KMAN PUBLISHING INSTITUTE
Journal Website

Article history:
Received 24 October 2024
Revised 21 December 2024
Accepted 06 January 2025
Published online 01 April 2025

Journal of Personality and Psychosomatic Research

Volume 3, Issue 2, pp 1-9

E-ISSN: 3041-8542

Cognitive Flexibility Mediates the Impact of Adverse Childhood Experiences on Somatization

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A r t i c l e I n f o

Article type:
Original Research

How to cite this article:
Rostami, M., & Baniasadi, T.M. (2025). Cognitive Flexibility Mediates the Impact of Adverse Childhood Experiences on Somatization. *Journal of Personality and Psychosomatic Research, 3*(2), 1-9.
https://doi.org/10.61838/kman.jppr.3.2.1

A B S T R A C T

This study aimed to examine whether cognitive flexibility mediates the relationship between adverse childhood experiences (ACEs) and somatization in adults. A descriptive correlational design was employed using data from 440 adult participants in the United States. The sample size was determined based on the Morgan and Krejcie table. Participants completed standardized self-report measures including the Adverse Childhood Experiences Questionnaire (ACE-Q), the Cognitive Flexibility Inventory (CFI), and the Patient Health Questionnaire-15 (PHQ-15) for somatization. Data were analyzed using SPSS-27 for descriptive statistics and Pearson correlation coefficients, and AMOS-21 was used for Structural Equation Modeling (SEM) to test the hypothesized mediation model. The results revealed that ACEs were positively correlated with somatization ($r = .46$, $p < .001$) and negatively correlated with cognitive flexibility ($r = –.39$, $p < .001$). Cognitive flexibility also showed a significant negative correlation with somatization ($r = –.41$, $p < .001$). The structural model demonstrated good fit ($\chi^2/df = 2.21$, GFI = .96, CFI = .97, RMSEA = .052). SEM results indicated that ACEs significantly predicted somatization both directly ($\beta = .31$, $p < .001$) and indirectly through cognitive flexibility ($\beta = .10$, $p < .001$), with a total effect of $\beta = .41$ ($p < .001$). The mediating role of cognitive flexibility was statistically significant, confirming partial mediation. These findings suggest that cognitive flexibility plays a protective role in the pathway from childhood adversity to physical symptom expression. Interventions aimed at enhancing cognitive flexibility may reduce the severity of somatization among trauma-exposed individuals and contribute to more effective trauma-informed care.

Keywords: *Adverse Childhood Experiences; Cognitive Flexibility; Somatization; Structural Equation Modeling; Trauma; Mediation Analysis.*

1. Introduction

Adverse childhood experiences (ACEs), encompassing various forms of abuse, neglect, and household dysfunction, are increasingly recognized as critical determinants of mental and physical health across the lifespan. Numerous studies have demonstrated that early traumatic experiences can become embedded in an individual's biopsychosocial development, influencing stress reactivity, emotional processing, and bodily awareness well into adulthood (Heule, 2025; Ma, 2025). One of the common psychological manifestations linked to ACEs is somatization—a process by which psychological distress is expressed through physical symptoms without an identifiable organic cause. Somatic symptoms can range from gastrointestinal discomfort and chronic pain to cardiovascular complaints and fatigue, often leading to functional impairment and increased healthcare utilization (Adamowicz et al., 2024; Prasetya et al., 2023).

The relationship between early adversity and somatization has been supported by a growing body of empirical evidence suggesting both direct and indirect pathways. Directly, ACEs may increase physiological stress sensitivity, contributing to autonomic dysregulation and inflammatory responses that manifest somatically (Rostami & Mehdiabadi, 2024; Wang, 2024). Indirectly, early trauma can disrupt emotional regulation, interpersonal functioning, and cognitive appraisal systems—factors that mediate the expression of psychological distress as physical symptoms (Lee & Yoon, 2024; Nudin & Mohamed, 2023). Importantly, the presence and severity of somatization symptoms often vary depending on the type, frequency, and cumulative burden of childhood trauma (Gardoki-Souto et al., 2022; Marmet et al., 2024).

