Glycemic Response to Fast and Slow Digestible Carbohydrate in High And Low Aerobic Fitness Men

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ABSTRACT

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The purpose of this study was to determine the difference in the glycemic response of high and low aerobic fitness men to glucose, and one novel slow and fast digestible carbohydrate (CHO). The glycemic index (GI) value of both novel CHO was determined for the high \( n = 6 \) and low \( n = 6 \) aerobic fitness men. Overall, GI values were 76.44±16.02 for the fast CHO and 48.96 ±17.30 for the slow CHO. Between the high and low fitness men the GI differed significantly, the fast CHO was found to be 86.71±16.15 and 66.17±7.12 and the slow CHO was found to be 59.13±8.50 and 38.79±18.38, for the low and high fitness men respectively. The glycemic response was significantly different among the trials (glucose, fast and slow CHO, \( p = .0001 \)). The GI of the novel CHO (fast and slow CHO) depended upon the aerobic fitness level of healthy young men.

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CHAPTER 1: INTRODUCTION

The glycemic index (GI) is a tool to describe the 2 hour blood glucose response following ingestion of carbohydrates (CHO). It is thought to be a useful tool in choosing foods that can improve an athletic performance (Burke, Collier, & Hargreaves, 1998), aid in the long term maintenance of blood glucose (Jenkins et al., 2002; Wolever, Jenkins, Jenkins, & Josse, 1991) and to assist in improving glycemic control for health implications as in diabetic populations (Jenkins et al., 1981). The glycemic response to ingestion of food, indicates the rate of digestion and absorption of the nutrients and their contribution to a change in blood glucose. Most often the glycemic response is numerically represented as the area under the curve (AUC); additional measurements to describe the glycemic response include maximal blood glucose value, percent increase and elapsed time to maximal blood glucose value.

Previous literature suggests that a GI value will be similar between individual’s regardless of characteristics, since the GI is calculated as the 2 hour glycemic response to a test meal as compared to the response to a standard glucose challenge (i.e., AUC for the test meal divided by the AUC for the standard glucose challenge; Wolever et al., 1991). However, additional literature has demonstrated that the glycemic response to fast and slowly digestible CHO is not be identical within individuals of high aerobic fitness levels (Wu, Nicholas, Williams, Took, & Hardy, 2003). Furthermore, only recently have GI trials characterized the difference in the glycemic response between individuals of high aerobic fitness and low aerobic fitness and sedentary individuals and some suggest that
the GI value may be different based on training status (Mettler, Lamprecht-Rusca, Stoffel-Kurt, Wenk, & Colombani, 2007; Mettler, Wenk, & Colombani, 2006).

Therefore, the purpose of this investigation was to determine the difference in the glycemic response of high and low aerobically fit men to a standard glucose challenge and to two test samples of one slowly digestible CHO and one fast digestible CHO, and to determine the GI values for each of the novel CHOs among the whole group and to determine if there were differences based on aerobic fitness.

**Statement of the Problem and Purpose of the Investigation**

The purpose of this investigation was to determine if there was a difference in the glycemic response of high and low aerobically fit men to a standard glucose challenge and to two test samples; one slowly digestible CHO and one fast digestible CHO. The current body of literature suggests that a GI value will be similar between individuals regardless of characteristics, since the GI is calculated as the 2 hour glycemic response to a test meal as compared to the response to a standard glucose challenge (Wolever et al., 1991). Recently, GI trials characterized the difference in the glycemic response between individuals of high aerobic fitness and low aerobic fitness and sedentary individuals (Mettler, et al., 2007; Mettler et al., 2006) using mixed meals as a test food. To the investigators knowledge, the current investigation is the first to test differences in the glycemic response between individuals of high aerobic fitness and low aerobic fitness using a pure CHO test food.
Hypotheses

The hypotheses for this investigation include:

1. The calculated glycemic index for each trial will be the same in high and low fitness subjects.

2. The 2 hour glycemic response will be lower in the slow digestible than the fast digestible carbohydrate for all subjects.

3. The glycemic response will be less in the high aerobic fitness subjects than the low aerobic fitness subjects.

4. The calculated glycemic index for the test foods will be a low glycemic index and high glycemic index food, for the slow CHO and fast CHO.

Limitations

This investigation will be limited by the following factors:

1. Test results depended on the cooperation of the subjects and their ability to follow all instructions given to them prior to their inclusion in this investigation.

2. The subjects were asked to maintain normal physical activity level during the period of the investigation and asked to abstain from increased physical activity 36 hours prior to the testing, subjects were expected to follow said instructions.

3. Subjects were asked to maintain constant dietary habit during the period of this investigation and asked to abstain from food and alcohol 24 hours prior to glycemic response trials and subjects were expected to follow said instructions.

4. All glycemic response trials were performed at approximately the same time of the day in the mornings (within 3 hours) to avoid effects of diurnal variation.
5. Subjects were asked to consume all test foods within a 15 minute time span and the investigator cannot account for small amounts of food remaining in the jar and cup containing the test food.

6. Total subjects ($N = 12$), and group subjects (high fitness, $n = 6$, low fitness, $n = 6$), previous literature used a minimum 10 subjects per group (Mettler et al., 2007); the limitation of total subjects ($N$) was acceptable because of the difficulty to recruit subjects.

**Delimitations**

The scope of this research study was delimited by the following factors:

1. The recruited subjects were males Caucasians, apparently healthy volunteers between the ages of 20 - 29 years attending Ohio University. The subjects were divided into two groups: high aerobic fitness men and low aerobic fitness fit men. The number of subjects in each group was 6 and 6 respectively.

2. The data collection procedures were performed on 3 days over 1 week, each trial at least 24 hours apart and maximum 72 hours apart from each other (in case of technical difficulties on the part of the subject or tester).

3. Subjects were asked to refrain from all physical activity (above normal daily activities), alcohol, and tobacco for 36 hours prior to testing. Furthermore, subjects were asked to come to each glycemic response testing session following an overnight fast (water only, 12 hours), remain seated and refrain from unnecessary extraneous movement during the glycemic response trials. The subjects were allowed to walk to the restroom.

4. The study was conducted from January 2007 to June 2007.

5. Subjects completed a health history questionnaire to determine their relative level of risk according to the standards set forth by the American College of Sports
Medicine (ACSM). Only individuals who were at low risk—non-smoking and did not have a metabolic condition—according to the aforementioned standards participated in the investigation.

6. Subjects read and signed a human consent form prior to participation.

7. The Institutional Review Board at Ohio University approved this project (see Appendix A).

**Significance of the Study**

The significance of this investigation lies in determining the influence of aerobic fitness levels on the GI of novel CHO test foods. Furthermore, determining the glycemic response in each of these groups will be valuable so that a more comprehensive knowledge of the use and limitation of using the GI for health, as it pertain to metabolic disorders, sports nutrition, and to the food industry for future development of commercial foodstuffs. Finally, the investigation will assess how body composition and aerobic fitness level will be valuable so that a more comprehensive knowledge of the use of the glycemic response.

**Definition of Terms**

This study considered the following definitions of the terminology to be used:

*Air displacement plethysmography.* A method used for assessing body volume and body composition by pressure changes precipitated between the test chamber and reference chamber by a moving diaphragm mounted on the common wall between the chambers; BOD POD is a brand name.

*Percent body fat.* The percentage of body mass that is not composed of lean muscle, water, bones or vital organs.
**Body composition.** Body composition is referred to as the breakdown of body make-up, i.e., fat, lean muscle, bone and water content

**Maximal oxygen consumption.** Maximal oxygen consumption (VO$_{2\text{max}}$) is the maximum rate for oxygen consumption by the body during maximum exercise.

**Glycemic index.** A tool to describe the 2 hour blood glucose response of a fixed amount of available carbohydrate from a test food compared to a standard food (glucose) consumed by the same subject (Jenkins et al., 2002).

**Glycemic response.** The amount of change in blood glucose from baseline after ingestion of foodstuffs numerically represented as the area under curve.

**Normoglycemia.** Blood glucose value $\leq$ 100 mg/dL, measured after an overnight fast (American Diabetes Association, 2007a; American Diabetes Association, 2007b).
CHAPTER 2: REVIEW OF LITERATURE

Carbohydrates (CHO) are an abundant fuel source for cells in the human body. Between 45 and 60% of total caloric intake is reported to come from carbohydrate ingestion in typical diets throughout the world (“Carbohydrates in human nutrition,” 1998). In the United States a trend towards increasing CHO ingestion is purported to coincide with a decrease in physical activity which are independently and collectively contributing to the increasing prevalence of obesity and diabetes (Augustin, Franceschi, Jenkins, Kendall, & La Vecchia, 2002).

Dietary CHO is ingested as monosaccharides, and disaccharides, collectively known as “simple sugars”, as well as polysaccharides also known as complex CHO or “starches”. Digestion of CHO begins in the mouth, but absorption of CHO into the blood stream does not occur until the small intestines. The majority of CHO is absorbed into the blood stream to be used as an immediate fuel or to be stored as glycogen for late use (Bouche, Serdy, Kahn, & Goldfine, 2004; Jequier, 1994; Levin, 1994). The glycemic index (GI) was proposed in 1981 (Jenkins et al., 1981) as a tool to describe the two hour blood glucose (glycemic) response following ingestion of CHO. The methodology for determining the GI of a CHO food is based on dependent on the mean glycemic response of a test sample (i.e., numerous individuals), the GI variability of each individual’s response contribute to the mean GI score. Clearly a GI score for a test sample of healthy individuals may have limited value for groups with specific and unique characteristics, such as above average aerobically fit group. The subsequent sections in this review of literature will focus on CHO metabolism, the GI and the application of the GI in individuals of above average aerobic fitness.
Carbohydrate Metabolism

Digestion and Absorption

CHO are classified by the degree of polymerization, where monosaccharides are composed of a single unit, disaccharides two units, oligosaccharides between 3 and 10 units, and polysaccharides greater than 10 units (Cummings & Englyst, 1995). Monosaccharides, including glucose, fructose and galactose, are the simplest and smallest form, with glucose being the most abundant. Common disaccharides include sucrose, lactose and maltose, which are digested in the small intestine by sucrase, lactase and maltase, respectively. The metabolism of each molecule of disaccharides results in the formation of two simple sugars–sucrose becomes a single glucose and fructose; lactose becomes glucose and galactose; and maltose becomes two glucose molecules. Polysaccharides are longer chains of monosaccharide consisting of hundreds of units and are commonly referred to as starches. Glycogen is the major animal starch in the human body, while amylase, amylopectin and resistant starch are the primary plant polysaccharides. Polysaccharides are digested beginning with hydrolysis by salivary amalyase in the oral cavity and pancreatic amylase in the small intestine (Levin, 1994). Once in the small intestines, glucose and galactose are actively transported, via secondary active transport, into the intestinal absorptive epithelial cells and absorbed across the enterocyte brush border membrane from the intestinal lumen by sodium dependent glucose transporters (SGLT-1) (Bouche et al., 2004). Although glucose and galactose compete for absorption, glucose typically is more completely absorbed. In contrast, fructose is absorbed by glucose transport protein-5 from the intestinal lumen and does not compete with the other simple sugars for absorption. On the basolateral membrane all
the simple sugars are transported by glucose transporter-2 (GLUT-2) for eventual incorporation into the blood (Lentze, 1995; Levin, 1994). Importantly, the majority of galactose and fructose that enter the blood are metabolically transformed into usable glucose in the liver (Bouche et al., 2004).

**Maintenance of Blood Glucose**

CHO are organic compounds that supply energy to the working body. Glucose is the most important CHO for human metabolism and its use, as opposed to other fuel sources, is largely governed by availability. Glucose is stored as glycogen in the liver and skeletal muscle tissue through a process known as glycogen synthesis (glycogenesis) and is broken down through glycogenolysis to dynamically maintain levels of blood and cellular glucose, respectively. Additionally, approximately five grams of free glucose is “stored” within the blood (Flatt, 1995). In general there is more total glycogen in the skeletal muscles at about 480g, because of its overall greater volume, but the liver contributes approximately 120g of glycogen as well (Flatt, 1995). However, depending on body size, and total CHO consumed, the amount of total glycogen stored in healthy adults has been estimated to be between 200-500g (Flatt, 1995). When ingestion of CHO exceeds its immediate metabolic need, glucose is converted into glycogen and stored for later use. However, continued overconsumption of CHO chronically increases insulin and glycogen concentrations, and reduces the need for fat as a fuel source, promoting CHO oxidation and storage (Jequier, 1994). Unfortunately, glycogen storage in the skeletal muscle and liver is limited because of the hydrophilic nature of carbohydrates (Flatt, 1995). Three grams of water is associated with each 1g of glycogen, thus imposing a physiologic limit due to energy density in the form of glycogen, and although
metabolically “wasteful” additional CHO may also be converted to fatty acids and stored as triglyceride in adipose tissue depots (Flatt, 1995). A chronic increase in insulin blunts fat metabolism and contributes to elevated blood glucose and lipids (Brooks, Fahey, & Baldwin, 2005). Moreover, overconsumption of CHO is often accompanied by an increase in dietary fat intake which is accompanied by a decrease in fat oxidation and only compounds these metabolic disturbances. This cascade may play a greater role in developing adipose tissue, as opposed to the minor pathway of converting CHO for storage as triglycerides within adipose tissue (Jequier, 1994).

