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Sensory Processing Sensitivity and Migraine-Related Disability: A Longitudinal Mediation Model Through Body Vigilance and Sleep Disturbance

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A B S T R A C T

This study aimed to examine the longitudinal association between sensory processing sensitivity and migraine-related disability and to determine whether body vigilance and sleep disturbance sequentially mediated this relationship among Canadian adults with migraine. A prospective four-wave longitudinal study was conducted with 352 Canadian adults diagnosed with migraine. Data were collected at baseline and at three-, six-, and nine-month follow-ups. Sensory processing sensitivity was assessed using the Highly Sensitive Person Scale, body vigilance was measured with the Body Vigilance Scale, sleep disturbance was evaluated using the PROMIS Sleep Disturbance Short Form, and migraine-related disability was assessed with the Migraine Disability Assessment questionnaire. The hypothesized serial mediation model specified baseline sensory processing sensitivity as the predictor, three-month body vigilance as the first mediator, six-month sleep disturbance as the second mediator, and nine-month migraine-related disability as the outcome. Longitudinal structural equation modeling with robust maximum likelihood estimation and full-information maximum likelihood was used. Earlier levels of the mediators and outcome, age, gender, migraine duration, monthly migraine days, migraine chronicity, and preventive medication use were controlled. The hypothesized serial mediation model demonstrated good fit, $\chi^2(44) = 68.21$, $p = .011$, CFI = .976, TLI = .958, RMSEA = .040, and SRMR = .035. Sensory processing sensitivity significantly predicted three-month body vigilance, $\beta = .310$, $p < .001$; body vigilance predicted six-month sleep disturbance, $\beta = .240$, $p < .001$; and sleep disturbance predicted nine-month migraine-related disability, $\beta = .280$, $p < .001$. The serial indirect effect was significant, $\beta = .021$, 95% CI [.010, .038], $p = .002$. The total indirect effect was also significant, $\beta = .094$, 95% CI [.049, .151], $p < .001$, while the remaining direct effect indicated partial mediation. Greater sensory processing sensitivity may contribute to subsequent migraine-related disability partly through increased monitoring of bodily sensations and later sleep disturbance, highlighting body vigilance and sleep as potentially modifiable intervention targets.

Keywords: *sensory processing sensitivity; migraine; migraine-related disability; body vigilance; sleep disturbance; longitudinal mediation*

1. Introduction

Migraine is a recurrent neurological disorder characterized not only by headache pain but also by sensory amplification, cognitive disruption, autonomic symptoms, sleep problems, emotional distress, and substantial interference with occupational, social, and domestic functioning. Its clinical burden varies considerably across individuals, even among patients with similar headache frequency, suggesting that migraine-related disability cannot be explained solely by attack count or pain intensity. Contemporary migraine research increasingly conceptualizes the disorder as the product of interacting neurological, psychological, behavioral, and environmental processes rather than as an isolated episodic pain condition. Neuroimaging investigations have identified potential biomarkers associated with treatment response in episodic migraine, illustrating the relevance of differences in central nervous system functioning to symptom expression and therapeutic outcomes (Wei et al., 2024). Similarly, emerging research protocols have emphasized the combined contribution of nociceptive, neurocognitive, psychological, and genetic characteristics to pain modulation among individuals with migraine (Fernández-de-las-Peñas et al., 2025). These developments indicate that clinically meaningful disability may arise from the interaction between neurobiological susceptibility and the ways in which individuals perceive, attend to, interpret, and respond to internal and external stimulation. Accordingly, examining dispositional sensitivity and modifiable cognitive and behavioral mechanisms may clarify why some individuals experience persistent disability despite receiving conventional migraine treatment.

Migraine manifests across the lifespan and is shaped by developmental, hormonal, psychiatric, and contextual influences. Pediatric migraine research has highlighted the importance of neurodevelopmental mechanisms, heterogeneous clinical phenotypes, and age-sensitive therapeutic approaches (Al-Beltagi, 2026), whereas research on migraine across the menopausal transition has demonstrated that changes in hormonal status may alter the occurrence, presentation, and management of migraine in adulthood (Korn & Bernstein, 2026). These life-course perspectives reinforce the view that migraine vulnerability is dynamically expressed within a broader biopsychosocial system. Headache psychiatry has likewise emerged as an integrative area concerned with the reciprocal relationships among headache disorders, mood disturbance, anxiety, sleep problems, cognitive processes, and behavioral responses (Otani, 2026). In primary headache management, sleep, anxiety, and mood disorders are not peripheral concerns but practical clinical issues that can influence symptom persistence, treatment adherence, and everyday functioning (Patrick Emanuell Mesquita Sousa & Peres, 2025). Migraine-related disability may therefore reflect more than the direct consequences of pain; it may also embody the cumulative effects of heightened sensory responsivity, persistent monitoring of bodily changes, disrupted sleep, anticipatory concern, and reduced capacity to disengage from symptom-related information.

Sensory processing sensitivity is a temperament-related individual difference involving increased responsiveness to subtle sensory input, deeper processing of environmental information, greater emotional reactivity, and a lower threshold for overstimulation. Individuals high in sensory processing sensitivity may notice minor variations in light, sound, smell, temperature, muscular tension, internal arousal, and affective states that other individuals process less intensely. This heightened responsivity is not inherently pathological and may support perceptual accuracy, empathy, and contextual awareness. However, in the presence of a recurrent neurological condition such as migraine, the same sensitivity may increase vulnerability to sensory overload, fatigue, anticipatory distress, and functional disruption. Migraine attacks are commonly associated with photophobia, phonophobia, osmophobia, motion sensitivity, and difficulty tolerating complex environments. A highly sensitive person may therefore experience migraine-related sensory phenomena as especially intrusive and may devote greater cognitive resources to detecting early signs of an attack. Neurobiological and neuropsychological disturbances described in multisystem conditions such as Ehlers–Danlos syndromes further illustrate how sensory, autonomic, cognitive, and bodily symptoms may cluster and contribute to functional difficulties (Westerman et al., 2025). Likewise, work on the interactions among neuroinfection, immune activity, and neurological disorders emphasizes that neurological symptoms can emerge from complex systemic processes rather than a single localized mechanism (Ngarka et al., 2022). These broader perspectives support investigation of sensory sensitivity as a trait that may interact with migraine-related neurophysiological vulnerability.

One plausible mechanism linking sensory processing sensitivity to migraine-related disability is body vigilance. Body vigilance refers to the tendency to direct sustained attention toward internal bodily sensations, actively scan for

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physiological changes, and interpret subtle somatic cues as salient or potentially consequential. In migraine, monitoring sensations such as pressure, visual disturbance, neck tension, nausea, dizziness, changes in temperature, or fluctuations in alertness may initially serve an adaptive function by facilitating early treatment. Nevertheless, persistent or inflexible monitoring may amplify symptom awareness, increase perceived threat, reinforce anticipatory anxiety, and interfere with ordinary activity. The Body Vigilance Scale has been examined psychometrically across cultural contexts, supporting the measurement of individual differences in attention to bodily sensations (Chahine et al., 2025). Evidence from chronic painful temporomandibular disorders indicates that hypervigilance can coexist with cognitive impairment, greater pain intensity, and catastrophizing (Antunes et al., 2025). Cognitive-behavioral-emotional phenotyping of painful temporomandibular disorders similarly suggests that pain experiences are shaped by interrelated perceptual, cognitive, emotional, and behavioral factors rather than by tissue-level pathology alone (Magri et al., 2025). A narrative review of temporomandibular disorders and somatization further highlights the relevance of bodily symptom interpretation in persistent pain conditions (Xue, 2026). Although temporomandibular pain and migraine are diagnostically distinct, these findings support the possibility that sustained somatic monitoring may contribute to the functional consequences of recurrent pain.

