Aerobic Training Effects on Rate of Force Production

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> Consider it pure joy, my brothers and sisters, whenever you face trials of many kinds, because you know that the testing of your faith produces perseverance. Let perseverance finish its work so that you may be mature and complete, not lacking anything. — James 1:2–4

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Dedication

This dissertation is dedicated to my family. First, my wife Carissa: You mean the world to me and you will always have my heart. I cannot wait to see what the Lord has in store for us next. To my two boys, Luke (2) and Jack (1 week): Luke, you are amazing! Your spirit is so contagious. I look forward to seeing you grow into the boy and man the Lord has created. And to Jack: You have been keeping a little secret the last 9 months. Though you are not what I had envisioned, I have been entrusted by the Lord to call you my son. I am so thankful for you and can't wait to see how you will continue to surprise your mom and me. I love each one of you!

Abstract

Aerobic training has been negated as a training modality for many anaerobic sports and their participating athletes. The trend is due to a growing theory that aerobic training has the potential to inhibit strength, power, and/or overall anaerobic performance. However, there are studies that have produced conflicting as well as inconclusive findings when aerobic conditioning is performed concurrently with strength training. PURPOSE: The primary purpose of this research was to investigate the effects of aerobic training on anaerobic output defined by vertical jump (VJ), broad jump (BJ), and Wingate testing results in male and female recreationally trained runners over a period of 16 weeks. Additionally, body weight (BW), percent body fat (PF), pre-exercise resting heart rate (RHR), VO_{2max} and anaerobic threshold (AT) were examined as exploratory measures. METHODS: A 16-week observational pre- and posttest design was used to determine the effects of a single phase aerobic training specific to preparing for a marathon on measures of anaerobic power as well as anthropometric measures and body composition. RESULTS: The primary findings demonstrated that after 16 weeks of a specified aerobic training for a marathon, there was no change in VJ ($p = 0.307$) and there was an increase in BJ ($p = 0.011$). There was no change in peak power (males $p = 0.08$, females $p = 0.114$). There was an improvement in fatigue index (males $p = 0.017$, females $p = 0.006$) during the 30-second Wingate cycle test among study subjects, however there was a decrease in PF (p=0.0001). In addition, marginal increases in VO_{2max} ($p = 0.055$) as well as decreases in PF ($p = 0.001$) and RHR ($p = 0.001$) were observed. CONCLUSION: The findings do not support the notion of the universal nature

that aerobic training has a negative effect in the development of rate of force production. However, the adverse effect may hold true in regards to explosive strength due to limited neural activation. The interference effect may also be present in a concurrent setting where aerobic and anaerobic training protocols are assessed in exclusively aerobically trained subjects. However, in a sport performance setting, as well as in general public health, the training for physical fitness requires the development of muscle strength, power and endurance. The present findings indicate that the influence of aerobic training is an important factor for not only overall fitness, but it does not appear to constrain anaerobic performance.

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Chapter 1

Introduction

Background

Aerobic training has been negated as a training modality for many anaerobic sports and their participating athletes. The trend is due to a growing theory that aerobic training has the potential to inhibit strength, power, and/or overall anaerobic performance. Previous research assessing the effect of aerobic training on anaerobic performance has shown decreased strength output and diminished vertical jump performance. However, the majority of the research involved concurrent aerobic training simultaneously with strength and weight training in varied sample populations (Dudley & Djamil, 1985; Hennessy & Watson, 1994; Kraemer et al., 1995; Powers & Howley, 2004; Staron et al., 1994). However, there are several other studies that have produced conflicting and inconclusive findings when aerobic conditioning is concurrently performed simultaneously with strength training (Balabinis, Psarakis, Moukas, Vassiliou, & Behrakis, 2003; Bell, Petersen, Wessel, Bagnall, & Quinney, 1991; Hickson, Dvorak, Gorostiaga, Kurowski, & Foster, 1988; Hikida et al., 1997; Hunter, Demment, & Miller, 1987; McCarthy, Agre, Graf, Pozniak, & Vailas, 1995; McCarthy, Pozniak, & Agre, 2002; Sale, MacDougall, Jacobs, & Garner, 1990; Wilmore & Costill, 2004). Several limitations and differences exist in the above-listed research. There are differences in the modality of the strength training, the modality of the aerobic conditioning, and the study participants. The most significant limitation is the lack of anaerobic power (rate of force production) as the dependent variable. Most studies have centralized their question

around strength output as the primary outcome variable, leaving a significant void in the theory that aerobic training may inhibit the performance of an anaerobic athlete.

In sport training and conditioning research there is a link specific to the terms *muscular strength* and *muscular power*, which have often have been interchanged and often misused or misinterpreted. Strength, or maximal force, is defined as the maximal force that a muscle or muscle group can generate (Tomlin & Wenger, 2001). Athletes are routinely anaerobically tested in a lab on a bench press or leg press (Abadie, Altorfer, & Schuler, 1999; Brooks, Fahey, & Baldwin, 2005; Kelly et al., 2007; Tomlin & Wenger, 2001), when strength carries no time or speed components, just force output specific to the sport outcome. Power relies on a time component and measures the rate at which the force is produced. Maximal power is equal to the force produced multiplied by the distance over which it acted, divided by the time it took to move the force that particular distance (Power = Force x Distance / Time),(Tomlin & Wenger, 2001). Power is measured by tests such as; vertical jump, broad jump, jumps measured via force plates, and the 30-second anaerobic Wingate power test (Hoffman et al., 2005; Hoffman, Cooper, Wendell, Im, & Kang, 2004; Hoffman & Kang, 2002). However, strength and power tests within research and sport settings are often used interchangeably, yet relinquish separate benefits to anaerobic athletes; synonymously interchanging these terms may be inaccurate by definition.

Aerobic conditioning benefits to the anaerobic athlete have been overlooked. Improving an athlete's aerobic conditioning level or fitness level, predominantly assessed by the VO_{2max}, can lead to a greater ability to replenish phosphocreatine stores which is

linked to increasing the ability to perform repeated high-intensity bouts (Bogdanis, Nevill, Boobis, & Lakomy, 1996; Laurent et al., 1992; McCully, Boden, Tuchler, Fountain, & Chance, 1989; McCully, Vandenborne, DeMeirleir, Posner, & Leigh, 1992; McMahon & Wenger, 1998; Takahashi et al., 1995; Tomlin & Wenger, 2001; Yoshida & Watari, 1993). Aerobically trained athletes possess a greater ability to reduce anaerobic glycolysis and limit lactate production while performing intense anaerobic bouts by the improved ability to utilize oxygen during the high-intensity working bout (Hamilton, Nevill, Brooks, & Williams, 1991). Additionally, athletes with an increased aerobic capacity are able to clear lactate more efficiently while recovering, thereby increasing power during subsequent working bouts (Oyono-Enguelle et al., 1990; Taoutaou et al., 1996). Inorganic intercellular phosphate, a contributing factor in muscle fatigue, is linked to faster removal rates in aerobically conditioned athletes as well (Wood et al., 2001). Finally, injury rate reductions have been linked to higher levels of aerobic capacity (Kaufman, Brodine, & Shaffer, 2000) as expressed in military studies.

Additional Purposes to the Primary Study

Public Health Implications

Participation in aerobic or endurance training has been well documented specific to public health and well-being. Reducing the risk of chronic disease and overall mortality has been documented throughout public health literature (Myers, 2003). However, the proposed study and its potential impact on the field of public health would predominantly stem from the pre- and posttest metabolic testing, heart rate response, and

body composition data. Fitness levels as defined by VO_{2max} have been associated with metabolic changes that improve longevity and quality of life.

Statement of the Problem

There is a lack of anaerobic power data collection in the context of aerobic conditioning among male and female college age athletes. Provided the lack of depth and breadth of research on concurrent strength and aerobic training and the conflicting results, it is clear that there is not enough research to conclude that aerobic conditioning specifically hinders anaerobic power or the rate of force production beyond that of concurrent training. A second confounder is the selection criteria of the participants. To this point, much of the data collected on the influence of aerobic conditioning and strength or power on minimally trained athletes or athletes that have not been exposed to previous aerobic conditioning. In the limited number of studies with aerobically trained subjects, there was no strength or power decreases, therefore suggesting that a history of aerobic conditioning may be the critical key to negating the affects found in some of the research. Consequently, the primary purpose of this study is to investigate the effects of aerobic conditioning on anaerobic power and force production. The present study will also provide unique insight with regard to public health specific to body composition, resting heart rates and increased aerobic capacity.

Significance of the Study

Specific to the anaerobic athlete, the ability to perform repeated high-intensity bouts as observed in anaerobic sports such as hockey, basketball, and football is of great importance and is potentially linked to an athlete's aerobic capacity. Similarly, injury

rates appear to be decreased by aerobic conditioning, as well as enhanced glucose uptake and heightened tissue restoration with the influence of aerobic conditioning.

Currently, three major issues in the literature are present. First, much of the data in terms of linking aerobic conditioning with strength or power have primarily been collected concurrently, and is widely dispersed in terms of protocol and methodology. Second, the limited amount of research that incorporated previously aerobically trained individuals creates an area of interest. As mentioned in the previous concurrent aerobic and strength training data, those subjects who had a history of aerobic conditioning were more resistant to the negative effects of strength development. Finally, the ability to produce beneficial data as it relates to the area of public health provides a framework for the proposed research.

Need for the Study

Aerobic conditioning is often thought by many sport practitioners to inhibit and interfere with anaerobic power production and maintenance. However, the scientific evidence is unclear on this topic. It is the purpose of this study is to determine what effect aerobic conditioning has on anaerobic power production on previously aerobically trained male and female athletes as well as produce meaningful data specific to public health.

Hypothesis

The following is the primary hypothesis for this study: Anaerobic power and rate of production of force will not be negatively affected by aerobic training in recreational runners.

The following is the secondary hypothesis for this study: Aerobic training will additionally improve body composition and cardiovascular measures in recreational runners.

Limitations

The sample selected for these study consisted of students enrolled in PE 1262– Marathon Training (course #902354) at the University of Minnesota, Twin Cities in the spring semesters of 2009, 2010, and 2011. These students possessed homogeneity, defined by their recreational status with most not competing in an anaerobic sport at the time of selection and never having previously completed a marathon. However, a wide range of training backgrounds may have existed.

Total training volume may yield unique results. Students were all volunteers, which may raise motivational issues of the participants. The sample population was not randomly selected; consequently, the application of the results may be limited due to the potential threats to validity.

Definition of Terms

Aerobic Conditioning/Endurance Training. As defined under the Methods Section, each participant trained to complete a marathon. Therefore, aerobic conditioning is defined as the time spent running in preparation for the marathon. Participants were required to complete a training protocol set forth by the instructor of the course. Each individual participant, via training logs, recorded aerobic conditioning training volumes. This served as a means to determine possible thresholds for aerobic conditioning and determine levels or strata of this independent variable.

Aerobic Conditioning from a Physiological Level. Physiologically, in aerobic activity, ATP (energy) production occurs in the mitochondria, which contains the appropriate enzymes for oxidative phosphorylation. The ATP production is caused by the multifaceted interaction between the Krebs cycle and the electron transport chain. The Krebs cycle's role is to complete the oxidation of the substrates obtained from food and form NADH along with FADH, which then enter the electron transport chain. The electron transport chain then converts NADH and FADH into ATP and water. The water is a result of oxygen-accepting electrons; for this reason, we breathe oxygen to be used as the acceptor of electrons in aerobic metabolism (Powers & Howley, 2004).

Anaerobic Power Output. The mean and peak power production quantified via the 30-second Wingate cycle ergometer expressed in watts (W). Relative peak power was expressed by dividing peak power by the participant's weight in kilograms. A fatigue index was also used during this test to determine the decline in power.

Anaerobic Rate of Force Production Evaluation: The rate of force production quantified by the two-leg vertical jump test as well as a two-leg broad jump test.

Body Composition: Percent lean and fat mass was collected via BodPod using whole body air displacement plethysmography.

Chapter 2

Literature Review

Effects of Aerobic Conditioning

Aerobic training on a structural level elicits a significant increase in activation frequency of motor units and a slight increase in oppositional load against motor units. Quintessential cardiovascular training that presents this stimulus to the muscle is represented in weight bearing settings (i.e., running) as well as non-weight bearing settings (swimming and cycling; Brooks et al., 2005).

Aerobic training results in little to no effect on the cross-sectional area of muscle and muscle fibers. However, specific to type I slow-twitch fibers, these fibers may experience slight increases in cross sectional size due to increases glycogen storage as well as oxidative metabolism (Rico-Sanz et al., 2003; Wilmore & Costill, 2004). The majority of adaptations to skeletal muscle occur within the metabolic realm. The adaptations to muscle under the influence of cardiovascular training produce changes in fiber type, capillary supply, myoglobin content, mitochondrial function, and oxidative enzymes (Wilmore & Costill, 2004).

Fiber Type

Aerobic training relies primarily on type I, slow-twitch muscle fibers. In response to the volume and intensity of training, type I fibers have minimal capability to become larger in cross-sectional area (Rico-Sanz et al., 2003). Fast-twitch fibers are not generally recruited to the same capacity as slow-twitch fibers during cardiovascular training and therefore do not experience similar responses in cross-sectional area. Similarly,

~~rearch~~research suggests little change in muscle fiber percentage between slow-twitch and fast-twitch fibers; however, changes have been documented in fast-twitch subtypes (Wilmore & Costill, 2004). Type IIx fast-twitch fibers are called upon less during aerobic training, and consequently, possess a lower aerobic capacity. The HERITAGE study suggests that type IIx fast-twitch fibers may transform to type IIa fast-twitch fibers. However, these results are far from conclusive (Wilmore & Costill, 2004).

Capillary Supply

A significant adaptation to aerobic training is the increase in capillaries surrounding the muscle fibers. Research has documented that cardiovascularly trained individuals have significantly greater capillary density compared to sedentary populations (Hermansen & Wachtlova, 1971). Dense capillary content allows a heightened interchange of gasses, heat, waste, and nutrients between the working skeletal muscle and the blood delivered (Wilmore & Costill, 2004).

Myoglobin Content

Upon entering the muscle, oxygen binds with an iron-containing compound that resembles hemoglobin called myoglobin. Myoglobin transports oxygen molecules from the cell membrane to the mitochondria. Of importance to cardiovascular training is the fact that myoglobin has the capacity to store and release oxygen, particularly when oxygen becomes sparse during muscle contraction. The release of oxygen often occurs during the lag time between the beginning of exercise and the amplified cardiovascular transportation of oxygen. Although the detailed distributions of oxygen delivery are not completely understood, cardiovascular training has been documented to increase

myoglobin content by 75–80%. This adaption of muscle significantly impacts the ability for oxidative metabolism (Wilmore & Costill, 2004).

Mitochondrial Function

Aerobic energy production occurs within the mitochondria. As a result, the effect on mitochondrial function is obvious. The major adaptations with cardiovascular training on the mitochondria are increases in size and number. Research has shown increases in the actual number of mitochondria by as much as 15%, as well as increases in the total size by as much as 35%, in as little as 27 weeks of training (Wilmore & Costill, 2004). As the volume of cardiovascular training rises, so do the number and the size of the mitochondria (Wilmore & Costill, 2004).

Oxidative Enzymes

Mitochondrial efficiency and the oxidative formation of ATP are increased by oxidative enzymes. Aerobic training has been shown to increase enzyme activity. Training induced enzymatic activity contributes not only to the number and size of the mitochondria, but also to the metabolic consequence of mitochondrial changes. It is suggested that increased enzyme activity creates a slower use of muscle glycogen and a reduced production of lactate during exercise at given intensities. This adaptation is likely to have importance to lactate threshold (Holloszy & Coyle, 1984).

Energy Sources

Aerobic training forces adaptations to the ability of the muscle to store and metabolize glycogen and fat. Muscle glycogen is often the primary substrate for aerobic exercise. The primary response to depleted glycogen storage is a heighted resynthesis

capacity. With proper recovery and dietary intake, cardiovascularly trained muscle stores significantly greater amounts of glycogen (Greiwe et al., 1999).

Additionally, aerobically trained muscle contains higher amounts of intermuscular triglycerides. Muscular enzymes responsible for lipid breakdown are increased with aerobic training. This adaptation allows for trained muscle to better oxidize lipids, decreasing the breakdown of glycogen (Wilmore & Costill, 2004).

Benefits of Aerobic Conditioning

Repeated Bouts

The influence of aerobic conditioning is believed by some to hinder an athlete's ability to produce force and may limit strength. One benefit of aerobic conditioning for the anaerobic athlete is a heightened ability to perform repeated bouts of high-intensity exercise. The enhanced ability to replenish phosphocretine (PCr) stores through aerobic conditioning is correlated with improved performance in repeat anaerobic bouts (Bogdanis, Nevill, Boobis et al., 1996; Laurent et al., 1992; McCully et al., 1989; McCully et al., 1992; McMahon & Wenger, 1998; Takahashi et al., 1995; Tomlin & Wenger, 2001; Yoshida & Watari, 1993). Bogdanis, Nevill, Boobis et al. (1996) noted that oxygen availability which is increased via aerobic conditioning contributed to the replenishment of PCr stores. This finding resulted in a correlation between a male athlete's aerobic metabolism and his ability to successfully perform repeated sprints, an anaerobic trait in high-intensity performance. A number of other investigations reported enhanced recovery levels of PCr in endurance-trained athletes compared with sprinters and controls (Laurent et al., 1992; McCully et al., 1989; McCully et al., 1992; Takahashi

et al., 1995; Yoshida &Watari, 1993). Takahashi et al. (1995) reported that a high oxygen consumption in trained endurance athletes results in a greater ability to resynthesize PCr, particularly in high-intensity activity. This finding was echoed by Yoshida and Watari (1993), where PCr resynthesis levels were higher in long-distance runners when compared to control subjects. Laurent et al. (1992) documented a smaller decrease in exercise-induced PCr levels in aerobically trained athletes compared to nonaerobically trained and control subjects. Laurent et al. (1992) also determined that restoration of PCr was heightened in trained subjects. Studies conducted by McMahon and Wenger (1998) and Tomlin and Wenger (2001) suggested similar relationships between aerobic fitness and power maintenance, as well as the recovery from high-intensity intermittent exercise. Both studies concluded that an athlete with a greater aerobic capacity, defined by VO_{2max}, has an increased ability to resynthsize PCr following high-intensity exercise as evidenced by repeat anaerobic bouts with greater maximal force production and sustained power. Finally, McCully et al. (1989) documented higher rates of PCr resynthesis in aerobically trained athletes following repeated exercise bouts than that of untrained subjects. These findings have been associated with improved power recovery, a very advantageous characteristic for the anaerobic athlete.

