EFFECT OF EXERCISE DURATION ON FOOD INTAKE, BODY WEIGHT, AND BODY COMPOSITION OF MICE

by

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INTRODUCTION

Obesity, or excessive body fatness, is a serious problem in the United States. It is estimated that at least 50 million adult men and 60 million adult women are obese (1). Obesity is not only undesirable to one's self image and perhaps social acceptance, but it also has been associated with at least 26 known medical conditions which could account collectively for 15-20% of the mortality rate (2). These conditions include hypertension, diabetes, renal disease, gallbladder disease, osteoarthritis, and impaired cardiac and respiratory function.

There is available to the American public a tremendous array of fad reducing diets, diet pills, reducing gadgets, and spas. In contrast, most reliable health professionals would suggest a sensible reduced calorie diet and exercise plan.

Few people would dispute the fact that exercise increases caloric expenditure and therefore may help prevent obesity. However, a lesser known effect of exercise is its effect on hunger and voluntary food intake. Although one might expect that the increased caloric expenditure during exercise would be followed by a compensatory increase in hunger and food intake, there is some evidence that this is not always true. A study of this relationship is presented in this thesis.

REVIEW OF LITERATURE

I. The Control of Food Intake

Energy balance is the relationship between energy intake and energy expenditure. When an individual is in positive energy balance he consumes more energy than he expends resulting in a net storage of energy in the body. Conversely, when an individual is in negative energy balance he expends more calories than he consumes and utilizes body energy stores. In zero energy balance, energy intake equals energy expenditure and there is no net gain or loss of body energy.

Many human beings and various animal species are exposed to a wide variety and overabundance of food but yet retain remarkably constant body weights. This suggests that there are physiological mechanisms which control the amount of energy consumed relative to energy expenditure. Although the exact nature of the control mechanism(s) is not clear, there are several theories which have been postulated. These theories can be roughly categorized as homeostatic or nonhomeostatic theories. They are described below.

Homeostatic Theories. According to homeostatic theories organisms are believed to have a genetically determined "set point" for body weight, body composition or food intake. Factors which affect the set point resulting in either an increase or decrease in food intake have been extensively reviewed (3). Although many homeostatic theories have been proposed, the most common ones are those involving gastrointestinal feedback, changes in body temperature, circulating levels of glucose or amino acids, amount of body fat, and control by neurotransmitters and hormones.

Some of the problems associated with studying the physiological control of food intake can be explained by the fact that the ingestion of a single food brings about many physical and chemical changes, and it is difficult to identify from these changes a single responsible homeostatic factor. No one theory has been conclusively accepted but the experimental evidence supporting or challenging each theory will be presented.

Feeding begins with taste sensations and a filling of the oral cavity. Appetite, or desire for food whether or not one is hungry, may be stimulated by the palatability of the food (4). It is not uncommon for an individual to consume a special dessert after he has been satisfied by an adequate meal. Texture and mouthfeel are also important influences on food intake (4). Although these factors may alter food intake, and thus energy intake, they are not major factors affecting energy balance.

Perhaps the oldest theory on the regulation of hunger was proposed by Cannon and coworkers in 1912 as cited in a review by Anand (5). They suggested that hunger was regulated by gastric contractions and stomach distention, and that these changes were mediated by messages sent by the vagus nerve. Anand (5) studied the effect of gastric distention on the hypothalamus centers of the brain. He found that gastric distention led to increased electrical activity of the "satiety" ventromedial hypothalamus (VMH), or satiety center, but no differences in activity of the feeding center.

Mayer (6) found that glucagon injection was followed by an inhibition of gastric contractions in rats. However, if VMH lesions were induced in these rats they did not exhibit the gastric contractions. This led some researchers to believe that the hypothalamus is involved in the gastric response to food intake.

A similar theory, as reviewed by Kissileff and Van Itallie (3), acknowledged the importance of gastric emptying but related it to caloric intake. This theory stated that stomach emptying will be slower and hunger will decrease when the caloric intake is greater than energy expenditure. However, studies have shown that glucose and xylose, a poorly metabolized pentose, leave the stomach at the same time and suppress hunger equally even though xylose does not contain any detectable calories. Furthermore, fructose was shown to leave the stomach twice as fast as glucose yet both were followed by a similar suppression in food intake. The theories involving the role of stomach distention on hunger were abandoned when it was found that gastrectomized patients still experienced hunger sensations and satiety (review by Anand).

Some investigators believe that the liver is the organ responsible for monitoring the state of the body's energy stores. In that hypothesis, reviewed by Kissileff (3), hunger occurs when insufficient fuel is delivered to the liver from the intestine and adipose tissue to maintain body function. The adequacy of these fuels is determined by hepatic receptors which in turn affect the sensation of hunger and satiety.

The thermostatic theory controlling food intake was proposed by Brobeck in 1948 as cited in a review by Anand (5). He believed that the important factors in the regulation of food intake was not its energy value but the amount of extra heat released, as a part of specific dynamic energy (SDE), following a meal. This release of energy signals the hypothalamus and results in an adjustment in the total quantity of food eaten. In this theory, the principle factor in satiety is the stress upon the body produced by the extra heat. The experimental evidence in support of this theory is based on the observation that most animals

increase food intake in cold temperatures, and reduce it in the hot temperatures (4). In a review by Anand (5) the preoptic anterior hypothalamus has been identified as the thermosensitive area of the brain. However, it has been argued that the thermostatic theory does not explain how the hypothalamus receptors distinguish heat released from SDE of a meal and the greater amount of heat released during exercise. Furthermore, it was shown that the addition of thyroprotein, which increases heat loss to food was followed by an increase in food intake where it may have been expected to decrease food intake.

Mayer (7) developed the concept of a glucostatic mechanism. In this theory, he proposed the presence of glucoreceptors sensitive to blood utilization in the VMH centers in the brain. He reasoned that glucose was the most logical effector because between meals carbohydrate stores are more influenced (decreased) than are stores of body fat or protein. Furthermore, glucose is the preferred energy source for the nervous system, of which the VMH centers are a part. This theory is supported by the finding that low levels of glucose utilization by the body cells corresponded to verbal descriptions of hunger in human subjects. Further evidence was demonstrated when a single injection of gold thioglucose into mice induced lesions in the VMH and was followed by permanent over-eating, impaired reaction to cold temperatures, exercise and caloric density adjustments. The VMH neurons were selectively poisoned because of the attraction of these cells for the glucose component of the molecule. Other evidence supporting this theory includes an observed increase in electrical activity in the satiety center during hyperglycemia but an increased activity in the feeding center during hypoglycemia (5). On the basis of the research done by Mayer and others, the glucostatic theory presents one

possible component of the physiological mechanism underlying hunger and satiety. There are, however, many other factors which may influence food intake.

The lipostatic theory was originally proposed by Kennedy in 1953 (8). Kennedy speculated that because the amount of fat depot relative to body weight is fairly consistent in adults, this factor may be involved in the regulation of food intake. In the lipostatic theory any deviation results in a release of an unknown metabolite which then signals the central nervous system. This is followed by an increase or decrease in food intake relative to the amount of body fat.

There is only circumstantial evidence to support this theory. Rats made obese by forced intragastric feeding ate less food until their normal lower body weights were attained (9). Further evidence supporting this theory was presented from earlier work by Hoebel et al. as reviewed by Hamilton (4). They showed that animals made obese by daily potamine zinc insulin injections and having VMH lesions did not exhibit hyperphagia. Not until they were fasted to a lower body weight did they begin to over-eat and regain weight. This has led researchers to believe that an important VMH function is the adjustment of the set point to achieve the genetically-determined amount of body fat.

The mechanism controlling food intake also may be related to plasma amino acid levels (10,11). Convincing evidence in support of this theory was demonstrated when rats fed a 5% protein diet while exposed to 25°C temperature did not eat and died. Rats fed a 5% protein diet and exposed to 8°C temperature thrived. In addition to cold and stress, exercise also was followed by an increase in food intake. Under these conditions, cold, stress, and exercise seem to alter some internal mechanism that leads to an

increase in food intake in low-protein-fed-rats, possibly to cover their protein requirement for survival (4). Animals fed diets lacking a single essential amino acid exhibit a reduction in food intake (11), but this finding is not well understood. It has been suggested that the observed anorexia may be a protective mechanism against the development of pathological lesions or other abnormalities resulting in death (11). In another study rats preferred diets containing all of the essential amino acids over those lacking in essential amino acids when given a choice (4). Furthermore, in the same study rats preferred a 25% protein diet over those containing higher or lower amounts of protein.

The neurotransmitters involved in the control of food intake also have been studied, particularly catecholamines in hypothalamic neurons (12,13). Because norepinephrin (NOREP) is an inhibitory transmitter, its elevation in the medial area of the hypothalamus has been shown to inhibit the electrical activity of satiety neurons, thus initiating feeding. Feeding was shown to be suppressed by a duodenally-activated mechanism that increases NOREP in the lateral area thereby inhibiting feeding center neurons.

Purified cholecystokinin (CCK), a humoral factor released from the intestine has also been implicated in satiety (13,14). Cholecystokinin also has been shown to alter the release of NOREP from receptor sites in the hypothalamus so the suppression of food intake seen with CCK may be attributed to its effect on noradrenergic neurons in both satiety and feeding centers (13).

Serotonin (5-HT) also is believed to be involved in controlling hunger and food intake. Waldbillig et al. (15) reported an inhibitory role of 5-HT on food intake in rats. Rats subjected to 5-HT depletion

became hyperphagic and gained weight. The results of the study thus demonstrated a reciprocal relationship between 5-HT levels and food intake. Wurtman and Wurtman (16) reported that MK-212, an anorectic drug which enhances serotoninergic transmission, reduced food intake when injected into rats. More specifically, this anorectic drug seemed to decrease consumption of carbohydrate without affecting protein intake when animals were presented with various combinations and proportions of food. Serotonin may be specifically involved in regulating the consumption of those nutrients (protein and carbohydrate) which affect its own synthesis and release.

The VMH content of gamma aminobutyric acid (GABA), an inhibitory neurotransmitter, may be affected by plasma glucose levels. The high concentration of this inhibitory neurotransmitter in the VMH increases during hypoglycemia which suggests GABA may inhibit satiety and stimulate eating (12).

Nonhomeostatic Theories. In contrast to the homeostatic theories on the regulation of food intake, nonhomeostatic theories do not recognize the importance of a set point in maintaining body weight. Rather they imply that the primary control of food intake is the result of environmental conditions, genetic make-up of the organism, or of psychological rewards or pleasures obtained from food.

The proponents of the nonhomeostatic theories have realized for some time that freely eating animals eat in discrete meals. For many animals the pattern of these meals is such that they seldom undergo short-term depletion and thus never experience hunger (17).

Meal frequency and duration, intensity of feeding, and food choices are all factors which can be affected by an animal's environment.

Herbivores usually eat frequent meals of long duration and at low intensity, while carnivores eat large infrequent meals at low intensity particularly if not threatened by predators. Small carnivores who are potential prey eat large meals at high intensities (18). Collier et al. (17) have concluded that the availability of food, its caloric density, the work and danger in acquiring food and the animal's position in the food chain determines the feeding pattern.

Kanarck (18) studied the food intake and meal pattern of rats offered three levels of caloric density: standard, diluted, and concentrated. Availability of the diets was limited by requiring the rats to press a bar for the food. The results indicated that the meal pattern of the rats was very sensitive to external environmental factors. Meal frequency decreased and meal size and duration increased when animals were required to press a bar to obtain the food. Although the meal frequency generally decreased with the greater required barpresses, meal frequency was greater on the diluted diet and less on the concentrated diet. It appears that rats can adapt to different feeding patterns depending on caloric density and work load in acquiring food in order to maintain energy balance (18).

Feeding is under the control of a dual neural mechanism in which the lateral hypothalamus (feeding center) initiates feeding and the VMH (satiety center) inhibits feeding (19). Electrical stimulation in certain areas of the brain have been shown to reinforce feeding activity while in other areas, stimulation results in feeding aversion. The lateral hypothalamus contains the reinforcing region while the VMH contains the aversion region. It appears that a correlation may exist between feeding and positive reinforcement (19). Hoebel and Teitelbaum (19) showed that when animals were hungry, lateral hypothalamic stimulation was more

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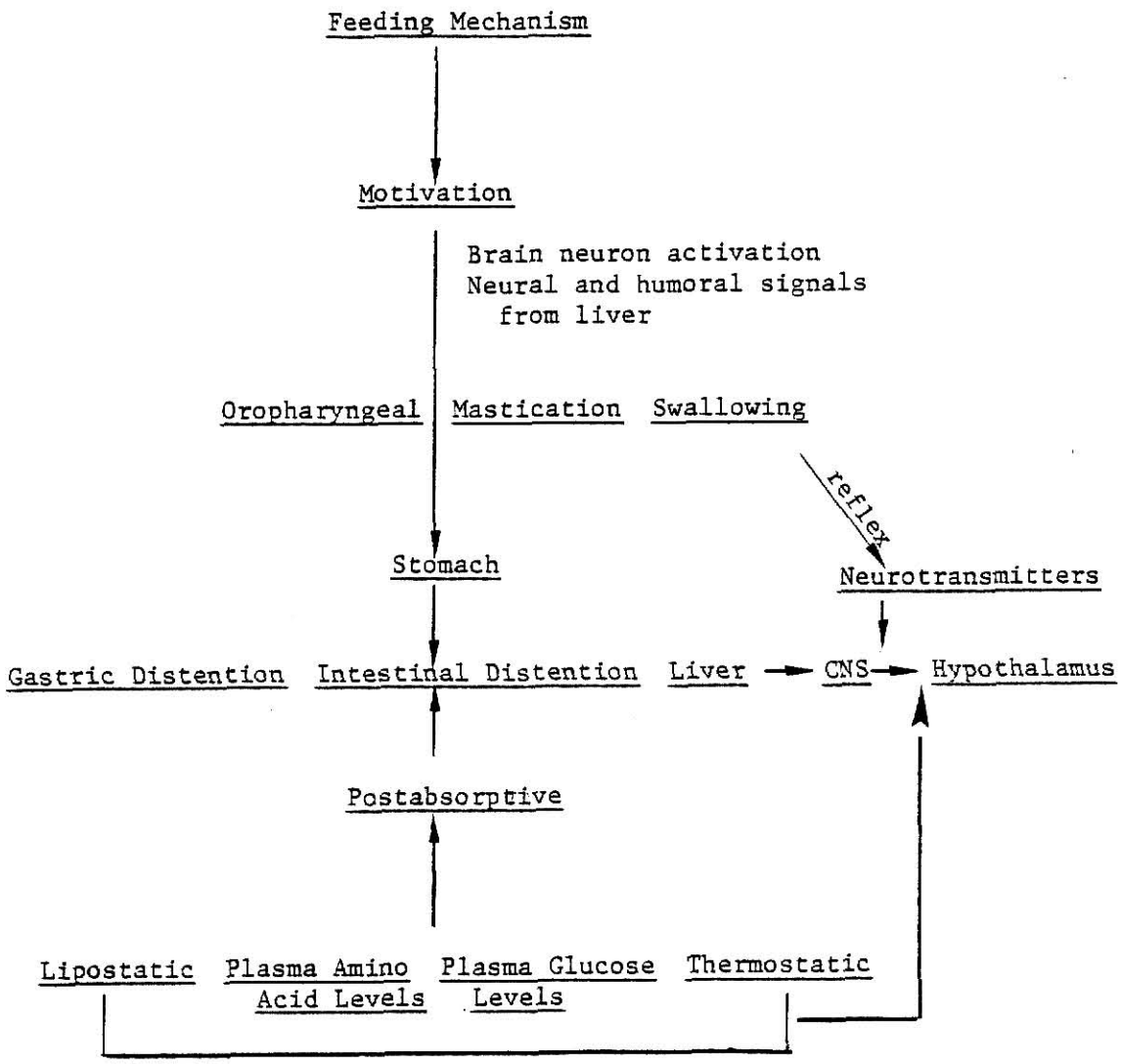

Fig. 1 Summary of factors influencing food intake.

reinforcing, however when satiated, stimulation was less reinforcing. They concluded that perhaps the pleasure of lateral hypothalamic stimulation is similar to the gratification obtained from eating. Those results showed that animals may control their food intake because of the rewards or reinforcements associated with feeding.

The variety of factors that influence feeding behavior and the theories proposed to account for them make it difficult to accept any single explanation. It is probable that food intake is a result of many complex factors and influences. Figure 1 summarizes many of these influences.

II. Studies by Jean Mayer and Coworkers: Effects of Exercise on Energy Intake and Body Weight

Exercise is usually not popular among obese individuals. Perhaps one reason is that they fear that exercise will stimulate their appetite and result in increased food intake. This is not always true, particularly with individuals who are usually sedentary.

Rat Study. Mayer et al. (20) demonstrated that light activity may actually result in reduced food intake. In their study, 21 adult female rats similar in age and weight were housed in individual cages and fed a stock diet and water ad libitum. Rats were then assigned to one of 10 groups varying in the length of time exercised. Rats were exercised either 0, 20, 40, 60, 120, 240, 300, 360, 420, or 480 minutes/day at 1.0 mph on a motor-driven zero-grade treadmill. The rats were exercised 5 days/week for 8 weeks, once in the morning and once in the afternoon. During each exercise period they were run 10 minutes alternating with 5 minutes of rest until the exercise sessions were completed. Prior to the

study all animals were adjusted to the treadmill by a preliminary training program of 15 days with daily short-term (3 minute) running periods.

The effect of exercise on food intake and body weight in the study by Mayer et al. is shown in figure 2. Rats that were exercised between 1 to 6 hours had a voluntary food intake proportional to the amount of time exercised. Body weights were similar among those groups suggesting that the energy intake was equal to the energy expended by the exercise. Rats exercised more than 6 hours had lower food intakes and body weights than animals exercised 5 hours, suggesting that beyond this point exhaustion occurred. Perhaps the most important finding was that rats exercised 20-60 minutes tended to consume less food and weigh less than rats which were confined to their cages. This suggests that light exercise does not stimulate food intake but may actually depress it.

Human Study. After performing the rat study, Mayer et al. (21) were interested in seeing whether these experimental results could be extrapolated to humans. The researchers studied 213 male workers in a West Bengal jute factory who were similar in height, weighing 67-198 pounds and were in good health.

Estimates of food intakes were obtained by dietary interviews and questionnaires. Repeated interviews and cross checks on individuals chosen at random gave good agreement of their general food intake. The nutrient composition of food consumed by the subjects was calculated using the Indian Food Composition Table for fat, protein, carbohydrate, calories, thiamin, riboflavin, niacin, and vitamin C. Other additional items of information obtained were religion and caste, income, educational levels, outstanding loans, amount spent on food, and number of dependents.

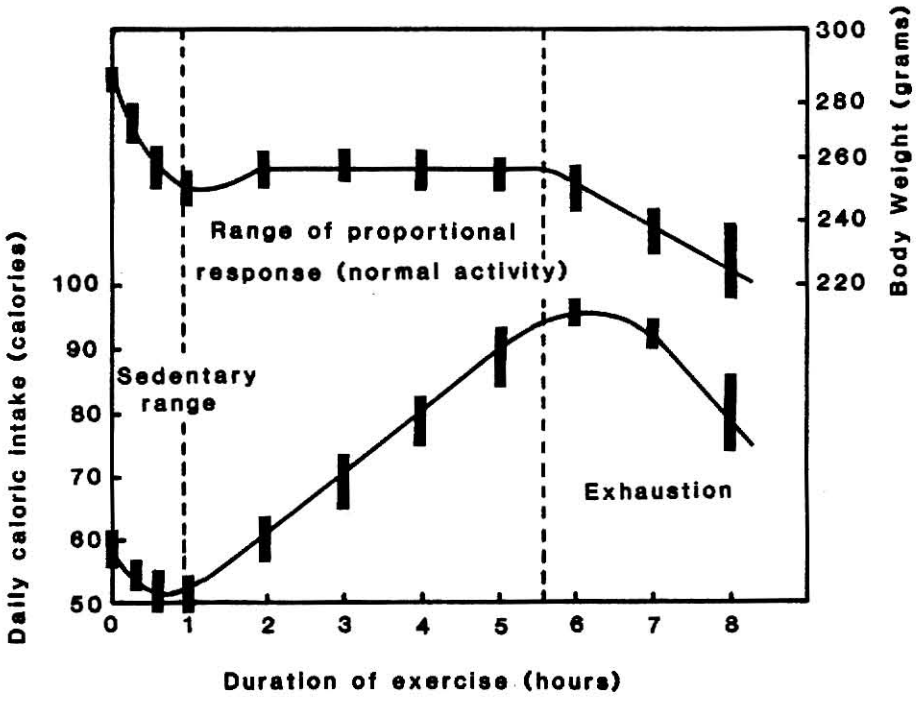

Fig. 2 Food intake and body weight as a function of exercise duration in normal adult rats, Mayer et al. (20).

