



Research paper

## Trait anger expression mediates childhood trauma predicting for adulthood anxiety, depressive, and alcohol use disorders

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## ARTICLE INFO

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Anger-In, anger expression–internal  
 Anger-Out, anger expression–external  
 AUD, alcohol use disorder  
 CTQ, retrospective childhood trauma questionnaire  
 GAD, generalized anxiety disorder  
 MDD, major depressive disorder  
 PD, panic disorder  
 STAXI, State-Trait Anger Expression Inventory  
 T1, Time 1  
 T2, Time 2  
 T3, Time 3

## ABSTRACT

**Background:** General aggression and evolutionary models posit that more severe early exposure experiences to trauma (physical, emotional, sexual abuse and/or neglect) place one at risk for adulthood psychopathology through heightened trait anger expression–internal (Anger-In) and external (Anger-Out). However, there are a dearth of empirical studies explaining the longitudinal childhood maltreatment–adulthood psychopathology relation.

**Objective:** Therefore, this study investigated if childhood maltreatment exposure severity predicted elevated adulthood major depressive disorder (MDD), generalized anxiety disorder (GAD), panic disorder (PD), and alcohol use disorder (AUD). Moreover, we tested if trait anger expression – internal and external – mediated the childhood maltreatment–adulthood MDD, GAD, PD, and AUD symptom associations.

**Method:** Participants took part in two waves of measurement spaced approximately 9 years apart. Time 1 childhood trauma severity (retrospectively-reported Childhood Trauma Questionnaire), Time 2 Anger-In and Anger-Out (State-Trait Anger Expression Inventory), and Time 3 adulthood MDD, GAD, PD (Composite International Diagnostic Interview–Short Form), and AUD (Alcohol Screening Test) diagnoses were measured.

**Results:** Anger-Out and Anger-In partially mediated the relations between childhood trauma severity and adulthood psychopathology diagnoses after adjusting for Time 2 symptoms. Higher Time 1 childhood trauma severity was related to greater Time 2 Anger-Out and Anger-In, and increased Time 2 Anger-Out and Anger-In were thereby related to elevated Time 3 adulthood MDD, PD and AUD, but not GAD severity. Trait anger accounted for 14 to 50% of the variance of childhood trauma–adulthood MDD, PD and AUD relations.

**Discussion:** Theoretical and clinical implications, such as the need for trauma-informed care, are discussed.

Childhood maltreatment is defined as repeated exposure to physical or emotional abuse and the lack of a consistently safe and supportive environment necessary for optimal child health and development, at the hands of a primary caregiver (Cicchetti and Toth, 2005). In the U.S., it is estimated that 1 in 4 children have endured some form of abuse and/or neglect in the past year or during their lifetime (Finkelhor et al., 2013). Further, it is well established that more frequent childhood maltreatment experiences significantly predict later development of various neurological changes and psychiatric disorders in adolescence and adulthood, including anxiety, depressive, and alcohol use disorders (Busso et al., 2017; Opel et al., 2019; Skinner et al., 2016). Such disorders then pose a risk for a wide array of cardiovascular, respiratory, neuroendocrine, cognitive, and autoimmune diseases (Lamers et al., 2019; Zainal and Newman, 2021; Zainal and Newman, in press). These disorders also incur substantial costs in terms of increased health care utilization, suicidality, disability, unemployment, and absenteeism

(Birnbaum et al., 2010; Newman et al., 2013). Given these individual and societal costs, understanding the mechanisms underlying the relations between childhood trauma and adulthood psychopathology is important.

*Developmental models* theorize that maltreated children exhibit increased problems with emotion regulation compared to those with minimal exposure to abuse or neglect. Further, the model proposes that these emotional dysregulation tendencies are linked to the development of later internalizing and externalizing psychopathology (Cicchetti and Rogosch, 2009; Cicchetti et al., 1995). Consistent with theory, studies have observed that more frequent exposure to childhood abuse and neglect was a precursor of later psychiatric disorders (Chen et al., 2010; Goodwin et al., 2005; Hagborg et al., 2020; Milojevich et al., 2019). For example, meta-analytic data (Chen et al., 2010) showed that across 22 prospective investigations, individuals with (vs. without) exposure to early childhood abuse were 3 times more likely to experience anxiety

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disorders, and 2.6 times more likely to develop depression during their lifetime. Extending those findings, exposure to physical and/or emotional abuse in childhood longitudinally predicted the development of major depressive disorder (MDD), but not generalized anxiety disorder (GAD), 4 to 14 years later in non-clinical adolescents (Milojević et al., 2019). Similarly, childhood sexual and physical abuse longitudinally forecasted panic disorder (PD) up to 21 years later in community-dwelling young adults (Goodwin et al., 2005). Recently, a study demonstrated that childhood maltreatment dovetailed with the development of alcohol use disorder (AUD) following 1 to 2 years in community-dwelling Swedish adolescents (Hagborg et al., 2020). Collectively, evidence indicates that more childhood maltreatment experiences were linked to higher future depression, anxiety, and AUD symptoms across various developmental stages.