Additional benefits surrounding repeated anaerobic bouts are also linked to an athlete's aerobic capacity. Aerobically trained athletes, defined by VO_{2max}, have been shown to consume higher levels of oxygen during intermittent intervals. Therefore, consuming more oxygen may reduce the dependence on anaerobic glycolysis, and limit lactic acid production equating to reduced hydrogen ion concentrations and greater

athletic performance (Hamilton et al., 1991). This physiological adaptation resulting from aerobic conditioning may improve performance.

Enhanced lactate clearance is an additional benefit that is improved by aerobic conditioning (Nelson, Arnall, Loy, Silvester, & Conlee, 1990; Taoutaou et al., 1996). An increased rate of lactate clearance during resting periods has been documented with aerobically trained athletes (Nelson et al., 1990; Taoutaou et al., 1996). Oyono-Enguelle et al. (1990) found that aerobically trained subjects reach their peak lactate level more quickly than untrained subjects after exercise implying a swifter removal of lactate from the active skeletal muscle in trained subjects. A similar cross-sectional study also linked athletes with greater aerobic capacities to enhanced blood lactate removal (Taoutaou et al., 1996). Taoutaou et al. (1996) showed increased lactate removal in various recovery tactics with the endurance-trained athletes when compared to those with lower aerobic capacities. However, methodological discrepancies do exist regarding lactate removal improvements with increased aerobic capacity. A difference in the timing of the lactate collection was apparent among the studies mentioned therefore creating issues specific to the validity of the data when comparing the research.

A final factor specific to aerobic conditioning is linked to an athlete's ability to perform repeated anaerobic bouts involving the removal of accumulated inorganic intracellular inorganic phosphate (P_i). Intracellular phosphate is thought to be a major contributor to muscular fatigue, which is particularly important as it relates to repeat anaerobic bouts. However, limited research exists on aerobic conditioning and the influence it may have on P_i reduction and improved repeat bouts. Yoshida and Watari

(1993) investigated the differences between aerobically conditioned subjects and untrained controls related to their metabolic responses to repeated anaerobic bouts. Although no statistically significant differences existed on P_i removal in the aerobically trained subjects compared to the untrained controls, the speed in which P_i levels were removed was significantly faster in the aerobically trained participants. Thus, aerobically trained subjects experienced a greater capacity to quickly remove inorganic phosphates, which contributes to a greater phosphocreatine restoration, as well as oxidative capacity of muscles.

Enhanced Glucose Uptake

Glycogen storage is a part of many anaerobic sports, particularly those involving high-intensity bouts followed by low activity recovery bouts. Progressive bouts of aerobic training that continually diminish muscle glycogen leads to a biological adaptation of increased muscle glycogen stores (Greiwe et al., 1999; Hickner et al., 1997). This "supercompensation," or an enhanced capacity to store glycogen, is an essential characteristic for peak performance in anaerobic athletes, who are highly reliant on glycogen to fuel high-intensity activity.

A cellular process leading to enhanced glycogen uptake as a result of aerobic training is an increase of muscle glucose transport protein-4 (GLUT-4). The GLUT-4 protein is a key component in skeletal muscle kinase. This adenosine monophosphate activated protein kinase is only signaled when exercise is greater than 60% of a person's VO_{2max}, which has been associated with aerobic training (Chen et al., 2003; Kraniou, Cameron-Smith, & Hargreaves, 2004). Kraniou et al. (2004) investigated the effects of

seven successive days of 60-minute bouts of aerobic training on male cyclists. There was a statistically significant increase in transcription of GLUT-4 following each bout of aerobic conditioning. Researchers also found a statistically significant overall increase in GLUT-4 protein that gradually accumulated over the weeklong analysis.

Injury Rate Decrease

Reductions in injury rates with greater levels of aerobic fitness have been documented in research (Almeida, Williams, Shaffer, & Brodine, 1999; Jones, 1993; Jones, Bovee, Harris, & Cowan, 1993; Knapik, Ang, Reynolds, & Jones, 1993; Kowal, 1980; Reynolds et al., 1994; Shaffer, Brodine, Almeida, Williams, & Ronaghy, 1999). Most injury research focuses on military recruits and is often strengthened by large sample sizes. An analysis conducted by Kaufman et al. (2000) correlated aerobic capacity to the reduction of basic training injuries, as well as "overuse" injuries. According to Almeida et al. (1999), military cadets with low levels of physical fitness, based on initial physical testing training, were more likely to sustain injury during training. An interventional trial to determine a potential cause of injury was conducted during an early Marine military recruit training phase. The investigators concluded that the recruits who were subjected to an intervention protocol with an emphasis on aerobic conditioning during their early training phase were significantly less likely to suffer an injury while in training. Specifically, lower extremity stress fractures were reduced by 55%, and overuse injuries were also significantly reduced in the aerobic intervention group.

Sport practitioners use a multidisciplinary approach to injury prevention and utilize aerobic training as a means to prepare the body and supporting structures for

intense activity and muscular contraction. Connective tissues (tendons, ligaments, cartilage) that provide musculoskeletal support may require several weeks or months of loading in order to prepare for the hypertrophy adaptations of muscle (Elliott, Wagner, & Chiu, 2007). However, muscle itself can experience hypertrophy in as little as a few weeks and begins to become denser in a much shorter period of time (Dawson et al., 1998; Staron et al., 1994). Many times athletes choose—or are prescribed by practitioners—to accelerate their training at a very rapid pace without the appropriate progression to develop the ligamentous joint complex, therefore increasing injury rates and decreasing performance (Kawakami, Muraoka, Ito, Kanehisa, & Fukunaga, 2002). Investigations of the stretch shortening cycle (SSC) indicate that if the connective tissues are strengthened in both quality and quantity, the rate of force production is greatly improved (Ettema, 2001).

Active Recovery/Local Circulation

Local circulation increased from sustained aerobic training has been documented to improve muscle restoration by withdrawing cellular debris and blood lactate as well as heightening nutrient transportation (Ahmaidi et al., 1996; Bogdanis, Nevill, Lakomy, Graham, & Louis, 1996; Hughson, 2003; Signorile, Tremblay, & Ingalls, 1993). Data from Ahmaidi et al. (1996), Bogdanis, Nevill, Lakomy et al. (1996), and Signorile et al. (1993) demonstrated the effectiveness of aerobic training in recovery as evidenced by the enhanced removal of blood lactate compared to passive settings. The mechanism is theorized to be the direct result of aerobic training. Specifically, aerobic exercise

vasodilatation of skeletal muscle, resulting in an increased blood flow to damaged cells (Ahmaidi et al., 1996; Bogdanis, Nevill, Lakomy et al., 1996; Signorile et al., 1993).

Hindrance of Aerobic Conditioning Theorized

Muscle Fiber Transformations

Transformation in muscle fiber structure, specifically as a function of isomyosin alterations, have been theorized as a potential cause of strength decreases under concurrent strength and endurance training conditions (Chromiak & Mulvaney, 1990; Dudley & Fleck, 1987). Chromiak and Mulvaney (1990), and Dudley and Fleck (1987) both articulated this theory in review articles without any applicable measurements and justified the argument based on physiological theory.

It has also been proposed that within an environment of concurrent strength and aerobic conditioning, another potential mechanism for inhibition may involve the specific transformation of Type IIa fibers (Costill, Fink, & Pollock, 1976; Houston, 1978; Staron et al., 1994). Costill et al. (1976) demonstrated fiber area ratio transformation, yet could not conclude fiber type transformation given a lack of significant differences in cross-sectional area of Type I, IIa, and IIx muscle fibers after training. Similar conclusions were also made by Staron et al. (1994) and therefore make it difficult to support the argument that muscle fibers experience fiber type transformations.

Finally, the functional properties of Type II fibers when compared to Type I are different and they respond differently when exposed to particular stresses. Physiologically, Type II fibers respond when exposed to stand-alone strength training with hypertrophy and greater cross-sectional fiber areas while Type I fibers experience

dramatic increases in mitochondrial and capillary density when limited to aerobic conditioning solely. However, when athletes are exposed to both anaerobic and aerobic protocols, distinct muscle fiber adaptations are not as clearly identified (Brooks et al., 2005; Dawson et al., 1998; Jacobs, Esbjörnsson, Sylven, Holm, & Jansson, 1987; Jansson, Esbjörnsson, Holm, & Jacobs, 1990).

Changes in Motor Recruitment/Neuromuscular System

The demands that are placed upon the neuromuscular system with strength training are far different than when participating in aerobic conditioning. Motor unit recruitment while under the influence of aerobic conditioning is primarily dependent on the duration and intensity (Henriksson & Reitman, 1976). Strength training, on the other hand, increases the coordination of the motor units to accommodate the increased force requirement of muscle contraction (Bandy, Lovelace-Chandler, & McKitrick-Bandy, 1990). It has been hypothesized that concurrent conditions will alter the motor unit recruitment patterns associated with each separate activity (Chromiak & Mulvaney, 1990; Elliott et al., 2007; Leveritt, Abernethy, Barry, & Logan, 1999). However, there is limited research to support the conclusion that motor unit recruitment plays a significant factor in the inhibition of force/power under concurrent force/power and endurance training. Dudley and Djamil (1985), as well as Chromiak and Mulvaney (1990), have only speculated on the potential involvement of neural factors in decreased strength development in documented research. However, both studies again focused on the concurrent setting of aerobic and strength training. Appropriate study designs (i.e.,

isolated training) have not been used to support the idea that aerobic conditioning, whether it be stand-alone or concurrent, negatively affects neural components.

Similar studies, specifically investigating myofibrillar ATPase activity have also been conducted with similar results. ATPase is a function of myosin heavy chain composition where it is believed that muscle containing myosin heavy chain Type II fibers are significantly faster than Type I (Fry, Allemeier, & Staron, 1994). Bell, Syrotuik, Martin, Burnham, and Quinney (2000) examined ATPase activity in university men and women and produced results that demonstrated greater activity with a strength training only group when compared to an endurance only group; however, a concurrent group also produced a greater ATPase activity than the endurance only group as well.

Endocrine Responses

Aerobic conditioning may also create a catabolic effect on muscle tissues. The catabolic state is the product of a decreased anabolic hormone release as well as an increase in catabolic hormones (Elliott et al., 2007). Kraemer et al. (1995) studied hormonal effects on army subjects stratified in four different training groups. Strength training groups did respond more positively with less exercise-induced cortisol responses, as well as greater testosterone production after exercise when compared to the concurrent strength and endurance group. However, the study design failed to produce equivalent training stimuli, which appears to be a contributing factor leading to the theorized overtraining effect of the strength and endurance group.

Bell et al. (1991), as well as Bell et al. (2000), sought to examine hormonal responses with training, but failed to reveal any differences in testosterone responses in

either group (strength or strength and endurance) when evaluating concurrent groups of strength and aerobic trained athletes. Bell et al. (2000) also discussed the necessity of adding strength training to endurance athletes programs for the purpose of maintaining testosterone levels and increasing anabolic environments.

Overtraining

Overtraining is a decrease in performance resulting from exposing the body to higher amounts of training volume and intensity (i.e., stress) than it can handle (Elliott et al., 2007). Though poorly defined within bodies of research, overtraining has often been speculated to be a potential result of combined strength and endurance training, particularly in review articles (Chromiak & Mulvaney, 1990; Dudley & Djamil, 1985; Elliott et al., 2007). In one of the first documented strength and endurance studies of note, it was speculated that the 80 minutes per day of training by the strength and endurance group was overtraining the subjects and that was the cause of strength decreases during the ninth and tenth weeks. However, the endurance only group received the same volume—80 minutes of cycle ergometer training—and did not produce the same decreases (Hickson, 1980). Therefore, it appears the authors were rather selective and biased in their conclusion that overtraining was the root of the strength decreases. Interestingly, similar studies of concurrent strength and endurance training have since been conducted and have used numerous different endurance- and strength-training protocols (intensity, duration, modality, frequency) making results difficult to interpret based on threats to external validity. Based on the vast differences in study design, it is

very difficult to conclusively state that overtraining is the contributor to decreases in strength and/or force production.

Protein Turnover

Aerobic training has been shown to diminish the total protein synthesis rates in skeletal muscle after acute bouts (Booth & Watson, 1985; Dohm, Kasperek, Tapscott, & Barakat, 1985; Rennie & Tipton, 2000). It has been discussed that endurance training combined with strength training may have the potential to impair adaptive responses involving protein synthesis and diminish force production (Kraemer et al., 1995). It is speculated by some that incorporating aerobic training on top of strength and power training may not only overtrain athletes, but the oxidative stress may also lead to decreased protein turnover and muscle atrophy. However, the discussion is speculative and is not supported in research nor has this disruption been theorized outside of Kraemer et al. (1995).

Aerobic Conditioning and Concurrent Strength

Researchers suggest that strength development may be impaired when aerobic conditioning is simultaneously performed at a moderate level (Bell et al., 2000; Bogdanis, Nevill, Lakomy et al., 1996; Casey, Constantin-Teodosiu, Howell, Hultman, & Greenhaff, 1996; Dudley & Djamil, 1985; Oyono-Enguelle et al., 1990; Sale et al., 1990; Thayer, Collins, Noble, & Taylor, 2000). Bell et al. (2000) specifically analyzed hormonal levels in four training groups. Cortisol levels increased in the concurrent strength and endurance training group after 12 weeks. The result implicates cortisol as a potential inhibitor to strength training gains and is the result of endurance training.

However, after 12 weeks, Bell et al. (2000) did not observe significant differences in testosterone, human growth hormone, or sex hormone in any of the four training groups and could only discuss that the findings suggest endurance training suppresses adaptations to strength training. Interestingly, a previous study by Bell et al. (1991) resulted in cortisol levels increasing not only with the concurrent group, but also in the strength group, an observation that was not present in the more recent 2000 study.

Similarly, Hennessy and Watson (1994) investigated four groups over an 8-week period to determine the effects of concurrent strength and aerobic conditioning. Results showed an increase in strength (1-repetition maximum) in the strength training only group, but failed to show any decreases in strength in lower body exercises for the concurrent strength and endurance group. Interestingly, results also showed an increase in upper body strength in the concurrent group. Data collected also showed slight improvements in vertical jump and 20-meter sprint times in the concurrent group but not enough improvement to cite statistical significance. Likewise, Dudley and Djamil (1985) presented results that indicated a significant difference in strength output between the strength group and the concurrent group. Interestingly, however, the concurrent group's strength was not hindered by the influence of aerobic conditioning but did not yield the improvements that were experienced by the strength only group thus contributing to the statistical difference.

Several studies exist that have not been able to significantly show the interference effect that the above authors have alluded to (Balabinis et al., 2003; Bell et al., 1991; Hickson et al., 1988; Hikida et al., 1997; Hunter et al., 1987; McCarthy et al., 1995;

McCarthy et al., 2002; Samuel, Holcomb, Guadagnoli, Rubley, & Wallmann, 2008; Staron et al., 1994). Much of the discrepancies between the studies are a result of study length, the use of trained and untrained subjects, the resistance and aerobic training modalities, and the training protocols used in each study.

Sale et al. (1990) found no difference in lower extremity strength when comparing a strength group to a concurrent strength and endurance group of subjects' posttests calculating the voluntary strength of an athlete's 1-repetiton maximum (1-RM) after 22 weeks resulted in no significant difference between groups. After 22 weeks of training, the majority of the participants in the concurrent strength and endurance group improved 1-RM for the leg press lower extremity strength test.

Bell et al. (1991) experienced similar outcomes when testing the effect of 16 weeks of training on lower extremity strength and hormonal levels. The concurrent strength and endurance group as well as the strength group both showed significant increases in leg press output. As mentioned previously, both groups also registered higher cortisol levels, a hormone Bell et al. (2000) speculated to limit strength output in the previous study. However, cortisol levels began to subside in the strength group after the eighth week.

In another study conducted by McCarthy et al. (1995), the concurrent endurance and strength groups, as well as the strength only group, showed similar improvements in 1-RM squat and bench press, vertical jump, and isometric knee extension tests. Improvements in both groups were seen over 10 weeks of training three days per week.

Results indicated that three days per week of concurrent training provided significant improvements in strength.

Finally, in a shorter seven-week trial by Abernethy and Quigley (1993) on upper body strength and endurance, researchers saw no strength interference with the concurrent strength and endurance group. Tests focused solely on the strength of the upper body via isokinetic elbow extension; aerobic conditioning was performed via upper body ergometry. As mentioned, results showed no inhibition of strength with the concurrent group; however, few studies have focused primarily on the upper body (i.e., subjecting the upper body to aerobic conditioning).

A common theme and potential threat to the above studies that note that concurrent strength and aerobic conditioning cannot coexist is the fact that each study had wide ranges of methodologies. The primary differences were in the modality of the strength training, the modality of the aerobic conditioning, and the study subjects. In previously mentioned studies that assessed primarily isotonic strength training movements, any statistically significant differences between the strength groups and the concurrent groups were only found when isotonic strength training measurements were tested as opposed to force/power measurements (Craig, Lucas, Pohlman, & Stelling, 1991; Dudley & Djamil, 1985; Hennessy & Watson, 1994; Hickson, 1980; Kraemer et al., 1995). Research involving solely upper body strength (Abernethy & Quigley, 1993) saw no inhibition in strength when concurrent aerobic conditioning was present; the same was true for studies that assessed isokinetic strength (Dudley & Djamil, 1985; Nelson et al., 1990). Of the studies, those that used running as the means of aerobic conditioning

resulted in a strength development inhibition when sample means of the concurrent strength and aerobic groups where compared to the stand-alone strength group (Craig et al., 1991; Hennessy & Watson, 1994; Hickson et al., 1988; Kraemer et al., 1995). The studies that involved cycling as a mode of aerobic conditioning have not resulted in consistent strength development interference (Dudley & Djamil, 1985; McCarthy et al., 1995; Nelson et al., 1990; Samuel et al., 2008). Finally, when rowing and upper body arm ergometers were used as a means of aerobic conditioning, there was no strength interference (Abernethy & Quigley, 1993; Bell et al., 1991; Bell et al., 2000). In terms of subject selection, studies where participants had been exposed to aerobic conditioning prior to their study participation showed no inhibition in strength development, therefore indicating that a history of aerobic conditioning may create a lesser susceptibility to strength inhibition under concurrent conditions (Bishop, Jenkins, Mackinnon, McEniery, & Carey, 1999; Hickson et al., 1988; Marcinik et al., 1991; McCarthy et al., 1995).

