

Association of Childhood Adversity with Emotion Regulation and Symptom Severity in Adults with Generalized Anxiety Disorder: A Prospective Observational Study

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Abstract

Background: Childhood adversity may influence the severity and course of generalized anxiety disorder (GAD), potentially through impaired emotion regulation.

Methods: This prospective observational study included 200 adults with GAD. Childhood adversity was assessed using the Childhood Trauma Questionnaire, emotion regulation using the DERS, and anxiety severity using HAM-A and GAD-7. Participants were reassessed after 8–12 weeks. Correlation, multivariable regression, and exploratory mediation analyses were performed.

Results: Greater childhood adversity was associated with higher DERS, HAM-A, GAD-7, depressive symptoms, and functional impairment. CTQ correlated with DERS ($p=0.360$) and HAM-A ($p=0.360$), while DERS showed a stronger association with HAM-A

($p=0.608$). DERS remained independently associated with baseline anxiety severity, and both CTQ and DERS predicted greater residual anxiety at follow-up. Exploratory mediation suggested partial mediation of the adversity–anxiety relationship through emotion dysregulation.

Conclusion: Childhood adversity and emotion dysregulation were associated with greater and more persistent anxiety severity in adults with GAD.

Keywords: Generalized Anxiety Disorder, Childhood Adversity, Emotion Regulation, Childhood Trauma, Anxiety Severity.

Introduction

Generalized anxiety disorder (GAD) is a persistent, chronic psychiatric disorder marked by uncontrollable worry, psychological and somatic symptoms, and a

disruption in functioning. Its clinical presentation is a result of the interplay between stressors in the present and developmental vulnerability. The impact of childhood adversity has been linked to a wide spectrum of psychiatric outcomes in adulthood, such that exposure to a greater number of adverse childhood events has been shown to be associated with increased long-term mental health burden¹. Epidemiological evidence also indicates that early adverse events can lead to vulnerability to later anxiety and depressive disorders, especially in the presence of later stress².

Emotional, physical, and sexual abuse and emotional and physical neglect are all forms of childhood adversity. The Childhood Trauma Questionnaire-Short Form (CTQ-SF) allows the assessment of these separate domains and an overall estimate of the burden of childhood adversity³. A potential route from these experiences to later anxiety is that emotion regulation is impaired. The Difficulties in Emotion Regulation Scale (DERS) is a measure of multiple facets of difficulty in emotion regulation, such as nonacceptance, impairment in goal-directed functioning, impulse-control problems, limited awareness and clarity, and diminished access to adaptive strategies⁴.

Emotion-regulation disturbance seems to be especially relevant to GAD. Mennin et al. found that people with GAD were more emotionally intense, less emotionally understanding and less emotionally regulated than comparison participants⁵. Turk et al. also showed that the emotion-regulation deficits of GAD are not the same as those of social anxiety disorder, indicating that emotion-regulation deficits may play a specific role in generalized anxiety symptoms⁶.

Additionally, the effects of childhood adversity and emotion dysregulation might interact in shaping

symptom burden. Huh et al. found that childhood trauma was correlated with increased anxiety and depressive symptoms in adulthood and that some of this relationship was mediated by maladaptive emotion-regulation strategies⁷. These mechanisms are clinically relevant, as treatments targeting emotion-regulation mechanisms have been shown to decrease symptoms in patients with GAD⁸.

Despite these findings, prospective studies investigating the relationship between childhood adversity, multi-dimensional emotion-regulation difficulties, and symptom severity in the same GAD sample remain limited. The present study therefore examined the relationship between childhood adversity and emotion-regulation difficulties and anxiety severity in adults with GAD, symptom change over an 8–12-week period, and whether emotion dysregulation partially mediated the relationship between childhood adversity and anxiety severity.

Methods

Study design and setting

The study was a prospective observational cohort study carried out in the Department of Psychiatry, District Hospital, Rampur from May 2024 to May 2026. 200 adults with clinically diagnosed generalized anxiety disorder (GAD) were recruited for the study and were assessed at baseline and followed up approximately 8–12 weeks later.