**Glucose Metabolism**

Glucose is metabolized in the cytosol of all cells in a process known as glycolysis. Glycolysis involves the breakdown of glucose to two molecules of pyruvic acid resulting in the relatively quick production of adenosine triphosphate (ATP; “usable” cellular energy). Pyruvic acid may also be converted to lactic acid to replenish nicotinamide adenine dinucleotide (NAD) a vital molecule that is essential for glycolysis to occur. While pyruvic and lactic acid are transported into the mitochondrion of the cell for further oxidation (aerobic glycolysis). When one molecule of glucose is completely metabolized it results in the formation of 32 molecules of ATP, however, if lactic acid is its fate, then the resulting ATP production is limited to two molecules (Bouche et al., 2004).

While at first, lactate may seem like an inefficient use of glucose, it can in and of itself be used as a fuel source. Research has demonstrated that lactate in the circulating blood is taken up by skeletal muscle, cardiac heart and the liver. In the liver the gluconeogenic pathway can convert it back to glucose, and in skeletal and cardiac muscle
it can be converted back to pyruvic acid for metabolism in the citric acid cycle (Bouche et al., 2004). During, aerobic conditions glycolysis readily occurs in the active red skeletal muscle tissue. Blood glucose serves as the immediate fuel source, and as blood glucose is depleted or exercise is extended beyond a few minutes, muscle glycogen is broken down to supply the active muscles with a “fresh” source of glucose (Flatt, 1995). Skeletal muscle is a glycolytic tissue and very active in the breakdown of glucose during exercise. When consuming CHO prior to exercise, rates of plasma glucose appearance increase, followed by a concomitant rise in glucose rate of disappearance (Febbraio, Chiu, Angus, Arkinson, & Hawley, 2000). Assuming that the glucose rate of disappearance matches glucose oxidation, the exogenous CHO consumed must have entered the pathway of glycolysis. Additionally, insulin levels increased following a CHO load and provided an additional stimulus for glucose disposal at rest, prior to exercise and muscle contraction (Febbraio et al., 2000). Thus, blood glucose and insulin levels prior to exercise provide indication of the fuel source used for exercise and endurance performance. Additional support of glycolytic flux through glycolysis is displayed after ingestion of CHO. For example, following the consumption of 75g CHO beverage solution the rate of glucose disappearance increased, while reducing the liver glucose output and lowering glycogenolysis and gluconeogenesis (Marmy-Conus, Fabris, Proietto, & Hargreaves, 1996). This suggests glucose metabolism results in the maintenance of plasma glucose during rest and even a subsequent bout of exercise. As a person transitions from a state of low to moderate and high need for glucose and energy, further metabolic processes increase to maintain blood glucose (in large part due to glycogenolysis).
Glycogenolysis

Glycogenolysis is regulated by the enzyme glycogen phosphorylase (Bouche et al., 2004; Hargreaves, 2000) that in turn is directly controlled by the hormones glucagon and epinephrine. When glucose is needed, glucagon and epinephrine concentrations in the blood increase in conjunction with an increase in rate of glucose appearance (Coker & Kjaer, 2005). The increase in the rate of glucose appearance supplies the active skeletal muscle with glucose. The ability of the epinephrine to directly stimulate the rate of glucose appearance in the blood stream is dependent upon it’s delivery to the liver (Coker & Kjaer, 2005). In particular, liver glycogen breakdown maintains blood glucose, while muscle glycogen provides immediate fuel for glycolysis within the working muscle (Bouche et al., 2004).

The metabolic state of the body may determine the use of glycogen as a fuel source. In particular, acute ingestion of CHO has been demonstrated to lower liver glucose output because of higher plasma glucose from dietary absorption (Marmy-Conus et al., 1996). Moreover, this rise in blood glucose triggers a rapid increase in pancreatic insulin levels that blunt glycogenolysis (Marmy-Conus et al., 1996). Conversely, during times of low blood sugar, which may be common during prolonged sub-maximal exercise lasting longer than 90 minutes, muscle glycogen may be a primary fuel source. A large body of research has demonstrated the outcomes of exercise and partially depleted blood sugar. Specifically, Febbraio et al. (2000) demonstrated that muscle glycogen concentrations decreased at 20 minutes and continued to decrease throughout 120 minutes of sub-maximal cycling. Similarly, consuming 75g of CHO 45 minutes prior to cycling to exhaustion research found that the vastus lateralis was significantly depleted of
glycogen (Kirwan, Cyr-Campbell, Campbell, Scheiber, & Evans, 2001; Kirwan, O’Gorman et al., 2001). These findings support the usage of muscle glycogen during prolonged exercise and demonstrate how exhaustion is often characterized by muscle glycogen depletion.

**Gluconeogenesis**

During a fasted state, or at times of low dietary CHO availability (metabolic stress), glucose must be formed from non-carbohydrate sources in a process known as gluconeogenesis (Bouche et al., 2004). Gluconeogenesis occurs in the liver and is accelerated by the release of the adrenal cortex hormone cortisol that is secreted in response to stress. Gluconeogenesis is an important process for the maintenance of blood glucose, especially considering the brain and central nervous system are dependent upon glucose as an energy substrate (Jequier, 1994). Importantly, the non-CHO sources for gluconeogenesis are abundant, and include, but are not limited to lactate, pyruvate, glycerol and select amino acids. However, while muscle and adipose tissue lack the specific enzymes to perform gluconeogenesis, the liver routinely performs this service for the body.

**Glycogenesis**

In contrast to liver glycogenolysis and gluconeogenesis, which function to increase blood glucose, glycogen synthesis (glycogenesis) functions to reduce blood glucose. Specifically, when the metabolic demands are low, but blood glucose is high, the pancreatic hormone insulin is secreted to assist glucose into cells and store the temporarily “excess” glucose as glycogen. Glucose is converted to glycogen primarily in the muscles; this process is controlled by the enzyme glycogen synthase (Bouche et al.,
Factors including the type of tissue, current levels of glucose in the blood and tissue, presence of insulin, translocation of glucose transporter 4 (GLUT-4), and glycogen status of the tissue determine the uptake of glucose. During times when muscle glycogen is low, contracting muscle increases their uptake of glucose by increasing glucose transport via GLUT-4 translocation (Wojtaszewski et al., 2003). Skeletal muscle is the primary site of GLUT-4 expression, GLUT-4 translocation occurs in both types of skeletal muscle, red and white muscle fibers, additionally; a single bout of exercise has been shown to increase the total number of GLUT-4 present at the plasma membrane surface (Goodyear, Hirshman, Smith, & Horton, 1991). Greater skeletal muscle mass is associated with a decrease in adipose tissue, and active muscle increases its uptake and storage of carbohydrate more rapidly than non-active muscle (Graham & Adamo, 1999). Endurance training increases the total amount of GLUT-4 in the skeletal muscle, or increases the number of GLUT-4 translocated to the cell surface; additionally, slow twitch type-I oxidative muscle fibers contain a larger concentration of GLUT-4 (Rodnick, Holloszy, Mondon, & James, 1990). GLUT-4 expression increases, and glycogen synthase activity increases as a result of exercise training (Christ-Roberts et al., 2004). Physically active populations and athletes are more sensitive to insulin, and display lower insulin concentrations; therefore, in combination with increased GLUT-4 translocation activity and increase in abundance of GLUT-4, less insulin is required to transport glucose across the plasma membrane. Therefore, higher fitness levels-increased muscle mass, GLUT-4 expression and translocation, and decreased adipose tissue-may alter how ingested CHO is metabolized due to the quality of the muscle (Graham & Adamo, 1999).
The liver and skeletal muscle plays critical roles in reducing blood glucose when it becomes high. The first step in glucose metabolism is transport into the cells for phosphorylation to glucose-6-phosphate (Bouche et al., 2004). Glucokinase expressed in the liver is responsible for this reaction. Glucokinase found in the liver has the ability to control and reduce blood glucose concentrations when they are high. Glucose 6-phosphate is then converted to glucose 1-phosphate which then forms uridine-diphosphate glucose (UDP-glucose). Glycogen synthase then catalyzes the conversion of UDP-glucose into glycogen (Nielsen & Richter, 2003). Glycogen synthase catalyzes the incorporation of UDP-glucose into glycogen.

**GLUT-4**

The movement of glucose from the blood into tissue where it is oxidized or stored for later oxidation is mediated by facilitated diffusion. Specifically, glucose transport by a series of specific glucose transport proteins is essential to the maintenance of blood glucose. Glucose transporter 1 (GLUT-1) is located on the sarcolemma of cells and involved in continuous glucose uptake (Klip & Paquet, 1990). The more abundant protein, GLUT-4, is moved (translocated) from its intracellular store to the membrane of metabolically active tissue. The translocation of GLUT-4 is a result of the pancreatic hormone insulin or calcium-mediated muscle contraction (Richter et al., 2004). While muscle contraction and insulin bring about GLUT-4 translocation independently, their result is the same—transport glucose into the cell. Glucose transport follows Michaelis-Menten saturation kinetics, therefore, during times when the metabolism of glucose increases (like during exercise) the rate of GLUT-4 movement to the plasma membrane and the total number of active GLUT-4 present at the membrane surface (translocation)
must be great to continue to transport glucose (Hayashi, Wojtaszewski, & Goodyear, 1997). Glucose gains entry into the cell via glucose transporter carrier proteins. Glucose transporter 4 (GLUT-4), translocation occurs from an intracellular location within muscle and adipose tissue; GLUT-4 is involved in insulin dependent glucose uptake and insulin independent glucose uptake.

**Glycemic Index**

The glycemic index (GI) is a concept that was developed to determine the glycemic responses of individual food stuffs in order to improve the current food tables provided by chemical analysis (Wolever et al., 1991). In theory, a GI number represents the potential of a food on subsequent blood glucose concentrations-how quickly a food stuff moves from the stomach to small intestine to bloodstream-this number may better reflect the physiological response to a better degree than chemical analysis. Knowing the glycemic effects of individual foods may provide further understanding of the physiological effects of whole diets, and allow for maintenance of glycemic responses, thus the glycemic effect of a diet remains constant (Wolever et al., 1991). The GI can be used to assist in improving glycemic control for health implications as in diabetic populations (Jenkins et al., 1981), and more recently has been considered a tool for athletic endeavors (Burke et al., 1998).

Specifically, the GI provides a numerical classification to different sources of CHO, and is calculated as the glycemic response of 50g of available CHO of a test food, expressed as a percentage of the same amount of available CHO of a standard food (Jenkins et al., 2002). Historically, comparisons of test CHO meals are made with 50g of a glucose solution or white bread (Jenkins et al., 2002). The GI is calculated as the area
under the glucose response curve (incremental area under the curve without consideration of relative hypo-glycemic values); the numerical value established to a food indicates the glycemic response in relation to glucose or white bread. As an example, a GI of 80 indicates that this particular food displays 80% the area under the curve that was demonstrated by glucose.

Several factors are involved in ranking foods according to the GI and performing the reference tests. Test food portion size is determined to be 50g available CHO- where availability therefore excludes resistant, or non-digestible CHO. Food with dietary fiber, an example of resistant starch, has been shown to underestimate the GI unless it is accounted for and the overall CHO content adjusted (Wolever et al., 1991). Test foods in a quantity above 50g display a tendency to flatten the glucose response curve, and dosages at 25g have not been shown to display a difference between glucose and bread (Jenkins et al., 1981). Because the dose response curve flattens from 50g to 100g, 50g of available CHO is most commonly accepted as the preferred amount for testing.