Regardless of the inconsistency in the current research, a potential conflict with the theory that aerobic conditioning and anaerobic strength cannot be combined has been the definition and interpretation of strength. The primary focus of current research has been force production, via a 1-RM bench press or leg press, or some form of isokinetic limb extension, and not the rate at which the force can be produced which is crucial for most anaerobic athletes.

Heart Rate Recovery/Deceleration

Heart rate activity, specifically heart rate recovery and heart rate deceleration (HRD), is at the forefront of much public health and medical research. It is hypothesized

that HRD postexercise is linked to mortality and that a retarded HRD may be a powerful predictor of all-cause mortality (Cole, Blackstone, Pashkow, Snader, & Lauer, 2011). Historically, the focus of most of the medical research has investigated changes in heart rate activity during exercise and stress testing (Ellestad & Wan, 1975; Lauer et al., 1996; Sandvik et al., 1995). However, a heightened interest in the decline in heart rate after the discontinuation of exercise has recently drawn interest.

Increased heart rate during exercise is the result of decreased parasympathetic activity and increased sympathetic activation (Arai et al., 1989; Brooks et al., 2005). The decline of heart rate after exercise is due to the reactivation of the parasympathetic nervous system (Brooks et al., 2005; Imai et al., 1994). Specifically, HRD is a result of vagal reactivation. Due to the relationship between vagal activity and HRD, the relationship may be used as a decisive prognostic sign for mortality risk (Cole et al., 2011).

Cole et al. (2011) analyzed HRD on 2428 patients and during the 6-year follow-up; an atypical HRD was significantly predictive of death. Of the 213 deaths, over 50% recorded abnormal HRD values. Interestingly, Cole et al. (2011) also discovered a strong correlation between HRD and exercise capacity in both men and women. Researchers noted that an athletic population showed improved HRD when compared to other populations.

Similar research links physical fitness, operationally defined by VO_{2max}, with HRD. Goldsmith, Bigger, Bloomfield, and Steiman (1997) and Goldsmith, Bloomfield, and Rosenwinkel (2000) show a relationship between vagal activation and modulation

with maximal oxygen uptake in healthy males when age was controlled. The relationship between aerobic capacity and HRD is a vital key to bolstering not only public health interventions, but also has implications to sports performance.

Heart Rate Recovery/Deceleration and Performance

Heart rate deceleration (HRD) has been identified as a powerful psychophysiological tool in predicting sport performance. The psychophysiological ability to manage heart rate and heart rate activity has been connected to not only increased performance, but also to greater attention to a salient stimulus, increased reaction time, and overall cognitive function and activity (Jennings & Wood, 1977; Lacey & Lacey, 1978).

Most competitive sports and in-game performance activities involve a preparatory period leading up to a critical juncture in a competition (i.e., a free throw in basketball, batting in baseball, snapping the ball in football). The preparatory period is thought to be a predictive marker of final performance (Guillot et al., 2005; Landers, Han, Salazar, & Petruzzello, 1994). The ability to decrease heart rate to an optimal level during this period prior to the task at hand is assumed to be a vital component for optimal attention processes, and thus predictive of ideal performance (Landers et al., 1994; Tremayne & Barry, 2001). Most literature linking HRD and performance has investigated aiming proficiency, such as archery (Landers et al., 1994; Robazza, Bortoli, & Nougier, 2000), rifle shooting (Konttinen, Lyytinen, &Viitasalo, 1998), pistol shooting (Tremayne & Barry, 2001), or golf putting (Boutcher & Zinsser, 1990; Hassmen & Koivula, 2001).

Greater HRD was revealed in elite level shooters and golfers when compared to less skilled archers and beginning golfers (Boutcher & Zinsser, 1990).

Performance measures rely on postural balance and control to maintain reference while aiming. In a study by Cottyn, De Clercq, Crombez, and Lenoir (2008), researchers investigated the effects of complex balance performance in reference to HRD. The authors found HRD to have a similar effect on the preparation phase of a balance beam acrobatic element in female gymnasts. However, results failed to show significant findings relating HRD and increased balance measures (Cottyn et al., 2008). Although this study did not show performance proficiency, it continued to support the previous literature related to neurocardiologic and physiologically mediated HRD. Nonetheless, performance research in sport has indicated HRD and performance are interrelated.

The connection between HRD and vagal modulation in public health settings and HRD and greater in-competition sport performance cannot go unnoticed. In a public health environment, empirical evidence exists directly linking HRD and mortality, and aerobic capacity appears to be a significant factor in that connection (Goldsmith et al., 1997). Sport performance also appears to be enhanced with greater HRD as cited above. However, the relationship between aerobic capacity and HRD has not been fully supported by empirical research for the anaerobic athlete and is an area of future investigation.

Public Health Outcomes

Oxygen Transport

There are a multitude of physiological benefits to cardiovascular exercise. One specific element is designed to seek the improvements in muscular function and the relationship to the body's ability to utilize and transport oxygen (VO_{2max}). Improved ability to utilize oxygen results in decreased fatigue when regular exercise is performed. This is of particular importance to individuals with cardiovascular disease. Individuals with cardiovascular or heart disease generally experience an exercise capacity lower than that of healthy populations. By improving VO_{2max}, and consequently oxygen utilization, this would lead to improvements in fatigue resistance. Cardiovascular training in relation to oxygen transport may also be improved by blood vessel dilation in response to hormonal activity stemming from exercise. These results, matched with improved vascular wall function and the physiologically improved ability to support skeletal muscle with oxygen during exercise leads to obvious public health implications (Myers, 2003).

Body Composition

The influence of cardiovascular training is hypothesized to positively affect body composition and reduce body fat, which directly affects the field of public health and well-being. Implications of this effect would potentially be twofold: 1) impact on resting metabolic rate (RMR) and 2) the physiological impact submaximal endurance training places on the body, tissues, and substrate utilization. Weight loss resulting in improved body composition, produced by an increase in endurance training with an unchanging

energy intake, is of obvious importance. Also, lack of weight loss with improved body composition can result in favorable health changes. Equally important is weight loss associated with lean tissue retention and RMR. Oftentimes, weight and fat loss following chronic endurance training is a result of a negative energy balance (Stiegler & Cunliffe, 2006). However, subsequent mechanisms may positively impact RMR contributing to improved body composition. The most noticeable association between cardiovascular training and RMR is the potential for skeletal muscle growth. Additionally, increases in RMR after endurance training during the post exercise recovery period (EPOC) may also be present. Furthermore, mechanisms such as increased protein turnover, sympathetic nervous system activity, and uncoupled respiration have been theorized (but not conclusively shown) to increase RMR (Stiegler & Cunliffe, 2006). Nevertheless, endurance training appears to be a factor in increasing RMR and improving body composition, which have significant implications to the field of public health (Stiegler & Cunliffe, 2006).

Endurance training at low to moderate intensity levels (<50% of VO_{2max}) relies on fats as the predominate substrate. As intensity rises (>50% of VO_{2max}), there is a progressive decrease in fat oxidation giving way to carbohydrate oxidation. As oxygen supplied by the heart and lungs to the contracting skeletal muscles is unable to meet the demand of the growing intensity, glucose is forced to become the primary fuel (Stiegler & Cunliffe, 2006). Regardless of the reciprocal decrease in fat oxidation for increases in glucose breakdown, the absolute quantity of fat oxidation may increase as the total amount of work at greater intensity exercise increases. Therefore, fat utilization may be

maximized at high intensities. Thus the primary goal of improving body composition from a public health standpoint may be to encourage higher intensity endurance training. However, individuals with low or diverse aerobic fitness (i.e., low VO_{2max}values) and poor body composition (high body fat values) may not be capable of high intensity endurance training thereby limiting the effect (Stiegler & Cunliffe, 2006).

Resting Heart Rate

Finally, potential outcomes on resting heart rate (RHR) values may be of value to general health. A lower resting heart rate is a result of chronic cardiovascular training and has been documented to decrease approximately 10 beats per minute after as little as 10 weeks of endurance training (Wilmore & Costill, 2004). However, the actual physiological mechanisms remain unclear. The most likely mechanism for decreased RHR is increased plasma volume, which allows for a greater return of blood back to the heart and increased stroke volume (Wilmore & Costill, 2004). More applicable to the public health arena is the importance of RHR as a predictive factor of cardiovascular disease and all-cause mortality. Clinical trial investigations examining heart-rate lowering drugs suggest that RHR reductions are of particular benefit to chronic heart failure, acute myocardial infarction, and angina patients (Fox et al., 2007). Similarly, pathophysiological data suggest a high RHR may have direct correlation to adverse effects on coronary atherosclerosis, the occurrence of myocardial ischemia and ventricular arrhythmias, and left ventricular function. Additionally, documented literature indicates increases in disease risk with RHR above 60-beats-per-minute (Fox et al.,

2007). Nonetheless, cardiovascular training as it relates to RHR is of particular importance to the field of public health and wellness.

Chapter 3

Methods

Participants

Participants were enrolled in PE 1262-Marathon Training (course #902354) at the University of Minnesota, Twin Cities in the spring semester of 2009, 2010 and 2011. Participants were preapproved by a physician to participate in the marathon course by participating in a complete medical physical. In order to enroll in the course, the student was informed that on the first day of the class, they would have to be able to complete a 30-minute run without stopping.

Participants were excluded if they had a known chronic health problem, did not have the ability to continuously run for a minimum of 30 minutes or have not been granted permission to enroll in PE 1262-Marathon Training (course #902354) at the University of Minnesota, Twin Cities in the spring semester of 2009, 2010 and 2011.It is essential that the participants came into the study with previous aerobic conditioning, as prescribed by the instructors for the course. Participants completed a prescreening questionnaire assessing their general health, competition and training status, and nutritional health before they were allowed to participate in the study.

Participants were excluded if they currently had a known chronic health problem, did not complete a physician's physical, or were not accepted into PE 1262-Marathon Training (course #902354) at the University of Minnesota, Twin Cities in the spring semester of 2009, 2010 or 2011.

Study Design

A 16-week observational pre- and posttest design was used to determine the effects of aerobic conditioning on measures of anaerobic power as well as anthropometric and body composition. The dependent variables were two-legged vertical jump and two-legged broad jump to determine power production and the Wingate cycle ergometer test to determine anaerobic power. Secondary dependent variables are maximal oxygen uptake, resting heart rate, maximum anaerobic threshold heart rate, and body composition. The independent variable was the aerobic conditioning running protocol set forth by the class, with the intention to complete a marathon at the conclusion of the course. The independent aerobic conditioning variable was additionally divided into strata depending on the volume and mileage recorded by each participant. Each subject kept training logs to record their adherence to the prescribed aerobic training protocol set forth by the course instructor. The training logs allowed the researcher the ability to create strata among the participants based on aerobic training volumes as well as mileage times. This design was able to test whether aerobic conditioning affected power performance and allowed the researcher to potentially fill a research gap in the current research trends related to aerobic training and its effects on anaerobic power.

Measures and Questionnaires

Participants were required to attend an orientation session where they were familiarized with the testing procedures as well as completed two questionnaires to assess training history, health history, injury history, nutritional history as well as oral contraceptive history and usage. Participants then returned to the laboratory for pre- and

posttesting. On each testing day, participants had their height, weight, and body composition recorded. Participants performed a 5-minute warm-up on a treadmill as a self-selected speed ranging from 3.0–3.5 mph. This was immediately followed by a two-legged vertical jump test as well as two-legged broad jump test to assess rate of force production. Following the completion of the anaerobic testing procedures in weeks 1 and 16, participants completed an 8- to 10-minute graded exercise test to assess maximal oxygen uptake (VO_{2max}). The rather rigorous maximal oxygen uptake test will only be conducted as a pre- and posttest. A Wingate cycle ergometer test was conducted after a 48-hour waiting/resting period following the maximal oxygen uptake test.

Anthropometry

Each participant's height (in meters) and weight (in kilograms) was measured using a certified scale and standard stadiometer. Height measurements were recorded to the nearest millimeter. Weight was measured to the nearest 0.1 kg. Anthropometric measures were collected at all testing sessions on each participant.

Maximal Oxygen Uptake (VO_{2max}) and Lactate Levels (LT)

Subjects performed a graded exercise test modified Bruce treadmill protocol to determine aerobic fitness and aerobic/lactate threshold based on their maximal oxygen uptake via treadmill on each visit to the laboratory. The subject was instructed to breathe into a mouthpiece while the intensity of the exercise gradually increased to induce voluntary fatigue. Inspired and expired airflow as well as CO2 and O2 were analyzed by a Medgraphics Ultima metabolic analyzing system (Medgraphics, Minneapolis, MN). Ventilation (Ve), CO_2 production (VCO_2), O_2 production ($V0_2$), and gas exchange ratio

(VCO_2 / VO_2; R) was measured directly breath-by-breath. Heart rate (HR) was also measured and monitored as the test progressed (Wasserman, Whipp, Koyal, & Beaver, 1973).

Maximal oxygen uptake is important in providing a measure of aerobic power output, otherwise known as work capacity (Brooks et al., 2005; Hickson et al., 1988). Oxygen uptake is heavily reliant on cardiovascular health, an influential variable to the aerobic assessment of this study. The relationship between maximal oxygen uptake and anaerobic output via maximal two-leg vertical jump, rate of force production (ground force reaction), and a 30-second Wingate test is a great value to the anaerobic athlete.

Anaerobic/lactate threshold (AT) is a vital measure to both the anaerobic and aerobic athlete (Brooks et al., 2005). The graded exercise test provided the determination of AT. The threshold was determined by the point of the nonlinear increase in Ve, the nonlinear increase in Vco_2, the increase in tidal O_2 without the decrease in tidal CO_2, and an increase in R (Wasserman et al., 1973).

Example Maximal Oxygen Uptake Protocol (based of each participant's 2-mile time trial speed):

Participant runs a 10min 2-mile (this would equal an average pace of 5:00/mile, or 12 miles per hour treadmill speed).

Sample stages:

Stage 1: 3 minute 3.0 mph @ 0.0% grade incline.

Stage 2–8: 1 minute stage length @ 1.0% grade incline. Speed starts at 9.2 mph and increasing 0.4 mph until reaching 12.0 mph at stage 8. Treadmill speed held

at 12.0mph and increase grade by 1.5% each minute after that until the participant reaches failure.

Body Composition

Body fat percentage was measured via Bod Pod (Life Measurement Inc., Concord, CA). Air displacement plethysmography determines percent body fat and fat free mass of the subject. A simple noninvasive 5-minute test consisting of measuring the subject's mass and volume (inside the Bod Pod chamber) was used to estimate the subject's body composition. Again, this procedure was conducted at each visit to the laboratory as a reasonable predictor of fat-free mass (Brooks et al., 2005; Hickson et al., 1988), and may have a significant correlation to anaerobic power output and or other public health effects.

Maximum Two-Leg Vertical Jump, Broad Jump and Rate of Force Production (Ground Reaction Forces)

Vertical jump is widely accepted as an accurate test of maximal leg power and is a recurring movement found in many sports such as basketball, volleyball, and track and field (18). Maximum vertical jump height was measured using a Vertec (MF Athletic Company, Cranston, RI). Subjects performed three maximal jumps on each testing day following a 5-minute warm-up. Subjects were required to stand with both feet flat on the ground, while lowering their body to the ground by producing flexion at the knee, hip, and trunk while extending the shoulders until knee flexion reached an approximate 90-degree angle. When the participants reached the 90-degree flexion point at the knee, they were then instructed to jump vertically as high as possible while extending their dominant

hand towards the Vertec system of markers. Maximal vertical jump height was measured by recording the highest marker moved on the Vertec system (Samuel et al., 2008).

A similar test was conducted to measure broad jump. Much like the above vertical jump test, the broad jump test assesses maximal leg power in a similar recurring linear movement found in several aspects of numerous sporting events. Subjects were again required to stand firmly on the ground, and then instructed to lower their body to the ground with ankle, knee and hip flexion. At the point of approximately 90-degree knee flexion, subjects were instructed to jump outward as far as possible. Back heel measurements were taken and the farthest measured distance was recorded.

Peak Anaerobic Power and Anaerobic Capacity

A Wingate anaerobic test via cycle ergometer was performed following the mandatory 48-hour waiting/resting period following the subject's maximal vertical jump and rate of force production tests. A mechanically braked cycle ergometer was used to determine anaerobic measures as a fixed resistance was applied to the flywheel of the cycle and the subject pedaled maximally for 30 seconds as power output is measured. Peak anaerobic power is calculated by force x distance (number of revolutions x distance of revolutions/ time in minutes). Anaerobic Capacity was measured by analyzing the total work during the test (Force x Total Distance in 30 seconds). Percent of peak power decrease during the 30-second test was additionally assessed and used as a measure of fatigue.

Performance on the Wingate anaerobic test is related to high-intensity activities such as the 50-meter sprint and 25-meter swim. It is less related to the maximal two-leg

vertical jump; however, will provide useful data when combined with anaerobic running program (Bell et al., 1991).

Pre-Exercise Resting Heart Rate

Resting heart rate measures were taken approximately 5 minutes prior to the graded exercise test. Polar heart rate monitors (Polar Electro Oy, Kempele, Finland) were placed on subjects after a 5-minute waiting period.

Statistical Methods

The analysis was completed using SPSS for Windows 18.0. The significance or alpha level for all analysis was set at $p < 0.05$.

The analysis for this research had two main objectives. The first objective is an exploratory analysis evaluating differences in anthropometry, body composition, aerobic capacity, lactate threshold, and resting heart rate in subjects pre- and posttraining. In addition to descriptive statistics, statistical difference in pre- and postdata was calculated to allow comparison with published data. The second objective is to explore the contribution aerobic training had on force production and anaerobic power. Matched pairs *t*-tests were used to determine the effect aerobic training had on anaerobic force production and the magnitude of relative change for the force and power production variables measured during the jumping and cycle ergometer tests. 2x2 ANOVA analysis was used to identify the effect training volume (high volume and low volume) had on the dependent variables. Correlational (Pearson's *r*) analysis was also conducted to determine predictive relationships among outcome variables.

