The Influence of Initial Blood Triglyceride Concentrations on the Change in Blood Lipids Following an Acute Bout of Exercise

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This thesis titled
The Influence of Initial Blood Triglyceride Concentrations on the Change in Blood
Lipids Following an Acute Bout of Exercise

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ABSTRACT

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The Influence of Initial Blood Triglyceride Concentrations on the Change in Blood Lipids Following an Acute Bout of Exercise

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The purpose of this investigation was to compare the absolute and relative acute exercise responses in average fitness men with lower (LOW = 43.33 ± 10.31) and higher (HIGH = 151.34 ± 28.90) initial triglycerides (TG) after expending 600kcal on a treadmill. All participants were screened for average fitness (47.37 ± 3.55) and body composition (19.14 ± 3.49), then completed a treadmill jog at 60% of their VO$_2$max and blood draws at 1 (1hPE), 24 (24hPE) and 48 (48hPE) hours postexercise. This investigation demonstrated significant absolute (mg/dl) TG reductions among groups with initially different TG values; however, these differences were relatively equal when considering the percent change. Secondly, groups divided solely on initial TG values had different postexercise HDL-C responses. This investigation adds to the growing body of literature in support of exercise’s beneficial influence over blood lipids and contributes towards a need for further research into the TG-HDL-C relationship.

Approved: _____________________________________________________________

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CHAPTER 1: INTRODUCTION

The National Cholesterol Education Program (NCEP) was founded in 1985 by the National Institutes of Health (NIH), a branch of the U.S. Department of Health and Human Services. In 2001, the third adult treatment program (ATP III) from the NIH was published focusing on the guidelines for treatment of adult blood lipid disorders (NIH, 2001). The majority of the scientific and public literature focuses on the need to reduce low-density lipoprotein cholesterol (LDL-C) or increase high-density lipoprotein-cholesterol (HDL-C) because of the strong association with significant reductions in coronary heart disease (CHD) risk (Manninen et al., 1992). More importantly, there is a significant body of literature indicating the reduction of serum triglycerides (TG) can also produce this positive effect, independent of other blood lipid variables (Guerin et al., 2001).

Each of the adult treatment programs previously published (ATP I, 1985 and ATP II, 1993) focused on specific areas of CHD risk. The ATP I (1985) was implemented and focused on nonspecific, but overall reductions in CHD risk in the at risk population (NIH, 2001), while the focus of ATP II (1993) was on interventions to specifically reduce LDL-C with the limited expectation that it would reduce morbidity and mortality due to CHD (NIH, 2001). What’s more, the NCEP’s ATP II (1993) recommendations considered TG values below 200mg/dl as normal; however, now, backed by a greater body of scientific knowledge and understanding, the most recent guidelines recommend TG be less than 150mg/dl. Therefore, based on these new findings, NCEP’s ATP III (2001) recommends giving more recognition to the possible
role of physical activity in modifying blood lipid profiles (including reducing TG and LDL-C, and increasing HDL-C) than ever before. This is not surprising considering the most influential factor contributing to the development of hypertriglyceridemia is physical inactivity, with obesity being a close second (Whaley et al., 2006).

The link between physical activity and the improvement of the blood lipid profile has been established by previous authors (Durstine, Grandjean, Cox, & Thompson, 2002). Furthermore, according to the NCEP’s ATP III (2001), physical activity and exercise are fundamental components of the “therapeutic lifestyle change,” a semi-drug free therapy that has unlimited potential. Focused meta-analyses demonstrate significant reductions in TG, ranging from 4% to 37%, with absolute reductions as much as 5 to 38mg/dl within 48 hours (h) postexercise (PE; Thompson & Rader, 2001). However, still unclear is the relationship between postexercise TG reductions and the initial TG concentrations.

Statement of Problem and Purpose of the Investigation

According to the current available literature, acute exercise often results in serum TG reductions; however, no work has specifically set out to compare the absolute and relative changes between those with lower (LOW) and higher (HIGH) initial TG concentrations. Thus, the purpose of this investigation was to compare both absolute (mg/dl) and relative (% of initial) blood lipid changes in the 48h following the expenditure of 600 Kilocalories (kcal) on a treadmill in men with lower (LOW) than the median initial TG and higher (HIGH) than the median initial TG concentrations.
Hypotheses

The hypotheses for this investigation included:

1. An acute bout of exercise resulting in the expenditure of 600 kcal will reduce triglyceride in men with average body fat and average aerobic fitness independent of initial levels of triglycerides (both LOW and HIGH TG groups).

2. Absolute reductions (mg/dl) in triglycerides following the expenditure of 600 kcal will be greater in the high triglyceride group as compared to the low triglyceride group.

3. Relative reductions (% change) in triglycerides following the expenditure of 600 kcal will be equal between the groups.

Assumptions

This investigation was limited by the following assumptions:

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

2. Test results depended upon the ability of subjects to maintain a normal physical activity level 72h prior to screening and testing periods and throughout the investigation.

3. Test results depended upon the ability of subjects to abstain from alcohol 72h prior to all blood sampling and to maintain constant dietary habits throughout the investigation.

4. Test results depended upon inherent TG variability between screen and baseline serum TG concentrations taken within a period of 2 weeks.
Delimitations

The parameters of this investigation were delimited by the following factors:

1. The recruited subjects were apparently healthy male volunteers between the ages of 20-34 years and divided into two groups of men: lower than median (LOW) triglycerides ( < 70mg/dl) and higher than the median (HIGH) triglycerides ( > 100mg/dl) for their age group.

2. Subjects were asked to refrain from all physical activity (above normal daily activities), alcohol, and tobacco for 72h prior to testing. Furthermore, subjects were asked to come to each triglyceride screening and testing session following an overnight fast (water only for a period of 12h).

3. Subjects completed a health history questionnaire to determine their relative level of risk according to the standards set forth by the American College of Sports Medicine (ACSM). Only those individuals who were considered low risk, non-smoking and free of any metabolic condition (according to the aforementioned standards) participated in this investigation.

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

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

Operational Definitions

This study considers the following definitions of the terminology:

*Above the median (HIGH).* Men with triglyceride concentrations in serum above the 50th percentile, > 100mg/dl; higher than the median concentration for their age.
**Absolute change (mg/dl Δ).** Measure of actual change (mg/dl) of postexercise (PE) blood lipid variables (1h = 1hPE, 24h = 24hPE and 48h = 48hPE) when compared to baseline.

**Acute exercise.** A single bout of exercise and the body’s response within a 72h period.

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

**Apolipoprotein (apo).** A protein found on the surface of lipoproteins with varying composition and use.

**Average aerobic fitness.** Men with a VO\(_2\)max between 40-52.5ml/kg/min after completing a graded exercise test.

**Average body composition.** Men between 12-25% body fat measured within the BOD POD, a brand name of a body density instrument.

**Below the median (LOW).** Men with triglyceride concentrations in serum below the 25 percentile, < 70mg/dl; lower than the median concentration for their age.

**Blood lipid profile.** The measurement of total triglycerides, total cholesterol and the contribution of high or low density cholesterol.

**Body composition.** Body composition is referred to as the breakdown of body make-up, i.e., fat, lean muscle, bone and water content.

**Chronic exercise.** The accumulated effect of training over several weeks.
Energy expenditure. The amount of Kilocalories (kcal) expended during a given time period.

Graded exercise test (GXT). Continuous, incremental stress test, specifically designed to produce maximal metabolic stress resulting in maximal oxygen consumption.

Hypertriglyceridemia. As defined according to the NIH, NCEP, ATP III (2001), any male above the age of 20 with triglycerides $\geq 150$mg/dl.

Kilocalorie (kcal). The amount of energy needed to raise the temperature of one liter of water by one degree Celsius.

Low-density lipoprotein (LDL). Lipoprotein predominantly composed of triglycerides and containing the apolipoprotein-B (apo B).

Low-density lipoprotein receptor (LDL-R). A receptor protein found predominantly within hepatic tissue and is the main acceptor of LDL.

Lipoprotein receptor protein (LRP). A receptor protein found predominantly within hepatic tissue and is the main acceptor of chylomicron lipoprotein remnants. Additionally, it may be used as a secondary route for LDL absorption.

Maximal heart rate. The maximal rate at which the heart beat reaches during the maximal graded exercise test.

Maximal oxygen consumption. Maximal oxygen consumption (VO$_2$max) is the maximum rate for oxygen consumption by the body during a graded exercise test.

Normal physical activity. Walking/bicycling to and from class, up and down stairs in order to accomplish one’s daily needs.
Percent body fat. The percentage of body mass that is not composed of lean muscle, water, bones or vital organs.

Plasma. The supernatant of blood found after centrifugation within chemical lined (to prevent clotting), pressurized test tube.

Relative change (% Δ). Percent change of postexercise blood lipid variable (1hPE, 24hPE and 48hPE) when compared to baseline as 100%.

Serum. The supernatant of blood found after centrifugation within a clotted, pressurized test tube.

Serum triglycerides (TG). A lipid based molecule composed of three fatty acid chains connected together via a glycerol backbone. All triglyceride measurements were taken from samples of serum and measured in mg/dl.

Whole blood. Blood that has recently been collected and analyzed as is, without separation within a centrifuge. More specifically, the blood collected and immediately analyzed for hemoglobin (Hb) and hematocrit (Hct).
CHAPTER 2: LITERATURE REVIEW

Lipoproteins transport and distribute various combinations of lipid and protein throughout the body, usually in a productive and healthy manner. However, when the circulatory system contains greater concentrations of fat and cholesterol, independent of the specific causative agent, the cardiovascular risk increases exponentially. Chronic exercise has demonstrated significant abilities to keep at least one aspect of the lipid delivery system at bay, triglycerides (TG). Muscles will predominantly metabolize a combination of carbohydrates and fat found within the circulation as well as within the muscle itself. Therefore, the acute exercise investigation allows the researcher to more precisely measure the benefits of a single bout of exercise and the short-term residual effects (within 72h) in order to better explore and prescribe the benefits of chronic exercise habits.

Cardiovascular Disease

Cardiovascular disease (CVD) includes all diseases related to the heart and the blood vessels and is believed to be the leading cause of death (Mitka, 2004). Coronary artery disease (CAD) is the result of atherosclerosis, the progressive build up of plaque within the coronary vessels, leading to the narrowing of the coronary lumen and the clinical result of coronary heart disease (CHD). When delivery of oxygen-rich blood to the coronary arteries is not sufficient to meet the metabolic demands of the heart, ischemic symptoms may appear resulting in injury or necrosis of the myocardium (myocardial infarction).
The Helsinki study of 1987 demonstrated the direct link between the blood lipid profile and the progression of CAD (Manttari et al., 1987). The literature suggests that cholesterol, primarily carried in low density lipoprotein particles may endure oxidative (Henriksen, Mahoney, & Steinberg, 1981) or enzymatic damage and become readily absorbed by interarterial macrophages and thereby, leading to the formation of foam cells and the fatty streaks that initiate atherosclerosis (Brown, Basu, Falck, Ho, & Goldstein, 1980). Thus, evidence suggests that there is a continuous and graded relationship between the concentration of blood cholesterol and the development of CAD.

Furthermore, triglycerides (TG) play a contributing role within lipid transport and have been independently linked to the risk of developing CAD (Leon & Sanchez, 2001). Additionally, a portion of this link can be accounted for by the occurrence of obesity (Howard & Wylie-Rosett, 2002), the result of a sedentary lifestyle.

Blood Lipid Metabolism

The lipoprotein is essential as the predominant transport mechanism of lipid in an aqueous environment, circulation. The lipoprotein distributes its core contents to the body’s extrahepatic cells for productive use and to maintain overall lipid homeostasis. Lipoproteins are lipid carriers composed of an outer surface of hydrophilic phospholipids and proteins surrounding a hydrophobic core of cholesterol, cholesterol esters (CE) and TG (Durstine et al., 2002). Phospholipids surround the core of the lipoprotein permitting lipid solubility within the water-based blood. Embedded within the surface of the lipoproteins are enzymes, transfer proteins, and apolipoproteins categorizing each lipoprotein class and their purported purposes in metabolism (Burnett & Barrett, 2002).
Cholesterol is a lipid-based molecule which can be gained through ingestion of cholesterol-rich foods, but can also be produced within all of the body’s cells. More importantly, the majority of cholesterol synthesis is done within the liver. Triglycerides are the major molecular fat supply primarily stored within adipose tissue, with smaller percentages stored within muscle, intramuscular TG (IMTG) and circulating in lipoproteins (Oscai, Essig, & Palmer, 1990).

In particular, lipoproteins containing derivatives of apolipoprotein-B (apo B) or β-lipoproteins are found to have the largest diameter and the least density (predominantly lipid). Those lipoproteins which contain derivatives of apolipoprotein-A (apo A) or α-lipoproteins are found to have the smallest diameter and greatest densities (predominantly protein). In the most general terms, greater concentrations of β-lipoproteins are associated with increased CHD risk based on their increased susceptibility to penetrate arterial walls and contribute to the production of fatty streaks. In contrast, the α-lipoproteins are associated with CHD risk reduction based on their role in reverse cholesterol transport; however, there is more to this story.

Each lipoprotein is categorized into classes by a gradient of size and density, from largest and least dense to smallest and most dense. The chylomicron is the largest and least dense, followed by the very low-density lipoprotein (VLDL), intermediate low-density lipoprotein (IDL), low-density lipoprotein (LDL), and lastly, the smallest and most dense particle is the apolipoprotein-A (apo A) associated, high-density lipoprotein (HDL) (see Burnett & Barrett, 2002 for further information). Additionally, within each lipoprotein class (Chylomicron, VLDL, IDL, LDL, and HDL) there is an even more
specific gradient of size and density. For example, LDL particles are thought to be heterogeneous, whereby the smallest, densest LDL particles are correlated to the greatest risk for vascular penetration which may lead to accelerated atherosclerosis (Mudd et al., 2007). On the other hand, larger, more buoyant HDL particles may be more effective within reverse cholesterol transport (Lamarche et al., 1995). Thus, each lipoprotein’s actions and relationships with CHD are dependent upon its size, density and composition. Furthermore, two pathways exist with the same general goal of hydrolyzing the predominantly lipid filled lipoprotein into a ‚leaner‘ version of itself, thereby distributing its contents for productive use. However, the exogenous and endogenous lipid transport pathways differ in their mechanism of action, location of release as well as the receptor mediated uptake out of circulation.

**Exogenous Lipid Transport**

The exogenous pathway is essential to the digestion and transport of all dietary fat through the intestinal lumen into the mucosal cells and eventually into circulation via the lymphatic system (van der Vusse, 2009). The majority of digestion and emulsification occurs within the small intestine (duodenum and ilium); here the presence of pancreatic lipases and bile salts combine with dietary fat and cholesterol to form lipid droplets surrounded by phospholipids, called micelles (Moffat & Stamford, 2006). These lipid droplets, composed of TG, phospholipids (PL) and cholesterol are then hydrolyzed into diffusible forms of fatty acids (FA) phospholipids (PL) and cholesterol and are absorbed within the mucosal cells of the intestine and then packaged back together, or re-esterified, successfully storing the lipid structure. The intestinal derived lipid components (TG, PL
and cholesterol) are packaged with apolipoprotein (apo) B-48, apo A-I and apo A-IV and released into circulation where it incorporates apo C-I, C-II, C-III and apo E via transfer from other circulating lipoproteins (Moffat & Stamford, 2006), thus producing the largest of all the lipoproteins, the chylomicron, containing approximately 85% TG (see Burnett & Barrett, 2002 for further information).

During chylomicron circulation, lipoprotein lipase (LPL), found on the surface of endothelial capillaries (Mead, Irvine, & Ramji, 2002), interacts with apo C-II, a cofactor which is embedded within the surface of the chylomicron in order to hydrolyze its TG-rich core into transferable free FA. Thus, these newly freed FA are available for neighboring tissues to absorb via membrane-associated fatty acid binding proteins (FABP) found on the surface of absorptive cells (Hargreaves & Spriet, 2006). Newly freed fatty acids are then absorbed through the phospholipid bilayer into the vesicle and re-esterified back into the original TG structure for storage within adipose tissue, storage within myocytes as intramuscular triglycerides (IMTG) or if needed, preferentially shuttled to the mitochondria for β-oxidation within working muscle (Hargreaves & Spriet, 2006). Hydrolysis of the chylomicron continues in circulation via LPL and to a lesser extent, hepatic lipase (HL) (Hargreaves & Spriet, 2006), until the originally TG-filled chylomicron is suitable for receptor-mediated catabolism by the liver via apo E specific receptors, LDL-receptor (LDL-R) or LDL receptor-related protein (LPR) as a lean chylomicron remnant consisting of cholesterol, phospholipids and much less TG (Burnett & Barrett, 2002). Additionally, the unabsorbed FFA binds with albumin and continues to circulate (van der Vusse, 2009) until the complex is either absorbed by other
tissues, via the same processes, or eventually absorbed into the liver to be repackaged and released as a very low density lipoprotein (VLDL) within endogenous lipid transport.

*Endogenous Lipid Transport*

As stated earlier, the major goal of both the endogenous and exogenous lipid transport systems is to supply lipid-based molecules to the body’s tissues for membrane structure, hormone production and/or bile acid secretion. However, differing are the sites of receptor-mediated uptake into and out of circulation. While the major lipid resource for exogenous distribution is from the diet and packaged and released from the small intestine, endogenous lipids are made up from circulating “leftover” lipoprotein remnants and albumin-bound FA, which are all absorbed, repackaged and distributed via the liver in the form of a very low-density lipoprotein (VLDL), the equivalence of the chylomicron in exogenous lipid transport. The VLDL is packaged with approximately 50% TG, 20% cholesterol and 8% is made up of apolipoproteins (apo) B-100, C-I, C-II, C-III, and E (see Burnett & Barrett, 2002 for further information). Similarly to the chylomicron, lipoprotein lipase (LPL) and hepatic lipase (HL) contribute to the hydrolysis of TG via the apo C-II; however, not all apolipoproteins are found embedded within the core of both lipoproteins. The VLDL contains apo B-100 and is packaged and released from the liver and immediately active in circulation. Alternatively, the chylomicron contains the apo B-48 and is packaged and released from the small intestine.

Lipoprotein lipase (LPL) continuously hydrolyzes the VLDL’s composition, reducing its size and increasing its density as the percentage of TG contained within its core is reduced, thereby creating the intermediate-low density lipoprotein (IDL). Further
delipidation of the IDL results in the gradual removal of the majority of TG from its core and the apolipoprotein from its surface, thereby forming the low density lipoprotein (LDL) composed of approximately 50% cholesterol, 6% TG, the remaining (44%) is split up evenly between phospholipids (PL) and the one major apolipoprotein, apo B-100 (see Burnett & Barrett, 2002 for further information). Finally, the LDL particle can be taken out of circulation via the LDL receptor (LDL-R) recognition of apo B-100.

The complex interactions of TG, FA, apolipoproteins, PL and cholesterol during exogenous or endogenous periods results in the replenishment of cellular cholesterol for important hormonal and digestive functions. However because of such complexities, overproduction in one area of the lipid cycle may only further atherosclerotic plaque development. In order to prevent atherosclerosis, cholesterol is collected by HDL and returned to the liver in a process called reverse cholesterol transport (RCT).

Reverse Cholesterol Transport

It is well known amongst the general healthcare industry that high density lipoproteins (HDLs) or the “good cholesterol” play a role in reducing CHD risk by collecting potentially hazardous cholesterol from circulation and returning it to the liver for productive use. According to Yancey et al. (2003), the reverse cholesterol transport (RCT) process occurs in three basic steps; a) efflux of free cholesterol (FC) from extra-hepatic cells on to the HDL surface, b) cholesterol esterification and transfer into the core of the HDL via lecithin cholesterol acyl transferase (LCAT), and c) the removal of HDL from circulation directly (75%) via scavenger receptor B-I (SR-B1) or indirectly (25%) through cholesterol-ester transfer protein (CETP) intravascular interactions with apo-B
associated lipoproteins and the removal via the hepatic derived LDL-receptor (LDL-R) recognition of apo B-100 (Yancey et al., 2003).

In its juvenile state, the pre-β-HDL consists of a lipid-poor protein band consisting of the HDL’s major apolipoprotein apo A-I. This lipid-poor protein will collect phospholipid (PL), free cholesterol (FC), and some TG to eventually produce the α-HDL, or commonly known as the HDL (Remaley et al., 2001). The primary fuel for HDL growth is the efflux of free cholesterol (FC) from extrahepatic cells into the core of the HDL, resulting in a change in size and shape. Thus, research has defined the α-HDL particles into subfractions based on the size and density, small and dense to large and buoyant HDL particles; HDL₃, HDL₂ and HDL₁, respectively (von Eckardstein, Huang, & Assmann, 1994). Based on these particle subfractions, comprehensive HDL-C screening techniques have been developed; however, this is only important for the mechanistic understanding of RCT and, in general, the total amount of cholesterol found within the HDL (HDL-C) has a strong inverse relationship with CVD risk.

Free cholesterol (FC) efflux can occur via two pathways, aqueous diffusion or protein-mediated diffusion (Yancey et al., 2003). Basic aqueous diffusion of free cholesterol from extrahepatic cells to the HDL surface is passively driven by the cholesterol concentration gradient and thus it is bidirectional and extremely inefficient (Rothblat et al., 1999). However, protein-mediated FC efflux augment the kinetics of simple diffusion via two receptors, ATP-binding cassette transporter A1 (ABCA1) and scavenger receptor class B type 1 (SR-B1). The majority of research into ABCA1 suggests unidirectional cholesterol efflux directly contributing to apo A-I lipidation,
thereby creating a more stable pre-β-HDL particle and therefore, indirectly contributing to RCT (Yancey et al., 2003).