Another methodological consideration includes controlling foods based on dry weight, in order to account for water content of cooked foods (Wolever et al., 1991). Furthermore, in choosing the standard food, one must consider that all white bread does not give the same GI value in all situations, this is most likely due to the brand and style of bread- white French bread has been reported at values from 70 to 97 GI (Foster-Powell, Holt, & Brand-Miller, 2002; Wolever et al., 1991). Interestingly, the current international GI tables utilize two GI values, one for glucose and one for white bread as the test foods respectively to accommodate potential discrepancy (Foster-Powell et al., 2002).
The test for the GI of a food is administered after an overnight fast. A test food is selected and 50g of available CHO is ingested by the test subject, during a 10 to 15 minute time span. Jenkins et al. (1981) used the set time of 10 to 15 minutes for ingesting the test meal and has subsequently become the standard time for current GI trials. Immediately following consumption of the test food, finger stick blood samples are collected through 120 minutes (2 hours). In general a sample is taken at 0, 15, 30, 45, 60, 90 and 120 minutes. Extending measurements beyond 2 hours, or the time at which the response returns to baseline reduces the differences in the GI of foods (Wolever et al., 1991). The time frame of 120 minutes is used because it relates to the early portion of the rate of CHO absorption in normal subjects (Wolever et al., 1991). The area under the curve for the 2 hour test period is expressed as a percentage of the same amount of glucose (as discussed previously). However, the measurement does not consider episodes of relative hypoglycemia; that is blood glucose below an initial (0 minute sample). Therefore, the values only consider the positive area under the curve. Many high GI foods display a hypoglycemic response resulting in a portion of the glucose response curve under baseline values, thus displaying a negative value (relative hypoglycemia). Therefore, only the area under the glycemic response curve at and above baseline values is used in determining the GI. Furthermore, foods that are slowly digestible (have low GI values) are likely to continue to maintain blood glucose above fasting for well over 2 hours.

The underlying theory of the GI involves the rate of carbohydrate digestion, the rate of glucose absorption from the small intestine, and the liberation of glucose into the blood stream (Jenkins et al., 2002). A low GI meal will experience prolonged absorption
and thus is usually referred to as a slowly digestible sugar. Some research also has demonstrated that insulinemic response in relation to the glycemic response of variable GI meals. A low GI would result in a slow postprandial rise in insulin and display a reduced area under the glycemic and insulin response curve (Augustin et al., 2002; Jenkins et al., 2002). In a recent review, Jenkins et al. (2002) demonstrated that because of the postprandial increase in insulin a resultant suppression of free fatty acids and lower blood glucose concentrations will be expressed along with the prolonged absorption of carbohydrate (Jenkins et al., 2002).

Several factors are involved in the classification of food according to the glycemic response. Those factors include the method of cooking, protein, fat and fiber quantity, the nature of the starch and the digestion and absorption rates of the test foods (Krezowski, Nuttall, Gannon, & Bartosh, 1986; Thorne, Thompson, & Jenkins, 1983). The glycemic effect of meals is of interest because of varying amounts of fat, and protein content. Recently, it was determined that the effects of protein and fat are negligible because the fat and protein contents of 14 different test meals did not correlate with the mean glucose area under the curve (AUC) (Wolever, Yang, Zeng, Atkinson, & Brand-Miller, 2006). The 14 different test meals varied in GI (35-100), protein (0-17.5g), fat (0-18.2g), and available CHO (15.5-79.4g); however, the CHO content and GI explained ≈90% of the variation in the mean glucose response (Wolever et al., 2006). Additionally, it has been previously established that a greater glucose response occurs after cooking a food, when compared to ingesting that food in a raw state (Collings, Williams, & MacDonald, 1981). For example, corn flakes which are pressurized, rolled and toasted display a greater GI value than that of raw corn (Brand, Nicholson, Thorburn, &
Furthermore, the GI value for various potatoes differed considerably depending on type and cooking method, the potatoes varied from a 56-89 GI value (Fernandes, Velangi, & Wolever, 2005). Other factors include, repeated heating and cooling and the degree of chewing which may aid or interfere with starch digestion in the small intestine (Cummings & Englyst, 1995). The temperature of food effects the GI, cold red potatoes elicited a lower glucose response when compared to hot red potatoes (Fernandes et al., 2005).

The CHO fraction of 50g is the sum of starch and sugars which are available for absorption in the small intestine; this does not include resistant starch (Foster-Powell et al., 2002). Resistant starch is calculated as the starch not hydrolyzed after 120 minutes of incubation; it is generally accepted that resistant starch is hard to measure; additionally, starch digestion is dependent upon the degree of processing (Cummings & Englyst, 1995). The largest percentage of resistant starch is found in raw potato (Cummings & Englyst, 1995). Raw potatoes are not a common food in the Western diet. Physically inaccessible resistant starch is a percentage of the total starch in the majority of starch containing foods; exceptions exist, such as shredded wheat which does not contain resistant starch. A different option to supplement the GI would be utilizing a rapidly available glucose value, which provides an easy to use guide for the amount of glucose available after consumption; the technique and application is discussed elsewhere (Cummings & Englyst, 1995).

Pure CHO is not commonly tested; foods ingested in a common diet are generally tested. For instance the current GI tables are separated into food groups. For example, the groups include bakery products, breakfast cereals, beverages, cookies, cereal grains,
snack foods, pasta and noodles etc., which serves a practical basis for the general public as opposed to a scientific basis (Foster-Powell et al., 2002).

A typical diet consists of variable amounts of fat, protein and carbohydrate percentages of the total daily caloric intake. In theory, the GI is unlikely to be affected with regard to normal dietary portions of fat and protein, because the amount of these macronutrients in normal circumstances is usually smaller than the quantity tested under experimental design (Beaton et al., 1979). Additionally, the GI tests the glycemic response regarding CHO content of foods, and many high CHO foods do not have a large quantity of fat and protein. The difference in fat and protein regarding the GI remains unclear. Whole and skimmed milk displayed similar blood glucose responses; additionally, cottage cheese displayed little effect on the blood glucose response (Jenkins et al., 1981). This indicates that the quantity of protein and fat in foods does not have a clear cut effect on the GI; further research is needed in this area.

Different CHO sources are responsible for a large portion of the variance in degree of postprandial glycemic response. These responses are displayed across a range of CHO sources; therefore, CHO require a further definition away from “simple” and “complex” in order to categorize the postprandial glycemic response. Starches are often classified as complex, while smaller chain monosaccharide’s are classified as simple CHO. One would expect the simple carbohydrates result in a spike in blood glucose response, and for the starches to have a reduced and flattened area under the glucose response curve; however that is not always the case. Potatoes display an increased blood glucose response, while fructose displays a reduced and flattened glucose response. To further complicate the situation, the degree of processing effects the GI; highly processed
foods degrade the amount of fiber, and intact grain, thus increasing the GI (Burke et al., 1998). Additionally, the GI is based on 50g of available CHO, thus 50g may be above the normal quantity consumed in a normal diet. For instance, carrots display an increased blood glucose response; however, 50g of carrots is equal to roughly 8 carrots, a quantity not commonly consumed at one sitting. Using the GI as a system to categorize the blood glucose response to food is a valuable tool, and can be used to replace the terms “simple” and “complex” when describing CHO (Burke et al., 1998).

**GI and health.** In healthy individuals, blood glucose levels peak approximately 1 hour after the start of the meal, and are controlled by a pancreatic release of insulin (Rendell & Jovanovic, 2006). It is suggested that postprandial glucose levels have a greater effect on the vascular system than elevated fasting plasma glucose, because in diabetic individuals insulin secretion may be insufficient to control the postprandial glucose response (Rendell & Jovanovic, 2006). Low GI foods may play an important aspect in controlling and treating postprandial hyperglycemia. Low GI foods do not raise blood glucose levels as great as high GI foods, therefore consuming a diet rich in low GI CHO may lead to less of a blood glucose peak value after a meal. Increased consumption of slowly absorbed CHO produces lower peaks in blood glucose and appears to be an advantage in maintaining glycemic control (Willett, Manson, & Liu, 2002). A reduction in the peak value of blood glucose following a low GI CHO will require less insulin secretion, and may lead to improved control of blood glucose in diabetics, and non-diabetics alike. The suppression of peak blood glucose value may lead to improvements in cardiovascular health and less reliance on exogenous insulin for diabetic patients.
Previous research has supported the importance of a low GI diet and health implications. A low GI diet has been shown to lower blood glucose profiles, reduce insulin secretion and postprandial glycemia, and improve lipid metabolism (Jenkins et al., 1987). A high intake of high GI foods has previously been shown to produce greater insulin resistance, increase postprandial free fatty acids and increase insulin demand comparatively to low GI foods (Willett et al., 2002). A low GI diet may decrease the demand for insulin and produce lower concentrations of blood glucose, thus improving insulin and glucose tolerance, improving cardiovascular health. To lower the risk of cardiovascular disease, including diabetes, the recommendation of ingesting a low GI diet appears to improve overall health. In addition to dietary factors, current fitness level is an important tool for assessing disease risk and the treatment of obesity and diabetes. Current physical activity level has been overlooked as a factor affecting the GI of foods. Previous research has suggested that subjects training state influences the GI of foods (Mettler et al., 2007). Therefore, the glucose response of healthy high fitness and low fitness subjects to different GI foods may offer an additional advantage in glycemic control. Overall, it appears that increasing ingestion of low GI foods and increasing physical activity are important health aspects for improving glucose and insulin tolerance.

Many sample populations have been tested regarding the blood glucose response to CHO containing foods. Some of the various groups tested include, normal healthy subjects, diabetic subjects, children and adults (Jenkins et al., 2002). In general, GI studies classify their subjects according to body mass index (BMI), but this does not allow for consistent interpretation and classification of the subjects. Studies involving a GI pre-exercise meal further classify subjects according to maximal oxygen consumption;
however, not all studies perform a GI trial prior to testing. Current GI tables are formulated according to “average” and “normal” subjects according to BMI. Furthermore, subject characteristics that may affect the blood glucose response include sex, age, body fatness, glucose tolerance and fasting blood glucose. Wolever et al. (1991) speculated with less than 60 subjects if these variables are standardized they do not appear to have a major effect on determining a GI, and thus will result in similar responses (Wolever et al., 1991). When comparing insulin dependent diabetes mellitus (IDDM) subjects to non-insulin dependent diabetes mellitus (NIDDM) subjects, the mean GI value of foods increased by approximately 5 (5%) and increased the variability of glycemic responses (Wolever, Jenkins, Josse, Wong, & Lee, 1987). When the area under the blood glucose response curve is expressed as the GI values, no significant differences between subjects is found, regardless of individual variation, it is therefore speculated that it is valid to apply GI values from one group of test subjects to different individuals displaying various characteristics (Wolever et al., 1991). The reason for this speculation is that there is dependence in the reference to test meal glycemic and insulinemic responses because the comparisons are only made within individuals. It is not logical to compare results of IDDM and NIDDM populations and apply those findings to the general population. This is especially true for individuals of high fitness levels, and on the other end of the spectrum, low fitness levels.

Individuals of high fitness levels and better body composition (greater fat free mass), often have a greater insulin sensitivity because of an increase in total concentration of GLUT-4 (Holloszy, 2005). This may lead to a lower than reported GI value of a test meal when the subjects are categorized into aerobic fitness levels above
and below average values. Therefore, to determine if subject characteristics do interfere with previously tested and reported GI values, those physical characteristics require testing. The current literature displays a general lack of diversity in subject characteristics pertaining to body composition and aerobic fitness levels. Subjects in GI trials are classified as healthy, according to BMI, or diabetic (Wolever et al., 1991, 2006). Body composition and glycemic response is measured when performing an exercise trial testing a pre-exercise, during exercise food; however, these trials do not follow the current GI methodology as strictly as a standard GI trial.

Previously, sedentary, moderately trained and endurance trained subjects’ influence on the GI has been studied (Mettler et al., 2007; Mettler et al., 2006). The present investigation is of importance because it used pure CHO test meals, not mixed meals, used the BOD POD (Life Measurement, Inc., Concord, CA) to determine body composition and not rely on three-site skinfold or body mass index assessment, and used a maximal treadmill test to determine maximal aerobic capacity and not rely on self reporting or cycle ergometry to determine fitness level. Although the same logic applies in higher aerobic fitness individuals, experimental evidence is not available and with the “growing” body of literature to suggest that the GI is a valuable tool for athletic endeavors, the difference in glycemic response between individuals of low and high aerobic fitness requires further investigation.

