Effects of Aerobic Exercise and Weight Reduction on Carbohydrate Metabolism during Submaximal Exercise in Sedentary, Overweight Women.

by

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(ABSTRACT)

Hormonal and metabolic responses to submaximal exercise were studied in 11 sedentary, overweight women who participated in an 8 week aerobic exercise program (80% VO2 max) while consuming a hypocaloric diet. A maximal and submaximal treadmill exercise test were performed before and after the program. During the submaximal exercise test, a graded portion (mean time 6.4 min) preceded the submaximal phase during which subjects worked at 80% VO2 max until exhaustion (mean time 12 min). Blood was sampled before and after the work via venipuncture. Whole blood was immediately analyzed for lactate accumulation. The remaining blood was centrifuged, separated, and frozen for subsequent serum glucose, cortisol, and insulin measurement. There was a significant increase in oxygen uptake (ml/kg/min), and a decrease in body weight, (6.7%), and body fat (14%). Resting heart rate was significantly

lower post-training (5.4%), as were exercise RQ (VCO_2/VO_2) ratios. Pretraining serum glucose and blood lactate significantly increased while nonsignificant decreases were noted in insulin and cortisol as a result of the submaximal exercise bout. The significant increases in glucose and lactate during exercise were blunted after the training program. However, only the post-training response of lactate was significantly different from the pretraining response. The insulin and cortisol response was not significantly different from that during the pretraining exercise test. A significant correlation was observed between RPE and lactate at the end of exercise both pretraining and post-training. In summary, the combined exercise and weight loss program resulted in exercise being less stressful, both metabolically and subjectively. This improvement enables greater exercise intensity to be performed prior to the significant accumulation of lactate and perception of fatigue which may inspire the sedentary, overweight female to establish and/or continue a regular exercise program.

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TABLE OF CONTENTS

Table of Contents (continued)

LIST OF TABLES

Chapter I

INTRODUCTION

The tendency for all living organisms to maintain a stable internal environment is called homeostasis. Physical exercise is a stress to the body because it disrupts homeostasis. Nearly all the physiological and metabolic changes brought on by repeated bouts of exercise tend to reduce the stressfulness of exercise to minimize changes in homeostasis.

The physiological changes in body function that occur in response to exercise are dependent upon the individuals' age, sex, activity level, percent fat, and disease status. Such changes are also dependent upon the type of exercise selected, and its' intensity, frequency, and duration.

In response to homeostatic perturbations induced by exercise, the cells of the body stimulate a complex response pathway. This pathway may involve changes in nerve activity, hormones, and/or changes within a given organ. Through such a pathway, signals are transmitted to organs which will eventually change their functions to produce the observed response to exercise. The exercise response opposes the homeostatic perturbations caused by the exercise.

During physical exercise training, an adaptation

pathway eventually causes a persistant change in the manner in which the body derives ATP for exercise, and adapts to homeostatic perturbations.

Statement of the Problem

Acute physical exercise causes a variety of alterations in body fuel homeostasis and hormonal regulation. Such alterations can be further modified by training as marked differences exist between physically trained and untrained individuals in their pattern of carbohydrate and fat metabolism during exercise.

LeBlanc et al. (1979) has proposed that the important differences observed in endurance trained athletes with regard to insulin responses to exercise are related to reduced adiposity and only indirectly related to enhanced maximal oxygen uptake capacity.

The object of the present study was to assess the effect of aerobic training on the patterns of fuel mobilization during an acute submaximal exercise test conducted at 80% maximal oxygen consumption before and after an 8 week aerobic walk/jog program. This program was administered to sedentary, overweight women (18-35) who followed a reduced calorie diet designed to result in weight loss of approximately 1 kg/week.

Emphasis was be placed on glucose homeostasis and the

hormonal and metabolic factors that contribute to its regulation. Percentage of adiposity of all subjects was obtained to assess its role in the control of fuel utilization and mobilization.

Research Hypotheses

HO: There is no difference between serum insulin, cortisol, glucose and blood lactate measured via venipuncture at rest and immediate post exercise, pretraining.

HO: There is no difference between serum insulin, cortisol, glucose, and blood lactate measured via venipuncture at rest and immediate post exercise, post-training.

HO: The treatment of an 8-week aerobic walk/jog program conducted at 80% of subjects' maximal oxygen consumption has no effect on subjects' serum insulin, cortisol, glucose and blood lactate measured at rest and immediately post exercise.

HO: The treatment of an 8-week aerobic walk/jog program conducted at 80% of subject's maximal oxygen consumption has no effect on subjects' body weight, body fat, and maximal oxygen consumption.

HO: There is no relationship between endurance exercise terminal RPE and immediate post exercise blood

lactate pretraining and post-training.

HO: The aerobic training treatment has no effect on the relationship between serum insulin and glucose.

HO: There is no relationship between the change in percent fat, or the change in VO2 max (L/min) with the change in insulin after training.

Significance of the Study

Presently there is a lack of information in the literature on training induced hormonal and metabolic alterations in fuel utilization and mobilization in originally sedentary, overweight women. Additionally, the effect of the variables adiposity and aerobic capacity on the hormonal response to exercise need to be addressed. These variables, modified by exercise training and weight loss, can affect the control of carbohydrate metabolism by the hormonal system. Thus this study will contribute information regarding the alterations in body fuel homeostasis and hormonal regulation in response to aerobic exercise with concomitant caloric restriction in a virtually unstudied population.

Delimitations

The following delimitations were imposed by the investigator:

1. Subjects were 11 sedentary, overweight (>22% body fat), female volunteers (18 to 35), presently attending VPI & SU.

2. Subject selection was dependent upon screening criteria.

3. Rate of Perceived Exertion was measured using Borgs' category-ratio scale.

4. The training program was conducted 3 mornings per week for 8 weeks.

5. The training consisted of aerobic exercise; walking or jogging at 80% of subjects' maximal oxygen capacity.

Limitations

The following limitations affect the generalizibility of the findings:

1. Due to a small sample size (n=11) results are limited to the experimental sample.

2. Adherence to dietary and exercise compliance was strictly voluntary so variation may have occurred.

3. Application of results is limited to an exercise intensity of 80% VO2 max.

4. Interpretation of changes in blood and serum levels of hormones and metabolic factors is limited because such levels are the net result of synthesis, mobilization and catabolism and do not reflect the turnover rate of hormones or metabolic factors.

Definitions and Symbols

Terms and symbols requiring clarifiation for use in this study are as follows:

Diet Program. is a 1200-1400 calorie diet based on the diabetic exchange list diet. The diet is designed to result in weight loss of approximately 1 kg/week.

Exercise Prescription. is a method of devising an exercise program for individuals determined by evaluation of physiological variables resultant from the GXT.

Graded Exercise Test (GXT). is a treadmill exercise test administered to subjects. The test is designed such that speed and grade increments are elevated until functional capacity is attained (Astrand & Rodahl, 1977).

Immediate Post Exercise (IPE). is any measurement derived from blood taken immediately after subjects submaximal exercise test.

Lactic Acid (LA). is the end product of anaerobic glycolysis.

Nutritional Program. is a weekly program designed to

give subjects information regarding nutritional principles, caloric content of foods, and behavior modification techniques.

Maximal Oxygen Uptake (VO2 max). is the maximum volume of oxygen consumed and utilized by the tissues.

Rate of Perceived Exertion. is the subjects' ability to rate the intensity of the exercise by utilizing perceptual cues as the primary source of information (Noble et al. 1983).

Respiratory Quotient (R). is the ratio between the carbon dioxide produced and oxygen consumed during metabolism of foodstuffs (Lamb, 1984).

Submaximal Exercise Test. is an exercise test that consists of approximately 5 graded increments and then continues at a speed and grade corresponding to 80% of subjects' VO2 max for as long as tolerated, but not longer than 20 minutes.

Terminal Rate of Perceived Exertion (terminal RPE). is the RPE reported prior to the last minute the endurance exercise test.

Basic Assumptions

1. Subjects exhibited a maximal performance on the treadmill GXT.

2. Subjects adhered to their respective exercise

prescription and did not engage in any other endurance activities in addition to the treatment program.

3. Subjects adhered to their reduced calorie diets.

Summary

Acute physical exercise causes a variety of alterations in body fuel homeostasis and hormonal regulation. Such alterations can be further modified by training as complex response pathways change their functions to oppose homeostatic perturbations. Eventually, an adaptation pathway will cause a persistant change in the manner in which the body derives energy for exercise and adapts to homeostatic perturbations.

This study will assess the effect of an 8 week aerobic walk/jog training program on the patterns of fuel utilization and mobilization during an acute exercise test conducted at 80% VO2 max before and after training. Emphasis was placed on glucose homeostasis and the hormonal and metabolic factors that contribute to its regulation. In addition, the effects of aerobic capacity and adiposity on the response of glucose to acute exercise were addressed.

Subjects for the study were sedentary, overweight (> 22% body fat), women (18-35). Presently there is a lack of information in the literature regarding hormonal and

metabolic alterations resultant from a combined diet and aerobic exercise program in such a population.

Chapter II

REVIEW OF THE LITERATURE

To present the reported knowledge pertinent to each topic under investigation in this study, the review of the literature is divided into six sections; Carbohydrate Metabolism, Cortisol, Insulin, Lactate, Rate of Perceived Exertion, and Obesity and Hormonal Status.

Carbohydrate Metabolism

The main function of carbohydrate is to serve as an energy fuel for the body (McCardle, Katch & Katch, 1981). The energy derived from the breakdown of carbohydrate is ultimately used to power muscular contractions as well as other forms of biological work.

Carbohydrate, stored in the muscle and liver as glycogen must first be broken down into the 6 carbon glucose molecule before it is released into the blood. Glucose can then enter cells to be used for work, or it can be stored as muscle or liver glycogen for later use. Glycolysis is the catabolism of glucose in the cell for energy. Glycogen stored in the muscle can be broken down during glycogenolysis to provide fuel for contractions. Liver glycogenolysis provides glucose which can circulate through the bloodstream to the working muscles.

There are two forms of glycolysis; anaerobic (without oxygen) and aerobic (with oxygen). Anaerobic glycolysis occurs in the sarcoplasm of the cell, rapidly breaking down the 6 carbon glucose molecules from either blood glucose or muscle glycogen into two lactic acid molecules, with a net production of two ATP molecules. The lactic acid produced can; (a) convert back to pyruvic acid and be broken down to carbon dioxide and water in the mitochondria; (b) diffuse to other muscles and be taken up and degraded for energy, or (c) be taken up by the liver where gluconeogenesis provides glucose to enter the blood and be transported back to the muscles for use or storage (the Cori Cycle).

Anaerobic glycolysis helps meet then energy requirements immediately at the onset of exercise, and during very maximal short bursts of energy. Anaerobic glycolysis cannot keep the muscles working longer than approximately 2 min at maximal workloads (Pernow, 1971). The excess lactic acid accumulating in the muscles may inactivate phosphorylase and phosphofructokinase so that glycolysis is depressed, ATP production declines, and work intensity must decrease (Gollnick & Hermansen, 1973).

During low and moderate intensity work, a lower rate of ATP production is required. Thus aerobic glycolysis and oxidation predominates. During aerobic glycolysis pyruvate

is not converted to lactate. Instead, pyruvate passes from the sarcoplasm into the mitochondria where a series of reactions breaks down each three-carbon pyruvic acid molecule into carbon dioxide and water. Ultimately carbon dioxide and water form 30 ATP molecules in addition to those derived from anaerobic glycolysis.

In the presence of oxygen, the mitochondria of the cells can produce energy from both carbohydrate and fat stores. The extent to which each substrate is utilized depends on the intensity, duration, and physical condition of the subject, and will concomitantly be reflected by the degree of blood glucose perturbation during such exercise.

During low intensity exercise (<60% VO2 max) there is greater activation of slow twitch fibers so that nearly all energy for ATP replacement is dependent upon the aerobic breakdown of fatty acids (Holloszy, 1973). Blood glucose values are not observed to decline during low intensity exercise unless the duration of the exercise bout is prolonged (3 hr) (Koivisto et al. 1982).

