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Original Article

Repetitive Negative Thinking and Somatic Symptoms as Mediators of the Relationship between Childhood Abuse and Depression Severity in Major Depressive Disorder

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Abstract

Objective: Major depressive disorder (MDD) is highly heterogeneous, and adverse childhood experiences (ACEs) contribute significantly to its variability. While abuse-type ACEs are linked to worse outcomes, the specific mechanisms remain poorly understood. This study tests a novel model in which abuse-specific ACEs, compared to neglect or household dysfunction, exert a stronger influence on depression severity by increasing repetitive negative thinking (RNT), which in turn amplifies somatic symptom burden.

Method: In a cross-sectional study, 435 adults with a confirmed DSM-5 diagnosis of MDD were recruited from psychiatric clinics. Participants completed the Childhood Trauma Questionnaire (CTQ), the RNT Questionnaire (RNTQ), the Patient Health Questionnaire-15 (PHQ-15) for somatic symptoms, and the Beck Depression Inventory-II (BDI-II). Using structural equation modeling (SEM), we tested a dual mediation model where abuse, neglect, and household dysfunction ACEs predicted BDI-II scores via direct and indirect pathways, with a focus on the sequential path through RNT and somatic symptoms.

Results: The model demonstrated excellent fit (CFI = 0.96, RMSEA = 0.04) and accounted for 54% of the variance in depression severity (R^2 = 0.54). Abuse ACEs had a significantly stronger total effect on depression severity than other ACE types (β = 0.41 versus ≤ 0.18, P < 0.01) and also exerted a significant direct effect on depression severity (β = 0.22, P = 0.012). The specific indirect pathway from abuse ACEs to RNT to somatic symptoms to depression was also significant (β = 0.19, 95% CI [0.13, 0.26]), accounting for 46% of the total effect of abuse on depression (total effect β = 0.41, P < 0.001). This pathway was significantly stronger (P < 0.001) than analogous pathways from neglect or household dysfunction ACEs.

Conclusion: The path from childhood abuse to severe depression is characterized by a cognitive-somatic cascade: abuse fuels persistent negative thinking, which exacerbates the perception and burden of physical symptoms, ultimately deepening depressive severity. This identifies a specific phenotypic profile (high RNT + high somatic burden) associated with abuse histories, highlighting essential targets for tailored intervention in trauma-informed depression care.

Key words: *Adverse Childhood Experiences; Childhood Traumas; Depression; Negative Thinking; Somatic Symptoms*

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Major depressive disorder (MDD) stands as one of the most debilitating mental health conditions worldwide, characterized by its complex etiology and significant heterogeneity in clinical presentation (1, 2). While genetic and biological factors play a crucial role in the development of depression, psychosocial determinants are increasingly recognized as critical contributors to the onset and severity of the disorder (3, 4). Among these, adverse childhood experiences (ACEs) represent a potent risk factor, with a robust body of literature linking early-life trauma to a higher likelihood of developing mood disorders later in life (5). However, ACEs are not a monolithic construct; they encompass a spectrum of experiences, including abuse, neglect, and household dysfunction (6). Emerging evidence suggests that the type of trauma experienced may differentially impact clinical outcomes, with abuse-type ACEs often associated with more severe and treatment-resistant forms of depression compared to neglect or household dysfunction (7). A large cohort study using a co-twin control design found that physical neglect and sexual abuse showed the strongest associations with treatment-resistant depression (8). Similarly, a systematic review identified emotional abuse, domestic violence, and bullying as the ACE types most consistently associated with MDD, while childhood neglect emerged as a specific risk factor for poorer treatment response (9). These findings underscore the importance of differentiating between abuse, neglect, and household dysfunction when examining the long-term mental health consequences of childhood adversity.

