There is a substantial body of evidence which supports the finding that caloric restriction results in a lowering of resting metabolic rate (RMR). Conflicting findings have been reported on the impact aerobic exercise may have in negating or even reversing this decline. It is widely believed that a combination of caloric deficit and exercise will accelerate weight/fat loss and prevent the decline of RMR. Some investigations, however, suggest that the effects of severe caloric restriction in tandem with moderate amounts of aerobic exercise may exacerbate the decline in RMR. The purpose of this study was to compare the resting metabolic rate and excess post-exercise oxygen consumption (EPOC) in active, normal calorie (NC) and low calorie (LC) dieting females.

Fifteen females between the ages of 18 and 25 were selected through their responses to questionnaires on diet and exercise habits, and were assigned to one of two groups based on their average daily caloric intake and exercise volume. Subjects assigned to the NC group ate a minimum of 1700 kcals · day⁻¹. Subjects in the LC group consumed an estimated 1200 kcals · day⁻¹ or less. Both groups of subjects engaged in aerobic exercise a minimum of four hours per week at ≥ 50% VO₂max. Independent t-tests were conducted on all dependent variables. Subjects demonstrated no significant differences in physical characteristics such as body weight, height, or body mass index, although the LC group did show a tendency toward greater percentage body fat (p = .08). No differences were seen in any physiological characteristics such as resting heart rate, blood pressure, or VO₂max. The only significant difference between the two groups was in estimated daily caloric intake (p < .001).

Subjects completed two RMR trials and a 45-minute submaximal treadmill test (65% VO₂max) to elicit EPOC. EPOC was measured for one hour. No significant
difference between groups was noted in RMR (.17 ± .03 vs .17 ± .03 L·min⁻¹, NC and LC, respectively) or EPOC (.24 ± .03 vs .25 ± .07 L·min⁻¹, NC and LC, respectively). At 60-minutes post-exercise, both groups still demonstrated a significant elevation over resting VO₂ (p < .01 and p < .05, NC and LC groups, respectively), with the NC group demonstrating a slightly greater absolute elevation in the last five minutes (.21 ± .02 vs .19 ± .03 L·min⁻¹). Based on the results of this study, it would appear that the level of caloric intake did not affect RMR or the magnitude of EPOC.
Comparison of Resting Metabolic Rate and Excess Post-Exercise Oxygen Consumption in Normal and Low Calorie Dieting Females

by

Carey Ann Hilbert

A THESIS
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Oregon State University

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APPROVED:

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Major Professor, representing Exercise and Sport Science

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Dean of Graduate School

I understand that my thesis will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my thesis to any reader upon request.

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Carey Ann Hilbert, Author
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COMPARISON OF RESTING METABOLIC RATE AND EXCESS POST-EXERCISE OXYGEN CONSUMPTION IN NORMAL AND LOW-CALORIE DIETING FEMALES

CHAPTER 1

INTRODUCTION

The role of exercise in the enhancement of physical, mental, or emotional well-being has received increasingly wide-spread support over the last decade. Lack of adequate physical exertion is now recognized by leading medical agencies as a contributing cause in increasing the risk for chronic diseases such as coronary artery and cardiovascular diseases, diabetes, and obesity (Pollock & Wilmore, 1990). Numerous studies have emphasized the integrative role that both quality and quantity of diet and exercise can have on increasing the health status of the individual. While it is important for this information to continue to be dispersed to those individuals who do not participate in any type of regular physical activity or have poor dietary habits, there is, conversely, a large number of individuals who have taken this message to extreme, often obsessive, levels.

In the past two decades there has been a tremendous increase in the numbers of women participating in intense athletic activities and exercise programs. An increasing socio-cultural emphasis on physical appearance, with thinness endorsed as the ideal, has been postulated as one of the motivating forces behind this rise in women's athletic participation (Davis, 1990; Wardle & Marsland, 1990). In addition, severe dieting behaviors are often combined with vigorous aerobic activity in an attempt to increase the energy deficit and hasten weight loss.

Conflicting findings have been reported on the impact exercise may have on increasing resting metabolic rate (RMR). Several studies with males and females utilizing aerobic exercise have not indicated an increase in resting metabolic rate (RMR) with exercise (Bingham, Goldberg, Coward, Prentice, & Cummings, 1989; Shah, Miller, & Geissler, 1988; Westerterp, Meijer, Schoffelen, & Janssen, 1994). In contrast, Tremblay and colleagues (1986) found that exercise training significantly increased RMR/fat-free mass (FFM) in lean, as well as obese, individuals. The inconsistency of these
investigations raises questions regarding mode, frequency, intensity, and duration of the exercise protocol used. Mole's (1990) review of the current literature suggests a definite intensity-duration threshold which needs to be attained for exercise to produce any lasting effects on RMR. When that threshold is exceeded, or is combined with a very low calorie diet, it is postulated by some that the result is an exacerbation of the decline in RMR seen with calorie deficit alone.

In addition to questions regarding the exercise components, dietary habits must be taken into consideration. There is strong supporting evidence for the findings that caloric restriction results in a lowering of the resting metabolic rate (Jebb, Goldberg, Coward, Murgatroyd, & Prentice, 1991; Mole, 1990; Thompson & Blanton, 1987). It is widely held that a combination of calorie deficit and exercise will accelerate weight/fat loss and prevent the decline of RMR (Ballor, Tommerup, Thomas, Smith, & Keesey, 1990; Lemons, Kreitzman, Coxon, & Howard, 1989; Mole, Stern, Schultz, Bernauer, & Holcomb, 1989; Svendsen, Hussager, & Christiansen, 1994). But if the body attempts to become more energy efficient during severe caloric restriction by lowering metabolic activity and energy expenditure, a question arises as to what ramifications that will have on the body's ability to increase metabolic rate in response to exercise. Phinney, LaGrange, O'Connell, and Danforth (1988) suggest that the effect of large amounts of aerobic exercise in tandem with severe caloric restriction may act to exacerbate the decline in RMR. Brownell, Steen, and Wilmore (1987) hypothesize that as weight falls below a level at which a person would normally regulate, energy efficiency will increase.

Elevation of metabolism following exercise is an issue of controversy. While there is an immediate elevation post-exercise (fast component), which is seen to decline rapidly within the first few minutes, disagreement exists primarily around what is known as the "slow component" (Mole, 1990). Numerous investigations show no indication of a prolonged elevation of post-exercise oxygen consumption (EPOC) (Brehm & Gutin, 1986; Freedman-Akabas, Colt, Kissileff, & Pi-Snyder, 1985; Kaminsky, Kanter, Lesmes, & Leham-Saeger, 1987; Knutgen, 1970; Pacy, Barton, Webster, & Garrow, 1985; Sedlock, Fissinger, & Melby, 1989), while at least as many other investigations report significant elevation of post-exercise oxygen consumption (Bahr, Inges, Vaage, Sejersted, & Newsholme, 1987; Chad & Quigley, 1991; DeVries & Gray, 1963; Edwards, Thorndike, & Dill, 1935; Maehlum, Grandmontantagne, Newsholme, & Sejersted, 1986; Quinn, Vroman, & Kertzer, 1994; Tremblay, 1988). The independent effects of intensity and duration of exercise, as well as the interaction between them, seem to indicate that intensity must be greater than 50 - 60% VO2max with a duration equal to or greater than 60 minutes for a significant elevation in EPOC to occur. Research studies provide
evidence for a prolonged elevation occurring in trained subjects who engage in exercise for greater than 45 min at a relatively high intensity (Belinski, Schultz, & Jequier, 1985; DeVries & Gray, 1963; Edwards et al., 1935; Maehlum et al., 1986). A thorough discussion of the issues surrounding both the controversy regarding RMR and EPOC will be presented in Chapter 2.

STATEMENT OF THE PURPOSE

The purpose of this study is to compare resting metabolic rate and post-exercise oxygen consumption measures between active, non-dieting females and active, low calorie dieting females, aged 19 - 25.

RESEARCH HYPOTHESES

The hypotheses undertaken in this study are:

a) caloric restriction below 1200 kcal/day accompanied by moderate amounts of aerobic exercise at an intensity above 50% VO\textsubscript{2max} and total volume equal to or greater than 4 hours per week, will result in a depressed RMR when compared to dietary intake of greater than 1700 kcal and exercise of the same intensity and volume, and

b) post-exercise oxygen consumption following moderate intensity and duration (greater than 60% VO\textsubscript{2max} and 20 minutes) will be of a smaller magnitude and shorter duration in low calorie intake (LC), exercising females as compared to females with comparable activity levels and normal caloric intake (NC).

While it is not the purpose of this study to demonstrate a prolonged EPOC, the elevation that might be expected to occur with an exercise intensity of 65% VO\textsubscript{2max} and duration of 45 minutes should be adequate to demonstrate any differences between groups.
STATISTICAL HYPOTHESES

The two statistical hypotheses outlining this study are as follows:
(a) \( H_0: \mu_{RMR} \) of Normal Calorie (NC) subjects is not different than the \( \mu_{RMR} \) of Low Calorie (LC) subjects, and;
\( H_a: \mu_{RMR} \) of NC subjects is greater than \( (>\) \( \mu_{RMR} \) LC subjects;
(b) \( H_0: \mu_{EPOC} \) of NC subjects is not different than the \( \mu_{EPOC} \) LC subjects, and;
\( H_a: \leq \mu_{EPOC} \) of NC subjects is greater than \( (>\) \( \mu_{EPOC} \) VLCD subjects

OPERATIONAL DEFINITIONS

In the course of the study, Low Calorie (LC) and Caloric Restriction refer to diets in which estimated total caloric intake does not exceed 1200 kcals per day. Chronic dieting defines individuals who have maintained LC's for a minimum of 6 months. These were measured by an initial self-report diet questionnaire (DEA) estimating average food intake and eating patterns and two 24-hour diet recalls (APPENDICES B and F).

Resting metabolic rate (RMR) is defined as the rate of energy expenditure taken at least 4 hours after a meal and any activity. While basal metabolic rate is considered a more precisely defined standard, it is more difficult to measure and verify. In this study, RMR will refer to the rate of energy expenditure taken within one hour of arising, at least 10 hours after any food and 12 hours after any physical activity.

BMI is the measure of Body Mass Index which is equal to body weight in kg + height in meters squared.

Excess post-exercise oxygen consumption (EPOC) is the elevation above resting oxygen consumption following a bout of vigorous exercise.

\( V_02_{max} \) is a quantified expression of an individual's maximal aerobic capacity or maximal aerobic energy transfer.

OC refers to a variety of oral contraceptive prescriptions.
ASSUMPTIONS

A primary tool for initial evaluation of physical activity participation and caloric intake will be self-report. It is well known that this method of acquiring data is dependent upon the accuracy and honesty of the participating subjects. While great effort will be exerted to obtain the most accurate and representative data from subjects, it has been noted that in the acquisition of dietary intake, an estimated 81% of subjects underreport intake by approximately 700 ± 379 kcals (Lee & Nieman, 1993).

LIMITATIONS

Shortcomings affecting the outcome of the study include a) limited sample size, which may not be representative of the larger population and which may have reduced the power of the statistical tests used; b) grouping of a variety of activities labeled "aerobic" which may not be truly reflective of a common aerobic intensity or demand across activities; and c) a quasi-experimental design, which precludes determination of a cause-and-effect relationship between LC diet and measures of RMR and EPOC.

DELIMITATIONS

Delimitations to this study include its focus on one gender. While it is well documented that males do engage in some of the same severe caloric restriction and hyperactivity that females do, especially in the sport of wrestling, it is generally considered to be of a more acute nature at the time of "making weight" for competition, and not on a prolonged or chronic basis.

A second delimitation reflects the age range of subjects, representing early adulthood. It is feasible that changes undergone in the adolescent body in severe caloric restriction and high volume exercise may have more pronounced manifestations in RMR, but these subjects are less accessible and require closer control measures, as well as being potentially more psychologically and physiologically vulnerable to the effects of caloric restriction and over-exercise. Older subjects may demonstrate changes in body composition known to occur with increasing age.
CHAPTER 2

LITERATURE REVIEW

A social phenomenon which has been steadily growing in our American culture since the early 1960's is the emphasis on the lean and thin female as the epitome of desirability. Magazines, billboards, television, fashion and almost any media campaign promote this image, and young women today are besieged with this "ideal woman." As strong and sleek females become more the model, the pursuit of this ideal has produced a population of driven, underfed, and overactive women. While promotion of health through physical fitness and good nutrition are highly valued pursuits, the "more is better" mentality often adopted by this sub-population takes these pursuits beyond optimal levels and onto a level of potentially detrimental behaviors. Professionals become faced with the challenge of determining if there is a point at which "enough" becomes "too much."

The beneficial role of exercise in weight reduction is well-known and uncontested. Exercise acts to increase caloric expenditure by the body, and when not accompanied by an increase in food consumption, helps to create a negative energy balance. Conflicting evidence exists, however, regarding the effects of prolonged aerobic exercise in affecting a chronic elevation in resting metabolism. Some factors contributing to the inconsistent findings in these studies include variations in the research methodology, as well as individual factors such as nutritional status and genetic variation. The currently held hypothesis is that a program of aerobic exercise in conjunction with moderate caloric restriction will provide an optimal stimulus for fat loss, decelerate or prevent lean muscle loss, and reverse or impede declining basal and resting metabolic rates normally encountered with caloric restriction (Nieman, Haig, De Guia, Dizon, & Register, 1988).

In conjunction with the use of exercise to more effectively lose weight, studies on acute responses to exercise also present conflicting evidence as to whether either the intensity and/or duration of an activity produce a prolonged elevation in oxygen uptake following exercise: excess post-exercise oxygen consumption (EPOC). Equivocal findings on the influence of aerobic training on this response further confound the issue. While a prolonged elevation may translate into minor caloric increases per exercise
session, over multiple sessions per week, it may contribute to increased overall caloric expenditure and negative energy balance.

While it is quite probable that the consequences of the interaction of restricted caloric intake and exercise are applicable to males as well as females, there are gender differences which must be considered. Women present a unique situation by virtue of their monthly menstrual cycle, which is regulated by a delicate balance of fluctuating hormone levels. These fluctuating hormone levels have been proposed to influence subtle differences in exercise responses and inherent differences in metabolism between the genders. Likewise, there may be cyclic changes within each individual that could alter her metabolic responses at rest or exercise during the different menstrual phases of each cycle. Discussion regarding the role of menstrual cycle and status is intended to provide supplemental information to foundational material used to develop the research hypotheses.

This chapter will examine some of the current literature in three main areas of research: the effect of caloric restriction and exercise on basal and/or resting metabolic rate (BMR or RMR); exercise intensity and duration on post-exercise oxygen consumption (EPOC); and lastly, the effect of menstrual cycle phase and status, and oral contraceptive use, on exercise performance.
THE EFFECT OF DIETING AND EXERCISE ON RESTING METABOLIC RATE

Prevalence Of Dieting Through Caloric Restriction And/Or Exercise

The practice of reducing caloric intake to affect a decrease in body weight is a highly common, well-accepted practice in our society today and is found just as frequently in populations of normal weight individuals as overweight. The 1985 National Health Institute Survey Findings (Stephenson, Levy, Sass, & McGarvey, 1987) on nutrition knowledge developed population estimates from its extensive data and predicted that 44% of women and only 25% of men were trying to lose weight through means of diet and/or exercise. When divided into weight categories -- overweight = BMI ≥ 27.8 for men and BMI ≥ 27.3 for women; lean = BMI < 20.5; normal = range between lean and overweight -- these same estimates found that more normal weight individuals (34.1 million) were attempting to lose weight than overweight individuals (22.6 million). Of the lean individuals trying to lose weight (approximately 3 million), 97% were women. Fifty-six percent of normal-weight women perceived themselves as overweight, and younger women were approximately four times more likely than same-aged men to view themselves as overweight. There is an elevated general awareness regarding weight among women, with an estimated 20% of lean women engaging in inappropriate dieting and weight loss. Wardle and Marsland (1990) investigated adolescent concerns about body weight and attitudes towards eating and reported the following findings: of 459 girls aged 11-18, 50% had feelings of being too fat, and dieting was common (26% of total females), especially in white, higher socioeconomic schools. When asked to rate thighs, bottom, and hips, 50-60% of girls responded "too big."

In a study of the role of exercise in the development of women's weight and diet concerns, Davis, Fox, Cowles, Hastings, and Schawass (1990) found that of 112 regular exercising women, 56% were on a diet and 76% wanted to lose weight. In addition, they found that the subject's basal metabolic rate (BMR), which was reduced during dieting, didn't return immediately with resumed refeeding. Guthrie (1985) found that among 384 intercollegiate athletes, 64% used their sport training as a means to control or lose weight; 54% complemented training with additional exercise; 26% used restrictive dieting or fasting; and 16% used laxatives, vomiting, diuretics, or other methods of purging.

In the past two decades there has been a tremendous increase in the numbers of women participating in intense athletic activity. During the same course of time there has
been a steady increase in the incidence and prevalence of the eating disorders of anorexia nervosa and bulimarexia. Shangold and Mirkin (1988) cite a probable increase of 100% since 1960. Prevalence of these disorders in the general population is estimated to be approximately 1 - 5% for anorexia nervosa and 5 - 10% for bulimarexia (Brownell, Rodin, & Wilmore, 1992; Shangold & Mirkin, 1988). In the arena of athletics, and with the cultural/societal emphasis on physical appearance, it is not at all uncommon to see the rates of incidence and prevalence higher than national or worldwide figures. Borgen and Corbin (1987) found convincing evidence for higher levels of symptoms associated with eating disorders among female athletes in sports which emphasized thinness (track, gymnastics), while Garner and Goldfinkle (1980) reported the prevalence of anorexia in female ballet dancers at 6.5% -- well above the national figures of 1%. Kirkley (1986) cites an estimated 5 - 20% of college-age women engage in bulimic behavior, whereas Halmi, Falk, and Schwartz (1981) reported that in a survey of 355 college students in New York, 19% of the women and 5% of the men engaged in bingeing practices.

The indication is that people, especially women, are compelled by personal and socio-cultural reasons to control their body weight. Dieting through caloric restriction and exercise are the two most widely used means, but desperate individuals may seek out more desperate means. An examination of the literature regarding the impact of diet and exercise on specific physiological parameters reveals contradicting and confusing information when attempting to determine beneficial levels of caloric restriction and increased physical activity for weight loss or maintenance.

**Effects of Caloric Restriction on RMR**

It is well documented that resting metabolic rate is decreased with prolonged caloric restriction. The pioneering work of Keys and colleagues (Keys, Brozek, Henshel, Mickelsen, & Taylor, 1950) in the area of human starvation provides the foundation for much of what is understood about physiological and psychological responses to food deprivation and increased energy efficiency of metabolically active tissue. Bray (1969) found that a decrease in caloric intake from 3500 kcal/day to 450 kcal/day resulted in a decline in RMR of 15% after three weeks of restriction, while Mole, Stern, Schultz, Bernauer, and Holcomb (1989) showed a decrease only five days after onset of restricted dieting (500 kcal/day). The initial reduction in RMR has been primarily attributed to a directly proportional decrease in metabolically active tissue (FFM). With any continued decrease in RMR, RMR/FFM is not additionally affected (Grande, Anderson, & Keys,
Lammert and Hansen (1982) compared the metabolic responses of nine non-obese subjects during two weeks of normal (2,000 - 3,250 kcal/day), overfeeding (+3,000 kcal/day), and semi-starvation (500 kcal/day) eating periods. During the period of overfeeding, both resting and exercise energy expenditure were increased above normal diet values, while in the semi-starvation period, mean resting and exercise energy expenditure decreased below initial values. While the decrease was not seen as significant, the semi-starvation period was initiated immediately post-overfeeding, which had produced an 11% increase in resting metabolism. Had the starvation period followed the normal diet period, it is quite conceivable decreases in RMR may have been more dramatic.

