Recovery from Exercise: The Influences of Supplementation and Rest Interval

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A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Kinesiology

by
Ryan William-Ignatius Miskowiec
B.S., Louisiana State University, 2007
M.S., Louisiana State University, 2012
May 2015
ACKNOWLEDGEMENTS

The process of completing this work has been difficult and time consuming. I would like to give thanks to those who have supported me in the effort and made this work possible.

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Dr. Johannsen has been generous enough to lend me both his ear and lab space. He has helped me become a better researcher by showing me clinical best practices and giving me his time.

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ABSTRACT

Recovery from exercise is immensely important and under studied. The experiments conducted throughout this research have focused on testing the effects of rest interval during resistance exercise and the efficacy of acetic acid feeding on recovery rate. To elucidate the relationship between rest interval and recovery energy expenditure two experiments were undertaken. These experiments were designed to alter only rest interval while maintaining work intensity and volume. No difference in the total energy expenditure during the short phase of recovery (first hour) was found, but energy expenditure during the prolonged phase (days post exercise) was minorly increased by lowering rest interval. These results combined with the metabolic and affective data collected have raised questions as to the underlying mechanisms which warrant further study. The third and fourth experiment focused on acetic acid consumption and recovery from exhaustive exercise. Previous studies in animal models have shown significantly improved recovery rates. This research was focused on testing the efficacy of acetic acid feeding in humans to improve performance. Participants therefore exercised, were given a time to rest, and a recovery beverage was consumed. After recovery participant’s ability to perform was assessed, and various metabolic measurements were assessed. The findings of these projects are promising, as acetic acid appears to have positive effects on recovery. The potential benefits to millions of athletes in a variety of sports are immense. The work completed throughout this Dissertation has successfully added to the field of recovery research, and provided more questions to be answered in the future.
CHAPTER I: INTRODUCTION

Recovery from exercise is an important area of study which is currently underserved. Understanding recovery is vital for optimal exercise programing, preparation, and performance. Without sufficient recovery, performance may suffer. Knowledge of the recovery process allows exercisers to adequately prepare and anticipate the physiological effects of exercise. The research conducted in this Dissertation has focused on the effects of rest interval on recovery energy expenditure and the ergogenic effects of vinegar supplementation on recovery.

When designing exercise programs using resistance exercise there are many variables to consider. The majority of past research into the bioenergetics effects of resistance exercise has focused on load intensity and volume of work. The variables are vital to exercise programing, but comprise only two of the many variables which comprise an exercise program. Rest interval is an important variable within resistance exercise programs which is understudied, and therefore its influence on recovery energy expenditure is unclear.

The recent surge in the popularity of “HIIT” training (high intensity interval training) using resistance exercise modalities prompted interest in this line of research. Very little is known about the physiological implications of this type of exercise from the perspective of recovery. A common goal for many recreational exercisers is weight loss or weight maintenance (1). Many contemporary exercise programs promise enhanced results through reduction of rest interval, but the literature is lacking in this area. The first investigation into this topic was therefore focused on gaining a clearer understanding of how this type of training impacted energy expenditure.

The first experiment performed in this Dissertation aimed specifically at establishing a relationship between rest interval and energy expenditure. While recovery was the primary focus
of the study the total energy expenditure (during work and recovery) was used as the technology available would not allow for a strict investigation of recovery energy expenditure. The majority of caloric expenditure secondary to resistance exercise occurs during the recovery, therefore these methods were concordant with the paradigm of the study (2).

Few previous studies have been conducted to elucidate the relationship between rest interval and recovery energy expenditure, but this research has been limited by flawed methodologies which have made interpretation difficult. The works of Ratamess et al. (3) and Farinatti (4) were convoluted by highly unmatched workloads (approximately 50% different in some cases). Other researchers have tested rest interval without making protocols highly different, and therefore the effect of rest interval on energy expenditure have not been adequately investigated (5, 6). To better understand rest interval an initial experiment was therefore developed to incorporate matched volume and intensity while exacerbating the difference in rest interval between treatments to such an extent as to demonstrate any potential effect on energy expenditure.

To follow up and further investigate rest interval’s effects on energy expenditure a second experiment was conducted. This investigation was aimed at elucidating the effects of rest interval the prolonged phase of recovery (days post exercise). Previous researchers linked lifting to increases in resting metabolic rate for several days post exercise (7, 8, 9). The mechanisms underlying this increase in metabolic rate are not fully understood, but damage to muscle tissue is a prevalent theory (7). The initial experiment conducted in this line of research examined only the short phase of recovery, and therefore the long term effects were not observed. Several participants of this study reported high levels of soreness following the limited rest protocol, but not the other. These anecdotal reports prompted a second study to assess rest interval’s effect on the prolonged phase of recovery. The aims of this study were to match volume and intensity while drastically
altering rest intervals and to assess the resultant resting metabolic rate days post exercise. Soreness was additionally assessed, and more lifts were added to more closely mimic typical recreational exercise programs.

The second element of this dissertation was testing the efficacy of vinegar as an ergogenic aid to recovery. Vinegar is comprised of water and acetic acid and past research in animal models indicates that acetic acid may significantly improve glycogen repletion rate (10,11,12). Muscle glycogen availability is highly correlated with high intensity performance (13). High intensity exercise depletes muscle glycogen rapidly, as it is preferentially utilized when energy demand is great (14). Full replenishment of muscle glycogen is a lengthy process which can take over 12 hours depending upon the intensity and duration of exercise (15). Many sports require athletes to compete in multiple events in a single day which may result in diminishing performance due to glycogen depletion. The consumption of acetic acid post exercise may therefore offer a means of increasing performance by increasing glycogen repletion rate.

Acetic acid has been shown to increase the rate of glycogen repletion in mice and horses, but this result has not been replicated in humans. Many athletes such as wrestlers and runners compete in multiple same day performances would benefit greatly from increased recovery of muscle glycogen. Therefore an initial investigation into the potential ergogenic effects of acetic acid on recovery rate was conducted. This study was designed to gauge high intensity performance capacity before and subsequent to fatiguing exercise and recovery. During recovery participants were provided with a carbohydrate based beverage which either contained or did not contain vinegar. The promising results obtained through this initial investigation prompted further more robust study.
To better understand the results obtained through the initial investigation into the ergogenic effects of acetic acid a second study was designed. The first study was conducted using untrained participants; a population which likely does not benefit from the research. The second study was therefore included participants who were better trained. To better understand the action of acetic acid during recovery more treatments were included and measurements of blood glucose and substrate utilization were collected. This second study was also designed with a different performance test to more thoroughly assess high intensity performance in an effort to gauge muscle glycogen content more effectively.

Ultimately, the research completed throughout this dissertation has focused on various aspects of recovery from exercise. The goal was been to contribute to the understanding of the interplay between exercise and recovery. The research presented is relevant to the millions of people throughout the world who seek to understand how to bolster their exercise programs, and derive maximal benefit with minimal investment. These studies are novel as rest interval’s effect on energy expenditure has not been effectively studied previously, and acetic acid’s effect on recovery in humans was previously untested. The following chapters detail each experiment individually and a general conclusion is included thereafter.
CHAPTER II: THE BIOENERGETIC EFFECTS OF REST INTERVAL DURING RESISTANCE EXERCISE: SHORT PHASE OF RECOVERY

PREFACE

This experiment was undertaken to gain an understanding of how rest interval affected the energy expenditure resulting from resistance exercise. Limited research has been conducted examining the significance of rest interval on energy expenditure during recovery. Studies which have attempted to explore this issue have failed to adequately isolate rest interval as the sole independent variable, and thus past results are difficult to interpret.

This experiment was designed to elucidate the effect of rest interval on energy expenditure maintaining equivalent volumes and intensities while manipulating only rest interval. Exercise performed with minimal rest was hypothesized to increased energy to a greater extent than equal work performed with greater rest.

METHODS

POPULATION

Healthy recreationally trained male participants were recruited from the Louisiana State University student body. Nineteen participants were recruited, 2 did not meet the inclusion criteria, therefore 17 participants were included. Participant characteristics are displayed in table 1.

The experiment’s procedures were explained verbally and in written form prior to participant completion of an informed consent approved by the Louisiana State University Institute Review Board. The PAR-Q was completed by all participants and no injuries or illnesses which contraindicated participation were reported. Additionally, participants were required to perform 3 sets of 10 repetitions on a bilateral leg press (Bodymasters™ CX123) using 180% of their body weight to be considered for inclusion.
Table 1: Participant Characteristics

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STUDY OVERVIEW

Participants completed the PAR-Q, discussed pertinent medical concerns, and gave their informed consent prior to participation. To familiarize participants with the equipment, and determine eligibility for inclusion participants completed three sets of ten repetitions on a bilateral leg press using 180% of their body weight.

Once screened participants completed two trials using a balanced crossover design with a minimum of one week wash out period. Each trial consisted of a brief warm up followed by 60 repetitions of bilateral leg press exercise. The leg press was performed using a load equal to 180% of the participant’s body weight with two distinct protocols. The work was performed either with low or high rest during the protocol. Participants’ metabolic activity was monitored continuously using indirect calorimetry before, during, and after each exercise bout. Blood glucose levels were assessed before and after the warm up, and again following completion of the last repetition during each protocol. Participants fasted overnight (a minimum of 10 hours pretrial), but were provided a small control meal which was administered two hours prior to commencement of each trial.

EXERCISE PROTOCOLS

Prior to each exercise protocol participants performed a warm up consisting of 20 repetitions using 100% of participant’s body weight on the bilateral leg press. The exercise protocol started three minutes after the beginning of the warm up.
The exercise protocols each consisted of 60 repetitions of bilateral leg press using 180% of each participant’s body weight as the prescribed load. The high rest protocol (HiR) consisted of 6 sets of 10 repetitions with approximately 6 minutes rest interval between sets. The low rest protocol (LoR) consisted of 60 repetitions of the same load completed as rapidly as possible. During the LoR protocol, participants were allowed to rest ad libitum, but verbally motivated to complete the workload quickly. All trials were conducted by the same researchers for consistency throughout the experiment.

**METABOLIC ASSESSMENT**

Oxygen consumption was assessed using indirect calorimetry (Parvo Medics TrueOne® 2400) in a quiet temperature controlled room. Participants rested for 15 minutes on the leg press prior to being fitted with the indirect calorimeter. Baseline metabolic activity was assessed for 10 minutes prior to the warm up. Without removing the indirect calorimetry equipment or moving from the leg press seat participants completed the warm up, exercise protocol, and recovery. Comparisons of the metabolic demands between protocols were made on a 1 hour period of breath gas assessment beginning at the start for the warm up.

**BLOOD GLUCOSE**

Blood glucose was assessed using a blood glucose monitor (ACCU-CHEK™ Compact Plus). Participants were prepared by cleaning the finger stick site with alcohol before each measurement. Then a lancet was used to prick the finger and a drop of blood was then applied to the test strip. A cotton ball was then pressed to the site until bleeding ceased.

**BODY COMPOSITION**

Participants’ body fat percentages were assessed using the men’s three site equation (chest, abdominal, and thigh sites) detailed in the ACSM’s Guidelines for Exercise Testing and
Prescription 9th edition. All skin fold assessments were conducted by the same researcher during participants’ initial screening using a hand held caliper (FEI™ Baseline Skinfold Caliper). Additionally, height and weight were measured during the screening visit.

**PRE-WORKOUT MEAL**

Participants fasted for 10 hours prior to each trial. To avoid exhaustion while maintaining a controlled metabolic state participants were provided with a 200 kilocalorie meal replacement bar which contained approximately 40% carbohydrate, 30% fat, and 30% protein. This meal was consumed 2 hours prior to each trial.

**STATISTICAL ANALYSIS**

Statistics analysis was completed using JMP Pro (version 11). Results are reported as mean plus or minus standard deviation. Differences in baseline energy expenditure, total energy expenditure, and respiratory quotient were assessed using paired t-tests. A 2X3 repeated measures ANOVA (Protocol X Time) was utilized to detect statistical differences between blood glucose measurements. Pierson’s correlations were utilized to assess relationships between time to complete the LoR protocol and various participant characteristics such as body fat percentage, weight, and BMI. Significance was determined at the p = 0.05 threshold.

**RESULTS**

Baseline energy expenditure of the HiR and LoR protocols were not significantly different [3.93±0.68 ml O2·kg⁻¹·min⁻¹ and 3.91±0.35 ml O2·kg⁻¹·min⁻¹ respectively (p = 0.85)] The average total energy expenditure of the HiR protocol was 6.44±0.51 ml O2·kg⁻¹·min⁻¹ whereas the LoR protocol resulted in 6.14±0.84 ml O2·kg⁻¹·min⁻¹ (p = 0.13). The minute by minute averages of oxygen consumption during each protocol is displayed in figure 1.
The RQ between the LoR and HiR protocols was significantly different [0.89±0.03 versus 0.92±0.03 respectively (p=0.01)]. The average RQ values during each protocol are shown in figure 2.

Participants completed the LoR protocol in 230.41±131.42 seconds. A significant correlation between participant’s body fat percentage and time to complete the LoR protocol (p = 0.02) was found. Time to complete the LoR protocol was not significantly correlated to weight or BMI (p = 0.46 and p = 0.24 respectively).
There were no significant interactions between time and protocol with respect to blood glucose levels \((p = 0.64)\). The blood glucose levels during each protocol were not significantly different with respect to protocol \((p = 0.15)\), but time was significant \((p = 0.04)\). On average blood glucose levels were significantly higher post exercise when compared to post warmup \((p = 0.02)\), but not pre exercise \((p = 0.07)\).

**DISCUSSION**

This experiment was undertaken to test the effects of rest interval on energy expenditure resulting from resistance exercise. The HiR protocol resulted in greater total energy expenditure than the LoR protocol, though this difference was not statistically or meaningfully different. These

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Figure 2: The mean respiratory exchange ratio (RQ) during each of the two trials. The first ten minutes depict the period of rest prior to warm up, and the remainder of the time follows the warmup, exercise, and recovery.
findings correspond closely to those of Haltom et al. (6) and Farinatti et al. (4) which both found no differences between high and low rest interval protocol in terms of total energy expenditure. The two previously mentioned works did result in significant differences, but their protocols did not differ greatly, which could explain the lack of findings. The present study differs from previous studies as the difference between protocols was exacerbated to a far greater extent. The null findings of the current study therefore support the results of previous research.

Indirect calorimetry can assess energy expenditure, but its application during resistance exercise is problematic. During indirect calorimetry the volumes of air moved is compared with the oxygen consumed and the carbon dioxide produced. This system therefore functions to monitor aerobic metabolic. While aerobic metabolism is occurring during resistance exercises anaerobic pathways (the phophagen system and glycolysis primarily) supply the majority of the energy during resistance exercise (16). These metabolic systems do not consume oxygen nor do they produce carbon dioxide as they generate energy. Therefore indirect calorimetry alone is insufficient to measure energy expenditure during resistance exercise because the anaerobic metabolism will not be accounted for during exercise (2). This known limitation of indirect calorimetry can be attenuated by measuring energy expenditure during recovery from exercise as these systems consume energy to be replenished (17). Total energy expenditure was therefore selected as the most appropriate means of assessing the energy expenditure of the exercise protocols.

Controlling volume of work, intensity of load, and rest interval concurrently is exceedingly difficult as fatigue results in diminished performance. Past studies such as Ratamess et al. (3) have attempted to perform multiple sets of work using extremely low rest intervals which have resulted in fatigue and uneven total work performed (over 50% less work in some instances). The paradigm
of rigid numbers of repetitions and sets can only be maintained if adequate time is allotted between sets for recovery. To assess the effects of rest on the energy expenditure of resistance exercise strict numbers of repetitions and sets were therefore not utilized, but instead a total volume was prescribed and participants were allowed to complete it at their own pace (during the LoR protocol). This unconventional approach to resistance exercise did result in differing times to complete the work, but produced equal work between protocols. A higher priority was placed on ensuring that volume and intensity consistency then temporal consistency, so this limitation was acceptable.

Intensity was determined solely by participant’s body weight. This decision was made to accommodate a wide variety of participants, and increased the generalizability of the results. The load of 180% of body weight was selected because this load is achievable by the majority of people when using a selectorized bilateral leg press machine. Participants with lower percent body fat completed the LoR protocol more rapidly than did those with higher percent body fat. Body fat is often inversely related to training status, which may have played a significant part in participants’ ability to rapidly complete the LoR protocol (18). Participants’ bodyweight was not significantly correlated to time to complete the LoR protocol. Therefore, participants’ time to complete the LoR protocol was not simply a function of the increased loads being prescribed to heavier participants.

This experiment consisted of only a single exercise. If a whole body workout were completed at the pace of the LoR protocol it is likely that the effects of fatigue would have negatively impacted participants’ total work performed. This in turn would likely result in reduced energy expenditure.
Respiratory quotient averages were significantly lower during the LoR treatment than the HiR treatment. While a low RQ is typically associated with fat utilization the highly anaerobic nature of the protocols may have convoluted these measurements. The LoR protocol in particular entailed a large volume of work in a short period of time, which likely resulted in significant anaerobic metabolism. Reductions in blood pH may result from intense work such as that of the LoR protocol. To prevent systemic acidosis buffers such as bicarbonate are employed by the body. With sufficient volumes of work using anaerobic metabolism the bicarbonate buffer system can become partially depleted. Following exercise reductions in RQ have often been associated with replenishment of the bicarbonate buffering system as metabolically produced CO2 may be converted to bicarbonate instead of being exhaled. The observed reduction of CO2 in breath gasses may therefore not be a result of increased fat oxidation, but instead is likely a product of bicarbonate replenishment (16).

Increased blood glucose levels were observed following both protocols. While blood glucose levels are controlled by a number of hormones the observed changes are likely due to the catecholamine release such as epinephrine, which is often observed during resistance exercise (19). Epinephrine is one of several hormones responsible for a feed forward type breakdown of liver glycogen which ultimately results in increased blood glucose (20).

Regular exercise is essential to general health and wellbeing, but many Americans do not meet their minimal recommended exercise levels due to time constraints. Understanding the effects of rest interval on energy expenditure is therefore vital to prescribing exercise for millions of individuals who have limited time to devote to exercise. The current study has shown that the time to complete a resistance exercise bout does not significantly impact the energy expenditure of the exercise. Therefore, while not particularly calorically advantageous performing lifts with
little to no rest interval may serve as a means for individuals to meet their recommended resistance exercise quota using minimal time. With limited research examining the efficacy of this type of exercise regime more work must be done to explore the potential health benefit and risks.
CHAPTER III: THE EFFECT OF REST ON RESTING METABOLIC RATE DURING THE PROLONGED PHASE OF RECOVERY FROM RESISTANCE EXERCISE

PREFACE

During the initial study examining the effects of rest interval on energy expenditure during the short phase of recovery several participants reported a great degree of soreness following the low rest protocol. Soreness is not uncommon following resistance exercise, as lifting often results in muscle damage, but participants did not report soreness following the high rest protocol.

Exercise induced muscle soreness typically occurs several days post exercise, and may be associated with increased RMR (21, 7).

The study was therefore undertaken to determine if rest interval may have an effect on energy expenditure during the prolonged phase of recovery. The low rest protocol was hypothesized to result in increased resting metabolic rate and greater soreness than the high rest protocol.

METHODS

POPULATION

Twelve recreationally trained females participated in this study. Participants were considered trained if they had lifted continuously for a minimum of 6 months prior to the commencement of the study, and could perform all of the lifting techniques using proper form.

Participants completed the PAR-Q and were free of any injuries or illnesses which contraindicate physical activity. Participants were nongravid, reported regular menstrual cycles (approximately 28 day), and denied the use of contraceptives which significantly impact metabolic rate. Descriptive information regarding participant characteristics is given in table 2.
Table 2: Participant Characteristics

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<td>Body Fat (%)</td>
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EXPERIMENTAL OVERVIEW

Participants reported to the Clinical Exercise Testing Lab (Louisiana State University, Baton Rouge, LA) on four consecutive mornings twice, with 28 days in between. Each morning RMR was assessed and ratings of perceived soreness were obtained. A full body resistance exercise protocol was completed on the second day of each four day trial period. The exercise protocols completed were identical with respect to volume and load intensity, but differed in rest interval between sets. This study was conducted in a balanced crossover design; half of the participants performed the low rest protocol (LoR) first and the other half performed the high rest (HiR) protocol first. The experimental overview is depicted in figure 3.

Figure 3: The procedures completed on each day during each experimental period. RMR: Resting Metabolic Rate, RPMS: Rating of Perceived Muscle Soreness, LoR: Low Rest Protocol, HiR: High Rest Protocol.
SUBJECT DESCRIPTIVES

Participants’ weight and body fat measured during each trial period. Body fat percentages were assessed with a hand held caliper (FEI™ Baseline Skinfold Caliper) and calculated using the ACSM’s Guidelines for Exercise Testing and Prescription 9th edition. Skin folds in three locations were measured (triceps, suprailiac, and thigh) by the same researcher during each assessment. Participants’ height was measured during this initial screening visit.

RESTING METABOLIC RATE

Prior to each RMR measurement participants rested in a supine position for approximately 30 min in a dimly lit, quiet, temperature controlled room. Once rested a hood which interfaced with an indirect calorimeter (Parvo Medics TrueOne® 2400) was fitted onto the participant; breath gasses were then measured for approximately 30 minutes. The last 10-15 minutes of RMR values were averaged for statistical analysis.

Assessments of RMR were conducted in the morning hours and participants were post prandial for a minimum of 12 hours. Participants were prohibited from outside exercise for 48 hours prior to each assessment. Participants reported compliance with the exercise, fasting, and rest instructions detailed above.

EXERCISE TREATMENT

Participants completed one bout of exercise during each trial period. The exercise protocol consisted of five exercises performed in a circuit type fashion in the following order: barbell squat, barbell bench press, lat pull, barbell overhead press, and barbell deadlift. Three sets of eight repetitions were completed for each exercise using 75% of participants’ predicted single repetition max. A metronome was utilized to pace each repetition. Repetitions were completed at a pace of one repetition every three seconds.
Each protocol entailed differing rest intervals. Participants were given 150 seconds of rest between each set during the high rest protocol. During the low rest protocol they were given 15 seconds between sets. Stopwatches were used to maintain consistent timing between each set.

**PREDICTED ONE REPETITION MAX**

Prior to any lifting participants had their form assessed; only those able to demonstrate proper technique were included for participation. Predicted single repetition maximal capacities were assessed for each lift using a five repetition max trial (22). This test was conducted as follows: to warm up participants lifted 50% of their self-predicted single repetition max ten times in a single set. Participants were then incrementally loaded until unable to perform five repetitions while maintaining safe techniques. Two minutes of rest were given between each attempt. Participants were spotted and their technique closely monitored during each attempt. Failure to maintain safe posture resulted in termination an attempt. The five repetition max was attained within five attempts or participants were reassessed at a later date.

**RATINGS OF PERCEIVED MUSCLE SORENESS**

Participants rated whole body muscle soreness each morning by self-selecting a numeric value ranging from 1 to 10. Selecting a value of “1” denoted “no apparent soreness/pain, while “10” was expressed as “the worse pain I’ve ever experienced.”

**DIETARY JOURNAL**

Participants recorded the foods they consumed 24 hours before the first day of each trial period, and for duration of each trial period. Participants were provided with a sample dietary journal, written guidelines, and a notebook for keeping track of daily food intake. Participants were asked to maintain typical dietary patterns throughout the experiment.
STATISTICAL ANALYSIS

Statistical analyses were completed using JMP Pro (version 11). Results are reported as mean plus or minus standard deviation. Changes in participants’ body fat and weight were assessed using paired T-Tests. Differences in RMR, RQ, RPMS, and diet logs were each analyzed using 2X3 repeated measures ANOVAs (Protocol X Time). Participants’ diets were analyzed using 2X4 a repeated measures ANOVA. Analyses were deemed significant if the p = 0.05 threshold was met or exceeded.

RESULTS

There were no significant time or protocol effects for RMR observations (p = 0.98 and p = 0.35 respectively), though interaction effect between time and protocol were significant (p < 0.01). On the second measurement post exercise (39 hours post) the LoR protocol was significantly higher than the HiR protocol (p < 0.01). The protocol*time interactions are illustrated in figure 4.

There were no observed effects of time or protocol on RQ (p = 0.08 and p = 0.49 respectively). There were no interactions between time and protocol with respect to RQ (p = 0.57). Participants’ body fat and weight did not change significantly between trial periods (p = 0.32 and p = 0.50 respectively).

Time had a significant effect on RPMS (p < 0.001). Both post exercise observations were significantly greater than baseline values (p < 0.05). Protocol also had a significant effect on RPMS (p = 0.016). There were interactions between protocol and time (p = 0.019). The LoR protocol resulted in greater RPMS than did the HiR protocol (p < 0.05). The protocol*time interactions are shown in figure 5.

Participants’ daily caloric intake was not significantly different across each trial period or between protocols (p = 0.97 and p = 0.57 respectively). There were no interaction effects between
protocol and time (p = 0.75). The sequence of protocols did not significantly impact caloric intake (p = 0.34).

Figure 4: The RMR interactions of protocol and time are illustrated above. Significant differences at the p < 0.05 threshold are denoted by the “*” symbol.

Figure 4: Resting Metabolic Rate
DISCUSSION

The primary aim of this study was to examine the effects of rest intervals the prolonged phase of recovery from a typical recreational lifting workout. Past research has indicated that RMR can be elevated for last long as 72 hours post exercise (23, 24). The mechanisms underlying this phenomenon are not entirely understood, but muscle repair may be a major player (7). Previous studies have found significant increases in RMR on the days following lifting protocols which were designed to elicit muscle damage. The intent was not only to establish a relationship between rest interval, muscle damage, and recovery from resistance exercise, but to do so using a
typical recreational lifting protocol. The repetitions, sets, and intensity were therefore selected to closely emulate the ACSM guidelines regarding resistance exercise.

During each trial post exercise RMR was measured approximately 15 hours after completion of the lifting protocol. No differences in RMR were observed between baseline and this first point of measurement. These findings were atypical as the majority of comparable studies have reported significant increases of between 4-9% in RMR during the first day post exercise (25, 26, 27, 28). The lack of RMR change at the first post exercise assessment point may be associated with the total volume lifted. Many comparable studies have entailed greater numbers of repetitions and sets with the specific intent of causing substantial muscle damage. Muscle damage is associated with increases in RMR days post exercise (7). The prescribed volume lifted may have been insufficient to elicit enough muscle damage to elevate participants’ RMR during the first day of recovery. Additionally, training status is an important consideration as training reduces exercise induced muscle damage (23). Participants were all active recreational lifters so the damaging effects of the exercise bout performed may have been blunted by adaptions secondary to their prior training.

The time of the initial assessment may have also played a significant role in the lack of observed change in RMR. Delayed onset muscle soreness (DOMS) typically occurs between 24-48 hours post exercise (21). DOMS is an immune system process by which the muscle repair and growth processes are stimulated. It is therefore possible that the first measurement (15 hours post exercise) occurred before the process of DOMS was in effect, and this may account for the lack of RMR change observed.

While neither treatment resulted in a change in RMR 15 hours post exercise the LoR treatment did result in a significant increase 39 hours post exercise. This is a novel finding as there
have been few studies which have isolated rest interval’s effects on RMR during recovery from resistance exercise. While no directly comparable studies exist several studies are analogous. Paoli et al. (25) found less rest to result in greater RMR 22 hours post exercise, but these results are convoluted by differences in both volume and intensity between protocols. Conversely, Hunter et al. (5) found rest interval to have no effect on RMR the first day post exercise, but again the treatments entailed differing volumes and intensities.

The mechanisms underlying the increased RMR 39 hours post exercise are not entirely clear, though muscle damage may have been a factor. During each assessment period RPMS was recorded. RPMS is a self-report scale, and is therefore not as accurate or reliable as direct measurements, but previous studies have found significant correlations between self-reported soreness and muscle damage (21, 29, 30). RPMS was elevated after both protocols, but the highest values were observed 39 hour post exercise. Therefore, the reduced rest intervals of the LoR protocol may have caused more muscle damage than the HiR protocol. While muscles damage is typically associated with mechanical loading metabolic acidosis can also stimulate muscle breakdown (31). Both protocols were matched in terms of volume and load intensity, but the rest intervals were drastically different (10 times less time between sets in the LoR protocol compared with the HiR protocol). The bioenergetics demands of the LoR protocol were therefore likely greater than the HiR protocol, which may have resulted in greater muscle damage secondary to increased systemic acidosis.

Substrate utilization was not affected by either protocol. Participants’ RQ values were not significantly different between protocols. This finding corresponds with previous studies which have similarly shown no change in substrate utilization during the prolonged phase of recovery
from resistance exercise (32, 2). Caloric intake was also not significantly different between protocols, and therefore the observed changes in RMR are likely not attributable to changes in diet.

Ultimately, this research has shown that rest interval reduction may increase RMR days following resistance exercise. These findings may be a result of greater muscle damage resulting from decreased rest. Though significant differences in RMR were recorded between protocols during a time point the differences were not large enough to meaningfully impact daily caloric balance. These results would likely have been magnified had the protocols included greater numbers of exercises, and more total volume of work.
CHAPTER IV: THE EFFECTS OF VINEGAR SUPPLEMENTATION ON RECOVERY RATE

PREFACE

Several studies have demonstrated a link between acetic acid feeding and increases in glycogen repletion rates in animal models (10, 11, 12). These studies were conducted on mice and horses, but these effects had not been verified on humans. The ability to perform at peak capacity is tightly linked with one’s stores of glycogen. Intense exercise rapidly diminishes muscle glycogen, which can lead to impaired performance (11). The process of glycogen repletion can be lengthy, and many sports entail multiple performances in a single day (12). The ability to recovery from each performance is therefore vital to maintaining peak physiologic capacity during each subsequent performance. The potential benefits of increasing the rate of glycogen repletion are therefore self-evident.

