

UNDERSTANDING THE EFFECTS OF EXERCISE ON GUT HORMONES &
OBESITY

By

© Matthew Miller

A thesis submitted to the
School of Graduate Studies
In partial fulfillment of the
Requirements for the degree of
Master's of Science (Kinesiology)

School of Human Kinetics and Recreation
Memorial University of Newfoundland

June, 2012

ABSTRACT

This study was designed to investigate the effects of a high-intensity circuit training exercise program on obese sedentary males, and circulating levels of pancreatic polypeptide. A total of 8 participants underwent an exercise intervention in which 12 exercise sessions over four weeks were completed. In each exercise session participants performed 7 resistance/body weight exercises in 3 circuits with as little rest as possible. Each week participants exercised 3 times and had blood work taken 2 times on alternating days. A dual-energy x-ray absorptiometry (DXA) scan was completed pre and post exercise program to measure body composition. The results indicated that the exercise program significantly changed the volume of work completed, resting heart rate, systolic blood pressure, body fat percentage, lean and fat tissue percentages, and trunk and leg fat percentages. There was no significant change in pancreatic polypeptide levels, diastolic blood pressure, time to completion, body mass, BMI, lean or fat body mass, or arm fat tissue. The results of this study indicate that 12 exercise sessions of a high-intensity circuit training exercise program are safe (for an otherwise healthy obese male) and effective in changing body composition, decreasing cardiovascular risk factors and increasing strength.

ACKNOWLEDGEMENTS

There are so many people that have helped and encouraged me along this journey. First and foremost, this Master's degree would never have been completed without my family. Their continued support, encouragement, unconditional love, and unwavering faith in my academic abilities gave me the motivation to complete this thesis. Without you, I would not be where I am today. Dr. Duane Button has been an ideal mentor, supervisor and friend. He has molded me as a student and individual and I am forever thank-full for his support. It is with the knowledge, guidance, and dedication from Dr. Button that I have succeeded. Andrew Baker has been a supportive friend and colleague throughout my time at MUN, without his friendship, and camaraderie I would never have finished this thesis.

The team of researchers I worked with during my data collection were amazing, Heather McCarthy, Gregory Pearcey, Shane Stratton, Steven Buckle, and Christy Nofall put in many hours of hard work and for this, I thank you. The faculty and staff in School of Human Kinetics and the team at Dr. Guang Sun's research lab provided me with the opportunity and resources to make this possible. Finally, to everyone I met here in Newfoundland, you have enriched my life and provided me with many good times that I will never forget.

Thank you.

"Old friends pass away, new friends appear. It is just like the days. An old day passes, a new day arrives. The important thing is to make it meaningful: a meaningful friend – or a meaningful day."

- Dalai Lama

TABLE OF CONTENTS

ABSTRACT.....	ii
ACKNOWLEDGEMENTS	iii
TABLE OF CONTENTS	iv
LIST OF TABLES.....	vi
LIST OF FIGURES.....	vii
LIST OF ABBREVIATIONS	viii
CHAPTER 1: INTRODUCTION.....	1-1
1.1 BACKGROUND OF STUDY	1-1
1.2 PURPOSE OF STUDY	1-3
1.3 SIGNIFICANCE OF STUDY	1-5
1.4 DEFINITIONS	1-6
1.5 REFERENCES.....	1-7
CHAPTER 2: REVIEW OF LITERATURE	2-1
2.1 INTRODUCTION	2-1
2.2 DEFINITION/MEASUREMENT OF OBESITY	2-3
2.3 OBESITY AND RISK FACTORS.....	2-5
2.4 OBESITY AND METABOLIC RATE.....	2-7
2.5 GUT HORMONES.....	2-9
2.6 PANCREATIC POLYPEPTIDE.....	2-11
2.7 EXERCISE	2-14
2.8 EFFECT OF EXERCISE TRAINING ON OBESITY	2-17
2.9 EFFECT OF EXERCISE TRAINING ON PANCREATIC POLYPEPTIDE	2-20
2.10 CONCLUSION	2-22
2.11 REFERENCES	2-24

CHAPTER 3: CO-AUTHORSHIP STATEMENT	3-1
---	------------

**CHAPTER 4: UNDERSTANDING THE EFFECTS OF
EXERCISE ON GUT HORMONES AND OBESITY 4-1**

4.1 ABSTRACT.....	4-2
4.2 INTRODUCTION.....	4-3
4.3 METHODS.....	4-7
4.4 RESULTS.....	4-14
4.5 DISCUSSION	4-17
4.6 REFERENCES.....	4-24
4.7 FIGURE LEGEND	4-27

LIST OF TABLES

Figure 4.1	Raw Data Values.....	4-29
------------	----------------------	------

LIST OF FIGURES

Figure 4.1	Timeline of Experimental Procedure	4-30
Figure 4.2 A-F:	Photos of Exercises Used in Experimental Procedure.....	4-31
Figure 4.3 A-D:	Exercise Intensity.....	4-37
Figure 4.4 A-B:	Change in Resting Heart Rate over 12 Exercise Sessions	4-38
Figure 4.5 A-B:	Change in Systolic and Diastolic Blood Pressure	4-38
Figure 4.6 A-D:	Body Mass, Body Fat, BMI, Lean vs Fat Mass.....	4-39
Figure 4.7 A-C:	Lean and Fat Tissue Percentages.....	4-40
Figure 4.8:	Pancreatic Polypeptide	4-41

LIST OF ABBREVIATIONS

BPM	Beats per minute
HR	Heart Rate
HRR	Heart Rate Reserve
DBP	Diastolic Blood Pressure
SBP	Systolic Blood Pressure
mmHg	Millimeters of Mercury
Pg/ml	Picograms per Milliliter
BMI	Body Mass Index
BF	Body Fat
HRmax	Heart Rate Maximum
RPE	Rate of Perceived Exertion
DXA	Dual-energy X-ray Absorptiometry
CHD	Cardiovascular Heart Disease
ADL	Activities of Daily Living
LDL	Low Density Lipoproteins
HDL	High Density Lipoproteins
BMR	Basal Metabolic Rate
RMR	Resting Metabolic Rate
EPOC	Excess Post-Exercise Oxygen Consumption
LHA	Lateral Hypothalamic Area
DVC	Dorsal Vagal Complex
PP	Pancreatic Polypeptide
PYY ¹⁻³⁶	Peptide Y-Y
CCK	Cholecystokinin
GIP	Glucose-Dependent Insulinotropic Polypeptide
GLP-1	Glucagon-Like Peptide 1
GLP-2	Glucagon-like Peptide 2
PHI/PHV	Peptide Histidine Isoleucine/Peptide Histidine Valine
HICT	High-intensity Circuit Training
TTC	Time to Completion
YOA	Years of Age
ELISAs	Enzyme-Linked Immunoabsorbent Assays
RIA	Radioimmuno-Assay

CHAPTER 1: INTRODUCTION

1.1 BACKGROUND OF STUDY

Obesity is becoming a worldwide health concern and the rate of obesity has increased significantly in the last 30 years. In Canada, the rate of obesity has tripled in the past few decades, and the most obese populations in Canada are located in the East coast (New Brunswick, Nova Scotia, Prince Edward Island, and Newfoundland & Labrador) (Durstine, Moore, Painter, & Roberts, 2009). There are a series of health impairments caused by obesity which include; hypertension, type II diabetes, heart disease, joint problems, breathing problems, liver malfunction, and some cancers (Anis, et al., 2010; Durstine, et al., 2009). Obesity not only affects the health of the individual, it also increases the health care costs associated with obesity because it contributes to, and causes other health impairments (Anis, et al., 2010). The cause of obesity is a complex interaction of several different factors, which include; poor diet, lack of physical activity, psychological and environmental mechanisms (i.e. socioeconomic status or mental disorders), as well as physiological mechanisms (i.e. hormone problems).

Appetite and satiety are two important physiological mechanisms that play a role in the development of obesity and these mechanisms are regulated by specific gastrointestinal hormones. Cholecystokinin (CCK), ghrelin, glucagon-like peptide 1 (GLP-1), glucagon-like peptide 2 (GLP-2), motilin, oxyntomodulin, pancreatic polypeptide, peptide YY (PYY₃₋₃₆), secretin, and somatostatin are the nine specific

hormones that directly regulate appetite and satiety (O. Chaudhri, Small, & Bloom, 2006; O. B. Chaudhri, Wynne, K., & Bloom, S. R., 2008). Appetite and satiety both contribute to how much a person eats and in obesity, the levels of these hormones found in the blood are altered (leading to an increase or decrease in appetite and a decrease in satiety) (Adrian, et al., 1976; Batterham, et al., 2003; O. Chaudhri, et al., 2006). The reason for this decrease is unknown.

Due to the rise of the incidence of obesity different ways to manage obesity have been investigated exercise has been shown to be a very effective way to manage obesity and its associated risk-factors (Durstine, et al., 2009). Supplementing gut hormones has been used as a method to manage obesity; however it is difficult and unrealistic (due to lack of availability and method of distribution) (Batterham, et al., 2003; O. Chaudhri, et al., 2006; Wynne, 2005). Although, exercise is a proven way to decrease weight and manage the associated risk factors of obesity, how exercise (and what type of exercise method) impacts gut hormones, and how this impact affects obesity is an area lacking research.

1.2 PURPOSE OF STUDY

Exercise intervention has been utilized in the management of many diseases and is also indicated in the management of obesity (Durstine & Moore, 2003; Martins, Kulseng, King, Holst, & Blundell, 2010; Martins, Morgan, Bloom, & Robertson, 2007). The proper method of exercise to utilize for obese individuals is debated, with some professionals prescribing aerobic exercise (due to its cardiovascular benefits, and increased in caloric expenditure during exercise bout), and others prescribing resistance training (due to its maintenance of lean muscle mass which contributes to an increase in caloric expenditure) (Gibala, Little, Macdonald, & Hawley, 2012; Jakicic, Marcus, Gallagher, Napolitano, & Lang, 2003; Martins, et al., 2010; Strasser & Schobersberger, 2011). To avoid losing any benefits of one type of training, a combination of resistance training and aerobic training may be more beneficial. Circuit training incorporates both resistance training and aerobic training in a high-intensity, short duration exercise bout. Exercises are completed one after another, with very little rest, which keeps the heart rate increased for the duration of the exercise (similar to aerobic exercise) while the exercises included are resistance exercises in nature (Gibala, et al., 2012; Jakicic, et al., 2003). Unfortunately, it is commonly thought that obese individuals not participate in high intensity training due to their 'unhealthy' state; there is no scientific evidence to support this claim.

The gut hormones that regulate appetite and satiety are important in the development and in the management of obesity so it is important to determine what hormones are most

important. Pancreatic polypeptide directly influences the higher brain centers to initiate satiety and decrease appetite (Batterham, et al., 2003; O. Chaudhri, et al., 2006; Martins, et al., 2007). Pancreatic polypeptide directly signals the brain (whereas other hormones send signals via second messengers) so it could play a more vital role in the control of appetite and satiety. There is no research to show the effect of resistance training on the levels of PP but these levels have been shown to increase after bouts of aerobic exercise (Martins, et al., 2010). Thus, the main purpose of this study was to investigate the effects of high-intensity resistance training exercise over a four week period on otherwise healthy obese individuals and the circulating levels of pancreatic polypeptide in these individuals.

1.3 SIGNIFICANCE OF STUDY

Obesity is an international concern and investigating any of the causes of and how to manage it is important. The role gut hormones play in obesity is clearly important, though, the mechanism that causes the circulating levels of these hormones to change in obesity are unknown. Future research conducted with gut hormones can shed some light into how and why obesity causes this physiological change

Exercise in particular has been shown to be an appropriate method to manage obesity and it's many associated risk factors. Further understanding of how exercise affects obesity is significant because it is cost effective, easy to implement, and can elicit positive benefits. Since the mode of exercise for obese individuals is often debated, it is important to determine what method of exercise will be the most beneficial. High-intensity circuit training may provide benefits of both aerobic and resistance training. Circuit training could potentially cause some significant effects in obese individuals as well as increase the levels of pancreatic polypeptide found in circulation (also adding to the benefits of exercise for obese individuals), these effects remain unknown.

1.4 DEFINITIONS

Obesity. The accumulation of excess body fat that often results in significant health impairments (Anis, et al., 2010).

