ABSTRACT

A Cross-Sectional Study of the Effects of Stressors upon Blood Glucose Levels in Non-Obese Patients in Rural Western Kenya

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In order to more effectively address the expansion of type 2 diabetes worldwide, this study investigated the role that stressors and the stress response play in the development of diabetes. This study analyzed clinical data collected from a sample population of 685 subjects of Luo ethnicity who attended a clinic in May, 2010, in rural western Kenya. Stressors were self-reported by the patients, and blood glucose levels were determined from blood samples. Diabetes, measured as blood glucose levels over 200 mg/dL, was found to be unrelated to body mass index, an accepted measure for obesity (X^2 = 2.51, df = 5, p = 0.7753).

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A CROSS-SECTIONAL STUDY OF THE EFFECTS OF STRESSORS UPON BLOOD GLUCOSE LEVELS IN NON-OBESE PATIENTS IN RURAL WESTERN KENYA

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TABLE OF CONTENTS

LIST OF FIGURES AND TABLES

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Dedication

This study is dedicated to my parents for their unending support, Jason and Christie for getting me on the plane that started this journey, Nathan and Haley for making my summers less productive if infinitely more enjoyable, and Mr. Dean Madison for the much needed prayer.

CHAPTER ONE

Introduction

Diabetes is growing at an alarming rate on a worldwide scale. While much has been done to describe the disease's manifestation in the biological sense, there is a gap in the literature when considering the socio-cultural origins of the disease. A clear understanding of all of the possible contributors to the development of a disease that is becoming a near pandemic is necessary in order to be able to contain it.

One area which has been overlooked in the field of diabetes is the role that stressors and the stress response play in the development of the disease. The Luo people of western Kenya are a group under significant social and biological stress; however, the incidence of obesity is low among this people group. Thus, by analyzing the blood glucose levels of non-obese Luo under significant stress, this study hopes to fill a gap in the literature by providing information on the prevalence of type 2 diabetes among people who are non-obese.

CHAPTER TWO

Review of Literature

In the developing world, type 2 diabetes is strongly associated with obesity. As the developing world begins to shift to a higher level of obesity, type 2 diabetes is increasing. However, diabetes mellitus is a metabolic disease characterized by hyperglycemia, or high blood sugar levels, and resulting when either the pancreatic islets (or *Islets of Langerhans*) are unable to secrete the required amount of insulin or the secreted insulin is not employed effectively[1–4]. Diabetes is divided into two major classifications depending upon the conditions in which it manifests: type 1 diabetes, which results from a deficit of insulin, and type 2 diabetes, which stems from an insufficient reaction to insulin[1,2,5,6].

Type 2 diabetes is the more common of the two, accounting for roughly 90% of diabetes cases in the developed world and holding an even higher percentage among developing countries[3,5–7]. The symptoms for diabetes include frequent urination, unusual thirst and hunger, unprecedented fatigue and irritability, frequent infections, blurred vision, wounds that are slow to heal, and tingling and numbness in the hands and feet[1,8,9]. However, persons with type 2 diabetes often show no symptoms of the disease[8]. This is because the onset of type 2 diabetes is gradual, and therefore, symptoms may be difficult to detect until complications have already arisen[9,10]. In addition to being more prevalent, type 2 diabetes results in a more complicated metabolic defect[2] being associated with hypertension, hyperlipidemia, and inflammation.

In order to understand the complexity of type 2 diabetes, one must first understand the influence of insulin in the body. Insulin is a hormone that is essential in regulating carbohydrate, protein, and fat metabolism through facilitation of energy storage in cells[1,2,4]. Insulin is produced in and secreted from the pancreas in response to the concentration of glucose in the blood[1–4]. Insulin primarily acts upon liver cells, muscle tissue, and adipose (fat) tissue by promoting the access of glucose to the cells and supporting the storage of glucose as glycogen[1,2]. The conversion of glucose into triglycerides rather than glycogen is preferred by insulin in adipose tissue, and recently created triglycerides are stored within the fat cells. Insulin likewise favors the entry of amino acids into cells to stimulate protein synthesis[1,2].

In type 2 diabetes, the pancreatic islets may secrete normal or increased volumes of insulin, but the tissues upon which the insulin is acting are unresponsive or incapable of reacting in the appropriate manner[1–3,11]. In time, many type 2 diabetics lose beta cell function and require insulin supplements as seen with type 1 diabetics. This sort of inadequate reaction to insulin is referred to as *insulin resistance*[1,2]. As a result of insulin resistance, glucose is absorbed normally, but it is not used effectively for energy and is not stored properly as glycogen[2]. Thus, excess glucose – which is neither utilized as energy nor stored as glycogen – amasses in the bloodstream, resulting in the distinctive increase in blood glucose levels, or hyperglycemia[2–4]. This surplus glucose is excreted with the urine; however, due to the high osmolarity of the blood in the presence of glucose, water moves from low osmolarity to high osmolarity, and, consequently, extreme amounts of water and electrolytes are excreted along with the urine. This can cause a fluctuation in water and acid-base balance within the body[2]. Furthermore, failure

to store glucose properly results in a perceived deficiency of glucose by the body. To counteract this apparent imbalance, protein is broken down into its building components, amino acids. The liver then uses the amino acids to create more glucose, enhancing the already existing hyperglycemia and leading to an increased loss of glucose, water, and electrolytes in the urine[2].

Hyperglycemia is not the only complication that diabetics face; however, it is the cause of a host of other complications affecting the vast majority of organ systems within the body[4]. Ketoacidosis can occur when insulin levels are lacking[2,12,13]. Without insulin, glucose is unable to enter the cells to be utilized as an energy source, and carbohydrate metabolism does not occur. Thus, fat is broken down in order to provide energy, producing ketones[2,12,13]. Acid ketones accumulate in the blood and are excreted with the urine. Through osmosis, water and electrolytes are similarly expelled from the body. If the hoards of ketone bodies overpower the body's natural buffer systems, the normal blood pH will be disrupted, resulting in ketoacidosis[2]. Critical acidosis can lead to coma, loss of cerebral function, and death[2,13]. Ketoacidosis is most common among persons with type 1 diabetes[2,11–15]; however, recent studies have shown that it is possible for a type 2 diabetic to experience ketoacidosis if there is a precipitating event to lower insulin levels[5,11,12,14,15]. The greater proportion of such cases occur in obese and comparatively young African Americans and Latino Americans, though diabetic ketoacidosis has been reported in European American, Native American, and Japanese American patients diagnosed with type 2 diabetes[11,14]. In one study of 138 patients admitted to the hospital for moderate-to-severe ketoacidosis, 30 had type 2 diabetes; 69.2% of the cases involving type 2 diabetics were the result of a discontinuation of medications and 48.4% involved

infections[14]. In another study[15] with a total of 121 patients with 137 episodes of ketoacidosis, 98 episodes occurred in patients with type 2 diabetes. Among ten patients suffering from repeated episodes of ketoacidosis, four were type 2 diabetics. Furthermore, infection was identified as the most common precipitating factor in type 2 diabetic patients, with respiratory tract and urinary tract infections predominating[15]. However, in the majority of diabetic ketoacidosis cases, no apparent precipitating cause was identified[11].

Persons with type 2 diabetes have the potential to become comatose as a consequence of the extreme hyperosmolarity of body fluids that results from severe hyperglycemia in the absence of ketosis[2,13]. The exaggerated rise in the osmolarity of the body fluids causes water to move, by osmosis, from the inside of the cells to the more concentrated extracellular fluid and then into the capillaries. As a result, the cells become shriveled and dehydrated. The kidneys also excrete the excess glucose with the urine, increasing dehydration if fluids are not replenished[16]. This disrupts the function of neurons which leads to coma[2].

Persons with diabetes can also suffer from hypoglycemia, a condition which occurs when blood glucose levels are too low[2,17–20]. Hypoglycemia requires a rise in insulin levels relative to the amount of carbohydrates to be metabolized[2,19]. A surplus of insulin causes a sheer drop in blood glucose levels and initiates a chain reaction called *insulin shock*[2]. In an attempt to establish balance, insulin secretion is inhibited, and the pancreas secretes glucagon while the adrenal medulla releases epinephrine to raise blood glucose through the conversion of glucagon[19]. Norepinephrine is discharged from the adrenal medulla and sympathetic postganglionic nerve terminals. This activation of the

sympathetic nervous system causes a rise in lipolysis in adipocytes, and the resulting increase of free fatty acids enables considerable glucose sparing[19]. In fact, the contribution of fatty acids in the blood has been estimated to make up 25% of the body's total defense against hypoglycemia[19]. If hypoglycemia continues uncontrolled or the body's measures are not adequate to achieve homeostasis, coma, seizures, and death can result[2,17]. While hypoglycemia is more common and typically more severe in those with type 1 diabetes, it has been shown to be relatively common in type 2 diabetics with prevalence of 70-80% in clinical trials among those administered insulin to enhance metabolic control[5,19]. In fact, duration of insulin treatment has been identified as an important predictor of the first episode of severe hypoglycemia, with the threat escalating by 33% each year of insulin administration[18]. In one study, symptoms of hypoglycemia were reported by 35.8% of the patients who had type 2 diabetes, with 11.6% labeling the symptoms as severe and 8.2% as very severe[20]. Though prevalence of severe hypoglycemia is significantly lower among type 2 diabetics than type 1 diabetics[17], it has been reported that, in patients with advanced type 2 diabetes treated with insulin, the glucagon response to diminishing blood glucose was virtually absent[19]. Therefore, patients with type 2 diabetes, similar to those with type 1 diabetes, are at risk for autonomic failure caused by hypoglycemia[19].

