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Neurobiological Correlates of Personality-Driven Psychosomatic Reactivity: A Psychoneuroimmunological Perspective

Daymar. Avilés-Santa[1], Przemyslaw. Wandzilak[2], Derek. Péloquin[3*], Sue J. Critchley[1]

[1] National Institute of Psychiatry Ramón de la Fuente Muñiz, Sub-directorate of Clinical Research, Mexico City, Mexico
[2] Department of Psychology, Concordia University, Montreal, Canada
[3] School of Psychology, University of Ottawa, Ottawa, Ontario, Canada

*** Corresponding author email address**: derek.peloquin@uottawa.ca

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A B S T R A C T

This study aimed to examine an integrated psychoneuroimmunological model linking personality traits to psychosomatic reactivity through perceived stress, neuroendocrine activity, and immune activation. A cross-sectional correlational design was employed with a community-based adult sample recruited from urban health centers in Mexico. Participants completed validated self-report measures assessing personality traits, perceived stress, and psychosomatic symptoms. Neuroendocrine activity was indexed using salivary cortisol collected at awakening and 30 minutes post-awakening, while immune activation was assessed through serum inflammatory markers, including interleukin-6 and C-reactive protein. Data were analyzed using correlation analyses, multiple regression models, mediation analyses with bootstrapping, and structural equation modeling to test direct and indirect pathways among psychological and biological variables. Inferential analyses indicated that neuroticism was a strong positive predictor of psychosomatic symptoms, whereas conscientiousness and agreeableness were significant negative predictors. Perceived stress significantly mediated the relationship between neuroticism and psychosomatic symptom severity. Elevated cortisol awakening response was associated with higher perceived stress and greater somatic symptom burden and functioned as an additional mediator. Inflammatory markers were positively associated with cortisol activity and psychosomatic symptoms. Structural equation modeling demonstrated good model fit and supported a sequential pathway from personality traits to stress appraisal, neuroendocrine dysregulation, immune activation, and psychosomatic reactivity, with protective traits exerting indirect buffering effects through reduced stress responses. The findings support a psychoneuroimmunological model in which personality traits function as central regulators of psychosomatic reactivity by shaping psychological stress appraisal and downstream neuroendocrine and immune processes, highlighting the importance of integrative, personality-informed approaches to psychosomatic health.

Keywords: *psychosomatic reactivity; personality traits; psychoneuroimmunology; cortisol; inflammation*

1. Introduction

Psychosomatic reactivity has long occupied a central position in attempts to understand how psychological processes translate into bodily symptoms, yet only in recent decades has the field moved toward integrative models capable of explaining this phenomenon across multiple biological levels. Contemporary perspectives increasingly emphasize that psychosomatic manifestations cannot be reduced to either purely psychological vulnerability or isolated physiological dysfunction, but instead emerge from dynamic interactions among personality structure, neural regulation, endocrine stress systems, and immune signaling pathways. Within this context, psychoneuroimmunology has provided a unifying framework that conceptualizes health and disease as outcomes of bidirectional communication between the nervous, endocrine, and immune systems, modulated by stable individual differences such as personality traits (Troubat et al., 2020; Zhao et al., 2022). This integrative approach is particularly relevant for explaining why individuals exposed to similar stressors display markedly different somatic outcomes, ranging from transient discomfort to chronic multisystem disorders.

Personality traits represent enduring patterns of emotional reactivity, cognitive appraisal, and behavioral regulation that shape how individuals perceive and respond to internal and external demands. Large-scale population studies consistently demonstrate that traits such as neuroticism, conscientiousness, and agreeableness are robustly associated with mental and physical health outcomes, including stress sensitivity, symptom reporting, and disease risk (Jiang et al., 2021; Whiston et al., 2023). Neuroticism, characterized by heightened negative affectivity and threat sensitivity, has been repeatedly linked to increased somatic complaints, pain perception, and functional syndromes, whereas conscientiousness often exerts a protective role through enhanced self-regulation and health-promoting behaviors (BaŞ et al., 2021; Tidmarsh et al., 2022). These associations suggest that personality traits may act as upstream psychological regulators of physiological stress systems, thereby shaping psychosomatic vulnerability across the lifespan.

