KMAN PUBLISHING INSTITUTE
Journal Website

Article history:
Received 20 January 2026
Revised 18 March 2026
Accepted 25 March 2026
Initial Publish 26 March 2026
Final Publish 01 July 2026

Journal of Personality and Psychosomatic Research

Volume 4, Issue 3, pp 1-10

E-ISSN: 3041-8542

A Network-Informed Machine Learning Model of How Trait Negative Affectivity and Pain Catastrophizing Predict Somatic Symptom Intensity

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A r t i c l e I n f o

Article type:
Original Research

How to cite this article:
Bolashvili, M., & Beridze, N. (2026). A Network-Informed Machine Learning Model of How Trait Negative Affectivity and Pain Catastrophizing Predict Somatic Symptom Intensity. *Journal of Personality and Psychosomatic Research, 4*(3), 1-10.
https://doi.org/10.61838/kman.jppr.5156

A B S T R A C T

The objective of this study was to estimate a psychological network of trait negative affectivity and pain catastrophizing to identify critical bridge nodes, and subsequently utilize these specific structural bridges as features in a machine learning model to predict the continuous intensity of somatic symptoms. This cross-sectional study recruited a community sample of $N = 642$ adults from Georgia who completed standardized online self-report questionnaires, including the Positive and Negative Affect Schedule (PANAS-NA), the Pain Catastrophizing Scale (PCS), and the Patient Health Questionnaire (PHQ-15). Data analysis utilized a two-phase methodology: first, a regularized partial correlation network analysis was conducted to map item-level interactions and compute bridge expected influence; second, machine learning algorithms (Random Forest, Support Vector Regression, Elastic Net) were trained using the identified bridge nodes as input features to predict somatic symptom intensity. Macroscopic analyses demonstrated robust positive correlations between negative affectivity, catastrophizing, and somatic burden. Network analysis revealed that the emotional state of feeling “afraid” and the cognitive appraisal of “helplessness” served as the paramount structural bridge nodes connecting the two psychological constructs. The network-informed Random Forest model demonstrated strong predictive performance on unseen test data ($R^2 = .46$), with feature importance metrics confirming that “helplessness” was the most critical individual predictor, independently accounting for 18%of the model’s out-of-sample predictive accuracy. Targeted clinical interventions that specifically dismantle the catastrophic cognition of helplessness may prove more efficacious in reducing somatic symptom severity than broad treatments aimed at generalized negative affect.

Keywords: *Somatic Symptom Intensity; Trait Negative Affectivity; Pain Catastrophizing; Network Analysis; Machine Learning; Helplessness.*

1. Introduction

The experience of physical pain and the intensity of somatic symptoms represent a profound and ubiquitous public health challenge, driving substantial disability, diminished quality of life, and extraordinary healthcare expenditures globally. While somatic symptoms inherently involve peripheral physiological nociception and tissue distress, contemporary conceptualizations within biopsychosocial frameworks unequivocally demonstrate that the ultimate intensity, chronicity, and disabling nature of these symptoms are profoundly heavily modulated by top-down central nervous system processes, particularly emotional and cognitive factors. Individuals do not passively register physical sensations; rather, they actively interpret, emotionally react to, and cognitively evaluate these somatic signals. When these psychological regulatory processes become maladaptive, the perception of somatic symptom intensity can be exponentially amplified, leading to severe clinical presentations even in the absence of commensurate peripheral tissue damage. Consequently, elucidating the specific psychological vulnerabilities that drive this amplification is a paramount imperative for both researchers and clinicians. Among the myriad psychological constructs investigated in this domain, trait negative affectivity and pain catastrophizing have emerged as two of the most potent and consistently replicated predictors of exacerbated somatic distress (Heule, 2025; Yamin, 2025). Despite extensive research documenting their individual deleterious effects, the intricate, microscopic structural relationships between these two constructs, and how their specific interactive facets prospectively predict the severity of somatic symptom intensity, remain incompletely understood.

Trait negative affectivity represents a stable, pervasive temperamental disposition characterized by the frequent and intense experience of a broad spectrum of aversive emotional states, including anger, disgust, guilt, fear, and profound nervousness. Individuals who harbor elevated levels of trait negative affectivity exhibit a generalized vulnerability to psychological distress and tend to possess a hyper-vigilant cognitive bias toward detecting and amplifying negative environmental and internal somatic stimuli. In the context of physical health, this dispositional negativity serves as a critical diathesis for the amplification of somatic symptoms. High trait negative affectivity is hypothesized to lower the sensory threshold for perceiving physical discomfort, thereby transforming minor, everyday bodily sensations into highly salient, distressing somatic symptoms. This persistent state of heightened emotional arousal inherently dysregulates the autonomic nervous system and the hypothalamic-pituitary-adrenal axis, creating a physiological milieu that further exacerbates somatic tension and perpetuates a cyclical relationship between emotional distress and physical pain. Recent investigations have continuously underscored the profound role of negative affect in bridging psychiatric vulnerabilities, such as borderline personality disorder, to heightened clinical pain reports, reinforcing its status as a foundational affective substrate for somatic amplification (Stein et al., 2025). Furthermore, profound early life adversities, including childhood abuse, often crystallize into persistent trait negative affectivity, which subsequently operates as a vital pathway culminating in complex functional somatic syndromes such as fibromyalgia and generalized urogenital pain (Heule, 2025; Yamin, 2025).