### Composition of Diet

The composition of a very low calorie diet (VLCD) has been considered as a factor in changes in weight/fat loss. Diets which emphasize low-carbohydrate and high-protein intake have been implicated as contributing to the retention of greater lean body tissue, thereby leading to greater amounts of metabolically active tissue. Nieman et al. (1988) found that despite a greater caloric deficit created through exercise, in addition to a restricted caloric intake, subjects in exercise and nonexercise groups displayed no differences in body composition after five weeks. This lack of difference was partly attributed to a protein composition in the diet of 63g (~1.27 g · kg⁻¹) which acted as a protective effect on lean body mass (LBM). Phinney, LaGrange, O'Connell, and Danforth (1988) compared twelve overweight, calorie-restricted individuals (mean 720 kcal/day) over 4 weeks. Six were randomly selected into a sedentary group, while the other six received aerobic exercise (27 hours total at 50% VO₂max). Both groups consumed a diet that included 1.5 g · kg⁻¹ protein based on ideal body weight. Lean tissue was well maintained in both groups and VO₂max was unaffected, while resting VO₂ of both groups declined 10% within the first week of dieting, and continued to decline another 17% in the exercise group by the third week. Preservation of LBM refutes the hypothesis that the reduction in RMR seen with caloric restriction and weight loss is a function of the loss of metabolically active tissue. In another study on metabolic responses to conditions of starvation, low-calorie ketogenic, and nonketogenic diets, 6 obese subjects ate either an 800 kcal low-carbohydrate, high-fat (5% and 70%, respectively), 800 kcal mixed-diet (45% CHO and 30% fat), with a constant protein content of 25% (Yang & Van Itallie, 1976), or were starved during three 10-day intervals.
over a 50-day study period. Each diet period was separated by 5 days of a 1,200 kcal mixed diet, during which weight loss was stabilized or modest weight gain occurred. Weight loss occurred in all diets, with an average loss of 2.78 ± .32 kg in the mixed diet and 4.66 ± .51 kg in the ketogenic diet. The total percentage of weight lost as protein was found to be statistically nonsignificant between the two diets (3.4 and 3.8%, respectively), although absolute values differed (.095 kg and .17 kg per day, respectively) and mean protein loss in the ketogenic diet was higher. Weight loss in the starvation group was much greater, and averaged to be approximately 22.52 ± 1.5 kg, with 6.7% (1.5 ± .13 kg) lost as protein. Basal metabolic rate declined directly as a function of the total weight loss (-12.3 ± 3.4% RMR: -13.3 ± 1.4% change in body weight) and was unaffected by the type of diet. The evidence then, seems to indicate that it is the magnitude of the caloric deficit that has the greatest effect on RMR, such that even when lean body mass is maintained, metabolic rate still declines in response to other factors, including total weight loss.

Effects of Exercise on RMR

A general disagreement exists in the literature regarding the effect exercise might have on resting metabolic rate. Poehlman, Melby, and Goran (1991) cite several factors which contribute to the inconsistency of the findings: a) the variety of modes, intensities, and durations utilized in the various studies make comparison ineffective; b) same day measurements (pre/post exercise) versus separate day baseline and exercise measurements, which introduces a factor of intra-individual variability; and, c) a variety of sample sizes, gender, age, and physical fitness levels may influence findings, as well as the timing of resting metabolic rate measurement. Additionally, findings have proven to be indefinite due the reliance on observational, cross-sectional studies, but this design does allow for comparative analysis between individuals having broad differences in physical activity levels and for control over physical variabilities such as body composition. Other weaknesses in the research include the random interpretation of "trained" versus "untrained" across studies, and the presence of genetic or other unknown factors which contribute to individual differences. Acknowledging the existence of these discrepancies, a brief examination of some of the available evidence both in support of and against findings of chronic changes in metabolic rate as a result of exercise follows.

Studies demonstrating increases in RMR with exercise. Several studies have reported increases in resting metabolic rate as a chronic response to exercise training.
Tremblay, Fontaine, and Nadeau (1985) compared eight trained endurance athletes to five untrained subjects eight hours postprandial and 16 hours post-exercise. While it cannot be determined from this design if an acute effect of prior activity influenced RMR measurement, the trained athletes exhibited a 15% higher absolute RMR over non-trained subjects. When normalized as kg · FFM⁻¹, this elevation was not significant. A finding that was of note, however, was that in a subgroup of the most highly trained subjects (12 - 16 h/wk) (n = 4), RMR was significantly greater than in those subjects training approximately six to ten h/wk (2.11 ± 0.14 vs 1.69 ± 0.04 ml · kg⁻¹ · min⁻¹, respectively). Also, since body weight and FFM were slightly lower in the highly trained group, their elevated RMR could not be attributed to greater lean mass. In another study, Tremblay et al. (1986) compared 20 trained and 39 non-trained males and found both absolute RMR and a regression of RMR on fat free mass to be significantly higher in the trained subjects. No significant difference in the slopes between the two groups was observed, but the intercept for the trained (.256 ± .638 kcal · min⁻¹; r = 0.61) was greater than for the untrained (-.177 ± .408 kcal · min⁻¹; r = .44). When the same study was duplicated on 8 moderately obese subjects placed on an 11-week program (5 h/week aerobic exercise at ~50% VO₂max), an increase in RMR corresponding to 8% of pre-training values was observed. The findings led authors to suggest that for a given quantity of FFM, a greater level of energy expenditure is found in very active individuals when compared to untrained.

Poehlman, Melby, and Badylak (1988) examined RMR and postprandial thermogenesis in trained and untrained men (n = 18). Measurement was taken 24-hours after the previous exercise bout, in an attempt to minimize the influence of elevated post-exercise O₂ consumption, and 12 hours postprandial. RMR was higher in the trained group both in absolute terms and kg · FFM⁻¹ by 9 and 11%, respectively. It is suggested that an association between the elevation in RMR and exercise training might only be apparent in very highly trained athletes (VO₂max ~ 70 ml · kg⁻¹ · min⁻¹), in support of similar findings by Tremblay et al. (1985, 1986). Interestingly, the thermic effect of a meal was significantly less in the trained group, which was consistent with findings by LeBlanc, Diamond, Cote, and Labrie, (1984) and Tremblay et al. (1985).

Poehlman, McAuliffe, Van Houten, and Danforth (1990) investigated the mediating factors of age and endurance training on metabolic rate in healthy men by measuring RMR 36 hours post-exercise in 42 younger (18 - 36 yr.) and 26 older (59 - 76 yr) men. Subjects were divided into four groups: younger trained (n = 22), younger untrained (n = 22), older trained (n = 11), and older untrained (n = 15). Training spanned 5.0 ± 1.1 yr for the young group and 15.4 ± 4.2 yr for the older group. When adjusted
for FFM, RMR in the younger and older trained groups was found to be between 5 and 7% higher, respectively, than in the non-trained groups. Age did not prove to be a factor when expressed as kg · FFM⁻¹. As in the earlier findings by Tremblay et al. (1986), the slopes of the two groups did not differ, but the intercept was greater in the trained groups (~ 1350 kcal · 24 hr⁻¹ trained vs ~1150 kcal · 24 hr⁻¹ untrained). The authors concluded that the level of physical activity in the form of endurance training may modify the energy requirements at rest.

Relatively little is known about the impact of resistance training alone on RMR, but because the energy cost in resistance training is considered to be lower than that of an aerobic workout, it is not viewed as placing the same type of demands on the oxygen energy systems. Anaerobic energy systems are the primary source of fuel utilization, with no increases in blood fatty acids or other lipids found with intense weight training. The volume rather than the intensity of work may be an important determining factor in fuel usage (Walberg, 1989). What has been considered in weight training are the positive effects it can have in increasing fat-free mass relative to fat mass. Since muscle tissue is more metabolically active, it would be expected that the greater the percentage of lean muscle tissue, the greater the metabolic rate and energy requirements of the body. A recent study by Casenhiser-Sutterluety, Kirby, and Sherman (1994) indicated that resistance training in conjunction with a VLCD resulted in a significantly smaller decline in RMR than the decline seen with aerobic exercise and a VLCD. This relationship, though, has not been clearly established and requires further inquiry.

**Studies demonstrating no increase in RMR with exercise.** For every study demonstrating an elevation in RMR with exercise training, there seems to be one which contradicts such findings. LeBlanc et al. (1984) compared seven untrained men and seven trained long-distance runners who had trained for a minimum of three years and were presently completing 100 - 160 km · wk⁻¹. Initial RMR measurement was taken 10.5 hours postprandial, with no mention of last exercise period. No significant differences between groups (3.51 ± 0.41 and 3.68 ± 0.42 ml · kg⁻¹ · min⁻¹, trained and non-trained, respectively) were found. It is unlikely that any post-exercise elevation was influencing the measurement, since the trained group actually demonstrated a lower RMR. As was seen in the work of Poehlman et al. (1988), a significantly reduced thermogenic response to food was observed in the trained subjects. Since this finding has not been consistently reported in studies which have used less well-trained subjects, it is suggested that level of training may be a factor. As a follow-up to this study, LeBlanc, Mercier, and Samson (1984) investigated diet-induced thermogenesis in relation to training level in females. Subjects were divided into three equal groups of ten and
classified by level of training into untrained, moderately, and highly trained groups. While no significant differences were detected in RMR between groups, there was a distinct trend for fasting VO$_2$ to increase with the level of training (3.00 ± 0.14, 3.21 ± 0.16, and 3.35 ± 0.15 ml · kg$^{-1}$ · min$^{-1}$ in untrained, moderately and highly trained subjects, respectively).

Hill, Heymsfield, McMannus, and DiGirolamo (1984) measured RMR 36 hours post-exercise in 4 highly trained (VO$_{2\text{max}}$ = 62 ± 3 ml · kg$^{-1}$ · min$^{-1}$) and 4 untrained (VO$_{2\text{max}}$ = 43 ± 2 ml · kg$^{-1}$ · min$^{-1}$) subjects. RMR · FFM$^{-1}$ was not determined to be significantly different between the two groups, but was noted to be 9% greater in the trained. The fact that a 9% difference was not statistically significant may reflect a problem of statistical power due to small sample size, as 9% of RMR/FFM may be equated to approximately 80 - 100 kcal. Poehlman, Despres, Bessette, Fontaine, Tremblay, and Bouchard (1985) measured RMR following a 12-hour overnight fast (prior exercise period not noted) in 14 trained and 10 non-trained males. No difference was seen between groups, but it was noted that a wide variation in RMR in the untrained groups may have contributed toward a masking effect if, indeed, a difference did exist.

Forty-four females, both lean and obese, aged 18 - 65 were measured for caloric expenditure in a study by Owen et al. (1986). Of these 44, eight were considered to be highly trained. The purpose of the study was to examine the accuracy of currently available tables and regression equations utilized in estimating RMR of healthy women. In the course of the study, no difference between the highly trained and non-trained groups was observed, but due to the small sample size of the latter group, statistical power may again have been a factor. As a note of interest, it was found that predictive methods for RMR may over- or under-estimate the value by 21 - 33% in non-athletes, whereas in well-trained athletes, these values may predict within 8 - 10% of measured values. Tremblay et al. (1989) followed five men who engaged in cycle ergometry exercise 6 day/week (intensity not specified) over a 100-day period. No dietary manipulations were included, but exercise was estimated to induce a 4.2 MJ (1000 kcal)/day deficit. While most training effects were noted within 25 days, RMR and the thermic effect of food were not significantly modified over the course of the study. If exercise does produce an elevation in RMR with training, but pronounced caloric deficit promotes its decline, the two mechanisms could be counteracting each other. Weight loss in these subjects corresponded to ~9% of initial body weight, a finding in agreement with earlier work and equivalent to the noted mean difference in RMR in trained and untrained subjects (Tremblay et al. 1986). It is hypothesized that if weight loss were substantially higher, a reduction in absolute RMR would have been noted. The authors propose
though, that even in such conditions, training provides for greater maintenance of RMR when compared to weight loss through caloric restriction alone.

A recent study by Westerterp et al. (1994) compared sleeping metabolic rate (SMR) in 11 women and 12 men before, during, and after a 40-week training period. Subjects decreased fat mass an average of 3.5 kg and increased LBM average of 2.7 kg, with an overall mean decrease in body mass of one kilogram. A small but insignificant decrease in SMR, which was related to the decrease in body mass, was noted at 40 weeks. Similarly, Goran, Calles-Escandon, Pochlman, O'Connell, and Danforth (1994) found no changes in RMR following 10 days of varying conditions of high flux energy balance (50% increases in activity and energy intake), negative energy balance (50% increase in energy expenditure), and positive energy balance (50% increase in energy intake) in three different groups of male subjects. However, the brief duration of this study may not have been sufficient to influence any chronic changes in RMR.

There seems some evidence indicating that a chronic change in RMR may be affected with exercise of adequate intensity and duration over a prolonged period of time. Due to inconsistencies in reporting conditions of baseline measurement, and the design of the studies, it becomes difficult to establish if RMR is being measured in a true resting condition or simply reflecting a prolonged elevation in post-exercise oxygen consumption. The standard definition of RMR states only that the measurement be taken following a minimum 10 - 12 hour fast and cessation of activity (Poehlman et al., 1991). If EPOC duration can potentially last from 12 - 24 hours post-exercise, as several studies show (Edwards, Thorndike, & Hill, 1935; Maehlum et al., 1986), and RMR measurement is taken prior to 24 hours post-exercise, it becomes difficult to confirm with certainty that any noted RMR elevation is a chronic effect rather than an acute response to exercise. Several of the studies previously cited in the review of literature either do not specify the interval of time between the exercise bout and measurement of RMR, or test within a twenty-four hour period. It does seem that aerobic exercise may result in an acute response of elevated metabolism, but genetic differences and level of training seems to be major factors confounding any final conclusions regarding the long-term effects.

Caloric Restriction in Conjunction with Exercise on RMR

The rationale behind the prescription of exercise in conjunction with calorie restriction is threefold: first, it is thought to accelerate fat loss by creating a greater negative-energy balance; secondly, it has been proposed to arrest or prevent the decline in
RMR known to occur with dietary restriction; and lastly, it is believed to help maintain fat free mass, which is often lost with fat weight in calorie restricted dieting (Walberg, 1989; Hill, Sparling, Shields, & Heller, 1987; Yang & Van Itallie, 1976). Some investigators counter this argument and report that severe caloric restriction in combination with large volumes of aerobic exercise may act to exacerbate the decline in RMR (Phinney et al. 1988; Poehlman, 1991), and still others report no changes occurring at all (Hill et al., 1987). It must be remembered when comparing these studies that a wide variety of protocols, subject characteristics and methodologies exist among them, as well as the fact that the majority of studies have been conducted on moderately overweight to obese subjects. How this applies to normal weight subjects has not been definitively established.

Studies demonstrating an increase in RMR with exercise during caloric restriction. Several reports of positive changes in RMR due to the addition of exercise in a hypo-caloric state have been made. Lennon, Nagle, Stratman, Shrago, and Dennis (1985) investigated the impact of 30 minutes of activity every other day for 12 weeks in overweight subjects who followed 1200, 1500, or 1800 kcal/day diets. RMR significantly increased in the activity groups over a non-active, dieting control group, leading the authors to conclude that "any level of exercise" would be beneficial, especially for females, in preventing the decline in RMR during caloric restriction (p. 39). In a study on obese subjects, RMR levels declined to an equivalent of 87% pre-dieting values after two weeks of dieting at 500 kcal/day (Mole et al., 1989). With the addition of daily exercise (30 min; 60% VO2max) RMR values returned to pre-dieting values.

Nieman et al. (1988) examined the effects of a 5-week exercise program, consisting of 45 minutes of a walk/jog protocol at 60% VO2max, 5 days a week, on 21 mildly obese females. Subjects ate a diet of 1300 kcals/day. When compared to a control group on the same diet without exercise, the exercising subjects showed a 6% increase in RMR 48 hours post-exercise. The results led Nieman and colleagues to suggest that intensity of exercise may play a role in increasing RMR.

Recently, Svendsen, Hassager, and Christiansen (1994) examined the effect of physical activity on body composition and RMR in overweight, post-menopausal women. They found that compared to the control group, the diet/exercise group demonstrated a significant increase in RMR of approximately 11% after 12 weeks. The authors concluded that in spite of age-associated declines in metabolic rate, moderate amounts of aerobic and anaerobic exercise could benefit overweight, post-menopausal women who are restricting caloric intake.
Other studies (Bailor, Katch, Beque, & Marks, 1988; Casenhis-Sutterluety et al., 1995; Lemons, Kreitzman, Coxon, & Howard, 1989; Walberg, 1989) have found that exercise, especially isotonic resistance training, provided a beneficial effect in increasing the RMR \cdot FFM^{-1} ratio during caloric restriction, thus improving overall RMR. Bailor et al. (1988) measured changes in LBM in obese women who weight-trained three times per week while consuming a diet 1000 kcal below maintenance. Lean body weight increased in the weight training and diet group while it decreased in the diet-only group. Resistance training did not increase fat loss relative to energy restriction, leading the authors to conclude that energy restriction and weight training act independently, with energy restriction acting to decrease body fat and weight training acting to increase lean body mass.

Lemons et al. (1989) divided four groups of ten overweight women into three exercise groups (aerobic, resistance, and a combination group) and a diet-only group. No significant differences were found between the diet-only and any exercise group, but the relative efficiency of the metabolic rate per kilogram fat-free mass in the resistance groups was significantly different (p < .05) by up to 10% of initial values. Casenhis-Sutterluety et al. (1995) recently found a significantly smaller decline in RMR in a group of subjects engaging in resistance training and a VLCD (800 kcal) when compared to a similarly dieting group who performed aerobic exercise. The resistance-trained group demonstrated a decline in RMR of 4.1 \pm 1.6\% initial resting VO_{2}, whereas the aerobic exercise group's RMR declined 16.2 \pm 2.4\% within 12 weeks. A diet-only group demonstrated a 17.6 \pm 2.5\% decline. The results of these studies indicates that weight training sustained during caloric deficit may attenuate or prevent the decline in RMR \cdot kg FFM^{-1}.

**Studies showing a decline or no increase in RMR with exercise during caloric restriction.** A large body of evidence supports the contention that exercise fails to arrest the decline of RMR occurring with caloric restriction. Van Dale and Saris (1989) found that while RMR decreased less in a group of "yo-yo dieters" who engaged in exercise four hr/week at 60\% VO_{2max} for 14 weeks, it decreased significantly for diet-only as well as diet/exercise participants alike. Prior to this study, Van Dale, Saris, Schofflen, and Ten Hoor (1987) studied obese women who were divided into diet-only and diet/exercise groups. Diets followed the same pattern in both groups, while the exercise groups participated in four hours/week at 50 - 60\% maximal aerobic power. Both groups showed a significant decline in RMR \cdot kg FFM^{-1} after 12 weeks. Warwick and Garrow (1981) investigated three women who were placed on an 800 kcal/day diet for 12 - 13 weeks and alternated between exercising and not exercising every three to four weeks (2
hour/day in 15 minute intervals on a bike ergometer). BMR was shown to decrease steadily throughout the study regardless of exercise periods. Krokiewski, Toss, Bjorntorp, and Holm (1981) found a significant decrease in the RMR of a group of diet/exercising obese women on a 500 kcal, three days/week, three-week long program, which could not be explained by changes in body composition, since a diet-only group demonstrated equivalent changes.

Phinney et al. (1988) found RMR to decline in both diet-only and diet/exercise groups (720 kcal; 30 min. at 50% VO_{2max} daily) by 10% within 7 days in a study of obese women. When exercise was increased to one-hour daily the second week and one-hour twice daily the third and fourth week, the decline increased to 17% below pre-dietary levels. This intensity and duration should have been adequate to reverse RMR decline if, indeed, exercise acts as a stimulus.

In another study of obese women, Hill et al. (1987) found no significant difference in the total decline in RMR between exercising and non-exercising diet-restricted subjects, (19.1% vs 17.3%, respectively) although the rate of decline was different between the two groups. After only one week, RMR declined significantly in both groups (5% and 16%, exercise and non-exercise, respectively), but then remained relatively stable in the non-exercising group while the exercising groups showed steady decline. The slopes of the weekly change between groups were also compared, and RMR of the exercising group changed at a rate of 2.02 ± 0.24 kcal · wk^{-1} while the rate of sedentary subjects changed at a rate of 1.20 ± 0.30 kcal · wk^{-1}. The decline could not be explained entirely by the loss in FFM, since the exercising group lost more weight from fat than from lean tissue but had no more weight loss in absolute values. In this study, exercise did not prevent the decline in RMR. Since caloric deficit was greater in the exercising subjects, and it has been suggested that RMR declines proportionally to the magnitude of caloric deficit, it could be expected that exercising subjects would show a greater decline in RMR, but this was not the case. The authors concluded that while moderate aerobic exercise may have positive effects for weight loss when combined with diets of only 800 kcal, the results were inconclusive. There was, however, "an indication that exercise may attenuate the expected decline in RMR" (Hill et al., p. 629). Poehlman et al. (1991) countered this conclusion in their review of the literature, stating that the decrease in RMR may be accelerated and exacerbated with moderate to large quantities of aerobic exercise and VLCD, especially if resulting in substantial weight loss.

