

## ABSTRACT

### The Effect of Age and Gender on the Prevalence and Severity of Hypertension in Rural Western Kenya

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In order to effectively lessen the drastically increasing morbidity of hypertension in sub-Saharan Africa, new prevalences must be calculated and the causes and aggravating factors of this disease must be more completely understood in this region. The literature repeatedly states how prevalence tends to be underestimated in this area of the world due to lack of blood pressure measurements and the highlighted focus on infectious diseases. Many studies have presented different, solid evidence on potential causes behind hypertension including the common ones of BMI, age, diet, and level of physical activity. The usual sample sizes have focused on older groups. The literature lacks concrete data concerning the nature of high blood pressure in younger groups. This cross-sectional study analyzes previously collected clinical data from a sample population of 685 patients who attended a clinic in May 2010 in rural western Kenya within the Nyando District. Data trends are inferred from blood pressure measurements, anthropometric measurements, blood samples, and the patient's self-report of symptoms. After creating the cut-off age of 18 years, the study's sample size reduced to 321 patients. For those between the ages of 18 and 44, the prevalence of high systolic blood pressure is 19.42%. For those equal to or over the age of 45, the prevalence of high systolic blood pressure is 53.50%. The prevalence of high blood pressure in males is 43.59% and the prevalence in females is 35.48%. BMI is found to be a statistically significant predictor of the severity and presence of high blood pressure only in the young group and the female group. These results show that hypertension is a silent, severe, proliferating problem in the rural area. The pathophysiology behind high blood pressure is typical in the female group, but there is clearly a different physiological process creating hypertension in those who are young and those who are thin.

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THE EFFECT OF AGE AND GENDER ON THE PREVALENCE AND SEVERITY  
OF HYPERTENSION IN RURAL WESTERN KENYA

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## DEDICATION

This study is dedicated to my parents, my brother, and my friends who have never stopped believing in me. This study is also dedicated to those living on the Nyakach Plateau, my wonderful friends in Kenya, and to all those who suffer from non-communicable diseases, especially hypertension

## CHAPTER ONE

### Introduction

#### *Hypertension: The Current World Status*

If this word, ‘hypertension’, is mentioned to anyone in suburban America, the person spoken to would nod and most likely shrug, minimizing it as another common lifestyle problem prevalent in the states. The same reaction would probably be given if the word ‘malaria’ were to come up in a conversation about the current health problems in developing countries. There would be no surprise – just a look of sympathy.

Unfortunately, communicable diseases are not the only pathological processes rising in numbers in developing countries. With the rise of urbanization and an increased interest in health research in the developing countries, a new issue has come into light. There is a new threat on the horizon of developing areas, quickly spreading, and that threat is non-communicable diseases (NCD).

Cardiovascular diseases are spreading rampant throughout Africa. With urbanization going on and different, more sedentary lifestyles being adapted, it does not come as a surprise to see high numbers of cardiovascular diseases in the cities. In most African cities now, a physician will find alarming rates of hypertension in patients. Even

in the rural areas, there have been surprising findings of high proportions of cardiovascular diseases. It is this silent and undetected hypertension that is leading to heart attacks and strokes, and recently, this is the issue making the headlines and dominating current research.

In 2000, almost one billion people in the world were counted as having hypertension. By the year of 2025, this number is expected to increase by 60%<sup>3-5</sup>. Centering in on sub-Saharan Africa, studies have shown that there were 75 million hypertensives in the area in 2008. By 2025, the number will shoot up to 125.5 million hypertensives. Along with this 2008 data, the numbers also show an overall prevalence of 20.7% of the urban population having hypertension and 13.7% of the rural population having hypertension<sup>73</sup>.

Although diseases such as malaria and HIV/AIDS are still very important to consider and study in Africa, scientists, researchers, and physicians should not neglect the potential negative effects non-communicable diseases could have upon the public.

### *The Pathophysiology*

Hypertension is considered a serious problem because of the many complications it could possibly lead to if not diagnosed, monitored, and treated. One of the many serious complications hypertension could lead to is a cerebral hemorrhage or stroke. Untreated hypertension can eventually lead to high blood pressures rupturing a cerebral artery, thus bringing damage to the brain<sup>15, 34</sup>.

There are many different types of hypertension. The most common one is called primary or essential hypertension. Primary hypertension can arise in two ways. If there is excessive vasoconstriction of the small arterioles, there can be high diastolic blood pressure. High systolic blood pressure can come as an effect of high peripheral resistance; to compensate for the resistance, the heart must pump harder. Because of the extra effort the heart has to make and because of the added pressure on the vessels, there are many effects on the heart, blood vessels, and kidneys<sup>15, 34</sup>.

Left ventricular hypertrophy can result from the heart working so hard. The muscles in the heart become enlarged. This does not cause immediate heart problems, but eventually the heart can descend into cardiac failure or congestive heart failure. Many of the symptoms include bloating, irregular pulse, fatigue, and swelling in the feet, ankles, and abdomen. Besides left ventricular hypertrophy, a patient could also develop hypertrophic cardiomyopathy, a similar condition. Another cardiac side-effect would be ischemic heart disease. This is when a heart attack occurs because the heart is not getting enough blood, and thus, oxygen. Symptoms involved include fatigue, irregular pulse, dizziness, and chest pain<sup>15, 34</sup>.

Besides heart problems, essential hypertension can lead to mild to severe vascular effects. A person can develop arteriosclerosis – the high, continuous pressure leads to arteriole injury. The injuries lead to degenerative changes and the lumen becomes narrowed. If the wall has reached a breaking point, it can turn necrotic and even burst<sup>15, 34</sup>.

Lastly, essential hypertension has been known to cause renal effects. One huge problem with hypertension is renal failure. The renal arterioles can narrow, thus restricting blood flow to the kidneys which could lead to the glomeruli and renal tubules dying<sup>15, 34</sup>.

Although primary hypertension is the most common, there are other types of hypertension as well. Conditions that can cause hypertension as a secondary effect include: endocrine gland dysfunction, chronic kidney disease, sleep apnea, coarctation of aorta, and preeclampsia<sup>15, 34</sup>.

Isolated systolic hypertension involves normal diastolic blood pressure while the systolic blood pressure is slightly or highly elevated. This is a hypertension primarily found in older people. With increasing age, the elasticity of the arteries decreases and can no longer absorb the force the ventricles exert. Because there is no excessive vasoconstriction, the diastolic pressure remains normal<sup>15, 34</sup>.

One last type of hypertension is hepatic portal hypertension. One of the main causes of portal hypertension is cirrhosis of the liver which causes scar tissue to block portal veins. With the combination of the vein blockage and high pressure of the blood coming through, there is usually leakage of fluid from the capillaries collecting in the abdominal cavity and causing ascites. To compensate for the vein blockages, the body will automatically create shunts or anastomoses, new pathways for the blood to flow<sup>15, 34</sup>.

Other conditions that have to do with hypertension include preeclampsia and metabolic syndrome. Preeclampsia results from high blood pressure during pregnancy and can lead to seizures. Metabolic syndrome refers to a combination of hypertension,

insulin resistance, and hyperlipidemia. There are currently two standards for metabolic syndrome. Based on the 2001 National Cholesterol Education Program Adult Treatment Panel, some risk factors for this syndrome include abdominal obesity (waist circumference >102 cm in men and >88 cm in women) and a blood pressure of +130/85 mmHg. WHO defines abdominal obesity by three measurements: a waist to hip ratio greater than 0.9, a BMI of at least 30 kg/m<sup>2</sup>, and a waist measurement of over 37 inches. Two other WHO classifications for metabolic syndrome include a cholesterol panel of at least 150mg/dl and a blood pressure of +140/90 mmHg<sup>15,34</sup>.

The following list shows the typical risk factors for hypertension:

- Black
- Family history of hypertension, heart disease, or diabetes
- Over the age of 55
- Overweight
- Not physically active
- Drink excessively
- Smoke
- Diet high in saturated fats or salt
- Illicit drug usage
- NSAIDS or decongestant drugs

The problem with hypertension exists in the fact that it is usually a silent disease. Hypertension does not usually alert the human with some textbook symptoms, but if hypertension is severe or malignant enough, possible symptoms will be produced:

- Pounding in chest, neck, or ears
- Irregular heartbeat
- Blood in urine
- Difficulty breathing
- Chest pain
- Vision problems
- Fatigue or confusion
- Severe headache

#### *Nyakach Plateau Clinic Observations*

In the clinic conducted on the top of the Nyakach Plateau, a physician found many cases of hypertension. The number noticed was alarming considering that the area was a strictly rural, not urban, area like Nairobi where hypertension is starting to run rampant.

It was also noted that there were all sorts of body types coming in with cases of high blood pressure. The situation presented then did not lie solely with the obese and did not follow the typical case of metabolic syndrome. There were many patients with high blood pressure who were young and many who were thin. These alarming findings opened many questions on the factors causing hypertension in the area. It was obvious from just observing that typical hypertension, as similar to that occurring in America, was not all that was happening in this area.

The high blood pressure mystery combined with the fact that most hypertension goes untreated in this area raises red flags and many questions. This is a puzzle waiting to be put together, but the pieces must be found first. Hypertension is a polygenic disease, and the aggravating factors behind it must be discovered. The rise of hypertension in developing countries is turning into a crisis.

Thus, this research investigates the possibility of finding a strange relationship between the young people and hypertension, a relationship not commonly found before in the rural area. This community-based research will hopefully contribute another piece of the puzzle to understanding rural health in Kenya. A better understanding could lead toward more effective interventions among this particular group of people, but it may also add to the general scientific knowledge of this devastating disease.



## CHAPTER TWO

### Review of Literature

#### *The Global Significance*

When people hear underdeveloped or developing nations, many think of infectious diseases as the foremost health problem. However, the truth is that although infectious diseases remain one of the top problems and killers, non-communicable diseases are starting to become an epidemic. Recent statistics show that infectious/parasitic diseases caused 5.5 million deaths in Africa in 2005. In the same year, non-communicable diseases (NCDs) caused over 2.4 million deaths. Non-communicable diseases are gradually climbing to the forefront in developing nations' health problems<sup>52</sup>. It has been predicted that by the year 2020, non-communicable diseases will be the leading cause of mortality and morbidity<sup>61</sup>. Already, the mortality rate from infectious diseases has begun to decline by 3% while the mortality rate from chronic diseases has increased by 17%<sup>1</sup>. More specifically, in 2000, a quarter of the world's adult population had cases of hypertension. By the year of 2025, the rate of hypertension is predicted to increase by 60%<sup>3,4</sup>.

### *The Importance and Significance of Anthropometric Measurements*

Particular anthropometric measurements indicate different things and can be more or less important depending on what disease is involved and what group is being studied. The top two standards of body fat used in the hypertension literature are body mass index (BMI) and waist-to-hip ratio (WHR).

The WHR is an extremely important variable in hypertension. According to Njelekela, with every 0.1 unit increase in WHR, there is a 70% increase in the odds of hypertension. Weight also has a direct correlation. A person can have an increase of 3.0 mmHg in systolic blood pressure for every 10 kilogram increase in weight <sup>9</sup>. A study by Fezeu stated that waist circumference (WC) is a better indicator of cardiovascular risk since it truly measures the adipose tissue than BMI or WHR does <sup>17,22, 23</sup>. This finding was further reinforced by a Nigerian study by Oldapo. Oladapo found that abdominal obesity was a better CVD risk factor to measure than overall obesity. A person was found to have higher risk for cardiovascular disease if a waist circumference was above 88cm in women and above 102cm in men and his or her blood pressure was above 130/85 <sup>58</sup>.

### *The Gender Differences*

Many studies compare men versus women and how the rates of hypertension differ between the two genders. Hypertension in females is more likely to be detected than in males because of women's higher rate of hospital admission due to pregnancy. While in the hospital, virtually all women have their blood pressures checked <sup>2, 35</sup>.

Although there is a greater amount of in-hospital blood pressure data on women, outside studies have found a trend of men having higher rates of hypertension than women. According to a study by de Ramirez, men had an overall higher hypertension prevalence of 24% compared to the women's 20%. Ramirez also found that the gender trends only related to systolic blood pressures (SBP) <sup>61</sup>.

The trend of males having higher hypertension rates than females is confirmed by other studies and further explained. In Njelekela's study, it was found that women had higher obesity rates (35% versus 13%) and a higher prevalence of abdominal obesity (58% versus 11%) compared to men. These findings would seem to point to women having more cardiovascular problems, but instead, Njelekela reinforced the finding of females having a lower rate of hypertension than males. In this particular study, females, compared to men, were found to have a 50% lower odds of having hypertension. Women had the higher waist-to-hip ratio (WHR), BMI, waist circumference, and hip circumference. Despite these being higher than men, women still had lower systolic and diastolic blood pressures with all data being statistically significant. Despite the high BMI and other high anthropometric measurements, women still had lower blood pressures than men due to potential protective factors. One explanation could be that the body fat in women is used in different ways by the body, such as for lactation, than the body fat in men, resulting in extra fat not leaving harmful effects. Because of these protective measures, women may need to reach a higher level of body fat than men require to become hypertensive <sup>19,24, 75</sup>.

One of the main reasons why women have a lower blood pressure than men, however, could be because of the protective effect estrogen has against increasing blood

pressures. Many studies have demonstrated that before puberty, boys and girls have similar values of systolic and diastolic blood pressures. Upon the onset of puberty, boys start to have higher blood pressure values than girls, and only at a later age do women surpass men in having higher blood pressure measurements. Studies like one done by Boschitsch also looked into the role of progesterone and aldosterone in hypertension in women. Although Boschitsch mainly looked at studies of USA, Canada, and Europe, the physiological process found can most likely be applied across all women of all ethnicities. Usually, angiotensin II, involved in the renin-angiotensin-aldosterone system, raises blood pressure. Angiotensin II leads to the release of aldosterone which also causes the cardiovascular system to retain salt and, in so doing, to retain water and increase blood pressure. However, progesterone, a female hormone, has been found to have an anti-aldosterone effect, and thus has the potential to lower blood pressure<sup>10</sup>. Fisher also found a sexual dimorphism in essential hypertension. As other studies have shown, only upon a later age of around 50, during the time of menopause, do women start to show higher blood pressures than men. This is due to the decrease at that time of the beneficial and protective effect of estrogen. Before menopause and after puberty, women have been shown to have lower peripheral resistance than men, and thus lower blood pressures. The physiology behind this involves the menstrual cycle and fluctuating hormones. In women, during their peak estrogen phases of the menstrual cycle (the follicular stage), there happens to be a complementary release of nitric oxide, a vasodilator. Fisher also noted that men have a higher concentration of endothelin which is a vasoconstrictor<sup>24</sup>. Furthermore, Hallberg studied gender specific associations with the vasoconstrictor endothelin – a contributing vascular factor to hypertension. Hallberg found that in

females, the ovarian hormones can suppress endothelin production. In lab studies, it has been found that testosterone, unlike estrogen, increases endothelin levels in the blood <sup>30</sup>. In another hypertension study, Hayes's article supports all these findings by demonstrating how the trend of women's systolic blood pressure starts from being lower than men's during early adulthood but after the age of 60, women's blood pressure becomes as high or higher than men's. The estrogen protective advantage only works during pre-menopause <sup>31</sup>. A study by Reyes shows the same blood pressure relationships between males and females. Reyes re-emphasizes how estrogen depresses blood pressure by releasing vasoactive substances and testosterone tends to hold onto sodium which increases volume retention, and thus higher blood pressure <sup>63</sup>.

Although most studies have displayed the relationship of men having higher rates of hypertension than women, there have been some contradictory studies suggesting that women have higher rates of hypertension than men. One of these studies is Muraguri's study among teenage secondary school students in Nairobi, Kenya. In the study, it was found that girls from age 13-15 had higher systolic blood pressure than boys, but the findings were not statistically significant. The second half of the study focusing on boys aged 15-18 years had statistically significant higher systolic blood pressures than girls. In the end, this follows the previously studied trends of pre-pubertal girls having equal or higher rates of hypertension than boys, and girls having reached menarche having lower blood pressures than boys. The same study has found that there are positive relationships between weight, height, and BMI with systolic blood pressure for boys, but for girls, positive relationships only exist between systolic blood pressure and weight and BMI, not height <sup>55</sup>.

### *The Age Group Differences*

It is not an unknown fact that with older age comes a higher chance of developing non-communicable diseases such as hypertension. The same trend occurs in developing nations, and most research studying non-communicable diseases or cardiovascular risks notes this trend<sup>15, 61</sup>. According to Ramirez's study, unlike how gender was only related to SBP, age was found to be correlated with systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial blood pressure (MABP)<sup>15</sup>. In one study, Fezeu showed that the group with the most chance of developing hypertension is the group age 55-74 years<sup>17,22</sup>.

