ABSTRACT

The Prevalence and Effect of Blood Lead Levels on Body Mass Index in Children in Rural Western Kenya

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According to the World Health Organization (WHO), lead poisoning accounts for 0.6% of the global disease burden. It has been described as the silent epidemic plaguing developing countries. Children have been found to be most vulnerable to exposure to lead. The insidious effects of this level on children's growth and learning are well documented. However, multiple studies have also shown the harmful effects of sub-clinical BLL (<10 μg/dl), effects that are irreversible but entirely preventable.

The goal of this community-based research study was to investigate the prevalence of lead poisoning and the potential relationship between clinical and sub-clinical blood lead levels and body mass index (BMI) in a remote, rural location of western Kenya. Among children who have other major impacts on their growth, it is important to know the contribution of clinical and sub-clinical levels of lead. This cross-sectional study analyzes 2012 data of 292 children 14 years of age and younger in Nyanza Province. The average blood lead level (BLL) was 3.601 μg/dL with a range of <3.3 μg/dL to 35.7 μg/dL (SD=3.166 μg/dL). Only 1.03% of the sample (n=3) had blood lead levels above 10 μg/dL but 30% (n=88) had BLL between 5 μg/dL and 10 μg/dL. A level of 4.6 μg/dL was shown in as early as 1 month of age. This indicates exposure long before the child is able to play in or ingest contaminated soil.

The relationship between lead and BMI was non-significant and was confounded by age. There seems to be decreased but constant exposure to lead across age groups. Adjusting for age and gender, episodes of malaria and lead were both significantly associated with BMI percentile ($F=6.61$, $p<0.0001$, $r^2=0.133$). The comorbidity of lead and repeated episodes of malaria show a decrease of 19-61% in BMI percentile within an age group. These results indicate that lead poisoning is a concern in this community. Efforts should be made to locate the source of lead while preventative measures are taken to decrease exposure pathways.

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# THE PREVALENCE AND EFFECT OF BLOOD LEAD LEVELS ON BODY MASS INDEX IN CHILDREN IN RURAL WESTERN KENYA

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# DEDICATION

This study is dedicated to the children of the Nyakach Plateau and all those who suffer from lead poisoning.

# CHAPTER ONE

## Introduction

The developing world carries an incredible proportion of disease burden compared to the developed world. Many of these are preventable diseases. They continue to wreak havoc on the developing countries and can be attributed to factors such as unclean water, unsanitary living conditions, or poor nutrition. Children are especially vulnerable to disease and feel the greatest impact. Their chances of contracting diseases are greatly increased by their curious nature and by their exploration in unsanitary environments. In regards to lead poisoning, children are at greater risk because of increased hand-to-mouth behaviors. These behaviors increase their risk of accidently ingesting soil or dirt.

In 1992, Jerome Nriagu reported that the estimated percentage of infants with lead poisoning in Africa was 15-30%. According to the World Health Organization (WHO), lead poisoning accounts for 0.6% of the global disease burden today (2007). Between 1960s and the 1990s, the United States Center of Disease Control and Prevention (CDC) reduced the clinical blood lead level (BLL) from 60 μg/dl to 10 μg/dl. The insidious effects of this level on children's growth and learning are well documented. However, multiple studies have also shown the harmful effects of sub-clinical BLL (<10 μg/dl). The only way to rid the body of lead, a heavy metal, is through a treatment called chelation therapy. Although an individual may undergo chelation therapy, the negative effects of having had lead in the body are not reversible. Most of those living in

developing countries lack the resources to undergo treatment for lead poisoning. Thus, the only way to prevent the negative systemic effects of lead is to prevent exposure.

Attention to toxic contaminants and environment hazards are disregarded in favor of the prevention of primary health care issues such as high infant and maternal mortality rates, malnutrition, and endemic communicable diseases. Although attention on diseases associated with helminthic infections in developing countries continues to increase, the effects of chemical contaminants and their effects on development is understudied. This is one the reasons why lead poisoning has been described as the silent epidemic plaguing developing countries.

# CHAPTER TWO

## Review Of Literature

### *Prevalence*

As more research is conducted, it has been found that no threshold of blood lead level is considered safe, especially in young children. In response to current research, the United States Center for Disease Control and Prevention (CDC) lowered the clinical threshold of blood lead levels. In the 1960s, the clinical threshold of blood lead level was 60 µg/dL. This level has been steadily lowered until in 1991, the threshold was set to 10 µg/dl, which is the current level of clinical significance to this day. The clinical threshold continues to be 10 µg/dL even though current research point to the effects of neurodevelopmental effects at sub-clinical (less than 10 µg/dL) levels. In 2012, the CDC revised the blood lead level of concern and has set the reference blood lead level to 5 µg/dL (CDC 2012). In the past 3 decades in the United States, as the CDC lowered the acceptable threshold for normal blood lead level and pushed towards eliminating sources of lead around the house, the percentage of children with blood lead levels greater than 10 µg/dL has dropped from 88% to 1.21% (Chandran 2010). Other developed countries have similarly lower percentages of lead poisoning.

The global prevalence of lead poisoning has steadily decreased but continues to remain a problem in developing countries. In 2003, Pruss-Ustun and colleagues at the WHO Geneva convention compiled the burden of lead in adults and children in 14 regions. It was found that in 2000, an estimated 120 million people had blood lead levels

of 5-10 μg/dL and 120 million people had levels above 10 μg/dL (Pruss-Ustun 2003). Among the children, the data showed that roughly 97% lived in developing regions with 40% having blood lead levels above 5 μg/dL and 20% having blood lead levels above 10 μg/dL (Pruss-Ustun 2003). Of the 20% of children who had lead levels above 10 μg/dL, 99% lived in developing regions (Pruss-Ustun 2003). This shows that children, especially those in developing countries, are most at risk for the outcomes of lead poisoning. Kenya fell within the "AfrE" category, which included most of southeastern Africa. The urban mean for the "AfrE" category was 9.8 μg/dL in children and 10.4 μg/dL in adults (WHO 2003). There was no reported rural mean blood lead level. Among children in this region, 19.1% had blood lead levels of 5-10 μg/dL, 8.9% had blood lead levels of 10-20 μg/dL, and 9.5% had blood lead levels higher than 20 μg/dL (Pruss-Ustun 2003). The prevalence found in Kenya was similar to the global prevalence.

A meta-analysis conducted by Ngueta and Ndjaboue concluded that of the 11,148 peer reviewed articles, published between 1 January 2000 and 30 January 2013, about lead poisoning, only 16 studies were conducted in Sub-Saharan Africa (2013). This shows that a disproportionately small number of studies about lead poisoning are done in high-risk areas. Ngueta and Ndjaboue found that the geometric weighted mean of lead poisoning in children below the age of 6 years was 13.1 μg/dL (2013). Six of the nine studies had samples where over 50% had a blood lead level greater than 10 μg/dL (2013). Ngueta and Ndjaboue concluded that young children living in Sub-Saharan Africa have higher geometric mean blood lead levels than children living in the United States (Ngueta 2013).

Of the 11,148 published papers from 2000-2013 regarding lead poisoning, the finding that only 16 of these articles focus on Sub-Saharan Africa is alarming. If 97% of the 240 million people with lead poisoning are in developing countries, there should be more focus on developed regions such as Sub-Saharan Africa. Of the studies done in Sub-Saharan Africa, the majority take place in urban settings (Table A).

**Table A**

| | | | | | Blood Lead Levels (ug/dl) | | | |
|---|---|---|---|---|---|---|---|---|
| **Study** | **Place of study** | **Children's age (sample size)** | **Method of Collection** | **Setting** | **Mean** | **Median** | **Range** | **Prevalence (%) of BLL ge 10** |
| Nriagu 1997 | South Africa | 2-5 yo and 8-10 yo (n=1200) | Venous blood | Rural<br><br>Semi-urban | 3.8<br><br>10 | NA<br><br>NA | NA<br><br>NA | NA<br><br>NA |
| Pfitzner 2000 | Nigeria | 6-35 mo (n=218) | Venous blood | Urban | 15.2 | NA | 1-60 | 70 |
| Wright 2005 | Nigeria | 6 mo - 5 yo (n=64) | Venous blood | Urban | 11.2 | NA | NA | 55 |
| Mathee 2006 | | (n=429) | Venous blood | | 6.4 | 6.1 | 1-24.5 | 10 |
| Diouf 2003 | Senegal | 8-12 yo (n=330) | Venous blood | Rural<br><br>Urban | 5.21<br><br>9.97 | NA | 0.58-18.9<br><br>3.1-22 | 16.67<br><br>58.93 |
| Mathee 2007 | South Africa | 1st grade (n=383) | Venous blood | | 9.1 | 8.9 | 1-18.1 | 35 |
| Nriagu 2008 | Nigeria | 2-9 yo (n=653) | Venous blood | Urban | 8.9 | 7.8 | 1-52 | 25 |
| Olewe 2009 | Kenya | 6-59 mo | Capillary blood | Urban | 6 | 5.4 | 3.3-24.7 | 7 |
| Rollin 2009 | South Africa | newborn (n=67) | Cord blood | Urban | NA | 2.4 | 1.5-8.7 | NA |
| Naicker 2010 | South Africa | newborn (n=618) | Cord blood | Urban | 5.9 | NA | 2-17 | >50 |
| Tuakuila 2010 | Democratic Republic of Congo | 0-5 yo (n=100) | Venous blood | Urban | 12.4 | NA | NA | 63.5 |
| Keating 2011 | Nigeria | 12-18 mo (n=218) | Venous blood | Urban | 11.1 | 9 | 1-43 | 44.7 |
| Tuakuila 2013 | Democratic Republic of Congo | 1-5 yo (n=55) | Venous blood | Urban | 11.2 | 11.5 | 3-37.8 | 70.9 |

*History and Major Events*

The use of lead dates long before the effects of lead on the body were known. The Ancient Romans used lead for a variety of purposes including pottery glazing, piping, utensils, and to sweeten wine as early as 4000 BCE (Chandran 2010). By the early second century BCE, lead poisoning was recognized as both an acute and chronic condition (Pearce 2007). Lead toxicities were documented through the ages and lead poisoning itself was also known as "plumbism" and "lead gout" among many other names. Before the industrial revolution, miners, shipbuilders, wine drinkers, and potters were the most likely to be exposed to lead (Chandran 2010).

The industrial revolution opened the doors for widespread contamination in the form of lead-based paints, food containers, gasoline, and metal works. In 1894, Turner and Gibson first described lead poisoning within children in Brisbane, Australia, which later resulted in the passing of a lead paint prevention act in 1920 (Turner 1897, Needleman 1993). Following Australia's example, other countries began to eliminate lead paint as well. In the United States, for example, lead toxicities in children were linked to lead-based paint in the early 1900s and the United States Consumer Product Safety Commission banned lead in paint in 1977 (Chandran 2010). Efforts have been made in individual countries to lower and eliminate potential sources of lead (e.g. banning lead in paint and gasoline).

However, developing countries such as Kenya have no such ban. A study done by Nganga and his colleagues showed that most paint brands in Kenya continue to have high levels of lead (2012). This study showed that the average lead concentration of thirty-one samples from the eleven most popular paint brands was 14,900 parts per million (ppm),

which is 166 times higher than the United States allowed limit of 90 ppm lead in household paint. The highest concentration of lead found in a paint sample was about 69,000 ppm, which is 766 times higher than the U.S. limit (Nganga 2012). Chipping paint helps spread lead into the environment.

## *Toxicology*

### *Lead chemistry*

Lead (Pb) is a stable metallic element with an atomic number of 82 and atomic weight of 207.2. It is a soft, silvery gray metal that is highly resistant to corrosion but soluble in hot sulfuric and nitric acids (Kujawa 1997). There are four naturally occurring isotopes (204Pb, 206Pb, 207Pb, and 208Pb) and three of these stable isotopes, 206Pb, 207Pb, and 208Pb, are the daughter nucleus resulting from the radioactive decay of uranium, actinium, and thorium (Alsaleh 1994). Lead may also form salts with organic acids such as lactic acid and acetic acid or with stable organic compounds such as tetraethyllead and tetramethyllead (Alsaleh 1994). Lead is considered a heavy metal along with cadmium, chromium, and copper (Ooyoo-Okoth 2010). These heavy metals interact in various pathways in the body.

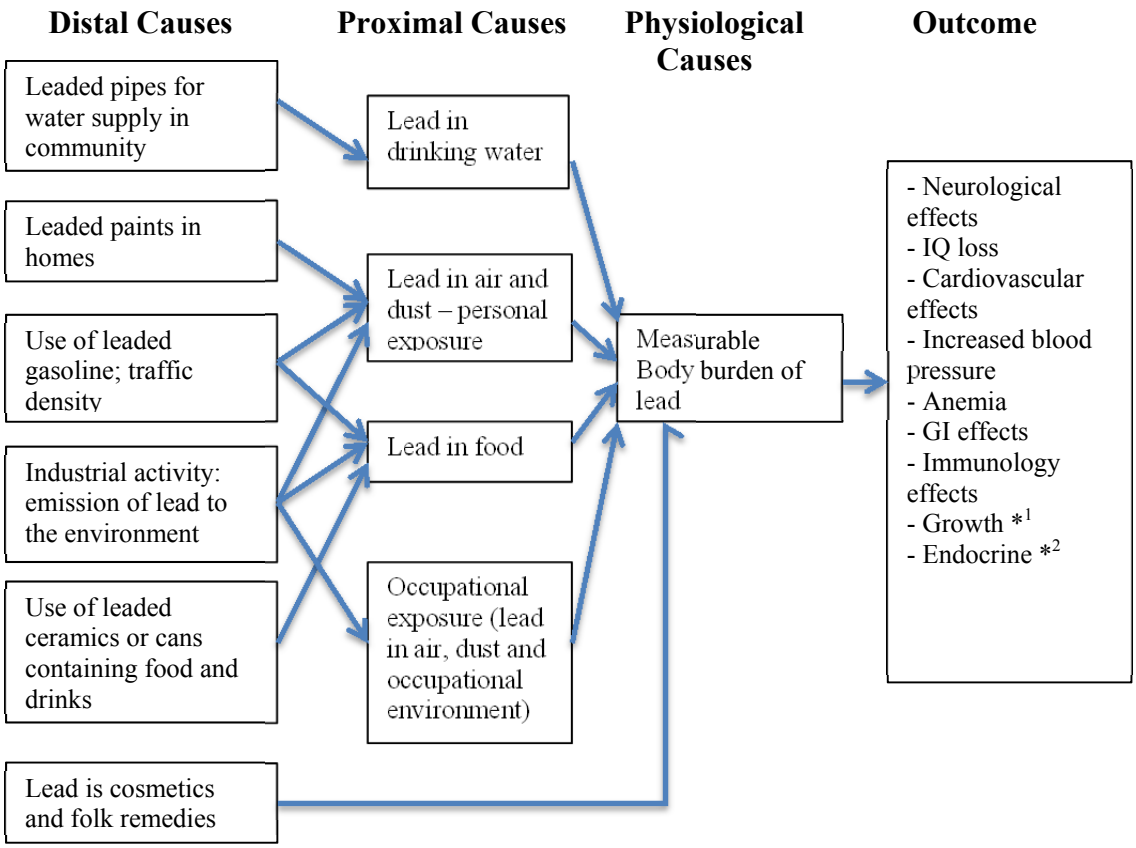
### *Exposure*

There are multiple potential routes in which an individual may be exposed to lead. The two most common methods of exposure to environmental lead is inhalation or ingestion. The majority, close to 99%, of total lead intake is ingested while 1% of lead in the body is from inhalation (Pizzol 2010). Children are more vulnerable than adults

because they exhibit increased hand to mouth behaviors both accidental and intentional (pica), have increased deposition of lead in soft tissue, greater neurotoxicity as a result of an immature blood-brain barrier, greater risk of damage at the cellular level during systemic development, or co-contaminant with another metal (Chandran 2010). The peak incidence of lead poisoning is around 18 to 30 months in most children (Chandran 2010). However, children with developmental delays (e.g. autism or pervasive developmental delay) that exhibit to prolonged and repetitive oral exploratory behaviors may have increased risk of ongoing and peaking blood lead levels (Chandran 2010).

