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Pain Catastrophizing as a Mediator between Anxiety Sensitivity and Somatic Symptoms

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A B S T R A C T

This study aimed to examine whether pain catastrophizing mediates the relationship between anxiety sensitivity and somatic symptoms in a community sample of Spanish adults. A descriptive correlational design was employed using self-report measures, including the Anxiety Sensitivity Index-3 (ASI-3), the Pain Catastrophizing Scale (PCS), and the Patient Health Questionnaire-15 (PHQ-15). A total of 422 adults from Spain were selected based on the Morgan and Krejcie sample size table and completed the questionnaires via online administration. Descriptive statistics and Pearson correlation analysis were conducted using SPSS-27, and Structural Equation Modeling (SEM) was performed using AMOS-21 to assess the hypothesized mediation model and model fit. Descriptive analysis revealed moderate levels of anxiety sensitivity (M = 38.47, SD = 8.62), pain catastrophizing (M = 26.38, SD = 10.17), and somatic symptoms (M = 14.92, SD = 5.78). Pearson correlations showed significant positive relationships between anxiety sensitivity and pain catastrophizing ($r = .61$, $p < .001$), anxiety sensitivity and somatic symptoms ($r = .49$, $p < .001$), and pain catastrophizing and somatic symptoms ($r = .57$, $p < .001$). The SEM results indicated an acceptable model fit ($\chi^2/df = 2.11$; CFI = .97; RMSEA = .051), with pain catastrophizing fully mediating the relationship between anxiety sensitivity and somatic symptoms. The indirect effect ($\beta = .28$, $p < .001$) was significant, while the direct effect ($\beta = .12$, $p = .128$) was not. The findings suggest that pain catastrophizing plays a central mediating role in the link between anxiety sensitivity and somatic symptoms. These results underscore the importance of targeting cognitive-affective factors such as catastrophizing in interventions aimed at reducing somatic distress in individuals with high anxiety sensitivity.

Keywords: *Anxiety sensitivity, Pain catastrophizing, Somatic symptoms.*

1. Introduction

Somatic symptoms, often manifested as physical complaints without sufficient medical explanation, present a significant challenge to both diagnostic clarity and therapeutic outcomes in clinical psychology and psychosomatic medicine. These symptoms can include pain, gastrointestinal issues, fatigue, and cardiopulmonary discomfort, which collectively contribute to heightened functional impairment and healthcare utilization (Nelson & Novy, 2021; Rodriguez et al., 2025). In recent years, researchers have increasingly focused on the psychological underpinnings of somatic symptoms, proposing that they may not be entirely rooted in physical pathology but are instead influenced by cognitive and affective processes (Zanotta, 2024). Understanding these psychological factors is vital to devising more effective interventions and improving the well-being of affected individuals.

A substantial body of evidence has identified anxiety sensitivity as a key vulnerability factor in the development and persistence of somatic symptoms. Anxiety sensitivity refers to the fear of anxiety-related sensations due to beliefs that such sensations have harmful physical, psychological, or social consequences (Ogawa et al., 2024). Individuals high in anxiety sensitivity are more likely to misinterpret benign physical symptoms as dangerous, potentially triggering a cascade of physiological arousal and attentional bias that exacerbates their experience of somatic distress (Lima et al., 2023). This mechanism has been observed across various contexts, including dental anxiety (Ogawa et al., 2024), orthodontic treatment pain perception (Lima et al., 2023), and chronic pain disorders (Abadi et al., 2024). The heightened vigilance and hyperawareness associated with anxiety sensitivity serve to amplify the salience of bodily sensations, making it a potent contributor to somatization.