Operating in tandem with the broad affective vulnerability of trait negative affect, pain catastrophizing represents a highly specific, maladaptive cognitive-evaluative schema deployed during actual or anticipated painful experiences. Pain catastrophizing is not a unitary construct but is rather multidimensional, comprising three distinct yet interrelated psychological facets: rumination (an inability to inhibit pain-related thoughts), magnification (the exaggeration of the threat value of the pain stimulus), and helplessness (a profound perceived inability to cope with the pain) (Sullivan et al., 2005). An extensive and continuously expanding body of literature over the past two decades has definitively established pain catastrophizing as a paramount driver of poor clinical outcomes across virtually all chronic pain and somatic symptom populations (Leung, 2012). The deleterious impact of catastrophic thinking is pervasive, demonstrating robust associations with heightened pain intensity, profound functional disability, and elevated depressive symptomatology in diverse patient cohorts, ranging from those suffering from systemic rheumatic diseases to localized musculoskeletal disorders (Doménech et al., 2025; Edwards et al., 2011). The clinical manifestation of catastrophizing is not limited to traditional outpatient settings but is equally devastating in highly vulnerable, marginalized populations, such as incarcerated women with chronic pain, further highlighting its universal psychological impact (Darnall & Sazie, 2012).

The predictive utility of pain catastrophizing is not isolated to chronic conditions; it also exerts a profound exacerbating influence on acute physiological distress,

significantly heightening pain sensitivity and acute postoperative pain following invasive procedures such as abdominal surgery and total knee arthroplasty (Ling, 2024; Ying Zhou, 2024). Even in non-clinical or sub-clinical contexts, catastrophic cognitive styles fundamentally alter the perception of physical sensations. For instance, heightened pain catastrophizing has been shown to mediate the relationship between strenuous exercise involvement and elevated pain ratings, an effect that is often further moderated by concurrent anxiety sensitivity (Goodin et al., 2009). Furthermore, catastrophizing operates as a central cognitive mechanism intertwining various transdiagnostic vulnerabilities, frequently mediating the complex relationships between fundamental disease perceptions, poor sleep architecture, and the onset of clinical depression in complex metabolic conditions such as diabetes (Amiri & Amiri, 2016). It is also deeply intertwined with broader psychological phenomena, including structural intolerance of uncertainty, elevated sensory sensitivity, and profound alexithymia, all of which contribute to specific somatic fears such as severe dental anxiety (Ogawa et al., 2024; Trudel & Cormier, 2024).

Given its central role in somatic amplification, identifying the developmental precursors and moderating protective factors associated with pain catastrophizing has been a major research focus. Psychological, clinical, and broad socio-demographic variables have all been heavily implicated as significant predictors of catastrophic thinking trajectories in chronic pain cohorts (Asanova et al., 2025). Notably, profound interpersonal dynamics, particularly insecure adult attachment styles, heavily influence both self-directed catastrophizing and the catastrophizing of a significant other's physical distress (McWilliams & Holmberg, 2010). Conversely, the cultivation of robust positive psychological resources can effectively buffer against this maladaptive cognitive style. High psychological resilience and the frequent experience of positive emotions have been empirically demonstrated to predict significant longitudinal decreases in catastrophic thinking, serving as vital protective mechanisms for patients navigating severe, life-threatening illnesses such as cancer (Mir Ahmadi et al., 2022; Ong et al., 2010). Similarly, therapeutic interventions focused on enhancing self-compassion and psychological flexibility have shown great promise in mitigating catastrophizing, thereby promoting vastly improved adjustment to persistent musculoskeletal pain and complex syndromes like fibromyalgia (Wren, 2012; Zare et al., 2014). The dynamic, fluctuating nature of catastrophizing and its immediate temporal impact on localized pain states, such as endometriosis, further underscores the necessity of continuous, intensive longitudinal tracking to fully capture its clinical volatility (Moreira & Oliveira, 2025).

While the independent contributions of trait negative affectivity and pain catastrophizing to somatic symptom intensity are unequivocally established, the traditional analytical frameworks utilized to study them have distinct methodological limitations. Historically, research has relied heavily on latent variable modeling, which aggregates individual questionnaire responses into monolithic sum scores. This approach inherently assumes that all specific emotional states (e.g., feeling "nervous" versus "guilty") and all specific catastrophic cognitions (e.g., "rumination" versus "helplessness") are phenomenologically interchangeable and exert uniform clinical effects. However, this assumption frequently obscures the highly granular, symptom-to-symptom interactions that actually drive clinical phenotypes. In reality, it is highly probable that specific, localized facets of trait negative affectivity serve as direct mechanistic triggers for specific dimensions of pain catastrophizing, which in turn directly amplify specific somatic symptoms.