Further complications of diabetes include an increased susceptibility to infection caused by the fact that pathogenic anaerobic bacteria show an affinity for 1) blood with elevated glucose levels and 2) oxygen-poor tissue resulting from arteriosclerosis caused both by inconsistencies in fat metabolism and elevated levels of lipids in the blood typical of diabetics. Complications resulting from arteriosclerosis such as strokes, heart attacks,

and gangrene of the lower extremities as a result of inadequate circulation also are common[2]. Persons with type 2 diabetes are at a higher risk of macrovascular disease and lower limb amputation, and can display anywhere from a two- to four-fold increase in cardiovascular disease, making cardiovascular disease the most significant killer in the diabetic population[1,5,21–27]. In fact, diabetes now ranks among smoking, high blood pressure, and cholesterol-related disorders as a major risk for cardiovascular disease. Additionally, persons with diabetes are more likely to suffer increasingly severe consequences of cardiovascular disease and less likely to survive long enough to reach the hospital in the event of a cardiovascular episode[24].

Diabetics are also susceptible to long-term complications that amplify as the duration of the disease progresses[1–3,5,7]. Blindness can result from degenerative changes in the small blood vessels supplying blood to the retina[1–3,5]. Diabetic retinopathy is the leading cause of blindness in adults: up to 15 years after diabetes manifests, 2% of sufferers will become blind, and another 10% will develop severe visual impairment[3,27]. Furthermore, diabetics are six times more prone to cataracts and 1.4 times more vulnerable to open-angle glaucoma than the general population[21,28,29]. The glomerular arterioles and capillaries of the kidney also undergo degenerative changes in diabetics that impair renal function and can result in kidney disease or failure[1–3,27]. Similarly, peripheral nerves degenerate, resulting in neuritis and neuropathy which cause tingling, pain, numbness or weakness and disrupted sensation in the extremities and can put sufferers in danger of injury, infection, and amputation[2,3,21,27,30].

Though the cause of insulin resistance is not completely understood, it is agreed that type 2 diabetes has a strong genetic component to its manifestation and is influenced

and promoted by environmental and behavioral factors such as obesity, physical inactivity, and the consumption of energy-dense foods with low nutritional value[1,5,6,10,31]. Type 2 diabetes typically appears in persons over the age of 40; however, in countries with rising rates of obesity and a growing proportion of the population that is overweight, type 2 diabetes is becoming increasingly prevalent in adolescents and children[5,6,10]. In fact, obesity has been identified as the primary risk factor for type 2 diabetes, with 80% of type 2 diabetics considered medically overweight[1,10,31]. Additionally, obesity is rapidly overtaking childhood under-nutrition as an imperative disease and is more demanding in light of its overall burden upon society[32].

Furthermore, the prevalence of obesity is on the rise. Approximately one-third of adults in the United States are obese: 27.6% of men and 33.2% of women. Additionally, one in six children and adolescents is overweight[33]. Obesity is not only impacting the United States, however; in 1980, 4.8% of men and 7.9% of women in the world were obese, compared with 9.8% of men and 13.8% of women today[34]. Moreover, the effects of obesity are not only seen among the affluent[32,35,36]. In one study, the prevalence of overweight and obese individuals in an urban setting increased by 35% during the observed period, and that increase was significantly larger among the poorest in society (+50%) than it was among the elite (+7%)[36]. Obesity is not only shifting from a few-high income countries to global prominence, but growing rapidly among low- to moderate-income countries[35].

With obesity and its complications extending across the globe, researchers are delving deeper into this near-pandemic. As contemporary Western culture, characterized by high food availability and a low-energy expenditure lifestyle, is shared across

increasingly distant continents, its non-communicable diseases are imparted as well[1]. One cause of the rapidly increasing obesity rates in developing countries is the shift in diet to mirror Western consumption, mainly in the categories of fat, caloric sweeteners, and animal source foods[35,37]. In remittance economies where money is sent home from family members who have left for urban areas or other countries, the selection at the local markets is vastly expanded to accommodate the improved finances of the village members[38]. One such newly introduced product is edible vegetable oil[35,38]. Between 1992 and 1996, global production of vegetable oils increased from 60 to 71 million metric tons[35]. Increased production was caused by increased consumption across destitute and affluent countries alike; however, the impact was greater among the low-income countries[35,39,40]. Caloric sweetener consumption has also increased across the globe, accounting for a greater proportion of total energy and total carbohydrates consumed. 306 kcal were consumed per person per day worldwide in 2000, approximately three times as much as in 1962[35]. Animal source foods consumption, such as meat and dairy products, is also on the rise. In the past 50 years, annual consumption of animal foods in China nearly tripled, escalating from 11 kg per capita in 1952 to 38 kg per capita in 1992. The increase was gradual before 1979, rising only by 0.2 kg annually to reach a total of 5.6 kg; from 1979 to 1992 consumption increased by 1.6 kg annually for a total of 21 kg[35,37].

The result of this nutrition shift among developing countries is that the rate of change in obesity in lower- and middle- income countries is significantly more profound than that in higher-income countries[35]. In fact, the rates of change in obesity in Asia, North Africa, and Latin America are two to five times higher than the annual rise in

prevalence of obesity among the men and women of the United States and Europe, which is approximately 0.25 for each[35]. Additionally, there is a greater likelihood among low- and moderate-income countries that adults from lower-income or lower-educated residencies will be overweight or obese relative to those adults of higher-income or higher-education[35,41].

Culture may also play a role in the rising prevalence of obesity among developing nations. When examining the origins of obesity, one must consider ethnicity, gender, and social class in order to draw conclusions regarding cultural beliefs on food and ideal body size[42,43]. For example, obesity occurs much more frequently among minority, low-income, and less educated persons, and minority ethnic groups generally have a higher-risk body fat distribution located around the abdomen[1,44]. Additionally, ethnic differences such as variable prevalence of obesity in adult females and gestational diabetes, parental practices such as overfeeding or providing high calorie foods and beverages that may be socially or culturally valued, lax attitudes toward physical activity, and exposure to ethnically targeted food marketing may all lead to higher rates of obesity among ethnic groups[42]. Furthermore, many ethnic groups are more accepting of those who are obese. Studies have shown that African American females experience significantly less pressure to be thin than their European American counterparts[45]. In one study[46] conducted among European American, Japanese, African American, Filipino, Chinese, Hawaiian, and multi-ethnic college students in Hawaii, it was found that men in general were more self-satisfied with a greater range of BMIs while women favored a smaller BMI. However, whereas European American males had high BMIs and were self-satisfied, Filipino males with high BMIs followed the female pattern of body

dissatisfaction. Chinese females had low BMIs and were very satisfied while Japanese females also had low BMIs but were very dissatisfied[46]. Another study conducted in Morocco and Tunisia discovered that half of all middle-aged women were obese, a prevalence three times greater than that seen in the men. Furthermore, in both countries, female obesity is seen as a sign of high social status and is a cultural symbol of beauty, fertility, and prosperity[47]. A study conducted in Gambia, an African country typical of many rapidly developing nations across the continent, used body image assessment for obesity and figure rating scale silhouette charts to map the body weight ideals among urban adults and determine differences with regards to age and gender. The results showed a high level of body satisfaction among the overweight and obesity was openly accepted, especially among middle-aged women. It has been speculated that acceptance of obesity within African populations could be a direct result of the thinness and frailty associated with the HIV/AIDS virus[38]. Obesity indeed appears to be a culture-bound syndrome[48].

With the prevalence of obesity and type 2 diabetes increasing across the globe, researchers are becoming increasingly interested in the body mechanisms that seem to be favoring weight gain. This search has lead to both the 'thrifty gene' and the 'thrifty phenotype' hypotheses. The thrifty gene hypothesis, as developed by Neel, essentially states that certain populations may have a genotype for glucose storage that, during times of feast and famine, offered a selective advantage and greater reproductive fitness for individuals with the ability to quickly release insulin to store energy in time of abundance and effectively utilize energy stores during times of scarcity[1,38,49–51]. However, shifts to modern lifestyles in which food is consistently available has made a once adaptive gene

injurious as it promotes the energy storage leading to obesity and, consequently, type 2 diabetes[1,49–51]. The thrifty gene hypothesis finds support among animals such as *Psammomys obesus* and human populations with a high susceptibility for type 2 diabetes such as Native North Americans and South Pacific Islanders[51]. In fact, one would expect that certain populations in developing countries would be more at risk for emergent obesity and diabetes[38]. For example, the ancestors of present-day Polynesians experienced long and stressful open ocean voyages and are speculated to have suffered cold stress and starvation during their settlement of the Pacific. These circumstances may have favored a high frequency of thrifty alleles and may account for the high prevalence of obesity and type 2 diabetes within the Polynesian population today. Alternatively, high frequencies of thrifty alleles could have manifested in the population through the founder or bottleneck effects when the Polynesians migrated from Taiwan. However, searches for thrifty genes within the Polynesian people have been unsuccessful so far[49]. In fact, while some studies have been able to find evidence for selection in certain loci, they have been unable to discover a consistent pattern of selection to provide conclusive confirmation of the thrifty gene hypothesis[50]. Indeed, it seems much more likely that the thrifty genotype is the result of multiple polymorphisms at several sites rather than the effect of a single abnormality[51]. Furthermore, the founder effect hypothesis, while still a valid concept, has neither proof nor any leads to ascertain the identity of such a thrifty gene; the fact that all of humankind has been subjected to periods of feast and famine should not grant the thrifty gene privileged status in explaining effects in populations of developing countries[38]. Additionally, in Gambia where 32.6% of women over the age of 35 are obese compared with less than two percent of men in the same age group, the severe

sexual dimorphism appears to suggest environmental and behavioral factors are at play rather than genetic facors[38]. The search for the elusive thrifty gene is a complicated and time-consuming task as a result of the fact that such a gene must encompass every facet of human energy balance from the search for resources right down to the efficiency of oxidative phosphorylation[38].

