The bidirectional relationship between sleep and physical activity following traumatic brain injury

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Abstract
Sleep and physical activity are both modifiable behavioural factors that are associated with better health and are potentially related. Following traumatic brain injury, damage to the brain caused by an external force, sleep disturbances are common. Exploring bidirectional relationships between sleep and physical activity might provide insight into whether increasing physical activity could decrease these sleep disturbances. The current study, therefore, examined inter- and intra-individual temporal associations between sleep and daytime physical activity in 64 people with traumatic brain injury reporting sleep problems or fatigue (47 males; mean age, 40 years). Sleep and physical activity were measured using actigraphy with corroborating sleep diaries over 14 consecutive days. Multilevel models were used to examine inter- and intra-individual associations between physical activity and sleep. Inter-individual variations showed that earlier bedtimes, earlier wake-up times and lower sleep efficiency were associated with more physical activity. Intra-individual temporal variations showed no significant association of daytime physical activity with sleep duration or continuity. However, shorter sleep time and less wake after sleep onset than usual were associated with more time spent in light-intensity activity the next day. Therefore, sleep may have more of an influence on physical activity than physical activity has on sleep in people with traumatic brain injury. In conclusion, the results do not confirm a potential beneficial effect of physical activity on sleep but suggest that improving sleep quality might be relevant to support of a physically active lifestyle in people with traumatic brain injury. Further research is necessary to confirm these results.

KEYWORDS
actigraphy, exercise, multilevel modelling, physical activity, sleep, traumatic brain injury

1 | INTRODUCTION

Sleep and physical activity (PA) are both modifiable behavioural factors that are associated with better health (Buysse, 2014; Warburton & Bredin, 2016). Epidemiological studies in the general population found that people who are less physically active tend to have poorer sleep and more sleep-related difficulties (Driver & Taylor, 2000; Loprinzi & Cardinal, 2011). Furthermore, in community sample
groups, treatment increasing PA was found to have beneficial effects on sleep (Kredlow et al., 2015). This relationship between sleep and PA is thought to be bidirectional, whereby poor sleep might be associated with less PA, and lower PA levels may be associated with poor sleep (Chennoufi et al., 2015; Kline, 2014).

People with traumatic brain injury (TBI) often report sleep disturbances, with a meta-analysis indicating that 53% of people with TBI have some form of objective sleep disturbance (Mathias & Alvaro, 2012; Ponsford & Sinclair, 2014). Sleep disturbances are strongly associated with high rates of fatigue and depression in individuals with TBI and may contribute to reduced activity in work, social and leisure activities (Beaulieu-Bonneau & Morin, 2012; Ponsford & Sinclair, 2014). Studies examining PA in people with TBI are limited and PA has not been well described in this population (Hamilton et al., 2015; Pawlowski et al., 2013). The few studies available have found that PA levels following TBI are lower than recommended by the World Health Organization (i.e., <150 min of moderate intensity exercise or 75 min of vigorous intensity exercise throughout the week) (Driver et al., 2016; Hamilton et al., 2015). Compared to the behaviour of the general population, however, results are inconsistent; some studies report similar PA (Morris et al., 2019), whereas others report reduced PA in people with TBI (Pawlowski et al., 2013).

The presence of sleep disturbances following TBI may lead to a tendency to rest more during daytime, and this may in turn lead to the maintenance of sleep disturbances (Ouellet et al., 2012). Because studies in the general population showed a positive association between PA and sleep (Driver & Taylor, 2000), increasing PA might be beneficial for improving sleep in people with TBI. However, to examine this we first need information about the bidirectional relationships between physical activity and sleep in people with TBI. Previous research in other populations often averaged sleep and PA variables over multiple days, which ignores day-to-day variation and restricts capacity to examine whether one behaviour may affect the other from one day to the next (McClinchey et al., 2014). The few studies that did examine these day-to-day variations in populations other than TBI found mixed results, suggesting that this relationship might depend on the population examined (Bernard et al., 2016; Best et al., 2019; Lambiase et al., 2013).

Therefore, this study examined the daily bidirectional relationships between daytime PA and sleep in people with TBI. Through a secondary analysis of historical baseline datasets examining treatments for sleep or fatigue, inter- and intra-individual variations between sleep and PA were examined in people with TBI with sleep problems or fatigue by corroborating a sleep diary with wrist actigraphy. Based on the premise that PA has a positive effect on sleep and this effect might be bidirectional (Kline, 2014), it was hypothesized that for inter-individual variations, higher average levels of PA, indicated by greater activity counts and more time spent in light activity, would be associated with better sleep, indicated by longer total sleep time (TST), higher sleep efficiency (SE), shorter sleep onset latency (SOL) and less wake after sleep onset (WASO). For intra-individual temporal variations, it was hypothesized that higher levels of PA during the day would be followed by better sleep at night, and that better sleep would be followed by more PA the next day. Because both sleep and physical activity are associated with levels of depression and have been shown to change with age, these variables were controlled for (Ponsford & Sinclair, 2014; Ramsey et al., 2018).

