Sports Medicine (2018) 48:2041–2051
https://doi.org/10.1007/s40279-018-0946-9

REVIEW ARTICLE

Mental Fatigue Impairs Endurance Performance: A Physiological Explanation

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Published online: 19 June 2018

Abstract

Mental fatigue reflects a change in psychobiological state, caused by prolonged periods of demanding cognitive activity. It has been well documented that mental fatigue impairs cognitive performance; however, more recently, it has been demonstrated that endurance performance is also impaired by mental fatigue. The mechanism behind the detrimental effect of mental fatigue on endurance performance is poorly understood. Variables traditionally believed to limit endurance performance, such as heart rate, lactate accumulation and neuromuscular function, are unaffected by mental fatigue. Rather, it has been suggested that the negative impact of mental fatigue on endurance performance is primarily mediated by the greater perception of effort experienced by mentally fatigued participants. Pageaux et al. (Eur J Appl Physiol 114(5):1095–1105, 2014) first proposed that prolonged performance of a demanding cognitive task increases cerebral adenosine accumulation and that this accumulation may lead to the higher perception of effort experienced during subsequent endurance performance. This theoretical review looks at evidence to support and extend this hypothesis.

Key Points

Mental fatigue impairs endurance performance, mediated primarily by an increase in perception of effort. The mechanism by which this occurs, however, is currently unclear.

We propose that with demanding cognitive activity, extracellular cerebral adenosine accumulates within active regions of the brain. We further propose that adenosine acts in two ways: by increasing perception of effort during subsequent effortful tasks, and by impairing motivation, or the willingness to exert effort, likely via an interaction with dopamine in the anterior cingulate cortex.

During an endurance test, both perception of effort and motivation can influence performance. We contend that any manipulation by which the accumulation of adenosine during mental exertion is reduced would minimise the impact of mental fatigue on subsequent endurance performance via these mechanisms.

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1 Introduction

Mental fatigue reflects a change in psychobiological state, caused by prolonged periods of demanding cognitive activity [1]. This change is gradual and cumulative and has subjective and objective manifestations including increased resistance against further effort [2], changes in mood [3] and feelings of 'tiredness' and 'lack of energy'. It is understood that mental fatigue impairs cognitive performance [4–6]; however, only more recently has it been demonstrated that aspects of physical performance are also impaired by mental fatigue [7]. Time to exhaustion during both high-intensity cycling [1] and a sustained isometric leg extension [8] were reduced following a mentally fatiguing task, and average running speed was slower during a 5-km treadmill [9] and a 3-km track running time trial [10]. Other measures of endurance performance are also impaired with mental fatigue, such as the low intensity component of an intermittent running protocol [11], and the distance completed during the Yo-Yo Intermittent Recovery test [12].

The mechanism behind the detrimental effect of mental fatigue on endurance performance is poorly understood. Variables traditionally believed to limit endurance performance, such as heart rate, lactate accumulation and neuromuscular function, are unaffected by mental fatigue [1, 8, 13]. Rather, it has been suggested that the negative impact of mental fatigue on endurance performance is primarily mediated by the greater perceived exertion experienced by mentally fatigued participants [14]. Whilst we acknowledge the contention surrounding the interchangeable use of perception of effort and perceived exertion within the literature [15, 16], this review uses the terms synonymously. In exercise science, perception of effort is defined as the conscious sensation of how hard, heavy and strenuous a physical task is [17]. Perception of effort can be quantified using psychophysical scales [15], most commonly, the rating of perceived exertion (RPE) scale, a linear scale ranging from 6 to 20, anchored by the descriptors 'no exertion at all' to 'maximal exertion' [18]. During a time-to-exhaustion test the impact of higher perceived exertion is demonstrated by participants reaching a terminal RPE more quickly and disengaging from the task earlier [1, 8]. In a time-trial setting, the average power output or speed produced for the same RPE is lower [9, 10].

