Multiple Models Can Concurrently Explain Fatigue During Human Performance

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ABSTRACT

Int J Exerc Sci 2(4): 280-293, 2009. One of the most commonly and thoroughly studied paradigms of human performance is fatigue. However, despite volumes of research there remains considerable controversy among scientists regarding definitive conclusions about the specific mechanism(s) contributing to fatigue. Within the literature there are three primary yet distinctly different governing ideas of fatigue; the traditionally referenced central model and peripheral model as well as the emerging central governor model (CGM). The CGM has recently been advocated by a limited number of researchers and is suggestive of a more integrative model of fatigue when compared the traditional peripheral and central models. However, more work is needed to determine the specific and perhaps synergistic roles of each paradigm during exercise or sport activity. This article contains three components; (1) a brief overview of the problems associated with defining fatigue, (2) a description of the models governing interpretation of fatigue and, (3) a presentation of multiple interpretations of selected data to demonstrate that some results can be reasonably explained using multiple models of fatigue, often concurrently. The purposes of this paper are to reveal that a) perhaps it is not the results that suggest a certain paradigm of regulation, yet that it may be a product of an *a priori* definition that is being employed and b) an integrative model of central and peripheral fatigue may present a plausible explanation for fatigue vs. adherence to the notion that each paradigm is mutually exclusive.

KEY WORDS: Central nervous system, feedback, feedforward, regulation, power

INTRODUCTION

Fatigue is a common phenomenon many individuals, athletes or not, routinely experience (36). However, mechanisms and characteristics involved in fatigue associated with exercise or sport performance are not well-understood. Within the literature, there are two established and one emerging models/theories present in the vast majority of fatigue research concerning sport and exercise performance (23, 31). If changes within the muscle are deemed the causal factor in a loss of power output, it is typically regarded as a result of peripheral fatigue (8, 18, 40, 43). Conversely, central fatigue is often associated in instances where the central nervous system has a diminished neural drive to muscle and, ultimately, is independent of the muscle's contractility (27, 33, 37, 38). In central

fatigue, as seen in peripheral fatigue, a diminished power output is immediately observable, however the principle difference rests in the underlying mechanism. There is, however, an emerging model that is integrative in nature. The central governor model (CGM), has been advanced recently in a series of studies by a number of researchers (3, 4, 16, 24, 30, 31, 36, 37, 38). This theory will posit that fatigue is a "feed-forward" process that is pre-determined prior to exercise initiation and is integrative in nature. That is, the brain acts as a "regulator" of the body as it interprets messages from the periphery as a result of work output.

PROBLEMS IN DEFINING FATIGUE

Defining fatigue, in many cases, has become as cryptic as determining the specific contributory mechanisms responsible (8). An exhaustive review of all models and mechanisms involved in fatigue research is beyond the scope of this article, but readers are directed to excellent reviews of this topic by Noakes (30) and Abbis and Laursen (1). Abbiss and Laursen (1), as well as Lambert et al. (24), have identified the "reductionist" idea of fatigue in exercise physiology as a common limiting factor prevalent in fatigue-related research. Traditionally, reductionism attempts to reduce a complex phenomenon to a singular determining variable. However, reductionism, as it applies to fatigue research, may also exist when conflicting models of fatigue (i.e., central vs. peripheral) are chosen *a priori* as the basis for the declines in human performance during laboratory or field testing (2, 24). In essence, many exercise physiologists will view fatigue as an unavoidable consequence of physical activity that will, at some point, lead to a critical point of metabolite accumulation (3, 5, 18). This critical point is thought to be the primary influence directly responsible for attenuated power and consequently exercise impairment/cessation (22). Conversely, many sport or exercise psychologists contend fatigue results from "sensations" or "feelings" (vs. metabolite accumulation) during physical activity that inevitably lead to voluntary discontinuation of exercise (2, 36). In many cases, reductionism results in fatigue being attributed to a presupposed idea rooted in the operational definitions that guide and govern the interpretation of sport and exercise performance. That is, an identical decline in muscular power may be attributed to entirely different causes based on the model believed beforehand to be responsible. However, multiple models of fatigue often offer an equally plausible explanation, thus explanations limited to a single model may be premature.