2 | METHODS

2.1 | Participants

This study aggregated a subset of 64 participants with TBI using baseline data from three studies with similar methodologies examining sleep and treatment for sleep problems and fatigue in patients with acquired brain injury (Grima et al., 2018; Sinclair et al., 2014). Participants with TBI were recruited through a rehabilitation programme or via referral by healthcare professionals at Epworth Healthcare, Melbourne, Australia. The inclusion and exclusion criteria were similar across the studies. Participants with a medically documented mild-severe TBI defined as a history of a blunt head trauma with loss of consciousness and post-traumatic amnesia, and with clinically significant self-reported sleep problems, daytime sleepiness or fatigue were included (Pittsburgh Sleep Quality Index >5; Epworth Sleepiness Scale [ESS] ≥10 or Fatigue Severity Scale [FSS] ≥4). Participants were excluded if they had: a pre-injury history of sleep problems, fatigue or a neurological condition other than TBI; recent shift work or travel across more than one time zone; a high risk of sleep apnea; or current use of sleep medications. The protocols for two of the studies can be found in the original papers (Grima et al., 2018; Sinclair et al., 2014).

2.2 | Procedure

All study protocols received hospital and university ethics approval and all participants provided informed consent. Demographic characteristics were collected at the start of the studies. Both PA and sleep were measured by corroborating a sleep diary with wrist actigraphy. Participants were asked to wear the actiwatch continuously for 2 weeks except when bathing or showering. Concurrent with wearing the actiwatch, participants completed a sleep diary. When receiving or after wearing the actiwatch, participants answered questionnaires about their subjective sleep, fatigue and mood.

2.3 | Sleep measurement

Actiwatch 2 and Actiwatch Spectrum (Mini-Mitter/Respironics Inc.) devices were used to measure both sleep and PA and were worn around the non-dominant wrist. This non-invasive device has been well validated to measure sleep in people with TBI (Kamper et al., 2016). The actiwatch was set to 60-s epochs and a medium sensitivity was used to define the sleep parameters extracted with
Actiware software version 6.0.9 (Philips, Respironics). Bedtime and wake-up time from the sleep diaries were used to define the rest interval for the sleep analyses and adjustments were made consistent with other studies (Grima et al., 2018; Sinclair et al., 2014). In addition, the sleep diaries were used to define the nap periods in the actiwatch data. Participants were asked to report their bedtime, wake-up time and naps during the previous day each morning. Sleep outcome variables were bedtime, wake-up time, TST, SE, SOL and WASO.

2.4 Physical activity measurement

Although the activewatches used in this study were primarily designed to measure sleep, multiple studies support the use of these devices to measure overall physical activity (Lambiase et al., 2014; Lee & Tse, 2019). To determine overall level of activity per participant, the raw activity counts per minute of the actiwatch from the wake periods (i.e., time between rising and retiring to bed) were averaged per day and over the total sampling period. To examine the effect of activity before bedtime, average activity counts in the 5 h before bedtime over the total sample period were calculated. Activity for the first and last day of the registration period were omitted because these did not include a complete 24-h window. Intensity of activity during the wake period was calculated using cut-off points, sedentary (<100 AC/min), inactive (100–500 AC/min), light activity (500–2,020 AC/min), moderate activity (2,020–5,999 AC/min) and vigorous activity (>5,999 AC/min), in line with previous research in people with TBI (Driver et al., 2016).

The primary physical activity outcomes were overall level of activity and time spent in light activity. Time spent in light activity was included in the analyses because time spent in moderate to vigorous activity was very limited for this group. Data were included if there were at least 4 days with more than 10 h of daytime activity data available (Migueles et al., 2017).

2.5 Questionnaires

Subjective sleep quality, daytime sleepiness and fatigue were assessed using the PSQI (Buysse et al., 1989), ESS (Johns, 1991) and FSS (Krupp et al., 1989) respectively. Anxiety and depression were assessed with the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snith, 1983). For all questionnaires, higher scores represent more symptoms/problems. Details of the TBI were collected with participant consent from the hospital database.

2.6 Data Analyses

To assess relationships between sleep and PA (averaged over the whole sample period), and personal characteristics such as age, time since injury, post-traumatic amnesia (PTA) and scores on the HADS, FSS, PSQI and ESS, Pearson correlation analyses were used. Due to non-normality, SOL was log transformed. These analyses confirmed that depression and age are the variables most associated with sleep and physical activity levels following TBI and they were therefore added as covariates in the multilevel models. To examine the relationship between physical activity in the 5 h before bedtime and SOL, multiple linear regression analysis was used with age and depression as covariates.

