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# The Impact of Physical Activity and Sleep on Physiology Following a mTBI

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ABSTRACT

***International Journal of Exercise Science 12(3): 919-931, 2019.*** A mild traumatic brain injury (mTBI) is a common injury that happens to people across a range of lifestyles. Rest has been regarded as the most effective remedy to restore impaired physiological functions in those who have experienced this kind of injury. However, recent data suggests that a gradual increase in physical activity post-injury can lead to quicker recovery from negative symptoms. The impact of physical activity on physiologic recovery is not completely understood and the purpose of this study was to better understand how physical activity and sleep influence physiological outcomes following a mTBI. The data collected in this study came from two groups of young adults: a healthy control (n=5) and those that had a mTBI ($n$ = 5). The daily activity was measured using an activity monitor for a week immediately after injury. Physiological measurements of excitability and inhibition within the motor cortex and neurotransmitter concentrations were collected within 72 hours of injury and again at two months to determine if physical activity or sleep within one week after injury had an influence on physiologic recovery. Control participants had higher levels of total ($d$ = 1.2), light ($d$ = 1.3) and moderate physical activity ($d$ = 0.7), and lower total sleep time ($d$ = -1.9), compared with the mTBI group. Across the two months, relative to the control group the mTBI group showed similar changes in excitability ($d$ = 0.13), but greater changes in inhibition ($d$ = -0.66). Changes in the excitatory ($d$ = 0.06) and inhibitory ($d$ = -0.16) neurotransmitters were similar between groups. Neither physical activity, nor sleep quality were significantly associated with the change in motor cortex excitability ($R^2 \leq 0.18$, $p \geq 0.22$), inhibition ($R^2 \leq 0.3$, $p \geq 0.08$) or neurotransmitter concentrations ($R^2 \leq 0.15$, $p \geq 0.20$) between 72 hours and two months post-injury. These preliminary results suggest there is no direct effect of physical activity on physiological outcomes.

KEY WORDS: Mild-traumatic brain injury, concussion, transcranial magnetic stimulation, motor evoked potential

## INTRODUCTION

Mild traumatic brain injuries (mTBIs) have a variety of negative impacts on physiology. MTBIs are clinically characterized by immediate impairments in brain function, including alteration of mental status or a loss of consciousness for less than 30 minutes, resulting from a mechanical force or trauma to the head (12) The resulting physical symptoms include disorientation, dizziness, headache, and visual sensitivities (12). For the majority of patients who suffer a mTBI, the symptoms resolve within the first week to 10 days (33). However, physiological symptoms such as higher levels of inhibition within the motor cortex can persist for months or years after

injury (3, 31). A mTBI can be challenging for healthcare professionals to identify and manage because of variations in symptoms and recovery. Therefore, identifying factors that impact recovery is critically important to providing high-quality care for patients with mTBI.

An increase in sleep has been suggested by clinicians to be an effective remedy for restoring impaired function to the brain, followed by a gradual return to activity (41). Sleep is critical in the metabolic homeostasis and its restoring properties may be related to the removal of neurotoxic waste that accumulates in the brain and central nervous system (22). In recent years, studies have begun to challenge the prescription of prolonged rest as a treatment for mTBI. Instead of maximum rest, some medical organizations are now suggesting that individuals engage in limited cognitive and physical activity shortly after injury (5). It has been shown that limited physical activity may decrease the time of recovery of negative cognitive and physical symptoms following a mTBI (22, 24, 28). However, the impact of sleep and physical activity on physiological outcomes of mTBI is unknown. A better understanding of the impact of sleep and physical activity on objective, physiological outcomes following mTBI can help to inform and improve recovery management.