It is usually within the operational latitude of the definition(s) of fatigue that the majority of discrepancies between theories governing fatigue and human performance are grounded. Perhaps the mechanism of fatigue is not as elusive as often believed and, ultimately, it is within the different 'interpretations' of fatigue that a common thread linking discrepancies throughout the literature may be found. This review is not meant to serve as an exhaustive presentation designed to advocate the 'pros and cons' of central, peripheral or a CGM model of fatigue. It is, however, intended to demonstrate the shared and sometimes distinct interpretative properties of multiple models of fatigue during human performance. Thus, a brief synopsis of the

ideas and principals accepted concerning peripheral, central, and CGM models of fatigue is warranted in an attempt to gain clarity concerning fatigue and to briefly present governing principles of interpretation found in the literature.

Perhaps a primary problem in defining fatigue is that it is quite often considered mutually exclusive to either a peripheral or central consequence. Indeed, the peripheral model of fatigue has been the more traditionally accepted of the models and is more widely accepted in the literature and throughout exercise physiology textbooks (18, 22, 36, 40). However, the potential of a central model of fatigue has been recognized for quite some time (8). Classically, a central model has been regarded as being, at the very least, subsidiary in the fatigue process. It also seems groups adhering strongly to one model or the other are resistant to considering the possibility of an integrated model in which ideas from both models provide reasonable explanations. Recently, there has been an ongoing controversy among central versus peripheral camps when discussing effects influencing human performance during sport and exercise performance. Perhaps the most notable of all central models of fatigue has been that of the "teleoanticipation" theory set forth by Ulmer in 1996 (42). This landmark idea has subsequently manifested the "complex model of fatigue" proposed by Lambert et al., (24) by way of the CGM theory proposed by Noakes, Ansley, Lambert, and St. Clair Gibson among others (3, 4, 16, 24, 30, 31, 36, 37, 38). It is important to note that a contrasting difference between a traditional central model of fatigue and the more contemporary CGM model is that a central model of fatigue is catastrophic in nature (similar to peripheral models) while the CGM model is anticipatory and is, by design, protective of catastrophic homeostatic perturbations.

DESCRIBING MODELS OF FATIGUE

Fatigue, either centrally or peripherally mediated, will lead to a marked decrease in power output or performance (8, 38). However, the CGM will argue that fatigue is an emotion elicited by the brain in order to achieve optimal performance and avoiding an overt threat to homeostasis, rather than a physical manifestation of a reduction in power output (34). Thus, there seems to be very little common ground shared among professionals supporting the contemporary models and those siding with the more traditional peripheral model. There are however, common characteristics beyond reductions in power or performance which seem to be loosely accepted by central and peripheral advocates. Some of the following found throughout the literature are (A) fatigue is coincided with a disruption of homeostasis (20, 21), (B) it is unavoidable (8, 21), (C) it is multifaceted and ubiquitous (8, 36), (D) develops following a myriad of processes (23), and (E) fatigue may happen anywhere along the chain of command (i.e., the brain to the muscle) (43). Again, the CGM will contest these points almost uniformly as it will posit that a fatigue, traditionally defined as reduced power, will not be necessarily observed if an individual is allowed to self-regulate intensity (34, 39, 41). While novel, reviewing and interpreting each individual definition for fatigue is beyond the scope of this paper, however, further investigation is warranted.