Patient selection

Eligible individuals were adults (18–65 years) who met DSM-5/DSM-5-TR criteria for GAD, had been seen in psychiatric care, had completed the necessary psychometric assessments, and had provided sufficient background data on childhood adversity, emotion regulation, and anxiety severity.

Patients were excluded if they had a current psychotic disorder, a manic or hypomanic episode, a severe substance-use disorder that interfered with assessment, a major neurocognitive disorder, an intellectual disability that made reliable assessment difficult, a severe neurological illness that made questionnaire completion difficult, a substance/medication-induced anxiety disorder, an acute medical condition in which anxiety was primarily due to the medical illness, a psychiatric emergency requiring immediate stabilization, unreliable recall of childhood experiences, or inability to participate in follow-up. There were no exclusion criteria for mild-to-moderate depressive symptoms or for common depressive comorbidity.

Clinical and psychometric evaluation

Baseline demographic and clinical data consisted of age, sex, **residence**, marital status, education, employment, socioeconomic status, family structure, age at onset of GAD, duration of illness, previous psychiatric treatment, current psychotropic treatment, psychotherapy/counselling, family psychiatric history, sleep disturbance, panic and somatic symptoms, and functional impairment.

Childhood adversity was measured by the Childhood Trauma Questionnaire-Short Form (CTQ-SF), which consists of five domains: emotional abuse, physical abuse, sexual abuse, emotional neglect and physical neglect. The total score for the CTQ was the sum of the five domain scores. Additionally, participants were classified as having a low, moderate, or high childhood-adversity burden based on the overall severity of the CTQ. The 36-item Difficulties in Emotion Regulation Scale (DERS) was used to assess emotion-regulation difficulties, with scores for nonacceptance, goals, impulse, awareness, strategies, clarity, and total DERS.

The Hamilton Anxiety Rating Scale (HAM-A) and the Generalized Anxiety Disorder-7 (GAD-7) were used to assess the severity of anxiety. The Patient Health Questionnaire-9 (PHQ-9) was used to measure depressive symptoms and functional impairment was measured on a 0–30 scale. Medical and selected psychiatric comorbidities were also recorded.

Follow-up assessment

The participants were reevaluated about 8–12 weeks after baseline. Follow-up measures were HAM-A, GAD-7 and DERS total scores. Treatment exposure during follow-up, including SSRI/SNRI treatment, short-term use of benzodiazepines, psychological intervention, medication adherence, and attendance at visits, was documented. The changes in HAM-A and GAD-7 were obtained by subtracting follow-up scores from baseline scores, with positive scores reflecting improvement.

Statistical analysis

Data for continuous variables were presented as mean $\pm$ SD or median [IQR] and for categorical variables as n (%). For continuous variables, ANOVA or Kruskal-Wallis tests were used for group comparisons, and for categorical variables, chi-square tests were used. Spearman's correlation was used to evaluate the association among the CTQ, DERS, HAM-A, GAD-7, PHQ-9, and functional impairment. Independent correlates of baseline and follow-up HAM-A were assessed using multivariable linear regression, and the results are presented as regression coefficients, 95% confidence intervals, and p values. Bootstrapping was used to perform an exploratory mediation analysis of the indirect pathway CTQ $\rightarrow$ DERS $\rightarrow$ HAM-A. All tests were two-tailed and $p < 0.05$ was used to indicate statistical significance.

Results

Participant characteristics and childhood adversity profile

The study included 200 adults with generalized anxiety disorder; the mean age was 36.2 ± 9.6 years and 125 (62.5%) were female. Childhood-adversity burden was classified as low in 120 (60.0%), moderate in 49 (24.5%), and high in 31 (15.5%) participants. Baseline demographic and clinical characteristics across adversity groups are presented in Table 1. Family history of anxiety or depression differed across groups, whereas most other demographic and treatment-history variables were broadly comparable.

