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ARTICLE

Excessive daytime sleepiness in adults with spinal cord injury and associations with pain catastrophizing and pain intensity

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Abstract

Study design Pre-post cohort mixed factorial design.

Objective Excessive daytime sleepiness (EDS) and chronic pain are major problems for people with spinal cord injury (SCI). However, the relationship between chronic pain and EDS requires clarification. The goal of the study was to determine associations between pain catastrophizing (PC) and pain intensity (PI) with EDS in adults with SCI.

Setting New South Wales, Australia.

Methods Participants included 45 adults with SCI and 44 able-bodied controls. The relationship between PI, PC, and EDS was explored by determining the influence of PC and PI on the performance of both groups in a behavioral test of EDS called the Oxford Sleep Resistance Test. PC and PI were assessed by self-report. The association between EDS, pain, and other relevant factors like fatigue and mood was established using multidimensional scaling in the SCI group data.

Results PC was found to have a significant association with EDS, with 33.3% falling asleep in the SCI group with low PC, compared with 70% in those with high PC. Only 10% of the controls fell asleep regardless of PC. PI did not significantly influence EDS in either group. Multidimensional scaling showed EDS was closely related to PC, PI, pain interference, fatigue, and mood.

Conclusions PC appears to be strongly associated with EDS in SCI. Findings suggest significant sleep benefits may occur in adults with SCI by treating cognitive biases like PC, as well as addressing associated factors like fatigue, pain interference, low mood, and so on.

Introduction

There exists a high rate of chronic pain associated with spinal cord injuries (SCI) that many struggle to deal with and which leads to diminished quality of life (QOL) [1–4]. Equally problematic, sleep disorder in neurological injuries like SCI can also lead to diminished QOL and social participation, especially in those with high lesions and motor/sensory complete injuries [5–9]. Excessive daytime sleepiness (EDS), closely associated with sleep disorder, autonomic imbalance, and fatigue are also prevalent conditions in SCI [5, 8–10]. For example, we have found EDS is positively associated with autonomic imbalance in adults with SCI [8]. Research has revealed reciprocal associations exist between sleep and chronic pain, finding that chronic pain interferes with sleep quality, while poor sleep increases chronic pain [11, 12]. It is not difficult to accept that chronic pain will disrupt sleep quality, however, it is perhaps less appreciated that poor sleep will increase pain [11, 12]. It has been argued that poor sleep and associated sleepiness will reduce top-down cortical resources (e.g., attention), resulting in diminished capacity to detect and modulate pain [13]. Further, it has been shown that pain catastrophizing (PC) diminishes top-down resources, resulting in increased pain and risk of psychological disorder [2, 14]. For example, Seminowicz and Davis [14] showed PC increased attention to pain, lowering the ability to distract/disengage from it, given increased PC was related to moderated prefrontal cortical vigilance resources. PC is a negative cognitive appraisal involving feeling helpless and pessimistic about

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one's pain, consistently focusing on the pain and its adverse consequences and magnifying its effects so that minor pain-related challenges become viewed as disastrous [2].

Noxious sensory stimuli (nociceptive bottom-up process such as pain intensity (PI)) demand immediate attention, ideally resulting in behavior that minimizes harm. Ignoring or not detecting the sensory noxious input will likely increase risk of injury and pain. Likewise, top-down processes such as distraction, helpful attention, optimistic appraisal-reappraisal will likely reduce pain, while factors like PC, depressive mood, and fear will increase pain [2, 3, 15]. However, research is needed to clarify how bottom-up factors like PI and top-down pain factors like PC relate to sleep disorder/EDS, as well as how demographic, injury, and psychosocial factors influence this relationship. PI remains an important bottom-up factor in SCI. Though spinothalamic tract damage in SCI diminishes bottom-up nociception information, the remaining nociceptive neurons in the spinal cord become hyperexcitable, leading to increased ascending PI [16].

This study was therefore designed to investigate the influence of PI (bottom-up) and PC (top-down) factors on EDS in adults with SCI with comparisons to able-bodied (AB) controls, as well as to determine associations between pain factors, EDS, sociodemographic and injury factors, fatigue, participation, psychosocial and mental health in adults with SCI. All factors were selected in the study given their relationship to how people adjust to SCI [6]. It was hypothesized that (i) participants with SCI would have increased propensity for EDS; (ii) participants with high levels of PC and PI would have the highest propensity for EDS; and (iii) multiple factors shown to be related to adjustment to SCI would be associated with pain factors and EDS, such as fatigue, sleep apnea, body mass index (BMI), participation, and mental health.

