The effects of acute high intensity aerobic exercise on cognitive performance:

a narrative review

Mizuki Sudo1\*, Joseph T. Costello2, Terry McMorris2,3, Soichi Ando4

1Physical Fitness Research Institute, Meiji Yasuda Life Foundation of Health and Welfare, Shinjuku, Tokyo, Japan

2Extreme Environments Laboratory, School of Sport, Health & Exercise Science, University of Portsmouth, Portsmouth, United Kingdom

3Institute of Sport, University of Chichester, Chichester, United Kingdom

4Graduate School of Informatics and Engineering, The University of Electro-Communications, Chofu, Tokyo, Japan

**\* Correspondence:**Soichi Ando
soichi.ando@uec.ac.jp

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Abstract

It is well established that acute moderate intensity exercise improves cognitive performance. However, the effects of acute high intensity aerobic exercise on cognitive performance has not been well characterized. In this review, we summarize the literature investigating the exercise-cognition interaction, specially focusing on high-intensity aerobic exercise. We discuss methodological and physiological factors that potentially mediate cognitive performance in response to high intensity exercise. We propose that the effects of high intensity exercise on cognitive performance are primarily affected by timing of cognitive task (during vs. after exercise). In particular, cognitive performance is more likely to be impaired during high intensity exercise when both cognitive and physiological demands are high and completed simultaneously. The effects may also be affected by type of cognitive task and physical fitness of participants. Second, we suggest that interactions between changes in regional cerebral blood flow, cerebral oxygenation, cerebral metabolism, neuromodulation by neurotransmitters/neurotrophic factors, and a variety of psychological factors are promising candidates that determine cognitive performance in response to acute high intensity exercise. The present review has implications for recreational, sporting, and occupational activities where high cognitive and physiological demands are required to be completed concurrently.

# Introduction

A growing body of evidence suggests that acute moderate intensity exercise improves cognitive performance (Lambourne and Tomporowski, 2010, Chang et al., 2012, Ando et al., 2020, McMorris, 2021). It has been speculated that the relationship between exercise intensity and cognitive performance is an inverted-U shaped (Lambourne and Tomporowski, 2010, Chang et al., 2012, McMorris, 2021). In the inverted-U theory, acute exercise gradually increases arousal to an optimal level from rest to moderate intensity and thus improves cognitive performance. A recent review summarized that improvements in cognitive performance following moderate intensity exercise are frequently accompanied by the changes in brain activation assessed by electroencephalogram (EEG) (Kao et al., 2020), which appears to support the theory that acute exercise alters brain activity and that this is associated with cognitive performance. Acute high intensity aerobic exercise leads to metabolic, circulatory, and neurohormonal changes at the level of the brain (Ide and Secher, 2000, Meeusen et al., 2001, Nybo and Secher, 2004, Ogoh and Ainslie, 2009, Seifert and Secher, 2011). In contrast to moderate intensity exercise, theoretically high intensity exercise may therefore also lead to altered, and potentially impaired, cognitive performance. Indeed, the inverted-U theory predicts that high intensity exercise increases arousal level beyond the optimal level and leads to a temporary reduction in cognitive performance. However, the current literature base detailing the effects of high intensity exercise on cognitive performance are not fully supported of this theory and is somewhat ambiguous and contradictory (Browne et al., 2017, Moreau and Chou, 2019, Cantelon and Giles, 2021, McMorris, 2021, Zheng et al., 2021).

Dietrich and Audiffren (2011) proposed the hypofrontality hypothesis to explain how acute high intensity exercise affects cognitive performance. The prefrontal cortex (PFC) orchestrates higher order brain function including cognitive function (Miller and Cohen, 2001, Cools and Arnsten, 2021), and is thought to play a central role in cognitive performance. Acute exercise activates brain regions including motor and sensory cortices, insular cortex, and cerebellum (Williamson et al., 1997, Christensen et al., 2000, Hiura et al., 2014). Hence, the hypofrontality theory speculates that extensive activation of motor and sensory systems during high-intensity exercise likely attenuates higher order functions of the PFC as the brain has finite metabolic resources (Dietrich and Audiffren, 2011). More recently, McMorris proposed an interoceptive model to explain the effects of high intensity exercise on cognitive performance (McMorris, 2021). This model offers a more holistic overview of the interaction as it incorporates motivation, perceived effort costs, and perceived availability of resources, together with regional activations and neurotransmitter releases in the brain. Nevertheless, to date, the (psycho)physiological mechanism(s) mediating the effects of acute high intensity exercise on cognitive performance is poorly understood.

