Review of: "Sustained Muscle EMG Activity to Contractile Failure During Incremental Exercise and Intense Constant Load Cycling: No Evidence of a Central Governor"

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Potential competing interests: No potential competing interests to declare other than I am the originator of the theory under review by the authors.

Thank you for asking me to review this article. I have already done so for another journal. The article was reviewed by at least 3 other reviewers who all came to the same conclusion. The article is based on a false premise. The authors are testing a prediction of the Central Governor that was never made by the authors of that theory and is not a feature of the model. I made this point very clearly, as did the other previous reviewers, to the original submission.

In short, the Central Governor Model cannot be tested in exercises in which the workload is fixed by the researchers. The only finding that the authors will ever find is exactly as these authors have found. The EMG activity will remain high until the athlete decides to terminate the exercise. For the simple reason that if the EMG activity fell before that point, the athlete, if running, would simply be ejected from the back of the treadmill. Since this never happens - the exhausted athlete always grabs the rails at the side of the treadmill the moment he chooses to stop exercising - the authors call this “voluntary exhaustion,” acknowledging that it is a brain-derived decision, exactly as the Central Governor model explains.

Unfortunately, the authors clearly believe they can continue to submit this article to other journals until they find one which does not have a decent peer review process, so that their article will be accepted. Instead, they should attempt to publish the data as is, instead of trying to score points on being more clever than those who developed the Central Governor Model.

I spent a great deal of time explaining why this is very poor science, but the authors have ignored everything I wrote as if it does not exist.

General comments written for the review of the original submission to the Journal of Sports Medicine and Health

I appreciate the careful data collection by these authors and the opportunity to review their paper. Before I begin the detailed point-by-point review, I’d like to begin with some more general comments.

When A.V. Hill proposed his model of how maximal exercise is “limited” by a catastrophic failure of coronary blood flow to the heart, causing blood flow to the exercising muscles to be limited, he ignored the key teaching in human physiology, which is that, directed by the brain, all functions in the human body are regulated “in anticipation” to maintain homeostasis and to prevent a catastrophic failure. Why a Nobel Laureate should have ignored this accepted physiological principle, I do

not know. Perhaps the concept of homeostasis was not yet fully developed by those who taught him in his university training.

So Hill came up with a model of exercise limitation that fundamentally violates that basic physiological teaching. And unfortunately, generations of exercise scientists, who might also not ever have undergone classical physiological teaching, believed that he was correct and so ensured that a fundamental core of teaching in the exercise sciences should perpetuate his erroneous understanding.

When I first gave this form of homeostasis, as it applies to exercise physiology, a name – the Central Governor – it was not as if I had magically discovered some new feature of human physiology. I was merely applying classical physiological principles to the study of exercise physiology.

It was essentially my attempt to convince the exercise scientists that their teachings are at variance with the core understanding of human physiology. I merely wished to point out that Hill had subverted our understanding of human physiology and that he had misled the world's exercise physiologists to follow him without understanding why Hill was in error.

My point is that from what we understand of how the body works in health and disease, there has to be homeostatic control by the brain in all forms of exercise performance in all environmental conditions. To disprove this requires one to disprove this fundamental, centuries-old understanding of how the body works, in which case we will have to revise all we teach in the modern courses of classic human physiology.

For example, I have also had an interest in two of the most tightly homeostatically regulated functions in the body – the blood glucose and sodium concentrations.

Thus, it is perfectly easy to show that homeostatic regulation of the blood glucose concentration can be driven to failure by having subjects fast overnight and then exercise without glucose ingestion for as long as it takes for them to become physically exhausted. By which time, their blood glucose levels will be low, and they will have cerebral symptoms of glycopenia. But they stop before their brains are harmed by such low blood glucose levels, showing that there is still a final "central governor" mechanism to prevent brain (in this case) damage when all else has "failed."

Similarly, there are athletes who have drunk so much fluid during exercise that they have developed fatal exercise-induced hyponatremic encephalopathy, even though, in health, the blood sodium concentration is very tightly homeostatically regulated. We found that only those with at least 2 biological variants will develop this condition. Even then, they should not die as they will fall unconscious and so will stop drinking. When fatalities occur, it's usually because the athletes have received inappropriate medical care (intravenous infusions of dilute sodium solutions).