Gut Hormones. A series of specific hormones that are released in the gastrointestinal system and regulate different aspects of the body.

Appetite. The desire to eat food. Often mistaken with hunger which is the physiological need to eat food.

Satiety. The feeling of fullness an individual feels after eating a meal.

Aerobic Exercise. A form of exercise that mainly utilizes the aerobic system of the body (i.e. running, cycling, brisk walking), or the continuous use of large muscle groups.

Resistance Exercise. A form of exercise training that involves strengthening muscles and utilizes the anaerobic system of the body (i.e. lifting weights, body weight exercises, plyometrics).

1.5 REFERENCES

- Adrian, T. E., Bloom, S. R., Bryant, M. G., Polak, J. M., Heitz, P. H., & Barnes, A. J. (1976). Distribution and release of human pancreatic polypeptide. *Gut*, 17(12), 940-944.
- Anis, A. H., Zhang, W., Bansback, N., Guh, D. P., Amarsi, Z., & Birmingham, C. L. (2010). Obesity and overweight in Canada: an updated cost-of-illness study. *Obesity Reviews*, 11(1), 31-40.
- Batterham, R. L., Le Roux, C. W., Cohen, M. A., Park, A. J., Ellis, S. M., Patterson, M., et al. (2003). Pancreatic polypeptide reduces appetite and food intake in humans. *J Clin Endocrinol Metab*, 88(8), 3989-3992.
- Chaudhri, O., Small, C., & Bloom, S. (2006). Gastrointestinal hormones regulating appetite. *Philos Trans R Soc Lond B Biol Sci*, 361(1471), 1187-1209.
- Chaudhri, O. B., Wynne, K., & Bloom, S. R. (2008). Can Gut Hormones Control Appetite and Prevent Obesity? [Review]. *Diabetes Care*, 31(2), 284-289.
- Durstine, J. L., & Moore, G. E. (2003). *Exercise Management for Persons with Chronic Disease and Disabilities* (Second ed.): Human Kinetics.
- Durstine, J. L., Moore, G. E., Painter, P. L., & Roberts, S. O. (2009). *Exercise Management for Persons With Chronic Diseases and Disabilities* (Third Edition ed.): Human Kinetics.
- Gibala, M. J., Little, J. P., Macdonald, M. J., & Hawley, J. A. (2012). Physiological adaptations to low-volume, high-intensity interval training in health and disease. *J Physiol*, 590(Pt 5), 1077-1084.
- Jakicic, J. M., Marcus, B. H., Gallagher, K. I., Napolitano, M., & Lang, W. (2003). Effect of exercise duration and intensity on weight loss in overweight, sedentary women: a randomized trial. *JAMA*, 290(10), 1323-1330.
- Martins, C., Kulseng, B., King, N. A., Holst, J. J., & Blundell, J. E. (2010). The effects of exercise-induced weight loss on appetite-related peptides and motivation to eat. *J Clin Endocrinol Metab*, 95(4), 1609-1616.
- Martins, C., Morgan, L. M., Bloom, S. R., & Robertson, M. D. (2007). Effects of exercise on gut peptides, energy intake and appetite. *J Endocrinol*, 193(2), 251-258.
- Strasser, B., & Schobersberger, W. (2011). Evidence for resistance training as a treatment therapy in obesity. *J Obes*, 2011, 9.
- Wynne, K., Stanley, S., McGowan, B., Bloom, S. (2005). Appetite Control. [Review]. *Journal of Endocrinology*(184), 291-318.

CHAPTER 2: REVIEW OF LITERATURE

2.1 INTRODUCTION

The level of obesity worldwide has increased substantially in the past 30 years, affecting over 300 million people and has become one of the largest world health concerns (Durstine, Moore, Painter, & Roberts, 2009). In Canada the rate of obesity has tripled in the past few decades, and the most obese populations in Canada are the citizens located in Eastern Canada (Nova Scotia, New Brunswick, Prince Edward Island, and Newfoundland & Labrador). Obesity is the accumulation of excess body fat that often results in significant health impairments (Durstine & Moore, 2003). There are many causes of obesity including hypothalamic, endocrine, and genetic disorders, however, poor diet and lack of physical activity are the leading causes (Durstine & Moore, 2003). Not only does obesity affect individual health, but it increases health care costs because it contributes to and causes various health impairments (Anis, et al., 2010). Some of these obesity-induced impairments include: hypertension, type 2 diabetes, heart disease, joint problems, some cancers, breathing problems, and liver malfunction (Fernandez-Sanchez, et al., 2011; Sizer, Whitney, & Piche, 2009). Each of these co-morbidities also has health risks. Thus, the prevention and management of obesity are very important for currently obese individuals and to those at risk of developing obesity.

The most effective way to manage obesity is often debated; however, exercise has been shown to be a very effective way to manage obesity and its associated co-morbidities (Durstine, et al., 2009). There are also physiological ways to manage obesity. Obesity can lower the levels of specific gastrointestinal hormones found in circulating blood which eventually leads to an increased appetite and decreased satiety in those who are obese. Although exercise is a proven way to decrease weight and manage the associated risk factors of obesity, the impact exercise has on gut hormones and how that effect translates into whether or not it will affect obesity is an area lacking research.

The purpose of the current literature review is to; 1) define obesity and associated risk factors, 2) investigate gut hormones associated with appetite and satiety, which directly influence obesity and, 3) investigate the effect exercise training has on obesity and gut hormones, and 4) examine how exercise plays an integral role in management of obesity as a whole.

2.2 DEFINITION/MEASUREMENT OF OBESITY

Obesity is defined by several different methods. One crude but commonly used method to define obesity is body mass index (BMI). A BMI of greater than 30 kg/m² indicates that someone is obese (Durstine, et al., 2009; Ehrman, 2009). However, BMI fails to take into account other factors that contribute to obesity (besides height and weight) such as muscle mass, bone mass, age, gender, and ethnicity (Kennedy, Shea, & Sun, 2009). Other methods of measuring body fat percent (BF%) have been proven to be more accurate, such as; dual-energy X-ray absorptiometry (DXA), air-displacement plethysmography, and underwater weighing (Dempster & Aitkens, 1995; Kennedy, et al., 2009; Salamone, et al., 2000; Sizer, et al., 2009). A study performed by Kennedy et al. (2009) compared BMI vs. DXA; it was found that over one-third of the males and females were misclassified by the BMI criteria when compared to DXA criteria (obese individuals were being classified as normal weight or overweight). Other research has found that BMI is not accurate in predicting adiposity status in normal to mildly overweight individuals, or in obese individuals (Adams, et al., 2007; Curtin, Morabia, Pichard, & Slosman, 1997; Romero-Corral, et al., 2008).

Due to the aforementioned obesity measurements, obesity has been categorically defined based on phenotype. Type 1 obesity is excess body mass or fat percentage, type 2 obesity is excess subcutaneous truncal-abdominal fat (android), type 3 obesity is excess abdominal visceral fat, and type 4 obesity is excess gluteal-femoral fat (gynoid) (Durstine & Moore, 2003). Performing a DXA scan, can distinguish between the android (typically

male) and gynoid (typically female) fat distributions. DXA is considered the gold standard for evaluating body composition (Kennedy, Shea, Sun, 2009). Future research on obese populations should be conducted with the DXA for the most reliable and accurate results.

2.3 OBESITY AND RISK FACTORS

Obesity has many associated co-morbidities including; stroke, gallbladder disease, osteoarthritis, sleep apnea, and at least eight different forms of cancer (Anis, et al., 2010). The two most prevalent co-morbidities are type II diabetes and hypertension (which leads to other cardiovascular diseases) (Anis, et al., 2010; Durstine, et al., 2009). Fat build up due to obesity can be distributed in different ways and in different areas. Subcutaneous fat is fat that lies between the skin and the internal organs, whereas visceral fat is fat that lies deep within the cavities of the body. Visceral fat is associated with more co-morbidities (compared to subcutaneous fat), and it is visceral fat that plays a role in metabolic disruptions because of its effects on insulin resistance and glucose intolerance (Ehrman, 2009).

Type II diabetes is characterized by increased insulin resistance, and often, individuals with type II diabetes are obese. Obesity contributes to the development of diabetes because it causes a decrease in insulin sensitivity (and diabetes is characterized by increased insulin resistance) (Durstine, et al., 2009). According to Fahey et al. (2010), obese individuals are at risk of increased cardiovascular heart disease (CHD) regardless of any other existing health problems (Fahey, 2010). Both CHD and Type II diabetes are significant co-morbidities associated with obesity, and thus, treating obesity can reduce the risks of these (and other) associated diseases (Church, 2011; Strasser & Schoberberger, 2011).

Managing obesity will not only reduce body weight but it will also serve as a secondary management tool for the associated health risks. Church (2011) found that a combination of resistance training and aerobic training is the best combination of exercise type (when compared to no exercise, or aerobic alone and resistance exercise alone) to decrease insulin resistance, indicating that exercise plays a role in managing diabetes. Not only has exercise been proven to manage diabetes but it has been shown to be an effective treatment for obese individuals since exercise improves cardiovascular function and capacity, systolic and diastolic blood pressure, insulin sensitivity, flexibility, muscular force production, improved performance of activities of daily living (ADL's) (de Deus, et al., 2011; Geliebter, et al., 1997) and an increased resting basal metabolic rate. Along with the above benefits of exercise, weight loss is the biggest benefit which not only contributes to the management of type II diabetes but a decrease in body weight can lead to an overall healthy lifestyle.

As reported by Haslam et al. (2006), a 10% weight loss is associated with a 10 mmHg decline in systolic and diastolic blood pressure, 40-60% decline in the incidence of diabetes, 10% decline in total cholesterol, and a 15% decline in LDL cholesterol, and a 20% decline in all causes of mortality (Haslam, Sattar, & Lean, 2006). With these proven benefits of weight loss, and using exercise as a tool to reduce weight, it is clear that further research should be completed investigating exercise as a management tool for obesity. Further discussion about obesity and exercise follows in a later section.

2.4 OBESITY AND METABOLIC RATE

Not only does increased fat cause the aforementioned co-morbidities, but it also contributes to decreased resting metabolic rate, and low fat free mass (or lean mass/muscle mass) is directly related to a lower metabolic rate as well (Wilmore, Costill, Kenney, 2008). A lower metabolic rate means that fewer calories will be expended at rest, leading to a lower chance of losing weight and an increased chance of gaining more weight (as opposed to an individual with a higher metabolic rate). Basal metabolic rate (BMR) is the amount of energy that a person uses while they are awake, lying down, fasted for 12 hours, and physically and mentally relaxed. Basal metabolic rate represents the energy requirements of the body in a resting state; it is estimated by measuring a person's rate of oxygen consumption (Stanfield, 2009). Generally, BMR will increase as body weight increases, and, muscle tissues have a higher resting metabolic rate (RMR) compared to adipose tissue (Stanfield, 2009). Having a low RMR is associated with having excess adipose tissue (and increased BMI) and a higher resistance to losing weight. Investigating the variables that contribute to RMR could prove to be useful treatments for obesity (Miller, 2012). For obese individuals, it is important to manage energy balance, and as stated above, muscle tissue and adipose tissue have different energy demands with muscles having a greater need for energy, thus an increase in caloric expenditure. Exercise can lead to increased muscle mass (or fat free mass) which will increase the body's need for energy, which in turn, will increase the RMR of an individual (Broeder, 1992; Wilmore et. al 2008). Exercise decreases body weight, and

increases muscle mass, subsequently increasing metabolic rate (i.e. increased caloric expenditure at rest).

Another aspect of exercise that plays a role in metabolic rate is excess post-exercise oxygen consumption (EPOC). EPOC is the body's increased metabolic need after work or exercise (Danduran, Dixon, & Rao, 2012). Thus, following a bout of exercise, the body's metabolic requirements and energy expenditure remains elevated (Mukaimoto & Ohno, 2012). The concept of EPOC could play a role in the management of obesity and should be further investigated. Along with the above discussion about obesity, the internal physiological mechanisms (such as hormones) that contribute to obesity are important to investigate. The next section will take an in-depth look at the role gut hormones play in obesity.

