The effects of low-intensity cycling on cognitive performance following sleep deprivation

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Abstract:

This study examined the effect of 24 h of sleep deprivation on cognitive performance and assessed the effect of acute exercise on cognitive performance following sleep deprivation. Young, active, healthy adults ($n = 24$, 14 males) were randomized to control (age = 24.7 ± 3.7 years, BMI = 27.2 ± 7.0) or exercise (age = 25.3 ± 3.3 years, BMI = 25.6 ± 5.1) groups. Cognitive testing included a 5-min psychomotor vigilance task (PVT), three memory tasks with increasing cognitive load, and performance of the PVT a second time. On morning one, cognitive testing followed a typical night's sleep. Following 24-h of sustained wakefulness, cognitive testing was conducted again prior to and after the acute intervention. Participants in the exercise condition performed low-intensity cycling (~ 40%HRR) for 15-min and those in the control condition sat quietly on the bike for 15-min. *t*-Tests revealed sleep deprivation negatively affected performance on the PVT, but did not affect memory performance. Following the acute intervention, there were no cognitive performance differences between the exercise and rested conditions. We provide support for previous literature suggesting that during simple tasks, sleep deprivation has negative effects on cognitive performance. Importantly, in contrast to previous literature which has shown multiple bouts of exercise adding to cognitive detriment when combined with sleep deprivation, our results did not reveal any further detriments to cognitive performance from a single-bout of exercise following sleep deprivation.

Keywords: Acute exercise | Sleep deprivation | Memory | Attention | Reaction time

Article:

1. Introduction

Prolonged wakefulness, through acute sleep deprivation or sleep restriction, can be detrimental to human performance and cognitive outcomes [1]. Although chronic sleep restriction (i.e. reduced sleep time) is more common, acute sleep deprivation (i.e. no sleep) can be experienced on

occasion and can be common in some occupational fields such as healthcare [2] and transportation [3]. There is substantial evidence showing that sleep deprivation increases the risk for human-error related accidents, with studies consistently showing that acute sleep deprivation causes individuals to perform similarly to intoxicated individuals [4], [5] and with one well-designed study indicating that performance after acute sleep deprivation was similar to performance in individuals with a blood alcohol content of 0.05–0.1% [6]. Considerable research has explored the effects of sleep deprivation on motor function and driving performance ([7], [8]) and has examined its influence on cognition [9]. Studies exploring the effects on cognition have demonstrated that sleep deprivation adversely affects working memory [10], [11], [12], attention [13], [14], and reaction time [12], [14], [15], [16]. It has been suggested that sleep deprivation lowers arousal, which can decrease neural efficiency or capacity (i.e. available cognitive reserves), thus blunting cognitive performance, unless additional arousal is elicited [17], [18]. Acute interventions that can increase arousal and improve cognitive performance, such as exercise, should be considered.

1.1. Cognitive performance and exercise

Though many studies have focused on neurobiological responses to sleep deprivation and have highlighted the negative implications for cognitive performance, few have examined acute interventions to counter the expected deficits. One possible behavioral intervention to consider is acute exercise because of its demonstrated positive effect on cognitive performance and its elevation of arousal. With respect to the effects of exercise on cognitive performance, meta-analyses have found that an acute bout of exercise has a small, but positive effect on cognitive performance by children, young adults, and older adults [19], [20]. Lambourne & Tomporowski suggested that acute exercise may improve rapid decision-making and automatic processing during exercise due to increases in arousal, however the physiological changes that remain after exercise may contribute to enhanced performance on more complex cognitive tasks such as memory processing. Exercise-induced arousal may increase the available reserves for basic attentional processing allowing necessary resources to be allocated for higher-order functioning. In addition to exercise-induced arousal, task complexity may affect task-specific performance outcomes after acute exercise. For example, Chang et al. [21] found an increase in activation in brain regions associated with arousal and higher-order processing after acute exercise in healthy, young adults. However, behavioral improvements were only found during complex tasks. These specific improvements may suggest a ceiling effect for simple tasks, in regard to exercise-induced arousal improvements, in a state where cognitive reserves are not decreased; however, tasks that are complex and elicit higher-order functioning may have a decreased threshold for arousal-induced improvements. According to the cognitive reserve theory, there needs to be some level of detriment to the reserves in order for significant improvements to occur in response to an intervention [22]. The detriment may be derived from the current status and demands placed on the individual, such as sleep deprivation, or by chronic conditions, such as aging or chronic disease. Given this evidence and the predictions of the cognitive reserve theory, it is anticipated that acute exercise may be beneficial for cognitive performance in sleep-deprived individuals in whom reserves are decreased.