The subjects were engaged in 15 different occupations in the factory, all varying in the degree of physical activity. These occupations were classified into 5 groups (sedentary, light work, medium work, heavy work, and very heavy work) according to physical demands. According to this classification, supervisors represented the sedentary workers, and clerks and mechanics represented light workers. Medium work characterized the drivers, heavy work was represented by millwaste carriers, and very heavy work was represented by the blacksmiths.

The relationship between caloric intake and occupation is shown in figure 3. Mayer et al. noted that the individuals who had the most sedentary jobs consumed more calories than those in jobs demanding light and medium work loads, and almost as many calories as those performing very heavy work. A comparison of body weights for each work load demonstrates that individuals with sedentary jobs tended to have heavier body weights than all occupations characterized by higher levels of activity.

Mayer and coworkers thus found striking similarities between results obtained in the rodent study and the human study. In both studies subjects undergoing light exercise had lower food intakes and body weights than the sedentary control groups. As exercise was further increased, food intake was proportional to the amount of exercise.

III. Methods of Exercising Animals

Swimming. Swimming has several advantages over treadmill running (22). Swimming requires simple and inexpensive equipment and generally, little training is required since rodents can swim naturally. The animals are highly motivated to exercise because if they did not swim they would drown. This motivation is believed to assure a high level of performance (23).

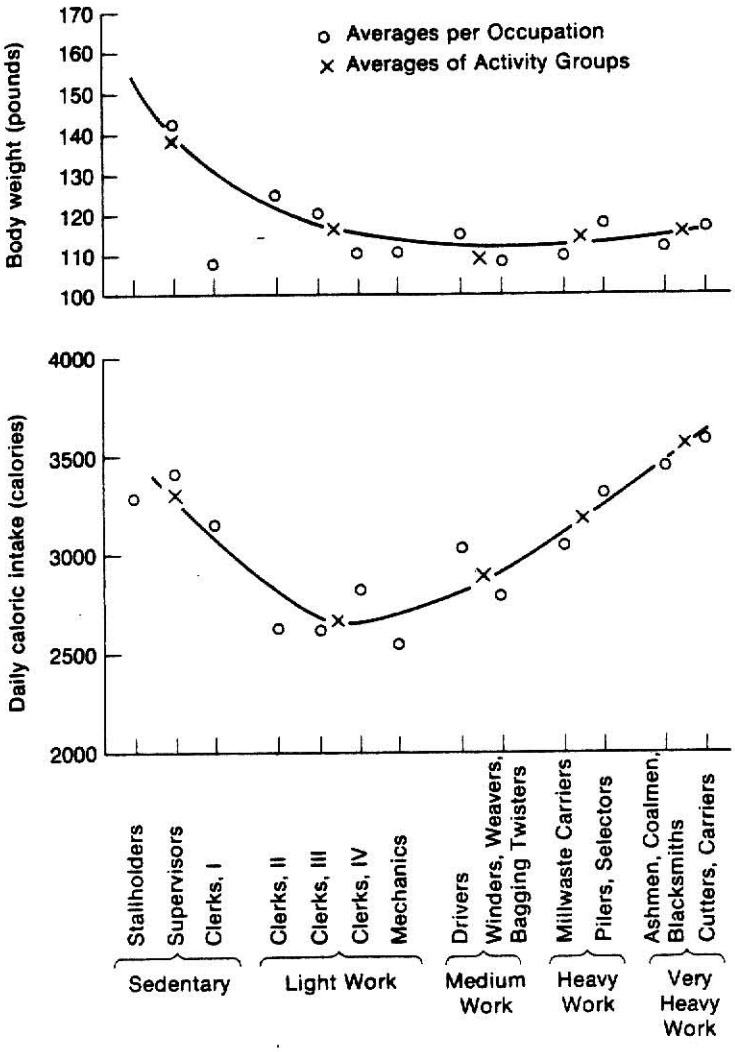

Fig. 3 Body weight and caloric intake as a function of physical activity (occupation) in humans, Mayer et al.(21).

Swimming used to test performance can be categorized as either endurance swimming to exhaustion, speed swimming, or maze swimming (23). In endurance swimming the animals swim with no escape route until exhaustion occurs. In speed swimming, the rat is placed in water a certain distance from an escape route. The maze swimming procedure is similar to the speed swim except the animals must maneuver obstacles before escaping from the water.

There are several factors affecting swimming performance. Water temperature must be regulated, because data indicate that the duration of the swim for all animals is maximal when the water temperature is at or near body temperature (22). One means of standardizing work load and reducing swim time involves attaching weights to the animals. Oscai and Molé (23) recommends that weights of 1-2% of body weight be attached to increase work load. Another source of variation in swimming occurs when air bubbles get trapped in the fur, which increases the buoyancy of the animals and reduces work load. Some investigators add a surface active agent or detergent to the water while others allow running tap water to stand for several hours before exercising animals (22).

Treadmill. Treadmill running is another popular method of exercising small lab animals (23,24,25) although one problem associated with this method is that some animals refuse to run. Such animals may require some form of motivation such as an electric shock system or jets of air (23), or treadmill accidents may result such as pinched tails and limbs. Untrained animals may be initially subjected to very mild exercise and then progressively increased work load levels by increasing the speed and/or duration of the treadmill belt. Increasing the slope of the treadmill also increases the workload for the animals. Once the final

workload is reached, the animals can be maintained at that level until they are sacrificed.

Other Methods. Less common methods of running animals include a live-in exercise drum (26), controlled running wheel (27), and a ladder ergometer for small animals (28). In exercise studies, it is useful to know how much stress the animals are exposed to and degree of training effect. In growing male rats, the amount of stress can be estimated by measuring the rate of weight gain compared to control animals (23). A dramatic weight loss in exercised animals may suggest they are over-stressed (23). Work capacity of animals can be evaluated by exercising them until exhaustion occurs and then compare selected measurements to those of a control group. For example, one measure of exhaustion in swimming or animals run on a treadmill is their inability to turn right side up when they are finished exercising (23).

IV. Other Selected Studies: Effect of Exercise on Food Intake, Body Weight, and Body Composition

Exercise and Food Intake. Data from several studies indicated that endurance exercise had an appetite suppressing effect on male rats (29-33), while others showed that food intakes of exercised rats are similar to those of sedentary control rats (31,34-36). A comparative summary of these studies is shown in table 1 for treadmill exercise and table 2 for swimming exercise. Katch et al. (32) reported that food consumption was depressed immediately following treadmill running in rats, and that a similar trend remained throughout the 24-hour period. This was particularly true for animals exercised at a high intensity level.

Dohm et al. (29) reported that male rats subjected to three different intensities of treadmill running (range of speed) all exhibited a

TABLE 1

Comparison of studies involving treadmill exercise of rats

Investigators	Speed m/min	Duration	Inclination %	Length of study wks	Exercise schedule days/wk	Food intake %[1]	body weight %[1]	Age days	Strain	Sex
Katch (32)	50.0	1 hr	0	2	5	-12	-7	-	Sprague-Dawley	M
Dohm (29)	20.0	1 hr	0	6	6	-12	-10	-	Holtzman	M
Taylor (33)	26.8	1 hr	0	16	5	-17	-23	42-56	Wistar	M
Dohm (29)	27.0	1 hr	0	6	6	-13	-15	-	Holtzman	M
Crews (30)	30.0	40-60 min	0	12	5	-20	-27	49	Wistar	M
Dohm (29)	35.0	1 hr	0	6	6	-9	-13	-	Holtzman	M
Deb (44)	26.8	1 hr	2	11	5	-4	-1	56	Zucker lean	M
Askew (37)	29.5	1 hr	8	12	5	-19	-25	45.5	Carworth CFN	M
Gleeson (51)	15.0	1 hr	8	8	7	+5	-11	-	Wistar	M
Askew (37)	29.5	2 hr	8	12	5	-12	-23	49	Carworth CFN	M
Oscai (50)	31.0	2 hr	8	12	7	-	-31.5	42	Wistar	M
Askew (46)	29.5	2 hr	8	13	5	-	-29	35	Carworth CFN	M
Askew (46)	35.0	1 hr	7.5	6	5	-	-11	35	Sprague-Dawley	M
Owens (34)	22.0	45 min	9	12	5	No change	+1.2	90	Long Evans	M
Katch (32)	16.0	50 min	10	2	5	-18	-7	-	Sprague-Dawley	M

[1] Percent food intake and body weight values are calculated by subtracting the mean of the control animals from the mean of the exercised animal ÷ by the mean of the control animals × 100.

TABLE 2

Comparison of studies involving swimming exercise of rats

Investigator		Duration	Additional workload[1]	Length of study	Exercise schedule	Food intake	Body weight	Age	Strain	Sex
		min	%	wks	days/wk	%[2]	%[2]	days		
Oscai	(50)	30	-	6	5	-	-5.6	70	Wistar	M
Oscai	(50)	60	-	6	5	-	-2.8	70	Wistar	M
Stevenson	(31)	60	-	4	4	-15	-	-	Sprague-Dawley	M
Lau	(35)	60	-	3	5	No change	-10	-	BHE	M
Oscai	(43)	120	1	18	5	-11	-39	42	Wistar	M
Stevenson	(31)	120	-	4	4	-10	-	-	Sprague-Dawley	M
Lau	(35)	120	-	6	5	-8.5	-14	-	BHE	M
Walberg	(38)	120	4	8	5	↑[3]	↓[3]	49	Zucker lean	F
Oscai	(50)	180	-	6	5	-	-7	70	Wistar	M
Stevenson	(31)	240	-	4	4	No change	-	-	Sprague-Dawley	M
Oscai	(36)	360	-	27	6	No change	↓[3]	5	Wistar	M
Oscai	(47)	360	-	12-14	6	No change	↓[3]	8	Wistar	M
Oscai	(40)	360	-	4½	6	+18	-4	70	Wistar	F
Oscai	(40)	360	-	4½	6	-	No change	42	Wistar	F
Oscai	(40)	360	-	4½	6	+2.3	-25	42	Wistar	M
Jones	(42)	360	3.5	15	6	-	↓[3]	91	Sprague-Dawley	M
Oscai	(39)	360	-	21	5	+31	-2	42	Wistar	F

[1] Weights were attached to the animals in these studies. Values indicate the added weight as a percent by body weight. [2] Percent food intake and body weight values are calculated by subtracting the mean of the control animals from the mean of the exercised animals ÷ by the mean of the control animals × 100. [3] Indicates trend shown by histograms. Actual number could not be calculated.

depression in food consumption when compared to controls. However there were no significant differences in food intake between rats in the three different intensity groups.

The effect of exercise duration on food intake also has been studied. Askew et al. (37) exercised male rats at the same intensity but at two different durations. Animals in both one hour and two hour exercise groups consumed less food than control animals, and rats in the two hour group consumed more food than those in the one hour group. In contrast, Stevenson et al. (31) swam male rats for 60, 120, 240 min/day. They found that rats in the 240 minute group had no change in food intake but those exercised 60 and 120 minutes exhibited a reduction in food intake. Lau et al. (35) reported no change in food intake in male rats swimming for 60 min/day while depression in food intake was seen in rats subjected to 2 hours of daily swimming. Lack of agreement between these studies reflects possible differences in experimental procedures and techniques as well as strain and age differences of the rats. However, the underlying trend seems to be a reduction in food intake by exercise.

Female rats respond differently to regular forced exercise than male rats. Walberg et al. (38) reported that lean female Zucker rats subjected to a 2 hour daily swim, had higher food intakes than control animals. Oscai et al. (39) reported that Wistar female rats increased their food intake approximately 31% when subjected to 6 hours of swimming. In another study (40) female rats swum 6 hours consumed 18% more food than the control rats but exercised male rats had food consumption similar to that of control rats. So the effect of exercise on food intake apparently is different in female rats than it is for male rats.

The involvement of the sex-specific gonadal steroids in this response is still unknown (41). It has been suggested that the differences observed in food intake between male and female rats may reflect a primary sex difference in the neural system that controls long-term energy balance (41).

Exercise and Body Weight. Numerous workers (29,30,32) have demonstrated that male rats subjected to endurance exercise gain less weight and have lower final body weights than sedentary freely-eating controls. The smaller weight gain observed in exercised male rats is presumably a result of an increase in caloric expenditure not compensated by proportional increase in caloric intake (30,36).

Lau et al. (35) reported that male rats subjected to 1 or 2 hours of swimming 5 days/week lost weight, but the same swimmers regained lost weight over the 2 day resting period which followed. The rate of weight gain over the resting period was somewhat greater for the swimmers than the control rats, but the differences were not significant.

In contrast to male animals, female rats subjected to regular exercise gained weight at approximately the same rate as sedentary control rats (38-40). Oscai et al. (39,40) demonstrated that female Wistar rats on a daily 6 hour swimming program maintained body weights similar to those of sedentary control rats. The maintenance of body weights by these animals was attributed to an increase in food intake proportional to the amount of exercise.

Exercise and Body Composition. The body composition of vertebrates includes several components such as protein, fat, water, glycogen, vitamins and minerals. The effect of exercise on these components has been studied, particularly its effect on body fat.

Jones et al. (42) found that the lower final body weights of exercising male rats as compared to those of sedentary rats could be attributed almost entirely to their lower fat content. Oscai and Holloszy (43) reported that 78% of the weight loss for exercising rats was a loss of body fat, or one third less body fat than those in the control group. Similar results have been shown by other investigators (29,38).

Stevenson et al. (31) reported that there was an inverse relationship between exercise duration and body fat. That is, as exercise duration was increased, body fat content tended to be decreased. In their rat study, the control group had 12.8% body fat while those in the 240 minute and 360 minute groups had 8.5% and 8.4% body fat, respectively.

Dohm et al. (29) studied the effect of different intensities of exercise on body fat. Rats were trained on three treadmill running speeds: 20 m/min, 27 m/min, and 35 m/min for 1 hour/day. The results of this study showed that exercising male rats daily resulted in 6%-7% body fat compared to 12% body fat for the control group, however, increasing the intensity by 70% caused no further reduction in body fat. Others have found that exercise reduces body fat. Deb and Martin (44) studied the effect of exercise on body composition in male lean and obese Zucker rats subjected to a moderate treadmill running program. Exercise resulted in 4.9% body fat in the lean rats compared to 6.4% for the control rats, while exercise resulted in 27.7% body fat in the obese rats compared to 39.0% body fat in the obese control rats. Walberg et al. (38) observed the effect of exercise on body composition in female lean and obese Zucker rats. Both groups when exercised had lower body fat contents than their sedentary freely-eating controls. Oscai et al. (39) reported that female

Wistar rats subjected to 6 hours of swimming had 9% body fat while sedentary control rats had 17% body fat.

The finding that the exercised rats are leaner than sedentary control rats suggests a lipid-mobilization effect of exercise (43). During prolonged exercise there is a stimulation of the sympathetic nervous system which may be responsible in part for the increased lipolysis and consequently, the increased fat loss.

Researchers also have examined the effect of exercise in specific fat depots. An example of a fat deposit commonly studied is the epididymal fat pad (EFP) surrounding the testis in male animals. The amount of fat stored in each depot is dependent on both the size and number of adipose cells. Factors influencing adipose cell number and size have been studied. Growth of EFP in rats is attributed to an increase in cell number and cell size. However cell proliferation ceases in rats between 15-28 weeks of age (36). Oscai (36,45,47) subjected 5 to 7-day-old rats to a 6 hours/day swimming program. The study was terminated at two different times, when the male rats reached 15 weeks (47) and 28 weeks (36,45). In these studies, exercised rats exhibited a reduction in the size and lipid content of the EFP when compared to sedentary-freely-eating control animals. The smaller EFP in the exercised rats was attributed to decrease in both total number of cells and size of cells. Furthermore because exercise early in life (less than 28 weeks) was shown to be effective in significantly reducing the rate of accumulation of cells in EFP of rats, the lower level of body fat were maintained in later life (36,45).

Askew et al. (46) subjected male rats to a strenuous treadmill running program for 2 hours/day 5 days/week for 13 weeks. Exercise did not affect the number of adipocytes per EFP in contrast to Oscai's studies. The

results may be attributed to an older age of the rats (5 weeks) at the onset of the exercise training program. The rats were possibly past the stage of the most marked proliferation of fat cells and consequently exercise may have exerted a lesser effect. In another study Askew et al. (37) subjected 6 1/2 week-old rats to two different treadmill running programs. One was designated as high intensity exercise, and the other as moderate intensity exercise. After 12 weeks of exercise, rats in both groups had EFP weighing 50% less than those of control animals. Exercised rats also had a smaller number and smaller size of adipocytes than freely-eating control rats.

Lau et al. (35) swam adult BHE rats for 1 hour/day for 3 weeks followed by 2 hours/day for another 3 weeks (5 days/week). There were no changes in adipose tissue in response to 1 hour of swimming at 3 weeks, however when swimming was increased to 2 hours/day (additional 3 weeks), weights of EFP and retroperitoneal depots of the swimmers were 50% lower than those in control animals. The lower fat depot weights observed in the 2 hour group were attributed to smaller cell size but not cell number. Data from this study indicate that adipose depot is affected by exercise duration.

Burowrecki et al. (48) swam adult male and female Wistar rats for 2-3 days/week with loaded weights corresponding to approximately 2% of their body weight. The exercise program lasted 7-11 weeks. Exercised male rats weighed 20% less and had EFP weighing 50-55% less than control rats. Total triglyceride content in fat pads and triglyceride content per adipocyte were smaller in exercised rats, however the number of adipocytes present in the two EFP was not significantly affected by exercise. Female rats exhibited a different response to exercise. Differences in

body weights of female controls and female exercised rats were much smaller (5.3%) than those of male rats. The parametrial fat pad weight, triglyceride content per adipocyte, and adipocyte diameter were all significantly smaller in exercised rats. Exercised female rats also had a smaller number of adipocytes than the control female rats.

The percent body protein and water in response to exercise also has been investigated. Walberg et al. (38) reported female lean Zucker rats subjected to 2 hours of swimming had total body protein contents similar to those of control rats. Percent body water was slightly higher for rats in the exercised group compared to sedentary control rats. Similar results were reported by Deb and Martin (44) who found that male lean Zucker rats exercised for 1 hour had slightly, but not significantly, higher body protein content and body water than sedentary control rats.

Jones et al. (42) reported that male rats subjected to a strenuous running program had an average total body protein content of 19.8% and 62.5% water content compared to an average 18.2% protein and 56.7% water content for the sedentary control rats. Crews et al. (30) ran male Wistar rats for 40-60 min/day and found that exercised rats had 18% protein compared to 15% protein found in the sedentary freely-eating controls. They suggested that exercise may promote considerable protection against the loss of protein, and that the fat-mobilizing effect of exercise could result in a greater availability of calories for the synthesis of lean tissue.