The thrifty phenotype hypothesis, developed by Hales and Barker, presents the idea that the associations between inadequate fetal and infant growth and the development of type 2 diabetes result from permanent alterations to the glucose-insulin metabolic pathway introduced by poor nutrition in early life. These changes reduce the capacity for insulin secretion and promote insulin resistance. These effects are imperative for early survival but become maladaptive when nutritional conditions improve in later life. When combined with obesity, aging, and low levels of physical activity, they become the most determining factors in the development of type 2 diabetes[1,38,52,53]. Furthermore, it has been demonstrated that both obesity and type 2 diabetes are more prevalent in children belonging to mothers who were diagnosed with diabetes during pregnancy, which suggests a predisposition rising from the influence of the intrauterine environment[52]. The thrifty phenotype hypothesis also rests upon the idea that manifestation of disease later in life is correlated with low birth weight. Among the Pima, both low and high birth weights are associated with a higher risk of developing type 2 diabetes, and low birth weight was found to be positively associated with parental diabetes, though only through the fathers[52]. However, there also exists evidence showing no correlation between intrauterine environment and later development of disease. One study conducted in both monozygotic and dizygotic twins revealed no association

between birth weight and either blood pressure or glucose intolerance[52]. Furthermore, while children born during famine in Holland exhibited significantly higher than normal blood glucose levels, children born during the siege of Leningrad demonstrated no long-term effects on blood glucose[52].

Recently it has been suggested that obesity is truly an inflammatory disease and excess body fat and diabetes are thus linked through inflammation. Research has discovered that obese individuals suffer from chronic, systemic low-grade inflammation – as obesity is characterized by the amassing of macrophages within adipose tissue[1,54]. Macrophages contribute to the basic inflammatory response that favors a catabolic state, and therefore acts as a suppressor of anabolic pathways, most notably the insulin signaling pathway; this contributes to obesity-linked inflammatory diseases such as diabetes[54,55]. Obesity is characterized by a broad inflammatory response, and inflammatory and stress-response genes are among the most highly monitored gene sets in the adipose tissue of obese animals[54].

Tumor Necrosis Factor- Alpha (TNF-α) was the first molecular link discovered between inflammation and obesity. TNF-α is overproduced in the adipose and muscles tissues of obese individuals and acts as an enhancer for inhibitory phosphorylation of serine residues, thereby inhibiting the actions of insulin[54,56–60]. Removal of TNF-α has been demonstrated to protect against insulin resistance in obese mice, and treatments that target pathways in which TNF-α is involved improves insulin sensitivity in humans[54,61,62]. Resistin is a newly discovered adipocyte peptide that increases both insulin resistance and fat storage. Levels of resistin increase with obesity, and drugs that cause a decrease in resistin have been shown to invoke weight loss[1,63].

Resistin levels can be decreased by the drug Rosiglitazone. Anti-resistin drugs improve insulin action in mice with diet-induced forms of obesity whereas treatment of normal mice with resistin impairs insulin action. Additionally, glucose uptake by adipocyte tissue stimulated by insulin is enhanced by neutralization of resistin, potentially linking obesity with diabetes[63].

The hormone leptin acts within the body to create a sensation of fullness. Studies have shown that leptin-deficient mice that receive leptin eat less and lose weight; however, the mice were shown to be more sensitive to a deficiency of leptin which promoted weight gain than they were to increased levels of leptin which diminished appetite and induced weight loss. Anti-obesity drugs developed with leptin as their bases have been ineffective for treating humans[1]. Moreover, mice and humans with deficient leptin function demonstrate impaired immunity, and leptin administration has been shown to reverse immunosuppression of starved mice[54].

Metabolic stresses can also activate the inflammation signaling pathway. Studies have shown that obesity overloads the capacity of the endoplasmic reticulum (ER); this stress on the ER leads to launching of inflammation signaling pathways which contribute to insulin resistance. However, insulin resistance has been found even in the absence of obesity when inflammatory cytokines or lipids were injected in animal cells[54]. Furthermore, stress affects metabolic activity by triggering the release of various hormones responsible for raising blood glucose levels[64–67]. Thus, stress is a potential contributor to hyperglycemia in diabetes, and patients with type 2 diabetes are ill-equipped to metabolize the elevated levels of blood glucose effectively. Moreover, regulation of stress hormones may be irregular in diabetes[64–66].

Both the number of cytokines and macrophages increases with psychosocial stress and the subsequent cellular inflammation and activation of the immune system can lead to type 2 diabetes[1]. Indeed, present evidence strongly suggests that type 2 diabetes is primarily an inflammatory disease which results in obesity-linked insulin resistance, hyperlipidemia, and hyperglycemia and is not an outcome itself[54]. Psychosocial stress has also been shown to increase cortisol secretion. Cortisol is a hormone which increases blood glucose levels and promotes storage of fat in the abdominal area[1,67]. Additionally, exposure to stress increases the number of individuals experiencing sleep disorders and sleep deprivation. In turn, short habitual sleep deprivation has been associated with reduced leptin levels. Therefore, individuals lacking sufficient sleep tend to have higher body mass indices as a result of increased appetite and energy intake[1].

Patients with diabetes are encumbered with greater anxiety and psychological tribulations as a result of their marred quality of life, especially regarding complications caused by the disease, and many patients believe that their development of diabetes was caused by an adverse or stressful life event[5,68,69]. In fact, both diabetes and obesity have been linked with stressful historical, social, and economic events[1]. One study[70] investigating the development of diabetes among Mexican-American migrant workers found that diabetics had a statistically significant greater number of years spent in migrant labor than non-diabetics. Additionally, 56% of diabetics, compared to 33% of non-diabetics, had one or fewer years of school. Furthermore, among those individuals with one or fewer years of school, participants who were diabetic had worked in an average of four states while those without diabetes had worked in only one state. The study also found that across diagnostic categories, 82% of the subjects who had been

migrants for more than 20 years had diabetes. The results of the study suggest that, although accepted as an important contributor, obesity is not the sole contributor to diabetes risk. Considering that obesity is a result of increased levels of cortisol in individuals experiencing stressful stimuli, obesity could actually be a tertiary factor[70]. Studies also show that family members, primary care physicians, and diabetologists are the main sources for psychosocial support when experiencing stressful life events[69].

However, while it has been demonstrated that psychosocial stress is related to a deterioration of glycemic control in diabetics, there is little evidence that stress can create diabetes in an otherwise healthy patient[68]. There are a large number of animal studies that support the idea that stress reliably produces hyperglycemia in persons with type 2 diabetes[64]. One study[71] conducted in Otsuka Long-Evans Tokushima Fatty rats, a model animal for type 2 diabetes in humans, explored the effects of environmental stress on metabolic derangements and the diabetes phenotype. The study found that acute environmental stress caused a temporary boost in blood glucose levels and a decrease in insulin secretion. Furthermore, stress-induced hyperglycemia amplified with age and was accompanied by increased plasma levels of both catecholamines and corticosterone[71]. Investigations into stress-induced hyperglycemia in type 1 diabetes have been contradictory: some cases have demonstrated that stress stimulates hyperglycemia and some that it causes hypoglycemia, while still others show no relationship at all between stress and blood glucose levels[65,66].

Whereas animal studies demonstrating the role of stress in the manifestation and development of type 2 diabetes are abundant, evidence for the role of stress in onset and outcome of type 2 diabetes in humans is sparce[65,66]. However, it is important to more

fully understand the complex causal pathway leading to type 2 diabetes so that it can be prevented more effectively. Diabetes is already one of the leading causes of death and disability in the United States, and total healthcare costs for its treatment reach about $174 billion annually[6]. The World Health Organization estimates that between the years of 2006 and 2015, China will spend $558 billion of national income solely on diabetes, heart disease, and stroke, and it is estimated that the costs of treating the complications of diabetes account for 5% to 10% of all that is spent on health care in the world[3]. Diabetes is not only a devastating disease in terms of morbidity but also with regards to mortality. In 2004 alone an estimated 3.4 million people died from consequences of high blood glucose[3]. In fact, diabetes is the cause of 5% of all deaths worldwide each year[72,73]. In people ranging from ages 35 to 64 years old, 6% to 27% of deaths are attributable to diabetes[72]. Moreover, deaths caused by diabetes are expected to double by 2030[3,73].

Not only is diabetes a destructive disease in terms of cost and mortality, but it is also becoming more prevalent. In 1985, an estimated 30 million people had diabetes worldwide[27,74]. The International Diabetes Federation predicts that number to increase to 324 million people in 2025, and the World Health Organization predicts an increase to 366 million in 2030[5,75,76]. With approximately seven million people developing diabetes each year, the predictions agree that type 2 diabetes is the most common chronic disease in the world and it is becoming more prevalent at an alarming rate[3,5]. It is estimated that in the next thirty-five years, the world-wide prevalence of diabetes will reach 25%, with 80% of cases appearing in developing countries and a greater proportion of those cases coming from the continents of Asia and Africa[5,21]. Developing countries are expected to see an increase of 170%, from 84 million to 228 million persons with type 2 diabetes[5].

Indeed, the World Health Organization reports that 80% of people with type 2 diabetes live in low- to middle- income countries and that 80% of deaths from diabetes and its associated complications will occur in developing countries[5,73,77]. Additionally, the vast majority of people with type 2 diabetes in low- and middle- income countries are between the ages of forty and sixty-five[5,73,78]. Thus, the disease is affecting them during the most productive period of their lives. Furthermore, because these individuals are diagnosed at an earlier age, they have more time to develop complications from the disease[5].

Currently, the lowest regional prevalence for type 2 diabetes, approximately 1.2%, is in Sub-Saharan Africa[1,79–81]. However, Africa is not escaping the grasp of diabetes. As developing countries on the continent shift from hunter-gatherer societies to industrial-based economies, they are trapped between the double threat of infectious diseases and the chronic diseases emerging as a result of the transition[5]. From 1959 through the middle 1980s, medical statistics showed a prevalence for diabetes of less than or equal to 4.9% throughout Africa. However, by 1994, prevalence of diabetes for the entire continent was reported as 3 million and was predicted to double or triple by 2010[21]. Indeed, in the fifteen years leading up to 2010, diabetes in Africa increased by 93%[5]. It is estimated that 7.1 million Africans were suffering from diabetes at the end of the year 2000, a number that is expected to rise to 18.6 million people by 2030[21]. The International Diabetes Federation estimated that in 2003 the prevalence in the African region among adults aged 20 to 79 to be 7.1 million persons and predicted that the prevalence would rise to 15 million persons by 2025[5]. The impending severity of diabetes on the African continent is such that epidemiologists predict its economic impact and death toll to exceed the devastation of HIV /AIDS virus in the near future[21].

