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A Comparison of the Effects of Two Acute Resistance Training Bouts on Post Exercise Oxygen Consumption

George Joseph Abboud

FLORIDA STATE UNIVERSITY

COLLEGE OF HUMAN SCIENCES

A COMPARISON OF THE EFFECTS OF TWO ACUTE RESISTANCE

TRAINING BOUTS ON POST EXERCISE OXYGEN CONSUMPTION

By

GEORGE JOSEPH ABBOUD

A Dissertation submitted to the
Department of Nutrition, Food and Exercise Sciences
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

Degree Awarded:
Spring Semester, 2009

The members of the Committee approve this Dissertation of George Joseph Abboud defended on March 30, 2009.

_____
Lynn Panton
Professor Directing Dissertation

_____
Andy Rudd
Outside Committee Member

_____
Emily Haymes
Committee Member

Approved:

_____
Bahram Arjmandi, Chairperson, Department of Nutrition, Food and Exercise Sciences

_____
Billie Collier, Dean, College of Human Sciences

The Graduate School has verified and approved the above named committee members.

There is only one person to whom I can dedicate this study. I dedicate this to my beautiful wife Winter. She has been my rock throughout this entire process. When I embarked on this journey she was there for me in every way and has continued to encourage, support and inspire me. I asked her to leave our home and families to pursue this dream and she did. Throughout the process she has selflessly put dreams of her own on hold and for that there are not enough words to express my gratitude. When a reasonable man would ask no more, I asked her to support my foray into the military while still working on a doctoral degree and again she provided me with unconditional love and support. We arrived at Florida State University with four Abbouds and left with six. Again it is Winter's strength that allowed us to expand our beautiful family during such stressful times. I can only hope that I have the opportunity to show her as much support and strength as she has shown me. I thank you. I love you. I dedicate this to you, Winterbrier.

ACKNOWLEDGEMENTS

There is no doubt that without the support of many there would be no dissertation. First I need to thank my immediate family. Without their love and understanding I could never have done this. Thank you Winter for being you. Thank you George and Autumn for understanding when Daddy was too busy sometimes to be or play with you, but know that I wanted to. Thank you Mike and Logan for bringing me smiles every day. Thanks go out to the rest of my family. Thank you Mom for always believing in me and continuing to support my dreams into my 40's. Thank you James for being a brother I could always and still do count on in many ways. Thank you Omar and Sara for the love and support you've provided me. Thanks go out to my subjects, without them there would be no study. Thanks go out to the many students who participated in supporting roles during the data collection. Thank you Sara Chelland Campbell for all you did for me and my family. You have always been an invaluable resource and true friend to me. Thanks to Paul Henning, Beau Greer, and Derek Kingsley for being friends who've been there to lean on and laugh with. A big thanks to all my friends at SSC who've always been supportive. And finally last but definitely not least. Thank you Dr. Panton. Thank you for opening your home to me and my family. Thank you for providing me with guidance throughout this entire process. Thank you for challenging me to raise the bar higher than I would have on my own. Thank you for your patience these last few years, for continuing to believe in me and for the blazing fast turnaround times you've provided on the way to the finish line. For these things I cannot thank you enough.

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ABSTRACT

A COMPARISON OF THE EFFECTS OF TWO ACUTE RESISTANCE

TRAINING BOUTS ON POST EXERCISE OXYGEN CONSUMPTION

Although there are limited data to support significant increases in resting metabolic rate (RMR) following resistance training, recent investigations have shown excess post-exercise oxygen consumption (EPOC) to be significantly elevated above baseline for up to 72 hours in untrained and trained men. **PURPOSE:** To compare the effects of two acute bouts of resistance exercise of differing loads on EPOC. **METHODS:** Eight experienced resistance trained males (22 ± 3 yrs.) were recruited to participate in this investigation. Subjects participated in two randomized acute resistance training bouts separated by at least one week with a total volume of weight lifted of 10,000 kg and 20,000 kg. A high intensity lifting protocol was used with subjects lifting approximately 85% of their 1 repetition maximum for each of the following 4 lifts; bench press, barbell squat, barbell row and Romanian deadlift. Exercise energy expenditure and resting metabolic rate (RMR) were measured by indirect calorimetry during both exercise bouts and for 30 minutes approximately 8.5 and 1.5 hours prior to each acute bout of exercise (baseline measurements) and again approximately 12, 24, 36, and 48 hours following exercise. Creatine kinase and ratings of perceived muscle soreness were measured with all post exercise metabolic measurements and immediately prior to and post exercise. Repeated measures analysis of variance was used to analyze dependent measures. Significance was accepted at $p<0.05$. **RESULTS:** During the 20,000 kg lift subjects expended significantly ($p<0.01$) more energy (484 ± 29 kcal) than the 10,000 kg lift (247 ± 18 kcal). Twelve hour creatine kinase (1159 ± 729 U/L) was significantly elevated ($p<0.05$) from baseline (272 ± 280 U/L) and immediately post exercise (490 ± 402 U/L) following the 20,000 kg lift. No significant differences were found in RMR following exercise between the 10,000 kg and 20,000 kg lifts nor were any significant differences detected among baseline RMR and RMR over the 48 hours following either of the acute bouts of resistance exercise. **DISCUSSION:** Contrary to previously published investigations, high intensity resistance training with loads of up to 20,000 kg using experienced resistance trained males does not significantly increase EPOC above baseline RMR.

CHAPTER 1

INTRODUCTION

According to a recent report in the Journal of the American Medical Association [1] 65.7% of the adult population in the United States (U.S.) is overweight or obese, with approximately half of that population being obese and of those individuals 5.1% are morbidly obese. Sixteen percent of the children in the U.S. between the ages of 6-19 years are overweight and 31% are at risk of becoming overweight. Men between the ages of 40-59 years who experience a substantial weight gain (>10% of their body weight) are at greater risk for cardiovascular disease and diabetes [2]. Globally it has been estimated that approximately 315 million people are now considered clinically obese [3]. It has been estimated that over 300,000 U.S. deaths annually can be attributed to obesity [4, 5]. It has also been reported that there is a significant reduction in life expectancy associated with obesity [6] and if the current trends in obesity continue there will be an additional 1% increase in disability annually [7] further burdening the U.S. health care system. This problem has reached epidemic proportions and any intervention that can help to reduce the effects of weight gain and obesity needs to be examined.

According to the American College of Sports Medicine (ACSM), aerobic exercise such as walking is recommended as an intervention strategy for overweight and obese adults to lose weight and successfully maintain their weight loss [8]. The current recommendation for aerobic exercise from the ACSM is a progressive program that builds to 150 minutes of moderate intensity exercise per week for positive health benefits [8]. For weight loss or weight maintenance the Institute of Medicine (IOM) and the International Association for the Study of Obesity (IASO) recommend at least 250-300 minutes per week [9]. The ACSM suggests that beginning exercisers progress to the aforementioned level of activity as the minimum requirement to achieve health benefits associated with aerobic exercise [8]. The goal of an aerobic exercise program for overweight and obese individuals is an energy expenditure of ≥ 2000 kilocalories (kcal) per week, which is approximately 250-300 minutes per week of aerobic activity. It has been found that the higher levels of energy expenditure are associated with greater success in the maintenance of long term weight loss [10, 11]. The same ACSM position stand that offers these recommendations regarding aerobic exercise and weight loss, found there to be minimal benefits to resistance training for weight loss when compared to caloric restrictions [12-16] and that weight loss associated with aerobic training was not enhanced by resistance training [17]. The ACSM recommends using resistance training as a

supplement to aerobic exercise to enhance muscular strength and endurance, which would improve functionality and possibly lead to the adoption of a more active lifestyle [8].

Compared to energy expenditure following exercise, energy expenditure that occurs during an aerobic exercise bout has been shown to be the most important contributor to weight loss due to the increase in energy expenditure above resting metabolic levels [18-20]. However it has been shown that resistance exercise can increase energy expenditure anywhere from 1 to 48 hours after the completion of an exercise bout [21-25]. This increase in energy expenditure after exercise is termed excess post-exercise oxygen consumption (EPOC). A number of mechanisms have been attributed to EPOC such as replenishment of oxygen stores in muscle and blood, increased circulation, increased lactate clearance, replenishment of adenosine triphosphate (ATP) and creatine phosphate (CrP) stores, increased ventilation, increased heart rate, increased body temperature, increased triglyceride/fatty acid cycling, substrate utilization shifts from carbohydrates to fats, glycogen resynthesis, and increased sympathoadrenal activity [18, 19, 26-29].

EPOC and the oxygen consumed during exercise are important factors in creating a negative energy balance for weight loss. Every liter of oxygen consumed metabolically costs approximately 5 kilocalories (kcals) in energy expenditure [30]. Therefore the more oxygen used during an exercise bout and after the exercise bout, the more kcals burned. Because losing weight, in most cases, is as simple as burning more kcals per day than are consumed, negative energy balance is desirable for weight loss.

Although statistically significant increases in metabolic rate post exercise have been documented when aerobic exercise is performed at great enough intensities and/or durations, this energy expenditure is minimal in most cases, when compared to the energy spent performing the exercise [31]. When examining resistance training, most data indicate that energy expenditure is relatively low even during high intensity bouts of exercise [32-34].

The positive effects of resistance training on health and human performance have been well documented and include, reduced percentage of body fat and total body fat, increased insulin sensitivity and blood glucose tolerance in the elderly, rehabilitation of orthopedic injuries, a reduction of systolic blood pressure, increased bone mineral density, and a moderate increase in basal metabolic rate (BMR) [32, 33, 35-39]. Yet due to the relatively small amount of energy utilized, even during the most strenuous resistance workouts, this type of training has not been recommended as an effective method for controlling weight [8]. Recently researchers in the field of Exercise Physiology have begun to question the role of anaerobic exercise such as resistance training on energy expenditure and metabolism [40, 41].

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Although two recent studies have reported significant increases in EPOC up to 48 hours following an acute bout of moderate to high intensity resistance exercise in both trained and untrained subjects [23, 24], the research examining the effects of resistance training on EPOC has not clearly established the link between energy expenditure and weight training, and no research that I am aware of has specifically looked at the effects of resistance loads on EPOC in trained individuals following an acute bout of exercise.

When examining resistance training the "load" is the amount of weight actually moved or lifted, expressed in pounds or kilograms. The load can be expressed per repetition, set, exercise or total weight moved per bout. Intensity is measured as a percentage of a subject's one repetition maximal lift (1RM) [42] and the volume is based upon the number of sets and repetitions [42]. Dolezal et al. [23] and Williamson et al. [23, 24] both found resting metabolic rate (RMR) to be significantly elevated 48 hours after a resistance training bout. Dolezal et al. used a high intensity low volume protocol [23] while Williamson et al. had subjects work at a moderate intensity level and a moderate volume [24]. Both these protocols elicited significant increases in EPOC 48 hours after the acute bouts, but only the subjects used by Dolezal et al. would have increased energy expenditure enough to impact weight loss, burning approximately an additional 528 kcals and 725 kcals in their trained and untrained groups respectively over a 48 hour period [23]. The total load lifted by these subjects was approximately twice that of the subjects used by Williamson et al. [24]. This would seem to indicate that the total load lifted might have to reach a threshold in order to have a significant effect on EPOC and calorie expenditure.

Statement of the Problem

The purpose of this study was to compare the effects of two acute bouts of resistance exercise of differing loads on EPOC. The two loads chosen for this study were 10,000 and 20,000 kg. The lighter load chosen for the present study was similar to the loads that were used in the protocol by Dolezal et al. for one muscle group [23]. The lighter load in the present study also approximated the loads that are used for single muscle groups by experienced weightlifters as determined by an informal survey conducted by this researcher. The heavier load was similar to loads used by both Gillette et al. [43] and Melby et al. [22] however these studies used a lower intensity than what will be used in the present study. To try and avoid the muscle damage that was shown to increase EPOC when using heavy loads, as used by Dolezal et al. [23], trained lifters and four different exercises on four different muscle groups were used to minimize muscle damage for both of the chosen loads.

Hypotheses

Hypothesis 1

EPOC will be significantly increased above resting metabolic rate (RMR) during the resistance training bouts and at 12, 24, 36 and 48 hours following both acute bouts of resistance exercises using a 10,000 kg and a 20,000 kg load.

Hypothesis 2

VO_2 will be significantly greater following the 20,000 kg bout of exercise when compared to the 10,000 kg bout of resistance training during the exercise bouts and 12, 24, 36 and 48 hours post resistance training.

Hypothesis 3

There will be a significant correlation between subjects lifting a greater amount of weight, relative to their body weight and increases in EPOC immediately following the acute resistance training bouts and at 12, 24, 36 and 48 hours.

Assumptions

The following assumptions were applied to this study:

1. All subjects accurately reported their past and current exercise histories.

2. Prior to each acute exercise bout and throughout the course of the study all subjects followed restrictions concerning food consumption, exercise and medication.

3. All subjects adhered to the exercise restrictions following the exercise bouts of acute exercise.

4. Analysis of variance was robust to variables that are not normally distributed and had unequal variances.

5. All equipment gave reliable and valid data.

Limitations

The following limitations were applied to this study:

1. The results of this study can only be applied to the subject pool utilized.

2. The subject pool was drawn from volunteers.

3. The subject pool was limited geographically to the areas surrounding Florida State University in Northwest Florida.

Delimitations

The following delimitations were applied to this study:

1. Eight male weight lifters with a minimum of 12 months experience resistance training were recruited from Tallahassee, FL and the surrounding area.

2. The subject age range was limited to 20-29 years of age.

3. The subjects were able to move a total load of 20,000 kg in an acute resistance training bout.

4. Subjects were considered normal weight (< 23% body fat) as determined by the assessment of body composition using a 3-site skinfold measurement.

Operational Definitions

Basal Metabolic Rate (BMR) – The lowest rate of body metabolism (energy use) that can sustain life, measured after an 8 hour overnight sleep in a laboratory under optimal conditions of quiet, rest, and relaxation and a 12 hour fast [44].

Body Mass Index (BMI) – Weight in kilograms divided by height in meters squared (kg/m^2)[3, 30]

Overweight – BMI of 25-29.9 [3]

Class I Obesity – BMI of 30 – 34.9 [3]

Class II Obesity – BMI of 35.0 – 39.9 [3]

Class III Obesity – BMI $\geq$ 40 [3]

Excess Post-exercise Oxygen Consumption (EPOC) – the additional amount of oxygen consumed metabolically above resting levels after the completion of exercise. This can be translated to caloric expenditure [31].

Fast EPOC component – EPOC that occurs < 1hour after the cessation of exercise [31].

Slow EPOC component – EPOC that occurs at or beyond 1hour after the cessation of exercise [31].

Experienced Weightlifter – Having at least 12 months of lifting experience with no more than 2 weeks rest at a time and no more than a total of 4 weeks off within the last 6 months and 9 weeks off within the last 12 months

Intensity of Aerobic Exercise – Expressed as high, moderate or low dependent upon the percentage of the VO_2 max:

High intensity aerobic exercise – $\geq$ 75% VO_2 max [44]

Moderate intensity aerobic exercise – 50-74% VO_2 max [44]

Low intensity aerobic exercise – $\leq$ 49% VO_2 max [44]

Intensity of Resistance Exercise [42]:

Low intensity - $\leq$ 67% of 1RM

Moderate intensity - 68–84% of 1RM

High intensity - $\geq$ 85% of 1RM

Load – total amount of weight lifted [42].

Oxygen Uptake (VO_2) – The ability of a person to take up and use oxygen expressed in either absolutely as liters per minute (L/min) or relative to body weight as milliliters per kilogram per minute ml/kg/min [42].

Maximal Oxygen Uptake (VO_2 max) – The greatest amount of oxygen that can be used for the entire body [42].

Resting Metabolic Rate (RMR) – The body's metabolic rate early in the morning following an overnight fast and 8 hours of sleep [44].

Volume of Resistance Exercise [42]:

Low volume – 2-6 sets of $\leq$ 6 repetitions

Moderate volume – 3-6 sets of 7-12 repetitions

High volume – 2-3 sets of >12 repetitions

CHAPTER 2

LITERATURE REVIEW

During exercise there is an increase in energy expenditure. This increased energy requirement is met by an increased uptake and consumption of oxygen (VO_2). The oxygen transport system of the human body cannot initially meet the energy demands made during exercise and the energy is supplied via anaerobic pathways. When exercise is completed the rate of oxygen consumption does not immediately return to resting levels. The elevated level of VO_2, from cessation of exercise until levels return to baseline is called excess post-exercise oxygen consumption (EPOC). The main body of research regarding VO_2 and EPOC has been conducted with aerobic exercise training. Oxygen consumption is highest during aerobic exercise and oxygen consumption following exercise is dependent upon multiple factors. Recent research has indicated that a sustained and significant EPOC can occur with high intensity resistance training. Investigating the effects of this type of training and EPOC will provide a greater understanding of whether resistance training may be an effective tool for weight loss or maintenance. This review will include the following: (a) background studies on EPOC, (b) potential mechanisms of the rapid and slow components of EPOC, (c) factors that affect the magnitude of EPOC, (d) and the effects of resistance training of different intensities and loads on EPOC.

Background

It is widely accepted that the first report of an increase in resting metabolic rate (RMR) following physical activity was by Benedict and Carpenter in 1910 [45]. They reported an 11.1% increase in RMR for 7-13 hours following "severe" exercise in their subjects. The problem with this study and many subsequent studies was their lack of controls and limited subject numbers. Many researchers initially neglected to report factors important to RMR like, exercise intensity or duration, diet, prior exercise history, time of testing, caffeine intake or temperature. These investigators did lay down the groundwork for Hill et al.'s oxygen debt hypotheses [46-49], which is the basis upon which all EPOC work is built upon.

Hill et al. [46-49] hypothesized that the increased RMR was a form of physiological repayment for the oxygen debt incurred at the onset of exercise. It was proposed that the oxygen debt was caused by the removal/oxidation of lactate. Margaria [50] refined the oxygen debt hypothesis by breaking down the payment schedule into two parts; a rapid (< 1 hour) "alactacid" phase and a slow ($\geq$ 1 hour) "lactacid" phase. Their assumptions were based upon the observations that blood lactate did not decline until after the rapid phase. Therefore the rapid phase must be

"alactacid" (not involving lactate) in nature. They ascribed the rapid component to Lundsgaard's [51] newly discovered phosphagens. The phosphagens proved to be ATP (adenosine triphosphate) and CP (creatine phosphate), which do indeed play a role in the rapid EPOC component. These phosphagens participate in a reversible biochemical process that provides most of the energy required for anaerobic physical exertion [44].

As research continued it became clearer that lactic acid, though present during muscle contraction and recovery, was not the main reason for the increase in energy expenditure post exercise. In a comprehensive review Gaesser and Brooks [52] explained in depth why oxygen debt was a misnomer and how lactic acid removal was not linked to oxygen consumption post-exercise either temporally or causally. Potentially more misleading is the fact that many early studies were done on amphibian muscle [53-55]. This poses a distinct problem because lactic acid removal is highly variable across species [52, 56-58]. Due to the extensive work of Brooks et al. [59-62] regarding metabolism and oxygen consumption following exercise and the lack of causality associated with the term, EPOC has now come to be the nomenclature used when identifying oxygen consumption following a bout of physical activity.

Rapid Component of EPOC

When examining EPOC it is important to understand the two components and the mechanisms involved with EPOC. The two components are the rapid and slow components. The rapid component is the EPOC observed in the first hour after exercise is completed and the slow component of EPOC is observed after the first hour. It is not the passage of time that delineates the components but the mechanisms behind each component which exhibit more differences than similarities. The rapid component will be presented first as the mechanisms are better understood. The mechanisms currently identified for the rapid component are; replacement of oxygen stores on the hemoglobin and myoglobin, the cost of ATP/CP resynthesis, lactate removal, increased heart rate, increased body temperature and increased ventilation.

Levels of mixed venous oxygen drop after exercise begins, as do oxygen levels of myoglobin. Replenishment of oxygen following exercise is necessary in both the hemoglobin and myoglobin and has been shown to occur within the first few minutes post-exercise [63]. This is a case of a true oxygen debt. Oxygen is repaid on a loan incurred during exercise.

As previously stated, the discovery of phosphagens led to early modifications of the oxygen debt hypothesis. It has been shown that during submaximal [64] and supramaximal exercise [65] ATP and CP are reduced. Consequently there is a metabolic cost to resynthesize these

phosphagens. Again these levels are restored to resting levels within minutes of cessation of exercise [64, 65].

The original basis for the oxygen debt hypothesis was based on lactate, its removal and subsequent conversion to glycogen. Originally it was hypothesized by Hill et al. that approximately 80% of lactate produced was converted to glycogen and approximately 20% was oxidized [46]. Although lactate removal still is a component in EPOC, evidence now suggests that 55-70% is oxidized post exercise and < 20% is converted to glycogen [52, 62].

Core body temperature certainly is an important factor when examining the rapid EPOC component. Brooks et al. [59, 66] have suggested that increased core body temperature has a negative effective on the resynthesis of phosphagens, by decreasing the phosphorylative coupling abilities of the mitochondria. This would translate to an energetically costlier resynthesis of ATP and CP.

Both heart rate and rate of ventilation are significantly increased during the rapid EPOC component [19]. The additional oxygen cost of increased heart rate and maintaining the greater ventilatory rate indicate both are contributors to the rapid EPOC component.

As is seen in the next section on the slow EPOC component, most of these mechanisms that explain the fast component are also involved in explaining some of the slow component of EPOC. However, their contributions to the slow component are substantially smaller, which indicate other mechanisms are involved in explaining the slow component [19].

Slow EPOC Component

As previously stated, increased circulation, ventilation and body temperature may contribute to the slow EPOC component, but the energy consumed via these mechanisms is insignificant [19]. Two mechanisms that have been identified and substantiated as major contributors to the slow EPOC component are: (1) a shift from carbohydrates as a substrate for energy expenditure to fat and (2) Triglyceride/Fatty Acid (TG/FA) cycling.

After prolonged and high intensity endurance exercise there is a shift from carbohydrate use as a substrate to fat [18, 26, 27]. Since using fat as a substrate is metabolically more costly in terms of energy expenditure [31] than carbohydrates it has been calculated that this shift could account for 10-15% of the slow EPOC component [19]. This contribution though significant is small compared to TG/FA cycling.

