REBOUND WEIGHT GAIN FOLLOWING EXERCISE OR MODERATE CALORIC RESTRICTION IN RATS

by

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

CHAPTER 1: REVIEW OF LITERATURE

I. THE DEVELOPMENT OF OBESITY

Obesity is the result of excess energy stores in the form of fat, mainly triglycerides, in adipose cells. Obesity may be characterized by an increase in fat cell number, or hyperplasia, and/or an increase in fat cell size, or hypertrophy. At one time the number of fat cells was believed to be fixed early in life. We now know that additional fat cells may also be formed under certain circumstances during adulthood.

In one study (1), adult rats of various strains and both sexes became obese when fed a high fat diet for 5 mo. Increases in both adipocyte size and number occurred, with the increases in cell number discovered first in the retroperitoneal fat pad. The researchers suggested that adipocytes had to reach a certain critical size before new adipocytes were formed. Some differences were observed among strains and between sexes. Male Osborne-Mendel and Sprague-Dawley rats showed increases in both adipocyte size and number; however, obese female Zucker rats showed no increase in adipocyte size, and lean female Zucker rats showed no increase in adipocyte number in the subcutaneous depot. This study shows that adult onset obesity may lead to a permanent increase in adipocyte number.

In another study (2), 11-wk-old rats were put on a food restriction regimen for 15 wk and then refed for an additional 5 wk. During refeeding, both hypertrophy and hyperplasia were observed. Female rats 5 mo of age fed a high fat diet until an age of 12 mo showed a

significant increase in both fat cell number and size over controls (3). These studies indicate that even though fat cell number may be normal in early life, an animal can still develop more fat cells later in life and exhibit hyperplastic obesity.

The aging process also seems to be associated with an increase in body fat. On an ad libitum rat chow diet, body fat of rats increased from 19.5 to 24.6 to 28.7% at ages of 6, 15, and 27 mo, respectively (4). Body weight increased at 6 and 15 mo, but decreased at 27 mo. The increases in body fat and weight may have been due to a decreasing metabolic rate, a more sedentary lifestyle, and other aspects of the aging process.

Certain types of diets may also be more likely to cause obesity. Rats fed diets containing 42% fat or 11% fat consumed a similar number of calories (5). However, at the end of 60 wk, the rats eating the high fat diet had gained 880 g and were 51% body fat. The rats on the low fat diet had gained only 666 g and were 30% body fat.

Similar results have been found in humans (6). Lean men overeating a high fat diet gained 30 pounds in 3 mo, but men eating even more calories on a high carbohydrate diet needed 7 mo to gain the same amount of weight.

Dietary sugar may also induce obesity. In a study cited but unpublished at this time (7), rats were placed on one of three diets: high fat (45% fat, 0% sugar), high sugar (55% sugar, 8% fat), and control (11% fat). At the end of the study, the rats on the high fat diet weighed 806 g and were 46% body fat. Those on the control diet were 647 g and 31% body fat. Thus, the source of calories is an

important determinant of diet-induced obesity.

In humans, the causes of obesity are similar to those in rats. Of particular importance are the high-fat/high-sugar diets and sedentary lifestyles in countries were obesity is prevalent. Humans eat for reasons other than hunger itself, such as boredom, depression, sensory satisfaction, or social interaction.

Several methods and models are used in studying obesity. In rats, certain strains such as the genetically obese Zucker rat are often employed in research studies. Osborne-Mendel rats are easily made obese on a highly palatable diet. Methods used to induce obesity in rats include ventromedial hypothalamic lesions induced electrolytically or by using gold thioglucose. Other approaches include feeding cafeteria and high-fat diets, decreasing litter size, and enforced inactivity.

Various methods have been used to induce weight loss in rats. Those include increasing litter size, decreasing feed intake, and exercise such as swimming and treadmill running. In feed restriction, the level of restriction varies, depending on the purpose of the study, from complete fasting or starvation to mild caloric restriction.

In humans, the influence of genetics and the environment on the development of obesity is examined through adoption studies, family studies, activity patterns, and clinical studies. In general, the tendency to be obese is believed to be inherited, but a faulty nutritional environment is needed to actually become obese.

The most popular methods to induce weight loss in humans are

exercise and diets reduced in calories. Other methods include appetite suppressants, fad diets, fiber pills, and other gimmicks available to the public.

II. EFFECTS OF MODERATE CALORIC RESTRICTION ON WEIGHT LOSS IN RATS

Weight is lost by maintaining a negative caloric balance. This is accomplished by reducing caloric intake, increasing the number of calories burned by the body, or both. The effectiveness of weight loss by reducing caloric intake will be examined below.

A. Weight Loss at Different Levels of Caloric Restriction

Different levels of caloric restriction will result in different rates of weight loss. In a study by Forsum et al. (8), one group of 7-wk-old sedentary male Sprague-Dawley rats was given feed ad libitum while restricted rats were given only 10 g/d. Since the ad libitum fed rats consumed 18.7 g/d, they gained 159 g compared to the smaller 36 g gain of the restriced rats over the 28-31 d period.

Oscai and Holloszy (9) restricted feed intake in 11.5-mo-old sedentary obese rats to match the weight loss of exercising obese rats. A group of sedentary control rats was also included. Over an 18-wk period, the feed-restricted sedentary rats consumed 6315 kcal compared to the 9985 kcal intake of sedentary control rats. Put another way, the caloric intake of the restricted rats was 63.2% of the control level. The feed-restricted rats lost 118 g or approximately 25% of their initial weight.

In another study (10), male Sprague-Dawley rats made obese by a

high-fat diet were switched to Purina Lab Chow and fed at a level of 50% of what the normal weight control rats were consuming of the chow diet. The obese rats lost 131 g in 21 d, which was a 16.1% reduction in weight.

Askew and Hecker (11) studied the effects of several levels of feed restriction on weight loss in 6.5-wk-old male rats. Over a 12-wk period, dietary restrictions at 84, 73, or 65% those of the control level led to a reduction in weight gain so that final weights were 93.8, 81.4, and 72.4%, respectively, of control rats.

Harris and Martin (12) placed 14-wk-old lean female rats on a restricted diet at 40% of that eaten ad libitum by control rats. After 22 d, the rats had lost 60 g, approximately 27% of their initial body weight. This study and the others previously described show that the rate and amount of weight loss depends on the severity and duration of caloric restriction.

B. Fat Weight vs. Lean Weight Loss

Simple measurement of weight loss does not indicate whether adipose tissue or lean body mass is lost. Therefore, it is important to determine whether moderate caloric restriction leads to the loss of fat weight or substantial amounts of lean weight as well.

Oscai and Holloszy (9) restricted feed intake in sedentary rats so that their weight loss would match that of exercising rats. Over the 18-wk period, the restricted rats consumed 70.2% as much feed as the exercising rats in order to maintain similar weights. Both groups of animals lost 182 g or about 25% of their initial body weights. The

feed-restricted rats lost about twice as much protein as the exercised rats; furthermore, they only lost 79% as much fat.

In a study by Harris and Martin (12), adult female rats consuming 40% of their normal feed intake lost 60 g of body weight in 22 d. Of this loss, 37% was fat and 20% was body protein. These studies suggest that a significant amount of lean mass is lost during moderate caloric restriction.

C. Changes in Basal Metabolic Rate

Basal metabolic rate (BMR) and specific dynamic action (SDA) are affected by the level of caloric intake in man and animals. This is a beneficial adaptation mechanism during starvation where a lowered metabolic rate could probably prolong survival. However, in deliberate attempts at weight loss, a downward adjustment of BMR in response to reduced caloric intake can make weight reduction more difficult.

Forsum et al. (8) divided male Sprague-Dawley rats into two groups: ad libitum fed rats, which consumed 18.7-18.9 g/d and those restricted to 10 g/d. This feeding program lasted 28-31 d and was followed by a 24 h fast for all rats. Rats were analyzed for body composition or placed in a respiration and then a metabolic chamber. The restricted rats had a BMR 30% lower than the ad libitum fed rats. The SDA of the ad libitum fed rats was 5% of the energy consumed compared to only 3% in the calorie restricted rats. This was a small yet statistically significant difference. From body composition measurements, rats restricted in feed intake before the calorimetry study were found to mobilize more protein and less fat compared to

rats fed ad libitum until the calorimetry and body composition studies. This study also found no difference in energy efficiency during activity unlike another study (13), which found that energy restriction increased the efficiency of physical activity. Thus, moderate caloric restriction may cause a decrease in BMR, which would make it difficult to continue losing weight at a consistent rate, and fewer calories would be needed to maintain a new, lower body weight than would be expected.

D. Adipocyte Number and Size

Several studies have been conducted to examine the effects of caloric restriction on the weight of fat pads, fat cell size, and fat cell number. Most research suggests that fat cells, once formed, cannot be eliminated by weight loss later in life.

In a study by Askew and Hecker (11), 6.5-wk-old male rats were subjected to different levels of feed restriction. Rats subjected to a 35% restriction in feed intake weighed 28% less than controls and had 30% lighter epididymal fat pat pads. Energy restriction had a greater effect reducing fat cell number than fat cell size, but this data might be attributed to the fact that energy retriction began at a young age in the rats.

