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# Positive reappraisal coping mediates the relationship between parental abuse and lack of affection on adulthood generalized anxiety severity

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#### ABSTRACT

Exposure to parental abuse and lack of parental affection during childhood are risk factors for adulthood psychopathology. Tendency to engage in positive reappraisal may be a plausible mechanism underlying this relationship. The current study examined if positive reappraisal coping mediated the relationship between maternal/paternal abuse/affection and adulthood generalized anxiety disorder (GAD) symptoms. Participant data (N = 3294) from the Midlife Development in the United States study was collected in three waves, spaced nine years apart. Longitudinal structural equation mediation modeling examined whether positive reappraisal coping at Time 2 mediated the relationship between maternal/paternal abuse/affection at Time 1 and GAD symptoms at Time 3, controlling for GAD symptoms at Time 1. Positive reappraisal coping mediated maternal/paternal childhood abuse – GAD symptom severity and maternal/paternal childhood affection – GAD severity relations. Maternal and paternal abuse was associated with lower positive reappraisal tendencies, predicting increased GAD symptom severity. Conversely, higher maternal/paternal affection revealed that childhood abuse to GAD severity via positive reappraisal path was significant for maternal but not paternal abuse, whereas affection from both parents remained significant. Positive reappraisal coping may be a possible mechanism linking childhood experiences to adulthood GAD severity.

# 1. Introduction

Generalized anxiety disorder (GAD) is a persistent mental disorder characterized by excessive worrying, tension, hypervigilance, and other somatic symptoms that persist for at least six months (American Psychiatric Association, 2013). GAD symptoms have also been shown to be highly comorbid with other mental disorders, such as major depressive disorder, panic disorder, and bipolar disorder (Barber et al., 2023; Silove & Marnane, 2013; Yapici-Eser et al., 2018), and has evidenced widespread consequences across many other domains. Examples include increased social disability (Newman et al., 2013b; Wittchen, 2002), poorer executive functioning (Majeed et al., 2023; Zainal & Newman, 2022), decreased work productivity (Hoffman et al., 2008) and increased primary care utilization (Maier et al., 2000; Porensky et al., 2009). Given that anxiety disorders are among the most common mental health disorders in the general population (Alonso et al., 2007; Kessler et al., 2005; Newman et al., 2013a) and that the detrimental impact of GAD symptoms is widespread, identifying and understanding risk factors and mechanisms associated with GAD symptoms is essential.

Childhood experiences have been shown to be a prominent factor in the development of GAD symptoms. Broadly, childhood experiences have been examined from the perspectives of both adverse (e.g., emotional, physical, or sexual abuse and household dysfunction) and positive (e.g., familial/social-emotional and social support; Bethell et al., 2019; Crandall et al., 2019; Felitti et al., 1998) events. Adverse childhood experiences, specifically in the form of parental childhood abuse, have been associated with poor outcomes ranging from difficulties in controlling/expressing anger toward self and others (Win et al., 2021), lower self-acceptance (Sanghvi et al., 2023), and higher somatic symptoms and medical utilization (Newman et al., 2000). More importantly, parental abuse during childhood has been linked to a wide range of mental health problems, including depression (Adrian &

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Hammen, 1993; Shih et al., 2006), externalizing issues (e.g., Deater-Deckard et al., 1998), and in particular, GAD symptoms (Copeland et al., 2018; Newman et al., 2016; Rudolph & Hammen, 1999; Sanghvi et al., 2023). Even decades after encounters of childhood abuse, retrospectively reported parental childhood abuse was found to increase risk of mental disorders in adulthood (Chapman et al., 2004). Taken together, parental abuse during childhood has been associated with lifelong increased risk of psychopathology, especially GAD symptoms (Green et al., 2010; Kessler et al., 2010).

Conversely, positive childhood experiences in the form of high parental affection have been linked to improved outcomes such as subjective and psychosocial well-being (Chen et al., 2019; Huppert et al., 2010). High levels of parental affection have also been inversely linked with mental health problems (Bartek et al., 2021; Chen et al., 2019; Enns et al., 2002), particularly anxiety symptoms (Butterfield et al., 2019). In contrast to the long-term effects of childhood abuse, Bethell et al. (2019) concluded that positive childhood experiences (e.g., parental affection) could have lifelong protective effects on mental health (including reduced pathological worry and other GAD symptoms).

One mechanism that might underlie the relationship between childhood experiences and GAD symptoms in adulthood is emotion regulation (for a review, see Dvir et al., 2014; Miu et al., 2022). Emotion regulation is defined as the process of shaping when and what emotions one has and the experience or expression of these emotions (Gross, 2014). Difficulty in regulating one's emotions has been identified as a transdiagnostic factor for many emotional disorders (Joormann, 2010; Nolen-Hoeksema et al., 2008), including anxiety disorders (Everaert & Joormann, 2019; Mennin et al., 2003; Newman & Llera, 2011; Teachman et al., 2012). One prominent strategy to regulate emotions is through the propensity to use positive reappraisal. Positive reappraisal is defined as cognitively reframing the meaning of distressing events to be less negative or more positive to reduce their negative emotional impact (Gross, 2014). Indeed, the utilization of positive reappraisal as a strategy to regulate emotions has been shown to be a strong factor in decreasing internalizing symptoms (Aldao et al., 2010; Kivity & Huppert, 2018; Liu & Thompson, 2017) due to decreased negative and increased positive experience of emotions (Gross & John, 2003) and better recovery from acute stressors (Jamieson et al., 2012). Collectively, deficits in tendencies to engage in positive reappraisal could result in the occurrence and maintenance of chronic psychopathology such as GAD and related anxiety disorders.

The development of emotional regulatory skills has been theorized to occur incrementally over the course of childhood (Gross & Muñoz, 1995), with researchers proposing a few theoretical models that might explain the underlying processes behind parental childhood abuse and resulting deficits in emotion regulation. For example, the theory of behavioral modeling of parents to children (Eisenberg et al., 1998; Rieder & Cicchetti, 1989) posits that children who observe emotion dysregulation in parents or caregivers may subsequently have difficulty regulating their feelings. Hence, it is highly likely that experiences of childhood abuse might result in impaired tendencies to engage in positive reappraisal, a component of emotion regulation, which might predispose one to emotional disorders (e.g., GAD symptoms). Conversely, experiences of parental affection would likely support the development of emotion regulation strategies, promoting the usage of positive reappraisal tendencies and reducing GAD symptoms over time. Taken together, understanding how tendency to positively reappraise is influenced by exposure to parental abuse/affection and its relation to GAD symptoms might provide crucial insights into our understanding of the mechanisms contributing to the onset and maintenance of GAD. To this end, such efforts may aid in more precisely identifying treatment targets and informing optimal preventive psychosocial interventions.

Much of the current empirical literature (e.g., Boyes et al., 2016;

Cloitre et al., 2019; Miu et al., 2022) has examined emotion regulation overall as a mediator between childhood abuse and psychopathology. In particular, Boyes et al. (2016) found that cognitive reappraisal tendencies were positively associated with mental health. Similarly, Miu et al. (2022) found that childhood adversity was negatively related to habitual cognitive reappraisal use, which in turn heightened the risk for future psychopathology. However, most of these studies were cross-sectional, which precluded weak causal inferences due to the absence of temporal precedence (Blackwell & Glynn, 2018) and did not explicitly examine GAD symptoms in adulthood. Furthermore, although negative associations between childhood parental affection and adulthood psychopathology have been established (Aunola et al., 2015; Jorm et al., 2003), there are a dearth of studies in the literature examining the mediating role of positive reappraisal, specifically on the relationship between childhood parental affection and GAD symptoms in adulthood.

In addition, a deeper examination of the differential impact of maternal and paternal figures on psychopathological symptoms in adulthood is warranted. Much of the existing research examining parental roles during childhood has disproportionately focused on maternal figures, often ignoring paternal figures (Brumariu & Kerns, 2010; Ding & He, 2022; Rutter, 1981). Researchers have suggested that both parental figures confer unique and independent effects on developmental outcomes (Grossmann et al., 2002; Pleck, 2010), which could be explained by varying roles within the family and different caregiving styles (Cox & Paley, 1997; Cui et al., 2018). Although there is growing research emphasis on the differential effects of parental roles on psychopathology, findings in the existing literature remain mixed. Most research (Kong & Martire, 2019; Moretti & Craig, 2013) has suggested that the maternal role might be a stronger predictor than the paternal role in mental health outcomes. The lasting influence and intricate dynamics between children and their mothers, as opposed to fathers, endured well into adulthood (Rosenthal & Kobak, 2010). Similarly, recent studies have observed that childhood abuse by mothers rather than fathers was associated with reduced psychological well-being, heightened risk of psychopathology, and increased distress (Kong & Martire, 2019; Kong, Martire, Liu et al., 2019). However, some researchers (Mattanah, 2001; Summers et al., 1998) have suggested that the paternal influence was a stronger predictor. Together, the dearth of research examining both parental roles and mixed findings in the current literature present a strong impetus to examine both parental roles in the perpetration of abuse and engagement in affection.