One model that highlights the role of perceived exertion during exercise regulation, and therefore has been used to explain the impact of mental fatigue on endurance performance, is the psychobiological model [19, 20]. This model, based on motivational intensity theory [21], proposes that performance throughout a constant-load power test is primarily determined by the interaction between perceived exertion and potential motivation [22]. Potential motivation is the greatest amount of effort a person is willing to exert to succeed in a task [23]. During this type of endurance test, when the effort required by the test is perceived to exceed potential motivation, or when perception of effort is so extreme that continuing seems impossible, the person consciously decides to stop exercising, or downregulates their level of effort. According to this model, any factor that influences perceived exertion and/or potential motivation influences endurance performance, even when the physiological capacity to perform the task is unchanged. It appears the negative impact of mental fatigue on endurance performance is mediated by an increase in perceived exertion [14]; however, the mechanism by which mental fatigue increases perceived exertion is still unclear. In 2014, Pageaux and colleagues were the first to propose that prolonged performance of a demanding cognitive task may increase cerebral adenosine accumulation and that this accumulation could contribute to the higher perceived exertion experienced during subsequent endurance performance [9, 13, 14, 24]. This theoretical review considers evidence to support and extend this hypothesis. Figure 1 provides a schematic representation of the proposed mechanism.

2 A Neurochemical Perspective

Although there are many potential contributors to fatigue during endurance performance, the neuromodulator adenosine is a promising candidate, particularly with regard to fatigue arising from mental exertion. Here we define fatigue as "difficulty in initiation of or sustaining voluntary activities" [25], which results most commonly in a sporting or exercise performance context in a reduction in physical output, observable in self-paced activity, or the cessation of exercise, when work-rate is dictated externally. Adenosine is known to accumulate within the brain during periods of wakefulness, before it dissipates with sleep [26, 27]; it also accumulates during intense physical exercise [28], and likely during effortful cognitive activity (see Sect. 3). The role of adenosine in these fatiguing contexts is supported by the ergogenic effect of the potent adenosine antagonist caffeine on cognitive [29] and sporting performance [30],

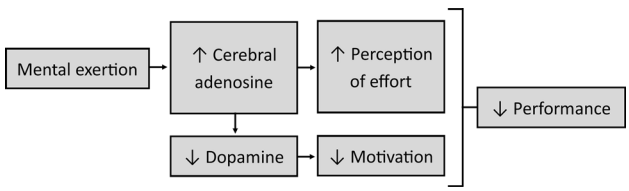

Fig. 1 Schematic representation of the proposed mechanism

and in reducing homeostatic sleep drive [31]. Fluctuations in basal adenosine are also believed to be linked to cerebral fuel [32], which is further discussed in Sect. 3. Adenosine acts through its neuromodulatory role, which typically inhibits its neural activity via inhibition of presynaptic neurotransmitter release [33] (including dopamine [34]) or hyperpolarisation postsynaptically [35]. Both actions are thought to occur through the A_1 receptor, one of the most abundant receptors in the brain [36]. Adenosine can also stimulate synaptic activity through the A_{2A} receptor, although this receptor is much less common than the A_1 receptor in most brain regions [32], and may be more involved in immune or circulatory functions [37].

Adenosine is ubiquitous, thanks largely to the fact that adenosine triphosphate (ATP) is produced by all cells. Adenosine is present in the extracellular space, including the synaptic cleft, due to breakdown of ATP, having been released directly, or conjointly with exocytosis [36]. Under certain conditions, where adenosine accumulates in the intracellular components (as seen in models of energy restriction [38]), adenosine may also be pumped directly into the extracellular space through specific channels [36]. While extracellular adenosine is rapidly cleared through metabolism or uptake into surrounding cells, endogenous release appears to be sufficient to exert its tonic inhibitory influence [39]. Estimates of resting extracellular adenosine vary between 10 and 30 nmol/L and 1 and 2 µmol/L, but can increase over 20-fold during different forms of stress, such as during energy restriction [40]. Although much of the research has focussed on basal levels of adenosine in modulating synaptic activity, it was recently discovered that following stimulated release, rapid transients of adenosine exist, lasting for from a few seconds up to ~30 s. These rapid adenosine transients are capable of modulating neural activity, including the inhibition of dopamine release via A_1 receptors [41]. The clearance rate of these rapid adenosine transients also appears to be influenced by the local metabolic state [42]. The existence and action of rapid adenosine transients is important in terms of mental fatigue. For instance, rapid adenosine transients may account for the fast recovery (over a few seconds) of performance in a cognitive task when an individual is interrupted from the task for a brief period. Traditional understandings of mental fatigue tend to centre on the relatively slow accumulation of fatigue over minutes or hours, and so the potential for rapid transients to influence performance should be kept in mind. A further complication when considering the role of adenosine in fatigue, particularly fatigue arising from mental exertion, is that adenosine receptor availability differs between individuals. It has been observed that individuals upregulate A_1 receptors differently after sleep deprivation, and the relative upregulation of these receptors has been able to distinguish between levels of sleepiness and cognitive performance [43]. Whether this variability is associated with a genetic predisposition or trainability remains uncertain at this stage.

