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Appetite, Catecholamine, Hunger and Resting Metabolic Rate Responses
of
Trained and Untrained Females

A Dissertation
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Doctor of Philosophy
Degree
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Judith Ann Flohr

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DEDICATION

To my loyal and faithful companions, Willie, Crawford, M.J., J.J., Puff, Spooker and G.B.

To the spirit and life of Marilyn Crawford, to the heart and soul of Rosie Rummel and to the love and confidence of Anne Gabbard Alley.

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ABSTRACT

The body weight and composition manifested by an individual at any point in time represents the cumulative effect of the energy intake and expenditure by that individual. The amount of weight lost or gained by an individual is related to his or her ability to maintain a state of negative or positive energy balance. Excessive weight gain will most likely be exhibited by excessive quantities of subcutaneous adipose tissue. Maintaining high levels of body fat can be detrimental to an individual's health. In recent years there has been a heightened interest in the interaction of exercise, nutrition and health. Simultaneous to the increased sensitivity to the role nutrition plays in health has been an increased appreciation for the the role it may play in physical performance. The number of females participating in physical activity for health and competitive reasons has also risen in the last twenty years. In spite of the increased participation by females, the majority of exercise and nutrition studies have used male subjects. Due to the physiological differences between males and females it is no longer acceptable or prudent, to extrapolate the data from males to females. The energy intake and expenditure by active females will determine the state of energy balance. Maintaining a positive or negative energy balance for an extended period of time will have significant consequences for performance. Exercise has been known to produce a hypophagic effect in some males and females. Circulating catecholamines have been proposed as a potential mechanism to explain this response. It has been suggested that female athletes chronically maintain an energy expenditure greater than their energy intake, without significant losses of body weight. Significant changes in resting metabolic rate and/or energy intake may explain the ability of some female athletes to maintain weight in spite of the energy deficits created by the high caloric demands of training. Hunger, appetite (food intake), and urinary catecholamine (epinephrine, E and norepinephrine, NE) responses to an acute bout of exercise and control session were studied in trained female runners (R) and swimmers (S). Resting metabolic rate (RMR) of the trained (T) subjects was compared to untrained (UT) sedentary controls (C). Mode did not influence the hunger or catecholamine response of the trained females. Hunger increased with time after the exercise session. Correlation coefficients indicated there was a significant relationship between the post exercise hunger ratings and circulating levels of NE. In addition NE was a significant predictor of appetite (food intake) following exercise, but not the control session. Hunger was not related to food intake following control or exercise session.

There were no differences in food intake across sessions or groups. Resting metabolic rate expressed as kcal/ kg LBW was lower in trained (T) females as compared to untrained (UT) controls. Hunger and appetite responses appear to be dissociated in female runners and swimmers. Appetite appears to be a function of a multitude of physiological, psychological and environmental stimuli. The role any one stimulus plays in the appetite response of the female is not clearly defined at this time. Female runners and swimmers resting metabolic rate may reflect an adaptation to chronic levels of low energy intake against high energy expenditures.

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CHAPTER I

Introduction

A person is said to be in energy balance when energy intake is equal to the energy expenditure, or when the caloric intake is equal to the caloric expenditure. A person in positive energy balance has a greater energy intake than expenditure; the reverse is true for someone in negative energy balance. A state of positive energy balance will lead to a gain in body weight; a negative energy balance will lead to a loss of body weight. Energy balance has consequences for physical performance as well as an individual's health. Various physical performances may be enhanced or hindered by the individual's ability to lose, maintain or gain weight. Furthermore, as the frequency and severity of adult onset obesity and childhood obesity continue to increase in the United States, it becomes paramount that methods are developed to control and prevent a positive energy balance in both children and adults [1, 2]. Additionally, it appears that those individuals who are carrying excess weight are carrying it as fat, which can lead to a variety of potential health problems [2]

Energy balance is related to the individual's tendency to maintain, gain or lose weight. It has been suggested that female endurance athletes chronically maintain an energy expenditure greater than their energy intake, yet significant weight losses are not experienced by these women [3-6]. However, it is important to recognize that the methods that are often utilized to estimate energy balance do not always account for the influence that exercise training may have on other aspects of the energy balance equation [4]. Furthermore, an editorial by Durnin eluded to the fact that the research in the area of energy expenditure and intake must be interpreted with a great deal of caution due to the lack of precision and control of the subject populations as well as the methodologies

utilized [7].

There are various factors which may significantly influence the female athlete's energy intake and expenditure. Resting metabolic rate (RMR), the thermic effect of feeding (TEF) and the thermic effect of activity (TEA) are all factors which will influence the energy expenditure side of the energy balance equation. The athlete's resting metabolic rate is the most significant factor in the energy balance equation; yet, there are conflicting reports on the impact that training has on the RMR of females [8-10].

There is some evidence that training elicits a reduced thermic effect of food [11-13]. Additionally, there continues to be debate as to the influence exercise has on hunger, appetite (food intake) and perhaps more importantly, that the response may differ for males and females. Moreover, exercise may exert an acute effect on the individual's perception of hunger, which may or may not influence the energy intake for that individual [14]. Furthermore, it is unclear what impact the mode and intensity of the activity may have on the perception of hunger or appetite immediately following a bout of exercise. The elevated catecholamine response and body temperature are mechanisms that have been proposed for the diminished food intake and appetite following exercise in male rats and humans [15, 16]. Obviously, the mode of activity or exercise can have a significant impact on the temperature and catecholamine response which then may indirectly influence the subject's postexercise appetite and food intake. Additionally, there are a number of reports that imply that the post exercise appetite or hunger response may not have an impact on the food intake subsequent to exercise [14, 17-19].

Resting metabolic rate represents the largest portion of total energy expenditure by sedentary humans and constitutes 60 to 75% of the total energy expenditure in humans. [20]. However, what is uncertain is exactly what fraction the RMR represents for

physically active females, specifically, female endurance athletes [10]. It is well established that there is a high correlation between RMR and fat free mass in both males and females [20]. Increases in fat free mass are normally experienced by both males and females as a result of physical training. Greater gains are normally achieved as a result of resistance training in contrast to endurance training. Yet it remains to be determined whether or not the type of endurance training has an influence on the RMR and whether the gains in lean body mass in females will in fact stimulate an increase in RMR.

There are a variety of factors that may influence the degree of change that a female athlete experiences in RMR as a result of endurance training. The mode of training, the change in body temperature, the degree of weight bearing activity, intensity of training, duration of training, cessation of training, and muscle damage are all potential factors that may indirectly influence the energy balance equation.

There is little evidence describing an elevated RMR in females due to chronic physical training, as has been reported for males [9, 10]. However, it is of practical significance for the athlete to be cognizant of changes in RMR due to the training itself or a reduction or cessation of training, especially if the change in RMR has a significant influence on the energy balance equation. In some cases the ability of the athlete to perform at his/her physical potential may be related to body weight and body composition. Small weight gains by athletes whose performance is indirectly related to body weight may be an advantage or disadvantage depending on their activity. For example, it is a disadvantage for the cross country runner to carry excess amounts of fat or body weight in general as this will increase the caloric cost per mile and cause them to run at a high percentage of VO_2 max.

Specifically, it is common for athletes to reduce training duration and intensity for weeks or days prior to special competitive events. In a recent investigation by Tremblay [21], trained male cyclists experienced a 6.6% decrease in RMR as a result of a three

day interruption in training. It is not known whether a cessation or reduction in training has a similar impact on the RMR of trained female athletes.

There are, thus, a number of questions that remain to be answered with regard to the energy balance equation for trained female athletes. The questions are of interest as the female athlete is a unique model, but perhaps more importantly, the female athlete may serve as a model that will help to answer questions about energy balance in all females. Understanding the intricacies of energy balance will assist coaches and health practitioners to design appropriate training and exercise and weight management programs for all females.

The number of female athletes and females participating in physical activity for the purposes of recreation and /or health is increasing. In spite of this, the majority of nutrition and exercise studies that have been conducted have utilized male subjects. Due to the physiological differences between males and females it is imperative that practitioners and researchers understand whether these differences preclude a variable response to exercise and diet. It is no longer acceptable or prudent to extrapolate the data from male subjects to females.

Therefore, this study was designed to examine the role hunger, appetite and resting metabolic rate play in the energy balance equation of trained females. The specific purposes of this study were; (a) to determine the influence mode of training had on the hunger response following an acute bout of exercise; (b) to determine the relationship between the hunger response and urinary catecholamine levels; (c) to determine if the hunger and/or urinary catecholamines responses were related to subsequent 24 hour food intake and (d) to determine if there were differences in RMR between trained and untrained females and trained female runners and swimmers.

CHAPTER II

Review of Literature

Introduction

Superficially, maintenance of the energy balance equation appears to be somewhat simplistic. That is, the majority of adults and athletes are able to maintain a stable body weight simply by balancing caloric intake with caloric expenditure. However, there are numerous adults and children who have difficulty maintaining energy balance; this is reflected in the current prevalence of adult onset obesity and the rampant increase of childhood obesity in the past twenty years [1].

There are several components of the energy balance equation; energy or food intake is only one side of the equation. Conversely, the expenditure side of the equation is determined by the individual's resting metabolic rate, the thermic effect of feeding and the thermic effect of activity (work and exercise). The relationship between energy intake and exercise is often difficult to determine due to the frequent misuse of terminology in the literature. The terms appetite, food intake and hunger are often incorrectly interchanged; each term has its own definition and importance to the energy balance equation. Most authors will define hunger as an internal state or level that disposes an animal or person to eat [22]. Appetite has been defined as the state of willingness to accept food or the desire to satisfy natural needs of the body [22]. Appetite (food intake) is the actual energy intake by the person or animal and is usually reported as the number of kilocalories consumed for a twenty four hour period [22]. An accurate assessment of energy intake or food intake is difficult due to the variety of physiological and psychological factors that influence the individual's perception of hunger and

appetite. Nevertheless, because energy intake is a significant component of the energy balance equation and can be manipulated, it is useful to understand how energy expenditure, specifically through exercise, may influence energy intakes in animals, trained athletes and the obese individuals.

Theories on Appetite

While it is understood that the long term maintenance of body weight is a result of physiological and behavioral mechanisms, the following section will focus entirely on the potential physiological controls of appetite. In light of the constancy of body weight over time and the short term changes in food intake, a two component regulatory system has been proposed for eating and maintenance of body weight. The short term component controls the onset and cessation of feeding for a given meal. Conversely, the long term component monitors the change and rate of change in the nutrient depots of the body [23]. Thus, the long term component enables humans to regulate body weight within a fairly narrow range.[24, 25]. It is postulated that the short term control, or the nature of the satiety signals, is either increased or decreased in accord with the long term component, which reflects the relative state of the nutrient depots of the body.[26].

Feeding is stimulated by a variety of external and internal cues to reflect a need for energy within the body. The caloric intake for a given meal may be in excess of what is actually needed; the excess is normally stored in the form of fat. Furthermore, in the adult, body weight changes are normally manifested by changes in the quantity of body fat. In unusual circumstances, large changes in body weight may also be related to changes in a person's lean body mass. Therefore, it is generally agreed that body fat is the primary tissue being regulated on a long term basis. The fat deposits of the body are considered to be very active metabolically, and will send metabolic or neuronal signals to reflect their size. The signals are believed to be sent to the brain where the output is compared to a reference point. Depending on the "error" of the signal, a variety of

mechanisms are activated to change the feeding signals [23]. The signals that ultimately converge to regulate appetite and body weight are more than likely integrated from a number of sites and tissues throughout the body.

The nutrient levels of a meal, and/or the distention of the gut, are potential signals that are monitored in the short term control of feeding. Theoretically, signals are sent from the gut to the brain during feeding to either stimulate or inhibit the feeding process [23]. Thus, the brain is believed to be actively involved in both long term and short term regulatory components. In recent years, an integrative hypothesis of how the brain regulates food intake has been formulated. The brain is believed to contain specific centers or areas that are sensitive to catecholamine neurotransmitters. Specifically, it is believed that feeding and satiety are modulated by the amount of catecholamine activity in various areas of the brain. The depletion of norepinephrine in specific brain areas has been shown to cause a decrease in daily food intake in rats [27]. Conversely, the infusion of a meal into the gut has been shown to cause changes in the levels of the neurotransmitters which then alter the feeding response [28].

In addition to the brain, the gastrointestinal track is implicated in the short term control of feeding, primarily in the areas of satiety or the inhibition of the feeding response. The signals from the gut are both neural and humoral. Glucoreceptors exist in the intestine which are known to send signals via the vagus nerve. In addition, receptors are believed to exist for the hormone cholecystokinin (CCK). There are a number of other brain-gut peptides that have been identified; as of yet the specific mechanisms of action have not been elucidated [23].

The liver is yet another organ that is involved in the short term regulation of feeding and body fat stores. The liver ultimately processes all the substrates of the body, thus, it is quite reasonable to assume it would have an active role in the regulation of appetite and body weight. The overall metabolic status of the liver is sensed by branches of the

vagus nerve which then relay the information to the brain. Thus, depending on the metabolic status of the liver, feeding will either be stimulated or inhibited [23].

The adipose tissue of the body is believed to be a potential site for the long term regulation of appetite and body weight. The lipostatic hypothesis is based on the underlying premise that adipose tissue regulates its own size via signals it sends to the brain which then influence the feedback upon itself. Unfortunately, the nature of the signals that are elicited by body fat have not been identified. Brobeck [29] contends that the activity of the adipose tissue serves to modulate the signals that are being transmitted by other organs due to the ingestion of food. The signals that have been suggested are serum levels of glycerol and free fatty acids. However, conclusive evidence for the regulatory function of these metabolites does not exist at the present time.

Despite the lack of a conclusive link between adipose tissue and the long term regulation of body weight, the levels of body fat are postulated to play an important role in the endocrine and autonomic nervous system's regulation of body weight. The role the fat stores play in regulating their size may be related to the activity of the autonomic nervous system. Specifically, Bray [30] postulates that a high amount of sympathetic nervous system activity is associated with low body fat stores and that high stores of body fat are often associated with lowered amounts of sympathetic nervous system activity.

The levels of insulin that pass through the blood brain barrier into the cerebrospinal fluid (CSF) are theorized to be influential in the long term maintenance of body weight [31]. According to the hypothesis, there is a positive correlation between the circulating levels of insulin in the cerebrospinal fluid and body fat stores. Specifically, a predictable fraction of circulating insulin will pass into the cerebrospinal fluid. The circulating levels of insulin provide the brain with information as to the size of the body

fat stores. Thus, if CSF levels of insulin increase this is a reflection of increasing adiposity; this in turn causes an inhibition of the feeding centers. Conversely, if the CSF insulin level decreases, the feeding centers are stimulated [32, 33].

In addition, the endocrine system is believed to interact with the nervous system and peripheral metabolic processes to regulate appetite and body weight. This hypothesis is sometimes referred to as the autonomic-endocrine hypothesis. In essence, the hypothesis states that lateral and medial portions of the hypothalamus are closely involved with the sympathetic and parasympathetic stimulation of insulin release, hepatic glycogen synthesis, fatty acid mobilization and peripheral norepinephrine turnover [33].

Theories on Exercise and Hunger

The liver has a critical role in modulating the state of the body's energy stores. "Hunger is believed to arise when the fuel delivery from the intestines and adipose tissue is inadequate for the maintenance of body functions without significant hepatic contributions." [34, p. 423]. This theory suggests that the brain receives information from the liver that endogenous sources of energy, primarily fat, are being utilized in place of readily available fuels to supply energy for various metabolic processes. Support for this theory has been provided by the measurement of increased food intake following liver glycogen depletion and increased serum free fatty acid levels.[35]. Exercise is known to cause an elevated fuel utilization; thus, in accordance with this theory, one should expect an increase in hunger following exercise. Despite the changes in energy utilization, several studies have shown that exercise precipitates a decreased hunger response [14, 36-41].

In a study by Maxwell [42], hunger was suppressed for approximately two hours following exercise of varying intensity and duration. Subjects completed four different treatments: control, no exercise (CN), exercise at 30% VO_2 max with energy

expenditure equivalent to 300 kcal (LE), exercise at 60% VO_2 max with an energy expenditure equivalent to 300 kcal (LH), and exercise at 60% VO_2 max short duration (SH). Each treatment was preceded by an overnight fast. When the overnight fast was extended by five hours, the subject's perception of hunger increased as did the serum levels of free fatty acids while the respiratory exchange ratio (RER) decreased. Both of these metabolic changes indicated a greater reliance on fat as the primary energy source. The subject's blood glucose did not change significantly as the level of hunger increased indicating active hepatic glycogenolysis or gluconeogenesis. The increased hunger in response to greater fat utilization is in agreement with Friedman's metabolic theory of hunger [34]

It was expected that the hunger ratings would be elevated following the exercise sessions and highest with the exercise causing the greatest energy expenditure. Surprisingly, this was not the case, although the RER and FFA levels indicated high rates of fat utilization. The hunger ratings were significantly depressed two hours following exercise for all exercise bouts. Serum free fatty acid levels and respiratory exchange ratio (RER) were measured throughout the recovery and exercise periods. The intensity and duration of exercise did not appear to have an impact on the reduction in hunger until 20 minutes post exercise. Twenty to sixty minutes post exercise, the hunger ratings were most affected by a combination of higher intensity and extended duration (LH). The intensity of exercise did not have an independent effect on hunger as the short duration exercise at 60% VO_2 max did not elicit a similar response. These findings suggested that perhaps there are additional factors associated with exercise which may override the normal signals associated with increased hunger and food intake during resting conditions.

Potential Mechanisms for the Hypophagic Effect of Exercise

The early reports suggested that acute bouts of exercise had a short term hypophagic

effect on appetite, however, the physiological mechanisms for the hypophagia are not clearly understood. Several factors have been proposed including increased plasma catecholamines [16, 43], increases in lactate [44], increases in core temperature [15, 45], and the production of an anorexigenic factor [36].

Belbeck et al.[36] proposed that the suppression of appetite following exercise was due to the increase of an anorexigenic (glycopeptide) agent in the plasma. The agent was originally identified by Stevenson et al.[46] in male rats. The presence of an anorexic agent in humans in response to exercise was examined by Belbeck et al.[36]. In the control period blood samples were obtained from seven men in the pre absorptive state, and sixty and ninety minutes post absorptive. Twenty four hours following the control measurements the subjects participated in exhaustive treadmill walking. Sixty and ninety minutes following the treadmill exercise venous blood samples were drawn. The plasma concentration of an anorexigenic substance remained constant throughout the control period. Following the treadmill exercise the plasma concentration of the anorexigenic substance increased significantly. The mean increase in the anorexic agent was 21%. Each subject manifested an increase, but the magnitude of the increase was variable across subjects. The injection of the anorexic substance identified by Stevenson [46] depressed food intake in rats by 50% for 24-48 hours. Belbeck et al.[36] speculated that an anorexigenic substance may be a factor responsible for the suppression of appetite immediately following exercise in humans. Unfortunately, there appears to be a lack of additional measurements of an anorexigenic substance in humans in response to exercise. Thus, while it is tempting to speculate that there may be such an agent responsible for the suppression of appetite frequently noted following exercise, adequate verification does not exist.

An association between an elevated core temperature and reduced food intake has been suggested by a number of authors [15, 42, 45, 47-50].

In the investigation by Maxwell [42], the rectal temperature of male subjects exposed to a variety of exercise protocols was obtained. A consistent reduction in the hunger response was observed as the core temperature increased above resting levels. The hunger ratings were the lowest during the first 90 minutes of recovery following the exercise bout that elicited the highest core temperature. However, it is important to note that there were no significant differences between the exercise treatments for hunger ratings; thus, this would imply that there are additional factors related to exercise that may reduce hunger.

Elevated catecholamine levels have also been suggested as a potential appetite suppressant. Catecholamine levels measured after exercise provide strong evidence as to the intensity of the exercise and the magnitude of change in metabolism in response to exercise. Specifically, the changes in the catecholamine norepinephrine provide indications as to the amount of sympathetic nervous system activity that occurred during exercise. It is not unusual for the norepinephrine concentration to increase by 600% during exercise, and although the epinephrine concentration does not change to the same magnitude, (increases 200%) it is a very powerful hormone. The changes in the catecholamines during exercise are directly related to the level of fuel mobilization from the liver and adipose tissue as well as the cardiovascular adjustments to an exercise bout. Catecholamines levels have also been linked to changes in food intake following exercise. Intraperitoneal injections of epinephrine have caused a decreased food intake in the animals [51].

The potential role of epinephrine in the hypophagia often demonstrated after exercise was recently examined by Guillard et al. [52]. Male rats were subjected to a nine week progressive swimming program that started with ten minutes of swimming per day and increased to sixty minutes per day. Urinary levels of norepinephrine and epinephrine and food intake were measured throughout the nine weeks. Catecholamine levels

increased significantly in response to exercise ranging from an increase of 38% to 70%. The food intake of the rats decreased in response to exercise, notably there was a significant positive correlation between the increase in urinary catecholamine levels and the decreased food intake. Guillard et al. [52] concluded that the increased catecholamine levels due to exercise were directly related to the reduced food intake and the depression of body weight gain in male rats.

The results of Guillard et al. [52] appear to support the theory that catecholamines may have a role in the control of food intake and body weight. Metabolically, catecholamines have a variety of effects that can in fact inhibit food intake; these are: (a) inhibition of gastrointestinal motility [53], (b) stimulation of hepatic glycogenolysis [54], and (c) mobilization of fat stores [55]. The mechanism responsible for reducing food intake via increased catecholamines is not completely clear. Nevertheless, one is tempted to speculate that epinephrine's ability to stimulate hepatic glycogenolysis [55] may contribute to a hypophagic response. Evidence to support this concept is derived from a study by Langhans et al. [56] that demonstrated propranolol's ability to block the anorectic effect of epinephrine.

Norepinephrine's role as an anorectic agent is related to its ability to stimulate lipolysis. Thus, it could be that, indirectly, norepinephrine acts to reduce food intake by stimulating lipolysis which then elicits a hypophagic response. Three important observations support the anorectic effect of norepinephrine: (a) norepinephrine generates a persistent elevation in free fatty acids [52, 57], (b) chronic infusions of norepinephrine in rats stopped weight gain [54], and (c) increased circulating levels of norepinephrine in response to exercise retarded body weight gains and food intake in rats [52, 58]. The linkage of catecholamines, exercise, reduced food intake and the control of body weight in humans has yet to be determined.

Acute Effect of Exercise on Hunger and Appetite (Food Intake)

Chronic exercise has been reported to cause increases, decreases and no change in the food intake of rats and humans [1, 17-19, 38, 40, 45, 59-65]. In contrast, there is a shortage of information which addresses the effect of acute exercise on hunger and post exercise food intake [14, 36, 37, 39-41]. Obviously, the energy intake pattern of an athlete will have a significant impact on whether or not he/she is in energy balance, surplus or deficit. Therefore, it may be useful to examine the relationship between the subject's post exercise rating of hunger and the subsequent food intake or appetite.

Jankowski and Foss [39] measured the 24 hour food intake of 14 sedentary men ages 21-30 years old. Each subject completed a 440 yard and one mile run twice during the study. Caloric intake was monitored via a carefully controlled food weighing technique. The subjects' 24 hour food intakes were evaluated for four days prior to engaging in the exercise and 24 hours following each exercise bout. Neither exercise bout had a significant effect on the subsequent 24 hour caloric intake. It is difficult to draw any conclusions from this study due to the brevity of the exercise bouts.

Unfortunately, due to the subjective nature of hunger, few studies have examined the hunger response following exercise. It is also important to realize that attempts to quantify hunger, or appetite (food intake) may themselves influence the subject's response.

Despite these difficulties, Reger et al. [41] measured the hunger and appetite response of untrained females prior to and following various treadmill protocols. Subjects engaged in four different experimental protocols: (a) (60C) 60 minutes of continuous treadmill exercise at 50% VO_2 max, (b) (30C) 30 minutes of continuous treadmill exercise, (c) (30I) 30 minutes of intermittent treadmill exercise (1 minute at 70% VO_2 max and 3 minutes at 40% VO_2 max) and (d) no exercise. Using a 5 point scale ranging from zero (acute pains, acute pangs, weak, dizzy) to four (4) (full, no hunger sensations), each subject was asked to rate her hunger prior to exercise,

immediately following exercise , one hour and two hours post exercise.

The 30I exercise protocol was associated with a reduced hunger response immediately following exercise. However, at one hour post exercise there were no significant differences in the subjects' perception of hunger across exercise protocols. It is significant to note that Reger et al. [41] also measured appetite. For the purposes of their study appetite was defined as a measure of the subject's desire to eat a specific food stimulus. Subjects rated their appetite on a 7 point scale ranging from 1 (indifferent to food) to 7 (strong craving). The appetite rating of the subjects was reduced following the 30I and 30C trials. Although the subjects' subjective ratings of hunger and appetite were reduced following exercise, the caloric content of the test meal, food intake record , or total calories did not vary significantly across the four experimental protocols [41].