A second cognitive-affective factor, pain catastrophizing, has also emerged as a critical mediator in the pathway linking anxiety sensitivity and somatic symptoms. Pain catastrophizing is defined as an exaggerated negative orientation toward actual or anticipated pain experiences and includes elements of rumination, magnification, and feelings of helplessness (Burri et al., 2018). Individuals who catastrophize tend to focus excessively on their pain, feel overwhelmed by it, and anticipate the worst possible outcomes. Pain catastrophizing has consistently been associated with increased pain intensity, emotional distress, and impaired functioning across diverse populations, including children with chronic pain (Miller et al., 2018) and adolescent girls experiencing menstrual pain (Payne et al., 2015). It also plays a mediating role in the relationship between psychological flexibility and pain-related outcomes in cancer patients and individuals with alexithymia (Abadi et al., 2024; Raiisi et al., 2022).

The literature increasingly supports the conceptualization of pain catastrophizing as a mechanism through which anxiety sensitivity influences somatic symptoms. This mediational role has been supported by studies indicating that individuals with higher anxiety sensitivity are more prone to catastrophize pain, thereby increasing their likelihood of experiencing intense somatic symptoms (Wong et al., 2016). The net suppression effect observed by Wong et al. demonstrated that catastrophic cognition significantly moderated the effect of anxiety sensitivity, suggesting a synergistic interaction that amplifies distress. Similarly, Conti et al. found that pain catastrophizing mediated the association between mindfulness and psychological distress in patients with chronic pain syndrome, reinforcing the idea that maladaptive cognitive styles can potentiate somatic experiences (Conti et al., 2020).

From a neurophysiological perspective, these associations may be partially explained by the role of central sensitization—an increased responsiveness of nociceptive neurons in the central nervous system to normal or subthreshold afferent input. Central sensitization has been shown to mediate the relationship between pain intensity and psychological variables such as catastrophizing and anxiety in individuals with persistent pain conditions (Shigetoh et al., 2019). In this context, the psychological and physiological mechanisms are not mutually exclusive but rather interdependent processes that reinforce each other over time.

The multidimensional nature of somatic symptoms is further complicated by their frequent comorbidity with mood disorders and neurodevelopmental conditions such as autism spectrum disorder (ASD). Larkin's work on somatic symptoms in autism highlights the intersection of sensory sensitivity, emotional dysregulation, and bodily awareness, suggesting that cognitive interpretations of physical symptoms may differ significantly across populations (Larkin, 2022). The broader construct of somatic symptom disorders thus requires an integrative model that accounts for individual differences in cognition, affect, and neurobiological function (Lamberty, 2024).

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The importance of developing such an integrative model is also evident in populations affected by illness-specific stressors. For instance, Tutelman et al. explored the psychological sequelae of childhood cancer survivorship and found that fear of cancer recurrence was closely linked to pain perception and emotional distress, mediated in part by catastrophizing thoughts (Tutelman et al., 2022). These findings underscore the need to consider pain-related cognition not as an isolated phenomenon but as part of a broader psychosocial framework that influences how individuals interpret and respond to bodily signals.

In addition to clinical and neurological perspectives, the empirical study of somatic symptoms benefits from theoretical frameworks such as the biopsychosocial model. Roenneberg et al. emphasized that functional somatic symptoms often arise at the intersection of biological vulnerability, psychological disposition, and social context (Roenneberg et al., 2019). Such models allow for a more nuanced understanding of how pain catastrophizing and anxiety sensitivity operate in tandem to generate and maintain somatic symptomatology. Similarly, Nahman-Averbuch et al. found consistent associations between psychological traits and pain outcomes across multiple experimental paradigms, demonstrating the replicability of these mechanisms in healthy populations (Nahman-Averbuch et al., 2021).

Despite the growing body of evidence supporting the role of anxiety sensitivity and pain catastrophizing in somatic symptoms, most studies to date have examined these variables in isolation or have focused on specific clinical populations. There remains a critical gap in understanding how these constructs interact in general community samples, particularly within diverse cultural contexts. Research by Ren et al. on self-pain sensitivity and empathy further supports the notion that cultural and individual factors can influence pain perception and somatic awareness, suggesting the need for broader cross-cultural investigations (Ren et al., 2020).