Methods

Study participants

Included 46 adults with SCI and 46 AB control participants. For SCI participants, recruitment occurred mostly through SCI rehabilitation unit, outpatient clinics, and by advertisements in SCI consumer organization newsletters. Inclusion criteria for SCI consisted of: (i) aged 18–80 years; (ii) English speaking; (iii) sustained a SCI and at least 6 months postinjury from inpatient rehabilitation; and (iv) no evidence of severe psychiatric disorder such as bipolar disorder or psychoses, as determined by a structured psychiatric interview. One SCI participant withdrew from the study leaving 45, and two AB controls failed to complete the study, leaving 44. Inclusion criteria for the controls included (i) and (ii) above, (iii) no history of neurological injury, and (iv) no evidence of a current severe psychiatric disorder, such as bipolar disorder or psychoses, as determined by a structured psychiatric interview. AB participants were included to make comparisons to how bottom-up (PI) and top-down (PC) pain factors influence EDS in SCI. The AB sample was stratified so that the two groups had similar numbers of males and females and similar ages.

Study design and procedure

A mixed factorial design was used. Two groups took part in a 40-min behavioral sleepiness test called the Oxford Sleep Resistance Test (OSLER; Stowood Scientific Instruments, Oxford, UK) during which electrophysiology recording occurred (electrocardiography and electrooculography (EOG)). Participants were tested first at baseline before the EDS, immediately after, and third, after a 5-min recovery period. However, only EOG data are reported in this paper, and only baseline data were used for analyses to address the third hypothesis. Participants were asked to refrain from drinking caffeinated/alcoholic beverages on the morning of the assessment. To control circadian influences [17], the experiment was conducted between 9 a.m. and 1 p.m. in a quiet semi-darkened room in the participant's home, health clinic, or research institution. The participants with SCI were seated in their wheelchair and controls were seated in a chair with armrests during the experiment.

Assessment

Completeness of the lesion was assessed by medical specialists according to the International Standards for Neurological Classification of SCI (http://ais.emsci.org/). BMI was assessed from self-reported height/weight and was classified according to World Health Organization guidelines: $<18.5\ kg/m^2$ (underweight), $18.5–24.9\ kg/m^2$ (normal), $25–29.9\ kg/m^2$ (overweight), $\geq 30\ kg/m^2$ (obese). Sections of the International SCI Pain Basic Data Set (v.2) were used to assess pain [18]. All participants rated average PI over the past week using a 0–10 numerical rating scale (0 = "no pain", 10 = "pain as bad as can be imagined"). Participants also rated pain interference associated with day to day activities, mood, and sleep (0 = no interference; 10 = extreme interference). The three items were summed for a total of 30.

The Pain Catastrophizing Scale (PCS), a cognitive bias towards thinking helplessly, pain rumination, and magnifying the problem, were used to asses PC [19]. The PCS is a 13-item 5-point Likert scale (0–4) with a range of 0–52 [19]. The PCS has acceptable reliability, validity, and internal consistency [2, 19]. Only total PCS scores are presented.

EDS was measured using the OSLER, a validated behavioral test of daytime sleepiness. It assesses the ability to remain awake (sleep latency) in a darkened room when required to tap a switch when a light at eye-level is turned on for 1 s every 3 s for up to 40 min [20]. A microsleep was recorded if a participant missed tapping the light or when their finger remained on the switch after the light switched off. After seven consecutive microsleeps (21 s), the participant was considered to have slept and the OSLER test terminated. For participants who had insufficient hand control to tap the switch with their finger, the switch was stabilized at chin level so they could lean forward and tap the switch with their chin. If they leant forward and slept, their chin remained on the switch and a microsleep recorded. This is a reliable means for completing the task in those with high lesions [8]. The total time participants remained awake is called sleep latency, a period of less than 40 min if they slept, or 40 min if they remained awake [20]. Its sensitivity/specificity in detecting daytime sleepiness is 85%/94%, respectively [20].

