A single training session affects exercise-induced muscle damage in the rat

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Exercise can cause transient damage to skeletal muscle, as has been demonstrated by numerous studies in humans (Ebbeling and Clarkson 1989) and in animals (after eccentric exercise: Armstrong et al. 1983, after concentric exercise: Amelink and Bär 1986). Damage is evidenced by elevations of serum enzymes such as CK as well as by histological signs of muscle damage. In humans this is often accompanied by muscle soreness and impaired muscle function (Bär et al. 1990).

A single bout of exercise can protect the muscle against damage caused by a subsequent work. This rapid adaptation is especially notable in humans after eccentric exercise (Ebbeling and Clarkson 1989). We describe the effect of one bout of exercise studied in a level (concentric) running rat model, on damage caused by a second, identical exercise session. Plasma CK activity was used as a marker for muscle damage. As we have shown before, most of the rise of the CK activity after exercise in this model is indeed derived from muscle (Amelink et al. 1988).

Experimental Methods

In the experiment 13 male Wistar rats (215 g ± 9) were used. A jugular vein cannula, reaching into the hart, was implanted one week before exercise. The rats were subjected to a 2 h level treadmill run at 19 m/min. After the first run the rats were randomly divided into group A (7 rats) and group B (6 rats). Both group A and group B rats were subjected to a second, identical, bout of exercise: group A after 1 day and group B after 6 days. The CK activity in serum was measured before and immediately after exercise.

Statistics

Because of the cannulation repeated blood sampling is possible, so that animals serve as their own controls. For statistical analysis of the data within one session the paired version of the Student's t-test was used, for comparison of data between sessions the unpaired t-test was used.

Results

Figure 1 shows the effect of one bout of exercise on muscle damage caused during a second exercise session 1 day or 6 days later. After the first bout the serum CK activity had increased significantly from 57 ± 13 to 123 ± 27 U/l (mean ± standard deviation, $p < 0.01$).

After 24 h, the resting CK activity (group A) was significantly lower (38 ± 5 vs 57 ± 13, $p < 0.01$). The increase in CK activity during the second exercise, however, was significantly higher than after the first bout (156 ± 40 and 123 ± 27 U/l resp, $p < 0.05$).

When the second exercise was performed 6 days after the first run (group B), the picture was completely different. Firstly, the CK resting values were not significantly different (54 ± 11 vs 57 ± 13 U/l). Secondly, the increase in CK activity after the second bout is much lower than the increase after the first run (76 ± 30 and 123 ± 27 U/l resp, $p < 0.01$).

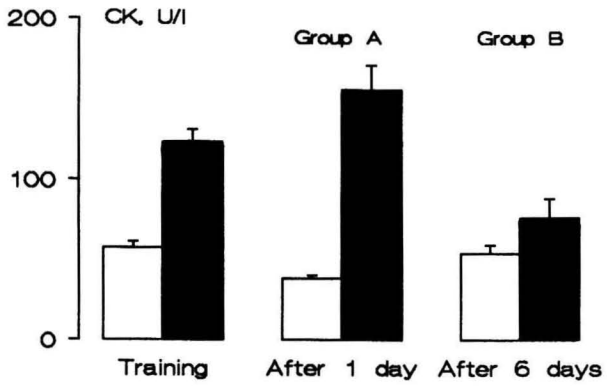

Figure 1. Plasma CK activity before (open bars) and immediately after (closed bars) a 2 h training run in 13 male rats. In 7 rats the exercise was repeated after 1 day (group A), in the remaining 6 rats the exercise was repeated after 6 days (group B).

Discussion

Based on the observation of a lower CK response after exercise in Group B there seems to be a protective effect of a single bout of exercise in rats that run for 2 h on a treadmill. This protection becomes apparent 6 days later. This is in keeping with Armstrong et al. (1983) who found a protective effect of a single bout of exercise when the exercise was repeated 3 days later and CK was measured 48 h after the second exercise. Thus, there is evidence for a rapid adaptation to exercise-induced muscle damage in rats after a single bout of exercise.

However, the fact that repeating the exercise regimen 24 h later resulted in an increased CK response points to an increased susceptibility to exercise-induced muscle damage within the first 24 h after exercise, before a protective effect of the first bout is established. Apparently it takes at least 24 h for the rat muscles to adapt. It is not known what entails the rapid adaptation to exercise-induced muscle damage. Rapid changes in passive resistance to stretch of the muscle (membrane) by reinforcement of the cytoskeleton, which is important in exercise-induced muscle damage (Waterman-Storer 1991) or the extracellular matrix (Stauber et al. 1990) could be responsible, as well as other e.g. metabolic factors.

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