Given the aforementioned evidence, the current study aims to investigate the mediating role of pain catastrophizing in the relationship between anxiety sensitivity and somatic symptoms in a Spanish adult population.

2. Methods and Materials

2.1. Study Design and Participants

This study employed a descriptive correlational design to examine the mediating role of pain catastrophizing in the relationship between anxiety sensitivity and somatic symptoms. A total of 422 participants were selected using a simple random sampling method based on the sample size estimation table by Krejcie and Morgan (1970), ensuring an adequate sample size for statistical validity. All participants were adults residing in Spain, and inclusion criteria required fluency in Spanish and the ability to provide informed consent. The sample included individuals from diverse demographic backgrounds in terms of age, gender, and educational level, aiming to enhance the generalizability of the findings within the Spanish population.

2.2. Measures

2.2.1. Somatic Symptoms

The PHQ-15 (Patient Health Questionnaire-15) is a widely used self-report measure developed by Kroenke, Spitzer, and Williams in 2002 to assess the severity of somatic symptoms. It consists of 15 items that evaluate common physical complaints such as stomach pain, back pain, headaches, fatigue, and shortness of breath experienced over the past four weeks. Respondents rate each symptom on a 3-point Likert scale (0 = “not bothered at all,” 1 = “bothered a little,” 2 = “bothered a lot”), with total scores ranging from 0 to 30. Higher scores indicate greater severity of somatic symptoms. The PHQ-15 has demonstrated excellent psychometric properties, with high internal consistency (Cronbach’s alpha ranging from 0.78 to 0.86) and strong construct and criterion validity confirmed in both clinical and non-clinical populations (Heidari et al., 2021; Schafer & Ferraro, 2013; Seyed Alitabar & Goli, 2024).

2.2.2. Pain Catastrophizing

The Pain Catastrophizing Scale (PCS), developed by Sullivan, Bishop, and Pivik in 1995, is a standard self-report questionnaire designed to assess exaggerated negative mental responses to actual or anticipated pain. The scale includes 13 items and measures three subscales: Rumination (e.g., "I can't stop thinking about how much it hurts"), Magnification (e.g., "I worry that something serious may happen"), and Helplessness (e.g., "There is nothing I can do to reduce the intensity of the pain"). Participants respond on a 5-point Likert scale ranging from 0 ("not at all") to 4 ("all the time"), with total scores ranging from 0 to 52. Higher scores reflect greater levels of pain catastrophizing. The PCS has shown excellent internal consistency (Cronbach’s alpha > 0.90) and robust construct validity across various

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populations and clinical conditions (Hooshmandi et al., 2024; Ogawa et al., 2024).

2.2.3. *Anxiety Sensitivity*

The Anxiety Sensitivity Index-3 (ASI-3), developed by Taylor et al. in 2007, is a revised version of the original ASI designed to provide a more nuanced measure of anxiety sensitivity. The ASI-3 comprises 18 items that assess fear of anxiety-related sensations across three subscales: Physical Concerns (e.g., fear of rapid heartbeat), Cognitive Concerns (e.g., fear of losing mental control), and Social Concerns (e.g., fear of others noticing anxiety). Respondents rate items on a 5-point Likert scale from 0 ("very little") to 4 ("very much"), yielding a total score ranging from 0 to 72. Higher scores indicate greater anxiety sensitivity. The ASI-3 has demonstrated strong psychometric properties, including high internal consistency (Cronbach's alpha ranging from 0.85 to 0.91) and confirmed validity in both clinical and community samples across multiple cultural contexts (Abadi et al., 2024; Bolouk et al., 2024; Hatami Nejad et al., 2024).

