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THE UNIVERSITY OF ALBERTA

THE EFFECTS OF PAIR-FEEDING AND EXERCISE ON THE UTILIZATION AND MOBILIZATION OF ENERGY SOURCES OF TRAINED AND NON-TRAINED RATS

by

© SUSAN MICHAEL CARY

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

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DEPARTMENT OF PHYSICAL EDUCATION

EDMONTON, ALBERTA

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FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "The Effect of Pair-Feeding and Exercise Upon the Storage and Mobilization of Energy Sources of Trained and Non-Trained Rats" submitted by Susan Michael Cary in partial fulfilment of the requirements for the degree of Master of Science.

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Date 7 December 1973

ABSTRACT

Fifty-four male Wistar rats were randomly assigned to a pair-fed and an ad-libitum feeding group with half of the animals in each group trained to run on a rodent motor-driven treadmill five days per week, for one hour per day at 26.8 meters per minute for nine to thirteen weeks. Approximately half of the exercised animals were sacrificed immediately following an exhaustive run of two to two and one-half hours. All other animals were sacrificed at rest.

Analysis of results indicated that chronic moderate exercise had little effect upon organ weights, glycogen storage, and FFA concentrations. Slightly increased adrenal and greatly decreased epididymal fat pad weights were found in chronically exercised rats. Exhaustive exercise resulted in a decrease in spleen and liver weights, decreased liver glycogen values and increased plasma and tissue FFA concentrations. Pair-fed animals demonstrated significantly reduced total body and organ weights as compared to the ad-libitum fed animals. No additional dietary effects were noted.

DEDICATION

To Mary and JoAnne for their blood, sweat and tears and to those little white rats for their blood, sweat and tissues.

ACKNOWLEDGEMENTS

I wish to express a special gratitude to my committee members; Dr. A.W. Taylor for his persistence, Dr. D.C. Secord for his faith and encouragement throughout the year, Dr. E. Bucholz for his continual educational opportunities, and Professor P. Conger for her additional support.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

It is well established that carbohydrates and fats participate in providing energy for different intensities and durations of exercise. Although carbohydrates were long considered to be the major, if not the only, fuel for muscular exercise, the importance of lipids during muscular exercise has been substantiated within recent years (Cobb and Johnson, 1963; Keul et al, 1967 and Taylor, 1972).

In brief, intensive exercise, the primary energy source is carbohydrate in origin, although Pruett (1970) found that, even at extreme exercise intensities, probably ten percent of the energy is derived from lipid stores. During moderate exercise in which there is no increase in the blood lactate level, the utilization of fat as a fuel may provide 25% to 90% of the energy for the aerobically working muscle (Gollnick et al, 1970; Issekutz et al, 1970). At this exercise intensity, however, it has been shown that the fuel substrate is in a transitory state from the beginning of exercise to a steady-state condition (Astrand and Rodahl; Pruett, 1970a), utilizing glycolytic resources while transferring to lipid metabolism. The initial level of muscle glycogen has been shown to affect the duration of the intensive exercise (Hultman, 1967), while that glycogen content in turn may be altered by the carbohydrate intake

(Bergstrom et al, 1967; Saltin and Hermansen, 1967).

Leveille and Chakrabarty (1967) determined that a sedentary rat fed ad-libitum may store an excessive amount of muscle glycogen for energy. Normally, rats feed in small amounts all day in order to maintain an essentially constant availability of nutrients for absorption from the intestine. The muscles investigated were smooth muscle; none of the exercise effects were included in the study. However, with an unlimited availability of food, exercised animals are known to restrict their intake to maintain a net energy balance with their level of activity (Thomas and Miller, 1958). To date, no conclusive data exists comparing the energy sources of exercised and sedentary rats with identical food intake levels.

Statement of the Problem

The purpose of this study was to investigate the effects of pair-feeding and exercise on the energy sources, total body weight, and organ weights of trained and sedentary rats. The following parameters were measured: total body weight, organ weights, muscle and liver glycogen, blood glucose, muscle and blood lactates, plasma and fat pad free fatty acids.

Rationale Behind the Study

In examining substrates available for muscular exercise, many authors have discussed some of the relationships of diet to the

storage of energy sources (Mitchell, 1926; Mayer et al, 1954; Pernow and Saltin, 1971). The effects of training and diet have been examined by Saltin and Hermansen (1967). Diets composed of varying amounts of carbohydrates have been studied to determine the effect upon muscle glycogen and fat deposition in sedentary animals (Bergstrom et al, 1967). In addition, a technique of equalized food intake between different experimental groups has been employed in various investigations to determine differences in utilization and mobilization of energy substrates. Chesters and Quarterman (1970) researched the effects of zinc deficiency employing the pair-feeding technique of food equalization on rats. Leveille and Chakrabarty (1967) examined the absorption and utilization of glucose of sedentary rats on a pair-feeding program. However, no previous investigations have been conducted to determine the storage and mobilization of energy source in exercise and sedentary rats maintained on a pair-fed diet.

Limitations and Delimitations of the Study

1. The study was confined to the male Wistar rat between 8 and 24 weeks of age.

2. Food intake was equalized on a pair-fed basis determined by the exercised animal.

3. Only one intensity of endurance training was used. In addition, some trauma could have resulted from the electrical shocking devise on the treadmill.

CHAPTER II

REVIEW OF LITERATURE

Diet

A number of investigators have studied the basic physiological aspects of diet as it is affected by exercise (Mitchell, 1926; Mayer et al, 1954; Thomas and Miller, 1958; Bergstrom et al, 1967; Hultman, 1967; Saltin and Hermansen, 1967; Gollnick and Taylor, 1968; Parks, 1969). It is of interest that Mayer et al (1954) noticed that, within a sedentary range of activity, rats show no increase in food intake. Additionally, no decrease in food intake is seen to follow a decrease in activity level. In support of this, Thomas and Miller (1958) found a decrease in food intake, and in spontaneous overnight activity, in rats given forced exercise during the day. However, an increase in food intake was noticed on the rest days. In this way, no over-all weight drop occurred due to the increased activity. It is not known whether disruption in the normal pattern for the nocturnal rat or a stress reaction from the exercise caused the decrease in food intake. None of the present hypotheses regarding regulation of food intake can explain this phenomena (Stevenson, 1967). Parks (1969) maintains that rats will regulate their food intake according to their utilization, adjusting to a new "set" position with a change in activity. Mayer et al (1955) attributes this to the blood glucose levels.

Mitchell and Beadles (1930) developed a method by which the effects of a variable food intake were eliminated to insure a clear interpretation of the results with reference to one deliberately imposed variable, in this case, exercise. In the original experiments, Mitchell and Carmen (1926) found that, on the identical diet, two rats may gain approximately the same weight during the same time interval, yet their body compositions may vary. If the diets of the same amount are nutritionally the same, the energy storage difference may be explained by the differences in the ability to utilize the various nutrients. Exercise variations are an example of treatments causing a difference in the utilization of the nutrients (Gollnick, 1963).

In determining the effects of exercise upon the energy storage within exercised and sedentary rats, the equalization of food intake among comparable animals may eliminate some of the dietary differences due to differing food intakes. However, as the exercised animal tends to consume less food than a control animal (Gollnick and Taylor, 1968), it is inevitable that growth will be restricted in the control rat (Mitchell, 1964).

Secord et al (unpublished results) found that liver glycogen content in chronic uremic rats tended to be much greater than that of normal rats in a pair-fed study. In an attempt to determine whether this glycogen deposit was normal or if the metabolic responses of the control animals were due to starvation or malnutrition, a study was undertaken in which one group of rats

was fed ad libitum and one fed the average amount of food consumed by the chronically uremic rats (23 - 25 grams/daily). From this it was determined that the results were normal with no starvation or malnutrition occurring. In later studies, it was determined that the chronic uremia in rats disrupted the normal metabolism and storage of liver glycogen, causing a deposition of glycogen with a greater-than-normal molecular weight which was unable to be mobilized by the animal.

Total Body and Organ Weights

The functions of various organs during exercise have been of interest to many investigators (Donaldson, 1915; Barcroft, 1927; Asahina, 1959; Badeer, 1964; Tipton, 1965; Gollnick et al, 1967; Bloor, 1968; Saville and Whyte, 1969; Reitsma, 1970). As early as 1915, Donaldson of the Wistar Institute published a review of the total body and organ weight changes due to exercise. This study reports that exercise fails to produce an animal larger in bone structure. Total body weight may be greater, though body size may not be larger, as fat content is less in the exercised animal. These findings have also been supported in more recent research (Gollnick and King, 1969a; Ostman and Sjostrand, 1971).

Stevenson et al (1964) found that the animals lost weight proportionally to the amount of exercise undergone. Running at 1 km per hour, 1 hour per day, for four days per week was sufficient

to depress the appetite of the rat. Ostman and Sjostrand (1971) also noticed a weight loss with exercising animals.

Heart weights, in general, were seen to increase (Donaldson, 1932; Badeer, 1964; Tipton, 1965). Badeer (1964) stated this as being due to the increase in the length and the thickness of myocardial fibers and the number of sarcomeres. In albino rats, conflicting results have been reported for exercise effects upon the liver. Bloor et al (1968) noted an increase in liver weights with training. Donaldson (1932), on the other hand, reported a decrease in liver weight due to the utilization of fat from liver storage. Accompanying the lighter liver was a lighter spleen. Spleen weights of the rat show a significant decrease following exhaustive exercise (Barcroft, 1925; Tipton et al, 1966; Gollnick et al, 1967). This has been attributed to the expulsion of blood with splenic contractions and possibly to a decrease in pressure in the portal vein (Barcroft, 1927). In addition, splenic contractions are stimulated neurally (Tipton et al, 1966). Therefore, spleen weight changes associated with training may be credited to alterations within the autonomic nervous system.

Kidney and testicle weights increased with exercise, according to Hatai (1915). Asahina et al (1959) found similar results in the kidney, but found the testicle in rats to be very resistant to change due to exercise.

Montoye et al (1960) found no significant differences in organ weights between exercised and control animals. However, differences in training programs must be taken in to account in the interpretation of results (Holloszy, 1967).

An organ reported to undergo cyclic changes with exercise is the adrenal. In rats, the stress reaction causes a liberation of ACTH to which the adrenals respond with a loss of fat from the adrenal storage (Bloor, 1968). Upon adaptation to the stressful stimuli, liberation of ACTH is lessened, causing a return to normal adrenal weight. However, a slight change in the training program or a decrease in adaptation will initiate the recurrence of the adrenal weight loss (Selye, 1950). In contrast to this finding by Selye is that of many other investigators that adrenals from exercised animals are heavier than in sedentary rats (Gollnick et al, 1967; Bloor, 1968; Ostman and Sjostrand, 1971). Such an effect may be due to the increased cortical size as an adaptation to the stress of training (Selye, 1950).

Blood Glucose and Tissue Glycogen

Most carbohydrates in the diet form glucose or fructose once digested. In the liver, fructose is readily converted into glucose in man prior to utilization. In the rat, fructose can be metabolized by adipose tissue, though its process is slower than that of glucose. Glucose levels in the blood are regulated closely

by the liver, the extrahepatic tissues, and several hormones in an attempt to maintain energy sources of glycogen for muscular work in the liver and skeletal muscle. The concentration of blood glucose is an important determinent in the rate of glucose uptake in the liver and other tissues. Glucogenesis occurs in almost every tissue of the body, but chiefly in the liver and skeletal muscle which allow easy permeability of glucose and subsequent conversion to glucose-6-phosphate. This substance is at the junction of glucogenesis, glycogenolysis, gluconeogenesis, glycolysis and the hexase-monophosphate shunt. A complicated series of enzymatic reactions ensue from this junction depending upon the bodily requirements at the time. Reactions are normally irreversible, maintaining a balance between catabolism and anabolism, and a relatively constant blood glucose level.

In fasting animals, the blood glucose level is regulated by the hepatic glucose release and the peripheral tissue removal of glucose (Reichard et al, 1961). In man, during moderate exercise at less than 50% of the maximal oxygen uptake, blood glucose levels are reported by Hermansen et al (1970) to remain relatively constant. The increased hepatic glucose release caused by sympathetic stimulation is balanced by an increased uptake by peripheral tissues. However, Christophe and Mayer (1958) consider this balance of the blood glucose levels to be a poor indication of the carbohydrate metabolism during exercise due to the many inconsistencies of glucose permeability and the alterations in the blood flow.

During short-term maximal work, significantly increased levels of blood glucose have been reported. Hermansen et al (1970), in a study with successive fatiguing exercise sessions, noted an increase of up to 179.3 mg. per 100 mls. of blood after the final trial. Levels of up to 200 mg. per 100 mls. of blood have been found in glucose infusion studies, with a concomitant fall in muscle glycogen that is smaller than normal. Low levels of muscle glycogen may facilitate the transport of glucose across the muscle cell membranes (Hultman, 1967). These studies involved human subjects.

In prolonged exercise, blood glucose levels have not been found to be consistent. The supply of blood glucose to the central nervous system must be continuous, thereby necessitating a blood glucose level which does drop below 60 - 70 mg. per 100 mls. of blood for an extended period of time (Harper, 1971). Yet, in severe prolonged exercise, Hultman (1967) reports a quantitatively small production of glucose from the liver, which is, therefore, small in comparison to the total carbohydrate utilization. This phenomena is attributed to the decreased hepatic blood flow during prolonged, continuous severe exercise. Gollnick et al (1967) also found a decreased blood glucose level in rats after exhaustive exercise. Toward the end of exercise, an increase in the release of liver glucose is seen continuing into recovery in humans (Bergstrom and Hultman, 1967).

Pruett (1970b) reported that plasma glucose levels in humans may be dependent upon the severity and duration of the exercise.

The results of that study suggest that exercise intensities of less than 20% of the maximal oxygen uptake may not cause a drop in the blood glucose level over a 6-hour exercise session. However, exercise intensities of 50% to 70% of the maximal oxygen uptake do produce a drop. Hermansen et al (1970) also found lowered blood glucose levels following moderate-heavy exercise (50% - 70% of the maximal oxygen uptake).

Liver glycogen levels are observed to undergo diurnal variations. For eight hours after eating, liver glycogen content increases linearly. After that period, a decrease in liver glycogen is nearly linear. It is suggested by Leveille and Chakrabarty (1967) that the increase and decrease may be due to the completion of intestinal absorption of carbohydrates and to the use of glycogen as an oxidative fuel.

In both humans and rats, the resting muscle glycogen content is elevated with training (Gollnick et al, 1970; [illegible] et al, 1971). This is consistent with the hypothesis that the capacity for prolonged exercise may be limited by the initial muscle glycogen content (Hermansen et al, 1967). However, this increased level of glycogen does not seem to be necessary for the increased efficiency obtained from training, for trained muscles may adapt to pre-training levels of glycogen while maintaining the same exercise capacity of training (Proctor and Best, 1932).

At 25% to 30% of the maximal oxygen consumption, glycogen depletion would require eight to ten hours of exercise. Yet, at

75% to 85% of the maximal oxygen uptake level, depletion would occur in one to two hours (Hermansen et al, 1967). Exercise intensity does not seem to affect the degree of depletion of muscle glycogen. Gollnick et al (1967) found resting levels in man to be approximately 2.3 g/100g of wet tissue and to approach 0.3 g/100 g wet tissue at fatigue. Hermansen et al (1967) found resting values of 1.58 g/100 g wet tissue in untrained men with exhaustive values of 0.06 g/100 g wet tissue. Trained subjects had initial contents of 1.69 g/100 g wet tissue and were exhausted at a level of 0.12 g/100 g of wet tissue at exhaustion.

The disappearance of muscle glycogen follows a triphasic curve. Initially, there is a very sharp decrease, followed by a steady fall which is said to be caused by a constant use of glucose and the production of lactic acid (Bergstram and Hultman, 1967). Only at the point where glycogen content is very low does the final slow use of glycogen occur. This is due to the increase in the hepatic glucose output paralled by an increased fat utilization. Recovery from depletion to the basal level is seen within one hour post-exercise (Bergstrom and Hultman, 1966b).

Muscle glycogen concentration in man can be increased considerably by the emptying of glycogen stores through exhaustive work followed by a carbohydrate-rich diet. On a normal, mixed diet, a glycogen content of 1.75 g/100 g wet tissue was seen by Bergstrom and Hultman (1966b). An exclusively fat and protein diet, however, will result in 0.6 g/100 g caused by a slow, incomplete resynthesis of

glycogen (Bergstrom et al, 1967). A carbohydrate diet not preceded by an exhaustion of previous glycogen stores will not alter the normal concentration, but a muscle group exercised to fatigue prior to this diet may show a storage of 3.5g/100 g muscle or greater (Astrand and Rodahl, 1970). This seems to be due to the inter-conversion of the two forms of glycogen synthetase which is regulated in order to hasten glycogen synthesis when the glycogen content is low and to inhibit synthesis when the tissue concentration is elevated (Saltin and Hermansen, 1967).

Bergstrom et al (1967) found human subjects on a high carbohydrate diet after a severe exercise bout to be able to continue at a fixed load surpassing 75% of their maximal oxygen uptake for 210 minutes. 80 minutes was the maximal performance time at the same fixed intensity following a fat diet. This indicates that the initial glycogen content could be a limiting factor in the exercise capacity of an individual.

Blood Lactate

During exercise, an interplay between oxidative and glycolytic metabolism takes place. As the exercise intensity increases, so also does the importance of glycogen as an energy substrate. At rest and in low intensity exercise, the oxidation of pyruvate is maintained. With an increased utilization of glycogen, puruvic acid becomes the final hydrogen acceptor through a reduction

reaction with $NADH_2$ and lactic acid is formed (Stetten and Stetten, 1960). Dawson et al (1971) consider lactates as an index of anaerobic metabolism and possibly as a limiting factor in exercise. As blood lactate levels rise, there is a concomitant reduction of both plasma free fatty acid and glucose oxidation (Issekutz et al, 1964). However, high blood lactate levels are not always parallel to glycogen storage depletion. The two are often correlated in fatigue following short-term severe exercise, although blood glucose levels show no close relation to the levels of lactic acid in the blood (Campos et al, 1928/29; Karlsson et al, 1971).

Muscle lactates more closely describe the production of energy during exercise. Within 3 minutes of the beginning of exercise, blood lactates appear (Strom, 1949). This is due to: the formation of lactates in different tissues; the rate of diffusion from the muscles; and the rate of utilization and the excretion of lactates. Blood acts as a carrier for the lactates, transporting them to tissues which may utilize the metabolite (Wasserman et al, 1965). Griggs et al (1966), for instance, suggest that the myocardium may be capable of dehydrogenation of lactates by a non-NAD linked enzyme system. Kidneys and liver, though primary locations for gluconeogenesis, may also be capable of utilizing lactates.