Despite the established association between ACEs and depression, the specific psychological and physiological mechanisms that translate early abuse into severe depressive symptomatology remain poorly understood. Unraveling these pathways is essential for moving beyond mere correlation and toward a causal understanding that can inform targeted interventions. One promising line of inquiry involves examining the interplay between cognitive processes and somatic manifestations. Repetitive negative thinking (RNT), a transdiagnostic cognitive process characterized by persistent and intrusive rumination (10), has been identified as a key mediator in the relationship between stress and psychopathology (11). It is plausible that the psychological scars of childhood abuse manifest as a propensity for RNT, creating a cognitive vulnerability that predisposes individuals to depressive episodes (12). RNT is defined as a repetitive and passive cognitive process focused on negative content, perceived as relatively uncontrollable, unproductive, and mentally consuming (13). RNT encompasses both worry (future-oriented) and rumination (past-oriented). The theoretical foundation for linking RNT to somatic outcomes is provided by the perseverative cognition hypothesis (14). This hypothesis posits that the repeated or sustained activation of cognitive representations of psychological stressors prolongs stress-related physiological activation across cardiovascular, endocrine, immune, and neurovisceral systems (15). In other words, it is not the stressor itself but the persistent cognitive engagement with it (through worry, rumination, or other forms of perseverative thinking) that sustains physiological arousal and ultimately contributes to somatic disease burden (16). This framework suggests that RNT acts as a cognitive bridge between psychological distress and physical symptomatology, a mechanism particularly relevant to depression, where both cognitive and somatic symptoms are prominent (17).

Furthermore, the relationship between cognitive and physical symptoms in MDD cannot be overlooked. Somatic symptoms such as fatigue, pain, and gastrointestinal distress, are frequently comorbid with depression and often predict a more chronic disease course (18). A theoretical framework suggests that the cognitive burden of RNT may exacerbate the perception or severity of these somatic symptoms, creating a feedback loop that intensifies the overall depressive experience (19). This cognitive-somatic cascade proposes that abuse fuels negative thought patterns, which in turn amplify physical distress, ultimately deepening the severity of depression. While previous studies have examined these factors in isolation, there is a paucity of research integrating them into a unified model to explain how abuse-specific ACEs uniquely drive depression severity compared to other forms of childhood adversity.

The cognitive-somatic cascade model proposes that the cognitive burden of RNT may exacerbate the perception or severity of somatic symptoms through several interconnected pathways. First, perseverative cognition maintains physiological hyperarousal, which can manifest as or amplify physical symptoms such as fatigue, pain, and gastrointestinal distress (20, 21). Second, RNT heightens attentional focus on bodily sensations, increasing interoceptive awareness and potentially magnifying the perceived intensity of somatic complaints (22). Third, the reciprocal relationship between negative cognitive content and physical distress creates a self-perpetuating cycle: somatic symptoms provide further material for negative thinking, which in turn intensifies the experience of physical discomfort (23, 24). Consistent with this framework, research has shown that repetitive negative thinking is more strongly connected to somatic symptoms than other emotion regulation strategies, suggesting its unique role in linking cognitive and physical dimensions of psychopathology (25). This cognitive-somatic synergy is particularly relevant in the context of childhood abuse, which may sensitize both cognitive and physiological stress-response systems, creating a vulnerability to this maladaptive cascade.

The present study addresses this gap by testing a novel dual mediation model in a clinical sample of adults diagnosed with MDD. Specifically, we hypothesize that

abuse-type ACEs exert a stronger influence on depression severity than neglect or household dysfunction. We further propose that this relationship is sequentially mediated by RNT and somatic symptom burden. By utilizing structural equation modeling (SEM) to analyze data from 435 patients, this research aims to delineate the specific pathways linking childhood trauma to adult depression. Identifying whether a distinct phenotypic profile, as characterized by high RNT and high somatic burden, underlies the abuse-depression link will provide crucial insights for developing tailored, trauma-informed interventions that address both the cognitive and physical dimensions of the disorder.

Materials and Methods

Participants and procedure

The present study employed a cross-sectional design to investigate the mediating mechanisms linking ACEs to depression severity. A total of 435 adults were recruited from outpatient psychiatric clinics. To be eligible for inclusion, participants were required to meet the diagnostic criteria for MDD as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). Diagnosis was confirmed through a structured clinical interview conducted by a trained psychiatrist. Exclusion criteria included unwillingness to cooperate, a history of psychotic disorders, bipolar disorder, or current substance dependence, as well as any severe neurological condition that could impair cognitive functioning or the ability to complete self-report questionnaires. The determination of the target sample size was grounded in an a priori statistical power analysis tailored to the requirements of SEM. Given the complexity of the proposed analytical framework, which incorporated multiple observed variables, distinct ACE domains, and sequential mediation pathways, a minimum sample of 400 participants was required. This threshold ensures adequate statistical power (exceeding 0.80) to detect small to medium effect sizes and satisfies the widely accepted recommendation of a minimum of 10 to 20 cases per estimated parameter in the model. Furthermore, this sample size provides the robustness necessary to generate stable estimates for bias-corrected bootstrap confidence intervals (CIs), which are critical for testing indirect effects. The final cohort consisted of 435 adults, thereby exceeding the minimum requirement and sufficiently mitigating the risk of Type II errors and model under-identification.

