Effect of Concurrent Sprint Interval and Resistance Training on Measures of Strength, Power, and Aerobic Performance

by

Richard Hutchinson Laird, IV

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Approved by

David D. Pascoe, Professor Kinesiology
L. Bruce Gladden, Professor Kinesiology
John C. Quindry, Associate Professor Kinesiology
John-Eric W. Smith, Principal Scientist, Head of GSSI IMG Academy

Abstract

PURPOSE: The purpose of this investigation was to determine if sprint interval training (SIT) performed concurrently with resistance training resulted in adaptive interference, had no effect, or caused synergistic enhancement to measures of strength, power, and VO_{2max} in recreationally active females when compared to resistance training alone. METHODS: 28 healthy females (20.3 ±1.7 yrs, 35.4 ± 4.0 $ml \cdot kg^{-1} \cdot min^{-1}$ VO_{2max}, 113 ± 17 lbs. 1 repetition max (1 RM) back squat) were asked to complete a 12 week resistance training study. Preliminary and post testing consisted of 1 RM back squat, maximal isometric squat, rate of force development (RFD), cycle ergometer based anaerobic power evaluations, lactate threshold (LT), and VO_{2max}. Following initial testing, participants were matched according to 1RM back squat and VO_{2max} values and randomly assigned to one of two groups: concurrent training (CT) that completed both resistance and SIT protocols, and resistance training (RT) which only completed the resistance training protocol. Training was completed 3 days per week and lasted for 11 total weeks. All resistance training was completed in the morning with each participant completing the protocol at the same time each day. Separated by at least 4 hours, CT participants returned and completed SIT. RESULTS: 1 RM squat and maximal isometric force values were significantly elevated following training in both RT and CT (both $P < 0.01$). RFD was not significantly altered in either group. Similarly, modified Wingate testing revealed

significant increases to peak and mean anaerobic power values in both ST ($P < 0.05$) and CT ($P < 0.01$) with no statistical difference between group response differences. VO_{2max} also increased as a result of resistance and concurrent training ($P < 0.01$). Predicted zero incline velocity that would elicit VO_{2max} (V_{max}) values were significantly elevated in both groups ($P < 0.01$) although concurrent training resulted in a significantly greater adaptive response ($P < 0.01$). LT values were not affected by training, although the velocity associated with LT (V_{LT}) increased significantly in both groups following training ($P < 0.01$). CONCLUSON: These data indicate that resistance training in isolation and sprint interval based concurrent training result in identical improvements to measures of strength, power, and VO_{2max} with no indication of adaptive interference. Only V_{max} adaptations supported the hypothesis of synergistic enhancement. These findings may be the result of commonalities between the adaptive responses to sprint interval and resistance training.

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List of Abbreviations

4E-BP1……..…….. eIF4E binding protein

AA……..……Amino Acid

ACSM ……..……American College of Sports Medicine

AMPK…….……..Adenosine Monophosphate Activated Protein Kinase

AMP……...……Adenosine Monophosphate

ATP……...……..Adenosine Triphosphate

a-v O_2……...…….Arteriovenous Oxygen

BMD……...……Bone Mineral Density

Ca++..……...……Calcium

CaMK…….……Calmodulin Dependent Kinases

COX…….…….Cytochrome c Oxidase

CS……..……..Citrate Synthase

CSA……..……Cross Sectional Area

CT……..Concurrent Training Group

DXA……..…….Dual-emission X-ray Absorptiometry

EMG……...……..Electromyography

HIIT……High Intensity Interval Training

IGF-1…….……..Insulin-like Growth Factor-1

Kcal……..Kilocalorie

LT……..Lactate Threshold

MHC……...…….Myosin Heavy Chain

MPB…….…..Muscle Protein Breakdown

MPS…….……..Muscle Protein Synthesis

MRI……Magnetic Resonance Imaging

MU…….…….Motor Unit

MUFF……...……Motor Unit Firing Frequencies

mRNA…….……. Messenger ribonucleic acid

mTOR……...…….. Mammalian Target of Rapamycin

NPB……..…….Net Protein Balance

p38……...…… Mitogen Activated Kinase p38

p70 S6k……...…… Ribosomal Protein S6 kinase

PI3-k…….……. Phosphatidylinositol 3-kinase

PGC-1α……..…… Peroxisome Proliferator-activated Receptor Gamma Coactivator

P_{max}…….……..Maximal Power associated with VO_{2peak}

Q……..…….Cardiac Output

RER……..Respiratory Exchange Ratio

RFD……...……Rate of Force Development

RM……..……Rep Max

RT……..…….Resistance Training

SIRT1……...…… sirtuin (silent mating type information regulation 2 homolog) 1

SIT……..……..Sprint Interval Training

SV……..Stroke Volume

T_{max}…….Maximal Exercise Time Completed at Velocity Associated with VO_{2max}

TFAM……..Transcriptional Factor A Mitochondria

TSC 1/2……..Tuberous Sclerosis Complex 1/2

V_{LT}……..……..Running Velocity Associated with Lactate Threshold

V_{max}…….……..Running Velocity Associated with VO_{2max}

VO_2……...……Volume of Oxygen Consumption

VO_{2max}……..…….Maximal Volume of Oxygen Consumption

CHAPTER I

INTRODUCTION

In 2011, the American College of Sports Medicine (ACSM) updated its position regarding recommended amounts of aerobic and resistance exercise needed to maintain and promote health [1]. For the development or maintenance of cardiovascular fitness current recommendations are; 1) at least 30 minutes per day of moderate activity 5 days per week, or 2) at least 20 minutes vigorous intense activity 3 days per week, or 3) some combination of both totaling 1000 Kcal per week [1]. Exercise meeting the above recommendations is categorized as endurance training. Weekly aerobic activity meeting these minimum requirements has been shown to result in increased aerobic fitness, decreased risk of cardiovascular disease, and decreased premature morbidity risk [1]. In addition, resistance training emphasizing dynamic multi-joint movements involving both concentric and eccentric contractions is encouraged 2-3 days per week to maintain and promote muscular fitness. Enhanced muscular fitness is associated with lower risk of all-cause mortality and the development of functional limitations [1]. While the ACSM suggests that minimal amounts of cardiovascular and resistance exercise are necessary to attain health, no recommendations are currently provided as to how training should be structured to both meet recommendations and maximize benefits.

Improvements to muscular strength and aerobic fitness are desirable adaptations in both athletes seeking improved performance and the general public looking to obtain

health benefits. The combination of resistance and aerobic exercise into a single program, in an effort to attain adaptations specific to each, is known as concurrent training. Research into the effects of concurrent training has been conducted for over 3 decades and has produced contradictory results. Several investigators [2-8], have reported that concurrent training results in decreased improvements to measures of muscular strength, power, and hypertrophy when compared to resistance training alone, a phenomenon which has been termed the interference effect. The interference effect has been attributed to overtraining [9], protocol design variations [10, 11], and an incongruence between signaling pathways responsible for adaptations to resistance and endurance exercise [12-15]. Improvement to measures of aerobic fitness have repeatedly failed to demonstrate any indication of interference [2-8, 16-20]. Although rarely observed, two studies [17, 21] have reported adaptive interference to VO_{2max} improvements with concurrent training. However each of these studies [17, 21] contains study design flaws that raise questions as to the reliability of results.

Support for the existence of adaptive interference with concurrent training is not universal. Other researchers report no indication of concurrent training based interference to measures of strength [8, 16-20, 22] and power [7, 20]. Furthermore, other studies [23-27] have reported synergistic enhancement of training adaptations when simultaneous resistance and aerobic exercise are performed. In contrast to traditional endurance training protocols typically utilized in concurrent training studies, the conditioning protocols of these investigations [23-27] closely resembled high intensity interval training (HIIT). As a result, it has been suggested that the adaptive responses to HIIT and resistance training contain more similarities than differences, thus accounting for synergistic enhancement [11].

The human neuromuscular system demonstrates a high degree of plasticity allowing skeletal muscle tissue to adapt favorably to specific tasks to which it is subjected. The Specific Adaptations to Imposed Demands (SAID) principal states that adaptations a physiological system undergoes in response to a training program are distinct, limited to systems that are utilized, and dictated by the level of overload experienced [28, 29]. Adaptive responses are dictated by a host of factors that include sex, initial fitness level, exercise intensity, duration, frequency, mode, and nutritional status. Ultimately a combination of these factors influences physical and biochemical adaptions of the system in accordance with the overload principle [28].

Skeletal muscle evolves in response to exertion-based cellular stress through altered fatigue resistance, aerobic / anaerobic enzyme concentrations, maximal contraction velocity, and myosin fiber remodeling [15, 29]. Stress resulting from exercise can generally be classified into one of three areas; 1) aerobic / endurance, 2) anaerobic / interval, or 3) resistance training. Traditional aerobic / endurance exercise consists of long duration (greater than 20 minutes), continuous, rhythmic, large muscle contractions completed at submaximal intensities. Adaptations to endurance training include increased a-v O2 difference [30, 31], stroke volume (SV) [32, 33], mitochondrial content [15], capillarity [31], oxidative enzyme activity [34, 35], decreases in total muscle size and cross sectional area, as well as fiber type transitions (IIx → IIa and II →I) [15, 36]. As a result, repetitive endurance training ultimately yields an increased capacity to load, transport, and utilize oxygen [15, 33, 37].

Anaerobic or interval exercise is classified as High Intensity Interval Training (HIIT) and consists of alternating work intervals performed near or above maximal intensities (> 90% $V0_{2max}$), interspersed with periods of low to moderate intensity exercise or complete rest. HIIT

interval duration varies greatly between protocols lasting between several seconds to several minutes with a negative relationship existing between duration and intensity. Over the past decade, HIIT has been shown to be equal to and in many cases more effective than endurance training at improving VO_{2max} [38, 39] as well as lactate threshold (LT) velocity and time trial performance [40-44]. This enhanced improvement may be due to greater peripheral adaptions which are generally more pronounced following HIIT [38]. HIIT has also been shown to result in increases in oxidative enzyme activity, markers of whole body lipid oxidization, and decreased glycogen and phosphocreatine depletion [42]. Sprint Interval Training (SIT), a specific type of HIIT which is characterized by supramaximal exercise intensities and very brief work intervals, has received more attention over the past several years. SIT has proven an effective method for improving aerobic fitness in a fraction of the time required with traditional endurance training [40, 42, 45].

Resistance training utilizes muscular contractions performed with additional external load usually limited to 20 repetitions or less per set. The adaptive response of skeletal muscle to resistance training varies greatly in response to protocol composition including: frequency of training, set and repetition schemes, exercise selection, resistance intensity, contraction velocity, and initial training status [29]. Adaptations to strength training include increased muscular strength and endurance, muscular hypertrophy, increases in bone mineral density (BMD), and increases in neuromuscular efficiency including: rate of force development (RFD), motor unit recruitment, synchronization, and increased frequency of stimulation [29].

While the body of knowledge available on the influence of concurrent training on adaptive responses is vast [2-8, 14, 16, 17, 20, 27, 46, 47], available data regarding HIIT and resistance training performed simultaneously is severely lacking or nonexistent. To the best of

our knowledge only one study has specifically examined the effects of concurrent training specifically utilizing HIIT [24]. Unfortunately this study lacked a strength-only control group making true inferences regarding the presence of adaptive interference difficult. Finally, high intensity and sprint interval exercise have been proposed as time efficient alternatives to traditional endurance training as lack of time is the most commonly cited barrier to physical activity [40-42]. However no research to date has examined adaptive strength and power responses to HIIT or SIT based concurrent training in recreationally active or untrained populations. Additionally, concurrent training studies employing all female subject pools are lacking. Therefore the purpose of this study was to determine if strength performance was impaired following concurrent resistance training and sprint interval running in untrained but active women. We hypothesize that the collaborative nature of adaptive processes resulting from sprint interval and resistance training will result in enhanced strength and power adaptations while improving aerobic fitness.

CHAPTER II

REVIEW OF LITERATURE

Introduction

The review of literature will provide a brief overview of the cardiovascular and musculoskeletal adaptations that take place in response to aerobic, interval, and supramaximal training protocols. Next a brief review of skeletal muscle adaptations in response to resistance training will be provided. Finally a review of available data on concurrent training will be presented including potential mechanisms linked to any interference effect between training adaptations.

Adaptations to Aerobic, Interval, and Supramaximal Exercise

The American College of Sports Medicine defines the minimum amount of aerobic exercise needed to maintain or improve cardiovascular fitness as 20 – 30 minutes of moderate to vigorous large muscle rhythmic activity such as walking, running, or swimming most days of the week [1]. Research has established that chronic participation in aerobic exercise results in numerous physiological changes which cumulatively act to increase aerobic fitness in an intensity dependent manor. Central adaptations to endurance training which include increases to: stroke volume (SV) [32, 33], myocardial contractility[48], left ventricular size [49], and plasma volume[50]; these function to increase the amount of oxygen loaded into the blood, and delivered to metabolically active tissue [37]. In addition, heart rate has been shown to decrease both at rest and with submaximal intensity exercise following endurance training [33, 51].

Peripheral adaptations, which occur in skeletal muscle tissue that experiences exercise induced cellular stress, include: increased capillarity [31], selective type I fiber hypertrophy [37], fast to slow fiber transformation [15, 36], decreased muscle Cross Sectional Area (CSA) [52], increased aerobic enzyme concentration and activity [34, 35], as well as mitochondrial biogenesis [15]. Peripheral adaptations ultimately result in increased oxygen extraction from the blood and as a consequence greater a-v O_2 differences [30, 31]. The degree to which central and peripheral adaptation occurs following training is directly related to exercise duration, mode, frequency, intensity, and the training status of the individual.

Fiber type shifting, particularly within the type II fiber pool, following both endurance and strength training is well documented in the literature [53-56]. Less certainty exists however regarding fiber conversions from fast (type II) to slow (type I) following endurance training. Fiber plasticity in response to aerobic training was first examined by Gollnick et al. [57] over a 5 month period. This pioneering study [57] reported no significant fast to slow fiber type transitions resulting from endurance training. Subsequent investigations also failed to observe fiber type shifting regardless of training protocol or subject pool [58, 59]. In contrast, other studies [60, 61] have reported that the percentage of type I fibers may be increased at the expense of the type II population with various aerobic training protocols including cycling [60] and long distance running [61]. Collectively these studies suggest that skeletal muscle fiber composition can be altered by endurance training, and that adaptive changes are likely governed by program design and participant training status.

The majority of improvements in aerobic fitness associated with endurance training can be attributed to increased oxygen extraction by active tissue which results from increased capillarity and mitochondrial biogenesis [35, 62]. Endurance training results in intracellular

perturbations including increased concentrations of cytosolic Ca+ , free radicals, and AMP, all of which are capable of initiating secondary signaling cascades within muscle cells [15]. In addition to binding troponin c, free Ca+ also binds and activates calmodulin dependent kinases (CaMK) capable of initiating signaling cascades which ultimately increase the oxidative capacity of muscle cells. Additionally, increases in AMP concentrations indicate changes in the energy status of the cell and causes activation of 5'adenosine monophosphate activated protein kinase (AMPK) [63]. Generally speaking AMPK initiates signaling pathways that inhibit ATP consuming processes and stimulates ATP generating processes. Finally, increased free radical production with endurance training results in activation of mitogen activated kinase p38 (P38) [15]. When activated, CaMK, AMPK, and P38 initiate signaling cascades which act to increase the activity of peroxisome proliferator-activated receptor gamma coactivator (PCG-1α), the master regulator of mitochondrial biogenesis in cells [64-66]. PCG-1α not only activates and assists transcriptional regulators responsible for increased mitochondrial biogenesis but also regulates many other endurance training derived adaptations including angiogenesis and fast to slow fiber type conversions [65], all of which result in an increased a-v O_2 difference [62]. A single bout of endurance training has been linked to increased mRNA expression of a number of genes associated with mitochondrial biogenesis and metabolism up regulation [67].

High Intensity Interval Training (HIIT), which is characterized by work periods at, near, or above intensities that elicit VO_{2max} lasting between 5 seconds and 5 minutes interspersed with periods of low intensity recovery or complete rest of equal or longer durations, has been the focus of a considerable amount of research since the early 2000's. HIIT protocols are designed to repeatedly stress physiological systems to a greater extent than typically required by daily activity or competition. HIIT has consistently been shown to be equal to if not more effective

than endurance training at eliciting adaptations to the cardiovascular system including increased cardiac output (Q), SV, oxidative enzyme activity, mitochondrial biogenesis, and VO_{2max} [38, 39, 44, 62, 68-70].

