THE EFFECTS OF EXERCISE ON GROWTH OF RATS RECOVERING FROM EARLY UNDERNUTRITION

by

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CHAPTER 1: REVIEW OF LITERATURE

Undernutrition and growth. Undernutrition during early stages of growth has permanent and irreversible effects. Widdowson and McCance (1) demonstrated that rats malnourished by litter expansion grew more poorly than those reared in smaller litters; and those differences persisted throughout their lives. When rats were underfed after three weeks of age, again, they grew more slowly than well-fed rats, but could recover with refeeding depending on the length of deprivation (2). These studies suggest that early stages of growth are more sensitive to nutritional inadequacy than later stages.

Enesco and LeBlond (3) have studied growth at a cellular level. Their method is based on the premise that the amount of deoxyribonucleic acid (DNA) per nucleus (6.2 pg in a rat diploid cell) is constant. The number of nuclei or cells in the tissue is estimated by dividing the total DNA in a tissue by the constant DNA content per nucleus. Cell size is estimated by dividing total tissue weight or protein content by the number of nuclei or cells (wt/DNA or protein/DNA).

Enesco and LeBlond used these calculations to describe three phases of growth in the rat. Phase one is characterized by an increase in cell number or hyperplasia. Growth during phase two is a combination of hyperplasia and hypertrophy (an increase in cell size). Phase three marks the end of almost all cell division; and growth occurs chiefly by hypertrophy.

Winick and Noble (4) investigated the effects of undernutrition during these three phases. Male rats were divided into three groups

where each group was deprived of food for 21 days at a different age. Group one was undernourished during suckling using larger than normal-size litters. Group two was given only one half of the required calories from 22 days to 43 days of age. Group three was restricted similarly for three weeks beginning at 65 days of age. All animals were refed after the deprivation period until sacrifice at 133 days of age. Total organ weight, protein, RNA, and DNA were measured after the period of caloric restriction and again after refeeding.

The results showed that rats underfed during suckling were unable to attain normal body weight, organ weight, or organ DNA content after refeeding. Animals in group two exhibited similar stunting except that brain and lung tissue had completely recovered. Rats underfed at the oldest age suffered no permanent depression in growth in any tissue measured.

These results were used by Winick and Noble to describe cellular growth of various organs (Fig. 1). All organ growth in rats until 22 days of age proceeds mainly by cell hyperplasia. From 22 to 42 days of age hyperplasia continues in all organs except brain and lung, because cell division is already complete in those organs. By 65 days all cell division is complete and growth proceeds by cellular hypertrophy.

They concluded that malnutrition at every stage inhibits increases in weight, total protein, and RNA. Increases in DNA, however, were diminished by caloric restriction only during critical early periods of growth. Thus, failure to grow can be attributed to two effects: reduction in cell number which results in permanent stunting or two, reduction in cell size which is reversible upon refeeding.

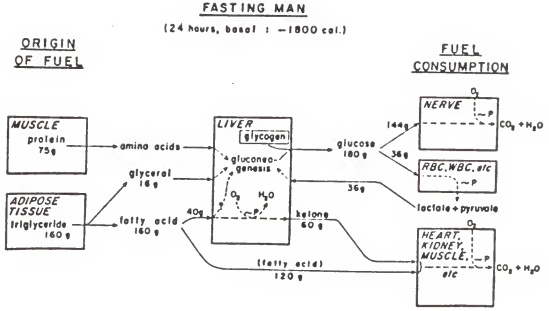

Figure 1. DNA (mg) during normal growth in the rat. DNA content reaches a maximum in every organ before growth stops. This indicates there must be a period of growth after which cells are no longer dividing, and the individual cell must be getting larger (5).

The growth of rat adipose cells is similar to that of other organs. Early growth of adipose tissue is mainly hyperplastic because undernutrition in suckling rats permanently reduces adipose cell number (6). On the other hand, malnutrition in adult rats decreases the size of fat deposits solely by decreasing cell size; cell number is unaffected (7, 8). Few new adipocytes are created in rats after 35 days of age. Rat adipocyte number is determined primarily during the preweaning period and is evidently fixed in adults (9).

Skeletal muscle growth and its response to undernutrition proves much more difficult to measure as it is multinucleated, consequently, DNA content (nuclear number) does not equate with cell number, and the ratio of protein/DNA does not reflect cell size (10).

Enesco and Puddy (11) studied the growth pattern of skeletal muscle in young rats and found that the increase in muscle size resulted from an increase in associated connective tissue, not an increase in muscle fiber number. The muscle fibers enlarged with age, DNA content increased due to increases in the number of nuclei. But DNA increases did not reflect fiber number increases.

Chronic food restriction reduced the diameter and length of muscle fibers in weanling rats (10) but did not change the number of fibers. Furthermore, DNA content was low in these deprived rats; the number of nuclei was reduced, but there was no change in measured cell number.

Some of these findings were supported by Picou et al. (12) and Cheek et al. (13) in studies on malnourished children. In children suffering from protein-calorie malnutrition, the major loss of muscle mass was due to loss of cell size rather than number.

As discussed, undernutrition inhibits cellular growth in organs and adipose tissue of suckling rats, and development of muscle fibers in young rats. In addition, stature is stunted in young rats. Kuramitsu et al. (14) reported rats fed diets deficient in either protein or energy had shorter femurs than controls because the malnourished rats experienced no growth spurt. Furthermore, appositional growth in bones slowed and eventually ceased during prolonged malnutrition in rats (15).

Metabolic effects of starvation. The metabolic events of starvation in man reviewed by Saudek and Felig (16) and Cahill (17), reflect the adaptations necessary to conserve protein and expend adipose tissue as a fuel source. They stated that even during brief starvation man's metabolism is markedly altered. The body adapts to maintain critical plasma glucose homeostasis, because glucose is required by certain tissues unable to metabolize fatty acids. Those tissues are nervous tissue, erythrocytes, leukocytes, bone marrow and renal medulla. If glucose is not provided by exogenous sources or declining glycogen stores, gluconeogenesis increases. Lactate and pyruvate are recycled into glucose via the Cori cycle which is driven by fatty acid oxidation (Fig. 2). Triglycerides also play a direct role as a glucose source, through the conversion of glycerol to glucose.

The most expensive source of glucose is the body's protein stores. Most tissues (muscle, heart, kidney, etc.) use little glucose during prolonged starvation, thereby sparing protein from gluconeogenesis. At the onset of starvation; however, the increase in gluconeogenesis demands the use of amino acids as a substrate. Pozefsky et al. (18) and

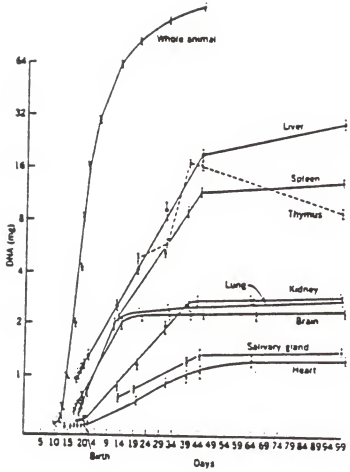

Figure 2. General scheme of fuel metabolism in a fasted man, showing the two primary sources, muscle and adipose tissue, and the three types of fuel consumers, nerve, pure glycolyzers and the remainder of the body that use fatty acids and ketones (17).

Felig et al. (19) measured the effect of brief starvation and found the amount of alanine, the principal nitrogenous glucose precursor, constituted greater than 30 percent of all amino acids released from the muscle. Consistent with this increase was the increased extraction of alanine by the liver for conversion to glucose. More alanine was converted into glucose in the liver than any other amino acid measured (20).

Alanine, however, is not the predominant amino acid in muscle, constituting only 5 to 7 percent of muscle protein. According to Goldberg and Odessey (21) and Odessey et at. (22) alanine is synthesized in the muscle by transamination of pyruvate with branched-chain amino acids (isoleucine, leucine, valine).

Adipose tissue is the largest energy reserve and the primary fuel source during starvation. Because citric acid cycle function is reduced, due to loss of substrates (particularly oxaloacetate) to gluconeogenesis, fatty acids are only partially oxidized to acetoacetate and beta-hydroxybutyrate (ketone bodies) (23, 24). Initially, the ketone bodies are metabolized for fuel by all organs with the exception of the tissues requiring glucose listed above. But in prolonged starvation, the body must reduce its need for glucose or protein, the major gluconeogenic source, would be fatally depleted after several weeks.

Owen et al. (25) found that one such adjustment occurs when the brain uses ketones instead of glucose as fuel during prolonged starvation. Up to 50 percent of the brain's energy requirements may be met by ketone bodies. Not only will these ketone bodies be used by the

brain as an energy source, but they provide a signal to the muscle to reduce amino acid catabolism and alanine output (26).

When rats are fasted they exhibit the same physiological adaptations seen in humans. Palou et al. (27) measured the effects of food deprivation for up to 24 hours on plasma metabolic parameters. Initially, liver and muscle glycogen were metabolized to maintain plasma glucose levels; liver glycogen levels dropped by 50 percent after three hours of fasting. Plasma glucose was maintained by gluconeogenic precursors. Lactate levels dropped sharply in the first three hours due to increased conversion to glucose. Fasting also induced a four- to five-fold increase in glucose synthesis from glycerol, and an increased formation of ketone bodies.

Hormonal responses to starvation. The metabolic events of starvation are triggered by a series of endocrinologic changes designed to conserve energy. Levels of hormones mediating fertility and growth decrease while hormones that maintain glucose homeostasis are enhanced. The metabolic response to hormonal flux can be broken into three basic phases (28). The absorptive phase begins after food intake and the subsequent rise in blood glucose concentrations. Rising insulin levels stimulate cellular uptake of glucose and amino acids, enhance lipogenesis, and inhibit lipolysis and gluconeogenesis. In the post-absorptive phase, growth hormone is secreted and, together with insulin, stimulates protein synthesis. In this way, growth hormone acts anabolically, mediating growth. Later, insulin levels decrease and glucose is cleared from circulation. In this phase, the fasting

phase, the action of growth hormone dominates. Growth hormone now mobilizes lipid stores, sparing plasma glucose and glycogen stores and, indirectly, protein. This anti-catabolic action of growth hormone is its second role.

These phases are greatly simplified because endocrine action during starvation is complex. Insulin is one of the key hormones in fasting metabolism though its levels are very low. Insulin, even in small amounts, indirectly inhibits peripheral lipolysis and proteolysis by lowering cyclic-AMP levels which, in turn, decrease catabolic enzyme concentrations (29, 17). Opposing and overriding insulin's effects during a fast are glucagon, growth hormone, thyroid stimulating hormone, and adrenocorticotropic hormone (17).

The low levels of insulin during starvation may also be directly associated with growth retardation. Children with diabetes mellitus, receiving inadequate insulin therapy, exhibited stunted growth though growth hormone levels were above normal (30).

Li et al. (31) found that muscle weight dropped in response to a 72-hour fast in rats, and that this was attributed to a decrease in the rate of protein synthesis. They proposed that a decrease in the level of plasma insulin was related to a block in peptide-chain initiation and a reduction in protein synthesis. Perfusion of the muscle in the presence of insulin removed the block, restoring protein synthesis.

The actions of insulin were similar to those of growth hormone in hypophysectomized rats (32, 33). Rapid growth was stimulated with slow-acting insulin and a high-carbohydrate diet. These rats grew heavier, longer, had increased epiphyseal cartilage width and increased nitrogen

retention compared to control animals. One explanation for these results was that insulin stimulated the utilization of amino acids and the synthesis of proteins while inhibiting protein catabolism. In the complete absence of insulin, growth hormone and other hormones exerted predominantly catabolic effects.