Chapter 4

Results

Tests for normality were conducted and all data exhibited constant variance, sufficing the assumptions for normality with the exception of Broad Jump (BJ), cycle ergometer, Wingate scores for differences in Peak Power (PP) and differences in Wingate Percent Decrease (%D). The data set for BJ appeared to contain an outlier that was a result of coding error and when removed, BJ variables were normally distributed. PP and %D contained no coding errors; therefore analysis was performed with nonparametric statistics. To look at the differences stratified by gender, matched paired *t*-tests were used to analyze the effects. Volume analysis was conducted by using 2x2 ANOVA.

Vertical Jump

Mean subject pretest scores were 17.67 +- 3.53 inches. No significant changes took place in VJ performance of subjects during the 16-week aerobic training period ($p = 0.307$). Female ($n = 60$) subjects also experienced no change in VJ (17.36 +- 3.52 inches, $p = 0.081$). Male (18.85 +- 3.33 inches, $n = 27$) VJ measurements additionally remained statistically unchanged ($p = 0.190$).

Stratum was created based on pretest heights to examine differences in subjects with greater anaerobic capacity upon the onset of the present study (Chu, 1996). Pretest jump scores for each subject were categorized into “below average” height (male VJ < 16 inches, female VJ < 12 inches) “average” (male VJ = 16.5–20 inches, female VJ = 12–16 inches) and “above average” (male VJ > 20.5 inches, female VJ > 16.5; Chu, 1996). The subjects with below average VJ ($n = 12$) experienced a 2.13-inch decrease in jump height

(p = 0.0001) after 16weeks of aerobic training. Subjects with average VJ scores (n = 62) saw no change in their jump height (p = 0.582). Above average subjects (n = 13) saw a 1.45-inch increase in their VJ heights after the aerobic training (p = 0.0001).

Table 1

Differences in Vertical Jump (Below Average)

		Paired Differences							
					95% Confidence Interval of the Difference				
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper	t	df	Sig. (2-tailed)
Pair 1	Post-training Vertical Jump (in.) - Prep-training Vertical Jump (in.)	-2.12500	1.15059	.33215	-2.85605	-1.39395	-6.398	11	.000

[a] Difference: Vertical Jump Stratum Code = -1 (BELOW AVERAGE)

Table 2

Differences in Vertical Jump (Average)

		Paired Differences							
					95% Confidence Interval of the Difference				
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper	t	df	Sig. (2-tailed)
Pair 1	Posttraining Vertical Jump (in.) - Pretraining Vertical Jump (in.)	-.08871	1.26272	.16037	-.40938	.23196	-.553	61	.582

[a] Difference: Vertical Jump Stratum Code = 0 (AVERAGE)

Table 3

Differences in Vertical Jump (Above Average)

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	Post-training Vertical Jump (in.) – Pre-training Vertical Jump (in.)	1.45833	.75252	.21723	.98020	1.93646	6.713	11	.000

[a] Difference: Vertical Jump Stratum Code = 1 (ABOVE AVERAGE)

As seen in Table 1, the below average values for VJ are significant ($t(12) = 6.398$, $p = 0.000$), suggesting that the subjects with below average VJ pretest scores experienced a significant decrease in VJ performance. Table 2 displays no significance between pre- and posttest scores ($t(62) = 0.553$, $p = 0.582$), suggesting average VJ pretest scores produced no significant differences in VJ performance. Finally, Table 3, the above average model shows significance ($t(12) = 6.713$, $p = 0.000$), suggesting subjects with pretest VJ score Above Average experienced a significant increase in VJ.

Subjects were also stratified by training volume and other dependent variables. Subjects who trained during the 16 weeks at low volume defined as less than 20 miles per week experienced no statistically significant difference in VJ changes than those subjects who performed high volume of running mileage of 20 miles or greater ($p = 0.962$).

Table 4

Differences in Vertical Jump Based on Overall Training Volume

Volume	Mean	Std. Error
Low	.020	.320
High	.038	.212

Table 4 illustrates changes in vertical jump after training for each volume.

Table 5

Differences in Vertical Jump Based on Training Volume and Gender

Gender	Volume	Mean	Std. Error
Female	Low	-.175	.326
	High	-.371	.262
Male	Low	.214	.551
	High	.447	.334

Table 5 shows that the difference in VJ after training.

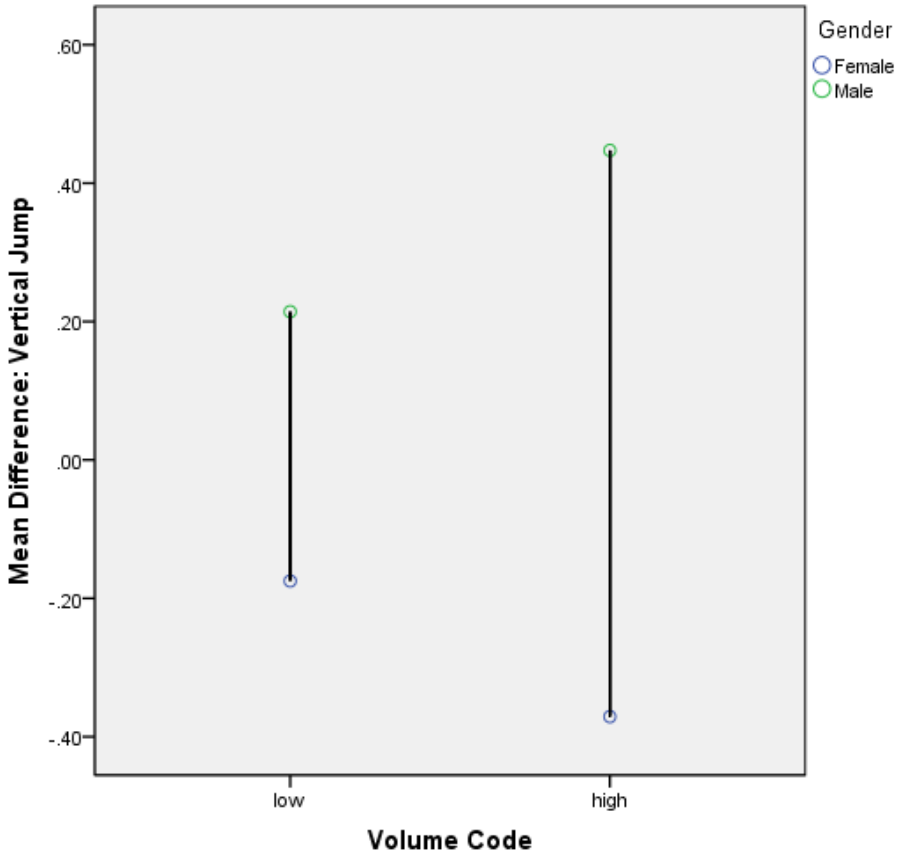

Figure 1. Difference in vertical jump by gender and volume level.

Table 6

Tests of Between-Subjects Effects: Differences in Vertical Jump Based on Training Volume and Gender

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	8.678[a]	3	2.893	1.362	.261	.053
Intercept	.048	1	.048	.023	.881	.000
Gender	5.251	1	5.251	2.473	.120	.033
Volume	.005	1	.005	.002	.962	.000
Gender * Volume	.663	1	.663	.312	.578	.004
Error	154.997	73	2.123			
Total	164.000	77				
Corrected Total	163.675	76				

[a] R Squared = .053 (Adjusted R Squared = .014)

As shown in Table 6, the overall corrected model shows no significance (F (3,73) = 1.362, p = 0.261), suggesting that none of the predictors makes a more significant contribution to the prediction of an individual's VJ than the mean across all participants.

Table 7

Pairwise Comparisons: Differences in Vertical Jump Based on Training Volume

					95% Confidence Interval for Difference	
(I) Volume	(J) Volume	Mean Difference (I–J)	Std. Error	Sig.[a]	Lower Bound	Upper Bound
Low	High	-.019	.384	.962	-.784	.747
High	Low	.019	.384	.962	-.747	.784

Note. Based on estimated marginal means.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 7 illustrates that the difference in VJ after training between high and low volume.

Broad Jump

Subjects tested for BJ performance experienced a 2.34-inch increase collectively in all 38 subjects [(males = 16, females = 22) $p = 0.011$]. Female subjects increased BJ performance by 1.91 inches ($p = 0.023$). Males experienced no change in BJ length ($p = 0.215$). Stratified data based on pretest performance were additionally analyzed (Chu, 1996). Subjects with average ($n = 18$) and above average ($n = 14$) BJ measurements produced increases in scores (average jump– $p = 0.080$, above average– $p = 0.041$). While subjects with pretest BJ scores below average ($n = 6$) experienced a 4.4-inch decrease in BJ ($p = 0.0001$).

Table 8

Differences in Broad Jump (Below Average)

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	Posttraining Broad Jump (in.) - Pretraining Broad Jump (in.)	-4.41667	1.31972	.53877	-5.80163	-3.03170	-8.198	5	.000

[a] Difference: Broad Jump Stratum Code = -1 (BELOW AVERAGE)

Table 9

Differences in Broad Jump (Average)

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	Posttraining Broad Jump (in.) - Pretraining Broad Jump (in.)	3.83333	5.44491	1.28338	1.12564	6.54102	2.987	17	.008

[a] Difference: Broad Jump Stratum Code = 0 (AVERAGE)

Table 10

Differences in Broad Jump (Above Average)

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	Posttraining Broad Jump (in.) - Pretraining Broad Jump (in.)	2.08333	3.11582	.89946	.10364	4.06303	2.316	11	.041

[a] Difference: Broad Jump Stratum Code = 1 (ABOVE AVERAGE)

As seen in Table 8, the overall corrected model is significant ($t(6) = 8.198$, $p = 0.000$).

Training volume additionally had no impact on BJ performance ($p = 0.962$). Subjects with low weekly training volume experienced no difference in BJ performance measures than when compared to those with high weekly running volumes.

Table 11

Differences in Broad Jump Based

Volume	Mean	Std. Error
Low	1.896	1.497
High	1.810	.976

Table 11 demonstrates changes in BJ after training for each volume.

Table 12

Differences in Broad Jump Based on Training Volume and Gender

Gender	Volume	Mean	Std. Error
Female	Low	1.958	1.339
	High	1.850	1.467
Male	Low	1.833	2.678
	High	1.769	1.287

Table 12 shows that the difference in BJ after training.

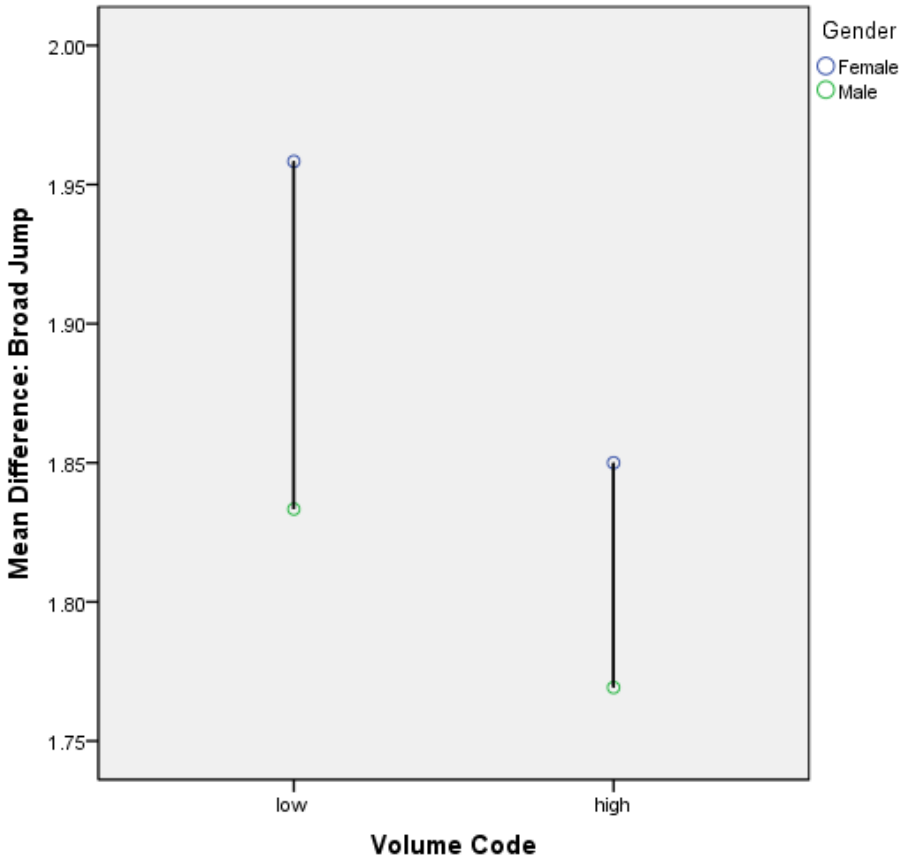

Figure 2. Difference in BJ by gender and volume level.

Table 13

Tests of Between-Subjects Effects: Differences in Broad Jump Based on Training Volume and Gender

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	.225[a]	3	.075	.003	1.000	.000
Intercept	92.524	1	92.524	4.300	.046	.112
Gender	.071	1	.071	.003	.954	.000
Volume	.050	1	.050	.002	.962	.000
Gender * Volume	.003	1	.003	.000	.990	.000
Error	731.604	34	21.518			
Total	862.625	38				
Corrected Total	731.829	37				

[a] R Squared = .000 (Adjusted R Squared = -.088)

As shown in Table 13, the overall corrected model shows no significance ($F(3,34) = 0.003$, $p = 1.0$), suggesting that none of the predictors makes a more significant contribution to the prediction of an individual's BJ than the mean across all participants.

Table 14

Pairwise Comparisons: Differences in Broad Jump Based on Training Volume

					95% Confidence Interval for Difference[a]	
(I) Volume	(J) Volume	Mean Difference (I–J)	Std. Error	Sig.[a]	Lower Bound	Upper Bound
Low	High	.086	1.787	.962	-3.545	3.718
High	Low	-.086	1.787	.962	-3.718	3.545

Note. Based on estimated marginal means

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 14 shows that the difference in BJ after training between high and low volume.

Wingate Power Analysis

Related Samples Wilcoxon Signed Rant Test was used to analyze Peak Power (PP) performance in the 30-second cycle ergometer Wingate Power Test stratified for gender (n = 15). Pre- and posttest male (n = 5) PP performance displayed no significant changes (mean pretest PP = 877.2 +-156.32 W, p = 0.08). Similarly, female subjects (n = 10) expressed no change in PP performance during the 30-second test (mean pretest PP = 677.2 W, p = 0.114). Volume stratification was also additionally analyzed in PP analysis. No significant differences existed between low and high training volume among the 15 subjects (p = 0.738).

Table 15

Tests of Between-Subjects Effects: Differences in Peak Power Based on Training Volume and Gender

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	1693.896[a]	2	846.948	.131	.878	.021
Intercept	57495.550	1	57495.550	8.894	.011	.426
Gender	82.689	1	82.689	.013	.912	.001
Volume	757.571	1	757.571	.117	.738	.010
Gender * Volume	.000	0	.	.	.	.000
Error	77576.733	12	6464.728			
Total	137726.960	15				
Corrected Total	79270.629	14				

[a] R Squared = .021 (Adjusted R Squared = -.142)

As shown in Table 15, the overall corrected model shows no significance (F (2,12) =0.131, p = 0.88), suggesting that none of the predictors makes a more significant contribution to the prediction of an individual's PP than the mean across all participants.

The results of the ANOVA also demonstrate that levels in volume, when we ignore the influence of gender, provided no predictive power in determining changes in PP after training (F (1,12) =0.117, p =0 .738).

Table 16

Pairwise Comparisons: Differences in Peak Power Based on Training Volume

					95% Confidence Interval for Difference[a]	
(I) Volume	(J) Volume	Mean Difference (I–J)	Std. Error	Sig.[a]	Lower Bound	Upper Bound
Low	High	20.817[a]	42.482	.633	-71.744	113.377
High	Low	-20.817[c]	42.482	.633	-113.377	71.744

Note. Based on estimated marginal means

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 16 shows that the difference in PP after training between high and low volume.

Table 17

Tests of Between-Subjects Effects: Difference in % Decrease in Power Based on Training Volume and Gender

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	149.807[a]	2	74.904	.824	.462	.121
Intercept	1215.620	1	1215.620	13.375	.003	.527
Gender	137.113	1	137.113	1.509	.243	.112
Volume	93.500	1	93.500	1.029	.330	.079
Gender * Volume	.000	0	.	.	.	.000
Error	1090.649	12	90.887			
Total	2640.190	15				
Corrected Total	1240.456	14				

[a] R Squared = .121 (Adjusted R Squared = -.026)

[b] Computed using alpha = .05

As shown in Table 17, the overall corrected model shows no significance (F (2,12) =0.824, p = 0.462).

The results of the ANOVA also demonstrate that levels in volume, when we ignore the influence of gender, provided no predictive power in determining changes in PP after training (F (1,12) =1.029, p = 0.330).

Table 18

Pairwise Comparisons: Differences in % Decrease in Power Based on Training Volume

(I) Volume	(J) Volume	Mean Difference (I–J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Low	High	2.314[a]	5.037	.654	-8.661	13.289
High	Low	-2.314[c]	5.037	.654	-13.289	8.661

Table 18 shows that the difference in %D after training between high and low volume.

VO_{2max}

VO_{2max} results for all the subjects displayed a mean 1.394-ml/kg/min improvement (pretest mean 50.74 +- 7.77 ml/kg/min, $p = 0.055$). Females did not show improvements (pretest mean 47.06 +- 5.24 ml/kg/min, $p = 0.90$), however, males did show statistical improvement (57.45 +- 7.3 ml/kg/min, $p = 0.001$).

Table 19

Mean Differences in VO_{2max} (All Participants)

Mean	Std. Error
1.394	.636

Table 19 demonstrates VO_{2max} increases of all participants.

Table 20

Differences in VO_{2max} Based on Gender

Gender	Mean	Std. Error
Female	.242	.722
Male	2.546	1.048

Table 20 demonstrates changes in VO_{2max} after training for each gender.