The relevance of body vigilance extends beyond pain disorders to conditions involving dizziness, autonomic symptoms, functional neurological presentations, gastrointestinal discomfort, and medically unexplained or disproportionate symptom burden. Persistent postural-perceptual dizziness is understood through an interaction among perceptual sensitivity, attentional focus, postural control, symptom expectations, and behavioral adaptation (Özdemir et al., 2026). Functional ear symptoms have also been examined through clinical models that integrate symptom experience, comorbidity, and etiological influences rather than assuming a single structural explanation (Scholfield et al., 2022). Research addressing post-concussion syndrome and functional neurological disorder has emphasized overlapping diagnostic interfaces and risk mechanisms that may sustain symptoms after the initial injury (Mavroudis et al., 2025), while related work has identified important points of intersection between persisting post-concussion symptoms and functional neurological presentations (Mollica et al., 2025). Multidisciplinary guidance for post-acute neurological sequelae of SARS-CoV-2 infection similarly recognizes that persistent neurological complaints require integrated assessment of cognitive, emotional, physical, and functional dimensions (Melamed et al., 2023). In autonomic illness, data-driven models linking trauma and postural tachycardia syndrome have further emphasized reciprocal relationships between bodily processes and brain-based interpretation (Crouch et al., 2026). Even theoretical work on irritable bowel syndrome has proposed that bodily sensation, sensory interpretation, and environmental forces may interact in shaping symptom experiences (Spiegel, 2022). Together, these literatures suggest that attention to the body can become a central pathway through which physiological vulnerability is translated into persistent distress and disability.

Body vigilance may also increase migraine-related disability indirectly by disrupting sleep. Individuals who closely monitor bodily sensations may find it difficult to disengage from internal cues during the pre-sleep period. Minor changes in heart rate, muscle tension, head pressure, gastrointestinal activity, temperature, or breathing may become cognitively salient, delaying sleep onset or promoting repeated awakenings. Research on sleep-onset insomnia has identified distinct hypervigilance profiles, including differences associated with psychiatric comorbidity, indicating that excessive monitoring and arousal are not uniform phenomena but clinically meaningful patterns (Abbattista et al., 2026). Experimental work on sleep-related attentional bias in insomnia has further shown that the processing of sleep-relevant information can be examined as a biased attentional decision process (Hui et al., 2026). These findings are compatible with the proposition that individuals who remain alert to bodily or environmental threat cues may experience difficulty transitioning from wakefulness to restorative sleep. Sensory processing sensitivity may intensify this process because highly sensitive individuals may be more reactive to light, sound, temperature, bedding discomfort, changes in breathing, or the expectation of nocturnal and morning migraine symptoms.

The relationship between sleep disturbance and migraine is likely reciprocal. Migraine attacks may interfere with sleep initiation and continuity, while insufficient or poor-quality sleep may reduce pain tolerance, alter nociceptive processing, increase emotional reactivity, and precipitate subsequent attacks. Experimental research on sleep restriction in migraine has directly examined changes in

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nociceptive evoked potentials, supporting a neurophysiological connection between insufficient sleep and pain processing (Omland et al., 2025). A meta-analysis of chronic pain populations has also identified multiple risk factors for sleep disorders, underscoring the frequency with which sleep disruption accompanies persistent pain (Ratu & Murni, 2024). Reviews of sleep disorders and orofacial pain similarly describe clinically important bidirectional relationships between disturbed sleep and painful conditions affecting the craniofacial region (McCloy et al., 2024). Because migraine-related disability includes missed work or study, reduced productivity, impaired household functioning, and withdrawal from social activities, sleep disturbance may magnify disability even when migraine frequency remains unchanged. Fatigue, slowed cognition, irritability, impaired executive functioning, and reduced coping capacity may make ordinary demands more difficult and may increase the perceived and actual impact of each migraine episode.

Sleep is also sensitive to environmental stress, trauma, work schedules, and emotional functioning, all of which may interact with sensory sensitivity and body vigilance. Research conducted in populations exposed to severe war-related trauma has described substantial neurobiological and psychological effects on sleep (Hamamra & Mahamid, 2026), while studies of genocide survivors have emphasized the epidemiology, neuropsychology, and culturally responsive treatment of persistent sleep disturbances (Kizilhan et al., 2026). Adverse childhood experiences have been associated with depression through pathways involving family functioning and insomnia, demonstrating that sleep can operate as an important intermediary between earlier adversity and later mental health outcomes (Tao et al., 2023). Occupational scheduling is another relevant influence, as debates concerning 12- versus 24-hour call duration in acute care surgery illustrate the potential consequences of prolonged work periods and disrupted recovery opportunities (Meshkin et al., 2025). These findings do not imply that all sleep disturbance in migraine is trauma- or occupation-related. Rather, they show that sleep is a responsive regulatory system affected by sustained arousal, cognitive preoccupation, environmental demands, and bodily discomfort. In individuals with high sensory processing sensitivity, these influences may have stronger effects because the threshold for detecting and reacting to stimulation is lower.

A serial pathway from sensory processing sensitivity to body vigilance, sleep disturbance, and migraine-related disability is therefore theoretically plausible. Sensory processing sensitivity may increase the detection and depth of processing of internal and external cues. When migraine is recurrent and unpredictable, heightened cue detection may become organized around the monitoring of prodromal sensations and possible triggers. Persistent body vigilance may then maintain physiological and cognitive arousal into the sleep period, resulting in nonrestorative or fragmented sleep. Sleep disturbance may subsequently increase pain sensitivity, fatigue, cognitive inefficiency, emotional instability, and difficulties performing daily roles. Network approaches to complex pain conditions support the examination of such interacting mechanisms rather than the isolation of a single dominant symptom (Valera-Calero et al., 2022). Studies attempting to discriminate fibromyalgia from localized or regional pain conditions through clinical, psychological, and cognitive patient-reported outcomes similarly demonstrate the value of multidimensional assessment (Cigarán-Méndez et al., 2025). The relevance of lived experience should also not be overlooked, because qualitative research involving families managing persistent and difficult-to-control behavior has shown that burden is shaped by meanings, vigilance, adaptation, and disruption of everyday life, not merely by symptom frequency (Pearson et al., 2026). Although the clinical population in that work differs from adults with migraine, the broader principle is applicable: functional disability emerges through the interaction between symptoms and the continuous demands placed on attention, emotion, family life, and daily routines.

Despite growing recognition of the relationships among sensory reactivity, somatic monitoring, sleep, and pain-related functioning, several limitations remain in the literature. Much of the available evidence is cross-sectional, which prevents determination of whether body vigilance and sleep disturbance prospectively mediate the association between a stable sensitivity trait and later disability. Research frequently examines sensory sensitivity, hypervigilance, insomnia, or pain outcomes separately, leaving their temporal ordering unclear. In addition, studies of migraine disability often prioritize headache frequency and intensity without sufficiently examining how dispositional sensitivity influences cognitive-attentional and sleep-related processes. This gap is clinically important because sensory processing sensitivity is relatively stable, whereas body vigilance and sleep disturbance may be more responsive to behavioral, cognitive, environmental, and sleep-focused interventions. Establishing a longitudinal indirect pathway could therefore identify modifiable

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mechanisms through which highly sensitive individuals become vulnerable to persistent migraine-related impairment. It could also clarify whether sensory processing sensitivity retains a direct relationship with disability after earlier symptom levels, migraine frequency, chronic migraine status, and medication use are considered.

The aim of this study was to examine the longitudinal association between sensory processing sensitivity and migraine-related disability among Canadian adults and to test whether body vigilance and sleep disturbance sequentially mediated this relationship across four measurement waves.

2. Methods and Materials

2.1. Study Design and Participants

This study employed a prospective, four-wave longitudinal design to examine the direct and indirect relationships between sensory processing sensitivity and migraine-related disability through body vigilance and sleep disturbance. Data were collected over a nine-month period, with assessments conducted at baseline and at three-, six-, and nine-month follow-ups. The temporal structure of the study was designed to strengthen the assessment of mediation by separating the predictor, mediators, and outcome across successive measurement occasions. Sensory processing sensitivity was assessed at baseline, body vigilance was examined as the first mediator at the three-month follow-up, sleep disturbance was assessed as the second mediator at the six-month follow-up, and migraine-related disability was evaluated as the primary outcome at the nine-month follow-up. Body vigilance, sleep disturbance, and migraine-related disability were also measured at earlier waves so that their preceding levels could be statistically controlled in the longitudinal mediation model.