Recent research has underscored that individuals exposed to multiple ACEs may be more vulnerable to developing somatic symptom disorders, especially in the absence of adaptive psychological resources such as cognitive flexibility (Ma, 2025; Zhang et al., 2023). Cognitive flexibility refers to the mental capacity to shift perspectives, adapt to changing demands, and integrate alternative interpretations of one's experiences. As such, it is considered a crucial component of executive functioning and a protective factor against maladaptive coping styles. While cognitive rigidity is often associated with psychological dysfunction and somatization, flexibility facilitates adaptive emotional processing and cognitive reappraisal, thereby mitigating the impact of traumatic experiences (Laube et al., 2024; Portilla-Saavedra et al., 2023).

The mediating role of cognitive flexibility in the relationship between ACEs and somatization is theoretically grounded in cognitive-behavioral and neurodevelopmental frameworks. From a cognitive-behavioral perspective, maladaptive schemas and ruminative thought patterns formed during childhood trauma can become entrenched if not challenged or reframed. Individuals with high cognitive flexibility are more likely to recognize these distortions and adjust their interpretations in emotionally adaptive ways, thereby reducing psychological distress and somatic symptom expression (Crismayanti et al., 2024; Lombardo, 2024). Neurodevelopmentally, ACEs are known to alter the functioning of the prefrontal cortex and limbic system, affecting executive control and emotional regulation. However, emerging evidence suggests that certain neurocognitive traits, including flexibility, may buffer against the long-term effects of such changes (Mancone et al., 2024; Tao, 2023).

Several empirical studies support the association between cognitive inflexibility and increased somatic complaints in trauma-exposed individuals. For example, individuals with a history of childhood maltreatment often report difficulty disengaging from negative emotional stimuli, which can intensify physical symptom reporting through mechanisms such as heightened interoceptive awareness and hypervigilance to bodily sensations (Adamowicz et al., 2024; Saadati et al., 2023). Moreover, cognitive rigidity may exacerbate the misinterpretation of benign bodily cues as threatening, thus reinforcing somatic distress (Heule, 2025; Lee & Yoon, 2024). In contrast, those with greater cognitive flexibility are able to challenge catastrophic interpretations, reframe distressing bodily sensations, and engage in problem-focused coping, ultimately experiencing fewer somatic symptoms (Mancone et al., 2024; Marmet et al., 2024).

Despite the growing recognition of these dynamics, few studies have systematically examined the mediating role of cognitive flexibility in the ACE-somatization pathway using robust statistical approaches such as structural equation modeling (SEM). Most existing studies have either assessed direct relationships or employed cross-sectional correlational designs without exploring mediation effects. In addition, many studies have focused exclusively on either the physiological or psychological outcomes of ACEs, without integrating both perspectives in a unified model (Crismayanti et al., 2024; Laube et al., 2024). This represents

JPPR
E-ISSN: 3041-8542

a significant gap in the literature, given that somatization lies at the intersection of psychological trauma and bodily expression.

Understanding the mediating role of cognitive flexibility has important implications for clinical practice and intervention. If cognitive flexibility serves as a protective buffer, interventions aimed at enhancing this capacity—such as cognitive-behavioral therapy, mindfulness-based approaches, and neurofeedback—may offer effective strategies for reducing somatic symptom severity in trauma-exposed individuals (Lombardo, 2024; Pichel & Suryadi, 2021). Moreover, identifying flexibility as a mediating mechanism supports a shift from purely symptom-focused treatments to resilience-oriented frameworks that foster adaptive cognition and emotion regulation (Gardoki-Souto et al., 2022; Portilla-Saavedra et al., 2023).

