BODY WEIGHT AND ADIPOSITY CHANGES OF OBESE GILTS PROVIDED *AD*

LIBITUM GROUND BEEF VERSUS HIGH CARBOHYDRATE DIETS

A Thesis
Submitted to the Graduate Faculty
of the
North Dakota State University
of Agriculture and Applied Science

By

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In Partial Fulfillment of the Requirements
for the Degree of
MASTER OF SCIENCE

Major Department:
Animal Sciences

May 2014

Fargo, North Dakota

North Dakota State University
Graduate School

Title

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The Supervisory Committee certifies that this ***disquisition*** complies with
North Dakota State University's regulations and meets the accepted standards
for the degree of

MASTER OF SCIENCE

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ABSTRACT

This study investigated the impact of providing a red meat (GB; cooked ground beef; 63% lean) or high-carbohydrate diet (CON) on growth performance and body composition of obese gilts as a biomedical model for humans. Treatment differences were observed for total intake (kg consumed/d; $P = 0.05$), average caloric intake (calculated kcals/d; $P = 0.003$), BW change ($P = 0.012$), and a treatment by harvest day interaction ($P = 0.001$) for pancreas weight. Subcutaneous backfat measured adjacent the 10th thoracic vertebra expressed as a percentage change from d0 tended ($P = 0.09$) to be less in GB gilts. There was no evidence of cardiac ventricular inflammation across treatments ($P > 0.21$). Despite consuming more total feed and more calories, the GB gilts gained less BW and deposited less subcutaneous fat over 84 days. More research is needed to further understand the physiological effect of food on human nutrition and health.

Key words: carbohydrates, gilts, humans, obesity, red-meat

ACKNOWLEDGEMENTS

Also to my family, mom, Angela, Jessica and Alex, thank you for you unending love, patience, guidance, understanding and support during my time at NDSU. I would not be here were it not for you pushing me to do something I didn't think I could.

To dad, I know you will not here to see me graduate, but I know that you have watched over me from the day you were taken from us. You were the one that showed me what life on the farm was all about and made me fully realize and understand the importance of agriculture and the bond that one can have with animals. You are my hero and the one that I will forever look up to. I know that although you are not here with me today you will forever be watching and believing in the decisions I will make. Know that I do not go a day without thinking about you. I love you!

Finally to my husband Adam, there are no words to express how thankful I am to have you in my life. You allowed me to take a leap of faith and continue my education. You allowed me to grow as an individual while still being there to pick me up when things were hard. Thank you for your unending love and support over the last six years. I cannot wait to see what the future holds.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ACRYONYMS

α…………………………………………………………………………alpha

ß…………………………………………………………………………..beta

°C………………………………………………………………..degrees Celsius

μU…..……………………………………………………………..microunit

ADG………………………………………………………average daily gain

ANPC…………………………………………Animal Nutrition and Physiology Center

ATP……………………………………………………….adenosine triphosphate

BF………………………………………………………………..back fat

BW……………………………………………………………body weight

CDC…………………………………………..Center for Disease Control and Prevention

cm………………………………………………………………centimeters

CON………………………………………………………………control

CP…………………………………………………………………crude protein

d……………………………………………………………………day

dL………………………………………………………………..deciliter

DM……………………………………………………………….dry matter

g………………………………………………………………….grams

g………………………………………………………………..gravity

GB……………………………………………………………ground beef

GI………………………………………………………………..glycemic index

GL……………………………………………………………glycemic load

GLUT4………………………………………………………glucose transporter 4

h……………………………………………………………………………….hour

IACUC…………………………………………Institutional Animal Care and Use Committee

kg……………………………………………………………………….kilogram

L………………………………………………………………………...liter

LM..…………………………………………………...longissimus muscle

LW……………………………………………………………...live weight

m^2………………………………………………………….meters squared

mg…………………………………………………………………….milligram

MIRKO…………………………………………muscle insulin resistant knockout

mL…………………………………………………………………….milliliter

mmol…………………………………………………………………...millimole

NDSU……………………………………………………North Dakota State University

NIH…………………………………………………...National Institutes of Health

NPB…………………………………………………………...National Pork Board

NRC……………………………………………………National Research Council

SAS……………………………………………………Statistical Analytical Software

US……………………………………………………………….United States

USDA…………………………………………United States Department of Agriculture

vs. ………………………………………………………………………versus

CHAPTER I. LITERATURE REVIEW

Introduction

Dietary decisions made by consumers are influenced by many factors including food type, cost, and convenience. For many consumers, the price of food is more important than overall quality (Blisard et al., 2004). Further, foods higher in fat and sugar are typically less expensive. Lower priced foods tend to be consumed more readily, regardless of benefits or disadvantages to overall health (Kaufman, 1997; Drewnowski and Spectar, 2004). Excessive caloric intake and diminished physical activity (exercise) are the two most common factors attributed with obesity and obesity-related diseases. Several factors related to increases in obesity include consumption of large portion sizes of foods recording a high glycemic index (**GI**). High GI foods cause a rapid release of insulin upon ingestion causing blood sugar and insulin levels to stay high or cycle up and down rapidly (Ludwig, 1999). When consumer health is compromised, debate over appropriate food choices becomes an issue as health officials seek consensus regarding what is considered a "healthy" food choice.

Diseases of Modern Civilization

Atherosclerosis, chronic liver disease, chronic obstructive pulmonary disease, type 2 diabetes, heart disease, and metabolic syndrome are diseases that appear to increase in frequency in industrialized countries as people accumulate more disposable income. An increase in disposable income allows the means to consume more dairy products, vegetable oils, sugary foods, and alcoholic beverages (Drewnowski, 2003). The amount of time once occupied by the physical demands of day to day survival are replaced by careers that are often much less physically demanding. People may become more sedentary, which in turn, may result in higher rates of obesity (Drewnowski, 2003).

1

In 2008, the Center for Disease Control and Prevention (CDC, 2008) found there were an estimated $147 billion dollars in medical costs associated with obesity. All 50 states had a prevalence of obesity greater than 20%, 36 states had an obesity rate of 25% or more, and 12 of those states had a prevalence of 30% or more (CDC, 2008). Obesity is a very complex issue attributed, but not limited to, over-consumption of easily digestible, energy-dense, convenient-access foods, combined with sedentary lifestyle and potential genetic predisposition which alters otherwise normal physiological mechanisms. Because of the obesity-related changes in physiology, obesity is a characteristic symptom of many diseases of modern civilization. It is characteristic of so many diseases that they are often described "obesity-related disorders" (Liese et al., 1998).

Obesity and diabetes have steadily increased over the last several decades. According to the American Diabetes Association (ADA, 2011), 8.3% (18.8 million people) of the United States population has been diagnosed with diabetes while another 7 million remain undiagnosed and 79 million are thought to possess symptoms of pre-diabetes. Based on these numbers, that would mean that 46.3% of the U.S. population is either diagnosed or undiagnosed with diabetes or pre-diabetes.

Another obesity-related disorder is the metabolic syndrome. Metabolic syndrome, defined several different ways, is a common metabolic disorder that originates from an increasing prevalence of central (midsection) obesity. Metabolic syndrome is also known as syndrome X, the insulin resistance syndrome, and (or) the deadly quartet (Eckel et al., 2005). The deadly quartet is comprised of upper-body obesity, glucose intolerance, hypertriglyceridemia, and hypertension (Kaplan, 1989). There are many underlying symptoms of metabolic syndrome that are manifested individually or in combinations. Diagnosable criteria

include glucose intolerance (type 2 diabetes, impaired glucose tolerance), insulin resistance, central adiposity, dyslipidemia, and hypertension (Lakka, et al., 2002).

Increasing rates of obesity in young people have resulted in diagnosis of type-2 diabetes and metabolic syndrome earlier in life than previously seen in the American population (Sinha et al., 2002; Weiss et al., 2004). Fagot-Campagna et al. (2000) reported that 33 to 45% of new diabetes cases are among adolescents. Mantzoros (2006) suggested these issues have become increasingly more prevalent in children and adolescents since the mid-1990s and increased diagnosis of childhood obesity and type-2 diabetes mellitus are not only common in the U.S., but worldwide. According to Bluher and Kiess (2006), type-2 diabetes mellitus and obesity are currently regarded as two of the most challenging health issues facing young children and adolescents.

It is the intent of this literature review to discuss relationships between macro-nutrients consumed in modern American diets and how this has generated a physiological and metabolic shift resulting in a change in the American phenotype in less than three decades. This shift has caused a cascade of diagnosis of the diseases of modern civilization, which have now come to be described as obesity-related metabolic diseases.

Physiological Aspects of Human Obesity

In the broadest sense, normal human growth and development follows this order; neural, skeletal, muscle, and adipose development. In the active growth phase, bone matures to provide a support structure and framework for muscle development. In mammalian development, pre-pubertal muscle growth and development are very energetically expensive and energy consumed from foodstuffs is principally used to fuel muscle hypertrophy. It may have been observed in an historical sense that pre-pubertal males and females could consume just about anything to fuel

the active growth of muscle and bone without leading to an increase in adiposity. Again historically, adiposity occurred post-puberty when muscle hypertrophy reached its plateau and the amount of energy necessary for active growth dramatically diminished. Therefore, post-pubertal diets should consider ingestion of energy for maintenance. These maintenance energy requirements should be considerably lower than previously necessary for active growth and development.

Insulin is a vital anabolic hormone responsible for intermediary metabolism and partitioning fuel nutrients for either storage or oxidation. Insulin serves four basic metabolic functions: (1) initiates glucose transport into cells, (2) triggers/initiates growth and gene expression resulting in a cascade of events that promote excess dietary energy to be stored as glycogen or adipose, (3) facilitates glycogen synthesis within the cell, and (4) promotes protein synthesis.