Identifying the factors that mediate the association between recurrent exposure to childhood maltreatment and adulthood depression, anxiety, and AUDs is essential to clinical science for several reasons. Perhaps most importantly, it can inform and refine abuse- and trauma-focused cognitive behavioral therapy (TF-CBT; Cohen et al., 2006), one of the most evidence-based treatments for children following abuse or neglect exposure thus far (Leenarts et al., 2013). A basic science study that uncovers the mediating factors underlying the naturalistic childhood maltreatment-adulthood psychopathology relation thus has the potential to optimize TF-CBT outcomes. For example, change in dysfunctional cognitions following trauma exposure mediated the reduction of posttraumatic stress symptoms throughout TF-CBT (Pfeiffer et al., 2017). On that note, parental support and children's abuse-related attributions and perceptions (e.g., self-blame, impaired trust) were also found to be important mediators of anxiety, depression, and PTSD symptom persistence and TF-CBT (Cohen and Mannarino, 2000). In addition, emotion regulation mediated improvement in internalizing and externalizing symptoms throughout the course of TF-CBT (Thornback and Muller, 2015).

One potential mechanism linking childhood maltreatment and depressive, anxiety, and AUDs is trait anger expression. It is common for children to feel prolonged anger after experiencing maltreatment, and the inability to cope with such emotions may lead to internalizing symptoms (e.g., avoidance, intrusive thoughts) and externalizing symptoms (e.g., lashing out behaviors). The *general aggression model* (Anderson and Bushman, 2002) proposes that frequent exposure to maltreatment by primary caregivers could, via modeling, promote the belief for the need to express anger to cope with life challenges. To date, at least four prospective studies have supported the general aggression model. For example, chronic neglect over any 2-year period during early childhood to pre-adolescence longitudinally predicted aggression and delinquency 2 to 14 years later in community-dwelling adolescents (Logan-Greene and Semanchin Jones, 2015). Consistent with those findings, neglect during the first 2 years of life predicted greater aggression after 2 to 6 years in relatively healthy kids (Kotch et al., 2008). Additionally, mother-to-child physical aggression predicted later parent-directed physical aggression and property damage following 2 to 3 years in nonclinical adolescents (Margolin et al., 2010). Moreover, parent-reported physical abuse significantly predicted greater anger 34 to 35 years later among community-based adults (Herrenkohl et al., 2012).

Relatedly, *evolutionary models* propose that relations between dispositional anger expression and future depression, anxiety, and AUDs may be explained by the behavioral inhibition system (BIS) and behavioral activation system (BAS) (Gray, 1990). The BAS is thought to regulate appetitive motivational responses and approach behavior, whereas the BIS modulates aversive motivational responses and avoidance behavior. Trait anger expression relates to both of these biological systems. More specifically, outward expression of anger (Anger-Out) was positively associated with BAS and negatively associated with BIS levels (Smits and Kuppens, 2005). This finding was explained as being due to BAS involving antagonism as an approach motivation. By

contrast, greater inward expression of anger (Anger-In) is thought to be related to higher BIS and lower BAS levels, because it involves inhibition of overt anger expression or hostility.

Additionally, *biopsychosocial models* propose that the presence of certain temperamental traits (e.g., high trait negative affect, negative emotional reactivity, anxiety sensitivity) places one at greater risk for future problems with anger expression control and psychopathology following early maltreatment (Spinhoven et al., 2016; Zhang et al., 2018). In particular, those more physiologically reactive to stress can become prone to anger subsequent to experiences of family conflict and related interpersonal stressors (El-Sheikh and Erath, 2011; Sun et al., 2020). Physiologically sensitive individuals may perceive familial dysfunction as more traumatic, stressful, and anger-provoking than those who are less reactive. Consistent with biopsychosocial theories, individuals with persistent or recently developed sensitivity to interpersonal conflict experienced significantly greater incidences of anger, hostility, and anxiety 2 years later compared to those without interpersonal sensitivity in a sample of nonclinical adolescents (Sun et al., 2020).

To date, six prospective studies (Harty et al., 2017; Izadpanah et al., 2017; Nozadi et al., 2015; Park et al., 2017; Stewart et al., 2010; Stringaris et al., 2009) and one cross-sectional study (de Bles et al., 2019) have shown that dispositional anger expression was linked to various mental health problems across the life span. First, anger rumination (a dimension of Anger-In) predicted depression and anxiety symptoms as well as aggression 5 years later in community-dwelling adults and adolescents (Izadpanah et al., 2017). Similarly, anger rumination longitudinally predicted heightened depression 6 years later in healthy middle-aged and elderly adults (Stewart et al., 2010). Likewise, irritability during early adolescence predicted GAD and MDD 20 years later in general adults (Stringaris et al., 2009). Also, Anger-Out predicted anxiety and depression symptoms following 4 to 8 months in nonclinical Mexican adolescents (Park et al., 2017). Further, anger-irritability (an Anger-Out dimension) predicted alcohol usage 5 years later in community-based adolescents with a history of attention-deficit disorder (Harty et al., 2017). Additionally, early childhood anger significantly predicted GAD symptoms 18 to 30 months later (Nozadi et al., 2015). A cross-sectional study also found that trait anger and anger attacks were associated with GAD symptoms (de Bles et al., 2019). Collectively, the evidence suggests that higher Anger-Out and Anger-In would plausibly be related to greater future depression, anxiety, and AUD symptom severity across long durations.