Cultural and contextual factors may also influence the relationships among ACEs, cognitive flexibility, and somatization. For instance, cultural norms surrounding emotional expression, bodily awareness, and help-seeking behavior may mediate how trauma is internalized and expressed physically (Nudin & Mohamed, 2023; Tao, 2023). In collectivist cultures, emotional suppression and somatization may be more socially reinforced, while individualist cultures may emphasize cognitive processing and verbal articulation of distress. These dynamics highlight the importance of considering sociocultural contexts in research and clinical interventions related to trauma and somatic symptoms.

In summary, there is compelling theoretical and empirical support for the hypothesis that cognitive flexibility may mediate the relationship between adverse childhood experiences and somatization. ACEs have consistently been linked to heightened psychological distress and physical symptomatology, while cognitive inflexibility has been identified as a key vulnerability factor that may explain the persistence of somatic complaints in trauma-exposed individuals (Saadati et al., 2023; Wang, 2024). At the same time, cognitive flexibility may offer a pathway to resilience by enabling trauma survivors to reinterpret, reframe, and regulate their internal experiences more adaptively (Ma, 2025; Zhang et al., 2023). Despite this promising evidence, research on the mediating role of cognitive flexibility remains limited, especially in large community samples using advanced statistical techniques.

To address this gap, the present study aims to examine the mediating role of cognitive flexibility in the association between adverse childhood experiences and somatization in a sample of adults residing in the United States.

2. Methods and Materials

2.1. Study Design and Participants

This study employed a descriptive correlational design to investigate the mediating role of cognitive flexibility in the relationship between adverse childhood experiences and somatization. The target population consisted of adults residing in the United States. Based on the sample size determination table developed by Morgan and Krejcie, a sample of 440 participants was deemed sufficient to ensure statistical power and representativeness. Participants were selected using convenience sampling through online platforms and community outreach. Eligibility criteria included being 18 years or older, having the ability to read and understand English, and providing informed consent. Demographic information such as age, gender, education level, and socioeconomic status was also collected to characterize the sample.

2.2. Measures

2.2.1. Somatization

The PHQ-15, developed by Kroenke, Spitzer, and Williams in 2002, is a widely used self-report instrument designed to assess the severity of somatic symptoms. It consists of 15 items that inquire about the frequency of common physical complaints (e.g., headaches, stomach pain, dizziness) experienced over the past four weeks. Each item is rated on a 3-point Likert scale ranging from 0 (not bothered at all) to 2 (bothered a lot), yielding a total score ranging from 0 to 30. Higher scores indicate greater levels of somatization. The PHQ-15 has demonstrated strong internal consistency (Cronbach's alpha > 0.80) and convergent validity with clinician-rated assessments and other somatic symptom measures, making it a reliable and valid tool in both clinical and research contexts (Ma, 2025; Mancone et al., 2024; Nakhaei Moghadam et al., 2024).

2.2.2. Cognitive Flexibility

The Cognitive Flexibility Inventory (CFI), developed by Dennis and Vander Wal in 2010, is a 20-item self-report scale created to assess individuals' tendencies to adapt to changing situational demands and to shift cognitive perspectives in response to challenging or stressful

experiences. The CFI comprises two subscales: Alternative Thinking (the ability to generate multiple alternative explanations for life occurrences and human behavior) and Control (the perception of situations as controllable). Responses are rated on a 7-point Likert scale from 1 (strongly disagree) to 7 (strongly agree), with higher scores reflecting greater cognitive flexibility. The CFI has shown strong psychometric properties, including high internal consistency (Cronbach's alpha > 0.80 for both subscales) and good construct validity across diverse populations and settings (Okur et al., 2025; Vakilian et al., 2024; Zhou et al., 2024).

