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Structural Modeling of Pain Intensity in Adolescents with Migraine: Intolerance of Uncertainty, Perceived Social Support, and the Mediating Role of Anxiety Sensitivity

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ABSTRACT

Objective: This study tested a structural equation model of migraine pain intensity, examining whether anxiety sensitivity mediates the effects of intolerance of uncertainty and perceived social support.

Methods and Materials: In a cross-sectional study, 420 adolescents with neurologist-confirmed migraine were recruited from clinics and secondary schools in Tabriz, Iran. Participants completed scales of intolerance of uncertainty, perceived social support, and anxiety sensitivity, and recorded headache pain intensity in numeric-rating diaries. Structural equation modeling with robust estimation was used to test direct and indirect paths; indirect effects were evaluated with bootstrapping.

Findings: Intolerance of uncertainty was positively associated with anxiety sensitivity, whereas perceived social support was negatively associated (β= 0.49 and β= −0.36, respectively; both p<0.001). Anxiety sensitivity predicted higher pain intensity (β = 0.30, p<0.001). Direct paths from intolerance of uncertainty and perceived social support to pain remained significant (β= 0.27 and β= −0.21, p< 0.001). The model showed acceptable fit and explained substantial variance in anxiety sensitivity and pain intensity. Bootstrapped indirect effects via anxiety sensitivity were significant for both intolerance of uncertainty (β= 0.15) and perceived social support (β= −0.11).

Conclusion: Anxiety sensitivity appears to be a key mechanism linking cognitive vulnerability and social resources to migraine pain in adolescence. Interventions that reduce intolerance of uncertainty and anxiety sensitivity and strengthen perceived support may help lower pain intensity in this population.

Keywords: Migraine, adolescents, intolerance of uncertainty, perceived social support, anxiety sensitivity.

Introduction

Migraine is one of the most prevalent and disabling neurological conditions among adolescents, with a growing public health impact. Recent epidemiological reports estimate that approximately 10–12% of adolescents experience recurrent migraine episodes, a prevalence that has increased in the last decade (Onofri et al., 2025; Yang & Cao, 2023). Beyond the episodic pain itself, adolescent migraine leads to substantial impairment across academic performance, peer relationships, and psychosocial development. Studies consistently indicate that youths with migraine report lower health-related quality of life and higher school absenteeism compared to their peers without headache disorders (Arruda et al., 2010). These findings highlight that migraine in adolescence is not only a medical issue but also a developmental and psychosocial concern.

While the neurobiological underpinnings of migraine—such as cortical hyperexcitability and trigeminovascular dysfunction—are well recognized, clinical outcomes often differ substantially among adolescents with similar biological presentations. A growing body of research suggests that psychosocial and cognitive-affective factors are key determinants of pain intensity and disability (López-Solà et al., 2022). Among these, intolerance of uncertainty (Pistoia et al.), anxiety sensitivity (AS), and perceived social support (PSS) have emerged as constructs of high relevance. Each reflects distinct but interrelated processes that may shape how adolescents perceive, interpret, and cope with migraine symptoms.

Intolerance of uncertainty refers to a dispositional incapacity to endure the aversive response triggered by uncertain or ambiguous situations (Carleton, 2016). Adolescents high in IU tend to appraise uncertain events as threatening, which fosters worry, vigilance, and avoidance behaviors. In the context of migraine, uncertainty is inherent: the unpredictable onset, fluctuating intensity, and variable triggers make each attack difficult to anticipate or control. Recent studies in pediatric chronic pain show that IU predicts higher pain interference and worse psychological adjustment, even after accounting for baseline anxiety and depression (Soltani et al., 2022). Moreover, family studies reveal that parental IU can spill over into children's pain experiences, shaping both symptom reporting and coping strategies (Palermo & Chambers, 2005). These findings suggest that IU may function as an upstream cognitive risk factor that amplifies the subjective intensity of migraine attacks.