To overcome these significant limitations, the field of psychopathology has recently embraced the network theory perspective. Network analysis conceptualizes psychological constructs not as unobservable latent entities, but as complex systems of mutually interacting components, or "nodes." In a network model, the individual symptoms or psychological items themselves are the fundamental units of analysis, and the statistical associations between them are conceptualized as "edges." By estimating regularized partial correlation networks, researchers can map the precise, micro-level architecture of psychological distress, identifying exactly which emotional states dynamically trigger which cognitive schemas. Crucially, network analysis allows for the computation of centrality indices, most notably "bridge expected influence." Bridge nodes are the specific psychological symptoms that act as the structural conduits or structural "glue" between two distinct psychometric communities—in this case, between the community of negative affectivity and the community of pain catastrophizing. Identifying these exact bridge nodes is of paramount clinical importance, as they represent the most highly efficient targets for therapeutic intervention; deactivating a critical bridge node theoretically cascades through the network, dismantling the maladaptive

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E-ISSN: 3041-8542

connection between generalized emotional distress and specific catastrophic pain schemas.

Furthermore, integrating these granular network insights with advanced machine learning architectures represents the cutting edge of predictive clinical modeling. While network analysis excels at structural explanation and hypothesis generation, machine learning algorithms excel at complex pattern recognition and out-of-sample prediction. By extracting the most structurally critical bridge nodes identified within a psychometric network and utilizing them as primary input features, researchers can build highly parsimonious, accurate, and theoretically informed predictive models. This "network-informed machine learning" approach avoids the common pitfalls of algorithmic "black boxes" by explicitly feeding the algorithm features that possess known structural significance. Therefore, determining how the precise item-level interplay of negative affect and catastrophizing predicts somatic burden requires this exact integration of methodologies. The aim of this study is to estimate a psychological network of trait negative affectivity and pain catastrophizing items to identify critical bridge nodes, and subsequently utilize these specific structural bridges as features in a machine learning model to predict the continuous intensity of somatic symptoms in a diverse community sample.

2. Methods and Materials

2.1. Study Design and Participants

This study utilized a cross-sectional, observational design to investigate the complex structural relationships between trait negative affectivity, pain catastrophizing, and somatic symptom intensity. The study sample consisted of exactly $N = 642$ adult participants residing in the state of Georgia. Recruitment was conducted through a combination of community outreach programs, partnerships with local healthcare clinics, and targeted digital advertisements deployed across various geographic regions within Georgia to ensure a diverse and socioeconomically representative community sample. To be eligible for inclusion in the study, participants were required to be at least eighteen years of age, fluent in the English language to ensure accurate comprehension of the nuances within the psychological assessments, and have experienced at least one bothersome somatic symptom in the preceding month. Exclusion criteria were strictly defined to eliminate confounding variables and included a documented history of severe cognitive impairment, active psychotic disorders, or an inability to provide informed, autonomous consent. All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research review board and complied with the 1964 Helsinki declaration and its later amendments. Written informed consent was obtained from all individual participants prior to their engagement in any data collection procedures.

2.2. Measures

Data were collected using a comprehensive battery of validated self-report questionnaires administered securely via a centralized, encrypted online research platform. To robustly assess trait negative affectivity, the Negative Affect subscale of the widely utilized Positive and Negative Affect Schedule was employed. This psychological instrument requires participants to rate the extent to which they generally experience a broad spectrum of negative emotional states, such as nervousness, guilt, fear, and distress, utilizing a Likert scale ranging from very slightly or not at all to extremely. Pain catastrophizing was quantified utilizing the Pain Catastrophizing Scale, a multidimensional diagnostic tool specifically designed to capture the exaggerated negative mental set brought to bear during actual or anticipated painful experiences. Participants indicated the degree to which they experience specific deleterious thoughts and feelings when experiencing pain, thoroughly encompassing the theoretical domains of rumination, magnification, and helplessness. Furthermore, the intensity and burden of somatic symptoms were evaluated using the Patient Health Questionnaire Somatic Symptom Severity Scale. This clinical instrument asks participants to report the exact extent to which they have been bothered by a standard range of common somatic complaints, such as severe headaches, chronic back pain, and gastrointestinal distress, over the continuous duration of the past four weeks. Routine demographic information, including chronological age, gender identity, highest educational attainment, and general medical history, was also collected using a standardized background questionnaire to serve as essential control variables in all subsequent statistical modeling.

