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 skeletal muscle recruitment reserve at fatigue proves that exercise physiology. In particular, there is no evidence that exercise performance is regulated by metabolic changes in the peripheral muscles, independent of any regulation by the central nervous system (CNS).

**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\(^7\)\(^8\)\(^9\) and colleagues\(^1\)\(^2\)\(^3\)\(^4\)\(^5\)\(^6\)\(^7\)\(^8\)\(^9\)\(^10\)\(^11\) in the 1920s. The classical theory, since defined as the cardiovascular/anaerobic/catastrophic model of exercise physiology,\(^14\)\(^15\) 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 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 coordinated 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.

**What is already known**

Beginning 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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