THE EFFECTS OF A SINGLE BOUT OF RESISTANCE EXERCISE ON MEASURES OF POSTPRANDIAL LIPEMIA, INFLAMMATION, AND ENDOTHELIAL FUNCTION FOLLOWING A HIGH FAT MEAL IN LEAN AND OBESE YOUNG WOMEN

by

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A Thesis for the Degree

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Chapter 1
Introduction

Background

The leading cause of death in the United States is heart disease, which is typically a result of behavioral and/or biological risk factors. Behavioral risk factors can include physical inactivity and a poor diet, among others factors. It is well known that physical activity reduces the mortality risk from cardiovascular disease (CVD), as well as lowers the risk of developing high blood pressure, type 2 diabetes mellitus, and certain types of cancer (34). Despite the current knowledge of the numerous health benefits of regular exercise, 60% of American adults do not regularly exercise and 25% are not active at all.

Besides inactivity, diet plays a major role in developing CVD. Elevated plasma triglycerides (TG), known as hypertrigylceridemia, are currently accepted as a synergistic risk factor for CVD (12, 31). Risk factors for developing CVD and/or type 2 diabetes mellitus such as hypertrigylceridemia, dyslipidemia, insulin resistance, glucose intolerance, hypertension, and visceral adiposity are collectively know as the metabolic syndrome (9, 34). Approximately, 24 percent of American adults have the metabolic syndrome, and 44 percent Americans over the age of 50 are affected by it (9). Individuals with the metabolic syndrome are 2.3 times more likely to develop CVD, three times more likely to develop coronary heart disease or experience a stroke, and they are six times more likely to have a cardiovascular-related death.

Metabolic syndrome risk factors been also been associated with an elevated postprandial lipemic response, compared to a healthy population (34). Postprandial lipemia (PPL) is the rise in plasma concentrations of TG and triglyceride-rich lipoproteins (TRL-TG) following a meal, and is considered an independent risk factor for CVD (31, 34, 44). Furthermore, the risk of developing CVD due to hyperlipemia is much greater in women than men, with a 76 percent

increased risk compared to 32 percent, respectively (16). The majority of Americans consume three or more high fat meals a day. When high fat meals are consumed on a regular basis, plasma TG levels are typically still elevated from the previous meal by the time the next high fat meal is consumed, causing chronic hypertriglyceridemia (20).

Repeated exposure to a hyperlipemic state, typical of the American diet, is linked to multiple disturbances of lipoprotein metabolism, which alters endothelial function (26). When blood TG levels remain elevated for an extended period of time, it causes an atherogenic effect on lipoproteins (9). This occurs because the lipoproteins exchange their triacylglycerol for cholesterol esters from cholesterol rich lipoproteins, which in turns causes a reduction in high-density lipoprotein cholesterol (HDLC), leaving a high proportion of low-density lipoprotein cholesterol (LDLC). Chronic hyperlipemia and acute postprandial hyperlipemia (PPHL) are also associated with leukocyte activation and an elevated thrombotic potential, therefore promoting endothelial cell dysfunction and vascular damage (42, 43). A change in endothelial function or endothelial dysfunction (ED) is known to cause a compensatory response in the vasculature, which causes the formation of procoagulant and vasoactive molecules, growth factors and inflammatory cytokines. These proinflammatory factors can alter endothelial cells such that they promote an atherogenic phenotype (17). Also, an increase in plasma TG during the state of PPHL can lead to an increase in reactive oxygen species (ROS) production (1, 3, 15). Increased ROS may ultimately lead to the degradation of nitric oxide (NO), an important vasodilator, which may cause further ED along with an increase in proinflammatory factors (15, 29). Therefore, continual PPHL leading to disturbances and alterations of endothelial cells can elicit a series of changes related to the initiation, progression, and complications of atherosclerosis (4, 15, 17, 37, 48).

Based on current research and clinical evidence, atherosclerosis is considered to be a postprandial phenomenon. Individuals who typically experience a heightened lipemic response in the postprandial state also tend to have symptoms of the metabolic syndrome, lipoprotein metabolism impairment, signs of atherosclerosis and ED (47). PPHL, as well as other metabolic disturbances typically linked with obesity, have even been indicated in normal weight, seemingly healthy young men and women (35). This may be partly due to chronic physical inactivity, poor dietary habits, alterations in their skeletal muscle metabolism, as well as increased visceral adiposity. Due to the increasing number of young adults in America with CVD and type 2 diabetes, any interventions to prevent or attenuate the negative effects of PPHL are of great value to researchers and the general public (20, 35).

Modalities that can temporarily decrease the negative effects of PPHL can result in a larger cumulative effect over the long term, such as significantly reducing a person's risk for developing CVD. Aerobic and resistance exercise, both acutely and chronically, have been used as modalities to blunt the negative effects of PPHL on the endothelium (47, 30, 14, 34, 46, 38, 25, 13, 27, 35). Many researchers have reported a decreased postprandial lipemic response after aerobic exercise training, possibly due to increased insulin sensitivity, improve glucose tolerance, increased TG clearance, and enhanced lipoprotein lipase (LPL) activity. Endurance trained individuals are shown to demonstrate a lower lipemic response to a meal than untrained individuals; however, this may be attributed to the triglyceride lowering effects of their most recent bout of exercise, known as the recency effect (6, 25, 47).

Although the majority of research has been conducted to examine the effect of aerobic exercise on PPHL and its related responses, a few research studies have been conducted to determine the effects of various forms of resistance exercise on the postprandial state (33, 35, 39,

40, 56). Petitt and colleagues (33) reported that the TG area under the curve (AUC) response was significantly lower 16 hours after a resistance exercise trial compared to an aerobic exercise trial of equal energy expenditure. Zotou et al. (56) also found a similar effect on plasma TG and TRL-TG in healthy untrained women following an acute bout of resistance exercise. It is suggested that the higher intensity, lower repetition contractile activity associated with resistance training can increase LPL activity, which could attenuate the PPHL response to a greater extent than less intense aerobic activity (13, 33, 35). Another mechanism behind the decreased TG response due to resistance exercise might be due to an increase in fat oxidation and TG clearance after the workout.

While the mechanism by which aerobic and/or resistance exercise attenuate the PPHL response is not fully understood, the timing of exercise sessions compared to meal consumption is shown to produce different responses (55). Research has shown that the greatest postprandial TG lowering effects occurs many hours after exercise, with the greatest effects seen between 14 and 16 hours post-exercise (33, 55, 56). Also, the total energy expenditure of an aerobic or resistance exercise session is thought to be a primary factor affecting the reduction of PPHL due to the exercise (46). An inverse correlation exists between the total energy expenditure of a workout and the degree of the postprandial response; meaning the greater the energy expenditure from exercise, the smaller the PPL response (33, 46). Tsetsonis et al. (46) found that even low-intensity aerobic exercise can provide beneficial effects on the PPL response, as long as the total energy expenditure matched that of a more intense exercise session.

There is less research and knowledge concerning the effects of resistance exercise compared to aerobic exercise on the postprandial state; however, some evidence suggests that resistance exercise can reduce fasting and postprandial TG concentration by increasing TG

clearance, increasing LPL activity, and increasing fat oxidation (33, 35, 56). Also, only a few previous studies of fasting and postprandial TG metabolism include young women, especially those under the age of 25 years, in their subject population (16, 33, 34, 39). Further studies on this topic utilizing women are necessary because women regulate TG metabolism differently than men, and women have an increased risk for developing CVD due to hypertriglyceridemia (23, 39). Also, a better understanding of pre-clinical risk factors for CVD in the young adult population is needed to prevent or slow the progression of this disease (16, 34). Research based evidence concerning the effects of resistance exercise on PPHL and its related effects have clinical implications for individuals suffering from the metabolic disorders such as the metabolic syndrome, CVD, type 2 diabetes, and ED. Resistance and/or aerobic exercise could serve as a realistic and non-medical intervention for individuals suffering from such metabolic diseases.

Purpose of the Study

The purpose of this study was to evaluate the effects of an acute bout of resistance exercise on PPHL, endothelial function, and inflammatory markers following a high fat meal in lean and obese, apparently healthy young women. Specifically, we examined fasting and postprandial plasma TG, endothelial function via flow-mediated dilation (FMD) and Nitrite/Nitrate concentrations, and systemic inflammation via measurement of interleukin-6 (IL-6), C-reactive protein (CRP), and total and differential leukocyte counts.

Hypotheses

It was hypothesized that among young women, a single bout of resistance exercise 14 to 16 hours prior to consumption of a high fat meal would:

1. Attenuate the PPHL response by reducing the plasma TG concentrations.
2. Increase FMD, therefore reducing postprandial ED.
3. Increase Nitrite/Nitrate concentrations, indicating an increase in NO production.
4. Attenuate the postprandial inflammatory response of IL-6.
5. Produce lower levels of CRP in the lean group compared to the obese.
6. Promote leukocytosis immediately after and 15 hr post-exercise

It was hypothesized that the high fat meal will induce leukocytosis in both EXS and CON trials.

Significance

The typical American spends much of the day in the postprandial state; therefore, a diet high in fat, typical of the American diet, would cause triglyceride levels to remain elevated for extended periods of time. Elevated postprandial triglycerides are linked to a decreased NO release from cells, causing endothelial dysfunction due to reduced vasodilatation. PPHL is also linked to disturbances in lipoprotein metabolism, increased expression of inflammatory markers, and the formation or progression of atherosclerotic plaques. Continually blunting the PPHL response is believed to lower the risk of CVD by increasing endothelial function. One modality to reduce this response is exercise. Relatively small decreases in the PPHL response arising from moderately intense exercise, most or all days of the week, could have an important cumulative effect on endothelial function in the long term. Even apparently healthy young adults are shown to exhibit signs of an increased risk of CVD, especially in obese or overweight individuals. Currently, most studies have focused on the affects of aerobic exercise on the postprandial lipemic response; therefore, less is known about the lipid lowering effects of resistance exercise, especially in the young female population. If the hypotheses are supported,

the results will demonstrate the beneficial therapeutic value of continual, life-long exercise as a means of reducing the risk of CVD, ED, and atherosclerosis in an apparently healthy population of young females. Also, appropriate preventive exercise programs could be formed with an enhanced understanding of how resistance exercises alter risk factors directly related to CVD.

Chapter II
Review Of Literature

Background

Cardiovascular disease (CVD) is the leading cause of death in the United States and is typically attributed to behavioral risk factors such as physical inactivity and poor diet habits. Physical activity is known to reduce the risk of mortality from CVD, as well as lower the chance of developing high blood pressure, type 2 diabetes mellitus, and certain types of cancer (37). Despite this current knowledge, 60% of American adults do not regularly exercise and 25% are not active at all. The western dietary habit, typical to most Americans, involves 3 or more high-fat meals a day causing plasma triglycerides (TG) to remain elevated between meals, causing a continual state of hypertriglyceridemia (19). This harmful dietary pattern also plays a major role in developing CVD and other metabolic diseases.

The metabolic syndrome is the collection of multiple metabolic disturbances, which together or separately could lead to the development of CVD and/or type 2 diabetes (9, 34). Components of the metabolic syndrome include: hypertrigylceridemia, dyslipidemia, insulin resistance, glucose intolerance, hypertension, and visceral adiposity. Approximately, 24 percent of Americans adults have the metabolic syndrome and it's prevalence increases with age, as 44 percent of Americans over the age of 50 are affected by it (9). With the metabolic syndrome, individuals have a 2.3 time more likely chance to develop CVD, a three time more likely chance to develop coronary heart disease or experience a stroke, and a six time more likely chance to have a cardiovascular-related death.

Elevated postprandial triglycerides, known as postprandial hyperlipemia (PPHL), is linked to endothelial cell dysfunction via lipoprotein metabolism disturbances, decreased vasodilator properties, increased expression of inflammatory markers, and the formation or

progression of atherosclerotic plaques (14, 19, 26, 42, 43). Blunting this PPHL response is believed to lower the risk of developing CVD by increasing endothelial function. Even young, apparently healthy individuals show certain risk factors for developing CVD or endothelial dysfunction (ED), especially in the obese and overweight population (20, 35). One modality to reduce the severity of the PPHL response is aerobic or resistance exercise. Continually blunting the PPHL response from a consistent exercise routine could have an important cumulative effect on endothelial function in the long term (47, 34).

Postprandial Lipemic Response

Postprandial lipemia describes the blood TG content following the consumption of a meal (20). This TG response is greatly increased following a high fat meal, especially a meal containing mostly saturated fat. The symptoms of the metabolic syndrome alone or combined are linked to elevated PPHL, which can cause the development of ED, atherosclerosis, and CVD (12, 16, 26, 34). Repeated exposure to a hyperlipemic state, typical of the American diet, is linked to multiple disturbances of lipoprotein metabolism, which alters endothelial function (26). Individuals can experience a near continuous state of hyperlipemia if they regularly consume high-fat meals, before their plasma TG levels have returned to resting levels from the previous meal (20). PPHL is associated with various metabolic changes, which can promote an atherogenic phenotype (9). Some of these changes include: increased levels of LDLC, decreased production of HDLC, increased chylomicron production, increased inflammatory markers, decreased vasodilator properties and the progression of atherosclerotic plaque formation. When blood TG levels remain elevated for an extended period of time, lipoproteins can exchange their triacylglycerol for cholesterol esters from cholesterol rich lipoproteins, resulting in a reduction in

HDLC, leaving a high proportion of LDLC. All of theses atherogenic changes due to a chronic state of PPHL can be attributed to ED and the development of CVD (9, 20).