At less than 50% of the maximal oxygen uptake, blood lactates show a small increase (Knuttgen, 1962). However, as exercise intensity increases, so also does the blood lactate concentration initially. In prolonged exercise, the lactate concentration

will decrease after the initial rise (Bang, 1932). The increased blood flow over the extended exercise session serves to distribute the blood lactates (Rommel and Strom, 1949). Rowell et al (1966) found that removal of blood lactate by gluconeogenesis occurred in the splanchnic area at a rate of 0.77 $\pm$ 2.5% of the total body lactate. Values of less than 70 mg. per 100 mls. of blood have been reported by Costill (1970) for exercise of 1 to 2 hours at an intensity associated with an increased lactate production. These values, however, were determined with very highly trained subjects.

In short-term exhaustive exercise, the blood lactate levels rise steeply and may approach maximal levels of approximately 200 mg. per 100 mls. of blood within 3 to 5 minutes after a fatiguing session of 3 minutes (Hermansen et al, 1970).

Evidence that the accumulation of metabolites may terminate exercise is as yet unclear. In short-term severe exercise, there is a parallel of low plasma glucose and high blood lactate levels. However, in long-distance running for instance, muscle glycogen may be depleted while blood lactate levels return to near resting levels (Costill et al, 1971). End-of-exercise glycogen stores may not be a critical factor in determining work capacity. It is possible that the initial muscle glycogen content may be more significant as the immediately available substrate for the metabolic apparatus (Hermansen, 1969). The critical blood lactate level for cessation of exercise is dependent upon many factors, such as the relative work intensity and the motivation of the subjects (Hermansen, 1971)

Free Fatty Acids

At the less severe exercise levels, muscle metabolism tends to be oxidative, relying upon lipids for the energy source (Pernow and Saltin, 1971). This has been only recently accepted in view of the relative participations of glycogen and fat as estimated by values for the respiratory quotient (CO_2 produced/O_2 consumed) (Wright, 1956). In addition, muscle tissues have recently been found to contain the necessary enzymes for the oxidation of lipids, demonstrating the ability to utilize long-chain fatty acids (Weinhouse et al, 1950; Wertheimer and Ben-Tor, 1956).

At rest, the fat content of plasma in humans is approximately 2% to 3% (Carlson, 1967). Gollnick et al (1970) reported plasma free fatty acid levels of 0.30 Eq/ml of blood for normal sedentary rats. Froberg (1971) found values ranging from 0.38 to 0.43 Eq/ml of blood for a similar sample.

The storage of fats is very efficient within the human body. Each molecule of fat contains an energy density of approximately 9 kcal/g, compared with carbohydrates 4 kcal/g, which is stored with water, reducing its efficiency as a stored fuel (Krogh and Lindard, 1920). Within the fat pad, there is a continual turnover of the triglycerides into glycerol and free fatty acids, while there are 3 classes of transport of fatty acids, all of which affect the plasma content and also the storage of fatty acids. These three classes are chylomicrons, lipoproteins, and free fatty acids (FFA).

Chylomicrons are the dietary fatty acids which are absorbed from the gastro-intestinal tract. Fatty acids transported from the liver to the other tissues are carried within the triglycerides of the lipo-proteins, the second major class. FFA are the form of fat continually being mobilized from the adipose tissue (Froberg, 1969). Issekutz et al (1966) state that it is this third class of albumin-bound fatty acids which is the important metabolic fuel during exercise.

During light exercise in the post-absorptive state, there is an initial fall and subsequent rise associated with the plasma FFA due to the facilitation of transcapillary exchange of the fatty acids. Such studies have been performed with dogs (Issekutz et al, 1964) and in humans (Havel et al, 1963).

The late rise in the plasma FFA level is probably due to the stimulation of fatty acid mobilization from the adipose tissues by catecholamines (Havel et al, 1963; Gollnick et al, 1970; and Taylor, 1972) and growth hormone (Basu, 1960), although various hormones of a nonadrenergic nature will also affect lipolysis (Gollnick et al, 1970).

After continued submaximal exercise, plasma FFA values above resting levels have been noted (Cobb and Johnson, 1963). Issekutz and Paul (1966) determined that oxidation of FFA alone could account for a four-fold increase in the energy expenditure of exercising dogs. The authors maintain that plasma FFA is a major energy source at any exercise level. However, the mobilization

of FFA differs from trained to untrained subjects, as the untrained subjects tend to incorporate more carbohydrates into metabolism at a lower exercise intensity than trained subjects (Issekutz et al, 1965a; Johnson et al, 1969).

Maximal exercise, determined as that above 70% of the maximal oxygen uptake, utilizes little fat. However, a decrease in plasma FFA concentration is seen with an increase in the presence of blood lactates (Issekutz et al, 1964). The release of the FFA is the system affected by lactates, and the presence of glucose and insulin also (Feltz, 1964; Pruett, 1970; Wertheimer et al, 1970).

Diet is an important consideration affecting the relative participation of fats and carbohydrates during exercise. The turnover of plasma FFA in fasting and fed humans differs after two hours of treadmill walking. The values obtained by Havel et al (1963) were 0.35 - 0.48 mEq/ml/minute and 0.57 - 0.64 mEq/ml/minute, respectively. Thus the availability of fat as an energy substrate may be altered by the type and amount of food intake. Leveille and Chakrabarty (1967) found that sedentary rats fed ad libitum stored a great deal of muscle glycogen as energy as opposed to animals fed once daily. Starved rats (Fabry et al, 1963) modified their storage of energy to accomodate greater lipogenesis as a reserve.

In exercised subjects, Bergstrom and Hultman (1967) determined different exercise durations resulting from varying diets. A high-carbohydrate diet provided increased muscle glycogen levels

along with an increased exercise endurance. A high-fat diet caused a decrease in both muscle glycogen values and exercise endurance. The high-fat diet does not supply increased FFA for moderate exercise and in an exercise session of 50% of the maximal oxygen consumption, blood lactates of statistical significance were recorded by Pruett (1970b) in humans.

CHAPTER III

METHODS AND PROCEDURES

Forty-six male Wistar rats (Woodlyn Farms, Guelph, Ontario) with initial body weights ranging from 160 to 250 grams at eight weeks of age were used in this study. The animals were housed in individual 7 x 10 x 7 inch suspended cages in an air-conditioned room controlled for room temperature (24 $\pm$ 1^{o} C) and a 12-hour light-dark cycle (8 a.m. to 8 p.m.).

The rats were randomly divided into two equal groups: Group I was divided into sedentary and exercised pair-fed sections, while Group II served as ad-libitum fed sedentary and exercised animals. Exercised animals were trained to run on a motor-driven treadmill (Quinton Rodent or Collins Treadmill) in daily exercise sessions that were progressively increased in speed and duration until the animals were able to run continuously for one hour at 26.8 meters per minute, five days per week, at the end of two weeks. Each exercised animal continued at this intensity for a training program of 9 - 13 weeks. Mild electrical stimulation (35 or 70mv) was used initially in training the rats to run. Paired controls received only the exercise natural for nutrition and excretion.

A unilateral excision of the right epididymal fat pad was performed under ether anaesthesia through the scrotum in five animals; three exercised and two controls. Similarly, a bilateral lipidectomy

was performed on six animals; two exercised and four controls. All animals were allowed sufficient time to recover from surgery prior to the initiation of the training regimen.

All rats received fresh water ad libitum throughout the study. Food was provided ad libitum only during the two-week training session, after which all exercise animals received crushed food pellets (Ralston Purina Co., St. Louis, Missouri) in a plaster feeder attached to each cage. A maximum of 40 grams daily set the food limit for the exercised rats. The amount of food for the control animals was then determined by the exercised animals (Mitchell and Beadles, 1932). Any food spilled by the exercised animal was weighed and used in the calculations of food eaten. This feeding schedule was maintained for the duration of the exercise program.

One-half of the exercise animals were sacrificed immediately after a fatiguing run. The other exercised animals and all control animals were sacrificed at rest. Control animals were sacrificed the day following their exercise-paired animal. Total body weights were measured on a Triple Balance Beam immediately prior to the exhaustive run in the fatigue rats and prior to sacrifice in the resting rats.

At the time of sacrifice, the animals were lightly anaesthetized with ether, the abdomen opened, and 8 - 10 mls. of blood withdrawn into heparinized syringes at the bifurcation of the abdominal aorta. The blood was centrifuged at 2000 rpm for 20 minutes and the plasma was then removed and frozen in dry ice and

alçohol. The whole liver was removed immediately and weighed; a small sample of approximately 400 mg was excised, weighed, inserted into a screw-top test tube and quickly forzen for glycogen analysis. The adrenals, spleen, heart and right kidney were removed and weighed. Testicles and epididymal fat pads, when present, were removed and weighed. The left biceps brachii and the gastrocnemius muscles were removed, weighed and frozen in dry ice and alcohol. All tissues and organ weights were measured on a Roller-Smith Precision Balance.

Glycogen Determination

The method developed by Lo, Russell, and Taylor (1970) was used for liver and muscle glycogen analysis. Frozen biceps brachii muscles, gastrocnemius muscle, and liver were hydrolyzed with 0.5 mls. of 30% potassium hydroxide containing saturated sodium sulfate (Na_2SO_4). In capped test tubes, the tissue samples were boiled for 20 - 30 minutes until dissolved, then cooled in ice.

Glycogen was then precipitated by the addition of 0.8 mls. of 95% ethanol to the digested solution. After continued cooling in ice for one-half hour or longer, the samples were centrifuged at 840 x g for 20 - 30 minutes. The supernatant was removed and the glycogen precipitate was dissolved in 10 mls of distilled water by vigorous shaking. From this glycogen solution, 1 ml., 0.5 ml. or 0.1 ml. of biceps brachii muscle, gastrocnemius muscle and liver

respectively was pipetted into a 150 x 20 mm test tube. One ml. of a 5% phenol solution was added to the test tube. Then 5.0 mls. of a 96% - 98% sulfuric acid (H_2SO_4) were added rapidly, with the acid stream directed at the surface of the liquid to insure proper mixing. After standing for ten minutes, the tubes were shaken and placed in a 25 - 30° C water bath for 10 - 20 minutes before colorimetric readings were taken.

Triplicate samples were prepared to minimize error due to contamination. Absorbance readings were taken at a wave length of 490 mu on a Beckman DU-2 spectrophotometer. Standard curves were determined daily. The glycogen content of the standard was subjected to the same phenol and sulfuric acid reactions as the muscles samples and the averages absorbance readings were plotted against the amount of glycogen used to produce standard curves.

Blood Glucose Determination

The GLUCOSTAT method (Worthington Biochemical Co., Freehold, N.J.), a prepared reagent for the quantitative, colorimetric determination of glucose, was used for the analysis of plasma glucose. After thawing, 0.1 ml. of plasma was added to 1.9 ml. water. To that mixture was added 1.0 ml. barium hydroxide ($Ba(OH)_2$), and then 1.0 ml. of zinc sulfate ($ZnSo_4$) solution. This mixture was then shaken and centrifuged at 2000 rpm for 10 minutes to form the 1:40 blood filtrate. Filtrate or standard was pipetted into labelled

test tubes to which the GLUCOSTAT reagent was added at timed intervals. After standing 10 minutes, the tubes had one drop of 4N hydrochloric acid (HCl) added to stabilize the color. The absorbancy of the solutions was read at 400 - 425 mu on a Beckman DU-2 Spectorphotometer after setting zero with a reagent blank. Measured in milligrams per 100 mls. of blood, glucose was calculated in the following manner:

$$\frac{Au}{As} \times Cs = Cu$$

where: A = Absorbance
C - Concentration of glucose

s = standard
u - unknown

Blood Lactate Determination

The Sigma Kit method was used to determine blood lactate (Sigma Chemical Co., St. Louis, Mo., 1965). Frozen plasma was thawed and 0.5 ml. transferred to a test tube containing 1 ml. of cold 8% perchloric acid (PCA) in order to precipitate the proteins. After standing for five minutes, the tubes were centrifuged at 3000 rpm for five minutes to obtain a protein-free filtrate. A reagent containing a lactic dehydrogenase (LDH) suspension, glycine-hydrozine buffer, B-diphosphopyridine nucleotide (B-DPN), and distilled water was mixed in an erlenmeyer flask.

A series of tubes were labelled, in duplicate, for the samples and the standards. To each tube was added a total of 3.0 mls. liquid. Animals sacrificed after a fatiguing run were tested with

1.0 ml. of the protein-free filtrate and 2.9 mls. of the reagent mixture. All other animals were tested with 0.2 mls. of the protein-free filtrate and 0.8 mls. of the reagent. The tubes were well mixed and allowed to stand for 45 minutes. The optical densities were read on a Beckman DU-2 Spectrophotometer at a wave length of 340 mu. The first tube of the standard curve was used to calibrate for zero optical density. The values for the unknowns were extrapolated from the standard curve to obtain blood lactate in milligrams per 100 milliliters of blood.

Tissue Lactate Method

The method utilized for tissue lactate was that of Lundholm et al (1963). Each muscle sample was homogenized in 29 ml. 6% perchlor acid (PCA) per gram for five minutes at medium speed in a Vitris Homogenizer. Then, the homogenate was spun down and 1 drop of methyl orange added to the decanted supernatant. This was neutralized with potassium carbonate (K_2CO_3) to a pH of 3.5 and cooled in ice for ten minutes.

The following reagents were brought to room temperature before use: 6% perchloric acid (PCA), 5 M potassium carbonate (K_2CO_3), glycine-hydrozine buffer, lactate dehydrogenase (LDH) and diphosphopyridine nucleotide (DPN).

Cuvettes, in triplicate, for the UNICAM SP 1800 ultraviolet Spectrophotometer were labelled control and experimental. Control cuvettes contained 1.35 [illegible] buffer, 0.15 ml. DPN, 1.50 ml. distilled.

Experimental cuvettes contained 1.35 ml. buffer , 0.15 ml. DPN, 1.40 ml. distilled water, and 0.10 ml. extract. Cuvettes were read twice (E_1) at 340 mu, three minutes apart to insure stabilization.

To each experimental cuvette was added 0.02 ml. LDH. On completion of the reaction (10 - 20 minutes), the optical density (E_2) was read twice at three minute intervals. The factor for the calculations was 158. Therefore, the mmoles of lactate per gram of tissue was calculated in the following manner: mmoles lactate/gram of tissue = $(E_2 - E_1) \times 158$.

Plasma Free Fatty Acid Determination

Plasma Free Fatty Acid content was assayed by the method of Dole and Meinertz (1960). In this assay, duplicated 1.0 ml. samples of fresh plasma were removed and added to 5.0 ml. of fat extraction mixture composed of heptane, isopropyl alcohol, and 1 N sulfuric acid (H_2SO_4) (10:40:1). The tubes were shaken and allowed to stand for about five minutes. Subsequently, 2.0 ml. of water and 3.0 mls. of heptane were added, the contents shaken, and the tubes allowed to stand for ten minutes. Three-milliter aliquots of the upper phase was taken and placed in 15 ml. conical centrifuge tubes with 1.0 ml. of Nile Blue A. Nile Blue A was used as the titration indicator in place of Thymol Blue as it gives a more reproducible end-point. The standards were made from recrystallized palmitic acid in heptane with a concentration of 0.200 ueq/ml. A blank

extract containing 1.0 ml. of water was employed in place of plasma. Nitrogen was blown through a column containing sodium hydroxide (NaOH) to eliminate carbon dioxide and delivered to the bottom of the titration tube through a capillary tube as a means of agitation and to prevent the solution from absorbing atmospheric CO_2.

Statistical Analysis

These data were analyzed using an analysis of variance technique with the two-way classification. Differences between mean scores were tested with the Scheffe's Multiple Comparison Between Ordered Means (Winer, 1971). Differences at the (.05) level were considered to be significant.

CHAPTER IV

RESULTS

Sedentary rats fed ad-libitum were significantly heavier than the sedentary pair-fed animals at the time of sacrifice. Regular endurance exercise retarded the weight gain of the trained animals ($\bar{x}$ 402.6 - 417.1 gm) so that the final total body weights of the pair-fed sedentary animals ($\bar{x}$ 391.6 gm) were similar to the ad-libitum trained animals (Fig. 1).

The weights of several specific organs (Fig. 2 to 6) (heart, kidney, testicles, gastrocnemius muscle and bicep brachii muscle) were not affected by the exercise. Mildly restricting the food intake of the pair-fed sedentary animals had no effect upon the final weights of the testicles, epididymal fat pad or gastrocnemius muscle. Pair-feeding did, however, result in decreased liver weights ($P < 0.05$). Exhaustive exercise caused a further decrease in liver weights in animals in both feeding programs (Fig. 7).