Kenya, like other quickly developing countries in Sub-Saharan Africa, has a rising prevalence of diabetes, with type 2 diabetes being the more common[31,82]. Furthermore, Kenyans are developing the disease at a younger age than their counterparts in developed countries and are at higher risk for life-threatening complications because they report to health centers only after symptoms have appeared and the disease has progressed[82]. The Ministry of Health in Kenya states that 1.2 million Kenyans live with diabetes, and if the rate at which the prevalence of the disease is increasing continues unabated, that number will increase to 1.5 million by 2025[82]. A diagnosis of diabetes can be a sentence to a lifetime of poverty, including refusal of access to education or fundamentals such as food[5]. Although diabetes has been thought to be a disease which affects the urban middle-class, its reach is extending to those living in poor and rural areas[74]. Additionally, the inaccessibility of information, medications, and appropriate foods stand in the way of treatment[5]. However, whereas diabetes is becoming ever more present in rural life and on the continent of Africa, obesity still remains rare throughout Sub-Saharan Africa as a whole[74,76].

This study investigates the relationship between stress and type 2 diabetes among the Luo people in the Nyanza district of Western Kenya where obesity is rare. The Luo, comprising approximately 2.7 million in population, are the second largest non-Bantu ethnic group in Kenya. The Luo migrated to the area roughly five centuries ago from the vicinity of Sudan[83]. As the Luo expanded from the level land surrounding the lake basin to the more rugged and heavily watered uplands, they adjusted their strategies for livelihood[84]. The first Luo immigrants lived a migratory lifestyle, primarily concerned with finding suitable pastures for their herds of cattle. However, when the population

began to grow, they adopted a sedentary way of life consisting of moderately secluded homesteads[83]. The shift away from pastoralism was encouraged by the growing population of tsetse flies in the wooded areas. The cattle, sheep, and goats tended by the Luo are an esteemed form of investment for their owners[84]. Cattle continue to govern ceremonial and economic activities, but agriculture and fishing have become increasingly significant for subsistence[83,84]. The Luo's agricultural endeavors began with sorghum, sesame, and finger millet but have since branched out to vegetables, coffee, groundnuts and sugarcane. Gill nets and long-line fishing are used to catch tilapia and other fish[83]. The same individuals, in different seasons of the year, are foragers, herders, farmers, and industrialists. Throughout the years, the Luo have both specialized and generalized their labor[84].

Traditionally, the head of the Luo homestead had his hut located near the cattle enclosure, and it was the setting for important deliberations on family and the community among the elders of the tribe; wives had separate huts and did not sleep in the hut of the head of homestead. Traditionally, a young woman whose suitor had given her parents enough cattle would be forcefully carried off by the bridegroom and his friends. Today, however, bride prices are paid in cash rather than cattle and marriages often are formalized by Christian sacrament[83]. Using the state, church, and local self-help movements to their advantage, the Luo have become internationally recognized for scholarship and proficiency in the English language[84]. They are a sizeable but geographically minor and economically underprivileged ethnic group in Kenya[84–86].

Luo society is organized through patrilinealism, meaning that all children born to a couple are deemed members of the father's kinship group[83,84,87,88]. Thus, women, as

mothers, supply children to their husband's kinship line and not their own; brothers add their progeny to the family line rather than sisters. The community organization among the Luo is typical of an agricultural society which is primarily patrilineal. In an agricultural society, production of food, especially when requiring large animals for plowing, is dominated by the males. Women play a smaller role in the food growing activities[87]. However, while women's roles in reproduction, organization, and production are not explicitly recognized in public, they do play an important role in the day-to-day activities of the community[84,89,90]. Seniority and sequence are vital aspects of family, lineage, and clan life. Performing tasks out of chronological or specified order brings upsetting spiritual and metaphysical threats to family, lineage, and community that must be addressed ritualistically. The power and prestige of the elders, while challenged by outspoken youths and changing socioeconomic times, are still present today[84]. Burial of the dead occurs within the homestead, and graves provide critical permanent fixtures on the landscape for reckoning of personal, familial, and political identities and allegiances[84,89].

From migrating to a different area to changing the economic activities of the community to the unsettling politics of the nation, the Luo people have been subjected to various stressors in their lengthy history. A stressor is defined as an external stimulus that elicits a defense response. Thus acute stress is the result of changes which disrupt the order of a person's life suddenly or severely, and chronic stress is the pressures which impinge on individuals daily or at consistent intervals[67]. One stressor which chronically plagues Sub-Saharan Africa is the HIV/AIDS virus. In fact, 70% of people worldwide infected with the HIV/AIDS virus are located in Sub-Saharan Africa[91]. In 1997, it was

estimated that 240,000 people had developed AIDS within Kenya alone since the virus's first appearance in 1984. Approximately one in eight adults in rural Kenya is infected with HIV primarily contracted through heterosexual contact[88]. 30% of children born to HIV positive mothers contract the disease during pregnancy or the birthing process, and the other 70% are at risk for becoming orphans[88]. Sub-Saharan Africa leads the world in HIV-related deaths, AIDS orphans, and number of infected women[91]. Sexual behavior has been deemed more perilous in rural areas as women from the countryside less frequently reported being a virgin at marriage, had a higher number of lifetime partners, and reported inconsistent condom use with nonspousal sexual partners[92]. Additionally, upon being informed of the risks of their sexual behaviors, 23% of women and 11% of men in rural areas made no change to their lifestyles compared to 16% of women and 7% of men in urban areas[88]. Among all the ethnic groups in Sub-Saharan Africa, the Luo of the Nyanza province have the highest prevalence of HIV at 30%[91]. The HIV/AIDS virus has implications beyond the actual destructive power of the disease: its effects reach throughout the entire community. One study found that children born to HIV positive parents, along with orphans and foster children, were significantly less likely to attend school than their counterparts. Furthermore, children of HIV-infected parents were more often underweight and wasted and received insufficient or inconsistent medical treatment[93]. Additionally, as the virus spreads across the African continent, elders are becoming increasingly responsible for the care of children made orphans by its ravages. These well-meaning elders do not have the proper resources to care for the children and are at risk for poor health themselves. One study found that while care-giving does not

directly affect physical well-being, it did act to decrease the mental health and the perceived health of the elders[94].

Education also plays a vast role in the community. Educated mothers have been shown to provide more efficiently for children under the age of five when it comes to infectious diseases such as typhoid, malaria, and influenza[95,96]. Education did not, however, protect against sexually transmitted infections or HIV/AIDS. One study revealed that most educated mothers within urban settings in Kenya experience socio-cultural and religious inhibitions which hindered them from providing meaningful sex – education to their pre-adolescent and adolescent daughters[96]. Education is also the single most important factor in determining marital age. Women with higher levels of education have a tendency to postpone marriage in favor of educational and career pursuits, and women who marry at a later age, while reducing the number of years of which they are available for childbearing, are spared the perils of early marriage. Early marriage is associated with early childbearing and the substantial health risks for both the mother and child which accompany it. Furthermore, these complications are more likely to arise when mothers are young and less equipped to deal with them, leading more frequently to maternal death. Changes within society have also changed the ways in which childbearing and education have been perceived. Traditionally, women gained status and power within the household and society by marrying and bearing children. Today, however, many women find respect and opportunity through education regardless of their marital status or whether or not they have children. Additionally, place of residence contributes to the determination of whether a woman will marry early or not. Rural areas tend to have institutional and normative structures such as kinship groups and

the community as a whole which promote early marriage and childbearing[97]. There exists a negative correlation between education and family size in Kenya. In order to receive income returns from educational investments in their children, Kenyan parents favor those children most likely to pursue higher education, typically the first born[98].

Child malnutrition is also a problem in Western Kenya. One study[99] found that 38% of children under the age of five in the community sample were malnourished and weighed 89% or less of the standard weight for their age. The most common causes of malnourishment were poor feeding and sickness. Mothers of malnourished children were likely to blame sickness while perceiving malnourished children of other women as underfed. Of mothers from Luo-speaking regions, 37% had co-wives, and of all mothers from the community sample, 47% reported their husbands as absent. From conversations with the mothers, the researchers found that the children became malnourished in the context of marital conflicts: many of the married women were cooperating poorly with their husbands, and some of the women left home for periods of time as a result of conflict and their child became sickly while in the care of another. Additionally, in some relationships, the husband devoted care and resources to another of his wives and her family[99].

Stability of marriage is one of the most striking features of the rural Luo community, yet discussions of marital problems are endemic[100], and the children are affected by their parent's marital and socioeconomic circumstances[101]. Children of divorced or never-married women have significantly higher probabilities of dropout before completion of the polio vaccine series and acute undernourishment than children of monogamously married mothers. Furthermore, the number of male household

members of working age greatly enhances the probability of full immunization and nourishment status of the children whose mothers are divorced or previously married. Studies show that while children of fathers married to more than one women are not typically nutritionally disadvantaged, polygyny is associated with higher polio dropout rates and a lower probability of full immunization than monogamous relationships[101].

Investigations into agricultural nutrition have uncovered the unprofitability of small, rural farms. The intensity of family labor on small household farms drives prices down, falling below zero for the smallest farms[102]. Additionally, families with the most narrow resources have the lowest food production for familial consumption and lowest average energy intake. These families also had the lowest food intake levels among young children and displayed higher incidence of stunting when compared with children belonging to families with a greater number of resources in their possession[103].

The Luo people of Western Kenya lead lives plagued by stressors. They face the perils of malnourishment and infectious diseases such as malaria, typhoid, and the HIV/AIDS virus. They live with the pain of lost loved ones in tight-knit kinship groups. Furthermore, they live within a complex culture affecting every facet of their lives from education, to occupation and employment, to family size and marital status. Moreover, even under the pressures exerted by acute and chronic stressors, they have escaped, to a large extent, the plague of the modern world - obesity. They have not, however, evaded the ravages of type 2 diabetes. This study seeks to fill a gap in literature by investigating whether stressors can result in type 2 diabetes in lieu of obesity.

CHAPTER THREE

Hypotheses

Research Questions and Hypotheses

Primary Research Question

How does body mass index affect blood glucose levels among the Luo?