Therefore, based on theory and empirical literature, the current study sought to examine the following hypotheses. First, we expected positive reappraisal tendencies to significantly mediate the relationship between parental childhood abuse and GAD symptoms in adulthood. Specifically, we predicted that increased maternal and paternal abuse (examined separately) would result in lower positive reappraisal coping, which in turn would lead to greater GAD symptom severity in adults. Next, we hypothesized that positive reappraisal tendencies would significantly mediate the relationship between maternal/paternal affection during childhood and GAD symptoms. Specifically, we predicted that higher maternal and paternal affection would separately result in increased positive reappraisal coping, which would, in turn, lead to lower experiences of GAD symptom severity in adulthood.

# 2. Method

## 2.1. Participants

Data for this study was taken from the Midlife in the United States project (MIDUS; Brim et al., 1999; Ryff et al., 2015; Ryff et al., 2007). MIDUS comprised three waves of data collected over approximately nine-year intervals: 1995–1996 (Time 1 [T1]); 2004–2005 (Time 2 [T2]); 2012–2013 (Time 3 [T3]). A total of 3294 participants were included in this study. Participants were between 20 and 74 years of age (M = 45.6, SD = 11.4) at baseline, of which 54.6% were female and 46.8% were college-educated. Most participants racially identified as White (89.01%), compared to 10.99% of participants who identified as African American, Native American, Asian, multiracial, and others. Refer to Table 1 for descriptive statistics and a correlation matrix of the study variables.

# 2.2. Procedures

The first wave of data collection (T1) was done via telephone interviews and self-administered questionnaires (SAQs).<sup>1</sup> The second (T2) and third (T3) data collection waves were done via SAQs. Modified versions of the assessments were administered via telephone for participants who did not complete SAQs at T2 and T3 (refer to MIDUS codebooks for more information; Brim et al., 1999; Ryff et al., 2007, 2015). The current study utilized data from 3,294 participants who completed telephone interviews and/or SAQs assessing GAD symptom severity at T1 and T3 because it offered data from participants who partook in most of the protocol aspects relevant to the current research aim. Measures that evaluated the frequency of childhood parental abuse and affection were completed at T1, and the measure of positive reappraisal was completed at T2.

#### 2.3. Measures

#### 2.3.1. Parental abuse during childhood

Retrospectively-reported experiences of childhood abuse were collected with the Conflict Tactics Scale (CTS2; Straus et al., 1996). The CTS2 examined emotional, physical, and serious physical forms of abuse experienced during childhood. Participants were asked to report the frequency at which each of their parents or people who raised them "Insulted or swore" at them (emotional abuse), "Pushed, grabbed, or shoved" them (physical abuse), and "Kicked, bit, or hit with a fist" (serious physical abuse). For this study, the abuse perpetrated by participants' mother or woman who raised them and father or man who raised them were examined separately. Participants rated their experiences on a 4-point scale (1 = Never to 4 = Often). Domains examined in the CTS2 demonstrated satisfactory internal consistency (Cronbach's  $\alpha s = .73$ , .71, and .75 for emotional, physical, and serious physical abuse, respectively). The CTS2 also had strong construct validity and good retest reliability across diverse samples (Chapman & Gillespie, 2019).

#### 2.3.2. Parental affection during childhood

Retrospectively reported maternal and paternal affection during childhood was collected at T1 (Rossi, 2001). Respondents rated their responses along a 4-point Likert scale ( $1 = Not \ at \ all$  to  $4 = A \ lot$ ). Examples of the items included "How much did she understand your problems and worries?" "How much love and affection did she give you?" and "How much time and attention did she give you when you needed it?". Both maternal and paternal affection scales were found to have good internal consistency ( $\alpha$ s = .91 and .93, respectively). This scale had also demonstrated good construct validity (Chen et al., 2019).

#### 2.3.3. Positive reappraisal coping

Positive reappraisal was measured at T2 as a part of an SAQ assessing primary and secondary control (Wrosch et al., 2000). Participants responded to five items, which included statements such as "I can find something positive, even in the worst situations," "I find I usually learn something meaningful from a difficult situation," and "Even when everything seems to be going wrong, I can usually find a bright side to the situation." Participants rated their positive reappraisal tendencies on a 4-point Likert scale (1 = *Not at all* to 4 = *A lot*). Positive reappraisal at T2 displayed good internal consistency ( $\alpha$  = .78). The primary and secondary control strategies scale (which includes positive reappraisal) displayed strong construct validity (Haynes et al., 2009; Wrosch et al., 2000).

# 2.3.4. Generalized anxiety disorder symptom severity

GAD symptom severity was measured at T1 and T3 using the Composite International Diagnostic Interview-Short Form (CIDI-SF; Kessler et al., 1998; Wittchen, 1994), which was derived from the GAD diagnostic criteria in the Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition (DSM-III-R; American Psychiatric Association, 1987). Participants administered the CIDI-SF over the telephone and were asked to report the frequency of 10 GAD symptoms over the past 12 months. Examples of items include "were restless because of your worry," "were keyed up, on edge, or had a lot of nervous energy," and "had trouble staving asleep because of your worry." Participants responded along a 4-point Likert scale (1 = never to 4 = on mostdays). The CIDI-SF demonstrated high sensitivity (96.6%) and specificity (99.8%; Kessler et al., 1998) and also had good internal consistency for this study (T1:  $\alpha = .96$ ; T3:  $\alpha = .97$ ). Psychometric property analyses were carried out to validate the utilization of the CIDI as a continuous measure of symptom severity. These analyses revealed evidence supporting convergent validity of the CIDI-SF GAD severity score, given significantly large and positive correlations of r = .81 with the Spielberger Trait Anxiety Inventory (Spielberger, 1983) and r = .78 with the Perceived Stress Scale (Cohen et al., 1983). The CIDI-SF GAD severity score also showed strong discriminant validity, based on consistently small and positive correlations of r values of .08 with the Social Contribution Scale and .06 with the Social Integration Scale (Keyes & Shapiro, 2004).

#### 2.4. Analytic plan

Longitudinal structural equation mediation modeling was conducted using the lavaan R package (Rosseel, 2012) in the RStudio software (R Version 4.2.2). To assess model fit, the following fit statistics were utilized: Chi-square (Hu & Bentler, 1999), model degrees of freedom and its probability of null outcomes (p) value, confirmatory fit index (CFI; Bentler, 1990), Tucker-Lewis index (Tucker & Lewis, 1973), root mean square error of approximation (RMSEA; Steiger, 1990) and its 90% confidence interval (CI), and standardized root mean square residual (SRMR; Byrne, 1998; Hu & Bentler, 1999). Two separate mediation models were constructed to examine T1 childhood maternal and paternal abuse predicting T3 GAD symptom severity via T2 positive reappraisal. Similarly, another two models measured T1 childhood maternal and paternal affection predicting T3 GAD symptoms via T2 positive reappraisal. Using the product of coefficients method of indirect effect ( $a \times b$ ), mediation analyses were conducted for the coefficients of latent composite scores derived for T1 parental abuse (maternal and paternal abuse separately), predicting the latent composite scores of T2 positive reappraisal (path *a*) and T2 positive reappraisal predicting T3 GAD symptom severity (path *b*). Additional mediation analyses were conducted with the same approach, examining T1 parental affection (both maternal and paternal affection separately) predicting T3 GAD symptom severity via T2 positive reappraisal. Also, we reported the unstandardized regression coefficients (β), standard errors (SE), z-scores, and p values (Cheung & Lau, 2008). Mediation effect sizes were presented as a proportion of indirect effect ( $a \times b$ ) relative to total effect  $(c = a \times b + c')$  (Preacher & Kelley, 2011; Wen & Fan, 2015). To increase analytic rigor, T1 GAD status was controlled for in all mediation analyses. Methodological researchers in causal inference advocate against adjusting for a mediating variable at baseline. Doing so could introduce

<sup>&</sup>lt;sup>1</sup> Although the MIDUS study Time 1 (T1) data collection had 7108 participants and Time 2 (T2) had 4512, only the 3294 participants had data for diagnostic assessments at both T1 and Time 3 (T3) (i.e., the participants selected for the present study).