So the point is that it is quite easy to argue that these two examples prove that catastrophic failure can happen in humans during exercise. But neither of these examples disproves the classical teaching that blood glucose and sodium concentrations are tightly regulated homeostatically by a central governor-type mechanism.

The next point is that we have clearly proven that this central governor mechanism acts “in anticipation,” as it must if it is to anticipate and then prevent a catastrophic failure. Some of these papers have been published in leading international journals of physiology, and the data and our interpretation are fully accepted by the scientific community.

Indeed, the promotion of the central governor model opened up the field of “pacing” as the ultimate anticipatory action in the exercise sciences. If the central governor does not exist, then what explains anticipatory exercise behaviours or even the “endspurt”? I have written extensively on these topics.

Surely every competitive 800-3000m running event ever run is a test of each athlete’s cardiovascular control mechanisms – either catastrophic or anticipatory – since it mimics maximal exercise testing in the laboratory to determine human cardiovascular “limitations,” according to the Hill catastrophic model. Yet I’m not aware that all these events always end with the development of cardiac failure/myocardial ischemia as Hill’s model predicts.

The final point is that one cannot test an anticipatory model of exercise control using a fixed work rate model, as these authors have. I wrote about this extensively many years ago, and the criticisms remain as valid today as then.

https://www.researchgate.net/profile/Timothy-Noakes/publication/5429159_Testing_for_maximum_oxygen_consumption_has_produced_a_brainless_model_of_human_exercise_performance/links/5de7c96e92851c836462656f/Testing-for-maximum-oxygen-consumption-has-produced-a-brainless-model-of-human-exercise-performance.pdf

And I am not alone in making this point:

https://www.cambridge.org/core/journals/comparative-exercise-physiology/article/abs/limitations-of-the-constant-load-and-selfpaced-exercise-models-of-exercise-physiology/52C0B2CED6DE640644AF31D8BAF6E402

In addition, as it directly relates to the topic in this research study,***there is clear evidence that AV Hill did not EVER establish the presence of the “plateau phenomenon”*** as I describe in this article on which I worked for many months many years ago.

https://bjsm.bmj.com/content/bjsports/42/7/574.full.pdf

To check this, I asked my PhD student Fernando Beltrami to spend some years researching whether or not the “plateau phenomenon” really exists.

This is his completed thesis

https://open.uct.ac.za/items/45584f23-392a-4a2b-9936-382dd7762ba3

and here are the papers describing some of his more important findings:

A high prevalence of false-positive maximal exercise tests:

https://www.sciencedirect.com/science/article/abs/pii/S1440244013001771

Conventional testing produces submaximal values of VO_2max

https://d1wqtxts1xzle7.cloudfront.net/50576049/Conventional_testing_methods_produce_sub20161127-6601-165xr2n-libre.pdf?1480273127=&response-content-disposition=inline%3B+filename%3DConventional_testing_methods_produce_sub.pdf&Expires=1699709837&Signature=IetzIUEfzBjD4JkmcYbWZ-hVcfGlJBfAFCs2isc8ZU4St0yhZd9m3Mg0KDTHIM~Dl49EzCUKEx8nSQ87MyEChShyBymQuib~cttBDCxhHkYDH9UKahHT80vGVJKu40XNqXDT6KIOYLvs8JHwJlezHjn~mELpbsRABl8JSUGSWvRbRTCxNiGu39y3r5VnLGEmJeMjXB-gLtIB7KhuCxBdxKNORJ70DZxrIA~h3WeYskKoPLA1FHHdwxF725ViACVkydVMSwguMzeyTLEL~QDJasaaymYyk0g1wD9oOiy0LcnD5x7F3yGjsGJjHz7V~qTBZNOKXaF0bWStv1PHboOGOg__&Key-Pair-Id=APKAJLOHF5GGSLRBV4ZA

More on conventional testing produces submaximal values of VO_2max

https://digitalknowledge.cput.ac.za/bitstream/11189/8851/1/Conventional_testing_produces.pdf

In summary, these studies show that the plateau phenomenon is not a marker of a maximal exercise test that has been terminated by myocardial ischemia producing a limiting blood flow to the exercising muscles.