In 2002, Laursen et al. [70] examined the effect of three HIIT protocols on peak oxygen consumption (VO_{2peak}), and other performance measures in comparison to traditional endurance training in well-conditioned cyclists. Results of this study indicated that HIIT protocols utilizing 60% of the maximal time that can be completed (T_{max}) at an intensity equal to that which elicits VO_{2peak} (P_{max}) resulted in greater improvements in performance measures. In addition, Edge et al. [44] examined the efficacy of HIIT utilizing 2 minute intervals at an intensity equal to between 120 and 140% of work rate associated with LT compared to a matched work traditional endurance training protocol. Results of this study showed that HIIT resulted in greater cycle ergometer repeated sprint ability, but equal VO_{2peak} and LT gains when compared to endurance training. Finally, when multiple protocols of varying intensity and durations were compared in recreationally active participants, VO_{2max} improvements were observed in an intensity dependent manor [39]. The 5 minute HIIT protocol resulted in VO_{2max} improvements that were greater than the 40 minute endurance training group completed at 75% VO_{2max}, and the 40 minute group demonstrated greater improvements than the 60 minute endurance training group completed at 50% VO_{2max}. When examined together these studies [39, 44, 70] suggest that HIIT protocols result in equal or greater adaptation when compared to endurance training and that improvements in aerobic fitness are directly related to the intensity of the exercise performed.

Further support for the potency of HIIT is provided by additional studies directly comparing exercise protocols existing at different points along the time-intensity continuum and matched for total work [38, 62]. Helgerud et al [38] compared the effect of four distinct

protocols (2 HIIT, 2 endurance training) on VO_{2max}, SV, and Q changes in trained male subjects. Findings of this study indicate that only HIIT protocols resulted in improvements in VO_{2max} and that these increases were driven by central adaptation to SV and Q. In 2008, Daussin et al. [62] conducted a study examining cardiorespiratory and mitochondrial functional responses to HIIT and endurance training. This study indicated that improvements in VO_{2peak} between the two groups were similar but that improvement resulted from changes to different factors affecting VO_{2max}. Endurance training based VO_{2peak} increases were mainly associated with improved oxygen extraction as measured by increases in a-v O_2 difference and improved capillarity density. Conversely, HIIT derived improvements were caused by increases in Q, SV, as well as a-v O_2 difference increases [62]. It should be noted that this study utilized untrained participants and the endurance training protocol consisted of relatively low intensity exercise equal to 60% of peak power. The exercise intensity could partially explain obtained results even though total work was matched. Collectively these studies suggest that improvements from HIIT may at least in part be due to central adaptations that occur to a lesser degree with endurance training.

Measures of oxidative enzyme content, activity, and mitochondrial biogenesis following as little as six HIIT sessions over two weeks indicate similar adaptive responses to those typically observed with traditional endurance training. Little et al. [68] examined the effect of six training sessions consisting of 8-12 repetitions of 60 second intervals completed at 100% of peak power interspersed with 75 seconds of recovery. Muscle biopsies obtained before and after training indicate increased maximal activity of citrate synthase (CS) and cytochrome c oxidase (COX) as well as increased total protein content of CS, COX, transcriptional factor A mitochondrial (TFAM), and PGC-1α [68]. Additionally, sirtuin (silent mating type information

regulation 2 homolog 1) (SIRT1) content, a proposed co-activator of PGC-1α and up regulator of mitochondrial biogenesis, increased by over 50% following HIIT. Taken together these results suggest that HIIT provides a potent stimulus for increasing mitochondrial capacity and that up regulation of TFAM, nuclear PGC-1α, and SIRT1 likely play a role in coordinating the observed adaptations [68].

Sprint interval training (SIT), a specific type of HIIT in which supramaximal intensities and brief work durations (< 1 min) are employed, has been increasingly studied over the past several years. SIT has been shown to up-regulate both glycolytic and oxidative enzymes, as well as increase maximal power output and VO_{2max} in a time efficient manor [71-73]. In one of the early studies to compare SIT to endurance training, Tabata et al. [74] utilized a 4 minute SIT protocol containing 8 repetitions of 20 second intervals on a cycle ergometer at an intensity equal to 170% of the work load that resulted in VO_{2peak} separated by 10 seconds passive recovery. Tabata [74] compared this group to an endurance training control consisting of 60 minutes of endurance training at 70% VO_{2peak} [74]. SIT resulted in VO_{2peak} improvements equal to those in the control group as well as significantly increased anaerobic capacity which was not found following endurance training.

More recently additional variations of SIT have been examined [40, 42, 45, 71]. Multiple researchers [40, 42, 45] have utilized repeated Wingate tests compared to 40–120 minutes of endurance training completed at 65% VO_{2peak}. Collectively their results suggest that SIT and endurance training resulted in significant improvements in VO_{2peak}, time trial performance, and measures of muscle oxidative capacity while only SIT increased maximal glycolytic enzyme activity and anaerobic capacity. Interestingly SIT participants in these

studies completed only 10% of the mechanical work of the endurance training controls indicating that SIT is a time efficient strategy for inducing rapid skeletal muscle adaptation.

While the body of knowledge regarding molecular responses to exercise is large, comparatively little is known about cellular responses which mediate adaptation to SIT. Paradoxically, in addition to the cellular adjustments made to endurance training and HIIT, SIT has repeatedly been shown to cause slow to fast (type I to type II) fiber transition [61, 75-77]. Dawson et al. [75] examined the effect of short (10 second) sprint training on fiber type distribution. Post training muscle biopsies revealed approximately a 15% increase in type II fibers and cross sectional area (CSA) with a concomitant equal decline in type I fiber number and CSA. These authors [75] concluded that the observed slow to fast fiber shifting, occurred in accordance with the SAID principal, and left the muscle more suited to produce maximal force during repeated sprint efforts [75]. Significant increases were also observed in supramaximal treadmill run time demonstrating that SIT also improves anaerobic power potentially caused by increased muscle buffering capacity and greater anaerobic energy contribution. Additional support for high velocity ambulatory contractions resulting in slow to fast fiber transitions can be found in the studies completed by Jansson [61, 76] and Andersen [77].

Summary

Adaptations to long term endurance training are well established [15, 32-34, 48] and include both peripheral and central changes which ultimately strive to deliver more oxygen in the blood, and increase oxygen extraction and utilization by active tissue. Although not universally accepted [57], it is generally believed that a long term adaptation of endurance training is fast to slow fiber shifting both within type II and between fiber types [60, 61], a change that would further increase the endurance capacity of a muscle. At the molecular level,

adaptations to aerobic exercise are driven by intracellular perturbations to concentrations of cytosolic Ca++ , free radicals, and AMP, all of which are capable of initiating secondary signaling cascades driving adaptive processes within the muscle [15]. PGC-1α, the master regulator of mitochondrial biogenesis in cells [64-66], is the final target of signaling cascades and is up-regulated by changes to concentrations of cytosolic Ca++ , free radicals, and AMP within the cell. Along with stimulating transcriptional regulators of mitochondrial biogenesis, PGC-1α also exerts regulatory control over angiogenesis and fast to slow fiber type conversions [65].

In recent years, both HIIT and SIT have been shown to be equal if not more effective than traditional endurance training at eliciting improvements in VO_{2max} [39, 40, 73]. Improvements to measures of aerobic fitness with this type of training may in part be due to greater central adaptations than typically observed with traditional endurance training [38, 62]. A unique observed adaptation to HIIT is the ability to increase both aerobic and anaerobic enzyme concentrations and activity simultaneously [42, 74]. Finally, SIT has been shown to elicit slow to fast fiber type transitions following training resulting in increases in the type II fiber pool at the expense of type I fibers [61, 75-77].

Adaptations to Resistance Training

A classic fable concerning the overload principal involves Milo of Crotona, a Greek farmer who completed his exercise sessions with a newborn calf draped across his shoulders. As the calf grew into a mature bull, Milo continued to complete the morning exercise sessions giving him unmatched strength and victory at the Olympiad [78]. In contrast to endurance training, resistance training utilizes muscular contractions performed against external load to induce cellular stress, and is usually limited to 20 repetitions or less at a time. Resistance

training derived adaptations are dependent on the intensity, frequency, and duration of training as well as the number of repetitions performed. Low intensity high repetition (12-20 reps) resistance training leads primarily to increased muscular endurance; moderate intensity moderate repetition (5-10 reps) results in the largest increases in muscular hypertrophy, and high intensity low repetition (1-3 reps) resistance training causes the largest increases in absolute muscular strength [29, 79].

Additionally, resistance training does not result in large changes to mitochondrial number or capacity and may actually decrease mitochondrial density with significant muscular hypertrophy [29]. Changes in muscular endurance, size, and strength do not occur in isolation. Rather each intensity and repetition combination results in the initiation of cellular signaling pathways which drive adaptive responses that will best cope with cellular stress brought on by the completed task, and smaller improvements in other areas in accordance with the theory of specificity [29]. Ultimately resistance training results in increased muscular strength through changes in muscle fiber composition, neural adaptations, and increases in skeletal muscle mass [15, 29, 80, 81].

Type II muscle fibers have been shown to have greater shortening velocity and force generation capacity than type I fibers [82]. As such, any increase in the number of type II fibers, or changes within type II fiber population allowing a greater percentage of fibers to be activated in response to a given stimulus, would result in greater maximal muscular strength. Research indicates that resistance training results in a fast to slow fiber remodeling in a manor distinct endurance training, in that it does not increase the type I fiber population [36]. Evidence is provided by various resistance training protocols which demonstrate the above fast to slow

conversions within type II fibers both with initial training [53, 54, 83], and retraining after a period of detraining [84].

The concept of resistance training based slow to fast fiber shifting at the expense of type I and IIx populations is more controversial. Indeed, neither 8 weeks of jump squats [59], nor 6, 9 [83], or 19 [53] weeks of resistance training resulted in any inter-fiber conversion leading to the conclusion that resistance training does not result in slow to fast fiber type shifting [36]. However, other investigations report that specific high contraction velocity based training results in increased type II fiber population [85, 86]. Liu et al. [86] compared two experimental resistance training protocols and their effects on myosin heavy chain (MHC) isoform expression. Participants trained for 6 weeks, 3 days per week with the control group performing traditional low repetition high intensity resistance training 3 days per week. The experimental group performed traditional resistance training on day one, 5 sets of 10 bench throws at 30% of max on day 2, and 5 sets of 10 plyometric push-ups on day 3. The control group, showed significant decreases in type IIx MHC, increases in type IIa HMC, and no change to type I MHC providing support for the unique fast to slow fiber shifting following resistance training. However the experimental group demonstrated no decrease in type IIx MHC, but an increase in type IIa MHC facilitated by a 50% decrease in type I MHC [86]. These results suggest that explosive resistance training protocols results in slow to fast fiber type shifting at the expense of the type I fiber populations [86].

Neural adaptations have also been demonstrated to play a significant role in increased strength expression, particularly with the initiation of resistance training [29, 80, 87]. In one of the first studies to examine the effect of resistance training on neural factors, Sale et al. [87] concluded that voluntary strength expression is a skilled act in which agonist and synergistic

stabilizing muscles must be activated maximally while being opposed by minimal antagonist activity. With the onset of resistance training, greater increases in strength occur than can be explained by hypertrophic increases in cross sectional area (CSA), which has been attributed to neural adaptations [80]. Increases in skeletal muscle activation following resistance training are mediated by increases in firing frequency, motor unit recruitment, neural drive, and changes to antagonist co-activation [29, 80].

Multiple researchers have utilized surface electromyography (EMG) to investigate changes to agonist muscle activation as a result of resistance training [18, 88, 89]. Median surface EMG readings are regarded as measures of motor unit recruitment. Increased median EMG measurements resulting from training have been interpreted to occur as a result of increased muscle activation, and partially explain increases in strength [88]. Unfortunately, results of studies utilizing surface EMG have produced inconsistent findings with some reporting significant increases [18, 90-92], while others report no change [89, 93, 94], in spite of increased strength measurements at the conclusion of all studies. Despite conflicting reports, Akima et al. [95] provide convincing evidence of increased motor unit recruitment with resistance training via observations that the volume of activated muscle was significantly greater with maximal voluntary contractions following only 2 weeks of high intensity isokinetic training. Inconsistent findings using EMG measurements can at least partially be explained by protocol variation, as well as measurement and interpretation issues including: reproducibility questions, relocating electrodes, variable impedance of skin and subcutaneous fat, as well as changes in muscle morphology [29]. Although not universally reported, it seems likely that alterations in maximal MU recruitment contribute to increase in strength observed independent of changes in CSA.

Two notable studies have examined neural drive through the analysis of Motor Unit Firing Frequencies (MUFF), obtained utilizing intra-muscular EMG recording techniques in humans [96, 97]. When measured in elderly women, MUFF rates are higher in weight trained women than their untrained counterparts suggesting that resistance training results in increased neural drive and motor unit firing frequency [96]. Additional support for the role of increases in MUFF accompanying increases in strength is provided by a 12 week training completed by Van Cutsem et al. [97], which demonstrated that fast ballistic contractions resulted in earlier motor unit activation and enhanced MUFF when compared to traditional resistance training. Study results were interpreted as indicative of increased motor unit firing rates at the onset of contractions. Collectively findings from these studies indicate resistance training results in increased neural drive and MU firing frequencies which contribute to greater agonist muscle activation and increased strength independent of CSA adaptation.

Antagonist muscle activation has the capacity to influence net force production via reciprocal inhibition. Greater levels of antagonist coactivation have been observed in untrained individuals compared to trained strength and power athletes [98, 99]. Studies examining the effects of training on antagonist muscle activation have produced conflicting results. Carolan et al. [100] utilized a within subject control experimental design to examine the effect of maximal isometric knee extensions performed 3 times per week on knee flexor coactivation. The most important finding of this study was that after only one week, knee flexor coactivation decreased by 20% in the resistance trained leg, and by 13% after two weeks in the untrained leg. Carolan et al. [100] concluded that early decreased knee flexor coactivation is an early adaptive response to resistance training and contributed to the 32.8% increase in trained maximal voluntary contraction. However, other studies have reported no changes in antagonist activation after 9

sessions [101], 14 weeks [102], or 6 months [103] of resistance training in middle aged or older populations. When combined, the results of these studies indicate that decreased coactivation of antagonist muscle groups may play a role in early increases in muscular strength with resistance training. It also seems that this response may dissipate with age. Nearly all of the studies examining antagonist coactivation have utilized single joint training and evaluation task. With the completion of more complex, full body, and high velocity movements, it seems plausible that the initial level of agonist activation could be higher allowing greater opportunity for reduced activation with training [29].

Skeletal muscle hypertrophy is documented as the primary adaptation to long term resistance training [104]. Comparison between the sexes reveals that the muscular hypertrophy adaptive response is blunted in women compared to men [79]. Sex based hypertropic differences have been attributed to decreased anabolic hormone concentrations, CSA, and smaller muscle fibers in women compared to men. It has therefore been suggested that neural adaptations play a larger role in female strength gains in response to resistance training than males [79].

Increases in skeletal muscle mass occur when the rate of muscle protein synthesis (MPS) exceeds the rate of muscle protein break (MPB) down consistently over weeks to months resulting in a positive net protein balance (NPB). Both resistance training [105-107] and amino acid (AA) consumption [108, 109] have been shown to increase MPS rates. In addition, simultaneous increases in MPS and decreases in MPB have been observed following resistance training in the fed state [110]. Post training MPS rates remain elevated for up to 48 hours in untrained participants [106], return to resting levels within 36 hours in trained individuals [111], and are reduced in participants when the same absolute work load is completed following

resistance training [107]. Ultimately muscular hypertrophy results in increased fiber CSA which allows for increased actin-myosin cross bridge interaction and force production [29].

Traditionally, initial increases in strength following the initiation of resistance training are attributed to neural adaptations, however changes to fiber CSA have been documented after as little as three weeks [112]. Narici et al. [93] utilized magnetic resonance imaging (MRI) to track the adaptive hypertropic response to 6 months heavy resistance training and observed linear whole muscle growth over the entire study with no indication of plateau. Although the exact time course of slowed muscular hypertrophy has yet to be documented, it is intuitive that the linear hypertropic increases cannot continue indefinitely. When CSA was examined after 24 weeks of heavy resistance exercise in a group of highly trained body builders (5 + years' experience) no changes in CSA or fiber area were observed [113].

Force produced by skeletal muscle contractions results in mechanical stretch and muscular tension whose severity is dependent on the opposing resistance, level of activation, and percentage of maximal voluntary contraction required [29]. Mechanical stretch of skeletal muscle and surrounding structural support components is the primary messenger of resistance training based adaptive signaling and results in acute secretion of Insulin-like Growth Factor-1 (IGF-1). IGF-1, when released, acts in an autocrine and paracrine fashion on surrounding muscle tissue [15]. IGF-1 asserts its effect through interaction with its membrane bound receptor IGF-1 binding protein, which in turn activates the phosphatidylinositol 3-kinase (PI3-k) - Akt – mammalian target of rapamycin (mTOR) cascade. mTOR, the end target of this cascade, then regulates the activity of multiple downstream effectors, most notably in relation to MPS, ribosomal protein S6 kinase (p70 S6k) and eIF4E binding protein (4E-BP1) [12]. P70 SK6 interacts with multiple substrate targets involved in cellular size maintenance and manipulation

as well as control of protein synthesis rates [15]. Increases in the concentration of active p70 S6k following resistance training has been observed in both animal [114] and human [115] models and is positively correlated with increases in maximal strength as well as type IIa CSA. Activated mTOR signaling also regulates 4E-BP1 function via hyper-phosphorylation resulting in suppression if its inhibitory effects eIF4E, a power promoter of MPS [15]. Current evidence suggests that acute changes in MPS rates are cause by increased mRNA translational efficiency and not to additional mRNA concentrations [12]. The role of acute and chronic changes in the quantity of mRNA and its role in the process of MPS remains an area of ongoing research.

