.
<p>4. Expand all existing federal, state, and local subsidies to help finance the lead hazard control efforts in economically distressed communities, or create subsidies for those communities without funds in place.</p>

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What influences do age, gender, and race/ethnicity have on the overall health consequences of excess lead in children?

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