A study on 11-wk-old obese male rats (2) showed that fat cell size actually increased when rats were fed diets 25 or 50% less than ad libitum fed controls for 15 wk. This suprising result was attributed to the meal eating pattern of the rats, which caused a rise in lipoprotein lipase activity and in serum insulin. In contrast,

another study (14) showed that fat cell size decreased during a greater than 40% restriction in feed intake.

Since fat cell number cannot be decreased in adulthood by weight loss, caloric restriction most likely causes weight loss by reducing the size of fat cells.

E. Effects on Hormone, Enzyme, and Substrate Levels

Levels of various substances in the blood and tissues are changed when weight loss occurs by moderate caloric restriction. These levels reflect the changes in lipid and glucose metabolism that occur with weight reduction and caloric restriction.

In one study (15), sedentary feed-restricted lean male Wistar rats 6 to 8-wk-old were pair weighed to a group of exercising rats fed ad libitum. The results showed that blood glucose, blood lactate, plasma free fatty acid (FFA) concentration, and adipose tissue FFA concentration in the feed-restricted rats were similar to those of sedentary ad libitum fed controls. Tissue lactate levels were slightly lower than controls, but differences were not significant.

Another study (12) found that circulating levels of L-3,3',5-triiodothyroxine were significantly decreased in feed-restricted lean female rats. Serum thyroxine, L-3,3',5'-triiodothyronine, insulin, corticosterone, and FFA levels were not affected by changes in body composition.

F. Rebound Weight Gain After Dietary Restriction

Often weight lost through dietary restriction, is regained when the restriction period is discontinued. The effects of refeeding after weight loss have been studied in animals.

Obese male Sprague-Dawley 150-d-old rats were subjected to two cycles of restriction and refeeding (10). After a dietary restriction to 50% of what control rats were consuming, the rats were allowed free access to a high fat diet. In the first restriction phase, the rats lost 131 g in 21 d, while in the second phase they lost 133 g in 46 d. Weight was also regained more rapidly after subsequent attempts at weight loss. Rats regained the weight in 46 d in the first cycle, but required only 14 d in the second cycle. Feed efficiency was higher in the restricted animals than controls during feed restriction and refeeding cycles. Furthermore, feed efficiency was even greater during the second cycle of restriction and refeeding than in the first cycle. There was no difference between the obese controls and obese cycling rats in body composition or insulin levels. No differences in cell size were observed among the adipocytes from the epididymal, retroperitoneal, and inguinal fat pads of the obese rats, but the obese cycling group did have an increased cell number and lipoprotein lipase activity in the retroperitoneal pad.

In another study (12) adult female rats fed 40 % of their usualintake lost 60 g from their original weight of 210 g in 22 d. Fat accounted for 37% of this loss while 20% was body protein. Upon refeeding, the rats were hyperphagic and regained the weight quickly. In 6 d, 86% of the fat was repleted, but 13 d were needed to replace

lost protein to normal levels. Body weight reached control levels after 12 d of refeeding. Hyperphagia became less pronounced at 6 d when most of the body fat was restored, but feed consumption did not return to control levels until day 12 when body weight returned to the control weight. Feed efficiency increased during the first 6 d of refeeding when fat was being replaced, but decreased when protein was being replaced.

Somewhat different results were found when using male rats. In one study (16), 84-wk-old male rats who had lost 21% of their body weight by feed restriction, epididymal and perirenal fat pad weights did not return to normal until body weight was back to contol levels. Thus, male rats took longer to restore body fat stores compared to female rats relative to the rate of total weight regain. Another study (17) found that fasted rats who had lost 25% of their body weight required 8 days to restore both fat and body weight to original levels.

In yet another study on refeeding (18), lean and obese female Zucker rats were subjected to four cycles of feed restriction and refeeding. A cycle consisted of 3 wk of feed restriction followed by 3 wk of ad libitum intake. After the fourth and final restriction feeding, both the cycling lean and the cycling obese had lower body weights and parametrial and retroperitoneal fat pad weights than the control ad libitum fed lean and obese rats. After the final refeeding period, the cycling lean and control lean rats had similar values for these measurements while the cycling obese rats still maintained significantly lower values than the control obese group. The cycling

lean rats showed a systematic pattern of weight loss and gain, recovering 70% of lost weight in the first week of each refeeding period. Even though the two lean groups consumed the same amount of feed, the cycling group gained more than the control group. In contrast, the cycling obese rats did not lose weight the first restriction period and gained significantly more weight than all other rats during the first refeeding period. In the subsequent restriction cycles, the obese rats did lose weight, but they never gained enough to catch up with the obese controls even though they always gained more than any other group of rats during the refeeding periods. During the refeeding periods, the cycling rats had greater feed efficiency ratios than their respective controls. These studies indicate that when rats have lost weight by caloric restriction, they are more efficient at regaining weight than rats that have never lost weight.

III. EFFECTS OF EXERCISE ON WEIGHT LOSS IN RATS

Another means to reduce body weight is to increase the number of calories expended by increasing physical activity. Arguments in favor of exercise over caloric restriction alone contend that it prevents loss of lean mass, counteracts the reduction in BMR, and more favorably changes the plasma lipid profile in comparison to caloric restriction.

A. Exercise, Weight Loss, and Changes in Body Composition

In a study by Oscai and Holloszy (9), 11.5-mo-old obese rats weighing about 706 g were subjected to an exercise program of swimming. After a training period, they swam 5 d/wk, 120 min/session for 4 wk. For the last 8 wk, their workload was increased. The exercising rats were provided with feed ad libitum. Over 18 wk, the rats lost 182 g, a 25% reduction in body weight, due to both an increased caloric output and a decreased appetite. The composition of the weight lost was 78% fat, 5% protein, 1% mineral, and 16% water. The measurements were based on comparisons to sedentary pair weighed animals. At the end of the study, percent body fat of the exercised rats was less than one-third that of the free-eating sedentary rats, less than one-half of initial levels, and slightly more than three-fourths that of the sedentary pair weighed rats. This study shows that exercise promotes the loss of fat while conserving lean mass as opposed to weight loss by caloric restriction.

B. Effects of Exercise on Adipocyte Size and Number

Weight loss by exercise results in a reduction in fat cell size. Ad libitum fed 16-wk-old male rats ran on a treadmill 5 d/wk for 8 wk (19). Fat cell size was reduced approximately 17%, and the amount of lipid per cell decreased. No effect was observed on fat cell number. Fat pad weights were also less in exercised rats than control rats. Similar results on the reduction of fat cell size have been reported (11,20,21,23).

Another study (20) demonstrated a significant decrease in fat

cell size when adult male rats swam 2 h/d, 5 d/wk but not when they swam only 1 h/d, 5 d/wk. In contrast, others found no difference in fat cell size when comparing male rats exercised on a treadmill 2 h/d and those exercised 1 h/d (11).

C. Effects of Exercise on Hormone, Enzyme, and Substrate Levels

Size of fat cells has been associated with hormone-stimulated lipolytic potential, but it has also been associated with exercise training. With sudden demands for energy, fatty acid mobilization is stimulated through the release of norepinephrine and the activation of hormone-sensitive lipase in adipose tissue. Lipolytic activity in adipose tissue increased when fat cell size was reduced in both trained and sedentary male rats (21), but when trained and sedentary rats having the same size fat cells were compared, the trained rats showed greater lipolytic activity. Similarly, when male rats were trained at two different intensity levels but had the same size fat cells, the rats training with more intensity had greater lipolytic activity (11). This indicates that exercise influences the sensitivity of adipocytes to lipolytic hormones in a manner other than the reduction of fat cell size.

Changes in levels of substances in the blood and tissues have been studied. In one study (15), male rats were exercised on a treadmill for 12 wk, 1 h/d, 5 d/wk. Resting blood glucose and lactate, plasma and adipose tissue FFA, and skeletal muscle lactate were similar to those of ad libitum fed and pair weighed sedentary controls. However, the skeletal muscle and liver glycogen stores were significantly

greater in the rested, exercising compared to the sedentary rats. Exhaustive exercise caused a decrease in blood glucose and glycogen stores and an increase in FFA levels. These results indicate that exercise increases the deposition of muscle and liver glycogen stores and the mobilization of FFA. Exercise decreased plasma cholesterol levels but did not influence triglycerides, glucose, or insulin levels.

D. Food Intake During Exercise Programs

Exercise modifies the feed intake in rats. However, the modification depends on the intensity of exerise and the gender of the rat.

In male rats, light exercise of extended duration does not affect feed intake while more severe exercise of shorter duration depresses appetite. Stevenson et al. (22) reported that 4 h of swimming, 4 d/wk had no effect on appetite and feed intake of male rats. Oscai et al. (23) also found no change in feed intake when male rats swam 6 h/d, 6 d/wk until 162 h of swimming had been completed. A study by Katch et al. (24) found that male rats exercised at low intensity had a depressed feed intake, but not as much as rats exercised at a high intensity. In these studies, even though low intensity exercise either had no effect or slightly decreased feed intake, a negative calorie balance resulted because feed intake did not increase to compensate for calories expended during exercise.