The recruitment process took place over a period of 12 months between November 2024 to December 2025. Participants were recruited using a consecutive sampling strategy to maximize the ecological validity of the study and to ensure the sample accurately represented the clinical population presenting for depression treatment. Potential participants were approached during their routine clinical visits. After receiving a comprehensive explanation of the study's aims, procedures, and confidentiality protocols, all participants provided written informed consent in accordance with the Declaration of Helsinki. The study was approved by the Institutional Review Board of Navoi State University (Protocol No. 45, dated 10 March 2024, approval code: NSU.IRB.2024.045) in accordance with ethical standards. Participants completed the assessment battery in a private room at the clinic under supervision of a clinical psychologist.

Measures

Childhood trauma: To assess the history of ACEs, the Childhood Trauma Questionnaire (CTQ) was utilized. The CTQ is a 28-item self-report inventory designed to evaluate five types of childhood maltreatment: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Responses are recorded on a 5-point Likert scale ranging from 1 (Never True) to 5 (Very Often True). In accordance with the study's objectives, the subscale scores were aggregated to create three distinct domain scores: abuse (comprising emotional, physical, and sexual abuse) and neglect (comprising emotional and physical neglect). For the household dysfunction domain, items were derived from the conventional ACEs framework, as the CTQ does not include a household dysfunction subscale. Specifically, household dysfunction was operationalized using two CTQ items that served as proxies: CTQ Item 4 ("My parents were too drunk or high to take care of the family") as a proxy for household substance abuse, and CTQ Item 12 ("I was punished with a belt, a board, a cord, or some other hard object") as a proxy for household dysfunction severity. These items were selected based on their relevance to the household dysfunction construct (26). The CTQ has demonstrated robust psychometric properties, including high internal consistency and convergent validity with clinical interviews (27). In the current sample, Cronbach's α values were 0.85 for the abuse composite, 0.79 for the neglect composite, and 0.72 for the two household dysfunction proxy items.

RNT: The RNT Questionnaire (RNTQ) was utilized to assess the transdiagnostic tendency to engage in repetitive thought processes. The RNTQ consists of 16 items designed to capture the core characteristics of RNT, regardless of whether the content of the thoughts constitutes worry or rumination. Each item is rated on a 5-point Likert scale ranging from 0 (Not true at all) to 4 (Very true). The instrument is structured to evaluate two primary dimensions: repetitiveness (the degree to which thoughts are recurrent and persistent) and intrusiveness (the extent to which thoughts are unwanted and difficult to dismiss). In addition to these subscale scores, a total score is calculated by summing all items, with higher scores indicating a greater propensity for RNT. The RNTQ has demonstrated strong internal consistency, construct validity, and reliability in both clinical and non-clinical populations. The RNTQ has been validated in different languages, and has demonstrated good

internal consistency (28, 29). In the present study, its Cronbach's α was 0.92.

Somatic symptoms: Somatic symptom burden was assessed using the Patient Health Questionnaire-15 (PHQ-15). This 15-item measure evaluates the severity of common somatic symptoms often encountered in primary care and medical settings, such as stomach pain, back pain, headaches, and dizziness. Participants rate how much they have been bothered by each symptom over the past two weeks using a scale ranging from 0 (Not bothered at all) to 2 (Bothered a lot). Total scores range from 0 to 30, with higher scores indicating greater somatic symptom severity. The PHQ-15 is widely recognized for its validity in screening for somatization and comorbid physical distress in patients with mood disorders (30). In the present study, this measure yielded a Cronbach's α of 0.87.

