Insulin Resistance in Hispanic Male Meatpackers

Roberta Rita Martine
University of Colorado at Boulder, roberta.martine@colorado.edu

Follow this and additional works at: https://scholar.colorado.edu/anth_gradetds
Part of the Biological and Physical Anthropology Commons, and the Medicine and Health Commons

Recommended Citation
Martine, Roberta Rita, "Insulin Resistance in Hispanic Male Meatpackers" (2012). Anthropology Graduate Theses & Dissertations. 12.
https://scholar.colorado.edu/anth_gradetds/12

This Dissertation is brought to you for free and open access by Anthropology at CU Scholar. It has been accepted for inclusion in Anthropology Graduate Theses & Dissertations by an authorized administrator of CU Scholar. For more information, please contact cuscholaradmin@colorado.edu.
INSULIN RESISTANCE IN HISPANIC MALE MEATPACKERS

by

Roberta R. Martine

B.A., St. Mary of the Plains College, 1965
M.A., University of Colorado, Denver, 1982
M.S., University of Colorado, Boulder, 1988

A dissertation submitted to the
Faculty of the Graduate School of the
University of Colorado in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
Department of Anthropology
2012
This dissertation entitled:
Insulin Resistance in Hispanic Male Meatpackers
written by Roberta R. Martine
has been approved by the Department of Anthropology

___________________________________
Greg Kandt, Ed.D., Committee Chair

____________________________________
Matthew Sponheimer, Ph.D., Committee Member

Date ______________

The final copy of this dissertation has been examined by the signatories, and we find that both the content and the form meet acceptable presentation standards of scholarly work in the above mentioned discipline.

IRB protocol # 0409.1
Abstract

Martine, Roberta R. (Ph.D., Anthropology)

Insulin Resistance in Hispanic Male Meatpackers

Thesis directed by:

Dr. Greg Kandt, Associate Professor, Department Health & Human Performance, Fort Hays State
Dr. Hong Wang, Assistant Professor, Department of Endocrinology, CU Medical Center
Dr. Rodger Kram, Associate Professor, Department of Integrative Physiology, CU, Boulder

Insulin resistance is the first stage in the development of diabetes. This study attempted to determine whether the physical fitness acquired through on-the-job physical activity influences whether an individual becomes insulin resistant and exhibits high fasting insulin levels. The biomarkers of adiponectin and apoB were also studied. The study suggests that a meatpacker’s adiponectin level is directly related to his level of physical fitness. Similarly, the study has shown, that fasting insulin, and therefore, insulin resistance, is inversely related to the meatpacker’s level of physical fitness. For the studied population of male Hispanic meatpackers in Dodge City, Kansas, there seems to be no relationship with physical fitness and his apoB level. Within the population under study, apoB varied only within normal ranges, and, thus, was not as subject to the effects of physical fitness as were the other health markers. The study did not directly establish that the physical fitness exhibited by the studied meatpackers was a result of on-the-job activity. Physical fitness, as measured by this study, was shown to be related to the meatpacker’s BMI, age, and HDL. The meatpacker’s job category and the minutes he exercised outside of work each week were not shown to be statistically related to his physical fitness measurements.
Figures

Figure 1: Factors Contributing to Each Type of T2DM (adapted from Mc Carthy, 1993) .................................................9
Figure 2: Gut Parts by Species (Milton, 1999b) ........................................................................................................16
Figure 3: Progression of Glucose Intolerance (adapted from De Meyts, 1993: 98) .........................................................36
Figure 4: Proposed Mechanism for obesity-induced insulin resistance (adapted from Boden, 2001: 806) ........41
Figure 5: Outcomes that Depend on Where Fat is Stored (from Ravussin & Smith, 2002: 368) .........................46
Figure 6: Meatpacking Job Classification Form ........................................................................................................98
Figure 7: A1C for Normal Glucose Tolerant Individuals by Ethnic Group .................................................................109
TABLES

Table 1: Prevalence of Glucose Intolerance (W.Knowler, 1993: 205) .................................................................12
Table 2: Worldwide Prevalence of Diabetes ...........................................................................................................13
Table 3: Expected Worldwide Growth in Diabetes ..............................................................................................13
Table 4: Hominin brain size, body size, and tooth morphology (adapted from Leonard, 2003) ......................20
Table 5: Diets of Modern Hunter-Gatherers by Biome (Cordain, 2000) ...............................................................22
Table 6: Development of Agriculture (Simmons, 1996; Edens, 2000; Kelly & Thomas 2010) .......................23
Table 7: Prevalence of skeleton abnormalities pre- and post-transition to agriculture ........................................25
Table 8: Actions of Insulin (adapted from Stachura, 1996:8) ............................................................................32
Table 9: Adipose Tissue Proteins Affecting Metabolism (data from Kershaw, 2004) ....................................42
Table 10: Study Statistical Tests ........................................................................................................................66
Table 11: Meatpacking Jobs ................................................................................................................................71
Table 12: Kruskal-Wallis Results Comparing Blood Tests by Job Categories ....................................................72
Table 13: Wilcoxon Test Results Comparing Blood Tests for Light and Heavy Job Categories .................72
Table 14: Wilcoxon Results Comparing Blood Tests for Light and Moderate/Heavy Job Categories ..........72
Table 15: Spearman Correlation Coefficients and Probability Results Comparing Blood Tests for Job Categories ........................................................................................................................................72
Table 16: Percentile Values for Maximal Oxygen Uptake (ml/kg.min) in Men ..................................................73
Table 17: Kruskal-Wallis Results Comparing Blood Tests by Fitness Categories – Exercised No More Than 80 Minutes per Week .................................................................................................................74
Table 18: Wilcoxon Test Results Comparing Blood Tests for VO2-1 and VO2-3 – ........................................74
Table 19: Wilcoxon Results Comparing Blood Tests for VO2-1 and VO2-2&3 – .............................................74
Table 20: Spearman Correlation Coefficients and Probability Results Comparing Blood Tests for Fitness Categories – Exercised No More Than 80 Minutes per Week .........................................................74
Table 21: Kruskal-Wallis Results Comparing Blood Tests by Fitness Categories – All Participants ..............75
Table 22: Wilcoxon Test Results Comparing Blood Tests for VO2-1 and VO2-3 – ........................................75
Table 23: Wilcoxon Results Comparing Blood Tests for VO2-1 and VO2-2&3 – .............................................75
Table 24: Spearman Correlation Coefficients and Probability Results Comparing Blood Tests for Fitness Categories – All Participants ........................................................................................................................................75
Table 25: Variable Means, Medians, and Ranges .................................................................................................76
Table 26: Significant and Notable Spearman Correlation Analyses between Variable Pairs (N=35) ...............78
Table 27: Linear Regression Results for Rockport Assessment vs Fasting Insulin, Adiponectin, ApoB, Minutes Exercised, and Job Category .............................................................................................................83
Table 28: Notable and Significant Linear Regression Results for Triglycerides ................................................84
Table 29: Multiple Regression Models ................................................................................................................85
Chapter 1: Introduction
The work people do can affect their health. The research described here attempts to understand how the physical work and the challenges of physical fitness associated with meatpacking affect the pre-diabetic, insulin-resistant state of Hispanic male meatpackers. Male meatpackers are an ideal population for studying the effects of on-the-job activity because there is a wide variation in the level of physical fitness it takes to perform various categories of meatpacking jobs.

Besides studying how on-the-job activity affects physical fitness, this research attempts to add to our current understanding of how low physical fitness, in general, contributes to diabetes. Much of physical fitness is the result of physical activity. However, unlike physical activity, which is a measure of what people do, physical fitness measures a set of attributes that people have or achieve that give them the ability to perform physical activity (Caperson et al., 1985). According to the American College of Sports Medicine (ACSM) (2009), fitness is a measure of the ability to perform moderate to high intensity exercise for prolonged periods of time. This study measures cardiovascular physical fitness, which has been found to be stronger than self-reported physical activity as a predictor of many health outcomes (Blair et al., 2004).

In particular, this research attempts to quantify the relationship between a worker's insulin-resistance (phase 1 of type 2 diabetes) status and that worker's level of cardiovascular physical fitness (referred to only as “physical fitness” in the remainder of this dissertation). In addition, it measures how a worker's adiponectin (a hormone related to diabetes and obesity) level is influenced by his physical fitness level and compares the worker's apoB level with his fitness level. ApoB is a apolipoprotein that
increases with insulin-resistance and is a better measure of atherogenic risk than is total cholesterol or LDL cholesterol (Sniderman and Cianflone, 1996).

**STUDY HYPOTHESES**

This research tests the following hypotheses within the Hispanic male meatpacker population:

1. Insulin resistance is inversely related to the physical fitness resulting from job related activities.

2. Adiponectin level is positively related to the physical fitness resulting from job related activities.

3. ApoB level is inversely related to the physical fitness resulting from job related activities.

**SIGNIFICANCE OF STUDY**

The scientific significance of the study is related to the vocational character of its participants. Because this research studies men who are otherwise sedentary, the health effects of physical activity in the workplace can be relatively easy to isolate and measure. Using a vocational group (rather than free-living, community group) of participants whose jobs were studied for levels of physical activity may bring more accuracy to the study since, in theory, mismatches of physical exertion and job categories may be identified.

The author is not aware of any previous study of the relationship of physical fitness and fasting insulin levels within a vocational group like our meatpacking population. Much has been studied about the relationship between physical activity and fasting insulin
levels, and, hence, insulin resistance. Less has been done to reveal the relationship between physical fitness and insulin resistance. Adiponectin presents a hope for the control of obesity in the future (Li et al., 2009). One study of women who varied by age and obesity (Ryan et al., 2003) found a significant relationship between physical fitness (VO_{2max}) and adiponectin levels. Additional studies of the relationship of physical activity to adiponectin levels have yielded somewhat contradictory results. Some studies indicate that adiponectin levels increase with physical activity and physical fitness (Bouassida et al, 2010; Vu et al., 2007; Ring-Dimitriou et al., 2006; Kritetos et al., 2004); while others observe no change in adiponectin levels with increases in physical activity and physical fitness (Vu et al., 2007; Hara et al., 2005; Polak et al., 2006;Hulver et al., 2002). This study may help clarify this hormone's relationship with physical fitness.

Adding an assay of apoB to the study strengthens the indication of insulin resistance in study participants. This is because apoB measurements of greater than 1.2 g/l, accompanied by triglyceride measurements greater than 1.5 mmol/l, are thought to be caused by insulin resistance and abdominal/central obesity (de Graaf, Couture, and Sniderman, 2008). High apoB levels are also an early indicator of later heart disease. This study may help us understand the relationship of physical fitness to an individual's apoB level. If physical fitness influences apoB level, then this would be another indication of the importance of physical exercise in an individual's fight against heart disease.
The incidence of type 2 diabetes has increased significantly over the past few decades. Although the prevalence of the disease in the United States is currently at 9%, among the Hispanic population the prevalence is higher and stands at 12.1% (CDC, 2011). The consensus view is that obesity is a major culprit. As the world's food supplies are increasing and electronic devices, like televisions and washing machines, are making it possible for people to become physically inactive, the rate of obesity, and, therefore, diabetes has been increasing, even in developing countries. In addition, with obesity occurring within our populations earlier and earlier, type 2 diabetes is a prominent disease even among our children and adolescents (CDC, 2011).

All study participants receive their blood test results and diabetes prevention information as prepared by the National Diabetes Education Program. Therefore, just participating in the study may help to guide these men toward a healthier future.

Diabetes is an expensive disease. In a 2009 report drawn from 10 million United Health Care members, it was revealed that it cost $20,700 per year to treat each person with diabetes-related complications, compared to spending $4,400 on people who are diabetes free (Berkrot, 2010).
Chapter 2: Background Information
Diabetes mellitus is a group of metabolic diseases characterized by high blood sugar (i.e., high plasma glucose). More than a disease, investigators have characterized diabetes mellitus as a chronic metabolic disorder wherein insulin actions are diminished, either because insulin is not secreted into the blood stream or because the liver, muscles, and adipose tissue of an individual are insulin resistant. Due to the diminished response to insulin, those who have diabetes exhibit high plasma glucose and metabolic abnormalities involving carbohydrates, proteins, and fats (Stachura, 1996).

To be diagnosed with diabetes mellitus, an individual’s fasting plasma glucose needs to be measured at greater than or equal to 126 mg/dl (CDC, 2011). In an individual’s development of diabetes, he or she may progress through the following stages:

1. normal glucose metabolism
2. insulin resistance (IR)
3. impaired glucose tolerance (IGT) or impaired fasting glucose (IFG)
4. full-blown, frank diabetes mellitus.

IGT was officially used by the World Health Organization (WHO) in 1980 to categorize persons whose plasma glucose levels at 2 hours after taking 75 grams of glucose orally is intermediate between normal and diabetic readings (between 140 and 199 mg/dl). IFG is a condition in which fasting glucose is elevated (between 100 and 125 mg/dl) after an overnight fast, but is not high enough to be classified as diabetes (Harris, 1984; CDC, 2011).
DIABETES THROUGHOUT HISTORY

Diabetes is not a new disease. As early as 600 BC, an East Indian physician, named Susruta, described an illness whose symptoms were extreme thirst and the passage of copious sweet urine (Stachura, 1996). At the time, diabetes was a condition exhibited by the rich, and Susruta thought it was caused by gluttonous overindulgence.

In 1st century Greece, Aretaeus was the first to name the disease (diabetes means “running water through a siphon”) and provide a clinical description of it. In 1674, mellitus (the Latin word for honey) was added to the name when the Englishman, Thomas Willis, made a distinction between this condition and other causes of excessive urination.

In the mid 1800s, two French physicians named Bouchardat and Lanceraux divided the diabetic populations into two types: diabetes ‘gras” or fat (shown as derived from an insulin action defect in Figure 1, below); and diabetes “maigre” or thin (shown as derived from an insulin secretory defect in Figure 1). In addition, Langerhans described the pancreatic islets in 1869 and by 1900 pancreatic islet beta-cells were shown to have been damaged in those dying of diabetes (Stachura, 1996; MacFarlane et al., 1997).

TYPES OF DIABETES

There are three types of diabetes (Keen and Barnes, 1997; Keen et al., 1982):

- Gestational diabetes mellitus (GDM) is a condition characterized by elevated plasma glucose during pregnancy. Women who develop GDM, and the offspring that they carry, are more prone to type 2 diabetes later in life. According to Cornier et al.
(2008), the prevalence of conversion of gestational diabetes to frank diabetes varies between 6 and 92%, depending on racial and ethnic background of the subjects, and the length of time they are studied.

- Type 1 diabetes (T1DM), or insulin-dependent diabetes mellitus, is a juvenile-onset, auto-immune condition characterized by the low or absent secretion of insulin.

- Type 2 diabetes (T2DM) is an adult-onset (generally after age 40) condition. This type of diabetes has either of two etiologies. Under the first etiology, insulin secretion is insufficient due to damage to the pancreas. Within the second, and more common, etiology, the individual develops a syndrome of inactivity and obesity. Eventually, insulin resistance, and, then, insulin insufficiency ensue (Mc Carthy, 1993). Figure 1 depicts both etiologies and lists the environmental and genetic factors that contribute to each type.

![Figure 1: Factors Contributing to Each Type of T2DM (adapted from Mc Carthy, 1993)]
This study concerns itself primarily with Type 2 diabetes, or T2DM, and especially with the type of T2DM described on the right side of Figure 1, above.

**PREVALENCE OF DIABETES**

According to the WHO, “the likelihood of developing diabetes…. rises steeply with increasing body fatness…approximately 85% of people with diabetes are type 2, and of these, 90% are obese or overweight” (WHO, 2004). To measure body fatness, the WHO and the Centers for Disease Control (CDC) use the Body Mass Index (BMI), which is an individual's weight in kilograms divided by the square of the individual's height in meters. Individuals with BMI's between 25 and 30 are considered overweight; those with BMI's greater than 30 are in the "obese" category (CDC, 2011). Statistics have shown that for every kilogram of weight increase, the risk of diabetes increases by 5.4%% (Must and McKeown, 2011).

With 68% of the US population being overweight or obese (CDC, 2003), and the number of obese individuals increasing by 0.5 (Australia) to 0.7% (UK) each year in the industrialized, English-speaking countries of the world alone (WHO, 2011), we can expect the number of people who suffer from T2DM to increase dramatically over the next few decades.

By the year 2030, the number of diabetic individuals in the US is expected to almost double and increase from 17.7 million to 30.3 million (Wild, 2004). This doubling is just a continuation of a recent pattern of steep increases in the number of diabetes sufferers. For example, between 1990 and 1998, a 33% increase was recorded in the United States. This increase was observed in both genders, in all ages, and in all ethnic
groups. People between the ages of 30 to 39 showed the largest increase (69.9%). The prevalence of diabetes among various populations as reported by the American Diabetes Association is as follows: 11.3% of the general population 20 years and older have diabetes; in addition, 14.2% of Native Americans, 11.8% percent of Latino/Mexican Americans and 12.6% of African Americans have the disease (CDC, 2011; diabetes.org, 2011).

In an article in the October 8, 2003, issue of JAMA, the CDC's chief epidemiologist, K.M. Narayan, analyzed the National Interview Survey (1984-2000) with regard to its insights into diabetes. A summary of his conclusions is as follows:

The average U.S. female born in 2000 has a 38.5 percent risk of developing diabetes, which will cut her life short by 14.3 years if she is diagnosed by age 40 and will reduce her quality of life for 18.6 years.

For males born in 2000, the risk of developing diabetes is 32.8 percent. The disease will shorten their lives by 11.6 years if diagnosed by age 40 and cut their quality of life for 22 years.

Female Hispanics run a 52.5 percent risk of diabetes from birth, while the risk for black women is 49 percent. The risk for male Hispanics is 51.9 percent and for black males 41.4 percent.

The diabetic state does not seem to favor either gender. In a statistical analysis of 1995 data (King, 1998), worldwide there were more women than men with diabetes (31 vs 20 million). However, in the developing countries, there were equal numbers of men and women with the disease (42 million each). Moreover, by the year 2025, the worldwide difference between the female and male disease prevalence is estimated to be reduced somewhat (159 vs 141 million), due mainly to a reduction in the percentage of female diabetics in established market economies and in former Soviet Socialist countries. Currently, 13 million American men aged 20 years and older have the disease; while 12.6 million women 20 years of age and older have it (CDC, 2011).
According to Cornier et al (2008), the difference in the prevalence among men and women may be due to differing socioeconomic status, work-related activities, and cultural views of body fat.

Although most cases of diabetes appear in the developed world, a disproportionate growth in diabetes has occurred and is expected to continue to occur among populations that are rapidly adopting a Western lifestyle (Knowler, 1993). The prevalence of glucose intolerance (IGT and T2DM) among some of these populations is shown in Table 1.

The growth in diabetes has been due in part to the increase in obesity within populations. The increase in obesity, in turn, is due to growing urbanization, an increase in the consumption of fatty diets, and the decrease in physical activity that comes with less on-the-job activity, more electronics in the home, and the replacement of more active leisure activities with passive television watching (Popkin, 2001).

Table 1: Prevalence of Glucose Intolerance (W.Knowler, 1993: 205)

<table>
<thead>
<tr>
<th>Population</th>
<th>Prevalence (%)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mapuche Indians, Chile</td>
<td>2</td>
<td>Populations with a traditional lifestyle</td>
</tr>
<tr>
<td>Rural Melanesians</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Rural Polynesians</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Rural Indians, India</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Urban Hispanics, USA</td>
<td>30</td>
<td>Populations in which rapid adoption of Western lifestyles has occurred</td>
</tr>
<tr>
<td>Urban Indians, Fiji</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>Micronesians</td>
<td>62</td>
<td></td>
</tr>
<tr>
<td>Pima Indians, USA</td>
<td>66</td>
<td></td>
</tr>
</tbody>
</table>

It is interesting to note that O’Dea (1984) has shown that when the Western lifestyle is replaced by the original indigenous lifestyle, obesity and diabetes quickly disappear. In a study where 10 diabetic Australian Aborigines reverted to their hunter/gatherer lifestyle and diet, O’Dea demonstrated that the symptoms of T2DM
could be reversed in as few as 7 weeks (O’Dea, 1984). During this period, study subjects reduced their weight from an average BMI of 27.2 to that of 24.5. In addition, their fasting insulin was reduced from 23 to 12 mU/l.

As of the year 2010, the worldwide prevalence of diabetes was estimated by the International Diabetes Federation (IDF, e-altas). Table 2 gives the estimated percentage of the population with diabetes for each area listed.

<table>
<thead>
<tr>
<th>Region</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Africa</td>
<td>3.2%</td>
</tr>
<tr>
<td>Middle East</td>
<td>7.7%</td>
</tr>
<tr>
<td>Europe</td>
<td>7.0%</td>
</tr>
<tr>
<td>North America</td>
<td>10.0%</td>
</tr>
<tr>
<td>South/Central America</td>
<td>6.5%</td>
</tr>
<tr>
<td>South East Asia</td>
<td>7.0%</td>
</tr>
<tr>
<td>Western Pacific</td>
<td>4.2%</td>
</tr>
</tbody>
</table>

By the year 2030, the IDF expects the prevalence of diabetes to grow in every region of the world for those between the ages of 20 and 79. Table 3 shows these growth estimates.

<table>
<thead>
<tr>
<th>Region</th>
<th>Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2010</td>
</tr>
<tr>
<td>Africa</td>
<td>3.2%</td>
</tr>
<tr>
<td>Middle East</td>
<td>7.7%</td>
</tr>
<tr>
<td>Europe</td>
<td>7.0%</td>
</tr>
<tr>
<td>North America</td>
<td>10.0%</td>
</tr>
<tr>
<td>South/Central America</td>
<td>6.5%</td>
</tr>
<tr>
<td>Southeast Asia</td>
<td>7.0%</td>
</tr>
<tr>
<td>Western Pacific</td>
<td>4.2%</td>
</tr>
</tbody>
</table>
**BIOLOGICAL HERITAGE**

According to Katherine Milton (1999b: 488), “the widespread prevalence of diet-related health-problems, particularly in highly industrialized nations, suggests that many humans are not eating in a manner compatible with their biology”. It may be helpful, therefore, to examine the digestive anatomy and physiology that our ancestors bequeathed to us to understand diabetes in modern populations. These will suggest what our ancestors might have eaten and give us clues to the type of diet to which our digestive anatomy is best adapted.

**Primate Diets**

Primate diets are typically categorized as faunivorous, folivorous, and frugivorous (Chivers and Hladik, 1980a; Hladik, 1981):

- Faunivorous diets are comprised of vertebrate and invertebrate animal protein and fat.
- Folivorous diets are composed of the structural parts of plants, including leaves, grasses, stems, bark, gum, etc.
- Frugivorous diets are composed of the reproductive parts of plants, including fruits, flowers, seeds, tubers.

Both folivorous and frugivorous diets are carbohydrate rich. In addition, frugivorous diets, through the incorporation of nuts, seeds and some fruits, can contain high
amounts of vegetable fat; while, folivorous diets contain the protein that is brought to them by the structural and leafy parts of plants (Milton, 1993).

Although the diet that an animal consumes is identified through observation, a gut differentiation coefficient is available (Chivers and Hladik, 1980b). This coefficient organizes species into faunivorous, frugivorous, or folivorous categories depending on the length of the organs in their gut. The coefficient is calculated by combining the surface areas of the stomach, cecum, and colon and then dividing the result by the surface area of the small intestine. Faunivores have coefficients between 0.1 and 0.5; coefficients for frugivores lie between 0.5 and 1.0; while folivores have coefficients greater than 1. It follows from the coefficient ranges, that the surface area of the faunivore small intestine is greater than the combined surface area of the stomach, cecum and colon; while the surface area of the small intestine for folivores is less than the combined area of the stomach, cecum, and colon. The coefficient for the chimpanzee (Pan troglodytes), for example, is approximately 1, meaning the surface of their small intestine is about equal to the combined surface area of their stomach, colon and cecum. This places them on the border between the frugivores and folivores. A coefficient of 1.0 for them would have us believe that they are frugivores who eat fruits, seeds and flowers, but whose diet may incorporate the leaves that are characteristic of the folivore diet. In addition, we know that frugivores, as a rule, supplement their diets with insects and/or leaves (Chivers and Hladik, 1980a). These supplements are confirmed for chimpanzees whose diet “consists primarily of ripe fruits supplemented by leaves and some animal prey” (Milton, 1993, 87). Baboons and macaques also fall
into the frugivourous category with gut differentiation coefficients of 0.6 and 0.8, respectively. (Chivers and Hladik, 1980b).

Generally, the gut differentiation coefficient reflects the specialization of different parts of the primate digestive anatomy, since different organs process different macronutrients. For example, the stomach is very involved in protein digestion. It is there that the proteins are denatured by the action of pepsin, trypsin, and chymotrypsin (Lambert, 1998). Lipids are digested almost entirely in the small intestine (Lambert, 1998; Whitney and Rolfes, 2002). Carbohydrates are processed in the mouth, stomach, cecum, and colon (Whitney and Rolfes, 2002). Primates that rely on carbohydrates (e.g., young leaves) utilize either a complex sacculated stomach or the cecum and colon for the fermentation and digestion (Lambert, 1998; Milton, 1999b); while primates that rely on fruits, do much of their food fermentation and processing in the stomach (Lambert, 1998; Whitney and Rolfes, 2002).

**Figure 2: Gut Parts by Species (Milton, 1999b)**
In addition, Milton (1999a) has argued that the digestive system exhibits a certain degree of plasticity. This may imply that the more an organ is needed in the digestion of the diet a species currently eats (or, has eaten in its evolutionary past), the more surface area that the organ contributes to the primate’s total digestive potential (Chivers and Hladik, 1980a; Lambert 1998; Lee et al., 1995). Hence, it is generally the case that carbohydrate-rich diets require more surface area in the stomach and colon for digestion, while diets that contain fat require more small intestine surface area (Milton, 1999a). Conversely, it should be noted that the absence of a feature (e.g., cecum) does not mean that a species lacks the ability to digest a related nutrient (e.g., leaves) (Milton, 1987). It is worthy to note that in modern humans, some digestion continues to persist in the cecum with the help of microbiotica (Whitney and Rolfes, 2002). Studies of mice (Turnbaugh et al., 2006; Ley et al., 2005) indicate that the amount of nutrients our guts can extract from our food may be dependent upon the proportions of microbiota types that live in the cecum. Obese mice tend to have 50% less Bacteroidetes (and a proportional increase in Firmicutes) compared to their lean littermates. Those of us who tend to put on weight more easily may have a mix of flora in our gut similar to the obese mice.

As gut proportions are a feature of the vertebrate anatomy that exhibit some plasticity, they can vary within the same species. In addition, the diets of the same species of primate show variety, as do the environments in which they live (Chapman, 2002). Studies of birds (McWilliams and Karasov 2001) and mammals (Hume et al., 2002) show that the gut exhibits plasticity as it reacts to a change in the quantity and
quality of the diet. For example, the small intestine of birds expands to accommodate occasions for higher food intake along the migratory pathway. In addition, in studies of marmots who had emerged from hibernation in April, and whose gut measurements were taken in July, it was shown that the stomach had increased by 105%, the small intestine by 259%, and the colon by 158% (Hume et al., 2002). Long term, birds respond to an increase in the quality of their diets by a decrease in gut length and an increase in amino acid uptake (McWilliams and Karasov 2001). These studies have not been done for human or primate subjects. Therefore, we do not know with certainty how much gut plasticity affects absorptive capacity. If humans are like other mammals, however, the more we eat, the more our intestines expand, and, consequently, the more we absorb.

Figure 2 compares the digestive anatomy of several hominoids, including humans. Note that gut proportions are generally dictated by phylogeny (Milton, 1999a). For example, all hominoids (apes and humans) show the same basic gut anatomy, which consists of a small stomach, a small intestine, a small cecum terminating in an appendix, and a markedly sacculated colon (Milton, 1987; Chivers and Hladik, 1980b). However, within that general framework, we can conjecture that digestive anatomy differs among the hominoids because each evolved in a different environment and, therefore, ate a different diet. For example, orangutans and gorillas followed an evolutionary path that kept them in the rainforests where they thrived on a diet of leaves, fruits, and other plant parts. For them, the size of their colon and cecum (see Figure 2) needed to adequately accommodate the digestion of their mostly herbivorous diet (Milton, 1999b).
A study of Figure 2 shows that the small intestine dominates the gut in humans. The fact that the small intestine is so prominent in the human digestive anatomy suggests that our anatomy specializes in the utilization of lipids. However, humans are not the only primates in whom this is the case. For example, baboons have a gut differentiation coefficient equal to 0.6; while the same measurement for macaques is about 0.8 (Chivers and Hladik, 1980b). This indicates that the small intestine contributes more than half of the digestive capacity in these species. This is very comparable to humans in whom the small intestine contributes 56% of the volume of the digestive system (Milton 1999b). Interestingly, baboons, African green monkeys, and macaques are prone to obesity and diabetes when they are physically inactive or when they are fed a calorically dense diet that they can enjoy at will (Kemnitz, 2002; Hansen and Bodkin, 1993; Wagner et al., 2006). In addition, baboons may have evolved in a savanna environment similar to the one in which the genus Homo emerged (Alberts et al., 2005).

**Hominin Diets**

Table 4 shows how our brain size, body size, and tooth morphology evolved through time. By the time modern Homo sapiens appeared on the planet, we had evolved into skillful hunters whose brains, and the cognitive abilities they endowed us with, were the most important part of our anatomy. In all probability, our elongated small intestine had emerged with the arrival of Homo erectus (Aiello and Wheeler, 1995) and the concomitant importance and meat and animal fats (Behrensmeyer, 1997; Reed, 1997) in our diet with life on the savanna (Milton, 1999a).
Table 4: Hominin brain size, body size, and tooth morphology (adapted from Leonard, 2003)

<table>
<thead>
<tr>
<th>Species</th>
<th>MYA</th>
<th>Brain Size (cc)</th>
<th>Weight Males (kg)</th>
<th>Weight Females (kg)</th>
<th>Tooth Surface (sq mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. afarensis</td>
<td>3.9 – 3.0</td>
<td>438</td>
<td>45</td>
<td>29</td>
<td>460</td>
</tr>
<tr>
<td>A. africanus</td>
<td>3.0 – 2.4</td>
<td>452</td>
<td>41</td>
<td>30</td>
<td>516</td>
</tr>
<tr>
<td>P. boisei</td>
<td>2.3 – 1.4</td>
<td>521</td>
<td>49</td>
<td>34</td>
<td>756</td>
</tr>
<tr>
<td>P. robustus</td>
<td>1.9 – 1.4</td>
<td>530</td>
<td>40</td>
<td>32</td>
<td>588</td>
</tr>
<tr>
<td>H. habilis</td>
<td>2.4 -1.6*</td>
<td>612</td>
<td>37</td>
<td>32</td>
<td>478</td>
</tr>
<tr>
<td>H. erectus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(early)</td>
<td>1.8 – 1.5</td>
<td>863</td>
<td>66</td>
<td>54</td>
<td>377</td>
</tr>
<tr>
<td>(late)</td>
<td>0.5 – 0.3</td>
<td>980</td>
<td>60</td>
<td>55</td>
<td>390</td>
</tr>
<tr>
<td>H. heidelbergensis</td>
<td>0.6 - 0.12</td>
<td>1250</td>
<td>60</td>
<td>55</td>
<td>350</td>
</tr>
<tr>
<td>H. sapiens</td>
<td>0.4 – 0.0</td>
<td>1350</td>
<td>58</td>
<td>49</td>
<td>334</td>
</tr>
</tbody>
</table>

*Leonard’s table lists “1.9-1.6” here. However, Conroy’s (2005) dates of 2.4-1.6 are more accurate.

