Myocardial Glycogen Changes with Exercise[1] (36588)

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The changes in cardiac glycogen concentration associated with single bouts of exercise involve more than simply a depletion during exercise and a return to control levels following exercise. They involve an additional period of relatively long duration during which the glycogen is significantly greater than control values (1). If trained or untrained rats are fasted shortly after a bout of exercise, the cardiac glycogenesis observed is greater than that observed when the rats are not exercised prior to a fast (1–3). Elevated myocardial glycogen levels have been reported in rats following 5 weeks of repetitive exercise (4), and these high glycogen stores could be a factor in the heart's ability for greater mechanical performance during periods of hypoxia (5).

These investigations suggest that single and repetitive bouts of exercise may change the heart's metabolic handling of its stored glycogen. The purpose of the present study was to compare the pattern of glycogen changes during and following single bouts of exercise in trained and untrained rats.

Methods and Materials. Male Wistar strain rats weighing between 150 and 250 g when purchased were used throughout these experiments. They were housed in individual cages and allowed free access to food and water. Some rats were trained by forcing them to run twice daily for 4 weeks on a motor driven treadmill at a speed of 1 mph. The running time was gradually increased to 1 hr so that during the final 2 weeks the rats were running for two 1-hr periods each day with a 4-hr rest period between exercising sessions.

At the end of 4 weeks of running, part of the trained rats along with control rats underwent a single bout of exercise during which they were forced to swim for 1 hr in a large sink filled with water at 25 $\pm$ 1°. Some rats were sacrificed either before or at 0, 1, 2, 4, or 8 hr after the swim and others were fasted for 48 hr with the fast beginning either before or at 0, 1, 2, 4, or 8 hr after the swimming. Fasting rats received water only.

All rats were sacrificed by injecting Nembutal and quickly excising the heart as soon as the rat was asleep. The hearts were trimmed of vessels and atria, blotted, and placed into tubes containing 30% KOH. The time between freeing the heart from its blood supply and placing it into the KOH was less than 10 sec. Total glycogen was extracted according to the general procedure of Good, Kramer and Somogyi (6) and quantitatively determined by the anthrone method of Seifter *et al.* (7) using a Beckman Model B spectrophotometer at a wave length of 620 mμ (8). Glycogen values were recorded in milligrams of glycogen per 100 g of wet tissue weight. Statistical analysis of the data was done by applying the Student's t test.

Results. Often we have seen an increase in heart weight–body weight ratio following the training program employed, but in this particular population there was no significant change. However, the trained rats did have a significantly lower body weight following the training program.

Figure 1 compares the myocardial glycogen concentrations in trained and control rats before and at various times following 1 hr of swimming. In both trained and control rats the glycogen levels were significantly lower

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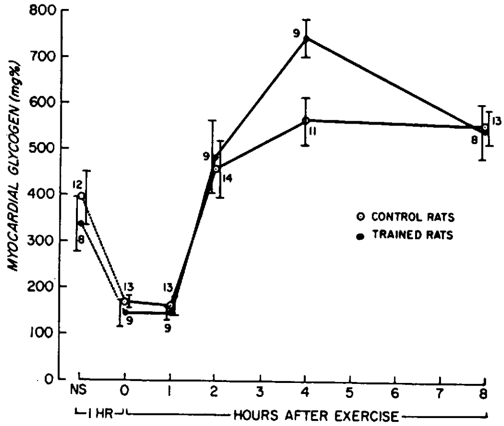

FIG. 1. Myocardial glycogen before and at various times following 1 hr of swimming in trained and control rats. Plotted are the mean values ± the standard error of the mean. Also shown is the number of rats associated with each point.

immediately after the exercise and continued to be significantly lower 1 hr later. After 2 hr the glycogen levels had returned to control values but continued to rise and were significantly elevated in trained and control rats at 4 and 8 hr following the exercise. Thus the extent of glycogen decrease during exercise and the pattern of glycogen recovery following exercise were the same for trained and control rats. However, the maximum glycogen concentration reached 4 hr after swimming was significantly higher ($p < .05$) in the trained rats.

Figure 2 compares the glycogen concentrations in cardiac and skeletal (gastrocnemius) muscles before and at various times following 1 hr of swimming. The data indicates that skeletal muscle glycogen, in contrast to cardiac glycogen, does not reach elevated levels during the recovery period being studied but merely returns to the control value.