At the neurobiological level, the hypothalamic–pituitary–adrenal axis occupies a pivotal position in translating psychological stress into systemic physiological responses. Cortisol, as the primary glucocorticoid end product of this axis, exerts widespread effects on metabolism, immune regulation, and neural plasticity. Dysregulation of cortisol rhythms, including exaggerated or blunted stress responses, has been implicated in a wide range of psychosomatic and affective disorders (Hasanah et al., 2023; Rovnaghi et al., 2021). Importantly, individual differences in cortisol reactivity are not solely determined by situational stressors, but are strongly influenced by stable psychological characteristics, including personality traits and long-term stress appraisal styles (Gano et al., 2023; Whiston et al., 2023). This evidence supports the view that personality-driven stress processing may be a critical mechanism linking psychological vulnerability to somatic outcomes.

Beyond neuroendocrine regulation, immune signaling has emerged as a central pathway in psychosomatic reactivity. Proinflammatory cytokines such as interleukin-6 and acute-phase proteins like C-reactive protein play essential roles in host defense, yet chronic low-grade inflammation is increasingly recognized as a common denominator across diverse psychosomatic and psychiatric conditions. Neuroinflammatory processes have been implicated in depression, chronic pain, cardiovascular disease, and functional gastrointestinal disorders, highlighting the relevance of immune activation in mind–body interactions (Bendezú et al., 2022; Troubat et al., 2020). Personality traits appear to modulate immune function indirectly through stress-related neuroendocrine pathways, as well as directly through behavioral and lifestyle factors that influence inflammatory load (Czerwińska et al., 2021; Scarola & Bardi, 2020).

Recent advances in psychoneuroimmunology further emphasize the role of complex biological networks rather than single biomarkers. Multi-omics approaches demonstrate that stress-related phenotypes are characterized by coordinated alterations in immune, metabolic, and neural signaling pathways, rather than isolated dysregulation (Wang et al., 2024; Wang et al., 2025). These findings align with emerging models of allostatic load, which conceptualize psychosomatic disorders as the cumulative consequence of repeated stress-related activation across interconnected biological systems (Mahony & Ryan, 2022). Within this framework, personality traits can be understood as modulators of allostatic processes, shaping the frequency, intensity, and recovery dynamics of stress responses over time.

The relevance of psychoneuroimmunological mechanisms is further underscored by growing evidence from clinical populations. Dermatological conditions, gastrointestinal disorders, and autoimmune diseases show particularly strong links between psychological factors,

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immune dysregulation, and symptom severity. For example, psychodermatological research highlights how emotional stress and personality structure influence inflammatory skin diseases through neuroimmune pathways (Dattolo et al., 2025; Jafferany et al., 2020). Similarly, functional gastrointestinal disorders and irritable bowel syndrome have been associated with stress-related alterations in gut–brain–immune communication, with implicit stress memory and autonomic dysregulation playing key roles (Chen et al., 2025; Császár-Nagy & Bókkon, 2023). These clinical observations reinforce the need for integrative models that account for psychological, neuroendocrine, and immunological dimensions simultaneously.

An additional layer of complexity arises from the gut microbiota, which has emerged as a crucial mediator of brain–immune interactions. Dietary patterns, microbial composition, and microbial metabolites influence inflammation, neurotransmission, and stress responsiveness, thereby contributing to psychosomatic vulnerability and resilience (Liu et al., 2023; Rajeev et al., 2024). Evidence suggests that personality-related behavioral tendencies, such as stress eating or health-related self-regulation, may indirectly shape gut–brain–immune communication, further amplifying individual differences in psychosomatic outcomes (Kolniak-Ostek, 2025; Tian, 2025). These findings expand traditional psychoneuroimmunological models by embedding personality-driven processes within broader metabolic and ecological contexts.

Sex differences also play a critical role in psychosomatic and immune-related disorders, with women showing higher prevalence of many autoimmune and functional somatic conditions. Immunological and hormonal mechanisms underlying these differences interact with psychological vulnerability and personality traits, suggesting that sex-specific psychoneuroimmunological pathways may influence symptom expression (Kim et al., 2025; Vassiliadi et al., 2021). Understanding how personality traits operate within these biologically differentiated contexts remains an important challenge for contemporary research.