Correlation matrix and descriptive statistics of study variables.

		1	2	3	4	5	6	7
1	MatAb (T1)	_						
2	MatAf (T1)	- 483 ***	-					
3	PatAb (T1)	.486 ***	242 ***	-				
4	PatAf (T1)	213 ***	.456 ***	463 ***	-			
5	PR (T2)	042 *	.084 ***	024	.105 ***	-		
6	GAD (T1)	.135 ***	145 ***	.132 ***	134 ***	089 ***	-	
7	GAD (T3)	.119 ***	094 ***	.087 ***	077 ***	128 ***	.346 ***	-
	Μ	4.65	22.84	4.96	19.77	12.28	13.58	13.09
	SD	1.95	4.90	2.14	5.76	2.43	6.46	6.33
	Min	3	7	3	7	4	10	10
	Max	12	29	12	29	16	40	40
	Skew	1.47	-0.87	1.28	-0.30	-0.29	1.83	2.15
	Kurtosis	2.05	0.16	1.23	-0.82	-0.44	2.56	3.87

Note. \*p < .05, \*\*\*p < .001.

T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); MatAb = childhood maternal abuse; MatAf = childhood maternal affection; PatAb = childhood paternal abuse; PatAf = childhood paternal affection; GAD = generalized anxiety disorder; PR = positive reappraisal.

bias by blocking part of the causal effect via the mediator (D'Onofrio et al., 2020; Rosenbaum, 1984). Hence, the authors chose not to control for T1 positive reappraisal. To deal with missing data (3.5% of the total observed dataset), the gold standard approach utilizing full information maximum likelihood (Lee & Shi, 2021) was conducted for data likely to be missing at random.

# 3. Results

# 3.1. T1 Childhood abuse predicting T3 GAD symptom severity via T2 positive reappraisal

The model examining T1 childhood maternal abuse predicting T3 GAD symptom severity via T2 positive reappraisal demonstrated good fit  $(\chi^2(df = 319) = 802.641, p < .001, CFI = .993, RMSEA = .023, 95\% CI$ [0.021, 0.025], SRMR = .030). All individual items had significantly high factor loadings for T1 maternal abuse ( $\lambda = 0.597 - 0.833$ ), T2 positive reappraisal ( $\lambda = 0.499 - 0.882$ ), and T3 GAD symptoms ( $\lambda =$ 0.804 - 0.879) (all p values < .001), offering evidence for the unidimensionality of all constructs of interest.<sup>2</sup> Greater childhood maternal abuse significantly predicted lower T2 positive reappraisal ( $\beta = -0.054$ , SE = 0.013, z = -4.233, p < .001, d = -0.456), which in turn significantly predicted higher T3 GAD symptom severity ( $\beta = -0.235$ , SE = 0.037, z = -6.330, p < .001, d = -0.682). Indirect effects of childhood maternal abuse  $\rightarrow$  T2 positive reappraisal  $\rightarrow$  adulthood GAD symptom severity were significant ( $\beta = 0.013$ , *SE* = 0.004, *z* = 3.397, *p* = .001, *d* = 0.366) with T2 positive reappraisal accounting for 19.70% of the relationship between maternal childhood abuse and adulthood GAD symptom severity. Refer to Tables 2 and 3 for a summary of longitudinal SEM mediation models. Refer to Fig. 1 for a path diagram of this analysis.

Similarly, the model examining T1 paternal abuse predicting T3 GAD symptom severity via T2 positive reappraisal demonstrated good fit ( $\chi^2(df = 319) = 832.807, p < .001$ , CFI = .992, RMSEA = .024, 95% CI [0.022, 0.026], SRMR = .031). All individual items loaded strongly onto their respective unidimensional constructs (T1 paternal abuse:  $\lambda = 0.614 - 0.890$ ; T2 positive reappraisal:  $\lambda = 0.500 - 0.880$ ; T3 GAD symptoms:  $\lambda = 0.805 - 0.879$ ) (all *p* values < .001). Increased childhood paternal abuse significantly predicted lower positive reappraisal at T2 ( $\beta = -0.036, SE = 0.010, z = -3.447, p = .001, d = -0.371$ ), which in turn significantly predicted higher T3 GAD symptom severity ( $\beta = -0.246, SE = 0.038, z = -6.551, p < .001, d = -0.705$ ). Indirect effects of paternal childhood abuse  $\rightarrow$  T2 positive reappraisal  $\rightarrow$ 

adulthood GAD symptom severity were significant ( $\beta = 0.009$ , SE = 0.003, z = 2.957, p = .003, d = 0.318) with T2 positive reappraisal accounting for 28.57% of the relationship between paternal childhood abuse and adulthood GAD symptom severity. Refer to Fig. 2 for a path diagram of this analysis. Taken together, both of these findings support Hypothesis 1.

As a sensitivity analysis, incremental predictions were tested to determine if positive reappraisal would mediate the paths between both maternal and paternal childhood abuse predicting adulthood GAD severity if measures reflecting abuse from both paternal and maternal figures were entered into the same model. This model had good fit ( $\chi^2(df = 396) = 1039.311, p < .001$ , CFI =.987, RMSEA =.024, 95% CI [0.022, 0.026], SRMR =.032). T2 positive reappraisal significantly mediated the childhood maternal abuse–T3 adulthood GAD severity association ( $\beta = -0.012, SE = 0.004, z = 2.780, p < .01, d = 0.281$ ), but not the childhood paternal abuse–T3 adulthood GAD severity association ( $\beta = -0.001, SE = 0.003, z = -0.255, p = .799$ ). The mediation pathway for maternal abuse. Refer to Table 4 for a summary of the longitudinal SEM mediation model.

# 3.2. T1 Parental affection predicting T3 GAD symptom severity via positive reappraisal

The model examining T1 maternal affection predicting T3 GAD symptom severity via T2 positive reappraisal showed good fit ( $\chi^2(df =$ 429) = 3711.636, *p* < .001, CFI = .928, RMSEA = .067, 95% CI [0.065, 0.069], SRMR = .031). All individual items loaded strongly onto their respective unidimensional constructs (T1 paternal abuse:  $\lambda = 0.614$  -0.890; T2 positive reappraisal:  $\lambda = 0.500 - 0.880$ ; T3 GAD symptoms:  $\lambda = 0.805 - 0.879$ ) (all *p* values < .001). Greater childhood maternal affection significantly predicted greater positive reappraisal at T2  $(\beta = 0.035, SE = 0.009, z = 4.056, p < .001, d = 0.379)$ , which in turn significantly predicted lower T3 GAD symptom severity ( $\beta = -0.204$ , SE z = 0.036, z = -5.636, p < .001, d = -0.526). Indirect effects of childhood maternal affection  $\rightarrow$  T2 positive reappraisal  $\rightarrow$  adulthood GAD symptom severity were significant ( $\beta = -0.007$ , *SE* = 0.002, z = -3.350, p = .001, d = -0.313) with T2 positive reappraisal accounting for 20% of the relationship between maternal childhood affection and adulthood GAD symptom severity. Refer to Tables 5 and 6 for a summary of these longitudinal SEM mediation models. Refer to Fig. 3 for a path diagram of this analysis.

Similarly, the model examining T1 paternal affection predicting T3 GAD symptom severity via T2 positive reappraisal showed good fit ( $\chi^2$  (df = 429) = 3590.401, p < .001, CFI = .933, RMSEA = .065, 95% CI [0.063, 0.067], SRMR = .028). All individual items loaded strongly onto their respective unidimensional constructs (T1 paternal abuse:

<sup>&</sup>lt;sup>2</sup> Due to poor factor loading, the fifth item of the positive reappraisal was dropped in all four models of analyses ( $\lambda s = 0.280, 0.279, 0.275, \text{ and } 0.231, \text{ in the original four models}$ ).

T1 Childhood Maternal Abuse Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD.