Fewtrell and colleagues proposed a general framework for exposure pathway, as seen in Figure A (2004). This model proposes potential sources and their distance from a particular health outcome. Distal causes may contribute to multiple sources of proximal sources that the individual may be in contact with on a regular basis. For example, the use of leaded gasoline or resulting automotive exhausts, emission of lead from industrial activity, and lead in ceramics or cans that contain food or drink, all distal causes, may all contribute to the lead concentration in food, which Fewtrell labels as a proximal cause. Some routes of exposure may account for more of the lead poisoning found depending on the location and culture of that population.

Figure A




*1 Lauwers et al. 1986, Vivoli et al. 1993, Kim et al. 1995
*2 ATSDR 2007

According to the World Health Organization, lead in global commerce primarily arises from recycling lead-acid batteries (2010). Fewtrell and colleagues show in their results that leaded gasoline, which can lead to both direct exposure through air pollution and indirectly through food and dust, remains a good indicator a country's efforts to remove lead (2004).

*Absorption*

There are many differences between adults and children regarding lead exposure pathways, metabolism, and toxicity effects. Once lead is in the body, it can accumulate in blood, soft tissue, or mineralizing systems, such as bone and teeth. Each of these destinations has varying levels of absorption and half-life. Ziegler and colleagues found that in infants and young children, the average fractional gastrointestinal absorption of lead is much greater than the absorption rate in adults (Ziegler 1978). Not only do children have a higher absorption fraction than adults, this absorption is increased when other nutritional deficiencies, e.g. calcium or iron deficiencies, are present (Bellinger 2004). The precise absorption rate and retention rate depends on a variety of variables and reminds highly variable throughout different studies. One study found that the net absorption rate averaged 41.5% while the net retention rate averaged 31.7% (Ziegler 1978). Mykkanen and colleagues showed that lead is rapidly taken up by the gastrointestinal tissue and slowly moved into the circulation (1981). Mykkanen and colleagues found that absorption rate and retention rate averaged around 40% (1981).

In blood, the half-life of lead varies from about 28-36 days (Rabinowitz 1976, ATSDR 2007). In soft tissue, the half-life of lead is about 30-45 days (Rabinowitz 1976, ATSDR 2007). Barry and colleagues show that the concentration of lead in soft tissue in children is comparable to the concentration of lead in soft tissue in female adults (1975). Small children absorb approximately 50% of ingested lead while adults absorb approximately 10% of ingested lead (Peraza 1998). The higher rate of absorption in children may be due to the increase in intestinal transport proteins in periods of rapid growth (Sargent 1994).

In bone, the half-life of lead is highly variable. In 1991, Rabinowitz and colleagues reported that with occupational exposure, lead half-life in bone could is between 5-19 years. Other studies report that lead half-life in bone is about 25-40 years (ATSDR 2007). Numerous studies have shown that the majority of the body burden of lead is found in bone. Barry and colleagues suggests that children retain lead in bone in a different capacity than adults (1975). Barry and colleagues estimate that in adults, over 90% of the body burden of lead rests in bone, 70% of which is in dense bone but the exact lead body burden in children is unknown (1975). Peraza and colleagues estimated that roughly 95% of the body burden of lead in adults is found in bone (1998). Once lead is stored in bone, it can be mobilized in the event of physiologic and pathologic conditions (Peraza 1998). One example of the mobilization of long-term blood stores of lead is the transfer of lead from mother to infant during pregnancy and or lactation (Peraza 1998).

### *Effects of micronutrients on lead toxicity*

The balance, or lack of there of, of critical micronutrients plays a vital role in the absorption and retention of lead in the body. Calcium and iron are two of the more commonly studied micronutrients including how these individual micronutrients interact with lead and how lead interrupts important pathways.

#### *Calcium*

Lead interrupts calcium pathways by: blocking the entry of calcium into nerve terminals thereby inhibiting the release of neurotransmitters, competing with calcium at uptake channels, and inhibiting varying types of calcium channels (Peraza 1998).

Numerous studies have shown that the absorption and retention of lead intake in the gastrointestinal system depends on the micronutrient status of the lumen, where calcium deficiency may lead to increased intestinal absorption and body lead retention (Peraza 1998, Mykkanen 1981, Six 1970). A study of pregnant women in Mexico City demonstrated the inverse relationship between dietary calcium and absorption of lead by the gastrointestinal tract (Peraza 1998). Having excess dietary calcium, on the other hand, indicated a small decrease in lead absorption (Peraza); however, this difference is less dramatic than the one caused by insufficient calcium. Peraza concluded that the sufficient balance of dietary calcium was the best method to combat lead intoxication (1988).

*Iron*

Nutritional and situational iron deficiency promotes lead toxicity and increase lead absorption. In a study of preschool children, dietary iron intake was found to have a negative relationship with blood lead and higher dietary iron intake was associated with lower blood lead levels (Hammad 1996). The co-occurrence of iron deficiency and lead poisoning are known to cause a more severe form of anemia when combined (Kwong 2004).

*Others*

There are many other components that affect the absorption of lead. Balanced nutrition as well as total food intake and higher fat intake have been linked with decreased gastrointestinal lead absorption (Peraza 1998). Children in developing countries however, may be unable to sustain a balanced diet as a result of lack of resources. These factors, along with calcium, iron, and vitamin D, may be a potential

intervention route in decreasing the severity of lead toxicity, especially in children (Peraza 1998). Nutritional intervention would not only improve overall health of an individual in vulnerable populations, but could also decrease or prevent lead intoxication.

### *Leads effect on body*

Individuals that have elevated blood lead levels present both symptomatic and asymptomatically. Some potential outcomes from lead toxicity include abdominal colic, growth failure, hearing loss, microcytic anemia, renal disease, seizures, encephalopathy, and cardiovascular disease (Chandran 2010, Fewtrell 2004 – Figure A)

*Systems affected by lead*

Lead poisoning and its neurological effects have been studied in great detail. Lead interacts in the nervous system in two ways: interference with neurotransmission and the disruption of cell migration, specifically cell adhesion molecules, during critical times of brain development (Chandran 2010). The neurobiological effects of lead give more specifically cognitive symptoms. Such symptoms include poor academic achievement, decreased intelligence quota (IQ), sensory-motor deficits, behavior disturbances, decreased attention, encephalopathy, and decreased language function (Alsaleh 1994, Needleman 1990).

Before the early to mid 1900s and the development of chelation therapy, a large proportion of children with high blood lead levels died. An estimated 45% of children, with greater than 60 μg/dL, who displayed signs and symptoms of encephalopathy ended up dying at an early age (Chandran 2010).

Bellinger and colleagues found that peripheral neuropathies tended to be more prominent in adults with lead poisoning while central neuropathies were more prominent in children (2004). Their results suggest that the peripheral nervous system damage in adults tend to reverse to some extent after the elimination of exposure but the central nervous system damage in children is irreversible (Bellinger 2004). This is another reason why early and active prevention to exposure is essential in children.

Significant studies have shown that there are no safe thresholds regarding the toxic effects of blood lead levels on the brain. Chandran and colleagues showed that each rise in blood lead level above 10 μg/dL resulted in a decline of 2-3 points in a child's IQ (Chandran 2010). More striking, however, was the data that suggest that decline in IQ may be higher in children with blood lead levels below 10 μg/dL than in children with blood lead levels above 10 μg/dL (Chandran 2010). A sustained elevated blood lead level at a young age has severe repercussions in the form of decreased academic and intellectual performance.

Cardiovascular effects of blood lead levels are mainly seen in adults. Elevated blood lead level may lead to an increase in blood pressure, which in turn could cause other cardiovascular diseases including ischemic heart disease (WHO 2003). For very 5 μg/dL increase in blood lead level between 5-20 μg/dL, blood pressure has been found to increase 1.25 mmHg in men and 2.4 mmHg in women (Fewtrell 2003). Other studies found that bone lead levels offer a better predictor of elevations in blood pressure than blood lead levels (ATSDR 2007).

At low exposures and low blood lead levels, there may also be renal effects. Proximal tubular nephropathy, glomerular sclerosis, and interstitial fibrosis characterize

lead nephrotoxicity (Diamond 2005, Loghman-Adham 1997). Decrease glomerular filtration is evident at blood lead levels below 10 μg/dL and this is more concerning than enzymuria and proteinuria, which is seen at blood lead levels above 30 μg/dL (ATSDR 2007). Pathological changes occur at blood lead levels over 50 μg/dL (ATSDR 2007). Increased blood pressure may be the cause of secondary decrease in glomerular filtration rate. At high exposures and high blood lead levels, colic is a consistent symptom of lead poisoning (ATSDR 2007). However, this occurs at blood lead levels greater than 60 μg/dL and is no longer commonly seen.

Lead has hematological effects as well. Lead inhibits t-cell function and affects cartilage mineralization (Chandran 2010). Blood lead levels may also inhibit rate-limiting enzymes such as ferrochelatase and delta amino leyulinic acid dehydrogenase in the heme synthesis pathway, leading to the accumulation of heme intermediates and microcytic hydrochronic anemia (Chandran 2010). Lead-induced anemia occurs at blood lead levels below 20 μg/dL (ATSDR 2007).

Since Nye's report in 1929, there have been a number of studies reporting the association between blood lead levels and anthropometric measures, such as height and weight (ATSDR 2007). There have been mixed results regarding the relationship between lead and anthropometric development. Sanin and colleagues found that in 1 month old Mexican infants, blood lead levels was inversely associated with weight gain (Sanin 2001). Similarly, in the Cincinnati Prospective Study, Dietrich and colleagues showed that a higher prenatal blood lead level was associated with reduced birth weight (Dietrich 1987). Schwarts and colleagues showered that the average blood lead level of children

who were on average 59 months old, was negatively associated with height, and had an estimated 1.5% decrease (Schwartz 1988).

Other studies show no significant relationship between lead and growth. A longitudinal cohort study performed by Kim and colleagues studied the relationship between chronic lead exposure and physical growth in children. Bone lead was not associated with physical growth but dentin lead had a positive association with BMI (Kim 1995). The non-significant results indicate that chronic lead exposure during childhood may result in resisting obesity in adulthood (Kim 1995). Evidence suggests that children exposed to lead in early childhood experience greater BMI between the ages of 7 and 20 years compared to those that were less exposed (Kim 1995). Similarly, Min and colleagues found a significant association between blood lead and decrease height and total arm length, but not an association between blood lead and weight or BMI (2008).

### *The Current Study*

This community-based research focuses on children on the Nyakach plateau in the Nyanza province of rural western Kenya, with the goal of developing interventions to decrease and ultimately prevent exposure to environmental lead, if found to be prevalent. The Nyakach Plateau is roughly 36 km (22.4 miles) southeast of Kisumu, Kenya and 12 km (7.5 miles) southeast of Lake Victoria. The dominant tribe of this area is Luo and speaks Dholuo as the primary language. The vast majority of residents have no modes of transportation other than walking. This very poor population has the highest HIV rate in

Kenya (Agot 2010). The majority of those living in this community survives by subsistence farming.

Some baseline testing of the fifteen most frequented water sources was conducted in 2010. These tests looked for heavy metals, including lead, and biological contaminants in the water. Due to a mix-up involving returning to the field to resample, the locations noted in the results did not correspond with original sampling locations. Two of the fifteen locations tested positive for lead. But because of the source mix-up, the exact location of lead contamination remains elusive. The positive results for two of the sources suggest that a possible source of lead is the water source, but this has not been confirmed.

Buildings in the area are traditional mud huts with thatched or metal roofs, or brick structures with concrete floors. Lead-based paint is not a likely source around the house but may be a potential source at schools or village market place shops. Since almost no one can afford a car on this high, rocky plateau, the transportation modes are limited to walking, bicycles, and the occasional motorcycle taxi, ruling out leaded gasoline as a likely cause.

As stated previously, out of the 240 million people with blood lead levels greater than 5 μg/dL, 97% live in developing regions. Developing regions carry a disproportionately large burden of lead poisoning. This shows that children, especially those in developing countries, are most at risk for the outcomes of lead poisoning.

It is important to gauge the prevalence of lead poisoning in the population on the plateau because of the negative, accumulating, and long-term effects that lead can have on multiple systems of he body. This study proposes to evaluate the prevalence of lead poisoning in children on the Nyakach plateau and its relationship with physical

development, which will be measured by body mass index. With this knowledge, there will be a better foundation from which to address the prevention of lead exposure and the search for lead in the environment.

# CHAPTER THREE

## Hypothesis

*Research Question #1* What is the prevalence of abnormal blood lead levels in Luo children in Western Rural Kenya?

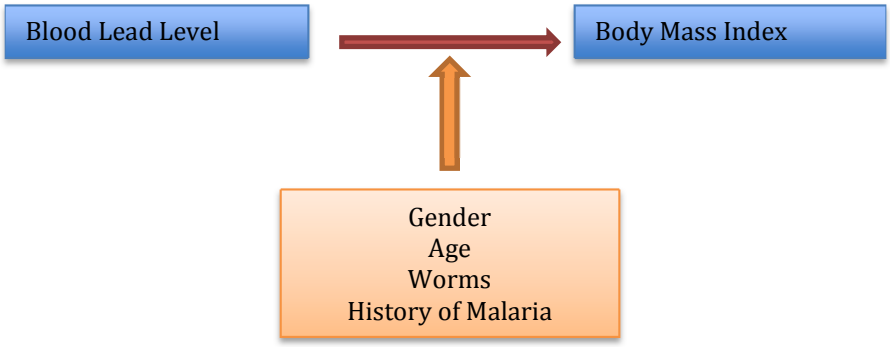
*Hypothesis #1:* There will be a similar proportion of abnormal blood lead levels in Luo children living in rural Kenya than that found in other rural communities in Africa.

*Null Hypothesis #1:* There will be a dissimilar proportion of abnormal blood lead levels in Luo children living in rural Kenya than that found in other rural communities in Africa.

*Research Question #2* How does blood lead levels affect physical development among Luo children?