Insulin-mediated glucose storage is the means for which blood sugar is regulated. Elevated concentrations of glucose in the blood will stimulate the release of insulin from pancreatic ß cells. Insulin will then stimulate uptake, utilization, and storage of glucose in target cells possessing insulin receptors. Insulin receptors are made up of 2 α-subunits that are linked to a β-subunit and to each other by disulfide bonds forming a $\alpha_2\beta_2$-heterotetramer (Cheatham and Kahn, 1995). Each subunit is responsible for performing different functions. The α-subunit is responsible for ligand binding while the β-subunit utilizes an insulin-stimulated protein kinase that is responsible for modifying other proteins (phosphorylation). Phosphorylation is important in reactions related to cellular signaling, regulation, and energy management (Bender and Mayes, 2006).

Humans tend to perform better when blood sugar concentrations remain stable. Significant spikes in blood sugar cause the brain to signal the pancreas to increase the amount of insulin secreted into the blood. The liver is an organ of glucose production and consumption. During normal feeding and fasting cycles, pancreatic insulin and glucagon will modulate the levels of blood sugar (glucose) necessary to fuel fasting periods. In periods of fasting (including time between meals), the liver serves as the primary source of glucose (Sherwin, 1980). When blood glucose concentrations exceed levels necessary for energy demands, the liver maintain blood glucose homeostasis by removing glucose from the blood and storing it as glycogen. When blood glucose declines, the liver will then produce new glucose to release into the blood. In a normal cycle, insulin facilitates blood glucose clearance and storage. As glucose concentration in circulation declines, this signals the pancreatic ß cells to reduce insulin production. In response to low blood glucose, the pancreatic α cells secrete the hormone glucagon. Glucagon serves the opposite function of insulin as it signals release of glucose from glycogen stored in hepatocytes. Blood glucose will rise to fuel the brain, metabolic processes, and physical activity. In normal metabolism, this cycle will continue to alternate between glucagon and insulin. When activity surpasses glycogen energy reserves in the liver, hunger receptors in the brain are activated and nourishment is sought out and consumed (Van Itallie and Kissileff, 1985). Or during periods of prolonged fasting, tissue protein catabolism provides gluconeogenic amino acids to the liver for conversion to glucose to increase blood sugar levels.

Even though the liver possesses a high percentage of its mass as glycogen, muscle is the largest source of glycogen. There are no glucagon receptors on muscle tissue and glucose/glycogen stored in muscle cannot be released into circulation to, for example, fuel the needs of the brain. This makes sense because muscle contraction is dependent on plentiful

5

supplies of ATP, which is hydrolyzed by the contractile protein myosin when it binds actin to facilitate the contractile power-stroke. Small quantities of ATP are stored in muscle to facilitate quick bursts of strength (as in the anaerobic fight or flight response). However, during periods of prolonged contraction (aerobic), ATP is generated from release of glucose from myofibrillar glycogen (a process called glycogenolysis) for entry into glycolysis or generated from fatty acid oxidation through the electron transport chain.

Consuming quantities of high glycemic carbohydrates results in the greater need for more insulin to facilitate clearance of circulating glucose into cells where they can be used immediately (active exercise) or stored as fuel during times of inactivity (Taubes, 2007). If the liver becomes saturated with glucose (glycogen), insulin assists in mediation of excess glucose for storage as triglyceride through conversion of the 6-C glucose into 2-C acetyl-CoA molecules which link in formation of FFA (Taubes, 2007). Insulin action on glucose uptake in muscle and fat results from a cascade of signaling events originating from insulin binding its receptor and ending in the translocation of the major insulin responsive glucose transporter (GLUT4) from intracellular spaces to the plasma membrane (Kahn, 1998). As the only glucose transporter regulated by insulin, GLUT-4 is primarily found in skeletal/cardiac muscle and adipose tissue (Cheatham and Kahn, 1995). If muscle tissue is saturated with glycogen, insulin receptors once present on the sarcolemma (muscle cell wall) down-regulate, now making the myofiber insulin resistant. The World Health Organization identifies insulin resistance as type-2 diabetes, impaired fasting glucose, glucose intolerance, or a glucose uptake below the lowest quartile for the average American. (Chaiken and Banerji, 2006). Insulin resistance is described as being a multifaceted syndrome that can be associated with numerous medical conditions such as obesity, hypertension, non-insulin dependent diabetes mellitus, dyslipidemia, and atherosclerotic

cardiovascular disease. An individual that is pre-diabetic may possess tissue-specific insulin resistance as a result of glycogen saturated down-regulation of myofibrillar insulin receptors. When individuals with this condition consume a carbohydrate-rich diet, more insulin will be secreted by ß cells in response, prompting greater synthesis and secretion of triglycerides into circulation or to be accumulated as liver fatty deposits (Taubes, 2007). Insulin is a major player in the life of the adipocyte which is one of the most insulin-responsive cell types (Kahn and Flier, 2000). As the primary anabolic hormone, insulin promotes storage of energy (Michael et al., 2000). If receptors on adipocytes remain viable, insulin will bind its receptor on the adipocyte and facilitate the clearance, transport, and storage of circulating triglycerides into the adipocyte. Therefore, sedentary lifestyle and dietary induced hyperglycemia ultimately leads to hyperinsulinemia, an accumulation of triglycerides, and increased adiposity. This metabolic scenario of tissue-specific insulin resistance suggests that eating a high glycemic, carbohydrate-rich diet can increase the risk of obesity-related metabolic disorders (Taubes, 2007).

Unnikrishnan (2004) noted that conceptually, it is better to consider insulin resistance as being tissue-specific, where individual phenotype is dependent on differing sensitivities of specific tissues. Unnikrishnan (2004) also stated that the multifaceted array of issues associated with metabolic syndrome indicates insulin resistance cannot be considered just a uniform alteration within the human body. Kim et al. (2000) noted several studies (Joshi et al.,1996; Accili et al.,1996) altering muscle insulin receptors in muscle insulin resistant knockout (MIRKO) mice during a 2-h hyperinsulinemic-euglycemic clamp resulting in basal plasma insulin concentrations increasing by approximately 50% compared to control mice. Insulin-stimulated whole-body glucose uptake decreased by 45% in the MIRKO mice and insulin-stimulated whole-body glycolysis was decreased by 25%. However, the most interesting finding

was an 87% decrease in insulin-stimulated whole-body glycogen/lipid synthesis in MIRKO mice. Insulin-stimulated rates of glycolysis and glycogen synthesis in skeletal muscle decreased by 73% and 88% in MIRKO mice. On the other hand, insulin-stimulated glucose transport activity in epididymal white adipose tissue was significantly (3 times) greater in MIRKO mice (Kim et al., 2000).

Dietary Glycemic Index and Glycemic Load

The glycemic index (GI) is a ranking of foods or foodstuffs on a scale from 0 to 100 according to the extent in which ingestion impacts blood glucose levels after consumption (Brand-Miller et al., 2002). The GI response is measured in vivo as the plasma glucose response to a standardized amount of ingested carbohydrate (Gross et al., 2004). Jenkins et al. (1981) published the first list of 51 foods and their GI indices. The GI of a particular food (or meal) is determined primarily by the nature of the carbohydrate(s) consumed. Foods with a high GI are typically highly digestible, triggering a rapid increase in blood sugar levels under normal metabolic conditions. It has been stated in the previous section that hyperglycemia stimulates insulin secretion, which promotes glucose uptake by muscle and adipose tissue. Within the first 2 h after a high GI meal is consumed, blood glucose concentrations can be as much as 2 times greater when compared to low GI meal containing identical nutrients and energy. Between 2 to 4 h after a high-GI meal, nutrient absorption begins to decrease, while high insulin levels and low glucagon levels continue. A high dietary GI may also impair ß cell function through direct effects of elevated levels of blood glucose and FFAs. Hyperglycemia has also been shown to cause ß cell dysfunction, otherwise known as glucotoxicity. Long-term studies in animal models have also shown that high-GI starch diets promote weight gain, visceral adiposity, and higher levels of lipogenic enzymes (Kabir et al., 1998; Pawlak et al., 2001).

Most refined starchy foods consumed in the United States possess a high GI, whereas vegetables, legumes, and fruits tend to have a lower GI. Variation in foodstuffs that contain similar carbohydrate content may vary due to true differences in physical and chemical characteristics of the specific food. Secondly, two similar foods may have been processed differently, causing differences in carbohydrate digestion and therefore, differing GI values. Also, differences in type of flour used, the moisture content, and cooking time can have an impact on the GI of food (Brand-Miller, 2002). Fat and protein foodstuffs have low GI and their inclusion as part of a whole meal will typically lower the overall GI (Bornet et al., 1987).