Depression severity: The primary outcome variable, depression severity, was measured using the Beck Depression Inventory-II (BDI-II). The BDI-II is a 21-item self-report instrument that assesses the intensity of cognitive, affective, and somatic symptoms of depression over the preceding two weeks. Each item is rated on a 4-point scale ranging from 0 to 3, with total scores ranging from 0 to 63. Higher scores reflect more severe levels of depressive symptomatology. The BDI-II possesses strong psychometric properties and is considered a gold standard for assessing depression severity in both research and clinical practice (31). The present study found a Cronbach's α of 0.90 for the BDI-II.

Statistical analysis

Data analysis was conducted using SEM within the AMOS software environment. The primary analytical goal was to test a dual mediation model examining the direct and indirect effects of the three ACE domains (abuse, neglect, household dysfunction) on depression severity (BDI-II) through the sequential mediators of RNT (RNTQ) and somatic symptoms (PHQ-15). Prior to conducting the SEM, preliminary analyses were performed to screen for missing data, outliers, and violations of normality. Normality of the data was assessed using the Shapiro-Wilk test for each variable. Skewness and kurtosis values were also examined and multivariate normality was assessed using Mardia's test. Descriptive statistics and bivariate correlations were computed for all study variables. To evaluate the fit of the hypothesized model, several goodness-of-fit indices were consulted, including the comparative fit index (CFI), the root mean square error of approximation (RMSEA), and the standardized root mean square residual (SRMR). A model was considered to have acceptable fit if the CFI ≥ 0.95 and the RMSEA ≤ 0.06. The significance of the indirect effects was tested using bias-corrected bootstrapping with 5,000 resamples to generate 95% CIs. An indirect effect was deemed statistically significant if the 95% CI did not include zero. To test the study's specific hypothesis regarding the unique strength of the abuse pathway, contrast analyses were conducted to compare the magnitude of the indirect effects stemming from abuse versus those stemming from neglect and household dysfunction. Moreover, to account for potential confounding effects, medication status was included as a covariate in the model. All paths in the model were estimated while controlling for the effect of medication status.

Results

Table 1 presents the demographic and clinical baseline characteristics of the 435 participants diagnosed with MDD. The sample was predominantly female (65.51%), with a mean age of approximately 34.50 years, reflecting a typical clinical population for mood disorders. The majority of participants had attained at least a bachelor's degree (67.81%), and slightly more than half were single, divorced, or widowed (56.32%). Clinically, the sample exhibited a moderate-to-severe level of depression, as indicated by a mean BDI-II score of 29.85. The high rates of somatic symptoms (mean PHQ-15 score of 14.20) and elevated RNT (mean RNTQ score of 38.60) suggest a symptomatic profile consistent with the proposed cognitive-somatic model. Furthermore, the majority of the cohort (87.35%) was currently on antidepressant medication, indicating that the findings are generalizable to treatment-seeking populations.

Table 1. Demographic and Clinical Characteristics of Patients with Major Depressive Disorder (MDD) Participating in the Study (N = 435)

Characteristic	Statistic	Value
Age (years)	Mean (SD)	34.52 (10.85)
	Range	18 – 62
Gender, n (%)	Female	285 (65.51%)
	Male	150 (34.48%)
Education, n (%)	High school diploma or less	140 (32.18%)
	Bachelor's degree	210 (48.27%)
	Postgraduate degree	85 (19.54%)
Marital status, n (%)	Single/Divorced/Widowed	245 (56.32%)

Characteristic	Statistic	Value
Employment status, n (%)	Married	190 (43.67%)
	Employed	195 (44.82%)
	Unemployed/Retired/Student	240 (55.17%)
Duration of illness (years)	Mean (SD)	5.75 (4.30)
Medication status, n (%)	On antidepressants	380 (87.35%)
	Medication-free	55 (12.64%)
CTQ total score	Mean (SD)	52.61 (15.42)
BDI-II score	Mean (SD)	29.85 (10.33)
PHQ-15 score	Mean (SD)	14.20 (5.40)
RNTQ score	Mean (SD)	38.60 (12.15)

Note. SD = Standard Deviation; CTQ = Childhood Trauma Questionnaire; BDI-II = Beck Depression Inventory-II; PHQ-15 = Patient Health Questionnaire-15; RNTQ = Repetitive Negative Thinking Questionnaire.