Accompanying the substrate shift during prolonged exhaustive exercise is a threefold increase in the TG/FA cycle [26, 27]. During prolonged exhaustive exercise only 25% of the FAs liberated from TGs are oxidized [19] and since the only pathways for FA use are oxidation and re-

esterification, 75% of these FAs must be re-esterified which in turn has been estimated to account for approximately 50% of the slow EPOC component. These two mechanisms account for approximately 60-65% of the slow EPOC component. Other mechanisms that have been proposed to contribute to the slow component, which may be more important during resistive exercise or intermittent aerobic exercise in elevating EPOC, are increases in protein synthesis and changes in energy efficiency at the mitochondrial level during recovery from exercise.

An elevated rate of whole body protein synthesis [29, 67, 68] and breakdown [29] have been observed as well as an increased muscle protein synthesis [69] following exercise. As protein synthesis is a metabolically costly mechanism it has suggested that this may contribute to the slow EPOC component. The second mechanism that may contribute to the slow component relates to mitochondrial uncoupling proteins (UCPs). Bangsbo et al. [63], Scott et al. [70], and Scott [41] have observed an underestimation of energy expenditure using oxygen consumption as the only measurement during heavy exercise. It has been postulated by Børsheim et al. [31] that this discrepancy may be due to UCPs, specifically UCP3 that is expressed abundantly in skeletal muscle tissue in humans [71-73]. UCPs are mitochondrial transporters that disrupt the proton gradient of the inner mitochondrial membrane. In effect they "uncouple" cellular respiration from ATP production, releasing stored energy as heat [71-73]. The expression of UP3 mRNA is related to sleeping metabolic rate and thyroid hormones. When examining activity levels, acute bouts of aerobic exercise up regulate the expression of UP3s and chronic endurance training down regulates the expression of UP3s [72]. To my knowledge, no research has been conducted examining UP3 expression and resistance training.

The mechanisms discussed in both the rapid and slow EPOC components explain to some degree why EPOC is observed, but they do not address the magnitude or duration of EPOC.   There are two components of exercise that affect the magnitude and duration of EPOC and they will be examined next. These two components are exercise duration and exercise intensity [31].

Magnitude of EPOC

When examining the body of research regarding EPOC, inconsistencies are evident almost immediately. Some data show EPOC lasting for hours with a significant impact on energy expenditure, while other data indicate EPOC is minor and contributes little to energy expenditure. The conflicting data are reconciled when intensity and duration of the acute exercise bouts are taken into account.

The absence of a sustained EPOC (beyond 1 hour) following exercise is found in most studies with exercise bouts of short durations (less than 80 minutes) [74-76]. An example of this is

seen in a study by Hagberg et al., who exercised 18 subjects (20-33 yrs of age), at 50, 65 and 80% of their VO_2 max for a 5-minute and a 20-minute bout of cycling at each intensity level. There were no significant differences in EPOC from baseline 35 minutes post exercise at 50 or 65% of VO_2 max when cycling for 5 or 20 minutes nor were there any differences at 80% of VO_2 max for the 5-minute bout. However when exercising at 80% of VO_2 max for 20 minutes there were significant increases ($p < .01$) in EPOC 35 minutes post exercise from baseline measurements and from the EPOC measurement 35 minutes post exercise of the 5-minute bout [74].

In a similar study by Maresh et al. they had 8 young, healthy males (mean age 27.6 yrs) randomly perform 4 cycling protocols. Two acute bouts of exercises were performed at 60% and 70% of subject's VO_2 max for 20 and 40 minutes at each intensity level. There were no significant increases in EPOC above baseline at minute 40 post exercise, for any of the 4 protocols [76]. In another study Pacy et al. exercised trained subjects at 33-55% of their VO_2 max for the initial 20 minutes of an hour for 4 consecutive hours and saw a significant increase in oxygen consumption for the first 40 minutes post exercise compared to pre exercise measurements. Even with intermittent exercise of 20-minute bouts over a 4 hour period, no significant elevation in EPOC was seen beyond 40 minutes following the last exercise bout. It is important to note that only 4 subjects (2 male and 2 female) participated in this study [75]. The results reported by these investigators [74-76] are typical of protocols utilizing short duration exercise when examining EPOC. None of these studies [74-76] saw significant increases in EPOC beyond 60 minutes. However, subsequent studies using similar intensities for longer exercise durations (approximately 80 minutes) have observed significant increases in EPOC beyond 60 minutes [18, 77, 78]. This would indicate a threshold for exercise duration is needed to significantly increase EPOC. Although the fast EPOC components of these and other studies [74-76, 79, 80] show significant differences from baseline or resting values, none of the investigators have reported increases beyond 40 minutes. Estimated caloric expenditures elicited from protocols of shorter durations (approximately < 80 minutes) would not be high enough to positively influence weight loss. Therefore, for EPOC to have an impact on weight loss aerobic exercise needs to be completed for at least 80 minutes. The differences in the protocols sited above could explain some of the conflicting EPOC results when examining the fast component.

In contrast the following studies found that EPOC magnitude was significantly greater and of a significantly longer duration when exercise bouts were of longer duration and established that exercise duration and EPOC magnitude had a linear relationship [18, 77, 78]. The following investigations indicate that for EPOC to be significantly increased beyond 1 hour, exercise must be

performed for at least 80 minutes at an intensity of no less than 29% of VO_2 max. These data also indicate that EPOC would not positively impact upon weight loss unless exercise was sustained for approximately 80 minutes at an intensity of 70% of VO_2 max.

Maehlum et al. [77] had 8 healthy subjects (mean age 22.1 yrs) cycle at 70% of their VO_2 max for 80 minutes then intermittently recorded oxygen consumption for the next 24 hours. The mean total oxygen consumption for exercise was significantly greater ($p>0.01$) compared to an identical control session in which the subjects were rested instead of exercised. The 24-hour oxygen consumption following exercise (EPOC) was 211 ± 16 L/12 hours and 185 ± 13 L/12 hours for the control session. This translates to approximately 260 extra kcals burned in the 24 hour period following exercise when compared to the control session [77]. The values from this exercise session could in fact positively impact weight loss.

In another study Bahr et al. had 6 male subjects (mean age 22.7 yrs.) cycle at 70% of their VO_2 max on separate days for 20, 40 and 76 minutes. At all durations EPOC was higher than control measures at 12 hours post exercise. The investigators did not report whether or not these differences were significant as this research was conducted to determine if there was a linear relationship between EPOC magnitude and exercise duration. In fact there was a linear relationship observed between duration of exercise and the magnitude of EPOC [78], the greater the exercise duration the greater the magnitude of the EPOC. The caloric expenditures for the 20, 40, and 76-minute bouts were approximately 56, 74 and 160 kcal respectively over the course of 12 hours. Although EPOC from all exercise bouts in this investigation was higher than the control session, it is important to note that only the 76-minute bout would have a practical impact on weight loss. Another note of interest is that these data conflict with the investigation by Maresh et al. [76]. Maresh et al. also had healthy males (8 subjects) perform aerobic exercise on a cycle ergometer for 20 and 40-minute exercise bouts at 70% of their VO_2 max. They found no significant increases in EPOC above baseline beyond 40 minutes. The only distinct differences in protocol that might explain this discrepancy is the subjects' respective VO_2 max measurements. Maresh et al. [76] used subjects with an average VO_2 max of 46.1 ml/kg/min. while Bahr et al. [78] used subjects with an average VO_2 max of 54.1 ml/kg/min. This could explain the differing results, as the subjects in the Bahr et al. investigation would have performed a greater absolute workload at 70% of their VO_2 max than the subjects in the Maresh et al. investigation. Of greater importance was that the work by Bahr et al. [78] established time as an important factor when examining EPOC and aerobic exercise and led to work examining different intensity levels.

Bahr and Sejersted used different intensities and long duration cycling exercise to determine if there were significant differences in EPOC at increasing intensities of exercise [18]. Their work established a clear curvilinear relationship between EPOC and intensity of exercise when duration is held constant. They had 6 male subjects (mean age 23 yrs.) exercise on a cycle for 80 minutes on 3 separate occasions at 3 different intensities 29%, 50% and 75% of VO_2 max. Subjects also came to the laboratory for one control session that was identical in its time course as the experimental protocols. During the control session the subjects rested in the supine position instead of exercising and followed the same bed rest recovery protocol as the exercise sessions. There was a significant increase in EPOC for each protocol, but this increase was an exponential increase above intensities of 50% of the VO_2 max.

By examining the majority of work regarding EPOC and aerobic exercise, Børsheim and Bahr [31] found the relationship between the intensity of exercise and the magnitude of EPOC to be curvilinear. They also found that aerobic exercise has to be at a threshold intensity of approximately 50-60% of VO_2 max before a linear relationship between increasing exercise durations and EPOC magnitude is established and that an exponential increase in EPOC magnitude is seen when duration is held constant and intensity of exercise is increased [18, 31]. This would indicate a synergistic relationship between duration and intensity rather than an additive relationship [31].

Until recently most of the research regarding EPOC has been focused on aerobic exercise. Early work with resistance training did not establish this mode of exercise as an effective means of burning calories post exercise. Recently some studies have shown significant increases in EPOC up to 48 hours following an acute bout of resistance exercise. There is still a great deal of conflicting data regarding this mode of exercise, but the answer may be in the load and intensity used during a resistance training bout.

Resistance Training and EPOC

Initially, when examining resistance training and EPOC, studies compared aerobic exercise and resistance training. Most of these studies attempted to match energy expenditure, intensity of exercise, and/or duration of exercise to determine which form of training would be more costly metabolically. Some researchers compared resistance training protocols, often comparing standard weight training protocols (multiple sets of 1 exercise are completed before moving to another exercise) with circuit training protocols (1 set of all exercises are completed before an exercise is repeated) [81, 82]. The research conducted until recently utilized protocols with relatively low to moderate total loads and showed little or no sustained EPOC beyond the first few hours following

an acute bout of resistance training [21, 82-84]. New research using relatively higher loads and intensities have shown significantly greater EPOC values for as long as 48 hours following the acute bouts of resistance training [23-25].

Elliot et al. examined subjects using an acute bout of aerobic exercise and 2 different resistance training protocols [81]. Nine healthy subjects, 4 males and 5 females (age range 22-30 years) were randomly assigned to 3 different 40 minute exercise protocols and one 2-hour control session to establish a baseline RMR. Subjects were considered physically active and experienced at both resistance and aerobic training. The aerobic exercise used a cycling protocol. Subjects pedaled at 75% of their VO_2 max for the prescribed 40 minutes. The resistance training protocols used a circuit training (CT) model (50% of 1RM) and a high intensity (HI) model (80-90% of 1RM) performed on the same resistance training equipment, except free weights were used for the bench press during the high intensity model. The CT model had subjects perform 4 circuits of 15 repetitions on 8 different pieces of equipment with a maximum of 30 seconds rest between each station. The HI model used 3 sets of 3-8 repetitions on the same equipment as the CT model, except where previously noted. Repetitions were performed to volitional fatigue. Both resistance-training protocols were conducted for a total of 40 minutes. Load values were not reported. Elliot et al. [81] observed a significantly ($p< .01$) greater amount of calories burned during cycling (432 ± 95 kcal) and CT (362 ± 167 kcal) than during HI lifting (248 ± 129 kcal). However both resistance-training protocols had significantly ($p<0.05$) higher EPOC expressed in caloric expenditure (HI: 51 ± 31 kcal; CT: 48 ± 20 kcal) values than the cycling protocol (32 ± 16 kcal) for the initial 30 minutes post exercise. Elliot et al. did not report the total load used during the resistance training protocols nor did they report the 1RM values. Elliot et al. also reported using subjects that were experienced in weight training and aerobic training, but did not report what the criteria was for "experienced". Time (40 minutes) was the only variable held constant for the 3 protocols. It is important to note that this study did not control for exercise intensity or caloric expenditure for any of the protocols, nor did they control for load, sets, repetitions or rest for the resistance protocols. Although the intensity of the HI protocol was enough to elicit a response in increasing metabolic rate, the EPOC values were relatively low [81].

Gillette et al. [43] compared aerobic exercise and resistance exercise by matching caloric expenditure during the acute bouts of exercises. Seven subjects, aged 22-35 years and considered "regular" exercisers were participating in a resistance training and aerobic training program at least 2 times per week for each mode of exercise at the time of the study. These subjects underwent VO_2 max testing for the aerobic bout and 1RM testing for the exercises in the resistance bout. The

control session and the aerobic session were randomized, but the resistance bouts always preceded the aerobic bouts. In an attempt to keep the caloric cost of both bouts similar, a preliminary test was conducted. The subjects completed the resistance-training bout while VO_2 was recorded. The resistance protocol included 5 sets of 8-12 repetitions for 10 different exercises. The exercises were made up of supersets. Four-minute intervals were allowed between each superset. The intensity of the resistance protocol was set at a moderate 70% of their 1RM. Average energy expenditure was measured during the preliminary lifts. The average energy expenditure was adjusted to account for body mass and no significant differences were found. For every 500 kg lifted during the preliminary tests approximately 11.5 kcals were expended. This average was used to determine how many calories each subsequent subject burned during their resistance bout. Each aerobic bout was then conducted at 50% VO_2 max of each subject and terminated when the predetermined calories of the resistance bout were reached. A 50% VO_2 max was used to represent an intensity normally used during a relatively long bout of aerobic training.

EPOC was significantly ($p< .05$) higher following the resistance-training bout (approximately 350-475 ml/min O_2) than the aerobic bout (approximately 325-375 ml/min O_2) and the control period (approximately 305-325 ml/min O_2) for the first 1.5 hours following exercise. EPOC remained significantly ($p < .05$) higher ($6.8 \pm 7.4\%$) for the resistance-training bout at hour 5 compared the aerobic training bout ($5.5 \pm 4.7\%$) and compared to the control session. EPOC was also significantly ($p< .05$) higher the next day, 14.5 hours following exercise when compared to the control. Exact data were not reported, but graph depiction indicated that approximately 100 kcals more were burned following the resistance bout at 14.5 hours post exercise [43]. The investigators determined that the caloric value above the control session for the first 5 hours following resistance training to be approximately 51 kcal. The investigators did not measure RMR beyond 14.5 hours. A 24-hour or 48-hour measurement post exercise could have been used to determine if EPOC would have impacted weight loss. The total load lifted was very high (25,405 kg), but the intensity of exercise was a moderate 70% of the 1RM and the load was distributed over 10 different exercises. Because the intensity was moderate and the load was spread out over 9 exercises each for a different body part (sit-ups were unloaded), the EPOC may have been attenuated [43].

Burleson et al. [83] matched time and percentage of VO_2 max to compare EPOC following treadmill exercise (TM) and weight training (WT). Fifteen males, (mean $\pm$ SD; 22.7 ± 1.6 years) with at least 6 months prior experience with weight lifting completed 2 protocols; a treadmill session and a resistance session. VO_2 max testing and 1RM testing was used to determine exercise intensities. Weight training was always performed first to facilitate matching of VO_2 percentages

and the timed circuit training was used to establish a 27-minute time frame. Each subject performed as many repetitions as possible at 60% of their 1RM in a 45 second time frame for 5 exercises using resistance equipment and a predetermined number of repetitions for 3 body weight exercises. The circuit was completed twice. After a subject completed the WT, the percentage of VO_2 max for that bout was matched for the TM bout. The average intensity for the WT bout was approximately 45% of VO_2 max. Burleson et al. found that VO_2 values between the 2 protocols were not significantly different at 30 (WT: 0.414 ± 0.016 L O_2/min; TM: 0.351 ± 0.027 L O_2/min), 60 (WT: 0.321 ± 0.020 L O_2/min; TM: 0.294 ± 0.025 L O_2/min), and 90 (WT: 0.328 ± 0.019 L O_2/min; TM: 0.298 ± 0.025 L O_2/min) minutes post exercise, respectively. The total oxygen consumption during the first 30 minutes following exercise was significantly ($p < .05$) greater for WT at 19.0L than TM at 12.7 L (no SEM reported). Compared to baseline values taken prior to the exercise protocol, VO_2 for WT recovery was significantly increased at both 30 and 90 minutes while the VO_2 for the TM protocol was not significantly increased over baseline measures [83]. Values were not reported for these data. Burleson et al. did see significant increases in EPOC with weight training when compared to baseline, but only for a very short period of time. The intensity of exercise for both exercise protocols was low and the duration of exercise and the total load lifted (3000-6000 kg) was also low for the 5 different exercises [83].

Haltom et al. [85] examined circuit weight training (CWT) with variable rest periods. Seven males with a minimum of 6 months weight training experience performed 2 CWT protocols on different days. Each session was identical except for the rest between exercises. Rest between exercises was either 20 seconds or 60 seconds. Each subject completed an 8-station circuit twice using both upper and lower body exercises. Subjects performed 20 repetitions at a predetermined 75% of their 20RM. Haltom et al. observed 1 hour EPOC to be significantly greater in the 20-second rest group (10.3 ± 0.57 L) when compared to the 60-second rest group (7.4 ± 0.39L). This translated to a significantly greater caloric expenditure for the 20-second group when compared to the 60-second group. When gross energy expenditure was examined to include energy expenditure during the exercise bouts and 1 hour EPOC, the 60 second group (277.23 ± 11.36 kcal) was significantly greater than the 20 second group (242.21 ± 8.13 kcal) [85]. This difference which seems contrary to significantly greater EPOC of the 20 second rest group is due to the longer exercise rest interval during the 60 second rest protocol, as the investigators did not differentiate between exercise time and rest time for O_2 consumption. Haltom et al. [85] would have had to use a low intensity protocol to establish a 20RM and only used 75% of that value for the CWT. They did not report load values. The 1-hour EPOC values translate to approximately 51.5 kcal and 37.0 kcal

burned in that period. This is a relatively high value. The investigators did not report EPOC beyond 1 hour.

Murphy and Schwarzkopf [82] examined the effects of resistance training protocols on EPOC using CWT and a 3 set standard weight training protocol (SWT). Ten untrained college aged males, average age 23.6 ± 3.9 years completed both protocols in random order. The exercises for both protocols were the same as was the order in which they were performed. The CWT used 50% of their predetermined 1RM for each exercise. Subjects circuited through the exercises with 30 seconds rest intervals. The CWT was completed 3 times and subjects performed an average of 10 repetitions per exercise. The 3 SWT differed in the intensity of exercise (80% of their 1RM), repetitions performed (to volitional fatigue), and rest intervals (60 seconds). Total load for both protocols was similar (CWT: 5510 ± 488 kg; SWT: 5293 ± 1113 kg), but the weight lifted per unit of time was much greater during the CWT (289 ± 28 kg/min) compared to the SWT (106 ± 22 kg/min). Statistical significance was not reported for any of the data. RMR was determined prior to each exercise protocol. Murphy and Schwarzkopf found the EPOC to be significantly ($p< 0.01$) greater and the duration longer following the CWT protocol. The investigators estimated the EPOC of CWT to be 4.95 L O_2 for a period of 20 minutes and EPOC of SWT to be 2.7 L O_2 for 15 minutes (standard deviations were not reported). Although a significant EPOC was observed following both exercise protocols, the magnitude and duration would not substantially impact weight loss. This investigation did match exercises and sets and found loads to be similar, but the authors recognized that intensity of exercise, rest periods and repetitions were not controlled for. The total load used in both protocols was relatively low, especially when divided over 6 separate exercises.

Melby et al. [21] also examined the effects of a SWT protocol (3 sets; 10 repetitions; approximately 70% of the 1RM) for 7 exercises on EPOC when compared to a control condition. Six healthy males 24.5 ± 6.1 years participated in the investigation. Oxygen consumption was measured 30 minutes prior to exercise and for 60 minutes following exercise. The time reported for the lifting protocol was 42 minutes. Upon completing the exercise protocol, subjects returned for a control condition where VO_2 was measured for 30 minutes followed by 42 minutes quiet sitting and followed by measurement of VO_2 for 60 minutes. The average load used for each subject was approximately 10,044 kg. Melby et al. found VO_2 to be significantly ($p< 0.01$) higher during the 60 minutes following exercise (343.0 ± 52.6 ml O_2/min) compared to preexercise (272.2 ± 41.1 ml O_2/min) and to the 60 minutes following quiet sitting (283.5 ± 41.0 ml O_2/min) [21]. This EPOC was approximately 19 extra kcals burned in an hour. The researchers did not measure beyond 60 minutes, but EPOC was significantly ($p< .05$) higher at that time point (exact values were not

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reported). The intensity of exercise during this protocol was of moderate levels at approximately 70% of the 1RM and the load moderate for 7 exercises. This was a well-controlled study that established the groundwork for future studies where EPOC was measured beyond 60 minutes.

Melby et al. [22] also conducted a study using 2 experimental protocols. Both protocols were designed to examine the effects of acute resistive exercise on EPOC with subjects experienced (training 3-4 times/wk) in resistance exercise, although no minimum amount of experience was reported. In Experiment 1 (EX1) 7 healthy male subjects, age 22-40 years had a RMR recorded at 0700 h, a preexercise RMR measured at 1330 h (used as a baseline for EPOC measurement), and the exercise bout was performed at 1400 h. EPOC values were measured for 2 hours following exercise and again at 0700 the following day. The resistance bout for EX1 had subjects perform 6 sets of 8-12 repetitions for 10 different exercises. The intensity was moderate with subjects using 70% of their 1RM. Weight was lowered if necessary so subjects could perform the prescribed number of repetitions for the protocol. Melby et al. found that for EX1 there was a large range of weight lifted among subjects when comparing total load (15,000-38,000 kg). Two hours post exercise VO_2 in EX1 was 11.4% higher than preexercise levels (7.0 ± 1.0 L O_2). They reported RMR to be significantly ($p < .01$) elevated 15 hours post exercise when compared to the same time point on the previous day ($2,110 \pm 80$ vs. 1930 ± 70 kcal) [22]. Even though Melby et al. recorded values separated by 24 hours, the time after exercise was only 15 hours. A true 24-hour measurement comparing the same time points following exercise was not recorded. Here is another case where a 24 and 48-hour measurement of EPOC would have been beneficial in determining if this type of exercise would have a beneficial impact on weight loss. This investigation did not report markers of muscle damage or muscle soreness, which also would have been beneficial in determining potential causes of the increased metabolic rate. The extra caloric expenditure 15 hours post exercise does translate to an additional 180 kcals burned following exercise. Without load values being held constant, load cannot be examined as a variable that would impact EPOC and its effect on weight loss.