In order for free cholesterol (FC) to be stored within the hydrophobic core of the lipoprotein, it must complete the process of esterification, or the simple addition of an ester group to the cholesterol compound (Moffat & Stamford, 2006). This process is accelerated by another enzyme, lecithin cholesterol acyl transferase (LCAT) producing a cholesterol ester (CE) compound for storage within the core of the lipoprotein (Jonas, 2000) and allowing more surface absorption of FC, resulting in HDL growth. Therefore, the FC efflux and the enzymatic work of LCAT perpetuate HDL growth and the recycling of potentially hazardous cholesterol back to the liver. Next, evidence suggests the liver-associated, scavenger receptor class B type 1 (SR-B1) accelerates the slow free cholesterol (FC) efflux from the HDL via selective uptake of its “on board” cholesterol, allowing the HDL particle to continue circulating (Acton et al., 1996). Therefore, the SR-B1 effectively prolongs the HDL’s time within circulation and thus, increases the chances of contributing to the indirect RCT pathway, via intravascular collisions with β-lipoproteins.

The indirect RCT theory suggests intravascular collisions between cholesterol ester (CE) filled HDL and TG-filled β-lipoproteins (Chylomicron, VLDL, IDL and LDL) activate the lipid transfer protein, cholesterol ester transfer protein (CETP) (Barter et al., 2003). Simply put, the CETP transfers TG to the HDL, in exchange for sending CE to the β-lipoprotein. This lipid exchange has both positive and negative results (Boekholdt et al., 2004); however, research suggests the most beneficial result of this lipid transfer of
TG to the HDL is the continual flux between HDL$_3$ (smaller and denser) and HDL$_2$ (larger and less dense) effectively lengthening the HDL circulation time and further contributing to RCT (Barter et al., 2003). Secondly, the CETP exchange allows the CE portion, now found within the β-lipoprotein, to essentially “hitch-hike” back to the liver via the β-lipoprotein liver-specific receptor, LDL-R (Boekholdt et al., 2004).

These basic areas of human blood lipid metabolism have been, and will continue to be, major points of interest for their role in the development of CHD and the research towards possible therapeutic disease prevention. These complex systems must work synergistically to prevent arterial plaque buildup; however, if one area works improperly, whether that be because of genetic abnormalities and/or lifestyle adaptations, it can have a drastic influence on one’s health risks.

Lipids, Lipoproteins and Health

According to the NIH in their latest NCEP’s ATP III (2001), blood lipid profile screening should be completed every 5 years in all adults at or over the age of 20 and should include total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C) and serum TG. The next section will outline these risks and provide evidence based hypotheses on how elevated TG concentrations can impact overall CVD risk.

The Blood Lipid Profile

A total cholesterol value of below 200mg/dl is considered desirable; however, both quantitative and qualitative (i.e., percentage of HLD-C to LDL-C) aspects must be considered when stratifying for CHD risk. The most recent NCEP ATP III
recommendations (2001) provide further evidence of the need to reduce LDL-C as the primary goal for treatment to reduce CHD risk. Specifically, the primary treatment for the reduction of LDL-C is via “therapeutic lifestyle changes” (TLCs) including dietary intervention, increased physical activity, and weight reduction or via a combination of TLCs and pharmaceutical intervention(s). However, based on covariate adjustments (TG, HDL-C), high variability among measurements (TG, apo B associated lipoproteins) and the cost of analyses (lipoprotein remnants, small dense LDL), many secondary risk factors are statistically dependent upon other lipid (LDL-C) and nonlipid (age, obesity, sex, etc.) characteristics in their CHD predictability (NIH, 2001). Therefore, the dispute over a risk factor’s independent or dependent categorization will continue to plague public health officials (Assmann, Schulte, Funke, & von Eckardstein, 1998; Hulley, Rosenman, Bawol, & Brand, 1980). Additionally, the emergence of the combined three-part dyslipidemia called, the “lipid triad” has shown a strong base of support. According to the latest ATP III, (2001) and its supporters (Austin, Hokanson, & Edwards, 1998; Krauss, 1998) the “lipid triad” consists of elevated TG, low HDL-C and elevated concentrations of small dense LDL, primarily based on their association with atherogenic, cholesterol-rich lipoprotein remnants, TG-rich lipoproteins (TRL) and their combined strength in predicting CHD risk and other chronic diseases, such as diabetes mellitus, obesity and metabolic syndrome.

Amidst the categorization of elevated TG as a secondary CHD risk factor by NCEP’s ATP III (2001) recommendations, there is still a strong base of literature providing evidence in support of TG’s independent predictive ability for future CHD
(Hokanson & Austin, 1996; Jacobson, Miller, & Schaefer, 2007). Meta-analyses have concluded the presence of elevated TG signifies an independent risk factor for CHD (Assmann et al., 1998). Jacobson et al. (2007) suggest TG testing should be the clinical norm, especially considering the increased prevalence of physical inactivity and its association with other chronic diseases (Alexander et al., 2003). Furthermore, those who do consider TG as a secondary indicator (Hulley et al., 1980), still support the strong association of the “lipid triad” and the inverse relationship between TG and HDL-C (Manninen et al., 1992).

The strongest base of evidence to consider low HDL-C as an independent and primary risk factor is the morbidity/mortality rates of the Helsinki Heart study (Henriksen et al., 1981; Manninen et al., 1992). In particular, Gordon et al. (1989) suggested that for every one mg/dl increase in HDL-C there is a 2-3% reduction in CHD risk (Gordon et al., 1989). Further support for the predictive ability of HDL-C was provided by Castelli (1986), who’s view of the Framingham investigation suggested the CHD predictability of TG was only as strong as the participants HDL-C; more specifically, only those with HDL-C <40mg/dl were at an increased risk, while those with normal or above average HDL-C were at no greater risk (Castelli, 1986). However, much like TG within NCEP’s ATP III (2001), HDL-C is also considered secondary based on its relationship with covariates of both lipid and nonlipid factors. Thus, among the blood lipid profile, LDL-C has been highlighted the most for its predictive ability of future CHD risk. However, the emergence of the “lipid triad” and its relationship with the proliferation of atherogenic
lipoprotein remnants, suggest these “secondary” factors should be given more respect for their predictive ability of CHD and the possibilities of future therapeutic interventions.

*Triglyceride-Rich Lipoproteins*

Regardless of primary or secondary rankings, hypertriglyceridemia strongly contributes to an elevated CHD risk by increasing the concentration of TG-rich lipoproteins (TRL) and their remnants within circulation (Hodis, 1999). The theory behind the atherogenicity of TRL and their remnants suggests that an elevated concentration of blood TG creates larger TRL. These large TRL contain a larger than normal amount of lipid to distribute and, thus, one hypothesis suggests, the extended lifecycle of the TRL in combination with the actions of CETP, provides the right circumstances to produce elevated concentrations of the most atherogenic particle, the small-dense LDL (Boekholdt et al., 2004). The result is a “pattern B” blood profile, the elevation of blood TG, lower HDL-C and greater concentrations of small dense LDL, first recognized by Austin et al. (1990) and now coined by the NCEP ATP III (2001) as the “lipid triad.”

At any one time-point, unless individuals are in a fasted state (12h of fasting), both the exogenous (chylomicron) and endogenous (VLDL, IDL and LDL) lipid delivery systems are simultaneously interacting among one another; however, according to Bjorkegren et al. (1996), these processes do not go without their problems. Bjorkegren et al. (1996) observed an ineffective VLDL hydrolysis during competition for the endothelial-bound LPL between TG-rich chylomicron and VLDL particles. Therefore, competition among the TRLs for endothelial bound-LPL suggests the TG-rich core of the
VLDL must “wait in line” for the larger and more effective chylomicron-LPL hydrolysis reaction, effectively increasing the VLDL time within circulation and potentially increasing negative CETP lipid interaction.

Secondly, the accumulation of lipids, in the form of TG and/or free fatty acids (FFA) has been demonstrated to influence the hepatic production (Lewis, 1997) and composition (Rader, Castro, Zech, Fruchart, & Brewer, 1991) of the VLDL particle thus resulting in the modification of its normal properties and functions. Furthermore, a constant surface thickness among all VLDL subfractions (VLDL_{1} – VLDL_{3}; largest to smallest, respectively), suggests the differences in size between VLDL_{1} and VLDL_{3} can only be accounted for by the variable amounts of its core contents, primarily TG (Sata, Havel, & Jones, 1972). Therefore, the combination of a less effective VLDL-LPL hydrolysis, larger TG-filled VLDL_{1} particles and potent CETP interactions provide significant evidence to consider the VLDL particle as a point of significant interest in the appearance of small dense LDL particles.

Cholesterol ester transfer protein’s (CETP) actions are predominantly beneficial when normal concentrations of blood TG are present; however, within a system of elevated blood TG, the action of CETP has been demonstrated to reduce RCT efficiency (Lagrost et al., 1995). As described earlier, CETP’s actions are intended to be a secondary route of reverse cholesterol transport (25%), transporting CE onto the \( \beta \)-lipoprotein and eventually back to the liver via hepatic absorption (LDL-R). The action of the HDL-bound CETP works toward a lipid-equilibrium, transferring CE for TG with other lipoproteins (Barter, Hopkins, & Calvert, 1982). Additionally, the triglyceride-rich
lipoprotein (TRL) transfers a portion of its TG to the HDL and under normal conditions this would contribute positively towards RCT. However, an elevated amount of substrate (TG) extends the delipidation processes effectively depleting the CE within the HDL core and resulting in smaller and less effective HDL (Newnham & Barter, 1990).

More importantly, this extended CETP interaction also affects the large TG-rich VLDL₁. Specifically, the extended CETP processes cause an accumulation of CE within the normally TG filled VLDL (Barter et al., 2003), creating the TRL-remnant. This is important when keeping in mind, the VLDL is primarily built to distribute TG, not cholesterol. Therefore, after an extended CETP exchange (TG for CE) resulting in an overabundance of TG within its core, the now CE-rich particle may not follow its normal VLDL–IDL–LDL lifecycle and instead, create the CE-rich VLDL remnant and eventually, the highly atherogenic small, dense LDL particle (Chung, Segrest, & Franklin, 1998).

Lastly, and least likely to occur, is the CETP-activity between two β-lipoproteins (Bjorkegren et al., 1996). This worst case scenario is believed to produce highly atherogenic remnant particles and further contribute to the reduction of LDL particle size (Guerin et al., 2001). More specifically, interaction between two β-lipoproteins can dramatically shift the lipid substrates of CETP and hepatic lipase (HL) to produce an increased appearance of small, dense LDL. Additionally, other authors have supported the relationship between elevated blood TG and elevated small, dense LDL particles (McNamara, Jenner, Li, Wilson, & Schaefer, 1992). However, according to Packard and Shepard’s review (1997), only when TG concentrations are > 1.5mmol/L (> 133.5mg/dl)
or > 1.6mmol/L (> 142.4mg/dl) do the small, dense LDL subfraction particles accumulate in men. More recently, this theory was supported by Boekholdt et al. (2004) in the EPIC (European Perspective Investigation into Cancer and Nutrition) Norfolk-Population Study. Boekholdt et al. (2004) demonstrated that elevated CETP levels (the most likely culprit contributing to an increased small dense LDL appearance) were associated with an increased risk of future CAD in apparently healthy individuals, but only in those with plasma TG concentrations above 1.7mmol/L (> 151.3mg/dl).

Triglyceride-rich lipoproteins and their remnants are prevalent within hypertriglyceridemic condition and can cause a dramatic shift in LDL particle size, decrease the efficiency of RCT via small ineffective HDL and thus, increase the potential atherosclerotic deposition and further CHD development (Jacobson et al., 2007). Furthermore, the majority of research suggests CETP has an inhibitory effect upon RCT, based on the evidence of elevated HDL-C in CETP deficient subjects (Inazu et al., 1994) and in mice, after in vivo CETP inhibition (Kee et al., 2006). However, under normal blood lipid circumstances, basic research has also demonstrated CETP to be the major indirect pathway of cholesterol ester disposal via either of the β-lipoprotein hepatic lipid receptors (LDL-R; Schwartz, VandenBroek, & Cooper, 2004).

The exploration and inhibition of CETP’s actions has been suggested to be a major pharmaceutical development in order curb the development of atherogenic particles and reduce CHD. More importantly, the negative impact of CETP and the production of TRL are highly dependent upon the concentrations of blood TG. Therefore, a much simpler and less expensive method of CHD reduction may be to
prevent the accumulation of blood-TG in the first place, via a simple bout of exercise in order to mobilize fat stores and keep blood TG concentrations within normal ranges.

Lipid Mobilization and Oxidation

Lipids are an important fuel source and generally contain three times the energy as carbohydrates. Energy provided by these fuels are broken down for quantification by the calorie, the amount of energy needed to increase one liter of water by one degree Celsius. In order to quantify these large numbers more precisely, exercise physiologists have adapted several conversions including the joule and the Kilo-joule, however for the remainder of this paper energy expenditure will be defined as Kilocalories (kcal = 1000 calories). Both the anaerobic and aerobic oxidative systems contribute to Adenosine-triphosphate (ATP) production, with a predominance of aerobic energy production via oxidative phosphorylation and the electron transport chain. The catecholamine release associated with exercise stimulates a combination of lipases increasing FA availability within circulation and ultimately, resulting in the mitochondrial-oxidation of FAs within skeletal muscle, however, much like the lipid delivery system, there is more to this story.

Bock et al.’s 1928 investigation into the individual contributions of fat and carbohydrate via indirect calorimetry, formulating the respiratory quotient (RQ) or RER, still hold true today. Havel et al. (1964) estimated about half of the expended energy during 2h of cycling at a moderate intensity (HR~130bpm) is accounted for by fat oxidation. Further evidence provided since Dill et al.’s 1930 work (Hargreaves & Spriet, 2006) suggests fuel utilization during exercise is predominantly lipid at low (< 50%) to
moderate (50-70%) intensities, contrastingly at higher intensities (> 75%), carbohydrate is more readily utilized.

This phenomenon has been supported by Bulow et al. (1981), who demonstrated an exercise-induced catecholamine release can induce a two-fold increase in adipose tissue blood flow and thus, increasing FFA availability for greater skeletal muscle FFA uptake. While during higher intensity exercise, the rise in catecholamine levels initiates an inhibitory α-adrenergic-effect to reduce adipose tissue hydrolysis as well as restrict adipose tissue blood flow via vasoconstriction (Hodgetts, Coppack, Frayn, & Hockaday, 1991). However, because indirect calorimetry measurements do not distinguish between sites of fat storage, specific contributions of different fat storage sites for skeletal muscle fat oxidation has been a methodologically controversial subject. Fatty acids are provided for ATP production via three currently measureable fat oxidation sources: a) total body fat oxidation (via indirect calorimetry or RER); b) circulating plasma FFA and the FAs contained with VLDL-TG (via blood collection and isotope tracers); and, c) the controversial contribution of intra-muscular TG (IMTG; via muscle biopsy, isotope tracer, quantitative ultrasound, or Hydrogen-Magnetic Resonance Spectroscopy; $^1$H-RMS). To date, the complete quantification of each of these lipid contributions at varying exercise intensities and their mechanisms are still under investigation. In Horowitz’s latest review (2003), he suggested subcutaneous adipose tissue (upper and lower body) provides nearly half of the total fat oxidation estimated via indirect calorimetry. Thus, the majority of fat oxidation can be attributed to the hydrolysis of
adipose tissue-TG stores versus the minor contributions from IMTG and VLDL associated TG.

*Adipose Tissue*

Until recently, evidence suggested in order for adipose tissue-stored TG to be used as a fuel source during exercise, the associated catecholamine release initiates the hormone sensitive lipase (HSL) cascade in order to hydrolyze its pool of intercellular TG. More specifically, exercise releases catecholamines which in turn, activates α and β-adrenergic receptors, resulting in the inhibitory or stimulatory effects, respectively (Hargreaves & Spriet, 2006). These adrenergic receptors initiate G-protein receptor interaction, starting the adenylate cyclase cascade, which converts ATP to cyclic adenosine-monophosphate (cAMP), activating its cAMP-dependent protein kinase in order to start the HSL phosphorylation cascade of the perilipins surrounding the adipose tissue-TG pool and thus freeing FAs for skeletal muscle use (Sztalryd et al., 2003).

Recent investigations using HSL-deficient mice demonstrated another contributor to TG hydrolysis, later named by Zimmermann et al. (2004), “adipose triglyceride lipase” (ATGL). Zechner et al. (2009) suggested that ATGL, in coordination with HSL, initiates the hydrolysis of the first of the three FAs from the glycerol backbone, creating the diaclyglycerol (DG). The DG is then hydrolyzed preferentially by HSL to the monoglycerol (MG). This hydrolyzing process is continued via monoglycerol lipase (MG) in order to free the last FAs from the glycerol backbone. Zimmerman et al. (2009) proposed a combined catecholamine, HSL and ATGL cascade in order to hydrolyze adipose tissue-TG; however, further evidence is needed to elucidate this phosphoylation
cascade theory. The hydrolysis of adipose tissue is the major contributor toward plasma FFA and thus, provides the majority of FFA for skeletal muscle absorption. However, there are other plasma FFA contributors to take into account, specifically, albumin-bound FA and lipoprotein-bound FAs (VLDL-TG).

Circulating Fatty Acids

There are three major contributing pathways to the use of circulating FFA, a) immediate absorption at the source (adipose tissue) of FA release via neighboring skeletal muscle fibers, b) circulating FFA bound to albumin, and c) FFA derived from LPL enzymatic actions on lipoproteins containing TG. The albumin bound FAs continue in circulation and are shuttled toward muscle fibers via increased skeletal muscle blood flow; however, if the albumin-FA complex is able to reach the liver, the FFA is repackaged into TG-form and released into circulation within the VLDL and the cycle continues.

Presumably, exercise completed in the fasted state relies on the liver to package and release VLDL-TG in order to support ATP production via fat oxidation. This is not just a presumption, because Borsheim et al. (1999) demonstrated an exercise induced reduction in the concentration of VLDL as well as the percentage of TG found within the VLDL as little as 4.5h postexercise (PE). According to Horowitz and Klein’s review (2000), evidence has shown negligible (Kiens & Lithell, 1989) to 5-10% (Ryan & Schwartz, 1965) contributions of plasma VLDL-TG toward total body fat oxidation. However, more recent investigations found plasma and VLDL-TG associated FA
contributions to be indistinguishable (Helge, Watt, Richter, Rennie, & Kiens, 2001; Teusink et al., 2003).

According to Morio et al. (2004) the liver contributes to greater VLDL-TG turnover during the early recovery phase (< 2hPE); however, they conceded the fact that while the VLDL-TG turnover demonstrated significant increases postexercise, it only contributed 13% to the total body fat oxidation. More interestingly, the novel glycerol tracing method used by Morio et al. (2004), demonstrated elevated VLDL-TG turnover during the first hour of recovery from a 45min bout of moderate intensity exercise in sedentary, nonobese subjects; however, there was no overall net change in VLDL-TG concentration. One explanation for the increased VLDL-G turnover without a reduction in concentration (Kimber, Heigenhauser, Spriet, & Dyck, 2003) is that the increased VLDL-TG turnover during the first hour of the recovery phase provides FA fuel for the replenishment of intramuscular TG (IMTG).

*Intramuscular Triglycerides*

Efforts to distinguish IMTG fat oxidation contributions have resulted in variable results between and within many investigations (Kimber et al., 2003; Watt, Heigenhauser, Dyck, & Spriet, 2002) as well as varying opinions within several reviews (Horowitz & Klein, 2000; Horowitz, 2003; van Loon et al., 2003; Watt et al., 2002). The primary reason for such discrepancies include, but are not limited to, the IMTG measurement technique, characteristics of the subjects and variability in the exercise treatment (Horowitz, 2003; van Loon et al., 2003; Watt et al., 2002). Even with the recent advances in IMTG measurement techniques, such as $^1$hydrogen-magnetic
resonance spectroscopy (\(^1\text{H-RMS}\)) and novel lipid tracers, a standard and accurate measurement of IMTG utilization is still needed. (For a more detailed review over IMTG methodology, see Horowitz, 2003 and van Loon et al., 2003).

The majority of investigations suggest that the IMTG pool can provide a dynamic fuel source during exercise (van Loon et al., 2003). Significant IMTG contributions toward total fat oxidation range from as little as 10% and as much as 50% during exercise (Horowitz, 2003). However, other authors have concluded insignificant IMTG contributions both during (Watt, Heigenhauser, & Spriet, 2002) and after exercise (Kimber et al., 2003). Watt et al. (2002) suggested that the primary stumbling point in IMTG measurement is centered on two areas: differences in IMTG measurement techniques and, the training status of willing participants. Additionally, it is a well known theory that endurance training improves the body’s ability to rely on fat oxidation in order to spare glycogen (Holloszy & Coyle, 1984). However, this increased fat oxidation does not equate to an increased rate of lipolysis; in other words, training has no effect on FA mobilization (Martin et al., 1993). Thereby, this provides a possible role for IMTG, because increases in fat oxidation due to training cannot be explained by the oxidation of circulating FA, but instead, maybe a more local intramuscular source.