Various factors determine an individual's PPL response to a meal. As previously mentioned, symptoms of the metabolic syndrome are linked to an exaggerated PPHL response (12, 16, 26, 34). Dyslipidemia, a symptom of the metabolic syndrome, results in a greater PPL response due to an elevated TG concentration for an extended period of time (12). Impaired insulin sensitivity, also a symptom of the metabolic syndrome, reduces TG clearance after a meal and therefore increase PPL (26, 31). Obesity is also linked to an exaggerated PPL response due to the tendency for a greater fat intake in this population, and a greater free fatty acid concentration from excess adipose tissue, all of which can impair TG clearance after a meal (44, 26, 27). Specifically, one study revealed positive correlation between the adiposity of an individual and the extent of the postprandial lipemic response in a resting condition, meaning the obese individuals experienced a greater PPHL response compared to the lean individuals (27). Gender is another determinant of an individual's PPL response. Women are shown to have a greater risk of developing CVD due to hyperlipemia than men, with a 76 percent increased risk compared to 32 percent, respectively (16). Other factors than can affect the PPL response are age, dietary choices, physical activity level, and genetics (20). Altogether, exaggeration of the PPL response have been linked to the development of CVD, ED, and atherosclerosis.

Inflammation as a Component of Atherosclerosis

It is now well known that atherosclerosis is a low-grade inflammatory disease, which can result in endothelial cell dysfunction (37). People with elevated levels of inflammatory markers are shown to have an increased risk of having a cardiovascular event (32). Even acute

inflammatory responses due to an infection, in otherwise healthy individuals, temporarily decrease endothelial function (17, 48). Proinflammatory factors, such as C-reactive protein (CRP), interleukin 1 (IL-1), tumor necrosis factor-α (TNF-α), oxidized LDL, and sometimes interleukin 6 (IL-6) can alter endothelial cells to promote this atherogenic characteristic (17). The endothelium is a vital endocrine organ that maintains vascular tone, regulates leukocyte and platelet adhesion molecules, regulates vascular cell growth and repair, and also regulates the productions of inflammatory cytokines (15). Maintaining the normal function of the vascular endothelial greatly effects the function of other metabolic parameters in the human body.

An atherosclerotic plaque, formed on the medial lining of the arterial walls, is more than just an accumulation of lipids, but instead it is also composed of a mixture of fibrous particles, cytokines, adhesion molecules, collagens and elastins (9, 37). Continual sheer stress or damage to the endothelial lining caused by an increased systemic blood pressure with the presence of an atherosclerotic plaque can lead a further increases in such inflammatory cytokines and particles. If the plaque ruptures a thrombosis can occur, which is a life-threatening event that occludes the artery and can lead to an ischemic myocardial infarction (37). In one study, Hyson et al. (20) found a significant correlation between the thickness of the medial arterial wall and the extent of the PPL response, suggesting a link between atherosclerosis and postprandial TG metabolism.

Chronic and postprandial hypertriglyceridemia are associated with leukocyte activation and an elevated thrombotic potential, therefore promoting endothelial cell dysfunction and vascular damage (42, 43). The relationship between hyperlipidemia and endothelial dysfunction is supported by the findings of Hyson et al. (19) who observed an activation of platelets and monocytes after a moderate to high fat meal. The results of *in vitro* studies also support this relationship since triglyceride-rich lipoproteins were shown to alter the normal function of

endothelial cells (14). ED can lead to a compensatory response, including the formation of procoagulant and vasoactive molecules, inflammatory cytokines, and growth factors. These proinflammatory factors can alter endothelial cells such that they promote an atherogenic phenotype (17). This can then cause the endothelium to further express adhesion molecules, monocyte chemotactic factors and gradient-dependent diffusion factors, which can accelerate the inflammatory process in the vasculature. Therefore, the vascular endothelium can be affected by and contribute to the inflammatory process in a cyclic manner that may lead to atherosclerosis (17, 37).

Endothelial Dysfunction and the progression of Atherosclerosis

Changes in endothelial function or ED can elicit a series of changes related to the initiation, progression, and complications of atherosclerosis (4, 17, 37, 48). ED can be caused by various factors such as elevated LDLC, decreased HDLC, decreased vasodilator properties, hypertension, type 2 diabetes mellitus, genetic alterations, elevated plasma homocystine levels, free radicals from cigarette smoking, and/or infectious microorganisms (37). Under normal conditions, the endothelium maintains a vasodilator, antithrombotic, and anti-inflammatory state; however, people who have risk factors for CVD may exhibit a loss of endothelium-derived nitric oxide (NO), a potent vasodilator (17, 18, 50, 51, 49). Furthermore, proinflammatory factors are responsible for down regulating the expression of endothelial nitric oxide synthase (eNOS), which also decreases NO production (17, 29, 48). Endothelium-derived NO works not only as a vasodilator, but it also inhibits platelet activity and adhesion of leukocytes to the endothelial surface. The loss of NO during proinflammatory periods is one of the modifications of

endothelial function that might be a critical mechanism that associates the inflammatory state to atherosclerosis (41, 51).

Numerous investigations have been conducted to further understand this link between ED, inflammation, and atherosclerosis. Various methods are used to measure the severity of this problem, which include flow-mediated dilation (FMD), TG area under the curve (AUC) or PPL, and various inflammatory markers. Some non-invasive techniques have been developed that utilize ultrasonographic imaging to observe preclinical signs of vascular disease and are now widely used in clinical studies (8, 10). One of these techniques is FMD, which records the vasodilator response to increased arterial flow that is dependent on NO bioavailability (10). Of the various non-invasive methods of accessing vascular endothelium function, FMD remains the most reproducible method. FMD uses a high frequency ultrasound to measure blood flow and brachial artery dilation, expressed as a percent change, after reactive hyperemia is induced by forearm or upper arm cuff occlusion (1). This technique provokes the release of NO, resulting in vasodilation that can be quantified as an index of vascular function (8). Determination of the NO radial itself is difficult because it has a relatively short half-life (29). Therefore, assays were developed to determine the stable end-products of NO, nitrite and nitrate, which together are useful indicators of NO radical formation.

Berry et al. (3) reported that after a high fat meal, endothelial dysfunction was increased as evidenced by a 3% reduction in FMD ($P<0.001$) (3). Many other investigators have reported a negative correlation between FMD and various inflammatory markers such as CRP, IL-6, TNF-α, oxidative LDL, and soluble intercellular adhesion molecule 1 (4, 17, 44, 45, 50). The inflammatory marker, CRP, correlating strongly with factors of the metabolic syndrome and is shown to be elevated in the state of ED (9). Conversely, Verma et al. (49) reported that in 1154

healthy, young firemen no correlation was observed between FMD, an assessment of ED, and CRP. FMD has also been shown to negatively correlate with PPHL, meaning an increase in FMD coincides with a decrease in TG AUC (3). Further studies comparing the FMD assessments of ED and other metabolic diseases are necessary to fully understand their relationship.

Exercise Effects on Postprandial Hyperlipemia

Modalities that can decrease the negative effects of PPHL on an acute basis can result in a larger cumulative effect over the long term, such as significantly reducing a person's risk for developing CVD. Aerobic and resistance exercise, both acutely and chronically, have been used as modalities to blunt the negative effects of postprandial hypertriglyceridemia on the endothelium (34, 47). Numerous authors have reported a decreased PPL response after aerobic exercise training, possibly due to improved free fatty acid, TG clearance, and increased LPL activity (13, 14, 25, 30, 38, 47). Tsetsonis et al. (46) observed a temporary reduction in TG concentrations in the fasted and postprandial state in both men and women due to an acute bout of aerobic exercise. A similar postprandial TG lowering effect was seen in individuals with the metabolic syndrome 11 hours after a 40, 60 and 70 percent maximum oxygen consumption (VO_2) exercise session (54). The PPL response was significantly lowered in each aerobic exercise condition by 30, 31, and 39 percent, respectively.

Endurance trained individuals demonstrate a lower lipemic response than untrained individuals; however, this may be attributed to the triglyceride lowering effects of their most recent bout of exercise, known as the receny effect (25, 47). The differences in the amount of PPL attenuation seen in various aerobic exercise trials may be due to the total amount of energy

expenditure (33, 46). In one study, researchers showed that the degree of reduction in the PPL response may be attributed to the type of exercise, the intensity of exercise and the exercise's total energy expenditure (33). Tstesonis et al. (46) observed that walking at either 32 percent VO_2 max for 3 hours or 63 percent VO_2 max for 1.5 hours produced similar results of a decreased PPL response, compared to a resting control condition. This provides evidence that even lower intensity exercise can provided health benefits for postprandial TG concentrations, which is has practical applications for the sedentary and/or obese populations who may not tolerate more intense exercise.

The majority of research has been conducted to examine the effect of aerobic exercise on PPL and it's related responses. However, on study conducted by Petitt and colleagues found that the TG AUC response was significantly lower after a resistance exercise trial compared to an aerobic exercise trial of equal energy expenditure, after adjusting for baseline differences (33). This leads to the theory that resistance exercise, despite being less researched, may have the ability to attenuate the PPHL response to a greater degree than aerobic exercise. Zotou et al. (56) examined a similar effect in a study composed of healthy untrained women. They found a significant decrease in both total plasma TG and TG-rich lipoproteins in a fasted and postprandial state following an acute bout of resistance exercise compared to a control trial. However, they did not observe a difference in fasting or postprandial non-TG-rich lipoproteins in either trial. Therefore, the 22-24% reduction in fasting and postprandial total TG was entirely due to the reduction in TG-rich lipoprotein concentration. Controversially, Shannon et al. (39) concluded that an acute bout resistance exercise failed to affect fasting or postprandial TG in resistance-trained women. In this study, the researchers controlled for the energy balance between the exercise and control trial by providing additional kilocalories after the resistance

exercise. The use of resistance trained women and the additional kilocalories provided in the exercise trial may have abolished the TG lowering effect of the exercise bout (23, 39, 56). Despite the inconclusive studies regarding the PPL response in women, the intensity or duration of an acute bout of resistance exercise does not appear to modify the TG response before or after a meal (39, 40).

Mechanism behind the ability of resistance exercise to attenuate the PPL response is still inconclusive. However, Petitt et al. (33) suggested that the higher intensity, lower repetition contractile activity associated with resistance training may increase LPL activity, which could attenuate the PPL response to a greater extent than less intense aerobic activity. Other mechanisms behind the decreased TG response in resistance exercise are similar to those for aerobic exercise, which include a possible increase in fat oxidation and/or increased TG clearance after exercise. Research also suggest that the PPL response after an exercise session is very individualized based on certain factors such as training status, fitness level, age, gender, weight, dietary habits, and genetic factors. As previously mentioned, trained individuals exhibit a better PPL response than untrained individuals, however, this may be entirely attributed to their most recent bout of exercise, instead of chronic physiological changes (25, 47). Weight seems to play a factor in the severity of the PPL response, with obese individuals producing a greater TG elevation than their lean counterparts; however, a single bout of exercise seems to blunt the PPL response in a similar fashion (5, 22, 34).

Only a few previous studies of fasting and postprandial TG metabolism include women in their subject population. Further studies on this topic utilizing women are necessary because women regulate TG metabolism differently than men and women have an increased risk for developing CVD due to hypertriglyceridemia (16, 34, 39). Compared to men, women have

lower total TG and VLDL-TG concentrations and greater VLDL-TG clearance rates, which resembles the changes in men after exercise (23, 56). Therefore, it is possible that exercise-induced hypotriglyceridemia occurs differently in women than in men. A possible mechanism behind this gender difference is a reduced rate of VLDL-TG secretion from the liver, which tends to be much greater in women compared to men (56). Further studies need to be conducted to observe the PPL response following resistance exercise in women, in younger individuals and in a healthy population.

Exercise and Inflammatory Markers

Exercise is known to improve a wide range of CVD risk factors, but results from past studies on the effects of exercise on inflammatory markers have been inconsistent (5, 6, 11, 14, 21, 30). Paton et al. (30) observed a postprandial increase in TNF-α after a high fat meal and this response was significantly blunted after 6 months of aerobic training at 50% and 70% VO_2Max, which indicates an improvement in inflammation. Paton et al. also observed that coagulation factors that reduce clotting and fibrinolysis, which increase the breakdown of fibrin material or blood clots, were both significantly reduced in the postprandial state after the 6 months of aerobic training. Harrison et al. (14) showed a postprandial increase in IL-6 and leukocytes after a high fat meal, but observed no difference in these variables after an aerobic exercise condition at 70% VO2Max compared to a control resting condition. However, leukocytes were higher the morning of the test meal in the exercise trial compared to the control trial and showed a small postprandial increase compared to the control trial. MacEneaney et al. (22) found similar responses in overweight and normal weight adolescent boys who showed an increase in IL-6 and leukocytes 6 hours after a high fat meal, but their was no difference between

the lean and obese groups or the aerobic or control conditions. These finding suggest that IL-6 and total leukocytes may be stimulated by a high-fat meal, but these effects do not produce significant differences in the presence or absence of an aerobic exercise session (14, 22). However, leukocytes alone can undergo an increase, known as leukocytosis, in response to a recent bout of exercise but this increase is not seen in the postprandial state (14).

Furthermore, MacEneaney's study of overweight and normal weight adolescent boys after a bout of aerobic exercise at 65% VO_2 max found no changes in CRP following the high-fat test meal, as well as no differences between the experimental conditions or weight classification (22). Researchers examining the postprandial effects of exercise on inflammatory markers have typically not included CRP as a variable. As previously mentioned however, CRP has been shown to negatively correlate with FMD (4, 17, 44, 45, 50). This correlation may indicate that CRP is an inflammatory marker that is affected due to chronic changes in an individual's health status such as the presence of obesity, long-term exercise training, and overall metabolic health. The presence of a single high-fat meal or an acute bout of exercise has not been shown to affect CRP (7, 14, 22).