In Experiment 2 (EX2) Melby et al. [22] used 6 male subjects, aged 20-35 years with the same type of resistance training experience as in EX1, and exposed them to the same exercise protocol as in EX1 except the subjects performed 1 less set of exercise. For EX2 subjects also underwent a control session on a separate day with subjects sitting quietly instead of exercising. Melby et al. reported significantly ($p < 0.01$) elevated oxygen consumption 2 hours following exercise when compared to the control session. Again exact values were not reported. Similarly they found oxygen consumption to be elevated 15 hours following exercise (2000 ± 110 kcal) when

compared to measurements taken at the same time on the previous day (1910 ± 110 kcal). The reason given by Melby et al. [22] for performing the second experiment was to better control for confounding variables. For the second experiment researchers added a control session, increased meals from 2 meals per day to 3 (to more accurately match normal eating patterns), lowered the sets from 6 to 5 and added an extra minute to the rest intervals. The last two changes were to avoid exercise induced nausea subjects complained of during the first experiment. Despite these changes, both experiments led to the same conclusions. Oxygen consumption was significantly elevated 2 hours post exercise when compared to control conditions or preexercise levels and that this elevation was still significantly elevated 14-15 hours following the resistance exercise used in this study [22].

The results from the above studies are indicating that certain types of resistance training protocols can elicit an EPOC beyond 1 hour and even up to 15 hours. These data suggest that this increase in EPOC may have an impact on weight loss. In previous investigations the absolute load used during resistance training bouts were not held constant [21, 22, 81], except in one case where rest periods were varied [85]. Although intensities for each individual protocol were held constant, the intensities themselves were often different between the studies and between protocols within studies. None of the investigations used high intensity protocols ($\geq$ 85% of 1RM), instead most of the protocols used intensities that were between 68-84% of 1RM [21, 22, 81, 85].

Schuenke et al. [25] conducted a study holding intensity constant for subjects. In this case the calories attributed to EPOC could definitely have an impact on weight loss. Schuenke et al. [25] studied 7 subjects with an average age of 20 years who were regularly weight training (3-4 times/wk) for a minimum of 6 months. Subjects had baseline VO_2 measurements taken 1 day prior to the exercise protocol and at 0700, 1200, and 1700 hours following a 30-minute supine rest period. Time points were matched to the measurements taken post exercise. On the resistance-training day, subjects had VO_2 measurement taken at 0700 and 1200 and exercised for 30 minutes prior to the 1700 measurement instead of resting supine. The resistance protocol consisted of 3 lifts (bench press, power cleans, and squats), of which the subjects performed 4 times in a circuit routine. Each lift had a variable intensity of 80%, 70% and 75% of the subjects' 1RM for the bench press, power clean, and squat, respectively. Subjects lifted until volitional fatigue, which was approximately 9-10 repetitions/set. Weights were adjusted if necessary to keep subjects in this repetition range. Schuenke et al found VO_2 to be significantly ($p < 0.05$) elevated above baseline values immediately following, 14, 19 and 38 hours following exercise. It was also reported that mean daily VO_2 values were also significantly ($p < 0.05$) greater for both days following exercise

when compared to the baseline day. Exact data were not reported, but Schuenke et al. did calculate the mean differences in caloric expenditure for the 2 days following exercise compared to the baseline day. They found that the average number of calories burned per subject above baseline was 404 kcals and 369 kcals for the first (24 hr) and second (48 hr) day following exercise, respectively. Exercise protocols of a similar intensity have recorded 175-600 kcal burned above resting levels during exercise itself [22, 81, 83] (differences may be attributed to variations in the respective protocols). In this study by Schuenke et al. [25], EPOC values alone could have had a positive impact on weight loss, which would have been further enhanced when the energy cost of the exercise is included. Unfortunately Schuenke et al. did not report values for the loads lifted by their subjects [25].

Another two studies [23, 24] conducted prior to the work of Schuenke et al. did report load values and had significant elevations in oxygen consumption 48 hours post exercise. These two investigations indicated that an acute bout of resistance training can elevate EPOC significantly for 48 hours, that load may play an important role in elevating EPOC, and in one case cause enough extra caloric consumption to positively affect weight loss. Williamson and Kirwan had 12 untrained older subjects (aged 59-77 years) perform a moderate intensity resistance protocol and measured BMR 48 hours post exercise. Subjects performed bench presses and single leg extensions but only the concentric phase of the lifts to minimize muscle damage. A control trial and data were reported, but not the specifics of how the control trial was conducted. The intensity of the exercise was approximately 70% of the subjects' 1RM and a total of 16 sets of 10 repetitions. The investigators reported the average load for the exercises performed. For the single leg extensions 18 ± 4 kg was reported for both legs and 36 ± 7 kg was reported for the bench press. This would make the average load for each subject approximately 11,500 kg. Williamson and Kirwan reported a significant ($p<0.006$) increase in BMR 48 hours following exercise (284.0 ± 34.0 kilojoules/hr) when compared to the control (274.9 ± kilojoules/hr). Energy expenditure calculated over a 24-hour period was significantly ($p<0.002$) greater for the exercise trial (1627 ± 193 kcal/24 hours) when compared to the control trial (1570 ± 193 kcal/24 hours). This translates to approximately an extra 57 kcal burned per day on both days following exercise. Measurements were only conducted at 48 hours post exercise, but it is possible, as seen with Schuenke et al [25], that EPOC at 24 hours may have been higher than at 48 hours leading to an even greater caloric expenditure over the entire 48 hours. Although the load for this investigation would be considered moderate to heavy, the subjects performed the concentric phase of the lift only. In effect the subjects only completed half the work. The subjects in this protocol were also considerably older than the other investigations

discussed thus far. The age range for the previous studies were approximately 19-40 years [21, 22, 25, 43, 81-83, 85], whereas in Williamson and Kirwan study the age range was 59-77 years. The age of the subjects and the concentric execution of the exercise make it unclear what the effects of this exercise on EPOC would be if younger populations completing full lifts were measured. Dolezal et al. [23] also observed significant changes in oxygen consumption 48 hours post exercise, but their investigation was considerably different from that of Williamson and Kirwan.

Dolezal et al. examined whether muscle damage caused by resistive exercise would influence RMR [23]. Nine trained (RT; a minimum 2yrs experience with lower body exercise 2 days/wk) and 9 untrained (UT) subjects (mean $\pm$ SD; age = 20.7 $\pm$ 2.1 years) had baseline RMRs determined prior to the exercise protocol. After completing RMR measurements on the day of the exercise protocol, subjects warmed up on a cycle ergometer for 5 minutes, subjects performed 8 sets of leg presses at a predetermined 6RM. The investigators chose to have subjects perform the lifts with a focus on the eccentric phase of the to evoke large amounts of muscle damage and leg presses because the investigators reported this exercise to induce more muscle damage. To ensure that the untrained subjects remained truly untrained, thigh cross-sectional area and regression analysis from a separate cohort was used to establish a 6RM for their subjects. The separate cohort was able to validate using thigh cross-sectional area to predict 6RM for leg press exercises [23]. By using this methodology the untrained subjects never performed any resistance training prior to the treatment in this investigation. Baseline RMR and RMR 72 hours post exercise were not significantly different between the 2 groups, trained vs. untrained (values not reported). Both the RT and UT groups had significant ($p < .05$) increases in RMR [kilojoules (kJ)/day and kJ /kg of fat free mass (FFM)] at 24 and 48 hours post exercise when compared to baseline and each other. The UT group had an RMR of 9705.4 $\pm$ 204.5 kJ/day at 24 hours and 8930.9 $\pm$ 104.4 kJ/day at 48 hours, while the RMR for RT group was reported at 9209.3 $\pm$ 535.3 and 8601.7 $\pm$ 353.7 kJ/day at 24 and 48 hours post exercise, respectively. When values were expressed relative to subjects' FFM, the significant differences remained between groups and from baseline at both 24 and 48 hours. Exact values were not reported. The investigators did in fact observe significantly ($p< 0.05$) increased markers of muscle damage and ratings of perceived muscle soreness (RPMS) for 72 hours following exercise. Values for CK of the UT group at 24, 48, and 72 hours were 320.4 $\pm$ 20.1 U/L, 1140.3 $\pm$ 37.1 U/L and 675.9 $\pm$ 41.7 U/L, respectively. Although unreported the graphed values for the RT group showed significant CK values of approximately 200, 850, and 450 U/L for the 24, 48 and 72 hour time points, respectively compared to baseline values of approximately 100 U/L for both groups.   There were also significant differences in RPMS for the UT group at 48hr (4.4 $\pm$ 0.5) and 72 hr (1.67 $\pm$

0.5) from baseline values of approximately 0. Significant values for RT subjects of RPMS were approximately 1, 3, and 1 for the 24, 48 and 72 hours time points, respectively. When RMRs are converted to daily caloric expenditure the average number of calories burned above baseline levels during the 48 hours were approximately 725 and 530 kcal/day for the UT and RT groups, respectively [23].

This investigation and the Schuenke et al. study [23, 25] both observed significant increases in RMR above baselines values over a 48-hour period and reported EPOC values that could have a positive impact upon weight loss. In both cases the intensity of exercise was relatively high (70-85 % of 1RM), and the exercises used large muscle groups. Schuenke et al. [25] did not report load values so it cannot be determined if load played a role in that investigation. Dolezal et al. [23] did report load values and they were relatively high (approximately 7200-9700 kg), but subjects focused on the eccentric phase of the lift and were assisted with the concentric phase, which in effect had subjects moving their load only partially through a full range of motion. Although this could have potentially diminished the impact moving these loads had on subjects, these subjects did see significant elevations in resting metabolic rate. Dolezal et al. also used only 1 exercise for their protocol, which does not reflect the typical work out that is done by a resistance training population.

Summary

When examining the body of work in the area of resistance training and EPOC, early work focused on comparing aerobic exercise with resistance exercise [43, 81, 83]. Consequently the focus was on matching energy expenditure or oxygen consumption when comparing the 2 modes of exercise, with little emphasis placed on controlling load or intensity of the resistance exercise. In one case, Elliot et al. only followed the oxygen consumption of their subjects for 90 minutes post exercise [81]. And although VO_2 values were not significantly greater than baseline at 90 minutes post exercise vs. baseline values it may have been beneficial to continue measurements for 24 to 48 hours post exercise to determine if the cumulative energy expenditure was significantly greater as was the case with Gillette et al. [43]. In the study by Gillette et al., they found that at 5.5 hours post resistance exercise, VO_2 values were higher than the control session, but not significantly. However, when compared to a control session the cumulative increase in energy expenditure was significantly greater when RMR was measured 14.5 hours post exercise. This investigation used a very heavy load (approximately 25,000 kg) but no further measurements were taken beyond 14.5 hours post exercise. As investigations in the area of resistance training and EPOC continued, often load values were not reported [25, 81, 85] and some, when reported used very low loads ranging from 3000-6000 kg [82, 83]. Those studies reporting low loads did not report significant elevations

in EPOC beyond 90 minutes. Investigators that reported heavy loads (loads ranging from approximately 11,000-26,000 kg) [22-24, 43], all saw significant increases in RMR 14.5-15 hours post exercise and in 2 cases saw significant increases in RMR 48 hours post exercise [23, 24]. However, these increases may have been due in part to muscle damage as seen by Dolezal et al [23]. No studies have used a full exercise protocol using both eccentric and concentric movements with a highly trained resistance population to minimize muscle damage and if possible affect weight loss in select groups of individuals. Therefore the purpose of the present study was to examine the effects of 2 acute bouts of resistance exercise on EPOC 24 and 48 hours post exercise using a 10,000kg and 20,000 kg load divided over four different exercises to minimize muscle damage.

CHAPTER 3

RESEARCH METHODOLOGY

The purpose of this study was to compare the effects of two acute bouts of resistance exercise of differing loads on EPOC. Resistance training is a recommended strategy in weight loss programs to maintain fat-free mass, but not as a strategy for weight loss itself [8]. Recent data indicate that following high intensity bouts of resistance exercise or bouts with heavy absolute loads, EPOC may be high enough to positively impact weight loss [23, 25]. This chapter will provide information pertaining to the research methodology that was used to conduct this study; (a) subjects, (b) the exercise protocols, and (c) the statistical analyses for this study.

Subjects

Subjects were eight healthy men between the ages of 19-29 years, recruited from Florida State University and the immediate Tallahassee area. Subjects were recruited by flyers (Appendix A) posted around campus, local gyms and fitness centers, and by word of mouth. Subjects had at least 12 months of lifting experience with no more than 2 weeks rest at a time, no more than a total of 4 weeks off within the last 6 months, and/or 9 weeks off within the last 12 months. Subjects were all lean (< 17% body fat). Subjects had no prior history of illegal performance enhancing substance use. Following pre-testing, subjects were randomly assigned to the respective resistance training protocols and acted as their own controls. This study was approved by the University Institutional Review board (Appendix B).

Research Protocols

Four days to one week prior to the initiation of the first protocol subjects arrived at the laboratory to sign informed consents (Appendix C), to have height/weight measurements, to have body composition assessment, to undergo protocol familiarization, and to have one repetition maximal (1RM) testing assessment. Subjects were given a food diary log to record their diet 3 days prior to the first acute bout of exercise and for the next 48 hours after the first protocol (Appendix D). This diet was replicated for the second protocol. Subjects refrained from resistance training and high intensity aerobic exercise 72 hours prior to each acute bout of exercise and from any aerobic exercise 48 hours prior to each bout of resistance exercise. On the evening prior to their first bout of exercise subjects arrived at the laboratory at approximately 2000 hours. They had a baseline evening RMR taken and slept overnight in the laboratory. Subjects were told not to eat or drink anything after 1600 hours except water prior to the baseline RMR. Following the evening baseline RMR, subjects were offered a meal from select items provided to them. Baseline RMR

was determined the following morning prior to the exercise protocol at approximately 0600 hours. Blood was drawn following the baseline RMR measurements to determine baseline creatine kinase levels. After the blood draw and 15-30 minutes before the exercise protocol subjects were fed a Gold Balance Bar containing 22g of carbohydrates, 15g of protein and 7g of fat. Subjects were randomly assigned to perform 1 of the 2 resistance exercise protocols. Subjects returned to the laboratory for the next 2 evenings at approximately 2000 hours to have blood drawn for creatine kinase measurements and RMR measurements taken and then stayed overnight to have blood drawn for creatine kinase measurements and RMR measurements taken in the morning. Subjects were provided a similar choice of foods to eat prior to going to sleep. Subjects repeated this schedule for the next protocol starting with diet replication no sooner than 24 hours after the last RMR measurement of the first protocol (see Fig. 1).

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Diet Log	Record all meals	Record all meals	Record all meals	Record all meals	Record all meals	
RMR PM			2130	2130	2130	
RMR AM				0600	0600	0600
RPMS			2130	0700 2200	0700 2200	0700
Exercise Protocol and VO$_2$ measurement				0730		
Creatine Kinase				0700 IPE 2030	0700 2030	0700
	Day 7	Day 8	Day 9	Day 10	Day 11	Day 12
Diet Log	Replicate all meals	Replicate all meals	Replicate all meals	Replicate all meals	Replicate all meals	
RMR PM			2130	2130	2130	
RMR AM				0600	0600	0600
RPMS			2130	0700 2200	0700 2200	0700
Exercise Protocol and VO$_2$ measurement				0730		
Creatine Kinase				0700 IPE 2030	0700 2030	0700

Figure 1. Research Protocol
RMR – resting metabolic rate; RPMS – rating of perceived muscle soreness; VO$_2$ – oxygen consumption; IPE – Immediately Post Exercise.

Pretesting

Four days to one week prior to testing, subjects arrived at the laboratory and had height and weight recorded in shorts and socks on a Seca balance scale (Seca Model 707; Columbia, MD). Subjects then had body composition measured using the Lange skinfold calipers. A 3-site sum of skinfolds was used [86] and body composition was determined use the Siri equation [87]. Following body composition assessment, subjects were familiarized with the exercises for the protocols. Upon completion of the familiarization of the exercise protocol subjects had their 1RM determined for each exercise. Subjects self reported their approximate 8RM weight for each exercise. This weight was applied to a prediction table [42] to determine the first weight attempted for their 1RM. The load attempted was approximately 10 pounds lighter than that predicted to allow for a margin of error. Ten-pound increments were used in most cases until a weight was attempted that the subject could not lift. The last successful lift was recorded as the 1RM. A maximum of 3 minutes was utilized as rest intervals between lifts.

Dietary Requirements

Three days prior to and two days following the first exercise protocol subjects maintained a diet record. The diet recorded for the first protocol was repeated for the same days prior to and following the second exercise protocol. Subjects were instructed to make food choices that could be easily replicated for the second protocol.

RMR Measurements

Subjects reported to the laboratory at approximately 2000 hours following a 3-4-hour fast. Subjects underwent a 30-minute period of supine quiet rest. Immediately following that period, VO_2 samples were collected to determine RMR and respiratory exchange ratio (RER) during a 30-minute period. A TrueMax 2400 metabolic cart (Consentius Technologies, Sandy, UT) was used to collect expired air and to determine RMR, exercising VO_2 and RER. Prior to all measurements the flow meter and gas were calibrated. On completion of the RMR subjects were fed a standardized meal then slept overnight in the laboratory to ensure 7-8 hours sleep prior to baseline RMR. The standardized meal choices were a turkey sandwich, oatmeal, fruit, soup, pretzels, trail mix, Gatorade, cheese, yogurt, cereal, granola bars, milk or herbal tea. Subjects were awakened at 0545 to perform personal hygiene. Subjects walked slowly to the testing area and underwent a 30-minute period of quiet supine rest followed by 30 minutes of metabolic gas collection from the metabolic cart for the determination of RMR. Subjects were allowed a light Gold Balance Bar 15 minutes prior to the first exercise protocol.

For the exercise protocols, subjects were fitted with a mouthpiece and nose clip and metabolic measurements were taken during exercise and averaged every minute to determine

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energy expenditure and RER during exercise. Subjects returned to the laboratory for the next two evenings to have RMR measurements taken at the same times as the baseline measurements for comparison to the baseline measurements. No earlier than 24 hours following day 6 of the first protocol did the second protocol (day 7) begin and RMR measurements began again on day 9 replicating the procedures from the first protocol (see Fig. 1).

Creatine Kinase Protocol

In order to determine the extent of muscle damage via creatine kinase markers, pre and post exercise blood samples were drawn from the subjects. Immediately prior to exercise subjects had approximately 25ml of blood drawn from the antecubital vein using sterile venipuncture techniques. The blood was collected in EDTA coated tubes and centrifuged for 10 minutes. Two clear unhemolyzed serum samples were separated and stored at -80 degrees Celsius for creatine kinase analysis. This protocol was repeated in conjunction to all of the remaining RMR measurements. When all samples were collected creatine kinase analysis was conducted.

Serum samples were analyzed through a series coupled enzymatic reactions using spectrophotometry. Values are derived based on the "absorptivity micromolar extinction coefficient" of NADH at 340 nm (0.00622). A unit per liter (U/L) of CK activity is that amount of enzyme which oxidizes one μmol/L of NADH per minute.

Resistance Protocols

Subjects refrained from resistance training and high intensity aerobic training 72 hours prior to the both acute exercise bouts and any aerobic exercise 48 hours prior to the acute exercise bouts. Following RMR measurements on the exercise day subjects rated perceived muscle soreness (RPMS) of the 4 body parts trained, using a visual analog scale (VAS; Appendix E) [88, 89] then performed an acute bout of resistance training while wearing a mouthpiece and nose clip for VO_2 measurements. Each bout consisted of 4 free weight exercises performed on a non-counterbalanced Smith Machine. The exercises performed were the bench press, squat, bent-over row and Romanian deadlift. Subjects all wore lifting belts during all exercises and wrist wraps for both the Romanian deadlift and the bent over row. One protocol entailed moving a combined load of 10,000 kg and another moving a combined load of 20,000 kg. Choosing either a marked or unmarked golf ball from a shopping bag randomized the order of the protocols. The loads were divided between the 4 exercises for both protocols to minimize muscle damage as follows; 35% to squats, 30% to bench press, 20% to bent over rows and 15% to Romanian deadlift. The weight lifted for each set was set at approximately 85% of the subjects' 1RM for 6-8 repetitions. The subjects continued to perform sets until the load for each respective protocol was reached. Rest

periods were set at a minimum 2 minutes between sets. Subjects moved from one exercise to the next as soon as the investigator had properly set the equipment. RPMS was assessed following all subsequent RMR measures and reported as an average of the 4 body parts trained (see Fig. 1).

Statistical Analysis

Statistical analysis was performed using SPSS for Windows version 15.0 and 16.0 (SPSS Inc., IL). Sample size estimation was determined *a priori* as a function of the significance criteria (α), the statistical power and the effects size (ES). Effect size was calculated using the following formula:

$$ES = (\mu_{1} - \mu_{0})/S_{0}$$

Where μ_{1} was the mean of the experimental value (2201.1 kcals/day), μ_{0} was the mean of the control value (1864.2 kcals/day) and S_{0} was the larger standard deviation ($\pm$ 127.9 kcals/day) of the two means, yielding the most conservative effect size of 2.63. For this experiment an effect size of 1.1 was used, based on a relevant literature review of an acute effect of resistance training exercise on EPOC [23]. Dolezal et al. measured EPOC 24 and 48 hours following an acute bout of resistance training using relatively high loads (approximately 10,000 kg). Statistical analysis was set at an $\alpha = 0.05$, ES = 1.1 and a statistical power of .80, yielding a minimum of 7 subjects. This number was raised to 8 to accommodate for individual attrition.

Values are presented in tables as means $\pm$ standard deviations and in figures as means $\pm$ standard errors. Dependent measures include VO_2, kcal, and RER measurements for RMR before and after the two acute resistance protocols. Data were analyzed using a repeated measures analyses of variance across time to determine differences in dependent variables. If significant interactions were found a Tukey post hoc test was used to determine where the differences existed. Pearson product moment correlations were used to evaluate creatine kinase and metabolic measurements. Pearson product moment correlations were also completed on the body weight of subjects and the increase in oxygen consumption to determine if lighter subjects had experienced a greater increase relative to their body mass. The level of significance for all tests set at $p< 0.05$.