2.3. *Data Analysis*

Data were analyzed using SPSS version 27 and AMOS version 21. Descriptive statistics were first calculated to summarize demographic characteristics and the central tendencies of the study variables. Pearson correlation coefficients were computed to examine the bivariate relationships between somatic symptoms (dependent variable) and the independent variables—anxiety sensitivity and pain catastrophizing. To evaluate the hypothesized mediation model, Structural Equation Modeling (SEM) was conducted using AMOS-21. Model fit was assessed using multiple indices including the Chi-square statistic, Comparative Fit Index (CFI), Tucker-Lewis Index (TLI), and Root Mean Square Error of Approximation (RMSEA). The significance of the indirect (mediated) effects was evaluated using bootstrapping with 5,000 resamples to obtain bias-corrected confidence intervals.

3. Findings and Results

The final sample consisted of 422 participants from Spain, including 243 females (57.58%) and 179 males (42.42%). Participants ranged in age from 18 to 65 years, with a mean age of 34.26 years (SD = 10.47). In terms of educational background, 98 participants (23.22%) had completed secondary education, 217 (51.42%) held a bachelor's degree, and 107 (25.35%) had a postgraduate qualification. Regarding employment status, 144 participants (34.12%) were employed full-time, 89 (21.09%) were part-time employees, 73 (17.30%) were students, 66 (15.64%) were unemployed, and 50 (11.85%) were retired. These demographic characteristics reflect a diverse adult sample from various socioeconomic and occupational backgrounds.

Table 1

Descriptive Statistics for Study Variables (N = 422)

Variable	Mean (M)	Standard Deviation (SD)
Anxiety Sensitivity	38.47	8.62
Pain Catastrophizing	26.38	10.17
Somatic Symptoms	14.92	5.78

The results presented in Table 1 indicate that the participants reported a moderate level of anxiety sensitivity (M = 38.47, SD = 8.62) and pain catastrophizing (M = 26.38, SD = 10.17). The average score for somatic symptoms was 14.92 (SD = 5.78), suggesting a notable presence of somatic complaints in the sample.

Prior to conducting the main statistical analyses, the assumptions of normality, linearity, homoscedasticity, and multicollinearity were evaluated and confirmed. Skewness and kurtosis values for all study variables ranged between –0.81 and +0.74, indicating acceptable normal distribution. Linearity and homoscedasticity were assessed using scatterplots and residual plots, which showed no evidence of violations. Multicollinearity diagnostics indicated that tolerance values ranged from 0.61 to 0.78 and variance inflation factor (VIF) values ranged from 1.28 to 1.63, all within acceptable thresholds. These results confirmed that the data met the necessary statistical assumptions for Pearson correlation and SEM analyses.

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Table 2

Pearson Correlations Among Study Variables (N = 422)

Variable	1	2	3
1. Anxiety Sensitivity	–		
2. Pain Catastrophizing	.61** (p < .001)	–	
3. Somatic Symptoms	.49** (p < .001)	.57** (p < .001)	–

Table 2 shows significant positive correlations between all variables. Anxiety sensitivity was positively correlated with pain catastrophizing (r = .61, p < .001) and somatic symptoms (r = .49, p < .001). Pain catastrophizing also correlated strongly with somatic symptoms (r = .57, p < .001), suggesting that higher levels of catastrophizing and anxiety sensitivity are associated with increased somatic complaints.

Table 3

Goodness-of-Fit Indices for the Structural Model

Fit Index	Value	Recommended Threshold
Chi-Square (χ^2)	128.74	–
df	61	–
χ^2/df	2.11	< 3.00
GFI	.94	≥ .90
AGFI	.91	≥ .90
CFI	.97	≥ .95
RMSEA	.051	< .06
TLI	.96	≥ .95

As shown in Table 3, the model demonstrated excellent fit to the data. The chi-square to degrees of freedom ratio was within acceptable limits (χ^2/df = 2.11). Fit indices such as GFI (.94), AGFI (.91), CFI (.97), and TLI (.96) all exceeded recommended thresholds, while RMSEA was .051, indicating a close fit of the model to the data.