Unlike insulin, secretion of growth hormone varies with the type and severity of malnutrition (34, 35, 30). Elevated growth hormone levels were measured in children suffering from kwashiokor, a disease due to a protein deficient diet. Researchers speculated that because growth hormone is protein sparing, more is secreted during malnutrition to prevent protein depletion. On the other hand, victims of marasmus, protein-calorie malnutrition, showed low, normal or elevated growth hormone levels. Raghuramulu and Jaya Rao (36) inferred from low plasma amino acid levels in kwashiokor that raised levels of growth hormone may be an attempt to increase the efficiency of utilization of the available amino acids. In marasmus, however, plasma amino acids were in normal ranges as was growth hormone.

When rehabilitating malnourished children with elevated growth hormone levels, dietary protein reduced growth hormone levels more than an increase in caloric intake. Glucose administered to kwashiorkor patients did not produce a fall in growth hormone levels, and sometimes resulted in a rise (30). Pimstone et al. (34) found a strong inverse relationship between growth hormone levels and plasma concentrations of leucine, isoleucine, valine and, especially, arginine.

Elevated growth hormone levels have also been considered a stress response to starvation (37). Alternately, they may be attributed to a

decreased metabolic clearance rate of growth hormone, not to increased secretion (38).

Undernourished children are stunted despite typically normal or elevated growth hormone levels. As mentioned above, chronically low insulin levels may be one explanation. However, research by Phillips and Young (39) suggests that the somatomedins may be responsible for skeletal growth or lack of it in undernourished rats. Cartilage growth activity dropped significantly after 72 hours of fasting as did somatomedin levels, independent of growth hormone levels. Thus, the depression of growth could not be attributed to growth hormone deficiency. Refeeding reversed depressed somatomedian levels followed by an increase in cartilage growth activity.

Other mediators in the anabolic effects of growth hormone are the insulin-like growth factors (IGFs) (40). Fasting resulted in a decrease in both types of IGFs. These falling plasma levels of IGFs are thought to be responsible for the change in growth hormone activites, from growth-promoting to substrate mobilization for fuel homeostasis.

Cortisol levels are elevated during fasting to maintain glucose homeostasis by scavenging glucose precursors for gluconeogenesis and inhibiting peripheral glucose utilization (37). As hypothesized for growth hormone, high levels of cortisol measured in children suffering from marasmus were attributable to impaired degradation of cortisol rather than excess secretion (41).

High levels of glucocorticoids also interfere with growth hormone's effect on growth. They may result in decreased growth hormone secretion, interferrence with somatomedin generation or action, or

direct action on the cartilage to stunt growth (30).

Rehabilitation after undernutrition. Growth can be restored to undernourished children through refeeding; however, these children develop more body fat once the expected weight for height is reached (42). Harris & Widdowson (43) discovered the same trend when rehabilitating underfed rats. After refeeding, the rats remained smaller than controls but possessed the same amount of total lipids as the controls. Therefore, the refed rats contained a higher proportion of body fat. Feed intake was not calculated in this study. Bjorntorp and Yang (44) did measure "food efficiency" or the increase in body weight per gram of food consumed and concluded that food efficiency increases fivefold in refed-fasted rats. They suggested that "starvation-induced energy conservation processes seemed to persist during refeeding."

Departing from traditional methods of rehabilitating undernourished children, Torun et al. (45) observed that children recovering from protein-calorie malnutrition benefitted from mild exercise. Subjects, two to four years old, were involved either in sedentary activities common to programs dealing with malnourished children, or in daily, mild walking and running while playing. In the six week study, both groups showed improvement but the active group experienced significantly greater increases in height and lean-body mass as measured by creatinine excretion. The researchers concluded that exercise had a growth-enhancing effect. With an increase in activity, they suggested dietary amino acids may be more efficiently used and incorporated into muscle proteins.

Exercise and growth. Studies have shown that regular exercise in rats promotes growth of muscle (46, 47, 48) and bones (49, 46, 50, 51) while discouraging development of adipose tissue.

Muscle hypertrophy occurs in both isometric and isotonic exercise, but in different muscle components. Muscle actomyosin increases with isometric exercise while muscle sarcoplasm (the energy-storing protein) increases with isotonic exercise (46).

Watt et al. (47) reported significant growth in forelimb and hindlimb muscles of rats undergoing two weeks of brief isometric exercise. The muscles of the active rats had accumulated higher DNA, RNA and protein contents than the muscles of the sedentary controls. Studies in which rats performed isotonic exercise, such as daily swimming, also showed that exercise resulted in increased DNA concentration in the hindlimb muscles (48).

Bone growth in rodents is accelerated by voluntary exercise. Exercised hamsters had greater axial and appendicular growth than sedentary hamsters. Appendicular skeletal growth continued at a higher rate than controls even after exercising ceased (50). The body length of rats also showed greater gains with voluntary exercise (51).

Bones of rats allowed access to running wheels exhibited increased bone density in a 22-week study (49). In contrast, Saville and Whyte (46) observed no increase in bone densities of exercised rats over that of sedentary controls. In fact, as a result of daily running, the bones hypertrophied in proportion with adjacent muscles.

The antagonistic effects of regular exercise on development of adipose tissue in rodents are well documented (52, 53, 54, 51, 55, 56,

57, 58). Oscai et al. (55) subjected eight-day-old male rats to a swimming program for 15 weeks. Compared with controls, the exercised rats gained weight slower and had significantly lower final body weights. Much of the difference in weight was due to differences in fat content. Exercised rats possessed fewer and smaller adipocytes in epididymal fat pads.

In contrast, exercise in adult rats reduces the size of adipocytes but not their number (59, 60, 61). Lower fat content was measured in seven-week-old rats which ran on a motorized treadmill one hour per day, five days per week for nine to seventeen weeks. Reduced fat content in exercised rats was attributed to diminished adipocyte size, not number, when compared to controls (57).

Effects of exercise on food intake. Alterations in body weight and composition in exercised rats may be attributed, in part, to changes in food intake. Mayer et al. (52) found that female rats exercised up to one hour exhibited decreased food intake and weight loss; but those exercised longer periods of time increased their intake proportionally so that weight was maintained. In contrast to Mayer's findings, more recent studies showed that prolonged exercise suppressed appetite in male rats (54, 62, 63). Crews et al. (54) noted reduced food intake in male rats subjected to two hours of daily treadmill running. Oscai et al. (62) observed that a swimming program (six hours per day, six days per week) depressed weight gain in male rats but not in female rats. Only the exercised female rats, not the exercised male rats, increased their food intake with increased energy expenditure.

The suppression of appetite is thought to be the result of exercise of high intensity rather than that of long duration (64, 63). Several factors may explain the effect of exercise on food intake. Catecholamines, which are secreted during exercise, are thought to be related to appetite suppression (62, 63). Baile et al. (65) found that high levels of blood lactate, comparable to those found during severe exercise, depressed food intake.

Metabolism during exercise. The substrates metabolized during exercise are primarily carbohydrates and lipids. The ratio of their usage depends upon exercise duration and intensity (66). Initially, carbohydrate stored as glycogen in the muscle are burned by the exercising muscle. As exercise continues and muscle glycogen stores drop, glucose is required from hepatic glycogenolysis.

This phenomenon was confirmed by Blawacka et al. (67). After ten minutes of moderate exercise, the glycogen level in the muscle of rats decreased by about 65 percent and in the liver by about 33 percent. After thirty minutes, the reduction was 85 percent for the muscles and 60 percent for the liver.

The exercising muscle continues to increase its dependency on blood-borne glucose from the liver over time. Wahren et al. (68) reported that uptake of blood glucose by exercising muscle accounted for 75 to 90 percent of its total carbohydrate oxidation after 40 minutes. Glucose utilization by active muscle peaks at 90 minutes and then decreases.

Concomitantly, fat usage in the form of plasma free fatty acids increases progressively with the duration of exercise. Ahlborg et al. (69) found that free fatty acids provided 40 percent of the total energy after 40 minutes of activity in men. This amount increased to 50 percent at 3 hours and over 60 percent at 4 hours. They speculated that the increased uptake of free fatty acids by the muscle are a consequence of increased availability rather than improved extractability (69). During light exercise, triglycerides are mobilized from peripheral adipose tissue stores; during intense exercise, triglycerides come from within the muscle (70). Muscle catabolizes the fatty acids while the glycerol moiety is shunted to the liver for gluconeogenesis.

Gluconeogenesis becomes increasingly vital to plasma glucose homeostasis during prolonged exercise. Alborg et al. (69) found that hepatic gluconeogenesis accounted for 45 percent of the glucose released after four hours of exercise, compared to 20 to 25 percent at rest and at 40 minutes of exercise. Splanchnic uptake of gluconeogenic precursors, alanine, lactate, and pyruvate were doubled; glycerol oxidation increased tenfold.

The source of alanine for gluconeogenesis is active muscle, analogous to the glucose-alanine cycle described in starvation metabolism (20, 71). Felig & Wahren (71) reported muscle output of alanine was proportional to exercise intensity in men bicycling. They suggested that alanine served not only as a glucose precursor, but as a carrier of ammonia away from the muscle. The ammonia is a product of branched-chain amino acid oxidation within the muscle supplying additional energy for activity (72). Thus, amino acids, are a minor

energy source during exercise, providing only 5 to 15 percent of the energy utilized (73).

Hormonal response to exercise. During prolonged exercise, energy demands are met while plasma glucose levels are maintained. Similar to a fasting situation, the substrate shifts during exercise are mediated by hormones. Hartley et at. (74) measured hormonal responses in bicycling men. As exercise progressed, insulin levels fell; whereas, all stress hormone levels rose. These hormones include glucagon, catecholamines, growth hormone and cortisol.

Hypoglycemia does not occur during exercise even though glucose utilization accelerates. Exercise results in a fall in plasma insulin levels which, in turn, allow more hepatic glycogenolysis and gluconeogenesis (68, 75). Glucagon is secreted in response to the falling blood glucose and insulin levels (76). The release of glucagon, however, is dependent on exercise intensity. Increased levels were noted in strenuous exercise but not in moderate exercise (75).

Glucagon is a powerful gluconeogenic hormone in addition to modulating lipid metabolism. It provides sufficient lipid metabolites to support exercising muscle. Glucagon's lipolytic action is twofold. It stimulates the release of fatty acids from adipose stores and enhances the hepatic conversion of fatty acids to ketone bodies while suppressing synthesis of triglycerides (77).

Glucagon release is also stimulated by catecholamines, epinephrine and norepinephrine, which act as the initial messenger for adaption to exercise (76). Catecholamine levels increase as work becomes more

intense. Elevated heart rate, vascular resistance, and dropping glucose levels trigger catecholamine release (78). These hormones exert powerful lipolytic action and stimulate glucogenolysis in the liver and muscle through the stimulation of cellular cyclic-AMP (76, 78).

As part of a generalized stress response to exercise, growth hormone levels rise. Hunter et al. (79) suggested that increased growth hormone levels during exercise were important in mobilizing lipids during moderate exercise. Yet, during prolonged exercise levels have been noted to drop (79, 74) while free fatty acid levels continue to rise. This phenomenon and the delayed effect of growth hormone on adipose, raise doubts about its lipolytic role (80).

Hartley et al. (74) found cortisol levels rose prior to exercise and remained elevated 60 minutes after exhaustion. Cortisol mediates gluconeogenesis and mobilzation of amino acids and fatty acids for fuel (81). Unlike glucagon and catecholamines, cortisol's effects are delayed due to its mechanism of action. Cortisol is a mobile hormone, penetrating its target cell to stimulate new nuclear RNA and protein synthesis of catabolic enzymes (76).

In conclusion, during fasting and during exercise, animals resort to a post-absorptive state of metabolism. Though carbohydrates are a preferred substrate, peripheral stores of energy, adipose and protein, also are consumed to conserve glycogen and maintain blood glucose homeostasis.

Unlike starvation, exercise has been shown to have an anabolic effect. One explanation may be the altered hormone levels observed during exercise. Oversecretion of growth hormone in exercised hamsters

was associated with accelerated growth (82). In addition, some studies found androgen levels stimulated by exercise (83, 84, 85). Growth hormone and androgens have been found to produce greater somatic growth than growth hormone alone (30). And while growth hormone levels may be elevated in starvation, androgen levels are not (37).