Table 21

Differences in VO_{2max} Based on Training Volume

Volume	Mean	Std. Error
Low	2.015	1.066
High	.774	.695

Table 21 illustrates changes in VO_{2max} after training for each stratified training volume.

Table 22

Differences in VO_{2max} Based on Training Volume and Gender

Gender	Volume	Mean	Std. Error
Female	Low	1.315	1.086
	High	-.831	.952
Male	Low	2.714	1.835
	High	2.378	1.012

Table 22, shows the difference in VO_{2max} after training.

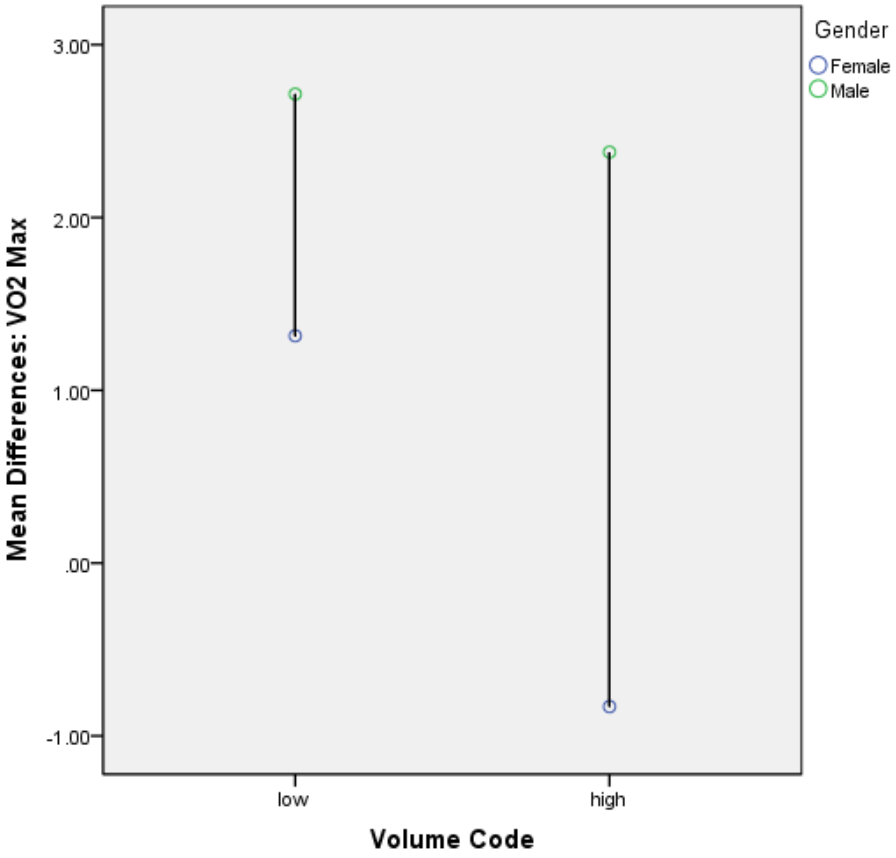

Figure 3. Difference in $VO_{2max}$by gender and volume level.

Table 23

Tests of Between-Subjects Effects: Differences in VO_{2max} Based on Training Volume and Gender

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	153.316[a]	3	51.105	2.168	.099	.083
Intercept	113.176	1	113.176	4.802	.032	.063
Gender	77.281	1	77.281	3.279	.074	.044
Volume	22.414	1	22.414	.951	.333	.013
Gender * Volume	11.919	1	11.919	.506	.479	.007
Error	1697.089	72	23.571			
Total	1931.280	76				
Corrected Total	1850.404	75				

[a] R Squared = .083 (Adjusted R Squared = .045).

As seen in Table 23, the overall corrected model shows no significance (F (3,72) = 2.168, p = 0.099).

The results of the ANOVA also demonstrate that levels in volume, when we ignore the influence of gender, provided no predictive power in determining changes in VO_{2max} after training (F (1,72) =0.951, p = 0.333). Figure 3 explains these differences further.

Table 24

Pairwise Comparisons: Differences in VO_{2max} Based on Training Volume

(I) Volume	(J) Volume	Mean Difference (I–J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Low	High	1.241	1.273	.333	-1.296	3.778
High	Low	-1.241	1.273	.333	-3.778	1.296

Note. Based on estimated marginal means

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 24 shows that the difference in VO_{2max}after training between high and low volume.

Pre-Exercise Resting Heart Rate

Pre-exercise resting heart rate measures of all subjects (n = 37, mean HR = 76 +- 9.4 bpm) displayed a 6.44 bpm improvement (p = 0.0001). Additionally, males (n = 17, mean HR = 73 +-10.35) witnessed heart rate improvements (p = 0.001). Females (n = 20, mean HR = 79.15 +- 8.0 bpm) saw a 5.08 bpm decrease in resting measures (p = 0.0001).

Table 25

Mean Differences in Resting Heart Rate

Mean	Std. Error
-6.443	1.022

Table 25 displays pre-exercise resting heart rate decreases.

Table 26

Differences in Resting Heart Rate Based on Gender

Gender	Mean	Std. Error
Female	-5.077	1.151
Male	-7.810	1.689

Table 26 demonstrates changes in the pre-exercise resting heart rate after training for each gender.

Table 27

Differences in Resting Heart Rate Based on Training Volume

Volume	Mean	Std. Error
Low	-7.744	1.701
High	-5.143	1.135

Table 27 demonstrates changes in the pre-exercise resting heart rate after training for each volume.

Table 28

Differences in Resting Heart Rate Based on Training Volume and Gender

Gender	Volume	Mean	Std. Error
Female	Low	-6.154	1.473
	High	-4.000	1.770
Male	Low	-9.333	3.066
	High	-6.286	1.419

The difference in resting heart rate after training was greatest for males with low volume, decreasing 9.33 beats per minute. Males with high volume also had large differences in resting heart rate (6.29 beats per minute), followed by females with low volume, who had an average decrease in resting heart rate of 6.15 beats per minute, while females with high volume experienced a decrease in resting heart rate of 4 beats per minute. These findings are shown graphically in Figure 4.

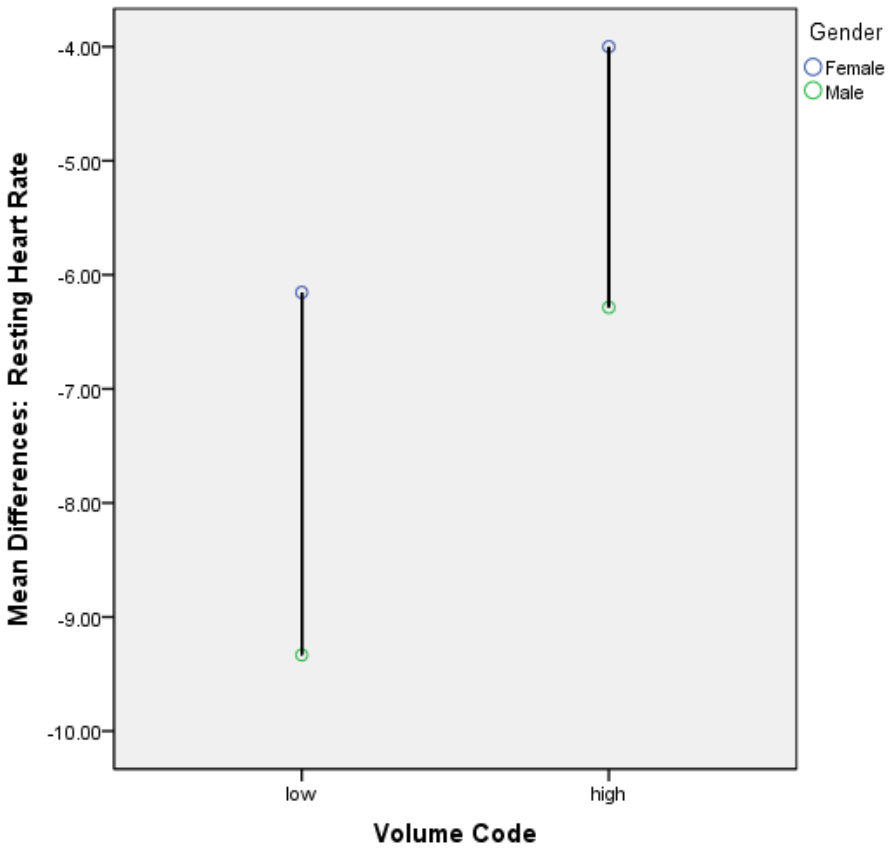

Figure 4. Differences in resting heart rate after training by gender and volume.

Table 29

Tests of Between-Subjects Effects: Differences in Resting Heart Rate Based on Training Volume and Gender

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	70.681[a]	3	23.560	.835	.484	.067
Intercept	1120.524	1	1120.524	39.726	.000	.532
Gender	50.386	1	50.386	1.786	.190	.049
Volume	45.640	1	45.640	1.618	.212	.044
Gender* Volume	1.348	1	1.348	.048	.828	.001
Error	987.216	35	28.206			
Total	2438.000	39				
Corrected Total	1057.897	38				

[a] R Squared = .067 (Adjusted R Squared = -.013).

As seen in Table 29, the overall corrected model shows no significance (F (3,35) = .835, p = .484), suggesting that none of the predictors makes a more significant contribution to the prediction of an individual's resting heart rate than the mean across all participants.

The results of the ANOVA also demonstrate that levels in volume, when we ignore the influence of gender, provided no predictive power in determining changes in resting heart rate after training (F (1,35) =1.62, p = .212). Figure 4 shown earlier explains these differences.

Table 30

Pairwise Comparisons: Differences in Resting Heart Rate Based on Training Volume

(I) Volume Code	(J) Volume Code	Mean Difference (I–J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Low	High	-2.601	2.045	.212	-6.751	1.550
High	Low	2.601	2.045	.212	-1.550	6.751

Note. Based on marginal means.

Table 30 shows that the overall difference in resting heart rate between participants with high and low volumes.

Anaerobic Threshold

Heart rates to estimate Anaerobic Threshold (AT) were collected on 84 subjects. No changes in AT estimates were observed after the 16weeks (p = 0.97). Female subjects (n = 55, mean AT 182.01 +-9.17 bpm), saw no change in AT performance (p = 0.467). Males (n = 29, mean AT 185.31 +- 10.13) additionally experienced no change in AT (p = 0.414).

Table 31

Mean Differences in AT Maximum Heart Rate

Mean	Std. Error	95% Confidence Interval	
		Lower Bound	Upper Bound
-.717	.982	-2.675	1.241

Table 31 shows AT maximum heart rate decreases.

Table 32

Differences in Maximum AT Heart Rate Based on Gender

Gender	Mean	Std. Error
Female	1.228	1.094
Male	-2.662	1.632

Table 32 demonstrates changes in the AT maximum heart rate after training for each gender.

Table 33

Differences in Maximum AT Heart Rate Based on Gender

Volume	Mean	Std. Error
Low	-1.048	1.641
High	-.387	1.080

Table 33 demonstrates changes in the AT maximum heart rate after training for each volume.

Table 34

Differences in Maximum AT Heart Rate Based on Training Volume and Gender

				95% Confidence Interval	
Gender	Volume Code	Mean	Std. Error	Lower Bound	Upper Bound
Female	Low	3.048	1.641	-.223	6.319
	High	-.593	1.447	-3.477	2.292
Male	Low	-5.143	2.843	-10.809	.523
	High	-.182	1.604	-3.378	3.014

The difference in maximum AT heart rate after training was greatest for males with low volume, decreasing 5.14 beats per minute. Females with low volume also had large differences in maximum AT heart rate, but in their case, the average maximum heart rate increased 3.05 beats per minute. For males and females with high volume, changes were much smaller. Females with high volume had an average decrease in maximum heart rate of .59 beats per minute, while males with high volume experienced a decrease in maximum heart rate of only .18 beats per minute. These findings are shown in Figure 5.

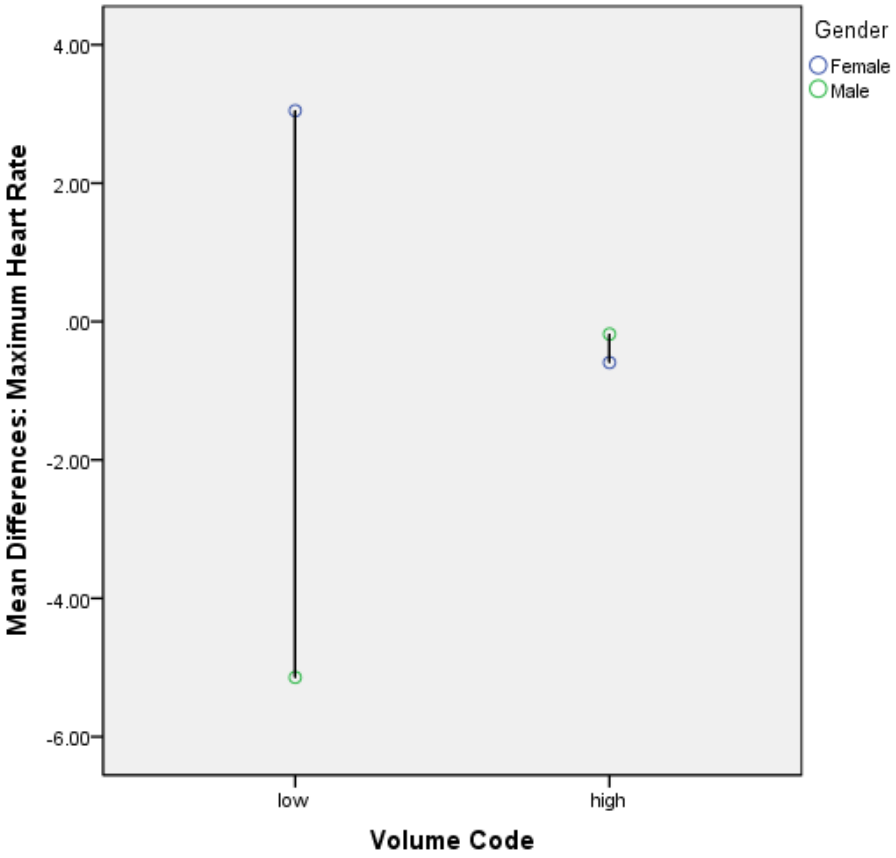

Figure 5. Differences in maximum AT heart rate after training by gender and volume.

Table 35

Tests of Between-Subjects Effects: Differences in Maximum Heart Rate Based on Training Volume and Gender

Source	Type III Sum of Squares	df	Mean Square	F	Significance	Partial Eta Squared
Corrected Model	389.568[a]	3	129.856	2.295	.085	.086
Intercept	30.168	1	30.168	.533	.468	.007
Gender	221.725	1	221.725	3.919	.051	.051
Volume	6.391	1	6.391	.113	.738	.002
Gender * Volume	271.027	1	271.027	4.791	.032	.062
Error	4129.601	73	56.570	.533		
Total	4520.000	77		3.919		
Corrected Total	4519.169	76				

[a] R Squared = .086 (Adjusted R Squared = .049).

As seen in Table 35, the overall corrected model shows no significance (F (3,73) = 2.295, p = 0.085), suggesting that none of the predictors makes a more significant contribution to the prediction of an individual's maximum AT heart rate than the mean across all participants.

The results of the ANOVA also demonstrate that levels in volume, when we ignore the influence of gender, provided no predictive power in determining changes in maximum AT heart rate after training (F (1,73) =0.113, p =0 .738). Figure 5 explains these differences further.

Table 36

Pairwise Comparisons: Differences in Maximum Heart Rate Based on Training Volume

(I) Volume Code	(J) Volume Code	Mean Difference (I–J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Low	High	-.660	1.965	.738	-4.576	3.255
High	Low	.660	1.965	.738	-3.255	4.576

Note. Based on estimated marginal means.

a. Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 36 shows that the overall difference in maximum heart rate between participants with high and low volumes was .66 beats per minute, but that the difference (p = .738) did not meet the statistical significance criterion of $p < .05$. Given the small average difference and the relatively high p-value, it is unlikely that a larger sample size would demonstrate a statistically significant difference between levels of volume. The results are presented graphically in Figure 5 shown earlier.

Percent Body Fat and Body Weight

Body fat measurements of subjects displayed significant improvements (p = 0.001). However, body weight data displayed no change from pre-to posttraining in all subjects. Male subjects (n = 30, mean %fat = 13.19 +-6.61) improved by 1.26 percent (p = 0.010), and collectively decreased body weight by 1.4 kgs (p = 0.089). Female subjects (n = 56, mean %fat = 26.69 +- 6.02) also decreased body fat measurements by 1.0 % (p =0.004), yet increased body weight by 1.2 kgs (p =0.123).

Table 37

Mean Differences in Body Weight

		95% Confidence Interval	
Mean	Std. Error	Lower Bound	Upper Bound
.541	.600	-.653	1.735

Table 37 shows body weight increases.

Table 38

Differences in Body Weight (Females)

		Paired Differences							
					95% Confidence Interval of the Difference				
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper	t	df	Sig. (2-tailed)
Pair 1	Post Weight (kg) - Pre Weight (kg)	1.17771	6.30656	.75378	-.32603	2.68146	1.562	69	.123

[a] Sex = F

Table 38 shows that on average across all female participants, the body weight increased by 1.18kg at the end of training.

Table 39

Differences in Body Weight (Males)

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Post Weight (kg) - Pre Weight (kg)	-1.35156	4.35645	.77012	-2.92223	.21911	-1.755	31	.089

[a] Sex = M

Table 39 shows that on average across all male participants, the body weight decreased by 1.35kg at the end of training.

Subjects stratified for a low training volume experienced a 0.85kg decrease in body weight. High-volume subjects produced 0.24kg increase in body weight. Training strata did not, however, produce statistically significant differences between low and high volume ($p = 0.612$). Body fat measurements for subjects stratified for low volume produced a 0.94% decrease and high-volume subjects decreased body fat by 1.0%. Training volume strata did not produce statistical differences ($p = 0.555$)

Table 40

Differences in Body Weight Based on Training Volume

Volume	Mean	Std. Error
Low	.847	1.020
High	.236	.634

Table 40 demonstrates changes in the body weight after training for each volume.