Summary

Resistance training, in accordance with SAID and overload principals results in selective adaptation to activated muscle fibers which collectively result in increased muscular size and strength with little to no influence on aerobic factors. Resistance training results in fast to slow muscle fiber type transitions within the type II fiber population with no increases to type I fibers [36]. In addition, explosive resistance training protocols completed with submaximal loads at maximal velocities have resulted in slow to fast fiber shifting increasing IIa fiber content at the expense of the type I populations [85, 86]. As type II fibers have been shown to have greater shortening velocity and force generation capacity than type I fibers [82], slow to fast fiber transitions contribute to strength gains observed with this type of training.

Neural adaptations have also proven to play a significant role in increased strength expression, particularly with the initiation of resistance training [29, 80, 87]. Alterations in neural output is accomplished through increases to firing frequency rates, motor unit recruitment, neural drive, and decreases in antagonist co-activation [29, 80]. Furthermore, it has

been suggested that neural adaptations play a larger role in strength gains obtained by women due to due to decreased hypertropic adaptations observed in this population [79].

Skeletal muscle hypertrophy is the primary adaptation to long term resistance training [104]. Increases in skeletal muscle mass occur when the rate of MPS, which remains elevated for up to 48 hours post training [106], exceeds the rate of muscle protein break (MPB) down consistently over weeks to months. Measurable increases in fiber CSA have been observed following as little as 3 weeks of resistance training [112], and have been shown to continue to increase in a linear fashion for 6 months [93].

Molecular signaling cascades which modulate adaptation to resistance training is primarily initiated by mechanical stretch [15, 29]. Contraction induced IGF-1 release from activated musculature initiates the PI3-k – Akt – mTOR signaling cascade [15]. Activity of P70 SK6 and 4E-BP1, which are well defined modulators of MPS rates, is increased in concert with mTOR activation [114, 115] . The end result of increased mTOR, p70-SK6, and 4E-BP1activity is elevated MPS rates resulting from increased mRNA translational efficiency [12].

Concurrent Training

Resistance training designed to increase muscular size and strength, performed simultaneously with aerobic training aimed at improvements in endurance is known as concurrent training. Traditionally, studies examining concurrent training have involved three groups: one that performs only strength training, one that performs only endurance training and a third group which performs both. Although the focus of research for over 30 years, studies examining the influence of concurrent training on the adaptive response have produced inconsistent results. Hickson et al. [2] first introduced research on the effect of concurrent

training in 1980 and found that concurrent training resulted in inhibited strength gains but identical VO_{2max} improvements when compared to each type of training performed in isolation.

In support of the initial findings of Hickson et al. [2], several other investigators have reported compromised adaptation to various indices of muscular performance including strength [3-7, 14, 116], power [3, 17, 117], and RFD [8, 18] following concurrent training. In addition, it appears that adaptive interference is limited to musculature that undergoes both endurance and strength training as shown in studies which measured no interference to upper body strength when trained concurrently with lower body endurance training protocols [20]. The negative effect of concurrent training on the adaptive response has since been described as the interference effect [2, 118]. Support for the existence an adaptive interference following concurrent training is far from universal. In fact an adverse effect of concurrent training on the developed VO_{2max} has rarely been observed [2, 3, 5, 7, 14, 16, 20, 117-120]. Only two studies have reported interference to aerobic adaptations and each contains study design flaws which raise questions as to their reliability [17, 21]. Nelson et al.[21] utilized experimental groups consisting of only 4-5 subjects, and collected VO_{2max} data on a treadmill while training was completed on a cycle ergometer. Glowacki et al [17], in spite of randomly assigning participants, began with significantly higher initial VO_{2max} values in concurrent training compared to endurance training groups. As a result each of these studies must be interpreted with caution.

In contrast to interference, other investigators have found no evidence to support the existence of an interference effect following concurrent training when measures of strength [8, 16-20, 22, 23] and power [20] are examined. Furthermore synergistic enhancement of certain physiological variables resulting from concurrent training has also been reported, including

measures of endurance [20], muscular hypertrophy and VO_{2max} [8], and anaerobic power [23]. Collectively these studies suggest adaptive interference is not an automatic end result of concurrent training.

Variance in reported presence of adaptive interference has been attributed to a multitude of protocol design factors which modulate the adaptive response. Training frequency varies greatly between studies and for the concurrent training group has generally ranged between 4 [8], and 11 [2] training sessions per week with both multiple training sessions per day [2, 121] and alternate training stimuli on each day [5, 116]. When multiple training sessions are used on the same day both consecutive training sessions [27, 121], and planned rest between session [20, 25] have been employed. Furthermore the mode of resistance training selected confounds study interpretation as it appears to influence the level of adaptive interference or lack thereof that is observed [11, 46]. The vast majority of concurrent training studies involving isoinertial resistance training have resulted in an inhibition of either strength or power development, while other methods such as isokinetic contractions result in less consistence interference [46].

Choice of endurance training mode also has adaptive interference implications. In a recent concurrent training meta-analysis, Wilson et al [11] analyzed the effect sizes of concurrent training studies utilizing running based endurance vs. concurrent training studies utilizing other modes endurance training. His analysis indicated that significantly greater interference is observed to measures of muscular hypertrophy and strength when run based endurance training is prescribed. It has been suggested that other exercise modes such as cycling might have a greater carry over effect to strength measurements assessed and that less eccentric based muscle damage is incurred with non-running, non-weight supporting protocols [11]. One might speculate that different types of contractions produce varying levels of

interaction between strength and endurance training, however future research examining this possibility is needed before contraction type can be considered a variable of interest.

Along with frequency and mode of exercise, individual variations in the adaptive response to concurrent training as a result of training history and initial training status have been suggested [46]. Larger adaptive responses in strength and power measurements following concurrent training were obtained in endurance trained athletes when compared to sedentary volunteers [117]. Unfortunately no data were collected on a control resistance training only group in this study so inferences regarding interference effect in trained endurance athletes from this study are not possible. Furthermore, concurrent training when utilized as a training strategy with athletes has resulted in improved strength and power measurements [27, 47]. Baker et al. [47] reported maintenance or improvements in measures of strength, power, and overall performance in college and professional rugby teams. Unfortunately, the nature of this study and others like it prevents comparisons to resistance training only control groups. It remains possible that participants with substantial training backgrounds are less susceptible to interference effect of concurrent training on strength development [46]. Participant training history and initial fitness level further confound our understanding of the effect of concurrent training.

Variations in dependent variable sensitivity may also influence the amount of interference that is observed with concurrent training [46, 122]. Abernethy et al. [123] examined the sensitivity and adaptive time course of various strength measurements (one repetition max, maximal isometric force, and isokinetic contractions (5 velocities)) in response to 4, 8, and 12 weeks of strength training. Their results suggest that the sensitivity of various strength measures were dissimilar and indicative of different mechanistic events. As a result, it has been suggested

that dependent variable selection likely influences the level of interference, if any, observed with concurrent training.

Indeed, when examining measures of maximal muscle performance, only those related to power have consistently resulted in universal interference. Dudley et al. [5] was the first to report a velocity dependent interference effect of concurrent training. After 7 weeks, impaired magnitudes of specific maximal torque with fast, but not slow velocities of contraction where measured in the concurrent training but not strength training group. Subsequent studies which evaluated power output, in some capacity, have supported Dudley's initial findings demonstrating interference in power adaptations in spite of no interference effect observed in measures of maximal strength [3, 8, 17, 18, 117].

Following 21 weeks of training, Mikkola et al. [8] reported identical increases in 1 RM values between groups but that only resistance training resulted in improved RFD. These results support Dudley's [5] initial suggestion that activities which require rapid force production may exhibit greater adaptive interference. Neural adaptations however, as measured by mean EMG amplitude, increased significantly with indifference toward group membership [8]. Additional evidence of contraction velocity specific interference effect is provided by a meta-analysis conducted by Wilson et al. [11] which analyzed the effect sizes of 21 training studies finding that only adaptations to power measurements were negatively affected by concurrent training. Furthermore, no significant differences were observed in measures of hypertrophy and strength between strength only and concurrent training groups when effect size statistical analysis was utilized [11]. Collectively these findings indicate that explosive contractions and power based activities are more likely to exhibit interference than either maximal strength or hypertrophy values.

Modulation of each of the variables mentioned above has resulted in decreased interference resulting from concurrent training [7, 8, 16-20, 22, 23]. On the contrary, the presence of interference has been reported across all fitness levels, frequencies of training, and exercise modalities with concurrent training [46]. While many researchers have examined variables that may be affected by adaptive interference resulting from concurrent training, relatively few have attempted to elucidate why such interference might occur. Of mechanisms that have been proposed, overtraining and incompatible molecular signaling pathways have received the most support although neither is unanimously accepted.

It has been proposed that the increased work load completed by the concurrently trained group could be responsible for the observed interference [2]. Indeed, the concurrent training group in the initial study of Hickson et al. [2] not only performed a much higher work load, but also completed strength and endurance training protocols separated by only 15 – 120 minutes. With this training protocol and others like it, chronic glycogen depletion, residual fatigue, and inadequate recovery (collectively called overtraining) could all effect optimal muscle tension development with subsequent resistance training. When chronically repeated over multiple training sessions, diminished tension development could contribute to inferior strength adaptations.

In 1985, Dudley et al. [5] demonstrated that overtraining alone could explain the existence of an interference effect with concurrent training. In this study endurance training was complete 3 days per week and consisted of 5 repetitions of 5 minute exercise periods on a cycle ergometer at an intensity that elicited VO_{2max} in the 5th minute. Resistance training was performed on 3 alternate days and consisted of only 2 repetitions of 30 seconds. While the concurrently trained group in this study still demonstrated an interference effect, it seems

unlikely that this drastically reduced training volume was enough of a stimulus to label overtraining the culprit [46]. When training frequency is moderate (2-4 days per week) and adequate recovery is allowed between training sessions (minimum 4 hours), consistent adaptive interference has not been observed indicating that with the exception of power measures, training organization and total volume likely affect the level of interference observed [11].

Another proposed cause of interference resulting from concurrent training is an apparent incapability of muscle adaptation to resistance and endurance training performed simultaneously [12, 13, 15]. Given the complexity of divergent signaling responses to endurance and resistance training, which operate at opposite ends of the adaptive continuum, it is reasonable to suggest that optimal adaptation to each is not possible when performed at the same time [15]. The first clue that strength and endurance training might initiate unique signaling pathways was provided by Atherton et al. [124], who utilized electrical contractions mimicking endurance and strength training in isolated rat muscle. Endurance-like contractions increased AMPK and PGC-1α activation levels while mimicked strength training resulted in increases in mTOR signaling suggesting that exercise specific adaptations are mediated by activation of separate molecular pathways.

Cellular responses to resistance training are initiated by contraction induced production of IGF-1 which binds to membrane bound IGF-1 receptors which initials the PI3-k – Akt – mTOR signaling cascade[12]. Phosphorylation of mTOR effectors p70 S6 and 4E-BP1has been implicated in increased NPB via increased RNA translational efficiency derived MPS rates, and decreased protein breakdown [15]. As a result, resistance training adaptations include increased myofibrillar CSA, type IIa MHC composition, anaerobic enzyme concentration / activity, and RFD with little to no effect on mitochondrial biogenesis [125]. Conversely, endurance training

adaptive signaling results from transient intracellular perturbations including elevated free cytosolic Ca++, free radical concentration, and AMP levels [63]. Resultant activation of CaMK, AMPK, and p38 up-regulate PGC-1α activity which is the cellular master switch controlling mitochondrial biogenesis [64-66]. PGC-1α activation results in increased capillarity and mitochondrial biogenesis which function to increase concentration of enzymes associated with aerobic energy production, the electron transport chain, and oxygen / substrate delivery and utilization to active tissue with no measurable increases in myofibrillar size.

Simultaneous activation of mTOR and PGC-1α pathways has been suggested to impair protein synthesis following resistance training [13, 78]. One mechanism by which cross talk between pathways could occur is via AMPK mediated activation of tuberous sclerosis complex 1/2(TSC 1/2) which inhibits mTOR activity and impairs contractile protein synthesis [12, 15]. Up regulation of TSC 1/2 by AMPK provides a biochemical link explaining why endurance exercise might inhibit optimal adaptation to resistance training [12].

Although attractive, interference derived from increased AMPK / TSC 1/2 activity has not been universally demonstrated in human skeletal muscle. In 2012 Lundberg et al. [25] examined the acute molecular responses to resistance training performed in one leg versus concurrent training performed on the other. Aerobic training was completed prior to resistance training and consisted of 45 minutes of one legged cycling at 70% of maximal wattage. Six hours after endurance training, each leg was subjected to a resistance training protocol which consisted of 7 maximal concentric and eccentric knee extensions. Surprisingly and in contrast to the proposed cross talk between adaptive pathways above, concurrent training resulted in greater mTOR and p70 S6k phosphorylation along with greater myostatin suppression, which is implicated in increased MPB [25]. The main finding of this study was that an acute bout of

concurrent training, provided adequate recovery between sessions, did not cause decreased mTOR activation and may result in enhanced cellular anabolic environments. In a follow up study utilizing an identical protocol completed 5 days per week for 5 weeks, Lundberg et al. [26] attempted to determine if initial acute differences in cellular signaling yielded pronounced differences in muscle hypertrophy between legs over time. Using MRI and muscle biopsy CSA measurements, significantly greater increases in muscle size were evident following concurrent training indicating that endurance training when completed prior to resistance training amplified hypertrophy signaling [26]. Collectively these studies suggest that concurrent training results in greater anabolic cellular environment and does not inhibit increases in muscular size, questioning the incompatibility of simultaneous strength and endurance signaling pathways. Additionally, these studies lend additional support to the observation that program design features play a role in dictating interference to desired adaptations [11, 126].

Simultaneous improvements in strength, power, and endurance are the desired training outcomes for many athletes. It has been demonstrated that athletes completing concurrent training experience smaller interference, no interference, or even synergistic enhancement of adaptive responses compared to recreationally active or untrained individuals [23, 24, 27, 47]. Although it is possible that the initial fitness level of athletes in these studies was responsible for decreased prevalence or complete ablation of interference, this seems unlikely as highly trained participants in other studies have demonstrated significant interference [3, 118]. Therefore the lack of interference or enhancement of the response of athletes in these studies [23, 27, 47] may be attributed to some other variable.

Team sport based concurrent training study design differs greatly from the majority of other concurrent training studies published in two major ways. First, rarely do scheduling and

time constraints of active athletic teams allow for the traditional 3 group experimental set up. Far more commonly the team is divided into two groups; one which continues with normal practice and training and a second whom completes additional resistance training. Since data have not been collected from athletes completing only resistance training for comparison, reports of synergistic enhancement or the absent interference must be interpreted with caution. It remains plausible that interference in some capacity, especially to measurements of power, may remain undetected due to lack of control group comparisons. Secondly, major difference exists in the endurance training protocols utilized by studies involving athletes and non-athletes. When current athletes are recruited as subjects, protocol design generally revolves around established practice and conditioning framework while other variables are manipulated. Conditioning protocols of such studies have been described in the literature as: "2-3 high intensity 20-30 minute training sessions per week" [47], and team practice sessions described as: "carrying a high degree of energy-system conditioning stimulus" [47]. However terms such as "high intensity" and "energy system conditioning" are poorly defined making determinations as to the true volume and intensity of conditioning stimulus difficult.

Only two studies [23, 24] to date have used a HIIT based condition, when assessing the effects of concurrent training. Balabinis et al. [23] observed that preseason concurrent training with basketball players led to synergistic enhancement of both strength and aerobic adaptive responses. Study duration was 7 weeks and employed endurance training which gradually transitioned from 5 miles of steady state running at 70% VO_{2max} in week 1 to 100, 200, and 400 M sprints by week 7 such that just under 50% of endurance training would qualify as HIIT [23]. Heavy resistance training was completed for the first 3 weeks of the study followed by 4 weeks of light strength training. Although no interference effect was observed, the unique design of

this training program makes between study comparisons difficult. In the only published concurrent training study utilizing SIT, Yung et al. [24] recruited professional soccer players and prescribed a SIT protocol consisting of 16 x 15 seconds sprinting at 120% of speed that elicited a player's VO_{2max} completed at a 1:1 work to rest ratio. Half of the team completed only soccer training while the other half received additional SIT and resistance training. The concurrent training group showed significant and greater improvements in 1RM back squat, 1RM bench press, vertical jump height, and 10 / 30 M sprint times than the soccer training only group [24].

