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Subjective sensory hypersensitivity in chronic acquired brain injury patients: the relationships with insomnia, hyperarousal and perceived stress

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ABSTRACT

Sensory hypersensitivity following acquired brain injury (ABI) is frequently reported and affects well-being, yet remains poorly understood. Research in neurotypical individuals suggests a link between hypersensitivity, insomnia, hyperarousal and perceived stress. This study examined the relationship between sensory sensitivity and insomnia in ABI patients and whether hyperarousal and/or perceived stress mediates this relationship. In an online cross-sectional cohort study among 188 chronic ABI patients of University Medical Centre Utrecht and 61 neurotypical controls, sensory hypersensitivity was measured using the Multi-Modal Evaluation of Sensory Sensitivity, insomnia using the Insomnia Severity Index, hyperarousal using the Hyperarousal Scale, and perceived stress using the Perceived Stress Scale. Associations were examined using multiple regression and mediation analyses with bootstrapping. The results confirmed that sensory hypersensitivity was frequent (66%) and persistent following ABI, mainly in visual and auditory modalities. Increased sensory hypersensitivity was related to higher severity of insomnia in ABI patients. This relationship was partially mediated by hyperarousal and perceived stress. Hyperarousal and perceived stress may underlie the link between sensory hypersensitivity and insomnia, although the direction of these effects remains unclear.

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Introduction

Acquired brain injury (ABI) refers to brain damage that occurs after birth, excluding injuries from congenital conditions, birth trauma or neurodegenerative diseases (Goldman et al., 2022). Among the many consequences of acquired brain injury (ABI), sensory hypersensitivity is one of the most frequently reported (Callahan & Lim, 2018). Despite the disruptive influences of sensory hypersensitivity in ABI patients, it remains largely overlooked both in clinical practice and research (Callahan & Lim, 2018; Thielen et al., 2023). It is suggested that sensory hypersensitivity is associated with insomnia, hyperarousal and perceived stress, which are other commonly observed symptoms following ABI (Callahan & Lim, 2018; Elliott et al., 2018; Engel-Yeger & Shochat, 2012; Miller et al., 1999; Milner et al., 2009). Nonetheless, the underlying mechanisms of sensory hypersensitivity and relationships to other constructs remain unclear (Callahan & Lim, 2018; Thielen et al., 2023).

Sensory hypersensitivity after ABI is defined as self-reported post-injury increase in perceived sensitivity to sensory stimuli, which may manifest itself as an altered response to sensory stimuli (Thielen et al., 2023). Sensory hypersensitivity can have profound effects on communication, cognition, physical and mental health, return to work and (social) participation (Callahan & Lim, 2018; de Sain et al., 2023). Consequently, it is also an important marker for prolonged recovery post-ABI (Chorney et al., 2017; Dischinger et al., 2009; Forrest et al., 2018). To date, research on sensory hypersensitivity primarily has focused on sensory hypersensitivity in mild traumatic brain injury (TBI) (Thielen et al., 2023). However, sensory hypersensitivity is also reported after other types of ABI, such as stroke (Mak et al., 2005). Furthermore, research has mostly concentrated on light and noise hypersensitivity (Thielen et al., 2023). Although sensory hypersensitivity is most common in these sensory modalities (auditory 56% & visual 53%), it is also prevalent in other sensory modalities including the perception of motion (23%), environmental temperature (21%), olfactory and gustatory stimuli (17%), and tactile stimuli (11%) (Thielen et al., 2024a).

Beyond sensory disturbances, sleep problems are another frequent and disabling consequence of ABI, with insomnia being one of the most prevalent. Insomnia affects about one-third of patients with ABI, is associated to poorer (mental) health and quality of life, and may hinder ABI recovery by disrupting neuroplasticity (Bassetti & Hermann, 2011; Daley et al., 2009; Duss et al., 2017; Leppävuori et al., 2002; Mathias & Alvaro, 2012; Ouellet et al., 2015). Earlier research found a significant link between sensory sensitivity and sleep, with higher sensory sensitivity associated with poorer sleep in neurotypical controls (Bastien et al., 2008; Devoto et al., 2005; Engel-Yeger & Shochat, 2012; Milner et al., 2009) and other patient populations such as individuals with cerebral palsy (van Rijssen et al., 2025). In veterans with TBI, sleep disturbances correlated significantly with the severity of sensory hypersensitivity, with insomnia being the

strongest predictor (Elliott et al., 2018). Hyperarousal is proposed as a key mechanism underlying this relationship (Elliott et al., 2018; Riemann et al., 2010; Woods et al., 2013). It is characterized by an abnormal state of increased responsiveness to stimuli marked by physiological and psychological symptoms such as elevated heart rate, respiration and increased levels of alertness and anxiety (Riemann et al., 2010). According to the Hyperarousal Model of Insomnia, chronic insomnia is accompanied by increased autonomic nervous system activity, with psychological symptoms contributing to its persistence (Riemann et al., 2010). Hyperarousal also lowers sensory thresholds (the lowest intensity at which a stimulus is detected), making it more difficult to regulate sensory input during both day – and nighttime, which can lead to sensory hypersensitivity and insomnia (Devoto et al., 2003; Milner et al., 2009; Woods et al., 2013). Evidence for this mechanism is found in Fragile-X syndrome, where hyperarousal is associated with increased sensory sensitivity (Miller et al., 1999), and in veterans with post-TBI sensory hypersensitivity, who show elevated heart rates during sleep even after controlling for PTSD (Elliott et al., 2018).