	Estimate	95% CI	Cohen's $d$
Regressions			
MatAb $[T1] \rightarrow GAD[T3]$	0.053 **	[0.016, 0.090]	0.299
MatAb $(T11 \rightarrow PR (T21)$	-0.054 ***	[-0.080, -0.029]	-0.456
$PR[T2] \rightarrow GAD[T3]$	-0.235 ***	[-0.307, -0.162]	-0.682
$GAD[T1] \rightarrow GAD[T3]$	0.317 ***	[0 268, 0 366]	1.356
Covariances	01017	[01200, 01000]	11000
$GAD[T1] \sim ~ MatAb[T1]$	0.089 ***	[0.064, 0.114]	0.757
Factor Loadings			
T1 MatAb 1	1.000	-	-
T1 MatAb 2	0.799 ***	[0.679, 0.920]	1.404
T1 MatAb 3	0.452 ***	[0.371, 0.534]	1.170
T3 GAD 1	1.000	-	-
T3 GAD 2	0.925 ***	[0.873, 0.977]	3.735
T3 GAD 3	1.003 ***	[0.945, 1.061]	3.667
T3 GAD 4	1.097 ***	[1.036, 1.158]	3.813
T3 GAD 5	1.013 ***	[0.950, 1.075]	3.413
T3 GAD 6	0.986 ***	[0.926, 1.045]	3.485
T3 GAD 7	0.850 ***	[0.796, 0.903]	3.356
T3 GAD 8	1.131 ***	[1.067, 1.196]	3.697
T3 GAD 9	1.020 ***	[0.957, 1.083]	3.407
T3 GAD 10	0.889 ***	[0.819, 0.958]	2.704
T2 PR 1	1.000	-	-
T2 PR 2	1.092 ***	[0.983, 1.201]	2.112
T2 PR 3	1.986 ***	[1.803, 2.168]	2.291
T2 PR 4	2.045 ***	[1.855, 2.234]	2.275
Residual Variances			
T1 MatAb 1	0.252 ***	[0.158, 0.345]	0.568
T1 MatAb 2	0.296 ***	[0.239, 0.353]	1.103
T1 MatAb 3	0.211 ***	[0.181, 0.241]	1.476
T3 GAD 1	0.118 ***	[0.099, 0.137]	1.301
T3 GAD 2	0.152 ***	[0.128, 0.176]	1.342
T3 GAD 3	0.125 ***	[0.105, 0.145]	1.331
T3 GAD 4	0.176 ***	[0.147, 0.204]	1.311
T3 GAD 5	0.165 ***	[0.140, 0.191]	1.372
T3 GAD 6	0.117 ***	[0.098, 0.137]	1.266
T3 GAD 7	0.130 ***	[0.111, 0.149]	1.438
T3 GAD 8	0.148 ***	[0.122, 0.173]	1.215
T3 GAD 9	0.133 ***	[0.111, 0.155]	1.271
T3 GAD 10	0.169 ***	[0.146, 0.192]	1.559
T2 PR 1	0.383 ***	[0.360, 0.406]	3.468
T2 PR 2	0.413 ***	[0.385, 0.440]	3.155
T2 PR 3	0.143 ***	[0.114, 0.173]	1.027
T2 PR 4	0.175 ***	[0.141, 0.209]	1.085
Residual Variances			
Variance of (MatAb)[T1]	0.570 ***	[0.471, 0.669]	1.214
Variance of (GAD)[T3]	0.333 ***	[0.295, 0.370]	1.879
Variance of (PR)[T2]	0.125 ***	[0.105, 0.146]	1.272
Variance of (GAD)[T1]	0.465 ***	[0.420, 0.510]	2.184
Defined Parameters			
Indirect Effect	0.013 ***	[0.005, 0.020]	0.366
Total Effect	0.066 ***	[0.028, 0.103]	0.371

*Note.* \*\* *p* < .01; \*\*\* *p* < .001.

T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); MatAb = childhood maternal abuse; GAD = generalized anxiety disorder; PR = positive reappraisal; CI = confidence interval; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual. Model fit indices:  $\chi^2(df = 319) = 802.641$ , p < .001, CFI = .993, RMSEA = .023, 95% CI [0.021, 0.025], SRMR = .030.

 $\lambda = 0.706 - 0.847$ ; T2 positive reappraisal:  $\lambda = 0.487 - 0.888$ ; T3 GAD symptoms:  $\lambda = 0.782 - 0.891$ ) (all *p* values <.001). Greater childhood paternal affection significantly predicted greater positive reappraisal at T2 ( $\beta = 0.043$ , *SE* = 0.007, *z* = 6.018, *p* < .001, *d* = 0.562), which in turn significantly predicted lower GAD symptom severity ( $\beta = -0.205$ , *SE* = 0.036, *z* = -5.623, *p* < .001, *d* = -0.525). Indirect effects of childhood paternal affection  $\rightarrow$  T2 positive reappraisal  $\rightarrow$  adulthood GAD symptom severity were significant ( $\beta = -0.009$ , *SE* = 0.002, *z* = -4.198, *p* < .001, *d* = -0.392) with T2 positive reappraisal accounting for 40.91% of the relationship between childhood paternal

Table 3

T1 Childhood Paternal Abuse Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD\.

	Estimate	95% CI	Cohen's d
Regressions			
PatAb[T1] $\rightarrow$ GAD[T3]	0.022	[-0.010, 0.053]	0.146
$PatAb[T1] \rightarrow PR[T2]$	-0.036 ***	[-0.057, -0.016]	-0.371
$PR[T2] \rightarrow GAD[T3]$	-0.246 ***	[-0.320, -0.172]	-0.705
$GAD[T1] \rightarrow GAD[T3]$	0.323 ***	[0.273, 0.373]	1.372
Covariances		·····	
GAD[ <i>T1</i> ] ~~ PatAb[ <i>T1</i> ]	0.096 ***	[0.069, 0.122]	0.758
Factor Loadings			
T1 PatAb 1	1.000	-	-
T1 PatAb 2	0.750 ***	[0.648, 0.852]	1.555
T1 PatAb 3	0.483 ***	[0.403, 0.563]	1.271
T3 GAD 1	1.000	-	-
T3 GAD 2	0.926 ***	[0.874, 0.979]	3.728
T3 GAD 3	1.004 ***	[0.946, 1.062]	3.644
T3 GAD 4	1.100 ***	[1.039, 1.161]	3.811
T3 GAD 5	1.013 ***	[0.950, 1.076]	3.396
T3 GAD 6	0.987 ***	[0.927, 1.047]	3.481
T3 GAD 7	0.849 ***	[0.795, 0.903]	3.340
T3 GAD 8	1.133 ***	[1.068, 1.197]	3.685
T3 GAD 9	1.020 ***	[0.956, 1.084]	3.379
T3 GAD 10	0.890 ***	[0.821, 0.959]	2.711
T2 PR 1	1.000	-	-
T2 PR 2	1.088 ***	[0.980, 1.197]	2.121
T2 PR 3	1.978 ***	[1.799, 2.158]	2.329
T2 PR 4	2.048 ***	[1.860, 2.235]	2.303
Residual Variances			
T1 PatAb 1	0.193 ***	[0.084, 0.301]	0.376
T1 PatAb 2	0.322 ***	[0.267, 0.378]	1.224
T1 PatAb 3	0.283 ***	[0.248, 0.318]	1.689
T3 GAD 1	0.119 ***	[0.099, 0.138]	1.304
T3 GAD 2	0.152 ***	[0.128, 0.176]	1.340
T3 GAD 3	0.125 ***	[0.105, 0.145]	1.324
T3 GAD 4	0.174 ***	[0.146, 0.202]	1.305
T3 GAD 5	0.166 ***	[0.140, 0.191]	1.380
T3 GAD 6	0.117 ***	[0.097, 0.137]	1.260
T3 GAD 7	0.131 ***	[0.112, 0.150]	1.449
T3 GAD 8	0.147 ***	[0.121, 0.173]	1.208
T3 GAD 9	0.134 ***	[0.111, 0.156]	1.268
T3 GAD 10	0.169 ***	[0.146, 0.192]	1.564
T2 PR 1	0.383 ***	[0.359, 0.406]	3.481
T2 PR 2	0.413 ***	[0.386, 0.441]	3.165
T2 PR 3	0.146 ***	[0.117, 0.175]	1.066
T2 PR 4	0.172 ***	[0.138, 0.206]	1.073
Residual Variances			
Variance of (PatAb)[T1]	0.735 ***	[0.623, 0.847]	1.380
Variance of (GAD)[T3]	0.333 ***	[0.295, 0.371]	1.868
Variance of (PR)[T2]	0.126 ***	[0.106, 0.147]	1.285
Variance of (GAD)[T1]	0.465 ***	[0.420, 0.510]	2.180
Defined Parameters			
Indirect Effect	0.009 **	[0.003, 0.015]	0.318
Total Effect	0.031	[-0.001, 0.062]	0.206

*Note.* \*\* *p* < .01; \*\*\* *p* < .001.