While neither of these studies [23, 24] gathered data on resistance training control groups for comparison, it remains possible that the additive adaptive effect of concurrent training in both studies can be explained by greater adaptive commonality between HIIT and resistance training. Indeed, cellular responses to endurance and resistance training exist at opposite ends of the adaptive continuum [78]. In the most general of terms, endurance training leads to increased resistance to fatigue at submaximal workloads and mitochondrial biogenesis while resistance training results are driven by neural adaptations and increases in MPS which over time decreases both oxidative enzyme and mitochondrial density. In contrast, fiber type transformation, type IIa fiber hypertrophy rates, and energy system utilization following HIIT more closely resembles adaptations observed with resistance rather than endurance training [36, 61, 77]. This observation led to the conclusion that adaptive responses to HIIT and resistance training could be more compatible and possibly complementary to one another [11, 13]. Many of the original studies that examined concurrent training and exhibited an interference effect utilized at least in part endurance training protocols lasting between 20 and 60 minutes at 70-85% of VO_{2max} [4, 46, 117]. Others utilized long interval high intensity endurance training

consisting of 5 minute intervals which resulted in oxygen consumption levels near VO_{2max} at the conclusion of each interval, but still found the presence of adaptive interference [2, 5]. While this long interval high intensity endurance training protocol and others like it could technically be classified as HIIT, the adaptive response it initiates is closer to that of endurance training rather than SIT or the conditioning programs completed by athletes in the studies above [5, 24, 47].

When compared across multiple studies, moderate negative correlations (r = -.75) have been reported between frequency and duration of endurance training and amount of interference that is observed [11]. Both HIIT and SIT have been shown to result in similar gains in aerobic fitness and performance improvements compared to traditional endurance training requiring only a fraction of the training volume [40, 42, 73]. It may be that the use of high intensity or SIT as a part of a concurrent training plan could maximize both resistance and aerobic adaptive gains both in the general public as well as athletic populations. To the best of this researcher's knowledge, no study to date has examined the effect of concurrent training utilizing SIT as the stimulus to stimulate aerobic adaptation. Furthermore the effect of HIIT-based concurrent training has not been examined in women regardless of training status.

Conclusion and Purpose

A significant body of research has been compiled over the last three decades examining the effects of concurrent training on a multitude of strength, power, performance, and molecular variables [2, 3, 5, 8, 14, 17, 20, 23-26, 116, 117]. Contradictory findings have been reported in response to concurrent training including interference between, identical responses of, and synergistic enhancement of training adaptations when strength and endurance are trained together. Between study differences in exercise mode, frequency, and volume utilized as well as

dependent variable selection have all demonstrated the capacity to modulate the level of adaptive interference observed [11, 46]. Furthermore, power measurements experience inhibited adaptation far more frequently that other measures of strength [11] in spite of no adaptive interference to measures of neural activation [8, 18].

Two of the most widely accepted hypotheses proposed to explain reported interference between strength and endurance adaptations have been overtraining [2, 9] and an incompatibility of simultaneous activation of signaling cascades initiated by resistance and endurance training respectively [12, 13, 15]. Residual fatigue and chronic glycogen depletion resulting from overtraining were initially proposed as causative factors causing adaptive interference in studies that utilized large training volumes [2], or dual daily training schedules [20, 117, 118]. However subsequent studies demonstrated that the presence of adaptive interference persist even when training volume was drastically reduced [5], or when endurance and strength training were completed on separate days [8]. Taken together, these studies demonstrate that overtraining alone cannot fully explain the presence of interference. Endurance training derived upregulation of AMPK has been suggested to inhibit post resistance training-based increases in MPS via phosphorylation of TSC 1/2 which inhibits mTOR activity [12], providing a biochemical link between endurance and strength training adaptations [121]. However, acute increases in cellular anabolic conditions have been reported with concurrent training when compared to strength training alone [25]. When repeated chronically this increased anabolic environment resulted in greater hypertrophy and muscle CSA [26], suggesting that concurrent training, at least under some circumstances, results in synergistic enhancement, not inhibition of muscular hypertrophy.

Improved adaptive responses across all measured variables including power in response to concurrent training has been reported by several studies which utilized athletes as participants [23, 27, 47]. The aerobic conditioning utilized in the each of these studies more closely resembled HIIT than traditional endurance training. While a significant amount of research has been completed, the adaptive response to concurrent training employing high intensity or sprint interval training in active but not trained subjects remains unknown. Furthermore, knowledge of the effect of concurrent training in all female subject pools is lacking or nonexistent when SIT protocols are employed. Therefore the purpose of this investigation was to determine if SIT performed concurrently with resistance training resulted in adaptive interference, had no effect, or caused synergistic enhancement to measures of strength, power, and VO_{2max} in recreationally active females when compared to resistance training alone.

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CHAPTER III

JOURNAL MANUSCRIPT

ABSTRACT

PURPOSE: The purpose of this investigation was to determine if sprint interval training (SIT) performed concurrently with resistance training resulted in adaptive interference, had no effect, or caused synergistic enhancement to measures of strength, power, and VO_{2max} in recreationally active females when compared to resistance training alone. METHODS: 28 healthy females (20.3 ±1.7 yrs, 35.4 ± 4.0 $ml \cdot kg^{-1} \cdot min^{-1}$ VO_{2max}, 112.7 ± 17.0 lbs. 1 repetition max (1 RM) back squat were asked to complete a 12 week resistance training study. Preliminary and post testing consisted of 1 RM back squat, maximal isometric squat, rate of force development (RFD), cycle ergometer based anaerobic power evaluations, lactate threshold (LT), and VO_{2max}. Following initial testing, participants were matched according to 1RM back squat and VO_{2max} values and randomly assigned to one of two groups: concurrent training (CT) that completed both resistance and SIT protocols, and resistance training (RT) which only completed the resistance training protocol. Training was completed 3 days per week and lasted for 11 total weeks. All resistance training was completed in the morning with each participant completing the protocol at the same time each day. Separated by at least 4 hours, CT participants returned and completed SIT. RESULTS: 1 RM squat and maximal isometric force values were significantly elevated following training in both RT and CT (both $P < 0.01$). RFD was not significantly altered in

either group. Modified Wingate testing revealed significant increases in peak and mean anaerobic power values in both ST ($P < 0.05$) and CT ($P < 0.01$) with no statistical difference between group responses. VO_{2max} also increased as a result of resistance and concurrent training ($P < 0.01$). Predicted zero incline velocity that would elicit VO_{2max} (V_{max}) values were significantly elevated in both groups ($P < 0.01$) although concurrent training resulted in a significantly greater adaptive response ($P < 0.01$). LT values were not affected by training, although the velocity associated with LT (V_{LT}) increased significantly in both groups following training ($P < 0.01$). CONCLUSON: These data indicate that resistance training in isolation and sprint interval based concurrent training result in identical improvements to measures of strength, power, and VO_{2max} with no indication of adaptive interference. Only V_{max} adaptations supported the hypothesis of synergistic enhancement. These findings may be the result of commonalities between the adaptive responses to sprint interval and resistance training.

INTRODUCTION

The human neuromuscular system demonstrates a high degree of plasticity allowing skeletal muscle tissue to adapt favorably to specific tasks to which it is subjected. Adaptive responses to resistance training, which is characterized by short duration maximal contractions, include muscular hypertrophy, fast to slow transitions in type II fibers (IIx to IIa), and neural adaptations which cumulatively result in increased strength with little or no effect on aerobic fitness [15, 29, 80, 81]. At the other end of the adaptive continuum lies endurance training, characterized by long duration, submaximal, rhythmic contractions. Endurance training ultimately results in an increased capacity to load, transport, and utilize oxygen which derives from increased stroke volume, cardiac output, mitochondrial biogenesis, and angiogenesis [15, 33, 37]. Given the complexity of divergent signaling responses to endurance and resistance training, which operate at opposite ends of the adaptive continuum, it has been proposed that optimal adaptation to each is not possible when performed at the same time [15].

The combination of resistance and aerobic exercise into a single program, in an effort to attain adaptations specific to each, is known as concurrent training. Research into the effect of concurrent training, which has been conducted for over 3 decades, has produced contradictory results. Hickson et al. [2] first reported that completion of both endurance and strength training simultaneously resulted in compromised strength gains. The nature and causes of adaptive interference observed with concurrent training has been

debated ever since. In agreement with this initial finding, several authors have reported inhibited adaptive responses in measures of maximal strength [2, 5, 117] , power [3, 17, 117], and rate of force development (RFD) [8, 18] following concurrent training, a phenomenon termed the interference effect. Conversely, other investigators have failed to detect the presence of adaptive interference with concurrent training reporting identical increases to both strength [8, 16-20, 22] and power [7, 20] measurements. Furthermore, others have reported synergistic enhancement of training adaptations when simultaneous resistance and aerobic exercise are performed [23-27] . Adaptive responses are dictated by a host of factors including sex, initial fitness level, exercise intensity, duration, frequency, mode, and nutritional status. Ultimately a combination of these factors influence the physical and biochemical adaptations which occur in response to training [28].

Sub-optimal adaptation with concurrent training has been attributed to both protocol design variations [10], which could contribute to overtraining based interference, and an incompatibility of signaling cascades mediating cellular responses to training at opposite ends of the adaptive continuum [12, 13, 121]. Chronic glycogen depletion, residual fatigue, and inadequate recovery have been suggested as contributing interference factors resulting from protocols utilizing high volume or multiple sessions per day training [2, 10]. However protocols with significantly reduced training volumes and alternate day programming, which are unlikely to have caused overtraining, have also displayed inhibition to adaptive results following concurrent training [5, 8]. These studies indicate that the greater volume of work completed with concurrent training protocols is not a prerequisite for the existence of adaptive interference.

Alternatively, given the complexity of signaling pathways initiating divergent adaptive responses, it has been proposed that simultaneous optimal adaptation to endurance and resistance exercise is not possible [12, 13, 121]. Both animal [124] and human [121] studies have provided evidence of a biochemical link between signaling pathways which has been proposed as a mechanism of interference [12, 13]. Endurance exercise results in increased 5'adenosine monophosphate activated protein kinase (AMPK) activity, which through phosphorylation of tuberous sclerosis complex 1/2 (TSC 1/2) has an inhibitory effect on the phosphatidylinositol 3-kinase (PI3-k) - Akt – mammalian target of rapamycin (mTOR) cascade via interaction with mTOR [15]. However, recent studies by Lundberg et al. [25, 26] reported synergistic enhancement of muscle hypertrophy. A single bout of concurrent training resulted in a greater anabolic stimulus as evidenced by greater phosphorylation of mTOR and ribosomal protein S6 kinase (p70 S6k) than did resistance training alone [25]. Furthermore, when repeated over 5 weeks of training, this group [26] reported significantly greater increases in muscle size following concurrent training. Collectively, these results [25] suggest that concurrent training does not always result in AMPK derived inhibition of strength adaptations as previously suggested [12, 13, 121]. A recent meta-analysis by Wilson et al. [11] revealed that only power and not strength or hypertrophy measurements demonstrated a significant adaptive incompatibility following concurrent training.

Dudley et al. [5] were first to report inhibited adaptive responses with high but not low velocity contractions. With subsequent studies [3, 8, 11, 17, 18, 117] finding additional evidence of power-based adaptive interference compared to measures of strength or hypertrophy adaptations, it has been suggested that indices of power are more susceptible to

inhibited adaptation than are other measures of strength [11]. Alternatively, others [23, 24, 27, 47] have reported either no inhibitory effects, or synergistic enhancement to measurements of power following concurrent training. Two protocol variables, active athlete subject population and HIIT based aerobic training, unique to these studies [23, 24, 47] could explain the adaptive enhancement that was observed. In contrast to endurance exercise, high intensity interval and resistance training display more similarity than differences in their adaptive profiles [11, 36]. Furthermore, Sprint Interval Training (SIT) has been shown to result in slow to fast fiber type transitions, an adaptation that presumably could enhance strength development [61, 75-77]. While synergistic enhancement across all adaptive measures has been demonstrated in athletes, training schedules prevented comparison to strength training only control groups in these studies allowing for the possibility that adaptive interference is present in some capacity, but remains undetected.

High intensity and sprint interval exercise have been proposed as time efficient alternatives to traditional endurance training as lack of time is the most commonly cited barrier to physical activity [40-42]. However the adaptive response of strength and power responses to HIIT or SIT based concurrent training in recreationally active or untrained populations has yet to be examined. Additionally, while a relatively large body of knowledge regarding the effects of various concurrent training protocols on subsequent adaptation is available, comparatively few studies have examined female populations. Therefore the purpose of this study was to determine if strength performance was impaired following concurrent resistance training and sprint interval running in untrained but active women. We hypothesized that the collaborative nature of adaptive processes resulting from

sprint interval and resistance training would result in enhanced strength and power adaptations while improving VO_{2max}.

METHODS AND PROCEDURES

Methods

Participants

Female volunteers between the ages of 19 and 30 were invited to participate in this study provided that they met the following inclusion criteria: 1) current exercise schedule that did not exceed 3 days per week of either aerobic or resistance training as determined by current activity questionnaire (Appendix A); 2) healthy as determined by Physical Activity Readiness Questionnaire (PAR Q) Screening (Appendix B); 3) and not currently taking any medications that would interfere with experimental variables (Appendix B) and; 4) VO_{2max} between 30 – 50 $ml \cdot kg^{-1} \cdot min^{-1}$. An all-female subject pool is warranted as no study to date has examined the effect of concurrent supramaximal HIIT and resistance training in women. Twenty eight participants met the inclusion criteria and volunteered to participate. Subject characteristics are summarized in table 6.

Preliminary Procedures and Assessments

Participants who met the initial written screening criteria were then scheduled for an additional screening visit to the Auburn University Thermal lab. This investigation was reviewed and approved by the Auburn University Institutional Review Board (IRB). Upon arrival to the lab for additional screening, participants were given and asked to sign an informed consent document (Appendix C). Volunteers were then asked to complete a

VO_{2max} test to determine initial aerobic fitness levels. Those volunteers who met all inclusion criteria then continued with collection of remaining anthropometric data and scheduled times for acclimation visits. A full study schedule can be viewed in Figure 8.

Anthropometric and Physiological Assessment

Anthropometric data including height and weight were obtained. Weight was measured to the nearest 0.1 kg using a calibrated scale (Michelli Scales, Harahan, LA) and height was assessed to the nearest 0.25 inches with a standiometer. Body composition was then obtained via Dual-emission X-ray Absorptiometry (DXA).

Acclimation

Over the next week, participants were scheduled for a minimum of three additional visits to the Auburn University Thermal laboratory where all resistance training and testing procedures were covered as described in Table 1. Participants were then given time to practice and become familiar with movements and testing protocols. Extra acclimation visits were offered to any participant who felt she needed more exposure to, or was uncomfortable with any portion of the study. Efforts were made to ensure that participants were comfortable with the exercises and tests, but that acclimation visits were not strenuous enough to constitute a training stimulus.

Preliminary Testing

Preliminary testing consisted of VO_{2max}, three 1 RM back squat efforts, maximal isometric squat, three modified Wingate tests completed with different resistance loads, and lactate threshold testing in a schedule summarized in Table 2. Each testing day began with 5 minutes of easy jogging, some light stretching, and any specific warm-up needed to prepare for daily activity. Participants kept a dietary record of all food and beverages consumed for

the week of testing including the two days before testing began. Participants were instructed to continue normal dietary practices and not to make any drastic dietary adjustments over the course of the study.

VO_{2max} Testing –

Prior to testing, participant hydration status was assessed using a urine refractometer (Atago Co., Tokyo, Japan). Only participants whose urine specific gravity (USG) was < 1.020 were allowed to complete VO_{2max} testing. Participants were then asked to warm-up on a treadmill for 5 minutes. Participants were free to adjust treadmill speed during this warm-up and were asked to determine a speed that they would be comfortable jogging for a minimum of 20 minutes. The self-selected speed was used as the initial velocity for VO_{2max} testing. Four submaximal stages each lasting 3 minutes were completed by each individual with 0° incline. Every three minutes for the first 9 minutes treadmill speed was increased by 0.5 miles per hour. After the completion of the 4^{th} stage (at minute 12) additional 1 minute stages were completed with a 2° increase in treadmill grade and no change to running speed, until voluntary exhaustion occurred.

Oxygen consumption (VO_2) measurements were collected by an automated metabolic testing system (True Max 2400 Metabolic Testing System, Parvo Medics, Salt Lake City, UT). The highest oxygen uptake average over 45 seconds was considered VO_{2max} if two of the following criteria were met: 1) respiratory exchange ratio (RER) ≥ 1.15, 2) heart rate within 10 beats per minute of age predicted max, and 3) volitional exhaustion / inability to continue the test. Average VO_2 values from each of the first four stages were used to create a regression equation unique to each individual. Using VO_{2max}

and the regression equation, zero grade velocity that would elicit VO_{2max} (V_{max}) was predicted. Vmax values were then used both to prescribe SIT velocity for each individual.