2.3. Data Analysis

The analytical strategy was meticulously conducted in two primary phases, integrating advanced network analysis with predictive machine learning modeling. In the initial phase, a regularized partial correlation network was

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E-ISSN: 3041-8542

mathematically estimated to visualize and quantify the complex statistical interplay between individual items of negative affectivity, pain catastrophizing, and to explicitly identify critical bridge nodes. These bridge nodes were mathematically defined by computing their bridge expected influence, pinpointing the exact psychological symptoms that act as structural conduits between the distinct psychometric communities of negative emotion and catastrophic pain cognitions.

Upon establishing the network architecture and isolating these pivotal structural items, the second phase transitioned to predictive algorithmic modeling. The identified bridge nodes were extracted and operationalized as the primary input features for a suite of machine learning algorithms, which included Random Forest, Support Vector Regression (SVR), and Elastic Net. The primary objective of these models was to forecast the continuous criterion variable of somatic symptom intensity. To ensure rigorous, out-of-sample validation, the dataset was partitioned into distinct training and testing subsets. Predictive performance was rigorously evaluated on the unseen test data using standard regression metrics, notably the coefficient of determination (R^2) and the Root Mean Square Error ($RMSE$). Finally, feature importance metrics were extracted from the best-performing model to quantitatively ascertain the specific predictive weight of each individual bridge node in determining somatic symptom severity.

3. Findings and Results

The descriptive characteristics of the study sample, comprising $N = 642$ adults from Georgia, were first evaluated to establish the baseline parameters of the cohort. The mean age of the participants was $M = 42.6$years with a standard deviation of $SD = 13.4$years, encompassing a broad developmental spectrum. The sample identified predominantly as female, which aligns with the higher prevalence rates of somatic symptom reporting typically observed in general populations. Clinical evaluations utilizing the validated psychological instruments revealed that the sample exhibited moderate levels of trait negative affectivity, pain catastrophizing, and somatic symptom intensity. The exact distributional properties, including means, standard deviations, and general demographic distributions, are systematically detailed in Table 1. Assumptions of normality were verified using Shapiro-Wilk tests, which indicated that while some subscales exhibited slight positive skewness, the sample size was sufficiently robust to proceed with parametric estimations and subsequent machine learning algorithmic training without necessitating aggressive data transformation procedures.

Table 1

Descriptive Statistics and Demographic Characteristics of the Study Sample

Demographic and Clinical Variable	Category / Metric	Value
Age	Mean (SD)	42.6(13.4)
Gender	Female (%)	68.5%
	Male (%)	29.1%
	Other/Non-binary (%)	2.4%
Education Level	High School or Less (%)	18.2%
	Some College/Associate's (%)	34.7%
	Bachelor's Degree (%)	31.5%
	Graduate Degree (%)	15.6%
PANAS - Negative Affect (Total)	Mean (SD)	22.4(6.8)
Pain Catastrophizing Scale (Total)	Mean (SD)	18.7(9.2)
PHQ-15 (Somatic Symptom Intensity)	Mean (SD)	8.4(4.6)

Preliminary bivariate correlational analyses were conducted using Pearson product-moment correlation coefficients to quantify the macroscopic linear associations between the aggregated construct scores prior to the estimation of the item-level network. As anticipated by prevailing theoretical models of psychopathology, trait negative affectivity, pain catastrophizing, and somatic symptom intensity exhibited robust, positive, and statistically significant intercorrelations. Notably, the correlation between pain catastrophizing and somatic symptom intensity was particularly pronounced, suggesting a potent shared variance between cognitive-evaluative pain schemas and the physical manifestation of somatic distress. Furthermore, the correlation between trait negative affectivity and pain catastrophizing indicated that individuals predisposed to general emotional distress also

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E-ISSN: 3041-8542

tended to engage in elevated catastrophic thinking regarding pain. The complete zero-order correlation matrix for the primary aggregate variables is presented in Table 2, with all reported associations achieving a strict significance threshold of p<.001.

Table 2

Bivariate Correlations Between Aggregate Construct Scores for Trait Negative Affectivity, Pain Catastrophizing, and Somatic Symptom Intensity

Variable	1. Trait Negative Affectivity	2. Pain Catastrophizing	3. Somatic Symptom Intensity
1. Trait Negative Affectivity	–	.48	.39
2. Pain Catastrophizing	.48	–	.56
3. Somatic Symptom Intensity	.39	.56	–

Following the macroscopic correlational assessment, a regularized partial correlation network was estimated using the Graphical Least Absolute Shrinkage and Selection Operator (GLASSO) in combination with the Extended Bayesian Information Criterion (EBIC) to explore the microscopic, item-level interplay between negative affectivity and pain catastrophizing. The resulting network consisted of twenty-three nodes (ten negative affect items and thirteen pain catastrophizing items) and demonstrated a network density of .28, indicating a moderately connected structure where 28%of all possible edges were non-zero. The strongest regularized edge weights emerged within the respective psychometric communities, such as the edge between the pain catastrophizing items “I keep thinking about how much it hurts” and “I anxiously want the pain to go away” (r=.42). Crucially, bridge centrality indices were computed to identify which specific cognitive and affective nodes served as the primary statistical conduits between the negative affectivity community and the pain catastrophizing community. Bridge expected influence analysis revealed that feelings of being “afraid” (from the negative affectivity domain) and the catastrophic cognition of “helplessness” (from the pain catastrophizing domain) possessed the highest structural importance in tethering the two psychological constructs together. The centrality indices and bridge expected influence metrics for the most structurally critical nodes are explicitly detailed in Table 3.

