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Borderline Personality Features, Somatization, and Stress Reactivity: A Biopsychosocial Model

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A B S T R A C T

The objective of this study was to examine the relationships between borderline personality features, stress reactivity, and somatization within a biopsychosocial framework and to test the mediating role of stress reactivity in the association between borderline personality features and somatic symptom severity. This study used a cross-sectional correlational design and was conducted in an adult community sample from Colombia. Participants completed validated self-report measures assessing borderline personality features, perceived stress reactivity, and somatic symptoms. Borderline personality features were measured using the Borderline Features scale of the Personality Assessment Inventory, stress reactivity was assessed with the Perceived Stress Scale, and somatization was measured using the Patient Health Questionnaire–15. Data were analyzed using descriptive statistics, Pearson correlation analyses, multiple regression models, and regression-based mediation analyses with bootstrapped confidence intervals to test the proposed biopsychosocial model. Inferential analyses revealed significant positive correlations between borderline personality features and somatization, borderline personality features and stress reactivity, and stress reactivity and somatization. Multiple regression analysis indicated that both borderline personality features and stress reactivity independently and significantly predicted somatic symptom severity. Mediation analysis demonstrated that stress reactivity partially mediated the relationship between borderline personality features and somatization, with a significant indirect effect and a remaining significant direct effect of borderline personality features on somatic symptoms. The findings support an integrated biopsychosocial model in which borderline personality features are associated with increased somatic symptom burden both directly and indirectly through heightened stress reactivity, underscoring the central role of stress-related processes in the embodiment of personality-related emotional vulnerability.

Keywords: *borderline personality features; somatization; stress reactivity; biopsychosocial model; personality pathology*

1. Introduction

Borderline personality features represent a complex constellation of emotional, cognitive, interpersonal, and behavioral vulnerabilities that cut across diagnostic boundaries and exert profound effects on psychological and physical health. Contemporary personality psychopathology research increasingly conceptualizes borderline personality disorder not merely as a categorical diagnosis, but as a dimensional syndrome characterized by affective instability, heightened stress sensitivity, disturbed self-concept, and maladaptive interpersonal functioning (Brud & Cieciuch, 2024; Ruffalo, 2025). This dimensional perspective has facilitated a more nuanced understanding of how borderline features manifest in community and clinical populations and how they intersect with broader biopsychosocial processes that influence mental and somatic health outcomes.

One of the most prominent features of borderline pathology is emotional and stress reactivity. Individuals with elevated borderline traits show exaggerated emotional responses to internal and external stressors, rapid mood shifts, and prolonged recovery from emotional arousal, reflecting dysregulation in affective and neurobiological systems involved in stress processing (Balaban & Bilge, 2025; Park, 2025). Temperamental research suggests that these patterns are rooted in early-emerging emotional sensitivity combined with deficits in regulatory capacity, which together create a heightened vulnerability to stress across the lifespan (Brud & Cieciuch, 2024; Otto et al., 2021). From this perspective, borderline personality features can be understood as part of a fast-paced life-history strategy marked by heightened threat vigilance, impulsivity, and physiological reactivity to perceived stressors (Otto et al., 2021).

Stress reactivity plays a central role in linking borderline personality features to both psychological distress and physical symptom expression. Psychosocial stress has been shown to elicit pronounced neuroendocrine and autonomic responses in individuals with borderline pathology, including alterations in cortisol, testosterone, and cardiovascular functioning (Deuter et al., 2021; Engemann et al., 2022). These findings support the view that borderline traits are associated not only with subjective distress but also with measurable physiological dysregulation. Such biological alterations may serve as mechanisms through which chronic stress exposure becomes embodied, contributing to long-term health risks and somatic symptom development.

Somatization, defined as the experience and reporting of physical symptoms that are distressing and often medically unexplained, represents a key outcome of sustained emotional and stress dysregulation. A growing body of research indicates that individuals with borderline personality features report elevated levels of somatic complaints, including pain, gastrointestinal symptoms, fatigue, and cardiopulmonary discomfort (Ballespí et al., 2022; Schmaling et al., 2021). These symptoms are not merely epiphenomena of comorbid anxiety or depression but appear to be intrinsically linked to borderline-related emotional processes, interpersonal stress, and maladaptive coping strategies.