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CHAPTER 2:

THE EFFECTS OF EXERCISE ON CATCH-UP GROWTH OF RATS RECOVERING FROM EARLY UNDERNUTRITION

INTRODUCTION

The growth retardation resulting from chronic undernutrition early in life has been extensively studied in man, rats, and other species (1-8). A return to normal nutriture and subsequent recovery is often characterized by a rapid rate of "catch-up growth" (9-12). This accelerated growth response is an attempt to normalize body size to a pre-retardation level. The degree of recovery may be affected by the type of diet used during rehabilitation (6,13-16), or the severity (17-19), duration (20), or age at onset (1-3) of the undernutrition period.

The objective of the present study was to examine the effects of exercise on growth during the recovery period in rats. Because growth is rapid during recovery, the animals may be particularly sensitive to external stimuli such as exercise. Exercise increases growth of skeletal muscle (21-23) and bones (24-26). Additionally, although exercise usually reduces voluntary feed intake in (male) rats (27-29), there are some reports that it increases intake in nutritionally-compromised animals (30,31).

Interest in this research stems from the previous finding that rats exercised during a feed deprivation regimen grew better than deprived animals not exercised (31). The present trial is conducted similarly except that rats began exercising after the feed deprivation period.

MATERIALS AND METHODS

Animals and Diet. Three-week-old male Wistar rats (Hsd:WI:BR, Harlan Sprague-Dawley, Indianapolis, IN), 55-85g each, were housed in wire-bottom, stainless steel cages (18 x 24 x 18 cm) in a temperature- and light-controlled room (21-23 °C, 12-h light/dark cycle). Rats were allowed water ad libitum throughout the study and were fed AIN-76A diet (Teklad Test Diets, Madison, WI), which is a nutritionally adequate, purified diet (Appendix 1). This diet has been previously described (32), and modified (33). Feed consumption was measured daily and rats were weighed twice weekly throughout the study.

Experimental protocol. Rats were randomly assigned to one of 2 groups for the first 4 wk of the study. Rats in one group received feed ad libitum; those in the other group were fasted every other day (EOD). Fasted EOD rats were given feed on alternate days in the morning hours, and the remaining feed was removed from the feed cups the following morning. This method of feed deprivation was chosen because of its simplicity and because it had been shown to cause growth retardation in young rats (31, 34-35).

After 4 wk, 8-9 rats from each group were killed to obtain baseline values and the remaining animals in both groups were divided into exercise and non-exercise sub-groups; all were allowed feed ad libitum. Exercised rats ran at a speed of 24 m/min (zero grade) for 75 min/d and 5d/wk (M-F) on a 1/2 hp Radiotrol treadmill (Boston Gears, Quincy, MA). To acclimitize the rats to running, the treadmill speed was progressively increased within the 75-min exercise period during the first 2 wk of the 8 wk exercise period.

Several rats were eliminated from the study. Three were eliminated from the first 4 wk of the study because they habitually dumped their feed from the feed cups and subsequently became underweight. Three more were dropped from the 8 wk exercise study: one for refusing to run on the treadmill, another was found to be a female, and the third had a chronic eye infection.

Killing procedures. After 8 wk, animals were killed by ether overdose, shaved, and dehaired with a commercial depilatory (Nair™, Carter-Williams Products Inc., NY). The lungs and the digestive tract were removed and, after decapitation, each carcass was weighed in air and then underwater to determine specific gravity (Appendix 2), and subsequently, body fat (36). Carcass lean mass was calculated as carcass weight minus carcass fat. Epididymal, retroperitoneal, and abdominal fat depots were removed and weighed. Livers, kidneys, hearts, brains, and vastus and gastrocnemius muscles from both hindlimbs were removed, weighed, and stored at -18°C for later analyses.

Analytical procedures. Femurs and tibias removed from left hindlimb were immediately cleaned of adherent tissue, dried in a 103°C forced air draft over for 24 h, and weighed after cooling in a desiccator. After bones were measured for length, they were soaked for 48 hr in deionized water and weighed underwater to determine specific gravity.

Protein and deoxyribose analyses were performed on livers, kidneys, brains, hearts, and vastus and gastrocnemius muscles (Fig 3). Appendices 3 and 4 describe reagents used in analyses. Whole livers were

homogenized on an Oster blender (Oster, Milwaukee, WI); other tissues were homogenized using a Polytron high speed homogenizer (Brinkman Instruments, Westburg, NY). Tissue protein was measured by the Lowry et al. procedure (37a). Deoxyribose was extracted from the tissues by the method described by Schneider (37b) and then assayed by the diphenylamine reaction (37c). Statistical tests employed were least significant differences tests following significant ($P < 0.05$) analysis of variance procedures (38). The computer program used for data analysis is in Appendix 5.

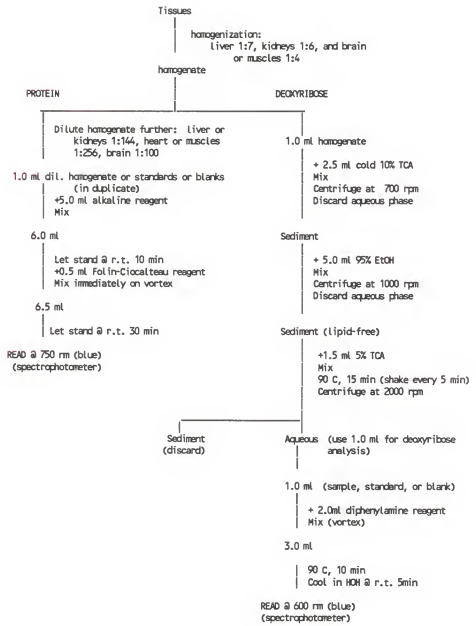

Fig 3. Protein and deoxyribose extraction and analysis

RESULTS

Weight gain, carcass composition and feed intake. Rats killed after fasting EOD the first 4 wk ate 46% less feed and gained 44% less weight than those fed ad libitum, even though they had free access to feed on alternate days (Table 1). Carcasses of the fasted EOD rats also contained 53% less fat and 37% less lean mass than those of the ad libitum animals. When the fasted EOD rats were refed for the next 8 wk they still remained smaller than rats that had been fed ad libitum, regardless of exercise treatments.

The 8-wk exercise program after the initial feeding period reduced weight gain in both groups of rats, but had a greater effect on rats that had been fasted EOD. Underfed rats that were exercised gained 24% less weight, and their carcasses contained 46% less fat and 8% less lean mass than underfed rats that were not exercised. In rats that had always been fed ad libitum, exercise reduced weight gain by 20% and carcass fat by 44%, but actually increased carcass lean mass by 2%.

Tissue Growth. The smaller weight gains of the fasted EOD rats after the 4-wk period were accompanied by smaller tissues and organs as well (Table 2). Livers, kidneys, brains, and gastrocnemius and vastus hindlimb muscles were lighter and had less protein contents in fasted EOD rats than ad libitum fed rats.

Exercise reduced liver weights and liver protein contents in both groups of animals. However, the effects of exercise on other tissues were different in early underfed and normal-fed rats. In previously ad libitum fed rats, exercise increased the weights and protein contents of

kidneys, brain, and hindlimb muscles; in fasted EOD rats exercise reduced growth of those tissues.

Perhaps the most dramatic effect of the 4-wk fasting EOD regimen was its effect on fat depots (Table 3). Epididymal, retroperitoneal, and abdominal fat pads were reduced in weight by 50 to 72%. When animals were refed they gained weight in those tissues; however the retroperitoneal depot still remained lighter in the fasted EOD rats even after 8 weeks of refeeding.

Exercise reduced weights of all three fat pads in both ad libitum and fasted EOD rats to a similar extent, between 37 to 44%, over the 8-week period. Exercise further seemed to reduce weight of all three fat pads comparably, without preference for any one location.

Hindlimb bone growth was also studied as a function of exercise and early nutriture. Fasting EOD for 4-wk reduced femur and tibia wt and length; those effects persisted even after refeeding. Exercise tended to increase bone weights in ad libitum fed rats even though the effects were not statistically significant. In contrast, exercise in underfed rats significantly reduced femur weight by 10%.

Tissue nucleic acid contents. We performed DNA analysis on tissues but found the values variable and higher than those usually reported. Consequently we did not include them in our research results, but they are shown in Appendices 12-16.

TABLE 1

Effects of exercise following normal or under-nutrition on weight gain, body composition, [1] and feed intake of growing male rats

Measurement	Ad Libitum			Fasted EOD			[3] ANOVA
	[2] (Baseline)	Followed by 8-wk -Exercise	+Exercise	(Baseline)	Followed by 8-wk -Exercise	+Exercise	
Initial wt, g	(72 ±2)	295 ±6	315 ±6	(70 ±2)	204 ±6	205 ±6	Fasted
Final wt, g	(297 ±8)	549 ±13	516 ±14	(196 ±9)	521 ±14	447 ±14	Fasted, Exercise
Total wt gain, g	(225 ±8)	254 ±12	200 ±12	(126 ±9)	317 ±12	242 ±12	Fasted, Exercise
Ave. daily wt gain, g	(8.0 ±0.3)	4.5 ±0.2	3.6 ±0.2	(4.5 ±0.3)	5.7 ±0.2	4.3 ±0.2	Fasted, Exercise
Carcass composition							
Wt, g	(253 ±7)	476 ±13	441 ±14	(152 ±7)	448 ±14	380 ±14	Fasted, Exercise
Fat, g	(45 ±3)	98 ±6	55 ±6	(21 ±3)	79 ±6	43 ±6	Fasted, Exercise
Fat, %	(18 ±1)	20 ±1	13 ±1	(14 ±1)	18 ±1	11 ±1	Exercise
Lean mass, g	(208 ±5)	378 ±9	385 ±10	(131 ±5)	368 ±10	337 ±10	Fasted
Lean mass, %	(82 ±1)	80 ±1	87 ±1	(86 ±1)	82 ±1	89 ±1	Exercise
Ave. daily feed intake							
total g/d	(19.8 ±0.5)	24.9 ±0.6	21.9 ±0.7	(10.7 ±0.5)	24.2 ±0.7	20.9 ±0.7	Exercise
per 100g body wt	(13.7 ±0.2)	6.0 ±0.1	5.4 ±0.1	(9.2 ±0.3)	7.1 ±0.1	6.7 ±0.1	Fasted, Exercise
Feed efficiency, g total gain/g total feed	(0.41 ±0.01)	0.18 ±0.01	0.16 ±0.01	(0.42 ±0.01)	0.23 ±0.01	0.21 ±0.01	Fasted, Exercise

[1] Values are means ± SEM for 8 or 9 rats.

[2] Baseline values obtained after the initial 4-wk feeding period.

[3] Two-way analysis of variance performed on the post exercise or non-exercise groups. Main treatment effects listed are significant (P 0.05).