There are a great number of studies in the literature examining the relationship of aging to high blood pressure among adults, but there are few studies looking into the relationship between high blood pressure and age among children or adolescents. The study done in the Ashanti region of Ghana, West Africa is one of the few studies looking into blood pressure patterns among children. The researchers found similar urban versus rural trends that have been repeatedly noticed in adults: blood pressure measurements are usually higher in urban than rural boys. For girls in this study, however, there was no difference in blood pressure noted between the rural, semi-urban, and urban areas. While this is an interesting find, females have also been noted to have a more fluctuating relationship between age and blood pressure due to puberty and the onset of hormones as explained in the above section of hypertension and gender<sup>4</sup>. Other studies done by

Amusa in the Tshannda Longitudinal Study showed the positive relationship between blood pressure, BMI and body fat for children. In this study of school children, they found that boys and girls had similar diastolic blood pressure and systolic blood pressure values until after grade level 5. Afterwards, the girls showed higher measurements of blood pressure than the boys. This is an interesting finding and seems to contradict the findings made by Fisher who showed the protective effect of estrogen against high blood pressures. It must be taken into account, however that this study focused on overweight children which could negate the protective effect of estrogen <sup>7, 24</sup>.

Another important cardiovascular study done on children was by Fourie who looked into how increased cardiovascular reactivity in children could lead to later development of cardiovascular disease. The pathophysiologic explanation given is that “a sympathetic overreaction such as elevated B-adrenergic activation pattern in young people over a long term leads to changes in the morphologic structure of blood vessels and to chronic elevated blood pressure” <sup>26</sup>.

### *The Problems in Africa: Potential Causes of Hypertension and other Non-Communicable Diseases*

#### *(1) Rural to Urban*

Non-communicable diseases are becoming a problem in developing countries presumably due to increasing urbanization. Western lifestyles are being adopted, which

has precipitated a shift in physical activity and diets <sup>56</sup>. The urbanization leads to sedentary lifestyles, obesity, unhealthy habits and diets. There are reported rates of non-communicable diseases increasing all over the world. In Tanzania, there have been reports of increasing hypertension rates in both urban and rural areas. Furthermore, an increase in the incidence of diabetes mellitus (DM) has also been reported in urban locations. Most notably, DM is becoming an increasing problem in Uganda and South Africa where the prevalence rates are between 8% and 13%. Hyperlipidemia is also becoming common with women and those over the age of thirty-five <sup>57</sup>.

Before urbanization, research reports in Kenya showed that there was a low incidence of hypertension in rural areas <sup>35</sup>. Urbanization and its effects, however, are becoming a major topic of study because of the proliferation of urban living – 40% of Africans currently live in urban areas, and by 2030, half of the Africans will live in urban areas <sup>47</sup>. Thus non-communicable diseases have been appearing in both urban and rural areas of developing countries. Yet they are more pronounced in urban areas, particularly hypertension. According to Agyemang, the age-adjusted systolic and diastolic blood pressure levels were revealed as lower in rural men and women than urban men and women <sup>3-5, 47</sup>. A study by Fezeu found that with increasing waist circumference, there was a greater increase in diastolic blood pressure in urban areas compared to rural areas. Urban and rural areas of developing countries generally consist of people with the same genetic make-up. What could contribute to the urban area's higher rates of cardiovascular risk factors are the environmental differences – these include diet and physical activity levels <sup>2, 19, 22, 24</sup>. In urban areas, the jobs people have are less physical than the duties required by rural life. The foods people eat in those areas are more “dense” than what is



consumed in rural areas. A combination of all these factors usually leads to the people in urban areas being more overweight or obese than those in the rural area. Kenya actually has one of the worst rates of obesity in SSA. In the study by Ziraba, an interesting trend was found. Instead of an increase in obesity in the wealthier women, it was noted that obesity increased in women of lower socio-economic class. This striking finding emphasizes an important lesson: although higher rates of non-communicable diseases are currently being found in the urban areas more than the rural areas, with time, the rates of non-communicable diseases will proliferate in the non-wealthy and non-urban areas <sup>79</sup>.

Most articles in the literature have thoroughly documented the increasing trend of non-communicable diseases like hypertension in urban areas of developing nations. What is lacking in the literature is an explanation of the rise of hypertension in non-urban settings. A most recent statistic notes 2.3% to 41.1% prevalence of hypertension in SSA <sup>17</sup>. A better understanding is especially urgent because studies have noted that cardiovascular diseases occur earlier among sub-Saharan African populations than any other populations <sup>51-53</sup>.

Comparing the urban and rural populations, Agyemang reiterated the common knowledge of the direct relationship between age and blood pressure. Despite the environment or social circumstances a person lives in, whether it be urban or rural, both systolic and diastolic blood pressure generally increase with age <sup>3-5</sup>.

## *(2) Genetics*

Hypertension has been defined in genetic terms as a polygenic disease, one that results from the environment and the interaction with several genes. The variety of factors playing into hypertension is the reason why hypertension in one person can be vastly different from the hypertension in another person. Because 40% of high blood pressure can be explained genetically, there have been myriad recent studies focusing on the genes causing high blood pressure <sup>14</sup>. Going further into the breakdown, 20-60% of hypertension can be explained by genetics while 0-16% can be explained by dietary factors <sup>64</sup>. According to Crook, one of the strongest linkages to hypertension could arise from the angiotensinogen gene <sup>14</sup>. Another study by Ehret looked at 200,000 individuals of European descent and found 16 important loci related to the regulation of blood pressure. Ehret also set forth “strong genetic risk scores” for systolic blood pressure in individuals of African background <sup>17</sup>.

Although many studies have been done on the genetics behind hypertension, there have been even more studies done on the environmental factors behind hypertension. In Africa especially, many studies have been focused on seeing repeated patterns of increasing hypertension in urban areas as compared to rural areas. Consequently, the conclusion has been that environmental circumstances are more closely linked to hypertension than to genetics <sup>9,11</sup>.

A study by Seedat has found that there are potential differences in the pathogenesis behind hypertension in whites and blacks. Upon further investigation into the blacks of Sub-Saharan Africa (SSA), it has been noted that there are significant

biochemical differences in their bodies versus other races. Blacks seem to have lower levels of low density lipoprotein (LDL) and higher levels of high density lipoprotein (HDL) <sup>78</sup>. HDLs are considered the “good cholesterol” while LDLs are considered to be the “bad cholesterol”. Furthermore, blacks are more “salt-sensitive”, while whites are more “salt-resistant”. Salt sensitivity refers to the physiological reaction of “renal vascular resistance” and “glomerular pressure” during high sodium intake. Because of these factors, black patients with hypertension are also sometimes subject to renal failure <sup>78</sup>. To make this case stronger, Tiffin’s study looked into the genes involved in salt sensitivity in indigenous South Africans and found that the salt sensitivity leading to hypertension is more prevalent in people of African origin <sup>73</sup>.

### *(3) Enzymes and Inflammation*

Some studies have found connections between certain enzymes and high blood pressure. According to Brewster’s study, there could be a potential linkage between increased levels of creatine kinase and higher chances of developing hypertension. Creatine kinase is present in vascular and cardiac muscle tissue. Its function is to help the cell under high stress or conditions of high demands by increasing salt retention in the renal tubules <sup>11</sup>.

In another article, Kuklinska showed that hypertension patients had higher CRP (C-reactive protein) concentrations. CRP is a marker of inflammation, and recently it has been used as a marker for cardiovascular activity <sup>38</sup>. Studies have shown that perhaps CRP is a measure of arterial stiffness. Recent studies have shown that chronic arterial

wall inflammation leads to endothelial dysfunction and thus, a higher chance of developing hypertension. Along with CRP, HTN patients also have high levels of BNP, interleukin-6, and tumor necrosis factors <sup>38</sup>.

#### *(4) Malnutrition and Low Birth Weight*

Recently, there has been a fair amount of literature investigating the relationship between birth size and weight and later incidence of hypertension. Omolola addressed the potential mechanism behind this relationship. It could be that low birth weight is a result of poor maternal nutrition and maternal iron deficiency anemia, and thus the baby is born with reduced vascular elasticity, which could lead to later on adult hypertension. In Omolola's study, low birth weight and later life hypertension was a result of parasitaemia (malaria) during pregnancy and not parasitaemia at delivery. Thus, it has been demonstrated that a low birth weight could lead to later cases of hypertension in life <sup>8</sup>. Woelk also investigated this issue and found an inverse relationship in England between systolic and diastolic blood pressures and birth weight. Although Woelk found these relationships in England, the study notes that this relationship can be applied to Africa due to the country's major problem of undernutrition <sup>77</sup>. Furthermore, stunting, a potential result of malnutrition, has been associated with high blood pressure. One study investigated the cardiovascular changes in stunted children. It found that vascular compliance (needed to maintain lower blood pressure) was lower in stunted children. With stunting being one of the most common nutritional problems in Africa, this is a huge concern since these early physiological changes can lead to later non-communicable

diseases. Although this study's findings reinforced previous studies, it found in the end, that after adjusting for BMI and heart rate, the systolic blood pressure and diastolic blood pressure were no different between stunted and non-stunted children. The lack of difference could be because as a child, the complications of reduced arterial compliance have yet to take its toll on the body <sup>64</sup>.

#### *(5) Secondary Hypertension*

It has been found that hypertension could also lead to chronic kidney disease or chronic kidney disease could lead to high blood pressures. Interestingly, while cardiovascular diseases are increasing at a rapid rate in underdeveloped nations, kidney diseases are also increasing. In Sub-Saharan Africa, hypertension (HTN) has been linked to the development of chronic kidney disease such as chronic glomerulonephritis. Chronic kidney disease has been mainly affecting young people age twenty to fifty years. Usually, this renal disease affects middle aged to elderly patients in developed nations <sup>56</sup>. Kidney function can be measured by assessing glomerular filtration rate (GFR). A crude estimate of GFR is serum creatinine which is a breakdown product of muscle tissue that should be almost totally cleared from the blood by the kidneys. A creatinine level of more than 1 standard deviation (SD) above the mean for normal, healthy adults is considered abnormal, and the progression of kidney disease can be monitored by following serum creatinine levels over time.

#### *Other Contributing Factors to Hypertension*

Diet and social factors are two of the main predictors of high blood pressure. Unfortunately, the realms of diet and/or social factors playing a role with hypertension are very large and complex and will not be investigated thoroughly in this study. It is important, however, to mention their significance.

### *(1) Diet*

Hypertension has been labeled the most common cardiovascular risk on the planet, and it is also the most preventable if humans understood how to manage it. There are many factors that go into the development of hypertension<sup>9</sup>. Diet has a significant impact on a person's likelihood of developing hypertension. With more high-fat foods and meat, a person has a greater chance of developing cardiovascular problems. According to the study by de Ramirez, who looked into hypertension of the rural areas of Malawi, Rwanda, and Tanzania, those who had a high diet of meat and foods with high-fat content had twice the odds of developing hypertension compared to those who lacked a diet of high-fat food and meats. It was further noted that those with a diet mainly of vegetables and fruits had a 54% smaller chance of developing hypertension<sup>17</sup>. Freedman, who studied the relationship between salt and blood pressure, found that a daily salt reduction of 100mmol leads to systolic pressure reduction from 1 to 6 mmHg.

Diet has thus been demonstrated to contribute to a person's likelihood of developing hypertension. Diet, however, by the THUSA BANA study, has been further broken down into specific components. The study's results only indicated that a deficiency of folic acid and biotin are potential dietary risk factors. Rooyen, one of the

study's authors, hypothesized that perhaps the lack of folic acid and biotin could lead to a reduction of arterial compliance, which in turn causes higher blood pressures. Rooyen addressed how magnesium, potassium, and calcium are known for their anti-hypertensive abilities. These three dietary factors were found in limited amounts in hypertensive patients, but yet they were also found in deficiencies in normotensive patients. Rooyen then stated that the causes of hypertension must be a mix of dietary, environmental, and genetic factors <sup>64</sup>.

## *(2) Social Factors*

Social factors also come into play when dealing with non-communicable diseases like hypertension. Effective intervention cannot be done unless the cultural values attached to particular risk factors, such as obesity, are understood. Many women may view their weight as normal while outsiders may view it as an obesity problem. Others do not want to lose any weight <sup>9</sup>. In some societies, obesity or having a great amount of weight is looked upon as a symbol of fortune or high status <sup>14</sup>. Furthermore, along with the positive relationship between blood pressure and age, BMI, and elevated glucose, Maher has shown higher rates of hypertension along those with no education or secondary education. Another study reinforced the inverse relationship between level of socio-economic status and blood pressure. Longo-Mbenza found in their study that children who had higher rates of blood pressure were usually from the lower socio-economic class <sup>43</sup>.

### *The Gap*

As observed in many prevalence studies, non-communicable diseases are on the rise in the developing world. One of these increasing conditions is hypertension. There is a great importance in being able to treat hypertension. The importance comes from many factors. One, although there are many prevalence numbers in the literature, many of them are underestimations. Much of the hypertension in developing countries may have gone undetected or at least unnoticed, and by the time the condition has been recognized by a physician, the patient has already reached an advanced and severe stage of cardiovascular disease or malignant hypertension. With this severe stage comes an increase in heart attack and stroke for the patient as well as a “loss of man hours, diminished work productivity, social burden, and increased health expenditure” for the economy and family <sup>58</sup>.

Studies have suggested a list of potential interventions for alleviating the effect that non-communicable diseases are having on developing countries. Researchers suggest increased physical activity and healthier diet programs and maintenance <sup>9</sup>. These measures can be implemented in an urban area quickly and effectively with the right resources and financial aid, but in a very underdeveloped, remote, rural area, the interventions are almost impossible. Thus, most of the studies that focus on new drug trials being conducted in major hospitals of developing nations’ capitals have little to do with the rural problem.

Because of the difficulties that come with studies in a rural, impoverished area, many studies do not venture into those areas to calculate prevalence rates of non-



communicable diseases, specifically hypertension. Therefore, in developing countries like Kenya, the prevalence rates of hypertension may have been underestimated.

Despite the large number of studies on the various issues related to hypertension, there are still a great number of questions to be explored. Hypertension in rural areas can result from a modifiable or a non-modifiable cause. It is important to narrow the causes down and attempt to identify the primary cause behind hypertension in a particular community. If the cause is modifiable (diet/physical activity/malnutrition), then the problem can be and should be addressed. If the cause is non-modifiable (genetics), then the problem will be harder to fix and will continue to plague society, but it is important, however, to implement treatment programs and interventions.

Furthermore, the literature of hypertension only contains studies reinforcing the patterns of how men are more hypertensive than women and how blood pressure increases with BMI and age. Few studies have addressed where young adults fit the picture of hypertension. This could be due to the fact that research and health policymakers generally have focused on the trend of increasing age with alarmingly increasing blood pressure. There is less attention given to atypical non-obese young adults who are hypertensive. This point of view is the wrong way to approach the problem of hypertension.

If the young people are walking around with untreated hypertension, then they become ticking time bombs. It is important, then, to study the risk factors and hidden hypertension in young adults. Only by doing so could there be a chance of preventing

further cardiovascular complications during old age. The goal should be to find and to eliminate the problem while people are young and the bodies are still actively changing.

Thus, what the literature lacks are studies looking into how hypertension affects rural villages in developing countries, and more specifically, how hypertension affects younger people in rural areas. The literature needs a study to carefully describe the rising phenomenon of hypertension in rural areas. This is essential in order to reduce the proliferating rates of hypertension.

## CHAPTER THREE

### Hypothesis

With the general objective of investigating how age and gender affect the prevalence of hypertension (i.e. high blood pressure) in rural western Kenya, this study proposes to test, via a cross-sectional study, on a sample size of 321 patients from the “Straw to Bread” 2010 clinic, these hypotheses:

#### *Research Questions*

Research Question One: “How does the age (i.e. two categories of young and old) of patients affect the relationship between blood pressure and BMI?”

Hypothesis 1A: Among old patients, there is a positive relationship between BMI and blood pressure.

Hypothesis 1B: Among young patients, there is no relationship between BMI and blood pressure.

Research Question Two: “Is the relationship between blood pressure and BMI stronger in men or women?”

Hypothesis 2A: Controlling for age, the positive relationship between BMI and blood pressure is stronger in male than in female patients.

Research Question Three: “Among old and young patients, will body fat distribution (i.e. truncal vs. peripheral fat) have a positive relationship with blood pressure?”

Hypothesis 3A: Among both old male and female patients, there is a positive relationship between truncal fat and blood pressure.

Hypothesis 3B: Among both young male and female patients, there is no relationship between truncal fat and blood pressure.

Hypothesis 3C: Among both old and young patients of both genders, there is no relationship between peripheral fat and blood pressure.