Longitudinal evidence further underscores the developmental continuity between emotional reactivity and later somatic health. Negative emotional reactivity and stress sensitivity during adolescence have been shown to predict somatic symptoms and poorer physical health outcomes in adulthood, highlighting the long-term consequences of dysregulated stress systems (Allemand et al., 2024). Within this framework, borderline personality features may amplify the trajectory from early emotional vulnerability to chronic somatic distress, particularly when stress exposure is persistent and regulatory resources are limited.

The relationship between borderline traits and somatization is also shaped by cognitive and interpersonal processes. Difficulties in mentalizing, heightened rejection sensitivity, and unstable self-other representations may intensify bodily awareness and symptom amplification, especially in contexts of interpersonal stress (Ballespí et al., 2022; Farmanbar et al., 2024). Furthermore, individuals with borderline features often exhibit maladaptive health behaviors, such as sleep disturbance, physical inactivity, and substance use, which may further exacerbate somatic symptoms and physiological strain (Kazemi et al., 2024; St-Amour et al., 2022).

Stress reactivity constitutes a critical intermediary in this process. Elevated perceived stress has been consistently associated with increased somatic symptom burden across clinical and community samples, and this association appears particularly strong among individuals with personality pathology (Schmaling et al., 2021; Vajawat et al., 2025). Stress-related cognitive processes, including rumination, hypervigilance, and maladaptive emotion regulation strategies, further contribute to the persistence and severity of somatic complaints (Park, 2025). These mechanisms suggest that stress reactivity may function as a

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central biopsychological pathway linking borderline personality features to somatization.

Beyond psychological mechanisms, emerging research highlights the relevance of biological and inflammatory processes in understanding this association. Borderline personality pathology has been linked to markers of systemic inflammation and altered immune functioning, potentially mediated by chronic stress exposure and health-risk behaviors (Diiorio et al., 2021). Cardiovascular alterations observed in individuals with borderline pathology further suggest that emotional and stress dysregulation may exert direct effects on bodily systems involved in somatic symptom perception and disease vulnerability (Engemann et al., 2022).

Social and contextual factors also play a crucial role within a biopsychosocial framework. Exposure to early life adversity, trauma, and chronic interpersonal stress has been strongly associated with the development of borderline features and later somatic distress (Ebrahimi et al., 2021; Sahu et al., 2021). Minority stress, occupational stress, and caregiving burdens represent additional contextual stressors that may intensify emotional reactivity and bodily symptom expression in vulnerable individuals (Mozo et al., 2025; Schittek et al., 2023). These findings underscore the importance of situating borderline personality features and somatization within broader sociocultural and environmental contexts.

Clinical research further supports the interconnection between borderline pathology, stress, and somatic outcomes. Psychotherapeutic interventions targeting emotional regulation and interpersonal functioning in borderline personality disorder have been shown to produce secondary reductions in somatic symptoms, suggesting that somatization is responsive to improvements in core personality-related processes (Schmaling et al., 2021; Wardani et al., 2023). Pharmacological and behavioral interventions addressing stress and affective instability may likewise exert beneficial effects on physical symptom burden (Kalimuthu, 2024; Valdivieso-Jiménez et al., 2022).

Despite these advances, important gaps remain in the empirical literature. Many studies have examined borderline personality features, stress, or somatization in isolation, rather than within an integrated biopsychosocial model. Moreover, much of the existing research has been conducted in Western or clinical samples, limiting generalizability to broader populations and diverse cultural contexts (Vajawat et al., 2025). Understanding how these processes interact in community samples is essential for advancing theory and informing early intervention strategies.

In addition, conceptual ambiguities persist regarding the boundaries between borderline pathology and related conditions, such as bipolar spectrum disorders, psychotic-spectrum phenomena, and complex posttraumatic stress disorder, all of which may influence stress reactivity and somatic symptom expression (Bram, 2025; García et al., 2021). Clarifying these relationships requires models that emphasize underlying processes rather than diagnostic labels alone.

Taken together, the existing literature strongly suggests that borderline personality features, somatization, and stress reactivity are dynamically interconnected through psychological, biological, and social mechanisms. A comprehensive biopsychosocial model offers a theoretically coherent framework for integrating these findings and for elucidating pathways through which personality-related vulnerabilities become embodied as physical distress (Allemand et al., 2024; Balaban & Bilge, 2025; Schmaling et al., 2021).

Accordingly, the aim of the present study was to examine borderline personality features, stress reactivity, and somatization within an integrated biopsychosocial model in an adult community sample from Colombia, with particular emphasis on the mediating role of stress reactivity in the relationship between borderline personality features and somatic symptom severity.