On the basis of the theoretical and empirical literature outlined earlier, we tested two overarching hypotheses to advance the understanding of the relations between childhood trauma and adulthood anxiety, depressive, and AUD severity. Based on theory and evidence, we hypothesized that greater retrospective childhood trauma would be associated with higher levels of Anger-In and Anger-Out. Moreover, we hypothesized that higher Anger-In and Anger-Out would be related to more adulthood GAD, MDD, PD, and AUD symptom severity. Further, we predicted that Anger-In and Anger-Out would significantly mediate the relations between childhood trauma and GAD (Hypotheses 1A and 1B), MDD (Hypotheses 2A and 2B), PD (Hypotheses 3A and 3B) and AUD (Hypotheses 4A and 4B) symptom severity.

## 1. Method

**Participants.** Participants were part of the Midlife Development in the United States (MIDUS) project across two waves of data collection: 2004 to 2005; and 2012 to 2013 (Brim et al., 2019; Ryff et al., 2017, 2019). This study did not require IRB approval, since it utilized a publicly available data set that was accessible through an online data repository (<https://www.icpsr.umich.edu/icpsr-web/ICPSR/series/203>). Participants ( $n = 3294$ ) averaged 45.62 years ( $SD = 11.41$ , range = 24–74 years) at baseline, 54.61% were female, and 41.70% had college education. The majority of the sample were White participants

(89.01%), and the remaining 10.99% were African American, Asian, Native American, or Pacific Islander. The proportion of participants who met diagnostic threshold for MDD, GAD, PD, and AUD were 9.93% ( $n = 327$ ), 1.94% ( $n = 64$ ), 5.19% ( $n = 171$ ), and 6.13% ( $n = 202$ ), respectively. Tables 1 and 2 show the descriptive statistics and correlation matrix of all the study variables.

**Procedures.** The present investigation focused on participants who consented to complete in-person psychiatric diagnostic interviews as well as self-report assessments of child maltreatment and trait anger expression. Time 1 (T1) self-reported retrospective childhood maltreatment exposure severity and Time 2 (T2) dispositional anger expression (Anger-In and Anger-Out) were measured from 2004 to 2005. T2 and Time 3 (T3) GAD, MDD, AUD, and PD symptom severity were assessed at both study visits (from 2004 to 2005 and from 2012 to 2013).

**T1 Retrospectively-Recalled Childhood Trauma.** Various childhood trauma experiences were evaluated with the retrospectively recalled 28-item Childhood Trauma Questionnaire (CTQ; Bernstein and Fink, 1998; Bernstein et al., 1994). It assessed the frequency and severity of traumatic childhood experiences along five sub-scales: Emotional Abuse (EA;  $\alpha = 0.89$ ), Physical Abuse (PA;  $\alpha = 0.86$ ), Sexual Abuse (SA;  $\alpha = 0.95$ ), Emotional Neglect (EN;  $\alpha = 0.91$ ), and Physical Neglect (PN;  $\alpha = 0.78$ ) (Bernstein and Fink, 1998). In this study, the Cronbach’s alpha for EA, PA, SA, EN, and PN were 0.89, 0.83, 0.94, 0.89, and 0.71, respectively. Respondents rated each CTQ item on a 5-point Likert-scale (1 = *Never true* to 5 = *Very often true*). Examples of items include “People in my family said hurtful or insulting things to me” (EA); “I got hit or beaten so badly that it was noticed by someone like a teacher, neighbor, or doctor” (PA); “Someone touched me in a sexual way, or tried to make me touch them” (SA); and “I had to wear dirty clothes” (PN). The scale also has good convergent and discriminant validity, as well as strong 2- to 6-month retest reliability (intraclass correlation = 0.88) (Bernstein et al., 1994).

**T2 Trait Anger Expression.** Anger proneness (trait anger) was assessed with the self-report State-Trait Anger Expression Inventory (STAXI; Spielberger, 1996)–Anger Expression subscales. The scale evaluates suppressing angry feelings and brooding over them internally (Anger-In;  $\alpha = 0.73$ – $0.74$ ) and externally expressing angry feelings by taking them out on other people or objects (Anger-Out;  $\alpha = 0.75$ – $0.77$ ) (Spielberger, 1996). In this study, the  $\alpha$ s for Anger-In and Anger-Out were 0.81 and 0.77 respectively. Respondents rated each STAXI item on a 4-point Likert-scale (1 = *Almost never* through 4 = *Almost always*). The STAXI also has good convergent and discriminant validity, as well as two-week test-retest reliability ( $r = 0.64$ – $0.82$ ) across various populations