T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); PatAb = childhood paternal abuse; GAD = generalized anxiety disorder; PR = positive reappraisal; CI = confidence interval; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual. Model fit indices:  $\chi^2(df = 319) = 832.807, p < .001$ , CFI = .992, RMSEA = .024, 95% CI [0.022, 0.026], SRMR = .031.

affection and adulthood GAD symptom severity. Refer to Fig. 4 for a path diagram of this analysis. Taken together, both of these findings support Hypothesis 2.

As a sensitivity analysis, incremental predictions were tested to determine if positive reappraisal would mediate the paths between both maternal and paternal childhood affection predicting adulthood GAD severity if measures reflecting affection from both paternal and maternal figures were entered into the same model. This model had good fit ( $\chi^2(df = 656) = 1980.437$ , p < .001, CFI = .988, RMSEA = .026, 95% CI [0.024, 0.027], SRMR = .030). T2 positive reappraisal significantly



Indirect effect  $\beta = 0.013^{***}$  (0.004), Total effect  $\beta = 0.066^{***}$  (0.019) Fit Statistics:  $\chi 2(df = 319) = 802.641$ , p < .001, CFI = 0.993, RMSEA = 0.023, 95% CI [0.021, 0.025], SRMR = 0.030

Fig. 1. Longitudinal SEM Mediation of T1 Childhood Maternal Abuse Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD. Note. \*\*p < .01; \*\*\*p < .001. T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); MatAb = childhood maternal abuse; GAD = generalized anxiety disorder; PR = positive reappraisal; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual.  $\beta$  = unstandardized beta regression weight with standard error in parenthesis;  $\varepsilon$  = item residual variances;  $\zeta$  = factor residual variances.

mediated the both childhood maternal affection–T3 adulthood GAD severity association ( $\beta = -0.006$ , *SE* = 0.003, *z* = -2.113, *p* < .05, *d* = -0.165), and the childhood paternal affection–T3 adulthood GAD

severity association ( $\beta = -0.009$ , *SE* = 0.003, *z* = -3.531, *p* < .001, *d* = -0.276). The mediation pathways for both maternal and paternal affection were still significant after adjusting for affection from either



Indirect effect  $\beta$  = 0.009\*\*\* (0.003), Total effect  $\beta$  = 0.031 (0.016)

Fit Statistics: χ2(df = 319) = 832.807, p < .001, CFI = .992, RMSEA .024, 95% CI [.022, .026], SRMR = .031

Fig. 2. Longitudinal SEM Mediation of T1 Childhood Paternal Abuse Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD. Note. \*\*p < .01; \*\*\*p < .001. T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); PatAb = childhood paternal abuse; GAD = generalized anxiety disorder; PR = positive reappraisal; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual.  $\beta$  = unstandardized beta regression weight with standard error in parenthesis;  $\varepsilon$  = item residual variances;  $\zeta$  = factor residual variances.

Supplemental incremental prediction analysis of T1 Childhood Maternal Abuse and T1 Childhood Paternal Abuse Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD.

	Estimate	95% CI	Cohen's d
Regressions			
$MatAb[T1] \rightarrow GAD[T3]$	0.056 *	[0.006, 0.105]	0.221
$PatAb[T1] \rightarrow GAD[T3]$	0.001	[-0.041, 0.043]	0.004
$MatAb[T1] \rightarrow PR[T2]$	-0.050 **	[-0.081, -0.020]	-0.323
$PatAb[T1] \rightarrow PR[T2]$	0.003	[-0.022, 0.028]	0.026
$PR[T2] \rightarrow GAD[T3]$	-0.239 ***	[-0.312, -0.166]	-0.643
$GAD[T1] \rightarrow GAD[T3]$	0.317 ***	[0.268, 0.367]	1.268
Covariances			
$GAD[T1] \sim \sim MatAb[T1]$	0.084 ***	[0.060, 0.108]	0.691
GAD[T1] ~~ PatAb[T1]	0.081 ***	[0.056, 0.107]	0.623
Factor Loadings			
T1 MatAb 1	1.000 ***	[1.000, 1.000]	-
T1 MatAb 2	0.861 ***	[0.790, 0.931]	2.394
T1 MatAb 3	0.489 ***	[0.429, 0.550]	1.587
T1 PatAb 1	1.000 ***	[1.000, 1.000]	-
TI PatAb 2	0.850 ***	[0.787, 0.912]	2.690
TI PatAb 3	0.547 ***	[0.488, 0.607]	1.812
I 3 GAD I T2 CAD 2	1.000 ***	[1.000, 1.000]	-
13 GAD 2 T2 CAD 2	1.005 ***	[0.874, 0.979]	3.478
13 GAD 3 T2 CAD 4	1.005 ***	[0.947, 1.062]	3.420
T2 CAD E	1.098 ***	[1.038, 1.139]	3.300
T3 GAD 5	0.084 ***	[0.946, 1.075]	3.132
T3 GAD 7	0.964	[0.924, 1.044]	3.102
T3 GAD 7	1 130 ***	[1.066, 1.195]	3 442
T3 GAD 9	1.020 ***	[0.957, 1.084]	3.160
T3 GAD 10	0.891 ***	[0.821, 0.960]	2.521
T2 PR 1	1.000 ***	[1.000, 1.000]	-
T2 PR 2	1.095 ***	[0.985, 1.205]	1.965
T2 PR 3	1.989 ***	[1.805, 2.173]	2.133
T2 PR 4	2.049 ***	[1.858, 2.239]	2.118
Residual Variances		<b>L</b> ,	
T1 MatAb 1	1.771 ***	[1.740, 1.802]	11.271
T1 MatAb 2	1.663 ***	[1.635, 1.691]	11.806
T1 MatAb 3	1.213 ***	[1.193, 1.232]	12.217
T1 PatAb 1	1.943 ***	[1.910, 1.976]	11.635
T1 PatAb 2	1.719 ***	[1.689, 1.748]	11.559
T1 PatAb 3	1.295 ***	[1.272, 1.318]	11.087
T3 GAD 1	1.321 ***	[1.297, 1.346]	10.670
T3 GAD 2	1.314 ***	[1.290, 1.338]	10.848
T3 GAD 3	1.328 ***	[1.304, 1.353]	10.625
T3 GAD 4	1.354 ***	[1.326, 1.381]	9.704
T3 GAD 5	1.317 ***	[1.291, 1.343]	10.083
13 GAD 6	1.306 ***	[1.282, 1.330]	10.669
T3 GAD 7	1.244 ***	[1.222, 1.200]	0.676
T3 GAD 0	1.332	[1.323, 1.300]	10 174
T3 GAD 9	1.250	[1.273, 1.325]	10.174
T2 PR 1	3 350 ***	[3 325 3 374]	27.059
T2 PR 2	3.152 ***	[3.127, 3.178]	24.211
T2 PR 3	2.923 ***	[2.896, 2.951]	21.007
T2 PR 4	2.857 ***	[2.828, 2.886]	19.610
Residual Variances		2	
Variance of (MatAb)[T1]	0.534 ***	[0.474, 0.594]	1.747
Variance of (PatAb)[T1]	0.661 ***	[0.598, 0.725]	2.056
Variance of (GAD)[T3]	0.332 ***	[0.295, 0.370]	1.748
Variance of (PR)[T2]	0.125 ***	[0.105, 0.146]	1.185
Variance of (GAD)[T1]	0.464 ***	[0.419, 0.509]	2.038
Defined Parameters			
Indirect Effect of MatAb	0.012 **	[0.004, 0.020]	0.281
Indirect Effect of PatAb	-0.001	[-0.007, 0.005]	-0.026
Total Effect	0.068 ***	[0.030, 0.106]	0.349

Note. \* p < .05; \*\* p < .01; \*\*\* p < .001.

T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); MatAb = childhood maternal abuse; PatAb = childhood paternal abuse; GAD = generalized anxiety disorder; PR = positive reappraisal; CI = confidence interval; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual. Model fit indices: ( $\chi^2(df = 396) = 1039.311$ , p < .001, CFI = .987, RMSEA = .024, 95% CI [0.022, 0.026], SRMR = .032).

#### Table 5

T1 Childhood Maternal Affection Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD.

