Heat Shock Protein 27 (HSP27) is found to translocate and accumulate on cytoskeletal structures during eccentric exercise (1). Moreover, in the days after exercise, the content of HSP70 seems to be up-regulated (2). However, the function of the exercise-induced stress protein response is not fully understood and in this study we investigate the stress protein response to two bouts of eccentric exercise. The stress protein response was studied in conjunction with expression of the regeneration marker CD56.

Twenty-two young and healthy males and females performed two bouts of 70 unilateral maximal eccentric actions with their arm flexors; the other (randomly chosen) arm served as control. Muscle biopsies were obtained one, 48, 96 and 168 hrs after bout 1 and one and 48 hrs after bout 2 that was performed three weeks after bout 1. Changes in isometric force-generating capacity were monitored for nine days after both bouts. Cross-sections of muscle specimens were immunohistochemically stained for HSP27, HSP70 and CD56.

The force-generating capacity was reduced immediately after bout 1 and 2 by 51±3 and 42±2%, respectively. After bout 1 muscle function did not recover within nine days, whereas after bout 2 the subjects recovered within three days. The number of HSP27 positive fibers peaked one hr after both bout 1 and 2, constituting 41±10 and 20±8% of counted myofibers, respectively (mean±SEM; p<0.05 compared to control). At 48 hrs after bout 1, 28±6% of the counted fibers were still HSP27 positive (p<0.05), whereas no increase in the number of HSP27 positive fibres could be detected at this time after bout 2. The number of HSP70 positive fibers was increased at 48, 96 and 168 hrs after bout 1, with a peak value of 27±5% of counted fibers at 48 hrs (p<0.05). After bout 2 the number of HSP70 positive fibers was 10±3% at 48 hrs, which was lower than after bout 1 (p<0.05). At 168 hrs after bout 1 and 48 hrs after bout 2, 19±11 and 11±3% of the counted fibers were CD56 positive, respectively (p<0.05).

The HSP27 staining showed a granular, myofibrillar stain; and the acute reduction in force-generating capacity correlated with the number of HSP27 stained fibers one hr after bout 1 (r=0.7; p<0.05). Especially at 48 hrs, HSP70 co-localized with HSP27; at 96 and 168 hrs HSP70 showed a more even, cytoplasmatic stain. At 168 hrs after bout 1 and at both time points after bout 2, CD56 strongly stained small myofibers with central nuclei. These apparently regenerated fibers – as a consequence of the necrosis after bout 1 – demonstrated also increased cytoplasmatic immunoreactivity against HSP27, but not HSP70.

It appears that HSP27 accumulate to disrupted myofibrils during eccentric exercise. In addition, HSP27 seems to be associated with late regeneration of myofibers, whereas HSP70 may be involved in the recovery and adaptation of surviving myofibers after eccentric exercise.

2. Paulsen et al. Submitted. AmJP.

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