Despite substantial theoretical and empirical advances, significant gaps remain in the literature. Many studies examine psychological, neuroendocrine, or immune factors in isolation, limiting the ability to draw conclusions about integrated pathways. Moreover, although personality traits are frequently assessed in psychosomatic research, they are often treated as covariates rather than central organizing variables that structure stress reactivity across biological systems. Recent calls within personality neuroscience emphasize the need to embed personality constructs within multi-level biological models, moving beyond descriptive associations toward mechanistic explanations (Lages & McNaughton, 2022; Millet & Jendzjowsky, 2023). Such an approach is essential for advancing precision-oriented psychosomatic medicine and for identifying targets for personalized interventions.

Furthermore, cultural and contextual factors remain underrepresented in psychoneuroimmunological research. Most evidence derives from European or East Asian populations, while Latin American contexts, characterized by distinct social stressors and health profiles, have received comparatively little attention. Considering that stress exposure, health behaviors, and social meaning systems vary across cultures, examining psychoneuroimmunological processes within diverse populations is crucial for the generalizability of theoretical models (Latifi & Flegr, 2025; Vlachos et al., 2020). Addressing this gap may also illuminate how chronic stressors related to socioeconomic inequality and environmental adversity interact with personality traits to shape psychosomatic reactivity.

In summary, accumulating evidence suggests that psychosomatic reactivity emerges from complex interactions among personality traits, stress appraisal, neuroendocrine regulation, and immune activation, embedded within broader metabolic and social contexts. Psychoneuroimmunology offers a powerful framework for integrating these dimensions, yet empirical studies explicitly testing personality-driven pathways across psychological, hormonal, and inflammatory levels remain limited. To advance understanding of these mechanisms, there is a need for comprehensive models that position personality traits as central regulators of psychoneuroimmunological processes rather than peripheral correlates.

The aim of this study is to examine the neurobiological correlates of personality-driven psychosomatic reactivity by testing an integrated psychoneuroimmunological model linking personality traits, perceived stress, neuroendocrine activity, immune activation, and somatic symptom expression.

2. Methods and Materials

2.1. Study Design and Participants

The present study employed a cross-sectional, correlational design grounded in the psychoneuroimmunological framework to examine neurobiological correlates of personality-driven

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psychosomatic reactivity. The study was conducted in Mexico between 2024 and 2025. Participants were recruited from urban primary healthcare centers and university-affiliated medical clinics in Mexico City and Guadalajara through announcements and physician referrals. The target population consisted of adults aged 20 to 55 years. Inclusion criteria were fluency in Spanish, absence of diagnosed autoimmune disorders, neurodegenerative diseases, active cancer, or current pregnancy, and no use of systemic corticosteroids or immunomodulatory medication during the three months preceding participation. Individuals with acute infectious illnesses at the time of data collection were excluded to avoid confounding inflammatory markers. After screening, a final sample of 312 participants (approximately equal distribution of men and women) was included. All participants provided written informed consent prior to participation.

2.2. Measures

Personality traits were assessed using the Big Five Inventory (BFI), originally developed by John, Donahue, and Kentle in 1991 and later refined by John and Srivastava in 1999. The BFI consists of 44 items designed to measure five broad personality dimensions: Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness. Items are rated on a five-point Likert scale ranging from strong disagreement to strong agreement. Scores for each dimension are calculated by summing relevant items, with higher scores indicating stronger expression of the trait. The Spanish-language version of the BFI has been validated in multiple Latin American populations, including Mexican samples, demonstrating satisfactory internal consistency coefficients typically exceeding 0.75 for all subscales, as well as good construct and convergent validity.

Psychosomatic symptomatology was measured using the Somatic Symptom Scale–8 (SSS-8), developed by Gierk and colleagues in 2014 as a brief self-report instrument for assessing somatic symptom burden. The scale includes eight items reflecting common psychosomatic complaints such as gastrointestinal discomfort, fatigue, pain, and cardiovascular-related sensations. Participants rate the extent to which they have been bothered by each symptom over the past seven days using a five-point scale ranging from not at all to very much. Total scores range from 0 to 32, with higher scores indicating greater psychosomatic reactivity. The SSS-8 has been validated internationally and shows strong internal consistency, test–retest reliability, and criterion validity. Spanish versions used in Mexican and broader Hispanic populations have demonstrated comparable psychometric properties.