1.2. Exercise and sleep deprivation on cognitive performance

Surprisingly, to our knowledge there is no research available on the effects of acute aerobic exercise as an intervention to counteract cognitive performance deficits associated with sleep deprivation. In the sleep deprivation literature, studies that have focused on exercise have used exercise as an additional stressor, and thus have specifically designed the exercise to be particularly challenging when coupled with sleep deprivation. Not surprisingly, these studies have tended to find that exercise combined with sleep deprivation results in negative effects. For example, performing multiple bouts of exercise every 2 h throughout a night of sleep deprivation has been shown to either have no impact on cognitive performance [23] or to negatively impact reaction time, attentional lapses, and mood [24]. Additionally, pairing continuous aerobic exercise with sleep deprivation, during an ultra-endurance event, resulted in deleterious effects on reaction time, attentional lapses, and false alarms during a psychomotor vigilance task [25]. These findings suggest that exercise paired with sleep deprivation may be designed in a way so as to add to the total physiologic load on the mind and body, decreasing performance in simple tasks, rather than increasing available reserves.

Despite this evidence that suggests multiple bouts of acute exercise or continuous exercise coupled with sleep deprivation has a negative effect on cognitive performance, the well-established beneficial effects of exercise in the absence of sleep deprivation suggest that it might be possible to use a single bout of exercise as a means of increasing arousal and, hence, improving cognitive performance following sleep deprivation. Although low, moderate, and vigorous intensity acute aerobic exercise can improve cognitive performance in a variety of populations, a single bout of low intensity exercise may benefit sleep-deprived individuals by increasing arousal and enhancing cognitive reserves, but without taxing the system to the point that deleterious effects on cognitive reserves would be seen. Hence, our purpose was to examine the changes in cognitive performance following 24 h of sleep deprivation and to assess the effects of a single bout of low-intensity exercise during the deprived state. We hypothesized that sleep deprivation would have a detrimental effect on cognitive performance, with greater effects on simple tasks, and that a single bout of low-intensity cycling would counter those deficits.

2. Method

2.1. Participants

Participants consisted of 30 young adults volunteers recruited from undergraduate and graduate classes and the community. Inclusion criteria were that participants were required to have a regular sleep pattern (self-reported 8 or more hours of sleep per night with onset typically occurring between 10 pm to midnight during the previous week), to self-report 6–8 h of sleep the night prior to participation in the study (i.e., not to be acutely sleep deprived before the beginning of the study), and to be recreationally active (self-report 30 min per day, 5 days per week, for the past 3 months). One participant was excluded for being acutely sleep deprived, two participants were excluded due to technical issues with sleep monitoring, and three participants were excluded for unforeseen events, which led to termination of testing during night two. The final sample consisted of 24 young adults (20–30 years). Participants were asked to refrain from participation in exercise 24 h prior to the first testing session and to abstain from caffeine prior to testing on morning one. All other typical daily patterns were maintained. Based upon self-reported medication use, none of the participants were taking any medications that would be

expected to influence sleep and self-reported caffeine intake between morning one and evening two ranged from 0 to 4 caffeinated beverages (M = 0.625 SD = 1.21). The University of North Carolina at Greensboro Institutional Review Board approved the testing protocol and all participants gave written informed consent and completed demographic questionnaires prior to participation.