Table 1: Baseline demographic and clinical characteristics according to childhood-adversity burden

Characteristic	Category	Low (n=120)	Moderate (n=49)	High (n=31)	Test statistic	p-value
Age, years	—	36.4 ± 10.2	35.9 ± 9.2	35.8 ± 8.4	$F=0.07$	0.928
Sex, n (%)	Female	73 (60.8%)	32 (65.3%)	20 (64.5%)	$\chi^2=0.36$	0.835
	Male	47 (39.2%)	17 (34.7%)	11 (35.5%)		
Marital status, n (%)	Unmarried	28 (23.3%)	4 (8.2%)	5 (16.1%)	$\chi^2=9.38$	0.052
	Married	78 (65.0%)	42 (85.7%)	25 (80.6%)		
	Separated/Divorced/Widowed	14 (11.7%)	3 (6.1%)	1 (3.2%)		
Education level, n (%)	Primary or less	13 (10.8%)	5 (10.2%)	5 (16.1%)	$\chi^2=4.02$	0.675
	Secondary	42 (35.0%)	18 (36.7%)	13 (41.9%)		
	Higher secondary	29 (24.2%)	9 (18.4%)	8 (25.8%)		
	Graduate or above	36 (30.0%)	17 (34.7%)	5 (16.1%)		
Duration of GAD, years	—	4.6 [2.7–7.0]	4.6 [3.1–6.6]	7.1 [5.1–9.4]	$H=11.53$	0.003
Age at GAD onset, years	—	30.6 ± 10.8	30.5 ± 9.5	28.0 ± 9.0	$F=0.83$	0.439
Depressive disorder/significant depressive symptoms, n (%)	Yes	35 (29.2%)	15 (30.6%)	16 (51.6%)	$\chi^2=5.78$	0.056
Previous psychiatric treatment, n (%)	Yes	58 (48.3%)	24 (49.0%)	18 (58.1%)	$\chi^2=0.96$	0.619
Family history of anxiety/depression, n (%)	Yes	24 (20.0%)	21 (42.9%)	8 (25.8%)	$\chi^2=9.34$	0.009

Values are mean $\pm$ SD, median [IQR], or n (%), as appropriate. F, one-way analysis of variance statistic; H, Kruskal-Wallis statistic; χ^2 , chi-square statistic; GAD, generalized anxiety disorder.

Psychometric findings and emotion regulation

CTQ scores increased across the prespecified adversity categories, with emotional neglect and emotional abuse

showing prominent gradients. Greater adversity burden was accompanied by higher DERS total and subscale scores, greater anxiety and depressive symptom severity, and greater functional impairment (Table 2).

Table 2: Psychometric findings according to childhood-adversity burden

Measure	Low (n=120)	Moderate (n=49)	High (n=31)	Test statistic	p- value	Effect size (ϵ^2)
CTQ total	30 [27–34]	46 [43–51]	73 [66–82]	H=152.67	<0.001	0.765
CTQ emotional abuse	5 [5–8]	11 [9–14]	18 [15–22]	H=107.40	<0.001	0.535
CTQ physical abuse	5 [5–5]	8 [5–11]	12 [9–16]	H=84.49	<0.001	0.419
CTQ sexual abuse	5 [5–5]	5 [5–9]	8 [5–12]	H=41.03	<0.001	0.198
CTQ emotional neglect	5 [5–8]	12 [7–15]	20 [18–24]	H=105.26	<0.001	0.524
CTQ physical neglect	5 [5–6]	7 [5–12]	15 [13–20]	H=90.98	<0.001	0.452
DERS total	84 [71–93]	90 [80–100]	106 [82–122]	H=22.85	<0.001	0.106
DERS nonacceptance	14 [11–16]	15 [12–19]	18 [14–21]	H=15.97	<0.001	0.071
DERS goals	11 [9–14]	12 [10–16]	14 [12–17]	H=17.80	<0.001	0.080
DERS impulse	12 [9–15]	13 [11–16]	15 [12–18]	H=9.04	0.011	0.036
DERS awareness	14 [11–15]	15 [12–16]	17 [14–19]	H=17.36	<0.001	0.078
DERS strategies	20 [17–22]	22 [17–25]	26 [18–29]	H=13.85	<0.001	0.060
DERS clarity	10 [8–13]	13 [11–14]	15 [12–18]	H=34.26	<0.001	0.164
Baseline HAM-A	21 [18–25]	23 [20–27]	27 [22–33]	H=21.50	<0.001	0.099
Baseline GAD-7	11 [9–13]	13 [11–15]	13 [11–16]	H=18.67	<0.001	0.085
PHQ-9	6 [4–8]	6 [4–9]	9 [6–12]	H=14.78	<0.001	0.065
Functional impairment score	10 [8–12]	13 [10–17]	16 [13–19]	H=35.51	<0.001	0.170

