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Prediction of Psychosomatic Symptoms Based on an Invalidating Environment with the Mediating Role of Alexithymia in Individuals with Generalized Anxiety Disorder

Zeinab. Hajirahimkhan[1], Seifollah. Rahmani[2*], Mahmoud. Goudarzi[3]

1 PhD student in Psychology, Department of Psychology, Sa.C., Islamic Azad University, Sanandaj, Iran
2 Assistant Professor, Department of Psychology and Counseling, Farhangian University, Tehran, Iran
3 Associate Professor, Department of Psychology, Sa.C., Islamic Azad University, Sanandaj, Iran
*Correspondence: rahmaniseifola@gmail.com

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ABSTRACT

The aim of the present study was to predict psychosomatic symptoms based on an invalidating environment with the mediating role of alexithymia in individuals with generalized anxiety disorder. The research method was descriptive –correlational using a structural equation modeling design. A total of 300 individuals with generalized anxiety disorder who had referred to counseling centers in Tehran in 2025 were selected through convenience sampling and completed the Patient Health Questionnaire, the Toronto Alexithymia Scale, and the Childhood Invalidating Environment Scale. Data analysis was performed using structural equation modeling in SPSS 26 and AMOS 26. The findings indicated that the relationship between an invalidating environment and psychosomatic symptoms was positive and significant ($\beta = 0.35$, $p < .01$). In addition, alexithymia played a mediating role in the relationship between an invalidating environment and psychosomatic symptoms ($\beta = 0.30$, $p < .01$). Therefore, it can be concluded that an invalidating environment affects psychosomatic symptoms in individuals with generalized anxiety disorder both directly and indirectly through the mediating role of alexithymia. It is suggested that psychologists and therapists apply appropriate therapeutic interventions, including dialectical behavior therapy and emotional self-regulation training, to reduce alexithymia and, consequently, mitigate the impact of childhood invalidating environments on psychosomatic symptoms.

Keywords: invalidating environment, alexithymia, psychosomatic symptoms, generalized anxiety disorder

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Introduction

Generalized anxiety disorder (GAD) is a chronic and disabling condition characterized by excessive and uncontrollable worry, heightened physiological arousal, and pervasive feelings of apprehension about everyday events and future threats (1). Individuals with GAD frequently report a broad range of somatic complaints, including fatigue, gastrointestinal discomfort, musculoskeletal pain, and cardiopulmonary symptoms, which often drive help-seeking in primary care and specialty medical settings (2, 3). These somatic manifestations are not merely secondary or peripheral features of anxiety; rather, they are central

components of the disorder's clinical presentation and contribute significantly to impairment, health-care utilization, and reduced quality of life (1, 4). From a biopsychosocial perspective, GAD exemplifies the tight interweaving of psychological processes and bodily functioning, making it an important context for studying psychosomatic symptoms and their underlying mechanisms (3, 5).

Psychosomatic symptoms are defined as physical complaints in which psychological and social factors play a central role in the onset, exacerbation, or persistence of bodily distress, even when a clear biomedical pathology is absent or insufficient to explain their severity (3). Large-scale epidemiological and clinical studies have shown that psychosomatic symptom clusters are highly prevalent and closely linked to emotional disorders, especially anxiety and depression (2, 4). The Patient Health Questionnaire-15 (PHQ-15) is one of the most widely used instruments for assessing somatic symptom burden, with robust psychometric properties across psychiatric outpatient samples, supporting its use as a valid index of psychosomatic distress (6). In Iranian populations, distinct psychosomatic profiles have been identified and shown to covary with a range of psychological problems, underscoring the clinical relevance of somatic symptom assessment in culturally specific contexts (7). Moreover, psychosomatic complaints do not simply reflect symptom reporting style; they are associated with real functional limitations, treatment preferences, and adherence patterns, such that patients may prioritize process-related aspects of care (e.g., time, communication, perceived validation) even more than outcomes when somatic distress is high (8). Recent integrative work highlights that psychosomatic symptomatology emerges from complex biopsychosocial mechanisms—spanning immune, neuroendocrine, cognitive, and interpersonal pathways—rather than from a single causal factor (5).