*Schematic Representation*

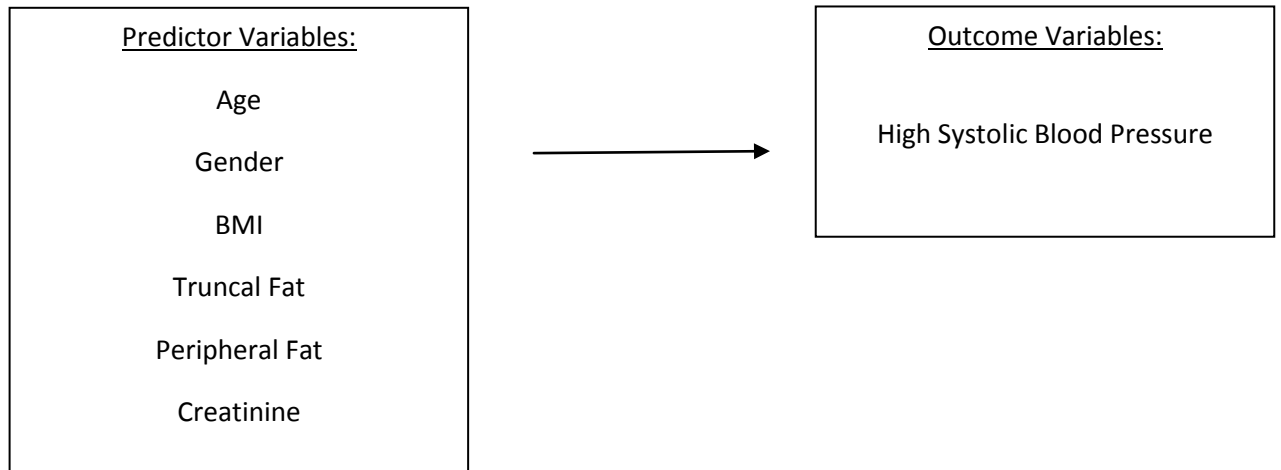


Figure III.1

*In-Depth Schematic Representation*

For Older Patients

in Correlation with Blood

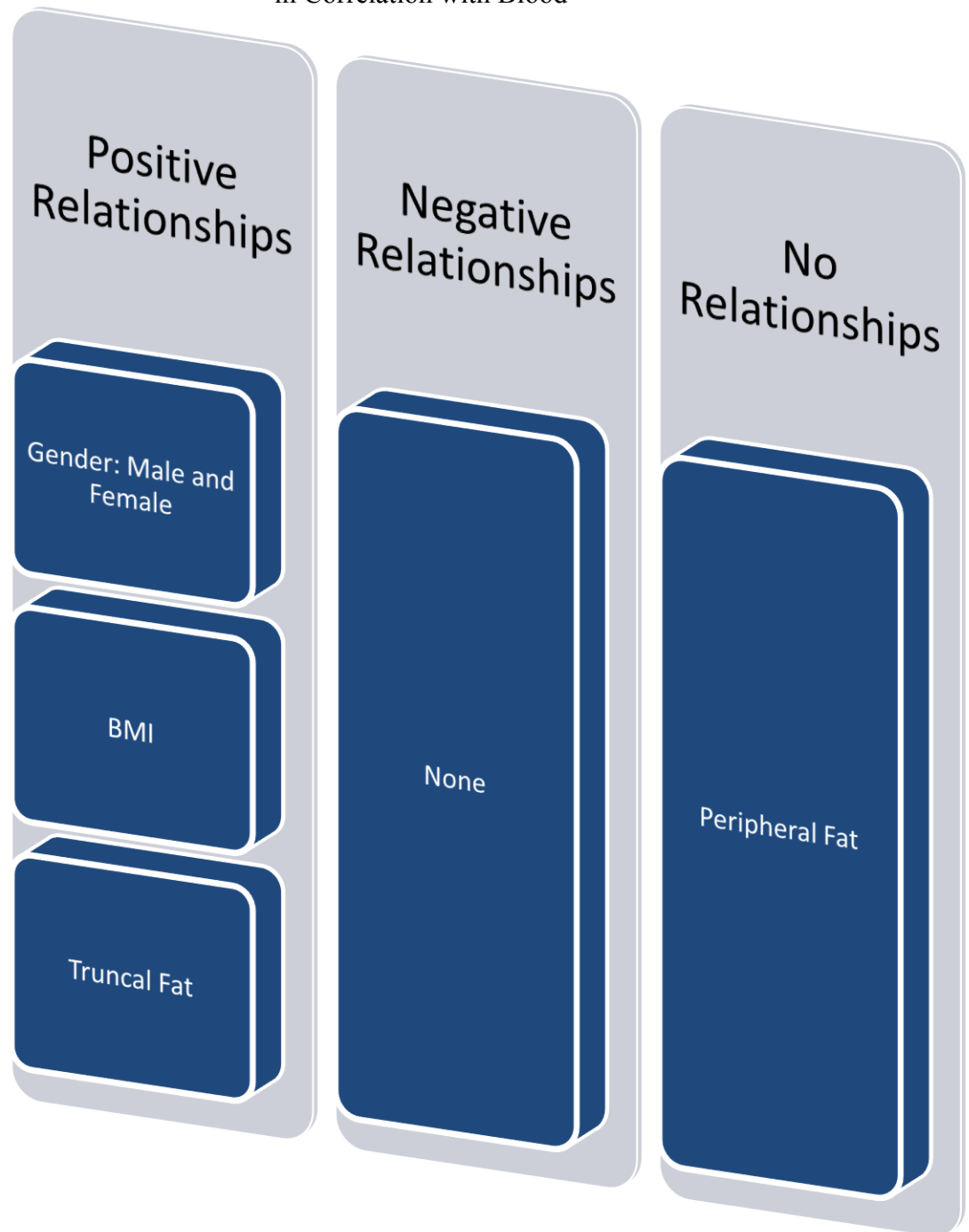


Figure III.2

*In-Depth Schematic Representation*

For Younger Patients  
in Correlation with Blood

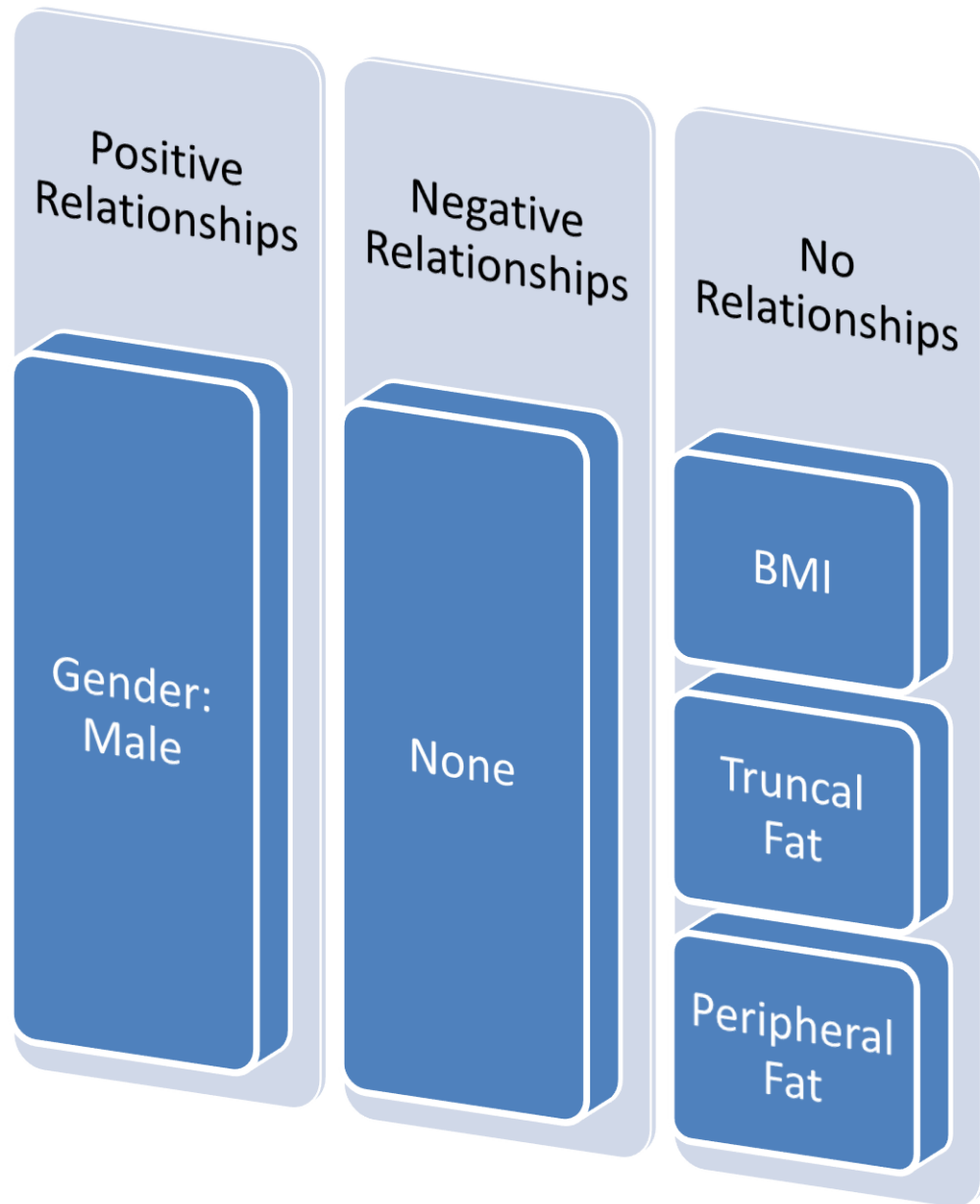


Figure III.3

## CHAPTER FOUR

### Materials and Methods

#### *Study Area and Population*

An analysis of cross-sectional clinical data was performed on previously collected data from the May 2010 Bethlehem Home clinic located in the rural area on the Upper Nyakach Division within the Nyando District in western Kenya. This location is approximately 36 km southeast of Kisumu, Kenya and 12 km southeast of Lake Victoria.

Each year since 2009, a temporary clinic has been set up by American medical professionals and students as part of a long-term collaborative medical/public health project. Patients are seen at no cost, and the doctors see 750 – 850 patients during the annual clinic. The data analyzed in this study came from this group of patients.

The area's local population mostly consists of people from the Luo ethnic group. Those who went to the clinic were mostly individuals who lived within walking distance. The medical clinic saw approximately 685 patients. This analysis was restricted to these 321 patients, who were 18 years of age and older. This age cut-off reflects the CDC 2005



- 2008 standard of looking at hypertension in America (CDC) <sup>60</sup>. The age of those included ranged from 18 years to 100 years.

### *Measurements*

All measurements were taken before the patient was seen by the physician. Weight in kilograms was measured with a spring scale purchased locally in Kisumu, Kenya. Height in centimeters was measured by using a long strip of measuring tape duct-taped to a wall and having patients stand with their backs against a wall. Tricep skin-fold measurements in millimeters were measured using Lange calipers (Beta Technology Inc.). Lange calipers measure subcutaneous tissue by using its pivoted tips and rounded corners to pinch the skin external to the muscle. The calipers can measure up to 60 mm of subcutaneous fat. Waist and hip circumferences were measured by a measuring tape in centimeters.

Creatinine values were measured by gathering a single venous blood sample from each patient and then using the i-STAT handheld device, manufactured by Abbott (Princeton, New Jersey). Controls were performed daily on all machines. The Lange calipers and i-STAT were all used appropriately by trained technicians and volunteers.

Blood pressure was measured using a sphygmomanometer with a standard adult cuff. Most measurements were taken on the right arm, but a second measurement was sometimes taken on the left arm or again on the right arm. Second measurements were taken into consideration if the first measurement seemed inaccurate (too high or too low).

With two measurements of blood pressures for certain patients, only the lowest blood pressure was considered for this study. Highest blood pressures were not considered due to possible random instrumental errors. While taking manual measurements via the sphygmomanometer, the cuff was inflated to a pressure higher than the systolic blood pressure. Then the cuff was deflated and upon the first ‘whooshing’ sound, the systolic blood pressure was recorded. Once the noise disappeared, the diastolic blood pressure was recorded. Blood pressures were only taken on adolescents and adults.

### *Study Design and Clinical Data*

This cross-sectional study used previously gathered clinical data from May 2010. All physical examinations and assessments were performed by Dr. Lisa Baker, a physician trained and board certified in the United States of America. Anthropometric measurements and lab data were taken by trained technicians and volunteers.

High blood pressure was defined as a blood pressure of 140/90 mmHg or above. Only systolic blood pressure was analyzed in this study.

Patients were stratified into categories of young and old, according to the CDC 2005 - 2008 categorization<sup>60</sup>. The young group consisted of patients between the ages of 18 and 44 years. The older group consisted of patients above the age of 44 years.

Body Mass Index (BMI) was calculated according to the formula: mass (kg)/(height<sub>m</sub>)<sup>2</sup>. The WHO uses six categories of BMI to focus on underweight individuals: severe thinness, moderate thinness, mild thinness, normal, pre-obese, and obese. This is named BMI WHO Severe Classification (Table IV.1).<sup>2</sup>

<b>BMI WHO Severe Classification</b>	
<b>Classification</b>	<b>Principal cut-off points (kg/m<sup>2</sup>)</b>
Severe thinness	<16.00
Moderate thinness	16.00 – 16.99
Mild thinness	17.00 – 18.49
Normal	18.50 – 24.99
Pre-Obese	25.00 – 29.99
Obese	≥30.00

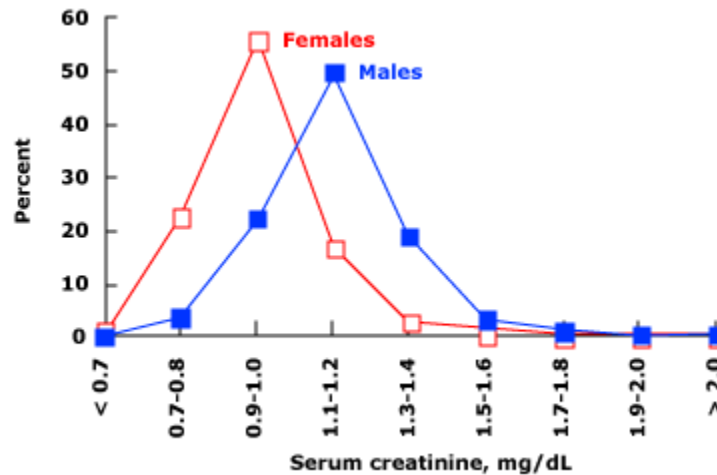
Table IV.1 <sup>28</sup>

Waist-to-hip ratio (WHR) was calculated according to the formula: waist circumference<sub>cm</sub>/hip circumference<sub>cm</sub>. The ratios were then categorized into quintiles (Table IV.2)

<b>WHR Quintile Cut-Offs</b>	
<b>Category</b>	<b>WHR Range</b>
Category 1	≤ 0.8172
Category 2	0.8173 - 0.8498
Category 3	0.8499 - 0.8777
Category 4	0.8778 - 0.9228
Category 5	≥ 0.9229

Table IV.2

Creatinine values were compared to American men and women from 1988 to 1994 (Jones 1998) (Graph IV.1).



Graph IV.1 <sup>16</sup>

### *Summary of Variables*

The variables used in the analysis were the following:

- Study ID
- Gender
- Age
- Systolic Blood Pressure
- Weight (kg)
- Height (cm)
- Waist Circumference (cm)
- Hip Circumference (cm)

- Tricep skinfolds (mm)
- Creatinine.

The variables created from the above initial variables were the following:

- High systolic blood pressure (vs. normal or low)
- BMI WHO 6 categories
- BMI (continuous)
- WHR
- Age Group (young vs. old).

### *Statistical Analysis*

All clinical data were coded and double-entered (blinded) into Microsoft Excel (Redmond, Washington) and then imported into SAS 9.2 (Carey, North Carolina). All variables were checked for errors and corrected when necessary. The statistical analysis was on three levels. The initial step was a descriptive analysis of the major variables crucial to this study: gender, age, systolic blood pressure, weight, height, waist circumference, hip circumference, and tricep skinfold. The bivariate, or, second, level of analysis relied on contingency table analysis, regression analysis, and ANOVA. The third tier of analysis was multivariate in approach; using regression and ANOVA, it was possible to estimate the effects of one predictor variable on an outcome variable while adjusting for the impact of additional variables. Alpha was set at 0.05.

### *Descriptive Statistics*

Descriptive univariate data included averages, medians, standard deviations, and ranges. Average systolic blood pressures, BMIs, tricep skinfold measurements, WHRs, waist circumferences, hip circumferences, and creatinine values are presented for each category within the variables for age groups, gender, and age-gender groups. Average systolic blood pressures, BMsI, tricep skinfold measurements, WHRs, waist circumferences, hip circumferences, and creatinine values were stratified by age and gender.