Values are median [IQR]. H, Kruskal-Wallis statistic. Effect size is epsilon-squared (ϵ^2). CTQ, Childhood Trauma Questionnaire; DERS, Difficulties in Emotion Regulation Scale; HAM-A, Hamilton Anxiety Rating Scale; GAD-7, Generalized Anxiety Disorder-7; PHQ-9, Patient Health Questionnaire-9.

Associations between adversity, emotion regulation, and symptom severity

Childhood adversity showed a positive correlation with emotion-regulation difficulty (CTQ total versus DERS total: Spearman $\rho=0.360$, $p<0.001$). CTQ total also correlated with baseline HAM-A ($\rho=0.360$, $p<0.001$) and GAD-7 ($\rho=0.308$,

$p < 0.001$). Associations of DERS total with baseline HAM-A ($\rho = 0.608$, $p < 0.001$) and GAD-7 ($\rho = 0.574$, $p < 0.001$) were stronger. The full correlation matrix is shown in Table 3, and the CTQ-DERS relationship is illustrated in Figure 1.

Table 3: Spearman correlation matrix for adversity, emotion regulation, symptoms, and functioning

Variable	CTQ total	DERS total	Baseline HAM-A	Baseline GAD-7	PHQ-9	Functional impairment
CTQ total	1.000	0.360 (< 0.001)	0.360 (< 0.001)	0.308 (< 0.001)	0.233 (< 0.001)	0.426 (< 0.001)
DERS total	0.360 (< 0.001)	1.000	0.608 (< 0.001)	0.574 (< 0.001)	0.325 (< 0.001)	0.600 (< 0.001)
Baseline HAM-A	0.360 (< 0.001)	0.608 (< 0.001)	1.000	0.716 (< 0.001)	0.480 (< 0.001)	0.673 (< 0.001)
Baseline GAD-7	0.308 (< 0.001)	0.574 (< 0.001)	0.716 (< 0.001)	1.000	0.349 (< 0.001)	0.643 (< 0.001)
PHQ-9	0.233 (< 0.001)	0.325 (< 0.001)	0.480 (< 0.001)	0.349 (< 0.001)	1.000	0.452 (< 0.001)
Functional impairment	0.426 (< 0.001)	0.600 (< 0.001)	0.673 (< 0.001)	0.643 (< 0.001)	0.452 (< 0.001)	1.000

Cells show Spearman ρ (p-value). HAM-A, Hamilton Anxiety Rating Scale; GAD-7, Generalized Anxiety Disorder-7.