I turn now to specific comments on statements in the text:

PAGE LINE COMMENT

2 5 It is not possible to evaluate an anticipatory control mechanism acting during exercise in humans using a fixed work load. I have provided two references above which explain the futility of this attempt.

7 What do you mean by volitional exhaustion? Volitional means “relating to the use of one's will,” as in "acceptance is a volitional act". But your entire argument is that whatever terminated this exercise bout, it has nothing to do with “volition” — a central brain regulatory effect. According to your explanation, it must be purely due to a failure of skeletal muscle contractile function in the face of increasing skeletal muscle recruitment. So you’ll need to use a different word – perhaps “non-volitional termination of exercise.” You need to be consistent in your terminology.

17-18 This is not inconsistent with the Central Governor Model (CGM). The CGM predicts only that the exercise will terminate before there is 100% recruitment of all available motor units in the exercising muscles. It also predicts that in health, exercise will terminate before the development of clear evidence for harm to major organs having occurred. You did not test whether or not 100% of the available motor units were active at exhaustion. Nor did you present any evidence for major organ damage terminating exercise. The CGM also predicts that fatigue will develop progressively as the exercise becomes more unbearable and will ultimately lead to the termination of exercise. Did you make any measurements to account for this clearly anticipatory effect?

3 4 With all respect, Tim Noakes is not a cardiologist, although he is South African. I’m not certain why my nationality has anything to do with my opinion.

10 In presenting a scientific argument, there is a certain protocol to which all ethical scientists need to adhere. For example, you reference 2 articles (3 and 4) in a way that you might wish your readers to believe that the arguments raised by those 2 authors have never been answered. But this is simply not true. I have spent a great deal of time rebutting both

https://link.springer.com/article/10.2165/11583950-000000000-00000

https://bmjopensem.bmj.com/content/4/1/e000277

The authors' ethical responsibility is to ensure that the readers of their article are fully informed that I have responded in great detail to both these articles. The readers can then be left to make up their own minds about whether there is information "anomalous to the CGM" and whether the CGM may violate "core principles". What exactly are those anomalies and core principles that I did not address in my rebuttal? That should be the starting point for these authors' concerns and criticisms. Not simply re-hashing the same complaints that Shephard and Robergs have already made and which have been fully answered, at least to my satisfaction and seemingly to the satisfaction of many others who have not raised these issues again in the intervening 5-13 years.

15-16. These issues are addressed in Fernando Beltrami's thesis referenced above. The point is, we have spent an inordinate amount of time and effort trying to understand this phenomenon, rather more than those involved in your reference 6. We actively tried to prove the models wrong by developing entirely novel methods of exercise testing.

22But this statement is neither logical nor supported by all your data. To repeat, the CGM is an anticipatory controller whose actions cannot be fully displayed in any form of exercise at a fixed work rate. Here are 2 articles showing the evidence for it during exercise in the heat:

https://www.researchgate.net/profile/Laurie-Rauch/publication/8566787_Impaired_exercise_performance_in_the_heat_is_associated_with_an_anticipatory_reduction_in_skeletal_muscle_recruitment/links/00b4951595053d45b7000000/Impaired-exercise-performance-in-the-heat-is-associated-with-an_anticipatory-reduction-in-skeletal-muscle_recruitment.pdf

https://pubmed.ncbi.nlm.nih.gov/16497719/

Now you want to say that the same must happen at a fixed work rate. But your alternative data show it happening: For example, on page 7, lines 4-7, you describe volitional fatigue as the inability to sustain a pedalling cadence within 20 rev/min of the predetermined value. Cadence is not directed by the muscles – it's directed by specific areas in the brain. So here is clear evidence, even at a fixed work rate, that the brain is trying to reduce the work rate even before "volitional exhaustion" happens. Please explain why this is not the action of a CGM trying to reduce the "volitional" work rate prior to the termination of exercise. Did you not see evidence for this on your EMG traces taken immediately prior to the termination of exercise, or were these data not analysed? Indeed, how do you interpret EMG data if the cadence is changing? I don't know; I'm just asking.

