To the Graduate Council:

I am submitting herewith a thesis written by Robin Denise Eldridge entitled "The Relationship Between Lean Body Mass and Resting Metabolic Rate." I have examined the final copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Human Performance and Sport Studies.

Edward T. Howley

Dr. Edward T. Howley, Major Professor

We have read this thesis
and recommend its acceptance:

David R. Bassett Jr.

Jean Pleas

Accepted for the Council:

Associate Vice Chancellor
and Dean of The Graduate School

The Relationship Between Lean Body Mass and Resting Metabolic Rate

A Thesis

Presented for the

Master of Science

Degree

The University of Tennessee, Knoxville

Robin Denise Eldridge

December, 1992

DEDICATION

This thesis is dedicated to my parents, Alice and Jack Eldridge. Thank you for all of your love, patience, confidence and encouragement. Without the two of you, none of this would have been possible. I really appreciate all of the things that you have done for me. I could not have asked for two better parents. I love you both very much. You're the greatest!

ACKNOWLEDGMENTS

I thank my advisor, Dr. Edward T. Howley and my other committee members, Dr. David Bassett and Dr. Jean Lewis.

Dr. Howley's willingness to go out of his way to help me understand the "simplest little things" is greatly appreciated.

I thank my subjects, all of whom showed up when they were supposed to. Thanks for making my life less stressful.

I thank Dr. Chuck Farmer and Dr. Judith Flohr for all of their statistical help. A special thank you to Judi, for lending her shoulders to cry on.

Last, but certainly not least, I thank my parents. Without their emotional and financial support, none of this would have been possible.

ABSTRACT

When dealing with exercise and its effect on resting metabolic rate, researchers have been concerned mainly with endurance training. It would be interesting, however, to see how different types of training affect this variable. For example, how does an anaerobic activity such as resistance training influence a person's resting metabolism? Does it cause adaptations within the muscle such that the RMR (kcal· kg LBM^{-1}· hr^{-1}) is higher than it is in runners? The purpose of this study was to answer that question.

A cross-sectional study was conducted on twenty-four apparently healthy, non-smoking males between the ages of 18-30 years. These individuals were placed into one of three different groups, sedentary, endurance trained or resistance trained.

All subjects were instructed to report to the laboratory at 6:00 a.m., after 12 hours of fasting and 36 hours of inactivity. Upon arrival, resting metabolic rate, residual volume and body composition measurements were obtained. Each measurement was made on two separate occasions in order to show reproducibility in the data. Based on the correlation coefficients of these three variables (r= 0.88, r= 0.99 and r= 0.99), there appeared to be good reproducibility between the values of Trial 1 and Trial 2.

A one-way ANOVA showed that the RMRs (kcal· kg LBM^{-1}· hr^{-1}) of sedentary people (1.32 ± 0.15) were significantly higher than those of endurance trained individuals (1.17 ± 0.11) and resistance trained individuals (1.15 ± 0.07). No significant difference was found to exist, however, between the latter two groups. Apparently, the two types of training had similar effects on resting metabolism (kcal· kg LBM^{-1}· hr^{-1}).

TABLE OF CONTENTS

LIST OF TABLES

TABLE **PAGE**

LIST OF FIGURES

FIGURE **PAGE**

CHAPTER I

INTRODUCTION

Metabolism can be defined as the process of chemical changes by which energy is provided for the maintenance of life [35]. It is believed to be influenced by a number of dynamic components such as the acute effects of exercise, diet, emotional status, ambient temperature, body size and age. Although gender is not dynamic in nature, it is also thought to elicit an effect on metabolism [27].

There are three major components of daily energy expenditure. They include resting metabolic rate (RMR), the thermic effect of feeding (TEF) and the thermic effect of activity (TEA). Of the three, resting metabolism represents the greatest portion of expenditure, accounting for 60-75% of the daily total in man [34].

"Resting metabolic rate constitutes the energy required to maintain body temperature and electrolyte gradients, to sustain cardiovascular and pulmonary work at rest, and to provide the energy used by the central nervous system and other chemical reactions" [9,34]. Given the functions of RMR, it is not surprising that it represents such a large percentage of daily energy expenditure.

The thermic effect of food is a second component of daily energy expenditure. It represents the increase in energy expenditure above RMR, upon the ingestion of a meal. TEF constitutes approximately 10% of the daily expenditure and includes the energy cost associated with the ingestion, absorption, metabolism and storage of food within the body [34].

The final component of daily energy expenditure is the thermic effect of activity. It is influenced by purposeful physical exercise, daily activities such as walking from place to place, and even muscular activity such as fidgeting. In short TEA accounts for all of the additional energy expended above RMR and TEF, and it also represents

the most variable component of expenditure. With respect to sedentary individuals, the thermic effect of activity is generally about 15% of the total daily energy expenditure. In individuals who regularly engage in physical activity, however, it has been found to contribute up to 30% of this total [34].

Although there are three different components of daily energy expenditure, researchers have been most concerned with resting metabolic rate. More specifically, they have been interested in the chronic effect that purposeful physical exercise may have on it. An overwhelming amount of evidence has recently begun to support the possibility of a relationship between these two variables [14,18,22,32,38,44-46]. If such a relationship really does exist, what exactly is it about physical activity that appears to influence RMR?

In search of an answer, researchers have been focusing more and more on the issue of lean body mass. Lean tissue is the most metabolically active tissue found within the body, and RMR has been shown to be strongly linked to it [5,6,26,27,32,34,47]. As a result, an individual who possesses a large amount of this particular tissue should have a higher absolute RMR (kcal $\cdot min^{-1}$) than a person who possesses a smaller amount of it. This does not necessarily mean, however, that the former individual will also have a higher RMR when expressed kcal$\cdot$ kg $LBM^{-1}\cdot$ hr^{-1}. In fact, differences between men and women disappear when resting metabolic rate is expressed in this manner.

Several studies have been conducted, comparing sedentary people to runners [14,18,22,32,38,44-46]. Based on the results of these investigations, it appears that trained individuals have the highest RMRs (kcal$\cdot$ kg $LBM^{-1}\cdot$ hr^{-1}). This suggests perhaps, that physiological changes occurring within muscle fibers themselves also influence resting metabolism. Simply put, there seems to be something different about

the lean tissue of a runner that requires a greater amount of energy to be expended for its maintenance.

Variations in resting metabolic rate have many practical implications. One of them deals with weight loss. At the present time, a variety of methods are used to reduce body weight. Some people try to reduce their weight solely by dieting. Others attempt to accomplish the same feat through exercise. The most successful weight loss program, however, incorporates both of these elements [5,6,17]. The reasons are simple. RMR is often reduced when dieting is the only method employed to achieve a specified weight loss. Purposeful physical exercise, however, has been shown to offset this reduction [3,5,6,17,21,26].

The chronic effects of such activity include an increase in the size and number of mitochondria contained within the lean tissue and an increase in capillary density. Many investigators believe that these changes enhance the metabolic quality of the lean tissue, resulting in the greater resting energy expenditure per kilogram of lean body tissue that trained athletes often exhibit [19,37].

When dealing with exercise and its effect on resting metabolic rate, researchers have been concerned mainly with endurance training. There is only one study that offers information about the effects of other types of training [6]. For instance, how does an anaerobic activity such as resistance training influence a person's resting metabolic rate? Does it cause adaptations within the muscle such that the RMR (kcal· kg LBM^{-1}· hr^{-1}) is higher than it is in runners? The purpose of this study was to answer that question.

CHAPTER II

REVIEW OF LITERATURE

ANIMAL STUDIES

Animal studies have commonly been used to help determine the relationship that exists between RMR and exercise. In 1970, for example, Terjung and Tipton measured the resting O_2 consumption of trained and untrained normal, hypophysectomized and thyroidectomized fasted male rats [43]. The data were collected at the end of a 10-week training program in which the rats ran for 1hr $\cdot$ day^{-1} at a speed of 1.6 km $\cdot$ hr^{-1}. Various endocrine systems have been shown to influence O_2 consumption. Based on the results of their study, however, Terjung and Tipton were unable to draw this conclusion. Initially, these researchers reasoned that because exercise could elicit an effect on the body's endocrine system, the highest RMRs should be seen in the normal rats. This, however, was not the case. Despite the presence or absence of certain endocrine glands, aerobic exercise did not appear to have a significant effect on resting metabolism.

Gleeson et al., on the other hand, found that such training did have a significant effect on RMR [14]. In this particular study, male Wistar rats were subjected to a 56 day training program in which they were required to run on an 8° incline at a speed of 0.9 km $\cdot$ hr^{-1}. Total exercise time was 1 hr $\cdot$ day^{-1}. At the conclusion of this training program, researchers had observed an overall rise of 10% in the resting metabolic rates of these rats.