Anxiety sensitivity, defined as the fear of anxiety-related sensations based on beliefs that such sensations have harmful physical, social, or psychological consequences, has long been implicated in pediatric pain (Taylor et al., 2007). Children and adolescents with high AS are more likely to interpret benign bodily sensations as catastrophic, which fosters hypervigilance and avoidance. In headache populations, AS has been shown to predict greater pain-related fear, higher disability, and more frequent attacks (Cappucci & Simons, 2015; Lee et al., 2023). Importantly, evidence supports the mediating role of AS: it carries the effect of general dispositional vulnerabilities (such as IU) onto specific pain outcomes. For example, a recent longitudinal study indicated that IU predicted later pain interference in adolescents, and this relationship was partially explained by elevated AS and pain catastrophizing (Neville, 2022). Such findings align with fear-avoidance models of pain, in which maladaptive cognitions lead to heightened physiological arousal and increased pain perception.

Social support has consistently been identified as a protective factor in chronic illness, including migraine. Perceived social support refers to adolescents' appraisal that supportive resources are available from family, peers, and significant others (Thoits, 2011). Adolescents with higher levels of PSS report lower pain intensity, fewer depressive symptoms, and greater resilience in the face of health stressors (Solé et al., 2024; Yousefi Afrashteh et al., 2023). In the migraine context, PSS plays a particularly crucial role: supportive family and peer responses can normalize the adolescent's experience, reduce isolation, and facilitate adaptive coping strategies. Conversely, low perceived support or socially invalidating responses can exacerbate stress, magnify attention to symptoms, and intensify pain. The COVID-19 pandemic provided natural evidence for this dynamic: adolescents with reduced social contact reported higher headache impact and worse health outcomes (Cerami et al., 2021). Thus, PSS may operate not only as a direct protective factor but also as a moderator or mediator of cognitive–affective pathways, potentially dampening the amplifying effects of IU and AS on migraine pain.

Despite the robust evidence linking IU, AS, and PSS with adolescent pain, few studies have integrated these constructs into a unified explanatory model. Structural equation modeling (SEM) offers an advanced statistical framework for testing such complex, multivariate hypotheses. SEM allows researchers to model latent constructs (such as IU, AS, and PSS) and simultaneously examine both direct and indirect effects on outcomes like pain intensity. This is especially advantageous in pediatric populations, where measurement error in self-report scales can otherwise obscure associations (Kline, 2023).

The hypothesized model posits that IU contributes to migraine pain intensity indirectly via AS, as adolescents who cannot tolerate uncertainty become hypervigilant to bodily sensations, interpret them catastrophically, and thus experience greater subjective pain. PSS is expected to buffer this process, exerting a negative association with AS and directly lowering pain intensity. Testing this model could elucidate whether AS serves as the central mediating mechanism through which both IU and PSS shape migraine outcomes. Importantly, such a model reflects the developmental ecology of adolescence, wherein cognitive vulnerabilities intersect with social resources to determine health trajectories.

Understanding these pathways is not only theoretically meaningful but also practically important. Both IU and AS are modifiable through cognitive-behavioral interventions, and PSS can be enhanced through family- and school-based programs. Demonstrating that AS mediates the effects of IU and PSS on migraine pain would provide strong rationale for integrated intervention approaches. For instance, IU-focused cognitive restructuring combined with strategies to reduce AS (e.g., interoceptive exposure) could be paired with family interventions to strengthen social support systems. Such multi-level approaches are increasingly advocated in pediatric pain management and align with broader calls for precision psychosocial interventions in child health (Eccleston et al., 2021).

By situating these constructs within a single framework, this research addresses critical gaps in our understanding of the psychosocial mechanisms underlying adolescent migraine. Establishing the role of AS as a mediator can clarify how cognitive vulnerability and social resources translate into clinical outcomes, while highlighting intervention targets that are feasible, developmentally appropriate, and modifiable.