2.5 GUT HORMONES

There are a series of gastrointestinal hormones (or gut hormones) that monitor the physiological processes within the gastrointestinal tract. Cholecystokinin (CCK), gastrin, ghrelin, glucose-dependent insulintropic polypeptide (GIP), glucagon-like peptide 1 (GLP-1), glucagon-like peptide 2 (GLP-2), motilin, oxyntomodulin, peptide histidine isoleucine/peptide histidine valine (PHI/PHV), pancreatic polypeptide (PP), peptide YY (PYY₃₋₃₆), secretin and somatostatin are the major gut hormones (O. Chaudhri, Small, & Bloom, 2006). One hormonal process of particular importance to obesity is appetite regulation. The regulation of appetite plays a clear role in obesity, because when appetite is increased, the desire to consume food is increased. This increase in food consumption increases the amount of energy that enters the body and without expending energy, this excess energy can be stored as fat. In obese individuals, the levels of the hormones that regulate appetite are decreased, which means appetite will be increased (leading to increased food consumption) (Chaudhary, Kang, & Sandhu, 2010). The mechanism behind this decrease of gut hormones is unknown. There are nine hormones that specifically regulate appetite; CCK, ghrelin, GLP-1, GLP-2, motilin, oxyntomodulin, PP, PYY₃₋₃₆, secretin and somatostatin.

The endocrine system and the nervous system work together to coordinate the functions of all the systems in the body, more specifically the nervous system uses neurotransmitters to send signals through the body and the endocrine system secretes hormones which are transported through the blood stream to regulate these functions. The

pancreas is one of the main organs in the secretion and uptake of hormones (Tortora, 2009). From the major gut hormones listed above; CCK, ghrelin, GLP-1, GLP-2, motilin, oxyntomodulin, PP, PYY₃₋₃₆, secretin and somatostatin are the hormones that specifically regulate appetite and satiety.

Of these hormones, only PP is secreted directly from the pancreas (O. Chaudhri, et al., 2006; Wynne, 2005). Three hormones released from the gut that are responsible for secretion of PP are ghrelin, motilin, and secretin, while somatostatin is responsible for the inhibition of PP (Parkinson, et al., 2002). It has been shown that leptin (a very commonly studied hormone that regulates food intake and obesity) does not regulate levels of PP (Arosio, et al., 2003; Hwang, Chan, Ntali, Malkova, & Mantzoros, 2008; Mochiki, Inui, Satoh, Mizumoto, & Itoh, 1997; Peracchi, Tagliabue, Quatrini, & Reschini, 1999).

2.6 PANCREATIC POLYPEPTIDE

Pancreatic polypeptide shares a very similar molecular make-up to PYY and both hormones serve similar roles. It has even been suggested that PP was produced as a clone of PYY (Batterham, et al., 2003). Pancreatic polypeptide is released specifically from the periphery (pancreatic F-cells) of the islets on the pancreas which makes it different from PYY (which is secreted from the L-cells of the small and large intestines) (Baynes, Dhillo, & Bloom, 2006; O. Chaudhri, et al., 2006). The release of PP is directly dependent on the number of calories consumed (there is a positive linear relationship between PP and calories consumed) (Adrian, et al., 1976; O. Chaudhri, Wynne, & Bloom, 2008; Tortora, 2009). Once PP is released into circulation, it circulates through the blood stream and binds to the Y4-receptor in the hypothalamus. Once PP has connected to the Y4-receptor, the CNS sends signals (via the vagal nerve) to the gut to inhibit gastric emptying and increase satiety and decrease appetite (Batterham, et al., 2003; O. Chaudhri, et al., 2006; Martins, Morgan, & Truby, 2008).

The hypothalamus houses specific neurons in the lateral hypothalamic area (LHA) that are known for appetite and satiety regulation (Larhammar, 1996; Sainsbury, et al., 2010). Pancreatic polypeptide is the only gut hormone that has a direct effect on the dorsal vagal complex (DVC) in the brain (both directly, and by stimulating the vagus nerve) in the fed state (Gardiner, Jayasena, & Bloom, 2008). This is of significance because the DVC directly triggers the higher brain centers to cause satiety; other hormones enter the arcuate nucleus which contains a series of second messenger systems.

This direct effect of PP on the DVC could indicate that PP signals satiety faster than other gut hormones (Gardiner, et al., 2008).

Food intake can directly affect the levels of PP or affect these levels indirectly through various disorders (i.e. obesity, anorexia, or Prader-Willi Syndrome) (O. Chaudhri, et al., 2006; O. Chaudhri, Wynne, & Bloom, 2008; Wynne, 2005). In obesity, the levels of PP are reduced which results in an increase in eating, whereas in anorexia, the levels of PP are increased. The mechanisms behind these changes in PP are yet to be discovered (Batterham, et al., 2003; Fujimoto, et al., 1997; Wynne, 2005). Since PP directly affects gastric emptying and appetite, it is important to investigate what role it plays in obesity. What is known is that there is a direct relationship between pancreatic polypeptide and obesity. (Glaser, Zoghlin, Pienta, & Vinik, 1988; Wynne, 2005; Zipf, O'Dorisio, Cataland, & Sotos, 1981).

Pancreatic polypeptide supplementation could be a potential therapy for obesity especially due to how long it stays in circulation (6-24 hours) (Batterham, et al., 2003; O. Chaudhri, et al., 2006; O. Chaudhri, Wynne, & Bloom, 2008; Wynne, 2005). Perhaps, combining PP supplementation with exercise could extend the length at which PP will stay in circulation to have prolonged effects on the feeling of satiety in obese individuals and a decrease in appetite for extended periods of time, which would potentially lead to more chronic weight loss (Martins, Morgan, Bloom, & Robertson, 2007). Pancreatic polypeptide serves a vital role in the gastrointestinal tract and the regulation of appetite

and gastric emptying. It is apparent that PP plays a role in obesity; however, this role still needs to be further investigated.

2.7 EXERCISE

Exercise can take many forms to suit various fitness goals; however the two most common forms of exercise are resistance training and aerobic training. Often, individuals will combine both common types. Although most exercise programs include a combination of resistance and aerobic training, there are advantages to both types of training. Typically aerobic training increases cardiovascular function, improves systolic and diastolic blood pressure, decreases resting heart rate, total cholesterol and triglyceride levels, as well as further benefits when combined with diet. However, aerobic exercise (running, biking, walking) does not maintain fat-free mass when compared to resistance training (Geliebter, et al., 1997). Resistance training (lifting weights)) can provide both healthy individuals and those with chronic diseases with; increased muscular force production, muscle endurance, flexibility, improved performance in activities of daily living, a decrease in resting heart rate and systolic blood pressure, as well as small increases in cardiovascular capacity (de Deus, et al., 2011). An exercise training program which combines both aerobic and resistance training (a program that includes an extended period of increased heart rate, as well as the inclusion of resistance exercises) may provide greater benefits for any individual trying to increase their fitness levels. Such an exercise program may be considered high intensity. One advantage of high intensity resistance training is a large volume of work can be completed within a short period of time. With the rest periods limited, the participants' heart rate will remain at an

increased level, which mimics an aerobic-type exercise program, while the exercises completed are resistance-training exercises.

One type of high intensity exercise program that combines both aerobic and resistance training and induces a prolonged increase in heart rate is called circuit training. Circuit training programs are often utilized to incorporate cardiovascular stimuli during a resistance training program. Circuit training typically incorporates 8-12 exercises while performing 10 to 15 repetitions of each exercise. The repetitions are performed within 30 to 60 seconds with 30 to 45 seconds rest between sets (LaFontaine, 1997). Circuit training (or a modification of circuit training) may be a more effective way to provide the combination of benefits of aerobic and resistance training. Research has shown that strength gains will occur in a circuit training program, but the cardiovascular benefits are only modest to non-existent (Gettman, et al., 1976; Hurley, et al., 1984). One important consideration when utilizing circuit training is the rest periods. Research completed by Castinheiras-Neto et al. (2010) found that if the rest interval (RI) (the amount of time spent resting between sets of work), is increased (i.e. more rest given between sets) in resistance training exercise sessions, the cardiovascular effect will be decreased. In other words, the more time spent resting in a resistance training program, there is a decreased chance of gaining cardiovascular benefits from this form of exercise. This suggests that when performing a resistance exercise program, or circuit training program, a short rest period is crucial in promoting cardiovascular benefits (Castinheiras-Neto, 2010).

Another important variable to consider when performing circuit training is intensity. It has been suggested that any physiological benefits gained from exercise training are mainly dependent on the intensity at which the individual exercises (Hofmann & Tschakert, 2011). The intensity of aerobic exercise should be above 64-70% of the individual's maximum heart rate (HRmax) and no higher than 94% of HRmax (*ACSM's Guidelines for Exercise Testing and Prescription*, 2006). Exercising at a higher intensity (i.e. closer to HRmax) has been shown to provide more benefits (i.e. increased cardiovascular responses, increased caloric expenditure, easier completion of ADL's etc.) when compared to exercise at a lower intensity while performing the same volume of work (Duncan, 2005; Lee & Paffenbarger, 2000). This suggests that regardless of training volume, higher workout intensity will yield more health benefits. Thus, a combination of both high intensity and small rest periods may be the key components to circuit training and provide an individual with the greatest physiological benefits. Furthermore, the above-cited research was based on normal weight and relatively healthy individuals. Specific recommendations and research on circuit training for obese individuals is lacking.

2.8 EFFECT OF EXERCISE TRAINING ON OBESITY

Exercise has been suggested as a treatment method for obesity due to its increase in caloric expenditure (Martins, Kulseng, King, Holst, & Blundell, 2010; Martins, Morgan, & Truby, 2008; Martins, Morgan, et al., 2007). In obese individuals, exercise training is important in creating a negative energy balance (energy out > energy in) which is effective in managing weight. Thus, exercise prescription should be highly considered when developing a management program for obesity (Fahey, 2010). A combination of nutrition and exercise are the most effective way to create a negative energy balance (Broeder, 1992). Aerobic exercise and resistance exercise both provide health benefits that can affect body weight and change body composition. However, resistance training can maintain and increase lean body mass, subsequently resulting in an increase in RMR, muscle mass and strength, as well as daily functioning, whereas aerobic exercise has mainly cardiovascular effects (Miller, 2012; Stanfield, 2009).

Little research exists about the most effective type of exercise to help manage obesity, and the research that does exist is conflicting. Some research suggests that aerobic exercise is better for and should be implemented with the obese population due to its cardiovascular benefits (Chaudhary, et al., 2010), whereas other research suggests that implementing an exercise program with obese individuals must include resistance training due to its maintenance of fat free mass (i.e. muscle tissue) which contributes to a increased metabolic rate and fat loss (McQueen, 2009). A 2011 review suggests that resistance training is as effective as aerobic training in reducing major cardiovascular

disease risk factors, improving body composition and maintaining reduced fat mass (Strasser & Schobersberger, 2011). It was also concluded that resistance training mobilizes visceral and subcutaneous fat in the abdominal region when compared to aerobic training (Strasser & Schobersberger, 2011). Perhaps to avoid losing any benefits from either resistance training or aerobic training, a combination of both would be the most beneficial to manage the many aspects of obesity. As discussed above (for normal weight individuals) circuit training is an effective way to combine both resistance training and aerobic training, and it could be indicated as an appropriate method to manage obesity. During circuit training, the short rest periods and high intensity work promotes cardiovascular, strength and increased muscle mass benefits, which will promote weight loss.

The intensity and type of exercise that is most effective in treating obesity is still debated. Jakicic et al. (2003) performed a study investigating the most effective intensity and duration to increase weight loss and maintain that weight loss over 12 months. The findings were inconclusive, exercising at a moderate intensity (40-59% heart rate reserve (HRR)) and moderate duration, showed the most weight loss (numerically) over 12 months, but this particular intensity and duration was not significantly different between other intensities and durations (vigorous-intensity (over 60% HRR) - moderate duration, moderate-intensity – high duration, vigorous intensity – high duration) (Jakicic, Marcus, Gallagher, Napolitano, & Lang, 2003). Another research study completed by Gibala (2012) showed that high intensity training (HIT; i.e. short bouts of intense all out maximal effort) on a bicycle improved cardiorespiratory fitness in the obese population,

and the improvements to cardiorespiratory health post-HIT were far better than the improvements post-endurance training (Gibala, Little, Macdonald, & Hawley, 2012).