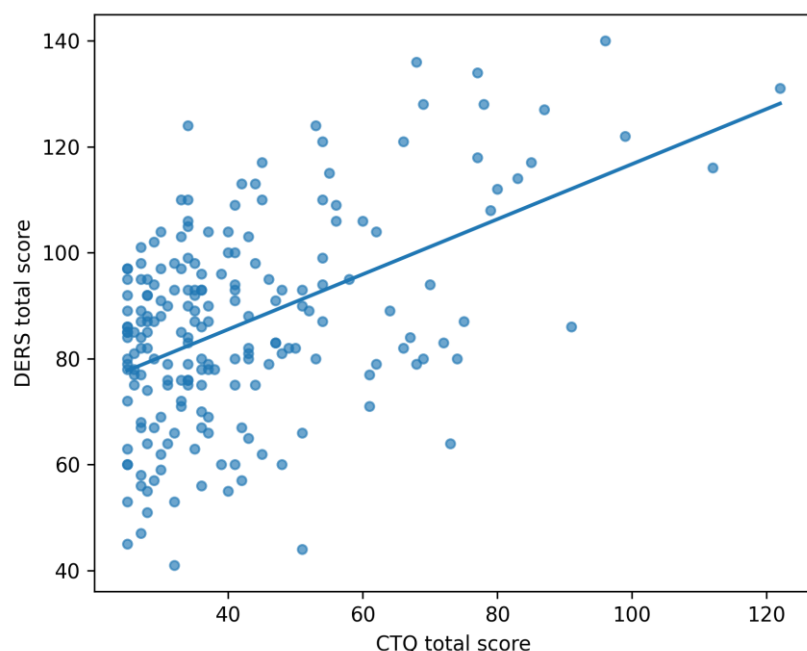


Figure 1: Association between childhood adversity (CTQ total) and emotion-regulation difficulties (DERS total). The fitted line represents the linear trend for visualization.

Prospective symptom course

Follow-up was completed by 185 (92.5%) participants. Anxiety symptoms improved overall during the 8-12-week follow-up, but residual HAM-A and GAD-7 scores were higher with increasing childhood-adversity burden, and improvement in HAM-A was smaller in the high-adversity group (Table 4). Follow-up completion did not differ significantly across adversity groups. The baseline-to-follow-up HAM-A distributions are shown in Figure 2.

Table 4: Prospective symptom course according to childhood-adversity burden

Measure	Low (n=120)	Moderate (n=49)	High (n=31)	Test statistic	p-value
Baseline HAM-A	21 [18–25]	23 [20–27]	27 [22–33]	H=21.50	<0.001
Follow-up HAM-A	11 [7–14]	15 [10–19]	18 [16–26]	H=33.78	<0.001
Change in HAM-A	10 [7–12]	8 [6–11]	7 [5–9]	H=13.32	0.001
Baseline GAD-7	11 [9–13]	13 [11–15]	13 [11–16]	H=18.67	<0.001
Follow-up GAD-7	7 [4–9]	7 [6–10]	10 [8–13]	H=16.50	<0.001
Change in GAD-7	4 [3–5]	5 [4–6]	3 [2–4]	H=11.97	0.003
Medication adherence: Poor	33 (27.5%)	17 (34.7%)	13 (41.9%)	$\chi^2=5.59$	0.232
Medication adherence: Partial	44 (36.7%)	15 (30.6%)	13 (41.9%)		
Medication adherence: Good	43 (35.8%)	17 (34.7%)	5 (16.1%)		
Follow-up completed	114 (95.0%)	44 (89.8%)	27 (87.1%)	$\chi^2=2.90$	0.234

Values are median [IQR] or n (%). Positive change scores indicate symptom improvement. H, Kruskal-Wallis statistic; χ^2 , chi-square statistic.

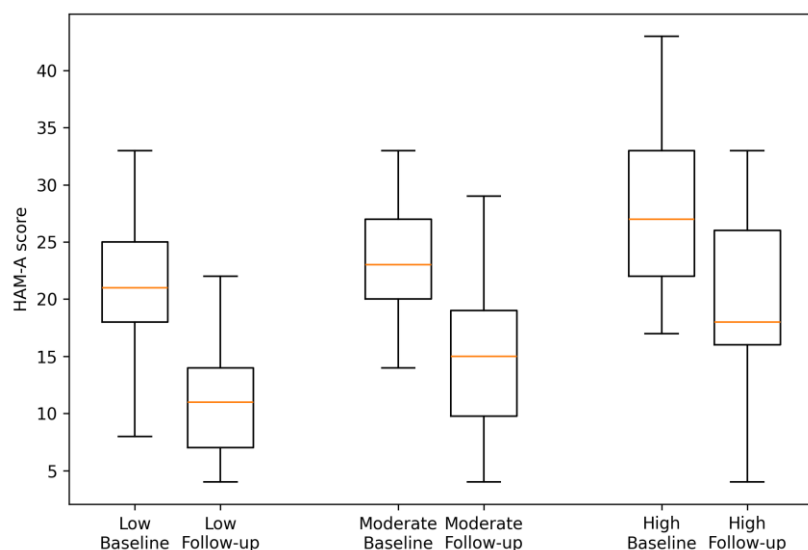


Figure 2: Baseline and follow-up HAM-A scores according to childhood-adversity burden.