Studies of rodent models of mTBI suggest that within days of an initial injury, there is a large release of the excitatory neurotransmitter glutamate, leading to a hyper-excitable state in the brain. This phase is followed by a longer-term spreading depression phase, involving inhibitory neurotransmitters such as GABA (12). These results in rodent models are supported by studies using Transcranial Magnetic Stimulation in humans. Using this technique, excitability of the motor cortex, as indicated by the motor evoked potential amplitude, has been shown to be lower in individuals with mTBI when compared to healthy individuals at 2 weeks post injury (6, 7). Further, inhibition within the motor cortex, as indicated by the cortical silent period, has been shown to be higher in individuals who have had a mTBI, weeks, months and years after injury (20, 24, 30). While these studies indicate that the concentration and actions of excitatory and inhibitory neurotransmitters in the brain may provide a physiological indication of mTBI, the factors affecting recovery of these aspects of injury remain unknown. It is possible that elements such as sleep and physical activity, which may impact recovery from mTBI, influence these underlying physiological consequences.

Although limited physical activity following an mTBI has been shown to be beneficial for cognitive and physical symptom recovery (13), its influence on the physiological consequences is not known. Therefore, the purpose of this pilot study was to explore the relationship between physical activity and sleep levels within the first week post-injury and physiological outcomes at two months post-injury. It was hypothesized that individuals who had more sleep and more physical activity would show greater improvements in neurotransmitter concentrations, excitability, and inhibition of the motor cortex at two months post-injury.

## METHODS

*Participants*

Ten individuals, (mean age = 21 ± 2.1 years) participated in this pilot study. This sample included five individuals who had been diagnosed by a medical professional as having suffered a mTBI, and five healthy age and sex-matched control participants who had no history of diagnosed brain injury. Clinical diagnosis of mTBI was made by an athletic trainer or a physician base on the injury description (immediate impairments in brain function, including alteration of mental status or a loss of consciousness for less than 30 minutes, resulting from a mechanical force or trauma to the head (12) and neurological testing including myotome, dermatome, and cranial nerve evaluations. Participants diagnosed with a mTBI came into the lab within 72 hours of the head injury and again at two months post injury. Control participants also completed two testing sessions, separated by two months.

Consistent with previous studies of the effects of mTBI (14, 29), and the understanding that approximately 90% of mTBIs occur without a loss of consciousness (16, 17, 31), individuals who suffered an mTBI must not have lost consciousness for more than one minute at the time of injury to be eligible for this study. Injuries occurred from rugby ($n$ = 2), ice hockey ($n$ = 1), bike crash ($n$ = 1), and basketball ($n$ = 1). For both groups, participants were excluded from the study if they 1) had experienced another mTBI within the past year prior to testing, or more than two mTBIs total in their lifetime (mTBI group), or one mTBI in their total lifetime (control group); 2) had a history of cognitive/attention deficiencies, independent of the injury; 3) had a history of other neurological impairment, musculoskeletal impairments, or seizures; 4) were taking medications known to affect brain neurotransmitter function; or 5) reported any contraindications to the use of transcranial magnetic stimulation and/or magnetic resonance measures. The study procedures were reviewed and approved by the Institutional Review Board and all participants provided written informed consent prior to beginning study. All data collection sessions were conducted through the Neurophysiology Laboratory at the University of Oregon. Participants were informed of the procedures but were not informed of the hypotheses of the study. Further, all data analyses were performed by the same experimenter, who was blinded to the group (control or mTBI) of the participants during analysis.

*Protocol*

At the first testing session, participants were provided with an accelerometer (GT3X, Actigraph, Pensacola, FL), which they wore on the wrist of the non-dominant hand for one week (1, 24). Data were collected in 60-s epochs on a 24-hour time scale to monitor levels of activity and sleep patterns (37). The first five full days of activity and sleep were analyzed using ActiLife software (Actigraph, Pensacola, FL). Physical activity was evaluated for total physical activity counts, and time spent in light, moderate, and vigorous activity, using established, validated cutoff values according to Freedson 1998 (11, 24). Sleep efficiency was calculated based on accelerations used to determine sleep time, number of wake times and duration of each wake period throughout the night. These sleep calculations were accomplished with ActiLife software, which provides a valid assessment of sleep time and overall sleep quality (36, 40). Physical activity and sleep values were averaged across the five days for each participant.