In contrast to light exercise, moderate exercise of greater intensity depresses feed intake in male rats. Crews et al. (25) found

that a 12-wk program of vigorous treadmill running 2 h/d decreased the feed intake of male rats. Stevenson et al. (22) also reported that 1-2 h of strenuous swimming 4 d/wk for 4 wk reduced feed intake in male rats. Oscai and Holloszy (9) who studied overweight male rats that were exercised by swimming 2 h/d, 6 d/wk, with attached weights, and Ahrens et al. (26) who exercised male rats on a treadmill 30 min/d for 8 wk both found a decrease in appetite and feed intake.

On the other hand, female rats show a different response in feed intake to exercise than male rats. Exercising female rats consume more feed than sedentary controls. Oscai et al. (23) subjected female rats to a swimming program 6 h/d, 6 d/wk, for a cummulative total of 162 h. The exercising rats consumed significantly more feed than sedentary controls. Crews and Aldinger (26) found that female rats subjected to 6 h of daily swimming had a 26% increase in feed intake. Because female rats respond differently to exercise than males in regard to feed intake, the effectiveness of exercise in promoting weight loss in female rats may be different than in males.

When considering spontaneous bouts of exercise, male rats were found to eat less on exercise days but increase intake on rest days (22). Spontaneous voluntary running on an activity wheel had no effect on feed intake in male rats but increased intake in female rats.

These studies indicate that the effectiveness of exercise on weight loss in rats may depend on the intensity of the exercise as well as the gender of the rats being studied. A summary showing the effects of treadmill exercise on weight gain and feed intake is shown

in Table 1.

E. Detraining and Rebound Weight Gain

When exercise is discontinued, weight gain rapidly follows. In rats, feed intake increases when physical activity subsides. This is similar to the greater feed intake of male rats during rest days between spontaneous bouts of exercise (22). In addition, feed efficiency increases during detraining. These effects were shown by Applegate et al. (28) who studied groups of male Osborne-Mendel rats on control diets or high-fat diets. After a 6-wk exercise program of treadmill running begun at 15 wk of age, a 2-wk detraining period followed. By the end of the detraining period, the detrained rats and the sedentary controls no longer differed in body weight. The detrained rats gained about twice as much fat as the sedentary controls in the 2-wk detraining period, even though they still had less than controls. With detraining, the number of fat cells in the retroperitoneal fat pad increased in both the high-fat and control groups, but in the epididymal pad for the high-fat group only. Fat cell size increased with exercise discontinuation, but the cells were still smaller than in the sedentary controls. Plasma triglycerides, insulin, and glucose were unaffected by detraining, but cholesterol increased after having dropped during exercise so that the levels were comparable to the sedentary controls. Discontinuation of exercise caused an increase in lipogenesis and lipoprotein lipase activity in adipose tissue. Another study (19) on male rats showed a similar response in weight gain except that while fat cell size increased, the

TABLE 1

Comparison of studies involving treadmill exercise in rats

Investigators (Ref.)	Speed (m/min)	Duration	Degree Inclination	Study length (wks)	Frequency (d/wk)	%Change Feed Intake[1]	%Change Body wt[1]	Age (wks)	Sex	Strain
Askew & Hecker (11)	29.5	2 h	8	12	5	-12.0	-23.5	6.5	M	Carworth CFN
Askew & Hecker (11)	29.5	1 h	8	12	5	-19.0	-25.4	6.5	M	Carworth CFN
Mazzeo & Horvath (4)	27.0	1 h	15	12	5	+27.9	+4.3	24	F	Fischer 344
Mazzeo & Horvath (4)	20.4	1 h	15	12	5	+17.8	-0.9	60	F	Fischer 344
Mazzeo & Horvath (4)	17.4	1 h	15	12	5	0	-4.0	108	F	Fischer 344
Taylor et al. (15)	26.8	1 h	0	12	5	-17.1	-23.5	6-8	M	Wistar
Askew et al. (21)	29.5	2 h	8	13	5	-24.1	-27.2	5	M	Carworth CFN
Stevenson et al. (22)	6.0	3 h	0	4	4	-15.0	-8.5	9	M	Sprague-Dawley
Applegate et al. (28)	20.0	50 min	0	6	6	0	-2.6	15	M	Osborne-Mendel
Booth et al. (19)	26.8	1 h	0	8	5	-	-14.5	7	M	Wistar
Booth et al. (19)	26.8	1 h	0	8	5	-	-3.6	16	M	Wistar
Booth et al. (19)	26.8	1 h	0	16	5	-	-1.3	7	M	Wistar
Dohm et al. (29)	35.0	1 h	0	6	6	-9.1	-13.8	-	M	Holtzman
Deb & Martin (30)	26.8	1 h	2	11	5	-4.4	-1.5	8	M	Zucker, lean
Askew et al. (32)	29.5	2 h	8	12	5	-3.5	-22.7	7	M	Carworth, CFN
McGarr et al. (33)	31.0	2 h	8	12	5	-	-19.4	-	M	Wistar CFN
Askew et al. (31)	29.5	2 h	8	12	5	-	-25.2	5	M	Carworth, CFN
Owens et al. (34)	22.0	45 min	9	12	5	0	0	12	M	Long Evans

[1] Percent changes in feed intake and body weight are calculated by dividing the difference between the values for the control and exercised animals by the value for the control animals.

number of fat cells remained the same.

Dohm et al. (29) found that detraining caused a dramatic increase in the rate of weight gain, with feed efficiency of detrained male rats being greater than untrained controls. Body fat increased from 6.8 to 8.4% in just 2 wk of detraining. The increased lipid deposition was due to an increase in lipogenic enzyme activity, including liver and adipose tissue fatty acid synthetase, adipose glucose-6-phosphate dehydrogenase, adipose citrate cleavage enzyme, and adipose malic enzyme.

These studies indicate that the beneficial effects of exercise in rats can be reversed when exercise is discontinued. The rapid fat deposition during detraining might be attributed to the reduction in activity, increased insulin sensitivity, or changes in thermogenesis.

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CHAPTER 2:

REBOUND WEIGHT GAIN FOLLOWING EXERCISE OR MODERATE CALORIC RESTRICTION IN RATS

INTRODUCTION

Animals subjected to weight loss regimens regain weight rapidly when the regimens are discontinued. The rapid rate of weight gain, or rebound weight gain, has been observed following caloric restriction or exercise. When rats lose 15 to 29% of their weight by caloric restriction, they quickly regain the weight in only 7 to 12 d (1,2,3). Repeated cycles of weight loss and gain further reduce the time needed to regain the weight. Termination of exercise also results in weight gain. Exercise-induced weight loss is regained in a 2-wk detraining period (4,5).

The rapid rate of weight gain has been attributed to several factors. Feed restriction results in a 30% reduction in basal metabolic rate (BMR) (6) possibly due to the significant loss of lean tissue (7,8). Caloric restriction is accompanied by an increase in feed efficiency, and when terminated, animals eat more feed as a compensatory response (1,8). On the other hand, when exercise is used to induce weight loss, lean body mass is spared, and most of the weight loss is from fat tissue (7). Thus, the the decrease in BMR may not be as dramatic as in feed restriction. However, when exercise is terminated, animals gain weight because of hyperphagia, an increase in feed efficiency, and an increase in the activity of lipogenic enzymes (4,5).

The present study was designed to compare the effects of exercise and caloric restriction on rebound weight gain. Composition of weight loss and gain was assessed longitudinally using total body electrical conductivity, a new method which can be used on live animals.

MATERIALS AND METHODS

Animals and housing. Retired breeder female Wistar rats, 8-10 mos of age, weighing 345-428g each, were obtained from Harlan Sprague-Dawley (Indianapolis, IN). The rats were housed in individual stainless steel cages at a room temperature of 23-25°C, with a 12-h light/dark cycle.

Diet and exercise protocols. Rats were assigned to one of three groups (N=10): 1) control, 2) exercised, or 3) feed-restricted, so that the weights of the rats were approximately equal among groups. All rats were fed a modified AIN-76A purified diet (Appendix 1) which contained a slightly increased content of vitamin and mineral mix replacing the usual sucrose content. Feed intake and body weights were recorded daily throughout the trial.

Exercised rats ran on a zero-grade treadmill (Boston Gears, Quincy, MA) at 18 m/min for 75 min/d (3x25 min with 5 min rest between bouts). They ran 5 d/wk with rest days on Wednesday and Sunday. The rats ran for a total of 7 wk; during the first 2 wk they were gradually acclimated to the treadmill. Feed-restricted rats were not exercised but were given a daily allotment of feed so that they weighed as much as the paired exercised rats.

After 7 wks, the exercise and feed-restriction regimens were discontinued, and rats were observed during a 2-wk rebound period. Body weight and feed intake measurements were also recorded during this time. Feed efficiency was calculated as g/d and g/100g body weight and was averaged for each week.