1 RM Back Squat

Initial 1 RM testing values in untrained participants have been shown to be unreliable indicators of initial strength levels due to unfamiliarity with the movement. Rhea et al. [127] successfully utilized a 3 day initial 1 RM testing protocol which has delivered reliable measures of initial strength in untrained populations. Values obtained on the second two days of this three day testing protocol have shown high correlation ($r = 0.98$) between days two and three with the greatest load lifted recorded as the 1 RM value [127, 128]. After a general warm-up participants completed 10 repetitions of a body weight squat followed by 5 repetitions with a 45 lb. barbell. Following 2 minutes rest, 5 repetitions were completed at an estimated 50% of the participant's 1 RM. After an additional 2 minutes rest a set of 3 was completed with an estimated 70% of max. Next, following 3 minutes rest, 1 repetition was performed with progressively heavier weight such that a 1 RM max value was attained in 3 sets or less. The first of these three back squat days was conducted on the last of the familiarization visits. A conservative loading approach was utilized on day one to gauge initial strength levels for each individual and to avoid injury. The subsequent two back squat days were based on the values from day 1.

Maximal Isometric Squat –

Isometric force-time curves, maximal isometric RFD, and maximal isometric squat force were measured on a force platform (AMTI ORG6-7, Watertown, MA). Set up for this test was included barbell placement against safety squat rack supports such two requirements were met for each participant: 1) participant knee angle was 140° when in

position prior to maximal contraction and; 2) that during the maximal effort, no movement occurred to any part of the apparatus. After initial measurements and adjustment, participants then were asked to stand on a force platform with the barbell resting on their backs and in light contact with the safety squat supports. Participants were instructed to maintain contact with supports, but not to exert any additional upward force until instructed to do so by auditory command. When signaled, participants exerted as much force as possible as quickly as possible upward against the barbell for a period of 3 seconds. Each participant completed three trials separated by a minimum of 2 minutes rest. Force values were recorded at a rate of 200 Hz for later evaluation. Maximal isometric force was calculated as the maximal force output attained in any of the three trials minus the average force exerted by the participant plus the barbell prior to maximal contraction. Maximal RFD (N per second) was analyzed and defined as the greatest increase in force over a given 50 ms time period, a method previously used and validated by Hakkinen et al. [103].

Maximal Anaerobic Power

On three separate occasions participants were asked to complete 10 seconds of maximal cycling exercise on Velotron cycle ergometer (Dynafit Pro, Racer Mate, Seattle WA). Each test was conducted with a different resistance with the goal of attaining maximal peak and mean anaerobic power. Testing was completed using Wingate software (Racer Mate, Seattle WA) which allowed for the test duration and resistance to be easily manipulated. Once equipment was properly adjusted, participants pedaled self-paced against 40 watts resistance for 20 seconds followed by an 8 second ramp up period in which maximal revolutions per minute were attained against no resistance. Following this unloaded acceleration a resistance equal to 7.5, 8.5, or 9.5 % of the participant's mass was

added; power output was recorded for 10 seconds, and both peak and mean anaerobic power were calculated for each participant. After each test participants completed at least 3 minutes of supervised light recovery.

Lactate Threshold

Lactate Threshold testing consisted of continuous treadmill running starting at 50% of predicted V_{max} with lactate measurements and a 5% velocity increase occurring every 4 minutes. A small amount of capillary blood was collected via finger puncture at the end of every 4^{th} minute and whole blood lactate concentration ($[La^-]$) was measured by a handheld lactate analyzer (Lactate Pro, Arkray, Inc. Kyoto, Japan). Testing was concluded once two La^- values over 4 mM were obtained or a clear exponential rise in whole blood lactate level was observed.

Post Testing

Post testing was conducted immediately following the 11^{th} week of training and repeated measures of all physiological variables were obtained. A summary of this schedule can be viewed in Table 5.

Experimental Procedures

Following preliminary testing, participants were placed into one of two experimental groups: concurrent resistance and SIT (CT), and resistance training (RT) in a matched pairs design based on initial 1RM back squat, peak anaerobic power, and VO_{2max} values. Training lasted a total of 11 weeks with a planned unload week taken following the 6^{th} week of training. Periods of reduced training (unload weeks) which was first proposed by Stone et al. [129], allow the body to dissipate accumulated fatigue, and has been associated with increases in strength gains and subsequent training loads [79, 125, 129]. Training took place

three days per week with all resistance training occurring before noon. Participants assigned to RT completed only the resistance training protocol while participants assigned to CT performed both resistance and SIT protocols on the same day separated by at least 4 hours such that SIT was completed in the afternoon. This time interval between sessions has been shown to be adequate to avoid fatigue based interference between sessions [3]. To eliminate post exercise nutritional variation, 8 oz. of chocolate milk was provided post workout to each participant. The nutritional profile of chocolate milk has been shown to stimulate muscle protein synthesis and aid in recovery [108]. After week 2, week 6, and prior to post training testing, participants were returned a copy of their dietary log and asked to ensure that no large dietary changes had occurred since the initiation of the study.

Resistance Training Protocol

Each participant completed resistance training at the same time each day. A general warm-up was completed prior to each training session and included light jogging and dynamic mobility work. The resistance training protocol consisted of two unique training schemes performed on alternate days throughout the study such that each protocol is completed 3 times every 2 weeks. The protocol is summarized in Table 3. This study utilized an undulating periodization model which has been shown to be at least [130, 131], if not more effective [127] at eliciting strength gains that linear periodization, such that intensity and volume were altered each time a resistance training protocol was repeated (Table 4). Three separate loading and repetition schemes were used for each protocol such that each was completed once every two weeks.

At the initiation of training, participants were allowed to self-select the weight lifted on all exercises for the first two training days of each resistance training protocol with the

exception of back squats for which loads were prescribed based on initial testing results. In the following weeks, 1 RM values were estimated utilizing the Wathan formula [132], whose 1 RM estimation accuracy which has been validated [133]. Using the Wathan formula, 1 RM estimates were calculated for each exercise using the maximal load successfully lifted for the prescribed number of repetitions. These 1 RM estimations were then used to prescribe subsequent exercise loading such that 70, 82.5, and 87.5 % of max were assigned on 10, 5, and 3 repetition days respectively. These percentages were selected as they represent 5% less than the theoretical max that can be lifted for a given rep scheme [28]. One open set was left on each exercise allowing participants to increase the load beyond prescribed levels as they were able thus providing more accurate 1 RM estimates for future loading.

Sprint Interval Training

SIT took place on the same 3 days per week as resistance training in the afternoon a minimum of four hours after the morning session and was only completed by participants assigned to the CT group. Following a brief general warm-up and dynamic flexibility, each CT participant completed an individualized SIT protocol based on the results of the initial VO_{2max} testing. The employed running based SIT protocol mimicked a cycle based SIT protocol used by Tabata et al. [74], which was shown to be effective at increasing VO_{2peak}, and anaerobic capacity measures. SIT consisted of 8 repetitions of 20 seconds of treadmill running, alternated with: 10 second passive recovery periods for a total exercise time of 4 minutes. The initial six sessions (2 weeks) were completed at 110, 115, and 120% of the predicted V_{max} determined from initial VO_{2max} testing. Once all 8 intervals were completed in 2 subsequent sessions, treadmill velocity was increased by an additional 3% (.1-.2 mph).

If 6 - 7 intervals were completed, or 8 completed intervals were not accomplished on consecutive days then the velocity was left unchanged. If at any point in training fewer than 6 intervals were completed then the running velocity was decreased by 1.5% for the subsequent training session. Average peak and mean percentages of VVO_{2max} attained during training were 154.5 ± 11.3 and 147 ± 9.1 respectively.

Statistical Analysis

Standard statistical methods were used for the calculation of means, standard deviations (SD). Descriptive statistics are presented as mean ± SD. The effects of training on principal dependent variables (1 RM Squat, maximal isometric force, RFD, anaerobic power tests, VO_{2max}, and LT) were analyzed using a 2 (time) x 2 (training intervention) mixed model ANOVA with random effects of participant and repeated measures for time. Planned pairwise comparisons were made both between training interventions and between measurement times within each protocol for all dependent variable measurements. The significance level for this study was set at $P < 0.05$. Statistics were analyzed using R software which is freely available on-line (URL http://www.R-project.org/).

Results

Participants

Each group began the study with 14 participants; however two participants from the CT group withdrew due for personal reasons. Pre-intervention, no significant differences were observed between the groups with respect to measures of height, mass, body composition, or lean mass (all $P > 0.5$). Furthermore, of all preliminary measures, only maximal isometric force exhibited significant differences between groups with greater initial strength observed in CT (113.2 ± 29 kg) as compared to RT (85.7 ± 23.9 kg) ($P < 0.05$). Of the 26 participants who completed the study, all completed at least 90% of the training sessions: CT (98.1 ± 1.3 %), RT (98.6 ± 1.4), which was required for inclusion in data analysis. Baseline descriptive characteristics of the participants are presented in Table 6.

Anthropometric Data

No group-by-time interactions were detected to any anthropometric measurements (all $P > 0.11$). Training did not significantly alter body mass, although composition was significantly effected following both resistance only and concurrent training. Body fat percentage decreased significantly by 8% only in RT (35.2 ± 6.7 to 32.5 ± 7.5, $P < 0.01$), while the 4% decrease resulting from concurrent training failing to reach significance (34.3 ± 7.5 to 33.1 ± 7.0, $P = 0.068$). Measures of lean mass increased significantly by 4 and 6% in RT and CT (both $P < 0.01$) with no difference between group response ($P = 0.45$). Results indicate that both groups had similar adaptive responses in total body mass and lean mass

increases, but that only RT displayed significant decreases in fat percentage.

Strength / Rate of Force Development

Significant increases in both 1 RM back squat and maximal isometric force measurements were observed within both groups following 11 weeks of training (Fig.1 and 2), with no group-by-time interactions detected (both $P > 0.47$). 1 RM back squat values significantly increased following training by 37% in RT (51 ± 10 to 69.2 ± 11.4 kg, $P < 0$ .01) and 34 % in CT (51.2 ± 4.2 to 68.2 ± 4.7 kg, $P < 0.01$). Maximal isometric force values also significantly increased following training in both RT by 33 % (85.7 ± 23.9 to 110.1 ± 23.8 kg, $P < 0$ .01) and in CT by 26% (113.8 ± 29 to 138.5 ± 30.3 kg, $P < 0.01$). RFD measurements were not altered by training with equal non-significant increases between groups (both $P > 0.11$). Results indicate that both protocols resulted in similar significant increases in maximal strength and non-significant increases in RFD.

Anaerobic Power

Peak and mean power values increased significantly as a result of training in both groups under all three resistance loads (Fig. 3 and 4), with no group-by-time interactions detected (all $P > 0.31$). RT resulted in increases to peak anaerobic power of 10% ($P < 0.01$), 10% ($P < 0.01$), and 4% ($P < 0.05$) to resistance loads of 7.5, 8.5, and 9.5% of mass in kg respectively. RT mean wattage values displayed a similar pattern of improvement with increases of 9, 12, and 7% (all $P < 0.01$) under the same conditions. Likewise, CT resulted in increased peak anaerobic power with gains of 9, 8, and 5% (all $P < 0.01$) to resistance loads of 7.5, 8.5, and 9.5 % mass (kg). CT mean wattage was also significantly elevated following training displaying increases of 11, 8, 9% (all $P < 0.01$). Results indicate that all measures of anaerobic power were significantly increased by both protocols.

Aerobic Measures

VO_{2max}, predicted V_{max}, and V_{LT} all displayed significant improvements as a result of each training protocol. VO_{2max} increased significantly by 6% after resistance training (35.0 ± 4.0 to 36.9 ± 4.1 ml/kg/min, $P < 0.01$, Fig. 5), and by 9% following concurrent training (35.8 ± 4.2 to 38.8 ± 4.0 ml/kg/min, $P < 0.01$, Fig. 6) with no difference in gains observed by either group ($P = 0.29$). Both protocols also resulted in V_{max} increases of 9% in RT (6.1 ± .7 to 6.7 ± .8 mph, $P < 0.01$) and 17% in CT (6.3 ± .9 to 7.3 ± 1.0 mph, $P < 0.01$). A significantly group-by-time interaction was detected with CT indicating that concurrent training resulted in significantly larger V_{max} improvements ($P < 0.01$, Fig. 7) than RT. Significant improvements to V_{LT} values of 18% (4.6 ± 0.6 to 5.4 ± 0.6 mph, $p < 0.01$) and 13% (4.6 ± 0.8 to 5.2 ± 1.0 mph, $P = 0.01$) were observed in RT and CT respectively with no difference observed between the gains of each group ($P = 0.39$). No significant changes were observed to LT values in either group as a result of training (both $p > 0.5$). Results indicate that both resistance training and concurrent training resulted in similar significant improvements in VO_{2max}, and V_{LT} while V_{max} improvements were significantly greater following concurrent training.

Discussion

To the best of our knowledge this was the first study to investigate the effect of concurrent training exclusively employing SIT on measures of strength, power, RFD, and aerobic fitness in a non-athletic population. The purpose of this study was to determine if SIT-based concurrent training resulted in adaptive interference, identical adaptation to, or synergistic enhancement of, strength, power, and RFD which have been shown to display adaptive interference following concurrent training [3-8, 14, 17, 18, 116, 117], when compared to strength training in isolation. The primary findings of this investigation were: 1) 1 RM and maximal isometric back squat values were significantly increased post training in both groups with no significant differences between responses; 2) RFD was not significantly increased by either training protocol; 3) peak and mean power values displayed equal significant increases following training across all resistance loads and for both training groups; 4) VO_{2max} and V_{LT} measurements significantly improved as a result of training while LT values were unaffected with no differences in responses between groups and; 5) CT exhibited significantly greater improvements in predicted Vmax than ST. Subsequently, results of this study are in agreement with others that have observed neither an interference effect nor synergistic enhancement of the adaptive process as a result of concurrent training to measures of strength [8, 16-20, 22] and power [7, 20].

Maximal Strength

Resistance training, when paired with aerobic conditioning, has been shown to result in adaptive interference and decreased strength gains in some [2-8] but not all [8, 16-20, 22] cases. Conflicting findings have been attributed to protocol design variations including exercise mode, duration, intensity, frequency, and participant fitness level [10, 11]. In the present study significant increases in both measures of maximal dynamic (33.6 – 37.4 %, $P < 0.01$) and isometric (25.6 – 32.9%, $P < 0.01$) force were observed following training with no differences in the adaptive response between ST and CT. These results indicate that neither interference nor synergistic enhancement of strength based adaptive responses occurred as a consequence of SIT-based concurrent training.

The average observed increases in 1 RM back squat strength in this study of 33-37% are greater than previously reported in similar participant populations of previously untrained women [134], or athletes [23, 24]. Utilizing linear periodization and untrained female participants, Kraemer et al. [134] reported that 12 weeks of training 3 days per week yielded increases of 24 % for 1 RM back squat values following both high (8-12) and moderate (3-7) volume resistance training. Similar strength gains (20-24%) have been reported in athletic populations following only 8 weeks of training [23, 24]. The greater increases observed in the present study could be a result of the prescribed undulating periodization model which has been shown to be more effective than linear periodization schemes at increasing maximal strength [127]. Additionally, while the current study duration was slight longer than the above studies [23, 24] which utilized athletes, it is unlikely that two additional training weeks alone could account for the observed discrepancy in percentage strength gains. Greater maximal strength observed in this study is

more likely attributable to the low initial muscular fitness of participants in addition to the novel application of undulating periodization-based strength training to this population. Equal significant increases were also observed in measures of maximal isometric strength between CT and RT. This finding is in agreement with other published literature [5, 8, 18], and shows no indication of adaptive interference to isometric strength measures. Both Hakkinen et al. [18] and Mikkola et al. [8] found identical 20-28% increases in isometric strength across all training groups in similar active but untrained populations as those who participated in the current study. Our finding along with these studies [8, 18] support the initial finding by Dudley et al. [5] that isometric and low velocity contractions were not affected by concurrent training.

In contradiction with previous reports which found either interference to [3-7, 14, 116] or enhancement of strength adaptations following concurrent training [8, 27], results of the present study support the growing body of literature reporting similar strength adaptive responses between concurrent and resistance only training [8, 16-20, 22, 23].

RFD / Power

Dudley et al. [5] were the first to report the existence of contraction velocity specific interference following concurrent training leading to the suggestion that power may be more susceptible to adaptive interference than strength. Other researchers [8, 18] have reported similar significant interference with RFD adaptations following concurrent training when compared to resistance training only controls (concurrent - 0%, strength only 38 – 50% increase). These studies [8, 18] add to a growing body of research supporting power specific adaptive interference resulting from concurrent training [3, 5, 8, 9, 11, 18, 135], although power specific interference has not been ubiquitously reported [7, 20]. In addition, a recent

concurrent training meta-analysis revealed that only measures of power consistently demonstrated an interference effect [11]. Our findings, in disagreement with the aforementioned studies [5, 8, 18], provide no evidence of adaptive interference with RFD measures resulting from concurrent training, although equal between group improvements did not reach significance (RT – P = 0.11, CT – P = 0.22). Noteworthy differences exist between the experimental protocols used in these studies [5, 8, 18] and our own. Both Hakkien et al. [18] and Mikkola et al. [8] utilized explosive resistance training for a portion (20%) of the total resistance training volume while our study did not, which could explain why RFD improvements did not reach significance by either RT or CT.