To assess inter- and intra-individual associations between the sleep variables and daytime PA, multilevel linear models (MLM) with the restricted maximum likelihood estimation method were used. An autoregressive error variance structure was used to take into account that consecutive sleep and physical activity observations might be correlated more highly than non-consecutive observations. Two sets of MLM were performed. In the first set of models, daytime PA variables (daily activity counts and time spent in light activity) were the independent variables and subsequent sleep variables (bedtime, wake-up time, TST, SE, WASO and SOL) were dependent variables, with depression, age, weekday (weekend versus week) and TST of the naps during the day added as covariates (naps were not added in the model including wake-up time). In the second set of MLM, sleep variables were the independent variables and next-day PA the dependent variables, with depression, age, weekday (weekend versus week) added as covariates. As independent variables, PA and sleep were divided into inter-individual and intra-individual variables in line with previous research (Bernard et al., 2016; Best et al., 2019). Inter-individual (between-person) variables were calculated as the average level of PA over the whole sample period for each person. Intra-individual (within-person) variables were centred at the person mean representing daily variations of physical activity, where positive values indicated higher scores than the person’s own average. Twelve different models were created. Analyses were performed in R version 3.5.1 with the nlme package (Pinheiro et al., 2018).

3 RESULTS

3.1 Sample characteristics

The main participant characteristics and average of the sleep and physical activity variables are presented in Table 1. The mean age was 40 years (standard deviation [SD], 12.9), with a range of 19 to 68 years. The causes of TBI were traffic accidents (81.3%), falls (15.6%) and assault (1.6%, N = 63). Based on PTA duration, TBI was severe (PTA > 7 days) in most cases (75%), moderate (PTA 1–7 days) in 13% and mild (PTA < 1 day) in 12% of cases (N = 60). Over 90% of participants reported clinically significant fatigue, 67% had at least mild symptoms of depression and 62% had at least mild symptoms of anxiety (Table 1). Almost 40% had at least mild excessive daytime sleepiness and over 80% reported poor sleep quality (Table 1). Participants wore the actiwatch on average for 12 days (SD, 1.3; median, 13; 95% confidence interval, 12.0–12.6 days) and 13 nights (SD, 1.7; median, 14; 95% confidence interval, 12.4–13.2 nights). There was no correlation between the number of days or nights the participant wore the actiwatch and the sleep or activity variables. On
average, participants spent 180 min in light activity per day, had a sleep duration of 7 h per night and an average sleep efficiency of 79%.

3.2 | Sleep and activity in correlation with covariates

There were no significant correlations between the sleep variables and time since injury, PTA duration, HADS anxiety score, fatigue (FSS) or daytime sleepiness (ESS). Significant low to moderate correlations were found between age and sleep, as well as depressive symptoms (HADS) and sleep. Older age was associated with earlier bedtimes ($r = -0.37, p = .002$), earlier wake-up times ($r = -0.30, p = .015$), shorter SOL ($r = -0.34, p = .007$) and better SE ($r = -0.25, p = .047$). Higher HADS depression scores were associated with later bedtimes ($r = -0.33, p = .009$) and later wake-up times ($r = -0.28, p = .029$). Worse subjective sleep quality (PSQI) was associated with later wake-up times ($r = -0.34, p = .007$). There were no significant correlations between the PA variables and age, time since injury, PTA duration, HADS anxiety score, fatigue (FSS), sleep quality (PSQI) or daytime sleepiness (ESS). Higher HADS depression scores were

### TABLE 1  Demographic characteristics, questionnaire results and average sleep and activity variables. If data were missing, the number of participants that data were available for is reported

| Variable                          | Mean | SD  | Min–max/N (%) |
|-----------------------------------|------|-----|---------------|
| **Age (years)**                   | 40.0 | 12.9| 19.1–68.7     |
| **Gender (male)**                 | 47 (73.4) |
| **Time since injury (months)**    | 54.9<sup>a</sup> | 56.4 | 3.2–250.7     |
| **PTA duration (days)**           | 29.9<sup>c</sup> | 29.7 | 0.0–150.0     |
| **Current employment (yes)**      | 18 (28.1) |
| **Education (years)**             | 14.7 | 3.0 | 8–19<sup>b</sup> |
| **Living independently or with family**<sup>b</sup> | 63 (100)<sup>b</sup> |
| **Current medication use**        | 39 (61.9)<sup>b</sup> |
| **Pain medication**               | 18 (28.6)<sup>b</sup> |
| **Antidepressants**               | 24 (38.1)<sup>b</sup> |
| **Anti-epileptic drugs**          | 7 (11.1)<sup>b</sup> |
| **PSQI global (sleep problems)**  | 9.6  | 4.1 | 2.0–21.0      |
| **Clinically significant (>5)**   | 52 (81.2) |
| **ESS (daytime sleepiness)**      | 8.2  | 4.4 | 0.0–18.0      |
| **Clinically significant (≥11)**  | 25 (39.1) |
| **HADS – depression**             | 8.7  | 4.5 | 0–22<sup>b</sup> |
| **Clinically significant (≥8)**   | 42 (66.7)<sup>b</sup> |
| **HADS – anxiety**                | 8.0  | 4.3 | 0–16<sup>b</sup> |
| **Clinically significant (≥8)**   | 39 (61.9)<sup>b</sup> |
| **FSS (fatigue)**                 | 5.4  | 1.3 | 0–18          |
| **Clinically significant (≥4)**   | 58 (90.6) |