The outcome of this particular study, however, was viewed with much uncertainty. Indirect calorimetry measurements were made too close to the last exercise session. Thus, it was unclear whether the increase in RMR was due to the chronic effects of physical conditioning or if it simply resulted from the residual effects of the previous exercise bout [34]. In search of a more definitive answer, a subsequent study

was conducted by Hill et al [18]. Their findings were in agreement with those of the previous study. According to these researchers, the effects of purposeful aerobic training did, indeed, appear to cause an elevation in RMR.

In this latter study, a physical conditioning program that consisted of swimming 2 hrs $\cdot$ day^{-1} for 148 to 168 days was used to elicit a training effect in twenty-two untrained female Charles River albino rats [18]. After being subjected to a period of fasting, initial resting metabolic rate measurements were obtained on each untrained rat. This variable was measured once again at the conclusion of the conditioning program. More specifically, it was obtained 24 hours upon completion. When expressed per kilogram of lean body mass, RMR was found to be higher in the trained state than it was in the untrained state. The 24-hour delay is of great importance. It should allow enough time for all thermic effects of activity to subside. As a result, any changes in RMR would likely be due to the chronic effects of physical conditioning and not the residual effects of the previous exercise session.

HUMAN STUDIES

Aside from the various animal studies that have been conducted, many experiments have also been done on humans [1,5,6,9,13,22,25,26,29-34,38,40,44-46]. To answer the question of whether or not the chronic effects of purposeful exercise have an effect on resting metabolism, researchers have been looking at the relationship that exists between RMR and lean body mass (LBM). Cross-sectional studies have frequently been used for this purpose.

When comparing two or more people, it is well known that the individual who possesses the greatest amount of LBM will have the highest absolute RMR, regardless of his conditioning level. More recently, researchers have become interested in the effect that purposeful physical exercise has on each kilogram of this lean tissue. Does the

individual who possesses the greatest amount of lean body tissue still have the highest RMR when expressed per kilogram of LBM? Or is it ultimately dependent upon their physical conditioning level?

Poehlman conducted a study in 1985 in which he compared the RMRs (kcal· kg LBM^{-1}· hr^{-1}) of 14 male long distance runners to those of 10 sedentary males [30]. Based on the results, he concluded that no significant difference existed between the two groups. Other research, however, indicates that trained individuals do, indeed, have higher RMRs per unit of lean body mass than their sedentary counterparts.

On two separate occasions, for example, resting metabolism (kcal· kg LBM^{-1}· hr^{-1}) was found to be, on average, 12% higher in trained athletes than it was in the untrained [22,32]. Similar results were obtained by Tremblay et al. [44,45], who found that trained individuals tended to have higher RMRs (kcal· kg LBM^{-1}· hr^{-1}). More specifically, in the two studies that they conducted, the values obtained on the trained athletes were found to be 19% and 6% higher, respectively. Finally, additional support comes from a study conducted by Hill et al. [18]. Based on the measurements that these researchers made, they found that the RMRs (kcal· kg LBM^{-1}· hr^{-1}) of trained subjects were 9% higher than those of untrained subjects.

The majority of these cross-sectional studies indicated that when compared to untrained or sedentary individuals, endurance trained athletes appear to have the highest RMRs (kcal· kg LBM^{-1}· hr^{-1}). It is very tempting to accept these findings and associate their existence with the different levels of physical conditioning found among the subjects. After all, only one of the available studies has shown anything different [30]. According to Mole´, however, when comparisons such as these are made between trained and untrained or sedentary individuals, the results must be analyzed cautiously. "Genetic differences and potential lack of important controls, including nutritional

status, physical activity patterns, and the degree of adaptation to training, may all lead to an erroneous interpretation of the data" [27].

Cross-sectional comparisons of resting metabolism in trained and untrained or sedentary subjects may be useful in detecting effects due to large differences in maximal aerobic power and training status. Training studies that attempt to assess changes in RMR, however, are potentially more informative. Unfortunately, conflicting results have been observed in the limited number of studies that have been performed.

Nine athletes who participated in either basketball, cross-country running, football or swimming were used as subjects in an early study conducted by Schneider and Foster [38]. The resting metabolic rate of each athlete was measured on before and after training. In the trained state, the RMR of seven athletes was found to be lower on average by 7% (ranging from 3% - 14%). The RMR of one cross-country runner, however, was found to be 12% higher in the trained state. Finally, with respect to the swimmers, no difference was found to exist between the two RMR values.

In addition to the athletes used in their study, Schneider and Foster also subjected three nonathletes to a 6-week training program [38]. Initial RMR measurements were obtained on each of these individuals before they began the program. The same measurement was made once again, at the conclusion of the training period. In comparing the two values, it became apparent that the program had a variable effect on each of the subjects. One individual, for example, experienced a decrease in RMR over the first 28-35 days of the study. By day 42, however, this value had risen. Resting metabolism was now 9% greater than it was in the untrained state. Another subject responded quite differently to the training program. An 11% increase in RMR was observed by day 14. Furthermore, at the end of the study, resting metabolism had achieved a value that was 22% greater than that of the untrained state. Finally, although not as great in

magnitude, the training program elicited an overall increase of 8% in the RMR of a third individual.

In 1986, Poehlman conducted a carefully controlled endurance training study that lasted 22 days [31]. Resting metabolic rate was measured at two different times. The first measurement was made before starting the program, and the second one was obtained 36 hours after completing it. Apparently, the training program had no effect on RMR (kcal· kg LBM^{-1}· hr^{-1}). When comparing the two values, it was obvious that this variable was unchanged. A more recent study that involved 10 lean and 10 obese men yielded similar results [40]. Each subject was required to ride the cycle ergometer at an intensity of 74% VO_2 max, 1 hr · day^{-1}, 4 times · $week^{-1}$. The training program lasted a total of 12 weeks. Initial RMR measurements were made before starting the program. Final RMR measurements were made 72 hours after completion of the last exercise session. Based on the results, RMR (kcal· kg LBM^{-1}· hr^{-1}) appeared to be unaltered by endurance training.

The longitudinal studies that have been discussed thus far indicate that exercise training does not generally cause an increase in RMR. Several other studies, however, state the contrary. Many researchers have found that exercise training does, indeed, lead to an increase in resting metabolism [22,32,33,45,46]. Six lean women were trained for 10 weeks in a study conducted by Lawson et al. [22]. During the first week, each was required to jog for 17 minutes, 3 times · $week^{-1}$. By week 10, however, these women were expected to progress to jogging for 77 minutes, 4 times · $week^{-1}$. Resting metabolism was initially measured prior to the onset of the training program. It did not change until the 4^{th} week of training. At this time, it was 12% higher on average than that of the sedentary control group. Furthermore, when measured at the end of week 10, these RMR values were found to be 13% higher than they were in the untrained state.

Somewhat similar results were also reported in an 11-week training study that involved 8 obese women [45]. Each subject's RMR was measured before the training program began and again 40-48 hours after its conclusion. A comparison of the two measurements indicated that RMR (kcal· kg LBM^{-1}· hr^{-1}) was 8% higher in the trained state.

The previous two studies found that resting metabolism increased with exercise training. In neither case, however, could this phenomenon be explained by a gain in lean tissue. Body composition measurements were made before and after the training periods of each study, and based on the results, there appeared to be no significant change in LBM. This finding suggests that energy requirements of the "active metabolic tissue" may be altered with aerobic exercise training.

Poehlman et al. found similar results [32]. In the absence of any increase in lean tissue, a higher RMR (kcal· kg LBM^{-1}· hr^{-1}) was still found to exist in highly trained male athletes of this cross-sectional study. Moreover, this effect persisted when the subjects were matched for body fat content. It appeared that differing amounts of adipose tissue had little impact on RMR. Instead, as these researchers suggested, perhaps high levels of physical training are the most important determinants of RMR.

Poehlman conducted another cross-sectional study in which he examined subjects with a wide range of fitness levels (40-80 ml· kg^{-1}· min^{-1}) [33]. He found a linear relationship between VO_2 max and RMR. Further, Poehlman discovered that the highest RMR values (kcal· kg LBM^{-1}· hr^{-1}) existed in the highly trained males (1.20 ± 0.04) relative to the moderately trained (1.06 ± 0.03) and untrained men (1.09 ± 0.03). These results gained additional support from Tremblay et al. [46]. In their study, higher RMRs were also evident in the group of trained athletes who possessed the highest VO_2 max values.

Some investigators have failed to observe this higher resting metabolism, even in trained individuals whose maximal aerobic power approaches 60 ml· kg^{-1}· min^{-1}. As Poehlman and Tremblay have shown, however, higher levels of maximal aerobic power and/or exercise training may be needed to observe these higher RMR (kcal· kg LBM^{-1}· hr^{-1}) [33,46]. The absence of this phenomenon may be due to the fact that an individual has a lower level of cardiorespiratory endurance.