**GI and high aerobic fitness populations.** Recent research has expanded the applications of the GI to include guidelines for athletic populations regarding pre-exercise meal, during exercise CHO supplementation and post-exercise recovery (Burke et al., 1998). Utilization of the GI may optimize the availability of CHO during exercise.
Prior to exercise a low GI meal is recommended; this may sustain blood glucose levels and promote greater utilization of CHO during the latter stages of exercise. During exercise a moderate to high GI food or drink is recommended to allow for easy digestion and absorption, therefore rapidly supplying energy (Burke et al., 1998). Post exercise a high GI food or drink is recommended for recovery of glycogen stores, the rapid supply of glucose from a high GI food may enhance post exercise refueling (Burke et al., 1998).

Low GI meals consumed before exercise may offer the benefit of maintaining glucose and prolonging endurance by maintaining blood glucose levels and sparing muscle glycogen (Wee, Williams, Tsintzas, & Boobis, 2005). Thus, a lower insulin response prior to exercise may result in increased fat oxidation during the subsequent bout of exercise, maintenance of blood glucose, and reduced or delayed onset of muscle glycogen usage (Wee et al., 2005). The GI does not consider gender, percentage of lean muscle mass and body fat percentage, age, and level of physical activity when classifying blood glucose response according to the GI. Therefore, the importance to GI to performance lies in the blood glucose response of physically fit populations. This may lead to further recommendations that may differ from non-active and diabetic populations and provide a non-invasive technique to determine the importance and abundance of GLUT-4 within athletic populations.

A nutritional plan for athletic populations should provide several goals, assure adequate CHO stores before exercise, provide an exogenous CHO source during exercise of long duration, and recover glycogen stores following post-exercise recovery (Burke et al., 1998). Current recommendations are for the ingestion of a pre-exercise meal 3 to 4 hours prior the competition; additionally, CHO should be ingested during prolonged
exercise lasting greater than 90 minutes or of increased intensity lasting 60-90 minutes. However, during many sporting activities, exogenous CHO sources are not available, and many events begin early in the morning, which may not be desirable to awake 3 to 4 hours before the event. Liver glycogen stores may be depleted after an overnight fast; therefore, a pre-exercise meal may optimize the liver and muscle glycogen stores in preparation for the following exercise session (Burke et al., 1998). Practical application of the GI to sports nutrition is desirable, with the goal of maintaining blood glucose throughout exercise, when the above events do not allow for adequate CHO ingestion. It is desirable during prolonged moderate intensity exercise to maintain CHO availability; therefore the following recommendations have been proposed (Coyle, 1991). A low GI meal should be consumed before exercise to promote maintenance of blood glucose and CHO availability; a moderate to high GI food or drink should be consumed during prolonged exercise, and high GI foods should be consumed during recovery to enhance glycogen storage (Coyle, 1991).

Prior to exercise (running, cycling), GI meals have been ingested at various time intervals, ranging from 30 minutes prior up to four hours, in an attempt to determine an advantageous time interval to optimize CHO utilization during exercise. Forty-five minutes prior to exercise, a low and high GI meal was consumed followed by cycling at 70% VO$_{2\text{max}}$ and then performing a 15 minute maximal work accomplished trial; the high GI food resulted in a hyperglycemic response, elevated insulin prior to exercise and reduced free fatty acid oxidation and plasma glucose during exercise (Sparks, Selig, & Febbraio, 1998). Similarly, when high and low GI meals were consumed 65 minutes prior to cycling to exhaustion plasma glucose levels were decreased compared to a low
GI meal during exercise (Stannard, Constantini, & Miller, 2000). Additionally, when low and high GI meals were consumed 60 minutes prior to cycling to exhaustion; the low GI meal improved endurance time by maintaining higher levels of free fatty acid and glucose during the exercise trial (Thomas, Brotherhood, & Brand, 1991). When ingesting a pre-exercise meal within 65 minutes prior to exercise, a hyperglycemic and hyperinsulinemic response is expected following the ingestion of a high GI meal. These responses result in suppressed fatty acid oxidation, and increased plasma glucose oxidation resulting in increased CHO oxidation and possible decreased performance times. When ingesting a low GI meal within 65 minutes prior to exercise, blood glucose levels are maintained and fat oxidation increases in respect to high glycemic index meals.

Meals ingested 3 hours prior to exercise have found similar results. Fat oxidation is shown to increase and CHO oxidation decreased while maintaining plasma glucose levels during 60 minutes of sub maximal treadmill running after ingestion of a low GI meal, in comparison to a high GI meal (Wu et al., 2003). The response of maintaining blood glucose after 60 minutes of running provides endurance performance benefits for prolonged training, exercise and competition, such as a marathon. After ingesting a low GI meal three hours prior running to exhaustion at 70% VO2max, the low GI index meal resulted in maintained glucose throughout exercise and increased fat oxidation in comparison to a high GI meal (Wee, Williams, Gray, & Horabin, 1999). These responses may be explained by the decreased area under the response time curve for glucose and insulin seen in the low GI meal (Wee et al., 1999). The increased insulin response in the high GI meal leads to suppression of fatty acids during exercise and increased rates of CHO oxidation. Running at 71% VO2max for 30 minutes after ingesting a low GI meal 3
hours prior resulted in reduced muscle glycogen utilization and increased fat oxidation during exercise (Wee et al., 2005). Ingesting a high GI meal resulted in increased muscle glycogen storage at rest and utilization during exercise. Blood glucose levels were maintained during 30 minutes of running in the low GI trial (Wee et al., 2005). Ingesting a low GI meal conserves muscle glycogen utilization, maintains blood glucose and increases fat oxidation during exercise, and therefore may be beneficial to endurance exercise and sporting events. Further health aspects include improving glycemic control, which may be beneficial for reducing oxidative damage (depression of serum antioxidants), and preserving high density lipo-protein (HDL) cholesterol (Jenkins et al., 2002). Additional areas of research and health include implications for obesity; the current assumption is that consuming a low GI meal early in the day may lead to less food consumption throughout the remainder of the day and the subsequent meals (Jenkins et al., 2002). The benefits of low GI meals coming to the forefront in the Western diet, further research is required to associate the GI with health benefits, expanding to topics including coronary heart disease (CHD), cancer, obesity, and diabetes. By investigating the difference in fitness level and glycemic response, further evidence may support, high fitness levels as a means for improving glycemic control, and glucose and insulin tolerance. Furthermore, if evidence suggests that a high fitness level results in a different glycemic response than that of a low fitness level for a specific GI food, the result would challenge current GI values. Physically active individuals may experience an attenuated metabolic response to high and low GI foods because of increased insulin sensitivity compared to low fitness/sedentary individuals (Roberts, 2003). This may lead to a
recommendation of the importance of increased physical activity, and not just diet as an important intervention factor for glycemic control.
CHAPTER 3: METHODS

Subjects

Participants in the study volunteered from Ohio University and the surrounding community. The research and all of the following methodology were approved by the Ohio University Institutional Research Board prior to data collection. The participants recruited were college age male students, 20-29 years of age, healthy, non-smoking, and had no recent illness, diagnosed disease, or taking any medication or dietary supplements known to affect metabolism. Potential students were initially screened in order to determine health history, current physical activity level, fasting blood glucose- to ensure normoglycemia, body composition and aerobic capacity analyses. A total of 33 potential students were recruited and underwent part or all of the initial screening. Of these potential students, 12 fulfilled the inclusion criteria and were placed into one of two groups. Six high fitness (VO$_{2\text{max}}$ between 63.0 - 52.3 mL/kg/min, $n = 6$) and six low fitness (VO$_{2\text{max}}$ between 41.0 – 42.9 mL/kg/min, $n = 6$) students completed this investigation. To be included in the study, the students’ VO$_{2\text{max}}$ were $\geq$70$^{th}$ percentile (49.0 ml/kg/min; for ages 20 to 29 years), classified as high aerobic fitness or below $\leq$50$^{th}$ percentile (44.2 ml/kg/min; for ages 20 to 29 years), classified as low aerobic fitness, according to the American College of Sports Medicine (ACSM) (Whaley et al., 2006). Since accepted norms for body composition do not exist, a range of 12.0 and 32.0% body fat was chosen and considered satisfactory for health (Whaley et al., 2006). Twenty-one students were recruited failed to meet the inclusion criteria. Five potential participants had VO$_{2\text{max}}$ values determined between 47.0 – 48.3 mL/kg/min; three potential participants had body composition values below the minimum12.0%; three potential
participants developed an undisclosed illness requiring medication during the course of participation and dropped out; and three potential participants did not have sufficient personal time after completing the initial screening.

Participants were tested for fasting blood glucose determined on two separate test mornings within 1 week, after a 12 hour overnight fast. Whole capillary blood was sampled before the trials began to ensure the subjects met criteria of normoglycemia (blood glucose value ≤ 100 mg/dL) (American Diabetes Association, 2007a; American Diabetes Association, 2007b). This method for fasting blood glucose value was previously used in glycemic index methodology (Jenkins et al., 1981; Wolever et al., 1991). The participants reported to the laboratory in the morning, finger capillary whole blood was collected by “finger stick”; approximately 30 microliters of blood was collected using heparinized Natelson collecting tubes (Fisherbrand Natelson ammonium heparin micro blood collecting tubes, ThermoFisher Scientific, Pittsburgh, PA). The blood was then tapped into a 0.5ml microcentrifuge tube (Fisherbrand General Purpose Microcentrifuge Tubes, ThermoFisher Scientific, Pittsburgh, PA) from which the sample was immediately analyzed for fasting blood glucose using an automated analyzer (YSI 2300 STATPLUS, Yellow Springs Instruments Inc., Yellow Springs, OH). The analyzer was calibrated according to manufacturer’s specifications, and reference standards (YSI D-glucose 500mg/dl, 900mg/dl, Yellow Springs Instruments Inc., Yellow Springs, OH; Pointe Scientific Glucose Standard 100mg/dl, Pointe Scientific Inc., Canton, MI) and unknown controls were tested daily (Sugar-Chex® II Aqueous Glucose Control, Streck Laboratories, Omaha, NE), before, during and after testing to verify the accuracy of the analyzer.
Prior to testing students submitted a 1 day food log that was analyzed for total calorie, carbohydrate, protein and fat consumption using Nutritionist Pro software (Nutritionist Pro Nutrition Analysis Software version 1.3 2003, Axxya Systems, Stafford, TX). The diet log was immediately returned to the students and the students were instructed to consume a similar diet prior to each test day to ensure a standardized protocol.

**Maximal Oxygen Consumption and Body Composition**

On a separate occasion, after at least 1 day of normoglycemia was determined, the students performed a graded exercise test on a treadmill ergometer to determine their maximal aerobic capacity (maximal oxygen consumption; VO$_{2\text{max}}$). In order to be considered a “true” VO$_{2\text{max}}$ the data obtained was required to meet the following criteria: (a) attainment of predicted max HR (plus or minus 10 beats per minute), (b) oxygen consumption leveling off or declining (defined as an increase of less than 150mL/min with increasing workload), and (c) RER greater than 1.1 (Howley, Bassett, & Welch, 1995). The information gained from this test was used to classify students according to levels of aerobic fitness.

For the test, students were fitted with a Hans Rudolph two-way non-rebreathing valve held in place using head gear while expired air was continuously analyzed for oxygen and carbon dioxide concentrations using a metabolic cart (TrueOne 2400 Metabolic Measurement System, ParvoMedics, Sandy, UT). The metabolic cart was calibrated according to the manufacturers’ specifications prior to each test. Before the subjects began exercise, they were made aware of the risks of maximal exercise, and read a paragraph discussing Borg’s rating of perceived exertion (RPE) (Robertson & Noble,
The Bruce Protocol was utilized as the maximal treadmill test. The Bruce protocol is a stage exercise test, each stage lasted 3 minutes. The first stage began at 1.7 miles per hour (mph) and 10% grade and increased as follows: stage two 2.5 mph and 12%, stage three 3.4 mph and 14%, stage four 4.2 mph and 16%, stage five 5.0 mph and 18%, and stage six 5.5 mph and 20%, respectively. Once the student reached volitional fatigue, and the previously mentioned criteria were met, the test was terminated; therefore, not all stages were utilized during every test. Throughout the test, students were continuously monitored for oxygen consumption, RER, and Borg’s ratings of perceived exertion based on a 6-20 point scale (Robertson & Noble, 1997). Heart rate was continuously monitored using a Polar heart rate monitor (Polar FS1 Heart Rate Monitor, Polar Electro Inc., Lake Success, NY). Students were strongly encouraged to continue the test to the point of volitional fatigue, and until the point at which three of the aforementioned criteria were met. If the students did not meet all three criteria the test was not considered a maximal exercise test. All students tested met three of the criteria, no students required retesting.