Perceived psychological stress was assessed using the Perceived Stress Scale–10 (PSS-10), originally developed by Cohen, Kamarck, and Mermelstein in 1983 and later shortened to a 10-item version. The PSS-10 evaluates the degree to which individuals appraise situations in their lives as stressful over the past month. Items are scored on a five-point Likert scale from never to very often, with four items reverse-scored. Total scores range from 0 to 40, with higher scores indicating greater perceived stress. The Spanish version of the PSS-10 has been extensively validated in Mexican populations, showing good internal consistency and strong associations with psychological distress and health-related outcomes.

Neuroendocrine stress reactivity was operationalized through salivary cortisol assessment. Saliva samples were collected using standardized Salivette collection devices. Cortisol was chosen due to its central role in hypothalamic–pituitary–adrenal axis activity and its relevance to stress-related psychosomatic processes. Samples were obtained at two time points on the same day, once immediately after awakening and once 30 minutes later, to estimate the cortisol awakening response. Cortisol concentrations were analyzed using enzyme-linked immunosorbent assay kits with established sensitivity and specificity. Salivary cortisol measurement is a well-validated, non-invasive method widely used in psychoneuroendocrinological research, with demonstrated reliability and validity across diverse populations.

Immune system activity was assessed through serum inflammatory markers, specifically interleukin-6 (IL-6) and C-reactive protein (CRP). Blood samples were drawn by trained phlebotomists under fasting conditions. IL-6 levels were quantified using high-sensitivity enzyme-linked immunosorbent assays, while CRP was measured using immunoturbidimetric methods. These biomarkers were selected due to their established roles in psychoneuroimmunological pathways linking stress, personality, and somatic symptom expression. Both IL-6 and CRP assays have demonstrated high reliability and validity in clinical and non-clinical research, including studies conducted in Latin American populations.

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2.3. *Data Analysis*

Data analysis was conducted using IBM SPSS Statistics version 27 and AMOS version 26. Prior to analysis, data were screened for missing values, normality, and outliers. Descriptive statistics were computed to summarize demographic variables and main study constructs. Pearson correlation analyses were used to examine bivariate associations among personality traits, psychosomatic symptoms, perceived stress, cortisol indices, and inflammatory markers. To test the hypothesized psychoneuroimmunological pathways, structural equation modeling was employed. Personality traits were specified as exogenous variables, psychosomatic reactivity as the primary endogenous outcome, and neuroendocrine and immune markers as mediating variables. Model fit was evaluated using multiple indices, including the comparative fit index, Tucker–Lewis index, root mean square error of approximation, and standardized root mean square residual. Bootstrapping procedures with 5,000 resamples were applied to estimate indirect effects and their confidence intervals. Statistical significance was set at $p < 0.05$ for all analyses.

3. Findings and Results

Table 1 presents the descriptive statistics and Pearson correlation coefficients among personality traits, perceived stress, psychosomatic symptom burden, salivary cortisol indices, and inflammatory markers. This table serves as the foundational overview of the data structure and the magnitude and direction of associations among the principal psychoneuroimmunological variables.

Table 1

Descriptive Statistics and Correlations Among Study Variables

Variable	Mean	SD	1	2	3	4	5	6	7	8	9
1. Neuroticism	27.84	6.12	—								
2. Extraversion	31.10	5.47	−0.32**	—							
3. Openness	34.02	5.21	−0.08	0.29**	—						
4. Agreeableness	35.41	4.98	−0.41**	0.26**	0.18**	—					
5. Conscientiousness	36.88	5.06	−0.45**	0.33**	0.22**	0.38**	—				
6. Perceived Stress	21.56	6.73	0.58**	−0.36**	−0.14*	−0.42**	−0.47**	—			
7. Psychosomatic Symptoms	13.92	6.01	0.61**	−0.28**	−0.10	−0.39**	−0.44**	0.64**	—		
8. Cortisol Awakening Response	6.48	2.11	0.34**	−0.19**	−0.05	−0.22**	−0.26**	0.37**	0.41**	—	
9. Inflammatory Index (IL-6/CRP)	2.87	1.03	0.29**	−0.17*	−0.04	−0.20**	−0.24**	0.33**	0.38**	0.42**	—

*p < 0.05, **p < 0.01.