Perceived stress is another potential factor in the sensory hypersensitivity-insomnia relationship. Perceived stress is defined as how much individuals evaluate situations in their lives as stressful (Cohen et al., 1983). While hyperarousal entails heightened physiological and psychological alertness, perceived stress focuses on the cognitive appraisal of situations. The two are closely related, but stress adds value by capturing the subjective experience of stressors, which can influence coping and emotional responses beyond physiological arousal. The anxiety hypothesis suggests that stress and anxiety can lead to a hyperaroused sympathetic nervous system, which in turn increases sensitivity to sensory stimuli (Shepherd et al., 2015). This is supported by evidence linking post-brain injury sensory hypersensitivity to anxiety and post-traumatic stress (Callahan & Lim, 2018; Elliott et al., 2018; Shepherd et al., 2019). Additionally, individuals with insomnia often perceive life as more stressful, rely more on emotion-focused coping strategies and report higher levels of bedtime hyperarousal than good sleepers (Morin et al., 2003). Taken together, these findings suggest that hyperarousal and perceived stress may be central factors in the relationship between sensory hypersensitivity and insomnia. This highlights potential targets for interventions to reduce sensory hypersensitivity and insomnia, support neurorehabilitation, and enhance daily functioning in ABI patients.

In this study, we obtained self-report measures to assess sensory sensitivity, insomnia, hyperarousal, and perceived stress in ABI patients and examined how the severity of these symptoms compares to those in neurotypical controls. Additionally, we investigated the relationship between sensory sensitivity and insomnia in patients with ABI and studied whether hyperarousal and perceived stress act as mediators in this relationship. This study will help provide a deeper understanding of these mechanisms and it will contribute to understanding the causes of both sensory hypersensitivity and insomnia following ABI.

Understanding what mediates this relationship might help identify intervention targets that may reduce sensory hypersensitivity and insomnia, support neurorehabilitation and enhance daily functioning in ABI patients.

Methods

Participants and procedure

The study was approved by the University Medical Centre Utrecht (UMCU) Medical Ethics Committee and the University of Utrecht's Ethical Review Board. The data for this online cross-sectional cohort study was obtained during the COVID-19 pandemic in 2020/2021. The study was conducted among chronic ABI patients and neurotypical controls. ABI patients, selected through a convenience sample, were recruited from the rehabilitation department of UMCU. All individuals who attended the rehabilitation outpatient clinic for the consequences of brain injury between January 2018 and January 2021 were invited via email by their attending physicians to participate. Neurotypical controls were recruited through a convenience sample within the researcher's social network.

Inclusion criteria were: (1) age eighteen years or older, (2) sufficient Dutch language proficiency, and (3) for ABI participants only, a diagnosis of ABI by a neurologist over six months ago (indicating that patients were in their chronic rehabilitation phase). ABI severity was not assessed, and no inclusion or exclusion criteria were applied based on ABI severity. Exclusion criteria applied to all participants involved (1) a diagnosis of psychotic disorder, (2) attention deficit hyperactivity disorder (ADHD), (3) autism spectrum disorder (ASD), (4) dementia, (5) aphasia, (6) tic disorder and (7) a primary sensory deficit that has not been compensated for (e.g., hearing loss or visual impairment). In addition, neurotypical controls were excluded if diagnosed with any brain disorder (i.e., any neurological condition).

To ensure comparability between ABI patients and neurotypical controls, participants were matched on age, gender, and educational level (Verhage scale (Verhage, 1964)). Since the control group initially included a disproportionate number of highly educated young women, 29 participants (32.2%) from this subgroup were randomly excluded using SPSS.

Participants received information about the study via mail and signed an online informed consent form before participation. Voluntary engagement was emphasized, allowing participants to withdraw from the study at any point without consequences. Using a weblink to the online study created by Gorilla Experiment Builder software (https://gorilla.sc), participants completed four self-report questionnaires on their personal computer or laptop at home. The questionnaires, assessing sensory hypersensitivity, hyperarousal, insomnia, and perceived stress, were presented in that order. The total participation duration was approximately 30 min.