Yet another problem facing the study of fatigue is that the interpretation of fatigue has proven to be problematic, as noted by Kay et. al. in 2001, "central to the study of fatigue is the definition that is employed." This is indeed problematic as, in many cases noted in this review, the definition adopted *a priori* by investigators may lead to interpretation of data that fits the model to which they adhere. However, as stated, the same data collected by a different group might offer an alternate, yet equally convincing explanation. Additionally, Abbiss and Laursen (2), as well as McKenna and Hargreaves (26) have recently described the problems associated with defining fatigue and the inherent problems this causes in disseminating research findings. However, this is problematic as, in many cases, the definition adopted *a priori* by investigators may lead to interpretation of data that fits the model to which they adhere. Alternatively, different researchers may be able to offer contrasting yet equally convincing explanations for data presented championing either a CGM, peripheral, or central model.

The on-going debate to determine an ultimate cause of fatigue during bouts of physical activity is certainly not a new idea in the field of sport and exercise science. The idea of a central influence was first introduced by Lombard during the late 1800s (25). In his study, Lombard reported an increased resistance to fatigue when active muscles were provided electrical stimulation when compared to voluntary contraction (25). This is a classic example of the central fatigue in the form of decreased neural drive during exercise, despite consciously providing maximal effort (22, 37). Since then studies have been published consistently demonstrating support and opposition to Lombard's original central fatigue hypothesis. In 1954, Merton conducted his classic study that has long been considered a paramount investigation that accurately describes and models the peripheral fatigue hypothesis. Contrary to Lombard's results that were reported decades earlier, Merton summarized his study by stating, "Fatigue is peripheral, for when strength fails, electrical stimulation of the motor nerve cannot restore it." (28). Merton also concluded that, "Neuromuscular block is not important in the fatigue of the volitional tetanus. Even in extreme fatigue, action potentials evoked by nerve stimulation are not significantly diminished." (28). Since Merton's original work in 1954 the vast majority of evidence reported, until recently, agree that the primary site of fatigue is within the muscle itself (i.e., peripheral) (10). However, it is possible that knowledge of this classic study has driven the mindset of some scientists to automatically adhere to a model which is always peripheral in nature. Consequent to this possibility would be the design and interpretation of numerous experiments which, while purported to agree with Merton's results, could be explained by the other models as well.

The rivalry amongst central and peripheral researchers was seemingly renewed again in the early 1980s, when Bigland-Ritchie published a report identifying potential sites for fatigue during exercise (5). In this report, eight potential sites at which fatigue may occur were identified; (i) excitatory input to higher motor centers, (ii) excitatory drive to lower motor neurons, (iii) motor neuron excitability, (iv) neuromuscular transmission, (v) sarcolemma excitability, (vi) excitation-contraction coupling, (vii) contractile mechanism, and (viii) metabolic

energy supply and metabolite accumulation. In this review, Bigland-Ritchie (5) identifies both central (sites i – iv) and peripheral (sites v – viii) that may be identified as factors potentially contributing to fatigue. A more probable scenario proposed by Bigland Ritchie (1984) and others (7, 10, 11, 24, 34) is that, in many cases, these sites work synergistically to during exercise and sport performance.

Following Bigland-Ritchie's report (5), the specific role of central and peripheral fatigue components have been typically regarded as task dependent (23, 29, 34, 43). Still, if it is true that sometimes fatigue can be attributed to central factors and sometimes peripheral factors, there must exist some "cross over point." It is unlikely that this cross-over point is concretely established within each individual; rather this point will potentially have tremendous interindividual variability. Indeed, there is most likely a "meshing" near the middle of this continuum between central and peripheral mediators, even if at the end of each spectrum there are well-established factors that are explained exclusively using one model or the other.