Research comparing the contracting and the noncontracting muscle via one-leg exercise demonstrated not only a reduction of IMTG within the exercising leg (~30%), but also provided further insight into the mechanism of IMTG utilization and storage (Sacchetti, Saltin, Osada, & van Hall, 2002). Sacchetti et al. (2002) suggested that their IMTG estimations demonstrate skeletal muscle uptake of circulating FFA for IMTG
storage in resting muscle fibers and the preferential shuttling of IMTG toward β-
oxidation in contracting muscle fibers. In support, other investigators (Dyck & Bonen,
1998; Morio, Holmback, Gore, & Wolfe, 2004) postulated that the IMTG controversy
can be explained by the simultaneous re-esterification and incorporation of FFA into the
IMTG-pool simply by the stimulatory effect of muscle contraction, thus maintaining a
relatively consistent IMTG concentration. Furthermore, recent evidence of muscle
associated HSL allows for the possibility of TG and FA shuttle toward β-oxidation within
muscle (Roepstorff et al., 2004). Therefore, research has provided evidence towards the
storage of TG within muscle and its preferential use and immediate replenishment via
circulating FFA, independent of where or how the FFA was mobilized (Teusink et al.,
2003), for a more concise review into IMTG utilization see the reviews by van Loon

Independent of the source, all free fatty acids (FFA) chosen for myocyte
absorption by membrane-specific fatty acid binding proteins (FABP) are guided through
the cytoplasm via transporters such as fatty acid transporter CD-36 (FAT-CD36) (Bonen,
Luiken, Arumugam, Glatz, & Tandon, 2000) toward the outer mitochondria membrane,
and guided across by creating a carnitine compound. Once inside the mitochondria, the
carnitine compound is replaced with CoA, creating the fatty acyl-CoA molecule for β-
Oxidation. This pathway includes the production of reducing equivalents NADH and
FADH$_2$ and acetyl-CoA. This acetyl-CoA is then used within the citric acid cycle to
produce more NADH and FADH$_2$ reducing equivalents for use within the electron
transport chain and the eventual production of usable ATP, via oxidative phosphorylation (Hargreaves & Spriet, 2006).

The Effects of Exercise Training on Blood Lipid Variables

Cross-sectional data (Halle, Berg, Konig, Keul, & Baumstark, 1997; Sgouraki, Tsopanakis, & Tsopanakis, 2001; Ziogas, Thomas, & Harris, 1997), meta-analyses (Halbert, Silagy, Finucane, Withers, & Hamdorf, 1999; Kodama et al., 2007; Kelley, 2004b), and growing experimental evidence support the notion that chronic exercise improves the blood lipid profile (Crouse et al., 1997; Katzmarzyk et al., 2001; Kraus et al., 2002; Slentz et al., 2007; Wilund, Ferrell, Phares, Goldberg, & Hagberg, 2002); however, not all investigations have supported the beneficial influence of exercise (Gaesser & Rich, 1984; Imamura et al., 2000). Halbert et al. (1999) cautioned the interpretation of their meta-analysis supporting the impact of exercise to improve blood lipid variables, stemming mainly from the extreme heterogeneity among the exercise training investigations. However, more recent meta-analyses by Kelley et al. (2004b) and Kodama et al. (2007) limited their analysis to only those exercise investigations that included a control group (Kelley et al., 2004b; Kodama et al., 2007). Moreover, they concluded exercise training has a beneficial effect on blood lipid variables, specifically, a modest reduction in LDL-C after walking of at least 8 weeks (Kelley et al., 2004b) and on average, a rise in HDL-C (2.53mg/dl) after an energy expenditure of 900kcal per week or 120min per week (Kodama et al., 2007). Authors suggest that these contradictory results on the major blood lipid variables (TChol, LDL-C and its subfractions, HDL-C and TG) are based on many varying factors, including exercise training variability.
(intensity, duration and volume) and training effects (weight, body composition and/or aerobic fitness). This section overviews the major findings of the chronic exercise effect on each of the major blood lipid variables (TChol, LDL-C, HDL-C and TG).

**Total Cholesterol**

The majority of research does not support the exercise-induced training effect in order to reduce plasma total cholesterol (TChol). According to Durstine et al. (2002) while few investigations have found reductions only after body composition improvements (Despres et al., 1999; Kiens & Lithell, 1989; Wirth, et al., 1985; Wood et al., 1988), the majority have not supported the exercise induced TChol reduction (Couillard et al., 2001; Kokkinos et al., 1995; Stefanick et al., 1998; Thompson et al., 1980; Warner, Ullrich, Albrink, & Yeater, 1989; Wood et al., 1988; Wood, Stefanick, Williams, & Haskell, 1991), unless accompanying dietary interventions (Durstine et al., 2001). Furthermore, no direct exercise-based mechanism has been suggested to influence the clearing of plasma cholesterol in order to reduce total cholesterol concentrations.

**Low Density Lipoprotein-Cholesterol**

The primary issue in the reduction of CHD risk is the reduction of low density lipoprotein-cholesterol (LDL-C); however, investigations into the effects of exercise training, without any dietary intervention, usually do not support a reduction of LDL-C (Despres et al., 1999; Gaesser & Rich, 1984; Kokkinos et al., 1995; Thompson, Thomas, Araujo, Albers, & Decedue, 1985; Wood et al., 1983). Ziogas et al. (1997) compared sedentary, recreational, and trained athletes demonstrating different cross-sectional levels of training, specifically a greater volume of training, may contribute towards a reduction
of proatherogenic LDL subfractions. Further supporting the exercise training induced, antiatherogenic distribution of LDL subfractions, Halle et al.’s (1997) cross-sectional investigation using hypercholesterolemic men (> 240mg/dl) reported lower concentrations of small, dense LDL with increased levels of physical activity. However, when experimentally investigated by Williams et al. (1990), no LDL-C distribution changes were reported after 1 year of exercise training even with significant weight loss in healthy, overweight men. Thus, until LDL-C and its subfractions can be measured directly (Can et al., 2010), there is insufficient evidence to support the training-induced reduction and/or redistribution of LDL-C (Durstine et al., 2002). Interestingly, both investigations that supported the effects of exercise training on LDL-C subfraction distribution (Halle et al., 1997; Ziogas et al., 1997), also demonstrated lower cross-sectional TG.

High Density Lipoprotein-Cholesterol

The majority of research suggests that exercise training has a dose-dependent influence over high density lipoprotein-cholesterol (HDL-C) changes (Couillard et al., 2001; Kodama et al., 2007; Seip et al., 1993; Stefanick et al., 1998; Thompson et al., 1985). According to Durstine et al. (2001), training of 12 weeks or longer has reported HDL-C absolute (2-8mg/dl) and relative (4-22%) increases; however, as is with acute exercise, HDL-C increases are dose dependent in terms of training volume. Kokkinos et al. (1995) took an applied approach by surveying middle-aged, healthy, nonsmoking males about the relationship between miles run per week and current concentrations of HDL-C, observing a gradual increase in HDL-C with increases in miles run.
Specifically, they observed an increase of 0.308mg/dl with every 1 mile run per week, while also finding significantly higher HDL-C concentrations at 7 or more miles per week when compared to a sedentary group. Similarly, a more recent training investigation suggested that a threshold of walking/jogging 11 miles per week is needed (Slentz et al., 2007). Meta-analyses of the exercise training investigations, including control trials, demonstrated HDL-C increases of 1.9mg/dl (Halbert et al., 1999) and 2.53mg/dl (Slentz et al., 2007) due to walking and exercise of 900kcal and/or 120mins, respectively. Moreover, the HDL-C increase was accounted for in the transfer from HDL\textsubscript{3}-C to HDL\textsubscript{2}-C, resulting in a significantly increased HDL\textsubscript{2}-C (Crouse et al., 1997; Campbell, Moffatt & Kushnick, 2011). The subfraction transfer theory results in an increased HDL particle size (Slentz et al., 2007; Thompson & Rader, 2001; Wilund et al., 2002) and therefore, considered antiatherogenic and suggested to fuel the RCT pathway (Durstine et al., 2002).

Other investigations set out to compare what factors might contribute to exercise in order to increase HDL-C, specifically, to explore the effects of aerobic fitness and/or body composition. Katzmarzyk et al. (2001) set out to investigate this issue by using the HERITAGE Family Study sample of a large population made up of both male and female healthy but sedentary subjects. Results from the HERATIGE Family Study suggested general body fatness was a better predictor of change in blood lipid levels than were changes in fitness. Furthermore, baseline blood lipid levels were the best predictors, suggesting an inverse relationship between baseline data and the direction of change due to training (Katzmarzyk et al., 2001). Moreover, Slentz et al. (2007) in their STRRIDE
investigation explored acute exercise, exercise training as well as the short- (72h) and long-term (5-15 days) effects of exercise cessation. They concluded that 30min of vigorous exercise (jogging) can sustain an improvement of HDL-C and HDL particle size over 2 weeks (15 days) posttraining. Past research (Newnham & Barter, 1990; NIH, 2001) has linked TG and HDL metabolism, and Slentz et al., (2007) supported this metabolic connection by demonstrating a reduction in VLDL-TG that was sustained for the same 2 weeks posttraining. Furthermore, HDL-C and TG were the only blood lipid variables which sustained these improvements over the 15 days of exercise cessation.

Triglycerides

As stated above, the inverse relationship between TG and HDL-C has a strong base of support (Halle et al., 1999; NIH, 2001); however, exercise training does not always significantly improve both variables. According to Leon and Sanchez (2001), an increase HDL-C is the most widely demonstrated result of exercise training, while the reduction in plasma TG does not occur as often. However, amongst a wide variety of populations (lean, obese, healthy, diseased, male, female, old and young), the majority of exercise training investigations have demonstrated TG reductions (Durstine et al., 2002; Grandjean et al., 1998; Kelley et al., 2004a; Slentz et al., 2007) with a significant mean of 11% (Kelley et al., 2004b) and ranging from as little as 7% (Thompson & Rader, 2001) and as much as 20% reductions of blood TG (Seip et al., 1993). The most recent data suggests that exercise training may have an influence over blood lipids, as much as 2 weeks post training (Slentz et al., 2007).
Slentz et al. (2007) compared three exercise methodologies in order to distinguish between the contributions of intensity versus volume of aerobic training. They compared high volume/high intensity (1,681 kcal/week at 65-80% VO$_2$max), low volume/high intensity (1,104 kcal/week at 65-80% VO$_2$max), and low volume/moderate intensity (1,075 kcal/week at 40-55% VO$_2$max) exercise prescriptions, relative to subjects’ initial fitness levels for a period of 8-9 months. The chronic exercise training results of this methodology provided evidence in support of both acute and chronic exercise TG reductions. Most importantly, the moderate intensity training regimen (40-55% VO$_2$max) resulted in TG reductions that were present 24 hours postexercise (24hPE) and were sustained 15 days after the last training session. Furthermore, both high intensity training regimens resulted in significant total TG reduction 24hPE; however, the reduction returned to baseline after only 5 days of exercise cessation. Therefore, the TG reductions brought on by the high intensity training program may be the result of the last training session, not necessarily the 8-9 months of high intensity exercise training.

Slentz et al. (2001) were not the first to investigate the sustained effects of exercise training and detraining over an extended exercise cessation period (Crouse et al., 1997; Giada et al., 1995; Thompson et al., 1985; Wang & Chow, 2004). Crouse et al. (1997) used methods similar to Slentz et al.’s (2007) investigation, comparing both the acute and chronic effects of exercise training at different intensities. However, no TG reductions were indicated in a sample of 12 hypercholesterolemic subjects, over a 24-week training period expending approximately 1,050 kcal/week. These conflicting TG results can be explained by two major methodological differences. First, the overall
length of exercise training differed by approximately 3-4 months between the two investigations (Crouse et al., 1997; Slentz et al., 2007). Second, the small sample size used by Crouse et al. (1997) may have diminished the exercise effect and statistical significance. However, both training regimens resulted in significant increases in HDL-C at high and moderate intensity exercise regimens, thus, if Crouse et al. (1997) had collected a larger sample, TG reductions might have reached the level of statistical significance.

In order to better understand the effects of exercise on the human body, researchers have used a more stable environment and timeframe in the acute exercise investigation model, typically measuring within 72h after completion of the single exercise treatment. More importantly, past literature (Crouse et al., 1997; Leon & Sanchez, 2001; Slentz et al., 2007) suggested that acute and chronic exercise can wield distinct and interactive blood lipid responses.

The Effects of Acute Exercise on Blood Lipid Variables

To date, the most common explanation for the acute exercise effect on the human body involves the mass and activity of lipoprotein lipase (LPL and LPLα), the essential enzyme needed to accelerate the body’s lipid distribution system. Exercise has been demonstrated to accelerate LPLα as well as significantly increase the concentration of LPL within the blood (Perreault, Lavely, Kittelson, & Horton, 2004). Physical inactivity has been shown to reduce LPLα and thus, allow greater accumulation of TG within the blood. Cross-sectional studies have also demonstrated that the more physically active subjects can have a greater LPLα (Hamilton, Hamilton, & Zderic, 2004). A single bout
of exercise may reverse the effects of inactivity, thus increase LPLa several times over baseline (sedentary) values (Hamilton et al., 2004; Perreault et al., 2004; Zhang et al., 2002). After moderate intensity exercise, these LPLa changes seem to have a delayed effect, ranging 4-48h postexercise (Zhang et al., 2002). Thus, greater physical activity levels may be attributed to increased LPL hydrolysis (Gupta, Ross, Myers, & Kashyap, 1993) and greater fat clearance. Furthermore, the increased skeletal muscle and adipose tissue blood flow associated with increases in physical activity can combine to enhance reverse cholesterol transport (RCT), resulting in a combination of benefits after just a single bout of activity.

Following a single bout of exercise, some literature demonstrates marked alterations in blood lipids during the hours following the bout, especially regarding HDL-C and TG. Generally, most of the current literature suggests there are no reductions in total cholesterol (TChol) or LDL-C during (Mestek et al., 2006), immediately after (Davis, Bartoli, & Durstine, 1992; Jafari et al., 2003; Visich et al., 1996), 24hPE (Davis et al., 1992; Visich et al., 1996), and 48hPE (Davis et al., 1992; Mestek et al., 2006). When the literature has indicated acute changes of TChol or LDL-C (Ferguson et al., 1998), it is often suggested that these changes may be the result of the inability to completely adjust for changes in plasma volume (Ferguson et al., 2003).

Literature seems to support one of two exercise stimulus theories. One leading theory suggests, based on training status, that there is an energy expenditure threshold with which an individual must reach in order to induce blood lipid changes (Visich et al., 1996). The other related theory suggests a graded response to energy expenditure; or in
other words, the more energy expended during exercise results in greater blood lipid changes. Much like the threshold theory, the graded response theory is dependent up on the training status of subjects (Durstine et al., 2001). Genetic makeup, sex, and pre-exercise blood lipid concentrations all may contribute to or hinder any postexercise blood lipid improvements. Recent investigations that have been unsuccessful at inducing significant changes in HDL-C in women (Imamura et al., 2000; Magkos, Wright, Patterson, Mohammed, & Mittendorfer, 2006; Weise, Grandjean, Rohack, Womack, & Crouse, 2005) highlight the need for further research into the combined effects of exercise and the menstrual cycle on acute blood lipid changes. Additionally, the presence of nonresponders, a subject population unresponsive to the exercise stimulus (Durstine et al., 2002), highlight the need for investigations based on strong statistical power. With these important factors in mind, the need for consistency among all aspects of the exercise investigation is paramount; unfortunately, consistency is not a strength of many acute exercise investigations.

*The Influence of Exercise Variables*

The majority of acute exercise investigations demonstrate HDL-C increases 18-72hPE with as much as 43% increases at an apex of 24hPE, (Bounds, Grandjean, O'Brien, Inman, & Crouse, 2000; Crouse et al., 1997; Dufaux, Order, Muller, & Hollmann, 1986; Ferguson et al., 1998; Foger et al., 1994; Gordon et al., 1994; Grandjean, Crouse, & Rohack, 2000; Kantor, Cullinane, Herbert, & Thompson, 1984; Kantor, Cullinane, Sady, Herbert, & Thompson, 1987; Mestek et al., 2006; Sady, Cullinane, Herbert, Kantor, & Thompson, 1984; Visich et al., 1996); however, attenuated over time
(Thompson & Rader, 2001). This is an important methodological finding, because if investigations set out to find changes in HDL-C, measurements must not be taken too early (1hPE and 6hPE; Visich et al. 1996) nor too late (120hPE and 192hPE; Foger et al. 1994), but instead, follow the established pattern of LPLa.

Acute exercise investigations measuring a possible drop in TG have produced mixed results (Bounds et al., 2000; Ferguson et al., 1998; Foger et al., 1994); however, the majority of investigations have shown a consistent drop in serum TG (Durstine et al., 2002). Meta-analyses demonstrate significant acute TG reductions, ranging from 4% to 37%, with absolute changes as much as 5 to 38mg/dl with exercise alone (Thompson & Rader, 2001). Several authors have investigated this reduction due to training (Crouse et al., 1997; Danner et al., 1984; Gaesser & Rich, 1984); however, the majority of these authors have concluded that reductions are only temporary, returning to previous values with a few days postexercise (48hPE) (Crouse et al., 1997; Danner et al., 1984; Morio et al., 2004; Raz, Rosenblit, & Kark, 1988). Only after regular acute bouts, within 48h of another, can the average person show consistent TG reductions due to exercise alone. However, still in question are the characteristics of the acute treatment protocols, such as energy expenditure, intensity, measurement timing, and the training/health status of subjects.

Carlson and Mossfeldt (1964) found an average 40% reduction in serum TG, starting 2-3h and persisting for at least 44h after completing a cross-country ski-race (85km). According to these authors, this was the first time a reduction in serum TG was directly attributed to a recent bout of exercise and was sustained beyond a few hours.
postexercise. Later, Holloszy et al. (1964) investigated the effects of 6 months of training on blood lipid variables and concluded that regular exercise, at least every 2 days (approximately 44h), would consistently reduce serum TG to manageable levels. Their findings and inferred conclusions are now the consensus and in clinical practice today (NIH, 2002). However, there have been many methodological innovations since the early exercise investigations of the 1960s. Most importantly, the plasma volume adjustment formula, created by Dill and Costill (1974). Dill and Costill, set the standard for plasma volume change estimation for all extended exercise-bout investigations that is still in use today. Prior to Dill and Costill (1974), the plasma volume shift due to an exercise-induced increase within intravascular pressures was suspected; yet it is still unquantifiable. Therefore these past investigations provided accurate but, unadjusted blood lipid results, as demonstrated by the extreme (> 40%) TG reductions (Carlson & Mossfeldt, 1964; Holloszy, Skinner, Toro, & Cureton, 1964). Post-Dill and Costill (1974), acute exercise research has provided support for a much more conservative blood lipid change, if at all. Thus, the only blood lipid results that will be discussed further are those that have applied the plasma volume correction method formulated by Dill and Costill (1974) or another equivalent method.

More recent acute exercise investigations, during (Ferguson et al., 2003) or immediately following exercise (1-3hPE), failed to show any significant blood lipid changes (Hurter, Peyman, Swale, & Barnett, 1972; Lampman et al., 1978). Methodological errors were suggested to be the culprit; specifically the timing of measurements did not persist through 44h, thus supporting the theory of delayed LPLa
contributing to postexercise blood lipid adjustments. Further research, across several disciplines of sport, demonstrated immediate increases in TG (3%) due to maximal intensity short-term treadmill exercise bouts (Sgouraki et al., 2004). Investigators attributed the overall increases in TG to the characteristics of the exercise treatment and timing of measurements. Specifically, the short duration and incremental designed protocol of the maximal exercise bout prevented the body from utilizing its lipid stores via β-oxidation. Therefore, the influence of real time-exercise will either have no effect or will increase serum TG. Furthermore, these conclusions support Oscai et al.’s (1990) theory, suggesting that serum TG contributes little as a fuel source, but rather helps to replenish intramuscular TG, thereby causing the delayed (~24h) depletion of serum TG.

The aerobically trained subject allowed many investigators to explore extreme amounts of energy expenditures in order to induce blood lipid changes. Kantor et al.’s (1984) results demonstrated significant HDL-C increases, from pre-exercise values (24h prior) of 15% (10mg/dl) and 20% (13mg/dl) and TG reductions of 36% (34mg/dl) and 27% (26mg/dl), 18h and 42h after running a marathon, respectively. Dufaux et al. (1986) analyzed blood samples taken after a 3h foot race at 75 ± 5% of their corresponding onset of blood lactate accumulation (OBLA, lactate = 4mmol/L) and concluded significant increases of 3mg/dl (6%) in HDL-C 1 day postexercise. Dufaux et al. (1986) linked this HDL-C change with an increased lecithin cholesterol acyl-transferase (LCAT) activity (LCATa). However, more recent investigations suggest acute exercise has no influence over LCATa (Grandjean et al., 2000). Grandjean et al. (2000) suggested that LCATa is
more dependent upon substrate availability, free cholesterol (FC) and less dependent upon the energy expended during exercise.

Sady et al.’s (1986) results further supported the main theory of a delayed LPLa by demonstrating a clear drop in fasting TG of 24mg/dl (26%) and an increase in HDL-C (10%), both associated with a 46% increase in LPLa after running a marathon. Frey et al. (1993) provided further support for LPL’s delayed effects by demonstrating TG reductions (31%) and beneficial HDL composition adjustments 20h after a 30km running race. Furthermore, the training status of subjects has been demonstrated to influence LPLa and, thereby, influence postexercise TG changes. More specifically, training status can significantly contribute to an increased (physically active) or decreased (sedentary) ability to clear VLDL from the blood via LPL hydrolysis (Perreault et al., 2004). Thus far, the majority of investigations have tested the trained subject exercising at extreme energy expenditures and intensities in order to induce blood lipid changes. More recent investigators have sought to better define, characterize, and specialize the exercise treatment needed in order to induce blood lipid changes in a wide range of subjects.