Measures

Sensory hypersensitivity

Sensory hypersensitivity was measured by the Dutch version of the *Multi-Modal Evaluation of Sensory Sensitivity* (MESSY-NL) (Thielen et al., 2024a). This self-report instrument, developed for ABI patients, measures subjective sensory sensitivity across several modalities (i.e., visual, auditory, tactile, olfactory, gustatory, motion, temperature and multisensory) (Thielen et al., 2024a). It was recently indicated that the MESSY is a reliable, valid and sensitive tool for post-injury sensory hypersensitivity following ABI (Thielen et al., 2024a). The questionnaire consists of two types of questions. The first type are eight yes/no items in which ABI patients are asked, for each modality separately, if they experienced an increase in their sensitivity from pre- to post-injury. Neurotypical controls are asked to evaluate a possible change in sensory sensitivity over the last month. The second type of questions consists of 30 multiple choice items which assess the severity of sensory sensitivity across the different sensory modalities. These items are answered on a five point Likert scale (1 = never/ not at all to 5 = very often/ extremely). The total score is ranging from 32–160, higher scores indicating higher severity of sensory sensitivity. Additionally, a sub-score per sensory modality can be calculated separately. In this study, the total MESSY score (sum) was used as measure of sensory sensitivity and the total sub-scores (sum) were used to assess sensory sensitivity in the different sensory modalities.

Insomnia

Insomnia was measured by the Dutch version of the *Insomnia Severity Index* (ISI) (Bastien et al., 2001), a 7-item self-report instrument in which participants are asked to evaluate their sleep over the past two weeks. The total score was used as measure of insomnia and ranges from 0 to 28 and the items are 5-point Likert scales (0 = none to 4 = very severe). The higher the total score, the more severe the insomnia. A cut-off of 14 was used for identifying insomnia disorder (Neven, 2014). Previous research has indicated that the ISI is a valid and reliable instrument to quantify perceived insomnia severity and presents a clinically useful tool in screening, as well as outcome measurement. The ISI possesses adequate internal consistency (Cronbach's alpha = .90) (Morin et al., 2011) and has been validated for online use (Thorndike et al., 2011). The Dutch ISI has been validated for use (Neven, 2014).

Subjective hyperarousal

Subjective hyperarousal was measured by the *Hyperarousal Scale* (HAS), a 26-item self-report instrument that measures tendencies to introspect, think about feelings, respond intensely to unexpected stimuli, and other behaviours that putatively involve cortical arousal (Hammad et al., 2001). The items are 4-point Likert scales (1 = not at all to 4 = extremely). The total score was used to

assess hyperarousal and ranges from 26 to 104, higher scores indicating more hyperarousal. Previous research has shown that the HAS-scores correlate with various neurophysiological measures, including EEG arousal measures, total EEG activity and the P300 event-related potential (Hammad et al., 2001; Regestein et al., 1993). Additionally, both the English version (Cronbach's alpha = .74; Hantsoo et al., 2013) and the Italian-translated version (Cronbach's alpha = .81; Bruno et al., 2020) exhibit adequate internal consistency. For the purpose of this study, the HAS was translated into Dutch by three psychology students and a psychologist, who are well-skilled in English. The first version of the questionnaire was back-translated into English by an English native speaker. After back-translation, comparison, and modification of the no-matching items, the final version of the translated scale was formed. When investigating hyperarousal in relation to sensory hypersensitivity, the sensory hypersensitivity related items (6, 12 and 17) were excluded from the total HAS score. The excluded items were part of the "react" sub-score (Pavlova et al., 2001) of the HAS, which concerns reactions to sensory stimuli. These items correlate strongly with the total MESSY score (respectively Pearson's $r = .72$, $r = .64$ and $r = .59$), much higher than the remaining HAS items (maximum of Pearson's $r = .39$). They were removed to avoid conceptual overlap, ensuring that the analysis reflects general hyperarousal rather than overlapping content with the sensory hypersensitivity measure.

Perceived stress

Perceived stress was measured by the Dutch version of the *Perceived Stress Scale (PSS)* (Cohen et al.,1983*),* a 10-item self-report instrument that assesses the perception of stressful experiences over the last month using a 5-point Likert scale (0 = never to 4 = very often). The scale consists of six negative worded items (1, 2, 3, 6, 9 and 10) and four positive worded items (4, 5, 7 and 8). The scores of the positive worded items were reversed, and the total scores ranges from 0 to 40, higher scores indicating more perceived stress. The PSS has demonstrated adequate reliability coefficients: Cronbach's alpha ranging from .75 to .91 (Cohen et al., 1983; Cohen & Williamson, 1988; Cole, 1999). The PSS has also shown validity evidence compared to health behaviours and perceived health (Cohen et al., 1983) and stressful life events and negative affect (Cohen et al., 1993) as criterion measures.

Data analyses

Analyses were conducted using IBM SPSS Statistics (Version 26.0) and JASP (Version 0.19.1). First, the measures of sensory sensitivity, insomnia, hyperarousal and perceived stress were tested for outliers, normality, multicollinearity, linearity, and homoscedasticity of residuals. The ISI compromised 1.5% missing data (max 1 item per participant was missing), which was addressed through

single imputations, calculated based on the non-missing answer's average for the respective participant.