All evoked potentials were recorded with surface electromyography (EMG) electrodes placed over the first dorsal interosseous (FDI) muscle on the dominant hand. The area was exfoliated with NuPrep and cleaned with an alcohol wipe prior to placement of the electrode to prevent potential signal disturbances. A bipolar Ag-AgCl electrode (DE-2.1, Delsys Inc., Boston, MA), with an inter-electrode distance of 1 cm was then secured to the FDI of the same hand, which was connected to an amplifier (Bagnoli, Delsys Inc., Boston, MA). A ground electrode was placed on the posterior position of the distal ulna. The signals were acquired 5 kHz with a 16-bit A/D converter (NI USB-6251, National Instruments, Austin, TX) using DasyLab software (Dasytec USA Inc., Amherst, NH).

Motor evoked potentials were induced in the FDI with transcranial magnetic stimulation (TMS), using a flat 70-mm figure-of-eight coil (MagStim 200$^2$, MagStim Company, Ltd., Whitland, UK). The position of optimal stimulation was found by locating the site on the head that induced the largest voltage of MEP response in the FDI muscle. The resting motor threshold (RMT) was determined by decreasing the stimulation intensity incrementally until the minimum level that induced a MEP of at least 50 μV in five out of ten trials was determined (10, 33). The stimulus was then set to 120% of the RMT to evoke six MEPs' of the FDI, with 10-15 seconds of rest between stimulations. The amplitude of the resting MEP was quantified using a custom-written program in MATLAB software (Mathworks, Natick, MA). All analyses were performed by the same individual for consistency.

The maximal voluntary contraction (MVC) force of the FDI was assessed by having participants push the index finger against a force transducer (MBP-5, Interface, Scottsdale, AZ) with maximum effort. As individuals with mTBI were tested within 72-hours after injury, it was important to minimize the total testing time. Therefore, the FDI, was chosen due to the relative ease of evoking responses in this muscle via TMS, compared with other muscles, limiting testing time and the total number of stimulations required. Further, previous TMS work in individuals with mTBI has focused on the FDI, and results of excitability and inhibition could therefore be compared with previous studies (2, 3, 7, 26, 27). Participants completed three trials of maximal effort, each lasting approximately 5 seconds, and separated by two minutes. The highest value across trials was taken as the MVC. This MVC value was not an outcome variable of the study, rather it was used to set a relative target force level for participants while evoking the cortical silent period (CSP). To evoke the CSP, participants were asked to apply force to 50% of their MVC and were provided with visual feedback. During the 50% contraction, TMS stimulation was delivered to the motor cortex at 120% RMT. The participants were asked to keep applying the force through the stimulation until the experimenter told them to relax. Six trials were obtained, with ~15 seconds rest between trials. The duration of the CSP was determined manually using a custom-written program in MATLAB. The CSP was measured from the end of the MEP to the beginning of the resumption of EMG activity (10, 31). For consistency, the same individual performed all analyses.

The excitatory and inhibitory neurotransmitters, glutamate and GABA, respectively, were measured using a 32-channel phased array head coil in a 3T MRI system (Skyra, Siemens, Munich, Germany). A functional localizer task where participants tapped the index finger of

their dominant hand for a total of 3 minutes was used to identify the location of the primary motor cortex. A 16x16x16mm voxel was then placed over this region for spectral analysis. A single-voxel PRESS sequence (TR/TE = 1500/30ms) [16] was used to assess glutamate and an adapted MEGA-PRESS sequence for GABA (TR/TE = 2000/68ms) (35). The area under glutamate (2.4 ppm) and GABA (3.0 ppm) peaks were calculated and expressed relative to total creatine using LCModel (Provencher). The concentration of creatine and phosphocreatine were taken to normalize the concentrations of the two target neurotransmitters.