It is obvious that the training studies discussed so far yield inconsistent results. Presently, researchers are still looking for answers to their question. Does chronic endurance training yield an increase in RMR (kcal· kg LBM^{-1}· hr^{-1})? With a solution in mind, many of these individuals have begun to incorporate some sort of dieting protocol into their studies. The rationale for this is simple. A reduction in caloric intake is known to decrease metabolism [29,47]. Thus, if endurance training really does have a chronic effect on resting metabolism (kcal· kg LBM^{-1}· hr^{-1}), there should be a noticeable reversal of the diminished RMR that was brought about by dietary restriction.

Warwick and Garrow, for example, conducted a metabolic ward study in which 3 obese women were placed on a reducing diet of about 800 kcal · day^{-1} for 12-13 weeks [47]. As a consequence, each subject's RMR (kJ · day^{-1}) was decreased. It was believed, however, that the initiation of a training program could eventually reverse this decline. The particular program that these women were placed on involved riding a cycle ergometer 2 hrs · day^{-1} for 3-4 weeks and resulted in the additional expenditure of 1640 ± 439 kJ · day^{-1}. The predicted outcome of this study did not occur. There was no reversal of the diminished RMR. It has been suggested that the exercise intensity was not great enough to produce a carry-over effect on this metabolic variable [27].

The relationship between diet, exercise and RMR has also been studied by Phinney et al. [29]. These researchers used 12 individuals to create 2 different groups, each consisting of 6 subjects. All twelve individuals were placed on a very low calorie diet, in which they consumed only 720 kcal $\cdot$ day^{-1}. In addition, one group was also expected to participate in an exercise program. It involved training at an intensity of 50% VO_2 max. The duration of this activity was 0.5 hr $\cdot$ day^{-1} for the first week, with a gradual progression to 1 hr $\cdot$ day^{-1} during the second week, and 2 hrs $\cdot$ day^{-1} throughout weeks three and four [29].

After 7 days, a 10% reduction was observed in the resting metabolism of both groups. The diet group remained at this depressed level for the next three weeks. RMR continued to fall, however, in the individuals who were also exercising. In fact, by the end of the study, there had been a 17% reduction in the RMR of this latter group. Apparently, the intensity and duration of exercise were not sufficient enough to result in a reversal of the declining RMR. Unfortunately, changes in body composition were not determined. As a result, it was not known whether the decrease in RMR was due to a reduction in the metabolic intensity of the active tissue (LBM), a loss of LBM, or both.

Genetic differences, timing of the exercise bout relative to metabolic measurements, different criteria to define trained and untrained individuals and different sample sizes have all been given as reasons for the conflicting results [34]. With respect to timing of the exercise bout, consider the study conducted by Segal [40]. Although the intensity of exercise (74% VO_2 max) was probably high enough to elicit changes in RMR, this variable was not measured until 72 hours after the last exericise bout. Research has shown that it is possible for detraining to occur during this period of inactivity [27]. This may explain why resting metabolism did not change between the untrained and trained states.

Despite the existence of conflicting results, most researchers have made careful attempts to control for all possible sources of error. As a consequence, the majority of studies seems to indicate that purposeful exercise does, indeed, have a carry-over effect on RMR. It appears that this activity causes an increase in RMR (kcal· kg LBM^{-1}· hr^{-1}), but the exact cause of this elevation remains to be established. The tissues and structures responsible for this phenomenon are unknown. Furthermore, the metabolic processes requiring this extra energy expenditure, as well as the regulating signals, also remain a mystery. Some have suggested that an increase in lean body mass may be responsible. Others believe that it is due to changes occurring within the muscle fibers themselves. Regardless, exercise training at an intensity of 60% VO_2 max or higher may be sufficient to cause elevations in resting metabolism [27].

Aerobic exercise stimulates an increase in the activity of mitochondrial proteins contained within the lean tissue [21,37]. In addition it also results in the increased size and number of mitochondria and capillary density. As a result it appears that RMR is not directly influenced by the quantity of lean body mass. Instead, this phenomenon is more likely to arise from an increase in the metabolic activity of each muscle fiber.

In addition to mitochondrial influences on resting metabolism, Lemon reviewed the role that protein metabolism has in exercise [23]. He concluded that the breakdown of this nutrient was magnified by physical activity. Similar results have been found based on the oxidation of leucine [36], but others still do not support this finding. The intensity and duration of exercise have also been shown to yield proportional increases in the plasma and urine concentrations of urea, which remained elevated for some time during recovery [16]. An elevation in the urinary excretion of urea has also been shown by Mole´ and Johnson [25], to exist during the first two days of recovery from intense exercise. If protein balance is to be maintained, its synthesis would need to be increased

accordingly. This activity is very costly. Thus, it could add yet another explanation for the elevated RMRs (kcal· kg LBM^{-1}· hr^{-1}) that are observed in trained athletes.

During the first eight weeks of a resistance training program, a gain in strength will occur largely from an increase in neural activity. After this initial time period, however, hypertrophy of the muscle itself becomes responsible for any such improvements [15]. Resistance training stimulates the production of actin and myosin. As a result, these contractile proteins cause an expansion or hypertrophy of the existing muscle fibers by increasing the cross-sectional area. With proper training weightlifters can achieve significant gains in lean body mass.

There is no question that endurance training elicits metabolic changes within the active muscle fibers. Trained athletes, for instance, experience an increase in the size and number of mitochondria contained within each of the different fiber types, as well as the capillary density. The activity of various mitochondrial enzymes is also altered [37]. Furthermore, protein metabolism has been shown to increase within the active tissues, as well [16,23,36]. Together, these changes are likely to be causes of the higher resting metabolism (kcal· kg LBM^{-1}· hr^{-1}) that is often observed in trained athletes.

How do weightlifters compare to these endurance athletes? Unfortunately, at the present time, there is not much literature on this topic. The only study that has been done was conducted by Broeder et al. [6]. They investigated the effects of high intensity resistance training on RMR. In this longitudinal study, forty-seven males between the ages of 18 and 35 were randomly assigned to one of three groups: control, endurance trained or resistance trained. The individuals of the latter two groups were subjected to a 12-week training program.

The resistance training program involved the use of both free weights and Nautilus equipment (Nautilus Sports/Medical Indus, Inc., Independence, VA). The following exercises were done throughout the 12-week period: bench press, parallel dip, behind-neck press, upright rows, tricep pressdown, leg press, leg extension, leg curl, lat pulldown, barbell curl, and abdominal crunch. Upper body exercises were done on Mondays and Thursdays, while lower body exercise were done on Tuesdays and Fridays. Abdominal exercises were done at the conclusion of each workout. During the first two weeks of the program, each subject performed 3 sets of 10-12 repetitions on each exercise. The resistance was such that the subject became fatigued upon performing the desired number of repetitions. During the last 10 weeks of the resistance program, some changes were made. The weight was now adjusted so that fatigue occurred after 10-12 repetitions in the first set, 8-10 repetitions in the second set and 6-8 repetitions in the third set. Abdominal exercises were still done after each workout session.

The endurance trained subjects participated in a walking or jogging program 4 times $\cdot$ week^{-1}. Each person was exercising at 70% VO_2 max for at least 40 minutes by the end of the 4th week. By the end of the week 8, however, each subject was exercising at 70-85% VO_2 max for 50 minutes. Fartlek-type intervals were added to the program during the final 4 weeks. These intervals had to be performed at an intensity of 90% VO_2 max for 2-5 minutes.

As a result of these training programs, the endurance trained athletes were able to maintain their fat-free weight, while simultaneously reducing their total fat weight. The resistance trained athletes, on the other hand, experienced an increase in fat-free weight, while their total fat weight was reduced.

Each subject's resting metabolism was measured before and after the training program. When comparing the pre-trained and post-trained values, Broeder found that

there was no significant difference. However, he also discovered that many of the subjects were in a state of negative energy balance. For example, during this period of increased caloric expenditure, there was also a small decline in caloric intake. Although endurance training and resistance training did not cause any elevations in RMR, it appears that they may be beneficial in helping to prevent the decrease in RMR that is often observed during periods of negative energy balance [6]. More research needs to be done in the area of resistance training.

When dealing with weight loss, most experts stress the importance of aerobic activity. After all, research has indicated that it can yield elevations in resting metabolism. What about anaerobic exercise? Can it be just as effective in a weight loss regimen? Weightlifters may provide the necessary evidence. The muscle adaptations caused by their mode of training are very different from those of an endurance athlete's. There is an increase in the activity of the contractile proteins rather than the mitochondrial proteins, and there is often a significant difference in the quantity of lean body tissue that each possesses. Research has shown that the more lean tissue an individual has, the greater his/her RMR ($kcal \cdot min^{-1}$) [5,6,26,27,33,34,47]. Does this phenomenon persist, however, when expressed per kilogram of lean body mass? If it does, then it suggests that resistance training may be just as effective at increasing RMR, as aerobic activity is.

CHAPTER III

METHODS

DESCRIPTION OF SUBJECTS

Twenty-four healthy, non-smoking males between the ages of 18 and 30 were recruited from the surrounding community to participate in this study. Eight of these individuals were used to form the control group. It consisted of sedentary people (all white) who had not participated in any form of regular physical activity for at least the past six months. The sixteen subjects that remained were used to help establish the experimental groups. One of these groups consisted of eight distance runners (all white) who ran 30-60 miles per week and engaged in no form of resistance training. The other was composed of eight weightlifters (6 white/2 black) who lifted 4-5 times per week and engaged in no type of aerobic activity.