With moderate to high intensity exercise (approximately 60% VO2 max) increased plasma glucose levels are observed (Bloom, 1976; Sutton, 1978). This intensity, referred to as the anaerobic threshold, results in lactic acid accumulation which inhibits fatty acid release from

fat cells (Jones et al. 1980). This results in a decrease of serum fatty acids, thus increasing the dependence on carbohydrate for energy. Glycogen is an important fuel in this type of exercise and it is catabolized both anaerobically and aerobically. There is no danger of depleting glycogen stores in this type of activity that is sustained for less than 40 minutes.

At workloads above 70% VO2 max, an increase in plasma glucose is observed (Pruett, 1970). This may be explained by hormonal changes which occur during exercise. At high intensity exercise hepatic glycogenolysis, with the increase in glucose output is stimulated by the increase in catecholamines, glucagon (Galbo, 1975) and glucocorticoids (Sutton, 1981). Insulin is the only hormone that inhibits hepatic glycogenolysis, and its' concentration decreases with intense exercise. The general effect during high intensity exercise is to favor hepatic glycogenolysis, and thus increase blood glucose. If moderately intense exercise is prolonged liver glycogen stores become limiting and gluconeogenesis increases. Liver glycogen stores can become depleted, but only under such circumstances as prolonged exercise or starvation (Felig & Wahren, 1975). Hypoglycemia results during exercise under these conditions.

Carbohydrate metabolism is affected by repeated bouts of exercise. Resting levels of plasma glucose have not been significantly affected by most physical training programs (Hartley et al., 1972; Rennie & Johnson, 1974; Winder et al. 1979). However, LeBlanc et al. (1981) reported significantly lower fasting levels of plasma glucose in trained versus untrained subjects. The insulin levels of the trained subjects were also significantly lower. Because the trained subjects were very fit (mean VO2 max of 90 ml/kg/min) the training adaption of decreased levels of both glucose and insulin was readily observable compared to the untrained subjects (mean VO2 max of 30 ml/kg/min).

The acute blood glucose response to an exercise bout has also been shown to be modified by endurance exercise training. During exercise at 40% VO2 max, a significant increase in plasma glucose levels following training has been reported (Kovisto et al. 1982). During such low intensity exercise there is increased utilization of fat for energy with less dependence on carbohydrate for energy. This adaptation in fat metabolism is important because the increased reliance on fat for energy allows both muscle glycogen and blood glucose to be spared.

Plasma glucose levels are observed to increase in

response to acute exercise post-training when the intensity is greater than 60% VO2 max (Gyntelberg et al. 1977; Hartley et al., 1972; Winder et al. 1979). Winder et al. (1979) reported that glucose appeared to be maintained at a slightly higher level in response to exercise following training. Galbo et al. (1977) observed no significant differences between the mean pretraining and post-training fasting levels of blood glucose in trained and untrained rats. However, the mean peak glucose value increased from 153.2 mg/100 ml before training to 159.5 mg/100 ml after training. Similar results were previously reported by Richer et al. (1976). They observed glucose concentrations in rats to increase more during exercise post-training vs pretraining after a 12 week training program. Rennie & Johnson (1974) observed the same response in humans who participated in a 4 week aerobic training program. Blood glucose concentrations during the post-training run (moderate speed) were significantly elevated compared to the values before training.

Contimuation of glucose output by the liver contributes to the elevated plasma glucose levels observed post-training. Although the hormonal responses to exercise are usually blunted in trained individuals, the need for blood borne substrates is not diminished after training

(Galbo et al. 1977). Although trained subjects secrete less catecholamines than untrained subjects at similar relative workloads, the catecholamines still stimulate hepatic glucose output in response to strenuous work (Wahren et al. 1971). Concomittant with hepatic glucose output is the lessened reliance on blood glucose for fuel by the trained muscles. Training increases the glycogen stores in both the muscles and the liver. These increased glycogen stores are depleted more slowly than the glycogen stores in the untrained. These training adaptations decrease the need to rely on blood and liver glucose for energy. Since the trained muscle is less dependent on blood glucose for energy, an elevated blood glucose level will be maintained or even increased in response to exercise post-training.

Endurance exercise training results in a blunting of the acute responses of plasma insulin (Hartley et al., 1972). Because insulin decreases lipase activity, the decrease in insulin during exercise leads to an increase in hormone sensitive lipase activity and increases the release of fatty acids from fat stores. This facilitates the use of more fatty acids and less glucose for energy.

Endurance trained muscles have more and/or larger mitochondria and there is greater activity of the

mitochondrial enzymes of the Krebs cycle, electron transport system, and beta oxidation for fatty acid catabolism. Thus the capacity of the muscles to oxidize carbohydrates and fats increases following training. This will result in less lactic acid production and less inhibition by lactate of the lipase enzymes which mobilize fatty acids from triglyceride stores in adipose tissue and muscle. Accordingly, endurance trained persons have a greater ability to mobilize and utilize fat (Henriksson, 1977).

Thus endurance exercise training stimulates more pathways to decrease the reliance on blood glucose during exercise than does an acute exercise bout. The increased muscle glycogen stores, muscle enzyme adaptations, and changes in hormonal responses to exercise training allow the trained person to spare their sources of carbohydrate and utilize fat during exercise.

Cortisol

At least 95% of the glucocorticoid activity of the adrenocortical secretions in humans results from the secretion of cortisol (Guyton, 1981). The secretion of cortisol is controlled almost entirely by adrenocorticotropic hormone (ACTH) secreted by the anterior pituitary, which in turn is controlled by corticotropin

releasing hormone (CRH) from the hypothalamus.

The circadian rhythm of the secretory rate of cortisol is high in the early morning and low in the late evening (Guyton, 1981). The plasma cortisol level ranges between a high of about 20 ug/100 ml and a low of about 5 ug/100 ml. Cortisol has a rather long half life of 60 to 90 minutes, one of the reasons being that in plasma it is bound to the corticosteroid-binding globulin (CBG).

The glucocorticoids are essential for organisms to resist stressful situations, including the stress of severe exercise training (Lamb, 1984). Tharp (1975) reported that adrenalectomized animals are much less able to endure exercise than normal animals. However, almost any type of stress, whether it be physical or neurogenic will cause an immediate increase in ACTH, followed within minutes by a greatly increased adrenocortical secretion of cortisol.

Cortisol functions in the metabolism of carbohydrates, fats, and proteins by; (a) stimulating gluconeogenesis-increasing liver conversion of amino acids into glucose thereby increasing liver glycogen and blood glucose; (b) mobilizing amino acids from tissues and increasing liver amino acids, and (c) mobilizing fatty acids from adipose tissue to increase blood free fatty acids. Although cortisol stimulates the metabolism of different substrates,

the substrate favored will depend on the situation. During short-term intense exercise energy will be mainly derived from carbohydrate metabolism. During prolonged exercise of moderate intensity, cortisol will promote fat utilization and conserve carbohydrates.

Ahlborg et al. (1974) have shown that a significant fraction of the glucose output from the liver during prolonged exercise comes from gluconeogenesis. This gluconeogenic contribution increases with the duration of exercise. Thus the gluconeogenic actions of the corticosteroids are important in supporting the blood glucose level during exercise.

Examination of the literature however, reveals a wide variety of glucocorticoid responses to acute as well as training adaptations to exercise. In their reviews of the response of cortisol during light to moderate intensity exercise, both Terjung (1979) and Tharp, (1975) have reported studies in which the the plasma cortisol concentrations have been observed to increase, decrease, or exhibit no change. Conflicting reports probably resulted from a failure to relate changes in glucocorticoid concentrations to relative workloads (White et al. 1976). Davies & Few (1973) reported that the change in total plasma cortisol concentration was positively correlated

with maximal oxygen intake (VO2 max) providing exercise intensity exceeds 60% VO2 max. Thus, studies reporting that plasma cortisol concentrations and urinary excretion rates of free cortisol are enhanced by prolonged intense (>80% VO2 max) exercise are more in agreement (Bonen, 1976; Desypris, 1980; Kuoppasimi, 1980; Gambert et al. 1981).

Tharp (1975) reported that plasma glucocorticoid levels in human subjects may exhibit a decrease at exhaustion. Such a response however, is most often seen in animal studies (Frenkl, 1971) due to the fact that humans will usually terminate the exercise before reaching this point.

Some evidence is suggestive of the variability in the corticosteroid response to exercise relative to fitness status. Metivier et al. (1969) found that in low intensity bicycle ergometer exercise, untrained individuals demonstrated a progressive rise in plasma cortisol, whereas well trained individuals showed an initial decrease, followed by no change during the work period. However, Hartley et al., (1972) observed no significant difference in the corticosteroid response to exhaustive bicycle exercise of moderate intensity (73% VO2 max) following a 7 week exercise conditioning program. Chin & Evonuk (1971) also reported no significant difference in plasma

corticosterone levels between the non-exercised control rats and the moderately exercised rats in response to training. However White et al. (1976) compared serum corticoid levels between a group of sedentary middle-aged males who underwent a 4 month physical fitness program and reported significantly lower mean corticosteroid levels during the post-training exercise tests as compared to the pretraining exercise tests. This result may be viewed as a reduction in the amount of physical stress, as well as in increase in stress tolerance Additionally, the duration of the program may have blunted the increase in the response of cortisol to exercise.

The difference in the effect of chronic exercise on corticosteroid response may be the result of differing durations of the conditioning programs. For example, while White et al. (1976) noted a significant decrease in mean corticosteroids in response to a 4 month program, Hartley et al., (1972) failed to demonstrate a significant reduction in corticosteroid response to exercise resulting from a 7 week training program.

It is recognized however that differential contribution of psychological and physiological components of stress during exercise probably accounts for some of the variability in the corticosteroid response to exercise

observed in research studies (Tharp, 1975). The classic study by Hill et al., (1956) on the Harvard rowing crew provided the first solid evidence of the importance of psychological factors on glucocorticoid secretion as related to exercise. They further reported that glucocorticoids increased significantly on race and time trial days over practice days even though the muscular work accomplished was comparable on those days. The added emotional stress of competition appeared to be the more important contributor to the increased glucocorticoid levels.

More recently, Mason et al. (1973) reported strikingly consistant individual responses and significant mean elevations of plasma cortisol (5 to 9 ug%) during a 20 min interval prior to the onset of the first exhaustive exercise (70% VO2 max) session in eight normal young men. Such consistant anticipatory psychoendocrine responses were not found prior to subsequent exercise sessions. These responses appear to reflect psychoendocrine reactions in immediate anticipation of severe exercise.

Nearly all exercise studies reported thus far have measured changes in plasma corticosteroid. It must be recognized that plasma levels provide only limited information about glucocorticoid response because such

levels can be modified by secretion rates, binding to plasma proteins, removal by tissues, degradation, and excretion.

Any changes in plasma cortisol reflect the net effect of both the rate of secretion, and the rate of removal from the plasma (Few, 1974). Serial measurements of plasma cortisol concentration give a useful indication of changes in the rate of cortisol secretion and the time when such changes occur (Cashmore et al. 1977). After analyzing data from ten experiments on men who exercised for one hour at constant workloads greater than 65% VO2 max after 3H cortisol. They reported that occasionally exercise at work exceeding 70% VO2 max fails to elicit a significant rise in plasma cortisol level. It was suggested that this could be due to failure to increase the rate of cortisol secretion or to an unusually high rate of cortisol removal.

Insulin

The hormone insulin is secreted from the beta cells of the pancreatic islets of Langerhans. It is composed of two amino acid chains that stimulates glucose uptake by many cells, of which muscle and adipose are most important. Insulin also promotes glycogen synthesis in the muscle and liver, and triglyceride synthesis in adipose tissue.

The hormonal response to moderate exercise is

characterized by a fall in plasma insulin. This was demonstrated by Bjorntorp, (1970) who exercised sedentary, overweight subjects. Similar findings have been reported in acute, moderate intensity (>60% VO2 max) exercise in non-obese subjects (Hartley et al., 1972; Gyntelberg et al. 1977; Wirth et al. 1979).