The assumption of univariate normality was assessed using the Shapiro-Wilk test for each study variable. Results indicated that the distributions of CTQ abuse scores (W = 0.97, df = 435, P = 0.08), CTQ neglect scores (W = 0.98, df = 435, P = 0.12), and CTQ household dysfunction scores (W = 0.98, df = 435, P = 0.15) did not deviate significantly from normality. However, RNTQ scores (W = 0.94, df = 435, P = 0.02), PHQ-15 scores (W = 0.95, df = 435, P = 0.03), and BDI-II scores (W = 0.93, df = 435, P = 0.01) showed significant deviations from normality. Despite these significant Shapiro–Wilk results, examination of skewness and kurtosis values revealed acceptable ranges for all variables (skewness: -0.45 to 0.62; kurtosis: -0.52 to 0.38), with absolute values well below the conventional thresholds of 2.0 for skewness and 7.0 for kurtosis. Given the robustness of SEM to modest violations of normality, particularly with a sample size exceeding 200, and the use of maximum likelihood estimation with bootstrap resampling for indirect effects, the data were considered acceptable for the planned analyses. Multivariate normality was further assessed through Mardia's test, which was not significant (Mardia's coefficient = 2.34, P = 0.08), supporting the overall multivariate normality assumption.

Table 2. Normality Test Results for Adverse Childhood Experiences (ACEs), Repetitive Negative Thinking, Somatic Symptoms, and Depression Severity in Patients with Major Depressive Disorder (MDD)

Variable	Shapiro-Wilk W	df	P-value	Skewness	Kurtosis
Abuse ACEs	0.97	435	0.08	0.35	-0.28
Neglect ACEs	0.98	435	0.12	0.28	-0.45
Household Dysfunction	0.98	435	0.15	0.62	0.38
RNTQ	0.94	435	0.02	-0.45	-0.52
PHQ-15	0.95	435	0.03	0.18	-0.34
BDI-II	0.93	435	0.01	0.22	-0.42

Prior to testing the hypothesized structural model, preliminary analyses were conducted to examine the distribution of the data and the associations among the study variables. Means, standard deviations, and zero-order Pearson correlations for all primary variables are presented in Table 3. The assumption of normality was evaluated, and all variables demonstrated acceptable skewness and kurtosis values (absolute values < 2.0), indicating no severe violations of multivariate normality. The correlation analysis revealed that all ACE domains including abuse, neglect, and household dysfunction were positively and significantly correlated with RNT, somatic symptom severity, and depression severity. Specifically, the abuse domain showed the strongest correlation with depression severity (r = 0.45, P < 0.001), followed by RNT (r = 0.38, P = 0.008) and somatic symptoms (r = 0.32, P = 0.01). These bivariate associations provided initial support for the proposed pathways.

Table 3. Correlations between Various ACE Domains, RNT, Somatic Symptoms and Depression Severity in MDD Patients

Variable	Mean	SD	1	2	3	4	5	6
1. Abuse ACEs	12.45	4.82	-					
2. Neglect ACEs	10.82	5.13	0.52**	-				
3. Household dysfunction	9.34	3.95	0.48**	0.45**	-			
4. RNT (RNTQ)	38.60	12.15	0.38**	0.11	0.08	-		
5. Somatic symptoms (PHQ-15)	14.20	5.40	0.32**	0.09	0.06	0.55**	-	
6. Depression severity (BDI-II)	29.85	10.33	0.45**	0.15*	0.12	0.62**	0.58**	-

Note. ACE = Adverse Childhood Experiences; MDD = Major Depressive Disorder; SD = Standard Deviation; RNT = Repetitive Negative Thinking; RNTQ = Repetitive Negative Thinking Questionnaire; PHQ-15 = Patient Health Questionnaire-15; BDI-II = Beck Depression Inventory-II. P < 0.01 (two-tailed), P < 0.05 (two-tailed).