Preliminary descriptive analyses described participant characteristics, sensory sensitivity, insomnia, hyperarousal and perceived stress in both ABI patients and neurotypical controls. Chi-square tests were used to compare categorical variables (gender and educational level) between ABI patients and neurotypical controls, and an independent samples t-test was used to compare age between groups. Independent t-tests facilitated a comparison of average scores between ABI patients and neurotypical controls across the total MESSY, the sensory modality sub-scores of the MESSY, the total scores of the ISI, the HAS and the PSS questionnaires. To control for multiple comparisons with the sensory modality sub-scores of the MESSY, a Bonferroni correction was applied, adjusting the significance threshold to alpha = .006 (with 8 tests). For all other tests, a significance level of alpha = .05 was applied. Additionally, for descriptive purposes, three frequencies were calculated: (1) the number of ABI patients who answered "yes" to questions asking whether they had become more sensitive since their brain injury for each sensory modality, (2) those exhibiting mild-moderate sensory sensitivity (> 1 *SD* above the *M* of neurotypical controls), and (3) those exhibiting severe sensory sensitivity (> 2 *SD* deviations above the *M* of neurotypical controls) in the different sensory modalities was calculated (see Table 2).

To assess the relationship between insomnia and sensory sensitivity in ABI patients, initially univariable regression analyses identified potential predictors of the outcome measure of sensory hypersensitivity. The demographic control variables age (continuous, in years), gender (dichotomized as female vs male) and educational level were examined, as well as the independent variable insomnia. Educational level was measured using the Dutch Verhage classification system, which ranges from 1 (no or incomplete primary education) to 7 (university degree; Verhage, 1964). This system was dichotomized into low-average education (Verhage 1–5, reflecting no/ primary education up to completed secondary or vocational education) vs high education (Verhage 6–7, reflecting higher professional education and university degrees). Variables with a *p*-value < .1 in the univariable analysis (i.e., correlations with the dependent variable) were included in subsequent multiple regression analyses. In Model I the selected control variables (age, gender, educational level) were added, after which in Model II insomnia was added. For all regression analyses, a significance level of alpha = .05 was used. Furthermore, bivariate Pearson's correlation analyses assessed which of the sensory modality sub-scores of the MESSY were related to insomnia. To control for multiple comparisons, a Bonferroni correction was applied, adjusting the significance threshold to alpha = .006 (with 8 tests). To complement this and reduce the risk of overlooking meaningful effects, effect sizes were calculated as Cohen's d for all correlations. Following Cohen's guidelines, d = 0.2, 0.5 and 0.8 were interpreted as small, medium and large effects, respectively (Cohen, 1988).

Moreover, to assess whether the relationship between sensory sensitivity and insomnia in ABI patients is mediated by hyperarousal and/or perceived stress, we used a structural equation modelling (SEM) approach in JASP, with 2000 bootstrapped samples to estimate confidence intervals for the indirect effects. SEM is seen as flexible and appropriate methods for testing mediation models (Gunzler et al., 2013). To ensure that multicollinearity did not bias the mediation analysis, we assessed the Variance Inflation Factors (VIFs) for the predictor (insomnia) and mediators (hyperarousal and perceived stress). A threshold of VIF < 5 was used to indicate acceptable levels of multicollinearity. For the mediation analyses, a significance level of alpha = .05 was used.

Results

Descriptive statistics and preliminary analyses

Samples

Among ABI patients, 203 participants met the inclusion and exclusion criteria and participated in the study. We excluded 7.4% (N = 15) of the participants due to missing data (incomplete questionnaires with more than one missing item), resulting in a total sample of 188 ABI patients. Of the neurotypical controls, 90 participants met criteria and participated in the study. Following the matching procedure described in the methods, 32.2% (N = 19) of the participants were excluded, leaving a final sample of 61 neurotypical controls (see Table 1 for demographic characteristics).

Table 1. Characteristics of ABI patients (N = 188) and neurotypical controls (N = 61).

Demographic characteristics			Statistics
Gender, N	ABI patients	Neurotypical controls	$\chi^2(2) = 0.33$, p = .85
Female	97 (51.6%)	32 (52.5%)	
Male	90 (47.9%)	29 (47.5%)	
Not specified	1 (0.5%)		
Age in years, *M (SD)*	57.5 (13.4)	50.2 (13.6)	t(247) = 4,01, *p*<.001
Educational level, *N*			$\chi^2(2) = 12.92$, *p*<.001
Low-average	71 (37.8%)	8 (13.1%)	
High	117 (62.2%)	53 (86.9%)	
Clinical characteristics			
Type of acquired brain injury, *N*			
Cerebrovascular accident	67 (35.6%)		
Subarachnoid hemorrhage	60 (31.9%)		
Transient ischemic attack	24 (9.8%)		
Brain tumour	33 (17.6%)		
Traumatic brain injury	19 (10.1%)		
Number of acquired brain injuries, *M (SD)*	1.27 (0.73)		
Time since (last) acquired brain injury in months *M (SD)*	30.45 (29.02)		

Note: *N* = Sample size, *%* = Percentage, *M* = Mean, *SD* = Standard deviation, χ^2 = chi-square test; t = independent samples t-test.