The compilation of the *International Tables of Glycemic Index* (Foster-Powell et al., 2002) provided the basis for the GI to be used as a tool for novel comparison of different carbohydrates relative to a foods potential for disease risk. In this case the GI is determined by comparing the postprandial glycemic response of a specific food with the postprandial glycemic response to the same amount of available carbohydrate from a standard food (white bread or glucose) in the same individual (Jenkins et al., 1981). There are 3 levels associated with the GI. Low GI foods (Table 1.1) span an index from 0 to 55 (Table 1.1; e.g. oatmeal, sweet potatoes, fruits, and non-starchy vegetables). The second level, ranging from 56 to 69 (Table 1.1; e.g. brown and wild rice) indicates moderate GI foods and the third level, high GI foods possess a range from 70 to 100 (Table 1.1; e.g. potatoes, bagels, white bread). Other specific non-meat foodstuffs can be seen in Table 1.2. Factors affecting the GI of a food include the type of starch and the amount of fiber, fat and protein. Fiber content of specific foodstuffs has also altered the rate of nutrient absorption from the gastrointestinal tract (Eisenhans et al., 1980; Johnson and Gee, 1980). High fiber meals produce a lower glucose response in both normal and diabetic patients (Jenkins et al., 1977; Potter et al., 1981). The rationale for the relationship between

9

starch digestion and glycemic response to higher fiber meals is due to a reduced rate of gastric

emptying (Leeds et al., 1978; Taylor, 1979) and a reduced rate of intestinal absorption

(Eisenhans et al., 1980; Johnson and Gee, 1980). Fat and protein content of different foodstuffs

also impact the GI content as well. Overall, both protein and fat reduce the glycemic response

seen by oral glucose in normal humans (Pi-Sunyer, 2002; Nuttall and Gannon, 1991). You will

notice that whole-muscle foods are missing from Table 1.1. According to the American Diabetic

Association, meat (protein and fat) does not have a GI because it does not contain glucose.

However, studies by Spiller et al. (1987) and Owen and Wolever (2003) suggest that adding fat

and protein to carbohydrate reduces glycemic responses nonlinearly, with the glycemic impact

plateauing as more and more protein and fat are added.

Table 1.1. Three levels associated with the glycemic index[1]

Level	Range
Low	0 to 55[2]
Moderate	56 to 69[2]
High	70 to100[2]

[1]Adapted from the American Diabetes Association, 201l.
[2]The glycemic index ranks foods and beverages based on how they affect blood sugar level. Foods are scored on a scale of 0 to 100.

Glycemic load (GL) is a product of the GI and combines both the quality and quantity of

the carbohydrate into one number (numeric index). The GL is a more accurate tool to assess the

impact of carbohydrate consumption. The values allow consumers to monitor the number of

carbohydrates they are consuming compared to the GI, which ultimately gives an indication of

how rapidly a particular carbohydrate is converted into sugar (Rakel, 2008). The formula for

calculating GL is [GI x grams of carbohydrate consumed] / 100. Thompson (2006) suggested the

daily sum of GL should be less than 500. The GL is an indication of how rapidly a carbohydrate is digested and released as glucose into the blood stream while GI does not take into account the amount (load) of carbohydrate of the foodstuff. Therefore, GL is a better predictor of how a carbohydrate will affect blood sugar (Brand-Miller, 2002). Brand-Miller et al. (2002) also showed that dietary GI and GL were independent predictors of type 2-diabetes risk and cardiovascular disease. Furthermore, The Nurses' Health Study (Salmeron et al., 1997) found those in the top tier of GL food consumption had a 50% increase in type-2 diabetes risk over a 6-yr follow-up period.

Understanding GI and GL is an important means for individuals to monitor healthful diets. Regular consumption of high-GI meals will cause blood glucose levels to rise more rapidly than consumption of low-GI meals. High blood glucose levels and subsequent excessive insulin secretion could create potential health issues such as nerve damage, heart disease, stroke, kidney disease, and other complications (National Institutes of Diabetes and Digestive and Kidney Diseases, 2011). Steady consumption of high-GI food results in higher average 24-h blood glucose and insulin levels, higher C-reactive protein excretion, and higher glycosylated hemoglobin concentrations in non-diabetic individuals (Jenkins et al., 1987; Miller, 1994). C-reactive protein is a protein found in the blood that typically appears after an injury, infection, or inflammation (Black et al., 2004). Research suggests that patients with prolonged elevated C-reactive protein levels are at increased risk of diabetes and metabolic syndrome, heart disease, and hypertension (Libby et al., 2004). Deron (2003) observed that C-reactive protein levels were typically found to be higher in those that smoke, are obese, and lack exercise. Chronic hyperglycemia can result in glucose covalently binding with the molecule hemoglobin. The glycosylated hemoglobin concentration represents the average blood glucose level over the

11

previous 2 to 3 months. In controlled diabetes mellitus, the concentration of glycosylated

hemoglobin is within the normal range, but in uncontrolled cases, levels may be 3 to 4 times the

normal concentration (Bayer, 2011).

Table 1.2. Non-meat foods associated with different levels of the glycemic index[1]

Level	Foodstuff
Low	oatmeal
	sweet potatoes
	corn
	barley
	peanuts
	lentils
	beans
	grapefruit
	soybeans
Moderate	wild rice
	white rice
	beets
	pineapple
	brown rice
	table sugar
	power bars
	chocolate bars
High	white bread
	potatoes
	cereals
	bagels
	jelly beans
	glucose
	soda crackers
	rice crackers

[1] Adapted from The University of Sydney, 2011. Jennie Brand-Miller, PI.

Dietary Intervention

Recent data from Gross et al. (2004) suggested high intake of refined carbohydrates may increase the risk of insulin resistance. Although there is an increase in the intake of refined carbohydrates in the form of processed grains, soft drinks, sweeteners, and refined flours, very little has been done to determine whether such changes in dietary composition are related to the current epidemic of obesity and type-2 diabetes in the United States (Gross et al., 2004). Data from the United States Department of Agriculture reveals modern carbohydrates are different from those that were consumed prior to the 20th century. Refining carbohydrates has changed the composition and impacted the bioavailability of the food products resulting in more rapid metabolic responses (Durtschi, 2001). Components that naturally held high-glycemic starches and sugars in check and slowing the glycemic response of the entire foodstuff have been removed leaving behind starches or sugars that add the most flavor, but have the highest glycemic response. For example, processing whole grains into white flour increases the caloric density by > 10%, reduces the amount of dietary fiber by 80%, and reduces the amount of dietary protein by almost 30% (Durtschi, 2001).

It has already been stated that low GI foods have less of an impact on blood sugar and subsequent insulin release (Jenkins et al., 1981). Regular consumption of low GI foods can assist in the maintenance of stable blood sugar and insulin levels that may decrease the risk of developing obesity-related metabolic disorders (Brand-Miller, 2003). Ludwig et al. (1999) stated consumption of high GI and GL meals resulted in a cascade of events leading to limit the availability of metabolic fuels, resulting in excessive hunger and overeating. For example, obese subjects consumed 81% more energy after two meals of instant oatmeal compared to two meals with the same amount of energy in the form of a vegetable omelet and fruit. Secondly, obese

subjects ate 53% more energy after consuming a high-GI instant oatmeal than they did after eating a medium-GI steel-cut oatmeal. Slabber et al. (1994) reported obese women whom were counseled to consume only low GI foods vs. those not counseled increased the amount of weight lost (4.24 ± 1.13 and 3.36 ± 1.92 kg , respectively). Bouche et al. (2002) found overweight men had significantly (~700 g) more fat mass loss after 5 weeks of low GI vs. high GI diets. Similarly, Spieth et al. (2000) observed children given a reduced-GI diet had a 1.15 kg/m^2 adjusted decrease in BMI. Halton and Hu (2004) reviewed 15 randomized controlled studies of higher-protein compared to lower-protein diets ranging from 7 days to 1 year on weight loss using a wide variety of macronutrient rations and methodologic designs. Seven of these studies found statistically significant decreases in body weight in higher protein diets. In one study, Skov et al. (1999) found obese subjects randomly assigned to a high-protein intake diet (25% energy) lost significantly more weight (8.8 vs 5.1 kg) and fat (7.6 vs 4.3 kg) after 6 months compared with a low protein diet (12% energy).

In 1982, geneticist James Neel hypothesized three scenarios regarding insulin-secretory responses that could lead to a predisposition of obesity and type 2-diabetes. He recognized these hypotheses as response to excessive glucose pulses resulting from refined carbohydrate and over-alimentation of many civilized diets.

The first scenario was denoted as a "quick insulin trigger," meaning the pancreatic insulin-secreting cells were hypersensitive to glucose in the bloodstream thus secreting too much insulin in response to increased blood sugar. This stimulated fat deposition and potentially insulin sensitivity in muscles. Ultimately this scenario suggested pancreatic cells would lose their capacity to respond to developing insulin resistance; possibly leading to type-2 diabetes (Taubes, 2007). Neel's second scenario suggested a tendency to become more insulin-resistant than

14

normal when dealing with a given amount of insulin in circulation (Taubes, 2007). Therefore, even the "normal" level of insulin response to changes in blood sugar would potentially result in insulin resistance because "the cycle" would begin again (Taubes, 2007). Neel's final scenario suggested appropriate amounts of insulin are secreted in response to "excessive glucose pulses" of a modern day meal (Taubes, 2007). The challenge with this was the relative sensitivity of muscle and fat cells to insulin (Taubes, 2007). Muscles become insulin resistant in response to excess excretion of insulin in the system stemming from excessive ingestion of refined carbohydrates (Taubes, 2007).

The American Diabetes Association (2011) suggests those trying to reduce their chances of becoming pre-diabetic or diabetic should consume fresh fruits and vegetables, nuts, and protein. Nuts are beneficial because they are a low GI food and provide adequate amounts of protein. Vegetarian diets have the potential to decrease the GI, which in turn will help decrease the insulin and glucose spikes in the blood, keeping levels more consistent while providing adequate nutrients. Several foods such as oysters, shrimp, and avocados can be used to replace high glycemic carbohydrates because they contain complete proteins and essential fatty acids to maintain and promote bodily functions. Diets that do not include animal products (meat, eggs, and dairy) eliminate these complete sources of essential amino acids. The strict vegan diet must combine vegetable sources of protein (i.e., corn and beans) to provide complete and adequate amounts of essential amino acids, yet may inadvertently end up consuming larger quantities of starch and thus unnecessarily increase the carbohydrate load of their diet.