Animals fed ad-libitum demonstrated greater heart and kidney weights ($P < 0.05$) (Fig. 2 and 3) than the pair-fed group. Chronically exercised animals did not differ significantly from the sedentary animals in heart and kidney weights. The ad-libitum feeding program produced larger spleens than did the pair-feeding regimen. Exhaustive exercise resulted in significantly lower spleen

FIGURE 1

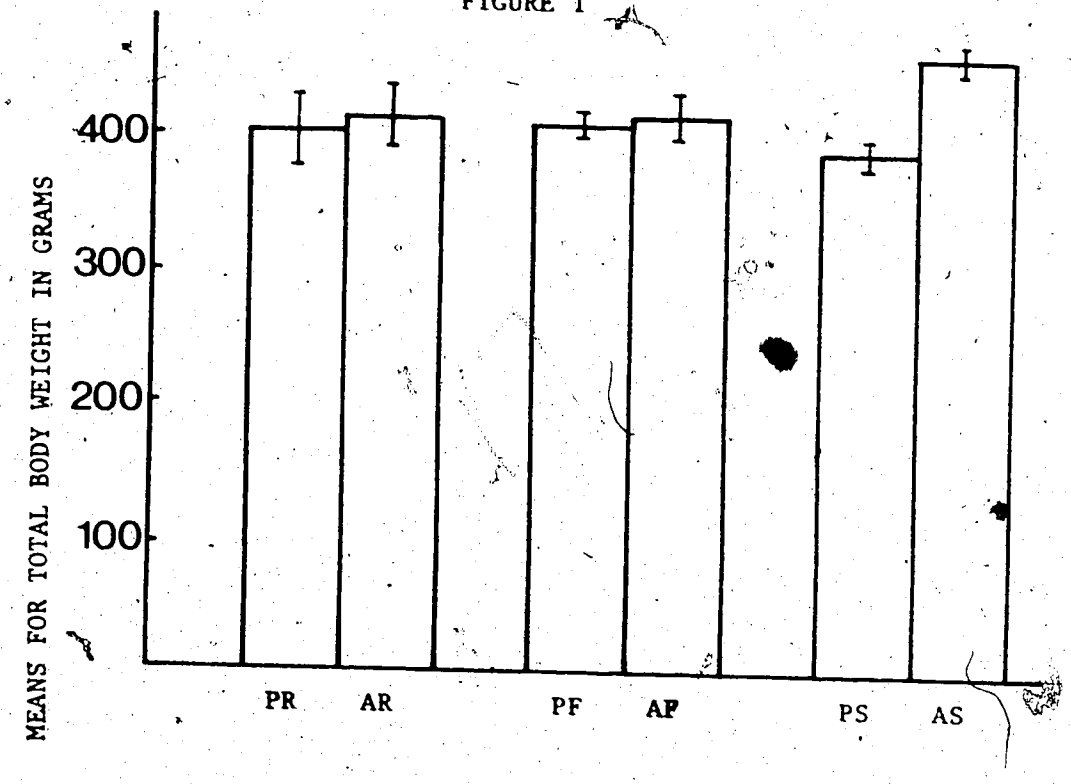
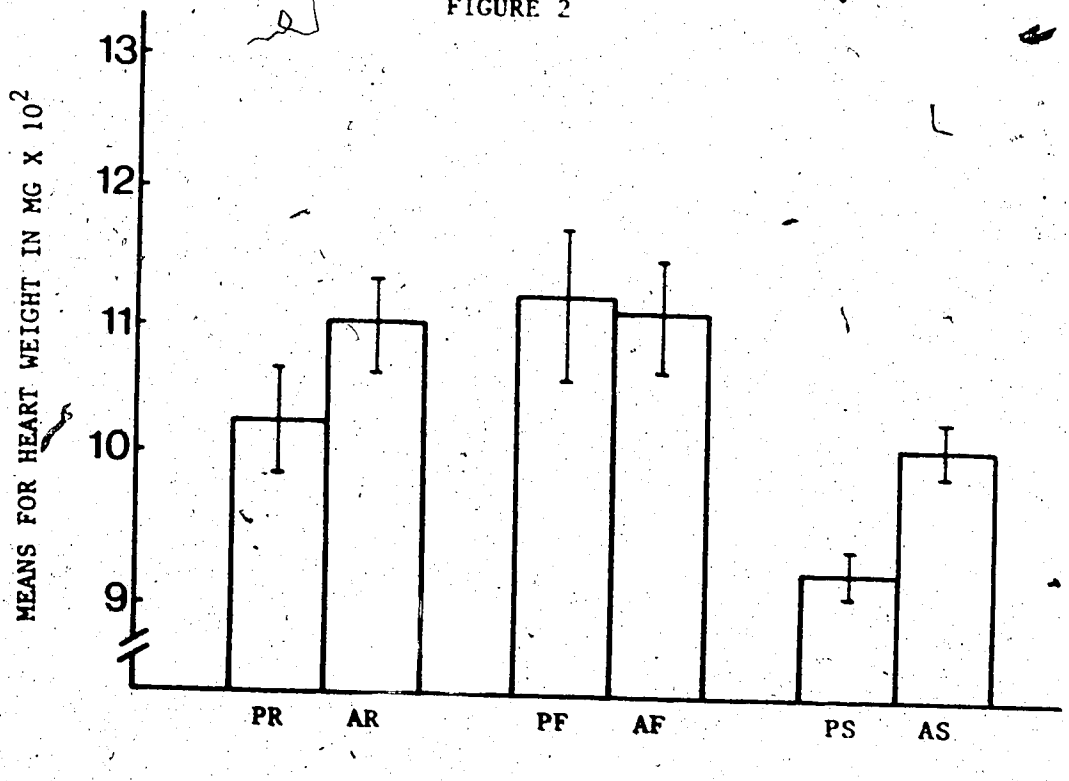


FIGURE 2



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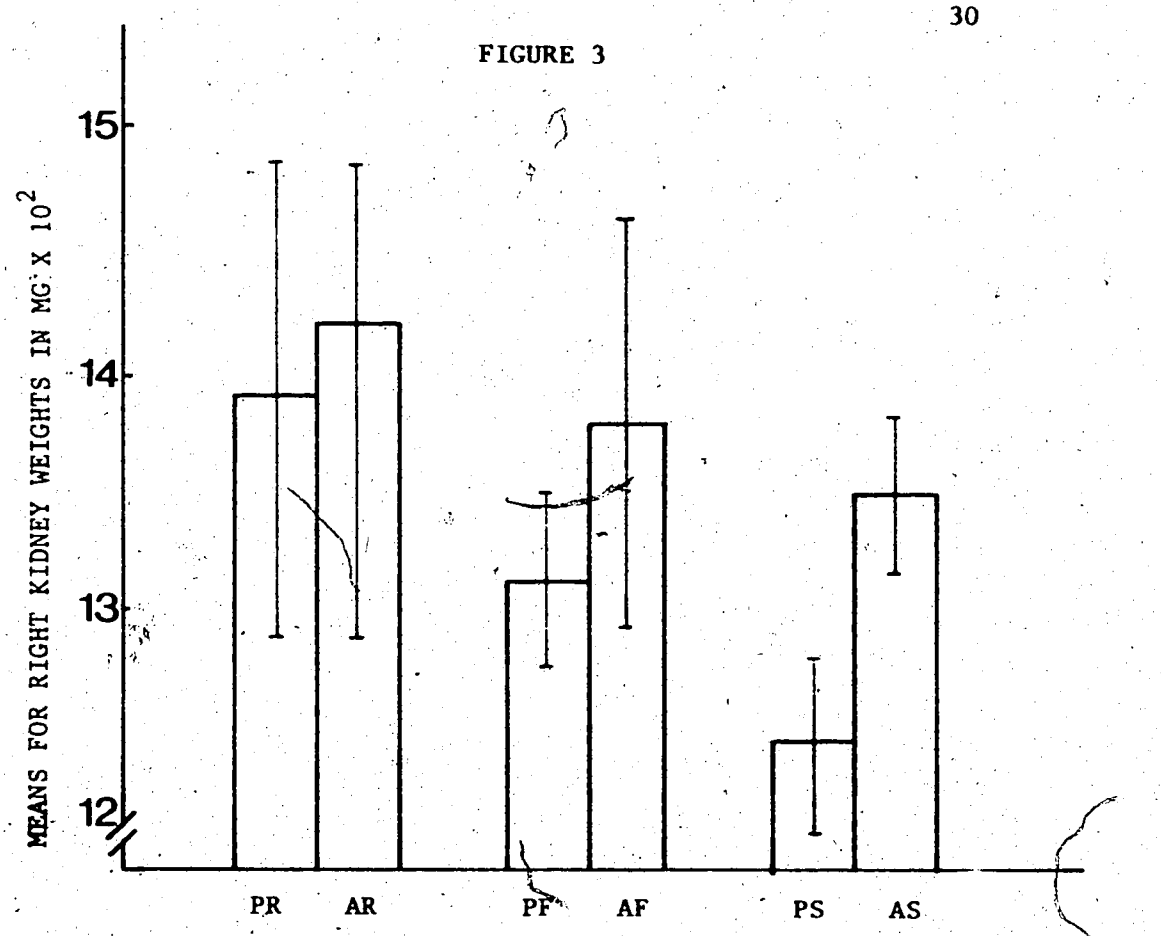

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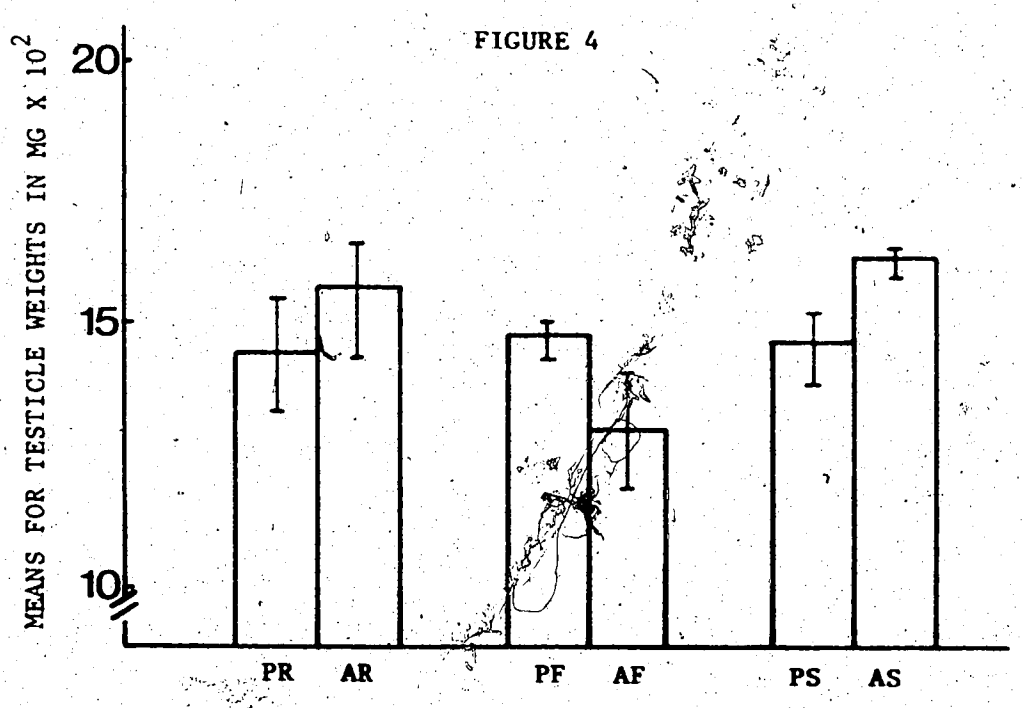

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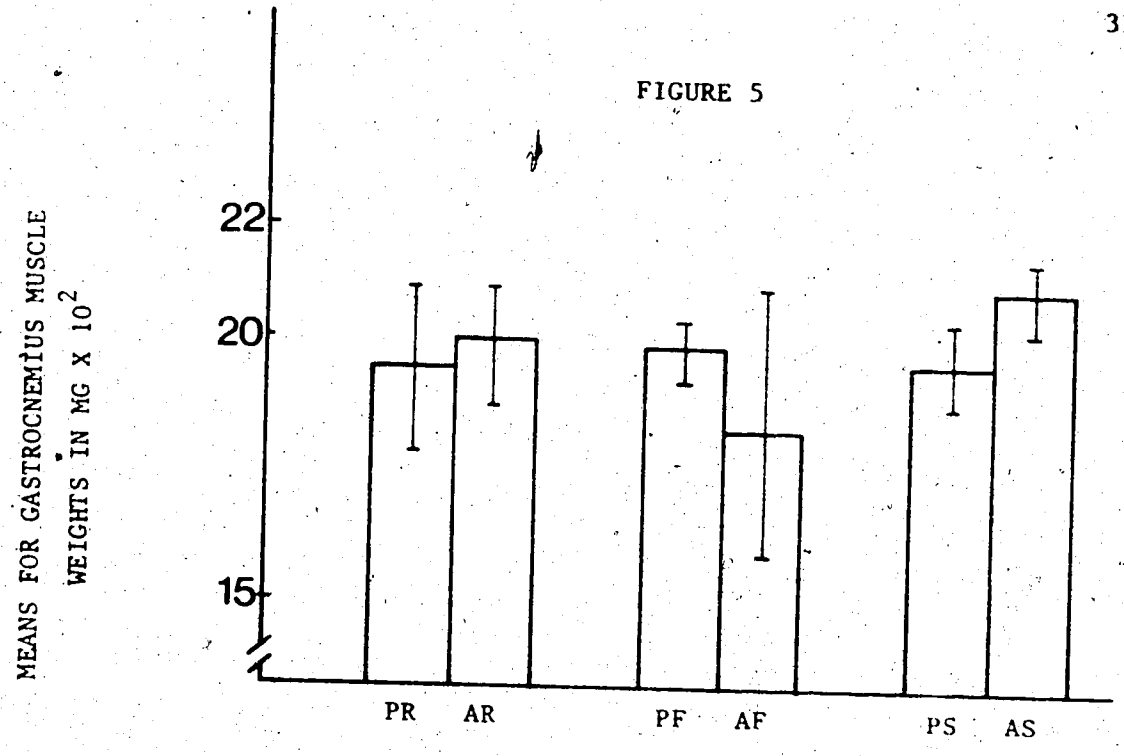

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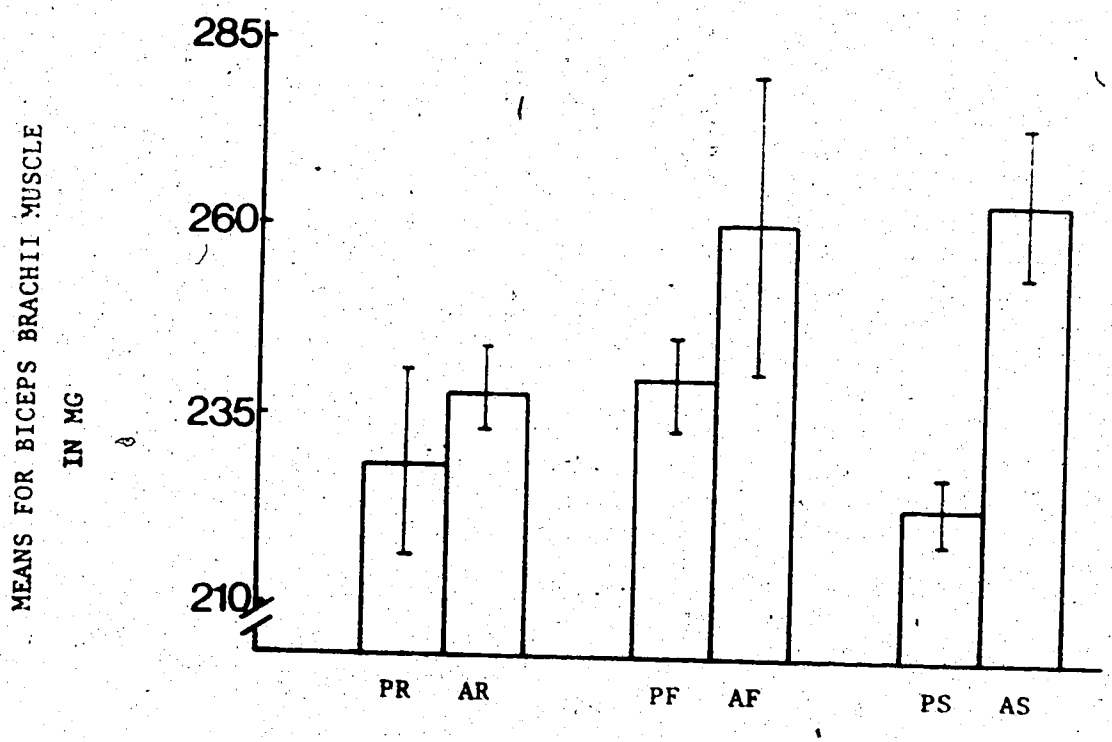

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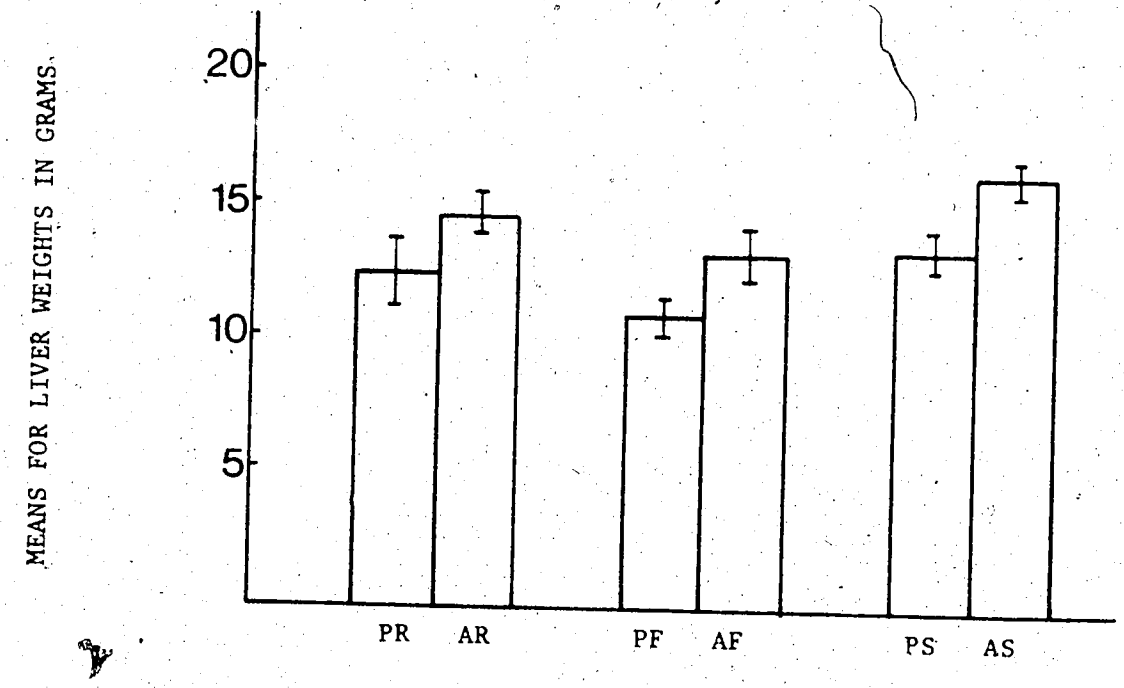
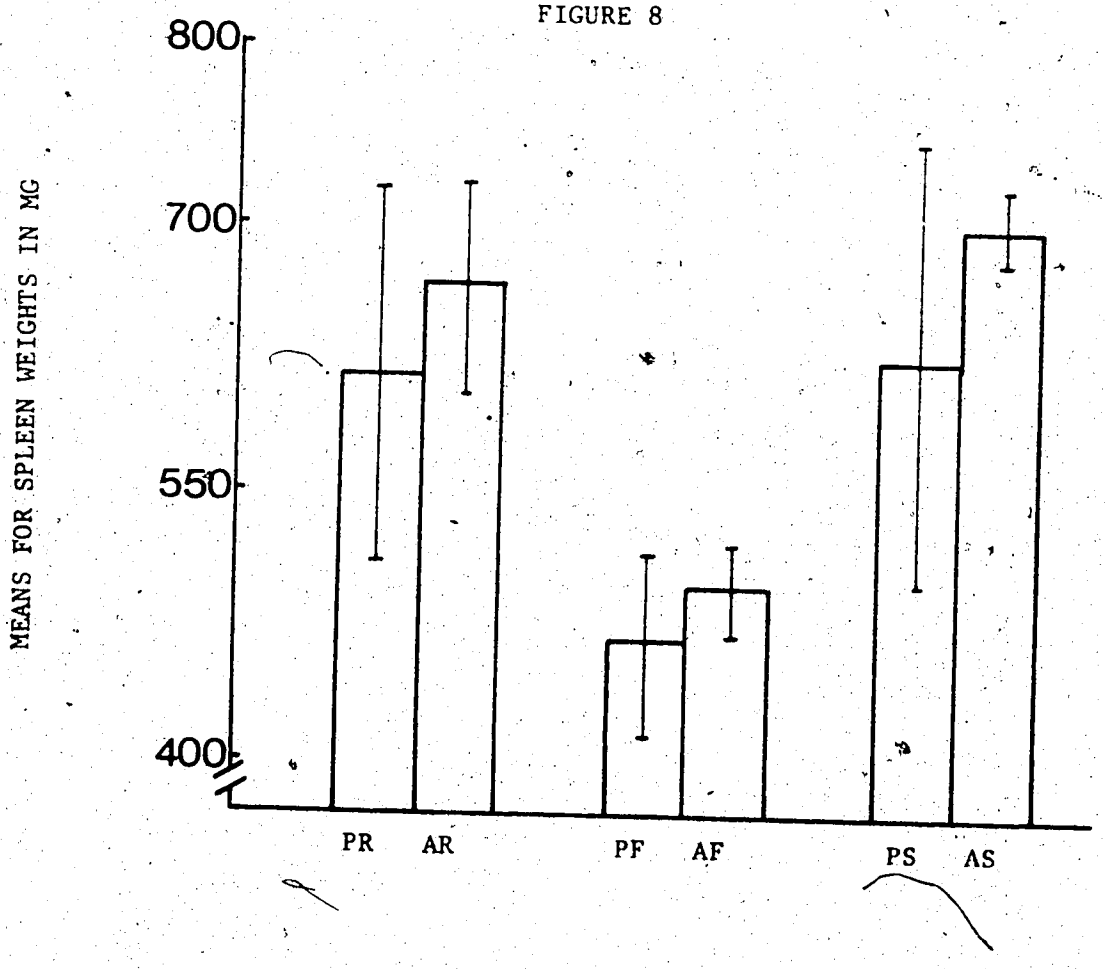