Fewer research studies have been conducted using resistance exercise as a mechanism to blunted the postprandial increases in inflammatory cytokines. However, a few studies have noted a resistance exercise-induced leukocytosis (24, 36). McFarlin et al. observed leukocytosis in postmenopausal women after an acute bout of resistance exercise and an increase in natural killer cell activity after resistance exercise training (24). A study of trained and untrained young men also saw a resistance exercise-induced leukocytosis as well as a decrease in natural killer cells, independent of training status (36). Despite inconclusive and minimal research over the effects of inflammatory markers in the postprandial state, exercise has been shown to positively

effect inflammation in some cytokines. However, these effects might only be seen after long-term exercise training and increased health status in certain inflammatory markers such as CRP. Also, overweight or obese and untrained individuals may not experience as great of a beneficial effect due to acute bouts of exercise compared to their lean and trained counterparts. Further studies involving inflammatory cytokines are necessary to better understand the relationship between exercise, particularly resistance exercise, and inflammation during the postprandial state.

Summary

CVD is the number one killer of Americans to date. It is known to be a multifactor disease with many contributing risk factors including genetic predispositions, a high-fat diet, physical inactivity, obesity, hypertension, dyslipidemia, hypertrigylceridemia, insulin resistance, and glucose intolerance (9, 34). Although research suggest that behavioral risk factors such as physical inactivity and poor dietary habits are an important contributing factors in the development of CVD, more research is needed to discover the mechanism behind how this occurs. Due to the typical American diet, many individuals spend most of the day in the postprandial hyperlipemic state (20). Such a continual elevation in postprandial triglycerides are linked to ED due to reduced vasodilatation, disturbances in lipoprotein metabolism, increased expression of inflammatory markers, and the formation or progression of atherosclerotic plaques (4, 15, 17, 37, 48). Together these symptoms can cause the slow progression of atherosclerosis and CVD over the course of many years.

Modalities that are able to blunt the PPHL response, such as aerobic or resistance exercise, are believed to lower the risk of CVD by increasing endothelial function (34, 47).

Minor decreases in the postprandial TG and inflammatory responses arising from moderately intense exercise, most days of the week, could have an important cumulative effect on endothelial function and CVD risk factors in the long term. Even apparently healthy young adults are affected by PPHL, especially in obese or overweight individuals. With continued research into aerobic and especially resistance exercise as a therapeutic modality to reduce the PPL response in all types of populations, preventive exercise programs could be formed with an enhanced understanding of how exercises attenuates the risk factors directly related to CVD, ED and atherosclerosis.

Chapter III
Methods

Participants

Nine sedentary lean and 10 sedentary obese women between 18 and 28 years of age participated in the study. For the purpose of this study, sedentary subjects were operationally defined as individuals who do not participate in a structured exercise program on a consistent basis, or no more than twice per week, for at least 6 months prior to testing. The mean age of the lean and obese participants was 20.22 $\pm$ 1.20 years and 22.60 $\pm$ 3.47 years, respectively. The mean body fat percentage of the lean and obese participants was 25.61 $\pm$ 3.26% and 35.00 $\pm$ 3.57%, respectively. Participants in both the lean and obese groups represented a variety of ethnicities, including 8 African Americans, 2 Asians, and 9 Caucasians. Exclusion criteria for the study consisted of diseases such as hypertension, diabetes mellitus, cardiovascular disease, amenorrhea or an eating disorder. Also, individuals who smoke tobacco, those on a special diet and/or those unable to exercise due an injury or long-term illness were excluded from the study. Excluded medications for the study included those for hyperlipidemia, hypertension, and/or hormonally based contraceptives. Participant's food preferences due to allergies or aversions to certain types of food were accommodated as long as the nutritional content of the high-fat meal could be maintained; otherwise, the participant was excluded from the study.

Experimental Protocol

Human Subjects approval was obtained prior to subject recruitment and data collection. Participants were recruited from the university campus and surrounding community via flyers and word-of-mouth. Advertisements were also posted in the university's bi-weekly mass emails, which are sent to all students, faculty and staff. Participants read and signed a consent form and

health information privacy form before participating in the study. They also completed a medical history questionnaire to indicate any contraindications to exercise. If any contraindications were found, a physician cleared the individual before they could participate in the study.

Experimental Design

This study utilized a three-factor design with a between group comparison of the lean and obese participants and a repeated measures analysis for time point and trial condition. Both groups participated in two experimental trials administered in a randomized order: a resistance exercise trial (EXS) and a resting control trial (CON). The trials consisted of either a resistance exercise session or rest session during the afternoon of Day 1 and then the consumption of the high-fat test meal on the morning of Day 2, followed by a 6-hour test period. In order to control for hormonal fluctuations, the participants completed both trials during the first 10 days of the follicular stage of their menstrual cycle, when estrogen levels are at their lowest. The follicular phase begins on the first day of menstruation and usually last 13 to 14 days; therefore, the two experimental trials were separated by at least 21 days.

Preliminary Testing. All preliminary testing occurred 1 to 3 weeks prior to the first experimental trial, depending on the randomized order of the trials. Participants first reported to the Texas Christian University Exercise Physiology Lab for anthropometric measurements. Participant's nude body weight was measured on an electronic scale (Nicol Scales Inc, Dallas, TX) to the nearest 0.1 kilogram. Height was measured on a stadiometer (Detecto, Webb City, MO) to the nearest 0.1 centimeter. Body mass index was determined using the subject's height and weight (kg/m^2). Body fat percentage was determined by Jackson-Pollock's seven-site skin

fold equation using the following sites measured to the nearest millimeter: tricep, subscapular, chest, mid-axiallary, abdominal, suprailiac, and thigh (2). Waist to hip ratio was measured to the nearest inch using a body measuring tape. The waist was defined as the smallest area below the rib cage and the hip was defined as the largest area of the buttocks (2). Participants were placed into the lean or obese groups if two of their three body composition measurements (BMI, waist to hip ratio, or percent body fat) categorized them as lean or obese. Ranges that were accepted for the lean and obese categories of these three anthropometric measures are listed below (Table 1).

Table 1. Anthropometric Categories.

	Lean	**Obese**
Body Max Index	18.5 - 25.0	30.0 - 45.0
Body Fat Percentage (%)	21.0 - 32.9	$\geq$ 39.0
Waist-to-Hip Ratio	$\leq$ 0.79	$\geq$ 0.85

On the same day, each participant was given a 24-hour food log with written and verbal instructions to thoroughly record their diet during Day 1 of their first experimental trial. They were asked to eat a typical diet that they could easily repeat on Day 1 of their second trial. The 24-hour food log was analyzed after the first experimental trial and returned to the participant for them to follow before their second trial.

At least two weeks prior to experimental testing, participants were introduced and familiarized with the resistance exercise machines used in the study, as well as tested for strength in Texas Christian University's Recreation Center. First, participants performed a 1-repetition maximum (1-RM) test of the 10 exercises in the resistance exercise protocol. The 10 exercises

address the total body and include: bench press, latissimus dorsi pull-down, seated row, shoulder press, shoulder fly, leg press, leg curl, leg extension, leg abduction, and leg adduction. The strength test was conducted by the ACSM 1-RM testing protocol consisting of one warm up set of 8 to 10 repetitions, followed by a second warm up set of 3 to 5 repetitions, and finishing with a progression in weight until a 1-RM is reached (2). At least 5 days following the strength assessment, the participants completed a practice workout session that included 3 sets of 8 to 12 repetitions at seventy percent of their pre-determined 1-RM weight. The exercises were structured in an alternating push-pull circuit, where two exercises were alternated until 3 sets of each were preformed. The exercises were ordered as follows: bench press/lat pull down, overhead press/seated row, leg press/shoulder fly, leg extension/leg curl, and abduction/adduction. Weight was adjusted as needed to complete at least 3 sets of 8 repetitions for each exercise. Neither the EXS or CON trial was conducted until at least a 7-day washout period following the practice workout session. Participants were monitored throughout each exercise session by a certified personal trainer.

Experimental Testing. Participants completed both an EXS and a CON 2-day trial separated approximately by 28 days. Day 1 consisted of initial assessments followed by the exercise bout or rest period and Day 2 involved baseline assessments prior to the consumption of the high-fat test meal, followed by a 6-hour testing period (see Figure 1). Participants were asked to abstain from any form of exercise, alcohol, nicotine, and caffeine 48 hours before each experimental trial. The participants were instructed to start recording their 24-hour food log on the morning of Day 1 of their first trial and to repeat this diet exactly on Day 1 of their second trial.

Day 1		**Day 2**		
Exercise or Control (4-6 PM)	14 to 16-hr Interval w/ Evening Meal	Baseline Sample (7-9 AM)	Test Meal (20 min)	6-hr PPL Test Period
Pre ↑ ♥		Base ↑ ♥		.5 ↑ 1 ♥ 1.5 ↑ 2 ♥ 3 ↑ 4 ♥ 5 ↑ 6hr ♥

Figure 1. Experimental Trial Timeline. (↑ = blood sample, ♥ = FMD analysis)

On Day 1 of both trials participants reported to the Exercise Physiology Lab between 4 and 6 pm for an initial blood sample and an assessment of endothelial function via a flow-mediated dilation (FMD). FMD was preformed with an Acuson ultrasound device using a color Doppler with an L5 or L10 transducer (Aspen, Acuson, Mountain View, CA). Transducer size was determined based on arm circumference of the participant. The FMD assessments consist of the participant lying in a supine position with their forearm resting on a stabilization pad and palm facing upward. The brachial artery of the upper arm was found using the appropriate size transducer and an 8-second video clip was taken. The position of the transducer was marked on the participant's skin in order to record the same location of the artery on each FMD assessment. Then the brachial artery is occluded for five minutes with a standard blood pressure cuff placed around the subject's forearm and inflated to 50 mmHg above the participant's systolic blood pressure. Upon the release of the cuff, 8-second video clips were recorded every 15 seconds for

3 minutes, totaling 13 video clips for the entire assessment. Assessments were stored on an MO-disk for later analysis.

After the initial blood sample and FMD assessment on Day 1 the participants either reported to the university's recreation center for the EXS trial resistance training or they rested in the lab for a comparable period of time during the CON trial. For the resistance exercise workout, participants completed the resistance exercise routine described above. After the exercise or resting session was completed, participants consumed a dinner of their choice before beginning a 12-hour overnight fast. Participants Total Volume Load (TVL) for each exercise was calculated by multiplying the weight they lifted in pounds, by 3 sets and 10 repetitions. The sum of the TVL for the 10 exercises was determined for each participant to obtain a TVL for the entire exercise session.

On Day 2 of the experimental trials, participants reported to the Exercise Physiology Lab 16 hours after the exercise or control resting session and at least 12 to 14 hours after their last meal or snack. Both the EXS and CON trials followed the same protocol for Day 2. Upon arrival to the lab, participants rested in a supine position for 10 minutes before a catheter was inserted into the antecubital vein for blood sampling (Becton Dickinson Saf-T-Intima, Sandy, UT). The baseline blood sample was drawn off the catheter after which the catheter was cleared with a saline flush. Then the baseline FMD measurement was obtained from the arm without the catheter.

After these samples were taken, participants consumed the high fat test meal in 20 minutes or less. The meals consisted of approximately 50 percent kilocalories (kcals) from fat (with 25 percent coming from saturated fat), 35 percent kcals from carbohydrate (mainly starch and refined sugar) and 15 percent kcals from protein. The meal was designed to provide a total

of 15 kcals/kg body weight for four predetermined 10-kg weight ranges (55-64.9 kg, 65-74.9 kg, 75-84.9 kg, and 85-94.9 kg). The food components of each high-fat test meal were pre-analyzed for weight, kilocalories, total fat, saturated fat, protein and carbohydrates using the Food Processor software (Viocare Inc., Princeton, NJ). The specific nutrient content of each pre-planned test meal is shown below in Table 2.

Table 2. High-Fat Test Meal Compositions.

55-64.9 Kg			**65-74.9 Kg**			**75-84.9 Kg**			**85-94.9 Kg**		
Kcal est: 900	**Kcal act: 920**		Kcal est: 1050	**Kcal act: 1069**		Kcal est: 1,200	**Kcal act: 1205**		Kcal est: 1350	**Kcal act: 1345**	
	Grams	**Pieces**		**Grams**	**Pieces**		**Grams**	**Pieces**		**Grams**	**Pieces**
Eggs	100	2	**Eggs**	100	2	**Eggs**	170	3+	**Eggs**	155	3+
Egg Whites	100	N/A	**Egg Whites**	120	N/A	**Egg Whites**	100	N/A	**Egg Whites**	135	N/A
Waffle	70	2	**Waffle**	70	2	**Waffle**	70	2	**Waffle**	105	2 1/2
Bacon	14	2 1/3	**Bacon**	22	3 2/3	**Bacon**	18	3	**Bacon**	21	3 1/3
Syrup	70	N/A	**Syrup**	95	N/A	**Syrup**	100	N/A	**Syrup**	105	N/A
Butter	40	N/A	**Butter**	45	N/A	**Butter**	52	N/A	**Butter**	55	N/A

The high fat meal consisted of Eggo® Homestyle Frozen Waffles, Hormel® Microwaveable bacon, Land o' Lakes® salted butter, Log Cabin® syrup, whole eggs, and egg whites (Table 3). It was prepared by the researchers in the Metabolic Lab Kitchen on the morning of Day 2 and was based on the participant's weight range and food preferences, if applicable. Participants were instructed to eat the entire meal; however; if they could not finish

the meal, they were then encouraged to eat as much as they could tolerate. Any remaining food was measured on a scale to the nearest gram, in order to ensure that the participant was given the same amount of food during their second trial. Participants were allowed to consume water ad-libitum throughout their first trial and were instructed to consume the same amount during their second trial.

Table 3. High-Fat Test Meal Ingredients.

Food	Brand	Wt (g)	Kcal	Fat (g)	Pr (g)	CHO (g)
Eggs	n/a	50	74	4	6.29	1.36
Egg whites	n/a	100	52	1.53	10.9	0.73
Waffle	Eggo®, Homestyle	70	190	6	4	29
Bacon	Hormel®, Microwaveable	12	60	45	3.7	0.1
Syrup	Log Cabin®	80	200	0	0	52
Butter	Land o' Lakes®, salted	14	100	11	0	0

Following the completion of the meal, blood samples were collected from the catheter every 30 minutes for the first 2 hours and then every hour for the next 4 hours. Therefore, post-meal blood sample time points included .5-hour, 1-hour, 1.5-hour, 2-hour, 3-hour, 4-hour, 5-hour and 6-hour. Post meal FMD assessments were obtained immediately after the blood draws at 1-hour, 2-hours, 4-hours, and 6-hours post meal.