Although insulin decreases during exercise, a small amount is necessary in regulating glucose uptake in the working muscles (Vranic et al. 1976). Because more blood is circulating through the muscles during exercise, the elevated plasma glucose concentration is less dependent on insulin to initiate uptake of glucose by the working muscles (Pruett, 1970). At the same time, blood flow to the liver is reduced so that there is less insulin circulating through the liver where insulin tends to block glucose delivery from the liver to the blood (Lamb, 1984). Thus the liver is able to supply additional glucose to the muscles.

The insulin decrease appears to be more pronounced in unfit or untrained subjects (Rennie & Johnson, 1974; Sutton, 1977, 1978; Winder et al. 1979). Trained or fit subjects exhibit a blunting of the decrease in the insulin response to moderate intensity exercise training. The elevation of blood catecholamines is proportional to

percent VO2. The exercise induced increase in catecholamines from the sympathetic nervous system simultaneously supresses insulin and stimulates hepatic glycogenolysis (Hartley et al., 1972). This catecholamine supression of insulin release would be expected to be less in trained subjects since catecholamines demonstrate a blunting response to exercise conducted at the same absolute workload postraining.

Another suggestion by LeBlanc et al. (1979) concerns the reduced adiposity of the better trained subjects. More insulin was secreted by obese subjects than trained subjects during a glucose tolerance test. Thus the authors suggest that it is the percentage of fat, not VO2 max that is related to the level of plasma insulin. Accordingly, by reducing the percentage of body fat, the insulin secretion rate would decrease proportionally.

Other factors suggested to explain the blunted response of insulin to exercise following training include: diminished pancreatic secretion of insulin (LeBlanc, 1982), enhanced peripheral sensitivity to insulin (Koivisto et al. 1980), and enhanced clearance of insulin (Wirth et al. 1981).

Controversial results regarding the effect of physical training on resting concentrations of insulin in the

plasma have been reported. When untrained subjects initiate a training program, no change in resting plasma insulin concentrations is noted (Gyntelberg et al. 1977; Winder et al. 1979). However, comparing endurance trained athletes to nonathletes, Wirth et al. (1981) reported that basal plasma insulin concentrations were lower in the athletes. Koivisto et al. (1979) and Lohman et al. (1978) have reported lower resting plasma insulin concentrations in highly trained athletes. Bjorntorp (1981) reported that insulin levels are inversely associated with the mass of adapted muscle tissue. This may suggest why highly trained endurance athletes readily demonstrate a lower plasma insulin level compared to subjects who have not acquired the same training status.

The decline in plasma insulin in response to exercise helps to minimize glucose uptake by nonactive tissues, thereby sparing blood glucose for active muscle and brain tissue. Also, the decreased insulin concentrations during exercise promote fat utilization by the muscles (Bukoweicke et al. 1980). The release of fatty acids into the blood from triglyceride stores is inhibited by elevated insulin and lactic acid levels. Thus this decreased level of insulin is one of the most important hormonal changes for activating the lipases and stimulating the release of fatty

acids.

Although the decrease in insulin is observed to be less in trained individuals, they are better able to rely on fat for energy. Trained individuals have more enzymes to increase the release of fatty acids from fat stores. They also have increased oxidative capacities to utilize fat for energy. Thus the overall effect is that trained individuals rely more on fat than glucose for energy during exercise versus untrained individuals (Astrand & Rodahl, 1977). This is reflected in the lower respiratory quotient (R) in trained versus nontrained subjects during exercise at the same absolute workload (Karlsson et al. 1973; Winder et al. 1979).

Lactate

Carbohydrate, glucose from the blood or glucose derived from glycogen is the only energy metabolite that can be used for energy anaerobically. Maximal exercise (>90% VO2 max) requiring short bursts of energy, as well as heavy intermittant exercise, relies primarily on anaerobic glycolysis to supply ATP (Pernow, 1971). The end product of anaerobic glycolysis is lactate. Thus the concentration of lactate in the blood reflects the degree to which anaerobic metabolism is occurring.

During aerobic exercise when the intensity exceeds

approximately 60% VO2 max, lactic acid accumulates in the blood (McCardle, Katch, & Katch, 1981). The inability to generate further energy aerobically is often referred to as the anaerobic threshold; " the level of exercise at which the anaerobic production of energy through glycolysis leads to the rapid accumulation of lactic acid in the blood" (Lamb, 1984).

Lactic acid is produced and removed from the blood at equal rates during rest and easy aerobic exercise (Brooks & Fahey, 1984). Although lactic acid turns over very rapidly, its concentration in the blood may not change. At heavier intensities (76% VO2 max) when blood lactate levels are elevating, latate production is exceeding removal. Once the circulation delivers additional oxygen needed for muscle respiration, the excess lactic acid will be removed.

In addition to anoxia, several other factors operate to change the blood lactate level. Fast contracting white skeletal muscle fibers produce lactate when they are recruited to contract whether oxygen is present or not (Holloszy, 1973). Sympathetic mediated release of glucagon in response to increasing exercise intensity causes glycogenolysis in both the muscle and the liver (Gyntelberg et al. 1977). This increase in glucose availability

results in an excess of pyruvate. This excess pyruvate is converted to lactate in the muscle and appears in the blood. Because blood is shunted to the muscles and away from the organs which ordinarily remove lactate (liver and kidney), blood lactate levels increase.

The increased hydrogen ion concentration causes the pH to decrease and within the muscle the lower pH may inhibit phosphofructokinase (PFK) and slow glycolysis. Hydrogen ion concentration may also interfere with muscle concentration by displacing calcium from troponin. High hydrogen ion levels also inhibit the release of free fatty acids into the circulation. Fat oxidation in the muscle is directly dependent upon the the circulating free fatty acid levels. Also, low pH levels may stimulate pain receptors, thus precipitating fatigue (Brooks & Fahey, 1984).

Fatigue will occur earlier in an untrained person exercising at 75% VO2 max than in an endurance trained person exercising at the same workrate. This may be partly related to the decreased lactate accumulation during exercise post endurance training. Physically trained individuals have lower blood lactate levels during submaximal exercise compared to untrained individuals (Holloszy, 1973). After a 10 week aerobic training study, Gyntelberg et al. (1977) reported blood lactate levels

increased significantly less during exercise at the same absolute work load after training. Winder et al. (1979) reported similar results from six initially untrained subjects following 9 weeks of endurance training. Sutton (1978) compared fit subjects to untrained subjects and reported similar lactate levels at rest, but significantly increased lactate levels in response to exercise in the untrained subjects, as compared to the fit subjects during an exercise test conducted at the same relative submaximal workloads.

Lower lactate levels after training indicate that; (a) less carbohydrate has been used anaerobically (Karlsson et al. 1973); (b) more lactic acid has diffused out of the muscles and into the blood and has been degraded for energy by other muscles, and/or (c) lactic acid has diffused to the liver where it is changed into liver glycogen via the Cori Cycle.

The lower lactate levels are beneficial since lactic acid inhibits fatty acid release from fat stores (Bukoweicki et al. 1980). Thus with less anaerobic metabolism there will be less lactic acid produced to inhibit the lipase enzymes which mobilize fatty acids from triglyceride stores in adipose tissue and in muscle. Consequently, more fatty acids will be delivered to the

working muscles to be utilized for energy. This training induced adaptation is also reflected by the lower R value at the respective work loads post-training, indicating that a higher proportion of fat, and less carbohydrate is being oxidized to produce ATP.

Rate of Perceived Exertion (RPE)

Borg (1970) initiated and validated research in measuring perceived exertion with the development of his model which linearly linked the perceptual response during exercise with heart rate as an index of strain. However, he had also acknowledged that perception of effort was dependent upon input from both "the musculature and the system of circulation". However, due to the linear association between RPE and heart rate, perceived exertion was most influenced by the circulatory system.

Having composited research regarding sensory inputs contributing to perceived exertion, Ekblom and Goldbarg (1971) suggested that a one factor model was too simplistic to interpret an individual's subjective rating of perceived exertion. Rather, they reaffirmed Borg's previous acknowledgement of an individual evaluating his perceived exertion during physical work from both local and central parameters, and suggested such parameters be addressed when investigating perceived exertion.

A most recent development by Borg (Noble et al. 1983) has readdressed the consideration that input from the musculature affects perception of effort. Lactate is a physiological variable associated with the musculature. In observing that this variable is related to exercise intensity according to a non-linear power function (Borg, 1970), a new psychophysical scale was developed. This new scale, referred to as the category-ratio scale of perceived exertion, parallels perceptual ratings with both muscle and blood lactate as a positively accelerating function.

The relevance of lactate metabolism to exercise performance is related not only to short term maximal intensity exercise, but is associated with steady state endurance exercise as well (Farrell et al. 1979; Kinderman et al. 1979). This was discussed by Jacobs (1981), who observed that ratings of perceived exertion, as measured by Borg's new category-ratio scale, were positively related to both blood and muscle lactate concentrations which accumulated during steady state exercise. These findings are in contrast to Lollogen et al. (1980) who observed no such relationship during a single exercise bout. This disparity may have occurred because Lollogen et al. (1980) used the Borg Scale, designed to increase linearly with heart rate, rather than the category-ratio scale.

Physical endurance training has been shown to reduce the accumulation of blood lactate during exercise at submaximal work rates (Davies et al. 1976). Also, physically trained individuals have lower blood lactate levels than untrained subjects during submaximal exercise at the same relative workloads (Ekblom et al. 1968). It is further suggested that a decreased accumulation of lactic acid may reduce subject's RPE because the feeling of exertion depends largely on the rate of lactic acid accumulation (Astrand & Rodahl, 1977; Noble et al. 1983).

Obesity and Hormonal Status

Many obese subjects have reduced insulin sensitivity compared to lean subjects. Also, some obese subjects have higher resting insulin levels which increase more in response to a glucose tolerance test compared to the insulin response of lean subjects (Bjorntorp et al., 1977). Some research has suggested that endurance exercise training reduces plasma insulin levels and increases sensitivity to insulin (LeBlanc et al. 1979).

LeBlanc et al. (1979) reported that highly trained subjects show a normal tolerance to injected glucose in spite of a markedly reduced insulin response compared to non-trained subjects. The authors suggested that these findings could be explained primarily by the reduced

adiposity observed in trained subjects rather than by the increase in VO2 max. They concluded that because of the decreased body fat the secretion of insulin is also reduced. However, the glucose tolerance is not changed by this condition because of the training induced enhanced sensitivity to insulin.

In continuation of this hypothesis LeBlanc et al. (1981) studied both trained and non-trained subjects who exercised for one hour at 70% of their respective VO2 max. To observe the effect of a nutritional factor on insulin sparing action of exercise the subjects ate ad libidum on one occasion, and were imposed a restricted diet on the other occasion. Under both conditions, the subjects refrained from exercising three days prior to the exercise test. Glucose tolerance was also studied under these conditions.

The trained subjects, when active and eating ad libidum had a lower insulin response to a glucose tolerance test compared to non-trained subjects eating ad libidum. When the trained subjects are inactive for three days but eat ad libidum, the rise in insulin during a glucose tolerance test is greater than when the same subjects maintain their training routine. However, when the trained subjects are put on a restrictive diet while

inactive for three days, they retain their low basal insulin levels and responded to a glucose test in the same manner as when they ate ad libidum and exercised. The non-trained subjects exhibited lower plasma insulin levels following a single bout of exercise which were similar to the low insulin levels observed in response to a three day restrictive diet while inactive. Thus, it was suggested that the beneficial effect of exercise training on insulin is not related to the VO2 max of the subject, but the insulin response is suggested as varying with the basal insulin levels.

The lower fasting blood sugar results from either exercise while eating ad libidum, or restrictive eating without exercise. The low basal insulin would increase the tissue sensitivity and reduce the tissue requirements during exercise.