Unfortunately, it is commonly thought that otherwise healthy obese individuals should not be prescribed high intensity exercise programs due to their 'unhealthy' state; however there is no scientific research to support this assumption. Perhaps high intensity circuit training, which has resistance exercise and aerobic components, would be more effective for managing obesity (de Deus, et al., 2011).

2.9 EFFECT OF EXERCISE TRAINING ON PANCREATIC POLYPEPTIDE

Aerobic exercise has been shown to increase the levels of circulating pancreatic polypeptide during and following an exercise bout in obese sedentary individuals, however, no research has been done investigating the effects of resistance training on PP (Martins, et al., 2010; Martins, Morgan, et al., 2007; Martins, Truby, & Morgan, 2007). Martins et al. (2007) investigated the effect of acute exercise on circulating levels of PP. Subjects were asked to have blood taken every 30 minutes for four hours. After the first blood draw they were given a meal and asked to perform aerobic exercise one hour later. After an hour of exercise, an hour of rest was given before a buffet meal was provided. The levels of PP increased after the meal (indicating a decrease in appetite and increase in satiety) and these levels increased even further after exercise (indicating an increased hormonal response due to exercise) (Martins, Morgan, et al., 2007). These findings suggest that there is a direct effect of exercise on the levels of PP in the blood; however this study did not investigate the long term effects of exercise on PP. Perhaps exercise would provide a longer term increase in PP levels, if it was performed consistently and over a longer period of time.

Although no research has been performed investigating the effects of resistance training on PP, a study conducted by Broom et al. (2009) demonstrated the effects of resistance training on ghrelin and PYY. It was found that hunger was suppressed during and shortly after resistance training, acetylated ghrelin was suppressed during resistance exercise, and PYY was non-responsive (Broom, Batterham, King, & Stensel, 2009). Due

to the similarities between PYY and PP (as discussed above) a similar response to resistance training could be seen in PP.

A recent review suggests that there is a lack of research on how exercise and gut hormones interact with, and affect appetite (Horner, Byrne, Cleghorn, Naslund, & King, 2011). More specifically, there is limited (if any) evidence of relationship between resistance training and circulating levels of pancreatic polypeptide which leaves room for research to be conducted in this area.

2.10 CONCLUSION

Obesity is a developing concern for the health of many individuals in the developed world. Along with the risks of obesity, there are associated co-morbidities that contribute to obesity being an extremely dangerous disease. BMI has been used to define obesity for years; however, another more accurate way to provide this definition is a DXA scan. DXA measures body fat and is considered the gold standard for defining obesity. There are various causes of obesity and the management of obesity is complex. Among these causes, are the internal gastrointestinal hormones that regulate appetite and satiety. In obesity, these hormones are decreased which causes an increase in appetite and a decrease in satiety which can lead to over eating. The reason for this decrease in hormone levels is unknown, but could shed some light on some important factors in the development of obesity.

Exercise helps treat obesity by decreasing body weight, increasing muscle mass, and reducing the effects of co-morbidities. The mode of exercise that is most beneficial for obese individuals can be debated. Aerobic training has been proven to increase caloric expenditure, affect body composition, and provide increased cardiovascular functioning; however aerobic exercise does not maintain muscle mass which is an important factor in prolonged caloric deficit. Resistance exercise maintains muscle mass however it does not provide as many cardiovascular benefits. A combination of resistance training and aerobic training may provide the largest spectrum of health benefits for obese individuals, and this could be obtained through high intensity circuit training. High intensity circuit

training provides the benefits of aerobic exercise by maintaining an increased heart rate, and also includes the benefits of resistance training by building muscle and maintaining fat free mass.

Exercise also affects gut hormones that control appetite and satiety which are important physiological mechanisms in obesity. It is proven that aerobic exercise will increase the acute (< 24 hours) levels of specific gut hormones in the blood which will decrease appetite and increase satiety, however, the effect that resistance training has on gut hormones, and specifically pancreatic polypeptide is unknown. The effect resistance exercise has on pancreatic polypeptide is important because PP directly sends signals to the brain centers in control of appetite and satiety, so investigating this hormone and how exercise affects the levels found in the blood stream has important implications in the treatment of obesity.

As the incidence of obesity is rapidly increasing the need for more research about the possible management techniques for obesity is increasingly important as well. Pancreatic polypeptide and other gut hormones are important in the regulation of appetite and satiety and the effect exercise has on each could have serious implications in the implementation of exercise in every treatment program for obesity.

2.11 REFERENCES

- Adams, T. D., Heath, E. M., LaMonte, M. J., Gress, R. E., Pendleton, R., Strong, M., et al. (2007). The relationship between body mass index and per cent body fat in the severely obese. *Diabetes Obesity & Metabolism*, 9(4), 498-505.
- Adrian, T. E., Bloom, S. R., Bryant, M. G., Polak, J. M., Heitz, P. H., & Barnes, A. J. (1976). Distribution and release of human pancreatic polypeptide. *Gut*, 17(12), 940-944.
- Anis, A. H., Zhang, W., Bansback, N., Guh, D. P., Amarsi, Z., & Birmingham, C. L. (2010). Obesity and overweight in Canada: an updated cost-of-illness study. *Obesity Reviews*, 11(1), 31-40.
- Arosio, M., Ronchi, C. L., Gebbia, C., Cappiello, V., Beck-Peccoz, P., & Peracchi, M. (2003). Stimulatory effects of ghrelin on circulating somatostatin and pancreatic polypeptide levels. *J Clin Endocrinol Metab*, 88(2), 701-704.
- Batterham, R. L., Le Roux, C. W., Cohen, M. A., Park, A. J., Ellis, S. M., Patterson, M., et al. (2003). Pancreatic polypeptide reduces appetite and food intake in humans. *J Clin Endocrinol Metab*, 88(8), 3989-3992.
- Baynes, K. C., Dhillon, W. S., & Bloom, S. R. (2006). Regulation of food intake by gastrointestinal hormones. *Curr Opin Gastroenterol*, 22(6), 626-631.
- Broeder, C. E., Burrhus, K. A., Svanevik, L. S., and Wilmore, J. H. (1992). The effects of either high-intensity resistance or endurance training on resting metabolic rate. *Am J Clin Nutr*, 80(10), 802-810.
- Broom, D. R., Batterham, R. L., King, J. A., & Stensel, D. J. (2009). Influence of resistance and aerobic exercise on hunger, circulating levels of acylated ghrelin, and peptide YY in healthy males. *American Journal of Physiology-Regulatory Integrative and Comparative Physiology*, 296(1), R29-R35.
- Castinheiras-Neto, A. G., Rodrigues da Costa-Filho, L., and, Farinatti, P. T. V. (2010). Cardiovascular Responses to Resistance Exercise are Affected by Workload and Intervals Between Sets. *Sociedade Brasileira De Cardiologia*, 95(4), 493-501.
- Chaudhary, S., Kang, M. K., & Sandhu, J. S. (2010). The Effects of Aerobic Versus Resistance Training on Cardiovascular Fitness in Obese Sedentary Volunteers. *Asian Journal of Sports Medicine*, 1(4), 177-184.
- Chaudhri, O., Small, C., & Bloom, S. (2006). Gastrointestinal hormones regulating appetite. *Physiol Trans R Soc Lond B Biol Sci*, 361(1471), 1187-1209.
- Chaudhri, O. B., Wynne, K., & Bloom, S. R. (2008). Can Gut Hormones Control Appetite and Prevent Obesity? [Review]. *Diabetes Care*, 31(2), 284-289.
- Church, T. (2011). Exercise in obesity, metabolic syndrome, and diabetes. *Prog Cardiovasc Dis*, 53(6), 412-418.
- Curtin, F., Morabia, A., Pichard, C., & Slosman, D. O. (1997). Body mass index compared to dual-energy x-ray absorptiometry: Evidence for a spectrum bias. *Journal of Clinical Epidemiology*, 50(7), 837-843.
- Danduran, M. J., Dixon, J. E., & Rao, R. P. (2012). Near infrared spectroscopy describes physiologic payback associated with excess postexercise oxygen consumption in

- healthy controls and children with complex congenital heart disease. *Pediatr Cardiol*, 33(1), 95-102.
- de Deus, A. P., de Oliveira, C. R., Simoes, R. P., Baldissera, V., da Silva, C. A., Orsini Rossi, B. R., et al. (2011). Metabolic and Cardiac Autonomic Effects of High-Intensity Resistance Training Protocol in Wistar Rats. *J Strength Cond Res*.
- Dempster, P., & Aitkens, S. (1995). A new air displacement method for the determination of human body composition. *Med Sci Sports Exerc*, 27(12), 1692-1697.
- Duncan, G. E., Anton, S. D., Sydeaman, S. J., Newton R.L, Corsica, J. A., Durning, P. E., Ketterson, T. U., Martin A. D., Limacher, M. C., and Perri, M. G. (2005). Prescribing exercise at varied levels of intensity and frequency: a randomized trial. *Arch Intern Med*, 165(20), 2362-2369.
- Durstine, J. L., & Moore, G. E. (2003). *Exercise Management for Persons with Chronic Disease and Disabilities* (Second ed.): Human Kinetics.
- Durstine, J. L., Moore, G. E., Painter, P. L., & Roberts, S. O. (2009). *Exercise Management for Persons With Chronic Diseases and Disabilities* (Third Edition ed.): Human Kinetics.
- Ehrman, J. K., Gordon, P. M., Visich, P. S., and Keteyian, S. J. (Ed.). (2009). *Clinical Exercise Physiology* (Second Edition ed.): Human Kinetics.
- Fahey, D. T., Insel, P. M., Roth, W. T., and Wong, I. (2010). *Fit & Well: Core Concepts and Labs in Physical Fitness and Wellness* (Second Canadian Edition ed.): McGraw-Hill Ryerson.
- Fernandez-Sanchez, A., Madrigal-Santillan, E., Bautista, M., Esquivel-Soto, J., Morales-Gonzalez, A., Esquivel-Chirino, C., et al. (2011). Inflammation, Oxidative Stress, and Obesity. *International Journal of Molecular Sciences*, 12(5), 3117-3132.
- Fujimoto, S., Inui, A., Naotoshi, K., Seki, W., Koide, K., Takamiya, S., et al. (1997). Increased cholecystokinin and pancreatic polypeptide responses to a fat-rich meal in patients with restrictive but not bulimic anorexia nervosa. *Society of Biological Psychiatry*(41), 1068-1070.
- Gardiner, J. V., Jayasena, C. N., & Bloom, S. R. (2008). Gut hormones: a weight off your mind. *J Neuroendocrinol*, 20(6), 834-841.
- Geliebter, A., Maher, M. M., Gerace, L., Gutin, B., Heymsfield, S. B., & Hashim, S. A. (1997). Effects of strength or aerobic training on body composition, resting metabolic rate, and peak oxygen consumption in obese dieting subjects. *American Journal of Clinical Nutrition*, 66(3), 557-563.
- Gettman, L. R., Pollock, M. L., Durstine, J. L., Ward, A., Ayres, J., & Linnerud, A. C. (1976). Physiological-Responses of Men to 1, 3, and 5 Day Per Week Training-Programs. *Research Quarterly*, 47(4), 638-646.
- Gibala, M. J., Little, J. P., Macdonald, M. J., & Hawley, J. A. (2012). Physiological adaptations to low-volume, high-intensity interval training in health and disease. *J Physiol*, 590(Pt 5), 1077-1084.
- Glaser, B., Zoghlin, G., Pienta, K., & Vinik, A. I. (1988). Pancreatic polypeptide response to secretin in obesity: effects of glucose intolerance. *Horm Metab Res*, 20(5), 288-292.

