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INVITED REVIEW

Fatigue development and perceived response during self-paced endurance exercise: state-of-the-art review

Rafael de Almeida Azevedo[1] · Marcos David Silva-Cavalcante[1,2] · Adriano Eduardo Lima-Silva[3] · Romulo Bertuzzi[1]

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Abstract

Performance in self-paced endurance exercises results from continuous fatigue symptom management. While it is suggested that perceived responses and neuromuscular fatigue development may determine variations in exercise intensity, it is uncertain how these fatigue components interact throughout the task. To address the fatigue development in self-paced endurance exercises, the following topics were addressed in the present review: (1) fatigue development during constant-load *vs.* self-paced endurance exercises; (2) central and peripheral fatigue and perceived exertion interconnections throughout the self-paced endurance exercises; and (3) future directions and recommendations. Based on the available literature, it is suggested (1) the work rate variations during a self-paced endurance exercise result in transitions between exercise intensity domains, directly impacting the end-exercise central and peripheral fatigue level when compared to constant-load exercise mode; (2) central and peripheral fatigue, as well as perceived exertion response contribute to exercise intensity regulation at the different stages of the trial. It seems that while neuromuscular fatigue development might be relevant at beginning of the trial, the perceived exertion might interfere in the remaining parts to achieve maximal values only at the finish line; (3) future studies should focus on the mechanisms underpinning fatigue components interactions throughout the task and its influence on exercise intensity variations.

Keywords Central fatigue · Peripheral fatigue · Pacing strategy · Endurance performance

Abbreviations

$[Ca^{2+}]$	Calcium concentration
$[H^+]$	Hydrogen concentration
$[K^+]$	Potassium concentration
[Pi]	Inorganic phosphate concentration
ATP	Adenosine triphosphate
CP	Critical power
CNS	Central nervous system
EMG	Electromyography signal
MLSS	Maximal lactate steady state
MVIC	Maximal voluntary isometric contraction
Qtw	Muscle twitch evoked torque.
RCP	Respiratory compensation point
RPE	Rating of perceived exertion
TT	Time trial
Inverted J shape	Pacing profile in "inverted J" format
J-shape	Pacing profile in "J" format
U-shape	Pacing profile in "U" format
VA	Voluntary activation
$\dot{V}O_2$	Oxygen uptake
$\dot{V}O_{2MAX}$	Maximal oxygen uptake
W'	Curvature constant

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✉ Romulo Bertuzzi
bertuzzi@usp.br

[1] Endurance Sports Research Group (GEDAE-USP), School of Physical Education and Sport, University of Sao Paulo, Av. Prof. Mello Moraes, 65 - Cidade Universitária, São Paulo, SP 05508-030, Brazil

[2] Sport Science Research Group, Post-Graduation Program Nutrition, Physical Activity and Phenotypic Plasticity, Academic Center of Vitoria, Federal University of Pernambuco, Recife, Pernambuco, Brazil

[3] Human Performance Research Group, Federal University of Technology – Parana (UTFPR), Curitiba, Parana, Brazil

Introduction

Performance in self-paced endurance exercise is determined by a complex interplay between psychophysiological and physical capacities, both components ultimately affecting

fatigue development (Abbiss and Laursen 2008; Enoka and Duchateau 2016). For instance, fatigue development has been shown to be directly impacted by task characteristics, such as different amount of muscle mass recruited (i.e. whole-body endurance exercise *vs.* single-leg knee extensor exercise) and contraction mode (i.e. isometric *vs.* dynamic) (Thomas et al. 2018; Weavil et al. 2018). While much of exercise-induced fatigue findings have been extremely relevant to provide mechanistic insights and a basic understanding of fatigue development, the reductionist approach of the majority of the studies designs eliminates the interaction and influence of various biological systems. For this reason, studying endurance exercises which involve large muscle mass and dynamic contractions (e.g. cycling) are of utmost relevance because of its demand in various physiological systems and practical significance (i.e. activities in daily living and sports competition) (Thomas et al. 2018; Weavil and Amann 2019).