*Statistical Analysis*

Due to the small sample size in this pilot study, all comparisons between groups were accomplished by calculating the effect sizes (Cohen's *d*). The effect size provides an indication of the magnitude of the difference in means and is less confounded by small sample sizes than tests of statistical significance (8). Cohen's *d* indicates a small effect when above 0.2, a medium effect when above 0.5 and a large effect when above 0.8 (8) and was calculated here where a positive *d* represents a higher mean in control participants than the mTBI group and a negative *d* indicates a lower mean in control participants than the mTBI group. Effect sizes were used to determine the difference between groups in age, height, weight, total, light, and moderate physical activity, as well as sleep efficiency and total sleep time. MEP amplitude, CSP duration, and glutamate and GABA concentrations were also compared between groups at the 72-hour and 2-month time points. Finally, the percent change in MEP amplitude, CSP duration, and glutamate and GABA concentrations from baseline (72 hours post-injury for individuals with mTBI) to the two-month testing session were calculated and were compared using Cohen's d. Relationships between each of the physical activity and sleep measures, and each of the physiological measures were examined with linear regression analyses. All analyses were performed with SYSTAT 13 (Crane Software International Ltd., Chicago, IL). All data are presented as mean ± SD, and significance for regression analyses was set at $p \leq 0.05$.

## RESULTS

Participant characteristics are shown in Table 1. The participants in the mTBI group were moderately older than those in the control group (*d* = -0.50), with only a small effect of group for weight (*d* = -0.27) and no effect for height (*d* = 0.13).

**Table 1.** Physical profile of healthy and concussed participants.

| Group | Age (years) | Height (cm) | Weight (kg) | Sex (M:F) |
|---|---|---|---|---|
| Control | 20 ± 1.1 | 173 ± 15 | 75 ± 16 | 3:2 |
| mTBI | 21 ± 2.9 | 171 ± 15 | 79 ± 14 | 3:2 |
| *d* | -0.5 | 0.13 | -0.27 | |

*Note.* mTBI = mild traumatic brain injury; *d* = effect size, where a positive effect reflects a higher value in the control group compared with the mTBI group; M = male; F = female

Activity and sleep data are shown in Table 2. Control participants had higher levels of total, light and moderate physical activity compared with the mTBI group. The effect for total (*d* = 1.20) and light (*d* = 1.33) physical activity counts was large, while the effect for moderate physical activity (*d* = 0.70) was moderate. The average total sleep time was greater in the mTBI group

compared with controls, and this was a large effect (*d* = -1.90). Sleep efficiency, however, had a small effect size (*d* = -0.39), as it was only slightly higher in the mTBI group compared with controls.

**Table 2.** Average activity and sleep for first five days post-injury.

| Group | Total (counts) | Light (counts) | Moderate (counts) | Sleep (min) | Sleep Efficiency (%) |
|---|---|---|---|---|---|
| Control | 702 | 488 | 215 | 440 | 92 |
| mTBI | 481 | 329 | 152 | 640 | 95 |
| *d* | 1.2 | 1.3 | 0.70 | -1.90 | -0.39 |

*Note.* mTBI = mild traumatic brain injury; *d* = effect size, where a positive effect reflects a higher value in the control group compared with the mTBI group

The MEP amplitude was higher in the mTBI group than controls at both testing times (Table 3). This effect was small at the 72-hour testing time (*d* = -0.44) but was large at two months post-injury (*d* = -1.37). The MEP amplitude in the control group increased by 50% across the two testing sessions and decreased by 37% in the mTBI group (Figure 1). This group difference in the percent change in MEP across time, however, was only a small effect (*d* = 0.13)

The duration of CSP was higher in the mTBI group than control at both testing times (Table 3). This effect was large at the 72-hour testing time (*d* = -0.96) and at two months post-injury (*d* = -1.53). The control group showed an overall decrease in CSP by 17% and the mTBI group showed an increase by 28% (Figure 2). This group difference in change in CSP across the two months, was a medium sized effect (*d* = -0.66).