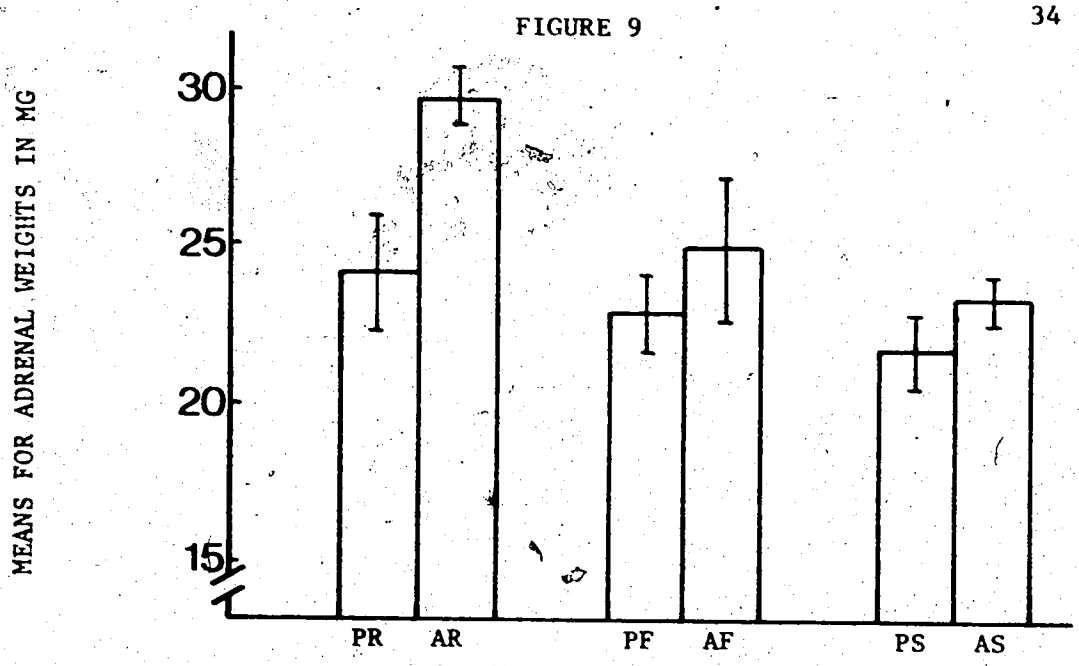
FIGURE 8



weights (Fig. 8). Ad-libitum fed animals had significantly larger adrenals than did the pair-fed rats. Animals engaged in regular endurance training had adrenals greater in size than the sedentary animals, although exhaustive exercise had no effect upon the adrenal size (Fig. 9). Neither ad-libitum nor pair-feeding programs produced any significant effect upon the epididymal fat pad weight (Fig. 10). Trained animals, however, demonstrated lighter fat pad wieghts ($P < 0.01$) than the sedentary animals. Exhaustive exercise did not affect the weight of the trained animals' fat pads. Bicep brachii muscle weights were significantly lower for the animals on the pair-feeding program (Fig. 6). Though neither chronic nor exhaustive exercise effects were significant, pair-fed animals exercised to fatigue tended to have greater bicep brachii weights than either the trained, sacrificed at rest or the sedentary animals (Fig. 10).

Pair-fed animals exhibited lower liver glycogen levels than animals fed ad-libitum, although the differences were not significant. No differences occurred between trained and sedentary animals (Fig. 11). Exhaustive exercise produced significantly lower liver glycogen levels than thos found in the trained animals sacrificed at rest. Gastrocnemius muscle glycogen levels were not affected by diet or chronic exercise, however, exercise to exhaustion tended to decrease the gastrocnemius muscle glycogen concentration (Fig. 12). The pair-fed animals demonstrated higher bicep brachii muscle glycogen levels than did the animals fed ad-libitum (Fig. 13). Training, in both diet programs, produced higher biceps brachii glycogen levels,

FIGURE 9



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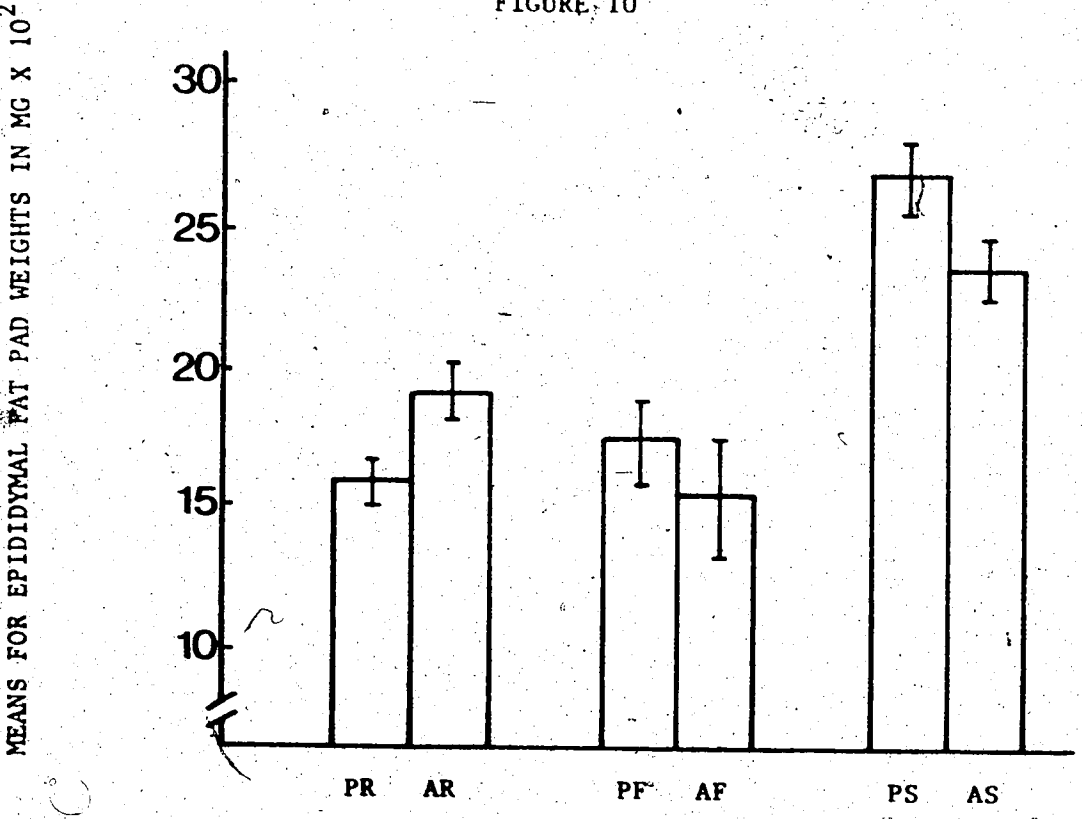

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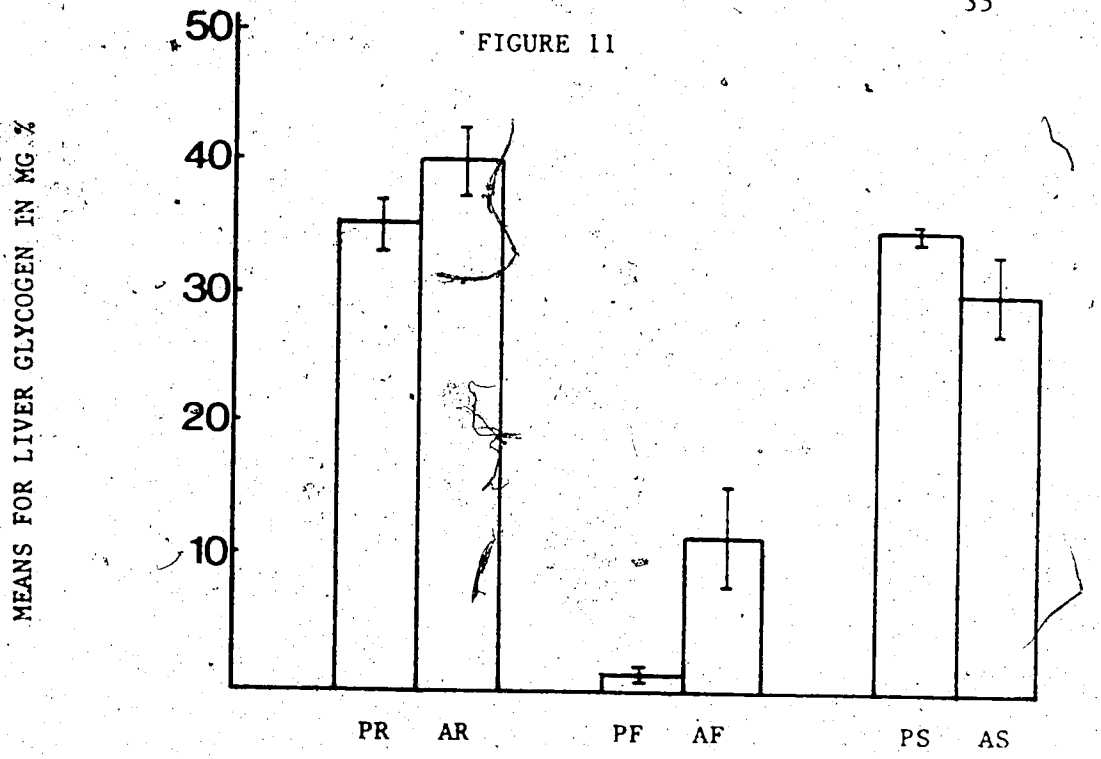

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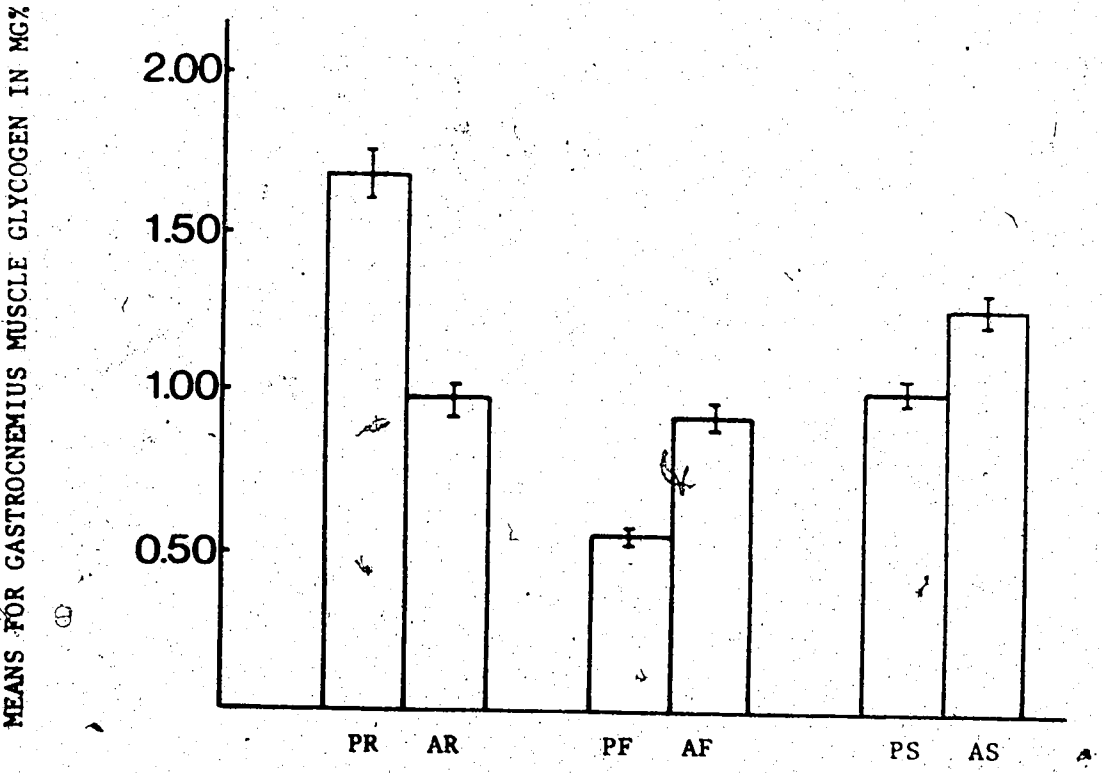

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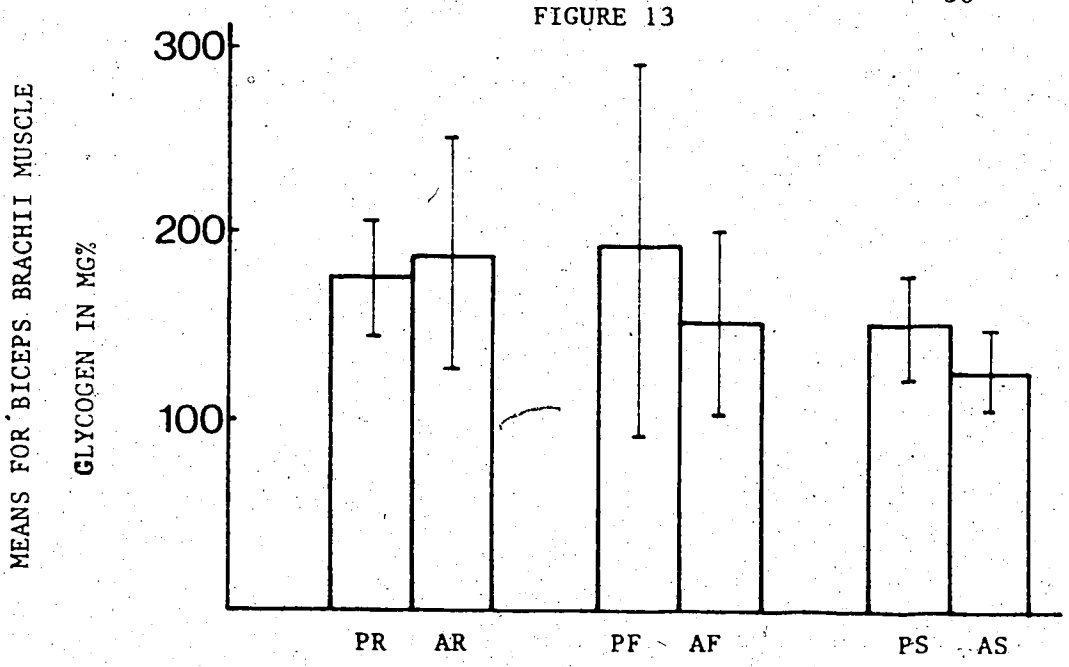
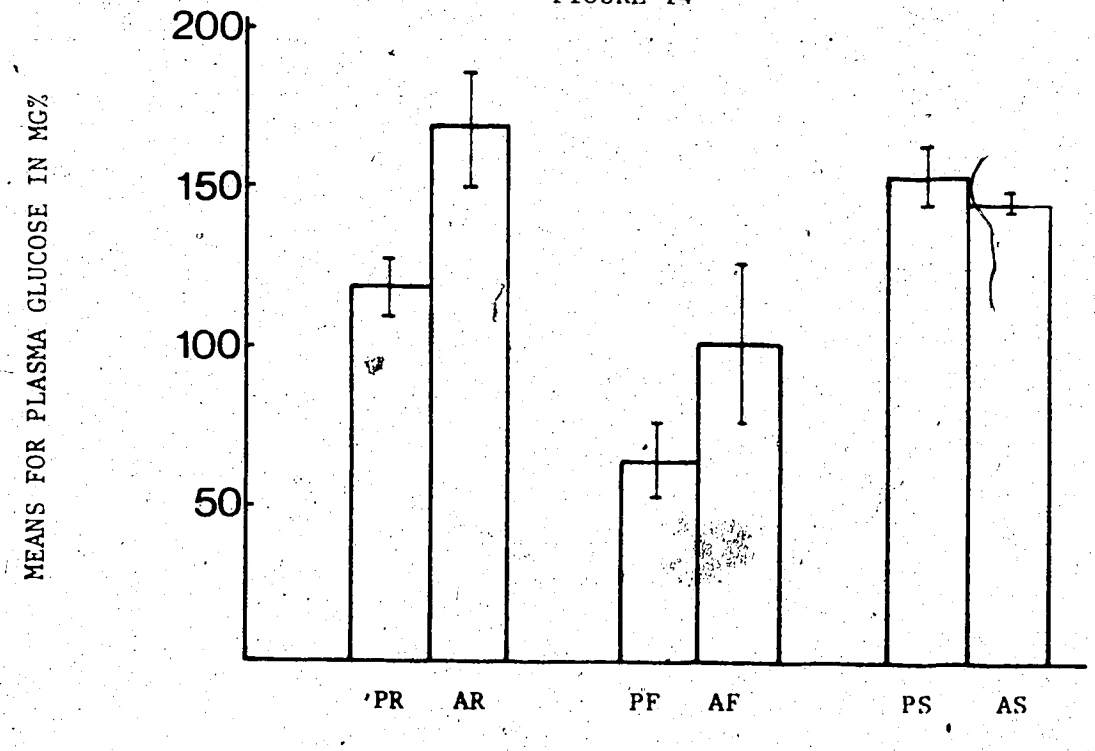


FIGURE 14



although no differences occurred as a result of exhaustive exercise. Plasma glucose levels were not affected by diet (Fig. 14). Animals sacrificed at rest were not significantly different from sedentary animals with respect to plasma glucose. However, animals sacrificed after exhaustive exercise had significantly lower plasma glucose levels than did the trained animals sacrificed at rest or the sedentary rats.

Gastrocnemius muscle lactates were not significantly altered by either diet or exercise programs (Fig. 15). Plasma lactates were not affected by the ad-libitum diet program (Fig. 16). However, the pair-feeding program did produce significantly higher plasma lactate levels for animals exercised to exhaustion with respect to sedentary or trained, sacrificed at rest animals.