CHAPTER 4

RESULTS
Subjects

Based on the sample size calculation ten subjects were recruited for the study. Two were unable to begin the pre-testing protocols due to scheduling conflicts. Therefore eight subjects were able to begin and eventually complete the entire protocol. Subject characteristics along with 1RM, and calorie intake data (calculated from the three day dietary log) are presented in Table 1. All subjects were experienced weight lifters who had been training for a minimum of 12 months. Subjects had no more than 4 total weeks off in the past 6 months or 9 total weeks off in the past 12 months. Although some subjects had previously participated in amateur bodybuilding and/or weight lifting competitions, none were currently training for such an event.

Table 1. Subject Characteristics (N=8)

Variables	Means ± SD	Range
Age (yrs)	22 ± 3	20 – 29
Height (cm)	176.9 ± 5.0	171.0 – 185.4
Weight (kg)	88.0 ± 8.7	80.1 – 101.4
BMI (kg/m^2)	28.1 ± 2.8	22.9 – 31.5
Body Fat (%)	9.9 ± 4.1	4.6 – 16.3
Lean Body Mass (kg)	79.0 ± 6.0	68.7 – 86.4
1RM Bench Press (kg)	137 ± 16	112 – 162
1RM Barbell Squat (kg)	177 ± 43	134 – 272
1RM Romanian Deadlift (kg)	114 ± 24	67 – 135
1RM Barbell Row (kg)	142 ± 34	95 – 193
Caloric intake (kilocalories)	2281 ± 462	1532 – 2791
Protein Intake (grams)	141.5 ± 46.8	79.4 -195.3
Percentage of diet from protein	24.5 ± 5.9	18.4 – 32.5

BMI = Body Mass Index; 1RM = One Repetition Maximum

Metabolic Measurements

The lighter exercise bout had subjects lifting a total of 10,000 kg divided over four exercises (squats, bench press, bent over rows and Romanian deadlifts). The heavier exercise bout had the subjects lifting 20,000 kg over the same four exercises. Data for the two exercise bouts are presented in Table 2. The exercise duration was significantly longer ($p \leq 0.05$) for the 20,000 kg protocol (90.3 ± 16.1 min) compared to the 10,000 kg protocol (43.6 ± 7.9 min). Energy expended in kilocalories for each protocol was also significantly different ($p \leq 0.05$) with an average 237 more kcals expended during the 20,000 kg protocol (484 ± 29 kcal) compared to the 10,000 kg protocol (247 ± 18 kcal). As expected average oxygen consumption was significantly greater ($p \leq$ 0.01) during both the 10,000 kg (12.9 ± 1.8 ml/kg/min) and 20,000 kg (12.3 ± 1.7 ml/kg/min) lifts when compared to their respective baselines (3.3 ± 0.3 ml/kg/min vs. 3.6 ± .8 ml/kg/min). The average relative oxygen consumption and respiratory exchange ratio were not different between the two protocols. If subjects were unable to complete 6 repetitions of a lift at any point in the protocol the weight was reduced by approximately 10% for subsequent lifts. Six subjects completed the 10,000 kg lift without having to reduce their starting weights for each lift during the protocol, and conversely five subjects found it necessary to reduce the starting weight used in the 20,000 kg lift for one or more exercises in order to complete the protocol.

Table 2. Exercise Measurements for the 10,000 kg and 20,000 kg protocols (N = 8)[a]

Variables	10,000 kg lift	20,000 kg lift
Exercise Time (min)	43.6 ± 7.9*	90.4 ± 16.1
Energy Expended (kcal)	247 ± 50*	484 ± 83
Average Energy Expenditure (ml/kg/min)	12.9 ± 1.8	12.3 ± 1.8
Average Respiratory Exchange Ratio	1.03 ± 0.02	1.00 ± 0.02

Values are means ± standard deviations
[a]Metabolic measurements were collected continuously and ten second averages were averaged over the entire protocol for mean values.
* $p \leq 0.01$, significantly different from 20,000 kg lift

The results of the morning and evening metabolic measurements for the 10,000 kg and 20,000 kg protocols are presented in Tables 3 and 4, respectively. The RMR measurements

following exercise were timed matched, statistically comparing morning to morning measurements. Evening RMR measurements were also time matched and statistically compared only with other evening measurements, to account for the natural upward (circadian) drift of RMR over the course of a day, due to an increase in body temperature. All measurements were taken over a 30-minute time period. Values for absolute measurements (l/min), relative measurements (ml/kg/min) and RER were averaged over the 30-minute time period. In the case of energy expenditure, total caloric values were used for the 30-minute measurement period. Thirty minute caloric consumption measures were 36 ± 5, 36 ± 6, and 37 ± 5 kcals for morning baseline, and 24 and 48 hours after the 10,000 kg protocol, respectively. No significant differences were observed across time. There were also no significant differences in absolute or relative metabolic rates across the 48 hours (Table 4). The only measurement where significant changes were observed was between the 24 and 48-hour RER.

Table 3. A.M. Metabolic Measurements for the 10,000 kg and 20,000 kg lifts (N = 8)[a]

Lift	Variables	A.M. Baseline	24h EPOC	48h EPOC
10,000 kg	RMR (ml/kg/min)	3.3 ± 0.3	3.4 ± 0.4	3.4 ± 0.3
	RMR (L/min)	0.30 ± 0.04	0.30 ± 0.05	0.30 ± 0.04
	Energy Expenditure (kcal)	36 ± 5	36 ± 6	37 ± 5
	RER	.89 ± .10	.86 ± .04*	.89 ± .05
20,000 kg	RMR (ml/kg/min)	3.5 ± 0.8	3.4 ± 0.3	3.5 ± 0.6
	RMR (L/min)	0.32 ± .09	0.30 ± .05	0.31 ± .05
	Energy Expenditure (kcal)	39 ± 11	36 ± 5	38 ± 6
	RER	.90 ± .11	.87 ± .08*	.90 ± .08

Values are means ± standard deviations
[a]Metabolic measurements were collected continuously and ten second averages were averaged over the entire protocol for mean values
[b]Energy expenditure in kcals were collected continuously are expressed as total kcals burned over 30 minutes.
* $p \leq 0.05$, significantly different from 48h EPOC
RMR = resting metabolic rate
kcal = kilocalorie
RER = respiratory exchange ratio

Evening measurements are presented in Table 4. As expected metabolic measurements recorded in the evening were higher than morning values although surprisingly not significantly different. As with 10,000 kg lift the only significant difference observed following the 20,000 kg lift was an increase in morning RER measurements. The significant increase occurred between the 24 hour and the 48 hour time points (.87 ± .08 and .90 ± .08, respectively).

Table 4. P.M. Metabolic Measurements for the 10,000 kg and 20,000 kg lifts (N = 8)[a]

Lift	Variables	P.M. Baseline	12h EPOC	36h EPOC
10,000 kg	RMR (ml/kg/min)	3.7 ± 0.4	3.8 ± 0.6	3.9 ± 0.6
	RMR (L/min)	0.33 ± 0.05	0.33 ± 0.07	0.34 ± 0.04
	RMR (kcal)	40 ± 5	41 ± 8	42 ± 8
	RER	.89 ± .10	.87 ± .10	.87 ± .09
20,000 kg	RMR (ml/kg/min)	3.6 ± 0.4	3.7 ± 0.4	3.8 ± 0.6
	RMR (L/min)	0.32 ± 0.06	0.33 ± .05	0.34 ± 0.07
	RMR (kcal)	39 ± 7	40 ± 6	42 ± 8
	RER	.86 ± .11	.88 ± .11	.88 ± .11

Values are means ± standard deviations
[a]Metabolic measurements were collected continuously and ten second averages were averaged over the entire protocol for mean values
[b]Energy expenditure in kcals were collected continuously are expressed as total kcals burned over 30 minutes.
RMR = resting metabolic rate
ml/kg/min = milliliters of oxygen consumed per kilogram body weight per minute
L/min = liters of oxygen consumed per minute
kcal = kilocalorie
RER = respiratory exchange ratio

No significant correlations were found between the measurements of metabolic rate at the different time points and body weight. Results between body weight and VO_2 are shown in Table 5.

Table 5. Correlations of Body Weight and VO_2

Time Points	10,000 kg lift	20,000 kg lift
EPOC Hour 12	r = + 0.17	r = + 0.03
EPOC Hour 24	r = + 0.63	r = + 0.38
EPOC Hour 36	r = + 0.06	r = + 0.52
EPOC Hour 46	r = + 0.25	r = - 0.13

EPOC = Excess Post-exercise Oxygen Consumption

Muscle Damage and Muscle Soreness

Following the 10,000 kg protocol subjects experienced no significant increases in their mean serum creatine kinase (CK) from baseline values nor in their ratings of perceived muscle soreness (RPMS). RPMS were not measured immediately post exercise as this measurement is indicative of delayed onset muscle soreness or DOMS, which would not be measurable immediately following exercise. All CK and RPMS measurements were repeated prior to and following the 20,000 kg lift. These results are presented in Table 6 and Figures 2-5.

Figures 2 and 3 show that there were no significant differences in serum CK levels or RPMS respectively for the 10, 000 kg protocol. For the 20,000 kg protocol the 12-hour measurement of CK (1159 ± 729 U/L) was significantly higher than the baseline (272 ± 280 U/L) and immediately post exercise (490 ± 402 U/L) measurements are shown in Figure 4. Despite the significant differences in CK levels, no significant differences were observed when measuring muscle soreness (RPMS) using the VAS as shown in Figure 5.

Table 6. Muscle Damage Measurements for the 10,000 and 20,000 kg protocol (N = 8)[a]

Lift	Variable	A.M. Baseline	Immediately Post Exercise	12h Post Exercise	24h Post Exercise	36h Post Exercise	48h Post Exercise
10,000 kg	Creatine Kinase (U/L)	309 ± 295	398 ± 344	729 ± 524	561 ± 400	492 ± 326	330 ± 189
	RPMS	.2 ± .2	N/A	.3 ± .4	.5 ± .7	.5 ± .4	.3 ± .2
20,000 kg	Creatine Kinase (U/L)	272 ± 280	490 ± 402	1159 ± 729*	981 ± 653	774 ± 588	506 ± 357
	RPMS	.3 ± .3	N/A	1.2 ± 1.3	1.2 ± 1.5	2.1 ± 2.2	1.5 ± 1.8

Values are means ± standard deviations
[a] Measurements were taken via blood samples immediately following resting metabolic rate measurements to determine creatine kinase levels
RPMS = ratings of perceived muscle soreness measured by the visual analog scale
* $p \leq 0.05$, significantly different from baseline and immediately post exercise measurements
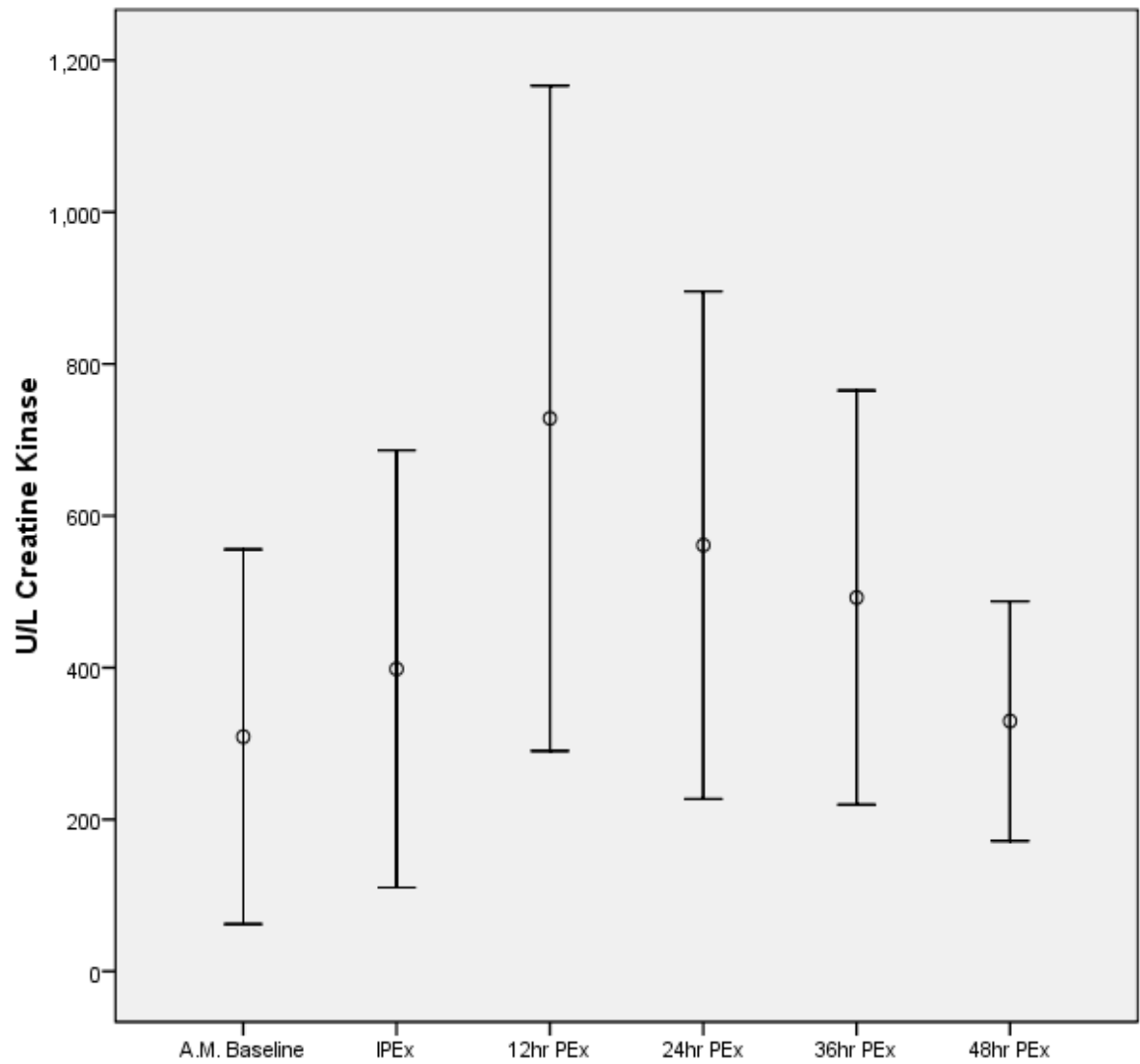
Figure 2. Creatine Kinase Measurements for the 10,000kg Lift (Mean ± SE)
U/L = units per liter; IPEx = Immediately Post-exercise; PEx = Post-exercise
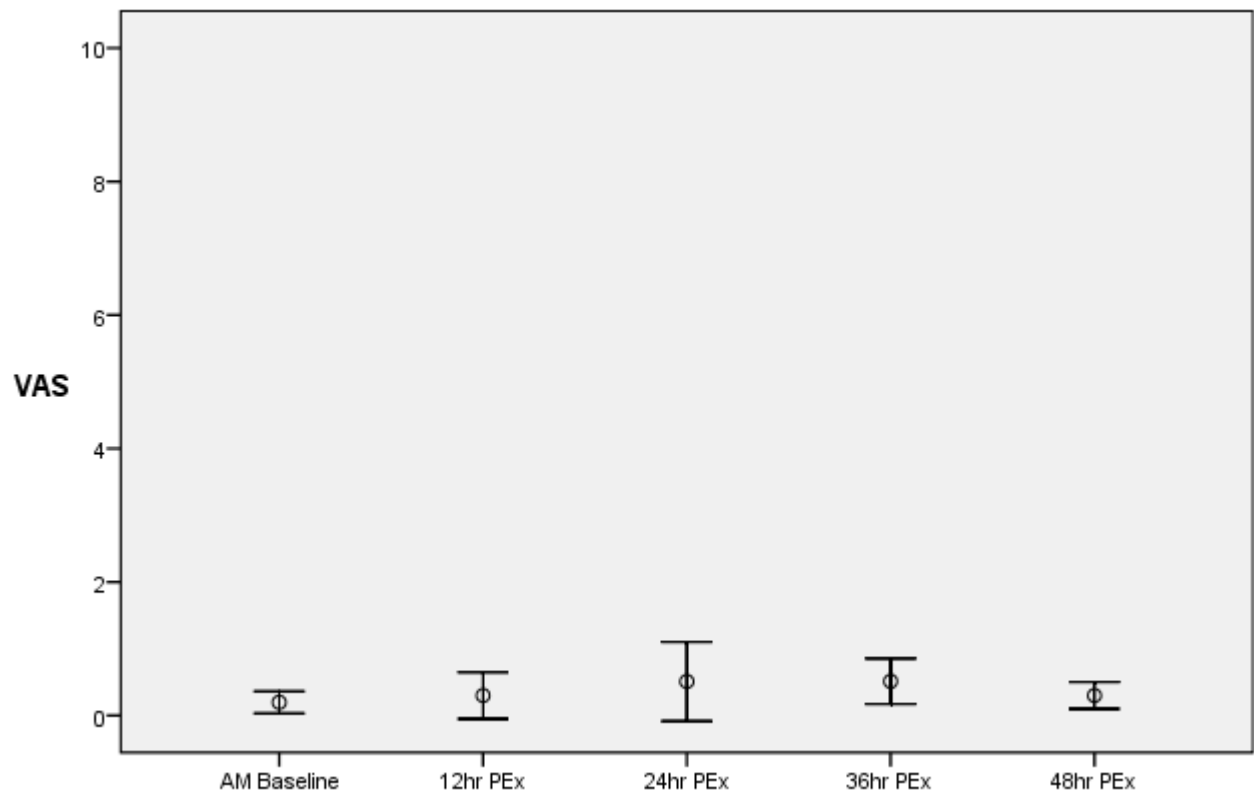
Figure 3. Ratings of Perceived Muscle Soreness 10,000 kg Lift (Mean ± SE)
VAS = Visual Analog Scale; IPEx = Immediately Post-exercise; PEx = Post-exercise
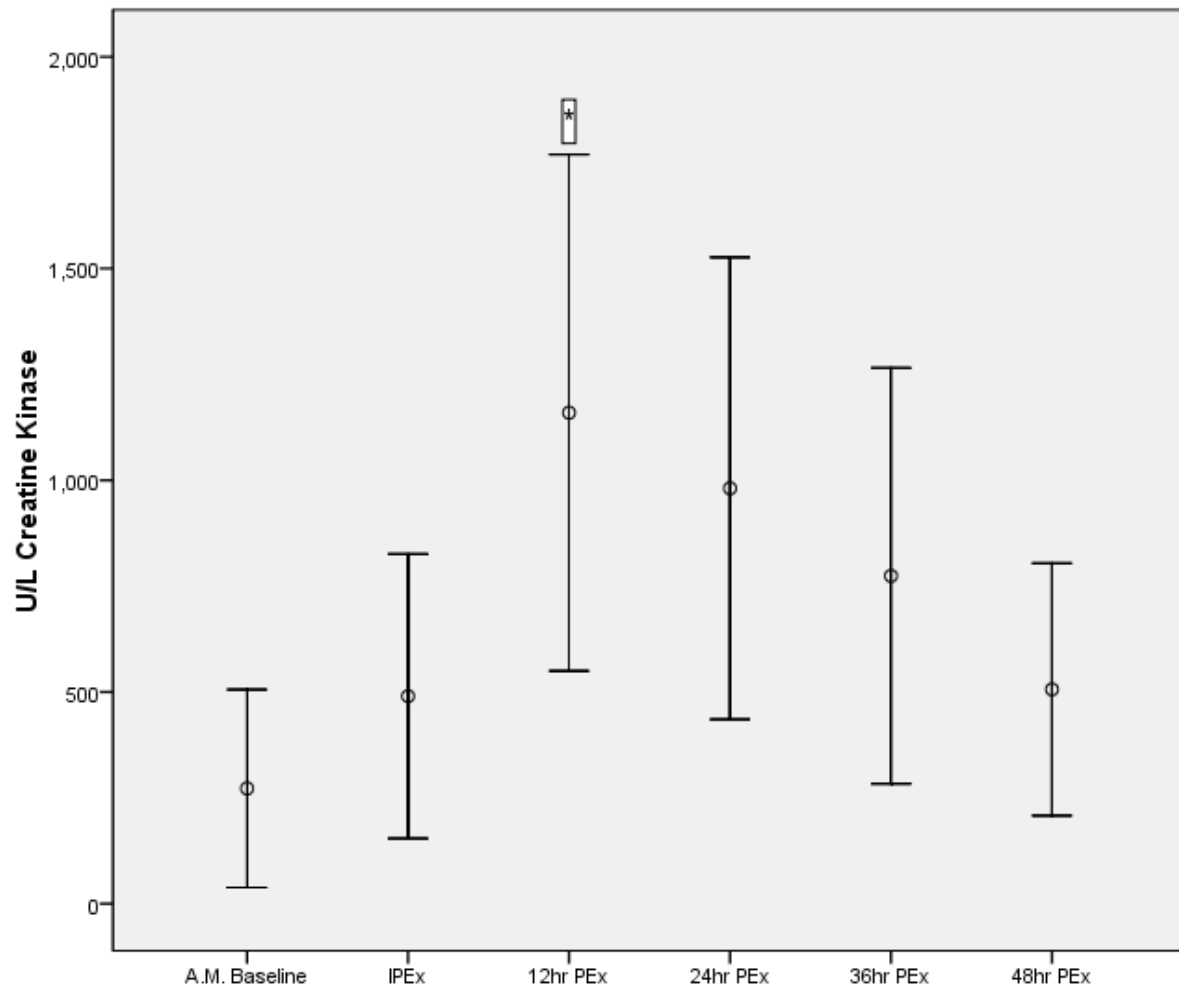
Figure 4. Creatine Kinase Measurements for the 20,000 kg Lift (Mean ± SE)
U/L = units per liter; IPEx = Immediately Post-exercise; PEx = Post-exercise
* $p \leq .05$, significantly different from A.M. Baseline and IPEx
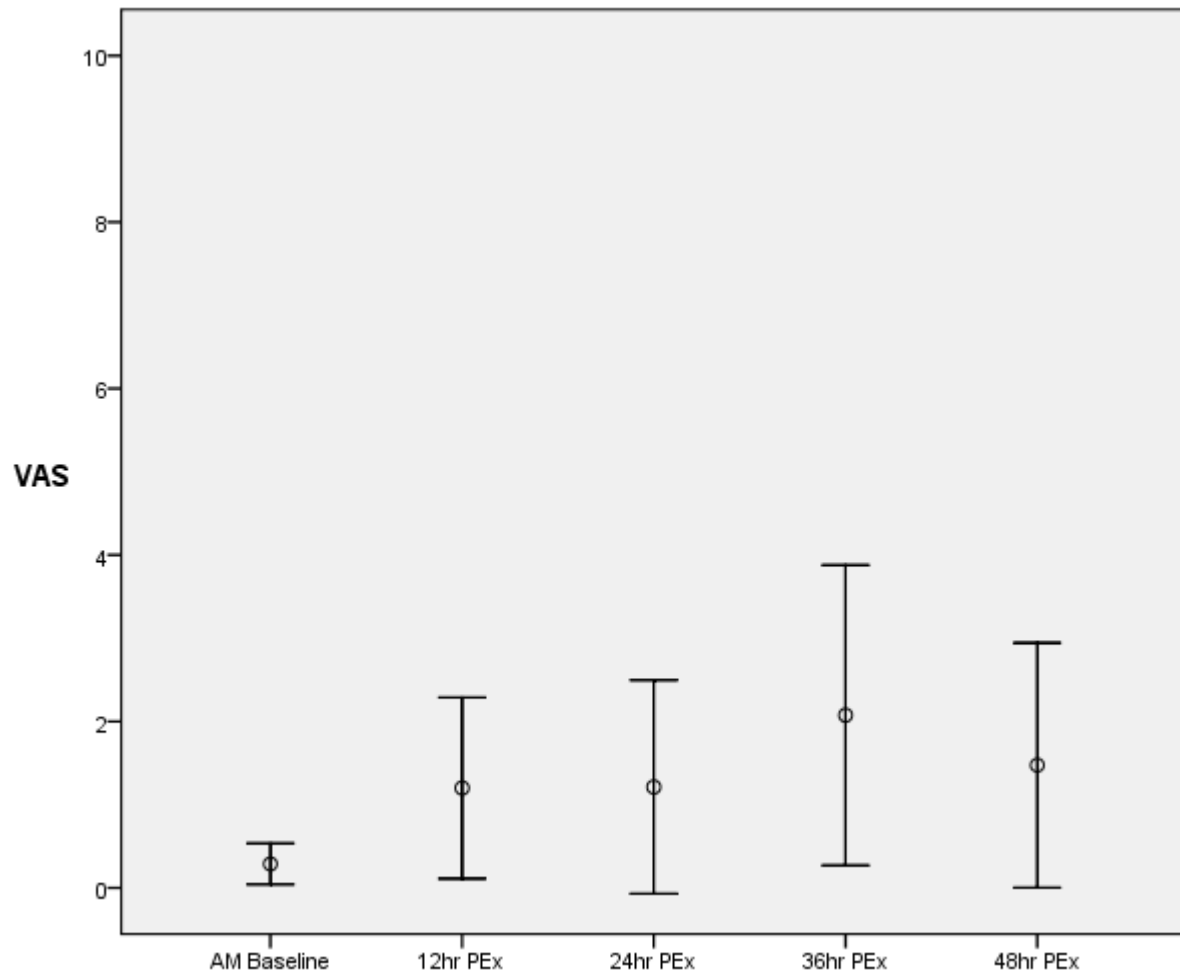
Figure 5. Ratings of Perceived Muscle Soreness 20,000 kg Lift (Mean ± SE)
VAS = Visual Analog Scale; IPEx = Immediately Post-exercise; PEx = Post-exercise