**Table 1**  
Correlation Matrix of Study Variables.

	1	2	3	4	5	6	7	8	9	10	11	12
1. Age	–											
2. Gender	–0.002	–										
3. T1 CTQ	–0.14***	.12***	–									
4. T2 STAXI–Anger-In	–0.24***	–0.04	.16***	–								
5. T2 STAXI–Anger-Out	–0.19***	–0.03	.18***	.18***	–							
6. T2 MDD Severity	–0.11***	.11***	.28***	.20***	.09**	–						
7. T2 GAD Severity	–0.08***	.07***	.16***	.14***	.04	.34***	–					
8. T2 PD Severity	–0.09***	.11***	.22**	.10***	.05	.27***	.15***	–				
9. T2 AUD Severity	–0.10***	–0.06**	.09***	.12***	–0.03	.11***	.07***	.14***	–			
10. T3 MDD Severity	–0.09***	.08***	.28***	.21***	.16***	.34***	.23***	.21***	.04	–		
11. T3 GAD Severity	–0.09***	.07***	.22***	.10***	.09**	.22***	.42***	.17***	.02	.34***	–	
12. T3 PD Severity	–0.10***	.08***	.15***	.10**	.08*	.20***	.13***	.34***	.05**	.27***	.23***	–
13. T3 AUD Severity	–0.11***	–0.07**	.06	.13***	.05	.04*	.01	.04*	.31***	.06***	.02	.09**

Note. \*\*  $p < .01$

\*\*\*  $p < .001$ . Note. \*\*\*  $p \leq 0.001$

\*\*  $p \leq 0.01$ .

\*  $p \leq 0.05$ .  $M$  = mean;  $n$  = number of participants;  $SD$  = standard deviation; Min = minimum; Max = maximum; CTQ = childhood trauma severity; STAXI-Anger-In = trait anger expression internal; STAXI-Anger-Out = trait anger expression external; MDD = major depressive disorder severity; GAD = generalized anxiety disorder severity; PD = panic disorder severity; AUD = alcohol use disorder severity; T1 = time 1; T2 = time 2; T3 = time 3; VF = verbal fluency.

**Table 2**  
Descriptive Statistics of Study Variables.

	$M$ or $n$	$SD$ or %	Min	Max	Skewness	Kurtosis
1. Age	45.62	11.41	20.00	74.00	0.24	–0.70
2. Gender	1.58	0.69	1.00	8.00	4.29	38.86
3. T1 CTQ	37.45	13.88	25.00	114.00	1.99	4.65
4. T2 STAXI–Anger-In	14.62	4.06	8.00	31.00	0.73	0.52
5. T2 STAXI–Anger-Out	12.83	3.13	8.00	28.00	0.90	1.30
6. T2 MDD Severity	0.60	1.71	0.00	7.00	2.71	5.83
7. T2 GAD Severity	0.13	0.87	0.00	10.00	7.66	61.93
8. T2 PD Severity	0.31	1.00	0.00	6.00	3.50	11.91
9. T2 AUD Severity	0.07	0.37	0.00	4.00	7.08	58.64
10. T3 MDD Severity	0.60	1.71	0.00	7.00	2.69	5.74
11. T3 GAD Severity	0.13	0.92	0.00	10.00	7.89	65.66
12. T3 PD Severity	0.27	0.92	0.00	6.00	3.73	13.94
13. T3 AUD Severity	0.10	0.46	0.00	5.00	6.37	48.49

Note.  $M$  = mean;  $n$  = number of participants;  $SD$  = standard deviation; Min = minimum; Max = maximum; CTQ = childhood trauma severity; STAXI-Anger-In = trait anger expression internal; STAXI-Anger-Out = trait anger expression external; MDD = major depressive disorder severity; GAD = generalized anxiety disorder severity; PD = panic disorder severity; AUD = alcohol use disorder severity; T1 = time 1; T2 = time 2; T3 = time 3; VF = verbal fluency.

(Bishop and Quah, 1998; Jacobs et al., 1988; Nakajima et al., 2016).

**T2 and T3 Symptom Severity.** T2 and T3 MDD, PD, and GAD symptom severity scores were based on the Diagnostic and Statistical Manual–Third Edition–Revised (DSM-III-R) Composite International Diagnostic Interview–Short Form (CIDI-SF; American Psychiatric Association, 1987; Kessler et al., 1998; Wittchen, 1994). All diagnoses were reported based on the prior 12 months. MDD severity was based on 7 symptoms (depressed mood, appetite changes, fatigue, suicidal ideation, sleep disturbances, anhedonia, concentration issues), wherein participants endorsed the presence (coded as ‘1’) or absence (coded as ‘0’) of each symptom. Thus, the MDD severity score could range from 0 to 7. For GAD severity, respondents reported the frequency and degree to which they experienced a series of symptoms due to their worries

(6-item; restlessness, muscle soreness or tension, irritability, trouble focusing, sleep disturbances, easily fatigued) on a 4-point Likert-scale (0 = *never* to 3 = *most days*) (i.e., score range = 0 to 18). For PD severity, respondents disclosed panic symptoms encountered in the past-year (6-item; heart palpitations, chest or stomach discomfort, hot flashes, trembling or shaking, sweating, surroundings seem unreal), such that respondents reported the presence (coded as '1') or absence (coded as '0') of each symptom (i.e., score range = 0 to 6). For MDD, PD, and GAD, symptom severity, the CIDI-SF has been shown to have good internal consistency (in this study, MDD, PD, and GAD  $\alpha$  = 0.94, 0.98, and 0.81 at T2;  $\alpha$  = 0.94, 0.99, and 0.83, respectively, at T3), strong retest-reliability, and excellent sensitivity (89.6–96.6%) and specificity (93.9–99.8%) (Kessler et al., 1998). To measure AUD symptom severity, participants disclosed any alcohol-related problems (4-item; paranoia or depression due to alcohol, strong desire to use alcohol, tolerance to the effects of alcohol, spending excessive time drinking) by responding "Yes" (coded as '0') or "No" (coded as '1') on the Alcoholism Screening Test (AST) (Selzer, 1971) (i.e., score range = 0 to 4). The AST has shown good internal consistency ( $\alpha$  = 0.94 at T2 and  $\alpha$  = 0.99 at T3 in this study), retest-reliability, sensitivity, and specificity compared to clinical interview-derived AUD diagnoses (Grzywacz and Marks, 1999; Selzer, 1971).