	Estimate	95% CI	Cohen's d
Regressions			
$MatAf/T11 \rightarrow GAD/T31$	-0.028	[-0.056, 0.000]	-0.181
$MatAf[T1] \rightarrow PR[T2]$	0.035 ***	[0.018, 0.051]	0.379
$PR[T2] \rightarrow GAD[T3]$	-0.204 ***	[-0.275, -0.133]	-0.526
$GAD[T1] \rightarrow GAD[T3]$	0.312 ***	[0.263, 0.360]	1.180
Covariances			
$GAD[T1] \sim \sim MatAf[T1]$	-0.089 ***	[-0.116, -0.063]	-0.619
Factor Loadings			
T1 MatAf 1	1.000	-	-
T1 MatAf 2	0.841 ***	[0.142, 0.183]	5.478
T1 MatAf 3	0.916 ***	[0.131, 0.166]	4.978
T1 MatAf 4	0.770 ***	[0.211, 0.259]	4.257
T1 MatAf 5	0.797 ***	[0.673, 0.780]	4.378
T3 GAD 1	1.000	-	-
T3 GAD 2	0.921 ***	[0.873, 0.970]	3.504
T3 GAD 3	0.987 ***	[0.934, 1.039]	3.413
T3 GAD 4	1.093 ***	[1.035, 1.150]	3.481
T3 GAD 5	1.008 ***	[0.950, 1.067]	3.152
T3 GAD 6	0.975 ***	[0.920, 1.030]	3.241
T3 GAD 7	0.855 ***	[0.804, 0.906]	3.057
T3 GAD 8	1.141 ***	[1.080, 1.201]	3.463
T3 GAD 9	1.028 ***	[0.969, 1.087]	3.190
T3 GAD 10	0.861 ***	[0.798, 0.923]	2.533
T2 PR 1	1.000	-	-
T2 PR 2	1.055 ***	[0.966, 1.144]	2.170
T2 PR 3	2.030 ***	[1.877, 2.183]	2.431
T2 PR 4	2.143 ***	[1.985, 2.300]	2.483
Residual Variances			
T1 MatAf 1	0.458 ***	[0.426, 0.490]	2.642
T1 MatAf 2	0.245 ***	[0.226, 0.264]	2.332
T1 MatAf 3	0.379 ***	[0.353, 0.404]	2.745
T1 MatAf 4	0.220 ***	[0.203, 0.237]	2.350
T1 MatAf 5	0.203 ***	[0.186, 0.220]	2.202
T3 GAD 1	0.113 ***	[0.098, 0.129]	1.326
T3 GAD 2	0.151 ***	[0.130, 0.172]	1.324
T3 GAD 3	0.133 ***	[0.116, 0.151]	1.389
T3 GAD 4	0.173 ***	[0.150, 0.197]	1.338
T3 GAD 5	0.164 ***	[0.143, 0.185]	1.420
T3 GAD 6	0.121 ***	[0.105, 0.137]	1.361
T3 GAD 7	0.123 ***	[0.108, 0.138]	1.474
T3 GAD 8	0.133 ***	[0.112, 0.154]	1.149
T3 GAD 9	0.121 ***	[0.104, 0.139]	1.286
T3 GAD 10	0.185 ***	[0.164, 0.206]	1.610
T2 PR 1	0.389 ***	[0.368, 0.410]	3.357
T2 PR 2	0.429 ***	[0.404, 0.454]	3.150
T2 PR 3	0.146 ***	[0.128, 0.164]	1.493
T2 PR 4	0.151 ***	[0.130, 0.173]	1.278
Residual Variances			
Variance of (MatAf)[T1]	0.726 ***	[0.673, 0.780]	2.471
Variance of (GAD)[T3]	0.341 ***	[0.305, 0.377]	1.721
Variance of (PR)[T2]	0.120 ***	[0.102, 0.138]	1.253
Variance of (GAD)[T1]	0.468 ***	[0.424, 0.512]	1.960
Defined Parameters			
Indirect Effect	-0.007 ***	[-0.011, -0.003]	-0.313
Total Effect	-0.035 *	[-0.063, -0.007]	-0.225

Note. \*p < .05; \*\*\* p < .001. T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); MatAf = childhood maternal affection; GAD = generalized anxiety disorder;  $\mbox{PR}\ =\mbox{positive}\ reappraisal;$   $\mbox{CI}\ =\mbox{confidence}\ interval;$  CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual. Model fit indices:  $\chi^2(df =$ 429) = 3711.636, *p* < .001, CFI = .928, RMSEA = .067, 95% CI [0.065, 0.069], SRMR = .031.

parent. Refer to Table 7 for a summary of the longitudinal SEM mediation model.  $^{\rm 3}$ 

# 4. Discussion

The current study examined the longitudinal effects of positive reappraisal coping as a mediator in the relationship between childhood experiences (parental childhood abuse and affection) on adulthood GAD symptom severity to understand better the mechanisms that parental abuse/affection may have had on the onset and maintenance of GAD symptoms in adulthood. Our findings showed positive reappraisal coping significantly mediated the relationship of both maternal and paternal childhood abuse (examined separately and in the same model) and adulthood GAD symptom severity. Similarly, positive reappraisal coping significantly mediated the relationship between maternal/ paternal affection and GAD symptom severity (examined separately and in the same model). Specifically, participants who retrospectively reported higher levels of abuse and lower levels of parental affection during childhood from either parental figure separately displayed decreased positive reappraisal tendencies nine years later. Subsequently, reduced inclination to use positive reappraisal resulted in increased GAD symptoms in adulthood.

Our findings lend support to the idea that lower tendencies to engage in positive reappraisal could be a mechanism linking increased maternal/paternal abuse during childhood to heightened GAD symptom severity in adulthood. Specifically, these findings lend credence to the theory that maternal/paternal abuse during childhood might result in poor acquisition, usage, and consolidation of positive reappraisal strategies. The lack of deployment of positive reappraisal to regulate emotions may then serve as a risk factor for the development of GAD symptoms in adulthood. Our findings align with existing cross-sectional research (e.g., Kim & Cicchetti, 2010; Kim-Spoon et al., 2013) and extend previous research by demonstrating the adverse effects of childhood abuse on tendencies to harness positive reappraisal in adulthood. Taken together, our findings show that parental abuse from both parental figures during childhood have considerable deleterious effects on adulthood mental health and underscores the importance of emotional coping strategies such as positive reappraisal in preventing the development of GAD symptoms.

Conversely, parental emotional socialization (i.e., parental modeling, responses, and engagement with children's emotions; Eisenberg et al., 1998) might be a plausible underlying reason behind why higher levels of both maternal and paternal affection during childhood independently predicted lower GAD symptom severity in adulthood via more frequent use of positive reappraisal strategies. Indeed, prior studies (e.g., Eisenberg et al., 1998; Fabes et al., 2002; Morris et al., 2007; Saarni, 1999) have found that the development of adaptive positive reappraisal in childhood was facilitated by parents who supportively engaged in emotion socialization. Our findings support the notion that positive reappraisal tendencies at midlife might be a possible mechanism linking maternal/paternal affection and GAD symptoms in later adulthood. It is also worth noting that no studies have examined positive reappraisal in the context of an 18-year period. Our findings thus extend existing literature and suggest that parental affection during childhood could be a significant protective factor in the development of GAD symptoms in adulthood through habitual utilization and practice of positive reappraisal.

Additionally, the current study had two related and noteworthy observations. Supplementary incremental prediction analyses including both maternal and paternal abuse in the same model revealed that only maternal but not paternal abuse during childhood was significantly associated with increased GAD symptoms via decreased positive Table 6

T1 Childhood Paternal Affection Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD.