TABLE 2

Effects of exercise following normal or under-nutrition on growth and protein contents in tissues of growing male rats [1]

Measurement	Ad Libitum			Fasted ECD			
	[2] (Baseline)	Followed by 8-wk -Exercise	Followed by 8-wk +Exercise	(Baseline)	Followed by 8-wk -Exercise	Followed by 8-wk +Exercise	[3] ANOVA
Liver							
Wt, g	(17.53±0.63)	24.79±1.26	22.88±1.33	(11.44±0.67)	21.76±1.33	18.10±1.33	Fasted, Exercise
Wt, g/100g body wt	(5.88±0.12)	5.31±0.23	5.18±0.24	(5.86±0.13)	5.20±0.24	4.86±0.24	N.S.
Protein, total g	(2.58±0.11)	3.79±0.19	3.40±0.20	(1.60±0.12)	2.99±0.20	2.76±0.20	Fasted
Kidneys (both)							
Wt, g	(2.66±0.05)	3.49±0.12	3.71±0.12	(1.86±0.05)	3.38±0.12	3.30±0.12	Fasted
Wt, g/100g body wt	(0.90±0.02)	0.75±0.02	0.84±0.02	0.95±0.02)	0.81±0.02	0.89±0.02	Fasted, Exercise
Protein, total mg	(388.6±8.8)	557.2±20.2	616.5±21.4	(268.3±10.0)	541.9±21.4	536.5±21.4	Fasted
Heart							
Wt, g	(1.10±0.13)	1.43±0.17	1.38±0.09	(0.71±0.05)	1.38±0.06	1.24±0.08	N.S.
Wt, g/100g body wt	(0.37±0.03)	0.26±0.02	0.27±0.01	0.36±0.02)	0.26±0.01	0.28±0.02	N.S.
Protein, total mg	(169.9±12.9)	216.9±37.7	206.0±25.0	(111.3±18.8)	207.4±31.9	209.6±15.2	N.S.
Brain							
Wt, g	(1.98±0.02)	2.19±0.04	2.25±0.04	(1.81±0.02)	2.16±0.04	2.06±0.04	Fasted, Fasted*Exercise
Wt, g/100g body wt	(0.67±0.03)	0.47±0.01	0.51±0.02	(0.93±0.03)	0.52±0.02	0.56±0.02	Fasted, Exercise
Protein, total mg	(194.3±2.8)	223.4±4.2	226.4±4.4	(178.0±3.0)	213.6±4.4	213.4±4.4	Fasted
Gastrocnemius muscle							
Wt, g	(3.11±0.09)	5.59±0.14	5.86±0.15	(2.25±0.09)	5.75±0.15	5.36±0.15	Fasted*Exercise
Wt, g/100g body wt	(1.05±0.03)	1.20±0.03	1.33±0.03	(1.15±0.03)	1.38±0.03	1.45±0.03	Fasted, Exercise
Protein, total mg	(504.0±17.2)	880.0±31.2	841.3±33.1	(355.0±18.2)	831.8±33.1	866.0±33.1	N.S.
Vastus muscle							
Wt, g	(3.06±0.08)	3.34±0.13	3.49±0.14	(2.15±0.08)	3.36±0.14	3.21±0.14	N.S.
Wt, g/100g body wt	(1.03±0.02)	0.72±0.03	0.79±0.03	(1.10±0.02)	0.81±0.03	0.87±0.03	Fasted, Exercise
Protein, total mg	(549.3±19.4)	601.6±15.3	665.3±16.3	(399.3±20.6)	622.4±16.3	582.3±16.3	Fasted*Exercise

[1] Values are means ± SEM for 8 or 9 rats. Muscles from both hindlimbs were pooled for measurements.

[2] Baseline values obtained after the initial 4-wk feeding period.

[3] Two-way analysis of variance performed on the post exercise or non-exercise groups. Main treatment effects listed are significant ($P < 0.05$).

TABLE 3

Effects of exercise following normal or under-nutrition on growth of fat depots and hindlimb bones of growing male rats[1]

Measurement	Ad Libitum			Fasted EOD			ANOVA[3]
	(Baseline)[2]	Followed by 8-wk -Exercise	Followed by 8-wk +Exercise	(Baseline)	Followed by 8-wk -Exercise	Followed by 8-wk +Exercise	
Fat depots							
Epididymal fat							
Wt, g	(3.6 ±0.2)	11.1 ±0.7	7.0 ±0.7	(1.6 ±0.2)	9.7 ±0.7	6.0 ±0.7	Exercise
Wt, g/100g body wt	(1.2 ±0.1)	2.4 ±0.1	1.6 ±0.1	(0.8 ±0.1)	2.3 ±0.1	1.6 ±0.1	Exercise
Retroperitoneal fat							
Wt, g	(5.8 ±0.4)	21.8 ±1.2	12.9 ±1.2	(1.6 ±0.5)	16.6 ±1.2	9.3 ±1.2	Fasted, Exercise
Wt, g/100g body wt	(1.9 ±0.1)	4.7 ±0.2	2.9±0.2	(0.8 ±0.1)	4.0 ±0.2	2.5 ±0.2	Fasted, Exercise
Abdominal fat							
Wt, g	(4.2 ±0.3)	13.8 ±0.8	8.3 ±0.9	(2.1 ±0.3)	12.4 ±0.9	7.0 ±0.9	Exercise
Wt, g/100g body wt	(1.4 ±0.1)	2.9 ±0.1	1.9 ±0.2	(1.1 ±0.1)	3.0 ±0.2	1.9 ±0.2	Exercise
Hindlimb bones							
Tibia							
Wt, mg	(389 ±7)	564 ±16	586 ±17	(286±7)	556 ±17	520 ±17	Fasted
Length, mm	(36 ±0)	40 ±0	40 ±0	(33 ±0)	40 ±0	40 ±0	N.S.
Specific gravity	(1.53 ±0.02)	1.64 ±0.02	1.57 ±0.02	(1.43 ±0.02)	1.58 ±0.02	1.60 ±0.02	N.S.
Femur							
Wt, mg	(417 ±6)	818 ±18	841 ±19	(309 ±6)	812 ±19	733 ±19	Fasted, Fasted*Exercise
Length, mm	(31 ±0)	38 ±0	39 ±0	(28 ±0)	38 ±0	37 ±0	Fasted
Specific gravity	(1.57 ±0.02)	1.53 ±0.02	1.52 ±0.02	(1.46 ±0.02)	1.52 ±0.02	1.52 ±0.02	N.S.

1 Values are means ± SEM for 8 or 9 rats.

2 Baseline values obtained after the initial 4-wk feeding period.

3 Two-way analysis of variance performed on the post-exercise or non-exercise groups. Main treatment effects listed are significant (P 0.05).

DISCUSSION

Our study shows that exercise during recovery from early undernutrition reduces catch-up growth in rats. Exercise following an intermittent fasting period reduced feed intake, feed efficiency, weight gain, and carcass contents of fat and lean mass. The smaller weight gains of the exercised underfed rats were accompanied by smaller visceral organs and hindlimb muscles and bones.

Exercise had somewhat different effects in rats fed ad libitum early in life. Exercise again (to a lesser extent) reduced feed intake, feed efficiency, weight gain, and carcass fat; but carcass lean mass was increased slightly and weights of kidneys, brains, and hindlimb muscles and bones were maintained or increased.

Our results suggest that the response to exercise can be modified by prior nutritional status. The underfed rats may not have been able to respond normally to exercise because of physiological impairments resulting from early undernutrition. Feed deprivation in young animals has been shown to reduce muscle growth (1,39-42) and oxidative capacity (43-45), and result in earlier fatigability during exercise (43). Previously undernourished rats exhibited poorer motor coordination and balance than control rats when tested later in life (46,47). Human studies demonstrate poor work capacity in malnourished children and adults (see review, ref. 48). These studies suggest that, at best, a prior period of undernutrition would make exercise difficult; it would also probably affect the normal training response as well.

On the other hand, there is some evidence that exercise may be a useful adjunct for the recovery from undernutrition. Torun et al. (49)

compared growth responses of active and sedentary 2 to 4-year-old Guatemalan children recovering from malnutrition. The active children participating in games involving moderate energy expenditure grew taller and had more lean body mass than children who followed the typical hospital level of physical activity (sedentary games). More recently, this laboratory found that young rats exercised during an intermittent fasting regimen ate more feed and gained more weight than fasted rats not exercised (31). Those data led to the question of whether exercise would have the same beneficial effects if begun after the period of undernutrition. As shown, exercise begun after the undernutrition period was not beneficial; it actually reduced growth.

A comparison of both these studies may reveal why exercise had apparently opposite effects. Both studies employed the same diet, method of feed deprivation, and exercise program; they differed only in the timing of exercise relative to the undernutrition period.

The physiological response of an animal to undernutrition is quite different from that response during recovery or "catch-up growth", and exercise may have different effects during those times. Undernutrition is characterized by a reduction in metabolic rate (50), growth rate (1-3), protein synthesis (14,38,51), and circulating levels of somatomedin C (52), thyroxine (53), and insulin (5,51,54). These physiological adjustments reflect the need to preserve "self" when resources are limited. Exercise may have been beneficial during that time because it allowed the animals to eat more on the days that they were allowed feed (31). Also, exercise is known to increase growth hormone levels

(55,56) and reduce the need for insulin (57,58) which is needed for growth.

Recovery following undernutrition is also a period of physiological stress, because demands imposed by rapid growth imply greater tissue deposition and subsequent nutrient need. During catch up growth voluntary food intake greatly increases (59) and efficiency of weight gain is improved (59,60). Serum levels of growth hormone (61) and insulin (51,54) increase, and those of corticosterone decrease (51) which are changes consistent with anabolic processes. Exercise during this rapid period of growth may have reduced growth further because it increases, irreversibly, the oxidation of branched chain amino acids (62,63), which are essential to growth.

Another potential factor affecting the growth response to exercise is the age of the rats when they begin to exercise. When rats were exercised during the undernutrition period they were younger and possibly more responsive to environmental stimuli. Winick and Noble (64) have shown that cellular growth of most organs and tissues in rats is complete by 44 days of age. Consequently exercise may have a greater impact when performed during the linear portion of the growth curve, when growth is more plastic and perhaps more easily modified by external factors. The clinical implications of our study suggest that exercise may be more valuable to growth if begun early in life before growth is complete (i.e. a "critical period" for exercise). Alternately, exercise may reduce recovery from undernutrition if begun at a later stage of the life cycle.

The effects of exercise on individual organs and tissues has previously been studied. Dohm and coworkers found exhaustive bouts of exercise significantly reduced portein levels of skeletal muscle and liver (66, 67). They reported that exercise caused a decrease in protein synthesis and an increase in protein degradation in muscle and liver. This protein loss was accompanied by increased free lysosomal activity. And in rats which were fasted before exercise, activity levels were severalfold greater. They suggested that fasting alters the structure of lysosomes which further enhances the catabolic effect of exercise. Our data on liver response confirm these findings. Muscle growth, however, was stimulated by exercise in rats fed ad libitum. Our rats were not exhaustively exercised; protein loss in these tissues may be dependent on the duration of exercise. Also protein synthesis has been found to increase after exercise (68) and may overcome any catabolic effects of exercise.

Recovery from undernutrition has also been examined for its effects on body composition. Harris and Widdowson (4) studied 3-wk-old rats fed a restricted diet for 10 wks followed by another 12 wks of unlimited diet. They found that the previously undernourished rats deposited more fat during rehabilitation than the control animals in the same number of days and over the same gain in body-weight.

Bjorntorp and co-workers have examined the physiological changes that occur during short-term fasting and refeeding in rats (60). They found that when 2-month-old rats were fasted for 65 hr and refed ad libitum their body weight and body composition returned to the levels of nonfasting controls by 8 days and that feed efficiency was higher during

the refeeding period than ad libitum eating. The refeeding period was characterized by an increase in glucose turnover, synthesis of glycogen and lipid in liver and adipose tissue, and repletion of protein losses in liver and muscle (54). When rats were exercised during the 8-day refeeding period they re-gained less body weight and fat without compromising the restoration of lean tissue (65).

Data from our study support those findings with only a few departures, which might be attributed to differences in experimental protocol. Our weanling (4-wk-old) male rats fed a restricted diet for 4 wk gained more weight and were more feed efficient during the refeeding period than rats that had been fed ad libitum; but they did not attain the body weights of the ad libitum fed rats over the 8-wk period. Their carcass percent fat was comparable at the end of the study. However exercise reduced the amount of fat re-gained and also, to a lesser extent, the amount of lean body mass re-gained in previously underfed rats. Our rats may not have been able to re-synthesize lean mass as effectively because their energy stores were low. This result of underfeeding coupled with the demands of exercise, may have reduced the energy available for growth.

