Journal of Affective Disorders 358 (2024) 138–149

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Research paper

Daily stress reactivity and risk appraisal mediates childhood parental abuse predicting adulthood psychopathology severity: An 18-year longitudinal mediation analysis

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ARTICLE INFO

Keywords:
Childhood parental abuse
Anxiety disorders
Major depressive disorder
Substance use disorders
Mediation analyses
Longitudinal structural equation modeling

ABSTRACT

Identifying mechanisms of childhood abuse-adulthood psychopathology relations could facilitate preventive efforts, but most prior studies used cross-sectional or two-wave designs and did not test the effects of childhood maternal and paternal abuse separately. Our 18-year three-wave study thus determined if Wave 2 daily stress reactivity and risk appraisal severity mediated Wave 1 retrospectively-reported childhood maternal and paternal abuse on Wave 3 generalized anxiety disorder (GAD), major depressive disorder (MDD), panic disorder (PD), alcohol (AUD), and substance use disorder (SUD) self-rated symptom severity. Longitudinal structural equation modeling was employed, adjusting for Wave 1 psychopathology severity. Higher childhood maternal and paternal abuse consistently predicted greater future daily stress reactivity and risk appraisal, and these mediators subsequently predicted increased GAD, MDD, and PD, but not AUD and SUD severity. Daily stress reactivity and risk appraisal consistently mediated the pathways between childhood maternal and paternal abuse predicting heightened adulthood GAD, MDD, and PD (Cohen's $d = 0.333$–0.888) but not AUD and SUD severity. Mediation effect sizes were stronger for childhood maternal (24.5–83.0%) than paternal (19.5–56.0%) abuse as the predictor. The latent interaction between Wave 1 childhood maternal and paternal abuse did not moderate the effect of Wave 1 maternal or paternal abuse on any Wave 3 adulthood psychopathology severity through Wave 2 daily stress reactivity and risk appraisal. Our research emphasizes the urgent requirement for continuous evaluation and intervention initiatives in trauma-informed care, both in inpatient and outpatient treatment settings.

1. Introduction

Childhood abuse (i.e., maltreatment of children and adolescents) represents a severe societal concern, impacting over a third of the population worldwide (Stoltenborgh et al., 2014). Annual incidences are approaching nearly one million children (Sedlak et al., 2010; U.S. Department of Health and Human Services, 2022). Child maltreatment's total lifelong economic impact is now estimated at $2 trillion (Peterson et al., 2018). Childhood maltreatment is typically perpetrated by someone responsible for the child's well-being, with approximately 80 % of cases involving mothers or fathers as the perpetrators (Hughes et al., 2017). Child maltreatment is associated with lifelong adverse biopsychosocial consequences (Chapman et al., 2004; Cicchetti and Handley, 2019). Childhood parental abuse events are additionally linked to increased odds of detrimental impacts on educational attainment and career prospects over long durations (Gilbert et al., 2009; Henkhaus, 2022). Numerous clinicians and scientists have thus long acknowledged the importance of early-life nurture in adulthood mental health since such efforts could identify prevention and treatment targets.

Recent decades of research have substantiated the validity of this proposition. Across 23 longitudinal studies, there was some meta-analytic indication of a dose-response association, with individuals exposed to multiple forms of childhood trauma having over threefold higher odds of developing a psychopathology (McKay et al., 2021). Another meta-analysis of 23 primarily cross-sectional studies showed

Abbreviations: AUD, alcohol use disorder; GAD, generalized anxiety disorder; MDD, major depressive disorder; PD, panic disorder; SUD, substance use disorder.

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https://doi.org/10.1016/j.jad.2024.04.068
Received 8 January 2024; Received in revised form 7 April 2024; Accepted 16 April 2024
Available online 23 April 2024

that the population-attributable fractions (i.e., proportion of cases of a specific health outcome in a population) linked to adverse childhood experiences for increased anxiety, depression, alcohol use disorder (AUD), and substance use disorder (SUD) severity varied between 27.5% and 41.1% across Europe and North America (Bellis et al., 2019). Moreover, epidemiological reports evidenced that childhood parental abuse conferred higher likelihood of developing future increased major depressive disorder (MDD), generalized anxiety disorder (GAD), and panic disorder (PD) severity (Hughes et al., 2017; Scott et al., 2010). Another meta-analysis found that individuals who experienced childhood traumatic events were at an elevated risk of future suicide attempts than the general population (Zatti et al., 2017). Collectively, understanding the mechanisms via which heightened childhood parental abuse might confer increased adulthood psychopathology risk is essential.

Daily stress reactivity and appraisal might be viable mechanisms through which childhood parental abuse predicts increased adulthood psychopathology severity. Myriad biopsychosocial theories postulate that childhood maltreatment might precede stress reactivity, suboptimal stress risk appraisal, and future adulthood psychopathology severity. Biologically, childhood abuse could directly disrupt development within neurobiological stress systems across time (Hakamata et al., 2022). Increased childhood maltreatment could also adversely affect the hypothalamic-pituitary-adrenal (HPA) axis, the primary neuroendocrine system responsible for the stress response (Koss and Gunnar, 2018), and the immune system (Danese and Baldwin, 2017). Psychologically, childhood maltreatment could diminish one's resilience to stress, making them more susceptible to anxious and depressive reactions with lower stress thresholds across extended periods (Shapero et al., 2014).