Hypothesis 1: Elevated blood glucose levels will be present among the Luo.

Null Hypothesis: There will be no elevated blood glucose levels among the Luo.

Hypothesis 2: Elevated blood glucose levels will be present among the non-obese Luo.

Null Hypothesis: There will be no elevated blood glucose levels among the non-obese Luo.

Hypothesis 3: Elevated blood glucose levels will not be related to body mass index (BMI) among the Luo.

Null Hypothesis: The obese will have a statistically significantly higher proportion of elevated blood glucose levels than the moderate and thin BMI Luo.

Secondary Research Question

Among the non-obese Luo in western Kenya, how do blood glucose levels compare in persons experiencing high levels of stressors with those experiencing low levels of stressors?

Hypothesis 4: Non-obese persons experiencing high levels of stressors will have significantly higher blood glucose levels than persons experiencing low levels of stressors.

Null Hypothesis: Among non-obese Luo in western Kenya, those persons experiencing high levels of stressors will manifest no significantly different levels of blood glucose than those persons experiencing low levels of stressors.

Theoretical Path Model Underlying Hypotheses

This study investigates the role that stressors, such as family history of death and disease, family size, home location, marital status, ages of children, education level, frequency and variety of meals, and employment status, play in raising blood glucose levels, a surrogate measure of Type 2 Diabetes, in the absence of excess body fat and a high body mass index that traditionally denotes obesity. Data was collected from patients seeking medical treatment at a temporary clinic in rural western Kenya in May of 2010. The stressors were self-reported by the patients.

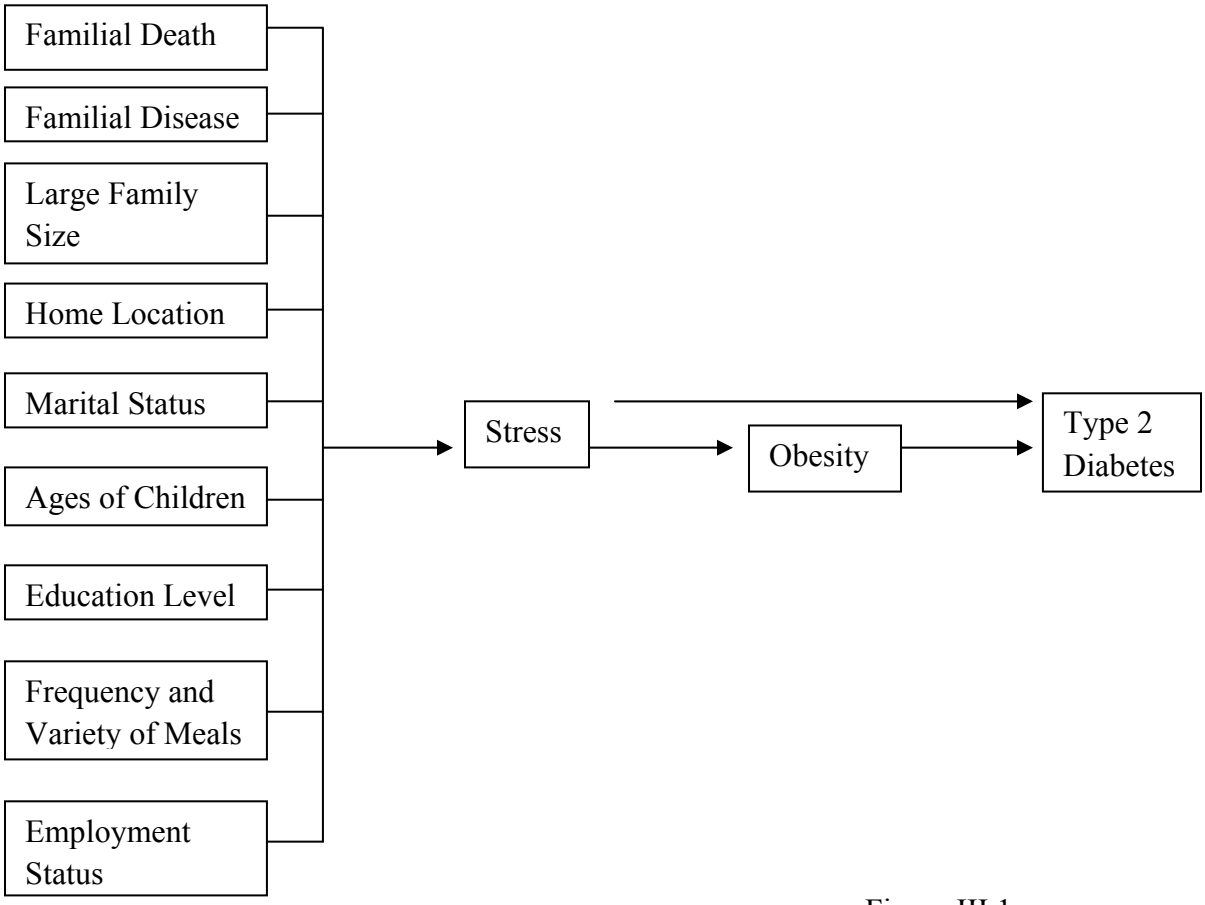

Figure III.1

CHAPTER FOUR

Methods

Study Area and Population

This study analyzed clinical data collected in the 14 day period of May 15 through May 29, 2010, from a single population of subjects. The clinic at which the data were collected was located in the Upper Nyakach Division of the Nyando District in western Kenya, approximately 36 km southeast of Kisumu and 12 km southeast of Lake Victoria at a latitude of $0^{o}21'23''$ S and an elevation of 1550 m.

The clinic attendees were primarily subsistence farmers and pastoral herders and their families who had homesteads within walking distance of the clinic. Additionally, almost all patients belonged to the Luo ethnic group.

Analyzing these data collected for clinical purposes was approved by the Institutional Review Board of Baylor University.

Measurements

A single venous blood sample was collected for each patient and then blood glucose levels were measured using the Chem-8 analysis and the i-STAT handheld device manufactured by Abott. A drop of blood was applied to the self-contained cartridge which was then inserted into the handheld i-STAT machine. The test analysis initiated automatically. The cartridge specifically designed for determining the blood glucose levels was the i-STAT G 06F09-01. Glucose levels reported by the i-STAT machine were recorded on patient-specific data sheets.

Skin-fold measurements were taken with Lange calipers. The tester pinched the skin to raise a double layer of skin and the underlying adipose tissue, but not muscle tissue, and the calipers were applied one centimeter below and at right angles to the pinch. A reading in millimeters was recorded both thirty and sixty seconds later. The skin-fold measurements were taken on the triceps and the subscapular region of the back.

The anthropometric measurements taken were height, weight, waist circumference, and hip circumference for all subjects, in addition to head circumference for those subjects under one year of age. The height measurements were taken from a tape measure pinned to the laboratory wall. The patients stood with their backs against the tape measure and their height was recorded in centimeters. The weight measurements were taken in kilograms from a spring-scale purchased locally in Kisumu. The waist, hip, and head circumference measurements were all taken from a tape measure and recorded in centimeters. Waist, hip, and head circumference measurements were taken from an unpinned tape measure and were recorded in centimeters.

Study Design and Clinical Data

The data of this study were collected by laboratory technicians and trained volunteers. Diabetes, for these analyses, is defined as a random blood glucose level of 200 mg/dL or higher. Blood glucose levels in the range of 100 mg/dL to 125 mg/dL were an indication of pre-diabetes. If high blood glucose levels were reported for a patient, urinalysis was then conducted to determine the amount of glucose excreted with the urine.

In order to determine the level of stress experienced by the patient, the number and type of stressors were based on the patients' self-report. Stressors were chosen

specifically as those deemed to be most present within Luo society and of the most lasting effects: stressors relating to kinship, education, employment, homesteads, nutrition, disease, and death.

The body mass of each subject was determined using the equation: $BMI = \frac{Weight\ (kg)}{Height\ (m)^2}$. BMI of the population was divided into categories by two methods. The first method divided the population into four BMI groups based upon the World Health Organization's (WHO) classification of BMI as underweight, normal, overweight, and obese. However, as the population was primarily underweight, patients were also divided by WHO criteria into six groups of severely underweight, very underweight, underweight, normal, overweight and obese.

Analysis

All clinical data were coded and double-entered blinded into Microsoft Excel and then imported into SAS statistical software version 9.2 (English) for Windows Vista. All variables were checked for errors and corrected when necessary. Univariate, bivariate, and multivariate analyses were performed using SAS (Cary, North Carolina). Alpha (α) was set at 0.05.

Analytic Statistics

Contingency table analysis and analysis of variance (ANOVA) were performed to evaluate the relationship between various social and biological stressors and elevated blood glucose levels. Regression analysis was performed to assess CRP and acidity.

Institutional Review Board

This study was approved by the Baylor University Institutional Review Board (IRB). The data abstracted from the clinical record used no unique personal identifiers. The variables used for this study were the following: study ID, age, gender, height, weight, blood glucose levels, family history of malaria, typhoid, HIV, diabetes mellitus, and serious illness, familial death, employment status, number of people living in a common space, marital status, education level, and food type and amount of consumption.

Variables created for statistical measurements which utilize the variables described above are the following: body mass index, diagnosis of diabetes, and age groups divided into children and adults as well as children, middle aged, and elderly.

CHAPTER FIVE

Results

Organization of Results

The results of the study are organized into four sections with the first being a general overview. The second section focuses upon social stressors and their effects on presence or absence of diabetes while the third section is dedicated to the contributing medical stressors to diabetes. The last section summarizes the results as they pertain to the original hypotheses.

General Overview

Information regarding the correlation of glucose levels, as measured by I-Stat, a diagnosis of diabetes based on World Health Organization divisions of blood glucose levels, age, and gender is provided in Tables V.1-3. A total of 685 patients attended the clinic between May 15 and May 29, 2010. Patients were grouped according to age by two methods. The first method divided the population into two age groups: younger than 18 years and greater than or equal to 18 years of age. The second method divided the population into three age groups: younger than 17 years, 17 to 52 years old (52 years of age is the age of forced retirement for many jobs in Kenya), and older than 52 years of age. Patients were diagnosed as diabetic if their random blood glucose value was greater than 200 mg/dL. Table V.4-5 demonstrates the relationship between diabetes and body mass index. Patients were divided into body mass index groups by two approaches. The first approach divided the population into four BMI groups based upon the World Health

Organization's classification of BMI as underweight, normal, overweight, and obese. However, as the population was primarily underweight, patients were divided into six groups of severely underweight, very underweight, underweight, normal, overweight and obese.