*Hypothesis #2:* Children with higher blood lead levels have a lower BMI than children with lower blood lead levels after controlling for age, gender, helminthic infection, and cases of malaria.

*Null Hypothesis #2:* Children with higher blood lead levels have a BMI that is not statistically significantly different than children with lower blood lead levels after controlling for age, gender, helminthic infection, and cases of malaria.




*Description of Study*

This cross-sectional study was designed to determine the baseline prevalence of blood lead levels in Luo children living in western rural Kenya and to study the effect of abnormal lead levels on growth while controlling for age, gender, helminthic infections, and history of malaria. A pilot study was conducted in 2011 in an annual temporary clinic run by the U.S.-based non-profit organization, Straw to Bread, as part of its comprehensive development project among the Luo community living on the Nyakach Plateau. Abnormal blood lead levels were documented among some of the Luo children, so a follow-up study was done in May of 2012, in which blood tests were done on all children who visited the clinic.

# CHAPTER FOUR

## Methodology

### *Setting*

All data were collected in a 10- day period from May 19 to May 29, 2012 from subjects who came to an annual temporary clinic set up through a U.S.-based non-profit organization, Straw to Bread. This temporary clinic was located on the Nyakach Plateau, in the Nyanza District in rural western Kenya (see Appendix A). Straw to Bread partners with this particular community in a comprehensive program of development including projects related to healthcare, education, clean water, sustainable agriculture, and small business. The location is approximately 36 km southeast of Kisumu, Kenya and 12 km southeast of Lake Victoria at a latitude of 0°21'23" and at an elevation of 1550m.

The local population attending the clinic belonged almost exclusively to the Luo ethnic group and spoke Dholuo as their native language. Clinic attendees were mostly subsistence farmers or pastoral herders and their families who lived within walking distance of the clinic. Some of the clinic attendees traveled from other areas on the plateau on motorized transport.

### *Sample*

The children who were studied were patients at the temporary clinic run by Straw to Bread. The exclusion criteria were children over the age of 14 and children from whom blood could not be drawn. A total of 292 children participated in the study.

*Study Design*

This cross-sectional study used the data that were previously collected for clinical purposes. When patients were registered, they were asked a series of questions as part of a larger Community Health Assessment Profile (CHAP) (see Appendix B). Data was collected by trained laboratory technicians and trained volunteers. The oldest relative present with the patient answered the questions. Patients that visited in 2012 were seen by one of three physicians trained and board certified in the United States. Patients were asked to go to lab where anthropometric measurements and blood samples were collected. Following lab, patients waited in line to see one of the available physicians.

A single venous blood sample was collected for each patient by a trained technician, using a lead-free plastic vacutainer containing an anti-coagulant. These samples were then analyzed using Magellan Diagnostics LeadCare II according to the user manual. This machine measures blood lead quantitatively and is sensitive from 3.3 μg/dL to 65 μg/dL. Values displayed as "<3.3 μg/dL" are recorded and analyzed as 0 μg/dL. All devices were used in accordance with procedures in their respective user manuals by trained technicians and volunteers.

Height was measured in centimeters by having the patient stand with his/her back to a measuring tape, which was installed on a wall. Mass was measured in kilograms using a spring-scale purchased locally in Kisumu, Kenya.

Body Mass Index (BMI) was calculated according to the formula: Mass (kg)/ Height$^2$ ($m^2$). BMI for age percentile was calculated using the World Health Organization Anthro software, version 3.2.2, and World Health Organization AnthroPlus software. WHO Anthro is used for patients under 5 years of age and WHO AnthroPlus is used for

patients 5 and older. Patients who were 3 standard deviations above or below the WHO standardizations were excluded from further analysis because the WHO Anthro software did not generate a numerical BMI percentile. BMI quartile designation refers to BMI in relation to the study sample.

Information for history of malaria was obtained as a part of past medical history. Patients (or caretaker) self-reported how many times they (or their charge) had contracted malaria in the past 6 months. This information was a part of the CHAP questionnaire, which was asked during registration.

Physicians diagnosed a patient as having worms based on the physical exam. Patients who were found to have worms were treated with Mebendazole.

### *Statistical Analysis*

Clinical data was used to assess prevalence of lead poisoning in children. All clinical data was coded and double entered into Microsoft Excel (Redmond, Washington) and then imported into SAS (Carey, North Caroline) statistical software version 9.3 for Windows 7. Descriptive statistics included frequencies, mean, median, standard deviation, and range (when appropriate). Analytic statistics included bivariate analysis using the chi-square statistic for contingency tables (categorical variables) or t-test and Pearson's r correlation with continuous variables. Multivariate analysis was done using multiple regression, logistical regression, and analysis of variance. Alpha was set at 0.05.

### IRB

This study was approved by the Baylor University Institutional Review Board.

# CHAPTER FIVE

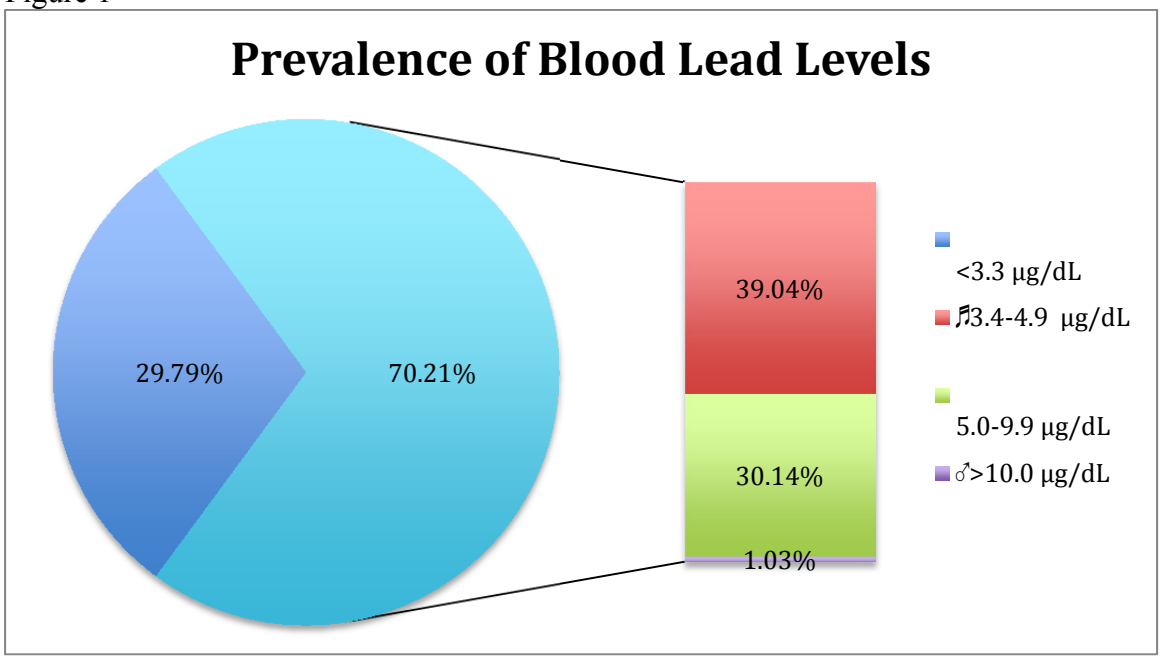
## Results

The study consisted of 292 children who were 14 years of age and younger. The average age was 7.241 years with a range from 1 month to 14 years. The standard deviation was 4.269 years. Of the 292, 45.55% were male (n=133) and 54.45% were females (n=159).

The average blood lead level was 3.601 µg/dL with a range of <3.3 µg/dL to 35.7 µg/dL (SD=3.166 µg/dL). The median blood lead level was 4.10 µg/dL. Of the total sample, 70% (n=205) tested positive for blood lead. The average blood lead level of those that tested positive for blood lead level was 5.00 with a range of 3.4 µg/dL to 35.7 µg/dL. Figure 1 shows the distribution of the sample by blood lead level. The proportion with blood lead levels less than 3.3 µg/dL was 30% (n=87). The lower sensitivity limit of the Magellan Diagnostic LeadCare II analysis was 3.3 µg/dL. Thus, all values with the reading of "<3.3 µg/dL" were analyzed as 0.0 µg/dL. Of our sample, 39% (n=114) had blood lead levels between 3.4-4.9 µg/dL. There were 30% (n=88) that had blood lead levels from 5.0-9.9 µg/dL. These blood lead values are between the new reference level as defined by the CDC 5 µg/dL and the current clinical threshold of 10 µg/dL (CDC 2012). Only 1% of the sample (n=3) had blood lead levels above 10.0 µg/dL.

Lead was not significantly related to BMI ($F=1.39$ $p=0.25$). However, age was significantly associated with both BMI ($F=28.13$ $p<0.0001$) and lead ($F=3.92$ $p=0.0209$), confounding the relationship between lead and BMI.

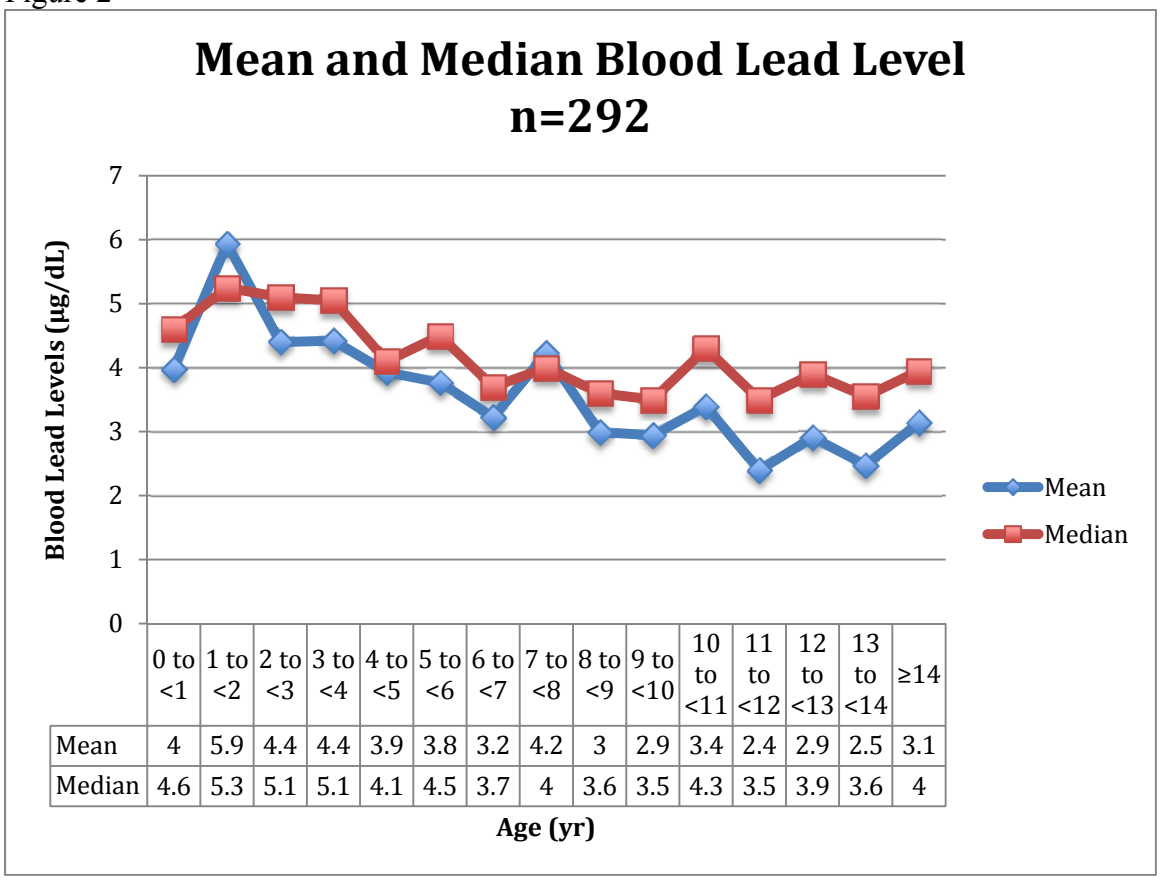
Figure 1




Blood lead levels were not statistically different between males and females but did vary significantly with age (F=1.94, p=0.023). When blood lead levels were distributed by age rounded to years, the younger children had a higher blood lead burden than the older children. As shown in Figure 2, children between 1 and 2 years of age, on average, had the highest blood lead (mean=5.94 median=5.25 n=29). The data shows that there is an early exposure to lead that continues until the age of 11. Children between the ages of 11 and 12 had the lowest blood lead (mean=2.39 median=3.5 n=23). The majority of the median blood lead values are higher than the mean blood lead values. This shows that the data is skewed to the left. The high frequency of 0.0 µg/dL values (n=87) is pulling the mean down. There is a statistically significant difference (t= 2.46 p=0.0171) between the group with the highest mean blood lead level (1-2 year olds) and the group with the lowest mean blood lead level (11-12 year olds). Because the half-life of lead in

blood is roughly 30 days, the fact that lead was measured at all ages indicates that there is repeated exposure to lead.

Figure 2




| | 0 to <1 | 1 to <2 | 2 to <3 | 3 to <4 | 4 to <5 | 5 to <6 | 6 to <7 | 7 to <8 | 8 to <9 | 9 to <10 | 10 to <11 | 11 to <12 | 12 to <13 | 13 to <14 | ≥14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mean | 4 | 5.9 | 4.4 | 4.4 | 3.9 | 3.8 | 3.2 | 4.2 | 3 | 2.9 | 3.4 | 2.4 | 2.9 | 2.5 | 3.1 |
| Median | 4.6 | 5.3 | 5.1 | 5.1 | 4.1 | 4.5 | 3.7 | 4 | 3.6 | 3.5 | 4.3 | 3.5 | 3.9 | 3.6 | 4 |

Figure 3 and 4 show mean blood lead level and median blood lead level, respectively, isolated and plotted with an estimated bone lead half-life curve. The average blood lead level for each age group represents an estimate of how the blood lead levels might look within an individual had they retained the majority of their blood lead as bone lead. The half-life curve starting from the beginning mean represents the course of bone lead if the child had only one initial exposure, the one within the first year of life. The bone half-life from the highest mean blood lead level shows the difference in bone lead burden. Any point above either bone lead half-life curve would reset the half-life line.