| Sleep variables                   | Mean | SD  | Min–max/N (%) |
|-----------------------------------|------|-----|---------------|
| **Bedtime (clock time 24 h; h)**  | 23:41| 1.44| 21:04–03:31   |
| **Wake-up time (clock time 24 h; h)** | 7:45 | 1.24| 05:47–11:00  |
| **Time in bed (h)**               | 8.0  | 1.1 | 5.1–10.3      |
| **Total sleep time (h)**          | 7.0  | 1.0 | 4.5–9.6       |
| **Sleep onset latency (min)**     | 28.7 | 22.9| 3.3–129.8     |
| **Sleep efficiency (%)**          | 79.2 | 7.1 | 58.0–91.3     |
| **Wake after sleep onset (min)**  | 60.1 | 22.2| 16.3–135.5    |

**Abbreviations:** ESS, Epworth Sleepiness Scale; HADS, Hospital Anxiety and Depression Scale; PTA, post-traumatic amnesia; PSQI, Pittsburgh Sleep Quality Index; SD, standard deviation; TBI, traumatic brain injury.

<sup>a</sup>Missing data for two participants.
<sup>b</sup>Missing data for one participant.
<sup>c</sup>Missing data for four participants.
| Predictors          | Bedtime |                         |                         | TST       |                         |                         | SE        |                         |                         | WASO      |                         | SOL       |
|---------------------|---------|--------------------------|--------------------------|-----------|--------------------------|--------------------------|-----------|--------------------------|--------------------------|-----------|--------------------------|-----------|
|                     | Estimates | CI (95%) | Estimates | CI (95%) | Estimates | CI (95%) | Estimates | CI (95%) | Estimates | CI (95%) | Estimates | CI (95%) | Estimates | CI (95%) |
| Overall activity (counts per min) |         |            |           |           |           |           |           |           |           |           |           |           |           |           |
| Inter-individual    | -0.004*  | -0.01 to -0.00  | -0.07    | -0.23 to 0.08 | -0.02    | -0.03 to 0.00 | 0.05 | -0.00 to 0.11 | 0.00 | -0.00 to 0.00 |         |            |           |           |
| Intra-individual    | -0.00    | -0.00 to 0.00  | -0.00    | -0.00 to 0.00 | 0.01    | -0.07 to 0.08 | 0.02 | -0.01 to 0.05 | -0.00 | -0.00 to 0.00 |         |            |           |           |
| Light activity (min) |         |            |           |           |           |           |           |           |           |           |           |           |           |           |
| Inter-individual    | -0.005** | -0.01 to 0.00  | -0.15    | -0.33 to 0.02 | -0.02    | -0.03 to 0.00 | 0.04 | -0.02 to 0.11 | 0.00 | -0.00 to 0.00 |         |            |           |           |
| Intra-individual    | 0.002**  | 0.00 to 0.00 | 0.00    | -0.00 to 0.00 | -0.09    | -0.19 to 0.00 | 0.00 | -0.01 to 0.01 | -0.02 | -0.05 to 0.02 | -0.00 | -0.00 to 0.00 |         |            |           |

Note: All models were adjusted for age, depression scores, weekday (weekend versus weekday) and total sleep time of naps taken during the day. The bold values indicate the significant associations. 707 daily observations were clustered in 63 participants.

Abbreviations: CI (95%), 95% confidence interval; SE, sleep efficiency; SOL, sleep onset latency; TST, total sleep time; WASO, wake after sleep onset.

*p < 0.05.

**p < 0.01.
associated with lower overall activity ($r = -0.31, p = 0.01$) and less time spent in light activity ($r = -0.39, p = 0.02$). Based on these correlation analyses the HADS depression score and age were added as covariates in the MLM analyses to examine the association between daytime PA and sleep. There was no association between activity in the 5 h before bedtime and SOL ($\beta = 0.00, p = 0.46$).

### 3.3 Inter- and intra-individual associations between PA and sleep

The HADS depression score was missing for one participant; therefore, the inter- and intra-individual variation analyses included 63 participants.

#### 3.3.1 Daytime physical activity as a predictor of sleep

The MLM examining inter- and intra-individual associations between daytime PA and sleep the following night only showed significant relationships between PA and the timing of sleep (Table 2).