2.2. Protocol

2.2.1. Overview

This study required 48 h of data collection for each participant. Prior to the start of the study, participants met with researchers for consenting, explanation of procedures, and questionnaires. Their activity and sleep were tracked with an accelerometer from that point until the completion of the study. After a typical night's rest at home (evening one), participants came to the lab for morning one procedures. After testing, the participants went about their typical day, avoiding napping or exercise, and returned to the lab that evening for overnight monitoring (evening two). Following 24 h of sustained wakefulness, morning two testing began. Morning two consisted of two cognitive testing sessions, one prior to and one after the intervention. Participants either exercised in between cognitive testing sessions or rested. The protocol is described in more detail below and depicted in Fig. 1.
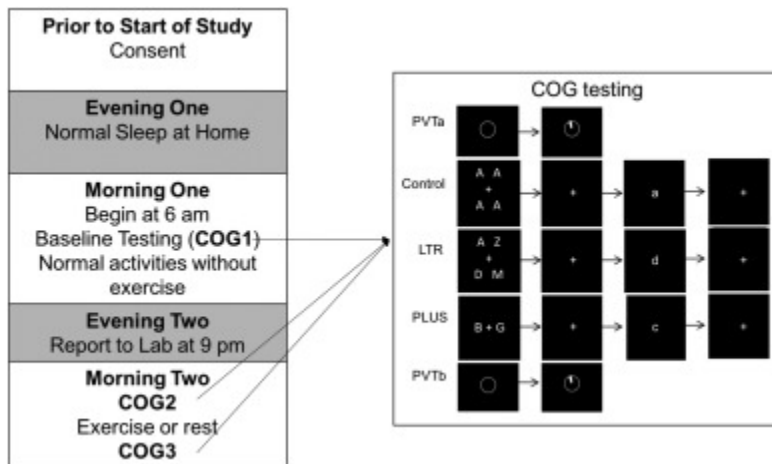

Fig. 1. Testing protocol with a visual display of the cognitive (COG) testing battery including Sternberg memory tasks (Control, LTR, PLUS) and psychomotor vigilance tasks (PVTa, PVTB) in testing order with examples for the Sternberg memory task indicating situations when the participant should respond that the letters match.

2.2.2. Evening one

All participants were given an Actigraph GT9X Link accelerometer (Actigraph, Pensacola, FL) when they were consented for participation to wear during the day and night prior to morning one and throughout testing in order to ensure compliance with activity and sleep requirements. Accelerometers were set to collect at 30 Hz for 48 h. The information was downloaded and analyzed with the Actilife software (Actigraph, Pensacola, FL). Sleep scoring was performed using the Sadeh algorithm for sleep detection [26]. Total sleep time on Evening One was a

primary variable of interest to ensure compliance, however participants were also asked "What time did you go to bed?" and "What time did you wake up?".

2.2.3. Morning one—baseline testing

Testing on morning one began at 6 am and consisted of 90 min of baseline testing. Baseline testing included an orthostatic challenge (assessing heart rate variability and resting heart rate in the seated position), cognitive testing (COG1), and balance testing; this paper will only report and discuss the cognitive findings. After baseline measures, participants were excused from the lab and reminded to participate in their typical daily activities while refraining from exercise.

2.2.4. Evening two—sleep deprivation

Participants reported back to the lab at 9 pm for overnight observation by research assistants who were instructed to ensure that participants did not fall asleep. Water was allowed all night, however after 11 pm other food or other beverages were prohibited until the completion of testing on the following day due to interference with biological samples collected for an ancillary study (not reported here). During the night, participants worked on school work, read, played video games, and socialized with each other (2–4 participants and 1 research assistant were at each session).

2.2.5. Morning two—intervention

24 h after baseline testing, participants were tested again on all baseline measures (COG2) presented in the same order. Participants were randomized to either exercise or control (no exercise) conditions. Exercise participants completed 15 min of low-intensity (35–40% heart rate reserve [HRR]) cycling on a Lode Excalibur Sport ergometer (Lode, Groningen, The Netherlands). The HRR range was calculated as: (% intensity $*$ $(220 - \text{age} - \text{HR}_{rest})) + \text{HR}_{rest}$. Control participants sat quietly on the cycle ergometer for 15 min with their feet in the pedals. Within 5 min of intervention completion, all participants completed the cognitive battery for a third time (COG3).

2.3. Cognitive battery

The cognitive battery was computer administered using E-prime (Psychology Software Tools, Inc., Sharpsburg, PA) and consisted of a psychomotor vigilance task (PVTa), three Sternberg memory tasks, and a second PVT (PVTb). The order of the cognitive battery along with a visual display of each task is outlined in Fig. 1.

2.3.1. Psychomotor vigilance task

Each PVT was 5 min in duration and participants were asked to respond by clicking the mouse as quickly as possible as soon as they noticed that the empty circle was beginning to fill in. This task was adapted from one originally used by Dinges & Powell [27] and used in its adapted form by Correa et al. [7] and by Gonzalez-Fernandez et al. [28]. The appearance of the stimuli occurred at varying intervals of 2 to 10 s. Performance was assessed using three different metrics

consistent with previous research [29], [30], [31]. Reaction time (ms) was averaged across trials if the time was > 100 ms. If reaction time was < 100 ms, the trial was recorded as a false alarm. If reaction time was > 500 ms the trial was counted as a lapse.

