**Cognitive fatigue effects on physical performance: a systematic review and meta-analysis**

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Abstract

Recent research has examined the effect that undertaking a cognitively fatiguing task for ≤ 90 minutes has on subsequent physical performance. Cognitive fatigue is claimed to affect subsequent physical performance by inducing energy depletion in the brain, depletion of brain catecholamine neurotransmitters or changes in motivation. Observation of the psychophysiology and neurochemistry literature questions the ability of 90 minutes’ cognitive activity to deplete energy or catecholamine resources. The purpose of this study, therefore, was to examine the evidence for cognitive fatigue having an effect on subsequent physical performance. A systematic, meta-analytic review was undertaken. We found a small but significant pooled effect size based on comparison between physical performance post-cognitive fatigue compared to post-control (g = -0.27, SE = –0.12, 95% CI -0.49 to -0.04, Z(10) = -2.283, p < 0.05). However, the results were not heterogenous (Q(10) = 2.789, p > 0.10, Τ2 < 0.001), suggesting that the pooled effect size does not amount to a real effect and differences are due to random error. No publication bias was evident (Kendall’s τ = -0.07, p > 0.05). Thus, the results are somewhat contradictory. The pooled effect size shows a small but significant negative effect of cognitive fatigue, however tests of heterogeneity show that the results are due to random error. Future research should use neuroscientific tests to ensure that cognitive fatigue has been achieved.

Key words. central executive: central fatigue: motivation.

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**1. Introduction**

 Cognitive fatigue is commonly viewed as a psychobiological state that occurs following an extended period of self-regulated activity, which leads to a decrement in the performance of reasoned cognitive processing tasks over a period of time [1,2]. Comparatively recent increased interest in physically-induced central fatigue (e. g. [3-8]) has led several authors (e. g. [9-12]) to utilize a cognitive fatigue-subsequent physical performance task paradigm to examine the effect of cognitive fatigue, induced by undertaking central executive tasks, on subsequent physical performance. The underlying rationale for such an experimental approach has intuitive appeal. Overcoming exercise-induced central fatigue is thought to involve inhibition of the desire to stop exercising, a process which has been claimed to take place in the pre-supplementary motor area (pre-SMA) and anterior cingulate cortex (ACC) [10, 11, 13]. The ACC and pre-SMA are seen by most cognitive neuroscientists as being part of the central executive process which Miyake et al. [14] termed “inhibition of prepotent responses” (p. 50), therefore researchers examining the effect of cognitive fatigue on subsequent physical performance have utilized the undertaking of central executive inhibition tasks to cognitively fatigue participants [9-11, 13, 15-18].

 Recent narrative reviews [19, 20] have concluded that the literature appears to support the hypothesis that cognitive fatigue would negatively affect subsequent physical performance. However, observation of the results, especially when outcome variables (e.g. time to complete the task and distance covered in a given time) are measured, shows almost equivocal findings. Moreover, examination of the broader cognitive fatigue and resource depletion literature shows that there is little evidence of fatigue taking place [21, 22], while claims concerning the mechanisms involved have been questioned. For example, one initially considered proposal that cognitive fatigue induces depletion of glucose levels in the brain [23], has been strongly criticized [24]. Indeed, reviews have shown very little evidence for any significant increase in brain metabolism during cognition [24, 25]. However, other underlying factors have also been put forward as possible reasons for performance of central executive tasks inducing a decrement in subsequent physical performance. Consistent with Marcora et al.’s psychobiological [10] and Noakes et al.’s [6] “central governor” theories of central fatigue , it has been argued that undertaking the central executive tasks results in the individual perceiving the effort required to undertake the exercise as being greater than in the control condition [10, 20]. It has been claimed that this has a negative effect on the person’s motivation for undertaking the subsequent physical task, resulting in poorer performance than in a control condition [20].