Chronic fatigue is defined as chronic tiredness involving feelings of exhaustion and negative emotions, such as anxiety and poor mood [10, 17]. It is the chronic nature of fatigue that distinguishes it from daytime sleepiness, or tiredness resulting from daily physical and mental exertion. Length of eye blink duration, calculated from EOG, is a sensitive physiological measure of fatigue with slow blink rates of around 500 ms or greater associated with fatigue [17, 21]. EOG was assessed at baseline and directly after the OSLER task for 5 min using the Biosemi^{TM} ActiveTwo System [8]. Eye blink duration was computed from the unfiltered EOG signal at 500 mV in 2-s intervals on a low-pass filter during the 5-min recordings. The blink rate of ≥500 ms for both groups was recorded. The Fatigue Severity Scale (FSS) was used as a self-report 9-item measure of fatigue [22]. Fatigue severity was calculated by adding up items and dividing by nine. Higher scores indicate greater fatigue and the FSS has acceptable reliability and validity [22]. The Berlin Questionnaire (BQ) was used to detect probable sleep apnea. BQ assesses snoring behavior, wake-time sleepiness and fatigue, and history of obesity or hypertension [23]. People scoring high of any two of three of these symptom groupings are at high risk of sleep apnea. The BQ has high internal consistency, sensitivity, and specificity [23].

Two measures of self-efficacy were assessed to determine relationships between self-efficacy beliefs about pain and beliefs about managing SCI. Self-efficacy beliefs about chronic pain were assessed using the 10-item pain self-efficacy questionnaire (PSEQ) [24]. Participants rated how confident they are about performing activities despite their pain. Each item is rated on a 7-point scale, where 0 = "not at all confident" and 6 = "completely confident". Higher scores indicate stronger pain-related self-efficacy beliefs. The PSEQ has acceptable reliability and validity [24]. The second measure of self-efficacy was a SCI-specific measure called the Moorong Self-Efficacy Scale, a 16-item scale, with items such as "I can avoid having bowel accidents" and "I can accomplish most things I set out to do". It is internally consistent, stable, and has acceptable construct validity [25]. Resilience was assessed using the Connor-Davidson Resilience Scale, which has demonstrated reliability and validity, with higher scores indicating greater resilience [26]. Anxiety and depressive mood were assessed by the depressive mood and anxiety domains of the Symptom Check List-90-R [27]. It is a 90-item self-report measure of negative symptoms having demonstrated validity and reliability [27].

Perception of actual social support was measured using the short-form version of the Social Support Questionnaire [28]. Higher scores indicate increased optimism about their social support and it has demonstrated reliability and validity [28]. Social participation and autonomy was assessed by the Impact on Participation and Autonomy Questionnaire, shown to be a reliable and valid instrument for assessing autonomy and participation in chronic disorders, with higher scores indicating poor autonomy and social participation [29]. Cognitive performance was assessed by the Neuropsychiatry Unit Cognitive Assessment Tool (NUCOG), a valid and reliable instrument measuring cognitive impairment in people with psychiatric and neurological disorders [30]. The NUCOG consists of 21 items assessing cognitive function across five cognitive domains: attention, memory, executive functioning, visuo-constructional, and language. Only the total score out of 100 (each of the five domains having total scores of 20) was used. It has been used to assess cognitive impairment in adults with SCI [30].

Statistical analysis

Descriptive statistics for all variables were calculated. Statistical power to detect group differences was calculated to be 88% ($\alpha = 0.05$; moderate effect size of 0.30). Participants were dichotomized into those with high PI and PC versus low PI and PC. Dichotomizing these two variables is commonly done in clinical practice. For PI, both groups were dichotomized around a moderate pain score of 4 (low PI group < 4; high PI group ≥ 4). For PC, the groups were dichotomized around the mean PCS for both groups (mean = 11.4; low PC < 11; high PC ≥ 11). While these cut-offs resulted in low numbers in some subgroups (see "Results"), there was no evidence that this negatively influenced the validity of the analysis results. EDS was calculated from sleep latency data generated from the OSLER test. To establish differences in sleep latency

between groups, Kaplan–Meier survival non-parametric analysis was used. Those who completed the 40-min task were judged to have "survived" and "censored". Those who failed seven consecutive 3-s switches (i.e., slept), were judged as an "event". The log-rank test statistic (Mantel–Cox test of equality of survival) determined sleep latency differences.