Sensory hypersensitivity

The results of the MESSY demonstrated that 66.0% (N = 124) of the ABI patients reported to have become more sensitive to sensory stimuli since ABI. Of this, post-injury sensory hypersensitivity concerning visual (46.3%) and auditory (47.9%) stimuli was most frequently reported (see Table 2 for an overview of the sensory hypersensitivity results). Furthermore, the severity of sensory sensitivity (MESSY total) of the ABI patients was significantly higher compared to neurotypical controls ($t(247) = 4.00$, $p < .001$, two-tailed, $d = 0.59$). Moreover, ABI patients reported significantly higher sensory sensitivity to multisensory, visual, auditory and motion stimuli (all $t > 3.11$, all $p < .002$, all $d > 0.46$), as compared to the neurotypical controls. There was no evidence for significant differences between the groups for sensory sensitivity to tactile, olfactory, temperature and gustatory stimuli (all $0 > t < 0.84$, all $p > .180$). In terms of severity, 73.9% (N = 139) of the ABI patients showed mild-moderate (>1 *SD* above *M* neurotypical controls) sensory sensitivity in at least one of the eight sensory modalities and 59.5% (N = 112) of the ABI patients in two or more sensory modalities. Furthermore, 47.9% (N = 90) of the ABI patients showed severe (>2 *SD* above *M* neurotypical controls) sensory sensitivity in at least one of the eight sensory modalities and 29.3% (N = 55) of the ABI patients in two or more sensory modalities.

Insomnia

Regarding the ISI results, 47.9% (N = 90) of the ABI patients reported one or more sleep complaint(s) and 10.1% (N = 19) of the ABI patients exceeded the ISI cut-off score of 14, suggestive of insomnia disorder. In the neurotypical controls this was respectively 37.7% (N = 23) and 1.6% (N = 1). ABI patients reported an average total score 2.34 higher on the ISI compared to neurotypical controls ($t(247) = 3.10$, $p = .002$, two-tailed, $d = 0.46$).

Hyperarousal

The hyperarousal scores of ABI patients was higher than that of neurotypical controls ($t(247) = 3.31$, $p = .001$ two-tailed, $d = 0.49$).

Perceived stress

The perceived stress scores of ABI patients were significantly higher compared to the neurotypical controls ($t(244) = 3.93$, $p < .001$ two tailed, $d = 0.62$).

Sensory hypersensitivity-insomnia relationship

Univariable and multivariable associations between potential predictors and sensory sensitivity are presented in Table 3. In the ABI patients, age was negatively related to sensory sensitivity (older individuals reported less sensory

Table 2. Overview of scores on MESSY, ISI, HAS, PSS of ABI patients and neurotypical controls and results of independent sample t-tests between mean scores of ABI patients (N = 188) and neurotypical controls (N = 61).

	ABI patients					Neurotypical controls		Independent sample t-test
	M	SD	Increased sensory sensitivity since ABI[a], n (%)	Mild-moderate sensitivity [b], n (%)	Severe sensitivity[c], n (%)	M	SD	t-values (p-values)
MESSY (total)	63.84	20.86		72 (38.3)	35 (18.6)	52.28	15.12	4.00 (.001)** $d = .59$
Multisensory	18.62	7.00	124 (66.0)	101 (53.7)	52 (27.7)	13.49	4.45	5.38 (.001)** $d = .79$
Visual	11.15	4.60	87 (46.3)	79 (42.0)	35 (18.6)	8.36	3.43	4.36 (.001)** $d = .64$
Auditory	12.32	5.78	90 (47.9)	85 (45.2)	47 (25.0)	9.10	3.53	4.12 (.001)** $d = .61$
Tactile	5.53	2.61	13 (6.9)	26 (13.8)	15 (8.0)	6.03	2.48	−1.35 (.180) $d = -.20$
Olfactory	4.77	2.55	19 (10.1)	24 (12.8)	9 (4.8)	5.08	2.47	−0.83 (.406) $d = -.12$
Temperature	5.02	2.36	23 (12.2)	47 (25.0)	20 (10.6)	4.82	1.69	0.60 (.548) $d = .09$
Gustatory	1.26	0.68	20 (10.9)	30 (16.0)	14 (7.4)	1.18	0.53	0.84 (.400) $d = .12$
Motion	5.17	2.25	29 (15.4)	65 (34.6)	25 (13.3)	4.21	1.51	3.11 (.002)** $d = .46$
ISI (total)	5.71	5.45				3.38	3.92	3.10 (.002)* $d = .46$
HAS (total)	57.21	9.70				52.72	7.47	3.31 (.001)* $d = .49$
PSS (total)	18.31	6.53				14.66	5.06	3.93 (.001)* $d = .59$

Overview of scores on the Multi-Modal Evaluation of Sensory Sensitivity (MESSY), the Insomnia Severity Index (ISI), the Hyperarousal Scale (HAS) and the Perceived Stress Scale (PSS) of acquired brain injury (ABI) patients and neurotypical adults and results of independent sample t-tests between mean scores of ABI patients (N = 188) and neurotypical controls (N = 61).