### *Analytic Statistics*

Regression analysis was performed to adjust for potential confounding factors. Regression analysis was also performed to evaluate the relationship between potential determinants. Blood pressure, as a continuous variable, was regressed on BMI, WHR, tricep skinfolds, truncal measurements (waist circumference and hip circumference), and creatinine. Adjusted  $r^2$  values showed the relationship between the single determinant to the variable of interest: blood pressure. Alpha was set at 0.05, and P values are reported. P values of less than 0.05 were considered to indicate statistical significance.

Analysis of variance (ANOVA) was also performed to evaluate the relationship between potential determinants. Blood pressure, as a dichotomous variable (normal versus high), is evaluated with BMI WHO six categories, age as a dichotomous variable, and WHR quintiles.  $R^2$  values show the relationship between the single determinant to

the variable of interest. Alpha was set at 0.05, and P values were reported. P values of less than 0.05 were considered to indicate statistical significance.

### *IRB*

This study was approved by the Baylor University Institutional Review Board. The data abstracted from the clinical record used no unique personal identifiers. All subjects remained anonymous.

## CHAPTER FIVE

### Results

#### *Organization of Results*

The results of this study are organized into six sections beginning with a general overview. Descriptive results are reported first followed by analytic results. Only systolic blood pressure measurements and analysis were taken into consideration in this analysis. Diastolic blood pressure measurements were not used in this analysis. Finally, when the term high blood pressure or hypertension (HTN) is used, it refers to high systolic blood pressure unless otherwise specified.

#### *General Overview*

A total of 685 patients attended the clinic between May 15 and May 29, 2010. Out of 685 patients, only 321 patients are included in this analysis because of the study's cut-off age of 18 years or older. Out of the study's sample size of 321 patients, the average



patient age was 47.6 years (SD=18.2) with a range of 18 years to 100 years. Patients were grouped according to age into two groups: the young group included patients 18 – 44 years and the old group included patients 45 years or more. There were a total of 111 patients found with high blood pressure. High blood pressure was defined as any blood pressure measurement equal to or greater than 140 mmHg. Out of the young group, 27 patients had high blood pressure and out of the old group, 84 patients had high blood pressure (Table V.1). When patients were grouped by gender, there were 34 males and 77 females with high blood pressure (Table V.2). Thus, in our study's sample, there are more old patients than young patients and more females than males.

Total Patients	685					
Study's Sample Size	321					
Age Group	(18 - 44 years) - 148			(45+ years)- 173		
Blood Pressure	112	27	9	73	84	16

Total High Blood Pressure                      111  
Total Normal Blood Pressure                      185  
Total Missing    25

	Normal Blood Pressure
	High Blood Pressure
	Missing

Table V.1

Total Patients	685			
Study's Sample Size	321			
Gender	Males - 78		Females - 217	
Blood Pressure	44	34	140	77

Total High Blood Pressure                      111  
Total Normal Blood Pressure                      184  
Total Missing    26

	Normal Blood Pressure
	High Blood Pressure
	Missing

Table V.2

The prevalence of elevated blood pressure (HTN) stratified by age in the population was higher in the old age group (53.50%) compared to the young age group (19.42%) (Table V.3). When the prevalence of HTN was stratified by gender, there was a higher prevalence found in the males (43.59%) compared to the females (35.48%) (Table V.3). The overall prevalence of high blood pressure in this study's sample size was 34.58% (Table V.3).

	High Systolic Blood Pressure Prevalence
Young	19.42%
Old	53.50%
Males	43.59%
Females	35.48%
Overall	34.58%

Table V.3

All BMI values were categorized into six groups based on WHO's BMI classification scheme: severe thinness, moderate thinness, mild thinness, normal or moderate BMI, pre-obese, and obese (Table IV.2). WHR (Waist to hip ratio) was divided into quintiles (Table IV.3).

Comparing the Nyakach Plateau region of this study to the United States shows the United States to have ten times the prevalence of obesity. The United States has approximately three times the prevalence of persons being pre-obese.

The Kenyan urban geographical area has BMI categorical percentages similar to the Nairobi Province, the capital of Kenya, and thus a very populated, urban area. When compared to the Nyakach Plateau, the Kenyan urban data shows double the pre-obesity rates of the Nyakach Plateau while the Nyakach Plateau presents almost five times the rate of underweight people as the Kenyan urban data.

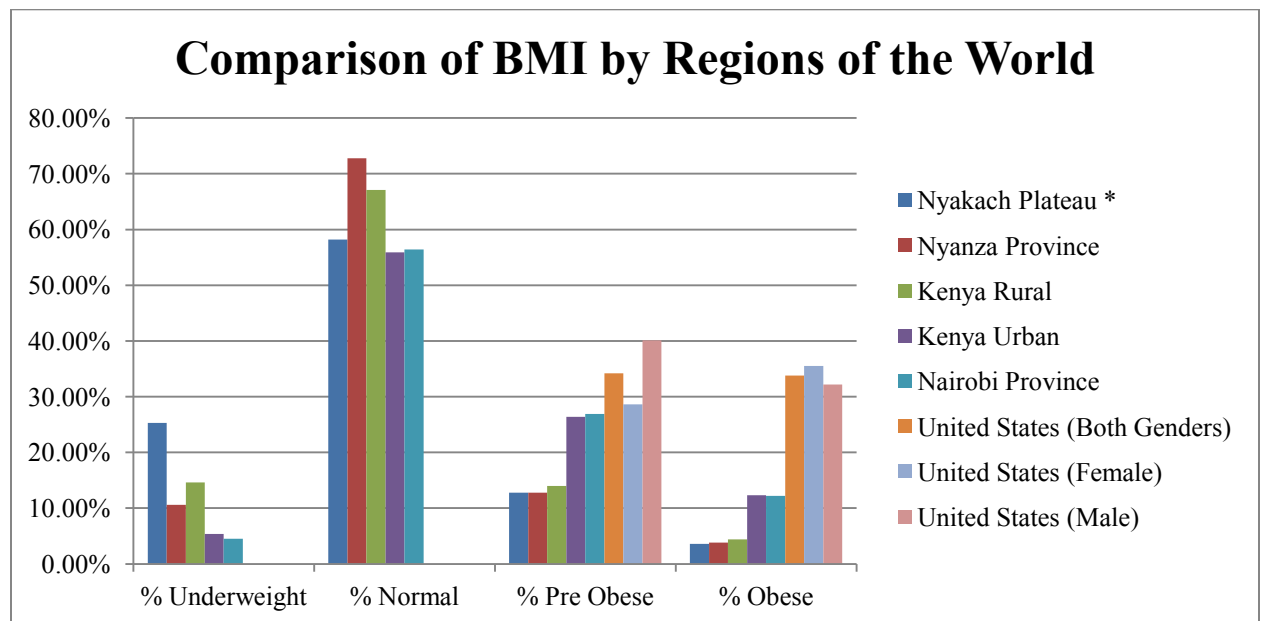
The Nyakach Plateau BMI percentages are relatively comparable to the Nyanza Province and the Kenyan rural areas. The area of this study has the highest prevalence of underweight people (Table V.4).

High Blood Pressure Prevalence: Comparison of Studies								
Geographical Region	Year of Study	Sample Size	Age Criteria	Prevalence by Gender		Prevalence by Age		Overall Prevalence
				Males	Females	Young (18 - 44 years)	Old (45+ years)	
Nyakach Plateau *	2010	321	18+	43.59%	35.48%	19.42%	53.50%	34.58%
Sub-Saharan Africa	2008	300+	15+	16.80%	15.70%			13.70%
Cameroon	2003	1762/1398	24-74	44.00%	34.10%			
United States	2005 - 2008	10,488	18+	30.60%	28.70%	10.50%	40.60% and 70.30%	29.90%

\*Data from this study

Not available

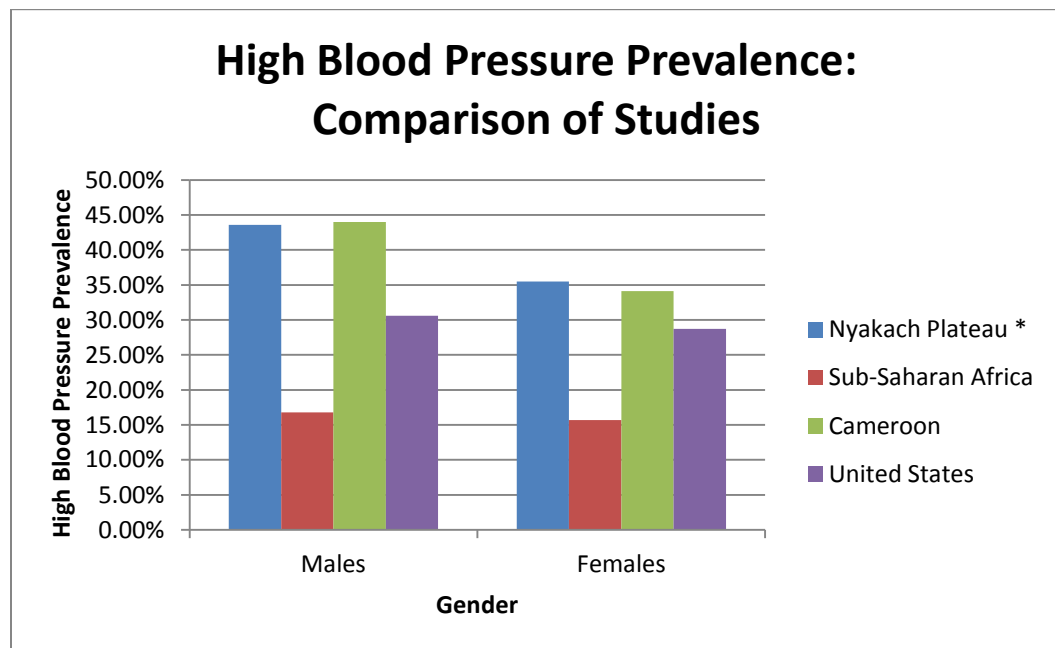
Table V.4<sup>2,22,28</sup>



Graph V.1<sup>28</sup>

When compared to other studies in Sub-Saharan Africa, Cameroon, and the United States, the Nyakach Plateau's high blood pressure prevalence numbers by gender are most similar to Cameroon's estimates. The prevalence of HTN is 40.60% for the age group 45-64 years and 70.30% for the age group 65+ years. When comparing the prevalence stratified by age, the Nyakach Plateau has a higher prevalence of high blood pressure in the young than does the United States. Out of all the regions, the highest

overall prevalence of high blood pressure lies in the region of this study on the Nyakach Plateau (Table V.4).



Graph V.2<sup>2,22,28</sup>

*Overall Data without Stratification*

*Descriptive Statistics*

<b>Averages by Variable Uncategorized</b>		
<b>Variable</b>	<b>N</b>	<b>Mean (SD)</b>
<b>Systolic Blood Pressure (mmHg)</b>	303	137 (21.2)
<b>BMI (kg/m<sup>2</sup>)</b>	301	21.23 (3.9)
<b>Age (years)</b>	321	47.6 (18.2)
<b>WHR</b>	288	0.87 (0.13)

Table V.5

Table V.5 summarizes the average systolic blood pressure, BMI (body mass index), age, and waist to hip ratio (WHR) for the overall study's sample size. The average systolic blood pressure was 137 mmHg (SD=21.2). The average BMI was 21.23 kg/m<sup>2</sup> (SD=3.9) and ranged from 13.98 to 34.84 kg/m<sup>2</sup>. The average BMI was in the normal range according to WHO standards. The range of BMI was between the severe thinness and obese class I categories. The average WHR was 0.87 (SD=0.13).

*Analytic Statistics*

<b>The Impact of Age, BMI, and Gender on Systolic Blood Pressure (mmHg)</b>						
Systolic Blood Pressure Regressed On:						
<b>Variable</b>	<b>Regression Coefficient</b>	<b>S.E.</b>	<b>t</b>	<b>P value</b>	<b>Incremental R<sup>2</sup></b>	<b>Cumulative R<sup>2</sup></b>
<b>Intercept</b>	94.37	7.45	12.67	0.0001		
<b>Age (years)</b>	0.47	0.06	7.37	0.0001	0.1495	0.1495
<b>BMI (kg/m<sup>2</sup>)</b>	0.96	0.29	3.34	0.0010	0.0293	0.1788
<b>Gender*</b>	-2.98	2.69	-1.11	0.2691	0.0005	0.1793
*0=Male, 1=Female						

Table V.6

Regressing systolic blood pressure, as a continuous variable, on the potential determinants created the regression coefficients in Table V.6-8. Out of all the potential determinants, age explained the most variance (14.95%) and was a significant predictor of blood pressure ( $p=0.0001$ ). When systolic blood pressure was regressed on age as a dichotomous variable, the explained variance was less than for age as a continuous variable. Thus, age was a better predictor of systolic blood pressure when analyzed continuously than when broken down into two categories of young and old. BMI was a significant indicator of blood pressure but explained less of the variance than age ( $p=0.0010$ ). When BMI was combined with age, they explained together a total of 17.88% of the variance: when the two variables were combined with gender, the explained variance increased minimally to 17.93%, but gender was not significant ( $p=0.2691$ ) while BMI and age remained significant ( $p=0.0010$  and  $p=0.0001$ ). Thus, gender is not a significant predictor of blood pressure (Table V.6).

<b>The Impact of Hip Circumference on Systolic Blood Pressure (mmHg)</b>						
Systolic Blood Pressure Regressed On:						
<b>Variable</b>	<b>Regression Coefficient</b>	<b>S.E.</b>	<b>t</b>	<b>P value</b>	<b>Incremental R<sup>2</sup></b>	<b>Cumulative R<sup>2</sup></b>
<b>Intercept</b>	130.87	4.65	28.15	0.0001		
<b>Hip Circumference (cm)</b>	0.070	0.05	1.26	0.2072	0.0020	0.0020

Table V.7

<b>The Impact of Hip Circumference (Obese) on Systolic Blood Pressure (mmHg)</b>						
Systolic Blood Pressure Regressed On:						
<b>Variable</b>	<b>Regression Coefficient</b>	<b>S.E.</b>	<b>t</b>	<b>P value</b>	<b>Incremental R<sup>2</sup></b>	<b>Cumulative R<sup>2</sup></b>
<b>Intercept</b>	117.51	23.74	4.95	0.0011		
<b>Hip Circumference (cm) on BMI WHO Severe Category: Obese</b>	0.42	0.24	1.77	0.1145	0.1919	0.1919

Table V.8

Hip circumference explained little of the variance in systolic blood pressure ( $R^2 = 0.0020$ ) ( $p=0.2072$ ) (Table V.7). However, when systolic blood pressure was regressed on hip circumference among those in the obese BMI WHO category, the potential determinant was able to explain 19.19% of the variance in blood pressure ( $p=0.1145$ ). The insignificant p value could be due to the small sample size of 10 patients in the BMI WHO obese category (Table V.8). The potential determinants of waist circumference, WHR, and tricep skinfolds were not significant predictors of blood pressure. When blood pressure was regressed on waist circumference and hip circumference combined, the p value remained insignificant. The same outcome of insignificant p value was demonstrated when BMI and waist circumference were regressed together with systolic blood pressure.

Analysis of Variance								
Variable							P	Total R-Square
AVERAGES Values in red are average systolic blood pressure measurements in mmHg								
BMI Classification by WHO	Severe Thinness	Mod Thinness	Mild Thinness	Normal	Pre-Obese	Obese	0.0266	0.043574
	133	136	138	134	138	158		
	N=9 SD=23.5	N=18 SD=32.0	N=44 SD=20.6	N=169 SD=19.9	N=39 SD=18.6	N=10 SD=25.8		
WHR (All 5 Quintiles)	Category 1	Category 2	Category 3	Category 4	Category 5		0.2348*	0.01951
	132	135	135	138	140			
	N=57 SD=18.1	N=57 SD=22.8	N=58 SD=16.0	N=56 SD=19.1	N=58 SD=27.5			

\* Not statistically significant

Table V.9

The potential impact of BMI on systolic blood pressure was explored using analysis of variance (ANOVA). BMI was analyzed using six WHO groups (Table V.9). The average systolic blood pressure was 158 mmHg for obese persons and 133 mmHg for the severely thin ( $p = 0.0266$ ,  $r^2 = 0.0436$ ). Waist-hip ratio (WHR), in quintiles, was not as good a predictor of systolic blood pressure as BMI (Table V.9).

Systolic blood pressure was regressed on BMI as a continuous variable ( $r^2 = 0.0293$ ,  $p = 0.0010$ ); BMI as a categorical variable explains more variance in systolic blood pressure than BMI as a continuous variable. It is clear than the BMI categories of the WHO have clinical relevance.