Body fat measurements. Percent body fat was measured before,

during, and after the weight loss period and also after each wk of the rebound period. Body fat was assessed by total body electrical conductivity (TOBEC) using a Dickey John 100 Ground Meat Fat Tester (Em-Scan, Inc., Springfield, IL). The TOBEC measurement is based on the induction of uniform current in the body by placement within a characteristic low-frequency electromagnetic field. Conductivity is highly correlated with lean body mass (9). The rats were fasted 12 h before the test and anaesthetized with diethyl ether. The rats were then carefully positioned on a plank using rubber bands and tape (Figure 1) and inserted into the machine. Four readings were obtained and the average was used to calculate lean body mass (LBM) and by difference, body fat. Readings for a single rat included Calc 1 (a beginning baseline reading), four Raw readings (with rat inside) alternating with four Empty readings (without rat), and Calc 2 (an ending baseline reading).

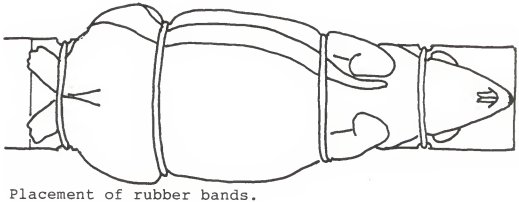
$$E = \frac{\text{Avg Empty - Avg Raw}}{\text{Avg Calc 1 \& Calc 2}}$$

$$\text{LBM (g)} = 30.84 + 0.396\ E - (4.85 \times 10^{-5} \times E^2)$$

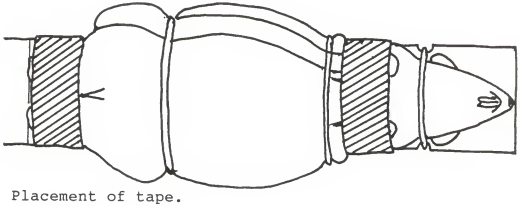
Terminal procedures. At the end of the 2-wk rebound period, rats were fasted 12 h and killed by decapitation. The heart, liver, kidneys, and gastrocnemius and vastus muscles of each rat were removed, weighed, wrapped in aluminum foil, and frozen at -70°C for further analysis. Omental, retroperitoneal, and gonadal fat pads were also removed and weighed.

Tissue analysis. Liver, kidney, heart, gastrocnemius, and

Plank with lines used for positioning the rat and notches used in the placement of rubber bands.



Placement of rubber bands.



Placement of tape.

Figure 1. Positioning of the rat for measurement of total body electrical conductivity (TOBEC).

vastus were analyzed for lipid content using the Folch gravimetric method (10) as modified by Chen et al. (11). Fifteen ml of a methylene chloride:methanol solution (2:1, v/v) was added to 1g of tissue in a test tube and homogenized using a high-speed Polytron homogenizer (Brinkman Instruments, Westburg, NY). The homogenate was filtered and washed with an additional 5 ml of the methylene chloride:methanol solution.

The filtrate was collected into centrifuge tubes, and 4 ml of 0.73% NaCl solution was added. The sample was mixed for 5 min on a wrist action shaker and was then centrifuged for 5 min. The top aqueous layer was aspirated off and 8 ml of the bottom (organic) layer was pipetted into an oven-dried pre-weighed aluminum dish and placed under a hood to evaporate the liquid. The plate was then dried in an oven at 101°C for 30 min, cooled in a dessicator, and weighed. Tissue fat was calculated as a percent of tissue weight.

Statistical analysis. Data were analyzed using Analysis of Variance procedures to differentiate among any treatment means (12). Each rat served as its own control or baseline. The computer program used for statistical analysis is shown in Appendix 11.

RESULTS

Weight gain and feed intake. Weight gain, feed intake, and feed efficiency for the 7-wk treatment period and 2-wk rebound period are shown in Table 2. During the treatment period, the control animals gained significantly more weight than rats in the exercise and feed-restricted groups. The exercised and feed-restricted rats gained a comparable amount of weight, as expected, because the feed-restricted rats were fed only enough so that they weighed as much as the exercised rats. During the rebound period, the exercised rats gained significantly more weight than the control and feed-restricted rats.

The control rats ate more feed (g/d and g/100g body weight) during the treatment period than exercised and feed-restricted rats, but feed intake was comparable between the two latter groups. During the rebound period, the exercised rats ate more feed than the control and feed-restricted rats when feed was expressed relative to body weight. Rats in the exercise and feed-restricted groups had lower feed efficiencies than the control group during the treatment period, but during the rebound period, feed efficiency of the exercise group was higher than both the control and feed-restricted groups.

Body composition. Average values for body weight and body composition are shown in Table 3. Control rats weighed more than exercised and feed-restricted rats during both the treatment and rebound periods. Exercised and feed-restricted rats weighed the same during the treatment period, but exercised rats gained more weight than feed-restricted rats during the rebound period.

Control rats had more body fat, in grams, than exercised and

TABLE 2

Weight gain and feed intake during

treatment and rebound periods in mature female rats[1]

Measurement	Treatment period (7-wk)	Rebound period (2-wk)
Weight gain, g		
Control	48±5[b]	-5±2[a]
Exercise	2±7[a]	13±4[b]
Feed-restricted	8±3[a]	0±5[a]
Feed intake, g/d		
Control	17.4±0.4[b]	14.5±0.5[a]
Exercise	14.6±0.5[a]	16.6±0.4[b]
Feed-restricted	13.7±0.3[a]	14.6±1.0[ab]
Feed intake, g/100g body wt		
Control	4.1±0.1[b]	3.3±0.1[a]
Exercise	3.7±0.1[a]	4.1±0.1[b]
Feed-restricted	3.4±0.1[a]	3.6±0.2[a]
Feed efficiency g wt gain/g feed intake		
Control	0.06±0.01[b]	-0.12±0.06[a]
Exercise	0.00±0.01[a]	0.22±0.06[b]
Feed-restricted	0.01±0.01[a]	-0.04±0.10[a]

[1] Values are means ± SEM for 9 or 10 rats. Means within each column not sharing common letters are significantly ($P<0.05$) different using least significant differences tests following analysis of variance procedures. Individual data are shown in appendix 3.

TABLE 3

Rebound weight gain following exercise or feed restriction periods in mature female rats[1]

	After 7-wk treatments	Rebound period, wks		
	0	1	2	% Change
Body wt, g [2]				
Control	438±7[b]	437±8[b]	433±7[b]	-1±1[a]
Exercise	391±10[a]	396±9[a]	404±8[a]	+4±1[b]
Feed-restricted	401±8[a]	400±9[a]	402±10[a]	0±1[a]
Body fat, g				
Control	190±5[b]	190±6[b]	190±5[b]	0±1
Exercise	168±3[a]	169±4[a]	174±4[a]	+4±2
Feed-restricted	173±4[a]	171±4[a]	175±5[a]	+1±2
Body fat, %				
Control	43±1	43±1	44±1	2±1
Exercise	43±1	43±1	43±1	0±1
Feed-restricted	43±1	43±1	44±1	1±1
Lean body mass, g				
Control	248±5[b]	248±6[b]	243±5	-2±1[a]
Exercise	223±8[a]	227±7[a]	230±6	+3±2[b]
Feed-restricted	229±8[ab]	229±7[ab]	227±8	-1±2[a]
Lean body mass, %				
Control	57±1	57±1	56±1	-1±1
Exercise	57±1	57±1	57±1	0±1
Feed-restricted	57±1	57±1	56±1	-1±1

[1] Values are means ± SEM for 9 or 10 rats. Means within each column not sharing common letters are significantly ($P<0.05$) different using least significant differences tests following analysis of variance procedures. Individual data are shown in appendices 4-6.

[2] Animals were matched for weight prior to 7-wk treatment periods; mean weight was 391±5 g.

feed-restricted rats during both the treatment and rebound periods. However, rats in all three groups gained a similar amount of fat during the rebound period. When body fat was expressed as percent of body weight, no significant differences were observed at any time among rats in any of the groups.

Control rats had more lean body mass than exercised rats during the treatment period, but exercised rats gained more lean body mass during the rebound period than either control or feed-restricted rats. When lean body mass was expressed as a percent of body weight, there were no differences among rats throughout the study.

Fat pads. Table 4 shows the fat pad weights after the rebound period. Control rats had more retroperitoneal fat than the feed-restricted rats but not more than the exercised rats. Control rats had more omental fat than rats in the exercise and feed-restricted groups. Gonadal fat (g) was not significantly different among the groups, but gonadal fat (g/100g body weight) was higher in exercised rats than feed-restricted rats.

Tissue weights and fat contents. Tissue weights and fat contents are shown in Table 5. Control rats had heavier livers than feed-restricted but not exercised rats. Feed-restricted rats had lighter livers, when expressed on a relative weight than exercised and control rats. Feed-restricted rats also had less fat in their livers than exercised and control rats.