### 1.1. Data analyses

Structural equation modeling (SEM) mediation analyses were conducted using the *lavaan R* package (Rosseel, 2012) with *RStudio* software (Version 1.3.959). To assess model fit, we used the confirmatory fit index (CFI; Bentler, 1990), root mean square error of approximation (RMSEA; Steiger, 1990), and standardized root mean square residual (SRMR; Hu and Bentler, 1999). Mediation analyses were carried out with a product-of-coefficients method of the indirect effects ( $a \times b$ ) for the regression coefficients of CTQ score predicting trait anger (Anger-In or Anger-Out) ( $a$  path), and trait anger predicting adulthood GAD, MDD, PD, or AUD severity ( $b$  path). We displayed the unstandardized regression coefficients ( $\beta$ ), standard errors ( $SE$ ), and utilized bootstrapping with 10,000 resampling draws (Cheung and Lau, 2008). The mediation effect size refers to the proportion of the indirect effect ( $a*b$ ) relative to the total effect,  $c = a*b + c'$  i.e., percentage of variance wherein trait anger explained the childhood trauma–adulthood psychopathology relation (Cheung and Lau, 2008; Preacher and Kelley, 2011; Wen and Fan, 2015). In total, there was 4.85% missing data. Full information maximum likelihood (FIML) was used to handle missing data, given that our data was missing at random (Graham, 2009). Also, Cohen's  $d$  effect size was computed for each beta weight with the formula ( $d = 2t/\sqrt{df}$ ), where  $d$  values of 0.2, 0.5, and 0.8 signified small, moderate, and large effect sizes, respectively (Cohen, 1988; Dunlap et al., 1996).

## 2. Results

### 2.1. Direct effects: childhood trauma predicting adulthood mental health outcomes

The model of T1 CTQ  $\rightarrow$  T3 diagnostic severity had good fit for T3 GAD ( $\chi^2(df = 116) = 182.34, p < .01, CFI = 0.92, TLI = 0.91, RMSEA = 0.07, 90\% CI [0.05, 0.09], SRMR = 0.07$ ), MDD ( $\chi^2(df = 149) = 232.874, p < .01, CFI = 0.98, TLI = 0.98, RMSEA = 0.04, 90\% CI [0.03, 0.05], SRMR = 0.04$ ), PD ( $\chi^2(df = 116) = 202.91, p < .01, CFI = 0.99, TLI = 0.99, RMSEA = 0.05, 90\% CI [0.04, 0.06], SRMR = 0.02$ ), and AUD ( $\chi^2(df = 62) = 127.74, p < .01, CFI = 0.99, TLI = 0.99, RMSEA = 0.05, 90\% CI [0.04, 0.06], SRMR = 0.02$ ). Greater baseline retrospective CTQ was significantly associated with higher T3 MDD ( $\beta = 0.01, SE = 0.00, t = 3.66, p < .01, d = 0.60$ ) and PD severity ( $\beta = 0.01, SE = 0.01, t = 1.95, p = .05, d = 0.36$ ). However, greater baseline retrospective CTQ was not significantly associated with T3 GAD ( $\beta = -0.02, SE = 0.01, t = -1.76, p = .08, d = -0.33$ ) or AUD severity ( $\beta = 0.01, SE = 0.01, t = 1.42, p =$

.16,  $d = 0.26$ ).

### 2.2. Indirect effects: childhood trauma predicting mental health outcomes via anger-in and anger-out

When Anger-In was the mediator, the model demonstrated excellent fit for GAD ( $\chi^2(df = 270) = 239.05, p = .91, CFI = 1.00, TLI = 1.02, RMSEA = 0.00, 90\% CI [0.00, 0.02], SRMR = 0.08$ ), MDD ( $\chi^2(df = 319) = 475.11, p < .01, CFI = 0.98, TLI = 0.98, RMSEA = 0.02, 90\% CI [0.02, 0.03], SRMR = 0.05$ ), PD ( $\chi^2(df = 270) = 298.56, p = .11, CFI = 1.00, TLI = 1.00, RMSEA = 0.01, 90\% CI [0.00, 0.02], SRMR = 0.04$ ), and AUD ( $\chi^2(df = 184) = 244.60, p < .01, CFI = 0.99, TLI = 0.99, RMSEA = 0.02, 90\% CI [0.01, 0.03], SRMR = 0.04$ ). Similarly, when Anger-Out was the mediator, the model illustrated good fit for GAD ( $\chi^2(df = 270) = 190.38, p = 1.00, CFI = 1.00, TLI = 1.05, RMSEA = 0.00, 90\% CI [0.00, 0.00], SRMR = 0.08$ ), MDD ( $\chi^2(df = 185) = 280.918, p < .01, CFI = 0.98, TLI = 0.98, RMSEA = 0.02, 90\% CI [0.02, 0.03], SRMR = 0.05$ ), PD ( $\chi^2(df = 270) = 330.34, p = .01, CFI = 0.99, TLI = 0.99, RMSEA = 0.02, 90\% CI [0.01, 0.02], SRMR = 0.04$ ), and AUD ( $\chi^2(df = 184) = 314.58, p < .01, CFI = 0.97, TLI = 0.97, RMSEA = 0.03, 90\% CI [0.02, 0.03], SRMR = 0.04$ ).