When correlations between creatine kinase levels and metabolic rates at 24 and 48 hours following the 20,000 kg bout were run, no significant correlations were observed between any time points with the 20,000 kg lift. Surprisingly, when correlations between creatine kinase levels and 24 and 48-hour metabolic measurements following the 10,000 kg lift were run, a significant correlation ($p \leq 0.05$) was observed at the 48 hr time point. The relationship of VO_2 and creatine kinase at 48 hours post exercise (r=0 .76) is shown in Figure 6.
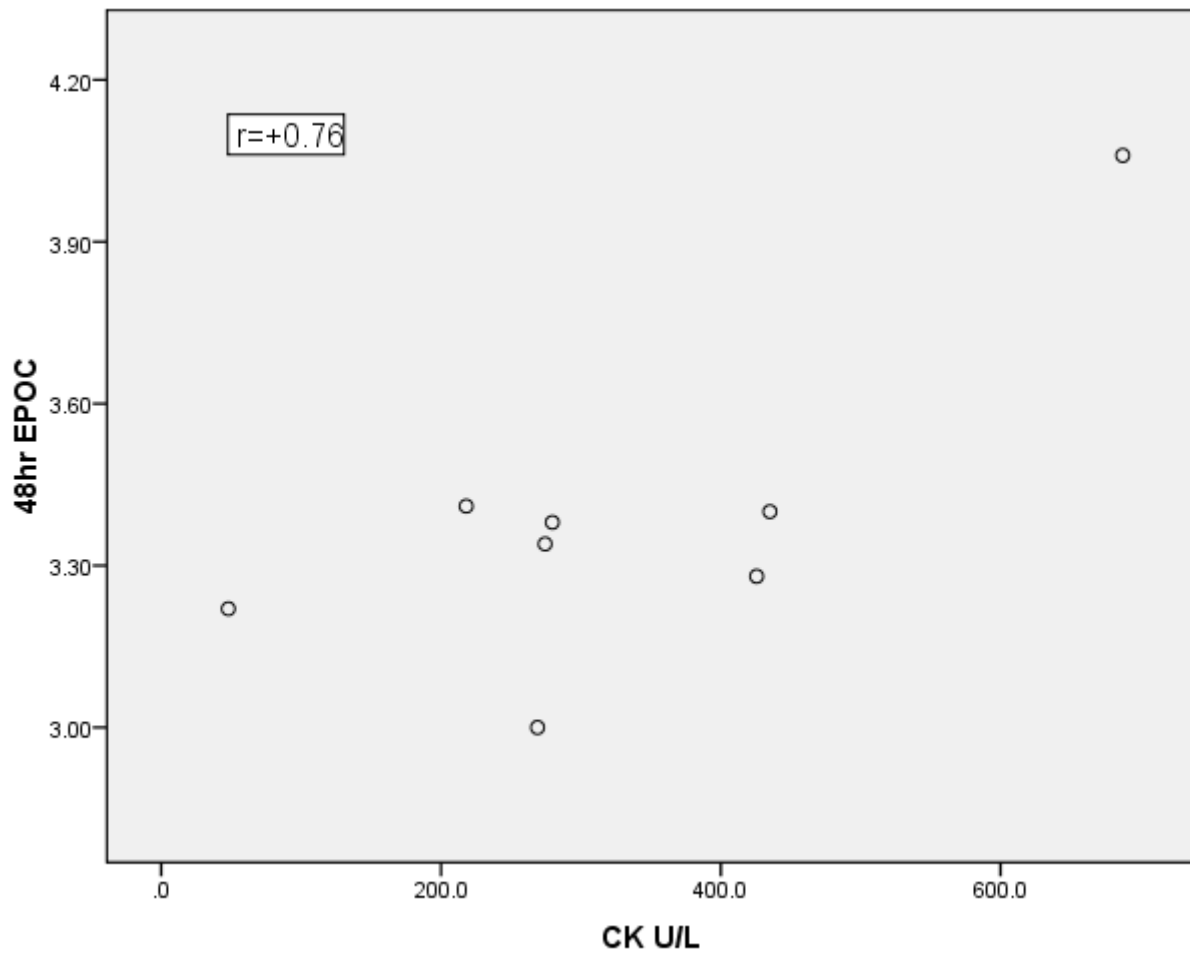
Figure 6. Correlation between Creatine Kinase and RMR 48 hours post10,000 kg lift
EPOC – excess post-exercise oxygen consumption; CK= creatine kinase; U/L=units per liter

CHAPTER 5

DISCUSSION

Initial research involving resistance training and EPOC focused on comparisons between aerobic exercise and resistance training [43, 81, 83] and resistance exercise was always shown to have significantly greater EPOC in each case. Durations of EPOC measured in these studies did not exceed 5 hours. This led researchers to investigate the effects of resistance training on EPOC exclusively. Investigations evaluating resistance training and EPOC manipulated rest periods during resistance training using either circuit training protocols [85] or compared circuit training with standard weight training (SWT) protocols [81, 82]. Still data collection only occurred for short periods of time after the exercise protocols with durations ranging from 20 to 60 minutes [81, 82, 85]. Although interesting, these studies did not answer the question as to whether the energy expenditure during and after resistance exercise could influence weight loss.

Melby et al. [21, 22] then conducted two investigations where the volume of the resistance protocols were set higher (loads ranging from approximately 10,000 kg to 38,000 kg) and intensities of 70% of subjects' 1RMs were used. These two studies focused solely on SWT protocols and extended the time in which metabolic measurements were taken. Significant increases in resting energy expenditure were recorded 15 hours post exercise [22]. The length of the increased metabolic rate and the extra caloric expenditure (approximately 180 kcals) indicated for the first time that resistance training could influence weight loss.

The earliest investigation to show increased energy expenditure above baseline and beyond 24 hours using resistance exercise occurred in 1997 by Williamson and Kirwan [24] and was supported by subsequent research [23, 25, 90]. The study by Williamson and Kirwan used relatively low loads in older male subjects and had significant but relatively low increases in energy expenditure after the exercise bout. Although their results (approximately 57 kcals/day) do not indicate that resistance training could influence weight loss, they did in fact show that EPOC could be significantly higher up to 48 hours post exercise. The subsequent research in this area [23, 25, 90] used much greater loads and higher intensities than previous research and in some cases elicited a great deal of muscle damage in their subjects [23, 90]. Muscle damage can lead to an increase in protein synthesis which has been found to be metabolically expensive [29, 91]. These studies do indicate that resistance training could influence weight loss.

Based on past studies it is now pretty well accepted that following an acute resistance training bout using either trained [23, 25, 90], untrained [22-24, 43, 90] or healthy [92] men, resting

energy expenditure can remain significantly elevated 1-72 hours post exercise. Of the studies reporting significant increases in resting metabolic rate, Dolezal et al. [23], Schuenke et al. [25] and Hackney et al. [90] all reported significant increases beyond 24 hours that could potentially influence weight loss. Dolezal et al. [23] and Schuenke et al. [25] used moderate to high intensity exercise with younger subjects and saw the greatest increases in EPOC following an acute bout of resistance training. These two studies were used to develop the protocol for the current investigation.

Schuenke et al. [25] utilized a moderately high intensity protocol with 4 sets of 10 repetitions divided among 3 exercises and performed a circuit protocol with extended rest periods of 2 minutes. The subjects were young males in their twenties and were considered healthy. Unfortunately Schuenke did not report load values only the intensity of the exercise. Schuenke et al. observed significant increases in resting metabolism up to 38 hours following the acute bout of resistance exercise. The exercise intensity was between approximately70-80% of the subjects' 1RM for the lifts. Their results translated to an additional caloric expenditure of approximately 773 kcal post exercise. The investigation by Schuenke et al. is the only study known to have used an Olympic lift, in this case a power clean, for their resistance training protocol. The power clean has a distinctly different power component which increases the intensity of the lift, involves more muscle groups, and increases the potential for muscle damage. Dolezal et al. [23] also observed significant increases in resting metabolism 48 hours following an acute bout of resistance exercise. The exercise was high intensity for their subjects at approximately 85% of their subjects' 1RM for the lifts they used. The load their subjects lifted was approximately 9725 kg, a load similar to our light lift protocol of 10,000 kg. However, Dolezal et al. utilized a protocol design that emphasized eccentric contractions.

Since the previous two investigations used young trained males in all or part of their investigations, we also used male subjects (mean age 22) with no less than 1 year of resistance training experience. Schuenke et al [25] used a 10RM protocol and Dolezal et al [23] used a 6RM protocol. The Dolezal et al. investigation saw significant increases in resting metabolic rate for a longer period of time than previous studies and used higher intensity exercise. Because high intensity exercise [21, 22] is performed with heavier weights being lifted, it was postulated that the absolute load lifted would have an impact on EPOC. Potentially the heavier the load lifted by the subjects the greater their increase in resting metabolic rate following exercise would be. Therefore in the current study we adopted a 6-8RM protocol with a lighter load similar to Dolezal et al. [23] and an even heavier load in an attempt to elicit an even greater metabolic response. Multiple

exercises were chosen as in the Shuenke et al. [25] investigation to better mimic a traditional resistance training session. We added a fourth exercise to limit the muscle damage found in the investigation by Dolezal et al. [23].

With these parameters established the purpose of the present study was to compare the effects of two acute bouts of resistance exercise of differing loads on EPOC. In this investigation EPOC would be considered the difference between baseline resting metabolic rate and resting metabolic rate during recovery. The two loads chosen for this study were 10,000 and 20,000 kg. Three hypotheses were postulated. The first hypothesis was that EPOC would be elevated above resting levels at 12, 24, 36, and 48 hours following both the 10,000 kg and 20,000 kg lifts. The second hypothesis stated that VO_2 would be significantly different during the 20,000 kg lift compared to the 10,000 kg and during all time points following the lifts. And finally the third hypothesis stated that there would be a significant correlation between EPOC and the weight lifted relative to the subjects' body weight.

The hypotheses proposed for this investigation were not supported. Since there were no significant differences in energy expenditure after the resistance training bouts when compared to baseline metabolic rates with either the 10,000 kg or 20,000 kg lift the first hypothesis was unsupported. Although energy expenditure during the 20,000 kg lift was significantly greater than the energy expenditure during the 10,000 kg lift, there were no significant differences in EPOC following the exercise bout between the 10,000 and 20,000 kg bouts thus the second hypothesis was unsupported. Finally, since there were no significant correlations between EPOC and the weight lifted relative to subjects' body weights, the third hypothesis was unsupported. Currently our data do not support the research that has been previously published [23-25, 43, 90, 92].

There are two areas that may have influenced the results of our study compared to previous research, the high strength levels of our subjects and our protocol design. When examining the strength levels of subjects from previous studies it is apparent that the strength of our subjects was much higher. Therefore, the relative training stimulus used to elicit increases in EPOC was much lower in the present study compared to that used by previous research. Our protocol kept our subjects lifting in the 6-8 repetition range. Two earlier studies that used training intensities of 70% of their subjects 1RM saw significant increases in resting energy expenditure. Melby et al. [22] had subjects perform 6 sets of 10 different exercises for a total of 60 sets at 70% of their 1RM. The repetition range for this protocol was 8-12 repetitions per set. This amounts to approximately 550 repetitions performed during the course of the acute exercise bout. The average load lifted by these subjects was approximately 25,000 kg. Gillette et al. [43] used a similar protocol having their

subjects complete 5 sets of 10 different exercises for a total of 50 sets and again had subjects perform these lifts at an intensity of 70% of their 1RM. The repetition range here was also 8-12 repetitions leading approximately to 500 repetitions being performed. The average load lifted by these subjects was again approximately 25,000 kg.

The difference in the load lifted in our protocol and the previous two protocols was only 5,000 kg and may seem small until you examine the number of repetitions our subjects used in order to move their 20,000 kg load. Our subjects lifted a similar load but with a drastically lower number of approximately 199 repetitions total. Melby et al. had subjects lift approximately 15,000 – 38,000 kg and Gillette et al. [22, 43] had subjects lift approximately 25,000 kg. If we had our subjects perform a similar number of repetitions the amount of weight our subjects lifted would have been close to 50,000 kg. Perhaps if our subjects had lifted this volume of weight they too may have experienced increases in 24 and 48-hour EPOC.

Another investigation has recently shown an increase in resting energy expenditure for 72 hours following an acute bout of resistance exercise. Hackney et al. [90] used 8 trained and 8 untrained subjects who performed 5 sets of 6 repetitions using weights ranging from approximately 51-65 kg for the bench press portion of their protocol. Our subjects performed the bench press for 6-8 repetitions with an average of approximately 137 kg. The entire load for their subjects was approximately 15,000 kg not including the weight used in the 3 familiarization sets. This is a relatively heavy load before the number of repetitions used to complete these lifts is considered. When examining the new data presented by Hackney et al. [90] the investigators reported using 8 exercises for 5 sets of 6 repetitions, a total of 240 repetitions to move a load of only 14,820 kg. In this case our subjects clearly moved more weight using far fewer repetitions. In other words given the strength of our subjects they would have moved a similar load with approximately 150 repetitions. This indicates that the exercise necessary to create a great enough perturbation in our subjects, compared to other investigations would have had to have been extreme in either volume or repetitions. Loads of that magnitude may be unreasonable for even highly trained resistance athletes to perform during the course of an exercise session. This leads this investigator to believe that initially resistance training may increase metabolic rate enough to promote weight loss, but once a certain level of training or fitness has been established, resistance training may no longer be a viable option for increasing EPOC or resting metabolic rate to help in promoting weight loss.

Like Dolezal et al. [23], Hackney et al. [90] focused on eccentric contractions and both saw significant increases in CK and RPMS. In the present study there were no significant increases in muscle damage measured by CK levels or RPMS in the subjects after the 10,000 kg lifting bout.

For most of the subjects the exercise protocol was similar to their normal workout load volume, but not their exercise distribution. There were however, significant increases in CK levels at 12 hours post exercise from baseline and immediately post-exercise for the 20,000 kg lift. Blood levels of CK in this investigation were similar to levels found in previous studies [23, 90, 93] using resistance trained athletes. Unlike Hackney et al. [90] and Dolezal et al. [23] our subjects did not experience a significant increase in resting energy expenditure. However when examining RPMS in these studies our resistance trained subjects reported no significant changes in their muscle soreness while the two previous studies that reported significant increases in both CK levels and resting metabolic rate following exercise also reported significant increases in RPMS.

Since Dolezal et al. [23] had experienced such prolonged and significant increases in resting metabolic rate following the eccentric muscle protocol, in our investigation we attempted to control for muscle damage by spreading out the 10,000 kg and 20,000 kg lifts over 4 body parts and choosing not to focus on the eccentric portion of the lift. An informal survey conducted prior to this investigation asked our participants what their habitual training volume was per body part during an acute bout of resistance training. This survey indicated that 10,000-15,000 kg per body part was lifted. Based on this information and to attenuate muscle damage each body part in this investigation was assigned approximately 50% of that amount on the heavier lifting day and approximately 25% on the lighter lifting day.

Based on results reported by both Dolezal et al [23] and Hackney et al. [90] it seems likely that by controlling for eccentric damage during our protocol this may have blunted protein synthesis and consequently the EPOC levels following our acute bouts of exercise. Dolezal et al. [23] emphasized the eccentric component using only one exercise. In this case the eccentric emphasis was used on one exercise; the leg press alone. Their subjects performed 8 sets of leg presses using a predetermined 6 repetition maximum. Hackney et al. [90] also emphasized the eccentric component by using a 1 second concentric contraction followed by a 3 second eccentric contraction on 10 different exercises to induce delayed onset muscle soreness. These investigations link moderate to high intensity exercise emphasizing eccentric muscle contraction to increased resting metabolic rate. The damage caused by this type of exercise may promote increased protein synthesis which is a costly metabolic process [91].

It is also possible that our subjects had a lower resting metabolic rate due to a relatively low caloric intake for their body weight. Our subjects had an average weight of 88 kg with an average lean body fat percentage of 10%. Compared to weight lifting subjects of similar body masses [94, 95] our average caloric intake of 2281 kilocalories was relatively low. Both Tarnopolsky et al. [95]

and Chen et al. [94] reported body masses of 80 kg and daily energy intake of 4802 kcal and 4597 kcal respectively while our subjects reported an average daily energy intake of approximately 2281 kcal. Although the only data available that I am aware of comparing restrictive eating and resting metabolic rate was done with females, Laessle et al. [96] and Platte et al. [97] both reported in their investigations that restrictive eating significantly reduces resting metabolic rate. It is possible then that our subjects, many of whom seemed very concerned with body image, may have what would be considered disordered eating patterns in their attempt to maintain a certain appearance. Our subjects may have even further restricted their caloric intake because they were asked to track their food over 5 days. Consciously or unconsciously this may have caused them to alter their intake. This could certainly be a confounding variable in our study and may have blunted their metabolic response to an acute resistance training bout. Future investigations could include a minimum caloric intake for the subjects relative to their body weight in order to control for this.

Future investigations using highly trained resistance athletes could focus on comparing EPOC following two bouts of acute resistance training of varying intensity levels. Using intensities similar to those used in previous investigations at 70% of the 1RM and the relatively high intensities our subjects used at 85% of their 1RM. This could better establish whether load or number of repetitions plays a greater role in EPOC during resistance training. Another potential area of investigation would be to compare two resistance protocols using identical exercises and loads and using only concentric contraction for one protocol and eccentric contraction only for the second protocol to determine if it truly is a mechanism of eccentric muscle damage that increases resting metabolic following resistance training.

Also in the current investigation, only acute bouts of exercise were examined. As this is rarely the way resistance exercise training is conducted. It is possible that a training protocol designed to be conducted over the course of 3-5 days would resemble the way most resistance training programs are carried out and would be more informative. One example would be to use whole body resistance training (training each major muscle group) on each day lifted. Another example would be to train single muscle groups on multiple days (chest on Mondays, back on Tuesdays, etc.). The cumulative effect of multiple training days on resting metabolic rate could raise resting energy expenditure to a level that would significantly impact energy expenditure and consequently affect weight maintenance or weight loss.

In conclusion, as our investigation did not see any increases in resting metabolic rate, our results support the ACSM's current position stand on resistance exercise and weight control [8]; that resistance training is an important part of a weight loss regimen by minimizing the loss of fat

free mass, but that there is no scientific evidence to suggest resistance training is superior to endurance exercise for weight loss in highly resistance trained males.

APPENDIX A

RECRUITMENT FLYER

Looking for healthy, 18-39 year old men,
experienced in resistance (weight) training

to participate in training sessions to determine
energy expenditure (calories burned)
following bouts of resistance training.

Please contact George Abboud at
gga3651@fsu.edu or 445-4611.