	Estimate	95% CI	Cohen's d
Regressions			
PatAf $(T1) \rightarrow GAD(T3)$	-0.013	[-0.036, 0.010]	-0.101
$PatAf[T1] \rightarrow PR[T2]$	0.043 ***	[0.029, 0.057]	0.562
$PR[T2] \rightarrow GAD[T3]$	-0.205 ***	[-0.276, -0.133]	-0.525
$GAD(T1) \rightarrow GAD(T3)$	0 314 ***	[0 266 0 363]	1 180
Covariances	0.011	[01200, 01000]	11100
$GAD[T1] \sim \sim PatAf[T1]$	-0.096 ***	[-0.123, -0.069]	-0.647
Factor Loadings	01050	[ 01120, 01000]	01017
T1 PatAf 1	1.000	-	-
T1 PatAf 2	0.803 ***	[0,779, 0,827]	6.080
T1 PatAf 3	0.836 ***	[0.809, 0.863]	5.636
T1 PatAf 4	0.784 ***	[0.760, 0.808]	5 960
T1 PatAf 5	0.871 ***	[0.846, 0.897]	6.339
T3 GAD 1	1 000	-	-
T3 GAD 2	0.921 ***	[0.873_0.970]	3 504
T3 GAD 3	0.921	[0.070, 0.970]	3 41 2
T3 GAD 4	1 093 ***	[1.035, 1.150]	3 481
T2 GAD 5	1.009 ***	[1.055, 1.150]	3 151
13 GAD 3 T2 CAD 6	0.075 ***	[0.930, 1.007]	2 241
T3 GAD 0	0.973	[0.920, 1.030]	3.057
T2 CAD 8	1 140 ***	[1.080 1.201]	3.463
	1.140	[1.060, 1.201]	2 100
13 GAD 9 T2 CAD 10	0.860 ***	[0.909, 1.067]	3.190
13 GAD 10 T2 DD 1	1.000	[0.796, 0.923]	2.333
12 PR 1 TO DD O	1.000	-	-
12 PR 2	1.055 ***	[0.966, 1.144]	2.108
12 PR 3	2.029	[1.6/0, 2.161]	2.430
12 PK 4	2.14/	[1.966, 2.303]	2.479
T1 DetAf 1	0 401 ***	TO 20F 0 4471	2 0 2 2
T1 DatAf 2	0.421	[0.395, 0.447]	2.922
T1 DatAf 2	0.244	[0.220, 0.202]	2.310
T1 DatAf 4	0.313	[0.295, 0.355]	2.091
TI Pataf 5	0.306 ***	[0.287, 0.320]	2.835
TI PALAI 5	0.243 ***	[0.226, 0.260]	2.334
TO GAD 1	0.113	[0.096, 0.129]	1.320
T2 CAD 2	0.131	[0.130, 0.172]	1.324
TO GAD 5	0.133 ***	[0.110, 0.151]	1.389
T2 CAD 5	0.1/3 ***	[0.150, 0.197]	1.338
TO GAD S	0.104 ***	[0.145, 0.185]	1.420
13 GAD 6	0.121 ***	[0.105, 0.137]	1.301
TO GAD 7	0.123 ***	[0.108, 0.158]	1.4/5
T2 CAD 0	0.133	[0.112, 0.134]	1.149
13 GAD 9	0.121 ***	[0.104, 0.139]	1.280
13 GAD 10	0.185 ***	[0.164, 0.206]	1.010
12 PR 1	0.389 ***	[0.368, 0.410]	3.357
12 PR 2	0.430 ***	[0.405, 0.455]	3.150
12 PR 3	0.14/ ***	[0.129, 0.165]	1.509
12 PK 4	0.150 ***	[0.128, 0.171]	1.267
Norionae of (Det A DIT13	0.060 ***	[0 00E 1 016]	2 150
Variance of (PatAI)[11]	0.900 ***	[0.905, 1.016]	3.130
Variance of (GAD)[13]	0.341 ***	[0.305, 0.378]	1./21
variance of (PR)[12]	0.119 ***	[0.102, 0.136]	1.251
variance of (GAD)[T1]	0.468 ***	[0.424, 0.512]	1.960
Indiroct Effect	0.000 ***		0 202
Total Effect	-0.009	[-0.013, -0.003]	-0.392
I UTAL FILCU	-0.044	1-0.040, 0.002	-0.102

*Note.*  $^{***}p < .001.$ 

T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); PatAf = childhood paternal affection; GAD = generalized anxiety disorder; PR = positive reappraisal; CI = confidence interval; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual. Model fit indices:  $\chi^2(df = 429) = 3590.401$ , p < .001, CFI = .933, RMSEA = .065, 95% CI [0.063, 0.067], SRMR = .028.

reappraisal. In contrast, when included in the same model, both maternal and paternal affection remained significantly associated with reduced GAD symptoms via increased positive reappraisal. These findings are worth noting as much of the extant literature in this area examining parental abuse or affection often did not distinguish between parental figures (e.g., Butterfield et al., 2021; Kim & Cicchetti, 2010) or did not account for paternal roles (Brumariu & Kerns, 2010; Rutter,

<sup>&</sup>lt;sup>3</sup> Sensitivity analyses showed that our results remained similar when we used multiple imputation as the missing data strategy.

Longitudinal SEM Mediation of T1 Childhood Maternal Affection Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD



Fit Statistics: χ2(df = 429) = 3711.636, p < .001, CFI = 0.928, RMSEA = 0.067, 95% CI [0.065, 0.069], SRMR = 0.031.

Fig. 3. Longitudinal SEM Mediation of T1 Childhood Maternal Affection Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD. Note. \*\*p < .01; \*\*\*p < .001. T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); MatAf = childhood maternal affection; GAD = generalized anxiety disorder; PR = positive reappraisal; CI = confidence interval; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual.  $\beta$  = unstandardized beta regression weight with standard error in parenthesis;  $\varepsilon$  = item residual variances;  $\zeta$  = factor residual variances.

Longitudinal SEM Mediation of T1 Childhood Paternal Affection Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD



Indirect effect  $\beta = -0.009^{***}$  (0.002), Total effect  $\beta = -0.022$  (0.012)

Fit Statistics:  $\chi^2(df = 429) = 3590.401$ , p < .001, CFI = 0.933, RMSEA = 0.065, 95% CI [0.063, 0.067], SRMR = .028.

Fig. 4. Longitudinal SEM Mediation of T1 Childhood Paternal Affection Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD. Note. \*\*p < .01; \*\*\*p < .001. T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); PatAf = childhood paternal affection; GAD = generalized anxiety disorder; PR = positive reappraisal; CI = confidence interval; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual.  $\beta$  = unstandardized beta regression weight with standard error in parenthesis;  $\varepsilon$  = item residual variances;  $\zeta$  = factor residual variances.

Supplemental incremental prediction analysis of T1 Childhood Maternal Affection and T1 Childhood Paternal Affection Predicting T3 GAD Severity via T2 Positive Reappraisal, controlling for T1 GAD.