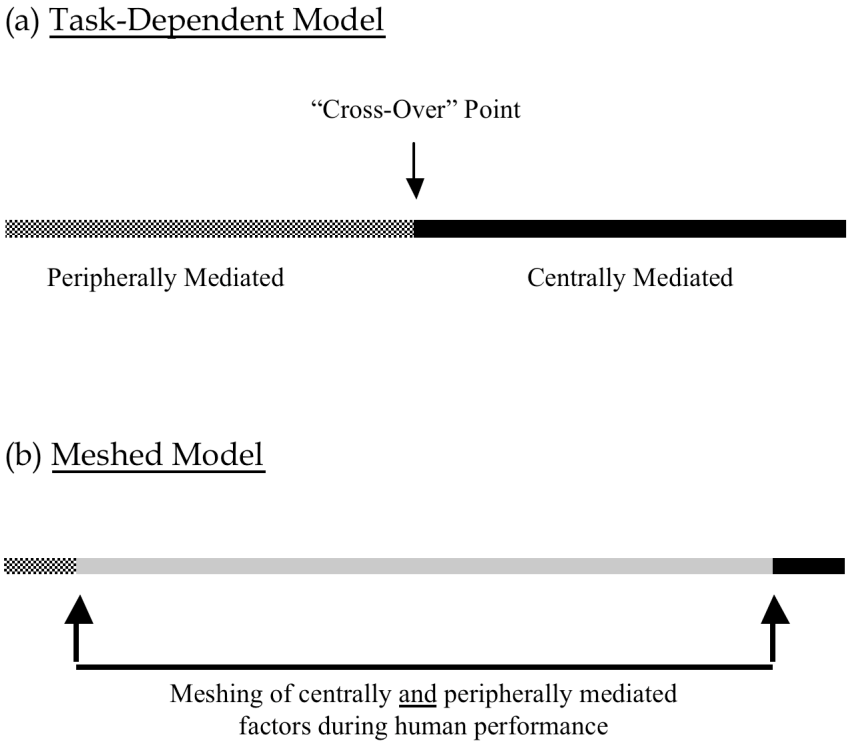

Figure 1. Schematic illustration of a task-dependent model (a) demonstrating a generic "cross-over" point from peripherally to centrally mediated exercise (arbitrary threshold proposed that regarding the role dominion in the regulation of exercise/sport performance. Also presented is a depiction of a meshed model (b) - a further elaboration of the integrative model (proposed by Lambert et al., 2005). This model posits that certain tasks are not relegated to either peripheral or central control, rather sport or exercise performance is largely regulated with central and peripheral factors functioning concurrently.

In much of the previously published literature, both peripheral and central mediators are considered to have contributory roles to subsequent fatigue during exercise bouts of maximal effort, however, it seems that one model is typically considered a primary contributor while the other relegated to be a less important secondary influence (5, 34). For example, a study by Kent-Braun (23) revealed that the relative contribution of central fatigue was minimal (20%) when compared to peripheral factors during high-intensity isometric exercise resulting in fatigue. Similarly, Nordlund et al., (34) found that there were contributions from both central and peripheral sites during nine bouts of 10 intermittent isometric maximal voluntary contractions (MVC) of plantar flexion. Results from this study revealed that there was no significant relationship between the decreases in level of activation throughout all nine bouts to the level activation achieved during the first bout. These results suggest that central fatigue had little influence on the participants' development of fatigue, as there were similar levels of neural innervation, as measured by twitch interpolation technique, identified throughout the bouts (34). Further, there was no relationship ($r = 0.00$) identified between central fatigue and level of activiation, whereas peripheral fatigue had a significant positive correlation with the level of activiation ($r = 0.57$) throughout the 10 exercises (34).

Conversely, Kay et al. (21) reported considerable central regulation during a 60-min self-paced cycling protocol that was interspersed with 10 1-minute all-out sprints. In this study, there was reduced neural drive, identified by integrated electromyography (iEMG), during sprints 2-4 with a noticeable increase in efferent output during later sprints (21). These authors concluded that this reduction in neural drive during the initial stages was a result of central regulation in order to provide ample energy reserves to maintain power outputs towards the end of the trial (21). These studies are offered as a brief example of the variability of results among studies, readers are directed to a number of excellent reviews that fully address the role of central (24, 26, 38) and peripheral (10, 14) factors during human performance. Many of these investigations ultimately consider and acknowledge the possible role of central and peripheral factors influencing fatigue, however, it is typical for a "primary" model to be identified and given noticeably more attention that the opposing model (16, 37, 40).