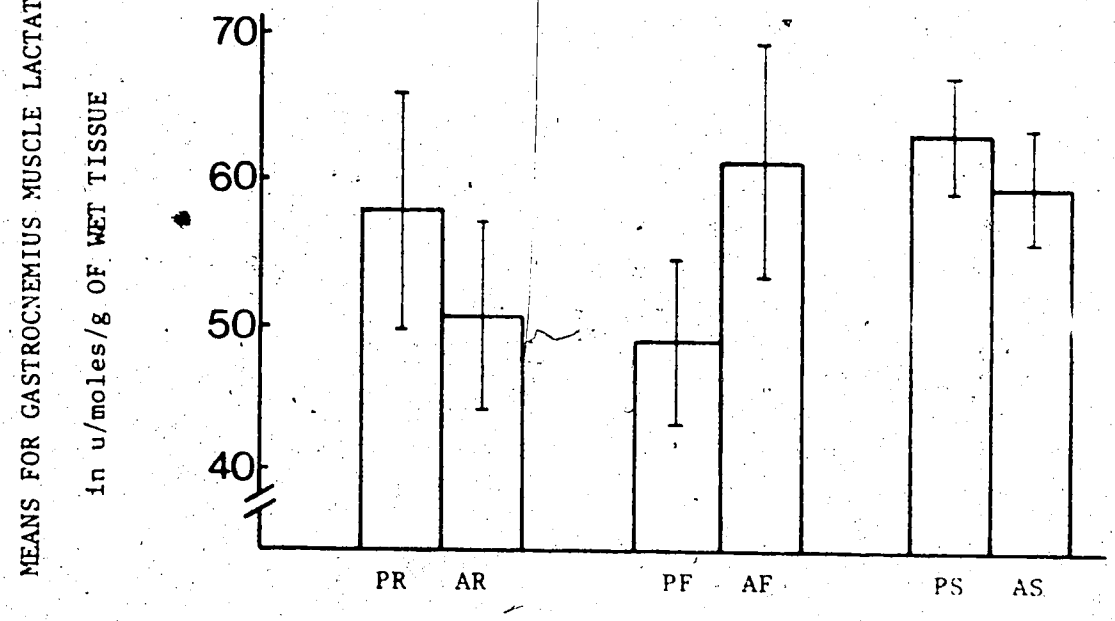
Diet did not affect the epididymal fat pad FFA levels (Fig. 17). The FFA levels in the fat pads of animals exercised to exhaustion were significantly greater than those of sedentary or trained, sacrificed at rest animals. No plasma FFA differences were noted between resting, trained animals and sedentary animals on the pair-feeding program (Fig. 18), although exhaustive exercise produced much greater plasma FFA levels than did regular training alone. The ad-libitum feeding program produced the following significant differences in plasma FFA:

a) resting trained animals had lower FFA levels than those exercised to exhaustion.

b) animals exercised to exhaustion produced higher levels than the sedentary animals.

c) trained animals sacrificed at rest had higher levels than the sedentary animals.

FIGURE 15



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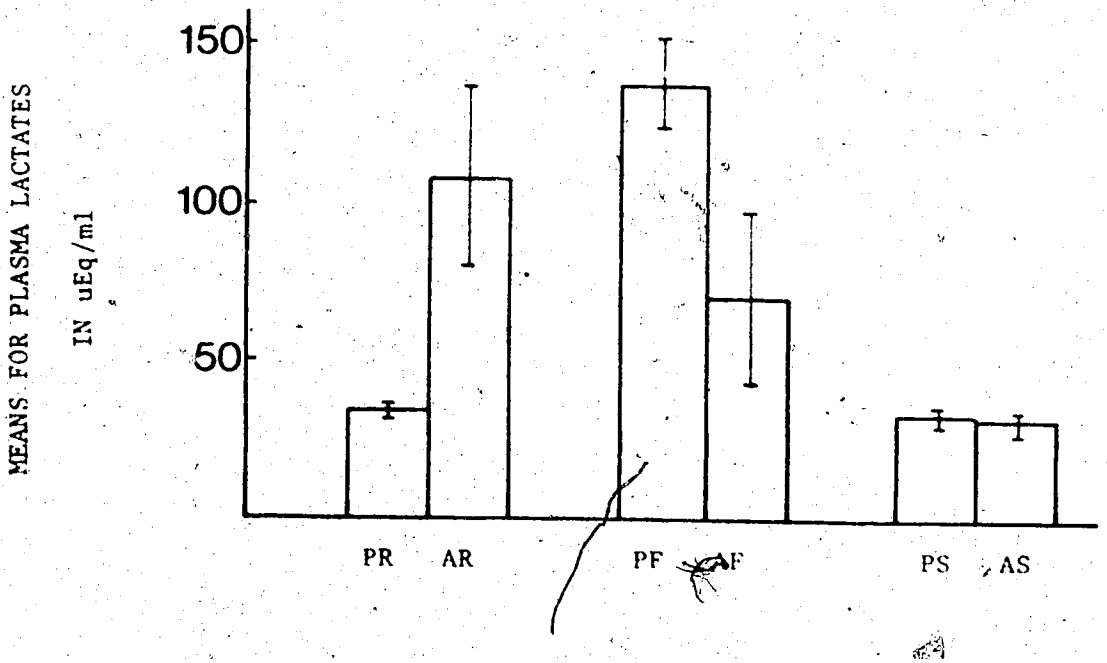

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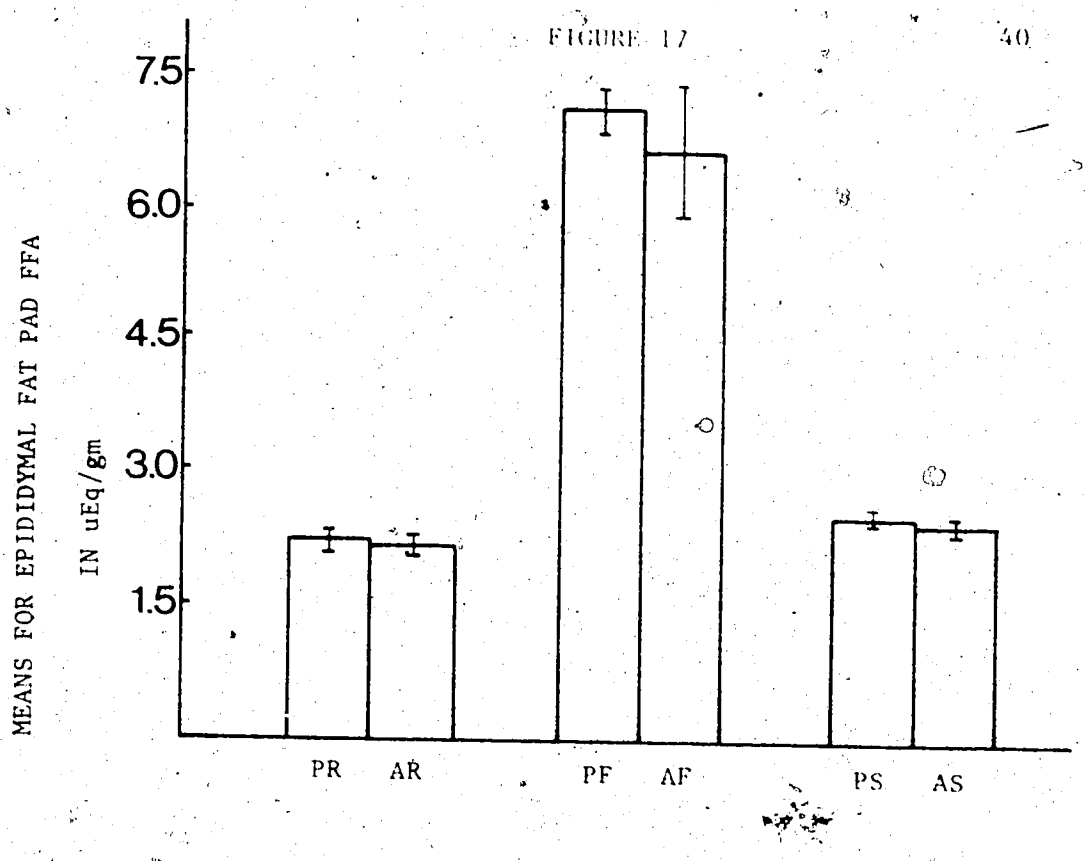

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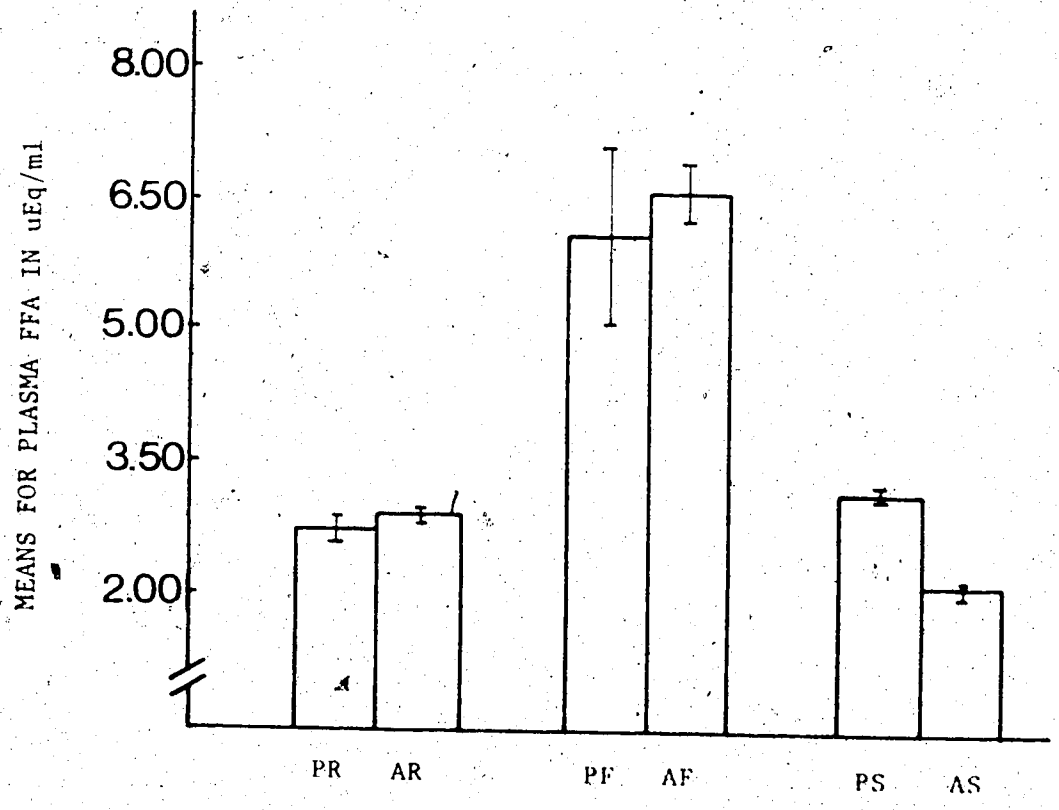

CHAPTER V

DISCUSSION

Total Body Weights and Organ Weights

Research on the alterations of total body weight resulting from different diets and exercise programs is conflicting. Gollnick (1963), Stevenson et al (1964) and Bloor et al (1968) have reported that trained animals gain less weight than sedentary control animals. However, Hatai (1915) found that exercise produced a larger, heavier rat. Stevenson et al (1964) determined that the animal lost weight proportionally to the amount of severe exercise. Running one km for one hour daily, four days per week, was sufficient to markedly depress the appetite of the animal. However, according to Thomas and Miller (1958), an increased food consumption on rest days was found to stabilize the weights of chronically exercised rats.

The results of this study concur with the findings of Gollnick (1963), Stevenson et al (1964) and Bloor et al (1968) that a decrease in the total body weights of the exercised animals occurs in comparison to the ad-libitum fed sedentary rats. The pair-fed sedentary animals were found to have weights less than those of the trained animals. Gollnick (1963), employing the group pair-feeding technique, found similar results and suggested that although the diets were theoretically isocaloric, the weight discrepancy was probably due to a difference in the available mobilizable energy

sources. The retarded weight gain in the exercised animal body weights were due to a decreased caloric intake and an increased calorie expenditure.

Mitchell and Carmen (1926), in some of the earliest studie involving food equalization among different groups of animals, maintained that animals of the different groups may gain approximately similar weights over a specified period of time, resulting in varied body compositions depending upon the energy requirements of the organisms. In this experiment, the pair-fed sedentary animals were found to weigh less than their exercised pairs. Donaldson (1932) found exercised animals to have less total body fat than the sedentary rats. Similarly, this experiment found epididymal fat pad weights to be significantly greater in sedentary rats of both feeding programs. The pair-fed sedentary rats had epididymal fat pad weights similar to those of the ad-libitum-fed sedentary rats, although their total body weights were less. The storage and composition of adipose tissue in ad-libitum sedentary animals may differ from that of pair-fed sedentary animals as a result of tissue requirements. Animals on a dietary program limiting feeding to a restricted time period daily (approximately one to two hours) are found to have an increased cell proliferation of adipose tissue (Hollifield and Parson, 1962; Tepperman and Tepperman, 1964 and Allman et al, 1965). In addition, Tuerkischer and Wertheimer (1942) have reported an increase in adipose tissue glycogen due to semi-starvation in laboratory rats. These findings may explain the proportional increase in epididymal fat pad weights of pair-fed sedentary rats in relation to the ad-libitum sedentary animals.

Liver weights may be altered as a result of diet and exercise. Donaldson (1932) reported a decrease in liver weight due to fat utilization after chronic exercise. Bloor et al (1968) conversely noted an increase in liver weight, which was interpreted as an increase in the liver glycogen content. Hatai (1915) also found an increase in liver weight due to exercise. In this study, decreased liver weights were noted for all pair-fed groups in relation to the ad-libitum fed group. There was also a decrease in the weights of heart, kidney, spleen, adrenal and bicep brachii muscle. It is possible that the alteration in feeding pattern from ad-libitum feeding during training to pair-feeding throughout the experimental period disrupted the normal feeding pattern to a permanent extent. However, Florence and Quarterman (1972) maintain that approximately two to three weeks is sufficient time for a laboratory rat to adjust to a newly-imposed feeding pattern. Also, it is possible that the random division of rats into feeding groups was unintentionally biased toward lighter rats in the pair-feeding group. In this study, liver weights were lighter in animals sacrificed after an exhaustive run than in sedentary animals and a significant decrease may be due to the acute exercise. Rowell (1971) suggested that this may be due to the decreased hepatic circulation during exhaustive exercise. As well, the decreased glycogen stores could account for a proportion of the weight loss with exhaustive exercise.

Heart weights in these experimental animals were not significantly different among the groups. Kidney and testicle

weights, as reported in the literature, were found to extremely resistant to exercise-induced change (Hatai, 1915).

Spleen weights have shown fairly consistent decreases with exhaustive exercise (Barcroft, 1925; Gollnick et al, 1967). In this study, both groups of animals sacrificed at exhaustion had significantly lower spleen weights than did the animals sacrificed at rest. The changes due to training alone seem to be unimportant. However, exercise to exhaustion does cause an expulsion of red blood cells and the spleen may remain contracted for a considerable period of time following exercise (Barcroft, 1929/30).

Adrenal weights were affected by exercise. According to the Selye stress theory, stress produces an increased ACTH secretion which in turn causes a loss of cortical lipids and a hypertrophy of cortical cells during the adaptation to training (Selye, 1950). Hearn and Wainio (1956) and Ostman and Sjostrand (1971) both found an increase in the adrenal weights of trained animals. Significant differences between the resting trained and sedentary animals were also noted in the present study, however, trained exhausted animals were intermediate to these groups and not significantly different from either.

Gastrocnemius and biceps brachii weights were not affected by training or by exhaustive exercise. The results in this study concur with those of Murray et al (1973) who found no increase in carcass muscle of exercised pigs and with Hearn and Wainio (1956) who reported no hypertrophy of gastrocnemius muscle in rats running

on a similar program. Saville et al (1969), however, found both an increase in bone density and muscle hypertrophy induced by exercise training in rats. The lack of skeletal muscle hypertrophy in the present study is undoubtedly due to the continuous type of exercise (isotonic) which does not result in muscle hypertrophy. Whereas the difference in the exercise regimen in Squille's study could explain for the increased size of the musculature.

Plasma Glucose and Tissue Glycogen

Variations in plasma glucose are due to the differences in rates of hepatic glucose release and peripheral tissue removal (Reichard et al, 1961). Difficulty occurs in determining the turnover rate of glucose as the plasma concentration remains fairly constant. However, it seems lilely that peripheral removal of glucose is the limiting factor in determination of plasma glucose levels. The increase in circulating catecholamines with exercise, according to Hermansen et al (1970), may also contribute to the increase in plasma glucose, although catecholamines are not necessary for the control of glycogenolysis during exercise (Gollnick et al, 1970). As utilization of energy sources during exercise fluctuates depending upon the severity and duration of the exercise, it is important to consider the effects of exercise upon the mobilization and interactions of the various substrates. The commencement of moderate exercise produces transient increases in plasma glucose and lactate concomittant with the inhibition of FFA mobilization. Glycolysis provides the major energy source. As moderate exercise continues, blood glucose

and lactate levels are shown to decrease (Gollnick et al, 1970; Hermansen et al, 1970 and Dawson et al, 1971) as the exercise becomes increasingly oxidative. Hermansen et al (1970) found marked changes in plasma glucose levels in humans; prolonged exercise at 50% to 70% of the maximal oxygen consumption was seen to decrease the blood glucose concentrations. Prolonged exercise of 75% to 80% of the maximal oxygen consumption produced no change in blood glucose levels in humans. Results from the present study indicated a significant decrease in plasma glucose levels following exhaustive exercise of two to two and a half hours' duration. In relation to the values of those animals sacrificed at rest, the values for animals sacrificed following exhaustive exercise are in the proper proportion. However, the raw data demonstrates individual values for animals sacrificed at rest in the range 100 to 200 mg%. It is more likely that these values are the result of incorrect measurement, as the literature is consistent in its reports of values for this condition being approximately 30 mg% regardless of the training program.

This exercise-induced hypoglycemia is caused by the increased uptake of glucose in the working muscle. Goldstein (1961) reported that an increased glucose transport system seems to exist humorally across the sarcolemma with increased energy requirements due to muscular contraction. The release of glucose from the liver is increased as exercise becomes progressively more difficult. Hultman (1967) and Hermansen et al (1970) have both suggested that

this could be due to the conversion of lactates to glucose within the liver as an adaptation to long-term exhaustive exercise.

Liver glycogen stores are in a state of constant flux due to the process of plasma glucose homeostasis (Mitchell, 1964).. Liver glycogen stores serve as the immediate source of energy to maintain necessary sources of available energy supply. Mobilization of glucose from liver glycogen is stimulated by the circulating catecholamines during exercise (Harper, 1971). Results from this experiment correlate well with the literature, indicating a decrease in the liver glycogen levels following prolonged exhaustive exercise. Animals sacrificed at exhaustion in this study demonstrated liver glycogen values approaching depletion as has been previously observed (Taylor et al, 1973).