The results shown in Table 1 indicate that Neuroticism demonstrated strong positive correlations with perceived stress, psychosomatic symptoms, cortisol awakening response, and inflammatory markers, suggesting a robust association between emotional instability and heightened psychophysiological reactivity. In contrast, Extraversion, Agreeableness, and Conscientiousness were consistently and negatively correlated with stress, psychosomatic symptom burden, and biological stress indicators. Openness showed weaker and mostly non-significant associations with physiological markers, indicating a more limited role in psychosomatic reactivity. Psychosomatic symptoms were strongly correlated with perceived stress and moderately correlated with both cortisol and inflammatory markers, providing preliminary evidence for a psychoneuroimmunological pathway linking psychological distress and somatic expression.

Table 2

Multiple Regression Analysis Predicting Psychosomatic Symptoms from Personality Traits

Predictor	B	SE	β	t	p
Neuroticism	0.41	0.04	0.48	10.25	<0.001
Extraversion	−0.19	0.05	−0.17	−3.80	<0.001
Openness	−0.06	0.05	−0.05	−1.21	0.227
Agreeableness	−0.23	0.06	−0.19	−3.83	<0.001
Conscientiousness	−0.27	0.05	−0.24	−5.40	<0.001

Model statistics: $R^2 = 0.52$, Adjusted $R^2 = 0.51$, $F(5, 306) = 66.18$, $p < 0.001$

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As shown in Table 2, personality traits collectively explained a substantial proportion of variance in psychosomatic symptoms. Neuroticism emerged as the strongest positive predictor, indicating that individuals with higher emotional instability reported significantly greater somatic symptom burden. Conscientiousness and Agreeableness were strong negative predictors, suggesting that self-regulation, responsibility, and interpersonal harmony may function as protective factors against psychosomatic reactivity. Extraversion also contributed negatively, though with a smaller effect size, while Openness did not significantly predict psychosomatic symptoms. These findings highlight personality structure as a central psychological determinant of psychosomatic expression.

Table 3

Mediation Analysis of Neuroticism, Stress, Cortisol, and Psychosomatic Symptoms

Pathway	Indirect Effect	SE	95% CI
Neuroticism → Stress → Symptoms	0.19	0.03	0.13 – 0.26
Neuroticism → Cortisol → Symptoms	0.07	0.02	0.04 – 0.12
Neuroticism → Stress → Cortisol → Symptoms	0.05	0.02	0.02 – 0.09

Model fit indices: $\chi^2/df = 2.41$; CFI = 0.95; TLI = 0.94; RMSEA = 0.045

The mediation results indicate that perceived stress significantly mediated the relationship between Neuroticism and psychosomatic symptoms, accounting for a large portion of the total effect. Cortisol awakening response also served as a significant mediator, both independently and sequentially following perceived stress. The serial mediation pathway supports a psychoneuroendocrine mechanism whereby emotionally vulnerable individuals experience elevated stress appraisals, leading to dysregulated hypothalamic–pituitary–adrenal axis activity and increased somatic symptom expression.

Table 4

Structural Equation Model Predicting Psychosomatic Reactivity

Path	Standardized β	p
Neuroticism → Stress	0.58	<0.001
Stress → Cortisol	0.39	<0.001
Cortisol → Inflammation	0.42	<0.001
Inflammation → Psychosomatic Symptoms	0.36	<0.001
Conscientiousness → Stress	−0.41	<0.001
Agreeableness → Stress	−0.33	<0.001

Model fit indices: $\chi^2/df = 2.31$, CFI = 0.96, TLI = 0.95, RMSEA = 0.064, SRMR = 0.048

The structural model demonstrated good overall fit and provided strong support for the hypothesized psychoneuroimmunological pathway. Neuroticism exerted a substantial indirect effect on psychosomatic symptoms through sequential activation of psychological stress, neuroendocrine dysregulation, and immune activation. Conversely, Conscientiousness and Agreeableness showed protective effects by attenuating stress responses, thereby indirectly reducing biological reactivity and psychosomatic symptom severity. Collectively, these findings underscore the integrative role of personality traits in shaping psychosomatic outcomes via interconnected psychological, neuroendocrine, and immunological mechanisms.