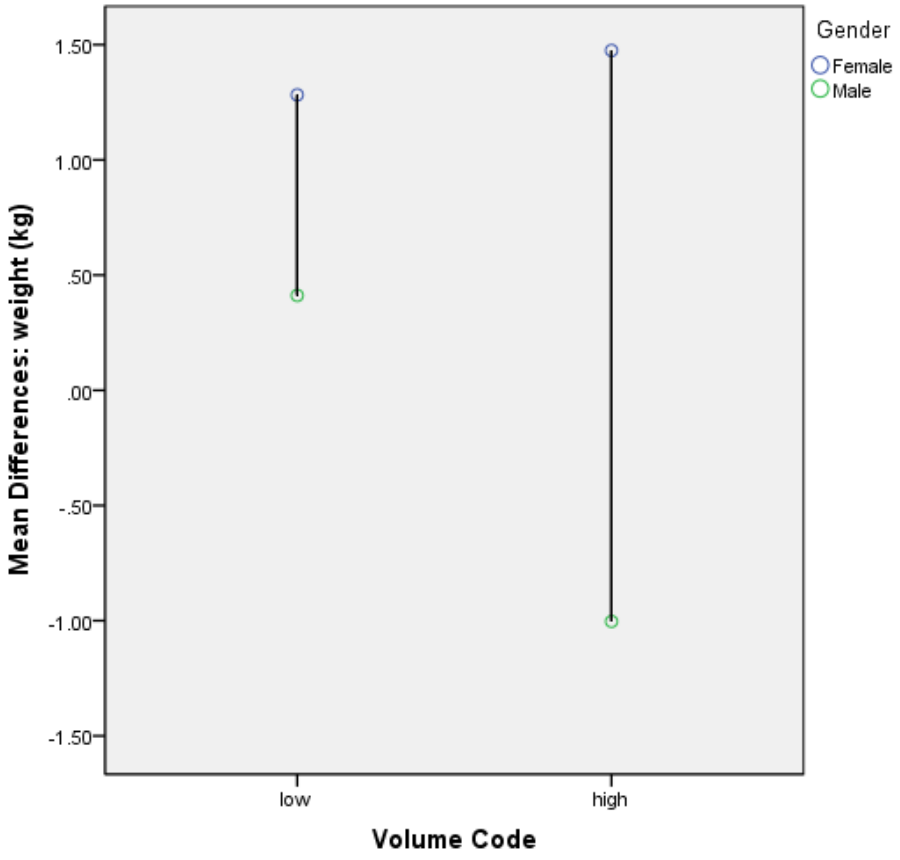

Figure 6. Differences in body weight after training by gender and volume.

Table 41

Tests of Between-Subjects Effects: Differences in Body Weight Based on Training Volume and Gender

Source	Type III Sum of Squares	df	Mean Square	F	Significance	Partial Eta Squared
Corrected Model	96.095[a]	3	32.032	1.435	.238	.049
Intercept	18.147	1	18.147	.813	.370	.010
Gender	43.398	1	43.398	1.944	.167	.023
Volume	5.777	1	5.777	.259	.612	.003
Gender * Volume	9.997	1	9.997	.448	.505	.005
Error	1874.862	84	22.320			
Total	2013.131	88				
Corrected Total	1970.957	87				

[a] R Squared = .049 (Adjusted R Squared = .015).
[b] Computed using alpha = .05.

As seen in Table 41, the overall corrected model shows no significance (F (3,84) = 1.435, p = .24), suggesting that none of the predictors makes a more significant contribution to the prediction of an individual's body weight than the mean across all participants.

The results of the ANOVA demonstrate that differences in gender, when we ignore the influence of volume, provided no predictive power in determining changes in body weight after training (F (1,84) =1.94, p = .167).

The results of the ANOVA also demonstrate that levels in volume, when we ignore the influence of gender, provided no predictive power in determining changes in Body Weight after training (F (1,84) =.259, p = .612).Finally, the ANOVA shows that

the interaction between volume and gender is not significant (F (1,84) =.448, p = .505), meaning that the effect of volume on body weight does not depend on the gender of the participant. Figure 6 above explains these differences further.

Table 42

Pairwise Comparisons: Differences in Body Weight Based on Training Volume

(I) Volume Code	(J) Volume Code	Mean Difference (I–J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Low	High	.611	1.201	.612	-1.777	2.999
High	Low	-.611	1.201	.612	-2.999	1.777

Note. Based on estimated marginal means

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 42 shows that the overall difference in body weight between participants with high and low volumes was .61 kg by the end of training, but that the difference (p = .612) did not meet the statistical significance criterion of $p < .05$. Given the small average difference and the relatively high p-value, it is unlikely that a larger sample size would demonstrate a statistically significant difference between levels of volume. The results are presented graphically in Figure 6 shown earlier.

Table 43

Mean Differences in Percent Body Fat

		95% Confidence Interval	
Mean	Std. Error	Lower Bound	Upper Bound
-.973	.323	-1.616	-.329

Table 43 shows percent body fat decreases.

Table 44

Differences in Body Fat (Females)

		Paired Differences							
					95% Confidence Interval of the Difference				
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper	t	df	Sig. (2-tailed)
Pair 1	Post Fat % - Pre Fat %	-.99643	2.51012	.33543	-1.66864	-.32421	-2.971	55	.004

[a] Sex = F

Table 44 shows percent body fat decreases (females).

Table 45

Differences in Body Fat (Males)

		Paired Differences					t	df	Sig. (2-tailed)
					95% Confidence Interval of the Difference				
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper			
Pair 1	Post Fat % - Pre Fat %	-1.26133	2.51075	.45840	-2.19886	-.32380	-2.752	29	.010

[a] Sex = M

Table 45 shows percent body fat decreases (males).

Table 46

Differences in Percent Body Fat % Based on Training Volume

Volume	Mean	Std. Error
Low	-.942	.545
High	-1.003	.347

Table 46 demonstrates changes in percent body fat after training for each volume.

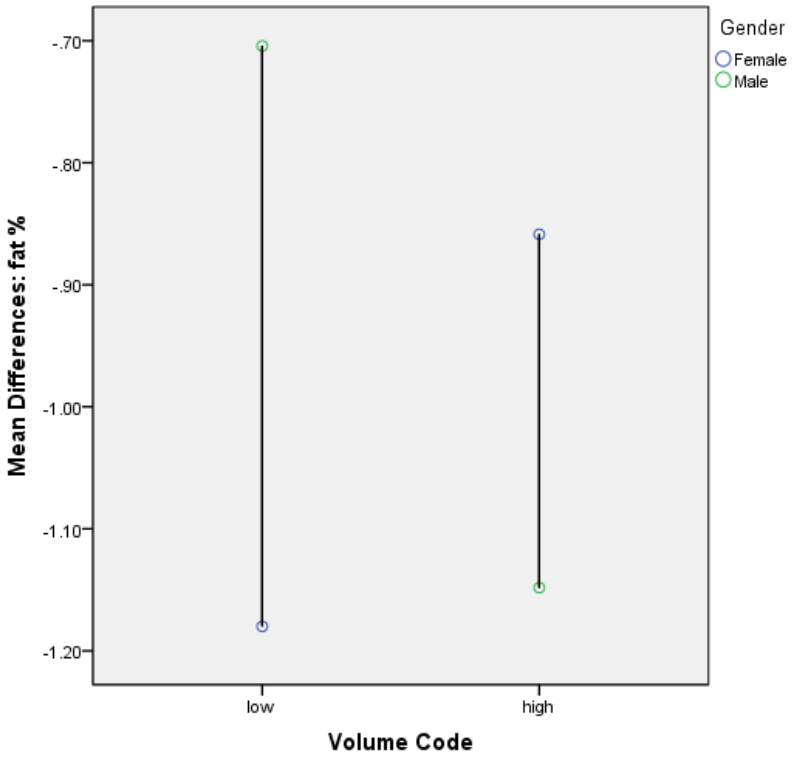

Figure 7. Differences in percent body fat after training by gender and volume.

Table 47

Tests of Between-Subjects Effects: Differences in Percent Body Fat Based on Training Volume and Gender

Source	Type III Sum of Squares	df	Mean Square	F	Significance	Partial Eta Squared
Corrected Model	2.336[a]	3	.779	.126	.944	.005
Intercept	55.909	1	55.909	9.071	.004	.108
Gender	.128	1	.128	.021	.886	.000
Volume	.055	1	.055	.009	.925	.000
Gender * Volume	2.163	1	2.163	.351	.555	.005
Error	462.276	75	6.164			
Total	545.301	79				
Corrected Total	464.612	78				

[a] R Squared = .005 (Adjusted R Squared = -.035).

[b] Computed using alpha = .05.

As seen in Table 47, the overall corrected model shows no significance (F (3, 75) = 0.126, p =0 .944), suggesting that none of the predictors makes a more significant contribution to the prediction of an individual's percent body fat than the mean across all participants.

The results of the ANOVA demonstrate that differences in gender, when we ignore the influence of volume, provided no predictive power in determining changes in Percent Body Fat after training (F (1, 75) =0.021, p = 0.886).

The results of the ANOVA also demonstrate that levels in volume, when we ignore the influence of gender, provided no predictive power in determining changes in

Percent Body Fat after training (F (1, 75) =0.009, p = 0.925). Figure 7 above explain these differences further.

Table 48

Pairwise Comparisons: Differences in Percent Body Fat Based on Training Volume

(I) Volume Code	(J) Volume Code	Mean Difference (I–J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Low	High	.061	.646	.925	-1.226	1.348
High	Low	-.061	.646	.925	-1.348	1.226

Note. Based on estimated marginal means.

[a] Adjustment for multiple comparisons: Least Significant Difference (equivalent to no adjustments).

Table 48 shows that the overall difference in percent body fat between participants with high and low volumes was .06 % by the end of training, but that the difference (p = .925) did not meet the statistical significance criterion of $p < .05$. Given the small average difference and the relatively high p-value, it is unlikely that a larger sample size would demonstrate a statistically significant difference between levels of volume. The results are presented graphically in Figure 7 shown earlier.

Correlational Measures

Pearson's Correlations measurements were performed and data were analyzed based on significance levels.

VO_{2max}

A significant negative correlation between VO_{2max} performance and body fat existed (p = 0.010) depicting as VO_{2max} improved, body fat percentages additionally improved. VO_{2max} and Wingate cycle ergometer PP shared no significant correlation (p = 0.447), however, $VO_{2max}$was significantly correlated to Wingate %D (p = 0.018), concluding that as VO_{2max}improved, as did the percent decrease in power over the 30-second power test. $VO_{2max}$and VJ shared a marginal positive correlation (p = 0.237) and $VO_{2max}$and BJ were significantly correlated (p = 0.020).

Table 49

Correlation Between $VO_{2max}$and Body Fat

		Differences: VO_{2max}	Differences: fat %
Differences: VO_{2max}	Pearson Correlation	1	-.282*
	Sig. (2-tailed)		.010
	N	83	82
Differences: fat %	Pearson Correlation	-.282*	1
	Sig. (2-tailed)	.010	
	N	82	86

*. Correlation is significant at the 0.05 level (2-tailed).

Table 49 demonstrates the relationship between VO_{2max} and % fat.

Table 50

Correlation Between VO_{2max} and Wingate % Decrease Peak Power

		Differences: VO_{2max}	Difference: % Decrease in Power
Differences: VO_{2max}	Pearson Correlation	1	-.258*
	Sig. (2-tailed)		.018
	N	83	83
Difference: % Decrease in Power	Pearson Correlation	-.258*	1
	Sig. (2-tailed)	.018	
	N	83	105

* Correlation is significant at the 0.05 level (2-tailed).

Table 50 demonstrates the relationship between$VO_{2max}$and %D.

Table 51

Correlation Between VO_{2max} and Broad Jump

		Differences: VO_{2max}	Difference: Broad Jump
Differences: VO_{2max}	Pearson Correlation	1	.403*
	Sig. (2-tailed)		.020
	N	83	33
Difference: Broad Jump	Pearson Correlation	.403*	1
	Sig. (2-tailed)	.020	
	N	33	39

* Correlation is significant at the 0.05 level (2-tailed).

Table 51 demonstrates the relationship between $VO_{2max}$and BJ.

Percent Body Fat

As described above VO_{2max} and body share a significant correlation ($p = 0.010$). Body fat also was significantly correlated to VJ performance ($p = 0.021$). However, body fat and BJ shared very little relationship ($p = 0.711$). Additionally, body fat shared no relationship with PP ($p = 0.437$), yet subjects experienced marginal relationships in body fat and %D ($p = 0.194$), depicting as body fat improves, as does %D power production.

Table 52

Correlation Between Body Fat and Vertical Jump

		Differences: fat %	Difference: Vertical Jump
Differences: fat %	Pearson Correlation	1	-.266*
	Sig. (2-tailed)		.021
	N	86	75
Difference: Vertical Jump	Pearson Correlation	-.266*	1
	Sig. (2-tailed)	.021	
	N	75	91

* Correlation is significant at the 0.05 level (2-tailed).

Table 52 demonstrates the relationship between %fat and VJ.

Chapter 5

Discussion

The primary purpose of this research was to investigate the effects of aerobic training on anaerobic output defined by, vertical jump (VJ), broad jump (BJ), and Wingate cycle ergometer, in male and female recreationally trained runners over a period of 16 weeks. Additionally, body weight (BW), body composition (PF), pre-exercise resting heart rate (RHR), $VO_{2max}$and anaerobic threshold (AT) were examined as exploratory measures. The primary findings demonstrated that after 16 weeks of aerobic training, there was no change in vertical jump, an increase in broad jump, no change in Peak Power (PP), and an improvement in percent decrease (%D) during the 30-second Wingate cycle tests collectively among study subjects. In addition, increases in $VO_{2max,}$ as well as decreases in percent body fat and pre-exercise resting heart rate were observed.

Vertical and Broad Jump

The present 16-week aerobic training period, despite the lack of any consistent concurrent anaerobic training, produced novel results. VJ scores of all subjects (n = 87) displayed no difference after the 16-week aerobic training protocol (p = 0.307). For meaningful sport performance application, VJ data were stratified based on pretest performance measurements. Pretest jump scores for each subject were categorized into "below average" height (male VJ < 16 inches, female VJ < 12 inches) "average" (male VJ = 16.5–20 inches, female VJ = 12–16 inches) and "above average" (male VJ > 20.5 inches, female VJ > 16.5; Chu, 1996). The subjects with below average VJ (n = 12) experienced a decrease in jump height (p = 0.001) as a result of aerobic training. Subjects

with average VJ scores (n = 62) saw no change in their jump height (p = 0.582). And interestingly, above average subjects (n = 13) saw increases in their VJ heights after the aerobic training (p = 0.001) with a 1.45-inch increase. These results appear similar to Sale et al. (1990), when researchers found no difference in lower extremity strength when comparing a strength only group of subjects to an endurance only group during 22 weeks of training. However, the results do not fully support the findings of Hennessy and Watson (1994), Dudley and Djamil (1985), Bell et al. (1991), and McCarthy et al. (1995) which also displayed no changes in in lower body strength output; however, concurrent anaerobic and aerobic conditions were structured in each study. It should be noted that, compared to the present study, only Hennessy and Watson (1994) measured jumping performance as a dependent variable in 56 rugby subjects. The other above mentioned studies evaluated lower extremity strength, particularly 1-RM leg press strength and knee extension strength after a period of concurrent aerobic and strength training.

Similar to VJ results, BJ performance expressed nearly identical results. The current study displayed an increase in BJ measurement collectively in all 38 subjects after aerobic training. Subjects were again stratified based on their pretest BJ performance Subjects with average (n = 18) and above average (n =14) BJ measurements, experienced increases in BJ scores (average jump–p = 0.08, above average– p = 0.041). While subjects with pretest BJ scores below average (n = 6) experienced a decrease in BJ (p = 0.0001).

The present study displays that the subjects with the lowest anaerobic jump scores entering the study experienced the greatest negative affect of aerobic training.

Conversely, the subjects with average or above average jumping ability prior to the 16-week aerobic training period experienced the most interesting effects. Both stratums increased in BJ scores. Average subjects saw no change in VJ, yet above average subjects increased their VJ. This is of particular importance to athletic populations and sports performance. Most anaerobic athletes would most likely be categorized in average and above average stratum. The current study appears to develop a theory that the greater the anaerobic capability of an athlete, the lesser effect aerobic conditioning has on anaerobic performance. The data also appears to lead to the inverse, suggesting an athlete with below average anaerobic jump performance may encounter larger unfavorable adaptions to aerobic training especially at higher weekly volumes. Furthermore, PF was expectedly decreased as a result of the aerobic training period as was an increase in VO_{2max}. VJ and BJ performance were strongly correlated to PF and VO_{2max} (VO_{2max} and BJ $r = 0.403$, $p = 0.020$; $VO_{2max}$and VJ $r = 0.141$, $p = .237$; PF and VJ $r = 0.266$, $p = 0.021$). Meaning, as VO_{2max}improved, there was a significant statistical enhancement with BJ ($p = 0.020$) and a positive correlation to VJ ($r = .141$). Similarly, as PF decreased, VJ was significantly increased ($p = 0.021$). The significance and strength of each relationship also appear to support the present theory limiting the effect aerobic training has on athletes especially those with greater anaerobic dispositions. These findings appear to support results published by Stauffer, Nagle, Goss, and Robertson (2010), when assessing jump tests in basketball players as well.

Wingate Power Analysis

Anaerobic power via Wingate cycle ergometer scores displayed novel results undocumented by previous literature. Subjects (n = 15) experienced minimal decreases in peak power (PP) levels after 16weeks of aerobic training, however, the decrease was not significant when stratified for gender (male – p = 0.080, female – p = 0.114). Both males and females saw significant improvements in peak power percent decrease (male – p = 0.017, female – p = 0.006). From a physiological standpoint, peak power decreases may be stemming from potential neuromuscular considerations. Bell et al. (2000) concluded that decreased myofibrillar ATPase activity was an interfering factor in university students when investigating 12-week strength and endurance measures. Endurance trained subjects did not possess as high ATPase activity as did the strength only and concurrent group when muscle biopsies were taken from the vastus lateralis muscle. The aerobic activity in the present study may have not recruited greater heavy chain muscle types (IIa and IIx) as speculated by Casey et al. (1996). The lack of any explosive or peak power training, therefore, recruiting heavy chain muscle types, may have been a contributor to decreased PP. Neuromuscular speculation may be apparent in the present study, however, muscle morphology, and metabolic enzyme activity was not documented in the subjects to make conclusive estimations. More apparent in the present study, may be the limited sample size as a larger contributing factor to decreases in PP among subjects. However, confounding the PP results from the present study were the VJ and BJ increases, particularly the effects on subjects with greater anaerobic capacities. Recently, however, limited sports performance application may be present with PP measures.