When comparing studies which investigate energy intake and exercise on RMR, a significant pattern emerges: In those studies which found a stimulated RMR post-exercise, caloric intake generally ranged from 1200 - 2000 kcal · day^{-1}, and exercise
sessions were equated at approximately 60% VO$_{2\text{max}}$ for 30 - 45 min. per session (300 - 320 kcal) (Lennon et al., 1985; Nieman et al., 1988). Studies which showed no reversal in RMR decline (due to caloric deficit) with the addition of exercise indicate subjects consumed less than 800 kcal/day and engaged in aerobic activity ranging between one and two hours at an intensity equivalent to either 50 - 60% VO$_{2\text{max}}$ or 392 ± 105 kcal (Warwick & Garrow, 1981; Phinney et al., 1988; Van Dale et al., 1987). Mole et al. (1989) found the RMR of five obese women decreased ~13% from pre-dieting levels after two weeks of a 500 kcal diet. When 30 min · day$^{-1}$ of exercise at approximately 60% VO$_{2\text{max}}$ was then added to the diet, the fall in RMR was reversed, returning to pre-dieting levels. It would appear that in diets of 1200 kcals or greater, the inclusion of exercise at a duration of less than 60 minutes and intensity ~60% of maximal aerobic capacity seems to affect an increase in RMR from the declining values seen as result of caloric restriction. Conversely, with caloric intake below 1200 kcals and exercise duration greater than 60 minutes, the same intensity of exercise produces a continued decline in RMR. Short bouts of exercise may provide a stimulus to RMR, especially when caloric intake is adequate to maintain required resting energy levels. When caloric intake drops below daily requirements for normal resting metabolism, prolonged exercise, such as what is commonly prescribed for "greater fat burning," acts to inhibit rather than stimulate RMR.

It is conceivable that if the individual is of normal weight, without large stores of fat to be lost, decline in BMR or RMR due to caloric restriction may even be greater with the addition of exercise. It is probable that an optimal level of both exercise and caloric restriction exists which would offset the decline in RMR and maintain lean tissue mass, but identifying this precise level has been elusive. To reiterate factors contributing to the difficulty in identifying optimal combinations of diet and exercise in the studies examined, consideration must be given to the heterogeneity of populations examined (obese v. non-obese, trained v. non-trained, etc.), differences in mode, frequency, intensity and duration of protocols, variance in definitions of "caloric restriction," composition of caloric intake, timing of intake, and subject sample size.
EXCESS POST-EXERCISE OXYGEN CONSUMPTION (EPOC)

Current knowledge and the development of understanding about EPOC is often attributed to the work of Edwards, Thorndike, and Hill (1935) and Hill, Long, and Luptin (1924), although documentation of studies identifying a prolonged elevation in oxygen consumption following exercise exists from 1913 and the early 1920's (Trost, 1994). Termed "O2 debt," Hill and Luptin (1923) proposed that an oxygen deficit occurred during exercise as a result of the oxidation of a small portion of lactate produced under the anaerobic conditions of exercise. The authors identified two phases of repayment of the O2 debt: an initial ("rapid") phase, thought to be due to the oxidative removal of lactic acid in the muscle, and a second ("prolonged") phase, thought to be due to oxidative removal of lactic acid which diffused from the muscle into the bloodstream (Trost, 1994).

It has since been determined that lactate removal is only a very small part of the increased post-exercise oxygen consumption, and that the extra energy cost needed to pay the "O2 debt" is comprised of several factors, including oxygen resynthesis of phosphocreatine, ATP and ADP, elevated catecholamine concentrations, lipolysis of triglycerides and FFA release, increased mitochondrial calcium uptake and respiration, and increased body temperature (Poehlman et al., 1991). The two phases of recovery noted by Hill and Lupton (1923) have been further refined into three components: the "fast component," which decays within 2 - 3 minutes after exercise and is independent of intensity and duration of exercise; the "slow component," which can last from 10 - 90 minutes post-exercise and is dependent on intensity and duration, and; the "ultra-slow component," in which metabolic rate could be elevated by 10 - 20% above pre-exercising value more than 2 hours post-activity (Mole, 1990). "Excess post-exercise oxygen consumption" (EPOC) is now considered a more accurate term, free of lactate-related "cause and effect" implications (Gasser and Brooks, 1984, cited in Poehlman, 1991).

Studies demonstrating prolonged elevation post-exercise. Edwards et al. (1935) reported that following two hours of intense American football, oxygen consumption remained elevated 25% above resting values for fifteen hours. The authors concluded that 50% of the total energy required for the two-hour participation was accounted for in the post-exercise oxygen elevation. Several weaknesses to this study must be noted, though. VO2 measurement was assessed only prior to activity, with no standardized baseline measure for comparison. No report of preceding activity or food intake is mentioned, and therefore one must question if these had been controlled. Passmore and Johnson (1960) determined post-exercise VO2 in 10 men following a 10
mile (4.0 mph) treadmill walk and found that VO$_2$ remained elevated above baseline at least seven hours. Again, no controlled baseline measure was taken, nor was the magnitude of the elevation (kcal · min$^{-1}$ or ml · kg$^{-1}$ · min$^{-1}$) determined.

Using a previous day's RMR measure as baseline, prior to which no activity had been performed, DeVries and Gray (1963) found post-exercise VO$_2$ to be elevated 8 - 28% above baseline for six-to-eight hours after 45 minutes of a combined exercise session of cycle ergometry, walking/running, and bench stepping. The energy cost of EPOC was estimated to be ~57 kcal. While only two male subjects were utilized and the intensity of the various exercise protocols lacked consistency, the use of multiple control and exercise days over a six-week period for mean comparison may in some ways compensate for these limitations. Bielinski, Schultz, and Jequier (1985) confined 10 male athletes to a respiratory chamber for two days. The first day was a rest day, which was followed on the second day by three hours of treadmill exercise at 50% VO$_{2\text{max}}$. Total energy cost of the session was ~2100 kcals. Meal intake was also accounted and controlled for, with meals on both days identical, and exercise the second day immediately preceding the lunch meal. During the 4.5 hours between meals, energy expenditure was 9% greater (40 kcal) on the exercise day. This elevation was not significant beyond 4.5 hours after exercise, although it did remain above resting values for a total of nine hours, but the morning following exercise metabolic rate was 4.7% higher in two of the subjects than on the morning following rest.

Maehlum et al. (1986) found a significant elevation in the metabolic rate in 8 male and female subjects for 12 hours following a 65 - 90 minute bout of cycle ergometry at 70% VO$_{2\text{max}}$ compared to resting conditions. The elevated VO$_2$ post-exercise accounted for approximately 120 kcals additional energy expenditure, or about 14% of the total energy cost of exercise. Twenty-four hours after exercise, the metabolic rate was significantly higher than in a similar period post-rest. In addition, the meal ingested post-exercise increased VO$_2$ significantly more than the same meal ingested during the control period. Due to the combined duration of EPOC with the increased thermogenic effect of food, the authors concluded that exercise may be advantageous during weight loss by increasing caloric expenditure and stimulating basal metabolic rate.

Two recent studies investigated the independent effects of duration and intensity of exercise on EPOC (Chad & Quigley, 1991; Quinn, Vromen, & Kertzer, 1993). Chad and Quigley compared trained and untrained females at intensities of 50 and 70% VO$_{2\text{max}}$ for 30 min. Both groups demonstrated a significantly greater post-exercise VO$_2$ following exercise at 50% than at 70% maximal oxygen consumption. At 180 minutes post-exercise, both trained and untrained groups continued to demonstrate a significant
elevation in VO₂ of approximately one L · min⁻¹, regardless of the intensity level of the exercise. Quinn et al. (1993) utilized a constant walking intensity of 70% VO₂max with varying durations of 20, 40, and 60 minutes on eight trained female subjects. EPOC was measured for three hours post-exercise and was found to be significantly elevated above pre-exercise values in all three duration trials, and total EPOC was significantly elevated in the 60 min trial (15.2 L) as compared to either the 20 (8.6 L) or 40 min trials (9.8 L).

**Studies demonstrating no prolonged elevation.** Several studies have failed to demonstrate any significantly prolonged elevation in VO₂ following exercise. Knuttgen (1970) had subjects complete 15 to 55 minutes of bicycle work at various intensities ranging from 45 - 98% of VO₂max. The magnitude of EPOC was positively correlated to both the intensity and duration of exercise. Fifteen minutes of work at 45% and 98% VO₂max resulted in equivalent energy expenditures of ~9 to 25 kcal, respectively. At 60% VO₂max, 15 and 55 minutes of exercise translated into energy expenditures during EPOC of only ~8 - 10 kcals, respectively. While the duration of EPOC was not reported, the low magnitude suggests it was very likely less than one hour.

Freedman-Akabas, Colt, Kissileff, and Pi-Snyder (1985) divided 23 male and female subjects into high, medium or low fitness categories, and had them perform 20 minutes of treadmill exercise at their predetermined anaerobic threshold. VO₂ was measured periodically for 220 minutes following exercise, and compared to an identical non-exercise control period. Within 40 minutes post-exercise, VO₂ had returned to baseline in all three groups. Using the highly fit group, these subjects also completed a second experiment. Alternately completing a fixed-duration, increased intensity, or fixed-intensity, longer duration treadmill run, VO₂ was only slightly and insignificantly higher when compared to the non-exercise control period. Pacy, Barton, Webster, and Garrow (1985) used an unusual exercise protocol to examine the effect of exercise on metabolism during fasted and fed states in lean subjects. The exercise protocol involved subjects working at 33 - 55% VO₂max on a cycle ergometer for four-20 minute bouts, each separated by 40 minutes recovery. Within 60 minutes post-exercise, VO₂ did not demonstrate any significant elevation above pre-exercise levels, although it did remain slightly elevated above pre-exercise level for an additional 25 minutes. Due to the nature of the protocol used, however, this study does not allow for easy comparison to others.

In attempting to assess the prescription for improving fitness in the general population, Brehm and Gutin (1986) investigated post-exercise metabolic rate in 8 runners who completed 4 different work rates (3.2, 6.4, 8.1, and 11.3 km · hr⁻¹) for 3.2 km, at intensities ranging from 18 to 68% VO₂max. The duration of EPOC lasted from 19 minutes for the lowest workrate (net energy cost ~4 kcal) to 48 minutes for the highest
(net energy cost ~17 kcal). A group of non-exercisers also completed the 3.2 km in 30 min, and showed no difference in EPOC from the runners. Kaminsky, Kanter, Lesmes, and Laham-Saeger (1987) reported similar findings in a study which had 14 men walk on a treadmill for 60 minutes at 35% peak VO₂ and another 10 men run for 30 minutes at 75% peak VO₂. Oxygen consumption during EPOC was not significantly different between groups and returned to resting levels within 15 minutes of recovery in both groups. Sedlock, Fissinger, and Melby (1989) examined EPOC under three varying conditions of exercise: a) high intensity, short duration (75% VO₂max, ~20 minutes); b) low intensity, short duration (50% VO₂max, ~30 minutes), and; c) low intensity, long duration (50% VO₂max, ~60 minutes). Using pre-exercise VO₂ as baseline, as opposed to a separate control condition, all conditions saw post-exercise VO₂ return to baseline within 34 minutes, and no condition produced an EPOC significantly different from another.

As mentioned previously, several factors may contribute to the discrepancies. The variation in modes, intensities, and durations used, ranging from 18 - 100% VO₂max, and from 15 minutes to four hours duration, contribute to inconsistency between studies. Not all studies controlled for food intake or compared resting postprandial to post-exercise postprandial, and some studies utilized a separate day control measure for baseline, introducing day-to-day variation, whereas others relied on a pre-exercise metabolic rate as baseline. Differences in the statistical tests used to determine the return of post-exercise VO₂ to baseline may result in studies which required VO₂ to return to absolute baseline to last longer than those stating that VO₂ was "not significantly different" from baseline (Poehlman et al., 1991). Based on a generalized evaluation of the literature, it would seem that "exercise of low to moderate intensity (< 50 to 75% VO₂max) does not produce a prolonged effect unless the duration of the exercise bout is long (80 - 180 min), and feeding occurs" (Poehlman, p.89). The relative importance of each of these individual components is much debated, but beyond the scope of this review to examine in detail. Those studies for which both intensity and duration are manipulated as independent variables seem to be the strongest in revealing the effect of exercise on EPOC. For a more in depth analysis of the impact of individual components on EPOC, one is referred to a review on the subject by Trost (1994).
Summary

It has been established within the course of this review that there remains great difficulty in establishing strong theories regarding both the impact of caloric restriction and exercise training on resting metabolic rate and the existence of a prolonged elevated post-exercise oxygen consumption. Utilization of a wide variety of investigator protocols, involving differences in mode, frequency, intensity and duration of exercise, the nutritional status of the individual, the composition of the diet, and the measurement techniques, exacerbate problems in consistency across studies. The comparison of findings from heterogeneous groups add yet another source of variation.

In analyzing the broad spectrum of studies, there does seem to be support for an interaction between the intensity and duration of chronic exercise to affect an increase in resting metabolism. That same interaction of intensity and duration, when combined with chronic and severe caloric restriction, seems to generate a decrease in resting metabolism. It is fair to recognize there must be a level at which the body can no longer sustain normal daily functioning without compromising its metabolic demands when fuel is in short supply. When additional demands for exertion are placed upon a system that is already compromised, it becomes difficult to expect the system to become, in effect, "less efficient" by increasing its metabolic needs. Therefore, based on this assumption, it is hypothesized in this study that moderately exercising, calorie restricted females will display a reduced RMR and EPOC when compared to moderately exercising females who consume a normal diet.

As previously mentioned, one additional consideration must be given to females who engage in vigorous exercise and restrict caloric intake, and that is the influence of the menstrual cycle. Numerous questions have been raised as to what metabolic changes the body might undergo as a result of monthly hormonal fluctuations. Does the flux of estrogen and progesterone have an additional impact on resting or exercise metabolism, such that menstrual status, cycle phase, or the use of oral contraceptives need to be considered when evaluating the role of exercise and/or diet in females? These questions provide the last topic for discussion. The review of literature which follows is presented as support material, and is not integral to establishing a foundation for which the research hypotheses are based.
THE ROLE OF THE MENSTRUAL CYCLE AND USE OF ORAL CONTRACEPTIVES ON EXERCISE PERFORMANCE

The normal menstrual cycle lasts approximately 28 days and is divided into four phases. The menstrual phase (days 0 - 4) is marked by the onset of bleeding (day 0), which signals the end of a cycle and the beginning of a new one (day 1). The follicular phase (~days 5 - 12) constitutes the development of mature follicles and is influenced by rising levels of the gonadotrophins lutenizing hormone (LH) and follicle stimulating hormone (FSH). FSH is responsible for the growth and development of the follicles in the ovary, while LH regulates estrogen production and secretion, ovulation, and the thickening of the endometrial lining (Wells, 1991). These changes are in response to increasing levels of estrogen and low levels of progesterone. Ovulation, preceded by a surge in estradiol, follows (~days 12 - 15), in turn followed by the luteal phase (~days 14 - 28). The luteal phase is influenced primarily by progesterone levels, although estrogens are still secreted, and continues until onset of bleeding (Mishell, 1982).

The roles of the specific hormones are varied and broad. Estrogens, primarily in the form of estradiol, estrone and estriol, promote sex-specific fat distribution in the breast, buttocks, and thighs. They may provide a beneficial decrease in both total cholesterol and low-density lipoproteins (LDL) and increase high-density lipoproteins (HDL), acting to protect against atherosclerosis. Of the more negative effects are changes in plasma fibrinolytic activity and platelet aggregation, leading to increased thrombosis. Edema and weight gain during periods of increased estrogen levels are also reported. Progesterone acts in many ways as an anti-estrogenic, inhibiting and decreasing synthesis of estrogen receptors. It may precipitate changes in body composition, cardiorespiratory function, hemodynamics, and thermoregulation, and has been implicated in the hyperventilation seen in pregnancy, as well as in the luteal phase of the menstrual cycle (Lebrun, 1994).

Some question has been raised as to whether hormonal fluctuations occurring during normal menstruation affect exercise performance. Since estrogen and progesterone, the primary hormones regulating menstruation, can each separately and in-tandem influence various metabolic processes, any one or combination of changes may result in altered physiologic functioning, and thereby potentially affect athletic performance (Lebrun, 1994). The majority of studies conducted prior to the early 1980's which investigated the role of menstrual cycle phase on exercise performance did not use plasma levels of ovarian hormones to confirm menstrual cycle phase, but relied instead
on basal body temperature and calendar day timing. Additionally, the variations between research studies regarding exercise intensity and duration, training status of subjects, sample size, timing of test procedures, and confirmation of menstrual phase, make comparison of results difficult. In the following discussion, all studies presented used two or more methods of cycle phase documentation, including urine analysis of LH or pregnandiol levels, serum progesterone levels, basal body temperature, and salivary hormone level analysis.

Core body temperature and basal metabolic rate in the menstrual cycle

The rise of progesterone seen during the luteal phase typically produces a subtle increase in core body temperature and basal metabolic rate (Kleitman & Ramsaroop, 1948). Pivarnek, Marichal, Spillman, and Morrow (1992) studied nine endurance trained females to determine whether phase of menstrual cycle affected temperature regulation during steady state exercise. Slight, but parallel, elevations in body temperature noted during luteal phase prior to and in early exercise became exaggerated at approximately 40 minutes of exercise when compared to follicular phase. Temperature during follicular phase exercise plateaued in 30 minutes, whereas during luteal phase it continued to rise and did not indicate equilibrium. One subject indicated LH surge at ovulation with no subsequent increase in progesterone and did not demonstrate the continuing rise in temperature in luteal phase. The authors therefore concluded that thermoregulatory differences during the luteal phase may likely be attributed to rising progesterone levels.

With the increase in core body temperature seen in the luteal phase, a concomitant rise in basal metabolic rate has been observed (Bisdee, James, & Shaw, 1989; Kleitman & Ramsaroop, 1948; Solomon, Kurzer & Calloway, 1982; Webb, 1986). Although intra-individual variability exist in both genders, in part due to energy intake and expenditure levels, it has been suggested that women have cyclic variations coinciding with menstrual cycle phase as well (Solomon et al., 1982). Solomon et al. (1982) investigated six normally menstruating females in a metabolic ward for 92 days during which exercise and diet were controlled for. Mean BMR for the women was $20.7 \pm 2.6 \text{kcal} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$. BMR declined to its lowest point approximately one week prior to ovulation in five of six subjects, and was followed by a rise preceding the next menses. There was an average difference of $.25 \text{kcal} \cdot \text{min}^{-1} (~360 \text{kcal/day})$ from before menses to post ovulation.
Webb (1986) conducted repeated 24-hour calorimetric measurements in ten normally menstruating women. One subject was taking oral contraceptives, but stopped use during one series of tests to investigate whether changes occurred as a result of discontinued use. For eight of the women, there was an increase in daily energy expenditure following ovulation ranging from 8 - 16%. In the subject using the oral contraceptive, there was a consistent level of energy expenditure for all four weeks of her cycle while on medication. When medication stopped, she showed a 14% increase in energy expenditure in the two weeks post-ovulation, consistent with the other subjects. It was concluded that the post-ovulatory increase in progesterone was the likely mechanism behind the elevated BMR.

Another study used women confined to a metabolic ward for 36 hours of whole body calorimetry during four phases of a single menstrual cycle under a variety of resting and exercise conditions (Bisdee et al., 1986). Total energy expenditure varied from 5.6 - 7.5% between subjects at different phases of the cycle, and when expressed as kg·FFM⁻¹, still indicated small differences in metabolic activity between phases. The greatest intra-individual difference occurred in the late luteal phase (3.8 - 7.8% total energy expenditure). The average 24-hour energy expenditure showed a consistent pattern of change throughout the cycle, with a decrease in late follicular and a rise to maximum values in the late luteal phase. A 2.5% overall difference between phases suggested to the authors that while the changes were small, they were likely dependent on larger alterations in RMR, rather than dietary or exercise induced change, and implied a complex control system regulating energy balance. While individual differences may vary, the bulk of evidence with adequate hormonal documentation seems to indicate small to moderate increases in basal metabolism during the luteal phase of the menstrual cycle.

Substrate metabolism during exercise

To clarify whether substrate metabolism is altered in exercise as a result of menstrual cycle phase, it is perhaps best to briefly review some of the literature on substrate utilization in general, and any observed differences between genders. It is well understood that the body is dependent on utilizing oxygen in the breakdown of ingested and stored fats, carbohydrates, and proteins for energy metabolism. Due to differences in the chemical composition of these fuels, the amount of oxygen required to completely oxidize each differs, resulting in different proportions of CO₂ produced to O₂ consumed,
and which is known as the Respiratory Exchange Ratio (RER or R). For activities ranging from rest to mild exercise, the R value reflects the metabolism of a combination of fat, protein, and carbohydrate (R = ~.80). As exercise intensity increases, there is a greater proportion of CO₂ expired in relation to O₂ consumed. This results in an increased R value (R value approaches or exceeds 1), and is reflective of a greater reliance on stored carbohydrate for metabolism. If the intensity of exercise does not increase, but is maintained for a prolonged period of time, or in the period following strenuous anaerobic exercise, the R value begins to shift downward below .8, reflecting a reduction in expired CO₂ and a greater reliance on stored fat for metabolism (McArdle, Katch and Katch, 1991). There is argument as to what mechanisms are ultimately responsible for driving the exercise-induced shifts in metabolism, ranging from, but not limited to enhanced stimulation of carbohydrate metabolism and/or entrapment of free fatty acids (FFA) within adipocytes during high-intensity exercise, inhibition of lipolysis, enhanced muscular uptake of free fatty acids (FFA) and triglycerides, and/or reesterification of FFA during lower intensity exercise (Hurley et al., 1986; Romijn et al., 1993).