On a separate occasion, participants were analyzed for body composition using a BOD POD (Life Measurement, Inc., Concord, CA). To qualify, the potential participants had to have a body composition between 12.0% and 32.0% body fat, accepted norms for body composition does not exist, a range of 12.0 and 32.0% body fat was chosen and considered satisfactory for health (Whaley et al., 2006). Initially, the criteria was set at a range of 12% to 25% to eliminate group differences in the mean body fat, however, due to the inability to recruit potential students within that range, the criteria was modified. Four of the low fitness students had reported body composition values at 29%, 31.5%,
26.4%, and 29.2% body fat, respectively. These students would have been excluded from the investigation if not for the modification to the body composition ranges; therefore, it was necessary to increase potential student participation in the investigation. Subjects reported for body composition testing after abstaining from food, drink for a minimum of 3 hours, and no previous exercise on the day of testing. Subjects voided, removed all jewelry, and dressed into the required minimal tight fitting clothing. Subjects were given the proper instruction on performing a test according to Life Measurement, Inc. Body Composition Tracking System Manual. Prior to each test the BOD POD was calibrated using a two point calibration test, the first test measured the BOD POD empty and the second test measured the chamber with a calibrated cylinder. The appropriate body density equation was selected according to the ethnicity and age of the subject, as selected from the included LMI software. Lung volume was measured from each subject for the required lung volume correction. The BOD POD is a dual-chamber plethysmograph that allows the determination of body composition by measuring body mass and volume in order to determine body density. Body density was then used to determine and calculate the relative proportions of fat and fat free mass, body fat was then calculated using the Siri equation (Siri, 1993). The accuracy and reliability of air plethysmography was previously reported; body volume measurement with an overall mean percentage error between measured and actual body volume of <0.1% (Dempster & Aitkens, 1995; McCrory, Gomez, Bernauer, & Mole, 1995).

**Glycemic Index Trials**

Subjects reported to the exercise physiology lab at a set time in the morning after a 12 hour fast, and having abstained from exercise (or physical activity above what was
considered their “typical daily schedule”) and alcohol consumption for the 36 hours prior to each trial. Three glucose challenges were utilized in all 12 subjects and included a standard, 50g of dextrose monohydrate, and two novel CHO meals consisting of modified slow and fast digestible CHO. Subjects were randomly provided one of three test meals consisting of 50g of available CHO with 500ml of water. One meal was the reference trial consisting of standard glucose as dextrose monohydrate (ADM Clintose® Dextrose, Archer Daniels Midland Co., Decatur, IL).

The novel CHO meals were prepared by Purdue University’s Whistler Center for Carbohydrate Research by way of a patent pending process, and then cooked according to their instructions prior to each subject consuming the meal. The fast and slow digestible CHO were tested, and reported to have GI values of 76.44 (16.02) and 48.96 (17.30), respectively, in a cross-section of subjects where no regards were given to their aerobic fitness levels or body composition. Briefly, biopolymer-entrapped CHO microspheres were prepared by dropping (21 G1½ hypodermic needle) and atomizing (Spray Systems Co., Wheaten, IL) a homogenous mixture of biopolymer blends and waxy corn starch (Tate & Lyle, Decatur, IL into a CaCl₂ solution (20 g/L). Following incubation (3 hours), the spheres were filtered, washed with distilled water and dried (12 hours, 45 °C). Total starch content of the microspheres, after powdering using a ball mill, had previously been determined using Megazyme Total Starch Kit (Megazyme International Ireland Ltd, Wicklow, Ireland), where the efficiency of preparation of the CHO was calculated from the actual amount of starch entrapped in the dry microspheres and the total amount of starch used for the preparation. Furthermore, in vitro starch digestibility of the cooked microspheres has been evaluated using the Englyst method (Englyst, Englyst, Hudson,
Cole, & Cummings et al., 1999) and determined a relatively small amount of resistant starch (<5%).

Instructions for cooking, place the dry volume 50g of novel CHO was placed in a glass jar, 500ml of water was added; the jar was sealed and placed in a pressure canner/cooker. The food was cooked for 15 minutes at 10psi, at this point is was removed from the heat source and allowed to cool to room temperature. The CHO was then placed in a refrigerator. Each fast and slow CHO was prepared the night before the trial. Prior to consuming the food, a non-caloric flavoring packet (Sugar Free Orange Early Rise, Great Value, Wal-Mart, Bentonville, AR) was added to the CHO, the CHO was placed in a drinking cup and the subjects were then instructed to drink the CHO and not to chew the microsphere CHO. A spoon was provided. No gastric distress was reported following the ingestion of the fast and slow CHO.

Each trial lasted 2 hours while the subjects were instructed to remain seated and not consume further food or drink throughout the trial, other than what was provided. The subjects were instructed to consume the food within 10 to 15 minutes, the standard time used by Jenkins et al. (1981). Capillary blood samples were obtained at 0, 15, 30, 45, 60, 90, and 120 minutes and collected using heparinized Natelson collecting tubes (Fisherbrand Natelson ammonium heparin micro blood collecting tubes, ThermoFisher Scientific, Pittsburgh, PA). The blood was then tapped into a 0.5ml microcentrifuge tube (Fisherbrand General Purpose Microcentrifuge Tubes, ThermoFisher Scientific, Pittsburgh, PA) from which the sample was immediately analyzed for fasting blood glucose using an automated analyzer (YSI 2300 STATPLUS, Yellow Springs Instruments, Yellow Springs, OH).
The “0” time point served as the baseline resting sample before the food was ingested and was collected with 5 minutes prior to initiation of the meal consumption. A total of seven capillary blood samples were taken and approximately 30μl of blood per sample. If necessary, the hands of the subjects were warmed using electric heating pads or lamps to ensure adequate blood flow. The finger was cleaned with an alcohol swab (Triad Sterile Medium Alcohol Prep Pads Isopropyl Alcohol 70% v/v, Triad Disposables Inc., Brookfield, WI ), allowed to air dry and the skin was then be pricked using a lancet (Unilet – Single Use Lancets 21G, Owen Mumford, Marietta, GA). The first “spot” of blood was cleaned away from the skin using a cotton swab and the subsequent blood was drawn into a heparinized Natelson collecting tube (Fisherbrand Natelson ammonium heparin micro blood collecting tubes, ThermoFisher Scientific, Pittsburgh, PA). The blood was then placed in a 0.5ml microcentrifuge tube (Fisherbrand General Purpose Microcentrifuge Tubes, ThermoFisher Scientific, Pittsburgh, PA) and presented to the analyzer for immediately determination of blood glucose (YSI 2300 STATPLUS, Yellow Springs Instruments, Yellow Springs, OH). The samples were analyzed in duplicate. The GI procedures are the standards proposed by Jenkins and colleagues (Jenkins et al., 1981).

The results for blood glucose were reported as mg/dl and utilized to determine the area under the glucose response curve. The area under the curve (AUC) was calculated using the incremental area under the curve method described by Wolever et al. (1991) by a JAVA™ platform (Sun Microsystems) software program developed by Dr. Genyi Zhang at Purdue University in 2006.
The AUC was used to determine the GI for the slow digestible CHO and fast digestible CHO in the equation, \( \text{GI} = \left[ \frac{(\text{AUC test food})}{(\text{AUC glucose})} \right] \times 100 \). The maximal blood glucose value was the maximal value recorded during the glycemic trials. The percent increase from baseline to maximal value was calculated as, percent increase \( = \left[ \frac{(\text{maximal value} - \text{baseline value})}{(\text{baseline value})} \right] \times 100 \), calculated by hand.

**Statistical Analysis**

The subject characteristics were compared by an independent \( t \)-test, and reported as mean (\( M \)) plus or minus standard deviations (\( SD \)). Normoglycemic data was compared using a 2 (group) \( \times \) 2 (day) repeated measures ANOVA. The data from the glycemic trials (GI, AUC, maximal value, percent increase, baseline blood glucose, time to peak) were analyzed using a 2 (group) \( \times \) 3 (trial) repeated measures ANOVA, the blood glucose AUC data were analyzed for each fitness level group (high fitness and low fitness) and for each test food (glucose standard, moderate GI slow digesting and low GI slow digesting) and used to determine the GI. *Tukey’s HSD* was used to determine the location of the significant differences, when applicable. *HSD* calculated by hand as described in *Statistics in Kinesiology* (Vincent, 2005). Pearson product-moment correlation (\( r \)) was used to establish relationships between fitness level and glycemic response, the coefficient of determination (\( r^2 \)) was calculated by hand. For statistical analysis SPSS 15.0 (SPSS Inc., Chicago, IL) was used. An alpha of 0.05 will be utilized to determine significant differences.
CHAPTER 4: RESULTS

Twelve men \( (N=12) \) completed this investigation with six men in the high fitness group \( (n = 6) \) and six in the low fitness group \( (n = 6) \). The high and low fitness groups were analyzed using an independent samples \( t \)-test, characteristics are presented as mean ± standard deviation \( (M \pm SD) \) and ranges (see Table 1). A \( t \)-test was utilized to compare age, body fat, the average absolute and relative VO\( _2 \)max scores, weight and height. The relative and absolute values for VO\( _2 \)max were significantly different between the groups \( (p = .0001 \) and \( p = .014, \) respectively) The relative and average VO2max scores were 3.55 ± 0.59 L/min and 42.05 ± 0.81 ml/kg/min for the low fitness group, and 4.40 ± 0.37 L/min and 56.9 ± 4.47 ml/kg/min for the high fitness group, respectively. The average percent body fat was significantly different \( (p = .002) \) at 25.99 ± 5.63% for the low fitness group and 16.15 ± 1.93% for the high fitness group. However, there was no difference between the groups regarding age, weight, and height (see Table 1).
A 2x2 (group x day) ANOVA was performed on fasting blood glucose values.

There was not a significant interaction ($p = .079$), nor were there significant main effects for day ($p = .184$) and group ($p = .079$). Furthermore, all subjects ($N = 12$) were classified as normoglycemic (fasting blood glucose $\leq 100.00$ mg/dl) using fasted whole capillary blood according to the American Diabetes Association on two separate mornings (American Diabetes Association, 2007a; American Diabetes Association, 2007b)(see Table 2).
Table 2

*Normoglycemia*

<table>
<thead>
<tr>
<th></th>
<th>Low Fitness ($n = 6$)</th>
<th>High Fitness ($n = 6$)</th>
<th>Total ($N = 12$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>$SD$</td>
<td>Mean</td>
</tr>
<tr>
<td>Day One (mg/dL)</td>
<td>83.59</td>
<td>4.14</td>
<td>78.28</td>
</tr>
<tr>
<td>(Range)</td>
<td>(78.90 – 90.40)</td>
<td>(74.40 – 82.10)</td>
<td>(74.40 – 90.40)</td>
</tr>
<tr>
<td>Day Two (mg/dL)</td>
<td>83.13</td>
<td>6.46</td>
<td>81.21</td>
</tr>
<tr>
<td>(Range)</td>
<td>(74.85 – 93.15)</td>
<td>(74.35 – 84.05)</td>
<td>(74.35 – 93.15)</td>
</tr>
</tbody>
</table>