This study was therefore conducted to provide proof of concept of the efficacy of acetic acid as an ergogenic aid for recovery from exhaustive exercise. The treatment containing acetic acid was hypothesized to improve performance post recovery to a greater extent that the placebo treatment.

METHODS

EXPERIMENTAL OVERVIEW

Recruitment and testing was conducted on the campus of Louisiana State University, Baton Rouge, LA. Participants first completed an informed consent and health history documents in accordance with the institute review board. Participants then had their height, weight, body fat, and peak power assessed. Participants underwent two trials of identical intensity to test the effects of vinegar supplementation on the rate of recovery from exercise. Trials began with a brief warm up and a series of sprints on the cycle ergometer (Monark® 818E). Participants then cycled for
30 minutes at 75% of their max. Low intensity resistance exercise was then performed to further fatigue the participants. Participants then consumed a recovery beverage with or without vinegar and rested for 2 hours. Following the recovery period participants performed a warmup and a sprint trial using the same protocol are the initial. The experimental overview is illustrated in figure 6.

![Experimental Overview Diagram]

Figure 6: Experimental Overview. Participants first performed a sprint trial which was followed by an exhaustive exercise protocol. Then participants consumed an experimental recovery beverage and were given two hours of recovery time. Following recovery they completed another sprint trial.

POPULATION

Eleven healthy female participants volunteered for this study. Participants did not regularly cycle, and were therefore considered untrained. Participants completed a PAR-Q, were consulted about the potential risks associated with participation, and gave their informed consent prior to exercise testing. Participants’ characteristics are included in table 3.

Table 3: Participant Characteristics

<table>
<thead>
<tr>
<th></th>
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<th>St.Dev.</th>
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<tr>
<td>Height (m)</td>
<td>1.63</td>
<td>0.07</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>20.81</td>
<td>3.64</td>
</tr>
<tr>
<td>Max Work (Watts)</td>
<td>136.36</td>
<td>13.06</td>
</tr>
</tbody>
</table>
DESCRIPTIVES

Participants’ height, weight, and body fat were assessed during each their trial period. Body fat percentages were estimated using the female’s three site equation detailed in the ACSM’s Guidelines for Exercise Testing and Prescription 9th edition (triceps, suprailiac, and thigh sites). All skin fold measurements were performed by the same researcher using a hand held caliper (FEI™ Baseline Skinfold Caliper) for consistency.

MAX POWER TEST

Prior to testing participants completed a 5 minute cycling warm up using low resistance. Without leaving the ergometer participants then were instructed to maintain a cadence of 100 rpm as the resistance on the fly wheel was gradually increased (25 Watt increments) every minute until the participant was unable to maintain cadence. The last wattage participants maintained for an entire minute was recorded as their maximal power.

SPRINT TEST

Following a 5 minute cycling warmup participants were instructed to maintain a cadence of 100 rpm, while 150% of their recorded maximal power was applied as resistance. This was maintained for 1 minute, or until the participant was unable to maintain cadence. Participants who completed the entire minute of work were given 2 minutes of rest, after which another minute of cycling began. Participants continued to cycle and rest in this fashion until they were unable to maintain cadence for 10 contiguous seconds, at which time the test was terminated. The total number of seconds completed during each 1 minute work interval was recorded as the participant’s sprint time. This test was administered prior to the exercise protocol, and following the recovery period.
EXERCISE PROTOCOL

Participants cycled for 30 minutes on a cycle ergometer with the resistance set at 75% of their previously determined maximal power. The cadence during this exercise protocol was determined by the participant, but kept equal between trials, and not less than 70 rpm. After cycling participants performed 3-5 sets to failure of leg extensions (Body Masters® CX110) using low resistance (10-20 lb.).

RECOVERY BEVERAGE

Participants were given a recovery beverage which consisted of 2 cups of grape juice, either with or without 2 tablespoons of red wine vinegar. Participants consumed the beverage with 5 minutes of completing the exercise protocol. While participants were not informed of the treatment being administered, though the strong flavor and smell of the vinegar additive belied the researcher’s attempt to blind participants to the contents of the treatment. Each recovery beverage was well tolerated.

STATISTICAL ANALYSIS

Statistical analyses were completed using JMP Pro (version 11). Results are reported as mean plus or minus standard deviation. Paired t-tests were utilized to determine the differences between pre exercise sprint durations, changes from pre to post exercise sprint durations, and the percent change in sprint duration between trials. A one way ANOVA was used to determine if the order of treatments affected the duration of sprint tests. Analyses were deemed significant if the p = 0.05 threshold was met or exceeded.

RESULTS

The duration of pre exercise sprints were not significantly different between trials [72.91±19.84 seconds versus 65.82±18.98 seconds respectively (p = 0.07)]. Both treatments
resulted in significant reductions in sprint duration (p < 0.05). The percentage change between pre exercise to post exercise sprint times were significantly lower with vinegar trials than placebo (p = 0.006). Figure 7 illustrates the pre and post exercise sprint durations for each treatment. The order of treatments did not significantly impact the duration of sprint trials (p = 0.37)

**Figure 7: Sprint Durations**

![Figure 7: Sprint Durations](image)

Figure 7 depicts the differences in performance either with or without vinegar supplementation following exhaustive exercise. The “*” symbol signifies a significant reduction in sprint performance.

**DISCUSSION**

This study was conducted to test the effects of acetic acid feeding on recovery from exhaustive exercise. Vinegar was therefore added to a carbohydrate based beverage and given to participants following exercise. The consumption of vinegar resulted in significantly faster recovery than did the placebo treatment. These findings support those of Fushimi and Nakao who previously found acetic acid feeding to increase recovery rate (10, 11, 12).
While the present study did not test for the mechanisms underlying the increase in performance past work in rodent models suggests that an increase in the rate of glycogen synthesis may be responsible (10, 11, 12). Increases in glycogen repletion and glycogen synthase activity have all been observed following acetic acid feeding post exercise.

The work in rodent models was later repeated by Waller et al. (33) who examined the effects of acetic acid feeding on horses. These researchers found that the addition of acetic acid to the post exercise meal of equestrian horses resulted approximately 3 times greater glycogen repletion than without during the first 4 hours of recovery. This study showed an initial increase in glycogen repletion, but 24 hours post exercise the groups were not different. The findings of the present study further support acetic acid’s role in recovery from exercise, and potential for improved rate of glycogen synthesis.

The cellular actions of acetic acid feeding on improving glycogen synthesis are not fully established, but past researchers have theorized acute changes in glucose metabolism to be a major player. Acetic acid feeding in mice has been shown to increase cytoplasmic citrate levels in mice (15). High concentrations of citrate inhibit PFK-1 (a major rate limiting enzyme of glycolysis) (34). Reductions in PFK-1 activity may result in increased glucose-6-phosphate concentrations which have been shown to stimulate glycogenesis (35, 36).

The ability to perform exercise at high intensity is closely related to muscle glycogen stores (14). The exercise protocol utilized in this study was designed to deplete muscle glycogen, as the modality and intensity of the exercise protocol were similar to previous designs which have demonstrated significant muscle glycogen depletion (37). Performance was assessed as a substitute for direct measurements of muscle glycogen, as they are highly correlated (38). Though
correlated, performance is not entirely dependent on muscle glycogen. Therefore while increased glycogen synthesis may have contributed to the observed results this theory cannot be validated at this time.

Statistical analysis of the pre exercise compared with post exercise sprint trials to be significantly less in the vinegar feeding group. Though these results were highly significant the baseline values may have skewed the results. While not statistically significant the pre exercise sprint times of the vinegar trials were approximately 7 seconds lower than those of the placebo. This disparity between trials may therefore have inflated the results. This difference in performance is not clear, though participants’ training status could have been a factor. Participants of this study were untrained, and therefore their work output may have been less consistent was would be expected from more trained participants.

Future research should be conducted to verify the findings of the present study further develop an understanding of the effects of acetic acid on recovery. Though significant improvements in recovery rate were observed following acetic acid feeding the underlying mechanisms of action are not known. To better understanding the effects of acetic acid on recovery metabolic responses should be monitored during future experiments. Additionally, the population should have a more appropriate training status. Rapid recovery from exercise primarily benefits athletes, and therefore future research should be conducted on trained participants.
CHAPTER V: VINEGAR SUPPLEMENTATION IN ATHLETIC POPULATIONS

PREFACE

The results of the initial study examining the effects of acetic acid feeding on recovery rate prompted this second investigation. The purpose of this study was to verify and expand on the initial findings. Acetic acid consumption resulted in significant improvements in recovery rate in the first experiment, but this was conducted on untrained participants. Sedentary populations do not benefit greatly from this research as repeated high intensity performances are not common events for this population. This research benefits athletes primarily, so this study was conducted with athletes as participants.

To better study acetic acid’s effects on recovery a more robust study design was devised. Participants completed more trials with a greater variety of treatments. The intent of completing more trials was to distinguish the effects of acetic acid from those of the carbohydrate consumption with greater clarity. The performance trials were more standardized, and better suited for assessment of muscle glycogen (the theorized mechanism of acetic acid’s action). Additionally measurements of blood glucose and substrate utilization were taken throughout the post exercise rest periods to elucidate the actions of acetic acid. The goal of this design was to further test the efficacy of acetic acid as an ergogenic aid for improved recovery from exhaustive exercise. The treatment containing both acetic acid and sugar was hypothesized to improve performance to a greater extent than the other treatments.

METHODS

POPULATION

Twelve healthy and active participants were recruited from the Baton Rouge area. All participants regularly exercised, though the modalities of their exercise were varied. The training
status of participants ranged from elite level competitive athletes to intermediate recreational exercisers. Participants completed a PAR-Q, were consulted about the potential risks associated with participation, and gave their informed consent prior to exercise testing. This experiment received approval from the Louisiana State University Institute Review Board. Participants’ characteristics are included in table 4.

Table 4: Participant Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>St.Dev.</th>
</tr>
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<tbody>
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</tr>
<tr>
<td>Weight (kg)</td>
<td>75.70</td>
<td>13.40</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.80</td>
<td>0.10</td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>12.00</td>
<td>2.30</td>
</tr>
<tr>
<td>VO$_{2\text{max}}$ (ml/kg/min)</td>
<td>46.48</td>
<td>5.02</td>
</tr>
<tr>
<td>Max Work (Watts)</td>
<td>262.50</td>
<td>49.50</td>
</tr>
</tbody>
</table>

**EXPERIMENTAL OVERVIEW**

Participants had their maximal aerobic power and VO$_{2\text{max}}$ assessed prior to beginning trials. On the same day as the VO$_{2\text{max}}$ test participants also completed a familiarization exercise to attenuate potential learning effects.

Participants completed four trials in a balanced crossover design. Each experimental trial commenced with a 20 kilometer best effort cycling bout. Participants then rested for 90 minutes. Blood glucose levels were measured at the beginning of the rest period, and every 15 minutes thereafter. Breath gas measurements were assessed throughout the rest period in 15 minute intervals. Then, 30 minutes into the rest period, participants consumed a recovery beverage. Following the rest period, participants completed 30 min of cycling with a fixed resistance of
approximately 70% of their maximal wattage assessed during their VO2max assessment. The experimental overview is displayed in figure 8.

![Experimental Overview Diagram]

Figure 8: Experimental Overview. BG: Blood Glucose, IC: Indirect Calorimetry

**TREATMENTS**

Participants consumed one of four beverages during each trial. Beverages were consumed during the recovery period and consisted of a combination of water, vinegar, and sugar. The contents of each beverage are displayed in table 5. All beverages were ingested using a straw and nose clip to diminish participants’ ability to detect differences between treatments.

<table>
<thead>
<tr>
<th></th>
<th>Beverage A</th>
<th>Beverage B</th>
<th>Beverage C</th>
<th>Beverage D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>100%</td>
<td>94%</td>
<td>92%</td>
<td>86%</td>
</tr>
<tr>
<td>Sugar</td>
<td>0%</td>
<td>0%</td>
<td>8%</td>
<td>8%</td>
</tr>
<tr>
<td>Vinegar</td>
<td>0%</td>
<td>6%</td>
<td>0%</td>
<td>6%</td>
</tr>
</tbody>
</table>

**GRADED EXERCISE TESTING**

Participants performed a graded exercise test to determine maximal cardiorespiratory capacities. Peak oxygen uptake (VO$_{2peak}$) and the workload for the steady-state exercise during the trials to follow were assessed using a metabolic cart (Parvo Medics TrueOne® 2400) and magnetically braked cycle ergometer (Racermate® Velotron).

Participants completed a brief warmup (5 minutes of cycling at low intensity) prior to commencement of the graded exercise test. Participants were then fitted with the breath gas analyzer and instructed to pedal at a rate of 70 rpm. The cycle ergometer was set to 35 watts.
initially and each stage (3 minutes) the wattage was increased by 35. Participants cycled at this rate until they reached volitional fatigue. The highest wattage maintained for a stage was designated as the participant’s maximal wattage.

**EXERCISE PROTOCOL**

The exercise protocol consisted of cycling for a simulated distance of 20 kilometers on a cycle ergometer (Racermate® Velotron). Participants were instructed to complete the distance as rapidly as possible. On trials subsequent to the first participants were paced using their initial performance.

**BLOOD GLUCOSE ASSESSMENT**

Blood glucose was measured using a blood glucose monitoring device (ACCU-CHEK™ Compact Plus). Participants’ fingers were sterilized with alcohol prior to being pricked with a lancet. Once the skin was broken a single drop of blood was applied to a test strip and the blood glucose levels were measured. Following collection of participants’ blood a cotton ball was applied to the break in the skin and pressure was applied until bleeding ceased.

**INDIRECT CALORIMETRY**

Breath gasses were assessed during the rest period using an indirect calorimeter (Parvo Medics TrueOne® 2400) using dilution protocols. Participants rested in a supine position in a quiet temperature controlled room before and after a hood device was applied for breath gas analysis. Breath gasses were assessed in 15 minute period with 15 minutes in between assessments a total of 3 times. During the rest period the hood was therefore applied at minute 15, 45, and 75, and removed at minute 30, 60, and 90.
PERFORMANCE ASSESSMENT

Following the rest period, participants completed a 30 minute cycling bout to assess their recovery work capacity. The resistance during this cycling bout was determined by participants’ maximal wattage and VO$_{2\text{peak}}$. The resistance was equal to the wattage produced by the participant at 70% of their VO$_{2\text{peak}}$, and then converted into pounds of drop weight to apply to the cycle ergometer. Participants were instructed to pedal as many times as possible throughout the 30 minute period. Participants were blinded to their work performance during this trial and free of distractions.

STATISTICAL ANALYSIS

Statistical analyses were completed using JMP Pro (version 11). Results are reported as mean plus or minus standard deviation. To determine the effects of the recovery beverages (treatment) and order of trials (sequence) on performance (total work performed during the performance trial) a 2X2 repeated measures ANOVA was performed. To determine the effects of treatments and time points on blood glucose a 2X2 repeated measures ANOVA was performed. Similarly a 2X2 repeated measures ANOVA was used to determine the effects of treatments on RQ and time points. Post hoc analyses were completed using LS Means student’s t-tests, and contrasts. Cohen’s d was used to determine effect size of each treatment. Significance was determined at an alpha level of 0.05 for all analyses.

RESULTS

Treatments did not have a significant effect on total work during the performance trial ($p = 0.22$); the total work performed as a result of each treatment are given in table 6. The order which the trials were performed likewise did not have a significant effect on total work ($p = 0.67$). There were significant treatment * order interaction ($p < 0.01$). Treatment B had a small effect size of -0.15, while treatments C and D had large effect sizes of 0.99 and 1.08 respectively.
Treatments had a significant effect on blood glucose levels (p < 0.01). Treatments C and D resulted in significantly higher blood glucose than did treatments A or B (p <0.05). There was a trend toward treatments B and D resulting in lower blood glucose levels than treatments A and C, though statistical significance was not reached (p = 0.13). The time also had a significant effect on blood glucose (p < 0.01). There were significant treatment * time interactions (p < 0.01). The treatment * time effects on blood glucose are displayed in figure 9.

![Figure 9: Blood Glucose During Rest](image)

Figure 9: Blood glucose responses during the 90 minute recovery period post exercise protocol.

Treatment trended toward having a significant effect on RQ (p = 0.08); these results are displayed in figure 10. Treatments B and D were not significantly different from treatments A and C (p = 0.86). The time of assessment significantly affected RQ (p < 0.01). The first period of
assessment (minutes 15-30 of the rest period) was significantly lower than the last two period of assessment (minutes 45-60, and 75-90) (p < 0.05).

The exercise protocol was completed in 37 minutes and 48 seconds on average, and did not differ significantly between trials (p = 0.99). Blood glucose did not differ significantly at the first point of assessment post exercise (p = 0.21), though the contrast between the first trial and last trial performed by each trial approached significance (p = 0.08). Blood glucose measurements at the beginning of each rest period are shown in figure 11.

Figure 10: The effects of each treatment on RQ throughout the 90 minute recovery period post exercise protocol.
DISCUSSION

The aim of this experiment was to further examine the effects of acetic acid feeding on recovery rate from exhaustive exercise. In order to gain a better understanding of the ergogenic properties of acetic acid, four treatments were tested. This allowed for more levels of comparison than the previously conducted experiment. Additionally, instead of performing a pre exercise and post exercise performance assessment there was only a single post exercise performance test. This protocol was designed to better assess glycogen levels post exercise, this moderate to high intensity
(~70% VO$_{2\text{max}}$) work bout should tax the glycogen stores to a greater extent than did the our previous study’s supramaximal sprints.

The treatments of this experiment did not significantly impact the total work performed. The lack of significant differences between the water and sugar water treatment (treatment A and C respectively) suggests that the intensity of the exercise may have been insufficient to adequately fatigue participants. The consumption of carbohydrates post exercise is a common practice for the recovery of glycogen, and therefore recovery from exercise. The administration of a carbohydrate containing beverage should therefore have significantly improved subsequent work performance more than the water only treatment if the initial fatiguing exercise protocol were sufficient to produce significant glycogen depletion or fatigue.

Alternatively the performance assessment may not have been of sufficient intensity to challenge participants and thus create significant differences. There was only a 5% difference in work performed between treatment A and treatment C. This minor change in performance could have been a result of participants innately pacing their pedaling cadence, or may have been a result of insufficient intensity. Participants were blinded to their performance during the work trial, but with their previous experiences on cycles it is possible that them may maintained a cadence which was familiar and maintained said rate regardless of their true capacities.

The minimal difference between performance trials may additionally be attributable to participant’s motivation. Performance was not incentivized and no external motivational cues were given during their performance. Participants’ lack of motivation to perform is evidenced by the total work performed on the final trial of each participant. Regardless of the treatment participants performed the lowest work on their last trial, which cannot be explained through
physiological mechanisms. Performance during the first trial was lower than the second and third, but this was likely a result of a learning effect. Participants performed a familiarization performance trial on the same day as their VO2max trial, but this may not have been enough to fully acclimate themselves to the cycle ergometer used during the experiment. Immediately following participants’ first exercise protocol their blood glucose levels were the highest. As participants completed subsequent trials their blood glucose levels became progressively lower. Increased blood glucose post exercise may be a result of sympathetic nervous system activity (19). Motor tasks which are unfamiliar result in greater sympathetic nervous system activity resulting from uncoordinated motor unit activation (39). When training motor patterns become more coordinated, therefore less motor units are recruited to accomplish a given task (40). With less muscle activation it is likely that the bioenergetics demands between exercise trials were unequal, and this could have resulted in less fatigue as participants became more accustomed to the performance trial. This should therefore have resulted in a continual improvement in performance had the participants been motivated to perform at their peak and the performance test been capable of gauging their ability to recover.

Though none of the treatments significantly altered work performance effect sizes of each treatment were determined against the control (Treatment A) to determine if there were any meaningful differences. Both treatments C and D resulted in large positive effect sizes, while treatment B was small and negative. Treatment D resulted in the greatest effect size, and therefore appears to have improved performance to the greatest extent. The consumption of glucose was likely made the largest contribution to these results, as glucose consumption is known to improve recovery post exercise.
Blood glucose was significantly increased by treatments containing sugar (C and D) when compared to the non-sugar treatments (A and B). Blood glucose was highest approximately 30 minutes post treatment consumption, which was expected given the typical rate of carbohydrate digestion. When the acetic acid containing treatments were contrasted with the non-acetic acid treatments a nearly significant decrease in blood glucose was observed. Therefore acetic acid feeding may have increased the uptake of blood sugar.

During recovery RQ values were lowest during the first post exercise assessment (approximately 0.7), which suggests that fat oxidation was the primarily substrate utilized during this period (41). The treatment effect on RQ was nearly significant, thus treatments containing sugar (C and D) resulted in increased RQ, while those without had a smaller increase. The reductions in RQ resulting from treatments B and D did not reach statistical significance, though the result may be meaningful. Treatment B contained water and acetic acid and resulted in an elevated RQ compared to treatment A (water only). When compared with the decreases in blood sugar concurrently observed these results may suggest that blood sugar was taken up to a greater extent and metabolized for energy. Treatment D contained sugar and acetic acid. This treatment resulted in a decreased in RQ following exercise, and a decrease in blood glucose. Previous authors have suggested that acetic acid may improve recovery be increasing glycogen storage mechanisms (10, 11, 12, 33). Improvements in glycogen repletion secondary to acetic acid feeding are theorized to be a result of reduction in PFK-1 activity secondary to citrate abundance (13). The acetic acid dosage in to current study was similar to the quantity administered in animal models, therefore may have been sufficient to increase citrate levels to the degree previously observed. The reduction in RQ following treatment D may therefore have been a result of inhibition of glucose metabolism.
Ultimately, the measurements of blood glucose and substrate utilization obtained during this experiment showed trends toward statistical significance and may be meaningful. These results alone suggest that acetic acid may increase glucose uptake, and may influence substrate utilization. The lack of difference in work performance between treatments makes any conclusions as to the efficacy of acetic acid as an ergogenic acid difficult as no improvements in performance were observed.
CHAPTER VI: CONCLUSION

The work completed throughout this dissertation has been directed toward gaining a deeper understanding of how various factors influence recovery from exercise. Recovery from exercise is vital to retention of performance over time, and is largely understudied. The experiments conducted throughout this research have focused primarily on rest intervals effects on energy expenditure during recovery and acetic acid as an ergogenic aid improving recovery rate.

During the short phase of recovery from resistance exercise the total energy expenditure between short and long rest was not significantly different. Rest interval did not have a significant effect on energy expenditure, and therefore may not be as important as volume of work performed or load intensity. The implications of this finding are subjective, yet meaningful to many populations. Those with limited time to devote to exercise will not be negatively impacted by completing the work at a faster pace. Conversely taking long breaks in-between sets is not detrimental to energy expenditure.

Reducing rest interval did result in significantly increased resting metabolic rate and muscle soreness during the prolonged phase of recovery. These results in concert imply that greater muscle damage may have resulted from reducing rest intervals, and this may have increased energetic demands during prolonged recovery. While soreness has been linked with muscle damage this is an indirect assessment, and therefore more robust research must be conducted before this relationship can be verified. The results of this study are directly transferable to recreational exercisers as many within this population are exercising in order to affect caloric balance. Reducing exercise duration while increasing caloric expenditure can benefit millions with limited time to devote to structured exercise programs.
In order to assess how recovery from exercise could be aided, the ergogenic effects of acetic acid were tested. The initial study of acetic acid’s effects on recovery was conducted to verify in humans the previous findings in animal models. Previous research indicated significant improvements in glycogen repletion secondary to acetic acid feeding. Muscle glycogen is essential to high intensity performance, and therefore the implications of this line of research are self-evident. Performance post recovery was significantly greater with acetic acid consumption, which may indicate a greater glycogen repletion rate.

The significant findings of the first investigation prompted additional study using a more robust study design. The second study conducted utilized a more trained population, more treatment levels, and some measures of metabolic activity. This study was performed to verify the initial findings and gain greater understanding of the ergogenic effects of acetic acid. Performance was not improved by any of the treatments as all four were not statistically different. These findings likely indicate a deficiency either within the exercise protocol or the performance assessment protocol, but the metabolic data collected was of value. Though not statistically significant acetic acid consumption appears to result in meaningful increases of glucose uptake. In the context of recovery from exercise, the ability to uptake glucose is vital, which may in itself justify the consumption of vinegar in conjunction with carbohydrates post exercise. The mechanisms underlying this phenomenon are not understood, but further research is warranted as the potential benefits of acetic acid feeding far out weight the risks.

Through completion of this research the goal has been to contribute in a meaningful way to the understanding of recovery. Ultimately this work has raised more questions than it has answered, which is to be expected, given the novel nature of this research. Rest interval may have an effect on prolonged recovery, but the mechanisms have not been established. Acetic
acid consumption post exercise has resulted in mixed performance results, but the true efficacy, dosage, and cellular mechanisms are unknown. The results of these works have implications for millions of people worldwide in both recreational and athlete populations. Future work will focus on continuation of this existing research in an effort to answer the questions which these studies have raised.
REFERENCES


34. Tornheim, K. Activation of muscle phosphofructokinase by fructose 2,6-bisphosphate and fructose 1,6-bisphosphate is differently affected by other regulatory metabolites. Journal of Biological Chemistry 1985; 260:7985–9.


APPENDIX I: GENERAL EXAM

INTRODUCTION

Energy Expenditure

The energy we use throughout the day for the various physiological operations and processes is known as daily energy expenditure. There are three general categories of energy expenditure, which in combination sum to the overall daily energy expenditure (42). The combination of the caloric expenditure of these three distinct areas comprises our daily energy expenditure (43). Daily energy expenditure can be used as a means of assisting with weight maintenance, loss, or gain. In the simplest of terms weight maintenance is just a balance between calories consumed, and daily energy expenditure (44). Taking in more calories than are expended on a given day will result in an increase in weight, while the opposite will result in a loss of weight.

Resting Metabolic Rate

The resting metabolic rate comprises all of the energy production necessary to maintain the homeostatic function of our various organ systems. The resting metabolic rate is typically the greatest contributor to our daily energy expenditure (60-75%) (45).

Thermic Effect of Food

Additionally there is a thermal effect associated with food consumption. This is essentially the energy required to breakdown the foods we eat into useable units for the body. The thermal effect of feeding can comprise as much as 15% of our daily energy expenditure (45).

Physical Activity

Thirdly the energy used for physical motion can make a large contribution to our caloric expenditure, or be nearly insignificant depending upon the activity level of the individual. All
motion produced by the body has an associated cost in energy (46). The energy expenditure associated with physical activity has a massive range of variation, as some people are sedentary while others are highly active.

**Excess Post-Exercise Oxygen Consumption**

The phenomenon of increased oxygen consumption following exercise was first termed by Hill and colleges as “Oxygen Debt” (47). This term was later revised by Gaesser and Brooks to Excess Post-Exercise Oxygen Consumption, or EPOC (48). EPOC, as it is now known was found to be a more accurate description of the condition, as the body is not necessarily paying off a debt of oxygen, but performing a plethora of operations. This period of recovery from exercise can be broken into three phases.

**Immediate Phase**

At the cessation of exercise there are several processes operating which contribute to the observed EPOC. The immediate phase comprises a very short period of time, when the systems which require little time to replenish themselves are contributing to the EPOC. While the other short phase and long phase processes are also ongoing this section deals strictly with the systems which recover within five minutes of cessation of exercise.

**Creatine Phosphate**

Unique to the first few minutes following intensive exercise there is a need to replenish creatine phosphate stores to homeostatic levels (17). Creatine phosphate is used for immediate ATP production, typically in the first few seconds of exercise, and is often associated with high power type movements. When exercise is sustained the creatine phosphate system is still working, though its relative contributing to exercise is relatively low compared to both the glycolytic and oxidative systems. Following exercise the creatine phosphate stores must be
restored as a means of readying for further use. This process is fairly rapid, and lasts for no longer than minutes for the majority of creatine phosphate levels to be recovered (49).

**Autonomic Control**

Additionally, following exercise, the heart rate remains elevated until the parasympathetic nervous system’s influence is restored, and the sympathetic nervous input is reduced (50). The body tends to operate in terms of feed-forward mechanisms, so as exercise ceases, this change in autonomic control may take several minutes, or much less for trained individuals (51). Depending upon a plethora of factors this process may take longer than five minutes, though typically this is not the case.

**Myoglobin / Hemoglobin Replenishment**

During exercise myoglobin and hemoglobin may have their stores of oxygen depleted, while this process may not be metabolically taxing, it does account for a portion of the observed increase in oxygen consumption following exercise (52).

**Short Phase of EPOC**

These processes, which contribute to EPOC, are those which last somewhat longer than the immediate phase, but take less time than the prolonged phase to recover. This short phase comprises the processes which take less than three hours to replenish, but more than a few minutes. The immediate and short phases of recovery are generally combined for the purposes of determining excess caloric expenditure post exercise. Increases in energy expenditure in excess of 40% above baseline values are often observed during this phase of recovery.

**Catecholamines**

During exercise catecholamines (Epinephrine and Norepinephrine) are continuously produced. They influence most every aspect of the physiological response to exercise, and can
account for a portion of the observed EPOC following exercise (53). Their role in EPOC is likely on several systems which then contribute to increased energy usage, and therefore demand. Catecholamines act to increase both heart rate and its contractile properties, which in turn results in an increased oxygen usage within the heart to produce the requisite ATP to maintain function (54). Following exercise catecholamine levels are elevated, as the body may require several hours to return these circulating hormones to resting values (53). While catecholamines are in the system, there will be an increased volume of oxygen consumed.

**Processing Lactic Acid**

The lactic acid produced though exercise at sufficient intensity must be dealt with by the body. This lactic acid accumulated will be either metabolized by being converted to pyruvate for oxidation, or converted to glycogen in the liver through gluconeogenesis (55). These processes each contribute to the observed increase in metabolic activity following exercise.