 These issues leave us concerned about the conclusions drawn by the narrative reviewers. The efficacy of central executive tasks to induce brain energy and neurotransmitter depletion is questionable but evidence from cognitive fatigue tasks does not supply any definitive answers. However, there is some qualitative information concerning the possible effects of cognitive fatigue-induced changes in perception of effort and motivation levels on subsequent physical performance. Therefore, we decided to attempt to clarify the situation. To do this, we undertook a systematic review with meta-analysis of the evidence for cognitive fatigue, induced by undertaking central executive tasks, having a negative effect on subsequent physical performance. The meta-analysis provides empirical evidence which will either support or fail to support the conclusions made by the narrative reviewers. Moreover, it will provide empirical evidence concerning the strength of any significant effect of cognitive fatigue on subsequent physical performance. This will help researchers determine whether to continue with this type of protocol or whether it needs to be refined. This is an important issue as this protocol is seen as providing useful evidence in the central fatigue hypothesis debate.

**2. Method**

 The reporting and protocol for this study followed the preferred reporting items for systematic reviews and meta-analysis (PRISMA) protocols. A systematic literature search using the databases PubMed and SCOPUS was undertaken. Each database was searched from their earliest available record up to November 2017. Key words used in the searches were combinations of “cognitive” “fatigue”, “exercise”, “physical”, “subsequent” and “performance”. In addition, reference lists from empirical reports and reviews were examined and screened for eligibility.

**2.1. Selection of studies**

 Two of the authors selected articles for inclusion. The titles and abstracts of publications obtained by the search strategy were screened. All trials classified as relevant by any of the authors were retrieved. Based on the information within the full reports, we used a standardized form to select the trials eligible for inclusion in the review. There was no blinding to study author, institution or journal at this stage.

 Studies were included if (a) they were performed on healthy humans: (b) within-subject design was used: (c) the study design included a control condition: (d) the cognitive fatigue condition (i) utilized a commonly accepted central executive task requiring conscious inhibition of prepotent responses, according to Baddeley [26] and/or Miyake et al. [14]; (ii) required an objective response; (iii) there was clear evidence of the brain neuroanatomy involved in undertaking the task (iv) subjectively scored emotional tasks were not included: (e) the requirement of the post-treatment physical performance was (i) to cover a given distance in as fast a time as possible; or (ii) to cover as much distance as possible in a given time; or (iii), with resistance exercise, maintaining a given force production to failure/exhaustion;(iv) to complete as many repetitions as possible in a given time, or (v) to complete as many repetitions as possible before voluntary exhaustion. Studies also had to present statistical information from which effect sizes could be calculated. English language restrictions were applied.

**2.2. Statistical treatment**

 Initially Cohen’s d effect sizes were calculated using the following formula: mean control – mean experimental/SD control. These were then transformed to Hedges’ g by applying the correction factor J [J = 1 – (3/4df – 1)]. A random effects group comparative model was used to calculate the pooled effect size using Comprehensive Meta Analysis Version 3.0 [27]. Statistical heterogeneity of the treatment between studies was evaluated using the Cochran Q test (consistent with other research, the threshold p value of 0.1 was considered statistically significant [28, 29]) and T2, and the I² test for inconsistency (values ​​greater than 75% were considered as indicative of high heterogeneity [28]). Publication bias was examined using Begg’s test [29].

**3. Results**

 Figure 1 outlines the stages of the literature research and choice of studies to be included. Following this procedure, there were eight studies. Of these studies, two undertook two separate experiments with different participants and so for the meta-analysis these were treated as separate studies. Similarly, one study tested two distinctly different groups of participants and this study was also treated as two separate studies, as it yielded two separate and independent effect sizes. This resulted in 11 effect sizes with N = 148.

*Insert Figure 1 about here*

 Ten experiments provided data for one outcome effect size, while one provided data for two effect sizes. In this study, effect sizes were combined to form one effect size as recommend by Borenstein et al. [27]. There was a total of 11 effect sizes. Table 1 shows the main outcomes from each experiment based on probabilities.