*p < .05

In order to compare the dietary analysis completed on 1 day’s diet prior to beginning the trials for total calories, total grams carbohydrate, total grams protein, total grams fat, total percentage carbohydrate, total percentage protein and total percentage fat an independent samples $t$-test was performed for each variable. The subjects were asked to follow a similar diet prior to each trial. This data is presented in Table 3 and indicates no significant differences ($p > .05$) between the groups for the characteristics of the diets.
### Subject Diet Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Low Fitness ((n = 6))</th>
<th>High Fitness ((n = 6))</th>
<th>Total ((N = 12))</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total kcal (g)</strong></td>
<td>2068.76</td>
<td>2131.99</td>
<td>2100.37</td>
</tr>
<tr>
<td>(Range)</td>
<td>(870.18 – 3227.42)</td>
<td>(1467.03 – 2655.00)</td>
<td>(870.18 – 3227.42)</td>
</tr>
<tr>
<td><strong>CHO (g)</strong></td>
<td>255.88</td>
<td>296.21</td>
<td>276.05</td>
</tr>
<tr>
<td>(Range)</td>
<td>(91.05 – 393.60)</td>
<td>(196.86 – 409.30)</td>
<td>(91.05 – 409.30)</td>
</tr>
<tr>
<td><strong>Protein (g)</strong></td>
<td>119.58</td>
<td>99.04</td>
<td>109.31</td>
</tr>
<tr>
<td>(Range)</td>
<td>(49.40 – 225.75)</td>
<td>(71.38 – 144.38)</td>
<td>(49.40 – 225.75)</td>
</tr>
<tr>
<td><strong>Fat (g)</strong></td>
<td>67.87</td>
<td>67.22</td>
<td>67.54</td>
</tr>
<tr>
<td>(Range)</td>
<td>(35.83 – 125.12)</td>
<td>(64.88 – 76.46)</td>
<td>(35.83 – 125.12)</td>
</tr>
<tr>
<td><strong>CHO (%)</strong></td>
<td>48.06</td>
<td>53.82</td>
<td>50.94</td>
</tr>
<tr>
<td>(Range)</td>
<td>(32.60 – 71.80)</td>
<td>(48.70 – 59.10)</td>
<td>(32.60 – 71.80)</td>
</tr>
<tr>
<td><strong>Protein (%)</strong></td>
<td>22.70</td>
<td>18.28</td>
<td>20.49</td>
</tr>
<tr>
<td>(Range)</td>
<td>(11.60 – 37.00)</td>
<td>(12.60 – 23.10)</td>
<td>(11.60 – 37.00)</td>
</tr>
<tr>
<td><strong>Fat (%)</strong></td>
<td>29.26</td>
<td>27.92</td>
<td>28.59</td>
</tr>
</tbody>
</table>

\*\(p < .05\)

A 2x3 (groups x trial) ANOVA was performed to determine differences in the time to ingest a test meal. The three trials were randomized; the time to ingest the test foods for each trial was determined to be similar among trials and between groups (see Table 4).
Table 4

Time to Ingest Meal

<table>
<thead>
<tr>
<th></th>
<th>Low Fitness (n = 6)</th>
<th>High Fitness (n = 6)</th>
<th>Total (N = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Glucose (min)</td>
<td>4.71</td>
<td>2.40</td>
<td>2.50</td>
</tr>
<tr>
<td>(Range)</td>
<td>(2.00 – 8.00)</td>
<td>(1.00 – 4.00)</td>
<td>(1.00 – 8.00)</td>
</tr>
<tr>
<td>Fast CHO (min)</td>
<td>9.75</td>
<td>5.29</td>
<td>6.36</td>
</tr>
<tr>
<td>(Range)</td>
<td>(5.00 – 20.00)</td>
<td>(2.15 – 11.00)</td>
<td>(2.15 – 20.00)</td>
</tr>
<tr>
<td>Slow CHO (min)</td>
<td>10.00</td>
<td>4.29</td>
<td>9.17</td>
</tr>
<tr>
<td>(Range)</td>
<td>(5.00 – 15.00)</td>
<td>(4.75 – 12.00)</td>
<td>(4.75 – 15.00)</td>
</tr>
</tbody>
</table>

*p < .05.

A 2x3 (groups x trial) ANOVA was performed to determine differences in the GI of the fast and slow digestible CHO. The group by trial interaction was statistically different, \( F(2, 3.72) = 417.70, p = .042 \). The model was then broken down into two simple ANOVAs comparing fast digestible CHO between low and high fitness and comparing slow digestible CHO between low and high fitness. Fast digestible CHO differed between low and high fitness \((86.71±16.15 \text{ and } 66.17±7.12, p = .017)\) and slow digestible CHO differed between low and high fitness \((59.13±8.50 \text{ and } 38.79±18.38, p = .034)\). Figure 1 illustrates the average GI values for the fast digestible and slow digestible CHO.
Figure 1. Glycemic index value. Depicted for fast digestible CHO, and slow digestible CHO. Mean value for glycemic index value, vertical lines depict standard deviations of the means for high fitness \((n = 6)\), low fitness \((n = 6)\) and total \((N = 12)\). *Significant difference between subject groups, \(p < .05\). Low fitness > High fitness.

A 2x3 (groups x trial) ANOVA was performed to determine differences in the glycemic response (reported as AUC). The mean glycemic responses- reported as the change in blood glucose resulting from food ingestion are reported in Table 5. The group by trial interaction was not statistically significant, \(F(2, 0.543) = 315189.28, p = .594\). The main effect of the trials (glycemic response of glucose, fast and slow digestible CHO) was statistically significant, \(F(2, 26.61) = 15695638.77, p = .0001\). Tukey’s post hoc on the main effects of trial found significant differences between the means of the test meals for glucose \((4318.24 \pm 1911.64)\) and the fast digestible CHO \((3328.45 \pm \)
1940.71), between glucose (4318.24 ± 1911.64) and the slow digestible CHO (2037.52 ± 1129.60) and between the fast digestible CHO (3328.45 ± 1940.71) and the slow digestible CHO (2037.52 ± 1129.60). Figure 2 illustrates the glycemic response as changes in blood glucose over time by trial and group.

Table 5

*Glycemic Index and Glycemic Response*

<table>
<thead>
<tr>
<th></th>
<th>Low Fitness (n = 6)</th>
<th>High Fitness (n = 6)</th>
<th>Total (N = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Glycemic Index</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fast CHO</td>
<td>86.71*</td>
<td>16.15</td>
<td>66.17*</td>
</tr>
<tr>
<td>(Range)</td>
<td>(63.80 – 107.28)</td>
<td></td>
<td>(59.08 – 73.39)</td>
</tr>
<tr>
<td>Slow CHO</td>
<td>59.13*</td>
<td>8.50</td>
<td>38.79*</td>
</tr>
<tr>
<td>Glycemic Response (AUC)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fast CHO</td>
<td>4106.03</td>
<td>2547.55</td>
<td>2550.87</td>
</tr>
<tr>
<td>(Range)</td>
<td>(1814.06 – 8835.00)</td>
<td></td>
<td>(1725.13 – 3235.37)</td>
</tr>
<tr>
<td>Slow CHO</td>
<td>2698.90</td>
<td>1206.78</td>
<td>1376.15</td>
</tr>
<tr>
<td>(Range)</td>
<td>(1112.92 – 3993.58)</td>
<td></td>
<td>(849.59 – 2085.75)</td>
</tr>
<tr>
<td>Glucose</td>
<td>4775.67</td>
<td>2641.75</td>
<td>3860.81</td>
</tr>
<tr>
<td>(Range)</td>
<td>(1691.00 – 9307.88)</td>
<td></td>
<td>(2865.39 – 4591.89)</td>
</tr>
</tbody>
</table>

*Note. AUC = area under curve, mg/dL/min.*

*Significant difference between subject groups, p < .05. Low Fitness > High Fitness.

*Significant difference among trials, p < .05. *Glucose > Fast CHO, Glucose > Slow CHO; b Fast CHO > Slow CHO, Fast CHO < Glucose; c Slow CHO < Fast CHO, Slow CHO < Glucose.
Figure 2. Glycemic response area under curve, (A) glucose, (B) fast digestible CHO and (C) slow digestible CHO, depicted for high fitness \((n = 6)\), low fitness \((n = 6)\) and total \((N = 12)\).
A 2x3 (groups x trial) ANOVA was performed to assess baseline blood glucose (see Table 6). There was not a significant interaction ($p = .467$), nor were there significant main effects for trial ($p = .326$) and group ($p = .467$), which indicate remarkably consistent measurements.

Table 6

*Baseline Blood Glucose*

<table>
<thead>
<tr>
<th></th>
<th>Low Fitness ($n = 6$)</th>
<th>High Fitness ($n = 6$)</th>
<th>Total ($N = 12$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Glucose</strong></td>
<td>Mean: 83.85, SD: 7.50</td>
<td>Mean: 80.12, SD: 2.28</td>
<td>Mean: 81.98, SD: 5.63</td>
</tr>
<tr>
<td>(Range)</td>
<td>(71.00 – 93.60)</td>
<td>(77.35 – 84.05)</td>
<td>(71.00 – 93.60)</td>
</tr>
<tr>
<td><strong>Fast CHO</strong></td>
<td>Mean: 82.02, SD: 6.31</td>
<td>Mean: 78.80, SD: 5.00</td>
<td>Mean: 80.44, SD: 5.69</td>
</tr>
<tr>
<td>(Range)</td>
<td>(74.85 – 92.90)</td>
<td>(71.90 – 85.50)</td>
<td>(71.90 – 92.90)</td>
</tr>
<tr>
<td><strong>Slow CHO</strong></td>
<td>Mean: 82.78, SD: 4.64</td>
<td>Mean: 82.37, SD: 4.16</td>
<td>Mean: 82.58, SD: 4.21</td>
</tr>
<tr>
<td>(Range)</td>
<td>(74.90 – 86.75)</td>
<td>(79.40 – 90.15)</td>
<td>(74.90 – 90.15)</td>
</tr>
</tbody>
</table>

*($p < .05$).

A 2x3 (group x trial) ANOVA was performed for glycemic response via maximal blood glucose. The interaction for this variable was not significant ($p = .594$). The main effect of glycemic response, as illustrated by the maximal blood glucose, within a trial (main effect for trial) in Table 7 was statistically significant, $F (2, 52.79) = 5506.47, p = .0001$. Post hoc analyses (Tukey’s HSD) revealed the slow digestible CHO, fast digestible CHO and glucose were all significantly different from each other ($p < .05$).
The fast digestible CHO was 135.33 mg/dl (22.26), the slow digestible CHO was 114.16 mg/dl (13.88), and glucose solution was 157.00 mg/dl (20.62) (see Table 7, Figure 3).

Table 7

Maximal Blood Glucose Value and Percent Increase

<table>
<thead>
<tr>
<th></th>
<th>Low Fitness (n = 6)</th>
<th>High Fitness (n = 6)</th>
<th>Total (N = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal Blood Glucose Value</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Glucose (Range)</td>
<td>162.33</td>
<td>28.35</td>
<td>151.67</td>
</tr>
<tr>
<td>Fast CHO (Range)</td>
<td>146.83</td>
<td>23.82</td>
<td>123.83</td>
</tr>
<tr>
<td>Slow CHO (Range)</td>
<td>119.83</td>
<td>15.43</td>
<td>108.48</td>
</tr>
<tr>
<td>Percent Increase</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Glucose (Range)</td>
<td>93.82</td>
<td>30.68</td>
<td>89.84</td>
</tr>
<tr>
<td>Fast CHO (Range)</td>
<td>78.66</td>
<td>24.67</td>
<td>57.02</td>
</tr>
<tr>
<td>Slow CHO (Range)</td>
<td>44.57</td>
<td>14.30</td>
<td>31.64</td>
</tr>
</tbody>
</table>

Note. Maximal blood glucose value (mg/dL/min), Percent increase (%).

Significant difference among trials, p < .05. a Glucose > Fast CHO, Glucose > Slow CHO; b Fast CHO > Slow CHO, Fast CHO < Glucose; c Slow CHO < Fast CHO, Slow CHO < Glucose.
Figure 3. Maximal blood glucose value. Responses to fast digestible CHO, slow digestible CHO and glucose. Mean value for percent increase, vertical lines depict standard deviations of the means for high fitness \((n = 6)\), low fitness \((n = 6)\) and total \((N = 12)\). \(^{a,b,c}\) Significant difference among trials, \(p < .05\). \(^{a}\) Glucose > Fast CHO, Glucose > Slow CHO; \(^{b}\) Fast CHO > Slow CHO, Fast CHO < Glucose; \(^{c}\) Slow CHO < Fast CHO, Slow CHO < Glucose.

The percent increase in blood glucose over the trials (from baseline to maximal value) was determined as: percent increase = \([(\text{maximal value} - \text{baseline value}) / \text{baseline value}] \times 100\), and are reported in Table 7 and illustrated in Figure 4. A 2x3 (group x trial) ANOVA was performed for percent increase in blood glucose. The interaction for this variable was not significant \((p = .307)\). The main effect of percent increase within a trial (main effect for trial) in Table 7 was statistically significant, \(F(2, \text{total participants}) = 8.34, p < .001\).
48.26) = 8635.51, \( p = .0001 \). Post hoc analyses (Tukey’s HSD) revealed significant differences among trials, the slow digestible CHO, fast digestible CHO and glucose were all significantly different from each other (\( p < .05 \)). The fast digestible CHO was 67.84\% (22.00), the slow digestible CHO was 38.11\% (13.59), and the glucose solution was 91.65\% (22.34), (all values were significantly different (\( p < .05 \)).