Multivariable analysis

In the baseline multivariable model, DERS total remained independently associated with HAM-A after

adjustment for CTQ total and relevant demographic and clinical factors (B=1.540 per 10-point increase, standardized $\beta=0.466$, $p<0.001$). The CTQ coefficient

was attenuated after inclusion of DERS (B=0.428 per 10-point increase, $p=0.058$). PHQ-9 also remained independently associated with HAM-A. The model explained 49.5% of adjusted variance in baseline HAM-A.

Table 5: Multivariable linear regression for baseline HAM-A severity

Predictor	B	Standardized β	95% CI	Test statistic	p-value
CTQ total, per 10-point increase	0.428	0.121	-0.015–0.871	$t=1.89$	0.058
DERS total, per 10-point increase	1.540	0.466	1.134–1.945	$t=7.44$	<0.001
Age, per 10-year increase	-0.424	-0.065	-1.103–0.255	$t=-1.22$	0.221
Female sex	-0.119	-0.009	-1.418–1.181	$t=-0.18$	0.858
GAD duration, per year	0.041	0.027	-0.120–0.203	$t=0.50$	0.616
PHQ-9, per 1-point increase	0.523	0.295	0.333–0.713	$t=5.40$	<0.001
Previous psychiatric treatment	-0.146	-0.012	-1.457–1.166	$t=-0.22$	0.828
Lower socioeconomic status	0.475	0.032	-0.900–1.850	$t=0.68$	0.499

Model adjusted $R^2=0.495$. B, unstandardized regression coefficient; β , standardized coefficient; CI, confidence interval. Maximum variance inflation factor=1.47.

Prospective regression

Among follow-up completers, baseline HAM-A was the strongest predictor of follow-up HAM-A. Higher CTQ and DERS scores remained associated with greater residual anxiety, whereas good and partial medication adherence were associated with lower follow-up severity relative to poor adherence. The prospective model had an adjusted R^2 of 0.799 (Table 6).

Table 6: Multivariable linear regression for follow-up HAM-A among follow-up completers

Predictor	Adjusted B	95% CI	Test statistic	p-value
Baseline HAM-A, per 1-point increase	0.733	0.633–0.832	$t=14.47$	<0.001
CTQ total, per 10-point increase	0.538	0.182–0.893	$t=2.97$	0.003
DERS total, per 10-point increase	0.670	0.355–0.984	$t=4.17$	<0.001
PHQ-9, per 1-point increase	-0.035	-.193–0.124	$t=-0.43$	0.667
Good adherence vs poor	-2.997	-4.198–-1.796	$t=-4.89$	<0.001
Partial adherence vs poor	-1.883	-3.071–-0.695	$t=-3.11$	0.002
Combined SSRI/SNRI + psychological intervention	0.108	-0.998–1.214	$t=0.19$	0.849
GAD duration, per year	0.046	-0.080–0.173	$t=0.72$	0.473

Analysis included 185 follow-up completers. B, adjusted unstandardized regression coefficient; CI, confidence interval. Model adjusted $R^2=0.799$.

Exploratory mediation analysis

Exploratory mediation analysis demonstrated a positive indirect association between childhood adversity and baseline HAM-A through emotion dysregulation

(standardized indirect effect=0.270, bootstrap 95% CI 0.191-0.356). The direct association remained non-zero after inclusion of DERS, consistent with a partial mediation pattern (Table 7).