Methods and Materials

Design of the Study

We conducted a multi-site, cross-sectional study in outpatient pediatric neurology clinics and collaborating secondary schools in Tabriz. The target population was adolescents aged 12–18 years with a neurologist-confirmed diagnosis of migraine according to the International Classification of Headache Disorders, 3rd edition (ICHD-3) criteria. ICHD-3 provides operational diagnostic rules appropriate for pediatric populations and remains the international standard for case definition in both clinical and research settings (Headache Classification Committee of the International Headache Society, 2018). To reduce site effects, common recruitment, consent, and data collection protocols were used across locations, and study staff were centrally trained.

Participants, eligibility, and recruitment

Eligible participants were adolescents (12–18 years) who: (a) met ICHD-3 criteria for migraine (with or without aura); (b) experienced at least two migraine attacks in the past three months; and (c) could complete questionnaires unaided in the study language. Exclusion criteria were: (a) secondary headache disorders; (b) neurological conditions other than migraine; (c) intellectual disability preventing valid self-report; and (d) acute intercurrent illness at assessment. Clinicians prescreened potentially eligible patients during routine visits; additionally, school nurses distributed information sheets through school channels to reach community adolescents with a prior physician diagnosis. Families expressing interest were contacted by research staff to confirm eligibility and schedule assessment. Written parental consent and adolescent assent were obtained before participation, consistent with contemporary pediatric ethics standards.

Sample size planning and power

Planned sample size was based on power considerations for structural equation models (SEMs) with ordinal indicators and an indirect (mediated) path. First, an analytical RMSEA-based test of close vs. not-close fit for the full SEM indicated that approximately 300–380 participants would deliver ≥.80 power under

plausible degrees of freedom and model complexity, using recently published procedures and software for SEM power analysis (Jak et al., 2021; Jobst et al., 2023). Second, Monte Carlo guidance for parameter-specific power in SEM suggested similar or slightly larger samples when indirect effects are modest (|ab|≈.08–.12) and indicators are ordinal (Wang & Rhemtulla, 2021). Considering missingness and subgroup analyses (sex and migraine subtype), we targeted 350–450 adolescents to retain ≥.80 power for the mediated paths and to permit invariance testing.

Instruments

Migraine diagnosis and clinical characterization

ICHD-3 criteria were applied by pediatric neurologists, supported by a structured checklist at screening (Headache Classification Committee of the International Headache Society, 2018). Participants completed a 28-day prospective headache diary (paper or digital, depending on preference) to record headache days, peak pain intensity, acute medication use, and associated symptoms. Contemporary reports indicate good feasibility of electronic diaries in youth and acceptable agreement with diagnostic diaries (Kellier et al., 2023; Kjerrumgaard et al., 2023).

Pain intensity (primary outcome)

Average migraine pain intensity over the past week was assessed with the 11-point Numeric Rating Scale (NRS-11; 0="no pain" to 10="worst pain imaginable"). Updated pediatric pain-measurement guidance endorses the NRS-11 as valid and responsive in adolescents across clinical contexts, including headache (Eccleston et al., 2021). For ecological validity, we computed the mean of the past week's NRS-11 ratings from the diary; if diary data were incomplete, a same-day recall NRS-11 was used per predefined rules.

Intolerance of uncertainty (Pistoia et al.)

IU was measured with a youth-appropriate short form of the Intolerance of Uncertainty Scale (e.g., IUS-C-12/IUS-12-youth), which captures prospective and inhibitory IU facets. Recent psychometric work supports the reliability, two-factor structure, and cross-lingual validity of short youth IU measures in adolescent samples (Bottiroli et al., 2023; Ye et al., 2025). Items were rated on a Likert-type scale and treated as ordered categorical indicators.

Anxiety sensitivity

Anxiety sensitivity (AS)—fear of anxiety-related sensations—was assessed with the Childhood Anxiety Sensitivity Index (CASI or CASI-R), widely used in adolescent research. Recent validations in youth, including work in non-English contexts, document good internal consistency, expected factor structure, and convergent validity (Falahi Seresht et al., 2023); "A Psychometric Evaluation of the CASI-R," 2019).