The results are somewhat encouraging in that, although the exercise protocols did not produce a chronic reduction in hunger or appetite, the 60C and 30C exercise bouts did cause a slight, yet statistically insignificant, reduction in food intake (128 kcal/day) 48 hours post exercise. A reduction of 128 kcal/day in energy intake coupled with an additional 218 kcal in energy expenditure due to the treadmill exercise amounts to a deficit of 397 kcal/day. Superficially, a deficit of 397 kcal would not appear to be instrumental in weight loss. However, were a subject to perform 60 minutes of low intensity treadmill walking every other day, this would amount to a loss of approximately 0.75 kg (1.65 lbs) of body fat per month. Moreover, this reduction, although small, could account for a loss of approximately 9 kgs (20 lbs) annually. Body fat losses of 9 kilograms per year would assist patients and health practitioners in the battle against "creeping obesity " that is so often experienced by middle aged adults.

Weight management is not simply a problem of middle aged adults: there are a number of athletic performances that can be hindered by a lack of control of body weight. Specifically, there is a tendency for athletes to gain fat in the "off" season which must be

lost before the next competitive season if one hopes to regain prior levels of performance. Thus, if exercise helps reduce the subject's perception of hunger this may influence the total calories that are consumed. Furthermore, it is tempting to speculate that the timing of exercise and meals may serve a viable role in the weight control process. That is, if exercise reduces a person's hunger, perhaps the caloric value of the subsequent meal will be reduced.

The impact of exercise intensity on the hunger response was examined by Thompson [14], who measured hunger ratings of male subjects following steady state cycling. Subjects participated in three experimental protocols: cycling at a low intensity (35% VO_2 max), cycling at high intensity (68% VO_2 max) or resting. The total energy expenditure for the cycling was equivalent in all groups and was set at approximately $4.1 \text{ kcal} \cdot \text{kg}^{-1}$ body weight. Subjects were asked to rate their hunger prior to exercise, at 5, 20, 35, 50 minutes following exercise, and following a post exercise meal. The subjects rated their hunger on a nine point paper and pencil scale ranging from 1 (extremely full) to 9 (extremely hungry). The authors used the hunger rating as an index of the subject's drive to eat. The results indicated that only the high intensity exercise had a significant effect on the subject's perception of hunger. However, the significant reduction in hunger following high intensity exercise was only present at 5 minutes post exercise and was absent after 20 minutes of recovery. Thus, although the high intensity exercise caused a reduced hunger response, it was a very acute reduction that did not significantly influence the volume or composition of the post exercise meal.

Even though high intensity activity caused a slight reduction in post exercise hunger and appetite, this level of exercise may or may not have a significant impact in a weight reduction regime. Perhaps activity of a greater duration or intensity may be useful in the suppression of hunger. Nevertheless, the suppression of hunger by itself will not be useful or significant in a weight reduction program unless it can be linked to a reduction

in appetite or food intake, as was shown by Reger et al. [41]. The potential anorexigenic effect of intense physical activity was illustrated in a simulation study conducted by Brouns et al. [37]. The subjects cycled for periods equivalent to various stages of the Tour de France bicycle race. The experimental duration and intensity were at levels only highly trained athletes were capable of maintaining. The exercise days were alternated with rest days. There was a significant reduction in food intake following the cycle activity. Total food intake for the subjects on the exercise days was significantly lower than on the rest days [37]. The design and findings of this particular study will be discussed in greater detail later in this review. Several factors have been proposed as potential underlying mechanisms for the anorexigenic effect elicited by exercise.

Chronic Changes in Appetite with Exercise (Animal)

The majority of the information that has been accumulated regarding energy balance in animals has been conducted using male and female rats. The classic study performed by Mayer [66] and colleagues was one of the first to carefully examine the role regular exercise had in food intake. Prior to the investigation by Mayer, it was assumed that obese individuals who engaged in exercise would increase their food intake to match the energy expenditure during exercise.[66].

The rats in Mayer's study were adult female rats, considered homogeneous with respect to age and weight. The rats were divided into two groups, an exercise group and a sedentary group. A variety of treadmill protocols were used to determine the relationship between length of exercise and subsequent food intake. Rats were engaged in treadmill running protocols of 20, 40, 60 minutes, and 1-2, 3-6 and above 6 hours. The food intake of the previously sedentary rats exercising 20 to 60 minutes decreased in the course of the study. However, for those rats exercising for durations of one to six hours, the food intake increased in proportion to the energy expenditure.

The results of this study were used to develop and define the classic energy intake

versus energy expenditure curve. The curve indicated that there were essentially three ranges of activity: sedentary, normal, and exhaustion. Mayer considered the sedentary and exhaustion ranges as zones of "unresponsiveness". Specifically, there was a point where a decrease in activity was not matched with a decrease in energy intake [66]. Additionally, there was also a range of excessive energy expenditure (greater than 6 hours) that was not matched with a concomitant increase in energy intake. The caloric intake matched the caloric expenditure in activity ranges of one to six hours. It is important to note that although caloric intake increased in response to exercise greater than one hour in duration, the exercising rats were able to maintain body weight below their sedentary counterparts. The authors cited their results as evidence indicating the value of exercise in weight control and perhaps prevention of obesity. When interpreting these results, it is very important to realize that the animals used were females, as later reports would indicate that there is a gender difference in the relationship between exercise and appetite [45, 67].

An accurate picture of the relationship between exercise and food intake can be developed if one is careful to distinguish between gender as well as the mode and the intensity of physical activity. Stevenson [68] reported that there was no change in food intake by male rats exposed to 4 hours of swimming 4 days per week. This finding was confirmed in a later study by Oscai [67] where male rats engaged in 6 hours of swimming per day. Average food intake in the exercising rats was 86 ± 2 kcal per day versus 84 ± 3 kcal per day in the sedentary controls. The exercise that these male rats were exposed to were considered to be of low intensity and long duration activity.

Conversely, there appears to be a different response by male rats when exposed to high intensity work of short duration as reported by Crews et al. [43]. Following 12 weeks of 2 hours per day of high intensity treadmill running, Crews et al. [43] reported that food intake decreased significantly in comparison to sedentary controls.

The exercising rats consumed a total of 6090 ± 261 kcal for the 12 weeks versus an intake of 7638 ± 224 kcal by the sedentary control rats. The reduced food intake in response to high intensity activity is in agreement with an earlier report by Stevenson et al. [68]. The results of that study indicated that male rats who were exposed to strenuous swimming for 60 to 120 minutes 4 days per week had a lower food consumption in comparison to the sedentary control group.

Further evidence for the reduction in food intake as a result of high intensity exercise was illustrated in a study by Katch et al. [45]. In an attempt to discern whether or not caloric cost or intensity of exercise influenced caloric intake, Katch divided rats into two groups: a sedentary control group and an exercise group. The exercise group was further divided into a low intensity (group I) and a high intensity exercise group (group II). In the first exercise week (week 2) of the study group I performed low intensity work and group II performed high intensity work. The groups exercised for 5 days and then rested 5 days at which point the exercise groups were switched (ie. group I became the high intensity group and group II, the low intensity group). The total caloric cost of exercise was kept constant within the exercising groups. Food intake was measured at one hour post exercise and every four hours for twenty four hours. Overall, the caloric intake was lower in the exercise groups. However, the high intensity exercise elicited the lowest caloric intake of all three conditions. Furthermore, the high intensity exercise resulted in the lowest gains in body weight. The authors concluded that exercise alone will elicit a reduction in caloric intake in comparison to a sedentary lifestyle. However, due to the fact that the high intensity group experienced a greater reduction in energy intake, one is left to conclude that the intensity of exercise may be significant when one is using exercise to potentiate a negative energy balance.

The reports reviewed above strongly suggest that the appetite suppression effect of exercise is at least moderately linked to the intensity of exercise. The mechanism

responsible for the reduced intake in response to high intensity exercise is not clearly understood. A number of factors have been suggested, ranging from an increased catecholamine response, increased core temperature, increased blood lactate and, in humans, a cognitive recognition of greater work [14-16, 43, 44]. Furthermore, the appetite response appears to differ between gender.

In the extensive literature reviews by Oscai [16] and Titchenal [63] it was stated that female rats do not always manifest the same appetite response to exercise as male rats. Support for this observation was provided by the data from numerous laboratory studies [16, 59, 63, 66, 67, 69-73]. The early investigation of Mayer [66] indicated that female rats exercising for durations of one hour or greater (up to 6 hours) matched increased energy expenditure with an increased energy intake. This finding is similar to that reported by Oscai [67]. Female and male rats were subjected to a swimming program and given unrestricted access to food. The exercising females consumed an average of 75 ± 1 kcal per day as compared to the sedentary controls who consumed an average of 61 ± 2 kcal per day. It is important to note that the male rats in the study did not change their food intake significantly in response to exercise.

In a later study by Johnson et al. [71], using male and female rats the female rats exhibited a different response to exercise than was noted by Mayer [66]. The female rats in Mayer's [66] study that engaged in swimming increased their energy intake as their energy expenditure increased. Conversely, the rats in Johnson's [71] study did not alter their food intake in response to increasing durations of treadmill running. The duration of exercise was gradually increased, starting at one-half hour per day and progressively increased to 1, 2, and 3 hours per day 5 days per week. The animals remained at a specific exercise duration (stages) until voluntary food intake and body weight leveled off (approximately two weeks). Upon completion of the third stage of exercise training the duration of training was reduced in a stepwise fashion decreasing

from 3 hours to one hour and eventually 30 minutes per day. The female rats did not alter their food intake during the detraining sequence and, consequently, achieved a body weight 10 grams greater than the control group. The physiological basis for the difference in the food intake response to chronic exercise between males and females as well as between modes of activity is not yet clearly understood

The observation that food intake differences exist between exercising male and female rats was further illustrated in a report by Applegate [59]. Male and female rats were divided into exercise and control groups. The exercise program initially consisted of running at 21.3 meters/min for 15 minutes per day that was gradually increased until the rats were running for 60 minutes per day. The body weight and composition, food intake, and blood lipids of the rats were measured. The exercise had a significant effect on weight, body composition and food intake (during the light cycle only) of the male rats. Conversely, the exercising female rat did not manifest significant differences in body weight or food intake as compared to their sedentary counterparts. It should be noted that the body fat content in the exercising females was significantly lower than that of the control animals. The majority of adults involved in weight reduction programs are seeking a substantial weight loss as a result of exercise. Therefore, in light of the data reported by Applegate and others [50, 59, 63], individuals participating in weight loss programs should be advised accordingly. Specifically, there may be significant changes in body fat without concomitant changes in body weight.

Applegate [59] noted that the food intake response by female rats is not in agreement with the observations of earlier investigations [16, 66, 67]. There are a variety of reasons for this discrepancy with suggestions ranging from the length of the adaptation period to the time during which the rats were exercised (light vs. dark cycle) as well as the mode of activity (running versus swimming). The conflicting data reported by Applegate [59] serve to confirm the differences between male and female rats. Thus, it

is essential that prior to the formulation of generalized statements regarding the response of male and female rats to exercise, only those studies using similar experimental designs be compared.

An additional variable that may influence the appetite response to exercise is body composition. The development of sound weight control programs for reduction of body fat levels for normal and obese humans is one of the goals of professionals involved in energy balance research. The debate that has transpired is twofold: first, do obese individuals respond to exercise in a pattern similar to lean individuals; and second, is exercise or food restriction more powerful in creating a caloric deficit? To answer these questions Walberg et al. [72] subjected obese and lean female Zucker rats to either an exercise (swimming) or food restriction regime for a period of eight weeks. The swimming program consisted of 120 minutes of swimming per day for a period of 8 weeks. Control animals were kept sedentary for the 8 week period. Both groups were allowed food and water ad libitum. An additional group of obese animals was exercised and food restricted for the 8 week training period.

Food intake for the obese and lean exercised rats increased above the weekly values observed for the sedentary controls. The exercise groups had body weights and body fat levels significantly lower than control animals. The most dramatic changes in body weight and body composition were illustrated in the exercise and food restricted animals. After 8 weeks of retirement the overall weekly food intake of the lean and obese swimmers were similar to their sedentary counterparts. It is interesting to note that in the first 4 weeks of retirement the obese swimmers had a food intake significantly greater than the obese sedentary animals. Overall, this study showed that food intake in lean and obese female rats increased in response to exercise. Additionally, the food intake of the obese exercised rats remained elevated for four weeks after cessation of swimming. Moreover, body composition was enhanced in both the obese and lean

exercised rats, and food restriction and exercise resulted in the greatest changes in body fat and weight, yet caused stunted muscle and skeletal growth. Swimming delayed but did not prevent the complete development of obesity in the obese female rat [72].

The appetite changes induced by exercise suggest that there is a variable response that perhaps is dependent on the intensity and mode of the activity as well as the gender of the animal. The results of numerous studies seem to indicate that, in general, the male rat decreases energy intake in response to treadmill running whereas females tend to increase their energy intake [43, 45, 60, 66, 68, 69, 71]. Swimming appears to cause an increase in the appetite of females; however, the male rat's response is either no change or a slight increase. [16, 63, 67, 72, 73]. Therefore, it is essential that one consider the gender, and the intensity and mode of activity prior to drawing any conclusions regarding the relationship between appetite and exercise in the animal model. The complexity of the appetite response to exercise is even greater in humans.

Chronic Changes in Appetite with Exercise (Humans)

Measuring the relationship between energy expenditure and intake is understandably a much more difficult task in humans in comparison to the rat. Even so, the intricacies of the energy balance equation in humans have been the subject of numerous investigations in the last fifty years. Durnin [74] and Edholm [75] conducted detailed studies examining the relationship between energy expenditure and intake on both a daily and a weekly basis. The studies revealed that there was a great deal of inconsistency between energy intake and energy expenditure for any given day. Durnin reported that only 10 subjects out of 69 showed a significant correlation between intake and expenditure on any one day [74].

The subjects in both studies exhibited significant variations above and below energy balance on a daily basis. It is interesting to note that the investigators observed greater consistency between average intake and expenditure when at least 10 subjects were used

and energy balance was measured for periods of one week or longer. Durnin [74] suggested, due to the fact that there was a significant correlation between intake and expenditure on a weekly basis, that this implied that the control of energy balance is a result of short and long term control mechanisms. The short term control of energy balance may be related to some physical mechanism that is affiliated with such factors as gut distention or motility. Subsequent to Durnin [74] and Edholm's [75] findings additional mechanisms were proposed. Various investigations have proposed that the short term control of energy balance may also be related to humoral factors such as plasma catecholamines [16, 43], lactate [44] or an additional physical factor such as core temperature [15].

Cross Sectional Studies

Mayer and colleagues [76] conducted a landmark study of the mill workers in West Bengal, India. The study was one of the first to critically examine the relationship between exercise and caloric intake in humans. The mill workers' uniform diet increased the reliability of the measurements taken by Mayer et al. [76]. A total of 213 male mill workers served as subjects for the study. The activity pattern of the workers ranged from very light work as observed for supervisors (sedentary) to very heavy work as reported for coalmen, blacksmiths and carriers. The relationship of exercise (job related physical activity) and caloric intake was very similar to the response that had been observed for rats as reported by Mayer et al. [66] two years earlier. The results of this study of humans indicated that there was a specific range or zone of activity that precipitated increases in food intake. Specifically, in this zone, increases in physical activity precipitated increases in caloric intake. Mayer et al. [76] referred to this zone as the zone of "normal activity". Likewise, there was a zone or range of activity below the normal zone that was termed the "sedentary" zone where decreases in activity were not matched with decreases in caloric intake, but on the contrary were matched with

increases in caloric intake. The relationship described by Mayer et al. [76] would imply that appetite regulation is based on some minimal level of activity. The accuracy of Mayer's [76] report has been both challenged and supported over the years; however, a clear picture of the mechanisms regulating appetite is still to be determined. Therefore, the remainder of this review section will focus on the chronic effects of regular exercise on energy balance.

A number of cross-sectional studies have been conducted contrasting the caloric intake of active individuals and sedentary subjects. The majority of the studies have compared the food intakes of moderately trained individuals and sedentary individuals. Results from these studies showed that active individuals tended to consume more calories than sedentary individuals regardless of gender. [3, 17, 19, 37, 76-81]. It could be inferred from these studies that very active or highly trained individuals would exhibit caloric intakes significantly greater than sedentary individuals or manifest significant weight losses in the course of training due to a high energy expenditure without a parallel increase in energy intake. Surprisingly, this is not necessarily the case, especially in highly trained female athletes [3, 5, 6, 81-85].

Duester [3] conducted a detailed nutritional survey of female marathon runners. The purpose of the survey was to obtain an extensive nutritional profile of highly trained female runners. The subjects had qualified for the First Olympic Marathon Trials; thus, they had completed 26.2 miles in a time of 2:51.19 or better. Eighty women participated in the study, completing a 3-day diet record, various diet record forms, an activity questionnaire, medical history forms and menstrual history forms. In addition to the dietary information, each subject's body composition was assessed via skinfolds, and a fasted blood sample was drawn to determine plasma concentrations of calcium, magnesium, zinc, copper and iron. The investigators excluded 29 subjects taking any form of an exogenous hormone .

The average caloric intake was $46.3 \text{ kcal} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$ or approximately 2397 kcal per day, with a range of 1067 to 4271 kcal per day. Although mean caloric intake of this population was above 38 kcal/kg/day (1800-2200 kcal/day) which is the RDA caloric intake for sedentary women, the intake appeared to be low for women who were running approximately 10 miles per day. The caloric cost of running 10 miles per day is approximately 800 kcal if one assumes an expenditure of 80 kcal/mile. Therefore, for these runners to maintain energy balance would require an energy intake of $54 \text{ kcal} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$ (2600 kcal/day). Greater than 75% of the subjects in Duester's study [3] were consuming less than 2600 kcal/day. More exceptional was that 25% of the runners were consuming less than the RDA for a sedentary woman of similar body weight; and even more astonishing was that 11% of the women were consuming less than 1500 kcal per day.

Maintaining a caloric intake of less than $38 \text{ kcal} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$ or 2200 kcal per day would appear to be detrimental to performance, or at the very least be inadequate, thereby producing a significant energy imbalance which could precipitate a significant weight loss. The significant departures in caloric intake reported by the runners could have been due to underreporting in the dietary record. Duester et al. [3] dismiss this proposal suggesting that these women were highly motivated and extremely interested in obtaining an accurate analysis of their nutritional status. Regardless of the level of motivation, the error that is normally assumed for dietary records ranges from 7 to 26 % depending on the meal [86]. The error in self reported food intake can be reduced by utilizing a seven day record and including a minimum of 10 subjects in the experimental design. As previously stated, Durnin [7] suggested that there is a significant correlation between intake and expenditure on a weekly basis. This correlation fits into his hypothesis that control of energy balance is a mixture of short and long term mechanisms. Despite the potential for error, the food intakes remained lower than what

one would expect for the level of activity engaged in by the women in Duester's [3] study. LeBlanc et al. [11] postulated that trained women may have an increased digestive efficiency, illustrated by a reduced postprandial heat production. Further studies are needed before an increased digestive efficiency can be accepted as an explanation for this apparent disparity in energy balance in highly trained women.

The food intake values reported by Duester et al. [3] are very similar to the values reported by Clark et al. [82] for elite female runners. The runners in both studies had similar marathon performances and weekly training distances. However, the runners who participated in the survey conducted by Clark et al. [82] revealed an even lower mean caloric intake of 2,000 kcal per day. It is important to note that a reduced caloric intake may cause an imbalance in the energy balance equation; however, of even greater consequence is the nutritional deficiencies that may be incurred [82].

Reduced caloric intakes were also found in a group of runners recently observed by Kaiserauer et al. [81]. In agreement with the findings of other authors [3, 82, 83], the caloric intakes were below the RDA and a number of micronutrient deficiencies were represented. Three different groups of women were represented: (a) amenorrheic runners (AR), (b) regular menstruating runners (RMR) and, (c) regular menstruating sedentary controls (RMSC). The caloric intakes were 1582, 2490, and 1688 kcal per day for the AR, RMR and RMSC groups, respectively. The amenorrheic runners in this study were running an average of 39.0 ± 6.2 miles, whereas the regular menstruating runners were running 31.0 ± 3.2 miles per week. The running groups had a mean VO_2 max of $56.0 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, thus they were considered highly trained. In spite of the training distances and seemingly caloric deficits maintained by the runners, body weight did not change significantly in the course of the study. The lack of weight loss is especially surprising in the AR runners. The amenorrheic runners had a weekly caloric expenditure similar to the regular menstruating runners. Failure of the

amenorrheic runners to lose body weight in spite of the caloric deficit suggests a physiological change or adaptation was made by these women. The possibility exists that the basal metabolic rate has been lowered in these women due to food restriction in combination with the nutritional deficiencies and/or exercise training [81].

Barr's [6] extensive review of studies measuring the food intake of female athletes further supported the findings of Clark [82] and Duester [3]. The mean caloric intake of female athletes was 2069 kcal per day, close to the RDA for sedentary young women. In addition, a significant number of the studies reviewed by Barr [6] indicated that a high percentage of female athletes tend to have food intakes significantly below the RDA. The mechanisms underlying maintenance of physical performance and rigorous physical training in spite of reduced energy intake remain to be determined. A number of factors ranging from a reduced resting metabolic rate, reduced thermic effect of food and the amount of physical activity performed outside of the training regime have been suggested that would allow female athletes to maintain reduced energy intake and still perform physical activity. Understandably, a lower than "normal" body weight would also explain a reduced caloric intake [6].

In an attempt to examine the effects of nutrition education on female athletes Welch et al. [83] developed a wellness based approach to individual nutritional counseling programs. Thirty nine female intercollegiate athletes, ages 17-22, participating in basketball, cross-country running, field hockey, golf, softball, swimming, tennis, track and field and volleyball served as subjects. Dietary data as well as anthropometrical data were collected during the team's preseason physical and at the end of each team's competitive season. The athletes were divided into two groups, with group one receiving from two to five individual nutritional counseling sessions. Twenty four hour dietary intakes were obtained on both groups by a trained interviewer pre and post season. The mean caloric intakes for group one were 1722 ± 1052 kcal and 1771 ± 852 kcal/day

pre and post season respectively. Group two had caloric intakes of 1788 ± 654 kcal/day and 1893 ± 648 kcal pre and post season respectively. The caloric intake of both groups was below the RDA of 2000 kcal per day for sedentary women. A closer examination of the dietary records illustrated that subjects who received counseling sessions did not make significant changes in total caloric intake. Nevertheless, it is important to note the athletes in group one did decrease their intake of fat and increase the intake of carbohydrates. It would appear that counseling sessions helped modify the diet of some athletes, but did not reduce or diminish their concern for body weight. The caloric intakes reported did not appear to be sufficient to provide the additional calories needed for training or performance. This evidence indicates that either female athletes are training and performing with less than optimal nutritional profiles or perhaps regular training induces adaptations in the energy balance equation that allow female athletes to maintain balance and performance with a reduced caloric intake. Somewhat less likely, but still a possible explanation is that the RDA of calories for females is too high.

In contrast to the findings of the studies discussed above [3, 6, 82, 83] were the findings of Short et al. [79] and Reggini [80]. Short [79] obtained the athletes' caloric intakes via twenty four hour and three day dietary records. The caloric intakes of female swimmers and members of the women's basketball, crew, lacrosse and volleyball teams were comparable to or greater than the RDA for young sedentary females. Unfortunately, the survey of female and male athletes completed by Short et al. [79] did not include runners. Due to the methodology employed to assess dietary intake, it is important to keep in mind that dietary records by design are not the most precise tool available to measure energy intake. In spite of the lack of accuracy in dietary records, the results do suggest that perhaps reduced caloric intakes in female athletes may be dependent on a variety of factors independent of caloric expenditure.

Short et al. [79] theorized that the caloric intake of basketball players and

swimmers was higher than other athletes due to body size and number of workouts per day, respectively. In a study conducted by Smith et al. [17] the dietary intake of female competitive and synchronized swimmers was contrasted to that of sedentary controls. Dietary records of nine competitive swimmers, fifteen synchronized swimmers and ten sedentary controls were assessed using a four day dietary intake record. The competitive swimmers swam 6 days per week, averaging 8000 yards per day for 24 weeks, the synchronized swimmers swam 3 days per week averaging 1500 yards per workout and the sedentary controls did not participate in any regular form of physical activity. The mean caloric intake for the competitive swimmers was 2468 ± 534 kcal per day which was within 250 kcal of the values reported by Short [79]. Intake by the synchronized swimmers was 2282 ± 758 kcal per day and the sedentary controls averaged 2030 ± 668 kcal per day. The values for the controls closely approximate the RDA for young females. Based on these findings, Smith et al. [17] concluded that as the level of physical activity increased so did the caloric intake.

Reggiani et al. [80] contrasted the dietary practices and lipoprotein profiles of twenty-one physically active young (mean age 22.6 ± 3.0 yrs) women with those of twenty-one sedentary controls. The active women were involved in a variety of activities: basketball, gymnastics, handball and swimming for a minimum of 2 hours per day 5 days per week. The mean caloric intake of the active women was 2169 ± 208 kcal per day versus a mean intake of 1756 ± 521 by the inactive controls. Clearly, the caloric intake by the active subjects was significantly greater than the inactive controls. Despite the increased caloric intake by the active females the total caloric intake by these individuals was within the RDA; however, the caloric intakes appeared to be somewhat low given the duration and frequency of physical activity. It is also significant to note that the subject pool did not include long distance runners, who tend to consume less than the Recommended Daily Allowance of calories [3, 82, 83].