A recent study attempted to determine the effects of exercise intensity and duration on FFA mobilization and utilization (Romijn et al., 1993). Five trained cyclists cycled for two hours at intensities of 25% and 65% VO₂max, and for 30 minutes at 85% VO₂max. Subjects were infused with isotopes and blood samples were collected at various periods during exercise and recovery. The authors found an inverse relationship between exercise intensity and FFA rate of appearance (Ra) after 30 minutes of exercise. Fat oxidation was not different between 25% and 85% VO₂max at 30 minutes, in spite of an increased intensity equivalent to three times as great. Sixty-five percent VO₂max elicited maximal rates of FFA oxidation, and both peripheral adipocytes and muscle triglyceride (TG) lipolysis contributed equally to fat oxidation. At 65% and 85% VO₂max, whole body oxidation of FFA was significantly higher than FFA tissue uptake, with the difference indicating a limited contribution of muscle TG stores. The authors assumed that glucose and FFA are oxidized by tissue and have no other function, and so determined that the maximal rate of FFA oxidation from peripheral lipolysis should be considered equal to plasma FFA uptake. If the primary mechanism regulating the decreased Ra of plasma FFA at 85% VO₂max were inhibition of whole body or peripheral lipolysis, it would be reflected by a concomitant decrease in plasma glycerol levels, since glycerol's appearance in the blood is a reliable indicator of lipolysis within adipocytes and exercising muscles. In fact, after 30 minutes of exercise, glycerol Ra was significantly higher at 65 and 85% VO₂max, while during recovery, glycerol Ra immediately decreased.
These observations suggested to authors that the shifting increase in FFA post-exercise was not due to an increase in lipolysis, a finding supported by Hurley et al. (1986). At 65% and 85% VO₂max, the rate of glucose uptake at the muscle was exceeded by values of total carbohydrate oxidation. The difference, again, seemed to signify a minimal contribution of muscle glycogen to carbohydrate oxidation. At 25% VO₂max, it was determined that carbohydrate oxidation was met solely by blood glucose uptake. An important finding, according to Romijn et al. (1993), indicated that lipolysis in peripheral adipose tissue is stimulated maximally by low intensity exercise (25%) and does not increase with an increase in exercise intensity. Lipolysis of intramuscular triglyceride is stimulated at higher intensities (65%), yet does not continue to rise with a continued increase in exercise intensity (85%). A physiological "steady state" seems to exist at 25% VO₂max, whereas at 65%, progressive increases in plasma FFA and glucose availability occur. The authors surmised that while the decreased availability of plasma FFA might contribute to part of the decline in fat oxidation with increasing intensity, it is offset by the gradual increase in blood glucose turnover. An issue not addressed in this study is the potential effects gender and training status may have on substrate metabolism and the responses observed in this study.

Hurley et al. (1986) placed non-endurance trained subjects on a twelve-week strenuous exercise program, and compared pre- and post-training weight, VO₂max, and peak cycling VO₂. Subjects demonstrated significant increases in VO₂max and peak cycle VO₂, decreased HR and lactate response, and showed a 90% increase in mitochondrial enzymes in quadriceps muscles from pre- to post-training biopsies. FFA and glycerol concentrations were significantly lower toward the end of prolonged exercise (90 minutes at 75% peak VO₂), and R values were significantly lower at all time points in the trained state. A significantly smaller proportion of energy was utilized from carbohydrate oxidation and proportionally greater amount was derived from fat during exercise. Muscle biopsies indicated that 41% less muscle glycogen was utilized in the trained state than pre-trained, due to a more than two-fold increase in muscle glycogen concentration in the quadriceps following prolonged exercise. The decrease in muscle TG concentration was approximately two times greater post-exercise.

A lower plasma FFA concentration during submaximal exercise in the trained state suggests a reduced rate of plasma FFA oxidation. An increase of greater than 60% in the amount of energy derived from fat implies a substantial increase in the proportion of calories utilized from fat (from 35% to 57%) post-training. The findings suggested that the increased utilization of fatty acids must be obtained from sources other than plasma FFA - specifically, muscle triglyceride. Since Romijn et al. (1993) tested subjects
at 65% and 85%, it becomes difficult to tell if the Hurley et al. study's workload (75% peak VO$_2$) would be consistent with the former's conclusion that muscle TG stores contribute minimally to exercise intensity above 65%. The 90% increase in mitochondrial enzymes of the quadriceps would suggest an enormous increase the muscle's capacity for FFA oxidation. Evidence seems to indicate that training induces adaptations in substrate oxidation, primarily a greater reliance on FFA utilization during prolonged and low to moderate intensity exercise. The exact mechanisms responsible for initiating these changes are still undetermined.

**Gender differences in substrate metabolism during exercise**

Do the alterations in substrate metabolism as a result of training apply equally to males and females, or do cyclic hormonal changes in females cause additional differences in this training response? Bunt (1990) demonstrated that estradiol decreases the rate of gluconeogenesis and glycogenesis, thereby increasing lipid availability and utilization. Conflicting reports regarding levels of fat oxidation during prolonged exercise in males and females are likely due to the effects of training, nutritional, and/or hormonal status in subjects. Tarnopolsky, MacDougall, Atkinson, Tarnopolsky, and Sutton (1990) attempted to control for these confounding variables by matching training levels and experience of all subjects, controlling nutritional intake, and testing females only during the mid-follicular phase of their menstrual cycle. During exercise of 65% VO$_2$max, the females consistently maintained a significantly lower R value, and this value remained fairly constant for both groups for the duration of exercise (~ 90 - 101 minutes). Resting and initial exercise plasma FFA and muscle glycogen concentration did not differ between groups, nor did FFA concentration change significantly during exercise over resting values. Females utilized a significantly greater amount of fat (47.6 ± 2.1g vs 26.9 ± 1.2g) and a reduced amount of carbohydrate (137.3 ± 6.0g vs 239.7 ± 10.3g) than males during exercise. Calorically, when expressed as g · kg$^{-1}$ lean body weight, expenditure did not differ significantly. Following exercise, males demonstrated a 25% greater glycogen depletion than females. They also demonstrated an increase in growth hormone levels throughout exercise, as well as a greater concentration at 60 and 90 minutes of exercise and 15 minutes post-exercise. Females exhibited a significant increase in plasma glucose concentration during exercise and the males showed no change. The females maintained this greater level throughout exercise. Lactate levels showed no difference between genders.
While the females' lowered R value during exercise is a strong indicator of greater lipid oxidation, the lack of a concomitant increase in their glycerol levels creates a discrepancy which has previously been indicated by Hurley et al. (1986) and Romijn et al. (1993). Two possibilities have been proposed to address this finding: 1) trained females have a greater FFA uptake than equally trained males; or 2) females have greater intramuscular TG utilization than their male counterpart. This second possibility is supported by the similar findings of Hurley et al. It is also feasible that trained females have greater intramuscular TG concentrations than males. Although this question was not investigated in the study, it certainly would lend greater understanding to the changes observed.

The greater sparing of muscle glycogen observed in the female subjects might also lend support to the theory of their greater fat utilization. The finding of similar resting glycogen concentrations between trained males and females has been corroborated, but Jansson (1986, cited in Tarnopolsky et al., 1990) also found that exercise at 65% VO2max for 25 minutes did not yield any differences between the genders. Since it has been proposed that trained subjects have a decreased rate of carbohydrate oxidation at submaximal levels of exercise, it is probable the duration utilized in Jansson's research was not adequate to stimulate any gender differences in the trained subjects. Tarnopolsky et al. (1990) do not give much insight on protein utilization, but suggest that increased urinary nitrogen excretion in the males, and no change in the females might indicate a greater protein catabolism during or post-exercise in males, whereas women have little or none. Females maintained plasma glucose levels better than males during exercise. Whether this could be related to menstrual cycle phase, and demonstrates a possible suppression of gluconeogenesis by ovarian hormones, has not received adequate inquiry. The authors concluded that during moderate intensity (65%), prolonged aerobic exercise, females derived a greater proportion of energy from fat and less from carbohydrate and protein oxidation than do males of similar training and nutritional status. While this study indicated some clear differences between males and equally trained females during the follicular phase of the menstrual cycle, it needs to be determined if hormonal changes occurring as females move through the menstrual cycle factor into substrate metabolism.
Substrate metabolism and menstrual cycle phase

The increase in core body temperature during the luteal phase, along with its associated rise in progesterone levels, has raised questions regarding changes in substrate utilization across the phases of the menstrual cycle. An investigation by Kanaley, Boileau, Misner, and Nelson (1992) cites a handful of research that indicates a) changes in estradiol (E2) and progesterone may influence metabolic hormones; b) basal androgen concentrations may be linked with growth hormone secretion, and c) females may have an elevation in growth hormone associated with their elevated estrogen levels during both rest and exercise. Other studies indicate no such metabolic changes occurring. Again, variations in exercise and testing protocols may preclude comparison of findings.

In the luteal phase, when levels of both estrogen and progesterone are high, it is postulated that metabolism is most efficient (Lebrun, 1993). Both Hackney (1990) and Nicklas, Hackney, and Sharp (1989) found that glucose uptake and storage was enhanced as estrogen levels rose. The presence of estradiol seems to initiate a shift towards FFA metabolism by increasing lipid synthesis and lipolysis in muscle and adipose tissue (Bonen, Hayes, Watson-Wright, Sopper & Pierce, 1983; Lebrun 1993). Additional investigation by Jurkowski, Jones, Toews, and Sutton (1981) concurs with this finding. However, Tarnopolsky et al. (1990) demonstrated greater fat and reduced carbohydrate and protein metabolism in trained women during mid-follicular phase when compared to equally trained males.

A study by Nicklas et al. (1989) required six normally menstruating, moderately trained females to cycle at 70% VO2max until exhaustion, during both mid-follicular and mid-luteal phases of their menstrual cycle. Muscle biopsies revealed no changes in glycogen content, nor were changes seen in blood glucose or FFA concentration between menstrual phases. FFA concentration from 75 min to exhaustion in luteal phase was greater when compared to follicular phase, and approached significance. The R value was lower at rest and during all exercise in the luteal phase, but was not significantly different. The authors believed that, although their data did not indicate significance, there may be physiological and performance responses to submaximal exercise as a result of cycle phase, based on their observed and almost significant findings.

Kanaley et al. (1992) had 6 amenorrheic and 7 eumenorrheic female athletes perform exercise at 60% VO2max for 90 minutes during both early and late follicular and mid-luteal phase. Menstrual phase did not significantly decrease resting glucose levels, nor was a difference found in substrate metabolism between the two groups, despite a lower steroid hormone concentration in the amenorrheic females. No change was
demonstrated in resting levels of serum growth hormone, which have been found to be increased by high levels of estrogen and which stimulate fat mobilization (Lebrun, 1993). It was concluded that fat and carbohydrate utilization were independent of menstrual status and phase. It was noted, however, that the effect of endurance training in these subjects may have overridden any potential differences between the two groups due to menstrual phase and status. Similar findings by Bonen et al. (1986), De Souza, Maguire, Rubin, and Maresh (1990), and Nicklas et al. (1989) all lend support to this possibility.

Nutritional status of subjects has not been carefully controlled for in the majority of studies on substrate metabolism and menstrual cycle phase. Bonen et al. (1983) conducted a series of treadmill tests under varying conditions of fasted, glucose-loaded and control conditions on 19 females during both follicular and luteal phase. Results were varied and diverse, and served primarily to emphasize the need for careful control of nutritional status in any study examining substrate metabolism and menstrual cycle phase.

In summary, research indicates that trained females exhibit some differences in substrate metabolism during exercise when compared to equally trained males, primarily greater lipid oxidation. While resting glycogen levels have not been found to be different, males have demonstrated greater glycogen depletion post-exercise, with exercise held equal between groups. Females also appear to have a greater increase in plasma glucose, which is not exhibited by the males during exercise, and this increase remains elevated throughout the course of exercise. Menstrual cycle phase may exert subtle influences on substrate metabolism, most notably increased glucose uptake and storage and increased FFA utilization during the luteal phase. In endurance trained females, however, many of these subtle differences are no longer readily apparent, suggesting that any phase-related changes that may exist are likely overridden by the effect of endurance training.

Menstrual cycle phase and performance

Numerous studies have investigated a variety of exercise parameters in relation to menstrual cycle phase. A cursory review of early studies not using any hormonal documentation of cycle phase indicate a strong tendency toward no observable changes in oxygen consumption between phases (Allsen, Parsons, & Bryce, 1977; Doolittle & Engebresten, 1972; Easton & Brooke, 1984; Stephenson, Kolka, & Wilkerson, 1982; Wells & Horvath, 1974), but equivocal findings on performance time, heart rate,
ventilation, and perceived exertion (Easton & Brooke, 1984; Stephenson et al., 1982; Wells & Horvath, 1974). Probably more important are the studies using hormonal documentation, which also give similar findings -- a strong indication toward no change in oxygen consumption due to menstrual cycle phase during exercise (De Souza et al., 1990; Dombovy, Bonekat, Williams, & Staats, 1987; Jurkowski et al., 1981; Nicklas et al, 1989; Pivarnek et al., 1992; Schoene, Robertson, Pierson, & Peterson, 1981), and more controversial findings on exercise parameters of performance time, heart rate, ventilation, and perceived exertion (De Souza et al., 1990; Jurkowsky et al., 1981; Lamont, 1986; Nicklas et al., 1989; Pivarnek et al., 1992; Schoene et al., 1981).

Lebrun (1994) attributes the likelihood of any changes in maximal performance to be due to the more subjective sensations of dyspnea than any real physiologic change. Schoene et al. (1981) compared exercise performance and respiration in eumenorrheic and amenorrheic athletes, and normally menstruating, non-athletic controls. Maximal VO2 values showed no significant changes in either group of athletes between phases, but a significant increase in maximal performance during the luteal phase of the control subjects. The athletes did not demonstrate any change in exercise performance between phases. This finding of no change in the trained groups follows the theory of increased tolerance, and perhaps motivation, of the endurance trained individuals.

De Souza et al. (1990) investigated the effects of menstrual status and phase on submaximal (80%) and maximal exercise parameters in eight normally menstruating and eight amenorrheic runners. No changes in VO2 occurred between phases or subjects at either sub- or maximal levels. Pivarnek et al. (1992) also found that in endurance trained females, VO2max values between phases were not different. Jurkowski et al. (1981) examined normally menstruating females in follicular and luteal phases of their menstrual cycle, at 33%, 66%, and 90% of their maximal power output. No difference was observed in oxygen uptake between the phases, although increased time to exhaustion was noted during the luteal phase.

Two studies finding change in VO2 values indicate conflicting findings. Lebrun (1993) cites a 1982 study by Horvath and Drinkwater, who found an increase in VO2 during luteal phase at rest in four females. This difference disappeared under the effects of exercise and heat stress. While the small sample size severely limited the statistical power, the authors surmised that the demands of exercise and heat most likely masked any small changes that might have been seen. Lebrun, McKenzie, Prior, and Taunton (1995) recently published a study in which 16 well-trained athletes tested on a variety of performance parameters showed a slight decrease in luteal phase in both absolute and relative VO2max, with no associated decrease in exercise performance.
Only a few studies have utilized precise hormonal documentation of menstrual cycle phase in conjunction with ventilatory changes and exercise. It has been fairly well established that progesterone is responsible for the hyperventilation seen in both pregnancy and at rest in the luteal phase of the menstrual cycle (Dombovy et al., 1987; Schoene et al., 1981). A study comparing respiratory drives and exercise performance in eumenorrheic and amenorrheic athletes and eumenorrheic non-athletic females found that all menstruating subjects demonstrated significantly higher resting minute ventilation (VE) in the luteal phase, but when compared as athletes vs. controls, no significant difference was noted (Schoene et al., 1981). Ventilatory equivalent (VE/VO2) was significantly higher during progressive exercise in the luteal phase of eumenorrheic subjects. There was no correlation, however, in the levels of increase in plasma progesterone and the degree of change in luteal and follicular phase respiration. Because trained subjects did not demonstrate any performance hindrance, while VO2max decreased in non-athletes during luteal phase exercise, it might be speculated that despite ventilatory differences between menstrual phases, endurance training may override these differences.

It is apparent that considerable controversy exists regarding the influence of ovarian hormones on a variety of exercise parameters. The need for rigorous control over documentation of cycle phase, timing of test procedures, nutritional and training status of subjects is highlighted by the equivocal findings in specific exercise responses. Keeping in mind the lack of confirming evidence regarding menstrual cycle phase and exercise performance, the role of oral contraceptives in altering normal hormonal responses to exercise will now be reviewed.

**Oral Contraceptives**

The use of oral contraceptives (OC) and subsequent potential impact on exercise performance has been the subject of inquiry because of their alteration of the natural cascade of menstrual events. While primarily used as a method of birth control, they are also commonly prescribed for symptoms of dysmenorrhea and premenstrual syndrome, cycle irregularity, hormone replacement for bone preservation in amenorrheic women, and endometrial fibrosis (Mishell, 1982). It has also been noted that some athletes and their coaches have used OC for the sole purpose of manipulating the menstrual cycle around important athletic events. Oral contraceptives exert influence over the menstrual cycle through a combination of estrogen and progestagen, or progesterone only. Natural
progesterone and synthetic progestins inhibit the "proliferative effect" of estrogen. The endometrial lining of the uterus contains specific receptor proteins, some of which bind estradiol and others, progesterone. If the receptor is inactivated, the hormones cannot bind, thereby inhibiting them from effecting changes in their targeted tissue. Progesterone acts to decrease synthesis of these receptors through its continued release in OCs. Additionally, it stimulates the enzyme estradiol-17 β dehydrogenase within the endometrial cell, which converts the more potent estradiol to estrone, to reduce estrogenic activity in the cell. This enzymatic activity increases during the luteal phase (Mishell, 1982). Bonen, Hayes and Graham (1991) conducted a study examining whether oral contraceptives caused any marked changes in substrate utilization during exercise over the course of normal menstruation and found no alterations. Oral contraceptive users had greater FFA concentration during mild exercise and lower glucose concentrations at rest and during mild and heavy exercise. This finding led authors to conclude that FFA metabolism is most likely greater during mild exercise in OC users, but at higher intensities, carbohydrate metabolism is not altered or impaired in this group.

In terms of what effect OCs have on exercise performance, there are no definitive data. Early studies are based on OC prescriptions which contained much higher doses of hormones than those currently prescribed, had a wide range of levels of estrogen and progesterone components, and involved subjects whose fitness levels varied considerably. Presently, OC dosage ranges from combinations of .50 mg mestranol + 1 mg norethindone, .35 mg ethinyl estradiol + 50, .75, and 1.0. mg norethindone, to .30 mg ethinyl estradiol + .3 mg norgestrel. A normal prescription would require the user to take a pill each of 21 days, followed by seven days of no use, or use of a inert formula.

**Studies demonstrating a change in exercise performance with oral contraceptive use.** Three studies give indications that OC use may alter ventilatory responses or oxygen consumption. Dagget, Davies and Boobis (1983) used maximal bicycle exercise to determine VO2 changes in seven females before, during, and after administration of oral contraceptives. At intervals of one and two months during use, significant decreases were observed in maximal VO2, but not at rest. Additionally, a significant reduction in mitochondrial citrate was reported during OC use. The authors concluded there was a "peripheral and metabolic effect on subjects taking oral contraceptives, with a return to normal values after cessation" (abstract). Interestingly, this study is cited elsewhere in the literature, but never in its manuscript form. Questions regarding methodology, statistical analysis, and exercise protocol might give insight to the findings, but remain unanswered and leave room for speculation.
Pearl (1993) cites an unpublished study by Lebrun (1991) which examined one of the newer low dose triphasic OC's effects on various performance parameters in seven elite female athletes. This study found a slight decrease in both absolute and relative VO₂max in the OC users when compared to controls, but insubstantial information is available to adequately assess this research, and Lebrun herself later suggests that, apart from minor changes seen in some variables, "for most women, there is no significant difference" in the effects of menstrual phase or administration of oral contraceptives on athletic performance (Lebrun, 1994, p.437).