In addition to RFD analysis, peak and mean power values have been used to investigate power specific interference resulting from concurrent training. Typically these measures are obtained from Wingate testing consisting of 30 seconds of maximal effort sprinting on a cycle ergometer against 7.5% of participant mass (kg). The validity of anaerobic measures gathered from Wingate testing have been questioned as contributions of the aerobic system to energy production and thus total power output has been estimated between 18 [136] and 44 [137] %. Significantly greater peak power measurements have been attained utilizing a 10 second modified Wingate test which consequently was proposed as a more appropriate test of peak and mean anaerobic power [138].

Kraemer et al. [3] observed significant increases in peak and mean power outputs (17 and 20% respectively) following 12 weeks of resistance training with no significant changes resulting from concurrent training. Alternatively, Balabinis et al. [23] reported equal significant increases in peak Wingate power measures in both strength and concurrent training groups following only 8 weeks of training utilizing HIIT style conditioning. In

contrast to the findings of Kraemer et al. [3] and in support of research by Balabinis et al. [23], we observed no evidence of adaptive interference for either peak or mean anaerobic power improvements as observed power improvements were equal between CT and RT (all $P < 0.03$). These findings do not support the concept of universal interference to all measures of power resulting from concurrent training.

The unique RFD and anaerobic power findings of the present study are likely a result of drastic differences in nonresistance exercise completed by concurrent training groups in the present study. It has recently been suggested that the adaptive response to sprint interval and resistance training harbor more similarities than differences [11]. If true, adaptive similarities between resistance and condition training could explain the lack of interference observed by the current investigation. Obtained peak and mean power values following training indicate that concurrent training neither aided nor inhibited maximal anaerobic power adaptations. It is possible that the extremely low volume of SIT utilized in this study did not reach a modality specific threshold necessary to initiate either significant adaptation to or interference with RFD measures. Therefore, further research is required to determine if RDF adaptations are selectively inhibited in response to sprint interval and explosive resistance training performed concurrently. Further investigations should employ explosive resistance training and evaluate different and higher volume HIIT and SIT protocols.

Aerobic Measures

Surprisingly, when pre and post VO_{2max} values were compared, both RT ($P < 0.01$) and CT ($P < 0.01$) exhibited significant improvements indicating that each protocol resulted in adequate cardiovascular stress to stimulate measurable adaptation. Significant aerobic improvements were not expected following resistance training as most researchers have not

reported increases in VO_{2max} following resistance training [2, 15, 139, 140]. However several studies [141-143] have reported increases to VO_{2max} values following resistance training in populations with low initial cardiovascular fitness. As the untrained participants of our study exhibited initial VO_{2max} values that were not far from those reported above [141-143], it is likely that this resulted in the significant improvements in VO_{2max} following resistance training.

Conversely, SIT has been shown to be an effective means by which to drive improvements in VO_{2max} [40, 42, 74, 75]. Our results, however, did not find significantly greater VO_{2max} improvements with the addition of SIT training when compared to resistance training alone ($p = 0.11$). The majority of research reporting significant maximal aerobic improvements resulting from SIT with extremely low exercise volume (< 6 minutes) has been conducted using cycle ergometers [40, 42, 74]. Similar studies employing running based SIT training have typically evaluated much larger training volumes (20 – 40 repetitions of 10 to 20 seconds maximal effort) [24, 75]. Our equal increase in VO_{2max} compared to RT may be due to SIT volume in the present study that was significantly less than has been previously employed with running based SIT protocols. This indicates that there may be a modality specific volume threshold required to drive aerobic adaptations from this type of training that was not reached.

In spite of identical VO_{2max} improvements, predicted V_{max} improvements revealed a significant group-by-time effect of concurrent training when compared to resistance training ($P < 0.01$). Greater improvements in V_{max} relative to similar improvements in VO_{2max} between groups suggest that SIT resulted in greater improvements in running economy. Neither training protocol resulted in significant changes in LT values (both $P > 0.5$). The

velocity at which threshold occurred however was significantly increased following strength ($P < 0.01$) and concurrent training ($P = 0.01$) which could be indicative of improved running economy, increased force production capacity of type I fibers, decreased lactate production at a given work load, increase lactate utilization / removal, or some combination of above explanations.

Anthropometric Data

Total body mass did not change in either group following training; however, body composition was significantly altered in both ST and CT. Surprisingly, fat tissue percentages decreased significantly in RT ($P < 0.01$), but failed to reach significance following concurrent training ($P = 0.068$). Skeletal muscle hypertrophy is documented as the primary adaptation to long term resistance training [104]. The chronic hypothesis originally proposed by Craig et al. [14] and further defined by several other researchers [12, 13, 15] suggest simultaneous activation of PI3-K – AKT – mTOR and AMPK – PGC-1α signaling cascades in response to resistance and endurance training respectively results in decreased mTOR activation via biochemical cross talk and impaired contractile protein synthesis. However other researchers have demonstrated synergistic enhancement of mTOR, P70 SK6, and 4E-BP1 activation which cumulatively resulted in greater muscular hypertrophy following concurrent training [25, 26]. In the present study both protocols resulted in significant increases in fat free mass (both $P < 0.01$), with no differences detected between group responses ($P = 0.45$). Fat free mass is composed of everything except fat tissue and no significant changes were observed in bone mineral density, or suspected to any non-muscle components of this measurement. It can be inferred from these findings that significant

muscular hypertrophy resulted from each protocol with no evidence of adaptive interference or synergistic enhancement following concurrent training.

Conclusion

This study found that 11 weeks of resistance or SIT-based concurrent training both resulted in similar significant adaptive responses in maximal muscular strength, power, anaerobic power, and cardiovascular fitness. Surprisingly, strength training resulted in significant VO_{2max} increases and body fat percentage decreases not typically associated with this type of training. It is likely that the low initial fitness status of participants is responsible for these findings. Only predicted V_{max} displayed any sign of adaptive synergistic enhancement following concurrent training with significantly larger increases, which suggest that sprint interval running results in greater running economy improvements than strength training in isolation. No indication of adaptive interference was detected in any performance variable. Measurements of power, which are traditionally the most susceptible to interference, demonstrated identical significant increases in peak and mean Wingate values and equal non-significant increases in RFD measures. It should be noted however that the SIT volume utilized in this study, which was drastically reduced compared to nearly all other concurrent training studies, may have failed to reach a threshold level necessary to initiate adaptive interference or synergism to adaptive responses. Additionally, no explosive resistance training was prescribed with this group of untrained participants. Further research into the interaction between adaptive responses produced by sprint or high intensity interval and resistance training is warranted and should include increased running-based SIT volume and / or high velocity resistance training contractions aimed at power development. Studies such as these may provide more definitive evidence for the existence of synergistic

enhancement or adaptive interference between concurrent HIIT and resistance training in untrained or recreationally active populations.

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Tables

Day 1	Day 2	Day 3
• Body Weight Squat • Isometric Squat • Vertical Jump	• Standing Press • Bench Press • Wingate Protocol • Bent over Row	• Deadlift • SIT Protocol • 1RM Back Squat walkthrough 1/3

Table 1 – Acclimation Schedule

Monday	Isometric Squat Anaerobic Power Test (1/3)
Tuesday	1 RM Back Squat (day 2/3) Anaerobic Power Test (2/3)
Wednesday	Rest Day
Thursday	Lactate Threshold
Friday	1 RM Back Squat (day 3/3) Anaerobic Power Test (3/3)

Table 2- Preliminary Testing Schedule

Protocol A	Protocol B
• Back Squat • Bent Over Row • Bench Press • Sit-ups	• Squat Jumps • Deadlift • Standing Press • Back Extensions

Table 3 – Resistance Training Breakdown

	Monday	Wednesday	Friday
Week 1	Protocol “A” 3 sets x 10 repetitions	Protocol “B” 3 sets x 10 repetitions	Protocol “A” 4 sets x 5 repetitions
Week 2	Protocol “B” 4 sets x 5 repetitions	Protocol “A” 5 sets x 3 repetitions	Protocol “B” 5 sets x 3 repetitions

Table 4 – Weekly undulating periodization scheme

Monday	Anthropometric Data Collection Isometric Squat Anaerobic Power Test (1/3)
Tuesday	1 RM Back Squat (day 2/3) Anaerobic Power Test (2/3)
Wednesday	Anaerobic Power Test (3/3)
Thursday	VO_{2max}
Friday	Lactate Threshold

Table 5- Post Testing Schedule

	Resistance Training	Concurrent Training
Anthropometric Data		
Age (yr)	20.4 ± 1.9	20.2 ± 1.5
Height (cm)	168.7 ± 2.2	170.8 ± 5.0
Mass (kg)	62.6 ± 8.2	63.3 ± 9.9
Body Fat %	35.2 ± 6.7	34.3 ± 7.5
Lean Mass (kg)	38.3 ± 2.5	38.9 ± 1.1
Strength / Power		
1 RM Back Squat (kg)	51.0 ± 10.0	51.2 ± 4.2
Isometric Squat (kg)	85.7 ± 23.9 *	113.2 ± 29 *
RFD (N/sec)	3862.8 ±1479	4,736 ± 1503
Anaerobic Power (% mass resistance)		
Peak Anaerobic Power		
7.5 (watts)	704.2 ± 110.4	709.3 ± 114.2
8.5 (watts)	820.1 ± 146.4	818 ± 146.1
9.5 (watts)	937.5 ± 160.4	938.4 ± 167.6
Mean Anaerobic Power		
7.5 (watts)	561.3 ± 77.9	543.8 ± 59.2
8.5 (watts)	601.0 ± 90.1	612.1 ± 91.7
9.5 (watts)	648.6 ± 86.9	641.0 ± 62.0
Aerobic Measures		
VO_{2max} (ml/kg/min)	35 ± 4.0	35.8 ± 4.2
V_{max} (mph)	6.1 ± 0.7	6.3 ± 0.9
Lt (mph)	4.6 ± 0.6	4.6 ± 0.8

Table 6 – Subject Characteristics. All values are presented as means ± standard deviations. * denotes significant differences between groups ($P < 0.05$)

	Pre	Post
Anthropometric Data		
Body Fat %	35.2 ± 6.7	33.3 ± 6.8 *
Lean Mass (kg)	38.3 ± 2.5	39.6 ± 2.6 *
Strength / Power		
1 RM Back Squat (kg)	51.0 ± 10.0	69.2 ± 11.4 *
Isometric Squat (kg)	85.7 ± 23.9	110.1 ± 23.8 *
RFD (N/sec)	3862.8 ±1479	4712 ± 1849.2
Anaerobic Measures (% mass resistance)		
Peak Anaerobic Power		
7.5 (watts)	704.2 ± 110.4	769.4 ± 103.7 *
8.5 (watts)	820.1 ± 146.4	892.3 ± 121.3 *
9.5 (watts)	937.5 ± 160.4	965.6 ± 148.7 *
Mean Anaerobic Power		
7.5 (watts)	561.3 ± 77.9	609.4 ± 63.2 *
8.5 (watts)	601.0 ± 90.1	666.5 ± 68.5 *
9.5 (watts)	648.6 ± 86.9	690.1 ± 75.8 *
Aerobic Measures		
VO_{2max} (ml/kg/min)	35 ± 4.0	36.9 ± 4.1*
V_{max} (mph)	6.1 ± 0.7	6.7 ± 0.8 *
Lt (mph)	4.6 ± 0.6	5.4 ± 0.6 *

Table 7 – RT Training Results. All values are presented as means ± standard deviations. * denotes significant differences from pre values ($P < 0.05$).

	Pre	Post
Anthropometric Data		
Body Fat %	34.3 ± 7.5	33.1 ± 7.0
Lean Mass (kg)	38.9 ± 1.1	41.0 ± 1.7 *
Strength / Power		
1 RM Back Squat (kg)	51.2 ± 4.2	68.2 ± 4.7 *
Isometric Squat (kg)	113.2 ± 29.0	138.5 ± 30.3 *
RFD (N/sec)	4,736 ± 1503	5434 ± 1699
Anaerobic Measures (% mass resistance)		
Peak Anaerobic Power		
7.5 (watts)	709.3 ± 114.2	769.6 ± 108.5 *
8.5 (watts)	818 ± 146.1	879.9 ± 144.3 *
9.5 (watts)	938.4 ± 167.6	984.1 ± 158.5 *
Mean Anaerobic Power		
7.5 (watts)	543.8 ± 59.2	605.2 ± 58.0 *
8.5 (watts)	612.1 ± 91.7	656.3 ± 62.5 *
9.5 (watts)	641.0 ± 62.0	697.6 ± 55.1 *
Aerobic Measures		
VO_{2max} (ml/kg/min)	35.8 ± 4.2	38.8 ± 4.0 *
V_{max} (mph)	6.3 ± 0.9	7.3 ± 1.0 *
Lt (mph)	4.6 ± 0.8	5.2 ± 1.1 *

Table 8 – CT Training Results All values are presented as means ± standard deviations. * denotes significant differences in adaptations between groups ($P < 0.05$)

	Resistance Training		Concurrent Training	
	Post Training	**% Change**	**Post Training**	**% Change**
Anthropometric Data				
Mass (kg)	63.5 ± 7.7	-1.6 ± 3.6	64.5 ± 10.1	-2.1 ± 2.7
Body Fat %	33.3 ± 6.8	-5.5 ± 4.5 **	33.1 ± 7.0	-3.8 ± 5.2
Lean Mass (kg)	39.6 ± 2.6	3.8 ± 2.1 **	41.0 ± 1.7	5.5 ± 2.3 **
Strength / Power Measures				
1 RM Back Squat (kg)	69.2 ± 11.4	37.4 ± 13.1 **	68.2 ± 4.7	33.6 ± 8.4 **
Isometric Squat (kg)	110.1 ± 23.8	32.9 ± 26.2 **	138.5 ± 30.3	25.6 ± 23.3 **
RFD (N/sec)	4712 ± 1849.2	38 ± 64.2	5434 ± 1699	22.2 ± 49.2
Anaerobic Measures (% mass resistance)				
Peak Anaerobic Power				
7.5 (watts)	769.4 ± 103.7	9.7 ± 6.4 **	769.6 ± 108.5	9.0 ± 6.7 **
8.5 (watts)	892.3 ± 121.3	9.7 ± 6.7 **	879.9 ± 144.3	8.1 ± 6.8 **
9.5 (watts)	965.6 ± 148.7	3.5 ± 6.2 *	984.1 ± 158.5	5.2 ± 5.2 **
Mean Anaerobic Power				
7.5 (watts)	609.4 ± 63.2	9.1 ± 5.7 **	605.2 ± 58	11.7 ± 7.4 **
8.5 (watts)	666.5 ± 68.5	11.8 ± 8.5 **	656.3 ± 62.5	8.2 ± 9.4 **
9.5 (watts)	690.1 ± 75.8	6.9 ± 6.1 **	697.6 ± 55.1	9.1 ± 6.1 **
Aerobic Measures				
VO_{2max} (ml/kg/min)	36.9 ± 4.1	5.8 ± 7.4 **	38.8 ± 4.0	8.8 ± 7.5 **
V_{max} (mph)	6.7 ± 0.8	9.1 ± 6.6 **	7.3 ± 1.0	17 ± 6.9 **|&
LT	5.4 ± 0.6	18.2 ± 14.2 **	5.2 ± 1.1	12.5 ± 19.5 **

Table 9. Final Values and % change. All values are presented as means ± standard deviations. * denotes significant change relative to preliminary values ($P < 0.05$). ** denotes significant change relative to preliminary measures ($P < 0.01$). & denotes significantly greater change than RT ($P < 0.05$).

Figures

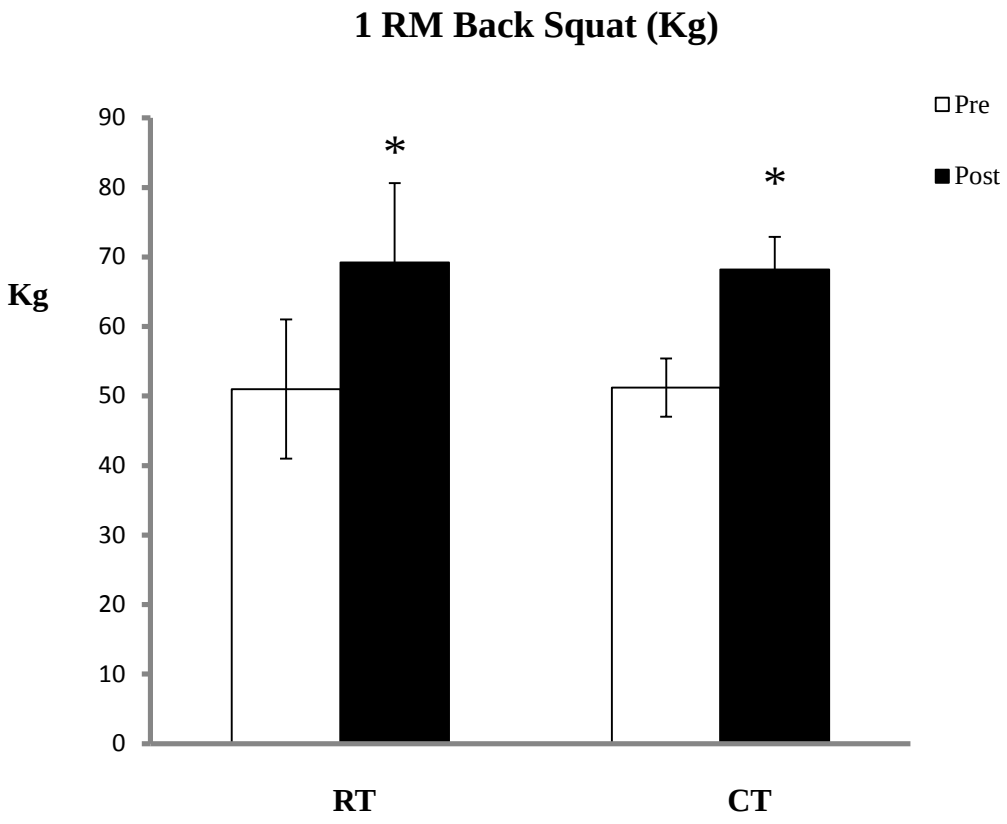

Figure 1. 1 RM back squat pre vs. post training. Values are reported as means ± SD. * Significantly increased from pre values ($P < 0.01$). No significant differences detected between training protocols.