**Temperature**

During exercise, as a result of the increased metabolism occurring to meet the demands of activity, the body increases in temperature. This temperature increase is beneficial to many physiological systems, and aids in the maintenance of activity (56, 57). Metabolism is greatly aided by having a higher temperature, as the metabolic pathways are governed by enzymes, which operate more quickly at slightly greater than normal temperatures; the result in a significant increase in metabolic rate (58). This increased metabolic rate does not cease instantly following exercise. Rather it persists until body temperature is restored to resting level. This process of giving off heat following exercise is dependent upon many factors, but typically can take up to several hours following exercise.
Prolonged Phase of EPOC

Following the initial short phases of EPOC there is a longer component which has been observed to last for days following a single bout of activity (59). The increase in metabolic rate during this phase is typically much more subdued (often below 15%), but even a very small increase in metabolic rate can amount to significant caloric expenditure over a long period of time.

Glycogen Re-synthesis

During exercise glycogen stores are taxed within the skeletal muscles. Glycogen stores are amongst the first energy reserves to be utilized during intense physical activity, and during high intensity activity serve as the primary source of fuel (13). Following exercise the body immediately begins to re-synthesis glycogen, but depending upon how much glycogen was used, this process may be quite lengthy. The process of completely replenishing glycogen stores may take days (60). The process of storing glucose as glycogen requires energy, so this prolonged process is a contributor to the minor increase in metabolic rate observed long after exercise (61).

Protein Synthesis

Following sufficient exercise stimulus there is an increase in protein synthesis to both repair the damage caused said exercise, and also to build new tissue (62). Exercise can stimulate hypertrophy of muscle, mitochondrial biogenesis, restructuring of tendons, or a myriad of other processes, all of which require energy to accomplish (63, 64, 65). These processes of tissue repair and growth therefore can be linked to the prolonged EPOC phase observed following exercise. These processes can last several days, and depending upon the volume of exercise, may be a major player in the prolonged increase in oxygen consumption following resistance exercise (66).
Traditional View of Resistance Training and Energy Expenditure

Role in Weight Management

When considering resistance training as a means of producing fat loss, or favorable body composition changes, the typical viewpoint considers mainly its ability to induce lean mass gain (44). It is well understood that resistance training has the benefit of inducing muscle hypertrophy, which has ramifications for one’s metabolic rate (67). Skeletal muscle accounts for approximately 22% of an average person’s metabolic rate (68). Therefore, should the quantity of muscle mass be increased there will be an accompanied increase in absolute resting metabolic rate.

While the fact that metabolic rate does increase from an absolute means there remains questions about the relative changes in metabolic activity with resistance training (69). Some forms of chronic physical activity have been shown to increase relative metabolic rate, which is to say that pound for pound fat free mass may have differing metabolic rates (70). Research into the effects of resistance training on relative changes in metabolic rate lack consistency, and will be analyzed in greater detail later in this review.

Resistance Training vs. Cardiovascular Exercise

Resistance training requires little caloric expenditure during the exercise, which may lead to the assumption that its contribution to daily energy expenditure is negligible (70). By comparison, cardiovascular exercise often generates much more energy expenditure for a given period of time. This difference in caloric expenditure makes sense when one examines the nature of the two exercises. During a traditional bout of cardiovascular exercise one is typically brought up to a steady work state and held there for a period of time. This steady state work produces a far greater caloric expenditure than typical resistance training during the activity,
which is comprised of working muscles at a much greater capacity for very short periods of time, and then taking breaks in between exertions (45). Though steady state work does entail greater caloric expenditure during exercise than interval type work, there tends to be an increase in caloric expenditure following said exercise.

The variable often overlooked in this equation is the lasting consequences of the exercises (69). During resistance training the caloric demand may be fairly low, but the Excess Post-Exercise Oxygen Consumption (EPOC), is generally greater following resistance training. The first few minutes following exercise is often solely considered the EPOC, but there is research suggesting that there may be significant increases in metabolic rate for several days following a single bout of exercise (71). When the entire EPOC is factored in, resistance training may produce a greater expenditure of energy than is typically considered.

When comparing the caloric expenditure of either resistance exercise or cardiovascular exercise both the energy expenditure during and after should be considered. Additionally, the chronic adaptations of resistance training, specifically muscle hypertrophy, cannot be ignored when comparing the effects of resistance training to cardiovascular training. Muscle is highly metabolic, therefore developing greater amounts of muscle mass is often associated with an increase in resting metabolic rate. When all of these factors are considered resistance training may result in greater energy expenditure rates, than has typically been associated with it.

Substrate Utilization

The energy, or ATP, used by the body for its various functions is derived by the various things we consume, which are in turn converted into useable substrate for the metabolic pathways of the body. In the simplest of senses, the body uses fat, carbohydrate, or protein to produce energy. The processes of producing usable energy from these macronutrients vary
depending upon many factors, one of which is rate (72). Fat oxidation for example provides the greatest amount of ATP production per gram of substrate, but there are many steps to its metabolism, and therefore it cannot be produced very quickly (73). By contrast glycolysis does not produce a large amount of ATP per glucose molecule, but the process is far faster (74). This difference in rate of energy production between the metabolic pathways of skeletal muscle drives the cross-over concept.

Cross-Over Concept

This concept, developed in the 1994, by Brooks and Mercier, describes how our bodies shift between primary energy producing metabolic pathways (75). This is to say that as work rate increases, and therefore ATP utilization, the body must metabolize substrates to meet the demand. When the body is using relatively small amount of energy, such as sitting still, or walking at a low intensity then fat oxidation may be the primary metabolic pathway in use by the body as a whole (76). In this example the rate of ATP utilization is low, and therefore a metabolic pathway that is slow can meet the demand. Conversely, as one begins to work harder, such as running, the oxidation of fat may not be fast enough to provide the ATP required to maintain that work rate. When the demands upon the energy production systems become too great for oxidation then faster metabolic systems must be called upon (77). This idea of shifting from one substrate to another is the cross-over concept. The theory simply states that one’s metabolic pathways begin to call upon glycolysis more as the work rate reaches maximal, and will call upon fat oxidation more as work rates are closer to rest.

Purpose of the Review
This review is intended to analyze the existing literature involving resistance type exercise and its post exercise effects on both energy expenditure and substrate utilization. Specifically this review will cover EPOC, recovery substrate utilization, and changes in metabolic activity (RMR/RER) secondary to resistance training.

**Scope of the Review**

This review will focus on the post exercise effects of resistance training. To this end the after effects of resistance training will be evaluated from both an acute and chronic perspective. Changes in lipid oxidation will be the main focus of the substrate utilization section.

The acute portion of this analysis will cover EPOC and the changes in substrate utilization following resistance exercise. This analysis of literature will be divided into the short and long phases of recovery. Articles included in this portion of the review must have reported acute post exercise volumes of oxygen for the energy expenditure portion, and ratios of oxygen to carbon dioxide for the substrate utilization portion.

The chronic portion of this review will focus on changes in resting metabolic rate and substrate utilization as a result of resistance training. These articles will be either longitudinal training studies or cross-sectional in nature. Changes in resting metabolic rate will be the topic of interest when considering resistance training’s effects on energy expenditure. Changes in resting respiratory exchange ratio will be the primary means of determining alterations in substrate utilization.
SHORT PHASE OF RECOVERY: ENERGY EXPENDITURE AND RESISTANCE EXERCISE

The short phase of EPOC typically lasts from several minutes to a few hours, depending upon the intensity and volume of the exercise performed. The initial response following exercise is a very large increase in oxygen consumption that is fairly transient; this immediate phase typically lasts only a few minutes before subsiding into a phase of less elevated oxygen consumption. Following the immediate phase of EPOC energy expenditure is typically elevated far beyond that of resting values, but not nearly as elevated as during the immediate phase. In the majority of research exploring resistance exercise and energy expenditure during recovery the immediate and short phases of EPOC are considered together. Therefore, for the purposes of this review they will be combined, and simply referred to as the short phase of EPOC.

Characterization of EPOC Response

It was not until the early 1990’s that researchers began looking into the effect of resistance exercise on EPOC. Many of the first articles published on resistance exercise and EPOC simply characterized the change in oxygen demand following resistance exercise.

In 1992 Melby et al. (78) had young trained male subjects perform 7 lifts (3 sets of 10 reps at 70% of their 1RM, with 2 minute rest intervals). As a result of this protocol they found an average of 27% increase oxygen consumption for the first hour of EPOC. While only the first hour post exercise was recorded, they did notice that energy expenditure was still elevated at the completion of their EPOC measurement time frame. Later this research team conducted a similar experiment, using largely the same methods, except for more sets and a few more exercises (27). In their second study they measured found energy expenditure to be elevated for the entire 2 hour measurement period. At the end of this period energy expenditure was still 12% greater than baseline.
These findings were later supported by Osterberg et al. (79), who found that over the 3 hour period of recovery recorded there was an average of 13% more energy expenditure following a single bout of resistance exercise. The protocol utilized in this study was similar to those employed by Melby et al. (78, 27), but young trained females were the subjects within this study.

In 2001 Binzen et al. (80) likewise found an average increase of 19% in energy expenditure during the short phase of EPOC as a result of resistance exercise. Unlike previous authors they observed the short phase of EPOC to end within 90 minutes of cessation of exercise. The shorter duration of EPOC observed in this study is somewhat puzzling as their exercise protocol was similar to the previous works (10 exercises, 3 sets, 10 reps, 70% of 1RM, 1 minute rest intervals). They also used young trained females, like Osterberg et al. (79), but the EPOC was half the duration. The cause of these discordant results is not clear, but some confounding factors must have been responsible.

Later, Shuenke et al. (81) performed a lower volume workout protocol (only 3 compound lifts), and found an average of 36% increase in metabolic rate during the recovery from work in their young trained male subjects. Subjects performed 4 sets of the bench press, the power clean, and the squat for 8-12 repetitions with a 2 minutes rest interval while working at 70%-80% of their 1RM. These researchers only measured the short EPOC for 30 minutes following exercise. Energy expenditure did not return to baseline within this period, so the duration of the short EPOC phase was simply not addressed by this study.

Melanson et al. (2) in 2005 found a 49% increase in energy expenditure over the first 30 minutes of recovery from resistance exercise. These untrained middle aged females performed 10 exercises (4 sets of 8-12 repetitions at approximately 70% of their 1RM with about 3 minutes
rest interval). This finding is of much greater magnitude than those listed previously, but what must be considered is that many previous works reported the average of several hours of recovery, whereas this study only reported the first 30 minutes. This study also used whole room indirect calorimetry as opposed to the typical metabolic carts utilized for this type of research. This method has the obvious advantage of allowing subjects to move, and breathe, naturally while exercising because there are no cumbersome apparatus attached to them.

In 2007 Ormsbee et al. (82) experimented on young trained males and found that there was only an approximate 10% increase in energy expenditure over the 45 minutes recorded following their bout of resistance training. Young trained male subjects performed 6 exercises using a fairly typical 3 sets of 10 repetitions at 70% of their 1RM protocol. While this workout was nearly identical to many of the previous works discussed, the magnitude of EPOC was much smaller. Other authors have reported responses of greater magnitude up to several hours post exercise, while these researchers observed a meager 10%. This differences may be the result of their having implanted a dialysis device onto their abdomens prior to exercise, which may have elevated their baseline energy expenditure or simply made subjects uncomfortable while moving (so perhaps they did not exert themselves to as greatly).

Benton et al. (83) found a 20% increase in energy expenditure as a result of resistance exercise over the 2 hour period of EPOC they recorded. These untrained middle aged female subjects performed 3 sets of 8-12 repetitions at approximately 80% of their 1RM on 8 exercises. When individual results were compared they noted that absolute volume of work and short EPOC magnitude appeared to be positively correlated.

Recently, in 2012 Scott et al. (84) characterized EPOC as not simply being the period of time after a whole bout of resistance exercise, but posited that after each interval of work, or set,
that there are minor EPOC periods. These finding imply that simply measuring EPOC following resistance exercise, without measuring energy expenditure throughout will not accurately gauge the recovery dynamics occurring as a result of resistance exercise.

The general consensus of research in the field of resistance exercise and EPOC is that there is a rather large transient elevation of energy expenditure immediately following exercise. Though all of these studies have detailed an increase in energy expenditure there seems to be a large degree of variability between findings. This is likely due to any number of factors including, but not limited to, differences in subject characteristics, modality of exercise, workout intensity, volume, and time to completion. What can be collectively gathered from these articles is that there is an increase in energy expenditure following resistance exercise, though the duration and magnitude of this response has a wide range of variability.

Comparisons of Resistance Exercise to Endurance Type Exercise

Another means of investigating the recovery energy expenditure responses of resistance exercise has been to compare it to endurance exercise modalities. To this end researchers have simply pitted resistance exercise against endurance exercise by means of calories expended, time completed, or intensity, and looked at the differences in energy expenditure following each respective bout.

The first to do so was Elliot et al. (85) in 1992. This group compared the effects of 40 minutes of traditional lifting, circuit training, or cycling on young active male subjects. A serious limitation of this study was their usage of varied intensity and modality with the only consistent factor being time to completion. While this was perhaps done to ascertain what type of exercise would result the greatest caloric expenditure for a given period of time the large number of unaccounted for variables make interpretation difficult. They observed that while
endurance training resulted in greater energy expenditure during work that both lifting treatments had short EPOC phases of significantly greater magnitude, about 59% more.

Later, Gillette et al. (86) compared an isocaloric bout of lifting with cycling and had similar results to Elliot. Resistance exercise resulted in significantly greater energy expenditure than either controls or the endurance exercise treatment over the 2 hour EPOC period recorded. These young trained male subjects all lifted before the cycling bout to determine the energy expenditure during the resistance exercise treatment, then cycled for at 50% of their VO\textsubscript{2max} for as long as it took to reach the same caloric expenditure. The lifting session included 5 sets of 10 exercises performing 8-12 repetitions per set at approximately 70% of their 1RM. While matching energy expenditure during exercise this experiment failed to account for the anaerobic portion of work performed during resistance exercise, which likely resulted in an underestimation of calories burned. Therefore, the work performed was almost certainly not matched, although this was the intent.

Burleson et al. (87) in 1998 conducted a study which was both isocaloric and isotemporal while comparing resistance exercise to treadmill exercise. Like many others they used young trained male subjects, but their exercise protocol was somewhat odd. Subjects performed 8 exercises, but only 2 sets and they did not count repetitions, but instead timed each set (30 seconds). Also the intensity was only 60% of their 1RM. The endurance training treatment was the same total time to completion with intensity modulated to match the same rate of caloric expenditure. They found greater energy expenditure during the first 30 minutes following resistance exercise, though by 60 minutes the difference was no longer significant. These researchers only measured the first 30 minutes of EPOC fully, but then at 60, and 90 minutes post exercise they measured their rate of energy expenditure again. Therefore the timing of
when their energy expenditure rates equalized seems to have occurred sometime after 30 minutes, but before 60 minutes.

Melanson et al. (88) approached this topic by matching neither caloric expenditure during activity nor time to complete the workload. They instead chose to match intensity of single repetition max to intensity of VO$_{2\text{max}}$; each treatment had an intensity of 70% (of 1RM for lifting, and VO$_{2\text{max}}$ for cycling). The lifting protocol consisted of 10 exercises with 4 sets of 10 repetitions using 70% of their 1RM with 3 minutes rest intervals. These trained young male subjects lifted for a total of 70 minutes, and cycled for 50 minutes. The resistance exercise treatment resulted in a 5% greater magnitude of short EPOC during the 30 minutes observed. The endurance exercise treatment, while resulting a smaller EPOC, required much more caloric expenditure during work. When the total energy expenditure of both work and EPOC were combined there were not significantly different in calories expended. The results of this work differ greatly from the observations of previous researchers. This is likely due to the matching work based on intensity of 1RM versus VO$_{2\text{max}}$.

Like the work of Gillette, Braun et al. (89) compared circuit weight training with endurance exercise, in this instance treadmill walking, on the basis of isocaloric work performed. These untrained young female subjects performed 8 exercises using 3 sets of 15 repetitions at 65% of their 1RM for the resistance exercise treatment. For the endurance training treatment they walked until they had matched the caloric expenditure of the resistance exercise treatment. When the two treatments were compared the resistance exercise elicited approximately 20% more energy expenditure than the endurance training over the 60 minute EPOC recorded.

When comparing the energy expenditure during recovery from both resistance and endurance exercise the existing literature has shown resistance exercise to evoke a greater
response. While the majority of the studies have shown this, it is important to recognize that matching caloric expenditure between the two modalities is problematic. Resistance exercise is largely anaerobic, and therefore the oxygen consumed during a resistance exercise bout is not wholly representative of the energy expended. Almost none of these authors measured the lactate responses during resistance exercise, and therefore the anaerobic portion of work was not accounted for. Therefore, it is likely that when several of the above authors have attempted to control their experiments by matching calories expended during exercise (on the basis of oxygen consumed during the resistance exercise bout) that they have underestimated the work performed. This in turn implies that their studies were not matched, and rather that the endurance training treatments did not require as much work as the resistance exercise treatments. Therefore the increased EPOC response observed following resistance exercise may simply a result of the greater work performed. This may have contributed to the disparity of EPOC response observed in some of these papers.

Effects of Intensity

Several researchers have sought to ascertain the effects of manipulating the intensity of load during resistance exercise and observing how this factor interacts with the recovery energy expenditure. Within this area of research an important, potentially confounding, variable to control is the total volume of work performed. Therefore within this section the studies with and without volume controlled for have been grouped separately.

Equal Volumes

The first study to investigate the effects of intensity on recovery energy expenditure was Olds et al. (90). In a crossover design their young active male subjects performed 2 sets of 7 exercises, using either 12 repetitions at 75% of their 1RM, or 15 repetitions at 60% of their 1RM.
EPOC was measured continuously throughout and for 60 minutes following each trial. The heavy condition elicited approximately 10% more energy expenditure than did the light. While statistically significant, this difference is fairly small from an absolute perspective, had the intensities been more distinct the influence of intensity may have been illustrated to a greater extent.

Later, Thornton et al. (91) performed such a study by separating the intensities much more drastically. Within this study subjects performed 2 sets of 9 exercises using either 15 repetitions at 45% of their 1RM, or 8 repetitions at 85% of their 1RM. Accordingly, their results were far different from those of Olds et al. (90). Thornton observed that during the first 20 minutes of EPOC that there was approximately 91% more energy expenditure with the heavy condition than the light. Following this initial large difference the remainder of their 120 minute EPOC assessment period did not show any differences between the treatments. What should be noted is that they did not record a full 120 minutes of breath gases, but instead only recorded at several time points within the short EPOC. Therefore, the point where the energy expenditure rates of the different treatments equalized could not be determined.

Wu et al. (92) then examined the effects of 75% versus 50% intensity, and similarly found that heavy lifts resulted in approximately 25% greater energy expenditure than light intensity over the 120 minute period of recovery assessed. These young trained male subjects performed 8 lifts using 3 sets of either 10 repetitions at 75% of their 1RM, or 15 repetitions using 50% of their 1RM.

Thornton et al. (93) later conducted another experiment using the same intensity scheme as their previous work (91). Once again, using young female subjects, they found that higher intensity resulted in a much larger magnitude short EPOC. In this instance when the entire 120
minute recovery period was averaged the high intensity treatment resulted in 49% greater energy expenditure than did the light treatment. While this work further confirms their initial findings, they did not record the full volumes of breath gases, but instead only captured several rates within the short EPOC. Therefore any fluctuations outside of the points they assessed were not recorded, and this is a limitation of their work.

All of these studies observed that lifting with greater intensity results in more recovery energy expenditure when volumes were equivalent. When considered together these studies show a sort of dose response between intensity and EPOC. The studies with a small divide between intensities found a more modest difference in response, whereas those with large differences in intensities observed large differences in energy expenditure response.

**Unequal Volumes**

In 2005 Kang et al. (94) compared three different intensities for differences in recovery energy expenditure. Their trained young male subjects performed only 1 exercise for 4 sets using either 15 (60% of 1RM), 10 (75% of 1RM), or 4 (90% of 1RM) repetitions per set. The light intensity treatment was 20% more work than the medium intensity treatment, but resulted in 5% less energy expenditure during the 40 minute EPOC recorded. The high intensity treatment resulted in the least energy expenditure of any, but it is important to consider that this treatment had less than half of the volume of the others. This study may illustrate an intensity and volume dynamic; whereby increasing the intensity such that volume is significantly reduced may attenuate recovery energy expenditure, even though intensity has previously been demonstrated to be positively correlated with EPOC.

Almeida et al. (95) in 2011 performed a similar study, but with only a heavy and light condition (80% and 55% respectively). Their young male subjects were untrained, and
performed 3 sets of 2 exercises using sets to failure with the intensities previously mentioned. They found that both conditions resulted in approximately the same magnitude of short EPOC, though they only measured EPOC for 10 minutes following exercise. Additionally, during the light treatment subjects performed approximately three times the volume of the heavy treatment. While the energy expenditure responses were not significantly different this study actually supports the role of intensity in increasing the magnitude of EPOC. With 3 times the work performed one would expect there to be greater recovery energy expenditure, but instead they were equivocal. Though, once again, having only measured 10 minutes of EPOC, the true implications of intensity on recovery energy expenditure was not thoroughly examined.

Lastly, Scott et al. (96, 97) published two papers on this topic in 2011. Both studies utilized young trained male subjects performing only the bench press. Collectively they used a range of intensities from as low as 36% to as high as 90% and they observed that heavier lifting resulted in greater energy expenditure relative to volume. Though, while greater intensity resulted in more recovery energy expenditure lighter intensity resulted in larger total energy expenditure from an absolute standpoint. The length of EPOC was not reported in either article, simply the net volume of oxygen above resting recorded before the energy expenditure rate reached baseline. These studies were based around either 1 or 2 sets of only 1 exercise, therefore the EPOC volumes were quite small in magnitude, and their generalizability to full workouts may be limited.

These studies, which did account for volume of work performed during work provide valuable information about the relationship between intensity and volume (which often are inversely related). Like the results of comparing resistance exercise with endurance exercise, the greater the intensity of work (based on 1RM) the greater the EPOC response. It is well known
that with higher intensity work more anaerobic metabolic pathways must be utilized. The metabolic pathways utilized during work may be an underlying cause of the differences often observed between work of differing intensities. Even when volumes are not matched greater intensity lifting seems to produce a larger energy expenditure response than lower intensity. This has potential ramifications for clinical populations and recreational exercisers as lifting at greater intensities may be a more time efficient means of producing caloric expenditure. With time being reported as a major limitation for many American’s ability to exercise regularly having a workout which takes less time, and gives similar results may be ideal.

Collectively the studies examining intensity as a modifier for the energy expenditure during recovery from resistance exercise demonstrate that intensity is positively correlated with recovery energy expenditure. This statement must be tempered by considering that lifting at greater intensities does not allow for as much volume to be lifted in a given number of sets. Therefore, though lifting with greater intensity of load seems to produce a greater energy expenditure the volume must be accounted for, or the result will be less predictable.

**Effects of Contraction Cadence and Time Under Tension**

Another important factor inherent to resistance training is the speed of contraction, which ties into time under tension. Several authors have endeavored to investigate the effect of manipulating the concentric and/or eccentric phase of contractions on the recovery energy expenditure of resistance exercise. Once again volume of work performed is an important factor, which not all researchers have accounted for, so studies with or without isovolumic protocols will be considered separately.
Equal Volumes

Mazzetti et al. (98) in 2007 investigated how “explosive” concentric contractions versus a slow velocity of concentric contraction would affect short EPOC. Their trained young male subjects performed 1 exercise with three different treatments. Two of the treatments utilized the same intensity (60% of 1RM), and entailed 4 sets of 8 repetitions. The key difference was that subjects either performed a 2 second controlled concentric motion, or an “explosive” concentric phase (moving the bar as rapidly as possible). The third treatment was also testing “explosive” repetitions, but used heavier weight (80% of 1RM), but with 6 sets of 4 repetitions. For all treatments the eccentric phase was a uniform 2 seconds. When intensity was constant the “explosive” concentric contractions resulted in 7% greater recovery energy expenditure than with 2 second concentric contractions over the duration of recovery measured. This 60% “explosive” treatment also induced a greater EPOC response than did the higher intensity treatment, but time may have been a confounder. The high intensity treatment entailed 50% more sets of work as compared to the lighter treatments, which also meant that to complete the same volume 50% more time was required. The EPOC measurements began strictly as soon as the bout of work was completed. If the same work is completed over a longer time than it is logical to conclude that perhaps recovery was already taking place during the sets. Therefore the observed response would not be as great following exercise.

In 2010 Barreto et al. (99) studied the effects of either 1 second or 2 second concentric and eccentric contractions. They used young to middle aged females who were trained, and had them perform 6 exercises (3 sets of 10 repetitions at 70% of their 1RM). Ultimately over the 60 minute EPOC period measured they did not observe a significant difference between treatments. This is likely attributable to the minor differentiation between treatments. With only a 1 second
difference between contraction phases this experiment may have lacked adequate separation between treatments, which may explain the null finding.

Later, Mazzetti et al. (100) performed another experiment similar to their previous work, which once again included “explosive” contractions. In this experiment both trained and untrained young male subjects performed 7 exercises with 3 sets of 10 repetitions. There were two variables of interest: training status and contraction speed. The three contraction speeds were 1 second concentric and eccentric, “explosive” concentric with a 2 second eccentric, or 2 seconds of concentric and eccentric. When these three treatments were undertaken by both trained and untrained subjects they observed that trained subjects had a significantly larger energy expenditure response to “explosive” contractions, while the untrained subjects did not differ regardless of the treatments.

Contrary to the previous research Scott et al. (101) found that with more time under tension the recovery energy expenditure response was greatest. Using only the bench press their young trained male subjects performed 3 sets of 5 repetitions at 70% of their 1RM. There were 3 treatment groups, 1.5 second concentric and eccentric, 4 second concentric with a 1 second eccentric, or 1 second concentric with a 4 second eccentric. Both of the longer cadences required an equal amount of recovery energy expenditure, which was greater than the shorter scheme, therefore time under tension was concluded to play an important role in the short EPOC of resistance exercise.

These studies as a whole have too much variability within the results to make draw any conclusions from them. Mazzetti et al. (98, 100) seem to observe that less time under tension, using intentionally rapid movement produces the best results, whereas Scott et al. (101) has found that more time under tension results in the greatest recovery energy expenditure. With
equal work volumes, there does not seem to be any consensus on the recovery energy expenditure of differing contraction cadences and time under tension.

**Unequal Volumes**

The three remaining studies examining contraction cadence and time under tension did not control the volume of work. The first of these was Hunter et al. (32) who in 2003 examined the effects of traditional lifting cadence versus “superslow” lifting. This study pitted traditional lifting (approximately 1 second concentric and eccentric contractions), versus “superslow” lifting (10 second concentric and 5 second eccentric contractions). These trained young men performed 10 exercises using either of the protocols (Traditional: 2 sets of 12 repetitions at 65% of their 1RM; Superslow: 1 set of 8 repetitions using 25% of their 1RM). A serious limitation of their protocol was the load intensity differential; to achieve this “superslow” training the intensity was much lower (about half of the other treatment). Ultimately they found that traditional lifting resulted in 25% more recovery energy expenditure than did “superslow”, but due to the great difference in intensities it is difficult to interpret their results. Beyond their having different volumes, intensities, rest intervals, and contraction cadences as confounders they also only measured the first 15 minutes of EPOC. This minor period of time does not allow for a full assessment of short EPOC, and therefore the results of this experiment only show the difference between these treatments during the very beginning of recovery.

Buitrago et al. (102) in 2012 examined both contraction speeds and intensity, and found a trend of faster contractions resulting in greater recovery energy expenditure. Their trained young male subjects over the course of 12 trials performed 1 set to failure with each combination of differing intensity (55%, 70%, 85%), and contraction cadence (1/1, 2/2, explosive/1, 4/4; concentric/eccentric). Their use of a variety of intensities made interpretation somewhat
difficult, and while statistically not significant, contraction schemes with less time under tension seemed to produce greater energy expenditure; as did those with higher intensity. Additionally, they only measured a 30 minute period of EPOC, but having only completed a single set of 1 exercise this may have been sufficient to assess the EPOC responses the various treatments elicited. Had more sets been completed the trends observed may have become statistically significant, but one set did not allow for sufficient differentiation between treatments.

Similarly, Mukaimoto et al. (103) in a 2012 publication found that fast contractions resulted in 20% more recovery energy expenditure than did slow contractions. Trained young male subjects performed 3 trials in a crossover design: Slow-Light: 4/4 (concentric/eccentric) with 50% of 1RM, Normal-Light: 1/1 (concentric/eccentric) with 50% of 1RM, or 1/1 (concentric/eccentric) with 80% of 1RM. All treatments consisted of 3 sets to failure on 4 exercises. In addition to faster contractions seeming to produce greater recovery energy expenditure, the high intensity treatment was nearly significantly better than the others, which concurs with the majority of previous findings. This experiment’s manipulation of both intensity and contraction speed make interpretation more difficult, but as with many others this articles appears to support the notion that rapid contractions may increase the energy expenditure during recovery from resistance exercise.

The results of these three studies are much more congruent then the studies using equal volumes. This result is somewhat expected as having less time under tension allows for more repetitions to be completed in a given set, and therefore more volume. It is difficult to posit whether the greater energy expenditure response observed in these studies were a result of the differences in the volume of work or the contraction speed schemes employed. When volume is
not controlled for contraction speed appears to be negatively correlated with recovery energy expenditure.

When comparing the studies examining contraction speed as a modifier of short EPOC there appears to be more evidence that faster contractions will result in greater recovery energy expenditure than does slow contractions. The high degree of variability within the findings which controlled volume suggests that there may not be a large effect of contraction speed on recovery energy expenditure. Conversely, studies which did not control for volume all showed time under tension to be negatively correlated with recovery energy expenditure. When time under tension is increased per repetition the number of repetitions completed per set is decreased, which in turn results in a smaller volume. Therefore, it is difficult to separate how much of the observed results are attributable to contraction cadence or volume lifted. When taken together these studies may serve as greater proof of volume’s effect on recovery energy expenditure than contraction speed or time under tension.