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APPENDIX

APPENDIX 1

Composition AIN-76A purified diet[1]

Ingredient	Amount
	g/kg
Casein, high protein	200.0
DL-Methionine	3.0
Sucrose	499.99
Corn starch	150.0
Corn oil	50.0
Non-nutritive fiber (cellulose)	50.0
Mineral mix[2]	35.0
Vitamin mix[3]	10.0
Choline bitartrate	2.0
Ethoxyquin	0.01

[1] Supplied by Teklad Test Diets, Madison, WI.

[2] Supplied as AIN-76 mineral mix in grams/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 grams/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 B12 (0.1% trituration in mannitol), 1.0; dry vitamin A palmitate (500,000U/g), 0.8; vitamin D3 trituration (400,000U/g), 0.25; dry vitamin E acetate, 10.0; menadione sodium bisulfite complex, 0.15; sucrose finely powdered, 981.08.

APPENDIX 2

Density of water at different temperatures[1]

Temperature (°C)	D_w (grams/ml)[2]
21	0.9980
22	0.9978
23	0.9975
24	0.9973
25	0.9971
26	0.9968
27	0.9965
28	0.9963
29	0.9960
30	0.9957
31	0.9954
32	0.9951
33	0.9947
34	0.9944
35	0.9941
36	0.9937
37	0.9934
38	0.9930
39	0.9926
40	0.9922

[1] Extracted from Weast, R.C. (Ed.): Handbook of Chemistry and Physics 54th ed., Cleveland: The Chemical Rubber Company, 1967, p. F-11.

[2] Rounded to 0.0001.

APPENDIX 3

Reagents for protein analysis (Folin-Lowry)[1]

1. Alkaline sodium carbonate solution (2% Na_2CO_3 in 0.1 N NaOH).
   Dissolve 20 grams Na_2CO_3 and 4 grams NaOH in a liter of deionized water.

2. Copper sulphate-sodium potassium tartrate solution (0.5% $CuSO_4$ in 1% Na, K tartrate).
   Dissolve 0.25 grams $CuSO_4$ in 50 ml 1% Na, K tartrate.

3. Alkaline solution prepare day of use 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. Stock standard protein solution (0.2 mg/ml):
   Dissolve 50 mg bovine serum albumin (Sigma Chemical Co., St. Louis, MO) in 250 ml deionized water, store refrigerated.

6. Working protein standards (0.0-0.2 mg/ml):
   The stock protein solution is diluted appropriately to obtain working standards.

Dilution factors for protein samples, diluted with ddH2O during homogenizing and before analysis: brains, 500 (5x100); livers, 1008 (7x144); kidneys, 864 (6x144); muscles, 1280 (5x256).

[1] Procedure described in: Plummer, D.T. (1971) An Introduction to Practical Biochemistry, McGraw Hill. London, UK. pp. 156-157.

APPENDIX 4

Reagents for deoxyribose analysis

1. Deoxyribose stock standard (0.4 mg/ml):
   Dissolve 40 mg of deoxyribose in 100 ml deionized water and store at 4 C. (Deoxyribose purchased from Sigma Chemical Co., St. Louis, MO).

2. Deoxyribose intermediate standard (40 ug/ml):
   Mix one ml of deoxyribose stock standard solution with 9.0 ml of deionized water.

3. Working deoxyribose standards (0-40 ug/ml):
   Prepare from intermediate standard, one ml is run parallel with samples.

4. 10% TCA:
   Ten grams of tricholoacetic acid is dissolved in deionized water and brought up to 100 ml.

5. Diphenylamine reagent:
   Dissolve 0.5 grams of diphenylamine in 50 ml glacial acetic acid and 1.37 ml of concentrated sulfuric acid, mix. Prepare fresh daily.

Dilution factors for nucleic acid samples, diluted with ddH2O during homogenizing and with 5% TCA during separation: brains, 12.5 (5x2.5); liver, 14 (7x2); kidneys, 12 (6x2); muscles, 10 (5x2).

```
//JCKEN     JOB (3830C9984,          ,,7),'DEBBIE-FN',TIME=(3,57)
/*ROUTE  PRINT LOCAL
/*REGION        500K
// EXEC SAS
//SYSIN DD *
DATA ONE;
TITLE EIGHT WEEK STUDY AFTER EXERCISE;
INPUT CID GROUP SATWT1 MONWT1 SATWT2 MONWT2 SATWT3 MONWT3 SATWT4
      MONWT4 SATWT5 MONWT5 SATWT6 MONWT6 SATWT7 MONWT7 SATWT8 MONWT8
      SATWT9 SP2 FEEDSS1 FEEDMF1 FEEDSS2 FEEDMF2 FEEDSS3 FEEDMF3
      FEEDSS4 FEEDMF4 FEEDSS5 FEEDMF5 FEEDSS6 FEEDMF6 FEEDSS7
      FEEDMF7 FEEDSS8 FEEDMF8 SP3 PERFAT LIVERWT KIDNEYWT HEARTWT
      ABDOMFAT RETROFAT EPIDFAT TIBIAWT TIBIALS TIBIAWI FEMURWT
      FEMURLE FEMURWI BRAINWT VASTUSWT GASTRWT DUMPER CARCASS EOD EXER
      FEMURBS TIBIABS FEMURSG TIBIASG VASTUSP GASTROCD
      GASTROCP BRAINP LIVERP KIDNEYD KIDNEYP)
      (2. 1. 17*1. 1. 3.1 4.1 3.1 4.1 3.1 4.1 #2 4.1 3.1 4.1 3.1
      4.1 3.1 4.1 3.1 4.1 1. 5.3 4.2 2*3.2 3.4 3. 3.1 2.1 3.
      3.1 2.1 3.2 #3 2*2.1 1. 3. 2*1. 2*3.2 2*4.3 3.3 4.1 3.2 4.1
      3.2 4.1 3.2 4.1 3.2 4.1);
VASTDNA=VASTUSD*VASTUSWT; VASTPRO=VASTUSP*VASTUSWT;
GASTDNA=GASTROCD*GASTRWT; GASTPRO=GASTROCP*GASTRWT;
BRNDNA=BRAIND*BRAINWT;    BRNPRO=BRAINP*BRAINWT;
LIVDNA=LIVERD*LIVERWT;    LIVPRO=LIVERP*LIVERWT;
KIDDNA=KIDNEYD*KIDNEYWT;  KIDPRO=KIDNEYP*KIDNEYWT;
VASTUSR=VASTPRO/VASTDNA; GASTROCR=GASTPRO/GASTDNA;
BRAINR=BRNPRO/BRNDNA; LIVERR=LIVPRO/LIVDNA; KIDNEYR=KIDPRO/KIDDNA;
TOTGAIN=SATWT9-SATWT1;
FDMF7=(FEEDMF1*FEEDNF2*FEEDMF3*FEEDMF4*FEEDMF5*FEEDMF6*FEEDMF8)/7;
FDSS7=(FEEDSS1*FEEDSS2*FEEDSS3*FEEDSS4*FEEDSS5*FEEDSS6*FEEDSS8)/7;
TOTFEED=FEEDSS1*FEEDMF1*FEEDSS2*FEEDMF2*FEEDSS3*FEEDMF3*FEEDSS4*
        FEEDMF4*FEEDSS5*FEEDMF5*FEEDSS6*FEEDMF6*FDSS7*FDMF7*
        FEEDSS8*FEEDMF8;
FEEDEFF=TOTGAIN/TOTFEED;
FEEDDAY=TOTFEED/56;
GAINWT1=MONWT1-SATWT1; GAINMF1=SATWT2-MONWT1;
GAINSS2=MONWT2-SATWT2; GAINMF2=SATWT3-MONWT2;
GAINSS3=MONWT3-SATWT3; GAINMF3=SATWT4-MONWT3;
GAINSS4=MONWT4-SATWT4; GAINMF4=SATWT5-MONWT4;
GAINSS5=MONWT5-SATWT5; GAINMF5=SATWT6-MONWT5;
GAINSS6=MONWT6-SATWT6; GAINMF6=SATWT7-MONWT6;
GAINSS7=MONWT7-SATWT7; GAINMF7=SATWT8-MONWT7;
GAINSS7=(GAINSS1+GAINSS2+GAINSS3+GAINSS4+GAINSS5+GAINSS6+
   GAINSS8)/7;
GAINMF7=(GAINMF1+GAINMF2+GAINMF3+GAINMF4+GAINMF5+GAINMF6+
   GAINMF8)/7;
AVSSGAIN=(GAINSS1+GAINSS2+GAINSS3+GAINSS4+
         GAINSS5+GAINSS6+GAINSS7+GAINSS8)/16;
AVMFGAIN=(GAINMF1+GAINMF2+GAINMF3+GAINMF4+
         GAINMF5+GAINMF6+GAINMF7+GAINMF8)/40;
GAINDAY=TOTGAIN/56;
AVSSFD1=FEEDSS1/2; AVMFFD1=FEEDMF1/5;
AVSSFD2=FEEDSS2/2; AVMFFD2=FEEDMF2/5;
AVSSFD3=FEEDSS3/2; AVMFFD3=FEEDMF3/5;
AVSSFD4=FEEDSS4/2; AVMFFD4=FEEDMF4/5;
AVSSFD5=FEEDSS5/2; AVMFFD5=FEEDMF5/5;
AVSSFD6=FEEDSS6/2; AVMFFD6=FEEDMF6/5;
AVSSFD8=FEEDSS8/2; AVMFFD8=FEEDMF8/5;
AVSSFD7=(AVSSFD1*AVSSFD2*AVSSFD3*AVSSFD4*AVSSFD5*AVSSFD6*
   AVSSFD8)/7;
AVMFFD7=(AVMFFD1*AVMFFD2*AVMFFD3*AVMFFD4*AVMFFD5*AVMFFD6*
   AVMFFD8)/7;
AVSSFD=(AVSSFD1*AVSSFD2*AVSSFD3*AVSSFD4*
        AVSSFD5*AVSSFD6*AVSSFD7*AVSSFD8)/8;
AVMFFD=(AVMFFD1*AVMFFD2*AVMFFD3*AVMFFD4*
        AVMFFD5*AVMFFD6*AVMFFD7*AVMFFD8)/8;
```

```
FDWTSS1=AVSSFD1/SATWT1*100;  FDWTMF1=AVMFFD1/MONWT1*100;
FDWTSS2=AVSSFD2/SATWT2*100;  FDWTMF2=AVMFFD2/MONWT2*100;
FDWTSS3=AVSSFD3/SATWT3*100;  FDWTMF3=AVMFFD3/MONWT3*100;
FDWTSS4=AVSSFD4/SATWT4*100;  FDWTMF4=AVMFFD4/MONWT4*100;
FDWTSS5=AVSSFD5/SATWT5*100;  FDWTMF5=AVMFFD5/MONWT5*100;
FDWTSS6=AVSSFD6/SATWT6*100;  FDWTMF6=AVMFFD6/MONWT6*100;
FDWTSS7=AVSSFD7/SATWT7*100;  FDWTMF7=AVMFFD7/MONWT7*100;
FDWTSS8=AVSSFD8/SATWT8*100;  FDWTMF8=AVMFFD8/MONWT8*100;
FDWTSS7=(FDWTSS1+FDWTSS2+FDWTSS3+FDWTSS4+FDWTSS5+FDWTSS6+
   FDWTSS8)/7;
FDWTMF7=(FDWTMF1+FDWTMF2+FDWTMF3+FDWTMF4+FDWTMF5+FDWTMF6+
   FDWTMF8)/7;
AVFDWTSS=(FDWTSS1+FDWTSS2+FDWTSS3+FDWTSS4+
         FDWTSS5+FDWTSS6+FDWTSS7+FDWTSS8)/8;
AVFDWTMF=(FDWTMF1+FDWTMF1+FDWTMF3+FDWTNF4+
         FDWTMF5+FDWTMF6+FDWTMF7+FDWTMF8)/8;
FDWTDAY=((AVFDWTSS*10)+(AVFDWTMF*40))/50;
LBM=(100-PERFAT);
FDEFFIC=TOTGAIN/TOTFEED;
CARCLBM=(LBM/100)*CARCASS;
CARCFAT=(PERFAT/100)*CARCASS;
ABDOMPER=(ABDOMFAT/SATWT5)*100;
RETROPER=(RETROFAT/SATWT5)*100;
EPIDPER=(EPIDFAT/SATWT5)*100;
DEPOPER=(ABDOMPER+RETROPER+EPIDPER);
FATSUM=ABDOMFAT+RETROFAT+EPIDFAT;
LIVERPER=(LIVERWT/SATWT5)*100;  KIDPER=(KIDNEYWT/SATWT5)*100;
HEARTPER=(HEARTWT/SATWT5)*100;  BRAINPER=(BRAINWT/SATWT5)*100;
VASTPER=(VASTUSWT/SATWT5)*100;  GASTPER=(GASTRWT/SATWT5)*100;
TIBIAPER=(TIBIAWT/SATWT5)*100;  FEMURPER=(FEMURWT/SATWT5)*100;
FLENPER=(FEMURLE/SATWT5)*100;   TLENPER=(TIBIALE/SATWT5)*100;
IF ID=35 THEN DELETE;
CARDS;
%ADD DEBJATA2
PROC GLM; CLASSES EOD EXER;
MODEL SATWT1 SATWT7 TOTGAIN GAINDAY CARCASS
   CARCFAT PERFAT CARCLEM LBM
   FEEDDAY FDWTDAY FEEDEFF ABDOMFAT ABDOMPER RETROFAT RETROPER
   EPIDFAT EPIDPER LIVERWT LIVERPER LIVERD LIVDNA LIVERP LIVPRO
   LIVERR KIDNEYWT KIDPER KIDNEYD KIDDNA KIDNEYP KIDPRO KIDNEYR
   BRAINWT BRAINPER BRAIND BRNDNA BRAINP BRNPRO BRAINR
   VASTUSWT VASTPER VASTUSD VASTDNA VASTUSP VASTPRO VASTUSR
   GASTRWT GASTPER GASTROD GASTDNA GASTRUCP GASTPRO GASTRUCR
   TIBIAWT TIBIAPER FEMURWT FEMURPER FEMURLE FLENPER TIBIALE
   TLENPER FEMURDS TIBIADS FEMURDG TIBIADG=EOD EXER EOD*EXER;

LSMEANS EOD EXER EOD*EXER/STDERR;
/*
```

