【国際セッション6】

Time-course and differential expression of heat shock proteins in human skeletal muscle following non-damaging treadmill exercise: is heat a mechanism of activation?J.P. Morton^{*}, D.P. MacLaren, A. McArdle[†] and B. Drust^{*}^{*}Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool, L3 2ET, UK and [†]School of Clinical Sciences, University of Liverpool, Liverpool, L69 3GA, UK

The exercise-induced expression of heat shock proteins (HSPs) as a cellular stress response in rodent models is relatively well defined (McArdle *et al.*, 2001). Comparable data from human studies, however, are limited and the mechanisms contributing to the exercise-induced production of HSPs are far from understood. Although exercise-associated hyperthermia is routinely cited as a possible signal responsible for inducing an increased production of HSPs following exercise, this hypothesis has not been formally tested in human skeletal muscle. The aim of the present study was to therefore characterise the time-course of response of the major HSP families in human skeletal muscle following a running exercise protocol and to test the hypothesis that increased muscle and core temperature mediate the increased production of HSPs following exercise.

Eight healthy active males performed 45 min of running exercise at a treadmill speed corresponding to their lactate threshold. Muscle biopsies were obtained from the vastus lateralis muscle immediately prior to and at 24 h, 48 h, 72 h and 7 days post-exercise. Seven healthy active males also underwent a passive heating protocol of 1 h duration in which one leg was immersed in a tank containing hot water (45 °C). The contra-lateral limb remained outside the tank and was not exposed to heat stress thus serving as a control leg. Muscle biopsies were obtained from the vastus lateralis of both legs immediately prior to and at the time of peak HSP expression induced by exercise (observed to be at 48 h and 7 days post-heating). The exercise and heating protocol induced significant increases ($P < 0.05$) in rectal (1.7 ± 0.3 and 1.5 ± 0.2 °C, respectively) and muscle temperature (3.8 ± 0.8 and 3.7 ± 0.6 °C, respectively). Muscle temperature of the control leg showed no significant change following heating (pre: 36.1 ± 0.5 , post: 35.6 ± 0.4 °C). Exercise induced a significant ($P < 0.05$) but variable increase in HSP70, HSC70 and HSP60 expression with peak increases (typically occurring at 48 h post-exercise) of $210 \pm 70\%$, $170 \pm 75\%$ and $139 \pm 23\%$ of pre-exercise levels, respectively. In contrast, heating failed to induce a significant increase in muscle content of these proteins. Neither heating nor exercise induced a significant increase in HSP27, α B-crystallin, MnSOD protein content or the activity of superoxide dismutase and catalase.

This study is the first in which the time-course and extent of HSP production in human skeletal muscle have been characterised following a moderately demanding running exercise protocol. Data demonstrate that the skeletal muscle of healthy active young males adapts to such stresses via an increased production of several HSPs (predominantly HSP70) where peak responses typically occur at 48 h post-exercise. We conclude that increases in both systemic and local temperature *per se* appear not to be mediating this response.

McArdle, A. et al. (2001). *Am J Cell Physiol*, 280: C621-C627.