In addition to certain requirements such as age, gender and training protocol, each subject also had other characteristics that made him eligible for participation in this study. For instance, each reported that he had been weight stable for at least the past six months, and had not engaged in any form of dieting. Furthermore, at the time of this study, none of the individuals were taking prescription drugs or steroids, and none were known to suffer from any type of metabolic disorder (i.e. thyroid problem).

Before beginning any of the testing procedures, each subject was informed of the following: the nature of the experiment; the specific testing procedures to be used throughout the experiment; the 36-hour period of inactivity and 12-hour fast that were required prior to testing; and the length of commitment. Once this had been accomplished, the participants were asked to sign an informed consent form. An example of this form can be seen in appendix A.

RESTING METABOLIC RATE

All subjects were instructed to report to the Resting Metabolic Rate Lab by 6:00 a.m. on the morning of the test. In addition, they were told to arrive after at least 12 hours of fasting and 36 hours of inactivity. For the purposes of this study, fasting was defined as abstinence from all food and beverage, except water. The thermic effect of food and the thermic effect of activity are both known to elevate the body's metabolism [27,34]. Therefore, it was very important to adhere to these guidelines if the most accurate measure of RMR was to be obtained.

Initially, each subject's height and weight were measured on a standard physician type scale. RMR was then determined by open-circuit spirometry. After being positioned comfortably onto a reclining, cushioned lounge chair, each subject was fitted with a mouthpiece and noseclip. A 30-minute habituation period was then allowed, so that each individual could become accustomed to breathing with the apparatus in place. At the conclusion of this period, the exhaled gases of each subject were collected into a 200-liter Douglas bag for a maximum of 20 minutes. Subsequently, the bag was analyzed for O_2 content, CO_2 content and volume. Resting metabolic rate was measured on two separate occassions, in order to show reproducibility in the data. Appendix B outlines the specific steps to be followed when measuring this variable.

BODY COMPOSITION

Body composition refers to the specific quantities of lean tissue and adipose tissue that make up the human body. In this particular study, it was determined via hydrodensiotometry (underwater weighing). The underwater weighing procedure is actually used to estimate body density, which is then used to calculate body fat percentages.

Siri and Brozek have developed some useful equations for calculating body fat percentages. The equations are based on density values of 0.900 g · cc^{-1} for fat tissue and 1.100 g · cc^{-1} for lean tissue. They can be written as follows:

$$\text{Siri [42]: Percent fat} = \frac{495}{\text{density}} - 450 \qquad (2.0)$$

$$\text{Brozek [7]: Percent fat} = \frac{4.570}{\text{density}} - 4.142 \cdot 100 \qquad (2.1)$$

Although equations 2.0 and 2.1 can be used with a great deal of accuracy, they are not reliable for all populations. For instance, the lean tissue of blacks is not 1.100 g · cc^{-1}. If accurate percentages of body fat are to be determined on these individuals, equations 2.0 and 2.1 must be modified. Schutte made the necessary modification in Siri's original equation [39]. For example:

$$\text{Blacks: Percent fat} = \frac{437}{\text{density}} - 393 \qquad (2.2)$$

Since two of the twenty-four subjects participating in this study were black, equation 2.2 was of particular interest. As a result, it was used to calculate the body fat percentages of those two individuals. The original Siri equation (2.0), on the other hand, was used for all of the remaining subjects.

Clearly, equations 2.0-2.2 are dependent upon accurate measures of body density. This variable can be easily calculated, however, if both the mass of the body and the volume of the body are known. Body mass can be measured on a standard, physician type scale. Body volume can be determined via hydrodensiotometry.

In determining an individual's underwater weight, residual lung volume must be accounted for. It represents the amount of air trapped in the lungs after complete expiration [35]. If this volume of air is disregarded, the individual's underwater weight will be underestimated, and as a result, his body fat percentage will be overestimated. Thus, it is extremely important that residual volume is measured as carefully as

possible. When using the underwater weighing technique, accurate estimations of body fat are dependent on it.

Closed-circuit spirometry was used to determine residual volume. This particular technique involved the dilution of a known concentration of O_2 (100%) with the volume of gas remaining in the lungs after maximum expiration. Closed-circuit spirometry was originally developed by Lundgaard and Van Slyke in 1918 [24], but it was later modified by Wilmore in 1969 [49]. Body fat percentages were determined on two separate occassions, in order to show reproducibility in the data. The specific steps to be followed when measuring residual volume and obtaining underwater weight are outlined in Appendixes C and D, respectively.

STATISTICAL PROCEDURES

All statistical tests were performed using the Statistics with Finesse program designed by James Bolding (P.O. Box 339, Fayetteville, AR 72702). Pearson correlation coefficients were derived for the resting metabolic rate ($L \cdot min^{-1}$), residual volume and body composition measurements obtained during Trial 1 and Trial 2. A one-way ANOVA was used to determine if any differences existed between the resting metabolism ($kcal \cdot kg\ LBM^{-1} \cdot hr^{-1}$) of sedentary individuals, runners and resistance trained individuals.

CHAPTER IV

RESULTS

Eight sedentary individuals, eight runners and eight weightlifters completed this study. The purpose of their participation was to determine whether or not different types of training caused significant differences in RMR. More specifically, did resistance training appear to cause adaptations within the muscle such that resting metabolism (kcal· kg LBM^{-1}· hr^{-1}) was higher than it was in runners? The physical characteristics of all twenty-four subjects are given in Table 1.

Before the resting metabolism (kcal· kg LBM^{-1}· hr^{-1}) of each group could be compared, three different variables had to be measured. They included RMR (L · min^{-1}), residual volume and body composition. Each of these measurements was made on two separate occasions. This was done in order to show the reliability of the testing procedures.

Resting metabolism (L · min^{-1}) was determined after a 12-hour fast and 36-hours of inactivity. A Pearson correlation coefficient was used to measure the strength of the relationship between Trial 1 and Trial 2. The values obtained on each subject are displayed in Figure 1. Based on this illustration, there appears to be a high correlation between the two measurements ($r = 0.88$, $p \leq 0.05$).

Residual volume was measured immediately after RMR. A Pearson correlation coefficient was used to measure the strength of the relationship between Trial 1 and Trial 2. The values obtained on each subject are displayed in Figure 2. Based on this illustration, there appears to be a high correlation between the two measurements ($r = 0.99$, $p \leq 0.05$).

Body composition was the last variable to be measured. A Pearson correlation coefficient was used to determine the strength of the relationship between Trial 1 and Trial 2. The values obtained on each subject are displayed in Figure 3. Based on this illustration, there appears to be a high correlation between the two measurements ($r = 0.99$, $p \leq 0.05$).

Table 1 Physical Characteristics of Subjects Classified by Mode of Training*

Characteristics	Sedentary (n = 8)	Runners (n = 8)	Weightlifters (n = 8)
Age (years)	23.63 ± 3.07	23.63 ± 3.42	21.63 ± 1.77
Height (m)	1.75 ± 0.06	1.78 ± 0.05	1.79 ± 0.06
Weight (kg)	87.21 ± 10.94	67.53 ± 6.92¥	91.07 ± 15.61
BMI (kg/m^2)	28.38 ± 2.84	21.28 ± 1.39¥	27.28 ± 2.92
Body fat (%)§	25.85 ± 5.37	9.74 ± 2.24	16.78 ± 4.67
LBM (kg)	64.31 ± 5.86	60.97 ± 6.62	75.41 ± 10.76¥

* Values are mean ± SD.
¥ Significantly different from sedentary, $^{*}p \leq 0.05$.
§ All groups are significantly different from each other, $p \leq 0.05$.