Table 4

Total, Direct, and Indirect Path Coefficients Between Study Variables

Path	b	S.E	Beta	p
Anxiety Sensitivity → Pain Catastrophizing	0.42	0.06	.58	< .001
Pain Catastrophizing → Somatic Symptoms	0.31	0.05	.49	< .001
Anxiety Sensitivity → Somatic Symptoms (Direct)	0.09	0.07	.12	.128
Anxiety Sensitivity → Somatic Symptoms (Indirect) via Pain Catastrophizing	0.13	0.03	.28	< .001
Anxiety Sensitivity → Somatic Symptoms (Total)	0.22	0.06	.40	< .001

Table 4 shows that anxiety sensitivity significantly predicted pain catastrophizing (b = 0.42, p < .001), which in turn significantly predicted somatic symptoms (b = 0.31, p < .001). The direct path from anxiety sensitivity to somatic symptoms was not statistically significant (b = 0.09, p = .128), indicating a full mediation. However, the indirect effect through pain catastrophizing was significant (b = 0.13, p < .001), supporting the hypothesis that pain catastrophizing mediates the relationship between anxiety sensitivity and somatic symptoms.

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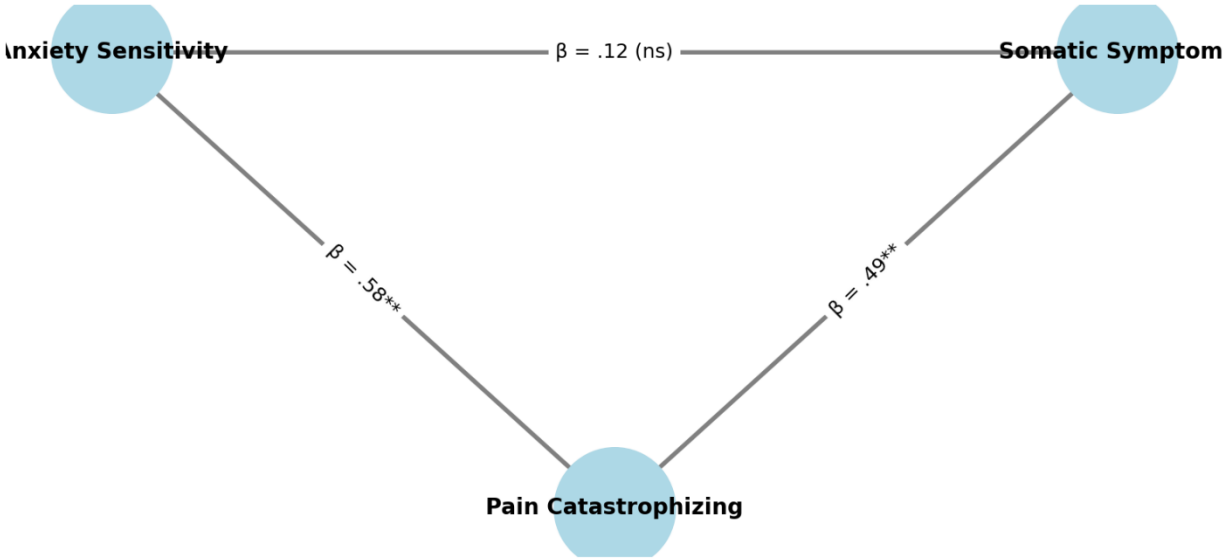
Figure 1

Model with Beta Coefficients



4. Discussion and Conclusion

The present study aimed to explore the mediating role of pain catastrophizing in the relationship between anxiety sensitivity and somatic symptoms in a community sample of Spanish adults. Using Pearson correlation analysis and Structural Equation Modeling (SEM), the results confirmed significant positive correlations between all variables. Specifically, anxiety sensitivity showed a strong positive correlation with both pain catastrophizing and somatic symptoms. Additionally, pain catastrophizing significantly correlated with higher levels of somatic symptoms. The SEM analysis supported a full mediation model, indicating that anxiety sensitivity indirectly affected somatic symptoms through pain catastrophizing. In other words, individuals with high anxiety sensitivity were more likely to engage in catastrophizing thoughts about pain, which in turn increased their experience of somatic symptoms.