The psychophysiological component of fatigue has been characterized by conscious sensations that regulate exercise intensity to preserve performer's integrity (Enoka and Duchateau 2016), which can be characterized by perceptual responses, such as the rating of perceived exertion (RPE) during exercise (Renfree et al. 2014; Smits et al. 2014). Briefly, exercise-induced changes within the muscles and auxiliary musculature (e.g. respiratory muscles) are sent to the CNS and combined with supraspinal information (e.g. emotions, feedforward and previous experiences) resulting in a conscious rating of effort (i.e. RPE) (Borg 1982; Ulmer 1996). On the other hand, the physical fatigue is characterized by the magnitude and/or rate of decline in objective measures of performance (e.g. power, speed, and torque) during and/or after a bout of exercise (Enoka and Duchateau 2016), thus without a direct influence of psychological aspects. Traditionally, the physical component of fatigue is based on the pre- to post-exercise decline in maximal voluntary isometric contraction (MVIC) (Enoka and Duchateau 2016), which the exercise-induced decrease in MVIC can be caused by a reduction in maximal muscle activation (i.e. central fatigue) and/or contractile function (i.e. peripheral fatigue) (Aagaard et al. 2002; Millet et al. 2011). Central fatigue originates from the reduced maximal neural drive from supraspinal and/or spinal pathways to the exercised musculature, which results in decreased voluntary activation (VA) (Gandevia 2001). VA is commonly quantified using the twitch interpolation technique; an exercise-induced decline in VA is a marker of central fatigue (Merton 1954; Gandevia 2001). In turn, peripheral fatigue is caused by alterations in intramuscular processes including metabolite accumulation (e.g. inorganic phosphate [Pi] and hydrogen ions [H^+]) and ion handling kinetics (e.g. extracellular potassium [K^+] accumulation and calcium [Ca^{2+}] handling) (Jones 1996; Hill et al. 2001; Blain et al. 2016). An exercise-induced reduction in evoked muscle torque from supramaximal electrical or magnetic peripheral motor nerve stimulation (Qtw) is commonly used as a marker of peripheral fatigue (Millet et al. 2011).

It is noteworthy that most of the current understanding about central and peripheral fatigue parameters during endurance exercise is derived from constant-load tests performed until task failure (Burnley et al. 2012; Burnley and Jones 2016; Thomas et al. 2016). This exercise mode does not allow variations in exercise intensity, which constrains fatigue characterization to a hermetic environment within a small range of possibilities and differ from a "real-world" situation. On the other hand, during self-paced endurance exercises (e.g. time trials [TT]), performers continually regulate their work rate throughout the task (Abbiss and Laursen 2008; Azevedo et al. 2019). Briefly, laboratory-based TT test mimics a real competitive sports setting, wherein the participant is asked to complete a given distance/duration as fast as possible, while the exercise intensity varies in a self-selected manner (i.e. self-paced) throughout the task. It is noteworthy that most of the current evidences are based on short to middle distance/duration trials (e.g. 1–40 km cycling and/or 3–21 km running races), since long distance/duration (e.g. ultra-endurance races) have received much less attention regarding the effects of fatigue in pacing. Additionally, long distance/duration races might also be influenced by other factors beyond the controlled environment in most of the laboratories settings (e.g. climate, oxygen availability, sleep deprivation, course characteristics) which could affect fatigue etiology (Millet 2011). Nevertheless, it has been suggested that these self-adjustments in exercise intensity throughout a short/middle distance/duration TT occur to avoid premature exercise cessation and development of critical levels of fatigue only at the finish line (Ulmer 1996; Abbiss and Laursen 2008; Azevedo et al. 2019).

For instance, some of the findings related to fatigue development in self-paced endurance exercises of short/middle duration have been restricted to pre- to post-TT assessments, which lack valuable information regarding the interactions between self-adjustments in exercise intensity and fatigue development throughout the trial. However, recent evidences investigating central and peripheral fatigue development throughout a middle-duration TT (i.e. 4 km) have highlighted that exercise intensity variations might be influenced by different fatigue components according to the TT phase (Azevedo et al. 2019). More specifically, while the high intensity adopted in the beginning of the 4 km TT seems to be affected by the rapid central and peripheral fatigue development, the self-adjustments in exercise intensity throughout the remaining TT parts (i.e. middle and end) might be more influenced by perceptual responses, such as the progressive increase in RPE until the finish line but rather stable central and peripheral fatigue levels (Azevedo et al. 2019).