In contrast to the other studies mentioned, Dohm et al. (29) reported that endurance training increased amino acid catabolism in rats. Protein levels of muscle and liver were significantly decreased by an exhaustive bout of exercise. The results of this study suggests that

protein is not spared but rather broken down at an increased rate during intense exercise, and the amino acids released could be available for energy.

The cardiac muscle and its adaptation to endurance exercise has interested many investigators. The only consistently observed cardiac adaptation to prolonged exercise is an increase in the ratio of heart weight to body weight (40,49,50).

An increase in the size of the heart relative to the body weight can play an important role in the increased work capacity caused by training. In the absence of cardiovascular disease, there seems to be a positive correlation between heart size and maximum cardiac output (50). An increase in heart weight to body weight ratio could be associated with an increase in the maximum capacity to deliver blood to the working muscle. Oscai et al. (40) demonstrated that female rats subjected to 6 hours of swimming had hearts weighing 22-28% more than those of control rats even though they had similar gains in body weight.

Hickson et al. (49) swam adult female rats for three 2 hour sessions/day 7 days/week. Exercised rats had significantly heavier heart weights than control rats after only 2 days of swimming. During the 28-day study, all of the observed differences in heart weights were attained during the first 14 days of the study. After 14 days, exercised rats had hearts that were approximately 30% heavier than controls. This study further showed that if animals ceased to exercise, heart weights decreased rapidly during the first 7 days losing about 60% of the cardiac hypertrophy.

In male swimmers, two variables seem to affect heart weight. These are the level of physical activity and body weight (50). This is particularly important because male exercised rats have significantly lower final

body weights than the sedentary freely-eating controls which in turn lead to differences in heart weight. Oscai et al. (40,50) reported that the hearts of male rat swimmers were lighter than those of the sedentary freely-eating animals, but significantly heavier than those of sedentary pair-weighted rats.

V. Effect of Exercise on Hormone Metabolism and Energy Utilization

One of the most important factors in directing and coordinating the metabolism of different body tissues is the influence of the endocrine glands. Endocrine glands produce their effects through production of hormones, which are carried by the blood throughout the whole body. The hormones secreted by these ductless glands may then act on target tissue cells by either influencing the rate of transport of the substance across a cell membrane or by affecting enzyme activity or synthesis within the cell (52).

Considerable information on the endocrine response to exercise has become available in recent years. Much of the information identifies endocrine response either during or after exercise. Some studies have shown specific changes in physiological response to exercise. Other studies have presented the possible physiological and biochemical significance of hormonal changes with exercise. Often the exact hormonal response to exercise cannot be identified because of inadequate measurement techniques used, experimental design, exercise conditions and/or the complexity of the endocrine system. Although much knowledge relating exercise to hormonal response has become available in recent years, further research is needed to uncover potentially important hormonal influences.

Perhaps the most important hormones involved in maintaining homeostasis of the body cells during exercise are cortisol, androgens, growth hormone (GH), thyroxine, insulin, glucagon, and catecholamines (52). These hormones will be discussed individually with particular emphasis on their response to exercise and their effect on energy utilization. Although most studies involve humans, several involve the effect of exercise in animals.

Androgens. There are many naturally produced hormones which have masculinizing effects but the most important are testosterone and androstenedione (52). In men, testosterone, secreted mainly by the testis, is released at a rate of 5-10 mg/day and androstenedione at 1-2 mg/day (53). The release rate for women, mainly from the ovaries, is less than 0.1 mg/day of testosterone and 2.4 mg/day of androstenedione (53). The higher levels of testosterone in men may partially explain why male athletes have greater muscle force and power than women athletes. Testosterone has been shown to have an enhancing effect on body structure, lean body mass (52) muscle glycogen and protein synthesis (53). Also androgens are mainly responsible for secondary sex characteristics (52).

With the development of sensitive techniques for measuring testosterone, serum androgen levels have been shown to respond to both exercise intensity and duration (54,55). Dohm et al. (54) subjected male Holtzman rats to a 1 hour daily treadmill running program for 6 weeks. A slight increase in testosterone levels was reported after 1 hour of exercise followed by a decline to less than 50% of the pre-exercise value 6 hours after exercise. The most dramatic decrease (75%) in testosterone was seen in rats immediately following an exhaustive exercise run.

Galbo et al. (55) reported that men subjected to short bouts of mild to maximum treadmill running exhibited no change in plasma testosterone levels. However, a significant rise in testosterone was found after 40 minutes of a prolonged exercise. The increase in testosterone levels was diminished during the latter part of the exhaustive run.

Dramatic increases in total plasma androgen levels after 20-90 minutes of strenuous maximal physical activity was reported in world class rowers and swimmers (53). In contrast Lamb et al. (53) studied plasma testosterone in untrained young males before and after a bicycle ergometer program. No significant changes in plasma testosterone were reported after the exercise.

The discrepancies found in the human studies may be a result of differences in physical conditioning and genetic background of the subjects, measurement of androgen levels, or workloads used by each laboratory. Further studies need to be carried out to determine the effect of exercise on testosterone metabolism.

Growth Hormone (GH). Growth hormone is secreted by the hypothalamus-pituitary gland system (52). A release of GH is stimulated by low plasma glucose concentration, exercise and heat exposure. Two of the best known actions of growth hormone are control of normal growth and regulation of protein metabolism (56). Growth hormone also increases the catabolism of adipose tissue for energy use and increases blood glucose level (52).

Exercise increases the release of GH but this increase is not proportional to increased work load (intensity) (52). Hartley (56) demonstrated that GH levels of men were significantly higher during graded bicycle exercise than at rest after 40 minutes of work at modern intensity. Mild

or maximal intensities of work did not increase the GH levels. There is no apparent explanation for this finding.

Sutton (57) subjected fit and unfit men to a cycle ergometer for 20 minutes at 85% of maximal oxygen uptake. Serum GH levels were similar in both groups at rest. There was no significant change in GH levels during exercise seen in the fit men, however a five-fold increase was seen in the unfit men. When the two groups were compared, GH levels during and following exercise were significantly elevated in the unfit men.

Glucocorticoids. The adrenal glands are two small highly vascular organs located at the upper poles of the kidneys. Two parts are distinguishable, the cortex and medulla, but they are different embryonically, histologically and functionally.

The adrenal cortex is stimulated by the hypothalamus-pituitary system to secrete cortisol. Although the changes in circulating glucocorticoids are not well understood, the basic metabolic actions have been determined. Glucocorticoids increase glycogen deposition in the liver, enhance liver glyconeogenesis, stimulate lipolysis, and act as anti-inflammatory agents (52). Cortisol stimulates the production of glucose from amino acids (protein) in the liver for use in energy or storage as glycogen.

The circulating levels of glucocorticoids are influenced by many factors including psychological and physical stress. Consequently, these factors may influence glucocorticoid responses in exercise studies.

Plasma cortisol levels are measured in most studies concerned with the effect of exercise on glucocorticoid metabolism. The plasma cortisol levels provide limited information, however, because those levels can be modified by the rate of cortisol synthesis, ability to bind with plasma proteins, removal rate, degradation and excretion (58). Hartley et al. (59)

reported a marked increase in plasma cortisol levels immediately prior to the onset of preliminary bicycle exercise program in men. A similar increase was not seen following training. Sutton (57) found plasma cortisol levels similar in the basal state of both fit and unfit men but following exercise it was increased in the unfit men and remained elevated for 50 minutes. This is possibly due to the greater ease in performing the task after training and also to the psychological ease of being familiar with the stress.

Catecholamines. Catecholamines, specifically norepinephrine (NOREP) and epinephrine (EPIN), are secreted by the nerve endings of the sympathetic nervous system. Some of these nerve endings are located on the heart, blood vessels, and the adrenal medulla (52). NOREP and EPIN have important metabolic actions, including mobilization of free fatty acids from adipose tissue and the catabolism of glycogen from the liver into glucose as an important energy source during exercise (52). NOREP and EPIN also have actions on the cardiovascular system by increasing heart rate, increasing force of heart contraction and increasing vasoconstriction of blood vessels (52).

Increased catecholamine secretions are stimulated by emotional and physical stress and pain but the exact mechanism are unknown. Hartley (56) suggested that during exercise, the central nervous system activates motor neurons to the skeletal muscle and sympathetic nerve fibers which simultaneously increase catecholamines.

Extensive activation of the sympathetic nerve system occurs during physical activity (56). Consequently, increases in NOREP can be found even in fairly easy work loads, but dramatic increases of two-to-six fold are found at maximal work loads (56). Hartley et al. (59) studied NOREP

and EPIN in normal men before and after intense bicycle exercise. NOREP was increased proportional to increased work intensity. Increased EPIN, however, was found only at maximal work intensity.

In another study, Hartley (56) reported that NOREP levels almost doubled after the first 40 minutes of exercise but there was no further change at exhaustion. Following a 6 week program of moderate endurance training plasma levels of NOREP were 50% of those seen before training.

Insulin and Glucagon. Insulin and glucagon are hormones secreted by the pancreas. Insulin secretion is stimulated by a high plasma glucose concentration while that of glucagon is stimulated by low plasma glucose. Glucagon increases glucose output from the liver by glycogenolysis (52). Insulin facilitates the transport of glucose and amino acids to the cell membrane. Insulin also increases the conversion of glucose to glycogen and the conversion of glucose into fat thus facilitating fat storage. Insulin also aids in the transport of amino acids into cells thus permitting protein synthesis (52).

During exercise, plasma glucagon increases and plasma insulin decreases while plasma glucose concentration does not change markedly but glucose turnover increases presumably because some glucose is taken up by exercising muscles (59). Hartley et al. (59) found that men subjected to exercise on a bicycle exhibited a drop in insulin levels during the first 40 minutes of exercise and remained depressed at exhaustion and during the first hour of recovery.

Thyroxine. A major function of thyroxine is that of regulating cellular energy transfer. Thyroid activity is under the influence of thyroid-stimulating hormone (TSH) secreted by the anterior lobe of the

pituitary gland, and is a feedback mechanism triggered by circulating thyroid hormone level (52).

The thyroid hormones have many important functions. The hormone accelerates cellular reactions in nearly all cells of the body resulting in increased oxygen consumption and basal metabolic rate. The hormones affect growth, rate of enzymatic reactions, and protein synthesis (60). These known functions have prompted many studies involving the effect of physical activity on thyroxine (T_4) concentration.

Exercise has been found to influence many thyroid hormone responses. Increased T_4 turnover first was noted in racehorses by Irvine (61). Irvine reported that racehorses subjected to 12 weeks of exercise exhibited increased T_4 secretion. In another study, Irvine (62) found greater T_4 turnover in male athletes than nonathletes. Athletes in moderately severe training had a thyroid degradation/secretion rate 75% above that of resting nonathletes. This rate, however, fell significantly after 3 days rest. It was concluded that exercise increases the rate of T_4 degradation in humans since the T_4 degradation was greater in athletes during training. The T_4 degradation rate decreased in athletes following termination of the exercise and the T_4 degradation rate increased in the sedentary non-athletes with exercise.

Wirth et al. (63) reported that rats subjected to a single bout of treadmill running exhibited an increase in plasma triidodthyronine (T_3), thyroxine (T_4), and triiodothronine/reverse triiodothronine ($T_3/_rT_3$) concentrations. However in trained rats, no change in these measurements were observed.

Several conclusions about thyroid hormones in response to exercise have been reviewed by Terjung and Winder (64). Exercise seems to enhance

the rate of utilization or disposal of thyroxine, with changes observed in as little as 6 days. The liver seems to be the major site for the increased uptake of T_4 from the blood. The concentration of T_4 in the muscle is not influenced by exercise. The enhanced T_4 degradation is not due to increased deiodinating enzymes in the liver, kidney or muscle but may be due to an increase in circulating free T_4 levels. Plasma levels of TSH were not found to be altered during or after exercise.

The changing plasma levels of these endocrine hormones which occur during exercise serve important functions. For example, the increase in glucagon and decrease in plasma insulin levels in response to exercise influence glucose metabolism. Increased levels of glucagon result in enhanced glucose output from the liver by glycogenolysis. Low plasma levels of insulin result in decreased conversion of glucose to glycogen, decreased conversion of glucose into fat, and a reduction in amino acid transport into cells (52). Increases in NOREP levels using mild-to-maximum work loads and increased plasma levels of EPIN during maximal work intensity stimulate mobilization of free fatty acids from adipose tissue and enhance the rate of glycogen breakdown to glucose in the liver (52). Elevated plasma GH levels during moderate work loads suggest GH may have an enhancing effect on lipolysis for energy use, as well as increasing blood glucose levels and protein synthesis (52). Elevated plasma androgen levels observed in some laboratories suggest that these hormones exert an enhancing effect on body structure, lean body mass as well as muscle glycogen and protein synthesis (52). Increased plasma cortisol levels found in untrained individuals may enhance liver gluconeogenesis, lipolysis and result in an inhibition of lipid synthesis in adipose tissue

(52). Elevated T_4 levels in exercising subjects enhance mobilization of free fatty acids from adipose tissue (52).

Although exercise is known to influence the metabolism of several hormones, which in turn influence the utilization of energy, further research is needed to uncover the exact mechanism of these changes and their potential for future use.

MATERIALS AND METHODS

Animals and Their Care

Fifty-four male Swiss Albino mice[1] weighing 29.0-34.0 g (7-8 weeks of age) were used for this study. They were housed individually in polypropylene cages (10" × 6.5" × 5.0") and fed a stock diet[2] and water ad libitum throughout the study. Room conditions were maintained at (20 ± 2°) with a 12-hour light-dark cycle. The cages were arranged on both sides of a battery which was rotated weekly so that different treatment groups would have uniform exposure to variables such as light intensity, ventilation and other daily disruptions.

Mice were randomly assigned to one of six treatment groups varying in the length of time exercised. Mice were exercised 0, 20, 40, 60, 120, or 240 minutes per day at 0.27 mph on a motor-driven zero-grade treadmill.[3] The duration of daily exercise in the different treatment groups was similar to that of Mayer et al. (20) for rats. The control animals were not exercised but were momentarily placed on the treadmill to correct for any excitement the animals received during handling. Mice were identified by earpunching procedures.

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[1]Obtained from the departmental breeding colony at the Dept. Foods and Nutrition, Kansas State University, Manhattan, KS. Original breeding stock for this colony was the HAP:(ICR)BR strain obtained from Harlan Sprague Dawley, Indianapolis, IN.

[2]Purina® Rodent Laboratory Chow 5001, St. Louis, MO. This diet contains approximately 23.4% protein, 4.5% fat, 5.0% fiber, 7.3% ash, and 49.8% nitrogen-free-extract (by difference).

[3]Radiotrol Treadmill, 1/2 h.p., Boston Gears, Quincy, MA.

The exercise periods were conducted 5 days a week (Mon.-Fri.) in late morning or early afternoon hours for 8 weeks. The 8-week period was preceded by a 2-week training period (table 3), a program of progressive exercise to familiarize the animals to the treadmill. Exercise periods were conducted once daily on exercise days.

During the 10-week period, body weight and feed intake were measured weekly. For body weight, each mouse was placed in a pre-weighed container and weighed on an Ainsworth balance (Model A-200). Weekly feed intake for each mouse was calculated by subtracting residual feed left at the end of the week from the initial feed weight.

During the 10-week exercise study we lost 5 mice in treadmill accidents. Three of these mice were in the group exercised for 120 minutes, one mouse was lost in each of the groups exercised for 60 or 240 minutes. Reported data do not include results from those animals.

Sacrifice Procedures

At the end of the 10-week exercise study, the mice were sacrificed by cervical dislocation. The digestive tract from the gastroesophageal sphincter to the anus were then removed from each mouse and discarded. This was done to minimize variations in body weight and composition caused by periodic feed intake. The mouse without the digestive tract was considered for analytical purposes, the mouse carcass.

The measurements taken at sacrifice included body weight, carcass weight, heart weight, and epididymal fat pad weight. The various tissues removed for weighing were returned to the rest of the carcass. Each mouse carcass was stored in an individual quart-size labeled canning jar at -18° until later analyses.

TABLE 3

Preliminary training schedule: minutes of progressive exercise during the two-week period

Week	Day	% of Target time	Group (target time)					
			0	20	40	60	120	240
1	Mon.	(10)	0	2	4	6	12	24
	Tue.	(15)	0	3	6	9	18	36
	Wed.	(20)	0	4	8	12	24	48
	Thur.	(30)	0	6	12	18	36	72
	Fri.	(40)	0	8	16	24	48	96
2	Mon.	(40)	0	8	16	24	48	96
	Tue.	(50)	0	10	20	30	60	120
	Wed.	(60)	0	12	24	36	72	144
	Thur.	(80)	0	16	32	48	96	192
	Fri.	(100)	0	20	40	60	120	240

Preparation and Homogenization of Mouse Carcasses

The flow chart for carcass homogenization is shown in figure 4. The jars containing the carcasses were allowed to sit at room temperature for 1 hour to thaw. Four volumes (4 × carcass weight) of deionized water were added to each jar and a blade, rubber gasket, and cap were placed on the jar opening. The caps were closed tightly on each jar and then unscrewed about 1/4 turn to allow the steam to escape during autoclaving. Jars were autoclaved in batches of 18 in a stainless steel tray for one hour at 121° and 15 psi. After autoclaving, jars were cooled in a 37° water bath for one hour. The eighteen jars included at least one mouse from every treatment group.

Each jar was removed from the water bath and prepared for homogenization. To prevent leakage, 2 rubber gaskets were placed between the jar mouth and blade, and another gasket was placed between the blade and an adaptor. The adaptor was screwed onto the jar and the whole unit was fitted onto an Osterizer blender. The mouse carcass was then homogenized one minute at low speed (stir) and 4 minutes at high speed (liquify). The homogenate was immediately filtered through one layer of cheesecloth into a 500-ml erlenmeyer flask. The remaining fur trapped in the cheesecloth was pressed 35 times against the side of the funnel with a spatula to remove any residual homogenate. The fur residual was returned to the jar and 2 volumes of water was added, followed by one minute of homogenization at highest speed in the blender. The mixture was again filtered and the filtrate was added to the erlenmeyer flask. Homogenization and filtration of the fur was performed a total of 3 times so that the last washing was fairly clear with little visible trace of homogenate. The

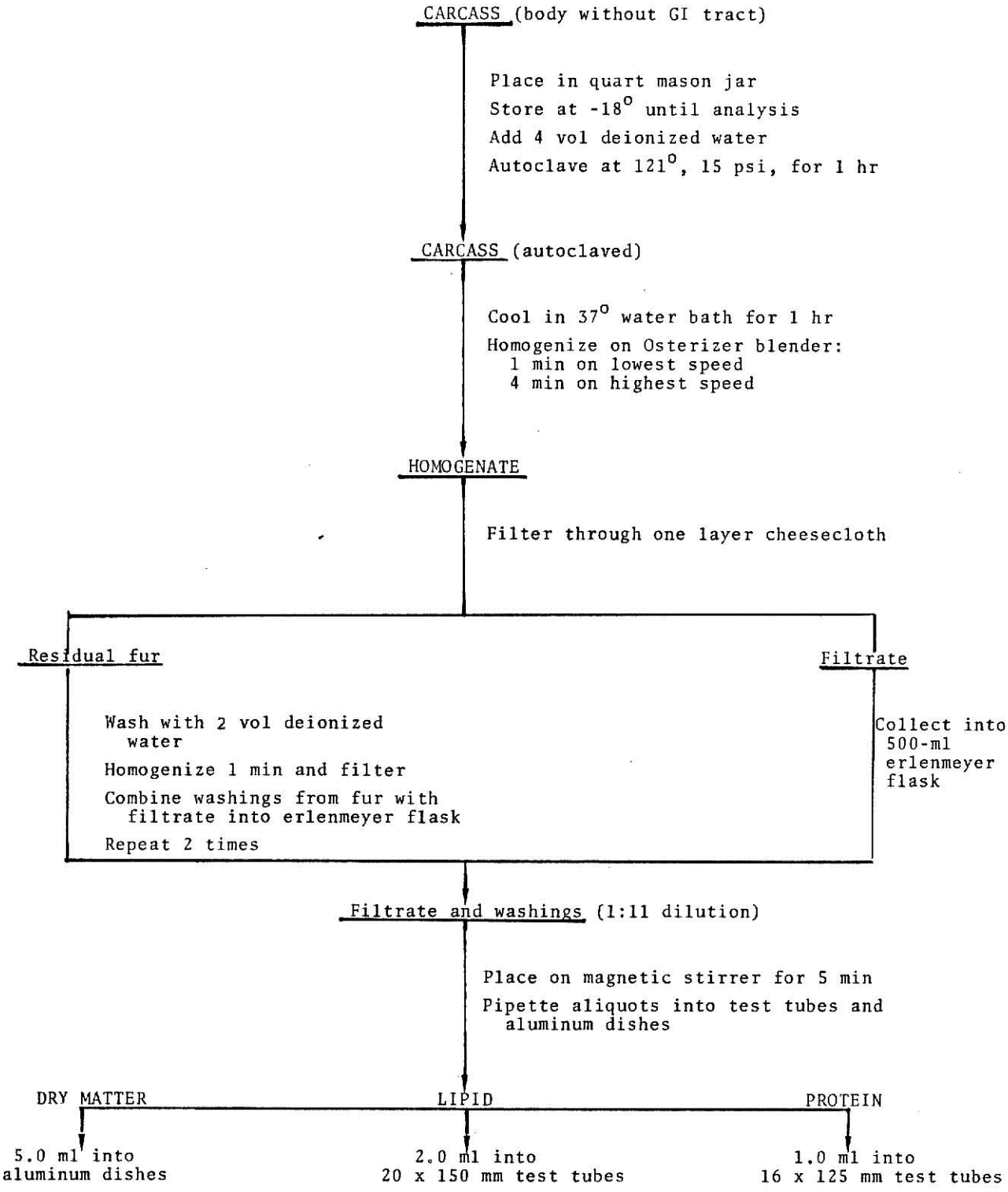

Fig.4 Preparation and Homogenization of Mouse Carcasses.

remaining fur residue, weighing approximately 1.5 grams, was discarded after homogenization and filtration.