**Adulthood GAD Severity.** Contrary to Hypothesis 1A, although childhood trauma was significantly positively associated with Anger-In ( $\beta = 0.05, SE = 0.02, t = 2.46, p = .014, d = 0.30$ ), Anger-In was not substantially related to adulthood GAD severity 9 years later ( $\beta = -0.00, SE = 0.10, t = -0.03, p = .97, d = -0.0037$ ). The childhood trauma  $\rightarrow$  Anger-In  $\rightarrow$  adulthood GAD symptoms indirect effect was also not significant ( $\beta = -0.00, SE = 0.01, t = -0.03, p = .97, d = -0.0037$ ). Similarly, contrary to Hypothesis 1B, childhood trauma was not related to Anger-Out ( $\beta = 0.01, SE = 0.01, t = 1.65, p = .10, d = 0.20$ ), and Anger-Out was not linked to adulthood GAD severity 9 years later ( $\beta = -0.20, SE = 0.18, t = -1.16, p = .25, d = 0.14$ ). The childhood trauma  $\rightarrow$  Anger-Out  $\rightarrow$  adulthood GAD symptoms indirect effect was also not significant ( $\beta = -0.00, SE = 0.00, t = -0.91, p = .36, d = 0.11$ ).

**Adulthood MDD Severity.** Supporting Hypothesis 2A, childhood trauma was significantly positively associated with Anger-In ( $\beta = 0.05, SE = 0.01, t = 4.92, p < .01, d = 0.55$ ), and Anger-In was significantly positively related to adulthood MDD symptoms 9 years later ( $\beta = 0.04, SE = 0.01, t = 3.24, p < .01, d = 0.36$ ). The childhood trauma  $\rightarrow$  Anger-In  $\rightarrow$  adulthood MDD severity indirect effect was also significant ( $\beta = 0.002, SE = 0.001, t = 2.87, p < .01, d = 0.32$ ). Anger-In accounted for 25% of the childhood trauma–adulthood MDD severity relation. Consistent with Hypothesis 2B, childhood trauma was also significantly positively associated with Anger-Out ( $\beta = 0.01, SE = 0.003, t = 4.42, p < .01, d = 0.49$ ), and Anger-Out was significantly positively related to future MDD symptoms ( $\beta = 0.09, SE = 0.03, t = 3.20, p < .01, d = 0.36$ ). Further, the childhood trauma  $\rightarrow$  Anger-Out  $\rightarrow$  adulthood MDD severity indirect effect was significant ( $\beta = 0.001, SE = 0.000, t = 2.73, p < .01, d = 0.31$ ). Anger-Out accounted for 14.29% of the childhood trauma–adulthood MDD severity relation.

**Adulthood PD Severity.** Supporting Hypothesis 3A, childhood trauma was significantly positively associated with Anger-In ( $\beta = 0.03, SE = 0.01, t = 4.38, p < .01, d = 0.53$ ), and Anger-In was significantly positively related to adulthood PD symptoms 9 years later ( $\beta = 0.11, SE = 0.04, t = 3.00, p < .01, d = 0.37$ ). The childhood trauma  $\rightarrow$  Anger-In  $\rightarrow$  adulthood PD severity indirect effect was also significant ( $\beta = 0.004, SE = 0.002, t = 2.49, p < .01, d = 0.30$ ). Anger-In accounted for 40% of the childhood trauma–adulthood PD severity relation. Supporting Hypothesis 3B, higher childhood trauma was significantly associated with greater Anger-Out ( $\beta = 0.01, SE = 0.003, t = 4.27, p < .01, d = 0.52$ ), and higher Anger-Out was significantly related to more adulthood PD symptoms 9 years later ( $\beta = 0.20, SE = 0.09, t = 2.15, p = .03, d = 0.26$ ). Moreover, the childhood trauma  $\rightarrow$  Anger-Out  $\rightarrow$  adulthood PD severity indirect effect was also significant ( $\beta = 0.002, SE = 0.001, t = 2.00, p = .05, d = 0.24$ ). Anger-Out accounted for 20% of the childhood trauma–adulthood PD severity relation.