Notelovitz et al. (1987) also used a low-dosage OC (Ovcon 35, containing .035 mg of ethinyl estradiol and .4 mg of norethindone) to determine its effect on cardiorespiratory fitness. Twelve women between the ages of 20 - 30 were randomly assigned into control (intrauterine device/diaphragm) or OC use group. Subjects engaged in a minimum of three hours exercise per week (runners 15 mi/wk; swimmers 6500 yd/wk). Each subject was tested twice at baseline (prior to contraceptive use) and at the end of six months for submaximal and maximal VO₂. The results indicated that OC users' maximal VO₂ decreased from a mean of 41.2 ± 11.8 to 38.4 ml · kg⁻¹ · min⁻¹ (7%), while the non-OC users' value increased from 42.6 ± 2.8 to 45.9 ± 5.9 ml · kg⁻¹ · min⁻¹. In both groups, submaximal values (3.0 mph, 5% grade) were not changed.

Additionally, the oxygen pulse, the volume of oxygen consumed per heart beat was increased in non-OC users by 9%, while OC users decreased 8%. Interestingly, there was a mean weight gain of approximately 2 kg in the OC user group by the end of the study, which was not seen in the non-user group, that may have accounted for some of the noted decrease in VO₂max. It is also interesting to note that the range of values for VO₂max in non-OC users varied from 39.8 - 45.4 ml · kg⁻¹ · min⁻¹ while range of values for OC users varied from 29.4 - 53.0 ml · kg⁻¹ · min⁻¹, indicating a much broader and varied extent of fitness levels, and which encompassed both below and above "normal" values (38 - 48 ml · kg⁻¹ · min⁻¹). Fitness levels of subjects was identified only as a minimum of three times/week, running at least 15 miles/week or swimming at least 6500 yards/week. No attempt to match subjects was made. It would seem that matching groups for fitness levels would provide more accurate assessment of changes, and eliminate exaggeration of any changes by lower fitness subjects due to anxiety regarding perceptions of exercise intensity. At submaximal values, oxygen uptake was unaffected, and the noted decrease in VO₂max did not have any effect on the subjects' normal exercise habits.

**Studies demonstrating no change in exercise performance with oral contraceptive use.** Montes, Lally, and Hale (1983) monitored changes in respiratory function during rest and exercise at three and six month intervals following the beginning
of OC use in 12 nulliparous females. No increase in VO2 was seen at either rest or exercise. There was insubstantial evidence to conclude that synthetic hormones available through OC's decreased exercise performance in untrained subjects during short duration exercise, but the implications regarding trained athletes could not be discerned.

A study investigating the effect of OC's on serum lipid profiles in women runners concluded that cardiovascular endurance, as well as lipid profiles, were not different for either control or OC groups when matched for percentage body fat and cholesterol intake (Gray, Harding, & Dale, 1983). This study did not incorporate use of a non-active control group for baseline comparison. Bonen, Hayes, and Graham (1991) hypothesized that if metabolic responses were different in normally menstruating females during follicular and luteal phases during exercise, carbohydrate and or lipid metabolism might be altered in the presence of synthetic steroids such as those found in oral contraceptives. Untrained subjects performed two bouts of treadmill exercise at 40% and 85% VO2max. The control group performed exercise during both the follicular and luteal phase, while the OC group performed during days 6 - 11 of an OC cycle and during days 3 - 5 when they were briefly abstaining from OC use. Normal menstrual phases were confirmed by progesterone and estradiol levels during the follicular phase and FSH and LH during the luteal phase, while phases in the OC group were confirmed by an increase in FSH during the non-use phase and a reduction during use. No difference in oxygen consumption was seen between groups or phases at either 40% or 85% VO2max. Additionally, FFA concentrations at rest were not different between groups, but during mild exercise FFA levels increased in the OC group. In addition, a modest change in the R value between trials in the OC group (R = .89 during use vs. .90 during non-use) suggested to authors a slightly greater FFA metabolism. This difference was not noted as significant, and seems relatively inconsequential in light of lack of nutritional control. During intense exercise, lactate levels remained similar for both groups and between phases, indicating that neither normal hormonal changes nor OC use altered carbohydrate metabolism.

Another study investigating the effects of oral contraceptives on metabolic and hormonal responses during exercise used eight OC users and eight normally menstruating females in a 90 minute treadmill test at 50% VO2max (Bemben, Boileau, Bahr, Nelson, & Misner, 1992). Menstruating subjects were tested during days 3 - 10 of the luteal phase of their cycle, and cycle phase was confirmed by serum progesterone levels. All subjects were considered moderately active, and maximal oxygen consumption was similar for both groups (44 ± 1.9 and 42.8 ± 1.3 ml · kg⁻¹ · min⁻¹, control and OC groups, respectively). Neither total energy expenditure nor total fat utilization was different between groups, although significant differences were seen in total grams · kg⁻¹ of
carbohydrate used (.82 ± .07 vs .63 ± .05 g · kg⁻¹, controls and OC users, respectively). No indication of changes in protein metabolism were noted to account for this difference. The results suggested to the authors that OC use affected carbohydrate metabolism while FFA utilization was not affected, in contrast with the earlier findings of Montes et al. (1991). The authors concluded that OC steroids produce significant changes in endocrine and metabolic responses that alter substrate utilization, especially during prolonged exercise.

In assessing the role of menstrual cycle and oral contraceptive use, Pearl (1993) acknowledges that only a handful of studies have been adequately controlled, and that competitive female athletes have won events in every phase of the menstrual cycle. Several reasons can be noted regarding why research on the effects of oral contraceptives has been so inconclusive; a) the lack of large, long-term studies; b) poor documentation and standardization of menstrual cycle phases; c) out-dated techniques for hormonal documentation d) difficulty in obtaining accurate hormonal measures in controls; e) the lack of trained female athletes in treatment groups; f) the use of a wide variety of OC types and dosages, and g) the use of a wide variety of testing protocols (Lebrun, 1994; Pearl, 1993). It was previously concluded that endurance training may have masked or eliminated any differences in substrate metabolism due to hormonal fluctuations in the menstrual cycle. It seems quite probable that, in the case of oral contraceptives, minor differences which exist beyond predicted intra-individual differences, may also be overridden or eliminated by training. It seems apparent that a large majority of evidence does not support any significant changes resulting from oral contraceptive use.
Summary

In conclusion, the research findings on the effects of menstrual cycle phase, status, and oral contraceptive use on exercise performance, reveal strongly indicative, but not unequivocal, results. The primary hormones regulating menstruation, estrogen and progesterone, are known to exert influence on various metabolic processes. Core body temperature and basal metabolic rate undergo slight increases during the luteal phase of the cycle, as a result of increasing progesterone levels. Studies examining differences in exercise performance due to cycle phase indicate no observable changes in submaximal or maximal endurance exercise, and those studies comparing eumenorrheic and amenorrheic females also indicate no differences in resting or performance comparisons. The use of oral contraceptives has not been shown to alter resting or exercise metabolism, or exercise performance. It appears very likely that in endurance-trained or active females, any differences which may exist due to cycle phase, status or OC use are rendered negligible or non-existent. While these factors have received strong support as non-influencing variables in resting and exercise states, rigorous control and accurate documentation will yield more far-reaching and precise results.
CHAPTER 3

METHODS AND PROCEDURES

The purpose of this chapter is to define the process of subject selection, the instruments and equipment utilized, the steps which led to the collection of data, and the means by which data were analyzed. This proposal was submitted to and reviewed by the Oregon State University Institutional Review Board for the Protection of Human Subjects prior to any contact with subjects or data collection. All subjects completed an Informed Consent before entering into the study (Appendix A).

SUBJECTS

Subjects (all female) aged 18 - 25 were selected through response to flyers and The Diet/Exercise Assessment (DEA) (Appendix B) distributed on the campus of Oregon State University and in surrounding communities within a 45-mile radius. To achieve a statistical power of .80 with alpha of each dependent measures set at .025, 9 subjects per group would be required to detect a moderate effect between means. Due to tightly defined criteria established for eligibility into the study and limited subject availability and follow-through, a total of 15 subjects completed the study - ten in the NC group and five in the LC group. Subjects met the following criteria:

**Low Calorie** - dietary intake at \( \leq 1200 \text{ kcal} \cdot \text{day}^{-1} \) and aerobic exercise at approximately 50% or greater of \( \text{VO}_2\max \) for a minimum of four hours per week for at least the previous nine months.

**Normal Calorie** - dietary intake at \( \geq 1700 \text{ kcal} \cdot \text{day}^{-1} \) and aerobic exercise at approximately 50% or greater of \( \text{VO}_2\max \) for a minimum of four hours per week for at least the previous nine months.

To further define the subject population, those subjects meeting initial requirements completed a Medical Questionnaire, the Eating Attitudes Test (EAT- 26) (Garner, Olmstead, Bohr & Garfinkle, 1982), The Bulimia Test (BULIT) (Smith & Thelan, 1984), and two 24-hour diet recalls (Appendices C, D, E, and F, respectively). Subjects attaining a score of \( \geq 20 \) on the EAT- 26 and \( \geq 102 \) on the BULIT were eliminated from
the study due to potential confounding factors associated with involvement in eating disorders.

All subjects fell within the normal or below normal range of Body Mass Index (20 - 26 BMI) as developed by the National Center for Health Statistics, and had no more than 25% ± 3% body fat as determined through hydrostatic weighing. The age of the subjects was between 19 - 25 years and the subjects selected into the study following completion of the DEA completed an Informed Consent form.

INSTRUMENTS

**EAT and BULIT.** Initial selection of subjects was determined through responses gained on the Diet/Exercise Assessment (DEA). The Eating Attitudes Test (EAT-26) (Garner et al., 1982) has been utilized as a 26-item, objective self-report measure of the symptoms of anorexia nervosa (reliability .90; validity .89 (P < .001)). The need to distinguish between individuals with bulimia and those with other eating disorders, as well as to distinguish between bulimics and those without eating disorders, has required development of a specific instrument. The cut-off score for identifying individuals at risk for anorexia nervosa has been set at 20 or above.

The BULIT (Smith & Thelen, 1984) has been correlated to other eating inventories (EAT, EDI, and the Binge Scale) and found to have correlations of .65 - .68 to measures of anorexia and .93 correlation to binge eating measures. Test-retest reliability of BULIT has been found to be .87, with a validity of .64. The lower predictive ability of BULIT in non-clinical settings may be an underestimation of the scale's actual measurement ability due to the initial test's "over representation of subjects with scores close to the designated cutoff score" who are presumed more difficult to identify than subjects with extreme scores (Smith & Thelen, 1984, p. 867). The cut-off score for identifying individuals at risk for bulimia has been set at 102 or greater.

**Food frequency and 24-hour recall.** The Health Habits and History Questionnaire (HHHQ), which is a food frequency questionnaire provided by the National Cancer Institute (Block, 1982) and two 24-hour diet recall were to be utilized to verify DEA statements and caloric intake. Due to a program flaw, the HHHQ was not utilized. While a variety of diet assessment measures are available, no single best method exists. Two important dimensions must be considered which affect dietary investigation, a) whether assessment is needed to determine an individual or groups' health/dietary status, and b) whether quantitative precision or classification on a continuum of high,
medium or low intake of nutrients is required. It has been noted that precise accuracy at the individual level is not needed to provide valid and representative information of diet and assessment (Block, 1982; Lee & Nieman, 1993).

Lee and Nieman (1993) cite several strengths and weaknesses of the food frequency questionnaire and 24-hour recall. Strengths of the frequency questionnaire include its ability to be self-administered and computer analyzed, its relative low cost to administer, and more reliable representation of usual intake over diet records of a few days. Weaknesses include its dependence on the ability of the participant to assess contents of food items, possible misrepresentation of usual foods or portion sizes, and the potential for intake data to be compromised when multiple foods are listed under a single heading. Strengths of the 24-hour recall include its low cost to administer, minimal effort and time required by participants to complete, and a detailed source of information on specific foods. Twenty-four hour recalls are considered by many researchers in the field to be more objective than food histories. Limitations include a tendency of participants to withhold or alter information, under-report binge eating or alcohol consumption, overestimate intake if the participant is aware it is low, and being an inadequate estimate if only a single day's report is used.

Factors affecting the validity of the all measures of dietary intake are differences between the usual diet and that measured, assessment method limitations and weaknesses, under- or over-reporting due to embarrassment or perceptions of investigator expectation, and observer characteristics. A one-year study designed to determine the number of days needed to obtain representative food intake records for groups of individuals cites three days as minimally required. This should result in a precision of ± 10% and confidence of 95% (Metz & Kelsey, 1984, cited in Lee & Nieman, 1993). In anticipation of using the HHHQ, two 24-hour recalls were considered adequate for the study.

APPARATUS

The Health Habits and History Questionnaire, would have been analyzed by HHHQ-DIETSYS. Analysis software, Version 3.5, National Cancer Institute (1994), but the program contained a fatal error which prohibited it from running. The 24-hour recall was analyzed on the Macintosh software program Food Processor II (1990) by ESHA Research, primarily for caloric intake, but also to assess average carbohydrate, protein and fat content.
Resting Metabolic Rate, $\text{VO}_{2\text{max}}$, submaximal workload, and EPOC were measured using a *Sensormedics 2900* metabolic cart, with subjects breathing through a Rudolph 2-way face mask model #7920. The treadmill used was a motorized *Sensormedics MAX-1*. Heart rate was continuously monitored via a Pace Heart Rate Monitor. A 3-L syringe was used to calibrate the flow meter preceding initial testing, while the gas analyzers were calibrated with gases of known concentration prior to each test.

Blood pressure measurement was taken with a *Prestige* sphygmomanometer.

The LFT 3000 *Go-Mi Inc.* Pulmonary Function Testing apparatus was utilized in determining lung volumes, including residual lung volume (RV). Residual volume was used to correct for body volume as determined through hydrostatic weighing.

Hydrostatic weighing is considered a highly reliable and valid technique for the assessment of body composition (Heyward, 1991). A Masstron Scale with incorporated load cell, read on a Toledo display was used. Analysis of body composition via hydrostatic weighing was carried out by *Body Composition Analysis 2.0* from Digithealth (1984).

All tests were administered by the principal investigator and the Oregon State University laboratory technician. Data were analyzed by the principal investigator.

**PROCEDURES**

Subjects were selected for initial participation in the study based on responses to the DEA. Those selected to complete the entire project attended four laboratory sessions, one which involved answering a series of exercise and diet questionnaires (total time 1.25 hours), two sessions of approximately 1.25 and 2.5 hours in duration during which metabolic measurements were conducted, and a fourth session of approximately 45 minutes in which a second RMR measure was conducted.

**Questionnaires.** During the first session, subjects met with the investigator at her campus office to complete six inventories or questionnaires -- the EAT-26, the BULIT, a food frequency questionnaire (HHHQ) and 24-hour recall, informed consent and medical history. Assessment of resting vital measures (blood pressure, heart rate), height, and weight were determined. Those subjects identified as having diets of less than or equal to 1200 calories were assigned participation as LC subjects, while those having diets of greater than 1700 kcals/day were assigned to the NC group. If any subject received scores of 20 or greater on the EAT and/or 102 on the BULIT, she would have been
eliminated from the study, due to confounding factors associated with eating disorders. No subject received such scores.

The second laboratory session was conducted at least three days following completion of questionnaires, and included the following measures:

**Body Composition.** Lung measures for expiratory reserve and residual volume were determined by having each subject breath into the pulmonary function equipment, and was then followed by body composition measurement by means of hydrostatic weighing. The water in the tank was near body temperature (35 - 37°C). Sitting on a chair that is suspended from a scale, each subject submerged herself at the end of a maximal exhalation and remained underwater for approximately 5 seconds while the scale was read. This was repeated 4 - 5 times. Subjects were weighed again on dry land.

**Maximal Aerobic Capacity.** The last assessment of the second laboratory session was VO$_{2\text{max}}$. After receiving verbal instructions and practice on the treadmill, subjects' maximal VO$_2$ consumption was measured using the following protocol: Subjects started at a 6% grade at 5 mph for a three minute warm-up. The protocol continued with another minute at 6% grade, 5 mph, after which the speed was increased by .5 mph/min until reaching a speed of 7 mph. At that point, the grade was then increased by 1% per minute until the subject became exhausted. The maximal oxygen consumption test took approximately 10 - 15 minutes. Attainment of VO$_{2\text{max}}$ was based on achieving at least two of the following three criteria: a) a plateau or increase of less than 2.0 ml · kg$^{-1}$ · min$^{-1}$ VO$_2$ over two consecutive stages of increasing work; b) heart rate greater than 90% of age predicted maximum, and; c) respiratory exchange ratio (R) greater than or equal to 1.1. The session concluded with a second 24-hour diet recall. Each subject was allotted approximately one-and-one-half hours for the entire testing session.

The third series of measurements were taken at least 36 hours following the completion of the VO$_{2\text{max}}$ test.

**Resting Metabolic Rate.** For the third session, each subject arrived at the lab between 6:30 and 9:30 a.m., within one hour after arising, and with a minimum of ten-hours since having eaten and 24-hours since having exercised. Using calendar day estimation, all attempts were made to avoid subjects' ovulatory phase of menstrual cycle. Upon arriving at the laboratory, subjects sat quietly for a minimum of fifteen minutes prior to testing. Resting metabolic rate was then measured by having subjects sit quietly and breathe for one half-hour into a Rudolph face mask attached by a 6-foot hose to the Sensormedics 2900 metabolic cart for gas analysis and determination of oxygen.
consumption. Measurement of RMR was conducted at a fourth and final laboratory session, following the identical protocol, at least one week, but not more than three weeks apart.

**Submax exercise and EPOC.** Following the initial RMR measurement taken at the third laboratory session, each subject underwent a 45-minute submaximal treadmill test at 65% of her VO_{2max}. Immediately following the submaximal work, subjects were asked to sit quietly and continue breathing through the face mask while expiratory gases were measured for EPOC. These gases were measured continuously for one hour.

**EXPERIMENTAL DESIGN**

Subjects were assigned to one of two groups -- a) Normal diet, moderate exercise (NC) or b) Low calorie, moderate exercise (LC). The NC group consisted of 10 females aged 18 - 25 whose daily caloric intake was estimated to be ≥ 1700 kcal and whose exercise volume was greater than or equal to than 4 hours per week. The LC group was composed of five females whose exercise volume and intensity was equivalent to the NC group's, but whose daily caloric intake was estimated at ≤ 1200 kcals. Subjects maintained both diet and exercise patterns for a minimum of nine months. No treatment was assigned, and measurements consisted of a single series of tests measuring body composition, maximal VO_{2}, and EPOC, and two measurements of RMR.

Due to non-random assignment into initial groups (NC vs LC), a quasi-experimental design was utilized. Scheduling of test measurement followed an independent t-test design.

**STATISTICAL ANALYSIS**

Measurement of resting metabolic rate was compared between groups over two trials, whereas EPOC and other comparisons of body composition and maximal VO_{2} were compared between groups over a single time period, with all data expressed as Mean ± standard deviation. Statistical analysis was conducted by means of an independent t-test to detect a moderate difference between means. Alpha was originally set at .025 for each dependent measure to achieve a power of .80 for detecting a moderate (1.5 - 1.75) effect size between groups, but due to low sample size, the anticipated power
of .80 was not attained. Statistical analysis of data collected was carried out on a Macintosh LC II using StatView 4.01 by Abacus Concepts.
RESULTS AND DISCUSSION

RESULTS

A total of fifteen subjects completed this study. Ten subjects were assigned into the Normal Calorie group (NC) (caloric intake ≤ 1700 kcal/day), while the other five were assigned into the Low Calorie group (LC) (caloric intake ≥ 1200 kcal/day). Four additional subjects initially recruited into the low calorie group discontinued participation for a variety of reasons -- one became pregnant, one's athletic team commitment in playoffs prevented her from adhering to the physical activity restrictions, and two took on jobs in addition to school commitments such that they felt unable to meet the demands of the study. Descriptive characteristics of participating subjects are outlined in Table 1. Normal and low calorie subjects demonstrated no difference in VO₂max (p = .93). Individual VO₂max results are presented in Appendix G, Table 7. The low calorie group appeared to have a higher total body fat composition, but this difference was not statistically significant (p = .07). The only characteristic in which subjects differed significantly was total caloric intake (p < .001), as determined by two 24-hour recalls and the DEA (outlined in Chapter 2).

EAT-26, BULIT, HHHQ, and 24 hour recalls. Subjects completed two questionnaires for the purpose of identifying any individual with an active eating disorder. No subject scored above recommended cut-off scores on either evaluation. On the EAT-26, designed to identify subjects with anorexia nervosa, NC subjects' scores ranged from 0 - 15 points, and LC subjects' scores ranged from 6 - 17 points. The cut-off score on the EAT-26 is a score ≥ 30. On the BULIT, designed to identify individuals with bulimarexia, NC subjects' scores ranged from 38 - 92, and LC subjects' ranged from 48 - 64. The cut-off score for this test is a score ≥ 102 points.