The 500-ml erlenmeyer flask containing the homogenate (initial homogenization and 3 washings) was placed on a magnetic stirrer and was mixed for about 5 minutes to keep the homogenate in suspension. Aliquots were pipetted into prelabeled test tubes for body composition analyses. Volumes of duplicate aliquots were as follows: 5.0 ml into pre-weighed aluminum dishes for dry matter determination, 2.0 ml into 20 × 150 mm test tubes for lipid analysis and 1.0 ml into 16 × 125 mm test tubes for protein analysis. Dry matter and protein determinations were performed on the day of homogenization. Samples for lipid analysis were stored at -18° before analysis. Analytical procedures for dry matter, protein, and lipid are shown in figure 5 and are explained in greater detail in the following paragraphs.

Determination of Dry Matter

Aluminum dishes (S/P catalog #D2165) were labeled, dried 2 hours in a 103° forced-air draft oven, cooled 1 hour in a desiccator and weighed to the nearest .001 g. Dishes were then arranged in the work area and 5 ml of each homogenate was pipetted into each pre-weighed dish while the homogenate was mixing on the magnetic stirrer. The dishes containing the homogenate samples were then weighed and dried at 103° for 5 hours. Dishes were cooled in a desiccator for 40 minutes and weighed. The percent dry matter was calculated by dividing the weight of the dried homogenate by the weight of the 5 ml homogenate before drying and multiplying by a correction factor of 110. This correction factor was obtained by multiplying 11 (initial dilution for homogenization) by 100 (to obtain

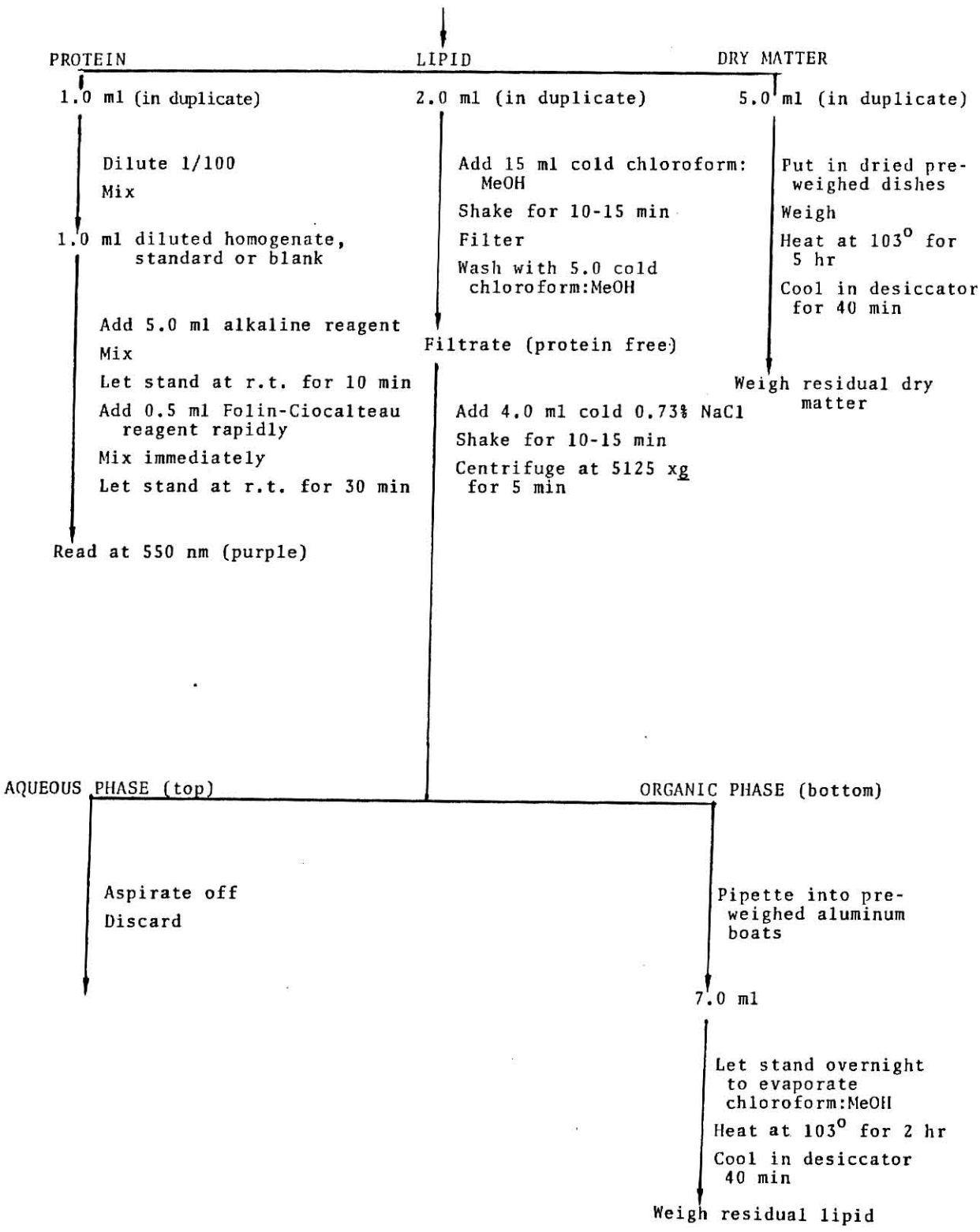

Fig. 5 Analyses for protein, lipid, and dry matter in mouse carcass homogenate.

percentage). Percent body water for each animal was calculated as 100 minus the percent dry matter.

Determination of Protein

Protein analysis was performed on 1.0 ml homogenate on the day of homogenization using the Folin-Lowry procedure (65). Each 1.0 ml sample was diluted 1/100 with distilled water and one ml of this mixture was used for further analysis. To each tube containing the diluted homogenate was added 5 ml alkaline reagent (see appendix table 1 for preparation of reagents). After mixing, each tube was allowed to stand at room temperature for 10 minutes. Then 0.5 ml Folin-Ciocalteau reagent was added rapidly and the tube contents were mixed immediately on a vortex mixer. After 30 minutes the optical density (purple color) was read at 550 nm on a spectrophotometer. Eight samples could be analyzed at a given time with a distilled water blank (for zeroing the spectrophotometer) and a protein standard solution (0.2 mg bovine serum albumin/ml). To correct for non-colored turbidity observed in each sample, a "sample blank" containing 1.0 ml diluted homogenate (1/100) was prepared and 5 ml alkaline reagent without the copper sulfate, and subtracted the absorbance reading of the "sample blank" tube from that of the "sample" tube. The resultant value was considered the corrected absorbance reading. Protein contents of each tube were determined by linear regression after comparing the corrected absorbance reading of each sample to those of the distilled water blank and protein standard. These values were then multiplied by a correction factor of 110 to get percent body protein. This factor was obtained by multiplying 11 (initial dilution for homogenization) by 100 (dilution of homogenate), divided by 1000 (conversion of mg to g), times 100 (to obtain percentage).

In this method of assay a purple-colored complex is formed when the copper in the alkaline reagent reacts with the peptide bonds in protein and the reduction of phosphomolybdate by tyrosine and tryptophan in the protein.

Determination of Lipids

The Folch method (66) was used to analyze fat content in the 2.0 ml homogenate. Fifteen ml cold chloroform-methanol (2:1, v/v) was added to the homogenate in each 20 × 150 mm test tube. Tubes were shaken 10-15 minutes on a wrist-action shaker. Samples were filtered through Whatman #1 filter paper into glass conical centrifuge tubes. An additional 5.0 ml chloroform-methanol was used to wash the filter paper. Four ml cold 0.73% NaCl was then added to each centrifuge tube and tubes were shaken again for 10-15 minutes. After centrifuging in a clinical centrifuge at highest speed (5125 x g), the tube contents had separated into 2 phases: the top layer being the aqueous phase and the lower layer the organic phase containing the lipids. The upper phase was aspirated off by a vacuum pump into an erlenmeyer flask trap. Seven ml of the organic phase was pipetted into pre-weighed aluminum dishes (prepared as for dry matter determination). The dishes were allowed to stand overnight during which time the chloroform-methanol evaporated leaving the lipid residue in the dishes. The following day the dishes were heated in a 103° oven for 2 hours, cooked in a desiccator 30 minutes, and weighed. Percent body fat (lipid) was calculated by multiplying mg lipid in each dish by 1021.4. This factor was obtained by multiplying 11 (initial dilution for homogenization), by 1.8571 (13 ml total organic phase ÷ 7 ml organic phase used), divided by 2 ml (because 2 ml homogenate was used), times 100 (to get percent).

Statistical Analyses

The effect of exercise duration on measurements were assessed by Analysis of Variance procedures (67) followed by Least Significant Differences tests (67). Statistical procedures were carried out using the computerized SAS (Statistical Analysis System) (68). A sample computer program for our data is shown in appendix table 2. The "predicted" body weights and food intakes for each group were mathematically computed as a function of time using Common Slopes Covariance Model followed by Least Significant Differences Tests (67). A sample computer program is shown in appendix table 3.

RESULTS AND DISCUSSION

Exercise and Body Weight

The effect of exercise duration on mean body weights of male mice during the 10-week study are presented in table 4. Mice exercised different lengths of time did not have significantly different body weights during the 10-week study. In contrast, other investigators (32,35,43,50) reported that exercised rats had lower body weights than sedentary freely-eating control rats. Lack of agreement may be attributed to different exercise methodologies, age, sex, and species of animals used in those studies.

The measurement of body weight gain is one way to assess growth. Although our mice were young adults, they continued to gain weight throughout the 10-week study. Large within group body weight variations also were observed in those mice. To correct for this variation the "predicted" body weights for mice in each group were computed mathematically from this data as a function of time using a Common Slopes Covariance Model followed by LSM in SAS (67). In figure 6, the calculated regression line for each exercise group is compared with that for the control group. Data from the last 8 weeks of the experiment were used because mice were exercised at their target exercise periods during that time. Statistical analyses revealed that there were no significant differences in body weight between the exercised and control animals.

At the onset of the study, the animals already had gained approximately 80-85% of their final body weight. Average weight gains during the 10-week study are shown in table 5; cumulative weight gains for

TABLE 4

Effect of exercise duration on body weights of Swiss albino mice: weeks 1-10[1]

Weeks	Time exercised (min)					
	0	20	40	60	120	240
	g					
1 (training)[2]	30.4 ±0.4	29.4 ±0.7	30.1 ±0.7	30.4 ±0.7	30.4 ±0.7	31.1 ±0.7
2 (training)[3]	31.3 ±0.4	30.5 ±0.6	30.9 ±0.7	31.5 ±0.6	29.9 ±0.9	31.4 ±0.5
3	32.0 ±0.4	31.3 ±0.6	31.2 ±0.9	31.4 ±0.9	31.0 ±1.0	31.7 ±0.7
4	33.4 ±0.5	32.4 ±0.7	32.5 ±0.9	32.9 ±0.7	32.3 ±0.9	33.2 ±0.8
5	34.3 ±0.5	33.2 ±0.7	33.3 ±0.9	34.3 ±0.8	33.5 ±0.9	34.1 ±0.8
6	35.4 ±0.4	34.3 ±0.7	34.7 ±1.0	35.2 ±0.9	34.5 ±0.8	35.2 ±1.0
7	35.4 ±0.4	34.5 ±0.7	34.8 ±0.9	35.8 ±0.9	34.5 ±0.9	35.4 ±0.8
8	36.2 ±0.4	35.6 ±0.7	35.7 ±0.9	36.4 ±0.7	35.3 ±0.9	36.2 ±0.8
9	36.4 ±0.4	36.0 ±0.7	35.6 ±1.0	36.6 ±0.8	35.2 ±0.9	35.7 ±1.1
10	36.5 ±0.4	36.6 ±0.9	36.2 ±1.2	37.2 ±0.8	36.1 ±1.1	36.6 ±0.8

[1] Results are expressed as means ± SEM for 6-9 mice in each group.
[2,3] During weeks 1 and 2, mice were not exercised for the time duration shown, but were on the progressive exercise schedule shown in table 3. There were no significant differences between treatment groups.

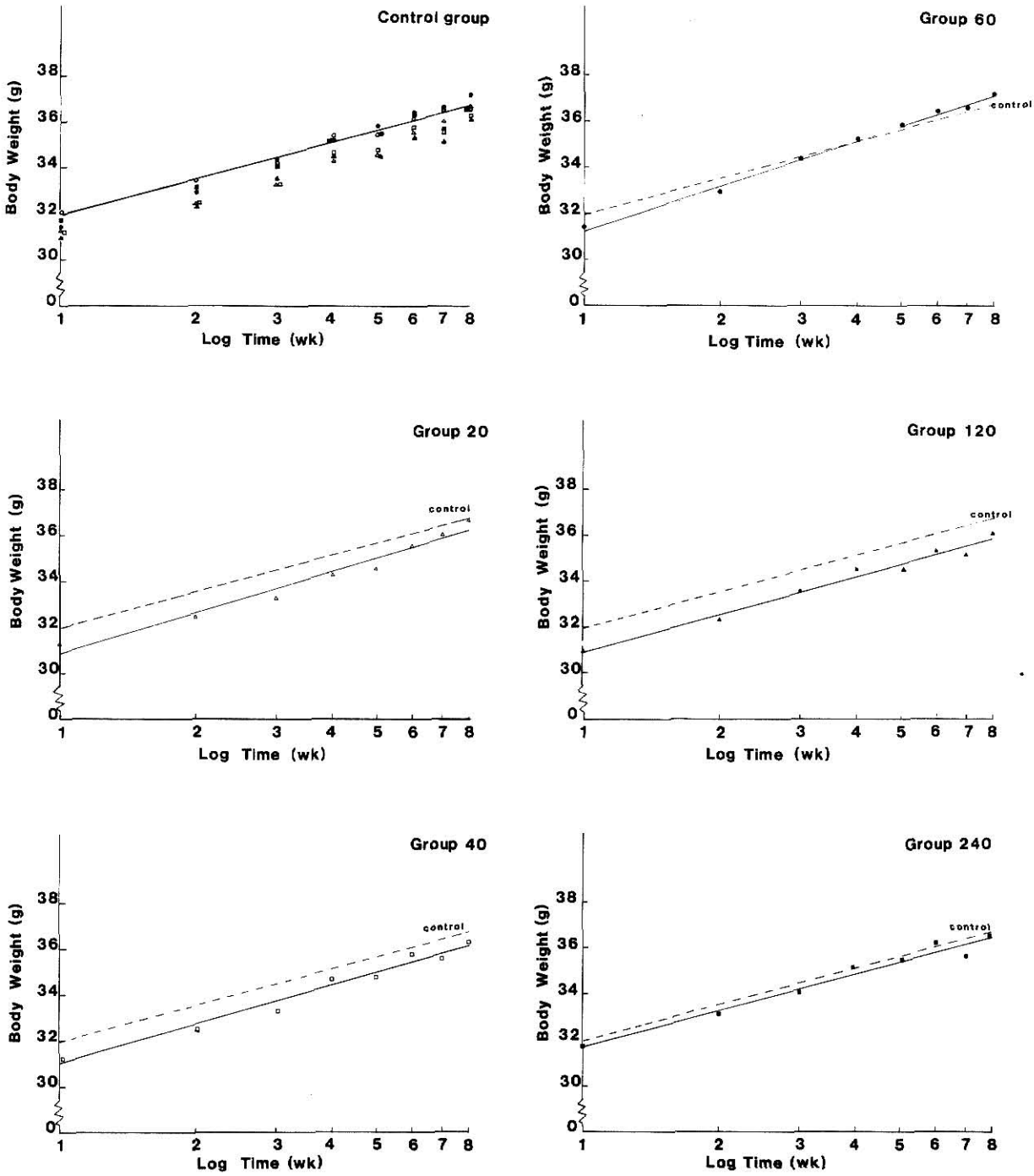

Fig. 6 Predicted body weights as a function of time in control and exercised mice during the 8-week study.

TABLE 5

Effect of exercise duration on weight gain of male Swiss albino mice[1]

Measurements	Time exercised (min)					
	0	20	40	60	120	240
			g			
Initial body weight	31.1 ±0.4	30.8 ±0.5	30.7 ±0.6	31.4 ±0.6	31.6 ±0.3	31.5 ±0.3
Weight gain week 1 and 2 (training)[2]	0.1[b] ±0.5	-0.4[b] ±0.4	0.1[b] ±0.2	0.1[b] ±0.2	-1.7[a] ±0.8	-0.1[b] ±0.3
Weight gain week 3-6	4.1 ±0.4	3.8 ±0.3	3.8 ±0.3	3.7 ±0.6	4.6 ±0.9	3.8 ±0.7
Weight gain week 7-10	1.1 ±0.2	2.3 ±0.5	1.5 ±0.3	2.0 ±0.3	1.6 ±0.4	1.4 ±0.3
Total weight gain	36.5 ±0.4	36.6 ±0.9	36.1 ±1.2	37.1 ±0.8	36.1 ±1.1	36.6 ±0.8

[1] Results are expressed as means ± SEM for 6-9 mice in each group.
[2] During weeks 1 and 2, mice were not exercised for the time duration shown, but were on the progressive exercise schedule shown in table 3.
[a-b] Means in the same row with different superscripts are significantly different ($P < 0.05$).

individual mice in each treatment group are shown in figure 7. Large variations in weight gain among animals in the same group were observed (figure 7). This seemed to be particularly true for the first 2 weeks (training period) of the study. The variation observed might be attributed to the animals' individual responses to the initial phase of the training program. The significantly lower weight gain of group 120 during the 2-week preliminary training program is not clear, however, it appears that the weight loss was regained during the next four weeks.

During weeks 3-6 the animals gained another 9-12% of their final body weight. The observed weight gain during this period was not significantly different among the treatment groups. However, the dramatic increase in body weight when compared to the training period suggests that perhaps the animals had become adjusted to their training program and environment and exhibited faster growth rates.