One brain region in which the actions of adenosine have been suggested to be particularly important in the context of mental fatigue is the anterior cingulate cortex (ACC) [13]. Both the continuous performance test (AX-CPT) [44] and the Stroop task [45]—cognitive tasks commonly employed to experimentally induce mental fatigue—are associated with activity in the ACC. This brain region is involved in effortful mental processes such as emotional processing and control [46], self-regulation [47] and performance monitoring [44], as well as appearing to be important in effort/reward processing [48], persevering with a task [49] and perceived exertion during endurance exercise [50]. Thus, with prolonged performance of a demanding cognitive task, such as the Stroop task or AX-CPT, an increase in local adenosine concentration is likely. Localised adenosine levels are also likely to rise further with the performance of a subsequent effortful cognitive task, or prolonged endurance test. One action of adenosine is to modulate the release of a number of neurotransmitters, including inhibition of the release of dopamine (see Sect. 5) [34]. Although adenosine may act across many brain regions, the presence of dopamine receptors in the ACC, and their role in regulating effort-based decision making [51], seems a further, particularly intuitive, means by which adenosine could modulate effort-related fatigue. The remainder of this review focuses on the role of adenosine, with specific reference to fatigue brought about by prolonged mental exertion.

3 Adenosine is Increased with Mental Exertion

Various physiological manipulations can increase extracellular adenosine [32]. Manipulations that cause the energy requirements of the brain to outstrip its ability to synthesize ATP, such as hypoglycaemia [52], hypoxia [53] and electrical stimulation [54], have all been shown to profoundly increase adenosine release. Brain metabolic activity increases from baseline with the performance of a cognitive task [55, 56], and greater activation is associated with greater task-specific cognitive demand [57]. Increased activation of the ACC [45] and right superior frontal region [58] have been observed with performance of an incongruent versus a congruent version of the Stroop task, with comparable results found within the prefrontal and parietal cortices for tasks of working memory [57, 59]. While assessment of adenosine within the human brain, particularly during cognitive or physical performance, is problematic, studies of rat brain slices have shown the formation and release of adenosine with electrical stimulation [53, 60, 61], moderate hypoglycaemia [52] and glycolytic inhibition [62, 63].

Before continuing, it must be emphasized that a model reliant on adenosine is not the same as one reliant on a limited physiological resource, despite clearly being related to localized cerebral fuel availability. In fact, we support the critique of the resource model of self-control [64–66], and wish to highlight where the hypothesis proposed in the present review diverges from this model. Mental fatigue research supports the statements made by the resource model in that performance of an initial task of self-control will impair performance on subsequent tasks of self-control, and that this effect can cross domains. We further support the notion that regular exertion of self-control, whether by taking a mentally challenging class or by participating regularly in effortful physical activity, can 'train' or improve self-control (see Sect. 7). As argued by others previously [67–69], we find the improbability of the resource model to arise from the belief that a common and depletable resource, namely glucose [70], fuels self-control and that deficits in self-control are mediated by depletion or a reduction of this fuel source. A reduction in blood glucose is not consistently observed with tasks of self-control [69, 71], nor are reports of performance restoration with glucose or carbohydrate supplementation [71–74]. Furthermore, a model based purely on a depletable physiological resource would not explain the performance benefits obtained from psychological strategies such as motivational self-talk [75], financial incentives [76, 77] and deception [78]. While these studies disprove glucose as the primary mediator of self-control, the idea that a cerebral fuel source may contribute to impaired physical performance following mental exertion cannot be disregarded completely. Often arguments discounting the role of brain fuels are based on whole brain metabolism measures [79], or make the assumption that peripheral blood glucose concentration accurately reflects cerebral metabolism [71]. A micro-dialysis study in rats assessed the glucose levels in hippocampal and striatum extracellular fluid (ECF) pre, during and post a spontaneous alternation task [80]. The level of glucose in the hippocampal ECF decreased sharply upon commencement of testing, by as much as 30% below baseline, and remained decreased for the duration of the task. At the same time, no decrease in striatal ECF glucose was observed. During this task, systemic blood glucose concentration was also analysed, increasing by up to 123% of baseline following completion of the task. This study highlights that glucose is consumed with mental exertion, but that this effect is localised in certain active regions of the brain. Although we propose that mental exertion will cause localised changes in cerebral fuel stores, which in turn can contribute to changes in cerebral adenosine concentration, we contend that it is the effect of adenosine on perception of effort (see Sects. 4 and 5) and motivation (Sect. 6) rather than a depletion in fuel that mediates changes in behaviour. Performing a prolonged and demanding cognitive task would conceivably both increase neuronal activation and reduce localized cerebral fuel availability; therefore, it is likely that in this scenario cerebral concentrations of adenosine would also increase.