Multidimensional scaling (MDS) is a multivariate technique used to demonstrate relationships visually in Euclidean distance (i.e., straight-line distances between variables in a two-dimensional space). Factors highly related will appear closer together, while factors that are less related will be placed at greater distances from each other. MDS has distinct advantages in representing relationships between variables compared with other strategies like scatter plots and correlation analyses [31]. MDS was conducted only in the SCI sample to reveal closeness of relationships (in distance) between the study factors of interest, as well as a variety of variables that have a historical or theoretical basis for a relationship to pain and sleepiness/fatigue, such as injury, mental health, and psychosocial factors. To perform MDS, an alternating least-squares algorithm (ALSCAL) was used, and the advantage of using MDS is that it is well suited for ordinal data analysis [31]. To conduct the MDS, relevant factors were placed into a 2-dimensional plot beginning with a dissimilarity matrix and then converting to a distance matrix. Given that many of the factors in this study are self-report and subjective, the analysis was directed to create distances from the data. SPSS was used to conduct the survival and MDS analyses (version 22; https://www.ibm.com/au-en/analytics/spss-statistics-software).

Results

Table 1 shows sociodemographics for the two groups. The groups were not significantly different for age, sex, years of education, BMI, or hours slept the night prior to the experiment. Participants with SCI had significantly increased risk of sleep apnea ($\chi^2_1 = 7.1$, $p < 0.01$). Injury characteristics are shown in Table 2. Table 3 shows comparisons between the two groups for the study factors. From Table 3, the SCI group had significantly higher PI, interference, and PC. Participants with SCI had significantly lower OSLER sleep latency, higher self-reported fatigue and eye blinks ≥500 ms, lower pain-related self-efficacy, and cognitive capacity. There was a trend for the SCI group to have higher levels of psychological distress.

Figure 1 shows the Kaplan–Meier sleep latency survival analysis plot for the four subgroups for PI: SCI low PI ($n = 15$), high PI ($n = 29$); AB low PI ($n = 37$), high PI ($n = 6$) subgroups (one SCI participant and one AB participant did not complete the OSLER task). There were no differences between the two SCI subgroups ($\chi^2_1 = 0.08$, $p =$ ns) or between the two AB subgroups ($\chi^2_1 = 0.68$, $p =$ ns). However, there were significant sleep latency differences between the SCI and AB groups ($\chi^2_1 = 18.5$, $p < 0.001$).

Table 1 Sociodemographics for the SCI ($n = 45$) and AB Control ($n = 44$) groups.

Factor	SCI	AB control	p value (t test or χ^2)
Age (mean, SD)	50.4 (18)	48.9 (19)	0.72
Sex			
Male	38	37	
Female	7	7	0.96
Years education (mean, SD)	13.9 (1.6)	14.2 (1.8)	0.42
Hours slept night before (mean, SD)	8.9 (1.4)	7.1 (1.2)	0.42
Body mass index (frequencies)			
Underweight <18.5	7	7	
Normal 18.5–24.9	15	9	
Overweight 25–29.9	16	18	
Obese >30	7	9	0.61
Sleep apnea (frequencies)			
No sleep apnea	18	30	
Sleep apnea	27	14	0.01

Figure 2 shows the Kaplan–Meier sleep latency survival plot for the four PC subgroups: SCI low PC ($n = 24$), high PC ($n = 20$); AB low PC ($n = 34$), high PC ($n = 9$) subgroups. Overall differences were found between the four subgroups ($\chi^2_3 = 26.8$, $p < 0.001$). There were no differences found between the two AB subgroups (3/34 or 8.8% slept in the low-PC-AB subgroup, 1/9 or 11.1% in the high-PC subgroup). However, the SCI subgroups were significantly different (8/24 or 33.3% slept in the low-PC-SCI subgroup, 14/20 or 70.0% slept in the high-PC subgroup: $\chi^2_1 = 4.5$, $p < 0.05$). Further, the AB subgroups were significantly different to the SCI low-PC subgroup ($\chi^2_2 = 6.9$, $p < 0.05$) and high PC subgroup ($\chi^2_2 = 30.3$, $p < 0.001$). The proportion of low (T4 or lower) versus high lesions (T3 or higher) was evenly distributed in the SCI subgroups for PI and PC.

Figure 3 shows the MDS plot for study factors and their relationship (in distance) between each other. PI, PC, pain interference (PBDS), sleepiness (OSLER latency), sleep apnea, and fatigue (FSS and slow eye blinks), form a distinct cluster along with depressive mood (POMS tension not entered given its close association to depressive mood), BMI, social support, and years of education. Self-efficacy factors sit to the left of this cluster, along with resilience. Cognitive capacity sits to the far left and social participation sits alone in the left-top corner.