Figure 3

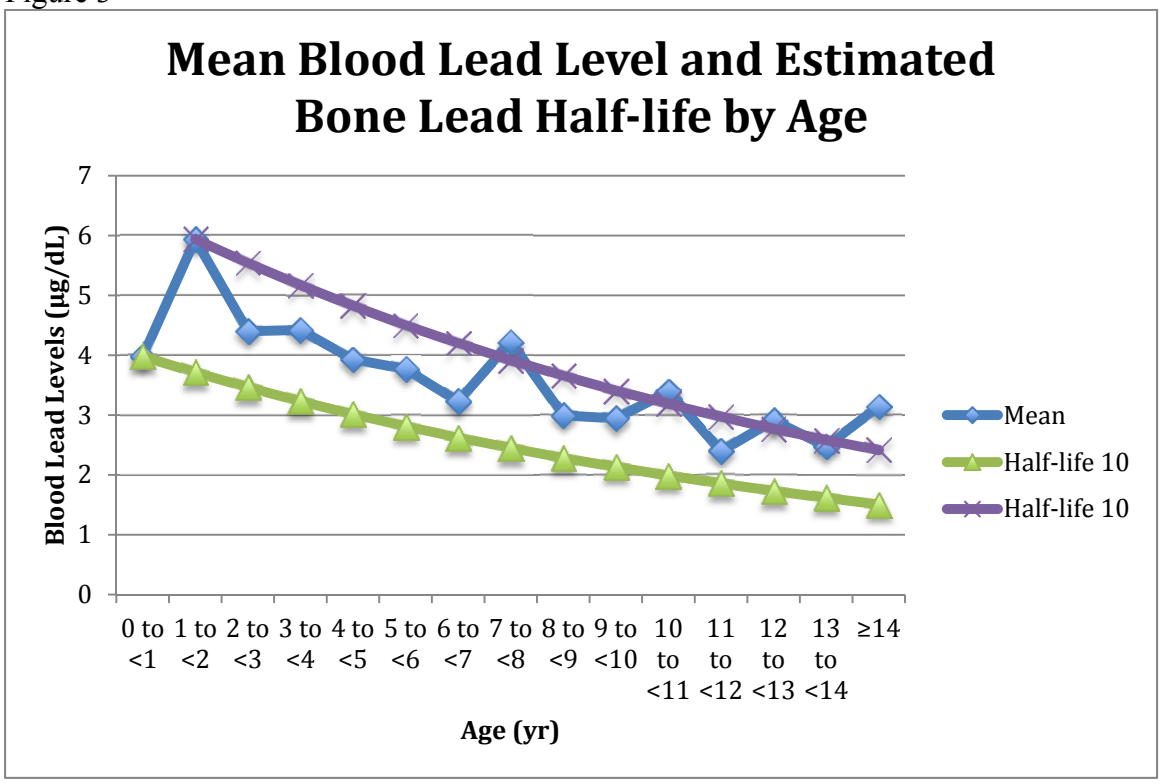
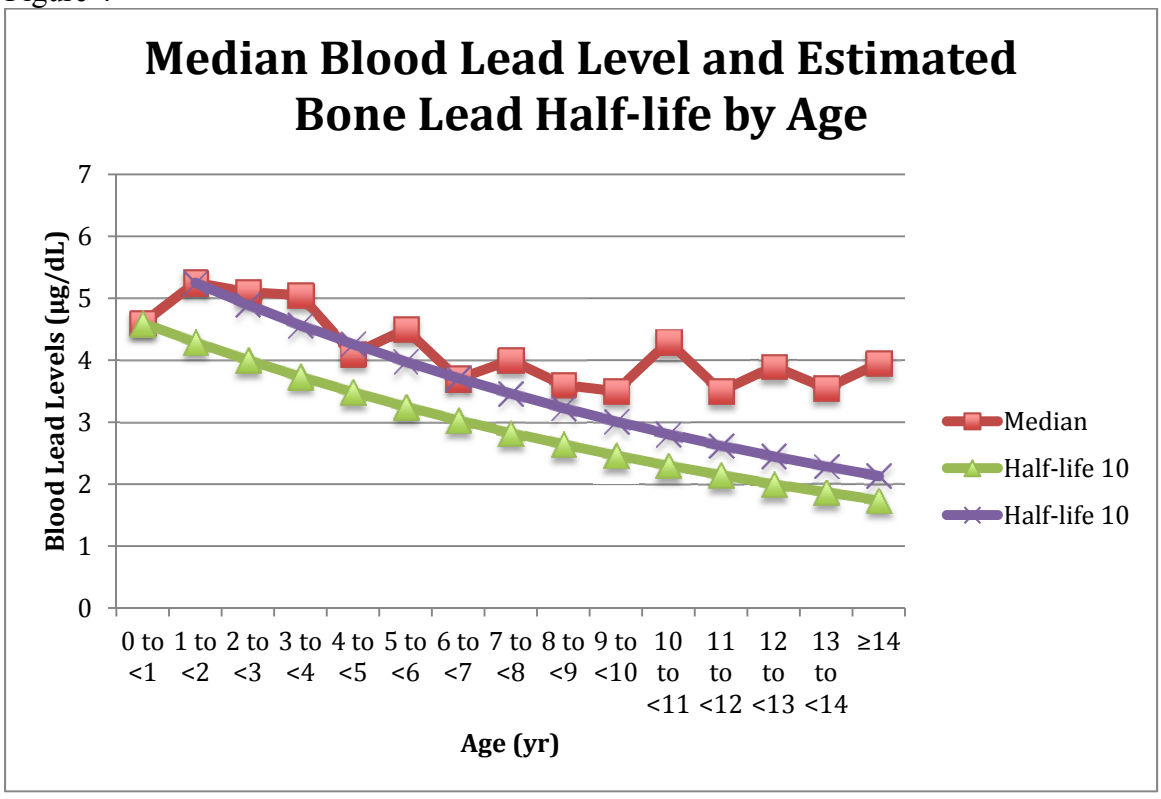


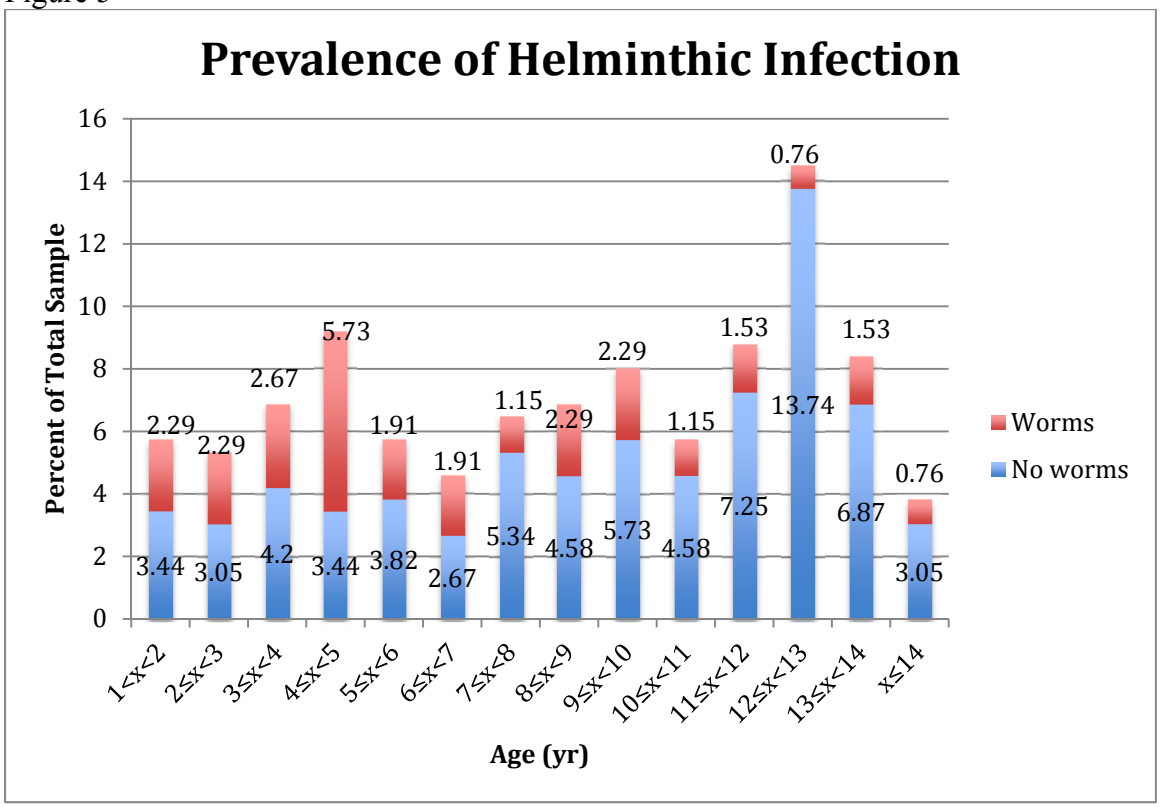

Figure 4



Helminthic infections were present within all age groups (Figure 5). Of the total sample, 25% were diagnosed and treated for worms and 75% did not have worms. Worms were found to be more prevalent in children under the age of 7. As shown in Figure 5, when stratified by age, children between the ages of 4 and 5 had the largest percentage of worms holding 5.73% of the worm burden. Children between the ages of 3 and 4 had the second highest worm burden at 2.67% (Figure 5). Children between the ages of 12 and 13 had the lowest worm burden at 0.75% (Figure 5).

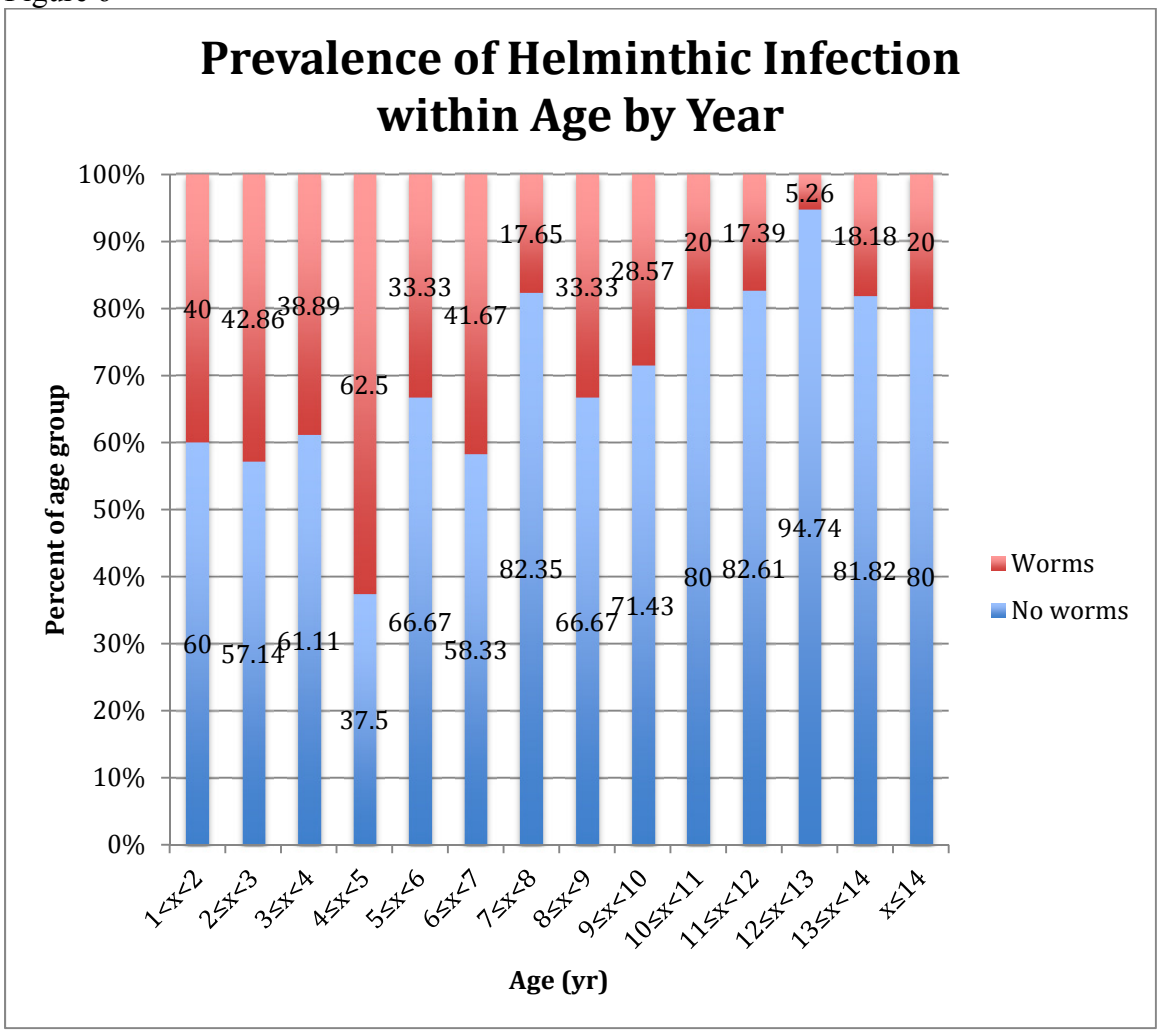
Helminthic infections were significantly related to BMI (t=-1.98 p=0.0492) but displayed a relationship contrary to the one found in literature. In the sample, having worms displayed a direct relationship with greater BMI. This variable was added to the regression model and analyzed further (Table 3).

Figure 5



In Figure 6, the data was rearranged to show the prevalence of worms within each age year. The younger children had a higher prevalence of helminthic infection. Two third of the individuals in the 4-5 year old age group had worms, as shown in Figure 6. The 12 year olds had the lightest worm burden. Only 5% of that age group was positive for worms.

Figure 6

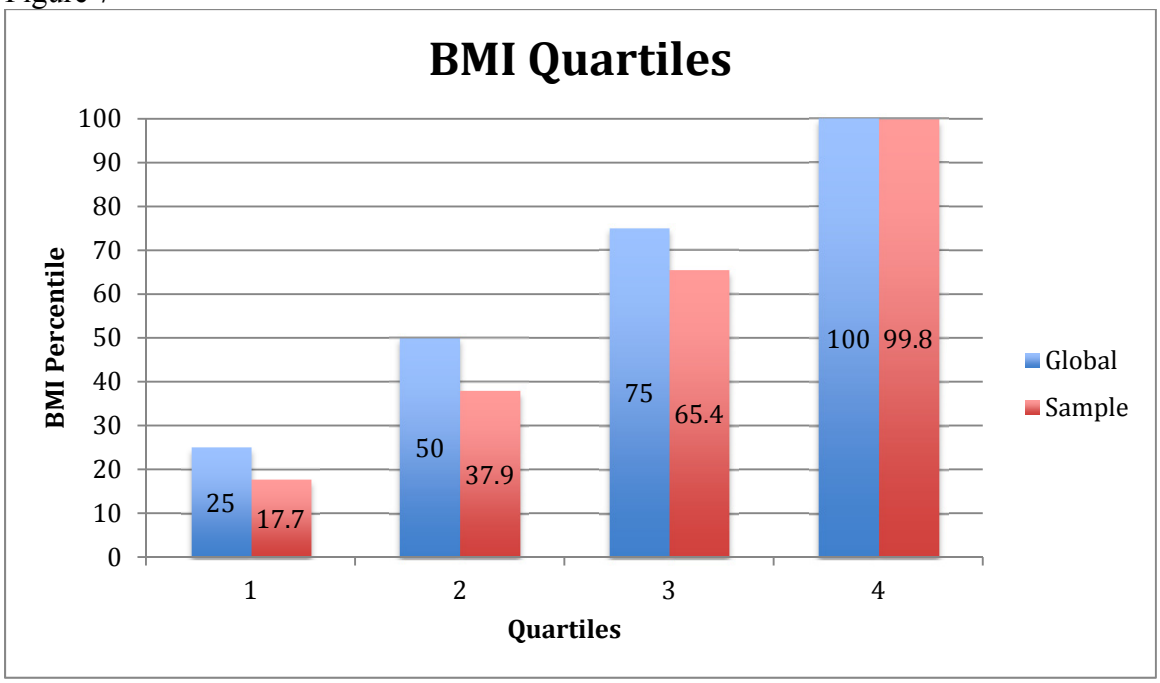



Age was significantly associated with BMI ($F=28.13$ $p<0.0001$). BMI percentiles were used instead of BMI z-scores because of its intuitive distribution. BMI percentile adjusts for age and gender. The mean BMI percentile was 42.52 with a range from 0.30

to 99.8 (SD=29.89 n=272), as seen in Figure 7. There was no significant difference in BMI between males and females. BMI quartile values for the sample were lower for all four categories as shown in Figure 6. Shockingly, 50% of the sample had BMI percentiles of 37.9 and below.