Blood Analyses

Plasma samples were collected in cold 5-mL EDTA tubes and serum samples were collected in cold 5-mL non-heparinized tubes then immediately placed back on chilled beads. Approximately 10 ml of blood was collected at each sampling time point. Collected samples were centrifuged at 2000 RMPs at 4^0 C for 10 minutes, aliquoted into 1.7 mL micro-tubes and frozen at -80 ºC. Total triglyceride (TG) concentrations were analyzed from plasma samples using a standard enzymatic spectrophotometric assay method. The inflammatory markers Interleukin-6 (IL-6) and C-Reactive Protein (CRP) were analyzed from plasma samples via a sandwich-like enzymatic immunoassay technique with commercially available kits (Quantikine, R&D Systems Inc., Minneapolis, MN). Nitrite/Nitrate were analyzed from plasma samples via commercially available Colorimetric assay kits (Caymen Chemical Co., Ann Arbor, MI). Total leukocytes and their 5-part differential counts were determined by plasma samples via an automated hematology analyzer (AcTdiff2, Beckman Coulter, USA). All samples from a single participant will be analyzed in the same batch.

Endothelial Function Analyses

The degree of the reactive hyperemic response, or change in vessel diameter, is the determinant of endothelial function and expressed as an absolute and a percent change in diameter. The artery diameters for each 8-second clip of the FMD assessments were determined using the Brachial Analyzer for Research software (5.8.7.SP1, Medical Imaging Applications LLC., Coralville, IA). The percent diameter change between the initial artery diameters before occlusion and the peak artery diameters after occlusion were calculated for each FMD time

point. This data was expressed as an absolute change in diameter (Delta FMD) and percent change in diameter (% Change FMD).

Statistical Analyses

A three-factor analysis of variance (ANOVA) was used for each dependent variable to determine differences between weight group (lean and obese), between conditions (EXS and CON), and over time (multiple sampling points depending on the variable). Specifically, a 2 x 2 x n, group by condition by time ANOVA with repeated measures on the condition and time factors was used. The number of levels of the repeated measures for the time factor varied from five to eight depending on the dependent measure. The dependent variables measured were blood levels of TG, nitrite/nitrate, IL-6 and CRP, total leukocytes with 5-part differentials and FMD responses expressed in absolute and relative units. A Huynh-Feldt correction for repeated sampling was applied to these analyses. The Newman Keuls *post hoc* analysis was used to isolate differences detected by the ANOVAs. TG area under the curve (AUC) was calculated using the trapezoidal rule and was analyzed using a two-factor ANOVA. Correlational analyses were also conducted to determine relationships between relevant dependent variables. Results are presented as means and standard deviations with significance accepted at $p < 0.05$.

Chapter IV
Results

Anthropometrics

Anthropometrics for the lean and obese groups are listed below in Table 4. The average age and height of the participants were not significantly different between the lean and obese groups. The lean group had a significantly lower weight, BMI and body fat percentage compared to the obese group (p=0.000), as well as a significantly lower waist to hip ratio (p=0.001). The lean group also had a significantly lower resting systolic blood pressure compared to the obese group (p=0.048); however, the resting diastolic blood pressures were not significantly different between groups.

Table 4. Participant Anthropometrics.

	Lean (Mean ± SD)	**Obese** (Mean ± SD)
Group Size	n = 9	n = 10
Age (years)	20.22 ± 1.20	22.60 ± 3.47
Height (cm)	168.67 ± 6.14	164.92 ± 6.36
***Weight** (kg)	67.35 ± 11.12	94.08 ± 11.50
***BMI** (kg/m^2)	23.58 ± 3.10	34.57 ± 3.30
***Body Fat %**	25.61 ± 3.26	35.00 ± 3.57
***Waist-to-Hip**	0.76 ± 0.06	0.86 ± 0.03

The * indicates significantly different group variables.

Dietary Intake

Based on the 24-hour food logs participants recorded on Day 1 of their first trial, the following nutritional data were obtained, which represents the participant's typical dietary intakes (Table 5). The average 24-hour kilocalories consumed by the lean and obese participants were not significantly different. There was not a significant difference in the percent of total kilocalories coming from carbohydrates, fat or protein consumed by the lean and obese group. Also, the percent of kilocalories coming from saturated fat was not significantly different between groups.

Table 5. 24-hour Food Log Nutritional Data.

	Lean (Mean ± SD)	**Obese** (Mean ± SD)
Total kcals	1,958.28 ± 856.96	2,138.48 ± 717.00
% Carbohydrate	50.67 ± 5.48	50.38 ± 6.16
% Fat	32.89 ± 5.06	35.38 ± 6.09
% Protein	16.44 ± 4.13	14.25 ± 2.92
% Saturated Fat	9.93 ± 4.43	10.07 ± 3.93

High-Fat Test Meal

Participants received approximately 15 kcal/kg body weight in the high-fat test meal during their first trial. Any leftovers were measured on a scale in grams so that participants received an equal amount of food during their second trial. There was not a significant

difference in leftover grams of food between the lean and obese participants, 96.61 ± 102.37g and 62.50 ± 76.13g, respectively (Figure 2).

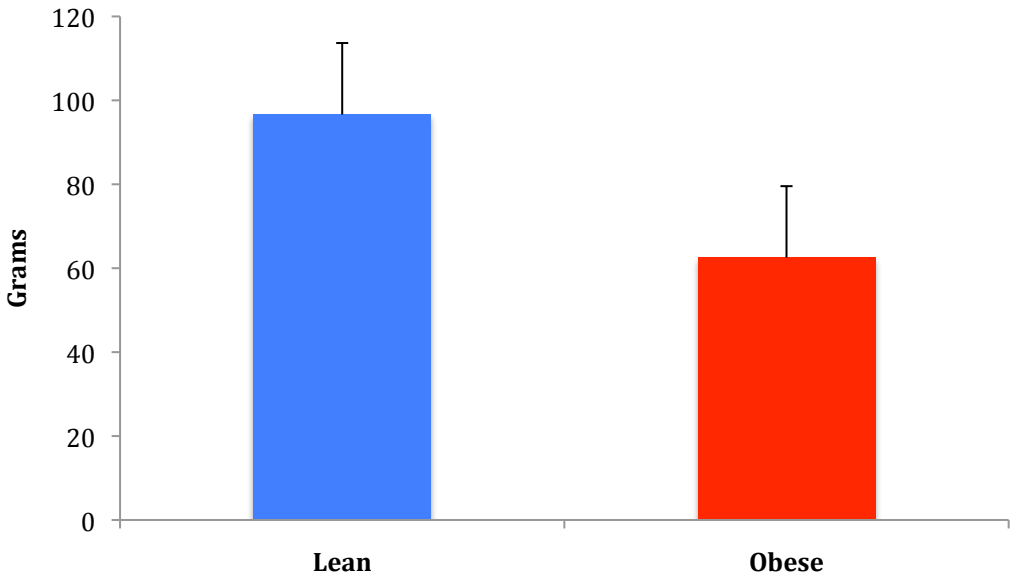

Figure 2. High-Fat Test Meal Leftovers. Values expressed as mean ± SE.

Resistance Exercise Session

Participant's TVL for each exercise was calculated by multiplying the weight lifted in pounds, by 3 sets and 10 repetitions. The sum of the TVL for the 10 exercises was determined for each participant to obtain a TVL for the entire exercise session. The average TVL for the lean and obese participants was not significantly different (22,963 ± 5,149 lb and 25,800 ± 5,548 lb, respectively) (Figure 3).

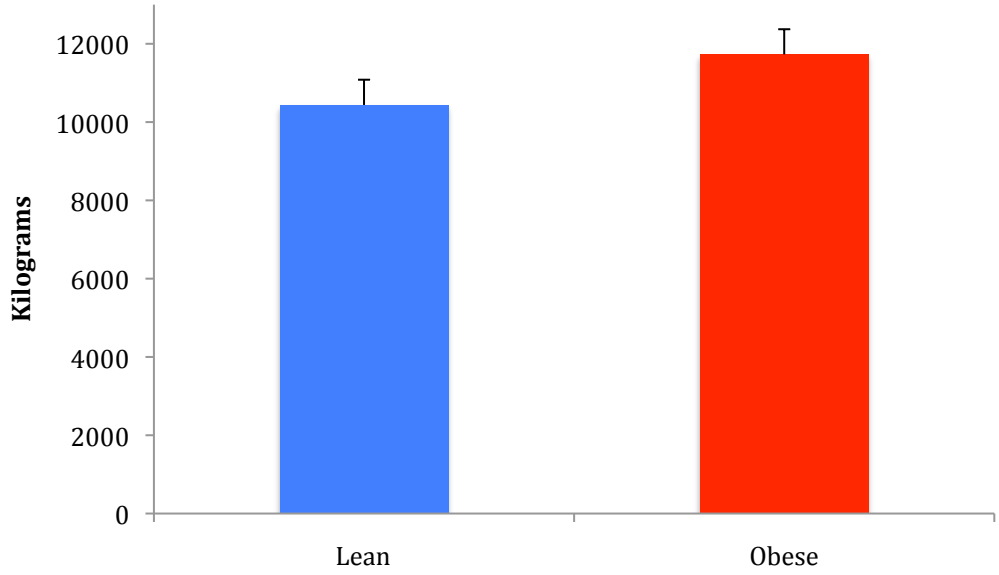

Figure 3. Total Volume Load of the Resistance Exercise. Values expressed as mean ± SE.

Triglyceride Responses

For triglyceride concentration, there was a significant main effect for group (p=0.019), with the lean having a lower TG concentration than the obese at all time points. There was a significant main effect for time (p=0.003). Post hoc testing revealed the Base time point was significantly lower than all other postprandial time points, except for the final 6-hr time point (Figure 4).

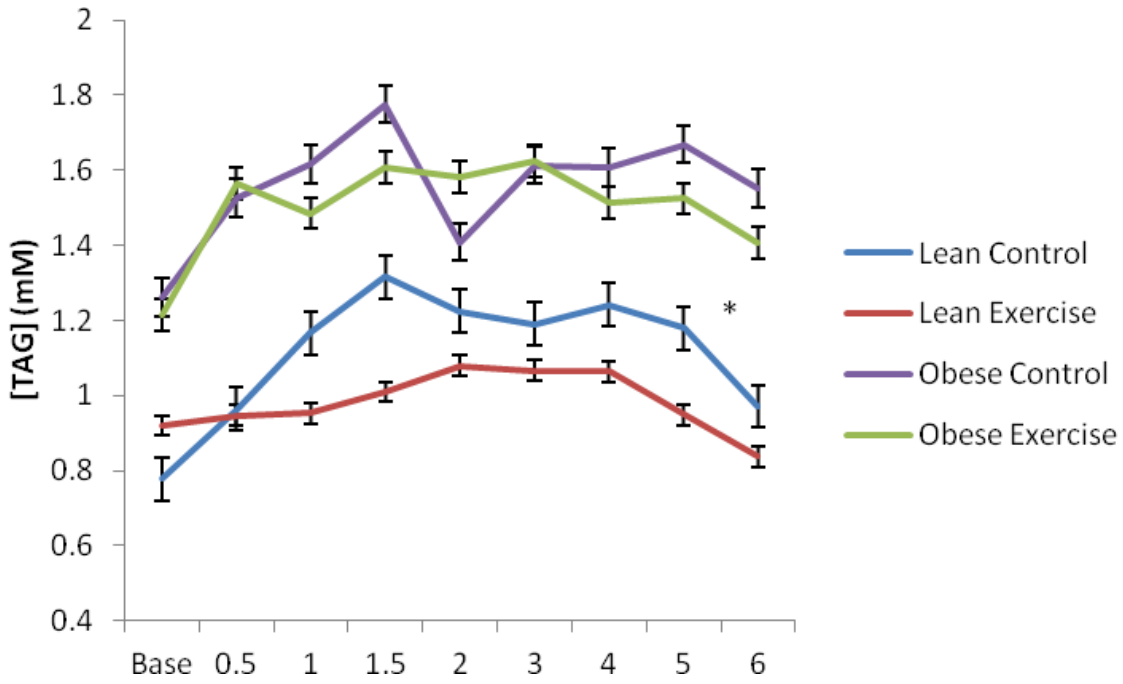

Figure 4. Triglyceride vs. Time Point. The * indicates the lean group is significantly lower than the obese group at all time points ($p = 0.019$). Values expressed as mean $\pm$ SE.

The total area under the triglyceride postprandial curve (AUC) had a significant main effect for group ($p=0.028$), due to both lean conditions being lower than the obese conditions (Figure 5).