Within this integrative perspective, environmental and interpersonal factors play a central role in shaping vulnerability to psychosomatic distress. One of the most influential frameworks for understanding the developmental origins of emotional and somatic dysregulation is Linehan's biosocial model, which proposes that chronic exposure to an invalidating environment in childhood disrupts the acquisition of adaptive emotion regulation skills (9). In this view, an invalidating environment is characterized by caregivers who dismiss, punish, trivialize, or distort the child's internal experiences—particularly emotional states—thereby teaching the child that his or her feelings are unacceptable, exaggerated, or untrustworthy (9). The Invalidating Childhood Environment Scale (ICES) was developed to operationalize these experiences and has demonstrated solid psychometric properties and meaningful associations with borderline personality symptomatology and other forms of psychological distress (10). More recent research has extended this construct to broader adult samples, showing that invalidating family environments are associated with maladaptive emotion regulation, distress intolerance, and interpersonal dysfunction (11).

Empirical studies support the notion that early adverse and invalidating experiences have long-term repercussions for mental health. Childhood abuse and neglect have been linked to late-life depression and chronic affective dysregulation, suggesting that early interpersonal trauma exerts enduring effects on vulnerability to mood and anxiety disorders (12). Attachment-based work indicates that insecure relational patterns are associated with medically unexplained somatic symptoms, partly through deficits in mentalization and difficulties making sense of internal states (13). In Iranian adults, childhood invalidating environments have been shown to predict problematic eating attitudes and behaviors via reduced self-compassion, low distress tolerance, and elevated impulsivity, highlighting the broad behavioral and

emotional sequelae of such environments (11). Parental alienation, as another form of relational invalidation, has also been conceptualized as a process that undermines the child's emotional security and distorts the meaning of attachment relationships, often with significant long-term psychological costs (14). These findings collectively suggest that early invalidation is a transdiagnostic risk factor that may contribute to the development of both internalizing symptoms and somatic complaints in adulthood.

Invalidating experiences are not only intrapsychic but also embedded within broader ecological and social contexts. Research in fields outside clinical psychology, such as conservation science, has shown that long-term management of complex human–environment interactions requires sustained, sensitive responses rather than punitive or dismissive strategies—whether the challenge is crop raiding by elephants or human distress signals (15). By analogy, chronic invalidation in family or community contexts can be conceptualized as a poorly managed "relational environment," in which individuals' emotional cues are repeatedly ignored or mishandled, perpetuating cycles of stress and maladaptive coping. Recent clinical work in neuropsychiatric populations underscores the salience of perceived invalidation: patients with drug-resistant epilepsy and functional dissociative seizures report distinct patterns of perceived invalidating environments and coping strategies, suggesting that perceived invalidation may interact with stress and neurobiological vulnerability to shape somatoform presentations (16).

Alexithymia—literally, "no words for feelings"—is a personality dimension characterized by difficulties identifying and describing one's emotions, a tendency to confuse emotional arousal with bodily sensations, and a cognitive style oriented toward external, concrete details rather than inner experiences (3). A growing body of evidence links alexithymia to a range of medical and psychiatric conditions, including cardiovascular disease, dermatological disorders, and mood and anxiety disorders (17, 18). In patients with cardiovascular disease, higher alexithymia and type D personality profiles predict greater emotional suppression, suggesting that difficulties in recognizing and articulating emotions may promote maladaptive coping and physiological strain (18). A systematic review in dermatology similarly shows that alexithymia is associated with worse morbidity and quality of life in cutaneous diseases, likely through interactions between stress reactivity, immune function, and self-care behaviors (17). Together, these findings support the conceptualization of alexithymia as a key vulnerability factor at the interface of mind and body.