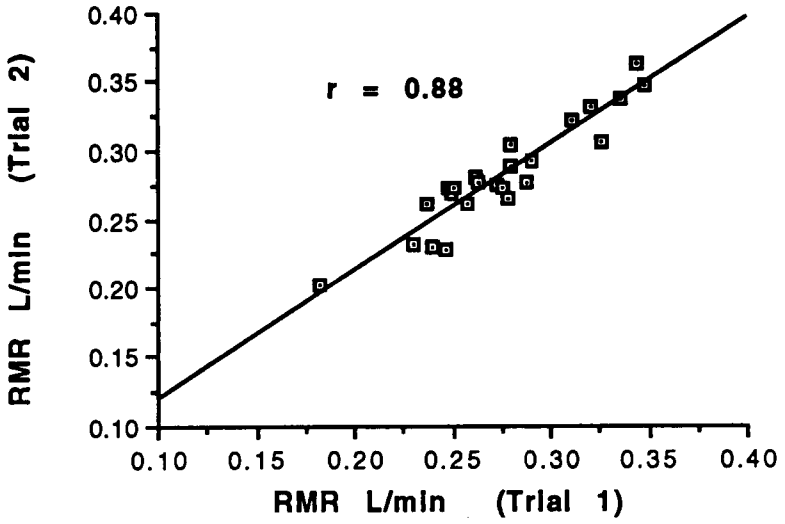

Figure 1 Correlation Between Resting Metabolic Rates (Trial 1 and Trial 2)

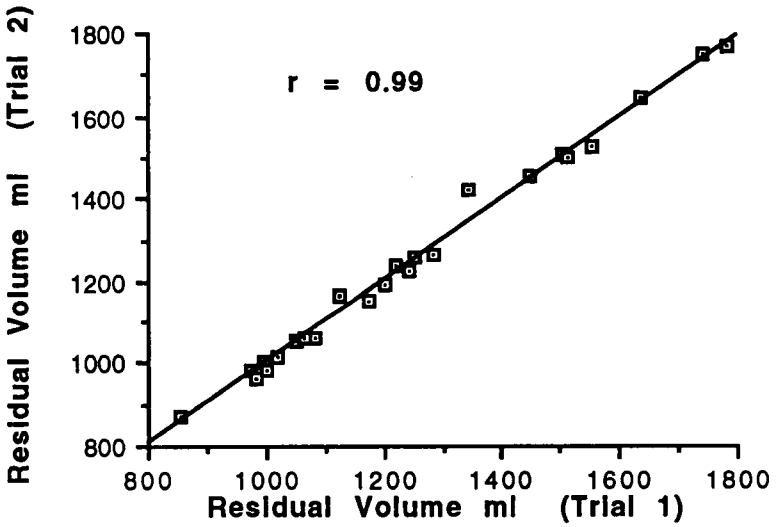

Figure 2 Correlation Between Residual Volumes (Trial 1 and Trial 2)

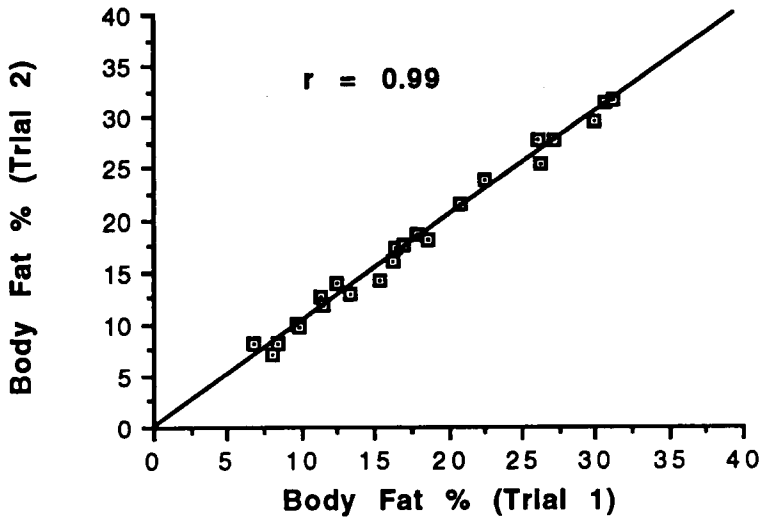

Figure 3 Correlation Between Body Fat Percentages (Trial 1 and Trial 2)

Clearly, Figures 1-3 illustrate good reliability in the testing procedures used for this particular study. As a result, the values obtained when expressing RMR in kcal· kg LBM^{-1}· hr^{-1} should be more credible.

With any statistical analysis, there are certain assumptions that dictate the validity and reliability of the results. For example, when performing ANOVA and/or ANCOVA, it is assumed that the results are based on data that are normally distributed.

In the present study, the Shapiro-Wilk test for normality indicated that the data were not normally distributed. This was due to an outlier that existed in the sedentary control group. When this individual was disregarded, however, the data were normally distributed. In addition, ANCOVA also assumes that the slopes (RMR kcal· hr^{-1} + LBM) of each group are equal. Based on regression equations derived in the present study, these slopes appeared to be unequal. This was the case, even when the outlier was

ignored.

One-way ANOVA was used to make comparisons between the resting metabolism of sedentary, endurance trained and resistance trained individuals. Of particular interest was the relationship between RMR (kcal· hr^{-1}) and lean body mass. The mean resting metabolic rate values are given in Table 2. According to the ANOVA ($F = 5.17$, $p \leq 0.05$), the runners and resistance trained individuals appeared to have significantly lower RMRs (kcal· kg LBM^{-1}· hr^{-1}) than the control group (see Table 3). ANCOVA was not used since the assumptions were not supported by the data.

Table 2 Mean Resting Metabolic Rates*

Variable	Sedentary (n = 8)	Runners (n = 8)	Weightlifters (n = 8)
R	0.839 ± 0.04	0.851 ± 0.03	0.829 ± 0.05
RMR (L· min^{-1})	0.292 ± 0.03	0.244 ± 0.03§	0.299 ± 0.04
RMR (kcal· kg BW^{-1}· hr^{-1})	0.975 ± 0.11	1.03 ± 0.10	0.957 ± 0.08
RMR (kcal· kg LBM^{-1}· hr^{-1})	1.32 ± 0.15	1.17 ± 0.11§	1.15 ± 0.07§

* Values are mean ± SD.
§ Significantly different from sedentary, $^{*}p \leq 0.05$.

Table 3 Analysis of Variance Table for Resting Metabolic Rates (kcal· kg LBM^{-1}· hr^{-1})

Source	Sum of Sqr.	DF	Var. Est.	F-Ratio	Prob. F
Among	0.13912	2	0.06956	5.1662	0.0150
Within	0.28276	21	0.01346		
Total	0.42188	23			

CHAPTER V

DISCUSSION AND CONCLUSIONS

The primary purpose of this study was to determine whether or not resistance training caused adaptations within the muscle such that RMR (kcal·kg LBM^{-1}·hr^{-1}) was higher than it was in runners. Researchers have been interested in resting metabolism for many years. This metabolic variable accounts for 60-75% of the total daily energy expenditure. Thus, it has many practical implications as far as weight loss is concerned.

In the present investigation, there was no significant difference between the RMRs (kcal· kg LBM^{-1}· hr^{-1}) of resistance trained individuals and runners. The value obtained for each group was 1.15 ± 0.07 and 1.17 ± 0.11, respectively. These results were similar to those of Broeder et al. [6]. These investigators conducted a longitudinal study on resistance trained athletes and runners. Although they found that neither mode of training caused changes in RMR (kcal· kg LBM^{-1}· hr^{-1}), the researchers also indicated that there was no significant difference between the resistance trained individuals and runners. The resting metabolic rate of each group was 1.21 ± 0.03 and 1.19 ± 0.03 kcal· kg LBM^{-1}· hr^{-1}, respectively.

At the present time, no other studies have examined the relationship between resistance training and RMR (kcal· kg LBM^{-1}· hr^{-1}). Many, however, have dealt with the relationship between this metabolic variable (kcal· kg LBM^{-1}· hr^{-1}) and endurance training. In each instance, the values obtained for resting metabolism were similar to those of the present investigation. Poelhman, for example, conducted two different studies in which the highly trained subjects had RMRs of 1.20 ± 0.04 and 1.18 ± 0.04 kcal· kg LBM^{-1}· hr^{-1}, respectively [32,33].

While the results obtained on the resistance trained individuals and runners of the present study were consistent with the data of other literature, the same was not

true for the sedentary subjects. This group of individuals, when compared to the control groups of other studies, was shown to have the highest RMR (kcal· kg LBM^{-1}· hr^{-1}). The average value for this group was 1.32 ± 0.15 kcal· kg LBM^{-1}· hr^{-1}. Other investigations, however, have indicated resting metabolic rates of 1.22 ± 0.02, 1.09 ± 0.03 and 1.05 ± 0.03 kcal· kg LBM^{-1}· hr^{-1} [6,32,33].

Why did the control group in this study have such a high RMR (kcal· kg LBM^{-1}· hr^{-1})? One possibility is "overfeeding", the consumption of an abnormally large amount of food in the period preceding the measurement. It is well known that the body's metabolism is increased upon the ingestion of a meal. This is more commonly known as the thermic effect of food (TEF). TEF accounts for approximately 10% of the total daily energy expenditure, and it is possible for it to last up to 8 hours after a meal has been consumed. The exact amount of time depends upon the nutritional state of the individual and the quantity of nutrients in the meal [25]. Under most circumstances, however, 8 hours is a sufficient amount of time for the food to be fully metabolized.

In the present study each subject was asked to fast for 12 hours before having his RMR measured. TEF will not ordinarily influence resting metabolism 12 hours after a meal has been consumed. Several studies, however, have shown that TEF can be elevated for longer periods of time if the individual has overeaten within the past 24 hours. Dauncey, for example, conducted a study in which the subjects were required to overeat for a period of 24 hours [25]. While these individuals were accustomed to a daily intake of 8400 kJ, they now had to consume 13830 kJ · day^{-1}. As a result, RMR (kcal· kg LBM^{-1}· hr^{-1}) was still elevated by 12%, 14 hours after the last meal had been consumed [25]. Overfeeding enhanced the metabolic activity of the tissue.