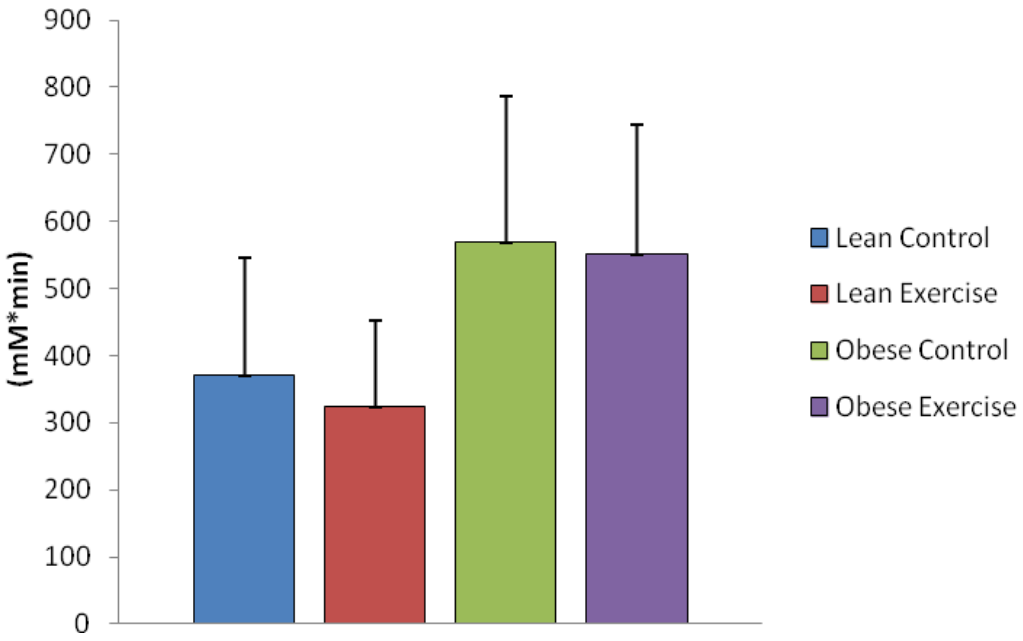

Figure 5. Triglyceride AUC. The * indicates that the lean group was significantly lower than the obese group. Values expressed as mean ± SE.

Flow-Mediated Dilation

There were no significant differences between group, condition, or time point for % change FMD (Figure 6). An ANOVA including just the Pre and Base time points of % change FMD was conducted to observe the effects of the resistance exercise, independent of the influence of the meal. This revealed a significant main effect for condition ($p=0.009$) due to a greater increase in % change in diameter in the EXS condition compared to the control condition (Figure 6). Also, % change FMD from Pre to Base approached a significant interaction effect for condition by group ($p=0.075$) with the Lean EXS condition having the largest diameter change.

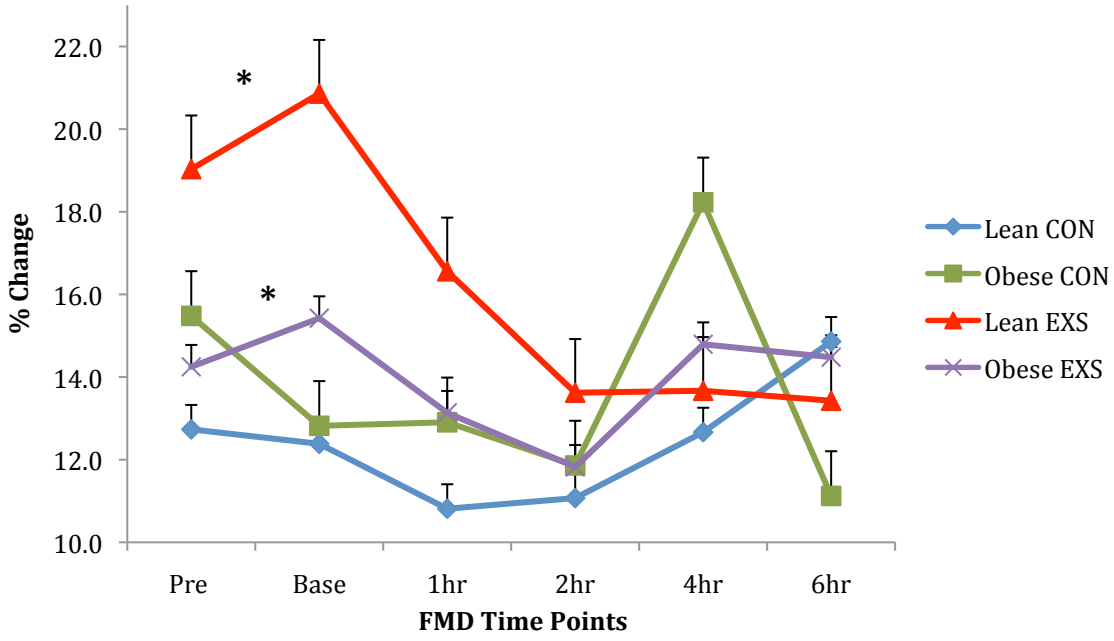

Figure 6. FMD Percent Change vs. Time Point. The * indicates a significant effect for condition for the Pre to Base time points only. Values expressed as mean ± SE.

The AUC for the 12 post-occlusion FMD clips in each time point was obtained to observe the total effect of dilation within that time (Figure 7). There was a significant main effect for weight group ($p=0.035$) due to the lean group having a lower AUC for both conditions across all time points. FMD AUC also had a significant main effect for condition ($p=0.034$)

because the CON conditions for each group had a lower AUC compared to the EXS conditions.

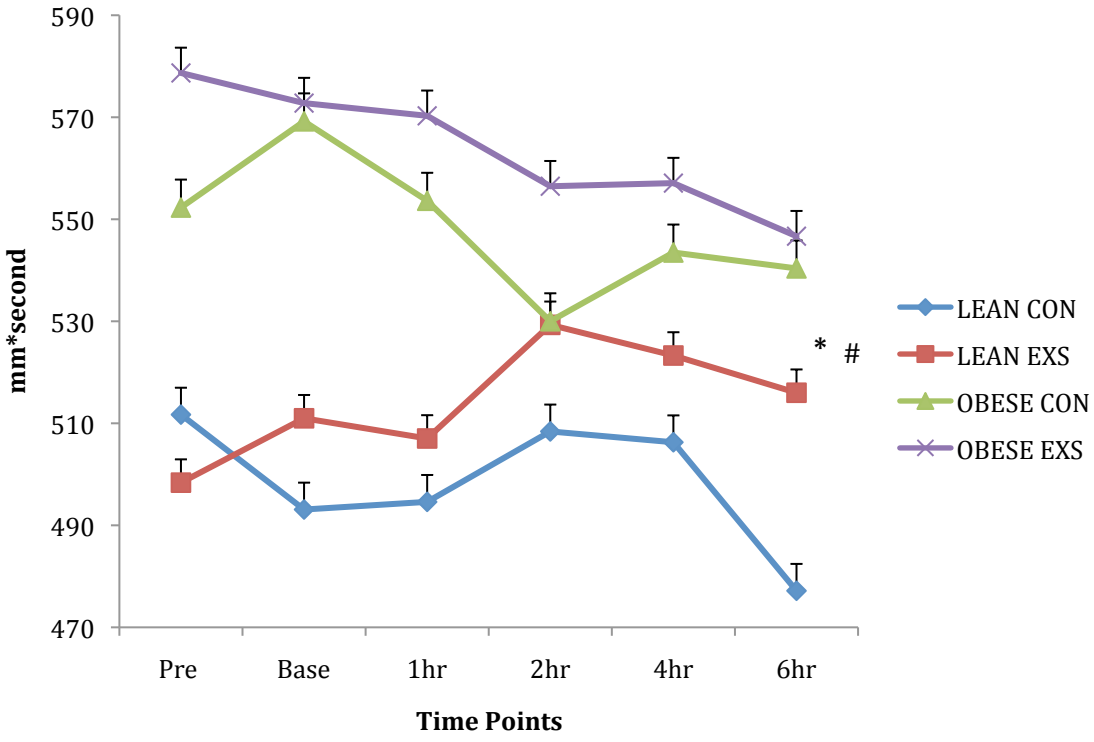

Figure 7. FMD AUC. The * indicates the lean conditions was significantly lower than the obese conditions ($p = 0.034$). The # indicates that the CON condition was significantly lower than the EXS conditions ($p = 0.034$). Values expressed as mean $\pm$ SE.

Nitrite & Nitrate Responses

Nitrite/Nitrate levels were not significantly different for group, condition or time but there was a tendency for an interaction effect for condition by time ($p=0.057$) and for condition by group by time ($p=0.051$) (Figure 8).

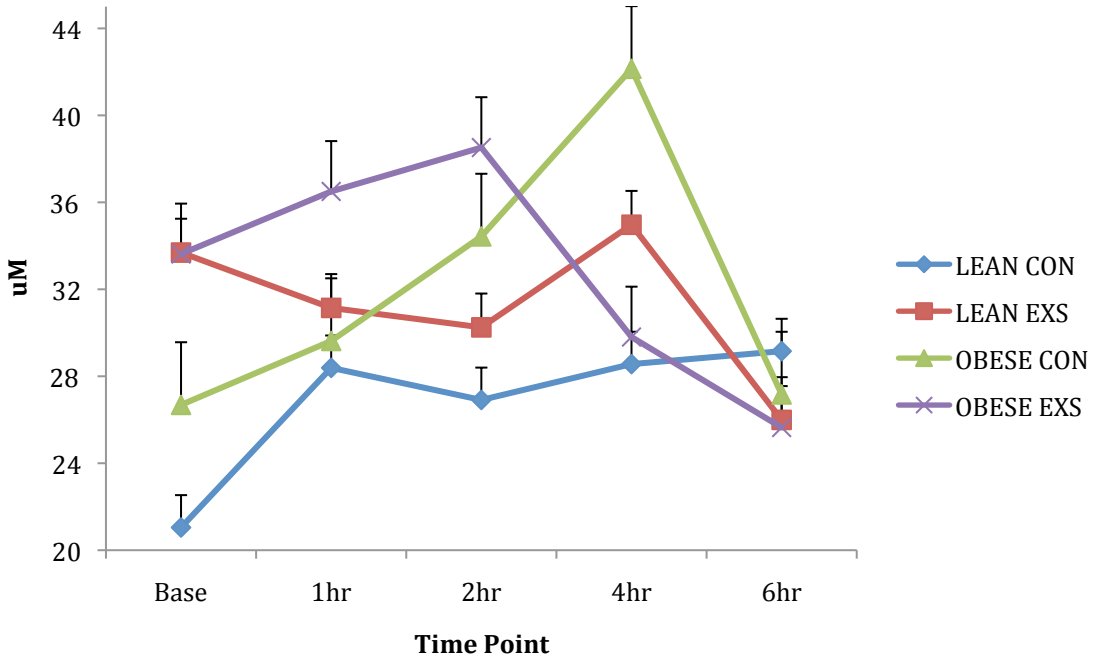

Figure 8. Nitrite/Nitrate vs. Time Point. Values expressed as mean ± SE.

Interluekin-6 Responses

For IL-6 concentration, there was a significant group by condition by time point interaction for IL-6 (p=0.019). The Newman Keuls pairwise analysis found that this significance difference is seen at the 6-hr time point where the obese CON condition (8.33 ± 3.00 pg/mL) is significantly greater than the obese EXS condition (5.41 ± 4.22 pg/mL) and the lean CON condition (5.13 ± 4.71 pg/mL). In addition, there was a significant main effect by time (p=0.000) (Figure 9). A post hoc analysis showed that the Base, .5-hr, and 1-hr time points were significantly lower than all the following postprandial time points.

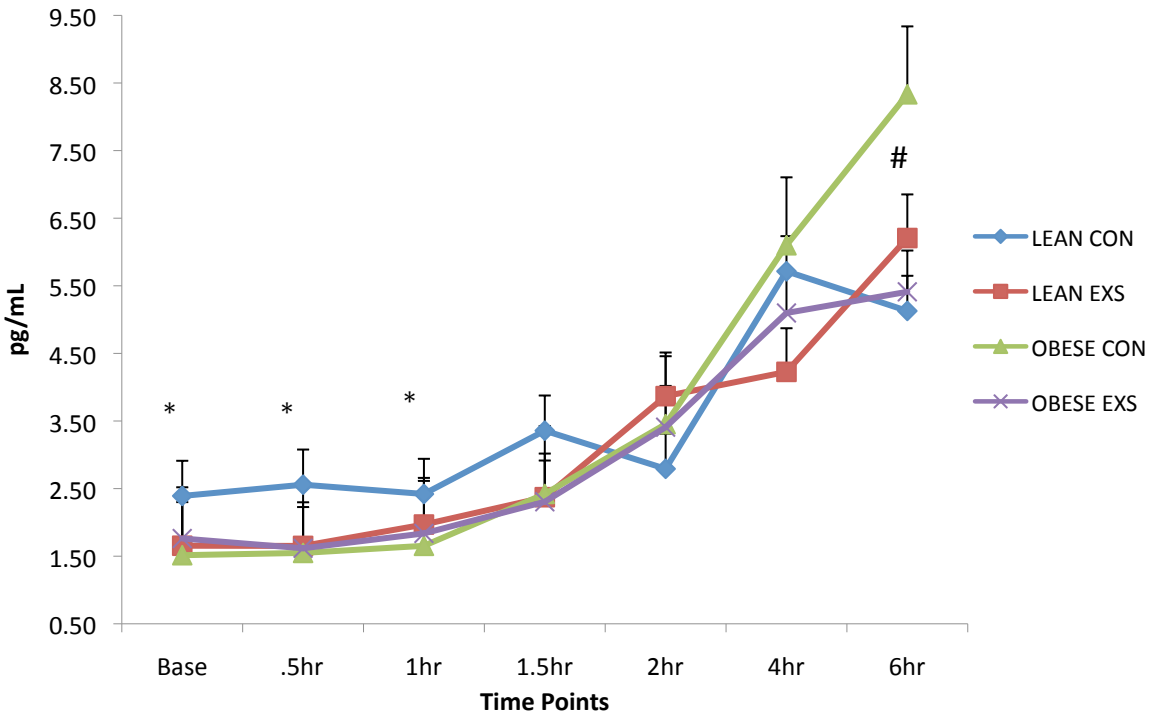

Figure 9. IL-6 vs. Time Point. The # indicates the Obese CON condition is significantly higher than the Obese EXS and Lean CON conditions. The * indicates that these time points are significantly lower than all others. ($P < 0.019$) Values expressed as mean $\pm$ SE.

C-Reactive Protein Responses

There was a significant main effect for group ($p=0.014$) for CRP with the lean having significantly lower concentrations than the obese across all time points (Figure 10).