The Block Health History Questionnaire (HHHQ) was not included in the final analysis of data. Due to a flaw in the program disk, it was not possible to run the compiled data. Several attempts were made to locate the error and re-analyze the data, to
no avail. Therefore, dietary intake was assessed only by the initial DEA and two 24-hour recalls. In the event that a subject's two 24-hour recalls demonstrated a difference of greater than 500 kcals, or would have placed her in a group different from the one assigned, a third recall was taken, and an attempt to have the subject identify the diet(s) most representative of her daily intake was made. All subjects were initially placed in the group which most accurately reflected their average daily caloric intake based on the DEA. The two 24-hour reports served to confirm that subjects were placed in the appropriate groups, as each subject's 24-hour recalls were found to corroborate initial DEA dietary intake range. The two 24-hour recalls resulted in estimated caloric intakes which varied approximately 200 - 500 kcals between reports in the NC group, and approximately 50 - 200 kcals in the LC group. Individual results for the DEA, two 24-hour recalls, BULIT, and EAT-26 are presented in Table 2.

Table 1.

Mean Physical and Training Characteristics of Normal and Low Calorie Groups.

<table>
<thead>
<tr>
<th>CHARACTERISTIC</th>
<th>NORMAL</th>
<th>LOW CALORIE</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE (years)</td>
<td>20.5 (2.5)</td>
<td>20.8 (2.3)</td>
</tr>
<tr>
<td>WEIGHT (kg)</td>
<td>59.2 (8.9)</td>
<td>56.6 (8.6)</td>
</tr>
<tr>
<td>HEIGHT (in)</td>
<td>65.4 (3.2)</td>
<td>65.7 (1.2)</td>
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<tr>
<td>% BODY FAT</td>
<td>16.9 (4.1)</td>
<td>21.2 (4.1)</td>
</tr>
<tr>
<td>RESTING BP (mmHg)</td>
<td>105/64</td>
<td>101/62</td>
</tr>
<tr>
<td>RESTING HR (bpm)</td>
<td>53.7 (5.5)</td>
<td>50.8 (11.26)</td>
</tr>
<tr>
<td>MEAN CALORIC INTAKE (kcal/day)</td>
<td>1949 (201) *</td>
<td>1206 (79) *</td>
</tr>
<tr>
<td>MEAN HOURS EXERCISE/WEEK</td>
<td>5 (1.0)</td>
<td>6 (1.2)</td>
</tr>
<tr>
<td>VO2max (ml · kg⁻¹ · min⁻¹)</td>
<td>51.0 (5.34)</td>
<td>50.8 (3.13)</td>
</tr>
</tbody>
</table>

All data expressed as Mean (standard deviation). * p < .001
Table 2.

Individual Dietary Intake Assessments and BULIT and EAT-26 Scores for Normal and Low Calorie Subjects

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>DEA</th>
<th>24 HR RECALL 1</th>
<th>24 HR RECALL 2</th>
<th>BULIT (&lt; 102)</th>
<th>EAT-26 (&lt; 30)</th>
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<tr>
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<td>1630</td>
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<td>1825</td>
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<td>2305</td>
<td>49</td>
<td>5</td>
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<td>77</td>
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<td>MEAN</td>
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<td><strong>5</strong></td>
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<td><strong>270.9</strong></td>
<td><strong>18.5</strong></td>
<td><strong>4.6</strong></td>
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<td>1325</td>
<td>1295</td>
<td>1025</td>
<td>58</td>
<td>17</td>
</tr>
<tr>
<td>1116</td>
<td>1200</td>
<td>1245</td>
<td>1190</td>
<td>64</td>
<td>12</td>
</tr>
<tr>
<td>1118</td>
<td>1200</td>
<td>1300</td>
<td>1290</td>
<td>53</td>
<td>14</td>
</tr>
<tr>
<td>1119</td>
<td>1250</td>
<td>980</td>
<td>1145</td>
<td>62</td>
<td>6</td>
</tr>
<tr>
<td>MEAN</td>
<td><strong>1258</strong></td>
<td><strong>1214</strong></td>
<td><strong>1182</strong></td>
<td><strong>57</strong></td>
<td><strong>11</strong></td>
</tr>
<tr>
<td>ST DEV</td>
<td><strong>53.9</strong></td>
<td><strong>119.1</strong></td>
<td><strong>93.6</strong></td>
<td><strong>5.9</strong></td>
<td><strong>4.4</strong></td>
</tr>
</tbody>
</table>
Menstrual status and cycle phase. Subjects were asked to average menstrual cycle phase length over the past three to six months, and to report the first day of their most recent cycle. Menstrual cycle was assessed by calendar day, with cycle phase length averaging from 21 to 30 days in twelve subjects and from 48 - 90 days in one subject. One subject had not menstruated in the previous three months, since stepping up her training for track season. One subject had not menstruated in the past nine months, due to treatment for endometriosis with oral contraceptives. She had discontinued use, by doctor's order, prior to participating in any metabolic testing, but did not menstruate at any time during her participation in the study. Menstrual status and estimated day of cycle in RMR trials 1 and 2 for all subjects are presented in Table 3. Four subjects were using oral contraceptives during the course of the study, and all had been taking them for at least six months prior to beginning the study. The various prescriptions used were Ortho-Novum (7-7-7), Lo-Ovral, Tri-Levlin, Micronor, and Modicum 28. Dosage levels of the various oral contraceptive prescriptions used by participants are given in Appendix H, Table 8.

Resting Metabolic Rate. All subjects had their resting metabolic rate measured on two different days, both within one-and-one-quarter hours of arising in the morning and following a ten-hour fast and 24-hour period of restricted physical activity. Subjects reclined quietly for fifteen minutes upon arrival at the lab, during which time heart rate and blood pressure measurements were taken. The Rudolph face mask was placed over the face and expired gases were collected for 30 minutes. Statistical analysis revealed no significant difference between RMR trials 1 and 2 within the same group (p = .60, NC group, and p = .85, LC group) or between groups in either trial (p = .27, trial 1, and p = .18 in trial 2). Table 4 summarizes the mean RMR findings between the two groups.

Individual subject data for both trials of RMR are presented in Appendices I and J, Tables 9 and 10. Inter-individual differences were noted between trials, ranging from .25 - 1.05 ml · kg⁻¹ · min⁻¹ (6.2 - 3.72 L · 60 min⁻¹) in the NC group and .13 - .82 ml · kg⁻¹ · min⁻¹ (.34 - 3.27 L · 60 min⁻¹) in the LC group. The mean difference between trials in the NC group was .51 ml · kg⁻¹ · min⁻¹ (1.84 · 60 min⁻¹) and .32 ml · kg⁻¹ · min⁻¹ (1.29 · L 60 min⁻¹) in the LC group.
Table 3.

Menstrual Status and Estimated Day of Cycle in RMR Trials 1 and 2 for Normal and Low Calorie Subjects.

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>CYCLE LENGTH</th>
<th>RMR I Estimated Day of Cycle</th>
<th>RMR II Estimated Day of Cycle</th>
<th>OC USE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NORMAL CALORIE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1101</td>
<td>28</td>
<td>day 5</td>
<td>day 24</td>
<td>+</td>
</tr>
<tr>
<td>1102</td>
<td>25</td>
<td>day 9</td>
<td>day 16</td>
<td>-</td>
</tr>
<tr>
<td>1103</td>
<td>*</td>
<td></td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>1105</td>
<td>23</td>
<td>day 20</td>
<td>day 4</td>
<td>+</td>
</tr>
<tr>
<td>1106</td>
<td>24</td>
<td>day 10</td>
<td>day 8</td>
<td>-</td>
</tr>
<tr>
<td>1107</td>
<td>28</td>
<td>day 21</td>
<td>day 2</td>
<td>+</td>
</tr>
<tr>
<td>1108</td>
<td>28</td>
<td>day 25</td>
<td>day 23</td>
<td>-</td>
</tr>
<tr>
<td>1110</td>
<td>21</td>
<td>day 16</td>
<td>day 15</td>
<td>-</td>
</tr>
<tr>
<td>1112</td>
<td>27</td>
<td>day 19</td>
<td>day 18</td>
<td>-</td>
</tr>
<tr>
<td>1117</td>
<td>29</td>
<td>day 5</td>
<td>day 18</td>
<td>-</td>
</tr>
<tr>
<td><strong>LOW CALORIE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1104</td>
<td>**</td>
<td></td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>1113</td>
<td>30</td>
<td>day 18</td>
<td>day 18</td>
<td>-</td>
</tr>
<tr>
<td>1116</td>
<td>28</td>
<td>day 22</td>
<td>day 5</td>
<td>-</td>
</tr>
<tr>
<td>1118</td>
<td>27</td>
<td>day 22</td>
<td>day 25 #</td>
<td>-</td>
</tr>
<tr>
<td>1119</td>
<td>60-90</td>
<td></td>
<td></td>
<td>+</td>
</tr>
</tbody>
</table>

* Using OC prior to testing for treatment of endometriosis - no menstrual cycle
** Athletic-induced amenorrhea; # - menses onset 10 hours post-test
- indicates non OC use; + indicates OC use
Five subjects conducted the two RMR testing trials during different estimated menstrual cycle phases. Three individuals demonstrated a higher RMR during estimated luteal phase, while the other two obtained higher values during estimated follicular phase. No difference between phases was noted (2.96 ± .32 vs 2.95 ± .48 ml · kg⁻¹ · min⁻¹, follicular vs. luteal, respectively).

Table 4.
Summary of Resting Metabolic Rate Trials 1 and 2 for Normal and Low Calorie Groups.

<table>
<thead>
<tr>
<th></th>
<th>NORMAL</th>
<th>LOW CALORIE</th>
</tr>
</thead>
<tbody>
<tr>
<td>RESTING HR (bpm)</td>
<td>53.7 (5.5)</td>
<td>50.8 (11.26)</td>
</tr>
<tr>
<td>RMR 1 (ml · kg⁻¹ · min⁻¹)</td>
<td>2.98 (.37)</td>
<td>2.83 (.45)</td>
</tr>
<tr>
<td>(L · min⁻¹)</td>
<td>.18 (.03)</td>
<td>.18 (.02)</td>
</tr>
<tr>
<td>(ml · kg⁻¹ · FFM⁻¹)</td>
<td>3.57 (.42)</td>
<td>3.55 (.56)</td>
</tr>
<tr>
<td>RMR 2 (ml · kg⁻¹ · min⁻¹)</td>
<td>3.07 (.42)</td>
<td>2.89 (.25)</td>
</tr>
<tr>
<td>(L · min⁻¹)</td>
<td>.16 (.03)</td>
<td>.16 (.03)</td>
</tr>
<tr>
<td>(ml · kg⁻¹ · FFM⁻¹)</td>
<td>3.68 (.39)</td>
<td>3.68 (.40)</td>
</tr>
<tr>
<td>MEAN RMR (ml · kg⁻¹ · min⁻¹)</td>
<td>3.02 (.27)</td>
<td>2.86 (.27)</td>
</tr>
<tr>
<td>MEAN KCAL /MIN</td>
<td>.88 (.10)</td>
<td>.79 (.13)</td>
</tr>
<tr>
<td>MEAN R VALUE</td>
<td>.84 (.04)</td>
<td>.89 (.06)</td>
</tr>
</tbody>
</table>

All data expressed as Mean (standard deviation)
Submaximal Exercise. Treadmill speed and grade was calculated for each subject based on achieving 65% of their determined VO$_{2\text{max}}$. Thirteen participants were able to maintain running speeds to complete the 45 minute test. Two subjects felt muscular or joint discomfort and were brought to a walking pace, while grade was increased to maintain the desired VO$_2$. Subjects in both groups averaged a submaximal level of 66% of determined VO$_{2\text{max}}$, with relative VO$_2$ ranging between 28 - 37 ml · kg$^{-1}$ · min$^{-1}$. Table 5 presents the mean metabolic responses of all subjects to submaximal and maximal exercise.

Table 5.
Mean Metabolic Responses to Exercise for Normal and Low Calorie Groups.

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>NORMAL</th>
<th>LOW CALORIE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SUBMAXIMAL EXERCISE (66% VO$_{2\text{MAX}}$)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MEAN HR (bpm)</td>
<td>167 (8)</td>
<td>168 (8)</td>
</tr>
<tr>
<td>MEAN VO$_2$ (ml · kg$^{-1}$ · min$^{-1}$)</td>
<td>33.5 (2.9)</td>
<td>33.3 (1.7)</td>
</tr>
<tr>
<td>MEAN KCAL · MIN$^{-1}$</td>
<td>9.89 (1.6)</td>
<td>9.31 (1.4)</td>
</tr>
<tr>
<td>MEAN R VALUE</td>
<td>.88 (.04)</td>
<td>.89 (.06)</td>
</tr>
<tr>
<td><strong>MAXIMAL EXERCISE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MEAN MAXIMAL HR (bpm)</td>
<td>191 (4)</td>
<td>187 (9)</td>
</tr>
<tr>
<td>VO$_{2\text{MAX}}$ (ml · kg$^{-1}$ · min$^{-1}$)</td>
<td>51.0 (5.4)</td>
<td>50.8 (3.1)</td>
</tr>
<tr>
<td>MEAN KCAL · MIN$^{-1}$</td>
<td>15.5 (2.7)</td>
<td>15.0 (2.0)</td>
</tr>
<tr>
<td>MEAN PEAK R VALUE</td>
<td>1.13 (0.3)</td>
<td>1.16 (0.2)</td>
</tr>
</tbody>
</table>

All data expressed as Mean (standard deviation)

EPOC. Two participants found it painful to wear the face mask throughout the two-and-one-quarter hour period and were switched to the mouthpiece with two-way valve when EPOC had dropped into the long-term phase. Only data for nine NC subjects
were used, as one individual experienced severe discomfort with the face mask and removed it before the mouthpiece was in place, thereby losing the first five minutes of her EPOC data collection.

Group mean recovery VO₂ and EPOC are summarized in Table 6. Absolute mean recovery VO₂ ranged from 10.37 - 18.75 L · 60 min⁻¹ in the NC group (μ = 14.56 L · 60 min⁻¹) and 10.31 - 17.23 L · 60 min⁻¹ in the LC group (μ = 14.35 L · 60 min⁻¹). Mean absolute recovery VO₂ in both groups was significantly different from absolute resting VO₂, indicating that the exercise duration of 45 minutes and intensity of 65% VO₂max was adequate to elicit EPOC (p < .001 and p = .007, NC and LC groups, respectively). No difference between groups in total recovery VO₂ was noted (p = .44). Individual EPOC data are presented in Appendix K, Table 11.

Table 6.
Mean Recovery VO₂ and Energy Expenditure for Normal and Low Calorie Groups.

<table>
<thead>
<tr>
<th>RECOVERY VARIABLE</th>
<th>NORMAL</th>
<th>LOW CALORIE</th>
<th>p =</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 9</td>
<td>n = 5</td>
<td></td>
</tr>
<tr>
<td>DURATION - SHORT (min.)</td>
<td>1:00 (:17)</td>
<td>1:24 (:33)</td>
<td>p = .11</td>
</tr>
<tr>
<td>TOTAL RECOVERY VO₂ (L)</td>
<td>14.56 (2.34)</td>
<td>14.35 (2.51)</td>
<td>p = .49</td>
</tr>
<tr>
<td>EPOC (L)</td>
<td>4.28 (1.23)</td>
<td>4.83 (2.27)</td>
<td>p = .36</td>
</tr>
<tr>
<td>EPOC (L · min⁻¹)</td>
<td>.24 (.03)</td>
<td>.25 (.07)</td>
<td>p = .36</td>
</tr>
<tr>
<td>MEAN R</td>
<td>.78 (.04)</td>
<td>.80 (.06)</td>
<td>p = .87</td>
</tr>
<tr>
<td>TOTAL RECOVERY ENERGY EXPENDITURE (kcal)</td>
<td>71.07 (8.41)</td>
<td>77.20 (21.32)</td>
<td>p = .45</td>
</tr>
<tr>
<td>NET RECOVERY ENERGY EXPENDITURE (kcal)</td>
<td>19.5 (5.83)</td>
<td>29.04 (18.84)</td>
<td>p = .08</td>
</tr>
<tr>
<td>ELEVATION LAST 5 MIN (L · min⁻¹)</td>
<td>.21 (.02)</td>
<td>.19 (.03)</td>
<td>p = .24</td>
</tr>
</tbody>
</table>

All data expressed as Mean (standard deviation)
Total increase in oxygen consumption for the 60 minutes post-exercise (EPOC) was determined by subtracting the same day's measured absolute mean RMR over 60 min from total recovery VO2. No difference between groups was noted (p = .36). To determine if either group demonstrated significant VO2 elevation above resting levels at the end of 60 minutes, or if there was a difference in the rate at which EPOC declined, EPOC was averaged in five minute intervals over the 60 minute collection period. Mean relative elevation in VO2 during the last 5 minutes of collection was .57 ml · kg⁻¹ · min⁻¹ (.21 L · min⁻¹) in the NC group and .55 ml · kg⁻¹ · min⁻¹ (.19 L · min⁻¹) in the LC group. No difference between the groups was demonstrated in relative or absolute VO2 in the last five minutes. At 60 minutes post-exercise, both groups still demonstrated a significant elevation over resting VO2 (p < .01 and p = .034, NC and LC groups, respectively), with the NC group demonstrating a slightly greater absolute elevation in the last five minutes (.21 ± .02 vs .19 ± .03 L · min⁻¹). The five minute averages of EPOC for each group are presented in Appendix L, Table 12.

Total energy expenditure during the 60 minutes post-exercise tended to be greater in the LC group, but was not significantly different between the groups (p = .28). When the net recovery energy expenditure was compared between groups, the difference was more noticeable, but still not significant (p = .16). One LC subject demonstrated distinct differences from all other subjects in all EPOC measures. She demonstrated the longest short phase of recovery, the greatest recovery VO2, and post-exercise energy expenditure equivalent. Due to her noticeably different results, analyses on EPOC were run both with and without her data, but exclusion of her data did not change the result of any finding in terms of statistical significance.
DISCUSSION

A lack of subjects able to complete participation in this study created difficulties in acquiring the proper statistical power and determining the appropriate tests to be conducted in analyzing the data. Despite this flaw, certain patterns and trends emerge which warrant discussion.

All subjects were generally similar in physiognomic characteristics, as well as age and average exercise participation per week. All subjects demonstrated bradycardia and above average aerobic capacity, reflective of their fairly high volume and intensity of physical activity.

General Characteristics. Notwithstanding the LC group's slightly lower body weight, this group demonstrated a higher body fat percentage, and it is possible that this difference would have been significant had groups been equal and the statistical power increased. It is interesting to note that in addition to the LC group's lower body weight and higher percentage body fat, they also engaged in an average of one hour more of exercise per week. It could be speculated that the LC groups' greater fat storage may act as a protective measure against periods of restricted intake, prolonged exercise, and potential weight loss. Lack of research in this area renders this question as conjecture. Two of three LC subjects with the greatest percentage body fat had periods of disordered eating two to three years prior, followed by weight cycling before achieving a more constant weight. In the past year, three of the five LC subjects had been on at least two diets to lose weight, none losing more than five pounds each time, of which some or most was regained. In the NC group, three subjects had said they had gone on one diet. No subject had dieted within the past five months. Research has indicated that weight cycling, or yo-yo dieting, may contribute to an increased fat gain with each successive weight loss/weight gain cycle (Hegarty, 1988). During the course of the study, no subject demonstrated a weight fluctuation of more than one pound in three different weighings.

Caloric Intake. Subjects were asked to maintain their current dietary habits as much as possible during the testing period. Because of the ten-hour fasting period prior to RMR testing, 24-hour recalls were not used on those days. The majority of subjects indicated that late night snacking while studying was common, and the ten-hour fast caused them to slightly change normal patterns. Estimated caloric intake showed greater
variation in the NC group between recalls, whereas the LC group demonstrated remarkable similarity between reports. Earlier discussion of the weaknesses and limitations of the 24-hour recall included possible misrepresentation of portion size and a tendency for subjects to under-report or alter information. Great effort was made to encourage subjects to be as accurate and detailed as possible regarding their recall. The relationship between the accuracy of their report and outcome of the tests being conducted was stressed, and all subjects seemed genuinely concerned with recalling their actions and eating of the previous day. No dietary patterns emerged other than in total intake, and all subjects but one from the LC group, who skipped both breakfast and lunch, ate the equivalent of breakfast, lunch, and dinner "meals." The Block HHHQ would have provided substantiation of estimated caloric intake, as well as an indication of overall nutritional status, revealing any possible macro- or micro-nutrient deficiencies in either group (most likely in the LC group).