Homo sapiens Diet

Modern Homo sapiens evolved in Africa around 200,000 years ago and spread throughout Europe and Asia by around 40,000 years ago. For the most part, the H. sapiens culture is categorized as Late, or Upper, Paleolithic (Jurmain, 2011). Their technology extended to bone tools, including fishing hooks, and microliths like arrow points, awls, hide-polishers (Klein, 1983). Their food procurement assemblage also included nets, traps, snares, and darts (Hoffecker, 2009). Therefore, they were much more adept at hunting than their ancestors (Klein and Cruz-UrIBE, 1999) and included the following in their diverse diet (Robbins et al., 1996; Richards 2002):

- Mammals: springhare, fox, mongoose, hyena, serval, zebra, rhinoceros, kudu, duiker, steenbok, hartebeest, eland, buffalo, reindeer (Hoffecker, 2009).
- Rodents: rat, gerbil, mouse.
• Other: Tortoise, lizard, snake, birds (Klein, 1983), fresh water fish, marine fish and mammals (Richards, 2009).

Although isotope analysis indicates that the diet of these people came primarily from animal protein (Richards, 2009), there is some indication they may have incorporated plant foods into their diet, since either fat or plant foods are required in the diet to dilute the nitrogen load that comes from eating meat (Jones, 2009; Flodin, 2010; Cordain, 2000). Candidates for plant foods in the diet vary by region (Jones, 2009). In Germany, the cherry, blackberry, dewberry, and raspberry may have provided the vegetal part of the diet; in Israel, acorns, pistachios, and almonds may have provided it; while in Spain, sloe, rosehips, and wild apple are candidates (Jones, 2009).

The skeletal remains uncovered at Upper Paleolithic *H. sapiens* sites would have us believe that they were well-adapted to hunting large prey and to meat eating, with a digestive anatomy that heavily utilized the elongated small intestine that had developed in earlier times. Our current diets provide foods for us that are very dissimilar from the prehistoric ones described here. Perhaps adding more lean meats to our diet would help us avoid diabetes.

The diet of modern hunter-gatherers may help us understand the intricacies of the subsistence habits of *H. sapiens* before the arrival of agriculture. Table 5 summarizes the types of diets eaten by 229 groups of modern hunter-gatherers (Cordain, 2000; Murdock, 1927). It indicates that as the contribution of plant matter decreases, an increase in the contribution of fish to the diet generally occurs. In fact, for societies living between 0 and 40 degrees latitude N or S, studies show that the combined contribution of plant matter and fish to the diet falls within 46-55% (Cordain,
So, animal proteins and fats, albeit sometimes in the form of fish protein, always make a significant contribution to hunter-gatherer diets.

**Table 5: Diets of Modern Hunter-Gatherers by Biome (Cordain, 2000)**

<table>
<thead>
<tr>
<th>Environment</th>
<th>Plant Contribution (%)</th>
<th>Fish Contribution (%)</th>
<th>Contribution of Animals other than Fish (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tundra</td>
<td>6-15</td>
<td>46-55</td>
<td>36-45</td>
</tr>
<tr>
<td>Northern coniferous forest</td>
<td>16-25</td>
<td>46-55</td>
<td>26-35</td>
</tr>
<tr>
<td>Temperate forest</td>
<td>36-45</td>
<td>36-45</td>
<td>16-25</td>
</tr>
<tr>
<td>Desert grasses &amp; shrubs</td>
<td>46-55</td>
<td>6-15</td>
<td>36-45</td>
</tr>
<tr>
<td>Temperate grasslands</td>
<td>26-35</td>
<td>6-15</td>
<td>56-65</td>
</tr>
<tr>
<td>Subtropical bush</td>
<td>36-45</td>
<td>26-35</td>
<td>26-35</td>
</tr>
<tr>
<td>Subtropical rain forest</td>
<td>36-45</td>
<td>6-15</td>
<td>46-55</td>
</tr>
<tr>
<td>Tropical grassland</td>
<td>46-55</td>
<td>16-25</td>
<td>26-35</td>
</tr>
<tr>
<td>Monsoon forest</td>
<td>36-45</td>
<td>26-35</td>
<td>26-35</td>
</tr>
<tr>
<td>Tropical rain forest</td>
<td>26-35</td>
<td>36-45</td>
<td>26-35</td>
</tr>
</tbody>
</table>

It is appropriate that we consider hunter-gatherer lifestyles in order to deduce what our ancestors ate in the Upper Paleolithic because only in the Holocene period -- during the last 10,000 years -- did we assume a sedentary lifestyle and attempt to control the availability of our food by becoming farmers and herders (see Table 6).

Fostering this change to farming was a dramatic change in the environment that occurred between 14,000 and 10,000 years ago when the temperature moderated and the climate became drier. With these new climatic conditions came migration, population aggregation and growth (Watson, 1995).

The process of domestication involved a gradual shift to dependence on domesticated plants and animals, starting with their use as supplements to what was already being hunted and gathered (Prince and Gebauer, 1995). In general, people were exposed to both the wild ancestors of domesticates (both plant and animal), and to the domesticates themselves, long before they adopted the agricultural lifestyle. Studies of strata at archeological sites suggest that foraging predominated in sedentary
communities before it was gradually replaced by farming (Renfrew and Bahn, 2000).

Many times the first groups to adopt agriculture were the foragers with the most complex societies, who occupied the richest lands (Hayden, 1995). In addition, farming practices, and the domesticates themselves, generally spread by diffusion across Eurasia in an east/west direction (Diamond, 1997).

Table 6: Development of Agriculture (Simmons, 1996; Edens, 2000; Kelly & Thomas 2010)

<table>
<thead>
<tr>
<th>Region</th>
<th>Year</th>
<th>Domesticate</th>
</tr>
</thead>
<tbody>
<tr>
<td>North America</td>
<td>10,000 BC</td>
<td>Dog</td>
</tr>
<tr>
<td>Mesoamerica</td>
<td>3,500 BC</td>
<td>Squash</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gourd</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bean</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Maize</td>
</tr>
<tr>
<td>South America</td>
<td>4,000 BC</td>
<td>Bean</td>
</tr>
<tr>
<td></td>
<td>2000 BC</td>
<td>Lima Bean</td>
</tr>
<tr>
<td></td>
<td>3500 BC</td>
<td>Guinea Pig</td>
</tr>
<tr>
<td>Middle East</td>
<td>10,000 BC</td>
<td>Dog</td>
</tr>
<tr>
<td></td>
<td>8,000 BC</td>
<td>Sheep</td>
</tr>
<tr>
<td></td>
<td>8,000 BC</td>
<td>Einkorn</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Emmer</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lentil</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pig</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Goat</td>
</tr>
<tr>
<td></td>
<td>6,500 BC</td>
<td>Cattle</td>
</tr>
<tr>
<td></td>
<td>3,000 BC</td>
<td>Zebu cattle</td>
</tr>
<tr>
<td></td>
<td>2,000 BC</td>
<td>Dromedary camel</td>
</tr>
<tr>
<td>Far East</td>
<td>5,000 BC</td>
<td>Rice</td>
</tr>
<tr>
<td></td>
<td>3,750 BC</td>
<td>Millet</td>
</tr>
<tr>
<td></td>
<td>3,000 BC</td>
<td>Horse</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bactrian camel</td>
</tr>
<tr>
<td></td>
<td>4,000 BC</td>
<td>Water Buffalo</td>
</tr>
</tbody>
</table>

Transition to Agriculture

A closer study of the sequence of human dietary history reveals what the transition to agriculture -- and its emergent growth, specialization and urbanization -- has meant in terms of health. Popkin (2002) has suggested that our human dietary
history has progressed through five modes or stages. Each mode, or stage, is characterized by its own dietary benefits and problems.

1. **Food Collection**: During this stage, people eat a variety of foods and physical activity is very high. Little obesity is found. This stage occurs with the hunter-gatherer lifestyle.

2. **Famine**: This stage is accompanied by the advent of agriculture. Because the diet is less varied, these are periods of acute scarcity where blight or poor weather make the yearly harvest impossible. The types of physical activities change, but the level of activity does not change.

3. **Receding Famine**: The diet begins to vary more and the contribution of fruits, vegetables and animal protein to the diet increases. Only in the last third of the last millennium have these changes become widespread. During this stage we see increased agricultural productivity.

4. **Nutrition-Related Noncommunicable Disease**: This stage is characterized by a diet high in fat, cholesterol, and sugar; but low in polyunsaturated fatty acids and fiber. Physical activity is reduced. There is a prevalence of obesity and degenerative disease is widespread. Pattern four is commonly seen among contemporary, Westernized societies, where physical activity is low.

5. **Behavioral Change**: During this stage, people are interested in preventing or delaying degenerative disease, so they incorporate more activity and a better diet in their lives.

   Although the transition to agriculture in many areas was gradual and based upon local experimentation with domesticates, there is also evidence that for some societies
the final step to total dependence upon agriculture was very abrupt and was
characterized by widespread famine (stage 2 of the Popkin classifications, above).
Denise Hodges (1989) of Northern Illinois University associates a slow transition to
agriculture, like the one that occurred in the Oaxaca Valley of Mexico, with fewer
physical problems like bone infections. Where the transition was more rapid, more
deleterious physical problems occur. For example, a study of human remains in the
Illinois River Valley in North America demonstrates how deleterious the transition from
foraging to agriculture could be. Goodman and Armelagos (1985) contrasted the health
of those who lived in the area, both pre- and post-agricultural transition. The valley’s
inhabitants were foragers until around 1050 AD. As foragers, they consumed grass
seed, fruits, berries, roots, tubers, vines, voles, birds, and fish. Upon their transition to
agriculture, their diet consisted mainly of corn, supplemented by products they foraged.
Table 7 summarizes the condition of the pre- and post-transition skeletons at the site.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Pre-Transition (Foragers)</th>
<th>Post-Transition (Farmers)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periosteal infections in tibias (shinbones)</td>
<td>26%</td>
<td>84%</td>
</tr>
<tr>
<td>Porotic hyperostosis (indicating anemia)</td>
<td>25%</td>
<td>50%</td>
</tr>
<tr>
<td>Childhood stunting</td>
<td>Less</td>
<td>More</td>
</tr>
<tr>
<td>Dental enamel defects</td>
<td>55%</td>
<td>80%</td>
</tr>
</tbody>
</table>

Some of the Goodman and Armelagos (1985) findings are summarized in Table 7. The table indicates that with the transition to agriculture, there was evidence that the people of the area suffered stress (low levels of nutrition and high levels of disease). According to Goodman and Armelagos, tibias (shinbones) were damaged because persistent bacterial infection injured the outer, periosteal, layer of the bone. Shinbones
were most affected because of their poor circulation. Porotic hyperostosis (a condition where a higher than normal percentage of the bone appears as marrow) occurred in the cranial bones in response to anemia. As the individual became more and more anemic, more of the interior of the bone was transformed to marrow and dedicated to the production of red blood cells. In severe cases, the outer layer of the bone disappeared and marrow portion showed on the outside of the cranium. Dental enamel defects and an increase in childhood stunting are evidence that these individuals suffered stress during the childhood periods of enamel and bone formation. According to Goodman and Armelagos (1985), the 40% of the pre-transition population suffered from degenerative defects, while 70% of the post-transition population suffered from them.

There could be many reasons for these deleterious effects. We know that the final Illinois River Valley agricultural site was used only as a seasonal site previously. Its suitability as a year-round site was not, therefore, established. In addition, all these effects could have resulted from disease and epidemics. The most obvious and proximal cause was the change in diet, however. Perhaps, nutritional stress was due to the fact that corn needs to be crushed and processed in lime to make all its nutrients available (Katz, 1975). Without proper processing, the corn would not have provided the people with the nutrients they required.

The people of the Illinois River Valley were not an isolated case. According to Larsen (1995:185), “the adoption of agriculture involved an overall decline in oral and general health. This decline is indicated by elevated prevalence of various skeletal and dental pathological conditions and alteration in skeletal and dental growth patterns… changes in food composition and preparation technology contributed to craniofacial and
dental alterations, and activity levels and mobility decline resulted in a general decrease in skeletal robusty." Larsen (2000) reports that dental pathologies and shorter stature were due to the food consumed and how it was prepared. He proposes that skeletal abnormalities were due to the indirect consequences of the agricultural transition: increased sedentism, population crowding, and poor hygiene. “Indeed,” he says (p. 105), “for almost every setting, there are increases in bone infection: for example, the eastern Mediterranean, west-central Illinois, the central Ohio River Valley, central Illinois, the lower Mississippi River Valley, and Ecuador.”

We know that the change in diet that the agricultural transition brought was detrimental to our overall health. According to Milton (1987), our evolutionary past has suited us for eating dicototyledonous plants like cabbage and carrots; however, monocots, like rye and wheat, are somewhat indigestible for us. Part of the reason for this is the high cellulose to hemi-cellulose ratio in monocots. Another reason is that monocot cereal grains are high in phytate, which readily combines with zinc, iron, and manganese. Therefore, any primate using monocot cereals as dietary staples may require other special foods (that are high in zinc, iron, and manganese) to avoid deficiencies in these minerals (Lloyd et al., 1978). A knowledge of nutrition tells us that deficiency in these minerals could have caused the anemia (iron), stunted growth (zinc and manganese), and poor skeletal quality found at the Illinois Valley site (Whitney and Rolfes, 2002). In addition, substituting a diet high in milled cereals for one rich in wild fruits, tubers and roots changed the type of fiber in our diet (Milton, 1999b). Since our digestive tract did not evolve to process the types of fiber monocots provide, eating this type of diet would not be optimum for our health.
Many times transitions have been difficult for the human species to negotiate. But their negotiation sometimes has resulted in either physical or cultural evolution. The transition from woodland to savanna is associated with the extinction of the australopithecines and the great evolutionary leap to the genus *Homo* (Jurmain, 2011). The agricultural transition in some places was responsible for the deleterious effects shown in Table 7, but it established conditions where population growth, trade specialization, and the emergence of cities could occur. However, in geographical pockets where famine occurred, it may have also endowed the survivors and their progeny with thrifty genes that conserved their energy and allowed them to survive in sparse times, but tended to ensure fat storage in times of plenty. These thrifty genes would tend to render us more susceptible to diabetes.

**Transition to an Industrial Economy**

The Industrial Revolution, although it eventually increased our standard of living significantly, initially wrought deleterious effects on our health. These deleterious effects are evidenced by records of stature at the beginning of the industrial revolution both in America and in Europe. Komlos (1998:786) tells us that:

*Consumers rearranged their consumptive bundle in response to an increase in relative nutrient prices first by substituting industrial commodities for food products and with the food budget itself by substituting carbohydrates for protein rich foods. This was most noticeable among farmers, workers, and among the lower middle class….who could economize on food expenditures while maintaining the same degree of satiation by substituting inexpensive starchy foods for more expensive meats and diary products.*

According to Komlos, Europe entered the Industrial Revolution first so we find that average heights were affected there earlier than in America. We know that in the United Kingdom the average height for soldiers born in 1775 (168 cm) was less than for
those born in 1760 (170 cm). This decline was found to be synchronous in four European areas: the United Kingdom, Sweden, the Hapsburg empire, and Bavaria.

In America, the stature reduction was apparent among Union soldiers, Georgia convicts, and West Point cadets beginning between 1830 and 1840. Only male black slaves, who continued to be well-fed because their worth was related to their strength and stature, showed an increase in stature for the same time period (Komlos, 1998).

Komlos also reports (1998:783) that physical stature was linked to “a rise in income inequality, an increase in real food prices, increased variability of income, urbanization, and industrialization.” For example, he points out that income and nutritional resources were unevenly distributed so that the stature of upper and middle income students in Germany improved at the end of the 18th century, while the height of lower class during the same time period declined. This disparity also occurred in America at the end of the 1840s between West Point cadets whose fathers were professionals and those whose fathers had more modest incomes.

The post-industrial revolution era is represented by stage 4 in the Popkin list of classifications, above. This stage is characterized by a diet high in fat, cholesterol, and sugar; but low in polyunsaturated fatty acids and fiber. Physical activity is reduced. There is a prevalence of obesity and degenerative disease, like diabetes, is widespread. In addition, following the Industrial Revolution, food-processing procedures created food forms that we had not consumed in our evolutionary past. We had never before encountered the dairy foods, milled cereals, refined sugars, vegetable oils, and fatty meats that are so prevalent in our current diets. These changes are too recent on our
evolutionary timescale for the human genome to adjust. Hence, degenerative diseases have developed (Eaton et al., 1988; Cordain et al., 2005).

**EVOLUTIONARY EXPLANATIONS FOR DIABETES**

The answer to why our abundant diets have brought degenerative disease, including diabetes, to our modern populations may lie in the evolutionary past. Various evolutionary possibilities are implied from the foregoing explanation of our biological heritage:

- Our digestive anatomy, with its long small intestine, is efficient at extracting nutrients from our diet. In fact, the small intestine is the only digestive organ where fats are extracted (Lambert, 1998) and fats are more than twice as calorically dense as proteins and carbohydrates (Whitney and Rolfes, 2002). Although our ancestors required this very efficient digestive organ to sustain life on the savanna, their diet did not include the marbled meats and rich pastries and snacks that our modern populations eat. Our small intestine might be just too efficient for our modern diet. Moreover, as diets contain less and less fiber, nutrients tarry in the small intestine (Milton, 1993) and more of them are extracted (Lambert, 1998; Milton, 1999a). If they are not used, they are eventually stored as fat (Whitney and Rolfes, 2002). As our bodies store more and more fat, the development of type 2 diabetes is more probable (Boden, 1997).

- We evolved in an environment where we needed to be physically active to obtain the foods and build the shelters that would sustain us (Eaton et al., 1988). But as we pursue the sedentary lifestyles that more office jobs and lighter leisure activities bring, we burn fewer calories and store more of them as fat. The estimated physical
activity level of *H. erectus* is estimated to be 1.54 times greater than the modern office worker (Lieberman, 2003). In addition, being overweight is not always the result of overeating, but the energy imbalance that comes from eating more than our metabolism burns is always the culprit. The inactivity that comes from watching too much television can also result in obesity, the primary cause of type 2 diabetes. One study found that watching television more than two hours daily was predictive of being overweight (Hughes, 2005).

- The transition to the cereal-rich agricultural diet may have left us with an abiding metabolic hunger for the dicototyledonous foods that nurtured us in the evolutionary past (Milton, 1987; Eaton et al., 1988). Yet, we turn to good-tasting modern foods, laden with sugars and fats, for satiation. Since these foods are metabolically inadequate, they are unable to quiet our primordial hunger pangs and we eat to the point of obesity. Again, obesity is the major cause of type 2 diabetes.

- During both Paleolithic and Holocene times, an adequate diet was not always available. Not all of *H. erectus*’ hunting expeditions returned with a kill (Garn and Leonard, 1989). In addition, famine, drought, and plagues have been periodic throughout human history. It has been suggested that to survive during these lean times, a thrifty gene was preserved in the human genome (Neel, 1962). Many types of thrifty genes have been proposed. Some of them spare carbohydrates; others make individuals store excess calories as fat more readily; still others modify insulin action. More detailed explanations of the thrifty gene are given later in this document.
During Pleistocene times, meat formed the core of our diet (Klein, 1983). Perhaps, increasing the amount of lean meats in our diets would render them more satisfying and improve the efficiency of our metabolism (Cordain et al., 2005; Eaton et al., 1988). The end result would be less obesity in our population, and, hence, less diabetes.

One or more of the above explanations may be the underlying culprit in making modern populations more obese, and, therefore more prone to diabetes. Perhaps, a study of the disease itself will yield more clues to its origin.

**THE ROLE OF INSULIN AND GLUCOSE METABOLISM**

We cannot understand diabetes without first considering insulin and its role in food metabolism. Insulin is a hormone secreted by the pancreas to control plasma (blood) glucose levels. When the pancreas detects a rise in the glucose in the blood, it secretes insulin into the blood stream. The actions of insulin are to promote the metabolism of the glucose that is already in the blood stream and to inhibit other metabolic processes that would increase plasma glucose levels. These actions are shown in Table 8, below.

<table>
<thead>
<tr>
<th>Table 8: Actions of Insulin (adapted from Stachura, 1996:8)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Promotes</strong></td>
</tr>
<tr>
<td>- Entry of glucose into cells</td>
</tr>
<tr>
<td>- Storage of glycogen* (glycogenesis)</td>
</tr>
<tr>
<td>- Storage of fat (lipogenesis)</td>
</tr>
<tr>
<td>- Storage of amino acids</td>
</tr>
<tr>
<td><strong>Inhibits</strong></td>
</tr>
<tr>
<td>- Synthesis of glucose (gluconeogenesis)</td>
</tr>
<tr>
<td>- Breakdown of glycogen* (glycogenolysis)</td>
</tr>
<tr>
<td>- Breakdown of fat (lipolysis)</td>
</tr>
<tr>
<td>- Breakdown of protein (proteolysis)</td>
</tr>
</tbody>
</table>

*Glycogen is excess glucose stored in the liver and muscles for later reconversion to glucose.
To understand T2DM, we must first understand how glucose metabolism works. Much of glucose metabolism is controlled by hormones released from clusters of cells, called islets of Langerhans, embedded in the pancreas. These clusters regulate fuel metabolism throughout the body and are able to (Stachura, 1996, 5):

- Sense the need for and the availability of glucose.
- Promote the storage of overabundant glucose.
- Maintain the level of plasma glucose within narrow limits.

The normal human pancreas contains about 1 million islets, most in the pancreatic tail. Each islet contains several types of cells, 60% of which are B (beta) cells, which produce insulin (Orci et al., 1976). Also, included within the islets are a (alpha) cells, which produce glucagon. Glucagon is released in response to low blood glucose concentration. It stimulates hepatic glucose production and elicits the release of glucose from storage (Felig et al., 1976).

The metabolic dance between these two hormones – insulin and glucagon – regulates plasma glucose levels as glucose metabolism switches between the fed state and the fasting state.

The Fed State

After food ingestion, circulating insulin levels rise within minutes in the individual with normal glucose metabolism. Insulin is released in two phases (Pfeifer et al., 1980). First, a rapid burst of insulin occurs within 1 or 2 minutes after food ingestion. This burst is complete within 10 minutes. The second phase begins 5 to 10 minutes after the initial increase and continues as long as the beta-cells are stimulated by elevated plasma
glucose levels. The insulin regulates glucose metabolism within the liver, the muscles, and adipose tissue (Whitney and Rolfes, 2002):

- Within the liver, insulin promotes the storage of glucose as glycogen (glycogenesis) and inhibits the breakdown of glycogen (glycogenolysis) and the creation of glucose (gluconeogenesis).
- Within the muscles, insulin promotes glycogenesis and inhibits glycogenolysis and protein breakdown (proteolysis).
- Within the adipose tissue, insulin promotes the entry of glucose into the cell and the formation of fat (lipogenesis). It also inhibits the breakdown of fat for energy (lipolysis).

Glucose metabolism occurs within the cell. Glucose transporters (GLUTs) are the mechanisms that the body uses to transport glucose and fatty acids from the bloodstream into the cell. They come in five varieties (Brooks et al., 2000):

- GLUT1: these are located throughout the body
- GLUT2: these are present in the gut, liver, and pancreatic islets
- GLUT3: these are present in the central nervous system and brain
- GLUT4: these are present in insulin-responsive tissues, skeletal muscle, adipose tissue, and heart
- GLUT5: these are present in the small intestine.

In skeletal muscle and adipose tissue, GLUT4 exists in intracellular vesicles. These vesicles are mobilized to the cell's plasma membrane by the stimulation of intracellular insulin receptors through the presence of extracellular insulin at the cell membrane (Brooks et al. 2000). In the absence of insulin, GLUT4 is not available at the
cell membrane to transport glucose from the blood to the cell. In addition, insulin resistance is associated with a decrease in GLUT4 transporter number and activity (Rajan, 2002).

**The Fasting State**

The fasting state occurs after glucose metabolism is completed. At the initiation of this stage, the pancreas releases glucagon into the blood stream. Glucagon induces a catabolic state in which glycogenolysis occurs in the liver and muscles. In addition, lipolysis occurs within the adipose tissue. During the lipolysis, fatty acids are freed from the stored triglycerides and the resulting glycerol structure is converted to glucose by the liver.

When an individual has diabetes mellitus, his or her glucose metabolism remains in a runaway fasting state. Besides promoting gluconeogenesis in the liver, this runaway state eventually results in insulin resistance in the liver, muscles, and adipose tissue. Thus, high levels of glucose are allowed to circulate in the plasma, along with high levels of plasma insulin, resulting in damage to blood vessels (with diabetes the vascular endothelium becomes abnormal) and internal organs (Stachura, 1996). Therefore, 63.7% of people with T2DM live, on the average, fewer years than those expected. (Arizona Mortality Statistics, 2009).

**PATHOGENESIS OF DIABETES**

Although diabetes mellitus is diagnosed at specified glucose levels, investigators have begun to consider diabetes not as a categorical state, but as an extreme in a continuum (Wareham, Franks, and Harding, 2002: 558). Moreover, it is thought that
reduction of diabetes to a binary state – where you either have it or you don’t -- might be somewhat artificial.

Figure 3: Progression of Glucose Intolerance (adapted from De Meyts, 1993: 98)

A better characterization of diabetes might be to view it as a process. Figure 3, above, illustrates this process for one patient (Granner & O’Brien, 1992). In the diagram, we see the stages through which the individual progressed as he or she moved from normalcy (stage 0), through insulin-resistance (IR - stage 1), through impaired glucose tolerance (IGT -- stages 2 and 3), finally arriving at T2DM (stages 4 and 5).

In Figure 3, the "N"s denote that the particular metabolic pointer (shown in the leftmost column of the diagram) was normal when the clinical status was as marked at the bottom of the column; asterisks denote events that mark the transition from one stage (0 through 5) to another. For example, entry into stage 1 (the insulin resistant -- IR -- stage) was marked by reduced peripheral glucose utilization, which probably resulted from inactivity; entry into stage 2 (the beginning of the impaired glucose
tolerance -- IGT -- stage) was marked by an increase in hepatic glucose production (HGP), even in the fed state; entry into stage 4 was marked by a reduction in insulin production; stage 5 was marked by a reduction of insulin production, a reduction in peripheral insulin utilization and an increase in hepatic glucose production. At the top of the diagram, are the organs that became involved in the disease process as the individual progressed from normalcy to T2DM.

It should be noted that IGT patients do not always develop T2DM. However, they do have a higher risk of developing it. Studies indicate that Pima Indian develop diabetes at a rate proportional to their plasma glucose concentrations (Hamman et al., 1978). Other studies show that people with IGT develop T2DM at a rate of 1 to 5 percent a year, compared to 1 percent of individuals classed as normal. However, these studies also showed that even after 10 years, the majority of persons remain in the IGT category, and a significant number return to normal (Keen et al., 1982; Qian et al., 2011; Kleber et al., 2010). Increased age, decreased physical activity and obesity seem to be the determining factors in whether an individual develops T2DM (Boden, 2001; CDC, 2011).

INSULIN RESISTANCE

The development of insulin resistance is the first step in the development of T2DM. As the tissues become insulin-resistant, the pancreas must increase its insulin production to obtain the same metabolic results. Eventually, the pancreas' beta-cells fail, pancreatic insulin output drops precipitously, and frank T2DM ensues (De Meyts, 1993; Cornier 2008; DeFronzo, 2010).
The Role of Free Fatty Acids

Current thinking is that the free fatty acids (FFAs), and the tissues where these FFAs are found play a role in the development of insulin resistance (Boden, 2001; McGarry, 2002; Ravussin & Smith, 2002; Kahn et al., 2006). Most foods hold their fats in triglyceride form. Each triglyceride contains one unit of glycerol to which three fatty acids are attached. Triglycerides are catabolized to fatty acid and glycerol in the small intestine so they can pass through the intestine wall. After passing through the intestine wall, fatty acids and glycerol units are reconstituted as triglycerides for transport (within chylomicrons) through the circulatory system and delivery to either the liver for catabolism or to the adipose tissue for storage (Whitney, 2002). In an individual with normal glucose metabolism, when the circulatory system is flooded with FFA and triglycerides for more than 3.5 hours, FFAs begin to affect liver function and start to be stored intramuscularly. This force feeding of lipids triggers a state of insulin resistance in these individuals and renders them "temporarily diabetic" (Boden, 2001b; Santomauro, 1999; McGarry, 2002; Brechtel, 2001; Krssak, 1999; Kahn et al., 2006)). In those individuals whose glucose metabolism is impaired (i.e., those with IGT and frank diabetes), FFAs are also thought to trigger insulin resistance. However, instead of FFA infusions, the root causes are thought to be an overabundance of fatty foods (Marshall, 2002; Pederson, 1991) and fat depots (Boden 1997), increased fat in the bloodstream (Boden, 1997), the idiosyncratic placement of fat depots in the body (McGarry, 2002; Forouhi, 1999), and abnormalities of the pancreas’ beta-cells (Kahn et al., 2006).

FFAs affect four organs that are instrumental in the etiology of T2DM: the muscle tissue, the liver, adipose tissue, and the pancreas itself. The first three of these organs become insulin-resistant. The mechanisms for this resistance are as follows.
• **Muscle tissue:** In the diabetic state, lipids may be stored intramuscularly. The presence of intramuscular lipids shuts down insulin-stimulated glucose uptake by immobilizing GLUT4s. During this process, the GLUT4s are prevented from translocating to the muscle cell's membrane where they normally react with plasma glucose molecules to bring them into the cell for metabolism. A reduction in insulin receptor substrate has been identified as one of the causes of this GLUT4 failure (Boden, 2003; McGarry, 2002). An adverse intrauterine environment during an individual’s fetal stage can also permanently modify the expression of genes that regulate beta-cell development in the pancreas and GLUT4’s in the muscle, making the individual diabetes prone (Pinney and Simmons, 2009).

• **Liver:** Insulin's normal action is to inhibit hepatic glucose production and glycogenolysis. However, in the presence of FFAs, the liver does not respond to insulin signaling and continues to produce glucose even postprandially (Boden, 2003; Kahn et al., 2006). It should be noted that when the liver becomes insulin-resistant, plasma insulin and FFAs are both elevated. Indeed, high levels of plasma insulin turn the liver into a "fat-producing factory" (McGarry, 2002). Hypertriglyceridemia may even result from the liver's insulin resistance.

• **Adipose tissue:** Insulin normally promotes lipogenesis and inhibits lipolysis. However, one of the reasons adipocytes fail to take in triglycerides from the bloodstream for lipogenesis is a reduction in insulin receptor substrate (Zhanguo, 2004). Normally, 75% of the FFAs released through lipolysis are
recycled in the adipose tissue and liver; 50% of this recycling occurs in the adipose tissue (Reshef et al., 2003). Insulin resistance interferes with this recycling process (lipogenesis) and allows FFAs to continue to circulate. Moreover, insulin resistance in adipose tissue leads to increased lipolysis at inappropriate times (i.e., postprandially), which also leads to an increase in circulating FFAs. This eventually leads to ectopic fatty deposits in the muscle and liver. These deposits interfere with normal FFA and glucose oxidation in muscle and liver cells and lead to insulin resistance in these organs. (Delarue and Magnan, 2007; Corcoran et al., 2007).