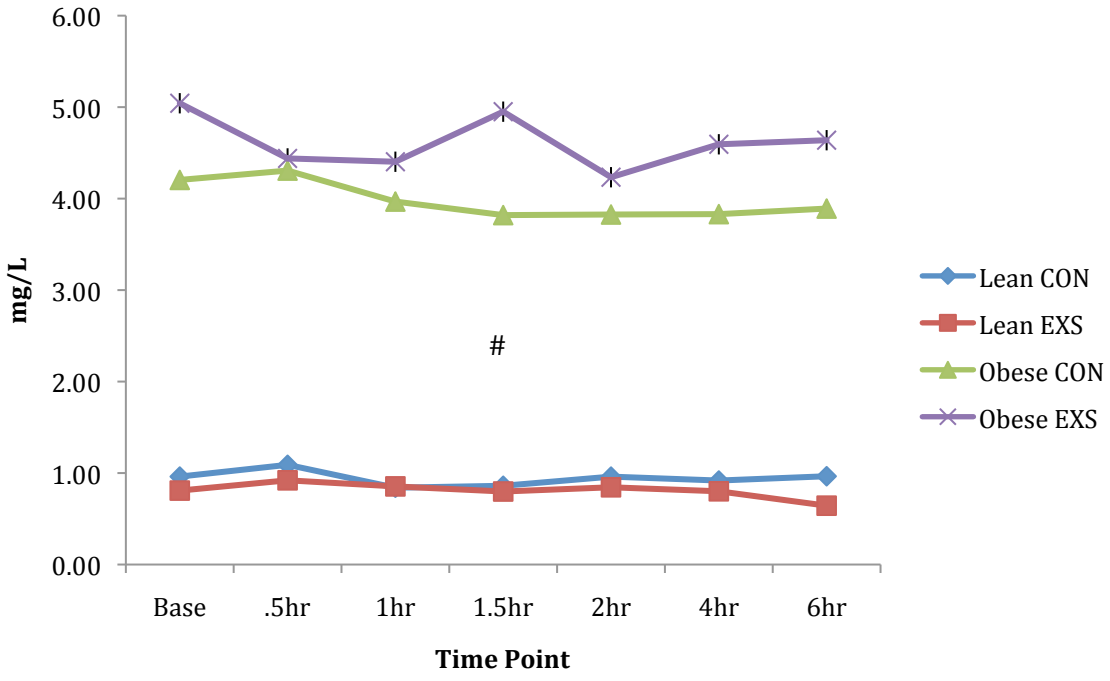

Figure 10. CRP vs. Time Point. The # indicates the obese time points are significantly different than the lean time points. Values expressed as mean ± SE.

Leukocytes & 5-Part Differential Responses

The total leukocytes had a significant main effect for time (p=0.000) (Figure 11). A post hoc analysis showed that the Pre time point was significantly higher than the Base, 1-hr, 2-hr and 3-hr time points and that the Base time point was significantly lower than the 3-hr through 6-hr time points. An ANOVA excluding the Pre time point also revealed a main effect for time (p=0.000). An ANOVA including just the Pre and Base time points was conducted to observe the effects of the resistance exercise, outside of meal influence, but only a significant main effect for time was found (p=0.000).

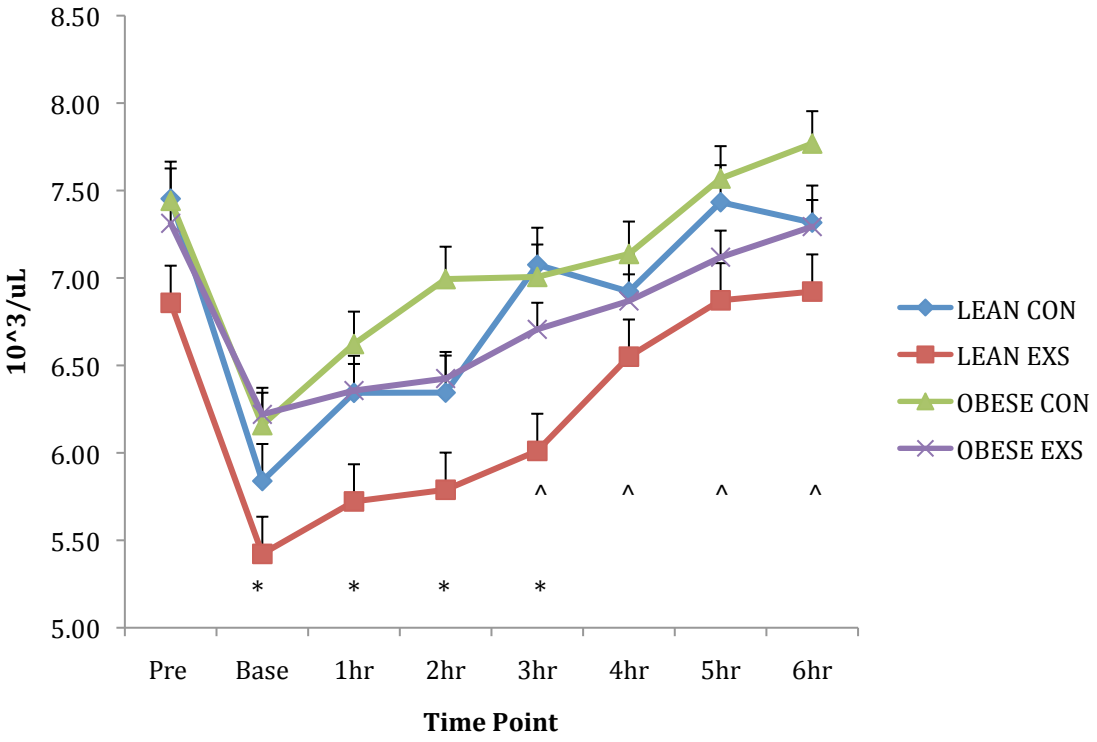

Figure 11. Total Leukocyte Count vs. Time Point. The * indicates a significant difference from Pre. The ^ indicates a significant difference from Base. Values expressed as mean ± SE.

The neutrophils showed a significant main effect for condition (p=0.035) with the EXS conditions in both groups being lower than the CON conditions at all time points (Figure 12). There was also a significant main effect for time (p=0.000) and a post hoc analysis showed that the Pre time point was significantly higher than the Base time point and that the Base time point was significantly lower than all other postprandial time points. An ANOVA excluding the Pre time point also revealed a significant main effect for condition (p=0.039) and a significant main effect for time (p=0.000).

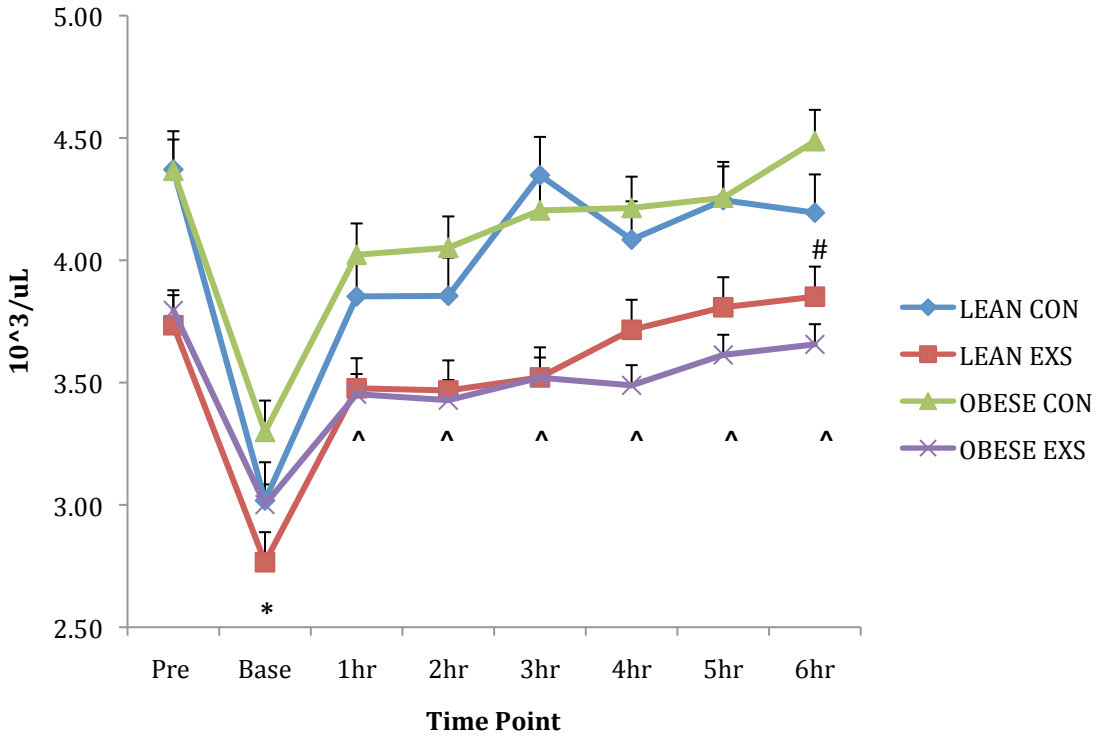

Figure 12. Neutrophil Count vs. Time Point. The * indicates a significant difference from Pre. The ^ indicates a significant difference from Base. The # indicates a significant difference between CON and EXS conditions in both groups. Values expressed as mean ± SE.

The lymphocyte count showed a significant main effect for time (p=0.000) (Figure 13). A post hoc analysis showed that the Pre time point is significantly higher than the 1-hr, 2-hr, and 3-hr time points. The Base time point is significantly higher than the 1-hr and 2-hr time points but significantly lower than the 5-hr and 6-hr time points. Also, the 1-hr time point is significantly lower than all the remaining postprandial time points.

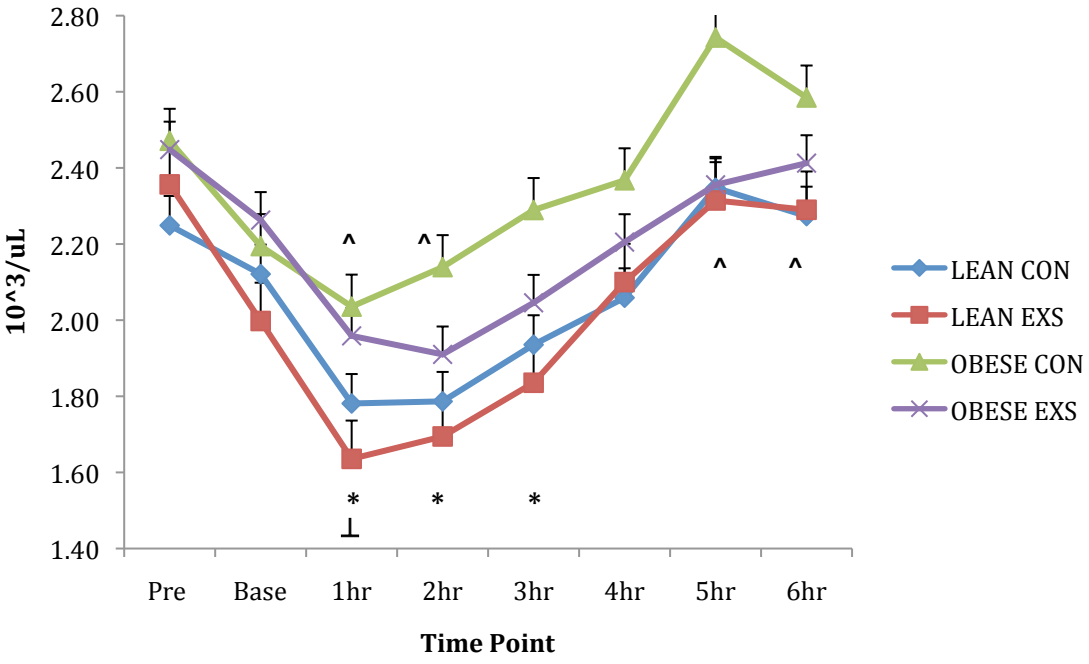

Figure 13. Lymphocyte Count vs. Time Point. The * indicates time points significantly different than Pre. The ^ indicates time points significantly different than Base. The ⊥ indicates the 1-hr time point is significantly different than all other postprandial time points. Values expressed as mean ± SE.

A significant main effect for time was seen for the monocyte count (p=0.000) (Figure 14). A post hoc analysis found that the Pre time point was significantly higher than the Base and 1-hr through 4-hr time points. Also, the Base, 1-hr and 2-hr time points were significantly lower than the 4-hr, 5-hr and 6-hr time points.

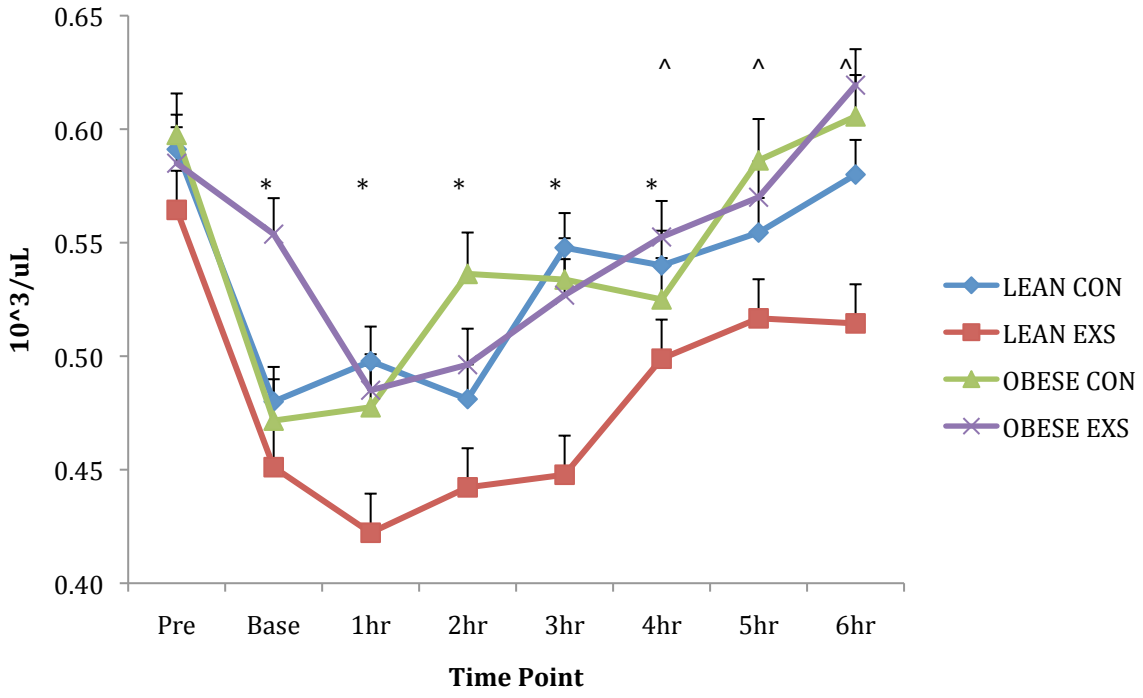

Figure 14. Monocyte Count vs. Time Point. The * indicates time points significantly different than Pre. The ^ indicates time points significantly different than Base, 1-hr and 2-hr time points. Values expressed as mean ± SE.