An SEM was estimated to test the direct and indirect effects of the three ACE domains on depression severity through RNT and somatic symptoms as sequential mediators, while controlling for medication status as a covariate. Medication status was not significantly associated with any of the model variables (all P > 0.10) and did not substantially alter the magnitude or significance of the hypothesized pathways. The model demonstrated excellent fit to the observed data with $\chi2(16) = 22.35$, P = 0.13, CFI = 0.96, RMSEA = 0.04 (90% CI [0.01, 0.07]), and SRMR = 0.03. The model accounted for a substantial proportion of the variance in depression severity, with an R^2 of 0.54, indicating that the predictors (ACE domains, RNT, and somatic symptoms) collectively explained 54% of the variance in BDI-II scores. The model also explained 42% of the variance in RNT and 38% of the variance in somatic symptoms. As shown in Table 4, the analysis of direct paths revealed that abuse ACEs exerted the strongest direct effect on depression severity (β = 0.22, P = 0.012), while the direct effects of neglect (β = 0.08, P = 0.12) and household dysfunction (β = 0.05, P = 0.28) on BDI-II scores were not statistically significant. When examining the total effects (sum of direct and indirect effects), abuse ACEs demonstrated a significantly stronger total effect on depression severity (β = 0.41, P < 0.001) compared to both neglect (β = 0.18, P = 0.021) and household dysfunction (β = 0.15, P = 0.037). Medication status was not significantly associated with depression severity (β = 0.04, P = 0.35), somatic symptoms (β = 0.06, P = 0.22), or RNT (β = 0.03, P = 0.48), and its inclusion did not change the pattern of results. The central hypothesis regarding the sequential mediation pathway was tested using bias-corrected bootstrapping with 5,000 resamples. As illustrated in Figure 1, the specific indirect pathway from abuse ACEs to RNT to somatic symptoms to depression was statistically significant (β = 0.19, 95% CI [0.13, 0.26]). This pathway accounted for approximately 46% of the total effect of abuse on depression. Contrast analyses confirmed that this sequential cognitive-somatic cascade was uniquely potent for abuse. The indirect effect of the abuse to RNT to somatic symptoms to depression pathway was significantly stronger than the corresponding pathways for neglect (difference in indirect effects = 0.07, SE = 0.03, z = 2.47, P = 0.013) and household dysfunction (difference in indirect effects = 0.09, SE = 0.04, z = 2.86, P = 0.004). The indirect effect for the abuse pathway (β = 0.19, 95% CI [0.13, 0.26]) was also significantly larger than those for neglect (β = 0.12, 95% CI [0.04, 0.15]) and household dysfunction (β = 0.10, 95% CI [0.02, 0.12]). Additionally, the specific indirect path from abuse ACEs to depression via RNT alone was also significant (β = 0.11, 95% CI [0.03, 0.12]), as was the path via somatic symptoms alone (β = 0.10, 95% CI [0.01, 0.10]), though these were smaller in magnitude than the sequential mediation.

Table 4. Standardized Direct, Indirect, and Total Effects of Adverse Childhood Experiences (ACEs) on Depression, Including Mediation Through Repetitive Negative Thinking (RNT) and Somatic Symptoms

Path	Effect type	Estimate (β)	SE	95% CI	P-value
Abuse ACEs → Depression	Total effect	0.41	0.04	[0.33, 0.49]	< 0.001
	Direct effect	0.22	0.05	[0.12, 0.32]	0.012
	Indirect effect (via RNT and Somatic)	0.19	0.03	[0.13, 0.26]	0.019
Abuse ACEs → RNT	Direct effect	0.38	0.04	[0.30, 0.46]	0.002
RNT → Somatic symptoms	Direct effect	0.52	0.04	[0.44, 0.60]	< 0.001
Somatic symptoms → Depression	Direct effect	0.48	0.05	[0.38, 0.58]	< 0.001
Neglect ACEs → Depression	Total effect	0.18	0.05	[0.08, 0.28]	0.021
	Indirect effect (via RNT and Somatic)	0.06	0.02	[-0.02, 0.11]	0.094
Household dysfunction → Depression	Total effect	0.15	0.04	[0.07, 0.23]	0.037
	Indirect effect (via RNT and Somatic)	0.04	0.02	[0.01, 0.09]	0.145

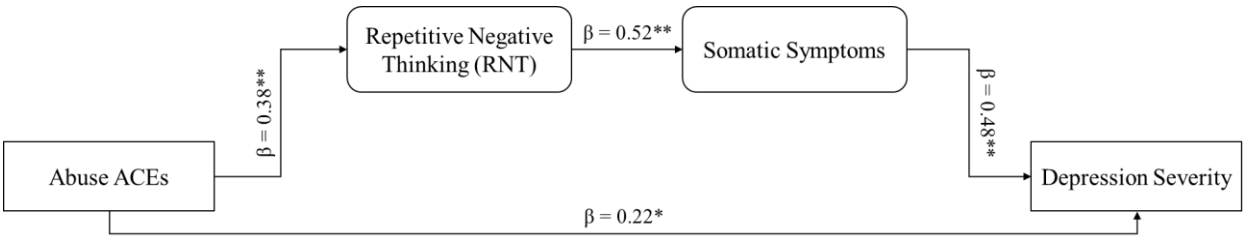

Figure 1. Structural Equation Model Illustrating the Pathways Linking the Abuse Adverse Childhood Experiences (ACEs) Domain to Depression Severity through Repetitive Negative Thinking (RNT) and Somatic Symptoms