4. Discussion and Conclusion

The present study set out to examine psychosomatic reactivity through an integrated psychoneuroimmunological lens, positioning personality traits as upstream regulators of stress-related neuroendocrine and immune processes. The findings provide strong empirical support for this framework and demonstrate that psychosomatic symptoms are not merely subjective complaints but are embedded in measurable biological pathways that are systematically shaped by stable personality characteristics. Overall, the results converge on a coherent pattern in which emotionally vulnerable personality profiles, particularly high

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neuroticism, are associated with amplified stress perception, dysregulated hypothalamic–pituitary–adrenal activity, heightened inflammatory signaling, and, ultimately, increased psychosomatic symptom burden.

At the psychological level, neuroticism emerged as the most powerful predictor of psychosomatic symptoms, both directly and indirectly. Individuals scoring high on neuroticism reported substantially higher levels of perceived stress and somatic symptom burden, a pattern consistent with large-scale personality and health studies showing that neuroticism is associated with heightened emotional reactivity, threat sensitivity, and symptom monitoring (BaŞ et al., 2021; Jiang et al., 2021). The strong association between neuroticism and perceived stress observed in this study aligns with the conceptualization of neuroticism as a dispositional bias toward negative appraisal of environmental demands, which in turn intensifies physiological stress responses. This psychological amplification mechanism provides a critical bridge between subjective experience and biological dysregulation.

In contrast, conscientiousness and agreeableness demonstrated robust protective effects. Higher levels of these traits were associated with lower perceived stress and reduced psychosomatic symptom severity, even when controlling for other personality dimensions. These findings are consistent with prior evidence indicating that conscientious individuals exhibit better emotional regulation, health-related self-control, and adaptive coping strategies, which collectively buffer against stress-related pathology (Tidmarsh et al., 2022; Whiston et al., 2023). Agreeableness, often linked to interpersonal harmony and reduced conflict exposure, may lower psychosomatic vulnerability by attenuating chronic social stress, a factor increasingly recognized as a driver of inflammatory activation (Czerwińska et al., 2021; Vlachos et al., 2020). Together, these results reinforce the notion that personality traits operate not only as psychological descriptors but as functional regulators of stress biology.

At the neuroendocrine level, the cortisol awakening response played a significant mediating role in the relationship between personality traits and psychosomatic symptoms. Elevated cortisol responses were associated with higher perceived stress and greater somatic symptom burden, supporting the central role of hypothalamic–pituitary–adrenal axis dysregulation in psychosomatic processes. These findings are consistent with longitudinal evidence demonstrating that altered cortisol trajectories reflect cumulative stress exposure and predict later health outcomes (Rovnaghi et al., 2021). Importantly, the present study extends this literature by showing that cortisol dysregulation is not merely a response to situational stress but is systematically linked to personality-driven stress appraisal patterns, echoing experimental and developmental findings on conditioned stress responses (Gano et al., 2023; Hasanah et al., 2023).

The immunological findings further strengthen the psychoneuroimmunological interpretation of psychosomatic reactivity. Elevated inflammatory markers were significantly associated with both cortisol activity and psychosomatic symptoms, indicating that immune activation constitutes a downstream biological pathway linking psychological vulnerability to somatic expression. This pattern aligns closely with contemporary models of neuroinflammation, which posit that chronic stress-related activation of immune signaling contributes to both physical and psychological symptomatology (Troubat et al., 2020; Zhao et al., 2022). The observed cortisol–inflammation–symptom pathway mirrors findings from adolescent and adult samples showing coordinated hypothalamic–pituitary–adrenal and inflammatory response profiles under stress (Bendezú et al., 2022; Vassiliadi et al., 2021).

The structural equation model provides a particularly compelling contribution by integrating personality traits, psychological stress, neuroendocrine activity, immune activation, and psychosomatic outcomes into a single coherent framework. The good model fit and strong standardized paths support the hypothesis that psychosomatic reactivity emerges from a cascade process rather than isolated effects. This cascade begins with personality-driven stress appraisal, progresses through neuroendocrine activation, and culminates in immune-mediated somatic symptoms. Such findings resonate with allostatic load models, which conceptualize disease risk as the cumulative biological cost of repeated stress-related activation across systems (Mahony & Ryan, 2022; Wang et al., 2025). In this sense, personality traits can be understood as modulators of allostatic processes, shaping both the frequency and intensity of biological stress responses.