The theory on overfeeding is very appealing. It seems to offer a reasonable explanation for why the resting metabolic rate (kcal· kg LBM^{-1}· hr^{-1}) of this study's control group was higher than that of control groups from other studies, as well as the

experimental groups of this study. Unfortunately, this cannot be accepted without question. There is no proof that the untrained individuals were overfed. A 3-day dietary recall was not requested. In addition, the R data of each group were virtually identical. The values for the sedentary, endurance trained and resistance trained groups were 0.839 ± 0.04, 0.851 ± 0.03 and 0.829 ± 0.05, respectively. Since the same fuel was being utilized at rest, the sedentary subjects had apparently adhered to the 12-hour fast. Unfortunately, however, their eating patterns before the fast were unknown. As a result, in anticipation of the 12-hour fast, it is possible that the sedentary individuals really did consume more food than they normally would have. If this were true, overfeeding could help explain the higher RMR (kcal· kg LBM^{-1}· hr^{-1}) found in this group of individuals.

In previous studies, sedentary people have been shown to have lower resting metabolic rates (kcal· kg LBM^{-1}· hr^{-1}) than trained athletes. The present investigation, however, indicated that the former individuals had the highest RMRs (kcal· kg LBM^{-1}· hr^{-1}). In order to explain this phenomenon, the elevations were attributed to overfeeding. Since 3-day dietary recalls were not obtained on any of the subjects and the R values of each group were virtually identical, however, this theory on overfeeding was just speculation. In the future, pertinent dietary information should be requested from all participants.

In conclusion, it is well known that resistance training and running yield distinctly different muscle adaptations. Based on the results of the present study, however, these different adaptations do not appear to yield significant differences between the RMRs (kcal· kg LBM^{-1}· hr^{-1}) of individuals participating in these activities. Apparently, the two types of training have similar effects on resting metabolism (kcal· kg LBM^{-1}· hr^{-1}).

REFERENCES

REFERENCES

1. Acheson, K.J., *et al.*, Thermic effect of glucose in man. *J. Clin. Invest.*, 1984. **74**: p. 1572 -1580.

2. Atwater, O. and F.G. Benedict, Experiments on the metabolism of matter and energy in the human body. *USDA Official Experimental Station Bulletin*, 1903. **136**: p. 1-357.

3. Ballor, D.L., *et al.*, Exercise training attenuates diet-induced reduction in metabolic rate. *J. Appl. Physiol.*, 1990. **68**(6): p. 2612-2617.

4. Brehm, B.A., Elevation of metabolic rate following exercise implications for weight loss. *Sports Medicine*, 1988. **6**: p. 72-78.

5. Broeder, C.E., *et al.*, The effects of aerobic fitness on resting metabolic rate. *Am. J. Clin. Nutr.*, 1992. **55**: p. 795-801.

6. Broeder, C.E., *et al.*, The effects of either high-intensity resistance or endurance training on resting metabolic rate. *Am. J. Clin. Nutr.*, 1992. **55**: p. 795-801.

7. Brozek, J., *et al.*, Densitometric analysis of body composition: Revision of some quantitative assumptions. *Annals of the New York Academy of Science*, 1963. **110**: p. 113-140.

8. Christie, R.V., The lung volume and its subdivisions I: Methods of measurement. *J. Clin. Invest.*, 1932. **11**: p. 1099-1118.

9. Danforth, E., Dietary induced thermogenesis: control of energy expenditure. *Life Science*, 1981. **28**: p. 1821-1827.

10. Dauncey, M.J., Metabolic effects of altering the 24 hour energy intake in man, using direct and indirect calorimetry. *British Journal of Nutrition*, 1980. **43**: p. 257-269.

11. Fanelli, M. and R. Kuczmarski, Ultrasound as an approach to assessing body composition. *Am. J. Clin. Nutr.*, 1984. **39**: p. 703-709.

12. Fanelli, M., R. Kuczmarski and M. Hirsch, Estimation of body fat from ultrasound measures of subcutaneous fat and circumferences in women. *Int. J. of Obes.*, 1988. **12**: p. 125-132.

13. Flatt, J.P., The biochemistry of energy expenditure., in *Recent Advances in Obesity Research: II*, G. Bray, Editor. 1978, Newman: London.

14. Gleeson, M., J.F., Brown and J.J. Waring, The effects of physical exercise on metabolic rate and dietary-induced thermogenesis. *Br. J. Nutr.*, 1982. **47**: p. 173-180.

15. Hakkinen, K., Factors influencing trainability of muscular strength during short term and prolonged training. *National Strength and Conditioning Association Journal*, 1985. **7**(2): p. 32-37.

16. Haralambie, G. and A. Berg, Serum urea and amino nitrogen changes with exercise duration. *Eur. J. Appl. Physiol. and Occup. Physiol*, 1976. **36**: p. 39-48.

17. Heshka, M.Y., *et al.*, Weight loss and change in resting metabolic rate. *Am. Soc. Clin. Nutr.*, 1990. **52**: p. 981-986.

18. Hill, J.O., J.R. Davis and A.R. Tagliaferro, Effects of diet and exercise training on thermogenesis in adult female rats. *Physiol. and Behav.*, 1983. **31**: p. 133-135.

19. Jackson, A. and Pollock, M.L., Practical assessment of body composition. *Phys. and Sports Med.*, 1985. **13**(5): p. 76-90.

20. Katch, F. and V. Katch, Measurement and prediction errors in body composition assessment and the search for the perfect prediction equation. *Res. Quarterly*, 1984. **51**(1): p. 249-260.

21. Kent-Braun, J.A., K.K. McCully and B. Chance, Metabolic effects of training in humans: a P-MRS study. *J. Appl. Physiol.*, 1990. **69**(3): 1165-1170.

22. Lawson, S., *et al.*, Effect of a 10-week aerobic exercise programme on metabolic rate, body composition and fitness in lean sedentary females. *Br. J. Clin. Pract.*, 1987. **41**: p. 684-688.

23. Lemon, P.W., Protein and exercise: update 1987. *Med. Sci. Sports Exerc.*, 1987. **19**: p. S179-S190.

24. Lundsgaard, C. and D.D. VanSlyke, Studies of lung volume I: Relation between thorax size and lung volume in normal adults. *J. Exptl. Med.*, 1918. **27**: p. 65-85.

25. Mole, P.A. and R. Johnson, Disclosure by dietary modification of an exercise-induced protein catabolism in man. *J. Appl. Physiol.*, 1971. **31**: p. 185-190.

26. Mole, P.A., *et al.*, Exercise reverses depressed metabolic rate produced by severe caloric restriction. *Med. Sci. Sports Exerc.*, 1989. **21**(1): p. 29-33.

27. Mole, P.A., Impact of energy intake and exercise on resting metabolic rate. *Sports Medicine*, 1990. **10**(2): p. 72-87.

28. Nash, H.L., Body fat measurement: weighing the pros and cons of electrical impedance. *Phys. and Sports Med.*, 1985. **13**(11): p. 124-128.

29. Phinney, S.D., *et al.*, Effects of aerobic exercise on energy expenditure and nitrogen balance during very low calorie dieting. *Metabolism*, 1988. **37**: p. 758-765.

30. Poehlman, E.T., *et al.*, Influence of caffeine on the resting metabolic rate of exercise-trained and inactive subjects. *Med. Sci. Sports Exerc.*, 1985. **17**: p. 689-694.

31. Poehlman, E.T., Heredity and changes in hormones and metabolic rates with short-term training. *Am. J. Physiol.*, 1986. **250**: p. E711-E717.

32. Poehlman, E.T., C.L. Melby and S.F. Badylak, Resting metabolic rate and postprandial thermogenesis in highly trained and untrained males. *Am. J. Clin. Nutr.*, 1988. **47**: p. 793-798.

33. Poehlman, E.T., *et al.*, Aerobic fitness and resting energy expenditure in young adult males. *Metabolism*, 1989. **38**(1): p. 85-90.

34. Poehlman, E.T., A review: exercise and its influence on resting energy metabolism in man. *Med. Sci. Sports Exerc.*, 1989. **21**(5): p. 515-525.

35. Powers, S.K. and E.T. Howley, Exercise Physiology. 1 ed. Vol. 1. 1990, Dubuque, IA: Wm.C. Brown.

36. Rennie, M. and R. Edwards, Effect of exercise on protein turnover in man. *Clin. Science*, 1981. **61**: p. 627-639.

37. Saltin, B. and P.D. Gollnick, Skeletal muscle adaptability: Significance for metabolism and performance., in *Handbook of physiology*, Peachey, L.D., R.H. Adrian and S.R. Geiger, Editors. 1983, Williams and Wilkins: Baltimore. **Section 10, chapter 19**.