A direct relationship between caloric expenditure and intake was first observed in female rats by Mayer et al. [66] and supported in later studies [16, 63, 67, 72]. Accurately assessing the ability of swimmers to balance the energy balance equation is compounded by the difficulty that exists in trying to accurately measure energy expenditure in swimming. Significant variations occur between individuals with respect to skill and efficiency, more so than that which exists in other activities such as running or cycling. Although it is tempting to speculate that swimmers are more likely to defend body weight with increasing work, additional studies are needed to verify this statement. In addition, it is possible that the chronic changes in catecholamines, lactate and body temperature in swimming are different from running and this may lead to variations in swimmer food intake stimuli. The strength or type of food intake stimuli may directly or indirectly affect the ability of the swimmer to maintain energy balance. Conversely, a more accurate assessment of energy intake and expenditures may reveal that, although caloric intakes are above the suggested RDA, they are still low in light of the high caloric output by swimmers participating in two workouts per day.

A study conducted by Blair et al.[77] clearly indicated that active male and female runners consume more calories than sedentary controls. The nutritional habits of 34 male and 27 female long-distance runners, 35-69 years of age, were examined. Participants in the study were required to average at least 24 kilometers of running per week during the previous year. The average training mileages were well above the established criterion, since the males ran an average of 65 km/week and the females 55 km/week. Dietary information was obtained via three day dietary records and verified by interviews with the investigators of the study. All subjects were given specific instructions on how to record food and beverage intake and advised to maintain their normal dietary patterns.

The male and female runners reported caloric intakes significantly greater than the

control subjects. Male runners' mean caloric consumption was 2959 kcal per day versus 2361 kcal per day for the controls. Female runners consumed on the average 2386 kcal per day versus the consumption of 1871 kcal per day by the female control subjects. The female runners were significantly lighter; thus, expressing caloric intakes relative to body weight further exaggerated the differences between the groups: 42 versus 30 kcal/kg for the males and 42 versus 27 kcal/kg for the females [77].

It is interesting to note the greater caloric intake by both the male and female runners, particularly the female runners, in comparison to data reported in studies by other investigators [3, 82, 83]. There are several potential explanations for this variation. First, it is entirely possible that the runners in Clark's [82] and Welch's [83] studies were more competitive and were voluntarily trying to maintain lower body weights to enhance their performance. Second, the older athletes may not have been quite as concerned with body image and body weight as the younger runners. Third, there is some evidence that at extremely high caloric expenditures the athlete is unable to consume adequate food to compensate for the energy imbalance [37, 76].

Brouns et al. [37] simulated a portion of the Tour de France cycling race in the laboratory. The race is a very demanding one that requires extremely high levels of caloric expenditure. Thirteen highly trained male subjects participated in the seven day study. The subjects were confined to a respiratory chamber for the duration of the study. Days one and two were preparation days followed by a rest day and then two days of exhaustive cycling, which were followed by a rest day. Food and fluid intake were monitored for each day of the study. The athletes were able to maintain energy balance on the rest days but were in negative balance on both exercise days. That is, although the caloric expenditures were extremely high on the exercise days, the athletes did not compensate with an increased caloric intake on those days. In contrast the caloric intake on the rest days was above the caloric expenditure. Thus, when examining the energy

balance equation for the entire study, the athletes were able to maintain balance by the incorporation of rest days. Brouns et al. [37] suggested that athletes who are engaged in intensive chronic training may have difficulty maintaining energy balance due to three factors: (a). the adverse effect between physical and mental stress and appetite, (b) intolerance for a intragastric food volume in the exercising athlete, and (c) the reduced number of "resting " hours available for the consumption and digestion [37 page S32].

Furthermore, Brouns [37] found that strenuous physical activity may have elicited a negative energy balance. The data that have been reported for male and female rats indicate that there is a gender difference in the maintenance of energy balance in light of increased energy expenditure due to exercise [16, 59, 63, 66, 67, 69, 71-73]. Further studies with humans are needed to determine if the gender differences are manifested in humans. It is important to recognize that the majority of the dietary records that have been collected and analyzed were recorded while the athletes were in training. Certainly it would be prudent to collect dietary information on females during periods of training and rest to accurately assess how they are able to achieve energy balance.

Longitudinal Studies

It is important to keep in mind that although the actual food intake and hunger ratings of active subjects are useful, individuals may have preconceived ideas as to how their appetite or hunger will be influenced by exercise. Evidence for this was presented in the study conducted by Harris and associates in 1984 [87]. Harris reported that the widely held view that exercise reduces appetite was not the case for a large group of active subjects that participated in the Perrier Study [87]. In fact, a majority (46%) of the respondents felt that exercise actually increased their appetite, 43 percent felt it had no effect and 9 percent felt it reduced their appetite . It was also noted that in those respondents who were involved in strenuous exercise (running), a fraction (29%) felt

exercise reduced their appetite and yet, 46 percent of the runners felt exercise increased their appetite. Although the survey provided some useful information, it lacked sufficient detail to accurately attribute the roles gender, mode and intensity of exercise have on appetite (food intake).

The results of numerous longitudinal studies have indicated that the mode, intensity and duration of exercise as well as gender and body composition influence the adaptive response of energy intake to exercise (energy expenditure) in both lean and obese individuals [18, 19, 38, 49, 61, 63-65, 88-98]. The responses to chronic exercise are as varied as the factors which influence the response, and include an increased caloric intake, no change and a decreased energy intake.

There are reports of both athletes and previously sedentary individuals increasing their energy intake in response to increased energy expenditure through exercise [91, 94, 97]. Parizkova measured significant increases (500 kcal/day) in the caloric intake of female gymnasts in response to training [97]. The caloric intake of the gymnasts was measured during periods of training, reduced training and no training. It also appears that athletes are not unique in demonstrating increases in caloric intake in response to training [94]. Kiens et al. [94] evaluated the impact of a moderate intensity endurance exercise program on middle-aged men who were previously sedentary. Caloric expenditure for the program was estimated at 1100 kcal per week and, yet, caloric intake of the subjects increased by 1800 kcal/week [94]. The results of Kiens et al. [94] implied that appetite control may be a combination of long term and short term adaptations that elicit an overcompensation early in the response, but may require greater lengths of time for a match to occur between intake and expenditure. In contrast to the overcompensation found by Kiens et al. [94], a high correlation was found between energy expenditure and caloric intake in non-obese female college students [91].

Epstein [91] assessed the relationship between exercise intensity, caloric intake and

weight. Seventeen female college students engaged in a running/jogging program for 5 weeks. Each subject's average running time was compared to her mean twenty-four hour caloric intake. The correlation coefficient for running time versus caloric intake was -0.69 ($p < 0.01$). This coefficient suggested that the faster running times were associated with greater caloric intake. A more careful examination of running times indicated that the subjects were exercising at low to moderate intensities. Even though caloric intake increased as the running intensity increased, the mean caloric intake (1520 kcal/ day) was significantly lower than the suggested RDA for young women (1800-2200 kcal/day). Therefore, while this study indicated that increased caloric intakes were concomitant to higher energy expenditures, the subjects were not meeting the RDA for calories similar to the several studies reported earlier in this review.

In contrast to the increases noted by Epstein [91], several studies have shown little or no change in the caloric intake of subjects in response to chronic aerobic exercise [49, 89, 92, 93, 96, 98]. For example, Dempsey [89] studied seven obese and non-obese young men who engaged in a total of 18 weeks of physical training that consisted of three phases: (a) daily training (8 weeks), (b) decreased training (5 weeks) and (c) daily training (5 weeks). The training phase was preceded by a three week pre-training period in which baseline measurements of caloric intake were recorded. Daily caloric intake was measured via food intake diaries completed by the subjects. Several anthropometrical measurements were recorded to provide for determination of body size and composition.

The mean caloric intake did not change significantly during the course of the study. Caloric intakes were 2003 ± 265 , 2148 ± 276 , 2190 ± 323 , and 2231 ± 237 for pre-training, phase one, phase two and phase three, respectively. Thus, although there appeared to be a slight increase in caloric intake, the change was not statistically significant. However, in his review, Wilmore [50] contended that, due to the additional

energy expenditure (400-600 kcal) by the subjects as a result of training, the appetite or caloric intake of the subjects actually decreased. To maintain energy balance the increased caloric expenditure due to training would have required the consumption of an additional 400-600 kcal per day. Surprisingly, the subjects did not maintain energy balance through the study. Verification for this statement is provided for in an examination of the significant body weight and composition changes that occurred in the study. The significant decreases in body weight and composition without concomitant increases in caloric intake may be interpreted as evidence of a decreased appetite. The results of this early study by Dempsey [89] are similar to the results of a longitudinal study that examined the effects of an endurance exercise program on the serum lipids of middle-aged men [92, 98].

The changes in the anthropometrical measurements of the subjects who were involved in the lipid profile study of Holloszy were reported by Skinner [98]. The subjects were running approximately 2-4 miles per day 3.3 times per week. There were no significant changes in body weight or caloric intake by the subjects. A lack of change in caloric intake implied that the exercise training actually induced a decrease in appetite due to the fact that the increased energy expenditure was not matched with an equal increase in energy intake [50]. The results of later studies [63, 93, 96] provided additional support for a lack of adaptation in appetite control in response to exercise.

The results of the longitudinal study by Katch et al. [93] implied that a similar response occurs in females. Katch and colleagues measured female swimmers and tennis players during their respective competitive seasons which were both approximately five months in duration. Caloric intake and composition were evaluated prior to and at the conclusion of the competitive season through the use of a 7-day dietary inventory. The swimming workouts consisted of 2000-4000 meters during the months of January through March and 1000-2000 meters from April through May. The tennis workouts

were approximately one hour in duration and consisted of 10 minutes of rope skipping, 45 minutes of play and a half-mile jog [93]. The mean caloric intake for the swimmers at the beginning of the season was 2091 kcal and did not change significantly in the course of the season. Although the caloric intake for the tennis players was lower (1811 kcal), the response paralleled that of the swimmers. The body weight and composition of all the subjects did not change significantly during the study. In spite of significant increases in energy expenditure the subjects did not significantly change their caloric intake to match the increased expenditure. Conversely, although the change was not significant, there was a trend to decrease caloric consumption throughout the season. There are several potential explanations for the lack of change in this population ranging from gender, mode, intensity or duration of training. Additionally it is possible that the failure of an increased expenditure to elicit a significant increase in appetite may be a reflection of regular training actually promoting a decrease in appetite.

In 1986 McGowan [96] examined the effects of no exercise, regular exercise and double exercise on seven previously trained male joggers. The subjects had engaged in an average of 2.4 -4.0 miles per day 5 days a week for at least 6 months prior to the study. Each subject participated in the three testing conditions in a counterbalanced manner. Subjects wore heart rate monitors to record heart rate and total heart beats during each exercise and resting session. Caloric intake was monitored through daily self-recording for a total of five weeks. Baseline caloric intakes were recorded two weeks prior to the beginning of the test conditions. No significant differences occurred in caloric intake across the test conditions. The caloric intake ranged from 2529 ± 845 kcal for no exercise to 2695 ± 980 kcal for double exercise [96]. This study suggested three important points. First, if exercise is utilized in long term weight control, then perhaps individuals should be cautioned that any reduction or cessation of exercise must be matched by a conscious reduction in caloric intake to avoid weight gains. Second,

although the increase in caloric intake was not significantly increased during the double exercise session it would appear as though the appetite control mechanisms are more sensitive to increases in caloric expenditure than decreases. A larger sample size or greater increases in expenditure may have elicited a significant response. Third, the responses indicated in this study are very similar to those suggested by Mayer et al.[76], that is, decreases in activity are not associated with decreases in caloric intake, but increases in energy expenditure are associated with increases in caloric intake.

In contrast to the studies which have reported increases [91, 94, 97] or no change [49, 89, 92, 93, 96, 98] in caloric intake in response to exercise, there are a number that have noted significant decreases in caloric intake [38, 61, 95]. Johnson et al. [61] developed an exercise program for college women that was designed to help reduce body fat, blood pressure and increase maximal oxygen consumption. Thirty two college age women participated in a 10 week cycling program. The sessions were held five days per week for thirty minutes each. The intensity of the sessions was gradually increased each week so that by week 10 the subjects were cycling at a mean work rate of 1005 kpm/minute for 30 minutes. The workrate was tripled by the conclusion of the study and mean work heart rate was increased by 50 beats per minute.

Caloric intake was derived from 3 day dietary records maintained by each subject during the first, fifth, and tenth weeks of the study. The mean caloric intake decreased from 1751 ± 107 to 1584 ± 92 kcal per week. Significant changes were observed in the sum of skinfolds indicating significant reductions in body fat, whereas total body weight did not change significantly in the course of the study. It is tempting to speculate that the exercise program caused a significant reduction in the caloric intake of college women. Regardless of the obvious decreases noted in caloric intake, this would not be a prudent deduction to draw for two reasons. First, the amount of change, although significant, is

within the error rate [86] that is thought to exist for self recording of caloric intake. Second, the authors did not indicate whether or not the subjects were advised not to change their eating habits during the study. Clearly, these subjects volunteered to participate in the study because they were interested in changing their blood pressure and body composition. Thus, it is conceivable that some or all of the subjects made a voluntary decision to reduce caloric intake that was not related to the exercise sessions. In spite of the reservations with the results mentioned above, they are in close agreement with a later study by Dickson-Parnell and Zeichner [38].

Thirty-three women, ages 18-31 years, participated in a 7 week cycle ergometer exercise program and were randomly assigned to either the high intensity (80% max heart rate), low intensity (55% max heart rate) or control group. The subjects exercised three days per week at either 80% or 55% of max heart rate for a period long enough to generate a caloric expenditure of 200 kcal/ session. Caloric intake was measured via a 7 day food record completed by each subject one week prior to training, during weeks 2,4, and 6 of the study and one week after completion of the study. Overall, there were no significant differences between the groups with respect to total caloric intake across weeks of study. Thus, all groups maintained energy balance in spite of the exercise sessions. Post hoc analysis revealed that both exercise groups had significantly higher caloric intakes on non-exercise days as compared to exercise days. Further analysis indicated that the high intensity group had a significantly lower intake on exercise days in comparison to the low intensity group. The caloric intake of the control group was significantly greater than the two exercise groups on the exercise days, whereas there were not significant differences between the groups on the nonexercise days. The findings of this study are similar to the results reported for animals [68, 93] and humans [99] in which it was observed that high intensity exercise caused a greater reduction in caloric intake as compared to low intensity

exercise, and caloric intake was greater on nonexercise days as compared to exercise days.

While it may appear as though high intensity exercise would be ideal for individuals trying to lose weight, there are additional risks associated with high intensity exercise. Conversely, low intensity activity has long been prescribed as a method for promoting a negative energy balance in obese individuals in order to reduce the incidence of injury and improve the rate of compliance and adherence. Based on the findings of Leon et al. [95], it would appear as though there are additional merits of low intensity exercise.

Leon et al. [95] studied the effects of a 16 week vigorous walking program on six previously sedentary obese men. The subjects walked for 90 minutes per day, 5 days per week, at an intensity high enough to invoke an energy expenditure of approximately 1100 kcal per session. An examination of food intake records of the subjects revealed that during the first four weeks caloric intake actually increased by 300 kcal per day. Subsequent to the first 4 weeks, caloric intake gradually decreased and by week 16 caloric intake was 130 kcal below the pretraining level. The initial increase, and then gradual decrease, may imply that low intensity, long duration exercise is capable of stimulating a reduced appetite, but that the response is not manifested until later in the training program.

All of the studies reviewed thus far relied on food diaries in order to determine caloric intake. While it is true that diaries are the best tool to insure "representativeness", they are not as accurate as direct observations by professionals [7, 86, 100]. To avoid the problems and inaccuracies associated with personal food diaries, Durrant et al. [90] studied lean and obese men and women in a metabolic ward. Subjects received food via automated food dispensing machines while staying on the metabolic ward. Each subject engaged in 3 days of sedentary living and three days of exercise on a cycle ergometer. Each exercise session was designed to elicit a minimal

expenditure of calories (100 kcal/session). The lean subjects increased their caloric intake in contrast to the lack of change in the obese subjects.

Woo et al. [18, 64, 65] and Pi-Sunyer [19] have conducted numerous metabolic ward studies in an attempt to understand energy balance in obese and lean subjects in response to exercise. Woo et al. conducted two metabolic ward studies using 6 obese [64] and 5 lean [18] women. The subjects in both studies were informed that they had volunteered to participate in a study designed to evaluate protein metabolism, and, thus, were unaware that food intake was being monitored. Each subject was given triple portions of a meal on platter. All food was weighed before and after each meal to determine caloric intake for the subject. There were three 19 day phases to each study: a sedentary evaluation phase and two exercise phases. The obese subjects engaged in treadmill walking at levels high enough to raise total daily energy expenditure to 110% (mild) and 125 % (moderate) of their sedentary level. The lean subjects performed exercise at 114 % and 129 % of their sedentary levels. The caloric intake of the lean subjects increased during both exercise conditions to a level where the increased expenditure was matched by caloric intake. Conversely, the caloric intake of the obese women did not change significantly, resulting in a negative energy balance. In a follow up study, Woo et al. [65] exposed obese women to 57 days of moderate exercise (125% of sedentary expenditure). The subjects were provided with a food surplus throughout the study; their intake was monitored covertly. The mean caloric intake was 1903 kcal per day; the treadmill exercise had no impact on their daily caloric intake. The mean energy expenditure for the subjects was 2882 kcal per day, thus the subjects maintained a negative energy balance of approximately 979 kcal per day throughout the study. Physical activity is just one of several physiological and psychological factors that will influence caloric intake. An additional factor that has been eluded to recently in females is the stage of the menstrual cycle.

The appetite of 21 females with clinically diagnosed premenstrual syndrome (PMS), and 13 controls was recently measured by Both-Orthman et al. [88]. Both groups had a higher appetite rating in the pre-menstrual phase (7 days prior to menses) as compared to the post menstrual phase (7 days post menses). Additionally, the PMS patients had a higher appetite rating in the premenstrual phase than was shown by the control group. This study showed in a prospective fashion that appetite and menstrual cycle phase appear to be related [88]. Due to the numerous factors that may influence caloric intake, it would seem prudent that when measuring the response of females that the menstrual phase in which they are evaluated be standardized as much as possible.

In general, regular aerobic exercise will cause either an increase in energy intake or no change. However, there are a few studies that have reported decreases in caloric intake in response to exercise. Regardless of the impact exercise has on caloric intake, individuals engaged in chronic physical training tend to lose body fat [50, 101]. Therefore, exercise either separately or in combination with diet is advised if an individual is attempting to produce a negative energy balance for the purposes of weight control.

The Assessment of Appetite

There are a variety of techniques that have been used to measure the food intake and or hunger of humans. [2, 6, 7, 19, 63, 86, 100, 102] Regardless of the technique utilized it is important to try to achieve a balance between accuracy and representativeness. Unfortunately, the process of monitoring an individual's food intake can, by itself, influence the eating habits of the subjects [19]. While it is true that observations or food weighing techniques may be the most accurate, they are not considered the most valid [63]. The methodologies used to assess food intake in free living subjects as opposed to a metabolic ward setting, are normally divided into two

major categories: those relying on memory in which the subject recalls the foods eaten, and those relying on record keeping, in which the subjects keep a diary or record of all foods and beverages consumed.

Twenty four hour recall is a useful technique to assess the food intake of a group, but it is highly suspect for accurately determining the intake of an individual [103, 104]. Variability of daily food intake by an individual is significantly greater than is the variability of food intake for a group for 24 hours. A 24 hour recall also tends to overestimate the caloric intake of individuals with low intakes and underestimate the intake of individuals with high caloric intakes [6, 105]. In spite of the problems with using a 24 hour recall it is still considered a viable tool to measure caloric intake [86, 102]. The reliability of the 24 hour recall can be improved by: a). conducting an interview with the subject by an individual trained in dietary record keeping and b). obtaining several random 24 hour recalls throughout the study period .

The three day and seven day records are common techniques used with free living subjects in an attempt to assess caloric intakes. Acheson et al. [100] found that the seven day record was not only the most practical but also the most accurate dietary record keeping technique. While it may seem logical to extend the record keeping period in order to improve accuracy and representativeness it appears as though subject compliance to the record decreases over time thus, reducing the validity and reliability of the record [100] 71. Stern's [86] review of energy intake techniques emphasizes that dietary records can be an effective tool for estimating caloric intake. This viewpoint is reinforced by Stunkard and Waxman [102] . When Stunkard compared dietary records or "self-reports" to actual observations, a correlation of 0.96 was found between measured food intake and "self-reports". In contrast to the high correlation recorded by Stunkard et al. [102], Stern [86] cautions that one must accept that records will normally yield an error of at least ten percent in spite of all attempts to improve

accuracy.

The accuracy of the dietary records can be improved by providing orientation sessions to the subjects prior to collection. It is particularly important to impress upon the subjects to maintain their usual dietary habits. Additional accuracy can be gained by providing measuring devices to assist the subjects in reporting accurate quantities of food [4]. The maintenance of a dietary record for several three day periods prior to the beginning of the study may improve the accuracy of the record. Lastly, using a dietary record in combination with interviews by trained personnel will also enhance the accuracy of the estimated values of caloric intake.

The report by Dalvit [106] implied that it may be important to obtain several dietary records when using female subjects. The caloric intake in menstruating females was 500 kcal greater during the luteal phase as compared to the follicular phase. Thus, it would be prudent to collect a dietary record or perform 24 recalls throughout the cycle or obtain 7 day food records during each phase of the cycle and average the intake from the two periods. Regardless of the subject population the investigator must balance accuracy and representativeness. If the focus of the research is on the response in the subject's natural environment, then it would appear as though either several random 24 hour recalls or 7 day records should be obtained if one is attempting to estimate the caloric intake of a subject.

Assessment of Hunger

Measuring hunger is significantly more difficult than appetite. However, several investigators have utilized a variety of scales to estimate the individuals internal drive to consume food [14, 40, 42, 107-109]. The most common scale used was a nine point scale in which the subjects rated their hunger with values ranging from 1 (very, very full) to 9 (very, very hungry). The most recent utilization of this hunger scale was by Maxwell [42] and Thompson et al. [14]. The subjects in both studies rated their hunger

before, during and after aerobic exercise at a variety of intensities.

Maxwell [42] provided an explanatory paragraph to each subject prior to testing that emphasized the importance of the subject concentrating on internal physical cues when rating their hunger. Hunger was significantly reduced following all exercise treatments. Hunger ratings gradually increased in the final 2 hours of recovery for all treatments, control and exercise. However, the hunger ratings at 90 minutes and 120 minutes following exercise were still lower than the control (no exercise) treatment [42]. The reduced hunger response recorded following exercise reported by Maxwell [42] lends support to the argument that exercise can have an anorexic effect. Thompson et al. [14] reported lower hunger ratings following exercise, however, the duration of the reduced hunger was very acute and did not appear to affect the calories consumed in the post exercise meal. Although the techniques currently available for measuring energy intake and perception of hunger are somewhat subjective, they are useful tools for providing representation of how exercise may affect eating behavior.

Summary

A thorough understanding of the relationship between exercise, hunger and appetite is extremely difficult due to the complications that are associated with the techniques used to take the measurements. In spite of the methodological problems it is imperative that studies are conducted which may provide additional knowledge that may enhance the practitioner's ability to develop logical and pragmatic weight management programs. It is equally important that coaches and athletes are given information which assists them in designing training programs to maximize physical performance.

The mechanisms that control the hunger response are complex and subject to the influence from numerous internal and external cues. In its simplest form, hunger is a state in which fuel delivery to the vital organs is inadequate and the liver is being stimulated to generate metabolic fuels. Based on this definition one would assume that

exercise would precipitate a significant depletion of energy supplies and, thus, increase the hunger response. Nonetheless, the majority of findings have indicated this is not the case. Most studies report a decreased hunger response following exercise [14, 37, 40-42].

The physiological mechanisms responsible for the anorexic effect of exercise have not been clearly delineated. Several factors have been proposed including increased plasma catecholamines [16, 43], increases in lactate [44], increases in core temperature [15, 45], and the production of an anorexigenic factor [36]. All of the suggested mechanisms are viable and it is quite possible that the anorexic effect may be the result of an integration of some or all these factors.

The majority of adults are able to maintain body weight within fairly narrow limits. The ability of humans and animals to control appetite and maintain body weight is the result of an integration of short term and long term control mechanisms [23]. Numerous organ systems are involved in the control of appetite; however, the fat deposits are most likely the system being regulated, as this is the body compartment that changes the most in adult life.

It is known that there are specific receptors for catecholamines in the brain [27, 28]. The activity of these receptors dictate whether a hunger or satiety response will be invoked by the brain [27, 28]. The gastrointestinal tract is also actively involved in appetite control as a result of the mechanical and humoral receptors relaying information about distention and glucose levels [23]. Coupled with the actions of the gut, the liver is continually sending information to the brain regarding circulating levels of fats, carbohydrates and protein. The brain, upon receiving the signals, interprets whether or not the endogenous sources of these fuels need to be activated. Although the liver sends a variety of signals regarding substrate levels, the primary tissue that is being regulated is the adipose tissue. Indirectly a fat cell helps to regulate its own size

via the circulating levels of insulin [32, 33]. As the level of insulin rises this increases the concentration of insulin in the cerebrospinal fluid signaling the brain that there is an increase in the body fat stores [32, 33]. Thus, the maintenance of body weight and control of appetite is dependent on the integration of short and long term controls by a number of systems throughout the body. The ultimate objective of the control is to regulate the size of the body's fat stores.