2.2.3. Adverse Childhood Experiences

The Adverse Childhood Experiences Questionnaire (ACE-Q), originally developed by Felitti et al. in 1998 as part of the CDC-Kaiser Permanente ACE Study, is a standardized screening tool used to assess exposure to traumatic childhood experiences before the age of 18. The ACE-Q consists of 10 items, each addressing different domains of childhood adversity, including emotional, physical, and sexual abuse, emotional and physical neglect, and household dysfunction (e.g., substance abuse, domestic violence, mental illness, incarceration, and parental separation). Each item is scored dichotomously (0 = no, 1 = yes), with total scores ranging from 0 to 10, where higher scores indicate greater cumulative exposure to childhood adversity. Numerous studies have confirmed the ACE-Q's test-retest reliability and its predictive validity in relation to various psychological and physical health outcomes in adulthood (Heule, 2025; Ma, 2025; Rahapsari & Levita, 2024; Torgah, 2024).

2.3. Data Analysis

Data were analyzed using SPSS version 27 and AMOS version 21. Initially, descriptive statistics were computed to examine the distribution and central tendencies of the variables. Pearson correlation coefficients were calculated to assess the bivariate relationships between somatization (dependent variable), adverse childhood experiences, and cognitive flexibility (independent and mediating variables). To test the hypothesized mediation model, Structural Equation Modeling (SEM) was performed using AMOS-21. Model fit was evaluated using common indices such as the chi-square statistic, Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), and Tucker-Lewis Index (TLI). The indirect effect of adverse childhood experiences on somatization through cognitive flexibility was assessed using bootstrapping procedures with 5,000 resamples to determine the significance of the mediation effect.

3. Findings and Results

The final sample consisted of 440 adult participants from the United States. Of these, 261 participants (59.3%) identified as female, 172 (39.1%) as male, and 7 (1.6%) as non-binary or preferred not to disclose their gender. The age of participants ranged from 18 to 65 years, with a mean age of 32.7 years (SD = 10.4). In terms of educational attainment, 46 participants (10.5%) had a high school diploma or equivalent, 163 (37.0%) held a bachelor's degree, 139 (31.6%) had completed some college or an associate degree, and 92 (20.9%) reported having a graduate or professional degree. Regarding employment status, 281 participants (63.9%) were employed full-time, 81 (18.4%) were employed part-time, 43 (9.8%) were students, and 35 (8.0%) were unemployed or retired at the time of the study.

Table 1

Descriptive Statistics for Study Variables (N = 440)

Variable	Mean	SD
Adverse Childhood Experiences (ACE)	4.76	2.53
Cognitive Flexibility	93.42	14.67
Somatization (PHQ-15)	13.28	5.41

Participants reported an average ACE score of 4.76 (SD = 2.53), suggesting moderate exposure to adverse experiences. The mean score for cognitive flexibility was 93.42 (SD = 14.67), which is consistent with normative adult samples. The average somatization score was 13.28 (SD = 5.41), indicating a mild to moderate level of physical symptom distress (Table 1).

Prior to conducting the main statistical analyses, key assumptions for Pearson correlation and Structural Equation Modeling (SEM) were examined and met. The data were

JPPR
E-ISSN: 3041-8542

assessed for normality using skewness and kurtosis values, which ranged from –0.81 to 0.67 and –0.94 to 1.12, respectively, falling within the acceptable range of ±2. The assumption of linearity was confirmed through scatterplots showing consistent linear patterns between variables. Homoscedasticity was evaluated using residual plots, which indicated evenly distributed residuals across predicted values. Multicollinearity was not a concern, as all Variance Inflation Factor (VIF) values were below 2.1. Additionally, the absence of significant outliers was confirmed using Mahalanobis distance (critical $\chi^2 = 16.27$, df = 3, $p < .001$), with only 3 cases exceeding the threshold and subsequently excluded from the final analysis. Therefore, the dataset met the assumptions required for both Pearson correlation and SEM procedures.

Table 2

Correlation Matrix for Study Variables

Variable	1	2	3
1. ACE	—	–.39** (p < .001)	.46** (p < .001)
2. Cognitive Flexibility		—	–.41** (p < .001)
3. Somatization			—

ACE scores were significantly negatively correlated with cognitive flexibility (r = –.39, p < .001), and positively correlated with somatization (r = .46, p < .001). Cognitive flexibility was also significantly negatively correlated with somatization (r = –.41, p < .001), indicating that individuals with higher cognitive flexibility tend to report fewer somatic symptoms (Table 2).