The concentration of glutamate and GABA in the primary motor cortex showed considerable changes over time. The concentration of glutamate was moderately higher in the control group at the 72-hour testing times (*d* = 0.65) with only a small difference at two months (*d* = 0.30) (Table 3). The concentration of glutamate decreased in the control group by -4.8% and the mTBI group by -4.9%. The effect size for the difference between groups in the change in glutamate was very small (*d* = 0.06) (Figure 3). The concentration of GABA was not largely different between groups at the 72-hour (*d* = 0.12) or two-month (*d* = -0.16) testing times (Table 3). The control group showed an overall increase in GABA concentration by 6% and the mTBI group, a decrease by 2%. Similar to the overall effect size in glutamate, the effect size of the difference between groups in the change in GABA concentrations was very small (*d* = -0.16) (Figure 4).

Overall, there were no significant relationships between physical activity or sleep and physiological outcomes (Table 4). However, the relationship between CSP duration and moderate physical activity approached significance (*p* = 0.08), suggesting a trend towards a relationship between physical activity and cortical inhibition.

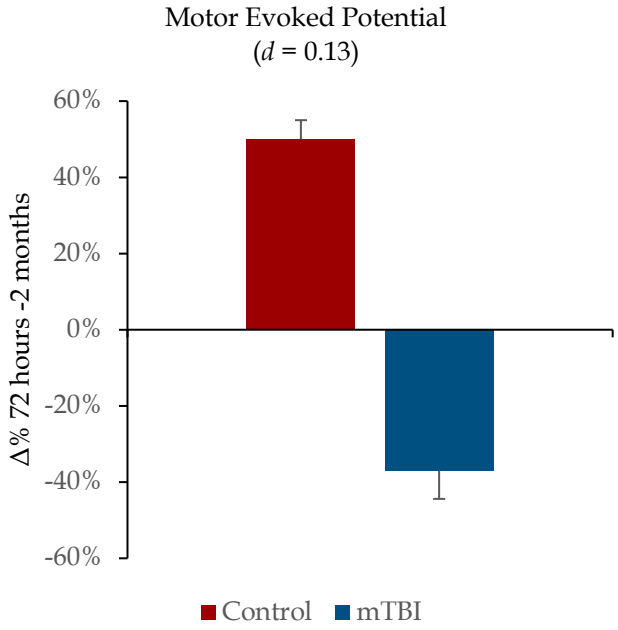