Importantly, alexithymia has been repeatedly implicated in the etiology and maintenance of psychosomatic symptoms. Theoretical models propose that when individuals cannot accurately identify or verbalize emotional states, affective arousal is more likely to be expressed through bodily channels, leading to heightened somatic complaints and health-care seeking (3). Empirical findings from Iranian samples indicate that emotion regulation difficulties, defensive styles, and insecure attachment patterns significantly predict psychosomatic symptoms, underscoring the role of emotional processing capacities in somatic distress (19). In a sample of Iranian adults, distinct psychosomatic profiles are associated with elevated psychological problems, further reinforcing the intertwined nature of emotional dysfunction and bodily symptoms (7). At a broader level, a recent meta-analysis on systemic lupus erythematosus shows that psychosomatic symptoms in chronic autoimmune conditions arise from interlocking biopsychosocial pathways, including emotional processing deficits and stress responsivity (5).

More fine-grained mechanistic work has begun to clarify how alexithymia functions as a mediator between stress, invalidating environments, and psychosomatic distress. In adults with histories of childhood trauma,

alexithymia has been found to mediate the relationship between early adverse experiences and current psychological distress, suggesting that early relational trauma may shape later vulnerability partly by impairing emotional awareness and symbolization (20). Similarly, in somatic symptom disorder, mindfulness-based cognitive therapy (MBCT) reduces psychosomatic symptom distress in part by enhancing self-compassion and decreasing alexithymia, supporting a causal role for emotional awareness and acceptance in alleviating somatic complaints (21). These findings are consistent with broader relational and attachment-based frameworks in which deficits in mentalization and emotional understanding link early invalidation to adult somatic distress (12, 13).

The clinical picture of GAD further underscores the importance of examining alexithymia and invalidating environments together. Patients with GAD often exhibit heightened physiological arousal, pervasive worry about health, and frequent presentations with physical symptoms, yet they may struggle to precisely label or differentiate their emotional states (1, 2). Long-standing anxiety can amplify bodily vigilance and catastrophizing about normal somatic sensations, thereby escalating psychosomatic symptom reporting (3). At the same time, individuals with GAD frequently describe family environments in which emotional expression was discouraged or minimized, which may contribute to long-term patterns of emotional suppression and alexithymia (9, 11). Despite these converging lines of evidence, relatively few studies have explicitly modeled the pathways from childhood invalidating environments to psychosomatic symptoms in adults with GAD, particularly with alexithymia as a mediating mechanism.

Dialectical behavior therapy (DBT), grounded in the biosocial model, directly targets the legacy of invalidation by fostering emotion regulation, distress tolerance, mindfulness, and interpersonal effectiveness (9). Findings from intervention and preference research show that patients are sensitive not only to treatment outcomes but also to relational and process characteristics such as validation, collaborative decision-making, and respect for their embodied experience of illness (2, 8). These clinical observations underscore that invalidation is not merely a historical factor but a continuing interpersonal process that may perpetuate psychosomatic distress unless explicitly addressed. Identifying the extent to which alexithymia transmits the impact of early invalidating environments to current psychosomatic symptoms could therefore inform the development and refinement of DBT-informed and MBCT-based interventions for GAD (9, 21).

Methodologically, structural equation modeling (SEM) offers a powerful framework for testing such mediational hypotheses, as it allows simultaneous estimation of relationships among latent constructs—such as invalidating environment, alexithymia, and psychosomatic symptoms—while accounting for measurement error (22, 23). Validated measures like the PHQ-15 for somatic symptoms, the ICES for childhood invalidation, and established alexithymia scales provide a solid psychometric foundation for constructing latent variables and examining complex pathways (6, 7, 10, 19). Integrating these methodological advances with contemporary biopsychosocial models of psychosomatic distress may clarify which aspects of early environment and emotional processing are most critical for clinical assessment and intervention in GAD (5, 21).

Given the high prevalence and burden of psychosomatic symptoms in individuals with GAD, the documented impact of invalidating childhood environments on emotional development, and the emerging evidence for alexithymia as a key mediator linking early adversity to adult psychosomatic distress, the present study aimed to examine whether psychosomatic symptoms in individuals with generalized anxiety

disorder can be predicted based on an invalidating environment, both directly and indirectly through the mediating role of alexithymia.