APPENDIX 6

Effects of exercise following normal or undernutrition on weight gain

in growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Beginning wt, g	75	250	303	70	201	207
	71	296	310	68	182	220
	73	333	331	62	200	218
	67	299	340	77	220	206
	77	321	303	76	202	200
	70	270	330	70	213	187
	68	310	290	66	203	188
	69	291	316	76	208	211
	77	285				
	72±2	295±6	315±6	70±2	204±6	205±6
Ending wt, g	283	582	531	172	546	440
	268	546	497	188	478	497
	338	632	560	148	497	447
	305	611	541	229	500	425
	254	571	465	207	558	456
	306	490	510	206	562	390
	282	548	488	177	522	454
	301	485	534	189	502	467
	339	473				
	297±8	549±13	516±14	196±9	521±14	447±14
Total wt gain, g	208	332	228	102	345	233
	197	250	187	120	296	277
	265	299	229	86	297	229
	238	312	201	152	280	217
	177	250	162	131	356	256
	236	220	180	136	349	203
	214	238	198	111	319	266
	232	194	218	113	294	256
	262	188				
	225±8	254±12	200±12	126±9	317±12	242±12
Ave. daily wt gain, g	7.4	5.9	4.1	3.6	6.2	4.2
	7.0	4.5	3.4	4.3	5.3	4.9
	9.5	5.3	4.1	3.1	5.3	4.1
	8.5	5.6	3.6	5.4	5.0	3.9
	6.3	4.5	2.9	4.7	6.4	4.6
	8.4	3.9	3.2	4.9	6.2	3.6
	7.6	4.2	3.5	4.0	5.7	4.8
	8.3	3.5	3.9	4.0	5.2	4.6
	9.4	3.4				
	8.0±0.3	4.5±0.2	3.6±0.2	4.5±0.3	5.7±0.2	4.3±0.2

APPENDIX 7

Effects of exercise following normal or undernutrition on carcass composition

in growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Carcass wt, g	242	508	454	131	478	378
	224	481	428	148	404	430
	288	554	486	110	423	378
	257	535	464	175	429	355
	214	502	391	165	488	389
	257	410	432	145	482	329
	243	478	412	138	453	382
	264	411	460	161	424	399
	287	407				
	253 ±7	476 ±13	441 ±14	152 ±7	448 ±14	380 ±14
Carcass fat, g	41	111	58	15	78	45
	35	107	68	24	52	70
	55	121	60	22	67	35
	42	122	67	17	80	20
	25	114	42	28	97	41
	55	80	36	16	84	41
	42	97	54	20	95	29
	46	54	57	24	81	65
	65	74				
	45 ±3	98 ±6	55 ±6	21 ±3	79 ±6	43 ±6
Carcass fat, %	17	22	13	12	16	12
	16	22	16	16	13	16
	19	22	12	15	16	9
	16	23	14	12	19	6
	12	23	11	17	20	10
	21	19	8	11	18	12
	17	20	13	15	21	8
	18	13	12	15	19	16
	23	18				
	18 ±1	20 ±1	13 ±1	14 ±1	18 ±1	11 ±1
Carcass lean mass, g	201	397	396	116	399	332
	189	374	360	123	352	360
	233	433	426	93	355	343
	215	414	397	153	349	334
	189	388	369	137	391	348
	201	330	396	129	398	288
	201	381	358	118	358	352
	218	357	403	137	343	334
	222	332				
	208 ±5	378 ±9	385 ±10	131 ±5	368 ±10	337 ±10
Carcass lean mass, %	83	78	87	88	84	88
	84	78	84	84	87	84
	81	78	88	85	84	91
	84	77	86	88	81	94
	88	77	89	83	80	90
	78	81	92	89	82	88
	83	80	87	85	79	92
	82	87	88	85	81	84
	77	82				
	82 ±1	80 ±1	87 ±1	86 ±1	82 ±1	89 ±1

APPENDIX 8

Effects of exercise following normal or undernutrition on feed intake of growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Ave. daily feed intake						
Total g/day	18.7	25.1	21.9	9.4	25.7	21.4
	16.8	25.9	20.5	10.6	20.4	24.1
	21.9	28.5	23.8	11.9	23.1	20.1
	20.6	27.1	24.9	11.0	23.8	19.8
	17.5	24.9	20.0	11.5	25.4	21.1
	20.6	22.0	20.4	11.1	26.8	18.2
	19.0	25.8	22.2	10.0	24.9	20.5
	20.2	22.0	21.7	10.1	23.7	22.1
	22.8	22.5				
	19.8 ±0.5	24.9 ±0.6	21.9 ±0.7	10.7 ±0.5	24.2 ±0.7	20.9 ±0.7
Ave. daily feed intake						
per 100 g body wt	13.0	6.1	5.4	8.3	7.2	7.0
	12.6	6.2	5.2	9.4	6.8	6.7
	13.9	6.1	5.3	8.7	7.0	6.3
	14.5	6.1	5.8	10.6	7.0	6.4
	12.6	5.7	5.3	9.1	7.1	6.7
	14.3	6.2	5.1	9.6	7.4	6.6
	13.8	6.0	5.8	9.6	7.4	6.7
	14.2	5.7	5.3	8.3	6.9	7.0
	14.0	6.1				
	13.7 ±0.2	6.0 ±0.1	5.4 ±0.1	9.1 ±0.3	7.1 ±0.1	6.7 ±0.1
Feed efficiency	0.40	0.24	0.19	0.39	0.24	0.19
g total gain	0.42	0.17	0.16	0.40	0.26	0.20
/g total feed	0.43	0.19	0.17	0.46	0.23	0.20
	0.41	0.20	0.14	0.46	0.21	0.20
	0.36	0.18	0.14	0.41	0.25	0.22
	0.41	0.18	0.16	0.44	0.23	0.20
	0.40	0.16	0.16	0.40	0.23	0.23
	0.41	0.16	0.18	0.40	0.22	0.21
	0.41	0.15				
	0.41 ±0.01	0.18 ±0.01	0.16 ±0.01	0.42 ±0.01	0.23 ±0.01	0.21 ±0.01

APPENDIX 9

Effects of exercise following normal or undernutrition on adipose tissue

of growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Epididymal fat	2.6	12.4	7.7	1.5	11.2	5.9
Wt, g	3.9	10.1	6.4	1.8	7.4	6.8
	4.5	10.8	8.7	2.0	8.3	6.9
	3.8	15.8	6.9	1.4	8.9	3.5
	2.7	12.2	4.8	2.0	13.2	6.0
	4.5	8.0	6.0	1.2	10.5	5.3
	3.4	12.0	7.2	1.4	11.7	5.1
	3.5	7.8	8.5	1.7	6.7	8.6
	3.9	11.1				
	3.6 ±0.2	11.1 ±0.7	7.0 ±0.7	1.8 ±0.2	9.7 ±0.7	6.0 ±0.7
Epididymal fat	0.9	2.6	1.8	0.9	2.5	1.6
Wt, g/100 g body wt	1.4	2.2	1.5	1.0	1.9	1.6
	1.3	2.0	1.8	0.9	2.1	1.9
	1.2	3.1	1.5	0.7	2.2	1.0
	1.0	2.5	1.2	1.0	3.0	1.6
	1.5	2.0	1.4	0.6	2.3	1.6
	1.2	2.5	1.7	0.8	2.8	1.4
	1.2	1.8	1.8	0.9	1.7	2.3
	1.2	2.6				
	1.2 ±0.1	2.4 ±0.1	1.6 ±0.1	0.8 ±0.1	2.3 ±0.1	1.6 ±0.1
Retroperitoneal fat	4.8	22.8	11.9	0.9	14.3	8.6
Wt, g	3.1	24.6	12.6	1.9	10.9	13.8
	6.8	28.4	15.7	2.3	16.6	8.4
	5.9	27.1	15.8	0.9	14.6	4.4
	3.8	21.6	11.0	2.1	20.8	8.1
	7.0	17.8	9.3	1.8	18.0	10.0
	5.4	20.1	12.2	1.1	18.9	5.9
	6.6	14.6	14.4	2.0	18.5	15.6
	8.8	19.7				
	5.8 ±0.4	21.8 ±1.2	12.9 ±1.2	1.6 ±0.5	16.6 ±1.2	9.3 ±1.2
Retroperitoneal fat	1.7	4.7	2.7	0.5	3.2	2.3
Wt, g/100 g body wt	1.2	5.3	2.9	1.0	2.9	3.3
	2.0	5.4	3.2	1.0	4.1	2.3
	1.9	5.3	3.5	0.4	3.6	1.2
	1.5	4.5	2.7	1.0	4.7	2.1
	2.3	4.4	2.2	0.9	4.0	3.0
	1.9	4.2	2.8	0.6	4.4	1.6
	2.2	3.4	3.2	1.0	4.7	4.0
	2.6	4.7				
	1.9 ±0.1	4.7 ±0.2	2.9 ±0.2	0.8 ±0.1	4.0 ±0.2	2.5 ±0.2
Abdominal fat	3.7	13.7	9.1	1.9	14.6	6.4
Wt, g	3.6	11.9	8.2	2.0	7.3	10.0
	5.6	16.5	9.9	1.9	13.2	6.7
	3.9	18.4	9.0	2.1	11.7	5.3
	3.2	15.6	6.0	2.6	15.6	7.0
	6.2	9.2	5.5	2.0	13.6	6.1
	2.6	14.5	10.5	2.2	13.6	5.8
	4.1	9.3	8.3	2.2	9.9	9.0
	5.4	15.0				
	4.2 ±0.3	13.8 ±0.8	8.3 ±0.9	2.1 ±0.3	12.4 ±0.9	7.0 ±0.9
Abdominal fat	1.3	2.8	9.1	1.1	3.3	1.7
Wt, g/100 g body wt	1.4	2.6	8.2	1.1	1.9	2.4
	1.7	3.2	9.9	0.8	3.3	1.8
	1.3	3.6	9.0	1.1	2.9	1.5
	1.2	3.2	6.0	1.3	3.5	1.8
	2.0	2.2	5.5	0.9	3.0	1.8
	0.9	3.0	10.5	1.2	3.2	1.6
	1.4	2.2	8.3	1.2	2.5	2.4
	1.6	3.6				
	1.4 ±0.1	2.9 ±0.1	1.9 ±0.2	1.1 ±0.1	3.0 ±0.2	1.9 ±0.2