Table 2 Injury and pain variables for SCI participants ($n = 45$).

Variable	Mean (SD)	Frequencies (%)
Years since injury mean (SD)	10.4 (13)	
Age at injury mean (SD)	40.1 (19)	
Cause of injury (%)		
Motor vehicle		14 (31.1)
Sport		6 (13.3)
Falls		19 (42.2)
Nontraumatic		3 (6.7)
Other		3 (6.7)
Level of injury		
Cervical		21 (46.7)
Thoracic		20 (44.4)
Lumbar/Sacral		4 (8.9)
AIS grade		
A		27 (69.3)
B		2 (5.1)
C		5 (12.8)
D		3 (7.7)
C/D		2 (5.1)
Compensation		
Yes (%)		43 (95.5)
Types of pain		
Neuropathic		31 (68.8)
Musculoskeletal		35 (77.7)
Visceral		10 (22.2)
Any pain		
Yes (%)		41 (91.1)

The AB control group had 67% with any pain, the majority of which was musculoskeletal (62.8%), with only 7% reporting neuropathic pain. Only one control reported they experienced visceral pain. Only 39 participants with SCI knew their AIS grade.

AIS American Spinal Injury Association Impairment Scale.

Discussion

Unsurprisingly, our data revealed that adults with SCI have an alarming profile consisting of elevated chronic PI, pain interference, fatigue, and elevated EDS and sleep apnea, and elevated PC [2, 5, 8, 10]. The SCI group had significantly higher PC scores than the controls. It is not unusual for chronic injury groups like SCI that have a high occurrence of persistent chronic pain and elevated anxiety and poor mood to have clinically elevated PC [2]. PC scores of 25 are indicative of very high PC [32]. In the SCI group, over one in five (22.7%) had PC scores of at least 26. Further, they had significantly decreased pain-related self-efficacy, a known mediator between pain and depressive mood, resulting in increased risk of depression and pain [3]. Mental health measures such as depressive mood and anxiety were trending higher in the SCI group compared with the controls. SCI adults are higher at risk of mental disorders [6].

SCI was associated with increased risk of EDS, most likely due to respiratory sleep disorders (e.g., obstructive sleep apnea), lesions to melatonergic loops, impaired temperature regulation, and increased psychological distress [6, 7]. It was hypothesized that clinically elevated levels of PI (≥4), would increase the risk of EDS. This was not supported. While large differences in EDS occurred between the two groups, no difference in EDS was found between the PI SCI subgroups. Arguably, this sleepiness impact is due to the influence of SCI rather than PI [8]. The lack of difference in EDS associated with PI may be due in part to the gating influence of sleep on ascending thalamic mechanisms.

It was hypothesized that elevated PC (<11 versus ≥11) would increase the risk of EDS. This hypothesis was supported. Many more participants in the SCI high-PC subgroup (70%) slept compared with the low-PC SCI subgroup (33%). While evidence has shown sleep disturbance is a greater problem in those with high lesions (e.g., T3 or higher) [8], the proportion of high lesions was evenly distributed within the SCI subgroups, suggesting the sleep latency differences were due, at least in part, to PC, and not to lesion level. Further, we have found no differences in EDS between complete versus incomplete lesions in these participants with SCI in the same dataset [8].

The above findings support the view that PC (a top-down factor) rather than PI (a bottom-up factor), has a greater influence on sleepiness [11, 12]. Prior research in healthy individuals has shown PC to be negatively related to prefrontal cortical regions active in top-down regulation of pain, suggesting that PC may weaken prefrontal cortical regulation of pain, reducing one's capacity to disengage from or suppress pain [14]. PC has also been shown to be associated with depressive mood states in adults with SCI [2]. Therefore, PC, in association with other relevant factors like depressive mood and fatigue (as suggested by the MDS analysis), likely contributes to diminished top-down processes (e.g., attention and affect/emotions) [2, 3, 10, 15] and the effectiveness of cortical circuitry involved in diffuse noxious inhibitory control of pain [30]. While the data gained from the univariate survival analyses are limited by lack of control of other potential contributors (like depression, fatigue, presence of sleep disorder, and so on), the finding does implicate and highlight the negative influence of PC on top-down resources that results in increased risk of EDS in adults with SCI. More research is required to understand the cause of this association, and whether treating cognitive biases like PC can improve sleep and daytime functioning.