4 Adenosine Increases Perception of Effort

The accumulation of adenosine with mental exertion is hypothesized to contribute to the increase in perceived exertion experienced by mentally fatigued participants [9]. There has, however, been no direct evaluation of the effect of adenosine on perceived exertion and fatigue during exercise performance in humans. While we acknowledge that it is not possible to directly quantify perceived exertion in animals, manipulations of adenosine agonists and antagonists profoundly affect effort-related choice behaviour and appear to make animals more sensitive to the work requirements of a task. In a feeding procedure in which rats could choose between pressing a lever for a preferred food or consuming readily available but less preferred lab chow, systemic injections of an adenosine agonist reduced the number of lever presses for the preferred food, but did not affect overall food intake [81]. Intracranial injections of an adenosine agonist reduced total responses to a task requiring a high level of effort to obtain a food reward [82], and intracerebroventricular administration of an adenosine receptor agonist reduced run time to exhaustion compared to the control condition, and without a change in any other variable [83].

Aside from periods of demanding mental exertion, adenosine is also proposed to accumulate during wakefulness, most notably in sleep deprivation. Direct measurement of adenosine in the forebrain of cats supports this hypothesis with extracellular adenosine progressively increasing during wakefulness and decreasing during subsequent recovery sleep [26]. In human studies, the effects of sleep deprivation on endurance performance are similar to those observed with mental fatigue. Without sleep, perceived exertion increases and endurance performance is worse, with no change in the peripheral physiological variables reported [84–86]. In humans, indirect support for the role of adenosine in the modulation of perceived exertion is found in studies involving the ingestion of caffeine. Caffeine is very similar in structure to adenosine and can bind to cell membrane receptors for adenosine, thus blocking their action [87]. Caffeine easily crosses the blood-brain barrier due to its lipophilic properties [88] and has been shown to counteract most of the inhibitory effects of adenosine on neuro-excitability [89], neurotransmitter release [90] and arousal [26]. The consumption of caffeine has been reported to reduce perceived exertion and improve endurance performance in both sleep-deprived [91] and mentally-fatigued participants [24]. Caffeine has also been reported to improve alertness and mood

[92], and combined caffeine and carbohydrate ingestion has been reported to lessen subjective mental fatigue during prolonged mental exertion [93]. In an exercise context, ingestion of caffeine reduced perceived exertion and increased power output during high-intensity efforts in cyclists [94], and in sleep-deprived participants, caffeine reduced perceived exertion and improved time to exhaustion back to baseline levels [91]. Specific to mental fatigue, cycling time to exhaustion was longest when mentally-fatigued participants consumed caffeine, compared to a placebo or control condition (being mentally-fatigued only) [24]. Compared to the placebo, perceived exertion was reported as lower during the early stages of the test following caffeine consumption [24].