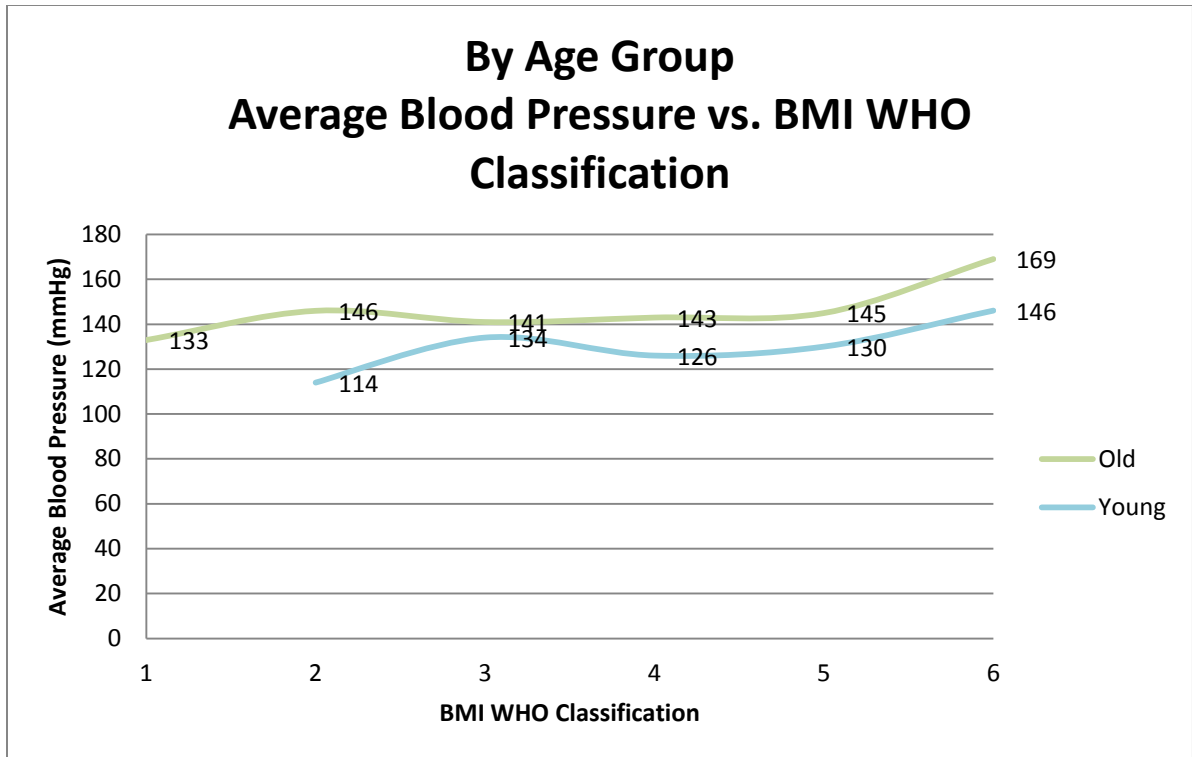
### *Data by Age Groups*

#### *Descriptive Statistics*

<b>Averages by Variable and Category</b>					
<b>Variable</b>	<b>N</b>	<b>Mean (SD)</b>	<b>Median</b>	<b>Minimum</b>	<b>Maximum</b>
<i>Young (18 - 44 years)</i>					
<b>Systolic Blood Pressure (mmHg)</b>	142	128 (15.4)	125	90	190
<b>BMI (kg/m<sup>2</sup>)</b>	141	21.63 (3.7)	20.47	16.29	34.84
<b>Age (years)</b>	148	31 (7.3)	30.5	18	44
<b>WHR</b>	138	0.87 (0.14)	0.86	0.36	1.54
<b>Waist Circumference (cm)</b>	138	69.72 (20)	74.4	26	114.5
<b>Hip Circumference (cm)</b>	138	81.15 (23.1)	88.5	25.7	118.6
<b>Tricep Skinfold (mm)</b>	139	15.3 (8.4)	14	3	41
<i>Old (45+ years)</i>					
<b>Systolic Blood Pressure (mmHg)</b>	161	144 (22.8)	142	95	226
<b>BMI (kg/m<sup>2</sup>)</b>	160	20.88 (4.1)	20.01	13.98	34.77
<b>Age (years)</b>	173	62 (11.5)	60	45	100
<b>WHR</b>	160	0.89 (0.08)	0.88	0.67	1.48
<b>Waist Circumference (cm)</b>	160	74.04 (20.5)	77.6	26	114.5
<b>Hip Circumference (cm)</b>	160	83.9 (22.4)	88.55	27.5	119
<b>Tricep Skinfold (mm)</b>	160	12.3 (9.2)	10	2	47

Table V.10

Table V.10 stratifies the sample by young and old age groups. Descriptively those patients who are older have a higher average systolic blood pressure than those patients who are younger. The highest blood pressure measurement of 226 mmHg was found in the old age group. The average BMI in the younger group was 21.63 kg/m<sup>2</sup> which falls in the BMI WHO normal category, and the average BMI in the older group was 20.88 kg/m<sup>2</sup> which also falls in the BMI WHO normal category. The highest average BMI value was higher in the young than the old patients (Table V.10).



Graph V.3

Graph V.3 shows the relationship stratified by age group between average blood pressures and the six categories of BMI by WHO. The old age group has higher average blood pressures across all six BMI categories than the young age group. Those patients in the old age group have high blood pressure averages, and those patients in the young group have normal to high blood pressure averages (Graph V.3).

<b>The Association of Hypertension and BMI Classification in Younger Patients</b>				
	<b>Systolic Hypertension</b>			<b>Percentage of those with hypertension in each BMI group</b>
	<b>Yes</b>	<b>No</b>	<b>Total</b>	
<b>Severe Thinness</b>	0 0.00	0 0.00	0 0.00	0.00
<b>Moderate Thinness</b>	1 16.67	5 83.33	6 4.32	3.7
<b>Mild Thinness</b>	7 36.84	12 63.16	19 13.67	25.93
<b>Normal</b>	12 13.19	79 86.81	91 65.47	44.44
<b>Pre-Obese</b>	3 16.67	15 83.33	18 12.95	11.11
<b>Obese</b>	4 80.00	1 20.00	5 3.60	14.81
<b>Total</b>	27 19.42	112 80.58	139 100	100
<b><math>\chi^2 = 17.18; p=0.0014</math></b>				

Table V.11

The Association of Hypertension and BMI Classification in Older Patients				
	Systolic Hypertension			Percentage of those with hypertension in each BMI group
	Yes	No	Total	
Severe Thinness	4 44.44	5 55.56	9 5.73	4.76
Moderate Thinness	7 50.00	7 50.00	14 8.92	8.33
Mild Thinness	15 55.56	12 44.44	27 17.20	17.86
Normal	42 51.85	39 48.15	81 51.59	50.00
Pre-Obese	11 52.38	10 47.62	21 13.38	13.10
Obese	5 100.00	0 0.00	5 3.18	5.95
Total	84 53.50	73 46.50	157 100	100
$\chi^2 = 4.86; p=0.4337$				

Table V.12

The association between BMI WHO categories and systolic hypertension (HTN) was explored using contingency table analysis. Among the younger age group (Table V.11), 19.42 % of the sample was hypertensive. Among the obese 80% were hypertensive and among the severely thin, 0.0 % were hypertensive ( $p = 0.0014$ ). Among the older adults (Table V.12) the association between BMI and HTN was not statistically significant; however, all 5 of the obese older adults were hypertensive.

The Impact of BMI on Systolic Blood Pressure (mmHg) Stratified by Age Group						
Systolic Blood Pressure Regressed On:						
Variable	Regression Coefficient	S.E.	t	P value	Incremental R <sup>2</sup>	Cumulative R <sup>2</sup>
Intercept	112.31	7.68	14.63	0.0001		
BMI (kg/m <sup>2</sup> )	0.73	0.35	2.08	0.0390	0.0238	0.0238
Intercept	125.84	9.49	13.25	0.0001		
BMI (kg/m <sup>2</sup> )	0.86	0.45	1.93	0.0552	0.0172	0.0172
						Young
						Old

Table V.13

The Impact of Waist and Hip Circumferences on Systolic Blood Pressure (mmHg) Stratified by Age Group						
Systolic Blood Pressure Regressed On:						
Variable	Regression Coefficient	S.E.	t	P value	Incremental R <sup>2</sup>	Cumulative R <sup>2</sup>
Intercept	119.84	4.90	24.46	0.0001		
Waist Circumference (cm)	0.09	0.07	1.41	0.1613	0.0072	0.0072
Hip Circumference (cm)	0.18	0.12	1.53	0.1275	0.0098	0.0170
Intercept	143.41	7.00	20.51	0.0001		
Waist Circumference (cm)	0.06	0.09	0.71	0.4807	-0.0032	-0.0032
Hip Circumference (cm)	-0.57	0.24	-2.35	0.0201	0.0284	0.0252
						Young
						Old

Table V.14

The Impact of WHR on Systolic Blood Pressure (mmHg) Stratified by Age Group						
Systolic Blood Pressure Regressed On:						
Variable	Regression Coefficient	S.E.	t	P value	Incremental R <sup>2</sup>	Cumulative R <sup>2</sup>
Intercept	139.20	8.28	16.82	0.0001		
WHR	-12.60	9.39	-1.34	0.1819	0.0058	0.0058
Intercept	97.58	19.29	5.06	0.0001		
WHR	52.26	21.70	2.41	0.0172	0.0299	0.0299
						Young
						Old

Table V.15

Regressing systolic blood pressure, as a continuous variable, stratified by age group, on the potential determinants produced the regression coefficients presented in Table V.13-15. BMI was found to explain 2.38% of the variance in blood pressure in the young and 1.72% of variance in the old, and is a significant predictor of blood pressure in both age groups ( $p=0.0390$  and  $p=0.0552$ ) (Table V.13). When waist circumference and hip circumference were analyzed together, they were shown to be significant predictors of blood pressure only in the old ( $p=0.0201$ ) with hip circumference explaining most of the variance (Table V.14). Lastly, WHR only was found to be a significant predictor with an  $R^2$  value of 0.0299 in the old age group ( $p=0.0172$ ) (Table V.15). Tricep skinfold measurements were found not to be statistically significant predictors of blood pressure.

Thus, for the young age group, BMI was the only significant predictor of blood pressure. For the old age group, BMI and WHR were significant predictors of blood pressure.

Analysis of Variance Stratified by Age Group								
Variable		AVERAGES					P	Total R-Square
		Values in red are average systolic blood pressure measurements in mmHg						
BMI Classification by WHO	Severe Thinness --- N=0 SD=---	Mod Thinness 114 N=6 SD=18.2	Mild Thinness 134 N=19 SD=19.8	Normal 126 N=91 SD=13.2	Pre-Obese 130 N=18 SD=14.1	Obese 146 N=5 SD=18.6	0.002	0.117835
	Severe Thinness 133 N=9 SD=23.5	Mod Thinness 146 N=14 SD=29.4	Mild Thinness 142 N=27 SD=22.7	Normal 143 N=81 SD=22.3	Pre-Obese 145 N=21 SD=19.4	Obese 169 N=5 SD=28.5	0.1288*	0.054474

WHR (All 5 Quintiles)	Category 1 127 N=32 SD=15.2	Category 2 129 N=32 SD=16.5	Category 3 132 N=28 SD=15.0	Category 4 128 N=20 SD=15.3	Category 5 124 N=25 SD=15.1		0.3941*	0.030277
	Category 1 139 N=25 SD=19.8	Category 2 142 N=25 SD=27.5	Category 3 137 N=30 SD=16.9	Category 4 144 N=36 SD=18.8	Category 5 153 N=33 SD=28.3		0.0576	0.06113

\* Not statistically significant

Young  
Old

Table V.16

The potential impact of BMI WHO classification and WHR, adjusted by age, on systolic blood pressure were evaluated using ANOVA (Table V.16). Among the younger adults, the average systolic blood pressure was 146 mmHg in the obese and 114 mmHg in the moderately thin (there were no young adults in the severely thin WHO category) ( $r^2 = 0.1178$ ,  $p = 0.002$ ). Similar trends were found among the older adults, but the differences in blood pressure between the BMI groups did not reach statistical significance. WHR was associated with blood pressure among the older adults ( $r^2 = 0.0611$ ,  $p = 0.0576$ ), but not among the younger adults.

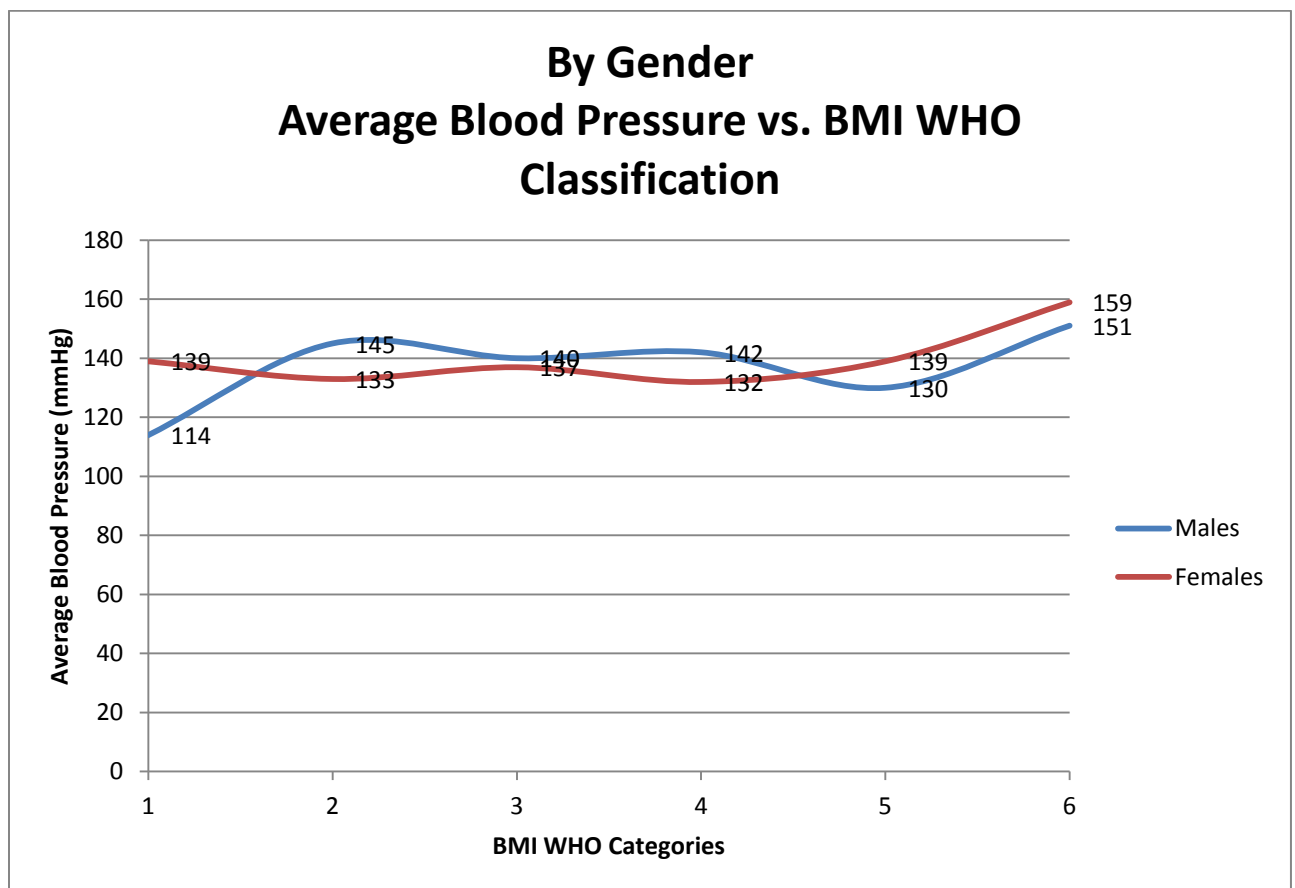
#### *Data by Gender*

##### *Descriptive Statistics*

<b>Averages by Variable and Category</b>					
<b>Variable</b>	<b>N</b>	<b>Mean (SD)</b>	<b>Median</b>	<b>Minimum</b>	<b>Maximum</b>
<i>Males</i>					
<b>Systolic Blood Pressure (mmHg)</b>	79	141 (21)	136	108	226
<b>BMI (kg/m<sup>2</sup>)</b>	82	19.81 (3.0)	18.96	15.51	31.29
<b>Age (years)</b>	87	55 (19)	59	19	86
<b>WHR</b>	80	0.88 (0.2)	0.89	0.008	1.19
<b>Waist Circumference (cm)</b>	81	72.99 (19)	77	28.3	114.5
<b>Hip Circumference (cm)</b>	81	82.75 (20)	87	31	115.8
<b>Tricep Skinfold (mm)</b>	81	8.81 (6.9)	5.8	2	30
<i>Females</i>					
<b>Systolic Blood Pressure (mmHg)</b>	222	135 (21)	130	90	200
<b>BMI (kg/m<sup>2</sup>)</b>	218	21.76 (4.1)	20.70	13.98	34.84
<b>Age (years)</b>	232	45 (17)	43	18	100
<b>WHR</b>	207	0.87 (0.1)	0.86	0.44	1.54
<b>Waist Circumference (cm)</b>	216	71.67 (21)	76.4	26	114.5
<b>Hip Circumference (cm)</b>	216	82.5 (24)	89	25.7	119
<b>Tricep Skinfold (mm)</b>	217	15.4 (8.9)	13	2	47

Table V.17

Table V.17 stratifies by males and females. Descriptively those patients who are males have a higher average systolic blood pressure than those patients who are females. The highest blood pressure measurement of 226 mmHg was found in the males. The average BMI in the males was 19.81 kg/m<sup>2</sup> which falls in the BMI WHO normal category, and the average BMI in the females was 21.76 kg/m<sup>2</sup> which falls in the BMI WHO normal category. Descriptively the highest average BMI value was higher in the females than the males (Table V.17).