While it is acknowledged that many factors will influence sleepiness and sleep disorder in SCI, the finding of

Table 3 Descriptive statistics for pain, sleep and psychosocial factors with *p* values for differences between groups.

Variable	SCI Group			AB Group			*p* value
	n = 45			*n* = 44			
	Mean	SD	±95% CI or %	Mean	SD	±95% CI or %	
Pain intensity	4.58	3.1	3.6–5.5	1.91	2.5	1.2–2.7	<0.001
Pain interference	12.1	9.8	9.1–15.1	4.74	6.7	2.7–6.8	<0.001
Pain Catastrophizing Scale	14.0	13.7	9.9–18.2	6.69	6.2	4.8–8.6	<0.01
OSLER sleep latency	28.9	13.7	24.8–33.1	38.6	4.8	37.2–40.1	<0.001
Fatigue severity scale	3.78	1.4	3.3–4.2	2.94	1.4	2.5–3.4	<0.01
% Post-task eye blink duration ≥500 ms	46.6			11.4			<0.001
PSEQ	42.5	12.8	38.6–46.4	53.1	8.8	50.4–55.8	<0.001
MSES	80.2	19.1	74.3–86.2	–	–	–	
CD-RISC	72.2	17.5	66.9–77.6	75.7	10.4	72.5–78.9	NS
POMS DM	11.1	11.0	7.7–14.5	6.86	15.7	2.0–11.7	NS
POMS anxiety	7.6	6.6	5.6–9.7	6.09	4.7	4.6–7.6	NS
SF-SSQ	24.4	19.5	18.4–30.4	24.8	15.0	20.2–29.4	NS
IPAQ	83.5	26.6	75.3–91.7	–	–	–	
NUCOG total score	93.35	5.4	91.7–94.9	96.26	3.5	95.2–97.3	<0.01

PSEQ Pain Self-Efficacy Questionnaire, *MSES* Moorong Self-Efficacy Scale, *CD-RISC* Connor-Davidson Resilience Scale, *POMS DM* profile of mood states depressive mood domain, *POMS Anxiety* profile of mood states tension domain, *SF-SSQ* Short-Form Social Support Questionnaire, *IPAQ* Impact on Participation and Autonomy Questionnaire, *NUCOG* Neuropsychiatry Unit Cognitive Assessment Tool.

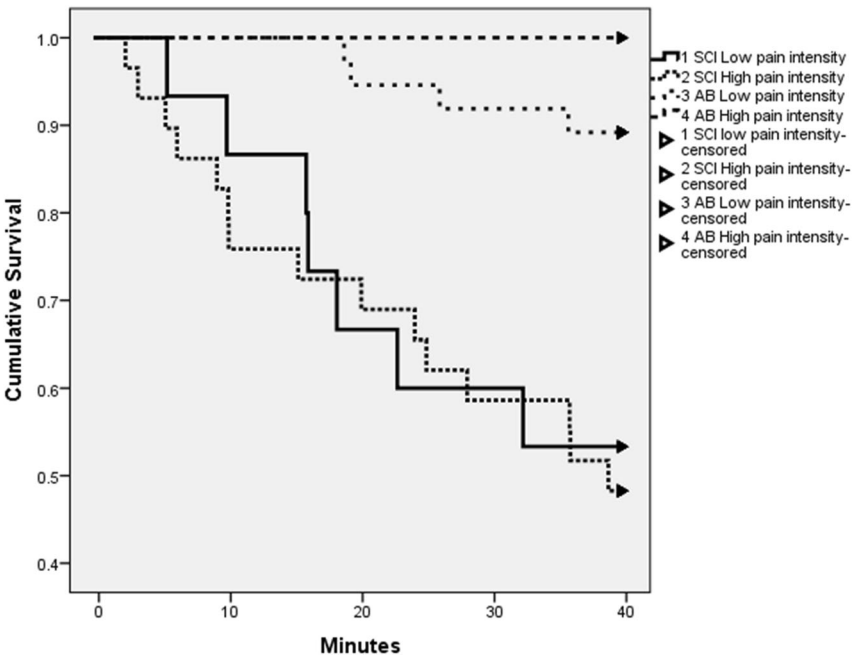

Fig. 1 Kaplan–Meier sleep latency survival estimate plots for the SCI and AB subgroups by PI. "Censored" indicates the percentage in each group that completed the 40-min task without sleeping. For the SCI low PI subgroup, 53.3% ($n = 8/15$) survived the task, for the SCI high PI subgroup, 48.3% ($n = 14/29$) survived. For the AB low PI subgroup, 89.2% ($n = 33/37$) survived, while 100% ($n = 6/6$) of the high AB high PI subgroup survived. One SCI and one AB did not complete the OSLER task, so they were removed from the analysis. The performance of the AB low PI group is shown by dots.