Similar findings were also reported in male rats (Richard & LeBlanc, 1980). Male rats were divided into three groups; sedentary free eating controls (C); exercise trained (T); and sedentary pair-weighted. After exercise training the animals were subjected to a glucose tolerance test. Results showed that exercise training led to a significantly smaller adipose tissue mass of both exercise trained and pair-weighted rats than that of the free eating

controls. There was also a reduction in basal as well as glucose-stimulated insulin without any impairment in glucose tolerance in both the trained and pair-weighted rats. These results suggest that the reduction in body fat mass rather than the level of physical training was the main factor responsible for the reduced insulin and enhanced hormone sensitivity.

Leon et al (1979) observed a 43% reduction in plasma insulin levels in 6 sedentary overweight men who participated in a 16 week vigorous walking program. Although no attempt was made to influence their diet, the dietary records of the subjects showed that the caloric intake failed to keep pace with the increased energy expenditure. As a result, the mean body fat dropped from 25.3% to 17.4%. The authors did not state whether the reduced insulin occurred because of training or body fat loss.

Reduced insulin levels have been observed in the absence of weight loss (Bjorntorp, 1970). These results were observed in patients with hyperplastic obesity who showed increased insulin sensitivity without a change in their amount of fat. However, Bjorntorp suggested that patients with hyperplastic obesity seem to be excluded from the normally observed weight decreasing effects of physical

training.

Thus the explanation for improved glucose tolerance and increased insulin sensitivity to decreased levels of insulin after physical training is not due soley to exercise training, but is probably due to the combination of the effects of decreased weight and body fat in conjunction with the exercise training.

Summary

Metabolic, hormonal, physical and psychological responses to acute exercise affect body fuel homeostasis and hormonal regulation. Such responses can be further modified by endurance exercise training. The preceding literature review presented the effect (s) of exercise on carbohydrate metabolism and the involvement of metabolic (lactate) and hormonal (insulin, cortisol) factors. The effect of obesity on hormonal status was also presented. Additionally, the psychological response to exercise was addressed in the section on rate of perceived exertion.

The manner in which one responds both physically and psychologically to exercise is dependent on the physical characteristics of the subject, as well as the intensity and duration of the exercise.

CHAPTER III

JOURNAL MANUSCRIPT

Effects of Aerobic Exercise and Weight Reduction on Carbohydrate Metabolism during Submaximal Exercise in Sedentary, Overweight Women.

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EFFECTS OF AEROBIC EXERCISE AND WEIGHT REDUCTION ON CARBOHYDRATE METABOLISM DURING SUBMAXIMAL EXERCISE IN SEDENTARY, OVERWEIGHT WOMEN.

by

Janine M. Toepfer

(ABSTRACT)

Hormonal and metabolic responses to submaximal exercise were studied in 11 sedentary, overweight women who participated in an 8 week aerobic exercise program (80% VO2 max) while consuming a hypocaloric diet. A maximal and submaximal treadmill exercise test were performed before and after the program. During the submaximal exercise test, a graded portion (mean time 6.4 min) preceded the submaximal phase during which subjects worked at 80% VO2 max until exhaustion (mean time 12 min). Blood was sampled before and after the work via venipuncture. Whole blood was immediately analyzed for lactate accumulation. The remaining blood was centrifuged, separated, and frozen for subsequent serum glucose, cortisol, and insulin measurement. There was a significant increase in oxygen uptake (ml/kg/min), and a decrease in body weight, (6.7%), and body fat (14%). Resting heart rate was significantly

lower post-training (5.4%), as were exercise RQ (VCO_2/VO_2) ratios. Pretraining serum glucose and blood lactate significantly increased while nonsignificant decreases were noted in insulin and cortisol as a result of the submaximal exercise bout. The significant increases in glucose and lactate during exercise were blunted after the training program. However, only the post-training response of lactate was significantly different from the pretraining response. The insulin and cortisol response was not significantly different from that during the pretraining exercise test. A significant correlation was observed between RPE and lactate at the end of exercise both pretraining and post-training. In summary, the combined exercise and weight loss program resulted in exercise being less stressful, both metabolically and subjectively. This improvement enables greater exercise intensity to be performed prior to the significant accumulation of lactate and perception of fatigue which may inspire the sedentary, overweight female to establish and/or continue a regular exercise program.

Introduction

Acute physical exercise causes a variety of alterations in body fuel homeostasis and hormonal concentrations. Such alterations can be further modified by training as marked differences exist between physically trained and untrained individuals. For example, trained individuals have lower blood concentrations of lactate, a metabolic factor reflective of fuel utilization during exercise. Lower blood lactate levels suggest a greater reliance on oxidative metabolism during exercise with less use of the anaerobic glycolysis pathway of ATP generation. Trained subjects may also exhibit a modification in endocrine function in response to exercise (11). Thus, training status may be evaluated by measuring the changes in hormonal and metabolic parameters which regulate carbohydrate and fat metabolism during exercise. Metabolic parameters during submaximal exercise are considered by some to be more reliable indicators for the state of conditioning than VO2 max (14).

The object of this study was to assess the effect of aerobic training on the patterns of fuel utilization and mobilization during an acute exercise test conducted before and after an 8-week walk/jog program administered to sedentary, overweight women. During the 8-week program,

subjects followed a weight reduction diet, thus the percentage of adiposity was be obtained for all subjects to assess the role of this variable in the control of fuel utilization and mobilization.

Methods

Eleven sedentary, overweight (> 22% body fat) (12) females (aged 18-35) volunteered to participate in this study. After the study was explained to the subjects, a written consent was obtained (Appendix C).

Pre-experimental testing included the determination of maximal oxygen consumption (VO2 max). A modification of a Balke ramp protocol was used (Appendix H). Subjects walked at 3.4 miles per hour. At two minute intervals the grade increased by 2.5% corresponding to an overall increase of 0.5 mets/workload until the subject voluntarily ended the test. Treadmill grade and velocity that would require VO2 levels corresponding to 80% VO2 max were determined for all subjects (1) (Appendix I). On another day within the same week, a submaximal exercise test was conducted. Subjects were instructed not to eat 2 hr prior to the test and to refrain from physical exercise for 24 hr prior to the test (Appendix F). The beginning of the exercise test followed the VO2 max protocol until the workload approximating 70% VO2 max was achieved. The speed and grade were then

adjusted to previously calculated workloads corresponding to 70% VO2 max. However, actual VO2 values showed that subjects were exercising at a mean VO2 (24.2 +1.6 ml/kg/min) which was approximately 9.5% above the mean calculated VO2 (22.1 +1.3 ml/kg/min). Although subjects were encouraged to maintain this workload for 20 min most subjects terminated the test due to volitional fatigue. This resulted in different exercise test durations for each subject. During the exercise test, rate of perceived exertion (RPE) using Borgs' category-ratio scale was recorded every fifth minute and at the minute prior to test termination (terminal RPE) (Appendix J). A5 ml blood sample was taken via venipuncture just before and immediately after exercise. Whole blood was immediately analyzed for lactate accumulation using the YSI Scientific Model 23L lactate analyzer. The remaining blood was then centrifuged, and serum was separated, and frozen. Serum glucose was measured by the O-Toludine technique (6) (Appendis K). Cortisol (8), and insulin (10), were measured by radioassay. The anti-insulin serum was obtained from Immunonuclear Laboratories. Coeffiecients of determination, evaluated by duplicatemeasurements of the same sample for cortisol and insulin were .97 and .99, respectively. The within and between assay variance of

glucose was evaluated by duplicate measurements of the same samples in two different assay runs. The intra-assay and inter-assay coefficients of determination for glucose are 4% and 5%, respectively.

The training began two days following the submaximal exercise test. Training consisted of a supervised 30 min aerobic walk/jog program conducted outdoors, 3 days/week, for 8 weeks. During this program subjects exercised at their respective exercise prescriptions. The exercise prescription was based on a heart rate value which corresponded to 80% VO2 max. The exercise leaders intermittently checked subjects heart rates to ensure that subjects were exercising at the proper intensity, as well as correctly monitoring their heart rates. Subjects attendance was recorded per exercise session, and subjects weight was recorded weekly at a diet counseling group session. Throughout the 8 week treatment, subjects were encouraged to follow a 1200-1400 calorie diet adapted from a diabetic exchange list diet. This diet was designed to result in a weight loss of 1 kg/week.

After the 8 week training, subjects were again administered a VO2 max GXT, and a submaximal exercise test. During the submaximal exercise test, duration, power output, and measurements taken were identical to the

pretraining submaximal exercise test.

SAS computer programs were used to conduct statistical analyses. Dependent t-tests were performed to evaluate differences between pretraining and post-training variables. Correlations were conducted to identify relationships between variables. The level of significance was set at $P<.05$.

Results

Efficacyof the training and diet program as determined by the classical criterion of maximal oxygen intake showed that there was a significant increase in relative oxygen intake (ml/kg/min), but the difference in average absolute oxygen intake (L/min) was not significant (Table 1). The training program significantly affected average body weight and fat of subjects. There was a mean decrease int he percent change of body weight (6.7%) and body fat (14%).

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Insert Table 1 Here.

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Additional evidence of metabolic and physiological adaptations to training was evident from measurements made during the submaximal exercise test. The post-training exercise test was conducted at the same absolute workload, for the same duration as the pretraining exercise test

(Table 2). Resting heart rate was significantly lower post-training as compared to pretraining. Although respiratory data was collected from only six subjects post-training, the subjects' mean respiratory exchange (R) was significantly lower than mean R values pretraining. Subjects' RPE values were lower both at minute 5, and at one minute prior to test termination post-training. However, these differences were not significant.

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Insert Table 2 Here.

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Table 3 presents the results of serum hormone and metabolic analysis. Because one subject did not follow dietary instructions prior to the exercise test, her serum glucose and insulin values are not included in the means presented.

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Insert Table 3 Here.

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Pretraining, the mean serum glucose at rest was approximately 14% below the normal range (80 mg/dl to 90 mg/dl). Significant increases were noted in the response of both glucose (35%) and lactate (84.5%) at the end of the exercise bout. The decrease observed in both insulin and

cortisol in response to pretraining exercise was not significant.

Post-training, mean resting glucose was significantly higher (25.3%) than pretraining. The mean resting lactate was significantly lower (39.8%). The mean resting concentration of cortisol was lower than the pretraining value (25%). The 30.1% increase in glucose in response to exercise posttraining was not significantly less than the increase pretraining. Lactate significantly increased in response to exercise post-training. However, this increase was significantly less than the increase in lactate in response to exercise pretraining. The decrease observed in both insulin and cortisol in response to post-training exercise was not significant.

When the relationship between variables is examined, a significant correlation was observed between terminal RPE and lactate pretraining (r= .51), and post-training (r= .64). However, the change in terminal RPE was not significantly related to the change in immediate post exercise lactate after training. Neither the change in body fat nor VO2 (L/min) was significantly correlated with the response of lactate post-training. The change in insulin was not related to the change in glucose in response to training.

Discussion

These non-trained sedentary subjects had below average initial VO2 max values, and elevated resting heart rates prior to training. After eight weeks of aerobic training and dietary intervention, significant differences were observed in their physical and cardiovascular parameters. These subjects, however, were considered to be highly motivated. They attended 92% of the nutritions sessions, and 88% of the exercise sessions. Target heart rate data collected during the exercise sessions showed that subjects were consistantly (90%) exercising within their target heart rate range.

The significantly elevated lactate after exercise suggests that these subjects of low fitness were in fact relying more on anaerobic carbohydrate metabolism pretraining than on aerobic metabolism to produce ATP. Thus, 80% VO2 max appeared to be above the anaerobic threshold for these women. The high rate of ATP regeneration that is necessary for moderately intense exercise, plus the inability of their untrained cardiovascular system to supply adequate oxygen to the muscles contributed to the accumulation of blood lactic acid. This excess lactic acid precipitated fatigue in the untrained subjects as reflected by their terminal RPE

values. Terminal RPE values were higher than expected due to the fact that subjects were actually exercising at an intensity approximately 9.5% above their calculated 70% VO2 peak. The inability of most of the subjects to endure such an exercise intensity is further indication of their untrained condition.