2.3.2. Working memory

Working memory performance was assessed by three Sternberg memory tasks; Control (CTL), Letter (LTR), and PLUS. The CTL and LTR tasks assessed maintenance of verbal working memory [12], [32]. For these tasks, four white capital letters were presented at the corners of an imaginary square on a black screen with a fixation point (+) in the center. After 500 ms, the letters disappeared and left only the fixation point. After 300 ms (CTL) or 3000 ms (LTR), a single lower case white letter appeared in place of the fixation point for 1500 ms and participants were instructed to click the left mouse button if the lower-case letter matched the upper-case letters and to click the right mouse button if they did not match. During the control task, the four capital letters were all the same. During the LTR task, the four letters were different.

The PLUS task also assessed maintenance of verbal working memory with the additional challenge of item manipulation [11]. During this task, two white capital letters were presented to the left and right of a fixation point on a black screen. After 500 ms seconds the letters disappeared, leaving the fixation point. After 3000 ms seconds a lowercase white letter replaced the fixation point for 1500 ms seconds and participants responded. Participants were instructed to advance the initial capital letters to remember the letters that succeeded them in the alphabet and then to identify whether or not the lowercase letter matched these new letters. Accuracy as a percentage and reaction time (ms) were used to assess memory on all three tasks [11].

2.4. Statistical analysis

All analyses were conducted using SPSS Version 23 (IBM Corp., Armonk, NY). Independent t-tests were utilized to ensure successful randomization of intervention groups based on participant demographics and sleep parameters. The effects of sleep deprivation on cognitive performance were analyzed using paired t-tests to investigate whether sleep deprivation had an effect on cognitive performance from morning one (COG1) to morning two (COG2). Subsequent analyses designed to examine the combined effects of sleep deprivation and exercise on cognitive performance were based on a mixed design to assess intervention effects. An analysis of covariance was used for each cognitive testing measure to assess whether low-intensity exercise affected expected detriments from sleep deprivation. Performance at COG2 should not have differed between the two intervention groups (exercise and control) because each participant was equally exposed to sleep deprivation at this time point. For this reason, performance at COG2 was used as a covariate while examining intervention effects (COG3). Alpha was set at $p = 0.05$.

3. Results

Table 1 includes participant demographics and sleep parameters. None of the measures were significantly different between the groups (all $p > 0.05$).

Table 1. Participant demographics.

Acute intervention group	Rest	Exercise
Participants total (n males)	12 (7)	12 (7)
Mean age in years (SEM)	24.67 (1.05)	25.25 (0.95)
Mean BMI (SEM)	27.19 (2.01)	25.62 (1.46)
Mean hours sleep (SEM)	5.32 (0.29)	5.43 (0.22)
Mean hours reported sleep (SEM)	7.03 (0.21)	6.71 (0.24)

SEM: standard error of the mean. Mean hours sleep determined with accelerometer and mean hours reported attained from self-report questionnaire.

3.1. Effect of sleep deprivation on cognitive performance

Sleep deprivation did not affect reaction time during PVTa ($p = 0.074$), but had a significant deleterious effect on reaction time during PVTb ($p = 0.000$) (Fig. 2).
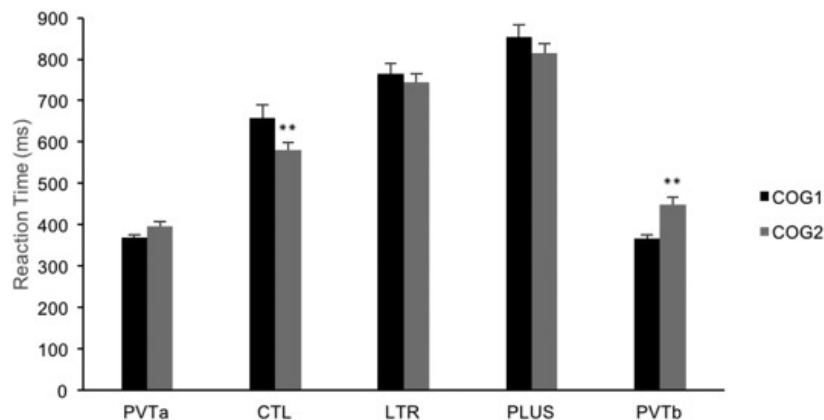