**Figure 1.** Percentage change in motor evoked potential from 72 hours to 2 months.

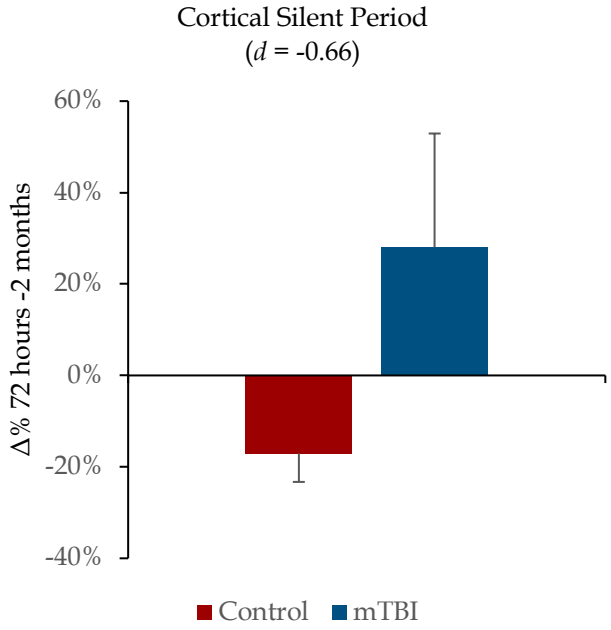


**Figure 2.** Percentage change in cortical silent period from 72 hours to 2 months.

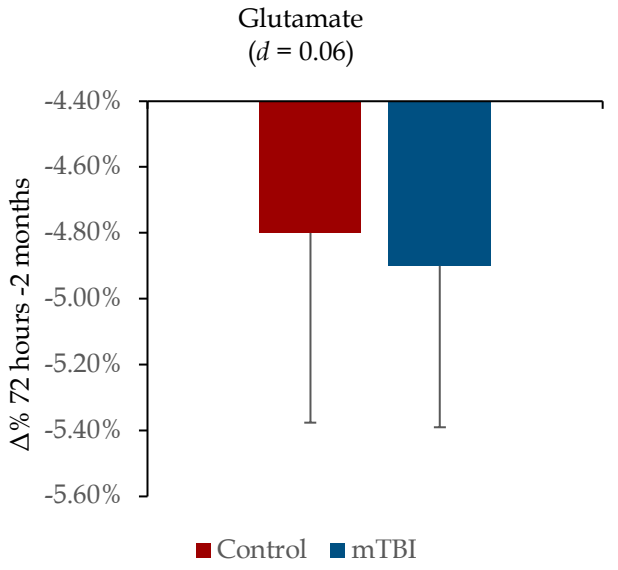


**Figure 3.** Percentage change in glutamate concentration from 72 hours to 2 months.

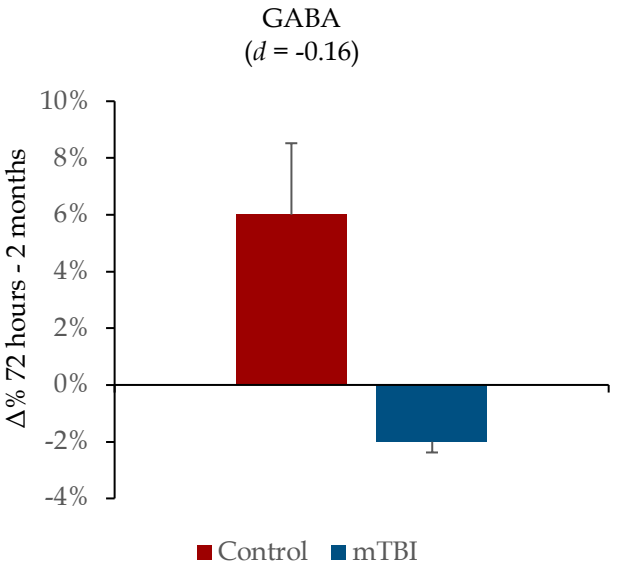


**Figure 4.** Percentage change in GABA concentration from 72 hours to 2 months.

**Table 3.** Average values of physiological measurements ± SD and percent change.

| Variable | Group | 72 hours | 2 months |
|---|---|---|---|
| MEP (mV) | Control | 0.55 ± 0.43 | 0.28 ± 0.2 |
| | mTBI | 0.79 ± 0.4 | 1.04 ± 0.79 |
| | *d* | -0.44 | -1.37 |
| CSP (ms) | Control | 80.63 ± 28.16 | 66.86 ± 16.25 |
| | mTBI | 112.45 ± 31.4 | 144.01 ± 73.85 |
| | *d* | -0.96 | -1.53 |
| Glutamate concentration (au) | Control | 0.94 ± 0.42 | 0.98 ± 0.13 |
| | mTBI | 0.98 ± 0.06 | 1.02 ± 0.1 |
| | *d* | 0.65 | 0.30 |
| GABA concentration (au) | Control | 0.013 ± 0.001 | 0.013 ± 0.002 |
| | mTBI | 0.014 ± 0.003 | 0.013 ± 0.007 |
| | *d* | 0.12 | -0.16 |

*Note.* MEP = motor evoked potential; CSP = cortical silent period; mV = millivolts; ms = milliseconds; au = arbitrary units; mTBI = mild traumatic brain injury; *d* = effect size, where a positive effect reflects a higher value in the control group compared with the mTBI group.