**Menstrual status and cycle phase.** Menstrual status and oral contraceptive use varied among participants. While stricter control over both parameters would have been ideal, several reasons exist for the inclusion of both OC users and amenorrheic subjects. As mentioned previously in this chapter, the subject pool was tightly defined and limited by the criteria established for age, caloric intake, and exercise levels. One-quarter of participants in the study used an oral contraceptive, and this percentage is representative of, if not slightly lower than, the percentage of OC users in the pool of subjects who initially were screened for the study but who did not meet age, dietary, or exercise criteria. Strong evidence was presented in Chapter 2 which clearly indicated recent research investigating the role of OC on exercise performance had shown minimal, if any, changes occurring with OC use (Bonen et al., 1983; Gray et al., 1983; Montes et al., 1983). Of the two studies which demonstrated small changes, one noted that while VO$_{2\max}$ was slightly diminished by OC use, submaximal performance was not affected at all (Notelovitz, 1987), and the other conceded that, for the majority of women, exercise performance is not affected by either OC use or menstrual cycle phase (Lebrun, 1994). Due primarily to this evidence, and because of the limited subject pool, the decision was made to not exclude subjects on the basis of OC use.

To verify whether any difference between the OC users and non-users within this study did actually exist, comparisons were made between the five OC users and ten non-
users. On the variables of weight (p = .65), percent body fat (p = .26), \( VO_{2\max} \) (p = .58), RMR (p = .58), and EPOC (p = .89), no significant differences between the groups were found.

In regard to inclusion of amenorrheic subjects, the limited subject pool again factored into this decision. Evidence presented in Chapter 2 was again strongly indicative that no compromising differences in exercise performance or resting metabolism exist between cycle phase or status, especially in trained subjects (De Souza et al., 1990; Dombovy et al., 1987; Jurkowski et al., 1981; Kanaley et al., 1992; Nicklas et al., 1989; Pivarnek et al., 1992; Schoen et al., 1981). Presence of amenorrhea made the use of calendar day estimation of menstrual cycle phase irrelevant. The use of calendar day estimation was used in attempt to avoid the ovulatory phase and menstruation during RMR testing, but recent research has indicated that normal menstruation cycles do not necessarily denote that ovulation has occurred or that the luteal phase is normal (Prior & Vigna, 1991). Tests conducted in each subject's estimated follicular phase took place on days four through ten, depending on the average cycle length. Tests conducted during the estimated luteal phase took place on days 16 - 25, again dependent on average cycle length. Since no hormonal or basal temperature documentation took place to verify ovulation and cycle phase, these estimates present a confound should evidence indicate a difference in metabolic responses at rest or exercise due to menstrual cycle phase. One subject later confirmed that approximately 10 hours following the second test, she began menstruation, three days earlier than expected. While this incident, in conjunction with fluctuations in R values, reflects the need for greater menstrual phase control, previous research has not provided strong evidence for menstrual status or luteal or follicular phase differences in trained females.

Resting Metabolic Rate. Three subjects admitted that the restriction on physical activity was the most difficult of the testing criteria to meet, but that they were able to comply with the requirement. All subjects demonstrated intra-individual variation between the two test periods. Although controls were placed on the timing of the last food intake and subjects were asked to eat as normally as possible during the course of the testing period, this control did not extend to the content of food intake. For example, one NC subject whose differences between trials was quite pronounced (\( -1.05 \text{ ml} \cdot \text{kg}^{-1} \))
· min⁻¹), was questioned about the 24-hour period preceding her second RMR trial. She revealed that her dietary intake consisted of more food than her estimated normal intake of approximately 1700 kcals, and was composed of mostly high fat fast food (hamburger, french fries, ice cream, burrito, refried beans, and chips), something she rarely, if ever, ate, especially in the same day.

While no statistical differences were apparent within each group between RMR trials, individual variation between trials may raise questions. The restriction placed on physical exercise in the 24 hours preceding testing did not require complete abstinence of physical activity. Subjects were allowed to engage in light walking or slow bike riding during the previous day to transport themselves to and from their classes and/or jobs. Minor differences in the amount of this type of activity may have influenced RMR results. Additionally, while an attempt to estimate menstrual cycle phase was made, it was not successful in one subject who underwent testing just 10 hours prior to onset of menstruation, and was unavailable for retesting. Lastly, several subjects were in the laboratory during stressful periods of mid-term or final exams and/or oral presentations. Despite instructions to remain as relaxed and quiet as possible during RMR determination, most subjects admitted reviewing facts or rehearsing in their head while lying on the laboratory table. It would seem that, given the differences between trials among subjects, the effort to control timing of intake, physical activity, menstrual phase, or pre-test standardization may not have been adequate to reduce intra-individual variations in RMR which can be attributed to these and possibly other unknown factors.

Despite the remarkably lower caloric intake, the LC group did not demonstrate a lower RMR than the NC group. Computer analysis of intake indicated that, on average, the normal group took in approximately 80 ± 10% of their recommended intake based on gender, age, weight, height and moderate physical activity, while the LC group's intake was estimated at approximately 54 ± 4% recommended values. It is well established that caloric restriction depresses metabolic rate (Bray, 1986; Lammert and Hansen, 1982; Mole et al., 1989), but the role of exercise in effecting a chronic elevation in RMR is more controversial. There is evidence that over a prolonged period of time, exercise of adequate intensity and duration may promote an increase in RMR (Poehlman et al., 1988 and 1990; Tremblay et al., 1986). When caloric restriction accompanies exercise, it is questioned whether or not exercise can counteract the decline in RMR. As Chapter 2
indicated, it may be that caloric restriction at approximately 800 kcals·day\(^{-1}\) or below, accompanied by exercise of 50 - 60% \(\text{VO}_2\text{max}\) or one-to-two hours in duration does not reverse the decline RMR and may even accelerate it (Krokiewsky et al., 1981; Phinney et al., 1988; Poehlman et al., 1991; Warrick & Garrow, 1981). Conversely, restriction of caloric intake to between 1200 - 2000 kcals·day\(^{-1}\) in combination with exercise sessions of approximately 60% \(\text{VO}_2\text{max}\) for 30 - 45 minutes duration was associated with increases in RMR from dieting-only levels (Lennon et al., 1985; Mole et al., 1989; Neiman et al., 1988; Svendsen et al., 1994). Since the LC subjects maintained dietary intakes between approximately 1000 and 1300 kcals/day and exercised approximately 45 minutes to 2 hours per session at intensities estimated to be between 50 - 70% \(\text{VO}_2\text{max}\), they may have been just above the "threshold" where exercise is no longer able to stimulate RMR.

R values showed little difference between the two groups at rest, although the LC group demonstrated a slightly higher R value in each RMR trial. Intra-individual variability ranged from differences of .01 - .10 between trials, but no difference between RMR trials was seen (\(p = .81\) and \(p = .48\), NC and LC groups, respectively). The greatest difference between trials was demonstrated in an NC subject and served to indicate the need for greater dietary control as well as hormonal documentation of menstrual cycle phase within the study. Additionally, several studies have indicated a lower R value during luteal phase of the menstrual cycle, but it has been suggested that endurance training may override this difference (Bonen et al., 86; De Souza et al., 90; Kanaley et al., 1992). In the five subjects undergoing RMR testing during two different phases of the menstrual cycle, all but one demonstrated a slightly lower R value during luteal phase.

**EPOC.** The short phase of EPOC was determined to be concluded when the rapidly declining phase in recovery \(\text{VO}_2\) was over and the very gradual decline began. The short phase of EPOC was not found to be significantly different between groups (\(p = .11\)), although one LC subject did demonstrate a slightly longer short phase (180 seconds) when compared to an average of 100 and 124 seconds in NC and LC subjects, respectively. These values for the short phase of \(\text{O}_2\) recovery from exercise are
consistent with the current understanding that the short phase is not dependent on intensity or duration of exercise and is complete within two to three minutes (Hill and Lupton, 1923).

The finding of a pronounced EPOC in both groups is in contrast to findings of Poehlman et al. (1991), who concluded that exercise intensities of 50 - 75% VO$_{2\text{max}}$ were not sufficient to produce EPOC unless duration was greater than 80 minutes and feeding occurred. Differences in training status of subjects, exercise protocol, control for food intake, and baseline VO$_2$ criteria are only a few of the variables which may have contributed to the discrepancies between the findings.

Although subjects of both groups demonstrated a slight elevation 60 minutes post-exercise, their average total energy expenditures fell within previously reported averages of 17 - 28 kcals post-exercise (Brehm & Guten, 1986; Freedman-Akabas et al., 1985; Knuttgen, 1970). Energy expenditure in the 60 minutes post-exercise remained significantly greater than expenditure at rest (p ≤ .01) in both groups. When the one LC subject who demonstrated such a wide discrepancy from other subjects in EPOC measures was questioned about the duration and intensity of the submaximal test as compared to her normal exercise patterns, she admitted that her normal routine was not as strenuous, although the duration was at least equivalent. No other subjects indicated subjective or objective differences in the intensity or duration of the exercise protocol as compared to their normal exercise.

The finding of a significant elevation in the last five minutes of the 60-minute EPOC in both groups also seems to counter the findings that, with similar ranges of exercise intensity and duration, EPOC had returned to baseline or near baseline values within 15 - 48 minutes (Brehm & Guten, 1986; Freedman-Akabas et al., 1985; Knuttgen, 1970). Had EPOC measurement continued until all subjects reached a specified percentage of RMR, a difference in duration of EPOC might have been seen between the groups. Whether this finding would be considered significant is not known. The 60-minute limit on post-exercise O$_2$ collection was set a) to accommodate laboratory, subject, and investigator availability, and b) because, based on research findings, the intensity and duration of the submaximal exercise protocol was not expected to elicit an EPOC beyond approximately 45 minutes.
SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

SUMMARY

A substantial body of evidence exists which supports the finding that caloric restriction results in a lowering of resting metabolic rate (RMR). Findings that aerobic exercise may negate or even reverse this decline, however, are conflicting. It is widely believed that a combination of caloric deficit and exercise will accelerate weight/fat loss and prevent the decline of RMR. When caloric intake equals or exceeds $\sim 1000 \text{ kcal} \cdot \text{day}^{-1}$ and exercise intensity remains around 60% $\text{VO}_{2}\text{max}$ with a duration of 30 - 45 minutes, reversal of the RMR decline has been documented. Some investigations, however, suggest that the effects of severe caloric restriction in tandem with moderate amounts of aerobic exercise may exacerbate the decline in RMR.

The effects of caloric restriction on EPOC have not been well researched, and even studies on mitigating factors of EPOC, primarily intensity and duration of exercise, are fraught with contradictions and inconsistencies. Some research suggests that unless exercise intensity is greater than 75% $\text{VO}_{2}\text{max}$ and duration greater than 80 minutes, EPOC will return to resting or near-resting $\text{VO}_{2}$ within 15 - 45 minutes.

The purpose of this study was to examine whether college-aged females engaging in chronic caloric restriction ($\leq 1200 \text{ kcal} \cdot \text{day}^{-1}$) and moderately intense exercise exhibit differences in resting metabolic rate and/or EPOC when compared to similar females who take in approximately 1700 kcals or greater per day. Two hypotheses were undertaken in this study: a) NC subjects would demonstrate a greater RMR than equally trained LC subjects, and b) NC subjects would demonstrate a greater EPOC than equally trained LC subjects.

Fifteen females were selected through their responses to questionnaires on diet and exercise habits, and were assigned to one of two groups based on their average daily caloric intake and exercise volume. Subjects assigned to the NC group ate a minimum of 1700 kcals $\cdot$ day$^{-1}$. Subjects assigned to the LC group consumed an estimated 1200 kcals $\cdot$ day$^{-1}$ or less. Both groups of subjects engaged in aerobic exercise a minimum of four hours per week at $\geq 50\% \text{VO}_{2}\text{max}$. Subjects completed two RMR trials controlled...
for food intake and activity, and a 45 minute submaximal treadmill test (65% VO2max) to elicit EPOC. EPOC was measured for one hour. Independent t-tests were conducted on all dependent variables.

Subjects demonstrated no significant differences between groups in age, weight, height, body mass index, resting heart rate, blood pressure or VO2max. The only significant difference between groups was exhibited in caloric intake (p < .001), although the LC group did show a tendency toward greater body fat composition. No significant difference between groups was noted in RMR (.17 ± .03 vs. .17 ± .03 L·min⁻¹, NC and LC, respectively) or EPOC (.24 ± .03 vs. .25 ± .07 L·min⁻¹, NC and LC, respectively). At 60 minutes post-exercise, both groups still demonstrated a significant elevation over resting VO2 (p < .01 and p = .034, NC and LC groups, respectively), with the NC group demonstrating a slightly greater absolute elevation in the last five minutes (.21 ± .02 vs .19 ± .03 L·min⁻¹). Had gas collection been continued until each subject reached a specified percentage of RMR, the total duration of EPOC may have indicated a slight, possibly significant, difference between the groups. It would appear, based on the results of this study, that the level of caloric intake did not affect RMR or the magnitude of EPOC.

CONCLUSIONS

The research hypotheses undertaken in this study were that a) in subjects whose caloric restriction was below 1200 kcal/day and who engaged in moderate amounts of aerobic exercise at an intensity above 50% VO2max and total volume equal to or greater than 4 hours per week, RMR will be depressed when compared to subjects with a dietary intake of greater than 1700 kcal and exercise of the same intensity and volume, and b) excess post-exercise oxygen consumption following moderate intensity and duration (greater than 55% VO2max and 20 min) will be of a smaller magnitude and shorter duration in low calorie intake (LC), exercising females as compared to females with comparable activity levels and normal caloric intake.

Due to the quasi-experimental design of the study and given the low power, conclusions must be drawn with caution. However, some possibilities can be explored. First, it seems evident that dietary intake between the two groups was remarkably dissimilar, while volume of exercise was approximately equal. In fact, when compared on a variety of physical and physiological characteristics, the only other noticeable, but not significant, difference between the groups was in body composition. No statistical
differences were noted between the LC and NC group in RMR. It would seem that the first research hypothesis underlying this study -- that the LC group would demonstrate a depressed RMR when compared to similar subjects with a greater caloric intake -- was not upheld.

Secondly, while both groups clearly demonstrated a significant elevation of post-exercise oxygen consumption, neither recovery VO₂ or EPOC were different between the two groups, such that the second stated research hypothesis was also not upheld. At 60 minutes post-exercise, both groups still demonstrated a significant elevation over resting VO₂ (p < .01 and p = .034, NC and LC groups, respectively). Had gas collection been continued until each subject reached a specified percentage of RMR, the total duration of EPOC may have indicated a slight, possibly significant, difference between the groups, and the second stated hypothesis would have been upheld. Based on the results of this study, it would appear that the level of caloric intake did not affect RMR or the magnitude of EPOC.

RECOMMENDATIONS

Three primary aspects of this study needed greater control to increase internal validity. First, the limited subject pool, specifically low calorie subjects, made achieving adequate statistical power impossible. On a university campus, where the prevalence of dieting among females is considered high, it was not thought that 10 low calorie subjects would be difficult to come by. In fact, at the outset, nine were selected. A monetary stipend may have enticed more individuals to participate or induced those who dropped out due to work to continue, although it would have had little influence on the other drop-outs in the study.

Dietary control in the 24-hour period prior to metabolic testing could help eliminate differences between trials that now cannot be explained with certainty. If a 24-hour diet recall was given at the time of the first RMR test, that diet could serve as a standardization for the next test. The only limiting factor to this would be those students living in dorms and sororities who did not have control over the food which was served. This was a large percentage of the subjects. Use of three or four-day diet records would provide greater confirmation of eating patterns and caloric intake over time, but issues regarding self-report under-estimation would still be of concern.

Hormonal documentation of serum progesterone and basal body temperature should have been conducted. This information would have helped to eliminate questions
regarding menstrual phase and status, verified that subjects experienced ovulation and that tests were not conducted at this time, and given greater accuracy to differences in R values between RMR tests. While evidence at this time does not suggest performance differences due to menstrual cycle phase or status, findings are equivocal on metabolic fluctuations due to cycle phase.

Additionally, by continuing EPOC measurement until all subjects reached a specified percentage of resting O2 consumption, clarification regarding whether the normal group did exhibit a longer EPOC than the NC group would have been possible. Time presented the major limitation in this decision. Laboratory, investigator, and subject availability made extending the two-and-one-quarter-hour period any longer infeasible.

A series of longitudinal intervention studies would be most beneficial for better control of factors such as exercise, caloric intake, and chronic versus acute effects of caloric restriction. Future studies investigating interactive effects of caloric intake and exercise would benefit by changing the experimental design to include four groups for comparison. This would allow for two control groups of Low Calorie/No Exercise and Normal Diet/No Exercise. In this manner, the effect of exercise could be isolated and the "threshold" level at which exercise is no longer able to attenuate the decline in RMR due to caloric restriction could be more easily identified.

Continuing research needs to address several questions which remain in regard to low caloric intake and metabolic responses at rest and exercise. The question of the role of exercise on resting metabolic rate still lacks unequivocal evidence in either direction. With health and fitness professionals everywhere espousing the importance of combining exercise with diet for effective weight loss and control, it seems imperative we understand the influence of these two factors individually and in tandem. Studies investigating the role of exercise alone on metabolic rate need to carefully define and control population characteristics in terms of body composition and fitness level. Studies need to be conducted on subjects at well-trained, moderately-trained and untrained fitness levels, and in obese, normal weight, and underweight populations. Consistency in the intensity and duration of the exercise protocol needs to be established across a series of studies within each population. The role of aerobic exercise versus and in conjunction with resistance training needs to be further investigated, as early evidence suggests that resistance work may act to increase FFM to a greater extent, thereby increasing metabolically active tissue.

Further inquiry into the effect of exercise in calorie restricted diets is of major importance. The "threshold" level below which exercise may no longer be able to
stimulate metabolic rate as a function of caloric restriction needs to be identified. To do this, caloric intake, and volume, intensity, and duration of exercise need to be meticulously controlled. Again, the level of fitness and body composition of subjects needs to be defined and multiple studies across each factor must be done. It has been suggested by some investigators that, rather than establishing a prescribed VO$_2$ as a workload for comparison, exercise should be prescribed to induce a similar energy expenditure between subjects (Poehlman et al., 1991). While this may be an interesting and valid suggestion, at this point in time, it might serve only to add confusion to the already difficult task of making a comparison between studies which have used various exercise protocols.

Continued work in the area of menstrual status and cycle phase should set standards for documentation of hormonal levels and basal temperature criteria. Lebrun (1995) has even suggested that the cycle phase under which testing takes place should also be standardized. Her multiple investigations in this area of research have begun a definite move in this direction by other researchers. Studies on or utilizing resting metabolic rate need to establish a standard for baseline measurement so that studies can be equated. Strict dietary and physical activity controls need to be effected, and the relation between fluctuations in substrate oxidation during menstrual cycle phase and resting metabolic rate needs to be established.
REFERENCES


APPENDICES
CONSENT FORM

Comparison of Resting Metabolic Rate and Excess Post-Exercise Oxygen Consumption in Normal and Low Calorie Dieting Females

Investigator: Carey A. Hilbert, Master's Candidate, Oregon State University

Purpose: The purpose of this study is to compare the resting metabolic rate and excess post-exercise oxygen consumption in two groups of active females, one eating a normal diet and the other eating a low calorie diet.

I have received an oral explanation of the study procedures and understand they entail:

1. **Completion of questionnaires**
   At the beginning of my involvement in this study, I will need to provide very detailed and accurate information regarding my present dietary and physical activity status and habits. Following initial testing, twenty subjects will be chosen, and I realize I may not be needed to complete the entire project.

2. **Body Composition**
   I will participate in three different laboratory sessions. During the first session, I will have my height and weight recorded, after which I will be asked to breathe into a canister type apparatus to measure my lung capacity. I will have my body composition measured by using an underwater weighing procedure in a specially designed indoor tank. Water in the tank will be near body temperature (35-37°C). Sitting on a chair that is suspended from a scale, I will submerge myself following a maximal exhalation and remain underwater for approximately 5 seconds while the scale is read. This procedure will be repeated 4-5 times.

3. **Maximal oxygen consumption (\(V_{02\max}\))**
   When I have completed the underwater weighing, I will change clothes and complete a test to measure my aerobic capacity. The test will be conducted on a motorized treadmill, starting at a slow speed and progressing with gradual increases in either speed or treadmill elevation until I become too fatigued to continue. The test will take approximately 10 to 15 minutes, with only the final few minutes being at a high intensity.