Cullinane et al. (1982) and Kantor et al. (1987) compared the trained and sedentary subject’s response to an equivalent amount of exercise. After stationary cycling at 80% of heart rate max for 1h (untrained), 1h (trained) and 2h (trained), Cullinane et al.(1982) demonstrated no change in HDL-C and a 33% TG reduction in only one of the three groupings, the trained group of cyclists who endured a 2h exercise session. Although acute exercise did not significantly impact HDL-C in these trained and untrained groups, their baseline HDL-C were significantly different between the trained
an untrained groups (56 vs. 45mg/dl, respectively). Testing an equivalent group of subjects, Kantor et al. (1987) found significantly different baseline differences in HDL-C concentrations between their trained (51mg/dl) and untrained (33mg/dl) groups. Furthermore, training status may have an influence over the HDL-C subfraction composition. More specifically, pre-exercise HDL-C subfractions were greater depending on the training status of subject groups. It was observed that the trained population has greater HDL\textsubscript{2}C, while the untrained had greater HDL\textsubscript{3}C. However, Kantor et al. (1987) demonstrated significant TG changes and an exacerbation of these same pre-exercise HDL-C subfraction differences, resulting in increases in both associated subfractions, HDL\textsubscript{2}C and HDL\textsubscript{3}C for the trained and untrained subjects, respectively. However, these results may just be a reflection of subjects’ pre-exercise HDL subfraction concentrations and not necessarily consistent for all untrained subjects.

In further analysis of these investigations, one area which has been improved upon in more recent investigations is the ability to quantify the exercise regimen and further control its treatment. The methodology of Cullinane (1982) and Kantor (1987) set out to investigate a typical “hard” workout session for a trained and untrained subject; however, the authors arbitrarily quantified the different exercise sessions according to duration, 1h vs. 2h at an equal intensity. Additionally, Kantor et al.’s (1987) intensity of cycling was controlled via heart rate monitoring, but this measure is considered inaccurate today. Even with adequate fluid replacement, according to the cardiac drift theory, heart rate increases with exercise duration due to increases in body temperature as well as arterial blood plasma volume shifts (Brooks, Fahey, White, & Baldwin, 2000).
Davis et al. (1992) set out to determine the effects of intensity on a single group of trained subjects. The authors designed two, random, equal energy expenditure (~952 kcal) exercise treatments, running on a treadmill at 50% (~90 min) and the other at 75% (~60 min) VO$_2$max, resulting in no blood lipid changes at 1, 24, 48 and 72 h PE. In conflict with Davis et al. (1992), Gordon et al.’s (1994) intensity investigation resulted in no TG reductions, but a 13% increase in HDL-C 24 h PE. Gordon et al. (1994) investigated 12 recreational runners expending 800 kcal at 60% and 75% VO$_2$max with 1-2 weeks separation in between the two trials. However, HDL-C increases were only found within the high intensity (75% VO$_2$max) exercise treatment and not within the 60% VO$_2$max exercise treatment group. Therefore, intensity played a significant role in improving HDL-C within a moderately fit group of recreational runners. However, these conclusions do not explain the reasoning for the conflicting results between Davis et al. (1992) and Gordon et al. (1994).

Davis et al.’s (1992) methodology accounted for many of the major issues seen in past research: an adjustment for possible plasma volume shifts, equal and measured energy expenditures (951 vs. 953 kcal), and adequate dietary control. However, there are still methodological limitations when compared with more recent literature. First, although a plasma volume shift was implemented (Greenleaf, Convertino & Mangseth, 1979), this plasma volume adjustment method does not use today’s gold-standard (Dill & Costill, 1979) and thus, may have confounded the results. Secondly, only the high intensity exercise resulted in a significant plasma volume change, while the low intensity exercise did not change over the 72 h of measurement. This suggests the low intensity
exercise treatment was set at an inadequate intensity (50%) and thus, unable to induce a plasma volume shift and/or any blood lipid improvements, which may very well be the case in a highly-trained group of subjects. Finally, all subjects (n = 10) participated in both the high and low intensity exercise treatments. In this case, only 1 week was given between the two exercise treatments and might have also confounded the results between the two exercise trials.

Generally, the untrained population does not need to endure such extreme energy expenditures in order to see blood lipid changes. Crouse et al. (1995) and Park and Ransone (2003) tested the untrained population (31.1 and 44.9ml/kg/min) by setting an extremely low acute bout energy expenditure limit. Additionally, both investigations added a second bout of exercise, with an adequate “flush out” period (1-2 weeks), in order to further test the effects of intensity. First and foremost, a single bout of exercise to expend 350-400kcal resulted in significant blood lipid improvements in both investigations. However, much like previous investigations (Davis et al., 1992; Gordon et al., 1994), the effects of intensity were in conflict. Crouse et al.’s (1995) results demonstrated HDL-C increases of 8% and TG reductions of 18% were independent of intensity (50% vs. 80%VO₂max), as long as the total energy expenditure was held constant. Thus, Crouse et al.’s (1995) results support the intensity-independent results of Davis et al. (1992).

In opposition, Park and Ransone (2003) demonstrated HDL-C increases of 7% and TG decreases of 10% after expending about 411kcal at the subject’s individual lactate threshold (on average 76.9 ± 8.6%VO₂max). However, no significant changes
were evident after an equivalent energy expenditure of 405kcal at 70% of their individual lactate threshold (45.1 ± 14.1%VO$_2$max). Therefore, according to Park and Ransone (2003), intensity does significantly influence the effects of a single bout of walking on a treadmill to expend about 400kcal. Furthermore, this result is in agreement with the previously literature (Gordon et al., 1994) demonstrated in a trained population.

Therefore, in review, Davis et al. (1992) subjected 10 well-trained runners to expend 950kcal, but it was not enough to induce blood lipid changes in either of the groups. Therefore, regardless of intensity, the amount of energy expenditure was inadequate for this trained population. Gordon et al. (1994) subjected “recreational” runners to a more modest amount of energy expenditure (800kcal) and demonstrated that exercise intensity needs to be considered, when trying to raise HDL-C via exercise. Next, Crouse et al. (1995) subjected an untrained group of subjects to only 350kcal resulting in HDL-C increases and TG decreases. However, these blood lipid improvements were equal among the two different exercise intensities. Lastly, Park and Ransone (2003) demonstrated a possible answer to this debate using an untrained population. Park and Ransone (2003) demonstrated that exercise-induced changes were dependent upon the relative lactate threshold of each subject and not a specific percentage of VO$_2$max. Therefore, exercise intensity had a significant influence over blood lipid changes; however, energy expenditure and the training status of subjects may be a larger part of the equation.

These results inspired other researchers to steer away from intensity comparisons and instead, to define the exercise “threshold” of total energy expenditure (kcal) needed
to optimize the benefits of a single bout of exercise and to specialize that treatment for a wide range of populations. Visich et al. (1996) and Ferguson et al. (1998) investigated equivalent groups of trained subjects (56.4 and 56.2ml/kg/min) at a variety of kcal expenditures ranging from 400-1,500kcal at equivalent intensities of 75% and 70% VO₂max, respectively. Visich et al. (1996) analyzed the blood of 12 trained runners after expending 400, 600 and 800kcal, only after collapsing all groups together did TG decrease (9mg/dl, 13%) and HDL-C increase (3mg/dl, 6%) 24hPE. The authors concluded that blood lipid improvement may be less dependent upon a kcal threshold and more dependent upon the duration of exercise and maybe, more importantly, the training status of subjects.

These theories were tested by Ferguson et al. (1998), who continued where Visich et al. (1996) left off, by testing the blood of an equivalent group of subjects (11 trained men) after they expended 800, 1,100, 1,300 and 1,500kcal. Ferguson et al. (1998) demonstrated exactly what Visich et al. (1996) noted as their limitations: a similar group of subjects did not improve HDL-C, but they did see a reduction in serum TG (31mg/dl, 26%) 24hPE. However, treatments equal to or beyond 1,100kcal produced significant HDL-C increases and TG reductions 24hPE. Thus, according to these results, the trained athlete must endure at least 800kcal and 1,100kcal of energy expenditure in order to see TG and HDL-C improvements, respectively. Additionally, as the energy expenditure increases, so too does the impact on blood lipid improvement, as seen in the most extreme energy expenditure of 1,500kcal which induced HDL-C increases (7mg/dl, 15%) and TG reductions (22mg/dl, 20%) through 48hPE.
Therefore, based on these results, the minimum energy expenditure for the trained subject approaches 800kcal, depending on the specific objectives, with variability due to genetics and training regimens. The threshold effect is nothing new: In order to cause adaptation within the body, individuals must push the body beyond the routine. Therefore, the trained athlete, who may expend over 1,000kcal per training session, will not see beneficial adaptations after merely 350kcal. However, the recreationally active/sedentary male may benefit from a more modest acute aerobic activity, depending on their relative fitness level and subject-specific health and fitness goals. Thus far, the most influential exercise variable has repeatedly been the training status of subjects. However, if this aspect is controlled for, via the screening of subjects, another variable emerges.

**The Influence of Blood Lipid Variables**

An extensive review of the literature provided a number of works in support of the inverse relationship between HDL-C and TG (Bounds et al., 2000; Crouse et al., 1995, 1997; Dufaux et al., 1986; Ferguson et al., 1998; Grandjean et al., 2000; Lamon-Fava, McNamara, Farber, Hill, & Schaefer, 1989; Park & Ransone, 2003; Sady et al., 1984; Visich et al., 1996) and thus, it has been well established among most researchers (Durstine et al., 2001; Miller, Langenberg, & Havas, 2007). Grandjean et al. (2000) correlated selected physiological variables and baseline lipid data of both normal and hypercholesterolemic subjects, resulting in a moderate, inverse correlation between HDL-C and TG (-0.761). Miller et al. (2007) suggests a combination of factors linking these two important variables. The first, the inefficient catabolism of TG-rich lipoproteins,
resulting in abnormal apolipoprotein distribution and the inefficient creation of nascent HDL particles. The second, the CETP-associated creation of TG-rich HDL allows for a more rapid depletion of its core contents (cholesterol ester, CE) and effectively reduces HDL-C.

However, other authors demonstrated that this inverse relationship is not always so obvious (Frey, Baumstark, & Berg, 1993; Gordon et al., 1994; Kantor et al., 1987). Some investigations demonstrate significant rise in HDL-C, without a reduction in TG (Gordon et al., 1994; Kantor et al., 1987). One explanation for the results provided by Gordon et al. (1994), suggests that the below average initial TG value of 69mg/dl, resulted in a reduced absolute TG reduction. Kantor et al.’s (1987) results also did not show significant TG reductions; however, inconsistent methodology (specifically a baseline blood measurement of 24h prior to exercise instead of immediately prior to the start of exercise) may have contributed to the differing TG results. Frey et al.’s (1993) investigation found just the opposite, demonstrating significant TG reductions without HDL-C increases. Again, methodological differences may be to blame for the inconsistent results, because the blood measurement occurred 20hPE rather than the consistently demonstrated 24hPE. However, Frey et al.’s (1993) results were aimed at providing insight into the possible metabolic changes prior to the 24hPE blood draw. Therefore, their results demonstrated significant metabolic exchange among the VLDL and HDL subfraction particles (HDL₂ and HDL₃) and consequentially, the HDL sub-particles are left with an increased cholesterol content. Secondly, the authors suggest the
HDL$_2$ sub-particle acts to transport TG-rich lipid remnants released during the spike of LPL$_{a}$ during exercise and the hours after (24hPE-48hPE).

The influence of acute exercise on blood lipid improvements has been shown consistently in a normolipidemic group of subjects; however, few authors have compared the acute effects of exercise in a normo and hyperlipidemic group of subjects. Crouse et al. (1995) analyzed the blood of an untrained, hypercholesterolemic (TC = 245mg/dl, >90$^{th}$ percentile) group of men after completing either a moderate (50$\%$VO$_{2\text{max}}$) or high (80$\%$VO$_{2\text{max}}$) intensity exercise bout to expend 350kcal. Demonstrated by past investigations and confirmed here, blood lipid changes were independent of intensity in a healthy and/or trained population. However, these intensity-independent results demonstrated the expected rise of HDL-C and reduction of serum TG in a unique hypercholesterolemic population and therefore, these results must be highlighted.

Examining this investigation further, the results show a population with above average LDL-C (173mg/dl), TC (254mg/dl) and borderline high TG (177mg/dl) prior to any exercise stimulation. As seen in the normocholesterolemic group of subjects, the hypercholesterolemic group demonstrated a rise in HDL-C at 24hPE and 48hPE (3mg/dl, 6.6%; 4mg/dl, 8.8%) and a drop in TG at 24hPE and 48hPE (33mg/dl, 19%; 26mg/dl, 15%, respectively). However, unlike the majority of normocholesterolemic subject investigations, adjustments of TC and LDL-C did accompany the 24hPE (no change and 10mg/dl, 6%) and 48hPE (12mg/dl, 5% and 14mg/dl, 8%, respectively) results of the hypercholesterolemic population, thus, exercise induced a unique postexercise blood lipid response in this hypercholesterolemic group (Crouse et al., 1995). Authors suggest a
dysfunctional mass and/or activity of the lipid transferring enzymes (CETP, LCAT and/or HL) as the primary difference between these groups of untrained subjects. However, more recent investigations dispute these delayed TC and LDL-C changes (Grandjean et al., 2000; Park & Ransone, 2003) and instead, suggest similar acute exercise responses between the normo- and hypercholesterolemic subject. Therefore, further research is needed to elucidate the influence of initial blood lipid concentrations on blood lipid responses to acute exercise.

Exercise’s beneficial effects, much like any other treatment, are limited by the body’s genetic minimum and/or maximum limits. Therefore, when compared to the untrained subject, the trained athlete needs to workout with greater intensity and frequency to achieve much smaller cardiovascular benefits. Taking this into account, subjects with initially above average HDL-C, whether that is the result of genetic advantages or exercise training, can also reach their individual ceiling and thus, demonstrate a much smaller absolute HDL-C rise. Several past investigations support the idea of baseline blood lipid concentrations influencing postexercise blood lipid changes (Cullinane, Lazarus, Thompson, Saratelli, & Herbert, 1981; Davis et al., 1992; Ferguson et al., 1998; Visich et al., 1996). Subjects with initially above average HDL-C (56mg/dl and 55mg/dl) demonstrated no statistically significant HDL-C increases (Cullinane, Siconolfi, Saritelli, & Thompson, 1982; Davis et al., 1992, respectively), while average HDL-C increases of 3mg/dl (8%) and 7mg/dl (17%) were demonstrated in those subject’s with an initially low HDL-C (41mg/dl and 40mg/dl; Ferguson et al., 1998; Visich et al., 1996, respectively). Gordon et al. (1994) tested this theory on their own data, concluding
the presence of a significant inverse relationship \( r = -0.49, p < .05 \) between the baseline and 24hPE HDL-C value. Furthermore, this inverse association is also true for those subjects with initially lower concentration of serum TG.

Previous research has demonstrated subjects with higher initial TG concentrations are associated with greater postexercise decreases in terms of absolute change (mg/dl) (Crouse et al., 1995; Ferguson et al., 1998; Foger et al., 1994; Grandjean et al., 2000). However, percentage TG changes between subjects with elevated TG and normal TG should be fairly equal. For instance, Visich et al. (1996) used trained subjects with below average initial TG concentrations (67mg/dl) and demonstrated a nine mg/dl (13.4%) reduction 24hPE; however, Crouse et al.’s (1995) above average initial TG concentrations were reduced 18mg/dl, (10.5%) after a similar 500kcal exercise bout. Therefore, when considering serum TG changes alone, absolute differences (9 vs. 18mg/dl) were much greater in those with elevated TG, while percentage differences are much closer in comparison. The most recent comparable acute exercise investigation discovered similar HDL-C and TG responses between hypercholesterolemic and normocholesterolemic groups (Grandjean et al., 2000). Based on the literature, this comparison best illustrates the absolute and relative TG response to acute exercise, however, differences in subject fitness levels suggest further research, with comparable subject groups is needed. Therefore, results with initially elevated TG may produce greater absolute TG changes postexercise and have a greater delayed effect versus a sample with initially average concentrations of blood TG.
After extensive review of the literature, no investigation has compared the absolute and percentage change between normal and above median TG subjects after a single bout of exercise with similar energy expenditure, mode and intensity through 48h. Many investigations have set out to compare other blood lipid variables after a drop in TG is achieved, in this investigation the TG reductions is the focus. Since there is no direct evidence, it is important to consider if the magnitude of change in postexercise TG concentrations is directly related to the initial concentration in both absolute (mg/dl) and relative (%) terms.

Secondly, primary research has demonstrated that acute exercise has a significant influence over the TG-HDL-C relationship. Therefore, it is also important to compare the indirect influence of initial TG concentrations, on the other blood lipid variables (HDL-C, TC, LDL-C).
CHAPTER 3: METHODS

Subjects

Participants between 20 and 34 years of age (24.29 ± 4.40; see Table 1) were recruited from Ohio University’s campus and Athens local communities. The majority of recruitment was completed by advertisement in the form of flyers and emails (see Appendix A). Subjects were nonsmokers and free of any orthopedic limitations that would interfere with their ability to exercise and not using any medications known to alter their metabolism. Prior to initial screening, all subjects were adequately informed of all benefits, risks, and the purpose of the study, as well as completing the informed consent process approved by Ohio University’s Institutional Review Board (see Appendix A). Overall, a total of 98 potential participants volunteered and were screened for qualification. However, only 17 qualified for one of two experimental groups: lower triglyceride (TG) group (LOW; n = 9) or higher TG group (HIGH; n = 8) and completed all components as laid out by the informed consent document. All disqualified subjects were excluded due to one of four informed consent violations explained in greater detail below; a) initial TG concentrations falling within established exclusionary limits (n = 32), b) body composition percentages falling outside the established range (n = 21), c) an inability to adhere to the informed consent guidelines (e.g. refraining from exercise and/or fasting prior to blood draws, n = 16), and c) aerobic fitness values falling outside the established range (n = 11). Lastly, one participant who qualified according to all preliminary screening tests, later revealed a difference between SCREEN and
BASELINE TG blood samples and thus, was excluded, further explanations are provided below (n = 32 + 21 + 16 + 11 + 1 = 81).

Preliminary Screening

All potential subjects demonstrated an aerobic fitness between the 25\textsuperscript{th} and 75\textsuperscript{th} percentiles according to standard values (40.5-52.5 ml/kg/min) as determined by a graded exercise treadmill test (Whaley et al., 2006) and within the 25\textsuperscript{th} and 75\textsuperscript{th} percentile for body fat percentage according to standard values (12-25\%BF), as determined by a BOD POD (Whaley et al., 2006) for men this age. Additionally, participants reported to the laboratory for a blood draw, denoted as blood sample SCREEN, in order to analyze serum TG concentrations. Based on this SCREEN sample, participants qualified for one of two groups defined as either the low (LOW) or high (HIGH) TG group. Below the 25\textsuperscript{th} (70mg/dl) and above the 75\textsuperscript{th} (139mg/dl) percentiles were originally used in order to accurately depict this age group (NIH, 2001; Whaley et al., 2006). However, due to low qualified enrollment numbers and the inherent variability of day-to-day TG, as well as a literature backed reduction of current (1991-2001) TG levels within the population (Ingelsson et al., 2009), the above average TG limit was lowered from the 75\textsuperscript{th} percentile (> 139mg/dl) to above the 50\textsuperscript{th} percentile (> 100mg/dl) in order to increase enrollment. Furthermore, the SCREEN sample was dually used as a disqualification factor as well as a subject specific control by comparing SCREEN and BASELINE TG data. The BASELINE blood sample was taken immediately prior to the experimental exercise trial and within 1 week of the SCREEN sample, thereby disqualification would occur if either serum TG sample (SCREEN or BASELINE) fell within 70-100mg/dl. Therefore, all
participants qualified according to their SCREEN and BASELINE serum TG concentrations consistent with the adjusted TG qualification values in either the low TG group (< 25th percentile; < 70mg/dl; LOW, n = 9) or the high TG group (> 50th percentile; > 100mg/dl; HIGH, n = 8, NIH 2002).

Body Composition

After potential subjects met the initial criteria, body composition was tested within the BOD POD after the participant was instructed to fast and refrain from exercise at least 4h prior. Participants wore a form-fitted swimsuit and a Lycra swim cap (provided) as per manufacturer’s specifications. The subject was fully enclosed within the BOD POD (an oval-shaped chair-chamber) for approximately 3-4, 1min periods. Using two different volumes of air, one known and one unknown and isolating pressure, this device calculated body density. The computer then estimated body fat percentage based on body density and estimates respiratory volumes with specific formulas pertaining to the population tested (Siri, 1993). Eligible participants include those within the 25th-75th percentile for body fat according to standard values (12-25%, as determined by a BOD POD; Whaley et al., 2006). Further accuracy and reliability on air plethysmography methods were reported by Dempster and Aitkens (1995).