The respiratory exchange ratio during the submaximal exercise bout provides further evidence for the reliance on anaerobic carbohydrate metabolism. The mean R of 1.0 suggests that most ATP was being provided from carbohydrate sources. Fatty acid oxidation was probably minimal due to the elevated lactic acid which would inhibit the release of fatty acids from fat stores (4).

The initial conflict between the theoretical increase in the use of carbohydrate for energy, with increased blood glucose observed during exercise may be explained by the hormonal changes which occur during exercise at 70% VO2 max (20). During high intensity exercise, hepatic glycogenolysis increases output of glucose into the blood. This is stimulated by the increase in catecholamines (7), and glucocorticoids (25) which occurs in response to exercise at such an intensity.

Cortisol was within normal range both pretesting and post-testing. However, cortisol was significantly lower at

rest post-training. This is attributed to a decrease in subjects psychoendocrine anticipatory the psychoendocrine anticipatory response to blood draw, as well as to exercise testing, experienced by the subjects (17). Glucose was lower than normal at rest pretraining, and near the upper end of the normal range post-testing. Thus a significant difference was observed between the two average concentrations. This is not the usual reported response as resting levels of plasma glucose are reported as not being significantly affected by most physical training programs (11, 22, 27).

Resting insulin was higher post-training than pretraining. Again, after considering the elevated response of resting glucose post-training, this insulin concentration does not seem too suprising. However, when untrained subjects with normal plasma insulin concentrations initiate a training program, usually no change in resting plasma insulin concentration is noted (9, 26). Mean resting insulin decreased after training (3, 16). Bjorntorp (3) reported that resting insulin concentrations are inversely associated with the mass of adapted muscle tissue which also suggests that trained subjects would demonstrate a lower concentration of insulin at rest.

A possible explanation for the unexpected increase in resting glucose and insulin may be due to a change in dietary consumption prior to blood sampling. Since subjects were only fasted for two hours to simulate normal exercise conditions during a weight reduction program, serum insulin could be affected by pre-exercise food choice.

As observed before training, glucose increased during exercise post-training. The continued use of carbohydrate substrate during the submaximal exercise test after training was evident from the high R (.96). The source of increased blood glucose would again be the result of hepatic glycogenolysis. However, the hormonal responses to exercise are usually blunted after training, and trained subjects would be expected to secrete less catecholamines and glucocorticoids than untrained subjects at similar relative workloads (26). Concomitant with the decreased hepatic glucose output, is the reduced reliance on blood glucose for fuel by the trained muscles. Training increases the glycogen stores in both muscle and the liver, and increases the capacity of muscle to oxidize fats (19). These training adaptations decrease the need to rely on blood and liver glucose for energy, and explain the reduced increase in blood glucose after aerobic training.

No significant change was observed in the response of insulin to exercise pretraining or post-training. Insulin usually decreases during exercise, and this response is usually blunted in trained subjects (9, 11, 22, 24, 28). Because the decline in plasma insulin is proportional to both the duration and intensity of exercise (5, 14, 21) the time course of the submaximal exercise test may not have been long enough for changes in insulin to be observed. At workloads of approximately 70% VO2 max significant changes in insulin are observed after a minimum of 15 minutes (11, 28).

Although subjects decreased their caloric intake below energy requirements throughout the training program, a significant change in resting levels of insulin was not observed. It has been shown that after subjects reduce their caloric intake, as well as their percentage of fat, basal insulin levels are reduced (15, 16, 23), independent of a change in VO2 max. The duration of fasting seems to be critical as the results of studies reporting a decrease in insulin used subjects who had fasted overnight (12 hr) prior to blood sampling.

The response of lactate to exercise post-training however, clearly demonstrated that subjects experienced a training effect. The decreased resting levels, along with

the reduced increase in lactate in response to the same relative workload was observed post-training. Lower lactate after training indicate that less carbohydrate has been used anaerobically (12). Although only a slight decrease was observed in the mean R values post-training, it further demonstrates less of a reliance on carbohydrate sources of metabolism post-training. Thus, with less anaerobic metbolism there will be less lactic acid produced to inhibit the lipase enzymes which mobilize fatty acids from triglyceride stores both in adipose tissue and in muscle (4). Consequently, more fatty acids will be delivered to the working muscles to be utilized for energy. This training induced adaptation indicated that a higher proportion of fat, and less carbohydrate was being oxidized to produce ATP.

Another consequence of the decreased lactate produced is a reduction in RPE during exercise since the perception of exertion depends largely on the rate of lactic acid accumulation (2, 20). There was a significant relationship observed between lactic acid and RPE pretraining and post-training as measured by Borgs' category-ratio scale.

Also, after 8 weeks of aerobic training and dietary intervention, significant differences were observed in subjects' physical (decreased body weight and body fat),

and cardiovascular (increased VO2 max, and decreased resting heart rate) parameters. These adaptations contributed to the altering of metabolic and hormonal parameters which reduced anaerobic metabolism during exercise, thus reducing the reliance on blood glucose during exercise.

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Table 1

Subject's body weight, VO_2max, and body fat.

		Begin	8-wk	T	PR> |T|
Weight (kg)	n=11	73.3±5.1	68.3±4.8*	-6.86	0.0001
VO_2max (ml/kg/min)	n=11	30.5±1.93	36.5±1.8*	2.69	0.0195
VO_2max (l/min)	n=11	2.3±0.06	2.4± .36	2.04	0.0650
Body fat (%)	n=11	32.3±1.3	27.8±2.2*	-6.39	0.0001

Values are means ± SEM

* Significantly different from the pretraining response ($p<0.05$)

Table 2

R, RPE, and Heart Rate during submaximal exercise.

		Pretraining	Posttraining	T	PR> |T|
Time (min)[a]		12 ±1.9	12 ±1.9		
Speed (mph)		5.0± .18	5.0± .18		
Grade (%)		2.2± .3	2.2± .3		
RQ[b]	n=6	1.0± .12	.96±.09*	-2.46	0.0572
RPE[b]	n=11	6.1±2.9	5.7±2.8	-2.08	0.0647
RPE[c]	n=11	9.5±1.8	9.0±2.1	-2.04	0.0683
Heart rate at rest ($bt \cdot min^{-1}$)	n=11	93 ±5.0	87.6±4.0*	-2.97	0.0141

Values are mean ± SEM

* Significantly different from the pretraining response (p 0.05)

[a] Mean number of minutes spent at 70% VO_2max

[b] Taken at minute 5 during the endurance phase

[c] Taken one minute prior to test termination

RPE = Rate of Perceived Exertion

$RQ = \frac{VCO_2}{VO_2}$

Table 3

Responses of serum glucose, insulin, cortisol, and blood lactate to submaximal exercise.

		Pretraining		Posttraining	
		Rest	IPE	Rest	IPE
Serum glucose (mg/dl)	n=10	68.7± 7.9	104.9± 12.6*	86.1± 9.9†	112.0±14.4*
Serum insulin (ng/ml)	n=10	.39 ±.1	.32± .1	.42± .1	.32± .0
Serum cortisol (ug/100 ml)	n=11	11.7 ±2.6	10.0 ± 1.7	8.4 ±1.5	7.2 ±1.7
Blood lactate (mmoles/L)	n=11	.9 ± .2	5.8 ± .5*	.68± .1	4.6 ± .4*††

Values are means ± SEM.

IPE = immediate post exercise

* Significantly different from the resting value ($p<.05$)

† Significantly different from the pretraining value ($p<.05$)

†† The change in the pretraining response (IPE - rest) is significantly different from the change in the posttraining response (IPE - rest) ($p<.05$)

Chapter IV

Summary

This study was conducted to assess the alterations in body fuel homeostasis and hormonal regulation that occur with aerobic training and dietary innervention in originally sedentary, overweight women.

Eleven women participated in an eight week aerobic walk/jog program conducted at 70% VO2 max. In addition to aerobic exercise, subjects were following a 1200-1400 calorie diet based on the diabetic exchange list diet, designed for weight loss of approximately 1-2 lbs/week. Before and after the training program, subjects underwent both a max GXT, and a submaximal exercise test (70% VO2 max). Blood (5ml) was taken before and after the submaximal exercise test via venipuncture. Whole blood was immediately analyzed for lactate accumulation using the YSI Scientific Model 23L lactate analyzer. The remaining blood was then centriguged, seperated, and frozen. Serum glucose was measured by the O-Toludine technique (6). Cortisol (8), and insulin (10), were measured by radioimmunoassay. The anti-insulin serum was obtained from Immunonuclear Laboratories. SAS computer programs were used to conduct statistical analyses. Dependent t-tests were conducted to examine differences between pretraining and post-training

post-training variables. Correlations were conducted to identify relationships between variables. The level of significance was set at $p<.05$.

Pretraining, serum glucose and blood lactate significantly increased. The decrease observed in both insulin and cortisol in response to pretraining exercise was not significant. Post-training the increase in both glucose and lactate was significant. However, only the increase in in lactate was significantly different from the pretraining response. A significant correlation was observed between RPE and lactate both pre ($r=.51$) and post-training ($r=.64$).

There was a significant increase in oxygen uptake (ml/kg/min), decrease in body weight (6.7%), and fat (14%). Resting heart rate was significantly lower post-training (5.4%), as were the mean RQ values. These differences have implications for the control of energy substrates used during exercise.

Research Implications. The results of this study contribute information regarding the metabolic and hormonal alterations that occur in response to both aerobic exercise and decreased caloric intake in originally sedentary women. With the decrease in RQ values, as well as the reduced response of lactate, it is hypothesized that a greater

percentage of fat was being oxidized to facilitate ATP production post-training. Additionally, the significant increase in VO2 max, decrease in body weight and fat allow this program to be of practical application to women, possessing characteristics similar to those of the subjects, who are interested in losing weight, and/or increasing their aerobic capacity.

This study supports the theory that the combination of aerobic exercise and reduced caloric intake is the most successful way to reduce weight. An increase in aerobic energy expenditure for approximately 30 min 3 d/wk, combined with reduced caloric intake (1200-1400 kcal) results in a significant reduction in body fat and weight within 8 wks.

Recommendation for Further Studies. A recommendation would be to further investigate the timing of the changes in the hormonal and metabolic responses to exercise with respect to caloric intake. The response of both insulin and glucose post-training is not in agreement with previously reported observations. This may have been due to the fact that subjects were not fasted overnight. However, since under normal circumstances exercise does not usually occur after a prolonged fast, it would be of interest to know how soon one can exercise after eating and

still demonstrate hormonal and metabolic changes which suggest preferential use of fat for ATP production. Also it would be interesting to note whether such time changes are dependent upon the composition of food ingested.

APPENDIX A

METHODOLOGY

APPENDIX A
METHODOLOGY

Subjects

Prior to subject selection, permission from the University Human Subjects Committee was obtained to conduct this study (Appendix B). The 11 subjects selected to participate in the present study were among volunteers who had responded to a public advertisement. Criteria for selection of subjects required the individuals to be:

1. Females, aged 18-35.
2. Sedentary, defined as not presently participating in a regular (three times/week) endurance activity.
3. Without any orthopedic or physical contraindications that would prevent them from participating in an endurance activity.
4. Overweight (>22% body fat).

General Method

Instructional Procedures. Prior to the initial testing, all subjects were given both a written and oral explanation of the study, including its risks and benefits. All subjects signed a written consent form agreeing to the procedures of the study (Appendix C).

Each subject completed a detailed medical history questionnaire (Appendix D) and underwent screening

measurements (Appendix E) to ensure that they were at minimal risk for problems caused by exercise testing.

Subjects then participated in an orientation session to familiarize themselves with the treadmill and breathing apparatus (mouthpiece) used to collect metabolic data. Subjects were also given instructions at this time (Appendix F).

Procedures. Prior to the onset of this investigation, a pilot study was conducted by the investigator to become familiar with the procedures of the testing protocol (Appendix G). On the first test day, skinfold measurements were taken to assess subject's percent fat. A symptom-limited max GXT was administered (Appendix H). Criteria for test termination included:

1. Achievement of age-predicted maximal heart rate.
2. Oxygen consumption that reaches a plateau or declines with increasing workloads.
3. Volitional fatigue.
4. An RQ > 1.00.