Table 7: Exploratory mediation analysis of CTQ total, DERS total, and baseline HAM-A

Effect/path	Standardized estimate	95% CI	Interpretation
Total effect (c)	0.450	—	Standardized total association of CTQ with HAM-A
Direct effect (c')	0.180	—	CTQ association with HAM-A after including DERS
a path	0.487	—	CTQ → DERS
b path	0.554	—	DERS → HAM-A adjusted for CTQ
Indirect effect (a×b)	0.270	0.191–0.356	Bootstrap 95% CI
Proportion mediated	59.9%	—	Exploratory

All effects are standardized. The indirect-effect confidence interval was obtained by bootstrap resampling. The mediation analysis was exploratory.

Discussion

Adults with generalized anxiety disorder who experienced more childhood-adversity burden reported more severe emotion-regulation problems, higher anxiety and depressive symptom scores, and lower functioning. The Childhood Trauma Questionnaire (CTQ) scores were correlated with DERS, HAM-A and GAD-7 scores, and DERS scores were correlated with anxiety severity scores, with the latter correlation being stronger. The coefficient of the CTQ was reduced and DERS was independently associated with baseline HAM-A in multivariable analysis. Adversity was associated with higher residual anxiety and less improvement during 8–12 weeks of follow-up; CTQ and DERS were also associated with follow-up HAM-A after controlling for baseline severity and treatment adherence. The exploratory mediation model also indicated that emotion dysregulation explained a significant, but partial, amount of the relationship between adversity and anxiety.

Our cohort's graded association between childhood adversity and adult anxiety is similar to that seen in large epidemiological studies. Kessler et al. examined the relationship between childhood adversity and psychiatric disorders in the WHO World Mental Health Surveys, which included 51,945 adults from 21 countries, and concluded that childhood adversities were highly interrelated, with abuse, neglect, and other measures of dysfunctional family functioning having particularly strong associations with psychiatric disorders. In the countries participating in the study, their population-attributable models suggested that childhood adversities statistically explained about 31% of anxiety disorders and nearly 30% of all psychiatric disorders⁹. More locally relevant is the study of Trivedi et al., which examined 909 Indian adults, with progressively higher odds of anxiety associated with higher ACE scores, and even stronger associations with depression in the medium, high and very high ACE categories¹⁰. These results are consistent with the graded pattern of HAM-A,

GAD-7, depressive symptoms and functional impairment found in our study across categories of adversity.

Emotion dysregulation was a unique factor in the explanation of symptom burden. The correlation of DERS with HAM-A ($\rho=0.608$) and GAD-7 ($\rho=0.574$) was stronger than the corresponding correlations with CTQ, and DERS was a strong independent correlate of baseline anxiety after adjustment. This is consistent with the meta-analysis by Aldao et al. that combined 114 studies with 241 effect sizes. Rumination, a maladaptive strategy, showed a large association with psychopathology, and avoidance and suppression showed medium-to-large associations; emotion-regulation strategies were also more consistently associated with internalizing disorders than with externalizing disorders¹¹. Therefore, the strength of the DERS–anxiety relationship in this current GAD sample is consistent with a larger literature that has found that regulatory dysfunction is a key transdiagnostic feature of internalizing psychopathology.

The relationship between childhood adversity and emotion regulation was less strong than the relationship between emotion regulation and current levels of anxiety severity, which is also biologically and developmentally plausible. In their review of 35 studies with 11,344 children and adolescents, Gruhn and Compas reported that emotion dysregulation was positively related to maltreatment ($r=0.28$) and overall emotion regulation was negatively related to maltreatment ($r=-0.24$). At the strategy level, maltreatment was correlated with increased avoidance, suppression and negative emotional expression, with correlations ranging from 0.24 to 0.25¹². Our CTQ–DERS correlation is slightly higher at 0.360, which may be due to the fact that we used an

adult clinical GAD sample where the regulatory vulnerabilities are already clinically relevant.