Note: *n* = sample size, % = percentage, *M* = mean, *SD* = standard deviation, *d* = Cohen's d,**p*-value <.05, ***p*-value <0.006 (Bonferroni-corrected).

[a]Self-reported increased sensory sensitivity since brain injury.

[b]> 1 *SD* above the *M* of the neurotypical controls.

[c]> 2 *SD* above *M* of the neurotypical controls.

Table 3. Results of univariate linear regression analyses and multiple regression analyses regarding the effects of age, gender, education and insomnia on sensory hypersensitivity in ABI patients (N = 187).

Predictor	Univariable regression analyses, *ß (p-value)*	Multiple regression analyses, *ß (p-value)*	
		Model I	Model II
Age[a]	−.24 (.001)*	−.23 (.001)	−.21 (.001)
Gender[b]	−.29 (.001)*	−.26 (.001)	−.23 (.001)
Education[c]	−.05 (.538)		
Insomnia	.35 (.001)*		.30 (.001)
R^2		.14 (.001)	.23 (.001)
ΔR^2			.09 (.001)

Note: ß = Standardised regression coefficient, R^2 = explained variance, ΔR^2 = change in explained variance, *p-values of <.1 were entered in the multiple regression analyses.
[a]in years.
[b]0 = female, 1 = male.
[c]0 = low/average education level (Verhage 1–5), 1 = high education level (Verhage 6–7).

hypersensitivity); female gender was positively related to sensory sensitivity and higher insomnia severity was positively related to sensory sensitivity. There was no evidence for a significant relationship between educational level and sensory sensitivity. In Model I of the multiple regression analysis for ABI patients, age and gender accounted for a significant 13.5% of the variance in sensory sensitivity ($R^2 = .135$, $F(2, 184) = 14.37$, $p < .001$, $f^2 = .156$). In Model II, insomnia was added to the regression equation and accounted for an additional 9.2% of the variance in sensory sensitivity ($\Delta R^2 = .092$, $\Delta F(1, 183) = 17.97$, $p < .001$, $f^2 = .101$). In combination, age, gender, and insomnia explained 22.8% of the variance in sensory sensitivity ($R^2 = .228$, $F(3, 183) = 17.97$, $p < .001$, $f^2 = .295$). When examining the association between insomnia and sensory hypersensitivity the strongest correlations with insomnia were observed for the multisensory (r = .411, d = .91), visual (r = .307, d = .64), and auditory (r = .312, d = .65) modalities, indicating medium-to-large correlations and effect sizes. Smaller correlations were observed for olfactory (r = .184, d = .37) and tactile (r = .155, d = .31) modalities, corresponding to small correlations and effect sizes. Correlations with environmental temperature (r = .102, d = .21), gustatory stimuli (r = .076, d = .15), and motion (r = .091, d = .18) were minimal.

Hyperarousal and perceived stress as mediators in the sensory hypersensitivity – insomnia relationship

Multicollinearity diagnostics indicated no significant issues, with all VIF values below the threshold of 5. Specifically, for the model predicting sensory hypersensitivity, VIF values were 1.226 for insomnia, 1.629 for perceived stress, and 1.825 for hyperarousal. In the models predicting the mediators, VIF values were 1. These results suggest that multicollinearity did not affect the interpretation of the mediation analysis.

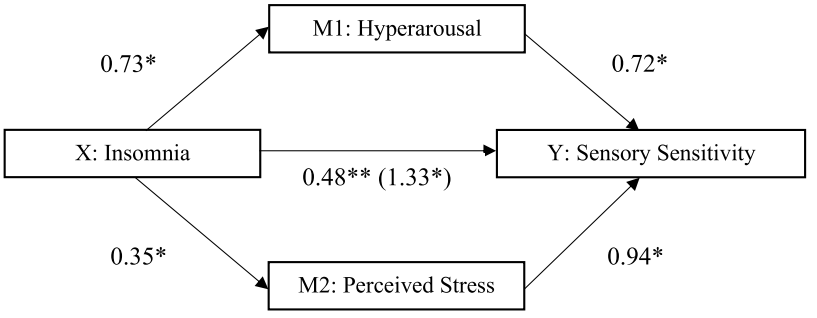

Figure 1. Unstandardized regression coefficients for the relationship between insomnia and sensory sensitivity as mediated by hyperarousal and perceived stress in ABI patients. The regression coefficient between insomnia and sensory sensitivity while controlling for hyperarousal and perceived stress, is in parentheses. Note: *p <.001, **p <.05.

