

Changes in Body Fat and Lipogenic Enzyme Activities in Rats after Termination of Exercise Training (39764)

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Introduction. During a recent investigation, we observed that trained rats gained weight at a remarkable rate after termination of training. Since training resulted in the loss of body fat (1), it was of interest to determine whether lipid deposition was enhanced after the termination of training (detraining). Thus, the present study was conducted to investigate alterations in body fat content during a 2-week detraining period. The activities of several lipogenic enzymes have also been measured to determine whether these enzymes play a role in the deposition of fat after termination of training.

Methods and materials. Male Holtzman rats (Holtzman, Madison, Wis.) weighing approximately 200 g at the start of the experiment were housed in individual cages and given water and commercial lab chow (Wayne Lab Blox, Allied Mills, Inc., Chicago, Ill.) *ad libitum*. Rats were divided into two groups: untrained, which remained sedentary in their cages; and trained, which were subjected to daily treadmill running at 35 m/min, 1 hr/day, 6 days/week for 6 weeks as previously described (2). Daily exercise of the trained rats was terminated after 6 weeks and the trained rats were allowed to remain sedentary in their cages during the detraining period. Food consumption and body weights were determined during the training and detraining phases of the experiment. Body fat content was measured by an established procedure (3).

After 2 weeks of detraining, six trained-detrained and six untrained rats were sacrificed by decapitation and the liver and epididymal fat pads were quickly excised and suspended in ice-cold 1.15% KCl. To assay for lipogenic enzymes, livers were homoge-

nized in a glass-Teflon Potter-Elvehjem homogenizer in 0.05 M potassium phosphate buffer, pH 7.4, containing 1 mM EDTA and 1 mM dithiothreitol; adipose tissue was homogenized in a glass-glass homogenizer in the same buffer. The particle-free supernatant solution was prepared by centrifuging the homogenates at 105,000g for 60 min followed by filtration through four layers of cheesecloth. Lipogenic enzymes in the particle-free supernatant solutions were assayed spectrophotometrically. Fatty acid synthetase was assayed according to the procedure of Plate *et al.* (4), citrate cleavage enzyme (EC 4.1.3.8) was assayed as described by Takeda *et al.* (5), malic enzyme (EC 1.1.1.40) was assayed according to Hsu and Lardy (6), and glucose-6-phosphate dehydrogenase (EC 1.1.1.49) was assayed according to the method of Langdon (7). All assays were performed under conditions of substrate saturation with the amount of enzyme as the rate-limiting component of the *in vitro* reaction. Protein concentrations of liver and adipose preparations were determined by the biuret (8) and Lowry *et al.* methods, (9), respectively.

The capacity of liver and adipose homogenates to synthesize glycerides was estimated by a modification of the method of Askew *et al.* (10). Adipose tissue was homogenized in 5 vol of 10 mM potassium phosphate buffer, pH 7.4, containing 1 mM EDTA and livers were homogenized in 5 vol of a solution containing 50 mM Tris-HCl, pH 7.5, 100 mM KCl, 5 mM MgSO₄, and 1 mM EDTA. The assay conditions were as described by Askew *et al.* (10) and the final reaction volume was 0.8 ml. All reactions were terminated by the addition of 3.0 ml of chloroform:methanol (1:2) and the lipids were extracted by the Bligh and

Dyer method (11). An aliquot (0.5 ml) of the lower phase was withdrawn, evaporated to dryness, and counted in a liquid scintillation counter (Beckman LS 233).

Results and Discussion. The effects of training and detraining on body weight are shown in Fig. 1. Training decreased body weight (compared to untrained controls), particularly during the last 2 weeks of training. This is reflected in the lower weight gain during the sixth week of training (Table I). However, as can be seen in Fig. 1, after training was terminated, there was a dramatic increase in body weight. The daily weight gain of the trained-detrained rats (Table I) was greater than the weight gain

for the untrained controls despite the fact that food consumption was similar for the two groups.

The increase in the ratio of weight gain to food consumed suggests a greater energy efficiency in trained-detrained rats than in control rats. Other investigators have shown that the feed efficiency of an animal is influenced by the nutritive value of the feed (12) as well as the level of feed intake and meal pattern (nibbling versus meal eating) (13). In addition, the genetic character of the animal also can influence the feed efficiency as has been demonstrated in obese Zucker rats (14). Although the factors affecting feed efficiency have been investigated, little is known about the mechanisms involved (13). We had hypothesized that the trained-detrained rats might be absorbing more food from the gut than the controls, thus deriving more nutritive value from an equal portion of food. However, analysis of protein, carbohydrate, and lipid in the feces demonstrated that absorption was not different in the trained-detrained rats than in the control rats (unpublished observation). Thus, further studies will be required to establish the mechanism that allows the trained-detrained rats to gain more weight than control rats while consuming equal amounts of food.