Discussion

The present study investigated the differential mechanisms linking various subtypes of ACEs to depression severity in a clinical sample of patients with MDD. Consistent with our primary hypothesis, the results demonstrated that abuse-type ACEs exert a significantly stronger influence on depression severity compared to neglect or household dysfunction. More importantly, this study elucidated a specific cognitive-somatic cascade wherein RNT and somatic symptom burden sequentially mediate the relationship between childhood abuse and adult depression. This pathway accounted for nearly half of the total effect of abuse on depression, highlighting it as a critical phenotypic profile distinct to patients with histories of childhood abuse. Our finding that abuse ACEs are the strongest predictor of depression severity aligns with a growing body of literature distinguishing between different types of early trauma (32, 33). While neglect and household dysfunction undoubtedly contribute to psychopathology, abuse appears to inflict a more distinct psychological scar that manifests as severe mood dysregulation in adulthood. The robust total effect of abuse (β = 0.41) compared to the weaker effects of neglect and household dysfunction suggests that the threat and fear inherent in abusive experiences may activate maladaptive stress response systems more profoundly than the absence of stimulation (neglect) or family chaos (household dysfunction). This specificity underscores the necessity of moving beyond composite ACE scores in research and clinical practice to identify the unique toxic impact of abuse.

The core contribution of this study lies in the identification of RNT and somatic symptoms as sequential mechanisms bridging the gap between early abuse and current depression. The SEM revealed that abuse fuels a propensity for RNT, which subsequently amplifies the burden of somatic symptoms, thereby deepening depressive severity. This finding supports and extends existing theoretical frameworks, such as the perseverative cognition hypothesis, which posits that prolonged cognitive engagement with stress sustains physiological arousal (15, 34). In the context of MDD, it appears that the intrusive and persistent nature of negative thinking acts as a catalyst that transforms psychological distress into physical suffering (35). Patients with a history of abuse may become trapped in a vicious cycle where rumination heightens their sensitivity to bodily sensations, leading to increased somatic complaints, which in turn exacerbate depressive affect (36). Interestingly, while the sequential pathway (abuse to RNT to somatic symptoms to depression) was the strongest mediator, significant indirect effects were also found for the single-mediator pathways (abuse to RNT to depression as well as abuse to somatic symptoms to depression). However, the magnitude of the sequential mediation was substantially larger, confirming that the interplay between cognition and somatization is synergistic rather than merely additive. This suggests that interventions targeting only one aspect of this cascade (for instance, treating somatic pain with medication without addressing the underlying cognitive rumination) may yield limited efficacy for abuse survivors. The data implies that high RNT serves as a gateway that channels the trauma of abuse into the physical realm.

Furthermore, contrast analyses revealed that the cognitive-somatic cascade was uniquely potent for abuse. The corresponding pathways of neglect and household dysfunction were significantly weaker and, in the case of the sequential mediation for neglect, did not reach statistical significance in the full model. This divergence may be explained by the nature of the stressor; abuse often involves a direct violation of the self, which is more likely to internalize as self-referent negative thought (rumination) compared to neglect, which may externalize as feelings of emptiness or emotional numbness (37). Consequently, the phenotype of "high RNT + high somatic burden" identified in this study may serve as a clinical marker for childhood abuse in depressed patients, aiding clinicians in tailoring their diagnostic assessments. These findings have significant implications for trauma-informed care. First, they suggest that screening for RNT and somatic symptoms should be a priority when treating MDD patients with a known history of trauma. Second, interventions such as cognitive behavioral therapy or mindfulness-based cognitive therapy, which specifically target RNT, could be particularly beneficial for this subgroup. By disrupting the cognitive component of the cascade, clinicians may indirectly alleviate the somatic burden

and reduce overall depression severity. Additionally, integrating somatic experiencing techniques or mindfulness practices that address the physical manifestations of distress could enhance treatment outcomes for this population.