There was a main effect for time point (p=0.000) for the eosinophil count (Figure 15). A post hoc analysis showed that Pre was significantly higher than 1-hr and 2-hr time points and that Base is significantly lower than the 5-hr and 6-hr time points. Also, the 1-hr and 2-hr time points were significantly lower than the 3-hr through 6-hr time points. Furthermore, a significant interaction effect is seen between trial condition and time (p=0.007). The Newman Keuls pairwise analysis found that this significance difference is seen at the 2-hr, 3-hr and 5-hr time points where the EXS conditions are significantly lower than the CON conditions in both groups.

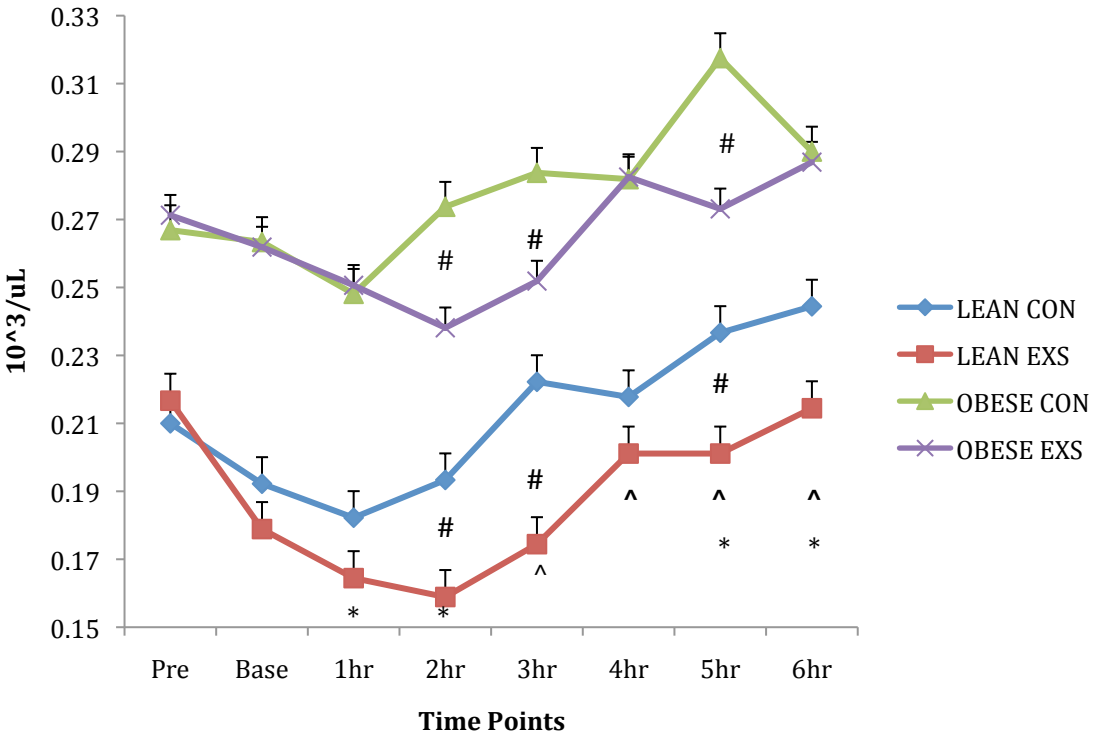

Figure 15. Eosinophil Count vs. Time Point. The * indicates a significant difference from Pre. The ^ indicates a significant difference from Base. The # indicates a significant difference between CON and EXS conditions in both groups. Values expressed as mean ± SE.

There were no significant differences in the basophil responses (Figure 16).

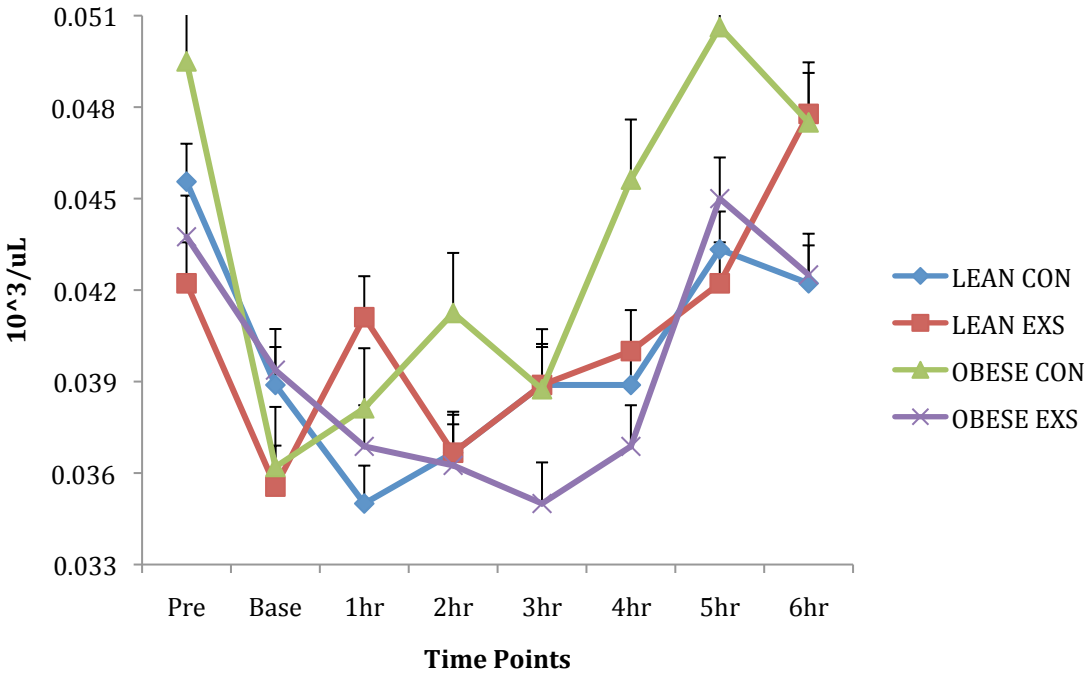

Figure 16. Basophil Count vs. Time Point. Values expressed as mean ± SE.

Chapter V
Discussion

Our study was designed to examine the effects of a single bout of resistance exercise (RE) on the postprandial concentrations of TGs, inflammatory markers and endothelial function in lean and obese young women following a high-fat meal. In order to observe the effects of both the RE and the consumption of a high-fat meal, the variables were tested before exercise (Pre), 16-hours after exercise (Base) and for a 6-hour period after meal consumption. Based on this design, we hypothesized that the RE would attenuate the PPHL response and the inflammatory response, as well as increase endothelial function following the meal. Even though the RE did not significantly blunt the PPHL response, differences seen in artery dilation, vasodilatory markers and inflammatory markers after RE suggest that our exercise manipulation was successful in blunting some of the harmful effects of a high-fat meal. The study design also revealed that the obese participants had greater response to the high-fat meal with or without the presence of the RE, suggesting that the obese population suffered from harmful metabolic effects due to their weight status alone. This is particularly important due to young age of the obese group (22.60 $\pm$ 3.47 years) and the fact that the group was classified on the lower end of the obesity scale, Class I Obesity, with an average BMI of 34.57 $\pm$ 3.30.

Postprandial Lipemic Response

Both groups experienced an elevated lipemic response to the high-fat meal as expected based on previous studies. The degree of the postprandial TG response is slightly less than the majority of previous PPL studies using a similar high-fat test meal (19, 33, 40, 47, 56). Our results show a 6-hour TG AUC in the lean versus obese of 623.07 versus 843.90 mM*hr in the CON condition and 530.20 versus 809.52 mM*hr in the EXS condition, respectively. Zotou et

al. (56) conducted a study using 30-year old recreationally active women found and found a slightly higher TG AUC response to their high-fat test meal, consisting of 60% fat. The resting condition produced a 712 mg/dL*h TG AUC and the resistance exercise condition produced 545 mg/dL*h. The smaller increase in TG elevation noted in the present studied compared to previous ones may be due to our high-fat meal consisting of 50% fat, while previous studies with higher postprandial TG values used meals consisting of between 60 and 67% fat (33, 40, 47, 56). Also, the use of a younger, healthy population may have caused the postprandial TG concentration to be slightly lower than previous studies due to better TG clearance.

The RE did not produce a significant difference in the postprandial lipemic response in either group, despite the small reduction noted in the EXS trials. Many previous studies have found a significant TG lowering effect of aerobic exercise and aerobic exercise training; however, fewer studies have observed a significant effect due to a single bout of resistance exercise. The previous studies that have shown a TG lowering effect due to resistance exercise mainly utilized males over the age of 25 years as participants. Therefore, our results may have differed to the differences in gender TG metabolism or because our participants were young, apparently healthy individuals that may be able to significantly improve their TG metabolism with exercise.

The TG AUC was reduced in the EXS condition by 14.81% in the lean and only 4.07% in the obese. The lack of a significant response to the exercise may be due to the intensity and duration of the resistance exercise or due to a small group size causing an effect on statistical significance. Petitt and colleagues (33) observed a significant 14% reduction in postprandial AUC TG compared to their control condition, after adjusting for baseline differences. This study involved 14 subjects compared to 9 subjects per group in our study. Greater statistical power

due to more subjects may be one reason why Petitt et al (33) showed significant an exercise-induced reduction compared to the insignificant 14% reduction observed in the lean group of our study. Also, the resistance exercise protocol of Petit et al. (33) was similar to our study in all factors except intensity; their subjects performed the exercises at their 10-RM instead of 70% of there 1–RM as in our study.

The TG elevation in both groups suggests that the amount and content of the high-fat test meal was great enough to produce a moderate PPHL response. However, the obese group had a significantly higher lipemic response than the lean, due to a higher concentration of resting and up to 6-hrs of postprandial TG's and a higher postprandial TG AUC. This difference in the lipemic response based on weight alone is particularly interesting due to the young age of the obese participants and their low level of obesity. These young women may not have suffered from obesity for many years, certainly not as long as older obese adults; therefore, the difference in their TG metabolism at such a young age is remarkable. This elevated TG response in the obese group may indicate pre-clinical signs of the metabolic syndrome, ED or atherosclerosis.

Endothelial Response

Initially observing the percent change FMD, only a tendency for an interaction between condition and time was found, although not significant. The data shows a tendency for a decrease in artery diameter change after the high-fat meal and later an increase in diameter change after the 2-hr postprandial time point. This increase continues until the 6-hr time point where it reached near base line measurements. A tendency for the EXS condition to increase diameter change was also seen in both groups; although, this increase was more pronounced in the lean.

The data in both groups and conditions show a percent change in FMD between 11% and 20%, which is approximately one and a half to three times greater than the percent changes observed in previous studies (3, 4, 52). A study of 29 hypertensive patients revealed a baseline FMD percent change of 7.9% and 8.1% in the experimental and control groups (52). Conversely a studied of young, apparently healthy men, with an average age of 27.1 years and BMI of 24.3, found a 7.3% change in FMD at baseline, which was then reduced to 6.0% and 4.3% after two different high-fat meals (3). Another studied comparing 30 patients with peripheral artery disease (PAD) to 30 healthy control subjects all over 60 years of age, found a lower percent change FMD in the PAD patients at 7.3%, compared to the healthy control subjects at 11.4% (4). This indicates that the young women in this study may not be near a state of ED and that the RE intervention may not be able to improve the function of their already healthy arteries. Despite that the obese groups showed other signs of the negative effects of obesity, their endothelial function most likely was not altered at this early stage in life.

We noted that the percent change FMD had some variations in the data, possibly due to errors in finding and obtaining the pre versus peak clips of the artery or errors in the placement of the transducer when taking the video clips of the artery. Individual and daily variations in the participant's artery dilation could be taken into account as well. The variations in the data are evidenced by approximately a 6% difference in percent change FMD between the Pre CON and Pre EXS time points in the lean group. The Pre measurement was a baseline assessment before the exercise and meal intervention; therefore, this time point should remain consistent between the CON and EXS trial within a single group.

When only the Pre and Base time points for percent change FMD were analyzed, however, a significant main effect for RE was found ($p=0.009$). This was due to an increase in

artery diameter change in the EXS conditions for both groups and a small decrease in artery diameter change in the CON conditions after the RE but before the high-fat meal. This indicates that the RE session was successful in increasing artery dilation after exercise, but this beneficial effect was diminished with the presence of the meal. Also, a tendency for an interaction effect for condition and group was seen when analyzing only the Pre and Base time points. The lean EXS condition saw a 1.83% change increase from Pre to Base indicating increased artery function, but the obese EXS condition did not experience as great of an effect with only a 0.87% change increase during this time. The total FMD AUC of the absolute artery diameter was analyzed within each time point. This revealed a significant effect between the lean and obese groups; however, this effect is most likely due to the obese women having an overall larger artery diameter, therefore a greater total FMD AUC. Therefore, the main effect seen between groups may be due to anatomical differences in the participant's systemic circulation due to the differences in body size. However, the AUC FMD analysis also showed that the EXS condition significantly increase artery dilation compared to the CON condition in both groups. This analysis is beneficial because it removes the chance of error for determining the artery pre and peak levels as necessary to determine the percent change FMD.