Data collected from this GXT was used to determine subjects' exercise prescription (ACSM, 1980). The exercise prescription was based on the heart rate value which corresponded to 70% of subject's max VO2 (Appendix I).

On the second test day, (at least one day after the

first test) subjects again reported to the laboratory to undergo a submaximal exercise test. It consisted of approximately five stages, and was followed by an endurance phase conducted at a speed and grade which corresponded to 70% of subject's VO2 max, as calculated from previous max GXT exercise data. Criteria used to terminate the endurance GXT consisted of:

1. Volitional fatigue, or
2. Achieving 20 minutes at 70% VO2 max.

RPE was recorded every fifth minute during the endurance phase, and at one minute prior to test termination. A blood sample (5ml) was taken via venipuncture at rest, and immediately post exercise for lactate analysis. The remaining blood was then centrifuged and serum seperatedand frozed for subsequent serum glucose, insulin, and cortisol analysis (see experimental procedures).

The training began 2 days following the submaximal exercise test. Training consisted of an aerobic walk/jog program held three times per week,for 8 weeks. Each session began with a 5 min warm-up, followed by a 30-minute stimilus phase where subjects walked/ jogged at a heart rate corresponding to their exercise prescription, and ended with a 5 min cool down. Subject attendence was recorded. All exercise sessions, held outdoors, were

supervised by the investigator.

Concomitant with the training, subjects were following a reduced calorie diet. Subjects also attended weekly nutritional sessions.

At the end of the 8 week training treatment, subjects reported back to the laboratory to undergo both another max GXT, and submaximal exercise test. Protocols were identical to the pretest and workload of the endurance test remained the same as the pretest, regardless of any changes in VO2 max.

Reliability Estimate. To estimate the consistancy of the measurement technique designed to assess subject's max VO2, a correlation coefficient was computed on ventilation (VE) data from subjects max GXT with VE data from subjects endurance exercise test. The estimate of reliability (0.96) was significant ($p<0.05$).

Validity Estimate. To validate the method of utilizing percent max VO2 as the method for predicting exercise training prescriptions, a correlation coefficient was computed on heart rate values corresponding to 70% of subject's max VO2 with heart rate values predicted for subjects using the Karvonen method. The Karvonen method is considered to be the criterion method for predicting training heart rate values (Davis & Convertino, 1975). The

estimate of reliability is consistant with a previous study which reported that the Karvonen method prediction of exercise intensity was not significantly different ($p<.05$) from measured intensity at a representative workload.

Experimental Procedures

Estimate of Body Composition. Subjects reported to the laboratory where data was collected to assess their respective body density. Subjects were weighed on a physicians scale and weight was recorded to the nearest 0.5kg. The skinfold technique provided estimates of subjects' body fat. Skinfold measurements (John Bull calipers, 10mm pressure) were taken at three sites; triceps, thigh, and abdomen. The measurement to the nearest 0.1mm was recorded at each site. Measurement data was put into an equation to determine subjects respective body density (Jackson et al., 1980).

VO2 Graded Exercise Test. An approximation of a Balke type ramp protocol (Appendix H) was used to determine subjects' max VO2 pre and post treatment (Buchfucher et al., 1983). Each subject began walking at a speed of 3.4 miles per hour. After 2 min, the grade increased to 2.5% and continued to increase in increments corresponding to an overall increase of 0.5 mets/workload, until the subject voluntarily ended the test. If a subject continued past 12

minutes, the speed was also changed to allow for successive increments of approximately 0.5 mets/workload to be achieved.

During the max GXT, FeCO2 and FeO2 were measured every 30 sec using a Beckman model LB-CO2 analyzer, and a Beckman model OM-1102 analyzer, respectively. Ventilation (VE) was measured every 30 sec using the Parkinson-Cowan CD-4 dry gas meter or the digital pneumotach. Calibration of the gas analyzers was performed before and after each test, with known reference gases to correct for drift.

Heart rate was measured via the electrocardiogram (CM5) at rest (both supine and standing), every 30 sec during exercise, and every minute post exercise until the heart rate returned to baseline.

Borg's category-ratio scale of perceived exertion (Appendix J) was used to assess RPE every minute.

The max VO2 was considered to be the highest oxygen uptake observed within the last minute of the test. From the VO2 data subject's target heart rate was prescribed. It was the heart rate value correspopnding to 70% of subject's max VO2.

Submaximal Exercise Test. Pre and post training, subjects underwent a submaximal exercise test which was not to exceed 20 minutes. The exercise test was conducted at a

speed and grade which corresponded to 70% of their respective VO2 max. Each subject followed the protocol described above until she reached a level just below 70% VO2 max (determined from initial GXT). The workload was then adjusted to correspond to the subject's respective exercise prescription. This phase continued for 20 minutes, or until the subject voluntarily ended the test.

RPE was taken every fifth minute during the endurance phase, and at the minute prior to test termination. Borg's category-ratio scale was used to assess RPE.

Blood samples (5ml) were taken via venipuncture at rest, (approximately 2 min before the endurance test), and immediately post exercise. Immediately after sampling, blood samples were analyzed for determination of lactate concentration by the YSI Scientific Model 23L lactate analyzer. The lactate analyzer was calibrated prior to testing, and after every third trial. The remaining blood was then centrifuged and serum seperated and frozen for subsequent determination of serum glucose (Feteris, 1965), insulin (Hales & Randle, 1963), and cortisol (Gwazdauskus et al. 1973) (Appendix K). All samples were assayed in duplicate. Subject data regarding measurements recorded is presented in Appendix L.

Exercise Training Treatment. The exercise training

treatment was designed so that subjects would exercise outdoors walking/jogging at a pace that would elicit subject's 70% max VO2 for 30 min, 3 days/week, for 8 weeks.

At the first session, subjects were instructed how to correctly assess their heart rate by radial palpation. Subjects recorded their resting, peak exercise, and recovery heart rates on an attendance chart provided by the investigator. The recommendation was made that subjects use the first three sessions to work up to achieving 30 minutes at their respective exercise prescription. All training sessions were supervised by the investigator.

Nutritional Program. Subjects followed either a 1200 kcal or 1400 calorie diet for the duration of the program. This diet was based on the diabetic exchange diet, designed for a weight loss of approximately 1 kg/week. Weekly sessions were conducted to provide subjects with information regarding nutritional and caloric content of food, as well as behavior modification techniques. Subjects also were weighed at this time.

Research Design.

External Validity. The characteristics of the subjects; sedentary females (18-35) allow the experimental findings from this study to be generalized only to a population possessing such characteristics.

Internal Validity. Variance was minimized by; (a) familiarizing subjects with the testing equipment prior to the initial endurance test, and (b) having the post-training exercise test follow the identical protocol as the pretraining exercise test.

Issues which may confound the claim that the observable results of the experimental study can be attributed to the manipulation of the independent variable (s) via the treatment rather than uncontrolled variance are;

1. Subjects were dieting.
2. Variation occurred in the length of subjects' exercise tests.

Design Procedure. A one-group pre-test-post-test design (VanDalen, 1979) was used.

Statistical Procedures. Dependent t-tests were administered to determine whether the following variables differed significantly in response to acute exercise before and after exercise training; serum glucose, insulin, cortisol, blood lactate, heart rate, RPE, terminal RPE, body weight, body fat, and VO2 max.

A simple correlation was used to determine the relationship between terminal RPE and immediate post exercise blood lactate both pre-training and post-training.

Correlations were also used to determine the relationship between insulin and glucose; and terminal RPE and lactate in response to training.

Data Analysis. Dependent t-tests revealed the following variables to be significantly different at P <.05 (Appendix L);

1. Body weight, body fat, and VO2 max measured pre-training and post-training.

2. The response of serum glucose and blood lactate to the submaximal exercise test pre-training and post-training.

3. Resting serum glucose and cortisol pretraining and post-training.

4. The response of blood lactate pretraining to post-training.

5. The decrease in post-training R values.

6. Heart rate, measured at rest pretraining and post-training.

Conclusions. Based upon the results of this study, the researcher failed to retain the following null hypotheses;

1. There is no difference between serum insulin, cortisol, glucose, and blood lactate measured at rest and immediate post exercise, pretraining and post-training.

2. The treatment of an 8-week aerobic walk/jog program conducted at 80% of subjects' maximal oxygen consumption has no effect on subjects' body weight, body fat, and maximal oxygen consumption.

3. There is no relationship between endurance exercise terminal RPE and immediate post exercise blood lactate levels, pretraining and post-training.

Conclusions are limited to the subjects of the study, and female persons possessing similar characteristics.

APPENDIX B

REQUEST TO HUMAN SUBJECTS COMMITTEE

REQUEST TO HUMAN SUBJECTS COMMITTEE, HPER DIVISION

TITLE: Metabolic and Hormonal Indicies Contributing to Perceived Exertion Before and After a Training Program for Overweight College Women.

PURPOSE: To investigate the relationship between perceived exertion and metabolic factors before and after an 8-week training program.

PROCEDURES: Approximately 20 female participants will be selected from the Virginia Tech campus. Only those 18-35 years of age will be considered for participation. Volunteers will be prescreened to assess any contraindications to exercise such as; diabetes, orthopedic disorders, severe anemia, heart disease, hypertension, pulmonary disease. Subjects selected will be assigned to one of two groups: 1) Nutrition education plus exercise; 2) Nutrition education plus exercise, plus a partnership program incorporated as a behavior modification technique. Subjects will be measured before and after an 8-week program for the following parameters: body composition, maximum aerobic capacity, plus lactic acid and plasma hormone concentration responses to an endurance exercise test. Nutrition classes will be conduced once per week while exercise classes will be attended 3 times per week. Exercise sessions will consist of a 5 minute warm-up, 30 minute stimulus phase (70% VO_2max), and a 5 minute cool down. Body weight and dietary records will be assessed regularly during the 8 weeks of treatment.

METHODS: Physical measures--Body composition via skinfold and girth measurements

--Maximum aerobic capacity via graded continuous treadmill test to voluntary exhaustion with continuous blood pressure, electrocardiogram, and respiratory gas analysis measurements. This test will be conducted prior to the training program only.

--Endurance exercise test conducted at 70% VO_2max for 30 minutes administered once before and once after the training program

--5mls of blood will be taken via venipuncture by a certified lab technician before and after the endurance exercise tests to assess lactic acid and plasma hormone concentrations

M417

Exercise Sessions--A jogging/running program 3 times per week consisting of a 5 minute warm-up, 30 minute stimulus phase (70% VO_2max), and a 5 minute cool down.

Nutrition Education--Subjects will be instructed to modestly decrease their kcal intake to achieve a weight loss not to exceed 1-1 1/2 lbs/week. Behavior modification and basic nutritional principles will also be presented

RISK: Fatigue may be experienced due to weight loss. Exercise testing and weekly sessions may cause temporary fatigue or muscle soreness. Blood draw will cause transient discomfort due to needle stick.

PROCEDURES TO MINIMIZE RISK: Prescreening to assess contraindications to exercise such as: diabetes, orthopedic disorders, severe anemia, heart disease, hypertension, pulmonary disease. Individuals possessing any such indications will be excluded. Blood draw will be done by a certified lab technician and limited to pre and post endurance exercise tests. Exercise tests will be performed in the Exercise Physiology Lab in HPER division by experienced technicians. The exercise sessions will be supervised by qualified exercise leaders. Subjects will be terminated from the study if health disturbances arise.

RISK/BENEFIT: The physical health benefits that may result from this program outweigh the minimal risk associated with the treatments.

APPENDIX C

INFORMED CONSENT

HUMAN PERFORMANCE LABORATORY

Division of Health, Physical Education and Recreation
Virginia Polytechnic Institute and State University

INFORMED CONSENT

I, _____, do hereby voluntarily agree and consent to participate in a testing program conducted by the personnel of the Human Performance Laboratory of the Division of Health, Physical Education and Recreation of Virginia Polytechnic Institute and State University.