**Table 4.** Relationships of physical activity and sleep with the percent change in physiological outcomes.

| Variable | Total PA | Light PA | Moderate PA | Total Sleep | Sleep Efficiency |
|---|---|---|---|---|---|
| Δ MEP | $R^2$ =0.02 | $R^2$ =0.003 | $R^2$ =0.18 | $R^2$ =0.02 | $R^2$ =0.01 |
| | $p$ = 0.63 | $p$ = 0.88 | $p$ = 0.22 | $p$ = 0.77 | $p$ = 0.79 |
| Δ CSP | $R^2$ =0.20 | $R^2$ =0.10 | $R^2$ =0.30 | $R^2$ =0.25 | $R^2$ =0.05 |
| | $p$ = 0.16 | $p$ = 0.32 | $p$ = 0.08 | $p$ = 0.22 | $p$ = 0.63 |
| Δ Glutamate | $R^2$ = 0.08 | $R^2$ = 0.10 | $R^2$ = 0.02 | $R^2$ =0.10 | $R^2$ = 0.0004 |
| | $p$ = 0.47 | $p$ = 0.73 | $p$ = 0.20 | $p$ = 0.95 | $p$ = 0.29 |
| Δ GABA | $R^2$ = 0.01 | $R^2$ =0.02 | $R^2$ =0.15 | $R^2$ =0.003 | $R^2$ =0.10 |
| | $p$ = 0.25 | $p$ = 0.36 | $p$ = 0.25 | $p$ = 0.5 | $p$ = 0.45 |

*Note.* MEP = motor evoked potential; CSP = cortical silent period; PA = physical activity

## DISCUSSION

In this pilot study, we sought to determine if there is an impact of physical activity and sleep within the first week following mTBI on neurophysiological measurements at two months post-injury. Relative to the control group, individuals with mTBI had higher motor cortex excitability, higher intracortical inhibition and no difference in glutamate or GABA concentrations. The mTBI group also had lower levels of physical activity and greater amount of sleep. In this pilot study, however, the change in the physiological measurements over the two months of testing was not significantly related to physical activity levels or sleep.

Following mTBI, there is a neurometabolic cascade, which includes a discharge of neuronal potassium, release of amino acids, and reduced cerebral blood flow (12). These ionic and metabolic changes have been shown to affect the excitability and inhibition of cortical neurons (31). Although there are no clinical normative values for MEP or CSP, the range of values reported here for both MEP (10, 26, 27, 31) and CSP (3, 10, 31, 39) are within the range previously reported by our lab and others. Altered levels of excitability and inhibition, assessed via TMS have been associated with clinical characteristics, such as slowed movement speed (3). Establishment of normative values for these measures may therefore be clinically relevant, in predicting those at risk for functional limitations following mTBI and to track the progress of recovery or evaluate the effectiveness of therapy. In contrast to previous studies where a population of individuals with concussion showed no significant difference in the MEP from 72 hours to 30 years post-injury (18, 31, 33), the current study showed that mTBI participants had a higher MEP amplitude at rest. This finding suggests that there is greater excitability 2 months following a mTBI. According to the physiological concussion cascade model, shortly following brain trauma, the brain would be in a hyper-excitable state, reflected by an increase in the glutamate levels. In a rodent model, the hyper-excitable state, reflected by increased glutamate levels, lasts 10 minutes after induced trauma (10). For humans, it is challenging to determine the length of the excitable state, but research suggests that there is a peak in this excitable state approximately five hours post-injury (31).

Previous research has shown that there is an increased duration of the cortical silent period after an mTBI, reflecting greater inhibition. This increased duration has been shown up to two months or several years following brain injury when compared to a controlled population (3, 6, 18, 31). The results of this experiment support these findings. The average cortical silent period of the mTBI group was longer than the controlled group at the first visit and increased at the two months follow up. The increasing level of inhibition is in agreement with the neurophysiologic cascade that has been demonstrated in rodent models (3, 12). Injury will induce an excitable state, followed by a state of neuronal depression, resulting in inhibition. The relationship between moderate physical activity levels after injury and the change in CSP duration approached statistical significance ($p = 0.08$). This trend is in line with the study hypothesis that a gradual return to activity has an effect on physiological outcomes. Different levels of physical activity may affect the recovery rate, thus further investigation into the impacts of moderate activity on inhibitory processes is suggested in order to provide insights to better understand recovery from mTBI.