In this review, we first summarize the findings of studies investigating the exercise-cognition interaction, specially focusing on high-intensity exercise. Then, we explore methodological and physiological factors which may alter cognitive performance after high intensity exercise. This review has implications for recreational, sporting and occupational activities where high cognitive and physiological demands are simultaneously required.

# Methodology

# A literature search was undertaken using Pubmed to identify studies that examined the effects of high intensity aerobic exercise on cognitive performance, assessed during and/or after exercise. The reference lists of relevant articles were also searched. The searches were undertaken in February 2022 and relevant articles were obtained. This review focused on healthy adults, and no restrictions were placed on publication date, study design, methodology or method of assessing cognitive performance. High intensity aerobic exercise was defined as exercise equating to ≥80% maximum power output (Browne et al., 2017), ≥80% maximal oxygen uptake (V̇O2) (McMorris, 2016b), or equivalent [e.g. ≥80% maximal heart rate (HR)]. Studies incorporating high intensity interval exercise (HIIE) were considered outside the scope of this review. We also did not include studies conducted in extreme environments, such as hypoxia and hot/cold environments. Instead, we referred evidence from HIEE studies, or studies in extreme environments, for discussion to better understand the physiological mechanisms underlying cognitive improvement/impairment.

Details of the included studies are described in Table 1, comprising a total of 40 studies (assessed during exercise, n = 19; assessed both during and after exercise, n = 2; assessed after exercise, n = 19). In many studies, cognitive performance was impaired during high intensity exercise (Chmura et al., 1994, McMorris and Keen, 1994, Brisswalter et al., 1997, McMorris et al., 2009, Labelle et al., 2013, Wang et al., 2013, Dutke et al., 2014, Mekari et al., 2015, Schmit et al., 2015, Smith et al., 2016, Gonzalez-Fernandez et al., 2017, Tempest et al., 2017, Komiyama et al., 2020, Stone et al., 2020). In these studies, impairments in both reaction time (RT) and accuracy were frequently observed. Four studies reported no changes in cognitive performance (Travlos and Marisi, 1995, Ando et al., 2011, Dutke et al., 2014, Ciria et al., 2019). Five studies reported improvement in RT and/or accuracy during high intensity exercise (McMorris and Graydon, 1997, Huertas et al., 2011, Shields et al., 2011, Davranche et al., 2015, Tempest et al., 2017). In four out of these five studies (Huertas et al., 2011, Shields et al., 2011, Davranche et al., 2015, Tempest et al., 2017), however, cognitive demands and/or exercise intensity were less demanding as compared with other studies. One study reported improvements in soccer-specific cognitive tasks in college soccer players (McMorris and Graydon, 1997), which suggests the improvements in cognitive performance may be associated with the cognitive tasks used in this study. Hence, available literature suggests that cognitive performance is more likely to be impaired during high intensity exercise when both cognitive and physiological demands are high.

Conversely, cognitive performance after high intensity exercise is heterogeneous; with improvements (Winter et al., 2007, Luft et al., 2009, Thomson et al., 2009, Griffin et al., 2011, Etnier et al., 2016, Hwang et al., 2016, Hill et al., 2019, Coco et al., 2020a, Loprinzi et al., 2021), impairments (Fery et al., 1997, McMorris et al., 2005, Coco et al., 2009, Thomson et al., 2009, Zimmer et al., 2016, Hill et al., 2019, Coco et al., 2020a), and no changes (Travlos and Marisi, 1995, Brisswalter et al., 1997, Kamijo et al., 2004a, Kamijo et al., 2004b, Luft et al., 2009, Chang et al., 2017, Sudo et al., 2017, Du Rietz et al., 2019, Marin Bosch et al., 2021) reported within the literature. These findings suggest that cognitive performance after high intensity exercise appears to be dependent on experimental design (*see below*). In the following sections, we discuss the methodological and (psycho)physiological factors that affect cognitive performance “*during*” and “*after*” high intensity exercise.

# Methodological factors

Here we discuss the potential methodological and experimental factors that contribute to the inconsistent findings. These include the following: timing of cognitive task (during or after exercise), type of cognitive task, and physical fitness of the participants.