Effects of Rest Interval

Only three studies have been published investigating rest interval as a means of manipulating recovery energy expenditure. The first of the three is a work by Haltom et al. (6), who in 1999 had male subjects perform 8 exercises using 2 sets of 20 repetitions at 75% of their 20RM with either 20 or 60 second rest intervals. They found that the short rest interval treatment resulted in 39% greater energy expenditure over the course of recovery than did long rest interval treatment. Conversely the longer rest interval treatment resulted in greater total energy expenditure than the short. This may be attributable to the longer time required to complete the long rest interval treatment. The minor EPOC periods in between each set, and the longer
overall time of physiologic perturbation may account for the difference in total energy expenditure observed between these two conditions (84).

Following Haltom’s work Ratamess et al. (3) conducted a much more complex study which involved 5 different rest intervals ranging from very short to long (30 seconds, 1 minutes, 2 minutes, 3 minutes, 5 minutes), with two different intensities (75% or 85% of 1RM). Trained young male subjects performed the bench press for 5 sets of either 5 repetitions (85% of 1RM) or 10 repetitions (75% of 1RM). These researchers found that the EPOC response was nearly identical between all treatments, but an important detail seemingly overlooked was the volumes lifted. The volume lifted during the 5 minutes rest interval treatment was approximately 25% more than when the rest intervals were 30 seconds. Repetitions completed per set was positively correlated with rest interval, and therefore as work was performed with less rest there was naturally less total volume completed. With the shortest rest interval comprising 25% less work, and producing the same recovery energy expenditure there may be some interaction between volume and rest interval. Only the first 30 minutes of EPOC were measured, but with such a small volume completed this may have been adequate for observing the differences between these treatments.

In 2011 Farinatti et al. (4) performed an experiment using trained young male subjects, and nearly the same protocol as Ratamess et al. (3), but using 2 exercises, and only 1 or 3 minute rest intervals. They found that rest interval had no statistically significant effect on EPOC, though there was a trend toward lower rest intervals producing greater recovery energy expenditure. This may have been due to the less extreme difference between treatments. Though Haltom had the same ratio of time between short and long rest intervals and had a large difference between treatments perhaps it is not simply a matter of ratio.
Any conclusions which could be drawn from these few studies would be tenuous at best. The trend within these studies is that there may be an interaction between rest interval and recovery energy expenditure, such that less rest may result in more energy expenditure. Though if the minor EPOC periods in between sets are considered then it is logical to conclude that having less time in between sets would result in a greater post exercise period of recovery. Therefore, when considering rest interval as a variable for manipulating the recovery energy expenditure from resistance exercise the total energy expenditure may be a more valid measure; as this includes both the traditional EPOC, and the small EPOC periods after each set.

Effects of Exercise Order

Several researchers have examined how exercise order, or combinations of multiple exercise modalities affect the energy expenditure during recovery. Much of this work has been comparing circuit training (performing single sets of a number of different exercises one after the other in a series) to more traditional forms of lifting (performing all of the sets of one exercise before moving to the next).

The first to do so was Murphy et al. (104) in 1992 who found that circuit training resulted in approximately 85% more energy expenditure during recovery than did traditional training. Their untrained young male subjects performed 3 sets of 6 exercises using either 80% of their 1RM with 2 minute rest intervals for the traditional lifting treatment, or 50% of their 1RM with 30 second rest intervals for the circuit treatment. The traditional treatment required 50 minutes to complete, while the circuit was finished in 19 minutes. The traditional training treatment was performed at greater intensity while the circuit treatment entailed more volume and less rest. The lack of rest within the circuit treatment almost certainly meant that there would be a greater EPOC response than the traditional treatment. Ultimately the numerous variations between each
treatment make separating the effects of circuit versus traditional training somewhat difficult in this interpret.

Pichon et al. (105) later found similar results, as their circuit routine produced much greater energy expenditure than did the traditional training treatment. Within this study both male and female subjects performed 2 sets of 4 exercises using either 10 repetitions (70% of 1RM, traditional lifting), or 20 repetitions (50% of 1RM, circuit lifting). While circuit training did result in a greater EPOC the volume of work performed was 42% more work than the volume lifting during the traditional training treatment. When the energy expenditure of recovery was evaluated per volume of work the traditional training treatment yielded a greater result than the circuit training treatment. Additionally, their method of assessing EPOC was by use of a Douglas bag, and only the first 5 minutes of recovery were assessed. Therefore the insight which can be gained from this experiment is minimal.

Da Silva et al. (106) then compared circuit training with super setting (performing two lifts, one then the other, until all of the sets are completed before moving onto the next exercises). Their untrained young female subjects performed 3 sets of 12 repetitions (50%-55% of 1RM) on 7 exercises using either method. Therefore, unlike the previous studies the order of sets was the only variable manipulated. Ultimately, they found that circuit training and super setting did not produce statistically different EPOC responses.

Kelleher et al. (107) similarly investigated super setting but compared it to traditional training. The trained young males of their study performed 4 sets to failure using 70% of their 1RM on 7 exercises. Likewise, when set order was the only variable manipulated they found no difference in the EPOC responses to either treatment.
In a departure from simply comparing different lifting schemes Farinatti et al. (75*) in 2009 investigated how performance of the same 3 lifts in different orders would affect the energy expenditure required during recovery. The three treatments differed only in which of the three lifts was performed in what order, but the repetitions, sets, and load were all the same. Young female subjects performed each of the three treatments using sets to failure with intensity equal to their 10RM on each lift. These researchers ultimately found no differences between treatments for EPOC or total energy expenditure. Though they did observe the last exercise performed in each order combination elicited the greatest energy expenditure. This supports the work of Scott et al. (84), and further illustrates that recovery from lifting is occurring during the workout, and therefore that recovery cannot simply be thought of as the time after a workout ceases. Instead, as these studies have shown, recovery begins as soon as an individual effort ceases, and it continues as other exercises are being performed.

Lastly, Balasio et al. (108) investigated the ordering effects of performing endurance type exercise and resistance exercise in a single workout. Their subjects ran or lifted first, then did the other directly after, or performed the same amount of each but in a mixed fashion (10 min of running, then a few lifts, then back to running). The young untrained females of this study performed a total of 30 minutes of running, and 3 sets (repetitions for 30 seconds using 55% of their 1RM for each set) of 8 exercises during each trial, just in varied orders and mixtures. They found that when separated into two distinct phases of a work the order in which each modality was performed did not affect the EPOC response. When running and lifting were performed in a mixed fashion significantly greater recovery energy expenditure resulted, approximately 33% more than other treatments.
Ultimately, the comparisons of various lifting schemes have not produced a coherent result. Several studies have found that circuit training produces greater energy expenditure, but the lack of control of highly relevant variables such as time to competition and volume lifted diminishes the impact of these results. When these confounding variables have been controlled for there has not been an effect of lifting scheme. Likewise, the order one uses to complete a lifting routine does not seem to make a significant impact upon the recovery energy expenditure. Though combining resistance and endurance exercise may significantly increase the energy expenditure of a workout. With such variation between findings it is unclear how exercise order interacts with EPOC, though the majority of limited research does not support there being an interaction.

Future Research

The research characterizing EPOC as a result of resistance exercise has been successful in illustrating the increased oxygen demands, though the reasoning behind this increased demand has not been determined. More mechanistic work must be undertaken to delve deeper into the underlying causes of this phenomenon. Some indications may be found in the research comparing endurance type exercise with resistance exercise. In several of these studies there was an equal work and/or time of exercise, but completely different EPOC responses. This perhaps suggests that working muscles more intensely triggers a physiological response which then results in a greater recovery energy expenditure. Therefore examining metabolic activity during resistance exercise may aid in better understanding why there is such a drastic difference in EPOC response.

Intensity of work as a modifier of EPOC has illustrated fairly well, though optimizing EPOC response has not. Increases in intensity also decrease volume, as does changing rest
interval, or increasing time under tension. There has been little work teasing out an optimal combination of these variables to produce the greatest EPOC response. More research at the various percentages of 1RM will provide a more complete illustration of how intensity interacts with volume and energy expenditure. More volume and intensity controlled studies must be conducted to determine what contraction scheme produces a more favorable energy expenditure response. In order to discern the effects of rest interval on recovery energy expenditure work using greater differences between treatments should be undertaken. This field is of special importance due to the recent rise in popularity of exercise programs which promote large volumes of work being completed with minimal rest with the promise of enhanced weight loss potential (P-90x, Crossfit, Insanity, etc.). With only three studies to date on this topic there is little scientific evidence for or against this type of exercise.

The order of exercises within a workout is also a field which has room for growth. Though the majority of the work has shown little difference, there is some indication that performing circuit training may result in more energy expenditure than traditional training. With more research into this field drawing conclusions would be more possible. Perhaps the most intriguing finding within this field has been the observations of Blasio et al. (108), that combining elements of endurance and resistance exercise instead of separating them results in more caloric expenditure. With the most important issue in the field of kinesiology being weight gain / obesity this type of exercise may possibly provide a method of producing larger energy expenditure for a given time period with the same volume of work. This study must be replicated, and more studies like it should be conducted in the future.

Additionally, the training status of subjects needs to be better addressed. Only one work has included this as a variable of interest, and there was a significant difference. Conduction
trials with highly trained subjects, not just trained or untrained, may result in further diversity of responses, and will add to our collective understanding of the issue. Further, the vast majority of all research has been conducted using young subjects, there have only been 3 of the 38 articles examining short EPOC which have used middle aged subjects. There has not been a single study using older subjects. Researching different age groups may result in additional insights, and contribute to the general understanding how age related differences interact with resistance exercise and its EPOC responses.

**Conclusion**

Research examining the effects of resistance exercise on the short phase of EPOC is limited by vast differences in study design, controlling volumes lifted, different exercise modality, and methodological difficulties of obtaining accurate energy expenditure data. While nearly all researchers recorded the entire EPOC as a volume of Oxygen consumed, some have only recorded rates of energy expenditure following exercise, which has limited value. Researchers have defined the response of energy expenditure directly following resistance exercise fairly well. There is a rapid rise in energy expenditure, which then subsides to a more subdued elevation of energy expenditure. This can last for several hours, but there has been almost no mechanistic research undertaken in conjunction with studying EPOC as a result of resistance exercise. Therefore, the physiological processes responsible for this are not entirely understood. Methodological limitations make comparisons of resistance exercise and endurance exercise somewhat arbitrary. In general resistance exercise seems to elicit a larger EPOC, though endurance exercise requires more energy during the work; though without an accurate means of accounting for the anaerobic portion of work it is difficult to accurately compare them.
Intensity of loading seems to make a significant impact upon the recovery energy expenditure, this factor has been the most studied, and has the greatest concordance, as shown in table 2.1 (while intensity is not presented, studies using low repetitions per set typically have used higher intensity than those with greater repetitions per set). Lifting with a load closer to maximal seems to increase the recovery energy expenditure more so than lifting lighter loads. Additionally, there is evidence that moving weight very rapidly will result in a larger energy expenditure response than performing slow contractions, but there is not enough agreement between studies to conclude anything at this time. The time in between sets within a workout may impact the recovery energy expenditure, such that lower rest interval results in a larger response, but there is only minimal evidence of this. Finally, the order which exercises are performed within a workout seems to be of little importance, but circuit training and alternating resistance exercise and endurance exercise in short bouts may produce greater energy expenditure. Ultimately, more research needs to be conducted in each of these distinct subjects in order to gain a more full understanding of their contribution to EPOC.

Table 2.1: Overview of Articles. This table outlines some of the basic parameters of the research conducted within each article reviewed. R: Resistance exercise, E: Endurance exercise, RI: Rest interval, Ex: Exercise, Expl: Explosive contractions, Rec: Recreational lifting speed (~1 second concentric, ~1 second eccentric), F: Fast contractions, S: Slow contractions, Hi: High intensity, Lo: Low intensity, Med: Medium intensity, TT: Traditional training, CT: Circuit training, SS: super sets, TEE: Total energy expenditure, NS: Not significant.

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>N</th>
<th># of Ex</th>
<th>Sets</th>
<th>Reps</th>
<th>EPOC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Melby et al. (78)</td>
<td>1992</td>
<td>6</td>
<td>7</td>
<td>3</td>
<td>10</td>
<td>1 Hour, 27%</td>
</tr>
<tr>
<td>Elliot et al. (85)</td>
<td>1992</td>
<td>10</td>
<td>10</td>
<td>4</td>
<td>10</td>
<td>R &gt; E, 59%</td>
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<tr>
<td>Murphy et al. (104)</td>
<td>1992</td>
<td>10</td>
<td>?</td>
<td>3</td>
<td>?</td>
<td>20 Min, CT &gt; TT, 85%</td>
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<tr>
<td>Melby et al. (27)</td>
<td>1993</td>
<td>13</td>
<td>10</td>
<td>6 or 5</td>
<td>8 to 12</td>
<td>2 Hours, 12%</td>
</tr>
<tr>
<td>Study</td>
<td>Year</td>
<td>Hi</td>
<td>Lo</td>
<td>Time</td>
<td>Outcome</td>
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</tr>
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</tr>
<tr>
<td>Olds et al. (90)</td>
<td>1993</td>
<td>7</td>
<td>7</td>
<td>12 or 15</td>
<td>Hi &gt; Lo, 1 Hour, 10%</td>
<td></td>
</tr>
<tr>
<td>Gillette et al. (86)</td>
<td>1994</td>
<td>10</td>
<td>10</td>
<td>8 to 12</td>
<td>R &gt; E, 2 Hours, Significant, but not well reported</td>
<td></td>
</tr>
<tr>
<td>Pichon et al. (105)</td>
<td>1996</td>
<td>8</td>
<td>4</td>
<td>10 or 20</td>
<td>5 Minutes, CT &gt; TT, 7% (TEE)</td>
<td></td>
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<tr>
<td>Burleson et al. (87)</td>
<td>1998</td>
<td>15</td>
<td>8</td>
<td>30 sec of lifting</td>
<td>R &gt; E @ 30 Minutes (49%)</td>
<td></td>
</tr>
<tr>
<td>Halton et al. (6)</td>
<td>1999</td>
<td>7</td>
<td>8</td>
<td>20</td>
<td>1 Hour, Short rest &gt; Long rest, 39%</td>
<td></td>
</tr>
<tr>
<td>Osterberg et al. (79)</td>
<td>2000</td>
<td>7</td>
<td>10</td>
<td>10 to 15</td>
<td>3 Hours, 13%</td>
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<tr>
<td>Binzen et al. (80)</td>
<td>2001</td>
<td>10</td>
<td>10</td>
<td>3</td>
<td>90 Minutes, 19%</td>
<td></td>
</tr>
<tr>
<td>Schuenke et al. (81)</td>
<td>2002</td>
<td>7</td>
<td>3</td>
<td>4</td>
<td>30 Minutes, 56%</td>
<td></td>
</tr>
<tr>
<td>Melanson et al. (88)</td>
<td>2002</td>
<td>7</td>
<td>10</td>
<td>4</td>
<td>R &gt; E, 30 Minutes, 5%</td>
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<tr>
<td>Thornton et al. (91)</td>
<td>2002</td>
<td>7</td>
<td>10</td>
<td>4</td>
<td>15 or 8 Hi &gt; Lo, 2 Hours, 190%</td>
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<tr>
<td>Hunter et al. (32)</td>
<td>2003</td>
<td>7</td>
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<td>8 or 12</td>
<td>15 Minutes, TT &gt; Super Slow, 25%</td>
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<tr>
<td>Melanson et al. (2)</td>
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<td>7</td>
<td>10</td>
<td>4</td>
<td>30 Minutes, 49%</td>
<td></td>
</tr>
<tr>
<td>Braun et al. (89)</td>
<td>2005</td>
<td>8</td>
<td>8</td>
<td>15</td>
<td>R &gt; E, 1 Hour, 20%</td>
<td></td>
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<tr>
<td>Kang et al. (94)</td>
<td>2005</td>
<td>11</td>
<td>1</td>
<td>4</td>
<td>Med &gt; Lo, &gt; Hi, 40 Minutes, (???)</td>
<td></td>
</tr>
<tr>
<td>Wu et al. (92)</td>
<td>2006</td>
<td>16</td>
<td>8</td>
<td>3</td>
<td>Hi &gt; Lo, 2 Hours, 25%</td>
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<tr>
<td>Ormsbee et al. (82)</td>
<td>2007</td>
<td>8</td>
<td>6</td>
<td>3</td>
<td>45 Minutes, 11%</td>
<td></td>
</tr>
<tr>
<td>Mazzetti et al. (98)</td>
<td>2007</td>
<td>9</td>
<td>1</td>
<td>4, 4, 6</td>
<td>1 Hour, Expl(Med) &gt; S, Expl(Hi); 7% and 15%</td>
<td></td>
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<tr>
<td>Ratamess et al. (3)</td>
<td>2007</td>
<td>8</td>
<td>1</td>
<td>5</td>
<td>30 Minutes, RI NS</td>
<td></td>
</tr>
</tbody>
</table>

91
<table>
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<tr>
<th>Study</th>
<th>Year</th>
<th>Sets</th>
<th>Reps</th>
<th>Time</th>
<th>Comments</th>
</tr>
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<tr>
<td>Benton et al. (83)</td>
<td>2009</td>
<td>45</td>
<td>8</td>
<td>3</td>
<td>8 to 12, 2 Hours, 20%</td>
</tr>
<tr>
<td>Farinatti et al. (75*)</td>
<td>2009</td>
<td>10</td>
<td>3</td>
<td>3</td>
<td>10, 20 Minutes, Ex Order NS</td>
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<td>Barreto et al. (99)</td>
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<td>20</td>
<td>6</td>
<td>3</td>
<td>10, 1 Hour, F = S</td>
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<td>Da Silva et al. (106)</td>
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<td>8</td>
<td>7</td>
<td>3</td>
<td>12, 30 Minutes, SS = CT</td>
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<tr>
<td>Kelleher et al. (107)</td>
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<td>10</td>
<td>7</td>
<td>4</td>
<td>to failure, 1 Hour, SS = TT</td>
</tr>
<tr>
<td>Thornton et al. (93)</td>
<td>2011</td>
<td>10</td>
<td>9</td>
<td>3</td>
<td>15 or 8, Hi &gt; Lo, 2 Hours, 49%</td>
</tr>
<tr>
<td>Almeida et al. (95)</td>
<td>2011</td>
<td>9</td>
<td>2</td>
<td>3</td>
<td>6-8 or 15-20, 10 Minutes, Hi = Lo</td>
</tr>
<tr>
<td>Scott et al. (96)</td>
<td>2011</td>
<td>10</td>
<td>1</td>
<td>2</td>
<td>to failure, Time not reported, NS</td>
</tr>
<tr>
<td>Scott et al. (97)</td>
<td>2011</td>
<td>13</td>
<td>1</td>
<td>1</td>
<td>to failure, Regression derived; Sets to failure resulted in steeper slope</td>
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<td>Mazzetti et al. (100)</td>
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<td>14</td>
<td>7</td>
<td>3</td>
<td>6 to 8, 2 Hours, Expl &gt; Rec, S, Not well reported</td>
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<td>10</td>
<td>2</td>
<td>5</td>
<td>10, 90 Minutes, RI NS</td>
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<tr>
<td>Scott et al. (84)</td>
<td>2012</td>
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<td>1</td>
<td>3</td>
<td>5, Time not reported, More time under tension &gt; less, 30%</td>
</tr>
<tr>
<td>Scott et al. (101)</td>
<td>2012</td>
<td>10</td>
<td>1</td>
<td>3</td>
<td>5, Time not reported, More time under tension &gt; less, 30%</td>
</tr>
<tr>
<td>Buitrago et al. (102)</td>
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<td>10</td>
<td>1</td>
<td>1</td>
<td>to failure, 30 Minutes, NS</td>
</tr>
<tr>
<td>Mukaimoto et al. (103)</td>
<td>2012</td>
<td>11</td>
<td>4</td>
<td>3</td>
<td>to failure, 3 Hours, NS</td>
</tr>
<tr>
<td>Blasio et al. (108)</td>
<td>2012</td>
<td>13</td>
<td>8</td>
<td>3</td>
<td>reps for 30 sec, 30 Minutes, Mixed &gt; Separate, 33%</td>
</tr>
</tbody>
</table>
SHORT PHASE OF RECOVERY: SUBSTRATE UTILIZATION AND RESISTANCE EXERCISE

Substrate utilization during exercise is fairly well understood. The cross-over concept succinctly illustrates how substrates are used as energy demand increases during exercise (75). Glycolysis tends to predominate as intensity of work increases, while oxidative processes primarily employed when intensity is low. While this idea is well understood, post exercise substrate utilization is not as clear; specifically post resistance exercise. The most common means of examining the usage of substrates during and after exercise is through indirect calorimetry. This process involves measuring the expired gases of a subject; substrate usage can be derived based upon the concentrations of oxygen and carbon dioxide. While this process is well established, and recognized as a valid means of determining substrate utilization, anaerobic work may confound post exercise observations. The process of bicarbonate buffer replenishment requires carbon dioxide, which is a chief component of the respiratory exchange ratio (RER). When aerobic metabolism produces carbon dioxide as a byproduct and it is not exhaled, the observed ratio of oxygen to carbon dioxide, or respiratory exchange ratio, may not be valid (109). This bicarbonate buffer replenishment process can cause respiratory exchange ratio to drop to levels which might under normal circumstances be indicative of nearly pure lipid oxidation, ketone metabolism, or readings not consistent with normal substrate utilization. Therefore, the measurement of respiratory exchange ratio directly following resistance exercise (a highly anaerobic activity) must be approached with care as these values are not necessarily representative of substrate utilization. Additionally, immediately following work the steep increase in respiratory exchange ratio may not be indicative of substrate utilization either. During exercise acidosis may occur, which then results in carbonic anhydrase catalyzing the breakdown of bicarbonate into carbon dioxide and water. This excess carbon dioxide is then
expired, which may explain respiratory exchange ratio values of greater than 1. Thus, the respiratory exchange ratio values, which are typically used to assess substrate utilization may be misleading if the acid-base controlling processes are not accounted for.

**Characterization of RER Response to Resistance Exercise**

Following most exercise there is a significant increase in respiratory exchange ratio which is followed by a gradual decrease back to resting values. With anaerobic work this decrease may drop below that of initial values, this generally accepted to be a result of bicarbonate buffer replenishment, but it is possible that fat oxidation is increased. Regardless of the cause there have been a number of researchers who have characterized the respiratory exchange ratio response to resistance exercise.

Melby et al. (78) in 1992 first examined how resistance exercise influences substrate utilization. Their trained young male subjects performed 3 sets of 7 exercises (10 repetitions using 70% of their 1RM). Indirect calorimetry was used for 1 hour post exercise, and they found respiratory exchange ratio was over 1.0, then after the first 6 minutes it dropped to 0.7, which was 0.15 lower than baseline. These results were attributed only to bicarbonate buffer replenishment. The next year this group performed another experiment using approximately the same lifting protocol, but with twice the sets (27). The results were roughly the same, though following this experiment respiratory exchange ratio dropped below 0.65 following work, and remained depressed for over an hour. This difference in response may have been the result of the greater work volume, and therefore more bicarbonate buffer replenishment, or there may have been a shift toward fat oxidation as this long workout is likely to have taxed glycogen supplies. No mechanisms beyond breath gases were assessed, and therefore the underlying processes responsible for the observed drop in respiratory exchange ratio are not known.
Osterberg et al. (79) found largely the same response in 2000 when they examined how resistance exercise affected respiratory exchange ratio in trained young females. They used a work protocol fairly similar to Melby et al. (27), and their subjects had 3 hours of EPOC recorded. Likewise they noted that respiratory exchange ratio increased, then rapidly dropped to under 0.7 within the first 30 minutes. Additionally, they noted that respiratory exchange ratio did not return to baseline values until 2 hours post exercise. They did not calculate any substrate utilization values because they suspected bicarbonate buffer replenishment to be the chief contributor to the observed results.

In 2001 Binzen et al. (80) measured respiratory exchange ratio for the first 120 minutes following a resistance exercise bout. Their trained young female subjects performed 10 lifts for 3 sets of 10 repetitions using 70% of their 1RM. Much like prior researchers they observed respiratory exchange ratio to be significantly lower than baseline values over the entire time measured (baseline: 0.85, post exercise: 0.75). This finding further confirmed what had been observed by other researchers, but blood lactate measurements were additionally obtained pre and post exercise within this study. During recovery blood lactate was significantly elevated for 60 minutes. Blood lactate was back to resting values for an hour while respiratory exchange ratio remained depressed for two. This may suggest that there is an increase in fat metabolism, as the process of bicarbonate replenishment should be completed once lactate levels have reached resting values. So, while blood lactate was not directly assessed this study does offer some evidence that bicarbonate buffering replenishment may not be the only process underlying the reduction in respiratory exchange ratio observed following resistance exercise.

Later, Schuenke et al. (81) examined the effects of a much smaller work volume. Their resistance exercise protocol only included 3 exercises for 4 sets of 8-12 repetitions using 70-80%
of their 1RM. The young trained male subjects of this experiment experienced a drop in respiratory exchange ratio like those of past studies, but to a lesser degree (from 0.89 to 0.79). Many of the prior studies have observed reductions in respiratory exchange ratio of 0.15 or more, but in this study it was only 0.1. While this is somewhat of a small difference perhaps having performed significantly less work the compensatory response in respiratory exchange ratio was smaller. This could either represent less bicarbonate buffer replenishment, or less of a shift to fat oxidation, as less work would likely have not taxed glycogen stores as greatly.

Melanson et al. (2) in 2005 was the first to observe no difference in respiratory exchange ratio following resistance exercise. Their untrained middle aged females underwent a 24 hour stay in a full room indirect calorimetry chamber. They performed 10 lifts using 70% of their 1RM (4 sets of 10 repetitions). Over the course of the 30 minute post exercise period recorded they reported that respiratory exchange ratio was not significantly different from that of baseline values. While initially this may be somewhat perplexing, as all studies before have found respiratory exchange ratio to decrease significantly following resistance exercise, these results may simply be the result of misrepresentation. The typical response based upon past studies of resistance exercise on respiratory exchange ratio is a large increase, followed by a significant decrease. This switch from high values to low usually occurs with the first 10-15 minutes following resistance exercise. Therefore, if the higher than baseline, and lower than baseline values are averaged over only a 30 minute time span the result may be not significantly different from baseline. Additionally, this was the first study examining the effects of resistance exercise on respiratory exchange ratio to include middle aged untrained subjects, so this may have been a factor as well. Perhaps these subjects being less trained increased their reliance on glycolysis/glycogenolysis following exercise, which prolonged the period of highly elevated
respiratory exchange ratio values. Had more than the first 30 minutes of EPOC been reported that perhaps more insight could be gained from this study.

In 2007 Ormsbee et al. (82) conducted a study which not only measured respiratory exchange ratio, but also Dialysate glycerol concentrations (a marker lipid metabolism) during and after resistance exercise. Their trained young male subjects performed 6 lifts using 3 sets of 10 repetitions at 70% of their 1RM. Prior to exercise a micro dialysis device was inserted into their subcutaneous abdominal fat; this device remained there measuring Dialysate glycerol concentrations throughout the bout of exercise, and during recovery. While respiratory exchange ratio fluctuated much the same as in previous studies (large increase followed by a depressed value) Dialysate glycerol concentrations were significantly increased following resistance exercise. The observed Dialysate glycerol readings showed an approximate 75% increase in fat metabolism. The results of this study are compelling evidence that bicarbonate buffer may not be the only process underlying the depression of respiratory exchange ratio following resistance exercise.

Lastly, Scott et al. (84) examined the dynamics of resistance exercise recovery not only from a post exercise perspective, but from a during workout perspective. Their young trained male subjects performed 3 sets of 5 repetitions at 70% of their 1RM on bench press. While the post exercise recovery was much the same as previous works (though of smaller duration), the between set variations were also observed. Following each set respiratory exchange ratio spiked to well over 1.0 (as high as 1.41 was recorded), then rapidly decreased to baseline or below. While this pattern has been observed by many other researchers, the results of this study suggest that recovery from resistance exercise cannot simply be assessed as the time following the last
repetition in workout. The between set recovery during a workout must be assessed in order to fully document the recovery from resistance exercise.

Ultimately, research examining the substrate utilization patterns acutely following resistance exercise has limitations. The bicarbonate buffer replenishment process following anaerobic metabolism confounds assessment of substrate utilization, as it alters respiratory exchange ratio. While the majority of researchers do not consider post resistance exercise respiratory exchange ratio depression to be indicative of increase lipid oxidation there is evidence to the contrary. The extent to which changes in substrate utilization or bicarbonate buffer replenishment are responsible for this phenomenon is unclear. What has been fairly well established is that following resistance exercise respiratory exchange ratio increases drastically, and within minutes it has a tendency to decrease below that of resting levels.

Resistance Exercise versus Endurance Exercise

Gillette et al. (86) was the first to compare endurance exercise and resistance exercise with respect to respiratory exchange ratio acutely post exercise. Their study consisted of trained young males performing either a bout of endurance exercise (cycling for as long as was necessary to match the calories burned during the resistance exercise protocol at a work rate of 50% of their VO2Max) or resistance exercise (10 exercises, 5 sets, 8-12 repetitions using 70% of their 1RM). The end result of this comparison of exercise modality was that resistance exercise resulted in a significantly lower respiratory exchange ratio than did the endurance exercise protocol (resistance exercise: 0.7, endurance exercise: 0.85). While respiratory exchange ratio is typically indicative of substrate utilization, in this instance it is logical to conclude that bicarbonate buffer replenishment may have played a significant role in the observed results. A major flaw of this work was the lack of accounting for anaerobic work performed. While the
researchers attempted to create an isocaloric experiment they did not take into account the anaerobic work performed, and thus the energy expenditure of the endurance exercise protocol was likely less than that of the resistance exercise. Additionally, as resistance exercise involves much more glycolytic activity metabolism than does working at 50% of one’s VO$_{2\text{Max}}$, the bicarbonate buffer system would require much more replenishment, which is the likely mechanism behind the difference in respiratory exchange ratio observed between these treatments.