CONCLUSION

In summary, a multitude of animal and human studies have hypothesized the consumption of high-carbohydrate, high GI diets (along with a lack of exercise) can result in postprandial hyperglycemia and hyperinsulinemia (McFarlane et al., 2003; Arora and McFarlane, 2005). Glycogen stores in the liver and muscle are typically maintained at higher levels in high GI diets. Consumption of high GI meals stimulate gluconeogenesis (Brand-Miller et al., 2002), causing greater dependence on carbohydrate and protein as sources of fuel 0 to 6 hours following eating. Therefore, increasing carbohydrate dependence may stimulate the need to consume more calories due to the feeling of hunger. These mechanisms can lead to overconsumption of carbohydrates and if this habit is maintained for long periods of time, could potentially lead to increased body weight gain and associated issues.

Understanding the human diet and the type of macro-nutrients consumed in modern American diets is crucial to the development and prosperity of the human population. Future research is necessary in order to continue to show how human diet choices have generated a physiological and metabolic shift in the American phenotype in less than three decades. A better understanding of this shift will allow for the development of strategies to optimize human health and well-being.

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19

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CHAPTER II. BODY WEIGHT AND ADIPOSITY CHANGES OF OBESE GILTS PROVIDED *AD LIBITUM* GROUND BEEF VERSUS HIGH CARBOHYDRATE DIETS

Abstract

The objective of this study was to evaluate the influence of diet on weight gain, body composition, and fat deposition in obese gilts fed a high calorie diet. Yorkshire × Duroc × Hampshire gilts (N = 21) born over a five-day period from a common sire were provided *ad libitum* access to a low lysine diet (Lys < 0.45%) to promote hyperphagia and adiposity. Upon reaching 3 cm subcutaneous backfat (10BF; 10/11th rib interface), dietary treatments were allocated across BW to either a ground beef (GB; n = 5) or control (CON; n = 5) treatment. The GB diet was 99.9% cooked ground beef (65:35 lean:fat) plus 0.1% calcium carbonate while CON comprised 70.55% ground corn, 15% vegetable oil, 8.5% distillers dried grains plus solubles, and 4.25% soybean meal. Both rations met NRC requirements for gilts of this size and weight. Intake and orts were recorded daily. Body weights (BW) and blood draws were collected on d0, 28, 56, and 84. Gilts were humanely slaughtered on d85 for tissue collections and body composition analysis. One gilt was removed from the GB due to foot infection. The CON gilts gained 0.77 while GB gilts gained 0.61. kg/d (P = 0.02). Estimated kilocalorie consumption/d was lower (P = 0.05) for CON (14,007 kcals/d) versus GB (16,557 kcals/d) from d 35 to 84. The percentage change of 10BF and BW from d0 were calculated as [(d28, 56 or 84 – d0)/d0]×100 and evaluated as repeated measure for the interaction of treatment by day. The GB gilts had a higher percent change in BW (P = 0.012) and a tendency for 10BF percent change (P < 0.09). No differences were observed (P ≥ 0.15) for pancreas, adrenal, liver, heart, or spleen weights. Despite consuming more total feed/food and more calories, the ground beef fed gilts gained less body weight and deposited less subcutaneous fat over the 84 days on test. More research is

needed to further understand the physiological effect of food and food combinations on human nutrition and health.

Key words: adiposity, carbohydrates, carcass characteristics, gilts

Introduction

The number of diagnosed cases of obesity and diabetes has steadily increased in the United States over the last several decades in children and adolescents. Data by Gross et al. (2004) suggested that a high intake of refined carbohydrates in the form of processed grains, soft drinks, and sweets is related to the current epidemic of obesity and obesity-related issues in the United States. Ludwig et al. (1999) stated that consumption of high glycemic index (GI) and glycemic load (GL) meals resulted in a cascade of events that limit the availability of metabolic fuels, leading to excessive hunger and overeating. The GI is a ranking of foods or foodstuffs on a scale from 0 to 100 according to the extent in which ingestion impacts blood glucose levels after eating (Brand-Miller et al., 2002). Glycemic load is a product of the glycemic index (GI) and combines both the quality and quantity of the carbohydrate into one number (numeric index). The glycemic load takes into account the impact of carbohydrate consumption. The GL values allow consumers to monitor the number of carbohydrates they are consuming compared to the GI, which ultimately gives an indication of how rapidly a particular carbohydrate is converted into sugar (Rakel, 2008). Skov et al, (1999) found that obese subjects that were randomly assigned to a high-protein diet (25% energy) lost significantly more weight (8.8 vs 5.1 kg) and fat (7.6 vs 4.3 kg) after 6 months compared with those on a low protein diet (12% energy). Ultimately, this study showed that the replacement of dietary carbohydrates with protein improved weight loss and increased clinically relevant weight loss. It has been shown that regular consumption of low GI foods assist in maintenance of stable blood sugar and insulin levels which may decrease risk of developing obesity and obesity-related disorders (Brand-Miller, 2003).

Pigs are omnivores and their anatomy and physiology are very similar to humans. A pig's gastrointestinal system, body composition, and nutrient requirements favor the use of the pig as an ideal model for evaluation of how diet influences physiological responses in growth and development (Tumbleson, 1986; Swindel et al., 1994; Tumbleson and Schook, 1996; Smith and Swindle, 2006). The National Institute of Health has stated that the pig is an excellent biomedical model with regard to the influence of diet on insulin regulation and function. This study tested the hypothesis that over consumption of carbohydrates would lead to increased fat deposition and weight gain compared to gilts provided a red meat diet. The pig as a model for humans has also been instrumental in the investigation of heart physiology, reproductive function, transplantation, skin physiology, brain and biochemical research as well as respiratory function and infectious disease related to humans. The molecular basis of the pig is much closer to that of humans than any other laboratory animal species, therefore becomes a useful model for the study of genetic components of human obesity and other related disorders (Kuzmuk and Schook, 2010).

Materials and Methods

This study was conducted at the North Dakota State University Animal Nutrition and Physiology Center (ANPC). All animal care and handling procedures were approved by the Institutional Animal Care and Use Committee (IACUC).

Animals and Diets

A pool of 21 crossbred gilts, approximately 90 days of age were selected from the NDSU swine unit and transported 0.8 kilometers to the ANPC (Fargo, ND). All pigs were born over a five-day period and had a common sire. Prior to being put on treatment, gilts were housed in individual pens (1.22 x 2.44 m) in the same room held in thermo-neutral, environmentally

controlled conditions. All gilts were allowed *ad libitum* access to water and a common diet

formulated using the guidelines of the National Research Council dietary recommendations for

growing swine (NRC, 1998). The common diet was purposefully low in lysine to promote

hyperphagia and increased adiposity. The choice of the low lysine diet was based on the findings

of several studies (Witte et al., 2000; Cisneros et al., 1996). The diet was analyzed for DM, ash,

CP, fat, and percent lysine (Table 2.1). Back fat thickness and loin muscle area (LMA) at the 10th

thoraxic vertebra were determined using an Aloka 500-SSD (Aloka America, Wallingford, CT).

Gilts were then ultrasounded every 14 d for the remainder of the study.

It should be noted that only the first 10 gilts to reach 3 cm of BF from the pool of 21 were

selected due to the extended amount of time it took to get each pig to the appropriate BF level.

Also, gilts were put on trial as a paired sample in order to ensure multiple pigs were slaughtered

on the same day and could also be compared across treatments. Upon reaching 3 cm of 10th rib

backfat, gilts were randomly assigned to one of two dietary treatments stratified across BW and

BF. The control gilts (CON; n = 5) received a standard commercial grower diet formulated to

NRC (1998) recommendations (Table 2.1). This diet consisted mainly of corn, soybean meal,

and dried distillers grains (DDGS) with an additional 15% fat added in the form of corn oil. The

treatment diet (GB; n = 5) was fully cooked 65:35 lean:fat ground beef (GB) top dressed with

calcium carbonate (0.10% GB, as fed; Table 2.1) to meet NRC requirements for a complete

balanced diet. This diet was fed four times per day at 0800, 1000, 1400 and 1600h to avoid

spoilage of this high fat diet. Although it was not provided as what would be traditionally

considered *ad libitum*, the GB pigs were allowed to consume as much as they wanted over these

four feedings each day.

Table 2.1. Ingredient composition and analyzed nutrient composition of experimental diets fed to gilts for 84 d

		Treatments	
Item	Fattening Diet[1]	CON	GB
Ingredient, % as fed			
Ground Beef	-	-	99.9
Corn	83.05	70.59	-
Corn oil	-	15.00	
DDGS	10.00	8.50	-
Soybean Meal	5.00	4.25	-
Di-calcium phosphate	0.345	0.29	-
Calcium	0.995	0.85	0.1
Salt	0.45	0.38	-
Swine vitamin premix[2]	0.03	0.03	-
Swine mineral premix[3]	0.14	0.12	-
Proximate Analysis			
Dry Matter	89.30	90.53	51.03
Crude Protein	9.68	11.09	16.84
Lysine	0.45	0.54	1.78
Crude Fat	4.39	16.54	36.9
Ash	3.79	3.32	0.69
Acid Detergent Fiber	5.39	7.4	-
Neutral Detergent Fiber	18.74	17.3	-

[1]Low lysine diet
[2]Vitamin premix content: vitamin A, 10,000,000 IU/lb; vitamin D3, 1,500,000 IU/lb; vitamin E, 50,000 IU/lb; vitamin B_{12}, 40 mg/lb; menadione, 4,000 mg/lb; biotin, 155 mg/lb; folic acid, 1,000 mg/lb; niacin, 50,000 mg/lb; d-panthothenic acid, 30,000 mg/lb; vitamin B_6, 3,000 mg/lb; riboflavin, 9,000 mg/lb, and thiamine, 3,000 mg/lb.
[3]Mineral premix content: Copper, 1.1%; Iodine, 240 ppm; Iron, 11.0%; Zinc, 11.0%. Manganese, 2.9%; Selenium, 200 ppm.