*Timing of cognitive task*

When participants perform cognitive tasks during exercise, they perform exercise and cognitive tasks simultaneously (i.e. using a dual task paradigm). However, when cognitive tasks are performed after exercise, participants only perform a single task. A meta-analysis reported higher effect sizes in single task conditions (after exercise) when compared with dual task conditions (during exercise) (Lambourne and Tomporowski, 2010), while another meta-analysis reported that effect sizes were not different between single and dual task conditions (Chang et al., 2012). Furthermore, McMorris and Hale (2012) undertook statistical analyses and found that there were no differences in effect sizes obtained during compared after exercise (McMorris and Hale, 2012). Nevertheless, as recently highlighted (McMorris, 2021), the timing of the cognitive tasks is typically less considered within the literature.

Table 1 indicates that the adverse effects are most prominent *during* high intensity exercise. These findings are corroborated by a recent review and suggest that impairments in cognitive performance are more likely to occur during high intensity exercise (Zheng et al., 2021). Based on the assumption that metabolic resources are limited in the brain, extensive activation in several brain regions (e.g. motor and sensory cortices) may attenuate higher order functions of the PFC and impair cognitive performance (Dietrich and Audiffren, 2011). Furthermore, in the majority of the included studies, cognitive performance was assessed using manual responses. Given that a limited capacity of the brain to simultaneously activate multiple regions involved in cognitive performance and high intensity exercise, it is plausible that cognitive performance is more likely to be impaired during high intensity exercise, particularly when both cognitive and physiological demands are high.

A recent meta-analysis demonstrated that acute high intensity exercise had a small, significant facilitating effect on cognitive performance *after* high intensity exercise (Moreau and Chou, 2019). In the current review, we observed that cognitive performance after high intensity exercise are inconsistent: and improvements, impairments, and no changes were reported. EEG studies reported reductions in P3 amplitudes after high intensity (Kamijo et al., 2004a, Kamijo et al., 2004b) or HIIE (Kao et al., 2017). On the contrary, Du Rietz and colleagues reported improvements in P3 amplitude and delta power reflecting executive and sustained attention after high intensity exercise (Du Rietz et al., 2019). These findings suggest that brain activity after exercise may be dependent on the experimental design employed (e.g. exercise intensity, time delay after exercise). Indeed, most physiological changes start to recover immediately after high intensity exercise (Ide et al., 2000, Gonzalez-Alonso et al., 2004, Curtelin et al., 2017, Sudo et al., 2017). Thus, a rapid recovery of physiological variables to homeostatic resting levels may, at least in part, explain the contradictory findings related to cognitive performance after high intensity exercise. Taken collectively, we propose that the effects of high intensity exercise on cognitive performance is closely related to, and impacted by, the timing of cognitive task (during vs. after exercise).

*Type of cognitive task*

Different brain regions are thought to be activated during different cognitive tasks (Macintosh et al., 2014, Chen et al., 2016a, Won et al., 2019). Thus, we can assume that type of cognitive task is one of the factors that determine how acute high intensity exercise impacts cognitive performance. This may be particularly relevant when exercise and cognitive task are concurrently performed since multiple brain regions are presumably activated. Indeed, cognitive improvements during high intensity exercise were observed in less cognitively demanding tasks (e.g. visual attention/detection task, flanker task). However, most of the studies included in this review assessed central executive performance, and we may not have been sufficiently powered to identify the effects of different cognitive tasks/domains. Further research is required to establish the effects of high intensity exercise of a variety of cognitive tasks and domains.

*Physical fitness level of participants*

It has been previously speculated that exercise-cognition interaction is influence by physical fitness (Lambourne and Tomporowski, 2010, Chang et al., 2012). For example, despite matched relative exercise intensity, individuals with lower physical fitness levels were more susceptible to cognitive impairments during high intensity exercise, when compared with those who had higher aerobic capacities (Brisswalter et al., 1997, Labelle et al., 2013). Furthermore, choice RT performance gradually improves during incremental exercise until at ~75% V̇O2max in young soccer players (Chmura et al., 1994). Physical fitness level has been suggested to be one of the moderators that affect cognitive performance in response to high intensity exercise (Browne et al., 2017). Further well powered studies are also necessary to clarify the relationship between cognitive performance and physical fitness before this theory can be confirmed.