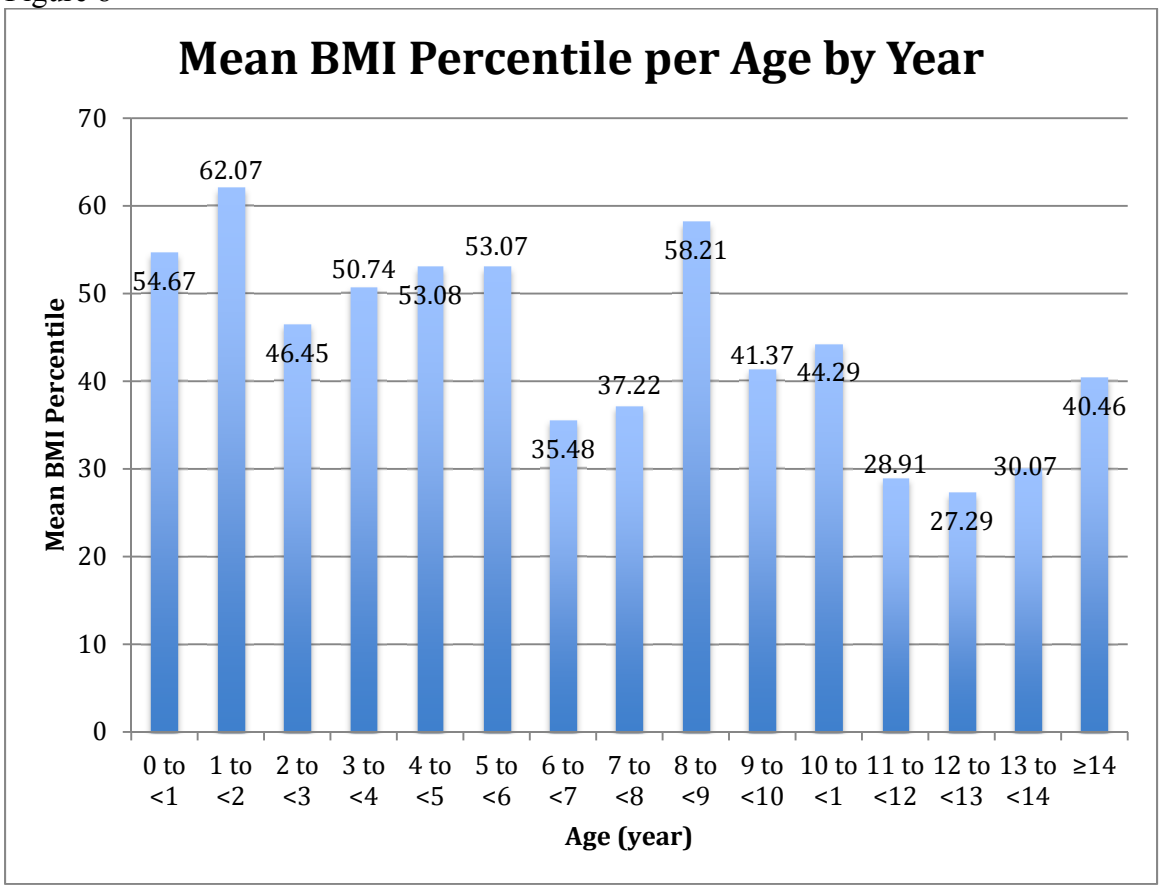
Twenty subjects had BMI z-scores greater than 3 standard deviations and were excluded from analysis because the WHO Anthro software, version 3.2.2, and WHO AnthroPlus software was unable to calculate a numerical BMI percentile. These twenty values showed up as "NA" and were left out of further analysis. Of these twenty z-scores, sixteen were in the positive direction (> +3SD) while four were in the negative direction (> -3SD). Fifteen subjects were 1 year old and younger, four subjects were between the ages of 1.5 and 3 years and one subject was 10 years old.

Figure 7



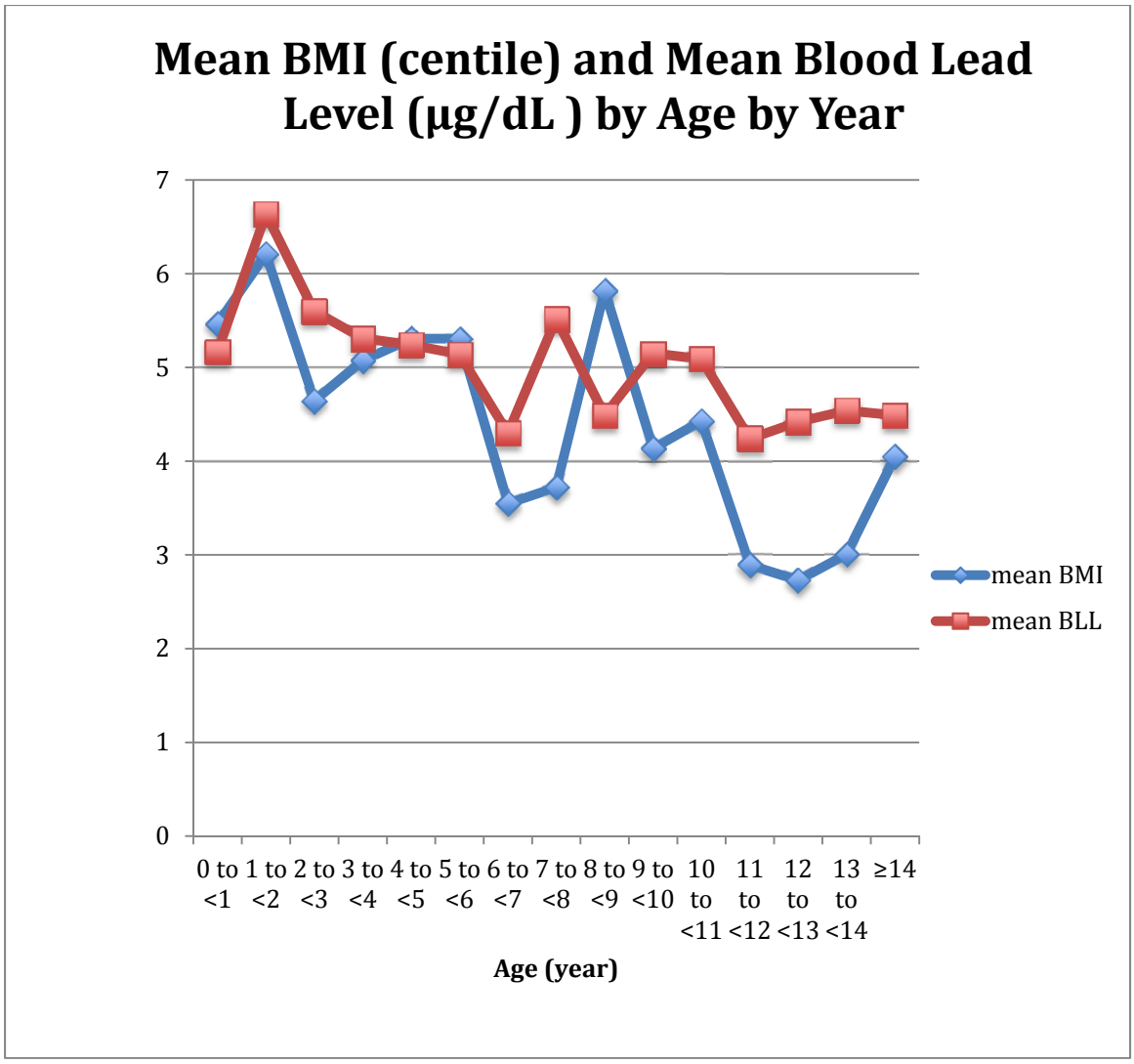
The average BMI percentile for each age group is shown in Figure 8. The younger children had a higher average BMI percentile than the older children. Children between the ages of 1 and 2 had the highest average BMI percentile at 62.07. Children between the ages of 12 and 13 had the lowest average BMI percentile at 27.29. These two groups are statistically different from one another ($t=4.35$ $p<0.0001$).

Figure 8




Overall, there is an inverse relationship between BMI percentile and age as shown in Figure 9 and 10. Figure 10 trichotemized the age groups into the youngest (1 month to 4.9 years old), middle (5 to 9.9 years old), and oldest children (10 years of age and older).

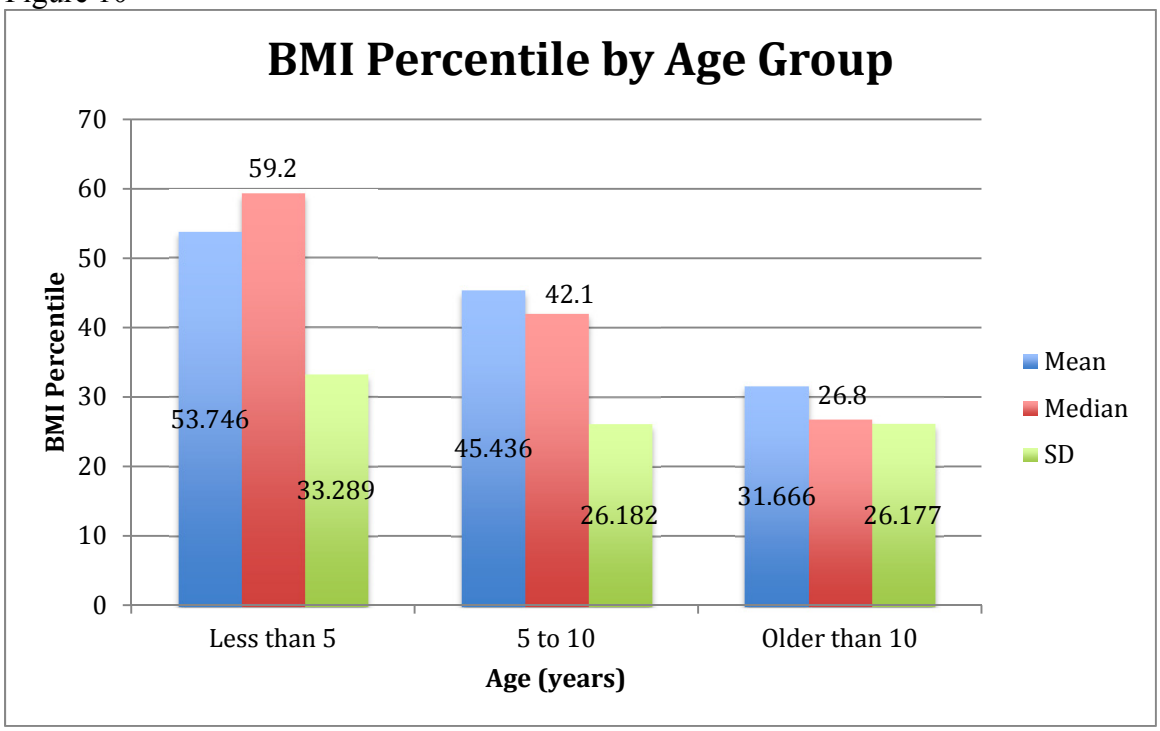
Figure 9




Blood lead levels were then trichotemized where group 1 represented those with no blood lead, group 2 represented those that had a BLL of 2.4-9.9µg/dL and group 3 represented those that had a blood lead level of greater than 10 µg/dL. This trichotemized lead designation was not a significant predictor of BMI percentile (F=1.39 p=0.250). Age was also a significantly related to blood lead level (p=0.0006 F=5.21). Age was a confounding variable. Adjusting for age, lead was a significant predictor of BMI percentile ($p<0.0001$ $F=28.58$ $r^2= 0.1209$). There is an inverse relationship between blood

lead level and BMI percentile: the greater the blood lead level, the lower the BMI percentile, as seen in Table 1 and Figure 11.

Figure 10

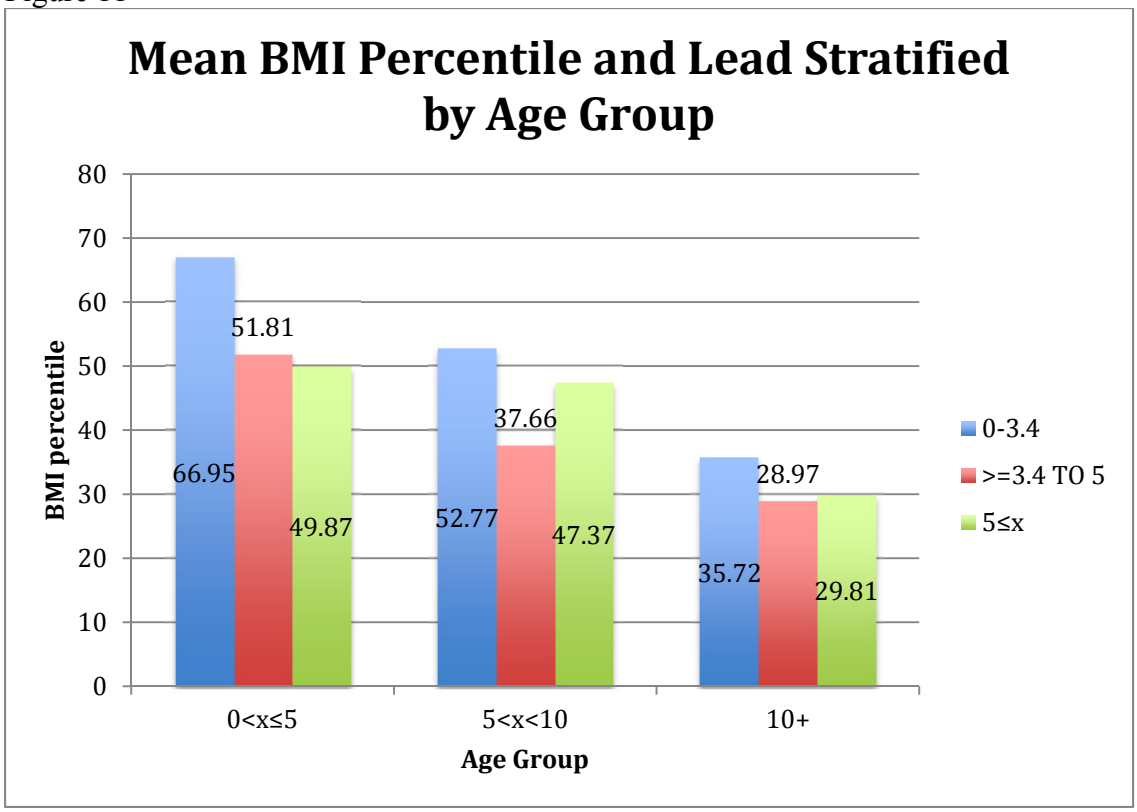



As seen in Table 1 and Figure 11, blood lead level and age were both stratified into three groups and the BMI percentiles for each category was plotted against each other. The BMI negative inverse trend between BMI percentile and age was seen in all lead categories. Within each age group however, only the youngest age group showed a decrease in BMI percentile as blood lead level increased. The older two age groups had more varying results.