Kidney weights were similar among rats in all three groups. However, kidney fat was significantly higher in the control group than the feed-restricted group. Heart weight and fat contents were

TABLE 4

Fat pad weights after 2-wk rebound period[1]

Fat Pad	Tissue wt g	Tissue wt g/100 g body wt
Retroperitoneal[2]		
Control	20.0 ± 2.2^{b}	4.5 ± 0.4^{b}
Exercise	15.5 ± 2.0^{ab}	3.8 ± 0.4^{ab}
Feed-restricted	13.8 ± 1.3^{a}	3.4 ± 0.3^{a}
Omental		
Control	18.7 ± 2.1^{b}	4.3 ± 0.4^{b}
Exercise	13.7 ± 1.4^{a}	3.3 ± 0.3^{a}
Feed-restricted	13.3 ± 0.9^{a}	3.3 ± 0.2^{a}
Gonadal[2]		
Control	19.3 ± 1.2	4.5 ± 0.3^{ab}
Exercise	16.6 ± 1.5	4.1 ± 0.3^{a}
Feed-restricted	20.3 ± 1.7	5.0 ± 0.3^{b}

[1] Values are means ± SEM for 9 or 10 rats. Means within each column sharing common letters are significantly ($P<0.05$) different using least significant differences test following analysis of variance procedures. Individual data are shown in appendix 7.

[2] Tissues from both sides were pooled for measurements.

TABLE 5

Tissue weights and fat contents after

2-wk rebound period[1]

Tissue	Tissue wt g	Tissue wt g/100g	Tissue fat %
Liver			
Control	11.3±0.4[b]	2.6±0.1[b]	14.2±1.5[b]
Exercise	10.5±0.4[ab]	2.6±0.1[b]	15.6±1.6[b]
Feed-restricted	9.4±0.5[a]	2.3±0.1[a]	10.1±0.7[a]
Kidneys[2]			
Control	2.3±0.1	0.5±0.0	5.0±0.2[b]
Exercise	2.3±0.1	0.6±0.0	4.8±0.1[ab]
Feed-restricted	2.2±0.1	0.6±0.0	4.4±0.1[a]
Heart			
Control	1.2±0.1	0.3±0.0	3.7±0.2
Exercise	1.1±0.0	0.3±0.0	4.0±0.2
Feed-restricted	1.0±0.0	0.3±0.0	3.4±0.2
Vastus			
Control	1.42±0.05	0.33±0.01[a]	2.5±0.1[b]
Exercise	1.43±0.02	0.35±0.01[ab]	1.9±0.1[a]
Feed-restricted	1.46±0.04	0.36±0.01[b]	2.2±0.1[ab]
Gastrocnemius			
Control	1.26±0.03	0.29±0.01[a]	2.7±0.1
Exercise	1.27±0.03	0.31±0.01[b]	2.4±0.1
Feed-restricted	1.25±0.04	0.31±0.01[ab]	2.3±0.1

[1] Values are means ± SEM for 9 or 10 rats. Means within each column not sharing common letters are significantly ($P<0.05$) different using least significant differences tests following analysis of variance procedures. Individual data are shown in appendices 8 and 9.

[2] Tissues from both sides were pooled for measurements.

not significantly different among the groups.

Vastus weight (g) was not significantly different among the groups. However, feed-restricted rats had heavier vastus muscles, when expressed relative to body weight, than control rats. Exercised rats had less fat in their vastus muscles than control rats but not feed restricted rats.

No significant differences in gastrocnemius weight (g) or fat content were found among the groups. When tissue weight was expressed relative to body weight, the exercised rats had heavier gastrocnemius muscles than control rats.

DISCUSSION

Our study shows that the termination of exercise is followed by a greater rebound weight gain than that observed after feed restriction. Our exercised rats gained more weight than control or feed-restricted rats, but they still did not catch up to the control rats during the 2-week period.

Dohm et al. (5) found that detraining results in a more rapid weight gain in previously exercised rats compared to controls, but at the end of a 2-wk detraining period, the body weights of the detrained rats were still less than the controls. Other studies have shown that exercised (4) or calorie-restricted rats (1,2,8,13,14) rapidly gain weight during a rebound period so that their body weights were at control levels in approximately 2 weeks.

The rats in our study may not have re-gained weight as rapidly because they were older, retired breeders, in contrast to the young male or female rats commonly used in weight loss studies. Perhaps the ability to re-gain weight is compromised in older rats, especially if it is preceeded by stressful treatments such as caloric restriction. When our feed-restricted rats were allowed free access to feed they did not eat more. Most other studies show that feed-restricted rats become hyperphagic and more feed efficient upon refeeding (1,2,8,14).

When our rats were exercised they voluntarily reduced their feed intake to about 84% of that consumed by the control animals. Other studies, in contrast, show that exercise increases feed intake of female rats (15,16). Male rats increase or do not change their feed

intake during exercise programs, depending on the intensity and duration of exercise (15,21). The difference between the present study and others may be in the age of our rats or their ability to adapt to an exercise program.

When exercise was discontinued our rats gained weight, and they gained more weight than feed-restricted or control rats during the 2-wk rebound period. This effect was likely due to their greater feed intake and feed efficiency, which has been observed in other studies during detraining (4,5,18). We do not know why the exercised rats gained more weight than feed-restricted rats. Both exercise and feed restriction, if discontinued, result in an increase in fat synthesis (4,5,8,22), although feed restriction would be expected to slow the replacement of lean tissue (8). Possibly, exercise kept our animals healthier than feed restriction so they could respond better. Most studies show that exercise reduces percent body fat and increases percent lean body mass (4,7,23). Another study found that compared to exercise, caloric restriction results in less loss of fat and a higher loss of lean tissue during weight reduction so that exercised rats have a lower percent body fat than pair-weighed calorie-restricted rats (7).

Our study also shows, importantly, that exercised rats gained more lean body mass during the 2-wk recovery period than control or feed-restricted rats. They also gained more fat but differences were not statistically significant. When body fat and lean body mass were expressed as a percent of body weight they did not vary throughout the study, possible because body composition in older animals is less

plastic and more resistant to change.

One of the objectives of our study was to evaluate the use of TOBEC in assessing longitudinal changes in body composition. We took TOBEC measurements in all animals at five different times throughout the study and consequently were able to observe repeatability in each rat and expected differences among treatment groups. The use of this instrument for live animals have been documented in only two published studies (24,25).

We found that TOBEC measurements in live animals were subject to several sources of error which must be minimized to attain an acceptable degree of precision and accuracy. In our hands, the TOBEC machine had a high degree of precision when obtaining readings from nonliving saline:oil emulsions of different sizes, but this was less so for live anaesthetized rats. The main sources of error in the rats seem to be the correct and consistent positioning of the animal in the machine and making sure that the physiological state of the animal (feeding, hydration, temperature) is reproducible at each measurement. These are probably not insurmountable difficulties, but methods used to minimize error must be documented in future studies before TOBEC can be used with confidence.

There is actually very little information specifying how to reduce sources of error. Walsberg mentioned that positioning of the animal was important but did not specify how this was to be accomplished (25). This study also showed that three days of dehydration affected TOBEC readings in quail, and that temperature reduction accomplished by the application of temperature-controlled plastic

bags affected readings in rats. He did not specify whether animals should be fasted before TOBEC measurements or whether water may be restricted for shorter periods of time to obtain consistent levels of gut filling and hydration.

We found, by trial and error, that animals should be fasted 12 hours before measurements. If they were not, their lean body mass measurements would be falsely high, probably because the electrolyte content of the diet was detected as lean body mass. We also developed a system to position the animals as consistently as possible in the TOBEC, which was at times difficult with 400+ g rats. Using these procedures we found that the percent fat would vary within 3% for 81% of our rats from one week to the next during the rebound period. However 1.5% of our rats exceeded the 5% variation in fat, which is not likely to be a true physiological response.

Future attention might be directed towards further refinement of animal positioning in the TOBEC machine (especially with head, paws, and tail). Other sources of error to be investigated include whether or not water should be restricted during the 12-h fast and whether rats should remain in anesthesia for a period of time before measurement (since excitation increases body temperature). These factors should be tested in mature rats where growth is slow and repeatability of measurements can be observed over time to establish an acceptable level of precision.

After repeatibility trials are completed, accuracy of TOBEC measurements should be established. This can be accomplished by a comparison of TOBEC readings to direct chemical analysis for fat.

The TOBEC values in our study are questionable because the average female rat had 43% body fat, much higher than values cited in other studies. Mazzeo and Horvath (23) have found that body fat of sedentary female rats 6 and 15 months is 19.5 and 24.6%, respectively. Exercise reduced body fat in those rats by 31 and 29%, respectively. In our study, exercise did not reduce body fat of the rats, according to our TOBEC measurements.

Comparison of TOBEC measurements to direct chemical analysis of fat should be accomplished in rats of different sizes, gender, and experimental treatment. These variables should be tested because they might result in a different distribution of fat within the animal, which may affect TOBEC readings. Direct chemical analysis is an important approach because previous studies involving TOBEC simply estimated body fat from specific gravity or by the difference between total body weight and lean body mass. Once the relationship between TOBEC reading and body fat is determined, a mathematical equation can be developed that will estimate body fat based on TOBEC readings, body weight, and saline-filled standards used to calibrate the machine.