#### 3.3.2 Sleep as a predictor of daytime physical activity

The MLM examining inter- and intra-individual associations between the sleep variables and next day physical activity revealed a similar inter-individual relationship between sleep timing and PA (Table 3). Participants who went to bed earlier and woke up earlier were more physically active, indicated by greater activity counts and more time spent in light activity. Inter-individual variations indicated that participants with better SE had fewer overall activity counts. Intra-individual variations in wake-up time showed a similar pattern; on days the participants woke up earlier then their average they were

| Predictors          | Overall activity (counts per min) | Light activity (min) |
|---------------------|-----------------------------------|----------------------|
|                     | Estimates CI (95%)                | Estimates CI (95%)   |
| Bedtime             |                                   |                      |
| Inter-individual    | $-19.34^*$ $-38.34$ to $-0.33$    | $-12.94$ $-30.12$ to $4.24$ |
| Intra-individual    | $-1.79$ $-7.19$ to $3.61$         | $-2.76$ $-7.01$ to $1.49$ |
| Wake-up time        |                                   |                      |
| Inter-individual    | $-29.99^*$ $-50.60$ to $-9.38$    | $-28.85^*$ $-46.97$ to $-10.73$ |
| Intra-individual    | $-5.72^*$ $-10.85$ to $-0.58$     | $-12.55^{***}$ $-16.49$ to $-8.61$ |
| Total sleep time    |                                   |                      |
| Inter-individual    | $-0.23$ $-0.62$ to $0.17$         | $-0.31$ $-0.65$ to $0.04$ |
| Intra-individual    | $-0.04$ $-0.12$ to $0.04$         | $-0.11^{***}$ $-0.18$ to $-0.05$ |
| Sleep efficiency    |                                   |                      |
| Inter-individual    | $-4.15^*$ $-7.91$ to $-0.38$      | $-2.95$ $-6.34$ to $0.45$ |
| Intra-individual    | $0.16$ $-0.60$ to $0.91$          | $0.10$ $-0.50$ to $0.69$ |
| Wake after sleep onset |                               |                      |
| Inter-individual    | $0.97$ $-0.15$ to $2.09$          | $0.57$ $-0.44$ to $1.58$ |
| Intra-individual    | $-0.12$ $-0.34$ to $0.10$         | $-0.23^*$ $-0.40$ to $-0.05$ |
| Sleep onset latency |                                   |                      |
| Inter-individual    | $0.14$ $-1.06$ to $1.35$          | $0.02$ $-1.05$ to $1.09$ |
| Intra-individual    | $-0.05$ $-0.24$ to $0.15$         | $-0.10$ $-0.25$ to $0.06$ |

Note: All models were adjusted for age, depression scores and weekday (weekend versus weekday). The bold values indicate the significant associations.
706 daily observations were clustered in 63 participants.
Abbreviation: CI (95%), 95% confidence interval.

* $p < .05$.
** $p < .01$
*** $p < .001$.  

Table 3 Results from multilevel models of inter- and intra-individual associations between nighttime sleep and physical activity during the following day, while adjusting for covariates.
more physically active, indicated by greater activity counts and more time spent in light activity. Intra-individual variations in TST and WASO revealed that when participants had shorter TST than their average or had less WASO than their average, they spent more time in light activity the next day. There were no significant associations between nightly changes in bedtimes, SE or SOL, and next day PA.

4 | DISCUSSION

This study examined the inter- and intra-individual daily relationship between PA and sleep in people with TBI. At the inter-individual level, results showed that earlier bedtimes, earlier wake-up times and lower SE were associated with more physical activity. Intra-individual daily variations in PA were not associated with the following night’s sleep duration or continuity, implying that more PA during the day did not improve sleep the following night. However, intra-individual nightly variations in sleep revealed that shorter TST and less WASO predicted more time spent in light activity the next day. Therefore, sleep might have a stronger influence on next day PA than PA has on the following night’s sleep.

Sleep timing is associated with the timing of the endogenous circadian rhythm (i.e., later bed and wake times indicate later circadian timing). Our finding that earlier bedtimes and wake-up times are associated with higher levels of PA, in conjunction with previous research showing that later bed and wake times are associated with less PA, indicates that there is likely to be an underlying circadian mechanism driving this relationship after TBI (Shechter & St-Onge, 2014). Contrary to our expectations, higher levels of PA were not associated with better sleep in this sample of people with TBI; rather, more PA was associated with lower SE. This differs from previous research in breast cancer patients with insomnia, which indicated a positive association between SE and PA at the inter-individual level (Bernard et al., 2016), and from studies in other populations that did not find a relationship between SE and PA (Atoui et al., 2020). There were no significant associations between daytime PA and other sleep variables such as TST, SOL and WASO at the inter-individual level, which were expected. This could be due to the large heterogeneity in people with TBI or to the fact that other potential relevant characteristics of PA were not taken into account, such as type of PA or the cognitive load, which varies across types of PA (Horne, 2013). Furthermore, although relationships have been demonstrated in previous research (Kredlow et al., 2015), associations were often small-to-medium and results inconsistent (Atoui et al., 2020). Therefore, these findings are unsurprising.

Temporal intra-individual associations showed that on days when TBI participants spent more time in light activity than usual, they went to bed later that night. In contrast with expectations, more PA during the day did not improve the following night’s sleep. However, shorter TST and less WASO were associated with more light activity the next day. In line with previous research, this suggests that intra-individual variability in sleep quality predicted next day PA but not vice versa (McGlinchey et al., 2014; Tang & Sanborn, 2014).