Graph V.4

Graph V.4 shows the relationship stratified by gender between average blood pressures and the six categories of BMI by WHO. Descriptively males had higher



average blood pressures in the BMI WHO groups of moderate thinness, mild thinness, and normal. Females, descriptively, had higher average blood pressures in the BMI WHO groups of severe thinness, pre-obese, and obese (Graph V.4).

*Analytic Statistics*

<b>The Association of Hypertension and BMI Classification in Male Patients</b>				
	<b>Systolic Hypertension</b>			<b>Percentage of those with hypertension in each BMI group</b>
	<b>Yes</b>	<b>No</b>	<b>Total</b>	
<b>Severe Thinness</b>	0 0.00	2 100.00	2 2.56	0.00
<b>Moderate Thinness</b>	3 50.00	3 50.00	6 7.69	8.82
<b>Mild Thinness</b>	12 48.00	13 52.00	25 32.05	35.29
<b>Normal</b>	17 41.46	24 58.54	41 52.56	50.00
<b>Pre-Obese</b>	0 0.00	15 83.33	2 2.56	0.00
<b>Obese</b>	2 100.00	0 00.00	2 2.56	5.88
<b>Total</b>	34 43.59	44 56.41	78 100	100
$\chi^2 = 6.05; p=0.3011$				

Table V.18

<b>The Association of Hypertension and BMI Classification in Female Patients</b>				
	<b>Systolic Hypertension</b>			<b>Percentage of those with hypertension in each BMI group</b>
	<b>Yes</b>	<b>No</b>	<b>Total</b>	
<b>Severe Thinness</b>	4 57.14	3 42.86	7 3.23	5.19
<b>Moderate Thinness</b>	5 35.71	9 64.29	14 6.45	6.49
<b>Mild Thinness</b>	10 47.62	11 52.38	21 9.68	12.99
<b>Normal</b>	37 28.46	93 71.54	130 59.91	48.05
<b>Pre-Obese</b>	14 37.84	23 62.16	37 17.05	18.18
<b>Obese</b>	7 87.50	1 12.50	8 3.69	9.09
<b>Total</b>	77 35.48	140 64.52	217 100	
<b><math>\chi^2 = 15.13</math>; <math>p=0.0098</math></b>				

Table V.19

Among males, BMI and HTN were not significantly associated ( $p = 0.3011$ ) (Table V.18). Among females, the relationship between BMI and HTN was statistically significant ( $p=0.0098$ ), but the pattern is difficult to interpret (Table V.19). The highest proportion (87.5%) of HTN was among the obese, but the next highest proportion (57.14) was among the severely thin group. The lowest proportion (28.46) of HTN was among the normal BMI group.

<b>The Impact of BMI and Age on Systolic Blood Pressure (mmHg) Stratified by Gender</b>						
Systolic Blood Pressure Regressed On:						
<b>Variable</b>	<b>Regression Coefficient</b>	<b>S.E.</b>	<b>t</b>	<b>P value</b>	<b>Incremental R<sup>2</sup></b>	<b>Cumulative R<sup>2</sup></b>
<b>Intercept</b>	117.60	18.10	6.50	0.0001		
<b>BMI (kg/m<sup>2</sup>)</b>	0.44	0.81	0.54	0.5894	-0.0093	-0.0093
<b>Age (years)</b>	0.21	0.12	1.69	0.0949	0.0242	0.0149
<b>Intercept</b>	83.42	7.84	10.65	0.0001		
<b>BMI (kg/m<sup>2</sup>)</b>	0.88	0.35	2.53	0.0122	0.0243	0.0243
<b>Age (years)</b>	0.60	0.07	8.11	0.0001	0.2258	0.2501
						Male
						Female

Table V.20

Regressing systolic blood pressure, as a continuous variable, on the potential determinants, stratified by gender, produced the regression coefficients presented in Table V.20. BMI was found to be significant only in the females explaining 2.43% of the variance in blood pressure ( $p=0.0122$ ). Age was found to be significant only in the females explaining 22.58% of the variance in blood pressure ( $p=0.0001$ ). Thus, when systolic blood pressure was regressed on BMI and age combined, the p value was significant only among the females with an  $r^2$  value of 0.2501 ( $p=0.0122$  and  $p=0.0001$ ) (Table V.20). The other potential determinants of systolic blood pressure - waist circumference, hip circumference, WHR, and tricep skinfolds - were found not to be statistically significant predictors (data not shown). The regression analyses did not produce any significant predictors of blood pressure among the males.

Analysis of Variance Stratified by Gender										
Variable		AVERAGES Values in red are average systolic blood pressure measurements in mmHg							P	Cumulative (Total) R-Square
BMI Classification by WHO	Severe Thinness 114 N=2 SD=8.5	Mod Thinness 145 N=6 SD=43.1	Mild Thinness 140 N=25 SD=18.9	Normal 142 N=41 SD=19.1	Pre-Obese 130 N=2 SD=7.1	Obese 150 N=2 SD=7.8			0.4836*	0.059049
	Severe Thinness 139 N=7 SD=23.8	Mod Thinness 133 N=14 SD=23.9	Mild Thinness 137 N=21 SD=22.7	Normal 132 N=132 SD=19.7	Pre-Obese 139 N=37 SD=18.9	Obese 159 N=8 SD=28.8			0.0095	0.069055
BMI Classification by WHO and Age (dichotmous)	Young 134 N=24 SD=17.4	Old 144 N=54 SD=22.2							0.4631*	0.104179
	Young 127 N=114 SD=14.9	Old 144 N=103 SD=23.4							0.0027 <.0001	0.231853

\* Not statistically significant

Males  
Females

Table V.21

An analysis of variance among the males revealed BMI not to be related to systolic blood pressure (Table V.21). Among the females, the relationship was statistically significant ( $p = 0.0095$ ), with BMI explaining 6.9% of the variance in systolic blood pressure; the pivotal difference between the BMI groups was the fact that the obese women had the highest average systolic blood pressure (159 mmHg). When the dichotomous age variable was added to the ANOVA model, the two variables of BMI and age explained 23% of the variance in systolic blood pressure among the women and 10.4% of the variance among the men.

*Data by Age Group and Gender*

*Descriptive Statistics*


<u>Averages by Variable and Category</u>					
Variable	N	Mean (SD)	Median	Minimum	Maximum
<i>Young Males (18-44 years)</i>					
<b>Systolic Blood Pressure (mmHg)</b>	24	134 (17.4)	132	108	190
<b>BMI (kg/m<sup>2</sup>)</b>	25	20.1 (2.8)	19.77	16.29	25.94
<b>Age (years)</b>	26	30 (6.8)	30.5	19	43
<b>WHR</b>	25	0.86 (0.16)	0.87	0.36	1.19
<b>Waist Circumference (cm)</b>	24	67.71 (18.7)	73.5	28.3	87.2
<b>Hip Circumference (cm)</b>	24	80.48 (19.9)	85	33	107.5
<b>Tricep Skinfold (mm)</b>	24	10.35 (8.1)	7	3	30
<i>Old Males (45+ years)</i>					
<b>Systolic Blood Pressure (mmHg)</b>	55	144 (22)	142	108	226
<b>BMI (kg/m<sup>2</sup>)</b>	57	19.68 (3.1)	18.83	15.51	31.29
<b>Age (years)</b>	61	66 (10.7)	65	47	86
<b>WHR</b>	55	0.89 (0.14)	0.90	0.78	1.10
<b>Waist Circumference (cm)</b>	57	75.2 (18.9)	78	29.3	114.5
<b>Hip Circumference (cm)</b>	57	83.7 (20.4)	88	31	115.8
<b>Tricep Skinfold (mm)</b>	57	8.16 (6.2)	5	2	29
<i>Young Females (18-44 years)</i>					
<b>Systolic Blood Pressure (mmHg)</b>	116	127 (14.8)	124	90	175
<b>BMI (kg/m<sup>2</sup>)</b>	115	21.95 (3.8)	20.66	16.49	34.84
<b>Age (years)</b>	120	31 (7.3)	32	18	44
<b>WHR</b>	111	0.87 (0.14)	0.85	0.44	1.54
<b>Waist Circumference (cm)</b>	113	70.1 (20.4)	75	26	114.5
<b>Hip Circumference (cm)</b>	113	81.15 (23.8)	89	25.7	118.6
<b>Tricep Skinfold (mm)</b>	114	16.24 (8)	15	3	41
<i>Old Females (45+ years)</i>					
<b>Systolic Blood Pressure (mmHg)</b>	106	144 (23.3)	141	95	200
<b>BMI (kg/m<sup>2</sup>)</b>	103	21.55 (4.4)	20.77	13.98	34.77
<b>Age (years)</b>	112	59 (11.4)	58	45	100
<b>WHR</b>	96	0.87 (0.09)	0.87	0.67	1.48
<b>Waist Circumference (cm)</b>	103	73.4 (21.4)	76.9	26	113.5
<b>Hip Circumference (cm)</b>	103	84 (23.5)	89	27.5	119
<b>Tricep Skinfold (mm)</b>	103	14.5 (9.7)	12	2	47

Table V.22

Table V.22 stratified by both age groups and gender, creating four groups: young males, old males, young females, and old females. Descriptively the old male patients and the old female patients have the highest average systolic blood pressure (144 mmHg). All average BMI measurements fall in the BMI WHO normal category. The highest average BMI measurement was in the young female group (21.95 kg/m<sup>2</sup>) (Table V.22).

*Analytic Statistics*

The Impact of BMI on Systolic Blood Pressure (mmHg) Stratified by Age-Gender Groups						
Systolic Blood Pressure Regressed On:						
Variable	Regression Coefficient	S.E.	t	P value	Incremental R <sup>2</sup>	Cumulative R <sup>2</sup>
Intercept	138.56	27.29	5.08	0.0001		
BMI (kg/m <sup>2</sup> )	-0.22	1.35	-0.17	0.8701	-0.0442	-0.0442
Intercept	128.71	19.38	6.64	0.0001		
BMI (kg/m <sup>2</sup> )	0.77	0.98	0.79	0.4348	-0.0072	-0.0072
Intercept	104.12	7.85	13.26	0.0001		
BMI (kg/m <sup>2</sup> )	1.03	0.35	2.93	0.0041	0.063	0.063
Intercept	123.56	11.55	10.69	0.0001		
BMI (kg/m <sup>2</sup> )	0.94	0.53	1.79	0.076	0.0212	0.0212



Young Males  
Old Males  
Young Females  
Old Females

Table V.23

The Impact of Waist and Hip Circumferences on Systolic Blood Pressure (mmHg) Stratified by Age-Gender Groups						
Systolic Blood Pressure Regressed On:						
Variable	Regression Coefficient	S.E.	t	P value	Incremental R <sup>2</sup>	Cumulative R <sup>2</sup>
<b>Intercept</b>	117.74	15.35	7.67	0.0001		
<b>Waist Circumference (cm)</b>	0.28	0.19	1.47	0.1560	0.0479	0.0479
<b>Hip Circumference (cm)</b>	-0.09	0.27	-0.34	0.7362	-0.0398	0.0081
<b>Intercept</b>	137.81	12.71	10.85	0.0001		
<b>Waist Circumference (cm)</b>	0.11	0.16	0.71	0.4825	-0.0095	-0.0095
<b>Hip Circumference (cm)</b>	-0.39	0.47	-0.84	0.4073	-0.0059	-0.0154
<b>Intercept</b>	120.00	5.03	23.85	0.0001		
<b>Waist Circumference (cm)</b>	0.07	0.07	0.99	0.3222	-0.0001	-0.0001
<b>Hip Circumference (cm)</b>	0.29	0.13	2.19	0.0309	0.033	0.0329
<b>Intercept</b>	145.89	8.49	17.18	0.0001		
<b>Waist Circumference (cm)</b>	0.04	0.11	0.39	0.6975	-0.0084	-0.0084
<b>Hip Circumference (cm)</b>	-0.65	0.29	-2.23	0.0279	0.0383	0.0299

Young Males

Old Males

Young Females

Old Females

Table V.24

Regressing systolic blood pressure, as a continuous variable, stratified by age group and gender, on the potential determinants produced the regression coefficients presented in Table V.23 and Table V.24. BMI was found to be a significant predictor of blood pressure only in the young females with an  $r^2$  of 0.063 ( $p=0.0041$ ) (Table V.23). When waist circumference and hip circumference were analyzed together, hip circumference became significant only in the old females ( $p=0.0279$ ) with hip circumference explaining most of the variance (Table V.24). The other potential determinants - waist circumference, hip circumference, WHR, and tricep skinfolds - were found not to be statistically significant predictors of blood pressure (data not shown).

Analysis of Variance Stratified by Gender and Age Group								
Variable		AVERAGES					P	Total R-Square
		Values in <b>red</b> are average systolic blood pressure measurements in mmHg						
BMI Classification by WHO	Severe Thinness --- N=0 SD=----	Mod Thinness <b>121</b> N=3 SD=18.1	Mild Thinness <b>148</b> N=6 SD=23.1	Normal <b>132</b> N=14 SD=12.0	Pre-Obese <b>125</b> N=1 SD=---	Obese --- N=0 SD=---	0.1130*	0.252969
	Severe Thinness <b>114</b> N=2 SD=8.5	Mod Thinness <b>168</b> N=3 SD=51.9	Mild Thinness <b>137</b> N=19 SD=17.3	Normal <b>148</b> N=27 SD=19.9	Pre-Obese <b>135</b> N=1 SD=---	Obese <b>151</b> N=2 SD=7.8	0.0697*	0.186419
	Severe Thinness --- N=0 SD=----	Mod Thinness <b>106</b> N=3 SD=17.8	Mild Thinness <b>128</b> N=13 SD=15.3	Normal <b>125</b> N=76 SD=13.3	Pre-Obese <b>131</b> N=17 SD=14.5	Obese <b>146</b> N=5 SD=18.6	0.0021	0.142071
	Severe Thinness <b>139</b> N=7 SD=23.8	Mod Thinness <b>140</b> N=11 SD=20.1	Mild Thinness <b>152</b> N=8 SD=25.9	Normal <b>141</b> N=54 SD=23.4	Pre-Obese <b>146</b> N=20 SD=19.7	Obese <b>182</b> N=3 SD=31.8	0.0680*	0.098994

WHR (All 5 Quintiles)	Category 1 <b>122</b> N=7 SD=11.7	Category 2 <b>160</b> N=2 SD=42.4	Category 3 <b>139</b> N=5 SD=14.5	Category 4 <b>142</b> N=3 SD=15.7	Category 5 <b>131</b> N=8 SD=7.6		0.0387	0.382817
	Category 1 <b>148</b> N=5 SD=23.7	Category 2 <b>138</b> N=6 SD=21.4	Category 3 <b>137</b> N=10 SD=18.8	Category 4 <b>141</b> N=12 SD=18.8	Category 5 <b>150</b> N=19 SD=27.2		0.576*	0.058495
	Category 1 <b>128</b> N=24 SD=16.3	Category 2 <b>127</b> N=30 SD=12.7	Category 3 <b>131</b> N=23 SD=15.1	Category 4 <b>126</b> N=17 SD=14.3	Category 5 <b>121</b> N=17 SD=16.8		0.3261*	0.042452
	Category 1 <b>136</b> N=20 SD=18.6	Category 2 <b>144</b> N=19 SD=29.5	Category 3 <b>137</b> N=20 SD=16.3	Category 4 <b>146</b> N=24 SD=18.9	Category 5 <b>157</b> N=14 SD=30.3		0.0695*	0.089173

\*Not statistically significant

	Young Males
	Old Males
	Young Females
	Old Females

Table V.25

An analysis of variance of systolic blood pressure among the four age-gender groups revealed BMI to be associated with blood pressure only among the young females ( $r^2 = 0.14$ ,  $p = 0.0021$ ) (Table V.25). WHR was associated with systolic blood pressure only among the young men ( $r^2 = 0.38$ ,  $p = 0.0387$ ).