During weeks 7-10 of the experiment, the animals gained weight at a slower rate than the preceding 4 weeks. Average weight gains during this time represented approximately 3-6% of their final body weight. The slower rate of weight gain seen in the last 4 weeks may suggest that animals had reached a plateau stage in their growth.

Exercise did not significantly affect mean body weight among the treatment groups. Exercise did, however, seem to affect the variability of individual body weights. In figure 7, individual body weights of exercised mice are compared to those of control mice during the 8-week target exercise period. The vertical bar after week 10 indicates the range between the heaviest and the lightest mouse in the control group. The range of body weights for each exercise group can easily be compared to that of the control group. Variation in body weights for the exercised

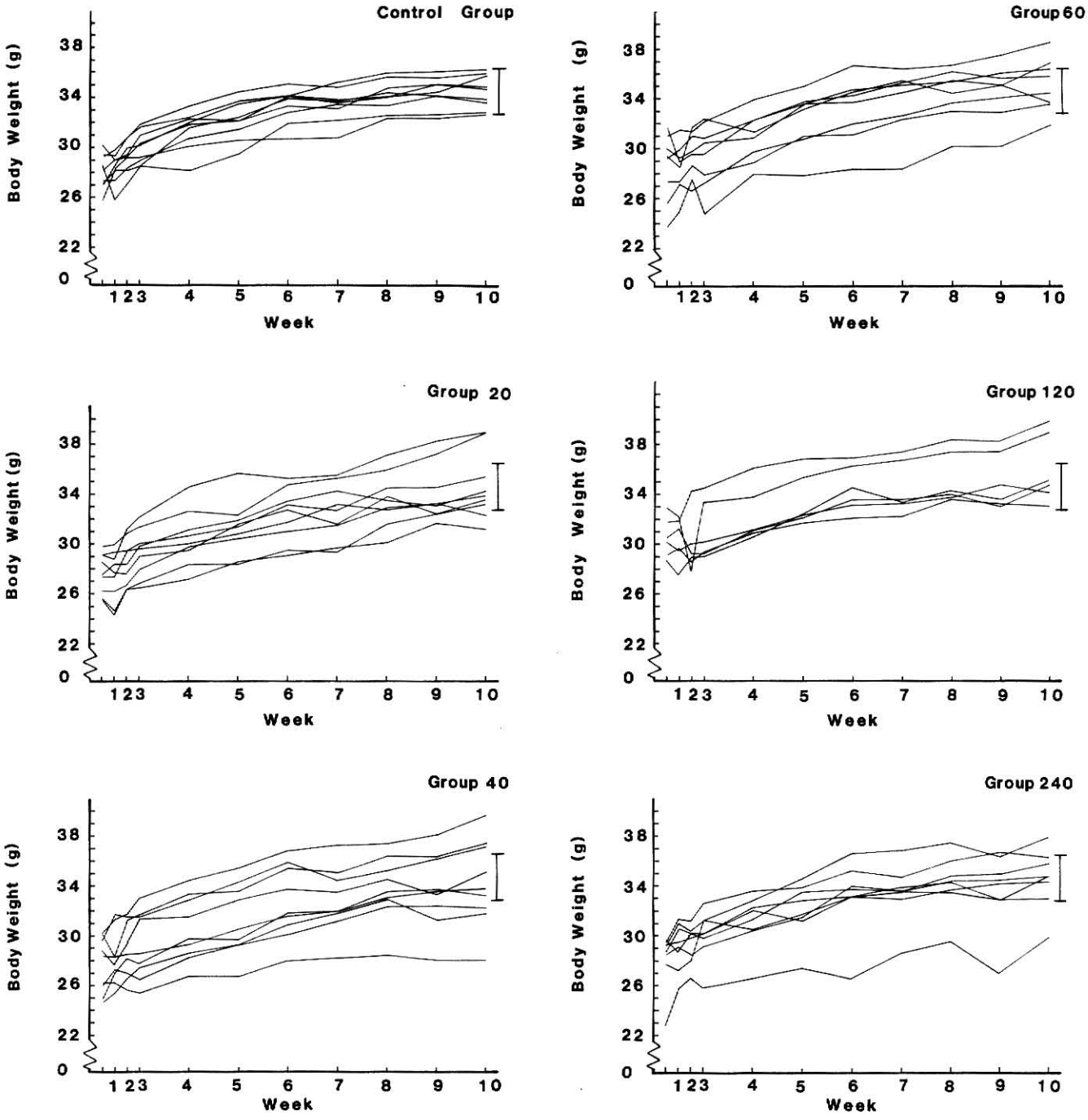

Fig. 7 Body weights of control and exercised mice during the preliminary training period and 8-week study. Each line represents weight gain for an individual animal. The vertical bar after week 10 represents the distance between the heaviest and lightest mouse in the control group.

animals seemed to be greater than that of the control group for all weeks, even though initially body weights were similar. Increased exercise duration was not followed by an increase in body weight variation.

The within group variation observed in our exercised mice may be attributed to several factors. First, animals within a given exercise group may have responded differently to that particular level of exercise. In this study some of the exercise animals ran at a smooth easy pace, whereas others ran at short bursts of speed alternating with rest periods. It is possible that these differences in exercise affected our measurements. Variation also could be attributed to different amounts of food in the digestive tract. Mice consume an average 5-6 grams food daily which is large in comparison to their average body weight of 25-35 grams. It is possible that exercise affected factors relating to food consumption such as time of food consumption or gastrointestinal transit time for the food consumed. Variation could be minimized perhaps by using a larger number of animals for each group. However this study was limited by the number of mice which could be exercised on the treadmill at one time. The treadmill had 18 cubicles for 18 animals and we chose to exercise two groups of 9 mice at the same time. Variation also could be minimized by using littermates for the different groups to insure that a comparison would be made among animals that were genetically similar.

Exercise and Food Intake

The effect of exercise duration on average weekly food intake of male mice during the 10-week study are presented in table 6. In table 7 total food intakes are compared during weeks 1 and 2 (training period), weeks 3-6, and weeks 7-10. In general exercise did not significantly affect total food intake for any of the treatment groups. However, animals

TABLE 6

Effect of exercise duration on weekly food intake of male Swiss albino mice: weeks 1-10[1]

Week	Time exercised (min)					
	0	20	40	60	120	240
	g/week					
1 (training)[2]	35.3 ±1.1	34.7 ±0.9	33.8 ±0.5	34.2 ±0.9	34.8 ±0.5	36.0 ±1.0
2 (training)[3]	35.6 ±0.7	34.5 ±0.9	33.4 ±0.9	34.2 ±0.9	35.3 ±1.6	34.2 ±0.7
3	40.2^{ab} ±0.9	38.3^{a} ±1.0	37.8^{ab} ±1.0	38.3^{ab} ±1.8	41.6^{bc} ±1.4	41.3^{c} ±0.8
4	39.4 ±1.6	39.0 ±0.8	39.0 ±1.1	39.9 ±1.5	41.1 ±0.9	42.7 ±0.9
5	36.8 ±0.6	34.4 ±0.8	35.3 ±1.1	36.5 ±1.4	37.5 ±1.1	39.5 ±0.8
6	36.8 ±0.6	35.0 ±0.5	37.2 ±1.1	37.6 ±0.9	37.2 ±0.9	38.7 ±1.1
7	38.0 ±0.6	36.6 ±0.9	36.6 ±0.8	38.3 ±1.0	38.0 ±1.4	39.4 ±0.8
8	39.4 ±1.1	37.8 ±0.6	38.0 ±0.9	39.1 ±0.9	40.9 ±1.0	40.0 ±0.6
9	38.7 ±1.0	36.7 ±0.5	37.6 ±1.0	37.8 ±0.6	38.2 ±0.9	38.8 ±1.1
10	36.0 ±0.8	35.9 ±0.7	37.5 ±1.4	36.9 ±0.9	37.3 ±1.2	38.9 ±0.8

[1] Results are expressed as means ± SEM for 6-9 mice in each group.
[2,3] During weeks 1 and 2, mice were not exercised for the time duration shown, but were on the progressive exercise schedule shown in table 3.
[a-c] Means in the same row with different superscripts are significantly different ($P < 0.05$).

TABLE 7

Effect of exercise duration on total food intake in male Swiss albino mice[1]

Measurements	Time exercised (min)					
	0	20	40	60	120	240
			g			
Food intake week 1 and 2 (training)[2]	70.9 ±1.7	69.1 ±1.7	67.2 ±1.2	68.5 ±1.7	70.1 ±2.0	70.2 ±1.4
Food intake week 3-6	153.3 ±3.5	146.8 ±2.7	149.2 ±4.0	152.3 ±5.4	157.4 ±3.8	162.3 ±3.1
Food intake week 7-10	152.2 ±3.4	147.1 ±2.5	149.7 ±3.7	152.1 ±3.0	154.4 ±3.8	157.1 ±2.9
Total food intake	376.4 ±7.9	363.0 ±5.6	366.1 ±8.6	372.9 ±9.5	382.0 ±9.1	389.6 ±5.4

[1] Results are expressed as means ± SEM for 6-9 mice in each group.
[2] During weeks 1 and 2, mice were not exercised for the time duration shown, but were on the progressive exercise schedule shown in table 3. There were no significant differences between treatment groups.

exercised for 240 minutes had higher food intakes than control mice and those exercised for 20, 40 and 60 minutes during week 3.

Total food intakes during the last 8 weeks (target exercise period) are compared in the bar graph in figure 2. We found a trend similar to that in his reported results (figure 8). That is, mice exercised for 20-40 minutes tended to have lower food intakes than the control mice did, but differences were not significantly different. We also found that mice exercised for 60 to 240 minutes tended to consume more food in amounts proportional to the amount of time exercised.

This study was designed to duplicate that of Mayer et al. (20) except that mice were used instead of rats. Although the trends were similar to those observed in the study by Mayer et al. (20) the differences were not marked. There are several possible reasons for these differences, and they should be recognized if further studies are to be performed in the future. First, it is difficult to compare responses of mice to those of rats. Mice tend to have a higher basal metabolic rate than rats, and also have a higher level of spontaneous activity, which may affect baseline values. Perhaps the baseline activity can be restricted by housing mice in smaller cages. Also our control animals were treated differently than those in the Mayer et al. study. Our control animals were removed from their cages and placed on the treadmill (without exercise) to correct for handling stresses. This handling of control animals was not mentioned in the Mayer et al. study. Perhaps our control animals would have gained more weight and consumed more food if they were not handled. This would result in greater differences between treatment groups. Furthermore, the rats in the Mayer et al. study were exercised at 1 mph. In the present study the

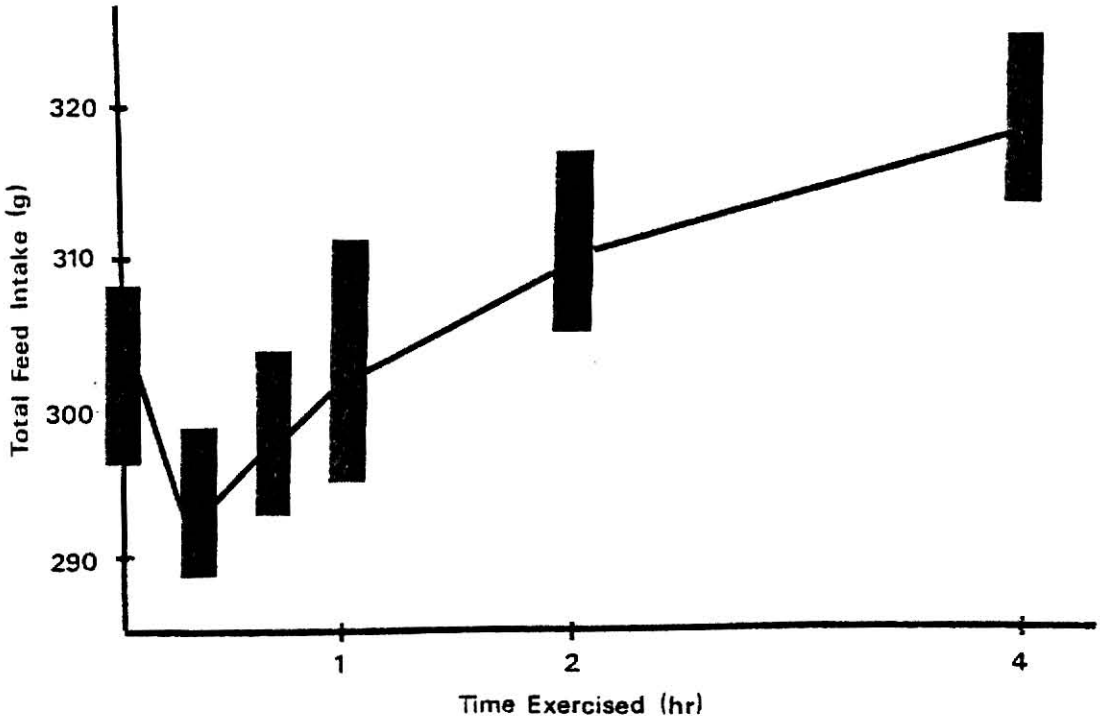

Fig. 8 Effect of exercise duration on total food intake of mice. Each bar represents means ± SEM for total food intake (8-week) in mice in response to increased exercise duration.

the speed was adjusted to .27 mph for mice to compensate for their smaller size.

To minimize variation in body weight within groups, animals could be littermates or matched for body weight at the beginning of the study. Variation in response to exercise might be reduced by exercising animals in a running wheel instead of a treadmill.

Mice sometimes consumed their own bedding (wood shavings) and feces (coprophagy) which may have affected caloric intake. To prevent this in future studies mice could be housed in wire-bottom cages. Another practice which could minimize variation in food intake would be the use of a more purified homogenous diet over the Purina Rodent Chow used in this study.

Because larger mice would be expected to consume more food than smaller mice weekly food/body weight ratios for mice in each of our exercise groups were calculated (table 8). In general, mice in the exercise groups of longer duration tended to have higher food/body weight ratios, with significantly higher ($P < 0.05$) food intakes by the 240 minute mice during four of the weeks they were exercised. The "predicted" food intakes for mice in each group were computed mathematically as a function of time using Common Slopes Covariance Model followed by LSM in SAS (67) (figure 9). The observed trends in food intake are similar to those for food/body weight ratios. That is, mice exercised for longer periods of time tended to have higher food intakes than those that were exercised shorter lengths of time.

Exercise and Body Composition

The effect of exercise duration on mean body weight, carcass weight, heart weight and epididymal fat pad (EFP) weight at sacrifice are presented

TABLE 8

Effect of exercise duration on food/body weight ratio[1] of male Swiss albino mice: weeks 1-10[2]

week	Time exercised (min)					
	0	20	40	60	120	240
	g food/g body wt					
1 (training)[3]	0.19 ±0.00	0.19 ±0.00	0.18 ±0.00	0.18 ±0.00	0.19 ±0.01	0.19 ±0.00
2 (training)[4]	0.18 ±0.01	0.18 ±0.00	0.18 ±0.00	0.18 ±0.01	0.20 ±0.01	0.19 ±0.00
3	0.18^{ab} ±0.00	0.17^{a} ±0.00	0.17^{a} ±0.00	0.17^{a} ±0.00	0.19^{c} ±0.00	0.19^{bc} ±0.00
4	0.17 ±0.00	0.17 ±0.00	0.17 ±0.00	0.17 ±0.00	0.18 ±0.00	0.18 ±0.00
5	0.15^{ab} ±0.00	0.15^{a} ±0.00	0.15^{a} ±0.00	0.15^{ab} ±0.00	0.16^{bc} ±0.00	0.16^{c} ±0.00
6	0.15^{ab} ±0.00	0.14^{a} ±0.00	0.15^{bc} ±0.00	0.15^{bc} ±0.00	0.15^{bc} ±0.00	0.16^{c} ±0.00
7	0.15 ±0.00	0.15 ±0.00	0.15 ±0.00	0.15 ±0.00	0.16 ±0.00	0.16 ±0.00
8	0.16 ±0.00	0.15 ±0.00	0.15 ±0.00	0.15 ±0.00	0.16 ±0.00	0.16 ±0.00
9	0.15 ±0.00	0.15 ±0.00	0.15 ±0.00	0.15 ±0.00	0.15 ±0.00	0.15 ±0.00
10	0.14^{ab} ±0.00	0.14^{a} ±0.00	0.15^{bc} ±0.00	0.14^{ab} ±0.00	0.15^{abc} ±0.00	0.15^{c} ±0.00

[1] Ratios calculated as average daily food consumption over body weight for that week. [2] Results are expressed as means ± SEM for 6-9 mice in each group. [3,4] During week 1 and 2, mice were not exercised for the time duration shown, but were on the progressive exercise schedule shown in table 3. [a-c] Means in the same row with different superscripts are significantly different ($P < 0.05$).

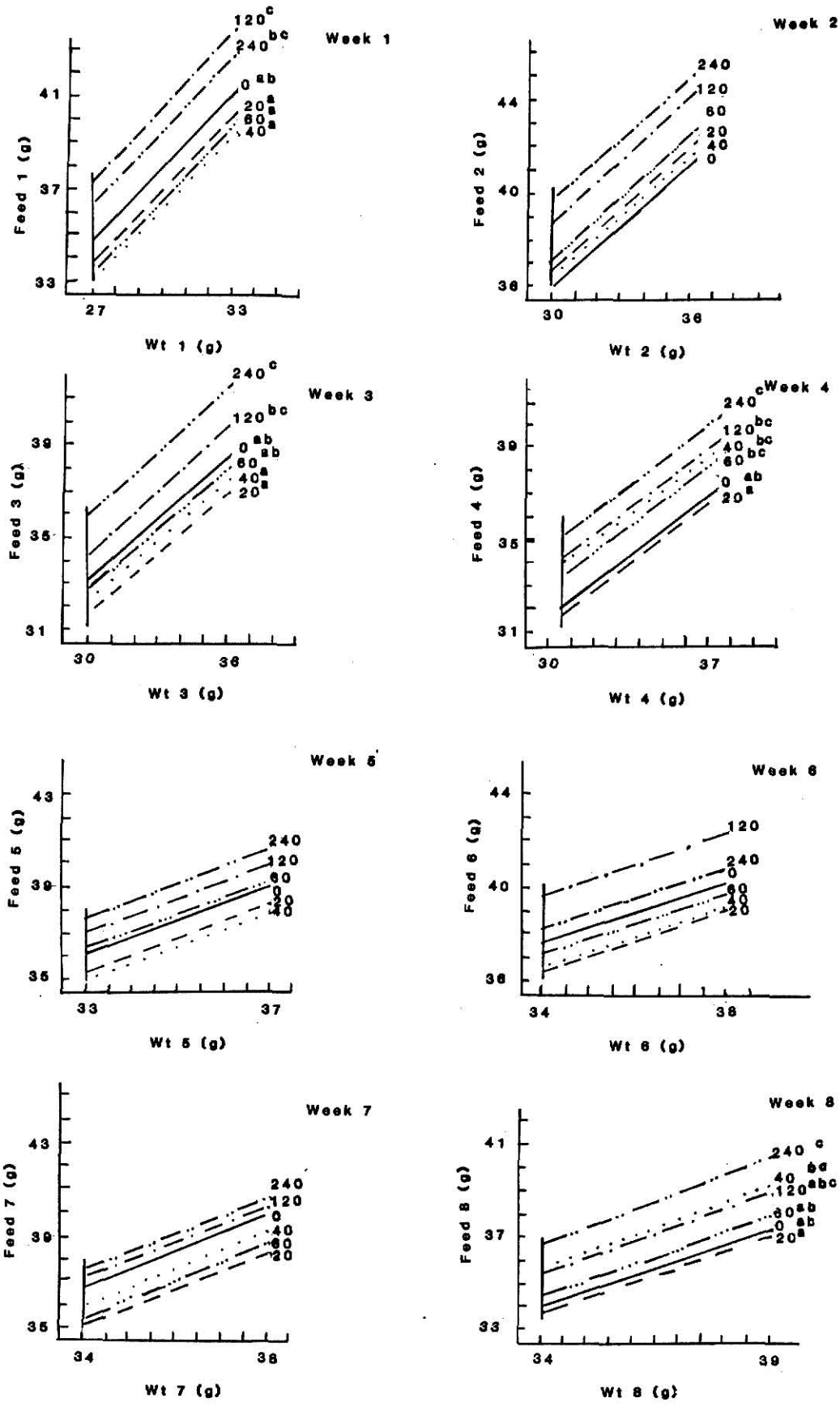

Fig. 9 Predicted food intake as a function of body weight in control and exercised mice. a-c means with different superscripts are significantly different ($P<0.05$).

in table 9. Increased exercise duration did not seem to affect final body or carcass weight at the time of sacrifice. In contrast to our results, other investigators (32,35,43,50) have found that body weights of animals subjected to exercise periods of long duration were smaller than those of sedentary freely-eating control animals. Lack of agreement may be attributed to different exercise speeds used in those studies, as well as age, species and sex differences.