The gilts on the *ad libitum* CON diet were provided 4.54 kg at 0800. If the CON rations were low at 1600 h, gilts were provided an additional 2.27 kgs to provide sufficient opportunity for *ad libitum* intake until the following morning. Orts were collected and weighed for both treatments prior to the 0800 feeding each day.

Ground Beef Preparation

The GB diet was prepared at the NDSU meat lab. Beef trimmings were obtained from a commercial meat processor (Long Prairie Packing Company, Long Prairie, MN). Upon arrival, trimmings were ground and the subsequent ground beef was spread evenly on 46 by 33 cm sheet pans and cooked until done (approximately twenty-five min) at 204 ^{o}C. After the ground beef was cooked, it was placed in the cooler (3° C) to chill. Allowing the product to chill ensured that the beef, fat, and juice were kept together in the pan. After the ground beef cooled for approximately 90 min, it was vacuum packaged for ease of storage. The cooked ground beef was then labeled with the date and weight, transported to ANPC, and then frozen until fed. Prior to use, ground beef packages were removed from the freezer the night before feeding to thaw. All ground beef was fed cold to maintain the consistency as the warm, soft fat appeared to be less palatable to the gilts.

Weight and Ultrasound Measurement Collection

Live body weights were obtained every 7 d from d0 (initiation of treatments). Back fat thickness and LMA were determined using an Aloka 500-SSD (Aloka America, Wallingford, CT) ultrasound machine every 28d. The same ultrasound technician was used throughout the duration of the study.

Feed intake was recorded daily. Orts were collected prior to the 0800 feeding and weighed. Prior to the 0800 feeding, all feeders were cleaned of their contents and new feed provided. Ground beef was provided to each gilt depending on their previous day's consumption. For example, if a gilt was provided 7 kg of GB on Monday and only consumed 4 kg, the following days amount would be decreased. That gilt would then be monitored at various times throughout the day to provide more GB if the allocation was consumed, thus allowing for *ad*

libitum intake to continue, yet decrease waste. The GB would be removed directly from the refrigerator prior to feeding, weighed, and provided to the gilts in the front pan of their feeder. Providing the diets in the back of their feeders did not allow for *ad libitum* intake as it would become stuck behind the paddle. The amount of feed weighed out was recorded on individual daily feed sheets and kept in chronological order to determine ADG and intake. The gilts on the GB treatment were fed first to ensure that they were eating what was provided. While the GB gilts were consuming their 0800 diets the CON diet feeders were cleaned. After feeder cleaning, each gilt was then provided with 4.54 kgs of CON diet. After initial feeding, if the majority of the GB in the feeders was gone, the GB pigs were provided an additional 1 to 1.4 kg of GB in order to simulate *ad libitum* intake, promote consumption, and ensure there was GB available for consumption. At feeding times, GB that was outside of the feeders (on slats) would be collected and weighed back as orts the following morning. This was done to minimize waste.

Calculated Estimate of Kcals Consumed

Calculated estimates of calories consumed are based on the standard acceptance that one gram of protein or carbohydrate contains four kilocalories (kcals) and one gram of fat contains nine kcals. Therefore, kcals consumed were calculated based on the proximate analysis of the GB and CON diets. Proximate analysis of CON diet was 69.05% carbohydrate, 11.09% crude protein, and 16.54% crude fat on an as-fed basis. Likewise, the GB diet had 0% carbohydrate, 16.84% crude protein, and 36.9% fat on an as-fed basis. Each percentage for the respective macro-nutrients was multiplied by grams of the total diet consumed and kcals consumed were calculated as the sum of four kcals per gram of crude protein and carbohydrate and nine kcals per gram of crude fat (Atwater and Bryant, 1899).

32

Statistical Analysis

      Body weight and subcutaneous back fat at the $10^{th}/11^{th}$ costae interface were collected on day –28, 0, 28, 56, and 84. A percentage change in BF relative to day 0 was calculated according to the following equations:

$$\text{Percent change BF on d28} = [(\text{d28 BF} - \text{d0 BF})/\text{d0 BF}]*100$$

$$\text{Percent change BF on d56} = [(\text{d56 BF} - \text{d0 BF})/\text{d0 BF}]*100$$

$$\text{Percent change BF on d84} = [(\text{d84 BF} - \text{d0 BF})/\text{d0 BF}]*100$$

      Similar equations were used to calculate percentage change in BW over time. These data collected over time were analyzed using generalized least squares (PROC MIXED, SAS Institute, Cary, NC) as repeated measures with the fixed effects of treatment, day, and treatment × day with pig ID serving as the repeated/subject variable.

      After 84 days on treatment, pigs were humanely euthanized and processed under USDA Food Safety and Inspection Service guidelines. Pigs were slaughtered according to the day that they went of test. Pigs were staggered in pairs across three different dates (four, two, and four pigs), depending on when they met the criteria for 3 cm of BF. Physical data were collected for off-test live weight, dressed carcass weight, subcutaneous fat depth adjacent the first, 10th, and last thoracic vertebra, and cross-sectional longissimus thoracis area (10th 11th costae interface). A limited necropsy was performed and weights were obtained for adrenal glands, heart, liver, pancreas, spleen, and peri-renal fat. Also, right and left ventricle thickness measurements (top, middle, and bottom) were collected. These data were analyzed using ordinary least squares (PROC GLM, SAS Institute, Cary, NC). The model included treatment, off test (harvest) day, and the treatment by day interaction. Means were separated using least significant difference.

Results and Discussion

Intake

On average, GB gilts consumed more kcals (Figure 2.1) than the CON group. Average

kcals consumed per day were significant for treatment ($P = 0.05$). For Weeks 3 to 12, the CON

averaged 14,007 kcals/d while the GB averaged16,557 kcals/d (Figure 2.2). These numbers

equivocates out to 18.21% more kcals consumed by the GB treatment than the CON treatment.

We hypothesize that it is not the amount of calories consumed, but rather the glycemic nature of

the diet consumed. Regular consumption of high-glycemic meals will cause blood glucose levels

to rise more rapidly than consumption of low-glycemic foods. Ludwig et al. (1999) observed an

increase in serum insulin concentrations after high GI meals compared to low and medium GI

meals that were consumed by obese teenage boys. This is believed to be due in part to the rapid

absorption of glucose into the system. In the same trial, plasma glucagon levels were suppressed

most likely due to the high plasma glucose and insulin concentrations in the system. The

decrease in consumption from week 6 to week 7 has been attributed to the change in GB batch.

Although not directly measured, after the first month on treatment, pigs on both treatments

seemed to become much more lethargic and acclimated to people. That said, GB pigs were

quicker to stand up when people entered the room and were easier to move to the scale. Further,

the GB gilts appeared to be more vigorous eaters who began consuming their allocation

immediately upon it being placed in the feeder pan. These behavioral observations were not

scientifically quantified and could be the focus of additional research. Another interesting note

related to the GB gilts was the appearance of oily skin. This appeared about 5 weeks after

beginning treatment. We are unsure of the mechanism behind this. One conclusion could be that

it was due to the high amounts of fat (36.9%; Table 2.1) that they were consuming or that

portions of the "grease" ended up on the pen slats. When the gilts laid on the slats, the grease

coated their bodies which ultimately collected more dust making the pigs appear darker and more
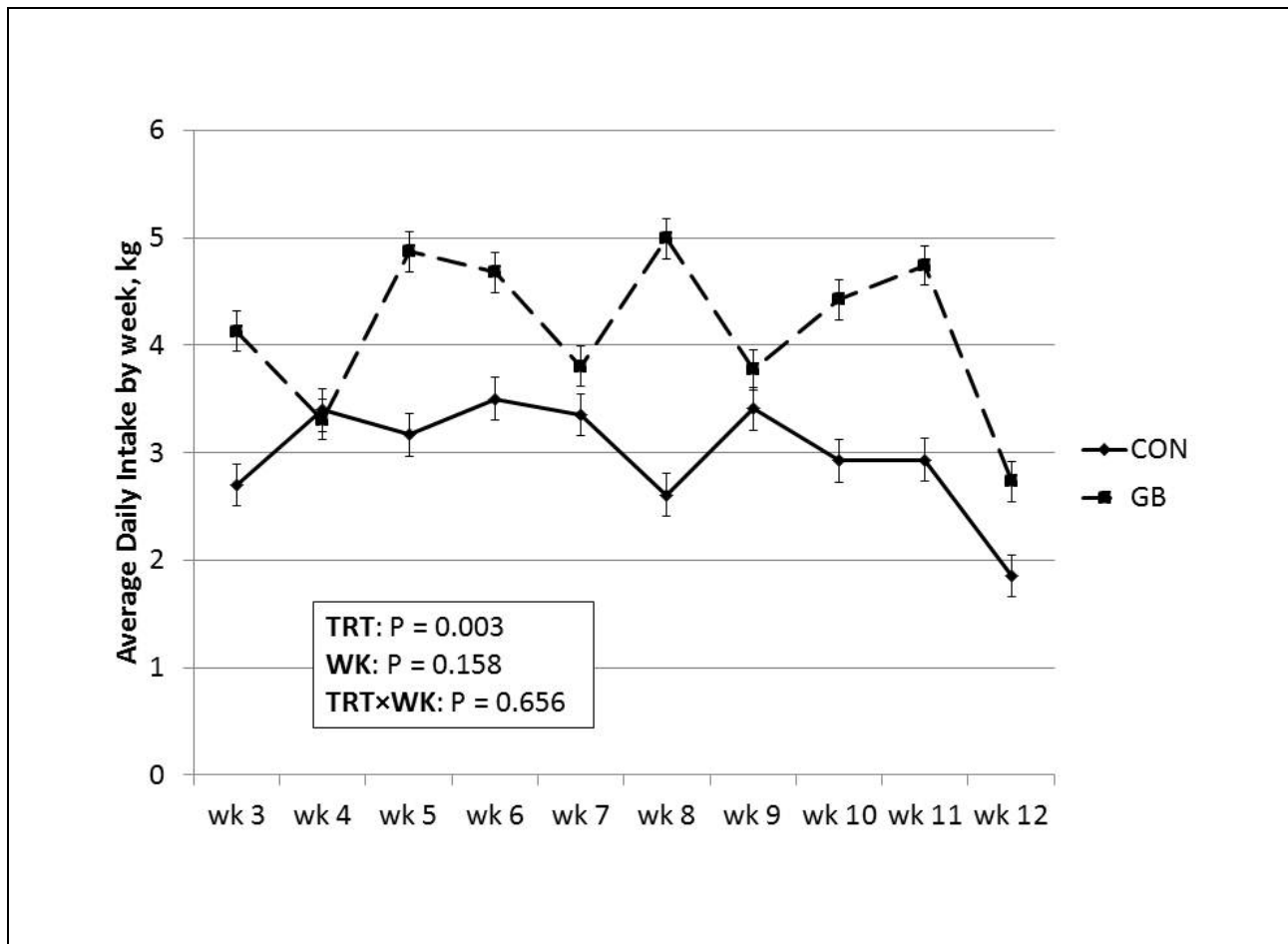
oily.