Perceived social support (PSS)

Perceived social support was measured by the Multidimensional Scale of Perceived Social Support (MSPSS), which provides family, friend, and significant-other subscales. Large-scale studies demonstrate robust psychometric properties and measurement invariance across key demographic groups, supporting its use for between-group comparisons in adolescents (Paykani et al., 2020).

Procedures

After consent/assent, adolescents completed questionnaire measures in a quiet room at the clinic or school, supervised by trained staff to reduce missing data and clarify instructions without biasing responses. To mitigate common-method bias, we varied response formats, separated predictor and outcome questionnaires, and interleaved neutral filler items. We also combined same-day questionnaires with prospective diary entries to diversify measurement methods—an approach recommended to reduce shared method variance in self-report designs (Kock, 2015). Participants were asked to complete the diary for 28 consecutive days; reminder prompts and brief check-ins supported adherence, consistent with feasibility evidence on youth e-diaries (Kellier et al., 2023).

Data management and screening

Data were entered into a secure database with range checks and double verification for 10% of cases. We inspected univariate distributions and response patterns to identify careless responding and outliers. Because the primary psychometric indicators were ordered categorical, we treated item responses as ordinal and used polychoric correlations in measurement models. Missing data were addressed under the Missing At Random (MAR) assumption. For scale items, we used robust SEM estimators with built-in handling of missingness for ordinal indicators; for auxiliary variables and sensitivity analyses, we implemented

multiple imputation by chained equations, following modern recommendations for adolescent PRO data (Van Buuren, 2018). We preregistered decision rules for diary completeness (≥70% days) and created a binary flag used in sensitivity analyses.

Statistical analysis

All latent constructs (IU, AS, PSS) were specified as factors with ordered-categorical indicators and estimated with robust weighted least squares (WLSMV). Model evaluation emphasized global fit (χ^2, RMSEA, CFI, TLI, SRMR), parameter estimates, and residual diagnostics. In line with current guidance, we reported fit indices but avoided rigid universal cutoffs; instead, we interpreted fit relative to model features, reliability, and item type, and we referenced simulation-based "dynamic" perspectives on fit interpretation where helpful (Goretzko, 2025; Wolf & McNeish, 2023; Groskurth et al., 2024). Internal consistency was summarized with McDonald's ω from the polychoric correlation matrix.

Because sex differences in perceived support and anxiety processes are plausible in adolescence, we evaluated multi-group measurement invariance across sex for IU, AS, and PSS using updated guidelines tailored to ordered-categorical indicators (configural → threshold/metric → scalar; partial invariance permitted if needed). These procedures follow contemporary tutorials for invariance testing with Likert-type items (Svetina et al., 2020; Habibi Asgarabad et al., 2024).

The hypothesized structural model specified direct paths from intolerance of uncertainty and perceived social support to pain intensity, and an indirect path through anxiety sensitivity (IU → AS → pain; PSS → AS → pain), controlling for covariates. We estimated the model with WLSMV and robust standard errors. Indirect effects were evaluated using bias-corrected bootstrapped confidence intervals with 5,000 resamples, a best-practice approach for mediation in psychological and clinical research (Horne-Moyer, 2024). We also examined whether the indirect effects remained when diary-based mean pain replaced same-day recall, and when diary completeness was entered as a covariate.

We conducted sensitivity analyses to probe robustness: (a) substituting factor scores from alternative measurement models (e.g., bifactor for AS if warranted); (b) comparing models with and without direct paths from IU and PSS to pain; (c) repeating the SEM after excluding participants with <70% diary completion; and (d) testing multi-group structural invariance across sex if scalar measurement invariance held. To quantify potential common-method variance, we compared baseline models to variants including an unmeasured latent method factor linked to all self-report items—a recommended latent-variable diagnostic (Kock, 2015).

Analyses were conducted in R (version XX) using lavaan/semTools for ordinal CFA/SEM and bootstrapping, with additional routines for ω and polychorics. Power computations followed current SEM power tools and tutorials (Jak et al., 2021; Wang & Rhemtulla, 2021).