Table 3

Fit Indices for the Structural Model

Fit Index	Value	Threshold for Good Fit
χ^2	112.84	—
df	51	—
χ^2/df	2.21	< 3.00
GFI	.96	≥ .90
AGFI	.94	≥ .90
CFI	.97	≥ .95
TLI	.95	≥ .95
RMSEA	.052	< .06

The SEM results indicated good model fit, with $\chi^2(51) = 112.84$, $p < .001$, $\chi^2/df = 2.21$, GFI = .96, AGFI = .94, CFI = .97, TLI = .95, and RMSEA = .052. All indices met or exceeded the recommended thresholds for acceptable to excellent fit, confirming that the hypothesized model adequately represented the data (Table 3).

Table 4

Total, Direct, and Indirect Path Coefficients in the Structural Model

Path	B	S.E	β	p
ACE → Cognitive Flexibility	–2.84	0.41	–.39	< .001
ACE → Somatization (Direct)	0.89	0.12	.31	< .001
Cognitive Flexibility → Somatization	–0.17	0.03	–.26	< .001
ACE → Somatization (Indirect via CF)	0.48	0.07	.10	< .001
ACE → Somatization (Total)	1.37	0.11	.41	< .001

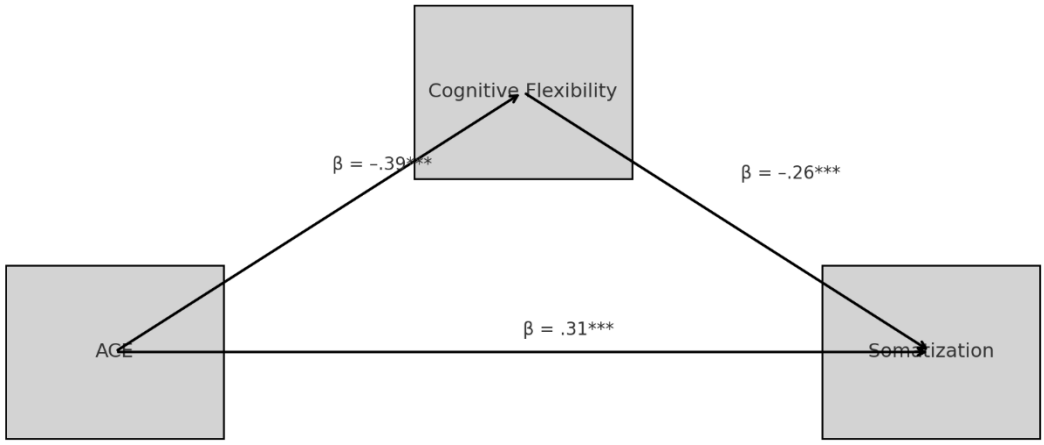
The direct path from ACEs to cognitive flexibility was significant (B = –2.84, β = –.39, p < .001), indicating that greater childhood adversity was associated with lower cognitive flexibility. The direct path from ACEs to somatization remained significant (B = 0.89, β = .31, p < .001), while cognitive flexibility was also a significant

JPPR
E-ISSN: 3041-8542

negative predictor of somatization (B = –0.17, β = –.26, p < .001). The indirect effect of ACEs on somatization through cognitive flexibility was also statistically significant (B = 0.48, β = .10, p < .001), supporting the mediating role of cognitive flexibility. The total effect of ACEs on somatization was B = 1.37 (β = .41, p < .001), suggesting that cognitive flexibility partially mediated the relationship between ACEs and somatic symptoms (Table 4).