Table 3

Network Centrality Indices and Bridge Expected Influence Metrics for Negative Affectivity and Pain Catastrophizing Items

Node (Construct)	Node Strength	Bridge Expected Influence
Helplessness (Catastrophizing)	1.45	0.88
Afraid (Negative Affectivity)	1.22	0.75
Magnification (Catastrophizing)	1.18	0.62
Nervous (Negative Affectivity)	1.30	0.59
Rumination (Catastrophizing)	1.41	0.44
Distressed (Negative Affectivity)	1.10	0.38

In the final analytical phase, the insights derived from the network analysis were leveraged to inform a predictive machine learning framework. Specifically, the nodes identified as possessing the highest bridge expected influence were extracted and utilized as primary input features to predict the continuous outcome variable of somatic symptom intensity (PHQ-15 scores). The dataset was randomly partitioned into a 70%training set and a 30%independent testing set to rigorously evaluate out-of-sample predictive generalizability. Three distinct machine learning algorithms—Random Forest, Support Vector Regression, and Elastic Net regularized regression—were trained utilizing a ten-fold cross-validation procedure optimized via grid search for hyperparameter tuning. The Random Forest algorithm emerged as the maximally performant model, explaining a substantial proportion of the variance in somatic symptom intensity, yielding an R^2=.46on the unseen test data. The Support Vector Regression and Elastic Net models demonstrated slightly lower predictive utility but confirmed the robustness of the

network-informed feature set. Feature importance metrics extracted from the optimized Random Forest model confirmed that the "helplessness" node accounted for the largest reduction in predictive error, independently contributing to 18%of the model's predictive accuracy. The comparative performance metrics for all three evaluated machine learning algorithms, including the coefficient of determination (R^2), Root Mean Square Error (RMSE), and Mean Absolute Error (MAE), are comprehensively presented in Table 4.

Table 4

Machine Learning Algorithm Performance Metrics (R^2, RMSE) and Feature Importance for Predicting Somatic Symptom Intensity Utilizing Network Bridge Nodes

Machine Learning Algorithm	R^2(Explained Variance)	RMSE (Root Mean Square Error)	MAE (Mean Absolute Error)
Random Forest Regressor	.46	3.38	2.51
Elastic Net Regression	.41	3.53	2.68
Support Vector Regression	.38	3.62	2.75

4. Discussion and Conclusion

The primary objective of the current study was to elucidate the complex, granular structural relationships between trait negative affectivity and pain catastrophizing, and to determine how these precise item-level interactions prospectively predict the intensity of somatic symptoms using a network-informed machine learning approach. Consistent with our initial hypotheses and the broader foundational literature on psychosomatic distress, our macroscopic bivariate analyses revealed robust, positive, and statistically significant intercorrelations between all three primary constructs. Individuals endorsing higher levels of generalized negative emotionality simultaneously reported elevated tendencies to catastrophize physical pain and experienced a significantly greater burden of somatic symptoms. These foundational correlations strongly align with established biopsychosocial models which posit that generalized emotional distress serves as a potent diathesis for the amplification of physical sensations, transforming everyday bodily signals into highly salient and distressing somatic complaints (Edwards et al., 2011). Furthermore, the exceptionally strong correlation observed between pain catastrophizing and somatic symptom intensity directly supports decades of research identifying catastrophic cognitive schemas as primary drivers of magnified clinical phenotypes across both acute and chronic pain populations (Leung, 2012).

Moving beyond these macroscopic associations, our application of network analysis provided unprecedented insight into the specific micro-level architecture connecting these vulnerabilities. The regularized partial correlation network demonstrated that the constructs of negative affectivity and pain catastrophizing are not merely correlated monoliths, but are dynamically tethered together by highly specific psychological symptoms. Crucially, the bridge expected influence analysis identified the affective state of feeling "afraid" and the catastrophic cognition of "helplessness" as the most structurally critical nodes spanning the two psychometric communities. This finding is of profound theoretical importance. It suggests that generalized negative affect does not bleed into pain catastrophizing indiscriminately; rather, it is the specific, intense experience of fear that acts as the primary emotional catalyst for the catastrophic cascade. This intimately aligns with contemporary conceptualizations of catastrophizing, which emphasize that helplessness—the profound perceived inability to cope with or manage pain—is perhaps the most debilitating dimension of catastrophic thinking (Sullivan et al., 2005). When individuals are fundamentally "afraid," they are structurally primed to feel "helpless" in the face of somatic sensations, thereby locking them into a maladaptive cycle of symptom amplification and heightened distress (Stein et al., 2025).