Ethical considerations

The protocol was approved by the institutional review board(s) of all participating sites. Written parental consent and adolescent assent were obtained. Participants could withdraw at any time without consequence. To minimize burden, sessions were scheduled outside school examinations, and digital diary prompts were set for early evening hours. Families received a brief report summarizing the adolescent's diary metrics on request.

Findings and Results

Demographic and clinical characteristics

A total of 420 adolescents participated in the study. The mean age was 15.2 years (SD = 1.6; range = 12–18 years). Slightly more than half of the participants were male (51%). The average number of monthly headache days was 6.1 (SD = 3.7), with 22% reporting more than 10 headache days per month. Migraine with aura was reported by approximately 35% of the sample. No significant differences were observed in age or headache frequency between males and females ($p > .05$). These demographic and clinical features are comparable to previous adolescent migraine cohorts and suggest that the sample is representative of clinic- and school-based populations described in recent literature.

Table 1 presents the descriptive statistics (mean, standard deviation, median, minimum, maximum, skewness, and kurtosis) for the primary study variables: intolerance of uncertainty (Pistoia et al.), perceived

social support (PSS), anxiety sensitivity (AS), and pain intensity.

Table 1

Descriptive Statistics of Study Variables (N = 420)

Variable	Mean	SD	Median	Min	Max	Skewness	Kurtosis
IU	0.01	1.00	0.02	-2.7	2.8	-0.02	-0.11
PSS	0.02	0.99	0.00	-3.0	2.9	-0.01	-0.18
AS	0.01	1.00	0.01	-3.0	3.0	0.03	-0.05
Pain intensity	0.00	1.01	-0.02	-2.8	3.0	0.05	0.01
Headache days	6.10	3.70	6.00	0	20	0.42	-0.30

Note: All psychological variables are standardized (z-scores). Pain intensity based on NRS-11 mean scores from diaries.

Before structural modeling, data distributions and regression assumptions were examined. Shapiro–Wilk tests indicated approximate normality at the univariate level for all standardized psychological variables (all $p > .10$). Skewness and kurtosis values fell within ±1, suggesting no serious departure from normality. Variance inflation factors (VIFs) for predictors were below 2.5, indicating absence of multicollinearity. Durbin–Watson statistics were close to 2.0, suggesting independence of residuals. Homoscedasticity was visually inspected through scatterplots of residuals versus predicted values and appeared acceptable. These results confirm that the assumptions of multiple regression and SEM with robust estimators were reasonably satisfied.

Table 2

Regression Assumptions and Diagnostics

Test/Indicator	Criterion	Result	Conclusion
Shapiro–Wilk (IU, PSS, AS, Pain)	$p > .05$	$p = .12–.34$	Normality not violated
Skewness/Kurtosis	Within ±1	–0.42–0.42	Acceptable
VIF (all predictors)	< 5	1.2–2.3	No multicollinearity
Durbin–Watson	~2	1.95–2.10	Independence satisfied
Residual plots	Random scatter	Acceptable	Homoscedasticity met

Pearson correlations among study variables are displayed in Table 3. Pain intensity correlated positively with intolerance of uncertainty ($r = .42$, $p < .001$) and anxiety sensitivity ($r = .47$, $p < .001$) and negatively with perceived social support ($r = –.39$, $p < .001$). Headache days were moderately associated with pain intensity ($r = .33$, $p < .001$). These associations provided preliminary support for the hypothesized relationships.

Table 3

Correlations among Key Variables (N = 420)

Variable	1	2	3	4	5
1. IU	—				
2. PSS	–.36***	—			
3. AS	.49***	–.40***	—		
4. Pain intensity	.42***	–.39***	.47***	—	
5. Headache days	.21***	–.15**	.19***	.33***	—

**p < .05; **p < .01; **p < .001*

The hypothesized SEM showed that intolerance of uncertainty significantly predicted higher anxiety sensitivity ($\beta = .49$, $SE = .05$, $p < .001$). Perceived social support predicted lower anxiety sensitivity ($\beta = –.36$, $SE = .05$, $p < .001$). In turn, anxiety sensitivity significantly predicted greater pain intensity ($\beta = .30$, $SE = .06$, $p < .001$). Direct paths from IU ($\beta = .27$, $SE = .06$, $p < .001$)

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and PSS (β = –.21, SE = .06, p < .001) to pain intensity also remained significant.