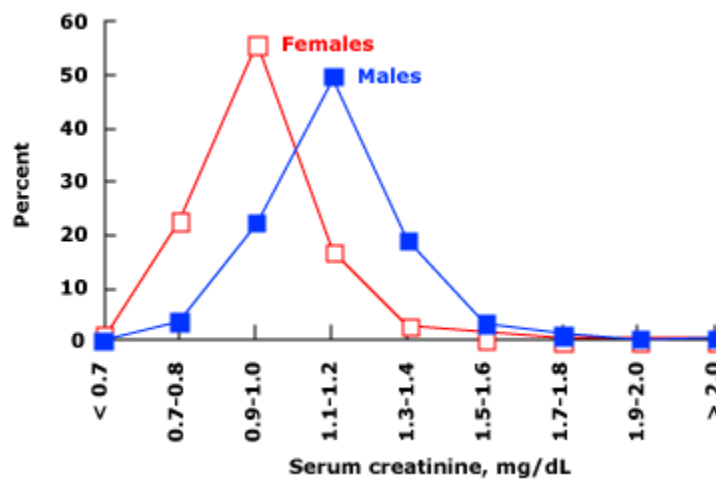
## Creatinine

### Descriptive Statistics

Averages by Variable and Category					
Variable	N	Mean (SD)	Median	Minimum	Maximum
<i>Young Males (18-44 years)</i>					
Creatinine	23	0.99 (0.17)	1	0.7	1.3
<i>Old Males (45+ years)</i>					
Creatinine	46	1.07 (0.36)	1	0.6	3
<i>Young Females (18-44 years)</i>					
Creatinine	92	0.76 (0.16)	0.7	0.5	1.4
<i>Old Females (45+ years)</i>					
Creatinine	84	0.79 (0.18)	0.8	0.5	1.9

Table V.26

Table V.26 stratified creatinine by both age groups and gender, creating four groups: young males, old males, young females, and old females. Descriptively the highest average creatinine value was in the old male group and the lowest average creatinine value was in the young female group. The highest creatinine value out of the entire sample size (3.00 mg/dL) also fell into the old male group (Table V.26).



Graph V.5<sup>16</sup>

The sample's creatinine values were compared to the creatinine values of American men and women from 1988 to 1994 (Jones 1998) (Graph V.5).

<b>Abnormal Creatinine Descriptive Statistics by Age and Gender</b>				
<b>Creatinine Range (mg/dL)</b>	<b>Number of Abnormal Creatinine Values</b>	<b>Age Range (years)</b>	<b>Blood Pressure Range (mmHg)</b>	<b>BMI Range (kg/m<sup>2</sup>)</b>
<i>Young Males (18-44 years)</i>				
All were 1.3	3	19 - 35	132 - 156	18.01 - 21.78
<i>Old Males (45+ years)</i>				
1.3 - 3.0	8	48 - 82	122 - 185	17.26 - 30.9617
<i>Young Females (18-44 years)</i>				
1.1 - 1.4	4	21 - 38	110 - 126	17.15 - 20.52
<i>Old Females (45+ years)</i>				
1.1 - 1.9	4	45 - 78	118 - 152	16.51 - 28.58

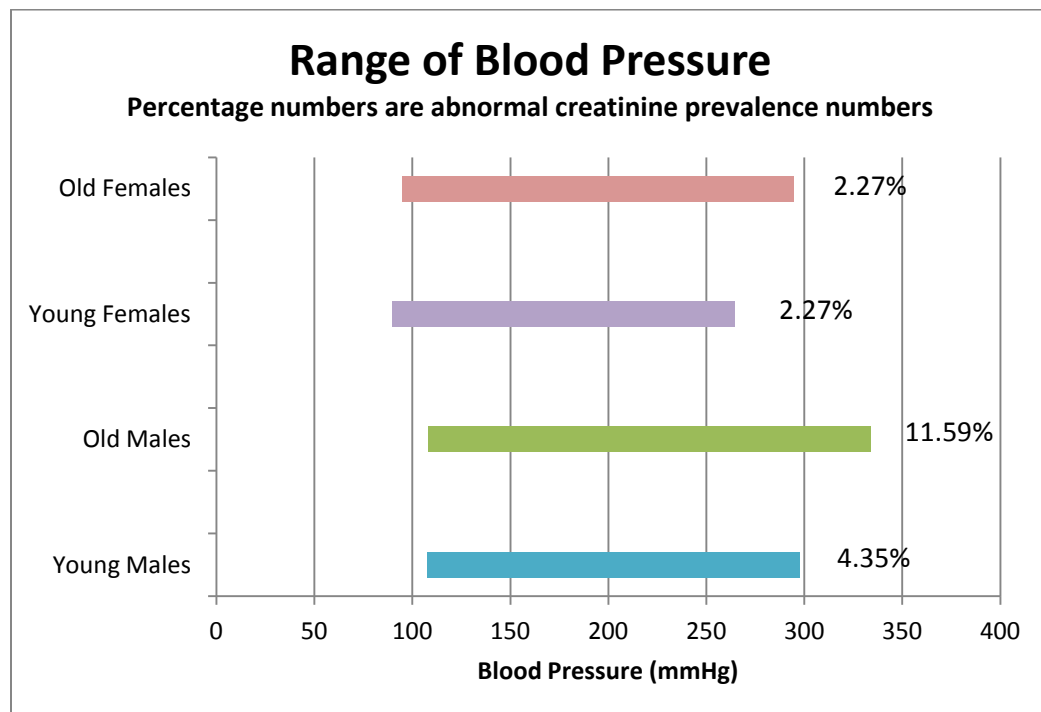
Table V.27

When systolic blood pressure, as a continuous variable, was regressed on creatinine, creatinine explained 1.34% of the variance in blood pressure ( $p=0.0382$ ).

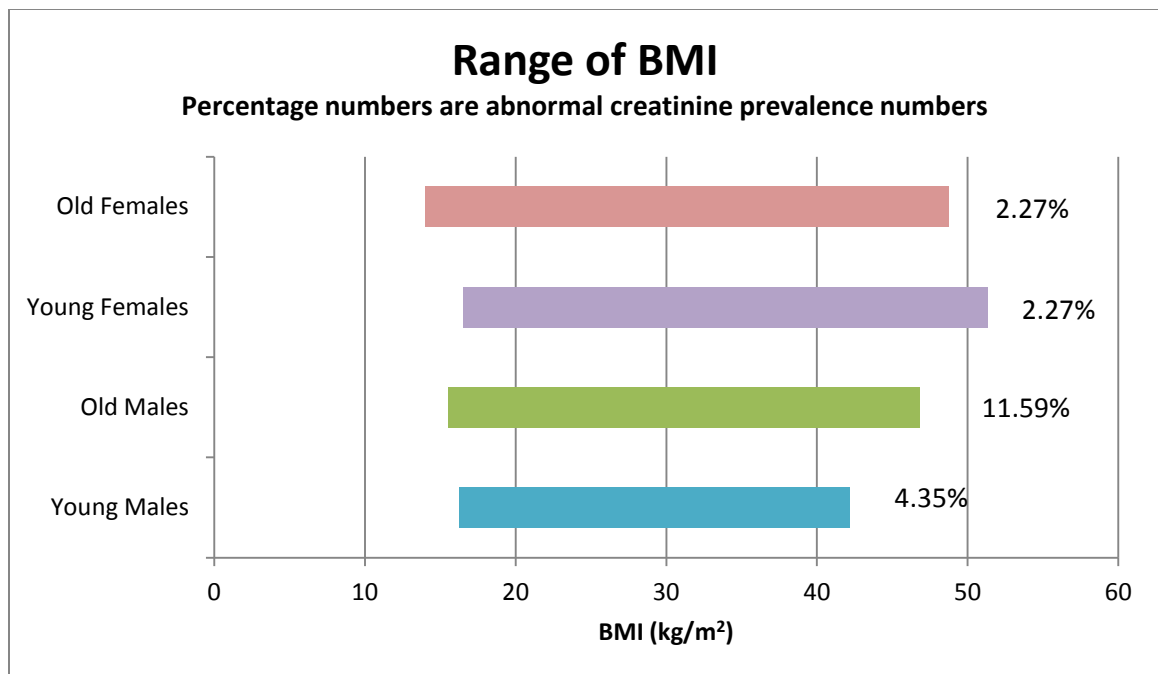
According to US standards presented in Graph V.5, the normal levels of creatinine in the blood are approximately 0.5 – 1.1 mg/dL in females. Out of the 176 women in the study sample with measured creatinine values, 8 of them were considered abnormal or above 1.1 mg/dL, and, thus, there was a 4.55% prevalence of abnormal creatinine in women. Six women had creatinine values of 1.1 mg/dL (+1 SD); 2 women were >2SD above the normal creatinine value. There was an equal amount of young women and old women with abnormal creatinine values. Among the young women with abnormal creatinine, their blood pressure measurements were considered mostly normal. Among the old women with abnormal creatinine, their blood pressure measurements

were mostly high and considered hypertensive. Finally, there was a prevalence of 2.27% of abnormal creatinine in both young and old females (Table V.27).

According to US standards (Graph V.5), the normal levels of creatinine in the blood are approximately 0.6 – 1.2 mg/dL in males. Out of the 69 males with measured creatinine values, 11 of them were considered abnormal or above 1.2 mg/dL; and, thus, there was a 15.94% prevalence of abnormal creatinine in men. Seven men had creatinine values that were +1SD above normal; four men were >2SD above the normal creatinine value. There was a greater amount of older men with abnormal creatinine values than younger men. Among the young men with abnormal creatinine, their blood pressure measurements were high. Finally, there was a prevalence of 4.35% of abnormal creatinine in young males and 11.59% of abnormal creatinine in old males (Table V.27).



Graph V.6



Graph V.7

Graph V.6 presents a graph showing the range of blood pressures stratified by age group and gender with accompanying prevalences of abnormal creatinine. The data shows descriptively that the highest blood pressures were found in the old male group, and the highest abnormal creatinine prevalence was in the same group (Graph V.6).

Graph V.7 presents a final graph showing the range of BMI stratified by age group and gender with accompanying prevalences of abnormal creatinine. The data shows descriptively that the highest BMI's are found in the young females group and some of the lowest BMI's are found in the old female group and the old male group. Among the young male patients, BMI ranges from moderate thinness to pre-obese. Among the old male patients, BMI ranges from severe thinness to obese. Among the young female patients, BMI range from moderate thinness to obese. Among the old female patients, people range from severe thinness to obese (Graph V.7).

### *Data Results as Predicted by Hypotheses*

Research Question One: “How does the age (i.e. two categories of young and old) of patients affect the relationship between high blood pressure and BMI?”

*Hypothesis 1A: Among old patients, there is a positive relationship between BMI and blood pressure.*

Table V.13 displays the results relevant to this hypothesis. According to the regression model, there was a positive relationship between BMI and blood pressure among old patients, but the relationship is not strong. BMI was shown to be a significant predictor of blood pressure in the old group with a small  $r^2$  of 0.0172 ( $p=0.0552$ ).

Therefore the data supports this hypothesis.

*Hypothesis 1B: Among young patients, there is no relationship between BMI and blood pressure.*

Table V.13 displays the results relevant to this hypothesis. According to the regression analysis, there was a positive relationship between BMI and blood pressure among young patients. BMI was shown to be a significant predictor of blood pressure in the young group with an  $r^2$  of 0.0238 ( $p=0.0390$ ). Therefore this hypothesis is rejected.

Upon further investigation, it was found that BMI as a categorical variable was an even better predictor of blood pressure than BMI as a continuous variable. When BMI was categorized using WHO criteria, it gained in explanatory power in the young group ( $r^2=0.117835$ ;  $p=0.0020$ ) (Table V.16).

Research Question Two: “Is the relationship between blood pressure and BMI stronger in men or women?”

*Hypothesis 2A: Controlling for age, the positive relationship between BMI and blood pressure is stronger in male than in female patients.*

Tables V.20 display the results relevant to this hypothesis. Table V.20 show that BMI was a significant predictor of blood pressure only in females ( $p=0.0122$ ). BMI was not statistically significant for the males ( $p=0.5894$ ).

Upon closer inspection in Table V.21, BMI as a categorical variable explained more variance than BMI as a continuous variable for females. BMI as a categorical variable remains statistically significant for females with an  $r^2$  of 0.069055 ( $p=0.0095$ ). BMI as a categorical variable was not statistically significant for males ( $p=0.4836$ ).

Table V.19 shows that BMI was a significant predictor of high blood pressure among female patients ( $p=0.0098$ ). BMI was not significant among males associated with HTN ( $p=0.3011$ ) (Table V.18). The highest prevalence of high blood pressure in males was in the mild thinness category, suggesting a negative relationship between blood pressure and BMI in males.

Therefore, the relationship between BMI and blood pressure seems to be stronger in female patients than male: a potential negative relationship exists between BMI and blood pressure in males while one does exist in females. Thus, we reject this hypothesis.

Research Question Three: “Among old and young patients, will body fat distribution (i.e. truncal vs. peripheral fat) have a positive relationship with blood pressure?”

*Hypothesis 3A: Among both old male and female patients, there is a positive relationship between truncal fat and blood pressure.*

Truncal fat in this study was measured by waist circumference, hip circumference, and WHR (waist to hip ratio). Table V.24 displays the results relevant to this hypothesis. Waist circumference as a single variable and hip circumference as a single variable were found not to be statistically significant predictors of blood pressure for both old male and old female patients. However, when waist circumference and hip circumference were analyzed together, hip circumference became significant only in the old female patients ( $p=0.0279$ ) with hip circumference explaining most of the variance.

WHR as a continuous variable was not a significant predictor of blood pressure; when WHR was categorized as quintiles, it remained insignificant (Table V.25).

Thus, the data showed no positive relationship between truncal fat and blood pressure in old male and female patients. Thus, we reject the hypothesis.

*Hypothesis 3B: Among both young male and female patients, there is no relationship between truncal fat and blood pressure.*

Table V.29 displays the results relevant to this hypothesis. Waist circumference as a single variable and hip circumference as a single variable were found not to be

statistically significant predictors of blood pressure for both young male and female patients.

WHR as a continuous variable was not a significant predictor of blood pressure, but when WHR was categorized as quintiles, it became significant in young male patients with an  $r^2=0.382817$ . This high variance, however, could be due to the small sample size of young male patients (Table V.25).

Hypothesis 3B is partially supported and partially rejected.

*Hypothesis 3C: Among both old and young patients of both genders, there is no relationship between peripheral fat and blood pressure.*

Peripheral fat in this study was measured by tricep skinfold measurements. Tricep skinfold was found not to be a statistically significant predictor of blood pressure for both old and young patients of both genders. Therefore, there was no relationship between peripheral fat and blood pressure. Thus, this hypothesis was supported by the data.



## CHAPTER SIX

### Discussion and Conclusions

#### *Overview*

The purpose of this study was to investigate the prevalence of high blood pressure and associated factors in a rural area of western Kenya with the long-term goal of finding a way to lessen the increasing morbidity and mortality rates of hypertension, an increasingly serious non-communicable disease in developing countries. Despite the world's international focus on infectious/parasitic diseases, there is a steep rise of non-communicable diseases in developing areas. In 2005, infectious diseases caused 5.5 million deaths in Africa. In the same year, non-communicable diseases (NCDs) caused over 2.4 million deaths. While the mortality rate of infectious diseases is still higher, the mortality rate of non-communicable diseases is not too far behind <sup>58</sup>. Thus, there is a need for more research in the area of NCD, especially with regard to hypertension.

Much of the literature concentrates on hypertension in urban areas in Africa. Even in these studies, however, many articles draw attention to the lack of studies done in rural areas. Thus, the hypertension prevalence numbers generated in studies may be underestimations of the population. In a 2008 study done in Sub-Saharan Africa, the

prevalence in the rural area was reported as 13.70%. In our 2010 study done in the Nyakach Plateau in rural western Kenya, the prevalence was found to be 34.58%. The prevalence number in this region more than doubles the prevalence stated in the Sub-Saharan African study. It is startling to see that this prevalence even exceeds the prevalence of hypertension in the United States. The CDC estimated the overall prevalence of hypertension in United States to be 29.90%.

When stratifying by gender, the prevalence of hypertension in 43.59% of males and 35.48% of females is comparable to Cameroon's 44.00% in males and 34.10% in females, lending face validity to the current study. The numbers from the Cameroon study were generated from urban and rural areas. The gender stratified prevalence numbers are higher than the United States (30.60% in males and 28.70% in females). According to Table V.8, the prevalence of hypertension in the United States is 40.60% among those aged 45 – 64 and 70.30% among those aged 65+. This study found that the age stratified prevalence for younger adults is 19.42% - almost double the prevalence rate of the United States (10.50%). These prevalence numbers of the Nyakach Plateau are startling, and they surpass the rural hypertension rates found in other relevant studies except for Cameroon. Because hypertension is a multifactorial disease, it is crucial to investigate further what factors are associated with alarming rates of high blood pressure in our rural area.