Muscle glycogen values were not affected significantly by exercise. Recent literature with human subjects has reported that the resting muscle glycogen values may be decisive in determining the maximal exercise time (Ahlborg, 1967 and Hermansen et al, 1967). Glycogen depletion occurs in a triphasic pattern. A rapid initial utilization associated with an increased production of lactate is followed immediately by a plateauing in the utilization of glycogen. The final phase is a slow, steady fall to a very low glycogen content (Hultman, 1967). This final slow depletion of muscle glycogen may be due to a decreased blood flow to the working muscle due to the increased contractions and pressure upon the capilaries. In addition, there is an increase in lipolysis in the working muscle. In the current study, gastrocnemius muscle glycogen and biceps

brachii muscle glycogen showed no significant decrease due to exhaustive exercise. However, a tendency toward depletion was noticed for gastrocnemius muscle glycogen. Glycogen content of the bicep brachii muscle of sedentary animals tended to be less than that of the trained animals. Taylor et al (1971) have reported that chronically exercised humans incorporate more glycogen in the trained muscle tissue than do untrained humans. Costill et al (1971) stated that substantial quantities of muscle glycogen were found in highly trained runners after either prolonged or short exhaustive exercise sessions; it was suggested that glycogen depletion was an unlikely explanation for the fatigue experienced by these subjects. The differences between humans and rats may be due to: genetic differences; the metabolism of FFA by the rats during steady state exercise; and, the differences in skeletal muscle fiber types being utilized.

Plasma and Muscle Lactate

Plasma and muscle lactate levels are indicative of the extent of glycoltic metabolism. In moderate exercise, an initial period of increasing lactate followed by a net utilization is seen (Dawson et al, 1971). Increased muscle lactates precede the increase in plasma lactates due to the time lag between muscle lactate production and diffusion of lactate into the circulation. The decline of plasma lactate is prolonged moderate exercise can be interpreted as a utilization of lactate by the myocardium (Griggs et al,

1966) and the metabolism in the Cori Cycle in the liver (Hultman, 1967 and Hermansen et al, 1971). The disappearance of lactate from the blood during prolonged exercise is a function of time (Crescitelli and Taylor, 1944). The present study found no differences in plasma or muscle lactate between the sedentary or trained animals on the ad-libitum feeding program. However, the pair-feeding program produced significant differences between the trained animals sacrificed at rest and those sacrificed at exhaustion. Exhaustive exercise caused a significant increase in the plasma lactate level of pair-fed animals. Technical error may account for the unusual inconsistency in the present data. If the extremely divergent values are excluded from the analysis, the results would more closely approximate those of the literature.

Free Fatty Acids

The importance of FFA in exercise has been well-documented (Gollnick et al, 1970 and Pernow and Saltin, 1971). Changes in the plasma FFA levels indicate only an imbalance between FFA mobilization from adipose tissue and the removal by the working muscle (Issekutz et al, 1966). Plasma FFA levels seem to be controled by the release rather than the uptake of FFA (Issekutz, 1966). Lipolysis is regulated in part by tonic control by the nervous system; in all likelihood the norepinephrine is released from the nerve endings (Engel et al, 1960 and Taylor, 1972). Taylor (1972) also supported the finding that catecholamines are rapid mobilizers of FFA. As

lactate levels rise, there is a concomitant reduction of plasma FFA turnover (Issekutz, 1964). Initially in exercise, a decrease in the plasma FFA concentration is often reported. This results from an increased blood flow to the working muscle and the consequent removal of plasma FFA; therefore, a decrease in mobilization is not necessarily the cause of the initial lag in plasma FFA concentration (Carlson and Pernow, 1961). Pruett (1970b) reported that exercise intensities of up to 70% - 80% of the maximal oxygen uptake activate agents increasing the plasma FFA level, thus increasing the substrate availability as exercise. Increases in exercise intensity result in a larger proportion of the energy derived from the glycolytic pathway, thereby inhibiting the mobilization of FFA. However, even at maximal intensities, energy provided by lipolysis may still produce 10% of the metabolic demand (Pruett, 1970a).

Taylor et al (1971) determined that FFA oxidation is the primary energy source for exercise intensities up to 50% of the subject's maximal oxygen uptake. Gollnick et al (1970) suggest that FFA is utilized as a primary energy source between 25% and 90% of the maximal oxygen uptake. Engel and White (1960) found evidence that, under certain conditions, excessive or persistent accumulation of FFA within the adipose tissue may eventually lead to an inhibition of glucose uptake, perhaps by altering the pH or the physical properties of the cytoplasm. The results of this study concur with the literature (Issekutz, 1964 and Taylor, 1972) that the plasma and fat pad FFA levels were significantly elevated by exhaustive exercise.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Fifty-four male Wistar rats were used to determine the effects of pair-feeding upon the storage and mobilization of energy sources of trained and sedentary rats.

The animals were randomly divided into two equal groups: Group I was divided into exercised and sedentary pair-fed groups while Group II served as ad-libitum fed, exercised and sedentary animals. Exercised animals were trained to run on a motor-driven treadmill by progressively increasing the speed and duration of the daily exercise session until they were capable of running continuously at 26.8 meters per minute, for one hour, five days per week. Each exercised animal continued to run at this intensity and duration for an additional nine to thirteen weeks. Approximately one-half of the exercised animals were sacrificed immediately after an exhaustive run of two to two and half hours' duration. All other animals were sacrificed at rest.

Analysis of results indicated that chronic moderate exercise had little effect upon organ weights, glycogen storage, and FFA concentrations. Slightly increased adrenal weights and greatly decreased epididymal fat pad weights were found in the chronically exercised rats. Exhaustive exercise resulted in a decrease in spleen and liver weights, decreased liver glycogen levels, and increased plasma and tissue FFA concentrations.

Pair-fed animals demonstrated significantly reduced total body and organ weights as compared to the ad-libitum fed animals. No additional dietary effects were noted.

Conclusions

Within the limitations of this study, it can be concluded that pair-feeding of exercised and sedentary rats does not significantly alter the storage and mobilization of energy sources.

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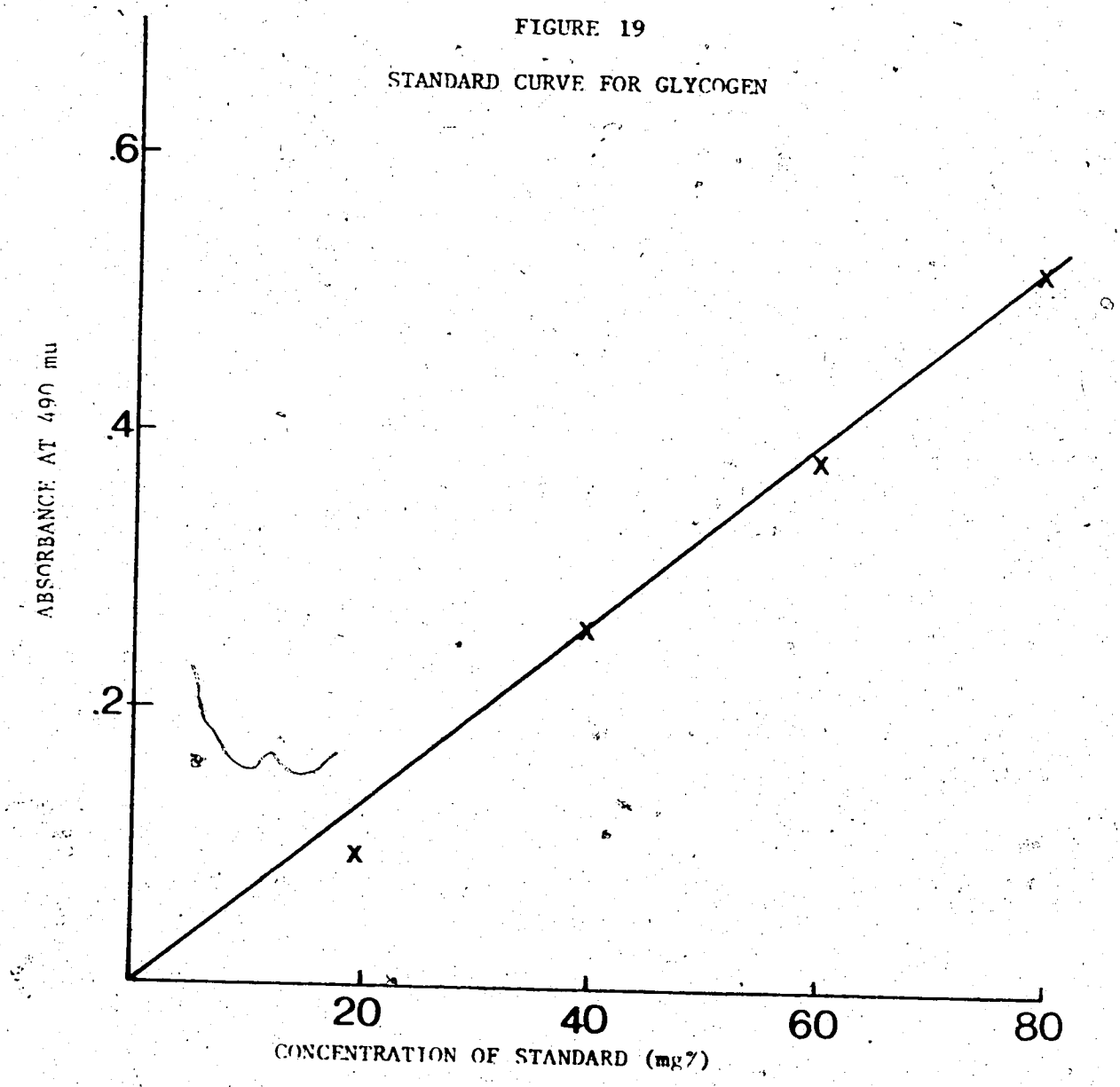
APPENDIX A

STANDARD CURVES

FIGURE 19

STANDARD CURVE FOR GLYCOGEN



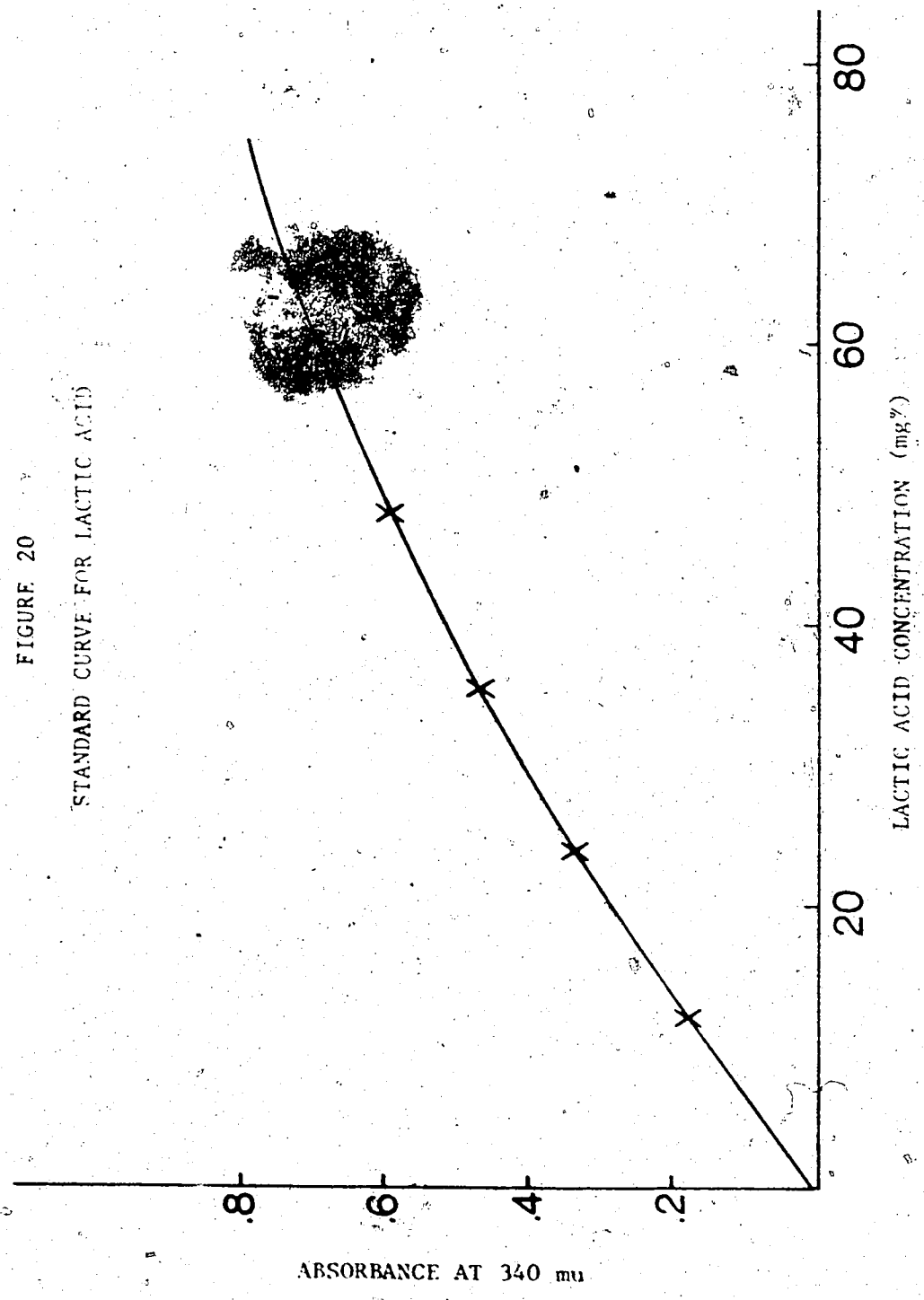


FIGURE 20

STANDARD CURVE FOR LACTIC ACID

FIGURE 21

STANDARD CURVE FOR FREE FATTY ACIDS

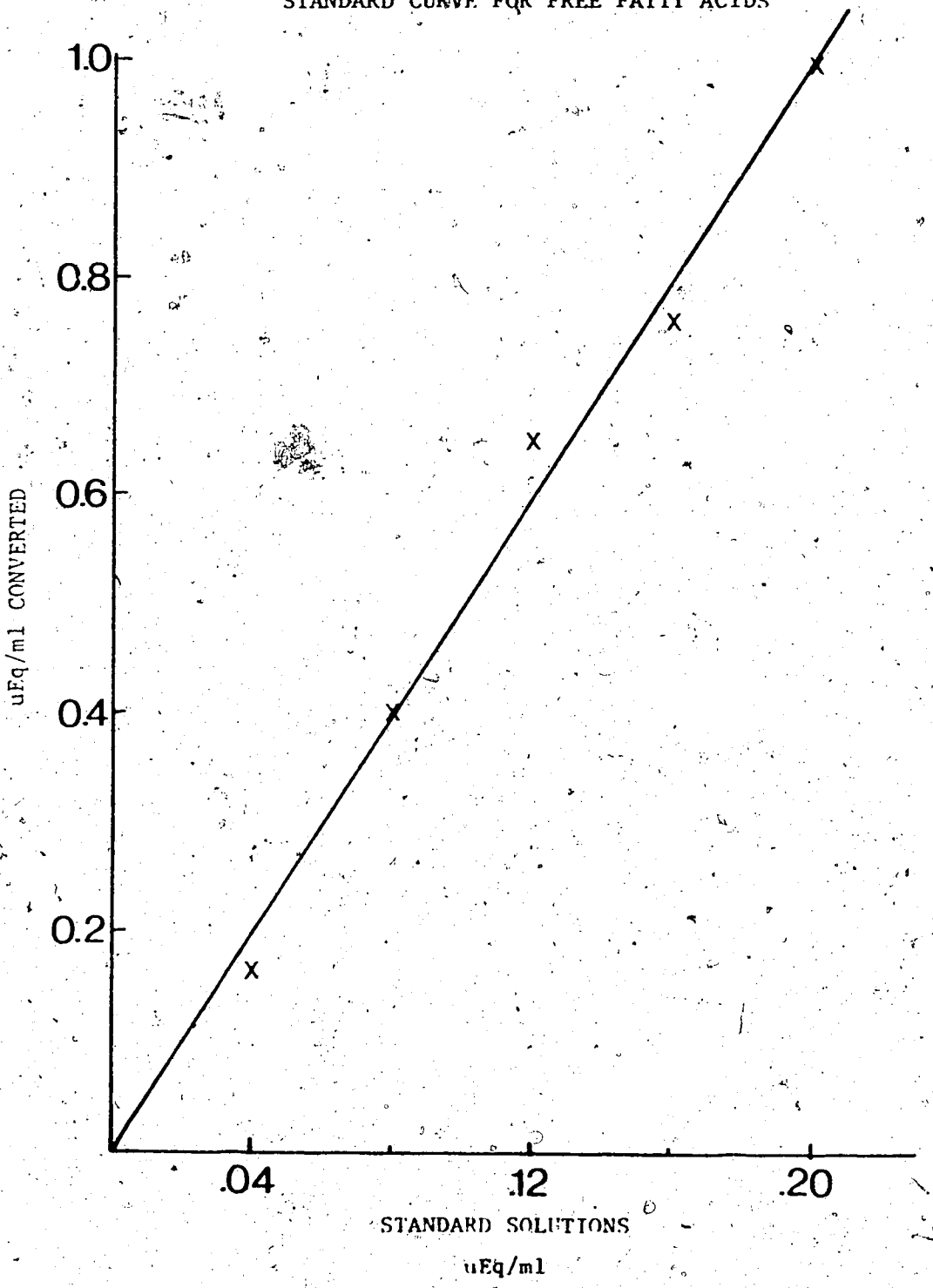
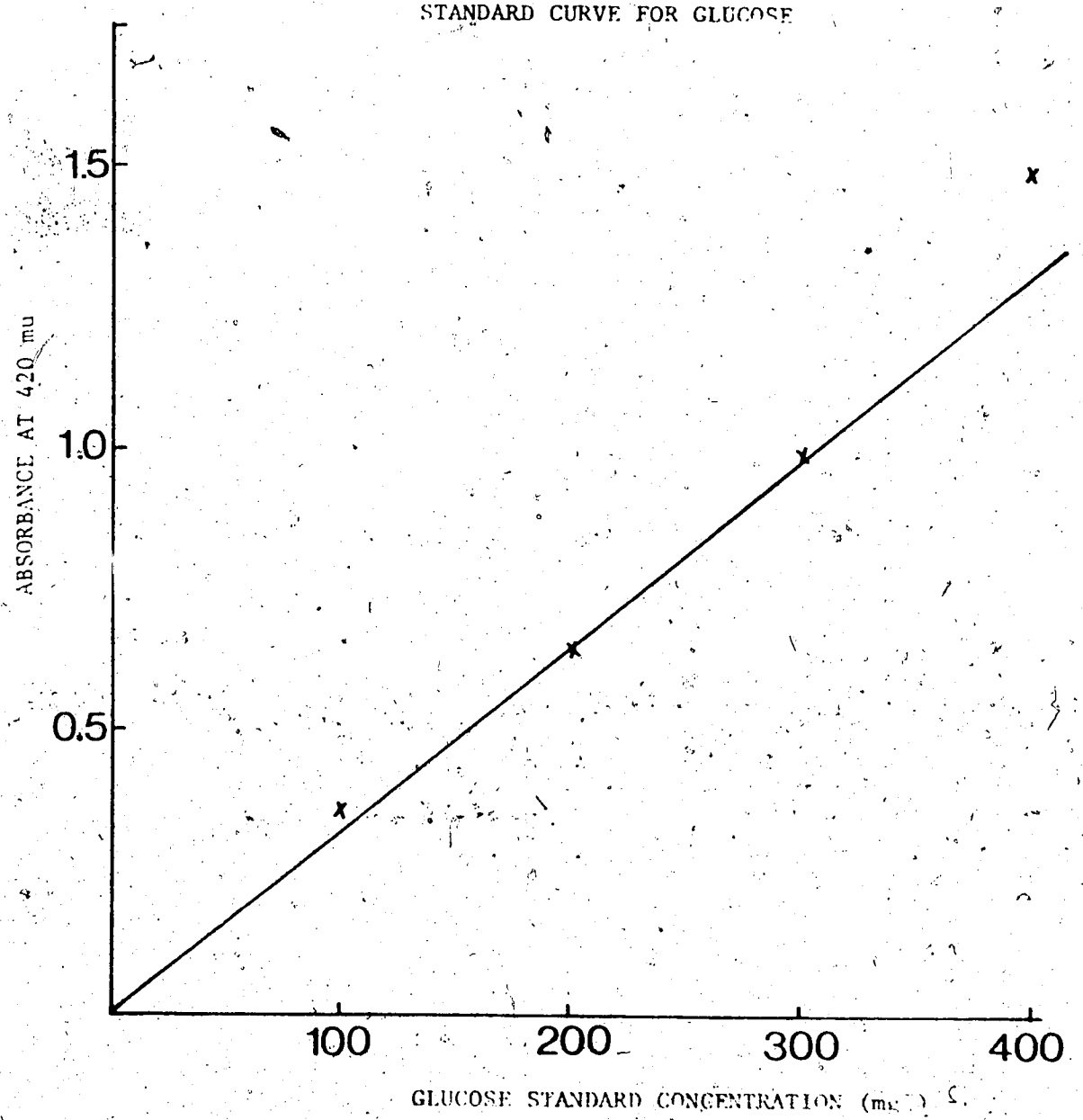