Nevertheless, the underpinning mechanism between the exercise intensity variations and fatigue development might by transient throughout a self-paced endurance exercise, wherein each phase might be influenced by distinct physical and psychophysiological variables.

In the present manuscript, we provided a comprehensive review of the interconnections amongst central and peripheral fatigue development and perceptual responses in exercise intensity variations throughout a self-paced endurance exercise of short/middle distance/duration. Specifically, the present review focused on (1) fatigue development during constant-load exercise and its translation to self-paced endurance exercise; (2) our current understanding of fatigue characteristics self-paced endurance exercise, considering each phase of the task (i.e. begining, middle, and end phases); and (3) future directions for further understanding fatigue development in self-paced endurance exercise. The present information and references were based on PubMed database search for the most relevant literature regarding fatigue in constant-load and self-paced endurance exercises. The searched terms "fatigue", "central fatigue" and "peripheral fatigue" were individually concatenated with "constant-load endurance exercise" and "self-paced endurance exercise".

Fatigue development: from constant-load to self-paced endurance exercise modes

A constant-load test is characterized by performing an exercise until task failure at a pre-determined work rate. Endurance exercise intensity is often categorized into three distinct exercise-intensity domains: moderate, heavy, and severe (Jones et al. 2008; Poole and Jones 2012; Burnley and Jones 2016; Black et al. 2017). The moderate- and heavy-intensity domains are distinguished by metabolic steady-state achievement within ~ 3 and 15 min from exercise onset, respectively, wherein stabilization of oxygen uptake ($\dot{V}O_2$), blood lactate concentration, and intramuscular metabolite levels occur (e.g.[Pi] and [H^+]) (Black et al. 2017). On the other hand, the severe-intensity domain does not present a metabolic steady-state (Poole and Jones 2012) and critical levels of intramuscular metabolites and maximal oxygen uptake ($\dot{V}O_{2max}$) are attained at task failure (Jones et al. 2008; Black et al. 2017). It is noteworthy, that the boundary between the heavy and severe domains can be estimated by distinct methods, such as the respiratory compensation point (RCP), maximal lactate steady state (MLSS) and critical power (CP) model, where the accuracy and reliability of each model have been extensively debated elsewhere (Keir et al. 2015; Jones et al. 2019) and it is beyond the scope of the present manuscript. Since most of the current evidences in the topic have utilized CP (Burnley et al. 2012; Burnley and Jones 2016; Pethick et al. 2020), this model was considered in the current study. Briefly, the CP can be characterized by the highest sustainable exercise intensity for a prolonged period of time and the finite work above this "threshold" relies on intramuscular substrate-level stores, commonly called as W' (Burnley and Jones 2016). Physiological responses in the severe intensity are due to the augmented contribution of substrate-level phosphorylation to the ATP turnover process, leading to faster intramuscular metabolite accumulation, and a four to five-fold shorter time to task failure compared to the heavy-intensity domain (Burnley and Jones 2016; Vanhatalo et al. 2016; Black et al. 2017). In addition, muscle contraction efficiency decreases as exercise intensity increases beyond moderate-intensity domain, as evidenced by a slow rise in $\dot{V}O_2$ for a given workload called "$\dot{V}O_2$ slow component" (Jones et al. 2011). The loss of muscle contraction efficiency within the heavy and severe domains is closely linked with greater recruitment of type II fibers, ultimately increasing the rate of metabolite accumulation and peripheral fatigue development (Keir et al. 2018), thus influencing exercise capacity because of increased metabolic cost for a given workload.