The mediation findings also bolster emotion regulation as one pathway between early adversity and adult anxiety, and argue against a deterministic model. Poole et al. studied 4,006 adult primary-care patients and discovered that the link between adverse childhood experiences and adult anxiety symptoms was mediated by emotion dysregulation. Importantly, the indirect pathway was seen to be different depending on resilience and was still present after controlling for sociodemographic factors¹³. In our sample, the indirect pathway via DERS was 0.270, and emotion dysregulation accounted for about 60% of the overall association. The findings were more in line with partial mediation than with emotion regulation being the only mechanism, as the direct effect remained present after inclusion of DERS.

This is also supported by longitudinal evidence. Weissman et al. re-assessed psychopathology two years later in 262 children and adolescents, 162 of whom had been exposed to abuse or domestic violence. Maltreatment was associated with greater emotional reactivity, expressive suppression and rumination, while emotional reactivity and rumination mediated, in part, later increases in a general psychopathology factor¹⁴. Their population was younger, but their outcome was transdiagnostic, and the prospective pattern is similar to our finding that emotion-regulation difficulty was related to subsequent anxiety severity even after we controlled for baseline HAM-A in the model.

However, not all studies agree that emotion dysregulation is the primary intermediate mechanism. Rosencrans et al. assessed 73 adults (68% female) with generalized or social anxiety disorder, and looked at

emotional abuse, emotional neglect, distress tolerance, emotion regulation, and quality of life. The association between emotional neglect and quality of life was mediated by distress tolerance, but not by emotion-regulation difficulty, and neither of these mechanisms significantly mediated the association involving emotional abuse¹⁵. This contrast is significant as it indicates that the psychological processes through which adversity domains may affect adult functioning may differ in part. Our results for emotional neglect and emotional abuse across CTQ categories, in addition to partial mediation by DERS, also suggest that other factors such as distress tolerance, attachment, cognitive schemas, or resilience may play a role.

The findings also have potential treatment implications for the future. Overall, anxiety decreased, but participants with high childhood adversity had higher HAM-A and GAD-7 scores at follow-up and less improvement on the HAM-A. Furthermore, although baseline severity was controlled for, CTQ and DERS continued to be associated with residual anxiety, and good and partial medication adherence were associated with better outcomes. Randomized evidence indicates that treatment response in GAD may, in fact, differ as a function of maltreatment history. Tang et al. randomly assigned 54 patients with GAD to 8 weeks of MBCT or psychoeducation. Mindfulness-based therapy showed greater improvement in worry overall, while anxiety improved similarly between groups; among those with childhood emotional abuse, however, greater reduction in anxiety was seen in the mindfulness-based therapy group than in the psychoeducation group, and mindfulness-based therapy appeared more effective among those with than without emotional-abuse histories¹⁶. These findings support our observations that

childhood adversity can impact not just severity of symptoms, but the short-term course of symptoms and responsiveness to interventions that target emotional processes.

Several strengths of the present study merit consideration. Participants had clinically diagnosed GAD, childhood adversity was assessed across multiple CTQ domains, emotion regulation was examined multidimensionally, and both clinician-rated and self-reported anxiety measures were incorporated. Prospective reassessment also allowed examination of residual symptom severity rather than limiting the analysis to cross-sectional associations. The mediation analysis adds mechanistic context, although it should remain exploratory because CTQ and baseline DERS were assessed concurrently.

The study also has limitations. Childhood adversity was assessed retrospectively and is therefore susceptible to recall and mood-congruent reporting biases. The single-centre sample may limit generalizability, and follow-up was restricted to 8–12 weeks. Treatment was observational rather than randomized, so associations between adherence, treatment exposure, and outcome may be affected by confounding. Depressive symptoms were common and may share variance with both emotion dysregulation and anxiety severity. Finally, although the prospective model adjusted for baseline anxiety, causal conclusions cannot be drawn regarding childhood adversity or DERS.

Conclusion

Childhood adversity was associated with greater emotion-regulation difficulties, higher anxiety severity, and poorer short-term symptom outcomes in adults with GAD. Emotion dysregulation emerged as an important independent correlate and partially accounted for the

relationship between childhood adversity and anxiety severity, supporting its potential relevance in clinical assessment and treatment planning.

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