![Figure 4](image-url)

**Figure 4.** Percentage increase. Responses to fast digestible CHO, slow digestible CHO and glucose. Mean value for percent increase, vertical lines depict standard deviations of the means for high fitness (\( n = 6 \)), low fitness (\( n = 6 \)) and total (\( N = 12 \)). \( a, b, c \) Significant difference among trials, \( p < .05 \). \( a \) Glucose > Fast CHO, Glucose > Slow CHO; \( b \) Fast CHO > Slow CHO, Fast CHO < Glucose; \( c \) Slow CHO < Fast CHO, Slow CHO < Glucose.

The glycemic response is also represented as the time to peak blood glucose and was determined as the time to reach the maximal glucose value within a trial. A 2x3
(group x trial) ANOVA was performed for time to peak blood glucose. There was no significant interaction and no significant main effects for glucose peak times within a trial (main effect for trial) \( (p = .165 \text{ and } p = .226, \text{ respectively}) \). The average peak times in the combined group (\( N=12 \)) was 52.5 ± 24.35 minutes (45.0 ± 13.42 and 60.0 ± 31.46, low and high fitness, respectively) for the fast digestible CHO, 46.25 ± 17.47 minutes (50.0 ± 22.58 and 42.5 ± 11.29, low and high fitness, respectively) for the slow digestible CHO, and 40.00 ± 9.77 minutes (37.5 ± 8.22 and 42.5 ± 11.29, low and high fitness, respectively) for the glucose trial.

To further examine some of the findings, correlations were calculated (Pearson’s \( r \)) and a general linear regression model was utilized to determine the predictive nature \( (r^2) \) of relationships. A correlation within the high fitness group (\( n=6 \)) between body composition and time to peak blood glucose value for the slow digestible CHO indicated a strong negative relationship for the high fitness group \( (r = -.980, p = .001) \). Similarly, the body weight and baseline blood glucose value indicated a strong correlation for the high fitness group \( (r = .912, p = .011) \). In the low fitness group, body weight and time to peak blood glucose value for the slow digesting CHO indicated a moderate negative correlation \( (r = -.871, p = .024) \). The subject characteristic and glycemic responses indicated low relationships for combined group totals for percentage body fat and GI of slowly digestible CHO \( (r = .614, p < .05) \), and low negative relationships for, absolute VO\(_2\)max and GI of fast digesting CHO \( (r = -.587, p < .05) \), absolute VO\(_2\)max and maximal blood glucose value of fast digesting CHO \( (r = -.695, p < .05) \), absolute VO\(_2\)max and percentage increase of fast digestible CHO \( (r = -.692, p < .05) \), absolute
VO₂ max and time to peak of slow digestible CHO ($r = -.615, p < .05$), and relative VO₂ max and GI of fast digestible CHO ($r = -.621, p < .05$).
CHAPTER 5: DISCUSSION

The purpose of the current investigation was to determine if there was a difference in the glycemic response of six high and six low aerobically fit men to glucose challenges. Three glucose challenges were utilized in all 12 subjects, a standard 50g of dextrose monohydrate, and two novel CHO meals consisting of modified slow and fast digestible CHO. The novel carbohydrate substrate for the meals was prepared by Purdue University’s Whistler Center for Carbohydrate Research by way of a patent pending process, and then cooked according to their instructions prior to each subject consuming the meal. As part of the glycemic response, the GI was calculated and this investigation found a significant difference between the GI when determined in subjects of different aerobic fitness levels (VO2max) and body composition. High aerobic fitness subjects demonstrated a significantly lower GI for two separate test meals, the slow digestible CHO ($M = 38.80, SD = 18.38$), and fast digestible CHO ($M = 66.17, SD = 7.12$) compared to low aerobic fitness students slow digestible CHO ($M = 59.13, SD = 8.50$) and fast digestible CHO ($M = 86.71, SD = 16.15$) (Table 4). The high and low fitness subjects differed in both VO2max (ml/kg/min) (high $M = 56.90, SD = 4.47$; low $M = 42.05, SD = 0.81$) and body composition (percentage body fat; high $M = 16.15, SD = 1.93$; low $M = 25.99, SD = 5.63$). These results suggest that the GI of 100% CHO foods (non-“mixed meal”) are lower for high compared to low aerobic fitness subjects and furthermore, that the entire glycemic response is dependent upon aerobic fitness level. From these results, it is suggested that when using the GI as a tool for meal planning, the individual’s aerobic fitness status and body composition must be considered.
In support of the hypothesis, that the glycemic response will be less in the slow
digestible CHO than the fast digestible CHO for both fitness levels, the mean total GI for
the slow digestible CHO ($M = 48.97, SD = 17.30$) and mean total area under the curve
glycemic response ($M = 2037.52, SD = 1129.60$) was significantly different from the
mean total GI for the fast digestible CHO ($M = 76.44, SD = 16.02$), and mean total area
under the curve glycemic response ($M = 3328.45, SD = 1940.71$). Opposing the
hypothesis that the calculated GI for each food trial will be the same in the high and low
aerobic fitness groups, the GI was found to be significantly different between the low and
high aerobic fitness subjects, for both the slow digestible CHO (high fitness, $M = 38.80,$
$SD = 18.38$; low fitness, $M = 59.13, SD = 8.50$ ), and the fast digestible CHO (high
fitness, $M = 66.17, SD = 7.12$; low fitness, $M = 86.71, SD = 16.15$). However, the
differences in glycemic response support the hypothesis that it would be less in the high
fitness subjects than the low fitness subjects; however, the calculated area under the curve
glycemic response was not significantly different between groups for the slow digestible
CHO (high fitness, $M = 1376.15, SD = 548.72$; low fitness, $M = 2698.90, SD = 1206.78$),
or the fast digestible CHO (high fitness, $M = 2550.87, SD = 587.26$; low fitness, $M =
4106.03, SD = 2547.55$) (all area under the curve units, mg/dL/2hrs). The final
hypothesis, the calculated GI for the test foods would be a low GI and high GI food, was
correct. The mean GI for the slow digestible CHO in the combined group was less than
the 55 value that is used to establish low GI foods ($M = 48.97, SD = 17.30, N = 12$) and
mean GI for the fast digestible CHO in the combined group was above the 70 value that
is used to establish high GI foods ($M = 76.44, SD = 16.02$) according to the international
tables of the glycemic index compiled from scientific literature (Foster-Powell et al., 2002).

The current investigation found a significant difference between the GI when determined in subjects of different aerobic fitness levels (VO2max) and body composition, this is consistent with the only published literature to date that reported different GIs between sedentary and endurance trained subjects, the sedentary subjects were neither involved in any exercise-like activity nor engaged in physically intense occupational or leisure activity, the endurance trained subjects trained at least four times per week (Mettler et al., 2007; Mettler et al., 2006), but in complete disagreement with previous literature that states the GI is food specific and not subject specific (Jenkins et al., 1981; Wolever et al., 1991; Wolever et al., 1987).

The majority of research indicates that subject characteristics do not appear to have an effect on the mean GI values of foods (Wolever et al., 1991). It has been reported that small differences are unlikely to be detected because the correlation coefficient of GI values in IDDM versus NIDDM was $r = 0.928$ ($p < .001$) (Wolever et al., 1987). The suggestion is that the test meal’s characteristic is a primary determinant of the GI and not the metabolic state of the subjects. However, the current investigation demonstrates that subject characteristics may have an effect on the glycemic response to all foods; if the subject characteristics are standardized they appear to influence all foods similarly and may only have a small effect on the GI value (Wolever et al., 1991).

The calculated mean total glycemic response for each group as the AUC was significantly different for glucose ($M = 4318.24$, $SD = 1911.64$ mg/dL/2hr, $N = 12$), slow digestible CHO ($M = 2037.52$, $SD = 1129.60$ mg/dL/2hr, $N = 12$) and fast digestible CHO
(M = 3328.45, SD = 1940.71 mg/dL/2hr, N = 12). This indicates that the glucose and fast digestible CHO meals resulted in a greater change in blood glucose than that of the slow digestible CHO. Generally, high GI foods stimulate a large insulin rise, followed by a rapid blood glucose fall, a lower GI value results in a slower glycemic response and a smaller rise in blood glucose (Augustin et al., 2002). Interestingly, subject characteristics, including the percent body fat (r = 0.544 and 0.614, for slow and fast digestible CHO, respectively) and relative VO₂ max (r = -0.621 and -0.449, for slow and fast digestible CHO, respectively), independently explained a good amount of the variation in the AUC (between 20-39%). This increase in glycemic response from the fast digestible CHO may likely result in an intense counter regulatory hormonal response and result in low circulating blood glucose levels (Roberts, 2003). The counter regulatory hormonal response would indicate overcompensation in insulin secretion as a result of the elevated blood glucose levels and may contribute to a decline in beta-cell function over time (Rendell & Jovanovic, 2006). This would imply a strong relationship between the fast digestible CHO (those with high GI values) and the potential for metabolic/cardiovascular disease as a result of the postprandial hyperglycemia.

Conversely, the slow digestible CHO resulted in a lower peak in area under the curve glycemic response (M = 2037.52, SD = 1129.60, N = 12) and this attenuated glycemic response may promote beta-cell function- that is, not exert the same deleterious effects on the body as the fast digestible CHO, due to elevated postprandial glucose experienced from the greater glycemic response (Rendell & Jovanovic, 2006).

Previous literature has failed to investigate the importance of the glycemic response, and accepted that the GI values will be similar when the subject characteristics
are standardized. Utilizing aerobic fitness level as a subject characteristic was neglected by previous research. Previous literature concentrated on age, sex, body fatness, glucose tolerance status, dose and timing of insulin, degree of diabetes control, and fasting blood glucose value on the day of the test, accepting that standardization will influence GI values to all foods similarly (Wolever et al., 1991). Based on the results of this investigation, having a high aerobic fitness and low body fat results in lower glycemic responses and potentially lower insulinemic stress. The significant difference between the GI of the high and low aerobic fitness levels is important because postprandial blood glucose levels have important implications in metabolic and cardiovascular health, as indicated by accelerated coronary heart disease prevalence in individuals with diabetes mellitus. A commonly accepted benefit of high aerobic fitness is well maintained blood glucose concentration and control (Roberts, 2003). Conversely lack of physical activity is associated with increased risk of diabetes mellitus and cardiovascular disease (Augustin et al., 2002; Brennan, 2005; Rendell & Jovanovic, 2006). In the postprandial state, blood glucose concentration starts to rise and there is a pulsatile insulin secretion response resulting in an increase of glucose uptake by the liver, muscle, kidney, adipose tissue, and other insulin dependent tissues. Greater aerobic fitness, greater lean muscle mass and GLUT-4 concentrations may require less insulin to control blood glucose levels in the postprandial state (Rendell & Jovanovic, 2006). The degree of rise in blood glucose is determined by the amount of glucose entering and the amount leaving the circulation, and the effects of hyperglycemia include a reduced response to secrete insulin, this reduced response characterizes NIDDM and impaired glucose tolerance (Rendell & Jovanovic, 2006). Improving aerobic fitness with and
without improving body composition level may be the key to improving blood glucose maintenance. Previous literature indicates that the effects of acute and chronic aerobic exercise lead to an increase in insulin-stimulated glycogen synthase activity and glycogen storage in muscle, in healthy, obese non-diabetic, and NIDDM individuals (Christ-Roberts & Mandarino, 2004). Aerobic exercise training enhances glucose disposal by increasing glucose uptake, glycogen synthase activity, and glucose storage (Christ-Roberts & Mandarino, 2004), and GLUT-4 expression in overweight non-diabetic and NIDDM subjects (Christ-Roberts et al., 2004). Additionally, aerobic exercise training utilizes insulin independent mechanisms to increase glucose uptake in the skeletal muscle and aid in the maintenance of blood glucose. Regardless of body composition and metabolic state (diabetes), acute and chronic exercise training effect the amount of glucose leaving the circulation by the above mentioned principles of glycogen synthase activity, glucose uptake (Christ-Roberts & Mandarino, 2004) and GLUT-4 expression (Christ-Roberts et al., 2004).