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APPENDIX

APPENDIX 1

Diet with adjusted vitamins and minerals[1]

Ingredient	Amount
	g/kg
Casein, high protein	200.0
DL-Methionine	3.0
Sucrose	493.49
Corn starch	150.0
Corn oil	50.0
Fiber (cellulose)	50.0
Mineral mix[2]	40.0
Vitamin mix[3]	11.5
Choline bitartrate	2.0
Ethoxyquin (antioxidant)	0.01

[1]This diet (TD 88317) is a modification of the AIN-76A Purified Diet (Rats/Mice) supplied by Teklad Test Diets, Madison, WI. Mineral mix and vitamin mix are each increased 15% over that in AIN-76A.

[2]Supplied as AIN-76 mineral mix in g/kg of mix: calcium phosphate, 500.0; sodium chloride, 74.0; potassium citrate monohydrate, 220.0; potassium sulfate, 52.0; magnesium oxide, 24.0; manganous carbonate, 3.5; ferric citrate, 6.0; zinc carbonate, 1.6; cupric carbonate, 0.3; potassium iodate, 0.01; sodium selenite, 0.01; chromium potassium sulfate, 0.55; sucrose finely powdered, 118.03.

[3]Supplied as AIN-76A vitamin mix in g/kg of mix: thiamin HCl, 0.6; riboflavin, 0.6; pyridoxine HCl, 0.7; niacin, 3.0; calcium pantothenate, 1.6: folic acid, 0.2; biotin, 0.02; vitamin B_{12} (0.1% trituration in mannitol), 1.0; dry vitamin A palmitate (500,000 U/g), 0.8; Vitamin D_3 tituration (400,000 U/g), 0.25; dry vitamin E acetate, 10.0; menadione sodium bisulfite complex, 0.15; sucrose finely powdered, 981.08.

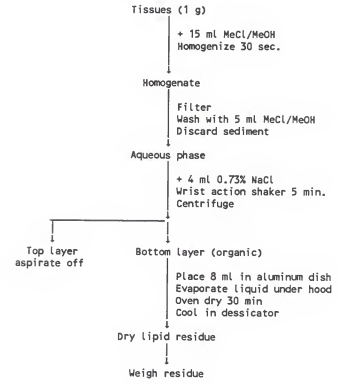
APPENDIX 2

Reagents for tissue fat analysis

1. Methylene chloride/methanol solution (2:1, v/v)

2. Sodium chloride solution (0.73% NaCl)
   7.3 g of sodium chloride is dissolved in deionized water and brought up to 1 L.

TISSUE FAT ANALYSIS PRODEDURE



APPENDIX 3

Weight gain and feed intake of mature female rats

	Treatment Period (7-wk)			Rebound period (2-wk)		
Measurement	Control	Exer	Feed-restr	Control	Exer	Feed-restr
Weight gain, g	24	-	12	0	-	25
	42	28	16	-12	11	-1
	79	-10	12	-1	9	-14
	43	30	15	-9	-3	12
	32	-28	-1	-11	16	-3
	54	6	-8	2	7	-8
	50	11	4	-18	9	17
	55	-22	-5	-7	32	3
	60	8	28	3	27	-21
	45	-7	4	6	12	-8
	48±5	2±7	8±3	-5±2	13±4	0±5
Feed intake g/d	16.1		13.3	14.1		19.0
	17.3	17.7	15.7	14.0	18.8	14.2
	18.4	13.9	12.6	15.6	16.0	12.6
	17.0	16.1	15.6	13.8	15.6	16.5
	16.4	13.6	13.8	14.4	16.8	15.3
	18.3	15.8	13.1	16.6	17.4	15.2
	16.0	12.8	13.3	11.1	15.2	19.5
	20.0	13.5	12.8	15.4	17.4	11.8
	17.0	14.1	13.5	14.7	17.5	9.4
	17.4	13.8	13.2	15.8	15.2	12.5
	17.4±0.4	14.6±0.5	13.7±0.3	14.5±0.5	16.6±0.4	14.6±1.0
Feed intake g/100g body wt	3.7		3.1	3.1		4.2
	3.9	4.1	3.7	3.0	4.1	3.2
	3.9	3.4	3.1	3.1	3.9	3.0
	3.9	3.8	3.8	3.0	3.6	3.8
	3.9	3.5	3.6	3.4	4.4	3.9
	4.4	4.1	3.5	3.7	4.0	3.9
	3.9	3.3	3.3	2.6	3.8	4.7
	4.8	3.5	3.3	3.6	4.6	3.1
	4.2	3.7	3.5	3.4	4.4	2.4
	4.3	3.7	3.5	3.8	4.1	3.4
	4.1±0.1	3.7±0.1	3.4±0.1	3.3±0.1	4.1±0.1	3.6±0.2
Feed efficiency g wt gain/ g feed intake	0.03		0.02	0.00		0.42
	0.05	0.03	0.02	-0.28	0.19	-0.02
	0.09	-0.01	0.02	-0.02	0.17	-0.33
	0.05	0.04	0.02	-0.21	-0.06	0.23
	0.04	-0.04	0.00	-0.23	0.26	-0.05
	0.06	0.01	-0.01	0.04	0.12	-0.15
	0.06	0.02	0.01	-0.49	0.17	0.26
	0.06	-0.03	-0.01	-0.14	0.49	0.07
	0.07	0.01	0.04	0.06	0.44	-0.62
	0.05	-0.01	0.01	0.11	0.21	-0.17
	0.06 ±0.01	0.00 ±0.01	0.01 ±0.01	-0.12 ±0.06	0.22 ±0.06	-0.04 ±0.10

APPENDIX 4

Body weight and fat of mature female rats during rebound weight gain

Measurement	Control	Exer	Feed-restr	Control	Exer	Feed-restr
Body wt, g	After 7-wk treatments			After rebound wk 1		
	443		435	450		440
	456	444	431	459	450	440
	490	399	419	495	411	419
	445	429	423	456	431	434
	421	361	391	423	371	393
	431	399	376	440	408	388
	426	393	401	429	394	410
	432	348	371	430	367	379
	428	379	396	435	395	392
	409	367	371	420	370	369
	438±7	391±10	401±8	437±8	396±9	400±9
	After rebound wk 2			% change		
	454		453	0.0		5.7
	460	457	439	-2.6	2.5	-0.2
	497	419	419	-0.2	2.3	-3.3
	449	430	438	-2.0	-0.7	2.8
	421	384	389	-2.6	4.4	-0.8
	446	467	385	0.5	1.8	-2.1
	415	402	422	-4.2	2.3	4.2
	428	383	378	-1.6	9.2	0.8
	433	406	380	0.7	7.1	-5.3
	422	372	365	1.5	3.3	-2.2
	433±7	404±8	402±10	-1±1	+6±1	0±1
Body fat, g	After 7-wk treatments			After rebound wk 1		
	188		171	207		179
	181	186	172	183	181	183
	228	180	184	225	184	178
	200	169	173	198	167	183
	173	160	156	179	140	151
	174	166	149	171	175	149
	194	166	173	192	178	169
	179	165	186	162	166	169
	191	161	191	192	165	184
	189	151	171	189	164	160
	190±5	168±3	173±4	190±6	169±4	171±4
	After rebound wk 2			% Change		
	188		186	0.0		8.7
	177	190	184	-2.7	1.7	7.3
	229	187	180	0.3	4.0	-2.1
	196	174	191	-2.0	3.1	10.6
	183	151	149	5.5	-5.6	-4.3
	181	176	144	3.6	6.5	-2.6
	188	169	185	-3.1	1.7	7.3
	180	181	172	0.9	9.7	-7.3
	190	178	183	-0.2	6.1	-4.4
	193	164	172	2.3	8.2	0.7
	190±5	174±4	175±5	0.5 ±0.9	3.9 ±1.5	1.4 ±2.1

APPENDIX 5

Percent body fat of mature female rats during rebound weight gain

Measurement	Control	Exer	Feed-restr	Control	Exer	Feed-restr
Body fat, %	After 7-wk treatments			After rebound wk 1		
	43		39	46		41
	40	42	40	40	41	42
	47	45	44	46	45	43
	45	39	41	44	40	43
	41	44	40	43	38	40
	40	41	40	39	43	39
	45	42	43	46	46	40
	41	47	50	38	45	45
	45	44	48	45	42	49
	46	41	46	46	45	45
	43±1	43±1	43±1	43±1	43±1	43±1
	After rebound wk 2			% Change		
	42		40	0.0		2.8
	40	42	43	0.0	-0.8	7.6
	47	46	44	0.5	1.8	1.3
	45	41	44	0.0	3.8	7.6
	45	40	39	8.4	-9.6	-3.6
	42	43	39	3.1	4.6	-0.5
	46	42	44	1.1	-0.5	2.9
	42	48	46	2.6	0.5	-8.1
	44	44	49	-0.9	-1.0	1.0
	47	43	47	0.8	4.7	2.9
	44±1	43±1	44±1	1.6±0.8	0.4±1.5	1.4±1.5