In 2002 Melanson et al. (88) likewise compared cycling with resistance exercise, but on the basis of intensity. They pitted cycling at 70% of VO$_{2\text{Max}}$ with lifting at 70% of 1RM. The trained young male subjects of this study cycled for 50 minutes during the endurance exercise protocol, and performed 4 sets of 10 repetitions on 10 exercises for the resistance exercise protocol. Through the use of whole room calorimetry no difference was observed between the two protocols with respect to respiratory exchange ratio over the 30 minute post exercise recovery period reported. This study may support the theory that the immediate drop in respiratory exchange ratio observed post exercise may be linked to bicarbonate buffer replenishment as both treatments in this experiment were of sufficient intensity to involve significant anaerobic metabolism.

Later Braun et al. (89) compared circuit weight training to endurance exercise, and found resistance exercise to result in a greater reduction of respiratory exchange ratio than did the endurance exercise treatment. Using an isocaloric, and isotemporal protocol subjects either lifted or exercised on a treadmill. The lifting treatment consisted of 8 exercises, using 3 sets of 15 repetitions at 65% of the young untrained females’ 1RM. The endurance exercise treatment was simply matched for time, and speed was set to consume an equivalent quantity of energy as that
consumed during the resistance exercise treatment. This study failed to consider the anaerobic portion of the energy expenditure, and like Gillette et al. (27) likely under estimated the caloric demand of the resistance exercise treatment. Ultimately, over the course of the hour recorded post exercise resistance exercise resulted in a 0.05 lower respiratory exchange ratio than did the endurance exercise treatment (0.75 and 0.80 respectively).

With only 3 studies having compared these different exercise modalities our knowledge of this topic is limited. With the significant differences in methodology, results, and subject characteristics between these studies it is difficult to interpret how respiratory exchange ratio post exercise differs between resistance exercise and endurance exercise. The amount of anaerobic work does seem to be inversely related with respiratory exchange ratio over the course of short term recovery. The traditionally accepted view is that this respiratory exchange ratio is reduced commensurate to the amount of bicarbonate buffer activity during exercise. While it is also possible that lipid oxidation could contribute in the reduced respiratory exchange ratio observed (as is typically the case with low respiratory exchange ratio) few studies to date have measured more than breath gases, and therefore the mechanism behind this respiratory exchange ratio remain unclear.

**Effects of Intensity**

In 1993 Olds et al. (90) examined how lifting with differing intensities would influence respiratory exchange ratio response to resistance exercise. Young active male subjects performed 2 sets of 7 exercises using either a low or high intensity load (60% or 75% of 1RM respectively). While this experiment did not account for volume of work performed, the respiratory exchange ratio was not significantly different between these two treatments over the hour of recovery recorded. The respiratory exchange ratio recorded had very similar
characteristics as observed by other researchers (rapid increase, followed by a reduction below baseline within minutes). The lack of difference between respiratory exchange ratio responses may have been a result of the treatments being too similar; the intensity may not have been distinct enough to result in dissimilar respiratory exchange ratios.

Thornton et al. (91) later conducted a similar experiment, but with greater differentiation between intensity levels. The young females of this study performed 2 sets of 9 exercises. They lifted either 45% of their 8RM (15 repetitions), or 85% of their 8RM (8 repetitions). Much like Olds et al. (90) they likewise did not find any significant differences in respiratory exchange ratio post exercise. This group in 2011 performed almost the same experiment, but with 3 sets instead of 2, and still found no difference between treatments for respiratory exchange ratio response (93). Like other researchers respiratory exchange ratio was significantly depressed below that of baseline values. Within both of these studies blood lactate was assessed, and within both studies neither treatment elicited significantly differences.

Wu et al. (92) in 2006 also examined for intensity influenced respiratory exchange ratio following resistance exercise. Using trained young males they were unable to find differences between the high and low intensity treatments (8 exercises, 3 sets, either 10 repetitions at 75% or 15 repetitions at 50% of 1RM). This work supports all of the previous studies which have observed no difference between respiratory exchange ratio responses following resistance exercise of differing intensities.

In 2007 Ratamess et al. (3) investigated not only how intensity influenced respiratory exchange ratio, but also rest interval. The trained young male subjects of this experiment performed 5 sets of bench press using every permutation of the 5 rest intervals (30 seconds, 1 minute, 2 minutes, 3 minutes, or 5 minutes) and 2 intensity levels (75% or 85% of 1RM).
Ultimately following these 10 trials no differences in respiratory exchange ratio were observed between treatments acutely following exercise. Respiratory exchange ratio responded much as other researchers have detailed, but with only 5 sets of work the recovery was of shorter duration. Blood lactate was assessed before and throughout the recovery from these bouts of resistance exercise. There were no differences between treatments reported, but it was highly related to fatigue rate and respiratory exchange ratio. With such a short period of recovery (from a small bout of work) these implications of their blood lactate findings are limited.

Lastly, Almeida et al. (95) performed another experiment using differing intensities to determine how respiratory exchange ratio following exercise would respond. Their untrained young male subjects performed 3 sets of 2 exercises using either a high intensity (6-8 repetitions at 80% of 1 RM) or a low intensity (15-20 repetitions at 55% of 1RM). The results of this experiment were nearly identical with those previously reviewed: no difference between treatments. While no significant difference was observed it should be noted that only the first 10 minutes of recovery were recorded, and therefore the decrease in respiratory exchange ratio typically observed was only in its beginning stages. Had more of the recovery been recorded there may have been differences, but this is unlikely due to past precedents.

All studies using different intensities have resulted in the same finding: No significant differences between treatments. With many studies using unequal volumes it is surprising that respiratory exchange ratio would not be affected. If the bicarbonate buffer system is wholly responsible for the commonly observed depression of respiratory exchange ratio following resistance exercise than one might expect work of differing intensity to influence the magnitude of this depression. Instead there has not been a single study which has found any difference, at
any time point between resistance exercise of various intensity levels. This lack of finding may suggest that there is more at work than simply acid-base buffering processes.

Future Research

With the respiratory exchange ratio response to resistance exercise having been fairly well defined the meaning of this change needs consideration. While the majority of researchers attribute the change in respiratory exchange ratio simply to bicarbonate buffer replenishment this mechanism has not been tested in conjunction with EPOC measurements following resistance exercise. Bicarbonate buffer replenishment does logically make sense, but there is evidence that fat metabolism post exercise is positively influenced as a result of resistance exercise. More research aimed at determining how much bicarbonate buffer replenishment contributes to the observed depression of respiratory exchange ratio following resistance exercise must be undertaken; without this knowledge determining substrate utilization post resistance exercise is not feasible. Measurements of blood pH have yet to be directly assessed which would aid greatly in determining whether the decrease in respiratory exchange ratio is due to fat metabolism or bicarbonate store replenishment.

Should bicarbonate buffer activity be found to play the predominant role in respiratory exchange ratio depression following resistance exercise a timeline of how long this process requires should be developed. Without a clear understanding of how long the process of bicarbonate buffer replenishment takes substrate utilization acutely post exercise cannot be determined.

Conclusion

Ultimately, the research examining substrate utilization for the first few hours post resistance exercise has been unsuccessful at clearly discovering what substrates are used.
Bicarbonate buffer activity is a confounding factor which has lead the majority of researchers to simply posit that post resistance exercise substrate utilization cannot be analyzed using breath gases. Carbon dioxide is thought to be lost to replenishment of the bicarbonate buffers, and therefore any post exercise depression of respiratory exchange ratio is not typically attributed to changes in substrate utilization. A major limitation of work in this field has been the lack of blood pH assessment. Only 4 studies have measured blood lactate, which is an indirect assessment of pH. Nearly all researchers have claimed bicarbonate buffer replenishment to be the major player underlying the change in respiratory exchange ratio post resistance exercise, but none have taken serious steps to actually test this theory.

Studies examining post resistance exercise substrate utilization have demonstrated that following resistance exercise respiratory exchange ratio tends to increase rapidly, then within minutes subside, often to below initial baseline values. Additionally, greater workloads (more volume lifted) tend to result in a more prolonged depression of respiratory exchange ratio, as is illustrated in table 3.1. Work examining how resistance exercise and endurance exercise modes of activity compare in terms of post exercise respiratory exchange ratio have had mixed results, but their findings seem to indicate that more anaerobic metabolism during work corresponds to lower respiratory exchange ratio values following work. Research investigating intensity as a modifier of respiratory exchange ratio response following resistance exercise has been unsuccessful at determining any relationship.

In conclusion, though many studies have found respiratory exchange ratio to change following resistance exercise, the observed responses have been attributed to bicarbonate buffer replenishment, and not substrate utilization. While there is evidence that lipid oxidation is up regulated following resistance exercise (82), the majority of researchers consider bicarbonate
buffer to be the major player underlying this phenomenon. Without more mechanistic work, or better means of determining post exercise substrate utilization, the substrates used following resistance exercise will not be clearly understood.

Table 3.1: Summary of Articles. This table outlines some of the basic parameters of the research conducted within each article reviewed. N: Number of subjects, # of Ex: Number of exercises, Dep: Depressed RER, EE: Post Endurance exercise, RE: Post resistance exercise, C: Control, NS: Not significant, DG: Dialysate Glycerol.

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>N</th>
<th># of Ex</th>
<th>Sets</th>
<th>Repetitions</th>
<th>RER Response</th>
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<tr>
<td>Melby et al. (78)</td>
<td>1992</td>
<td>6</td>
<td>7</td>
<td>3</td>
<td>10</td>
<td>1 Hours Dep, RE: 0.7</td>
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<td>1993</td>
<td>13</td>
<td>10</td>
<td>6 or 5</td>
<td>8 to 12</td>
<td>2 Hours Dep, RE: 0.65</td>
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<tr>
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<td>1993</td>
<td>7</td>
<td>7</td>
<td>2</td>
<td>12 or 15</td>
<td>1 Hours Dep, RE: 0.7</td>
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<tr>
<td>Gillette et al. (86)</td>
<td>1994</td>
<td>10</td>
<td>10</td>
<td>5</td>
<td>8 to 12</td>
<td>15 Minutes Dep, EE: 0.85, RE: 0.7</td>
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<td>2000</td>
<td>7</td>
<td>10</td>
<td>5</td>
<td>10 to 15</td>
<td>2 Hours Dep, RE: 0.7</td>
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<tr>
<td>Binzen et al. (80)</td>
<td>2001</td>
<td>10</td>
<td>10</td>
<td>3</td>
<td>10</td>
<td>2 Hours Dep, 0.75</td>
</tr>
<tr>
<td>Schuenke et al. (81)</td>
<td>2002</td>
<td>7</td>
<td>3</td>
<td>4</td>
<td>8 to 12</td>
<td>30 Minutes Dep, 0.79</td>
</tr>
<tr>
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<td>2002</td>
<td>14</td>
<td>9</td>
<td>2</td>
<td>15 or 8</td>
<td>2 Hours Dep, NS (45% or 85% of 8RM)</td>
</tr>
<tr>
<td>Melanson et al. (88)</td>
<td>2002</td>
<td>10</td>
<td>10</td>
<td>4</td>
<td>10</td>
<td>30 Minutes Dep, NS (EE vs. RE)</td>
</tr>
<tr>
<td>Melanson et al. (2)</td>
<td>2005</td>
<td>7</td>
<td>10</td>
<td>4</td>
<td>10</td>
<td>30 Minutes, NS (RE vs. C)</td>
</tr>
<tr>
<td>Braun et al. (89)</td>
<td>2005</td>
<td>8</td>
<td>8</td>
<td>3</td>
<td>15</td>
<td>1 Hours Dep, RE &lt; EE (0.75 vs. 0.8)</td>
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<tr>
<td>Wu et al. (92)</td>
<td>2006</td>
<td>16</td>
<td>8</td>
<td>3</td>
<td>10 or 15</td>
<td>2 Hours Dep, NS (75% or 50%)</td>
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<tr>
<td>Ormsbee et al. (82)</td>
<td>2007</td>
<td>8</td>
<td>6</td>
<td>3</td>
<td>10</td>
<td>45 Minutes Dep, RE: 0.71, DG: +75%</td>
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<tr>
<td>Ratamess et al. (3)</td>
<td>2007</td>
<td>8</td>
<td>1</td>
<td>5</td>
<td>10 or 5</td>
<td>30 Minutes Dep, NS (75% vs. 85%)</td>
</tr>
<tr>
<td>Thornton et al. (93)</td>
<td>2011</td>
<td>10</td>
<td>9</td>
<td>3</td>
<td>15 or 8</td>
<td>2 Hours Dep, NS (45% or 85% of 8RM)</td>
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<tr>
<td>Almeida et al. (95)</td>
<td>2011</td>
<td>9</td>
<td>2</td>
<td>3</td>
<td>6-8 or 15-20</td>
<td>10 Minutes Dep, NS (80% vs. 55%)</td>
</tr>
<tr>
<td>Scott et al. (84)</td>
<td>2012</td>
<td>10</td>
<td>1</td>
<td>3</td>
<td>5</td>
<td>Time Not Reported, RE: 0.8</td>
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PROLONGED PHASE OF RECOVERY: ENERGY EXPENDITURE AND RESISTANCE EXERCISE

Resistance exercise has often been found to elicit increased energy expenditure for much longer than the initial short phase of EPOC; this prolonged energy expenditure is referred to as the prolonged phase of EPOC. The acute effects of resistance exercise on energy expenditure during this prolonged EPOC period are largely understudied. With approximately 15 articles published the results are understandably mixed, and vary to a great extent. Studies have been divided by research question, and will be compared with studies with similar intents.

Length and Magnitude of Prolonged EPOC

In 1994 Gillette et al. (86) compared the prolonged effects of either cycling or resistance exercise on young active males, and found a 4% increase in energy expenditure for 14.5 hours following the resistance exercise bout. The resistance training protocol consisted of 5 sets of 8 to 12 repetitions using approximately 70% of the 1RM on 10 separate exercises which comprised a full body workout. The cycling treatment consisted of working at approximately 50% of their VO2max for as long as it took to consume the same volume of oxygen as the resistance training protocol. This means that this study was not randomized, and that the resistance training may have interacted with the cycling treatment without taking this interaction into account. Additionally, while the volume of oxygen consumed was measure, the anaerobic processes, which are typically predominant in resistance exercise, were not measured. It is therefore likely that the cycling condition did not perform an equivocal bout of exercise though the calories burned were not reported to be significantly different. Further, with only 10 subjects, the cycling results were not reported due to outliers who made the data too variable to analyze.

Melby et al. (27) in 1993 conducted two experiments seeking to ascertain the energy expenditure response of resistance exercise 16 hours post exercise. This study used a resistance
training program which was nearly identical to that of Gillette et al. (86) and also had young trained males as subjects. Their findings were understandably similar as well, though of slightly greater magnitude: 9% and 5% increase in energy expenditure respectively. The discrepancy in magnitude of energy expenditure may have to do with the subject’s characteristics. The participants in study 1 were on average not as strong, and did not weigh as much. This may suggest that they were not very well trained, and by comparison the subjects in study 2 were larger and stronger. If there were difference in training status than perhaps this may suggest that an equivocal bout of work may have differing effects on prolonged EPOC based on subject characteristics.

Later Osterberg et al. (79) found a nearly identical 4% increase when examining the prolonged energy expenditure of resistance exercise 16 hours post exercise. This study examined the effects of resistance exercise on trained young female subjects. The resistance exercise treatment was very similar to the previous two studies discussed, but the repetitions within each set were slightly greater (10-15 instead of 8-12). Though the results of this study were quite consistent with those previously reviewed, there were some possible confounders. First, this experiment only required their subjects to have not exercised for 36 hours prior to the experiment. This potential oversight could suggest that subjects may have already been experiencing a prolonged EPOC during their initial baseline resting metabolic rate measurements. Additionally, menses was not taken into consideration, and therefore this could have also significantly affected their energy expenditure, as measurements were taken over the course of several days.

Melanson et al. (88, 2) in 2002 and 2005 conducted studies of total energy expenditure in which subjects had their energy expenditure measured for a period of 24 hours. During this time
they performed a resistance exercise bout, and had their responses measured. The first of the studies used young active males, while the other used untrained middle aged females. Though using different populations they each had nearly identical resistance training treatments, and had a similar 20% increase in total energy expenditure as a result. While total energy expenditure was greatly increased much of this energy expenditure was accounted for by the resistance exercise and the short phase of EPOC. The prolonged EPOC accounted for the approximate 4-5% of the total energy expenditure increase. These were the only studies to actually measure the volume of oxygen consumed over the prolonged period of recovery, which in these studies was 17.5 hours in duration.

In stark contrast Hunter et al. (5) did not find any elevation of their trained young female subjects’ energy expenditure 19 hours following resistance exercise. While this study had a much greater number of subjects the lack of prolonged EPOC may be linked to the conditions of their experiment. This experiment was performed at the end of a long term resistance training study, during which subjects lifted at the same intensity and volume for months. Subjects were then tested to observe any potential prolonged EPOC response to this same bout of work. Perhaps performing the same workout repetitively dampened the metabolic effect. Additionally, there was much less volume of work performed during this experiment when compared with the previous studies examined. This study only used 2 sets of 8 exercises, while most studies have been using at least 4 sets of 10 exercises. Though no effect was observed, this may indicate that performing the same workout repetitively will eventually result in less prolonged metabolic effect.

Jamurtas et al. (26) in 2004 observed a 9% increase in energy expenditure 24 hours following resistance exercise using the same resistance exercise protocol employed by Melby et
al. (27). These findings are nearly identical to those of Melby, who also used young trained males, but within this study energy expenditure was elevated up to 24 hours post exercise. Additionally, Jamurtas measured creatine kinase, thyroid hormone levels (T₃ & T₄), and collected self-report measures of muscle soreness. Also an endurance training treatment was compared against the resistance exercise protocol. The endurance training protocol resulted in an EPOC of greater duration, but did not result in muscle soreness. Thyroid was unaffected by resistance exercise, but both creatine kinase and soreness were increased. Both of these measures of muscle damage were elevated for up to 24 hours post exercise, which correlated well with the increase in resting energy expenditure. The increase in both creatine kinase and muscle soreness as a result of resistance exercise may suggest that protein synthesis may have a large role in the prolonged EPOC period following resistance exercise. This is further supported by the lack of muscle soreness observed as a result of the endurance training treatment. So while, both endurance training and resistance exercise evoked large prolonged EPOC responses, the mechanisms underlying each are likely different in nature.

Schuenke et al. (81) found similarly that energy expenditure stayed elevated far beyond the initial bout of resistance exercise. With trained young male subjects performing 3 full body exercises for 4 sets of 8 to 12 repetitions at 70%-80% of their 1RM they found that EPOC lasted for up to 38 hours. The magnitude of the increased energy expenditure was not well reported, only that is was significantly greater than baseline.

Williamson et al. (110) observed that EPOC did not end until 48 hours post exercise. The energy expenditure increase during EPOC was variable, starting at a greater amount and eventually subsiding out to a minor 3% observed 2 days following exercise. Their untrained older male subjects lifted at 75% of their 3RM for 18 sets of 10 repetitions on a combination of
leg extensions and bench press. Aside from only performing 2 exercises this group only had their subjects perform the concentric phase of each lift.

These studies utilized methods which were largely different, and therefore have a great degree of variation in results: for example some researchers have observed elevated energy expenditure as long as 48 hours or more following resistance exercise, while other have observed EPOC to end within 24 hours. This variation is likely due in large part to the vast differences in resistance exercise protocols employed. The length of exercise bout has been variable from lasting less than 30 minutes, to taking over 90 minutes. The intensities used have ranged from very mild loads with many repetitions to lifting very heavy loads for only a few repetitions. These fundamental differences are likely at the heart of the drastic variations in both magnitude and duration of prolonged EPOC. In general, it appears that within the first 24 hours following resistance exercise, energy expenditure may be elevated between 4-18%, and over the next 24 hours these values either return to baseline or remain slightly elevated.

**Effects of Rest Interval and Intensity**

Fatouros et al. (23) in 2009 investigated the effects of three different loading schemes using various intensities. Their untrained older male subjects performed 3 sets of 10 exercises using either 80% (heavy), 60% (medium), or 45% (light) of their 1RM. Repetitions were matched with intensity such that the heavy treatment only performed 7 reps, while the medium performed 10, and the light 14. Therefore the low and medium treatments were roughly matched for volume, while the high condition entailed the least amount of work performed. Ultimately they observed the heavy treatment resulted in the greatest increase in energy expenditure during the prolonged phase of recovery. Additionally, while the light and medium intensity groups experienced an increase in energy expenditure for 48 hours, the heavy condition elicited a
significant increase in energy expenditure for up to 72 hours post exercise. Cortisol was measured throughout this study, and interestingly the heavy treatment evoked the smallest response in cortisol, which has been known to increase metabolic rate (111). The contradiction between mechanism and observed response may suggest that cortisol levels are not a major player in the increased energy expenditure observed following resistance exercise.

Paoli et al. (25) have recently conducted an investigation using young trained males to test the effects of rest interval and intensity on energy expenditure during the prolonged phase of EPOC. This group compared a bout of high intensity interval training (5 exercises, 3 sets, 3-6 repetitions using approximately 85% of their 1RM) to more traditional lifting (8 exercises, 4 sets, 8-12 repetitions using approximately 70% of their 1RM). They found that although the traditional training treatment entailed a significantly larger volume of work, the high intensity interval training treatment had an approximately 18% greater energy expenditure 22 hours after exercise.

Petrofsy et al. (112) also examined how a short vigorous bout of work would affect prolonged recovery energy expenditure. In this study active young male and female subjects exercised for only 6 minutes, performing bodyweight exercises continuously. The result of this short duration, high volume workout was a 9% increase in energy expenditure for as long as 24 hours post exercise. Though no other treatments were administered to serve as a basis for comparison the results are fairly impressive. A potential limiting factor was their unorthodox resting metabolic rate assessment procedures. Resting metabolic rate was only assessed for 5 minutes, without a period of familiarization during each assessment. This short time of assessment could potentially result in fluctuations in gas exchange simply due to the subjects’ initial reaction to having a mask placed over their mouth and nose. Their resting metabolic rate
assessment methods may be problematic, but the potential implications of a 6 minute bout of exercise provoking such a large metabolic response make this kind of research highly relevant to many individuals whose largest barrier to exercise is lack of time.

Heden et al. (113) in 2011 investigated how volume of work affects energy expenditure during prolonged EPOC. They compared full body workouts using either one or three sets. This experiment used untrained young male subjects, and additionally compared muscle soreness with energy expenditure. Muscle soreness was determined using a scale based upon their perceived muscle soreness. Ultimately they observed that although one treatment entailed 3 times the work of the other that they were not statistically different with respect to prolonged EPOC. It should also be noted that an increase in energy expenditure was observed 72 hours post exercise (approximately 5% above baseline). Muscle soreness was correlated highly with the increase in energy expenditure, but while the two treatments did not differ in energy expenditure, only the 3 set treatment produced muscle soreness for 48 hours (they both produced muscle soreness up to 24 hours). These finding suggest that intensity is perhaps of greater importance than volume for producing prolonged energy expenditure.

In stark contrast to these studies Hunter et al. (32) has found no difference in energy expenditure 22 hours after either a moderate intensity or low intensity bout of lifting. This work compared super slow lifting (1 set, 8-12 repetitions, 15 seconds per rep using 25% of 1RM) to tradition lifting (2 sets, 8-12 repetitions, 2 seconds per rep using 65%) using trained young males.

The study of rest interval and intensity as modifiers of prolonged EPOC has such a limited quantity of research that the conclusions drawn must be approached with skepticism. Though this research is limited, the studies within this area have generally observed that rest
interval/time to competition may be negatively correlated with recovery energy expenditure. Additionally, intensity may be positively correlated with prolonged energy expenditure. Volume of work does not seem to play as much of a role as intensity for evoking a prolonged EPOC response, though this is preliminary at best. Overall, the factors inherent to resistance exercise (intensity, volume, rest interval, etc.) do seem to have an effect on the prolonged energy expenditure response, though the research investigating their contributions is quite limited.

Effects of Training Status

Dolezal et al. (7) in 2000 studied the training status of young male subjects, as well as creatine kinase, and muscle soreness as a variable of interest while investigating the prolonged effects of resistance exercise on energy expenditure. This study used long eccentric phases of contraction (4 seconds), using a fairly intense leg press workout (8 sets of 6RM). They found that across all time points the untrained subjects had a greater relative increase in energy expenditure than did the trained subjects (both remained elevated of 48 hours post exercise). Serum creatine kinase and muscle soreness (based on self-report) was additionally elevated, and untrained subjects had significantly higher levels than did trained subjects following resistance exercise. Taken together these results imply that protein resynthesis may be major player in the elevated energy expenditure observed following resistance exercise, as creatine kinase is a blood marker for muscle damage (114). This would explain the observed increase in energy expenditure response from untrained subjects, and the blunted affect in trained subjects, as habitual resistance exercise seems to make individuals more resistant to the muscle damage induced by lifting (115). The greater values of soreness reported by untrained subjects further supports this hypothesis, as soreness can be related to the amount of muscle damage sustained through exercise. Having a greater soreness would then potentially imply that more muscle was
damaged, and therefore more protein resynthesis must take place, and this requires more metabolic activity.

The results of Dolezal were later supported by Hackney et al. (8) who likewise observed that untrained subjects had larger energy expenditure as a result of resistance exercise than did trained subjects. They also used young males, and based their work around the eccentric phase of contraction (3 second eccentric with a 1 second concentric), but instead of doing only one exercise they performed a full body workout (8 exercises, 8 sets, 6 repetitions using rating of perceived exertion (RPE) as a means of determining intensity). Though RPE is not a typical means of determining intensity this protocol must have been effective as an increase in energy expenditure was observed for as long as 72 hours post exercise. Additionally, like Dolezal et al. (7), creatine kinase and rating of perceived muscle soreness was measured. This study also observed both creatine kinase and muscle soreness to be increased following their lifting session; with untrained subjects have a greater response for a longer period of time in all variables.

Taken together these studies suggest that when an equivalent bout of resistance exercise is undertaken by individuals of differing training status the resultant energy expenditure will be affected by the level of training. The EPOC response seems to be negatively correlated with training status based upon the limited research available. These studies also present some of the only mechanistic research in this field, which has provided compelling evidence that muscle damage and subsequent repair plays a major role in the prolonged increased energy expenditure period following resistance exercise.

Future Research

In order to gain a more clear understanding of the factors contributing to the prolonged
energy expenditure observed following resistance exercise more mechanistic research must be conducted. Though some authors have investigated creatine kinase levels, muscle soreness, and other factors, these works have not compared resistance exercise of various intensities. Intensity of loading, though results are mixed, may be an important factor relating to prolonged EPOC magnitude. Future work investigating serum creatine kinase levels at different load intensities will aid in teasing out how much muscle damage (and subsequent repair) accounts for the observed increase in prolonged energy expenditure from resistance exercise. Intensity effects must be explored to a greater extent in order to devise lifting programs which provide the greatest metabolic effect for a given amount of volume / time.

Additionally, there has been no work examining the influence of glycogen resynthesis on prolonged energy expenditure as a result of resistance exercise. Long bouts of resistance exercise may tax glycogen stores enough induce glycogen long term resynthesis, how much this contributes to the increase in energy expenditure observed following resistance exercise has yet to be researched. More work is also required to determine how rest interval affects the acute prolonged energy expenditure. There is only one article which has explored this issue to date, and within this study rest intervals were not the sole variable manipulated, so the conclusions which can be drawn from it are extremely limited (25). With the increased popularity of short intense workouts it is vital that researchers examine the merits of rest interval a means of manipulating energy expenditure.

The volume of work needed to be performed to elicit optimal prolonged energy expenditure in persons of differing training status also needs to be researched. There has been almost no work teasing out how important volume is in relation to the other fundamental factors inherent to resistance exercise. Also, there is no work comparing contraction schemes (i.e.}
concentric vs. eccentric vs. isometric) and prolonged EPOC. There are several studies which have examined differences in energy expenditure following resistance exercise bouts using different concentric and eccentric manipulations, but there are no such studies looking at the prolonged effects.

Beyond the potential for manipulating the various components of resistance exercise the methods used to assess prolonged EPOC are have been to date largely flawed. All but 2 studies examining prolonged EPOC have simply determined the rate of oxygen use for a period of 20-40 minutes at various time points following exercise. While this method is much less resource intensive, and does provide some information, this is simply a rate, not a volume of oxygen usage. It is possible that there could be fluctuations during the prolonged EPOC that may not be accounted for with such gross estimations. Therefore, to take a more accurate account of the prolonged EPOC research using whole room continuous assessment should be conducted for more extended periods. To date there has only been approximately 18 hours of prolonged EPOC studied (88, 2). Learning more about when energy expenditure is elevated in a timeline will allow for more accurate estimations of the mechanisms which are potentially contributing to said increase in energy expenditure.

With the majority of research on prolonged EPOC being centered on young subjects there is much work to be done in both middle and older age groups. The obesity epidemic is centered primarily on both the middle and older age groups, so the effects of resistance training on caloric expenditure are much direr in these groups than the young. Further, most research has focused on trained subjects. More research on untrained subjects may lead to a greater understanding of resistance exercises’ ability to increase energy expenditure, which may in turn serve as an impetus for more participation in this type of activity. Additionally, the vast majority of studies
have focused on male subjects, while females have gone largely unstudied. Resistance exercise may have at one point been a more gender specific activity, but in our society females are becoming more and more involved in lifting. Ascertaining how resistance exercise may affect females’ energy expenditure is therefore more vital.

Finally, research examining popular supplements, and their effects on prolonged EPOC following resistance exercise needs to be undertaken. The ergogenic aid industry is massive, and there are millions of individuals who use these products, but there is no research into their effects on EPOC.