this association between PC and sleepiness is important as it enhances our understanding of how to manage chronic pain and sleep disturbance. Although establishing effective treatments for sleep disorders and EDS is challenging, especially in SCI, studies investigating the efficacy of treatments for PC in adults with SCI and chronic pain are rare [33], though PC has been shown to be significantly reduced in adults with SCI in a broad-based pain-management program [34]. A possible strategy for treating pain and sleep disorder/sleepiness in adults with SCI could be to target PC [2], best perhaps within a multimodal pain-management approach. Establishing efficacy for such an approach should be a priority for future research.

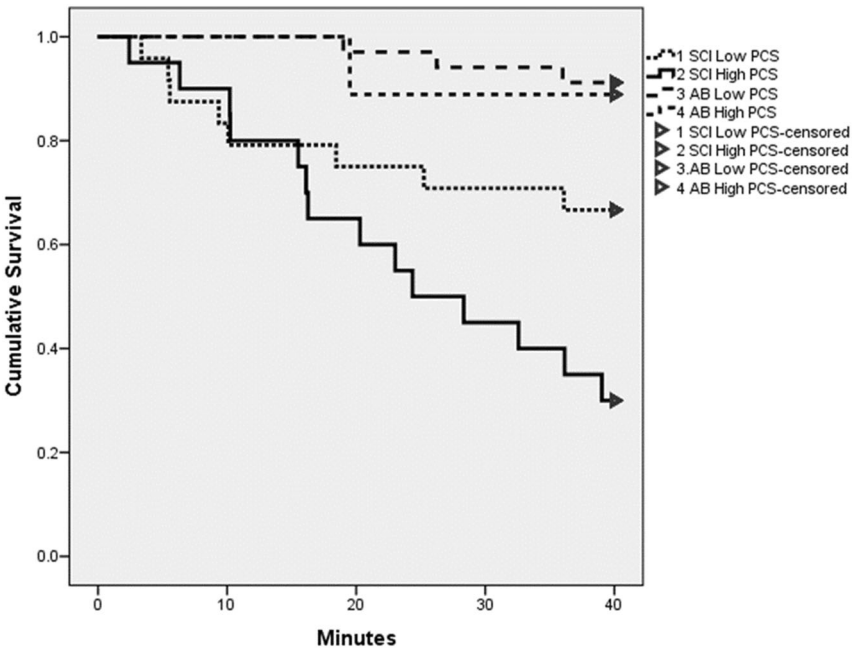

Fig. 2 Kaplan–Meier sleep latency survival estimate plots for the SCI and AB subgroups by PC. "Censored" indicates the percentage in each group that completed the 40-min task without sleeping. For the SCI low-PC subgroup 66.7% ($n = 16/24$) survived the task, for the SCI high-PC subgroup 30% ($n = 6/20$) survived. For the AB low-PC subgroup 91.2% ($n = 31/34$) survived while 88.9% ($n = 8/9$) of the high AB high-PC subgroup survived. Two SCI and one AB did not complete the OSLER task, so they were removed from the analysis.