Ample empirical studies offered evidence consistent with these ideas. Childhood abuse predicted unhealthy thinking patterns that contributed to stress dysregulation, which thereby increased the risk of heightened psychopathology (Weissman et al., 2019). Heightened trait anger expression (internal and external; Win et al., 2021) and reduced self-acceptance (Sanghvi et al., 2023) mediated the relations between increased childhood maltreatment and adulthood depression, anxiety, and SUD severity across 9 to 18 years. Another prior ecological momentary assessment (EMA) study revealed a correlation between a higher frequency of maternal childhood abuse and a heightened perception of daily stressors' severity (Kong et al., 2019). This study further showed that adults who experienced maternal childhood abuse more frequently displayed heightened emotional reactivity in response to everyday stressors. Moreover, individuals with notably reduced positive affect during days with stressors had a higher risk of developing future MDD and anxiety disorders (Rackoff and Newman, 2020). On the whole, existing evidence suggests the plausibility of heightened daily stress reactivity and risk appraisal mediating the pathway of childhood parental abuse predicting increased adulthood psychopathology severity.

Previous research had limitations the current study aimed to overcome. First, most prior studies used cross-sectional or two-wave longitudinal designs. Optimal mediation approaches require three or more time points (Cole and Maxwell, 2003; Maxwell and Cole, 2007) to establish weak causal inference in prospective-observational studies (Blackwell and Glynn, 2018). Second, most prior studies did not separately examine the effects of maternal and paternal abuse. We explored paternal and maternal abuse as distinct predictors, acknowledging that each parent may possess unique caregiving styles and roles within the family, potentially yielding varying effects on a child's future mental health (Cox and Paley, 1997; Cui et al., 2018). Third, high levels of abuse by one parent might be buffered by non-abuse or affection by another, yet interactive effects have not been tested in prior research, which is a gap our study remedied.

Therefore, we focused on addressing a pivotal translational question: identifying targets for mitigating the impact of childhood parental abuse on adulthood psychopathology severity. First, we tested the prediction that increased childhood maternal abuse (Hypothesis 1A) and paternal abuse (Hypothesis 1B) would predict future increased daily stress reactivity, thereby predicting higher GAD, MDD, PD, AUD, and SUD severity. Second, we evaluated the prediction that increased childhood maternal abuse (Hypothesis 2A) and paternal abuse (Hypothesis 2B) would predict future elevated daily stress risk appraisal severity, thereby predicting higher GAD, MDD, PD, AUD, and SUD severity.

2. Method

2.1. Participants

The inclusion criteria comprised Midlife Development in the United States (MIDUS) participants with relevant data at all three time points: 1995–1996 (Wave 1; W1), 2004–2005 (Wave 2; W2), and 2012–2013 (Wave 3; W3; Brim et al., 2020; Ryff et al., 2019; Ryff et al., 2017). Although the sample size started with 7108 at W1 and 4963 at W2, only 3294 completed the MIDUS study. The current study was a secondary data analysis of a publicly available data set, exempting it from Institutional Review Board approval. At W1, eligible participants had mean age of 46.65 years ($SD = 10.35$, range 20–86), with 54.95% female and 42% having a college education. The racial distribution consisted of 89.7% Caucasian, 3.3 % African American, and 7.0% Asian, Native American, Pacific Islander, other ethnicities or declined to disclose.

Table 1
Descriptive variables of clinical and sociodemographic variables (N = 3294).

Continuous variables	*M*	(*SD*)
W1 Age (years)	46.65	(10.35)
W2 Age (years)	54.70	(10.30)
W3 Age (years)	62.51	(9.35)
Disorder severity		
W1 GAD severity	0.136	(0.859)
W3 GAD severity	0.131	(0.921)
W1 MDD severity	0.692	(1.822)
W3 MDD severity	0.601	(1.706)
W1 PD severity	0.376	(1.087)
W3 PD severity	0.273	(0.921)
W1 AUD severity	0.073	(0.434)
W3 AUD severity	0.039	(0.318)
W1 SUD severity	0.211	(0.651)
W3 SUD severity	0.713	(0.772)
W1 Maternal emotional abuse	1.774	(0.909)
W1 Paternal emotional abuse	2.106	(1.243)
W1 Maternal physical abuse	1.669	(0.815)
W1 Paternal physical abuse	1.711	(0.854)
W1 Maternal severe physical abuse	1.212	(0.571)
W1 Paternal severe physical abuse	1.286	(0.661)

Binary variables	*n*	(%)
Sex-at-birth		
Men	1484	(45.05)
Women	1810	(54.95)
Race		
Missing data	129	(3.92)
Multiracial	28	(0.85)
White	2956	(89.74)
African American	111	(3.37)
Native American	11	(0.33)
Asian	16	(0.49)
Other	43	(1.31)
W1 GAD diagnosis	76	(2.31)
W3 GAD diagnosis	64	(1.94)
W1 MDD diagnosis	385	(11.69)
W3 MDD diagnosis	327	(9.93)
W1 PD diagnosis	222	(6.74)
W3 PD diagnosis	171	(5.19)

W1, wave 1; W2, wave 2; W3, wave 3; GAD, generalized anxiety disorder; MDD, major depressive disorder; PD, panic disorder; AUD, alcohol use disorder; SUD, substance use disorder.