Conclusion

From the available research few conclusions can be drawn about the inherent factors comprising resistance exercise, though the magnitude and duration of prolonged EPOC has some fairly well defined characteristics. The length of the prolonged component following resistance exercise seems to vary in length greatly, but may last as long as 3 days. Most studies have observed the magnitude of this prolonged energy expenditure to be a 4-10% increase in energy expenditure, as shown in Table 4.1. The factors governing this response are largely unknown, but exercise parameters such as intensity, volume, and rest interval seem to influence the prolonged EPOC response. Intensity seems to be positively correlated with energy expenditure, whereas rest interval appears to be negatively correlated. Volume of work has not been proven to significantly influence the prolonged energy expenditure from resistance exercise, though research on this is extremely limited. Training status appears to also be negatively correlated with the prolonged recovery energy expenditure of resistance exercise, though there is very limited evidence. While the prolonged increase of energy expenditure following resistance exercise is only of small magnitude, over the course of several days this small percentage increase can easily meet or exceed the total calories expended during the initial workout. This prolonged period of EPOC is therefore highly important when considering the total energy expenditure of resistance.

Table 4.1: Article Overview. This table outlines some of the basic parameters of the research conducted within each article reviewed. N: Number of subjects, NR: Not reported, NS: Not significant, T: Trained subjects, U: Untrained subjects, L/M: Light and medium intensity
treatments, H: Heavy intensity treatment, Reps: Repetitions, # of Ex: Number of exercises within the resistance exercise protocol.

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>N</th>
<th># of Ex</th>
<th>Sets</th>
<th>Reps</th>
<th>EPOC</th>
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<tr>
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<td>1993</td>
<td>13</td>
<td>10</td>
<td>5 to 6</td>
<td>8-12</td>
<td>5%, 9%; 16 Hours</td>
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<tr>
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<td>1994</td>
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<td>10</td>
<td>5</td>
<td>8-12</td>
<td>4%; 14.5 Hours</td>
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<td>1997</td>
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<td>2</td>
<td>8</td>
<td>10</td>
<td>3%; 48 Hours</td>
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<td>2000</td>
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<td>1</td>
<td>8</td>
<td>6</td>
<td>U: 15%, T: 10%; 48 Hours</td>
</tr>
<tr>
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<td>2000</td>
<td>7</td>
<td>10</td>
<td>5</td>
<td>10-15</td>
<td>4%; 16 Hours</td>
</tr>
<tr>
<td>Melanson et al. (88)</td>
<td>2002</td>
<td>10</td>
<td>10</td>
<td>4</td>
<td>10</td>
<td>5% over 17.5 Hours</td>
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<td>Schuenke et al. (81)</td>
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<td>7</td>
<td>3</td>
<td>4</td>
<td>8-12</td>
<td>38 Hours</td>
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<tr>
<td>Hunter et al. (32)</td>
<td>2003</td>
<td>7</td>
<td>10</td>
<td>1 or 2</td>
<td>8-12</td>
<td>NS. 22 Hours</td>
</tr>
<tr>
<td>Jamurtas et al. (26)</td>
<td>2004</td>
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<td>10</td>
<td>4</td>
<td>8-12</td>
<td>6%; 24 Hours</td>
</tr>
<tr>
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<td>2005</td>
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<td>10</td>
<td>4</td>
<td>10</td>
<td>4% Over 17.5 Hours</td>
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<td>Hunter et al. (5)</td>
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<td>55</td>
<td>8</td>
<td>2</td>
<td>10</td>
<td>NS. 22 Hours</td>
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<tr>
<td>Hackney et al. (8)</td>
<td>2008</td>
<td>16</td>
<td>8</td>
<td>8</td>
<td>6</td>
<td>L/M: 48 Hours, H: 72 Hours</td>
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<tr>
<td>Fatouros et al. (23)</td>
<td>2009</td>
<td>40</td>
<td>10</td>
<td>3</td>
<td>7, 10, 14</td>
<td>U: 9%, T: 7%; 72 Hours</td>
</tr>
<tr>
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<td>2011</td>
<td>8</td>
<td>10</td>
<td>1 or 3</td>
<td>10</td>
<td>5%; 72 Hours</td>
</tr>
<tr>
<td>Petrofsky et al. (112)</td>
<td>2011</td>
<td>10</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>9%; 24 Hours</td>
</tr>
<tr>
<td>Paoli et al. (25)</td>
<td>2012</td>
<td>17</td>
<td>8 or 5</td>
<td>4 or 3</td>
<td>8-12, 10</td>
<td>18%; 22 Hours</td>
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</table>
PROLONGED PHASE OF RECOVERY: SUBSTRATE UTILIZATION AND RESISTANCE EXERCISE

Following exercise, energy expenditure is often increased for hours, and in some instances days. During this period of recovery from exercise the substrates utilized have been largely unstudied. The therefore the effect of resistance exercise on prolonged substrate utilization are unclear. There are approximately a dozen studies published which have examined this issue and methodologies, research questions, and protocols employed are highly varied. Though research in this field is scarce, studies have been grouped together based upon common research aims, in an effort to compare findings of like study design.

Characterization of Substrate Utilization Response

The studies presented within this subsection all examined substrate utilization as a result of resistance exercise. The aim of these experiments was to characterize both how respiratory exchange ratio was affected by resistance exercise, and over what duration. Respiratory exchange ratio is the most prevalent method of ascertaining substrate utilization from a whole body perspective. This method involves measuring the ratio of oxygen to carbon dioxide being inhaled and exhaled. Based upon the ratio of these expired breath gases substrate utilization can be estimated. High respiratory exchange ratio values are indicative of more glycolytic metabolism while lower respiratory exchange ratio values represent fat metabolism. Acid-base buffer activity is not a confounder within these studies as replenishment of these systems doesn’t last longer than the short phase of EPOC.

Melby et al. (27) first investigated the effects of resistance exercise on prolonged substrate utilization in 1993. They conducted two experiments, which were ultimately pooled for statistical purposes. They used young trained male subjects within this experiment, all of whom performed 5–6 sets of 8–12 repetitions on 10 exercises using approximately 70% of their
1RM. Substrate utilization was measured before, and 15 hours following the bout of resistance exercise. Though a trend toward resistance exercise resulting in a lower respiratory exchange ratio existed, statistical significance was not reached (Baseline: 0.82, Resistance exercise: 0.79).

In 1997 Williamson et al. (110) examined how resistance exercise would affect prolonged substrate utilization, but measured it for a longer time post exercise. This study measured untrained older males’ substrate utilization before, and 48 hours post exercise. Their subjects performed 8 sets of 2 exercises (10 repetitions using 75% of their 3RM). The lifting protocol was somewhat odd in that they only performed the concentric portion of each repetition, and had quite a large volume of work split between only 2 exercises. Ultimately no change was observed in substrate utilization from pre to 48 hours post exercise.

Later, Osterberg et al. (79) investigated this issue using trained young female subjects. Their subjects performed 10 exercises (5 sets of 10-15 repetitions using 70% of their 1RM). This group found that resistance exercise resulted in a prolonged depression of respiratory exchange ratio 16 hours post exercise (Baseline: 0.84, Post Exercise: 0.81). This reduction signified that fat metabolism was increased compared to baseline, and contradicted the findings of the previous researchers.

In 2002 Schuenke et al. (81) similarly explored how resistance exercise influenced prolonged substrate utilization, but instead of just measuring a single before and after time point they measured 11 different times (5 before, and 6 after exercise). The time span measured was from 34 hours pre exercise to 48 hours post exercise. Their young trained male subjects performed 4 sets of 3 exercises (8 – 12 repetitions using 70%-80% of their 1RM). Respiratory exchange ratio was significantly reduced by this resistance exercise protocol, but the only time points which were significant were the 29 hours pre-exercise (0.9) compared to the 43 hours
post-exercise time point (0.84). While this is a rather large difference in respiratory exchange ratio, and could potentially signify an increase in fat metabolism, the lack of other time points being significantly different leaves it suspect. With 11 points to compare, these researchers may have simply run paired comparisons until one was significantly different. So while this is a positive result the lack of transparency in the presented data makes the validity of these findings questionable.

Finally, Melanson et al. (2) in 2005 used whole room calorimetry to examine how a bout of resistance exercise could affect substrate utilization. The middle aged untrained females of this study performed 4 sets of 10 repetitions using 70% of their 1RM on 10 exercises. Substrate utilization was measured over the course of a 24 hour period, which included pre, during, and post workout. Ultimately, while respiratory exchange ratio was significantly elevated during exercise there was no resultant decrease in respiratory exchange ratio observed following exercise. They therefore concluded that resistance exercise did not affect substrate utilization following cessation of work.

The researchers examining the prolonged effects of resistance exercise on substrate utilization have presented contradictory findings. Approximately half of the studies have reported increase fat metabolism in the prolonged period of recovery following resistance exercise, while the others have found no change whatsoever. With only five studies examining this topic any conclusions will be tenuous, but training status may be an important factor in substrate utilization following resistance exercise. In the two studies which reported decreases in respiratory exchange ratio subjects were trained (79, 81). Additionally, Melby et al. (27) used trained subjects and had a trend toward resistance exercise resulting greater fat metabolism. The two studies which did not find any difference following resistance exercise used untrained
subjects (110, 2). If training status was an influential variable than perhaps more efficient use of glucose/glycogen (common amongst trained individuals) may have caused the increase in observed fat metabolism following resistance exercise (116). This observed relationship may be coincidental, as the number of studies is quite limited. These studies alone do not present enough evidence to conclude that substrate utilization is changed during the prolonged phase of recovery from resistance exercise.

**Comparisons of Resistance and Endurance Exercise**

Several studies have used comparisons of endurance type exercise with resistance exercise as a means of gaining insight into their potential benefits and differences. The primary metabolic systems utilized during resistance exercise and endurance exercise are largely different (depending upon intensity of work), but it is this difference that can potentially delineate the mechanisms underlying recovery substrate utilization. With the understanding that endurance exercise is mostly aerobic (at lower intensities) and resistance exercise is largely anaerobic the prolonged recovery substrate utilization response to each may indicate how using various metabolic pathways could contribute to changes in substrate utilization. Additionally, resistance exercise typically results in more muscle damage and subsequent repair, while endurance exercise typically depletes glycogen stores to a greater extent. The process of repairing muscle and replenishing glycogen seems to have a role in the prolonged EPOC secondary to resistance exercise. How these processes influence substrate utilization has not been extensively studied, perhaps they may play a role in recovery substrate utilization. The following studies have all measured the prolonged response of substrate utilization following both an endurance exercise and resistance exercise treatment.
Gillette et al. (86) in 1994 used trained young male subjects to assess the effects of either a resistance exercise protocol or cycling. Their intent was to control for work performed by first having their subjects perform the lifting treatment, then based upon the breath gases analyzed have their subjects cycle (50% of VO2max) for as long as it took to match the caloric expenditure. The lifting treatment consisted of 10 exercises during which 5 sets of 8–12 repetitions were completed using 70% of their 1RM. Substrate utilization was assessed before and 14.5 hours following each treatment. Ultimately, they found that the resistance exercise treatment elicited a greater reduction in respiratory exchange ratio than the endurance exercise treatment (0.84 and 0.87 respectively). While this was a significant finding, and indicates a greater fat metabolism during the prolonged recovery phase following resistance exercise, the work performed during each treatment was likely not matched. The anaerobic metabolism during the resistance exercise treatment was not accounted for when the cycling workload was prescribed. With lifting involving a large quantity of anaerobic metabolism the cycle treatment was presumably far less demanding. Therefore, while resistance exercise did have a greater response the disparity between the volumes of work completed confounds these findings.

In 2002 Melanson et al. (88) performed a similar experiment comparing lifting with cycling, but used whole room calorimetry. The resistance exercise protocol was largely the same as that of Gillette et al. (86), but the endurance exercise treatment consisted of 50 minutes of cycling at 70% of their VO2max. Substrate utilization was determined over the course of the 24 hours stay in the metabolic chamber, and various time points before and after each treatment were reported. No differences between treatments or control were reported in substrate utilization. Respiratory exchange ratio was elevated during each treatment, but there was no change following work.
Later, Jamurtas et al. (26) but compared resistance exercise, and running at 70%–75% of their VO$_{2\text{max}}$ for 60 minutes. The trained young males of this study followed the same resistance exercise protocol utilized by Melby et al. (27). While calorically the endurance exercise treatment was much more demanding, the respiratory exchange ratio values 10 and 24 hours following each treatment were the same. Both the endurance exercise and resistance exercise treatments resulted in a depression in respiratory exchange ratio at 10 hours (-7%), and at 24 hours (-3%). Substrate utilization was also assessed at 48, and 72 hours post exercise, but no differences were found. Both the endurance exercise and resistance exercise treatments of this experiment involved a significant anaerobic metabolism and both elicited significant reductions in respiratory exchange ratio. Additionally, creatine kinase levels were measured concurrently with each assessment of substrate utilization. Creatine kinase, often linked to muscle damage, was elevated during the first 24 hours following each treatment. These findings suggest that working at intensities which necessitate anaerobic work, and result in muscle damage, may stimulate an up-regulating of fat metabolism during the prolonged phase of recovery following said exercise.

Lastly, Hunter et al. (5) in 2006 had trained young females perform either a bout of resistance exercise or endurance exercise. The resistance exercise treatment consisted of 8 exercises (2 sets of 10 repetitions using 80% of 1RM). The endurance exercise treatment was a 40 minute treadmill walk or jog at 80% of their max heart rate. Substrate utilization was assessed before and 19, 33, and 67 hours post exercise. Neither treatment resulted in a statistically significant difference in respiratory exchange ratio at any post exercise time point. Perhaps the lower volume of work performed during this study’s resistance exercise treatment
was insufficient to elicit a depression of respiratory exchange ratio which has been observed by other researchers using greater workloads.

These four studies present a variety of responses to endurance exercise and resistance exercise protocols. While half of these articles have found significant findings in substrate utilization following at least one modality of exercise the other half has found no result from either. Studies which have reported changes in substrate utilization have been of fairly low magnitude (0.03 change in respiratory exchange ratio on average), which is approximately what has been reported within the first section of articles reviewed. Likewise, the proportion of null findings is approximately that of the last section as well. While there are a meager number of studies from which to draw conclusions low intensity work does not appear affect substrate utilization during prolonged recovery, while working at higher intensity may. With only half of researchers finding significant results in substrate utilization during the prolonged period following exercise it is difficult to speculate as to how either of these modalities compare.

Effects of Manipulating Resistance Training Parameters

The studies reviewed within this section have tested for differences in substrate utilization during the prolonged phase of recovery using varied protocols of resistance exercise. These works have either altered the volume, intensity, rest interval, contraction cadence, or some combination of these factors inherent to conventional resistance exercise.

Hunter et al. (32) compared the effects of “super slow” resistance exercise with traditional training. The young trained male subjects of this study performed 10 exercises using either traditional training (2 sets of 12 repetitions using 65% of their 1RM with a contraction cadence of approximately 1 second per eccentric and concentric contraction) or super slow (1 set of 8 repetitions using 25% of their 1RM with 10 second concentric and 5 second eccentric
contractions). Ultimately, when substrate utilization was assessed 22 hours post exercise there were no differences between either treatment or the control.

In 2011 Heden et al. (113) compared two resistance exercise protocols with differing volumes. Untrained young males performed 10 exercises with either 1 or 3 sets of 10 repetitions using approximately 70% of their 1RM. Respiratory exchange ratio was assessed 24, 48, and 72 hours post exercise, as well as a rating of perceived muscle soreness. While perceived muscle soreness was significantly greater than baseline up to 48 hours there were no changes in respiratory exchange ratio observed in either treatment.

Later Paoli et al. (25) examined how intensity of load and rest interval influenced substrate utilization during the prolonged phase of recovery from resistance exercise. The high intensity interval training treatment consisted of 5 exercises, 3 sets, and 3-6 repetitions using approximately 85% of their 1RM with 2.5 second rest intervals, while the traditional lifting was 8 exercises, 4 sets, and 8-12 repetitions using approximately 70% of their 1RM with 1-2 minute rest intervals. Substrate utilization was assessed before and 22 hours post exercise. The higher intensity treatment resulted in a significant reduction in respiratory exchange ratio (Pre Trial: 0.83, Post Trial: 0.8) while the traditional training treatment did not have a significant result. The traditional training treatment entailed much more volume than then high intensity treatment, but did not have any effect on respiratory exchange ratio during prolonged recovery. This may imply that intensity is a more important factor then volume when considering how resistance exercise influences recovery substrate utilization.

These three studies have utilized largely dissimilar methodologies, and examined a variety of questions. The quantity of literature examining manipulation of resistance exercise variables as a means of altering prolonged recovery substrate utilization is miniscule. Therefore,
while volume and contraction velocity do not seem to affect prolonged substrate utilization, and intensity may be negatively correlated with respiratory exchange ratio, these observations of are limited by only having a minimal body of evidence.

**Future Research**

In general there is a scarcity of research exploring the effects of resistance exercise on prolonged substrate utilization. With less than half of all the studies in this area reporting any change in substrate utilization more work must be done to tease out if there is an effect. A potential limiting factor has been the low number of subjects within these studies, and thus trends toward greater fat oxidation have often been observed that did not reach the 0.05 threshold of statistical significance. With greater subject pools there would be more certainty as to whether substrate utilization is altered by resistance exercise. Additionally, the time course of how substrate utilization is affected by resistance exercise has yet to be established based upon the current literature. While several studies have investigated how substrate utilization may be changed several days following resistance exercise, the 3-14 hours post exercise timespan has not been well studied. Further, only 2 research groups have studied the prolonged effects of resistance exercise on substrate utilization in age groups other than young people. While younger subjects are undoubtedly more accessible to most researchers their responses may differ from those of middle aged and older individuals. These middle and older aged populations must be studied because often these are the individuals with the greatest weight management problems. If resistance exercise could serve a means of increase fat oxidation for a prolonged period of time following a workout than these populations may benefit from this research. Additionally, there have been no studies directly comparing trained with untrained subjects to ascertain how training status impacts substrate utilization during prolonged recovery. The
potential mechanisms underlying changed in substrate utilization following resistance exercise have likewise been largely understudied. With such a small number of studies comprising the body of evidence in this area it is difficult to draw conclusions about how resistance exercise affects substrate utilization.

**Conclusion**

The effect of resistance exercise on prolonged recovery substrate utilization is not entirely clear. The majority of studies have observed there to be no response in prolonged substrate utilization following resistance exercise. Those studies which have found a change have all reported resistance exercise to promote an increased fat metabolism during prolonged recovery. Respiratory exchange ratio reductions have been observed by some researchers for as long as 48 hours post exercise, while others have been unable to find a change 15 hours following resistance exercise. When various exercise protocols have been compared there are no apparent trends within intensity, volume, repetition scheme, training status, or gender which would account for the great disparity of findings with this literature, as shown in Table 5.1. The magnitude of change reported by researchers who have observed increased fat oxidation has been fairly minimal, which may suggest that the non-significant findings were just above the threshold of significance, while some where just below. This could therefore suggest that substrate utilization is only altered to such a minor extent that it is often not considered statistically significant. Regardless of the cause how substrate utilization changes during prolonged recovery as a result of resistance exercise is not well understood.
Table 5.1: Summary of Articles. This table outlines some of the basic parameters of the research conducted within each article reviewed. N: Number of subjects, # of Ex: Number of exercises performed during the treatments, RER: Respiratory exchange ratio, NS: No statistically significant findings, RE: Post resistance exercise, C: Control or baseline, EE: Endurance exercise, Hi: Post high intensity resistance exercise

<table>
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<tr>
<th>Study</th>
<th>Year</th>
<th>N</th>
<th># of Ex</th>
<th>Sets</th>
<th>Reps</th>
<th>RER Response</th>
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<tr>
<td>Melby et al. (27)</td>
<td>1993</td>
<td>13</td>
<td>10</td>
<td>6, 5</td>
<td>8-12</td>
<td>15 Hours; NS</td>
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<td>Williamson et al. (110)</td>
<td>1997</td>
<td>12</td>
<td>2</td>
<td>16</td>
<td>10</td>
<td>48 Hours; NS</td>
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<td>10-15</td>
<td>16 Hours; RE: 0.81, C: 0.84</td>
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<td>4</td>
<td>8-12</td>
<td>48 Hours; RE: 0.84, C: 0.9</td>
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<td>10</td>
<td>4</td>
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<td>Jamurtas et al. (26)</td>
<td>2004</td>
<td>10</td>
<td>10</td>
<td>4</td>
<td>8-12</td>
<td>10, 24 Hours; RE=EE &lt; C (7%, 4%)</td>
</tr>
<tr>
<td>Hunter et al. (5)</td>
<td>2006</td>
<td>55</td>
<td>8</td>
<td>2</td>
<td>10</td>
<td>67 Hours; NS</td>
</tr>
<tr>
<td>Hunter et al. (32)</td>
<td>2003</td>
<td>7</td>
<td>10</td>
<td>1, 2</td>
<td>8-12</td>
<td>22 Hours; NS</td>
</tr>
<tr>
<td>Heden et al. (113)</td>
<td>2011</td>
<td>8</td>
<td>10</td>
<td>1, 3</td>
<td>10</td>
<td>72 Hours; NS</td>
</tr>
<tr>
<td>Paoli et al. (25)</td>
<td>2012</td>
<td>17</td>
<td>8 or 5</td>
<td>4, 3</td>
<td>8-12, 10</td>
<td>22 Hours; HI: 0.83, C: 0.8</td>
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CHRONIC ADAPTATIONS TO RESISTANCE EXERCISE: RESTING METABOLIC RATE

Resistance exercise performed regularly over a long period of time has some definite effects, such as hypertrophy of skeletal muscle, maintenance or gain of bone mineral density, and increased strength (63, 67). The effect of long term resistance exercise on metabolic rate is not entirely clear; some research shows there to be an absolute increase in resting metabolic rate, but not relative to fat free mass, while others have observed no response. The mixed results are likely linked to the variations in population, age group, or training programs, though even when studies are largely similar there is often much discordance between results. This review will group studies by likeness in the hopes of discerning what effects resistance training has on resting metabolic rate.

Cross-Sectional Research

Some of the first research undertaken to determine the long term effects of resistance training on resting metabolic rate were cross-sectional in design. These investigations gathered individuals who had been performing resistance exercise habitually for a criterion period of time, and measured their resting metabolic rate versus those who had not. These studies are fairly simple to conduct, and their impact is limited. When no baseline is measured prior to training it is difficult to separate individual differences from those of training adaptation. Additionally, the variety within resistance training programs is immense, and therefore the adaptations are likely to be greatly varied, which may introduce a great deal of variability between results.

Amongst the first to conduct this type of research on resistance exercise was Poehlman et al. (117) in 1992. These researchers were interested in comparing the differences between resistance trained, endurance trained, and untrained young males. They found resistance trained subjects to have the greatest resting metabolic rate (+7% when compared to endurance training,
+13% compared to the untrained group) from an absolute perspective. Though, when their resting metabolic rate per kilogram of fat free mass was assessed endurance training resulted in the greatest response (+5% vs. resistance training, and +10% vs. untrained group). The subjects within this group were likely good representatives of their respective training modalities, as the resistance training group had much more fat free mass than did the endurance training and untrained groups. Likewise the endurance training group on average had a much greater \( \text{VO}_{2\text{max}} \) than either of the other groups.

This finding was contradicted at Ballor et al. (118) the same year, who conducted nearly the same investigation but with young females. They found the resistance trained and endurance trained groups to have a similar resting metabolic rate, which was still 7% greater than the untrained group. Their comparison of resting metabolic rate relative to fat free mass revealed only endurance training to result in a significantly increased metabolic rate. Menstrual cycle was accounted for, and the fat free mass of each group was not significant different. The lack of difference in fat free mass suggests that perhaps the lifting group was not highly trained (as habitual resistance training typically results in hypertrophy), and therefore the results may not be representative of the effects of resistance training on resting metabolic rate.

Toth et al. (119, 120) conducted two studies in 1995 using middle aged subjects, one with females and one with males. In both of Toth’s studies the resistance trained and endurance trained groups had the same absolute resting metabolic rate, which was 17% greater than the untrained group. When comparing resting metabolic rate relative to fat free mass resistance training had no significant effect in males (119), while females has 12% greater resting metabolic rate relative to fat free mass than their untrained counterparts (120). What should be mentions is that within both of these studies the resistance training group did not have
significantly more fat free mass than either of the other groups, which calls into question how trained their subjects were. Regular resistance training over a long period of time should result in significant fat free mass increase, so perhaps some subjects were not as trained as was reported. It is interesting that the study using females had large significant differences in resting metabolic rate relative to fat free mass, while the study with males did not.

In contrast to other studies in this area, Bosselaers et al. (121) studied body builders compared with untrained subjects using whole room indirect calorimetry. They observed the resistance trained group to have an approximately 14% greater resting metabolic rate than the untrained group, but when fat free mass was controlled for the groups were not different. This study further supports the notion that resistance training only chronically increases resting metabolic rate through its ability to hypertrophy muscle. This study used mostly young male subjects (and one female) which were likely highly trained as they had almost 10 kg of fat free mass more than their untrained counterparts.

Though previous studies has found resistance training to at least have some effect on resting metabolic rate, either relative or absolute, Byrne et al. (122) found neither. This research group sought to compare both mode and intensity of habitual exercise on resting metabolic rate of young females. Ultimately they found that regardless of the method of training that there were no differences in resting metabolic rate, though trends of higher intensity exercise modalities producing larger responses were observed. These finding are puzzling, as five previous studies have found significant results, while this one would seemingly suggest that no matter the type of exercise there is no result in resting metabolic rate. A possible explanation for this result could be the methods employed in grouping individuals. Their method of separating subjects was entirely based on the number of days per week they exercised, and their perceived
level of training. This method of differentiation is highly subjective, and likely led to subjects being misclassified. There were no differences in fat free mass between the resistance trained and endurance trained groups, which indicates that subjects were likely not well trained.

Ultimately, these six studies present many conflicting findings, but the most consistent observation has been a significant increase in resting metabolic rate as a result of habitual resistance training. Most studies have found this increase to be linked solely to the increase in fat free mass often associated with resistance training, though there is limited research which suggests that resting metabolic rate may increase even relative to fat free mass. The lack of consistent findings within these cross-sectional studies is likely linked to subject characteristics and selection. The vast number of different resistance training methodologies makes recruiting like subjects problematic, and a major factor which was not accounted for by many of these studies was fat free mass as an indicator of training status. In several studies there were criterion values for VO$_{2}\text{max}$ required for inclusion into endurance trained groups, while there were no criteria for inclusion into resistance trained groups other than reporting a certain number of days per week of training for a certain duration. Based upon these studies resting metabolic rate seems to be increased by resistance training, though this increase appears to be highly related to increases in fat free mass.

**Short Term Training Studies**

This group of studies focuses on resistance training intervention studies which had a length of less than six months. Of the research dedicated the chronic resting metabolic rate adaptations as a result of resistance training, these studies are the most numerous. The advantage to training subjects as opposed to measuring individuals who were already trained is that baseline characteristics can be assessed. Then following training subjects can be reassessed, and the
differences can then more accurately attributed to the intervention. There still are significant limitations with training studies such as individual participation levels, controlling for diet, other physical activity, and environmental factors. Additionally, as the work of Sale et al. (123) has shown peripheral nervous system adaptations comprise the majority of the initial physiological changes observed during a training program. Therefore short term training interventions may not be highly indicative of the true physiological adaptations associated with resistance training. While these types of studies do have their limitations they do allow for a greater degree of control than does cross-sectional research.

The first training study to investigate resistance training was Broeder et al. (124) in 1992. This study sought to evaluate how resistance training or endurance training would affect the resting metabolic rate of their young male subjects after 12 weeks of training. Subjects of this study performing 11 exercises 2 days per week using variable intensities (not clearly stated) that allowed for subjects to complete 8-10 repetitions in each of 3 sets. They found that neither intervention increased resting metabolic rate, though there was a significant 2.1kg increase in fat free mass as a result of the resistance training treatment. Increasing fat free mass often results in greater resting metabolic rate values, but these subjects were in negative caloric balance throughout the study (as determined by diet recall). While there was no increase in resting metabolic rate, resistance training was able to attenuate the decrease in resting metabolic rate often associated with negative caloric balance.

Then Pratley et al. (125) examined how resistance training affected not only resting metabolic rate, but norepinephrine and thyroid hormone as potential mechanisms. The middle to older aged male subjects performed resistance training 3 days per week, using 14 exercises which comprised a full body workout. They only performed 1 set of 15 repetitions on each
exercise with a variable intensity that was adjusted throughout each set to allow for completion of all repetitions. Their 16 week intervention resulted in a 7% increase in resting metabolic rate, which was still significant when compared with fat free mass (gain of 1.6kg), though of smaller magnitude (5%). Additionally, they found that norepinephrine was increased by 36%, while thyroid hormone levels had not changed. Therefore norepinephrine was associated with the increase in resting metabolic rate that fat free mass did not account for. A possible confounding factor within this study was the length of time between the last workout of the intervention, and the post trained measurements of resting metabolic rate. There were only 22-24 hours between last workout and these measurements. Therefore the resting metabolic rate data obtained may have been confounded by a prolonged increase in energy expenditure common to resistance exercise recovery. Additionally, there was no control group attached to this study, and therefore the seasonal variations within resting metabolic rate were not accounted for.

In 1995 Ryan et al. (126) also studied resistance training and norepinephrine levels, though this group did not find norepinephrine to be related to the increase in resting metabolic rate observed. This study compared obese and non-obese post-menopausal women performing resistance training workouts 3 days per week (14 exercises, and 2 sets of 15 repetitions) over a 16 week period. These researchers observed there to be an approximate 50 kcal increase in the resting metabolic rate within both group despite neither experiencing a significant change in fat free mass. Without having controlled for diet during training, and the lack of a control group to help account for seasonal variations this increase in resting metabolic rate may or may not be the result of the resistance training protocol undertaken by these women.