**(Psycho)Physiological factors**

As noted above, high intensity exercise induces a variety of physiological effects on the human brain. We summarize and discuss physiological factors that are linked to cognitive performance in response to high intensity exercise. Here we discuss the potential (psycho)physiological factors that may contribute to the inconsistent findings. These include the separate and combined effects of cerebral blood flow (CBF), cerebral oxygenation, cerebral metabolism, neuromodulation by neurotransmitters and neurotrophic factors, and various psychological factors.

*Cerebral blood flow (CBF)*

During exercise, CBF is regulated by complex interactions between neural activity and metabolism, partial pressure of oxygen, carbon dioxide (CO2), blood pressure, cardiac output, and sympathetic nervous system activity (Ogoh and Ainslie, 2009, Smith and Ainslie, 2017). CBF gradually increases during mild- to moderate intensity exercise in response to neural activity and metabolism (Ogoh and Ainslie, 2009). However, during high intensity exercise, hyperventilation-induced hypocapnia constricts the cerebral vessels, thereby reducing CBF (Ogoh and Ainslie, 2009, Smith and Ainslie, 2017). This suggests that brain metabolic demands might be inadequate during high intensity exercise. Ogoh et al. (2014) reported that an increase in CBF, achieved using CO2 inhalation, did not affect cognitive performance during prolonged moderate intensity exercise (Ogoh et al., 2014). More recently, Komiyama and colleagues tested the hypothesis that a reduction in CBF is directly linked to impairment in cognitive performance during high intensity exercise (Komiyama et al., 2020). By restoring CBF via CO2 inhalation, the authors demonstrated that middle cerebral artery (MCA) velocity (a surrogate for CBF) did not prevent impaired cognitive performance during high intensity exercise. These results suggest that a reduction in CBF *per se* may not be responsible for impaired cognitive performance during high-intensity exercise. However, given that CBF supplies oxygen and nutrients, the association between cognitive performance and regional CBF (e.g. blood flow to the PFC) in response to high intensity exercise should be further investigated. In particular, a recent study indicated physiological “uncoupling” between the PFC oxygenation and MCA velocity during high intensity exercise with CO2 inhalation (Hansen et al., 2020). Follow-up studies using sophisticated neuroimaging methods (e.g. fMRI or PET) are helpful to fully understand the association between regional CBF and cognitive performance in response to high intensity exercise.

*Cerebral oxygenation*

Cerebral oxygenation reflects the balance between cerebral oxygen availability and utilization (Boushel et al., 2001, Komiyama et al., 2017), which is generally measured from the PFC. Cerebral oxygenation reduces during high intensity exercise close to maximal intensity (Rooks et al., 2010). Some studies suggest that a reduction in cerebral oxygenation is not associated with impairments in cognitive performance during high intensity exercise (Ando et al., 2011, Schmit et al., 2015, Tempest et al., 2017). In contrast, others have indicated that impairments in cognitive performance was accompanied by reduction in cerebral oxygenation during high intensity exercise (Mekari et al., 2015, Stone et al., 2020). The latter studies suggest that impairments in cognitive performance may be associated with attenuated PFC oxygenation. However, several studies have shown that cognitive performance improved during acute moderate intensity exercise in hypoxia despite substantial reductions in cerebral oxygenation (Ando et al., 2013, Komiyama et al., 2015, Komiyama et al., 2017). Hence, it is likely that a reduction in cerebral oxygenation, in isolation, does not result in impaired cognitive performance. However, reduction in cerebral oxygenation during high intensity exercise may impair cognitive performance in concert with other physiological factors.

Cerebral oxygenation starts to recover immediately after maximal exercise (Gonzalez-Alonso et al., 2004, Sudo et al., 2017). Notably, the degree of recovery of cerebral oxygenation following maximal exercise may be associated with cognitive performance (Sudo et al., 2017). This finding suggests that recovery of cerebral oxygenation after high intensity exercise may, at least in part, account for the differential effects of high intensity exercise on cognitive performance between single (i.e. after) and dual (i.e. during) conditions.