38. Schneider, E. and A. Foster, The influence of physical training on the basal metabolic rate in man. *Am. J. Physiol.*, 1931. **98**: p. 595-601.

39. Schutte, S.A. and H.M. Linkswiler, Calcium., in *Nutrition Reviews' present knowledge in nutrition* . 1984, The Nutrition Foundation: Washington, D.C. p. 400-412.

40. Segal, K., et al., Effect of physical training on resting energy and caarbohydrate metabolism in lean and obese men. *Med. Sci. Sports Exerc.*, 1989. **21**(2): S44.

41. Sinning, W.E., Anthropometric estimation of body density, fat and lean body weight in women gymnasts. *Med. Sci. Sports*, 1978. **10**(4): p. 243-249.

42. Siri, W.E., Gross composition of the body., in *Advances in biological and medical physics*, Lawrence J.H. and C.A. Tobias, Editors. 1956, Academic Press: New York. **4**: p. 239-280.

43. Terjung, R.L. and C.M. Tipton, Exercise training and resting oxygen consumption. *Int. Z. angew. Physiol.*, 1970. **28**: p. 269-272.

44. Tremblay, A., *et al.*, Contribution of postexercise increment in glucose storage to variations in glucose induced thermogenesis in endurance athletes. *Can. J. Physiol. Pharmacol.*, 1985. **63**: p. 1165-1169.

45. Tremblay, A., *et al.*, The effect of exercise-training on resting metabolic rate in lean and moderately obese individuals. *Int. J. Obes.*, 1986. **10**: p. 511-517.

46. Tremblay, A., *et al.*, Long term exercise training with constant energy intake 2: effect on glucose metabolism and resting energy expenditure. *Int. J. Obes.*, 1988. **14**: p. 75-84.

47. Warwick, M. and J. Garrow, The effect of addition of exercise to a regime of dietary restriction on weight loss, nitrogen balance, resting metabolic rate and spontaneous physical activity in 3 obese women in metabolic ward. *Int. J. Obes.*, 1981. **2**: p. 25-32.

48. Weinsier, R.L., Y. Schutz and D. Bracco, Reexamination of the relationship of resting metabolic rate to fat-free mass and to the metabolically active components of fat-free mass in humans. *Am. J. Clin. Nutr.*, 1992. **55**: p. 790-794.

49. Wilmore, J., The use of actual, predicted and constant residual volumes in the assessment of body composition by underwater weighing. *Med. Sci. Sports*, 1969. **1**(2): p. 87-90.

APPENDIXES

APPENDIX A

INFORMED CONSENT FORM

DESCRIPTION OF PROJECT:

The purpose of this project is to study the relationship between lean body mass and resting metabolic rate (amount of energy you use at rest). These two variables will be measured in the morning, after 12 hours of fasting and 36 hours of inactivity. This protocol should ensure that each individual is truly in a resting state.

The entire RMR procedure, which lasts approximately 50 minutes, will take place while you are lying comfortably in a lounge chair. During this time, you will have a respiratory valve in your mouth. Further, a nose clip will be used to block nasal breathing. The first 30 minutes will be used to get you accustomed to breathing through the aforementioned apparatus. You will simply lie in the chair and breathe as normally as possible. A single 20-minute collection of your exhaled air will be made during the latter part of this time period.

Your body fatness will be measured with the underwater weighing technique. After your height, weight and residual volume (air remaining in your lungs after a complete exhalation) have been determined, you will enter the tank and sit on the weighing sled. While in this position, you will exhale completely while you bend forward to pull your head underwater. We ask that you hold your breath for 5-10 seconds before "coming up" to breathe again. You will repeat this step 5-6 times. This procedure will be carried out on the same day of 2 consecutive weeks. More specifically, it will be obtained on the same days as RMR.

You will receive a careful measurement of your resting metabolic rate and your body fat percentage. These values are helpful in planning weight control or exercise programs.

I have read the statement above and understand my role in the experiment, as well as the risks involved. I have had the procedures explained and demonstrated to me. In addition, I am aware that:

1. My name and the results will remain confidential;
2. I am entitled to have any further inquiries answered regarding the procedures;
3. I may withdraw my consent and discontinue participation at any time without prejudice toward me;
4. In the event of physical injury because of my participation in this study, financial compensation is not available and medical treatment will not be provided free of charge.

Signature: _____ **Date:** _____

Witness: _____ **Date:** _____

Dr. Edward T. Howley
Rm. 336, HPER Bldg (974-5111)

Robin D. Eldridge
Rm. 305, HPER Bldg (974-5111)

APPENDIX B

RESTING METABOLIC RATE PROCEDURES

1. Turn on the flow meter of the S3-A O_2 analyzer 30 minutes before analyzing gas samples.
2. Push the operate button on the LB2-CO_2 analyzer 30 minutes before analyzing gas sample.
3. Change the AQUASORB.
4. Record the barometric pressure, temperature, relative humdity and water vapor pressure of the laboratory. These values will be used to calculate a standard temperature and pressure dry (STPD) correction factor.
5. Measure Resting Metabolic Rate (RMR)
   a. Position each subject comfortably onto a reclining, cushioned lounge chair.
   b. Fit each subject with a 2-way Daniel's valve and mouthpiece. Hoses should be attached to both sides of this respiratory valve. The subject will inspire through one and expire through the other.
   c. Fit each subject with a noseclip. This will help to ensure that breathing occurs only at the mouth.
   d. Allow a 30-minute habituation period so each subject can become accustomed to breathing with all of the apparatus in place.
   e. Collect each subject's expirate into a 200-Liter Douglas bag for 20 minutes. This can be done by turning the 3-way valve that connects the expiratory hose to the bag. It may be necessary to terminate collection prior to 20 minutes, if the bag fills too quickly.
   f. At the conclusion of the collection period, turn the 3-way valve so that no more air is allowed to flow into the Douglas bag.

g. Remove the Douglas bag from the expiratory hose and place the rubber stopper in it. This will prevent the loss of any exhaled gas.

h. Analyze the bag for O_2 content, CO_2 content and volume.

6. Calibrate the analyzers

a. Check the temperaure of the O_2 sensor located on the S3-A O_2 analyzer. Turn the Range dial to "T.C. 0-10" and observe a temperature reading of 6.790 ± 0.003.

b. Turn the Range dial to "%O_2" and move the switch located just below it from "unknown" to "reference adjust". Use the potentiometer to adjust the value on the meter face to 20.93% (this represents the percentage of O_2 in the atmosphere). Move the Range dial back to "unknown" once this has been done.

c. Zero the analyers by allowing N_2 to flow from the N_2 tank into the analyzers for 20-30 seconds. Adjust the Beckman LB2-CO_2 analyzer to zero with the "zero control" knob. Simply observe a reading of 00.00 ± 0.02% on the meter face of the S3-A O_2 analyzer.

d. Set the span of both analyzers. Let the high CO_2/low O2 gas flow from its tank into each analyzer for 20-30 seconds. Use the "gain" knob to set the value (5.98%) on the Beckman LB2-CO_2 analyzer. Use the "cell zero" knob the set the value (14.99%) on the S3-A O_2 analyzer. Follow the same procedures for the low CO_2/high O_2 gas. The percentages of CO_2 and O_2 contained within this tank are 2.96-1.96% and 17.97%, respectively.

7. Analyze the Douglas bags

a. Record the values that are shown on the meter face of each analyzer. These numbers represent FIO_2 and $FICO_2$.

b. Shake the bag to ensure that the expired gases are thoroughly mixed before being analyzed.

c. Remove the clip from the rubber tubing and attach the tubing to the AQUASORB.

d. Allow the expirate to flow into the analyzers for one minute. Record the corresponding percentages of O_2 and CO_2 shown on each respective meter face.

e. Remove the bag from the tube of AQUASORB and place the clip back on the rubber tubing. This will prevent air from leaking out of the bag.

f. Turn off the flow meter of the S3-A O_2 analzyer and push the operate button on the Beckman LB2-CO_2 analyzer.

8. Measure the volume of expirate

a. There are many valves located on the 120-Liter Tissot gasometer. Open the one to which the Douglas bag will eventually be attached, but close all others.

b. Push the inverted bell as far down as possible, close the only opened valve and observe the meterstick on the side of the Tissot. Record the initial displacement value.

c. Remove the rubber stopper from the Douglas bag and attach it to the Tissot.

d. Open the valve to which the Douglas bag is connected and squeeze all of the expirate out of it.

e. Close the valve and record the final displacement measurement shown on the meter stick.

f. Linear displacement is the difference between the final and initial measurements obtained off of the meterstick. This variable is used to help calculate the equivalent volume of gas contained within the Douglas bag. To obtain the most accurate measurement, however, the amount of gas that is lost during 1 minute of analysis (0.6 Liters) must be added back in.

g. The procedures outlined in steps (a - f) may need to be repeated more than one time. As air is squeezed into the Tissot, the inverted bell begins to rise. It is

undesirable to break the seal that exists between this bell and the water of the gasometer. To keep this from occuring, then, it is necessary to prevent the bell from rising too high. Thus, any given Douglas bag may need to be squeezed more than one time, before it has been sufficiently depleted of its contents. Remove the Douglas bag from the Tissot and place the rubber stopper in it before repeating the procedures.