A considerable number of studies have examined the relationship between exercise and energy intake in both animals and humans. The results of the animal studies have indicated that male rats subjected to treadmill running tend to decrease their food intake; conversely, female rats show no change or increase their energy intake. Swimming does not appear to change the caloric intake significantly in males but will increase the intake in females. The mechanism behind the dimorphic response remains to be elucidated.

The food intake response in humans to exercise has been measured in cross sectional and longitudinal studies. The cross sectional studies have suggested that trained men tend to increase their caloric intake in response to an increased caloric expenditure. The majority of studies that have studied the trained female have suggested that physical training will elicit a variable appetite response. Specifically, the nutritional surveys [3, 6, 81-83] have indicated that highly trained female runners tend to have caloric intakes significantly below what would be predicted given the high rates of energy expenditure experienced by these athletes. In contrast Blair et al.[77] reported that the caloric intake of middle-aged female runners was significantly greater than their sedentary counterparts. In addition, surveys and studies of female athletes involved in various team and individual sports have found that these women tend to have caloric intakes close to the RDA. The caloric intake of the females approximated the suggested RDA and were above the average intake of female runners, and yet they were below the

values that would seem necessary to maintain energy balance in light of the increased energy expenditure [1, 79, 80].

The results of longitudinal studies of appetite and exercise have varied; however, the majority have suggested that caloric intake will not change appreciably [49, 89, 92, 96] , or will increase slightly [91, 94, 97] as the expenditure of energy increases. To add to the confusion surrounding appetite and exercise there are a few reports of a decreased food intake in response to exercise [38, 61, 95]. There does not appear to be a gender difference in the appetite response of humans to increased energy expenditure via exercise. Nevertheless, it is essential to note that the majority of studies tracking appetite and exercise have been conducted using either obese females or lean males. Furthermore, the studies have employed either jogging/running or cycling for very limited periods. In light of the increased risk of orthopedic problems associated with jogging or running it would seem prudent to examine the impact swimming has on the appetite of males and females of various body sizes and composition.

In contrast to the mixed response by lean subjects, obese individuals tend to maintain caloric intake at pretraining values. The response of the obese serves as another example of the inconsistent and conflicting reports that exist within the literature. The perception of hunger, the concomitant appetite response following exercise, and the response to chronic training is indeed complex. Clearly, additional study is warranted that will help illustrate the mechanisms producing hunger, controlling appetite and assisting in the maintenance of body weight.

Resting Metabolic Rate

Standards

Basal metabolic rate represents the lowest metabolic rate in humans and is a significant portion of an individual's total 24 hour energy expenditure, yet it is very difficult and somewhat impractical to measure. In contrast, the resting metabolic rate

(RMR) is the best predictor of the individual's overall energy requirements and is normally 60-75% of the total daily energy requirements [110]. RMR is an estimate of the energy cost of maintaining the body temperature, electrolyte balance, and the functioning of the central nervous system, cardiovascular system, pulmonary system and other necessary general chemical reactions [20]. The RMR is normally measured while the individual is in a rested, but fasted state. The resting metabolic rate of an individual is influenced by an assortment of factors such as age, gender, body size and composition, temperature, thermogenic hormones and prior exercise.

Recent technological advances have facilitated the measurement of RMR, and with the advent of respiratory chambers and whole body calorimeters, the measurement of the 24 hour energy expenditure has also increased. However, in spite of the technological advances, the use of a respiratory chamber remains extremely limited. Although a respiratory chamber improves the accuracy of energy expenditure measurements, it is difficult to establish an environment that is similar to the subject's natural free living environment. The few studies that have been completed using a respiratory chamber, or whole body indirect calorimeter, have produced different results. Specifically, it is difficult at this time to determine whether body weight or fat free mass is the best predictor of 24 hour energy expenditure. Ravussin et al.[111] measured the 24 hour energy expenditure of lean, moderately obese and obese males and females. The fat free mass had the highest correlation ($r = 0.95$) to 24 hour energy expenditure. This finding was in agreement with the study by Webb [112, 113] who found a correlation of $r = 0.95$ between 24 hour energy expenditure and fat free mass. In contrast, de Boer et al.[114] measured 29 lean and overweight women and observed that body weight provided the highest correlation to 24 hour energy expenditure. These results imply that gender, age and body size differences between subjects can be compensated for by expressing energy expenditure in relation to fat free mass.

A report by Owen [110] strongly suggested that the prediction equations that were developed in the first half of the twentieth century are inadequate and in general tend to overestimate the RMR of both lean and obese subjects [110]. There is a variety of ways RMR is expressed: the units of expression can be in relation to time, body surface area, total body weight, fat free mass or lean body mass. In a study of 104 healthy lean and obese men and women Owen [110] reported the best predictor ($r = 0.74$) of RMR was total body weight. In view of the ease with which body weight is measured and obtained and the high degree of accuracy that is possible, Owen [110] suggested that body weight can be used to estimate RMR, in place of previous prediction equations. The equations generated by Owen using body weight predicted RMR in female athletes within 8-10% of the measured values. It is of interest to note that body weight was more highly correlated to RMR in female athletes ($r = 0.94$) than nonathletes ($r = 0.74$). Similar data for men were not available. It is also significant to note that if fat-free mass is known, a single regression equation can be utilized to estimate RMR in men and women. The convenience of using equations to predict RMR should be limited in view of the variability of RMR within a non-athletic population [12, 115-117]. In general the normal range of RMR for women is 900-1500 kcal/24 hour and for men, 1100-2200 kcal/24 hr. Obviously size, gender, activity and body composition will ultimately influence an individual's RMR [110]. The RMR in young healthy subjects weighing 50-70 kilograms will range from 1100-1800 kcal/hour. Thus, there appears to be a slight discrepancy among authors as to the "normal" range. This discrepancy further precipitates discussion on whether or not obese individuals have a "normal" RMR [78].

The findings of Owen differ somewhat with other reports as to the most appropriate anthropometrical variable that should be utilized to predict RMR. [12, 115, 117, 118]. Hoffmans et al. [117] compared the metabolic rates of obese (33.6% fat) and normal (20.4% fat) women. The fat free mass was the best predictor of RMR for the normal

($r=0.67$) and the obese ($r=0.75$) women. When the two groups were combined, resulting in a greater range of body weight and body fat, the fat free mass ($r=0.64$) and body weight ($r=0.64$) showed similar correlations to RMR. Further analysis revealed that the best prediction of RMR was obtained when fat free mass and fat weight ($r=0.74$) were used as independent variables in a linear regression equation [117]. In addition, it was surprising that the RMR per kilogram of fat free mass was higher in the obese group as compared to the normal women. The RMR of these subjects was also correlated to fat mass and body surface area. The results of this study strongly suggested that no single variable of body size or composition is adequate for estimating RMR in a population of women who differ significantly in body size and composition.

The level of obesity and/or the homogeneity of the population being measured appear to influence the variable that serves as the best predictor of RMR. Halliday et al. [119] measured the RMR of 22 obese women. RMR was correlated with a number of variables, and the highest correlations were found with body weight ($r=0.79$), surface area ($r=0.79$), creatine excretion ($r=0.69$) and lean body mass ($r=0.84$, potassium, $r=0.76$, water). The high correlation of lean body mass to RMR was in contrast to the findings of Bray et al. [120]. Bray et al. [120] measured the RMR of 18 grossly obese women and found that RMR was better correlated to body weight, body fat and surface area as compared to lean tissue measures such as total body water, or potassium and creatine levels. It should be noted that although the subjects in both of these studies were obese, the subjects in Bray's [120] study weighed considerably more. Specifically, 14 of the 18 subjects in Bray's [120] study were greater than 120 kgs, whereas only one subject in Halliday's investigation weighed 120 kgs. Thus, the degree of obesity in itself may influence the anthropometrical variable that will ultimately serve as the best predictor of RMR.

The RMR of 30 obese, moderately obese and normal subjects was measured by

Ravussin and associates [111]. Although the correlation of RMR to body weight was high for all groups ($r = 0.74$), a higher correlation was found between RMR and fat free mass ($r = 0.82$). The absolute RMR (kcal/day) was higher in the obese subjects when compared to the normal subjects; this is in agreement with other reports [117, 118, 121]. The results of this study were later supported in a follow up investigation in which Ravussin et al. [115] studied the RMR and 24 hour energy expenditure of 177 male and females. Fat free mass was found to be the best predictor ($r = 0.81$) of RMR in both men and women. Similar findings for males were reported by Poehlman et al. [12] and Astrup et al. [122]. Although some studies have found a good correlation between body weight and RMR the best predictor appears to be fat free mass in normal and obese populations. Furthermore, the majority of reports have found that the RMR is higher in obese subjects than in normal subjects. This finding should not be surprising in light of the additional lean tissue that is necessary to support and carry the excessive body weight of the obese individual. However, there is not universal agreement on this issue since some investigations have reported RMR within the normal range in obese individuals [118].

Resting Metabolic Rate and Exercise

Cross Sectional Studies

The impact that exercise has on RMR has been examined through cross sectional studies [10-12, 78, 110, 123-128], and longitudinal studies [8, 78, 129-135]. The studies that have examined the RMR of trained and untrained subjects have produced varying results. The reports have shown: a higher RMR in highly trained males [12]; a higher RMR in trained versus untrained males that did not persist when corrected for lean body mass [9, 136]; no difference in the female athlete [10] or male athlete [13] as compared to untrained subjects; and a lower RMR in the trained female [6]. Some of the discrepancy that has occurred between studies may have been due to the varying

times at which the measurement was taken in relationship to the last exercise session.

It is difficult to speculate on the relationship between training and RMR in females due to the scarcity of reports in this area. Owen et al. [110] reported significant differences between the regression equations for nonathletic and athletic females. In addition, they reported that the female athletes had greater increases in RMR per unit of body weight gain in comparison to nonathletic females. In agreement with other reports Owen et al. [110] found that if the RMR was reported per unit of lean body mass, the influence of gender on RMR was removed. However, the RMR of the population of the study did not include male athletes, therefore, a comparison of athletic male and female athletes was not possible. The results of the study by Owen et al. [110] differ from data reported earlier [10, 116]. LeBlanc et al. [10] measured the RMR of 30 females during the postovulatory phase. The women were categorized as either: highly active, moderately active or sedentary. There were no significant differences between the groups in RMR. Owen et al. [110] did not report the VO_2 max of his subjects and thus it is difficult to compare the data with those reported by LeBlanc [10]. Regular training may have a chronic impact on RMR but without adequate information regarding the VO_2 max of the subjects, comparisons across studies is limited. Specifically, in these two studies the classifications may not be based on equivalent levels of aerobic capacity, which restricts one's ability to combine the data and draw any generalizations about the relationship between RMR and VO_2 max.

In an earlier study by Owen et al. [116] the RMR of 36 female nonathletes and 8 athletes was measured and the authors found no significant difference between the two groups. This finding is somewhat difficult to understand based on a later report by Owen [110] that showed greater increases in RMR per unit of body weight gain in athletic women as compared to nonathletic women. The correlation of body weight to RMR was 0.94 for the female athletes and 0.74 for the nonathletes.

The apparent lack of a training effect on RMR was also described by LeBlanc et al. [10]. The RMR of thirty healthy young women (mean age 26 years) was measured. Subjects were placed in one of three groups: 1. trained subjects, that included distance runners, cross country skiers and a racing cyclist; 2. moderately trained subjects who exercised at least three times per week for 60 minutes per day at 60% of their maximal heart rate; 3. untrained subjects who did not participate in any form of regular physical activity. The VO_2 max was 57.5 ± 0.95 , 49.9 ± 0.81 and 38.9 ± 0.147 $\text{ml}\cdot\text{kg}^{-1}\text{min}^{-1}$ groups 1, 2, and 3, respectively. Although there were no significant differences in RMR between the groups, the trend appeared to indicate that the greater the activity, the higher the RMR. It is possible that if greater differences existed in fitness and activity levels that significant differences would have been demonstrated in RMR. Furthermore, a larger population of subjects from each group would provide greater confidence in the results presented by Owen and LeBlanc [10, 116].

Brehm et al. [124] found statistically insignificant differences in RMR between trained and untrained males and females. The lack of statistical significance is not surprising in light of the low numbers of subjects. A total of 8 males and 8 females, four trained and four untrained, were measured.

Although there is not an abundance of studies regarding the RMR of trained versus untrained males, the evidence available does imply that trained males have an elevated RMR [12, 126, 136]. Poehlman et al. [12] measured the RMR of 18 male subjects between the ages of 18-37 years. The subjects were divided into two groups based on their activity patterns. The trained subjects were distance runners who trained 100-600 km/week. The untrained subjects were not involved in any form of regular physical activity. Maximal oxygen consumption values were measured for both groups with a mean VO_2 max of 70.5 ± 1.8 and 53 ± 2.4 $\text{ml}\cdot\text{kg}^{-1}\text{min}^{-1}$ for the trained and untrained groups, respectively. It is important to note that although the untrained group was not

involved in regular physical activity, a VO_2 max of $53.0 \text{ ml}\cdot\text{kg}^{-1}\text{min}^{-1}$ is considered above normal for that age group of inactive males. The absolute (kcal/min) and relative values (kcal/kg of fat free weight) of RMR were determined for each subject. The RMR of the trained subjects was greater than the untrained when expressed on an absolute (1.29 kcal/min vs. 1.17 kcal/min) or relative (1.18 kcal/kg FFW vs. 1.05 kcal/kg FFW) basis. The elevated RMR in the trained subjects was sustained when the subjects were matched for body fat content. This finding suggested that high levels of physical activity play a significant role in the RMR of an individual. That is, it is well known that trained individuals tend to have lower body fat levels than their sedentary counterparts and yet body composition differences did not explain the differences measured in RMR.

The relationship between RMR and physical activity was further supported in a recent investigation by Poehlman et al [126]. Twenty eight subjects with a wide range of physical activity patterns ranging from sedentary to running 6-7 days per week, and VO_2 max values ranging from 45 to $70 \text{ ml}\cdot\text{kg}^{-1}\text{min}^{-1}$, served as subjects. There were three groups: sedentary subjects who reported no regular physical activity, moderately trained subjects who ran 3-4 times per week, and highly trained subjects who ran 6-7 times per week. Surprisingly, there was no significant difference in the fat free weight between the groups. As would be expected the highly trained group had significantly lower levels of body fat in comparison to the untrained and moderately trained groups. The RMR expressed in relation to fat free weight (FFW) was greatest in the highly trained group ($1.20 \text{ kcal}\cdot\text{kg FFW}^{-1}\text{hour}^{-1}$), followed by the untrained ($1.09 \text{ kcal}\cdot\text{kg FFW}^{-1}\text{hour}^{-1}$) and the moderately trained ($1.06 \text{ kcal}\cdot\text{kg FFW}^{-1}\text{hour}^{-1}$). There was significant correlation between VO_2 max and RMR ($r = 0.55$ $p < .01$). It should be noted that not all investigations have reported an elevated RMR in trained subject [11, 125]. This apparent discrepancy may be related to the differences in activity and/or the maximal aerobic capacities of the subjects. The highly trained subjects in Poehlman et

al's. [12, 126] investigations had average VO_2 max values of $70 \text{ ml}\cdot\text{kg}^{-1}\text{min}^{-1}$ or higher, whereas, the mean VO_2 max was approximately $60 \text{ ml}\cdot\text{kg}^{-1}\text{min}^{-1}$ in earlier reports [11, 125]. This difference suggested that perhaps very high fitness and activity levels are necessary before the RMR of the male is significantly impacted.

The report by Tremblay et al. [136], which agrees with the majority of other evidence, strongly suggested that high levels of activity promote an elevation in RMR in males. The RMR of the untrained group and a group training 6-10 hours per week were essentially the same. In contrast, the group training 12-16 hours per week demonstrated an elevation in RMR.

Brehm et al. [124] found no significant differences between trained and untrained males and females. The lack of statistical significance is not surprising in light of the low numbers of subjects. A total of 8 males and 8 females, four trained and four untrained, were measured. In light of these data, and the differences reported in the majority of studies, it would appear as though either high levels of activity and/or high levels of fitness precipitate an elevation in RMR in males. Conversely, the few studies that have reported RMR values for trained women implied that training does not elicit an elevation in RMR. These studies should be interpreted with a great deal of caution in light of the low number of subjects and the results that longitudinal studies have demonstrated. A conservative evaluation of the studies indicates there is insufficient data available to conclude whether or not training has an impact on the RMR of women.

Longitudinal Studies

The impact that exercise has on RMR has also been examined in longitudinal studies of obese and lean men and women [4, 8, 9, 129-132]. The limited longitudinal studies with female subjects seem to indicate that in trained and untrained females chronic endurance exercise will either precipitate a small yet significant increase in RMR or no change in RMR.

Tagliaferro et al. [135] studied the impact of a 10 week jogging program on the RMR, VO_2 max, the thermic effect of food (TEF), and body composition of 10 healthy young women. The subjects participated in a 10 week jogging program 3-5 days per week for 30 minutes at an intensity of 70-80% of heart rate range. A training heart rate was calculated for each individual using the Karvonen formula. The subjects showed significant improvement in VO_2 max (20%), as well as a mean decrease of 2.4% in body fat, which represents a change of 10.4%. In spite of the significant changes in body composition and cardiorespiratory function, the changes in RMR were extremely varied (-21 to + 2%) among the subjects [135]. Those individuals who manifested the greatest improvements in VO_2 max had a greater tendency to demonstrate an increased RMR and TEF. Conversely, those individuals who showed only a slight or no increase in VO_2 max tended to show a decrease in RMR.

The large variance in the RMR at the end of the study may be related to the caloric intake changes that were observed for some subjects. The subjects were asked to maintain a constant caloric intake. The mean caloric intake did not change significantly during the 10 week program (1828.7 ± 437 to 1706 ± 456 kcal/day). Although there was no change in the mean caloric intake, there were some changes by individual subjects that deserve examination. First, the subjects were asked to maintain caloric intake at pretraining levels and yet the majority either did not comply or the exercise precipitated an unconscious decrease in food intake. Six of the nine subjects had a lower daily caloric intake at the conclusion of the study. The decreases in caloric intake ranged from 95 to 508 kcal/day. In five of the six subjects who decreased caloric intake their RMR also declined at the end of 10 weeks. Second, the observed caloric intake indicated that the majority of the subjects were consuming less than the RDA of calories for their age and gender. Not only were some of the subjects taking in fewer calories than the RDA, but of the five subjects who demonstrated a reduced RMR, three had caloric intakes in

the range of 1013 to 1291 kcal. Severe restriction of energy intake is known to depress RMR [137]. Thus, the combination of caloric expenditure through jogging and the low caloric intake may have produced a significant negative energy balance which stimulated a decreased RMR. A closer examination of the data reveals that each subject who decreased her caloric intake also demonstrated a reduction in RMR. It is tempting to speculate that although exercise may have a stimulating effect on RMR, this effect can be negated by a low caloric intake that in combination with exercise produces a significant negative energy balance.

Belko et al. [130] have also reported a lack of a statistically significant change in RMR as a result of exercise. In their study [130] they examined the impact of a negative energy balance achieved by diet and exercise, and by diet alone in moderately overweight (115% of ideal weight as established by Metropolitan Life Insurance Company tables) females. The subjects lived in a metabolic unit for 8 weeks. During the first two weeks of the study baseline values for BMR, RMR, TEF, VO_2 max, body fat and energy requirements for weight maintenance were determined. Upon completion of the baseline period the subjects were divided into two groups: group 1 followed a moderate energy/exercise regimen (MEEX); group 2 followed a low energy/sedentary routine (LESD). The energy expenditure and/or energy intake was manipulated so as to produce a total daily energy deficit which was 50% of the baseline value. The exercise regimen consisted of treadmill walking 6 days per week, on the seventh day energy intake was manipulated so as to maintain a 50% energy deficit.

It is somewhat surprising that 6 weeks of a 50% energy deficit did not precipitate a significant change in BMR or RMR. In spite of this observation the investigators [130] did measure a slight increase in the BMR of the exercise group (MEEX). The total weight loss was greatest in the LESD group, and the fat losses were similar between the groups. In defense of the efficacy of diet and exercise as mechanisms of weight loss, Belko [130]

and her colleagues maintained that the improvements in body composition and VO_2 max achieved by the MEEEX group would ultimately raise the overall health status of an individual as opposed to dieting alone. Moreover, near the end of the study the MEEEX group was losing more fat than the LESD group. In light of a 50% energy deficit a decrease in RMR would certainly be expected. However, in spite of the deficit, the caloric intake that was imposed on the subjects did not drop below 1200 kcal/day. In view of a 1200 kcal/day intake one would not expect significant changes in RMR as this is the recommended minimum caloric intake necessary to preserve RMR during weight loss [138].

The inconsistent results of the studies by Tagliaferro et al. [135] and Belko et al. [130] were in sharp contrast to the results by Lawson et al. [8] who studied the impact of a 10 week jogging program on RMR. The training time was gradually increased from 17 minutes three days per week to 77 minutes per day, four days per week. The RMR of each subject was determined on a nonexercise day using a ventilated hood system. The body composition and resting heart rate of the subjects were also measured. The subjects were instructed to eat ad libitum throughout the jogging program. There were significant changes in the RMR, (213.2 ± 10.4 ml O_2 /min to 241.5 ± 9.0 ml O_2 /min.) [8]. The jogging program failed to precipitate the significant changes in body composition that have commonly been observed in other studies using female subjects. The lack of a significant change in body composition, specifically fat free mass, led the authors to conclude that the underlying cause of the increased RMR was related to either an increased caloric intake, or a prolonged thermogenic effect or a combination of both. The caloric intake of the subjects was not reported, therefore it is difficult to confirm or refute the authors' conclusions.

Several studies have measured elevated resting metabolic rates in highly trained males (VO_2 max > 69 ml/kg/min) [9, 12, 126, 136]. Unfortunately, there is

insufficient evidence as to whether or not highly trained females typically manifest a similar response to chronic training. Vallieres et al. [4] measured the RMR of 6 trained female swimmers twice over a 30 day period of training and found no significant change in RMR despite a reduction in training time (18 to 14 hours/week) that was equivalent to a reduction in energy expenditure of 360 kcal/day. Any implications that might be drawn from this study are limited due to the lack of untrained controls. Moreover, although the RMR did not change, the mean values for the swimmers were not reported, thus, making a comparison to other published data impossible.

Several longitudinal studies have examined the relationship between diet, exercise and resting metabolic rate in obese men and women [9, 129, 131-134, 137, 139-141]. It is difficult to make generalizations about the impact diet and exercise have on RMR in obese men and women due to the varying degrees of obesity, caloric restriction and exercise training. However, Tremblay et al. [9] and Lennon et al. [131] using different exercise and diet protocols, measured statistically significant increases in RMR due to regular aerobic exercise in overweight adults. Lennon et al. [131] randomly assigned 75 overweight men and women (15-35% above ideal weight as defined by Metropolitan Life Insurance tables) to one of three groups: 1. control group, diet only (C); 2. a diet and exercise group, in which the exercise was self-selected (D) and 3. a prescribed diet and exercise program (P). Caloric intake assignments for each subject were estimated by the formula: caloric intake = $10 \times \text{ideal weight}$, and the value was rounded either 1200, 1500 or 1800 kcal/day. The caloric intakes were 1200, 1500 and 1800 for groups C, D and P respectively. The subjects in the diet-exercise group (D) were given a list of potential activities and asked to participate in one of those activities every day for 30 minutes. The prescribed diet and exercise group (P) was given a jogging program based on the results of their exercise test data. Each subject performed the assigned activity every other day for 12 weeks.

Both exercise groups achieved significant increases in VO_2 max as a result of the diet and exercise regimen. VO_2 max increased by 9.11 % and 11.6 % in groups D and P, respectively. Resting metabolic rate increased by 10.1 % in group P. The data indicated that there was a tendency for RMR to rise in the subjects in the self selected exercise group (group D). Body composition changed in all groups: body fat decreased by 4.7, 5.2, and 7.1 % in groups C, D, and P respectively.

It is well established that if duration and frequency are held constant, improvements in VO_2 max will be related to the training intensity maintained by the subject. Lennon et al. [131] reported that the exercise groups had similar total caloric expenditures from exercise, however, the intensity of training was higher in the prescribed exercise group. The subjects in group P were expending approximately 100 kcal more per exercise session than those subjects performing self-selected exercise. There was a significant correlation between the percent change in VO_2 max and RMR for the female subjects. The authors suggested that the significant correlation between VO_2 max and RMR suggests that changes in RMR in females due to training may be dependent on the training intensity. In addition, Lennon et al. [131] suggested that perhaps the critical threshold of training intensity necessary to elicit RMR changes is that intensity which promotes a 11-12 percent increase in VO_2 max. Evidence to support this proposition was found in a careful examination of group D results. The subjects in group D had a 9.11 percent increase in VO_2 max, but did not manifest a significant increase in RMR. The changes in body composition further support the hypothesis that there is a critical training intensity necessary to elicit RMR changes. Specifically, the change in body fat was similar across all groups and yet only group P manifested significant changes in RMR. Thus, the results of this study suggest that perhaps there is a link between training intensity and RMR changes in women.