The analytical sample consisted of exactly 352 adults with migraine residing in Canada. Participants were recruited from outpatient neurology and headache clinics, primary healthcare practices, university-affiliated health centers, migraine support organizations, and online advertisements distributed through Canadian patient advocacy networks. Recruitment was conducted across several Canadian provinces to increase geographical and sociodemographic diversity. Individuals who expressed interest in the study completed an initial online screening questionnaire and were subsequently contacted by a trained research assistant for eligibility confirmation. Migraine status was verified through a self-reported physician diagnosis followed by a structured screening interview based on the diagnostic characteristics described in the third edition of the International Classification of Headache Disorders. Participants were required to be between 18 and 65 years of age, currently reside in Canada, be able to read and understand English, have access to the internet, and report at least two migraine attacks or four migraine days per month during the three months preceding enrollment. Participants were eligible regardless of whether they experienced migraine with aura or migraine without aura and regardless of whether they were using acute or preventive migraine medication, provided that their medication regimen had remained stable for at least four weeks before baseline assessment.

Individuals were excluded when they reported a major neurological condition other than migraine, including epilepsy, multiple sclerosis, traumatic brain injury with persistent neurological complications, stroke, or a neurodegenerative disorder. Additional exclusion criteria included a current psychotic disorder, an acute manic episode, active substance dependence, untreated obstructive sleep apnea, narcolepsy, or another diagnosed sleep disorder that could independently account for severe sleep disruption. Individuals working rotating or overnight shifts were excluded because irregular work schedules could substantially influence sleep disturbance independently of migraine and sensory processing sensitivity. Participants who were unable to provide informed consent or who were enrolled in another intensive behavioral migraine intervention at the time of recruitment were also excluded. The use of common migraine medications and stable psychological or behavioral treatments was permitted and documented at each assessment.

An a priori Monte Carlo power analysis was performed to determine the sample size required to detect a small longitudinal indirect effect within a serial mediation model. Assuming standardized path coefficients ranging from .15 to .25, a two-sided significance level of .05, and at least 80% statistical power, the analysis indicated that approximately 300 participants would be required. The recruitment target was increased to account for anticipated attrition across the nine-month follow-up period. A total of 401 individuals completed the preliminary screening, of whom 367 met the initial eligibility requirements. Fifteen eligible individuals declined participation or did not complete the baseline consent procedure, resulting in a baseline cohort of 352 participants. All 352 participants provided baseline data and

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were retained in the primary analyses through full-information maximum likelihood estimation. A total of 318 participants completed all four measurement waves, corresponding to a complete follow-up rate of 90.3%. Participants who missed one or more follow-up assessments were not automatically removed from the primary analyses, provided that they had completed the baseline assessment and contributed data to at least one relevant study variable.

Baseline data were collected through a secure web-based research platform. Follow-up questionnaires were distributed electronically three, six, and nine months after baseline. Participants received an email invitation containing an individualized survey link, followed by two standardized reminders when an assessment was not completed within the designated response period. At each wave, participants reported any changes in migraine frequency, acute or preventive medication use, psychological treatment, major medical conditions, and sleep-related diagnoses. They also reported the number of migraine days experienced during the preceding 28 days. The data collection schedule was standardized so that each follow-up assessment was completed within a two-week window surrounding the planned assessment date.

2.2. Measures

Sensory processing sensitivity was measured using the Highly Sensitive Person Scale developed by Aron and Aron. The scale contains 27 items assessing heightened sensitivity to internal and external stimuli, depth of information processing, emotional responsiveness, susceptibility to overstimulation, and awareness of subtle environmental changes. Participants rated each statement on a seven-point Likert scale ranging from 1, representing “not at all,” to 7, representing “extremely.” Items were summed to produce a total score, with higher scores indicating greater sensory processing sensitivity. The total score was used as the principal predictor because the study focused on sensory processing sensitivity as a broad temperamental trait rather than on isolated components of sensitivity. The scale has been widely used in adult populations and has demonstrated acceptable internal consistency and construct validity. In the present study, sensory processing sensitivity was assessed at baseline, before the proposed mediating processes and subsequent migraine-related disability were evaluated.

Body vigilance was assessed using the Body Vigilance Scale. This instrument measures the extent to which individuals consciously attend to, monitor, scan, and detect changes in bodily sensations. The scale evaluates attentional focus on internal physical states, perceived sensitivity to bodily changes, the amount of time spent monitoring the body, and the degree of attention allocated to specific physical sensations. These sensations include changes in heart rate, breathing, dizziness, gastrointestinal activity, muscular tension, pain, temperature, and other internal cues. Higher scores indicate stronger and more persistent monitoring of bodily sensations. The scale was selected because body vigilance represents a theoretically relevant cognitive-attentional mechanism through which highly sensitive individuals may become increasingly aware of migraine-related physiological cues. Body vigilance was measured at baseline and at the three-, six-, and nine-month assessments. The three-month body vigilance score served as the first mediator in the primary longitudinal model, while baseline body vigilance was included to account for pre-existing individual differences in bodily monitoring.

Sleep disturbance was measured using the eight-item Patient-Reported Outcomes Measurement Information System Sleep Disturbance Short Form. The instrument assesses perceived sleep quality, difficulty falling asleep, difficulty remaining asleep, restless or unrefreshing sleep, satisfaction with sleep, and the extent to which sleep is experienced as problematic. Participants rated each item according to their sleep experiences during the previous seven days using a five-point response scale. Raw scores were summed and converted to standardized T-scores according to the established scoring procedure, with higher scores indicating greater sleep disturbance. The PROMIS measure was chosen because it provides a concise and psychometrically supported assessment of perceived sleep problems without restricting sleep disturbance to a single symptom such as sleep duration or insomnia severity. Sleep disturbance was assessed at all four study waves. The six-month sleep disturbance score was treated as the second mediator, while earlier sleep disturbance was controlled to determine whether body vigilance predicted subsequent changes in sleep-related difficulties.

Migraine-related disability was evaluated using the Migraine Disability Assessment questionnaire. The questionnaire assesses the number of days during the preceding three months on which migraine caused complete absence from work or education, reduced productivity at work or education, inability to perform household activities, reduced productivity in household activities, and missed participation in family, social, or recreational activities. The five disability items were summed to calculate the total

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migraine-related disability score, with higher scores representing a greater number of days affected by migraine and more severe functional impairment. The supplementary items concerning headache frequency and average pain intensity were recorded descriptively but were not included in the disability total. Migraine-related disability was assessed at baseline and at every follow-up. The nine-month total score constituted the primary outcome, while prior disability scores were included in the longitudinal model to distinguish prospective associations from the continuation of pre-existing disability.

Migraine frequency was assessed through a 28-day electronic headache diary completed before each major assessment. Participants recorded whether they experienced a migraine on each day, the approximate duration of symptoms, maximum pain intensity, presence of aura, use of acute medication, and interference with usual activities. Monthly migraine days were calculated as the total number of days during the diary period on which participants experienced a migraine headache or used migraine-specific acute medication for a headache consistent with their usual migraine presentation. Participants reporting fewer than 15 headache days per month were classified as having episodic migraine, whereas those reporting at least 15 headache days per month, including at least eight days with migraine characteristics, were classified as having chronic migraine. Monthly migraine days and chronic migraine status were considered potential covariates because both are strongly associated with migraine-related disability.