The roles of peripheral and central regulation of power output, however, have grown increasingly more complex and are now being intertwined in integrative models in order to better describe fatigue during exercise of different modes, intensities, and durations. A model of fatigue permitting exploration of both the influence of central and peripheral mediators (as proposed by the CGM) offers the advantage of identifying factors contributing to fatigue among various exercise paradigms without subscribing (especially *a priori*) to the idea that fatigue must exclusively result from either one or the other. The idea that fatigue may result from a combination of factors (including central and peripheral) is analogous to other systems in an intact physiological system. For example, ventilation is responsive to multiple input pathways which function collectively rather than any

single mediator that is universally dominant in all situations.

Recently, Lambert et al. (24) produced a "complex model" of fatigue incorporating an integrative approach using peripheral and central regulators working synergistically to attenuate challenges to homeostasis during various exercise paradigms. While integrative, this approach draws principally on the role of a central regulator/governor proposed by Noakes et al (33) and, consequently, this novel idea has attracted much criticism (43). It seems few researchers are ready to concede to an integrative regulatory model and instead adhere to the traditional task-dependent model dictated by a predominant influence (i.e., peripheral or central). As stated, this can be problematic as the cause of fatigue can be presupposed due to the operational definition offered *prior to* data interpretation. Indeed, cases exist in which the cause of fatigue may be considered strictly peripheral (28) or central (21). However many studies could be well-defended using a central, CGM and peripheral model, provided that sufficient data to implicate such mechanisms. To demonstrate this idea, selected studies have been re-interpreted in a manner alternative to the original authors' initial interpretations. This will demonstrate that conclusions for many fatigue studies may be viewed from multiple vantage points without adhering solely to a single model. Consequently, when analyzing data, assuming a position with no pre-determined operational definition of fatigue may often necessitate presentation of multiple plausible explanations for a given data set. While potentially criticized for ambiguity, our approach is arguably preferable over allowing a pre-established idea to dictate attribution of fatigue peripherally or centrally without consideration of an equally reasonable alternative explanation.

The following two studies (3, 18) are similar in that both reported on pacing strategies involved in 4-km cycling time trials, however, different methodological approaches were taken by each group. Accordingly, conclusions from each author conflict (with each other) in determining an appropriate model of fatigue. Ansley et al. (3) concluded that pacing strategies adopted in their trials were most likely a result of CGM regulation. Conversely, Hettinga et al. (18) concluded peripheral mediation of pacing. These studies are subsequently presented with a summary of the original findings put forth by their respective authors. Plausible alternative interpretations implicating an opposing fatigue model will be presented, to demonstrate the openness to interpretation often evident in research concerning fatigue. However, for the sake of brevity, these interpretations will primarily address the concepts typically associated with a traditional peripheral model of fatigue and the emerging new model of regulation, the CGM. This, of course, is not meant to discredit the notion of a central model of fatigue; rather the focus of this article is intended to demonstrate the shared and sometimes distinct interpretative properties of both multiple models of fatigue during human performance. This will help substantiate our current stance that multiple models of fatigue, in many cases, can be identified as "contributory" influences regulating human performance depending upon the operational definition used to govern the interpretation of results. Our stance is that data may often be

interpreted from multiple vantage points and care should be taken that adherence to preconceived notions regarding fatigue mechanisms not be permitted to dictate conclusions to the point of discrediting an alternate theory.

Study 1

Ansley et al. (3) had subjects complete a VO_{2peak} cycling test, and seven days later complete three successive 4-km time trials, each separated by 17-min of recovery; [10-min were passive, 5-min performed at a self-selected intensity, and a 2-min session performed at 35–40 $km \cdot h^{-1}$ prior to initiation of each subsequent time trial]. Throughout trials only distance covered was provided as feedback with participants encouraged to complete each trial as quickly as possible.