		Gender		
		Female	Male	Total
Elevated Blood Glucose	Yes	8 (0.0223)	6 (0.0259)	14 (0.0237)
	No	351 (0.9777)	226 (0.9741)	577 (0.9763)
	Total	359 (0.6074)	232 (0.3926)	591 (1.00)
	χ^2= 0.0780, df= 1, p = 0.7800			

Table V.1

Table V.1 compares the patients determined to be diabetic (by having blood glucose levels equal to or greater than 200 mg/dL) by gender (χ^2 = 0.0780, p = 0.7800). There was no association between diabetes and gender.

		Age		
		Less than 18 years of age	Greater than or equal to 18 years of age	Total
Elevated Blood Glucose	Yes	4 (0.0133)	10 (0.0336)	14 (0.0234)
	No	296 (0.9867)	288 (0.9664)	584 (0.9766)
	Total	300 (0.5017)	298 (0.4983)	598 (1.00)
	χ^2= 2.6744, df= 1, p = 0.1020			

Table V.2

Table V.2 compares diabetes and by their age, sorting between patients who are under 18 years of age and those who are 18 years or older. Although not statistically significant, the older patients were more likely (0.0336) than the younger patients (0.0133) to have elevated blood glucose levels (χ^2 = 2.6744, df = 1, p =0.1020).

		Elevated Blood Glucose		
		Yes	No	Total
Age	<17 years	4 (0.0137)	287 (0.9863)	291 (0.4932)
	17-52 years	6 (0.0309)	188 (0.9691)	194 (0.3288)
	>52 years	4 (0.0381)	101 (0.9619)	105 (0.1780)
	Total	14 (0.0237)	576 (0.9763)	590 (1.00)
	χ^2= 2.6214, df= 2, p = 0.2696			

Table V.3

Table V.3 depicts the relationship between diabetes and age, categorized into three groups: under 17 years of age, 17 to 52 years of age, and greater than 52 years old. This age grouping also revealed no correlation with diabetes (χ^2 = 2.6214, df = 2, p = 0.2696) . Analysis of variance on continuous blood glucose levels and the three age group categories revealed similar results. Those patients under the age of 17 had a mean blood glucose level of 114.89 mg/dL. The 17 to 52 year old age group had a mean blood glucose level of 116.26 mg/dL. The over 52 years old group had a mean blood glucose

level of 120.24 mg/dL. While blood glucose levels were higher with each older age group, the increase was not statistically significant (r^2= 0.002, $F_{2,568}$= 0.57, p = 0.5658).

		BMI				
		Underweight <18.50	Normal 18.51-25.0	Overweight 25.01-30.0	Obese >30.0	Total
Elevated Blood Glucose	Yes	7 (0.0222)	6 (0.0290)	1 (0.0244)	0 (0.00)	14 (0.0244)
	No	309 (0.9778)	201 (0.9710)	40 (0.9756)	10 (1.00)	560 (0.9756)
	Total	316 (0.5505)	207 (0.3606)	41 (0.0714)	10 (0.0174)	574 (1.00)
	χ^2 = 0.5002, df = 3, p = 0.9188					

Table V.4

Table V.4 shows a comparison between body mass index as determined by the World Health Organization and divided into four groups, underweight, normal, overweight, and obese. The proportion of persons with elevated blood glucose was 0.022, 0.0290, and 0.0244 among the underweight, normal, and overweight BMI groups, respectively; all three of these proportions were very close to the overall proportion of persons with elevated blood glucose levels (0.0244). There were no persons with elevated blood glucose levels among the WHO categorization of obese – albeit that there

were only 10 obese patients in the sample. There clearly was no association between BMI and elevated blood glucose levels ($\chi^2 = 0.5002$, df = 3, p = 0.9188).

		Body Mass Index						
		Severely Underweight <16.0	Very Underweight 16.01-17.0	Underweight 17.01-18.50	Normal 18.51-25.0	Overweight 25.1-30.0	Obese >30.0	Total
Elevated Blood Glucose	Yes	4 (0.0203)	0 (0)	3 (0.0405)	6 (0.0290)	1 (0.0244)	0 (0)	14 (0.0204)
	No	193 (0.9797)	45 (100)	71 (0.9595)	201 (0.9710)	40 (0.9756)	10 (100)	560 (0.9756)
	Total	197 (0.3432)	45 (0.078)	74 (0.1289)	207 (0.3606)	41 (0.0714)	10 (0.017)	574 (1.00)
	$\chi^2 = 2.5080$, df = 5, p = 0.7753							

Table V.5

Table V.5 depicts the relationship between elevated blood glucose and further division of body mass index into the six groups labeled by the WHO as severely underweight, very underweight, underweight, normal, overweight, and obese. Again, elevated blood glucose varied little by BMI ($\chi^2 = 2.5080$, df = 5, p = 0.7753). It is crucial to note that the severely underweight had approximately the same proportion (0.0203) of elevated blood glucose as those in the overweight BMI groups (0.0244). As BMI is an accepted measure for obesity, this finding supports the hypothesis that, among Luo in this sample, elevated blood glucose was present in the absence of obesity.

Employment and Education

		Employed		
		No	Yes	Total
Elevated Blood Glucose	Yes	13 (0.0248)	1 (0.0132)	14 (0.0233)
	No	511 (0.9752)	75 (0.9868)	586 (0.9767)
	Total	524 (0.8733)	76 (0.1267)	600 (1.00)
	$\chi^2 = 0.3954$, df = 1, p = 0.5295			

Table V.6

Table V.6 represents the relationship between employment in the family and diabetes. First of all, it is pertinent that 87.33% of the patients seen at the clinic were members of families in which no one had employment (self, spouse, brothers, sisters, parents, or children). This may be due to the selection bias that employed persons could not take off the time to attend the clinic. Approximately 2.5% of the unemployed had elevated blood glucose levels, compared to 1.3% of those from employed families ($\chi^2 = 0.3954$, df = 1, p = 0.5295). Analysis of variance revealed the mean blood glucose level for those patients who were employed to be 119.08 mg/dL while the mean blood glucose

level for those who were unemployed was 115.73 mg/dL (r^2= 0.000676, $F_{1,577}$= 0.39, p = 0.5324).

Division of the population into two age groups, under 18 years and 18 years or older, revealed that among adults mean blood glucose levels were lower among the unemployed (116.32 mg/dL versus 126.33 mg/dL) (r^2= 0.004729, $F_{1,277}$= 1.32, p = 0.2523), although this difference was not statistically significant.

		Education			
		No School	Primary School	Secondary School and Higher	Total
Elevated Blood Glucose	Yes	5 (0.0182)	7 (0.0294)	2 (0.0227)	14 (0.0233)
	No	269 (0.9818)	231 (0.9706)	86 (0.9773)	586 (0.9767)
	Total	274 (0.4567)	238 (0.3967)	88 (0.1467)	600 (1.00)
	χ^2 = 0.0698, df = 2, p = 0.7053				

Table V.7

Table V.7 depicts the relationship between diabetes and education level with the lowest education level being no education at all, the middle education level being primary level, and the highest education level encompassing both secondary education and

college or technical school. There were no significant differences in the proportion of elevated glucose levels across education levels (χ^2 = 0.0698, df = 2, p = 0.7053).

Food Types and Meals

Elevated blood glucose was compared with the types of food consumed by the patients: porridge, fruit, vegetables, milk, and meat. The patient responded how often a week each food group was consumed, with frequency ranging from once per week to five times per week. There was no association between elevated blood glucose levels and: porridge consumption (χ^2 = 1.73, df = 1, p = 0.7861); fruit consumption (χ^2 = 4.48, df = 1, p = 0.3455); vegetable consumption (χ^2 = 0.2022, df = 1, p = 0.9952); milk consumption (χ^2 = 1.68, df = 1, p = 0.7948); or, meat consumption (χ^2 = 4.00, df = 1, p = 0.4054). Patients also reported how many meals per day they consumed, on a scale from 1 to 7. Low meat consumption paired with high porridge consumption for one or two meals per day was not associated with elevated blood glucose (χ^2 = 0.1279, df = 1, p = 0.7207). Analysis of variance comparing continuous blood glucose levels with amount of meat consumption revealed that the highest blood glucose levels were seen among those patients consuming the most meat per week (r^2= 0.0145, $F_{4,528}$= 1.94, p = 0.1019). This supports the biological theory of higher fat consumption contributing to higher blood glucose levels. Comparing elevated blood glucose levels among patients who reported eating one, two, or three meals a day showed no relation (χ^2 = 0.4971, df = 2, p = 0.7799). Elevated blood glucose levels comparing persons who ate only one meal per day with those eating two or more meals revealed no association (χ^2 = 0.2747, df = 1, p = 0.6002). Overall, no association was found between amount or type of food consumption and the presence of elevated blood glucose levels.

Relationship Stressors

Table V.8 shows the relationship between marital status among patients and diabetes. The unmarried were slightly more likely to have elevated blood glucose levels than those who were married (5% vs. 3.6%; $\chi^2 = 0.1043$, df = 1, p = 0.7468), but the differences were not statistically significant.

		Married		
		Yes	No	Total
Elevated Blood Glucose	Yes	9 (0.0359)	1 (0.0500)	10 (0.0369)
	No	242 (0.9641)	19 (0.9500)	261 (0.9631)
	Total	251 (0.9262)	20 (0.0738)	271 (1.00)
$\chi^2 = 0.1043$, df = 1, p = 0.7468				

Table V.8

Analysis of variance comparing blood glucose levels and marital status showed relatively equal mean values for blood glucose levels among married and unmarried patients. Unmarried patients had a mean blood glucose level of 118.60 mg/dL while married patients had a mean blood glucose level of 117.73 mg/dL (r^2= 0.000018, $F_{1,269}$= 0.00, p = 0.9438).