APPENDIX B

INSTITUTIONAL REVIEW BOARD (IRB)

Florida State
UNIVERSITY

Office of the Vice President For Research
Human Subjects Committee
Tallahassee, Florida 32306-2742
(850) 644-8673 · FAX (850) 644-4392

APPROVAL MEMORANDUM (for change in research protocol)

Date: 6/21/2006

To:
George Abboud
MC 1493

Dept: NUTRITION FOOD AND MOVEMENT SCIENCES

From: Thomas L. Jacobson, Chair

Re: Use of Human subjects in Research
Project entitled: A comparison of the effects of two acute resistance training bouts on post exercise oxygen consumption

The memorandum that you submitted to this office in regard to the requested change in your research protocol for the above-referenced project have been reviewed and approved. Thank you for informing the Committee of this change.

A reminder that if the project has not been completed by 3/14/2007, you must request renewed approval for continuation of the project.

By copy of this memorandum, the chairman of your department and/or your major professor is reminded that he/she is responsible for being informed concerning research projects involving human subjects in the department, and should review protocols of such investigations as often as needed to insure that the project is being conducted in compliance with our institution and with DHHS regulations.

This institution has an Assurance on file with the Office for Protection from Research Risks. The Assurance Number is IRB00000446..

cc: Lynn Panton
APPLICATION NO. 2006.0209

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APPENDIX C

INFORMED CONSENT

INFORMED CONSENT FORM

1. I freely and voluntarily and without element of force or coercion, consent to be a participant in the research project entitled "A comparison of the effects of two acute resistance training bouts on post exercise oxygen consumption." George Abboud and Lynn Panton, Ph.D., a doctoral candidate and faculty member, are conducting this research respectively, at Florida State University in the Department of Nutrition, Food and Exercise Sciences.

2. The purpose of this study will be to compare the effects of two acute bouts of resistance exercise of differing loads on exercise post oxygen consumption (EPOC).

3. My participation in this project will involve coming to the Exercise Physiology labs at Florida State University for 7 sessions. On the first visit I will be oriented to the study, sign an informed consent, and complete questionnaires on health, exercise, and tobacco histories. I will also have my height and weight measured and my body composition assessed using a 3 site skinfold method. I will then undergo a familiarization period with the exercises (bench press, squat, bent-over row, Romanian deadlift, which is a lift designed to ease the stress on the knees and low back because the subject does not bring the weight to the ground as in a traditional deadlift) that I will be performing during this study and complete a 1 repetition maximum (1RM) test for each exercise. I will be given a food diary log to record my exact diet 3 days prior to my second visit and for 48 hours following my first exercise bout. This log will be used to exactly replicate my diet 3 days prior to and during my second series of visits. I will choose a golf ball from a bag to randomly determine which exercise bout I will complete first, either a 10,000 or 20,000 kg bout spread over four exercises.

On my second visit I will report to the laboratory at 2100 hours following a 3 hour fast. I will undergo a 30-minute period of supine quiet rest. Immediately following that period my oxygen (VO_2) samples will be collected beneath a ventilated hood to determine my resting metabolic rate (RMR) during a 30-minute period. I will be provided a standardized meal such as a turkey sandwich or a bowl of cereal then sleep overnight in the laboratory to ensure 7-8 hours of sleep prior to my baseline RMR measurements. I will be awakened at 0545 to perform personal hygiene. I will slowly walk to the testing area and undergo a 30-minute period of quiet supine rest followed by 30 minutes of metabolic gas collection under the ventilated hood for the determination of RMR. After the determination of my RMR I will have approximately 25 ml of blood taken in sterile vacutainer tubes from an antecubital vein using sterile venipuncture techniques. My blood will be collected in EDTA coated tubes and centrifuged for 10 minutes. Samples will be separated and stored at -80 degrees C until analysis. My blood will be analyzed to determine Creatine Kinase activity (a marker of muscle damage) and cholesterol levels and will be measured in duplicate using an enzymatic assay kit.

I then will have my cardiovascular function evaluated before and after the exercise session. I will have a strap placed around my chest and thighs and a foot support to keep me from slipping off the table. After 30 minutes of rest on a padded table, I will be tilted up to an almost standing position. I will stay in this position for 10 minutes. My heart rate and blood pressure will be continuously monitored to see how quickly my heart rate and blood pressure change. My heart rate will be measured by putting three electrodes on my chest and placing a little cuff around my forefinger that will monitor my blood pressure.

Subject's Initials

_____

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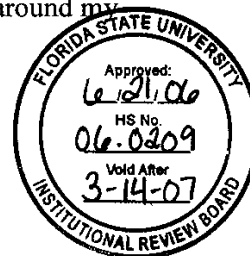
5. The possible benefits of my participation in this research project include learning my strength levels and how many kilocalories I burn when performing the resistance training in this study. I will also be given a number of tests free of charge and the results will provide me with an accurate assessment of my body composition and blood cholesterol.

I will also learn about my cardiovascular reflexes status. It is important to know how my heart rate and blood pressure are modulated in response to exercise and positional changes.

6. The results of this research study may be published but neither my name or identity will be revealed. Information obtained during the course of the study will remain confidential, to the extent allowed by law. My name will not appear on any of the results. No individual responses will be reported. Only group findings will be reported in publications. Confidentially will be maintained by assigning each subject a code number and recording all data by code number. Dr. Lynn Panton, will keep the only record with the subject's name and code number in a locked drawer in her office. Data will be kept for 10 years and then destroyed.

7. In case of an injury first aid will be provided to me by the laboratory personnel working on the research project, and any other treatment or care will be provided at my expense.

8. I will not be paid for my participation in this research project.

9. Any questions I have concerning the research study or my participation in it, before or after my consent, will be answered by the investigators or they will refer me to a knowledgeable source. I understand that I may contact Dr. Lynn Panton at work (850) 644-4685 or at home (850) 893-3159 for answers to questions about this research project or my rights. Group results will be sent to me upon my request.

10. In case of injury, or if I have questions about my rights as a subject/participant in this research, or if I feel I have been placed at risk, I can contact the chair of the Human Subjects committee, Institutional Review Board, through the Office of the Vice President for Research, at (850) 644-8633.

11. The nature, demands, benefits and risks of the project have been explained to me. I knowingly assume any risks involved and if I decide to withdrawn at any time for any reason, I may do so without penalty.

I have read the above informed consent form. I understand that I may withdraw my consent and discontinue participation at any time without penalty or loss of benefits to which I may otherwise be entitled. In signing this consent form, I am not waiving my legal claims, rights or remedies. A copy of this consent form will be given to me.

_____
(Subject Signature & Date)

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After this test I will then be allowed a light Gold Balance Bar 15 minutes prior to the first exercise protocol. I will be asked to assess my muscle soreness via the Visual Assessment Scale (VAS).

I will then put on a mouthpiece and nose clip and perform the first acute bout of exercise while my VO_2 samples are collected. I will perform 6-8 repetitions of each exercise that will consist of the bench press, squat, bent-over row and Romanian deadlift at approximately 85% of my 1RM. I will perform as many sets as necessary to completely lift the prescribed load for each exercise. I will wear a weight belt during the entire exercise protocol to reduce lower back stress.

I will arrive for visit 3, 24 hours following my arrival for visit 2 and will follow the exact same protocols as visit 2, but I will not perform any exercise after spending the night in the laboratory, I will only have VO_2 measurements recorded.

I will arrive for visit 4, 24 hours following my arrival for visit 3 and will follow the exact same protocols as visit 3.

I will replicate the first protocol no sooner than 24 hours following the last testing session of the first protocol. The acute exercise bout of the second protocol will be either lighter or heavier depending on which absolute weight I performed in the first protocol. Replication will begin by exactly matching my dietary intake 3 days prior to my second acute exercise session as documented in my food diary log.

4. I understand there is a possibility of a minimal level of risk involved if I agree to participate in this study. The risks will be minimized by using trained technicians and by teaching me proper techniques in testing and training. I will complete a health history questionnaire before I can participate in the study. I will not be able to participate in this study if I yes to any of the question on the questionnaire or have any other condition that may be contraindicated for exercise testing and training.

I understand that there is the possibility of muscle soreness following the acute exercise bouts. The risk of muscle soreness will be minimized by using 4 exercises specific to 4 different muscle groups and soreness will be tracked via a visual analog scale (VAS) to rate my soreness.

The risks of drawing blood are small; there may be some discomfort at the site of needle placement with possible bruising, swelling or infection. The risk will be minimized by the use of skilled technicians using sterile techniques and equipment.

There are minimal risks or discomforts with answering the enclosed questionnaires. If I choose not to complete the questionnaires I will not be able to participate in the study.

The risk associated with head-up tilt test may be a feeling of dizziness. If I experience dizziness the test will be immediately stopped and I will be placed back in a horizontal position. These possible side effects will disappear within a few minutes after the termination of the test.

Subject's Initials

_____

APPENDIX D

FOOD LOG DIARY

Day 1		Day 2		Day 3	
Food Eaten	Quantity	Food Eaten	Quantity	Food Eaten	Quantity

Day 4		Day 5		Day 6	
Food Eaten	Quantity	Food Eaten	Quantity	Food Eaten	Quantity

Day 7		Day 8		Day 9	
Food Eaten	Quantity	Food Eaten	Quantity	Food Eaten	Quantity

Day 10		Day 11		Day 12	
Food Eaten	Quantity	Food Eaten	Quantity	Food Eaten	Quantity

APPENDIX E

VISUAL ANALOG SCALE

Visual Analog Scale

No
Pain

Chest

Unbearable
Pain

No
Pain

Quadriceps

Unbearable
Pain

No
Pain

Back

Unbearable
Pain

No
Pain

Hamstrings

Unbearable
Pain

APPENDIX F

SPSS DATA ANALYSIS

```
GLM
      BaseRMR10kg2kcal EPOC10kg24kcal EPOC10kg48kcal
   /WSFACTOR = amRMR10kg 3 Polynomial
   /MEASURE = Kcal
   /METHOD = SSTYPE(3)
   /PLOT = PROFILE( amRMR10kg )
   /EMMEANS = TABLES(amRMR10kg) COMPARE ADJ(BONFERRONI)
   /PRINT = DESCRIPTIVE ETASQ
   /CRITERIA = ALPHA(.05)
   /WSDESIGN = amRMR10kg .
```

General Linear Model

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data I.sav

Within-Subjects Factors

Measure: Kcal

amRMR10kg	Dependent Variable
1	Base RMR10kg2kcal
2	EPOC10kg24kcal
3	EPOC10kg48kcal

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR10kg2kcal	35.8213	4.67481	8
EPOC10kg24kcal	36.1338	6.10614	8
EPOC10kg48kcal	36.4963	5.20895	8

Mauchly's Test of Sphericity[b]

Measure: Kcal

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
amRMR10kg	.881	.762	2	.683	.893	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
   Design: Intercept
   Within Subjects Design: amRMR10kg

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Tests of Within-Subjects Effects

Measure: Kcal

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
amRMR10kg	Sphericity Assumed	1.826	2	.913	.068	.935	.010
	Greenhouse-Geisser	1.826	1.787	1.022	.068	.918	.010
	Huynh-Feldt	1.826	2.000	.913	.068	.935	.010
	Lower-bound	1.826	1.000	1.826	.068	.802	.010
Error(amRMR10kg)	Sphericity Assumed	188.901	14	13.493			
	Greenhouse-Geisser	188.901	12.508	15.103			
	Huynh-Feldt	188.901	14.000	13.493			
	Lower-bound	188.901	7.000	26.986			

Estimated Marginal Means

amRMR10kg

Estimates

Measure: Kcal

amRMR10kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	35.821	1.653	31.913	39.729
2	36.134	2.159	31.029	41.239
3	36.496	1.842	32.141	40.851

Pairwise Comparisons

Measure: Kcal

(I) amRMR10kg	(J) amRMR10kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.313	2.128	1.000	-6.967	6.342
	3	-.675	1.631	1.000	-5.775	4.425
2	1	.313	2.128	1.000	-6.342	6.967
	3	-.363	1.713	1.000	-5.719	4.994
3	1	.675	1.631	1.000	-4.425	5.775
	2	.363	1.713	1.000	-4.994	5.719

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRMR10kg2ml EPOC10kg24ml EPOC10kg48ml
  /WSFACTOR = amRMR10kg 3 Polynomial
  /MEASURE = Relative
  /METHOD = SSTYPE(3)
```

```
/PLOT = PROFILE( amRMR10kg )
/EMMEANS = TABLES(amRMR10kg) COMPARE ADJ(BONFERRONI)
/PRINT = DESCRIPTIVE ETASQ
/CRITERIA = ALPHA(.05)
/WSDESIGN = amRMR10kg .
```

General Linear Model

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Within-Subjects Factors

Measure: Relative

amRMR10kg	Dependent Variable
1	Base RMR10kg2ml
2	EPOC10kg24ml
3	EPOC10kg48ml

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR10kg2ml	3.3363	.29737	8
EPOC10kg24ml	3.3813	.42673	8
EPOC10kg48ml	3.3863	.30355	8

Mauchly's Test of Sphericity[b]

Measure: Relative

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser	Huynh-Feldt	Lower-bound
					Epsilon[a]		
amRMR10kg	.777	1.514	2	.469	.818	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: amRMR10kg

Tests of Between-Subjects Effects

Measure: Relative

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	272.229	1	272.229	1931.491	.000	.996
Error	.987	7	.141			

Estimated Marginal Means

amRMR10kg

Estimates

Measure: Relative

amRMR10kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	3.336	.105	3.088	3.585
2	3.381	.151	3.024	3.738
3	3.386	.107	3.132	3.640

Pairwise Comparisons

Measure: Relative

(I) amRMR10kg	(J) amRMR10kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.045	.199	1.000	-.668	.578
	3	-.050	.131	1.000	-.459	.359
2	1	.045	.199	1.000	-.578	.668
	3	-.005	.162	1.000	-.513	.503
3	1	.050	.131	1.000	-.359	.459
	2	.005	.162	1.000	-.503	.513

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRMR10kg2L EPOC10kg24L EPOC10kg48L
  /WSFACTOR = amRMR10kg 3 Polynomial
  /MEASURE = Absolute
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( amRMR10kg )
  /EMMEANS = TABLES(amRMR10kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE ETASQ
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = amRMR10kg .
```

General Linear Model

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Within-Subjects Factors

Measure: Absolute

amRMR10kg	Dependent Variable
1	Base RMR10kg2L
2	EPOC10kg24L
3	EPOC10kg48L

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR10kg2L	.2950	.03964	8
EPOC10kg24L	.2988	.04794	8
EPOC10kg48L	.2988	.04454	8

Mauchly's Test of Sphericity[b]

Measure: Absolute

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
amRMR10kg	.812	1.249	2	.536	.842	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: amRMR10kg

Tests of Between-Subjects Effects

Measure: Absolute
Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Intercept	2.124	1	2.124	500.361	.000	.986
Error	.030	7	.004			

Estimated Marginal Means

67

amRMR10kg

Estimates

Measure: Absolute

amRMR10kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.295	.014	.262	.328
2	.299	.017	.259	.339
3	.299	.016	.262	.336

Pairwise Comparisons

Measure: Absolute

(I) amRMR10kg	(J) amRMR10kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.004	.016	1.000	-.055	.048
	3	-.004	.011	1.000	-.038	.031
2	1	.004	.016	1.000	-.048	.055
	3	.000	.015	1.000	-.045	.045
3	1	.004	.011	1.000	-.031	.038
	2	.000	.015	1.000	-.045	.045

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRMR10kg1kcal EPOC10kg12kcal EPOC10kg36kcal
  /WSFACTOR = pmRMR10kg 3 Polynomial
  /MEASURE = Kcal
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( pmRMR10kg )
  /EMMEANS = TABLES(pmRMR10kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE ETASQ
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = pmRMR10kg .
```

General Linear Model

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data I.sav

Within-Subjects Factors

Measure: Kcal

pmRMR10kg	Dependent Variable
1	Base RMR10kg1kcal
2	EPOC10kg12kcal
3	EPOC10kg36kcal

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR10kg1kcal	40.0888	5.86115	8
EPOC10kg12kcal	40.5025	7.93596	8
EPOC10kg36kcal	41.5675	7.56294	8

Mauchly's Test of Sphericity[b]

Measure: Kcal

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
pmRMR10kg	.968	.198	2	.906	.969	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: pmRMR10kg

Tests of Within-Subjects Effects

Measure: Kcal

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
pmRMR10kg	Sphericity Assumed	9.312	2	4.656	.823	.459	.105
	Greenhouse-Geisser	9.312	1.937	4.807	.823	.456	.105
	Huynh-Feldt	9.312	2.000	4.656	.823	.459	.105
	Lower-bound	9.312	1.000	9.312	.823	.394	.105
Error(pmRMR10kg)	Sphericity Assumed	79.162	14	5.654			
	Greenhouse-Geisser	79.162	13.561	5.838			
	Huynh-Feldt	79.162	14.000	5.654			
	Lower-bound	79.162	7.000	11.309			

Estimated Marginal Means

pmRMR10kg

Estimates

Measure: Kcal

pmRMR10kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	40.089	2.072	35.189	44.989
2	40.503	2.806	33.868	47.137
3	41.568	2.674	35.245	47.890

Pairwise Comparisons

Measure: Kcal

(I) pmRMR10kg	(J) pmRMR10kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.414	1.214	1.000	-4.210	3.383
	3	-1.479	1.264	.841	-5.432	2.474
2	1	.414	1.214	1.000	-3.383	4.210
	3	-1.065	1.081	1.000	-4.447	2.317
3	1	1.479	1.264	.841	-2.474	5.432
	2	1.065	1.081	1.000	-2.317	4.447

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRMR10kg1ml EPOC10kg12ml EPOC10kg36ml
  /WSFACTOR = pmRMR10kg 3 Polynomial
  /MEASURE = Relative
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( pmRMR10kg )
  /EMMEANS = TABLES(pmRMR10kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE ETASQ
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = pmRMR10kg .
```

General Linear Model

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Within-Subjects Factors

Measure: Relative

pmRMR10kg	Dependent Variable
1	Base RMR10kg1ml
2	EPOC10kg1 2ml
3	EPOC10kg3 6ml

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR10kg1ml	3.7113	.37650	8
EPOC10kg12ml	3.7488	.59595	8
EPOC10kg36ml	3.8713	.59772	8

Mauchly's Test of Sphericity[b]

Measure: Relative

					Epsilon[a]		
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser	Huynh-Feldt	Lower-bound
pmRMR10kg	.955	.275	2	.872	.957	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: pmRMR10kg

Tests of Within-Subjects Effects

Measure: Relative

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
pmRMR10kg	Sphericity Assumed	.112	2	.056	1.132	.350	.139
	Greenhouse-Geisser	.112	1.914	.059	1.132	.349	.139
	Huynh-Feldt	.112	2.000	.056	1.132	.350	.139
	Lower-bound	.112	1.000	.112	1.132	.323	.139
Error(pmRMR10kg)	Sphericity Assumed	.693	14	.049			
	Greenhouse-Geisser	.693	13.400	.052			
	Huynh-Feldt	.693	14.000	.049			
	Lower-bound	.693	7.000	.099			

Estimated Marginal Means

71

pmRMR10kg

Estimates

Measure: Relative

pmRMR10kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	3.711	.133	3.396	4.026
2	3.749	.211	3.251	4.247
3	3.871	.211	3.372	4.371

Pairwise Comparisons

Measure: Relative

(I) pmRMR10kg	(J) pmRMR10kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.038	.110	1.000	-.382	.307
	3	-.160	.121	.687	-.540	.220
2	1	.038	.110	1.000	-.307	.382
	3	-.123	.101	.795	-.439	.194
3	1	.160	.121	.687	-.220	.540
	2	.123	.101	.795	-.194	.439

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRMR10kg1L EPOC10kg12L EPOC10kg36L
  /WSFACTOR = pmRMR10kg 3 Polynomial
  /MEASURE = Absolute
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( pmRMR10kg )
  /EMMEANS = TABLES(pmRMR10kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE ETASQ
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = pmRMR10kg .
```

General Linear Model

```
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data I.sav
```

Within-Subjects Factors

Measure: Absolute

pmRMR10kg	Dependent Variable
1	Base RMR10kg1L
2	EPOC10kg1 2L
3	EPOC10kg3 6L

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR10kg1L	.3275	.04979	8
EPOC10kg12L	.3313	.06556	8
EPOC10kg36L	.3400	.06392	8

Mauchly's Test of Sphericity[b]

Measure: Absolute

					Epsilon[a]		
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser	Huynh-Feldt	Lower-bound
pmRMR10kg	.951	.302	2	.860	.953	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: pmRMR10kg

Tests of Within-Subjects Effects

Measure: Absolute

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
pmRMR10kg	Sphericity Assumed	.001	2	.000	.885	.435	.112
	Greenhouse-Geisser	.001	1.906	.000	.885	.431	.112
	Huynh-Feldt	.001	2.000	.000	.885	.435	.112
	Lower-bound	.001	1.000	.001	.885	.378	.112
Error(pmRMR10kg)	Sphericity Assumed	.005	14	.000			
	Greenhouse-Geisser	.005	13.344	.000			
	Huynh-Feldt	.005	14.000	.000			
	Lower-bound	.005	7.000	.001			

Estimated Marginal Means

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pmRMR10kg

Estimates

Measure: Absolute

pmRMR10kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.328	.018	.286	.369
2	.331	.023	.276	.386
3	.340	.023	.287	.393

Pairwise Comparisons

Measure: Absolute

(I) pmRMR10kg	(J) pmRMR10kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.004	.009	1.000	-.033	.025
	3	-.013	.011	.837	-.046	.021
2	1	.004	.009	1.000	-.025	.033
	3	-.009	.009	1.000	-.037	.019
3	1	.013	.011	.837	-.021	.046
	2	.009	.009	1.000	-.019	.037

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRMR20kg2kcal EPOC20kg24kcal EPOC20kg48kcal
  /WSFACTOR = amRMR20kg 3 Polynomial
  /MEASURE = Kcal
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( amRMR20kg )
  /EMMEANS = TABLES(amRMR20kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE ETASQ
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = amRMR20kg .
```

General Linear Model

```
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data I.sav
```

Within-Subjects Factors

Measure: Kcal

amRMR20kg	Dependent Variable
1	Base RMR20kg2kcal
2	EPOC20kg24kcal
3	EPOC20kg48kcal