Literature by Stauffer, Nagle, Gross, and Robertson (2010), demonstrated insignificant and weak correlations with PP Wingate scores and BJ, Overhead Throw and 30 meter sprint times in Division I basketball players. Hoffman, Epstein, Einbinder and Weinstein (2000), concluded comparable results when investigating PP Wingate scores and 30-second sprint speed and VJ. Hoffman et al. (2000) found insignificant correlation in PP and 30-second sprint speed as well as marginal correlations between PP and VJ when researching 17-year-old national basketball players. The increasing amount of published sports performance data not only highlights a potential threat to the external validity of the Wingate ergometer PP test in some populations, but also leads to support the present studies finding where PP was not found to be significantly related to VJ or BJ (BJ and PP r = .275; BJ and PP r = 0.281). Furthermore, McLester, Green, Wickwire, and Crews (2008), concluded the only significant correlation that existed with PP was body fat percentage when studying male college aged students. Although not significantly supported by the present study, this notion would, however, indicate that the possession of greater relative body fat would decrease power production, be it Wingate PP values or VJ given the above significant correlation of PF and VJ.

Fatigue was also calculated from the Wingate test. As previously stated, subjects experienced improvements in peak power percent decrease (%D) after the 16 weeks (p = 0.006). Potentially, a more predictive performance measure for sport than the Wingate PP value is the Wingate %D. A vital key in sport performance is the prevention and minimization of fatigue. In the present study, evidence supports the contribution of aerobic capacity and the ability to sustain anaerobic power and minimize the decrease in

power. Collectively, subjects improved VO_{2max} as well as %D. Moreover, results additionally presented a correlation of $VO_{2max}$and fatigue (r = 0.258, p = 0.018). Not only did subjects improve %D after 16 weeks, but also those possessing greater VO_{2max}improvements also significantly improved their %D measurements. One previous study could be identified that looked at relationships between VO_{2max} and fatigue variables in the Wingate cycle test. Koziris et al. (1996) discovered a strong relationship between aerobic power (VO_{2max}) and Wingate fatigue in recreational subjects. The same results were replicated in female subjects. The results of Koziris et al. (1996) and the present study support the contribution of the aerobic energy system with increasing anaerobic power capacity and the minimization of fatigue. Evidence would suggest (Bogdanis, Nevill, Lakomy et al., 1996; Laurent et al., 1992; McCully et al., 1989; McCully et al., 1992; McMahon & Wenger, 1998; Takahashi et al., 1995; Tomlin & Wenger, 2001; Yoshida & Watari, 1993). As supported by the present study, in many anaerobic sport settings, involving repeated efforts of shorter durations without adequate recovery, athletes may improve performance and decrease fatigue when aerobic measures are improved. Previous literature concluded that aerobic metabolic pathways contribute to an increasingly larger portion of ATP when high-intensity bouts are repeated consecutively (Bangsbo, Krustrup, Gonzalez-Alonso, & Saltin, 2001; Gaitanos, Williams, Boobis, & Brooks, 1993; Putman, Matsos, Hultman, Jones, & Heigenhauser, 1999). Bangsbo et al. (2001) and McLester et al. (2008) found aerobic mechanisms provide large amounts of ATP after the first bout of anaerobic exercise, and therefore an enhanced aerobic capacity would be beneficial during repeated maximum intensity bouts.

In addition, if aerobically trained subjects are capable of greater lactate clearance (Bassett, Merrill, Nagle, Agre, & Sampedro, 1991), there is potential for attenuated diminishing of force in repeated bouts. Though lactate was not assessed during this investigation, it can be theorized that blood lactate levels can reach extreme levels during the Wingate test. Research has documented that H+ concentration due to lactate accumulation contributes to fatigue (Fitts, 1994; Powers & Howley, 2004), which is associated with decreased force production. Therefore, it can be justified that a greater lactate clearing capacity, via oxidative metabolism, should lead to improved performance and power production in repeated bouts.

It is interesting that all of the correlations with significance as well as the matched pairs analysis around power production and power sustainment (BJ, VJ, PP %D) and the influence of aerobic capacity (VO2) max and body fatness (PF). These data support the relationship of body fat and performance whether it be anaerobic or aerobic in nature. Similarly, aerobic capacity is advantageous in decreasing fatigue and has an insignificant impact on power production. Moreover, aerobic training only appeared detrimental to those with below average anaerobic output and for those with average to above average jumping ability. It would be speculation that aerobic training may be enough of a neuromuscular stimulus to elicit positive outcomes and no disadvantages. However, greater aerobic fitness was required when anaerobic power production sustainment was assessed (Wingate peak power percent decrease) leading to the theory that greater aerobic fitness is a vital component in longer anaerobic or repeated bouts.

VO_{2max} and Pre-Exercise Heart Rate

VO_{2max} measurements were marginally improved in all subjects ($p = 0.055$). Male subjects displayed significant improvement ($p = 0.001$), however, female subjects showed no improvement in VO_{2max}. ($p = 0.9$). Female subject outliers are speculated to have heavily influenced the analysis. The improved body composition in women may have been offset by lack of weight loss and complicated by weight gain.

Pre-exercise heart rate measurements did improve significantly ($p = 0.001$) for the total group and individually in both male and female stratum (male p– 0.001; female p = 0.001). Although this data do not fully statistically support the hypothesis stated, and traditionally what may be physiologically theorized (Brooks et al., 2005), VO_{2max} improvements were still supported in the correlational analysis. VO_{2max} was significantly correlated with improved body composition (PF; $p = 0.010$), BJ ($p = 0.020$) and Wingate %D ($p = 0.018$), and was marginally related to VJ ($r = .237$). This leads to the notion that the improvement of VO_{2max} has the potential to create enough physiological stimuli to improve aerobic and anaerobic performance. Of critical interest is the link between VO_{2max} and fatigue (%D) leading to the potential for improved recovery. As mentioned above, aerobic mechanisms play very critical roles in ATP regeneration as well as expediting the removal of byproducts. Muscle mitochondrial density is additionally improved with aerobic training, which supports aerobic metabolism. Research by McMahon and Wenger (1998), as well as Tomlin and Wenger (2001), cite that those athletes with higherVO_{2max} values have enhanced abilities to resynthesize PCr after repeated high-intensity exercise bouts. Tomlin and Wenger (2001) also suggest that a

greater VO_{2max} leads to increased recovery from multiple anaerobic bouts due to the following mechanisms: a higher aerobic response to excess post exercise VO_2; greater and more efficient lactate removal; and improved PCr regeneration.

VO_{2max} and heart rate recovery has additionally been linked as a mechanism to increased recovery. Hoffman, Epstein, Einbinder, and Weinstein (1999) cited strong predictive relationship between VO_{2max} and heart rate recovery when evaluating successive bouts of repeated line running drills in basketball players. The present study would support those results. Current study subjects experienced significant correlations between post-VO_{2max} and post-RHR (r = -0.45). Subjects with greater post-VO_{2max} measures appear to possess lower postexercise heart rate values. The likely mechanism is the increased vagal activation and modulation (Goldsmith et al., 1997; Goldsmith et al., 2000). Current research has demonstrated that with improved oxygen capacity (VO_{2max}), vagal activity is often substantially higher leading to not only lower resting heart rate values, but additionally, improving sport performance by improving heart rate deceleration. Heart rate deceleration may not only support the physiological function of increased recovery, but improving heart rate deceleration may contribute to a greater psychophysiological component in sport. The psychophysiological ability to manage heart rate and heart rate activity is not only beneficial for greater athletic performance, but may also lead to great attention capacity to a salient stimulus, increased reaction time and overall greater cognitive function and activity (Jennings & Wood, 1977; Lacey & Lacey, 1978). The present study results and the correlation of a greater $VO_{2max}$with a

lower RHR would lead to support the findings linking aerobic capacity with lower heat rates and also greater heart rate deceleration.

Body Composition and Body Weight

Body composition evaluation produced interesting results. Collectivity, subjects displayed significant improvements in percent fat measurements ($p = 0.001$). Most intriguing were the overall results as they relate to body weight. The findings demonstrate that female subjects showed statistically significant percent body fat reductions ($p = 0.004$), yet failed to produce similar reductions in body weight ($p = 0.123$). The differences in body fat and body weight/body mass index most likely stem from gains in muscle mass and the additional water weight with heighted mitochondrial and glycogen storage. These results align with the adaptations documented by Greiwe et al. (1999) and Hickner et al. (1997), with their evaluations of muscle glycogen accumulation after aerobic exercise. Muscle glycogen concentration, muscle GLUT-4 content, and glycogen synthase activity were all heightened when evaluating both trained and untrained individuals. The repeated depletion of glycogen stores as a result of aerobic training most likely results in super-compensation of glycogen storage. Although not measured in the current study, research would lend to the notion of an increase in muscle glucose transport protein-4 (GLUT-4), as well as a greater muscular capacity for glycogen storage (Kraniou et al., 2004). The difference in this adaptation in men and women is remarkable. The lack of significant weight change in women may be an evolutionary adaptation through compensatory eating to maintain fat stores needed for reproduction.

The evidence from this research would suggest that body weight is not an appropriate measurement tool, particularly in the early stages of an intervention or cardiovascular protocol in healthy populations, specifically to fitness gains. The study population displayed insignificant reductions in body weight, and many female subjects increased body weight. However, reductions in percent body fat measures were statistically significant in both men and women. The offsetting physiological adaptions described above to the aerobic training protocol were most noted in the female subjects.

Volume Calculations

Weekly running volume stratification was conducted via subject provided training logs recording weekly training volume in mileage on the dependent variables. Low volume runners were defined as participating in <20 miles per week based on 16-week averages. High-volume subjects were operationally defined as training >20 miles per week based on 16-week averages. Although no significant differences existed among any of the outcome variables ($p > 0.05$) as a predictive means to determine a change based on training volume, meaningful data could still be gathered. VO_{2max}among low volume subjects, both male and female displayed the greatest improvements (female = 1.35 ml/kg/min, male 2.714 ml/kg/min) when compared to high-volume subjects. Although the results were not found to be statistically significant at the $p < 0.05$ level, the findings would align with previous research citing lower volume and intensity training is sufficient for improved oxygen transport (Myers, 2003). Moreover, extreme mileage and training amounts do not appear to be necessary for greater oxygen utilization and cardiovascular function. Additionally, pre-exercise resting heart rate readings showed a

greater reduction in low volume runners (male and female) as well as greater weight loss reductions when compared to their high-volume counterparts. These results would lend to support previous public health research that demonstrates greater plasma volume and increased stroke volume are elicited in as little as 10 weeks upon the inception of cardiovascular training even at low levels of training (Wilmore & Costill, 2004). It is, however, speculated by the researchers that the low volume subjects entered the study with a lesser aerobic capacity and training exposure, therefore, creating a greater capacity for adaptation within the 16-week training protocol. The diminished returns training principal would then apply to the high-volume subjects, citing that their previous training history and aerobic predispositions would lend to less marked improvements in these areas. Again, although these findings were not found to be statistically significant, the results would certainly lend to future research initiatives. The lack of high intensity or interval training most likely contributed to less physiological change in the higher mileage group. A limitation of this research was the lack of a control group, understanding that the minimal stimulus was 20 miles per week.

Significance of the Results

The study's primary hypothesis was supported. The study context, aerobic training did not lead to any diminished anaerobic performance results and in some areas of the study sample, aerobic training appeared to provide sufficient training stimulus to elicit positive anaerobic performance results.

The most significant outcome form this study was that of jump performance. Specifically, subjects with below average jumping capacity in both VJ and BJ were the

only group negatively affected by aerobic training. All other stratum (average and above average) were either unaffected or their anaerobic jump performance was subsequently improved. Similarly, the correlational analysis of jump performance and PF and VO_{2max} produced novel insight. The strength and statistical significance of the positive relationship between VJ and BJ performance and VO_{2max} and PF support a theory that as one reduces percent body fat and improves their aerobic capacity, anaerobic performance is consequently improved. These two primary results suggest that in athletes with average to above average anaerobic output, aerobic training does not appear to interfere with rate of force production. Additionally, and the reduction of body fat and an improved aerobic capacity may contribute to improve the rate of force production.

Wingate power analysis provided significant results as well. Although average PP performance was decreased as a result of aerobic training, the minimal decrease was not enough to support any statistical significance ($p > 0.05$). Wingate peak power %D was, however, statistically improved after the aerobic training. This would support a theory that aerobic training may lead to delayed fatigue, a particular performance criteria in anaerobic sport. Additionally, the statistically significant correlational analysis of %D and VO_{2max} would also support the need for the anaerobic athlete to add aerobic training in the combating of fatigue, specifically for repeated anaerobic high intensity bouts.

Body weight as it relates to body composition additionally produced meaningful results. Female subjects did not produce statistically significant reductions in body weight after the 16-week protocol. However, females did show significant improvements in total percent body fat. The present study would suggest that the use of body weight might not

be an effective assessment tool in the beginning stages of anaerobic training program specific to a health profile, specifically in healthy female populations. Moreover, weight loss appears to be an impractical motivating factor to measure gains in overall health, especially form a cardiovascular standpoint.

Finally, although the stratification of study subjects based on weekly volume-running logs did not present any statistically significant results, the data collected appears somewhat to steer momentum towards low volume training as a more effective means in overall aerobic body composition adaptations in fairly sedentary populations. Although results were unclear and insignificant and researcher speculation about the low volume subjects initial training history and fitness levels, the results would suggest future research specific to training volume or an alteration in intensity would provide meaningful data as it pertains to overall gains in health.

Limitations

The most critical limitation is the lack of a control group. The present study does not have a neutral control to compare the results. The major threat to adding a control group is the ability to appropriately define the controls and the ability to accurately access their anaerobic and aerobic training protocols and volumes. However, a control group would increase the validity in determining aerobic training's effect on anaerobic output.

Another limitation for the present study is the sample population. The sample selected were students that were enrolled in PE 1262 Marathon Training at the University of Minnesota in the spring semesters of 2009, 2010 and 2011. None of the subjects currently participate in anaerobic sport competition. There is also a relatively wide

anaerobic and aerobic performance range of study subjects. However, all study subjects were healthy, and upon the inception of the study possessed a history of aerobic training, fulfilling a prerequisite for the study. Study subjects accurately fit the title "recreational runners." Provided the above limitation in the present study, researchers stratified for pretest anaerobic performance as a means to increase the external validity of the research. As described, pretest VJ and BJ measurements were used to stratify participants for further analysis with the intention of categorizing groups. Researchers were able to identify above average, average and below average anaerobic performance thresholds to determine the effects of aerobic training on different levels of anaerobic differentiation among the subjects.

Training volumes were quantified and participants were stratified for high or low weekly training volumes based on self-reported mileage. Regardless of stratum, weekly training volume is vastly higher than those measuring anaerobic output in previous research. Low mileage runners were defined as participating in <20 miles per week based on 16-week averages. High-volume subjects were operationally defined as training >20 miles per week based on 16-week averages. Volunteerism of subjects may pose motivational bias as well.

Improvements

An improvement to the current study includes the use of a control group. As mentioned above, the present study lacks a control group to measure the magnitude of the findings. The single most important improvement would be to accurately define a control group to match the aerobic training group.

A second improvement to this study would be the use of predetermined training volumes. Subjects were required to self-report training volume based on mileage and time. However, predetermining equivalent subject training volume stratum would provide greater control over aerobic volume and potentially clearer and more meaningful results particularly to the anaerobic athlete. The use of accelerometers or GPS tracking devises would have given more precise values specific to training volumes and intensities.

An additional improvement to the current study would be to expound upon the pretest anaerobic stratification of the jump performance. The use of force plates would control for any countermovement in the vertical jump. The lack of control for the countermovement before the jump would be a substantial limitation in this research. The present study created stratum based on pretest jumping capacity, however, equal strata was not accomplished. Future areas of study would benefit from equivalent stratum based on anaerobic performance as measured by different assessments.

Finally, controlling for and administering concurrent anaerobic (strength) training may have provided greater insight especially to an athletic population. Much of the research to date evaluating the effects of aerobic training on anaerobic output has resided under the umbrella of concurrent training. Most research has outlined the effects of aerobic conditioning on anaerobic performance while subjects are concurrently participating in both forms of training as would be expected during in-season sports performance. This controllable factor may lead to greater insight to the effect of the training.

Future Research

Future research should divulge into two areas: sports performance and public health. From a sports performance aspect, future studies would benefit from utilizing sports specific athletes and evaluating the effects of aerobic training on various sport specific anaerobic performance measures. Research gaps would continue to be filled with the use of concurrent training protocols and modalities. As outlined in the present study, incorporating the use of greater externally valid sports performance measures such as VJ and BJ, as well as other on field actions as opposed to stand-alone strength measures that are presented in previous research would continue to evolve sports performance research and enhance the inference of the results. Lactate level evaluation would also be a direction for future research that would provide insight to anaerobic athletes. Weekly training volume and pretest anaerobic performances stratification as mentioned above would be additional areas of improved research.

From a public health stance a greater emphasis on the effects of resting values as well as post exercise and EPOC metabolic data would provide novel results. Controlling for resting and pre-exercise heart rate and metabolic activity as well as post bout and EPOC metabolism would be critical areas of continued research. Additionally, controlling for training volumes and intensities would provide insight for exercise prescription in various populations.

Conclusion

There are no parametric indicators that aerobic training leads to any attenuation or potentiation in the ability to produce anaerobic force. Moreover, in the present study,

some stratum appeared to be physiologically stimulating enough through aerobic training to experience heightened anaerobic output. Only when stratified for anaerobic jump performance was any interference present in below average populations. Conversely, those subjects with average to above average anaerobic output experienced no change or even increased jump performance after the 16weeks of aerobic training. Based on the evidence of this research, jump performance was also significantly correlated with VO_{2max} and PF, leading to the idea that in an anaerobic setting, as an athlete improves both their VO_{2max} and body composition, their anaerobic performance is subsequently improved. This theory was further supported in the Wingate cycle ergometer analysis citing the subjects with greater VO_{2max} performances and lower percent fat numbers were correlated with improved peak power percent decrease performance.