- Haslam, D., Sattar, N., & Lean, M. (2006). ABC of obesity. Obesity--time to wake up. *BMJ*, 333(7569), 640-642.
- Hofmann, P., & Tschakert, G. (2011). Special needs to prescribe exercise intensity for scientific studies. *Cardiol Res Pract*, 2011, 209302.
- Horner, K. M., Byrne, N. M., Cleghorn, G. J., Naslund, E., & King, N. A. (2011). The effects of weight loss strategies on gastric emptying and appetite control. *Obesity Reviews*, 12(11), 935-951.
- Hurley, B. F., Seals, D. R., Ehsani, A. A., Cartier, L. J., Dalsky, G. P., Hagberg, J. M., et al. (1984). Effects of High-Intensity Strength Training on Cardiovascular Function. *Medicine and Science in Sports and Exercise*, 16(5), 483-488.
- Hwang, J. J., Chan, J. L., Ntali, G., Malkova, D., & Mantzoros, C. S. (2008). Leptin Does not Directly Regulate the Pancreatic Hormones Amylin and Pancreatic Polypeptide. *Diabetes Care*, 31(5), 945-951.
- Jakicic, J. M., Marcus, B. H., Gallagher, K. L., Napolitano, M., & Lang, W. (2003). Effect of exercise duration and intensity on weight loss in overweight, sedentary women: a randomized trial. *JAMA*, 290(10), 1323-1330.
- Kennedy, A. P., Shea, J. L., & Sun, G. (2009). Comparison of the Classification of Obesity by BMI vs. Dual-energy X-ray Absorptiometry in the Newfoundland Population. *Obesity*, 17(11), 2094-2099.
- LaFontaine, T. (1997). Resistance training for patients with hypertension. *Strength and Conditioning*, 19(1), 5-9.
- Larhammar, D. (1996). Structural diversity of receptors for neuropeptide Y, peptide YY and pancreatic polypeptide. *Regul Pept*, 65(3), 165-174.
- Lee, I. M., & Paffenbarger, R. S., Jr. (2000). Associations of light, moderate, and vigorous intensity physical activity with longevity. The Harvard Alumni Health Study. *Am J Epidemiol*, 151(3), 293-299.
- Martins, C., Kulseng, B., King, N. A., Holst, J. J., & Blundell, J. E. (2010). The effects of exercise-induced weight loss on appetite-related peptides and motivation to eat. *J Clin Endocrinol Metab*, 95(4), 1609-1616.
- Martins, C., Morgan, L., & Truby, H. (2008). A review of the effects of exercise on appetite regulation: an obesity perspective. [Review]. *Int J Obes*(32), 1337-1347.
- Martins, C., Morgan, L. M., Bloom, S. R., & Robertson, M. D. (2007). Effects of exercise on gut peptides, energy intake and appetite. *J Endocrinol*, 193(2), 251-258.
- Martins, C., Truby, H., & Morgan, L. A. (2007). Short-term appetite control in response to a 6-week exercise programme in sedentary volunteers. *British Journal of Nutrition*, 98(4), 834-842.
- McQueen, M. A. (2009). Exercise aspects of obesity treatment. *Ochsner J*, 9(3), 140-143.
- Miller, W. M., Spring, T. J., Zalesin, K. C., Kaeding, K. R., Nori Janosz, K. E., McCullough, P. A., and Franklin, B. A. (2012). Lower Than Predicted Resting Metabolic Rate Is Associated With Severely Impaired Cardiorespiratory Fitness in Obese Individuals. *Obesity (Silver Spring)*, 20(3), 505-511.
- Mochiki, E., Inui, A., Satoh, M., Mizumoto, A., & Itoh, Z. (1997). Motilin is a biosignal controlling cyclic release of pancreatic polypeptide via the vagus in fasted dogs.

- American Journal of Physiology-Gastrointestinal and Liver Physiology*, 272(2), G224-G232.
- Mukaimoto, T., & Ohno, M. (2012). Effects of circuit low-intensity resistance exercise with slow movement on oxygen consumption during and after exercise. *J Sports Sci*, 30(1), 79-90.
- Parkinson, C., Drake, W. M., Roberts, M. E., Meeran, K., Besser, G. M., & Trainer, P. J. (2002). A comparison of the effects of pegvisomant and octreotide on glucose, insulin, gastrin, cholecystokinin, and pancreatic polypeptide responses to oral glucose and a standard mixed meal. *J Clin Endocrinol Metab*, 87(4), 1797-1804.
- Peracchi, M., Tagliabue, R., Quatrini, M., & Reschini, E. (1999). Plasma pancreatic polypeptide response to secretin. *Eur J Endocrinol*, 141(1), 47-49.
- Romero-Corral, A., Somers, V. K., Sierra-Johnson, J., Thomas, R. J., Collazo-Clavell, M. L., Korinek, J., et al. (2008). Accuracy of body mass index in diagnosing obesity in the adult general population. *Int J Obes (Lond)*, 32(6), 959-966.
- Sainsbury, A., Shi, Y. C., Zhang, L., Aljanova, A., Lin, Z., Nguyen, A. D., et al. (2010). Y4 receptors and pancreatic polypeptide regulate food intake via hypothalamic orexin and brain-derived neurotrophic factor dependent pathways. *Neuropeptides*, 44(3), 261-268.
- Salamone, L. M., Fuerst, T., Visser, M., Kern, M., Lang, T., Dockrell, M., et al. (2000). Measurement of fat mass using DEXA: a validation study in elderly adults. *Journal of Applied Physiology*, 89(1), 345-352.
- Sizer, F. S., Whitney, E., & Piche, L. A. (2009). *Nutrition: Concepts and Controversies* (1 ed.). Toronto, Ontario: Nelson Education.
- Stanfield, C. L., and Germann, W. J. (Ed.). (2009). *Principles of Human Physiology* (Third ed.); Pearson.
- Strasser, B., & Schobersberger, W. (2011). Evidence for resistance training as a treatment therapy in obesity. *J Obes*, 2011, 9.
- Tortora, G. J., Neilsen, M. T. (2009). *Principals of Human Anatomy* (11th ed.); John Wiley & Sons Ltd.
- Wilmore, J. H., Costill, D.L., & Kenney, W. L. (2008). *Physiology of Sport and Exercise* (4th ed.); Human Kinetics.
- Wynne, K., Stanley, S., McGowan, B., Bloom, S. (2005). Appetite Control. [Review]. *Journal of Endocrinol*(184), 291-318.
- Zipf, W. B., O'Dorisio, T. M., Cataland, S., & Sotos, J. (1981). Blunted pancreatic polypeptide responses in children with obesity of Prader-Willi syndrome. *J Clin Endocrinol Metab*, 52(6), 1264-1266.

CHAPTER 3: CO-AUTHORSHIP STATEMENT

I will address my contributions to this thesis in five statements:

- i) This research idea was an addition to the 'Newfoundland Study' performed by the Dr. Sun lab.
- ii) Allied Health Services allowed our research team to utilize their space and equipment for the duration of the study. Memorial Varsity Athletics also allowed us access to their private gym facilities for the implementation of the resistance training program. Heather McCarthy and I recruited all participants and performed all research testing on each participant. The experimental procedure required two researchers at all times.
- iii) The exercise prescription was performed by three Canadian Society for Exercise Physiology (CSEP) certified personal trainers (CPT) under the supervision of Heather McCarthy, Dr. Duane Button, and myself.
- iv) Raw data was collected by Heather McCarthy, Farrell Cahill, and myself. I performed all data analysis procedures.
- v) With the Guidance of Dr. Button, I prepared the thesis.

CHAPTER 4: UNDERSTANDING THE EFFECTS OF EXERCISE ON GUT HORMONES AND OBESITY

MATTHEW B. MILLER, HEATHER MCCARTHY and DUANE C. BUTTON

School of Human Kinetics and Recreation
Memorial University of Newfoundland
St. John's, Newfoundland Canada

Corresponding Author: Duane Button Ph.D
School of Human Kinetics and Recreation
Memorial University of Newfoundland
St. John's, Newfoundland, Canada, A1C 5S7
(709) 864-4886 (telephone)
(709) 864-3979 (fax)
dbutton@mun.ca

4.1 ABSTRACT

The effect of high-intensity resistance training on gut hormones and obesity is relatively unknown. Thus, the objective of this study was to determine how a high-intensity circuit training program affects body composition, physiological measures, and pancreatic polypeptide levels in obese sedentary males. Eight obese apparently healthy male participants (34.3 ± 12.1 YOA, 179.1 ± 5.1 cm, 112.4 ± 20.1 kg), volunteered for this study. Participants' body composition, and blood hormone levels were measured before and after the exercise program. The exercise program lasted four weeks with exercise sessions three times a week (12 sessions total). Participant's heart rate, blood pressure, rating of perceived exertion, work volume and time to completion were measured each exercise session. The high-intensity circuit training program elicited significant changes in resting heart rate, systolic blood pressure, volume of work, body fat percentage, lean and fat tissue percentage, and leg and trunk fat percentage. There was no change in the circulating levels of pancreatic polypeptide. Participants experienced these changes in 6 hours (12 sessions x 30 minutes) of exercise. Throughout the duration of the exercise program participants' working HR was not lower than 85% of their age predicted HRmax.. Thus, healthy obese sedentary males can be prescribed a high-intensity circuit training program to significantly improve overall health.

4.2 INTRODUCTION

Obesity has been increasing worldwide at an alarming rate, and in Canada, the rate of obesity has tripled in the last two decades. Obesity not only has a direct effect on the health of the individual, there are also other concomitant health risks including, type II diabetes, cardiovascular disease (due to high cholesterol, high blood pressure, inactivity etc.), and some types of cancers (Durstine, Moore, Painter, & Roberts, 2009). Due to the increasing incidence of obesity, new ways to manage obesity and its risk factors are being explored. Exercise is a management technique that is increasingly being prescribed to obese individuals (Church, 2011; Durstine, et al., 2009; Gibala, Little, Macdonald, & Hawley, 2012; Jakicic, Marcus, Gallagher, Napolitano, & Lang, 2003). The proper method of exercise to manage obesity is often debated. Some professionals suggest aerobic exercise be prescribed due to the cardiovascular benefits and increase in caloric expenditure, whereas, other professionals prescribe resistance training due to the maintenance of lean muscle mass (which also increases basal caloric expenditure) (Chaudhary, Kang, & Sandhu, 2010; LaFontaine, 1997).

One form of training that combines both aerobic and resistance exercise is circuit training. Circuit training incorporates both the aerobic benefits of exercise (by keeping the heart rate elevated for the duration of the exercise session) as well as resistance training benefits (by utilizing resistance training exercises) (LaFontaine, 1997). Circuit training is often performed in a short period of time moving from exercise to exercise quickly, giving the participant as little rest as possible (Castinheiras-Neto, 2010;

Hofmann & Tschakert, 2011). The rest interval between exercises and the intensity at which the participant works at are important variables in high-intensity circuit training. The shorter the rest interval between exercises the more cardiovascular benefits are gained (Castinheiras-Neto, 2010). The intensity at which the individual exercises is a key factor in obtaining cardiovascular benefits. According to the American College of Sports Medicine 's Guidelines for Exercise Testing and Prescription (Donnelly, et al., 2009) the exercise intensity must be at a level at 64-70% of the participants maximum heart rate (HRmax) or higher, but no higher than 94%. It has been proven that exercising at a higher intensity provides more cardiovascular benefits when compared to exercising at a lower intensity while performing the same volume of work (Duncan, 2005; Lee & Paffenbarger, 2000). Because of this, a high-intensity, short rest interval exercise program should provide an individual with many cardiovascular benefits. Circuit training is a beneficial form of exercise for healthy participants, but unfortunately it is often assumed that performing this form of exercise with otherwise healthy obese individuals be avoided, however there is no scientific evidence to support this claim.

From a physiological basis, hormones also contribute to obesity. Many hormones have been studied in relation to obesity and exercise (Adrian, et al., 1976; Batterham, et al., 2003; O. B. Chaudhri, Wynne. K., & Bloom, S. R., 2008; Martins, Kulseng, King, Holst, & Blundell, 2010), however, the relationships between obesity, gut hormones and exercise have not been well established. Specifically, there are gastrointestinal hormones that directly influence appetite (motivation to eat) and satiety (the feeling of fullness), which are important contributors to the development of obesity (Batterham, et al., 2003;

O. Chaudhri, Small, & Bloom, 2006). Pancreatic polypeptide (PP) and peptide-YY (PYY₃₋₃₆) are both hormones that affect the upper brain centers to regulate satiety and appetite; PP has been described as a clone of PYY₃₋₃₆ but is different because PP is released from the pancreas, whereas PYY₃₋₃₆ is released from the small intestine (Chaudhary, et al., 2010). PP directly affects the upper brain centers (which makes it unique when compared to other gut hormones that affect the upper brain centers via secondary messengers) to regulate appetite and satiety. In obese individuals, the circulating levels of PP are decreased; leading to an increase in appetite and a decrease in satiety (Baynes, Dhillon, & Bloom, 2006; Glaser, Zoghlin, Pienta, & Vinik, 1988; Murphy & Bloom, 2004).