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR20kg2kcal	38.8050	10.95368	8
EPOC20kg24kcal	35.7938	5.17706	8
EPOC20kg48kcal	37.8913	5.92126	8

Mauchly's Test of Sphericity[b]

Measure: Kcal

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
amRMR20kg	.417	5.248	2	.073	.632	.707	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: amRMR20kg

Tests of Within-Subjects Effects

Measure: Kcal

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
amRMR20kg	Sphericity Assumed	38.139	2	19.069	.444	.650	.060
	Greenhouse-Geisser	38.139	1.263	30.186	.444	.568	.060
	Huynh-Feldt	38.139	1.413	26.985	.444	.588	.060
	Lower-bound	38.139	1.000	38.139	.444	.527	.060
Error(amRMR20kg)	Sphericity Assumed	601.522	14	42.966			
	Greenhouse-Geisser	601.522	8.844	68.014			
	Huynh-Feldt	601.522	9.893	60.802			
	Lower-bound	601.522	7.000	85.932			

Estimated Marginal Means

amRMR20kg

Estimates

Measure: Kcal

amRMR20kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	38.805	3.873	29.647	47.963
2	35.794	1.830	31.466	40.122
3	37.891	2.093	32.941	42.842

Pairwise Comparisons

Measure: Kcal

(I) amRMR20kg	(J) amRMR20kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	3.011	3.088	1.000	-6.646	12.668
	3	.914	4.286	1.000	-12.491	14.319
2	1	-3.011	3.088	1.000	-12.668	6.646
	3	-2.098	2.078	1.000	-8.598	4.403
3	1	-.914	4.286	1.000	-14.319	12.491
	2	2.098	2.078	1.000	-4.403	8.598

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

General Linear Model

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Within-Subjects Factors

Measure: Absolute

amRMR20kg	Dependent Variable
1	Base RMR20kg2L
2	EPOC20kg24L
3	EPOC20kg48L

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR20kg2L	.3150	.08701	8
EPOC20kg24L	.2988	.04518	8
EPOC20kg48L	.3113	.05276	8

Mauchly's Test of Sphericity[b]

Measure: Absolute

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
amRMR20kg	.425	5.130	2	.077	.635	.712	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: amRMR20kg

Tests of Within-Subjects Effects

Measure: Absolute

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
amRMR20kg	Sphericity Assumed	.001	2	.001	.214	.810	.030
	Greenhouse-Geisser	.001	1.270	.001	.214	.712	.030
	Huynh-Feldt	.001	1.424	.001	.214	.737	.030
	Lower-bound	.001	1.000	.001	.214	.657	.030
Error(amRMR20kg)	Sphericity Assumed	.038	14	.003			
	Greenhouse-Geisser	.038	8.891	.004			
	Huynh-Feldt	.038	9.970	.004			
	Lower-bound	.038	7.000	.005			

Estimated Marginal Means

amRMR20kg

Estimates

Measure: Absolute

amRMR20kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.315	.031	.242	.388
2	.299	.016	.261	.337
3	.311	.019	.267	.355

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Pairwise Comparisons

Measure: Absolute

(I) amRMR20kg	(J) amRMR20kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.016	.025	1.000	-.061	.093
	3	.004	.034	1.000	-.102	.110
2	1	-.016	.025	1.000	-.093	.061
	3	-.013	.016	1.000	-.064	.039
3	1	-.004	.034	1.000	-.110	.102
	2	.013	.016	1.000	-.039	.064

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRMR20kg2ml EPOC20kg24ml EPOC20kg48ml
  /WSFACTOR = amRMR20kg 3 Polynomial
  /MEASURE = Relative
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( amRMR20kg )
  /EMMEANS = TABLES(amRMR20kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = amRMR20kg .
```

General Linear Model

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Within-Subjects Factors

Measure: Relative

amRMR20kg	Dependent Variable
1	Base RMR20kg2ml
2	EPOC20kg24ml
3	EPOC20kg48ml

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR20kg2ml	3.5575	.77210	8
EPOC20kg24ml	3.3875	.33737	8
EPOC20kg48ml	3.5338	.59042	8

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
amRMR20kg	Pillai's Trace	.168	.606[a]	2.000	6.000	.576
	Wilks' Lambda	.832	.606[a]	2.000	6.000	.576
	Hotelling's Trace	.202	.606[a]	2.000	6.000	.576
	Roy's Largest Root	.202	.606[a]	2.000	6.000	.576

a. Exact statistic

b.

  Design: Intercept
  Within Subjects Design: amRMR20kg

Mauchly's Test of Sphericity[b]

Measure: Relative

					Epsilon[a]		
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser	Huynh-Feldt	Lower-bound
amRMR20kg	.481	4.387	2	.112	.658	.751	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

  Design: Intercept
  Within Subjects Design: amRMR20kg

Tests of Within-Subjects Effects

Measure: Relative

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
amRMR20kg	Sphericity Assumed	.136	2	.068	.211	.812
	Greenhouse-Geisser	.136	1.317	.103	.211	.722
	Huynh-Feldt	.136	1.502	.090	.211	.752
	Lower-bound	.136	1.000	.136	.211	.660
Error(amRMR20kg)	Sphericity Assumed	4.500	14	.321		
	Greenhouse-Geisser	4.500	9.219	.488		
	Huynh-Feldt	4.500	10.514	.428		
	Lower-bound	4.500	7.000	.643		

Tests of Within-Subjects Contrasts

Measure: Relative

Source	amRMR20kg	Type III Sum of Squares	df	Mean Square	F	Sig.
amRMR20kg	Linear	.002	1	.002	.004	.950
	Quadratic	.133	1	.133	1.335	.286
Error(amRMR20kg)	Linear	3.800	7	.543		
	Quadratic	.699	7	.100		

Tests of Between-Subjects Effects

Measure: Relative

Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	292.811	1	292.811	704.334	.000
Error	2.910	7	.416		

Estimated Marginal Means

amRMR20kg

Estimates

Measure: Relative

amRMR20kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	3.558	.273	2.912	4.203
2	3.388	.119	3.105	3.670
3	3.534	.209	3.040	4.027

Pairwise Comparisons

Measure: Relative

(I) amRMR20kg	(J) amRMR20kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.170	.259	1.000	-.641	.981
	3	.024	.368	1.000	-1.128	1.176
2	1	-.170	.259	1.000	-.981	.641
	3	-.146	.195	1.000	-.757	.464
3	1	-.024	.368	1.000	-1.176	1.128
	2	.146	.195	1.000	-.464	.757

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Multivariate Tests

	Value	F	Hypothesis df	Error df	Sig.
Pillai's trace	.168	.606[a]	2.000	6.000	.576
Wilks' lambda	.832	.606[a]	2.000	6.000	.576
Hotelling's trace	.202	.606[a]	2.000	6.000	.576
Roy's largest root	.202	.606[a]	2.000	6.000	.576

Each F tests the multivariate effect of amRMR20kg. These tests are based on the linearly independent pairwise comparisons among the estimated marginal means.

a. Exact statistic

```
GLM
  BaseRMR20kg1kcal EPOC20kg12kcal EPOC20kg36kcal
  /WSFACTOR = pmRMR20kg 3 Polynomial
  /MEASURE = Kcal
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( pmRMR20kg )
  /EMMEANS = TABLES(pmRMR20kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE ETASQ
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = pmRMR20kg .
```

General Linear Model

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Within-Subjects Factors

Measure: Kcal

pmRMR20kg	Dependent Variable
1	BaseRMR20kg1kcal
2	EPOC20kg12kcal
3	EPOC20kg36kcal

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR20kg1kcal	39.2913	6.72313	8
EPOC20kg12kcal	40.1725	5.58156	8
EPOC20kg36kcal	41.5875	8.41044	8

Mauchly's Test of Sphericity[b]

Measure: Kcal

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
pmRMR20kg	.909	.572	2	.751	.917	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: pmRMR20kg

Tests of Within-Subjects Effects

Measure: Kcal

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
pmRMR20kg	Sphericity Assumed	21.471	2	10.735	.629	.547	.083
	Greenhouse-Geisser	21.471	1.833	11.712	.629	.535	.083
	Huynh-Feldt	21.471	2.000	10.735	.629	.547	.083
	Lower-bound	21.471	1.000	21.471	.629	.454	.083
Error(pmRMR20kg)	Sphericity Assumed	238.759	14	17.054			
	Greenhouse-Geisser	238.759	12.832	18.606			
	Huynh-Feldt	238.759	14.000	17.054			
	Lower-bound	238.759	7.000	34.108			

Estimated Marginal Means

pmRMR20kg

Estimates

Measure: Kcal

pmRMR20kg	Mean	Std. Error	95% Confidence Interval Lower Bound	Upper Bound
1	39.291	2.377	33.671	44.912
2	40.173	1.973	35.506	44.839
3	41.588	2.974	34.556	48.619

Pairwise Comparisons

Measure: Kcal

(I) pmRMR20kg	(J) pmRMR20kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.881	1.808	1.000	-6.537	4.775
	3	-2.296	2.013	.875	-8.592	4.000
2	1	.881	1.808	1.000	-4.775	6.537
	3	-1.415	2.338	1.000	-8.728	5.898
3	1	2.296	2.013	.875	-4.000	8.592
	2	1.415	2.338	1.000	-5.898	8.728

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRMR20kg1ml EPOC20kg12ml EPOC20kg36ml
  /WSFACTOR = pmRMR20kg 3 Polynomial
  /MEASURE = Relative
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( pmRMR20kg )
  /EMMEANS = TABLES(pmRMR20kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE ETASQ
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = pmRMR20kg .
```

General Linear Model

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Within-Subjects Factors

Measure: Relative

pmRMR20kg	Dependent Variable
1	Base RMR20kg1ml
2	EPOC20kg12ml
3	EPOC20kg36ml

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR20kg1ml	3.6625	.43605	8
EPOC20kg12ml	3.7475	.38351	8
EPOC20kg36ml	3.8650	.63130	8

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Mauchly's Test of Sphericity[b]

Measure: Relative

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
pmRMR20kg	.978	.136	2	.934	.978	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: pmRMR20kg

Tests of Within-Subjects Effects

Measure: Relative

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
pmRMR20kg	Sphericity Assumed	.165	2	.083	.539	.595	.072
	Greenhouse-Geisser	.165	1.956	.085	.539	.591	.072
	Huynh-Feldt	.165	2.000	.083	.539	.595	.072
	Lower-bound	.165	1.000	.165	.539	.487	.072
Error(pmRMR20kg)	Sphericity Assumed	2.147	14	.153			
	Greenhouse-Geisser	2.147	13.692	.157			
	Huynh-Feldt	2.147	14.000	.153			
	Lower-bound	2.147	7.000	.307			

Estimated Marginal Means

pmRMR20kg

Estimates

Measure: Relative

pmRMR20kg	Mean	Std. Error	95% Confidence Interval Lower Bound	Upper Bound
1	3.663	.154	3.298	4.027
2	3.748	.136	3.427	4.068
3	3.865	.223	3.337	4.393

Pairwise Comparisons

Measure: Relative

(I) pmRMR20kg	(J) pmRMR20kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a] Lower Bound	Upper Bound
1	2	-.085	.189	1.000	-.675	.505
	3	-.203	.188	.952	-.791	.386
2	1	.085	.189	1.000	-.505	.675
	3	-.118	.210	1.000	-.774	.539
3	1	.203	.188	.952	-.386	.791
	2	.118	.210	1.000	-.539	.774

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRMR20kg1L EPOC20kg12L EPOC20kg36L
  /WSFACTOR = pmRMR20kg 3 Polynomial
  /MEASURE = Absolute
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( pmRMR20kg )
  /EMMEANS = TABLES(pmRMR20kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE ETASQ
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = pmRMR20kg .
```

General Linear Model

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Within-Subjects Factors

Measure: Absolute

pmRMR20kg	Dependent Variable
1	Base RMR20kg1L
2	EPOC20kg12L
3	EPOC20kg36L

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRMR20kg1L	.3250	.05657	8
EPOC20kg12L	.3300	.04781	8
EPOC20kg36L	.3400	.06633	8

Mauchly's Test of Sphericity[b]

Measure: Absolute

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser	Huynh-Feldt	Lower-bound
					Epsilon[a]		
pmRMR20kg	.964	.219	2	.896	.965	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: pmRMR20kg

Tests of Within-Subjects Effects

Measure: Absolute

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
pmRMR20kg	Sphericity Assumed	.001	2	.000	.434	.657	.058
	Greenhouse-Geisser	.001	1.931	.000	.434	.650	.058
	Huynh-Feldt	.001	2.000	.000	.434	.657	.058
	Lower-bound	.001	1.000	.001	.434	.531	.058
Error(pmRMR20kg)	Sphericity Assumed	.015	14	.001			
	Greenhouse-Geisser	.015	13.516	.001			
	Huynh-Feldt	.015	14.000	.001			
	Lower-bound	.015	7.000	.002			

Estimated Marginal Means

pmRMR20kg

Estimates

Measure: Absolute

pmRMR20kg	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.325	.020	.278	.372
2	.330	.017	.290	.370
3	.340	.023	.285	.395

Pairwise Comparisons

Measure: Absolute

(I) pmRMR20kg	(J) pmRMR20kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a] Lower Bound	Upper Bound
1	2	-.005	.016	1.000	-.055	.045
	3	-.015	.015	1.000	-.063	.033
2	1	.005	.016	1.000	-.045	.055
	3	-.010	.018	1.000	-.066	.046
3	1	.015	.015	1.000	-.033	.063
	2	.010	.018	1.000	-.046	.066

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRER10kg2 RER10kg24h RER10kg48h
  /WSFACTOR = amRER 3 Polynomial
  /MEASURE = C02toO2ratio
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( amRER )
  /EMMEANS = TABLES(amRER) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = amRER .
```

General Linear Model

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Within-Subjects Factors

Measure: C02toO2ratio

amRER	Dependent Variable
1	Base RER10kg2
2	RER10kg24h
3	RER10kg48h

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRER10kg2	.8925	.05726	8
RER10kg24h	.8588	.04016	8
RER10kg48h	.8900	.05292	8

Mauchly's Test of Sphericity[b]

Measure: C02toO2ratio

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
amRER	.634	2.739	2	.254	.732	.877	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: amRER

Tests of Within-Subjects Effects

Measure: C02toO2ratio

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
amRER	Sphericity Assumed	.006	2	.003	4.787	.026
	Greenhouse-Geisser	.006	1.464	.004	4.787	.042
	Huynh-Feldt	.006	1.754	.003	4.787	.033
	Lower-bound	.006	1.000	.006	4.787	.065
Error(amRER)	Sphericity Assumed	.008	14	.001		
	Greenhouse-Geisser	.008	10.245	.001		
	Huynh-Feldt	.008	12.275	.001		
	Lower-bound	.008	7.000	.001		

Estimated Marginal Means

amRER

Estimates

Measure: C02toO2ratio

amRER	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.893	.020	.845	.940
2	.859	.014	.825	.892
3	.890	.019	.846	.934

Pairwise Comparisons

Measure: C02toO2ratio

(I) amRER	(J) amRER	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a] Lower Bound	95% Confidence Interval for Difference[a] Upper Bound
1	2	.034	.012	.077	-.004	.071
	3	.003	.015	1.000	-.045	.050
2	1	-.034	.012	.077	-.071	.004
	3	-.031*	.009	.024	-.058	-.005
3	1	-.003	.015	1.000	-.050	.045
	2	.031*	.009	.024	.005	.058

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRER10kg1 RER10kg12h RER10kg36h
  /WSFACTOR = pmRER 3 Polynomial
  /MEASURE = C02toO2ratio
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( pmRER )
  /EMMEANS = TABLES(pmRER) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = pmRER .
```

General Linear Model

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Within-Subjects Factors

Measure: C02toO2ratio

pmRER	Dependent Variable
1	Base RER10kg1
2	RER10kg12h
3	RER10kg36h

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRER10kg1	.8900	.10296	8
RER10kg12h	.8725	.10457	8
RER10kg36h	.8738	.08943	8

Mauchly's Test of Sphericity[b]

Measure: C02toO2ratio

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser	Huynh-Feldt	Lower-bound
					Epsilon[a]		
pmRER	.625	2.818	2	.244	.727	.869	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: pmRER

Tests of Within-Subjects Effects

Measure: C02toO2ratio

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
pmRER	Sphericity Assumed	.002	2	.001	.511	.610
	Greenhouse-Geisser	.002	1.455	.001	.511	.558
	Huynh-Feldt	.002	1.738	.001	.511	.587
	Lower-bound	.002	1.000	.002	.511	.498
Error(pmRER)	Sphericity Assumed	.021	14	.001		
	Greenhouse-Geisser	.021	10.183	.002		
	Huynh-Feldt	.021	12.166	.002		
	Lower-bound	.021	7.000	.003		

Estimated Marginal Means

pmRER

Estimates

Measure: C02toO2ratio

pmRER	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.890	.036	.804	.976
2	.873	.037	.785	.960
3	.874	.032	.799	.949

Pairwise Comparisons

Measure: C02toO2ratio

(I) pmRER	(J) pmRER	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.018	.019	1.000	-.042	.077
	3	.016	.013	.802	-.026	.058
2	1	-.018	.019	1.000	-.077	.042
	3	-.001	.024	1.000	-.076	.074
3	1	-.016	.013	.802	-.058	.026
	2	.001	.024	1.000	-.074	.076

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.


```
GLM
  BaseRER20kg2 RER20kg24h RER20kg48h
  /WSFACTOR = amRER 3 Polynomial
  /MEASURE = C02toO2ratio
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( amRER )
  /EMMEANS = TABLES(amRER) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = amRER .
```

General Linear Model

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Within-Subjects Factors

Measure: C02toO2ratio

amRER	Dependent Variable
1	Base RER20kg2
2	RER20kg24h
3	RER20kg48h

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRER20kg2	.9025	.10727	8
RER20kg24h	.8725	.08379	8
RER20kg48h	.9025	.08345	8

Mauchly's Test of Sphericity[b]

Measure: C02toO2ratio

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhous e-Geisser	Huynh-Feldt	Lower-bound
					Epsilon[a]		
amRER	.672	2.387	2	.303	.753	.914	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: amRER

Tests of Within-Subjects Effects

Measure: C02toO2ratio

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
amRER	Sphericity Assumed	.005	2	.002	4.541	.030
	Greenhouse-Geisser	.005	1.506	.003	4.541	.046
	Huynh-Feldt	.005	1.829	.003	4.541	.035
	Lower-bound	.005	1.000	.005	4.541	.071
Error(amRER)	Sphericity Assumed	.007	14	.001		
	Greenhouse-Geisser	.007	10.540	.001		
	Huynh-Feldt	.007	12.800	.001		
	Lower-bound	.007	7.000	.001		

Estimated Marginal Means

amRER

Estimates

Measure: C02toO2ratio

amRER	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.903	.038	.813	.992
2	.873	.030	.802	.943
3	.903	.030	.833	.972

Pairwise Comparisons

Measure: C02toO2ratio

(I) amRER	(J) amRER	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	.030	.013	.150	-.010	.070
	3	.000	.013	1.000	-.042	.042
2	1	-.030	.013	.150	-.070	.010
	3	-.030*	.008	.016	-.054	-.006
3	1	.000	.013	1.000	-.042	.042
	2	.030*	.008	.016	.006	.054

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseRER20kg1 RER20kg12h RER20kg36h
  /WSFACTOR = pmRER 3 Polynomial
  /MEASURE = C02toO2ratio
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( pmRER )
  /EMMEANS = TABLES(pmRER) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = pmRER .
```

General Linear Model

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Within-Subjects Factors

Measure: C02toO2ratio

pmRER	Dependent Variable
1	Base RER20kg1
2	RER20kg12h
3	RER20kg36h

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRER20kg1	.864	.1090	8
RER20kg12h	.879	.1058	8
RER20kg36h	.876	.1116	8

Mauchly's Test of Sphericity[b]

Measure: C02toO2ratio

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
pmRER	.839	1.050	2	.592	.862	1.000	.500

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: pmRER

Tests of Within-Subjects Effects

Measure: C02toO2ratio

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
pmRER	Sphericity Assumed	.001	2	.001	.421	.664
	Greenhouse-Geisser	.001	1.723	.001	.421	.636
	Huynh-Feldt	.001	2.000	.001	.421	.664
	Lower-bound	.001	1.000	.001	.421	.537
Error(pmRER)	Sphericity Assumed	.017	14	.001		
	Greenhouse-Geisser	.017	12.063	.001		
	Huynh-Feldt	.017	14.000	.001		
	Lower-bound	.017	7.000	.002		

Estimated Marginal Means

pmRER

Estimates

Measure: C02toO2ratio

pmRER	Mean	Std. Error	95% Confidence Interval Lower Bound	Upper Bound
1	.864	.039	.773	.955
2	.879	.037	.790	.967
3	.876	.039	.783	.970

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Pairwise Comparisons

Measure: C02toO2ratio

(I) pmRER	(J) pmRER	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.015	.017	1.000	-.067	.037
	3	-.013	.021	1.000	-.077	.052
2	1	.015	.017	1.000	-.037	.067
	3	.003	.015	1.000	-.044	.049
3	1	.013	.021	1.000	-.052	.077
	2	-.003	.015	1.000	-.049	.044

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

CORRELATIONS
 /VARIABLES=Weight EPOC10kg12ml
 /PRINT=TWOTAIL NOSIG
 /STATISTICS DESCRIPTIVES
 /MISSING=PAIRWISE.

Correlations

Notes

Output Created		14-Jan-2009 13:11:38
Comments		
Input	Data	C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	8
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.