Fig. 2. Reaction time during psychomotor vigilance tasks (PVTa, PVTb) and Sternberg memory tasks (CTL, LTR, PLUS) at baseline (COG1) and after 24 h of sleep deprivation (COG2). Error bars reflect standard error ($n = 24$). $**p < 0.01$.
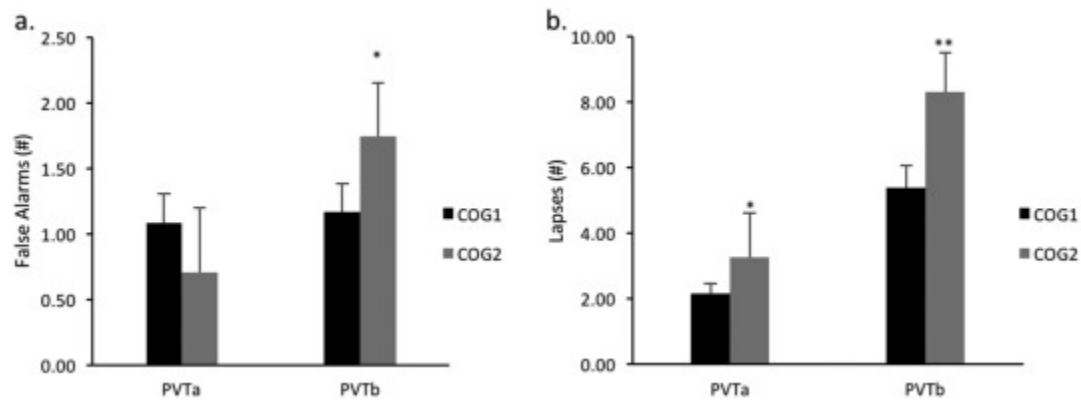
Fig. 3. a) Number of false alarms and (b) lapses during psychomotor vigilance tasks (PVTa, PVTb) at baseline (COG1) and after 24 h of sleep dep (COG2). Error bar reflect standard error ($n = 24$). $**p < 0.01$, $*p < 0.05$.

Additionally, false alarms were not significantly affected after sleep deprivation during PVTa ($p = 0.885$), but were significantly increased during PVTb ($p = 0.016$) (Fig. 3a). Lapses during both PVTa and PVTb were significantly increased ($p = 0.015$, $p = 0.000$, respectively) (Fig. 3b).

Though reaction times appeared to decrease across all Sternberg memory tasks in response to sleep deprivation, the only task for which this reached significance was the control condition ($p = 0.008$). There were no significant changes in accuracy as a function of sleep deprivation (see Table 2).

Table 2. Effects of sleep deprivation.

	COG1 (SE)		COG2 (SE)		t (23)	p value
Reaction time (ms)						
PVTa	368.78	(6.68)	395.69	(12.65)	− 1.87	0.074
CTL	658.17	(31.91)	579.62	(19.79)	2.92	**0.008****
LTR	764.53	(25.87)	744.30	(20.21)	0.95	0.351
PLUS	854.51	(28.35)	813.81	(23.62)	1.43	0.166
PVTb	367.83	(7.42)	449.80	(17.84)	− 4.78	**0.000****
Accuracy (%)						
CTL	0.91	(0.03)	0.96	(0.01)	− 1.80	0.085
LTR	0.88	(0.02)	0.86	(0.03)	0.63	0.537
PLUS	0.70	(0.03)	0.68	(0.04)	0.80	0.431
False Alarms						
PVTa	1.08	(0.22)	1.17	(0.49)	− 0.15	0.885
PVTb	0.71	(0.22)	1.75	(0.40)	− 2.59	**0.016***
Lapse						
PVTa	2.17	(0.30)	5.38	(1.36)	− 2.64	**0.015***
PVTb	3.25	(0.68)	8.29	(1.20)	− 4.82	**0.000****

Means (standard error) for reaction time of all cognitive tasks, number of false alarms, and number of lapses during PVT, and accuracy during memory tasks on morning one (COG1) and morning two (COG2). Bold p values indicate a statistically significant difference between COG1 and COG2.
* $p < 0.05$.
** $p < 0.01$.

Table 3. Effects of the intervention.