Figure 2.1. Average daily intake (kg) by week (wk) for gilts fed *ad libitum* corn-soybean control (CON) and 65:35 (lean:fat) blend ground beef (GB) dietary treatments (TRT) for 84 days (12 weeks).

Figure 2.2. Average daily caloric intake (calculated kcals[1]) by week (wk) for gilts fed *ad libitum* corn-soybean control (CON) and 65:35 (lean:fat) blend ground beef (GB) dietary treatments (TRT) for 84 days (12 weeks).
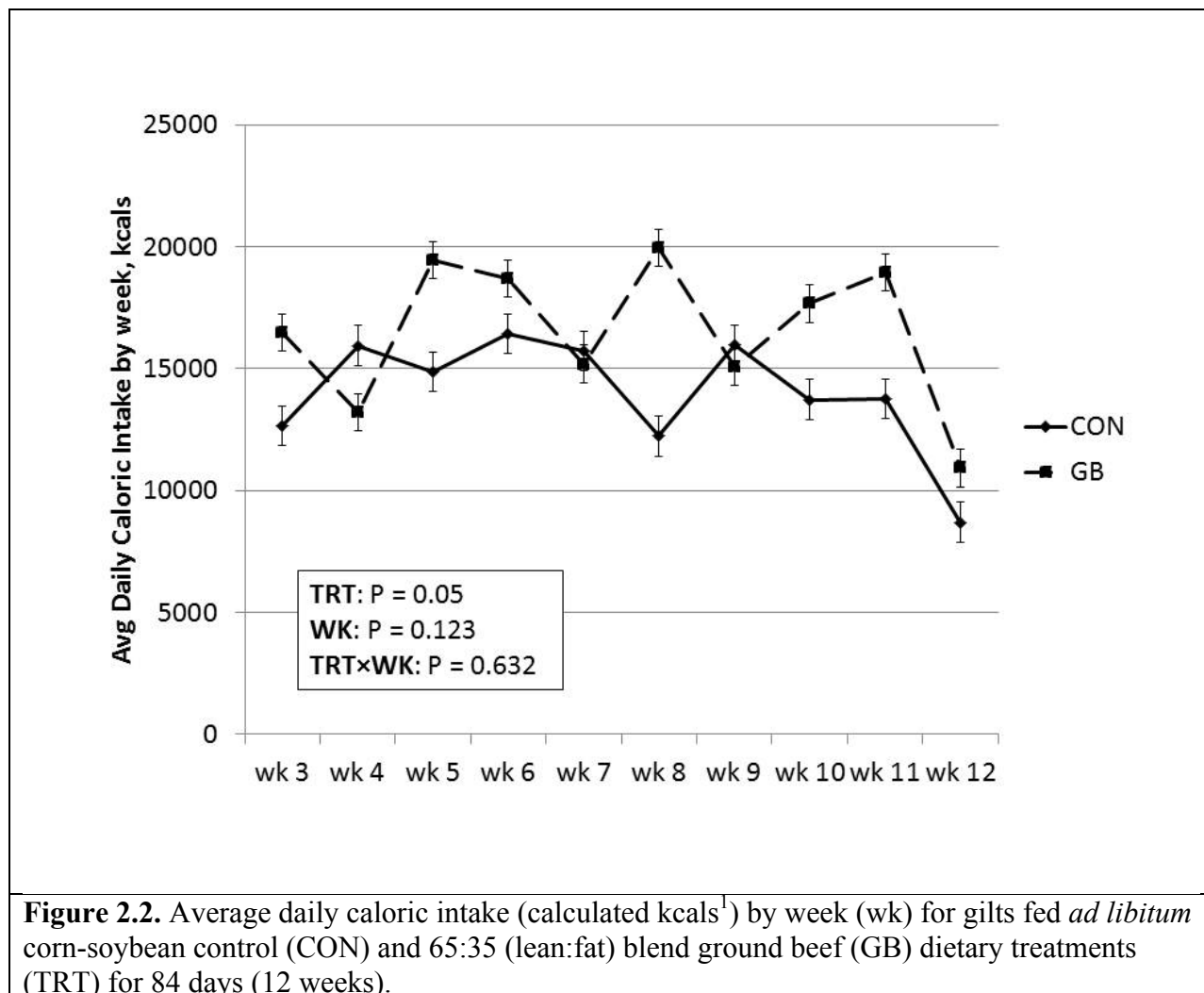
Growth and Development Characteristics

Gilts continued to gain weight (Day effect, $P < 0.001$) (Figure 2.3), with CON gaining an average of 0.77 kg/d and the GB gilts gained 0.61 kg/d, however BW was not affected ($P = 0.25$) by treatment (Figure 2.4). That said, CON gained significantly more BW ($P = 0.012$) when expressed as a percentage of weight gained from the start on test (d0; Figure 2.4).
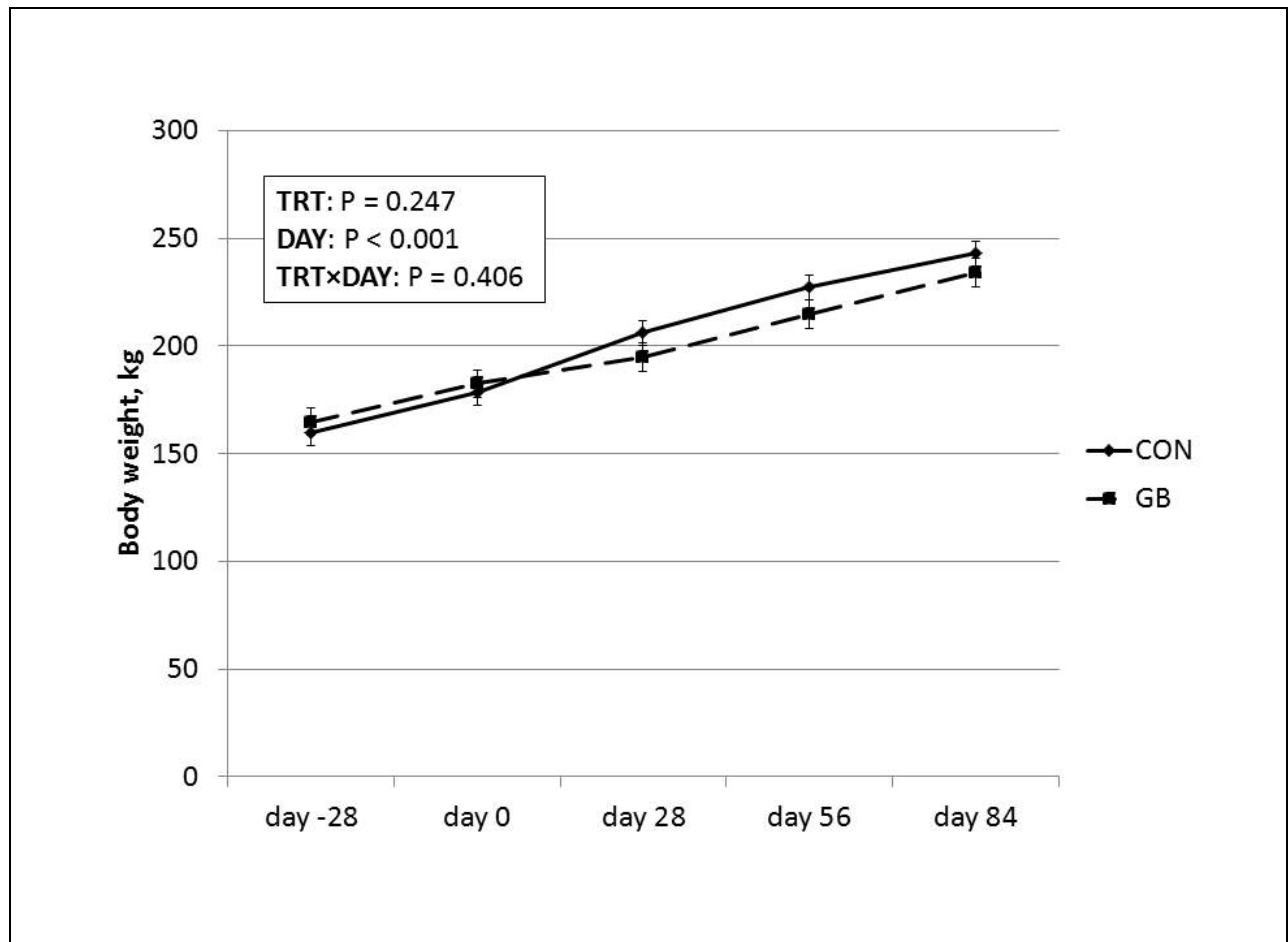
36