**Adulthood AUD Severity.** Supporting Hypothesis 4A, childhood trauma was significantly positively associated with Anger-In ( $\beta = 0.02$ ,  $SE = 0.01$ ,  $t = 4.26$ ,  $p < .01$ ,  $d = 0.63$ ), and Anger-In was significantly positively related to adulthood AUD symptoms 9 years later ( $\beta = 0.21$ ,  $SE = 0.06$ ,  $t = 3.59$ ,  $p < .01$ ,  $d = 0.53$ ). The childhood trauma  $\rightarrow$  Anger-In  $\rightarrow$  adulthood AUD severity indirect effect was also significant ( $\beta = 0.01$ ,  $SE = 0.002$ ,  $t = 2.92$ ,  $p < .01$ ,  $d = 0.43$ ). Anger-In accounted for 50% of the childhood trauma–adulthood AUD severity relation. Supporting Hypothesis 4B, childhood trauma was significantly positively connected with Anger-Out ( $\beta = 0.01$ ,  $SE = 0.00$ ,  $t = 4.41$ ,  $p < .001$ ,  $d = 0.65$ ), and Anger-Out was significantly positively linked to adulthood AUD symptoms 9 years later ( $\beta = 0.34$ ,  $SE = 0.17$ ,  $t = 2.02$ ,  $p = .04$ ,  $d = 0.30$ ). The childhood trauma  $\rightarrow$  Anger-Out  $\rightarrow$  adulthood AUD severity indirect effect was also significant ( $\beta = 0.004$ ,  $SE = 0.002$ ,  $t = 2.05$ ,  $p = .04$ ,  $d = 0.30$ ). Anger-Out accounted for 40% of the childhood trauma–AUD severity relation.

### 3. Discussion

This study builds upon existing knowledge concerning the mechanism that places one at risk of adulthood psychopathology following childhood maltreatment. To our knowledge, this is the first study to examine dispositional anger expression as a mediator of the childhood maltreatment–adulthood psychopathology relation. After controlling for TrTime 2 psychopathology, both Anger-Out and Anger-In significantly mediated the relations between childhood trauma and adulthood MDD, PD, and AUD symptom severity (but not GAD severity). Anger-In accounted for 25%, 40%, and 50% of the variance of the association between childhood trauma and adulthood MDD, PD, and AUD, respectively. Simultaneously, Anger-Out explained 14%, 20%, and 40% of the variance of the connections between retrospective childhood trauma and adulthood MDD, PD, and AUD, respectively. Overall, findings partially support our hypotheses and are generally consistent with developmental, evolutionary, and general aggression models (Anderson and Bushman, 2002; Cicchetti and Rogosch, 2009; Gray, 1990). We offer potential explanations to refine frameworks on this subject matter.

Why did experiences of childhood trauma lead to subsequent increase in dispositional anger expression levels? Based on the general aggression model and related literature, it is plausible that individuals maltreated during childhood were more likely to experience dispositional anger, via behavioral modeling of hostility reinforced by their primary caregivers with emotion regulation deficits (Bandura and Rosenthal, 1978; Wang et al., 2020). In addition to behavioral modeling, tendencies toward violence and anger rumination can also develop due to deficiencies in social information-processing skills often present in individuals with early history of trauma (see meta-analysis by Luke and Banerjee, 2013). Specifically, children raised in a dysfunctional home environment may constantly look for subtle cues of a caregiver's emotional change to prepare themselves for a potential outburst. This can result in developing a bias toward interpreting hostility in the ambiguous actions and intentions of others, which, in turn, can lead to higher dispositional anger expression. Supporting these speculations is evidence that hostile attribution bias was associated with aggression in individuals who experienced early trauma (Zhu et al., 2020). Our longitudinal results extend such findings, and future prospective research can try to replicate and evaluate our propositions.

With regard to Anger-In and Anger-Out as mediators between retrospective childhood trauma and adulthood MDD, AUD, and PD, the present findings support biopsychosocial theories of childhood maltreatment. Biopsychosocial models propose that physiologically sensitive individuals with deficiencies in regulating high intensity, low threshold anger (e.g., self-blame, difficulties with self-soothing), may cope with relationship stressors in various maladaptive ways that exacerbate psychiatric problems (Crow et al., 2014; Poole et al., 2017; Rosenstein et al., 2018). Explained differently, those who are naturally more physiologically sensitive will experience greater levels of stress

and negative emotions in response to trauma compared to those who are less sensitive, and their intense reactions to the traumatic events may exacerbate their emotional reactivity, leading to difficulty regulating and coping with their anger in the future. These assertions are consistent with evidence that frequent childhood maltreatment and biological alterations (e.g., buildup of cortisol, blunted prefrontal cortex activation) coincided with greater future impulsive and other emotionally dysregulated behaviors (Bridgett et al., 2015). Further, less belief in one's own ability to regulate anger longitudinally predicted greater internalizing and externalizing symptoms (Di Giunta et al., 2018). Moreover, greater problems in managing anger predicted higher future substance usage in adolescents (McKee et al., 2020; Mergler et al., 2018). Perhaps this reflects how some individuals attempt to manage their anger by numbing themselves with alcohol or similar, harmful drug dependencies. Future studies can test these hypotheses.