	SD + rest (SE)		SD + exercise (SE)		F (1, 21)	p value
Reaction time (ms)						
PVTa	396.88	(13.91)	388.50	(13.91)	0.16	0.689
CTL	544.71	(15.46)	531.23	(15.46)	0.37	0.550
LTR	721.47	(20.36)	756.80	(20.36)	1.48	0.237
PLUS	771.33	(31.74)	778.41	(31.74)	0.02	0.878
PVTb	461.37	(20.18)	452.67	(20.18)	0.09	0.764
Accuracy (%)						
CTL	0.95	(0.01)	0.97	(0.01)	0.92	0.348
LTR	0.86	(0.04)	0.82	(0.04)	0.58	0.454
PLUS	0.70	(0.04)	0.70	(0.04)	0.00	0.982
False alarms						
PVTa	1.94	(0.45)	0.73	(0.45)	3.65	0.070
PVTb	1.97	(0.51)	1.11	(0.52)	1.35	0.259
Lapses						
PVTa	4.29	(1.15)	4.55	(1.15)	0.02	0.880
PVTb	9.79	(1.34)	8.54	(1.35)	0.43	0.521

Means (standard error) for reaction time of all cognitive tasks, number of false alarms, and number of lapses during PVT, and accuracy during memory tasks (COG3) after combining sleep deprivation with rest (SD + rest) or exercise (SD + exercise).

3.2. Effects of acute exercise following sleep deprivation

There were no intervention effects for any of the reaction time measures on either of the PVTs or the Sternberg memory conditions ($p > 0.05$). The number of false alarms and lapses during PVTa and PVTb were not different between the conditions. In addition, accuracy on the Sternberg memory conditions was not affected by intervention ($p > 0.05$) (see Table 3).

4. Discussion

4.1. Effects of sleep deprivation on cognitive performance

After 24 h of sleep deprivation, performance during the PVT was negatively affected. Specifically, detrimental effects to reaction time, false alarms, and lapses were apparent and these detriments were more pronounced after also completing the Sternberg memory tasks. These findings are consistent with our hypotheses and support the theoretical framework of the cognitive reserve theory. Congruent with previous research, it appears that sleep-deprivation decreases available cognitive reserves, negatively affecting simple, monotonous tasks that do not elicit task-induced arousal [10], [30] and further increasing the cognitive load (i.e. through the addition of three memory tasks) decreased reserves yielding a greater detriment on PVTb. Although not assessed in this study, these findings are also consistent with previous neuroimaging studies investigating the role of thalamic activation in explaining the link between arousal and attention [10], [18]. In contrast to the negative effects seen on the PVT, we do not provide evidence of sleep deprivation being detrimental to complex working memory tasks. Reaction time and accuracy during the LTR and PLUS tasks were unchanged from morning one to morning two testing. This is consistent with past literature in that complex working memory tasks have been shown to be more resilient to sleep deprivation [10]. The threshold where thalamic activation can compensate for a decreased reserve state may be influenced by task complexity. Performance on complex tasks such as the Sternberg memory tasks, in a sleep-deprived state may not be as vulnerable to detriment due to a higher threshold for thalamic compensation prior to decreasing performance in response to increasing cognitive load.

4.2. Effects of acute exercise following sleep deprivation

Given the prior findings of increased arousal preserving cognitive performance on complex tasks, we aimed to investigate an acute intervention that has been shown to increase arousal and activation of the thalamus. Exercise has been suggested as an arousal-provoking activity [20] and it has been shown that an acute bout of cycling can increase activation of the alerting network which includes the thalamus, right frontal cortex, right parietal cortex [21]. We expected exercise to positively affect cognitive performance detriments following sleep deprivation. Interestingly, there were no significant differences between the control or exercise groups on any of the cognitive performance measures. Importantly, though unexpected, these results indicate that a single bout of low-intensity exercise can be performed without further impairing cognitive performance after 24 h of sleep deprivation.

4.3. Strengths and limitations

This study has some strengths and limitations that should be discussed. In terms of strengths, to our knowledge this is the first study to purposefully investigate how a single bout of low-intensity exercise affects cognitive performance in a sleep-deprived state. Additionally, a strength of this study is the inclusion of a control group. Initial studies were paramount to our understanding of the effects of exercise on cognitive performance when sleep-deprived, however these studies have been limited by the failure to include a control group. By including a control group in this study, we provide a valuable extension to the existing literature.