Tremblay et al. [9] performed two experiments designed to examine the relationship

between RMR and exercise. The first experiment was a cross sectional examination of the RMR of 59 young males, 20 trained and 39 untrained. The mean VO_2 max of the subjects was 50.8 ± 1.0 for the untrained and 68.4 ± 0.7 ml/kg/min for the trained. The RMR of the trained individuals was 11 percent greater than that of the untrained subjects.

In the second experiment Tremblay et al.[9] measured the changes in RMR, VO_2 max, and body composition of eight moderately obese women as a result of an 11 week aerobic exercise program. The aerobic exercise consisted of a variety of activities including cycling, swimming, jogging and aerobic dance. All exercise was performed at a mean intensity of 50% VO_2 max for five hours per week. Following 11 weeks of aerobic activity, there was a significant reduction in total body weight and fat mass in previously sedentary women. In addition the RMR of the subjects increased by 8% above pretraining values. Although the increase in RMR in women was consistent with the findings of Lennon et al. [131], it is somewhat surprising in light of the low intensity of activity that was performed by the subjects and the significant loss of body weight [9]. Based on the results of Lennon et al. [131] and Tremblay et al. [9] it would appear as though there is a threshold of training intensity and duration that is necessary to elicit increases in RMR in overweight individuals.

A later investigation by Nieman et al. [132] provided additional evidence that aerobic exercise may precipitate an increase RMR in untrained subjects. Twenty four female volunteers who were obese (20-40% overweight) were randomly divided into a nonexercise or exercise group. All subjects were fed a 1300 kcal/day diet for five weeks. The exercise group participated in a supervised walk/jog program 5 day per week for 45 minutes per day at 60% VO_2 max. The investigators estimated that approximately 320 kcal were expended per exercise session which was in line with the recommendations by the American College of Sports Medicine [138] for weight loss. At the conclusion of the study both groups demonstrated significant decreases in body

weight and body fat; however, there was no significant difference between the groups with respect to changes in body weight or composition. The exercise group had a 3.2% increase in VO_2 max in contrast to a 2.1 % decrease in the nonexercise group. Resting metabolic rate increased significantly (5.9%) in the exercise group following training. In contrast the nonexercise group manifested a slight decrease in RMR (2.2%). A significant correlation($r = 0.75$) was found between the per cent change in VO_2 max and RMR in the exercise group. The results of this study supported two important arguments: 1. Exercise may help prevent the typical decline in RMR that accompanies weight loss via caloric restriction alone, and 2. Exercise may help to increase RMR which would potentiate the negative energy balance that is required to lose weight.

The majority of data published has indicated that restricting caloric intake to 900 kcal/day or less will lead to a decrease in RMR. [129, 133, 134, 137, 140, 142, 143]. The role exercise has in reversing the decline in RMR is controversial. In a report by Henson et al.[140] seven moderately obese women volunteered for a nine week diet and exercise protocol. During the first three weeks the subjects had a caloric intake of 800 kcal/day. Exercise training was added in week four and the caloric restriction remained at 800 kcal/day. The exercise training consisted of 30 minutes of cycling at 70% VO_2 max five day per week. In spite of the significant increases in VO_2 max, the 13 % decrease in RMR following three weeks of dieting was not reversed by the addition of exercise to the regimen. The authors concluded that exercise of sufficient intensity and duration to increase VO_2 max did not appear to reverse the decrease in RMR due to caloric restriction.

In contrast to the results reported by Henson et al. [140] the results of studies by Mole et al. [129] and others [142, 143], suggested that the depression in RMR with caloric restriction can be reversed with aerobic exercise. Mole et al. measured the RMR, body composition and VO_2 max of four overweight females (body fat 32-42%) and

one male (body fat = 26.1%) who participated in a diet and exercise protocol. The experiment contained four consecutive stages: 1. a weight maintenance period, 2. a period of caloric restriction (500 kcal/day), 3. a period of caloric restriction and exercise and, 4. a short period of increased caloric intake (1200 kcal/day) without exercise [129]. During the caloric restriction and exercise period subjects engaged in 30 minutes of exercise per day at approximately 60% of VO_2 max. This was equivalent to a caloric expenditure of approximately 200 kcal/day. As expected, there were changes in total body weight and composition by the end of the study. It is interesting to note that the majority of the 6.6 kgs of weight loss was fat loss (5.5 kgs) with only a small decrease in lean body weight (1.1 kg). The resting metabolic rate was depressed 14 % from the pretraining values following the period of caloric restriction. Following the period of caloric restriction and exercise RMR values returned to their pre diet level. In spite of an increased caloric intake in period four the withdrawal of exercise seem to precipitate a fall in RMR. Specifically, RMR gradually decreased throughout the fourth period such that metabolic rate was depressed by approximately 10% from predicted values. The results of this study, and those of Hewitt et al. [142] and Schultz et al. [143] are clearly in contrast to the results of Henson et al. [140]. Part of the discrepancy among these studies may be related to the level of obesity of the subjects, the level and duration of severe caloric restriction, and the exercise protocol. Clearly, greater consistency in experimental protocols would allow researchers to determine if exercise can be used to offset the RMR changes reported to occur with severe dieting.

It is not known at this time whether intense physical training promotes an increase in RMR in females. Conversely, it does appear as if males adapt to intensive physical training with an increase in RMR. Additional studies that follow the energy intake and expenditure of female athletes on a cross sectional and on a longitudinal basis are needed. Currently, there is lack of information on the impact that mode of activity has on RMR.

In addition, it appears that exercise of sufficient intensity to promote increases in VO_2 max may be useful in offsetting the decline in RMR due to severe caloric restriction in untrained populations. However, additional data are needed to determine the appropriate levels of training and caloric restriction to insure safe weight loss.

Potential Mechanisms for Resting Metabolic Changes with Training

Several studies have illustrated a higher RMR in highly trained males [9, 12, 126, 136] and in untrained females [8, 129, 131, 132] following physical training. Despite the findings of these studies, the mechanisms which may be responsible for the changes in RMR have not been identified. The thyroid hormones are known to serve a major role in the energy metabolism of an individual. In spite of the variances noted in the RMR of trained and untrained males, Poehlman [12, 126] did not observe significant differences in the thyroid hormones of trained and untrained males. Data is currently lacking with regards to the thyroid hormone levels in untrained and trained females. Additional cross sectional and longitudinal studies are necessary before it can be determined whether thyroid hormones play a role in RMR changes.

Poehlman [20] speculated, based on the findings of Woo et al. [144], that perhaps RMR is a function of the total "energy flux" maintained by the individual. Woo et al. [144], engaged 6 males in an experimental protocol that consisted of randomly assigned periods that were varied in degree of energy balance. The conditions were : 1. sedentary, energy intake matched to energy expenditure (SD), 2. elevated energy intake and expenditure (155% above sedentary levels) (EE), and 3. energy intake at 155 % of sedentary but expenditure equivalent to sedentary levels (OF). The RMR during the EE and OF conditions was elevated by 10% and 5.5% respectively. These results implied that although a positive energy balance increased RMR, a greater increase in RMR was noted when both intake and expenditure of energy were increased [144]. It is also important to note that despite the RMR changes experienced by these subjects, there

were not significant differences across conditions in the plasma concentrations of the thyroid hormones T3 and T4.

In light of the high fluctuations in energy expenditure that are common among male endurance athletes it seems plausible that a high energy exchange may stimulate an elevation in RMR. Additional evidence to support this theory was found in the reports of Tremblay et al. [21] and Poehlman et al. [126]. Poehlman et al. [126] reported a significant correlation between energy intake and RMR ($r = 0.54$) and between RMR and $\text{VO}_2 \text{ max}$ ($r = 0.77$) in trained males. In addition, Tremblay et al. [21] found a significant decrease (6.6%) in RMR in trained males following a three day interruption in training. Subjects maintained a moderately high caloric intake of 3500 kcal per day following the initial measurement of RMR. In light of the fact that the subjects were not training, this caloric intake could be interpreted as excessive and therefore elicited an elevated RMR due to overfeeding. These data, in combination with the relationships reported by Poehlman et al. [126], lend support to the "energy flux" argument. Although, these studies do not provide conclusive evidence for the "energy flux" theory they do imply that the degree of energy intake and expenditure may influence RMR.

Additional mechanisms which may explain an elevation in RMR as a result of physical training include changes in the quantity of lean tissue (muscle specifically), increased protein synthesis and increased rates of substrate cycling due to an elevation in plasma catecholamines. Tremblay et al. [136] suggested that the differences in RMR recorded between trained and untrained individuals was not related to the fat free mass. Evidence to support this argument was found in the body composition and RMR measurements taken on a group of sedentary males, moderately and highly trained male athletes. The highly trained athletes had a RMR significantly greater than the sedentary or moderately trained individuals. In contrast, the moderately trained subjects had a greater body weight and fat free mass. Before a morphological explanation is ruled out, additional

studies should be completed on males and females across a continuum of fitness levels.

In his review Poehlman [20] speculated that a potential mechanism for the elevation of RMR in trained male athletes was an elevation in protein synthesis following exercise. There is some support for this suggestion due to the fact that protein synthesis is a metabolic process which is considered energetically "expensive." Although this explanation is tempting, additional studies of trained males and females are necessary to establish the relationship between protein metabolism and RMR.

Newsholme [145] suggested that the ultra slow phase of recovery period that has been observed 8-9 hours following exercise could be characterized as a period during which there is an elevated rate of substrate cycling occurring in the muscle. The "prolonged stimulation of cycles after exercise could be caused by a prolonged elevation in the level catecholamines" [145, page 404].

A more recent report by Sims and Danforth [146] suggested that perhaps skeletal muscle is the most likely site of facultative thermogenesis. The authors cited the findings of Fagher et al. [147] who reported that increased sympathetic nervous system activity was related to an elevation in the thermogenesis in muscle. These findings, coupled with Newsholme's [145] suggestion that muscle is the major site of "futile substrate cycling", tend to indicate that perhaps a relationship exists between the plasma catecholamines and the fat free mass (muscle) in regulating the RMR of trained individuals. Additional studies are necessary before it can be determined whether or not body composition and/or plasma catecholamines play a significant role in the regulation of RMR.

Energy balance, although simplistic in definition, is subject to the influence of numerous physiological, psychological and environmental factors. Exercise is just one of the factors which may have an impact on the direction of the energy balance equation. The influence exercise has on the energy balance equation may be a multifaceted phenomenon.

Specifically, there are several ways exercise may control the direction of the equation. First, exercise by itself will cause an increase in daily energy expenditure. Second, exercise may promote or increase RMR. Third, the thermogenic effect of food may be altered depending on the state of training and/or exercise prior to a meal. Last, exercise may play a significant role in the intake side of the equation by influencing appetite and/or hunger. Thus, a parametric study of the effects exercise has on the energy balance equation and its components is needed to understand the relationship between exercise, performance and weight loss. The present investigation was initiated to (a) determine the influence the mode of training has on the hunger response following an acute bout of exercise, (b) determine the relationship between the hunger response and urinary catecholamine levels, (c) determine if the hunger and /or catecholamine responses are related to subsequent 24 hour food intake and, (d) determine if there are differences in RMR between trained and untrained females and trained runners and swimmers.

CHAPTER III

Methods

Subjects

Thirty six females ranging in age from 17-24 volunteered to serve as subjects for the study. All subjects were healthy and had no clinical disease or were taking any medications, with the exception of aspirin on an occasional basis. The subjects ranged in weight from 71.7 to 46.8 kgs, and height from 152 to 175 cms. The subjects formed three groups, runners (R, n= 12), swimmers (S, n=12) and untrained (UT, n=12) based on either their mode of training or lack of training. The majority (10) of the runners were members of an intercollegiate cross country team that trained an average of 49 ± 7.1 miles (79.5 kilometers) per week, engaged in weight training three days per week for approximately one hour per session, and participated in deep and shallow water running two days per week for approximately twenty minutes a day. The two runners who were not members of the cross country team ran at frequencies and distances equal to the team members and also engaged in water activities one day per week. The swimmers were members of an intercollegiate swim team that averaged 14 .5 hours (approximately 37,000 meters) of swimming per week and participated in weight training three days per week for approximately one hour per session. In order to participate in the study, the runners and swimmers must have participated in their respective sport for a minimum of five years. The control subjects did not engage in regular physical activity and had not participated in regular activity for a minimum of six month prior to the study.

Orientation Sessions

Each subject signed an informed consent form and completed a medical history and

activity questionnaire (see Appendixes A, B, C), prior to participation in the study. Approval for all procedures was obtained from the Institutional Review Boards of The University of Tennessee and James Madison University.

Prior to any testing subjects were asked to attend two orientation sessions. The first session was designed to acquaint the subjects with the general purpose and design of the study. The subjects were not informed as to the potential relationship between each of the variables being measured. The second session was devoted to signing the informed consent, completing the health history questionnaire, and the subjects were given an introduction to the laboratory procedures for the assessment of body composition, resting metabolic rate, maximal oxygen consumption, and the protocol for the experimental and control sessions. Each subject was given written instructions (see Appendixes D and E) for each of the laboratory tests. In addition subjects were called 12-24 hours prior to each testing date to remind them of the special procedures that were to be followed prior to testing. Separate orientation sessions were held for each group (During the orientation session for the runners and swimmers the subjects were allowed to examine plastic samples of typical serving sizes of food, to assist them in providing an accurate recall of twenty-four hour food intake). The subjects were reminded several times throughout the study to maintain their current eating and exercise habits. To standardize the influence of the menstrual cycle all measurements were completed during the follicular phase (days 2 to 12), and in the afternoon, with the exception of body composition, which was completed in the morning without regard for the menstrual cycle phase. The subject's body weight was measured prior to the assessment of body composition, maximal oxygen consumption , and resting metabolic rate.

Body Composition

Prior to the assessment of body density all subjects were weighed on a physician type

scale that was calibrated before each test session. Before weighing (dryland and water) all subjects were asked to void completely. The body density of all subjects was determined via underwater weighing. All subjects were weighed in the shallow end of a swimming pool using a Chatillon scale. The underwater weight was determined from the first three trials to have a range of 100 grams or less [148]. Residual volume was measured in the respiratory therapy unit of a local hospital using the helium dilution method. Body fat was calculated using the Siri [149] equation.

Resting Metabolic Rate

The resting metabolic rate (RMR) of all subjects was measured using a computerized metabolic cart (MMC Horizon , SensorMedics, Yorba Linda, California). Subjects were instructed to abstain from all caffeine, and fast for three hours prior to the measurement of RMR. In addition, the subjects were instructed to abstain from physical activity a minimum of 20 and a maximum of 26 hours prior to the measurement. Upon arrival at the laboratory subjects rested for twenty minutes in a reclined position and precautions were taken to minimize any external environmental disturbances that might influence the RMR measurement. Following the twenty minute rest period, subjects were fitted with a face mask (for the collection of expired gases) and instructed to remain at rest while breathing through the mask. Expired gases were collected for twenty minutes. Oxygen consumption, carbon dioxide production and ventilation were computed using the Nutritional Assessment Critical Care software of the MMC Horizon System. Oxygen consumption was converted to kilocalories per day using a variation of the modified Weir (Sensormedics, Yorba Linda) equation. The gas analyzers were calibrated prior to the beginning of each test. The mean oxygen consumption and carbon dioxide production were used to calculate mean daily caloric expenditure. RMR, expressed as $\text{kcal}\cdot\text{kg}^{-1}$ lean body weight (LBW), was calculated by dividing the mean daily caloric expenditure by the subject's lean body weight. The RMR measurements for

all subjects was determined during the afternoon (1200-1700) hours.

Maximal Oxygen Consumption

The maximal oxygen consumption of the runners and swimmers was determined. The maximal aerobic power ($\text{VO}_2 \text{ max}$) of the runners was assessed using a progressive and continuous treadmill test designed to bring the subjects to max in 8-10 minutes. Subjects started at a speed that approximated their mean daily training pace. Prior to the determination of $\text{VO}_2 \text{ max}$ all equipment was calibrated and the subjects were familiarized with the testing equipment and procedures. The $\text{VO}_2 \text{ max}$ of the swimmers was determined using a graded tethered swimming test against a progressively increasing resistance. Markers were placed at 3 foot intervals on the bottom of the pool, and all subjects started with approximately the same resistance in the tether cord. Oxygen consumption was determined using a metabolic cart (MMC Horizon SensorMedics, Yorba Linda, California).

Experimental (Exercise) and Control Sessions

To evaluate the relationship between mode of physical activity, hunger and the catecholamine response, subjects participated in an experimental and control session. Both sessions were completed during the follicular phase of the ovarian cycle (day 2-12). The order of the sessions was randomly assigned in a counterbalanced manner. The experimental and control sessions were separated by a minimum of 72 and a maximum of 120 hours. During the experimental session the subjects ran or swam at an intensity of 65-70% of $\text{VO}_2 \text{ max}$ (80-85% of maximum heart rate). The duration of the sessions was determined by calculating the time necessary for each subject to expend approximately 600 kcal. The caloric expenditure for each subject was based on the oxygen consumption value that represented 65-70% of their $\text{VO}_2 \text{ max}$. The caloric expenditure per minute was approximated. Test time was calculated as the amount of time necessary to expend approximately 600 kcal per session. This caloric expenditure

was considered to approximate the normal daily caloric expenditure by these female athletes. All experimental and control session were conducted in the afternoon (1200-1800 hours). The protocol for the experimental and control sessions is illustrated in Figure 1.

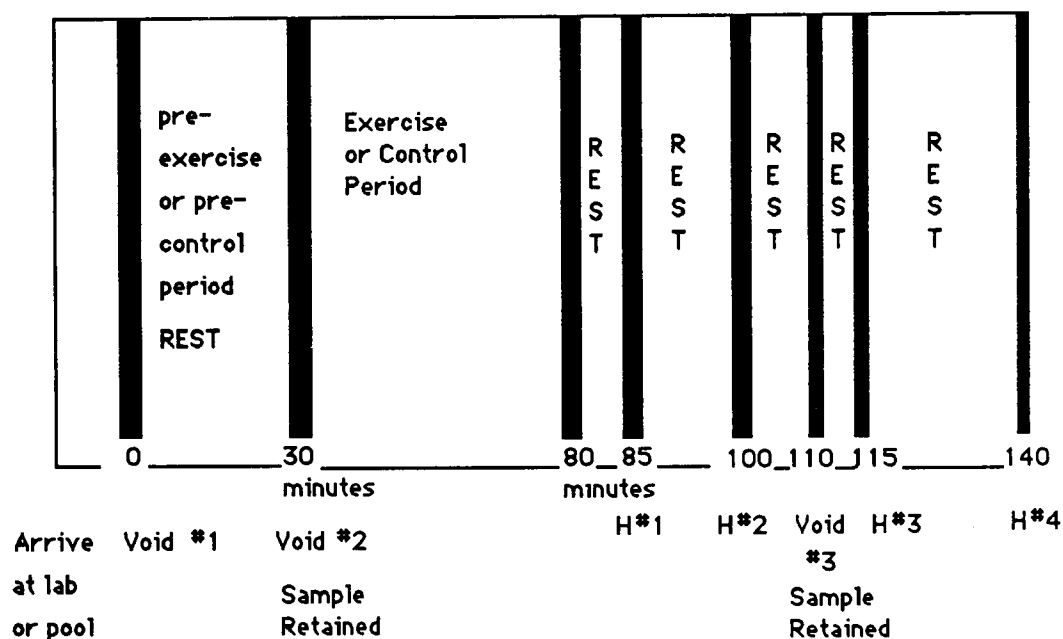


Figure 1 Protocol for Experimental and Control Sessions

Urinary Catecholamines

Upon arrival at the laboratory each subject was asked to void completely. The time was recorded, and this urine sample was discarded. Each subject then rested for a period of no less than 30 minutes. Following the rest period subjects were asked to void again. The pH of the urine sample was adjusted to 2.5-3.0, and the volume of the sample and time of voiding were recorded (this sample became the rest sample). Each urine sample was stored and frozen in a polyethylene bottle for later analysis. After providing the urine sample, subjects ran or swam for a time period long enough to result in a caloric expenditure of 600 ± 3.8 kcal. The runners performed continuous exercise during the

experimental session on a motor driven treadmill at a speed and grade that elicited a 65-70% VO_2 max response. The swimmers swam continuously in a 25 yard pool at a speed equivalent to 65-70% VO_2 max. The heart rate of the subjects was monitored throughout the experimental session to ensure that an intensity of 80-85% maximum heart rate was maintained for the duration of the exercise session. Five, twenty, thirty-five and sixty minutes following completion of the experimental session, the subjects were asked to rate their hunger on a 9 point paper and pencil scale (see Appendix G) ranging from 1 (extremely full) to 9 (extremely hungry) [14, 42]. Thirty minutes after completion of the run or swim portion of the session, subjects were asked to void again. The pH of the sample was adjusted to 2.5-3.0, and the volume and time of voiding were recorded (this became the exercise sample). Water was available through out the exercise and rest periods. Twenty-four hours following the experimental and control sessions the subjects were telephoned by a graduate student in dietetics and asked to recall the type and amount of food and beverages they had consumed in the 24 hours following the conclusion of the experimental and control sessions. The caloric content and composition of the athletes diet was determined using a computer software program (Food Processor, ESHA Research, Salem, Oregon.).

During the control session the subjects were asked to sit quietly and minimize their movements for the time period equivalent to their exercise period. The subjects were allowed to read and study. Not all subjects complied with the investigator's instructions and several appeared to be very "fidgety" at times during the control session. The protocol for the urine collection, hunger ratings and food intake were identical to the protocol followed for the experimental session. Upon completion of the control session, subjects were instructed to abstain from physical activity for 24 hours.

The absorption of the catecholamines (CA) were performed according to the method of Viktora et al. [150] with modifications (see Appendix F). A known sample of

norepinephrine (NE) was added to each urine sample to determine recovery. All urine samples were analyzed in duplicate. Total catecholamines were determined by oxidizing the eluate at pH 6.5. Epinephrine was determined by oxidizing the eluate at pH 2.85. The values for epinephrine (E) and norepinephrine (NE) were corrected to 100% recovery. The NE was calculated according to Euler and Floding [151]. Exercise and control excretion of norepinephrine and epinephrine were calculated and reported in nanograms per minute (see Appendix F for calculation formula).

Statistical Procedures

All statistical test were performed using SAS [152], a statistical analysis system. A two-way repeated measures ANOVA was used to determine if there were differences in hunger ratings between the control and experimental sessions for the runners and swimmers. A correlation matrix was developed to assess the relationship between urinary catecholamines and the hunger response. Multiple linear regression (stepwise selection) was used to determine the best predictors of 24 hour food intake following exercise (experimental) and rest (control). A one-way ANOVA was used to determine if there were significant differences in RMR between runners, swimmers and controls. Significance was accepted at the 0.05 level. A priori orthogonal contrasts were used to test for differences between the trained (runners & swimmers) and the untrained (controls).

CHAPTER IV

Results

Physical and Descriptive Characteristics of the Subjects

Twelve runners, twelve swimmers, and twelve untrained subjects completed the study. The values for physical, descriptive and training characteristics of the subjects are presented in Table 1.

Hunger

The runners and swimmers were asked to rate their hunger at 5, 20, 35, and 60 minutes following the experimental and control sessions. The mean hunger ratings are listed in Table 2 and presented graphically in Figure 2. A two-way repeated measures ANOVA (see Table 3) indicated that there were no differences between the runners and swimmers hunger ratings following the experimental session. Although, there were no differences between the groups' perception of hunger following exercise, there was a significant ($F=37.98$, $p < 0.0001$) increase in the hunger response with time. A hunger rating difference was generated for each subject by subtracting the experimental hunger rating from the control rating for each hunger rating interval. The hunger difference scores are illustrated in Figure 3. A two-way repeated measures ANOVA (see Table 4) was used to determine whether or not the difference in hunger ratings were distinct for runners and swimmers and across time. There were no significant differences ($F= 0.16$, $p= 0.695$) in the hunger difference response between the two groups. However, there was a significant ($F= 3.04$, $p= 0.0348$) decrease in the hunger difference for both groups with time. In addition there was evidence that ($F= 0.53$, $p= 0.6648$) the runners and swimmers had similar patterns of hunger ratings across time.