*Insert Table 1 about here*

 Examination of Table 1 shows that from the 11 studies, there were 12 outcome dependent variables based on probabilities. Half of the variables for physical performance were significantly, negatively affected by mental fatigue. All experiments except one demonstrated negative effect sizes. Shücker and MacMahon (study 2) [17] showed a positive but near zero effect size. The pooled effect size was g = -0.29 (SE = –0.12), 95% CI -0.49 to -0.04 (Z(10) = -2.283, p = 0.02). However, the results showed little dispersion of effects across the selected studies (Q(10) = 2.787, p = 0.99, Τ2 < 0.001, Ι2 <0.001%), suggesting that the pooled effect size does not amount to a real effect and differences are due to random error [27, 28] (see Figure 2).. No publication bias was evident, as measured by the Begg and Mazumdar [29] formula (Kendall’s τ = -0.07, p = 0.38, one-tailed). The classical fail-safe N [30] was significant (Z = -2.29, p = 0.02) and estimated that five more studies showing positive effects would result in non-significance.

*Insert Figure 2 about here*

**4. Discussion**

 The findings from this meta-analytical review, indicate that the evidence for cognitive fatigue, induced by central executive inhibition tasks, having a negative effect on subsequent physical performance appears to be currently unclear. The results, based on probabilities (see Table 1), are equivocal, while the meta-analysis data also provide a somewhat mixed picture. The pooled effect size is small but significant, when measured by comparison between post-mental fatigue and post-control condition results. However, the Q value is non-significant, which means that the dispersion between the studies is less than one would expect by chance. More importantly, the very small Τ2 result indicates that there was no real significant effect and that differences are due more to random error. This empirical approach differs from the conclusions made by the authors of the two narrative reviews [19, 20]. To some extent, this may be due to small differences in the criteria for inclusion in those reviews and in the present meta-analysis, but observation of the probability results of the studies reviewed by those authors provide very similar results to the probabilities reported in this study. Indeed, Van Cutsem et al. [20] were cautious in interpreting their data, due to the fact that their results were close to being equivocal. We too are cautious because one set of data supports a difference, while two others do not. Moreover, our sample size is small, as indeed are those of the narrative reviewers [19, 20]. However, while the probability results and the pooled effect size results are affected by sample size, the Τ2 statistic is not, as this measure depends on scale. Therefore, our results provide more support for the argument that the data are due to random error rather than a true treatment effect [27]. Nevertheless, one can not simply ignore the fact that the pooled effect size was significant, although small but Inzlicht and colleagues [21, 22] have questioned the efficacy of at least part of the underlying rationale by raising doubts concerning whether or not cognitive tasks really do affect brain metabolism [21, 22].

 We chose central executive inhibition tasks because in many of the studies reviewed, authors stated or implied [9, 10, 13, 16] that during endurance exercise, the athlete must inhibit perceptions of fatigue if the goal is to be achieved. Therefore, the performance of cognitively fatiguing central executive tasks, which require inhibition of prepotent responses, would deplete resources necessary to inhibit perceptions of fatigue, leading to impairment of the participant’s physical performance relative to the control condition. As highlighted in the Introduction, research has proposed that this activity is dependent on activation of the pre-SMA and ACC [10]. As a result, the studies have logically and sensibly utilized central executive inhibition tasks, including the Stroop color-word test [31], go/no go task (see [32]) and the AX-Continuous Performance Task (AX-CPT) {33], as the cognitively fatiguing conditions. These tasks are all thought to activate similar neural pathways. The color-word interference condition in the Stroop test activates a wide range of regions including ACC, dorsolateral prefrontal cortex (DLPFC), SMA, inferior frontal gyrus (IFG) and medial superior parietal cortex [34, 35]. The go/no go task also activates the ACC, SMA and the right IFG but in addition, the pre-SMA and the subthalamic nucleus (STN) [36-38]. The AX-CPT engages the ACC, bilateral DLPFC, left premotor cortex and the bilateral IFG [39, 40]. These collective brain regions are part of the dopaminergic and noradrenergic pathways, which depend upon the supply of the catecholamines neurotransmitters dopamine and noradrenaline for activation. One other study considered in this review [12] also included the 1-back test [41], a central executive task which requires dopamine and noradrenaline for activation but is not an inhibition task [42]. However, the main task used in the studies considered in this review was the Stroop color word task.