*Cerebral metabolism*

It is generally accepted that blood glucose is the primary energy source for the brain at rest (Gold, 1995). Komiyama and colleagues (2016) reported that cognitive performance improves during moderate intensity exercise after skipping breakfast (Komiyama et al., 2016). This finding suggests that substrates other than glucose may compensate for reduced availability of blood glucose during moderate intensity exercise. It is plausible that the same would be true for high intensity exercise. Indeed, blood glucose uptake is thought to be reduced in the brain during high intensity exercise (Kemppainen et al., 2005). In contrast, blood lactate substantially increases during/after high intensity exercise, and it is taken up by the brain (Ide et al., 2000, Gonzalez-Alonso et al., 2004, Quistorff et al., 2008, Siebenmann et al., 2021). Several studies suggested that blood lactate would provide energy that contributes to improvements in cognitive performance following HIIE (Tsukamoto et al., 2016, Hashimoto et al., 2018, Herold et al., 2022). In particular, Hashimoto and colleagues directly measured lactate uptake in the brain after HIIE, and suggested that lactate production in extra-cerebral tissues supports brain function (Hashimoto et al., 2018). On the contrary, Coco and colleagues suggested that high levels of blood lactate have detrimental effects on cognitive performance (Coco et al., 2020b). Interestingly, Coco et al. indicated that intravenous lactate infusion of a lactate solution impaired attentional performance (Coco et al., 2009). Hence, further studies are warranted to investigate how blood lactate acts as a mediator of exercise-induced alterations in cognitive performance (Ando et al., 2022).

*Neuromodulation by neurotransmitters and neurotrophic factors*

In humans, it is less clear how acute exercise affects central neurotransmitters release due to technical and methodological challenges. Nevertheless, given that rodent studies indicate that acute exercise releases neurotransmitters in the brain (Meeusen et al., 2001, Hasegawa et al., 2011, Goekint et al., 2012, Chen et al., 2016b), acute exercise is likely to influence brain circuits involving a number of neurotransmitters including dopamine and noradrenaline (McMorris, 2016a, Ando et al., 2020). Dopamine and noradrenaline modulate the strength of the PFC network connections, and regulation of dopamine and noradrenaline is required for appropriate prefrontal cognitive function (Arnsten, 2011). Furthermore, excess noradrenaline and dopamine appear to weaken the signal to noise ratio, which may result in impairments in the PFC function (Arnsten, 2011, Cools and Arnsten, 2021). Hence, the available literature suggests that excess of neuromodulators in the brain may have adverse effects on cognitive performance during/after high intensity exercise. High intensity exercise also seem to increase brain-derived neurotrophic factors (BDNF) (Ferris et al., 2007, Winter et al., 2007, Fernandez-Rodriguez et al., 2021) and insulin-like growth hormone factor-1 (IGF-1) (Sudo et al., 2017). Several studies have implicated that changes in BDNF is associated with cognitive improvement induced by acute exercise (Winter et al., 2007, Lee et al., 2014, Skriver et al., 2014, Hwang et al., 2016). However, BDNF and IGF-1 are known to play a crucial role in angiogenesis, synaptogenesis, and neurogenesis following long-term exercise (Cotman and Berchtold, 2002, Voss et al., 2011, Nieto-Estevez et al., 2016). It may be premature to conclude that changes in BDNF and IGF-1 play a role in cognitive performance during/after high intensity exercise.

*Psychological factors*

In most studies, psychological factors are typically not considered when attempting to elucidate the effects of high intensity on cognitive performance. However, as suggested by McMorris (2021), psychological factors such as motivation and perception of effort may affect the acute exercise-catecholamine-cognition interaction (McMorris, 2021). Cantelon and Giles also suggested that psychological factors are moderating factors that affects exercise-cognition interaction (Cantelon and Giles, 2021). At present, and given the lack of empirical evidence, further investigations are needed to investigate the association between psychological factors and cognitive performance during and after high intensity exercise.

*Integration of (psycho)physiological factors*

It is likely that the effects of high intensity exercise on cognitive performance are multifactorial and determined by the integration of several physiological and psychological factors. We propose that interactions of these factors influence neural activity associated with cognitive performance and that this determines cognitive performance during and after high intensity exercise. However, the current literature base is insufficient to substantiate this speculation and this should be the focus of future research in this area.

**Conclusion**

# This narrative review summarized the literature examining the effects of acute high intensity exercise on cognitive performance. We propose that the effects of high intensity exercise on cognitive performance are primarily affected by a variety of methodological and (psycho)physiological factors (Figure 1). Specifically, these include the timing of cognitive task (during *vs.* after exercise), cognitive task(s), and fitness level. It is also likely that a complex interaction between changes in regional CBF, cerebral oxygenation, cerebral metabolism, neurotransmitters/neurotrophic factors, and a variety of psychological factors explains many of the heterogeneous findings. The review is likely to have implications for recreational, sporting, and occupational activities where high cognitive and physiological demands are required simultaneously.