VO_2, HR, and lactate concentration [La] were not significantly different among the three repeated 4-km time trials (3). However, peak power outputs were significantly lower in the final two time trials vs. trial one (3). There were no significant differences in average power among all trials (3). Rectus femoris iEMG recordings during trials revealed lower values (~ 25%) than corresponding maximal voluntary contraction (MVC) values and the sequential measurements were not significantly different among 4-km trials (3). Accordingly, Ansley et al. (3) determined their results substantiated a centrally-mediated regulator. This notion hinged on four primary findings of (a) no significant differences in time to completion in the trials with a concomitant reduction in peak power output in the second and third trials and (b) lack of 100% muscle recruitment (c) observation of an increased power that was "tracked" by iEMG during the final 60-s of all trials and (d) evidence of the endspurt phenomenon (3). These factors, while not the only criteria associated with this notion, are certainly considered "hallmark" indications of CGM influence (3, 21, 35).

The first finding supporting CGM model of regulation is the uniformity of completion times with significantly different peak power. This, of course, is indicative of the proposed "learning effect" employed to achieve an optimal pacing strategy (3), which is a primary tenet of central regulation. A primary goal is to regulate power output via feed-forward mechanisms that respond to undulating metabolic response, prior experience, perceptual response, as well as distance covered (or distance remaining) throughout a given exercise session, consequently creating optimal power to ensure both successful completion and avoid critical disruption of homeostasis (3, 6, 16, 24, 33, 35, 36, 39, 41, 42). Secondly, tracking iEMG with increased power output also is considered a hallmark indication of the CGM (3, 21, 23, 35, 37). Proponents of central regulation will argue that peripheral fatigue can only be considered when there is a concomitant reduction in power output with increasing neural drive (35), which was not observed in Ansley (3).

Probably the most significant finding from Ansley et. al. supporting the notion of the CGM, is the presence of the end spurt phenomenon during the conclusion of each interval. Similar to the notion of tracking iEMG with power output stated previously, the endspurt phenomenon relates an individual's innate ability to produce a power output similar to (in some cases exceeding) initial power outputs during

exercise or sport performance. The idea of an endspurt is well-supported by Ansley and colleagues in that all of the individuals had knowledge of an anticipated endpoint (i.e., distance covered feedback) and were able to adjust (subconsciously) there pacing strategy via the brain's teleoanticipatory center to create the proper "algorithm" (37). This adjusted (subconsciously) pacing process is employed to ensure successful completion and reserve ample metabolic reserve and regulate inhibitive metabolic by-products (i.e., H^+, [La], etc.) that enable a subject to produce an endspurt (37).

Conversely, using data from the Ansley study (3) there is ample evidence for alternate interpretations identifying a peripheral influence. There was a significant reduction in peak power output from the first 4-km time trial to subsequent trials (3). This reduction in peak power was coincided with a marked (although not statistically significant) VO_2 increase and increased time to completion in both the second and third time trials, with time to completion reaching significant difference between the first and second trial (3). Additionally, blood lactate levels were elevated to near maximal levels and increased (although not significantly) with each successive trial (3). The accumulation of blood lactate and consequent pH reduction has been linked to fatigue in various pacing strategies (12). Still, others claim accumulated lactate and lowered pH have little effect, if any, on optimal performance during intense exercise (3, 19, 29). While there seem to be overall equivocal ideologies regarding lactate accumulation and pH, there are other physiologic consequences that can be linked, even if not considered causal, to peripherally regulated performance during intense exercise (14).