The pancreas itself, instead of becoming resistant in the presence of FFAs, is potentiated by their presence. That is, in the presence of FFAs, the production of insulin is enhanced. In most individuals, this enhanced insulin production is sufficient to meet the needs of the insulin-resistant organs for more stimulation. However, in diabetic individuals, the beta-cells begin to exhibit abnormalities (Kahn et al., 2006) and eventually fail and die (Boden, 2003; Kashyap, 2003a).

**The Role of Obesity**

The fact that insulin resistance increases with weight gain and decreases with weight loss indicates that fat accumulation is not only associated with, but in fact, causes insulin resistance (Boden, 2001a). Boden has hypothesized that the adipose tissues of obese persons release FFAs that cause insulin resistance in skeletal muscle and in the liver (Boden, 1997). This relationship is shown below.
Numerous studies have found that BMI is more related to the development of T2DM than the physical activity level (Barclay, 2004; Brancati, 1999; Mayer-Davis, 1998; Sundquist, 2000). Furthermore, a Johns Hopkins study (Brancati, 1999) indicates at a man’s BMI status at age 25 predicts whether or not he will develop diabetes later on in life. The study concludes that “in men, [the status of] overweight at 25 years of age strongly predicts diabetes risk in middle age, largely through its association with overweight at 45 years of age and average BMI to 49 years of age.”

**The Role of Adipose Tissue**

The properties of adipose tissue may provide clues to why a high BMI predicts the diabetic state. A study of adipose tissue by Kershaw and Flier (2004) characterizes adipose tissue as an endocrine organ. “Adipose tissue,” they write, “is a complex, essential, and highly active metabolic and endocrine organ. Besides adipocytes, adipose tissue contains connective tissue matrix, nerve tissue, stromovascular cells, and immune cells.” Adipose tissue has been found to secrete hormones that act both locally and systemically to modify and control metabolism.
Table 9 summarizes the actions of proteins manufactured in adipose tissue. Note that, of the proteins listed, TNFα and IL-6 interfere with insulin action and promote an increase in FFAs. TNFα and IL-6, therefore, would be adaptive in environments that required carbohydrate-sparing mechanisms to promote survival. ASP (acylation stimulating protein) promotes fat synthesis in environments that promote obesity; while resistin inhibits glucose uptake in the same environments.

Table 9: Adipose Tissue Proteins Affecting Metabolism (data from Kershaw, 2004)

<table>
<thead>
<tr>
<th>Protein</th>
<th>Action</th>
<th>Relationships</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin</td>
<td>Signals energy sufficiency; acts with adiponectin in those with adipose tissue insufficiency to increase insulin sensitivity.</td>
<td>At low levels, it signals energy insufficiency; high levels indicate obesity.</td>
</tr>
<tr>
<td>TNFα</td>
<td>Suppresses expression of genes involved in glucose uptake and metabolism and fatty acid oxidation; enhances expression of genes that promote cholesterol and fatty acid synthesis; impairs insulin signaling</td>
<td>Important in the pathogenesis of obesity; positively associated with obesity.</td>
</tr>
<tr>
<td>IL-6</td>
<td>Induces hyperlipidemia, hyperglycemia, and insulin resistance; decreases insulin signaling by reducing insulin receptor signaling; inhibits adipogenesis and adiponectin secretion.</td>
<td>Expression of IL-6 is 2-3 times greater in visceral adipose tissue than in adipose tissue depots elsewhere in the body. Negatively associated with obesity.</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>In the liver, increases insulin sensitivity, increases fatty acid oxidation, and reduces hepatic glucose production. In the periphery, enhances nitric oxide production, and, therefore, exercise-induced glucose uptake.</td>
<td>Inverse association with obesity. Artificial increases in adiponectin lead to improved insulin sensitivity, and glucose tolerance. It also decreases the number of circulating FFAs.</td>
</tr>
<tr>
<td>ASP (acylation stimulating protein)</td>
<td>Promotes FFA uptake by increasing lipoprotein lipase activity, promotes triglyceride synthesis and decreases lipolysis.</td>
<td>Positively associated with obesity, insulin resistance,</td>
</tr>
<tr>
<td>Resistin</td>
<td>Impairs insulin-stimulated glucose uptake.</td>
<td>Positively associated with obesity*. More active in visceral adipose tissue. Lower resistin levels improve fasting glucose levels.</td>
</tr>
</tbody>
</table>

*There may be a weak relationship between resistance and insulin-sensitivity in non-obese individuals (Heilbronn, 2004)

Adiponectin enhances the metabolic actions that promote good glucose metabolism. Its actions include (Berg, 2001; Chadran, 2003; Duncan, 2004; Pandzic,
2010; Saltiel, 2001; Schulze, 2004; Sowers, 2008; Stein, 2004; Wozniak, 2009; Weyer, 2001; Yamauchi, 2001):

- Promotion of intramuscular FFA clearance, and uptake of extramuscular glucose (independent of insulin action)
- Inhibition of visceral fat, insulin resistance, and hepatic glucose production

In fact, adiponectin is said to be responsible for approximately 35% of the variance in insulin responsiveness. (Borst, 2007)

Note that the amount of plasma adiponectin is inversely related to amount of body fat and plasma testosterone (Chadran, 2003). Hence, men and overweight individuals, especially those with central adiposity, produce less of it. Moreover, the amount of plasma adiponectin is found to be ethnicity related, with Asian (Abate et al., 2004) and Pima Indians (Weyer, 2001) having lower values of plasma adiponectin than Europeans of the same BMI. In Asian populations, T2DM often occurs at lower BMI and waist circumference readings. Reasons for this include the facts that Asians have high body fat, high truncal, subcutaneous and intra-abdominal fat and low muscle mass (Misra & Khurana, 2011). This propensity for higher body fat would tend to make Asian adiponectin readings lower.

**Relationship of Insulin Resistance to Lipoproteins**

Insulin resistance is also associated with the overproduction of non-HDL cholesterol and apolipoprotein B (apoB). ApoB appears to be a more accurate clinical measurement of atherogenic risk from low-density lipoproteins (LDL) than is total cholesterol and LDL (Sniderman and Cianflone, 1996). In fact, apoB measurements of greater than 1.2 g/l, accompanied by triglyceride measurements greater than 1.5 mmol/l,
are thought to be caused by insulin resistance and abdominal/truncal obesity (de Graaf, Couture, and Sniderman, 2008).

**Interrelationships among Age, Obesity, and Diabetes**

A fluctuation in insulin sensitivity occurs in the normal life cycle. Insulin resistance is more likely to occur during puberty, pregnancy, and with aging (Kahn et al., 2006). In aging, the increase in insulin resistance may be due the increase in the fat to muscle ratio. It is known that insulin sensitivity declines in 35 to 70 year olds at about the same rate and correlates closely with the increase in body fat. Moreover, in obese patients with T2DM, lowering plasma FFA levels proportionately increases insulin sensitivity.

As of the year 2000, the global prevalence of diabetes had been shown to increase with age as follows (Wild, 2004):

- 1 percent of individuals 30-34 have diabetes
- 4 percent of individuals 40-44 have diabetes
- 6 percent of individuals 50-54 have diabetes
- 12 percent of individuals 60-64 have diabetes

In the United States, the same trend is shown, with 3.7% of those 20-44 years old exhibiting diabetes; 13.7% of those 45-64 with the disease; and an estimated 26.9% of individuals 65 and over with the disease (CDC, 2011). The prevalence of the metabolic syndrome, a precursor to T2DM, has also been shown to increase consistently with age (Cornier et al., 2008).
**Ectopic Fat Deposits**

When an individual consumes more calories than he or she expends, fat accumulates. Fat may accumulate in currently existing adipocytes or the proliferation and differentiation of preadipocytes may be induced, allowing the fat to accumulate in these new cells. Individuals with a low capacity for the differentiation of preadipocytes into mature adipocytes are susceptible to the bloating of the current adipocytes and ectopic fat storage in muscle, liver, and pancreas. Individuals with this type of ectopic fat storage exhibit lowered glucose uptake in the muscles, lowered suppression of hepatic glucose production, and finally, a reduction in the secretion of insulin with prediabetes. Such individuals are very susceptible to T2DM (Ravussin & Smith, 2002). The relationship of the pattern of fat accumulation and T2DM is shown in Figure 5.

J. Denis McGarry (2002), at the Department of Internal Medicine at the University of Texas Southwestern Medical Center in Dallas, believes that insulin sensitivity appears to correlate not so much with total body fat, but with where the fat is stored. Apparently, the abnormal accumulation of fat in muscle and other tissues plays an important role in the etiology of insulin resistance. Moreover, he finds that intramuscular fat deposits correlate more tightly with insulin resistance than other measurements like BMI, waist to hip ratio, or total body fat. Defects in fat oxidation may cause fat accumulation and lipotoxicity in the skeletal muscle. This lipotoxicity in the skeletal muscle interrupts insulin signaling and causes insulin resistance (Muoio, 2010; Goodpaster and Wolf, 2004).

In addition to the ectopic placement of fat within the cells playing a role in the etiology of diabetes, studies of the Dogrib Indians of Canada's Northwest Territory...
substantiate the fact that where fat is stored within the body plays a role in the development of T2DM. Among the Dogrib, hyperglycemics did not have a significantly higher BMI than do normal individuals. However, deposits of the fat in the upper trunk were significantly higher in hyperglycemics. (Szathmary, 1983: 147-162) Therefore, when comparing the susceptibility of individuals with an “apple-shaped” physical morphology, with those with a “pear-shaped” morphology, it is the apple-shaped individual who is more prone to T2DM. Perhaps this is because of the deposition of fat around the liver and the inability of the liver to deal with such high concentrations of FFAs in the portal circulation (Kahn et al., 2006; Cornier et al., 2008), rendering it insulin-resistant.

It is known that central obesity causes high levels of circulating FFAs. In addition, individuals who exhibit central obesity produce an enzyme that converts inactive cortisone to biochemically active cortisone, resulting in more central obesity and more insulin resistance (Dushay, 2005).

![Figure 5: Outcomes that Depend on Where Fat is Stored (from Ravussin & Smith, 2002: 368)](image-url)
Role of Exercise

Independent of BMI, the amount of exercise an individual performs is directly related to whether he or she develops T2DM. This is because exercise, whether moderate or strenuous (Mayer-Davis, 1998; Hu, 1999), results in increasing the following:

- GLUT4s (Dela, 1994; Houmard, 1991)
- Mitochondria
- Plasma lipase activity (Ross, 2003)
- Intramuscular capillaries

Since the translocation of GLUT4s to the cell membrane are the transport mechanism that muscle cells use to transfer glucose from the blood to their interior, the more GLUT4s the muscle cells contain, the more glucose uptake capability the cells have. More mitochondria give the cell greater ability to burn metabolites for energy. More plasma lipase activity ensures that the adipocytes are breaking down their fats for energy as it is needed. Moreover, as more metabolites are burned, fewer FFAs are available. More capillaries ensure that nutrients pass through areas where they can be transferred into the cell's interior for metabolism. All of this has the overall effect of increasing the body's sensitivity to insulin (Wojtaszewski, 2000), which in turn reduces the following:

- Hepatic glucose production: The liver becomes more insulin sensitive. Therefore, there is a reduction in hepatic glucose production. (Segal, 1991).
- \( \gamma \)-glutamyltransferase: This is a marker for insulin resistance in the liver that decreases, indicating an increase in insulin sensitivity (Wannamethee, 2000).
- **Plasma insulin levels**: the amount of insulin that the body needs postprandially to induce insulin-stimulated glucose uptake is reduced (Rosenthal, 1983, Wasserman, 1989)

In addition to insulin-stimulated glucose uptake in muscle tissue, muscle contraction also stimulates glucose uptake (Rajan, 2002). Roberts et al (1997) found that contraction-induced glucose transport in skeletal muscle is nitric-oxide and calcium (Ca+) dependent. When muscles contract, Ca+ induces the GLUT4s to translocate to the muscle cell membranes where they can combine with plasma glucose and move it into the cell's interior for metabolism (Hayashi, 1997). This means that although a muscle may be insulin-resistant, exercise can be beneficial in the treatment of diabetes because contraction-induced glucose uptake occurs in distinct insulin-independent pathways. Indeed, exercise has been shown to be more effective than low-calorie diets (Arciero, 1999; Sakamoto, 1998) or weight loss (Ross, 2003) in inducing effective glucose disposal.

Moreover, exercise may delay the onset of insulin resistance, of T2DM, or of its debilitating effects. The risk for T2DM is inversely associated with regular exercise training (Church, 2011). In addition, regular, rigorous exercise and a diet program have been found to greatly reduce the incidence of insulin resistance (Chahwala and Arora, 2009). A Finnish study showed that the risk of type 2 diabetes was reduced by 58% when individuals included physical conditioning into their lifestyles (Tuomiletho et al., 2001). The Diabetes Prevention Program showed similar results (Tuomilehto et al., 2001). The Nurses’ Health Study, which evaluated 5125 female nurses with type 2 diabetes for 14 years, showed that increased physical activity, including regular
walking, is associated with a substantial reduction in the cardiovascular events associated with T2DM (Vasey et al., 2001). A similar study among U.S. physicians showed that exercise reduced the development of T2DM even after adjusting for BMI. Conversely, a lack of exercise and prolonged sedentary behavior has been found to be predictive of T2DM (Hu, 2001). In general, any increase in physical activity was shown to reduce the risk of T2DM in both the Finnish Diabetes Prevention Study (Laaksonen et al., 2005) and the Strong Heart Study (Fretts et al., 2009).

Insulin sensitivity increases in as few as 6 weeks after the onset of exercise training (DeFronzo, 1987). Moreover, insulin sensitivity is heightened for from 2 to 5 days after a single bout of exercise (Mikines, 1988). However, detraining reverses these results. Within 90 days after the termination of resistance training, in particular, insulin-mediated glucose uptake is decreased significantly (Andersen, 2003).

**DIET AND DIABETES**

We know that elevated plasma and intramuscular FFAs decrease insulin sensitivity. However, there is some controversy as to whether their origin is ingested fat or body fat. The high correlation between obesity and diabetes suggests that body fat is one cause of the problem. According to Jequier (2000), there are three factors that support the link between ingested fat and obesity:

1. The efficiency of nutrient utilization is higher for fat than for carbohydrates or proteins.

2. Dietary fats are preferentially stored rather than oxidized.
3. Fat induced appetite control signals are too weak and too delayed to prevent excessive fatty food intake.

Jequier’s findings are supported by other research that indicates that high-fat diets that are particularly high in saturated fats also contribute to insulin resistance (Costacou and Mayer-Davis, 2003; Marshall, 1997 and 2002; American Diabetes Association, 2002) through both general weight gain and the immediate availability of FFAs through digestion. This may be due to the fact that high fat diets reduce mitochondrial activity. Over time, mitochondrial dysfunction may result, causing a prediabetic/insulin-resistant state (Sparks et al., 2005).

Other than the fact that they contain twice as many calories per gram as carbohydrates and proteins do, monounsaturated fats seem to improve insulin sensitivity (Costacou and Mayer-Davis, 2003; American Diabetes Association, 2002). This may be because they may improve insulin signaling by increasing membrane fluidity (van Dam, 2002).

Replacement of fats in the diet with carbohydrates also has its drawbacks. High carbohydrate diets increase postprandial plasma glucose and insulin levels as well as triglycerides, and may decrease high-density lipoproteins (HDLs) (Costacou and Mayer-Davis, 2003). The higher carbohydrates are in fiber, however, the more they contribute to insulin-sensitivity, as dietary fiber intake is inversely related with insulin levels (Ludwig, 1999). This may be because fiber slows digestion and some types are indigestible and merely add bulk to the diet, rather than calories. In addition, fiber speeds the passage of food through the intestine. Foods that are high in fiber are low in their ability to load sugar into the blood stream (i.e., their glycemic index is low). A diet
high in fiber, especially cereal fiber, and low in high glycemic index foods lowers the risk for diabetes (Harvard School of Public Health website, http://www.hsph.harvard.edu/nutrionalsource/diabetesprevention). Moreover, carbohydrates like potato chips, potatoes and sugar-sweetened beverages are directly associated with permanent weight gain; while vegetables, whole grains, and fruits have an inverse relationship with it (Mozaffarian et al., 2011).

Although both proteins and carbohydrates provide four calories per gram, proteins do not have as many negative metabolic side-effects as carbohydrates (Costacou and Mayer-Davis, 2003). Long-term, though, proteins may negatively affect kidney function and aggravate diabetic nephropathy (Costacou and Mayer-Davis, 2003).

In summary, diets that prevent diabetes are primarily low-fat, with dietary carbohydrates derived from vegetables, rather than from high-sugar items, or from foods with a high glycemic index. Maintenance of protein intake at recommended levels (0.8 grams/kilogram of body weight) seems advisable (Whitney & Rolfes, 2002).

**GENETICS**

We know that factors that people can control -- like obesity and inactivity -- play a major role in the development of diabetes. But we also think that diabetes is a genetic disease because familial aggregations of the disease are common, identical twins have a high concordance for the disease, and high-risk populations, such as the Pima, exist (see Table 1). In addition, we know that diabetes, for the most part, is a polygenic disease. That is, there are many genes that interact within an individual to finally render him or her diabetic (Frayling, 2007).
In an effort to find the genes that cause the diabetic condition, geneticists have turned to linkage analysis within families and ethnic groups to help them in their quest. Linkage analysis investigates whether within groups of related individuals, diseases or other traits tend to be associated with alleles of a genetic marker. Intrafamilial or intragroup association is usually present when the disease locus and marker reside on the same chromosome close enough to each other to restrain separation in crossovers during meiosis. Linkage is a powerful technique in finding causes for Mendelian, monogenic disorders. Using linkage analysis, researchers discovered that the genetic cause for the monogenic disorder MODY (Maturity Onset Diabetes of the Young) resided in chromosomes 12q24 and 20q12-q13.1 (Menzel, 2002). As a result of genome-wide associated studies, also, a gene was discovered that increases BMI and promotes fat storage (the FTO gene), predisposing an individual to diabetes by altering BMI (Frayling, 2007). Obesity, however, is not the only cause of diabetes. That is, some obese individuals never develop the disease; and some thin individuals develop it early.

One fact that may help us to isolate the genetic basis of T2DM is that T2DM is highest in ethnic groups that have rapidly adopted Western lifestyles. Many of these groups are composed of populations which withstood periods of marked food scarcity, and developed a "thrifty" metabolism to help them lay up fatty caches to help them during times when they did not find food. This thrifty metabolism was proposed 50 years ago by the American geneticist James Neel. Neel (1962) suggested that under the conditions of feast and famine, which brought about fluctuating and often sparse food
supplies, people with a "thrifty gene" were better able to store food as fat during periods of abundance.

Because North American Indians fit the profile for those who would have developed this "thrifty" gene, perhaps we should study them to understand this anomaly. For example, the Pima have the highest risk for T2DM, with over 70% of the US population of Pima between ages 45 and 74 suffering from the disease. In the Pima population, genetic markers on chromosome 4q and 7q have been linked to insulin resistance (Narayan, 1997). In addition, a mutation in the beta-3-adrenergic-receptor gene, which regulates lipolysis and thermogenesis in adipocytes, has been shown to cause a slower burning of calories in this population. Moreover, in an unexpected discovery among the Objiway and Cree Indians of Canada, researchers (Young, 2000) have found a mutation in one gene that promotes fat storage. A person with one copy of the gene has two times the risk of developing diabetes. If the individual has inherited two copies of the gene, one from the mother the other from the father, the risk is 15 times as great.

Since Mexican Americans have a 2.72 greater risk of developing T2DM (Haffner, 1991), they form another candidate population for the "thrifty" gene. For the past 25 years, Craig Hanis, Ph.D., of the University of Texas, Houston, has been working with 330 pairs of diabetic brothers and sisters from the Mexican American community in Starr County, Texas. In 1996, Graeme Bell, Ph.D., of the University of Chicago, collaborated with the Hanis team and was able to demonstrate a linkage between an increased risk of diabetes in this population and an unknown gene located near one end of chromosome 2, which they called T2DM1. The T2DM1 gene codes for a new
protein, calpain 10, one of a family of calpain proteases, which regulate the function of other proteins by snipping off pieces, altering the protein and making it more or less active.

This calpain 10 anomaly was also found among the Pima (Baier, 2000). Pima Indians with a particular version of the calpain 10 gene demonstrate many of the characteristics associated with a more frugal energy balance, such as a decreased sleeping metabolic rate and a tendency to hoard rather than burn glucose.

The calpain 10 mutation does not cause diabetes by itself, but interacts with lifestyle factors such as diet, exercise and other genes. It is said to account for about 14% of the T2DM in Mexican American communities and about 4% of the T2DM in European populations (Horikawa et al., 2000; Wade, 2001).

The gene that controls adiponectin has also been shown to be T2DM-related. Recent genome-wide scans have mapped a susceptibility locus for T2DM to chromosome 3q27, where the gene encoding for adiponectin is located. Decreased adiponectin correlates with increased insulin resistance. Adiponectin decreases insulin resistance by decreasing intramuscular lipids and hepatic glucose production. Certain ethnic groups have been shown to have lower adiponectin plasma levels. Lower adiponectin could also be the culprit among of ethnic groups that have been shown to be T2DM-prone (see above).

It has also been determined that the susceptibility of pancreas’ beta-cells to failure is genetic. When individuals experience a high-fat diet and little physical exercise, they become insulin-resistant, which increases insulin demands made on the beta-cells. In T2DM-prone individuals, the combination of increased insulin demand...
and a detrimental diet and exercise pattern result in beta-cell dysfunction and decreased beta-cell mass, which leads to IGT, and eventually T2DM (Kahn et al., 2006).

**BEHAVIORAL FACTORS**

There is strong evidence that genetic and environmental factors are both important in the etiology of T2DM. So far, genetic factors we can identify account for only a maximum of 14% of the cases reported for given ethnic groups. However, ethnic groups not only share genes, but can share behavioral patterns (Wareham, Franks, and Harding, 2002: 557). Moreover, 90% of individuals with T2DM are obese (see above). Therefore, instead of attributing the development of diabetes to genes alone, we might look to over-abundant, but poor, diets and a lack of physical activity (characteristics that ethnic groups might share) as the major causes of the disease. These, at least, enable the fat creation and ectopic fat placement that mark insulin resistance and the beginnings of T2DM.

To substantiate this conclusion, we might point to public health studies of lifestyles of minority people in the US that indicate the following behavior patterns: (Hogue et al., 2000)

- Only 40% of adult minority Americans eat a healthy, balanced diet (i.e, one adhering to the RDA guidelines) 4 to 7 days per week.
- African Americans are 0.6 times as likely as whites to eat a healthy, balanced diet.
- Hispanic Americans are 0.8 times as likely as whites to eat a healthy, balanced diet.
- Only 24% of the minorities sampled said that they exercised hard at least four days a week.
In the sample, married women were only 0.6 times as likely as unmarried women to report a routine, physical exercise schedule.

Low-income households are 0.3 times as likely to exercise regularly as middle-income households.

African American men reported exercising 0.7 times as much as white men. (There was no significant difference between Hispanic and white men.)

Unemployed men were only 0.3 times as likely to exercise as those who were employed.

From these findings, we can look forward to a diabetic population that is increasingly minority, married, poor, and physically inactive, which is quite consistent with the current and projected demographics. Unlike Susrata, who found diabetes among the rich in ancient times (Stachura, 1996: 3), in America, for the most part, we find it among our poor minorities, who not only live in a way that renders them more susceptible to the disease, but who may also have genes that leave them with a propensity for it (CDC, 2011).

Of course, there are subtleties that color the broad strokes of this conclusion. For example:

- There is evidence that Mexican immigrants to America are less likely to become obese than the Mexican Americans who were born here (Sundquist 2000).

- Mexican Americans who do not speak English are more likely to be obese than those who have been acculturated and do speak English (Sundquist 2000).
• The longer that Puerto Rican immigrants live in America, the more likely they are to develop obesity (Himmelgreen, 2004).

However, even considering nuances like these, we are left with the fact that 12 percent of Latino/Mexican Americans and 13 percent of African Americans develop the disease before they die (CDC, 2011). Much of the reason lies in lifestyle, while some of it lies in genes.

SUMMARY

This section has examined our biological heritage as primates and *Homo sapiens* and articulated our current understanding of the causes of the diabetes. At the nexus of the two lie the physical characteristics that allowed us to adapt to the harsh existence of the sporadic food and physical exertion that came with the foraging lifestyle. Yet, these same characteristics render us obese and insulin-resistant now that rich foods come easy and our greatest physical exertion consists of getting out of bed in the morning. These physical characteristics are species-wide, but they vary enough to make some ethnic groups more T2DM-prone than others. They include things like:

• *Length and efficiency of the small intestine.* The more efficient this organ is, the more food is digested and the more calories are available for energy metabolization or storage.

• *Level of adipose tissue hormones.* Some of these slow the metabolism; others make it more efficient (adiponectin). All may have been adaptive during our foraging past. Some are maladaptive now (TNFa and IL-6).
• *Level of ectopic fat storage.* Whether individuals store fat centrally effects how prone they are to be insulin-resistant and to develop diabetes later in life. Hormone levels, especially of cortisol, could be responsible and could vary by group.

• *Beta-cell strength.* Not all obese or insulin-resistant individuals develop T2DM. Only the ones whose beta-cells are too weak to keep up with the ever increasing insulin demand do. How robust the beta-cells are could be another physical characteristic that could vary by group.

To these physical characteristics, we must add the behavioral characteristics that contribute to obesity. These include:

- A high-calorie, high fat diet
- A lifestyle that includes little exercise

These behavioral characteristics, like their physical counterparts, can vary by group and make some ethnic groups more T2DM-prone than others.
Chapter 3: Study Population and Methods
To test insulin resistance in the Hispanic community, I chose to study Hispanic, male meatpackers in Dodge City, Kansas. Dodge City is the site of two meatpacking facilities – Cargill and National.

**STUDY POPULATION**

All study participants had to be:

- Meatpackers
- Between the ages of 18 and 55.
- Hispanic
- Male
- Non-diabetic (as measured by the Bayer Hemoglobin A1C test, a non-fasting test with immediate results). That is, if the A1C test results show that the meatpacker's hemoglobin is $\geq 6.5\%$ glycolated (Buell C 2007, ADA 2009), the meatpacker was referred to a physician or clinic for possible diabetes, and he was disqualified from the study as this indicated that he has had a hyperglycemic episode during the past 2 or 3 months prior to the test (Goldstein et al. 2004.)
- Sedentary outside of work. To determine this, the men were asked whether they exercise outside of work more than 30-40 minutes twice a week. When answering this initial question, those who indicated that their exercise exceeded this limit were excluded from the study.
- Performing their current job more than three months to make sure their exertion at work during the study period is representative and that their A1C test reflects their time on their current job;
• Low and moderate risk individuals, as indicated by all "No" answers to the questions on the PAR-Q portion of the "Cardiovascular Risk Assessment" questionnaire (available in Appendix B). That is, high risk individuals were excluded from the study.

• Exhibiting a systolic blood pressure of \( \leq 160 \) and a diastolic blood pressure \( \leq 100 \).

Although high blood pressure alone does not place an individual in the high-risk category according to the American College of Sports Medicine (ACSM, 2009), the ACSM views high blood pressure as "a unique risk factor that may be aggravated by acute exercise." Therefore, if the individual's systolic blood pressure was more than 140 (but less than or equal to 160) and/or his diastolic blood pressure was more than 90 (but less than or equal 100), a physician was present during the Rockport exercise test for physical fitness. Individuals with a systolic blood pressure greater than 160 or a diastolic blood pressure greater than 100 were excluded from the study.

The first language of all of the participants was Spanish. Most were born in Mexico or Central America and immigrated to the United States of America as adults.

**STUDY PLACE AND TIME**

The study took place on Sundays at Our Lady of Guadalupe Cathedral, Dodge City, Kansas, 67801. Male meatpackers were recruited through announcements at the Spanish masses, advertising in church newsletters, the local Hispanic newspaper, the local Hispanic grocery store, and on local Dodge City Hispanic radio stations (see Appendix D for the advertising used to attract participants). Positioning the testing
facilities in Dodge City took advantage of the presence of two nearby meatpacking facilities -- Cargill and National.

LOGISTICS OF THE STUDY

Data were collected for this study between February 15, 2011 and June 26, 2011. The study is a cross-sectional, observational one that actively recruited the study population (see above). Participants performed meatpacking jobs that were classified as Light, Moderate, or Heavy. These job categories were the result of ergonomic studies conducted by the meatpacking industry for jobs performed there.

For participants, the study progressed through three sessions:

- **Session 1** – During this session, the church receptionist determined whether the individual was qualified in as much as he was sedentary outside of work and whether he had been performing his current job for three months. She explained the consent form (see Appendix B: Forms) to qualified individuals, one page at a time. One at a time, participants who had signed the consent form proceeded to the principal investigator who logged them into the study, gave them an ID number that would later be associated with their health data and blood test results, and measured their height, weight, and waist and hip circumferences. Next, participants proceeded to a bilingual nurse who determined whether the participant did light, moderate, or heavy meatpacking work. The nurse asked questions to assess the risk level for each participant, took his blood pressure, and both administered and provided participants with the results the hemoglobin A1C test (see pages 1 and 2 of the Nurses Interview Form in Appendix B). The nurse also informed the participant of the time he was expected for his second and third sessions and asked him to
record his diet (see Appendix B: Forms) for three consecutive days before arriving for session 2.

- **Session 2** – During this session, the principal investigator administered the modified Rockport walking test to pre-qualified subjects. In addition, the bilingual nurse collected the completed diet records, took blood samples and interviewed participants to obtain some medical and personal history and an assessment of their own physical fitness and activity (see pages 3 through 6 of the Nurses Interview form in Appendix B). Participants received a $15 grocery certificate when they completed the walking test. A volunteer from the Dodge City medical center transported the blood samples, centrifuged them and sent them for analysis to the CTRC laboratory at the Anschutz Medical Center in Denver.

- **Session 3** – During this session, each participant received the results of his blood and physical fitness tests (i.e., Rockport Walking test) in a sealed envelope. An explanation of the stages of diabetes and the need to change their diet and exercise regimen was provided, along with a handout on diabetes prevention prepared by the National Diabetes Education Program. (See Appendix C: Feedback).

**PROCEDURES**

The following explains the procedures that were used to measure glycolated hemoglobin, physical fitness, body size, blood pressure, and diabetes-related hormones for each study participant.

**Glycolated hemoglobin.** Diabetes status was assessed during session 1 through the Bayer A1C test. This is a finger stick test that the bilingual nurse administered. It took 5 minutes for the test to provide results and no fasting was required. Outcomes of 6.5% or
greater indicate that the individual is diabetic. Outcomes between 5.7 and 6.4% indicate that the individual is prediabetic (Lorenzo et al., 2010). In addition, those with outcomes between 6.0 and 6.4% have 16 times the normal risk of developing diabetes in the future; outcomes of between 5.5 and 5.9 % indicate 5 times the normal risk; those between 5.0 and 5.4% indicate twice the normal risk (Cheng et al., 2011).