### *Primary Hypertension*

The most common cause of high blood pressure in the United States and in many cases around the world is primary, or essential, hypertension. Excessive vasoconstriction is frequently associated with and exacerbated by obesity, a component of metabolic syndrome. However, in a part of rural Kenya where the majority of people are malnourished and thin, the data has shown that many cases of hypertension are occurring without the pattern that we have to come to think of as typical. Hypertension associated with increased weight seems to be occurring in the females more than the males of this study. While BMI has a weak, albeit positive relationship with blood pressure in males, BMI shares a stronger positive relationship with blood pressure in females. BMI was also proven to be a statistically significant predictor of the severity of high blood pressure for the females. Furthermore, the average BMI in females ( $21.76 \text{ kg/m}^2$ ) is higher than the average BMI in males ( $19.81 \text{ kg/m}^2$ ). This indicates that the female patients in our study have higher body fat content than the male patients. Upon further investigation and stratification by age group and gender, it was found that BMI was a statistically significant predictor of the severity of blood pressure specifically in younger females. While this finding seems surprising, the descriptive statistics show the young female patients having the highest BMI in the study sample compared to old females, old males, and young males.

When BMI was broken down categorically, it revealed more into the reason why hypertension may be the explanation of high blood pressures in females. BMI was broken down into six categories using WHO BMI cut-off standards. Excluding the category of

normal BMI, the high percentage of women (18.18%) with high blood pressure is in the pre-obese BMI category (Table V.19). Graph V.4 shows a linear relationship between BMI and average blood pressure for females. As the BMI category jumps from normal to pre-obese and to obese, average blood pressure spikes in the obese category (151 mmHg) (Graph V.4). The data conclusively shows that women follow the typical primary hypertension pattern. With increasing BMI, women tend to have increasing blood pressures.

Age was found to be a better predictor of blood pressure than BMI in females. Thus, when BMI and age were combined, together the variables were able to explain 25% of the variance in blood pressure. The data shows that women follow the typical primary hypertension pattern. With increasing BMI and increasing age, women tend to have increasing blood pressures.

#### *BMI vs. WHR*

There were two measures of body fat content used in this study. BMI was still considered a statistically significant predictor of blood pressure for the old patients, but BMI was found to be a better predictor of blood pressure for the young patients. Furthermore, BMI was found to be a better predictor of blood pressure than WHR for the young patients. WHR, however, was found to be a better predictor of blood pressure than BMI for old patients. This finding brings in the discussion of apple-shaped bodies versus pear-shaped bodies.

Apple-shaped bodies are at more risk of developing cardiovascular diseases and hypertension than pear-shaped bodies. This is due to the fact that apple-shaped bodies have more abdominal adiposity or central obesity and a larger waist circumference than pear-shaped bodies which carry fat in their hips and thighs. WHR rather than BMI is a better measure of central obesity. This explains why WHR is a better predictor of blood pressure for the old group because there are more old people than younger people with central obesity. BMI is not the best measure for abdominal adiposity and may not be a good measure of hypertension risk in old people. BMI remains a better predictor of blood pressure in the young since most young people do not take on the apple-shaped bodies.

Lastly, when BMI was operationalized categorically, it became a better statistically significant predictor of the severity of blood pressure than continuous BMI. Thus, this shows the BMI WHO categories are clinically relevant.

One interesting finding in our study shows WHR being a very significant predictor of blood pressure in young males. WHR in young males was able to explain around 38% of the variance in blood pressure (Table V.25). This could be due to random error from a small sample size or because of another physiological reason. WHR could act as a better predictor of the severity of blood pressure than BMI in young males primarily because BMI is not the best predictor of cardiovascular or hypertensive risk due to the fact that muscle mass is never taken into consideration. Because muscle weighs more than fat and young males tend to have more muscle than other age-gender groups, WHR may eclipse BMI as a better predictor of blood pressure.

Lastly, although much of the literature states how waist circumference is an excellent indicator of cardiovascular risk, this study has found that waist circumference was not a statistically significant predictor of blood pressure for any age group or gender.

### *Curvilinear Relationship of Blood Pressure*

Another interesting finding in these data was a curvilinear blood pressure versus BMI relationship in both young and old. In the old age group, there is an increase in average blood pressure in the moderate thinness BMI category and in the pre-obese and obese categories versus the average blood pressure in the normal BMI category. In the young age group, there is an increase in average blood pressure in the mild thinness BMI category and in the pre-obese and obese categories. The curvilinear relationship trends can be viewed in Graph V.3. When comparing prevalence, there were greater percentages of people with high blood pressure in the mild thinness category (25.93% and 17.86%) compared to the percentages of people with high blood pressure in the pre-obese and obese categories (Table V.11-12).

What we find in our study sample is a curvilinear relationship. Instead of having high blood pressures concentrated in those with high body fat content, high blood pressures are being concentrated among those who are thin and those who are fat; however, blood pressure remains normal for those in the normal BMI category. This trend is happening in both the young and the old. Those who have high blood pressure and are in the pre-obese and obese categories follow the typical blood pressure pattern of higher BMI and higher age equals higher blood pressure. What is curious are those who

have high blood pressure and are in the thin BMI categories. There are a number of reasons why this relationship could happen. There could be a different pathophysiological process going on that involves genetics. Another explanation could be that the thinner people are more likely to have secondary hypertension not associated with the typical weight pattern. Studies have also shown that perhaps low birth weight or childhood malnutrition could lead to a later incidence of hypertension. If the mother has poor nutrition and the baby is born with a low birth weight, there could be some physiological differences in the infant. Infants could develop less vascular elasticity, leading to adult hypertension. Those children who are malnourished and stunted could have less vascular compliance and thus, later cases of hypertension. This is a reasonable explanation for hypertension in thin people in this sample given the frequency of drought, famine, and poverty. However, it is difficult to test this hypothesis in this population since very few babies are born in a hospital and are weighed.

### *Secondary Hypertension*

In our analysis, we have found that there are few variables that could be significant predictors of blood pressure for males. BMI, age, waist circumference, and WHR were found not to be statistically significant predictors of blood pressure for males. Looking at the prevalence, there is a high percentage of males with high blood pressure in the mild thinness category. In Graph V.4, it shows a peak of average blood pressure for males in the moderate thinness category and a drop in average blood pressure in the pre-obese category. Thus, there are more males with high blood pressure who are thin rather than fat.

This is a very interesting and odd finding and could be explained in terms of secondary hypertension, where another disease is causing hypertension. There are multiple reasons for secondary hypertension, including primary renal disease, Cushing's disease, thyroid disease, hyperaldosteronism, pheochromocytoma, obstructive sleep apnea, and coarctation of the aorta. The only one of these diseases that this study was able to explore was renal disease using serum creatinine values. Through the results indicate that renal disease may indeed be associated with hypertension in this sample, it is impossible to know if the renal compromise was the cause or the effect of the hypertension. When analyzing creatinine values, it was found that there are a higher percentage of males rather than females with abnormal creatinine values. Abnormal creatinine values are an indicator of renal problems, specifically a decrease in glomerular filtration rate. Upon closer inspection, the results showed that those male patients with abnormal creatinine values have mostly high blood pressure measurements. Their BMI's revealed that about half of the males with abnormal creatinine values are considered mildly thin. There is also a higher number of older males with abnormal creatinine values than young males, and as mentioned before, there are more old patients who are thinner than young patients. All of these findings together point toward the potential of a kidney disease causing high blood pressures among the thin males. A potential kidney disease causing high blood pressures could be chronic glomerulonephritis which the literature has indicated may be causing high blood pressure in Africa.

People who are malnourished may have lower creatinine values than expected for their age and gender. Because this study is looking at creatinine values which are higher



than the normal and because this study has revealed a high percentage of underweight patients, the prevalence numbers of abnormal creatinine could be underestimated.

One other cause of secondary hypertension is the use of oral contraceptives. Though the use of oral contraceptives in this population was not measured, informal reports suggest that it is very low. Nevertheless, oral contraceptive use would be a useful variable to include in future studies.

### *Systematic Threats to Internal Validity*

Although BMI and age explained much of the variance in blood pressure in females and WHR explained much of the variance in blood pressure in young males, there is still much of blood pressure that cannot be explained in any of the groups. Therefore, there must be other factors contributing to blood pressure of which we are unaware or were unable to measure in this study. Furthermore, this study was done on a clinic sample, and therefore, the prevalence of hypertension could be overestimated for the general population.

### *Random Threats to Internal Validity*

The study found small to moderately large p-values in the regression and analysis of variance models. Although some p-values may be moderately large, they were less than alpha. In some regression and analysis of variance models, p values larger than alpha were found. Therefore, this study could have been significantly affected by random errors such as small sample size when variables were stratified into smaller groups.

Furthermore, the sample has potential bias in that it represents only those able to travel to the clinic, only those in close proximity to the clinic, only those who were aware of the clinic, only those able to be seen in the 14 days of clinic, and only those willing to endure the 8 or more hours waiting in line for the clinic.

### *Construct Validity*

A non-automated sphygmomanometer was used to determine high blood pressure. Automated readings of blood pressure are more accurate, and the manual technique was a source of potential systematic bias. BMI and WHR are accepted measurements of body fat, and the WHO BMI categories used are widely accepted. Weight could have been more precisely measured, but whatever error was present was at least consistent between patients since only one scale was used.

The greatest threat to construct validity in this study is the method used to determine if a patient has hypertension. Because blood pressure was only taken at a single point in time, this method of determining high blood pressure as a chronic disease is only suggestive. And though care was taken to train those who performed the blood pressure measurements, some degree of inter-observer and intra-observer variability is possible. Nevertheless, this method offers the best approximation of high blood pressure in a clinic where most patients cannot be seen multiple times due to time constraints and travel. Follow-up measurements of blood pressure would provide a better standard in whether patients truly had chronic high blood pressure or whether the abnormal measurement was due to chance, an error in measurement, or a transient response to

illness or stress. It would be expected that some of the elevation in blood pressure would be due to acute illness that brought the patient to the clinic. However, almost none of the adults were severely ill as indicated by temperature  $> 101^{\circ}\text{F}$  or a clinical evaluation by the physician of severe illness.

### *Generalization across Persons, Settings, and Time*

By identifying only patients with blood pressure greater than 140/90 mmHg, the researchers attempted to minimize falsely labeling as positive those who may truly be normal or pre-hypertensive. Some features of this study can be generalized to similar, rural populations in sub-Saharan Africa. To the degree that this group of Luo people is genetically unique, however, the study may only describe the prevalence among this tribe.

### *Conclusion*

This study reveals many different concerning points about hypertension in developing countries. It has shown that perhaps there is a greater prevalence of high blood pressure in rural areas than many assume. This could be due to the difficulty of venturing into a rural area and taking measurements. The other frustration that could lead to researchers not spending enough time studying rural areas could be the difficulty of maintaining a treatment program to help the people.

This study has shown how females follow the typical blood pressure correlation with higher BMI and age. This study has also shown that there are a great many more

younger patients with this silent disease than previously thought. It is unclear how much high blood pressure is resulting from a non-modifiable factor (genetics) compared to the contribution of modifiable factors (diet/lifestyle/childhood nutrition), especially in those patients who are young, those patients who are male, and those patients who are thin. The degree of hypertension in these groups suggests that there may be a particularly strong genetic component in this ethnic group. Addressing modifiable risk factors and identifying treatable causes of secondary hypertension, especially among young patients, would reduce the problem of future hypertension during old age.

In conclusion, this community-based research which has grown out of troubling clinical observations has indeed confirmed surprising findings of high blood pressure in those patients who are thin and those patients who are young. Results have shown BMI and age to be significant predictors of blood pressure in the females and not in males, and have also shown how females fit the model of metabolic syndrome with obesity. BMI was proven to be a better measurement of high blood pressure in the young and WHR was proven to be a better measurement of high blood pressure in the old. The study has also demonstrated the possibility of kidney disease causing hypertension in the groups where BMI and age cannot explain high blood pressure. Hypertension can lead to many life-threatening or fatal cardiovascular diseases such as heart attack and stroke, and the rates are increasing at an alarming rate. It is essential to the future of developing countries that we investigate and try to find a solution to high blood pressure before it succeeds in taking its devastating toll on millions of people and countless communities.

## APPENDICES

## APPENDIX A

### *Bethlehem Home*

#### *The “Straw to Bread” Project*

Bethlehem Home is located on the Nyakach Plateau. It was started by Pastor Habil Ogola as a ministry to care for sixty orphans and destitute elders in 2001. Through the partnership of Pastor Habil Ogola, four other local people, the “Straw to Bread” project (started by Dr. Lisa Baker, MD PhD), and Baylor University students, this project has grown beyond the task of providing food and healthcare to the people. It is not just a “band-aid” mission trip and project; it is a growing relationship and friendship born out of humility, care, compassion, equality, and love.

The “Straw to Bread” project seeks to create ways for Luo people to live independently. Most mission trips arrive in an impoverished place and provide temporary healthcare and food supplies to the starving people. This is a temporary cure to what ails poverty in third world regions. The “Straw to Bread” project aims to establish something different. By building a school and creating plans to build a permanent clinic and guest house, this project, along with Pastor Habil Ogola, has a firm dream and mission of creating ways for the members of Bethlehem Home to be able to permanently support and sustain themselves.

While this dream seems far from reach, progress has already bloomed amongst the hopeful community. Gardens are being planted in the school's backyard to teach the children about growing their own vegetables. Water sources are being improved. Gutters are being installed on the roofs of huts. Through many partnerships with people in the U.S., several Luo young people are being sent to pharmacy school, tailoring school, and medical school.

In 2009, a 20,000-liter cistern was built to collect drinking water and thatched roofs were replaced with sheet metal. Students built wooden bed frames for the elders and healthcare was provided to over 800 people living in the Nyakach Plateau area.

In 2010, rain gutters were installed to harvest rainwater for drinking, and fruit trees were planted at homes. Goats were purchased to provide milk for drinking and nutrition supplementation, and a swing-set was constructed for the orphans. The clinic improved by using a medical laboratory to test blood samples for diabetes, anemia, and malaria while "Straw to Bread" became the first in Kenya to use adjustable glasses for people with poor vision through Global Vision 2020.

In 2011, 800 patients were seen by Dr. Lisa Baker and Dr. Bob Dimski. Business seminars were hosted on the plateau, and there was advancement in the water tanks and gutter systems installer around the area. Education in reproductive health issues were trained to the "Village Mothers", an established group of nine local women leaders. Clinical research in asthma, high blood-lead levels, anemic patients, and hypertensive patients continued.

In 2012, four physicians and two business professors will travel to the Nyakach Plateau. A garden will be started and given to the school children and one business professor will begin to train the orphans how to take care of the goats. Clinical research will continue.



## APPENDIX B

### **Love There, Love Here, Love Near**

Here is Kenya – the poorest of the grounds  
But we look closer and around  
And we see through the eyes above  
Oh here it is, here is the meaning of love

Love is seeing Habil with tears in his eyes  
Hugging every person with joyous cries  
No matter the religious, racial walls  
For in this world, we accept all

Love is taking a photo with a child  
Running with them through plains and wild  
Teaching them new letters and words  
And them teaching us games unheard

Love is witnessing plant fledglings' growth  
Of colors, avocados, mangoes, or both  
Pushing new seeds into the soil  
And praying and watching these farmers' toils

Love is the mother and baby goats  
And organizing scattered data notes  
And milk so fresh and white and clear  
And the elders giving care so dear

Love is van trips up and down  
Sleeping and dreaming of a future so sound  
Hearing 'Mzungo!' being shouted in the streets  
And just being with these people in African heat

Love is gazing at the clear skies of stars  
Appreciating home and friends so far  
Giving head rubs and back rubs every night  
Conversing of life and love by dim light

Love is an elder smiling so beautiful and shy  
And seeing a very sick baby live not die  
It is the woman with a swollen leg and desperate tears  
And one physician who can quiet her fears

Love is understanding pain  
Dancing with the children in the rain  
Huddled under rain jackets and boots  
Watching water soak in dry roots

Love is heavy rain turned into a waterfall  
And moving through greens and trees so tall  
It is hearing the people's prayers and low hums  
It's how shared spirituality can make us humbly succumb

Love is understanding beauty is not the same  
Depending on who you are and what you have to claim  
But giving a pen, the gift of words  
And seeing in a child, a mind suddenly stirred

Love is the temporary clinic on the Nyakach Plateau  
And gazing at Lake Victoria and the world of Kenya below  
It is watching a man not take a meal  
So more orphans will eat and their hungers will heal

Love is dancing and singing with these people of faith  
And despite language, being able to relate  
Handing blankets and shoes and toys  
To ecstatic elders, teenagers, girls, and boys

Love is working with them even though we are apart  
It is forever leaving in Kenya my heart  
And together uplifting our spirits to a level above  
Oh here it is, here is the meaning of love

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