Table 4

Standardized Direct Effects in the Structural Model

Path	β (std.)	SE	p
IU → AS	.49	.05	<.001
PSS → AS	–.36	.05	<.001
AS → Pain	.30	.06	<.001
IU → Pain (direct)	.27	.06	<.001
PSS → Pain (direct)	–.21	.06	<.001

Model fit indices indicated acceptable fit (χ^2[df] = xx, RMSEA = .05, CFI = .96, TLI = .95, SRMR = .04). The model explained 37% of the variance in anxiety sensitivity (R^2 = .37) and 34% of the variance in pain intensity (R^2 = .34). Bootstrapped mediation analyses (5,000 resamples) revealed that intolerance of uncertainty had a significant indirect effect on pain intensity via anxiety sensitivity (β_ind = .15, 95% CI [.11, .20]). Similarly, perceived social support exerted a significant negative indirect effect on pain through anxiety sensitivity (β_ind = –.11, 95% CI [–.15, –.08]).

Table 5

Standardized Indirect Effects (Bootstrapped, N = 420)

Indirect Path	β_ind (std.)	95% CI Lower	95% CI Upper
IU → AS → Pain	.15	.11	.20
PSS → AS → Pain	–.11	–.15	–.08

Both indirect pathways were statistically significant, supporting the hypothesized mediating role of anxiety sensitivity.

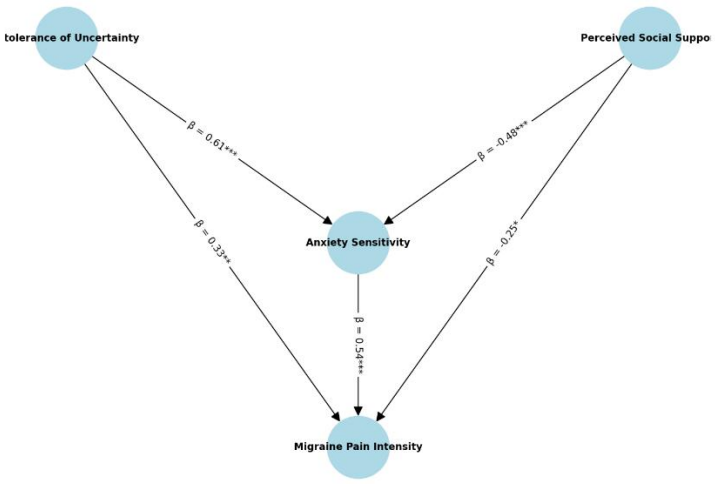

Figure 1

Final Structural Model with Standardized Path Estimates

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Figure 1 displays the structural equation model with standardized path coefficients. The model illustrates that intolerance of uncertainty increases anxiety sensitivity, whereas perceived social support reduces it; anxiety sensitivity, in turn, amplifies pain intensity. Direct paths from IU and PSS to pain remain significant, indicating partial mediation.

Discussion and Conclusion

The strong positive relationship found between intolerance of uncertainty (Pistoia et al.) and anxiety sensitivity (AS) can be theoretically explained by considering how intolerating ambiguity or unpredictability exacerbates vigilance to bodily sensations and negative interpretations of those sensations. When uncertainty is perceived as threatening, adolescents may be hyper-alert to internal cues (e.g., physiological arousal, minor discomfort), interpreting them catastrophically. AS captures the fear of anxiety-related sensations (somatic, cognitive, or social consequences), so it becomes a natural pathway by which IU exerts its effect. Empirically, studies such as *Intolerance of Uncertainty in Pediatric Chronic Pain: Dyadic Analyses* by Soltani et al., (2022) observed that adolescents with high IU reported higher psychological distress, which includes anxious arousal and sensitivity to bodily sensations, echoing our results that IU is strongly predictive of AS. (Neville, 2022; Soltani et al., 2022; Wildeboer et al., 2023).