APPENDIX 6

Lean body mass of mature female rats during rebound weight gain

Measurement	Control	Exer	Feed-restr	Control	Exer	Feed-restr
Lean body mass, g	After 7-wk treatments			After rebound wk 1		
	258		264	263		257
	219	264	259	220	257	250
	260	259	235	250	250	238
	201	235	250	231	238	244
	233	250	235	233	244	227
	227	235	227	207	227	235
	183	227	228	203	235	248
	212	228	185	231	248	204
	216	185	205	203	203	192
	220	205	200	225	192	197
	248±5	223±8	229±8	248±6	227±7	229±7
	After rebound wk 2			% Change		
	265		274	3.1		3.8
	221	274	246	0.8	3.8	-5.3
	252	246	225	-3.2	-5.3	-4.3
	226	225	244	12.4	-4.3	-2.5
	230	244	239	-1.6	-2.5	1.6
	233	239	223	2.7	1.6	-1.8
	199	223	233	8.7	-1.8	1.9
	228	233	202	8.0	1.9	9.0
	215	202	192	-0.2	9.0	-6.2
	222	192	191	0.8	-6.2	-4.6
	243±5	230±6	227±8	-2±1	+3±2	-1±2
Lean body mass, %	After 7-wk treatments			After rebound wk 1		
	58		61	54		59
	60	58	60	60	59	58
	53	55	56	54	55	57
	55	61	59	56	60	57
	59	56	60	57	62	60
	60	59	60	61	57	61
	55	58	57	54	54	60
	59	53	50	62	55	55
	55	56	52	55	58	51
	54	59	54	54	55	55
	57±1	57±1	57±1	57±1	57±1	57±1
	After rebound wk 2			% Change		
	58		60	0.0		-1.8
	60	58	57	0.0	0.6	-5.0
	53	54	56	-0.4	-1.4	-1.0
	55	59	56	0.0	-2.5	-5.2
	55	60	61	-5.8	7.6	2.4
	58	57	61	-2.1	-3.3	0.3
	54	58	56	-1.0	0.4	-2.2
	58	52	54	-1.8	-0.5	8.1
	56	56	51	0.7	0.8	-0.9
	53	57	53	-0.7	-3.3	-2.5
	56±1	57±1	56±1	-1.1±0.6	-0.2±1.1	-0.8±1.2

APPENDIX 7

Fat pad weights of mature female rats after 2-wk rebound period

Measurement	Control	Exer	Feed-restr	Control	Exer	Feed-restr
	g			g/100 g body wt		
Retroperitoneal fat pad wt	23.5		20.9	5.3		4.5
	27.7	27.7	11.3	6.2	6.1	2.6
	34.3	13.5	14.6	7.0	3.3	3.6
	20.9	14.5	16.1	4.8	3.4	3.7
	16.1	12.5	8.2	3.9	3.3	2.1
	16.3	11.3	8.1	3.8	2.8	2.2
	14.8	23.4	18.1	3.6	5.8	4.3
	13.8	12.5	12.7	3.2	3.3	3.4
	18.7	13.7	16.2	4.3	3.4	4.3
	13.4	10.2	11.8	3.2	2.7	3.3
	20.0	15.5	13.8	4.5	3.8	3.4
	±2.2	±2.0	±1.3	±0.4	±0.4	±0.3
Omental fat pad wt	21.1		18.9	4.8		4.1
	22.0	20.9	14.2	5.0	4.5	3.3
	35.2	13.0	10.2	7.2	3.2	2.5
	19.1	18.9	14.9	4.4	4.4	3.4
	14.9	8.6	10.6	3.6	2.3	2.7
	18.4	11.5	9.5	4.2	2.8	2.6
	16.9	16.8	12.6	4.1	4.2	3.0
	10.8	10.6	13.9	2.5	2.8	3.7
	15.2	12.3	13.9	3.5	3.0	3.7
	13.8	10.3	14.2	3.3	2.7	3.9
	18.7	13.7	13.3	4.3	3.3	3.3
	±2.1	±1.4	±0.9	±0.4	±0.3	±0.2
Gonadal fat pad wt	17.1		28.3	3.9		6.2
	20.1	22.2	18.5	4.5	4.9	4.3
	25.4	20.6	19.7	5.2	5.0	4.9
	17.0	22.1	26.9	3.9	5.2	6.2
	26.9	9.8	20.2	6.6	2.6	5.2
	16.4	15.7	11.7	3.8	3.9	3.2
	18.2	14.3	25.8	4.5	3.6	6.2
	19.0	13.0	17.4	4.5	3.4	4.7
	14.5	18.4	19.4	3.4	4.5	5.2
	18.6	13.2	15.2	4.5	3.5	4.2
	19.3	16.6	20.3	4.5	4.1	5.0
	±1.2	±1.5	±1.7	±0.3	±0.3	±0.3

APPENDIX 8

Organ weights in mature female rats after 2-wk rebound period

Measurement	Control	Exer	Feed-restr	Control	Exer	Feed-restr
	----------g----------			-----g/100 g body wt------		
Liver wt	10.8		12.2	2.4		2.7
	11.3	12.5	9.8	2.5	2.7	2.3
	13.8	10.6	9.6	2.8	2.6	2.4
	10.9	12.5	11.7	2.5	2.9	2.7
	11.7	10.1	8.7	2.9	2.7	2.2
	12.3	9.4	8.6	2.8	2.3	2.3
	11.8	9.7	7.4	2.9	2.4	1.8
	8.6	9.2	8.3	2.0	2.4	2.2
	11.6	10.1	9.2	2.7	2.5	2.5
	10.5	10.6	8.7	2.5	2.8	2.4
	11.3	10.5	9.4	2.6	2.6	2.3
	±0.4	±0.4	±0.5	±0.1	±0.1	±0.1
Kidney wt	2.5		2.6	0.6		0.6
	2.4	2.4	2.2	0.5	0.5	0.5
	2.1	2.4	2.3	0.4	0.6	0.6
	2.1	2.5	2.4	0.5	0.6	0.6
	2.4	2.6	2.2	0.6	0.7	0.6
	2.6	2.2	2.1	0.6	0.5	0.6
	2.1	2.0	2.1	0.5	0.5	0.5
	2.2	2.3	1.9	0.5	0.6	0.5
	2.4	2.1	2.2	0.6	0.5	0.6
	2.6	2.3	2.4	0.6	0.6	0.7
	2.3±0.1	2.3±0.1	2.2±0.1	0.5±0.0	0.6±0.0	0.6±0.0
Heart wt	2.0		1.2	0.5		0.3
	1.1	1.1	1.1	0.2	0.2	0.3
	1.2	1.3	0.9	0.2	0.3	0.2
	1.0	1.2	1.1	0.2	0.3	0.3
	1.1	1.3	1.0	0.3	0.3	0.3
	1.2	1.0	1.1	0.3	0.2	0.3
	1.0	1.0	1.0	0.2	0.2	0.2
	1.0	1.0	0.9	0.2	0.3	0.2
	1.1	1.0	1.0	0.3	0.2	0.3
	1.2	1.0	1.0	0.3	0.3	0.3
	1.2±0.1	1.1±0.0	1.0±0.0	0.3±0.0	0.3±0.0	0.3±0.0

APPENDIX 9

Hindlimb muscle weights in mature female rats after 2-wk rebound period

Measurement	Control	Exer	Feed-restr	Control	Exer	Feed-restr
	----------g----------			--g/100 g body wt--		
Vastus wt	1.37		1.62	0.3		0.4
	1.25	1.40	1.55	0.3	0.3	0.4
	1.27	1.41	1.56	0.3	0.3	0.4
	1.64	1.55	1.43	0.4	0.4	0.3
	1.43	1.43	1.56	0.3	0.4	0.4
	1.37	1.40	1.40	0.3	0.3	0.4
	1.38	1.31	1.48	0.3	0.3	0.4
	1.79	1.42	1.36	0.4	0.4	0.4
	1.29	1.53	1.29	0.3	0.4	0.3
	1.37	1.39	1.31	0.3	0.4	0.4
	1.42	1.43	1.46	0.33	0.35	0.36
	±0.05	±0.02	±0.04	±0.01	±0.01	±0.01
Gastrocnemius wt	1.19		1.41	0.3		0.3
	1.28	1.29	1.32	0.3	0.3	0.3
	1.17	1.24	1.31	0.2	0.3	0.3
	1.40	1.27	1.32	0.3	0.3	0.3
	1.32	1.23	1.22	0.3	0.3	0.3
	1.20	1.25	1.34	0.3	0.3	0.4
	1.25	1.28	1.33	0.3	0.3	0.3
	1.38	1.19	1.16	0.3	0.3	0.3
	1.14	1.48	1.11	0.3	0.4	0.3
	1.24	1.20	1.01	0.3	0.3	0.3
	1.26	1.27	1.25	0.29	0.31	0.31
	±0.03	±0.03	±0.04	±0.01	±0.01	±0.01