Maximal Oxygen Consumption

Maximal oxygen consumption (VO2max) and ventilatory threshold (Beaver, Wasserman & Whipp, 1986) values were determined during a maximal graded exercise test (GXT) on a motor driven treadmill (TMX-425, Full Vision Inc., Newton, KS) using the Bruce protocol in order to measure ventilatory threshold (Bruce, 1963). Gas
exchange and ventilatory parameters were measured with a metabolic system (Truemax 2400 Metabolic Measurement System, ParvoMedics, Sandy, UT). The system provided and recorded immediate calculation of the respiratory exchange ratio (RER; VCO₂/VO₂) and rates of VO₂ and energy expenditure at select time intervals. Indirect calorimetry data was used for the estimation of exercise intensity and the contribution of fat and carbohydrate to energy expenditure (RER). The metabolic system was calibrated according to manufacturer’s recommendations using a gas mixture of known concentrations of O₂ and CO₂ (4%O₂/16%CO₂, Scott Medical Products, Plumsteadville, PA) and a 3L calibration syringe (no. 5530, Hans Rudolph, Inc., Kansas City, MO).

Blood pressure was monitored during the first three stages and heart rate measurements were recorded every 30 seconds via an interface with the metabolic system (Polar CIC Inc., Port Washington, NY) to ensure appropriate physiological progression was made as intensity increased and during recovery until resting values were attained. To be considered a maximal oxygen consumption two of the four criterion were met: a) respiratory exchange ratio (RER) > 1.1; b) heart rate reached within ± 10 beats per minute (bpm) of heart rate max; c) Rating of Perceived Exertion (RPE) ≥ 18; 4) less than 2ml/kg/min increase with an increase in workrate; although it was not encountered, if a participant’s GXT failed to elicit two of the four criterion, they would have been retested (no subjects who qualified for this investigation were retested). The GXT was continued until volitional fatigue. Expired gases were collected through a Hans Rudolph one-way valve (Kansas City, MO) mouthpiece and analyzed for fractions of oxygen and carbon dioxide (TrueOne 2400, ParvoMedics Systems, Sandy, UT). Participants’ ventilator
thresholds were calculated according to the manufacturer’s specifications (Beaver, Wasserman & Whipp, 1986). Maximal oxygen consumption was measured to characterize participants and to estimate intensity of the exercise performed (60 ± 5%) in the experimental exercise trial protocol.

*Three-Day Diet Log*

Subjects completed a 3-day (2 weekdays and 1 weekend day) food diary within a week prior to their first experimental trial. The diets were analyzed for total daily kcal, grams of macronutrients and percentage of total kcal of each macronutrient with Nutritionist Pro software (Version V, Axxya Systems, Stafford, TX). Those with an average dietary fat intake either less than 8% or more than 45% were disqualified from participation for the reason that extreme amounts of dietary fat can alter blood lipid analyses (Warnick & Albers, 1978).

*Exercise Trial*

All exercise trial data collection occurred between the hours of 6:00 a.m. and 10:00 a.m. after an overnight fast (water only; 10-12h). A blood sample was taken by venipuncture procedure from a vein in their arm prior to initiation of exercise and labeled as BASELINE. Subjects were setup for an estimated accumulated kcal exercise bout using the same equipment and setup as the maximal aerobic consumption treadmill test. According to the GXT response, speed and grade were adjusted to induce a 2-3 min low intensity steady state warm-up, within 5-10 min subjects were walking/jogging at a pace to illicit 60 ± 5% of the subjects VO2max (L/min). The accumulated kcal expended were estimated via a variation of Katsanos et al.’s (2004) investigation, measuring direct and
estimating indirect gas volumes throughout the exercise bout. Initially, speed and grade were modified to bring each participant to their previously determined 60 ± 5% VO$_2$max (57 ± 3.8 average; 52-68% VO$_2$max range) to expend a total of 600kcal (608 ± 15kcal average; 585-654kcal range). Modifications were made throughout the trial and water was provided ad libitum during the exercise bout. Subjects rested (seated or semirecumbent position) for 1h after completing the exercise bout and had the first of three postexercise (PE) blood draws, 1h (1hPE) before leaving the laboratory. Subsequently, the subject returned to have a 24h and 48h postexercise (24hPE and 48hPE, respectively) sample collected. As explained within the informed consent process, during this time away from the laboratory, the subject refrained from any exercise outside of their daily required tasks (e.g., walking to and from class) and completed two 10-12h overnight fasts (water only) prior to returning to the laboratory for the 24hPE and 48hPE blood samples.

**Blood Sampling and Biochemical Analyses**

Subjects were instructed to sit down for 10min prior to sampling in order to prevent hemoconcentration of blood variables (Maw, Mackenzie, & Taylor, 1995). Blood draws were taken for qualification and baseline (SCREEN and BASELINE) and 1hPE, 24hPE, and 48hPE and collected from an antecubital vein using a 23-gauge butterfly needle (Vaculet, 26766, Excel, Int., Los Angeles, CA) into a test tube containing EDTA (456023, K2EDT, Greiner Bio-One, Monroe, NC). Hemoglobin and hematocrit were determined and the remaining whole blood was centrifuged (x 2,100g) within 2h of collection at 3°C for 15 minutes using a refrigerated centrifuge (D3720 Sorvall Legend;
Kendro Laboratory products; Germany). Following centrifugation, plasma samples were aliquoted into microcentrifuge tubes (02-682-550, Fisherbrand, Fisher Scientific, Hampton, NH) and stored in an ultra-low freezer at -86°C (5700 series, VWR, Bristol, CT) for determination of blood lipid variables via batch analysis to limit variability of measurement.

Hemoglobin was assessed in duplicate using a portable analyzer (Hgb Pro, ITC, Edison, NJ) by introducing 25 microliters of whole blood onto a preprogrammed analytical strip. If reported concentrations are > 0.3mg/dl different, a third sample was analyzed and the values averaged. The coefficient of variation was small (CV = 0.01%).

Hematocrit was determined in triplicate using the microcapillary method where whole blood is placed into heparinized micro-hematocrit capillary tube (no. 22-362-566, Fisher Scientific, Pittsburgh, PA) and centrifuged using an IEC Micro MB centrifuge (Model 3411, International Equipment Company, Needham Heights, MA) for 7min. Hematocrit (%) was then determined using a circular microcapillary tube reader (Model 2201, International Equipment Company, Needham Heights, MA). The coefficient of variation was small (CV = 0.01%).

Hemoglobin and hematocrit concentrations were used to estimate changes in plasma volume relative to SCREEN values according to the Dill and Costill (1974) plasma volume adjustment formula validated by Maw, Mackenzie, Comer, and Taylor, (1996):

\[
PV (\% \text{ change}) = 100 \left( \frac{PV_a - PV_b}{PV_b} \right)
\]

where \( PV_a = BV_a - CV_a \), \( CV_a = BV_a (Hcta) \), and \( BV_a = BV_b (Hbb / Hba) \)
Metabolic data was then adjusted for shifts in plasma volume prior to statistical analyses. Thus, unadjusted concentrations were determined with the following formula:

\[ PV \text{ Unadjusted Metabolic values} = PV \text{ Adjusted Metabolic value} - (\text{Adjusted Metabolic value} \times \%\text{PV change}), \text{ where (PV= plasma volume).} \]

Assays for the determination of TG, TC and HDL-C were performed according to manufacturer specification using manual procedures and spectrophotometer (Evolution 300, ThermoElectron Corporation, Waltham, MA). Determination of serum TG was performed with a commercially available reagent (T7532, Pointe Scientific, Canton, MI). This procedure is based on McGowan et al.’s (1983) methods, in which TG are enzymatically hydrolyzed to free fatty acids and glycerol. The glycerol is phosphorylated by adenosine triphosphate with glycerol kinase to produce glycerol-3-phosphate and adenosine diphosphate. The Glycerol-3-phosphate is oxidized to dihydroxyacetone phosphate by glycerol phosphatase oxidase producing hydrogen peroxide. Hydrogen peroxide reacts with 3-aminoantipyrine and 3, 5 dichloro-2-dyroxybenzene sulfonate to produce a red dye. The absorbance of this dye at 500nm is proportional to the concentration of TG present in the sample and then compared against the absorbance of glycerol standards (M-103, Document-Casco Standards, Fremont, CA) of known concentration. The coefficient of variation for TG was determined for all unknown and control samples (LIP101, Thermo-MAS, Hampton, NH; CV = 4.88%).
Total cholesterol was assayed according to the technique developed by Allain et al. (1974) using commercially available reagents (C510; Pointe Scientific, Canton, MI). Cholesterol esters are enzymatically released to free cholesterol and fatty acids. The free cholesterol is oxidized by cholesterol oxidase to cholest-4-en-3-one and hydrogen peroxide. The hydrogen peroxide combines with hydroxybenoic acid and 4-aminoantipyrine in the presence of peroxidase to form a quineimine dye which is spectrophotometrically measured at 500nm. The intensity of absorbance is directly proportional to the TC concentration in the sample. Absorbance readings for unknown samples were compared to the known concentrations to generate a standard curve (ES5010, Ever Scientific, Exton, PA) and to quantify their cholesterol concentration. A sample of known cholesterol concentration was analyzed (every 10th sample) to ensure validity of the measurement. The coefficient of variation for TC was determined for all unknown and control samples (LIP101, Thermo-MAS, Hampton, NH; CV = 3.83%).

Determination of plasma HDL-C was performed following the procedures developed by Warnick and Albers (1978), and Gidez et al. (1982). First, a heparin-manganese reagent is prepared to precipitate Apo-B containing lipoproteins, leaving only the cholesterol in the HDL fraction. A small amount (0.5mL) of this supernatant is drawn off and analyzed in the same fashion as described above for TC. The coefficient of variation for HDL-C was determined for all unknown and control samples (LIP101, Thermo-MAS, Hampton, NH; CV = 3.2%).

Plasma low density lipoprotein cholesterol (LDL-C) was calculated according to the Friedewald Equation (Friedewald, Levy, & Fredrickson, 1972):
\[ \text{LDL-C} = \text{TC} - \left[ \text{HDL-C} \times \frac{\text{TG}}{5} \right] \]

Statistics Analysis

All statistics were computed using SPSS 17.0 (Window’s version, SPSS Inc., Chicago, IL). Group means and standard deviations are reported within the results section. Groups were evaluated using group x time repeated measures ANOVAs for main effects, interaction and between group differences of blood lipid variables and changes, respectively. Significance was determined as an alpha \( \alpha < .05 \), any non-spherical data used the Greenhouse-Geisser corrections. Any interaction effect was further distinguished with a one-way ANOVA. Bonferroni’s pairwise comparison post hoc test was used when appropriate; all \( F \) and \( p \) values are reported within the results.
CHAPTER 4: RESULTS

Subjects

A total of 98 participants volunteered to participate in this investigation. However, only 17 qualified and completed all tests, leaving a total 81 ineligible participants (see Figure 1). Subject characteristics of the low triglyceride (TG; LOW; n = 9) and high TG (HIGH; n = 8) groups were compared using independent samples $t$-tests and listed per group as a mean ± standard deviation ($M \pm SD$) and range. There were no differences for subject characteristics between groups (see Table 1).

![Figure 1. Distribution of subject ineligibility. TG, Triglyceride screen = > 70 or < 100mg/dl; BF%, body fat percentage = < 12 or > 25%; Other, unable to fast/refrain from exercise according to consent form guidelines; VO$_2$max, aerobic fitness = < 40.5 or > 52.5ml/kg/min.](image-url)
Table 1

Subject Characteristics

<table>
<thead>
<tr>
<th></th>
<th>LOW Group (n=9)</th>
<th>HIGH Group (n=8)</th>
<th>Combined (N=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td></td>
<td>(Range)</td>
<td>(Range)</td>
<td>(Range)</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>24.56 ± 3.50</td>
<td>24.00 ± 5.48</td>
<td>24.29 ± 4.40</td>
</tr>
<tr>
<td></td>
<td>(20.00 – 30.00)</td>
<td>(20.00 – 34.00)</td>
<td>(20.00 – 34.00)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.81 ± 0.06</td>
<td>1.80 ± 0.09</td>
<td>1.80 ± 0.07</td>
</tr>
<tr>
<td></td>
<td>(1.73 – 1.93)</td>
<td>(1.68 – 1.96)</td>
<td>(1.68 – 1.96)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>83.55 ± 11.12</td>
<td>83.69 ± 9.52</td>
<td>83.61 ± 10.07</td>
</tr>
<tr>
<td></td>
<td>(63.64 – 96.00)</td>
<td>(70.00 – 97.30)</td>
<td>(63.64 – 97.73)</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>18.98 ± 2.86</td>
<td>19.31 ± 4.30</td>
<td>19.14 ± 3.49</td>
</tr>
<tr>
<td></td>
<td>(15.90 – 25.00)</td>
<td>(13.00 – 24.00)</td>
<td>(13.00 – 25.00)</td>
</tr>
<tr>
<td>Fat Mass (kg)</td>
<td>15.93 ± 3.63</td>
<td>16.42 ± 5.04</td>
<td>16.16 ± 4.22</td>
</tr>
<tr>
<td></td>
<td>(12.00 – 22.27)</td>
<td>(9.10 – 23.45)</td>
<td>(9.10 – 23.45)</td>
</tr>
<tr>
<td>Fat-Free Mass (kg)</td>
<td>67.61 ± 8.72</td>
<td>67.27 ± 5.58</td>
<td>67.45 ± 7.19</td>
</tr>
<tr>
<td></td>
<td>(51.23 – 78.91)</td>
<td>(57.46 – 74.27)</td>
<td>(51.23 – 78.91)</td>
</tr>
<tr>
<td>VO_{2max} (ml/kg/min)</td>
<td>47.02 ± 4.04</td>
<td>47.75 ± 3.13</td>
<td>47.37 ± 3.55</td>
</tr>
<tr>
<td></td>
<td>(41.20 – 52.00)</td>
<td>(43.30 – 52.40)</td>
<td>(41.02 – 52.40)</td>
</tr>
<tr>
<td>VO_{2max} (L/min)</td>
<td>3.93 ± 0.64</td>
<td>4.02 ± 0.53</td>
<td>3.97 ± 0.57</td>
</tr>
<tr>
<td></td>
<td>(2.75 – 4.89)</td>
<td>(3.46 – 5.00)</td>
<td>(2.75 – 5.00)</td>
</tr>
<tr>
<td>Ventilatory Threshold (%)</td>
<td>55.67 ± 7.52</td>
<td>56.13 ± 8.20</td>
<td>55.88 ± 7.60</td>
</tr>
<tr>
<td></td>
<td>(48.00 – 73.00)</td>
<td>(48.00 – 68.00)</td>
<td>(48.00 – 73.00)</td>
</tr>
</tbody>
</table>

Note. No differences between group characteristics.

Subjects recorded a 3-day diet log prior to testing and followed that similar diet throughout the 3 days of consecutive testing. Dietary characteristics were compared by a 2 x 3 (group x day) repeated measures ANOVA for the 3-day average amount of total kilocalories (kcal), total grams and percentage of total kcal of each macro-nutrient.
(protein, carbohydrates and fat) per day for each group, reported as standard deviation ($M \pm SD$) (see Table 2). On average, the LOW group ($M = 2965.11, SD = 764.29$) consumed greater kcal than the HIGH group ($M = 2173.92, SD = 439.58, F(1, 1.374) = 6.60, p = .021$) over the 3 days of recording and it was accounted for in grams of Fat (LOW, $M = 109.57, SD = 25.20$ > HIGH, $M = 109.57, SD = 439.58$; $F(1, 2) = 17.68, p = .001$; see Table 2). No further differences were found between or within grams or percentages of macronutrients consumed over the 3 days of testing.

Table 2

3-Day Dietary Characteristics

<table>
<thead>
<tr>
<th></th>
<th>LOW Group (n=9)</th>
<th>HIGH Group (n=8)</th>
<th>Combined (N=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td></td>
<td>(Range)</td>
<td>(Range)</td>
<td>(Range)</td>
</tr>
<tr>
<td>Daily Total (kcal)</td>
<td>2965.11 ± 764.29 $^a$ (2095.00 – 4353.00)</td>
<td>2176.92 ± 439.58 (1539.67 – 2732.67)</td>
<td>2592.78 ± 736.42 (1539.67 – 4353.00)</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>139.16 ± 70.11 (67.78 – 269.00)</td>
<td>91.17 ± 28.65 (47.59 – 121.83)</td>
<td>116.57 ± 58.54 (47.59 – 269.00)</td>
</tr>
<tr>
<td>Protein (%kcal)</td>
<td>17.86 ± 6.07 (8.83 – 27.23)</td>
<td>17.02 ± 4.78 (7.90 – 22.80)</td>
<td>17.46 ± 5.35 (7.90 – 27.23)</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>370.34 ± 111.05 (235.70 – 586.17)</td>
<td>277.19 ± 76.26 (123.53 – 381.45)</td>
<td>326.51 ± 104.92 (123.53 – 586.17)</td>
</tr>
<tr>
<td>Carbohydrate (%kcal)</td>
<td>49.32 ± 7.61 (37.23 – 61.37)</td>
<td>51.35 ± 8.96 (36.77 – 65.50)</td>
<td>50.28 ± 8.07 (36.77 – 65.50)</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>109.57 ± 25.20 $^a$ (70.63 – 149.20)</td>
<td>67.24 ± 13.91 (47.93 – 82.67)</td>
<td>89.65 ± 29.60 (47.93 – 149.20)</td>
</tr>
<tr>
<td>Fat (%kcal)</td>
<td>32.83 ± 4.11 (28.07 – 39.17)</td>
<td>28.01 ± 6.60 (20.80 – 41.97)</td>
<td>30.56 ± 5.80 (20.80 – 41.97)</td>
</tr>
</tbody>
</table>

$^a$ LOW > HIGH, $p < .05$. 
Exercise Trial

Independent \( t \)-tests were utilized to compare exercise trial time (min; \( p = .236 \)), energy expenditure (kcal; \( p = .326 \)) and percentage of ventilatory threshold (%; \( p = .145 \)), resulting in no significant differences between groups, data is reported as means and standard deviations (\( M \pm SD \); see Table 3).

Table 3

*Exercise Trial Data*

<table>
<thead>
<tr>
<th></th>
<th>LOW Group (n=9)</th>
<th>HIGH Group (n=8)</th>
<th>Combined (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SD (Range)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy Expended (kcal)</td>
<td>612.33 ± 16.97</td>
<td>604.63 ± 13.95</td>
<td>608.71 ± 15.65</td>
</tr>
<tr>
<td></td>
<td>(601.00 – 654.00)</td>
<td>(585.00 – 630.00)</td>
<td>(601.00 – 654.00)</td>
</tr>
<tr>
<td>Exercise Trial Time (min)</td>
<td>57.56 ± 7.59</td>
<td>55.19 ± 6.71</td>
<td>58.00 ± 6.90</td>
</tr>
<tr>
<td></td>
<td>(50.50 – 75.00)</td>
<td>(43.50 – 65.00)</td>
<td>(50.50 – 75.00)</td>
</tr>
<tr>
<td>Ventilatory Threshold (%( VO_2 )max)</td>
<td>55.98 ± 2.33</td>
<td>58.72 ± 4.76</td>
<td>57.27 ± 3.82</td>
</tr>
<tr>
<td></td>
<td>(52.26 – 59.08)</td>
<td>(51.56 – 68.41)</td>
<td>(51.56 – 68.41)</td>
</tr>
</tbody>
</table>

*Note.* No differences between group characteristics.

Measurements of \( VO_2 \) and RER were anticipated to vary over the exercise trial, based on a physiological phenomenon that occurs primarily as the result of changing hormonal concentrations and substrate utilization (Hargreaves, 2006); therefore, each trial was divided into quarters and reported as means and standard deviations (\( M \pm SD \)).
Absolute VO$_2$ (L/min) differed when compared with a 2 x 4 (group x quarter) repeated measures ANOVA across the four quarters of the exercise trial ($F(1, 1.675) = 3.70, p = .046$). However, post hoc tests using Bonferroni’s pairwise comparisons revealed no significant differences between the quarters for VO$_2$ (L/min; $M \pm SD$; 1$^{st}$ = 2.22 ± 0.29; 2$^{nd}$ = 2.28 ± 0.33; 3$^{rd}$ = 2.27 ± 0.35; 4$^{th}$ = 2.31 ± 0.33; see Figure 2).

There were significant differences in the respiratory exchange ratio (RER) when compared with a 2 x 4 (group x quarter) repeated measures ANOVA across the four quarters of the exercise trial ($F(1, 1.883) = 56.59, p < .001$). Bonferroni’s pairwise comparisons revealed significant decreases over time among all the averaged RER quarter values (VCO$_2$/VO$_2$; 1$^{st}$ > 2$^{nd}$, $p < .001$; 1$^{st}$ > 3$^{rd}$, $p < .001$; 1$^{st}$ > 4$^{th}$, $p < .001$; 2$^{nd}$ > 3$^{rd}$, $p = .006$; 2$^{nd}$ > 4$^{th}$, $p < .001$; 3$^{rd}$ > 4$^{th}$, $p = .002$), indicating an increase in fat oxidation over the exercise trial (see Figure 2). No differences were revealed with interactive effects ($F(1,1.883) = 0.667, p = .513$), nor main effects for between groups ($F(1,1.883) = 0.194, p = .666$).
Figure 2. Exercise trial characteristics during the 1st, 2nd, 3rd and 4th quarters. RER = respiratory exchange ratio (VCO₂/VO₂), VO₂ (L/min) = volume of oxygen, a 1st > 2nd, 3rd, 4th, b 2nd > 3rd, 4th, c 3rd > 4th, p < .05.