Central and peripheral contributions to fatigue development appear to change in an exercise intensity-dependent manner (Burnley and Jones 2016). Thomas and colleagues (Thomas et al. 2016) compared the level of central and peripheral fatigue following constant-load cycling bouts performed at three different intensities: two within the severe domain (i.e. 100% and ~ 76% of peak power output) and one within the heavy domain (i.e. 64% of peak power output). While the higher exercise intensities resulted in greater peripheral fatigue (i.e. Qtw reduced to − 33%, − 16%, and − 11%, respectively), the lower exercise intensity elicited greater central fatigue (i.e. VA reduced to − 2%, − 6%, and − 9%, respectively). Additionally, similar levels of end-exercise peripheral fatigue have been reported regardless of the relative intensity within the severe-intensity domain (Burnley et al. 2012; Schäfer et al. 2019), which supports the assumption that peripheral fatigue develops more rapidly with increasing exercise intensity until the attainment of critical levels that carries to an interruption of exercise. Indeed, it has been suggested that exercise intensity variations might be regulated by the rate of W' utilization and reconstitution (Jones and Vanhatalo 2017). Briefly, while W' utilization follows a linear decline, its reconstitution is better represented by a curvilinear response, thus reinforcing the assumption of precise management of the finite work capacity above CP throughout a TT until the finish line (Chidnok et al. 2013a). In this context, exercise cessation within the severe domain have been associated with excessive W' utilization regardless of the exercise intensity above CP (Schäfer et al. 2019). Thus, it can be assumed that fatigue development within the heavy-intensity domain

can be attributed to both central and peripheral mechanisms, whereas the severe domain is mainly ascribed to peripheral mechanisms (Burnley et al. 2012; Burnley and Jones 2016; Thomas et al. 2016).

However, during a self-paced endurance exercise, the work rate varies, ultimately resulting in transitions between exercise intensity domains. Conversely, these adjustments may directly impact central and peripheral fatigue developments and overall physiological responses. For example, intramuscular disruptions (i.e.[Pi] and pH) following a constant-load severe-intensity bout to task failure can demonstrate partial recovery if the exercise intensity is reduced to the heavy domain (Chidnok et al. 2013b). Indeed, depending on the length of the TT, self-regulated exercise intensity often shifts from severe (non-metabolic stable) to heavy (metabolic stable) domains (Chidnok et al. 2013a; Black et al. 2015), which could result in a partial recovery of the metabolic disturbance provoked by prior severe-intensity part of the trial, preventing premature exacerbation of peripheral fatigue development. Interestingly, some findings have shown that pacing is directly linked with W' utilization, where regardless the pacing profile adopted (i.e. enforced or self-paced) the work above CP does not change (Chidnok et al. 2013a). Although, it must be acknowledged that calculation of work done within the severe domain during a self-paced exercise has a caveat of boundary estimation since the CP might be downshifted throughout the task (Clark et al. 2018). Additionally, the self-regulation of exercise intensity might also be influenced by the decrease in muscle contraction efficiency, which result from the metabolite accumulation and peripheral fatigue development for a given workload within the heavy and severe domains (Jones et al. 2011; Keir et al. 2018). Indeed, previous findings have shown that muscle contraction efficiency (i.e. estimated by gross efficiency) decreases rather linearly in high intensity and short distance cycling TT (i.e. 4000 m) (Noordhof et al. 2015).

Considering the abovementioned task-dependency characteristic of fatigue, Fig. 1 shows a schematic representation of physiological and central and peripheral fatigue development during each exercise mode (i.e. constant-load