A demographic and clinical information form was administered at baseline to collect data on age, gender, province of residence, educational level, employment status, relationship status, household income category, age at migraine onset, duration since diagnosis, migraine with or without aura, family history of migraine, common migraine triggers, co-occurring health conditions, and current medication use. Participants identified all acute and preventive migraine medications they had used during the preceding month. Changes in preventive medication, initiation of behavioral treatment, or major changes in migraine management were documented at follow-up. The same clinical update form was administered at the three-, six-, and nine-month assessments to identify changes that could influence the longitudinal associations.

The internal consistency of the multi-item measures was evaluated separately at each measurement wave using Cronbach's alpha and McDonald's omega coefficients. Confirmatory factor analysis was used to examine whether the measurement structure of the sensory processing sensitivity, body vigilance, and sleep disturbance instruments was adequately represented in the Canadian sample. Before calculating total scores, item-level data were inspected for values outside the permitted response range, unusually rapid completion, repetitive response patterns, and excessive missingness. Questionnaire responses were excluded from scale scoring when more than 20% of the items on a measure were missing. When 20% or fewer of the items were missing, the participant's mean score on the completed items of that scale was used to replace the missing item values.

2.3. Data Analysis

Data analyses were conducted using IBM SPSS Statistics and Mplus. Preliminary analyses included data screening, descriptive statistics, examination of score distributions, reliability estimation, attrition analysis, and evaluation of the bivariate relationships among the study variables. Continuous variables were summarized using means, standard deviations, observed ranges, skewness, and kurtosis, while categorical variables were described using frequencies and percentages. Histograms, normal probability plots, standardized residuals, and Mahalanobis distance were examined to identify substantial departures from normality and potentially influential multivariate observations. Because migraine-related disability scores were expected to display positive skewness, all principal path estimates were calculated using robust maximum likelihood estimation, which produces standard errors and test statistics that are less sensitive to non-normality.

Differences between participants who completed all four assessments and those who missed at least one follow-up assessment were examined using independent-samples tests, chi-square tests, and logistic regression analysis. Baseline sensory processing sensitivity, body vigilance, sleep disturbance, migraine-related disability, monthly migraine days, demographic characteristics, and medication status were included as predictors of follow-up completion. Missing longitudinal data were addressed using full-information maximum likelihood estimation under the missing-at-random assumption. This approach allowed all 352 participants to contribute available information to the estimation of the model and avoided the loss of statistical power and potential bias associated with complete-case analysis.

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The hypothesized serial mediation model was tested using longitudinal structural equation modeling. Baseline sensory processing sensitivity was specified as the independent variable, body vigilance at the three-month follow-up was specified as the first mediator, sleep disturbance at the six-month follow-up was specified as the second mediator, and migraine-related disability at the nine-month follow-up was specified as the dependent variable. The model estimated the path from sensory processing sensitivity to subsequent body vigilance, the path from body vigilance to later sleep disturbance, and the path from sleep disturbance to subsequent migraine-related disability. A direct path from sensory processing sensitivity to nine-month migraine-related disability was retained to determine whether the indirect processes fully or partially accounted for the prospective association.

To provide a more rigorous test of temporal mediation, the model adjusted for baseline body vigilance, earlier sleep disturbance, and earlier migraine-related disability. Specifically, three-month body vigilance was predicted while controlling for baseline body vigilance, six-month sleep disturbance was predicted while controlling for three-month sleep disturbance, and nine-month migraine-related disability was predicted while controlling for six-month migraine-related disability. Autoregressive paths were therefore included to account for the stability of the mediators and outcome over time. Covariances among the baseline study variables were freely estimated. Age, gender, monthly migraine days, chronic versus episodic migraine status, duration of migraine, and preventive medication use were included as covariates because these variables could plausibly influence sleep disturbance or functional disability.

The principal indirect effect was defined as the longitudinal serial pathway from baseline sensory processing sensitivity to three-month body vigilance, from three-month body vigilance to six-month sleep disturbance, and from six-month sleep disturbance to nine-month migraine-related disability. Two specific indirect pathways were also estimated: the pathway from sensory processing sensitivity to migraine-related disability through body vigilance and the pathway from sensory processing sensitivity to migraine-related disability through sleep disturbance. Indirect effects were evaluated using 10,000 Monte Carlo draws to generate 95% confidence intervals. An indirect effect was considered statistically significant when its confidence interval did not contain zero. Standardized path coefficients, unstandardized estimates, standard errors, confidence intervals, and exact probability values were reported.

Overall model fit was evaluated using the chi-square statistic, comparative fit index, Tucker–Lewis index, root mean square error of approximation with its 90% confidence interval, and standardized root mean square residual. Comparative fit index and Tucker–Lewis index values of .90 or greater were considered indicative of acceptable fit, with values approaching .95 interpreted as evidence of good fit. Root mean square error of approximation and standardized root mean square residual values below .08 were interpreted as acceptable, while values below .06 were considered indicative of close fit. Model evaluation was based on the combined pattern of fit indices rather than on the chi-square test alone because the chi-square statistic is sensitive to sample size.

Several supplementary analyses were conducted to evaluate the robustness of the findings. The serial mediation model was repeated using only participants who completed all four assessments. It was also estimated separately after excluding participants who initiated or substantially changed preventive migraine medication during the follow-up period. Multigroup analyses examined whether the principal structural pathways differed between participants with episodic and chronic migraine. A competing model in which sleep disturbance preceded body vigilance was estimated and compared with the hypothesized temporal sequence. Finally, the mediation model was repeated with monthly migraine days included as a time-varying covariate to determine whether the proposed indirect relationships remained evident beyond changes in migraine frequency. All statistical tests were two-sided, and a probability value below .05 was considered statistically significant.

3. Findings and Results

The final analytical sample consisted of 352 adults with migraine residing in Canada. Participants ranged in age from 18 to 65 years, with a mean age of 39.68 years and a standard deviation of 11.24 years. Of the participants, 278 were women (79.0%), 68 were men (19.3%), and 6 identified as nonbinary or another gender identity (1.7%). Geographically, 132 participants resided in Ontario (37.5%), 67 in Quebec (19.0%), 54 in British Columbia (15.3%), 39 in Alberta (11.1%), 31 in the Atlantic provinces (8.8%), 19 in Manitoba or Saskatchewan (5.4%), and 10 in one of the Canadian territories (2.8%). Regarding educational attainment, 261 participants held a university degree or

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another postsecondary degree (74.1%), 58 had completed a college diploma or vocational certificate (16.5%), and 33 had completed high school or a lower level of education (9.4%). A total of 198 participants were employed full-time (56.3%), 56 were employed part-time (15.9%), 47 were unemployed, on medical leave, or receiving disability-related support (13.4%), and 51 were students, retired, self-employed without fixed hours, or classified in another occupational category (14.5%). The mean duration of migraine since symptom onset was 15.26 years, with a standard deviation of 10.08 years. At baseline, participants reported an average of 10.84 migraine days during the preceding 28 days, with a standard deviation of 5.73 days. Based on headache-frequency criteria, 248 participants had episodic migraine (70.5%), whereas 104 met the criteria for chronic migraine (29.5%). Migraine with aura was reported by 154 participants (43.8%), while 198 reported migraine without aura (56.3%). At baseline, 144 participants were using at least one preventive migraine medication (40.9%), and 319 reported using an acute migraine medication during the preceding month (90.6%). Follow-up assessments were completed by 341 participants at three months (96.9%), 329 at six months (93.5%), and 318 at nine months (90.3%). Participants who completed all follow-up assessments did not differ significantly from those who missed at least one assessment in age, gender distribution, migraine subtype, chronic migraine status, monthly migraine days, baseline sensory processing sensitivity, body vigilance, sleep disturbance, or migraine-related disability. A multivariable logistic regression model predicting nine-month study completion was also nonsignificant, $\chi^2(8) = 9.84$, $p = .276$, suggesting that attrition was not systematically associated with the principal baseline variables.