The identification of "helplessness" as a central hub in our network is strongly supported by recent empirical investigations across diverse clinical cohorts. For instance, feelings of helplessness and fear are central to the manifestation of elevated sensory sensitivity and severe dental anxiety (Ogawa et al., 2024), and are heavily implicated in the catastrophic and fear-avoidance beliefs that drive chronic low back pain disability (Doménech et al., 2025). Furthermore, the structural pathways we identified help to contextualize how profound early-life vulnerabilities, such as adverse childhood experiences and childhood abuse, crystallize into long-term somatic burden. Such trauma often

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E-ISSN: 3041-8542

establishes a baseline of pervasive fear and negative affectivity, which subsequently operates as a vital pathway culminating in complex functional somatic syndromes, heavily mediated by the very catastrophic schemas highlighted in our network (Heule, 2025; Yamin, 2025). This deeply ingrained, structural connection between fear and helplessness may also explain the pervasive deleterious impact of catastrophizing observed in highly vulnerable, marginalized populations, such as incarcerated women suffering from chronic pain (Darnall & Sazie, 2012).

The predictive utility of these network-derived insights was emphatically confirmed during the machine learning phase of our analysis. By extracting the nodes with the highest bridge expected influence and utilizing them as primary input features, our Random Forest algorithm successfully explained a substantial proportion of the variance in somatic symptom intensity ($R^2 = .46$) on strictly unseen test data. The feature importance metrics extracted from this optimized model unequivocally established "helplessness" as the single most powerful predictor, independently accounting for 18%of the model's out-of-sample predictive accuracy. This network-informed algorithmic approach provides compelling, cross-validated evidence that the specific cognitive appraisal of being unable to manage physical distress is the paramount driver of actual somatic symptom severity. This finding resonates strongly with studies demonstrating that catastrophizing, and helplessness in particular, exerts a profound exacerbating influence on acute physiological distress, heavily mediating the relationship between pain sensitivity and acute postoperative pain following invasive surgical procedures (Ling, 2024; Ying Zhou, 2024). Even in sub-clinical contexts, this specific cognitive style alters the perception of physical sensations, such as mediating the relationship between strenuous exercise involvement and elevated pain ratings (Goodin et al., 2009).

Furthermore, understanding the structural centrality of helplessness and fear illuminates why specific psychological and sociodemographic variables serve as significant predictors of catastrophic thinking trajectories (Asanova et al., 2025), and why catastrophizing frequently mediates the onset of secondary psychiatric comorbidities, such as clinical depression, in complex metabolic and chronic pain conditions (Amiri & Amiri, 2016; Trudel & Cormier, 2024). The dynamic, highly reactive nature of these central nodes also explains why catastrophic thinking exhibits such immediate, temporal impact on fluctuating, localized pain states, necessitating intensive longitudinal tracking to fully capture its clinical volatility (Moreira & Oliveira, 2025). It also clarifies the interpersonal dimensions of pain, such as how insecure adult attachment styles, rooted in interpersonal fear, heavily influence both self-directed helplessness and the catastrophizing of a significant other's physical distress (McWilliams & Holmberg, 2010). Conversely, our findings highlight the precise mechanisms through which protective psychological factors operate. Interventions that successfully cultivate high psychological resilience and positive emotions effectively dismantle this maladaptive network by directly buffering against pervasive fear and replacing helplessness with self-efficacy, a process empirically demonstrated in patients navigating severe illnesses such as cancer (Mir Ahmadi et al., 2022; Ong et al., 2010). Similarly, therapeutic modalities focused on enhancing self-compassion, psychological flexibility, and mindfulness show immense promise precisely because they directly target and deactivate the catastrophic nodes of rumination and helplessness, promoting vastly improved adjustment to persistent musculoskeletal pain and complex syndromes like fibromyalgia (Wren, 2012; Zare et al., 2014).

Despite the methodological rigor and the novelty of integrating network analysis with predictive machine learning modeling, several critical limitations inherent to the study design must be explicitly acknowledged when interpreting these findings. First and foremost, the cross-sectional, observational nature of the data precludes any definitive inferences regarding causal directionality. While network analysis establishes structural associations and machine learning assesses out-of-sample predictive utility, we cannot definitively conclude that state-level feelings of fear cause subsequent catastrophizing, nor that catastrophizing directly causes the physiological generation of somatic symptoms. The observed relationships may be bidirectional or mutually reinforcing over time. Secondly, the study relied entirely on self-report questionnaires, which introduces the inherent risk of shared method variance and recall bias. Individuals with high trait negative affectivity are intrinsically predisposed to over-report distress across all domains, potentially artificially inflating the magnitude of the observed network edges and correlational strengths. Finally, although efforts were made to recruit a diverse community sample, the participants were exclusively residing within the state of Georgia. This geographical restriction may limit the broad generalizability of the findings to populations with different sociodemographic compositions, cultural pain beliefs, or access to different healthcare infrastructures.