Body weight data may additionally be a misleading assessment tool when measuring health gains during the first few months of an activity intervention in healthy populations. The present aerobic conditioning protocol did not illicit weight loss in female populations. However, percent body fat did decrease as a result of the 16-week program. The gains in muscle mass and mitochondrial adaptation that are generally a result of aerobic training are the likely factors to the presented results particular to women. Therefore, the use of body weight as a motivator for weight loss and overall health may be an impractical assessment tool. The use of percent body fat appears to be a more reliable measure; however, the practicality and availability of such assessment are difficult.

Finally, the present data do not support the notion of the universal nature that aerobic training has an interference effect in the development of rate of force production. However, the interference effect may hold true in regards to explosive strength with limited neural activation of trained tissues unmeasured in the current study. The interference effect may also be present in a concurrent setting where aerobic and anaerobic training protocols are assessed against anaerobic only subjects. However, in a sport performance setting as well as public health, the training for physical fitness requires the development of muscle strength, power, and endurance, which the present findings indicate that the influence of aerobic training is an important factor for not only overall fitness, but does not appear to constrain anaerobic performance.

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Appendix A

IRB Exemption

UNIVERSITY OF MINNESOTA

Research Exempt from IRB Committee Review Category 4:

EXISTING DATA: RECORDS REVIEW & PATHOLOGICAL SPECIMENS

Route this form to:	U Wide Form: UM 1573
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Submission Instructions:
E-mail a copy of this application and any other materials required to the Research Subjects' Protections Programs Office: RSPPeRev@umn.edu

Electronically submitted protocols must be sent from a University of MN e-mail account. Original signatures are not required. U of M x.500 IDs have been deemed by the University of Minnesota to constitute a legal signature.

Academic Advisors and Co-Investigators should be carbon copied (Cc) on the submission e-mail.

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see http://www.research.umn.edu/irb/download/ or call 612-626-5654

1.1 Project Title (Project title must match grant title. If different, also provide grant title):

Rate of Force Production Under Aerobic Conditioning

1.2 Principal Investigator (PI)

Name (Last name, First name MI): Carroll, Christopher, K	M.Ed.
5550 Chatsworth St. N Shoreview MN, 55126	651-503-2459
	Pager or Cell Phone Number: 651-503-2459
	Fax:
U of M Employee/Student ID: 2516330	carro194@umn.edu
Carro194	Kinesiology
Occupational Position: ☐Faculty ☐Staff ☒Student ☐Fairview Researcher ☐Gillette Researcher ☐Other:	
Human Subjects Training (one of these must be checked--refer to training links at the end of this section): ☒ CITI , ☐ Investigator 101, ☐ NIH training (EXCEPT for 5/8/06 to 2/29/08), ☐ UM/RCR (between 1994-2003) ☐ Other - Indicate training received, when and from which institution:	HIPAA Training (Required if Data Contains PHI): ☐ HIPAA
As Principal Investigator of this study, I assure the IRB that the following statements are true: The information provided in this form is correct. I will seek and obtain prior written approval from the IRB for any substantive modifications in the proposal, including changes in procedures, co-investigators, funding agencies, etc. I will promptly report any unexpected or otherwise significant adverse events or unanticipated problems or incidents that may occur in the course of this study. I will report in writing any significant new findings which develop during the course of this study which may affect the risks and benefits to participation. I will not begin my research until I have received written notification	

of final IRB approval. I will comply with all IRB requests to report on the status of the study. I will maintain records of this research according to IRB guidelines. The grant that I have submitted to my funding agency which is submitted with this IRB submission accurately and completely reflects what is contained in this application. If these conditions are not met, I understand that approval of this research could be suspended or terminated.

carro194	8/1/11
x.500 of PI	Date

Training Links:
FIRST (Fostering Integrity in Research, Scholarship and Training):
http://cflegacy.research.umn.edu/first/humansubjects.htm
HIPAA: http://www.research.umn.edu/first/AdditionalCourses.htm
- "UM/RCR" includes all human subjects protection training offered in-person or online at the University of Minnesota from 1994-2003.
- The online NIH tutorial offered during the period May 8, 2006-February 29, 2008 is NOT acceptable to meet this requirement.
- If you completed a version of this training not included on the list provided, provide details as indicated
- The University of Minnesota uses two methods to verify records about completion of human subjects protection training: 1) training registration online, or 2) researcher must provide copy of completion certificate. To check your online training record, go to http://www.research.umn.edu/first/Reports.htm

1.3 Department, Division Head, or Dean Information
Please note as the researcher, you are responsible for confirming and following your departmental standards and requirements for research.

Li LiJi, Ph. D.
Name of Department Head, Division Head, or Dean

1.4 Are there additional Co-Investigators and Staff?
☐ Yes. Download an extra personnel sheet and include it with your application.
☒ No. Continue to 1.5.

1.5 Is the PI of this research a student?
☐ Yes. *Include Appendix J.*
Electronically submitted protocols must be carbon copied (Cc) to their advisor.
☐ No. Continue to 2.

Academic Advisor to the Student Investigator	
Advisor's Name (Last name, First name MI): Stacy Ingraham	Kinesiology
Mailing Address: 221A CookeH 1900 University Ave SE Minneapolis, MN 55455	Phone Number: 612-626-0067
	Email: ingra013@umn.edu
	U of M x.500 ID (ex. smith001): Ingra013

2. Funding

2.1 Is this research funded by an internal or external agency?
☐ Yes. *Include Appendix A.*
☒ No.

If no, explain how costs of research will be covered:

The costs of the research will be funded by the Principal Investigator, Christopher Carroll.

3. Institutional Oversight

3.1 Will this research be utilizing Fairview Health System resources or medical records?

☐ Yes.
☒ No.

3.2 Will this research be utilizing Gillette Children's Specialty Healthcare or medical records?

☐ Yes.
☒ No.

3.3 Is this research proposal being reviewed by any other institution or peer review committee?

☐ Yes. It is the responsibility of the PI to secure the appropriate approval from these committees and document that approval to the IRB. Attach a copy of documentation of approval, if received, and indicate committees below.
☒ No.

If yes, then please list which committees will review this proposal:

4. Conflict of Interest

Federal Guidelines emphasize the importance of assuring there are no conflicts of interest in research projects that could affect the welfare of human subjects. Reporting of financial interests is required from all individuals responsible for the design, conduct or reporting of the research. If this study involves or presents a potential conflict of interest, additional information will need to be provided to the IRB. Examples of conflicts of interest may include, but are not limited to:

- A researcher participating in research on a technology, process or product owned by a business in which the researcher or family member holds a significant financial interest or a business interest
- A researcher participating in research on a technology, process or product developed by that researcher or family member
- A researcher or family member assuming an executive position in a business engaged in commercial or research activities related to the researcher's University responsibilities
- A researcher or family member serving on the Board of Directors of a business from which that member receives University-supervised Sponsored Research Support
- A researcher receiving consulting income from a business that funds his or her research
- A researcher receiving consulting income from a business that could benefit from the results of research sponsored by a federal agency (i.e. NIH)

"Family Member" means the covered individual's spouse or domestic partner, dependent children, and any other family member whom the covered individual reasonably knows may benefit personally from actions taken by the covered individual on behalf of the University.
"Business Interest" means holding any executive position in, or membership on a board of a business entity, whether or not such activities are compensated.

For additional details and definitions, please refer to the appropriate policy:
University of Minnesota Researchers, please refer to:
http://www.policy.umn.edu/Policies/Operations/Compliance/CONFLICTINTEREST.html

University of Minnesota Researchers involved in clinical health care in the Academic Health Center, also refer to:
http://www.policy.umn.edu/Policies/Operations/Compliance/CONFLICTINTEREST_APPA.html

Fairview Health System Researchers, please refer to:
http://www.fairview.org/Research/index.htm

Gillette Children's Specialty Healthcare Researchers, please refer to:
http://www.gillettechildrens.org/

4.1 Do any of the Investigators or personnel listed on this research project have a business interest or a financial interest of $10,000 or more ($5,000 or more if involved in clinical health care with an appointment in the Academic Health Center, AHC) associated with this study when aggregated for themselves and their family members?

☒ No.
☐ Yes.

If yes, identify the individual(s) and complete section 4.3:

4.2 Do any of the investigators or personnel (when aggregated for themselves and their family members) listed on this research have:

Ownership interests less than $10,000 ($5,000 if in clinical health care with an appointment in the AHC) when the value of interest could be affected by the outcome of the research?
☒ No. ☐ Yes.

Ownership interests exceeding 5% interest in any one single entity?
☒ No. ☐ Yes.

Compensation less than $10,000 ($5,000 if in clinical health care in the AHC) when the value of the compensation could be affected by the outcome of the research?
☒ No. ☐ Yes.

If yes, identify the individual(s) and complete section 4.3:

4.3 Has the business or financial interest been reported?

☒ No.
If you are a University of Minnesota researcher, please report your business or financial interest online via the Report of External Professional Activities (REPA) at:
http://egms.umn.edu/quickhelp/EGMS_Instructions/prepa.html
If you are a Fairview Health System researcher, please complete the Fairview Health Services Conflict of Interest Disclosure forms at:
http://www.fairview.org/Research/BusinessOperations/ConflictsofInterest/index.htm
and submit the completed forms to the Fairview Office of Research.
If you are a Gillette Children's Specialty Healthcare researcher, please contact the Director of Research Administration, at 651-229-1745.

☐ Yes.
If yes, have you been informed that a Conflict of Interest Review Committee is reviewing the information you reported on your REPA? ☐ No. ☐ Yes.

The IRB will verify that a management plan is in place with the Conflict of Interest (COI) Program. If the COI Program does not have an approved management plan in place for this research, they will contact the individual(s) listed in question 4.1 for additional information.

Final IRB approval cannot be granted until all potential conflict matters are settled. The IRB receives a recommendation from the Conflict of Interest Review Committee regarding disclosure to subjects and management of any identified conflict. The convened IRB determines what disclosure language should be in the consent form.

5. Use of Protected Health Information (PHI): HIPAA Requirements

5.1 As part of this study, do you:

a. Collect protected health information (PHI)* from subjects in the course of providing treatment/experimental care; or
b. Have access to PHI* in the subjects' records?

Please read the definition of PHI below before answering.

*PHI is defined under HIPAA as health information transmitted or maintained in any form or medium that:

1. identifies or could be used to identify an individual;
2. is created or received by a healthcare provider, health plan, employer or healthcare clearinghouse; and
3. relates to the past, present or future physical or mental health or condition of an individual; the provision of health care to an individual; or the past, present or future payment for the provision of healthcare to an individual.

The following records ARE EXEMPTED from the definition of PHI even though they may contain health-related information: student records maintained by an educational institution and employment records maintained by an employer related to employment status. If your study uses these kinds of records, it is not subject to HIPAA. However, existing IRB rules on informed consent and confidentiality still apply.

Health-related information is considered PHI if (any of the following are true):

1. the researcher obtains it directly from a provider, health plan, health clearinghouse or employer(other than records relating solely to employment status);
2. the records were created by any of the entities in "1" and the researcher obtains the records from an intermediate source which is NOT a school record or an employer record related solely to employment status; OR
3. the researcher obtains it directly from the study subject in the course of providing treatment to the subject.

Health-related information is not considered PHI if the researcher obtains it from:

1. student records maintained by a school;
2. employee records maintained by an employer related to employment status; OR
3. the research subject directly, if the research does NOT involve treatment.

☐ Yes. If yes to a or b above, complete Appendix H to show how you will satisfy HIPAA requirements for authorization to use PHI in research.

☒ No.

6. Summary of Activities

Use lay language, do not cut and paste from or refer to grant or abstract.

6.1 Briefly state what is your research question.

Under the presence of Aerobic Training, what happens to the rate of force production (ability to broad and vertical jump) as well as identify public health effects such as oxygen consumption and resting heart rate.

6.2 Describe the source of the records; medical, educational, employment, existing data set, or pathological specimens (waste).
For approval in this category you must plan to use an existing data set without access to identifiers, records review to which you have permissible access to records when the chart is older than January 1, 1997, or where the patient has signed a consent form which is in the file after January 1, 1997, or collecting waste tissue after it has been released to pathology.

We plan to use existing educational data records collected in PEA-1262, Marathon Training form the spring semesters of 2009, 2010, and 2011.

6.3 Number of records or specimens to be used:

In the last three years, there have been approximately 100 participant data collected.

6.4 How long do you anticipate this research study will last from the time you are determined to meet the criteria for exempt research?
Exempt research is generally considered short-term in nature. This office routinely inactivates exempt applications after five years from the time it was determined to meet the exempt criteria. If you think your project will extend beyond five years, contact the IRB office (612-626-5654 or irb@umn.edu).

Less than 3 months

6.5 Is the data you are gathering publicly available?

- ☐ Yes. Continue to 7.1
- ☒ No. Continue to 6.6

6.6 Do you already have permissible access to the records or specimens (i.e. through a job, volunteer work, internship etc.)?

- ☒ Yes. Describe how you have permissible access to the records.

The instructor and director of the course (PEA-1262) made the data available to the principal investigator.

- ☐ No. Continue to 6.6a

6.6a Will the records you receive be stripped of all identifiers that would make it possible for you to identify a subject?

- ☒ Yes. Continue to 6.7
- ☐ No. This research does not qualify for exempt status. Please complete the full IRB application, requesting expedited review if appropriate.

6.7 Confirm that the data/specimens you wish to review already exist

- ☐ The data set exists.
- ☐ The data set does not already exist.
  If the data is not already collected, the research does not qualify for exempt category four research. Please complete the full IRB application requesting expedited review if appropriate.

6.8 Please confirm that you will not have access to, or create a link, which would make it possible to identify subjects.

☒ I will not have access to, or create, a link.

☐ I will have access to a link.

If you have access to, or create a link you do not qualify for exempt category four research. Please complete the full IRB application requesting expedited review if appropriate.

6.9 Describe the identifying information to which you will have access to prior to recording data:

I will have no identifying information to which I will have access to prior to any data collection.

6.10 Describe the identifying information you will record:

Please note in order to proceed with exempt research under category four, you may not record information in such a manner that subjects can be identified directly or through identifiers linked to subjects.

I will not be recoding any identifying information. All of the data has been recorded and all identifiers removed.

7. Confidentiality

See Protecting Private Data Guideline from the Office of Information Technology (OIT) for information about protecting the privacy of research data.

7.1 Describe provisions taken to maintain confidentiality of data:

All the data released was free of any identifiable markers. The data was kept under password protected files and no access was permitted to the P.I.

7.2 Describe the security plan for data including how and where stored and duration of storage (i.e., password protection, encrypted data, etc.):

The data was kept under password protected files.

7.3 Will identifiable data be made available to anyone other than the PI?

☐ Yes.

☒ No.

If yes, explain who and why they will have access to the identifiable data:

This regulation does not apply to FDA regulated research.

You have reached the end of this form. Please make sure that you have responded to every question on this application (even if your response is "not applicable").

Appendix B

Sample Training Logs

Name: Sample Training Log #1

Subject #: 1

Week #	Date	Days run	Time (min.)	Distance
1	15-Dec-08	5	122	10
2	22-Dec-08	3	60	6
3	29-Dec-08	3	88	9
4	5-Jan-09	4	94	10
5	12-Jan-09	0	0	0
6	19-Jan-09	3	95	9
7	26-Jan-09	4	108	12
8	2-Feb-09	2	112	12
9	9-Feb-09	3	158	13
10	16-Feb-09	3	196	19
11	23-Feb-09	4	230	23
12	2-Mar-09	4	238	23
13	9-Mar-09	3	117	12
14	16-Mar-09	3	135	13.5
15	23-Mar-09	4	249	24
16	30-Mar-09	2	240	22
17	6-Apr-09	3	190	18
18	13-Apr-09	2	267	23
19	20-Apr-09	2	135	13
20	27-Apr-09	3	331	27
21	4-May-09	3	180	19
22	12-May-09	4	146	14
23	19-May-09	3	409	32
TOTAL		70	3900	363.5
Average week:		3.0434783	169.5652174	15.8043478

Name: Sample Training Log #2

Subject #: 8

Week #	Date	Days run	Time (min.)	Distance
1	15-Dec-08	0	0	0
2	22-Dec-08	1	30	3
3	29-Dec-08	2	60	6
4	5-Jan-09	1	30	3
5	12-Jan-09	3	90	9
6	19-Jan-09	3	110	13
7	26-Jan-09	3	120	14
8	2-Feb-09	4	175	21
9	9-Feb-09	3	164	18
10	16-Feb-09	4	210	25
11	23-Feb-09	3	135	15
12	2-Mar-09	3	210	25
13	9-Mar-09	4	209	25
14	16-Mar-09	4	230	26
15	23-Mar-09	4	275	30
16	30-Mar-09	4	320	36
17	6-Apr-09	3	190	22
18	13-Apr-09	1	180	20
19	20-Apr-09	2	90	11
20	27-Apr-09	3	260	30
21	4-May-09	3	210	25
22	12-May-09	4	230	27
23	19-May-09	3	280	32
TOTAL		65	3808	436
Average week:		2.826087	165.5652174	18.9565217

Name: Sample Training Log #3

Subject #: 11

Week #	Date	Days run	Time (min.)	Distance
1	15-Dec-08	4	90	10
2	22-Dec-08	4	95	10
3	29-Dec-08	4	110	11
4	5-Jan-09	2	80	7.85
5	12-Jan-09	3	100	9.15
6	19-Jan-09	4	150	15.1
7	26-Jan-09	4	160	17
8	2-Feb-09	5	205	22
9	9-Feb-09	4	200	20.1
10	16-Feb-09	4	235	23.2
11	23-Feb-09	4	240	24.5
12	2-Mar-09	4	265	26.3
13	9-Mar-09	4	233	24.3
14	16-Mar-09	4	188	20
15	23-Mar-09	4	283	29.3
16	30-Mar-09	4	326	33.5
17	6-Apr-09	4	270	28.1
18	13-Apr-09	4	350	37
19	20-Apr-09	4	269	28.6
20	27-Apr-09	4	333	35.7
21	4-May-09	4	208	21.8
22	12-May-09	5	280	30.6
23	19-May-09	4	322	34.9
TOTAL		91	4992	520
Average week:		3.9565217	217.0434783	22.6086957