Syntax		CORRELATIONS
		/VARIABLES=Weight EPOC10kg12ml
		/PRINT=TWOTAIL NOSIG
		/STATISTICS DESCRIPTIVES
		/MISSING=PAIRWISE.
Resources	Processor Time	00:00:00.047
	Elapsed Time	00:00:00.032

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8
EPOC10kg12ml	3.7488	.59595	8

Correlations

		Weight	EPOC10kg12ml
Weight	Pearson Correlation	1.000	.170
	Sig. (2-tailed)		.687
	N	8	8
EPOC10kg12ml	Pearson Correlation	.170	1.000
	Sig. (2-tailed)	.687	
	N	8	8

```
* Chart Builder.
GGRAPH
 /GRAPHDATASET NAME="graphdataset" VARIABLES=EPOC10kg12ml Weight MISSING=LISTWISE
REPORTMISSING=NO
 /GRAPHSPEC SOURCE=INLINE.
BEGIN GPL
 SOURCE: s=userSource(id("graphdataset"))
 DATA: EPOC10kg12ml=col(source(s), name("EPOC10kg12ml"))
 DATA: Weight=col(source(s), name("Weight"))
 GUIDE: axis(dim(1), label("EPOC10kg12ml"))
 GUIDE: axis(dim(2), label("Weight"))
 ELEMENT: point(position(EPOC10kg12ml*Weight))
END GPL.
CORRELATIONS
```

```
/VARIABLES=Weight EPOC10kg24L
/PRINT=TWOTAIL NOSIG
/STATISTICS DESCRIPTIVES
/MISSING=PAIRWISE.
```

Correlations

Notes

Output Created		14-Jan-2009 13:17:36
Comments		
Input	Data	C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	8
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=Weight EPOC10kg24L /PRINT=TWOTAIL NOSIG /STATISTICS DESCRIPTIVES /MISSING=PAIRWISE.
Resources	Processor Time	00:00:00.110
	Elapsed Time	00:00:00.063

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8
EPOC10kg24L	.2988	.04794	8

Correlations

		Weight	EPOC10kg24L
Weight	Pearson Correlation	1.000	.626
	Sig. (2-tailed)		.097
	N	8	8
EPOC10kg24L	Pearson Correlation	.626	1.000
	Sig. (2-tailed)	.097	
	N	8	8

Correlations

Notes

Output Created		14-Jan-2009 13:15:13
Comments		
Input	Data	C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	8
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.

Syntax		CORRELATIONS
		/VARIABLES=Weight EPOC10kg36ml
		/PRINT=TWOTAIL NOSIG
		/STATISTICS DESCRIPTIVES
		/MISSING=PAIRWISE.
Resources	Processor Time	00:00:00.094
	Elapsed Time	00:00:00.047

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8
EPOC10kg36ml	3.8713	.59772	8

Correlations

		Weight	EPOC10kg36ml
Weight	Pearson Correlation	1.000	.059
	Sig. (2-tailed)		.890
	N	8	8
EPOC10kg36ml	Pearson Correlation	.059	1.000
	Sig. (2-tailed)	.890	
	N	8	8

```
* Chart Builder.
GGRAPH
 /GRAPHDATASET NAME="graphdataset" VARIABLES=EPOC10kg36ml Weight MISSING=LISTWISE
REPORTMISSING=NO
 /GRAPHSPEC SOURCE=INLINE.
BEGIN GPL
 SOURCE: s=userSource(id("graphdataset"))
 DATA: EPOC10kg36ml=col(source(s), name("EPOC10kg36ml"))
 DATA: Weight=col(source(s), name("Weight"))
 GUIDE: axis(dim(1), label("EPOC10kg36ml"))
 GUIDE: axis(dim(2), label("Weight"))
 ELEMENT: point(position(EPOC10kg36ml*Weight))
END GPL.
CORRELATIONS
```

```
/VARIABLES=Weight EPOC10kg48ml
/PRINT=TWOTAIL NOSIG
/STATISTICS DESCRIPTIVES
/MISSING=PAIRWISE.
```

Correlations

Notes

Output Created		14-Jan-2009 13:18:01
Comments		
Input	Data	C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	8
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=Weight EPOC10kg48ml /PRINT=TWOTAIL NOSIG /STATISTICS DESCRIPTIVES /MISSING=PAIRWISE.
Resources	Processor Time	00:00:00.062
	Elapsed Time	00:00:00.048

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8
EPOC10kg48ml	3.3862	.30355	8

Correlations

		Weight	EPOC10kg48ml
Weight	Pearson Correlation	1.000	.252
	Sig. (2-tailed)		.547
	N	8	8
EPOC10kg48ml	Pearson Correlation	.252	1.000
	Sig. (2-tailed)	.547	
	N	8	8

Correlations

Notes

Output Created		30-Jan-2009 17:58:58
Comments		
Input	Data	C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav
	Active Dataset	DataSet2
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	8
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.

Syntax		CORRELATIONS
		/VARIABLES=Weight EPOC20kg12ml
		/PRINT=TWOTAIL NOSIG
		/STATISTICS DESCRIPTIVES
		/MISSING=PAIRWISE.
Resources	Processor Time	00:00:00.078
	Elapsed Time	00:00:00.140

[DataSet2] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8
EPOC20kg12ml	3.7475	.38351	8

Correlations

		Weight	EPOC20kg12ml
Weight	Pearson Correlation	1.000	.033
	Sig. (2-tailed)		.939
	N	8	8
EPOC20kg12ml	Pearson Correlation	.033	1.000
	Sig. (2-tailed)	.939	
	N	8	8

CORRELATIONS
 /VARIABLES=Weight EPOC20kg12ml
 /PRINT=TWOTAIL NOSIG
 /STATISTICS DESCRIPTIVES
 /MISSING=PAIRWISE.

Correlations

Notes

Output Created		14-Jan-2009 13:18:35
Comments		
Input	Data	C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	8
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=Weight EPOC20kg24ml /PRINT=TWOTAIL NOSIG /STATISTICS DESCRIPTIVES /MISSING=PAIRWISE.
Resources	Processor Time	00:00:00.094
	Elapsed Time	00:00:00.047

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8
EPOC20kg24ml	3.3875	.33737	8

Correlations

		Weight	EPOC20kg24ml
Weight	Pearson Correlation	1.000	.379

			.354
	N	8	8
EPOC20kg24ml	Pearson Correlation	.379	1.000
	Sig. (2-tailed)	.354	
	N	8	8

```
CORRELATIONS
 /VARIABLES=Weight EPOC20kg24ml
 /PRINT=TWOTAIL NOSIG
 /STATISTICS DESCRIPTIVES
 /MISSING=PAIRWISE.
```

Correlations

Notes

Output Created		14-Jan-2009 13:16:39
Comments		
Input	Data	C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	8
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=Weight EPOC20kg36L /PRINT=TWOTAIL NOSIG /STATISTICS DESCRIPTIVES /MISSING=PAIRWISE.
Resources	Processor Time	00:00:00.094
	Elapsed Time	00:00:00.046

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[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8
EPOC20kg36L	.3400	.06633	8

Correlations

		Weight	EPOC20kg36L
Weight	Pearson Correlation	1.000	.520
	Sig. (2-tailed)		.186
	N	8	8
EPOC20kg36L	Pearson Correlation	.520	1.000
	Sig. (2-tailed)	.186	
	N	8	8

```
CORRELATIONS
 /VARIABLES=Weight EPOC20kg48ml
 /PRINT=TWOTAIL NOSIG
 /STATISTICS DESCRIPTIVES
 /MISSING=PAIRWISE.
```

Correlations

Notes

Output Created		14-Jan-2009 13:19:14
Comments		
Input	Data	C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>

	Split File	<none>
	N of Rows in Working Data File	8
Missing Value Handling	Definition of Missing	User-defined missing values are treated as missing.
	Cases Used	Statistics for each pair of variables are based on all the cases with valid data for that pair.
Syntax		CORRELATIONS /VARIABLES=Weight EPOC20kg48ml /PRINT=TWOTAIL NOSIG /STATISTICS DESCRIPTIVES /MISSING=PAIRWISE.
Resources	Processor Time	00:00:00.063
	Elapsed Time	00:00:00.046

[DataSet1] C:\Documents and Settings\gabboud\My Documents\Dissertation\Correct Data II.sav

Descriptive Statistics

	Mean	Std. Deviation	N
Weight	87.9900	8.70611	8
EPOC20kg48ml	3.5338	.59042	8

Correlations

		Weight	EPOC20kg48ml
Weight	Pearson Correlation	1.000	-.130
	Sig. (2-tailed)		.759
	N	8	8
EPOC20kg48ml	Pearson Correlation	-.130	1.000
	Sig. (2-tailed)	.759	
	N	8	8

```
GLM
  BaselineCK10kg2 IPECK10kg CK10kg12h CK10kg24h CK10kg36h CK10kg48
```

```
/WSFACTOR = MuscleDamage 6 Polynomial
/MEASURE = CreatineKinase
/METHOD = SSTYPE(3)
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/PRINT = DESCRIPTIVE
/CRITERIA = ALPHA(.05)
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```

General Linear Model

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Within-Subjects Factors

Measure: CreatineKinase

MuscleDamage	Dependent Variable
1	Baseline CK10kg2
2	IPECK10kg
3	CK10kg12h
4	CK10kg24h
5	CK10kg36h
6	CK10kg48

Descriptive Statistics

	Mean	Std. Deviation	N
BaselineCK10kg2	309.013	295.2505	8
IPECK10kg	398.288	344.3001	8
CK10kg12h	728.450	524.0688	8
CK10kg24h	561.325	399.9020	8
CK10kg36h	492.338	326.3130	8
CK10kg48	329.638	188.7325	8

Mauchly's Test of Sphericity[b]

Measure: CreatineKinase

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
MuscleDamage	.000	44.905	14	.000	.425	.615	.200

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: MuscleDamage

Tests of Within-Subjects Effects

Measure: CreatineKinase

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
MuscleDamage	Sphericity Assumed	1011174.087	5	202234.817	6.362	.000
	Greenhouse-Geisser	1011174.087	2.123	476216.073	6.362	.009
	Huynh-Feldt	1011174.087	3.073	329032.004	6.362	.003
	Lower-bound	1011174.087	1.000	1011174.087	6.362	.040
Error(MuscleDamage)	Sphericity Assumed	1112563.970	35	31787.542		
	Greenhouse-Geisser	1112563.970	14.863	74852.286		
	Huynh-Feldt	1112563.970	21.512	51717.695		
	Lower-bound	1112563.970	7.000	158937.710		

Tests of Between-Subjects Effects

Measure: CreatineKinase
Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	10596057.2	1	10596057.20	16.996	.004
Error	4364132.280	7	623447.469		

Estimated Marginal Means

MuscleDamage

Estimates

Measure: CreatineKinase

MuscleDamage	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	309.013	104.387	62.177	555.848
2	398.288	121.728	110.445	686.130
3	728.450	185.286	290.318	1166.582
4	561.325	141.387	226.999	895.651
5	492.338	115.369	219.533	765.142
6	329.638	66.727	171.853	487.422

Pairwise Comparisons

Measure: CreatineKinase

(I) MuscleDamage	(J) MuscleDamage	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-89.275	22.358	.079	-186.651	8.101
	3	-419.438	106.864	.086	-884.860	45.985
	4	-252.313	92.867	.448	-656.772	152.147
	5	-183.325	92.951	1.000	-588.150	221.500
	6	-20.625	79.871	1.000	-368.485	327.235
2	1	89.275	22.358	.079	-8.101	186.651
	3	-330.163	96.693	.168	-751.286	90.961
	4	-163.038	97.867	1.000	-589.277	263.202
	5	-94.050	103.532	1.000	-544.962	356.862
	6	68.650	95.812	1.000	-348.639	485.939
3	1	419.438	106.864	.086	-45.985	884.860
	2	330.163	96.693	.168	-90.961	751.286
	4	167.125	72.786	.830	-149.880	484.130
	5	236.113	103.068	.836	-212.775	685.000
	6	398.813	134.463	.314	-186.810	984.435
4	1	252.313	92.867	.448	-152.147	656.772
	2	163.038	97.867	1.000	-263.202	589.277
	3	-167.125	72.786	.830	-484.130	149.880
	5	68.988	40.986	1.000	-109.519	247.494
	6	231.688	81.023	.365	-121.188	584.563
5	1	183.325	92.951	1.000	-221.500	588.150
	2	94.050	103.532	1.000	-356.862	544.962
	3	-236.113	103.068	.836	-685.000	212.775
	4	-68.988	40.986	1.000	-247.494	109.519
	6	162.700	50.676	.223	-58.008	383.408
6	1	20.625	79.871	1.000	-327.235	368.485
	2	-68.650	95.812	1.000	-485.939	348.639
	3	-398.813	134.463	.314	-984.435	186.810
	4	-231.688	81.023	.365	-584.563	121.188
	5	-162.700	50.676	.223	-383.408	58.008

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
  BaseCK20kg2 IPECK20kg CK20kg12h CK20kg24h CK20kg36h CK20kg48h
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  /MEASURE = CreatineKinase
  /METHOD = SSTYPE(3)
  /PLOT = PROFILE( MuscleDamage20kg )
  /EMMEANS = TABLES(MuscleDamage20kg) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = MuscleDamage20kg .
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General Linear Model

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Within-Subjects Factors

Measure: CreatineKinase

MuscleDamage20kg	Dependent Variable
1	Base CK20kg2
2	IPECK20kg
3	CK20kg12h
4	CK20kg24h
5	CK20kg36h
6	CK20kg48h

Descriptive Statistics

	Mean	Std. Deviation	N
BaseCK20kg2	272.163	279.6116	8
IPECK20kg	490.013	401.8173	8
CK20kg12h	1159.425	729.3767	8
CK20kg24h	980.838	652.5022	8
CK20kg36h	774.113	587.5499	8
CK20kg48h	506.000	356.4704	8

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
MuscleDamage20kg	Pillai's Trace	.865	3.831[a]	5.000	3.000	.149
	Wilks' Lambda	.135	3.831[a]	5.000	3.000	.149
	Hotelling's Trace	6.385	3.831[a]	5.000	3.000	.149
	Roy's Largest Root	6.385	3.831[a]	5.000	3.000	.149

a. Exact statistic

b.
Design: Intercept
Within Subjects Design: MuscleDamage20kg

Mauchly's Test of Sphericity[b]

Measure: CreatineKinase

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
MuscleDamage20kg	.000	47.580	14	.000	.404	.570	.200

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.

Design: Intercept
Within Subjects Design: MuscleDamage20kg

Tests of Within-Subjects Effects

Measure: CreatineKinase

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
MuscleDamage20kg	Sphericity Assumed	4481268.967	5	896253.793	9.681	.000
	Greenhouse-Geisser	4481268.967	2.022	2216192.720	9.681	.002
	Huynh-Feldt	4481268.967	2.848	1573559.292	9.681	.000
	Lower-bound	4481268.967	1.000	4481268.967	9.681	.017
Error(Muscle Damage20kg)	Sphericity Assumed	3240223.853	35	92577.824		
	Greenhouse-Geisser	3240223.853	14.154	228919.868		
	Huynh-Feldt	3240223.853	19.935	162539.558		
	Lower-bound	3240223.853	7.000	462889.122		

Estimated Marginal Means

MuscleDamage20kg

Estimates

Measure: CreatineKinase

MuscleDamage20kg	Mean	Std. Error	95% Confidence Interval Lower Bound	Upper Bound
1	272.163	98.858	38.401	505.924
2	490.013	142.064	154.085	825.940
3	1159.425	257.874	549.651	1769.199
4	980.838	230.694	435.332	1526.343
5	774.113	207.730	282.908	1265.317
6	506.000	126.031	207.983	804.017

Pairwise Comparisons

Measure: CreatineKinase

(I) MuscleDamage20kg	(J) MuscleDamage20kg	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-217.850	54.223	.076	-454.006	18.306
	3	-887.263*	175.737	.022	-1652.645	-121.880
	4	-708.675	179.698	.084	-1491.311	73.961
	5	-501.950	193.635	.537	-1345.282	341.382
	6	-233.838	125.006	1.000	-778.272	310.597
2	1	217.850	54.223	.076	-18.306	454.006
	3	-669.413*	127.714	.018	-1225.644	-113.181
	4	-490.825	161.190	.281	-1192.850	211.200
	5	-284.100	193.003	1.000	-1124.682	556.482
	6	-15.988	141.398	1.000	-631.813	599.838
3	1	887.263*	175.737	.022	121.880	1652.645
	2	669.413*	127.714	.018	113.181	1225.644
	4	178.588	131.293	1.000	-393.230	750.405
	5	385.313	202.264	1.000	-495.604	1266.229
	6	653.425	201.408	.213	-223.764	1530.614
4	1	708.675	179.698	.084	-73.961	1491.311
	2	490.825	161.190	.281	-211.200	1192.850
	3	-178.588	131.293	1.000	-750.405	393.230
	5	206.725	87.247	.745	-173.260	586.710
	6	474.838	121.783	.089	-55.561	1005.236
5	1	501.950	193.635	.537	-341.382	1345.282
	2	284.100	193.003	1.000	-556.482	1124.682
	3	-385.313	202.264	1.000	-1266.229	495.604
	4	-206.725	87.247	.745	-586.710	173.260
	6	268.113	85.072	.242	-102.401	638.626
6	1	233.838	125.006	1.000	-310.597	778.272
	2	15.988	141.398	1.000	-599.838	631.813
	3	-653.425	201.408	.213	-1530.614	223.764
	4	-474.838	121.783	.089	-1005.236	55.561
	5	-268.113	85.072	.242	-638.626	102.401

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Bonferroni.

```
GLM
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  /EMMEANS = TABLES(MuscleDamage) COMPARE ADJ(BONFERRONI)
  /PRINT = DESCRIPTIVE
  /CRITERIA = ALPHA(.05)
  /WSDESIGN = MuscleDamage .
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General Linear Model

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Within-Subjects Factors

Measure: RPMS

MuscleDamage	Dependent Variable
1	Base RPMS20kg2
2	RPMS20kg12h
3	RPMS20kg24h
4	RPMS20kg36h
5	RPMS20kg48h

Descriptive Statistics

	Mean	Std. Deviation	N
BaseRPMS20kg2	.288	.2949	8
RPMS20kg12h	1.200	1.3005	8
RPMS20kg24h	1.213	1.5338	8
RPMS20kg36h	2.075	2.1585	8
RPMS20kg48h	1.475	1.7589	8

Mauchly's Test of Sphericity[b]

Measure: RPMS

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a] Greenhouse-Geisser	Huynh-Feldt	Lower-bound
MuscleDamage	.045	16.764	9	.063	.595	.923	.250

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: MuscleDamage

Tests of Within-Subjects Effects

Measure: RPMS

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
MuscleDamage	Sphericity Assumed	13.293	4	3.323	3.322	.024
	Greenhouse-Geisser	13.293	2.381	5.582	3.322	.054
	Huynh-Feldt	13.293	3.692	3.601	3.322	.028
	Lower-bound	13.293	1.000	13.293	3.322	.111
Error(MuscleDamage)	Sphericity Assumed	28.012	28	1.000		
	Greenhouse-Geisser	28.012	16.669	1.680		
	Huynh-Feldt	28.012	25.841	1.084		
	Lower-bound	28.012	7.000	4.002		

Estimated Marginal Means

MuscleDamage

Estimates

Measure: RPMS

MuscleDamage	Mean	Std. Error	95% Confidence Interval	
			Lower Bound	Upper Bound
1	.288	.104	.041	.534
2	1.200	.460	.113	2.287
3	1.213	.542	-.070	2.495
4	2.075	.763	.270	3.880
5	1.475	.622	.005	2.945

Pairwise Comparisons

Measure: RPMS

(I) MuscleDamage	(J) MuscleDamage	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.913	.405	.588	-2.543	.718
	3	-.925	.513	1.000	-2.993	1.143
	4	-1.788	.679	.339	-4.525	.950
	5	-1.188	.525	.580	-3.301	.926
2	1	.913	.405	.588	-.718	2.543
	3	-.013	.406	1.000	-1.646	1.621
	4	-.875	.554	1.000	-3.109	1.359
	5	-.275	.436	1.000	-2.033	1.483
3	1	.925	.513	1.000	-1.143	2.993
	2	.013	.406	1.000	-1.621	1.646
	4	-.863	.518	1.000	-2.948	1.223
	5	-.263	.572	1.000	-2.568	2.043
4	1	1.788	.679	.339	-.950	4.525
	2	.875	.554	1.000	-1.359	3.109
	3	.863	.518	1.000	-1.223	2.948
	5	.600	.282	.711	-.537	1.737
5	1	1.188	.525	.580	-.926	3.301
	2	.275	.436	1.000	-1.483	2.033
	3	.263	.572	1.000	-2.043	2.568
	4	-.600	.282	.711	-1.737	.537

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

Correlations

Notes

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Correlations

		EPOC10kg48ml	CK10kg48
EPOC10kg48ml	Pearson Correlation	1.000	.755[*]
	Sig. (2-tailed)		.030
	N	8	8
CK10kg48	Pearson Correlation	.755[*]	1.000
	Sig. (2-tailed)	.030	
	N	8	8

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

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BIOGRAPHICAL SKETCH

GEORGE JOSEPH ABBOUD

Personal Data:
    Date of Birth: December 26, 1966
    Place of Birth: Passaic, NJ
    Marital Status: Married

Education:
    Montclair State College, Upper Montclair, NJ;
        Speech and Theater, BFA, 1992
    Montclair State University, Upper Montclair, NJ;
        Exercise Science, MA, 2001

Professional
Experience:
    Assistant Professor; Salem State College
        Salem, MA; 2006-Present

Presentations:
    Abboud, G., Dion, S. & Fuller, H. Teaching About Healthy Weight. AAHPERD Eastern District Conference. Newport, Rhode Island. Feb. 2008

    Abboud, G., Excess Post-exercise Oxygen Consumption and Heavy Resistance Training. Presented at Salem State College, Oral Presentation, Feb. 2007.

    Abboud, G., Panton, L., Toole, T., Holton, E., & Kingsley, D. Relationships of continuous scale-physical function performance indices (CS-PFP) to gait and balance parameters in older women. Abstract presented to the Southeast Regional American College of Sports Medicine. Atlanta, GA, Feb. 2003

    Abboud, G., Tobin, R., A Comparison of Strength in 20, 30, 40, and 50 year old trained weight lifters. Presented at Southeast Regional Chapter ACSM in Atlanta, GA. Oral presentation Feb. 2002

Professional
Associations:
    New England American College of Sports Medicine
    National Education Association
    Massachusetts Teachers Association
    Massachusetts State College Association

Activities:
    College Teaching Certification
    Red Cross CPR/ First Aid certified
    1LT U.S. Army National Guard, Medical Services
    Completed Project Adventure Training
        Adventure Programming
        Technical Skills Intensive