	Estimate	95% CI	Cohen's $d$
Regressions			
$MatAf[T1] \rightarrow GAD[T3]$	-0.023	[-0.058, 0.011]	-0.103
$PatAf[T1] \rightarrow GAD[T3]$	0.002	[-0.027, 0.030]	0.008
$MatAf[T1] \rightarrow PR[T2]$	0.026 *	[0.004, 0.048]	0.177
$PatAf[T1] \rightarrow PR[T2]$	0.038 ***	[0.020, 0.056]	0.325
$PR[T2] \rightarrow GAD[T3]$	-0.235 ***	[-0.309, -0.161]	-0.489
$GAD[T1] \rightarrow GAD[T3]$	0.323 ***	[0.273, 0.372]	0.998
Covariances			
$GAD[T1] \sim MatAf[T1]$	-0.087 ***	[-0.113, -0.062]	-0.524
$GAD[T1] \sim PatAf[T1]$	-0.096 ***	[-0.123, -0.069]	-0.547
Factor Loadings	1 000		
11 MatAf 1 T1 MatAf 2	1.000	-	-
T1 MatAf 2	0.007	[0.830, 0.904]	3.360
T1 MatAf 4	0.764 ***	[0.000, 0.074]	3 1 2 7
T1 MatAf 5	0.822 ***	[0.785, 0.859]	3.379
T1 MatAf 6	0.531 ***	[0.496, 0.565]	2.343
T1 MatAf 7	0.773 ***	[0.727, 0.818]	2.614
T1 PatAf 1	1.000	-	-
T1 PatAf 2	0.806 ***	[0.780, 0.832]	4.720
T1 PatAf 3	0.816 ***	[0.786, 0.845]	4.294
T1 PatAf 4	0.787 ***	[0.760, 0.814]	4.467
T1 PatAf 5	0.877 ***	[0.849, 0.905]	4.836
T1 PatAf 6	0.680 ***	[0.649, 0.710]	3.385
TI PatAf 7	0.724 ***	[0.692, 0.756]	3.468
T3 GAD 1	1.000	-	-
T3 GAD 2	0.925	[0.871, 0.979]	2.041
T3 GAD 5	1.011	[0.931, 1.070]	2.399
T3 GAD 4	1.021 ***	[0.957, 1.086]	2.415
T3 GAD 6	0.994 ***	[0.932, 1.056]	2.443
T3 GAD 7	0.854 ***	[0.799, 0.909]	2.395
T3 GAD 8	1.142 ***	[1.075, 1.208]	2.622
T3 GAD 9	1.033 ***	[0.968, 1.098]	2.420
T3 GAD 10	0.898 ***	[0.826, 0.969]	1.909
T2 PR 1	1.000	-	-
T2 PR 2	1.071 ***	[0.953, 1.188]	1.397
T2 PR 3	1.969 ***	[1.773, 2.165]	1.537
IZ PR 4 Residual Variances	2.013	[1.812, 2.215]	1.530
T1 MatAf 1	0 515 ***	[0.475_0.554]	1 001
T1 MatAf 2	0.254 ***	[0.232, 0.277]	1.761
T1 MatAf 3	0.406 ***	[0.377, 0.435]	2.162
T1 MatAf 4	0.259 ***	[0.238, 0.280]	1.899
T1 MatAf 5	0.212 ***	[0.192, 0.231]	1.654
T1 MatAf 6	0.222 ***	[0.204, 0.240]	1.891
T1 MatAf 7	0.387 ***	[0.359, 0.414]	2.171
T1 PatAf 1	0.433 ***	[0.399, 0.467]	1.96
T1 PatAf 2	0.247 ***	[0.228, 0.266]	1.992
TI PatAf 3	0.356 ***	[0.333, 0.380]	2.32
T1 DotAf 5	0.308 ***	[0.286, 0.331]	2.122
T1 PatAf 6	0.242	[0.222, 0.202]	2 200
T1 PatAf 7	0.422 ***	[0.395, 0.449]	2.366
T3 GAD 1	0.123 ***	[0.103, 0.143]	0.933
T3 GAD 2	0.157 ***	[0.132, 0.182]	0.955
T3 GAD 3	0.124 ***	[0.104, 0.144]	0.943
T3 GAD 4	0.177 ***	[0.148, 0.206]	0.94
T3 GAD 5	0.164 ***	[0.137, 0.190]	0.961
T3 GAD 6	0.116 ***	[0.095, 0.136]	0.857
T3 GAD 7	0.131 ***	[0.111, 0.151]	1.011
T3 GAD 8	0.145 ***	[0.118, 0.171]	0.837
T3 GAD 9	0.128 ***	[0.106, 0.151]	0.867
13 GAD 10 T2 DD 1	0.10/ ***	[0.143, 0.191]	1.07
T2 PR 2	0.300 ***	[0.330, 0.404]	2.303 2.208
T2 PR 3	0.140 ***	[0.107, 0.173]	0.654
T2 PR 4	0.179 ***	[0.142, 0.217]	0.739
Residual Variances		,*.=-/3	
Variance of (MatAf)[T1]	0.670 ***	[0.612, 0.728]	1.775
Variance of (PatAf)[T1]	0.949 ***	[0.889, 1.008]	2.443
Variance of (GAD)[T3]	0.330 ***	[0.292, 0.367]	1.337

Table 7 (continued)

Estimate	95% CI	Cohen's d
0.127 ***	[0.105, 0.150]	0.876
0.458 ***	[0.413, 0.503]	1.565
-0.006 *	[-0.012, 0.000]	-0.165
-0.009 ***	[-0.014, -0.004]	-0.276
-0.037 *	[-0.067, -0.007]	-0.187
	Estimate 0.127 *** 0.458 *** -0.006 * -0.009 *** -0.037 *	Estimate         95% CI           0.127 ***         [0.105, 0.150]           0.458 ***         [0.413, 0.503]           -0.006 *         [-0.012, 0.000]           -0.009 ***         [-0.014, -0.004]           -0.037 *         [-0.067, -0.007]

*Note.* \**p* < .05; \*\**p* < .01; \*\*\**p* < .001.

T1 = time 1; T2 = time 2 (9 years after T1); T3 = time 3 (9 years after T2, 18 years after T1); MatAf = childhood maternal affection; PatAf = childhood paternal affection; GAD = generalized anxiety disorder; PR = positive reappraisal; CI = confidence interval; CFI = confirmatory fit index; RMSEA = root mean square error of approximation; SRMR = standardized root mean squared residual. Model fit indices:  $(\chi^2(df = 656) = 1980.437, p < .001, CFI = .988, RMSEA = .026, 95\%$  CI [0.024, 0.027], SRMR = .030)

1981). A small handful of studies pointed to maternal figures as having significantly more impact than paternal figures in terms of effects on psychological well-being (Rosenthal & Kobak, 2010) and common mental disorders (Sanghvi et al., 2023). However, other studies suggested that paternal figures might have been stronger predictors of mental health outcomes (Summers et al., 1998). Our findings seem to align more with the extant literature, which suggests that maternal (vs. paternal) abuse is especially deleterious on tendencies to engage in positive reappraisal, which may, in turn, lead to increased GAD symptoms. Maternal abuse might present a more immediate risk for adult psychopathology than paternal abuse, potentially shaped by differences in interaction frequency with each parent (Moretti & Craig, 2013). On the other hand, our findings also highlight the importance of both parental figures in the development and tendencies to engage in positive reappraisal via parental affection (perhaps via positive behavioral modeling and related processes) and its significant association with reduced GAD severity in adulthood. Taken together, these findings are vital in informing treatment targets and prevention efforts geared toward improving positive reappraisal tendencies in individuals exposed to parental abuse (especially maternal abuse) and low affection from both parental figures during childhood.

The current study had some limitations. First, parental abuse and affection were measured retrospectively, which might have been susceptible to recall bias. However, empirical evidence has supported the construct validity and retest reliability of retrospective reports of childhood experiences (Cav et al., 2022; Schauss et al., 2021; Yancura & Aldwin, 2009; Zanotti et al., 2018). Further, retrospective reports of childhood experiences demonstrated stability over time and were independent of mood (Gerlsma et al., 1993, 1994). Thus, it is unlikely that retrospective reports of childhood experiences in this study were affected by recall biases. Second, only one aspect of emotion regulation, positive reappraisal, was examined in this study. Other emotion regulation strategies exist, such as acceptance, avoidance, problem-solving, rumination, and suppression (Gross, 2014; Marr et al., 2022), which were not included in the scope of this study. Emotion regulation strategies such as suppression have been found to be maladaptive in nature and were associated with poorer outcomes, including psychopathology (Dryman & Heimberg, 2018; Hu et al., 2014). Future research should examine how childhood parental abuse or affection may affect the development and utilization of these other emotion regulation strategies in adulthood and their potential to function as mechanisms for childhood experiences predicting future GAD symptom severity. Lastly, participant demographics in the current research were mostly White, highly educated, financially and physically healthy, and married individuals (Radler & Ryff, 2010). Furthermore, the current data set did not include information on the participant's family structure during childhood. Hence, these findings may not be entirely generalizable to more culturally or socio-economically diverse contexts and could not account for non-traditional family structures. For example, childrearing

norms might differ across racial/ethnic groups and various family structures in the U.S. (Pachter et al., 2006; Weinraub & Wolf, 1983), which might substantially alter the results, warranting further research. However, the study could be a basis for exploration by future researchers on the etiology of GAD symptoms in more diverse populations. Limitations notwithstanding, study strengths included the use of longitudinal structural equation mediation modeling in ways that reduced measurement error, established temporal precedence, and improved the inferential rigor of our findings (Cole & Maxwell, 2003). Another strength was the novelty of the research question. Specifically, examining both parental roles in the context of abuse and affection during childhood separately allowed for the determination of their potentially different effects on positive reappraisal and GAD symptom severity in adulthood.

In summary, the present study found that positive reappraisal significantly mediated the relationship longitudinally between higher childhood parental abuse and lower childhood parental affection on GAD symptoms in adulthood. Examined separately, childhood maternal and paternal maltreatment was associated with decreased positive reappraisal, which led to increased GAD symptoms in adulthood. Lower childhood maternal and paternal affection were independently associated with reduced positive reappraisal, which resulted in increased GAD symptoms in adulthood. Examined concurrently, only maternal abuse was significantly associated with elevated GAD symptoms via decreased positive reappraisal tendencies. However, both maternal and paternal affection remained significant predictors of lower GAD symptoms via positive reappraisal coping. These findings highlight positive reappraisal as a potential underlying mechanism linking childhood experiences to the development and maintenance of psychopathology, which may have important practical implications for the treatment and prevention of elevated GAD symptoms.

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# CRediT authorship contribution statement

Zainal Nur Hani: Methodology, Supervision, Writing – original draft, Writing – review & editing. Ng Matthew H. S.: Conceptualization, Data curation, Methodology, Visualization, Writing – original draft, Writing – review & editing. Newman Michelle Gayle: Supervision, Writing – original draft, Writing – review & editing.

### **Declaration of Competing Interest**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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