Increased exercise duration did not seem to affect mean heart weight at the time of sacrifice because no significant differences were observed between treatment groups. Lack of significant differences may be attributed to large within group variability in individual body weights or possibly exercise at a level inadequate to cause cardiac hypertrophy. Because there is a relationship between heart weight and body weight, Common Slopes Covariance Model followed by LSM (67) was used to test for significance between the standardized heart weight/carcass weight relationship. No significant differences were observed for the heart weight-carcass weight relationship between the treatment groups. In contrast, other investigators (40,49,50) reported that exercise increased heart weight-body weight ratio.

Common Slope Covariance Model followed by LSM (67) also was used to test for any significant differences between EFP-carcass weight relationship. Increased exercise duration did not affect mean EFP weights at the time of sacrifice because no significant differences were observed between the treatment groups. In contrast, other investigators (37,46) reported a reduction in EFP weights in response to exercise. Lack of agreement may be attributed to the large variation in individual body weights as well as different exercise methodologies, age, sex, and species differences.

TABLE 9

Effect of exercise duration on weight measurements of male Swiss albino mice at sacrifice[1]

Measurement	Time exercised (min)					
	0	20	40	60	120	240
Body weight, g	36.7 ±0.5	36.3 ±0.7	36.3 ±1.1	36.7 ±0.8	36.3 ±1.1	35.8 ±0.9
Carcass weight, g	31.5 ±0.4	31.5 ±0.6	31.4 ±0.9	31.6 ±0.7	31.0 ±0.9	30.9 ±0.8
Heart weight, mg	158 ±5.4	157 ±6.7	152 ±6.3	166 ±8.4	153 ±6.8	156 ±5.4
% of body weight	(0.5 ±0.4)	(0.5 ±0.0)	(0.5 ±0.0)	(0.5 ±0.2)	(0.5 ±0.0)	(0.5 ±0.0)
Epididymal fat pad, g	0.7 ±0.1	0.7 ±0.1	0.7 ±0.0	0.7 ±0.1	0.6 ±0.1	0.6 ±0.1
% of body weight	(2.2 ±0.2)	(2.4 ±0.1)	(2.2 ±0.1)	(2.1 ±0.2).	(1.9 ±0.2)	(1.8 ±0.2)

[1] Results are expressed as means ± SEM for 6-9 mice in each group after the 10-week trial. There were no significant differences between treatment groups.

The effect of exercise duration on body composition at sacrifice is shown in figure 10. Significant differences were observed when comparing percent body fat of the treatment groups. Mice in treatment groups 240, and 120 had significantly less ($P < 0.05$) body fat than those in group 20 and the control group. Our data corroborate the findings of other investigators (31,38,39). Exercise did not seem to affect percent protein among the treatment groups.

Significant differences ($P < 0.05$) were observed in body water content between the treatment groups. Mice that were exercised 240 minutes had significantly more body water than mice that were exercised 20, 40, or 60 minutes or control mice. Higher body water contents in exercised mice has been observed by other researchers (30,38,42). The finding that the mice exercised 240 minutes had the highest percent body water was expected because this group also had the lowest percent body fat, and it is well known that body fat is inversely proportional to body water (60).

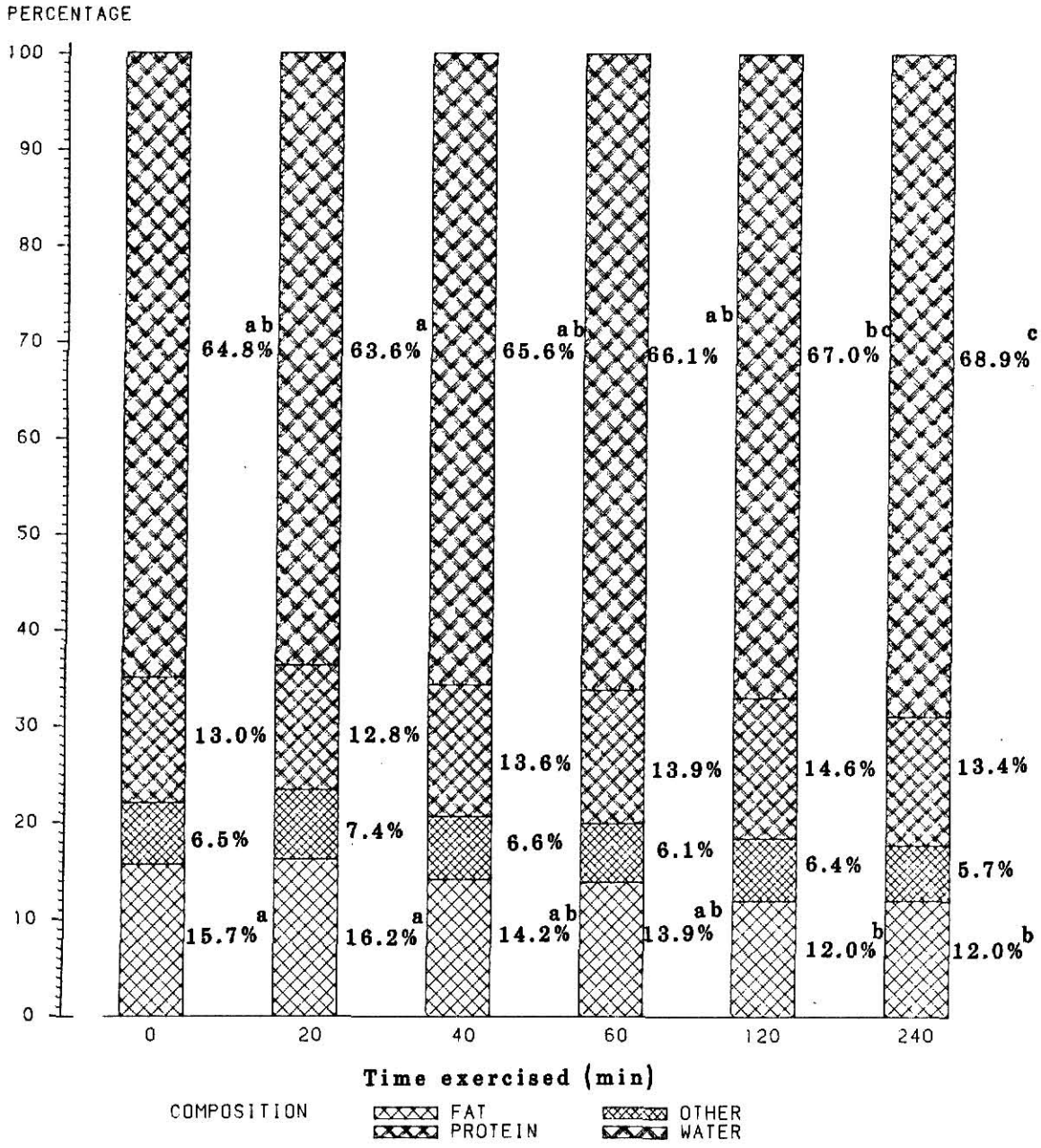

Fig. 10 *Influence of exercise duration on body composition of mice.* $^{a-c}$ **means with different superscripts are significantly different ($p<0.05$).**

SUMMARY

The effects of exercise duration on food intake, body weight, and body composition were studied in adult male Swiss Albino mice. Fifty-four mice were randomly assigned to one of six treatment groups: a control group (not exercised) or exercise groups receiving either 20, 40, 60, 120, 240 minutes of treadmill exercises 5 days/week for 8 weeks.

Throughout the study mice exercised 20 or 50 minutes tended to consume less food than control animals; and mice exercised more than 60 minutes tended to have greater food intakes proportional to the length of time exercised, but these differences were not statistically significant.

Analyses of body composition of the mice revealed that mice exercised for 240 minutes had significantly lower ($P < 0.05$) percent body fat and significantly higher percent body water ($P < 0.05$) than mice exercised either 0 or 20 minutes per day. Percent body protein, heart weights, and epididymal fat pad weights were not significantly different among treatment groups.

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LITERATURE CITED

1. Abraham, S. & Johnson, C. L. (1980) Prevalence of severe obesity in adults in the United States. Am. J. Clin. Nutr. 33, 364-369.

2. Select Committee on Nutrition and Human Needs. (1977) Diet related to killer diseases II. Part II. Obesity. U.S. Senate.

3. Kissileff, H. R. & Van Itallie, T. B. (1982) Physiology of the control of food intake. Ann Rev. Nutr. 2, 300-371.

4. Hamilton, C. L. (1973) Physiologic control of food intake. J. Am. Dietet. Assoc. 62, 35-40.

5. Anand, B. K. (1961) Nervous regulation of food intake. Physiol. Rev. 41, 677-708.

6. Mayer, J. (1965) The ventromedial glucostatic mechanism as a component of satiety. Postgrad. Med. 38, A101-110.

7. Mayer, J. (1953) Glucostatic mechanism of regulation of food intake. N. Engl. J. Med. 249, 13-16.

8. Kennedy, G. C. (1953) The role of depot fat in the hypothalamic control of food intake in the rat. Proc. Royal Soc. Lond. (Biol) 140, 578-592.

9. Cohn, C. & Joseph, D. (1962) Influence of body weight and fat on appetite of "normal" lean and obese rats. Yale J. Biol. Med. 34, 598-607.

10. Andik, I., Donhoffer, Sz., Farkas, M. & Schmidt, P. (1963) Ambient temperature and survival on a protein-deficient diet. Brit. J. Nutr. 17, 257-261.

11. Leung, P. & Rogers, Q. (1969) Food intake: regulation by plasma amino acid pattern. Life Sci. 8, 1-9.

12. Gradison, L. & Guidotti, A. (1977) Stimulation of food intake by muscimol and beta endorphin. Neuropharmacology 16, 533-536.

13. Myers, R. D. & McCaleb, M. L. (1980) Feeding: satiety signal from intestine triggers brain's noradrenergic mechanism. Science 209, 1035-1037.

14. Smith, G. P. (1973) Humoral hypothesis for the control of food intake. In: Obesity in Perspective (Bray, G. A., ed.), Part 2, DHEW Publication No. (NIH) 75-708.

15. Waldbillig, R. J., Bartness, T. J. & Stanley, B. G. (1981) Increased food intake, body weight, and adiposity in rats after regional neurochemical depletion of serotonin. J. Comp. Physiol. Psychol. 95, 391-405.

16. Wurtman, J. J. & Wurtman, R. J. (1979) Drugs that enhance central serotoninergic transmission diminish elective carbohydrate consumption by rats. Life Sci. 24, 895-904.

17. Collier, G., Hirsch, E. & Hamlin, P. H. (1972) The ecological determinants of reinforcement in the rat. Physiol. Behav. 9, 705-716.

18. Kanarek, R. B. (1976) Energetics of meal patterns in rats. Physiol. Behav. 17, 395-399.

19. Hoebel, B. G. & Teitelbaum, P. (1962) Hypothalamic control of feeding and self-stimulation. Science 135, 375-377.

20. Mayer, J., Marshall, N. B., Vitale, J. J., Christensen, J. H., Mashayekhi, M. B. & Stare, F. J. (1954) Exercise, food intake and body weight in normal rats and genetically obese adult mice. Am. J. Physiol. 177, 544-548.

21. Mayer, J., Roy, P. & Mitra, K. P. (1956) Relation between caloric intake, body weight, and physical work: studies in an industrial male population in West Bengal. Am. J. Clin. Nutr. 4, 169-175.

22. Dawson, C. A. & Horvath, S. M. (1970) Swimming in small laboratory animals. Med. Sci. Sports 2, 51-78.

23. Oscai, L. B. & Molé, P. A. (1975) Laboratory techniques involving small animals in exercise physiology research. Exercise Sport Sci. Rev. 3, 351-370.

24. Wranne, B. & Woodson, R. D. (1973) A graded treadmill test for rats: maximal work performance in normal and anemic animals. J. Appl. Physiol. 34, 732-735.

25. Andrews, R. J. (1965) Treadmill for small laboratory animals. J. Appl. Physiol. 20, 572-574.

26. Jenness, M. E. & Byrd, R. J. (1974) "Live-in" exercise drums for small animals. J. Appl. Physiol. 36, 509-510.

27. Wells, R. L. & Heusner, W. W. (1971) A controlled-running wheel for small animals. Lab. Animal Sci. 21, 904-912.

28. Russell, J. C., Campagna, P. D. & Wenger, H. A. (1980) Small animal ergometer. J. Appl. Physiol. 48, 394-398.

29. Dohm, G. L., Beecher, G. R., Stephenson, T. P. & Womack, M. (1977) Adaptations to endurance training at three intensities of exercise. J. Appl. Physiol. 42, 753-757.

30. Crews, E. L., Fuge, K. W., Oscai, L. B., Holloszy, J. O. & Shank, R. E. (1969) Weight, food intake, and body composition: effect of exercise and of protein deficiency. Am. J. Physiol. 216, 359-363.

31. Stevenson, J. A. F., Box, B. M., Feleki, V. & Beaton, J. R. (1966) Bouts of exercise and food intake in the rat. J. Appl. Physiol. 21, 118-122.

32. Katch, V. L., Martin, R. & Martin, J. (1979) Effects of exercise intensity on food consumption in the male rat. Am. J. Clin. Nutr. 32, 1401-1407.

33. Taylor, A. W., Cary, S., McNully, M., Barrod, J. & Secord, D. C. (1974) Effects of food restriction and exercise upon the deposition and mobilization of energy stores in the rat. J. Nutr. 104, 218-222.

34. Owens, J. L., Fuller, E. D., Nutter, D. D. & Digirolame, M. (1977) Influence of moderate exercise on adipocyte metabolism and hormonal responsiveness. J. Appl. Physiol. 43, 425-430.

35. Lau, H. C., Flaim, E. & Ritchney, S. J. (1979) Body weight and depot fat changes as influenced by exercise and dietary fat sources in adult BHE rats. J. Nutr. 109, 495-500.

36. Oscai, L. B., Babirak, S. P., Dubach, F. B., McGarr, J. A. & Spirakis, C. N. (1974) Exercise or food restriction: effect on adipose tissue cellularity. Am. J. Physiol. 227, 901-904.

37. Askew, E. W. & Hecker, A. L. (1976) Adipose tissue cell size and lipolysis in the rat: response to exercise intensity and food restriction. J. Nutr. 106, 1351-1360.

38. Walberg, J. L., Molé, P. A. & Stern, J. S. (1982) Effect of swim training on development of obesity in the genetically obese rat. Am. J. Physiol. 242, R204-R211.

39. Oscai, L. B., Molé, P. A., Krusack, L. M. & Holloszy, J. O. (1973) Detailed body composition analysis on female rats subjected to a program of swimming. J. Nutr. 103, 412-418.

40. Oscai, L. B., Molé, P. A. & Holloszy, J. O. (1971) Effects of exercise on cardiac weight and mitochondria in male and female rats. Am. J. Physiol. 220, 1944-1948.

41. Nance, D. M., Bromley, B., Barnard, R. J. & Gorski, R. A. (1977) Sexually dimorphic effects of forced exercise on food intake and body weight in the rat. Physiol. Behav. 19, 155-158.

42. Jones, E. M., Montoye, H. J., Johnson, P. B., Martin, J. M., VanHuss, W. D. & Cederquist, D. (1964) Effects of exercise and food restriction on serum cholesterol and liver lipids. Am. J. Physiol. 207, 460-466.

43. Oscai, L. B. & Holloszy, J. O. (1969) Effects of weight changes produced by exercise, food restriction or overeating on body composition. J. Clin. Invest. 48, 2124-2128.

44. Deb, S. & Martin, R. J. (1975) Effects of exercise and of food restriction on the development of spontaneous obesity in rats. J. Nutr. 105, 543-549.

45. Oscai, L. B., Babirak, S. P., McGarr, J. A. & Spirakis, C. N. (1974) Effects of exercise on adipose tissue cellularity. Fed. Proc. 33, 1956-1958.

46. Askew, E. W., Barakat, H., Kuhl, G. L. & Dohm, G. L. (1975) Response of lipogenesis and fatty acid synthesis to physical training and exhaustive exercise in rats. Lipid 10, 491-496.

47. Oscai, L. B., Spirakis, C. N., Wolff, C. A. & Beck, R. J. (1972) Effects of exercise and of food restriction on adipose tissue cellularity. J. Lipid Res. 13, 588-592.

48. Bukowiecki, L., Lupien, J., Follea, N., Paradis, A., Richard, D. & LeBlanc, J. (1980) Mechanism of enhanced lipolysis in adipose tissue of exercise-trained rats. Am. J. Physiol. 239, E422-E429.

49. Hickson, R. C., Hammons, G. T. & Holloszy, J. O. (1979) Development and regression of exercise-induced cardiac hypertrophy in rats. Am. J. Physiol. 236, H268-H272.

50. Oscai, L. B., Molé, P. A., Brei, B. & Holloszy, J. O. (1971) Cardiac growth and respiratory enzyme levels in male rats subjected to a running program. Am. J. Physiol. 220, 1238-1241.

51. Gleeson, M., Brown, J. F. & Waring, J. J. (1982) The effect of physical exercise on metabolic rate and dietary-indiced thermogenesis. Br. J. Nutr. 47, 173-183.

52. Berger, R. A. (1982) Applied Exercise Physiology, pp. 57-65, Lea & Febiger, Philadelphia.

53. Lamb, D. R. (1975) Androgens and exercise. Med. Sci. Sports 7, 1-5.

54. Dohm, G. L. & Louis, T. M. (1978) Changes in androstenedione, testosterone and protein metabolism as a result of exercise. Proc. Soc. Exp. Biol. Med. 158, 622-625.

55. Galbo, H., Hummer, L., Petersen, I. B., Christensen, N. J. & Bie, N. (1977) Thyroid and testicular hormone responses to graded and prolonged exercise in man. Europ. J. Appl. Physiol. 36, 101-106.

56. Hartley, L. H. (1975) Growth hormone and catecholamine response to exercise in relation to physical training. Med. Sci. Sports 7, 34-36.

57. Sutton, J. R. (1978) Hormonal and metabolic responses to exercise in subjects of high and low work capacities. Med. Sci. Sports 10, 1-6.

58. Tharp, G. D. (1975) The role of glucocorticoids in exercise. Med. Sci. Sports 7, 6-11.

59. Hartley, L. H., Mason, J. W., Hogan, R. P., Jones, L. G., Kotchen, T. A., Mougey, E. H., Wherry, F. E., Pennington, L. L. & Ricketts, P. T. (1972) Multiple hormonal responses to prolonged exercise in relation to physical training. J. Appl. Physiol. 33, 601-607.