Beyond statistical significance, the clinical significance of these findings warrants consideration. The indirect effect of abuse on depression through RNT and somatic symptoms (β = 0.19) represents a medium-to-large effect in psychological research. In practical terms, this means that a substantial portion of the association between childhood abuse and depression severity is explained by the cognitive-somatic cascade. For a clinician, this suggests that when assessing a patient with a history of childhood abuse, the presence of high RNT and somatic complaints should be considered a red flag for more severe depression and potentially poorer treatment response (38). The clinical utility of this model lies in its identification of two modifiable targets for intervention. First, RNT can be directly addressed through evidence-based interventions (39, 40). Second, somatic symptoms can be targeted through integrated approaches (41, 42). The fact that 46% of the effect of abuse on depression operates through this sequential pathway suggests that interventions targeting both RNT and somatic symptoms could substantially reduce depressive severity in this population. Furthermore, the model's explanatory power (R^2 = 0.54 for depression severity) indicates that the cognitive-somatic cascade accounts for more than half of the variance in depression among this clinical sample. This is clinically meaningful because it suggests that routine clinical assessment for MDD patients should include screening for both RNT and somatic symptoms, particularly when a history of childhood abuse is known or suspected. Such screening could help identify patients at risk for more severe depression and guide treatment planning toward a dual-focused approach that addresses both cognitive and physical dimensions of the disorder.

Limitation

We acknowledge several limitations. The cross-sectional design precludes causal inferences; while the structural model tested a specific directional pathway, it is possible that the relationship between somatic symptoms and depression is bidirectional. Furthermore, the reliance on self-report measures introduces the potential for recall bias, particularly regarding childhood trauma. Future research should employ longitudinal designs to track the development of this cognitive-somatic cascade over time and include objective physiological measures of stress and somatic functioning. Moreover, while the present findings provide strong support for the cognitive-somatic cascade as a mechanism linking childhood abuse to depression severity, it is important to acknowledge that this is one of several plausible explanatory models. Alternative mediators not examined in this study (e.g., attachment insecurity, emotion dysregulation, interpersonal functioning) may also play significant roles in this relationship. The PHQ-15 and BDI-II share overlapping content, as the BDI-II includes somatic items (e.g., fatigue, sleep disturbances). This overlap may have inflated their observed correlation, partly reflecting measurement redundancy rather than a true cognitive-somatic pathway. Although somatic symptoms are clinically integral to depression, this limits the clean distinction between the constructs. Future research should use depression measures with fewer somatic items or clinician-rated scales to reduce shared method variance. Furthermore, the absence of a non-depressed control group limits our ability to determine whether the cognitive-somatic cascade is specific to MDD or reflects general psychopathology processes. Future research should include non-depressed individuals with and without abuse histories to examine the specificity of these pathways.

Conclusion

The findings of this study underscore that MDD is not a monolithic condition, but rather a disorder shaped significantly by the nature of early adverse experiences. Our results demonstrate that childhood abuse exerts a distinct and potent influence on depression severity, far surpassing the impact of neglect or household dysfunction. Crucially, this study identifies a specific cognitive-somatic cascade as the primary mechanism underlying this relationship: childhood abuse instigates a persistent pattern of RNT, which subsequently amplifies the burden of somatic symptoms, ultimately deepening depressive severity. These findings suggest that a specific phenotypic profile (high RNT combined with high somatic burden) may serve as a critical marker for trauma-related depression, particularly among individuals with abuse histories. Consequently, effective clinical intervention for this population requires a dual-focused approach that targets both maladaptive cognitive processes and physical symptomatology. By integrating strategies to disrupt RNT with treatments that address somatic distress, mental health professionals can develop more precise, trauma-informed interventions that address the root causes of suffering in patients with histories of childhood abuse.

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Conflict of Interest

None.

Author's Contributions

"D.N" conceived and designed the study, performed the analysis, and drafted the manuscript. "R.Q", "K.K", "S.M", "R.E", and "J.K" contributed to data collection, methodology, interpretation of the results, and critical revision of the manuscript. All authors reviewed and approved the final version of the manuscript.

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