Plasma Volume

As has previously been illustrated, it was anticipated that plasma volume would fluctuate. Therefore, to allow for determination of the effects of exercise on the blood plasma variables of interest, descriptive data (M ± SD) for blood volume characteristics (hemoglobin, hematocrit and plasma volume percentage change) were investigated (see Table 4) and the concentrations of variables of interest were normalized according to standardized procedures for plasma volume shifts (Dill & Costill, 1974). Blood volume characteristic data were compared using a 2 x 5 (group x time) repeated measures ANOVA for SCREEN, BASELINE, 1hPE, 24hPE and 48hPE).
Hemoglobin values were compared using a 2 x 5 (group x time) repeated measures ANOVA. No interaction was determined ($F(1,4) = 0.907, p < .443$); however, there was a significant main effect over time ($F(1,4) = 10.991, p < .001$). A Bonferroni pairwise comparison revealed SCREEN was significantly greater than BASELINE ($p = .011$), 1hPE ($p = .008$), 24hPE ($p = .010$) and 48hPE ($p = .008$). No between group differences were identified ($F(1,4) = 0.571, p < .462$; see Table 4).

Hematocrit values were compared using a 2 x 5 (group x time) repeated measures ANOVA. No interaction was determined ($F(1,4) = 14.13, p = .257$). However, differences were revealed for main effect over time ($F(1,4) = 4.454, p = .013$). A Bonferroni pairwise comparison revealed SCREEN was significantly greater than 48hPE ($p = .013$). No between group differences were identified ($F(1,4) = 0.839, p = .374$; see Table 4).

The percentage of plasma volume changes from SCREEN – BASELINE, BASELINE – 1hPE, BASELINE – 24hPE and BASELINE – 48hPE were compared using a 2 x 4 (group x time) repeated measures ANOVA. The repeated measures ANOVA determined there were no interactive ($F(1,3) = 1.306, p = 0.284$), main effects over time ($F(1,3) = 0.797, p = 0.502$) nor any main effects between group differences ($F(1,3) = 0.064, p = 0.804$; see Table 4).
Table 4

**Blood and Plasma Volume Descriptive Data**

<table>
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<tr>
<th></th>
<th>SCREEN</th>
<th>BASELINE</th>
<th>1hPE</th>
<th>24hPE</th>
<th>48hPE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Range</td>
<td>Mean ± SD</td>
<td>Range</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Hemoglobin (mg/dl)</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>LOW</td>
<td>15.72 ± 0.09</td>
<td>14.40–17.05</td>
<td>14.86 ± 1.28</td>
<td>12.10–16.70</td>
<td>14.88 ± 1.30</td>
</tr>
<tr>
<td>HIGH</td>
<td>15.79 ± 0.18</td>
<td>14.40–17.05</td>
<td>15.04 ± 0.74</td>
<td>12.10–16.70</td>
<td>15.12 ± 0.67</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>LOW</td>
<td>41.93 ± 0.42</td>
<td>41.83–49.67</td>
<td>44.94 ± 4.23</td>
<td>37.50–53.00</td>
<td>44.52 ± 4.29</td>
</tr>
<tr>
<td>HIGH</td>
<td>46.84 ± 0.74</td>
<td>41.83–49.67</td>
<td>45.04 ± 0.82</td>
<td>37.5–53.00</td>
<td>44.86 ± 1.02</td>
</tr>
<tr>
<td>Combined</td>
<td>45.94 ± 0.59</td>
<td>41.83–49.67</td>
<td>44.99 ± 3.04</td>
<td>37.5–53.00</td>
<td>44.68 ± 3.11</td>
</tr>
<tr>
<td>PV ∆ from SCREEN (%)</td>
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<td></td>
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</tbody>
</table>

*a SCREEN > BASELINE, 1hPE, 24hPE and 48hPE; b SCREEN > 48hPE, 1,24,48hPE = # hours postexercise; Combined -n = 17, NPVA-TG = non-plasma volume adjusted –TG, TGΔ = TG change , TGΔ (%) = percent change from BASELINE as 100%, p < .05.PV ∆ = plasma volume change from SCREEN, n/a = not applicable.

Blood Lipid Concentrations

**Triglycerides**

SCREEN TG concentrations were dually used as a disqualification factor as well as to ensure subject consistency within the grouping concentrations. SCREEN and plasma volume adjusted (PVA) -BASELINE TG were compared with a 2 x 2 repeated measures ANOVA and reported below as means and standard deviations (M ± SD). No interactive effects (F(1,1) = 3.45, p < .083) and no main effect differences over time (F
(1,1) = 0.027, \( p = .871 \) were determined. However, between groups main effects revealed an overall greater TG in HIGH versus LOW \( (F(1,1) = 131.55, \ p < .001) \).

### Table 5

**Within Subjects Triglyceride Control (mg/dl)**

<table>
<thead>
<tr>
<th></th>
<th>LOW Group (n=9)</th>
<th>HIGH Group (n=8)</th>
<th>Combined (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td></td>
<td>(Range)</td>
<td>(Range)</td>
<td>(Range)</td>
</tr>
<tr>
<td>SCREEN TG</td>
<td>48.27 ± 12.46</td>
<td>151.34 ± 28.90</td>
<td>96.77 ± 57.06</td>
</tr>
<tr>
<td></td>
<td>(31.00 – 64.80)</td>
<td>(108.20 – 183.00)</td>
<td>(31.00 – 183.00)</td>
</tr>
<tr>
<td>PVA-BASELINE TG</td>
<td>43.33 ± 10.31</td>
<td>154.97 ± 23.25</td>
<td>96.94 ± 61.40</td>
</tr>
<tr>
<td></td>
<td>(25.45 – 59.96)</td>
<td>(118.09 – 187.97)</td>
<td>(25.45 – 187.97)</td>
</tr>
</tbody>
</table>

\(^{a}\) HIGH > LOW, BASELINE TG is plasma volume adjusted (PVA) based on SCREEN TG data, \( p < .05 \).

In order to compare both the actual en vivo effect of exercise to the effect of the plasma volume adjustment on blood concentrations, the nonplasma volume adjusted TG (NPVA-TG; BASELINE, 1hPE, 24hPE and 48hPE) were analyzed with a 2 x 4 (group x time) repeated measures ANOVA and reported as means and standard deviations \( (M ± SD) \) or standard error \( (M ± SE) \), where appropriate. No interactive effects were determined \( (F(1,3) = 3.574, \ p = .062) \) nor any main effects over time \( (F(1,3) = 3.616, \ p = .061) \). However, between groups effects revealed an overall greater NPVA-TG in HIGH versus LOW \( (F(1,3) =144.53, \ p < .001) \; \text{see Table 6} \).

To determine the effect of exercise on blood lipid variables the concentrations were adjusted to reflect plasma volume shifts based on the SCREEN sample. Plasma
volume adjusted TG were compared with a 2 x 5 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. Significant interaction between groups was identified ($F(1, 4) = 3.383, p = .041$). A one-way repeated measures ANOVA was run for each group, however, it revealed no significant differences over time for either group (LOW, $F(0, 4) = 2.075, p = .107$; HIGH, $F(1, 4) = 3.657, p = .065$). Bonferroni’s pairwise comparison was used in order to reveal significant TG reductions over time (main effect; $F(1, 4) = 3.944, p = .025$) and revealed that 24hPE was significantly less than SCREEEN ($p = .028$) and BASELINE ($p = .032$). Lastly, main effects for between groups ($F(1, 4) = 107.22, p < .000$) revealed an on average, overall less TG in the LOW ($M = 42.57, SE = 6.66$) versus the HIGH ($M = 141.04, SE = 7.06$; see Table 6 and Figure 3).

![Figure 3](image)

*Figure 3*. Plasma volume adjusted triglyceride concentrations over time. *a* Combined 24hPE < Combined BASELINE and Combined SCREEN, $p < .05$. 
Absolute TG changes in mg/dl (TGΔmg/dl) from BASELINE – 1hPE, BASELINE – 24hPE and BASELINE – 48hPE were analyzed with a 2 x 3 (group x time) repeated measures ANOVA and are reported as means and standard deviations (M ± SD) or standard error (M ± SE), where appropriate. No interactive effects were determined (F(1,2) = 2.189, p = .148), nor any main effects over time (F(1,1.389) = 1.114, p = .326). However, main effects between groups revealed an overall greater absolute (mg/dl) TG change in HIGH versus LOW (F(1,1.389) = 13.787, p = .002; see Table 6).

Relative TG changes as % (TGΔ%) from BASELINE – 1hPE, BASELINE – 24hPE and BASELINE – 48hPE were analyzed with a 2 x 3 (group x time) repeated measures ANOVA and reported as means and standard deviations (M ± SD) or standard error (M ± SE), where appropriate. No interactive effects (F(1,2) = 2.779, p = .078), main effects over time (F(1,2) = 2.097, p = .140), nor main effects between groups (F(1,2) = 3.506, p = .081) revealed any significant differences (see Table 6).

Total Cholesterol

Nonplasma volume adjusted TChol (NPVA-TChol) over time (BASELINE, 1hPE, 24hPE and 48hPE) were analyzed with a 2 x 4 (group x time) repeated measures ANOVA and reported as means and standard deviations (M ± SD) or standard error (M ± SE), where appropriate. No interactive effects were determined (F(1,3) = 2.801, p = .051), nor any main effects over time (F(1,3) = 0.157, p = .924). However, main between groups effects revealed an overall greater NPVA-TChol in HIGH versus LOW (F(1,3) = 20.337, p < .001; see Table 7).
Table 6

**Triglyceride Data**

<table>
<thead>
<tr>
<th></th>
<th>BASELINE</th>
<th>1hPE</th>
<th>24hPE</th>
<th>48hPE</th>
<th>Mean Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SE</td>
</tr>
<tr>
<td>NPVA-TG (mg/dl)</td>
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<td></td>
<td></td>
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<tr>
<td>LOW</td>
<td>40.64 ± 12.56</td>
<td>43.28 ± 6.86</td>
<td>34.92 ± 7.43</td>
<td>36.92 ± 8.09</td>
<td>38.94 ± 5.12</td>
</tr>
<tr>
<td>HIGH</td>
<td>140.90 ± 19.88</td>
<td>116.89 ± 31.07</td>
<td>113.19 ± 29.32</td>
<td>143.34 ± 37.52</td>
<td>128.58 ± 5.43</td>
</tr>
<tr>
<td>Combined</td>
<td>87.82 ± 53.97</td>
<td>77.92 ± 43.36</td>
<td>71.75 ± 45.01</td>
<td>87.00 ± 60.39</td>
<td>83.76 ± 3.73</td>
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<td>TG(mg/dl)</td>
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<td>LOW</td>
<td>43.33 ± 10.31</td>
<td>47.47 ± 9.95</td>
<td>38.08 ± 5.88</td>
<td>41.41 ± 8.05</td>
<td>42.57 ± 6.66</td>
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<tr>
<td>HIGH</td>
<td>157.24 ± 25.38</td>
<td>127.93 ± 28.37</td>
<td>132.71 ± 38.56</td>
<td>146.29 ± 39.86</td>
<td>141.04 ± 7.06</td>
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<tr>
<td>Combined</td>
<td>96.94 ± 61.40</td>
<td>85.33 ± 45.99</td>
<td>82.61 ± 55.12</td>
<td>90.77 ± 60.32</td>
<td>91.81 ± 4.85</td>
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<td>TGΔ (mg/dl)</td>
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<td></td>
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<tr>
<td>LOW</td>
<td>n/a</td>
<td>-4.14 ± 10.26</td>
<td>-5.25 ± 8.44</td>
<td>-1.92 ± 13.77</td>
<td>-1.01 ± 3.81</td>
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<td>HIGH</td>
<td>n/a</td>
<td>-29.31 ± 27.11</td>
<td>-24.54 ± 24.00</td>
<td>-10.96 ± 18.81</td>
<td>-21.60 ± 4.04</td>
</tr>
<tr>
<td>Combined</td>
<td>n/a</td>
<td>-11.60 ± 25.89</td>
<td>-14.32 ± 19.65</td>
<td>-6.17 ± 16.47</td>
<td>-11.30 ± 2.77</td>
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<tr>
<td>TGΔ (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOW</td>
<td>n/a</td>
<td>12.72 ± 24.64</td>
<td>-8.72 ± 20.84</td>
<td>1.42 ± 33.08</td>
<td>1.81 ± 5.90</td>
</tr>
<tr>
<td>HIGH</td>
<td>n/a</td>
<td>-18.10 ± 15.24</td>
<td>-16.55 ± 16.30</td>
<td>-8.21 ± 12.98</td>
<td>-14.29 ± 6.26</td>
</tr>
<tr>
<td>Combined</td>
<td>n/a</td>
<td>-1.78 ± 25.62</td>
<td>-12.40 ± 18.70</td>
<td>-3.11 ± 25.16</td>
<td>-6.24 ± 4.30</td>
</tr>
</tbody>
</table>

a 24hPE < BASELINE; b HIGH > LOW on average for NPVA-TG, TG and Absolute TGΔ (mg/dl); 1, 24, 48hPE = the # hours postexercise, Combined (y-axis) = averaged across for all groups (LOW and HIGH), Mean Estimate (x-axis) = average across all time points (BASELINE - 48hPE), NPVA-TG = non-plasma volume adjusted-TG, TGΔ = TG change, TGΔ (%) = percent change from BASELINE as 100%, p < .05.

Plasma volume adjusted TChol over time (BASELINE, 1hPE, 24hPE and 48hPE) were analyzed with a 2 x 4 (group x time) repeated measures ANOVA and reported as means and standard deviations (M ± SD) or standard error (M ± SE), where appropriate. No interactive effects were determined (F(1, 3) = 1.5149.45, p = .224), nor any main effects over time (F(1, 3) = 0.169, p = .917). However, between subjects effects revealed an overall greater TChol in HIGH versus LOW (F(1, 3) = 13.55, p = .002; see Table 7).
Absolute (mg/dl) change of TChol from BASELINE – 1hPE, BASELINE – 24hPE and BASELINE – 48hPE were analyzed with a 2 x 3 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. No interactive effects were determined ($F(1, 2) = 01.341, p = .277$). No main effects over time ($F(1, 2) = 0.252, p = .779$), nor any main between groups effects ($F(1, 2) = 1.859, p = .193$; see Table 7).

Relative TChol changes (%) from BASELINE – 1hPE, BASELINE – 24hPE and BASELINE – 48hPE were analyzed with a 2 x 3 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. No interactive effects ($F(1,2) = 1.284, p = .292$), main effects over time ($F(1,2) = 0.237, p = .790$), nor main effects between groups ($F(1,2) = 0.971, p = .340$) were determined (see Table 7).

**High Density Lipoprotein-Cholesterol**

Nonplasma volume adjusted HDL-C (NPVA-HDL-C) over time (BASELINE, 1hPE, 24hPE and 48hPE) were analyzed with a 2 x 4 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. No interactive effects ($F(1,3) = 1.309, p = .285$), no main effects over time ($F(1,3) = 0.142, p = .857$), nor between groups main effects ($F(1,3) = 0.168, p = .688$) were determined (see Table 8).
Table 7

Total Cholesterol Data

<table>
<thead>
<tr>
<th></th>
<th>BASELINE Mean ± SD</th>
<th>1hPE Mean ± SD</th>
<th>24hPE Mean ± SD</th>
<th>48hPE Mean ± SD</th>
<th>Mean Estimate Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>NPVA-TChol (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOW</td>
<td>145.99 ± 18.50</td>
<td>143.32 ± 22.30</td>
<td>138.18 ± 20.44</td>
<td>135.99 ± 18.80</td>
<td>140.87 ± 10.34</td>
</tr>
<tr>
<td>HIGH</td>
<td>205.61 ± 33.57</td>
<td>207.54 ± 43.22</td>
<td>210.01 ± 40.60</td>
<td>212.24 ± 48.04</td>
<td>208.85 ± 10.97</td>
</tr>
<tr>
<td>Combined</td>
<td>174.05 ± 40.07</td>
<td>173.54 ± 46.45</td>
<td>171.99 ± 47.91</td>
<td>171.87 ± 52.21</td>
<td>174.86 ± 7.54</td>
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<tr>
<td>TChol (mg/dl)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOW</td>
<td>159.57 ± 30.98</td>
<td>156.61 ± 29.48</td>
<td>152.90 ± 32.28</td>
<td>158.16 ± 28.93</td>
<td>156.81 ± 14.25</td>
</tr>
<tr>
<td>HIGH</td>
<td>230.59 ± 49.72</td>
<td>231.05 ± 57.90</td>
<td>237.32 ± 56.23</td>
<td>234.15 ± 55.00</td>
<td>233.28 ± 15.12</td>
</tr>
<tr>
<td>Combined</td>
<td>192.99 ± 53.82</td>
<td>191.64 ± 58.03</td>
<td>192.63 ± 61.57</td>
<td>193.92 ± 57.19</td>
<td>195.04 ± 10.39</td>
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<td>TCholΔ (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HIGH</td>
<td>0.45 ± 13.80</td>
<td>6.73 ± 10.85</td>
<td>3.56 ± 10.73</td>
<td>3.58 ± 3.88</td>
<td>3.58 ± 3.88</td>
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<tr>
<td>Combined</td>
<td>-1.36 ± 13.41</td>
<td>-0.36 ± 15.57</td>
<td>0.93 ± 12.01</td>
<td>-0.051 ± 2.66</td>
<td>-0.051 ± 2.66</td>
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<tr>
<td>TCholΔ (%)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOW</td>
<td>-1.38 ± 8.92</td>
<td>-3.79 ± 10.89</td>
<td>-0.47 ± 8.77</td>
<td>-0.33 ± 1.57</td>
<td>-0.33 ± 1.57</td>
</tr>
<tr>
<td>HIGH</td>
<td>0.27 ± 6.16</td>
<td>2.70 ± 4.12</td>
<td>1.24 ± 4.80</td>
<td>-1.88 ± 2.16</td>
<td>-1.88 ± 2.16</td>
</tr>
<tr>
<td>Combined</td>
<td>-0.86 ± 7.53</td>
<td>-0.74 ± 8.82</td>
<td>0.34 ± 7.02</td>
<td>1.22 ± 2.29</td>
<td>1.22 ± 2.29</td>
</tr>
</tbody>
</table>

aHIGH > LOW on average for NPVA-TChol and TChol; 1, 24, 48hPE = the # hours postexercise, Combined (y-axis) = averaged across for all groups (LOW and HIGH), Mean Estimate (x-axis) = average across all time points (BASELINE-48hPE), NPVA-TChol = non-plasma volume adjusted-TChol, TCholΔ = TChol change, TGΔ (%) = percent change from BASELINE as 100%, p < .05.

Plasma volume adjusted HDL-C over time (BASELINE, 1hPE, 24hPE and 48hPE) were analyzed with a 2 x 4 (group x time) repeated measures ANOVA and reported as means and standard deviations (M ± SD) or standard error (M ± SE), where appropriate. No interactive effects (F(1,3) = 2.492, p = .072), no main effect over time (F(1,3) = 0.747, p = .530), nor main between group effects (F(1,3) = 0.110, p = .745) were determined (see Table 8).
Plasma volume adjusted HDL-C absolute change (BASELINE – 1hPE, BASELINE – 24hPE and BASELINE – 48hPE) were analyzed with a 2 x 3 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. Significant interaction was present for the absolute (mg/dl) HDL-C change ($F(1, 2) = 3.975, p = .029$). A one-way repeated measures ANOVA was performed for each group (LOW, $F(0,2) = 4.488, p = .028$; HIGH, $F(0,2) = 0.895, p = .431$) and indicated significantly greater HDL-C increases from BASELINE – 24hPE than at BASELINE – 1hPE, for the LOW group only ($p = .005$; see Table 8).

Relative HDL-C change (BASELINE – 1hPE, BASELINE – 24hPE and BASELINE – 48hPE) were analyzed with a 2 x 3 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. No interactive effects ($F(1,2) = 3.214, p = .054$), no main effects over time ($F(1,2) = 2.457, p = .103$), nor main between groups effects ($F(1,2) = 0.736, p = 0.405$) were determined (see Table 8).