   During the tests, I will breathe room air through a mouthpiece so that the amount of oxygen I am using can be determined. My heart rate will be continuously monitored by a watch-type apparatus strapped around my chest. Trained laboratory personnel, certified in CPR, will administer the exercise tests.
4. **Resting metabolic rate**
The second laboratory session will require me to arrive at the laboratory in the morning, within one hour of getting up, having had no food for at least 10 hours and no vigorous physical activity for 24 hours. I also must not be in the ovulatory phase of my menstrual cycle. My heart rate and blood pressure will be taken while I sit quietly for 15 minutes. Then I will be asked to breathe for one half-hour into a face mask attached by a 6 foot hose to a large cart for collected gas to be analyzed. I will be asked to come to the lab at least one week after this day to repeat this test under identical circumstances.

5. **Submaximal exercise**
After 30 minutes seated, I will get on the treadmill and run for 45 minutes at a moderate pace. During this time, I will still be breathing into the apparatus which collects the air I exhale.

6. **Excess post-exercise oxygen consumption**
The last test I will perform will immediately follow the 30 minutes of submaximal exercise. I will be brought to a resting, seated position and asked to continue breathing into the facemask. Expired air will be collected continuously for up to one hour.

7. **Risks**
There is a remote risk of death associated with the test of maximal oxygen consumption. In large, varied populations, this risk is one death per 10,000 tests. Since I am from a low risk segment of the population (young and healthy) and will be screened to exclude individuals with known symptoms of heart disease, the risk is considerably less. Furthermore, personnel trained in test administration and CPR will be administering all tests and monitoring for signs of exercise intolerance.

There are no identified risks involved in hydrostatic weighing or determination of resting metabolic rate. Any exercise activity involves a degree of risk of physical injury. I will receive detailed instruction and opportunity to practice on the treadmill prior to testing. Since I am from a low risk segment of the population (young and healthy), and trained personnel will be administering all tests, there is minimal risk involved in my participation.

8. **Benefits**
I will benefit from my participation by contributing to the understanding of the effect of caloric restriction on resting and post-exercise metabolic rate, which are the minimum amount of energy I use to sustain my body at rest and the amount of energy I use after exercise to recover. I will also gain information about my nutritional intake, body mass index, blood pressure, lung capacities, maximal aerobic capacity and percent body fat.

Participation in this study will entail four sessions requiring between 45 to 150 minutes each. The initial laboratory session will entail completing several different questionnaires regarding my health, and diet and exercise patterns. The second session will involve being weighed under water in a tank and running on a treadmill to measure my maximum oxygen consumption. The third session will take place early in the morning after I have fasted for ten hours and not exercised for 24, and require me to sit quietly for one-half
hour while I breathe into a facemask. Then I will run on the treadmill again at an intensity lower than the one I tested in at the previous session. Lastly, I will sit back down again and continue to breath into the mask for another hour while gases are collected. The final session will be a repeat of the early morning session where I must not eat for ten hours or exercise for 24, and sit quietly for one half hour and breathe into the mask.

My anonymity will be maintained by assigning me a code number upon entry into the study. All data will be recorded using the code number. The list containing the names of the subjects and their appropriate code numbers will only be available to the researchers in this study. I will not be identified in any way in the presentation or publication of the results of the study.

Questions about the research or any aspects of my participation in it should be directed to Carey Hilbert (345-6957). I understand that the University does not provide a research subject with compensation or medical treatment in the event the subject is injured as a result of participation in the research project.

I have been completely informed and understand the nature and purpose of the research project. The researcher has offered to answer any further questions that I may have. I understand that my participation in this study is completely voluntary and I may withdraw from the study at any time without prejudice or loss of benefits to which my participation entitles me.

I have read the foregoing and agree to participate in this study.

_________________________________________  ____________________________
Subject's Signature                      Date

_____________________________________
Subject's Address

_________________________________________  ____________________________
Investigator's Signature                  Date
Appendix B

DIET AND EXERCISE ASSESSMENT

YOUR HONESTY AND ACCURACY IN ANSWERING THESE QUESTIONS WILL BE EXTREMELY IMPORTANT. THANK YOU FOR YOUR PARTICIPATION. YOUR CONFIDENTIALITY IS ASSURED.

Age: ________ Height: ________ Weight: ________

Please refer to the following code when responding to #s 1 - 4:

A - always (7 times/week)
U - usually (5-6 times/week)
O - occasionally (3-4 times/week)
R - rarely (1-2 times/week)
N - never (0 times/week)

1. Do you eat breakfast? A U O R N (circle one)
   If so, please describe your most "typical" breakfast(s). Wherever possible, be specific to amounts (cups, oz, slices, etc.) as well as brand names ("Cheerios" etc.)

2. Do you eat lunch? A U O R N (circle one)
   If so, please describe your most "typical" lunch(es). Wherever possible, be specific to amount (cups, oz., slices) as well as brand names ("Yoplait", "Campbell's" etc.)

3. Do you eat dinner? A U O R N (circle one)
   If so, please describe your most "typical" dinner(s). Wherever possible, be specific to amount (cups, oz., slices) as well as brand names ("Domino's", "Good Season's" etc.)
4. Do you snack?  
A U O R N (circle one)

If so, please describe your most "typical" snack(s). Wherever possible, be specific to amount (cups, oz., slices) as well as brand names ("Doritos", "Hagen Daz" etc.)

5. For what length of time would you estimate you have been following this pattern of eating?

6. Do you engage in any regular exercise (at least 3 days per week)?  Yes  No

7. Please list types of any activity you engage in on a regular (≥3 p/w) basis, and indicate how often and the length of time per session at that activity.

8. How long would you estimate that you have been doing each of the above listed activities.

9. Given the following scale, please rate how hard you judge your exercise to be. If various activities correspond to various intensities, please indicate how often you work at a given intensity for each activity. (Circle most appropriate answer)

6 - 8 light, easy (i.e. casual walking)
9 - 11 moderately light (i.e. fast walking)
12 - 15 moderately hard (i.e. dancing, aerobics)
16 - 18 hard (i.e. competitive training)
19 - 20 very hard (i.e. all out to complete exhaustion)

10. Are you presently on birth control pills or taking shots for birth control?  Yes  No

11. Have you ever had an eating disorder (anorexia or bulimia)?  Yes  No

If yes, how long ago, and for how long?

If you are interested in participating further in a diet and exercise study, please fill in below:

name ___________________________ phone number (___)___________

address ________________________________________________________
Appendix C

MEDICAL QUESTIONNAIRE

Name ___________________________ Date ________________
Address __________________________ Phone ________________
Age _______ Height _______ Weight ________________
Date of last menstrual period _______ Number of days between periods ______

Circle the appropriate responses:

1. Have you smoked cigarettes regularly in the last 5 years?
   yes  no

2. Have you been diagnosed as having high blood pressure?
   yes  no

3. Have you ever been treated for "sugar diabetes" (diabetes mellitus)?
   never  with pills  with insulin injections

4. Have you, either of your parents, or any of your siblings had a "heart attack," bypass surgery, or other known heart problems prior to the age of 55 years?
   yes  no

6. Have you ever had an elevated blood cholesterol level (over 240 mg/dl)?
   do not know  no  yes

   Do you know your blood cholesterol level? __________ mg/dl

7. Do you ever get spells of dizziness or feel faint?
   yes  no

8. Have you ever been told you have a bone or joint problem that has been aggravated by exercise, or might be made worse with exercise?
   yes  no

9. List any prescription medications you are taking and give amounts.
Appendix D

EATING ATTITUDES TEST

Please place an (X) under the column which best applies to each of the numbered statement. All of the results will be strictly confidential. Please answer each question carefully. Thank you.

A=Always U=Usually O=Often S=Sometimes R=Rarely N=Never

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<td>( ) ( ) ( ) ( ) ( )</td>
<td>1. Am terrified about being overweight</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>2. Avoid eating when I am hungry</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>3. Find myself preoccupied with food</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>4. Have gone on eating binges where I feel I may not be able to stop</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>5. Cut my food into small pieces</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>6. Aware of the calorie content of foods that I eat</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>7. Feel that others would prefer I eat more</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>8. Vomit after I have eaten</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>9. Feel extremely guilty after I have eaten</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>10. Am preoccupied with a desire to be thinner</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>11. Exercise strenuously to burn off calories</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>12. Weigh myself several times a day</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>13. Have regular menstrual periods</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>14. Other people think that I am too thin</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>15. Am preoccupied with the thought of having fat on my body</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>16. Take longer than others to eat my meals</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>17. Avoid foods with sugar in them</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>18. Eat diet foods</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>19. Feel that food controls my life</td>
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<td>( ) ( ) ( ) ( ) ( )</td>
<td>20. Display self-control around food</td>
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A U O S R N

21. Feel that others pressure me to eat
22. Give too much time and thought to food
23. Feel uncomfortable after eating sweets
24. Engage in dieting behavior
25. Like my stomach to be empty
26. Enjoy trying rich new foods
Appendix E

The BULIT

Directions: Answer each question on the following pages by circling the appropriate letter. Please respond to each item as honestly as possible; remember, all of the information you provide will be kept strictly confidential.

1. Do you ever eat uncontrollably to the point of stuffing yourself (i.e. going on eating binges)?
   A) ONCE A MONTH OR LESS (OR NEVER)
   B) 2-3 TIMES A MONTH
   C) ONCE OR TWICE A WEEK
   D) 3-6 TIMES A WEEK
   E) ONCE A DAY OR MORE

2. I am satisfied with my eating patterns
   A) AGREE
   B) NEUTRAL
   C) DISAGREE A LITTLE
   D) DISAGREE
   E) DISAGREE STRONGLY

3. Have you ever kept eating until you thought you'd explode?
   A) PRACTICALLY EVERY TIME I EAT
   B) VERY FREQUENTLY
   C) OFTEN
   D) SOMETIMES
   E) SELDOM OR NEVER

4. Would you presently call yourself a "binge eater"?
   A) YES, ABSOLUTELY
   B) YES
   C) YES, PROBABLY
   D) YES, POSSIBLY
   E) NO

5. I prefer to eat:
   A) AT HOME ALONE
   B) AT HOME WITH OTHERS
   C) IN A PUBLIC RESTAURANT
   D) AT A FRIEND'S HOUSE
   E) DOESN'T MATTER

6. Do you feel you have control over the amount of food you consume?
   A) MOST OF THE TIME
   B) A LOT OF THE TIME
   C) OCCASIONALLY
   D) RARELY
   E) NEVER

7. I use laxatives or suppositories to help control my weight.
   A) ONCE A DAY OR MORE
   B) 3-6 TIMES A WEEK
   C) ONCE OR TWICE A WEEK
   D) 2-3 TIMES A MONTH
   E) ONCE A MONTH OR LESS (OR NEVER)

8. I eat until I feel too tired to continue.
   A) AT LEAST ONCE A DAY
   B) 3-6 TIMES A WEEK
   C) ONCE OR TWICE A WEEK
   D) 2-3 TIMES A MONTH
   E) ONCE A MONTH OR LESS (OR NEVER)

9. How often do you prefer eating ice cream, milk shakes, or puddings during a binge?
   A) ALWAYS
   B) FREQUENTLY
   C) SOMETIMES
   D) SELDOM OR NEVER
   E) I DON'T EVER BINGE

10. How much are you concerned about your eating binges?
    A) I DON'T BINGE
    B) BOTHERS ME A LITTLE
    C) MODERATE CONCERN
    D) MAJOR CONCERN
    E) PROBABLY MY BIGGEST CONCERN IN LIFE RIGHT NOW

11. Most people I know would be amazed if they knew how much food I can consume at one sitting.
    A) WITHOUT A DOUBT
    B) VERY PROBABLY
    C) PROBABLY
    D) POSSIBLY
    E) NO
25. What is the most weight you've ever lost in 1 month?
   A) OVER 20 LBS.
   B) 12 - 20 LBS.
   C) 8 - 11 LBS.
   D) 4 - 7 LBS
   E) LESS THAN 4 LBS.

26. If I eat too much at night I feel depressed the next morning.
   A) ALWAYS
   B) FREQUENTLY
   C) SOMETIMES
   D) SELDOM OR NEVER
   E) I DON'T EAT TOO MUCH AT NIGHT

27. Do you believe that it is easier for you to vomit than it is for most people?
   A) YES, IT'S NO PROBLEM FOR ME
   B) YES, IT'S A LITTLE EASIER
   C) ABOUT THE SAME
   D) NO, IT'S LESS EASY
   E) I DON'T VOMIT

28. I feel that food controls my life.
   A) ALWAYS
   B) ALMOST ALWAYS
   C) FREQUENTLY
   D) SOMETIMES
   E) SELDOM OR NEVER

29. I feel depressed immediately after I eat too much.
   A) ALWAYS
   B) FREQUENTLY
   C) SOMETIMES
   D) SELDOM OR NEVER
   E) I DON'T EAT TOO MUCH

30. How often do you vomit after eating in order to lose weight?
   A) LESS THAN ONCE A MONTH (OR NEVER)
   B) ONCE A MONTH
   C) 2 - 3 TIMES A MONTH
   D) ONCE A WEEK
   E) 2 OR MORE TIMES A WEEK

31. When consuming a large quantity of food, at what rate of speed do you usually eat?
   A) MORE RAPIDLY THAN MOST PEOPLE HAVE EATEN IN THEIR LIVES
   B) A LOT MORE RAPIDLY THAN MOST PEOPLE
   C) A LITTLE MORE RAPIDLY THAN MOST PEOPLE
   D) ABOUT THE SAME AS OTHER PEOPLE
   E) MORE SLOWLY THAN OTHERS (OR NOT APPLICABLE)

32. What is the most weight you've ever gained in one month?
   A) OVER 20 LBS
   B) 12 - 20 LBS.
   C) 8 - 11 LBS.
   D) 4 - 7 LBS.
   E) LESS THAN 4 LBS.

33. My last menstrual period was
   A) WITHIN THE LAST MONTH
   B) WITHIN THE PAST 2 MONTHS
   C) WITHIN THE PAST 4 MONTHS
   D) WITHIN THE PAST 6 MONTHS
   E) MORE THAN 6 MONTHS AGO

34. I use diuretics (water pills) to help control my weight.
   A) ONCE A DAY OR MORE
   B) 3 - 6 TIMES A WEEK
   C) ONCE OR TWICE A WEEK
   D) 2 - 3 TIMES A MONTH
   E) ONCE A MONTH OR LESS

35. How do you think your appetite compares with that of most people you know?
   A) MANY TIMES LARGER THAN MOST
   B) MUCH LARGER
   C) A LITTLE LARGER
   D) ABOUT THE SAME
   E) SMALLER THAN MOST

36. My menstrual cycles occur once a month
   A) ALWAYS
   B) USUALLY
   C) SOMETIMES
   D) SELDOM
   E) NEVER
12. Do you ever eat until you feel sick?
   A) VERY FREQUENTLY
   B) FREQUENTLY
   C) FAIRLY OFTEN
   D) OCCASIONALLY
   E) RARELY OR NEVER

13. I am afraid to eat anything for fear that I won't be able to stop.
   A) ALWAYS
   B) ALMOST ALWAYS
   C) FREQUENTLY
   D) SOMETIMES
   E) SELDOM OR NEVER

   A) ALWAYS
   B) FREQUENTLY
   C) SOMETIMES
   D) SELDOM OR NEVER
   E) I DON'T EAT TOO MUCH

15. How often do you intentionally vomit after eating?
   A) 2 OR MORE TIMES A WEEK
   B) ONCE A WEEK
   C) 2-3 TIMES A MONTH
   D) ONCE A MONTH
   E) LESS THAN ONCE A MONTH (OR NEVER)

16. Which of the following describes your feelings after binge eating?
   A) I DON'T BINGE EAT
   B) I FEEL OKAY
   C) I FEEL MILDLY UPSET W/ MYSELF
   D) I FEEL QUITE UPSET W/ MYSELF
   E) I HATE MYSELF

17. I eat a lot of food when I'm not even hungry.
   A) VERY FREQUENTLY
   B) FREQUENTLY
   C) OCCASIONALLY
   D) SOMETIMES
   E) SELDOM OR NEVER

18. My eating patterns are different from patterns of most people.
   A) ALWAYS
   B) ALMOST ALWAYS
   C) FREQUENTLY
   D) SOMETIMES
   E) SELDOM OR NEVER

19. I have tried to lose weight by fasting or going on crash diets.
   A) NOT IN THE PAST YEAR
   B) ONCE IN THE PAST YEAR
   C) 2-3 TIMES IN THE PAST YEAR
   D) 4-5 TIMES IN THE PAST YEAR
   E) 5+ TIMES IN THE PAST YEAR

20. I feel sad or blue after eating more than I planned to eat.
   A) ALWAYS
   B) ALMOST ALWAYS
   C) FREQUENTLY
   D) SOMETIMES
   E) SELDOM, NEVER, OR NOT APPLICABLE

21. When engaged in binge eating, I tend to eat more starches and sugar.
   A) ALWAYS
   B) ALMOST ALWAYS
   C) FREQUENTLY
   D) SOMETIMES
   E) SELDOM OR NOT APPLICABLE

22. Compared to most people, my ability to control my eating behavior seems to be:
   A) GREATER THAN OTHER'S
   B) THE SAME AS OTHERS
   C) LESS THAN OTHERS
   D) MUCH LESS THAN OTHERS
   E) I HAVE NO CONTROL

23. One of your best friends suggests you both eat at a new buffet restaurant tonight. Although you'd planned on eating something light at home, you go and eat a lot, feeling uncomfortably full. How would you feel about yourself?
   A) FINE, & GLAD I'D TRIED A NEW RESTAURANT
   B) REGRETFUL I'D EATEN SO MUCH
   C) DISAPPOINTED IN MYSELF
   D) UPSET WITH MYSELF
   E) TOTALLY DISGUSTED WITH MYSELF

24. I would presently label myself a "compulsive eater"
   A) ABSOLUTELY
   B) YES
   C) YES, PROBABLY
   D) YES, POSSIBLY
   E) NO, PROBABLY NOT
24-Hour Recall Form

Courtesy of the Human Nutrition Information Service, USDA.

This record is for: ________________________  
PERSON'S FIRST NAME

This person's date of birth is: ___ ___  
MONTH  DAY  YEAR

DAY ONE is from 12:00 AM to 11:59 PM yesterday. That date was: ___ ___  
MONTH  DAY  YEAR

(CIRCLE NUMBER FOR DAY OF WEEK)

Sunday 1  
Monday 2  
Tuesday 3  
Wednesday 4  
Thursday 5  
Friday 6  
Saturday 7

Your cooperation is entirely voluntary. This information will be used to estimate the types and amounts of foods and beverages consumed by people like you. Results will be used to help ensure an adequate and safe food supply for all. This survey is authorized by law. (If asked, ask: National Agricultural Research, Extension and Teaching Policy Act of 1977, Section 1428, 7 U.S.C. 3178.)

All information will be kept confidential and will be reported as statistics only.
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Appendix G

Table 7.
Individual VO$_{2\text{max}}$ Results for Normal and Low Calorie Subjects.

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Table 8.
Dosage Levels of Various Oral Contraceptives Used by Subjects.

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<td>3) 1 mg norethindrone; .035 mg ethinyl estradiol</td>
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<td>Tri-Levlen</td>
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### Table 9.

Trial 1 Mean RMR and Estimated Menstrual Cycle Phase for Normal and Low Calorie Subjects.

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<th>RMR 1 (KCAL/60 min)</th>
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* Menstrual phase not established; F - follicular phase; L - luteal phase
Table 10.

Trial 2 Mean RMR and Estimated Menstrual Cycle Phase for Normal and Low Calorie Subjects.

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* Menses not established; # Menses began ~10 hrs. post-test
F - follicular phase; L - luteal phase
Appendix K

Table 11.
Individual Recovery VO₂ and Energy Expenditure for Normal and Low Calorie Subjects.

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>TOTAL RECOVERY (L/60)</th>
<th>EPOC (L)</th>
<th>EPOC (L/min)</th>
<th>ENERGY EXPENDITURE (kcal/60)</th>
<th>NET RECOVERY ENERGY EXP (kcal)</th>
<th>ELEVATION LAST 5 MIN (L/min)</th>
<th>RMR I (L/min)</th>
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<tbody>
<tr>
<td><strong>NORMAL CALORIE</strong></td>
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<td>4.40</td>
<td>0.22</td>
<td>64.07</td>
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# Appendix L

Table 12.

Five Minute Averages for EPOC for Normal and Low Calorie Groups.

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<tr>
<th>TIME</th>
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<th>NC</th>
<th>LOW CALORIE</th>
<th>LC</th>
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<td>ml · kg⁻¹ · min⁻¹</td>
<td>L · min⁻¹</td>
<td>ml · kg⁻¹ · min⁻¹</td>
<td>L · min⁻¹</td>
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<tr>
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<tr>
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<td>0.22</td>
<td>3.69</td>
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</tr>
<tr>
<td>min 26 - 30</td>
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<td>0.22</td>
<td>3.69</td>
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<tr>
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<tr>
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<td>0.16</td>
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