From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans

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It is a popular belief that exercise performance is limited by metabolic changes in the exercising muscles, so called peripheral fatigue. Exercise terminates when there is a catastrophic failure of homeostasis in the exercising muscles. A revolutionary theory is presented that proposes that exercise performance is regulated by the central nervous system specifically to ensure that catastrophic physiological failure does not occur during normal exercise in humans.

Certainly truths are self evident; others are more deeply hidden. Perhaps the most interesting question in exercise physiology is the one that is not often asked: why do muscles not develop rigor during exercise of high intensity and short duration (seconds to minutes) or when their fuel reserves are depleted during more prolonged exercise lasting two or more hours? Consider for example, the following. Human skeletal muscle can increase its peak rate of ATP utilisation to about 3 \( \mu \text{mol/s/kg wet weight} \) during seconds of beginning intensive exercise. Yet skeletal muscle ATP concentrations are 5–7 \( \mu \text{mol/kg wet weight} \). Thus, as little as seven seconds of peak exercise would deplete the muscle ATP stores and induce rigor as a catastrophic event if the peak rate of muscle ATP production was less than 80% of demand. Indeed, Newsholme and Start\(^2\) have calculated that total ATP stores in the flight muscles of the fly, \textit{Lucilia sericata}, would sustain about 0.1 second of running respectively if the oxygen cost of flight, and the white leg muscles of the rabbit about 1.0 second of running respectively since, as soon as the rate of ATP production lags behind its rate of use, muscle ATP concentrations fall in oxidative ATP production cannot be remedied. Rather, the muscles “fatigue” so that either the exercise must be terminated or it can continue only at lower intensities.

These concepts are based on the “limitations” or “catastrophe” models of exercise physiology, which conjecture that fatigue develops only after one or more bodily systems are stressed beyond their capacity, leading rapidly to the complete system failure that we recognise as exhaustion. The most long standing example, perhaps the earliest source of this intellectual mindset, is the theory that a limiting oxygen delivery to muscle, leading to skeletal muscle hypoxia or anaerobiosis, is the cause of fatigue during high intensity exercise of short duration, the so called cardiovascular/anaerobic/catastrophic model of exercise physiology.\(^6\)

However, the overlooked flaw in this explanation is that fatigue cannot be the immediate consequence of an imbalance in the rates of muscle ATP production and ATP use. For, if that were true, as soon as the rate of ATP production lags behind its rate of use, muscle ATP concentrations must fall inexorably, leading ultimately to muscle rigor. But, if muscle rigor does not develop during any form of exercise in vertebrates, then some other control mechanism must exist to terminate exercise even though the rates of skeletal muscle ATP production and use are in balance and skeletal muscle ATP concentrations are sufficiently high to prevent the development of rigor.

In a symposium at the 2001 American College of Sports Medicine Annual Conference in Baltimore, three presentations, reproduced here in an updated format, were delivered that, together, presented a different solution to this historical dilemma.

In the first paper, Noakes and St Clair Gibson present an historical overview of the evolution of the different models used to explain the physiological factors that are currently thought to limit human performance in exercise of either short duration and high intensity or lower intensity and longer duration. They pay special attention to the cardiovascular/anaerobic/catastrophic model because it is this model that has had the greatest influence on teaching and research in the exercise sciences since its first formulation in the early 1920s by AV Hill and his colleagues in London.

As astrophysicist Steven Hawking has written: “A theory is a good... model if it describes a wide class of observations and if it predicts the results of new observations. Beyond that it makes no sense to ask if it corresponds to reality, because we do not know what is reality independent of a theory. How can we know what is real, independent of a theory or model with which to interpret it?” Rather, the value of a model is that it makes predictions, the veracity of which can then be evaluated. If these predictions prove untrue, then the model must be relinquished: “Eventually the theory becomes a creaking and ugly edifice. Then someone suggests a new theory in which all the awkward observations are explained in an elegant and natural manner.”

Accordingly, in the second paper, St Clair Gibson and Noakes begin to establish the basis for a new model which they believe is better able to explain all the current observations. They argue that the peripheral model of fatigue is an example of a linear dynamic system in which system failure results from the progressive increase in the quantity of some, perhaps metabolic, variable until an absolute maximal concentration is reached. Once the system has failed catastrophically, causing physical exhaustion, a period of rest is required before exercise can again commence and during which the metabolites responsible for the fatigue are removed. However, they argue that, in humans with an intact CNS, no single metabolite has yet been identified that causes fatigue according to this linear model. More importantly, no example of absolute system failure, such as skeletal muscle rigor, has ever been reported; yet this is the logical prediction of the catastrophe model of Edwards.