Methods and Materials

Study Design and Participants

The present study employed a descriptive–correlational design using structural equation modeling. The statistical population consisted of all individuals with generalized anxiety disorder who referred to counseling centers in Tehran in 2025. In structural equation modeling, Loehlin (2004) considers a minimum sample size of 100 and an optimal sample size of 200, while Kline (2015) recommends a minimum sample size of 200. Given that the present study also used a correlational design with structural equation modeling—and that sample size is critically important considering the data analysis method—a sample size of 200 participants was deemed appropriate. To allow for overestimation, 220 participants were selected. Sampling was conducted using a convenience sampling method. Inclusion criteria—alongside informed consent—included a minimum education level of a high school diploma, age between 20 and 50 years, diagnosis of generalized anxiety disorder based on a clinical interview by a specialist, and absence of other medical or psychological disorders as diagnosed by a psychiatrist. Exclusion criteria included providing incomplete information and withdrawing from completing the questionnaires. To adhere to ethical principles, participants were informed that the questionnaires were being used to gather data for an academic research project, that their responses would remain confidential and anonymous, and that they could withdraw at any stage without any negative consequences.

Data Collection

Patient Health Questionnaire (PHQ). The 15-item Patient Health Questionnaire was used to assess psychosomatic disorders. The original questionnaire was developed by Kroenke et al. (2002). The 15-item scale assesses somatic symptoms and evaluates the range of symptoms experienced by individuals over the past month. Response options range from 0 (not bothered at all) to 2 (bothered a lot). The overall reliability of the scale was reported as .87 by Han et al. (2009), with a test–retest reliability of .65. In Iran, Shobe, Feyzi, Afshar, and Hassanzadeh (2016) reported a reliability of .92 and sensitivity and specificity of 73.80% and 76.20%, respectively. Han et al. (2009) also found significant correlations between this questionnaire and the Beck Depression Inventory (r = .559) and the 12-item General Health Questionnaire (r = .435).

Toronto Alexithymia Scale (TAS-20). This questionnaire was developed by Taylor in 1986 and revised by Bagby, Parker, and Taylor in 1994. The Toronto Alexithymia Scale consists of 20 items and includes three subscales: Difficulty Identifying Feelings, Difficulty Describing Feelings, and Externally Oriented Thinking. The scale is scored on a 5-point Likert scale ranging from strongly disagree to strongly agree. The components and corresponding items are as follows: Difficulty Identifying Feelings (DIF), assessing the individual's ability to recognize emotions and differentiate them from bodily sensations, including items 1, 3, 6, 7, 9, 13, and 14; Difficulty Describing Feelings (DDF), assessing the ability to verbalize emotions, including items 2, 4, 11, 12, and 17; and Externally Oriented Thinking (EOT), assessing the extent of introspection and emotional reflection, including items 5, 8, 10, 15, 16, 18, 19, and 20. The psychometric properties of the Toronto Alexithymia Scale have been examined and confirmed in multiple studies. In the

Persian version, Cronbach's alpha coefficients were .85 for the total alexithymia score, .82 for Difficulty Identifying Feelings, .75 for Difficulty Describing Feelings, and .72 for Externally Oriented Thinking, indicating good internal consistency. Concurrent validity was confirmed based on correlations between the subscales and measures of emotional intelligence, psychological well-being, and psychological distress (Elahi-Mehr, Shahqolian, Abdollahi, & Rajabi, 2021).

Invalidating Childhood Environment Scale (ICES). This questionnaire was developed by Manfredi and colleagues in 2004 and includes two components—negative responses and lack of support—in both mother and father forms. Scoring is based on a Likert scale ranging from 1 (never) to 5 (always). The minimum score is 14 and the maximum score is 70. In a study by Robertson (2013), the psychometric properties of this questionnaire were assessed, showing good reliability in a nonclinical sample, with Cronbach's alpha coefficients of .90 for the father form and .88 for the mother form. Convergent validity was supported by correlations between invalidating environment scores and borderline personality disorder symptoms. In Iran, Rahmati (2022) reported Cronbach's alpha coefficients for the mother-related items as .99 for the total scale, .98 for negative maternal responses, and .98 for maternal lack of support; and for the father-related items as .99 for the total scale, .99 for negative paternal responses, and .98 for paternal lack of support. The correlations between the total mother form, negative maternal responses, and maternal lack of support with distress tolerance were –.235, –.229, and –.187, respectively; and for the father form –.247, –.235, and –.202 at $p < .001$, indicating support for the divergent validity of the scale.