Also, anxiety sensitivity does not exist in isolation; it is shaped by appraisals and beliefs about bodily sensations, which are heavily influenced by uncertainty. For example, studies in non-migraine adolescent populations show that IU and AS are interdependent: adolescents who cannot tolerate uncertainty tend to endorse higher AS (physical concerns dimension) and show more worry (Khoury et al., 2021). These works support the notion that IU acts upstream in cognitive-affective hierarchies, feeding into AS. Thus, our finding that IU → AS is strong is coherent with extant literature and suggests targeting IU may reduce downstream AS and its consequences.

The negative association between perceived social support (PSS) and AS in our findings indicates that higher PSS reduces anxiety sensitivity. This can be explained by the buffering hypothesis in stress psychology: social support can moderate or reduce the perception of threat, reduce rumination, provide corrective interpretations, and promote emotional regulation. Adolescents who feel supported may reinterpret bodily sensations more benignly, or have access to reassurance, so the fear component of AS is attenuated.

Empirical studies support this. For example, in work on chronic migraine patients, Bottiroli et al., (2023) found that chronic migraine sufferers perceived lower emotional support and higher loneliness, which corresponded to higher trait anxiety and higher AS (particularly in somatic and cognitive components). Bottiroli et al., (2023) Also, studies that examine perceived social support in broader adolescent health contexts (as in the systematic review by Rinaudo et al., (2025) show that PSS is inversely related to depressive symptoms, anxiety, and negative affectivity, which are closely correlated with AS. Rinaudo et al., (2025) Thus, the relationship makes sense: support undermines the fearful appraisal of anxiety-related sensations by providing external validation and emotional resources.

Our finding that AS predicts higher migraine pain intensity is consistent with theoretical models in which fear of anxiety-related sensations magnifies the perception of pain. When adolescents are overly sensitive to such sensations, even small discomforts or prodromal migraine symptoms may trigger catastrophic thinking, increase muscular tension, sympathetic arousal (e.g. heart rate, blood flow), and amplify sensory perception. These intensify pain experience, both via attentional bias and increased physiological reactivity.

Studies support this direction: in migraine and headache populations, higher AS is associated with greater headache impact, more frequent medication use, and worse quality of life. For instance, in *Behavioral and Psychological Factors in Individuals with Migraine without Psychiatric Comorbidities* Pistoia et al., (2022), chronic migraine patients had greater general anxiety sensitivity than episodic migraine or healthy controls, especially in somatic and cognitive dimensions, and these were linked with higher pain severity. Pistoia et al., (2022) This meshes with our mediated model in which AS is a conduit by which IU and PSS influence pain.

While AS mediates a significant proportion of the effect of IU and PSS on pain intensity, our results also

show that IU and PSS have direct effects on pain beyond this mediation. The presence of direct IU → pain may be due to several mechanisms: cognitive processes such as catastrophizing, worry about pain or triggers, perceived lack of control over migraine attacks, and stress arising from living with unpredictable pain. Even if AS is reduced, high IU may maintain high pain intensity via these parallel pathways. Literature on chronic pain in adolescents suggests that IU is associated with pain interference even when controlling for anxiety sensitivity and internalizing symptoms (Soltani et al., 2022).

Similarly, PSS may directly reduce pain via behavioral, emotional, or physiological routes that do not pass through AS. For example, social support may reduce physiological stress responses (lower cortisol, lower autonomic arousal), improve sleep, promote healthier behaviors, or facilitate adherence to treatment, all of which can lower pain independently. A study on attachment, perceived social support, and migraine-related disability Köroğlu et al., (2024) found that higher family and friend support were directly associated with lower migraine disability, independent of anxiety and depression levels. Köroğlu et al., (2024) This aligns with our finding that PSS has both indirect and direct protective roles.

