The Effects of Prior Exercise on Myocardial Glycogenesis During a Fast[1] (35437)

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(Introduced by E. C. Hoff)

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Previous reports have suggested that a prolonged exercise program produces changes in cardiac metabolism which lead to greater myocardial glycogenesis in trained animals during a fast than in fasted, control animals (2, 5, 7). Our experiments were undertaken to determine if repetitive exercise or merely a single bout of exercise causes this enhanced glycogeneis observed in the trained, fasted rat.

Methods and Materials. Male rats of the Wistar strain were used throughout these experiments. Both trained and control rats were taken from the same population and were allowed free access to food and water. Rats were trained by forcing them to run twice daily for approximately 30 days on a motor driven treadmill at a speed of 1 mile/hr. The running time was gradually increased from 15 min to 1 hr so that during the final 10 days the rats were running for two 1-hr periods each day.

Immediately following the last exercising period in the training program the trained rats were started on a 48 hr fast along with control animals. During the fast, the rats were allowed free access to water but were deprived of all food. Following the fasting period each rat was given pentobarbital and 5 min were allowed for the anesthetic to take effect. The heart of each rat was rapidly excised by opening the thorax, exposing the heart, and with a single cut freeing it from the body. The atria and large vessels were cut away and discarded. The ventricles were blotted and transferred to a preweighed tube containing 30% KOH. The hearts of control nonfasted rats were similarly excised. The time between freeing the heart from its circulatory supply and putting it in the preweighed tube was less than 10 sec. The weight of the tissue sample was determined by the difference in the weight of the tube before and after the muscle was added.

To compare the effect of prolonged training and a single exercising period, a second group of experiments was performed. One hr of swimming was used as the form of exhaustive exercise in these experiments involving only a single exercise period. These rats were sacrificed at 0, 1, or 6 hr after the swimming period and their cardiac glycogen levels were compared with those of control rats. Other rats were fasted for 48 hr with the fast being initiated at 0, 1, or 6 hr after the swimming period. Control rats were also fasted for 48 hr.

Total glycogen was extracted according to the general procedure of Good *et al.* (3), and quantitatively determined by the anthrone method of Seifter *et al.* (6), using a Beckman Model B spectrophotometer at a wavelength of 620 mμ (4).

Glycogen values were reported in milligrams of glycogen per 100 g of wet tissue weight. Statistical analysis of the data was done by applying Student's t test or analysis of variance and Duncan's multiple range test.

Results. Table I gives the results of experiments comparing control and trained rats. The training regime was severe enough that the exercising rats gained less body weight than the control animals, resulting in a significantly lower body weight in the trained population ($p < .05$). There was no indication of the development of cardiac hypertrophy during training since the heart weights and the heart weight/body weight

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TABLE I. Body Weights, Heart Weights, Heart Weight/Body Weight Ratios, and Myocardial Glycogen Concentrations in: Nonfasted Control Rats; Fasted Control Rats; and Fasted Trained Rats.

Values represent mean and the standard error of the mean. Glycogen values are in milligrams per 100 g of wet tissue weight.

	Wt		$\frac{HW}{BW} \times 10^3$	Myocardial glycogen (mg/100 ml)
	Body (g)	Heart (mg)		
Control				
nonfasted	381 ± 10	927.9 ± 16.7	2.44 ± 0.06	313.8 ± 23.4
fasted	360 ± 10	877.6 ± 25.0	2.45 ± 0.05	549.6 ± 29.4[c]
Trained				
fasted	324 ± 7[a]	833.5 ± 17.0[b]	2.47 ± 0.12	722.2 ± 27.2[d]

[a] $p < 0.05$, trained vs control fasted or control nonfasted.
[b] $p < 0.01$, trained vs control nonfasted.
[c] $p < 0.01$, control fasted vs control nonfasted.
[d] $p < 0.01$, trained vs control fasted or control nonfasted.

ratios were not significantly elevated. The lower heart weight in the trained rats compared with the control nonfasted rats is due to the combined effects of the training program and the 48-hr fast. Fasting produced a significant increase in cardiac glycogen in both trained and control animals. However, myocardial glycogen was significantly higher in the fasted, trained rats than in the fasted, control rats ($p<.01$).

Figure 1 shows results of experiments involving only a single bout of exercise. Shown by the lower line in Fig. 1 are the cardiac glycogen concentrations before and at various times following 1 hr of swimming. Myocardial glycogen levels decreased during the swimming period and returned to near control levels by 1 hr following the exercise. However, the myocardial glycogen concentrations continued to rise and were significantly higher than control levels 6 hr following the exercising period.

Figure 1 also shows the effects of a 48-hr fast on cardiac glycogen concentrations. The fasting was begun in exercised rats at 0, 1, or 6 hr following exercise. The cardiac glycogen levels were significantly greater in all groups of exercised, fasted rats than in the control, fasted rats ($p<.05$) or all groups of nonfasted rats ($p<.01$).

Discussion. Our results concur with those previously reported (2, 5, 7) that myocardial glycogenesis during a fast is greater in trained rats than in untrained rats. The critical factor in producing the enhanced glycogenesis in the trained rats, however, appears to be merely the last bout of exercise in the training program rather than the process of repetitive exercise. This is indicated by the fact that acute exercise in nontrained rats

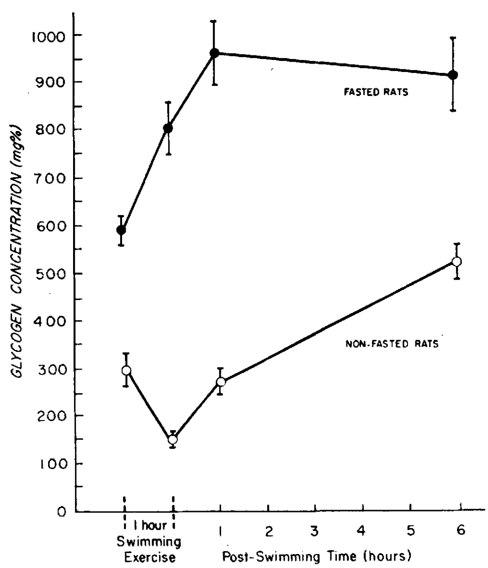

FIG. 1. Myocardial glycogen concentrations in nonfasted rats before and at 0, 1, and 6 hr following 1 hr of swimming and in 48-hr fasted rats with the fasting period beginning before or 0, 1, or 6 hr following 1 hr of swimming. Given are the mean values ± the standard error of the mean. Each point represents the results for 10 rats.

will produce a similarly enhanced glycogenesis during a fast.

Our results indicate that the effects of acute exercise are of relatively long duration. The data supporting this include the fact that cardiac glycogen is significantly elevated as long as 6 hr following exercise. Also, a fast begun 0, 1, or even 6 hr following acute exercise produces higher glycogen levels than that observed in fasted, control rats. Blount *et al.* (1) reported a similar pattern of glycogen utilization and synthesis following 15 min of swimming. With their shorter period of exercise, however, elevated glycogen levels occurred within 1 hr following the exercise. It has yet to be determined whether the extent of utilization and speed of recovery of myocardial glycogen following a single bout of exercise differs between trained and untrained animals.

Summary. The enhanced myocardial glycogenesis observed in trained rats can no longer be attributed to the training process but appears to be due merely to the last bout of exercise in the training program.

The pattern of changes in cardiac glycogen during and following exercise involves more than simply depletion and recovery. It involves an additional period of relatively long duration during which the glycogen is significantly greater than control levels.

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