Figure 1

Model with Beta Coefficients



4. Discussion and Conclusion

The present study aimed to explore the mediating role of cognitive flexibility in the relationship between adverse childhood experiences (ACEs) and somatization among adults in the United States. Using Pearson correlation and Structural Equation Modeling (SEM), we found significant positive correlations between ACEs and somatization, as well as a significant negative correlation between ACEs and cognitive flexibility. Additionally, cognitive flexibility was negatively associated with somatization. SEM analysis revealed that cognitive flexibility partially mediated the relationship between ACEs and somatic symptoms, supporting our hypothesized model. These findings suggest that ACEs may predispose individuals to heightened somatic symptomatology, in part through impairments in cognitive flexibility—a critical adaptive resource for managing distress.

The direct positive relationship between ACEs and somatization observed in this study aligns with a substantial body of literature indicating that early trauma is a strong predictor of physical symptom expression later in life. Individuals exposed to childhood abuse, neglect, or household dysfunction often demonstrate heightened interoceptive awareness, altered stress responses, and maladaptive emotion regulation, all of which may manifest somatically (Heule, 2025; Ma, 2025). For instance, Adamowicz et al. reported that specific types of childhood maltreatment were linked to elevated somatic symptoms, particularly among individuals with emotional dysregulation or alexithymia (Adamowicz et al., 2024). Similarly, Crismayanti et al. emphasized that adolescents exposed to trauma frequently somatize psychological distress as a culturally or socially acceptable means of expression (Crismayanti et al., 2024). These studies support the conclusion that somatization can function as a conduit for unresolved psychological trauma, reinforcing the need for early intervention.

The observed inverse relationship between ACEs and cognitive flexibility also supports findings from prior research. Childhood trauma can hinder the development of executive functioning, including the capacity to shift cognitive strategies, tolerate ambiguity, and reframe distressing experiences (Rostami & Mehdiabadi, 2024; Wang, 2024). Marmet et al., in a cohort study of emerging adults, found that greater exposure to adverse events was associated with elevated psychological distress and

JPPR
E-ISSN: 3041-8542

decreased psychological resilience, including impaired cognitive flexibility (Marmet et al., 2024). Similarly, Saadati et al. reported that adults with childhood trauma histories displayed rigid thought patterns and cognitive biases, which in turn contributed to both emotional dysregulation and psychosomatic responses (Saadati et al., 2023). Our results echo these findings by confirming that ACEs are negatively associated with flexibility in cognitive processing—a skill central to managing internal conflict and somatic awareness.

Importantly, the mediation analysis in this study provides empirical support for the theoretical model proposing that cognitive flexibility can buffer the adverse effects of ACEs on somatization. That is, while individuals with high ACE exposure are more likely to develop somatic symptoms, those who maintain greater cognitive flexibility demonstrate fewer symptoms, even in the presence of trauma history. This mediating effect is consistent with the cognitive-behavioral framework, which posits that flexible thinking enables individuals to reinterpret negative events, reduce catastrophic misinterpretation of bodily cues, and engage in more adaptive coping (Laube et al., 2024; Lee & Yoon, 2024). The results extend the findings of Mancone et al., who demonstrated that cognitive rigidity predicted increased somatic complaints among teachers under stress, and that flexible cognition mitigated this link (Mancone et al., 2024). Our findings further suggest that targeting cognitive flexibility may be a promising intervention point for trauma survivors presenting with somatic symptoms.

Additionally, this study's findings resonate with Lombardo's psychoanalytic and neuropsychological framework, which argues that maladaptive emotional processing and cognitive rigidity contribute to somatic presentations in clinical populations (Lombardo, 2024). The role of flexibility as a protective factor is also evident in Portilla-Saavedra et al.'s work, which highlighted how cognitive distortions resulting from early parental alienation could be moderated by adaptive cognitive reframing and resilience (Portilla-Saavedra et al., 2023). Likewise, Tao explored how childhood experiences informed artistic expression and cognitive transformation, further supporting the notion that internalized trauma can either stifle or shape adaptive psychological mechanisms depending on flexibility and self-reflection (Tao, 2023).