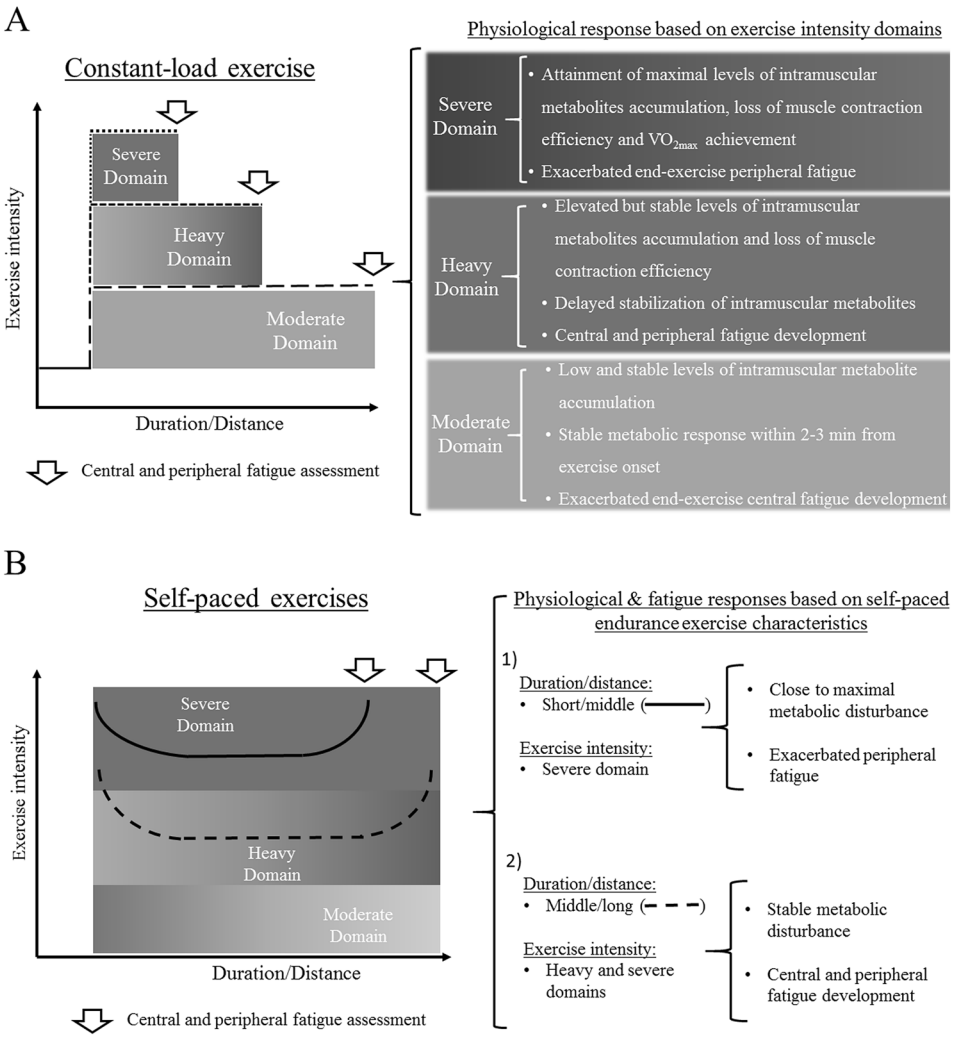

Fig. 1 Relationship between endurance exercise model and fatigue responses. Schematic representation of physiological and fatigue responses at the end of either constant-load or self-paced endurance exercises with distinct intensity domains. Panel **a**: constant-load endurance exercises to task failure within moderate, heavy and severe intensity domains. Note that end-exercise physiological and fatigue responses are distinct depending on the intensity domain utilized. Briefly, from the moderate to severe domains, the time to task failure decreases, which is accompanied by progressive metabolite accumulation and peripheral fatigue development. Panel **b**: end-exercise physiological and fatigue responses in self-paced endurance exercises. Note that depending on the task duration/distance, the overall exercise intensity might be performed amongst domains, which result in a mixture of physiological and fatigue responses. Briefly, as shorter the self-paced endurance task, probably, higher the exercise intensity and greater the metabolic disturbance and peripheral fatigue development

and *vs*. self-paced endurance exercises). In panel A, physiological variables (e.g. $\dot{V}O_2$) are characterized by stable (moderate- and heavy-intensity domains) to a non-stable (severe-intensity domain) responses. While central fatigue development is more exacerbated during moderate to heavy intensity domains, end-exercise peripheral fatigue level is maximal after an exercise performed within the severe-intensity domain. On the other hand, in panel B, depending on the TT duration, exercise intensity will vary between domains, ultimately allowing for partial recovery of metabolite accumulation and slowing down the fatigue development. Nonetheless, previous findings have shown that if the TT distance/duration is short enough to maintain exercise intensity within the severe domain during the entire trial, the overall response will be characterized by exacerbated metabolic disturbance and peripheral fatigue development (Thomas et al. 2015). The following section will explore how exercise intensity variations, also referred as pacing, throughout a TT might vary amongst domains and how it may be linked to central and peripheral fatigue development.

Central and peripheral fatigue during self-paced endurance exercise

Given that changes in exercise intensity across the domains may elicit different metabolic responses, mainly when performed near the boundary between the severe- and heavy-intensity domains, the question that emerges is: Is the central and/or peripheral fatigue development the cause or the consequence of exercise intensity variations throughout a TT?