A JASP SEM mediation analysis showed that, in ABI patients, the relationship between insomnia and sensory sensitivity was positive and significant when not accounting for mediators (total effect: B = 1.33, SE = 0.26, t(187) = 5.09, $p < .001$, 95% CI[0.81, 1.83]). Furthermore, a positive relationship was found between insomnia and hyperarousal (B = 0.73, SE = 0.11, t(187) = 6.49, $p < .001$, 95% CI[0.50, 0.93]) and insomnia and perceived stress (B = 0.35, SE = 0.08, t(187) = 4.18, $p < .001$, 95% CI[0.18, 0.54]). When hyperarousal and perceived stress were included as mediators, the direct effect of insomnia on sensory sensitivity was positive but not significant (B = 0.48, SE = 0.24, t(187) = 1.96, $p = .050$, 95% CI[−0.03, 0.98]). Although the coefficient is numerically positive, the 95% confidence interval includes zero, indicating that we cannot conclude a statistically significant direct effect. This suggests that most of the effect of insomnia on sensory sensitivity is transmitted via the mediators. In this mediated model, hyperarousal (B = 0.72, SE = 0.18, t(187) = 4.11, $p < .001$, 95% CI[0.41, 1.01]) and perceived stress (B = 0.94, SE = 0.24, t(187) = 4.02, $p < .001$, 95% CI[0.49, 1.36]) were positively and significantly associated with sensory sensitivity. The indirect effect of insomnia on sensory sensitivity via hyperarousal (B_indirect = 0.52, 95% CI[0.26, 0.82]) and via perceived stress (B_indirect = 0.33, 95% CI[0.17, 0.61]) were both significant, as the 95% confidence intervals did not include zero. These results indicate that hyperarousal and perceived stress partially mediate the relationship between insomnia and sensory sensitivity in ABI patients (see also Figure 1).

Discussion

Post-injury sensory hypersensitivity, insomnia, perceived stress, and hyperarousal are frequently reported in patients with ABI. However, the exact nature of their relationships remains unclear. In this study we investigated the prevalence and severity of these symptoms, examined the relationship between sensory sensitivity severity and insomnia and explored the potential mediating roles

of hyperarousal and perceived stress. Our findings revealed a high prevalence of sensory hypersensitivity in ABI patients, a significant relationship between severity of sensory sensitivity and insomnia, and partial mediation by perceived stress and hyperarousal.

In our sample of 188 ABI patients, two-thirds of the patients reported increased sensitivity to sensory stimuli since their ABI. During the chronic phase of rehabilitation after ABI, approximately half of the patients reported severe sensitivity in at least one sensory modality. This predominantly included auditory (25% of patients) and visual (19% of patients) stimuli, which aligns with previous research (Kashluba et al., 2004; Dikmen et al., 2010). Of the other sensory modalities, post-injury hypersensitivity to motion was most commonly reported, consistent with prior research on motion dysfunction in TBI (Marcus et al., 2019). Furthermore, ABI patients also reported significantly higher levels of insomnia, (subjective) hyperarousal and perceived stress compared to neurotypical controls.

A key finding of this study was the positive relationship between sensory sensitivity and insomnia in chronic ABI patients. This is in accordance with previous research that has linked high sensory sensitivity to poorer sleep quality in both neurotypical controls and veterans with TBI (Bastien et al., 2008; Devoto et al., 2005; Elliott et al., 2018; Engel-Yeger & Shochat, 2012; Milner et al., 2009). Notably, our findings revealed that insomnia was mostly related to sensitivity in the multisensory, visual and auditory modalities, while the other modalities (olfactory, temperature, tactile, gustatory and motion) showed a smaller or no such relationship. This distinction provides new insight, as prior research has not explicitly differentiated the role of these sensory modalities in relation to insomnia.

Furthermore, the relationship between sensory sensitivity and insomnia was found to be partially mediated by hyperarousal and perceived stress, with hyperarousal playing a more prominent role. Notably, once these mediators were included, the direct effect of insomnia on sensory sensitivity was no longer significant, suggesting that the link between sensory hypersensitivity and insomnia may largely operate through these underlying mechanisms rather than reflecting a direct association. These findings align with existing hypotheses on the mechanisms underlying this relationship in other clinical populations (e.g., Elliott et al., 2018; Shepherd et al., 2019). A possible explanation for this relationship is that insomnia is associated with increased hyperarousal and perceived stress, leading to lower sensory thresholds and, in turn, increased sensory sensitivity. This perspective is in line with the anxiety hypothesis, which suggests that heightened physiological arousal due to stress or anxiety can make it more difficult to regulate sensory input both during the day and at night, thereby contributing to sensory hypersensitivity and insomnia (Devoto et al., 2003; Milner et al., 2009; Woods et al., 2013).