Table 1 Physical, Descriptive and Training Characteristics of the Subjects

Variable	Runners		Swimmers		Controls	
	Mean	SD	Mean	SD	Mean	SD
n	12		12		12	
Age (years)	19.80	± 1.6	19.40	± 1.3	21.00	± 1.1
Height (cm)	164.2	± 5.6	163.8	± 6.6	165.4	± 6.0
Weight (kgs)	53.80	± 3.8	61.30	± 5.0	59.70	± 8.5
Body fat (%)	14.22	± 4.2	20.40	± 2.9	26.50	± 4.3
LBW (kgs)	46.10	± 3.2	48.70	± 3.4	43.80	± 5.8
VO ₂ max (ml·kg ⁻¹ min ⁻¹)	60.90	± 6.2	52.50	± 4.0	----	----
VO ₂ max (l/min)	3.260	± 0.31	3.300	± 0.37	----	----
Training (km/week)	79.50	± 7.5	38.30	± 1.3	----	----

Table 2 Mean Hunger Ratings

		Groups					
Session	Time	Runners			Swimmers		
		Mean		S.D.	Mean		SD
Control	5	5.67	±	0.89	6.25	±	0.97
Control	20	5.92	±	0.90	6.58	±	0.79
Control	35	6.58	±	0.79	6.92	±	0.79
Control	60	7.17	±	0.58	7.50	±	0.52
Experimental	5	4.92	±	1.24	4.92	±	1.68
Experimental	20	5.25	±	1.06	5.75	±	1.60
Experimental	35	6.08	±	1.24	6.42	±	1.62
Experimental	60	6.92	±	1.00	7.0	±	1.04

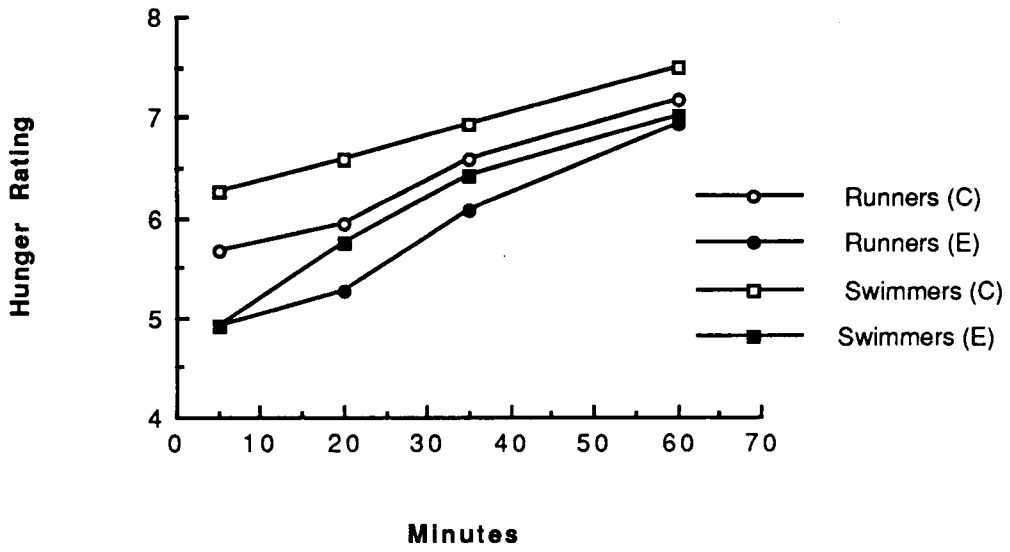


Figure 2 Mean Hunger Ratings

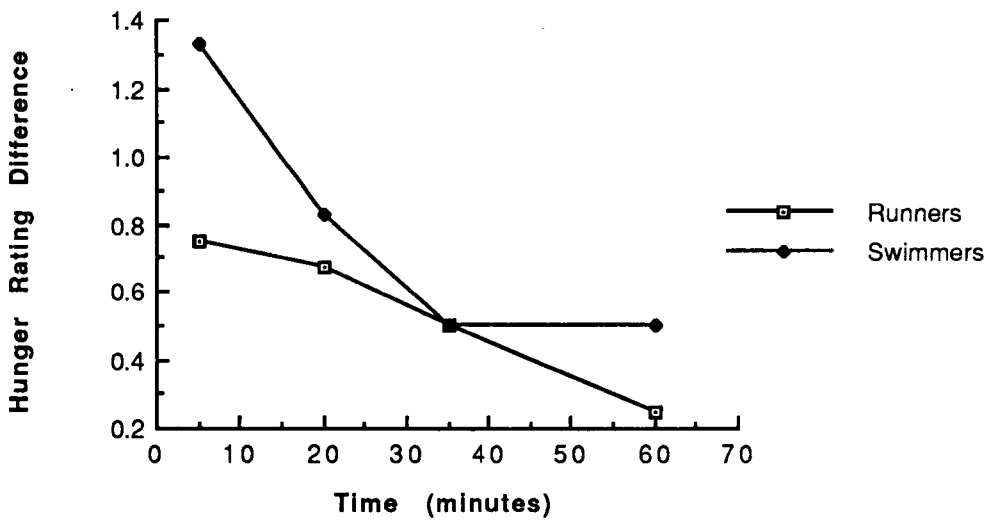


Figure 3 Hunger Rating Difference (control-experimental rating)

Table 3 Repeated Measures Analysis of Variance for Runners and Swimmers on Experimental Hunger Ratings.

Source	SS	df	MS	F
<i>Between Subjects</i>				
Mode	1.260	1	1.260	0.22
Error	124.145	22	5.642	
<i>Within Subjects</i>				
Time	56.864	3	18.954	37.98*
Time*Mode	0.9479	3	0.3159	0.63
Error (Time)	32.937	66	0.4990	

* $p < 0.0001$

Table 4 Repeated Measures Analysis of Variance for Runners and Swimmers on Hunger Differences

Source	SS	df	MS	F
<i>Between Subjects</i>				
Mode	1.500	1	1.500	0.16
Error	211.33	22	9.606	
<i>Within Subjects</i>				
Time	6.25	3	2.083	3.04*
Time*Mode	1.083	3	0.3611	0.53
Error (Time)	45.16	66	0.6843	

* $p \leq 0.05$

Catecholamines and Hunger

Urinary excretion rates of epinephrine and norepinephrine were determined for each subject for both the control and experimental sessions. The mean epinephrine and norepinephrine excretion values are reported in Table 5. The strength of the relationship between the hunger responses and urinary catecholamines was evaluated by the correlation coefficients. The norepinephrine, and epinephrine levels were correlated with the hunger responses at all four time periods following the control and experimental sessions. The correlation coefficients for the control session (see Table 6) indicated that there was a significant inverse relationship ($r = -0.45$, $p = 0.04$ and $r = -0.49$, $p = 0.02$) between the hunger rating at 20 and 35 minutes following the control session, and the urinary levels of norepinephrine. The relationship between the subject's perception of hunger and norepinephrine at 20 and 35 minutes following the control session are illustrated in Figures 4 and 5.

The correlation coefficients for the experimental hunger responses at 5, 20, and 35 minutes indicated a significant inverse relationship ($r = -0.54$, $p = 0.009$, $r = -0.58$, $p = 0.004$, and $r = -0.68$, $p = 0.0005$) between hunger and experimental norepinephrine values (see Table 7). The results indicated that as the levels of norepinephrine increased, the hunger response following exercise decreased. The relationship between perception of hunger and experimental excretion rates of norepinephrine are illustrated in Figures 6-8.

Appetite (Food Intake) and Catecholamines

Appetite following the both the control and experimental session was assessed through the use of a 24 hour dietary recall that was obtained approximately 24 hours following the conclusion of the experimental and control sessions. The caloric content of the athletes' diets was determined using a computer software program (Food Processor, ESHA Research, Salem, Oregon). Correlation coefficients were used to assess the

Table 5 Experimental and Control Urinary Catecholamines

Session	E Excretion ng/min	NE Excretion ng/min
Control	18.52 ± 18.66	25.06 ± 18.75
Experimental	36.60 ± 33.90	84.16 ± 28.73

Values are mean ± SD, N = 20, E = epinephrine, NE = norepinephrine

Table 6 Correlation Coefficients for Urinary Catecholamines (epinephrine, norepinephrine), Hunger(H) and Food Intake Following the Control (C) Session.

	Epi	Norepi	HC5	HC20	HC35	HC60	FIC
Epi		-0.09	0.11	-0.11	-0.35	0.00	0.20
Norepi			-0.29	-0.45¥	-0.49¥	-0.42	-0.21
HC5				0.83+	0.78+	0.51*	-0.02
HC20					0.76+	0.69+	0.26
HC35						0.39	0.18
							0.33

¥ p ≤ 0.05

* p ≤ 0.01

+ p < 0.0001

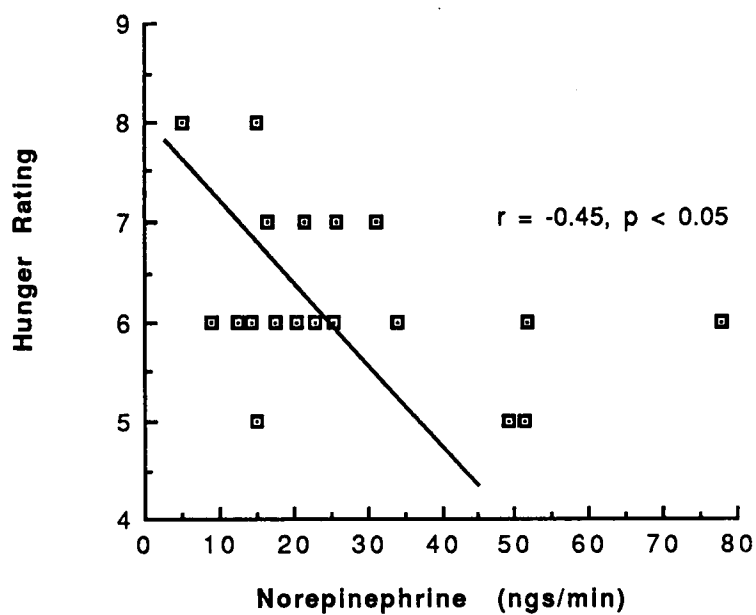


Figure 4 Correlation of Control Hunger Rating 20 Minutes to Norepinephrine

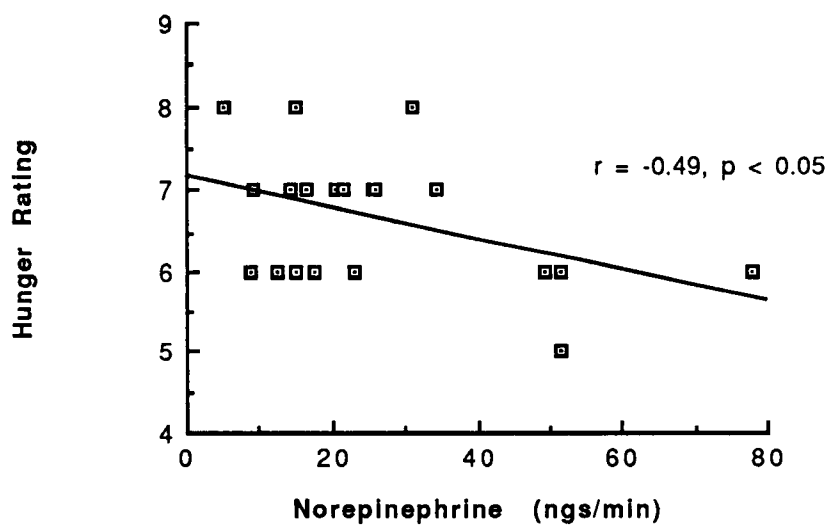


Figure 5 Correlation of Control Hunger Rating 35 Minutes to Norepinephrine

Table 7 Correlation Coefficients for Urinary Catecholamines (Epi, Norepi), Hunger (HE) and Food Intake (FIEX) for the Experimental Session (E).

	Epi	Norepi	HE5	HE20	HE35	HE60	FIEX
Epi		-0.16	0.17	0.28	0.15	-0.13	-0.13
Norepi			-0.54*	-0.59*	-0.68†	-0.38	0.45¥
HE5				0.83+	0.75+	0.51*	0.03
HE20					0.90+	0.63†	0.15
HE35						0.68†	0.19
HE60							0.19

¥ $p \leq 0.05$

* $p \leq 0.01$

† $p \leq 0.001$

+ $p \leq 0.0001$

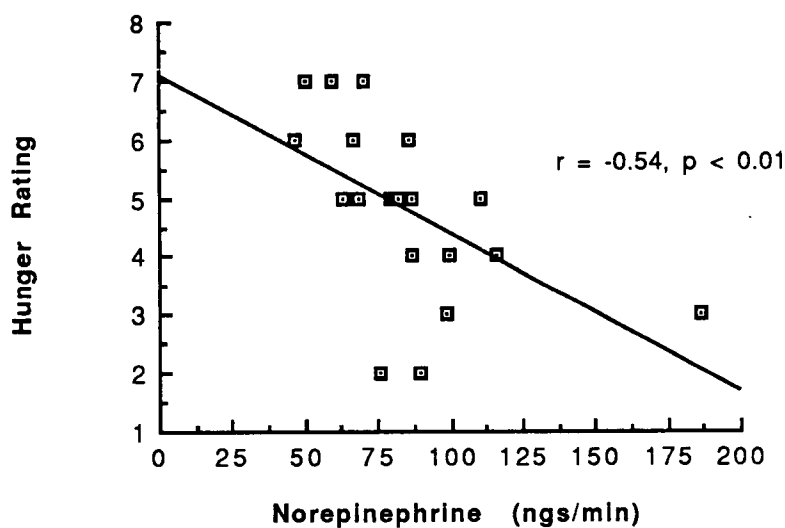


Figure 6 Correlation of Experimental Hunger Rating 5 Minutes to Norepinephrine

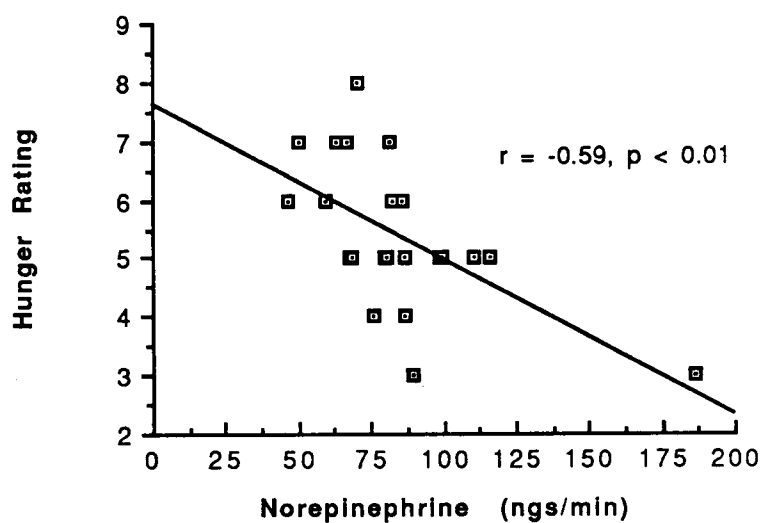


Figure 7 Correlation of Experimental Hunger Rating 20 Minutes to Norepinephrine

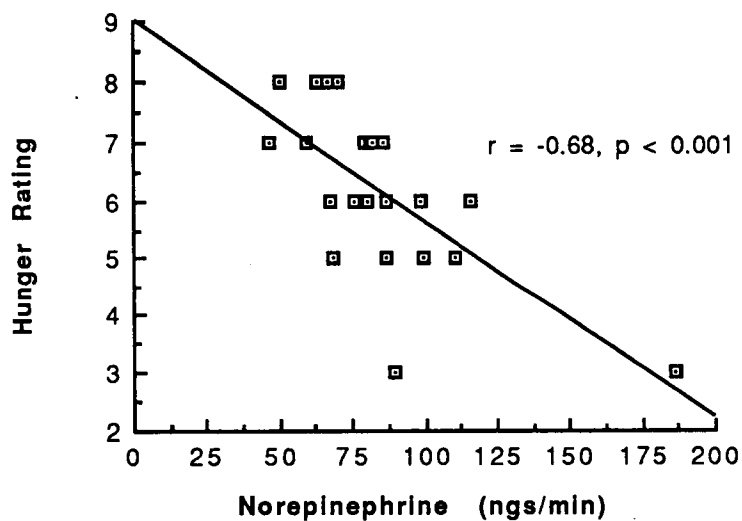


Figure 8 Correlation of Experimental Hunger Rating 35 Minutes to Norepinephrine

relationship between food intake and catecholamines. The mean values for appetite (food intake) are presented in Table 8. A two-way repeated measures ANOVA (see Table 9) was used to determine whether swimmers and runners differed in food intake and whether there were differences between the control and experimental sessions.

The hunger response following the experimental and the control sessions was not related to food intake. The results also indicated that catecholamine levels were not related to food intake following the control session. However, there was a positive relationship ($r = 0.45$, $p = 0.03$) between food intake and norepinephrine following the experimental session, as illustrated in Figure 9.

A stepwise selection regression analysis was performed to determine whether the hunger ratings, catecholamines and mode of training had an impact on food intake following the control and the experimental sessions. The model tested is written below:

$$Y (\text{food intake}) = \beta_0 + \beta_1 (\text{mode}) + \beta_2 (\text{norepi}) + \beta_3 (\text{epi}) + \beta_4 (H1) + \beta_5 (H2) + \beta_6 (H3) + \beta_7 (H4)$$

Mode = runner (0) or swimmer (1), norepi= norepinephrine, epi= epinephrine

H1,2,3,4 = Hunger rating at 5, 20, 35 and 60 minutes respectively.

Norepinephrine ($R^2 = 0.20$, $F = 5.11$, $p = 0.03$) was the first variable added to the model indicating that it explained the greatest amount of variation when using catecholamines and hunger to predict food intake following the experimental session. The addition of the second hunger rating (20 minutes) following exercise had a significant effect ($R^2 = 0.49$, $F = 10.77$ (partial), $p = 0.0039$) on the model and was a significant predictor of experimental food intake. The remaining variables in the model did not meet the 0.1500 significance level for entry into the model. The R squared values, coefficients, the standard errors and probabilities for the regression equation are presented in Table 10.

Table 8 Mean Food Intake (kcal/24 hours) Following the Control and Experimental Sessions.

	Session	
	Control	Experimental
Runners	1723.5 ± 710	1988.58 ± 713
Swimmers	1996.00 ± 499	2267.83 ± 905

Table 9 Repeated Measures Analysis of Variance for Food Intake (Experimental and Control)

Source	SS	df	MS	F
<i>Between Subjects</i>				
Mode	913284.18	1	913284.18	1.27
Error	15787310.29	22	717605.01	
<i>Within Subjects</i>				
Sessions	864838.52	1	864838.52	2.67
Sessions*Mode	136.69	1	136.69	0.00
Time (Error)	7131559.29	22	324161.78	

Table 10 Stepwise Regression for Experimental Food Intake

Variable	R2 partial	R2 model	β	SE	F	p
Norepi	0.2037	0.2037	23.17	5.55	17.32	0.0005
HE2	0.2881	0.4917	404.72	123.33	10.77	0.0039

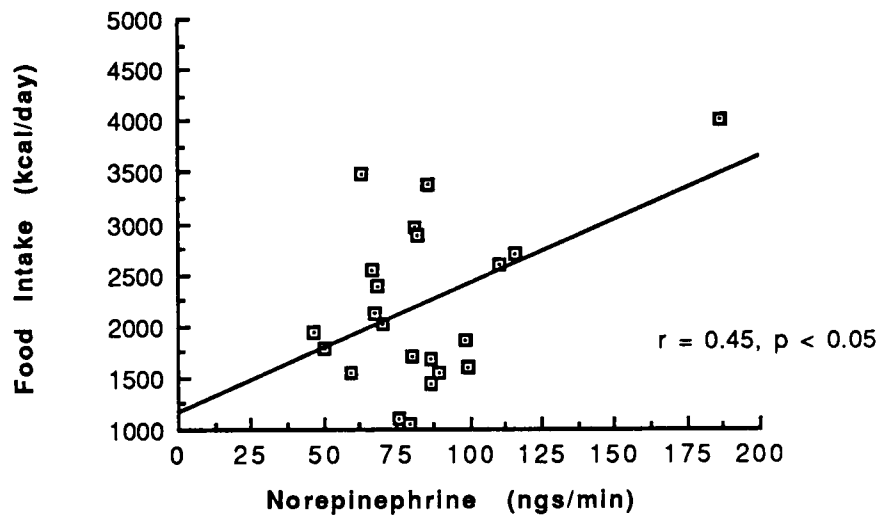


Figure 9 Correlation Experimental Appetite to Norepinephrine

The stepwise selection regression analysis performed on the control session data, indicated that the fourth hunger rating (60 minutes) explained the largest portion of the variance in food intake using the model proposed above ($R^2 = 0.19$, $F = 4.48$, $p = 0.04$). The remaining variable in the model did not meet the 0.1500 significance level for entry into the model. The R squared values, regression coefficients, standard errors and probabilities are presented in Table 11.

Resting Metabolic Rate

Mean values for resting metabolic rate (RMR) are presented in Table 12. One-way ANOVA (see Table 13-15) tests were used to determine whether or not there were differences in resting metabolic rate (RMR) among the groups. Resting metabolic rates expressed in $\text{kcal}\cdot\text{kg BW}^{-1}\cdot\text{day}^{-1}$, kcal/day and kcal/kg LBW were compared among the three (runners, swimmers and untrained) groups. Orthogonal contrasts were performed to determine whether or not significant differences existed between the trained (runners and swimmers) and untrained subjects, the runners and swimmers.

When RMR was expressed relative to body weight (kgs) there were no differences ($F = 0.06$) among the groups. In addition, the orthogonal contrasts indicated that there were no differences in RMR between the trained and the untrained ($F = 0.07$) subjects or between runners and swimmers ($F = 0.06$). Resting metabolic rate was significantly ($F = 3.71$, $p = 0.0353$) different among the three groups when expressed in kcal/day . Orthogonal contrasts indicated that there were no differences ($F = 1.39$, $p = 0.2466$) between the trained and untrained subjects, however, significant ($F = 6.02$, $p = 0.0196$) differences did exist between the runners and swimmers.

To account for differences in lean body weight, RMR was expressed relative to the subject's lean body weight. The results indicated that there were significant ($F = 10.13$, $p = 0.0004$) differences among the groups in RMR expressed as kcal/kg LBW .

Table 11 Stepwise Selection Regression for Control Food Intake.

Variable	R2 partial	R2 model	β	SE	F	p
HC 60 min	0.1907	0.1907	527.30	249.21	4.47	0.0478

Table 12 Mean Values of Resting Metabolic Rate.

Variable	Runners	Swimmers	Untrained
RMR (kcal·kg ⁻¹ ·day ⁻¹)	27.34 ± 3.81	27.08 ± 2.18	27.48 ± 1.99
RMR (kcal/day)¥	1470.9 ± 204	1661.8 ± 121	1645.7 ± 227
RMR (kcal/kg LBW)*	31.86 ± 3.65	34.19 ± 2.50	37.70 ± 3.28

All values expressed as the mean ± SD, LBW = lean body weight (kgs).

¥ p ≤ 0.05

* p ≤ 0.001

Table 13 Analysis of Variance Table for Resting Metabolic Rate (kcal·kg⁻¹·day⁻¹)

Source	SS	df	MS	F
Mode	0.99555	2	0.497777	0.06
Error	255.4141	33	7.739823	
Total	256.4097	35		
Contrasts				
T vs. UT	0.568888	1	0.568888	0.07
R vs. S	0.426666	1	0.426666	0.06

Table 14 Analysis of Variance for Resting Metabolic Rate (kcal/day)

Source	SS	df	MS	F
Mode	268960.22	2	134480.11	3.71*
Error	1196855.42	33	36268.35	
Total	1465815.64			
Contrasts				
T vs. UT	50456.05	1		1.39
R vs. S	218504.17	1		6.02¥

* p = 0.0353

¥ p = 0.0196

Table 15 Analysis of Variance Table for Resting Metabolic Rate (kcal/ kg LBW)

Source	SS	df	MS	F
Mode	207.24	2	103.62	10.13¥
Error	337.66	33	10.23	
Total	544.90			
Contrasts				
T v. UT	174.60	1		17.06*
R vs.S	32.64	1		3.19

¥ p = 0.0004

* p = 0.0002

The contrast between the trained and untrained indicated a significant ($F = 17.06$, $p = 0.0002$) difference in RMR in kcal/kg LBW. In addition, there was no significant ($F = 3.19$, $p = 0.0833$) differences between the runners and swimmers RMR when expressed in kcal/ kg LBW.

CHAPTER V

Discussion and Conclusions

Hunger and Mode of Exercise

The first purpose of this investigation was to examine the hunger response of trained female runners and swimmers following an acute bout of exercise that was equivalent in intensity and energy expenditure. The relationship between exercise, appetite and hunger is somewhat difficult to discern due to the lack of consistency in the use of the terms, hunger and appetite. It is not uncommon for the terms appetite and hunger to be used interchangeably. Appetite is accurately defined as the state of willingness to accept food, or the desire to satisfy the natural needs of the body, it is usually reported as the number of kilocalories consumed in a twenty four hour period [22]. Hunger is an internal state or level that disposes an animal or person to eat [22]. The majority of studies that have measured hunger have used a rating scale [14, 41, 42]. The scale is a subjective evaluation by the subject, and in reality is the subject's perception of his/her hunger.