5 Generation of Perceived Exertion

Despite the obvious importance of perceived exertion in the regulation of endurance performance, particularly when mentally fatigued, it is not understood exactly how perceived exertion is generated. It is currently proposed that perceived exertion is related to the activity within various regions of the motor cortex, including the premotor and primary motor areas [95]. Indeed, the corollary discharge theory postulates that an efference copy of the central motor command is sent directly from motor to sensory areas of the brain in order to assist in the generation of perceptions associated with motor output [96–99]. With increasing exercise intensity or muscle fatigue, a greater number of motor units are recruited and firing frequency increases, as does the number of efferent copies received by sensory regions within the brain [95, 100]. As it is an inhibitory neuromodulator, we consider that localised accumulation of adenosine creates a scenario in which a greater stimulatory input is required to produce a motor output. This stimulatory signal presumably arises from motivational and other higher brain centres. We contend that it could be the increased stimulatory requirements that, as may occur with mental exertion, translates into an increase in perceived exertion. Supporting this proposition, ingestion of caffeine reduced RPE during submaximal isometric knee extensions, and reduced activity of premotor and motor areas of the cortex, reflected in the amplitude of motor-related cortical potential (MRCP) [98]. These changes in RPE and MRCP occurred without significant change in muscle activation and force output. The explanation for the caffeine-induced reduction in MRCP during muscle contraction was suggested to be increased central nervous system (CNS) excitability [101, 102], thereby requiring less activity in premotor and motor areas of the cortex to produce the same degree of muscle activation after caffeine ingestion. Considering these findings in another way, the lesser premotor and motor area activity required for the muscle contraction could also be explained by a caffeine-induced reduction in inhibition. Studies of transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) also appear to support this model of perceived exertion. A study of tDCS, with the anodal electrode placed over the left motor cortex and cathodal electrode above the shoulder, improved time to exhaustion during an isometric leg extension compared to placebo or control [103]. The improvement in time to exhaustion was paralleled by a reduction in RPE, and it was proposed that the stimulation reduced the threshold required for the firing of the descending motor drive. In an opposing manner, but likely via similar mechanisms, TMS to reduce primary motor cortex excitability increased perceived exertion during a force-matching, grip force task [104].

It is proposed that a primary site of this neuromodulation is the ACC [13]. As mentioned in Sect. 2, this brain region is involved in both effortful cognitive activities [44–47] and perception of effort during exercise [50]. A reduction in task-related brain activity has also been shown in the ACC following either cognitive or motor skills training in young adults [105], potentially providing a partial explanation for the lower perceived exertion reported by experienced compared to less experienced athletes on a particular exercise task [106]. This may also contribute to the reduced impact of mental exertion on a subsequent cycling time trial in professional cyclists compared to recreational cyclists [107]. Lastly, the ACC has been implicated in cost/reward decision making relating to the expenditure of effort [48, 108], such as the downregulation of power output that occurs during a time trial in a mentally fatigued person [9, 10]. While we focus on the ACC as the current strongest candidate for the neuroanatomical basis of the increase in perceived exertion with mental fatigue, it would be ill-considered to suggest that other cortical regions are not also involved. For example, the insular cortex has been suggested to be associated with changes in perception of effort during exercise both under hypnosis [50] and during conscious exercise [109]. When mental exertion is effortful, however, we propose that adenosine accumulates within the ACC. The inhibitory influence of adenosine means greater stimulatory input is needed to produce the required force output, the corollary discharge is subsequently increased, and perceived exertion is therefore greater.

6 Adenosine Impairs Motivated Behaviours

With reference to the psychobiological model, we further contend that the negative effect of adenosine on endurance performance is twofold, affecting not only a person's perceived exertion during a challenging task, but also their motivation. Although a central component of mental fatigue is described as an "increased resistance to further effort" and