However, the directionality of the relationship between sensory hypersensitivity and insomnia remains unclear. On the one hand, as discussed above, insomnia-related hyperarousal and perceived stress may lower sensory thresholds, leading to increased sensory sensitivity. On the other hand, individuals with increased sensory sensitivity may struggle to effectively regulate sensory stimuli both while awake and during sleep (Engel-Yeger & Shochat, 2012). Research suggests that difficulties in selective attention may contribute to sensory hypersensitivity, as individuals with heightened sensitivity often have trouble filtering and prioritizing relevant sensory inputs (Thielen et al., 2024b). Supporting this notion, ABI patients are shown to often experience deficits in selective attention (Alnawmasi et al., 2022; Alnawmasi & Khuu, 2022), making them particularly vulnerable to difficulties in filtering sensory information. In line with this, studies on post-stroke populations have shown that deficits in selective attention are linked to sensory hypersensitivity, particularly in response to visual stimuli (Thielen et al., 2024b). This impaired sensory processing can disrupt the brain's ability to differentiate and modulate relevant sensory inputs, potentially leading to difficulties in transitioning into and maintaining restorative sleep states (Milner et al., 2009). This alternative mechanism suggests that sensory processing deficits during wakefulness may contribute to sleep onset difficulties and disrupted sleep continuity, thus highlighting the complex relationship between sensory processing and insomnia. It is even plausible that a cyclical relationship develops, where sensory hypersensitivity and insomnia perpetuate each other. For instance, increased sensory hypersensitivity disrupts sleep, leading to insomnia, while the resulting lack of restorative sleep and accompanying stress further exacerbate sensory hypersensitivity (Fernández-Mendoza et al., 2010), potentially creating a self-reinforcing loop. The independent mediating roles of hyperarousal and perceived stress in this relationship underscore the interplay between subjective hyperarousal and psychological stress. Hyperarousal has been linked to neuroticism personality traits (Cellini et al., 2017), while perceived stress reflects a sense of control, in which coping plays an important role (Morin et al., 2003). This suggests that individual factors, including personality and coping styles, may influence the sensory hypersensitivity-insomnia relationship. Future research should establish causal directions of these relationships and explore how such individual factors influence these relationships and treatment outcomes.

Given that this study relied on cross-sectional, self-reported data, which limits our ability to establish causal relationships between the variables, further research is needed. Longitudinal research tracking sensory hypersensitivity and insomnia over time is essential to clarify the directionality of these relationships and establish causality. Additionally, the generalizability of the results may be limited by demographic characteristics of the sample, which consisted primarily of younger, highly educated patients. Another factor that limits the generalizability is that ABI severity was not assessed. Given that all participants were

living at home and able to attend outpatient rehabilitation, the sample likely reflected individuals with relatively mild impairments. Future studies should include standardized severity measures to clarify its impact on sensory hypersensitivity and insomnia. Furthermore, data collection took place during the COVID-19 pandemic, which may have influenced stress levels unrelated to ABI and thereby affected the observed relationships between stress, hyperarousal, sensory hypersensitivity and insomnia. Moreover, due to COVID-19 restrictions, all data relied on subjective and self-report measures. Although self-reports are valuable for capturing personal experiences and perceptions, the use of subjective measures, particularly for hyperarousal, may have influenced the results. Hyperarousal was measured subjectively, which may differ from objective physiological measures like heart rate variability or EEG. Furthermore, subjective hyperarousal can overlap with or be influenced by perceived stress. The key distinction is that perceived stress reflects how an individual appraises stressors, while hyperarousal indicates a heightened physical state, such as increased heart rate or alertness. Future studies incorporating objective measures such as polysomnography, EEG, actigraphy and biomarkers like cortisol levels could strengthen the reliability of the current findings on sleep, hyperarousal and perceived stress.

This study highlights the critical need for greater clinical and scientific attention to sensory hypersensitivity symptoms in ABI patients, particularly regarding its link to insomnia. While evidence-based treatments for insomnia are available in this population (Ford et al., 2020), interventions specifically targeting sensory hypersensitivity remain underdeveloped. It is essential to explore comprehensive treatment plans that account for both insomnia and sensory sensitivity complaints. For example, interventions such as sensory modulation therapy or environmental adaptations could complement existing insomnia treatments. Moreover, it would be valuable to investigate whether interventions for insomnia also positively impact sensory hypersensitivity. A reduction in insomnia might alleviate hyperarousal and perceived stress, potentially breaking the negative feedback loop between insomnia and sensory hypersensitivity. Additionally, our study emphasizes the importance of addressing hyperarousal and perceived stress, which were found to mediate the relationship between sensory hypersensitivity and insomnia. Based on these results, it could be valuable to explore interventions that specifically target stress reduction, such as relaxation exercises, mindfulness-based approaches or other forms of stress management, as potential ways to alleviate insomnia and support neurorehabilitation outcomes in individuals with ABI.

In conclusion, this study highlights sensory hypersensitivity as a prevalent and impactful consequence of ABI that deserves greater clinical and scientific attention. This is especially important as it can have detrimental effects on patients' overall well-being, while knowledge about the underlying mechanisms remains insufficient. Our results demonstrate a positive relationship between insomnia

and sensory sensitivity in chronic ABI patients, partially mediated by hyperarousal and perceived stress. Future research should focus on establishing their causal relations and exploring individual differences that may influence these relationships. Also, it is crucial to assess the impact of existing treatments on sensory hypersensitivity and to develop targeted interventions aimed at alleviating these symptoms in ABI patients. Such efforts are essential for creating comprehensive, evidence-based strategies to improve the quality of life of ABI survivors.

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