Heat Shock Proteins in Exercise: A Review

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Abstract

This review has attempted to highlight some of the most recent and original studies involving the stress response. It is clear that, especially regarding *in vivo* human studies, there is a paucity of research regarding the SPR stimulus, it's signalling system, measurement concepts, chronic adaptations, and it's practical use in both the health and elite sporting performance fields. In addition, the differences in exercise intensity, mode duration, subjects, training status etc. contribute to disparity in findings from previous studies.

Key Words: **% Body Fat, Flexibility, Cardiovascular Endurance, VO_2 max**

Introduction

Contemporary exercise physiology research has termed changes in the homeostatic balance within the whole organism as stress. Stress is defined as 'the process by which environmental events threaten or challenge the organism's well being and by which that organism responds to this threat' *(Turner, 1994)*. Stressors of any nature act initially at the cellular level and are manifested as protein damage or impairment, which compromises the function and integrity of the cell. Without intervention, stressors would develop throughout cell lines and eventually affect tissues, organs and perhaps in time, the whole organism. However, a set of highly conserved proteins have been shown to accumulate at such instances and have thus been termed stress proteins (SP). It has been postulated that expression of these proteins convey a response initiated to maintain cellular homeostasis after exposure to stress. The response has been denoted in all types of cells investigated to date, and although the precise function of these proteins remains unclear, their expression in the cell allows it to survive normally lethal stresses.

The most commonly cited SP is the heat shock protein (HSP), so called because the original stressor that elicited the SP response was hyperthermia *(Ritossa, 1962)*. Yet, to date, investigators have been unable to directly ascertain the stimulus and mechanism by which SP are synthesised, and further, their role is asserting stasis. Nonetheless, HSP's have been associated with a number of cytoprotective functions, including the protection of stable proteins *(Dobson and Ellis, 1998)*, chaperoning and folding of nascent polypeptides *(Beckman et al., 1990)*, and degradation of aggregated proteins *(Chiang et al., 1989)*.

A number of conditions, including hyperthermia, ischemia, pH alterations, energy depletion, calcium variations, abnormal protein generation and oxidative phosphorylation uncoupling are known to induce the cellular stress protein response (SPR). The majority of these stressors that are met with the SPR accompany exercise and therefore exercise physiologists have experienced difficulties in drawing firm