EDUCATION

Logical limitations to the "catastrophe" models of fatigue during exercise in humans

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A central debate in the exercise sciences is the cause of the fatigue that develops especially during high intensity exercise of short duration. The most popular theory holds that this form of exercise is limited by a peripherally based, metabolite induced failure of skeletal muscle contractile function, independent of reduced muscle activation by the central nervous system; so-called peripheral fatigue. This theory arose originally from studies undertaken by Nobel Laureate Sir Archibald Vivian Hill and colleagues in Manchester, UK in the 1920s. In turn, their interpretations were crucially influenced by the earlier 1907 findings of Sir Frederick Gowland Hopkins, Nobel Laureate for his discovery of the vitamins, and Walter Morley Fletcher. The original model of Hill and his colleagues proposed that performance during exercise of high intensity was limited by skeletal muscle anaerobiosis that developed as the result of a limiting skeletal muscle blood flow, following the onset of myocardial ischaemia. Such skeletal muscle anaerobiosis ultimately prevented the neutralization of the lactic acid that, Hill believed, initiated muscle contraction. The resulting lactic acid accumulation impaired skeletal muscle relaxation, causing the (involuntary) termination of exercise. The evolutionary progression of this model led to the "catastrophe theory" of Richard Edwards, which posits that exercise terminates when the physiological and biochemical limits of the body are exceeded, causing a catastrophic failure of intracellular homeostasis.

This paper addresses six hallmark physiological requirements that must be correct if Hill's cardiovascular/ anaerobic/catastrophic model is the exclusive explanation for the fatigue that develops during maximum exercise to exhaustion. This leads to a review of the evidence supporting other, related "catastrophe" models that have been developed to explain fatigue during exercise of lower intensities and longer durations.

It is concluded that there is little published evidence supporting the theory that fatigue occurs only after physiological homeostasis fails according to the prediction of these catastrophe models. Rather, it is proposed that fatigue in any form of exercise may form part of a regulated, anticipatory response co-ordinated in the subconscious brain. The ultimate goal of this regulation is to preserve homeostasis in all physiological systems during exercise, regardless of intensity or duration or the environmental conditions in which it is undertaken.

A popular teaching in exercise physiology is that fatigue during most forms of exercise is due to a peripherally based, metabolite induced failure of skeletal muscle contractile function (peripheral fatigue)[1–6], independent of reduced skeletal muscle activation by efferent output from the motor cortex of the central nervous system (CNS); so-called central fatigue.

HISTORICAL DEVELOPMENT OF THIS MODEL OF PERIPHERAL FATIGUE

The origins of this belief can be traced to the pioneering studies of Fletcher and Hopkins[1 2] and Hill[3 7–11] and colleagues[12–15] in the 1920s. The classical theory, since defined as the cardiovascular/anaerobic/catastrophic model of exercise physiology,[16 17] postulates that fatigue during high intensity exercise of short duration results from a skeletal muscle "anaerobiosis" (see Addendum) that develops when the oxygen requirement of the active skeletal muscles exceeds the heart's capacity to further augment oxygen delivery to exercising muscle by increasing the cardiac output. As a

Abbreviations: ATP, adenosine triphosphate; CNS, central nervous system; EMG, electromyographic; MR, magnetic resonance; MVC, maximal voluntary contraction NIRS, near infrared spectroscopy; PCr, phosphocreatine; RPE, rating of perceived exertion

What is already known

Beginnning with the foundation studies of the British Nobel Laureates A V Hill and F G Hopkins in the early 1900s, the A V Hill cardiovascular/anaerobic/ catastrophic model of exercise physiology has evolved to dominate teaching and research in the exercise sciences. This model posits that exercise is regulated by metabolic changes in the peripheral muscles, independent of any regulation by the central nervous system (CNS).

What this study shows

This review shows that the published literature does not support the six hallmark predictions of the Hill model of exercise physiology. In particular, there is no evidence that skeletal muscle recruitment is ever total during voluntary exercise to exhaustion in humans. The presence of skeletal muscle recruitment reserve at fatigue proves that exercise performance is regulated by the CNS, specifically to ensure that a catastrophic failure of homeostasis does not occur during voluntary exercise in humans.

result, any additional increase in energy generation in the active muscles can come only from ''anaerobic'' metabolism leading to fatigue, as the ''maximum oxygen intake is inadequate, lactic acid accumulating, a continuously increasing oxygen debt being incurred, fatigue and exhaustion setting in''.[12]

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