To address this question, Azevedo and colleagues (Azevedo et al. 2019) performed a comprehensive study evaluating central and peripheral fatigue at different phases of a 4000 m cycling TT. Pacing during this TT length has a parabolic (*U*-shaped) profile, with power residing within the severe-intensity domain at the beginning and at end of the trial, and within the heavy-intensity domain during the middle (Abbiss and Laursen 2008). Thus, VA and Qtw were measured at the end of the fast-start (600 ± 205 m), at the end of the middle portion (3600 ± 190 m) and following the end-spurt (4000 m). Both VA and Qtw decreased immediately after the fast-start phase, indicating an early development of central and peripheral fatigue; however, no further changes occurred during the remaining phases, implying no additional central nor peripheral fatigue until the end of the trial. Interestingly, the attainment of early exacerbated levels of central and peripheral fatigue coincided with an abrupt reduction in muscle recruitment (inferred from electromyography signal, EMG) and consequent reduction in power, demarcating the end of the fast-start phase. Thereafter, both muscle recruitment and power were relatively stable, characterizing an even-paced phase (Azevedo et al. 2019). Power during the middle was self-selected and resided near the boundary between the severe- and heavy-intensity domains, suggesting that exercise intensity was downregulated to a zone wherein muscle recruitment and the rate of metabolite accumulation were not large enough to produce additional central and/or peripheral fatigue (Burnley et al. 2012; Thomas et al. 2016; Black et al. 2017). In this context, previous findings have suggested that self-adjustments in exercise intensity within the severe domain would be also based on depletion of W', where nadir levels would only occur at the finish line (Chidnok et al. 2013a). Thus, the exercise intensity adopted during the fast-start phase was unsustainable and resulted in early central and peripheral fatigue development, demanding a power adjustment to avoid premature exercise cessation (Azevedo et al. 2019).

The physiological mechanism by which pacing is adjusted after an early accumulation of central and peripheral fatigue involves complex interactions between the central nervous system (CNS) and peripheral fatigue mechanisms (Amann 2011; Hureau et al. 2017). It has been suggested that afferent feedback from intramuscular mechano- and metabo-sensitive receptors from multiple ascending pathways (i.e. type III and IV afferent fibers) are largely activated after the accentuated intramuscular perturbations and peripheral fatigue accumulation caused by the fast-start phase (Amann et al. 2009; Blain et al. 2016). Such signals are sent to subcortical and cortical structures (i.e. thalamus, limbic system and prefrontal cortex), which modulate descending motor neural drive (Chaudhuri and Behan 2000; Almeida et al. 2004). Indeed, when afferent feedback from active musculature is pharmacologically blocked (via intrathecal fentanyl), central motor drive during the fast-start phase of a "5,000 m" cycling TT is increased, resulting in exacerbated power and prolonged fast-start phase duration (Amann et al. 2009; Blain et al. 2016). Compared to a control condition, these alterations lead to a significant reduction in power during the last half of the trial and greater end-exercise peripheral fatigue (Amann et al. 2009; Blain et al. 2016). Additionally, a recent study demonstrated that a considerable reduction in pre-exercise Qtw via a stretch–shortening cycle protocol (100 drop jumps), which is believed to increase afferent feedback signals, suppressed the fast-start phase during a "4,000 m" (the equivalent of 4 km) cycling TT (Silva-Cavalcante et al. 2019). Together, these findings suggest that pacing adjustments during the beginning of a trial may be closely regulated by peripheral fatigue development, likely via afferent feedback from active muscles, which limits descending central motor drive.

Perceived responses during self-paced endurance exercise

Given that exercise-related perceptive exertion response is interconnected with central and peripheral fatigue development, ultimately characterizing overall fatigue symptom, an important question that arises is: how do perceived exertion responses and central and peripheral fatigue interact throughout self-paced endurance exercise and determine exercise intensity variations?