Furthermore, this intra-individual association between shorter TST and more PA the next day has also been found in previous studies (Bernard et al., 2016; Lambiase et al., 2014). Nonetheless, a meta-analysis did not indicate consistent unidirectional associations between PA and sleep (Atoui et al., 2020). These results, together with the small associations that we found, suggest that the association between sleep and PA might differ across individuals with TBI. Very recent research has suggested this might also be the case in the general population (Atoui et al., 2020). In addition, it might be mediated by other factors such as light exposure, timing of the physical activity, season or physical fitness, which we did not control for (Atkinson & Davenne, 2007). Furthermore, the different study populations and diverse range of measurements and devices used, make comparisons between studies difficult. Therefore, standardized procedures for the assessment, analysis and reporting of this type of data are needed.

In both patient and non-patient samples, PA is associated with better health (Booth et al., 2012; Loprinzi & Cardinal, 2011). People with TBI who exercise regularly have better general health and are less depressed (Gordon et al., 1998). In addition, positive effects of PA treatment have been found on sleep, cognitive functioning, mood and quality of life in individuals with TBI (Hoffman et al., 2010; Vanderbeken & Kerckhofs, 2017). These studies indicate that PA has positive effects in multiple domains in people with TBI. In line with previous studies, the results of this study showed that more PA was related to fewer depressive symptoms; however, the direction and causality could not be examined with this study.

4.1 | Limitations

This study has strong ecological validity, as participants were examined in their own environment, and recall bias was limited because of the use of objective measures of both sleep and PA. Nonetheless, there were some limitations. Although actigraphy is often used to measure sleep and activity in the field, and studies comparing the Actiwatch 2 with devices commonly used to measure activity, such as the ActiGraph WGT3X-BT and the ActiGraph GT1M, show strong correlations between the devices, limitations with actigraphy measures in general remain (Lambiase et al., 2014; Lee & Tse, 2019). Most notably, actigraphy is not able to distinguish between different types of activity and the level of exertion required. Additionally, there are activities for which the device cannot be worn (e.g., swimming) and, due to its location, the device may not register the activity (e.g., cycling). Although measures of sleep timing can be obtained from actigraphy, it cannot provide insight into how PA may impact sleep architecture. A further limitation of this study is the use of historical datasets. There were no specific questions regarding PA and only people with TBI with clinically significant fatigue or sleep disturbances were included. This may limit the generalizability of these findings to the wider TBI population. However, sleep disturbances are common following TBI and especially for this group it is important to examine whether sleep disturbances might
be associated with decreased physical activity. Finally, the results are from observational data examining whether PA during the day predicts sleep that night and vice versa and are therefore limited in providing insight into the underlying mechanisms. Future studies geared towards exploring the causal link between PA and sleep, such as randomized-controlled intervention studies, may shed light on the impact of PA on sleep.

5 | CONCLUSION

The results suggest an association between PA and timing of sleep, but more physically active TBI participants did not sleep better. Therefore, these results do not suggest a beneficial effect of physical activity on sleep following TBI. However, the lack of this association might be related to individual differences. The TBI population is known to be a particularly heterogeneous group in terms of neuropathology, and in cognitive and behavioural symptoms, and therefore PA treatment for sleep disturbances following TBI might need to be individually tailored. Furthermore, day-to-day variations showed that sleep may have more of an influence on physical activity than physical activity has on sleep in people with TBI. Therefore, improving sleep quality may be an important first step to encourage a more physically active lifestyle and potentially improve health and quality of life after TBI. However, further studies are necessary to confirm these results.

CONFLICTS OF INTEREST

One out of the three historical datasets included in this paper received funding from the National Health and Medical Research Council, approval ID: 1028733. EMW is supported by an Australian Government Research Training Program (RTP) Scholarship. For the remaining authors, no conflicts of interest were declared.

AUTHOR CONTRIBUTIONS

NG, KS and LY acquired the subjects and collected data. NG, KS, LY, JM and JB preprocessed data. JB, JP, NG and JM determined the study hypotheses. JB conducted statistical analyses and prepared the initial draft. All authors provided a critical review of the manuscript.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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REFERENCES

