PROTECTIVE MECHANISMS IN EXERCISE-INDUCED HYPERTERMIA

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The capacity for both prolonged and high-intensity short-term exercise is diminished in hot environmental relative to normothermic conditions. Furthermore, during submaximal exercise in hot conditions, there is an increase in both thermal and cardiovascular strain. Although it is clear that the increased physiological strain relates to less efficient avenues of heat loss, the precise mechanisms that contribute to an earlier onset of fatigue in the heat, remain unclear. Possible candidates include:- 1) central nervous inhibition via increased serotonergic/dopaminergic activity due to the attainment of a critical core temperature; 2) altered cardiovascular control resulting in changes in blood pressure regulation, secondary to peripheral blood volume displacement and dehydration, and 3) depletion of metabolic substrates due to increased reliance on muscle glycogenolysis. Despite this uncertainty, it is evident that strategies can be employed to delay the onset of fatigue and protect the individual from heat injury. These primarily relate to acclimatization processes to both the exercise stress and environmental condition resulting in cardiovascular and thermal adaptations ultimately delay the attainment of a critical core temperature and the onset of fatigue. Whilst the cardiovascular and thermoregulatory mechanisms responsible for these beneficial adaptations are understood, the underlying cellular mechanisms are less well defined, although the role of heat shock proteins, at a skeletal muscle level at least, have been ruled out. These adaptive responses are important in not only allowing performance to continue but also in decreasing the risk of adverse events.

This is of particular importance in populations where heat loss is compromised, for example, the elderly.