# Conflict of Interest

The authors declare that they have no competing interests.

# Author Contributions

MS, JTC, TM, and SA drafted the manuscript. All authors approved the final version of the manuscript.

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Table 1 Summary of the findings

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Authors | Participants (F) | Cognitive task(s) | Exercise modality/intensity | Timing of cognitive task | Physiological variables | Main findings |
| Chmura et al. (1994) | N =22 (0) | Choice RT | Near maximal (300W) | During | Blood catecholamine, lactate | RT: impairment |
| McMorris & Keen (1994) | N = 12 (4) | Simple RT | Cycling100% maximum workload | During | - | RT: impairment |
| McMorris & Graydon (1997) | N =12 (0) | Soccer specific visual search taskSoccer specific decision making task | Cycling100% maximum power output | During | - | Visual search: improvementDecision making: improvement |
| McMorris et al. (2009) | N = 24 (0) | Flanker task | Cycling 80% maximum aerobic power | During | Blood catecholamine, adrenocorticotropin hormone, cortisol | RT: impairmentAccuracy: impairment |
| Ando et al. (2011) | N = 12 (0) | Flanker task | Cycling80% V̇O2peak | During | Cerebral oxygenation | RT≈Accuracy≈ |
| Huertas et al. (2011)  | N = 18 (0) | Attention network test | Cycling95% LT | During | Blood lactate | RT: improvement |
| Shields et al. (2011) | N = 30 (15) | Visual threat-detection task | Cycling80% Maximal HR | During | - | RT: improvementAccuracy: improvement |
| Labelle et al. (2013) | N = 37 (18)High fit = 16Low fit = 21 | Stroop task | Cycling 80% peak power output | During | - | Accuracy: impairmentRT variability: impairment in lower fit |
| Wang et al. (2013) | N = 80 (31) | Wisconsin card sorting test | Cycling80% HRR | During | - | Performance: impairment |
| Dutke et al. (2014) | N = 60 (14) | Word comparisonInterval production (press a button every 2s) | Cycling120% AT | During | - | Number of correct response≈Response time≈Interval production error: impairment |
| Davranche et al. (2015) | N = 14 (3) | Simon task | Cycling20% above VT | During | - | RT: improvementAccuracy≈ |
| Mekali et al. (2015) | N = 19 (12) | Stoop task | Cycling85% peak power output | During | Cerebral oxygenation | RT: impairmentAccuracy: impairment |
| Schmit et al. (2015) | N = 15 (5) | Flanker task | Cycling85% maximal aerobic power | During | Cerebral oxygenation | RT≈Accuracy: impairment |
| Smith et al. (2016) | N = 15 (9) | Go/No-Go task | Running90% HRR | During | - | RT: impairmentAccuracy: impairment |
| González-Fernández et al. (2017) | N = 24 (12) | Psychomotor vigilance task | Cycling100% ventilatory anaerobic threshold | During | - | RT: impairment |
| Tempst et al. (2017) | N = 14 (5) | Flanker task2-back task | Cycling10% above VT | During | Cerebral oxygenation | RT (flanker task): improvementAccuracy (n-back): impairment |
| Ciria et al. (2019)  | N = 20 (0) | Oddball task | Cycling80% V̇O2peak | During | EEG | RT≈Accuracy≈ |
| Komiyama et al. (2020) | N = 17 (0) | Spatial delayed response taskGo/No-Go task | Cycling80% V̇O2peak | During | Middle cerebral arteryCerebral oxygenation | RT≈Accuracy: impairment |
| Stone et al. (2020) | N = 13 (5) | Cedar Operator Workload Assessment Tool | CyclingNear maximal | During | Cerebral oxygenation | Accuracy: impairment |
| Travlos & Marisi (1995) | N = 20 (0)High fit = 10Low fit = 10 | Random number generation testChoice RT | Cyclingduring 80% V̇O2max & after volitional exhaustion | During/After | - | Random number generation test≈RT(after exercise)≈  |
| Brisswalter et al. (1997) | N = 20 (0)High fit = 10Low fit = 10 | Simple RT | Cyclingduring 80% maximal aerobic power & after volitional exhaustion | During/After | - | RT (during exercise): impairment in only low fitAccuracy≈ in both groups |