**Physical fitness estimates.** Here “physical fitness” is synonymous with “aerobic, cardiovascular fitness,” which is essentially the maximal aerobic capacity, or VO$_{2max}$. In the study, VO$_{2max}$ was estimated in two ways.

- First, it was estimated using the formula of George et al (1997). See Appendix A.
- Second, we estimated the VO$_{2max}$ from a modified Rockport walking test of physical fitness (ACSM, 2009).

The Rockport walking test is a one-mile walk where the participant is instructed to self-select a fast, but less than maximal, steady exercise pace. (On a 10-point scale, the investigator indicated that the participant was to walk at a level-6 or “fuerte” pace.) The Rockport walking test gives valid and reliable estimates of physical fitness across a broad population and is meant to be administered in the field. Since the individual’s heart rate plateaus after three minutes of walking, a quarter-mile test is just as valid as the extended full-mile test (Greenhalgh et al., 2001). Here the participants took the quarter-mile version of the test. (See Appendix A.) The Rockport walking test was administered during session 2.

**Body size (anthropometric) and blood pressure measurements.** During sessions 1 and 2, the bilingual nurse measured the participant’s blood pressure, and thickness of the triceps and subscapula skinfolds. Skinfold measurements were ascertained using
standard protocols (Lohman et al., 1988). The mean arterial blood pressure was calculated \((DBP + (SBP-DBP)/3)\) (Sesso et al., 2000). The principal investigator measured the participants height, weight and waist and hip circumferences during session 1.

*Diabetes-related hormone measurements.* Clinical tests on each participant's blood samples included an adiponection assay, an apoB assay, a lipid panel, and tests for levels of fasting insulin and fasting glucose. All these blood analyses occurred at the CTRC at the University of Colorado Medical Center. The bilingual nurse and a volunteer from the local hospital drew the blood for these tests the Sunday morning after the meatpackers completed their 3-day diet recording period. A volunteer from the local medical center transported the blood samples to the Dodge City medical center, where the blood was centrifuged and frozen. Frozen blood samples were sent every month to the CTRC for testing. One set of samples drawn from each individual was kept at the medical center in case the other set (used for testing at the CTRC laboratory in Denver) was lost or broken.

Note that fasting insulin levels of \(>12.2\) mU/l or \(15\) ulU/mL indicate insulin resistance in non-diabetic individuals. In addition, individuals with normal glucose metabolism have a fasting glucose levels of \(<100\) mg/dL; prediabetes is indicated by fasting glucose levels of \(\geq100\) and \(\leq126\) mg/dL; those with diabetes have fasting glucose levels of \(>126\). The average adiponectin level for men is \(7.7\) µg/ml. Levels below this would tend toward insulin-resistance (Weyer et al., 2001). Levels of apoB above \(1.2\)g/l would indicate high atherogenic risk. Also, if triglycerides exceed \(1.5\) mmol/l, regardless of apoB level, the metabolic syndrome and accompanying insulin
resistance may be indicated (Sniderman et al., 2010). The blood for the tests was drawn during session 2.

**INTERVIEWS**

Since literacy could not be assured, interviews were used, instead of questionnaires. A bilingual nurse conducted an interview with each meatpacker during sessions 1 and 2 to obtain his demographic information (i.e., age, level of physical training, and level of education) and get information related to the his Perceived Functional Ability and Physical Activity Rating. The interview forms that the bilingual nurse used appear in Appendix B: Forms.

<table>
<thead>
<tr>
<th>Table 10: Study Statistical Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Statistical Test</strong></td>
</tr>
<tr>
<td>Kruskal-Wallis Chi-square</td>
</tr>
<tr>
<td>Chi-Square</td>
</tr>
<tr>
<td>Spearman Correlation Coefficients</td>
</tr>
<tr>
<td>Wilcoxon Exact Test</td>
</tr>
<tr>
<td>Linear Regression</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
DATA ANALYSIS

Table 10 lists the statistical tests that were performed on the variables listed using the SAS statistical package, version 9.2.
Chapter 4: Results
As planned, this study was performed at Our Lady of Guadalupe Cathedral, Dodge City, Kansas. Although an effort was made to recruit participants directly from the meatpacking facilities and from a local Hispanic grocery store, the overwhelming majority of the participants came from the pool of cathedral parish members. The study was conducted from February through June of 2011 on Sunday mornings and afternoons at the cathedral.

In total, 46 participants were recruited; 43 of them went through session 1, where data on their height, weight, waist and hip circumference, blood pressure, family history and A1C readings were obtained. Of these 43 participants, 35 remained interested enough to complete session 2, when they submitted their diet records, and took the Rockport test for physical fitness and the George self-assessment of physical fitness. At session 2, their blood was also drawn for total cholesterol, triglyceride, HDL, fasting insulin, fasting glucose, adiponectin, and apoB testing. The blood samples were immediately centrifuged and frozen at the local Dodge City medical center, and later sent to the CTRC laboratory at the University of Colorado medical center for analysis. As the results were emailed back from the laboratory, session 3 was scheduled where the participants received the results of their blood tests and listened to a presentation on the etiology of diabetes as it related to their diet and exercise patterns (see Appendix C: Feedback).

**TYPES OF RESULTS**

Two types of results were obtained from this study:

- Those that characterize the differences among the groups represented in the study; and
Those that characterize the study population in general.

The remainder of this chapter describes the results of the statistical analyses used to obtain these results.

**Differences Among the Groups**

During this study, the male meatpackers who participated in the study were analyzed by:

- Job Category; and
- Physical Fitness Category.

Both analyses were necessary to thoroughly test the hypotheses on which the study was based. To state the hypotheses again, they are:

- Insulin resistance is inversely related to the physical fitness resulting from job related activities.
- Adiponectin level is positively related to the physical fitness resulting from job related activities.
- ApoB level is inversely related to the physical fitness resulting from job related activities.

**Analyses by Job Category**

There are many jobs within meatpacking. Some have been ergonomically classified as requiring light physical exertion; some require moderate exertion; others require heavy exertion. The following table lists the jobs that were known to the principal investigator to fall within these classifications at the time of the study:
Table 11: Meatpacking Jobs

<table>
<thead>
<tr>
<th>Activity Level/Division</th>
<th>Fabrication</th>
<th>Kill Floor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heavy</td>
<td>chuck boner, tender puller, strip boner, re-hang rounds, forequarter marker, boxer</td>
<td>gutter, split saw operator, fold/stack hides, chisler/templer, face plates, 1&amp;2 legger, cheeker</td>
</tr>
<tr>
<td>Moderate</td>
<td>navel boner, brisket boner, top butt boner, strip boner, skirt trimmer, top butt trimmer, strip trimmer, brisket trimmer, chuck trimmer, loose meat bagger</td>
<td>air knife operator, weas and rodder, fat puller, head hanger</td>
</tr>
<tr>
<td>Light</td>
<td>CO₂ injector, low temp/beef tissue belt, finger meat bagger, defect picker/sorter, spinal cord processor, frock sewer, upgrade trimmer, navel trimmer</td>
<td>case ready crew, vacuum operator, ear tickets, intestine flusher, yard worker, spinal cord worker, liver wrapper, contamination counter, hock processor, paper on/off worker</td>
</tr>
</tbody>
</table>

Note that some of the jobs are performed on the kill floor in the process of butchering the animal for delivery to the public. Other jobs are performed in the process of fabrication, where the animal parts are boned, trimmed, and stuffed for packaging.

Of the 35 men who completed the study, only 30 reported exercising no more than 80 minutes per week outside of their normal work activities (and, were, presumably, “sedentary outside of work”). Of these 30, only 24 had jobs that were ergonomically studied and included within the table above. The other 6 had jobs within the maintenance, shipping, quality control, supervisory, and training functions at the meatpacking plants. Typically, the workload of these 6 participants varied from day to day, from week to week, and from season to season. Therefore, they were excluded from the studies performed by job category.

Because the sample size was small and the results were not normally distributed and could not be transformed to be normally distributed, non-parametric tests were performed. The following tables summarize the results of Kruskal-Wallis (an ANOVA
substitute), Wilcoxon Exact (a t-test substitute), and correlation tests that analyze the relationships between the job categories listed in Table 11 and the results of the fasting insulin, adiponectin, and apoB blood tests. None of the outcomes showed significance.

**Table 12: Kruskal-Wallis Results Comparing Blood Tests by Job Categories**

<table>
<thead>
<tr>
<th>Variable Tested</th>
<th>Light Exertion</th>
<th>Moderate Exertion</th>
<th>Heavy Exertion</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>6</td>
<td>10</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>16.83(±9.88)</td>
<td>16.7(±6.25)</td>
<td>16.13(±7.02)</td>
<td>0.928</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>8.78(±5.01)</td>
<td>6.2(±3.37)</td>
<td>7.0(±4.70)</td>
<td>0.544</td>
</tr>
<tr>
<td>ApoB</td>
<td>87.5(±12.42)</td>
<td>76.1(±11.44)</td>
<td>85.0(±15.26)</td>
<td>0.226</td>
</tr>
<tr>
<td>Physical Fitness</td>
<td>28.61(±2.91)</td>
<td>25.77(±12.04)</td>
<td>29.89(±6.5)</td>
<td>0.762</td>
</tr>
</tbody>
</table>

**Table 13: Wilcoxon Test Results Comparing Blood Tests for Light and Heavy Job Categories**

<table>
<thead>
<tr>
<th>Variable Tested</th>
<th>Light Exertion</th>
<th>Heavy Exertion</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting Insulin</td>
<td>16.83(±9.88)</td>
<td>16.13(±7.02)</td>
<td>0.489</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>8.78(±5.01)</td>
<td>7.0(±4.70)</td>
<td>0.377</td>
</tr>
<tr>
<td>ApoB</td>
<td>87.5(±12.42)</td>
<td>85.0(±15.26)</td>
<td>0.465</td>
</tr>
<tr>
<td>Physical Fitness</td>
<td>28.61(±2.91)</td>
<td>29.89(±6.5)</td>
<td>0.194</td>
</tr>
</tbody>
</table>

**Table 14: Wilcoxon Results Comparing Blood Tests for Light and Moderate/Heavy Job Categories**

<table>
<thead>
<tr>
<th>Variable Tested</th>
<th>Light Exertion</th>
<th>Moderate/Heavy Exertion</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>6</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>16.83(±9.88)</td>
<td>16.44(±6.44)</td>
<td>0.416</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>8.78(±5.01)</td>
<td>6.55(±3.87)</td>
<td>0.193</td>
</tr>
<tr>
<td>ApoB</td>
<td>87.5(±12.42)</td>
<td>80.06(±13.63)</td>
<td>0.150</td>
</tr>
<tr>
<td>Physical Fitness</td>
<td>28.61(±2.91)</td>
<td>27.61(±9.93)</td>
<td>0.372</td>
</tr>
</tbody>
</table>

**Table 15: Spearman Correlation Coefficients and Probability Results Comparing Blood Tests for Job Categories**

<table>
<thead>
<tr>
<th>Job Category</th>
<th>N</th>
<th>Fasting Insulin vs Physical Fitness Coeff</th>
<th>P value</th>
<th>Fasting Insulin vs Physical Fitness Coeff</th>
<th>P value</th>
<th>Adiponectin vs Physical Fitness Coeff</th>
<th>P value</th>
<th>ApoB vs Physical Fitness Coeff</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light</td>
<td>6</td>
<td>.0857</td>
<td>.436</td>
<td>.3714</td>
<td>.234</td>
<td>.4058</td>
<td>.2124</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>10</td>
<td>-.3547</td>
<td>.157</td>
<td>.3091</td>
<td>.1924</td>
<td>.0307</td>
<td>.4665</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heavy</td>
<td>8</td>
<td>-.0240</td>
<td>.4775</td>
<td>-.2619</td>
<td>.2655</td>
<td>-.7306</td>
<td>.0198</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate &amp; Heavy</td>
<td>18</td>
<td>-.3264</td>
<td>.093</td>
<td>.1404</td>
<td>.289</td>
<td>-.1060</td>
<td>.3377</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Analyses by Physical Fitness Category

In analyzing the results by job category, it became apparent that the jobs within each category appeared along a continuum rather than in distinct categories. That meant, that in many cases the categories “bled” into another category and the standard deviation of one category severely encroached upon the standard deviation of the adjacent category. To overcome this problem, physical fitness categories were created using the percentile measurements of physical fitness shown in Table 16 as follows:

- $VO_{2\text{max}}$ category 1 = 0 – 26.799 ml/kg.min (percentiles 1-10 for ages 60 and above)
- $VO_{2\text{max}}$ category 2 = 26.8 – 37.899 ml/kg.min (percentiles 10-20 for ages 20 to 29)
- $VO_{2\text{max}}$ category 3 = 37.9 – 50 ml/kg.min (percentiles >20 for ages 20 to 29)

The results of the fasting insulin, adiponectin, and apoB tests were analyzed using these categories. The categories were based on percentiles published by the American College of Sports Medicine (ACSM, 2009).

Table 16: Percentile Values for Maximal Oxygen Uptake (ml/kg.min) in Men

<table>
<thead>
<tr>
<th>Percentile</th>
<th>20-29</th>
<th>30-39</th>
<th>40-49</th>
<th>50-59</th>
<th>60+</th>
</tr>
</thead>
<tbody>
<tr>
<td>90</td>
<td>55.1</td>
<td>52.1</td>
<td>50.6</td>
<td>49.0</td>
<td>44.2</td>
</tr>
<tr>
<td>80</td>
<td>52.1</td>
<td>50.6</td>
<td>49.0</td>
<td>44.2</td>
<td>41.0</td>
</tr>
<tr>
<td>70</td>
<td>49.0</td>
<td>47.4</td>
<td>45.8</td>
<td>41.0</td>
<td>37.8</td>
</tr>
<tr>
<td>60</td>
<td>47.4</td>
<td>44.2</td>
<td>44.2</td>
<td>39.4</td>
<td>36.2</td>
</tr>
<tr>
<td>50</td>
<td>44.2</td>
<td>42.6</td>
<td>41.0</td>
<td>37.8</td>
<td>34.6</td>
</tr>
<tr>
<td>40</td>
<td>42.6</td>
<td>41.0</td>
<td>39.4</td>
<td>36.2</td>
<td>33.0</td>
</tr>
<tr>
<td>30</td>
<td>41.0</td>
<td>39.4</td>
<td>36.2</td>
<td>34.6</td>
<td>31.4</td>
</tr>
<tr>
<td>20</td>
<td>37.8</td>
<td>36.2</td>
<td>34.6</td>
<td>31.4</td>
<td>28.3</td>
</tr>
<tr>
<td>10</td>
<td>34.6</td>
<td>33.0</td>
<td>31.4</td>
<td>29.9</td>
<td>26.7</td>
</tr>
</tbody>
</table>

Because the categories were not dependent upon job categories, more participants were represented. Two analyses by fitness categories were performed: one for those who exercised no more than 80 minutes per week (see Tables 17 through 20);
and one for all the study participants, including those who exercised more than 80 minutes per week (see Tables 21 through 24).

Table 17: Kruskal-Wallis Results Comparing Blood Tests by Fitness Categories – Exercises No More Than 80 Minutes per Week

<table>
<thead>
<tr>
<th>Variable Tested</th>
<th>VO2-1</th>
<th>VO2-2</th>
<th>VO2-3</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>14</td>
<td>12</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>20.71(±10.12)</td>
<td>15.5(±7.11)</td>
<td>16.25(±7.41)</td>
<td>0.366</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>5.3(±2.66)</td>
<td>7.66(±4.99)</td>
<td>10.2(±2.03)</td>
<td><strong>0.046</strong></td>
</tr>
<tr>
<td>ApoB</td>
<td>84.64(±16.84)</td>
<td>84.83(±10.15)</td>
<td>73.75(±13.5)</td>
<td>0.357</td>
</tr>
<tr>
<td>Physical Fitness</td>
<td>19.17(±4.91)</td>
<td>31.57(±3.24)</td>
<td>40.48(±2.51)</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>

Table 18: Wilcoxon Test Results Comparing Blood Tests for VO2-1 and VO2-3 – Exercised No More Than 80 Minutes per Week

<table>
<thead>
<tr>
<th>Variable Tested</th>
<th>VO2-1</th>
<th>VO2-3</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>14</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>20.71(±10.12)</td>
<td>16.25(±7.41)</td>
<td>0.211</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>5.3(±2.66)</td>
<td>10.2(±2.03)</td>
<td><strong>0.006</strong></td>
</tr>
<tr>
<td>ApoB</td>
<td>84.64(±16.84)</td>
<td>73.75(±13.5)</td>
<td>0.156</td>
</tr>
</tbody>
</table>

Table 19: Wilcoxon Results Comparing Blood Tests for VO2-1 and VO2-2&3 – Exercised No More Than 80 Minutes per Week

<table>
<thead>
<tr>
<th>Variable Tested</th>
<th>VO2-1</th>
<th>VO2-2&amp;3</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>14</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>20.71(±10.12)</td>
<td>15.68(±6.94)</td>
<td><strong>0.088</strong></td>
</tr>
<tr>
<td>Adiponectin</td>
<td>5.3(±2.66)</td>
<td>8.3(±4.51)</td>
<td><strong>0.025</strong></td>
</tr>
<tr>
<td>ApoB</td>
<td>84.64(±16.84)</td>
<td>82.06(±11.69)</td>
<td>0.455</td>
</tr>
<tr>
<td>Physical Fitness</td>
<td>20.71(±10.12)</td>
<td>33.80(±4.98)</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>

Table 20: Spearman Correlation Coefficients and Probability Results Comparing Blood Tests for Fitness Categories – Exercised No More Than 80 Minutes per Week

<table>
<thead>
<tr>
<th>Fitness Category</th>
<th>N</th>
<th>Fasting Insulin vs Physical Fitness</th>
<th>Adiponectin vs Physical Fitness</th>
<th>ApoB vs Physical Fitness</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Coeff</td>
<td>P value</td>
<td>Coeff</td>
</tr>
<tr>
<td>VO2-1</td>
<td>14</td>
<td>-0.044</td>
<td>0.881</td>
<td>-0.3451</td>
</tr>
<tr>
<td>VO2-2</td>
<td>12</td>
<td>-0.084</td>
<td>0.794</td>
<td>-0.599</td>
</tr>
<tr>
<td>VO2-3</td>
<td>4</td>
<td>-0.8</td>
<td>0.10</td>
<td>-0.8</td>
</tr>
<tr>
<td>VO2-2&amp;3</td>
<td>16</td>
<td>-0.009</td>
<td>0.495</td>
<td>-0.087</td>
</tr>
<tr>
<td>Overall</td>
<td>30</td>
<td><strong>-0.244</strong></td>
<td>0.097</td>
<td>0.255</td>
</tr>
</tbody>
</table>
Table 21: Kruskal-Wallis Results Comparing Blood Tests by Fitness Categories – All Participants

<table>
<thead>
<tr>
<th>Variable Tested</th>
<th>VO2-1</th>
<th>VO2-2</th>
<th>VO2-3</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>15</td>
<td>15</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>21.60(±10.34)</td>
<td>14.80(±6.65)</td>
<td>15.60(±6.5)</td>
<td>.155</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>5.42(±2.60)</td>
<td>7.73(±4.52)</td>
<td>10.28(±1.77)</td>
<td>.019</td>
</tr>
<tr>
<td>ApoB</td>
<td>86.73(±18.13)</td>
<td>83.47(±13.24)</td>
<td>75.60(±12.40)</td>
<td>.510</td>
</tr>
<tr>
<td>Physical Fitness</td>
<td>19.33(±4.77)</td>
<td>31.98(±3.34)</td>
<td>40.45(±2.17)</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>

Table 22: Wilcoxon Test Results Comparing Blood Tests for VO2-1 and VO2-3 – All Participants

<table>
<thead>
<tr>
<th>Variable Tested</th>
<th>VO2-1</th>
<th>VO2-3</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>15</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>21.60(±10.34)</td>
<td>15.60(±6.5)</td>
<td>.135</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>5.42(±2.60)</td>
<td>10.28(±1.77)</td>
<td>.007</td>
</tr>
<tr>
<td>ApoB</td>
<td>86.73(±18.13)</td>
<td>75.60(±12.40)</td>
<td>.653</td>
</tr>
</tbody>
</table>

Table 23: Wilcoxon Results Comparing Blood Tests for VO2-1 and VO2-2&3 – All Participants

<table>
<thead>
<tr>
<th>Variable Tested</th>
<th>VO2-1</th>
<th>VO2-2&amp;3</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>15</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>21.60(±10.34)</td>
<td>15.00(±6.46)</td>
<td>.033</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>5.42(±2.60)</td>
<td>8.37(±4.12)</td>
<td>.017</td>
</tr>
<tr>
<td>ApoB</td>
<td>86.73(±18.13)</td>
<td>81.50(±13.18)</td>
<td>.271</td>
</tr>
<tr>
<td>Physical Fitness</td>
<td>19.33(±4.77)</td>
<td>34.10(±4.84)</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>

Table 24: Spearman Correlation Coefficients and Probability Results Comparing Blood Tests for Fitness Categories – All Participants

<table>
<thead>
<tr>
<th>Fitness Category</th>
<th>N</th>
<th>Fasting Insulin vs Physical Fitness</th>
<th>Adiponectin vs Physical Fitness</th>
<th>ApoB vs Physical Fitness</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Coeff</td>
<td>P value</td>
<td>Coeff</td>
</tr>
<tr>
<td>VO2-1</td>
<td>15</td>
<td>-0.023</td>
<td>.4672</td>
<td>-0.321</td>
</tr>
<tr>
<td>VO2-2</td>
<td>15</td>
<td>-0.03</td>
<td>.459</td>
<td>-0.465</td>
</tr>
<tr>
<td>VO2-3</td>
<td>5</td>
<td>-0.70</td>
<td>.094</td>
<td>-0.70</td>
</tr>
<tr>
<td>VO2-2&amp;3</td>
<td>20</td>
<td>0.008</td>
<td>.486</td>
<td>0.017</td>
</tr>
<tr>
<td>Overall</td>
<td>35</td>
<td>-0.293</td>
<td>.044</td>
<td>.282</td>
</tr>
</tbody>
</table>

RESULTS FOR WHOLE STUDY

Other than the group analysis by fitness category that included all participants, above, additional results obtained in this study characterize the whole population under
study. For these results, no one was excluded from the statistical analyses and all 35 of the participants who completed session 2 are represented in the results.

<table>
<thead>
<tr>
<th>Table 25: Variable Means, Medians, and Ranges</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
</tr>
<tr>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>BMI</td>
</tr>
<tr>
<td>Waist Circumference</td>
</tr>
<tr>
<td>Waist/Hip Ratio</td>
</tr>
<tr>
<td>Systolic BP</td>
</tr>
<tr>
<td>Diastolic BP</td>
</tr>
<tr>
<td>Fasting Insulin</td>
</tr>
<tr>
<td>Fasting Glucose</td>
</tr>
<tr>
<td>Adiponectin</td>
</tr>
<tr>
<td>Total Cholesterol</td>
</tr>
<tr>
<td>Triglycerides</td>
</tr>
<tr>
<td>HDL</td>
</tr>
<tr>
<td>LDL</td>
</tr>
<tr>
<td>ApoB</td>
</tr>
<tr>
<td>Physical Fitness Self-Assessment</td>
</tr>
<tr>
<td>Rockport Physical Fitness Assessmen</td>
</tr>
</tbody>
</table>

Of particular interest are the descriptive statistics shown in Table 25, above, the analyses of the A1C test results, correlation analyses, and linear and multiple regression
tests performed on the data collected. Also, included are family history statistics for diabetes and heart disease.

**A1C Test Results**

The A1C test was performed on everyone who came to session 1. The test identified those who already had diabetes so they could be excluded from the study. It also served the purpose of recruiting men to the study since it provided results within 5 minutes. Therefore, all 43 participants took the A1C test. For this population, the average reading was 5.48; the median was 5.6; and the mode was 5.6.

The A1C results identify people at a particular risk for developing diabetes. That is, given the risk categories shown below, individuals have the indicated chance of developing the disease (Cheng et al., 2011):

- **A1C category 0**: < 5.0 – normal chance of developing diabetes
- **A1C category 1**: 5.0-5.4 – twice the normal chance of developing diabetes
- **A1C category 2**: 5.5-6.0 – 5 times the normal chance of developing diabetes
- **A1C category 3**: 6.1-6.4 – 16 times the normal chance of developing diabetes

For this population, 4 men fell into category 0; 16 were classed as category 1; 19 as category 2; and 4 as category 4. These A1C categories were analyzed by both job and physical fitness categories using chi-square analysis and neither analysis yielded significant results. The P value for the Job Category vs A1C category was 0.33; while the P value for the Fitness Category vs A1C category was 0.71.
Correlation Results

Correlation analyses were performed on every possible pair of study variables. The significant (P values less than or equal to .05) and notable (P values between .05 and .10) correlation probabilities and the correlation coefficients on which they are based as given in Table 26. See Appendix E for a correlation matrix.

Table 26: Significant and Notable Spearman Correlation Analyses between Variable Pairs (N=35)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Correlating Variable</th>
<th>Correlation Coeff</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Physical Fitness Self-Assessment</td>
<td>0.239</td>
<td>.083</td>
</tr>
<tr>
<td></td>
<td>Rockport Assessment</td>
<td>0.263</td>
<td>.063</td>
</tr>
<tr>
<td></td>
<td>Total Cholesterol</td>
<td>-0.2717</td>
<td>.057</td>
</tr>
<tr>
<td></td>
<td>ApoB</td>
<td>-0.263</td>
<td>.063</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>-0.242</td>
<td>.081</td>
</tr>
<tr>
<td></td>
<td>LDL</td>
<td>-0.329</td>
<td>.027</td>
</tr>
<tr>
<td>BMI</td>
<td>Waist Circumference</td>
<td>0.805</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>Physical Fitness Self-Assessment</td>
<td>-0.554</td>
<td>.0003</td>
</tr>
<tr>
<td></td>
<td>Waist/Hip Ratio</td>
<td>0.561</td>
<td>.0002</td>
</tr>
<tr>
<td></td>
<td>Rockport Assessment</td>
<td>-0.669</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>Fasting Insulin</td>
<td>0.4977</td>
<td>.0012</td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td>0.28956</td>
<td>.045</td>
</tr>
<tr>
<td></td>
<td>ApoB</td>
<td>.255</td>
<td>.07</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>.370</td>
<td>.014</td>
</tr>
<tr>
<td></td>
<td>Diastolic BP</td>
<td>0.287</td>
<td>.047</td>
</tr>
<tr>
<td>Variable</td>
<td>Correlating Variable</td>
<td>Correlation Coeff</td>
<td>P Value</td>
</tr>
<tr>
<td>----------------------------------</td>
<td>----------------------</td>
<td>-------------------</td>
<td>---------</td>
</tr>
<tr>
<td>Waist Circumference</td>
<td>BMI</td>
<td>0.805</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>Waist/Hip Ratio</td>
<td>0.721</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>Physical Fitness Self-Assessment</td>
<td>-0.607</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>Rockport Assessment</td>
<td>-0.600</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>Fasting Insulin</td>
<td>0.571</td>
<td>.0002</td>
</tr>
<tr>
<td></td>
<td>HDL</td>
<td>-0.2373</td>
<td>.085</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>0.458</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>Diastolic BP</td>
<td>0.277</td>
<td>.05</td>
</tr>
<tr>
<td>Waist/Hip Ratio</td>
<td>Waist Circumference</td>
<td>0.721</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>BMI</td>
<td>0.561</td>
<td>.0002</td>
</tr>
<tr>
<td></td>
<td>Physical Fitness Self-Assessment</td>
<td>-0.4524</td>
<td>.0032</td>
</tr>
<tr>
<td></td>
<td>Rockport Assessment</td>
<td>-0.2422</td>
<td>.08</td>
</tr>
<tr>
<td></td>
<td>Fasting Insulin</td>
<td>0.479</td>
<td>.002</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>BMI</td>
<td>0.370</td>
<td>.014</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>-0.242</td>
<td>.081</td>
</tr>
<tr>
<td></td>
<td>Rockport Assessment</td>
<td>-0.219</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>Physical Fitness Self-Assessment</td>
<td>-0.234</td>
<td>.088</td>
</tr>
<tr>
<td></td>
<td>Fasting Insulin</td>
<td>0.367</td>
<td>.015</td>
</tr>
<tr>
<td></td>
<td>ApoB</td>
<td>0.357</td>
<td>.017</td>
</tr>
<tr>
<td></td>
<td>Waist Circumference</td>
<td>0.458</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>HDL</td>
<td>-0.335</td>
<td>.025</td>
</tr>
<tr>
<td></td>
<td>LDL</td>
<td>0.344</td>
<td>.021</td>
</tr>
<tr>
<td>Variable</td>
<td>Correlating Variable</td>
<td>Correlation Coeff</td>
<td>P Value</td>
</tr>
<tr>
<td>-------------------</td>
<td>----------------------</td>
<td>-------------------</td>
<td>---------</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic BP</td>
<td></td>
<td>0.507</td>
<td>.001</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>BMI</td>
<td>0.287</td>
<td>.05</td>
</tr>
<tr>
<td></td>
<td>Rockport Assessment</td>
<td>-0.348</td>
<td>.10</td>
</tr>
<tr>
<td></td>
<td>Adiponectin</td>
<td>-0.222</td>
<td>.10</td>
</tr>
<tr>
<td></td>
<td>Waist Circumference</td>
<td>0.277</td>
<td>.05</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>0.507</td>
<td>.001</td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>BMI</td>
<td>0.4977</td>
<td>.0012</td>
</tr>
<tr>
<td></td>
<td>Physical Fitness Self Assessment</td>
<td>-0.2390</td>
<td>.083</td>
</tr>
<tr>
<td></td>
<td>Rockport Assessment</td>
<td>-0.2934</td>
<td>.043</td>
</tr>
<tr>
<td></td>
<td>Waist Circumference</td>
<td>0.5714</td>
<td>.0001</td>
</tr>
<tr>
<td></td>
<td>Waist/Hip Ratio</td>
<td>0.479</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td>0.39416</td>
<td>.009</td>
</tr>
<tr>
<td></td>
<td>HDL</td>
<td>-0.34615</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>LDL</td>
<td>-0.235</td>
<td>.086</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>0.367</td>
<td>.015</td>
</tr>
<tr>
<td>Fasting Glucose</td>
<td>Adiponectin</td>
<td>0.335</td>
<td>.025</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>Rockport Assessment</td>
<td>0.2817</td>
<td>.05</td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td>-0.2715</td>
<td>.057</td>
</tr>
<tr>
<td></td>
<td>Fasting Glucose</td>
<td>0.335</td>
<td>.025</td>
</tr>
<tr>
<td></td>
<td>HDL</td>
<td>0.2401</td>
<td>.08</td>
</tr>
<tr>
<td></td>
<td>LDL</td>
<td>0.269</td>
<td>.056</td>
</tr>
<tr>
<td></td>
<td>Diastolic BP</td>
<td>-0.222</td>
<td>.10</td>
</tr>
<tr>
<td>Variable</td>
<td>Correlating Variable</td>
<td>Correlation Coeff</td>
<td>P Value</td>
</tr>
<tr>
<td>------------------------</td>
<td>----------------------</td>
<td>-------------------</td>
<td>---------</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td>Age</td>
<td>-0.2717</td>
<td>.057</td>
</tr>
<tr>
<td></td>
<td>ApoB</td>
<td>-0.8256</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>HDL</td>
<td>-0.3806</td>
<td>.012</td>
</tr>
<tr>
<td></td>
<td>LDL</td>
<td>0.8162</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>BMI</td>
<td>0.28956</td>
<td>.045</td>
</tr>
<tr>
<td></td>
<td>Fasting Insulin</td>
<td>0.39416</td>
<td>.009</td>
</tr>
<tr>
<td></td>
<td>A1C</td>
<td>.22581</td>
<td>.10</td>
</tr>
<tr>
<td></td>
<td>Adiponectin</td>
<td>-0.2715</td>
<td>.057</td>
</tr>
<tr>
<td></td>
<td>Rockport Assessment</td>
<td>-0.3081</td>
<td>.036</td>
</tr>
<tr>
<td></td>
<td>ApoB</td>
<td>0.216</td>
<td>.11</td>
</tr>
<tr>
<td></td>
<td>HDL</td>
<td>-0.4842</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>LDL</td>
<td>-0.426</td>
<td>.005</td>
</tr>
<tr>
<td>HDL</td>
<td>Rockport Assessment</td>
<td>0.4267</td>
<td>.005</td>
</tr>
<tr>
<td></td>
<td>Fasting Insulin</td>
<td>-0.34615</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>Adiponectin</td>
<td>0.2401</td>
<td>.08</td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td>-0.4842</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>Waist Circumference</td>
<td>-0.2373</td>
<td>.085</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>-0.335</td>
<td>.025</td>
</tr>
<tr>
<td></td>
<td>Total Cholesterol</td>
<td>-0.3806</td>
<td>.012</td>
</tr>
<tr>
<td>LDL</td>
<td>Age</td>
<td>-0.329</td>
<td>.027</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>0.344</td>
<td>.021</td>
</tr>
<tr>
<td></td>
<td>Fasting Insulin</td>
<td>-0.235</td>
<td>.086</td>
</tr>
<tr>
<td>Variable</td>
<td>Correlating Variable</td>
<td>Correlation Coeff</td>
<td>P Value</td>
</tr>
<tr>
<td>------------------------</td>
<td>----------------------</td>
<td>-------------------</td>
<td>---------</td>
</tr>
<tr>
<td>Adiponectin</td>
<td></td>
<td>0.269</td>
<td>.056</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td></td>
<td>0.8162</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Triglycerides</td>
<td></td>
<td>-0.426</td>
<td>.005</td>
</tr>
<tr>
<td>ApoB</td>
<td></td>
<td>0.684</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>ApoB</td>
<td>Systolic BP</td>
<td>0.357</td>
<td>.017</td>
</tr>
<tr>
<td>BMI</td>
<td></td>
<td>0.2548</td>
<td>.07</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td>-0.263</td>
<td>.06</td>
</tr>
<tr>
<td>Rockport Assessment</td>
<td></td>
<td>-0.242</td>
<td>.08</td>
</tr>
<tr>
<td>Triglycerides</td>
<td></td>
<td>-0.2715</td>
<td>.11</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td></td>
<td>-0.8256</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>LDL</td>
<td></td>
<td>0.684</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Physical Fitness Self-Assessment</td>
<td>BMI</td>
<td>-0.554</td>
<td>.0003</td>
</tr>
<tr>
<td></td>
<td>Waist/Height Ratio</td>
<td>-0.4524</td>
<td>.0032</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.239</td>
<td>.083</td>
</tr>
<tr>
<td></td>
<td>Fasting Insulin</td>
<td>-0.2390</td>
<td>.083</td>
</tr>
<tr>
<td></td>
<td>Waist Circumference</td>
<td>-0.607</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>-0.234</td>
<td>.088</td>
</tr>
<tr>
<td>Rockport Assessment</td>
<td>BMI</td>
<td>-0.669</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.263</td>
<td>.063</td>
</tr>
<tr>
<td></td>
<td>Adiponectin</td>
<td>0.2817</td>
<td>.05</td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td>-0.3081</td>
<td>.036</td>
</tr>
<tr>
<td></td>
<td>Fasting Insulin</td>
<td>-0.2934</td>
<td>.043</td>
</tr>
</tbody>
</table>
### Linear Regression Results