Despite notable strengths, this study is not without its limitations. First, there was high variability in the performance changes from morning one to morning two, which may suggest that there is a wide range of sensitivity to sleep deprivation. Research has shown that the physiological response to sleep deprivation may be highly stable and trait-like despite significant inter-individual variability [33]. This suggests that while the between-person variability is large, the response within a single individual is quite stable. If this is true, then the cognitive responses should also be stable across time, suggesting that there may be individuals for whom sleep deprivation is problematic while it is less so for others. Barclay & Myachykov [34] found that 18 h of sustained wakefulness had less of a detrimental effect on attention in evening-type participants compared to morning-type participants. This moderation effect of chronotype suggests the importance of considering this potential moderator during sleep deprivation research and our failure to do so is a shortcoming of this study. An additional limitation of this study is the reliance on self-report measures of typical behavior (sleep patterns and exercise). It is possible that sleep and/or exercise information was misreported which may have influenced the assumed homogeneity of the sample. We expected that our participants were regularly physically active and had a regular sleep pattern and this was important, particularly given our small sample size, as a way of controlling for variability in responses to sleep deprivation and/or exercise.

Additionally, although thalamic activation has been identified as a potential mediator of the arousal and attention [18], we did not use fMRI in this study. To better understand the potential mechanisms underlying the detrimental effects of sleep deprivation on arousal and cognitive performance, the moderating role of task complexity in this relationship, and the potential compensatory benefits of exercise-induced arousal following sleep deprivation, future research would benefit from including a measure of arousal such as heart rate or perceived arousal and should also assess thalamus activation.

It would also be worthwhile for future research to investigate the implications of varying exercise intensities following sleep deprivation. We selected low-intensity exercise because we anticipated that this may improve cognitive performance by increasing cognitive reserves rather than hindering cognitive performance by adding an additional drain on cognitive reserves. This is the first study to investigate a single bout of exercise to counter the effects of sleep deprivation on cognitive performance and more research is needed to further our understanding of whether or not exercise can be implemented to counter cognitive deficits as a result of sleep deprivation. A major shortcoming here is our sample size that was adequately powered to be sensitive to moderate size effects from sleep deprivation on cognitive performance and large effects of group differences in cognitive performance following sleep deprivation and exercise or rest. It is possible that the effects of exercise may be at a smaller magnitude than anticipated, and that we were therefore unable to detect differences given the relatively small sample size. Future

research should include samples that are sensitive to smaller-sized effects or incorporate larger samples.

Also, the cognitive load may not have been great enough to elicit the performance detriments typically shown after sleep deprivation or to be sensitive to the benefits of exercise. Others have used similar tasks with sleep deprivation, but have included multiple exposures to the tests during each testing session [11]. In this study, each testing session was comprised of one experimental period including a single test for each of the memory tasks. Though this did seem to increase cognitive load, shown by decreased performance at PVTb, we did not see any detriments during any of the memory tasks and this is not consistent with previous findings.

Finally, the main goal of this study was to assess the effects of acute exercise and sleep deprivation on cognitive performance and task measures were chosen based on the sleep deprivation literature. The effects of sleep deprivation on cognitive performance has previously been assessed using the PVT [35] and Sternberg memory tasks [10], [11]; However, exercise may not be as beneficial to performance on those tasks as compared to tasks highly sensitive to acute exercise such as the Stroop color and word tasks, choice reaction time, and free recall [19]. Future research may consider using more exercise-sensitive tasks, however sensitivity to sleep deprivation may be reduced.

5. Conclusion

To our knowledge, this is the first study to investigate the effects of low-intensity acute exercise on cognitive performance following 24 h of sleep deprivation. We provide support for the previous literature of sleep deprivation having negative effects on cognitive performance during simple tasks. However, our results did not show that acute low-intensity exercise benefits vigilance performance. Of importance, a single bout of low-intensity exercise does not appear to add any further detriment to cognitive performance after sleep deprivation which suggests that individuals may perform low intensity exercise without exacerbating cognitive deficits from sleep deprivation. If future studies, with larger samples, find support for benefits of low-intensity exercise on particular cognitive measures (e.g., a reduction of false alarms), this could be very important for improving performance for those who experience sleep deprivation. Occasional sleep deprivation is experienced by many individuals in the fields of healthcare and transportation, and future studies are needed to identify interventions to minimize human-error related accidents which are primarily associated with impaired cognitive performance.

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