The level of plasma lactate concentrations and other metabolic data confirm subjects in Ansley et al. (3) were indeed performing high-level, near-maximal work. Further, it has been suggested that repeated bouts of high-intensity exercise, as in Ansley et al. (3), can lead to marked reductions in glycogen (14) as well as phosphocreatine (PCr) stores and, perhaps more detrimental, may affect fatigability at the cellular level by disrupting excitation-contraction (E-C) coupling through various intracellular mechanisms adequately addressed in previous literature (11, 14). Ansley et al. (3) correctly argue that the rest period allowed the subjects provide ample time between trials for adequate PCr recovery (cited in 6, 17), but the notion of reduced glycogen stores is left largely unexplained. Furthermore, Fitts and Balog (11) have posited that recovery in large muscle groups (i.e., quadriceps) from high-intensity exercise is a biphasic process, in that there is a "rapid phase" (1-2-min) as well as a "slow exponential phase" (50-60-min). The effect of repeated high-intensity exercise and allotted recovery period observed in the study by Ansley et al. (3) may have led to only a partial recovery prompting a negative effect on E-C coupling (11, 14). Indeed, it is possible to surmise the peripherally mediated E-C coupling effect possibly leading to declines in iEMG which served as the direct evidence of central regulation originally acknowledged in Ansley (3). This alternative interpretation may serve as evidence implicating a peripheral influence offering plausible explanations to the significant reduction in peak power and

increased time to completion observed by Ansley et al. (3).

Study 2

In a study similar to Ansley et al. (3), Hettinga et al. (18) had subjects perform a maximal incremental test, followed by four 4-km trials on different days. The first was used to determine the power outputs to be maintained throughout the remaining time trials, which were performed using various dictated pacing strategies (18). In a randomized order, subjects performed an even, submaximal, or supramaximal pacing strategy (dictated by design) during the beginning 2-km, whereupon they were allowed to self-select their pace for the remaining 2-km while being encouraged to finish as fast as possible (18).

Results revealed a significant increase in total power during the second interval (i.e., 2000-4000-m) during the submaximal (negative split) trial, no significant difference in total power output between the first half and second half of the trial during the even paced trial, and a significant decrease in total power during the latter half of the 4000-km time trail during the supramaximal (positive split) trial (18). Hettinga et al. (18) demonstrated VO_2 significantly increased during the final 2000-m across all three pacing trials. However, anaerobic power significantly increased during the second half of the submaximal paced trial, did not significantly change during the remaining 2000-m during the even trial, and significantly decreased during in the final 2000-m of the supramaximal trial (18). During the final 2000-m of the 4-km time trial, there was a significant HR and [La] increase for all pacing approaches, while RPE significantly increased during the final section of the submaximal and supramaximal trials, with no significant changes during the even paced trial. Results from iEMG revealed significant increases as a percent MVC between the first and second 2000-m intervals between the vastus lateralis and biceps femoris across all three pacing trials with no significant difference for rectus femoris across three trials (18).

Hettinga et al. (18) found similar results as previous studies (3) regarding iEMG for rectus femoris, however, there were striking differences in iEMG data obtained from the VL and BF also investigated in this study. The inclusion of additional muscle groups (as opposed to only RF as in previous studies) was beneficial as previous research has found variation in iEMG patterns among monoarticular (RF) and biarticular muscle (VL, BF) groups (18). Similar to previous investigations (3, 35), muscle fiber recruitment failed to reach 100% throughout trials in, not only the RF, but also VL and BF (18). Accordingly, in Ansley et. al. (3) and St Clair Gibson et. al. (35) the operational definition of peripheral fatigue was determined by stating that peripheral fatigue may only be satisfied if 100% of MVC is achieved prior to failure or fatigue. In Hettinga et al. (18) though, authors indicate it is problematic to use this criterion as (a) it is a different muscle contraction (i.e., isometric vs. isotonic) and (b) averaging iEMG activity over a time period involving cyclic movement will inevitably contain sums of zero; thus negatively impacting an overall average of total muscle recruitment (18). While Hettinga et al. (18) interpreted their results in favor of peripheral fatigue despite sharing similar results seen in a similar

study (3) concerning iEMG response during 4-km time trials; vastly different interpretations have emerged, both warranting merit, but consequently providing overall equivocal evidence for peripheral and CGM regulation. The pitfalls of iEMG interpretation to determine mediating factors of fatigue have been given considerable attention in a recent review by Weir et al. (43). Nonetheless, it is easily observed that similar data are often interpreted multiple, potentially correct ways.