 As stated above, the evidence that undertaking central executive tasks really does induce cognitive fatigue has been questioned [21, 22] and in a series of comprehensive literature reviews, Raichle [43, 44], and Raichle and Gusnard [45] concluded that there is little evidence to show that in normoxia and normothermia, brain energy supplies are depleted by cognition. Indeed, this does question the use of the term “cognitive fatigue” in this type of protocol. Unfortunately, based on the data available, we can not state whether or not brain metabolism was depleted in the cognitive fatigue tasks reviewed, but brain metabolism is not the only factor involved in cognitive fatigue. Marcora and colleagues [10, 13] proposed that undertaking a cognitive task can affect motivation for subsequent physical performance and this we can examine, albeit qualitatively, from the studies examined. However, observation of Table 1 shows that there was no reported significant diminution of motivation following completion of the central executive tasks or prior to undertaking the exercise in the control condition in any of the studies. Nevertheless, in eight of the 11 experiments, participants reported significantly increased subjective perceptions of fatigue and/or effort following the inhibition task (see Table 1) and it is possible that this could have affected subsequent physical performance, even if only to a small extent. That the effect is small may also be due to the fact that our data suggest that there was no diminution of motivation (see Table 1). This would have a positive effect on the decision to continue exercising rather than stop, as has been shown in studies examining the effect of pre-exercise motivational self-talk [46]. Observation of Table 1 and Figure 3 also suggests that there is a possible gender factor, in that the all male studies tended to show the highest negative effects. Unfortunately the data are such that sub-group analyses are not viable.

4.1. Limitations and future research

 The sample size was large enough to carry out a basic meta-analysis but did not allow for comparison between possible moderators, such as duration of the central executive task. Examination of the effects of other central executive tasks, e.g. sustained attention, would be interesting. The fact that there were no physiological, psychophysiological or neurochemical measures during the central executive tasks means that comment on their efficacy is somewhat speculative although based on research into effects of cognitive fatigue on similar tasks. Moreover, Van Cutsem et al. [20] suggested that cognitive fatigue may affect endurance tasks but not sprint/power/strength tasks and this could be examined in future research. Potential gender differences should also be examined.

**5. Conclusion**

 This analysis shows a small but significant pooled effect size based on comparison between physical performance post-mental fatigue compared to post-control. However, results for heterogeneity, especially Τ2, indicate that effects are likely due to random error rather than a true intervention effect. Observation of results of the cognitive fatigue tasks (see Table 1) shows that although motivation was not negatively affected, perception of effort following the mental task was higher than in the control condition. This may account for the small but significant pooled effect size, if it affected the individual’s decision regarding the energy costs involved in continuing the exercise in the post-cognitive fatigue condition. That the effect is small and its significance questionable may be due to the fact that motivation does not appear to have been affected by cognitive fatigue, which may have allowed participants to overcome the post-cognitive fatigue higher perception of effort. This strongly suggests that research, in which motivation, is manipulated is necessary before definitive conclusions can be made with regard to the effects of cognitive fatigue on subsequent physical performance.

**Compliance with ethical standards**

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**Conflict of interest**

Terry McMorris, Martin Barwood, Beverley J. Hale, Matt Dicks and Jo Corbett declare that they have no conflicts of interest relevant to the content of this review.

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**Figure headings**

Figure 1. Stages of the literature research and choice of studies to be included

Figure 2. Forest plot showing the pooled and individual effect sizes and 95% confidence intervals. Negative results indicate that performance following cognitive fatigue was poorer than in the control condition. Positive effect sizes show that performance following the central executive task was better than in the control condition.

Note. pro professional cyclists: rec recreational cyclists: CI confidence interval.