Table 1: BMI percentile and lead stratified by age

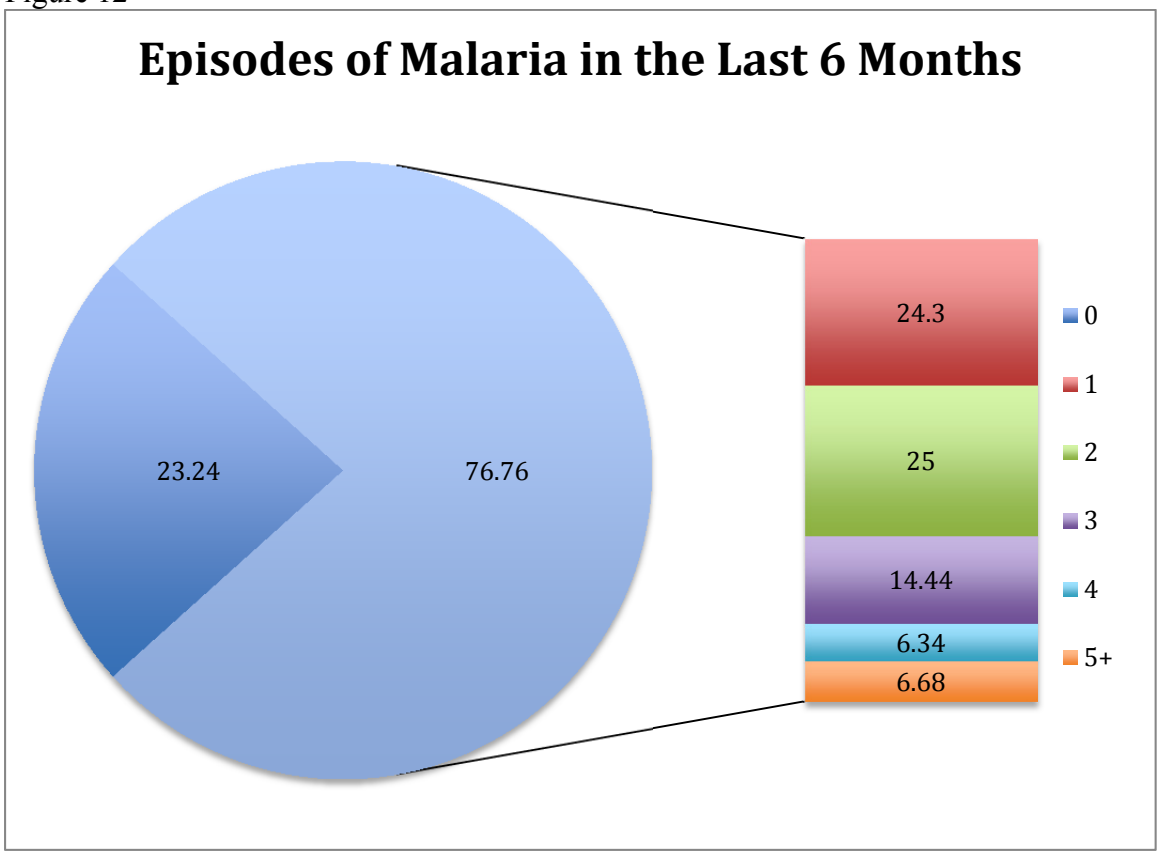
| Age (yr) | 0 to 4.9 | | | 5 to 9.9 | | | 10 and older | | |
|---|---|---|---|---|---|---|---|---|---|
| Blood Lead | n | Mean | SD | n | Mean | SD | n | Mean | SD |
| 0-3.4 | 16 | 66.95 | 34.91 | 26 | 52.77 | 25.06 | 41 | 35.72 | 28.49 |
| >=3.4 TO 5 | 23 | 51.81 | 29.66 | 31 | 37.66 | 24.68 | 52 | 28.97 | 24.52 |
| 5≤x | 43 | 49.87 | 34.02 | 26 | 47.37 | 27.47 | 14 | 29.81 | 25.43 |

Figure 11




Within the sample, 75% (n=218) reported that they had at least 1 episode of malaria in the last 6 months. This 75% group was further split up where, 24.3% the sample had 1 episode of malaria, 25.0% of the sample had 2 episodes of malaria, and 27.46% had three or more episodes of malaria in the last 6 months. See Figure 12.

Figure 12




As seen in Table 2 and Figure 13, history of malaria was significantly related to BMI percentile ($F=4.97$ $p=0.0076$). This meant that the greater the episodes of malaria, the lower the BMI percentile. There was a slight increase in BMI percentile within the group that did not have any malaria in the past 6 months in the middle (5 to 9.9 year olds) and older (10 and older) age group. Subjects that had at least 1 episode of malaria within the last 6 months had a slower decrease in BMI percentile from group 1 to group 2 and a steeper decrease from group 2 to group 3.

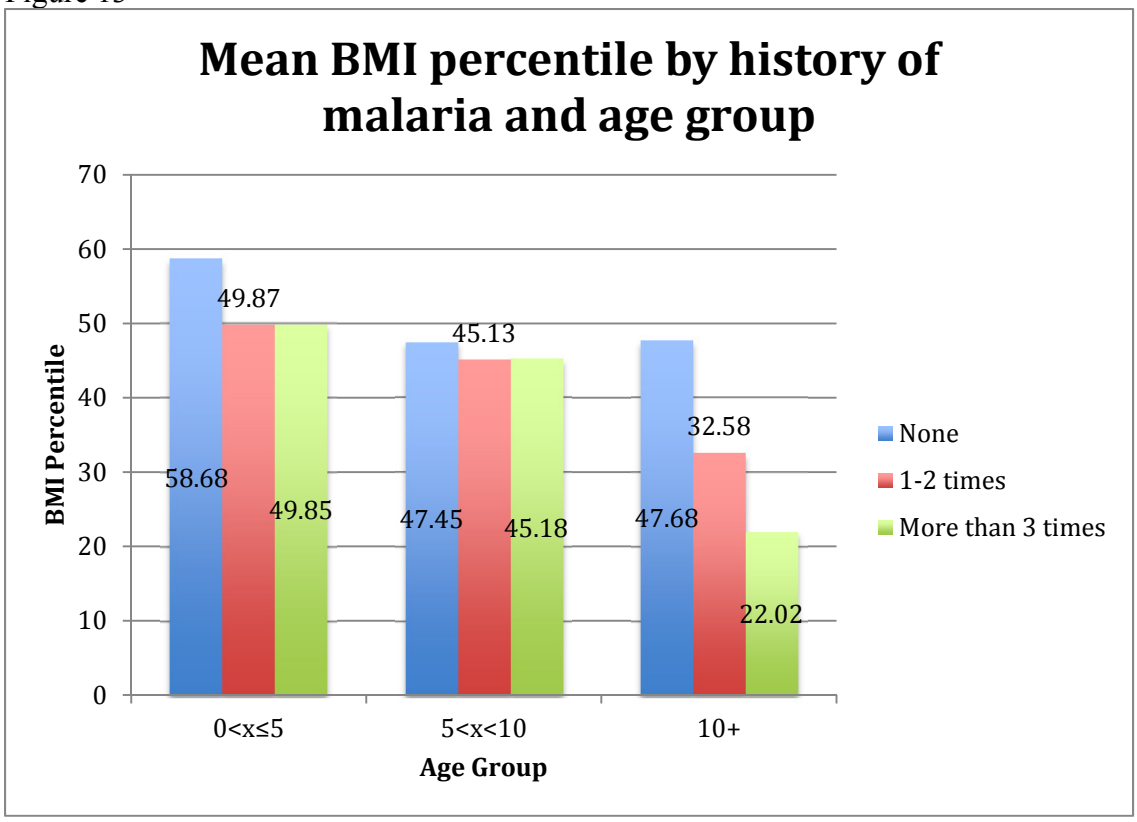
Lead and a history of malaria both negatively influence BMI percentile. Lead shows a lesser decrease in BMI percentile while history of malaria shows a greater decrease in BMI percentile. Both have a greater negative impact with age. However,

blood lead level seems to have a slightly modulating effect compared to the effect of malaria cases.

Table 2: BMI percentile and History of Malaria stratified by Age

| Age (yr) | 0 to 4.9 | | | 5 to 9.9 | | | 10 and older | | |
|---|---|---|---|---|---|---|---|---|---|
| History of Malaria | n | Mean | SD | n | Mean | SD | n | Mean | SD |
| None | 19 | 58.68 | 30.27 | 18 | 47.45 | 27.53 | 18 | 47.68 | 28.54 |
| 1 to 2 times | 42 | 49.87 | 34.51 | 41 | 45.13 | 26.17 | 54 | 32.58 | 25.49 |
| More than 3 times | 15 | 49.85 | 32.52 | 23 | 45.18 | 26.57 | 35 | 22.02 | 21.99 |

Figure 13




Lead, age, number of episodes of malaria in the last 6 months, and worms were regressed on BMI percentile. Overall, the model was significant ($F=6.61$, $p<0.0001$,

$r^2$=0.133). The positive relationship between worms and BMI dropped out. This was probably just an artifact of the study. Lead, age, and episodes of malaria explained 13.3% of the variance in BMI. Gender and age are accounted for when using BMI percentile. Therefore, this regression model shows that blood lead levels and episodes of malaria have some significant effect on BMI percentile.

Table 3: Regression Model

| Variable | F= | p= |
|---|---|---|
| Age | 24.86 | <0.0001 |
| Episodes of Malaria | 3.63 | 0.0278 |
| Blood Lead Level | 3.64 | 0.0277 |
| Worms | 0.28 | 0.596 |

To further study the complex relationship between episodes of malaria and blood lead level, a comorbidity variable was created. This variable combined trichotomized lead and trichotomized history of malaria and created a good and bad score. The comorbidity of lead and malaria will be referred to as "lead/mal." Subjects with no lead and no history of malaria were given a score of "0." Subjects with some lead and/or some history of malaria were given a score of "1." Subjects with high blood lead and more than 3 episodes of malaria were given a score of "2." Frequencies of lead/mal are shown in Table 4. Lead/mal was a significant indicator for BMI percentile among girls (p=0.0213 t=2.33) but non-significant among boys (p=0.251 t=1.15).

Table 4: Lead and malaria comorbidity frequency – "Lead/mal"

| leadmal | Frequency | Percent | Cumulative Frequency | Cumulative Percent |
|---|---|---|---|---|
| **0** | 20 | 7.04 | 20 | 7.04 |
| **1** | 241 | 84.86 | 261 | 91.9 |
| **2** | 23 | 8.1 | 284 | 100 |

Table 5: BMI percentile per lead and malaria comorbidity stratified by age

| Age (yr): | **0 to 4.9** | | | **5 to 9.9** | | | **10 and up** | | |
|---|---|---|---|---|---|---|---|---|---|
| | n | Mean BMI Perc | SD | n | Mean BMI Perc | SD | n | Mean BMI Perc | SD |
| Lead/mal = 0 | 5 | 78.4 | 35.24 | 5 | 57.84 | 30.75 | 7 | 43.84 | 28.37 |
| Lead/mal = 1 | 64 | 49.92 | 31.76 | 68 | 44.6 | 25.72 | 95 | 31.55 | 26.14 |
| Lead/mal = 2 | 7 | 52.89 | 38.64 | 9 | 46.81 | 29.47 | 5 | 16.9 | 19.02 |
| | p=0.1769 F=1.77 | | | p=0.5540 F=0.60 | | | p=0.2128 F=1.57 | | |

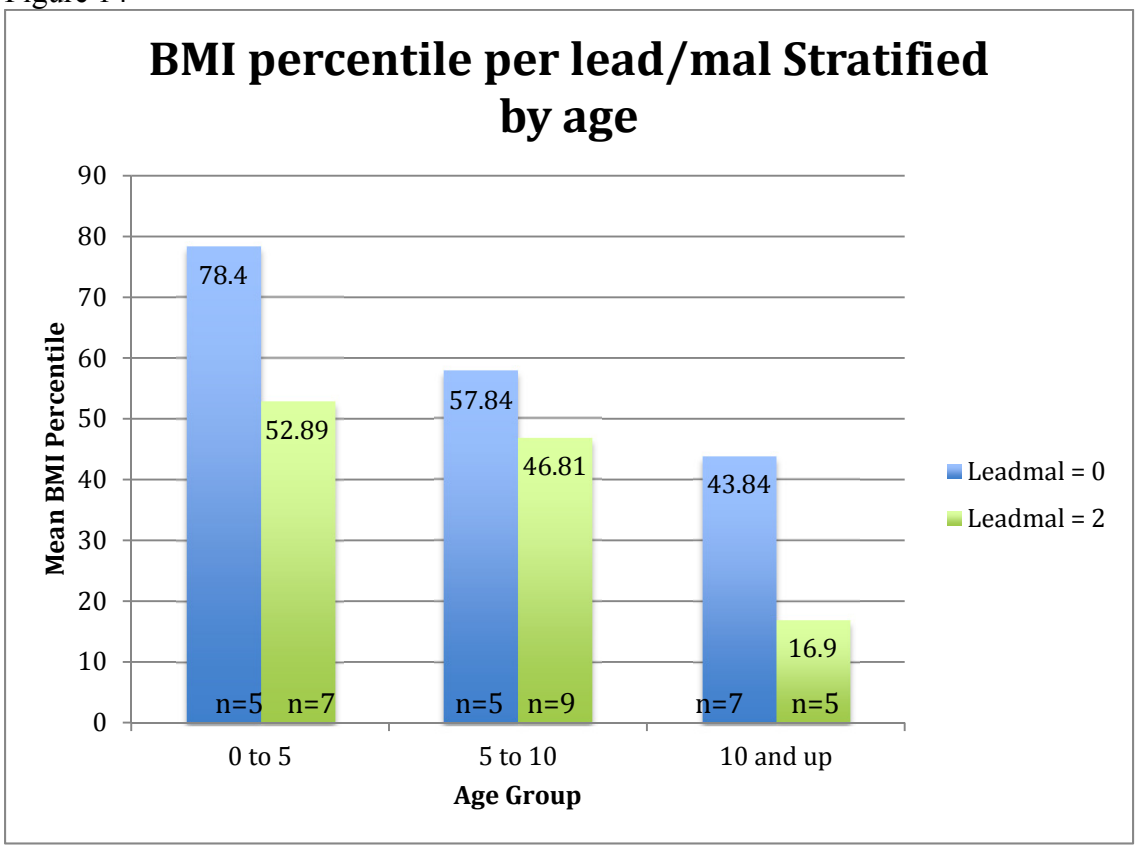
Trends are non-significant (see Table 5) but patterns are consistent (see Figure 14 and Figure 15). When organized by lead/mal, all age groups within each lead/mal designation show a decreasing BMI percentile trend. Of the subjects that were in lead/mal group 0, the 1-month to 5-year-old kids that had neither lead nor cases of malaria began with the highest BMI percentile (78.4$^{th}$ percentile). There was a 32.5% decrease in BMI percentile in children that had both high blood lead levels and high episodes of malaria in the youngest age group.