Figure 2.3. Body weight (kg) accumulation of gilts fed *ad libitum* corn-soybean control (CON) and 65:35 (lean:fat) blend ground beef (GB) dietary treatments (TRT) from day – 28 to day 84.
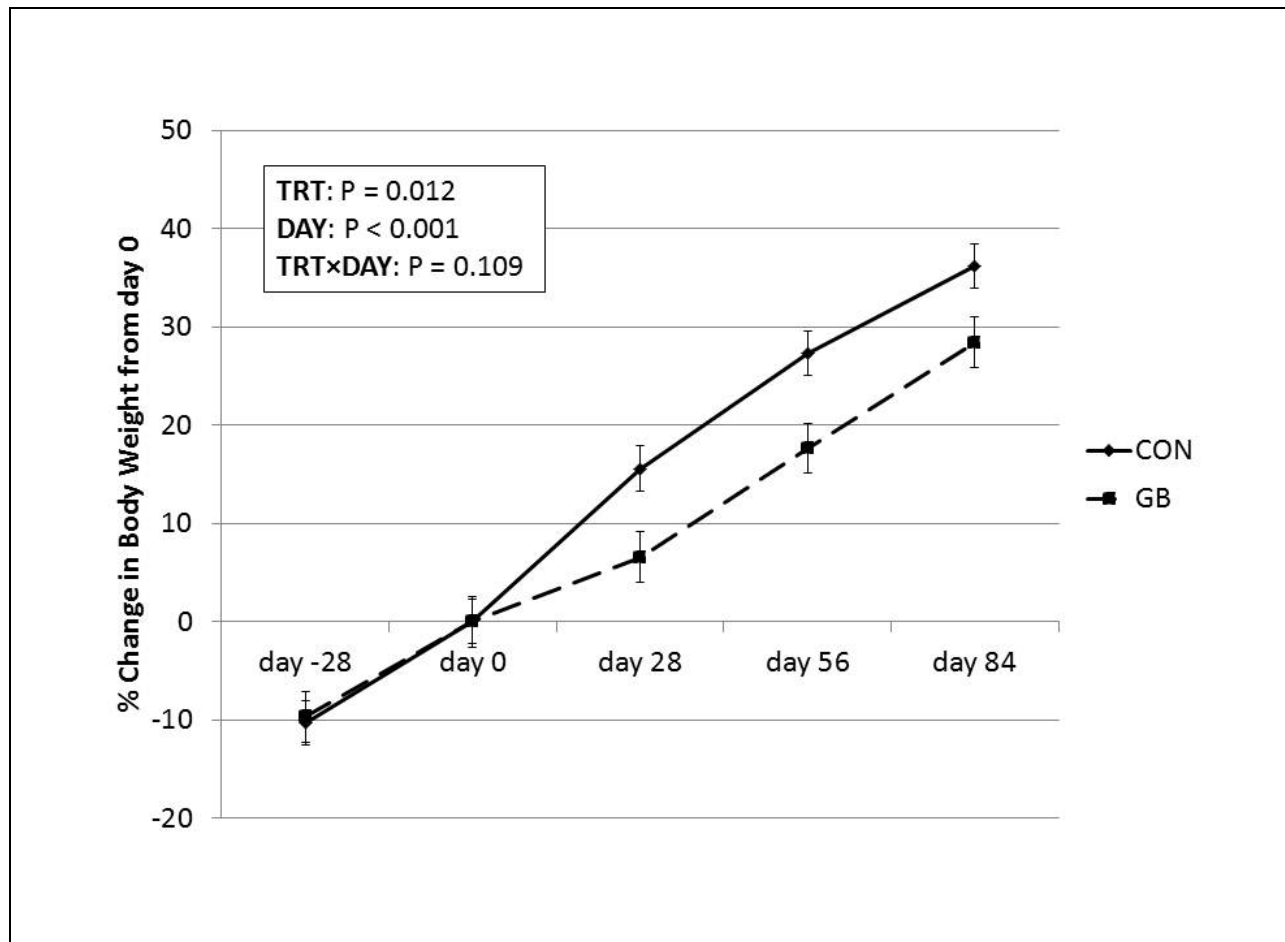
Figure 2.4. Body weight change expressed as the percentage change from day 0 (start on test) for gilts fed *ad libitum* corn-soybean control (CON) and 65:35 (lean:fat) ground beef (GB) dietary treatments (TRT).

Overall, subcutaneous backfat thickness was not affected ($P = 0.35$) by treatment (Figure 2.5). However, CON fed gilts tended ($P = 0.09$) to have a more rapid and greater accumulation of BF when expressed as a percentage change from the beginning of dietary treatment (d0; Figure 2.6). Gilts consuming the standard corn/soy finishing ration with 15% additional fat increased subcutaneous fat by an average of 12% over the same period of time while consuming fewer calories. There was an effect of day ($P = <0.001$) for percent change in subcutaneous