The nonsignificant relationship between childhood trauma and adulthood GAD (vs. PD, AUD, and MDD) via anger is counterintuitive, as it largely does not align with the majority of past literature. For example, our results differ from a previous study's finding that anger at 54 months of age positively predicted mother-reported GAD symptoms 18 to 30 months later (Nozadi et al., 2015). In addition, it was at odds with the cross-sectional result that higher levels of trait anger and anger attacks were significantly associated with greater GAD (de Bles et al., 2019). Likewise, our findings are contrary to the meta-analytic data across 7 prospective studies that found those with early exposure to childhood abuse (vs. without) were significantly more likely to experience anxiety disorders in their lifetimes (Chen et al., 2010). However, one study that was partially aligned with our results found that adolescents with greater exposure to sexual or emotional abuse, but not neglect, longitudinally predicted MDD, but not GAD (Milojevich et al., 2019). These discrepancies may be in part explained by differences in sample characteristics (adults vs. children), measures used, and study design (e.g., cross-sectional nature of the de Bles et al. (2019) paper compared to this study). Upcoming investigations can test these speculations.

Some direct effects were also observed, such that greater childhood trauma significantly predicted trait anger expression, MDD, and PD severity in adulthood. These findings offer more support for biopsychosocial theories of childhood maltreatment. A plausible explanation may be that individuals with early maltreatment exposure who are also predisposed to anxiety sensitivity (fear of anxious arousal-related sensations) and negative emotional reactivity may be at greater risk of developing later PD and MDD symptoms. Stated differently, such individuals are likely to be bothered by both the maltreatment event(s) and their own fearful or anxious reactions to them. For example, anxiety sensitivity positively predicted posttraumatic stress symptom severity 6 to 12 months later in adults who were victims of physical abuse from familial violence (Marshall et al., 2010). These speculations warrant further investigation.

This study has several limitations. First, since childhood trauma data was collected retrospectively, CTQ-anger associations were cross-sectional. Relatedly, given the large age range (24 to 74 years herein), there was large heterogeneity with regard to the duration between occurrences of the traumatic events and their retrospective report in adulthood. However, we determined that the pattern of results remained the same after age was controlled for as a covariate in all of the models. Second, since we neither assessed chronicity nor first onset of disorders in this study, they may be confounding variables. Third, the relations between anger and common psychiatric disorders are complex and bidirectional, and thus merit more attention. Moreover, we cannot determine causality from our prospective study, as causality can only be established using experimental designs (Shadish et al., 2002). Also, unmeasured third variables, such as genetics (e.g., presence of specific gene markers or genetic variants), self-blame, and thought control strategies may have contributed to our findings (Dorresteyn et al., 2019; Lekman et al., 2008; Meiser-Stedman et al., 2014; Mick et al., 2014). Further, CIDI-SF scales used in this study were based on DSM–III–R

criteria. Therefore, replication with the DSM-5 is necessary to ensure that its revisions (e.g., 4 out of 7 depressive symptoms in DSM-III-R vs. 5 out of 9 symptoms in DSM-5 for MDD) will uphold the current findings. Another limitation includes the lack of ethnic and economic diversity in the present study sample. Future research could recruit a more inclusive sample to maximize generalizability. Nonetheless, study strengths include the use of a latent variable approach which minimizes measurement error, and large sample size that maximizes power.

The clinical applications of our basic science study warrant attention. To facilitate treatment, clinicians should encourage clients who have experienced childhood maltreatment to engage in acceptance, compassion, or forgiveness for caregivers who have wronged them, as this is a first step toward reducing anger rumination and aggression. For example, a 6-week forgiveness intervention was found to significantly reduce dispositional anger expression in adults who experienced distressing past interpersonal conflicts (Harris et al., 2006). Clinicians should also train clients to modulate their anger through strategies that encourage adaptive, goal-oriented behavior that reduce the risk of an adulthood onset of anxiety, depression and AUDs. Such constructive anger regulation strategies have been shown to improve psychopathology. For example, mindfulness coping techniques longitudinally reduced depression symptoms in individuals with dysregulated anger (Cassello-Robbins et al., 2020). Moreover, our study highlights the importance of continuing work on trauma-informed care in assessment, diagnosis, and treatment in various inpatient and outpatient treatment settings (Bendall et al.). In summary, shifting the priorities of trauma-focused therapies to managing dispositional anger expression may improve mental health outcomes.

#### Author contributions

My research team, Ms. Zainal, Dr. Newman, and I conceived of the presented idea, developed the theory, and performed the statistical analyses. Ms. Zainal verified the analytical methods and outputs. Dr. Newman encouraged Ms. Zainal and I to refine the analyses and supervised the findings of this work. We take full responsibility for the data, the accuracy of analyses and interpretation, as well as conduct of the research. All authors have (1) made substantial contributions to analysis and interpretation of the study and its findings; (2) drafted and revised the article for intellectual content; and (3) gave their final approval of the version submitted. The manuscript has been read and approved by all three authors.

#### Statement of ethics

This study was conducted in compliance with the American Psychological Association (APA) ethical standards in the treatment of human participants and approved by the institutional review board (IRB). Further, this research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Informed consent was obtained from participants as per IRB requirements at Harvard University, Georgetown University, University of California at Los Angeles, and University of Wisconsin. Since this study used a publicly available dataset, it was exempt from IRB approval.

#### Declaration of Competing Interest

My research team, Ms. Zainal, Dr. Newman, and I, do not have any conflicts of interest in regard to the authorship, research, and/or publication of this article, and have enclosed the manuscript and tables in Microsoft word format. All authors are affiliated with PSU.

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#### Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jad.2021.03.086.

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