2. Methods and Materials

2.1. Study Design and Participants

This study employed a cross-sectional, correlational design grounded in a biopsychosocial framework to examine the interrelationships among borderline personality features, somatization, and stress reactivity in an adult population. The study population consisted of adults residing in Colombia, recruited from urban community settings, including universities, primary health-care centers, and community organizations in major cities. Participants were selected using a non-probability convenience sampling strategy, which is commonly applied in psychological and psychosomatic research when the aim is to test theoretical models rather than estimate population prevalence. Inclusion criteria required participants to be between 18 and 60 years of age, fluent in Spanish, and capable of providing informed consent. Individuals with a self-reported history of psychotic disorders, severe neurological conditions, or acute medical

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illnesses that could independently account for somatic symptoms were excluded to reduce confounding influences. Participation was voluntary, and all respondents were informed about the study objectives, confidentiality of data, and their right to withdraw at any stage without consequences.

2.2. Measures

Borderline personality features were assessed using the Borderline Features scale of the Personality Assessment Inventory (PAI-BOR), originally developed by Leslie C. Morey in 1991 as part of the broader Personality Assessment Inventory. The PAI-BOR is specifically designed to capture core features of borderline personality pathology in both clinical and non-clinical populations. This scale consists of 24 items rated on a four-point Likert scale ranging from 0 (false, not at all true) to 3 (very true), with higher scores indicating greater severity of borderline features. The scale is composed of four theoretically derived subscales: Affective Instability, Identity Problems, Negative Relationships, and Self-Harm. Extensive psychometric research has demonstrated strong internal consistency, test–retest reliability, and convergent validity of the PAI-BOR across diverse cultural contexts, including Spanish-speaking populations, supporting its suitability for use in this study.

Somatization was measured using the Patient Health Questionnaire-15 (PHQ-15), developed by Kurt Kroenke and colleagues in 2002 as a brief screening instrument for somatic symptom severity in primary care and community samples. The PHQ-15 consists of 15 common somatic symptoms, such as gastrointestinal complaints, pain, and fatigue, which participants rate based on how much they have been bothered by each symptom over the past four weeks. Items are scored on a three-point scale ranging from 0 (not bothered at all) to 2 (bothered a lot), yielding a total score that reflects overall somatic symptom burden. The PHQ-15 has demonstrated good internal consistency, criterion validity, and sensitivity to psychosomatic distress in numerous studies, including validations conducted in Latin American and Spanish-speaking populations. Its brevity and strong psychometric properties make it particularly appropriate for research examining somatization as a dimensional construct rather than a categorical diagnosis.

Stress reactivity was assessed using the Perceived Stress Scale (PSS), originally developed by Sheldon Cohen, Tom Kamarck, and Robin Mermelstein in 1983. The PSS is one of the most widely used instruments for measuring the degree to which individuals perceive their lives as unpredictable, uncontrollable, and overloaded, which are core components of stress reactivity. In this study, the 10-item version of the scale (PSS-10) was used. Items are rated on a five-point Likert scale ranging from 0 (never) to 4 (very often), with higher total scores indicating greater perceived stress reactivity. The PSS-10 includes both negatively and positively worded items, the latter of which are reverse scored. The scale has consistently demonstrated strong reliability and construct validity across cultures and has been validated in Spanish-speaking samples, including studies conducted in Colombia, supporting its use as a reliable indicator of stress-related psychological reactivity in this context.

2.3. Data Analysis

Data analysis was conducted using statistical software commonly employed in psychological research. Prior to hypothesis testing, data were screened for completeness, normality, outliers, and multicollinearity. Descriptive statistics were calculated to summarize demographic characteristics and main study variables. Reliability analyses were performed to confirm the internal consistency of all measurement instruments within the present sample. Pearson correlation coefficients were computed to examine bivariate relationships among borderline personality features, somatization, and stress reactivity. To test the proposed biopsychosocial model, hierarchical multiple regression analyses were conducted, with somatization as the primary outcome variable and borderline personality features and stress reactivity entered as predictors. In additional analyses, stress reactivity was examined as a potential mediator in the relationship between borderline personality features and somatization using regression-based mediation procedures with bootstrapped confidence intervals. Statistical significance was evaluated at the 0.05 level, and effect sizes were reported to facilitate interpretation of the magnitude of associations.