Low Density Lipoprotein-Cholesterol

Nonplasma volume adjusted LDL-C (NPVA-LDL-C) over time (BASELINE, 1hPE, 24hPE and 48hPE) were analyzed with a 2 x 4 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. Significant interaction ($F(1,3) = 3.655, p = .019$) was identified. A one-way repeated measures ANOVA were performed for each group (LOW, $F(3) = 3.023, p = .049$; HIGH, $F(3) = 1.531, p = .236$) and revealed there was a significant
difference over time in the LOW group; however, a Bonferroni pairwise comparisons
indicated no significant differences. Main effects over time indicated significant
differences \((F(1,3) = 14.684, p = .002)\). A Bonferroni pairwise comparison revealed an
overall greater NPVA-LDL-C for the HIGH versus the LOW \((F(3,1 = 14.68, p = .002;\)
see Table 9). LDL-C was calculated via Friedewald Equation (Friedewald, Levy, &

Table 8

*High-Density Lipoprotein-Cholesterol Data*

<table>
<thead>
<tr>
<th></th>
<th>BASELINE Mean ± SD</th>
<th>1hPE Mean ± SD</th>
<th>24hPE Mean ± SD</th>
<th>48hPE Mean ± SD</th>
<th>Mean Estimate Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>NPVA-HDL-C (mg/dl)</td>
<td></td>
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</tr>
<tr>
<td>LOW</td>
<td>39.54 ± 12.66</td>
<td>38.88 ± 9.85</td>
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<td>40.86 ± 8.52</td>
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<td>HIGH</td>
<td>38.52 ± 9.84</td>
<td>39.08 ± 9.69</td>
<td>37.04 ± 9.50</td>
<td>38.35 ± 9.90</td>
<td>38.29 ± 3.37</td>
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<td>Combined</td>
<td>39.06 ± 11.08</td>
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<td>39.39 ± 9.55</td>
<td>39.77 ± 8.98</td>
<td>39.24 ± 2.32</td>
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<td>HDL-C (mg/dl)</td>
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<tr>
<td>LOW</td>
<td>42.75 ± 13.34</td>
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<td>HIGH</td>
<td>43.11 ± 12.35</td>
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<td>41.32 ± 9.30</td>
<td>42.40 ± 10.58</td>
<td>42.59 ± 3.80</td>
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<tr>
<td>Combined</td>
<td>42.92 ± 12.48</td>
<td>42.84 ± 10.72</td>
<td>43.50 ± 10.45</td>
<td>44.77 ± 10.32</td>
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<td>4.12 ± 6.51</td>
<td>0.69 ± 1.35</td>
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<td>n/a</td>
<td>0.42 ± 4.22</td>
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<td>-0.71 ± 8.56</td>
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<td>Combined</td>
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<td>-0.08 ± 4.67</td>
<td>0.58 ± 6.20</td>
<td>1.85 ± 7.71</td>
<td>-0.68 ± 1.97</td>
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<td>HDL-CΔ (%)</td>
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<tr>
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<td>0.80 ± 11.95</td>
<td>8.01 ± 11.18</td>
<td>12.44 ± 14.54</td>
<td>4.06 ± 3.53</td>
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<td>1.51 ± 21.54</td>
<td>7.08 ± 4.84</td>
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<tr>
<td>Combined</td>
<td>n/a</td>
<td>1.36 ± 11.36</td>
<td>4.04 ± 16.61</td>
<td>7.30 ± 18.45</td>
<td>1.03 ± 5.14</td>
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</table>

^24hPE > 1hPE; 1, 24, 48hPE = the # hours postexercise, Combined (y-axis) = averaged across all
groups (LOW and HIGH), Mean Estimate (x-axis) = average across all time points (BASELINE-48hPE),
NPVA-HDL-C = non-plasma volume adjusted-HDL-C, HDL-CΔ = HDL-C change, HDL-CΔ (%) =
percent change from BASELINE as 100%, \(p < .05\).
Plasma volume adjusted, estimated LDL-C over time (BASELINE, 1hPE, 24hPE and 48hPE) were analyzed with a 2 x 4 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. Significant interaction was identified ($F(1,3) = 3.257, p = .030$) and a one-way repeated measures ANOVA was performed for each group and revealed no significant interactive effects (LOW, $F(0,3) = 1.027, p = .398$; HIGH, $F(0,3) = 2.386, p = .098$). No main effects over time were determined ($F(1,3) = 0.195, p = .899$). However, main effects between groups did reveal an overall greater LDL-C in the HIGH versus the LOW ($F(1,3) = 10.5, p = .005$; see Table 9).

The absolute (mg/dl) change of LDL-C from BASELINE – 1hPE, BASELINE – 24hPE and BASELINE – 48hPE were analyzed with a 2 x 3 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. No interactive effects ($F(1,2) = 2.175, p = .131$), nor main effects over time ($F(1,2) = 0.183, p = .833$) were determined to be different. However, main effects between groups revealed an overall greater change in the HIGH versus the LOW ($F(1,2) = 4.617, p = .048$; see Table 9).

Relative LDL-C changes (%) from BASELINE – 1hPE, BASELINE – 24hPE and BASELINE – 48hPE were analyzed with a 2 x 3 (group x time) repeated measures ANOVA and reported as means and standard deviations ($M \pm SD$) or standard error ($M \pm SE$), where appropriate. No interactive effects ($F(1,2) = 2.128, p = .137$), main effects over time ($F(1,2) = 0.058, p = .944$), nor main effects between groups ($F(1,2) = 2.405, p = .142$) were identified (see Table 9).
Table 9

Low-Density Lipoprotein-Cholesterol Data

<table>
<thead>
<tr>
<th></th>
<th>BASELINE Mean ± SD</th>
<th>1hPE Mean ± SD</th>
<th>24hPE Mean ± SD</th>
<th>48hPE Mean ± SD</th>
<th>Mean Estimate Mean ± SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>NPVA-LDL-C (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOW</td>
<td>98.32 ± 19.49</td>
<td>95.79 ± 21.60</td>
<td>89.72 ± 23.14</td>
<td>87.74 ± 21.22</td>
<td>92.89 ± 9.30</td>
</tr>
<tr>
<td>HIGH</td>
<td>138.91 ± 26.61</td>
<td>145.09 ± 38.98</td>
<td>150.34 ± 36.38</td>
<td>145.04 ± 40.29</td>
<td>144.84 ± 9.87</td>
</tr>
<tr>
<td>Combined</td>
<td>117.42 ± 30.59</td>
<td>118.99 ± 39.26</td>
<td>118.24 ± 42.66</td>
<td>114.71 ± 42.48</td>
<td>118.87 ± 6.78</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOW</td>
<td>108.16 ± 30.65</td>
<td>104.89 ± 27.05</td>
<td>99.84 ± 31.34</td>
<td>103.01 ± 28.17</td>
<td>103.98 ± 12.39</td>
</tr>
<tr>
<td>HIGH</td>
<td>156.03 ± 38.33</td>
<td>161.93 ± 50.90</td>
<td>169.47 ± 51.14</td>
<td>162.49 ± 43.44</td>
<td>162.48 ± 13.14</td>
</tr>
<tr>
<td>Combined</td>
<td>130.68 ± 41.47</td>
<td>131.73 ± 48.59</td>
<td>132.61 ± 54.02</td>
<td>131.00 ± 46.46</td>
<td>133.23 ± 9.03</td>
</tr>
<tr>
<td>LDL-CΔ (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOW</td>
<td>n/a</td>
<td>-3.27 ± 17.83</td>
<td>-8.32 ± 18.32</td>
<td>-5.15 ± 14.26</td>
<td>-5.58 ± 4.53</td>
</tr>
<tr>
<td>HIGH</td>
<td>n/a</td>
<td>5.90 ± 16.53</td>
<td>13.44 ± 15.74</td>
<td>6.46 ± 7.57</td>
<td>8.60 ± 4.80</td>
</tr>
<tr>
<td>Combined</td>
<td>n/a</td>
<td>1.05 ± 17.34</td>
<td>1.92 ± 20.04</td>
<td>0.31 ± 12.74</td>
<td>1.51 ± 3.30</td>
</tr>
<tr>
<td>LDL-CΔ (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LOW</td>
<td>n/a</td>
<td>-0.96 ± 17.32</td>
<td>-6.61 ± 16.74</td>
<td>-3.79 ± 13.38</td>
<td>0.44 ± 2.73</td>
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<tr>
<td>HIGH</td>
<td>n/a</td>
<td>2.65 ± 9.57</td>
<td>7.63 ± 8.57</td>
<td>3.73 ± 4.86</td>
<td>4.76 ± 3.97</td>
</tr>
<tr>
<td>Combined</td>
<td>n/a</td>
<td>0.74 ± 13.91</td>
<td>0.09 ± 15.03</td>
<td>-0.25 ± 10.71</td>
<td>-3.79 ± 3.74</td>
</tr>
</tbody>
</table>

*a HIGH > LOW on average for NPVA-LDL-C and LDL-C; 1, 24, 48hPE = the # hours postexercise, Combined (y-axis) = averaged across for all groups (LOW and HIGH), Mean Estimate (x-axis) = average across all time points (BASELINE-48hPE), NPVA-LDL-C = non-plasma volume adjusted -LDL-C, LDL-CΔ = LDL-C change, LDL-CΔ (%) = percent change from BASELINE as 100%; p < .05. LDL-C was calculated via Friedewald Equation (Friedewald, Levy, & Fredrickson, 1972).
CHAPTER 5: DISCUSSION

The main finding of this investigation was that expending $608.71 \pm 15.65$ kcal in a group of average fitness men ($n = 17; 47.37 \pm 3.55$ ml/kg/min) resulted in significant reductions in triglycerides (TG) ($14.32 \pm 19.65$ mg/dl) 24hPE regardless of initial TG concentrations. Mean group comparisons collapsed over all time points revealed significantly less absolute TG reductions ($\Delta$mg/dl) for the LOW group ($-1.01 \pm 3.801$ mg/dl) versus the HIGH group ($-21.60 \pm 4.04$ mg/dl). However, when these between groups effects were translated into relative change ($\%\Delta$), there were large differences among the groups, but not significant differences in TG$\%\Delta$ as the result of exercise for either group (LOW = $1.81 \pm 5.9\%$ and HIGH = $-14.29 \pm 6.26\%$ at 24hPE, $p = .054$) through 48hPE.

First Hypothesis

The results of this investigation support the first hypothesis: An acute bout of 600kcal did reduce blood TG concentrations at 24hPE. This postexercise TG reduction is in line with the majority of published work on changes in blood lipid following a single bout of exercise (Bounds et al., 2000; Crouse et al., 1995 & 1997; Cullinane et al., 1982; Dufaux et al., 1986; Ferguson et al., 1998; Foger et al., 1994; Frey et al. 1993; Grandjean et al., 2000; Kantor et al., 1984; Lamon-Fava et al., 1989; Park & Ransone, 2003; Thompson, Cullinane, Henderson, & Herbert, 1980; Sady et al., 1984), which demonstrate significant TG reductions of 5-38mg/dl (4-37%) with a consistent apex occurring 24hPE (Thompson & Rader, 2001).
Previous published investigations that demonstrate reductions in TG without statistical significance (Angelopoulos, 1993; Gordon, 1994; Kantor, 1987) have failed to control for intensity (percentage of heart rate max; Kantor, 1987) or total energy expenditure (overall exercise time; Angelopoulos, 1993) as possible explanations for their lack of findings. In fact, Gordon et al. (1994) were the first to report a “real-time” respiratory gas measurement collection method to estimate energy expenditure even though the investigation did not report TG reductions. However, although Gordon et al. (1994) failed to find a significant TG reduction with acute exercise it was no doubt, in part, due to large standard deviations among the TG data. It can be speculated that the multiple exercise trial paradigm exacerbating the inherent variability in day-to-day TG. The current investigation’s methods were tailored specifically to help account for the inherent variability of day-to-day TG by using a single trial paradigm, and thus resulting in generally smaller standard deviations among the TG data. Research published “post-Gordon et al. (1994)” in this area of investigation, utilize “real-time” respiratory gas measurements to better administer the exercise treatment.

Second Hypothesis

This investigation supports the second hypothesis: An acute bout of 600kcal induced greater absolute (Δmg/dl) TG change in the HIGH versus the LOW when collapsed over all time points (TGΔmg/dl, LOW = -1.01 ± 3.81 and HIGH = -21.6 ± 4.04; p = .002). Previous research coincidentally has demonstrated subjects with higher initial TG concentrations are associated with greater postexercise decreases in terms of absolute change (Δmg/dl) (Crouse et al., 1995; Ferguson et al., 1998; Foger et al., 1994;
Grandjean et al., 2000), but this was not their stated intent. This investigation was unique because of the group defining initial TG concentrations, and therefore, directly demonstrated the significant influence of initial TG concentrations over the postexercise TG reduction (TGΔmg/dl); however, relatively (TGΔ%) the postexercise TG changes were large but not significantly different (TGΔ%, LOW = 1.81 ± 5.90, HIGH= -14.29 ± 6.26; \(p = .054\)).

**Third Hypothesis**

Therefore, results of the current investigation also support the third hypothesis: An acute bout of 600kcal did not result in different relative (%) TG change (TGΔ%) between groups with different initial TG (TGΔ% on average for the LOW = 1.81 ± 5.90 and HIGH = -14.29 ± 6.26; \(p = .054\)). These results support previous works demonstrating relative TG changes (Δ%) between subjects with elevated and normal TG concentrations should be fairly equal (Crouse et al.1995; Visich et al., 1996). For instance, Visich et al. (1996) used trained subjects with below average initial TG concentrations (67mg/dl) and demonstrated a 9 mg/dl (13.4%) reduction 24hPE; however, Crouse et al.’s (1995) above average initial TG concentrations (177mg/dl) were reduced 18mg/dl (10.5%) after a similar 500kcal exercise bout. Thus, in general absolute TG change (TGΔmg/dl; 9 vs. 18mg/dl) was much greater in those with elevated TG, while percentage differences were much closer in comparison (TGΔ%; 13.4 vs. 10.5%; Visich et al., 1996; Crouse et al., 1995, respectively).

The results of this investigation aligned with the current, but to this point, unsubstantiated view that the initial TG concentration influences the post- “acute
exercise” blood lipid response (Durstine et al., 2002; Moffat & Stamford, 2006). Since the aforementioned Gordon et al., 1994 publication (post-Gordon, 1994; see Appendix B), nearly all reports have illustrated TG reduction following acute exercise when the initial TG concentrations were at least 99mg/dl (Bounds et al., 2000; Crouse et al., 1995, 1997; Ferguson et al., 1998; Grandjean et al., 2000; Park & Ransone, 2003); however, when baseline TGs are reported below 99mg/dl, statistically significant TG reductions are less common (Visich et al., 1996; see Appendix B). Furthermore, while these conclusions support the current views within acute exercise research, it has not been directly tested until now.

Additionally, these results support previous investigations that followed TG at 1hPE (Crouse et al., 1997; Cullinane et al., 1982; Davis et al., 1992; Frey et al., 1993; Gordon et al., 1994; Thompson et al., 1980; Visich et al., 1996) and 48hPE (Angelopoulos et al., 1993; Davis et al., 1992; Dufaux et al. 1986; Kantor et al., 1984; Thompson et al., 1980) without demonstrating significant change. However, more recent acute exercise investigations that collected blood at or near the 48hPE, demonstrated significant TG reductions of 11-28mg/dl (6-25%; Bounds et al., 2000; Crouse et al., 1995, 1997; Ferguson et al., 1998; Foger et al., 1994; Granjean et al., 2000). Despite these conflicting results, there are viable explanations.

The results of the current investigation demonstrate groups with differing initial TG concentrations (LOW = 43.33, HIGH = 157.24) respond differently to a similar exercise up to 24hPE, depending on the energy expenditure. Those conflicting investigations that demonstrated a significant TG reduction effect over 48h (Bounds et
al., 2000; Crouse et al., 1995, 1997; Grandjean et al., 2000) exhibited initial TG concentrations ranging from 113-177mg/dl, much greater than the current investigation’s combined initial TG concentration of 96.94 ± 61.40mg/dl. Thus, the main idea of the current investigation, to create two separate groups defined by higher and lower TG concentrations, resulted in attenuating the postexercise TG reduction effect through 48h. Additionally, the large standard deviations created by the separation of LOW and HIGH TG groups did not aid in sustaining statistical significance through 48hPE.

Fat Metabolism

The major source of FA oxidation during exercise is derived from the release of FAs from subcutaneous adipose tissue via the stress induced hormone sensitive lipase (HSL) cascade (Hargreaves & Spriet, 2006). These released peripheral FA combine with localized intramuscular TG (IMTG) to provide a nonstop lipid fuel source for beta-oxidation (Horowitz, 2003). These readily available lipid fuels limit the need for lipoprotein-bound TG to contribute directly to muscle exercise; however, the hours after exercise (4-48h), peripheral FA (adipose and intramuscular) must be replenished. Previously published literature has established a delayed activation of endothelial-bound lipoprotein lipase activity (LPLa) an enzyme needed to hydrolyze lipoprotein TG. Zhang et al. (2002) demonstrated significantly greater LPLa at 24hPE versus baseline, 4hPE, 8hPE and 12hPE after an exercise routine similar to the current methods (treadmill exercise for 1h at 60%VO\(_{2}\)max). Thus, greater physical activity levels may be attributed to increased LPL hydrolysis ranging from 4-48hPE (Horowitz, 2003) and peaking at 24hPE (Zhang et al., 2002).
In light of the delay in LPLa following an acute bout of exercise, it has been suggested that lipoprotein-bound TG contributes only a small amount of fatty acid substrate for working muscle. It is believed that these lipoprotein-bound TG sources are more likely replenishing peripheral (adipose or intramuscular) TG sources that were the primary fat source during the bout and the hours after exercise (Horowitz, 2003). Recent FA availability research by Helge et al. (2001) demonstrated after adaption to a fat-rich diet, exercise induced an increased FA oxidation rate with a greater contribution from lipoprotein-bound (serum) TG. Thus, a fat-rich diet may contribute to the remodeling of the TG associated lipoproteins, in this case, implicating the very low density lipoprotein (VLDL) as the altered TG distributing lipoprotein, and thus, an important fuel source during fasted exercise.

Acute Exercise

This investigation contributes to an established body of literature, which demonstrates that the acute exercise TG-response appears to be directly associated with the amount of energy expenditure (Bounds et al., 2000; Crouse et al., 1995; Ferguson et al., 1998; Grandjean et al., 2000; Park & Ransone, 2003; Visich et al., 1996). The training status of subjects stands out from the rest of the manipulative variables because of its strong influence over the effects of an acute bout of exercise to expend X-amount of kcal. In the current investigation all men tested for statistically similar fitness (47.37 ± 3.55ml/kg/min) and body composition (19.14 ± 3.49% body fat) as well as similar energy expenditures (608.71 ± 15.65kcal) during the exercise trial. Other investigations in agreement with these results differ in the training status of subjects (Bounds et al., 2000
Crouse et al., 1995; Cullinane et al., 1982; Dufaux et al., 1986; Ferguson et al., 1998; Fogel et al., 1994; Grandjean et al., 2000; Kantor et al., 1984; Lamon-Fava et al., 1989; Park & Ransone, 2003; Sady et al., 1984; Thompson et al., 1980; Visich et al., 1996); however, these differences are accounted for in the proportional amount of energy expenditure. For instance, for those investigations that defined the amount of kcal expended (post-Gordon et al., 1994, see Appendix B), a trained sample of subjects (50.2, 56.2 and 56.4 ml/kg/min) needed at least 800 kcal of energy expenditure in order to induce significant TG reductions (Bounds et al., 2000; Ferguson et al., 1998; Visich et al., 1996, respectively). On the other hand, a sedentary/untrained population (31.3, 33.4 and 44.9 ml/kg/min) only needed 350-500 kcal in order to induce significant TG reductions (Crouse et al., 1995; Grandjean et al., 2000; Park & Ransone, 2003, respectively).

Therefore, in the current investigation, a population of average fitness (47 ± 3.55 ml/kg/min) subjects demonstrated significant 24hPE TG reductions after an average amount of acute exercise-energy expenditure (608.71 ± 15.65 kcal); however, exceptions unsuccessful at inducing an acute exercise TG reduction, with equivalent fitness and energy expenditures (37 ml/kg/min and 450 kcal), must also be considered (Campbell, Moffat, & Kushnick, 2011). Perhaps this exception to the norm, was the result of the intermittent-exercise intervention, forcing the body to use a greater proportion of its anaerobic stores than it would during a continuous, primarily fat burning, exercise bout.

Previous publications have demonstrated postexercise TG reductions can be independent (Crouse et al. 1995; Davis et al., 1992) as well as dependent (Gordon et al., 1994) upon the absolute intensity (%VO2max) at which exercise is completed. These
conflicting results stem primarily from two influential acute exercise characteristics already discussed, the aerobic fitness of subjects and the adequate and equal energy expenditures. However, more recently, Park and Ransone (2003) suggested intensity does have some influence over the acute exercise response, when completed at the subject’s relative lactate threshold. Park and Ransone’s (2003) unique methodology, of matching the subject-specific relative lactate threshold with the exercise trial intensity, resulted in satisfactory lipid oxidation and therefore, significant postexercise TG reductions. The results of the current investigation draw similar comparisons with Park and Ransone (2003) because, on average, the relative ventilatory thresholds (55.88 ± 7.6VO₂max) collected during the VO₂max tests are similar to the intensity at which the exercise trails were completed (57.27 ± 3.82 %VO₂max). Thus, these results support previous intensity-dependent findings (Gordon et al., 1994; Park & Ransone, 2003) and suggest if the exercise trial intensity was significantly below or above the average ventilatory threshold for this group of subjects, it would have resulted in a reduced postexercise blood lipid response. Therefore, with adequate energy expenditure, exercise should be done at or near a subject’s ventilatory threshold in order to adequately stimulate lipid oxidation from various lipid sources, including adipose tissue, lipoprotein-bound, and intramuscular associated lipids (Park & Ransone, 2003).

According to the results, the initial TG concentrations were the only screened distinguishable differences among the LOW and HIGH groups; however, after batch analysis of all SCREEN samples, other initial blood lipid variables became significant. Not surprisingly, the HIGH group’s initial non-plasma volume adjusted (NPVA)-TChol
(205.61 ± 33.57mg/dl) and initial NPVA-LDL-C (138.91 ± 26.61) were greater than the LOW group’s initial concentrations (145.99 ± 18.5 and 98.32 ± 19.49mg/dl, respectively); although, initial HDL-C was equivalent (LOW = 39.54 ± 12.66 and HIGH = 38.52 ± 9.84mg/dl). Due to the fact that TG are broken-down to supply circulation with a constant stream of free fatty acids, TG concentrations are constantly shifting and thus, are considered a secondary health indicator among the other blood lipids. These initial blood lipid concentrations alone were significant, in that groups screened solely on serum TG concentrations resulted in significantly greater atherogenic blood-cholesterol concentrations, qualifying for borderline high TChol (> 200mg/dl) and LDL-C (130-159mg/dl; NIH, 2001) and therefore, providing further support for TG screening as a primary health indicator. Nevertheless, while the initial atherogenic cholesterol concentrations were greater in the HIGH group, combined average concentrations did not exceed the normal TChol (174.05 ± 40.07) and LDL-C (117.42 ± 30.59) concentrations for this age group.

