

Skeletal muscle heat shock protein response to lower limb unloading and reloading in humans

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Skeletal muscle atrophy is a consequence of ageing, many chronic diseases and a sedentary lifestyle. Skeletal muscle disuse often accompanies each of these conditions and is likely to contribute to at least some of the muscle atrophy. Growing evidence suggests that heat shock proteins (HSPs) may be involved in the regulation of muscle mass during disuse, as they have been shown to inhibit apoptosis, activate satellite cells, attenuate unloading-induced muscle atrophy, and are responsive to changes in muscle loading in rodents. Nonetheless, no studies have investigated the HSP response to altered muscle loading in humans. We, therefore, investigated the effects of unilateral lower limb suspension (ULLS, unloading) and subsequent strength training (reloading) on human skeletal muscle HSP protein expression.

Vastus lateralis muscle biopsy samples were collected from the unloaded leg prior to (Pre) and immediately after 23 days of ULLS (Unload) and following 4 weeks of resistance training (Reload). A DXA scan was also performed 1 day prior to each biopsy to assess changes in leg lean mass. Skeletal muscle HSP72 and HSP27 expression was assessed by Western blotting and immunofluorescence.

Unloading caused left thigh mass to decrease in all participants, however, the mean decrease of $5.4 \pm 4.5\%$ was not statistically significant ($p = 0.061$). Reloading caused left thigh mass to increase $5.7 \pm 1.9\%$ ($P = 0.001$) compared to Unload. Western blotting revealed that HSP72 expression increased $60.2 \pm 48.2\%$ ($P = 0.025$) from Pre to Unload and was no longer different from Pre following reloading ($P = 0.296$; see Figure). These results were confirmed by immunofluorescence. HSP27 expression was unchanged following unloading and reloading.

These data provide evidence that human skeletal muscle HSP72 expression is responsive to changes in muscle loading and may therefore be a viable target for the prevention of muscle atrophy.