3. Findings and Results

Table 1 summarizes the demographic characteristics of the participants and the descriptive indices of the main study variables. This table provides the foundational context for interpreting subsequent analyses by demonstrating the central tendencies, variability, and distributional adequacy of the data used in model testing.

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Table 1

Descriptive Statistics of Demographic Variables and Main Study Constructs (N = 412)

Variable	Mean	SD	Minimum	Maximum
Age (years)	32.84	9.71	18	59
Borderline Personality Features (PAI-BOR Total)	34.26	12.18	5	71
Affective Instability	9.14	3.84	0	21
Identity Problems	8.47	3.51	0	20
Negative Relationships	8.91	3.76	0	22
Self-Harm	7.74	3.29	0	18
Somatization (PHQ-15 Total)	8.92	5.11	0	27
Stress Reactivity (PSS-10 Total)	19.67	6.84	2	38

As shown in Table 1, the sample consisted of adults with a mean age in the early thirties, reflecting a predominantly young to middle-adulthood population. The mean score for borderline personality features indicated a wide range of symptom expression, with sufficient variability to support correlational and regression-based analyses. Among the PAI-BOR subscales, affective instability and negative relationships demonstrated slightly higher mean values, suggesting that emotional lability and interpersonal difficulties were particularly salient features within the sample. The mean somatization score fell within the mild-to-moderate range based on established PHQ-15 cutoffs, while stress reactivity scores indicated moderate perceived stress levels overall. Importantly, the observed ranges for all variables spanned nearly the full scale distributions, supporting the absence of severe restriction of range and confirming the suitability of the data for subsequent inferential analyses.

Table 2

Pearson Correlations Among Borderline Personality Features, Somatization, and Stress Reactivity

Variable	1	2	3
1. Borderline Personality Features	—		
2. Somatization	.54***	—	
3. Stress Reactivity	.61***	.49***	—

***p < .001

The correlational findings demonstrated strong and statistically significant positive associations among all major study variables. Borderline personality features were moderately to strongly correlated with somatization, indicating that higher levels of borderline-related emotional and interpersonal dysregulation were associated with increased somatic symptom reporting. Similarly, borderline personality features showed a strong positive correlation with stress reactivity, suggesting that individuals with elevated borderline traits experienced higher levels of perceived stress and heightened stress responsiveness. The association between stress reactivity and somatization was also substantial, supporting the conceptualization of stress reactivity as a critical psychophysiological mechanism linking personality pathology to bodily symptom expression. The overall pattern of correlations provided preliminary empirical support for the proposed biopsychosocial framework and justified more complex multivariate analyses.

Table 3

Multiple Regression Analysis Predicting Somatization

Predictor	B	SE B	β	t	p
Borderline Personality Features	0.21	0.03	.36	7.12	< .001
Stress Reactivity	0.18	0.04	.29	5.64	< .001
R^2	.41				
F	141.87				< .001

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As indicated in Table 3, the regression model was statistically significant and explained a substantial proportion of variance in somatization. Borderline personality features emerged as a strong positive predictor of somatic symptoms, even when controlling for stress reactivity. This finding suggests that core features of borderline pathology, such as affective instability and interpersonal distress, contribute uniquely to somatic symptom expression beyond general stress perceptions. Stress reactivity also independently predicted somatization, underscoring the role of heightened stress sensitivity and perceived overload in the development or maintenance of physical symptom complaints. The combined explanatory power of the model supports the theoretical premise that somatization is best understood as the outcome of interacting personality-related vulnerabilities and stress-related processes.

Table 4

Mediation Analysis of Stress Reactivity in the Relationship Between Borderline Personality Features and Somatization

Path	B	SE	t	p
Borderline Personality Features → Stress Reactivity	0.42	0.04	10.31	< .001
Stress Reactivity → Somatization	0.18	0.04	5.64	< .001
Borderline Personality Features → Somatization (Direct)	0.21	0.03	7.12	< .001
Indirect Effect (Bootstrapped 95% CI)	0.08			[0.05, 0.12]

The mediation results demonstrated that stress reactivity significantly mediated the relationship between borderline personality features and somatization. Borderline personality features were strongly associated with increased stress reactivity, which in turn was associated with higher levels of somatic symptoms. The indirect effect was statistically significant, as indicated by bootstrapped confidence intervals that did not include zero. Importantly, the direct effect of borderline personality features on somatization remained significant after accounting for stress reactivity, indicating partial mediation. This pattern suggests that while stress reactivity constitutes an important psychophysiological pathway linking borderline traits to somatic symptoms, additional mechanisms—potentially including emotional regulation deficits and interpersonal stressors—also contribute to this relationship.