APPENDIX 10

Effects of exercise following normal or undernutrition on tibia measurements

of growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Tibia						
Wt, mg	393	594	565	260	580	488
	368	606	540	261	496	592
	401	620	608	315	507	494
	404	633	626	316	554	522
	390	624	566	302	587	501
	397	447	602	273	632	517
	362	531	567	283	522	525
	414	516	618	282	572	520
	370	503				
	389±7	564±16	586±17	286±7	556±17	520±17
Length, mg	36	40	41	33	41	39
	36	41	40	32	39	41
	37	42	42	36	40	39
	37	40	40	34	40	39
	35	41	39	34	41	41
	35	38	40	33	40	39
	37	40	40	33	39	40
	36	41	41	33	39	39
	37	39				
	36±0	40±0	40±0	33±0	40±0	40±0
Specific gravity	1.57	1.56	1.52	1.52	1.58	1.53
	1.61	1.54	1.65	1.46	1.60	1.55
	1.58	1.71	1.55	1.39	1.69	1.61
	1.44	1.57	1.58	1.40	1.60	1.63
	1.49	1.56	1.54	1.45	1.54	1.65
	1.52	1.60	1.59	1.44	1.54	1.65
	1.51	1.66	1.56	1.31	1.58	1.60
	1.51	1.83	1.61	1.49	1.52	1.57
	1.50	1.73				
	1.53±0.02	1.64±0.02	1.57±0.02	1.43±0.02	1.58±0.02	1.60±0.02
Breaking strength, kg	2.68	3.68	2.52	1.32	3.20	3.00
	2.60	1.24	2.52	2.44	3.88	4.64
	2.04	4.80	4.32	2.56	1.20	2.04
	2.08	3.88	2.08	1.88	3.28	3.40
	1.80	3.60	4.56	2.28	2.40	2.88
	1.92	1.76	1.96	2.80	4.44	3.36
	2.80	1.80	3.24	3.32	7.00	2.96
	1.48	4.28	3.72	2.68	2.48	3.00
	2.12	3.04				
	2.17±0.17	3.12±0.41	3.12±0.44	2.41±0.18	3.48±0.44	3.16±0.44

APPENDIX 11

Effects of exercise following normal or undernutrition on femur measurements

of growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Femur						
Wt, mg	414	818	840	293	827	679
	399	879	769	282	753	818
	446	881	899	331	866	686
	393	866	839	330	786	709
	410	891	810	330	829	730
	434	703	874	307	876	742
	419	790	812	291	745	760
	418	800	888	307	811	742
	421	733				
	417±6	818±18	841±19	309±6	812±19	735±19
Length, mm	31	38	39	27	38	37
	30	38	37	27	38	38
	32	40	40	30	38	36
	31	38	39	29	37	37
	31	39	38	28	38	38
	30	36	39	28	38	37
	32	38	38	28	37	38
	31	39	39	28	37	36
	32	36				
	31±0	38±0	39±0	28±0	38±0	37±0
Specific gravity	1.53	1.50	1.53	1.50	1.53	1.46
	1.53	1.53	1.49	1.44	1.50	1.46
	1.69	1.52	1.59	1.39	1.41	1.58
	1.54	1.53	1.48	1.61	1.52	1.58
	1.54	1.47	1.56	1.40	1.59	1.53
	1.50	1.48	1.56	1.38	1.49	1.49
	1.55	1.65	1.46	1.39	1.57	1.55
	1.61	1.57	1.52	1.57	1.53	1.48
	1.62	1.58				
	1.57±0.02	1.53±0.02	1.52±0.02	1.46±0.02	1.52±0.02	1.52±0.02
Breaking strength, kg	1.24	2.60	1.32	2.56	4.04	2.40
	----	3.32	2.44	2.68	2.40	2.88
	1.88	2.92	4.80	2.72	2.76	3.76
	1.32	2.80	2.04	2.04	4.32	1.68
	3.20	2.92	2.08	1.36	4.24	1.88
	1.36	2.16	3.76	1.00	2.68	2.16
	2.36	2.40	2.76	1.24	1.44	3.40
	1.80	1.88	2.88	2.16	2.72	3.64
	2.36	2.24				
	1.94±0.24	2.58±0.29	2.76±0.31	1.97±0.24	3.08±0.31	2.72±0.31

APPENDIX 12

Effects of exercise following normal or undernutrition on protein and nucleic acid contents in livers of growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Liver	16.10	31.66	25.51	9.96	24.76	20.54
Wt, g	15.27	26.21	22.81	11.30	22.56	25.66
	21.31	29.50	23.86	11.63	22.71	17.29
	17.92	30.07	26.05	12.68	21.10	13.22
	14.28	21.18	19.95	12.78	22.85	16.62
	19.14	18.43	22.27	11.08	26.93	16.01
	16.28	22.72	20.34	10.53	19.22	17.42
	16.74	21.88	22.27	11.52	13.94	18.07
	20.75	21.49				
	17.53±0.63	24.79±1.26	22.88±1.33	11.44±0.67	21.76±1.33	18.10±1.33
Wt, g/100 g body wt	5.69	6.60	5.81	5.79	5.63	5.51
	5.70	5.65	5.34	6.01	5.97	6.11
	6.30	5.63	4.87	5.08	5.66	4.72
	5.88	5.92	5.72	6.40	5.20	3.69
	5.62	4.41	4.94	6.17	5.15	4.37
	6.25	4.53	5.16	5.38	5.97	4.90
	5.77	4.77	4.75	5.95	4.54	4.94
	5.56	5.12	4.87	6.10	3.52	4.74
	6.12	5.12				
	5.88 ±0.12	5.31 ±0.23	5.18 ±0.24	5.86 ±0.13	5.20 ±0.24	4.86 ±0.24
DNA, total mg	36.71	75.03	55.36	20.02	62.64	41.90
	-----	58.44	53.60	-----	50.99	52.60
	48.59	71.68	57.02	24.42	35.20	40.80
	35.48	48.71	34.12	27.77	34.60	31.20
	25.27	35.79	34.71	23.00	42.50	29.91
	35.02	36.31	47.21	16.40	55.21	28.33
	31.42	39.76	43.73	14.53	53.24	41.11
	37.16	44.63	49.88	22.00	37.08	39.93
	36.73	48.35				
	35.80±2.02	51.00±3.64	46.96±3.87	21.16±2.16	46.43±3.87	38.20±3.87
Protein, total g	2.61	4.82	3.76	1.49	3.83	3.28
	----	4.52	3.25	----	3.20	3.90
	3.15	4.27	3.63	1.65	3.04	2.55
	2.68	4.50	3.69	1.81	2.72	2.00
	2.12	3.00	2.95	1.71	2.66	2.65
	2.47	2.69	3.34	1.65	3.50	2.37
	2.22	3.41	3.16	1.34	2.58	2.75
	2.25	3.34	3.46	1.57	2.41	2.57
	3.12	3.55				
	2.58 ±0.11	3.79 ±0.19	3.40 ±0.20	1.60 ±0.12	2.99 ±0.20	2.76 ±0.20
Protein/DNA, mg/mg	71.18	64.22	67.83	74.48	61.15	78.33
	-----	77.30	60.68	-----	62.88	74.24
	64.78	59.63	63.68	67.67	86.52	62.58
	75.61	92.41	108.09	65.25	78.66	64.07
	84.01	83.79	86.88	74.50	62.58	88.50
	70.49	74.21	70.85	100.47	63.41	83.73
	70.78	85.83	72.18	92.39	48.41	66.86
	60.63	74.85	69.28	71.52	65.00	64.30
	84.86	73.47				
	72.8 ±3.9	76.4 ±4.0	74.7 ±4.2	78.0 ±4.1	66.1 ±4.2	72.8 ±4.2

APPENDIX 13

Effects of exercise following normal or undernutrition on protein and nucleic acid contents in kidneys of growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Kidneys (both)	2.52	3.71	3.67	1.81	3.72	3.68
Wt, g	2.69	3.39	3.78	1.68	3.32	3.58
	2.90	3.63	4.03	2.04	3.22	3.20
	2.58	4.02	3.80	2.00	3.45	2.88
	2.41	3.83	3.11	1.94	3.62	3.48
	2.80	3.24	3.54	1.72	3.79	2.81
	2.75	2.66	3.77	1.78	3.14	3.66
	2.63	3.69	3.95	1.87	2.75	3.10
	2.71	3.28				
	2.66 ±0.05	3.49 ±0.12	3.71 ±0.12	1.86 ±0.05	3.38 ±0.12	3.30 ±0.12
Wt, g/100 g body wt	0.89	0.77	0.84	1.05	0.84	0.99
	1.00	0.73	0.88	0.89	0.88	0.85
	0.86	0.69	0.82	0.89	0.80	0.87
	0.84	0.79	0.83	1.01	0.84	0.80
	0.95	0.80	0.77	0.93	0.81	0.92
	0.92	0.80	0.81	0.83	0.84	0.86
	0.98	0.56	0.88	1.00	0.74	1.01
	0.87	0.86	0.86	0.98	0.69	0.81
	0.80	0.78				
	0.90 ±0.02	0.75 ±0.02	0.84 ±0.02	0.95 ±0.02	0.81 ±0.02	0.89 ±0.02
DNA, total mg	8.22	12.28	12.96	6.50	13.21	11.33
	9.01	11.83	14.51	3.67	10.16	12.85
	10.96	14.12	14.43	5.47	10.30	11.65
	6.76	13.02	11.67	9.34	12.14	10.60
	7.50	12.26	9.33	6.21	12.67	10.61
	7.64	10.21	11.86	6.11	13.03	8.73
	9.52	11.46	11.95	7.10	10.96	10.98
	7.04	11.29	12.60	----	9.21	10.23
	6.23	11.94				
	8.10 ±0.47	12.05 ±0.46	12.41 ±0.49	6.73 ±0.53	11.46 ±0.49	10.87 ±0.49
Protein, total mg	410.5	641.1	648.5	283.8	620.5	578.5
	391.7	546.1	630.5	254.0	534.8	597.1
	461.1	591.0	696.4	272.3	531.3	521.0
	370.0	569.6	607.2	290.4	532.0	498.0
	362.5	619.9	513.2	269.1	530.0	552.0
	377.4	469.2	568.9	242.2	609.0	429.6
	387.2	453.8	651.4	264.5	542.6	625.9
	353.5	529.1	616.2	-----	434.8	490.1
	384.0	596.3				
	388.6±8.8	557.2±20.2	616.5±21.4	268.3±10.0	541.9±21.4	536.5±21.4
Protein/DNA, mg/mg	50.0	52.2	50.0	43.7	47.0	51.0
	43.5	46.2	43.4	39.9	52.6	46.5
	42.1	41.8	48.3	49.8	51.6	44.7
	54.7	43.7	52.0	31.1	43.8	47.0
	48.4	50.5	55.0	43.3	41.8	52.0
	49.4	46.0	48.0	39.7	46.7	49.1
	40.7	39.6	54.5	37.2	49.5	57.0
	50.1	46.9	48.9	----	47.2	47.9
	61.6	50.5				
	48.9 ±2.1	46.3 ±1.3	50.0 ±1.4	40.7 ±2.4	47.5 ±1.4	49.4 ±1.4