These findings align with and extend existing literature by offering empirical support for a cognitive-affective pathway linking anxiety sensitivity to somatic symptomatology through pain-related cognitive distortions. This model builds on previous research showing that anxiety sensitivity amplifies the perception of bodily sensations, often leading to maladaptive interpretations and behavioral responses (Lima et al., 2023; Ogawa et al., 2024). The current results corroborate findings from Wong et al., who demonstrated that pain catastrophizing mediates the relationship between anxiety sensitivity and distress in clinical and non-clinical samples (Wong et al., 2016). Similarly, Burri et al. showed that both anxiety-related traits and catastrophizing share genetic and environmental contributions that influence pain and somatic experiences, further validating the interconnectedness of these variables (Burri et al., 2018).

Our findings also echo the results of Abadi et al., who reported that interventions targeting cognitive patterns such as catastrophizing and anxiety sensitivity were effective in reducing chronic pain symptoms, underscoring the psychological component in somatic distress (Abadi et al., 2024). Additionally, the role of catastrophizing in modulating pain outcomes has been demonstrated in adolescent girls with chronic menstrual pain (Payne et al., 2015), children with chronic pain (Miller et al., 2018), and cancer patients experiencing heightened psychological vulnerability (Raiisi et al., 2022). These studies collectively support the premise that catastrophizing acts as an emotional and cognitive amplifier of distress, often translating into heightened somatic awareness and functional impairment.

The full mediation model confirmed in the current study provides evidence that anxiety sensitivity may not directly lead to somatic symptoms but rather operates through its influence on pain-related cognitive processes. This finding is supported by Conti et al., who demonstrated that pain catastrophizing mediates the relationship between psychological flexibility and emotional distress in chronic pain sufferers (Conti et al., 2020). Similarly, Nahman-Averbuch et al. found strong associations between

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psychological characteristics and pain perception across diverse experimental settings, reinforcing the role of cognitive mediators like catastrophizing in somatic symptomatology (Nahman-Averbuch et al., 2021). Our results align well with these findings and highlight the importance of addressing cognitive biases in the management of somatic symptoms.

From a neurophysiological standpoint, the observed pathway may reflect the influence of central sensitization—a condition where the central nervous system amplifies pain signals due to prolonged exposure to stress and anxiety. Shigetoh et al. demonstrated that central sensitization mediates the relationship between pain intensity and psychological factors, particularly catastrophizing and anxiety-related cognitions (Shigetoh et al., 2019). This neurobiological explanation aligns with our findings by suggesting that anxiety sensitivity primes the individual for hyper-responsiveness, and catastrophizing fuels this sensitivity by maintaining a focus on threat and helplessness.

Additionally, our findings reinforce the biopsychosocial view of somatic symptom disorders, as previously outlined by Roenneberg et al. and Nelson & Novy, who emphasized the multidimensional nature of somatization (Nelson & Novy, 2021; Roenneberg et al., 2019). In this framework, psychological vulnerabilities such as anxiety sensitivity and maladaptive thought patterns like catastrophizing are central to understanding and treating functional somatic symptoms. This integrative perspective has been echoed in more recent theoretical models, including those proposed by Rodriguez et al. and Zanotta, who conceptualized somatic symptom disorders as the product of cognitive-affective dysregulation interacting with bodily awareness and environmental stressors (Rodriguez et al., 2025; Zanotta, 2024).