| Fery et al. (1997) | N =13 (0) | Short-term memory task | CyclingVolitional exhaustion | After | - | RT: impairment |
| Kamijo et al. (2004a) | N = 12 (0) | Go/No-Go task | CyclingVolitional exhaustion | After | EEG | EMG-RT≈ |
| Kamijo et al. (2004b) | N = 12 (0) | Go/No-Go task | CyclingVolitional exhaustion | After | EEG | EMG-RT≈ |
| McMorris et al. (2005) | N =12 (0) | Whole body choice RT | Cycling100% maximal power output | After | Blood lactate | RT: impairment |
| Winter et al. (2007) | N = 27 (0) | Vocabulary learning task | RunningVolitional exhaustion | After | Blood catecholamine, BDNF | Learning speed: improvementRT: improvement |
| Coco et al. (2009)  | N = 17 (0) | Attention and Concentration task | CyclingVolitional exhaustionLactate infusion (N = 6) | After | Blood lactate | RT: impairmentAccuracy: impairment |
| Luft et al. (2009) | N = 30 (7) | Simple RT, Choice RTWorking memory taskShort-term memory taskContinuous monitoring task | RunningVolitional exhaustion | After | - | Working memory: improvementOthers≈ |
| Thomson et al. (2009) | N =163 (0) | Speed discrimination (decision-making) | RunningVolitional exhaustion | After | - | Time: improvementAccuracy: impairment  |
| Griffin et al. (2011) | N = 47 (0) | Face-name matching taskStroop task | RunningVolitional exhaustion | After | Blood BDNF, IGF-1 | Pairs recalled: improvementAccuracy≈ |
| Etnier et al. (2016) | N = 16 (7) | Rey Auditory Verbal Learning Test | RunningVolitional exhaustion | After | Blood BDNF | Memory performance: improvement(24h later) |
| Hwang et al. (2016) | N = 58 (26) | Stroop testTrail making test | RunningTarget HR corresponding to 85-90% V̇O2max | After | Blood BDNF | Stroop test: improvementTrail making test: improvement |
| Chang et al. (2017) | N = 36 (36) | Stroop test | Running80% HRR | After | Cerebral oxygenation | RT≈ |
| Sudo et al. (2017)  | N = 18 (0) | Spatial DR taskGo/No-Go task | CyclingVolitional exhaustion | After | Cerebral oxygenation, Blood catecholamine, BDNF, IGF-1, lactate | RT≈Accuracy≈ |
| Ziemmer et al. (2017) | N = 119 (78) | Tower of London | CyclingVolitional exhaustion | After | Blood lactate | Thinking time: impairment |
| Du Rietz et al. (2019) | N = 29 (0) | Cued continuous performance taskFlanker taskChoice RT task | Cycling20% delta (difference between gas exchange threshold and V̇O2peak) | After | EEG | RT≈Accuracy≈ |
| Hill et al. (2019) | N = 13 (0) | Flanker task | Cycling & arm crankingVolitional exhaustion | After | - | Cycling: impairmentArm cranking: improvement  |
| CoCo et al. (2020) | N = 30 (?)Young = 15Old = 15 | Simple RTStroop Color Word TestTrail Making Test | CyclingVolitional exhaustion | After | Blood lactate | Simple RT: impairmentStroop Color Word Test: impairmentTrail Making Test: improvement |
| Loprinzi et al. (2021) | N =120 (77) | Word list memory task | Running75% HRR | After | - | Memory: improvement |
| Marin Bosch et al. (2021) | N =18 (0) | Associative memory task | Cycling75% maximal cardiac frequency | After | Neural activity (fMRI), Blood endocannabinoids, BDNF | Accuracy≈ |

F: females; N: number of participants; RT: reaction time; W: watts; V̇O2peak: peak oxygen uptake; LT: lactate threshold; HR: heart rate; HRR: heart rate reserve; AT: anaerobic threshold; VT: ventilatory threshold; EEG: electroencephalogram; V̇O2max: maximal oxygen uptake; EMG: electromyogram; BDNF: brain derived neurotrophic factor; IGF-1: insulin-like growth hormone factor 1. ≈no effect.

**Figure captions**

Figure 1 *upper*: Summary of methodological factors that affect cognitive performance in response to high intensity exercise. *lower*: potential (psycho)physiological factors that mediate cognitive performance.