APPENDIX 10

Tissue fat in mature female rats after 2-wk rebound period

Measurement	Control	Exer	Feed-restr	Control	Exer	Feed-restr
Tissue fat		Liver			Kidney	
	13.8		12.2	4.2		4.4
	17.1	23.0	8.4	5.9	5.1	5.0
	22.3	12.2	11.0	5.9	4.4	4.1
	11.4	21.2	12.3	4.8	4.2	4.8
		19.1	7.1		4.2	4.3
	10.8	9.9	7.2		5.1	3.8
	14.0	15.5	10.5	4.8	5.3	4.2
	8.3	9.6	12.2	5.0	4.7	4.6
	16.8	12.8	11.3	4.8	5.1	4.1
	12.2	17.4	8.4	4.2	4.8	4.6
	14.2 ±1.5	15.6 ±1.6	10.1 ±0.7	5.0 ±0.2	4.8 ±0.1	4.4 ±0.1
		Heart			Vastus	
	4.0		4.4	2.8		2.1
	4.2	3.5	3.5	2.7	2.0	2.0
	3.8	3.3	3.3	2.4	1.7	2.1
	4.9	3.8	3.9	3.2	1.7	2.2
		3.8	2.7		2.6	2.1
	2.9	3.6	2.7	2.7	2.2	2.2
	3.5	4.9	2.6	2.4	1.7	2.2
		4.2	4.8	2.1	1.7	2.6
	3.3	4.2	3.2	2.1	1.5	2.2
	3.0	4.4	2.9	2.0	2.4	2.2
	3.7±0.2	4.0±0.2	3.4±0.2	2.5±0.1	1.9±0.1	2.2±0.1
		Gastrocnemius				
	2.4		2.3			
	3.1	2.0	2.0			
	3.2	2.6	2.3			
	2.9	2.6	2.4			
		2.2	1.7			
	2.6		2.3			
	2.3	2.7	2.4			
	1.9	2.2	2.7			
	3.1	2.1	2.1			
	2.6	2.6	3.1			
	2.7±0.1	2.4±0.1	2.3±0.1			

APPENDIX 11

Computer program

```
//JACQUE    JOB (388609984,XXXXXXXXX,,3),'KATHY-FN',TIME=(3,59)
/*REGION         1000K
// EXEC SAS
//SYSIN DD *
DATA ONE;
INPUT (RAT GROUP INIWT WTWK1 WTWK2 WTWK3 WTWK4 WTWK5 WTWK6 WTWK7
       ENDWT WTREBWK1 WTREBWK2 FASTWK1 FASTWK2 FDWK1 FDWK2 FDWK3
       FDWK4 FDWK5 FDWK6 FDWK7 FDREWK1 FDREWK2 LIVER KIDNEY HEART
       VASTUS SP1 GASTROC ABDOFAT RETROFAT GONADFAT TOBEC1 TOBEC2
       TOBEC3 TOBEC4 TOBEC5 LIVERF KIDF HEARTF VASTUSF GASTROCF)
       (1. 1. 13*3. 9*3.1 3.1 2*2.1 3.2 2. #2 3.2 3*3.1 5*3. 3. 4*2.);
TOTGAIN=ENDWT-INIWT; REBWT=FASTWK2-ENDWT;
GAINPER=TOTGAIN/INIWT*100; REBPER=REBWT/ENDWT*100;
TOTFAT=ABDOFAT+RETROFAT+GONADFAT;
FATPER=TOTFAT/FASTWK2*100;
LIVERPER=LIVER/FASTWK2*100; KIDPER=KIDNEY/FASTWK2*100;
VASTPER=VASTUS/FASTWK2*100; GASTPER=GASTROC/FASTWK2*100;
RETROPER=RETROFAT/FASTWK2*100; GONADPER=GONADFAT/FASTWK2*100;
HEARTPER=HEART/FASTWK2*100; ABDOPER=ABDOFAT/FASTWK2*100;
LIVERFAT=LIVERF/10; KIDFAT=KIDF/10; HEARTFAT=HEARTF/10;
VASTFAT=VASTUSF/10; GASTFAT=GASTROCF/10;
MA=4.915+(0.0063188*(TOBEC1+62.56));
M1=SQRT(MA);
LBM1=(-2.217+M1)/0.003156;
MB=4.915+(0.0063188*(TOBEC3+62.56));
M3=SQRT(MB);
LBM3=(-2.217+M3)/0.003156;
MC=4.915+(0.0063188*(TOBEC4+62.56));
M4=SQRT(MC);
LBM4=(-2.217+M4)/0.003156;
MD=4.915+(0.0063188*(TOBEC5+62.56));
M5=SQRT(MD);
LBM5=(-2.217+M5)/0.003156;
LBMPER1=LBM1/INIWT*100; FATPER1=100-LBMPER1;
LBMPER3=LBM3/ENDWT*100; FATPER3=100-LBMPER3;
LBMPER4=LBM4/FASTWK1*100; FATPER4=100-LBMPER4;
LBMPER5=LBM5/FASTWK2*100; FATPER5=100-LBMPER5;
FAT1=FATPER1*INIWT/100; FAT3=FATPER3*ENDWT/100;
FAT4=FATPER4*FASTWK1/100; FAT5=FATPER5*FASTWK2/100;
FDWT1=FDWK1/WTWK1*100; FDWT2=FDWK2/WTWK2*100;
FDWT3=FDWK3/WTWK3*100; FDWT4=FDWK4/WTWK4*100;
FDWT5=FDWK5/WTWK5*100; FDWT6=FDWK6/WTWK6*100;
FDWT7=FDWK7/WTWK7*100; FDWTREB1=FDREWK1/WTREBWK1*100;
FDWTREB2=FDREWK2/WTREBWK2*100;
FATGAIN=(FAT5-FAT3)/FAT3*100; FATPGAIN=(FATPER5-FATPER3)/FATPER3*100;
LBMGAIN=(LBM5-LBM3)/LBM3*100; LBMPGAIN=(LBMPER5-LBMPER3)/LBMPER3*100;
AVWTA=(WTWK1+WTWK2+WTWK3+WTWK4+WTWK5+WTWK6+WTWK7)/7;
AVFDA=(FDWK1+FDWK2+FDWK3+FDWK4+FDWK5+FDWK6+FDWK7)/7;
AVFDWTA=(FDWT1+FDWT2+FDWT3+FDWT4+FDWT5+FDWT6+FDWT7)/7;
TOTFDA=AVFDA*49; FDEFFA=TOTGAIN/TOTFDA;
AVWTB=(WTREBWK1+WTREBWK2)/2; AVFDB=(FDREWK1+FDREWK2)/2;
AVFDWTB=(FDWTREB1+FDWTREB2)/2; TOTFDB=AVFDWTB*14;
FDEFFB=REBWT/TOTFDB;
CARDS;
$ADD JACDATA
PROC ANOVA;
CLASSES GROUP;
MODEL FASTWK1 FASTWK2 REBPER FAT3 FAT4 FAT5 FATGAIN FATPER3
      FATPER4 FATPER5 FATPGAIN LBM3 LBM4 LBM5 LBMGAIN LBMPER3 LBMPER4
      LBMPER5 LBMPGAIN LIVER LIVERPER LIVERFAT KIDNEY KIDPER KIDFAT
      HEART HEARTPER HEARTFAT VASTUS VASTPER VASTFAT GASTROC GASTPER
      GASTFAT RETROFAT RETROPER GONADFAT GONADPER ABDOFAT ABDOPER
      GAINPER TOTFAT FATPER LBM1 LBMPER1 FATPER1 FAT1 FDWT1 FDWT2
      FDWT3 FDWT4 FDWT5 FDWT6 FDWT7 FDWTREB1 FDWTREB2 AVWTA AVWTB
      TOTFDA TOTFDB=GROUP;
MEANS GROUP/LSD;
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REBOUND WEIGHT GAIN FOLLOWING EXERCISE OR MODERATE CALORIC RESTRICTION IN RATS

by

JACQUE STRUCKHOFF

B.S., Kansas State University, 1988

AN ABSTRACT OF A THESIS

submitted on partial fulfillment of the

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MASTER OF SCIENCE

Department of Foods and Nutrition

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1989

ABSTRACT

We compared rebound weight gain in mature female rats that had been exercised, feed restricted, or had received no treatment (control) for 7-wk periods. Exercised rats ran on a zero-grade treadmill (speed, 18 m/min; duration, 75 min/d; frequency, 5 d/wk). Animals in another group were feed-restricted so that their body weights were similar to those of the exercised animals. These treatments were discontinued and the animals were observed for an additional 2-wk rebound period. The exercised rats gained more weight, consumed more feed, and had higher feed efficiencies than control or feed-restricted rats. They also gained more lean body mass during the rebound period, according to measurements by total body electrical conductivity (TOBEC). This study suggests that mature female rats are more likely to recover or "rebound" from exercise than feed restriction.