FIGURE 22

STANDARD CURVE FOR GLUCOSE



APPENDIX B

TABLE OF MEANS

TABLE 1

GROWTH CHART

Group	Weight (g)				
	Initial	Week 5	9	13	Final
Pair-fed, exercised	192.7	308.1	360.1	394.9	405.6
Pair-fed, sedentary	194.6	314.5	343.9	365.6	392.0
Ad-libitum, exercised	208.6	300.9	381.5	428.9	416.2
Ad-libitum, sedentary	206.0	311.5	414.1	448.7	471.5

TABLE 2

MEANS FOR TOTAL BODY WEIGHT
IN Gms

Diet	EXERCISE		
	R	F	S
P	402.6*	408.1	391.6
	±28.8	± 8.1	±10.4
A	416.6	417.1	471.5
	±15.1	±22.5	±11.0

* means ± standard error of the mean

LEGEND FOR ALL TABLES IN APPENDICES B, C, D

Diet: P - pair-feed
a - ad-libitum

Exercise: R - trained, sacrificed at rest
F - trained, sacrificed at fatigue
S - sedentary

TABLE 3

MEANS FOR LIVER WEIGHTS
IN Gms

Diet	EXERCISE		
	R	F	S
P	12.4*	11.5	13.4
	±1.2	±0.6	±0.6
A	14.7	13.3	16.4
	±0.8	±0.9	±0.5

* means ± standard error of the mean

TABLE 4

MEANS FOR HEART WEIGHTS
IN Mg

Diet	EXERCISE R	F	S
P	1026.1*	112.6	936.9
	± 41.5	±67.5	±24.5
A	1101.1	1120.8	1095.6
	± 34.3	± 48.6	± 22.4

* means ± standard error of the mean

TABLE 5

MEANS FOR RIGHT KIDNEY WEIGHTS
IN Mg

Diet	EXERCISE R	F	S
P	1396.01*	1312.48	1255.15
	± 76.51	± 25.88	± 42.38
A	1411.45	1393.68	1353.29
	± 43.14	± 77.84	± 29.64

* means ± standard error of the mean

TABLE 6

MEANS FOR SPLEEN WEIGHTS
IN Mg

Diet	EXERCISE R	F	S
P	617.2*	464.9°	627.6
	±128.7	±54.1	±138.0
A	667.0	484.1	708.1
	±48.8	±24.1	±21.2

* means ± standard error of the mean

TABLE 7

MEANS FOR ADRENAL WEIGHTS
IN Mg

Diet	EXERCISE R	F	S
P	24.38*	22.97	22.19
	±2.01	±1.09	±1.38
A	29.62	25.16	23.17
	±0.79	±2.41	±0.88

* means ± standard error of the mean

TABLE 8

MEANS FOR TESTICLE WEIGHTS
IN Mg

Diet	EXERCISE		
	R	F	S
P	1474.1*	1483.4	1477.4
	± 72.3	± 26.8	± 48.7
A	1546.6	1363.4	1591.7
	± 51.6	± 85.1	± 22.9

* means ± standard error of the mean

TABLE 9

MEANS FOR FAT PAD WEIGHTS
IN Mg

Diet	EXERCISE		
	R	F	S
P	1564.0*	1723.7	2696.4
	± 75.9	±162.2	±154.3
A	1894.0	1530.9	2454.0
	±104.8	±240.3	±133.8

* means ± standard error of the mean

TABLE 10

MEANS FOR GASTROCNEMIUS MUSCLE WEIGHTS IN Mg

Diet	EXERCISE R	F	S
P	1936.07*	1974.22	1951.09
	±109.09	± 38.73	± 53.97
A	1991.69	1836.30	2112.08
	± 83.63	±183.62	± 67.91

* means ± standard error of the mean

TABLE 11

MEANS FOR BICEPS BRACHII MUSCLE WEIGHTS IN Mg

Diet	EXERCISE R	F	S
P	227.71*	244.56	224.93
	±15.45	± 7.37	± 6.14
A	240.38	261.00	266.53
	± 5.48	±20.56	± 9.74

* means ± standard error of the mean

TABLE 12

MEANS FOR LIVER GLYCOGEN
IN Mg%

Diet	EXERCISE R	F	S
P	34.8*	01.3	36.1
	±05.0	±00.2	±00.2
A	40.7	10.3	30.5
	±04.6	±04.6	±03.1

* means ± standard error of the mean

TABLE 13

MEANS FOR GASTROCNEMIUS MUSCLE GLYCOGEN
IN Mg%

Diet	EXERCISE R	F	S
P	1.71*	0.66	1.28
	±0.56	±0.05	±0.18
A	1.07	0.95	1.55
	±0.27	±0.25	±0.39

* means ± standard error of the mean

TABLE 14

MEANS FOR BICEPS BRACHII MUSCLE GLYCOGEN
IN Mg%

Diet	EXERCISE		
	R	F	S
P	1.76*	1.91	1.52
	±0.39	±1.12	±0.27
A	1.87	1.48	1.25
	±0.63	±0.50	±0.23

* means ± standard error of the mean

TABLE 15

MEANS FOR GASTROCNEMIUS LACTATE

IN uEq/gm OF WET TISSUE

Diet	EXERCISE		
	R	F	S
P	58.00*	49.33	63.75
	±8.31	±5.60	±4.01
A	51.25	61.00	60.11
	±6.58	±8.84	±3.79

* means ± standard error of the mean

TABLE 16

MEANS FOR PLASMA LACTATE
IN uEq/ml

Diet	EXERCISE		
	R	F	S
P	31.1*	132.2	35.6
	±3.9	±17.3	±2.7
A	107.8	66.8	35.5
	±39.9	±33.3	±3.6

* means ± standard error of the mean

TABLE 17

MEANS FOR FAT PAD FREE FATTY ACID
IN uEq/gm

Diet	EXERCISE		
	R	F	S
P	2.04*	7.07	2.18
	±0.13	±0.26	±0.08
A	2.02	6.77	2.16
	±0.13	±0.66	±0.09

* means ± standard error of the mean

TABLE 18

MEANS FOR PLASMA FREE FATTY ACIDS
IN uEq/ml

Diet	EXERCISE		
	R	F	S
P	0.273*	0.609	0.289
	±0.020	±0.090	±0.010
A	0.283	0.652	0.208
	±0.010	±0.030	±0.010

* means ± standard error of the mean

TABLE 19

MEANS FOR PLASMA GLUCOSE
IN Mg%

Diet	EXERCISE		
	R	F	S
P	122.3*	62.4	153.3
	± 13.6	±14.9	± 11.3
A	171.9	100.6	148.5
	± 21.5	±28.9	± 5.3

* means ± standard error of the mean

APPENDIX C

STATISTICAL ANALYSIS

TABLE 20

SHEFFE MULTIPLE COMPARISON BETWEEN ORDERED MEANS: EFFECT OF EXERCISE

VARIABLE	GROUPS		
	Trained rest	Trained fatigue	Sedentary
LIVER WEIGHT; means (gm)	13.5	12.4	14.9
1		1.6168	1.9463
2			3.8002**
t(0.05)>2.509* 5(0.01)>3.159**			
SPLEEN WEIGHT; means (mg)	642.1	474.5	667.8
1		4.1284**	0.7321
2			5.4738**
t(0.05)>2.5179* t(0.01 >3.1654**			
LIVER GLYCOGEN; means (Mg%)	37.8	5.8	33.3
1		6.5208**	1.1280
2			6.5626**
t(0.05)>2.509* t(0.01)>3.159**			

TABLE 21

ANALYSIS OF VARIANCE OF SIMPLE MAIN EFFECTS FOR DIET

Source	SS	DF	MS	F
		KIDNEY WEIGHTS		
Diet	0.8769375E	1	0.8769375E	4.066
Error	0.1251072E	58	0.2157020E	
		HEART WEIGHTS		
Diet	0.1815834E	1	0.1815834E	12.704
Error	0.8290400E	58	0.1429379E	
Diet	0.1313848E	1	0.1313848E	11.023
Error	0.6793800E	57	0.1191895E	
		ADRENAL WEIGHTS		
Diet	0.1570388E	1	0.1570388E	4.038
Error	0.4744875E	122	0.3889241E	
		LIVER WEIGHTS		
iet	0.1066727E	1	0.1066727E	19.634
r	0.3151200E	58	0.5433103E	
		SPLEEN WEIGHTS		
Diet	0.5523634E	1	0.5523634E	4.522
Error	0.6718400E	55	0.1221527E	

TABLE 22

ANALYSIS OF VARIANCE OF SIMPLE MAIN EFFECTS FOR EXERCISE

Source	SS	DF	MS	F
		PLASMA GLUCOSE		
Exercise	0.4552426E	1	0.4552426E	2.564
Error	0.9944200E	56	0.1775750E	
		EPIDIDYMAL FAT PAD WEIGHTS		
Exercise	0.2853622E	1	0.2853622E	0.530
Error	0.6574522E	122	0.5388952E	
		LIVER GLYCOGEN		
Exercise	0.3965378E	1	0.3965378E	0.002
Error	0.1102192E	58	0.1900331E	
		ADRENAL WEIGHTS		
Exercise	0.4208511E	1	0.2104255E	5.410
Error	0.4744875E	122	0.3889241E	
		LIVER WEIGHTS		
Exercise	0.6183270E	1	0.3091635E	5.690
Error	0.3151200E	58	0.5433103E	
		HEART WEIGHTS		
Exercise	0.1255994E	1	0.6279969E	4.393
Error	0.8290400E	58	0.1429379E	
		SPLEEN WEIGHTS		
Exercise	0.3189963E	1	0.1594981E	13.057
Error	0.6718400E	55	0.1221527E	

TABLE 23

PRIMARY MAIN EFFECTS FOR SIGNIFICANT DIET AND EXERCISE INTERACTIONS

Source	SS	DF	MS	F
		PLASMA LACTATES		
Pair-fed	0.62174687E	2	31087.34	37.86
Error	0.23810187E	29	821.04	
Ad-libitum	0.29776937E	2	14888.47	3.73
Error	0.11566306E	29	3988.38	
		PLASMA FFA		
Pair-fed	0.68454647E	2	0.34	93.15
Error	0.10655499E	29	0.00	
Ad-libitum	0.77568531E	2	0.39	279.08
Error	0.40302277E	29	0.00	
		TOTAL BODY WEIGHT		
Pair-fed	0.17250000E	2	862.50	0.38
Error	0.65395000E	29	2255.00	
Ad-libitum	0.25893000E	2	12946.50	5.02
Error	0.82554000E	29	2579.81	

APPENDIX D

RAW DATA

TABLE 24

ORGAN AND MUSCLE WEIGHTS FOR GROUP PR

Rat No.	Total Body (g)	Liver (g)	Heart (mg)	Right Kidney (mg)	Spleen (mg)	Right Adrenal (mg)	Left Adrenal (mg)	Biceps Brachii (mg)
3	499	16.2	1189.2	1686.2	841.2	24.2	29.2	276.2
11	394	13.4	983.2	1207.3	587.8	28.2	26.4	242.8
33	444	11.6	1058.6	1458.0	637.0	24.1	22.2	247.8
59	454	15.6	1118.6	1504.6	649.8	39.2	28.0	-
82	425	13.8	1022.2	1322.2	642.2	27.8	29.2	253.6
124	303	8.0	900.2	1054.4	412.6	18.8	14.2	150.6
125	299	8.4	901.4	1090.4	550.8	22.0	7.6	213.0

TABLE 24 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP PR

Rat No.	Right Testicle (mg)	Left Testicle (mg)	Right Fat Pad (mg)	Left Fat Pad (mg)	Right Gastrocnemius (mg)	Left Gastrocnemius (mg)
3	1506.0	1429.0	2166.2	2024.2	2545.6	2484.2
11	1572.9	1583.8	1352.8	1382.9	2139.7	2039.7
33	1500.0	1434.0	1686.0	1686.0	2085.2	1730.2
59	1516.4	1547.8	1759.0	1693.8	2228.2	2232.8
82	1694.2	1694.2	1254.2	1370.2	1649.8	2164.2
124	1673.6	1683.0	-	1440.4	1355.8	1665.8
125	1082.2	710.0	-	1203.8	1202.4	1588.2

TABLE 25

ORGAN AND MUSCLE WEIGHTS FOR GROUP PF

Rat No.	Total Body (g)	Liver (g)	Heart (mg)	Right Kidney (mg)	Spleen (mg)	Right Adrenal (mg)	Left Adrenal (mg)	Biceps Brachii (mg)
4	406	9.0	964.0	1295.6	460.0	25.8	26.2	266.0
7	403	10.5	1135.6	1267.6	414.2	19.8	19.6	210.4
8	452	14.1	1464.0	1349.6	486.4	28.4	16.4	215.4
10	389	11.2	858.6	1283.6	387.0	26.0	26.4	247.8
26	373	11.0	980.0	1252.0	523.0	23.0	24.0	272.0
29	400	11.4	1153.8	1337.4	426.8	14.0	14.0	255.6
57	394	10.6	1013.8	1225.4	458.8	28.2	28.2	244.6
123	420	11.8	1041.9	1310.0	466.4	21.8	21.8	230.0
126	436	14.0	1121.4	1491.0	562.0	25.2	24.8	262.8

TABLE 25 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP PF

Rat No.	Right Testicle (mg)	Left Testicle (mg)	Right Fat Pad (mg)	Left Fat Pad (mg)	Right Gastrocnemius (mg)	Left Gastrocnemius
4	1562.4	1528.0	1367.4	1330.6	2013.4	1881.4
7	1452.8	1410.0	1492.8	1326.2	2126.2	1909.2
8	1782.2	1682.8	3819.0	2535.0	2108.6	1992.8
10	1456.4	1435.6	1012.2	1106.6	2145.0	1983.4
26	1498.0	1473.0	980.0	1052.0	1551.0	1897.0
29	1438.2	1428.0	2155.0	2077.6	2164.0	1960.0
57	1381.8	1377.2	1800.2	1579.4	1921.2	1850.2
123	1306.4	1402.8	1862.9	1777.3	2191.0	2100.0
126	1513.0	1572.0	1706.8	2201.8	1713.4	2032.8

TABLE 26

ORGAN AND MUSCLE WEIGHTS FOR GROUP PS

Rat No.	Total Body (g)	Liver (g)	Heart (mg)	Right Kidney (mg)	Spleen (mg)	Right Adrenal (mg)	Left Adrenal (mg)	Biceps Brachii (mg)
16	454	14.2	1032.2	1151.8	503.8	20.2	19.4	206.2
17	431	15.0	992.4	1605.0	746.4	25.3	32.0	
20	434	14.2	933.0	1360.0	667.4	17.8	20.4	223.4
22	424	16.0	905.0	1229.4	717.2	12.8	12.8	232.4
23	389	13.0	860.2	1186.0	540.0	26.4	31.8	205.0
24	325	8.8	894.0	1059.2	555.6	17.2	18.8	184.0
37	410	11.8	878.0	1155.0	700.8	10.4	13.8	249.2
38	335	9.5	748.0	970.0	308.8	13.4	7.4	211.8

TABLE 26 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP PS

Rat No.	Right Testicle (mg)	Left Testicle (mg)	Right Fat Pad (mg)	Left Fat Pad (mg)	Right Gastrocnemius (mg)	Left Gastrocnemius (mg)
16	1519.8	1466.4	3840.4	2442.4	2362.2	2122.2
17	1682.2	1719.4	[illegible]	2184.8	2331.0	2341.4
20	1768.2	1773.0	[illegible]	2462.4	2223.2	2089.8
22	1640.0	1626.2	4181.0	4417.2	2295.2	2040.0
23	1330.0	1319.8	3525.0	3024.8	1876.6	[illegible]
24	574.2	593.8	[illegible]	3341.6	2060.2	1998.0
37	1448.0	1474.8	[illegible]	2249.0	2263.6	2335.0
38	1461.2	1433.4	1067.2	1084.6	1896.2	[illegible]