APPENDIX 14

Effects of exercise following normal or undernutrition on protein and nucleic acid contents in brains of growing male rats

Measurement	CONTROL (Baseline)	CONTROL - Exer	CONTROL + Exer	FASTED ECD (Baseline)	FASTED ECD - Exer	FASTED ECD + Exer
Brain						
Wt, g	1.90	2.08	3.67	1.72	2.16	1.78
	2.05	2.18	3.78	1.73	2.20	1.91
	1.89	2.24	4.03	1.85	2.05	2.20
	1.97	2.26	3.80	1.79	2.17	2.11
	1.94	2.20	3.11	1.82	2.09	2.16
	2.02	2.30	3.54	1.89	2.39	2.00
	2.07	2.23	3.77	1.86	2.13	2.24
	2.01	2.21	3.95	1.83	2.13	2.08
	1.93	2.00				
	1.98±0.02	2.19±0.04	2.25±0.04	1.81±0.02	2.16±0.04	2.06±0.04
Wt, g/100 g body wt	0.67	0.43	0.53	1.00	0.49	0.48
	0.76	0.47	0.55	0.92	0.58	0.45
	0.56	0.43	0.45	0.80	0.51	0.60
	0.64	0.44	0.49	0.90	0.53	0.59
	0.76	0.45	0.51	0.87	0.47	0.57
	0.66	0.56	0.54	0.91	0.53	0.61
	0.73	0.46	0.53	1.05	0.50	0.62
	0.67	0.51	0.48	0.97	0.54	0.54
	0.57	0.47				
	0.67±0.03	0.47±0.01	0.51±0.02	0.93±0.03	0.52±0.02	0.56±0.02
DNA, total mg	2.72	2.37	2.88	3.03	2.74	2.70
	3.42	3.75	3.18	2.96	2.97	3.36
	2.65	3.14	3.40	2.42	2.87	2.88
	2.78	3.16	2.83	2.76	2.76	2.68
	3.32	3.19	3.00	2.71	3.20	2.74
	2.91	3.13	3.03	3.33	3.42	2.86
	3.46	2.92	3.13	3.39	3.04	2.98
	3.06	2.93	3.18	3.06	3.26	3.18
	3.22	2.98				
	3.05±0.11	3.06±0.09	3.08±0.09	2.96±0.11	3.03±0.09	2.92±0.09
Protein, total mg	197.6	211.1	234.8	180.9	207.8	194.9
	210.1	223.4	243.4	182.5	220.0	204.0
	186.7	232.5	219.3	178.0	215.2	233.6
	194.6	234.6	234.2	174.5	208.8	216.3
	189.2	231.0	208.0	177.4	211.5	226.8
	194.3	238.7	229.1	191.3	233.0	213.0
	199.1	237.5	227.0	167.0	207.7	215.5
	198.6	206.6	215.5	168.7	204.9	202.8
	178.5	195.0				
	194.3±2.8	223.4±4.2	226.4±4.4	178.0±3.0	213.6±4.4	213.4±4.4
Protein/DNA, mg/mg	72.7	89.0	81.6	59.8	75.7	72.0
	61.4	59.6	76.5	61.7	74.1	60.7
	70.6	74.1	64.6	73.4	75.0	81.1
	72.6	74.1	82.7	63.3	75.7	80.7
	57.0	72.4	69.4	65.4	66.1	82.7
	66.8	76.3	75.6	57.5	68.2	74.5
	57.6	81.3	72.5	49.3	68.2	72.3
	65.0	70.3	67.7	55.2	62.9	63.7
	55.4	65.4				
	64.3±2.3	73.6±2.4	73.8±2.5	60.7±2.5	70.7±2.5	73.5±2.5

APPENDIX 15

Effects of exercise following normal or undernutrition on protein and nucleic acid contents in gastrocnemius muscles of growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Gastrocnemius muscle						
Wt, g	2.9	5.6	5.6	2.1	5.8	5.1
	2.9	6.1	5.8	2.1	5.4	6.4
	3.6	6.0	6.5	2.7	5.8	5.4
	3.2	6.0	6.2	2.0	5.7	5.2
	2.9	5.5	6.1	2.5	6.6	5.4
	3.0	5.5	5.6	2.4	5.9	4.9
	3.3	5.7	5.1	1.8	5.3	5.1
	3.0	5.1	6.0	2.4	5.5	5.4
	3.2	4.8				
	3.11±0.10	5.59±0.14	5.86±0.15	2.25±0.10	5.75±0.15	5.36±0.15
Wt, g/100 g body wt	1.02	1.17	1.28	1.22	1.32	1.37
	1.08	1.31	1.36	1.12	1.43	1.52
	1.06	1.14	1.33	1.18	1.45	1.48
	1.05	1.18	1.36	1.01	1.40	1.45
	1.14	1.14	1.51	1.21	1.49	1.42
	0.98	1.35	1.30	1.16	1.31	1.50
	1.17	1.20	1.91	1.01	1.25	1.42
	1.00	1.19	1.31	1.27	1.39	1.42
	0.94	1.14				
	1.05±0.03	1.20±0.03	1.33±0.03	1.15±0.03	1.38±0.03	1.45±0.03
DNA, total mg	1.16	1.29	1.40	0.88	1.86	1.17
	1.04	1.16	1.74	0.76	1.94	1.66
	1.69	1.98	1.88	1.16	1.68	2.11
	1.38	2.04	1.49	1.02	2.00	1.56
	1.25	1.60	2.01	1.02	2.24	1.78
	1.05	1.65	2.07	0.94	1.53	1.72
	1.62	1.77	1.38	0.68	1.54	1.43
	1.20	2.09	1.68	0.94	1.54	1.84
	1.34	1.39				
	1.30±0.07	1.66±0.10	1.71±0.10	0.92±0.87	1.79±0.10	1.66±0.10
Protein, total mg	553.0	935.2	863.5	330.5	790.5	868.0
	523.4	1057.7	857.2	350.7	673.9	1027.8
	470.2	798.6	736.4	321.3	798.1	718.7
	446.4	802.8	773.8	272.4	766.1	948.5
	464.0	897.6	901.6	440.0	869.9	850.0
	518.4	950.4	881.4	368.6	1065.0	818.3
	487.7	824.2	806.3	312.1	895.7	894.5
	501.0	894.5	910.2	445.4	795.3	801.9
	571.5	758.9				
	504.0±17.0	880.0±31.2	841.3±33.1	355.0±18.1	831.8±33.1	866.0±33.1
Protein/DNA, mg/mg	476.8	726.1	616.8	374.8	425.9	740.0
	501.4	912.6	492.7	463.9	346.7	617.7
	277.9	403.3	390.7	276.7	474.5	341.3
	324.4	393.5	520.0	267.1	384.0	608.0
	372.1	562.8	447.9	429.3	387.6	477.0
	493.7	576.0	425.4	393.8	694.2	477.1
	301.6	466.4	585.6	456.3	582.8	626.4
	417.5	427.8	541.8	475.9	516.4	436.8
	425.2	545.2				
	399.0±27.7	557.1±43.2	502.6±45.8	392.2±29.4	476.5±45.8	540.5±45.8

APPENDIX 16

Effects of exercise following normal or undernutrition on protein and nucleic acid contents in vastus muscles of growing male rats

	CONTROL			FASTED ECD		
Measurement	(Baseline)	- Exer	+ Exer	(Baseline)	- Exer	+ Exer
Vastus muscle						
Wt, g	3.1	3.1	3.2	2.0	3.5	3.5
	2.9	3.3	3.7	2.0	3.3	3.6
	3.5	3.5	4.1	2.6	2.9	3.3
	2.9	4.1	3.1	2.0	3.3	3.2
	2.9	3.3	4.3	2.3	3.4	3.0
	2.9	3.1	2.9	2.3	4.1	2.8
	2.9	3.8	3.1	1.9	3.2	3.1
	3.0	3.0	3.5	2.1	3.2	3.2
	3.3	2.9				
	3.04±0.08	3.34±0.13	3.49±0.14	2.15±0.08	3.36±0.14	3.21±0.14
Wt, g/100 g body wt	2.00	0.64	0.73	1.16	0.80	0.94
	1.08	0.71	0.87	1.06	0.87	0.86
	1.04	0.67	0.84	1.14	0.72	0.90
	0.95	0.81	0.70	1.01	0.81	0.89
	1.14	0.69	1.06	1.11	0.76	0.79
	0.95	0.76	0.67	1.12	0.91	0.86
	1.03	0.80	0.72	1.07	0.76	0.86
	1.00	0.70	0.76	1.11	0.81	0.84
	0.97	0.69				
	1.03±0.02	0.72±0.03	0.79±0.03	1.10±0.02	0.81±0.03	0.87±0.03
DNA, total mg	1.18	0.59	1.02	0.88	1.30	0.80
	1.22	0.82	1.04	0.68	1.85	0.83
	1.68	1.30	1.35	1.20	0.81	2.05
	1.28	0.70	0.81	0.54	0.96	0.86
	0.96	0.89	1.20	0.74	1.16	0.87
	1.07	1.12	1.39	0.90	0.78	0.64
	1.19	1.03	0.83	0.76	0.90	0.81
	0.78	0.78	1.16	0.52	1.76	0.96
	1.09	0.67				
	1.16±0.08	0.88±0.11	1.10±0.12	0.78±0.08	1.19±0.12	0.98±0.12
Protein, total mg	523.9	504.1	628.8	367.4	672.0	548.8
	508.7	576.5	670.1	326.4	544.8	608.4
	636.3	613.9	719.1	490.9	580.9	578.8
	536.5	635.1	650.7	410.8	595.6	610.2
	534.5	629.3	663.1	431.2	602.8	572.1
	553.0	620.9	608.7	406.2	658.5	496.4
	484.3	622.4	626.8	336.9	688.0	616.9
	501.0	625.8	755.0	424.6	636.8	626.6
	665.3	586.4				
	549.3±19.4	601.6±15.3	665.3±16.3	399.3±20.6	622.4±16.3	582.3±16.3
Protein/DNA, mg/mg	444.7	855.8	614.1	417.5	518.9	681.7
	417.6	698.8	646.8	480.0	294.8	734.8
	378.8	474.0	531.5	410.4	715.4	282.9
	420.4	911.2	807.3	760.7	622.4	706.3
	558.5	706.3	550.7	585.9	521.5	657.6
	515.4	556.4	437.3	452.8	845.3	770.9
	407.3	606.7	748.9	443.2	767.8	765.4
	642.3	802.3	653.6	808.8	361.8	652.7
	610.9	879.1				
	488.4±43.0	721.2±52.6	623.8±55.8	544.9±45.6	581.0±55.8	656.5±55.8

THE EFFECTS OF EXERCISE ON GROWTH OF RATS RECOVERING FROM EARLY UNDERNUTRITION

by

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AN ABSTRACT OF A THESIS

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ABSTRACT

The effects of an 8-wk exercise program were studied on growth of rats following a period of normal nutrition or undernutrition. Male Wistar weanling rats, underfed by fasting on alternate days for 4-wk, gained 44% less weight than ad libitum fed rats (carcass: 53% less fat; 37% less lean mass), and had markedly smaller fat depots, visceral organs, brains, and hindlimb muscles and bones. Rats in both groups were then allowed free access to feed and subdivided into exercise or non-exercise groups. Eight wk of treadmill exercise (speed, 24 m/min; duration, 75 min/d; frequency, 5 d/wk) reduced weight gain in both normal-fed and underfed rats, but had a greater effect on underfed rats. Underfed rats that were exercised gained 24% less weight (carcass: 46% less fat; 8% less lean mass), and tended to have smaller tissues than underfed rats that were not exercised. Data suggest that exercise following a postweaning period of undernutrition (between 4-8 wks of age) reduces "catch-up" growth in rats.