Treuth et al. (127) also conducted a 16 week long study using post-menopausal women, and found a 9% increase in absolute resting metabolic rate. When compared with fat free mass
gain the increase in resting metabolic rate was not significant. This group used a resistance training protocol which was nearly identical to Ryan et al. (126), though they did include dietary recall as a form of controlling for potential changes in resting metabolic rate based on eating habits. Once again no control group was included, so these results may be the result of seasonal variation. Additionally, their use of postmenopausal women limits the application of these findings to the general population. While there are a significant number of postmenopausal women in the world their altered hormonal levels and subsequent physiology make these findings less applicable to other populations.

Sale et al. (128) compared endurance training, resistance training, and a non-exercise control group over a 12 week period, and found no change in resting metabolic rate between any of the groups of middle aged women. These results are somewhat expected as the resistance training protocol was comprised of using hand weights of approximately 2 pounds in a group exercise type setting, and the endurance training group simply walked. This stimulus was not sufficient to promote a significant response, which was evident by the lack of fat free mass change observed. Additionally, although menstrual cycle was well controlled for they only allowed 22-24 hours between the last bout of exercise, and the post training resting metabolic rate measurements. Should the training stimulus been of sufficient intensity that one might expect an interaction with the prolonged portion of EPOC. The lack of any difference in resting metabolic rate serves as further evidence that the training protocols used were simply not of rigorous enough to elicit any adaptations.

Van Etten et al. (129) approached resistance training related changes in metabolic rate from a different perspective; this group examined the effects of resistance training on sleeping metabolic rate. Subjects exercised 2 days per week performing full body workouts (14
exercises) with an intensity and scheme of sets that was different for each exercise, though
seemed to be fairly intense as a whole. Ultimately they found that after 12 weeks of training
their middle aged male subjects did not experience any change in sleeping metabolic rate
whatsoever, though it should be mentioned that their baseline measures were taken
approximately one week after the training program had already begun. Taking pre measures
during training may have inflated the baseline measures, and therefore made the post training
measures less representative of the changes which may have occurred. Additionally, without
having accounted for seasonal changes through use of a control group, resistance training may
have increased sleeping metabolic rate, but a shift in acclimatization could have reduced it. This
theory is further supported by the significant increase in fat free mass gained over the course of
this study without any difference in sleeping metabolic rate. Also diet was not controlled, which
is another potential confounding factor.

In 1998 Dolezal et al. (130) compared resistance training, endurance training, and
combined training (resistance training and endurance training) over a 10 week training period.
Young male subjects were selected for this study. The resistance training group performed 13
exercises 3 days per week using loading scheme which gradually increased to 3 sets of 80% of
their 1RM (repetitions per set were also variable). Though not easily defined this resistance
training program must have been fairly effective as there was an average 2.1 kilogram increase in
fat free mass. The endurance training group increased intensity gradually, starting with 65% of
heart rate max, and ending with approximately 80%, for 25 to 40 min 3 days per week. The
combined training group performed both the resistance training and endurance training workouts.
Though this was a fairly short training period these researchers found that both the resistance
training and the combined group had an increased resting metabolic rate following training (6%
and 5% respectively), though not significant once fat free mass was taken into account. The endurance training group actually had a significant reduction in resting metabolic rate (-3%). As with many of the previous studies the lack of a control group calls into question just how much the resting metabolic rate change can be accounted for by the different training protocols.

Trevisan et al. (131) observed a 9% increase in resting metabolic rate following their 16 week long training program utilizing post-menopausal women. This study did control for seasonal variations by use of a control group. The resistance training group performed 10 exercises 3 days per week with a variable intensity which allowed for 3 sets of 8 to 12 repetitions. This program was seemingly effective as there was an average increase in fat free mass of 1.8kg. Though this increase in resting metabolic rate was one of the largest observed over a short term training period it was not significant once fat free mass was taken into account, and the post testing values may have been inflated. The post training resting metabolic rate testing was only 24 hours following the last workout treatment, and diet was not taken into account whatsoever. The prolonged portion of EPOC has been reported to last as long as 72 hours (2, 23). This suggests that some of the observed increase in resting metabolic rate may have in part been due to EPOC and not adaptations to training.

Again in 1997 Van Etten et al. (132) examined the effects of resistance training on sleeping metabolic rate, and found similar results to their first investigation, i.e. no effect. This investigation differed from their first in 1995 (129), mainly due to their adding a control group, dietary recall, and increase in study duration (18 weeks instead of 12). The training protocol was largely the same, which was once again effective at increasing fat free mass (2.1kg gained). Having controlled for the factors they initially did not, and achieving ultimately the same result this study strengthen the observations of the first study they performed in 1995.
Byrne et al. (133) compared the effects of 20 weeks of resistance training or combined resistance exercise and endurance training on resting metabolic rate. Their middle aged male subjects gained 1.9 kilograms of fat free mass in the resistance training group. They exercised 4 days per week (splitting upper and lower body, so actually only doing a whole body workout 2 days per week). Their loading scheme was such that the intensity was gradually increased throughout the study, but 3 sets of each exercise were performed. Resistance training resulted in an absolute increase in resting metabolic rate (3%), while the combined treatment resulted in a reduction in resting metabolic rate (-4%), and the control group had no change. When fat free mass change was taken into account, the change in resting metabolic rate observed in the resistance training treatment was no longer significant. A major confounding factor for the combined group was their having performed the endurance training portion of training outdoors during the summer. Heat acclimatization can significantly reduce resting metabolic rate, and this may account for the drop in resting metabolic rate observed over the course of training (134). Diet was controlled through diet logs which the subjects maintained over the course of training.

The majority of research examining the effects of short term resistance training on resting metabolic rate has resulted in chronically increased resting metabolic rates. Though many researchers have observed this, the widespread lack of control reduces the impact these studies have. Diet was fairly well taken into account by use of recall in the majority of studies, though there were a few who did not do any kind of dietary analysis. Changes in diet can influence resting metabolic rate, and therefore training studies must include some measurement of diet to account for this confounder. Half of these studies did not have control groups, so changes in seasonal weather conditions alone may have altered resting metabolic rate through the effects of acclimatization. Additionally, many studies have lacked sufficient training intensity to elicit
changes in fat free mass, and therefore a null finding could be expected. Overall, despite lacking control measures for confounding variables the existing literature indicates that resistance training programs of less than 6 months duration are effective at increasing resting metabolic rate from an absolute perspective, but not relative to fat free mass.

**Long Term Training Studies**

The studies within this section are all of at least six months in duration. Studying the effects of resistance training over a longer period of time makes for a more thorough assessment of the effects of habitual resistance training than shorter term studies. The initial adaptations to resistance training are largely neurological, and when untrained subjects are put into any exercise intervention they are likely to have large rapid changes (123). Training subjects for longer period of time allows researchers to gauge more clearly the true physiologic adaptations associated with habitual resistance training. The major limitation of conducting research of this nature is the time required, and subject retention. Additionally, without control groups the climate change over any given 6 month period can make resting metabolic rate measurements problematic as acclimatization can significantly enhance or suppress resting metabolic rate. All of the long term resistance training studies have used a mixture of free weights and weight machines during their protocols. Additionally, all of these studies have conducted full body training programs (including approximately 10 exercises) three days per week.

The first study to examine resistance training over a long term training period was Taaffe et al. in 1995 (135). Their study examined the effects of either low or high intensity (40% or 80%) utilizing an equal volume on older female subjects. No change in resting metabolic rate was observed over the course of this yearlong study, though the lack of change in fat free mass suggests that there must have been some participation or study design problems. Older
individuals often have under developed musculature, and therefore typically respond well to resistance training, but for some unaccounted for factor no changes in fat free mass were elicited by this protocol. This may explain the lack of resting metabolic rate change observed within this study. Also, the methodology used to assess resting metabolic rate was somewhat questionable. Douglas bags were employed, and breath gasses were measured for only 10 minutes, with only a 5 minute rest period prior to assessment. The potential difference in resting metabolic rate associated with resistance training is somewhat small in magnitude; therefore a rest period of more than 5 minutes should have been taken before obtaining breath gas measurements, as this may have been insufficient to bring the body to homeostatic resting levels. Though each measurement may not have been well controlled, they averaged three consecutive mornings at each time point assessed, and therefore the variations should have been mitigated.

Hunter et al. (136) in 2000 then similarly examined the effects of resistance training on elderly subjects of the same approximate age as Taaffe et a. (135), though within this study both males and females were included. Intensity of load was approximately 65%-80% for 2 sets of 10 repetitions. Unlike Taaffe, Hunter observed there to be a significant increase in resting metabolic rate as a result of their 6 month training program from both an absolute and relative (to fat free mass) perspective (6.8% and 3.5%). These results are somewhat questionable due to the lack of a control group and any dietary control. The increase in resting metabolic rate may have been the result of seasonal acclimatization to either warm to cold weather, or an overall increase in food consumption. These limiting factors therefore reduce the impact of this study’s results.

In 2002 Poehlman et al. (137) used a single set protocol with intensity similar to Hunter et al. (136) for the same 6 month duration, and found no change in resting metabolic rate. The subjects of this experiment were young females as opposed to Hunter’s older subjects, and
gained 1 kilogram of fat free mass over the course of training. This increase in fat free mass should have elicited an increase in resting metabolic rate from an absolute perspective; it is therefore odd that there was no significant change. This study included a control group, so environmental acclimatization related changes in resting metabolic rate were controlled for. These researchers did fail to control for any dietary factors, and therefore changes in eating behaviors may have contributed to the discrepancies within this study.

Later, Dionne et al. (138) in 2004 performed an experiment using a fairly rigorous intensity of 80% for 3 sets using either young or older females; in order to compare age related differences. Though both young and old subjects gained fat free mass (young more so than old) only the young group experienced an increase in resting metabolic rate (5%), though this difference was not significant when fat free mass was accounted for. Like Hunter et al. (136) this study did not account for diet or include a control group, so the same limitations apply.

To compare intensity effects on long term resistance training Fatouros et al. (9) split subjects into 3 groups, and had them train for 6 months, and then detrained for another 6 months. Groups were as follows: Low (14 reps, 45%-50%), Medium (10 reps, 60%-65%), and High (8 reps, 80%-85%). Although the volume lifted was unequal (intensity was inversely related to volume) the intensity was positively correlated with resting metabolic rate change. The high intensity group experienced a 9% increase in resting metabolic rate, while the medium had a 7%, and the low intensity group had only a 3% increase. This study did include a control group, which had no change in resting metabolic rate over the training period. Following the 6 month period of detraining, intensity still seemed to have an effect, as it once again was positively related with maintenance of resting metabolic rate. Diet was not controlled for by these researchers, so this may cast doubt upon the results.
In 2009 Kirk et al. (139) experimented with a minimal lifting program of only 1 set of 3-6 repetitions of whole body lifting using 85%-90% intensity. Though this workout required only 11 minutes to complete resting metabolic rate was increased by 7% following 6 month of training. Diet was controlled for through recall, and there was a control group to account for environmental factors. The control group also experienced a nearly significant increase in resting metabolic rate; their increase was large enough that when the experimental group was compared to the control group the increase in metabolic rate from lifting was no longer significant. This suggests that the increase in resting metabolic rate observed through resistance training was likely due in large part to environmental changes. A major strength of this study was their use of whole room indirect calorimetry and their control of the major confounding factors inherent to this type of research. All measures of resting metabolic rate were obtained for a full day.

Ultimately, the long term intervention studies examining the effects of resistance training have yielded mixed results amidst poor experimental control. While the majority of research has found resistance training to result in increases in resting metabolic rate, most studies have observed this to only be related to the increase in fat free mass secondary to training. The use of higher intensity seems to be beneficial in eliciting increases in resting metabolic rate, while some studies have found no differences regardless of intensity. With the exception of one study all resting metabolic rate data has been based upon rate of O$_2$ consumed over a given period of time. This rate of oxygen usage has then been extrapolated based upon the assumption that the rate at this point of the day (the morning in these studies), is constant throughout the day. Therefore the effects of long term resistance training have only been tested on early morning metabolic rate, in nearly all studies reviewed. It may be possible that resistance training affects metabolic rate at
different points in the day, but testing only in the morning will not detect this. Diet is a confounding factor that has not been well controlled for, with the exception of Taaffe et al. (135) and Kirk et al. (139). When examining the long term repercussions of any training program dietary control is essential. Subject’s diet in this instance may explain the loss or gain of fat free mass or overall body weight, but without any assessment of diet any result may be difficult to interpret. Other major limiting factors inherent to this research which have been overlooked by many researchers are environmental factors such as climate, social behaviors linked to time of year, and circadian rhymes. Long term training studies lasting 6 months in duration are likely to begin when it is either cold or warm outside (winter or summer), and conclude during the opposite condition. Research has shown that resting metabolic rate is suppressed during heat acclimatization, and enhanced during cold acclimatization (134, 140). Without control groups to account for the normal shift in resting metabolic rate due to environmental changes one is left to wonder whether the changes observed following training are a result of the treatment or a confounding environmental factor. In conclusion, studies examining the long term effects of resistance training have demonstrated that resting metabolic rate is significantly increased, but methodological confounders reduce the impact of these findings.

Training Studies with a Dietary Component

Resistance training protocols have also been investigated for their possible interactions with diet. The majority of these studies have traditionally focused on weight loss as a major outcome variable, but resting metabolic rate is also of great interest within these studies. Caloric intake tends to be positively correlated with resting metabolic rate (141). Therefore, when a diet which induces negative caloric balance is undertaken, resting metabolic rate tends be reduced, and the impact of said diet is blunted. The research in this area has largely tested resistance
training as a means of attenuating the reducing of resting metabolic rate often observed during negative caloric balance. The following studies have all imposed at least one diet treatment on subjects while a resistance training program was undertaken.

Byner et al. (142) examined the effects of resistance training on maintenance of resting metabolic rate during extreme dieting (800 kcal/day) on obese middle aged subjects (17 female and 3 male). Over the course of this 12 week study subjects performed 10 resistance exercises (full body workout) 3 days per week. The intensity of lifting was not clearly stated, but based upon the repetition scheme (8-15reps/set) it would appear that they were using approximately 65-70% of their 1RM, and they progressed from 2 to 4 sets over the course of training. Despite having such a limited caloric intake subjects actually gained 3.6% resting metabolic rate, which when compared with fat free mass became even larger (5.3%). This result, though impressive may have been a result of their lack of stringent control during resting metabolic rate pre and post testing. Female subjects were not tested in their follicular phase (which is the norm), but where instead simple tested initially during whatever phase they were in at the time, and post tested during the same approximate phase. Though this may seem like a reasonable means of controlling for the inherent variability in resting metabolic rate due to menstrual cycle some of the females (3 of the 17) were not tested during the same cycle, so it is possible that the results were skewed by these individuals.

Similarly, Wadden et al. (143) studied the not only the effects of resistance training with a very low calorie diet protocol on middle aged females, but also endurance training and combining both resistance training with endurance training. In this study the obese subjects consumed 900 calories and exercised 2 to 3 days per week doing full body lifting workouts (1-2 sets, 10-14 reps) for resistance training portion of exercise. The endurance training consisted of
step aerobics, for 40 minutes 3 days per week. Ultimately, the 3 exercise treatments resulted in better maintenance of resting metabolic rate over this 48 week training study; with a trend of the endurance training group having the least reduction. Additionally there were no changes in fat free mass between any of the groups.

Later, Hunter et al. (144) also examined the effects of a very low caloric intake diet (800 kcal) combined with resistance training, endurance training, or no exercise. With a large subject pool of over 100 young females these researchers found that resting metabolic rate was not changed over the course of this study in the resistance training group, while both of the other groups experienced significant reductions. Within this study the endurance training group either walked or jogged on treadmills for the endurance training treatment, while the resistance training group performed workouts very similar to those in Byner et al. (142) and Wadden et al. (143), but with a greater intensity (80% of 1RM). Menses was well controlled for when resting metabolic rate was assessed as all testing occurred during the follicular phase. The difference in resting metabolic rate retention secondary to resistance training between Hunter et al. (144) and Wadden et al. (143) may be in part due to the higher intensity used by Hunter. Additionally, the endurance training and resistance training groups’ workouts had a greater differentiation than that of Wadden, and this may have contributed more to the distinct difference in findings.

Kerksick et al. (145) conducted a diverse study of resistance training with several diet conditions including three negative caloric balance conditions (based around manipulating the percentage of carbohydrate within the diet; all 1200 kcal), one positive caloric balance condition (2700kcal), one with exercise and no diet condition, and a control condition. Subjects were obese middle aged females. There was no mention with this article of any measures being taken to control for menstrual cycle. Without controlling menstrual activity there may have been a
large degree of variability within pre and post training assessments. Their resistance training program differed greatly from previous works, as they did not have a set number of repetitions or intensity, but instead simply had their subjects complete 2 circuits of 14 full body exercises where each station was timed for 30 seconds of work. This resistance training program was likely not sufficient to elicit hypertrophy as all of the negative caloric diets experienced significant reductions in fat free mass, and the positive caloric balance diet had no change over this 14 week period. Resting metabolic rate was reduced by all negative caloric balance diets, while the high energy diet group experienced a significant increase in resting metabolic rate. With the odd resistance training program used by these researchers, and the lack of consistent measurement of resting metabolic rate this study has limited impact.

Ballor et al. (146) examined how a large reduction in body weight and subsequent resistance training program influences resting metabolic rate. The older subjects (5 female and 4 male) of this study lost an average of 9 kilograms of weight and then undertook either a 12 week resistance training or endurance training program. A significant increase in resting metabolic rate (8%) was observed as a result of the resistance training treatment, while the endurance training group experienced a reduction in resting metabolic rate. The increase in resting metabolic rate in the resistance group was linked to the increase in fat free mass as a result of their training program (3 days per week, 8 exercises, 3 sets, 8 reps, at 80% of 1RM). While subjects were in negative caloric balance prior to training their diets were not controlled during the exercise interventions. When diet was assessed post intervention they found that most subjects were in neutral or positive caloric balance. While calorie intake is positively correlated with metabolic rate typically this does not wholly account for the changes observed post exercise training. The endurance training group experienced a further decrease in resting metabolic rate.
even though they were not in negative caloric balance. This appears to be strong evidence that resistance training significantly increases resting metabolic rate following weight loss.

In contrast with many of the diet manipulation studies, Campbell et al. (147) had their older subjects (8 male, 4 female) consume a diet designed for weight maintenance, but with differing protein contents (either 0.8 or 1.6 grams per kilograms of body weight). For 14 weeks these subjects performed resistance exercise 3 days per week doing only 4 exercises (which were not compound motions) for 3 sets of 8 to 12 repetitions using approximately 80% of their 1RM. Over the course of this training period subjects’ resting metabolic rate increased by an average of 7% regardless of the protein content, but their method of performing resting metabolic rate assessment was somewhat odd. Subjects had their resting metabolic rate measured using a standard ventilation hood, but they were only 30 minutes post prandial. Though the meals were the same before and after training the possible interactions of training and thermogenesis of feeding confound these results.

Lastly, Hambre et al. (148) recently performed an unorthodox study which had young male subjects performed resistance training and either consume an additional meal of fast food (1350 kcal, 41g protein), or take a whey protein supplement (33g protein) in addition to their normal meals during the day. The resistance training program was not well defined, as subjects seemingly were told to exercise 3 days per week lifting for 1 hour per session at a local gym for 3 months. Exercise was therefore not supervised or quantified in a uniform manner. In spite of this limitation resting metabolic rate increased by approximately 9% within both groups and by about 5% relative to fat free mass. Additionally, the lack of a control group weakens this study as seasonal variations may have accounted for change in resting metabolic rate.
Studies examining the effects of combining dietary interventions with resistance training programs have demonstrated that fat free mass retention may be possible given sufficient training stimulus, though there has been a large degree of variability in diet and training protocols. During extreme caloric restriction resting metabolic rate is often suppressed, but some studies have shown that resistance training can be an effective means of attenuating this effect. During weight maintenance or weight gaining diets resistance training appears to increase resting metabolic rate secondary to fat free mass gain. The impact of these results may be limited as some studies have lacked stringent control measures or had methodological deficiencies. When control groups are not included seasonal changes may account for changes in resting metabolic rate. Many of the resistance training programs have been poorly devised or lacked adequate stimulus to elicit any change in fat free mass. Additionally, much of the research has been conducted on females, and the inherent variability of resting metabolic rate over the course of the menstrual cycle has not always been accounted for. These limiting factors detract from the strength of this research, though ultimately there does appear to be an effect of resistance training on increasing or maintaining resting metabolic rate when dietary condition are imposed.

**Future Research**

This field of study, above all else, must have more literature which has controlled for confounding factors. Far too many studies have not included control groups, had dietary control or analysis, or have failed to account for menstrual cycle. Additionally, the lack of consistency with which resistance training programs have been executed makes analysis of the resulting data difficult from a broad perspective. Therefore, researchers should adopt a more uniform means of prescribing resistance exercise, unless the resistance exercise protocol itself is a variable of
interest.

While cross-sectional research may offer limited control over any confounding factors, more studies should be done not only to compare resistance training trained subjects to untrained subjects, but to compare different modes of resistance training. For example highly trained power lifters could be compared with gymnasts, or Olympic lifters. In general research in this field has broadly classified resistance training subjects based upon their reported frequency of performing resistance exercise, but no study has included a criterion which addresses the quality of this training. Studies of this kind must adopt a physiologic criterion by which to assess how trained subjects are, and therefore more accurately gauge the effects of resistance training on resting metabolic rate.

Future training studies should adopt a more uniform means of prescribing resistance training, such as the ACSM guidelines for lifting. It is peculiar that there are guidelines which have been derived for general use by the public, yet few researchers have based their resistance training programs around them. Additionally, more work exploring the mechanisms beyond fat free mass gain, should be undertaken. While many studies have shown the increase in resting metabolic rate to be highly correlated with fat free mass there are a few researchers who have observed increases in resting metabolic rate even relative to fat free mass. Further, while fat free mass may be largely linked to increases in resting metabolic rate, it does not account for all of the variation. These additional factors may be sympathetic nervous system activity, fiber type changes, or any number of other hormonal variations. There are few studies which have addressed potential mechanisms, and therefore our knowledge is limited. Additionally, a major business associated with resistance training is supplements. There are almost no studies which have examined the effects of popular supplements on the resting metabolic rate changes.
secondary to resistance training. To gain a more robust understanding of this field of study the dietary/supplemental intake must be accounted for and researched in addition to the act of lifting alone.

**Conclusion**

Though largely confounded by limiting factors resistance training does seem to increase resting metabolic rate chronically. This increase is largely associated with the fat free mass gain secondary to resistance training, though the other possible mechanisms are under studied. Regardless of age or gender resistance training appears to positively influence resting metabolic rate, though the studies using young males seemed to benefit more so than other groups (which is likely tied to their greater capacity for anabolism) (149). Increased metabolic rate as a result of resistance training has appears to occur in as little as 3 months, and the magnitude of this increase is typically dependent upon the quantity of fat free mass gained, as shown in Table 6.1. Ultimately, the majority of chronic research has demonstrated resistance training to positively influence resting metabolic rate, though the methods of many researchers have been riddled with limiting factors which have confounded their results.

**Table 6.1:** Overview of Articles. This table outlines some of the basic parameters of the research conducted within each article reviewed. UT: Untrained, RT: Resistance Trained, Lo: Low Intensity, Med: Medium Intensity, Hi: High Intensity

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>N</th>
<th>Age Group</th>
<th>Weeks</th>
<th>RMR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poehlman et al. (117)</td>
<td>1992</td>
<td>96</td>
<td>Young</td>
<td>X</td>
<td>RT &gt; UT (13%)</td>
</tr>
<tr>
<td>Ballor et al. (118)</td>
<td>1992</td>
<td>81</td>
<td>Young</td>
<td>X</td>
<td>RT &gt; UT (6.5%)</td>
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<tr>
<td>Broeder et al. (124)</td>
<td>1992</td>
<td>22</td>
<td>Young</td>
<td>12</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Campbell et al. (147)</td>
<td>1994</td>
<td>12</td>
<td>Older</td>
<td>14</td>
<td>6.80%</td>
</tr>
<tr>
<td>Bosselaers et al. (121)</td>
<td>1994</td>
<td>10</td>
<td>Young</td>
<td>X</td>
<td>14%</td>
</tr>
<tr>
<td>Study</td>
<td>Year</td>
<td>Participants</td>
<td>Age Group</td>
<td>Sample Size</td>
<td>Effect Size</td>
</tr>
<tr>
<td>------------------------------</td>
<td>------</td>
<td>--------------</td>
<td>---------------</td>
<td>-------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Pratley et al. (125)</td>
<td>1994</td>
<td>13</td>
<td>Middle - Older</td>
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</tr>
<tr>
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<td>87</td>
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<td>UT = RT</td>
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<tr>
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</tr>
<tr>
<td>Ryan et al. (126)</td>
<td>1995</td>
<td>15</td>
<td>Middle - Older</td>
<td>16</td>
<td>Significant, but not well reported</td>
</tr>
<tr>
<td>Sale et al. (128)</td>
<td>1995</td>
<td>41</td>
<td>Middle</td>
<td>12</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Van Etten et al. (129)</td>
<td>1995</td>
<td>21</td>
<td>Young - Middle</td>
<td>12</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Taaffe et al. (135)</td>
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<td>Older</td>
<td>52</td>
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</tr>
<tr>
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<td>1997</td>
<td>26</td>
<td>Young</td>
<td>18</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Dolezal et al. (130)</td>
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<td>Young</td>
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<td>15</td>
<td>Older</td>
<td>26</td>
<td>6.80%</td>
</tr>
<tr>
<td>Byrne et al. (122)</td>
<td>2001</td>
<td>61</td>
<td>Young</td>
<td>X</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Byrne et al. (133)</td>
<td>2001</td>
<td>28</td>
<td>Middle</td>
<td>20</td>
<td>3%</td>
</tr>
<tr>
<td>Poehlman et al. (137)</td>
<td>2002</td>
<td>48</td>
<td>Young</td>
<td>26</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Dionne et al. (138)</td>
<td>2004</td>
<td>31</td>
<td>Young or Older</td>
<td>26</td>
<td>5.20%</td>
</tr>
<tr>
<td>Fatouros et al. (9)</td>
<td>2005</td>
<td>50</td>
<td>Older</td>
<td>26</td>
<td>Lo: 3%, Med: 7%, Hi: 9%</td>
</tr>
<tr>
<td>Trevisan et al. (131)</td>
<td>2007</td>
<td>30</td>
<td>Middle - Older</td>
<td>16</td>
<td>8.70%</td>
</tr>
<tr>
<td>Hunter et al. (144)</td>
<td>2008</td>
<td>103</td>
<td>Young</td>
<td>25</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Kerksick et al. (145)</td>
<td>2009</td>
<td>161</td>
<td>Middle</td>
<td>14</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Kirk et al. (139)</td>
<td>2009</td>
<td>39</td>
<td>Young</td>
<td>26</td>
<td>7%</td>
</tr>
<tr>
<td>Hambre et al. (148)</td>
<td>2012</td>
<td>24</td>
<td>Young</td>
<td>13</td>
<td>9%</td>
</tr>
</tbody>
</table>
Habitual exercise training typically stimulates physiological adaptations. The extent to which substrate utilization is impacted by regular resistance training is unclear. Typical skeletal muscle adaptations to resistance training include hypertrophy and fiber type shifts. While an increase in fat free mass is associated with increased metabolic rate, there remains debate as to how substrate utilization is affected. Shifts in fiber type from fast twitch to more slow type fibers may result in changes in resting substrate utilization, as these different fibers have greater capacities to perform either anaerobic or aerobic metabolism. Additionally, increased sympathetic nervous system activity as result of exercise has been suggested as a potential contributor to alterations in substrate utilization secondary to chronic exercise training, though there is limited evidence to substantiate this idea. Research into the effects of chronic resistance training on substrate utilization has been cross-sectional or intervention based, which provides a fairly comprehensive overview of the topic.

**Training Studies**

The research investigating chronic resistance training’s influence on resting substrate utilization is fairly scarce. Within this section all training studies have been grouped together in spite of the methodological differences which may make comparisons of their results difficult. While subsections for studies with varied aims are ideal there are too few articles to make this type of analysis productive. Rather, all research examining how substrate utilization is impacted by resistance training interventions will be reviewed together, and their commonalities will be compared.

The first training study examining how resistance training affects substrate utilization was Campbell et al. (147) in 1994. Over the course of a 14 week period older male and female
subjects performed 3 sets of 8–12 repetitions at 80% of their 1RM on 4 exercises 3 days per week. Additionally, subjects were split into one of two dietary groups. Both were isocaloric in nature, and the level of protein consumption was the variable of interest (either 0.8 grams or 1.6 grams per kilogram of bodyweight). The dietary portion of this study was not significant, as both groups had similar results. Over the course of this study, subjects’ body fat decreased by 2.2%, and they gained an average of 1.4 kg of fat free mass. Resting respiratory exchange ratio was significantly reduced (baseline: 0.9, post-training: 0.82). These findings indicate that fat oxidation was significantly increased as a result of resistance training, but these findings are questionable due to the lack of any control group to account for the environmental changes which could also contribute to a shift in substrate utilization that may have occurred over these 14 weeks. Additionally, these measurements were assessed only 30 minutes post prandial. Though both pre and post training measures were assessed in this manner the resistance training program may have impacted post feeding substrate utilization, such that these results were not entirely indicative of resting substrate utilization alterations.

In 1995 Ryan et al. (126) examined how resistance training would affect the substrate utilization of post-menopausal subjects who were grouped as either obese or normal weight. For 16 weeks these women lifted for 3 days per week (14 exercises, 3 sets using an intensity which was altered throughout each set to allow them to finish 15 repetitions). No control group was included in this study, which calls into question how much environment may have contributed to their results. Neither group experienced a change in respiratory exchange ratio. Additionally, dietary changes were not measured during the program as evidenced by the obese group losing 4.1% body fat, while the normal weight group gained 1.2%. Both groups gained fat free mass during this study (Obese: +0.5 kilogram, Normal: +1.1 kilogram). Additionally, norepinephrine
levels were assessed before and after training, but no correlations were found between substrate utilization and norepinephrine.