Data analysis

Data analysis was conducted using structural equation modeling in SPSS 26 and AMOS 26.

Findings and Results

The descriptive indices of the participants' demographic characteristics indicated that most participants held a bachelor's degree (49.66%) and the majority were women (62.67%). Most participants were employed in office jobs (41.0%) or were self-employed (37.33%).

Table 1. Mean, Standard Deviation, and Cronbach's Alpha of the Study Variables

Variable	Mean	Standard Deviation	Cronbach's Alpha (Total Scale)
Invalidating Environment – Maternal Lack of Support	2.42	0.756	—
Invalidating Environment – Paternal Lack of Support	2.38	0.782	—
Invalidating Environment – Maternal Negative Responses	2.34	0.744	—
Invalidating Environment – Paternal Negative Responses	2.63	0.745	—
Invalidating Environment – Total Score	2.44	0.635	.839
Difficulty Identifying Feelings	2.32	0.942	—
Difficulty Describing Feelings	2.24	0.951	—
Externally Oriented Thinking	2.82	0.923	—
Alexithymia – Total Score	2.46	0.808	.792
Psychosomatic Symptoms – Total Score	0.997	0.293	.783

Table 1, in addition to presenting the mean and standard deviation of the study variables, shows the Cronbach's alpha coefficients. Results indicate that all alpha coefficients are close to or above .70, demonstrating that the questionnaire items used to measure the variables possess acceptable internal consistency.

The assumptions of univariate normality, skewness, and kurtosis of total scores in the variables are presented in Table 2.

Table 2. Skewness, Kurtosis, and Univariate Normality Tests of the Study Variables

Variable	Shapiro–Wilk	Kolmogorov–Smirnov	Skewness	Kurtosis
Invalidating Environment	.939***	.147***	.749	1.536
Alexithymia	.921***	.178***	.972	.989
Psychosomatic Symptoms	.988***	.076***	.203	–.275

Table 2 shows that the absolute values of kurtosis and skewness for all variables fall within ±2. This indicates that the univariate distribution of the variables does not deviate significantly from normality. However, the normality tests indicate that variable distributions are non-normal. Examination of individual scores revealed that nine scores did not meet the assumption of univariate normality; these scores were identified and removed on a case-by-case basis.

To evaluate the assumption of multivariate normality, Mahalanobis distance values were analyzed.

Table 3. Multivariate Normality Tests

Test	Minimum	Maximum	Mean	Standard Deviation	N
Mahalanobis Distance	0.061	15.869	1.9993	2.666	289
Cook's Distance	0.000	0.061	0.004	0.006	289

The results of Table 3 indicate that the maximum Mahalanobis distance value (15.869) exceeds the critical chi-square value with 2 degrees of freedom (18.42), suggesting the presence of multivariate outliers. Two such scores were removed on a case-by-case basis.

The assumption of multicollinearity was assessed using Variance Inflation Factor (VIF) and tolerance values. Results are shown in Table 4.

Table 4. Variance Inflation and Tolerance of Predictor Variables

Variable	Tolerance	VIF
Invalidating Environment	.834	1.199
Alexithymia	.733	1.364

Table 4 shows that the assumption of multicollinearity is satisfied, as the tolerance values for all predictors exceed .10 and VIF values are below 10. According to Meyers et al. (2006), tolerance values below .10 and VIF values above 10 would indicate violation of the multicollinearity assumption.

Table 5. Correlation Matrix of Total Variable Scores

	1	2	3
1. Invalidating Environment	1	—	—
2. Alexithymia	.326**	1	—
3. Psychosomatic Symptoms	.294**	.444**	1

***p < .05*

Table 5 indicates that there is no correlation above .90 between the independent variables. Additionally, there are positive and significant correlations among all variables.

To evaluate linearity, a multiple scatterplot matrix was used. Results indicated that relationships between variables were linear. Homogeneity of variances was examined using a scatterplot of standardized residuals, which confirmed that the assumption of homoscedasticity was met.