Table 1

Descriptive Statistics and Correlations Among the Principal Study Variables

Variable	Mean	SD	Observed range	1	2	3	4	5	6	7	8
1. Baseline sensory processing sensitivity	4.72	0.83	2.22–6.81	—							
2. Baseline body vigilance	23.74	7.29	4–40	.38***	—						
3. Three-month body vigilance	24.61	7.12	6–40	.42***	.68***	—					
4. Three-month sleep disturbance	59.21	7.38	38.8–78.7	.27***	.34***	.39***	—				
5. Six-month sleep disturbance	59.84	7.46	39.3–78.7	.31***	.32***	.43***	.71***	—			
6. Six-month migraine-related disability	34.76	23.95	0–121	.22***	.24***	.29***	.37***	.41***	—		
7. Nine-month migraine-related disability	35.87	24.69	0–128	.25***	.25***	.32***	.39***	.46***	.76***	—	
8. Baseline monthly migraine days	10.84	5.73	4–28	.13*	.15**	.17**	.23***	.26***	.54***	.56***	—

As presented in Table 1, sensory processing sensitivity was positively associated with all proposed mediating and outcome variables. Baseline sensory processing sensitivity was moderately correlated with baseline body vigilance, $r = .38$, $p < .001$, and three-month body vigilance, $r = .42$, $p < .001$, indicating that participants characterized by greater sensitivity to environmental and internal stimulation also reported more persistent monitoring of bodily sensations. Sensory processing sensitivity was also positively related to three-month sleep disturbance, $r = .27$, $p < .001$, six-month sleep disturbance, $r = .31$, $p < .001$, six-month migraine-related disability, $r = .22$, $p < .001$, and nine-month migraine-related disability, $r = .25$, $p < .001$. These correlations provided preliminary support for the hypothesized longitudinal associations but were not sufficiently large to indicate substantial conceptual overlap among the constructs. Body vigilance demonstrated considerable temporal stability, as shown by the correlation between baseline and three-month body vigilance, $r = .68$, $p < .001$. Similarly, sleep disturbance displayed a strong association between the three- and six-month assessments, $r = .71$, $p < .001$, and migraine-related disability exhibited strong stability between the six- and nine-month assessments, $r = .76$, $p < .001$. Three-month body vigilance was positively correlated with six-month sleep disturbance, $r = .43$, $p < .001$, and nine-month migraine-related disability, $r = .32$, $p < .001$. Six-month sleep disturbance was positively related to nine-month migraine-related disability, $r = .46$, $p < .001$. Baseline monthly migraine days had its strongest correlations with six-month disability, $r = .54$, $p < .001$, and nine-month disability, $r = .56$, $p < .001$, confirming the importance of controlling migraine frequency when evaluating psychological and sleep-related predictors of functional impairment. None of the correlations exceeded .80, indicating that multicollinearity was unlikely to compromise estimation of the structural model.

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Table 2

Longitudinal Descriptive Statistics, Internal Consistency, and Distributional Characteristics of the Study Measures

Measure and assessment wave	Valid n	Mean	SD	Skewness	Kurtosis	Cronbach's α	McDonald's ω
Sensory processing sensitivity, baseline	352	4.72	0.83	−0.18	−0.31	.91	.92
Body vigilance, baseline	352	23.74	7.29	0.09	−0.47	.86	.87
Body vigilance, three months	341	24.61	7.12	0.04	−0.39	.87	.88
Body vigilance, six months	329	24.38	7.25	0.08	−0.42	.88	.88
Body vigilance, nine months	318	24.19	7.31	0.11	−0.38	.87	.88
Sleep disturbance, baseline	352	58.92	7.31	0.21	−0.25	.89	.90
Sleep disturbance, three months	341	59.21	7.38	0.18	−0.29	.90	.90
Sleep disturbance, six months	329	59.84	7.46	0.14	−0.32	.91	.91
Sleep disturbance, nine months	318	59.47	7.51	0.16	−0.26	.90	.91
Migraine-related disability, baseline	352	35.42	23.71	1.08	0.76	.85	.86
Migraine-related disability, three months	341	35.11	23.28	1.04	0.69	.86	.87
Migraine-related disability, six months	329	34.76	23.95	1.11	0.88	.87	.87
Migraine-related disability, nine months	318	35.87	24.69	1.15	0.94	.88	.88

Table 2 demonstrates that the study measures showed acceptable to excellent internal consistency across all assessment waves. The Highly Sensitive Person Scale had a Cronbach's alpha coefficient of .91 and a McDonald's omega coefficient of .92 at baseline, indicating that the items provided a highly reliable assessment of sensory processing sensitivity. Reliability coefficients for body vigilance ranged from .86 to .88 for Cronbach's alpha and from .87 to .88 for McDonald's omega. Sleep-disturbance reliability coefficients ranged from .89 to .91, while those for migraine-related disability ranged from .85 to .88. The similarity between alpha and omega estimates indicated that reliability conclusions were not dependent on the assumption of equal item loadings. Sensory processing sensitivity, body vigilance, and sleep disturbance exhibited limited skewness and kurtosis, with all values remaining within conventional thresholds for acceptable univariate distributions. Migraine-related disability showed greater positive skewness, reflecting the presence of a smaller group of participants with very high disability scores. Consequently, robust maximum likelihood estimation was retained for the structural analyses. Mean levels of body vigilance, sleep disturbance, and migraine-related disability remained relatively stable across the nine-month study period. Repeated-measures analyses indicated no significant overall linear change in body vigilance, $F(1, 317) = 1.72$, $p = .191$, sleep disturbance, $F(1, 317) = 2.31$, $p = .129$, or migraine-related disability, $F(1, 317) = 0.68$, $p = .411$. This absence of a substantial sample-wide mean trend was consistent with the observational nature of the study and indicated that the longitudinal associations were not attributable to a uniform increase or decrease in symptom severity. Confirmatory factor analyses supported the proposed measurement structures of sensory processing sensitivity, body vigilance, and sleep disturbance. Longitudinal tests of body vigilance and sleep disturbance supported configural, metric, and scalar invariance, with changes in the comparative fit index remaining below .01 and changes in the root mean square error of approximation remaining below .015. These findings indicated that changes and temporal associations involving these measures were unlikely to reflect alterations in the interpretation or measurement of the constructs over time.

Table 3

Model-Fit Indices for the Primary, Competing, and Sensitivity Models

Model	n	χ^2	df	p	CFI	TLI	RMSEA [90% CI]	SRMR	AIC	BIC
Primary hypothesized serial mediation model	352	68.21	44	.011	.976	.958	.040 [.020, .058]	.035	17,142.60	17,421.90
Complete-case model	318	66.10	44	.018	.974	.955	.040 [.017, .059]	.036	15,471.28	15,742.44
Stable-medication sensitivity model	301	62.74	44	.033	.977	.961	.038 [.012, .058]	.034	14,608.51	14,875.06
Model including time-varying migraine days	352	78.33	52	.011	.974	.956	.038 [.018, .055]	.037	17,098.42	17,408.55
Competing reverse-order mediation model	352	96.84	44	<.001	.949	.917	.059 [.043, .075]	.052	17,176.90	

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The primary hypothesized serial mediation model demonstrated good overall fit to the data, $\chi^2(44) = 68.21$, $p = .011$, CFI = .976, TLI = .958, RMSEA = .040, 90% CI [.020, .058], and SRMR = .035. Although the chi-square test was statistically significant, the remaining fit indices were consistently within the ranges associated with good model fit, and the upper boundary of the RMSEA confidence interval remained below .06. The complete-case analysis involving the 318 participants who completed all four waves produced an almost identical pattern of fit, with a CFI of .974, TLI of .955, RMSEA of .040, and SRMR of .036. This similarity indicated that the results obtained through full-information maximum likelihood estimation were not materially dependent on incomplete follow-up data. The model restricted to 301 participants whose preventive medication regimen remained stable also showed good fit, suggesting that medication changes did not account for the proposed mediation pattern. The sensitivity model incorporating monthly migraine days as a time-varying covariate retained favorable fit indices, with a CFI of .974 and RMSEA of .038. By contrast, the competing model in which sleep disturbance temporally preceded body vigilance showed weaker fit, including a lower CFI and TLI, higher RMSEA and SRMR, and increases of 34.30 points in AIC and 34.30 points in BIC relative to the primary model. The indirect serial pathway in the reverse-order model was also not statistically significant. These results provided greater support for the hypothesized temporal sequence in which sensory processing sensitivity predicted increased body vigilance, which subsequently contributed to sleep disturbance and later migraine-related disability. A multigroup comparison further indicated that constraining the principal structural paths to equality across episodic and chronic migraine groups did not significantly worsen model fit, $\Delta\chi^2(3) = 5.82$, $p = .121$. Thus, there was no sufficient evidence that the principal mediation mechanism differed between participants with episodic and chronic migraine.