Again in 1995, Treuth et al. (127) investigated the effects of resistance training on older women’s substrate utilization. Subjects performed 3 sets of 12 repetitions using approximately 70% of their 1RM on 12 exercises 3 days per week over a 16 week time span. Using whole room calorimetry they observed a significant reduction in respiratory exchange ratio from pre to post training (0.9 and 0.82 respectively). Though diet was controlled through recall there was a lack of control group, which would have strengthened the impact of their findings. No significant changes in fat or fat free mass were observed over the course of this intervention.

Van Etten et al. (129) also examined substrate utilization as a result of resistance training in 1995, but used young to middle aged male subjects. This group compared how resistance training would affect substrate utilization while subjects slept. The intervention had a duration of 12 weeks, during which subjects lifted 2 days per week (14 exercises with sets a different number of sets, repetitions, and intensity for each exercise performed). Subjects experienced a loss of 2.3 kilograms of body fat, and gained 1.1 kilograms of fat free mass. There was no control group, or diet assessment throughout this study. The lack of control limits the implications of this study, but there were differences in substrate utilization observed. In general, respiratory exchange ratio was lower post training, but they noted a trend of those who initially had a low respiratory exchange ratio had an increase, while those with a high increase decreased. Another potential confounding factor present within their experimental design was their method of assessing baseline substrate utilization. Subjects had their baseline respiratory exchange ratio assessed following the first week of resistance training. Therefore, the initial values were not true baseline measurements.
In 1996 Ballor et al. (146) assessed how resistance training or endurance training would affect substrate utilization following a large reduction in body weight. Extremely obese subjects within this study first lost an average of 9 kilograms of weight. Then both male and female subjects (older in age) exercised for 12 weeks doing either a resistance training program (3 days per week, 8 exercises, 3 sets of 8 repetitions using between 50% and 80% of their 1RM) or endurance training (3 days per week, 20-60 minutes per session, at an intensity of over 50% of their \( \text{VO}_{2\text{max}} \) on treadmills). No significant differences were observed in respiratory exchange ratio or fat mass for either group, but significant differences were observed in fat free mass. The resistance training group increased fat free mass by an average of 1.5 kilograms while the endurance training group decreased fat free mass by 0.6 kilograms. Diet, however, was not controlled during the course of this study, and no control group was included to factor out environmental effects on the observed results.

Hunter et al. (136) later conducted similar research on older individuals to assess how resistance training affects substrate utilization. This study was of much greater duration than past studies (26 weeks). Both male and female subjects performed 2 sets of 10 repetitions using 65%-80% of their 1RM on 11 exercises 3 days per week. Following training subjects had an average of 3.4% less body fat, and 2 kilograms of fat free mass gained. Additionally, a significant decrease in respiratory exchange ratio was observed, signifying that resting fat oxidation was up regulated (0.86 to 0.83). Unfortunately, there was no control group or diet control during this experiment, so the changes observed in respiratory exchange ratio could be attributed to factors other than the training protocol.

In 2001 Byrne et al. (133) examined how resistance training or resistance training with the addition of walking would affect substrate utilization in obese middle aged females. Subjects
lifted regularly for 20 weeks (4 days per week, 15 exercises, 3 sets using approximately 70%-80% of their 1RM, and performed a variety of repetition schemes dependent upon the exercise), and half of the subjects walked in addition to the resistance training. Ultimately, there were no significant differences between treatments, and neither resulted in a change in respiratory exchange ratio or body fat. Fat free mass was increased by an average of 1.9 kilograms. Diet was controlled by the use of dietary logs, and they did include a control group to account for environmental factors. Neither diet nor environmental factors seemed to be significant players in the observed results. This study was the first to include control of highly pertinent confounding variables, and therefore the results are likely of greater value than those which have not.

Poehlman et al. (137) examined how endurance training or resistance training influenced substrate utilization in young females. Over the course of the 26 week training period subjects either performed 1 set of 10 repetitions using 60%-80% of their 1RM on 9 exercises 3 day per week, or an endurance training program which started as a jogging program that progressed to interval training. Following training neither treatment group experienced any change in respiratory exchange ratio or fat mass, and only the resistance training group had a significant change in fat free mass (+1.3 kilograms). Diet was not controlled, but there was a control group to account for environmental factors; though a potentially major limitation of this study was their lack of accounting for menstrual cycle. This was the first study to investigate young individuals, but unfortunately this study was not well controlled, and therefore the results are questionable.

In 2005 Fatouros et al. (9) investigated the effects of intensity of resistance training and substrate utilization in older male individuals. These subjects trained for 26 weeks then detrained for another 26 weeks. The resistance training program entailed 10 exercises being performed 3 days per week for 3 sets using either 50% (14 repetitions), 60%-65% (10
repetitions), or 80%-85% (8 repetitions) of their 1RM. Ultimately, regardless of intensity level, there were no observed changes in respiratory exchange ratio post training or following detraining. A control group was employed to account for environmental changes, but diet was not tracked. The long duration and variety within resistance training programs utilized within this study strengthen the observed results of this study. This article is strong evidence for the lack of association between resistance training and substrate utilization change.

Trevisan et al. (131) also examined substrate utilization change secondary to resistance training in menopausal subjects. Their exercise protocol lasted for 16 weeks, during which they performed 10 lifts 3 days per week. Each set consisted of 8-12 repetitions using 60%-80% of their 1RM (3 sets per exercise). A control group was included, but diet was not controlled. No significant differences were observed in respiratory exchange ratio from pre to post training. Additionally, no changes in fat mass were observed, while there was an average fat free mass gain of approximately 2 kilograms. A potential limitation of this study was their testing of substrate utilization within only a 24 hour period following last exercise. While the existence of a substrate utilization perturbation during the prolonged phase of recovery from resistance exercise may be debatable, it is possible that there could have been an effect on their post training measurements.

Lastly, Kirk et al. (139) in 2009 studied how a very minimal program of resistance training would influence substrate utilization. The young male subjects of this study performed regular resistance training for 26 weeks. The lifting program consisted of only 1 set of 3-6 repetitions using 85%-90% of their 1RM on 9 exercises 3 days per week. Using whole room indirect calorimetry, respiratory exchange ratio was measured over the course of 24 hours both before and after training. No significant differences were noted between pre and post respiratory
exchange ratio values. Environmental factors which may have contributed to a change in respiratory exchange ratio were controlled with a control group, and diet was controlled through use of dietary recall throughout the training period. No significant differences were observed in fat mass of the resistance training group, while the control group gained an average of 2.3 kilograms. Conversely, the resistance training group gained an average of 1.5 kilograms of fat free mass, while the control group did not significantly change.

The majority of training studies investigating the effects of chronic resistance training on substrate utilization have resulted in null findings. Only 3 of the 11 studies concerning this topic have reported a change in substrate utilization, all of which were reductions in respiratory exchange ratio (signifying an increase in fat metabolism). Approximately half of the studies included control groups, which is an important element to any research study examining chronic changes, as there are various environmental factors which may significantly impact these measurements. Additionally diet was generally not well controlled, which may potentially interact with substrate utilization change, as substrate intake can be linked to substrate utilization (150). These training studies have varied from 12 to 52 weeks in duration, which should be an adequate length of time to elicit some change in substrate utilization if such an adaptation existed. While some research indicates a chronic increase in fat metabolism as a result of resistance training, the studies with contradictory findings have been more well controlled, and numerous. Therefore, based upon intervention studies there does not appear to be a significant change in respiratory exchange ratio as a result of chronic resistance training.

Cross-Sectional Studies
While training studies allow researchers to control the various confounding factors inherent to experimentation and measure both before and after training to see progression, their limitation is often duration. Cross-sectional research, while flawed, does allow researchers to gain insight into the potential adaptations to long term training. Finding individuals who have performed a type of exercise for many years is not difficult, but training individuals for many years is often too costly and implausible. This type of study designs has allowed researchers to gather information about the substrate utilization patterns in those who have trained habitually for a long duration. The major limitation of this research is the lack of pre-exercise data, and control over how the exercise has been performed. Therefore, while differences may have been found it is entirely possible that those drawn to resistance training could inherently share characteristics such as eating patterns and fiber typing dispositions which may account for said differences. Cross-sectional studies are in essence a “snap shot” of individual’s current physiological condition, but this is still value in some instances.

Bosselaers et al. (121) was the first to test the substrate utilization patterns of habitual lifters. Both young male and female subjects were included in this experiment, and they had to have been lifting regularly for several years. When compared with an age matched control group who did not exercise the lifters had approximately 9 more kilograms of fat free mass, and they had nearly the same body fat. Using whole room indirect calorimetry it was determined that the resistance training group had a higher respiratory exchange ratio than did the controls (0.88 vs. 0.85). As both groups were in energy balance during their stay in the metabolic chamber the researchers ultimately concluded that the difference was either as a result of habitual diet differences or physiological adaptations.
In 1995 Toth et al. (120) assessed the substrate utilization rates of middle aged females who either performed resistance training or endurance training regularly. The criterion of inclusion to this study was solely frequency of exercise, without regard to quality of exercise. This lack of differentiation in subjects based on skill level presents a potential limitation as the groups did not seem to be different in terms of fat free mass. The resistance training group had only 2 kilograms more fat free mass than did the controls and endurance training group. Habitual resistance exercise typically promotes accumulation of fat free mass, and this minor difference could suggest that the individuals selected were not well trained. The resistance training group had significantly less body fat percentage than did the control or endurance training groups (Controls: 30%, Resistance training group: 16%, Endurance training group: 25%). Ultimately no differences in substrate utilization were found between subjects of any group, which may or may not be a function of the seeming low level of physiological development between groups.

Lastly, Byrne et al. (122) investigated how both endurance training and resistance training influenced substrate utilization, and if intensity of work made an impact. Young female subjects were studied, and the number of times per week that they exercised was used to group them into intensity levels. Neither intensity nor mode of exercise significantly impacted respiratory exchange ratio at rest. Resistance training and endurance training individuals had similar body fat percentages (21% and 19% respectively), while the control group was 33% body fat. Fat free mass was also nearly equal between endurance training and resistance training groups (48 and 49 kilograms), and significantly lower in controls (45 kilograms). The lack of difference in fat free mass between endurance trained and resistance trained groups may suggest that the resistance trained group was not well trained.
Based upon the limited number of studies examining how habitual resistance training influences substrate utilization there is not a clear understanding. Two studies have reported no significant differences, and one has found greater respiratory exchange ratio with resistance trained subjects. Though only one study has found respiratory exchange ratio to be significantly different, this was also the only study to have large differences in fat free mass between control and the trained group. Therefore, this study likely had a highly trained group of subjects, while the other two studies, which did not find significance, may not have. The increase in respiratory exchange ratio found by Bosselaers et al. (121) is puzzling as the intervention based research which found significant results has only reported decreases in respiratory exchange ratio. No mechanisms have been researched in conjunction with the substrate utilization patterns in these cross-sectional studies, so it is difficult to explain these findings. The meager number of studies examining long term resistance training and substrate utilization make conclusions impossible, but based on the available evidence there does not seem to be a consistent effect.

**Future Research**

Research in investigating the effects of chronic resistance training on substrate utilization is scarce. There are only 14 articles published which have addressed this issue using direct measurements of substrate utilization (indirect calorimetry). Though the number of studies is few, and the majority of findings have not shown a change in substrate utilization. A major failing of the current literature is the lack of variety in populations studied. Within the cross-sectional research mostly young subjects have been studied, while the intervention based studies have used older individuals, as seen in Table 7.1. More research with greater subject pools will allow for a more clear understanding of how or if resistance training influences resting substrate utilization.
Additionally, research including muscle fiber sampling, serum norepinephrine, and other potential mechanisms should be undertaken. The lack of significant findings within many of these studies could be related to exercise protocols lacking an effective stimulation. Studying some of the underlying processes common to resistance training adaptation in conjunction with substrate utilization may remove doubt as to the effectiveness the resistance training program employed, and therefore make a more compelling argument for or against its effects.

Training studies of longer duration should also be conducted. Many studies have only been of 3-4 months duration. Adaptations to resistance training, such as muscle hypertrophy, typically take much longer than this period of time to be highly significant. Additionally, cross-sectional research using highly trained subjects should be undertaken. Past studies have used frequency of workouts as the criteria for inclusion. This could explain the null results often observed within these studies, as group who are not greatly different should not show a statistically significant difference.

Conclusion

Studies examining substrate utilization as a result of habitual resistance training have largely resulted in null findings. There have been 14 studies examining this issue, and only 5 have reported a change in substrate utilization. Within the studies which have found significant results the majority have observed a decrease in respiratory exchange ratio which is indicative of increased fat oxidation. While there is some indication that resting fat oxidation may be increased as a result of resistance training the majority of studies do not support this finding. The majority of both cross-sectional (long term training) and intervention (shorter term training) studies have found no significant change in respiratory exchange ratio as a result of resistance training, and therefore substrate utilization is not considered to be greatly affected. In
conclusion, the large number of null findings compared to significant differences observed indicates that resistance training may not influence substrate utilization.


<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>N</th>
<th>Age</th>
<th>Duration</th>
<th>Findings</th>
</tr>
</thead>
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<td>Older</td>
<td>14 wks</td>
<td>Pre: 0.9, Post 0.82</td>
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<td>Young</td>
<td>X</td>
<td>RT: 0.88, C: 0.85</td>
</tr>
<tr>
<td>Ryan et al. (126)</td>
<td>1995</td>
<td>15</td>
<td>Middle - Older</td>
<td>16 wks</td>
<td>NS</td>
</tr>
<tr>
<td>Treuth et al. (127)</td>
<td>1995</td>
<td>13</td>
<td>Older</td>
<td>16 wks</td>
<td>Pre: 0.9, Post 0.82</td>
</tr>
<tr>
<td>Van Etten et al. (129)</td>
<td>1995</td>
<td>21</td>
<td>Young - Middle</td>
<td>12 wks</td>
<td>sleeping SU sig. at 1 time point (0.82 to 0.79)</td>
</tr>
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<td>Toth et al. (120)</td>
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<td>54</td>
<td>Middle</td>
<td>X</td>
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<td>1996</td>
<td>18</td>
<td>Older</td>
<td>12 wks</td>
<td>NS</td>
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<td>Older</td>
<td>26 wks</td>
<td>Pre: 0.86, Post: 0.83</td>
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<td>2001</td>
<td>19</td>
<td>Middle</td>
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<td>48</td>
<td>Young</td>
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<tr>
<td>Fatouros et al. (9)</td>
<td>2005</td>
<td>50</td>
<td>Older</td>
<td>26 wks training, 26 wks detraining</td>
<td>NS</td>
</tr>
<tr>
<td>Trevisan et al. (131)</td>
<td>2007</td>
<td>30</td>
<td>Middle - Older</td>
<td>16 wks</td>
<td>NS</td>
</tr>
<tr>
<td>Kirk et al. (139)</td>
<td>2009</td>
<td>39</td>
<td>Young</td>
<td>26 wks</td>
<td>NS</td>
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</table>
CONCLUSION

The metabolic effects of resistance exercise from both an acute and chronic perspective are not clearly understood. This lack of clarity is due in large part to the scarcity of research, and methodological issues. The primary means of assessing whole body metabolic activity is through indirect calorimetry. This technique involves measuring the volumes of gases inhaled versus those expired (carbon dioxide and oxygen); based upon both the ratio and quantities both metabolic rate and estimations of substrate utilization can be obtained. Indirect calorimetry has some irreconcilable limitations when used in during resistance exercise. Lifting is highly anaerobic, which means that a large amount of the energy used to fuel this type of exercise does not use oxygen. Carbon dioxide is a byproduct of oxidation, which is an aerobic process. Therefore, if creatine phosphate and glycolytic processes are being performed to primarily fuel an activity, breath gas analysis will not detect this. This makes measuring energy expenditure during resistance exercise inaccurate; typically leading to an underestimation of the calories burned.

Additionally, the byproducts of glycolysis/glycogenolysis can decrease pH of blood which is can be problematic from a systemic perspective. Bicarbonate stores aid in buffering these acids, which consequently makes substrate utilization immediately following resistance exercise difficult to quantify. The respiratory exchange ratio, that is the ratio of carbon dioxide to oxygen in breath gasses, is the primary means of assessing which substrates are utilized from a whole body perspective. When the bicarbonate buffers are depleted they must be replenished. The process of replenishing bicarbonate stores requires carbon dioxide. Therefore, once exercise has ceased there is typically an observed decrease in respiratory exchange ratio. Lower respiratory exchange ratios are often considered to be indicative of increased fat metabolism, but
the sequestering of carbon dioxide to replenish bicarbonate stores interferes with accurate
substrate utilization assessment. These anaerobic confounders have made the study of resistance
exercise and metabolism difficult. Some researchers have addressed these limitations, and
attempted to compensate for them, while others have ignored them. Aside from the inherent
limitations of studying metabolism immediately following resistance exercise there have been
some consistent, and some discrepant findings.

**Short Phase of Recovery**

The most studied period of post resistance exercise metabolic activity has been the short
phase of EPOC. Studies have found that following resistance exercise, as with most exercise,
there is a period of great ventilation during the first few minutes of recovery which rapidly
decreases, and then gradually subsides back to near resting state. The short phase of EPOC
typically lasts for an hour or less, but some research has observed it to be somewhat longer. The
increase in metabolic rate is often considered to be somewhat negligible, but several studies have
reported increases of more than 40% above resting for more than an hour post exercise.
Respiratory exchange ratio during this period is often observed to increase drastically, and then
decrease to below baseline values for duration similar to that of the short EPOC. This decrease
would typically be considered an indication of increase fat metabolism, but bicarbonate buffer
system replenishment confounds these findings. Consequently, substrate utilization during the
period of short EPOC remains largely unclear.

There have been some conflicting results within this field of study, which may be a result
of methodological and population differences. Most studies have utilized a scheme of sets,
repetitions, and intensity similar to those recommended by the ACSM, while others have had
some more unique protocols. Comparing resistance exercise with endurance exercise has been a
common means of assessing recovery energy expenditure effects. The majority of this research has found resistance exercise to elicit greater recovery energy expenditure, and larger reductions in respiratory exchange ratio than endurance exercise. These findings may or may not be erroneous due to the anaerobic nature of resistance exercise; a factor which has largely been ignored or unaddressed. This has led many researchers to compare unequal bouts of work, and therefore comparisons may not be equitable.

Another common means of assessing the metabolic effects of resistance exercise on short term recovery has been through the modulation of various workout parameters. Resistance exercise bouts inherently have characteristics such as the volume lifted, intensity of loading, number of sets and repetitions per set, rest interval between sets, contraction speed during each repetition, and exercise order. The manipulating of these variables has comprised much of the research into short term recovery energy expenditure and substrate utilization. A major limitation of many of these studies has been the manipulation of multiple variables simultaneously. Many studies have not controlled for the volume lifted when comparing a variable such as intensity or rest interval, and the end result is one condition performing more work than the other. This confounds any results as the protocols in some cases have been so different that the results are difficult to attribute to any single factor.

Intensity has been the most studied variable. Though the findings are not entirely consistent there is evidence that lifting with at greater intensity, relative to 1RM, elicits a greater magnitude and duration of short EPOC. This response appears to be somewhat dose responsive, as studies with a great differentiation between treatments have resulted in more pronounced responses, while those with similar intensities have had marginal differences. The study of intensity and resultant respiratory exchange ratio has produced no significant findings.
Therefore based upon the existing literature there does not appear to be a relationship between intensity of loading and respiratory exchange ratio post exercise.

Contraction speed and time under tension has also received a relatively large amount of attention. The results of these studies have been far less consistent than those of intensity modulation, and therefore conclusions are more difficult to posit. The majority of researchers have found faster contractions to result in a greater magnitude of short EPOC, while there are some which have found either no difference or the opposite. With the small number of studies examining this issue and the lack of congruent findings, no conclusions can be made at this time regarding the effects of time under tension/contraction speed on recovery energy expenditure. Respiratory exchange ratio has not been studied with respect to contraction speed modulation.

Additionally, rest interval manipulation has received very little attention. There are no consistent findings when rest interval has been the primary variable altered. Short phase recovery energy expenditure and substrate utilization as a result of resistance exercise is unclear, largely due to the lack of scientific evidence.

Exercise order and arrangement has also been investigated. Means of performing resistance exercise such as circuit training, traditional training, super-setting, and other arrangements have produced very similar recovery energy expenditure. The existing literature does not support any particular arrangement of resistance exercises or orders of execution resulting in differing metabolic results. However, one study has found a very significant effect of combining endurance exercise and resistance exercise bouts, which evoked a much larger response than separating the two (25). The effect of exercise order of execution on respiratory exchange ratio has not been studied, and therefore no conclusions can be made.
Prolonged Phase of Recovery

Beyond the first few hours post exercise there is evidence that a minor increase in energy expenditure can be detected for as long as three days. This subdued increase in energy expenditure is referred to as the prolonged phase of EPOC. Our understanding of this phenomenon is limited as there have been few studies investigating it. Similar to the study of the short phase of EPOC much of the research investigating prolonged EPOC has been descriptive in nature. There exists great discordance between the findings within this field as some researchers have detected increased energy expenditure for three days post resistance exercise, while others have not observed any increase in energy expenditure beyond the short phase of EPOC. The majority of researchers have detected a significant increase in energy expenditure when prolonged EPOC has been tested for, but the magnitudes of these responses are quite varied. Many researchers have only recorded rates of oxygen consumption, and have failed to collect full volumes. This is a major limitation of common indirect calorimetry assessments, as rates are only indicative of metabolic activity during the period assessed, therefore conclusions about the remainder one’s daily energy expenditure is assumed. Within the first 24 hours following resistance exercise there have been increases of 4%-20% in energy expenditure observed by various researchers. The majority of studies have not found energy expenditure to be increased for one day, but only a few have actually tested beyond a single time point during prolonged EPOC. The studies which have assessed post resistance exercise energy expenditure for more than the first day have observed an increase of as much as 15% at 48 hours post, and 9% at 72 hours post. No studies have found energy expenditure to be elevated beyond 72 hours post exercise. The variety of results observed is likely attributable to differences in population characteristics and protocols employed.
Though the number of studies investigating prolonged EPOC and resistance exercise is quite small there have been a few who have researched methodological factors which may modify the energy expenditure response. Rest interval between sets during resistance exercise has had some study, and the preliminary findings are that less rest may result in a greater prolonged EPOC response. Similarly lifting at a greater intensity may result in a longer duration and magnitude of EPOC. Additionally, trained subjects do not seem to have as great of a response as those who are not trained. This may suggest that through training subjects adapt such that they are more resistant to the effects of a single resistance exercise bout and this in some way blunts their energy expenditure response during.

Substrate utilization has received even less study than energy expenditure during prolonged recovery from resistance exercise. Though research is fairly sparse the majority of findings do not support the existence of a prolonged change in substrate utilization as a result of resistance exercise. The studies which have reported significant changes in substrate utilization have all found respiratory exchange ratio to decrease. These results suggest that resistance exercise may not change substrate utilization, or could increase fat metabolism. Therefore from the perspective of body composition change resistance exercise has only had neutral or positive results.

**Chronic Adaptations**

Most training studies have been effective in producing increased resting metabolic rate, even in as little as 3 months of training; though these increases are linked to fat free mass primarily. Resistance training has been proven to significantly increase fat free mass through training, which in turn tends to increase resting metabolic rate. Muscle tissue accounts for a majority of the metabolic activity within the body. If one gains fat free mass then they often
have an increased in metabolic rate on account of their having more metabolically active tissue in their body. The magnitude of resting metabolic rate gain during many studies has been between 3%-17% depending upon population, duration of training, and the methods employed. There have been studies which have not observed increases in resting metabolic rate as a result of resistance training, but confounding factors have largely been to blame. The field of chronic resistance training metabolic research has been plagued with lack of control groups, dietary control, and training protocols utilizing ineffective lifting schemes. While null results have been observed in these studies the majority of the well-designed studies have produced positive results. Cross sectional research comparing habitual lifters with age matched counterparts has largely confirmed the findings of training based studies, as those with more fat free mass tended to lift, and have greater resting metabolic rate.

Resistance training has not been conclusively proven to promote changes in resting substrate utilization. While few studies have observed declines in respiratory exchange ratio as a result of training the vast majority have found no differences when compared to their age matched controls. The seldom detected decrease in respiratory exchange ratio may mean that resistance training could result in a greater resting fat metabolism, though this finding has been observed in the minority of studies. Ultimately, resistance training appears to either increase or not affect fat metabolism, with the former being the prevalent result of most research.

**Conclusion**

With only a handful of mechanisms studied throughout all of the research investigating the metabolic effects of resistance exercise during the short, prolonged, or chronic phases, the causes of any of the observed results remain unclear. When the short and prolonged recovery research is considered in combination, the result is a fairly cohesive illustration of how resistance
exercise acutely influences metabolism. Immediately following resistance exercise caloric expenditure is greatly increased compared to resting measures. This increased metabolic rate diminishes greatly during the first few hours post exercise, but stays above baseline for up to three days. This EPOC response to resistance exercise has often been considered fairly trivial when considered for its real world implications, but when both short and prolonged EPOC phases are considered together the caloric expenditure is often of greater magnitude than the calories expended during the initial bout of work. For example in Petrofsky et al. (112) the resistance exercise bout consumed an average of 50 kilocalories to complete, but the subsequent EPOC (short and prolonged combined) resulted in approximately 350 kilocalories expenditure. These results are of somewhat greater magnitude than the norm, but based upon this study, and others like it the energy expenditure during resistance exercise recovery cannot be considered insignificant. Acute substrate utilization as a result of resistance exercise is not clearly understood. The confounding nature of anaerobic metabolism on respiratory exchange ratio makes measurements of substrate utilization during short EPOC erroneous. Further, most studies have reported no change in respiratory exchange ratio during the prolonged phase of EPOC, which suggests that resistance exercise likely does not acutely affect substrate utilization in a meaningful way.

Chronic resistance training appears to affect metabolism primarily through increasing fat free mass, which in turn increases resting metabolic rate, while substrate utilization does not appear to be significantly impacted. Many researchers have concluded that increases in resting metabolic rate as a result of resistance training are not significant. This is largely due to resting metabolic rate not changing in relation to fat free mass. For example some research has found endurance exercise to result in an increase in resting metabolic rate relatively to fat free mass,
which essentially means that per kilogram of fat free mass there is more energy expenditure per day. While this is a significant finding many of the resistance training groups have had greater absolute resting metabolic rate, which is of greater importance.

Ultimately, resistance exercise acutely and chronically results in significant alterations in energy expenditure. The increase in caloric expenditure from a single bout of work can easily make a significant impact on energy balance. When considering that many individuals lift multiple times per week they may have a prolonged recovery occurring every day, all day. The implications of resistance exercise positively affecting energy balance are therefore evident.

Further, as a result of this training habitually is an increase in fat free mass, which is in itself highly metabolic. When all phases of recovery are considered together there can be little doubt that resistance training can make a significant positive impact upon total energy expenditure on a daily basis.
APPENDIX II: IRB APPROVAL DOCUMENT

ACTION ON PROTOCOL APPROVAL REQUEST

TO: Arnold Nelson  
  Kinesiology
FROM: Dennis Landin  
  Chair, Institutional Review Board
DATE: June 19, 2014
RE: IRB# 3504
TITLE: Effects of oral vinegar ingestion on recovery in trained athletes


Review type: Full __ Expedited ____  Review date: 6/13/2014
Risk Factor: Minimal ______ Uncertain X ______ Greater Than Minimal ______

Approved ______ Disapproved ______

Approval Date: 6/13/2014  Approval Expiration Date: 6/12/2015

Re-review frequency: (annual unless otherwise stated)

Number of subjects approved: 30

LSU Proposal Number (if applicable): ______

Protocol Matches Scope of Work in Grant proposal: (if applicable) ______

By: Dennis Landin, Chairman ______

PRINCIPAL INVESTIGATOR: PLEASE READ THE FOLLOWING –
Continuing approval is CONDITIONAL on:

1. Adherence to the approved protocol, familiarity with, and adherence to the ethical standards of the Belmont Report, and LSU’s Assurance of Compliance with DHHS regulations for the protection of human subjects*.
2. Prior approval of a change in protocol, including revision of the consent documents or an increase in the number of subjects over that approved.
3. Obtaining renewed approval (or submittal of a termination report), prior to the approval expiration date, upon request by the IRB office (irrespective of when the project actually begins); notification of project termination.
4. Retention of documentation of informed consent and study records for at least 3 years after the study ends.
5. Continuing attention to the physical and psychological well-being and informed consent of the individual participants, including notification of new information that might affect consent.
6. A prompt report to the IRB of any adverse event affecting a participant potentially arising from the study.
8. SPECIAL NOTE: All investigators and support staff have access to copies of the Belmont Report, LSU’s Assurance with DHHS, DHHS (45 CFR 46) and FDA regulations governing use of human subjects, and other relevant documents in print in this office or on our World Wide Web site at http://www.lsu.edu/irb

*All investigators and support staff have access to copies of the Belmont Report, LSU’s Assurance with DHHS, DHHS (45 CFR 46) and FDA regulations governing use of human subjects, and other relevant documents in print in this office or on our World Wide Web site at http://www.lsu.edu/irb
VITA

Ryan Miskowiec, son of Michael and Mary Miskowiec, was raised in Southern California until middle school aged when his family relocated to South East Louisiana. He has since resided in Louisiana and completed a Bachelor’s of Science, a Master’s of Science, and Doctorate of Philosophy at Louisiana State University’s main campus in Baton Rouge. His professional interests gravitate toward performance enhancement and preventative medicine.

Ryan Miskowiec grew up playing several sports such as basketball, street hockey, and volleyball. In addition to outdoors activities electronic sports are a passion of his in. He has a wonderful family which includes his wife, Nadia Miskowiec, and two children (Marek and Elora).