Figure 3 gives the myocardial glycogen concentrations in trained and control (sedentary) rats following a 48-hr fast. The fast was begun before or at the indicated times following 1 hr of swimming. Fasting in trained and control rats not having previously swum produced significant glycogenesis in cardiac muscle (no change in skeletal muscle glycogen levels was observed). In control rats the extent of glycogenesis was even greater if the fast was begun 0, 1, or 2 hr after 1 hr of swimming. Similarly in trained rats the glycogenesis was significantly enhanced if the fast was begun 1 or 2 hr following the swimming. Thus the single bout of exercise affected the extent of glycogenesis that occurred over the following 48-hr fast in trained and control animals.

Discussion. These results concur with those previously reporting that the pattern of changes in cardiac glycogen during and following exercise involves more than simply depletion and recovery. It includes an additional period of relatively long duration in the course of which the glycogen is significantly greater than control levels (1). These studies also show the extent of glycogenolysis during exercise as well as the pattern of glycogen recovery following exercise to be similar in trained and untrained rats. However, the maximum glycogen level reached during recovery was significantly higher in the trained rats. This would indicate that training does alter to some extent the manner in which the heart handles its endogenous glycogen.

Lamb *et al.* (9) did not observe a supercompensation of myocardial glycogen in guinea pigs following exercise probably because they chose to measure glycogen levels

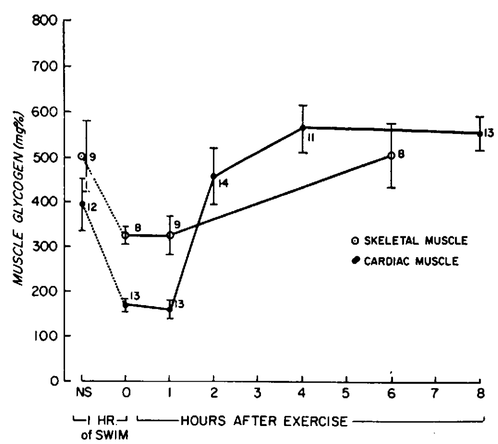

FIG. 2. Glycogen levels in skeletal and cardiac muscles before and at various times following 1 hr of swimming. Plotted are the mean values ± the standard error of the mean. Also given is the number of rats associated with each point.

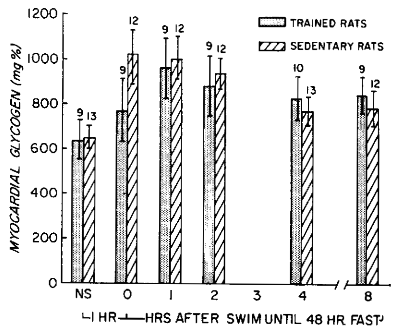

FIG. 3. Myocardial glycogen in trained and control (sedentary) rats following a 48-hr fast which was begun before or at varius times following 1 hr of swimming. Given are the mean values ± the standard error of the mean and the number of rats associated with each bar.

only at zero and 48 hr after the exercise by which time the cardiac glycogen might have returned to control values. They did, however, observe a supercompensation of skeletal muscle glycogen at this time. In contrast, our studies using rats showed a supercompensation of cardiac glycogen which reached a peak 4 hr after exercise but no supercompensation of skeletal muscle glycogen during this same period. These results suggest that a supercompensation of glycogen following exercise probably occurs in both types of muscle but much more slowly in skeletal muscle.

The results reported in this paper are in agreement with those previously reporting that myocardial glycogenesis during a fast is enhanced in both trained and control rats if the fast is begun shortly after a bout of exercise (1). The elevated glycogen levels in fasted, trained rats reported by several investigators (1–3) is probably due to the last bout of exercise rather than the program of repetitive exercise. Scheuer *et al.* (4), however, found a difference in cardiac glycogen between fasted trained and fasted control rats which occurred only after 5 weeks of training. If a single bout of exercise in the training program were responsible for this difference, it should have been manifested from the very beginning of the training program. The full significance of their work, however, would depend on how much time was allowed to elapse between the last bout of exercise and the beginning of the fast, since the length of this period will affect the extent of fasting myocardial glycogenesis. Their results appear to show effects of training which are not produced by a single bout of exercise and which are manifested only after a long training regime.

Summary. The extent of glycogenesis during exercise and the pattern of glycogen recovery following exercise is similar in trained and untrained rats. However, the maximum value reached during recovery is significantly higher in the trained group. A supercompensation of skeletal muscle glycogen was not observed during this same period. The degree of glycogenesis during a fast is the same in trained and untrained rats and in both groups is enhanced if the fast is begun following a bout of exercise.

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