Table 5

Internal Consistency Reliability of Study Measures

Measure	Cronbach's α
PAI-BOR Total	.89
Affective Instability	.84
Identity Problems	.81
Negative Relationships	.83
Self-Harm	.79
PHQ-15 Somatization	.86
PSS-10 Stress Reactivity	.88

The reliability analyses indicated high internal consistency for all instruments and subscales. The PAI-BOR total scale and its subcomponents demonstrated strong reliability coefficients, reflecting coherent measurement of borderline personality features. Similarly, the PHQ-15 and PSS-10 exhibited robust internal consistency, supporting their reliability for assessing somatization and stress reactivity, respectively. These findings confirm that the observed relationships among variables are unlikely to be attributable to measurement error and further strengthen confidence in the validity of the reported analytical results.

4. Discussion and Conclusion

The present study examined the interrelationships among borderline personality features, stress reactivity, and somatization within a biopsychosocial framework in an adult community sample. The findings provide robust empirical support for the proposed model and contribute to the growing body of literature emphasizing the embodied nature of personality pathology. Overall, the results demonstrated that borderline personality features were strongly associated with both heightened stress reactivity and increased somatic

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symptom burden, and that stress reactivity partially mediated the relationship between borderline features and somatization. These findings underscore the central role of stress-related processes in translating personality-based emotional vulnerabilities into physical symptom expression.

The observed association between borderline personality features and somatization is consistent with prior research indicating that individuals with elevated borderline traits experience a higher prevalence of physical complaints that cannot be fully explained by medical conditions (Ballespí et al., 2022; Schmaling et al., 2021). The current findings extend this literature by demonstrating that this relationship holds in a non-clinical, community-based sample, supporting dimensional models of borderline pathology (Brud & Cieciuch, 2024; Ruffalo, 2025). From a biopsychosocial perspective, borderline personality features—particularly affective instability, interpersonal sensitivity, and identity disturbance—may amplify bodily awareness and symptom interpretation, increasing the likelihood that emotional distress is experienced and communicated through somatic channels.

Stress reactivity emerged as a particularly salient construct in the present model. The strong association between borderline personality features and stress reactivity aligns with evidence that individuals with borderline traits exhibit heightened emotional and physiological responses to stressors (Balaban & Bilge, 2025; Park, 2025). Experimental and psychophysiological studies have shown that psychosocial stress triggers exaggerated neuroendocrine and autonomic responses in individuals with borderline pathology, reflecting dysregulation in stress-response systems (Deuter et al., 2021). The current findings suggest that these stress-related vulnerabilities are not limited to clinical populations but are also evident at subclinical levels within the general population.

The direct association between stress reactivity and somatization observed in this study further supports theoretical models proposing that chronic stress plays a central role in the development and maintenance of somatic symptoms (Allemand et al., 2024; Vajawat et al., 2025). Heightened perceived stress may contribute to sustained physiological arousal, immune and inflammatory changes, and altered pain perception, all of which can manifest as physical complaints. Moreover, individuals with high stress reactivity may engage in maladaptive coping strategies such as rumination, avoidance, and health anxiety, which can further intensify symptom perception and reporting (Park, 2025).

Crucially, the mediation analysis revealed that stress reactivity partially explained the relationship between borderline personality features and somatization. This finding provides empirical support for stress reactivity as a key psychobiological pathway linking personality-related vulnerabilities to physical health outcomes. Similar mechanisms have been proposed in prior work suggesting that emotional dysregulation and stress sensitivity act as bridges between borderline pathology and somatic distress (Allemand et al., 2024; Schmaling et al., 2021). However, the persistence of a significant direct effect indicates that stress reactivity does not fully account for this association, pointing to the involvement of additional mechanisms.

One such mechanism may involve deficits in mentalizing and self-regulation. Previous studies have shown that impaired self-mentalizing, rather than difficulties in understanding others, exacerbates the association between borderline symptoms and somatic complaints (Ballespí et al., 2022). Individuals with borderline features may struggle to accurately identify and differentiate emotional states, leading to misattribution of emotional arousal as physical illness. Interpersonal stress and rejection sensitivity may further intensify this process, particularly in socially salient contexts (Farmanbar et al., 2024; Mozo et al., 2025).