High GI foods and fast carbohydrate absorption may result in large blood glucose fluctuations and disrupt hormonal balance (high insulin and low glucagon levels) inducing increased glucose storage and inhibiting lipolysis, creating a hypoglycemic environment (Augustin et al., 2002). Utilizing insulin independent mechanism as a result of aerobic exercise training may aid in glucose maintenance and may not require as great of an insulin response as seen from ingesting high GI foods, and decrease the likelihood of hypoglycemia. The metabolic state of hypoglycemia may be seen as a fasting state and trigger glucagon release and hunger signals (Augustin et al., 2002), this could lead to overeating. Mechanisms responsible for improving glycemic response are likely related
to GLUT-4 transporters, glycogen synthase enzyme content, and insulin secretion and sensitivity (Christ-Roberts et al., 2004; Hayashi et al., 1997; Holloszy, 2005). The concentration and activity of GLUT-4 may be responsible for the difference in the GI between low and high fitness subjects. Previous research has shown that aerobic exercise training increases GLUT-4 expression, translocation. Glycogen synthase activity, increasing glucose disposal has also been demonstrated to be affected by fitness levels (Christ-Roberts et al., 2004). Individuals of high fitness levels and greater fat free mass with less fat mass, display increased insulin sensitivity because of an increase in total concentration of GLUT-4 (Holloszy, 2005).

Furthermore, improving glycemic control may be beneficial for reducing oxidative damage (depression of serum antioxidants), and preserving high density lipoprotein (HDL) cholesterol (Jenkins et al., 2002). Additional areas of research include implications for obesity, the current assumption is that consuming a low GI meal early in the day may lead to less food consumption throughout the remainder of the day and the subsequent meals (Jenkins et al., 2002), and maintain glucose and insulin at moderate levels potentially avoiding a hypoglycemic state (Augustin et al., 2002).

The difference in the level of exercise training may have lead to a difference in the GI. The current investigation found similar results to Mettler et al. (2007) and Mettler et al. (2006) where the current training state of subjects influenced the GI. Endurance trained subjects were shown to have a lower GI for commercially available breakfast cereals than that of sedentary subjects, thus supporting the notion that the GI is subject dependent rather than food specific (Mettler et al., 2007; Mettler et al., 2006). The current investigation and Mettler et al. (2007) and Mettler et al. (2006) cannot
account for the acute effect for the previous exercise bout, as each subject group was allowed a different amount of time abstaining from exercise ranging from the day before the trial (less than 24 hours) to 2 days prior. Holloszy (2005) reports that exercise-induced increase in GLUT-4 translocation and contraction and insulin-stimulated glucose transport is seen 16 hours or longer after exercise, and an increase in GLUT-4 is not involved in the increase in muscle insulin sensitivity seen 2 to 4 hours after exercise (Holloszy, 2005). The current investigation attempted to control for this aspect by requiring all subjects to abstain from exercise for 36 hours prior to testing. However, this investigation cannot determine the underlying effect of the observed training state, or the degree of translocation and activation of GLUT-4, since no direct GLUT-4 concentrations were investigated. Previous literature is yet to elucidate the underlying cause of the variance in GI by aerobic fitness alone; however, the current investigation is in agreement with Mettler et al. (2007) and Mettler et al. (2006) that the aerobic fitness of young men will impact the GI of CHO foods.

Additionally, during exercise circulating glucose levels are maintained at a relative constant level, this is due largely to exercise induced changes in insulin and glucagon (Coker & Kjaer, 2005) as well as the hepatic contribution though glycogenolysis and gluconeogenesis. The literature suggests that physically active populations and athletes are more sensitive to insulin, and display lower insulin concentrations(Christ-Roberts & Mandarino, 2004; Christ-Roberts et al., 2004); therefore, in combination with increased GLUT-4 translocation activity and increase in concentration of GLUT-4, less insulin is required to transport glucose across the plasma membrane (Christ-Roberts & Mandarino, 2004; Christ-Roberts et al., 2004; Holloszy,
Skeletal muscle is the primary site of GLUT-4 expression, GLUT-4 translocation occurs in both types of skeletal muscle, red and white muscle fibers, additionally; a single bout of exercise has been shown to increase the total number of GLUT-4 present at the plasma membrane surface (Goodyear et al., 1991). Put together, this information suggests that increasing aerobic fitness may be responsible for altering the glycemic response, and explain the subject specific GI.

The current investigation found a significant difference between the aerobic fitness of the high and low fitness subjects (VO₂max (ml/kg/min), high $M = 56.90$, $SD = 4.47$; low $M = 42.05$, $SD = 0.81$) and body composition expressed as percentage body fat (high $M = 16.15$, $SD = 1.93$; low $M = 25.99$, $SD = 5.63$), and found a difference in the GI (high fitness, slow digestible CHO $M = 38.80$, $SD = 18.38$, fast digestible CHO $M = 66.17$, $SD = 7.12$; low fitness, slow digestible CHO $M = 59.13$, $SD = 8.50$, fast digestible CHO $M = 86.71$, $SD = 16.15$). Previous research has used healthy aged, and young men, IDDM and NIDDM subjects, hyperlipidemic subjects, and different race and ethnicity groups in order to determine if differences exist within GI, for these specific populations. A review of subject characteristics and the GI is available elsewhere (Jenkins et al., 2002; Wolever et al., 1991). No previous differences have been discovered and it is accepted that the GI is food specific and not subject specific, subject characteristics do not appear to have a major effect on the mean GI of foods (Wolever et al., 1991).

In addition to supporting evidence that the GI is subject specific, the difference in the CHO test food for this investigation support the GI as test food specific. Significant differences were found within the maximal blood glucose values of each test food for combined group totals (slow digestible CHO, $M = 114.16$, $SD = 13.88$; fast digestible
CHO, $M = 135.33$, $SD = 22.26$, all values mg/dl, $N = 12$) and the percentage increase from baseline value to maximal value within each test food for combined group totals (slow digestible CHO, $M = 38.11$, $SD = 13.59$; fast digestible CHO, $M = 67.84$, $SD = 22.00$, all values percent, $N = 12$). The fast digestible CHO resulted in a greater increase in blood glucose from baseline compared to the slow digestible CHO. Incorporating slow digestible CHO foods into diets for helping control and maintain fluctuations in blood glucose, because of the lesser degree of percentage increase compared to the fast digestible CHO. These findings support the GI as food specific because there were not differences between the low and high aerobic fitness subjects. No differences were found within the time to peak blood glucose concentration between groups or within test foods. The time to peak blood glucose from time of ingestion of food to time of maximal value for combined group totals were not significantly different (slow digestible CHO, $M = 46.25$, $SD = 17.47$; fast digestible CHO, $M = 52.5$, $SD = 22.35$; glucose $M = 40.00$, $SD = 9.77$, all values minutes, $N = 12$). These times indicate that the slow digestible CHO indicated a trend towards reaching a peak blood glucose value at a faster rate than that of the fast digestible CHO. The current investigation cannot determine whether the observed effect is subject specific for the difference in maximal values because insulin responses were not collected. Calculation of the insulin index has not shown significant differences between sedentary, moderate trained, and endurance trained subjects (Mettler et al., 2007). Insulin sensitivity is mediated by translocation of GLUT-4 and exercise-induced muscle activity (Holloszy, 2005). The insulin secretion may be related not only to the food but also to the subject, because of possible differences in GLUT-4 and insulin sensitivity (Christ-Roberts et al., 2004; Holloszy, 2005; Mettler et al., 2007). Improved
insulin sensitivity should improve the glycemic responses for all of the CHO test foods and thus not impact the relationship between different glycemic responses (Mettler et al., 2007).

A limitation to the current investigation was the lack of insulin data and measures of glucose disposal including GLUT-4 sacrolemmal and subcellular concentrations, using glucose as a reflection of the insulin response is a large speculation. The differences in maximal blood glucose only allow for speculation, high fitness subjects displayed lower maximal value concentrations, though these values were not significant. Impact on GI remains unknown, this investigation supports a subject training state specific GI, but cannot ignore the factors affecting a food specific GI.

The present investigation has a practical application to support that the GI may depend upon the subjects’ current fitness level. High fitness subjects displayed greater glucose tolerance and less glycemic response to all of the CHO test foods, compared to lower fitness subjects. This may be due to an increase insulin tolerance, less insulin secretion, and increased activation of GLUT-4. The response of insulin between fitness groups is equally important. From a health aspect, people of lower fitness levels should be encouraged to increase fitness levels, this improvement in fitness level may result in similar glycemic responses that was witnessed by high fitness subjects in this investigation. Current fitness level of subjects has often been neglected by GI researchers and should continue to be an important aspect and population group when investigating glycemic and insulin sensitivity responses. It is important to expand on the findings that training state does affect the GI of pure CHO foods.
REFERENCES


Flint, A., Gregersen, N. T., Gluud, L. L., Moller, B. K., Raben, A., Tetens, I., et al. (2007). Associations between postprandial insulin and blood glucose responses,


Appendix A: Institutional Review Board Documents

The following research study has been approved by the Institutional Review Board at Ohio University for the period listed below:

**Project:** The Glycemic Response to Fast and Slow Digestible Carbohydrates in High and Low Aerobic Fitness Men

**Researcher(s):**
- Adam Jackson
- Michael Kushnick

**Advisor:**
- Michael Kushnick

**Department:** Recreation and Sport Sciences

**Approval Date:** 12/19/06

**Expiration Date:** 12/19/07

This approval is valid until expiration date listed above. If you wish to continue beyond expiration date, you must submit a periodic review application and obtain approval prior to continuation.

The approval remains in effect provided the study is conducted exactly as described in your application for review. Any additions or modifications to the project must be approved by the IRB (as an amendment) prior to implementation.

Adverse events must be reported to the IRB promptly, within 5 working days of the occurrence.
The amendment, detailed below, and submitted for the following research study has been approved by the Institutional Review Board at Ohio University. Approval date of this amendment does not affect the expiration date of the original approval.

Amendment: Two vancomycins instead of two fingersticks to determine if subject is normoglycemic. Consent form revised accordingly.

Project: The Glycemic Response to Fast and Slow Digestible Carbohydrates in High and Low Aerobic Fitness Men

Project Director: Adam Jackson
Michael Kushnick

Advisor: Jacqueline Legg, M.B.A., Chair
Institutional Review Board

Date: 2/10/07

Department: Recreation and Sport Sciences
Appendix B: Health History Questionnaire

HEALTH HISTORY

Please indicate whether any of the following apply to you. If so, please place a check in the blank beside the appropriate item. Thank you.

___________ Hypertension or high blood pressure

___________ A personal OR family history of heart problems or heart disease
   If yes, please elaborate: ___________________________________

___________ Diabetes

___________ Orthopedic problems

___________ Cigarette smoking or other regular use of tobacco products

___________ Asthma or other chronic respiratory problems

___________ Illness, fever or Gastrointestinal Disturbances (diarrhea, nausea, vomiting)
   within the last month

___________ Known food allergies (especially to sorghum based products)

List any prescription medications, vitamin/nutritional supplements or over-the-counter medicines you routinely take or have taken in the last five days (including dietary/nutritional supplements, herbal remedies, cold or allergy medications, antibiotics, migraine/headache medicines, aspirin, ibuprofen, birth control pills, etc.)

I certify that the information that I provided on the health history form of the above stated research project was true, accurate and complete to the best of my knowledge.

______________________________
Signature

______________________________
Date
Appendix C: Data Collection Sheet

Criteria Met: VO₂max  Y  N
BF%:  Y  N

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Date: __________
Time: __________
Time to ingest: __________
Alcohol 36hrs:  Y  N
Exercise 36hrs:  Y  N
Fasting 12hrs:  Y  N
Appendix D: Subject Recruitment Flyer

Exercise Investigation

Men… How fit and fat are you?
How do carbohydrate “snacks” affect your blood glucose?

Exercise Physiology Research

Adam Jackson/Dr. Kushnick

aj226905@ohio.edu

Needed:
Men ages 18-29
NON-smoking and healthy

Will require:
A max treadmill test
Body Fat Assessment (BOD POD)
Glycemic Index Trial (11 finger pricks)
Dietary composition analysis

Will receive:
#1 VO₂max – Aerobic Capacity/Fitness
#2 Body Fat determined by the BOD POD
#3 Dietary composition profile
#4 Fasting and post-“snack” blood glucose levels
#5 Experience in the Exercise Physiology Laboratory!