Linear regression analyses were run to determine the overall relationship between the Rockport Physical Fitness test results and fasting insulin, adiponectin, apoB, minutes of exercise per week, and job category. Analyses results are shown in Table 27. Note that “R^2” represents the proportion of the variation in the Rockport assessment that can be explained by the variation in the variable named in the first column of the table.

**Table 27: Linear Regression Results for Rockport Assessment vs Fasting Insulin, Adiponectin, ApoB, Minutes Exercised, and Job Category**

<table>
<thead>
<tr>
<th>Tested Variable</th>
<th>P value</th>
<th>R-squared</th>
<th>Relationship to Rockport Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting Insulin</td>
<td>.023</td>
<td>.115</td>
<td>Inverse</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>.065</td>
<td>.069</td>
<td>Positive</td>
</tr>
<tr>
<td>ApoB</td>
<td>.067</td>
<td>.066</td>
<td>Inverse</td>
</tr>
<tr>
<td>Minutes of Exercise/Week</td>
<td>.18</td>
<td>.024</td>
<td>Positive</td>
</tr>
<tr>
<td>Job Category</td>
<td>.35</td>
<td>.006</td>
<td>Positive</td>
</tr>
</tbody>
</table>
Of all the blood test results, triglycerides were most pervasive in its relationship with (and, perhaps, influence on) other test results. Therefore, linear regression analyses were performed to better understand these relationships. The following table summarizes the significant and notable results. Linear regression analyses were also performed to test the relationships of fasting glucose, total cholesterol, systolic blood pressure, waist circumference, and apoB with triglycerides, but these results were not significant.

### Table 28: Notable and Significant Linear Regression Results for Triglycerides

<table>
<thead>
<tr>
<th>Tested Variable</th>
<th>P value</th>
<th>R-squared</th>
<th>Relationship to Triglycerides</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rockport Physical Fitness</td>
<td>.0712</td>
<td>.064</td>
<td>Inverse</td>
</tr>
<tr>
<td>Fasting Insulin</td>
<td>.0042</td>
<td>.192</td>
<td>Positive</td>
</tr>
<tr>
<td>A1C</td>
<td>.049</td>
<td>.08</td>
<td>Positive</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>.089</td>
<td>.054</td>
<td>Inverse</td>
</tr>
<tr>
<td>LDL</td>
<td>.0082</td>
<td>.162</td>
<td>Inverse</td>
</tr>
<tr>
<td>HDL</td>
<td>.0045</td>
<td>.19</td>
<td>Inverse</td>
</tr>
</tbody>
</table>

### Multiple Regression Results

Sometimes more than one variable contributes significantly to a particular outcome. The outcomes of fasting insulin, adiponectin, apoB, triglycerides, and physical fitness (Rockport walking test) were tested using multiple regression analyses to determine which variables contributed most significantly to their variability. Aggregately, the variables that contribute to the multiple regression combination are called the "model." Each model has a P value, an $R^2$ value, and a regression equation. "$R^2$" represents the proportion of the variation in the outcome that can be explained by the variation in the other variables. The P value represents the significance of the model. The regression
equation is the numerical representation of the multiple regression line. If you fill in the values for a set of variables, the equation will yield a value along the regression line. The George and Rockport equations in Appendix A are examples of regression equations. Table 29 is a summary of the regression line formulas this study has provided.

**Table 29: Multiple Regression Models**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Contributing Variables/Regression Formula</th>
<th>P value</th>
<th>R-squared</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting Insulin</td>
<td>Waist/Hip Ratio, Systolic BP, Triglycerides, Family History of Diabetes(1 or 0)</td>
<td>&lt;.0001</td>
<td>0.5853</td>
</tr>
<tr>
<td>Regression Formula</td>
<td>Fasting Insulin=113.546(W/H Ratio)+0.1137(SBP)+0.0404(Triglycerides)+5.23 (FH_Diabetes)-107.79</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ApoB</td>
<td>Total Cholesterol, HDL</td>
<td>&lt;.0001</td>
<td>0.8153</td>
</tr>
<tr>
<td>Regression Formula</td>
<td>ApoB=13.603+0.59(Total Cholesterol)-0.705(HDL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adiponectin</td>
<td>Fasting Glucose, LDL, Family History of Diabetes(1 or 0)</td>
<td>0.027</td>
<td>0.2538</td>
</tr>
<tr>
<td>Regression Formula</td>
<td>Adiponectin=0.2299(Fasting Glucose)+0.0465(LDL)-2.387(FH_Diabetes)-17.652</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triglycerides</td>
<td>Total Cholesterol, LDL, HDL, Fasting Insulin</td>
<td>&lt;.0001</td>
<td>1.0000</td>
</tr>
<tr>
<td>Regression Formula</td>
<td>Triglycerides = 5.00 (Total Cholesterol) – 5.00 (LDL) -5.00 (HDL) + 6.59178 E-12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical Fitness</td>
<td>BMI, Age, HDL</td>
<td>&lt;.0001</td>
<td>0.6024</td>
</tr>
<tr>
<td>Regression Formula</td>
<td>Physical Fitness (Rockport walking test) = 77.98 – 1.614 (BMI) -0.308 (Age) +0.147 (HDL)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Family History Data**

Data on family history with regard to heart attacks and diabetes were collected during session 1. In general, the population was not prone to high blood pressure with the average systolic blood pressure coming in at 119 and the average diastolic blood pressure being 73. This blood pressure data correlates well with the family history, with only 2 out of the 43 participants on whom family history data were collected reporting a family history of heart attacks; while 20 of the 43 reported a family history of diabetes.
**SUMMARY**

The findings of this study were as follows:

- Statistics suggest that a meatpacker’s adiponectin level is significantly and directly related to physical fitness for those who report exercising no more than 80 minutes per week outside of work. As a meatpacker’s physical fitness increases, so does his adiponectin level increase. (See Tables 17-20.) When those who exercised more than 80 minutes per week were included in the analysis (see Tables 21-24), the relationship between adiponectin and physical fitness became more significant than for those who reported exercising no more than 80 minutes per week outside of work.

- Statistics also suggest that a meatpacker’s fasting insulin level is inversely related to his physical fitness. That is, as his physical fitness increases, his fasting insulin levels decrease. This relationship was true at the borderline (p = .088) significant level for those who reported exercising no more than 80 minutes per week outside of work. (See Tables 17-20.) When those who exercised more than 80 minutes per week were included in the analysis, the relationship between fasting insulin and physical fitness became more significant, with a P value of .033 (see Tables 21-24).

- For all meatpackers in the study, their apoB levels have no statistical relationship to their physical fitness levels. (See Tables 17-24.)

- There is no statistical relationship between physical fitness levels and A1C readings.
• The regression formulas for fasting insulin and adiponectin show a strong family history component, indicating that there are probable aspects of these variables that are hereditary. (See Table 29.)

• Linear regression analysis indicates that triglycerides have a strong positive relationship with fasting insulin and A1C levels, suggesting that these markers may have their etiology in triglyceride levels. (See Table 28.)

• Multiple regression analysis indicates that physical fitness, as measured by the Rockport walking test, is related to BMI, age, and HDL. Linear regression analysis indicates that there is no statistical relationship between a meatpacker’s physical and his job category and the minutes he exercises per week outside of work (See Tables 27 and 29).
Chapter 5: Discussion
STUDY DIFFICULTIES

During this study, both population and statistical difficulties were encountered.

Population Difficulties

Population difficulties occurred in the areas of recruitment, meatpacker classification, and meatpacker activity accuracy.

Recruitment: The most difficult aspect of this study occurred in the area of population recruitment. The easiest way to recruit meatpackers in Dodge City would have been to recruit the meatpackers within the meatpacking facilities themselves. With the cooperation of meatpacking management, a one-time event, similar to a health fair, could be staged to recruit interested individuals. Both the National and Cargill plant personnel departments and plant managers were approached with a list of positive outcomes for the meatpackers and for the meatpacking facility itself. Both plants declined participation. Cargill is usually more sympathetic than is National to similar community events, but even they claimed they needed the agreement of their corporate offices to undertake such an event. I sent a letter to their corporate offices via the plant manager and have yet to hear a decision.

Even at the cathedral, meatpacker recruitment was sparse most Sundays, amounting to three or four individuals. Despite announcements made at the mass and ads in the church newsletter and city newspaper, eight individuals was the highest number of meatpackers recruited any Sunday.
Naturally, these recruitment difficulties made the original estimate of 120 participants impossible to achieve within the 6 months allotted by the Graduate School for the study after the IRB approval. The final count for study participants was 43. Dedicated telephone calls to participants in session 1, kept participant losses between session 1 and session 2 to a minimum, and the count for meatpackers arriving for the physical fitness test and blood draws in session 2 was 35.

**Classification of Meatpackers:** The categories shown in Table 11 of this document were obtained in 2006 from the manager of health services at Cargill. Because of the current refusal of Cargill to cooperate with the study, these categories were not updated when the study began. Also, there are many jobs performed within the plant that are not mentioned in the chart. Where would we put plant supervisors, trainers, or shippers, as similar jobs were not mentioned? Of the 35 men who completed the study, only 30 were sedentary outside of work and exercised no more than 80 minutes per week outside of their normal work activities. Of these 30, only 24 had jobs that were ergonomically studied. The other 6 had jobs within the maintenance, shipping, quality control, supervisory, and training functions at the meatpacking plants. Typically, the workload of these 6 participants varied from day to day, from week to week, and from season to season. Therefore, they were excluded from the studies performed by job category.

**Activity Accuracy:** Before potential participants signed the consent form, they were told that they could not participate in the study unless they exercised no more than 30 or 40 minutes once or twice a week outside of work. However, some meatpackers agreed to this condition despite the fact that they exercised more than the stated ceiling. This discrepancy was identified by an additional question within the nurse’s questionnaire that
asked for number of minutes the individual exercised outside of work per week. Meatpackers who stated that they exercised more than 80 minutes a week were excluded from the group analyses by job category. There were two group analyses by physical fitness categories: one for those who reported that they exercised no more than 80 minutes per week; the second, for all study participants who completed session 2 of the study, including those who exercised more than 80 minutes per week. Also, all study participant data was represented in the descriptive statistics, correlation statistics, linear and multiple regression analysis, and the analysis of A1C data.

**Statistical Difficulties and Considerations**

Statistical difficulties occurred in the areas of the diet record and skinfold measurement accuracy. In addition, special consideration is given in this section to the operational definition of “sedentary,” validity of recalled exercise data, the normality of the data, and study population limitations.

**Diet Record:** The original intention of the diet record was not to count calories or nutrients, but to identify diet anomalies that might contribute to out-of-range blood results. Because of the education level or motivation of some of the meatpackers, even this goal could not be met because many times the diet record was completed in the most haphazard way. When triglycerides were very high, however, and the meatpacker came to session 3, he was asked whether manteca (i.e. pork lard) was used in the preparation of his food and how many tortillas he consumed each day. Most times, a likely cause of the high triglycerides was found to be the 12 to 15 tortillas (made with manteca) that the meatpacker ate on a daily basis.
**Skinfold Measurements:** Subscapular and tricep skinfold measurements were performed during session 2 of the study. However, a known inconsistency in the measurements occurs when more than one person performs the measurements. The first bilingual nurse quit the study when she was scheduled for a back operation and our substitute nurse was absent one time when the skinfold measurements were taken. Therefore, because the results were so inconsistent, they could not be used in the study.

**Operational Definition of Sedentary:** Operational definitions of “sedentary” vary across studies. In a paper presented by Hong et al., at the 2004 American Association of Public Health, they indicated that in 46 randomized control trials of exercise intervention for “sedentary” seniors, “sedentary” was defined five ways: 1) no definition given; 2) no regular exercise participation; 3) no prior exercise within a specified period of time; 4) duration or frequency of prior exercise specified; and 5) failure to meet the ACSM guideline. In studies where duration/frequency of exercise or regularity of exercise were used to define sedentary, the completion of the exercise regimen by participants in the randomized trial was significantly higher.

For this study, the operational definition of “sedentary outside of work” was “exercising no more than 30 or 40 minutes once or twice a week outside of work.” This definition meets the duration/frequency criteria for a good operational definition of “sedentary” as shown, above, and is very close one provided by ACSM as the threshold for a sedentary lifestyle (ACSM, 2009). According to the ACSM someone is sedentary when they do not participate in at least 30 minutes of moderate intensity physical activity on at least 3 days of the week for at least 3 months.
Validity of Recalled Exercise Data: There have been major studies (Mayer-Davis et al., 1998; Regensteiner et al., 1991) performed that relate diabetes health markers to physical exercise levels using self-reported physical activity as the exercise measurement.

The Regensteiner et al. study was part of the San Luis Valley Diabetes study. During this study, subjects were asked to recall their activities over the past 7-days and to place them in categories of active, very active, or extremely active. In addition, subjects were asked “to recall work activities and then nonwork activities….Activities not considered to be specifically work related were classified as nonwork by default.” (Regensteiner et al., 1991:1067) Based upon their answers, subjects were put into one of three terciles of exercise activity and diabetes markers were analyzed for each tercile.

For the Mayer-Davis et al. study two approaches were used to access physical activity. One approach asked subjects to indicate the number of times per week they engaged in vigorous physical activity. Choices available were “rarely to never,” “5 or more times per week,” etc. The second approach was through a 1-year recall of physical activity.

Asking the meatpackers in this study to recall their recent minutes of physical activity outside of work would tend to be just as accurate as the yearlong exercise recall required of participants in the Mayer-Davis study, or the week-long detailed recall required of the Regensteiner study. In addition, in this study, diabetes markers were not analyzed with regard to minutes of exercise, but with regard to physical fitness, which was measured in two ways: once with the self-assessment of the George test; and again, with the Rockport walking test. Although the meatpackers seemed to overestimate their
physical fitness with the George self-assessments, the results of both the George and Rockport tests placed the average reading below the 25th percentile for the average age of the participants, suggesting that the majority of the participants were sedentary.

Statistical analyses sought the relationship of diabetes markers only with the results of the Rockwell walking test since it was presumed to be more accurate than self-assessments. Note that physical fitness is stronger than self-reported physical activity as a predictor of many health outcomes (Blair and Church, 2004).

**Normality of the Data:** Both ANOVA and t-test analyses require that the data be normally distributed for results to be valid. When data are not normally distributed, the usual remedy is to substitute the e-log for the original reading. This process is known as the “log-transformation of the data.” The logs are usually normally distributed, but not always. In the case of the apoB and adiponectin readings, which were needed to test the study hypotheses, the data were not normally distributed, but neither were the logs. Therefore, nonparametric tests were used instead of the ANOVA and t-tests that were planned. The Kruskal-Wallis test was substituted for the ANOVA; the Wilcoxon Exact test was substituted for the t-test.

**Study Population Limitations:** For testing the diabetes health-markers of fasting insulin, adiponectin, and apoB, the study recruited thirty-five meatpackers. To overcome the limitations presented by this small population, non-parametric testing was used. In addition, for the analyses of job and physical fitness categories, categories 2 and 3 were combined so that the testing more closely met the sample size requirements for 80% likelihood of finding at least a 0.05 P value through t-tests that compare two groups. The sample size requirement was 33 individuals.
STUDY STRENGTHS

Despite the fact that many difficulties were encountered in the process of data collection, the study yielded many notable and significant results. These positive results occurred not only with regard to the statistical analyses, but in relationship to potential health outcomes among the Hispanic male meatpacking community in Dodge City, Kansas.

Besides the above attempts to ensure the integrity of the data, many attempts were made to ensure each meatpacker's understanding of his blood test results and his need to visit a physician. Understanding was assured through translations by native speakers, a feedback session where results were explained, and checks with the church-going meatpackers to assess whether they were still on target.

Translations: All participant documents were translated by native speakers. These documents included the consent forms, parts of the interview forms, a Power Point presentation that was used in the feedback sessions, and the letter that conveyed the blood test results. Also, Spanish speakers explained the consent forms, did the interviews, and provided feedback.

Feedback Sessions: During the feedback session (session 3), each meatpacker received his results in a sealed envelope, along with a letter explaining what each result meant and a direction for them to visit their physician should they have more questions. A bilingual nurse was available during the session to answer any questions and each meatpacker heard and received a copy of a Spanish language Power Point presentation that explained diabetes, its stages, and what could be done to prevent its development.
The presentation was based on information provided by the “Step by Step” program of the National Diabetes Educational Program.

Informal Checks: Frequently, those who administered the study would see study participants at church. Participants would provide information on their changed diets or exercise programs, their visits with their doctors, or their intentions to visit their doctors soon. Some said they were taking new prescriptions.

HYPOTHESES TESTING RESULTS

Initially, the study hypotheses were tested using the job categories listed in Table 11. To state the hypotheses again, they were:

- Insulin resistance is inversely related to the physical fitness resulting from job related activities
- Adiponectin level is positively related to the physical fitness resulting from job related activities.
- ApoB level is inversely related to the physical fitness resulting from job related activities.

Because the fitness level for meatpackers performing the demanding heavy and moderate categories was lower than for meatpackers performing light tasks (see Table 14), the use of the meatpacking industry’s classifications became questionable for ascertaining whether fasting insulin, adiponectin, and apoB varied by fitness level.

Although the form that helped the meatpacking industry classify jobs isolated the strength and flexibility needed to perform the job, it made no mention of any capabilities consistent with cardiovascular physical fitness (see Figure 6). Therefore, there were individuals hired for more difficult jobs that were stronger or more flexible than those who
were hired for jobs classified as light. However, strength and flexibility is no guarantee of cardiovascular fitness.

To determine whether fasting insulin, adiponectin, and apoB varied by fitness level, therefore, instead of job categories, fitness levels were used directly and drawn from the meatpackers’ Rockport Walking test results and ACSM percentile values for maximal oxygen uptake in men (see Table 16). The categories used were as follows:

- **VO_{2max} category 1** = 0 – 26.799 ml/kg.min (percentiles 1-10 for ages 60 and above)
- **VO_{2max} category 2** = 26.8 – 37.899 ml/kg.min (percentiles 10-20 for ages 20 to 29)
- **VO_{2max} category 3** = 37.9 – 50 ml/kg.min (percentiles >20 for ages 20 to 29)

The percentile value for age 60 and above was used for the lowest physical fitness level because it allowed reasonable graduations above it. The values for categories 2 and 3 were taken from the 20-30 age group because this age group showed the widest difference in values. Percentiles were not used because categories for physical fitness values that did not consider age were sought. These categories for physical fitness yielded the statistical results shown in Tables 17-24. Although the population fell into the three fitness categories shown above, both three category tests (Kruskal-Wallis—Tables 17 and 21) and two category tests (Wilcoxon Exact – Tables 18,19, 22, and 23) were performed because two category tests have more statistical power given small sample sizes (see Study Population Limitations, above).

Note that a count was done to determine how job categories related to VO_{2max} (fitness) categories. The results were as follows:

- Of the 6 individuals in the light exertion job category, 1 was classed as VO_{2max} category 1 and the other 5 were in VO_{2max} category 2.
### POST OFFER PREPLACEMENT QUESTIONNAIRE

**PART A - TO BE COMPLETED BY HUMAN RESOURCES (Please Print)**

<table>
<thead>
<tr>
<th>Name (Last, First, Middle)</th>
<th>Social Security No.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Sex:  

<table>
<thead>
<tr>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Birth Date / /  

<table>
<thead>
<tr>
<th>Job Offer Date</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

**POSITION APPLIED FOR OR NOW HOLDING**

<table>
<thead>
<tr>
<th>Title</th>
<th>Department:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Human Resources Contact**

<table>
<thead>
<tr>
<th>HR Phone No. (Area Code) Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>(620) 338-4425</td>
</tr>
</tbody>
</table>

**Division**

<table>
<thead>
<tr>
<th>Facility</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dodge City, KS</td>
</tr>
</tbody>
</table>

**Human Resources Signature**

---

**PART B - TO BE COMPLETED BY HUMAN RESOURCES BEFORE EXAMINATION**

<table>
<thead>
<tr>
<th>Exam Type:</th>
<th>Job Transfer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Reason for Request:**

<table>
<thead>
<tr>
<th>New Hire</th>
<th>Job Transfer</th>
<th>Medical Qualification</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Examination Appointment:**

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

**PART C - ESSENTIAL JOB FUNCTION REQUIREMENTS**

(See the appropriate box for each requirement essential to the duties of this position. List any additional essential factors in the blank spaces.)

**Torso**

- Heavy lifting/carrying greater than 60 pounds
- Moderate lifting/carrying equal to 40-60 pounds
- Moderate lifting/carrying 20-40 pounds
- Light lifting/carrying 0-20 pounds
- Frequent carrying of lifting greater than 40 times per hour
- Overhead or above shoulder reaching required
- Bending below the waist required
- Bending at waist, 180° per hour
- Push/pull maximum weight required, pounds unwheeled
- Push/pull maximum weight wheeled
- Twisting torso at waist

*This job is classified as producing light/moderate/heavy (circle one) stresses to the spine.*

**Upper Extremity Requirements**

(See appropriate boxes and circle appropriate italicized items.)

- Use of fingers required
- Both hands required
- Acceptable prosthesis allowed
- Ability to grasp light/medium/heavy with right/left hands
- Ability to flex/extend right/left hands
- Ability to do repetitive work with hands: low repetition (>30 sec/cycle) / moderate (30 sec/cycle) / high (15 sec/cycle)
- Ability to exert light/medium/heavy arm/hand force with right/left hands
- Ability to wear gloves, right/left hands
- Ability to use tools, right/left hands
- Ability to use vibrating tools, right/left hands

*This job is classified as producing light/moderate/heavy stresses to the hands and upper extremity.*

**Environmental**

- Ability required to work at the following temperatures:
  - <32°F
  - 32-65°F
  - 66-77°F
  - >77°F
- High humidity
- Ability to work outside/inside
- Ability to work on slippery, uneven or unstable surfaces
- Ability to work with solvents or chemicals specified:

- Ability to work with hands in water
- Ability to work around moving machinery
- Ability to work at unprotected heights
- Ability to operate crane, truck, forklift, or other moving vehicles
- Ability to work with dust and fumes
- Ability to wear a respirator

---

**Cognitive Problems**

- Vision:
  - Vision correctable to 20/20 in one eye and 20/40 in the other eye
  - Vision correctable to 20/40 in one eye and 20/100 in the other eye
  - Specific eye requirements:

- Both eyes required

---

*Figure 6: Meatpacking Job Classification Form*
• Of the 10 individuals in the moderate exertion job category, 6 were in VO$_{2\text{max}}$ category 1, 2 were in VO$_{2\text{max}}$ category 2, and 2 were in VO$_{2\text{max}}$ category 3.

• Of the 8 individuals in the heavy exertion job category, 2 were in VO$_{2\text{max}}$ category 1, 5 were in VO$_{2\text{max}}$ category 2, and 1 was in VO$_{2\text{max}}$ category 3.

That is, meatpackers in the light exertion job category were more likely to be in VO$_{2\text{max}}$ category 2 (5 out of 6) than were meatpackers performing jobs that required heavy exertion (5 out of 8). Only one individual from the heavy exertion category was in VO$_{2\text{max}}$ category 3; none of the individuals in the light exertion category exceeded VO$_{2\text{max}}$ category 2.

This lack of fit between job categories and fitness categories could have been caused by the following:

• inaccuracy of the job categories,

• inaccuracy of the bilingual nurse’s interpretation of the job category given by the meatpacker, or

• the fact that meatpacking jobs are not progressively related to aerobic physical fitness, with an increase in categories of exertion reflected with an accompanying increase in aerobic physical fitness.

The job categories were obtained from Cargill about 5 years ago. They could have changed in the interim. However, the mismatch reflected in the statistical analysis by job category and the results of the count, above, reflects a major change, not a minor one. The meatpackers we interviewed did not identify such a change. Both of the nurses who interviewed the meatpackers were fluent Spanish speakers. There is a possibility that
when they listed the jobs for the meatpackers, the meatpackers did not understand the categories well enough to be able to respond accurately. If personnel records could have been accessed, any interview inaccuracies could have been overcome, but this was not allowed. The last possibility is that the job categories are not a good proxy for and an accurate and progressive scale of aerobic fitness. This lack of fit is supported by the results of the multiple and linear regression analyses of physical fitness (see Tables 27 and 29 and the discussion of physical fitness later in this Discussion section). The regression analyses tell us that job categories do not significantly statistically contribute to and are not significantly statistically related to physical fitness. Moreover, the averages and standard deviations for the analyses by job category (see Tables 13-16) indicate that the jobs within each category appear along a continuum rather than in distinct categories. That means that, in many cases, the averages for the categories “bled” into another category and the standard deviation of one category severely encroaches upon the standard deviation of the adjacent category. The job categories may not have reflected the fact that a job in the moderate exertion category could have been as strenuous, aerobically, as a job in the heavy exertion category. Another reason for the discrepancy between job category and physical fitness might be that meatpackers in jobs that require heavy exertion may use power tools, like chain saws, that hang from the ceiling and bear a large part of the workload; while a job in a category of lower exertion might depend solely on the exertion of an unaided meatpacker. Without a study of meatpacker jobs that considers aerobic physical fitness, and not just strength and flexibility, the reasons for the mismatch between aerobic physical fitness and meatpacking jobs are unlikely to be uncovered. Given the results of the multiple regression analysis for physical fitness, an
individual’s BMI, age, and HDL level contribute most to his physical fitness level. His job category was not significantly related to physical fitness.

Note that the following convention was used when compiling the results of statistical tests on study data. When there was a choice of one-sided or two-side test results, one-sided results are provided because the results were related to physical fitness in the direction (positive or inverse) anticipated in the stated hypotheses. The results of this hypothesis testing are discussed below.

Adiponectin

Adiponectin is an adipose tissue derived hormone that decreases with increased body adiposity. The thinner the individual is, the higher his adiponectin level is.

In this study, adiponectin was shown to be statistically directly related to physical fitness for those individuals who reported that they exercised no more than 80 minutes per week outside of work. That is, as physical fitness increased for these individuals, so did adiponectin increase. For this group, the probabilities for this conclusion were shown to be 4.6% for the Kruskal-Wallis test (see Table 17) and 2.5% for the Wilcoxon Exact test (see Table 19). For the entire population of meatpackers who completed session 2, including those who exercised more than 80 minutes per week, the probabilities for this conclusion were shown to be 1.9% for the Kruskal-Wallis test (see Table 21) and 1.7% for the Wilcoxon Exact test (see Table 23). The positive direction of the relationship between adiponectin and physical fitness was indicated by a positive correlation coefficient (+.255 [Table 20] and +.282 [Table 24]) for the overall population and a positive direction in the relationship between the Rockport assessment and adiponectin in the linear regression analysis for the whole study (see Table 27). These study results are consistent with
those of another study that indicates that increases in physical activity (and perhaps, physical fitness) induce increases in adiponectin levels (Ring-Dimitriou, 2006; Kriketos et al., 2004).