Title of Study: Metabolic vs. Hormonal Indices Contributing to Perceived Exertion Before and After a Training Program for Overweight College Women.

PURPOSES: To investigate the relationship between metabolic factors and perceived exertion before and after training in overweight women.

I voluntarily agree to participate in this testing program. It is my understanding that my participation will include:

Pre and Post Program Testing to include:

1. An exercise test requiring your maximum effort, conducted by qualified personnel at the Exercise Physiology Lab, before the training program.

2. An endurance exercise test at 70% VO_2max for 20 minutes, conducted by the above, before and after the training program.

3. Body composition will be analyzed by measurement of skinfolds and circumferences at three sites on your body before and after the training program.

4. Blood lactic acid and plasma hormone levels will be analyzed from a 5ml blood sample taken via venipuncture, at the Exercise Physiology Lab before and after your pre and post endurance exercise test. Two other blood samples will be taken during the endurance test by finger prick (.2ml each).

Weekly:

1. Attendance at eight weeks of exercise and nutrition education classes will be required. Nutrition classes are once per week and exercise classes are 3 times per week.

I understand that participation in this experiment may produce certain discomforts and risks. These discomforts and risks include:

> Fatigue and muscle soreness may be transiently experienced as a result of the exercise test or exercise classes. Temporary discomfort will be experienced due to needle puncture with blood drawing.

Certain personal benefits may be expected from participation in this experiment. These include:

I will receive information regarding proper nutrition and/or exercise procedures for healthy, effective weight loss. In addition I will be informed of my dietary profile, body composition, and exercise capacity at the end of the program.

I understand that many data of a personal nature will be held confidential and will be used for research purposes only. I also understand that these data may only be used when not identifiable with me.

I understand that I may abstain from participation in any part of the experiment or withdraw from the experiment should I feel the activities might be injurious to my health. The experimenter may also terminate my participation should he feel the activities might be injurious to my health.

I understand that it is my personal responsibility to advise the researchers of any preexisting medical problem that may affect my participation or of any medical problems that might arise in the course of this experiment and that no medical treatment or compensation is available if injury is suffered as a result of this research. A telephone is available which would be used to call the local hospital for emergency service.

I have read the above statements and have had the opportunity to ask questions. I understand that the researchers will, at any time, answer my inquiries concerning the procedures used in this experiment.

Scientific inquiry is indispensable to the advancement of knowledge. Your participation in this experiment provides the investigator the opportunity to conduct meaningful scientific observations designed to make significant educational contribution.

If you would like to receive the results of this investigation, please indicate this choice by marking in the appropriate space provided below. A copy will then be distributed to you as soon as the results are made available by the investigator. Thank you for making this important contribution.

____ I request a copy of the results of this study.

Date _____ Time _____a.m./p.m.

Participant Signature _____

Witness _____
HPL Personnel

Project Director Dr. Janet Walberg Telephone 961-7545

HPER Human Subjects Chairman Dr. Jon Sebolt

Telephone 961-5104

Dr. Charles Waring, Chairman, Institutional Review Board for Research Involving Human Subjects. Phone 961-5283.

APPENDIX D

MEDICAL HISTORY FORM

Basic CHD Risk Screening

PART I. SELF-ADMINISTERED INTERVIEW

Name_____ Date_____

Occupation_____

Age_____ Sex_____

Campus Address_____

Home Address_____

Work Phone No._____ Home Phone No._____

Family Physician _____ City _____

MEDICAL HISTORY

1. Indicate nature of condition* for male members of immediate family.

Personal _____

Grandfather

Maternal_____

Paternal_____

Father _____

Brother(s) _____

Uncle(s) _____

* Coronary artery disease, angina pectoris, coronary thromoosis, rheumatic fever, cardiac enlargement, valvular heart disease, arrhythmia, other.

2. Have you ever experienced any of the following (please check the circumstances in which they occur):

____ chest pain	___at rest	___exertion	___cold weather	___emotion
____ chest pressure	___at rest	___exertion	___cold weather	___emotion
____ discomfort/pain in jaw	___at rest	___exertion	___cold weather	___emotion
____ discomfort/pain in teeth	___at rest	___exertion	___cold weather	___emotion
____ discomfort/pain in throat	___at rest	___exertion	___cold weather	___emotion
____ discomfort/pain in elbow	___at rest	___exertion	___cold weather	___emotion
____ discomfort/pain in wrist	___at rest	___exertion	___cold weather	___emotion
____ palpitations/skipped beats	___at rest	___exertion	___cold weather	___emotion

3. Have you ever had an exercise or fitness evaluation? _____ If yes, please explain_____
4. Are you taking any medications on a regular basis? No_____ Yes_____
5. If yes, please list any and all medications you are taking (both prescription and non-prescription drugs.)

Name of Medication	Dosage	Doses per Day
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

EXERCISE/ACTIVITY HISTORY

1. Are you currently involved in a regular exercise program (3 days/week)? _____
2. Do you regularly walk or run one or more miles continuously? _____
   If yes, average number of miles you cover per workout or day:_____miles
   What is your average time per mile? _____ minutes:seconds
3. Please check the following sports/activities in which you have participated over the past 6 months.

Activity	Days/Week	Min/Day	Intensity		
			Light	Moderate	Vigorous
basketball	_____	_____	_____	_____	_____
bicycling	_____	_____	_____	_____	_____
calisthenics	_____	_____	_____	_____	_____
dancing	_____	_____	_____	_____	_____
chopping wood	_____	_____	_____	_____	_____
golf (without cart)	_____	_____	_____	_____	_____
handball	_____	_____	_____	_____	_____
mountain climbing/ hiking	_____	_____	_____	_____	_____
racquetball	_____	_____	_____	_____	_____
swimming	_____	_____	_____	_____	_____
tennis	_____	_____	_____	_____	_____
weight training	_____	_____	_____	_____	_____
other_____	_____	_____	_____	_____	_____
other_____	_____	_____	_____	_____	_____
other_____	_____	_____	_____	_____	_____

APPENDIX E

SCREENING MEASUREMENTS

SCREENING MEASUREMENTS

NAME_____

DATE_____

HEIGHT (CM) _____

WEIGHT (KG) _____

AGE_____

SKINFOLD MEASUREMENTS

TRICEPS _____

THIGH _____

ABDOMEN _____

PERCENT FAT _____

APPENDIX F

PRETESTING INSTRUCTIONS

PRETESTING INSTRUCTIONS

1. Subjects should come dressed in running attire.

2. Subjects should get a good nights sleep (7 hours) prior to test day.

3. Subjects should abstain from eating 2-3 hours prior to testing.

4. Subjects should abstain from any medications that could alter heart rate or pulmonary responses.

5. Subjects should abstain from drinking any beverages that contain caffeine or alcohol in the 8 hours prior to the test.

APPENDIX G

PILOT STUDY DATA

Min.	HR	V_E	VO_2	VCO_2	R
2	94	7.5	.38	.29	.76
2.3	88	7.2	.38	.30	.77
3	100	8.0	.43	.33	.76
3.3	107	8.6	.46	.36	.78
4	107	9.6	.53	.40	.76
4.3	115	10.0	.54	.43	.80
5	115	10.8	.61	.48	.79
5.3	115	12.2	.63	.51	.81
6	125	12.5	.65	.56	.85
6.3	136	13.8	.70	.61	.86
7.	136	15.6	.73	.67	.92
7.3	150	16.4	.83	.73	.88
8	150	17.6	.79	.76	.95
8.3	150	17.8	.84	.80	.95
9	166	19.0	.94	.88	.94
9.3	166	19.8	.87	.87	1.00
10.0	170	24.1	1.08	1.11	1.02
10.3	170	24.8	1.00	1.05	1.04
11	170	27.7	1.05	1.17	1.11
11.3	180	27.2	1.07	1.15	1.07
12	180	27.0	1.06	1.08	1.01
12.3		26.6	1.10	1.09	.98
13	170	26.2	1.08	1.05	.96
13.5	170	26.8	1.16	1.13	.96

APPENDIX H

GXT PROTOCOL

Min.	mph	Grade (%)	FeO_2	$FeCO_2$	V_E	HR	RPE	BP
0.00	3.4	0						
.15								
.30								
.45								
1.00								
.15								
.30								
.45								
2.00		2.5						
.15								
.30		3.5						
.45								
3.00		5.0						
.15								
.30		6.5						
.45								
4.00		7.5						
.15								
.30		9.0						
.45								
5.00		10.0						
.15								
.30		11.5						
.45								
6.00		12.5						
.15								
.30		14.0						
.45								
7.00		15.0						
.15								
.30		16.0						
.45								
8.00		17.5						
.15								
.30		19.0						
.45								
9.00		20.0						
.15								
.30		21.0						
.45								
10.00		22.0						
.15								
.30	4.0	18.0						
.45								
11.00	4.0	19.0						
.15								
.30		20.						
.45								
12.00		21.5						
.15								
.30		23.						
.45								
13.00		25.						
.15								
.30		26.						
.45								
14.00		27.0						
.15								
.30								
.45								

GXT Treadmill Protocol

NAME _____

M/E / Exp

APPENDIX I

SUBMAXIMAL EXERCISE WORKLOAD CALCULATION

Calculation of Energy Requirement for Submaximal Exercise

Horizontal Speed Component

VO_2 (ml/kg·min) = speed in m/min x 0.1 O_2 ml/kg·min per m/min + 3.5 ml/kg·min

Vertical Work Component

VO_2 (ml/kg·min) = .70 x speed in m/min x 1.8 ml/kg·min

Subject	Calculated 70% VO_2max (ml/kg·min)	Actual VO_2 (ml/kg·min)
1	28.0	35
2	20.5	23
3	20.7	21
4	24.0	30
5	17.7	20
6	14.1	17
7	20.3	19
8	26.2	29
9	23.0	25
10	21.6	23
11	27.3	24
$\bar{X}$	22.1	24.2

$\bar{X}$ difference = 9.5%

APPENDIX J

RPE SCALE

THE CATEGORY-RATIO SCALE OF PERCEIVED EXERTION

0	NOTHING AT ALL	
0.5	VERY, VERY WEAK	(JUST NOTICEABLE)
1	VERY WEAK	
2	WEAK	(LIGHT)
3	MODERATE	
4	SOMEWHAT STRONG	
5	STRONG	(HEAVY)
6		
7	VERY STRONG	
8		
9		
10	VERY, VERY STRONG	(ALMOST MAX)
•	MAXIMAL	

APPENDIX K

ASSAY PROCEDURES

O-TOLUDINE GLUCOSE ASSAY

Reagents.

Benzoic acid - 0.25%

Glucose stock standards - (Harleco)

O-Toludine reagent - 6% (u/v) (Sigma)

Preparations of Standards.

1. 25 mg/100 ml = 125 ul Glucose stock standard
   4875 ul Benzoic acid (0.2%)
2. 50 mg/100 ml = 250 ul Glucose stock standard
   4750 ul Benzoic acid (0.2%)
3. 75mg/100 ml = 375 ul Glucose stock standard
   4625 ul Benzoic acid (0.2%)
4. 100mg/100 ml = 500 ul Glucose stock standard
   4500 ul Benzoic acid (0.2%)
5. 150mg/100 ml = 750 ul Glucose stock standard
   4250 ul Benzoic acid (0.2%)
6. 200 mg/111 ml = 1000 ul Glucose stock standard
   4000 ul Benzoic acid (0.2%)
7. Blank 10.5000 ul Benzoic acid (0.2%) Note. Usually only need following standards: 25, 50, 75, 100, Blank

Methods.

1. Sample volume - 100 ul serum
2. O-Toluidine volume - 5.0 ml

3. Wave length setting - 630 nm - use red bulb and red filter

4. Reaction temperature - 100 C

5. Reaction time - 7 min

Procedure Outline.