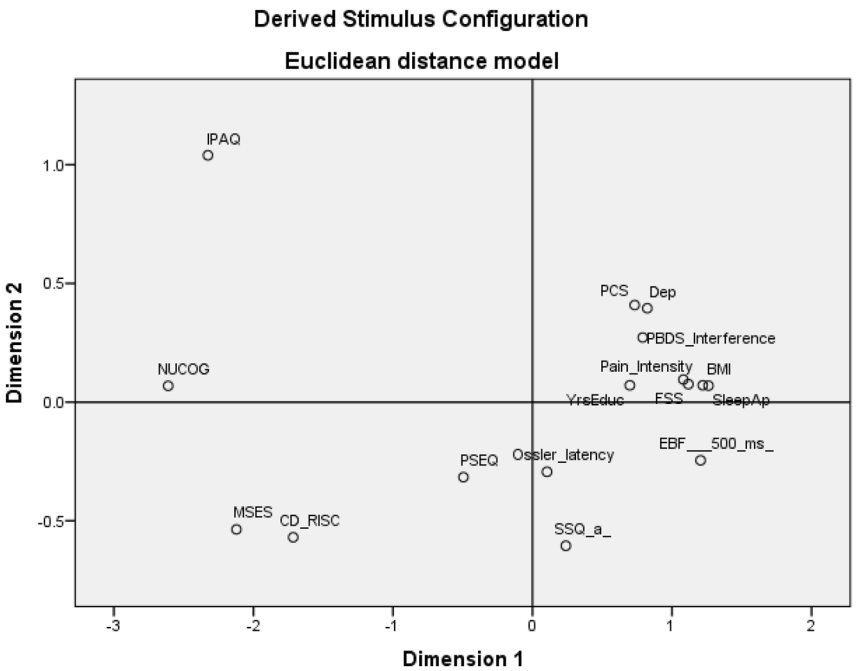

Fig. 3 Shows the multidimensional scaling 2-dimensional plot of pain, sleepiness, fatigue factors, and the relationship in distance to psychosocial factors in Euclidean space for the total SCI group. PCS pain catastrophizing, Dep POMS depressive mood, PBDS interference Pain Basic Data Set interference, BMI body mass index, SleepAp Berlin Questionnaire, FSS Fatigue Severity Scale, YrsEduc years of education, EBF 500 ms eye blink duration ≥500 ms, SSQ a Social Support Questionnaire actual support, PSEQ Pain Self-Efficacy Questionnaire, CD-RISC Connor-Davidson Resilience Scale, MSES Moorong Self-Efficacy Scale, NUCOG Neuropsychiatry Unit Cognitive Assessment Tool, IPAQ Impact on Participation and Autonomy Questionnaire.

It was further hypothesized that multiple factors would be associated with EDS and pain factors. This hypothesis was supported. Figure 3 shows how factors relate to EDS and pain. A distinct cluster was found, containing PI, PC and pain interference, as well as sleepiness, sleep apnea, and self-reported and physiological-based fatigue. This demonstrates the close relationship that sleep/sleepiness/fatigue has with pain factors. This cluster also contained BMI, depressive mood, and years of education, and these were not unexpected, given elevated BMI is related to sleep

disturbance such as sleep apnea [5], while increased depressive mood and lower education have been shown to be associated with increased chronic pain in SCI [3].

The above data support the SCI Adjustment Model (SCIAM) [35]. SCIAM distinguishes between preinjury factors, moderators, mediators, and outcomes. The cluster revealed in the MDS analysis contains prominent moderators that influence adjustment, including PI, pain interference, sleep factors, fatigue, social support, mood states, and BMI. Notably, PC was located in this "moderator" cluster. As a moderator, PC may be a result of an underlying cognitive attentional bias influencing cognitive dimensions, pain being one. In addition, SCIAM classifies cognitive status as a preinjury and postinjury moderator, so its independent placement from the moderator cluster seems reasonable, as is the placement of the resilience and self-efficacy factors, which are classified as mediators in SCIAM [35, 36]. Social participation is best viewed as a moderator, so its placement outside the moderator cluster requires clarification.

Limitations include sleep apnea diagnosis based on the BQ screen. Future research on sleep disorder, EDS, and pain should employ gold standard assessment for sleep apnea to clarify its possible contribution to daytime sleepiness and pain. Another limitation involved participants not being monitored for use of substances just prior to the experiment. However, most reported they complied with the request. A further limitation involves possible confounding of the survival analyses by other factors that influence EDS. In conclusion, prior research using the same dataset has found that EDS in adults with SCI was related to autonomic imbalance, suggesting sleep disturbance in SCI could be managed by improving autonomic balance [8]. The current findings also provide positive direction for improving clinical management of pain and sleep in SCI. Interventions could include, in addition to heart rate and respiratory feedback training to improve function like autonomic balance [8], anti-PC cognitive restructuring strategies based within a pain-management program in addition to improving skills in managing psychosocial stresses [2, 34]. It is hoped such treatments may result in increased alertness and productivity during the day, reduced pain, improved social access, and QOL.

Data availability

The dataset generated during the current study are available from the corresponding author on reasonable request.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethics statement We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during the course of this research. The study was approved by the Northern Sydney Central Coast Human Research Ethics Committee (0812-257M 08/HAWKE/167/168).

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