Adiponectin is thought to increase glucose uptake and lipid oxidation in muscle, leading to improved insulin sensitivity and less insulin-resistance (Yamauchi et al., 2001; Richardson et al., 2006). Theoretically, then, a negative correlation between adiponectin levels and blood glucose levels should exist. This meatpacker study shows a surprising positive correlation (+.335, P value 2.5%) of adiponectin with fasting glucose. The reason for this positive relationship could be the lack of data attributable to the small sample size.

There are two forms of adiponectin receptors in skeletal muscles (AdipoR1 and AdipoR2). The expression of both forms of the receptors is thought to be lower in Mexican-Americans with a family history of diabetes and is thought to be positively correlated with the insulin-resistance of this population (Civitarese et al., 2004). Although adiponectin originates in the fat cells, its expression is also regulated in the muscles and liver by the AdipoR1/2 receptors there. In this meatpacker study, a linear regression analysis was run to determine the relationship of a family history of diabetes and adiponectin. No significance was found. However, family history did figure prominently in the multiple regression analysis for adiponectin (see Table 29), indicating that there are probable aspects of this variable that are hereditary. No tests were performed to determine the number of AdipoR receptors in skeletal muscles, nor how family history affects this number. Civitarese et al. (2004) speculate that the lower expression of AdipoR1/R2 receptors in individuals with a family history of diabetes may be part of reason they are predisposed to developing diabetes.
It should be noted that adiponectin expression is positively correlated with glucose disposal. Much of glucose disposal depends on the number of GLUT4 receptors in muscle. We know that the number of GLUT4s increases with exercise (Dela, 1994; Houmard, 1991); perhaps exercise increases the number of AdipoR receptors as well. We do know that as exercise increases in obese and/or diabetic individuals, AdipoR expression also increases in skeletal muscle (Vu et al., 2007).

Adiponectin also functions as an insulin sensitizer by decreasing hepatic glucose output (Trujillo & Scherer, 2005; Richardson et al., 2006) during the fed state. Because much of the adiponectin activity occurs in the liver, Trujillo and Scherer (2005) have speculated that the decreased production of adiponectin in visceral adipose tissue depots in obese individuals may explain the inverse association between visceral adiposity and adiponectin levels. In this study, no significant or notable relationship was found between waist circumference and adiponectin levels. Again, this result may be due to the lack of data, which is attributable to the small sample size.

Four men in this study exhibited very low adiponectin levels despite the fact that they were physically fit and were not insulin resistant. Perhaps, these men should be followed after this study since even in the absence of any other indicators of insulin resistance, low adiponectin levels predict the development of type 2 diabetes (Lindsay et al., 2002; Spranger et al., 2003).

ApoB

Most people are familiar with the categorization of HDL as the “good” lipoprotein, and LDL as the “bad” lipoprotein. But few know that LDL is actually a calculated amount that is derived from the blood levels of total cholesterol, HDL, and triglycerides (LDL = Total Cholesterol – (HDL + Triglycerides/5)). The apoB measurement, however,
is an assay is a count that includes the actual number of low density lipoproteins (i.e., LDLs) in the blood.

In this study, for those individuals who reported exercising no more than 80 minutes per week outside of work and for the total population under study, there was shown to be no significant relationship with physical fitness and an individual’s apoB level. In fact, the apoB levels for this population were shown to vary only within the normal range, with very few approaching the level for risk of insulin resistance (i.e., 1.2 g/l). This finding was strengthened by the findings for both systolic and diastolic blood pressure for this population, with the mean systolic BP reading set at 119 and the diastolic BP set at 72.5. In fact, there were only two individuals in the study for whom we recorded a family history of heart attacks. It should be noted that the Mexican American population in general has been shown to have a decreased prevalence of hypertension (Haffner et al., 1990; CDC, 2010), coronary heart disease (Rewers et al., 1993; CDC, 2010), and ischemic heart disease mortality (Becker et al., 1988; CDC, 2010) compared to the non-Hispanic white population.

The immigrant status of this particular population could have contributed to their favorable apoB results. According to a study that compared the mortality rates of non-Hispanic whites, US-born Mexican Americans, and foreign-born Mexican Americans, foreign-born Mexican Americans experienced only half the mortality rates of the other two groups (Wei et al., 1996). Part of this is due to a “healthy migrant effect,” that indicates that migration itself filters out unhealthy individuals, who must stay home because of their debilitated condition. Another advantage of this population may be diet.
Because of their lower socioeconomic and economic level, this group may not be able to afford a diet richer in fatty meats.

In the San Antonio Family Heart Study, lipoprotein variance was three times more likely to result from genes than from environmental factors (Mitchell et al., 1996). Mexican Americans seem to be favored with genes that do not predispose them to heart disease. However, once individuals become diabetic, heart disease becomes a factor in their mortality. In the Hispanic population in general, the most frequently cited underlying causes of diabetes-related deaths were heart disease (31%), cancer (8%), and stroke (6%) (Smith and Barnett, 2005).

Note that despite the fact that physical fitness did not affect apoB levels according to the analyses of physical fitness groups (see Tables 17-24), according to a linear regression analysis (see Table 27), there was a borderline significant inverse relationship between the apoB measurement and the Rockport assessment for the whole population. This may be due to the fact that the relationship between physical fitness and apoB levels is more linear than the physical fitness categories reflected in the Wilcoxon tests. The multiple regression analysis done on the whole population yielded the following regression formula:

\[ \text{ApoB} = 13.603 + 0.59(\text{Total Cholesterol}) - 0.705(\text{HDL}) \]

The analysis indicated that total cholesterol and HDL accounted for 81% of the variation in the apoB readings.

**Fasting Insulin**

Type 2 diabetes develops in three stages: insulin resistance; prediabetes; and frank diabetes. The hallmark of insulin resistance is an elevated fasting insulin level;
while the markers for prediabetes and frank diabetes are elevated fasting glucose levels. As the muscles and liver become more and more insulin-resistant because of elevated free fatty acids (Boden, 1997), more and more insulin is required to activate glucose uptake into the muscles and to shut off glucose production in the liver during the fed state. Therefore, although blood glucose may not be high (because elevated insulin levels have done their job), insulin levels are higher than normal (De Meyts, 1993). This constant need for relatively high insulin levels puts a strain on the pancreas' beta-cells, where the insulin is produced, and leads to their demise and to prediabetes, and, eventually, frank diabetes (Kahn et al., 2006).

For meatpackers who exercised no more than 80 minutes per week outside of work, fasting insulin levels were shown to have a borderline significant relationship (P value= .088) with physical fitness (see results of the Wilcoxon test, Table 19). For the entire population of meatpackers who completed session 2, including those who exercised more than 80 minutes per week, fasting insulin levels showed a significant relationship (P value = .033) with physical fitness (see Table 23). Also, a significant inverse relationship (P value=.023) was found between the Rockport assessment and fasting insulin when a linear regression analysis was run for the whole population (see Table 27). The difference between the borderline significant result for those who exercised no more than 80 minutes/week (30 individuals) and the results for the entire population who finished session 2 (35 individuals) could have been due to the larger sample size that was represented in latter analyses. Alternatively, the improvement in significance could have been due to the fact that the effect of exercise on fasting insulin levels becomes increasingly apparent as the individual exercises more outside of work.
Blood hormones, blood lipoproteins, and anthropometrics accounted for only 58% of the variability in the fasting insulin measurements according to the following multiple regression formula (see Table 29):

\[
\text{Fasting Insulin} = 113.546(W/H \text{ Ratio}) + 0.1137(\text{SBP}) + 0.0404(\text{Triglycerides}) + 5.23 (\text{FH_Diabetes}) - 107.7
\]

The formula indicates that body fat (waist to hip ratio) figures prominently in fasting insulin level, while an individual’s systolic blood pressure, family history of diabetes, and blood triglycerides contribute to a lesser extent. The family history component in the regression formula may indicate that there are aspects of this variable that are hereditary.

The results of the Spearmen correlations for fasting insulin, shown in Table 26, strengthen the conclusion that body (BMI, Waist Circumference) and dietary (some portion of the triglycerides) fat affect an individual’s level of free fatty acids (FFAs), and, therefore, fasting insulin.

Absent from the formula, is the contribution from dietary fats, which may account for much of the remaining variability (42%). The San Luis Valley Diabetes Study analyses indicated that dietary fats – particularly saturated and trans-fat – significantly predict the risk of type 2 diabetes and that dietary fiber and starch were inversely associated with fasting insulin levels (Marshall et al., 1994 and 1997). Hu et al. (2001) remind us that consuming more saturated and trans fats could adversely affect glucose metabolism and insulin resistance. They recommend a higher amount of fiber and minimally processed whole grain products to lower the risk of diabetes. In this study, based on interviews with participant meatpackers at feedback sessions, high daily intake of tortillas made with manteca may account for some of the dietary problem. For this population, whole grain tortillas made with a minimal amount of fat would be a good substitute.
OTHER FINDINGS

This study yielded interesting and significant findings that were outside its initial charter to investigate its stated hypotheses. These additional findings were in the areas of physical fitness, A1C, measurements of central adiposity, and triglycerides.

Physical Fitness

Both multiple and linear regression tests were applied to determine which variables contributed most to an individual’s physical fitness as measured by the Rockport walking test. Multiple regression results indicated that BMI, age, and HDL contributed 60% of the variability of physical fitness levels. This was at a <.0001 significance level. Both BMI and age were inversely related to physical fitness; while HDL was positively related. That is, as an individual’s BMI or age increased, his physical fitness decreased. However, if his HDL increased, so did his physical fitness. An individual’s job category and minutes he exercised per week outside of work were tested using linear regression to determine their relationship with physical fitness. No significant relationship was found.

A1C

In general, the Mexican American population has a higher average A1C percentage than does the non-Hispanic white population (see Figure 8, below).
In this study, the average participant age was 41 and the average A1C reading was 5.5. This was significantly higher for this age group than the 5.2 Figure 8 shows for the Mexican American population in general. In fact, it was equivalent to average A1C readings for Mexican American individuals aged 65, possibly rendering the Dodge City population susceptible to diabetes at an earlier age. This disparity could be due to the particular dietary habits of the Dodge City population.

We administered A1C tests to 43 men. Of those, 19 had 5 times the normal chance of developing diabetes in the future; and 4 had 16 times the normal chance of developing the disease. In fact, 14 of the men showed A1C readings between 5.7 and 6.4, which put them in the prediabetes category (Choi et al., 2011). More exercise and fewer saturated fats in their diets might help this population subset avoid diabetes in the future.

**Measurements of Central Adiposity**

In this study (see Table 26), waist circumference (-0.600 correlation coefficient, <.0001 P value) was found to have a higher correlation with the Rockport physical fitness
result than did the waist/hip ratio (-0.2422 correlation coefficient, .08 P value). In addition, waist circumference (0.5714 correlation coefficient, .0001 P value) correlated better with fasting insulin (and, therefore, insulin resistance) than did waist/hip ratio (0.479 correlation coefficient, P value .002). This is consistent with the findings of Wang et al. (2005), which state the waist circumference is a better predictor of risk for type 2 diabetes than is the waist/hip ratio. Both this study and the Wang study were of all men. Perhaps, the waist/hip ratio has a higher correlation with the physical fitness, fasting insulin levels, and insulin resistance of women.

Note that it is not only central adiposity, but adiposity in general, that contributes to high triglyceride levels. Eric Swanson, MD, (2011) reported that after liposuction there was a 43% reduction in triglyceride levels for those whose levels exceeded 150 mg/dL before the procedure. Adiposity continues to be a significant predictor of insulin sensitivity, with central adiposity being the most important single factor (Racette et al., 2006).

**Triglycerides**

Linear regression analyses done on the whole study population showed that triglycerides (and, therefore, their derivative free fatty acids) had an inverse relationship with an individual’s physical fitness, and adiponectin, LDL, and HDL levels. That is, the higher an individual’s triglycerides were, the lower was their physical fitness level. Their adiponectin, LDL, and HDL levels were similarly related, with these levels decreasing with a triglyceride rise. The influence of triglycerides on adiponectin makes sense as we know that adiponectin decreases as body fats rise. However, the inverse influence of
triglycerides on LDL and HDL is more obscure. The relationship with LDL is more evident if we examine the following formula:

\[ \text{LDL} = \text{Total Cholesterol} - (\text{HDL} + \text{Triglycerides}/5) \]

Given the formula, the higher the triglycerides are, the lower is the calculated LDL reading. If you solve the same formula for HDL, you will see that higher triglycerides also cause lower HDL readings.

Linear regression analysis also showed that there was a positive, or direct, relationship between triglycerides and individual’s fasting insulin level and A1C reading. That is, the higher the triglyceride level was, the higher the fasting insulin and A1C readings were. High triglycerides mean that there are high free fatty acids (Boden, 2003). High free fatty acids cause insulin resistance, and, hence, high fasting insulin. Given the insulin-resistant state of individuals with high triglycerides and high fasting insulin, there many have been times in the two or three months before an A1C reading, when blood glucose was high because insulin production had not caught up with the need for glucose clearance after a meal. At these times, hemoglobin glycolation could have occurred. Hence, higher A1C readings may have resulted.
RELEVANT OBSERVATIONS

One observation that is not a result of this study, but may underlie the insulin-resistant state of the Mexican American population in general, is the persistence of the use of manteca (pork lard used for cooking) in the Mexican American diet today. Recent investigation into the sale of manteca in grocery stores in Baja California, Mexico, indicates that vegetable oil has long ago substituted for manteca on the shelves. This has been because of the high current cost of manteca and the purposeful substitution of a healthier product. In Mexican American grocery stores in Denver and Dodge City, however, manteca still holds a prominent place. In a large store in Denver, in fact, manteca takes up a whole aisle. This could be a cultural “founder’s effect.” Just as an older variation the French language persists in Quebec City and Montreal, the remnants of a culture that long ago settled eastern Canada, manteca persists in the Southwestern United States, a remnant of the culture of the Mexicans who originally settled the area. When new Mexican immigrants arrive, they settle into the extant culture and enjoy the cheap manteca and delicious tortillas. We know that diet contributes to identity and feelings of belonging. Therefore, even when immigrants have manteca very seldom in their native Mexico, they may switch to it upon their arrival in an attempt to belong to their new cultural group. This disparity between the eating habits of native Mexicans and Mexican Americans may explain why the prevalence of diabetes in Mexico is about 10% (IDF, e-atlas), while it is 12% among Mexican Americans.
Dietary acculturation is “the process by which immigrants adopt the dietary practices of the host country” (Satia-About et al., 2002: 1105). In this case, the Mexicans who immigrate from Mexico are acculturated into the Mexican American culture. During this acculturation, the taste preferences of the new arrivals change. Even though there were earlier socioenvironmental influences that established them among their family and peers in Mexico, once they have immigrated, the immediate environment is most influential (de Castro, 2002). This is especially true if the immigrants reside in a Mexican American barrio (Keefe, 1992).

On the whole, women and men born in Mexico tend to be healthier than Mexican Americans born here. Their diets meet heart-healthy dietary guidelines and recommended nutrient intakes (Dixon et al., 1999). However, as they are acculturated into the Mexican American communities, their diets tend to change. For newcomers, the consumption of traditional foods decreases, and the adoption of new food practices increases (Romero-Gwynn and Gwynn, 1997). Not all the changes are good ones. For example, the consumption of plain boiled rice increased from 9% of the population in Mexico to 32% of the population after immigration to the United States. The consumption of plain white bread was 15% in Mexico, but 66% after immigration. While 25% consumed cookies in Mexico, 61% of the population consumes cookies after immigration. Only 14% of the Mexican population consumes flour tortillas, but 38% of the Mexican immigrants consume them in their core diet (Grivetti et al., 1996) once they settle in America.
Although many Mexican immigrants claim that they do not use manteca in their cooking, they neglect to the count the flour tortillas that they consume. A visit to “The Pioneer Woman” cooking website reveals the following recipe for flour tortillas:

(http://thepioneerwoman.com/cooking/2010/05/homemade-flour-tortillas)

- 2 ½ cups all-purpose flour
- 2 ½ teaspoons baking powder
- 1 teaspoon kosher salt
- ½ cup lard (manteca)
- 2 tablespoons additional lard
- 1 cup hot water.

When you visit the site, you will see a large blue and green tub of Morrell pork lard pictured. This is the commodity that lines the shelves of Mexican grocery stores and is consumed by Mexican Americans.

Habits differ within ethnic groups. Variables that might influence the food habits of immigrant Mexicans are educational status, length of residency, place of birth, and geographical location (Algert et al., 1998; Jerome, 1980). Romero-Gwynn and Gwynn (1997) report that the use of manteca decreased from 67% in Mexico to 28% after immigration. These statistics do not explain the fact that no manteca was found on the grocery shelves in Baja California Norte, while there is a whole aisle dedicated to it in Denver Mexican American grocery stores. Because Mexico is a big country, with many states and ecological niches, each area may vary in its cooking fat preferences. More investigation into the prevalence of manteca on a area-by-area basis (both in Mexico...
and in the United States) is needed to determine how diets change in this regard once Mexican immigrants are integrated into Mexican American barrios in the United States.

With regard to the application of this information to the meatpackers in this study, when they were interviewed, few meatpackers knew that tortillas were made with manteca. Many thought they were eating a healthy diet by avoiding cooking with manteca, but they neglected to eliminate tortillas made with manteca from their diets. Although we educated the men in the study about the dangers of manteca, education does not guarantee a change in behavior, especially when food imparts much of their group identity.
Chapter 6: Conclusion
CURRENT STUDY

For all meatpackers in the study (both for those who reported exercising no more than 80 minutes per week outside of work and for those who exercised more than this threshold), their adiponectin levels were directly related to their levels of physical fitness. Moreover, for those who reported exercising no more than 80 minutes per week outside of work, the study has suggested, at the borderline significant level, that fasting insulin tends to be inversely related to the level of physical fitness. When all meatpackers who completed session 2 were represented in the analysis, including those who exercised more than 80 minutes per week, fasting insulin was shown to be significantly inversely related to their physical fitness levels. The more physically fit a meatpacker is, the lower his fasting insulin levels tend to be and the less insulin-resistant he is.

For the tested population of male Hispanic meatpackers in Dodge City, Kansas, there seems to be no relationship between an individual’s physical fitness or job category and his apoB level. Within the population under study, apoB varied only within normal ranges, and, thus, was not as subject to the effects of either job stresses or physical fitness as were the other health markers (adiponectin and fasting insulin). When a linear regression analysis was performed on the whole population, linear regression analysis showed borderline significant inverse relationship between a meatpacker’s apoB level and his level of physical fitness.

In general, we can conclude that physical fitness does matter in an individual’s diabetes-related health markers.
The study did not establish a link between physical fitness and on-the-job activity. Physical fitness itself was shown to be more related to the meatpacker’s BMI, age, and HDL than it was to his job category or the minutes he exercised outside of work each week.

**FUTURE STUDIES**

Although not a part of this study, certain variables seem to have an effect on the health markers of the studied population. Among these variables are: 1) their immigrant status and the “healthy immigrant effect”; 2) the degree to which their current dietary habits differ from those they followed before their immigration; 3) the degree to which the health of Mexican Americans differ from that of native Mexicans; 4) the degree to which the “cultural founders effect” has influenced their current health. All of these variables are interesting in themselves and would provide opportunities for further research into the health status of the Mexican American population. In addition, an intervention study that would determine the benefits of testing for insulin resistance in an individual’s journey toward diabetes would be helpful. Armed with the beneficial outcomes of such a study, on-the-job testing for insulin resistance and diabetes educational workshops might become commonplace. We test for cholesterol to prevent heart disease. Why not test for fasting insulin to prevent insulin resistance and diabetes?
REFERENCES CITED:


Blair, S, Church, TS. The fitness, obesity, and health equation. Is physical activity the common denominator? JAMA 2004, 292(10), 1232-1234


Cordain L. 2002. The paleo diet, John Wiley and Sons, Hoboken, NJ.


Klein RG and Cruz-Uribe. 2000. Middle and later stone age large mammal and tortoise remains from Die Kelders Cave 1, Western Cape Province, South Africa. Journal of Human Evolution. 38:169-195.


Appendix A: Formulas
Formula 1 -- George Formula for Estimated VO2\textsubscript{max} (George et al., 1997)

\[ \text{VO2max (ml.kg}^{-1}.\text{min}^{-1}) = 43.513 + (6.564 \times \text{gender}) - (0.749 \times \text{BMI}) + (0.724 \times \text{PFA}) + (0.788 \times \text{PA-R}) \]

Where: Gender = 1 for Male; 2 for Female
BMI = body weight (kg)/stature (m)\(^2\)
PFA = Perceived Functional Ability (9) from list of questions
PA-R = Physical Activity Rating (10) from list of questions

Formula 2 -- Mean Arterial Blood Pressure

\[ (\text{DBP} + (\text{SBP-DBP})/3) \]
where
DBP is Diastolic Blood Pressure
SBP is Systolic Blood Pressure

Formula 3 -- Estimated VO2\textsubscript{max} via Rockport Walking Test (ACSM, 2009)

\[ \text{VO2max (ml.kg}^{-1}.\text{min}^{-1}) = 132.853 + 6.315(\text{gender}) - 0.3877(\text{age}) - 0.1692(\text{wt in lbs}) - 3.2649(\text{time in min}) - 0.1565(\text{HR}) \]

Where: Gender = 1 for Male; 0 for Female

Time is multiplied by 4 to obtain the time for a total mile given a quarter-mile walk
Appendix B: Forms
PARTICIPANT INFORMED CONSENT FORM
February 15, 2011

Please read the following material that explains this research study. Signing this form will indicate that you have been informed about the study and that you want to participate. We want you to understand what you are being asked to do and what risks and benefits—if any—are associated with the study. This should help you decide whether or not you want to participate in the study.

You are being asked to take part in a research project conducted by Roberta Martine, a graduate student in the University of Colorado at Boulder’s Department of Anthropology, 233 UCB, Boulder, CO 80309-0233. This project is being done under the direction of Dr. Greg Kandt. Roberta Martine can be reached at (303)579-4202. Dr. Kandt can be reached at (785)628-4371. Also, if you need help during the study, you can come to the offices at Our Lady of Guadalupe Cathedral where a bilingual receptionist will help you.

Project Description:

This research will study whether the type of work you do affects your risk of developing diabetes. You are being asked to be in this study because you are a male meatpacker and you do not currently have diabetes. It is entirely your choice whether or not to participate in this study. About 160 workers will be invited to participate in this research.

Procedures:

If you agree to take part in the study, you will be asked to participate in the following procedures. All procedures will occur at the offices of Our Lady of Guadalupe Cathedral in Dodge City, Kansas:

1) A hemoglobin A1C test. You will not need to fast before this test, which will indicate immediately whether you already have diabetes. To get blood for the test, a nurse will prick one of your fingers with a needle. If the A1C test indicates that you may already have diabetes, you will not be able to participate in the study and will be referred to a local agency or physician for treatment outside of work. A bilingual nurse will administer the test and give you the results.

2.) An interview about you, your medical history, and your physical activity. During the interview, a nurse will ask you questions like: “Do you feel pain in your chest when you do physical activity? Do you lose your balance because of dizziness or do you ever lose consciousness? What is your age?”

3.) Measurements of your height, weight, waist circumference, hip circumference, blood pressure and the thickness of a skinfold on your back right under your shoulder, and another skinfold at the back of your right upperarm. These measurements will be taken by the nurse who interviews you.
4.) **A Walking Fitness Test.** This will be administered in the offices of Our Lady of Guadalupe Cathedral and will require you to walk a quarter of a mile (400 meters) at a steady, but fast pace. The researcher (Roberta Martine) will take you through this procedure, but a nurse will be present in the clinic and available if needed. The fitness test should only take about 10 minutes.

5.) **A diet record.** We will need a record of the foods you eat during the 3-day period. We will provide the forms that you use to record this information. We will collect the forms the same day that you take your walking test. If you need help completing the forms, come to the offices of Our Lady of Guadalupe cathedral where a receptionist will help you.

7.) **Blood samples.** These blood samples will allow us to measure the levels of your diabetes related hormones. The blood samples will be taken on Sunday mornings so you will have to fast from Saturday night until Sunday morning. A little more than 3 teaspoonfuls of blood will be drawn from a vein on the inside of your elbow or the back of your hand. That area will be cleaned with antiseptic, and a tourniquet (an elastic band) will be placed around your upper arm to apply pressure and limit blood flow through the vein. A sterile needle will be inserted into the vein, and the blood collected in a syringe. During the procedure, the tourniquet will be removed to restore circulation. Once the blood has been collected and the needle is removed, the area will be covered to stop any bleeding.

Participating in this study will mean that you will need to attend a total of 3 sessions on 3 different days. All sessions will take place in the offices of Our Lady of Guadalupe Cathedral in Dodge City, Kansas. What you will be asked to do in each of the session, and our best estimate of the time it will take is outlined below:

1.) **Session 1** – During this session, a nurse will administer the hemoglobin A1C test and give you the results. The hemoglobin A1C test should take only a minute or two to draw the blood and five minutes to get the results. During the five minute wait, the nurse will be taking your measurements and obtaining your information. In total, the session should take about 30 minutes.

2.) **Session 2** – During this session, a nurse will interview you to obtain some medical and personal history and your assessment of your own physical fitness and activity. After the interview, we will take some blood samples and you will take the walking fitness test by walking a quarter of a mile at a fast pace. At the end of the session, you will receive a grocery certificate in appreciation of your participation in the study. This session will take about 30 minutes on a Sunday morning. You will need to fast from midnight on Saturday night until Sunday when the blood is drawn.

3.) **Session 3** – At this session, you will receive the results of your blood and physical fitness tests in a sealed envelope. A handout and/or film from the National Institute of Health on diabetes prevention will be provided. The session will last about a half-hour on a Sunday morning or early afternoon.
Risks and Discomforts:

When you participate in this study you must take a walking fitness test and provide blood samples. There are some potential risks to these parts of the study. If you do not have heart trouble, the risks are very low for the fitness test, which has been given to thousands of adults between the ages of 18 and 65 without any lasting adverse effects. However, whenever people exercise, there is always the possibility of risks and physical discomfort. Some of the risks during and right after the test include abnormal blood pressure, dizziness, fainting, muscle soreness, irregular heart rhythm, vomiting, stroke, acute myocardial infarction (heart attack), and death. Even if people exercise at their maximum level, the risk of any one of these is very low, less than 2 in every 1000 tests. Since your level of exercise will be less than maximum, your level of risk will also be lower. The risks associated with providing blood samples are slight and include the risk of excessive bleeding, light-headedness, the accumulation of blood under the skin, infection (a slight risk anytime the skin is broken), and multiple skin punctures to find veins.

This risk of anyone other than you and the study administrators obtaining your health information, will be kept to a minimum because only the researchers (Roberta Martine and Greg Kandt) and the bilingual nurse will see your individual data. In addition, the data will be kept in a place that is locked and inaccessible to unauthorized people.

Benefits:

The benefits of being in this study are that you will learn about how physically fit they are, about your blood test results, and about diabetes prevention.

Subject Payment:

You will be paid $15.00 for your participation in the study in the form of a grocery certificate. If you choose to withdraw before the end of session 2, however, you will receive nothing.

Source of Funding:

None.

Cost to Participant

There are no direct costs to you for participation in this study.

If You Are Injured or Harmed:

If you feel that you may have been harmed while participating in this study, you should inform Roberta Martine (303-579-4202) immediately. The cost for any treatment will be billed to you or your medical or hospital insurance. The University of Colorado at Boulder has no funds set aside for the payment of health care expenses for this study.

If you experience injury that requires medical attention, contact the investigator Dr. Greg Kandt (785-628-4371) and your personal physician immediately (if it is a medical emergency, first call 911).
Ending Your Participation:

You have the right to withdraw your consent or stop participating at any time. You have the right to refuse to answer any question(s) or refuse to participate in any procedure for any reason. Refusing to participate in this study will not result in any penalty or loss of benefits to which you are otherwise entitled.

Confidentiality:

We will make every effort to maintain the confidentiality of your data. That means only the researchers (Roberta Martine and Greg Kandt) and the nurse will see your individual data. No one else will know the outcome of your tests or whether you are at risk for diabetes. In addition, you will be assigned a code number and that number will be used throughout the investigation. Your code number will be tied to your name through a master list of names and code numbers, which will be kept in a locked file cabinet. All coded data will be kept in a separate location. The records will be destroyed by shredding them after 10 years.

Other than the researchers and nurse, only regulatory agencies such as the Office of Human Research Protections and the University of Colorado at Boulder Institutional Review Board may see your individual data as part of routine audits.

Questions?

If you have any questions regarding your participation in this research, you should ask the investigator before signing this form. If you should have questions or concerns during or after your participation, please contact Dr. Greg Kandt at (785)628-4371.

If you have questions regarding your rights as a participant, any concerns regarding this project or any dissatisfaction with any aspect of this study, you may report them -- confidentially, if you wish -- to the Institutional Review Board, 3100 Marine Street, Rm A15, 563 UCB, University of Colorado at Boulder, (303) 735-3702.
Authorization:

I have read this paper about the study or it was read to me. I know the possible risks and benefits. I know that being in this study is voluntary. I choose to be in this study. I know that I can withdraw at any time. I have received, on the date signed, a copy of this document containing 4 pages.

Name of Participant (printed) __________________________________________

Signature of Participant ___________________________ Date ______________

{Also initial all previous pages of the consent form.)

Name of Form Reader: __________________________

Signature of Reader ___________________________ Date ______________

(needed if participant consent was obtained through another party having read the consent form aloud)

I (name ) ___________________________ witnessed the consent process and saw the study participant sign the form.

Signature of Witness ___________________________ Date ______________

(needed if participant consent was obtained through another party having read the consent form aloud)

Name of Person to be Contacted in Case of Emergency: ______________

Phone Number: ______________
Resistencia a la insulina en hombres hispanos empaquetadores de carne
Investigadora principal: Roberta Martine

FORMULARIO DE CONSENTIMIENTO INFORMADO PARA EL PARTICIPANTE
15 febrero, 2011

Por favor, lea la siguiente información que explica este estudio de investigación. Al firmar este formulario, usted indicará que ha sido informado sobre este estudio y que desea participar en él. Queremos que comprenda lo que se le pide que haga y los riesgos y beneficios, si existieran, que pueden estar relacionados con este estudio. Esto deberá ayudarle a decidir si desea, o no, participar en este estudio.

Está usted siendo invitado a participar en un proyecto de investigación dirigido por Roberta Martine, una estudiante graduada del Departamento de Antropología de la Universidad de Colorado en Boulder (University of Colorado at Boulder), 233 UCB, Boulder, CO 80309-0233. El proyecto está supervisado por el Dr. Greg Kandt. Puede ponerse en contacto con Roberta Martine: (303) 579-4202. Para contactar al Dr. Greg Kandt: (785)628-4371. Además, si necesita ayuda durante este estudio, puede venir a las oficinas de la Catedral de Nuestra Señora de Guadalupe, en donde una recepcionista bilingüe le ayudará.

Descripción del proyecto:

Esta investigación estudiará si el tipo de trabajo que hace afecta al riesgo de desarrollar diabetes. Se le pide que participe en este estudio porque es usted empaquetador de carne y hombre, que en el presente no tiene diabetes. Es su elección si desea participar o no en este estudio. Se les invitará a participar a unos 160 trabajadores.