Table 4

Standardized Direct, Indirect, and Total Effects in the Longitudinal Serial Mediation Model

Effect	Standardized estimate	SE	95% confidence interval	p
Sensory processing sensitivity → three-month body vigilance	.310	.050	[.212, .408]	<.001
Three-month body vigilance → six-month sleep disturbance	.240	.050	[.142, .338]	<.001
Six-month sleep disturbance → nine-month migraine-related disability	.280	.050	[.182, .378]	<.001
Sensory processing sensitivity → six-month sleep disturbance	.130	.045	[.042, .218]	.004
Three-month body vigilance → nine-month migraine-related disability	.120	.050	[.022, .218]	.017
Sensory processing sensitivity → nine-month migraine-related disability, direct effect	.090	.042	[.008, .172]	.031
Sensory processing sensitivity → body vigilance → migraine-related disability	.037	.015	[.009, .076]	.014
Sensory processing sensitivity → sleep disturbance → migraine-related disability	.036	.013	[.012, .067]	.006
Sensory processing sensitivity → body vigilance → sleep disturbance → migraine-related disability	.021	.007	[.010, .038]	.002
Total indirect effect	.094	.026	[.049, .151]	<.001
Total effect of sensory processing sensitivity on migraine-related disability	.184	.045	[.096, .272]	<.001

As shown in Table 4, baseline sensory processing sensitivity significantly predicted higher body vigilance at the three-month follow-up after baseline body vigilance and the designated covariates had been controlled, $\beta = .310$, SE = .050, $p < .001$. This result indicated that a one-standard-deviation increase in sensory processing sensitivity was associated with an increase of approximately .31 standard deviations in subsequent body vigilance. Three-month body vigilance, in turn, significantly predicted greater sleep disturbance at six months while controlling for earlier sleep disturbance, $\beta = .240$, SE = .050, $p < .001$. Six-month sleep disturbance was subsequently associated with greater migraine-related disability at nine months after prior disability and migraine frequency had been controlled, $\beta = .280$, SE = .050, $p < .001$. Sensory processing sensitivity also had a smaller direct prospective relationship with six-month sleep disturbance, $\beta = .130$, $p = .004$, and three-month body vigilance retained an independent association with nine-month migraine-related disability, $\beta = .120$, $p = .017$. The direct effect of sensory processing sensitivity on nine-month migraine-related disability remained statistically significant after both mediators and all covariates were included, $\beta = .090$, $p = .031$, indicating partial rather than complete mediation. The specific indirect effect through body vigilance alone was significant, $\beta = .037$, 95% CI [.009, .076], as was the specific indirect effect through sleep

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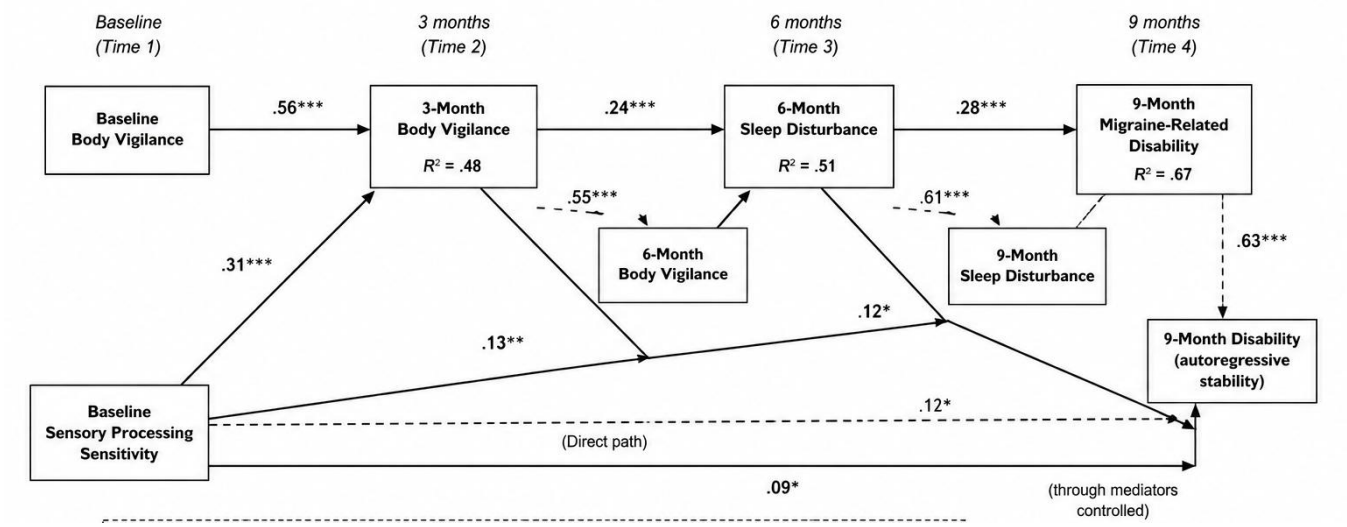
disturbance alone, β = .036, 95% CI [.012, .067]. Most importantly, the hypothesized serial indirect effect from sensory processing sensitivity through body vigilance and then sleep disturbance to migraine-related disability was statistically significant, β = .021, 95% CI [.010, .038], p = .002. The total indirect effect was .094, 95% CI [.049, .151], and accounted for approximately 51.1% of the total association between sensory processing sensitivity and subsequent migraine-related disability. The total effect remained significant, β = .184, 95% CI [.096, .272], p < .001. Among the covariates, baseline monthly migraine days was the strongest predictor of later disability, β = .290, p < .001, while chronic migraine status had a smaller independent effect, β = .120, p = .006. Age, gender, migraine duration, and preventive medication use were not significant independent predictors after migraine frequency, prior disability, and the proposed mediators were considered.

Figure 1

Standardized Path Coefficients for the Longitudinal Serial Mediation Model Linking Sensory Processing Sensitivity to Migraine-Related Disability Through Body Vigilance and Sleep Disturbance



The path model illustrated in Figure 1 showed a sequential pattern in which greater baseline sensory processing sensitivity predicted increased body vigilance three months later, which subsequently predicted greater sleep disturbance at six months and greater migraine-related disability at nine months. All three constituent paths of the proposed serial mechanism were statistically significant after the autoregressive effects of baseline or preceding measures had been included. Baseline body vigilance strongly predicted three-month body vigilance, β = .560, p < .001, three-month sleep disturbance strongly predicted six-month sleep disturbance, β = .610, p < .001, and six-month migraine-related disability strongly predicted nine-month disability, β = .630, p < .001. These autoregressive effects demonstrated substantial temporal stability and established that the significant prospective pathways represented prediction of relative changes in the mediators and outcome rather than simple cross-sectional associations. The model explained 48.0% of the variance in three-month body vigilance, 51.0% of the variance in six-month sleep disturbance, and 67.0% of the variance in nine-month migraine-related disability. The direct path from sensory processing sensitivity to migraine-related disability was reduced but remained significant after body vigilance and sleep disturbance were introduced, further supporting a partial mediation interpretation. The serial indirect pathway remained significant in the complete-case analysis, β = .019, 95% CI [.008, .037], in the stable-medication analysis, β = .022, 95% CI [.010, .041], and in the model controlling for time-varying monthly migraine days, β = .018, 95% CI [.007, .035]. In contrast, the alternative pathway in which sleep disturbance preceded body vigilance was not significant, β = .006, 95% CI [−.001, .018], p = .098. Collectively, the primary and sensitivity analyses indicated that sensory processing sensitivity was prospectively associated with migraine-related functional impairment through both heightened attention to bodily sensations and subsequent disruption of sleep, and that this association could not be explained solely by pre-existing disability,

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migraine frequency, medication status, or the temporal stability of the study variables.