It has been shown that aerobic exercise can increase the levels of PP in the blood during and shortly after an exercise bout (Martins, et al., 2010; Martins, Morgan, Bloom, & Robertson, 2007; Martins, Truby, & Morgan, 2007). However, this research was only conducted for a short period of time (levels were tracked for three hours after exercise) and only with low-intensity aerobic exercise. There is no research conducted when monitoring the hormones levels in the long term (up to a week, month, or longer), or investigating how resistance exercise in the form of circuit training affects these hormones levels.

The purpose of the current research study was to investigate the effects of a four week high-intensity circuit training program on sedentary overweight or obese individuals, and how this form of exercise changes body composition, and pancreatic

polypeptide concentrations in the blood. It was hypothesized that high-intensity circuit training will provide a decrease in fat mass, but not over all body weight, a decrease in systolic blood pressure, heart rate, and increase strength. It is also hypothesized that the levels of PP will be increased following the completion of the exercise intervention.

4.3 METHODS

Participants:

A total of eight participants (34.3 ± 12.1 YOA, 179.1 ± 5.1 cm, 112.4 ± 20.1 kg) were recruited from the Province of Newfoundland and Labrador (NL). Participants were all sedentary overweight/obese ($36 \pm 3.1\%$) with an average BMI of 34.5 ± 4.5 . The participants did not have any metabolic, endocrine, or cardiovascular diseases. Participants were considered sedentary because they only performed activities of daily living (ADL's) and did not engage in any further exercise throughout the week (a 'physical activity and medical' questionnaire was used to obtain this information). Participants were verbally informed of all procedures, and if willing to participate, read and signed a written consent form and a Physical Activity Readiness Questionnaire (PAR-Q) prior to participation. The Memorial University of Newfoundland Human Investigation Committee approved this study.

Experimental procedure:

Before the start of the resistance training protocol participants were tested on each of the dependent variables (blood pressure, body composition, heart rate, and the gastrointestinal hormone pancreatic polypeptide). Once preliminary data was collected the participants began a resistance training exercise program for the duration of four weeks. Exercises included in the program were; squat, bench press, partial curl-up, dead lift, burpee, bent over row and shoulder press. For the duration of the four-week training period participants exercised on Monday, Wednesday, and Friday (a total of 12 sessions).

Blood pressure testing occurred each session throughout the duration of the study and blood samples were taken twice weekly (Tuesday and Thursday). See Figure 4.1 for details on the experimental timeline.

Independent variable:

Exercise Protocol:

Prior to the start of each training session, participants performed a five-minute warm-up consisting of functional body weight exercises using a combination of the exercises completed in the program or step-ups and jumping jacks. Heart rate and rating of perceived exertion (RPE) were monitored for intensity. Following the warm-up the participants completed the exercise program. The following description for each exercise was verbally instructed to each subject.

Squat - Start with feet about shoulder width apart, and toes pointed slightly outward. Keeping the back neutral and chest up throughout the movement, let the hips move backward then immediately bend the knees. Lower until the crease formed at the hips is horizontally aligned with the tops of knee. For each repetition, return to the starting position by reversing the motion (see Figure 4.2 A).

Bench Press - Lie on a bench, ensuring the body maintains contact with the floor and bench at five points: both of the feet, the hips, the upper back, and the head. Start with a grip slightly wider than shoulder width. Lower the weight slowly until the bar lightly touches the lower portion of the chest. While maintaining the five contact points,

press the weight upwards through the same movement path used in the downward movement (see Figure 4.2 B).

Partial Curl-up - Lying in supine position with knees bent at 90 degrees the participant will slowly curl-up the spine so that the tip of the middle finger reaches marked area of mat at 10cm. On the return, the shoulder blades and head must make contact with the mat and the tip of the middle fingers must return to the 0 cm mark.

Dead-Lift - Stand with feet shoulder-width apart, the knees slightly bent, and the back flat in a neutral position. Grasp the bar (away from the rack) with the hands slightly wider than shoulder width. Using the legs, lift the bar to knee height while maintaining a neutral spine with the chest up. Once the bar reaches the top of the knees, move to an upright position. While keeping the knees slightly bent, bend at the hips until the hands are ~30 cm from the floor while keeping a neutral spine (see Figure 4.2 C.).

Burpee - Begin in a squat position with the hands on the floor in front of you. Kick the feet back to a push-up position. Lower the upper body down until the elbows make an angle of 90 degrees. Immediately push the body upward and return feet to the squat position. Jump vertically (see Figure 4.2 D)

Bent over Row - Using a dumbbell stand with feet shoulder width apart and slightly bend the knees. Bend over at the hips so that the back is at a 60 to 80 degree angle and the spine is in a neutral position. Leading with the elbows, pull the weight upwards until it touches the mid torso. Bring the weight back to the starting position in a controlled manner following the same path as the upward movement (see Figure 4.2 E).

Shoulder Press - Start holding the weight at shoulder width and height. Keeping the back flat and chest up press the weight upwards until the arms are fully extended. Return to the starting position by reversing the motion (see Figure 4.2 F).

The exercises were modified (easier or harder) depending on the capability of the participant and the participant's technique. Participants were asked to complete 8-12 repetitions on all exercises. If the participant failed to reach eight repetitions on a given set, the weight was reduced and if the participant reached 12 repetitions the weight was increased. Participants completed a total of three sets of all exercises during each session and there was limited rest given between exercises and sets. The participant only rested when absolutely necessary. This exercise protocol was followed on each exercise day and weight was increased as necessary for each exercise session, for the curl-up and burpee, participants completed maximal repetitions for each set.

Dependent Variables:

Heart Rate:

A Polar T-31 heart rate monitor was used to measure heart rate before, during, and after each exercise session for each participant. HR was monitored to determine the intensity (% of age predicted HR max) each participant worked at during each exercise session (heart rate was recorded at the end of each circuit and averaged) and to determine the effects of the training program on resting HR.

Rating of Perceived Exertion

Rating of perceived exertion was recorded (at the end of each circuit and averaged) using the Borg's RPE scale. Participants rated their subjective exercise intensity from a scale of 6-20; six being equivalent to complete rest, and 20 being equivalent to the hardest effort they have ever experienced.

Blood Pressure:

Blood pressure was taken using an electronic blood pressure cuff (Physio Logic Auto Inflate BP Monitor; AMG Medical Inc. Montreal, QC.). Participants were seated with the arm supported at an angle of 15 degrees from the trunk. An appropriate size cuff was chosen and applied firmly to the participants left arm. The lower margin of the cuff was at heart level and two to three cm above the antecubital space. Changes in resting blood pressure were measured to track the cardiovascular response of the participants throughout the research study.

Body Composition:

Obesity is often defined by body mass index (BMI), however, this is a crude measurement which often mistakenly categorizes individuals as normal weight, or overweight when based on body fat percentage, when they are actually obese (Adams, et al., 2007; Kennedy, Shea, & Sun, 2009; Romero-Corral, et al., 2008). Thus, in the current study a dual-energy X-ray absorptiometry (DXA) that measures body fat percentage, which is the gold standard for measuring body composition (Kennedy, et al., 2009), was used to measure the participants body composition.

Body composition measurements included standing height and weight, and BMI. BMI was defined as body mass divided by height squared (kg/m^2). Percentage body fat (% BF), percentage trunk fat (% TF), and percentage arm fat (% AF) were measured by using dual-energy X-ray absorptiometry (DXA; Lunar Prodigy; GE Medical Systems, Madison, WI). Version 12.2 of the enCORE software package (GE Medical Systems) was used for DXA analysis. The experimental protocol for DXA methods has been previously published (Shea, et al., 2007; Sun, Vasdev, Martin, Gadag, & Zhang, 2005). Briefly, participants were required to fast for 12 h prior to the DXA scan and during the DXA scan participants lay in a supine position, with all metal accessories removed. The DXA scan took approximately 10 – 15 minutes to complete. These measurements were used to track changes in body composition throughout the duration of the research study.

Biochemical measurements using fasting blood samples:

Fasting (12 hours of fasting) blood samples were obtained from all participants by a registered nurse. Whole PP (Millipore Corporation Pharmaceuticals, Billerica, MA) concentrations were measured in duplicate with enzyme-linked immunosorbent assays (ELISAs). The intra-assay serum variation ranged from 3.3% - 5.0% and inter-assay serum variation ranged from 4.4% - 9.8%. The detection limit for the PP ELISA kit was 12.3 pg/ml with a 50µl sample size.

Statistical analysis:

All data analysis was conducted using SPSS statistics computing program version 17.0 (SPSS Inc. © 1993-2007). Data was analyzed using a one-way ANOVA repeated measures design. An alpha level of $p < 0.05$ was considered statistically significant. If significant differences ($p < 0.05$) were detected a Tukeys post hoc analysis was performed. Descriptive statistics for all data was reported as mean \pm SD.

4.4 RESULTS

Exercise Program

All eight subjects completed the study (with at least 80% adherence rate) and all were included in the analysis. There were no notable complications related to the exercise program. Table 4.1 includes all mean \pm SD values for raw data collected pre-exercise program and post-exercise program.

Exercise Intensity

Throughout the exercise program total volume of work, time to completion of work, rate of perceived exertion and heart rate were measured during each exercise session. The total volume of weight lifted on average increased by 50.7% ($p < 0.05$) from pre to post exercise. See Figure 4.3A for the change in total volume of weight lifted between each exercise session. The time it took to complete each exercise session was similar for each participant. There was no statistical significant difference for change in time to completion for each exercise session. Change in time to completion between the beginning and the end of the exercise program can be found in Figure 4.3 D. On average, participants working heart rate was maintained at 85 ± 3.6 % of their average age predicted HRmax throughout all 12 exercise sessions. Average working heart rate for each session can be found in Figure 4.3 A. The average rating of perceived exertion from session to session was 16.5 ± 2.5 . There was no statistical significant difference in RPE for any exercise session. The ratings of perceived exertion for each exercise session can be found in Figure 4.3 C.

Physiological Related Measures

The average resting heart rate decreased by 16.0% ($p < 0.05$) from pre to post exercise program, see Figure 4.4 A-B. Systolic blood pressure (SBP) decreased by 5.5% ($p \leq 0.04$) from pre to post exercise program, see Figure 4.5 A. Diastolic blood pressure (DBP) decreased by 3.4%, there was no significant difference in DBP, between pre and post exercise program, see Figure 4.5 B.

Body Weight and Body Composition

The participant's body fat percentage significantly ($p \leq 0.01$) decreased by 3.6% from pre to post exercise program, see Figure 4.6 B. There was no significant difference in the participant's body mass (Figure 4.6 A), BMI (Figure 4.6 C), lean body mass (Figure 4.6 D), or fat body mass (Figure 4.6 D) between pre and post exercise program. However, when tissue is expressed as a percentage of total mass, the percent fat tissue significantly ($p = 0.00$) decreased and the percent lean tissue significantly ($p = 0.00$) increased by 3.6% and 2% respectively, between pre and post exercise program. See Figure 4.7 A and 4.7 C for details. In addition there was a trend ($p = 0.09$) for decreased arm fat percentage 4.7% (Figure 4.7 C) and a significant ($p \leq 0.01$). decrease by 4% and 3% in leg and trunk fat percentages respectively, from pre to post exercise program (Figure 4.7 C).

Pancreatic Polypeptide

There was no significant difference in levels of circulating pancreatic polypeptide between pre and post exercise program, See Figure 4.8.

4.5 DISCUSSION

The current research study investigated the effect that a four week (12 exercise sessions) high-intensity circuit training (HICT) exercise intervention had on body composition, heart rate, blood pressure, and the circulating levels of pancreatic polypeptide in obese sedentary males. The most important findings were 1) a high intensity program was completed by all individuals with no complications thus; this type of training could be prescribed to an apparently healthy young obese individual in a safe manner, 2) a significant drop in resting heart rate and systolic blood pressure occurred with this exercise program, suggesting that a combination of resistance training and aerobic exercise can provide cardiovascular benefits 3) a significant drop in total body fat percentage, trunk and leg body fat percentage, as well as fat tissue percentage occurred due to this exercise program. A significant increase in lean tissue percentage also occurred, suggesting that not only did this exercise program decrease fat mass, but it also increased lean mass.