a "decrease in the level of commitment to the task at hand" [2], few studies within the mental fatigue and physical performance literature have reported a reduction in self-reported motivation [76, 110]. Within experimental research, motivation is extremely difficult to accurately and objectively quantify, and the authors believe that currently no appropriate self-report measure of motivation is available. Self-reported motivation data are confounded by both the nature of voluntary participation in research and social desirability bias, whereby it is likely that participants will avoid reporting low motivation (these being potential limitations of a range of self-report data). In the case of mental fatigue, when motivation is experimentally manipulated in the form of a financial incentive, mentally fatigued participants perform better on tests of physical endurance than they do when no incentive is offered [76, 111]. Furthermore, financial incentives do not have a significant effect on the endurance performance of participants who are not mentally fatigued, suggesting that within this participant sample manipulating motivation was only advantageous when they were mentally fatigued, and that further benefit beyond baseline cannot be achieved [76]. Outside of the physical performance literature, increasing task motivation has also restored cognitive performance following previous mental exertion [77]. Changes in motivation may also be reflected by changes in the reported RPE. For example, the presence of an attractive female observer reduced RPE in males, compared to no observer, or the presence of a male observer [112]. The authors suggested that, for the male participants, the motivation to portray physical competence may have been increased compared to when an observer of the same sex was present. Similarly, the presence of a competitive avatar during a cycling time trial increased power output compared to cycling alone, with no difference in perceived exertion [113]. Although the reported 'motivation to perform' did not differ between conditions, all participants expressed a wish to 'beat the competition' [113]. Similar findings have also been observed in studies of deception [78, 114, 115]. These studies suggest that changes in motivation may influence the reported RPE, and further highlight that the current methods of quantifying motivation may be limited.

Aside from highlighting the potential flaws in the current tools used to quantify motivation, we suggest adenosine could impair motivation due to its ability to inhibit the release of dopamine [34], as well as its ability to modify the affinity of dopamine receptor binding [116]. Activation of adenosine receptors has been linked to inhibition of neuronal firing [117], inhibition of neurotransmitter release [118] and decreasing locomotor activity [119]. Adenosine injected into the brains of rats also changes their behaviour similarly to mentally fatigued participants, in that they shy away from tasks that are effortful. Injection of an adenosine agonist into the nucleus accumbens of rats produces effects similar to those observed with dopamine depletion or antagonism [81]. Dopamine antagonism causes rats to reallocate their behaviour away from food-reinforced tasks that have high work requirements towards less effortful types of food seeking [120]. As mentioned in Sect. 2, one brain region where an interaction between adenosine and dopamine may be particularly important for mental fatigue is the ACC. Adenosine [121] and dopamine receptors [122] are both found within this region, and manipulations of D_1 receptors have shown to effect effort-based decision making in rats [51].

7 Could Training Preserve Cerebral Fuel and Reduce Adenosine Accumulation?

An exception to the consistent reporting of a reduction in endurance performance with mental fatigue is the unaffected time trial performance of professional road cyclists following 30 min of mental exertion [107]. In this study, professional and recreational cyclists were exposed to an identical cognitive task and experimental procedures. During the performance of a subsequent cycling time trial, the recreational cyclists recorded a lower mean power output and a slower average speed in the mental exertion condition, whereas the endurance performance of the professional cyclists was unchanged [107]. Although the mental fatigue manipulation may seem short, 30 min of performance of the same cognitive task has also impaired time trial performance in recreational runners [9]. It is plausible that training-induced adaptations, which would conserve cerebral fuel availability (and thereby minimise accumulation of extracellular adenosine), would enable the professional cyclists to tolerate a greater cognitive load before experiencing the detrimental effects on endurance performance. In rats, cerebral glycogen supercompensation has been observed following as little as 4 weeks of endurance training [123]. The human brain also appears to have capacity for adaptation with an acute increase in cerebral glycogen stores following a single bout of insulin-induced moderate hypoglycaemia [124]. Supercompensation of cerebral glycogen has also been shown in rainbow trout following 10 days of fasting [125], in mice following insulin-induced hypoglycaemia [126], as well as transient forebrain ischaemia [127]. Adenosine could also be reduced via an increase in the efficiency of neuronal processing, with enhanced neural efficiency observed in athletes performing at higher levels, assessed via both electroencephalography [128] and functional magnetic resonance imaging [129]. Increased neuronal efficiency would lead to greater conservation of cerebral fuel, similar to the preservation of skeletal muscle glycogen observed with changes in movement economy [130]. Any mechanism by which a greater supply of cerebral fuel is available is likely to minimize the accumulation of adenosine with mental exertion,

and training associated with performance mastery appears to achieve this in a number of ways.