Atkinson, G., & Davenne, D. (2007). Relationships between sleep, physical activity and human health. Physiology & Behavior, 90(2), 229–235. https://doi.org/10.1016/j.physbeh.2006.09.015
Atouf, S., Chevance, G., Romain, A. J., Kingsbury, C., Lachance, J., & Bernard, P. (2021). Daily associations between sleep and physical activity: A systematic review and meta-analysis. Sleep Medicine Reviews, 57(101426), 1. http://dx.doi.org/10.1016/j.smrv.2021.101426
Beaulieu-Bonneau, S., & Morin, C. M. (2012). Sleepiness and fatigue following traumatic brain injury. Sleep Medicine, 13(6), 598–605. https://doi.org/10.1016/j.sleep.2012.02.010
Bernard, P., Ivers, H., Savard, M. H., & Savard, J. (2016). Temporal relationships between sleep and physical activity among breast cancer patients with insomnia. Health Psychology, 35(12), 1307–1315. https://doi.org/10.1037/heao0000408
Best, J. R., Falcik, R. S., Landry, G. J., & Liu-Ambrose, T. (2019). Analysis of dynamic, bidirectional associations in older adult physical activity and sleep quality. Journal of Sleep Research, 28(4), e12769. https://doi.org/10.1111/jsr.12769
Booth, F. W., Roberts, C. K., & Laye, M. J. (2012). Lack of exercise is a major cause of chronic diseases. Comprehensive Physiology, 2(2), 1143–1211. https://doi.org/10.1002/cphy.c110025
Buysse, D. J. (2014). Sleep health: Can we define it? Does it matter? Sleep, 37(1), 9–17. https://doi.org/10.5665/sleep.3298
Buysse, D. J., Reynolds, C. F. 3rd, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. Psychiatry Research, 28(2), 193–213. https://doi.org/10.1016/0165-1781(89)90047-4
Chennauoi, M., Armal, P. J., Sauvet, F., & Léger, D. (2015). Sleep and exercise: A reciprocal issue? Sleep Medicine Reviews, 20, 59–72. https://doi.org/10.1016/j.smrv.2014.06.008
Driver, H. S., & Taylor, S. R. (2000). Exercise and sleep. Sleep Medicine Reviews, 4(4), 387–402. https://doi.org/10.1053/smrv.2000.0110
Driver, S., Rachal, L., Swank, C., & Dubiel, R. (2016). Objective assessment of activity in inpatients with traumatic brain injury: initial findings. Brain Impairment, 17(1), 55–63. https://doi.org/10.1017/Brimp.2015.20
Gordon, W. A., Sliwinski, M., Echo, J., McLoughlin, M., Sheerer, M. S., & Meili, T. E. (1998). The benefits of exercise in individuals with traumatic brain injury: A retrospective study. Journal of Head Trauma Rehabilitation, 13(4), 58–67. https://doi.org/10.1097/00001199-199808000-00006
Grima, N. A., Rajaratnam, S. M. W., Mansfield, D., Sletten, T. L., Spitz, G., & Ponsford, J. L. (2018). Efficacy of melatonin for sleep disturbance following traumatic brain injury: A randomised controlled trial. BMC Medicine, 16(1), 8. https://doi.org/10.1186/s12196-017-0995-1
Hamilton, M., Williams, G., Bryant, A., Clark, R., & Spelman, T. (2015). Which factors influence the activity levels of individuals with traumatic brain injury when they are first discharged home from hospital? Brain Injury, 29(13–14), 1572–1580. https://doi.org/10.3109/02692066.2015.1075145
Hoffman, J. M., Bell, K. R., Powell, J. M., Behr, J., Dunn, E. C., Dikmen, S., & Bombardier, C. H. (2010). A randomized controlled trial of exercise to improve mood after traumatic brain injury. PM&R, 2(10), 911–919. https://doi.org/10.1016/j.pmrj.2010.06.008
Horne, J. (2013). Exercise benefits for the aging brain depend on the accompanying cognitive load: Insights from sleep electroencephalogram. Sleep Medicine, 14(11), 1208–1213. https://doi.org/10.1016/j.sleep.2013.05.019
Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth sleepiness scale. Sleep, 14(6), 540–545. https://doi.org/10.1093/sleep/14.6.540
Kamper, J. E., Garofano, J., Schwartz, D. J., Silva, M. A., Zeitzer, J., Modares, M. O., Barnett, S. D., & Nakase-Richardson, R. (2016). Concordance of actigraphy with polysomnography in traumatic brain injury neuromodulation admissions. Journal of Head Trauma Rehabilitation, 31(2), 117–125. https://doi.org/10.1097/HTR.0000000000000215
Kline, C. E. (2014). The bidirectional relationship between exercise and sleep: Implications for exercise adherence and sleep improvement.
American Journal of Lifestyle Medicine, 8(6), 375–379. https://doi.org/10.1159/1559827614544437
Kredlow, M. A., Capozzoli, M. C., Hearon, B. A., Calkins, A. W., & Otto, M. W. (2015). The effects of physical activity on sleep: A meta-analytic review. Journal of Behavioral Medicine, 38(3), 427–449. https://doi.org/10.1007/s10865-015-9617-6
Krupp, L. B., LaRocca, N. G., Muir-Nash, J., & Steinberg, A. D. (1989). The fatigue severity scale. Application to patients with multiple sclerosis and systemic lupus erythematosus. Archives of Neurology, 46(10), 1121–1123. https://doi.org/10.1001/archneur.1989.00520460115022