Within this context, exercise-related perceived exertion responses reflect changes in the sensations regulating performer integrity (Enoka and Duchateau 2016), wherein the output is based on a combination of sensory inputs (i.e. afferent feedback) and cognitive processes (i.e. assessment of all potential risks and behaviors) (Ulmer 1996; Kluger et al. 2013; Hureau et al. 2017). One of the most accepted perceived exertion parameters during endurance exercise is the RPE response, which represents a summation of signals from afferent feedback (Ulmer 1996; Hureau et al. 2017) and supraspinal mechanisms (e.g. feedforward and corollary discharge from cortical areas related to motor planning) (Ulmer 1996; Marcora 2009; Hureau et al. 2017). The integrative nature of RPE has been extensively demonstrated in self-paced endurance exercise modes, wherein the rate of increase in RPE in percentage of remaining time is constant, regardless of task distance or duration, and the achievement of maximal values occur only at the finish line (Faulkner et al. 2008; Thomas et al. 2015; Froyd et al. 2016). Exercise intensity alterations may thus occur based on a "template" in the rate of RPE increase throughout the trial (Faulkner et al. 2008).

In this scenario, the RPE is anchored to the teleoanticipation concept (Ulmer 1996). Briefly, intensity alterations during a self-paced endurance exercise (e.g. cycling TT) are suggested to have a considerable anticipatory component based on pre-exercise expectations and experiences (Tucker 2009), which also contribute to the decision-making process during the task (Renfree et al. 2014; Smits et al. 2014). RPE responses represent the conscious/verbal manifestation of a pre-programmed strategy (Tucker 2009) and are evidenced by adjustments in exercise intensity based on its template as multiple afferent signals inform fatigue development during the trial (Lambert et al. 2005). In line with these suggestions, an index derived from the product of the momentary RPE and the fraction of the race distance remaining, the so-called Hazard Score, has been suggested to determine the likelihood that athletes will change their pacing and represents the manner by which an anticipatory regulation of exercise intensity occurs (Koning et al. 2011). This index suggests that athletes continuously monitor their present and expected feelings in a given moment. Interestingly, the Hazard Score reaches maximal values (i.e. highest risk of premature fatigue) after ~ 30% of the trial, which coincides with a sudden reduction in exercise intensity characterizing the end of the fast-start phase.

As mentioned in the previous section, at the end of the fast-start phase, there is a significant amount of both central and peripheral fatigue (Azevedo et al. 2019). However, at the end of the fast-start phase, the RPE is relatively low. Indeed, several studies have reported that the performers achieve considerable high values of power at the fast-start phase while reporting the lowest RPE, when compared to the remaining parts of the trial (Thomas et al. 2015; Silva-Cavalcante et al. 2018, 2019; Azevedo et al. 2019). The reasons for this dichotomic response between the central and peripheral fatigue and RPE responses is not fully understood, but it might be related to the delay in afferent feedback from the active musculature to RPE processing within the CNS. During the fast-start phase of a TT, the time required to achieve stabilization in the intramuscular metabolites and other physiological variables (e.g. VO_2) related to perceived exertion (i.e. RPE) is greater (> 10 min, depending on the domain) than the duration of this phase of the trial (~ 60 s) (Whipp 1994; Xu and Rhodes 1999; Jones et al. 2008; Azevedo et al. 2019). This temporal misalignment could produce delayed RPE processing, despite a considerable central and peripheral fatigue level (Azevedo et al. 2019). In turn, there is an alignment between the early development of central and peripheral fatigue level with high Hazard Score at the beginning of the trial, which represents a higher perceived risk of premature exercise cessation before the finish line (Fig. 2).

Another intriguing point resides in the final portion of a TT, wherein an "end-spurt" (i.e. final sprint) is frequently reported (St Clair Gibson et al. 2001; Noakes and Gibson 2004; Noakes et al. 2005). The "end-spurt" appearance challenges the notion that central and peripheral fatigue solely influences exercise intensity variations, as an increase in muscle recruitment and power occur even under considerable levels of central and peripheral fatigue (St Clair Gibson et al. 2001; Noakes and Gibson 2004; Noakes et al. 2005). In other words, a brief high-intensity exercise is performed even when central and peripheral fatigue and physiological parameters are near maximal levels. This phenomenon is consistently evidenced during TTs performed under distinct environmental conditions and with different distances/durations (Amann et al. 2006; Thomas et al. 2015), highlighting a conscious decision to increase exercise intensity regardless of circumstances. An end-spurt initiates when the risk of premature exercise cessation, measured via Hazard Score, is low (Ulmer 1996; Koning et al. 2011). Interestingly, some authors have suggested that the end-spurt occurrence might