backfat thickness from day -28 to day 84 (Figure 2.6). Following the first 30 days, both groups of gilts continued to linearly increase their levels of backfat. Blouet et al. (2006) reported that rats provide a high-protein, low-carbohydrate diet showed decreased fat deposition along with improved oral glucose tolerance and insulin sensitivity compared to restricted normal protein or normal protein levels. This is similar to the results presented by Lacroix et al. (2004) that showed lower fat mass in rats provide a high protein low carbohydrate diet over a 6-mo period. Future research using tissues collected from the current project will evaluate leptin and ghrelin receptors in the hypothalamus of GB and CON gilts to provide insight regarding mechanisms driving the continued consumption even at levels exceeding the metabolic energy needs of the pig.
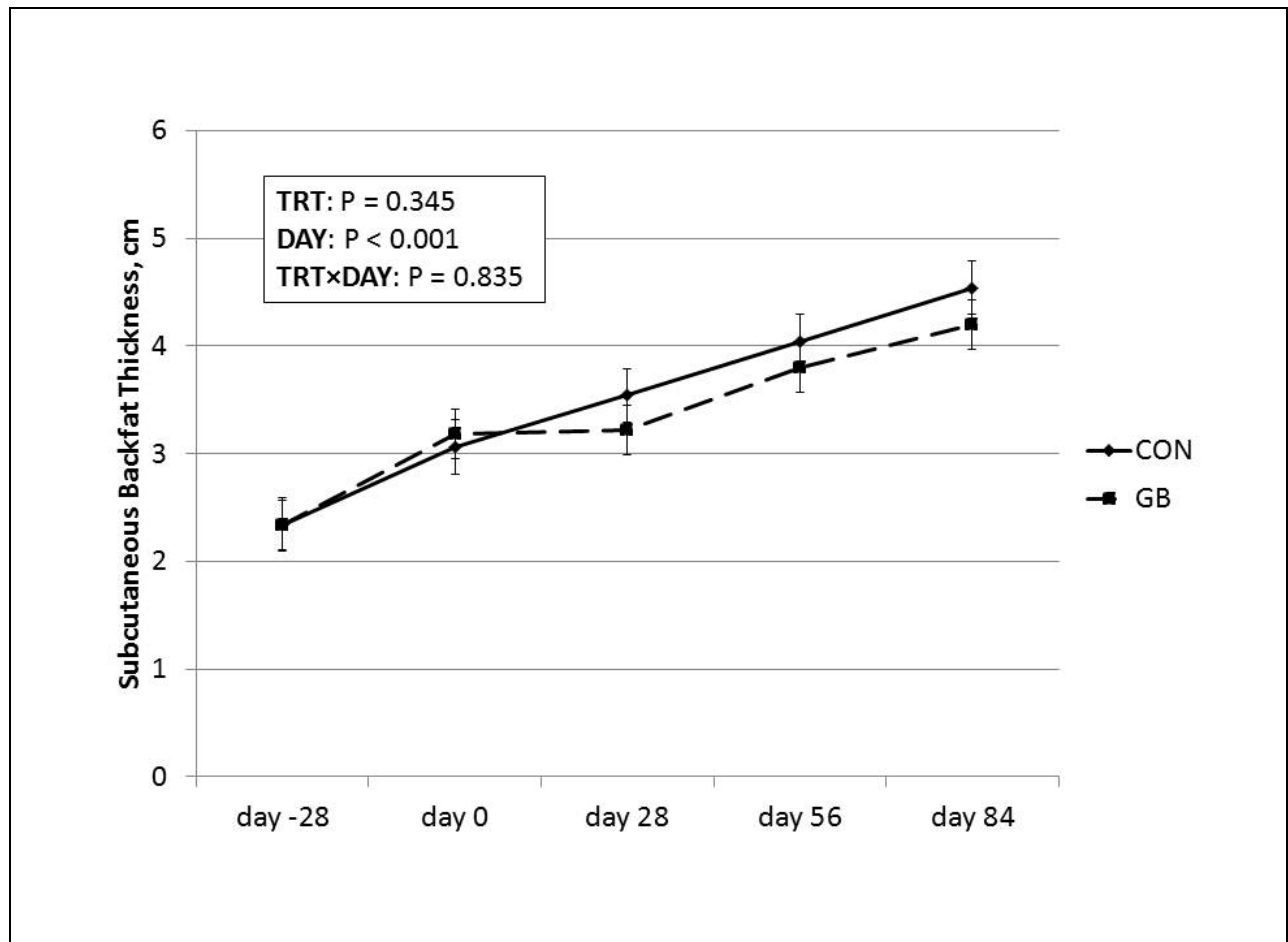
Figure 2.5. Subcutaneous backfat (cm) accumulation of gilts fed *ad libitum* corn-soybean control (CON) and 65:35 (lean:fat) blend ground beef (GB) dietary treatments (TRT) from day – 28 to day 84.
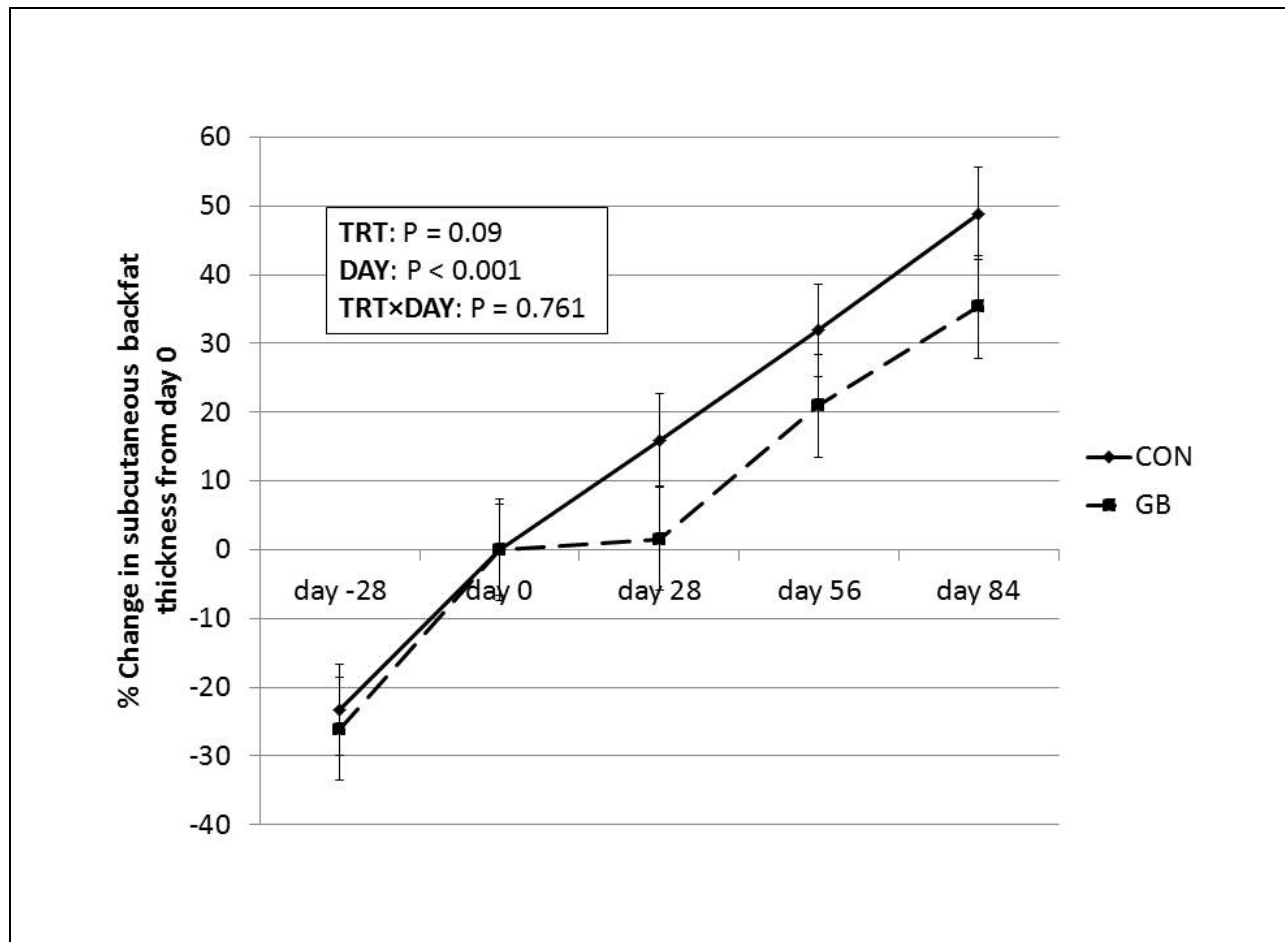
Figure 2.6. Subcutaneous backfat (cm) expressed as the percentage change from day 0 (start on test) for gilts fed *ad libitum* corn-soybean control (CON) and 65:35 (lean:fat) blend ground beef (GB) dietary treatments (TRT).

Final Body Composition

Final BW and carcass weight did not differ ($P \geq 0.52$). Furthermore, there was no impact of treatment on LMA, intramuscular fat, first rib fat depth, tenth rib fat depth, and last rib fat depth measurements. The calculated lean body mass (NPB, 2000) expressed as a percentage of carcass weight did not differ for CON (46.5%) vs. GB (47.1%). Gilt liver, adrenal glands, pancreas, heart, spleen and perirenal fat weights and weights expressed as percentage of final

BW did not differ ($P \geq 0.15$) by dietary treatment (Table 2.2). Gilt back fat measurements were not affected by ($P \geq 0.55$) dietary treatment nor were there differences in intramuscular fat (Table 2.2). We hypothesized that the CON gilts would deposit more backfat and presumably more intramuscular fat (a.k.a. marbling) than the GB gilts due to the large amount of carbohydrates being consumed. This would have been consistent with our hypothesis that over time hyperglycemia would lead to myofibril IR down-regulation while adipocytes would remain viable to insulin binding and subsequent insulin mediated adiposity. Witte et al., (2000) hypothesized that inadequate dietary lysine limited the synthesis of muscle-specific proteins and increases the amount of energy available for fat deposition. Similarly, Cisneros et al. (1996) observed that feeding lysine-deficient diets during the last five weeks of the finishing stage increased intramuscular fat content of pork. In the current study, the CON did contain adequate amounts of lysine for gilts of this size and stage of development.

Additionally, there was no effect on ventricular thickness (top, middle, or bottom sections) of the heart ($P = \geq 0.21$) in either treatment group. Even though both the right and left ventricle thickness's were not significant, the effects of left ventricle thickness play a larger role in the health of the animal. The left ventricle is more widely studied in both human and animal models because it provides blood flow to the entire body, compared to the right ventricle which only completes the pulmonary circulation system (Voelkel et al., 2006). Implications of this demonstrate that the cardiovascular health of the animals was not a factor affecting the overall health of the animals. Measuring ventricle wall thickness provides a predictor of the diastolic properties of the muscle indicating whether healthy diastolic pressures are present (Grossman et al., 1974). Seeing no significant difference in either treatment group reveals that the animals had similar blood pressures and delivery of nutrients to the body at the time of death. It is important

to note that it is a challenge to directly compare the findings in human research compared to animal models. The inability to compare findings is due to the abnormalities of cardiac structure and function more commonly present in animal models compared to humans affected by obesity. This may be due in part to the fact that the specific genetic abnormalities seen in many animal models are not observed in most humans with obesity (Abel et al., 2008).

CONCLUSION

Both groups of gilts increased adiposity. Gilts receiving the ground beef treatment consumed more feed and total calories than gilts fed the traditional corn/soybean meal ration. Despite consuming more total feed/food and more calories, the ground beef fed gilts gained less body weight and deposited less subcutaneous fat over the 84 days on test. Furthermore, there was no evidence of cardiac ventricular inflammation across treatments. More research is necessary involving the physiological response to food or combinations of food that expands beyond the standard human dietary advice of "consume fewer calories.

Table 2.2. Carcass Characteristics and organ weights of gilts fed CON or GB diet for 84 d.

Item	Treatment		SEM	*P*-value
	CON	GB		
Live Weight, kg	241.47	236.25	5.36	0.52
Hot Carcass Weight, kg	158.95	155.77	4.04	0.60
LMA, cm^2	69.68	67.53	3.00	0.63
Subcutaneous-fat, cm				
1st rib	6.23	6.53	0.64	0.75
10th rib	4.79	4.44	0.39	0.55
Last rib	4.40	4.37	0.45	0.96
Intramuscular fat*	2.32	2.80	0.04	0.66
Adrenals, g	8.54	10.03	0.87	0.29
Adrenal glands, % BW	0.003	0.004	0.0015	0.23
Heart, g	616.67	596.60	20.30	0.52
Heart, % BW	0.26	0.25	0.009	0.70
Left Ventricle				
Top, mm	26.41	26.50	0.81	0.94
Middle, mm	25.02	22.18	1.33	0.21
Bottom, mm	17.33	18.25	3.17	0.84
Right Ventricle				
Top, mm	12.02	11.08	1.36	0.64
Middle, mm	9.67	10.58	1.54	0.69
Bottom, mm	7.68	7.83	1.15	0.93
Peri-renal Fat, g	5.61	5.21	0.58	0.64
Peri-renal fat, % BW	2.32	2.15	0.22	0.61
Liver, g	2.11	2.24	0.12	0.46
Liver, % BW	0.88	0.95	0.04	0.25
Pancreas, g	216.67	231.67	6.09	0.16
Pancreas, % BW	0.09	0.10	0.002	0.15
Spleen, g	263.33	273.33	12.18	0.58
Spleen, % BW	0.11	0.12	0.007	0.47

*Intramuscular fat scores correspond to the visual estimate of the percentage of intramuscular lipid as viewed in the longissimus thoracis at the cut lean surface of 10/11th rib interface (NPB, 2000)

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46

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