Procedimientos:

Si usted acepta participar en este estudio, se le pedirá que participe en los siguientes procedimientos. Todos los procedimientos se harán en las oficinas de la Catedral de Nuestra Señora de Guadalupe, en Dodge City, Kansas:

1) **Un examen de hemoglobina A1C.** No necesitará estar en ayunas para este examen, que indicará inmediatamente si usted ya tiene diabetes. Para conseguir la sangre para el análisis, una enfermera le pinchará un dedo con una aguja. Si el examen A1C indica que usted tiene diabetes, no podrá participar en este estudio y se le referirá a una agencia local o médico para que obtenga tratamiento fuera del trabajo. Una enfermera bilingüe hará el análisis de sangre y le dará los resultados.

2) **Una entrevistas acerca de usted, de su historial médico y de su actividad física.** Durante la entrevista, una enfermera le hará preguntas como: "¿Nota dolor en el pecho cuando hace ejercicio físico? ¿Pierde el equilibrio debido a un mareo, o pierde la conciencia? ¿Cuántos años tiene?"

3) **Medidas de altura, peso, cintura, cadera, presión sanguínea, la masa corporal/el grosor de la piel en su espalda, debajo de su hombro y el grosor de la piel en la parte de atrás de su brazo derecho.** Estas medidas las tomará la enfermera que le entrevistó.

4) **Un examen de condición física** (también llamados exámenes submaximales V02). Este examen se hará en las oficinas de la Catedral de Nuestra Señora de Guadalupe y requiere que usted camine la cuarta parte de una milla (400 metros) a un paso fijo pero a la vez rápido. La investigadora (Roberta Martine) le guiará en este procedimiento, pero habrá además una enfermera que estará presente y disponible si hiciera falta. Este examen durará unos 10 minutos.

5) **Historial de su dieta.** Necesitaremos documentación de la comida que ingiera durante un tres día periodo. Se le entregarán unos formularios que deberá llenar con esta información. Recogeremos los formularios el mismo día que camina la cuarta parte de una milla. Si necesita ayuda para completar los formularios, venga a las oficinas de la Catedral de Nuestra Señora de Guadalupe, en donde una recepcionista le ayudará.
6) **Muestras de sangre.** Estas muestras de sangre nos permitirán medir los niveles de las hormonas relacionadas con la diabetes. Las muestras de sangre se recogerán el domingo por la mañana, así que deberá estar en ayunas desde el sábado por la noche hasta la mañana del domingo. Se le extraerá un poco más de 3 cucharaditas de sangre de una vena del brazo, o de la parte superior de la mano. Esa zona se limpiará con un antiséptico. Luego se le pondrá un torniquete (una banda elástica) alrededor del brazo (parte superior), para aplicar presión y limitar así la circulación de la sangre por la vena.

Se le insertará una aguja esterilizada en la vena y se le extraerá sangre con una jeringuilla. Durante la extracción, se le quitará el torniquete para restablecer la circulación. Una vez se haya sacado la aguja, se cubrirá esa zona para parar cualquier hemorragia.

Si participa en este estudio, deberá asistir a un total de 3 sesiones en 3 días diferentes. Todas las sesiones serán en las oficinas de la Catedral de Nuestra Señora de Guadalupe en Dodge City, Kansas. Se le pedirá lo siguiente en cada sesión, que aproximadamente durará el tiempo que le indicamos abajo:

1) **Sesión 1** – Durante esta sesión, una enfermera le hará el examen de hemoglobina A1C y le dará los resultados. Este examen dura un minuto, o dos, para extraerle sangre y otros 5 minutos para obtener los resultados. Mientras tanta, una enfermera le entrevistará y le tomará su medidas. Entotal, la sesión durará unos 30 minutos.

2) **Sesión 2** – En esta sesión, una enfermera le entrevistará para obtener algunos datos sobre su historial médico y personal y su evaluación de su propia condición física y de su actividad. Después de la entrevista, se le sacará muestras de sangre y usted hará un examen de condición física por caminar la cuarta parte de una milla a un paso rápido. Al terminar la sesión, usted recibirá un certificado de supermercado como forma de aprecio por su participación en el estudio. Esta sesión durará unos 30 minutos un domingo por la mañana. Será necesario que usted esté en ayunas desde la medianoche el sábado por la noche hasta el domingo cuando se saca la muestra de sangre.

3) **Sesión 3** – En esta sesión, usted recibirá los resultados de su examen de sangre y examen fisico en un sobre sellado. Se le entregará también un boletín o una placa del Instituto Nacional de la Salud de prevencion de la diabetes. La sesión durará una media hora el domingo por la manana o temprano por la tarde.

**Riesgos e incomodidades:**

Cuando usted participe en este estudio, deberá hacer un examen de condición física por caminar la cuarta parte de una milla a un paso rápido y deberá hacerse un análisis de sangre. Hay ciertos riesgos potenciales en esta parte del estudio. Si usted no tiene problemas de corazón, los riesgos al hacer el examen de condición física son muy bajos. Este examen se le ha hecho a miles de adultos de edades comprendidas entre los 18 y 65 años, sin que tuvieran efectos adversos duraderos. Sin embargo, cuando las personas hacen ejercicio, siempre existe la posibilidad de riesgo e incomodidad física. Algunos de los riesgos que pueden ocurrir durante el examen VO2, y justo después del examen, son: presión arterial anormal, mareo, dolor muscular, ritmo cardíaco irregular, vómitos, coágulo en el cerebro, ataque de corazón y la muerte. Dado el caso en el que una persona haga ejercicio a nivel máximo, el riesgo de que cualquiera de estos riesgos ocurra es muy bajo, menos de 2 casos en cada 1000 exámenes. El nivel de ejercicio que usted haga no será el máximo, así que el nivel de riesgo será también más bajo. El riesgo asociado con el análisis de sangre es mínimo e incluye sangrar demasiado, mareo o náusea, o la acumulación de sangre debajo de la piel (un morado), infección (un leve riesgo en cualquier momento que se rompa la piel) y múltiples pinchazos en la piel para encontrar las venas.

Se mantendrá al mínimo el riesgo de que otras personas obtengan información sobre su salud, porque únicamente usted y los que administran los exámenes para este estudio (Roberta Martine y Greg Kandt), más la enfermera bilingüe verán sus resultados y datos individuales. Los datos se mantendrán en lugar seguro, protegido e inaccesible a personas no autorizadas.

**Beneficios:**

Los beneficios de participar en este estudio son que usted sabra sobre su salud física, los resultados de los análisis de sangre, y sobre la prevención de la diabetes.
Pago por Participación:

Usted recibirá un certificado por 15 dólares en despensa por su participación en el estudio. Si usted decide abandonar antes del final de la sesión 2 no lo recibirá.

Fuentes de financiamiento:

Ninguna.

Coste para el participante

No existen costes directos para usted por participar en este estudio.

En caso de accidente o daño:

Si usted cree que se ha dañado mientras participaba en este estudio, deberá informar a Roberta Martine (303-579-4202) inmediatamente. El coste de cualquier tratamiento se le cargará a usted o a su seguro médico.

La Universidad de Colorado en Boulder no tiene fondos para pagar los gastos de los cuidados médicos para este estudio. Si se lesionó y necesita atención médica, contacte a la Dr. Greg Kandt ((785)628-4371) y a su médico inmediatamente (si es una emergencia médica, primero llame al 911).

Terminar su participación en este estudio:

Usted tiene el derecho a retirarse o finalizar su participación en cualquier momento. Tiene el derecho a no contestar a cualquier pregunta que se le haga, o puede rehusar participar en cualquier procedimiento, por cualquier motivo. Negarse a participar en esta investigación no resultará en ninguna penalización o pérdida de beneficios a los que tenga derecho.

Confidencialidad:

Haremos todo lo posible por mantener confidencial sus datos e información. Eso significa que únicamente las investigadoras Roberta Martine y Greg Kandt, más la enfermera independiente, verán los resultados de sus exámenes. Nadie más conocerá los resultados de sus exámenes, o si usted tiene el riesgo de contraer diabetes. Además, se le asignará un código con números que será usado durante toda la investigación. Su código con números será unido a su nombre por medio de una lista maestra de nombres y códigos con números, y ésta estará guardada en un armario seguro. Todos los datos codificados se guardarán en un lugar separado. Toda la información va a ser destruida después 10 años con una máquina trituradora.

En adición a las investigadoras y la enfermera, agencias reguladoras como la Oficina de Protección de Investigaciones Humanas (Office of Human Protections) y el Comité de Investigaciones (Institutional Review Board) de la Universidad de Colorado en Boulder podrán considerar su información individual como parte de sus intervenciones rutinarias.

¿Tiene usted preguntas?

Si tiene cualquier pregunta relacionada con su participación en esta investigación, deberá preguntarle a la investigadora antes de firmar este formulario. Si tuviera preguntas o cuestiones que le preocupen durante o después de su participación, haga favor de contactar a la Dr. Greg Kandt ((785)628-4371).

Si tiene preguntas acerca de sus derechos como participante, o preocupaciones relacionadas con este proyecto, o cualquier queja con respecto a cualquier aspecto de este estudio, podrá informar, confidencialmente si lo desea, al...
Autorización:

He leído este formulario sobre el estudio, o se me ha leído. Conozco los posibles riesgos y beneficios. Sé que participar en este estudio es voluntario y escojo participar en el estudio. Sé que puedo retirarme en cualquier momento. He recibido, en la fecha de la firma, una copia de este documento que contiene 6 páginas.

Nombre del participante (letra de imprenta) ____________________________________________

Firma del participante __________________________________ Fecha ____________
(Escriba las iniciales de su nombre en cada página de este formulario de consentimiento informado).

Nombre de la persona que leyó el formulario: ________________________________________

Firma de ese lector/a: __________________________________ Fecha ____________
(Se necesita si el consentimiento del participante se obtuvo a través de una persona que leyó el formulario de consentimiento al participante)

Yo (nombre) __________________________________________ fui testigo del proceso de consentimiento informado y vi firmar el formulario al participante del estudio.

Firma del testigo __________________________________ Fecha ____________
(Se necesita si el consentimiento del participante se obtuvo a través de una persona que leyó el formulario de consentimiento al participante)

Nombre de la persona que debe contactarse en caso de emergencia:

________________________________________________________________________

Número de teléfono: ______________________________________________________
For Nurse

Cardiovascular Risk Assessment
Insulin Resistance in Hispanic Male Meatpackers Study

Participant number:  
Emergency contact:  
Phone:  

GENERAL HEALTH QUESTIONS

Yes / No 1. Do you use tobacco?
Yes / No 2. Have you been told you have high blood pressure? What is it?
Yes / No 3. Have you been told you have high cholesterol? What is it?
Yes / No 4. Do you have any allergies? What medications are you taking for them?
Yes / No 5. Do you have any problems with your joints, like arthritis or other joint disorders?
Yes / No 6. Do you have any other medical conditions we should be aware of?
Yes / No 7. Are you on a special diet, like a vegetarian diet or a weight loss diet?
Yes / No 8. When is the last time you were examined by a doctor?

SCREENING FOR CARDIOVASCULAR DISEASE  (Questions from the Physical Activity Readiness Questionnaire (PAR-Q))

Yes / No 1. Have you ever been told that you have a heart condition and that you should only do physical activity recommended by the doctor?
Yes / No 2. Have you ever been told that you have diabetes, or a thyroid condition, or a kidney or liver condition?
Yes / No 3. Do you feel pain in your chest, neck, jaw or arms when you do physical activity?
Yes / No 4. In the past month, have you had pain when you were not doing physical activity?
Yes / No 5. Do you lose your balance because of dizziness or do you ever lose consciousness?
Yes / No 6. Do you take drugs (for example, water pills) for your blood pressure or heart condition?
Yes / No 7. Do you have any of the following symptoms: heart palpitations, rapid heart rate, swollen ankles, shortness of breath, heart murmur, trouble breathing at night, burning or other pain in your legs when walking short distances?
Yes / No 8. Do you know of any other reason why you should not do physical activity?
Yes / No 9. Do you suffer from excessive fatigue?
Yes / No 10. Do you have any known lung problems like COPD or asthma?

SCREENING FOR CORONARY DISEASE RISK AND STUDY QUALIFICATIONS
Job (Circle One)

<table>
<thead>
<tr>
<th>Activity Level/Division</th>
<th>Fabrication</th>
<th>Kill Floor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heavy</td>
<td>chuck boner, tender puller, strip boner, re-hang rounds, forequarter marker, boxer</td>
<td>gutter, split saw operator, fold/stack hides, chisler/templer, face plates, 1&amp;2 legger, checker</td>
</tr>
<tr>
<td>Moderate</td>
<td>navel boner, brisket boner, top butt boner, strip boner, skirt trimmer, top butt trimmer, strip trimmer, brisket trimmer, chuck trimmer, loosemeat bagger</td>
<td>air knife operator, weasand rodder, fat puller, head hanger</td>
</tr>
<tr>
<td>Light</td>
<td>CO₂ injector, low temp/beef tissue belt, finger meat bagger, defect picker/sorter, spinal cord processor, frock sewer, upgrade trimmer, navel trimmer</td>
<td>case ready crew, vacuum operator, ear tickets, intestine flusher, yard worker, spinal cord worker, liver wrapper, contamination counter, hock processor, paper on/off worker</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
For Nurse

Time on Job _____ Yrs ________Months
Time spent exercising:  ____________ minutes ______ times per week.
AIC (Circle One):  1       2        3        4         5           6            7           8           >8

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height</td>
<td>ft     in</td>
</tr>
<tr>
<td>Weight</td>
<td>Lbs</td>
</tr>
<tr>
<td>Waist Circumference</td>
<td>in</td>
</tr>
<tr>
<td>Hip Circumference</td>
<td>in</td>
</tr>
<tr>
<td>BMI [lbs / 2.2]/[height in inches*2.54/100]^2</td>
<td></td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>/</td>
</tr>
<tr>
<td>Mean Arterial Blood (DBP + (SBP-DBP)/3)</td>
<td></td>
</tr>
</tbody>
</table>

1. Did anyone in your family have a heart attack or die suddenly?
2. If yes, who?
3. If yes, at what age did they have the heart attack or die?
4. Have you smoked in the last six months?
5. If yes, how many cigarettes per day?
6. How often do you drink alcoholic beverages?
   a. No, never.
   b. Yes, _____times a day.
   c. Yes, _____times a week.
   d. Yes, _____times a month.
7. Does anyone in your family have diabetes?
8. If yes, at what age did they get the disease?
9. Did anyone in your family ever die of the disease?
   Who?
   Age when they started symptoms?
   Age at death?
10. Which group best describes you:
    a. Black
    b. White
    c. Hispanic American
    d. Hispanic (not born in America) Years here:_______ Country of origin:________________________
    e. Asian
    f. Native American
    g. Other  ____________
## Insulin Resistance in Hispanic Male Meatpackers Study

### Interview Form -- For Nurse

**Participant number ______________________________ Date ______________

Current Medications: ______________________________________________________
_____________________________________________________________________
_____________________________________________________________________

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triceps Skinfold</td>
<td>cm</td>
</tr>
<tr>
<td>Subscapular Skinfold</td>
<td>cm</td>
</tr>
<tr>
<td>PFA (Perceived Function Ability) -- see Page 4 -- Use only one scale -- whichever participant feels most comfortable with.</td>
<td></td>
</tr>
<tr>
<td>PA-R (Physical Activity Rating) -- see Page 5</td>
<td></td>
</tr>
<tr>
<td>Calculated VO$_{2\text{max}}$ = 50.077 - (0.749\text{BMI}) + (0.724-PFA) + (0.788\text{PA-R})</td>
<td></td>
</tr>
</tbody>
</table>
### Perceived Functional Ability (PFA) Questions

**Suppose you were going to exercise continuously on an indoor track for 1 mile. Which exercise pace is just right for you --- not too easy and not too hard?**

Circle the appropriate number (any number, 1 to 13)

<table>
<thead>
<tr>
<th></th>
<th>Walking at a slow pace (18 minutes per mile or more or 11.25 minutes per km or more)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Walking at a medium pace (16 minutes per mile or 10 minutes per km)</td>
</tr>
<tr>
<td>4</td>
<td>Walking at a fast pace (14 minutes per mile or 8.75 minutes per km)</td>
</tr>
<tr>
<td>6</td>
<td>Jogging at a slow pace (12 minutes per mile or 7.5 minutes per km)</td>
</tr>
<tr>
<td>8</td>
<td>Jogging at a medium pace (10 minutes per mile or 6.25 minutes per km)</td>
</tr>
<tr>
<td>10</td>
<td>Jogging at a fast pace (8 minutes per mile or 5 minutes per km)</td>
</tr>
<tr>
<td>12</td>
<td>Running at a fast pace (7 minutes per mile or less or 4.5 minutes per km or less)</td>
</tr>
</tbody>
</table>

**How fast could you cover a distance of 3 miles and NOT become breathless or overly fatigued? Be realistic.**

Circle the appropriate number (any number, 1 to 13).

<table>
<thead>
<tr>
<th></th>
<th>I could walk the entire distance at a slow pace (18 minutes per mile or more; 11.25 minutes per km or more)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>I could walk the entire distance at a medium pace (16 minutes per mile; 10 minutes per km)</td>
</tr>
<tr>
<td>4</td>
<td>I could walk the entire distance at a fast pace (14 minutes per mile; 8.75 minutes per km)</td>
</tr>
<tr>
<td>6</td>
<td>I could jog the entire distance at a slow pace (12 minutes per mile; 7.5 minutes per km)</td>
</tr>
<tr>
<td>8</td>
<td>I could jog the entire distance at a medium pace (10 minutes per mile; 6.25 minutes per km)</td>
</tr>
<tr>
<td>10</td>
<td>I could jog the entire distance at a fast pace (8 minutes per mile; 5 minutes per km)</td>
</tr>
<tr>
<td>12</td>
<td>I could run the entire distance at a fast pace (7 minutes per mile or less; 4.5 minutes per km or less)</td>
</tr>
</tbody>
</table>
**Physical Activity Rating (PA-R)**

Select the number that best describes your overall level of physical activity for the last 6 months. Please include on-the-job activities.

<table>
<thead>
<tr>
<th>Number</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>I avoid walking or exertion. I never use the stairs; drive where possible instead of walking.</td>
</tr>
<tr>
<td>1</td>
<td>Light activity: I walk for pleasure, but I routinely use the stairs and occasionally exercise sufficiently to cause heavy breathing and perspiration.</td>
</tr>
<tr>
<td>2</td>
<td>Moderate Activity: I have 10 to 60 minutes per week of moderate activity -- such as golf, horseback riding, calisthenics, table tennis, bowling, weight lifting, yard work, cleaning house, walking for exercise.</td>
</tr>
<tr>
<td>3</td>
<td>Moderate Activity: I have over 1 hour per week of moderate activity as described in #2.</td>
</tr>
<tr>
<td>4</td>
<td>Vigorous Activity: I run less than 1 mile (or 1.6 km) per week or spend less than 30 minutes per week in comparable activity, such as running or jogging, lap swimming, cycling, rowing, aerobics, skipping rope, running in place, or engaging in vigorous aerobic activity (like soccer, basketball, tennis, racquetball, or handball).</td>
</tr>
<tr>
<td>5</td>
<td>Vigorous Activity: I run at least 1 (1.6 km) but less than 5 miles (8 km) per week or spend at least 30 but less than 60 minutes per week in comparable physical activity as described in #4.</td>
</tr>
<tr>
<td>6</td>
<td>Vigorous Activity: I run at least 5 (8 km) but less than 10 miles (16 km) per week or I spend at least 1 hour but less than 3 hours per week in comparable physical activity as described in #4.</td>
</tr>
<tr>
<td>7</td>
<td>Vigorous Activity: I run at least 10 (16 km) but less than 15 miles (24 km) per week or I spend at least 3 hours but less than 6 hours per week in comparable physical activity as described in #4.</td>
</tr>
<tr>
<td>8</td>
<td>Vigorous Activity: I run at least 15 (24 km) but less than 20 miles (32 km) per week or I spend at least 6 hours but less than 7 hours per week in comparable physical activity as described in #4.</td>
</tr>
<tr>
<td>9</td>
<td>Vigorous Activity: I run at least 20 (32 km) but less than 25 miles (40 km) per week or I spend at least 7 but less than 8 hours per week in comparable physical activity as described in #4.</td>
</tr>
<tr>
<td>10</td>
<td>Vigorous Activity: I run 25 miles (40 km) or more per week or I spend 8 or more hours per week in comparable physical activity as described in #4.</td>
</tr>
</tbody>
</table>
### Demographic Information

1. What is your age?  
2. What is your marital status?  
   a. Single  
   b. Married  
   c. Divorced  
   d. Widowed  
   e. Separated  
3. What was your highest grade in school?  
4. Have you ever done regular physical exercise outside of work:  
   a. No, never.  
   b. Yes, _____times a week, for _____minutes. Type of exercise:_______________
Evaluación del riesgo cardiovascular
Estudio sobre resistencia a la insulina en hombres hispanos trabajadores en frigoríficos

Número de participantes:          contacto de emergencia: 
Teléfono: 

PREGUNTAS GENERALES DE SALUD

Sí / No 1. ¿Consume tabaco? 
Sí / No 2. ¿Le han dicho que tiene presión arterial alta? ¿Cuál es? 
Sí / No 3. ¿Le han dicho que usted tiene el colesterol alto? ¿Cuál es? 
Sí / No 4. ¿Tiene alguna alergia? ¿Qué medicamentos está tomando para ello? 
Sí / No 5. ¿Tiene problemas con sus articulaciones, como la artritis u otros trastornos de la articulación? 
Sí / No 6. ¿Tiene alguna otra condición médica que debemos tener en cuenta? 
Sí / No 7. ¿Está usted en una dieta especial, como una dieta vegetariana o una dieta para bajar de peso? 
_______ 8. ¿Cuándo fue la última vez que lo examinó un médico? 

DETECCIÓN DE ENFERMEDADES CARDIOVASCULARES (Preguntas del Cuestionario de Actividad Física de Preparación (PAR-Q))

Sí / No 1. ¿Alguna vez le han dicho que tiene una enfermedad del corazón y que sólo debe realizar actividad física recomendada por el médico? 
Sí / No 2. ¿Alguna vez le han dicho que usted tiene diabetes o una enfermedad de la tiroides, o un riñón o enfermedad del hígado? 
Sí / No 3. ¿Siente dolor en el pecho, cuello, mandíbula o brazos cuando realiza alguna actividad física? 
Sí / No 4. En el último mes, ¿ha tenido dolor, cuando no estaba haciendo actividad física? 
Sí / No 5. ¿Pierde el equilibrio debido a mareos o alguna vez ha perdido la conciencia? 
Sí / No 6. ¿Toma medicamentos (por ejemplo, píldoras de agua) para la presión arterial o enfermedad del corazón? 
Sí / No 7. ¿Tiene alguno de los siguientes síntomas: palpitaciones, taquicardia, tobillos hinchados, falta de aliento, soplo cardíaco, dificultad para respirar durante la noche, ardor o algún otro dolor de en las piernas al caminar distancias cortas? 
Sí / No 8. ¿Sabe usted de cualquier otra razón por la que no debe hacer actividad física? 
Sí / No 9. ¿Sufre de fatiga excesiva? 
Sí / No 10. ¿Tiene algún problema pulmonar conocida como EPOC o asma?
Habilidad funcional percibida Preguntas

Suponga que va a hacer ejercicio continuado de 1 milla en una pista interior. ¿Qué ritmo de ejercicio es el adecuado/correcto para usted --- que no sea demasiado fácil ni demasiado duro?

Haga un círculo en el número apropiado (cualquier número del 1 al 13).

<table>
<thead>
<tr>
<th>Número</th>
<th>Opción</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Caminar a ritmo lento (18 minutos por milla o más, o 11,25 minutos por km o más)</td>
</tr>
<tr>
<td>2</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Caminar a ritmo medio (16 minutos por milla o 10 minutos por km)</td>
</tr>
<tr>
<td>4</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Caminar a ritmo rápido (14 minutos por milla u 8,75 minutos por km)</td>
</tr>
<tr>
<td>6</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Hacer “jogging” (o correr con ritmo cómodo) a un ritmo lento (12 minutos por milla o 7,5 minutos por km)</td>
</tr>
<tr>
<td>8</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Hacer “jogging” a ritmo medio (10 minutos por milla o 6,25 minutos por km)</td>
</tr>
<tr>
<td>10</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Hacer “jogging” a ritmo rápido (8 minutos por milla o 5 minutos por km)</td>
</tr>
<tr>
<td>12</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Correr a ritmo rápido (7 minutos por milla o menos, o 4,5 minutos por km o menos)</td>
</tr>
</tbody>
</table>

¿Cuánto tiempo tardaría en hacer la distancia de 3 millas sin quedarse sin aliento y sin fatigarse excesivamente? Sea realista.

Haga un círculo en el número apropiado (cualquier número del 1 al 13).

<table>
<thead>
<tr>
<th>Número</th>
<th>Opción</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Podría caminar las 3 millas a ritmo lento (18 minutos por milla o más; 11,25 minutos por km o más)</td>
</tr>
<tr>
<td>2</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Podría caminar las 3 millas a ritmo medio (16 minutos por milla; 10 minutos por km)</td>
</tr>
<tr>
<td>4</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Podría caminar las 3 millas a ritmo rápido (14 minutos por milla; 8,75 minutos por km)</td>
</tr>
<tr>
<td>6</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Podría correr las 3 millas a ritmo lento (12 minutos por milla; 7,5 minutos por km)</td>
</tr>
<tr>
<td>8</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Podría correr las 3 millas a ritmo medio (10 minutos por milla; 6,25 minutos por km)</td>
</tr>
<tr>
<td>10</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Podría correr las 3 millas a ritmo rápido (8 minutos por milla; 5 minutos por km)</td>
</tr>
<tr>
<td>12</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Podría correr las 3 millas a ritmo rápido (7 minutos por milla o menos; 4,5 minutos por km o menos)</td>
</tr>
</tbody>
</table>
**Clasificación de actividad**

Seleccione el número que mejor describa su nivel de actividad física en general, en los últimos 6 meses. Por favor incluya las actividades físicas que haga en el lugar del trabajo.

<table>
<thead>
<tr>
<th>Nivel</th>
<th>Descripción</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Evito caminar o hacer esfuerzo. Siempre uso las escaleras; manejo en coche a los lugares cuando es posible, en vez de caminar.</td>
</tr>
<tr>
<td>1</td>
<td>Actividad ligera: Camino porque me gusta, pero uso las escaleras por rutina y ocasionalmente hago ejercicio suficiente que me produce una respiración fuerte y sudoración.</td>
</tr>
<tr>
<td>2</td>
<td>Actividad moderada: Hago de 10 a 60 minutos de actividad moderada por semana, como por ejemplo golf, montar a caballo, calistenia (gimnasia o deporte), tenis de mesa, jugar a los bolos, levantar pesas, trabajar en el jardín, limpiar la casa, caminar como ejercicio.</td>
</tr>
<tr>
<td>3</td>
<td>Actividad moderada: Hago 1 hora de actividad moderada por semana, como la descrita en # 2.</td>
</tr>
<tr>
<td>4</td>
<td>Actividad enérgica: Corro menos de 1 milla (o 1,6 km) por semana, o practico un ejercicio similar menos de 30 minutos por semana, como por ejemplo correr, &quot;jogging,&quot; nadar en piscinas, montar en bicicleta, remar, ejercicio aeróbico, saltar a la cuerda, correr en un mismo lugar, o practico ejercicio aeróbico vigoroso (como jugar al fútbol, baloncesto, tenis, racketbol o balonmano).</td>
</tr>
<tr>
<td>5</td>
<td>Actividad enérgica: Corro al menos 1 milla (1,6 km) pero menos de 5 millas (8 km) por semana, o paso al menos 30 minutos pero menos de 60 minutos por semana haciendo una actividad física similar a la descrita en # 4.</td>
</tr>
<tr>
<td>6</td>
<td>Actividad enérgica: Corro al menos 5 millas (8 km) pero menos de 10 millas (16 km) a la semana, o paso al menos 1 hora pero menos de 3 horas por semana haciendo una actividad física similar a las descrita en # 4.</td>
</tr>
<tr>
<td>7</td>
<td>Actividad enérgica: Corro al menos 10 millas (16 km) pero menos de 15 millas (24 km) por semana, o paso al menos 3 horas pero menos de 6 horas por semana haciendo una actividad física similar al descrito en # 4.</td>
</tr>
<tr>
<td>8</td>
<td>Actividad enérgica: Corro al menos 15 millas (24 km) pero menos de 20 millas (32 km) por semana, o paso al menos 6 horas pero menos de 7 horas por semana haciendo una actividad física similar a la descrita en # 4.</td>
</tr>
<tr>
<td>9</td>
<td>Actividad enérgica: Corro al menos 20 millas (32 km) pero menos de 25 millas (40 km) por semana, o paso al menos 7 horas pero menos de 8 horas haciendo una actividad física similar a la descrita en # 4.</td>
</tr>
<tr>
<td>10</td>
<td>Actividad enérgica: Corro 25 millas (40 km) o más por semana, o paso 8 ó más horas por semana haciendo una actividad física similar a la descrita en # 4.</td>
</tr>
<tr>
<td>Time</td>
<td>Description of Food</td>
</tr>
<tr>
<td>------</td>
<td>---------------------</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Hora</td>
<td>Descripción de los alimentos</td>
</tr>
<tr>
<td>------</td>
<td>-----------------------------</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix C: Feedback
Insulin Resistance Among Male Hispanic Meatpackers Study
Study Participants
Re: Your Results

Dear Participant,

The results of your blood tests and your walking test are enclosed. The blood tests that you should look at are the fasting insulin, fasting glucose, triglycerides (a blood fat), LDL (a protein that measures the number of bad cholesterol carrying particles in the blood stream), and adiponectin (that measures how well insulin is working). Your body may be insulin-resistant if your fasting insulin, glucose and triglyceride measurements are high and the adiponectin is low. Your metabolism may be insulin-resistant if your blood levels cross these thresholds:

- Fasting insulin more than 15 ulU/mL
- Fasting glucose more than 100 mg/dL
- Triglycerides more than 150 mg/dL
- Adiponectin less than 7.7 µg/ml

Insulin resistance occurs when your body needs more insulin to maintain normal levels of blood glucose and triglycerides. This condition may tend to overwork your pancreas in its attempt to supply the insulin that your body needs. Diabetes occurs when the pancreas is so overworked that it can no longer make enough insulin.

Since insulin resistance is related to being inactive and overweight, insulin-resistant individuals would tend to have more bad cholesterol (LDL) in their blood. If your LDL is above 130 mg/dL, you may be more likely to develop hardening of the arteries and heart disease in later years.

VO2max is a measurement of your physical fitness. To find out how physically fit you are compared to other men your age, find your age and your VO2max measurement in the following chart. The higher the percentile in which your VO2max falls, the more physically fit you are compared to others in your age group. For example, someone who is 42 years old with a VO2max of 40 would be between the 40th and the 50th percentile. That is, he would be more physically fit than a little more than 40% of the population.