8 Recommendations for Future Research

The aim of this theoretical review was to present evidence to support and extend the hypothesis, originally proposed by Pageaux and colleagues in 2014, that adenosine plays a critical role in the impaired endurance performance of mentally fatigued persons. We acknowledge that adenosine may be only one of several factors that contribute to fatigue; however, in the case of mental fatigue, with other peripheral physiological variables ruled out, we propose the adenosine hypothesis as a promising explanation. This review only proposes a hypothesis that is yet to be experimentally tested. Nevertheless, hypotheses such as these are important as they often lead to direct testing of theories, explanations and thus understanding, which will allow the literature in the area to move forward. To help direct this future research, a small number of recommendations have been provided below.

Firstly, a major limitation of mental fatigue and physical performance research is the way in which we quantify motivation. As discussed in Sect. 6, any time participants are asked to self-report motivation it is likely that their response will be biased through the concept of social desirability [131]. Although voluntary, when a participant agrees to take part in a research project it is expected that they will give the task their best effort. In the case of reporting task motivation, little can be done to evaluate if the reported value is truthful, whether that be a conscious or unconscious decision [132]. One method that has been suggested to reduce or detect the presence of response bias is to obtain measures of the predictor and criterion variable from different sources [133]. Regarding motivation, self-report is the only measure available. Perception of effort on the other hand, as an example of another self-report measure, can be obtained directly from the participant, but can also be associated indirectly with performance outcomes. For example, an RPE of 11 (light) would be viewed as questionable at the completion of a maximal incremental cycling task. Although all self-report data are susceptible to bias, measurements that can be obtained from a secondary source, and thus have increased transparency, are likely to be less prone to falsification. Furthermore, social desirability has previously been reported to bias reported motivation to undertake work on an online crowdsourcing service [134], as well as reported goal orientation in young athletes [135]. As currently no other measure of motivation is available, it is difficult to make any recommendation other than to consider self-reported motivation carefully. We suggest that experiments that manipulate motivation in mentally fatigued participants [76, 111] are currently the best approach to evaluating the impact of mental exertion on motivation.

Secondly, it must be considered that mental fatigue may not only arise from the performance of a prolonged and demanding cognitive task. Common regions of the brain appear to be active during mentally effortful tasks, whether that be a cognitive task such as the Stroop task, controlling one's emotions, or maintaining focus during a long endurance race. By examining effort more globally, as opposed to simply thinking of mental fatigue as a state arising from the performance of a demanding cognitive task, we may uncover further linkages between effortful tasks and how they are regulated.

Finally, although direct assessment of adenosine concentration during prolonged mental and physical exertion in humans would be ideal, at present we have not developed the technology to permit such an approach. Pharmacological manipulation may serve as one alternate method by which we can investigate neurochemical changes with mental fatigue. Substances that inhibit or promote adenosine, as well as inhibit or promote dopamine, may be used during exercise performance in mentally fatigued and non-mentally fatigued participants. Manipulations of diet and feeding prior to, or post, mental exertion may also be used to determine any potential protective role of increased cerebral fuel availability on the negative effects of mental fatigue on endurance performance.

9 Conclusion

The findings presented in the current theoretical review provide a physiological rationale for the impairment of endurance performance undertaken in a state of mental fatigue. We propose that extracellular cerebral adenosine accumulates due to the greater neuronal activity required by a demanding cognitive task, as well as through a reduction in local fuel availability. Adenosine then acts in two ways: increasing perceived exertion during subsequent endurance exercise, and impairing motivation to expend effort, most likely in the ACC. While this review focuses on the role of adenosine, dopamine and glucose/glycogen in the impact of mental fatigue on endurance performance, it is likely that other fuel sources, neurotransmitters and peripheral mechanisms also contribute [136–138]. Nonetheless, by narrowing our focus to these particular facets we are able to present our view on what is a central aspect of this important and complex psychophysiological phenomenon. For it is not until we understand how mental fatigue impairs endurance performance that we can best find ways to combat it.

Acknowledgements The authors would like to acknowledge the anonymous reviewers who contributed to earlier versions of this manuscript and improved both the quality and the content of the article.

Compliance with Ethics Standards

Funding No sources of funding were used to assist in the preparation of this article.

Conflicts of interest Kristy Martin, Romain Meeusen, Richard Keegan, Kevin G. Thompson and Ben Rattray declare that they have no conflicts of interest relevant to the content of this review.

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