Analysis of the variables for the number of people living in the patient's home and the age of the patient revealed the two variables to be unrelated (data not shown). Analysis of variance of the house number and blood glucose levels revealed that elevated blood glucose was not related to the number of people living in a common space (data not shown).

Table V.9 depicts the lack of a relationship between stress caused by familial death and elevated blood glucose. A death in the family in the past year was compared to persons with no deaths in the family; there was no difference in the proportions of elevated blood glucose between the two groups ($\chi^2 = 0.0091$, df = 1, p = 0.9241).

		Familial Death		
		Yes	No	Total
Elevated Blood Glucose	Yes	1 (0.0164)	7 (0.0181)	8 (0.0179)
	No	60 (0.9836)	379 (0.9819)	439 (0.9821)
	Total	61 (0.1365)	386 (0.8635)	447 (1.00)
$\chi^2 = 0.0091$, df = 1, p = 0.9241				

Table V.9

Analysis of variance with blood glucose levels and stress caused by familial death revealed that patients who had deceased family members had a slightly lower mean blood

glucose level: 114.56 mg/dL for those with familial deaths compared with 116.32 mg/dL among those with no familial deaths (r^2= 0.000186, $F_{1,430}$= 0.08, p = 0.7773). When analysis of variance was performed on blood glucose levels and stress caused by familial death adjusting for age of the sample in three age groups, it revealed that, among the youngest age group, mean blood glucose levels were higher among those experiencing familial death (120.66 mg/dL versus 113.95 mg/dL) (r^2= 0.006971, $F_{1,196}$= 1.38, p = 0.2422). For the middle age group, mean blood glucose levels were lower among patients who had deceased family members (105.53 mg/dL versus 116.16 mg/dL) (r^2= 0.005699, $F_{1,147}$= 0.84, p = 0.3602). Additionally, among the elderly, mean blood glucose levels were also lower among patients with death in the family (111.22 mg/dL versus 121.92 mg/dL) (r^2= 0.002537, $F_{1,81}$= 0.021, p = 0.6511). None of these differences were statistically significant.

HIV/AIDS

		Age		
		<18 years old	18 years and older	Total
HIV Positive	Yes	0 (0.00)	9 (0.0346)	9 (0.0182)
	No	234 (1.00)	251 (0.9654)	485 (0.9818)
	Total	234 (0.4734)	260 (0.5263)	494 (1.00)
	$\chi^2 = 8.25$, df = 1, p =0.0041			

Table V.10

Table V.10 compares the patient's HIV status and age, which is divided into two groups: children and adolescents in one group and adults in another. None of the children or adolescents were HIV positive, while 3.5% of the adults were ($\chi^2 = 8.25$, df = 1, p = 0.0041).

		Elevated Blood Glucose		
		Yes	No	Total
Patient HIV Status	Positive	1 (0.1111)	8 (0.8889)	9 (0.0364)
	Negative	5 (0.0210)	233 (0.9790)	238 (0.9636)
	Total	6 (0.0243)	241 (0.9757)	247 (1.00)
	$\chi^2 = 2.97$, df = 1, p =0.0848			

Table V.11

When patient HIV status among adults was compared with elevated blood glucose levels, patients with HIV were more likely to have elevated blood glucose (11.11 % vs. 2.10 %; $\chi^2 = 2.97$, df =1, p = 0.0848) (Table V.11). Additionally, comparing the HIV status of the spouse of the patient with elevated blood glucose levels revealed that HIV positive-spouse status was positively associated with elevated blood glucose (12.50 % vs. 2.09 %; $\chi^2 = 3.54$, df = 1, p = 0.0600) (Table V.12).

		Elevated Blood Glucose		
		Yes	No	Total
Spouse HIV Status	Positive	1 (0.1250)	7 (0.8750)	8 (0.0324)
	Negative	5 (0.0209)	234 (0.9791)	239 (0.9676)
	Total	6 (0.0243)	241 (0.9757)	247 (1.00)
$\chi^2 = 3.54$, df = 1, p =0.0600				

Table V.12

Diabetes Symptoms

When elevated blood glucose was compared with symptoms of diabetes such as inappropriate hunger and thirst, frequent urination, and a diagnosis of neuropathy, it was revealed that while the adults experienced some of the symptoms of diabetes ($\chi^2 = 3.20$, df = 1, p = 0.0737), the younger age group did not ($\chi^2 = 0.0552$, df = 1, p = 0.8143). There was no association between inappropriate hunger and thirst or frequent urination and diabetes for both age groups (data not shown). Additionally there was no association between diabetes and a diagnosis of neuropathy for the children's age group (data not shown). However, among the adult age group, the proportion of patients with elevated blood glucose with a diagnosis of neuropathy was 50%, compared to 3.05% among those without elevated blood glucose ($\chi^2 = 13.46$, df = 1, p = 0.0002).

Diagnosis groups

Diagnoses were originally grouped based on whether or not they were chronic. The chronic diagnoses were further divided into stable and deteriorating. Among the stable diagnoses were musculoskeletal diseases and non-musculoskeletal diseases. The musculoskeletal diagnoses included arthritis, back pain, generalized pain, pain on the right side of the body, and chest pain. When chronic musculoskeletal diagnoses were compared with elevated blood glucose levels, there was no association ($\chi^2 = 0.0117$, df = 1, p = 0.9137); this lack of association also held among the youth and the adults. The non-musculoskeletal diagnoses were gastroesophageal reflux, cerebral palsy, dysmenorrhea, endometriosis, heavy menstrual periods, neurofibromatosis, thyroid dysfunction, seizures, fatigue, ringworm, fungus, Bell's palsy, cleft lip, microcephaly, hydrocephaly, unspecified rash, headache, malnutrition, and chronic abdominal pain. When chronic non-musculoskeletal diagnoses were compared with elevated blood glucose, there was no association among the adults ($\chi^2 = 0.0636$, df = 1, p = 0.8009). However, among the children 75% of those with elevated blood glucose had chronic non-musculoskeletal diagnoses, with a relative risk of 7.6 ($\chi^2 = 4.39$, df = 1, p = 0.0362).

The deteriorating diagnoses were divided into cardiovascular and non-cardiovascular. The chronic cardiovascular diagnoses were angina, cardiovascular chest pain, hypertension, congestive heart failure, and pitting edema. Of those with a cardiovascular chronic disease, 8.45 % had elevated blood glucose levels, compared to 1.57 % of those without a cardiovascular disease having elevated blood glucose levels (Relative Risk = 5.4, $\chi^2 = 12.48$, df = 1, p = 0.0004).

		Elevated Blood Glucose		
		Yes	No	Total
Chronic Cardiovascular Disease	Yes	6 (0.0833)	66 (0.9167)	72 (0.2408)
	No	4 (0.0176)	223 (0.9824)	227 (0.7592)
	Total	10 (0.0334)	289 (0.9666)	299 (1.0000)
$\chi^2 = 7.3017$, df = 1, p =0.0069				

Table V.13

The chronic non-cardiovascular diagnoses included chronic cough, possible cancer, kidney disease, developmental delay, failure to thrive, diagnosis of hypotonia, poor feeding, diagnosis of neuropathy, goiter, HIV, possible HIV, tuberculosis, autoimmune disease, leiomyoma, diagnosis of chronic diarrhea, and diagnosis of chronic vomiting. Comparison of chronic non-cardiovascular diagnoses with elevated levels of blood glucose revealed a non-significant increased risk of elevated blood glucose levels for patients suffering from chronic non-cardiovascular diagnoses (overall relative risk = 2.85, $\chi^2 = 2.11$, df = 1, p = 0.1465).

Elevated blood glucose was also compared with malaria. Among the entire sample, the relative risk was 2.28, indicating that people with malaria were approximately two times as likely to have elevated blood glucose ($\chi^2 = 2.51$, df = 1, p =

0.1133). Among the children, the comparison of malaria and diabetes elevated blood glucose found that all of the children with blood glucose greater than 200 mg/dL had malaria; 4 of the 141 (2.84 %) youth with malaria had elevated blood glucose levels (χ^2 = 4.51, df = 1, p = 0.0336). Among the adults, the proportion of those with malaria who had elevated blood glucose levels was 5.63 %, compared to 2.88 % of those without malaria (χ^2 = 1.12, df = 1, p = 0.2894). This is most likely stress hyperglycemia due to severe illness. However, infection can precipitate type 2 diabetes, so a high level of blood glucose during severe malaria may be both an indication of physiological stress and also the herald of type 2 diabetes.

A comparison of acute illnesses with diabetes indicated that acute illness and elevated blood glucose were not related for the overall sample (χ^2 = 0.5376, df = 1, p = 0.4634), the children (χ^2 = 0.4539, df = 1, p = 0.5005), or the adults (χ^2 = 0.3474, df = 1, p = 0.5556).

Finally, a diagnosis of worm infection was compared with elevated blood glucose. There was no association between a diagnosis of worms and elevated blood glucose among the entire sample (χ^2 = 1.21, df = 1, p = 0.2710), the children (χ^2 = 0.1607, df = 1, p = 0.6886), or the adults (χ^2 = 0.1158, df = 1, p = 0.7337).

CRP

Analysis of variance of C-reactive protein (CRP) and elevated blood glucose revealed that the mean value of CRP was significantly higher for those with elevated blood glucose than those without elevated blood glucose, 53.79 mg/dL versus 14.48 mg/dL, respectively (r^2= 0.029892, $F_{1,580}$= 17.87, p = 0.0001).

Regression analysis for CRP on blood glucose levels and malaria is found in Table V.14. Malaria was the major determinant of CRP levels (incremental r^2= 0.1238, p = 0.0001); blood glucose levels did contribute to the overall explained variance (incremental r^2= 0.0185, p = 0.0067).