4 1 The peripheral governor model, clearly described by MacIntosh,

https://pubmed.ncbi.nlm.nih.gov/21326373/

, and which in no way detracts from the CGM. The CGM does not dictate that there can be no peripheral fatigue during exercise. In fact, we have provided some of the better studies quantifying the development of peripheral fatigue during exercise.

For example, https://pubmed.ncbi.nlm.nih.gov/30487392/

4 And why would the brain want to have influence over the development of “contractile failure”? It’s not its job. Its job is to prevent a catastrophic failure. And how do you define “contractile failure,” and how does this differ from a peripheral governor, etc.?

6 Whether or not sEMG activity “dampens” at volitional fatigue is not relevant to the CGM for reasons already described. A fixed work rate test is not how humans exercise. We work by anticipating the future and adjusting the work rate accordingly. The only prediction the CGM makes is that exercise will terminate before there is 100% motor unit recruitment in the exercising limbs. Because if there is 100% recruitment, then it's very likely that harm will occur. If exercise terminates before that point and harm has not occurred, then the CGM has worked effectively even if it allowed the sEMG to increase near the end of exercise. But at “volitional fatigue,” EMG activity will have dropped sharply. So what does one classify as the end of the EMG recording?

10 Wrong. This is not evidence for a disturbance of homeostasis. Rather, it is more likely a method to prevent the development of pulmonary hypertension and acute pulmonary edema in those athletes with very large cardiac outputs which, when circulating through the pulmonary circulation, risk causing pulmonary edema.

https://pubmed.ncbi.nlm.nih.gov/15107409/

11-26 Not sure how this is relevant. Peripheral fatigue is a fact. Whether or not it is caused by these changes is uncertain. What is important is that the presence of peripheral fatigue does not invalidate the CGM.

27-32 But to properly study this question, you would need to show that any progressive increase in sEMG was associated with harm and that all motor units were active at the point of exhaustion. You don’t explain why a progressive increase in sEMG disproves the CGM. If the athlete decides, as each does somewhere in the middle to end of the exercise bout, for how long he or she is prepared to continue exercising, and if he or she is developing peripheral fatigue, then the only way to sustain that “volitional” exercise to “volitional” exhaustion (and not to terminate exercise prematurely) is to maintain the same muscle power output by recruiting more fibers, each of which is producing less force. Then ultimately, the athlete still decides to give up the “volitional” effort and to stop exercising as part of an anticipatory CGM action that anticipates that if the constant work rate exercise continues much longer, there is a real risk of harm.

Until you can disprove that argument, you have not disproved how the CGM may also be working, even in exercise at a constant work rate.

11 18-19 These data appear clear – neural output is increasing to the point of what you still term “volitional fatigue”. But as I’ve asked repeatedly – what is volitional fatigue, and what causes the subjects suddenly to be unable to keep pedalling at the required cadence? And how do you know that the choice to stop exercising is not an action of the CGM acting in anticipation of future harm if the exercise continues? The answer is you cannot. So your data are not some definitive proof against the CGM.

13 9-24 None of these findings dispute the CGM for reasons already mentioned. The athletes chose to stop exercising at the point of “volitional fatigue”. If you had been using a non-fixed work rate, somewhere near the end of exercise, the subjects would have started to pedal slower and reduce their work rates with reductions in sEMG activity. This would then identify the anticipatory nature of the choice to slow down. But your experimental research design prevented your subjects from doing that.

Thus, your experimental method determined the outcome you would record, even before the experiments began. You could only ever record either an increase or no change in sEMG activity; you could not expect to measure a falling sEMG activity whilst the athletes were continuing to exercise at the same fixed work rate.

14 17 To restate: The CGM does not predict a reduction in sEMG activity prior to the termination of exercise at a fixed work rate, so your findings do not disprove the CGM. What you clearly show is that sEMG activity increases progressively near the end of exercise and **despite this, the subjects still stop exercising.** Why do they stop exercising according to your model? **They stop because they make the conscious/subconscious decision to stop, even though they should be able to continue according to your findings.** That is the paradox in your data that you do not choose to understand.

14 24. This explanation cannot be the complete story because it does not explain why subjects reduce their pedalling frequency, which is a centrally regulated (i.e., brain) phenomenon. Clearly, part of a CGM effect is reducing work output to protect the athlete – in my not so humble opinion.