1. Prepare standards

2. Pipet 100 ul of Blank and each standard into tubes (use duplicates for standards).

3. Pipet 100 ul into four tubes of each pool sample (use 2 pools if possible).

4. Pipet 100 ul of each sample into tubes (can do 53 tubes total on each run).

5. Add 5.0 ml of O-Toluidine reagent to each tube and cap; vortex to mix.

6. Immerse tubes in 100 C incubator (mixture must be covered with water) for exactly 7 min.

7. Remove tubes and cool immediately to room temperature by placing in an ice water bath for 3 min.

8. Transfer tube contents to spec. tubes and take reading at 630 nm (should read within 20 to 25 min. to have true color readings.

CORTICOID HORMONE EXTRACTION FROM PLASMA AND QUANTIFICATION BY CPB ASSAY

Part I: Extraction and Preparation for Assay

Reagents. Distilled - Isoctane
Methylene Chloride
Methanol

Procedure.

1. Label extraction tubes provided.
2. Add 10 ul of 3H-Cortisol (2900 dpm or 1200 cpm)
3. Allow to air dry
4. Add 1.0 ml plasma to extraction tube
5. Incubate in water bath at 45C for 10 min.
6. Add 5 ml isoctane
7. Rhythmically shake tubes for 1 min. Freeze samples - 15 min.
8. Pour off and discard isoctane
9. Repeat steps 6-8
10. Add 10 ml methylene chloride
11. Rhythmically shake tubes for one min.
12. Freeze methylene chloride and plasma in a slant position, pour off methylene chloride into clean labelled test tube.
13. Dry down methylene chloride in water bath at 45C

14. Redissolve in 2 ml methylene chloride:methanol (9:1)

15. Split the redissolved extract into labelled vials and culture tubes as follows:

0.2 ml- mini vial (recovery)

0.3 ml- 10 x 75 mm culture tube

0.6 ml- 10 x 75 mm culture tube A predetermined amount could be put in a "SAVE" culture tube at this time

16. Dry down

17. Add 5 ml recovery scintillation cocktail to recovery aliquot; store recovery vials at 4C.

18. Wash extraction tubes from step 1 - acetone rinse followed by water.

Part II: Assay 1. Prepare 1 set of standards for each of the 19 unknowns (set up in sets of 48). Cortisol standards 0-0, 0., .1, .25, .5, 1.0, 1.5, 2.0, 3.0 ng.

2. CBG Preparation

a. Add 100 ul (7.6 x 10 -6 dpm) of 3H corticosterone stock to a 125 ml Erlenmyer flask

b. Dry down

c. Add 2.5 ml dog plasma - incubate 10 min at 45C

d. Add 97.5 ml deionized H2O

e. Let sit for 10 min. before use

f. Keep on ice until finished

3. Add 1.0 ml CBG preparation to each unknown and standard tube

a. Vortex

b. Incubate 5 min at 45C

c. Incubate 10 min at 4C (ice bath)

d. Place dextran coated charcoal (DCC-Corticoid) solution and distilled water on ice

e. Put centrifuge carriers on ice

f. Set clock for 5 min.

g. Place DCC solution on magnetic stirrer

h. Start clock before adding .2 ml DCC to all tubes (0-0 tube receives .2 ml of water only)

i. Vortex after adding DCC

j. Place tubes in carriers

k. Centrifuge at 4C for 10 min at 3000 RPM (1732 g)

l. Place carriers in ice bath

m. Take off .5 ml supernatant and add 5 ml scintillation cocktail together into a mini-vial

n. Vials are now ready to be counted in the Searle Beta Scintillation Counter.

IMMUNONUCLEAR SERUM INSULIN ASSAY

Preliminary Removal of Insulin Antibody-Bound Insulin Prior to RIA for Determination of the "Free" Insulin

1. Aliquot 250 ul of each patient specimen into a 12 x 75 mm glass tube, and mix well.
2. Add 250 ul of 25% PEG to each patient sample. Mix well.
3. Centrifuge at room termerature for 20 minutes at 760 x g.
4. Pipette 200 ul samples in duplicate from the clear supernatant into 12 x 75 mm glass assay tubes. CAUTION: Do not disturb the pellet.
5. Assay according to the RIA procedure.

Assay Procedure

1. Reconstitute lyophilized reagents and allow any frozen reagents to come to 20-25C before use. Mix gently.
2. Set up labeled 12 x 75 mm tubes in duplicates. All volumes are in microliters.
3. Add reagents to the tubes as follows:
    a. Total count tubes
    100 ul of 125I insulin
    Set aside until step number 10.
    b. Nonspecific binding (NSB)

200 ul of 0 standard

c. 0 standard

200 ul of insulin standard

100 ul of guinea pig anti-insulin serum

d. Insulin standards

200 ul of insulin standard

100 ul of guinea pig anti-insulin serum

e. Quality control and unknown sera

200 ul of serum

100 ul of guinea pig anti-insulin serum

4. Add 100 ul of 125I insulin to all tubes.

5. Vortex gently without foaming and incubate at 2-8C for 16-20 hours.

6. Add 500 ul rabbit anti-guinea pig precipitating complex (RAGP-PPT) to all tubes.

7. Vortex gently without foaming and incubate for 15-25 minutes at 20-25C

8. Centrifuge at a minimum of 760 x g for 20 minutes at 20-25C

9. Decant or aspirate supernatant

10. In a gamma scintillation counter, count the precipitate of each tube and the total count tubes for 60 seconds or longer.

APPENDIX L

DATA

Table 1. Effect of 8-wk training on body weight, VO_2max, and body fat.

Subject	Age	Weight (kg)		VO_2 (ml/kg/min)		VO_2 (L/min)		Body fat (%)	
		Begin	End	Begin[1]	End[2]	Begin	End	Begin	End
1	21	56.4	53.9	41.0	41.0	2.3	2.2	29.1	25.8
2	20	59.7	56.4	32.7	32.0	1.9	1.7	30.7	29.8
3	18	71.7	63.7	29.3	32.8	2.1	2.2	34.0	29.0
4	30	65.9	62.4	37.3	41.0	2.5	2.5	28.0	25.3
5	35	118.4	112.4	20.0	22.0	2.4	2.5	42.6	40.8
6	22	69.9	66.4	32.1	38.0	2.2	2.5	27.2	26.3
7	20	71.7	66.0	36.2	43.0	2.6	2.8	33.1	30.7
8	20	63.2	61.2	38.9	40.6	2.5	2.5	29.1	26.7
9	34	84.2	78.5	24.6	34.7	2.1	2.7	35.5	31.0
10	19	79.1	69.1	29.8	38.0	2.4	2.6	33.5	32.2
11	20	65.5	60.6	34.0	38.0	2.2	2.3	32.2	29.1
$\bar{X}$	24	73.2	68.3*	30.5	36.5*	2.3	2.4	32.3	27.8*
SE	1.2	5.1	4.8	3.4	1.8	.06	.09	1.3	2.2

* Significantly different from the pretraining responses ($p<.05$)

1 expired gas system

2 inspired gas system

Table 2. The concentration of serum glucose, insulin, cortisol, and blood lactate in response to the pretraining exercise test.

Subject	Time[c] (min)	Time[d] (min)	Glucose (mg/dl)		Insulin (ng/ml)		Cortisol (ug/100 ml)		Lactate (mmoles/L)		Speed/ Grade (%)
			rest	IPE	rest	IPE	rest	IPE	rest	IPE	
1[a]	8	15	67.1	130.9			26.1	17.1	.7	6.6	5.3/5
2	17.7	24	67.1	113.0	.3	.16	4.3	6.6	1.1	5.9	5.0/2.5
3	9	18.3	79.2	121.9	.21	.11	6.9	7.0	.4	6.0	5.3/1
4	5	13	73.1	89.0	.31	.27	9.5	5.1	.4	7.3	5.2/3.5
5[b]	20	25.3			.72	.50	21.2	19.5	2.5	3.7	3.3/1
6	5	11.3	73.1	121.9	.40	.14	11.5	5.3	1.3	7.7	5.0/1.5
7	20	25.3	73.1	98.0	.45	.15	10.3	8.5	.4	2.5	5.3/1
8	6.2	11.15	61.0	92.0	.45	.61	5.4	9.9	1.2	4.3	5.4/3.5
9	6	13	88.3	136.9	.30	.12	4.0	4.9	.5	7.5	4.7/1.5
10	13	20.4	97.4	149.0	.45	.99	26.2	17.9	1.0	7.5	5.0/1.5
11	20	28.0	76.2	101.0	.40	.18	3.3	7.9	.5	5.2	5.0/3
$\bar{X}$	12.2	18.6	68.7	104.9	.36	.32	11.7	10.0	.9	5.8*	5.0 2.2
SE	3.7	5.6	7.9	12.6	.11	.10	3.5	3.0	.30	.51	.18 .30

* Significant ($p<.05$)

[a] Subjects' insulin score is not included in the means presented

[b] Subjects' glucose score is not included in the means presented

[c] Time of endurance phases at 70% VO_2max

[d] Total time on treadmill

Table 3. The concentration of serum glucose, insulin, cortisol, and blood lactate in response to the post-training exercise test.

Subject	Time[c] (min)	Time[d] (min)	Glucose (mg/dl) n=11		Insulin (ng/ml) n=11		Cortisol (ut/100 ml) n=11		Lactate (mmoles/L) n=11	
			rest	IPE	rest	IPE	Rest	IPE	rest	IPE
1[a]	8	15	108.8	182.9			12.7	8.7	.5	6.5
2	17.7	24	92.7	123.8	.34	.23	16.2	2.3	.5	5.9
3	9	18.3	98.5	111.8	.36	.16	10.6	11.1	.4	3.8
4	5	13	92.7	100.5	.23	.21	2.7	1.8	.8	5.5
5[b]	20	25.3			.75	.42	14.7	18.3	1.2	2.6
6	5	11.3	78.2	131.3	.22	.16	6.8	5.3	.7	6.0
7	20	25.3	92.7	116.3	.35	.32	7.9	6.7	.6	2.3
8	6.2	11.15	78.2	74.9	.58	.41	3.1	1.6	1.2	3.5
9	6	13	89.8	131.3	.17	.32	1.8	4.5	.6	5.2
10	13	20.4	123.2	131.7	.92	.28	9.8	15.2	.7	4.9
11	20	28	92.7	127.6	.24	.20	5.3	2.6	.3	4.2
$\bar{X}$	12.2	18.6	86.1	112.0	.42	.31	8.3	7.1	.68	4.6*
SE	3.7	5.6	9.9	14.4	.07	.04	1.4	1.7	.09	.42

* Significant ($p<.05$)

[a] Subjects' insulin score is not included in the means presented

[b] Subjects' glucose score is not included in the means presented

[c] Time of endurance phase at 70% VO_2max

[d] Total time on treadmill

Table 4. RQ, RPE, and heart rate responses to submaximal exercise.

Subject	RQ [1]		RPE[1]		RPE[2]		Resting Heart Rate ($b \cdot min^{-1}$)	
	Pre-training	Post-training	Pre-training	Post-training	Pre-training	Post-training	Pre-training	Post-Training
1	-	-	4	4	10	10	-	-
2	1.0	1.0	5	4	10	10	115	94
3	1.1	1.0	7	6	10	8	88	98[a]
4	-	-	10	9	10	9	83	68
5	-	-	8	9	10	10	75	96[b]
6	1.3	.95	10	9	10	9	100	94
7	.96	.93	1	1	4	3	79	84
8	-	-	5	4	10	10	88	83
9	1.1	.92	9	9	10	10	115	88
10	1.1	1.0	4	4	10	10	100	107
11	-	-	4	4	10	10	83	75
$\bar{X}$	1.0	.96*	6.1	5.7	9.5	9.0	93	87.6*
SE	.5	.4	1.8	1.7	2.9	2.7	4.8	3.7

* Significantly different from pretraining response ($p<.05$)
RQ= repiratory quotient
RPE = rate of perceived exertion
- = data not available
a = subject ill at time of test
b = subject had not taken medication (Lopressor 55 mg)
1 = taken at minute 5 of endurance test
2 = taken one minute prior to test termination

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