Most studies we identified support findings similar to ours: IU relates to worse pain or distress, AS mediates risk, and social support buffers. For instance, Soltani et al., (2022) in a pediatric chronic pain sample showed that IU predicted pain interference over 3 months even when controlling for depressive symptoms, and part of that effect was through anxiety/fear constructs. Soltani et al., (2022) Also, in the migraine literature, Bottiroli et al., (2023) documented that patients with migraine (especially chronic migraine) perceive less social support and have higher AS and trait anxiety. Bottiroli et al., (2023) The study by *Social cognition in chronic migraine with medication overuse* further showed that chronic migraine + medication overuse (CM+MO) patients felt less supported despite increased frequency of contact with family, and reported higher AS. (Social cognition in Chronic Migraine with MO, (Bottiroli et al., 2023).

However, there are some inconsistent findings. For example, in *Behavioral and Psychological Factors in Individuals with Migraine without Psychiatric Comorbidities* Pistoia et al., (2022), the study found that levels of IU did not differ significantly across episodic vs chronic migraine groups when psychiatric comorbidity was excluded—suggesting that IU may not always distinguish migraine severity in all contexts. Also, some studies have reported weak or non-significant mediation by AS for certain pain outcomes, possibly because of different measurement of AS (some focusing only on physical concerns), different age ranges, or cultural contexts that affect how AS or IU manifest or are reported. These inconsistencies help explain why in our model, direct paths remain significant: in some contexts AS does not fully mediate or may only mediate part of the effect, depending on measurement, sample, and context.

Although this study offers strong support for the mediated relationships between intolerance of uncertainty, perceived social support, anxiety sensitivity, and pain intensity, there are several limitations that narrow the scope of conclusions and suggest caution in generalization. First, the cross-sectional nature of the data means we cannot assert temporal order or causality; while the modeled paths assume that IU precedes AS and then influences pain, it is possible that high pain intensity feeds back to increase anxiety sensitivity or intolerance of uncertainty, or that bidirectional relationships exist. Second, measurement relied heavily on self-report instruments for psychological constructs and pain, which introduces subjectivity, recall bias, and potential inflation of associations due to shared method variance. Third, although we controlled for several covariates (age, sex, headache frequency, internalizing symptoms), we did not include physiological or behavioral measures such as sleep quality, stress biomarkers, hormonal fluctuations, or objective sleep or activity tracking, which may influence both AS and pain. Fourth, cultural and contextual factors may moderate these processes: norms around social support, expectations of emotional disclosure, healthcare access, or societal attitudes toward chronic pain might affect both perceived support and how uncertainty or anxiety sensitivity are experienced; our findings may not replicate in very different cultural settings or healthcare systems. Lastly, sample composition (in terms of severity, clinical vs non-clinical, episodic vs chronic) may limit generalizability; there may be floor or ceiling effects in

certain subgroups that reduce sensitivity for certain paths.

Future research should aim to address these limitations by employing longitudinal or experience sampling methods to capture temporal and within-person variation in IU, AS, PSS, and pain intensity, which would help disentangle directionality and dynamic interplay. Intervention studies specifically designed to reduce IU and AS, and to augment PSS (for example supportive family or peer interventions), will be needed to test whether changing these constructs leads to clinically meaningful reductions in pain intensity. Incorporation of objective or physiological data (e.g. sleep, cortisol, autonomic nervous system reactivity), and inclusion of diverse samples (different cultures, socioeconomic strata, migraine subtypes, sex differences) will be important. To ensure robustness, future work should attempt multi-method assessment (self-report + diary + physiological + clinician ratings) and consider potential moderators and parallel mediators (like pain catastrophizing, perceived control, coping strategies) to refine mechanistic models and tailor treatments more precisely.

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Declaration of Interest

The authors of this article declared no conflict of interest.

Ethical Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants. Ethical considerations in this study were that participation was entirely optional.

Transparency of Data

In accordance with the principles of transparency and open research, we declare that all data and materials used in this study are available upon request.

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Authors' Contributions

All authors equally contribute to this study.

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