Our findings also align with culturally diverse perspectives on somatization and trauma. Nudin et al. noted that adult victims of intimate partner violence in Malaysia who had childhood trauma histories often expressed psychological pain through physical symptoms, a process intensified by cultural taboos against emotional disclosure (Nudin & Mohamed, 2023). Similarly, Pichel's review of young women who experienced sexual violence revealed a strong link between somatization and developmental trauma, especially when emotional processing was impaired (Pichel & Suryadi, 2021). These insights are consistent with our conclusion that trauma exposure, combined with cognitive rigidity, increases the likelihood of somatic symptomatology in adulthood.

The results of this study also offer indirect support to the neurodevelopmental literature. Studies such as that by Zhang et al. have shown that anxious attachment moderates the relationship between childhood trauma and pain symptoms, further suggesting that relational and emotional factors may exacerbate or buffer somatic experiences (Zhang et al., 2023). Heule's research similarly demonstrated that pain catastrophizing and negative affect mediated the relationship between ACEs and urogenital pain, aligning with our finding that cognitive factors play a critical intermediary role (Heule, 2025). Our use of SEM to model cognitive flexibility as a mediator provides a statistically rigorous extension to these findings, illustrating a clear pathway from trauma to somatic distress via cognitive mechanisms.

This study's contributions are also in harmony with Gardoki-Souto et al., who found that fibromyalgia patients frequently reported childhood trauma histories, and that trauma severity predicted pain intensity and emotional comorbidity (Gardoki-Souto et al., 2022). Such research supports the biopsychosocial model of somatization, which views symptom expression as an interaction of psychological trauma, cognitive processing deficits, and physiological dysregulation. By demonstrating that cognitive flexibility partially mediates the relationship between ACEs and somatic symptoms, the present study affirms the importance of integrative models in understanding complex clinical presentations.

Despite its contributions, this study has several limitations. First, the cross-sectional design precludes any definitive conclusions about causality. While the findings support a mediating role of cognitive flexibility, longitudinal studies are needed to confirm whether cognitive flexibility develops as a protective response or is inherently diminished by trauma. Second, the use of self-report measures introduces the potential for response bias, including social desirability and retrospective inaccuracies, particularly in recalling childhood experiences. Third, while the sample size was statistically adequate, it consisted entirely of U.S.-based participants, limiting the generalizability of findings

to other cultural contexts. Lastly, although the mediation model included cognitive flexibility, other possible mediators—such as emotional regulation, resilience, or attachment style—were not examined and may further clarify the pathways between ACEs and somatization.

Future studies should adopt longitudinal and experimental designs to establish temporal and causal relationships among ACEs, cognitive flexibility, and somatization. Such designs could help determine whether interventions that improve cognitive flexibility also result in reductions in somatic symptoms over time. Moreover, incorporating neurocognitive assessments and biomarkers of stress reactivity (e.g., cortisol, heart rate variability) would offer a more comprehensive understanding of the psychophysiological processes involved. Expanding research to include culturally diverse and clinical populations—such as trauma survivors, chronic pain patients, or individuals with somatic symptom disorder—could enhance the applicability and relevance of findings. Finally, future models could test multiple mediators or moderators simultaneously, such as emotional clarity, coping strategies, or social support, to build more robust explanatory frameworks.

From a practical perspective, these findings underscore the importance of incorporating cognitive flexibility training into therapeutic interventions for individuals with a history of childhood trauma. Mental health practitioners should consider integrating approaches that foster flexible thinking, such as cognitive restructuring, mindfulness-based cognitive therapy, and acceptance-based practices. Screening for ACEs in primary care and mental health settings could also help identify individuals at risk for somatic symptomatology. Furthermore, psychoeducation on the relationship between early trauma, cognitive processing, and physical symptoms may help reduce stigma and promote more holistic treatment planning. Healthcare systems should prioritize trauma-informed care models that recognize the interplay between psychological and physical health, offering integrated services that address both domains.

Authors' Contributions

Authors contributed equally to this article.

Declaration

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

Transparency Statement

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

Acknowledgments

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

Declaration of Interest

The authors report no conflict of interest.

Funding

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

Ethics Considerations

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

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JPPR
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