Of the subjects in the lead/mal = 1 group, those that had either lead and or some cases of malaria began with a BMI percentile of 49.92 and had an average difference of -18.37 between the time they were 0-5 years old to older than 10 years old. There was a 19.07% decrease in BMI percentile in children that had both high blood lead levels and high episodes of malaria in the middle age group.

Subjects that were in the lead/mal=2 group, began with a BMI percentile of 52.89 and had an average difference of -35.99 between the time they were 0-5 years old to older than 10 years old. There was a 61.45% decrease in BMI percentile in children that had both high blood lead levels and high episodes of malaria in the oldest age group.

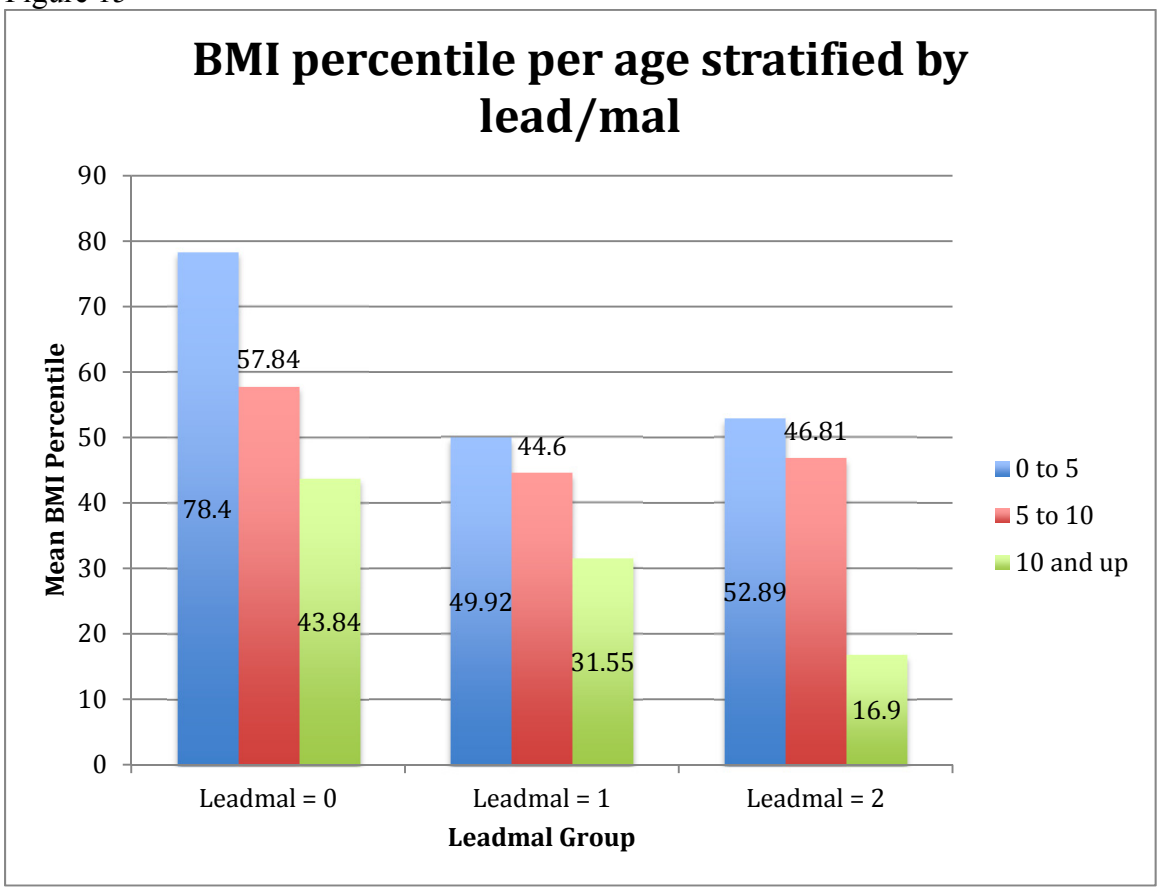
However, since this is a cross-sectional study, we can only hypothesize longitudinal trends based on our sample.

Figure 14




Another way to visualize the effects of lead and malaria on BMI percentile is to stratify by category (Figure 15). Within all lead/mal groups, BMI percentile decreases over time for all three age groups.

Figure 15


BMI percentile per age stratified by lead/mal
Mean BMI Percentile
90
80
70
60
50
40
30
20
10
0
78.4
57.84
43.84
49.92
44.6
31.55
52.89
46.81
16.9
Leadmal = 0
Leadmal = 1
Leadmal = 2
Leadmal Group
0 to 5
5 to 10
10 and up

# CHAPTER SIX

## Discussion

### *Research Question #1*

The first research question this study tried to answer was: What is the prevalence of abnormal blood lead levels in Luo children in rural western Kenya? The hypothesis was that there would be a similar proportion of abnormal blood lead levels in Luo children living in rural Kenya than that found in other rural communities in Sub-Saharan Africa.

The high prevalence of lead poisoning in children is astounding. The mean blood lead level of our sample was 3.601 μg/dL with a range of <3.3 to 35.7 μg/dL and a standard deviation of 3.166 μg/dL. The mean and standard deviation display a negative curtosis, i.e. the distribution is skewed to the left. In the sample of 292 children in rural western Kenya, 70% had an abnormal blood lead level (at least 3.4 μg/dL). More concerning, however, was the fact that 31.17% (n=91) of our sample had at least 5 μg/dL, and 1.03% (n=3) of our sample had greater than 10 μg/dL. Our mean blood lead level of 3.601 μg/dL is closest to the mean blood lead level of 3.8 μg/dL, which was seen in a study by Nriagu and colleagues in 1997 in rural South Africa. In their rural sample, 3% had blood lead levels greater than 10 μg/dL, which was 3 times higher than our results (Nriagu 1997).

The proportion of children with blood lead levels greater than 10 μg/dL is less than proportions found in studies done in Sub-Saharan Africa (Table A). In this rural

setting, the prevalence of blood lead level above 10 μg/dL was about 1%. Diouf and colleague (Table A) reported that the prevalence of blood lead level above 10 μg/dL was 16% in rural Senegal. These proportions were significantly lower than those found in urban settings, which averaged around 50% of the sample having above 10 μg/dL. However, these studies did not provide proportions of blood lead levels above 5 μg/dL so there the reference level blood lead level cannot be compared. The effects of sub-clinical blood lead levels may be exacerbated by the problem of poverty, poor nutrition, lack of access to treatment, or possible increase in exposure pathways.

The children that were between the ages of 1 and 2 years had the highest mean blood lead level (mean=5.94, median=5.25, n=29). This indicates that exposure is early in a child's life, earlier than the age where hand-to-mouth behaviors are expected. This points to maternal influence in a child's lead levels. Children between the ages of 11 and 12, on the other hand, had the lowest mean blood lead level (mean=2.39, median=3.5, n=23). The significant blood lead level difference between the 1-2 year olds and 11-12 year olds indicate that there are exposure sources for the younger children that differ from the sources for the older children. Mean blood lead level decreased across all age groups until they began to increase again starting at age 12 (Figure 2).

These mean blood lead levels cannot be translated to the history of blood lead in a child's life because this is a cross-sectional study. However, the means suggest a pattern of what blood lead may be doing over time in children in this setting. The 30-day half-life of lead in blood would not explain the measured blood lead levels across all ages. Because there is measurable blood lead levels across all age groups, it can be deduced

that there is renewed, repeated exposure to lead. Exposure decreases but is still present in later years.

*Research Question #2*

The second research question this study aimed to answer was: How do blood lead levels affect physical development among Luo children? It was hypothesized that children with higher blood lead levels have a lower BMI than children with lower blood lead levels after controlling for age, gender, helminthic infection, and cases of malaria.

Age and number of episodes of malaria in the last 6 months were significantly and inversely related to BMI. Worms had a significant and direct relationship with BMI. Lead and gender did not have a significant relationship with BMI. However, when all of these variables were regressed on BMI percentile, which is standardized for both age and gender, only episodes of malaria ($p=0.0278$), and blood lead level ($p=0.0277$) had a significant relationship with BMI percentile.

The results suggest that there was a complex relationship between lead and malaria that had an impact on BMI percentile. The combined effects are shown in Figure 14. The BMI percentile of children with normal lead levels and no malaria cases, displayed a weathering effect over time. The youngest children (0 to 5 years old) had the highest mean BMI percentile (78.4) if they did not have blood lead levels and if they did not have any episodes of malaria. Children (10-14 years old) had the lowest mean BMI percentile (16.9) if they had a blood lead level of at least 5 μg/dL and at least 3 episodes of malaria. The BMI percentile of children with high blood lead levels and at least 3 episodes of malaria also displayed the weathering effect of time. Most striking was the

result that, within each age group, the presence of high blood lead levels and multiple episodes of malaria caused BMI percentile to decrease by 19-61%.

On average, having higher blood lead levels and multiple episodes of malaria caused a decrease in BMI percentile of children within that age group. Children in other rural, tropical climate settings may experience a similar trend in BMI percentile if they exhibit similar blood lead levels and historical episodes of malaria. Not only is a child's BMI negatively impacted by poor nutrition, but high lead levels may have a noticeable impact on BMI as well. High lead levels not only impact BMI but may also have neurological and systemic effects that this study did not measure.

### *Limitations and potential sources of error*

The average for each age group was used as a representation of the estimated blood lead level trends throughout a child's growth. However, the cross-sectional design of this study limited the conclusions that can be drawn from blood lead levels over time. Furthermore, because of the short half-life of lead in blood, it is not as accurate a measure of total body lead burden.

This clinical sample offers potential sampling bias in that it represents only a subset of the community on the Nyakach Plateau. Because blood was drawn only from children who visited the clinic, the sample was limited to only those that were aware of the clinic, those that were well enough to travel the clinic, and those that were willing to wait in line to see the physicians. Blood lead level was analyzed in child patients under the age of 14 years from whom blood could be drawn. Outcomes may be more pronounced and improved if a larger sample size was attained.

Additionally, it is possible that blood samples for the lead test may have been contaminated, which could have been controlled for had each blood sample been run multiple times. Expensive tests and limited resources restricted the sample size and did not allow for retests. Accuracy might have been improved if each blood sample could have been tested for lead 3 times and the average had been analyzed. The blood lead machine was calibrated every morning, but this is also a possible source of error.

One of the biggest sources of error concerned the anthropometric measurements, especially in younger children. There may have been inconsistencies particularly in measuring height accurately. If younger children were bundled up in multiple layers of clothes and blankets, height may have been overestimated. If a baby was not totally stretched out while being measured lying down, then height was underestimated, and BMI was overestimated. If height was overestimated, BMI was underestimated. Similarly, weight measurements may not have been accurate if volunteers rounded to convenient numbers, or if mothers were weighed with their babies and then mother's weight was subtracted. If weight was underestimated, BMI was underestimated, and if weight was overestimated, BMI was overestimated.

The variable "episodes of malaria" was a self-reported variable. Children or parents of younger children may have self-diagnosed past episodes of malaria based on symptoms and signs that were not diagnosed by a licensed physician or community health worker. The diagnostic tools that the physicians used to diagnose helminthic infections were specific but not sensitive.

# CHAPTER SEVEN

## Conclusion

This study provides prevalence of lead poisoning in a rural western Kenya. The goal of this study was to see how lead impacted a child's developmental growth. The trend of decrease in BMI as lead level increased was consistent with the literature. This study, however, helps contribute to the literature by providing prevalence of a rural community in Kenya. It also contributes to the study of comorbidity of lead poisoning and repeated episodes of malaria and its effect on BMI.

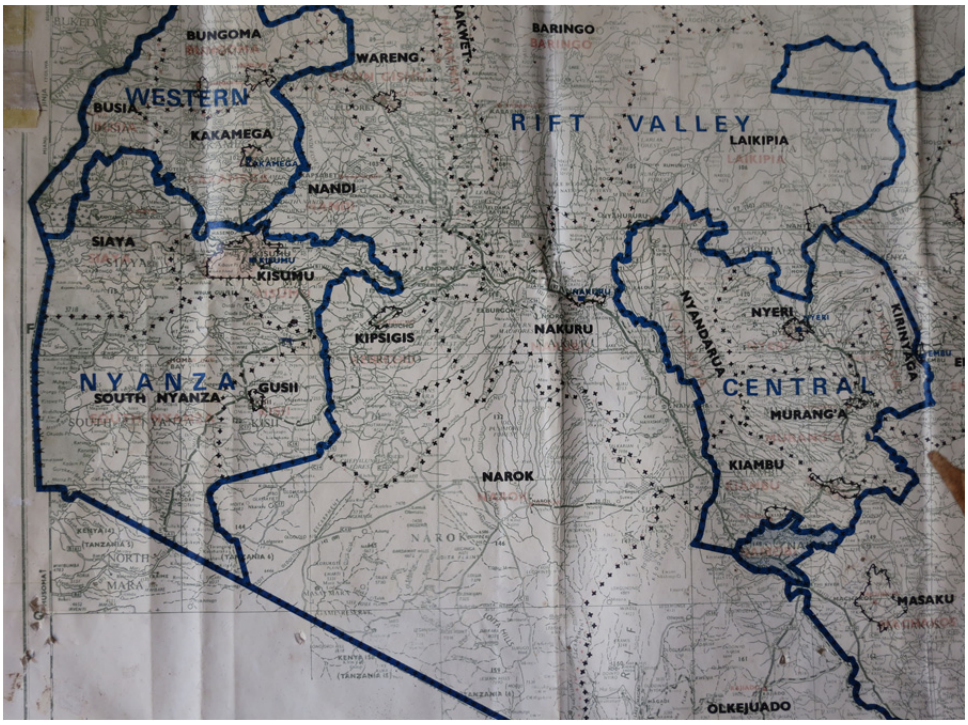
Current interventions include educational seminars that explain the dangers of lead poisoning, especially in young children, and possible ways to block potential exposure pathways. Without knowing the exact source, however, concrete preventive measures cannot be taken.

Future programs and projects should be aimed towards finding the sources of lead so that a more effective preventive intervention can be implemented. For example, if the lead is from the water, filtration methods should be researched. Or if the source is from the dirt or soil, effort should be made to wash hands in clean water or decrease hand to mouth behaviors in children. The findings of this study provide greater understanding of the prevalence of lead poisoning in the community on the Nyakach Plateau. Future studies, programs, and interventions may be implemented by considering exposure pathways for this community.