The following conceptual model was tested:

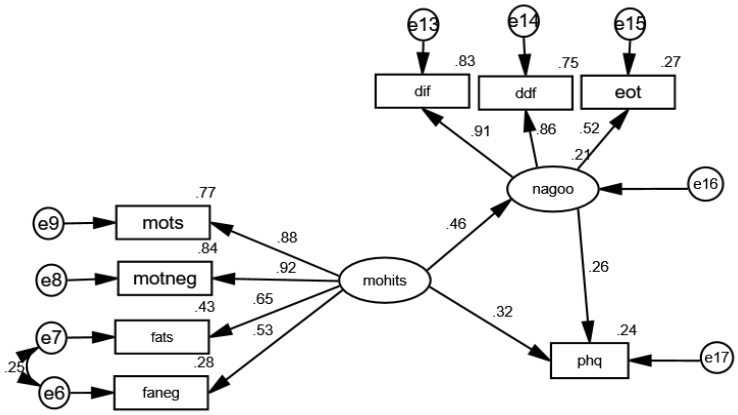

Figure 1. Conceptual model of alexithymia as a mediator in the association between an invalidating environment and psychosomatic symptoms.

Table 6. Goodness-of-Fit Indices of the Conceptual Model

Fit Index	Initial Model	Revised Measurement Model	Cutoff Point	Result
Chi-square	86.366	137.820	$p < .001$	Confirmed
Degrees of Freedom	18	17	—	—
χ^2/df	4.798	4.115	< 3	Confirmed
GFI	.928	.945	> .90	Confirmed
AGFI	.856	.883	> .85	Confirmed
CFI	.936	.940	> .90	Confirmed
RMSEA	.115	.104	< .08	Confirmed

Table 6 shows that the goodness-of-fit indices support the acceptable fit of the initial measurement model with the collected data. In the revised model, covariance was added between the error terms of paternal lack of support (fats) and paternal negative responses (faneg).

Table 7. Factor Loadings of the Measurement Model (Confirmatory Factor Analysis)

Indicators	Path (Latent → Indicator)	b	S.E.	β	Critical Ratio
Invalidating Environment → Paternal Negative Responses	1.000	—	.527	—	
Invalidating Environment → Paternal Lack of Support	1.263	.136	.653	9.263***	
Invalidating Environment → Maternal Negative Responses	1.677	.179	.922	9.345***	
Invalidating Environment → Maternal Lack of Support	1.700	.183	.870	9.306***	
Alexithymia → Difficulty Identifying Feelings	1.000	—	.988	—	
Alexithymia → Difficulty Describing Feelings	.848	.077	.800	10.986***	
Alexithymia → Externally Oriented Thinking	.510	.053	.496	6.160***	

Table 7 presents the results of confirmatory factor loadings for the measurement model. All factor loadings exceed .40 and are statistically significant. After confirming the measurement model and adequate Cronbach's alphas, the conceptual model was tested.

Table 8. Standard Path Coefficients of Observed Variables in the Model

Causal Variables	Path Coefficient	Alexithymia	Psychosomatic Symptoms	Label
Invalidating Environment	Direct	.46***	.32***	c
	Mediated	—	.30***	—
	Indirect	—	.482**	a

Alexithymia	Direct	—	.26***	b
	Mediated	—	—	—
	Indirect	—	—	—

According to Table 8, the total path coefficient between an invalidating childhood environment and psychosomatic symptoms (β = .32, p < .01) is positive and significant. In other words, higher levels of invalidating childhood environment experiences predict higher levels of psychosomatic symptoms. Using Baron and Kenny's method, the indirect effect between an invalidating childhood environment and psychosomatic symptoms through alexithymia was positive and significant (β = .30, p < .01). Thus, alexithymia positively and significantly mediates the effect of an invalidating childhood environment on psychosomatic symptoms. However, because the direct path remains significant, the mediation is partial.

Effect sizes of the independent variable—invalidating childhood environment—on alexithymia and psychosomatic symptoms are presented in Table 9.

Table 9. Multiple Correlation Coefficient (R^2) of Effects of Independent Variables on Alexithymia and Psychosomatic Symptoms

Dependent Variable	R^2
Alexithymia	.212
Psychosomatic Symptoms	.244

As shown, invalidating environment and alexithymia together explain 24.4% of the variance in psychosomatic symptoms. Invalidating childhood environment alone explains 21.2% of the variance in alexithymia.