During the exercise intervention, the participant's average working heart rate, did not drop below 85% of their age predicted HRmax. Furthermore, the participant's RPE, and time to completion remained unchanged throughout the entire exercise program. This indicates that the intensity at which the participants worked also remained unchanged, however, the volume of work (sets x reps x kilograms) increased significantly (by ~50%) between the beginning and end of the exercise program. Participants were psychologically (via RPE) and physiologically (via HR) working at the same intensity

during each session, but their volume of work (or amount of weight lifted) completed increased substantially, indicating an increase in intensity as well as neuromuscular capacity.

Resting systolic blood pressure significantly decreased from 147.9 mmHg to 139.7 mmHg in only 12 exercise sessions indicating that only 12 sessions of high intensity circuit training can decrease systolic blood pressure from a hypertensive level to a high-normal level (Durstine, et al., 2009). Along with this decrease in blood pressure, there was a significant decrease in resting heart rate from 84.2 bpm to 70.6 bpm was also found. Similar to other research studies, there was no change in diastolic blood pressure (Chaudhary, et al., 2010). Decreased RHR is not commonly seen during and/or following resistance training and it has been suggested that aerobic exercise has a more profound effect on RHR than resistance training (Chaudhary, et al., 2010; Nybo, et al., 2010). In the current study, participants did experience a drop in RHR. Perhaps the change in RHR could be accounted for by the intensity at which they worked and lack of rest time. Castinheiras-Neto et al. in 2010 found that the shorter the rest interval is, the more cardiovascular benefits are obtained in resistance exercise (a decrease in HR and SBP was found in resistance training with lower rest intervals) (Castinheiras-Neto, 2010). In another study it was found that the intensity and exercise duration at which participants worked did not have an effect on their cardiovascular fitness (decreased RHR or BP) (Jakicic, et al., 2003). Despite this somewhat conflicting research about intensity of exercise, the results of the current study are not different from other research indicating that exercise training will decrease SBP, RHR, but not DBP (Castinheiras-Neto, 2010;

Chaudhary, et al., 2010; Moraes, et al., 2012; Nybo, et al., 2010). These research studies found this effect over 6 weeks (Chaudhary, et al., 2010) 12 weeks (Moraes, et al., 2012; Nybo, et al., 2010), and 52 weeks respectively (Jakicic, et al., 2003). These findings contradict the research done by Jakicic et al. (2003) which indicates exercise should be completed between 50-75% of HRmax for the safest form of exercise with the most health related benefits (increased caloric expenditure and cardiovascular improvements) (Jakicic, et al., 2003). However, in just 6 hours of exercise (12 sessions x ~30 minutes each) there were no injuries, and significant physiological and anthropometric changes occurred. Other research studies found similar results (i.e. decreasing rest interval provides more cardiovascular benefits) in ~9 hours (Chaudhary, et al., 2010), ~18 hours (Moraes, et al., 2012; Nybo, et al., 2010), and ~78 hours respectively (Jakicic, et al., 2003). This suggests that the intensity and rest intervals that the participants use are key components to the exercise program. The current research had short rest intervals and maintained a high intensity throughout the program which produced significant changes in RHR and SBP..

There was no significant decrease in body mass, or BMI; however, there was a significant decrease in body fat percentage. There was a significant increase of lean tissue percentage and decrease of fat tissue percentage, which together contribute a healthy change in body mass of approximately six pounds (27. Kgs). Similar research failed to find a change in body fat percentage when utilizing a 10 week resistance training program (Donges & Duffield, 2012) or a 12 week circuit training program (Kang, 2012). These programs only used resistance training (not circuit training) so the HR was not

elevated for a longer period of time (Donges & Duffield, 2012) and only performed aerobic exercise at 50-70% of HRR (Kang, 2012). This further supports the current findings that limited rest and higher intensity exercise, performed in 6 hours of work, are beneficial for weight loss.

An increase in lean tissue percent indicates that there was an increase in muscle mass, which is reflected in the lack of change in overall body mass. It is known that muscle tissue is denser than fat tissue, thus, when muscle increases and fat tissue decreases in a specific area, it will weigh more. It is for this reason that body mass index is an inaccurate measurement. Overall body fat percentage decreased significantly, however there was no change in BMI. This could be accounted for by the increase in muscle mass (lean tissue increased by 1.1 kgs). To see absolute changes in body composition a body fat measurement is more accurate (than BMI) and provides more accurate details about changes that are occurring in the body. As seen in the current research study, an increase in muscle mass and decrease in fat mass is not reflected in the BMI calculation, but is reflected in a DXA body analysis. If participants in the current research study only received BMI information, they would have been told no change in their body composition occurred, when in fact there were significant changes. For this reason, future research investigating changes in body composition should be conducted using a DXA scanner and not BMI.

The changes in fat percentage for the arms, legs, and trunk area due to this exercise program provide interesting insight into how and where fat breaks down. It is

commonly thought (by the general population) that the phenomenon of 'spot reduction' (exercising a specific body part to lose fat in that area) is the best way to lose fat in the desired location. However, as the results of this study show, spot reduction does not exist. There were significant changes in the fat percentage in the legs and the trunk, however not in the arms. Due to the nature of the exercises (mostly full body) and the area's in which fat was lost (legs and trunk) it is clear that no exercise can provide fat loss in a specific area. For example, participants performed a shoulder press and bench press (both upper body exercises), as well as a deadlift, squat, upright row, and burpee which also incorporate the upper body, but there was a trend towards significance for fat lost in the arms. This could be accounted for by the fact that less fat is carried in the arms (in males) in the first place, however this indicates that regardless of what exercise is being performed (at an appropriate intensity), there will be total body fat loss, and not fat loss in one area.

The levels of circulating pancreatic polypeptide did not change significantly. There are many factors that could contribute to this finding such as; genetic variants between participants, the classification of obesity that each participant fell into, and most significantly the participant's diet. The participant's diet was not monitored during the research study and participants were not asked to change their diet in any way. This could have a significant effect on the participants levels of pancreatic polypeptide since the release of pancreatic polypeptide is directly associated with the amount of calories consumed (Batterham, et al., 2003). If participants were changing their diets it is likely that the caloric consumption would also change and affect the levels of pancreatic

polypeptide in their circulation. Levels of PP could have increased (the opposite effect) and exercise could have induced more hunger (due to increased caloric expenditure), leading the participants to consume more calories leading to an increase in pancreatic polypeptide. The exercise program may have had an effect on the levels of PP; however, the lack of dietary information may have masked this effect.

This research was intended to investigate the effects that a high intensity circuit training program has on physiological measures, anthropometric measures, and pancreatic polypeptide. The participants were not asked to change their diet and diet was not monitored. Keeping track of the participant's diet would be a beneficial way to see changes that occur due to their eating habits. Due to the high variability associated with the levels of pancreatic polypeptide, eight subjects were probably not enough to see a change in the circulating levels of PP. To increase the power of the study more subjects would need to be recruited, and diet should be monitored or controlled. Including BMR as a dependent variable would also increase the information about the physiological changes occurring within the subjects, and further shed light into how the obese individual responds to high-intensity exercise.

Suggestions for future research

The results of this study showed that a high intensity circuit training protocol can provide participants with dramatic results in a short period of time. In the future, research should be conducted over a longer period of time (> 6 months), the diet of all participants should be monitored, and more participants should be used. Obtaining the participants

BMR values before and after the exercise intervention would also provide valuable information about how the participants' bodies are changing and adapting to the high intensity exercise. Investigating why pancreatic polypeptide occurs at lower levels in the obese population is important and more research should attempt to uncover the link between pancreatic polypeptide and other gut hormones and obesity.

Summary

Overall, a high intensity circuit training exercise program is very effective in providing significant physiological (RHR, SBP, volume of work) and anthropometric (BF%, lean and fat tissue percentage, trunk and leg fat percentage) changes in 12 exercise sessions or 6 hours of exercise. As discussed above, this is substantially shorter than other exercise protocols with similar findings. This research was conducted with obese sedentary males, and resulted in no injuries, with all participants completing the exercise program. A high-intensity circuit training program can be prescribed as a safe and effective management technique for obesity, and that the results are of great benefit to the otherwise healthy obese sedentary male population. The levels of pancreatic polypeptide remained unchanged; however, diet was not taken in account. The diet plays a large role in the amounts of PP in circulation, so there may have been an exercise effect that was masked by the participants' caloric intake.

4.6 REFERENCES

- Adams, T. D., Heath, E. M., LaMonte, M. J., Gress, R. E., Pendleton, R., Strong, M., et al. (2007). The relationship between body mass index and per cent body fat in the severely obese. *Diabetes Obesity & Metabolism*, 9(4), 498-505.
- Adrian, T. E., Bloom, S. R., Bryant, M. G., Polak, J. M., Heitz, P. H., & Barnes, A. J. (1976). Distribution and release of human pancreatic polypeptide. *Gut*, 17(12), 940-944.
- Batterham, R. L., Le Roux, C. W., Cohen, M. A., Park, A. J., Ellis, S. M., Patterson, M., et al. (2003). Pancreatic polypeptide reduces appetite and food intake in humans. *J Clin Endocrinol Metab*, 88(8), 3989-3992.
- Baynes, K. C., Dhillon, W. S., & Bloom, S. R. (2006). Regulation of food intake by gastrointestinal hormones. *Curr Opin Gastroenterol*, 22(6), 626-631.
- Castinheiras-Neto, A. G., Rodrigues da Costa-Filho, I., and, Farinatti, P. T. V. (2010). Cardiovascular Responses to Resistance Exercise are Affected by Workload and Intervals Between Sets. *Sociedade Brasileira De Cardiologia*, 95(4), 493-501.
- Chaudhary, S., Kang, M. K., & Sandhu, J. S. (2010). The Effects of Aerobic Versus Resistance Training on Cardiovascular Fitness in Obese Sedentary Volunteers. *Asian Journal of Sports Medicine*, 1(4), 177-184.
- Chaudhri, O., Small, C., & Bloom, S. (2006). Gastrointestinal hormones regulating appetite. *Philos Trans R Soc Lond B Biol Sci*, 361(1471), 1187-1209.
- Chaudhri, O. B., Wynne, K., & Bloom, S. R. (2008). Can Gut Hormones Control Appetite and Prevent Obesity? [Review]. *Diabetes Care*, 31(2), 284-289.
- Church, T. (2011). Exercise in obesity, metabolic syndrome, and diabetes. *Prog Cardiovasc Dis*, 53(6), 412-418.
- Donges, C. E., & Duffield, R. (2012). Effects of resistance or aerobic exercise training on total and regional body composition in sedentary overweight middle-aged adults. *Appl Physiol Nutr Metab*, 37(3), 499-509.
- Donnelly, J. E., Blair, S. N., Jakicic, J. M., Manore, M. M., Rankin, J. W., & Smith, B. K. (2009). American College of Sports Medicine Position Stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci Sports Exerc*, 41(2), 459-471.
- Duncan, G. E., Anton, S. D., Sydean, S. J., Newton R.L., Corsica, J. A., Durning, P. E., Ketterson, T. U., Martin A. D., Limacher, M. C., and Perri, M. G. (2005). Prescribing exercise at varied levels of intensity and frequency: a randomized trial. *Arch Intern Med*, 165(20), 2362-2369.
- Durstine, J. L., Moore, G. E., Painter, P. L., & Roberts, S. O. (2009). *Exercise Management for Persons With Chronic Diseases and Disabilities* (Third Edition ed.): Human Kinetics.
- Gibala, M. J., Little, J. P., Macdonald, M. J., & Hawley, J. A. (2012). Physiological adaptations to low-volume, high-intensity interval training in health and disease. *J Physiol*, 590(Pt 5), 1077-1084.