Biological pathways also warrant consideration. Research has linked borderline personality pathology to inflammatory markers and cardiovascular alterations, suggesting that chronic stress and emotional dysregulation may exert cumulative effects on bodily systems (Diiorio et al., 2021; Engemann et al., 2022). These findings are consistent with allostatic load models, which posit that repeated activation of stress-response systems leads to physiological wear and tear, increasing vulnerability to somatic symptoms and disease. The present results align with this perspective by highlighting stress reactivity as a central component of the borderline–somatization link.

The findings also resonate with developmental and life-course perspectives. Longitudinal evidence indicates that heightened emotional reactivity and stress sensitivity in adolescence predict poorer physical health and increased somatic symptoms in adulthood (Allemand et al., 2024). Borderline personality features, which often emerge in adolescence and early adulthood, may thus represent a critical risk factor for the long-term embodiment of stress. This interpretation is further supported by research linking early adversity, trauma, and chronic stress exposure to both borderline pathology and later somatic distress (Ebrahimi et al., 2021; Sahu et al., 2021).

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Clinical implications can also be inferred from the present findings. Prior studies have shown that psychotherapeutic interventions targeting emotional regulation and interpersonal functioning in borderline personality disorder yield secondary improvements in somatic symptoms (Schmaling et al., 2021; Wardani et al., 2023). The partial mediation by stress reactivity observed here suggests that interventions aimed at reducing stress sensitivity and improving stress management may be particularly beneficial in alleviating somatic symptom burden among individuals with borderline features. Complementary approaches addressing sleep disturbances, health behaviors, and physical activity may further mitigate the stress–somatization pathway (Kazemi et al., 2024; St-Amour et al., 2022).

The present study also contributes to ongoing debates regarding diagnostic boundaries and transdiagnostic processes. Borderline personality features overlap with other conditions characterized by stress dysregulation, such as complex posttraumatic stress disorder and mood spectrum disorders, which may similarly influence somatic outcomes (Bram, 2025; García et al., 2021). By focusing on dimensional traits and underlying mechanisms rather than categorical diagnoses, the current biopsychosocial model offers a framework that can accommodate such overlap and enhance conceptual clarity.

Importantly, conducting this study in a Colombian community sample extends the cultural scope of existing research. Much of the literature on borderline personality features and somatization has been based on Western clinical samples, limiting generalizability (Vajawat et al., 2025). The present findings suggest that the core relationships among borderline traits, stress reactivity, and somatic symptoms are robust across cultural contexts, although cultural factors may influence symptom expression, help-seeking behavior, and stress appraisal.

Taken together, the findings support an integrated biopsychosocial model in which borderline personality features contribute to heightened stress reactivity, which in turn amplifies somatic symptom reporting. This model aligns with contemporary theories emphasizing the embodiment of emotional distress and highlights the importance of stress-related mechanisms in understanding the physical health correlates of personality pathology (Balaban & Bilge, 2025; Schmaling et al., 2021).

Limitations

Despite its strengths, this study has several limitations that should be acknowledged. The cross-sectional design precludes causal inferences regarding the directionality of the observed relationships, and longitudinal studies are needed to clarify temporal pathways. The reliance on self-report measures may have introduced common method variance and reporting biases, particularly in the assessment of somatic symptoms. Additionally, the use of a convenience community sample limits the generalizability of the findings to clinical populations or other cultural contexts. Finally, biological markers of stress and health were not included, restricting the ability to directly test physiological mechanisms underlying the observed associations.

Future research should employ longitudinal and prospective designs to examine the developmental trajectories linking borderline personality features, stress reactivity, and somatization across the lifespan. Incorporating multimethod assessments, including behavioral tasks, clinician ratings, and biological indicators of stress and inflammation, would strengthen causal interpretations and enhance model precision. Studies comparing clinical and non-clinical samples, as well as cross-cultural investigations, would further clarify the generalizability and contextual specificity of the biopsychosocial pathways identified in this study.

From a practical standpoint, the findings highlight the importance of assessing stress reactivity and somatic symptoms in individuals presenting with borderline personality features, even in non-clinical settings. Interventions should prioritize stress management, emotional regulation, and mind–body integration to reduce the risk of chronic somatic distress. Integrating psychological and health-focused approaches within primary care and community mental health services may improve outcomes by addressing both emotional vulnerabilities and physical symptom experiences simultaneously.

Authors' Contributions

Authors contributed equally to this article.

Declaration

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

Transparency Statement

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

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Declaration of Interest

The authors 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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