# APPENDICES

APPENDIX A

Nyanza Province and Nyakach District




Appendix B

## COMMUNITY HEALTH ASSESSMENT PROFILE (CHAP)—General Questions

1-Date: _____(mon/dy/year) 2-Interviewer: _____ 3-Patient ID: _____

4-Setting: __ Straw to Bread Clinic(1) __ Permanent plateau clinic(2) __ At home of participant(3) __Elsewhere in community(4)

5-Age: _____yrs (proportion of year if <1 yr, e.g. 6 mon = .5 yr)

6-Gender: __M(0) __F(1) 7-Bethlehem Home: __Y(1) __N(0)

### Medical History (switch spacing for HIV and others

Malaria
8-Number of times since Jan 2012:
_____ times
9-Num of time since May 2011: _____
10-Take medicine to treat it?
____ N/A(99)
____ Yes(1)
____ No(2)

11-Time since last episode:
_____N/A(99)
_____ wks

12-Had immunizations?
_____ None(0)
_____ Polio(1)
_____ Tetanus(2)
_____Diph/Pertussis/Tetanus(3)
_____ Hep B(4)
_____ HIB(5)
Other: _____

13-Completed all required immunizations:
_____Yes(1) _____No(2)

14-Does patient have HIV?
____Yes(1) ___No(2)
____Don't know(99)

15-When diagnosed? ____N/A(99)
___yrs (proportion of year if <1 yr)

16-Treatment now?
___yes(1) ____No(0) ___N/A(99)

17-How long? ____N/A(99)
___yrs (proportion of year if <1 yr)

19-Last time treated for worms : ___months ago ___not treated(99)

18-Ever unable to get treatment?
___Yes(1) ___No(0) ___N/A(99)

20-Taking meds currently for this problem? (mark with 'yes' or 'no' for each category) Yes = 1 No=0

20a_____ HTN 20d_____ Skin 20g_____ Indigestion, vomiting, constipation
20b_____ Diabetes 20e_____ Malaria 20h_____ Breathing problems, cough, congestion
20c_____ Pain 20f_____ Vitamins

Instructions: If yes, put a number in the box: Put 1 if only one person has it, OR e.g. put 2 if 2 brothers have the disease

| | 21-asthma | 22-diabetes | 23-HTN | 24-sickle cell | 25-stroke | 26-heart attack | 27-HIV | 28-Employed | 29-Died within the last year | 30-Drinks alcohol at least once a month | 31-Smoker |
|---|---|---|---|---|---|---|---|---|---|---|---|
| a. Self | | | | | | | | | | | |
| b.Mother | | | | | | | | | | | |
| c.Father | | | | | | | | | | | |
| d.Bro(s) | | | | | | | | | | | |
| e.Sister(s) | | | | | | | | | | | |
| f.Spouse | | | | | | | | | | | |
| g.child(ren) | | | | | | | | | | | |

### Social History

32-Number living in pt's household: _____ 33-How many people in the household smoke? _____

33-Number of each category living in household:
a.___ pt's child or child-in-law
b.___ pt's spouse
c.___ pt's grandchild
d.___ non-family member
e.___pt's grandparent or spouse's GP
f.___pt's parent or spouse's parents
g.___pt's sibling or in-law

Patient ID:

## Social History (cont.)

34-How many wives does the man have who is the head of the household ? _____
35-How many wives live in the household? _____

36-Is the pt married:
_____ Yes(1)
_____ Widowed(2)
_____ Divorced(3)
_____ Never married(4)
_____ No, too young(5)
37-Number of times married _____
38-Yrs married to current or last spouse _____
39-Number of pregnancies
_____N/A(99)
_____#
40-How many living children does the pt have? _____
41-What is the age of the pt's oldest living child?
_____ yrs (proportion of year if <1 yr)

42-If the pt has a paying job what is it? _____
43-How much school completed
_____ still in school(1)
_____ primary(2) _____ trade school(5)
_____ secondary(3) _____too young for school(6)
_____ university(4) _____never went to school(7)

44-Source of main household income
_____paid by someone else for doing a job(1)
_____selling things that the family grows or makes(2)
_____no household income(3)

45-**Main** household income provider?
_____self(1)
_____spouse(2)
_____children(3)
_____parents(4)
_____no household income(5)

## Nutrition

Mark with 'x' when applicable

| | 46-Morning Meal | 47-Mid-day Meal | 48-Evening Meal |
|---|---|---|---|
| **a.Protein** (Meat/fish/Poultry/ Dagaa/Omena/ Eggs) | | | |
| **b.Other Protein** (beans) | | | |
| **c.Milk** | | | |
| **d.Green Vegetable/Tomatoes** | | | |
| **e.Fruit** | | | |
| **f.Starch** (bread/porridge, ugali, maize, rice, chapati, pots, sweet pots, cassava, beans) | | | |
| **g.Added Sugar** | | | |
| **h.Soft Drinks** | | | |

49-Has the pt ever eaten things that were not food, such as mud or dirt?
_____ yes(1) _____ no(0)

50-When was the last time the pt ate something that was not food (e.g. mud)?
____ never(0)
____ in the last week(1)
____ not in the last week, but in the last month(2)
____ not in the last month, but in the last year(3)
____ years ago when you were a child(4)

51-How often does the pt drink alcohol?
_____Never(1)
_____1-2 times a year(1)
_____1-2 times a month(2)
_____1-2 times a week(3)
_____3-6 times a week(4)
_____daily(5)
52-When pt drinks alcohol, how much?
_____1 drink(1)
_____2 drinks(2)
_____>2 drinks(3)

53-Breastfeeding
____N/A -- pt is child or male or never pregnant(99)
_____ No, didn't breastfeed(0)
Shortest time: _____ N/A(99) _____ # days
Longest time: _____ N/A(99) _____ # months

54-Add salt to food?
_____never(0)
_____seldom(1)
_____at least one meal per day occasionally(2)
_____at least one meal per day most of the Time(3)
_____at least one meal every day(3)

55-Use **iodized** salt?
____yes(1) ____no(0) ____do not know(99)

Patient ID:

## Electricity, House, Water

56-Electricity in house used for:
- _____ N/A—no elec(99)
- _____ lights(1)
- _____ TV(2)
- _____ radio(3)
- _____ stove(4)
- _____ refrigerator(5)
- _____ computer(6)

57-Any household member own
- _____Bicycle(1)
- _____motorbike(2)
- _____car/truck(3)

58-Does pt pay someone for transportation?
__never(0) __daily(1)
__weekly(2) __monthly(3)
__few times a year(4)

59What is the one **main** source of drinking water for pt's household?
- _____ river, stream, spring(1) Location: _____
- _____ pond(2) Location: _____
- _____ well(3) Location: _____
- _____ tap(4) Location: _____
- _____ shared tank(5) Location: _____
- _____ tank at your home(6)
- _____ rainwater caught in buckets(7)

60-What kind of toilet facilities do you use when you are at home?
- ____none, go in the bush(0)
- ____community latrine(1)
- ____open pit latrine(2)
- ____closed pit latrine(3)
- ____flush to pit latrine(4)
- ____flush to piped sewer system(5)

61-House questions
a.Roof: __thatch(1) __metal(2) __tile(3)
b.Wall: __mud/dung(1) __brick(2) __metal(3)
c.Floor : __dirt/dung(1) __concrete(2) __tile(3)

d.Cooking fire:
- ____inside open wood fire(1)
- ____outside open wood fire(2)
- ____inside stove(3)
- ____none(0)

e.Where does pt sleep:
- ____dirt floor(1)
- ____concrete floor(2)
- ____bed(3)

f.How many separate bedrooms? ___#

62a-Have a mosquito net? ____Yes(1) ____No(0)
b.Insecticide treated net? __Yes(1) __No(0)
___N/A(99)
c.How long have you had your current net?
___1-3 months(1) ___4-6 months(2)
___> 1 year(3) ___N/A(99)
d.Sleep under a mosquito net?
- ____never(0)
- ____seldom(1)
- ____occasionally(2)
- ____more than half the time(3)
- ____almost always(4)

e.Sleep under a net last night? __Yes(1) __No(0)
f.Net have holes in it? __Yes(1) __No(0) __N/A(99)

63-Windows:
- ___ none(0)
- ___ glass(1)
- ___ curtains, but no glass(2)
- ___ open with no closure(3)

64a-Water tank at pt's home? ___ yes(1) ____ no(0)

b.Straw to Bread tank? ___yes(1) ____no(0) ____N/A(99)
c.Keep it covered? (partially covered is "no")
_____all the time(1) _____ most of the time(2)
_____ less than half the time(3) _____ not covered(0)
d.Do you share your tank water with people outside the household?
___yes(1) ___no(0) ____N/A(99)
e.How many people share the tank water? _____
f.How long does tank have water during the dry season? ____months

g.Water purifier? ___yes(1) ____no(2)
h.Straw to Bread water purifier? ___yes(1) ___no(0) ____N/A(99)

i.Are there mosquitoes on, around, or inside your home water tank?
- _____ N/A, no water tank(99)
- _____ all the time(1)
- _____ most of the time(2)
- _____ less than half the time(3)
- _____ none of the time(0)

65-Farming
a.Does household grow food to eat themselves?
____ Yes(1) ____ No(0) ____ Sometimes(2)
b.Does household grow food to sell?
____ Yes(1) ____ No(0) ____ Sometimes(2)
c.Do you use pesticides on the food grown by household?
____ Yes(1) ____ No(0) ____ Sometimes(2)
d.If so, what petsicides?_____ (list)

e.Do you store pesticides in the house?
____ Yes(1) ____ No(0)
f.Is the pesticide container open or closed?
____ N/A(99) ____open(1) ____closed(2)
g.Do any of the neighbors next to where you live use pesticides on the food they grow?
____ Yes(1) ____ No(0)

66-How many of these animals does pt's household have? (list number of each)

| | | | |
|---|---|---|---|
| a.____cattle | b.sometimes indoors: | ____yes(1) | ____no(0) |
| c.____goats | d.sometimes indoors: | ____yes(1) | ____no(0) |
| e.____sheep | f.sometimes indoors: | ____yes(1) | ____no(0) |
| g.____chickens | h.sometimes indoors: | ____yes(1) | ____no(0) |
| i.____donkeys | j.sometimes indoors: | ____yes(1) | ____no(0) |
| k.____dogs | l.sometimes indoors: | ____yes(1) | ____no(0) |
| m.____cats | n.sometimes indoors: | ____yes(1) | ____no(0) |

Patient ID:

**Miscellaneous**

67-Is the person wearing shoes?
____ Yes(1) ____ No(0)
68-Is person pregnant?
____ Yes(1) ____ No(0) ____ does not know(2) ___N/A(99)

69-How would pt describe their health in general?
____very good(1)
____good(2)
____fair(3)
____poor(4)
____very bad(5)

70-How often does pt go to the local clinic:
____ Never(0)
____ Almost never(1)
____ a few times a yr(2)
____ a few times a month(3)
____ a few times a week(4)
____ almost every day(5)
71-Purpose of going to local clinic:
____ prenatal care, deliver baby(1)
____ family planning/birth control(2)
____ HIV testing(3)
____ HIV treatment(4)
____ sick(5)
____ child's immunizations(6)
____ take sick family member(7)
____ N/A never go(99)

72-Main reason for not going to clinic more often:
____not sick(1)
____money(2)
____family member doesn't want me to(3)
____no transportation, cannot walk there(4)
____too crowded, cannot be seen(5)
____do not think it helps(6)

73-How often does pt leave the plateau to go to…
a.Sondu
____Never(0)
____a few times a yr(1)
____a few times a month(2)
____a few times a week(3)
____almost every day(4)
b.Kisumu and beyond
____Never(0)
____a few times a yr(1)
____a few times a month(2)
____a few times a week(3)
____almost every day(4)
74-How many times has pt been to Nairobi? _____
75-How many times has pt been out of Kenya? _____

76-What are the reasons that pt leaves the plateau? (check all)
a.School, return home at night __Yes(1) ___No(0)
b.Boarding school ___Yes(1) ___ No(0)
c.Shopping/entertainment ___Yes(1) ___No(0)
d.Business ___Yes(1) ___No(0)
e.Job ___Yes(1) ___No(0)

77-Birth control used:
____N/A(99)
____condom(1)
____pill(2)
____shot(3)
78-How often birth control:
____Never(0)
____Almost never(1)
____occasionally (2)
____often(3)
____regularly all the time(4)

79-Circumcised? N/A ____ yes ____ no

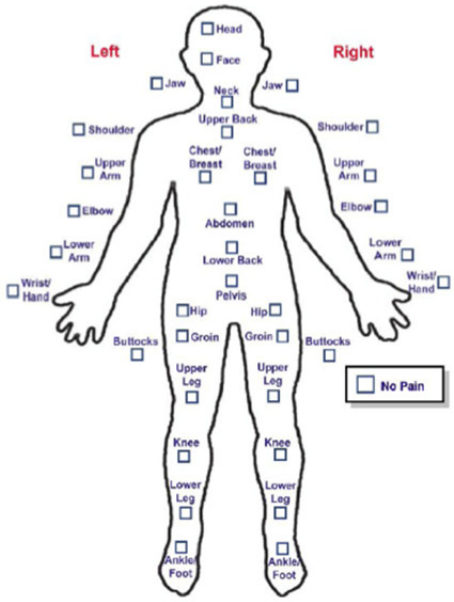
Patient ID:

**Chronic Pain Questions:**

**80-Have you had pain for at least 3 months?**

_____Yes(1) ____No(0)

**81-Indicate on the figure all the locations in which you have been having pain for at least 3 months:**




**82-Have you had any of the following problems for at least the last 3 months?**

Feeling unusually tired _____Yes(1) ____No(0)
Trouble with sleep _____Yes(1) ____No(0)
Anxiety or depression _____Yes 1) ____No(0)
Trouble thinking or remembering _____Yes(1) ____No(0)

**Use the following scale to describe how bad the problem has been over the last 3 months:** **(indicate with an 'x' in the appropriate box)**

| | a. No problem | b. Slight or they come and go | c. Moderate; often present | d. Severe and continuous |
|---|---|---|---|---|
| 83-Pain | | | | |
| 84-Fatigue | | | | |
| 85-Trouble with seep | | | | |
| 86-Anxiety or depression | | | | |
| 87-Trouble thinking or remembering | | | | |

**88-What do you think is the most likely cause of these problems?**

- Do not know(1)
- Frequent hunger(2)
- Working hard physically(3)
- Getting older(4)
- A previous injury(5)
- A long-term illness that you have (such as HIV or asthma)(6)
- Upsetting things going on in your family or communities(7)
- Worries about the future(8)

**89-What do you think is the next likely cause of these problems?**

- Do not know(1)
- Frequent hunger(2)
- Working hard physically(3)
- Getting older(4)
- A previous injury(5)
- A long-term illness that you have (such as HIV or asthma)(6)
- Upsetting things going on in your family or communities(7)
- Worries about the future(8)

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