More recent authors have explored the effect of acute exercise in the untrained, dyslipidemic profiled subject with mixed results (Crouse et al., 1995, 1997; Grandjean et al., 2000; Park & Ransone, 2003; Zhang et al., 2002). For instance, Crouse et al.’s (1995) hypercholesterolemic group of subjects demonstrated the expected TG reduction and rise in HDL-C; however, unexpectedly, there was also a rise in TC (12mg/dl; 4%) and LDL-C (14mg/dl; 8%). This hypercholesterolemic-subject, acute exercise response was unique when compared to the results of others (Grandjean et al., 2000; Park & Ransone, 2003), demonstrating unchanged TC and LDL-C acute exercise responses, similar to the normo-
cholesterolemic subject. Therefore, the results of this investigation support the majority of past research, suggesting acutely, exercise has no influence over TC and LDL-C changes; however, other baseline blood lipid variables did exert their influence over the acute exercise blood lipid response. Furthermore, while this investigation does not support the acute exercise effect to reduce TC nor LDL-C, it does not dispute the established need for chronic exercise to remove excess cholesterol and establish an improved line of lipoproteins within the bloodstream to prevent coronary artery disease (CAD).

The inverse relationship between HDL-C and TG has been well established among researchers (Brites et al., 2000; Durstine et al., 2002; Halle et al., 1999; Miller et al., 2007) and significantly demonstrated via primary acute exercise evidence (Bounds et al., 2000; Crouse et al., 1995, 1997; Dufaux et al., 1986; Ferguson et al., 1998; Grandjean et al., 2000; Park & Ransone, 2003; Sady et al., 1984; Visich et al., 1996). These past researchers suggest exercise contributes to the promotion of reverse cholesterol transport (RCT) via an improved efficiency, measured by the amount of cholesterol found within the HDL particle. On a more basic level, the breakdown of TG via LPLa and an exercise-induced increase in blood flow allow the HDL particle to collect valuable remnants released during exercise, resulting in a short-lived and enhanced reverse cholesterol transport. In the current study, group interactions demonstrated the LOW group responded (Δ1hPE = -0.52 ± 1.75 vs Δ24hPE = +2.69 ± 1.38, p = .005) differently than the HIGH group in absolute (Δ1hPE = +0.42 ± 1.49 vs Δ24hPE = -1.80 ± 2.65) HDL-C changes. More specifically, a subject with a below average initial TG
concentration demonstrated a significant rise in HDL-C, while those with higher concentrations of initial TG did not, despite demonstrating a greater absolute (mg/dl) postexercise TG reduction. These findings are in contrast to the majority of past investigations (Bounds et al., 2000; Crouse et al., 1995, 1997; Dufaux et al., 1986; Ferguson et al., 1998; Grandjean et al., 2000; Sady et al., 1984; Park & Ransone, 2003; Visich et al., 1996) which demonstrated significant HDL-C increases in a wide spectrum of participants (healthy, trained, untrained and unhealthy/hyperlipidemic) and cannot be explained by laboratory variance as a known (control) was measured every 10 samples to maintain consistency, nor poor methodology as these methods are similar to previous investigations demonstrating significant HDL-C adjustments (Grandjean et al., 2000). Therefore, we can only speculate (discussed below) as to why the HIGH group did not also increase their postexercise HDL-C. The results of this investigation may provide further insight as to the intravascular actions that occur during exercise; however, further basic research is needed to elucidate the TG-HDL-C relationship in hyperlipidemic populations.

The results of this investigation illustrate that the LOW group’s HDL-C acute exercise response ($\Delta_{1hPE} = -0.52 \pm 1.75$ vs $\Delta_{24hPE} = +2.69 \pm 1.38$, $p = .005$) were significantly different from the HIGH group’s HDL-C response ($\Delta_{1hPE} = +0.42 \pm 1.49$ vs $\Delta_{24hPE} = -1.80 \pm 2.65$). While the majority of previous research suggests HDL-C increases in response to a single bout of exercise (Bounds et al., 2000; Crouse et al., 1995, 1997; Dufaux et al., 1986; Ferguson et al., 1998; Grandjean et al., 2000; Park & Ransone, 2003; Sady et al., 1984; Visich et al., 1996; see Appendix B), some do report
that a failure of acute exercise to elicit positive changes in HDL-C (Angelopouls, 1993; Fogar, 1994; Lamon-Fava, 1989).

The lack of change in the LOW group’s HDL-C may be related to the exercise bout or physiological differences within this group. Specifically, Gordon et al. (1994) demonstrated large (9mg/dl), but not significant reductions in TG and increases in HDL-C (5mg/dl or 12%) in fit men (57.7ml/kg/min). However, these increases in HDL-C were only seen when participants exercised at a higher intensity (75% versus 60%VO_{2}\text{max}) and expended 800kcals. On the other hand, Park et al. (2003), using a group of sedentary (44.9ml/kg/min) hyperlipidemic men (initial TG = 262mg/dl) demonstrated significant TG reductions and an increase in HDL-C after an acute bout of exercise to expend 405kcal at 75% of their lactate threshold (~60%VO_{2}\text{max}). What’s more, the “average fitness” subjects selected for this investigation within the HIGH group may have needed to expend more kcals through exercise to elicit HDL-C regardless of intensity (the current investigation used 60%VO_{2}\text{max} to expend 600 kcal of energy expenditure).

To put it in perspective, the majority of published research has utilized participants on the far ends of the spectrum, highly trained (Bounds et al., 2000; Ferguson et al., 1998; Fogar et al., 1994; Frey et al., 1993; Gordon et al., 1994; Lamon-Fava et al., 1989; Visich et al., 1996) or very sedentary participants (Crouse et al., 1995, 1997; Grandjean et al., 2000; Park & Ransone, 2003), and some using participants with variable morbid conditions (hypertriglyceridemia and/or hypercholesterolemia). However, the current investigation tested an average fitness group of participants (47.37ml/kg/min) after expending an average amount kcal (600kcal). To that end, the current body of
knowledge “loosely” demonstrates that to induce a lipid effect, the energy expenditure of an acute bout of exercise must be greater when aerobic/metabolic “fitness” is greater (Bounds et al., 2000; Ferguson et al., 1998; Visich et al., 1996). Therefore, possibly the most influential aspect of acute exercise research, the training status of subjects, may have exerted its influence over the current HDL-C unresponsive results.

Another possible explanation for this lack of response in HDL-C of the high TG group may have been an inability to completely adjust for shifts in plasma volume. Interestingly, the individual subject responses were quite variable, as changes in plasma volumes postexercise included large reductions (-13.95%) and even greater increases (+29.97%) throughout the 48h of blood sampling; however, on average the range of change was not as large (+6.83% to +11.10%). While this is not an uncommon finding for acute exercise research as several other investigations demonstrated somewhat-similar plasma volume changes (Crouse, 1995 = -6.6% to +4.0; Granjean, 2000 = -14.0% to +8.0%; Park, 2003 = 3.22% to 3.23%), the Dill and Costill adjustment for acute shifts in plasma volume, although validated in this type of exercise model (Maw et al., 1996), may not be ideal for such large shifts over such a short time. Finally, while research does suggest these results are uncommon, more recent basic research may provide further insight.

Importantly, the current results are supported by basic work from Brites et al. (2000), who demonstrated an altered HDL-C metabolism when comparing the serum samples of normo- and hypertriglyceridemic subjects via cultured measurements of cholesterol efflux. Brites et al. (2000) suggested that these differences are primarily
based on the greater cholesterol ester transfer protein activity (CETPa) within the hypertriglyceridemic group. And these results are in line with many investigations (Boekholdt et al., 2004; Wilund et al., 2002; Yancey et al., 2003). Therefore, the influence of initial TG status on HDL-C increases, may be explained in part by a disadvantageous environment (elevated serum TG) for the activity of CETP (the transfer of TG to HDL-C), resulting in a reduced HDL-C and smaller HDL particle size allowing for increased extraction via renal and hepatic vessels.

Further studies into the distribution of HDL particle subfractions have demonstrated significantly different acute exercise responses among various groups. Kantor et al. (1987) demonstrated a difference in HDL-C subfraction response to an acute bout of exercise when comparing a sedentary and trained group of subjects. More specifically, both groups of subjects demonstrated an increased HDL-C; however, this was accounted for in different subfraction particles, the trained group increased the cholesterol content of the HDL$_2$, while the sedentary did the same in the HDL$_3$ particle. More recently, Wilund et al. (2002) suggested these differences were based on genetic differences in the enzyme function of CETP and thus, resulted in a different HDL-C postexercise response.

What’s more, several of the acute exercise investigations demonstrating significant HDL-C increases were only revealed after collapsing two or three different groups into one large group (Crouse, 1995, 1997; Granjean; 2000; Visich, 1996), primarily because of the smaller effect size of HDL-C changes (i.e., smaller % change than TG) and thus, statistically, a larger $n$ is needed to reach significance. Finally, we can
only speculate to how the unresponsive HDL-C results may change with a greater number of subjects included within the HIGH group.

The two most comparable acute exercise investigations similar to the current investigation are Grandjean et al. (2000) and Zhang et al. (2002); however, differences in methodology as well as the conclusions do exist. Grandjean et al. (2000) established two groups with different baseline cholesterol levels in order to compare the postexercise changes in several blood lipid variables. Grandjean et al.’s (2000) results both support and oppose the current investigations conclusions, demonstrating significant improvement in both TG and HDL-C variables; however, this postexercise blood lipid response was also found to be similar among the normo- and hypercholesterol-defined groups. Therefore, past acute exercise literature has failed to design an investigation in order to specifically compare groups with differing initial TG concentrations.

On the other hand, Zhang et al. (2002) compared a similar group of subjects, using TG as the defining variable; while the experimental methodology differed from the usual acute exercise investigation paradigm. Zhang et al. (2002) used a unique multitrial methodology which consisted of five randomized trials comparing the postexercise responses of a normotriglyceridemic (75-150mg/dl) and hypertriglyceridemic (200-400mg/dl) group of subjects without any significant TG or HDL-C changes 24hPE. Thus, suggesting similar blood lipid responses among normotriglyceridemic and hypertriglyceridemic subjects and therefore, in opposition to the majority of past literature (Durstine et al., 2002; Moffat & Stamford, 2006).
However, surprisingly, the baseline lipid data provided support for the TG-HDL-C inverse relationship with significantly greater HDL-C concentrations found within the normotriglyceridemic group versus the hypertriglyceridemic group. Zhang et al. (2002) also demonstrate an overall greater concentration of lecithin cholesterol acyltransferase activity (LCATa) within the hypertriglyceridemic subjects when compared to a normotriglyceridemic group. This is contradictory to the current knowledge of LCAT because this enzyme is termed the “driving force” of reverse cholesterol transport (RCT) and thus, one would postulate a proportional increase in HDL-C concentrations. Thus, until Zhang et al.’s (2002) unique methodology is replicated, one can only speculate to the causes of their unique findings.

Unfortunately this investigation was limited to blood lipid concentrations and thus, important mechanistic enzymes (LCAT and CETP) were not measured. Importantly, the recent research exploring CETP and CETPa (Brites et. al., 2000) as well as LCAT and LCATa (Zhang et. al., 2002) suggests there is still much to confirm about the effects of acute exercise on blood lipids in abnormal populations. Future applied and basic research should explore the actions of CETP in a population with elevated blood TG in order to better understand the current investigations results.

Another important limitation to this investigation is the significantly greater amount of calories consumed by the LOW group (2965.11 ± 764.29kcal) than the HIGH group (2176.92 ± 439.58kcal; \( p = .021 \)). More importantly, this difference was accounted for in grams of fat (LOW = 109.57 ± 25.2gram > HIGH = 67.24 ± 13.91gram; \( p = .001 \)) and therefore, may have contributed to the significantly less amount of absolute TG
reduction within the LOW group. Bounds et al. (2000) demonstrated this nutritional limitation does not acutely affect the postexercise fasted blood lipid results (Bounds et al., 2000); however, while the difference in nutritional balance has been shown to have no effect over the acute exercise blood lipid results, this article cannot show evidence to dispute the fact that the LOW group consumed a greater amount of kcal than the HIGH group and therefore, may have contributed to a more positive caloric balance and thus, significantly influenced the postexercise blood lipid results (Braun & Brooks, 2008).

Other limitations that may require further investigation include the fact that free fatty acids went unmeasured in this investigation and therefore, overall body fat oxidation can only be assumed via respiratory exchange ratios. Furthermore, the need to define the origin and mobilization of FAs and their lipases during exercise and the hours/days after, are still a necessity in order to fully understand the effects of acute exercise.

Conclusion

In conclusion, this investigation demonstrated significant absolute (mg/dl) TG reductions among groups with initially different TG concentrations; however, these differences were relatively equal when considering the percent change. Secondly, and unique to these results, groups divided solely on initial TG concentrations had different postexercise HDL-C responses. This investigation adds to the growing body of literature in support of exercise’s beneficial role within blood lipids and points to a need for further research into the TG-HDL-C relationship. Practically speaking, these results lend evidence to suggest that individuals with above average TG should adopt chronic
physical activity habits in order to reduce serum TG and further increase their ability to raise HDL-C more effectively through continued exercise training.
REFERENCES


Thompson, P. D., & Rader, D. J. (2001). Does exercise increase HDL cholesterol in those who need it the most? *Arteriosclerosis, Thrombosis, and Vascular Biology, 21*(7), 1097-1098.


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

Project: The Effect of Acute Exercise on Blood Lipid Profiles of Men with Above Average Triglycerides

Researcher(s): Mark McGlynn

Advisor: Michael Kushnick

Department: Recreation and Sport Sciences

Institutional Review Board

Approval Date 8/1/07
Expiration Date 9/5/08

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

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

Adverse events must be reported to the IRB promptly, within 5 working days of the occurrence.
The following research study has been approved by the Institutional Review Board at Ohio University for the period listed below.

**Project:** The Effect of Acute Exercise on Blood Lipid Profiles of Men with Above Average Triglycerides

**Researcher(s):** Mark McGlynn

**Advisor:** Michael Kushnick

**Department:** Recreation and Sport Sciences

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Adverse events must be reported to the IRB promptly, within 5 working days of the occurrence.
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**Project:** The Effect of Acute Exercise on Blood Lipid Profiles of Men with Above Average Triglycerides

**Researcher(s):** Mark McGlynn

**Advisor:** Michael Kushnick

**Department:** Recreation and Sport Sciences

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# APPENDIX B: SUMMARY OF PREVIOUS INVESTIGATIONS

<table>
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<tr>
<th>Reference</th>
<th>Status</th>
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<th>Base</th>
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<th>Δ</th>
<th>% Δ</th>
<th>48hPE</th>
<th>Δ</th>
<th>% Δ</th>
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<td>80% VO2max</td>
<td>TG/ HDL-C</td>
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<td>Combined Trials</td>
<td>TG/ HDL-C</td>
<td>45</td>
<td>48*</td>
<td>3*</td>
<td>7</td>
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### APPENDIX B. Summary of Previous Investigations (mg/dl)

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<th>48hPE</th>
<th>Δ</th>
<th>%Δ</th>
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<td>TG HDL-C</td>
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<td>600 kcal run</td>
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<td>TG HDL-C</td>
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<td>71*</td>
<td>-28*</td>
<td>-28</td>
<td>85</td>
<td>-14</td>
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<td></td>
<td></td>
<td>TG HDL-C</td>
<td>39</td>
<td>45*</td>
<td>6*</td>
<td>16</td>
<td>42</td>
<td>3</td>
<td>7</td>
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<tr>
<td></td>
<td></td>
<td>1,500 kcal run</td>
<td>70% VO2max</td>
<td>TG HDL-C</td>
<td>110</td>
<td>70*</td>
<td>-40*</td>
<td>-36</td>
<td>88*</td>
<td>-16*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TG HDL-C</td>
<td>42</td>
<td>54*</td>
<td>12*</td>
<td>29</td>
<td>52</td>
<td>10</td>
<td>24</td>
<td></td>
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<tr>
<td>17 Bounds ’00</td>
<td>T</td>
<td>1,000 kcal run</td>
<td>70% VO2max</td>
<td>TG HDL-C</td>
<td>113</td>
<td>95</td>
<td>-18</td>
<td>-16</td>
<td>85*</td>
<td>-28*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TG HDL-C</td>
<td>48</td>
<td>53*</td>
<td>5*</td>
<td>10</td>
<td>52</td>
<td>4</td>
<td>8</td>
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<tr>
<td>18 Grandjean ’00</td>
<td>UT</td>
<td>500 kcal run</td>
<td>70% VO2 HC</td>
<td>TG HDL-C</td>
<td>155</td>
<td>140</td>
<td>-15</td>
<td>-9</td>
<td>136</td>
<td>-19</td>
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<td>TG HDL-C</td>
<td>41</td>
<td>43</td>
<td>2</td>
<td>5</td>
<td>46</td>
<td>5</td>
<td>12</td>
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<td></td>
<td>70% VO2 NC</td>
<td>TG HDL-C</td>
<td>132</td>
<td>115</td>
<td>-17</td>
<td>-13</td>
<td>117</td>
<td>-15</td>
<td>-11</td>
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<td>TG HDL-C</td>
<td>43</td>
<td>49</td>
<td>6</td>
<td>14</td>
<td>50</td>
<td>7</td>
<td>16</td>
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<td>Combined Trials</td>
<td>TG HDL-C</td>
<td>144</td>
<td>128*</td>
<td>-16*</td>
<td>-11</td>
<td>128*</td>
<td>-16*</td>
<td>-11</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TG HDL-C</td>
<td>42</td>
<td>46*</td>
<td>4*</td>
<td>10</td>
<td>48*</td>
<td>6*</td>
<td>14</td>
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<tr>
<td>19 Park &amp; Ramsone ’03</td>
<td>UT</td>
<td>411 kcal run</td>
<td>L.T. (~77%)</td>
<td>TG HDL-C</td>
<td>267</td>
<td>240*</td>
<td>-27*</td>
<td>-10</td>
<td>NR</td>
<td>----</td>
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<tr>
<td></td>
<td></td>
<td>TG HDL-C</td>
<td>47</td>
<td>51*</td>
<td>4*</td>
<td>9</td>
<td>NR</td>
<td>----</td>
<td>----</td>
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<tr>
<td></td>
<td></td>
<td>405 kcal run</td>
<td>70% LT (45%)</td>
<td>TG HDL-C</td>
<td>258</td>
<td>258</td>
<td>0</td>
<td>0</td>
<td>NR</td>
<td>----</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TG HDL-C</td>
<td>51</td>
<td>51</td>
<td>0</td>
<td>0</td>
<td>NR</td>
<td>----</td>
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</tr>
</tbody>
</table>

TG = Triglyceride, HDL-C = High Density Lipoprotein-Cholesterol, T = trained, UT = untrained; kcal = Kilocalories, Status = training status of subjects; 24hPE = 24 hours postexercise, 48hPE = 48 hours postexercise, Δ = blood lipid changes (mg/dl), %Δ = percentage change, NR = not reported, * = P < 0.05.

1 = Thompson, 1980; 12 trained men completed a marathon; blood draws @ 5min, 1h, 4h, 18h*, 42h*, and 56hr postexercise
2 = Cullinane, 1982; 10 sedentary men cycled 1h @50rpm and 9 trained cyclist completed 2 trials of 60 and 120min @60rpm
3 = Kantor, 1984; trained men running a Marathon; with postexercise blood draws at 18h and 42h
4 = Dufaux, 1986; moderately trained men completed a 3h running test (covering 19.3-27.5 miles); no plasma volume correction
5 = Sady, 1986; 10 trained men ran a marathon (147-236 min); postexercise sample was collected 18h after finishing
6 = Kantor, 1987; 2 groups of subjects, T and UT completed different kcal expenditures—not measured (1h vs 2h)
7 = Lamon-Fava, 1989; 6 men completed a triathlon; blood collections taken immediately after, 1, 2, 4 and 6 days postexercise
8 = Davis, 1992; 1 healthy group of men completed two separate exercise sessions at LI (low intensity) and HI (high intensity)
9 = Angelopoulous, 1993; 7 untrained men completed a 30 minute treadmill exercise, blood collections were taken at 5, 24, and 48h
10 = Frey, 1993; 13 trained men completed a running field test of (130 min); triglyceride data reported as overall means
11 = Fogel, 1994; 144 mile bike marathon, approximately 11h
12 = Gordon, 1994; 12 “recreational” men completed two 800 kcal exercise protocols at 60 and 75% VO_2 max
13 = Crouse, 1995; hypercholesterolemic men completed either a 50% or 80% VO_2 max exercise bout expending 350 kcal
14 = Visich, 1996; 12 trained men ran three separate protocols at 400, 600, and 800 kcal—no measurement through 48h PE
15 = Crouse, 1997; 26 hypercholesterolemic men trained 3x wk @2 bicycle intensities; blood lipid data per group, collapsed
16 = Ferguson, 1998; a group of 11 trained men ran 4 different running protocols to expend 800, 1100, 1300 and 1500 kcal
17 = Bounds, 2000; diet and exercise investigation with trained runners
18 = Grandjean, 2000; comparison of hyper and normal cholesterolemic, untrained men after 500 kcal expenditure on a treadmill
19 = Park & Ransone, 2003; intensity comparison, LT vs 70% LT threshold in hypertriglyceridemic and hypercholesterolemic groups