The subjects in the present study were asked to rate their hunger four times (5, 20, 35, 60 minutes) following an acute bout of exercise performed at 65-70% of their VO_2 max. Each subject ran or swam for a predetermined time and intensity. Subjects expended approximately 600 kilocalories during the exercise session. Mode did not influence the hunger response following exercise since there was no significant differences between the runners' and swimmers' hunger ratings. Although the mode of exercise did not appear to have an influence on hunger ratings following exercise, there was a significant effect due to time. The mean difference in hunger ratings following exercise for the runners decreased from 0.75 ± 1.86 at five minutes to 0.25 ± 1.06 at

60 minutes. The mean difference in hunger ratings for the swimmers decreased from 1.33 ± 2.10 at five minutes to 0.50 ± 1.00 at 60 minutes. Thus, with time the hunger responses following the control and experimental session approximated each other to a greater degree. The actual ratings following the control session for the runners increased from 5.67 ± 0.89 at five minutes to 7.17 ± 0.58 at sixty minutes. The swimmers response was similar with ratings increasing from 6.25 ± 0.97 at five minutes to 7.50 ± 0.52 at sixty minutes. The ratings following exercise increased from 4.92 ± 1.24 at five minutes post exercise, to 6.92 ± 1.00 at 60 minutes post exercise for the runners and from 4.92 ± 1.68 to 7.0 ± 1.04 for the swimmers. The increase in hunger with time was significant for both groups.

Few studies have examined the hunger response following an acute bout of exercise. Of those, the majority have used males as subjects with the mode of activity being cycling [14, 42]. The hunger rating scale used in the present study was similar to the one used in the studies conducted by Maxwell [42] and Thompson et al. [14]. The pattern of the response in the present study was similar to the responses reported by Thompson et al. [14] for high intensity exercise (68% VO₂ max). Specifically, subjects felt "satisfied" (a rating of 4.92), neither hungry or full, immediately (5 minutes) following exercise at an intensity of 65-70% VO₂ max. In addition, time appeared to precipitate an increase in the subject's perception of hunger. However, in light of the similarity in responses between the present study and the results of Thompson et al. [14] it would appear as though the intensity, and not the mode, of exercise influences an individual's perception of hunger. Moreover, gender does not appear to affect a subject's perception of hunger following exercise. Female subjects in the present study, and those from an earlier investigations by Reger, Allison, and Kurucz [41], had similar hunger responses to the male subjects of previous investigations [14, 42] .

The evidence currently available suggests that exercise performed at an intensity of

65 -70% VO_2 max may suppress hunger for a brief period [14, 41]. The results of the present study, and those of Thompson [14], suggest that the suppressive effect may only last five minutes. These results are in contrast to the extended (up to 120 minutes) suppression noted by Maxwell [16, 42]. What remains to be determined is the mechanism responsible for the acute suppression of hunger following aerobic exercise. Various physiological and psychological mechanisms have been suggested for the reduced hunger and appetite responses associated with exercise. One of the physiological factors that has been proposed is an increased level of catecholamines [Guilland, 1988 #54, 43, 54, 56, 57].

Hunger and Catecholamines

The second purpose of this study was to examine the relationship between urinary catecholamines and the subject's perception of hunger. Hunger ratings following the control and experimental sessions were correlated to urinary levels of norepinephrine and epinephrine. There was a significant inverse relationship between the hunger ratings and norepinephrine at twenty and thirty-five minutes following the control session. However, epinephrine levels were not related to perception of hunger following the control session. Concentrations of norepinephrine were significantly related to hunger ratings at 5, 20, and 35 minutes following the experimental session. The presence of an inverse relationship between hunger and norepinephrine indicated that as the level of norepinephrine increased, the perception of hunger decreased for a period of 5-35 minutes following the control session and submaximal exercise. This data suggests a "metabolic link" between activity of the autonomic (sympathetic) nervous system and subsequent perception of hunger. However, one must be extremely cautious in the interpretation of the correlations, remembering that they do not confirm a cause and effect and, at the very best, they suggest a relationship that should be examined further.

Prior to drawing any conclusions from these data it is important to remember that

the measured levels of norepinephrine provide a glimpse of the activity of the autonomic nervous system during the exercise that preceded the measurement of hunger ratings. Thus, it seems prudent prior to drawing any conclusions or proposing any implications of these data, to discuss the variables that can influence the catecholamine response and the hunger rating. Although the correlation data indicated a strong relationship between norepinephrine and hunger one must be reminded of the limitations of the hunger rating. Specifically, the rating is a subjective perception of hunger by each subject that may be influenced by an assortment of physiological, psychological, and environmental variables. Regardless of the subjective nature of the hunger rating, it has been established that "hunger is not an all or none phenomenon and it can be graded in magnitude; it therefore has motivational properties that can be measured by amount and rate of consumption, or rate of performance or an arbitrary behavior to obtain food" [22, p. 373]. In spite of the limitations of the hunger measurement, this study suggested that a relationship may have existed between an individual's perception of hunger and a measurable physiological variable (ie. catecholamines), that is known to reflect the metabolic state of the body. The potential relationship between hunger and catecholamines is certainly intriguing and complex, and requires an examination of the variables that influence the catecholamines response during exercise.

In humans, epinephrine constitutes the major fraction (greater than 90%) of the total catecholamines released from the adrenal medulla into the circulation "[153]. In contrast to the origin of epinephrine, norepinephrine levels are indicative of the activity of the sympathetic peripheral nerve terminals. The values measured from the plasma, or urine, reflect the amount of leakage of norepinephrine as a neurotransmitter. Furthermore, the levels of epinephrine observed are believed to be an indicator of the amount of emotional stress, where as norepinephrine values represent the amount of physical stress [154].

In light of the information presented above it is not unreasonable to expect large increases in norepinephrine with exercise and moderate changes in epinephrine. The increase in circulating levels of catecholamines are dependent on the intensity and duration of exercise. Norepinephrine levels have been reported to increase 50-600 percent as a result of aerobic exercise [154-159]. The majority of studies report a gradual linear increase in norepinephrine up to 65-75% of VO_2 max [154, 156-159]. Workloads greater than, or equal to, 75% of VO_2 max appear to elicit a rapid and exponential increase in norepinephrine [156-158]. In the present study norepinephrine and epinephrine increased 236 (25.06 to 84.15 ngs/min) and 97 (18.52 to 36.60 ngs/min) percent respectively. These changes are in agreement with the data reported by a number of authors using similar exercise intensities [156-159].

It is important to keep in mind that the absolute values that are reported for catecholamines may differ between active and sedentary populations due to the effect that training appears to have on resting, submaximal and maximal secretory levels of catecholamines [160-162]. The evidence for a training effect on resting and exercise levels of catecholamines is variable [160, 161, 163]. In general, trained individuals will demonstrate lower levels of norepinephrine during submaximal exercise. The epinephrine response to training is not clearly defined at this time due to the conflicting research [160, 163]. Thus, while the norepinephrine levels in the present study are somewhat low in relation to an earlier study of untrained males [155], they are in agreement with other values reported for trained male subjects [158, 164]. The resting levels of catecholamines observed in the present study are in close agreement with the values [165] reported for female basketball players and track and field athletes. However, the post exercise epinephrine values observed in the present study are somewhat lower than those reported by Pierce et al. [165]. and the norepinephrine values are somewhat higher. The norepinephrine differences are most likely related to

the greater exercise duration in the present study . The disparity in epinephrine values are most likely related to differences in training intensity. Moreover, the subjects in the present study were highly trained females. Therefore, one might expect that, due to their high training state, norepinephrine levels would be lower than the moderately trained males subjects of Howley's investigation [158]. In contrast the epinephrine values are somewhat higher values than those previously reported for males by Howley [158], and in close agreement with the values reported by Cronan and Howley [155]. The variability of the epinephrine response observed in the present study is not a new finding [158]. The analytical procedures were extremely reproducible, with the exception of two runners and two swimmers whose control data was not used in the statistical analysis . In view of the high reproducibility of the analytical procedures other factors may be precipitating an elevated epinephrine response. It is well established that stressful situations can influence the catecholamine response. Although, precautions were taken to minimize the impact that mental or emotional stress may have on the epinephrine response it should be noted that subjects were asked to run for a period of approximately 50 minutes in an atypical environment, or swim continually for an atypical length of time (the majority of a normal swimming workout is interval work). Furthermore, the subjects were student-athletes who may have been somewhat anxious with the time commitment of the test sessions that were additions to their regular academic and training schedules. It is also interesting to note that the majority of the subjects had been subject to drug testing and reflected on that unpleasant experience in the course of this study. Finally, the subjects were asked to refrain from caffeine twenty four hours prior to the test sessions. Failure to comply with this request may cause an elevation in resting and exercise levels of epinephrine in trained individuals.

Interpretation of the results of this study are further complicated by the conflicting

evidence on the role gender plays in the catecholamine response. In studies conducted by Friedmann and Kindermann [166] and Favier et al. [167], the catecholamine response to exercise by trained and untrained males and females was similar. These two studies did not support the concept of a gender difference, but did provide additional evidence for a training response. However, the data on gender similarities must be interpreted with a great deal of caution in light of the evidence reported by Goldstein et al. [168]. In a well controlled study of young females (20-25 yrs), Goldstein et al. found plasma and urinary catecholamines levels were significantly lower during the follicular phase of the ovulatory cycle. Additional studies are needed to clarify the role the menstrual cycle may have in resting and exercise excretion rates of catecholamines. The catecholamine levels observed in the present study were recorded during the follicular phase of the menstrual cycle. In view of the catecholamine values reported by Goldstein et al. [168], it would appear as though the phase of the menstrual cycle may influence circulating levels of catecholamines, however, additional studies are needed to clarify the role of the menstrual cycle in catecholamine excretion.

The secretion of catecholamines is important due to the role they play in modulating cardiac function, blood-flow distribution and substrate mobilization processes during physical performance. The degree of hepatic glycogenolysis, adipose tissue lipolysis, muscle glycogenolysis and lipolysis is a function of the intensity of the beta sympathetic activation. Thus, the substrate availability during prolonged exercise, similar to the exercise bout in the present study, is most likely related in part to the circulating levels of epinephrine and norepinephrine. During exercise of extended duration, a rise in the circulating levels of epinephrine and norepinephrine promotes the mobilization of free fatty acids (FFA) from adipose tissue. Coupled with the mobilization of FFA are increases in glucose mobilization from the liver, and gluconeogenesis by the liver. The degree of lipolysis during submaximal exercise is known to be influenced by the state of

training. Specifically, trained persons will have higher rates of lipolysis in comparison to untrained persons [166]. It has been suggested that the intensity or extent of lipolysis may subsequently effect the hunger response.

The current theories on hunger would suggest that there are peripheral and central receptors that are responsive to changes in circulating levels of fuels, specifically the levels of glucose and free fatty acids [23, 30, 35, 51, 169, 170]. Centrally, it is proposed that a satiety center and a feeding center exist within the hypothalamus [171]. Specifically, an increase in blood glucose would elicit an increase in the electrical activity of the satiety center in the ventromedial nuclei of the hypothalamus, and a decrease in the activity of the feeding center of the lateral nuclei [30]. In addition, various researchers have suggested that the liver may contain glucoreceptors that provide information to the brain regarding the intrahepatic concentration of glucose [34, 51, 169, 170]. Furthermore, the studies of Friedman and Ramirez [170] suggested that hepatic nerve activity may also be related to the levels of fatty acids in the liver. Moreover, the role of fat storage and mobilization may also be important in the nature of the hunger response that is elicited. Specifically, if there is an increased mobilization (lipolysis) the supply of fuel to the tissues is increased and the hunger response is low.

In light of the elevated fuel mobilization that occurs during exercise, these theories would suggest that hunger is depressed during and following exercise (for a brief period of time) due to the increased activity in the satiety center. The information that is provided to the brain (satiety center) during exercise indicates that circulating and hepatic levels of glucose and free fatty acids are high, thus, hunger is depressed. The catecholamines, epinephrine and norepinephrine are then implicated as anorexic agents due to the impact they have on lipolysis and glycogenolysis. However, it is interesting to note that during periods of fasting or starvation levels of norepinephrine may be

elevated and a subject's perception of hunger may also be elevated. Thus, the catecholamine levels may have an impact on hunger and satiety, yet it is important to recognize that changes physiological variables such as in blood lactate or body temperature may also play a role in precipitating the hypophagic response that has been noted after exercise.

Therefore, the inverse correlation between norepinephrine and hunger ratings reported in the present study imply that the hunger response following exercise may be directly related to the degree of fuel mobilization, which in turn is related to the secretion of catecholamines. The level of catecholamine secretion is related to the intensity of the exercise and the degree of emotional or mental stress. In addition, if indeed catecholamines indirectly suppress hunger, the effect would not persist for an extended period of time following exercise due to the extremely short half-life of the catecholamines. The results of the present study indicated that the increase in hunger with time following exercise follows the same pattern of catecholamine reabsorption. Specifically, the subjects indicated they were satisfied at 5 and 20 minutes following exercise. In contrast, at 35 minutes following exercise the mean hunger rating was 6.08, and 6.42 for the runners and swimmers respectively, which indicated the subjects were "slightly hungry". Furthermore, based on the findings of the present study and those of Thompson et al. [14], exercise, irrespective of mode, appears to suppress hunger but for only a brief period of time, which may be related to the removal rate of catecholamines, which indirectly influence substrate utilization. However, of greater significance to energy balance is the role the catecholamine response may have on food intake following exercise.

Appetite (Food Intake)

The third purpose of the present study was to measure the food intake of runners and swimmers following an exercise bout and a control period. Secondary to this purpose was

to examine the relationship between urinary catecholamines, hunger and food intake following an exercise bout and a control period. Of particular interest was an attempt to determine whether urinary levels of catecholamines and/or hunger ratings were significant predictors of 24 hour food intake following submaximal exercise and a control period.

There was no significant relationship between the hunger ratings following the control period and the subjects' food intake. This finding would imply a dissociation between an individual's hunger response and his/her food intake on non exercise days. This observation is in agreement with the findings of Thompson et al. [14] and Reger et al. [40, 41] who reported no relationship between the hunger response and the total caloric consumption in the test meal following a control period. Furthermore, the results of the present study indicated there was no relationship between hunger and food intake following the experimental (exercise) session. This finding is also in agreement with the reports of Reger et al. and Thompson et al. [14, 40, 41] who found no significant correlation between a subject's food intake in a test meal and her perception of hunger following exercise.

Although, it is recognized that hunger is not an all or none phenomenon, it does not appear likely that the varied perceptions of hunger have an effect on reducing or increasing the food intake on an acute level. The lack of a relationship between the subject's perception of hunger and food intake is not surprising in view of the multitude of physiological, psychological and environmental factors that may influence an individual's food intake. Specifically, food intake may be related to adipose reserve, ambient temperature, hypoglycemia, osmolarity, palatability or a combination of any of the aforementioned variables [51]. Moreover, it is possible that food intake is also related to a variety of conditional stimuli, for example, time of day, environment in which the meal is consumed, and the presentation of the meal (ie. color and texture).

The separation of the hunger and food intake response in the present study is further supported by the findings of a several investigations [29, 107-109, 172]. In 1975 Brobeck [29] suggested that perhaps food intake is more closely related to the satiety signals than hunger signals. This appears to be reinforced with a later investigation by Brala and Hagen [172] who showed that hunger ratings did not correlate with total caloric consumption. Brala and Hagen [172] postulated that their findings indicated that short term intake of food was more closely related to "hedonistic" aspects of taste than were calories.

The observations reported by Durrant and Royston [109] would imply that on a chronic basis hunger may not be a good predictor of total caloric intake. In spite of three (1000 kcal 7 days, 500 kcal 7 days and 1000 kcal for 7 day) dietary manipulation periods the authors did not observe a correlation between hunger and intake. Perhaps, the relationship between hunger and food intake is best described by Monello [108]. Monello [108] theorized that for survival an organism must be made aware of an energy deficit, via conscious sensations, conversely, satiety, or the actual food intake may take place as a result of signals at an unconscious or subconscious level, ie. hypothalamic control. Thus, in light of the results of the present study coupled with the reports of earlier investigations it would appear as though food intake is not related to the subject's perception of hunger.

It has been reported by several investigators that chronic endurance exercise tends to decrease food intake [3, 6, 38, 52, 61, 81-83, 95]. Catecholamines have been proposed as a potential explanation for the suppression of appetite (food intake) following an acute bout of exercise and during periods of regular physical training [43, 52]. At the present time the nature of the relationship between urinary catecholamine levels food intake has yet to be accurately described or defined.

The results of the present study indicated a significant ($r = 0.45$) positive

correlation between norepinephrine and food intake the 24 hours following exercise, however, no relationship was apparent between epinephrine and experimental food intake. Furthermore, hunger did not correlate with the urinary catecholamines or food intake following the control session.

In order to determine which of the variables (hunger, mode of activity, and catecholamines) measured in the present study may serve predictors of food intake a regression model was tested. Using stepwise regression, norepinephrine and the subject's hunger rating at 20 minutes were significant predictors of food intake following exercise, and accounted for 49 percent of the variance in food intake. Conversely, following the control session food intake was best predicted by the subject's hunger rating at 60 minutes. The model accounted for 19 percent of the variation in food intake following a control session.

It is difficult to draw discuss these data in light of the lack of studies with similar protocols and subjects. However, the results of the present study tend to support the findings reported by Guillard et al. [52]. Guillard et al. [52] reported a significant positive correlation between an increase in twenty four hour levels of norepinephrine and epinephrine and a suppression in 24 hour food intake in male rats that were involved in a swimming program. The results of Guillard et al. [52] and those of the present study suggest that increasing the total catecholamine secretion via endurance exercise during a twenty four hour period may reduce appetite.

In addition, the gain in body weight normally experienced by developing rats was greater in the control rats compared to the exercising rats. Thus, it would appear as though chronic exercise will tend to raise the overall daily (total twenty four) secretion of urinary catecholamines above sedentary levels in rats and this may in turn depress gains in body weight. However, it is tempting to speculate that norepinephrine may be an anorectic agent due to its ability to stimulate lipolysis. Thus, it may be the case that

norepinephrine acts indirectly to reduce food intake by stimulating lipolysis which then elicits a hypophagic response. Three important observations support the anorectic effect of norepinephrine in the rat model: (a) norepinephrine generates a persistent elevation in free fatty acids [52, 57], (b) chronic infusions of norepinephrine in rats stopped weight gain [54], and (c) increased circulating levels of norepinephrine in response to chronic exercise retarded body weight gains and food intake in rats [52, 58].

Unfortunately, an equivalent amount of information is not available for the human model. However, two important observations have been recorded in humans: (a) a significant correlation has been recorded between the urinary output of catecholamines the day after heavy exercise and the activity of lipoprotein protein lipase in skeletal muscle [173, 174], and (b) the activity of lipoprotein lipase (LPL) is increased in skeletal muscle and adipose tissue in trained subjects compared with the LPL activity in sedentary control subjects [175]. Catecholamines, as a potential mechanism for regulation of appetite in the trained individual, will be examined in greater detail later in this paper.

The food intake values that were observed in the present study must be interpreted with caution in light of two major confounding factors which may have influenced caloric intakes reported by the subjects. First, in spite of the procedures followed in this study to improve the accuracy of the 24 hour dietary recall, it has been shown to overestimate caloric intake for individuals with low intakes and underestimate values for individuals with high caloric intakes [6, 105]. Second, in spite of the orientation provided to the subjects, Stern's data would imply the technique has an inherent error of ten percent [86].

The food intake values for the swimmers and runners in the present study were not significantly different from each other. In addition there did not appear to be a difference between caloric intake on exercise versus non-exercise days. The food intake values (1735-1988 kcal/day) observed for the runners in the present study are in close

agreement with the values that have been reported in cross sectional studies of female runners [3, 6, 81, 82]. The intake for the swimmers (1996-2267 kcal/day) is slightly below what has been previously reported for competitive female swimmers [1, 79, 176]. The food intake values observed in both groups are within the recommended daily allowance (RDA) for a sedentary female 18-22 years of age.

It appears as though the subjects in the present study, and female athletes in general, are meeting the RDA for a sedentary female, and it is also true that they are consuming a greater number of calories than their sedentary counterparts [80, 177]. However, in view of the high daily energy expenditures reported for these athletes it is difficult to understand why energy intake values are not higher. A number of cross sectional [3, 6, 81-83, 176, 177] and longitudinal studies have reported lower than expected caloric intakes for active females [38, 61, 71, 95, 178]. The mechanism responsible for the suppression of appetite in response to chronic endurance training remains to be clearly determined.

Catecholamines have been proposed as one potential variable to explain the appetite response in endurance trained individuals. The anorectic effect that is attributed to catecholamines is related to their ability to mobilize lipids and carbohydrates. The increased mobilization of fuel during exercise is directly related to the secretion of epinephrine and norepinephrine. In general, trained individuals will demonstrate lower levels of norepinephrine at rest and during submaximal work, and elevated levels of epinephrine during high intensity work [160, 161, 163]. The effect of training on resting epinephrine levels is controversial. Kjaer et al. [163] reported that in several investigations conducted in their laboratory an increased epinephrine response at rest was reported for trained individuals, while other studies have reported decreases or no change in resting epinephrine levels [155, 160, 162].

Regardless of the changes training may or may not elicit in resting epinephrine

levels there are other changes noted with training that may enhance the resting lipolysis of trained individuals. Training has been reported to cause an increase in the enzyme lipoprotein lipase which is central to the lipolysis of fats [174, 175]. The catecholamine and enzyme changes noted for trained individuals at rest and during exercise may elicit a hypophagia response which may precipitate the mismatch of energy expenditure and intake in trained subjects. Specifically, in a report by Crampes et al. [179], endurance trained subjects demonstrated an increased sensitivity of the subcutaneous abdominal adipose tissue to the lipolytic effect of epinephrine. Prior to accepting catecholamines as the variable that explains the appetite and expenditure mismatch additional cross sectional and longitudinal studies are needed that employ female subjects. In addition, it would be premature to accept the catecholamines as the mechanism responsible for appetite suppression in view of the additional variables (ambient and muscle temperature, blood and muscle lactate) that may effect substrate mobilization and utilization.

Finally, appetite is a multifaceted phenomenon that is a result of a variety of physiological, psychological and environmental stimuli, and at best can only be partially explained by the interaction of catecholamines and metabolism. In the present study the variable appetite and catecholamine response do not serve to confirm or deny a relationship between appetite and catecholamines. The findings of the present study suggest a need for additional studies that carefully examine the interaction of the hormones and enzymes that help to regulate appetite and substrate utilization.

Resting Metabolic Rate

The fourth purpose of this study was to measure and compare the resting metabolic rate (RMR) of trained and untrained females and trained runners and swimmers. Resting metabolic rate accounts for the largest fraction of the daily energy expenditure.

Therefore, alterations in RMR, as a result of endurance training, may become significant

in weight loss programs that combine diet and exercise to induce a negative caloric balance.

The results of the present study indicated that when RMR was expressed relative to total body weight ($\text{kcal}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$) there were no differences between trained and untrained subjects or between runners and swimmers. A lack of differences between trained and untrained subjects is similar to the data of previous studies [10, 116, 124]. It is difficult to discuss the lack of differences between runners and swimmers due to the scarcity of RMR data for trained female swimmers and runners. The significance of RMR to the energy balance equation is discussed later in this paper.

Resting metabolic rate expressed as kcal/day was significantly different among the three groups in the present study ($F=3.71$). However, the orthogonal contrasts indicated that there were no differences between the trained and untrained subjects. The contrast of runners and swimmer indicated significant ($F= 6.02$) differences in resting metabolism. Thus, when RMR was expressed in absolute values, differences did not appear to exist between trained and untrained females. This finding is in agreement with previous reports [10, 116, 124]. However, the runners, perhaps due to their lower total body weight, did have significantly lower RMR (kcal/day) values in comparison to the swimmers. Unfortunately, there is a paucity of research available on resting metabolism and trained females. Moreover, a recent energy balance study of female swimmers failed to report the values observed for RMR [4]. Clearly, additional cross sectional and longitudinal studies are needed of moderately and highly active females.

Based on the results of several studies [12, 111, 113, 115, 117, 119] fat free mass, or lean body weight, appears to be the best predictor of resting metabolic rate in males and females. The results of the present study indicated that there were significant ($F= 17.06$) differences in RMR expressed as kcal/kg LBW between trained and untrained females, and no differences between runners and swimmers.

The values reported for RMR (kcal/day) for all three groups in the present study are above results reported by some studies [116, 177] , but in close agreement with the values that have been previously reported for control or sedentary females of ideal or normal weight [111, 117] . Previous investigations have utilized a 12 hour fast, and some have also measured the energy expenditure in a metabolic ward setting. The protocols followed in earlier investigations [111, 116 , 117, 177] resembled the procedures that are normally given for the determination of basal metabolic rate (BMR). Measurements of basal metabolic rate are normally 90% of resting metabolic rate. Although, the studies by Myerson et al. [177] and Owen et al. [116] reported the energy expenditure values as RMR, it may be more appropriate to classify the measurement as BMR. The discrepancy in values is most likely explained by the difference in experimental protocols. Resting metabolism was measured in the present study after a 3 hour fast and the subjects had refrained from physical training at least 20 hours prior to the measurement. In spite of the abstinence from training the subjects were allowed to walk to the lab, and to and from classes prior to the measurement. When the values of the current study are corrected for the 10-12 percent difference that is known to exist between RMR and BMR the values are in agreement for control subjects or previously reported studies [111, 116 , 117, 177]. It is also important to recognize that extraneous movements by the subject while the measurement is being taken will understandably increase the RMR measured. Furthermore, Ravussin et al. [115] postulated that a large portion (100-800 kcal/day) of the variability in energy expenditure among subjects may be due to differences in the amount of spontaneous physical activity, also known as "fidgeting". Subjects in the present study asked to refrain from extraneous movements during the assessment of RMR.