They next review the contrasting central neural or central governor model of fatigue, which is based on the evidence that there is always skeletal muscle motor unit “recruitment reserve” in all forms of fatigue, indicating that the CNS regulates and indeed limits the extent of skeletal muscle recruitment, specifically to ensure that homoeostasis is maintained and catastrophe averted.

This system would probably be controlled by either feed forward regulation from higher cortical structures or reflex inhibition of efferent neural command by type III and IV chemoreceptors reacting to changes in peripheral substrate or metabolite concentrations, or even perhaps from the input of skeletal, cardiac, or respiratory muscle mechanoreceptors. On the basis of a number of their own other recent findings, they next propose an updated and more comprehensive version of their original model, described by Noakes et al.

According to their central governor model, they propose that during self paced exercise, which is the natural, as opposed to laboratory, form of human physical activity, the CNS continuously modifies the pace as part of a complex, non-linear dynamic system. In this model, the power output (work rate) during exercise is continuously adjusted on the basis of metabolic calculations performed at a subconscious CNS level. These calculations take into account prior knowledge acquired during previous exercise bouts, the planned end point of the current exercise bout, and the current metabolic rate, among many other potential variables. These
Integrative neural regulation of fatigue

subconscious calculations create a continuously adjusting power output, and hence pace, during exercise. This model provides the revolutionary insight that fatigue is not a physical event but rather a sensation that is the conscious manifestation of these subconscious CNS processes.20 29 30

Thus the model predicts that the ultimate control of exercise performance resides in the brain’s ability to vary the work rate and metabolic demand by altering the number of skeletal muscle motor units recruited during exercise. Thus the key question becomes: what physiological parameters determine the number of motor units recruited by the CNS in actively exercising skeletal muscles?

In the third paper, Lambert, St Clair Gibson, and Noakes11 examine peripheral regulation of metabolic activity, and how peripheral processes integrate with the regulatory centres in the CNS. They review the evidence showing that peripheral metabolites can provide information to central (brain) regulatory centres by way of afferent peripheral pathways and that they are therefore an integral part of the regulatory process. Alterations in efferent neural command generated as a result of peripheral afferent sensory input has a natural time delay, which leads to a continuously adjusting, oscillating power output, or pace, as part of a whole body, complex, non-linear, dynamic system.

Lambert, St Clair Gibson, and Noakes11 further argue that these alterations in power output may also be determined partly by regulatory systems that also exist in the periphery. These peripheral systems contribute to a hierarchical system of control with redundant properties, and this redundancy of command processes creates a more robust—that is, a complex—control system capable of maintaining homeostasis more efficiently than would single, isolated control processes that lack a central integration or the presence of redundancies.

Finally, they show that the neural and metabolic responses to exercise may be “reset” by factors such as antecedent exposure to exercise. Even a single bout of exercise before a particular event can alter metabolic activity and the conscious sensation of fatigue during the subsequent exercise bout, indicating that the central governor processes are mutable and can be continuously reset on the basis of continuing and novel experiences.

The ideas presented here are revolutionary as opposed to evolutionary because they make three novel predictions.

● Firstly, as at rest, now also during all forms of exercise, all physiological functions are homeostatically regulated by CNS control mechanisms to ensure that bodily harm does not result.

● Secondly, the conscious sensation of fatigue does not arise directly from the action of metabolites in the periphery, but rather from the regulatory centres in the subconscious parts of the brain, the function of which is to ensure homeostasis during exercise.30 Therefore the distinct sensation of fatigue is not directly related to a physical end point, but is rather an interpretation of the effect of the current level of activity on future exercise capacity and any threats that immediate and future events pose to the maintenance of homeostasis.30 31

● Thirdly, as the sensation of fatigue is an emotion rather than a physical state, pacing strategies and their control during self regulated exercise—the journey and not just the end point—are probably the most important phenomena in exercise physiology.

The authors therefore develop the argument that the “limitations” or “catastrophe” models of exercise physiology fail specifically because they do not explain what is most obvious. That far from being limited by the failure of one or more physiological systems during exercise, the reality is that all physiological systems in the body, both at rest and during physical activity, are homeostatically regulated in a continuous process, specifically to prevent catastrophic failure, including irreversible physical exhaustion, widespread cell damage, or even death, as the outcome of exercise.

What is already known

A popular teaching is that exercise performance is limited by metabolic changes in the exercising muscles, so called peripheral fatigue. This model predicts that exercise terminates when there is a catastrophic failure of homeostasis in the exercising muscles.

What this study adds

In a series of five articles introduced here, we present a revolutionary theory that explains the key findings better than the traditional catastrophe model of exercise physiology. The model proposes that exercise performance is regulated by the CNS specifically to ensure that catastrophic physiological failure does not occur during normal exercise in humans.

REFERENCES


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