- Glaser, B., Zoghlin, G., Pienta, K., & Vinik, A. I. (1988). Pancreatic polypeptide response to secretin in obesity: effects of glucose intolerance. *Horm Metab Res*, 20(5), 288-292.
- Hofmann, P., & Tschakert, G. (2011). Special needs to prescribe exercise intensity for scientific studies. *Cardiol Res Pract*, 2011, 209302.
- Jakicic, J. M., Marcus, B. H., Gallagher, K. I., Napolitano, M., & Lang, W. (2003). Effect of exercise duration and intensity on weight loss in overweight, sedentary women: a randomized trial. *JAMA*, 290(10), 1323-1330.
- Kang, H., Lee, Y. S., Park, D., & Kang, D. (2012). Effects of 12-week circuit weight training and aerobic exercise on body composition, physical fitness, and pulse wave velocity in obese collegiate women. *Soft Comput*(16), 406-410.
- Kennedy, A. P., Shea, J. L., & Sun, G. (2009). Comparison of the Classification of Obesity by BMI vs. Dual-energy X-ray Absorptiometry in the Newfoundland Population. *Obesity*, 17(11), 2094-2099.
- LaFontaine, T. (1997). Resistance training for patients with hypertension. *Strength and Conditioning*, 19(1), 5-9.
- Lee, I. M., & Paffenbarger, R. S., Jr. (2000). Associations of light, moderate, and vigorous intensity physical activity with longevity. The Harvard Alumni Health Study. *Am J Epidemiol*, 151(3), 293-299.
- Martins, C., Kulseng, B., King, N. A., Holst, J. J., & Blundell, J. E. (2010). The effects of exercise-induced weight loss on appetite-related peptides and motivation to eat. *J Clin Endocrinol Metab*, 95(4), 1609-1616.
- Martins, C., Morgan, L. M., Bloom, S. R., & Robertson, M. D. (2007). Effects of exercise on gut peptides, energy intake and appetite. *J Endocrinol*, 193(2), 251-258.
- Martins, C., Truby, H., & Morgan, L. A. (2007). Short-term appetite control in response to a 6-week exercise programme in sedentary volunteers. *British Journal of Nutrition*, 98(4), 834-842.
- Moraes, M. R., Bacurau, R. F., Casarini, D. E., Jara, Z. P., Ronchi, F. A., Almeida, S. S., et al. (2012). Chronic conventional resistance exercise reduces blood pressure in stage 1 hypertensive men. *J Strength Cond Res*, 26(4), 1122-1129.
- Murphy, K. G., & Bloom, S. R. (2004). Gut hormones in the control of appetite. *Exp Physiol*, 89(5), 507-516.
- Nybo, L., Sundstrup, E., Jakobsen, M. D., Mohr, M., Hornstrup, T., Simonsen, L., et al. (2010). High-intensity training versus traditional exercise interventions for promoting health. *Med Sci Sports Exerc*, 42(10), 1951-1958.
- Romero-Corral, A., Somers, V. K., Sierra-Johnson, J., Thomas, R. J., Collazo-Clavell, M. L., Korinek, J., et al. (2008). Accuracy of body mass index in diagnosing obesity in the adult general population. *Int J Obes (Lond)*, 32(6), 959-966.
- Shea, J., Randell, E., Vasdev, S., Wang, P. P., Roebathan, B., & Sun, G. (2007). Serum retinol-binding protein 4 concentrations in response to short-term overfeeding in normal-weight, overweight, and obese men. *Am J Clin Nutr*, 86(5), 1310-1315.
- Sun, G., Vasdev, S., Martin, G. R., Gadag, V., & Zhang, H. W. (2005). Altered calcium homeostasis is correlated with abnormalities of fasting serum glucose, insulin

resistance, and beta-cell function in the Newfoundland population. *Diabetes*, 54(11), 3336-3339.

4.7 FIGURE LEGEND

Figure 4.1: Timeline of the experimental Procedure

Figure 4.2: A: squat, B: bench press C: deadlift, D: burpee, E: row, F: shoulder press

Figure 4.3: Exercise Intensity. A. The total volume of work completed over the duration of the 12 sessions compared to the average heart rate for each session. For clarity, standard deviation bars are only represented in one direction for each data set. B. The change in total work between session 1 and session 12 of the exercise program. C. The rate of perceived exertion for each session throughout the exercise program. D. The change in time to completion between the beginning and end of the exercise program. Significant difference is indicated by a * with $p < 0.05$. Data represented as means \pm SD

Figure 4.4: Resting Heart Rate and Change in Heart Rate. **A:** The average resting heart rate (bpm) for each participant over each session. * indicates there was a significant difference between the indicated exercise session compared to the first exercise session **B:** The change in resting heart rate from the beginning of the exercise program to the end of the exercise program. * represents a significant difference at $p < 0.05$. Data represented as means \pm SD.

Figure 4.5: Change in Systolic and Diastolic Blood Pressure. The average change in **A:** systolic and **B:** diastolic blood pressure (mmHg) between the beginning of the exercise

program and the end. * represents a significant difference at $p < 0.05$. Data represented as means \pm SD.

Figure 4.6: Body mass, body fat, BMI, and lean vs. fat mass. **A.** The change in body mass, **B.** The change in body fat percentage, **C.** The change in BMI, **D.** The change in lean mass and fat mass, between the beginning and end of the exercise program. * represents a significant difference at $p < 0.05$. Data represented as means \pm SD.

Figure 4.7: Fat and lean tissue percentages. **A.** Over all body fat tissue percentage, **B.** The lean tissue percentage, **C.** The percent fat found in the arm, leg, and trunk, between the pre and post exercise program. * represents a significant difference at $p < 0.05$. Data represented as means \pm SD

Figure 4.8: The change in circulating levels of pancreatic polypeptide in the blood between pre and post exercise program. Data represented as means \pm SD

Table 4.1. Summary of all raw data values (mean \pm SD)

	Pre-Exercise Program		Post-Exercise Program		<i>p</i>
	Mean	SD	Mean	SD	
Volume of Work (kgs)	5473.9	2790.6	8249.4	4257.8	*0.04
Time to completion (mins)	28.7	5.8	28.5	8.7	0.91
HR (bpm)	84.2	11.8	70.6	9.8	*0.04
SBP (mmHg)	147.8	8.2	139.7	9.7	*0.03
DBP (mmHg)	82	4.6	79.2	9.1	0.38
Body Fat (%)	36	3.1	34.7	3.2	*0.01
Weight (kgs)	112.4	20.2	111.1	20.3	0.26
BMI (kg/m ²)	34.5	4.5	34.1	4.6	0.17
Lean Body Mass (kgs)	66.6	9.3	67.6	1.0	0.26
Fat Body Mass (kgs)	40.1	9.4	38.5	9.9	0.1
Lean Tissue (%)	62.7	3.2	64.0	3.3	*0.00
Fat Tissue (%)	37.3	3.2	35.9	3.3	*0.00
Arm Fat (%)	28.7	4.9	27.4	5.7	0.09
Leg Fat (%)	32.2	4.2	30.9	3.9	*0.01
Trunk Fat (%)	41.9	3.0	40.6	3.1	*0.00
Pancreatic Polypeptide (pg/ml)	163.6	181.9	156.5	199.6	0.61

Heart rate (HR), beats per minute (BPM), systolic blood pressure (SBP), diastolic blood pressure (DBP), body mass index (BMI)

Figure 4.1: Timeline of Experimental Procedure

Monday	Tuesday	Wednesday	Thursday	Friday
	Pre-testing Blood Work	Exercise Session 1	Blood Work	Exercise Session 2
Exercise Session 3	Blood Work	Exercise Session 4	Blood Work	Exercise Session 5
Exercise Session 6	Blood Work	Exercise Session 7	Blood Work	Exercise Session 8
Exercise Session 9	Blood Work	Exercise Session 10	Blood Work	Exercise Session 11
Exercise Session 12	Post-Testing Blood Work			

Figure 4.2 A: Squat



Figure 4.2 B: Bench Press

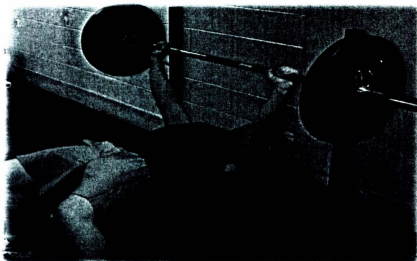


Figure 4.2 C: Dead Lift

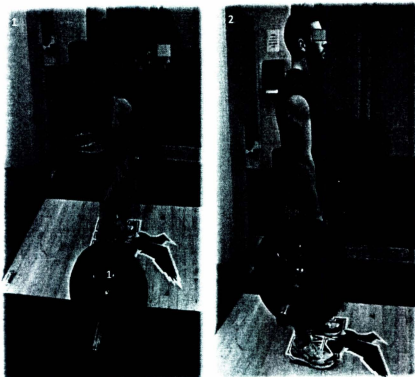


Figure 4.2 D: Burpee



Figure 4.2 E: Row

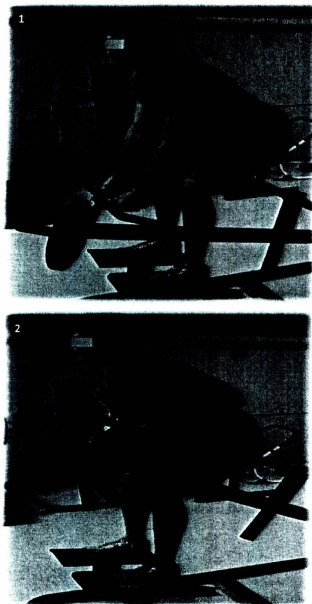


Figure 4.2 F: Shoulder Press

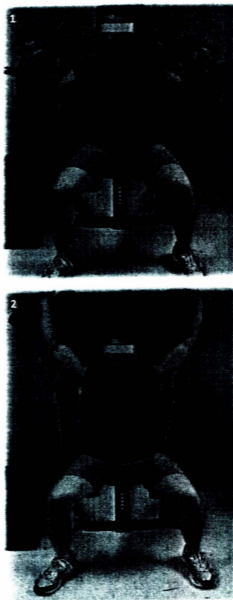


Figure 4.3 A-D: Exercise Intensity

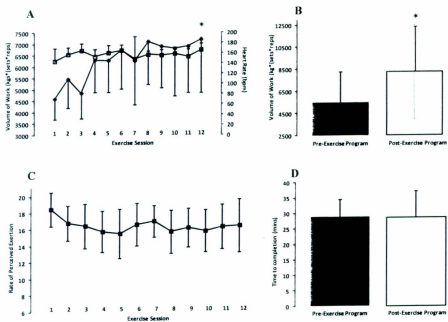


Figure 4.4 A-B: Change in Resting Heart Rate over 12 Exercise Sessions.

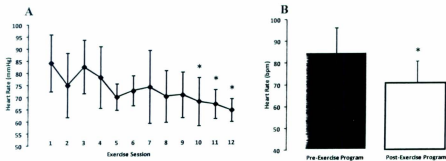


Figure 4.5 A-B: Change in Systolic and Diastolic Blood Pressure

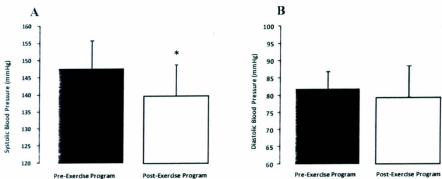


Figure 4.6 A-D: Body Mass, Body Fat, BMI, Lean vs Fat Mass

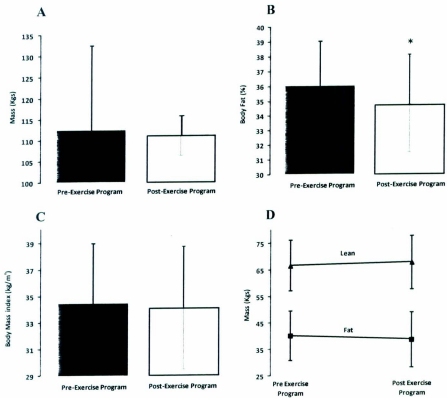


Figure 4.7 A-C: Lean and Fat Tissue Percentages

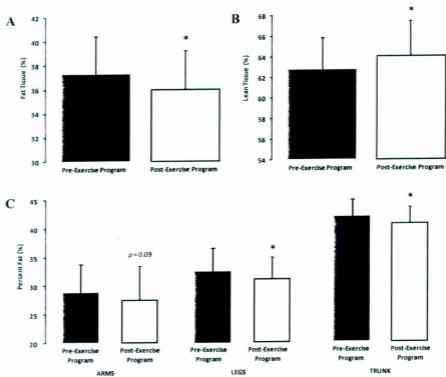


Figure 4.8: Pancreatic Polypeptide