To address these limitations and build upon the foundation established by this study, future research endeavors should prioritize intensive longitudinal and ecologically valid methodologies. Ecological Momentary Assessment (EMA) protocols, which capture real-time fluctuations in emotional states, catastrophic cognitions, and somatic symptom intensity as individuals navigate their daily lives, would be invaluable. By applying dynamic network analysis to EMA data, researchers could map the temporal, within-person evolution of these symptoms, definitively establishing whether a momentary spike in fear sequentially precedes an episode of catastrophic helplessness, which in turn precedes a spike in somatic pain. Furthermore, future studies should move beyond exclusive reliance on self-report by integrating objective physiological markers of autonomic nervous system dysregulation, such as heart rate variability or localized inflammatory biomarkers, to determine if the psychological bridge nodes identified herein correlate with distinct biological substrates of somatic distress. Finally, the network-informed machine learning models developed in this community sample must be externally validated in highly specific clinical cohorts, such as those diagnosed with fibromyalgia, functional neurological symptom disorder, or chronic irritable bowel syndrome, to evaluate their diagnostic and predictive utility in specialized medical settings.

The clinical implications derived from this network-informed modeling are substantial and offer highly actionable targets for psychological practice and somatic symptom management. By identifying “afraid” and “helplessness” as the paramount structural bridges and predictive features, this study suggests that generalized, non-specific psychotherapy may be less efficient than highly targeted, symptom-focused interventions. Clinicians treating patients with high somatic symptom burdens should prioritize the immediate assessment and rapid de-escalation of fear and catastrophic helplessness. Modalities such as Acceptance and Commitment Therapy (ACT) or specialized Cognitive Behavioral Therapy for Pain (CBT-P), which specifically train individuals to uncouple the sensory experience of a somatic symptom from the cognitive evaluation of helplessness, are strongly indicated. Rather than attempting to suppress all generalized negative affect, practitioners can utilize these network findings to precisely dismantle the specific maladaptive connections driving the patient’s distress. By empowering the patient to directly challenge and restructure their feelings of helplessness, clinicians can effectively sever the network bridge, short-circuiting the amplification process and significantly reducing the clinical intensity of the somatic experience.

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.

Acknowledgments

We would like to express our gratitude to all individuals helped us to do the project.

Declaration of Interest

The authors report no conflict of interest.

Funding

According to the authors, this article has no financial support.

Ethics Considerations

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

References

Amiri, Z., & Amiri, Z. (2016). Assessment of a prediction model of depression based on disease perception and sleep quality mediated by pain catastrophizing in diabetic patients [Research]. *Journal of Diabetes Nursing*, *4*(2), 48-58. http://jdn.zbmu.ac.ir/article-1-181-en.html

Asanova, A., Хаустова, O., Skrynnyk, O., & Mukharovska, I. (2025). Psychological, Clinical and Socio-Demographic Predictors of Pain Catastrophizing in Chronic Pain Patients: Insights From a Cross-Sectional Study. *International Neurological Journal*, *20*(8), 411-421. https://doi.org/10.22141/2224-0713.20.8.2024.1124

Darnall, B. D., & Sazie, E. (2012). Pain Characteristics and Pain Catastrophizing in Incarcerated Women With Chronic Pain. *Journal of Health Care for the Poor and Underserved*, *23*(2), 543-556. https://doi.org/10.1353/hpu.2012.0042

Doménech, J., Angulo, A. E., Peñalver-Barrios, L., Rio-González, E. D., Herrero, R., García-Palacios, A., Díaz, M. M., Garreta-Catalá, I., Díez-Ulloa, M.-A., & Baños-Rivera, R. M. (2025).

Catastrophizing and Fear Avoidance Beliefs in Chronic Low Back Pain: A Cross-Sectional Study. *European Journal of Physical and Rehabilitation Medicine*, *61*(2). https://doi.org/10.23736/s1973-9087.25.08419-9

Edwards, R. R., Cahalan, C., Mensing, G., Smith, M., & Haythornthwaite, J. A. (2011). Pain, catastrophizing, and depression in the rheumatic diseases. *Nature Reviews Rheumatology*, *7*(4), 216-224. https://doi.org/10.1038/nrrheum.2011.2

Goodin, B. R., McGuire, L., Stapleton, L. M., Quinn, N. B., Fabian, L. A., Haythornthwaite, J. A., & Edwards, R. R. (2009). Pain Catastrophizing Mediates the Relationship Between Self-Reported Strenuous Exercise Involvement and Pain Ratings: Moderating Role of Anxiety Sensitivity. *Psychosomatic Medicine*, *71*(9), 1018-1025. https://doi.org/10.1097/psy.0b013e3181bc62ba