Discussion and Conclusion

The present study examined the predictive role of an invalidating childhood environment on psychosomatic symptoms in individuals with generalized anxiety disorder (GAD), with alexithymia tested as a mediating variable. The results demonstrated that an invalidating environment significantly and positively predicted psychosomatic symptoms, and this relationship was partially mediated by alexithymia. These findings provide strong empirical support for developmental–emotional models of psychosomatic distress, particularly those emphasizing the long-term effects of emotional invalidation and deficits in emotional awareness on somatic symptom expression.

The direct relationship identified between an invalidating childhood environment and psychosomatic symptoms aligns with a growing body of evidence demonstrating that early relational experiences shape adult emotional and physiological functioning. In particular, research has shown that emotionally invalidating caregiving—characterized by dismissive, punitive, or inconsistent responses to the child's emotional states—disrupts the development of adaptive emotion regulation capacities (9). Within the biosocial framework, such disruption increases vulnerability to chronic emotional dysregulation and somatic distress. Consistent with this model, prior studies have documented significant links between invalidating environments and a variety of psychological and behavioral difficulties, including maladaptive coping, distress intolerance, and impulsivity (11). The findings of the current study extend this line of research by demonstrating that invalidation is also strongly implicated in the somatic symptom burden of individuals with GAD.

These results are also supported by studies examining childhood adversity and emotional development. For instance, childhood abuse and neglect have been shown to exert long-lasting effects on affective functioning and depressive symptoms in later life (12). Similar relational patterns appear in individuals reporting high psychosomatic symptom loads, especially when early emotional experiences were characterized by unpredictability, rejection, or blame. The current study's findings resonate with such work by showing that even in the absence of overt trauma, persistent invalidation in childhood may shape maladaptive emotional schemas that manifest physically many years later. The findings also resonate with attachment-based studies demonstrating that insecure attachment styles and impaired mentalization are associated with medically unexplained somatic symptoms (13). This suggests that disruptions in emotional and relational development provide a credible pathway through which invalidating environments contribute to bodily distress.

The significant mediating effect of alexithymia highlights the critical role of emotional processing deficits in psychosomatic experiences. Individuals exposed to chronic invalidation often learn to suppress, ignore, or misinterpret their emotional experiences due to repeated messages that their feelings are inaccurate or unacceptable (9). Over time, these adaptive responses become habitual patterns of emotional unawareness, consistent with the core features of alexithymia. The present findings are consistent with prior research documenting strong associations between childhood trauma, invalidation, and alexithymia (20). In this respect, alexithymia can be conceptualized as a psychological mechanism through which early relational experiences exert influence on somatic symptomatology.

The mediational results mirror previous empirical findings showing that individuals with high alexithymia have greater difficulty differentiating physical sensations from emotional arousal, thereby increasing bodily vigilance, misinterpretation of physical sensations, and ultimately somatic symptom reporting (3). Studies in both psychiatric and medical populations have reinforced this pattern. For example, alexithymia has been repeatedly associated with more severe morbidity in dermatological conditions (17), heightened emotional suppression in cardiac patients (18), and broader psychosomatic vulnerability across chronic conditions (5). Taken together, these findings support the premise that deficits in emotional awareness and articulation contribute substantially to somatic distress. The present study adds to this literature by confirming this mediational pathway specifically among individuals diagnosed with GAD, a population known to exhibit elevated physiological hyperarousal and somatic sensitivity.

Furthermore, the partial mediation effect observed in the model suggests that invalidating environments influence psychosomatic symptoms not only through alexithymia but also through other pathways. These may include heightened stress reactivity, developmental impacts on the autonomic nervous system, maladaptive schemas about bodily sensations, and interpersonal patterns that maintain anxiety and somatization (2, 3). Some of these mechanisms are evident in research showing that individuals with chronic anxiety exhibit increased somatic amplification and excessive health worry, which may be rooted in early relational contexts where emotional discomfort was minimized or pathologized (1).

