Response to: Lessons from Popper for science, paradigm shifts, scientific revolutions and exercise physiology

Robergs[1] claims that the central governor model (CGM) of exercise performance has been re-stated so frequently that it can no longer be properly falsified according to the Popperian model of scientific disproof. In response I argue that the CGM is based on our and others' absolute disproof of the Hill model that has dominated the teaching of the exercise sciences for the past century. The basic disprovable assertion of the CGM is that the brain regulates exercise performance to ensure that a catastrophic failure of homeostasis does not occur. Even though the CGM is now more than 21 years old, no one has yet published experimental data that refute it.

THE TRUE HISTORY OF HOW THE CGM CAME ABOUT

Before the CGM, there was one undisputed truth in the exercise sciences: exercise performance is limited by the development of anaerobiosis in the exercising muscles.[2 3] This explanation was developed by Nobel Laureate A V Hill[4 5] in clear breach of Popperian science. Hill had formulated his theory without ever attempting to disprove any existing models, the most popular of which was perhaps that of Musso.[6] In 1904, Musso[7] described what he considered the features of exercise fatigue: 'The first is the diminution of the muscular force. The second is fatigue as a sensation. That is to say, we have a physical fact which can be measured and compared and a psychic fact which eludes measurement' (p 154). Interestingly Robergs does not accuse the Nobel Laureate A V Hill of 'pseudoscience'.

In the 1980s, we began to question the Hill's model as we were consistently unable to detect its most important underpinning, the so-called 'plateau phenomenon'.[8] We have since confirmed that this is a highly variable, indeed elusive, phenomenon.[9 10] Thus, by 1988, we had established that Hill presumed, but did not ever prove, the presence of this mysterious phenomenon,[8] thereby disproving the foundation myth on which Hill conceived his model.[11] Robergs ignores this information.

As Robergs writes, he was present in 1996 when I presented the first iteration of how our ideas had progressed by then.[12] The key advance was my realisation that Hill's model violates a fundamental physiological principle necessary to maintain health—the preservation of homeostasis in all bodily systems (regardless of the imposed stress). But Hill's model requires that skeletal muscle anaerobiosis develops as a result, he proposed, of a developing but regulated cardiac failure.[5 13] Since this model must lead to an inevitable myocardial ischaemia, it cannot be correct. We subsequently labelled Hill's model, the catastrophic anaerobic model of exercise performance,[14] to indicate its deviance from normal physiology as is currently taught.

These new ideas have been vigorously rebutted by those defending Hill's model.[3 15] We have rebutted all these criticisms[13 16 17] and have completed novel experiments designed to determine whether Hill's model or the CGM is more probable.

OUR STUDIES DISPROVING HILL'S CATASTROPHIC MODEL

In 2001, we described the evidence that formed the intellectual basis for our new model: '…peak cardiovascular function is reduced during maximal exercise in both acute and chronic hypoxia with no evidence for any primary alterations in myocardial function. Since peak skeletal muscle electromyographic activity is also reduced during hypoxia, these data support a model in which a central, neural governor constrains the cardiac output by regulating the mass of skeletal muscle that can be activated during maximal exercise in both acute and chronic hypoxia'.[18]

In a 2004 paper, we proved that, when exercising in the heat, athletes reduce their pace 'in anticipation' by decreasing the extent to which they recruit their exercising muscles.[19] By showing that athletes do not continue to exercise at an intensity that, if maintained, would cause physical harm (heat injury), we definitively disproved Hill's catastrophic model. This anticipatory slowing confirmed an observation we had made previously.[20]

In 2005, we collated published evidence from more than 100 scientific papers that are logically incompatible with Hill's catastrophic model.[14]

We then showed that persons exercising at a fixed rating of perceived exertion (RPE) completed less exercise in the heat than in cooler conditions,[21] confirming the presence of a central regulator of their exercise performance. Amann *et al*,[22] among many others, have provided independent confirmation for this anticipatory control of exercise performance, most unambiguously shown during exercise in hypoxia.

Hill's catastrophic model also predicts that exercise can only terminate after there has been 100% recruitment of all the skeletal muscle fibres in the exercising limb.[14] In contrast, we have shown that athletes do not recruit 100% of their active muscles during maximal running exercise.[23] The same applies during maximal voluntary exercise with the knee extensors.[24]

CONCLUSION

We appreciate Robergs' letter that allows us to collate the evidence we have provided to disprove Hill's catastrophic model. As Robergs points out, our model has gone through multiple reiterations since first conceptualised and that is a marker of openness, honesty and good science.

The basic disprovable assertion of the CGM is simple: the brain regulates exercise performance to ensure that a catastrophic failure of homeostasis

does not occur.[12 13 16–18 25] Our most recent iteration which represents a further significant advance in the development of this model of exercise regulation has just been published.[26]

I note that even though the CGM is now more than 21 years old, no one has yet published experimental data that refute it.

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