C-reactive Protein Regressed On:						
Variable	Variable Coefficient	Standard Error	t-value	p-value	Incremental Adjusted r^2	Total Adjusted r^2
Intercept	-3.2675	3.8440	-0.85	0.3957		
Malaria	24.9062	2.7485	9.06	0.0001	0.1238	0.1238
Elevated Blood Glucose	0.0835	0.0307	2.72	0.0067	0.0185	0.1423

Table V.14

Hypertension

Comparison of hypertension with elevated blood glucose (Table V.15) was statistically significant among adults (χ^2 = 9.47, df = 1, p = 0.0021). Among those with hypertension, 9.8 % had elevated blood glucose levels; among the non-hypertensives, 1.75 % had elevated blood glucose levels. However, this association may be attributed to the fact that hypertension is a potential outcome of diabetes.

		Hypertension		
		Yes	No	Total
Elevated Blood Glucose	Yes	6 (0.0984)	4 (0.0175)	10 (0.0345)
	No	55 (0.9016)	225 (0.9825)	280 (0.9655)
	Total	61 (0.2103)	229 (0.7897)	290 (1.00)
$\chi^2 = 9.47$, df = 1, p = 0.0021				

Table V.15

Acidity

Comparison of blood acidity levels with elevated blood glucose showed no association for the entire sample ($\chi^2 = 0.0669$, df = 1, p = 0.9671). Comparison of acidity and elevated blood glucose stratified by age resulted in no association among the adults ($\chi^2 = 0.8974$, df = 1, p = 0.6385) or among the children ($\chi^2 = 1.3087$, df = 1, p = 0.5198). Analysis of variance further confirmed the conclusion of no relationship: the mean CO_2 level for those without elevated blood glucose was 23.94 mEq/L whereas it was 22.77 mEq/L for those with elevated blood glucose ($r^2 = 0.003547$, $F_{1,520} = 1.85$, p = 0.1742).

Synopsis

The majority of the potential social stressors (unemployment, education, types of food and amount consumed, marital status, number of people living in home, and familial death) had no association with elevated blood glucose levels. HIV status of the patient and HIV status of a spouse were associated with elevated blood glucose levels. Neuropathy in adults, non-musculoskeletal chronic disease among the youth, malaria among youth, hypertension among adults, and CRP in the total sample were medical conditions associated with elevated blood glucose levels.

Data Results as Predicted by Hypotheses

Hypothesis 1: Elevated blood glucose levels will be present among the Luo

Null Hypothesis: There will be no elevated blood glucose levels among the Luo.

Tables V.1, V.2, and V.3 display the results relevant to this hypothesis. There were patients with elevated blood glucose levels among the Luo, a people known to have a small proportion of obesity, stratified across age and gender.

Hypothesis 2: Elevated blood glucose levels will be present among the non-obese Luo.

Null Hypothesis: There will be no elevated blood glucose levels among the non-obese Luo.

Tables V.4 and V.5 show that there were Luo individuals with elevated blood glucose levels among the underweight and severely underweight BMI groups.

Hypothesis 3: Elevated blood glucose levels will not be related to body mass index (BMI) among the Luo.

Null Hypothesis: The obese will have a statistically significantly higher proportion of elevated blood glucose levels than the moderate and thin BMI Luo.

Tables V.4 and V.5 make it clear that elevated blood glucose levels were not related to BMI. The proportion of persons with elevated levels of blood glucose among the underweight (0.02222 from Table V.4) and the severely underweight (0.0203 from Table V.5) are very similar to the proportion of the overall sample.

Four of the diabetics were classified as severely underweight, three as underweight, six as normal, and one as overweight. Thus, among the Luo sample, there exist non-obese persons with elevated blood glucose levels.

Hypothesis 4: Non-obese persons experiencing high levels of stressors will have significantly higher blood glucose levels than persons experiencing low levels of stressors.

Null Hypothesis: Among non-obese Luo in western Kenya, those persons experiencing high levels of stressors will not manifest significantly different levels of blood glucose from those persons experiencing low levels of stressors.

The results of the comparison of elevated blood glucose and the social stressor did not support the hypothesis; the null hypothesis could not be refuted. In some instances, what the researchers hypothesized to be stressors appeared to be protective against diabetes. Such was the case with HIV status of the patient, HIV status of the spouse, and familial death. Furthermore, no association was found for type and amount of food consumed and elevated blood glucose levels; the same lack of association was found with

various biological disorders. The study revealed that risk of elevated blood glucose increased in malaria patients and those patients with elevated levels of blood glucose had higher levels of inflammation as measured by C-reactive protein. Further investigation into hyperglycemia in rural Kenya may reveal that it has its origin in critical illness.

One limitation of the study concerned a question on the patient intake form. This question asked the patient if they were fasting at the time (i.e. had consumed nothing since awaking that morning). However, in addition to this question being a difficult one to translate and convey in the Luo language, into the week in which the research was conducted, it became apparent that the Luo people drink tea with sugar in the morning. As the translation of the fasting question typically resulted in the translators asking the patients whether they had eaten, patients most likely reported that they were fasting even if they had had tea with sugar. Thus, it was decided to conservatively treat everyone as non-fasting and the benchmark for elevated blood glucose levels was set at 200 mg/dL.

CHAPTER SIX

Discussion and Conclusion

Overview

The purpose of this study was to fill in the gap in the understanding of the development of elevated blood glucose. While the role which obesity plays in the development of type 2 diabetes has been well-documented and researched, there has been relatively little investigation into the role in which stressors and the stress response contribute to the disease development. It has been suggested that stress – whether biological, metabolic, or social in origin – can result in diabetes.

The physical examination and diagnosis of each patient attending the clinic were performed by a single physician trained and board certified in the United States. There was potential sampling bias in that the sample represents only a proportion of those who are ill in the community. Patients must have been well enough to travel to the clinic, aware of the clinic, and willing to wait eight or more hours in a line outside the clinic to be examined by the physician.

This study found body mass index, an acceptable measure for obesity, and blood glucose levels to be unrelated. This is contrary to the popularly accepted notion that obesity is the cause of diabetes. However, among the Luo people of western Kenya, the high blood glucose levels, and thus diabetes, observed in the general population is not a result of increased body fat. As expected, blood glucose levels and diabetes were independent of gender. However, they were also independent of age.

All potential social stressors were found to be unrelated to diabetes except for HIV status among patients and spouses. Contrary to expectation, having no or limited education did not result in higher blood glucose levels. All analyses of the type and amount of food consumption failed to reach statistical significance and showed no real difference between the blood glucose levels of those eating more nutritionally well-rounded meals or more meals and those who ate less of a variety of food or fewer meals altogether. Analysis confirmed the biological idea that consumption of foods higher in fat results in an elevated blood glucose level as opposed to consumption of lower amounts of fat, but the relationship was not statistically significant. Furthermore, what were considered as relationship burdens had no effect upon blood glucose levels. In fact, individuals who were not married exhibited higher blood glucose levels than those who were married. Perhaps the truly stressful situation is the former in which there is no partner to help lessen the load and burdens of life. In the United States, non-married men and men with older wives have a lower life expectancy whereas married women have a lower life expectancy[104]. Additionally, no difference was observed between the number of people sharing a living space in the homes of persons with elevated blood glucose and those with non-elevated levels. Furthermore, familial death did not seem to cause significant stressful chronic rises in blood glucose levels. In fact, the analyses suggested that those individuals who have experienced a death within their family actually have lower blood glucose levels than those who have not had a family member die.

Positive HIV status of patients and their spouses was associated with elevated blood glucose levels. Whether this exposure should be called social stressors or biologic

stressors or both is beyond the scope of this study. Clearly, HIV status was related in this sample to elevated blood glucose levels.

Upon analysis of the identified biological stressors, many resulted in no association between their manifestations and diabetes. There was no identifiable relationship between chronic musculoskeletal diagnoses and elevated blood glucose levels, nor was there a relationship between chronic non-musculoskeletal diagnoses and diabetes. Among the children, however, it was discovered that three out of four persons with elevated levels of blood glucose were diagnosed with chronic non-musculoskeletal diseases: seizures, ringworm, and fungal rash. There was a positive association between elevated blood glucose and chronic cardiovascular disease among adults; however, while it is to be expected, the association does not necessarily provide insight into the manifestation of diabetes. There was no association between the diagnosis of chronic non-cardiovascular disease and elevated blood glucose levels. Finally, analysis of acute illness and worm infection revealed that neither was associated with increased blood glucose levels.

Adult patients diagnosed with malaria were approximately two times as likely to be hyperglycemic as those not diagnosed with malaria, although this relationship was not statistically significant. Among the youth, all four patients with hyperglycemia had malaria and their blood glucose levels were raised as a result of their sickness ($p = 0.0336$). This was further affirmed when comparison of elevated blood glucose levels with the *symptoms* of diabetes showed a slight, though statistically insignificant, correlation among adults but no correlation at all among the children. The children were not experiencing any of the symptoms of diabetes.

Analysis of C-reactive protein revealed that those with elevated blood glucose levels in the study had significantly higher levels of CRP, a measure of high-grade inflammation, than did those without elevated blood glucose levels – even after adjusting for the effects of malaria. However, this increased inflammation may be the result of the additional sicknesses contracted by those with hyperglycemia.

A positive and statistically significant correlation was found between hypertension and diabetes among the adult patients. This association was most likely due to hypertension's role as an outcome of diabetes.

Overall, this study was successful in finding a population in which blood glucose levels were not correlated with obesity. However, no social stressors except HIV status could explain this association. Perhaps the most interesting discovery is the relationship between blood glucose levels, high grade inflammation, and biological stress. This may be the result of stress hyperglycemia, which is elevated blood glucose levels associated with critical illness. Stress hyperglycemia is the result of increased cortisol, catecholamine's, glucagon, growth hormone, gluconeogenesis, and glycogenolysis. Uncontrolled hyperglycemia is associated with poor outcomes in critically ill patients. Additionally, as insulin resistance is demonstrated in 80% of patients with stress hyperglycemia, it is possible that continued exposure to infection and critical illness could cause people to be in a continual state of stress hyperglycemia, effectively resulting in type 2 diabetes[105]. Further investigation into the relationship between malaria and other critical illnesses and elevated blood glucose levels would be meaningful research.

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