TABLE 26 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP PS

Rat No.	Total Body (g)	Liver (g)	Heart (mg)	Right Kidney (mg)	Spleen (mg)	Right Adrenal (mg)	Left Adrenal (mg)	Biceps Brachii (mg)
40	337	16.8	1162.2	1553.9	749.4	20.2	20.4	203.0
42	369	12.0	854.6	1106.0	540.0	42.0	41.6	238.2
45	384	15.4	938.8	1326.0	588.6	23.8	24.0	252.2
55	407	14.0	1001.4	1370.8	835.0	26.3	27.5	266.2
68	455	14.0	1001.0	1229.8	840.2	18.2	28.6	227.7
69	380	11.0	902.6	1136.0	562.4	22.6	20.4	245.8
92	393	14.0	815.9	1243.6	529.7	21.9	21.9	200.0
93	428	14.6	981.9	1330.0	656.2	23.2	27.8	203.4

TABLE 26 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP PS

Rat No.	Right Testicle (mg)	Left Testicle (mg)	Right Fat Pad (mg)	Left Fat Pad (mg)	Right Gastrocnemius (mg)	Left Gastrocnemius (mg)
40	1570.4	1553.8	1933.0	2012.0	1437.0	1589.0
42	1475.2	1399.8	1805.4	2138.0	1538.4	1553.8
45	1623.0	1555.8	3864.0	3246.4	2162.2	1966.4
68	1556.0	1521.0	2786.4	3037.2	2196.0	2192.6
69	1638.2	1678.6	2716.6	2478.8	1836.2	1668.6
92	1504.6	1536.9	1730.9	2089.6	2010.9	1997.4
93	1692.6	1647.8	2927.6	3015.6	1526.0	1142.2
55	1508.3	1034.0	2502.5	-	1734.6	1920.3

TABLE 27

ORGAN AND MUSCLE WEIGHTS FOR GROUP AR

Rat No.	Total Body (g)	Liver (g)	Heart (mg)	Right Kidney (mg)	Spleen (mg)	Right Adrenal (mg)	Left Adrenal (mg)	Biceps Brachii (mg)
97	403	14.0	971.6	1278.2	650.0	28.3	24.7	223.8
98	343	13.2	1136.0	1589.0	761.0	29.0	31.0	225.0
99	442	17.6	1040.3	1430.2	764.2	21.2	28.2	239.4
100	408	13.7	1064.6	1541.2	605.5	30.4	29.6	253.2
101	455	16.4	1288.4	1449.2	807.3	31.0	33.7	255.5
105	472	16.4	1104.0	1442.0	668.4	32.0	33.4	292.4
106	434	16.0	1040.2	1255.0	711.9	29.1	32.8	236.5
108	376	10.4	1164.2	1306.2	367.8	29.8	29.8	200.0

TABLE 27 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP AR

Rat No.	Right Testicle (mg)	Left Testicle (mg)	Right Fat Pad (mg)	Left Fat Pad (mg)	Right Gastrocnemius (mg)	Left Gastrocnemius (mg)
97	1700.0	1650.0	1571.9	1631.0	1938.2	2008.2
98	1494.0	[illegible]	1270.0	1286.0	1518.0	1094.0
99	1347.4	1543.8	2333.2	2227.8	2109.2	2138.2
100	1583.9	1703.2	2350.2	2050.9	2078.6	2028.2
101	1776.6	1782.8	2390.1	2724.2	2303.1	2204.6
105	1643.6	1674.0	1929.6	1679.2	2427.6	2353.0
106	1551.9	1590.1	1820.0	1847.3	2176.4	1954.2
108	1380.4	1358.2	1596.2	1596.2	1719.6	1820.6

TABLE 28

ORGAN AND MUSCLE WEIGHTS FOR GROUP AF

Rat No.	Total Body (g)	Liver (g)	Heart (mg)	Right Kidney (mg)	Spleen (mg)	Right Adrenal (mg)	Left Adrenal (mg)	Biceps Brachii (mg)
31	381	12.6	1064.0	1299.0	480.0	18.0	18.0	226.0
84	419	11.6	1084.3	1176.6	433.8	28.2	33.6	251.9
102	529	15.4	1300.2	1572.6	473.0	16.2	14.2	316.6
103	301	11.1	1044.0	1350.0	-	28.0	31.0	211.0
107	447	16.0	1147.6	1570.2	549.8	32.4	32.0	301.8

TABLE 28 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP AF

Rat No.	Right Testicle (mg)	Left Testicle (mg)	Right Fat Pad (mg)	Left Fat Pad (mg)	Right Gastrocnemius (mg)	Left Gastrocnemius (mg)
31	1412.0	1403.0	1384.0	1201.0	1719.0	1677.0
84	1054.6	1116.4	1426.7	1671.4	2334.6	2384.6
102	1530.0	1507.2	2773.5	2671.0	2227.2	2194.4
103	1376.0	1412.0	-	-	741.0	967.0
107	1868.0	907.8	1962.4	1040.0	2124.4	2201.4

TABLE 29

ORGAN AND MUSCLE WEIGHTS FOR GROUP AS

Rat No.	Total Body (g)	Liver (g)	Heart (mg)	Right Kidney (mg)	Spleen (mg)	Right Adrenal (mg)	Left Adrenal (mg)	Biceps Brachii (mg)
27	396	14.2	942.8	1217.2	599.2	25.0	26.2	252.0
41	483	16.8	1162.2	1553.9	749.4	20.2	20.4	270.2
50	571	21.2	1224.6	1522.0	816.4	27.3	28.2	213.0
71	449	13.8	973.2	1216.2	694.2	19.4	22.2	323.8
72	472	17.2	1017.8	1282.2	792.8	21.4	23.8	323.8
95	525	19.2	1165.6	1373.6	792.4	26.0	25.4	342.3
109	450	16.2	1148.0	1249.0	-	24.0	28.0	257.0
110	466	18.4	1092.0	1269.0	-	20.0	23.0	282.0
111	416	15.2	925.6	-	565.8	9.0	9.6	227.8

TABLE 29 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP AS

Rat No.	Right Testicle (mg)	Left Testicle (mg)	Right Fat Pad (mg)	Left Fat Pad (mg)	Right Gastrocnemius (mg)	Left Gastrocnemius (mg)
27	1400.2	1560.0	1697.8	2103.2	2006.2	1080.4
41	1570.4	1553.8	1599.8	2593.2	2525.2	2480.8
50	1849.8	1849.8	4625.9	4611.8	2687.8	2067.8
71	1590.8	1471.8	1991.6	1772.2	2123.2	1774.2
72	1532.0	1532.6	1952.4	2181.8	2424.8	2489.4
95	1505.6	1568.8	3118.9	3023.3	1515.8	1058.2
109	1596.0	1617.0	1909.0	1864.0	2220.0	1964.0
110	1598.0	1576.0	2975.0	3032.0	1923.0	2168.0
111	-	-	1825.4	2139.0	2023.0	1967.0

TABLE 29 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP AS

Rat No.	Total Body (g)	Liver (g)	Heart (mg)	Right Kidney (mg)	Spleen (mg)	Right Adrenal (mg)	Left Adrenal (mg)	Biceps Brachii (mg)
112	487	19.2	1225.8	1603.8	792.6	27.2	26.4	260.4
113	484	15.6	1063.7	1320.9	681.9	20.9	22.8	271.9
114	456	16.4	1252.0	1269.4	625.6	12.0	11.8	248.6
115	484	14.6	1160.2	1466.8	873.0	28.2	28.2	254.0
116	414	12.0	1022.0	1234.6	630.2	22.4	21.8	180.2
117	552	18.4	1134.0	1440.6	655.5	28.4	26.0	330.0
118	393	13.0	1005.4	1215.4	692.2	17.8	25.8	230.2
119	517	17.0	1168.2	1427.8	742.2	22.2	27.8	296.2
120	475	16.7	1086.0	1466.0	798.6	25.4	24.3	234.6
121	468	17.2	1047.2	1394.2	625.2	31.8	30.0	272.4

TABLE 29 (Cont'd)

ORGAN AND MUSCLE WEIGHTS FOR GROUP AS

Rat No.	Right Testicle (mg)	Left Testicle (mg)	Right Fat Pad (mg)	Left Fat Pad (mg)	Right Gastrocnemius (mg)	Left Gastrocnemius (mg)
112	1061.0	1677.8	1792.4	2105.8	2234.0	2345.4
113	1661.9	1684.6	2040.0	2054.6	1860.9	2264.9
114	1581.6	1598.8	2550.2	2547.0	2024.2	2143.0
115	1880.0	1679.2	2757.4	2674.5	2425.2	2221.2
116	1634.6	1590.6	2672.6	2701.0	1642.2	1723.4
117	1680.0	1720.0	2773.0	2711.4	2853.0	2832.4
118	1718.6	1680.2	1960.2	2572.2	2107.6	2143.4
119	1488.2	1457.8	3487.6	3431.0	2306.3	2878.8
120	1713.8	1647.6	2232.7	1962.8	2376.3	2070.6
121	1508.0	1450.4	3532.8	-	1544.0	1776.4

TABLE 30

BLOOD LACTATES, GLUCOSE AND FREE FATTY ACIDS AND FAT PAD FREE FATTY ACIDS FOR GROUP PR

	LACTATES	FREE FATTY ACIDS			GLUCOSE
Rat No.	Plasma mg%	Plasma uEq/ml	Right Fat Pad uEq/g	Left Fat Pad uEq/g	Plasma mg%
3	36	1.62	1.83	0.280	117
11	40	1.86	2.61	0.321	182
33	28	1.62	2.46	0.314	127
59	47	2.13	2.13	0.278	79
82	24	1.13	1.96	0.153	142
124	17	-	2.63	0.268	130
125	26	2.48	1.48	0.300	79

TABLE 31

BLOOD LACTATES, GLUCOSE AND FREE FATTY ACIDS AND FAT PAD FREE FATTY ACIDS FOR GROUP PF

	LACTATES	FREE FATTY ACIDS			GLUCOSE
Rat No.	Plasma mg%	Plasma uEq/ml	Right Fat Pad uEq/g	Left Fat Pad uEq/g	Plasma mg%
4	48	0.557	7.13	6.32	60
7	160	0.538	6.32	6.57	37
8	180	0.555	6.13	-	-
10	90	0.764	5.91	8.46	40
26	130	0.692	-	-	24
29	82	0.713	8.13	8.13	40
57	120	0.543	6.81	7.43	91
123	200	0.624	7.31	8.02	154
126	180	0.492	6.37	5.97	53

TABLE 32

BLOOD LACTATES, GLUCOSE AND FREE FATTY ACIDS AND FAT PAD FREE FATTY ACIDS FOR GROUP PS

	LACTATES	FREE FATTY ACIDS			GLUCOSE
Rat No.	Plasma mg%	Plasma uEq/ml	Right Fat Pad uEq/g	Left Fat Pad uEq/g	Plasma mg%
16	45	0.303	3.12	2.00	163
17	38	0.246	1.97	1.87	133
20	27	0.321	1.97	2.46	143
22	28	0.316	2.31	2.76	113
23	200	0.327	2.12	2.12	174
24	39	0.286	2.11	1.83	110
37	54	0.308	2.78	2.31	143
38	39	0.300	1.79	-	200

TABLE 32 (Cont'd)

BLOOD LACTATES, GLUCOSE, FREE FATTY ACIDS AND FAT PAD FREE FATTY ACIDS FOR GROUP PS

	LACTATES	FREE FATTY ACIDS			GLUCOSE
Rat No.	Plasma mg%	Plasma uEq/ml	Right Fat Pad uEq/g	Left Fat Pad uEq/g	Plasma mg%
40	48	0.281	1.75	1.37	105
42	37	0.217	2.16	1.96	105
45	25	0.262	1.17	2.81	231
55	24	0.332	2.64	2.64	171
68	50	0.262	2.13	2.13	117
69	23	0.284	1.81	1.81	116
92	30	0.280	2.63	2.63	194
93	43	0.341	2.04	2.04	186

TABLE 33

BLOOD LACTATES, GLUCOSE, FREE FATTY ACIDS AND FAT PAD FREE FATTY ACIDS FOR GROUP AR

	LACTATES	FREE FATTY ACIDS			GLUCOSE
Rat No.	Plasma mg%	Plasma uEq/ml	Right Fat Pad uEq/g	Left Fat Pad uEq/g	Plasma mg%
97	25	0.241	1.83	2.41	123
98	24	0.300	1.23	1.61	174
99	40	0.304	2.14	1.79	168
100	35	0.322	3.31	1.78	200
101	37	0.205	1.92	1.86	177
105	200	0.300	2.13	1.93	147
106	35	0.312	1.32	2.22	124
108	180	0.280	2.44	2.43	109

TABLE 34

BLOOD LACTATES, GLUCOSE, FREE FATTY ACIDS AND FAT PAD FREE FATTY ACIDS FOR GROUP AF

	LACTATES	FREE FATTY ACIDS			GLUCOSE
Rat No.	Plasma mg%	Plasma uEq/ml	Right Fat Pad uEq/g	Left Fat Pad uEq/g	Plasma mg%
31	39	0.583	-	-	113
84	200	0.631	4.41	6.67	31
102	30	0.592	7.13	6.78	184
103	33	0.478	-	-	135
107	32	0.813	8.31	7.33	40

TABLE 35

BLOOD LACTATES, GLUCOSE, FREE FATTY ACIDS AND FAT PAD FREE FATTY ACIDS FOR GROUP AS

	LACTATES	FREE FATTY ACIDS			GLUCOSE
Rat No.	Plasma mg%	Plasma uEq/ml	Right Fat Pad uEq/g	Left Fat Pad uEq/g	Plasma mg%
27	32	0.319	1.63	1.14	100
41	40	0.302	2.33	2.31	144
50	32	0.341	1.98	2.07	188
71	31	0.263	2.31	2.62	184
72	26	0.242	1.37	2.43	148
95	34	0.394	3.32	-	153
109	30	0.316	2.31	2.16	153
110	46	0.298	2.43	1.76	132
111	90	0.300	2.41	2.33	194

TABLE 35 (Cont'd)

BLOOD LACTATES, GLUCOSE, FREE FATTY ACIDS AND FAT PAD FREE FATTY ACIDS FOR GROUP AS

	LACTATES	FREE FATTY ACIDS			GLUCOSE
Rat No.	Plasma mg%	Plasma uEq/ml	Right Fat Pad uEq/g	Left Fat Pad uEq/g	Plasma mg%
112	90	0.253	2.41	2.30	153
113	25	0.317	2.13	2.32	158
114	31	0.312	2.14	2.12	142
115	21	0.316	2.32	1.91	121
116	39	0.302	1.82	2.16	131
117	30	0.330	-	-	142
118	31	0.300	1.87	1.96	163
119	53	0.278	1.87	1.93	153
120	25	0.264	1.97	1.87	126
121	20	0.341	2.31	2.04	137

TABLE 36

TISSUE GLYCOGEN AND LACTATE FOR

GROUP PR

	GLYCOGEN			LACTATE
Rat No.	Liver mg%	Gastrocnemius mg%	Biceps mg%	Gastrocnemius umoles/gm
3	34.4	0.66	0.99	10
11	49.7	4.91	2.99	65
33	10.6	0.75	1.45	64
59	37.9	1.52	3.45	71
82	39.4	0.90	0.80	76
124	4.58	1.44	1.44	-
125	2.56	1.73	1.19	62

TABLE 37

TISSUE GLYCOGEN AND LACTATE FOR

GROUP PF

	GLYCOGEN			LACTATE
Rat No.	Liver mg%	Gastrocnemius mg%	Biceps mg%	Gastrocnemius umoles/gm
4	0.70	0.70	0.34	58
7	1.99	0.58	0.72	26
8	2.74	0.61	0.78	40
10	0.44	0.50	0.97	27
26	1.02	0.56	0.99	43
29	0.92	0.58	0.98	71
57	1.54	0.55	-	71
123	1.23	1.49	1.49	50
126	08	0.54	0.54	58

TABLE 38

TISSUE GLYCOGEN AND LACTATE FOR

GROUP PS

	GLYCOGEN			LACTATE
Rat No.	Liver mg%	Gastrocnemius mg%	Biceps mg%	Gastrocnemius umoles/gm
1	36.1	0.75	0.83	57
17	31.1	0.70	-	65
20	82.9	0.83	1.26	69
22	45.2	1.24	1.64	61
23	32.3	1.18	1.95	73
24	11.6	0.93	2.07	78
37	8.0	0.58	1.21	64
38	59.3	0.98	0.72	21

TABLE 38 (Cont'd)

TISSUE GLYCOGEN AND LACTATE FOR

GROUP PS.

	GLYCOGEN			LACTATE
Rat No.	Liver mg%	Gastrocnemius mg%	Biceps mg%	Gastrocnemius umoles/gm
40	26.7	2.47	3.78	94
42	26.5	3.19	3.66	58
45	38.1	1.12	0.89	48
55	33.3	0.73	0.78	62
68	56.5	1.09	0.46	51
69	35.6	1.79	3.50	77
92	20.95	0.96	1.86	74
93	33.5	1.99	4.10	68

TABLE 39

TISSUE GLYCOGEN AND LACTATE FOR

GROUP AR

	GLYCOGEN			LACTATE
Rat No.	Liver mg%	Gastrocnemius mg%	Biceps mg%	Gastrocnemius umoles/gm
97	43.7	0.84	2.65	35
98	31.1	2.59	3.57	62
99	43.8	0.79	1.07	64
100	26.0	0.74	0.90	29
101	37.7	5.26	3.23	49
105	5.1	0.78	0.78	37
106	65.1	1.03	0.69	69
108	2.8	0.50	0.86	77

TABLE 40

TISSUE GLYCOGEN AND LACTATE FOR

GROUP AF

	GLYCOGEN			LACTATE
Rat No.	Liver mg%	Gastrocnemius mg%	Biceps mg%	Gastrocnemius umoles/gm
31	25.00	1.39	1.75	72
84	2.28	0.53	0.77	74
102	5.16	0.59	0.75	49
103	16.90	1.72	3.33	78
107	1.97	0.53	0.82	32

TABLE 41

TISSUE GLYCOGEN AND LACTATE FOR

GROUP AS

	GLYCOGEN			LACTATE
Rat No.	Liver mg%	Gastrocnemius mg%	Biceps mg%	Gastrocnemius umoles/gm
27	32.6	0.70	0.73	76
41	35.8	0.67	0.94	69
50	21.3	0.29	0.53	27
71	27.1	0.58	0.74	43
72	50.8	1.64	2.75	79
95	18.7	2.17	1.01	72
109	16.6	4.24	4.00	74
110	21.1	3.49	4.36	84
111	43.2	0.76	0.92	56

TABLE 41 (Cont'd)

TISSUE GLYCOGEN AND LACTATE FOR

GROUP AS

	GLYCOGEN			LACTATE
Rat No.	Liver mg%	Gastrocnemius mg%	Biceps mg%	Gastrocnemius umoles/gm
112	43.7	0.74	0.83	70
113	18.3	0.89	0.53	34
114	60.9	0.71	1.05	59
115	12.9	1.02	0.98	66
116	18.6	1.51	0.71	68
117	44.4	1.02	1.15	48
118	19.5	0.68	0.85	41
119	26.2	0.44	0.86	63
120	40.9	0.62	0.87	41
121	26.9	0.94	2.47	72