While there were no significant differences in time to completion among the trials, similar to Ansley et al. (3); the results presented by Hettinga et al. (18) were interpreted in favor of a primary peripheral model of fatigue. During their study, Hettinga et al. (18) found that during the first 2000-m of the supramaximal trial blood [La] were highest and, subsequently, was the only trial that revealed a significant loss of power output during the second half of the trial. Furthermore, the submaximal trial had the opposite effect having the lowest [La] and having a significant increase in power output (18). These results were interpreted to suggest peripheral regulation of exercise is involved to prevent "unsustainable metabolic disturbances," which could severely disrupt homeostasis (18).

It is important to mention that Hettinga and colleagues did discuss a possible central influence; however, the idea of central regulation was presented as auxiliary at best. Therefore, as with Ansley (3), we will put forth a plausible interpretation of the data presented by Hettinga et al. (18) suggesting central, as opposed to peripheral, regulation is perhaps an appropriate model.

The lack of significant differences in completion time, despite mandated initial speed (i.e., submaximal, even, supramaximal) lends support to the notion of a central governor or, more specifically, teleoanticipation (38, 42). St Clair Gibson et al. (38) have suggested individuals employ an "internal clock" constantly reassessing progress towards a known end-point (in this case 4-km) by regulating a pre-determined power output and level of perceived exertion (RPE) at completion. Power from Hettinga et al. (18) and RPE suggest that, despite having an enforced increase or decrease in preferred power, the subconscious teleoanticipatory center appropriately up-regulated or down-regulated power during the second half of all trials to preserve homeostasis and subsequently, regulate completion time (38). This is further substantiated by examining [La] in Hettinga et al. (18). It appears that despite varied [La] after the first half of the 4-km (which would have been expected due to the different intensities employed) the ending lactate was strikingly similar. When this evidence is considered in conjunction with consistent RPE and modified power, it seems to suggest the possible presence of a subconscious central regulator potentially assessing peripheral input and, subsequently, successfully regulating exercise performance (9, 24, 36, 38, 39, 41).

CONCLUSION

We propose that, when legitimate and defensible interpretations of data using multiple models of exercise regulation are possible, they should be presented in such a

manner. It seems there are vast discrepancies among researchers supporting either central or peripheral models of fatigue. Subsequently, data are often interpreted in a manner that best supports a preferred model, sometimes disallowing alternative explanations. We feel it is in the best interest of our field to be able to demonstrate flexibility and, consequently, embrace opposing theories to objectively present what seem to be largely unknown mediators of fatigue processes regulating a myriad of human performance variables, in support of all possible theories when this approach is appropriate. Further, we hope that by providing many, but certainly not all, of the "guidelines" that satisfy conditions as either central, peripheral or a CGM and subsequently and successfully applying them to identical data will further illustrate our main intention of this report.

In this article we have presented some, but not all, of the encumbrances associated with fatigue research, and more importantly, data interpretation implicating, in this case, either a CGM or peripheral model of regulation. This article did not serve to present a thorough review of the volumes of literature concerning the disparities between central and peripheral models. It was, however, intended to propose an alternative avenue to interpretation. That is, to abstain from developing *a priori* definitions or biases that may create "blinders" to alternative, yet equally viable data interpretations. There are few studies, to these authors' knowledge, that can be presented in only one manner (e.g., Merton, 1954). Indeed, it is more common than not that explanations using multiple models are possible as was done in this review and in other recent work from our laboratory (15). This certainly does not infer that authors presenting only one side are biased. However, perhaps pre-determined models drive data interpretation when alternate explanations are equally feasible.

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