The variability in energy expenditure between individuals can be minimized by expressing RMR relative to fat free mass or lean body weight [115, 122]. Myerson et

al. [177] measured the RMR of amenorrheic and eumenorrheic runners and sedentary controls. The amenorrheic runners had a lower resting metabolic rate than their eumenorrheic or sedentary cohorts, and the eumenorrheic runners had a lower RMR than the controls. The pattern of differences between trained and the controls in the present study are in agreement with the data reported from the investigation by Myerson et al. [177]. The majority (19/24) of the trained subjects in the present study were classified as eumenorrheic. The results of the present study indicated that there were significant ($F=10.13$) differences among the three groups in RMR expressed as kcal/ kg LBW. The trained subjects had a significantly lower RMR (kcal/kg LBW) when compared to the control subjects. Runners and swimmers had similar values for RMR relative to lean body weight.

Role of RMR and Appetite in Energy Balance

The results of the present study, and those of Myerson et al. [177], suggest that the trained individuals are manifesting some form of energy conservation. A theoretical energy balance equation for the exercise day is constructed for the runners and swimmers of the present study:

Net theoretical energy balance = Appetite - (RMR + TEF* + TEA**)

Runners: 1988 kcal - (1471 kcal + 147 kcal + 732 kcal) = -362 kcal

Swimmers 2267 kcal - (1661 kcal + 166 kcal + 749 kcal) = -309 kcal

*The equation reflects estimated values of TEF (10% of RMR)

** 600 kcal expended in the exercise session and an additional 9% of the RMR to account for normal daily activity.

Discrepancies between energy expenditure and intake by the athletes in the present study are not surprising in view of the numerous studies that have previously reported a similar disparity in moderately and highly trained females [1, 3, 6, 79, 81, 82, 176, 177]. One would normally expect that maintaining an energy deficit as theoretically

calculated in the equations above would lead to significant weight losses over time. However, weight did not change during the course of the present study. To accurately assess the impact of an energy deficit similar to the values observed in the present study longitudinal measurements of energy intake and expenditure should be taken.

Understandably, it is difficult to comprehend why female athletes with extremely high daily energy expenditures would demonstrate a reduced RMR as was reported by Myerson et al. [177]. One potential explanation for this type of response may be related to the degree of the energy imbalance and the length of time the imbalance has been maintained. Myerson et al. [177] approximated the caloric deficit of runners to be 600-800 calories per day. It is difficult to determine precisely what energy deficit was maintained by the athletes in the present study without the thermic effect of feeding values and energy expenditure for all daily activities. However, from a theoretical viewpoint one is tempted to conclude that the trained athletes in the present study were manifesting some form of energy conservation. The RMR values measured in the present study for the control subjects would also suggest that perhaps a more critical examination of the RMR for sedentary populations is necessary. Furthermore, the results of the present study suggest that current RDA values for energy intake may also need to be examined.

Clearly, further studies are needed that examine the relationship between energy intake and the components of the energy expenditure portion of the energy balance equation. In view of the competitive pressures on female athletes to be "thin", additional information must be gathered if attempts are going to be made to truly understand the role that diet and training have on performance.

Summary

As the number of girls and women participating in sport has increased, the pressure to perform has also increased. Coupled with the added competitive pressures to be the

best, are alarmingly high numbers of athletes with poor nutritional habits, and the incidences of eating disorders continues to rise among female athletes at a alarmingly high rate. Furthermore, even small amounts of excessive non-essential fat can have a considerable influence on physical performance. Performance is most certainly linked with the athlete's ability to maintain energy balance.

Energy balance

The physical training and nutritional practices of female athletes will influence the energy balance equation which will indirectly impact performance. Energy balance is the interaction of energy intake (appetite), and expenditure (resting metabolic rate, the thermic effect of eating and the thermic effect of activity). The variety of physiological and psychological factors that may influence appetite make it a difficult phenomenon to study and understand. Despite the difficulties associated with the measurement of appetite (food intake) it is useful to understand how the caloric intake may be manipulated to create an energy deficit, or surplus, depending on the desired outcome set by an individual. Furthermore, it is important to understand how exercise, specifically the mode and intensity, may influence an individual's appetite. The results of the present study indicated that female runners and swimmers have similar hunger, catecholamine and appetite responses to work that is equivalent in intensity and expenditure.

The majority of studies that have been undertaken to understand the relationship between eating behavior, body composition and exercise have been designed to assess the impact of chronic training on these variables. The acute response to exercise may have a greater role in the long term changes that are manifested in energy balance by exercise training. Specifically, the data of this study suggested that urinary catecholamines may play a role in the appetite of the female athlete.

It has been suggested that female endurance athletes chronically maintain an energy expenditure greater than their energy intake, yet significant weight losses are not

experienced by these women. Superficially, energy balance is a phenomenon that appears to be easily achieved in female distance runners as evidenced by their low levels of body fat. It has been suggested that the low levels of body fat reported in female runners are a result of an elevated energy expenditure that is a consequence of intensive training (mileage and time). It is not likely that the differences in body fat between the runners and swimmers in this study are due to differences in training status. The runners and swimmers in this study were classified as highly trained as a result of the mean VO_2 max values achieved.

Numerous studies have been conducted using trained female runners in an attempt to understand the influence chronic physical training has on body composition and food intake. Specifically, researchers have been interested in determining how a chronic elevation of energy expenditure through exercise influences body composition, and energy intake. However, few studies have examined the role hunger may have on the appetite. Second, very few studies have examined how the mode and/or intensity of training may influence the hunger response.

Exercise is often prescribed as an important component of a conscientious weight loss program for healthy and obese populations. In addition, as athletes and coaches train to enhance performance, more precision will be needed in the design of the training program. The present study has illustrated the complexity of the factors that influence the energy balance equation in females. Future studies are needed to clarify the role each factor plays and the degree of manipulation that is possible through exercise.

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APPENDIXES

APPENDIX A

The University of Tennessee*
Knoxville, Tennessee
Consent for Investigation Procedures
(Informed Consent)

I, _____, hereby agree on _____ (date) to participate in the research project entitled: Hunger, Appetite and Catecholamine Responses in Trained Females. The proposed study will take approximately 6 weeks to complete.

Note: In the event you find that any of the following information is not clear, please ask the investigator to explain immediately.

Regular exercise is known to help increase energy expenditure and, therefore, might be helpful in weight loss or weight control. The purpose of this study will be to determine body composition, cardiovascular endurance, and resting metabolic rate.

In this study I understand that the following measurements or tests will be conducted on separate occasions: a). recording body weight and height, and an underwater weighing, b). measurement of lung volumes, c). estimation of maximal aerobic capacity (VO_2 max), d). two exercise test sessions, and two control sessions, e). recall of 24 hour food intake, and f). resting metabolic rate. I understand that I will fast three hours prior to measurement of all of the physiological variables and the exercise sessions.

Body weight will be measured using a standard laboratory weighing scale; body height will be measured with a meter stick. Underwater weighing will be done in the swimming pool, and lung volumes will be measured in the Pulmonary Function Laboratory at Rockingham Memorial Hospital. Maximal aerobic capacity (VO_2 max), resting metabolic rate, the control and exercise sessions will take place in the Human Performance Laboratory on the treadmill or in the swimming pool. Furthermore, I understand I will be breathing through a mouthpiece and be wearing a noseclip or face mask, as well as being connected to an electrocardiogram to monitor my heart rate during the exercise tests. Prior to and after the exercise and control test sessions I agree to submit urine samples. I understand that the urine samples will not be analyzed for the presence of any illegal or banned substances. All urine samples will be analyzed exclusively for the presence of adrenalin and noradrenalin (epinephrine and norepinephrine). I also agree to participate in a dietary recall 24 hours following each exercise and control session.

I understand the purpose of the study and that the benefits to me are primarily informational. However, the data collected may add to the body of knowledge regarding the benefits of exercise in weight maintenance.

I have been informed of the risk associated with the measurement of underwater weighing and VO_2 max. I may experience some slight discomfort during the measurement of my maximal aerobic capacity (VO_2 max) and body composition (underwater weighing). I also understand that I can withdraw from the study at any time without penalty.

Subject's Signature _____

Date

Witness Signature _____

Date

Judith A. Flohr: Department of Physical Education: Phone # 703-568-3448

*Used for runners and swimmers

APPENDIX B

The University of Tennessee*
Knoxville, Tennessee
Consent for Investigation Procedures
(Informed Consent)

I, _____, hereby agree on _____ (date) to participate in the research project entitled: Hunger, Appetite and Catecholamine Responses in Trained Females. The proposed study will take approximately 3 weeks to complete.

Note: In the event you find that any of the following information is not clear, please ask the investigator to explain immediately.

Regular exercise is known to help increase energy expenditure and, therefore, might be helpful in weight loss or weight control. The purpose of this study will be to measure resting metabolic rate in trained and untrained females.

In this study I understand that the following measurements or tests will be conducted on separate occasions: a). recording body weight and height, an underwater weighing, and b). resting metabolic rate. I understand that I will fast three hours prior to measurement of all of the physiological variables. Body weight will be measured using a standard laboratory weighing scale; body height will be measured with a meter stick. Underwater weighing will be done in the swimming pool, lung volumes and resting metabolic rate, will be measured in the Human Performance Laboratory (Godwin 209) Furthermore, I understand I will be breathing through a face mask,

I understand the purpose of the study and that the benefits to me are primarily informational. However, the data collected may add to the body of knowledge regarding the benefits of exercise in weight maintenance.

I have been informed of the risk associated with the measurement of underwater weighing and resting metabolic rate. I may experience some slight discomfort during the measurement of my body composition (underwater weighing). I also understand that I can withdraw from the study at any time without penalty.

Subject's Signature _____

Date

Witness Signature _____

Date

Judith A. Flohr: Department of Physical Education: Phone # 703-568-3448

*Used for controls

APPENDIX C

The University of Tennessee
Screening for Health Related Fitness Testing and Participation

Health Status Questionnaire: Please check yes or no by each of the following.

	Yes	No	
1.	_____	_____	Has your doctor ever said you have heart trouble cardiovascular disease?
2.	_____	_____	Have you experienced pains of pressure in the left midchest area, left neck, shoulder or arm at rest or in response to exertion?
3.	_____	_____	Do you often feel faint or have spells of severe dizziness?
4.	_____	_____	Have you ever been told you have high blood pressure?
5.	_____	_____	Do you have a bone or joint problem that has been aggravated by exercise, might be made worse by exercise, or that would interfere with exercise?
6.	_____	_____	Do you have symptoms of back pain that might limit you in flexibility or aerobic conditioning exercise?
7.	_____	_____	Are you currently taking medication? If yes the name of the medication and purpose. _____
8.	_____	_____	Could this medication or any over the counter drug you are taking alter your response to exercise? If so please explain. _____
9.	_____	_____	Are you currently taking oral contraceptives?
10.	_____	_____	Have you ever had (or presently have) kidney disease?
11.	_____	_____	Are you now receiving treatment for hypocalcemia ? Have you ever been prescribed a calcium supplement? yes_____ no_____

- | | Yes | No | |
|--|-------------|--------------|--|
| 12. | _____ | _____ | Have you ever been treated for any hormone abnormality? |
| 13. | _____ | _____ | Is your menstrual cycle regular? |
| 14. | _____ | _____ | Have you ever been told by your physician not to exercise or limit your level of physical exertion? If yes, what limitations were Recommended? |
| <hr/> | | | |
| 15. | _____ | _____ | Do you have three or more of the following secondary risk factors? |
| | | | a. family history of coronary heart disease |
| | | | b. obesity |
| | | | c. sedentary lifestyle |
| | | | d. type A behavior, with a stressful occupation and/or lifestyle |
| | | | e. diabetes |
| 16. How would you rate the amount of physical activity you perform during the day? | | | |
| _____ | very little | _____ little | _____ moderate |
| _____ | | | active |
| active | | | _____ very |
| 17. How would you rate the amount of physical activity you perform during leisure time? | | | |
| _____ | very little | _____ little | _____ moderate |
| _____ | | | active |
| active | | | _____ very |
| 18. Are you presently performing any standard or regular exercise program? If so, please explain the type, frequency and duration of activity. | | | |
| _____ | yes | _____ | no |

APPENDIX D

Instructions for Test Sessions•

It is important that you follow the special instructions listed below for each test session. If for some reason you were not able to comply with the test preparation instructions please notify the investigator upon arrival to the laboratory or pool (prior to the beginning of the test session).

Underwater Weighing (Body Composition)

All underwater weighing will take place in the Godwin pool. Please arrive in a swim suit. Prior to entering the pool you will be asked to weigh yourself on the scale in the Women's locker room across from the pool. It is important that you perform the underwater weighing with an empty bladder, so please void just prior to the test. This session will take 15 - 20 minutes to complete.

My appointment for underwater weighing is

Residual Lung Volume

All lung volume measurements will be completed at Rockingham Memorial Hospital in the Pulmonary Function Laboratory. The lab is on the first floor of the hospital. The easiest way to find the lab is to take the elevators that are off of the main lobby and simply follow the signs. This session will take 15 - 20 minutes to complete.

My appointment for the residual lung volume is

Resting Metabolic Rate (RMR)

A minimum of 22 and a maximum of 26 hours should have elapsed from the time of last exercise session you engaged in until the resting metabolic rate (RMR) measurement is taken. For example if you exercised from 3:00 pm to 4:00 pm on a Wednesday you would schedule your RMR appointment for anytime between 2:00 and 6:00 pm on Thursday. Prior to the RMR measurement your activity should be as low as possible. **DO NOT RUN TO THE LAB**, should you be late do not try to make up lost time by running to make your appointment. **WALK or DRIVE** to the lab, exerting as little energy as possible. The 24 hours prior to the measurement of RMR should be as relaxed as possible.

Please arrive at the lab in a fasted state haven eaten approximately 3 hours prior to the measurement of RMR. It is extremely important that you maintain your normal daily diet throughout the entire experiment. However, if you normally consume caffeine, please abstain from caffeine at least 24 hours prior to the measurement of RMR. Wear comfortable clothes, you will be resting and not exercising for this measurement. This measurement will be taken only once, unless an unforeseen complication arises during the test that would influence the test results. This measurement should be taken sometime in the first 14 days of your menstrual cycle. If you are confused about any procedure, please ask questions. This measurement is taken in room 209 Godwin. This session will take 45 - 50 minutes to complete.

My appointment for Resting Metabolic Rate is:

VO₂ max (Maximal Oxygen Consumption)

It is not essential that you are fasted for this measurement, however, it is suggested that you follow your normal pre practice meal size and time. Dress to run or swim which ever is appropriate. Please complete your normal stretching routine prior to your appointment time. A moderate warm-up is incorporated into the test session. All runners will perform this test in room 209 Godwin, all swimmers in the Godwin pool. This session will take 30- 40 minutes to complete.

My appointment for VO₂ max determination is

Experimental and Control Sessions

Please arrive at the lab (Godwin 209) or pool having fasted for 3 hours. In addition, your last exercise session should have been completed 20-24 hours from your test time. In addition to the 3 hour fast it is essential that you abstain from caffeine 24 hours prior to the experimental session. Please drink copious amounts of water the 12 hours prior to the experimental or control session. It will be extremely desirable if you can avoid voiding 1-2 hours prior to your arrival at the lab. You will have approximately 45 minutes after completion of the exercise session to study or read so please feel free to bring your books. During the control session you may study or read so please bring your books and/or reading materials. You should complete one exercise and one control session sometime in the first 14 days of your menstrual cycle. All control sessions will take place in Godwin 209. The experimental and control sessions will take approximately 2 hours each to complete.

My appointment for the experimental session is :

My appointment for the control session is:

Please call me at 3448 or 434-8677 if you have questions or concerns. Thanks for all your help.

*Used for runners and swimmers

APPENDIX E

Instructions for Test Sessions•

It is important that you follow the special instructions listed below for each test session. If for some reason you were not able to comply with the test preparation instructions please notify the investigator upon arrival to the laboratory or pool (prior to the beginning of the test session).

Underwater Weighing (Body Composition)

All underwater weighing will take place in the Godwin pool. Please arrive in a swim suit. Prior to entering the pool you will be asked to weigh yourself on the scale in the Women's locker room across from the pool. It is important that you perform the underwater weighing with an empty bladder, so please void just prior to the test. This session will take 15 - 20 minutes to complete.

My appointment for underwater weighing is.

Residual Lung Volume

All lung volume measurements will be completed at Rockingham Memorial Hospital in the Pulmonary Function Laboratory. The lab is on the first floor of the hospital. The easiest way to find the lab is to take the elevators that are off of the main lobby and simply follow the signs. This session will take 15 - 20 minutes to complete.

My appointment for the residual lung volume is

Resting Metabolic Rate (RMR)

A minimum of 22 and a maximum of 26 hours should have elapsed from the time of last exercise session you engaged in until the resting metabolic rate (RMR) measurement is taken. For example if you exercised from 3:00 pm to 4:00 pm on a Wednesday you would schedule your RMR appointment for anytime between 2:00 and 6:00 pm on Thursday. Prior to the RMR measurement your activity should be as low as possible. **DO NOT RUN TO THE LAB**, should you be late do not try to make up lost time by running to make your appointment. **WALK or DRIVE** to the lab, exerting as little energy as possible. The 24 hours prior to the measurement of RMR should be as relaxed as possible.

Please arrive at the lab in a fasted state haven eaten approximately 3 hours prior to the measurement of RMR. It is extremely important that you maintain your normal daily diet throughout the entire experiment. However, if you normally consume caffeine, please abstain from caffeine at least 24 hours prior to the measurement of RMR. Wear comfortable clothes, you will be resting and not exercising for this measurement. This measurement will be taken only once, unless an unforeseen complication arises during the test that would influence the test results. This measurement should be taken sometime in the first 14 days of your menstrual cycle. If you are confused about any procedure, please ask questions. This measurement is taken in room 209 Godwin. This session will take 45 - 50 minutes to complete.

My appointment for Resting Metabolic Rate is:

*Used for controls

APPENDIX F

Urine Catecholamine Procedure

Preparation of columns

1. Put the small plastic columns on the wooden rack and add enough of the resin to fill the small diameter barrel and a little more. **Be consistent across columns.**
2. Add 0.1% EDTA pH 6.5 to each column sufficient to fill it.
3. Let the columns flow and use your judgement that they are flowing at about the same rate.

Preparation of urine sample

1. Measure out about 3 minutes worth of urine (1/20 of a 1 -hour collection) and put into each of four different 100 ml beakers. (**Record volume of urine used**).
2. Add 0.1 ml of 5% EDTA to each beaker for each 5 ml of urine.
3. Add 0.1% EDTA to each beaker to bring the volume to about 20 ml.
4. Adjust pH of each beaker to 6.5 with 1N NaOH by adding and swirling around.
Don't use magnetic stirrer.
5. Add 1 ml of 0.2 M phosphate buffer, pH 6.5
6. Put beaker in front of column stand.
7. Add internal standard (1000 μ l of 0.5 g/ml NE) to the third and fourth beaker in each pair.
8. **Stir or swirl contents** and pour on column.

Elution of catecholamines

1. After urine has run through the column, add about 10 ml of 0.1% EDTA.
2. After this has run through, put conical centrifuge tube under column and add 9 ml 4% Boric Acid.
3. Remove conical tube and add 4% boric acid to each to make all tubes read 10.0 ml
4. **COVER TUBE WITH PARAFILM AND INVERT TUBE SEVERAL TIMES TO MIX CONTENTS.**

Measurement of total catecholamines

1. Make fresh NaOH/Asb/EDA solution; mix thoroughly by gently inverting the glass stoppered graduated cylinder; keep in ice water.
2. For each boric acid tube put 2 fluorometer tubes out. Add 1 ml (1000 μ l) of the boric acid sample to each fluorometer tube.
3. Add 1 ml of pH = 6.5 phosphate buffer to each tube.
4. Add 100 μ l 0.01 1N HCl to each tube except the external standards.
5. Add 100 μ l of NE (0.5 μ g/ml) to the NE external standard tube. **Note: the external tube consists of the boric acid from the urine sample plus the 100 μ l amount of NE. In this way, the external standard is being run under conditions of the sample.**
6. Add 100 μ l ZnSO₄ to each sample tube.
7. With the clock running, add 100 μ l potassium ferrocyanide to the first sample tube and **VORTEX**; do remaining tubes at 10 second intervals.
8. At exactly 3 minutes from the start of oxidation, add 1 ml of the NaOH/Asb/EDA solution from the 1 ml repipet and **VORTEX**. Stay on time sequence.

9. Add 1 ml of the NaOH/Asb?EDA to each of the "blank" tubes and **VORTEX**. Then add 100 μ l of the ZnSO₄ and 100 μ l potassium ferrocyanide solutions and **VORTEX AGAIN**.
10. Read florescence with filter set 360/515 for each sample and blank.
11. Obtain net florescence by subtracting blank from sample.
12. Obtain the external standard by subtracting the net florescence from the urine sample tube from that to which you added the 100 μ l of NE.
13. Calculate recovery by subtracting from the mean of the net florescence of the third and fourth column (the two to which you added the NE to the urine sample) the florescence of the one and two columns, then divide by the net florescence value for the external NE standard.
14. Calculate total catecholamines: [net fl of urine sample + net fl of NE standard] $\times$ 10 ml $\times$ 0.5 μ g/ml. Divide this by the recovery value to have the quantity expressed at 100% recovery.

Measurement of Epinephrine

1. For each boric acid sample tube put 4 fluorometer tubes out. Add 1 ml (1000 μ l) of the sample to each fluorometer tube.
2. Add 1 ml of pH = 2.85 0.5 M citrate/phosphate buffer to each tube.
3. Add 100 μ l 0.01 N HCl to each tube except the external standards.
4. Add 100 μ l of EPI (0.5 μ g/ml) to the EPI external standard tube.
NOTE: the external standard tube consists of the boric acid from the urine sample plus the 100 μ l amount of E. In this way, the external standard is being run under conditions of the sample.
5. Add 100 μ l ZnSO₄ to each sample tube.
6. With the clock running, add 100 μ l potassium ferrocyanide to the first sample tube and **VORTEX**; do remaining tubes at 10 second intervals.
7. At exactly 5 minutes from the start of oxidation, add 1 ml of the NaOH/Asb/EDA solution from the 50 ml Eppendorf pipet and **VORTEX**. Stay on time sequence.
8. Add 1 ml of the NaOH/Asb?EDA to each of the "blank" tubes and **VORTEX**. Then add 100 μ l of the ZnSO₄ and 100 μ l potassium ferrocyanide solutions and **VORTEX AGAIN**.
9. Read florescence with filter set 360/515 for each sample and blank.
10. Obtain net florescence by subtracting blank from sample.
11. Calculate EPI: [net fl + net fl of E external standard] $\times$ 10 ml $\times$ 0.5 μ g/ml. Divide by the recovery value to express the epinephrine at 100% recovery.

Calculation of catecholamines in urine

1. The calculation above gives you the quantity of total CA or E in the 10 ml conical tube. This came from the 3 minutes of urine that was added to the beaker. To calculate the catecholamines in the urine do the following:

$$\mu\text{g CA/tube} \times \text{total urine vol/vol/analyzed} \div \text{minutes over which urine sample was collected.}$$
2. Do for both total catecholamines and epinephrine. Subtract E from total to get NE.

APPENDIX G**Hunger-Fullness Perception Scale**

<u>Rating</u>	<u>Description</u>
1	very, very full
2	very full
3	moderately full
4	slightly full
5	satisfied
6	slightly hungry
7	moderately hungry
8	very hungry
9	very, very hungry

Explanatory Paragraph

The body's need for nourishment is signaled by a variety of cues. For example, people often experience the feeling of emptiness or hear stomach noises between meals. Likewise, during and following food consumption, the body signals various stages of fullness. These signals add up to the physical drive to eat. Of course a person's eating behavior results from many physiological, environmental, and emotional cues. This scale is designed to help you focus on physical sensations that create feelings of hunger and fullness.

You will be asked, "How hungry or full do you feel"? Focus on signals inside your body. A rating of 1 is equivalent to the fullest you have ever felt, eg. after Thanksgiving dinner. A rating of 5 is equivalent to just being satisfied, i.e. not feeling one way or the other, not thinking about eating. A rating of 9 is equivalent to the most hungry you think you could ever feel. [Maxwell, 1985]

VITA

Judith A. Flohr was born in Bainbridge, Maryland on July 25, 1952. She attended The University of California, Davis and in May 1975 received the degree of Bachelor of Science in Physiology. She received the Master of Arts degree at The University of California, Davis in September 1979 in Physical Education. In June of 1988 she entered The University of Tennessee, Knoxville and in May 1991 received a Doctor of Philosophy in Education.

She has taught at Oberlin College in Ohio, and the College of Wooster in Ohio. She is currently on the faculty in the Department of Physical Education and Sport at James Madison University, Harrisonburg, Virginia.