60. Pkke, R. L. & Brown, M. L. (1975) Nutrition: An Integrated Approach, pp. 199-201, Wiley & Sons, Inc., New York.

61. Irvine, C. H. (1967) Thyroxine secretion rate in the horse in various physiologic states. J. Endocr. 39, 313-320.

62. Irvine, C. H. (1968) Effect of exercise on thyroxine degradation in athletes and non-athletes. J. Clin. Endocr. 28, 942, 948.

63. Wirth, A., Holm, G., Lindstedt, G., Lundberg, P. A. & Bjorntorp, P. (1981) Thyroid hormones and lipolysis in physically trained rats. Metabolism 30, 237-242.

64. Terjung, R. L. & Winder, W. W. (1975) Exercise and thyroid function. Med. Sci. Sports 7, 20-26.

65. Plummer, D. T. (1971) An Introduction To Practical Biochemistry: The Folin-Lowry Method of Protein Assay, p. 156, McGraw-Hill Book Co. Limited, England.

66. Folch, J., Lees, M. & Sloane Stanley, G. H. (1957) A simple method for the isolation and purification of total lipids from animal tissue. J. Biol. Chem. 226, 497-509.

67. Snedecor, G. W. & Cochran, W. G. (1976) Statistical Methods, Iowa State University Press, Ames.

68. Helwig, J. T. & Council, W. G. (eds.). (1979) SAS User's Guide, SAS Institute, Inc., Cary.

APPENDIX

APPENDIX TABLE 1

Reagents for analysis of protein and lipid

Protein

1. Alkaline sodium carbonate solution. (2% Na_2CO_3 in 0.1 N NaOH). Dissolve 20 g Na_2CO_3 and 4 g NaOH in a liter of deionized water. This was used in place of the "alkaline solution" for sample blanks.
2. Copper sulfate-sodium-potassium tartrate solution, (0.5% $CuSO_4$ in 1% Na, K tartrate). Dissolve .25 g $CuSO_4$ in 50 ml 1% Na, K tartrate. Prepare fresh daily.
3. "Alkaline solution." Prepare fresh daily by mixing 50 ml of (1) and 1 ml of (2).
4. Folin-Ciocalteau reagent. Dilute commercial reagent (Fisher Scientific, St. Louis, Mo.) with an equal volume of deionized water on the day of use.
5. Standard protein solution (0.2 mg/ml). Dissolve 50 mg bovine serum albumin (Sigma Chemical Co., St. Louis, Mo.) in 250 ml deionized water. Refrigerate.

Lipid

1. Chloroform-methanol (2:1, v/v). Mix 667 ml chloroform with 333 ml methanol. Refrigerate and keep cold during analysis by placing automatic pipettor in a container of crushed ice.
2. 0.73% NaCl. Dissolve .73 g NaCl in 100 ml water. Refrigerate and keep cold during analysis as for chloroform.

APPENDIX TABLE 2

Sample SAS[1] computer program for LSM procedure

```
//PRS0958 JOB (XXXXXXXXXXXX1,10) 'Becky Haack'
//EXEC SAS
//SYSIN DD *
DATA ONE:
INPUT;
CARDS;
PROC SORT;
DATA TWO;
INPUT;
CARDS:
PROC SORT;
PROC PRINT; BY GROUP;
PROC GLM;
CLASSES GROUP;
LSMEANS GROUP/STDERR PDIFF;
/*
```

[1] Statistical Analysis System.

APPENDIX TABLE 3

Sample SAS[1] computer program for Common Slopes Covariance Model procedure

```
//XPRS7646 JOB (XXXXXXXXXXX,3), 'Becky Haack'
//EXEC SAS
//SYSIN DD *
DATA ONE;
INPUT;
CARDS;
PROC SORT; BY MOUSE GROUP;
DATA; SET ONE;
PROC SORT; BY WEEK;
PROC GLM;
CLASSES GROUP;
MODEL FEED=GROUP WT/SOLUTION;
LSMEANS GROUP/PDIFF;
/*
```

[1] Statistical Analysis System.

APPENDIX TABLE 4

Effect of exercise duration on body weights of male Swiss albino mice: weeks 1 and 2

Week	Time exercised (min)					
	0	20	40	60	120	240
	g					
1 (training)[2]	29.4	26.7	29.0	29.4	29.7	33.3
	30.2	32.0	30.3	30.4	31.3	30.7
	30.3	30.8	33.4	30.9	27.6	31.1
	27.8	26.4	29.7	31.2	29.6	27.7
	30.7	29.4	28.2	31.9	31.9	31.5
	31.9	30.4	27.4	26.9	32.3	33.0
	31.0	29.7	33.6	29.3	--	29.2
	31.4	28.2	29.3	33.5	--	32.6
	31.1	31.4	30.3	--	--	--
	30.4[1]	29.4	30.1	30.4	30.4	31.1
	±0.4	±0.7	±0.7	±0.7	±0.7	±0.7
2 (training)[3]	30.5	28.4	30.2	30.7	28.7	33.2
	30.2	33.0	33.3	33.7	29.3	32.2
	31.2	33.1	33.7	31.5	29.0	30.5
	29.2	28.4	31.4	31.7	30.1	28.6
	32.0	31.5	27.8	33.0	34.3	31.8
	32.9	30.4	28.6	29.6	27.9	32.4
	31.4	29.6	33.5	28.6	--	30.0
	32.9	28.7	29.0	33.4	--	32.2
	31.3	31.5	30.6	--	--	--
	31.3	30.5	30.9	31.5	29.9	31.4
	±0.4	±0.6	±0.7	±0.6	±0.9	±0.5

[1] Results are expressed as means ± SEM for 6-9 mice in each group.
[2,3] During weeks 1 and 2, mice were not exercised for the time duration shown, but were on the progressive exercise schedule shown in table 3.

APPENDIX TABLE 5

Effect of exercise duration on body weights of male Swiss albino mice: weeks 3-6

Week	Time exercised (min)					
	0	20	40	60	120	240
				g		
3	31.0	28.5	29.8	29.9	29.4	34.6
	30.5	33.4	33.7	34.3	29.3	31.9
	31.2	34.3	35.0	31.5	29.1	31.1
	30.5	28.9	33.3	32.5	30.2	27.8
	32.2	31.7	27.4	32.8	34.5	32.1
	33.7	31.8	29.5	26.7	33.4	33.2
	33.0	31.0	33.5	29.3	--	31.1
	33.9	30.0	28.5	34.2	--	32.1
	32.3	32.0	30.6	--	--	--
	32.0[1]	31.3	31.2	31.4	31.0	31.7
	±0.4	±0.6	±0.9	±0.9	±1.0	±0.7
4	32.8	29.2	31.8	30.9	31.0	35.6
	33.6	34.7	35.3	33.3	31.1	33.3
	32.1	36.6	36.4	34.3	30.7	32.4
	30.2	30.4	33.5	32.9	31.2	28.6
	34.0	32.0	28.7	34.3	36.2	34.2
	34.5	33.1	30.6	30.0	33.9	34.8
	34.4	31.5	34.9	31.7	--	32.5
	35.4	31.8	30.3	35.9	--	34.1
	33.9	32.7	31.3	--	--	--
	33.4	32.4	32.5	32.9	32.3	33.2
	±0.5	±0.7	±0.9	±0.7	±0.9	±0.8
5	33.5	30.6	31.7	33.0	31.8	35.8
	34.5	34.3	35.6	35.2	32.4	35.5
	32.6	37.7	37.4	35.6	32.5	33.5
	31.5	30.4	34.9	35.6	32.2	29.4
	35.6	32.8	28.7	35.7	36.9	34.8
	35.8	33.9	31.3	29.9	35.5	36.6
	34.2	33.6	36.3	32.8	--	33.7
	36.5	32.5	31.3	37.0	--	33.2
	34.2	33.4	32.5	--	--	--
	34.3	33.2	33.3	34.3	33.5	34.1
	±0.5	±0.7	±0.9	±0.8	±0.9	±0.8
6	34.9	31.1	33.8	33.1	32.2	37.2
	36.0	36.7	37.4	36.6	33.2	35.7
	32.7	37.3	38.8	35.7	34.7	36.0
	34.0	31.5	35.7	36.7	33.7	28.6
	36.1	33.7	30.0	36.3	37.0	35.1
	36.2	35.4	32.9	30.4	36.4	38.6
	36.2	34.7	37.9	34.0	--	35.1
	37.1	33.0	32.1	38.7	--	35.2
	35.4	35.3	33.6	--	--	--
	35.4	34.3	34.7	35.2	34.5	35.2
	±0.4	±0.7	±1.0	±0.9	±0.8	±1.0

[1] Results are expressed as means ± SEM for 6-9 mice in each group.

APPENDIX TABLE 6

Effect of exercise duration on body weights of male Swiss albino mice: weeks 7-10

Week	Time exercised (min)					
	0	20	40	60	120	240
			g			
7	35.5	31.7	33.9	34.4	32.3	36.7
	35.7	37.3	37.0	37.5	33.3	35.5
	32.8	37.5	39.2	36.5	33.4	35.5
	34.2	31.3	35.5	37.2	33.6	30.6
	35.6	35.2	30.2	37.4	37.5	35.9
	37.3	36.2	33.8	30.4	36.9	38.8
	35.8	33.6	36.4	34.6	--	35.6
	36.9	33.5	33.2	38.4	--	35.0
	35.1	34.7	33.9	--	--	--
	35.4[1]	34.5	34.8	35.8	34.5	35.4
	±0.4	±0.7	±0.9	±0.9	±0.9	±0.8
8	35.4	32.1	35.5	35.0	33.7	38.0
	36.4	37.9	38.4	36.5	33.8	36.8
	34.4	39.1	39.3	37.5	34.4	35.5
	34.6	33.6	36.5	37.4	34.0	31.6
	36.1	34.7	30.4	38.2	38.5	36.3
	38.0	35.5	34.9	32.2	37.5	39.4
	36.1	35.8	37.1	35.7	--	36.4
	37.7	34.9	34.3	38.7	--	35.7
	36.8	36.5	35.0	--	--	--
	36.2	35.6	35.7	36.4	35.3	36.2
	±0.4	±0.7	±0.9	±0.7	±0.9	±0.8
9	36.2	33.7	35.6	34.9	33.3	38.7
	36.2	39.2	38.3	37.1	34.9	36.9
	34.4	40.3	40.0	37.1	33.7	34.9
	34.7	34.4	35.3	38.1	33.2	29.0
	36.5	35.2	30.0	37.7	38.4	34.9
	38.1	35.1	33.2	32.2	37.5	38.3
	37.1	34.4	38.1	36.1	--	36.5
	37.6	35.2	34.3	39.6	--	36.2
	37.1	36.5	35.5	--	--	--
	36.4	36.0	35.6	36.6	35.2	35.7
	±0.4	±0.7	±1.0	±0.8	±0.9	±1.1
10	35.7	33.2	35.2	35.6	33.2	38.3
	35.8	41.0	39.4	38.9	34.3	37.8
	34.7	41.0	41.6	35.7	35.2	36.8
	34.9	35.5	37.1	38.4	34.8	31.8
	37.8	34.3	30.0	37.8	40.0	35.0
	38.3	36.3	33.7	33.9	39.1	39.9
	36.9	35.2	39.1	36.5	--	36.8
	38.0	35.9	34.2	40.6	--	36.3
	36.8	37.4	35.8	--	--	--
	36.5	36.6	36.2	37.2	36.1	36.6
	±0.4	±0.9	±1.2	±0.8	±1.1	±0.8

[1] Results are expressed as means ± SEM for 6-9 mice in each group.

APPENDIX TABLE 7

Effect of exercise duration on weight gain of male Swiss albino mice

Weeks	Time exercised (min)					
	0	20	40	60	120	240
				g		
1-2 (training)[2]	-0.2	-0.6	0.0	0.8	-2.3	1.2
	-0.5	-0.3	0.4	1.0	-2.0	-0.2
	2.5	1.2	1.1	-0.8	-1.5	-0.4
	-1.9	-1.2	0.5	-0.3	-2.4	-1.2
	1.4	2.4	-0.5	0.5	1.9	0.4
	1.3	-0.5	-0.4	0.0	-4.1	0.4
	-1.4	-1.8	0.6	-0.8	--	-1.4
	-0.5	-1.7	0.1	0.1	--	0.0
	0.6	-0.8	-0.7	--	--	--
	0.1[1]	-0.4	0.1	0.1	-1.7	-0.1
	±0.5	±0.4	±0.2	±0.2	±0.8	±0.3
3-6	4.4	2.7	3.6	2.4	3.5	4.0
	5.8	3.7	4.1	2.9	3.9	3.5
	1.5	4.2	5.1	4.2	5.7	5.5
	4.8	3.1	4.3	5.0	3.6	0.0
	4.1	2.2	2.2	3.3	2.7	3.3
	3.3	5.0	4.3	0.8	8.5	6.2
	4.8	5.1	4.4	5.4	--	5.1
	4.2	4.3	3.1	5.3	--	3.0
	4.1	3.8	3.0	--	--	--
	4.1	3.8	3.8	3.7	4.6	3.8
	±0.4	±0.3	±0.3	±0.6	±0.9	±0.7
7-10	0.8	2.1	1.4	2.5	1.0	1.1
	-0.2	4.3	2.0	2.3	1.1	2.1
	2.0	3.7	2.8	0.0	0.5	0.8
	0.9	4.0	1.4	1.7	1.1	3.2
	1.7	0.6	0.0	1.5	3.0	-0.1
	2.1	0.9	0.8	3.5	2.7	1.3
	0.7	0.5	1.2	2.5	--	1.7
	0.9	2.9	2.1	1.9	--	1.1
	1.4	2.1	2.2	--	--	--
	1.1	2.3	1.5	2.0	1.6	1.4
	±0.2	±0.5	±0.3	±0.3	±0.4	±0.3
1-10 (total study)	5.0	4.2	5.0	5.7	2.2	6.3
	5.1	7.7	6.5	6.2	3.0	5.4
	6.0	9.1	9.0	3.4	4.7	5.9
	3.8	5.9	6.2	6.4	2.3	2.0
	7.2	5.2	1.7	5.3	7.6	3.6
	6.7	5.4	4.7	4.3	7.1	7.9
	4.1	3.8	6.2	7.1	--	5.4
	4.6	5.5	5.3	7.3	--	4.1
	6.1	5.1	4.5	--	--	--
	5.4	5.8	5.4	5.7	4.5	5.1
	±0.4	±0.5	±0.6	±0.5	±1.0	±0.6

[1] Results are expressed as means ± SEM for 6-9 mice in each group. [2] During weeks 1 and 2 mice were not exercised for the time duration shown but were on the progressive exercise schedule shown in table 3.

APPENDIX TABLE 8

Effect of exercise duration on weekly food intake of male Swiss albino mice: weeks 1 and 2

Week	Time exercised (min)					
	0	20	40	60	120	240
	g/week					
1 (training)[2]	32.3	33.9	34.5	33.3	32.9	37.0
	35.1	33.2	34.7	34.3	35.2	32.4
	39.4	30.6	36.7	35.5	36.4	35.0
	29.1	32.9	32.8	35.3	34.1	39.9
	37.6	39.1	32.5	36.4	35.2	37.8
	35.0	35.9	32.3	29.8	35.2	36.3
	34.3	37.5	34.5	31.5	--	31.9
	36.1	33.2	32.6	37.9	--	37.4
	39.1	35.7	33.4	--	--	--
	35.3[1] ±1.1	34.7 ±0.9	33.8 ±0.5	34.2 ±0.9	34.8 ±0.5	36.0 ±1.0
2 (training)[3]	36.4	33.4	31.3	33.6	30.8	35.4
	32.7	36.0	38.7	36.6	32.6	35.2
	37.6	34.2	35.8	35.2	39.8	35.6
	32.4	30.4	33.9	33.9	32.9	34.9
	38.6	40.3	29.6	38.2	40.6	37.1
	35.0	34.8	34.3	32.7	34.9	31.9
	34.2	35.8	32.0	29.4	--	31.0
	36.0	31.7	32.8	34.4	--	32.7
	37.3	33.6	32.5	--	--	--
	35.6 ±0.7	35.4 ±0.9	33.4 ±0.9	34.2 ±0.9	35.3 ±1.6	34.2 ±0.7

[1] Results are expressed as means ± SEM for 6-9 mice in each group.
[2,3] During weeks 1 and 2, mice were not exercised for the time duration shown, but were on the progressive exercise schedule shown in table 3.

APPENDIX TABLE 9

Effect of exercise duration on weekly food intake of male Swiss albino mice: weeks 3 to 6

Week	Time exercised (min)					
	0	20	40	60	120	240
	g/week					
3	40.0	34.5	35.6	33.4	38.8	43.2
	39.0	38.4	42.6	41.6	39.6	37.2
	41.3	43.0	40.3	42.4	42.7	41.4
	37.7	34.6	38.7	42.9	37.9	38.5
	42.3	42.5	33.2	40.6	44.4	43.0
	36.0	38.3	39.4	29.0	46.2	41.9
	37.7	39.0	37.2	34.5	--	42.6
	43.9	38.2	34.6	42.1	--	42.3
	43.7	36.3	38.4	--	--	--
	40.2[1]	38.3	37.8	38.3	41.6	41.3
	±0.9	±1.0	±1.0	±1.8	±1.4	±0.8
4	38.8	36.1	32.2	33.7	38.1	42.9
	40.4	38.5	43.3	38.0	38.5	41.0
	42.2	41.7	41.6	43.9	42.6	42.6
	31.0	36.3	39.6	45.0	41.9	38.4
	45.1	42.5	37.2	44.0	42.7	47.0
	33.8	38.4	37.5	35.6	42.6	43.0
	37.1	37.6	41.4	37.7	--	41.8
	42.4	39.0	39.1	41.1	--	45.3
	44.2	41.1	38.9	--	--	--
	39.4	39.0	39.0	39.9	41.1	42.7
	±1.6	±0.8	±1.1	±1.5	±0.9	±0.9
5	35.7	34.4	29.6	32.7	34.1	41.5
	35.2	33.4	40.2	39.7	36.0	40.6
	37.7	37.6	37.7	37.7	38.0	38.6
	35.9	32.3	35.5	42.4	36.7	34.9
	40.6	37.5	30.8	38.1	41.9	41.1
	34.4	34.0	35.8	30.8	38.3	41.2
	36.0	35.9	38.1	33.3	--	39.1
	38.2	30.5	34.0	37.5	--	39.4
	37.9	34.5	35.7	--	--	--
	36.8	34.4	35.3	36.5	37.5	39.5
	±0.6	±0.8	±1.1	±1.4	±1.1	±0.8
6	38.5	31.3	33.9	34.3	33.8	41.2
	37.0	34.7	41.3	37.5	35.4	36.6
	37.4	36.0	41.0	38.8	39.3	41.2
	34.7	34.1	38.9	41.3	37.6	32.4
	37.5	37.0	31.4	38.4	38.4	38.6
	33.3	35.7	37.1	33.7	38.7	40.4
	35.8	35.6	38.6	36.9	--	38.6
	38.1	34.8	35.5	39.9	--	41.0
	39.0	35.6	37.0	--	--	--
	36.8	35.0	37.2	37.6	37.2	38.7
	±0.6	±0.5	±1.1	±0.9	±0.9	±1.1

[1] Results are expressed as means ± SEM for 6-9 mice in each group.