In animal models of mTBI, it has been demonstrated that in the early post-injury stages there is an increase in the release of the excitatory neurotransmitter glutamate. In the later post-injury stages, there is a greater level of the inhibitory neurotransmitter GABA. In the current investigation, however, we did not find any differences in the concentration of glutamate or GABA in the motor cortex region of the individuals with mTBI compared with healthy controls. Direct measurements of neurotransmitters in humans with mTBI are limited and, while we can compare the direction of differences across studies, direct comparisons of absolute values are limited by the arbitrary units, and the dependence on the magnet used. The values we observed here, however, are in line with our previous findings in a larger group of healthy young adults

(42). In contrast to the lack of group differences observed in the current study, Henry et al. (2010) found lower levels of glutamate in individuals with mTBI at one-week post-injury. However, in agreement with our results, Tremblay et al. (2014) showed no differences in GABA concentrations between individuals with mTBI and controls at 3 years post-injury. The divergent results may indicate a time-sensitivity of changes in glutamate post-injury.

The physiological outcomes from mTBIs can be variable but studies have shown that the negative effects on motor responses can lead to a period where the brain is at risk of further injury (12, 15). Studies have demonstrated that the symptoms of mTBI typically resolve in a relatively short period (7-14 days) in the majority of patients (29, 31); however, functional deficits and neurophysiological changes can persist beyond the resolution of symptoms (11). Currently, the best-accepted treatment plan is for patients to rest in the first hours-days following injury. In line with the prescription of rest, the mTBI patients in this study had lower levels of physical activity, particularly moderate intensity activity, and greater total sleep time than the control participants.

The clinical suggestion for rest stems from the idea that nonessential physical activity diverts essential oxygen and glucose away from injured brain cells, including neurons, and delays recovery from mTBI. However, recent studies suggest that low, controlled levels of physical activity in the early post-injury time may be beneficial to recovery (21-24). Further, it has been suggested that prolonged rest past the first few days from head injury may actually slow the rate of recovery (24, 30). These previous studies of the impacts of physical activity and sleep have focused largely on recovery of physical symptoms. A novel aspect of the current study was the measurement of unsupervised activity and sleep, and objective neurophysiological outcomes. In this study, we did not find a relationship between physical activities or sleep on changes in neurophysiological outcomes at 2 months post-injury.

As this is a pilot study, the sample size was small, which is a limitation. Additional studies with larger sample sizes are warranted and would permit examination of physical activity and sleep patterns across days. In this study, it was also unknown if the clinician suggested extended rest or a gradual increase in daily activity in the injured group. The observed difference in the activity levels and sleep between the two groups reflected such a suggestion, however, the clinicians' orders should be specifically tracked in future studies. Further, activity and sleep were tracked with Actigraph accelerometers. Although these devices have been validated for measures of activity (11, 24) and sleep (36, 40), all measures are based on accelerations of the device, which may limit their accuracy. It is therefore recommended that future work, building on this pilot study, use activity and sleep logs to help verify information obtained from the accelerometers.

We hypothesized that individuals with higher levels of physical activity and sleep in the first days following an mTBI would demonstrate greater improvements in neurophysiological outcomes at two months post-injury. This hypothesis was based on the traditional prescription of rest and recent data showing better symptom recovery in patients who performed a low-level, supervised exercise protocol after injury (21-24). However, in this small pilot project, we did not

find an association between habitual, unsupervised activity levels or sleep and recovery of neurophysiological assessments. Further work is therefore necessary to fully understand the benefits and risks of physical activity and sleep on recovery from mTBI. Research of how physical activity, and sleep influence physiological outcomes after mTBI will significantly benefit recovery management.

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