Nitrite plus nitrate is used as an indicator of NO production that occurs during vasodilatation. Therefore, an increased level of nitrite plus nitrate following exercise indicates a beneficial effect of exercise on endothelial function due to increased vasodilatation. Our results showed a tendency for a beneficial exercise effect at the Base time point (after exercise but before the meal) and up to 2 hours postprandially, but the presence of the high-fat meal eventually diminishes this effect after 2 hours. When the Base time point was analyzed alone, to determine any potential effects of exercise, a significant difference between the CON and EXS

conditions was noted in both groups. These results suggested that the RE produced higher levels of NO, therefore higher levels of nitrite and nitrate, due to increased vasodilatation after the exercise. Despite this beneficial effect eventually being diminished in the later hours of the postprandial state, it did have a tendency to affect the participant's endothelial function in the immediate hours after the meal. In conclusion, the RE did show a beneficial effect on percent change in artery diameter and markers of vasodilatation the day after the exercise session, but these positive vasodilatory effects were diminished in the postprandial hours after a high-fat meal.

Inflammatory Response

IL-6 produced a post meal inflammatory response in both conditions and groups reaching a peak 6 hours after the meal was consumed; however, this meal-induced inflammation was the most severe in the CON condition for the obese participants with a concentration of 8.33 pg/ml. This 6-hr IL-6 concentration was significantly higher than the obese EXS condition with a concentration of 5.41 pg/ml, indicating an exercise related inflammatory reduction. This exercise-related effect on the postprandial IL-6 response was not seen in the lean group; however, there was a significant difference in the CON conditions between the lean and obese groups, suggesting that in a normal resting state, the obese individuals have a higher postprandial IL-6 inflammatory response compared to their lean counterparts. This response mimicked the significant 6-hr postprandial IL-6 increase seen by Harrison and colleagues (14) in their control condition compared to their aerobic exercise condition. Harrison et al. (14) studied young, lean, recreationally active men and their peak IL-6 concentrations of approximately 3.5 and 2.5 pg/mL in the control and exercise conditions, respectively, was much lower than the peak

concentrations of 8.3 and 5.4 pg/mL observed in the obese women in our study. These data suggest an effect of obesity of IL-6 concentration in the postprandial state with and without the presence of exercise. Further investigation of the postprandial inflammatory response was conducted by determining a total IL-6 AUC; however, there were no significant differences between trial condition or group due to the collapsed time points and the late onset of inflammatory in the obese CON condition.

CRP produced interesting results for inflammation in the fact that no differences were seen due to exercise or the high-fat meal ingestion, but there was a significant difference among the lean and obese groups. CRP showing no exercise or meal effects coincides with previous research that suggest that CRP is an inflammatory variable that only changes due to alterations in chronic health conditions, such as impaired endothelial function or metabolic status, obesity or physical inactivity (22, 30). The lean group's CRP concentrations averaged between 700 to 1,000 ng/ml in both conditions across all time points, which is comparable to previous studies of postprandial CRP in a healthy population, before and after exercise training. However, the obese participants in our study showed an average concentration between 3,000 to 5,000 ng/ml, which is three to five times greater than the concentrations seen healthy subjects in other studies.

MacEneaney et al. (22) studied the postprandial inflammatory effects in lean and obese adolescents, and observed CRP levels between 1,000 and 1,500 ng/ml in their obese group including both the control and exercise trials (22). Therefore, the concentration obtained in our obese young women was extremely high, indicating that their obesity has severely affected their chronic inflammatory state, compared to obese adolescents less than a decade younger than them. The Center for Disease Control (CDC) and the American Heart Association (AHA) have stated that high sensitivity-CRP concentrations great than 3000 ng/ml places an individual in the

high risk category for chronic systemic inflammation (9). The CRP concentration obtained by our obese participants is particularly interesting due to the young age of the other wise apparently healthy young women. Additionally, the obese women were classified on the Class 1 Obesity level, suggesting that even minimal levels of obesity could have negative systemic health effects, even at a young age.

Leukocytes and all 5 differential counts showed a significant effect for time due to an overnight diurnal drop from the Pre to Base time points and meal-induced leukocytosis, which was expected based on previous research (14, 22, 24, 36). This diurnal and meal effect on leukocytes caused the morning pre-meal time point (Base) to produce the lowest concentration while the evening pre-exercise time point (Pre) and the 6-hour postprandial time point showed the highest concentration. Exercise-induced leukocytosis was not observed 16 hours after the resistance exercise bout as expected due to prior research (14, 22, 36). However, when the pre-exercise time point was excluded from the analysis, a significant exercise-related reduction in leukocytosis is seen in the lean group at the 3-hour postprandial time point. A significant blunting effect of exercise is also observed between both groups in the neutrophils and esinophils. Neutrophils showed a blunted effect of the resistance exercise at all time points and the esinophils showed a significant reduction at the 3-, 4-, and 5-hour time points. The Harrison et al. (14) studied the inflammatory response to a high fat meal in young men and observed a similar blunting effect on leukocytosis due to prior aerobic exercise, after adjusting for baseline differences.

Conclusion

The purpose of this study was to determine the effects of an acute bout of resistance exercise on postprandial concentrations of TGs, inflammatory markers and on endothelial function in young, lean and obese women. It has been proposed that both acute aerobic and resistance exercise have the ability to blunt the negative effects of high fat meals on various metabolic factors. The present study supports this idea due to a significantly blunted postprandial IL-6 and leukocyte concentration and an increased postprandial artery dilation and vasodilatory marker concentrations in young, apparently healthy women.

Our study differs from many of the previous PPL studies because we used a three-factor design accounting for two weight groups, an EXS and CON condition and an extended two-day testing period, including 6 hours of postprandial measurements. Many of the previous exercise and PPL related studies have used a single population of subjects, typically young to middle-aged men, involving mostly acute or chronic aerobic training with a reduced postprandial testing period. Therefore, we studied a unique population in the less researched area of acute resistance exercise, as well included a wide array of variables. Another strength of our study was the multi-ethic subject pool composed of healthy young women. Participants in each group represented the variety of ethnicities, which included: African-American, Asian, and Caucasian. Another strength of our study is that the two groups of participants, lean and obese, did not vary significantly in age, dietary habits or strength levels. The participants generally consumed the same amount of kilocalories in a day and their diet was composed of roughly the same percentage of nutrients. Therefore, the high-fat test meal should have induced a similar level of response between both groups. The two groups also had a similar TVL in their RE session, contributing to a similar volume and intensity of the exercise session prior to the experimental

trial. The lean and obese groups only differed in variables regarding their body composition, with the obese having a higher weight, BMI, body fat percentage, and waist to hip ratio, as well as a significantly higher systolic blood pressure.

The design of our study could be improved with a larger number of participants per group and by using individuals who are resistance-trained compared to sedentary. Improvements in the study design could also be achieved by standardizing the pre-experimental diet to better control for individual dietary variations. In the present study, within subject variations in pre trial diet was controlled but between subject variations was not controlled. In future studies, the effects on PPL, ED and inflammation due to RE training of 12 or more weeks is needed to understand the chronic effects of RE on these variables. Also, more time points in the later hours of the postprandial period as well as including other inflammatory markers such as TNF-a, IL-1 and coagulation factors could help to better understand the inflammatory responses to exercise and a high-fat meal.

Even though our hypothesis that the postprandial TG concentration would be significantly blunted due to an acute bout of RE was not supported, our hypothesis that endothelial function would improve due to the RE proved to be true. However, the presence of a high-fat meal diminishes the significance of this beneficial exercise effect on artery dilation. Similar exercise-induced effects were observed in the nitrite-nitrate concentrations where the CON conditions produced less artery dilation, evidenced by less NO production compared the EXS conditions; however, this effect was diminished 2-hours after the meal consumption. Our hypothesis that RE would blunt the increase in postprandial inflammatory markers was also proven to be true. IL-6 and leukocytes both showed beneficial effects due to exercise in the postprandial state. This suggests that even acute bouts of exercise can produce beneficial effects

on inflammation in both lean and obese women. In conclusion, our study observed that the negative effects of CVD risk factors including PPHL, increased postprandial inflammation and decreased postprandial endothelial function are seen in apparently healthy young women solely due to their obesity. We also found that an acute bout of RE can blunt the some of the negative effects of reduced artery dilation and NO production in the postprandial state, as well as reduce some postprandial inflammatory markers in both lean and obese young women.

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ABSTRACT

THE EFFECTS OF A SINGLE BOUT OF RESISTANCE EXERCISE ON MEASURES OF POSTPRANDIAL LIPEMIA, INFLAMMATION, AND ENDOTHELIAL FUNCTION FOLLOWING A HIGH FAT MEAL IN LEAN AND OBESE YOUNG WOMEN

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The present study determined the effects of an acute bout of resistance exercise on postprandial concentrations of TGs, inflammatory markers and on endothelial function in young, lean and obese women. Nine sedentary lean (20.22 + 1.20 years) and 10 sedentary obese (22.60 + 3.47 years) women participated in two experimental trials, a resistance exercise trial (EXS) and a resting control trial (CON). The following day they consumed a high-fat test meal after a 12-hour fast, followed by a 6-hour testing period.

The lean group had a lower overall triglyceride response to the meal compared to the obese group ($p=0.028$) and all groups observed a postprandial triglyceride elevation. Both groups experienced an increase in artery dilation after the resistance exercise compared to the control trial ($p=0.034$). Only the obese group observed a significantly blunted IL-6 response due to the resistance exercise ($p=0.019$). The lean group had a significantly lower concentration of CRP compared the obese group across all time points ($p=0.014$). Furthermore, both the lean and obese groups in both conditions observed leukocytosis after the high-fat meal and throughout the test period ($p=0.000$). However, this elevation was blunted in the exercise trials in both groups.

The results of this study suggest that even acute bouts of exercise can produce beneficial effects on inflammation in both lean and obese women. It also suggest that the negative effects of cardiovascular disease risk factors including postprandial hyperlipemia, increased postprandial inflammation and decreased postprandial endothelial function are seen in apparently healthy young women solely due to their obesity.

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CAREER GOALS:

Apply my skills and knowledge as an exercise physiologist to promote life-long health using exercise as rehabilitative tool.

EDUCATIONAL BACKGROUND:

Master of Science in Kinesiology, Concentration: Exercise Physiology, December 2011

Texas Christian University – Fort Worth, Texas
Graduate Assistant
Harris College Outstanding Academic Achievement Award, GPA – 3.51
Thesis – "*Effect of prior resistance exercise on postprandial lipemia, endothelial function and inflammatory markers following a high fat meal in lean and obese young women.*"

Bachelor of Science in Exercise Science, Concentration: Exercise Science, May 2009

Abilene Christian University – Abilene, Texas
Double Minor – Nutrition, Coaching
Summa Cum Laude, GPA - 3.82

RELAVENT EXPERIENCE:

Aug 2011 – Present	Multiple Sclerosis Society of Fort Worth - Adaptive Physical Trainer for Mind Set: Assisted Physical Training
Sept 2009 – Present	Colonial Country Club - Personal Trainer & Service Specialist
June 2011 – Aug 2012	North Richland Hills Recreation Center - Personal Trainer
Feb 2012	Lecturer: Exercise & Aging – Fort Worth YMCA
March 2011	Textbook Reviewer for Lippincott Williams & Wilkins. *Exercise Physiology: Integrating Theory and Application* By William J. Kraemer, Steven J. Fleck, and Michael R. Deschenes
May 2010 – Aug 2010	Cardiac and Pulmonary Rehabilitation Intern Baylor All-Saints Medical Center – Fort Worth
June 2009 – Sept 2009	LA Fitness - Personal Trainer
Fall 2007 – Spring 2009	Abilene Christian University Student Athlete Tutor – Kinesiology
February 2008	USA Track & Field Level 1 Coaching Clinic attendee
Fall 2008	Abilene Christian University Cross-Country Coaching Intern

LABORARTORY PROFICIENCIES:

Exercise Blood Pressure/Heart Rate/Oxygen Saturation monitoring
$V0_2$ Max and Wingate Testing

EKG Interpretation
Body Composition Testing (Skin-fold, Bioelectrical Impedance, Underwater Weighing)
Phlebotomy and Basic Blood Biochemistry

CERTIFICATIONS:

American Heart Association - Advanced Cardiovascular Life Support (ACLS)
American Heart Association - Basic Life Support (BLS) for Healthcare Provider
Cooper Institute - Certified Personal Trainer
SCW – Group Exercise Specialty Certification
SCW – Kettle Weights Specialty Certification

MEMBERSHIPS:

2009 – Present	American College of Sports Medicine (ACSM)
2006 – 2009	TX. Association of Health/Physical Education/Recreation/Dance (TAHPERD)
2006 – 2009	Student Athlete Advisory Committee - ACU Chapter
2005 – 2009	Abilene Christian University Track & Field/Cross Country varsity athlete

HONORS AND AWARDS:

April 2011	TCU's Harris College Outstanding Academic Achievement Award
March 2011	Texas ACSM Master's Student Research Development Award
2010 – 2011	Kinesiology Graduate Assistantship - Texas Christian University
2006 – 2009	Abilene Christian University Dean's List (seven of eight semesters)
2005 – 2009	Abilene Christian University Academic Scholarship
2007	Academic All-American – Track & Field
2006 & 2008	Academic All-Conference – Track & Field

VOLUNTEER:

September 2009	NHL's Dallas Stars Preseason VO_2 Max Testing Assistant
July 2009	Luke's Locker Junior Wellness and Running Camp Counselor
2008 – 2009	Peer Health Educator - ACU Chapter
July 2006	Ultimate Fitness Training - Distance Running Camp Counselor
2004 – 2005	Keller Independent School District Special Olympics Leader