4. Discussion and Conclusion

The present longitudinal study examined whether sensory processing sensitivity predicted subsequent migraine-related disability through the sequential influence of body vigilance and sleep disturbance among Canadian adults with migraine. The findings supported the proposed model. Higher baseline sensory processing sensitivity predicted greater body vigilance at the three-month follow-up, even after controlling for initial body vigilance and relevant demographic and clinical characteristics. Greater body vigilance subsequently predicted higher sleep disturbance at six months, and sleep disturbance predicted greater migraine-related disability at nine months after previous disability and migraine frequency were taken into account. The hypothesized serial indirect effect was statistically significant, indicating that sensory processing sensitivity was associated with later disability partly because highly sensitive individuals became more attentive to internal bodily sensations and subsequently experienced greater sleep disruption. The specific indirect pathways through body vigilance alone and sleep disturbance alone were also significant. However, the direct association between sensory processing sensitivity and migraine-related disability remained significant after inclusion of both mediators, indicating partial rather than complete mediation. Collectively, these findings suggest that sensory processing sensitivity may function as a distal dispositional vulnerability, whereas body vigilance and sleep disturbance represent more proximal and potentially modifiable processes through which this vulnerability is translated into functional impairment.

The positive association between sensory processing sensitivity and later body vigilance is theoretically consistent with the defining characteristics of sensory processing sensitivity. Individuals with elevated sensitivity tend to notice subtle environmental and internal changes, process stimulation more deeply, and become more easily overwhelmed when sensory demands are intense or persistent. In the context of migraine, these characteristics may increase awareness of early physiological changes such as head pressure, neck tension, nausea, visual disturbance, dizziness, temperature fluctuations, muscular discomfort, or changes in alertness. Although attention to these cues can facilitate timely use of acute medication or avoidance of triggers, repeated monitoring may gradually become rigid and threat-oriented. The present findings indicate that sensory sensitivity was not merely correlated with body vigilance at one point in time but prospectively predicted relative increases in bodily monitoring. This observation is compatible with research showing that hypervigilance is related to pain intensity, catastrophizing, and cognitive difficulties in chronic painful temporomandibular disorders (Antunes et al., 2025). It also accords with cognitive-behavioral-emotional models of persistent pain in which bodily sensations acquire heightened salience through reciprocal interactions among attention, interpretation, emotional responses, and behavior (Magri et al., 2025). The association between temporomandibular disorders and somatization further illustrates how increased attention to bodily symptoms may contribute to persistent symptom burden and functional disruption (Xue, 2026).

The finding that body vigilance predicted later sleep disturbance provides an important explanation for how attentional processes may extend the effects of sensory sensitivity beyond waking migraine symptoms. Sleep requires a gradual reduction in cognitive, sensory, and physiological monitoring. Individuals who remain focused on internal sensations may experience difficulty disengaging from subtle changes in breathing, heart rate, muscular tension, head pressure, gastrointestinal activity, or temperature during the pre-sleep period. The present findings suggest that body vigilance may maintain a state of alertness that interferes with the transition into sleep and contributes to fragmented or nonrestorative sleep. This interpretation is supported by evidence that sleep-onset insomnia can involve distinct hypervigilance profiles characterized by persistent cognitive and physiological arousal (Abbattista et al., 2026). Research using attentional decision-making models has likewise shown that individuals with insomnia display biased processing of sleep-related information, indicating that attention itself may participate in the maintenance of sleep disturbance (Hui et al., 2026). In highly sensitive individuals with migraine, the combination of heightened sensory responsivity and repeated scanning for possible attack-related cues may make it particularly difficult to reduce vigilance at bedtime.

The prospective relationship between sleep disturbance and migraine-related disability was also consistent with the broader migraine and pain literature. Experimental evidence indicates that sleep restriction alters nociceptive processing in individuals with migraine, supporting a neurophysiological mechanism through which insufficient

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sleep may intensify vulnerability to pain (Omland et al., 2025). Sleep disruption can reduce pain-inhibitory capacity, heighten emotional reactivity, impair attention and executive functioning, and increase fatigue. These consequences can amplify the functional effects of migraine even when the number of migraine days remains stable. A person who is sleep-deprived may be less able to work efficiently, manage household responsibilities, maintain social engagement, or recover between attacks. Meta-analytic findings from chronic pain populations similarly indicate that sleep disorders are associated with a range of clinical and psychological risk factors and are deeply embedded in the persistence of pain-related problems (Ratu & Murni, 2024). Reviews of sleep disorders and orofacial pain also emphasize a bidirectional relationship in which pain impairs sleep and disturbed sleep subsequently intensifies pain sensitivity and disability (McCloy et al., 2024). The present results extend these observations by showing that sleep disturbance may occupy a longitudinal mediating position between body-focused vigilance and later migraine-related disability.

The significant serial indirect pathway is particularly important because it provides a temporally ordered explanation of how a relatively stable trait may lead to a clinically meaningful outcome. Sensory processing sensitivity did not appear to influence disability through a single isolated mechanism. Instead, it was associated with increased bodily monitoring, which was followed by worsening sleep and then greater functional impairment. This pattern is compatible with multidimensional and network-based accounts of chronic pain, which emphasize that symptoms and psychological processes reinforce one another over time (Valera-Calero et al., 2022). In fibromyalgia and other pain conditions, combinations of clinical, psychological, and cognitive patient-reported outcomes have shown greater discriminatory value than individual symptoms considered in isolation (Cigarán-Méndez et al., 2025). Migraine may similarly be better understood through interconnected processes involving sensory responsivity, attention, arousal, sleep, pain modulation, and everyday functioning. The current model therefore complements neurobiological investigations of migraine by identifying psychological and behavioral processes through which central sensitivity may acquire functional significance.

The findings also align with recent efforts to conceptualize migraine through an integrated neuropsychiatric framework. Migraine is increasingly recognized as a condition in which neurological symptoms interact with anxiety, mood disturbance, sleep problems, and cognitive responses (Otani, 2026). Practical recommendations for managing primary headaches likewise emphasize the clinical relevance of sleep, anxiety, and mood rather than treating these concerns as secondary complications (Patrick Emanuell Mesquita Sousa & Peres, 2025). The present results add sensory processing sensitivity and body vigilance to this framework. Highly sensitive individuals may experience migraine-related environmental stimulation more intensely, monitor prodromal sensations more closely, and become increasingly concerned about whether an attack is developing. This anticipatory process may contribute to avoidance of activity, reduced confidence in bodily functioning, and increased sleep-related arousal. The persistence of a significant direct effect after controlling for body vigilance and sleep disturbance suggests that additional mechanisms may also connect sensory processing sensitivity to disability, including emotional reactivity, avoidance of sensory environments, reduced tolerance of uncertainty, trigger-related fear, autonomic arousal, or difficulty recovering after overstimulation.

The results are also compatible with evidence from conditions in which perceptual sensitivity and symptom monitoring contribute to persistent functional impairment. Persistent postural-perceptual dizziness has been conceptualized as involving an interaction among altered sensory processing, attentional focus, symptom expectations, and behavioral adaptation (Özdemir et al., 2026). Similar integrative models have been applied to functional ear symptoms, in which symptom experience is understood through the combined influence of comorbidity, bodily attention, and experiential factors (Scholfield et al., 2022). Research on persistent post-concussion symptoms and functional neurological disorder has likewise emphasized overlapping mechanisms such as heightened symptom attention, expectation, cognitive load, and maladaptive adaptation (Mavroudis et al., 2025; Mollica et al., 2025). These findings do not imply that migraine disability is functional or psychogenic. Rather, they demonstrate that neurological symptoms and attentional processes can interact without invalidating the biological basis of the condition. In migraine, body vigilance may amplify awareness and behavioral consequences of genuine neurological sensations, thereby increasing disability beyond that attributable to attack frequency alone.