Our findings also resonate with those of Tutelman et al., who found that pain-related fears and catastrophizing were prominent among survivors of childhood cancer, often predicting long-term psychological distress and somatic complaints (Tutelman et al., 2022). These results highlight the durability of catastrophizing as a psychological trait that influences somatic health across the lifespan and under different contextual stressors. Moreover, Larkin's work on autism and somatic symptoms reinforces the broader relevance of catastrophizing beyond chronic pain syndromes, suggesting it is a transdiagnostic factor associated with heightened bodily distress (Larkin, 2022).

Interestingly, Ren et al. explored the potential of self-pain sensitivity as a proxy for empathy toward others' pain and found that individual differences in sensory processing significantly affect emotional responses (Ren et al., 2020). These findings indirectly support our model by illustrating how heightened sensitivity, when filtered through cognitive schemas like catastrophizing, may intensify internal distress and lead to somatization. In this regard, catastrophizing serves not only as a cognitive bias but also as an emotional amplifier, particularly in individuals with elevated anxiety sensitivity.

The present study offers several theoretical and practical implications. Theoretically, it supports a cognitive-affective model of somatic symptoms, highlighting the mediating role of pain catastrophizing in the relationship between anxiety sensitivity and bodily complaints. Practically, the results underscore the importance of early psychological screening and intervention for individuals displaying high levels of anxiety sensitivity or catastrophizing. These interventions may involve cognitive-behavioral approaches, mindfulness-based strategies, or psychoeducation, all aimed at reducing the tendency to interpret bodily sensations as threatening. The findings advocate for a paradigm shift in somatic symptom treatment from purely somatic to integrative psychological approaches.

Despite its strengths, this study has several limitations that should be acknowledged. First, the cross-sectional design limits the ability to make causal inferences regarding the directionality of the relationships among anxiety sensitivity, pain catastrophizing, and somatic symptoms. Longitudinal designs would better capture the temporal order of these variables. Second, all data were collected through self-report instruments, which may be subject to recall bias and social desirability effects. Third, although the study involved a large and diverse Spanish adult sample, cultural factors unique to Spain may limit the generalizability of findings to other populations. Additionally, the study did not include physiological measures or clinical interviews that could corroborate the presence and severity of somatic symptoms, nor did it assess potential moderating variables such as gender, socioeconomic status, or comorbid mental health conditions.

Future research should adopt longitudinal or experimental designs to examine causal relationships among anxiety sensitivity, pain catastrophizing, and somatic symptoms over time. This would allow for the identification of dynamic patterns and intervention-sensitive windows. Furthermore, expanding the sample to include participants from different cultural, clinical, and age groups would enhance generalizability. Incorporating qualitative methods such as in-depth interviews or narrative analysis could

provide richer insights into the lived experiences of individuals with high anxiety sensitivity and somatic distress. Researchers may also consider examining additional mediators or moderators—such as emotion regulation strategies, mindfulness, or perceived control—to develop more comprehensive models of somatization. Finally, the integration of physiological and neurobiological measures, such as cortisol levels or brain imaging data, could help bridge the gap between cognitive theories and biological mechanisms.

The current findings have practical implications for clinical and community-based interventions. Mental health professionals should consider assessing both anxiety sensitivity and pain catastrophizing in clients presenting with somatic symptoms, as these factors may underlie or exacerbate their physical complaints. Intervention programs that incorporate cognitive restructuring, exposure-based techniques, and emotional regulation skills can help reduce the cognitive distortions and anxiety-driven interpretations associated with bodily sensations. Moreover, psychoeducation about the role of thoughts and beliefs in physical symptom experiences may empower individuals to reframe their bodily experiences in less threatening terms. Preventive programs in schools, workplaces, and primary healthcare settings could also be designed to teach emotional resilience and bodily awareness, potentially reducing the progression from anxiety to somatic distress. Integrating these psychological elements into routine care may ultimately lead to more effective, patient-centered approaches in managing somatic symptom disorders.

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

The author report no conflict 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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