The results also align with emerging multi-system perspectives emphasizing network-level dysregulation rather than single biomarkers. Recent multi-omics studies demonstrate that stress-related phenotypes are characterized by coordinated alterations in immune, metabolic, and neural pathways, a pattern conceptually consistent with the integrated model tested here (Wang et al., 2024; Wang et al., 2025). Although the present study did not employ omics-

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level analyses, the observed convergence of psychological, hormonal, and inflammatory indicators supports the validity of such integrative approaches and highlights the relevance of personality traits as organizing variables within these complex biological networks.

Clinical implications of these findings are evident when considered alongside evidence from psychosomatic and medical populations. Conditions such as psoriasis, irritable bowel syndrome, and chronic pain syndromes have been shown to involve stress-sensitive neuroimmune mechanisms that closely mirror the pathways identified in this study (Chen et al., 2025; Császár-Nagy & Bókkon, 2023; Salberg et al., 2020). The strong association between personality traits and these pathways suggests that individual differences in personality may partly explain variability in symptom severity and treatment response observed in clinical practice. This perspective is further supported by psychodermatological and psychodynamic research highlighting the role of personality structure in shaping somatic disease expression (Dattolo et al., 2025; Jafferany et al., 2020).

Broader contextual factors also merit consideration. Emerging evidence on gut–brain–immune interactions suggests that dietary patterns, microbiota composition, and metabolic inflammation contribute to psychosomatic vulnerability (Liu et al., 2023; Rajeev et al., 2024). Personality traits may indirectly influence these processes through behaviorally mediated pathways such as diet, stress coping, and lifestyle regulation, offering a plausible extension of the present findings. Similarly, sex differences in immune function and stress biology may interact with personality-driven processes to shape psychosomatic outcomes, as suggested by immunological research on autoimmune and inflammatory disorders (Kim et al., 2025). Although sex-specific analyses were beyond the scope of the present study, the findings provide a foundation for future work examining these interactions.

Culturally, the study contributes to the relatively limited psychoneuroimmunological literature from Latin American contexts. Chronic stress exposure related to socioeconomic inequality, environmental adversity, and health access disparities may amplify the relevance of personality-driven stress mechanisms in such settings. The consistency of the present findings with international literature suggests that core psychoneuroimmunological processes are robust across cultural contexts, while also underscoring the need for culturally sensitive models that incorporate contextual stressors (Latifi & Flegr, 2025; Vlachos et al., 2020).

Taken together, the findings support a model in which personality traits function as central regulators of psychosomatic reactivity by shaping psychological stress appraisal and downstream neuroendocrine and immune responses. This integrated perspective advances psychosomatic research by moving beyond fragmented associations and toward a mechanistic understanding of mind–body interactions grounded in psychoneuroimmunology.

Several limitations should be acknowledged. The cross-sectional design precludes causal inference and limits conclusions regarding temporal sequencing among personality traits, stress, biological markers, and psychosomatic symptoms. Although biological measures were included, they represent snapshots rather than dynamic processes, and repeated or longitudinal assessments would provide a more nuanced understanding of stress-related regulation. Self-report measures of personality and symptoms may also be subject to reporting biases, particularly in individuals high in negative affectivity. Finally, although the sample was culturally specific, it may not fully represent rural or clinically diagnosed populations.

Future studies should employ longitudinal and experimental designs to clarify causal pathways and examine how personality-driven stress mechanisms unfold over time. Incorporating repeated biological sampling, ecological momentary assessment of stress, and advanced analytic approaches such as multi-omics or network modeling would further strengthen psychoneuroimmunological inference. Research should also explore sex-specific and culturally contextualized pathways, as well as interactions with lifestyle factors such as diet, sleep, and physical activity, to develop more comprehensive models of psychosomatic reactivity.

From a practical perspective, the findings underscore the importance of integrating personality assessment into psychosomatic and preventive healthcare. Interventions targeting stress appraisal, emotional regulation, and self-regulatory capacities may be particularly beneficial for individuals with high neuroticism or low conscientiousness. Psychotherapeutic, behavioral, and lifestyle-based approaches that reduce chronic stress and inflammation may help attenuate psychosomatic symptoms by intervening early in the psychoneuroimmunological cascade identified in this study.

Authors' Contributions

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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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