Effect of exercise duration on weekly food intake of male Swiss albino mice: weeks 7 to 10

Week	Time exercised (min)					
	0	20	40	60	120	240
	g/week					
7	39.0	33.1	33.6	36.4	35.2	40.4
	36.1	34.0	37.5	39.9	34.7	40.1
	39.4	40.6	41.0	40.8	38.3	40.7
	36.5	35.5	36.5	40.5	35.8	34.0
	40.9	40.5	32.9	41.2	42.9	40.8
	35.9	37.5	38.5	32.7	41.4	39.7
	36.0	35.4	35.4	37.6	--	40.4
	39.4	37.6	36.4	37.3	--	39.1
	39.1	35.3	37.4	--	--	--
	38.0[1]	36.6	36.6	38.3	38.0	39.4
	±0.6	±0.9	±0.8	±1.0	±1.4	±0.8
8	42.1	35.1	35.6	36.9	41.3	40.5
	36.3	36.8	39.5	38.6	37.8	41.4
	40.9	41.6	41.8	40.9	41.6	40.3
	38.0	38.0	41.0	43.4	38.2	35.9
	43.0	38.4	33.2	41.2	44.7	40.2
	33.8	35.8	39.3	36.3	41.8	39.6
	36.5	37.3	37.6	37.2	--	41.9
	43.1	39.3	36.4	38.3	--	39.9
	40.9	37.8	37.2	--	--	--
	39.4	37.8	38.0	39.1	40.9	40.0
	±1.1	±0.6	±0.9	±0.9	±1.0	±0.6
9	41.7	34.8	35.4	37.1	36.2	41.8
	37.4	36.8	40.5	37.6	40.5	41.7
	39.4	39.5	42.9	36.9	37.1	36.3
	37.1	37.6	36.2	39.0	35.8	32.5
	41.7	38.6	33.0	39.6	41.3	40.2
	32.7	35.7	35.5	34.2	38.3	38.3
	37.1	34.7	39.3	38.1	--	40.1
	40.7	36.7	37.2	39.6	--	39.9
	40.6	36.4	38.8	--	--	--
	38.7	36.7	37.6	37.8	38.2	38.8
	±1.0	±0.5	±1.0	±0.6	±0.9	±1.1
10	39.2	32.1	32.7	36.3	34.7	38.8
	34.0	34.2	40.5	39.0	33.6	39.7
	38.2	39.1	43.7	34.8	37.6	43.4
	33.9	36.5	38.1	39.3	38.1	34.7
	36.9	36.7	32.4	38.5	41.5	38.7
	33.5	35.0	35.6	32.6	38.6	38.9
	33.4	35.3	43.0	35.7	--	38.1
	38.5	37.9	35.6	39.0	--	38.7
	36.9	36.5	36.3	--	--	--
	36.0	35.9	37.5	36.9	37.3	38.9
	±0.8	±0.7	±1.4	±0.9	±1.2	±0.8

[1] Results are expressed as means ± SEM for 6-9 mice in each group.

APPENDIX TABLE 11

Effect of exercise duration on total food intake of male Swiss albino mice

Weeks	Time exercised (min)					
	0	20	40	60	120	240
	g total					
1-2 (training)[2]	68.7	67.3	65.8	66.9	63.7	72.4
	67.8	69.2	73.4	70.9	67.8	67.6
	77.0	64.8	72.5	70.7	76.2	70.6
	61.5	63.3	66.7	69.2	67.0	74.8
	76.2	79.4	62.1	74.6	75.8	74.9
	70.0	70.7	66.6	62.5	70.1	68.2
	68.5	73.3	66.5	60.9	--	62.9
	72.1	64.9	65.4	72.3	--	70.1
	76.4	69.3	65.9	--	--	--
	70.9[1]	69.1	67.2	68.5	70.1	70.2
	±1.7	±1.7	±1.2	±1.7	±2.0	±1.4
3-6	153.0	136.3	131.3	134.1	144.8	168.8
	151.0	145.0	167.4	156.8	149.5	155.4
	158.6	158.3	160.6	162.8	162.9	163.8
	139.3	137.3	152.7	171.6	154.1	144.2
	165.5	159.5	132.6	161.1	167.4	169.7
	137.5	146.4	149.8	129.1	165.8	166.5
	146.6	148.1	155.3	142.4	--	162.1
	162.6	142.5	143.2	160.6	--	168.0
	164.8	147.5	150.0	--	--	--
	153.3	146.8	149.2	152.3	157.4	162.3
	±3.5	±2.7	±4.0	±5.4	±3.8	±3.1
7-10	162.0	135.1	137.3	146.7	147.4	161.5
	143.8	141.8	158.0	155.1	146.6	162.9
	158.2	160.8	169.4	153.4	154.6	160.7
	145.5	147.6	151.8	162.2	147.9	137.1
	162.5	154.2	131.5	160.5	170.4	159.9
	135.9	144.0	148.9	135.8	160.1	156.5
	143.0	142.7	155.3	148.6	--	160.5
	161.7	151.5	145.6	154.2	--	157.6
	157.5	146.0	149.7	--	--	--
	152.2	147.1	149.7	152.1	154.5	157.1
	±3.4	±2.5	±3.7	±3.0	±3.8	±2.9
1-10 (total study)	383.7	338.7	334.4	347.7	355.9	402.7
	362.6	356.0	398.8	379.9	363.9	385.9
	393.8	383.9	402.5	386.9	393.7	395.1
	346.3	348.2	371.2	403.0	377.8	356.1
	404.2	393.1	326.2	396.2	413.6	404.5
	343.4	361.1	365.3	327.4	396.0	391.2
	358.1	364.1	377.1	351.9	--	385.5
	396.4	358.9	354.2	387.1	--	395.7
	398.7	362.8	365.6	--	--	--
	376.4	363.0	366.1	372.9	382.0	389.6
	±7.9	±5.6	±8.6	±9.5	±9.1	±5.4

[1] Results are expressed as means ± SEM for 6-9 mice in each group. [2] During weeks 1 and 2 mice were not exercised for the time duration shown but were on the progressive exercise schedule shown in table 3.

APPENDIX TABLE 12

Effect of exercise on food/body weight ratio of male Swiss albino mice: weeks 1 and 2[1]

Week	Time exercised (min)					
	0	20	40	60	120	240
	g food/g body wt					
1 (training)[3]	0.19	0.18	0.17	0.16	0.19	0.18
	0.18	0.17	0.20	0.19	0.18	0.17
	0.19	0.20	0.17	0.20	0.22	0.19
	0.19	0.19	0.19	0.20	0.18	0.20
	0.20	0.21	0.17	0.18	0.20	0.19
	0.16	0.18	0.20	0.15	0.20	0.18
	0.17	0.19	0.16	0.17	--	0.21
	0.20	0.19	0.17	0.18	--	0.18
	0.20	0.16	0.18	--	--	--
	0.19[2] ±0.00	0.19 ±0.00	0.18 ±0.00	0.18 ±0.00	0.19 ±0.01	0.19 ±0.00
2 (training)[4]	0.18	0.18	0.15	0.16	0.19	0.18
	0.19	0.17	0.18	0.16	0.19	0.18
	0.19	0.18	0.18	0.20	0.21	0.20
	0.15	0.18	0.18	0.20	0.20	0.19
	0.20	0.19	0.19	0.19	0.18	0.21
	0.15	0.18	0.19	0.17	0.22	0.19
	0.17	0.18	0.18	0.19	--	0.20
	0.18	0.19	0.19	0.17	--	0.20
	0.20	0.19	0.18	--	--	--
	0.18 ±0.01	0.18 ±0.00	0.18 ±0.00	0.18 ±0.01	0.20 ±0.01	0.19 ±0.00

[1] Ratio calculated as average daily food consumption over body weight for that week. [2] Results are expressed as means ± SEM for 6-9 mice in each group. [3,4] During weeks 1 and 2, mice were not exercised for the time duration shown, but were on the progressive exercise schedule shown in table 3.

APPENDIX TABLE 13

Effect of exercise duration on food/body weight ratios of male Swiss albino mice: weeks 3-6[1]

Week	Time exercised (min)					
	0	20	40	60	120	240
	g food/g body wt					
3	0.18	0.17	0.17	0.16	0.19	0.18
	0.18	0.16	0.18	0.17	0.19	0.17
	0.19	0.18	0.16	0.19	0.21	0.19
	0.18	0.17	0.17	0.19	0.18	0.20
	0.19	0.19	0.17	0.18	0.18	0.19
	0.15	0.17	0.19	0.15	0.20	0.18
	0.16	0.18	0.16	0.17	--	0.19
	0.18	0.18	0.17	0.17	--	0.19
	0.19	0.16	0.18	--	--	--
	0.18[2] ±0.0	0.17 ±0.0	0.17 ±0.0	0.17 ±0.0	0.19 ±0.0	0.19 ±0.0
4	0.17	0.18	0.14	0.15	0.17	0.17
	0.17	0.16	0.17	0.16	0.18	0.17
	0.19	0.16	0.16	0.18	0.20	0.19
	0.15	0.17	0.17	0.19	0.19	0.19
	0.19	0.19	0.18	0.18	0.17	0.20
	0.14	0.16	0.17	0.17	0.18	0.18
	0.15	0.17	0.17	0.17	--	0.18
	0.17	0.17	0.18	0.16	--	0.19
	0.19	0.18	0.18	--	--	--
	0.17 ±0.0	0.17 ±0.0	0.17 ±0.0	0.17 ±0.0	0.18 ±0.0	0.18 ±0.0
5	0.15	0.16	0.13	0.14	0.15	0.16
	0.14	0.14	0.16	0.16	0.16	0.16
	0.16	0.14	0.14	0.15	0.17	0.16
	0.16	0.15	0.14	0.17	0.16	0.17
	0.16	0.16	0.15	0.15	0.16	0.17
	0.14	0.14	0.16	0.15	0.15	0.16
	0.15	0.15	0.15	0.14	--	0.16
	0.15	0.13	0.15	0.14	--	0.17
	0.16	0.15	0.16	--	--	--
	0.15 ±0.0	0.15 ±0.0	0.15 ±0.0	0.15 ±0.0	0.16 ±0.0	0.16 ±0.0
6	0.16	0.14	0.14	0.15	0.15	0.16
	0.15	0.13	0.16	0.15	0.15	0.15
	0.16	0.14	0.15	0.15	0.16	0.16
	0.14	0.15	0.15	0.16	0.16	0.16
	0.15	0.16	0.15	0.15	0.15	0.16
	0.13	0.14	0.16	0.16	0.15	0.15
	0.14	0.15	0.14	0.15	--	0.16
	0.15	0.15	0.16	0.15	--	0.17
	0.16	0.14	0.16	--	--	--
	0.15 ±0.0	0.14 ±0.0	0.15 ±0.0	0.15 ±0.0	0.15 ±0.0	0.16 ±0.0

[1] Ratios calculated as average daily food consumption over body weight for that week.
[2] Results are expressed as means ± SEM for 6-9 mice in each group.

APPENDIX TABLE 14

Effect of exercise duration on food/body weight ratios of male Swiss albino mice: weeks 7-10[1]

Week	Time exercised (min)					
	0	20	40	60	120	240
	g food/g body wt					
7	0.16	0.15	0.14	0.15	0.15	0.16
	0.14	0.13	0.14	0.15	0.15	0.16
	0.17	0.15	0.15	0.16	0.16	0.16
	0.15	0.16	0.15	0.15	0.15	0.16
	0.16	0.16	0.15	0.16	0.16	0.16
	0.14	0.15	0.16	0.15	0.16	0.15
	0.14	0.15	0.14	0.15	--	0.16
	0.15	0.16	0.16	0.14	--	0.16
	0.16	0.14	0.16	--	--	--
	0.15[2] ±0.0	0.15 ±0.0	0.15 ±0.0	0.15 ±0.0	0.16 ±0.0	0.16 ±0.0
8	0.17	0.16	0.14	0.15	0.17	0.15
	0.14	0.14	0.15	0.15	0.16	0.16
	0.17	0.15	0.15	0.15	0.17	0.16
	0.15	0.16	0.16	0.16	0.16	0.16
	0.17	0.16	0.16	0.15	0.16	0.16
	0.13	0.14	0.16	0.16	0.16	0.14
	0.14	0.15	0.14	0.15	--	0.16
	0.16	0.16	0.15	0.14	--	0.16
	0.16	0.15	0.15	--	--	--
	0.16 ±0.0	0.15 ±0.0	0.15 ±0.0	0.15 ±0.0	0.16 ±0.0	0.16 ±0.0
9	0.16	0.15	0.14	0.15	0.15	0.15
	0.15	0.13	0.15	0.14	0.16	0.16
	0.16	0.14	0.15	0.14	0.16	0.15
	0.15	0.16	0.15	0.15	0.15	0.16
	0.16	0.16	0.16	0.15	0.15	0.16
	0.12	0.14	0.15	0.15	0.14	0.14
	0.14	0.14	0.15	0.15	--	0.16
	0.15	0.15	0.15	0.14	--	0.16
	0.15	0.14	0.16	--	--	--
	0.15 ±0.0	0.15 ±0.0	0.15 ±0.0	0.15 ±0.0	0.15 ±0.0	0.15 ±0.0
10	0.16	0.14	0.13	0.14	0.15	0.14
	0.13	0.12	0.15	0.14	0.14	0.15
	0.16	0.14	0.15	0.14	0.15	0.17
	0.14	0.15	0.15	0.15	0.16	0.15
	0.14	0.15	0.15	0.14	0.15	0.16
	0.12	0.14	0.15	0.14	0.14	0.14
	0.13	0.14	0.16	0.14	--	0.15
	0.14	0.15	0.15	0.14	--	0.15
	0.14	0.14	0.14	--	--	--
	0.14 ±0.0	0.14 ±0.0	0.15 ±0.0	0.14 ±0.0	0.15 ±0.0	0.15 ±0.0

[1] Ratios calculated as average daily food consumption over body weight for that week.
[2] Results are expressed as means ± SEM for 6-9 mice in each group.

APPENDIX TABLE 15

Effect of exercise duration on measurements of male Swiss albino mice at sacrifice

Measurement	Time exercised (min)					
	0	20	40	60	120	240
Body weight, g	36.7	34.8	35.8	34.9	33.5	37.4
	35.9	39.3	39.2	37.9	36.0	37.9
	34.6	40.2	41.4	35.6	34.9	34.4
	34.5	34.5	37.8	37.5	34.1	30.9
	37.9	34.9	29.7	38.8	40.7	34.3
	37.9	36.3	34.6	32.7	38.7	39.2
	36.9	35.4	37.7	36.8	--	36.0
	39.4	35.2	33.9	39.6	--	36.1
	36.6	35.9	36.4	--	--	--
	36.7[1] ±0.5	36.3 ±0.7	36.3 ±1.1	36.7 ±0.8	36.3 ±1.1	35.8 ±0.9
Carcass weight, g	31.5	29.7	31.4	30.1	28.8	32.4
	31.0	34.8	34.6	33.4	30.6	32.0
	29.6	34.9	35.0	30.3	29.8	30.4
	29.7	30.1	32.0	32.4	29.3	25.8
	32.1	30.2	26.1	32.5	34.5	30.3
	33.3	31.5	29.4	28.5	33.3	34.0
	31.6	30.6	32.9	31.2	--	31.3
	33.3	30.6	29.8	34.6	--	30.9
	31.4	30.9	31.4	--	--	--
	31.5 ±0.4	31.5 ±0.6	31.4 ±0.9	31.6 ±0.7	31.0 ±0.9	30.9 ±0.8
Heart weight, mg	171	132	145	157	181	171
	148	158	149	178	153	168
	141	189	190	162	134	175
	150	156	162	191	139	132
	189	143	131	202	161	151
	151	175	131	126	152	162
	142	181	148	154	--	147
	174	140	146	158	--	142
	154	142	170	--	--	--
	158 ±5.4	157 ±6.7	152 ±6.3	166 ±8.4	153 ±6.8	156 ±5.4
Epididymal fat pad weight, g	0.59	0.65	0.65	0.65	0.40	0.45
	0.78	0.89	0.83	0.60	0.54	0.67
	0.49	1.14	0.98	0.56	0.47	0.46
	0.72	0.58	0.64	0.76	0.80	0.37
	0.62	0.66	0.44	0.50	0.79	0.56
	1.10	0.75	0.61	0.51	0.54	0.95
	0.77	0.70	0.75	0.69	--	0.52
	0.67	0.90	0.66	1.20	--	0.56
	0.58	0.54	0.72	--	--	--
	0.7 ±0.06	0.7 ±0.06	0.7 ±0.05	0.7 ±0.08	0.6 ±0.07	0.6 ±0.06

[1] Results are expressed as means ± SEM for 6-9 mice in each group after the 10-week trial.

APPENDIX TABLE 16

Effect of exercise duration on body composition of male Swiss albino mice at sacrifice

Measurement (% of carcass)	Time exercised (min)					
	0	20	40	60	120	240
Dry matter, %	32.1	37.4	34.5	34.9	30.9	28.3
	35.6	37.2	36.2	32.8	31.5	31.5
	34.8	39.0	40.7	32.0	32.3	31.2
	37.9	32.6	31.9	34.3	39.0	29.0
	33.2	35.3	32.8	30.7	33.8	32.2
	40.1	36.2	34.9	33.3	30.8	37.8
	35.2	36.4	27.7	34.2	--	26.4
	32.0	38.8	35.6	38.9	--	32.3
	35.4	34.4	35.0	--	--	--
	35.2[1]	36.4	34.4	33.9	33.0	31.1
	±0.9	±0.7	±1.2	±0.9	±1.3	±1.2
Body water, %	67.9	62.6	65.5	65.1	69.1	71.7
	64.4	62.8	63.8	67.2	68.5	68.5
	65.2	60.9	59.3	68.0	67.7	68.8
	62.0	67.4	68.1	65.7	60.9	71.0
	66.8	64.7	67.2	69.3	66.2	67.8
	59.8	63.8	65.1	66.7	69.2	62.2
	64.7	63.6	72.3	65.8	--	73.6
	68.0	61.2	64.4	61.1	--	67.7
	64.6	65.6	65.0	--	--	--
	64.8	63.6	65.6	66.1	66.9	68.9
	±0.9	±0.7	±1.2	±0.9	±1.3	±1.2
Fat, %	09.2	14.3	13.8	12.8	10.7	09.2
	14.3	16.3	15.8	15.3	10.2	15.3
	21.4	18.9	16.3	10.2	09.2	11.8
	17.4	12.3	11.2	13.8	19.4	09.7
	12.8	14.8	11.2	10.2	13.3	10.7
	22.0	14.8	12.3	12.8	09.2	16.8
	14.3	16.8	12.8	15.8	--	11.2
	13.8	21.4	18.4	20.4	--	11.2
	16.3	16.8	15.8	--	--	--
	15.7	16.3	14.2	13.9	12.0	12.0
	±1.4	±0.9	±0.8	±1.2	±1.6	±0.9
Protein, %	13.3	12.7	12.0	13.0	14.1	13.1
	12.7	13.7	13.7	15.1	13.7	13.2
	15.7	14.1	15.4	17.5	19.2	13.6
	12.0	13.9	14.2	13.3	14.6	12.0
	12.0	12.3	12.9	12.3	13.9	13.0
	11.6	11.6	13.3	13.6	12.3	13.8
	14.1	13.4	13.7	13.2	--	16.2
	12.1	11.7	13.9	13.3	--	12.3
	13.6	12.4	13.7	--	--	--
	13.0	12.9	13.7	13.9	14.6	13.4
	±0.4	±0.3	±0.3	±0.6	±1.0	±0.4

[1] Results are expressed as means ± SEM for 6-9 mice in each group after the 10-week trial.

EFFECT OF EXERCISE DURATION ON FOOD INTAKE, BODY WEIGHT, AND BODY COMPOSITION OF MICE

by

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ABSTRACT

The effects of exercise duration on food intake, body weight, and body composition were studied in adult male Swiss Albino mice. Fifty-four mice were randomly assigned to one of six treatment groups: a control group (not exercised) or exercise groups receiving either 20, 40, 60, 120, 240 minutes of treadmill exercise 5 days/week for 8 weeks.

Throughout the study mice exercised 20 or 40 minutes tended to consume less food than control animals; and mice exercised more than 60 minutes tended to have greater food intakes proportional to the length of time exercised, but these differences were not statistically significant.

Analyses of body composition of the mice revealed that mice exercised for 240 minutes had significantly lower ($P < 0.05$) percent body fat and significantly higher percent body water ($P < 0.05$) than mice exercised either 0 or 20 minutes per day. Percent body protein, heart weights, and epididymal fat pad weights were not significantly different among treatment groups.