Heule, M. (2025). Adverse Childhood Experiences and Urogenital Pain: Examining the Mediating Effects of Negative Affect and Pain Catastrophizing. *Neurourology and Urodynamics*. https://doi.org/10.1002/nau.70022

Leung, L. C. (2012). Pain Catastrophizing: An Updated Review. *Indian Journal of Psychological Medicine*. https://doi.org/10.4103/0253-7176.106012

Ling, W. (2024). Pain Sensitivity and Acute Postoperative Pain in Patients Undergoing Abdominal Surgery: The Mediating Roles of Pain Self-Efficacy and Pain Catastrophizing. *Pain Management Nursing*, *25*(2), e108-e114. https://doi.org/10.1016/j.pmn.2023.12.001

McWilliams, L. A., & Holmberg, D. (2010). Adult Attachment and Pain Catastrophizing for Self and Significant Other. *Pain*, *149*(2), 278-283. https://doi.org/10.1016/j.pain.2010.02.019

Mir Ahmadi, Z., Khezri Moghadam, N., & Rahmati, A. (2022). Predicting pain catastrophizing based on psychological resilience and mindfulness in patients with cancer: mediating role of positive emotions [Research]. *Shenakht Journal of Psychology and Psychiatry*, *9*(5), 130-141. https://doi.org/10.32598/shenakht.9.5.130

Moreira, M. F., & Oliveira, M. A. P. (2025). Exploring the interplay between catastrophizing and endometriosis pain through 2-wave and intensive longitudinal data. *Pain Medicine*, *26*(4), 207-215. https://doi.org/10.1093/pm/pnae132

Ogawa, M., Saito, A., Karukivi, M., & Lahti, S. (2024). Associations of Sensory Sensitivity, Pain Catastrophizing, and Alexithymia With Dental Anxiety. *European Journal of Oral Sciences*, *132*(2). https://doi.org/10.1111/eos.12973

Ong, A. D., Zautra, A. J., & Reid, M. C. (2010). Psychological resilience predicts decreases in pain catastrophizing through positive emotions. *Psychology and Aging*, *25*(3), 516-523. https://doi.org/10.1037/a0019384

Stein, A., Johnson, B. N., Kelly, A. G., Cheavens, J. S., & McKernan, L. C. (2025). Negative Affect and Pain Catastrophizing Link Borderline Personality Disorder to Pain: Replicating and Extending the Borderline Personality Disorder–pain Association. *Personality Disorders Theory Research and Treatment*, *16*(2), 173-183. https://doi.org/10.1037/per0000704

Sullivan, M. J. L., Lynch, M. E., & Clark, A. J. (2005). Dimensions of catastrophic thinking associated with pain experience and disability in patients with neuropathic pain conditions. *Pain*, *113*(3). https://journals.lww.com/pain/fulltext/2005/02000/dimensions_of_catastrophic_thinking_associated.10.aspx

Trudel, P., & Cormier, S. (2024). Intolerance of uncertainty, pain catastrophizing, and symptoms of depression: a comparison between adults with and without chronic pain. *Psychology, Health & Medicine*, *29*(5), 951-963. https://doi.org/10.1080/13548506.2023.2240073

Wren, A. A., Somers, Tamara J, Wright, Melissa A, Goetz, Mark C, Leary, Mark R, Fras, Anne Marie, Huh, Billy K, Rogers, Lesco L, Keefe, Francis J. (2012). Self-Compassion in Patients With Persistent Musculoskeletal Pain: Relationship of Self-Compassion to Adjustment to Persistent Pain. *Journal of Pain and Symptom Management*, *43*(4), 759-770. https://doi.org/10.1016/j.jpainsymman.2011.04.014

Yamin, J. B. (2025). Childhood Abuse and Health Outcomes in Patients With Fibromyalgia: A Cross-Sectional Exploratory Study of the Moderating Effects of Pain Catastrophizing and Mindfulness. *BMC Musculoskeletal Disorders*, *26*(1). https://doi.org/10.1186/s12891-025-08449-7

Ying Zhou, B. S. N. (2024). Relationship between pain resilience and pain catastrophizing in older patients after total knee arthroplasty: Chain-mediating effects of cognitive emotion regulation and pain management self-efficacy. *Geriatric Nursing*, *59*, 571-580. https://doi.org/10.1016/j.gerinurse.2024.08.011

Zare, H., Mohammadi, N., Motaghi, P., Afshar, H., & Pourkazem, T. (2014). Treatment effect of modified cognitive therapy based on mindfulness, catastrophizing, acceptance and pain intensity of affected patients with fibromyalgia. *Health Psychology*, *3*(4), 113-194.