<table>
<thead>
<tr>
<th>Percentile</th>
<th>20-29</th>
<th>30-39</th>
<th>40-49</th>
<th>50-59</th>
<th>60+</th>
</tr>
</thead>
<tbody>
<tr>
<td>90</td>
<td>55.1</td>
<td>52.1</td>
<td>50.6</td>
<td>49.0</td>
<td>44.2</td>
</tr>
<tr>
<td>80</td>
<td>52.1</td>
<td>50.6</td>
<td>49.0</td>
<td>44.2</td>
<td>41.0</td>
</tr>
<tr>
<td>70</td>
<td>49.0</td>
<td>47.4</td>
<td>45.8</td>
<td>41.0</td>
<td>37.8</td>
</tr>
<tr>
<td>60</td>
<td>47.4</td>
<td>44.2</td>
<td>44.2</td>
<td>39.4</td>
<td>36.2</td>
</tr>
<tr>
<td>50</td>
<td>44.2</td>
<td>42.6</td>
<td>41.0</td>
<td>37.8</td>
<td>34.6</td>
</tr>
<tr>
<td>40</td>
<td>42.6</td>
<td>41.0</td>
<td>39.4</td>
<td>36.2</td>
<td>33.0</td>
</tr>
<tr>
<td>30</td>
<td>41.0</td>
<td>39.4</td>
<td>36.2</td>
<td>34.6</td>
<td>31.4</td>
</tr>
<tr>
<td>20</td>
<td>37.8</td>
<td>36.2</td>
<td>34.6</td>
<td>31.4</td>
<td>28.3</td>
</tr>
<tr>
<td>10</td>
<td>34.6</td>
<td>33.0</td>
<td>31.4</td>
<td>29.9</td>
<td>26.7</td>
</tr>
</tbody>
</table>

Everyone, especially overweight and insulin-resistant people, should be aware that diet and exercise can improve their metabolism. Eating a heart healthy diet and exercising at least 30 minutes five times a week is important to keep you healthy with a low risk of heart disease and diabetes.

If you have any questions, please bring this information to your physician and ask for his or her guidance.
Estudio sobre resistencia a la insulina entre hombres hispanos que trabajan en los frigoríficos.

Estudio de los participantes
Re: los resultados

Estimado participante,

Los resultados de los análisis de sangre y su prueba física se adjuntan. Los análisis de sangre que usted debe ver son la insulina en ayunas, glucosa en ayunas, triglicéridos (grasa en la sangre), LDL (una proteína que mide el número de partículas portadoras de colesterol malo en la sangre), y la adiponectina (que mide como trabaja la insulina). Su cuerpo puede ser resistente a la insulina si la insulina la glucosa en ayunas, y las mediciones de triglicéridos son altos y la adiponectina es baja. Su metabolismo puede ser resistentes a la insulina si sus niveles de sangre pasa estas medidas:

De insulin en ayunas superior a 15 uU/ml
Glucosa en ayunas mayor de 100 mg / dl
Los triglicéridos más de 150 mg/dl
La adiponectina menos de 7,7 µg / ml

La resistencia a la insulina se produce cuando el cuerpo necesita más insulina para mantener los niveles normales de glucosa en la sangre y los triglicéridos. Esta condición puede tender al exceso de trabajo del páncreas en su intento de suministrar la insulina que su cuerpo necesita. La diabetes ocurre cuando el páncreas es tan sobrecargado de trabajo que ya no puede producir suficiente insulina.

Como la resistencia a la insulina está relacionada con la inactividad y el sobrepeso, los individuos resistentes a la insulina que tienden a tener más colesterol malo (LDL) en la sangre. Si su LDL está por encima de 130 mg/dl, usted puede estar más propenso a desarrollar endurecimiento de las arterias y enfermedades del corazón en el futuro.

VO2max es una medida de su aptitud física. Para saber cómo está físicamente en comparación a otros hombres de su edad, encuentre su edad y su medida de VO2 máx en el siguiente cuadro. Cuanto más alto el número de percentil de su VO2máx, indica que usted tiene mejor aptitud física comparado con otros hombres de su edad. Por ejemplo, alguien que tiene 42 años de edad, con un VO2máx de 40 estará entre el percentil 40th y 50th. Es decir, él estará en mejor condición física que un poco más del 40% de la población.

<table>
<thead>
<tr>
<th>Percentil</th>
<th>20-29</th>
<th>30-39</th>
<th>40-49</th>
<th>50-59</th>
<th>60+</th>
</tr>
</thead>
<tbody>
<tr>
<td>90</td>
<td>55.1</td>
<td>52.1</td>
<td>50.6</td>
<td>49.0</td>
<td>44.2</td>
</tr>
<tr>
<td>80</td>
<td>52.1</td>
<td>50.6</td>
<td>49.0</td>
<td>44.2</td>
<td>41.0</td>
</tr>
<tr>
<td>70</td>
<td>49.0</td>
<td>47.4</td>
<td>45.8</td>
<td>41.0</td>
<td>37.8</td>
</tr>
<tr>
<td>60</td>
<td>47.4</td>
<td>44.2</td>
<td>44.2</td>
<td>39.4</td>
<td>36.2</td>
</tr>
<tr>
<td>50</td>
<td>44.2</td>
<td>42.6</td>
<td>41.0</td>
<td>37.8</td>
<td>34.6</td>
</tr>
<tr>
<td>40</td>
<td>42.6</td>
<td>41.0</td>
<td>39.4</td>
<td>36.2</td>
<td>33.0</td>
</tr>
<tr>
<td>30</td>
<td>41.0</td>
<td>39.4</td>
<td>36.2</td>
<td>34.6</td>
<td>31.4</td>
</tr>
<tr>
<td>20</td>
<td>37.8</td>
<td>36.2</td>
<td>34.6</td>
<td>31.4</td>
<td>28.3</td>
</tr>
<tr>
<td>10</td>
<td>34.6</td>
<td>33.0</td>
<td>31.4</td>
<td>29.9</td>
<td>26.7</td>
</tr>
</tbody>
</table>

Todos, especialmente personas con sobrepeso y resistencia a la insulina, deben ser conscientes de que la dieta y el ejercicio pueden mejorar su metabolismo. Comer una dieta saludable para el corazón y hacer ejercicio por lo menos 30 minutos cinco veces a la semana es importante para mantenerse saludable con un bajo riesgo de enfermedades cardiovasculares y diabetes.

Si usted tiene alguna pregunta, favor lleve esta información a su médico pidale consejo.
Next Steps Feedback Presentation – English Contents

Slide 1:

Next Steps

What comes next??

Slide 2:

Incidence of Diabetes

- 8% of general population
- 12% (1 in 8) of Hispanic population
- By 2050, the percentage of Hispanics with diabetes is expected to be 50%
  - Heredity (15%)
  - Culture (85%)

Slide 3:

What is Diabetes?

- Diabetes is a disease in which the body does not produce or properly use insulin.
- Insulin is a hormone that is needed to convert sugar, starches, and other foods into energy needed for daily life.

Slide 4:

More on Diabetes

- Diabetes is a disease that is characterized by:
  - High blood glucose (sugar) – greater than 125
  - Low blood insulin
  - Death of B-cells in the pancreas
  - Many vascular complications that can lead to amputations, blindness, kidney and heart disease
  - Nerve damage
Slide 5:

Diabetes Complications

- Retinopathy (blindness)
- Nephropathy (kidney problems)
- Feet ulceration and/or amputations
- Heart attack
- Stroke
- Erectile dysfunction (inability to have an erection)

Slide 6:

What is prediabetes?

- Higher than normal blood glucose (100-125)
- Lower blood insulin
- Diabetes results in 10 years
- More likely to develop heart disease and stroke

Slide 7:

What is insulin?

- A hormone produced in the B-cells of the pancreas
- Regulates blood sugar
- After a meal, causes the muscles to use the blood sugar and the liver to stop releasing stored sugar into the blood.

Slide 8:

What is insulin resistance?

- Blood glucose can be normal (below 100)
- Fasting insulin is high
- Liver, muscles, and pancreas are resistant to insulin...therefore, more and more insulin is needed to clear the blood of sugar.
- Pancreas grows tired clearing blood sugar, B-cells may die
- Diabetes may result within years, but not always. Some people have strong pancreases!!
Slide 9:

Progression of diabetes

- Normal glucose metabolism
- Insulin resistance
- Prediabetes
- Diabetes

Slide 10:

Your blood results

- Fasting insulin – High levels = resistance
- Fasting glucose – Levels between 100 and 125 = prediabetes
- Adiponectin – Low levels = resistance
- Triglycerides – High levels = resistance
- LDL – High levels = lead to clogged arteries
- ApoB – High levels = lead to clogged arteries

Slide 11:

A1C

- 5.0 y 5.4 = 2 x the normal chance of getting diabetes
- 5.5 y 5.9 = 5
- 6.0 y 6.4 = 16
- 6.5 = Diabetes

Slide 12:

Our goal -- Restore normal metabolism!!

How??

- Wake up insulin metabolism
- Eat a good diet.
- Improve physical fitness through daily exercise.
Slide 13:

Wake up insulin metabolism
- Lose 7% of your body weight...
  - Liver will become less insulin resistant
- Start exercising
  - Muscles will become less insulin resistant

Slide 14:

Good diets:
- Use portion control
- Have less fat
- Have less sugar
- Have less salt

Slide 15:

Portion control
- Use smaller plates
- Restrict portion sizes:
  - Rice, pasta, beans – no more than size of ice-cream scoop
  - Low-fat cheese – no more than size of four-dice
  - Meat and fish – no more than size of deck of cards
  - Butter and margarine
  - Sugar
  - Salt
- Increase portion sizes:
  - Green vegetables
  - Fruit
  - Egg whites

Slide 16:

Do's and Don'ts of a Good Diet

Don't
Regular Soda

Whole Milk Products
Sour Cream
Milk
Yogurt
Mayonnaise

Fried Foods

Salty Snacks

Supersize

Manteca

Do

Diet Soda or Water

Skim Milk/Low Fat Products
Sour Cream
Milk
Yogurt

Light Mayonnaise

Grilled or Steamed Foods

Fruits and Vegetables

Regular Size

Vegetable or Olive Oil

Slide 17:

Read Food Labels
☐ Look for fewer calories, less fat, higher fiber, less salt
Slide 18:

Current level
- See physical fitness chart in envelope
- How to read the percentiles...

Slide 19:

Physical Activity Rule!!
- Get at least 30 minutes of moderate-intensity physical activity (e.g., brisk walking, yard work, and actively playing with children, for example, riding bicycles or playing soccer) 5 days a week.

Slide 20:

Improve Physical Fitness
- Walk More
  - Build automatic walking into day
    - Take stairs
    - Park far away
    - Walk at lunch time
  - Goal – 10,000 steps/day
- Move More
  - Do some strength exercises
  - Get into moderate category at work – more activity = more money
Pasos a seguir

¿Qué hacer ahora?

Incidencia de diabetes

- 8% de la población general
- 12% (1 de 8) de la población hispana
- Para 2050 se espera que un 50% de la población hispana tenga diabetes
  - Herencia (15%)
  - Cultura (85%)

¿Qué es la diabetes?

- La diabetes es una enfermedad en la que el cuerpo no produce insulina o la usa inadecuadamente.

- La insulina es una hormona necesaria para convertir el azúcar, los almidones y otros alimentos en la energía necesaria para realizar las actividades diarias.

Más sobre la diabetes

La diabetes es una enfermedad que se caracteriza por:

- Glucosa elevada en la sangre (azúcar)- más de 125
- Insulina baja en la sangre
- Muerte de las B-células en el páncreas
- Muchas complicaciones vasculares que pueden llevar a amputaciones, ceguera, enfermedad de los riñones y del corazón
- Daño al sistema nervioso

Slide 5:

Complicaciones de la diabetes
- Retinopatía (ceguera)
- Nefropatía (afección de los riñones)
- Úlceras en los pies o amputaciones
- Ataque cardiaco
- Accidente cerebrovascular
- Disfunción eréctil (in capacidad para tener erección del pene o impotencia)

Slide 6:

¿Qué es la prediabetes?
- La glucosa en la sangre está más alta de lo normal (100-125)
- Menor insulina en la sangre
- Resultado en posible diabetes en 10 años
- Tendencia a desarrollar una enfermedad del corazón o derrame cerebral/embolia

Slide 7:

¿Qué es la insulina?
- Una hormona producida en las B-células del páncreas.
- Regula el azúcar en la sangre
- Después de una comida, hace que los músculos usen el azúcar de la sangre y hacen que el hígado deje de enviar azúcar almacenada a la sangre.

Slide 8:

¿Qué es resistencia a la insulina?
- La glucosa de la sangre puede ser normal (por debajo de 100)
- El nivel de insulina en ayunas es alto
El hígado, los músculos y el páncreas se hacen resistentes a la insulina por eso se necesita cada vez más insulina para limpiar el azúcar de la sangre. El páncreas se cansa de limpiar el azúcar de la sangre, las B-células pueden morir. La diabetes puede aparecer en algunos años, pero éste no es siempre el caso. Hay personas con páncreas fuertes.

Slide 9:

Evolución de la diabetes
- Metabolismo normal de glucosa
- Resistencia a la insulina
- Prediabetes
- Diabetes

Slide 10:

Su Sangre
- El nivel de insulina en la sangre en ayunas-niveles altos 15 = resistencia
- El nivel de glucosa en la sangre en ayunas- niveles entre 100 y 125 = prediabetes
- La adiponectina-niveles bajos 7,7 = resistencia
- Los triglicéridos-niveles altos 150= resistencia
- LDL-niveles altos 130 = lleva a arterias obstruidas

Slide 11:

A1C
- 5.0 y 5.4 = 2
- 5.5 y 5.9 = 5
- 6.0 y 6.4 = 16
- 6.5 = Diabetes
Slide 12:

Nuestra meta – Recuperar un metabolismo normal

¿Cómo?
- Despierte el metabolismo de la insulina
- Coma una dieta equilibrada
- Mejore su forma física con ejercicio diario.

Slide 13:

Despierte el metabolismo de la insulina
- *Pierda 7% de su peso corporal*
  - El hígado se hará menos resistente a la insulina
- *Comience a hacer ejercicio*
  - Los músculos se harán menos resistentes a la insulina

Slide 14:

Dietas buenas:
- *Controle las porciones*
- *Coma menos grasa*
- *Coma menos azúcar*
- *Coma menos sal*

Slide 15:

Control de las porciones
- *Use platos más pequeños*
- *Haga las porciones más reducidas:*
  - Arroz, pasta, frijoles – limite la porción a una cucharada (el tamaño de una cucharada de helado)
  - Queso bajo en grasa – limite la porción a 4 cubitos/trozos del tamaño de un dado
  - Carne y pescado – limite la porción al tamaño de una baraja de cartas
  - Mantequilla y margarina
Azúcar
Sal
☐ Haga porciones más grandes:
Verduras y hortalizas verdes
Fruta
Claras de huevo

Slide 16:

El Sí y No de una buena dieta

No
☐ Bebidas carbonadas regulares
☐ Productos de la leche entera
☐ Crema agria
☐ Leche
☐ Yogurt
☐ Mayonesa
☐ Comida frita
☐ Aperitivos salados
☐ Porciones enormes
☐ Manteca

Sí
☐ Bebidas carbonadas bajas en azúcar o agua
☐ Leche descremada o productos lácteos bajos en grasa
☐ Crema agria
☐ Leche
☐ Yogurt

☐ Mayonesa light
☐ Comida a la parrilla/plancha o al vapor
☐ Fruta y verduras
☐ Porciones regulares
☐ Aceite vegetal o de oliva

Slide 17:

Lea el etiquetado de los productos alimenticios
☐ Opte por los alimentos con menos calóricas, menos grasa, más fibra y menos sal
Slide 18:

**Nivel actual**
- Vea el cuadro de forma física en el sobre
- Cómo leer los porcentajes...

Slide 19:

**Physical Activity Rule!!**
- Haga al menos 30 minutos de actividad física, de intensidad moderada, 5 veces a la semana (por ejemplo: caminar vigorosamente, trabajar en el jardín o huerto, jugar activamente con niños, como por ejemplo montar en bicicleta, o jugar al fútbol).

Slide 20:

**Mejore su forma física**
- **Camine más**
  - Introduzca el caminar en su rutina diaria
  - Tome las escaleras
  - Aparque lejos del lugar al que va
  - Camine durante el almuerzo
  - Meta – 10.000 pasos al día
- **Muévase más**
  - Haga ejercicios de fuerza (como levantar pesas)
  - Haga trabajos de categoría o nivel moderado– más actividad = más dinero
Appendix D: Advertising
Free diabetes screening!

Now 1 in 8 Latinos have diabetes

Help us find out more about the disease. Join our research study and Earn a grocery certificate

Where: Our Lady of Guadalupe Cathedral
Dodge City, KS
When: 3 Sunday mornings, before and after the Spanish Mass. 30 minutes each time.
Requirements: You must be a Latino, male meatpacker

For more information please contact:
The cathedral office receptionist
620-227-9240
or
Roberta Martine
University of Colorado
Boulder, CO
303-579-4202
roberta.martine@colorado.edu
Detección de diabetes gratis!

Ahora 1 de cada 8 latinos tiene diabetes

Ayúdenos a saber más sobre la enfermedad. Únase a nuestro estudio de investigación y Gane un certificado para compra de alimentos.

**Lugar:** Catedral de Nuestra Señora de Guadalupe  
Dodge City, KS  
**Cuándo:** Tres domingos por la mañana, antes y después de la misa en español (30 minutos cada vez).  
**Requisitos:** Ud. debe ser latino, hombre y empacador de carne

**Para obtener más información, póngase en contacto con:**

La recepción de la oficina de la catedral  
620-227-9240  
o  
Roberta Martine  
Universidad de Colorado  
Boulder, CO  
303-579-4202  
roberta.martine@colorado.edu
VOLUNTEERS NEEDED FOR RESEARCH STUDY ON DIABETES

Do you have reason to be concerned about diabetes? Have you seen people in your family, neighborhood, or church slowly die of the disease? It is a fact that people are becoming more and more susceptible to the disease. For example, did you know that females born in 2000 have a 38.5 percent risk of developing the disease; while males born in that year have a 32.8 percent risk of developing it? However, for Hispanics born in 2000, the risk of getting diabetes is known to be even higher and runs at about 50%. For those who develop diabetes by age 40, the disease will shorten their lives by 11.6 years and cut their quality of life for 22 years, through conditions like heart disease, kidney disease, and blindness.

We are providing diabetes screening and conducting a diabetes research study at Our Lady of Guadalupe Cathedral, here in Dodge City. For the study we are recruiting 160 male meatpackers. If you qualify for the study, you will receive free diabetes screening. You qualify if you can answer “Yes” to the following questions:

- Are you currently diabetes free?
- Are you relatively physically inactive outside of work?
- Are you healthy?
- Have you been performing your job more than three months?

Come help us fight the disease. To sign up, contact the receptionist at the cathedral offices at 620-225-4802. The study will require a time commitment of three 30-minute sessions at Our Lady of Guadalupe Cathedral, before or after the Sunday Spanish Mass. When you complete the second session, you will receive a $15.00 grocery certificate. During the third session, you will receive your blood test results.
SE NECESITAN VOLUNTARIOS PARA UN ESTUDIO DE INVESTIGACIÓN SOBRE LA DIABETES

¿Tiene usted una razón para estar preocupado por la diabetes? ¿Ha visto usted a gente de su familia, vecindario o de su iglesia morir lentamente de esta enfermedad? Es un hecho que las personas son cada vez más susceptibles a contraer esta enfermedad. Por ejemplo, ¿sabía usted que las mujeres nacidas en el año 2000 tienen un 38,5 % de riesgo de desarrollar la diabetes; mientras que los hombres nacidos en ese año tienen un 32,8 % de riesgo de desarrollar esta enfermedad? Sin embargo, para los hispanos nacidos en el 2000, el riesgo de desarrollar la diabetes es sabido que es incluso más alto y está en el 50 %. Para aquellos que desarrollan la diabetes a los 40 años, la enfermedad reducirá sus vidas 11,6 años e interrumpirá la calidad de vida durante 22 años, con condiciones como la enfermedad de corazón, la enfermedad de riñón y la ceguera.

Estamos proporcionando detección de diabetes y realizando un estudio de investigación de la diabetes en la Catedral Nuestra Señora de Guadalupe, aquí en Dodge City. Para el estudio se están reclutando a 160 hombres empacadores de carne. Si usted califica para el estudio, usted recibirá detección de diabetes gratis. Podrá participar si puede contestar “Sí” a las siguientes preguntas:

¿En este momento no padece la diabetes?
¿Es usted relativamente inactivo físicamente fuera del trabajo?
¿Está usted sano?
¿Ha estado trabajando en su trabajo más de tres meses?

Ayúdenos a combatir la diabetes. Para participar, contacte a la recepcionista de la oficina de la. El estudio necesitará de tres sesiones de 30 minutos cada una de su tiempo en la Catedral Nuestra Señora de la Guadalupe, ya sea antes o después de la misa en español de los domingos. Cuando usted complete la segunda sesión, usted recibirá su certificado de 15 dólares en despensa. Durante la tercera sesión, usted recibirá los resultados de sus pruebas de sangre.
FOR IMMEDIATE RELEASE: March 15, 2011

Help wanted: Little work, short hours, lifelong rewards

By Linn Ann Huntington
Special to The Dodge City Daily Globe

Wanted: 160 Hispanic meatpackers -- men, between the ages of 18 and 55.
To do: Not a whole lot.
For how long: 90 minutes
Rewards: $15 in free groceries. Oh yeah, it might save one’s life too.

Roberta Martine is a woman with a mission. Not a giant, worldwide mission—just a small mission to save a few lives in Dodge City, Kan.

But she needs some help.

Martine, a doctoral student at the University of Colorado, is seeking volunteers for her dissertation project. That research project, in biological anthropology, is examining ways to help Hispanic men lower their risk of developing type 2, or adult-onset, diabetes.

Martine’s study comes in the wake of findings published by a scientist at the Centers For Disease Control in Atlanta, Ga., who analyzed a 12-year study on diabetes. Those findings showed that the average American male born in 2000 runs a 32.8 percent risk of developing diabetes sometime during his life. But for the average Hispanic male, that risk is much higher—51.9 percent.

Martine recalls that she was so startled by those figures, she had to read them again. One in two male Hispanics in the U.S. will develop diabetes.

That’s when Martine says she knew she had to do something to try to change that statistic.

Martine is searching for 160 male Hispanic meatpackers in Dodge City, Kan., who would like to be part of her research study. To qualify, men must be between the ages of 18 and 55, must currently be diabetes-free, physically inactive outside of work, be in relatively good health and must have been at their jobs for more than three months.

“We want to identify people at risk and help prevent them from getting diabetes,” Martine said. “That’s one big purpose of the study.

“Also, we are trying to relate the level of physical activity with the risk of diabetes. Meatpackers have jobs that range from light to heavy exertion. We think on-the-job activity will affect the level of susceptibility to the disease.”

Martine, who lives in Hays and teaches at Fort Hays State University, is currently driving from Hays to Dodge City several times a month. All of the screening tests are being administered at Our Lady of Guadalupe Cathedral in Dodge City under the supervision of registered nurses. The study requires a time commitment of three 30-minute sessions at the church before or after the Sunday Spanish Mass.

After the second session, participants will receive a $15 grocery certificate. During the third session, participants will receive the results of their blood tests, Martine said.

According to the CDC, type 2 diabetes, in which the body gradually loses its ability to use insulin, accounts for 90-95 percent of all diabetes cases. The CDC also predicts that if current trends don’t change, as many as one-third of all U.S. adults could have diabetes by 2050.

Diabetes raises the risk of high blood pressure, stroke, and heart disease. It can also lead to kidney failure, blindness and amputations.

“We know that a structured lifestyle program that includes losing weight and increasing physical activity can prevent or delay type 2 diabetes,” said Ann Albright, PhD, director of the CDC’s Division of Diabetes Translation, in a 2011 news release.

183
Risk factors cited by the CDC include race, ethnicity, family history, older age, obesity, and a sedentary lifestyle.

Hispanics, African Americans and American Indians are among the groups at greatest risk, according to an article published by the CDC in January. While 26 million Americans have been diagnosed with the disease, another 27 percent, or about 7 million people, do not even know that they have it, the article states.

Martine wants to change that—at least in the Dodge City area. She notes that as grim as the statistics are, the CDC article also offers hope. Better screening has resulted in more people being able to manage their diabetes and live longer, more productive lives.

That’s why Martine’s study is offering free screening. She urges male Hispanic meatpackers in the Dodge City area “to come help us fight the disease.” To sign up, individuals may contact the receptionist at the cathedral office at 620-225-4802.
Se necesita ayuda: Poco trabajo, pocas horas, recompensas de por vida

Linn Ann Huntington
Especial de The Dodge City Daily Globe

Se necesitan: 160 hombres hispanos empaquetadores de carne entre 18 y 55 años
Para: No demasiado trabajo
Recompensa: $15 para comprar comestibles. Y además, puede que le salven la vida.

Roberta Martine es una mujer con una misión. No es una misión gigantesca ni mundial, sino una misión pequeña que salvará unas cuantas vidas en Dodge City, Kansas. Pero necesita ayuda.

Martine, una estudiante de doctorado de la Universidad de Colorado, busca voluntarios para su estudio doctoral. Su proyecto de investigación en antropología biológica examina maneras de ayudar a bajar el riesgo de desarrollar Diabetes tipo II, o Diabetes de inicio en la edad adulta, en hombres hispanos.

El estudio de Martine está en correlación directa con los resultados publicados por un científico de los Centros para el Control de Enfermedades en Atlanta, Georgia., que analizó un estudio sobre la diabetes durante 12 años. Los resultados mostraron que el hombre común americano nacido en el año 2000 tenía un 32,8 % de riesgo de desarrollar la diabetes en algún momento de su vida. Pero para el hombre común hispano, ese riesgo era mucho mayor—51,9 %.

Martine explica que se sorprendió tanto por los resultados de ese estudio que tuvo que leer los resultados de nuevo. Uno de cada dos hombres hispanos en los EEUU desarrollará la diabetes. Es entonces cuando Martine supo que tenía que hacer algo para cambiar esa estadística.

Martine busca 160 hombres hispanos empaquetadores de carne en Dodge City, Kansas, a los que les gustaría formar parte de su estudio de investigación. Las condiciones para poder participar son: hombres entre 18 y 55 años, que no tengan diabetes en el presente, que sean físicamente inactivos fuera del trabajo, que tengan relativamente buena salud y que hayan trabajado en su puesto de trabajo más de tres meses. “Queremos identificar a las personas que tienen propensión a la diabetes y ayudarles a que no la desarrollen,” dijo Martine. “Éste es uno de los grandes propósitos de este estudio.”

“También intentamos relacionar el nivel de actividad física con el riesgo de desarrollar la diabetes. Los empaquetadores de carne tienen trabajos que varían de esfuerzo ligero a esfuerzo pesado. Creemos que la actividad física en el puesto de trabajo afectará el nivel de susceptibilidad, riesgo o propensión a desarrollar esta enfermedad.”

Martine, que vive en Hays e imparte clases en la Universidad de Fort Hays, en el presente se desplaza varias veces al mes de Hays a Dodge City. Todas las pruebas de detección se están administrando en la Catedral Nuestra Señora de
Guadalupe en Dodge City, bajo la supervisión de enfermeras registradas. Su estudio requiere el compromiso de tres sesiones de 30 minutos en la Catedral, antes o después de la Misa en español de los domingos. Después de la segunda sesión, los participantes recibirán un certificado de $15 para comprar comida. Durante la tercera sesión, los participantes recibirán los resultados de sus analíticas, dijo Martine.

De acuerdo con los CDC, la Diabetes tipo II o no insulinodependiente, en la que el cuerpo pierde gradualmente la habilidad de usar la insulina, representa el 90-95% de todos los casos de diabetes. Los CDC a su vez predicen que si las tendencias actuales no cambian, un tercio de todos los adultos en EEUU podrían tener diabetes para 2050.

La diabetes eleva el riesgo de tener la presión alta, de tener una embolia, y enfermedades del corazón. También puede llevar a la insuficiencia renal, a la ceguera y a amputaciones.

“Tenemos contancia de que tener un estilo de vida estructurado que incluye perder peso e incrementar la actividad física puede prevenir, o retrasar, la aparición de la Diabetes tipo II,” dijo Ann Albright, PhD, directora de los “CDC’s Division of Diabetes Translation,” en un boletín informativo de 2011. Los factores de riesgo citados por los CDC incluyen la raza, la etnia, la herencia familiar, la edad avanzada, la obesidad y el estilo de vida sedentario.

Los hispanos, los afro-americanos y los indios americanos son los grupos que tienen mayor riesgo, de acuerdo con un artículo publicado en los CDC en enero. Mientras a 26 millones de americanos se les ha diagnosticado esta enfermedad, otro 27%, o unos 7 millones de personas, no saben que padecen de esta enfermedad, expone el artículo.

Martine desea cambiar esto-al menos en el área de Dodge City. Explica que aún siendo la estadística tan desalentadora, el artículo de los CDC ofrece a su vez esperanza. El perfeccionamiento en las pruebas de detección ha resultado en que las personas con diabetes pudieran ser capaces de controlar su enfermedad y vivir más años, vidas más productivas.

Por esta razón, el estudio de Martine ofrece pruebas de detección gratuitas. Martine anima a los hombres hispanos empaquetadores de carne del área de Dodge City a “que nos ayuden a luchar contra esta enfermedad.” Para inscribirse, pueden contactar con la recepcionista en la oficina de la Catedral 620-225-4802.

Traducido por Chita Espino-Bravo, PhD, Profesora Asistente de español, Departamento de Lenguas Modernas, FHSU.
Detección de diabetes gratis!

Únase a nuestro estudio de investigación y gane un certificado para compra de alimentos.

Lugar: Catedral de Nuestra Señora de Guadalupe, Dodge City, KS
Cuándo: Tres domingos por la mañana, antes y después de la misa en español (30 minutos cada vez).
Requisitos: Usted debe ser latino, hombre y empaqueter de carne

Llame 620-225-4802 para hacer una cita.
Appendix E: Correlation Matrix
Matrix of Correlation Outcomes for All Study Participants

|                         | BMI     | Waist Circumference | Waist/Hip Ratio | Systolic BP | Diastolic BP | Fasting Insulin | fasting glucose | Fasting Cholesterol | Total Cholesterol | HOMA | HOMA IR | QUICK CIRC | Waist Circumference | Age |
|-------------------------|---------|---------------------|------------------|-------------|--------------|----------------|-----------------|--------------------|--------------------|------|---------|-----------|---------------------|-----|--------|
| BMI                     | NS      | NS                  | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| Waist Circumference     | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| Waist/Hip Ratio         | NS      | <0.001              | <0.001           | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| Systolic BP             | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| Diastolic BP            | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| Fasting Insulin         | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| fasting glucose         | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| Fasting Cholesterol     | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| Total Cholesterol       | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| HOMA                    | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| HOMA IR                 | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| QUICK CIRC              | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| Waist Circumference     | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |
| Age                     | NS      | NS                 | NS               | NS          | NS           | NS             | NS              | NS                 | NS                 | NS   | NS      | NS        | NS                  | NA  | NS     |

NS = Not Significant; NA = Not Applicable
(Includes both Borderline and Significant Results -- Significant Results are in Bold)