APPENDIX C

RESIDUAL VOLUME PROCEDURES

1. Turn the vacuum pump on and switch the N_2 analyzer from "standby" to "operate". Allow at least 30 minutes for warm-up before calibration.
2. With the bell pulled all the way up, check the water level of the Collin's 9-Liter Tissot gasometer. It should be in the middle of the window. If not, use distilled water to ensure that it is.
3. Record the water temperature of the gasometer.
4. Calibrate the N_2 analyzer
   a. Occlude the valve tubing and observe a value of zero on the meter face.
   b. Expose the needle valve to room air.
   c. Turn the needle valve counterclockwise until a maximum value is obtained.
   d. Turn the "adjust" knob until a value of 79.6% is shown on the meter face.
   e. Turn the needle valve clockwise until a reading of 76.6% is obtained.
   f. Turn the "adjust" knob once again, until a value of 79.6% is shown.
   g. To check the calibration, make sure that the analyzer reads 79.6 ± 0.1% as room air is pulled into the bell.
5. Fill the bell with O_2
   a. Open the stopcock that connects the O_2 tank to the spirometer.
   b. Flush the bell 3 or more times with O_2. To accomplish this attach the white tubing, 3-way valve and mouthpiece to the spirometer and allow the bell to fill slightly with the valve closed. Next, open the valve and force the air out of the bell by pushing down on it. This action causes air to flow out past the sensor, allowing the N_2 concentration of the bell to be determined. Continue flushing with O_2 until a value of 0.3% is shown on the meter face of the analyzer.

c. Fill the bell with 6000 ml or 6500 ml of O_2. This initial amount is dependent upon the lung capacity of each subject. Nevertheless, the pen that is located on the chart recorder should move from 7000 ml down to 1000 ml or 500 ml, depending on the initial volume of the bell.

6. Measure residual volume

a. The subject is seated with the knees bent prior to the measurement. This is done in order to similate the subject's body position during the underwater weighing procedure.

b. A mouthpiece, which is connected to a 3-way valve, is placed inside of the subject's mouth. He is also required to wear a noseclip at this time.

c. The 3-way valve should be closed so that the subject is breathing room air.

d. Upon command, the subject takes a deep breath in and exhale until all air has been forced out of the lungs.

e. After maximal expiration has been achieved, the 3-way valve is turned to connect the subject to the bell of O_2.

f. The subject is instructed to take 6-8 rapid, deep breaths. After taking the first breath in, the value shown on the N_2 analyzer should be recorded. It represents the IN_2. Next, the researcher records two values (one during inspiration and one during expiration) as they begin to stabilize within 0.2-0.3% of each other. Their average is used to represent EN_2. Finally, the subject is instructed to exhale maximally until all air is out of the lungs. The 3-way valve is turned back to room air, and the value shown on the N_2 analyzer is recorded. It represents AfN_2.

g. Determine the amount of air that was expired by looking at the markings on the chart paper. For the test to be valid, this value should be within 50 ml of the

initial volume of air that filled the bell.

h. Continue residual volume measurements until two similar IN_2, EN_2 and AFN_2 readings are obtained. This generally occurs after 2-3 trials. The average of each respective variable is then used to calculate the subject's residual volume.

i. Check the calibration of the N_2 analyzer before each trial. The analyzer needs to be recalibrated if it does not show a value of 79.6 ± 0.02% when room air is pulled into the bell.

APPENDIX D

HYDROSTATIC WEIGHING PROCEDURES

1. Make sure that the tank is clean.
2. Close the drain and fill the tank with tap water. Keep the temperature between 31-35 °C.
3. Turn on the digital display unit and the physiograph. In order to allow a sufficient warm-up, do this at least 30-minutes prior to testing.
4. Calibrate the hydrostatic weighing apparatus
   a. With the subject crouched in the back right corner of the tank and nothing on the weighing sled, use the "zero" knob to obtain a value of ± 00.00 kg on the digital display unit.
   b. Place the 4.26 kg weight belt on the sled. Situate it such that its weight is distributed evenly between the back two force transducers. If a value of 4.26 kg is not shown on the digital display unit, use the "gain" knob to make adjustments.
   c. With the belt still in place, use the "zero" knob to tare the display unit from 4.26 kg back down to ± 00.00 kg.
   d. Place the 1.98 kg weight belt in the center of the heavier belt. If a value of 1.98 ± 0.02 kg is not shown on the digital display unit, remove both belts from the sled and recalibrate the equipment. If a value of 1.98 ± 0.02 kg is shown on the digital display unit, remove both belts from the sled, and observe that the display unit shows a value of -4.26kg. If it does not, recalibrate the equipment.
5. Prepare the subject and obtain the measurement
   a. The subject removes all air bubbles from the hair, skin and swimsuit, while crouching in the corner of the tank.
   b. The subject places the 4.26 kg weight belt around his waist.

c. Using the railings for support, the subject eases his backside onto the sled. He should be facing away from the entry area of the tank and seated comfortably, with his knees bent and his feet secured under the foot rest.

d. The subject puts on a noseclip and ensures that no part of his body is touching the sides of the tank.

e. While holding on to the sides of the sled, the subject takes in a deep breath and begins to exhale maximally as he pulls himself underneath of the water.

f. The subject holds his breath for as long as possible, once all of the air has been forced out of the lungs. During this time, his underwater weight is monitored by the physiograph.

g. Repeat the underwater weighing procedure until 2-3 similar values are obtained. This may take as many as 4-7 trials.

h. Recheck calibration at the conclusion of the test. Have the subject remove the 4.26 kg belt from his waist and place it on the sled. The weight of this belt should be evenly distributed between the back two force transducers, and the digital display unit should give a reading of 00.00 ± 0.02 kg.

APPENDIX E

ORIGINAL DATA

Table A-1 Original Data (Sedentary)*

ID#	Age (yrs)	Ht (m)	Wt (kg)	R	RMR ($L \cdot min^{-1}$)	RV (ml)	BF (%)
CB	21	1.82	92.73	0.8726	0.2845	1065	23.17
SD	25	1.71	95.85	0.8076	0.3265	865	31.07
MG	23	1.71	90.97	0.8427	0.2745	1254	29.68
TK	22	1.73	86.32	0.9098	0.3370	1016	26.97
DL	30	1.70	74.46	0.8744	0.3365	992	27.36
RR	24	1.71	75.37	0.8194	0.2375	1072	21.16
TR	20	1.87	104.88	0.8118	0.3160	999	31.40
GT	24	1.77	77.07	0.7756	0.2915	981	16.04

* All data represents the mean of Trial 1 and Trial 2.

Table A-2 Original Data (Runners)*

ID#	Age (yrs)	Ht (m)	Wt (kg)	R	RMR ($L \cdot min^{-1}$)	RV (ml)	BF (%)
SF	28	1.78	73.04	0.8307	0.2485	1235	13.19
SJ	24	1.70	59.59	0.8565	0.1915	1541	11.92
NK	27	1.78	70.88	0.8607	0.2345	1505	8.27
BL	20	1.76	61.97	0.8693	0.2605	1744	7.51
RM	28	1.82	74.06	0.8076	0.2590	1775	8.26
GM	23	1.74	57.20	0.8122	0.2305	1143	9.73
SP	23	1.86	74.46	0.8747	0.2585	1638	7.43
DP	18	1.80	69.06	0.8516	0.2695	1165	11.65

*All data represents the mean of Trial 1 and Trial 2.

Table A-3 Original Data (Resistance Trained)*

ID#	Age (yrs)	Ht (m)	Wt (kg)	R	RMR ($L \cdot min^{-1}$)	RV (ml)	BF (%)
TB	22	1.75	82.12	0.8344	0.2705	1229	14.82
JB	22	1.83	101.98	0.8735	0.3480	1273	17.28
EB	18	1.82	80.99	0.8401	0.2735	1383	9.74
MF	24	1.70	76.95	0.8919	0.2820	972	16.87
KH	21	1.73	79.51	0.7936	0.2910	1051	13.14
EM	22	1.80	107.89	0.7559	0.3160	1198	25.79
OW	23	1.82	81.61	0.7828	0.2610	1452	18.33
PY	21	1.89	117.53	0.8559	0.3535	1505	18.28

* All data represents the mean of Trial 1 and Trial 2.

VITA

Robin Denise Eldridge was born in Petersburg, Virginia on August 1, 1969. She graduated from Hopewell High School in June 1987, and began attending James Madison University in August of the same year. She received her Bachelor of Science degree in Health Science and Athletic Training in May 1991. Upon graduation, she entered graduate school at the University of Tennessee, Knoxville.

She received her Master of Science degree in December 1992. She is currently serving as the athletic trainer for the women's basketball team at James Madison University.