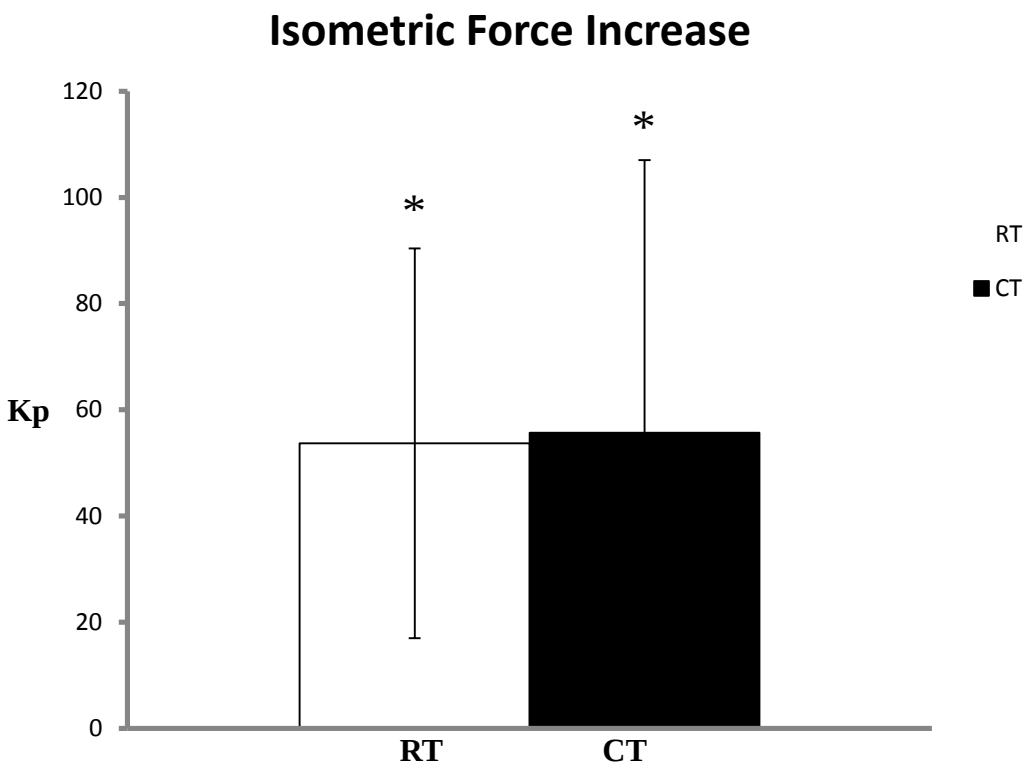

Figure 2. Maximal isometric force increases from pre values. Values are reported as means ± SD. * Significantly increased from pre values ($P < 0.01$). No additional significant differences detected post training.

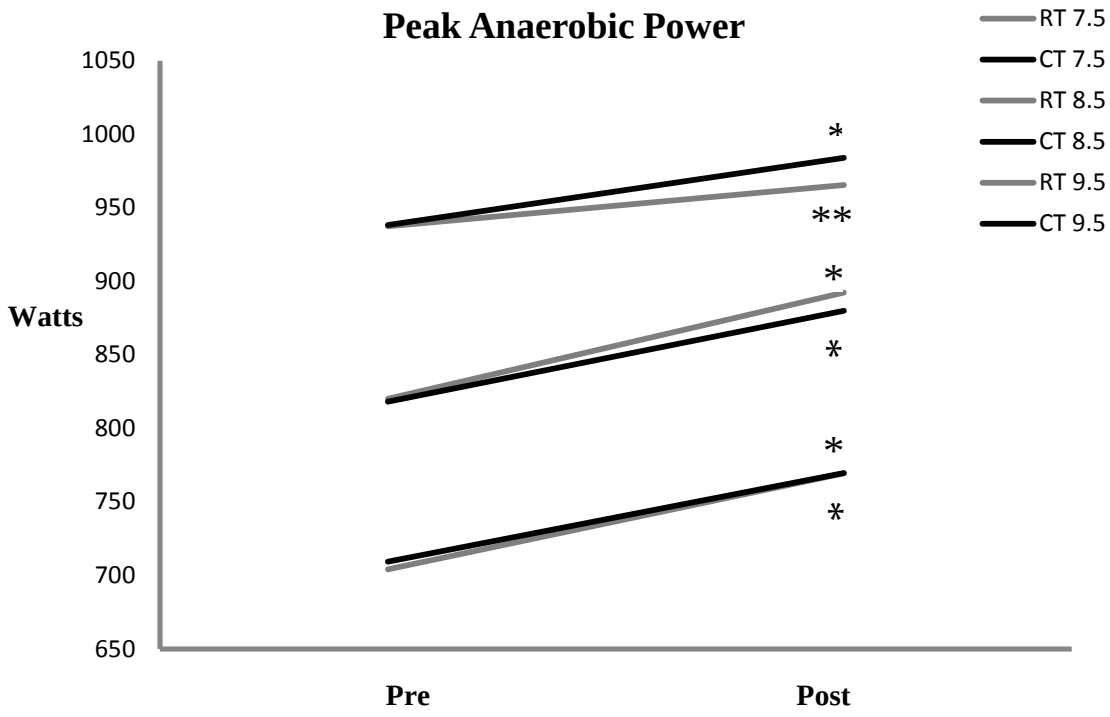

Figure 3. Peak anaerobic power pre and post training at three different resistances (7.5, 8.5, and 9.5 % mass). Values are reported as means. ** Significantly increased values vs. pre ($P < 0.05$). * Significantly increased vs. pre values ($P < 0.01$). No significant differences were detected between groups.

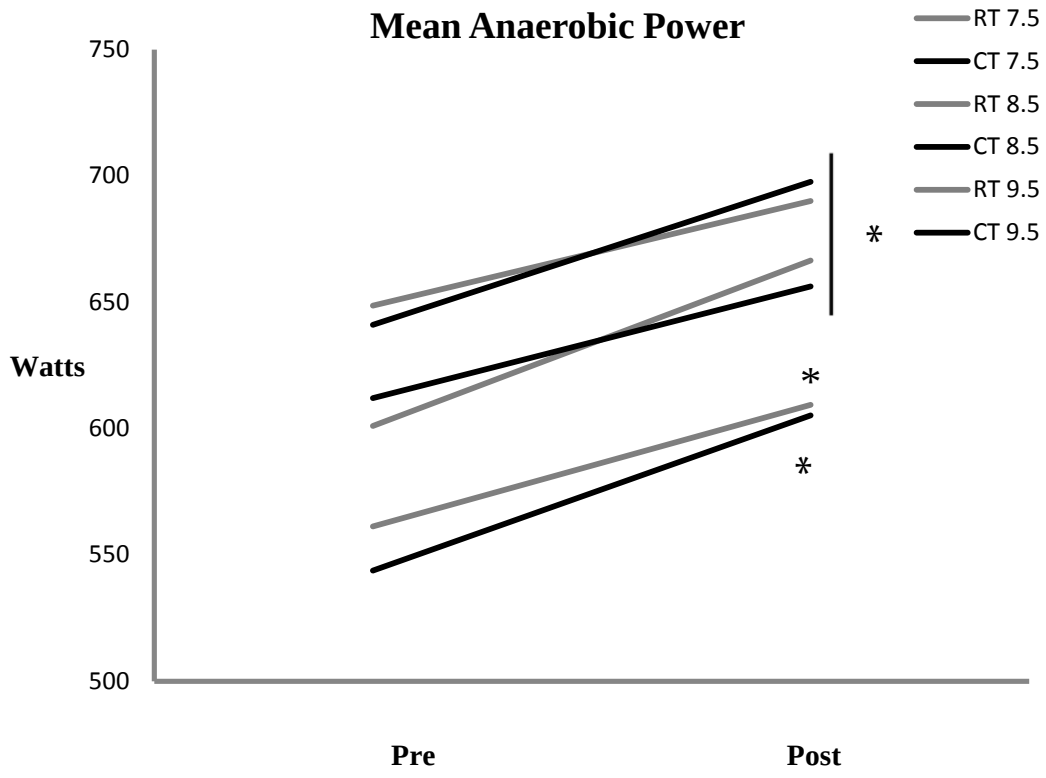

Figure 4. Mean anaerobic power pre and post training at three different resistances (7.5, 8.5, and 9.5 % mass). Values are reported as means. * Significantly increased vs. pre values ($P < 0.01$) No significant differences were detected between groups.

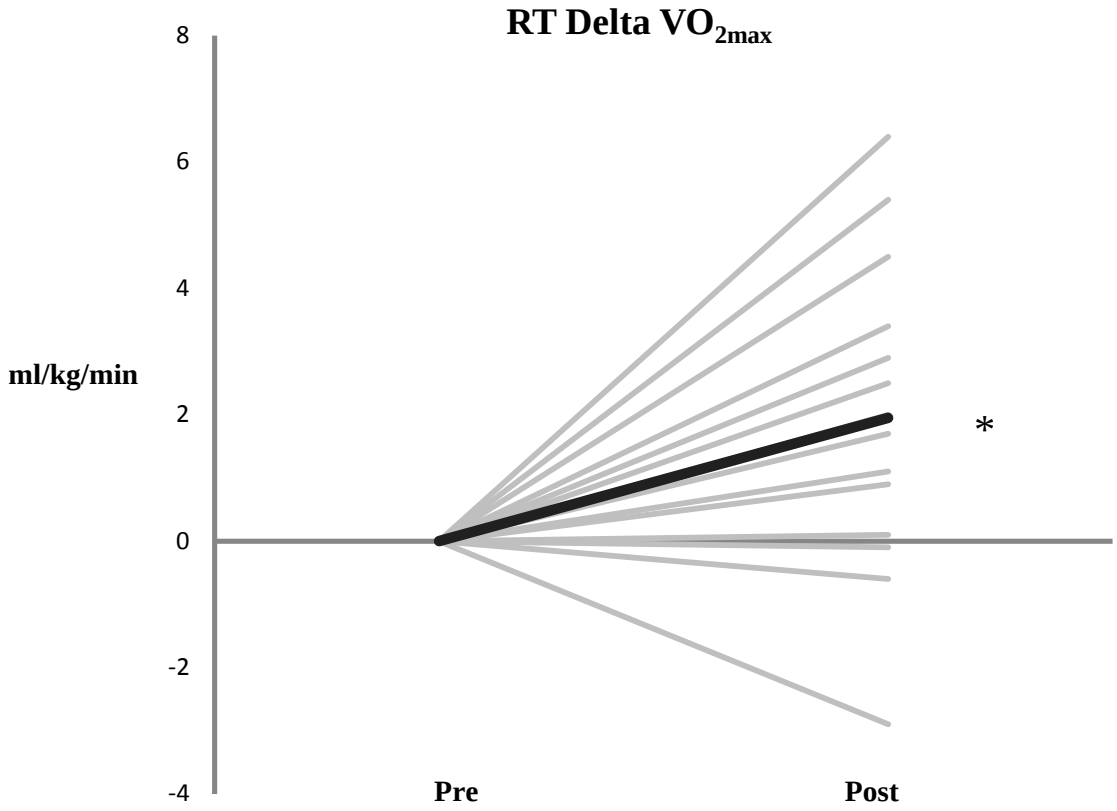

Figure 5. Change in VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) in RT following training. Values are reported as post minus pre (ml/kg/min). * Significant change from pre values ($P < 0.01$).

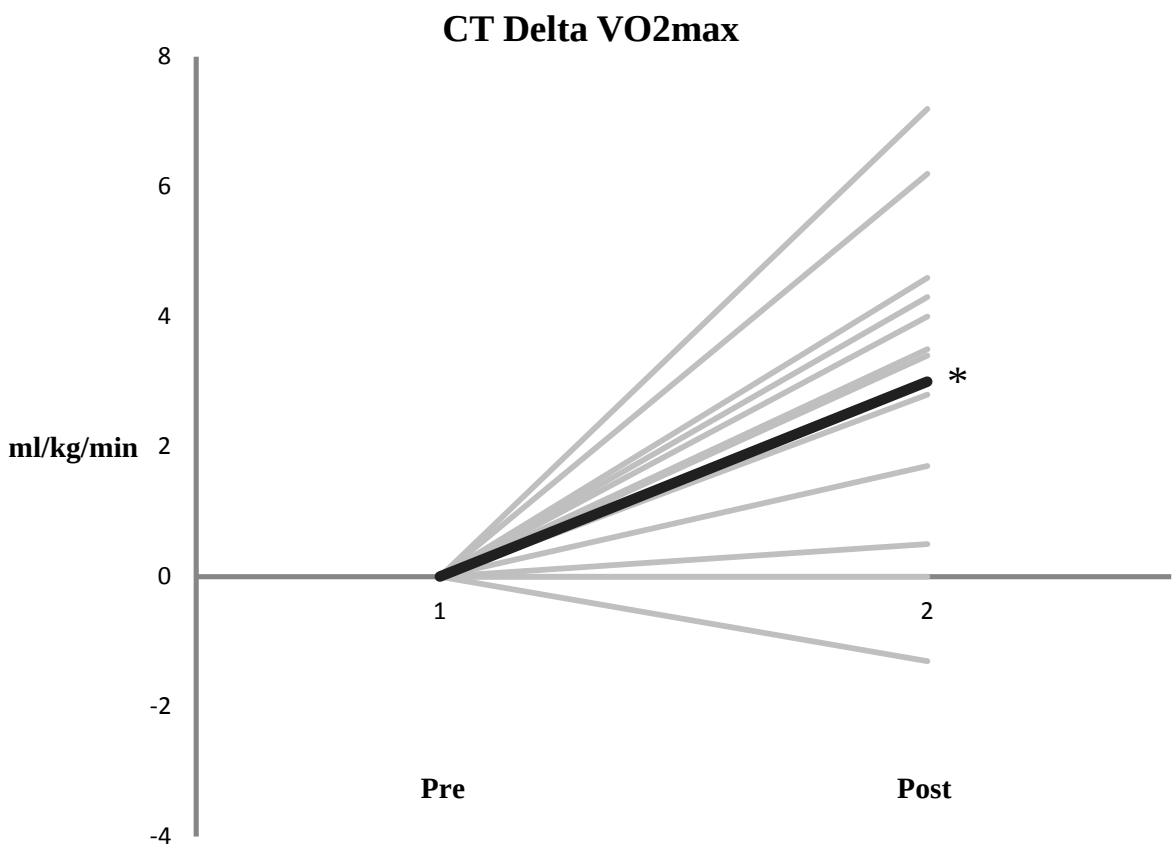

Figure 6. Change in VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) in CT following training. Values are reported post minus pre (ml/kg/min). * Significant change from pre values ($P < 0.01$).

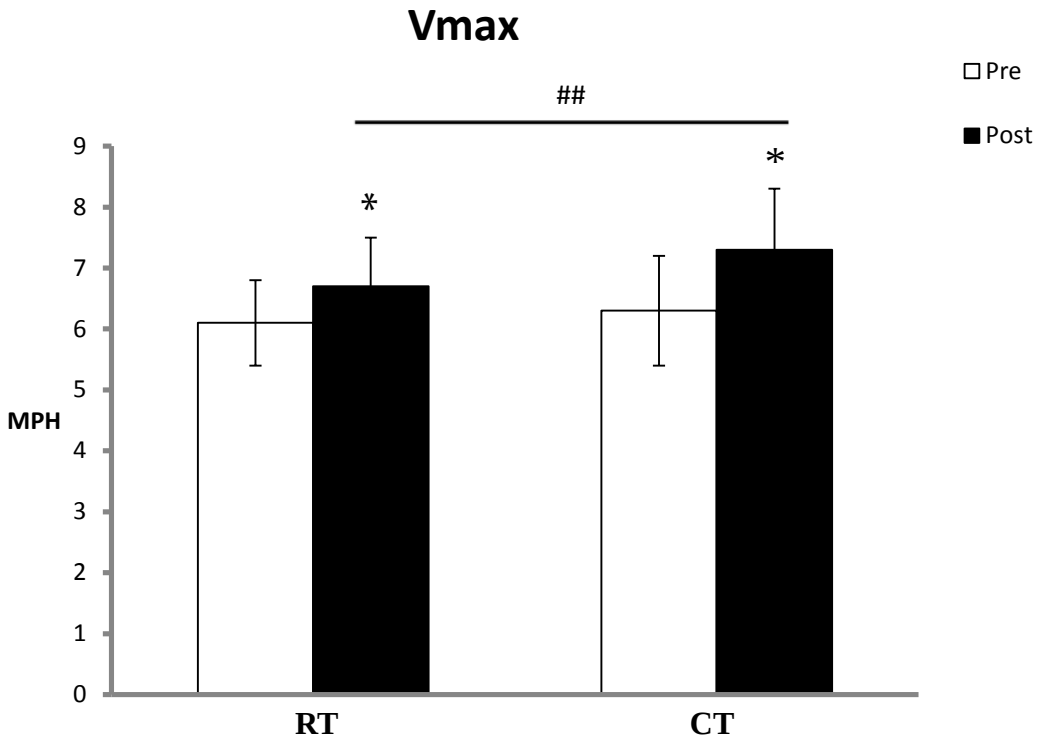

Figure 7. Predicted V_{max} pre and post training. Values are reported as means ± SD.
* Significantly increased from pre values ($P < 0.01$). ## Significantly different post values between groups ($P < 0.01$).