The relationship among bodily monitoring, arousal, and sleep can also be interpreted within broader models of

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systemic dysregulation. Research linking trauma-related processes with postural tachycardia syndrome has demonstrated that bodily and brain-based mechanisms may interact through sustained vigilance, autonomic activation, and interpretation of internal states (Crouch et al., 2026). Neurobiological and neuropsychological disturbances reported in Ehlers–Danlos syndromes similarly illustrate the coexistence of sensory, autonomic, cognitive, and pain-related symptoms (Westerman et al., 2025). Theoretical models of irritable bowel syndrome have also proposed that bodily sensation and perceptual interpretation contribute to symptom persistence (Spiegel, 2022). These bodies of research reinforce the view that attention to internal sensations is neither exclusively psychological nor exclusively physiological. Instead, body vigilance may operate at the interface between nervous-system reactivity, learned expectations, environmental demands, and behavioral responses.

The robustness analyses strengthened confidence in the primary interpretation. The hypothesized model showed good fit in the full sample, among participants who completed all four waves, and among those whose preventive medication regimen remained stable. The serial indirect effect also remained significant when monthly migraine days were modeled as a time-varying covariate. Thus, the mediation pattern was not explained solely by changes in headache frequency or preventive treatment. The competing model in which sleep disturbance preceded body vigilance fit the data less well and did not yield a significant serial indirect pathway. This result supports, although does not definitively prove, the proposed temporal order. Body vigilance may be especially likely to precede sleep disruption because sustained bodily monitoring can maintain pre-sleep arousal and interfere with disengagement from symptom-related information. Nevertheless, reciprocal effects remain plausible, as poor sleep may also increase interoceptive sensitivity and threat monitoring on the following day.

The absence of significant differences between episodic and chronic migraine groups suggests that the proposed mechanism may operate across levels of migraine frequency. Chronic migraine was associated with greater disability, but the pathways linking sensory processing sensitivity, body vigilance, sleep disturbance, and disability were statistically similar across migraine subtypes. This finding implies that sensory sensitivity and attentional processes may influence functional burden even among individuals who do not meet criteria for chronic migraine. It also supports a dimensional interpretation in which vulnerability is distributed across the migraine population rather than restricted to those with the most frequent attacks. Developmental and hormonal perspectives on migraine further indicate that symptom expression is heterogeneous and shaped by multiple interacting influences (Al-Beltagi, 2026; Korn & Bernstein, 2026). Sensory processing sensitivity may represent one source of such heterogeneity by helping to explain why individuals with comparable migraine frequency experience markedly different levels of interference.

The study further contributes to research on the interaction between sleep, stress, and persistent arousal. Sleep disturbance in populations exposed to war and severe trauma has been linked to sustained neurobiological and psychological activation (Hamamra & Mahamid, 2026), and studies of genocide survivors have emphasized the enduring neuropsychological consequences of disrupted sleep (Kizilhan et al., 2026). Adverse childhood experiences have also been associated with later depression through pathways involving insomnia and family functioning (Tao et al., 2023). Although the present study did not focus on trauma, these findings demonstrate that sleep is a central regulatory process affected by persistent vigilance and stress-related activation. Occupational studies concerning prolonged clinical shifts similarly show that environmental demands and restricted recovery opportunities can influence sleep and functioning (Meshkin et al., 2025). In individuals with migraine, heightened sensitivity and body vigilance may constitute an internally generated source of sustained arousal that operates alongside external stressors.

The findings have implications for understanding disability as a multidimensional outcome rather than a direct reflection of symptom frequency. Migraine-related disability includes lost productivity, missed responsibilities, impaired domestic functioning, and withdrawal from social or recreational activities. These outcomes are shaped by the meanings individuals assign to symptoms, the extent to which they monitor and anticipate attacks, the restorative quality of sleep, and the resources available for adapting to recurrent illness. Qualitative research on families living with persistent behavioral and health-related demands has shown that burden is produced through ongoing vigilance, adaptation, and disruption of daily routines rather than by symptom frequency alone (Pearson et al., 2026). Similarly, multidisciplinary guidance for persistent neurological sequelae emphasizes the need to assess cognitive, emotional, physical, and functional dimensions together (Melamed et al., 2023). The current findings support applying this broader

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perspective to migraine by showing that sensory sensitivity, bodily attention, and sleep contribute independently and sequentially to disability.

The study should be interpreted in light of several limitations. First, the principal variables were assessed using self-report instruments, creating the possibility of shared-method variance and response bias. Second, although the four-wave design improved temporal ordering, it did not establish causality, and reciprocal relationships among body vigilance, sleep disturbance, and disability may still exist. Third, migraine diagnosis was verified through structured screening and prior physician diagnosis rather than an in-person neurological examination conducted by the research team. Fourth, the sample included a high proportion of women and participants with postsecondary education, which may limit generalizability to men, individuals with lower educational attainment, and populations with reduced access to healthcare or digital research platforms. Fifth, sleep disturbance was measured subjectively and was not supplemented by actigraphy or polysomnography. Sixth, sensory processing sensitivity was treated as a global trait, although specific components such as ease of excitation, low sensory threshold, and aesthetic sensitivity may have different relationships with migraine. Finally, despite statistical control of migraine frequency, medication use, and earlier symptom levels, unmeasured factors such as anxiety, depression, trauma exposure, pain catastrophizing, menopausal status, caffeine consumption, occupational schedule, or comorbid pain conditions may have influenced the observed pathways.

Future research should replicate the model in larger and more diverse samples, including men, adolescents, older adults, culturally heterogeneous populations, and individuals with limited access to specialist migraine care. Studies with more frequent assessments could examine whether daily changes in sensory overload and body vigilance predict same-night sleep disruption and next-day migraine burden. Ecological momentary assessment, electronic headache diaries, actigraphy, and physiological indicators of autonomic arousal would reduce reliance on retrospective self-report. Future studies should also test reciprocal cross-lagged models to determine whether sleep disturbance increases later body vigilance as well as being influenced by it. Examination of the separate dimensions of sensory processing sensitivity may identify which aspects are most strongly related to migraine-related impairment. Additional mediators, including pain catastrophizing, avoidance, anxiety sensitivity, emotional dysregulation, perceived stress, and intolerance of uncertainty, should be evaluated alongside body vigilance and sleep. Intervention studies are also needed to determine whether reducing maladaptive bodily monitoring or improving sleep weakens the association between sensory processing sensitivity and disability.

In clinical practice, assessment of adults with migraine should extend beyond headache frequency and pain intensity to include sensory sensitivity, attention to bodily sensations, sleep quality, and the functional consequences of anticipatory monitoring. Clinicians can help patients distinguish flexible awareness of meaningful prodromal signs from persistent scanning that increases arousal and restricts activity. Psychoeducation should validate sensory sensitivity as an individual difference while explaining how excessive monitoring may intensify distress and interfere with sleep. Cognitive-behavioral strategies, attentional flexibility training, relaxation, stimulus control, sleep scheduling, and behavioral treatment for insomnia may be especially useful for highly sensitive patients. Environmental modifications involving light, sound, temperature, and workload should be individualized rather than used as broad avoidance strategies that reinforce fear of stimulation. Neurologists, primary care clinicians, psychologists, and sleep specialists should coordinate treatment when migraine disability co-occurs with persistent hypervigilance and sleep disruption. Such an integrated approach may reduce functional impairment by addressing not only migraine attacks but also the attentional and sleep-related processes through which sensory sensitivity contributes to everyday disability.

Authors' Contributions

Authors contributed equally to this article.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

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Declaration of Interest

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Ethics Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants.

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