Lambiase, M. J., Gabriel, K. P., Chang, Y. F., Kuller, L. H., & Matthews, K. A. (2018). Temporal relationships between physical activity and sleep in older women. Medicine and Science in Sports and Exercise, 45(12), 2362–2368. https://doi.org/10.1249/MSS.000000000000361
Lee, P., & Tse, C. Y. (2019). Calibration of wrist-worn ActiWatch 2 and ActiGraph wGT3X for assessment of physical activity in young adults. Gait & Posture, 68, 141–149. https://doi.org/10.1016/j.gaitpost.2018.11.023
Loprinzi, P. D., & Cardinal, B. J. (2011). Association between objectively-measured physical activity and sleep, NHANES 2005–2006. Mental Health and Physical Activity, 4(2), 65–69. https://doi.org/10.1016/j.mhpa.2011.08.001
Mathias, J. L., & Alvaro, P. K. (2012). Prevalence of sleep disturbances, disorders, and problems following traumatic brain injury: A meta-analysis. Sleep Medicine, 13(7), 898–905. https://doi.org/10.1016/j.sleep.2012.04.006
McGlinchey, E. L., Gershon, A., Eidelman, P., Kaplan, K. A., & Harvey, A. G. (2014). Physical activity and sleep: Day-to-day associations among individuals with and without bipolar disorder. Mental Health Phys Act, 7(3), 183–190. https://doi.org/10.1016/j.mhpa.2014.05.003
Migueles, J. H., Cadenas-Sanchez, C., Ekelund, U., Delisle Nyström, C., Mora-Gonzalez, J., Löf, M., Labayen, I., Ruiz, J. R., & Ortega, F. B. (2017). Accelerometer data collection and processing criteria to assess physical activity and other outcomes: A systematic review and practical considerations. Sports Medicine, 47(9), 1821–1845. https://doi.org/10.1007/s40279-017-0716-0
Morris, T. P., Tormos Muñoz, J.-M., Cattaneo, G., Solana-Sánchez, J., Bartrés-Faz, D., & Pascual-Leone, A. (2019). Traumatic brain injury modifies the relationship between physical activity and global and cognitive health: Results from the Barcelona brain health initiative. Frontiers in Behavioral Neuroscience, 13(135), 1–7. https://doi.org/10.3389/fnbeh.2019.00135
Ouellet, M.-C., Beaulieu-Bonneau, S., & Morin, C. M. (2012). Sleep-wake disturbances and fatigue in individuals with traumatic brain injury. In C. E. Espie, & C. M. Morin (Eds.), The Oxford handbook of sleep and sleep disorders (pp. 1–73). Oxford: Oxford University press. https://doi.org/10.1093/oxfordhb/9780195376203.001.0001
Pawloski, J., Dixon-Ibarra, A., & Driver, S. (2013). Review of the status of physical activity research for individuals with traumatic brain injury. Archives of Physical Medicine and Rehabilitation, 94(6), 1184–1189. https://doi.org/10.1016/j.apmr.2013.01.005
Pinheiro, J., Bates, D., DebRoy, S., Sarkar, D., & R Core Team. (2018). nlme: Linear and nonlinear mixed effects models. R package version, 3(1), 111. https://CRAN.R-project.org/package=nlme
Ponsford, J. L., & Sinclair, K. L. (2014). Sleep and fatigue following traumatic brain injury. Psychiatric Clinics of North America, 37(1), 77–89. https://doi.org/10.1016/j.psc.2013.10.001
Ramsey, J., Driver, S., Swank, C., Bennett, M., & Dubiel, R. (2018). Physical activity intensity of patient’s with traumatic brain injury during in-patient rehabilitation. Brain Injury, 32(12), 1518–1524. https://doi.org/10.1080/02699052.2018.1500715
Shechter, A., & St-Onge, M.-P. (2014). Delayed sleep timing is associated with low levels of free-living physical activity in normal sleeping adults. Sleep Medicine, 15(2), 1586–1589. https://doi.org/10.1016/j.sleep.2014.07.010
Sinclair, K. L., Ponsford, J., & Rajaratnam, S. M. (2014). Actigraphic assessment of sleep disturbances following traumatic brain injury. Behavioral Sleep Medicine, 12(1), 13–27. https://doi.org/10.1080/15402002.2012.726203
Tang, N. K. Y., & Sanborn, A. N. (2014). Better quality sleep promotes daytime physical activity in patients with chronic pain? A multilevel analysis of the within-person relationship. PLoS One, 9(3), e92158. https://doi.org/10.1371/journal.pone.0092158
Vanderbeken, I., & Kerckhofs, E. (2017). A systematic review of the effect of physical exercise on cognition in stroke and traumatic brain injury patients. NeuroRehabilitation, 40(1), 33–48. https://doi.org/10.3233/nre-161388
Warburton, D. E., & Bredin, S. S. (2016). Reflections on physical activity and health: What should we recommend? Canadian Journal of Cardiology, 32(4), 495–504. https://doi.org/10.1016/j.cjca.2016.01.024
Zigmond, A. S., & Snaith, R. P. (1983). The hospital anxiety and depression scale. Acta Psychiatrica Scandinavica, 67(6), 361–370. https://doi.org/10.1111/j.1600-0447.1983.tb09716.x

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