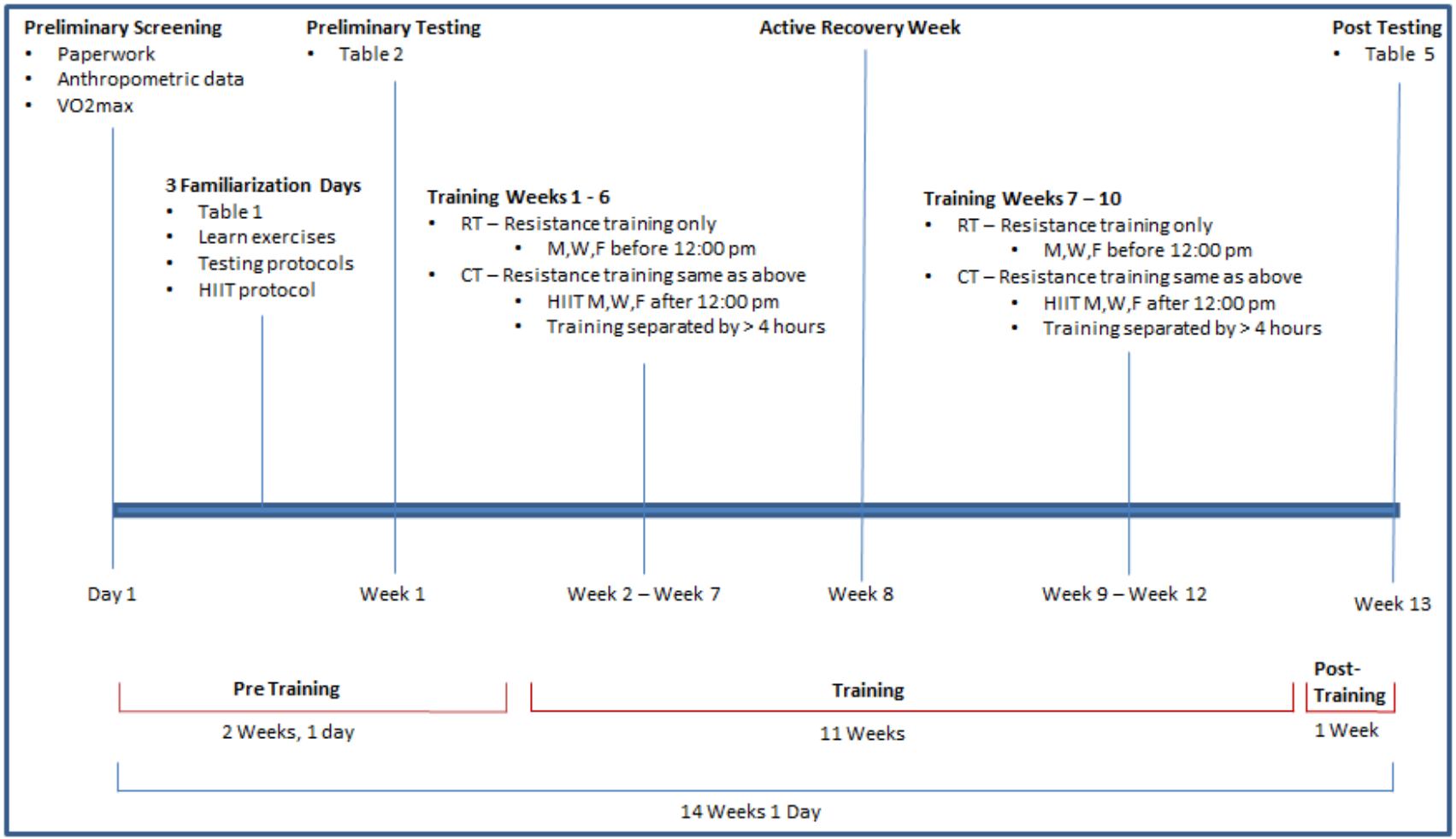

Figure. 8 - Study design

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Appendices

Appendix A

RECENT TRAINING HISTORY QUESTIONNAIRE

Subject code number:_____ Date:_____

Please answer these questions regarding your recent training level. Interval training is defined as alternating periods of high intensity or maximal effort alternated with periods of low intensity of complete recovery.

1. On average, how many days per week do you perform resistance exercise? How many minutes on average for each session?
_____

2. On average, how many days per week do you perform endurance exercise? How many minutes on average for each session?
_____

3. Have you made any changes to your exercise habits in the last 3 months?
_____

4. Did you do any interval training during this time? If yes, please describe.
_____
_____
_____
_____

Participants Signature_____ Date _____

Appendix B

PAR Q Medical Questionnaire*

Please read each question carefully and answer honestly. If you do not understand the question, please ask the investigator for clarification. Check the appropriate answer.

_____ _____ 1. Are you under 19 year old?

_____ _____ 2. Do you presently smoke or have been a regular smoker?

_____ _____ 3. Has your doctor ever said you have heart trouble?

_____ _____ 4. Do you have a family history of early cardiovascular death before the age of 50?

_____ _____ 5. Have you ever had a heart murmur, rheumatic fever or respiratory problems?

_____ _____ 6. Have you ever been told that you have a fast resting heart rate?

_____ _____ 7. Have you ever been told by your doctor or nurse that your blood pressure is too high?

_____ _____ 8. Have you ever been told that your cholesterol is too high?

_____ _____ 9. Have you been told that you have a kidney disorder?

_____ _____ 10. Have you been told that you have diabetes or that your blood sugar is too high?

_____ _____ 11. Have you been told that your electrocardiogram (EKG), 12 lead EKG or stress test is not normal?

_____ _____ 12. Do you have any rashes or reactions that result from hot or cold exposures (hot or cold uticaria)?

_____ _____ 13. Have you been hospitalized in the past year?

_____ _____ 14. Are you taking prescription medicine?
If so, what? _____

** Note that taking certain medications may cause you to be excluded from participation in this study including those that cause increases in heart rate, or other drugs that may increase the risk of participation.

_____ _____ 15. Do you have any orthopedic issues that would prevent participation in this study?

_____ _____ 16. Do you have any reason to believe that your participation in this investigative effort may put your health or well being at risk? If so, please state reason. _____

_____

Signature of subject _____ Date _____

*Adapted from British Columbia Department of Health and Michigan Heart Association.

Appendix C

Auburn University

Department of Kinesiology	**Telephone: (334) 844-4483**
2050 Memorial Coliseum	**Fax: (334) 844-1467**
Thermal Lab (Room 2118)	**Thermal Lab: (334) 844-1479**

Informed Consent for a Research Study Entitled

"Effect of simultaneous high intensity sprint interval running and ground based barbell strength training versus strength training on measures of strength and power"

Project Overview: You are invited to participate in a research study that will examine the effect of two different training programs on aerobic, strength, and power outputs in recreationally active females. We are recruiting participants to complete a 13 week study. Participants will be assigned to either a strength training only, or concurrent strength training and Sprint high intensity interval training (HIIT).

Purpose: The purpose of this investigation is to examine aerobic, strength, and power changes that occur following each training intervention.

Participation Requirements: To be eligible, you must be:

1. Female participant between 19 and 35 years of age
2. Maximal oxygen consumption levels between 30 and 50 ml/kg/min as determined by preliminary testing.
3. Low risk for medical complications (as determined by physical activity readiness questionnaire (PARQ)).
4. Currently engaging in no more than three days per week of moderate strength and/or endurance training (as determined by recent activity questionnaire).
5. Currently not taking any medications that will increase the risk of participation, or interfere with testing variables. Note that taking certain medications may cause you to be excluded from participation in this study including those that cause increases in heart rate, or other drugs that may increase the risk of participation.

You must meet all of the requirements to be eligible for participation in this study.

Time commitment for participation in this study will be 13 weeks, lasting a total of between 36 and 46 hours dependent on training group assignment.

Day 1: On the first visit to the lab, you will complete the PARQ Questionnaire, complete the current activity questionnaire, and read and sign the University-approved informed consent form. Either Richard Laird or David D. Pascoe will be present for all informed consent briefings. In ineligible for participation for any reason (participation requirements or PAR-Q) all forms will be returned to the subject and no record kept by the researchers.

Descriptive data will be obtained (age, height, weight, DEXA(body composition)). Hydration level will be assessed using a urine refractometer. If adequately hydrated, you will then be familiarized with the Woodway treadmill and complete a VO2 max test. This is an incremental treadmill running test designed to determine your maximal oxygen uptake. We will monitor you closely and ensure that you complete a thorough cool-down by walking for several minutes at a comfortable pace.

The total time for the exercise testing will be approximately twenty minutes (including a warm-up and cool down) while descriptive data will not take more than 40 minutes, making total time commitment for the entire visit approximately one hour.

Day 2 – Day 10: Over this 9 day period you will return to the lab on three separate occasions to be familiarized with testing procedures including vertical jump, isometric squat, back squat, standing press, monarch cycle ergometer, and treadmill running protocol. Additional familiarization opportunities will be provided as needed. On the last familiarization day a practice run will be completed on the one repetition max test for both the back squat and standing press. Total time commitment for each visit is 45 minutes.

You will be asked to refrain from other physical activity during the course of the study

Testing - Week 1 – Testing will take place on Monday, Tuesday, Thursday, and Friday on the timeline provided below. Participants will be given a dietary log on Monday to record throughout the week. Logs will be collected on Friday by the researcher. Detailed descriptions of each test can be found at the end of this document.

Monday: Vertical Jump, Isometric Squat, and Wingate testing
Tuesday: 1 RM testing for back squat and standing press (day 2)
Wednesday: Off
Thursday: Lactate threshold testing
Friday: 1 RM testing for back squat and standing press (day 3)

Time commitment for the week = 3.5 hours

After testing is completed you will be assigned to one of two experimental groups: strength training only, or strength training plus sprint HIIR. Both exercise protocols are described below.

Strength Training Protocol -

- General warm-up will be completed before each session
- Two alternating training days will be used
- Sets, reps, and intensity will be altered after every two training sessions.
- Each program will be individualized based on you testing variables

- Training will occur at the same time each day (before 12 noon) and will not exceed 45 minutes
- All training will be overseen by a certified strength and conditioning coach
- Time commitment = 2.5 hours/week

Sprint HIIR Protocol

- 3 minute warm-up will be completed consisting of a jog at 50% VO2 max
- Participants will be allowed to stretch as needed prior to intervals
- Total exercise time will be 4 minutes broken down into 8 intervals of 20 seconds running, 10 seconds passive recovery
- Speed of the run will be individualized and based on testing outcomes
- Training will occur at the same time each day (after 12 noon) and will not exceed 20 minutes
- Time commitment = 1 hour / week

Training Weeks 2 – 12

- Training will take place three days per week: Monday, Wednesday, and Friday
- Participants assigned to the concurrent training group will strength train in the morning and run intervals in the afternoon separated by a minimum of 3 hours
- Participants will be asked to consume at least 1 pint of water before reporting for the workout
- Week 7 (Spring Break) will be used as an unload week. Participants will be given instructions as to what light activity to complete over break
- Time commitment 2.5 – 3.5 hours per week depending on group assignment.

Post Testing – Week 13 – will be a retest of all variables examined in week 1. Dietary logs will be returned to participants on Friday of week 12 and you will be asked to replicate dietary intake from the first week. Testing schedule is found below.

Monday: Vertical Jump, Isometric Squat, and Wingate testing
Tuesday: 1 RM testing for back squat and standing press
Wednesday: Collect anthropometric data
Thursday: VO2 Max
Friday: Lactate threshold testing

Time commitment for the week = 4 hours

Total time commitment = 13 weeks – between 36 and 46 hours depending on groups assignment

Test Descriptions:

VO2 Max – perform incremental treadmill test with O2 consumption monitored via a True Max Metabolic Testing System.

Vertical Jump - 3 attempts to attain Maximum vertical jump will be given. Each attempt will be separated by at least 1 minute rest.

Isometric Squat - In a squat rack knee angle will be set to 150° with participant standing on a force platform. At the instruction of the researcher the participant will exert as much force as possible upwards on the bar

1 RM Back Squat – Participants will warm-up; followed by a set of 10 squats with an empty bar. An additional set of 5 and 2 sets of 3 will be used to warm up. 1 RM will be obtained in no more than 4 attempts

1 RM Standing Press - Participants will warm-up; followed by a set of 5 standing presses with an empty bar. An additional set of 5 and 2 sets of 3 will be used to warm up. 1 RM will be obtained in no more than 4 attempts

Wingate – Participants will be required to warm-up and cool down for 3-5 minute before and after the test at a light resistance. The 30 second test will be conducted on a monarch cycle ergometer.

Lactate Threshold – After warming up participants will complete 4 separate 6 minute intervals corresponding to 55, 65, 75, and 85% of VO2 max. A small amount of blood will be collected at the end of each trial for blood lactate content analysis via finger prick by a trained phlebotomist.

Potential Risks:

1. While performing any exercise there is a chance of muscle strains, sprains, pulls, and even death. The American College of Sports Medicine estimates the risk of death to be 0.5 per 10,000 individuals.

2. Due to the high intensity nature of some of the exercise you may feel nauseous and/or light-headed after completing the intervals.

3. With any blood collection procedure there is a risk of infection, bleeding, bruising, irritation at injection site, and/or fainting.

"Note" It is important for you to realize that you are responsible for any costs incurred in the event of an injury.

Precautions:

1. Although the training for this trial is of higher intensity, it is of short duration and at a comfortable environmental temperature and humidity level. Heart rate will be recorded throughout the trial. We have additionally employed the use of a modified PARQ to assist in eliminating participants that have potential medical or orthopedic identified risks. During the trials you will always be accompanied by researchers who maintain current CPR Certifications.

2. After each exercise bout you will be monitored and be given a chance to cool-down.

3. Both investigators participating in data collection (Richard Laird and David Elmer) are certified in phlebotomy. Only new, sterile blood-gathering equipment and aseptic techniques will be utilized throughout all data collection and analysis processes.

4. All training program design and oversight will be handled by Rich Laird who is a Certified Strength and Conditioning Coach with more than 4 years experience working in the field. Proper lifting technique, volume and intensity manipulation, and spotting will be employed to decrease the risk of injury.

5. Should an emergency arise, we will call 911 and follow our emergency action plan. You are responsible for any cost associated with medical treatment.

Benefits: You will receive 12 weeks of organized and supervised training, along with performance assessments including body composition, Wingate assessment, VO2 max, 1 RM squat, 1 RM bench, Vertical Jump, Isometric squat force, and lactate threshold.

Your participation is completely voluntary. If you change your mind about participating, you can withdraw at any time during the study. If you choose to withdraw, you can request to have your data withdrawn. Your decision about whether or not to participate or to stop participating will not jeopardize your future relations with Auburn University, the Department of Kinesiology, or the Thermal Lab.

Your privacy will be protected. Any information obtained in connection with this study will remain anonymous.

If you have any questions, we invite you to ask us now. If you have questions later, you can contact Richard Laird (rhl0003@auburn.edu), David Pascoe (pascodd@auburn.edu), or call 334-844-1479. You will be provided with a copy of this document for your records. For more information regarding your rights as a research participant, you may contact the Auburn University Office of Human Subjects Research or the Institutional Review Board phone number (334) 844-5966 or email at hsubjec@auburn.edu or IRBChair@auburn.edu.

HAVING READ THE INFORMTION PROVIDED, YOU MUST DECIDE WHETHER OR NOT YOU WISH TO PARTICIPATE IN THIS RESEARCH STUDY. YOUR SIGNATURE INDICATED YOUR WILLINGNESS TO PARTICIPATE.

Participant's signature	Printed Name	Date
_____	_____	_____
Investigator obtaining consent	Printed Name	Date
_____	_____	_____
Co-Investigator	Printed Name	Date
_____	_____	_____