In an attempt to determine whether the weight gain was due to an increase in lipid deposition, we analyzed the body fat content of the trained and untrained rats at the end of training and 2 weeks after the termination of training. Body fat content of the trained rats increased from 6.8 to 8.4% dur-

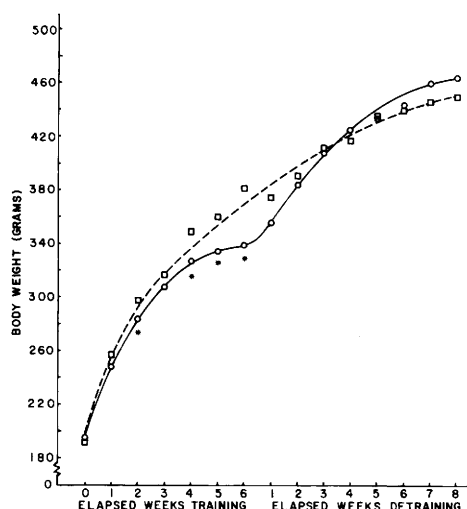


FIG. 1. Effect of exercise training on body weight. □---□, untrained rats, ○—○, trained-detrained. (*) indicates a significant ($P < 0.05$) difference between trained and untrained rats.

TABLE I. EFFECTS OF TRAINING AND DETRAINING ON BODY WEIGHT, FOOD CONSUMPTION, EPIDIDYMAL FAT PAD WEIGHTS, AND CARCASS FAT CONTENT.

	Training		Detraining	
	Untrained	Trained	Untrained	Trained-De-trained
Body weight (g)	406 ± 5 ^a	350 ± 6*	431 ± 6	397 ± 5*
Daily weight gain (g/day)	2.93 ± .26 ^b	1.84 ± .24 ^{b*}	1.98 ± .13 ^c	3.48 ± .33 ^{c*}
Daily food consumption (g/day)	24.4 ± .2 ^d	22.2 ± .2 ^{d*}	23.8 ± .5 ^e	24.2 ± .5 ^e
Fat pad weight (g)	5.70 ± .37	3.10 ± .11*	5.54 ± .21	4.37 ± .21*
Carcass fat content (%)	11.1 ± .6	6.8 ± .2*	12.0 ± .5	8.4 ± .3*

^a Values are mean ± SEM for at least six observations.

^b Daily weight gain during the sixth week of training.

^c Average daily weight gain over the 2-week detraining period.

^d Average daily food consumption for the 6-week training period.

^e Average daily food consumption for the 2-week detraining period.

* Significant difference ($P < 0.05$) between untrained and trained.

TABLE II. EFFECTS OF TRAINING AND DETRAINING ON ENZYMES INVOLVED IN LIPID SYNTHESIS IN LIVER AND ADIPOSE.

	Enzyme activity (nmole/min/mg of protein)			
	Adipose		Liver	
	Untrained	Detained	Untrained	Detained
Fatty acid synthetase	5.0 ± .6 ^a	12.1 ± 2.4*	5.0 ± .4	6.3 ± .5*
Glucose-6-phosphate dehydrogenase	43.8 ± 2.0	66.0 ± 8.1*	23.3 ± 1.9	27.8 ± 3.6
Citrate cleavage enzyme	5.8 ± .8	17.6 ± 3.5*	12.1 ± .8	14.9 ± 1.2
Malic enzyme	6.2 ± .6	23.6 ± 6.6*	17.1 ± 2.3	19.6 ± 2.4
Glyceride synthesis	15.7 ± 1.0	18.8 ± 1.1	3.99 ± 2.6	3.94 ± .17

^a Values are mean ± SEM for at least six observations.* Statistically significant ($P < 0.05$) difference between trained-detained and untrained.

ing the 2 weeks of detraining.

To establish whether the increase in carcass fat could have been the result of increased activities of the lipogenic enzymes, we have measured the activity of several enzymes involved in the synthesis of fatty acids and glycerides. We previously reported that training decreased the activities of hepatic fatty acid synthetase (15) and adipose tissue glucose-6-phosphate dehydrogenase (16) and increased the capacity for glyceride synthesis in adipose tissue (10, 16). The results of the present experiment were in agreement with previous findings and thus the data taken at the end of training have not been reproduced here. After 2 weeks of detraining, there was a general increase in lipogenic enzyme activities in the trained-detained rats, compared to controls, with statistically significant differences found for liver and adipose fatty acid synthetase, adipose glucose-6-phosphate dehydrogenase, adipose citrate cleavage enzyme, and adipose malic enzyme. Thus, it seems clear that an increase in the activities of lipogenic enzymes in both liver and adipose may play a role in the rapid deposition of fat after the termination of exercise training.

Summary. Exercise training lowered the body fat content of rats but, after training was terminated, there was a rapid increase in body weight and carcass fat. After 2 weeks of detraining, there were increases in the activities of liver and adipose tissue fatty acid synthetase, adipose glucose-6-phosphate dehydrogenase, adipose citrate cleavage enzyme, and adipose malic enzyme. The increase in the activities of lipogenic enzymes may play a role in the rapid deposition of fat after termination of exercise training.

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