Additionally, the present results align with recent findings demonstrating that perceptions of invalidation continue to influence coping strategies and somatic presentations in adulthood. In neuropsychiatric populations, individuals with functional dissociative seizures, for example, report more negative early emotional experiences and distinct patterns of coping compared to those with drug-resistant epilepsy (16).

These findings support the idea that invalidation may contribute to somatization across a wide range of clinical presentations. From a biopsychosocial perspective, relational, emotional, and cognitive factors converge to produce a profile of heightened bodily distress and impaired emotional communication. The current study's results strengthen this conceptualization by showing that both invalidation and alexithymia contribute significantly to psychosomatic symptoms in individuals with GAD.

Methodologically, the use of structural equation modeling (SEM) provided a rigorous analytic approach for evaluating the direct and indirect pathways among variables. SEM is recognized as one of the most robust methods for testing complex mediational models, as it accounts for measurement error and allows simultaneous modeling of latent constructs (22, 23). The constructs examined in this study—invalidating environment, alexithymia, and psychosomatic symptoms—are multidimensional psychological processes that lend themselves to latent variable modeling. The model fit indices obtained in this study indicate that the hypothesized model was theoretically justifiable and empirically supported. Prior studies have effectively used SEM to investigate similar pathways, such as the mediating effects of alexithymia in trauma-related distress (20) and the effectiveness of mindfulness-based interventions in reducing psychosomatic symptoms through self-compassion and reduced alexithymia (21). Thus, the methodological approach further validates the study's findings and strengthens the interpretability of the results.

Overall, the study provides empirical evidence for a conceptual model wherein an invalidating environment serves as a significant antecedent to psychosomatic symptoms in GAD, with alexithymia acting as a meaningful psychological mechanism linking early emotional experiences to adult somatic distress. The findings underscore the importance of early relational experiences in shaping emotional and bodily well-being and offer clear implications for prevention, assessment, and treatment of psychosomatic symptoms in GAD populations.

Despite its significant contributions, the present study has several limitations. First, the use of self-report instruments introduces the possibility of response bias, including social desirability and recall bias regarding childhood experiences. Second, the cross-sectional design limits the ability to establish temporal or causal relationships among variables. Longitudinal studies would be necessary to confirm the developmental trajectory implied by the model. Third, the sample was limited to individuals seeking counseling services in a single metropolitan area, which may reduce the generalizability of findings to broader clinical or community populations. Fourth, the study did not account for potentially confounding variables such as current life stress, comorbid psychiatric conditions, medication use, or cultural factors that may influence both alexithymia and somatic symptom reporting. Lastly, the reliance on a single type of mediator restricts the exploration of other potential mechanisms that may link invalidation to psychosomatic symptoms.

Future research should employ longitudinal or prospective designs to more clearly establish causal pathways between early invalidation, alexithymia, and psychosomatic symptoms. Expanding the sample to include diverse populations—such as nonclinical individuals, different age groups, or cross-cultural samples—would enhance generalizability. Future studies may also incorporate multi-method assessments, including clinical interviews, physiological measures, or informant reports, to reduce the limitations of self-report. Additionally, research could explore multiple mediators simultaneously, such as mentalization, distress tolerance, attachment styles, or emotion regulation strategies, to construct a more comprehensive model. Experimental or intervention-based studies examining whether reductions in alexithymia lead to

corresponding decreases in psychosomatic symptoms would be particularly valuable in confirming the mediational role identified in the present study.

Clinicians working with individuals who exhibit psychosomatic symptoms and generalized anxiety should consider assessing histories of emotional invalidation and levels of alexithymia during intake and treatment planning. Interventions that target emotional awareness, labeling, and expression may be especially beneficial. Therapeutic modalities such as dialectical behavior therapy, emotion-focused therapy, and mindfulness-based interventions can support the development of emotional processing skills and promote healthier responses to bodily sensations. In clinical practice, fostering validation and strengthening emotional communication may help reduce somatic distress and enhance treatment engagement.

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Authors' Contributions

All authors equally contributed to this study.

Declaration of Interest

The authors of this article declared no conflict of interest.

Ethical Considerations

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

Transparency of Data

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

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