Fig. 2 Schematic representation of fatigue components responses throughout a self-paced endurance exercise. Note that the self-paced endurance exercise was divided according to the exercise intensity variations, which were performed within different domains. The open arrows indicate central and peripheral fatigue assessments at the end of each phase, wherein their time course change is represented on the righthand side of the figure. Concomitantly, the time course of perceived responses (i.e. rating of perceived exertion and hazard scores) are also represented throughout each phase of the task (i.e. at the bottom righthand side)

also be based on the amount of W' available until the finish line, where this information would also be taken into account for the RPE response and teleoantecipation model (Ulmer 1996; Jones and Vanhatalo 2017) thus, the end-spurt occurrence may be a decision-making process based on the low likelihood of premature exercise cessation.

Taken together, it is evident that the perceived exertion responses are also determinant of exercise intensity variations throughout a self-paced endurance task, rather than solely central and peripheral fatigue development. As shown in Fig. 2, even though the precise mechanism which perceived exertion and central and peripheral fatigue interact is not fully understood, it is suggested that rapid peripheral fatigue development at the beginning of the TT might affect perceived responses (i.e. Hazard Score) through afferent feedback to the CNS, ultimately downregulating exercise intensity. On the other extreme, the "end-spurt occurrence" suggests a conscious decision to increase the exercise intensity despite high levels of peripheral fatigue, which might be explained by the low perceived risk of premature exercise cessation.

Future directions for characterization of fatigue development during self-paced endurance exercise

Despite of the relevance of self-paced endurance exercise, a limited number of studies have examined the connection between perceived responses and central and peripheral fatigue development during this exercise mode. To improve our understanding, simple recommendations for future studies must be provided.

The majority of previous studies using self-paced endurance exercise did not report the time expended within each exercise intensity domain. This point is relevant since end-exercise central and peripheral fatigue levels are profoundly impacted by the metabolic demand imposed during the task (Burnley et al. 2012; Thomas et al. 2016; Black et al. 2017). In addition, different pacing profiles within the same task (e.g. "U"-, "J"-, or "inverted J"-shape) can potentially influence the overall metabolic demand by modulating the time spent within different exercise intensity domains (Hettinga et al. 2006) and, consequently, fatigue development. Future studies are encouraged to identify the exercise intensity domain(s) in which each phase of the trial was performed and, preferably, report the time expended at each domain. To have more insights regarding the interaction between exercise intensity variations and fatigue development, central and peripheral fatigue should be assessed, whenever possible, during different phases of a TT rather than only at post-exercise.

From an integrative perspective, it is important to understand the interconnection between central and peripheral fatigue and perceived exertion response during self-paced endurance exercise. Thus, interventions focused on manipulating perceived responses, such as cognitive task (Silva-Cavalcante et al. 2018) and/or non-localized exercise-induced peripheral fatigue before the TT (Bertuzzi et al. 2020) provide interesting insights about the interconnection

between central and peripheral fatigue development and perceived effort during TT. Moreover, it is unknown if the decision-making nature of self-paced endurance exercises (Renfree et al. 2014; Smits et al. 2014) could exacerbate perceived responses due to, potential, higher CNS demand compared to constant-load endurance exercises. For example, external decision-making stressors (i.e. high cognitive task) while exercising elicit greater perceptual responses (i.e. stress and anxiety) resulting in shorter time to task failure compared to control condition (Yoon et al. 2009). However, it is currently unknown if the decision-making process in self-paced endurance exercises would also affect central fatigue development compared to constant-load exercise mode.

Conclusion

Fatigue development during self-paced endurance exercise results from a complex interaction between physical and psychophysiological components, ultimately influencing exercise intensity alterations. It is plausible to suggest that while central and peripheral fatigue may largely contribute to exercise intensity regulation at the beginning of a self-paced task, perceived responses may dictate exercise intensity at the end of the trial to achieve maximal exertion only at the finish line. It must be highlighted that more empirical data characterizing physical and psychophysiological components of fatigue at different moments/phases of a self-paced endurance exercise is needed.

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Compliance with ethical standards

Conflict of interest The authors declare there are no competing interests.

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