Table 1 presents more details on the demographic and clinical attributes.

2.2. Procedures

This study centered on 3294 participants who underwent telephone interviews and/or self-reports, measuring the severity of psychopathology symptoms at W1 and W3. Participants also filled out assessments measuring the frequency of childhood emotional, physical, and severe physical abuse experiences at W1 and daily diary reports on stressful events, reactivity, and risk appraisals for eight days at W2. The following subsections detail each measure.

2.3. Measures

2.3.1. W1 Childhood abuse

Incidents of abuse were retrospectively self-reported using the Revised Conflict Tactics Scale (CTS2; Straus et al., 1996). The CTS2 assessed childhood abuse across three categories: emotional abuse, physical abuse, and severe physical abuse. Respondents rated their experiences using a 4-point Likert scale (1 = *Never* to 4 = *Often*). Each category was independently rated for abuse events involving the individual's maternal or primary female caregiver and paternal or primary male caregiver. Regarding emotional abuse (6 items), respondents reported whether the perpetrators exhibited the following behaviors: "did or said something to spite you; insulted you or swore at you; sulked or refused to talk to you; smashed or kicked something in anger; stomped out of the room; and threatened to hit you." Concerning physical abuse (3 items), respondents indicated if the perpetrators engaged in the following actions: "threw something at you; slapped you; pushed, grabbed, or shoved you." Finally, respondents reported any instances of severe physical abuse (5 items) when the perpetrators exhibited the following actions: "beat you up; burned or scalded you; choked you; hit or tried to hit you with something; kicked, bit, or hit you with a fist." CTS2 scores demonstrated satisfactory internal consistency herein, as Macdonald's omega (ω) values were .711 and .709 for maternal and paternal abuse, respectively. We employed Macdonald's ω (Dunn et al., 2014) to assess reliability, recognizing the shortcomings of Cronbach's alpha (α), which assumes homogeneous variances in true scores, perfect associations, and non-correlated error variances among items. CTS2 scores also exhibited robust validity and reliability when assessed across diverse samples (Chapman and Gillespie, 2018).

2.3.2. W2 Daily stress reactivity

The assessment of negative affect (NA) in response to any stressor(s) utilized a scale specifically designed for the MIDUS National Survey of Daily Experiences (NSDE; Almeida et al., 2002; Kong et al., 2019; Wardecker et al., 2022). Participants used a 5-point Likert scale (0 = *none of the time* to 4 = *all of the time*) to respond to 14 items inquiring about their daily emotional experiences, framed as "How much of the time today did you feel…?" Responses were aggregated within each individual, with elevated scores indicating greater stress reactivity. The Daily Inventory of Stressful Experiences (DISE; Almeida et al., 2002) was employed to assess the exposure, frequency, and nature of daily stressors. Participants were queried about the occurrence of a particular type of negative event in the past 24 h (0 = *no* or 1 = *yes*). These events encompassed arguments, avoided arguments, workloads, domestic pressures, and network stressors, which referred to stress-inducing situations involving close friends or relatives that affected the respondent.

2.3.3. W2 Daily stress risk appraisal

Respondents also recorded information regarding their perceived level of risk associated with the stressor affecting various aspects of their personal lives on a 4-point Likert scale (1 = *not at all at risk* to 4 = *at risk a lot*; Kong et al., 2019). This approach extended the domains of primary appraisal as outlined by Lazarus (1999). The risk areas comprised (a) disruptions to daily routines, (b) external perceptions of the respondent, (c) financial well-being, (d) future plans, (e) personal health and safety, and (f) self-concept (Almeida et al., 2005). The daily diary scale scores in the present study showed good internal consistency scores (between-person ω = .875, within-person ω = .884). Higher levels of risk indicated appraising stressors as more threatening and less controllable.

2.3.4. W1 and W3 Psychopathology severity

The symptom severity scores for GAD, MDD, PD, AUD, and SUD were determined according to the Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition (DSM-III-R; American Psychiatric Association, 1987) criteria, utilizing the World Health Organization's Composite International Diagnostic Interview-Short Form (CIDI-SF; Kessler et al., 1998). Continuous scales were employed to evaluate GAD, MDD, and PD symptom severity in the past 12 months. GAD severity was reported on a scale of 0 (*lowest worry*) to 10 (*highest worry*). It assessed symptoms of GAD related to excessive and uncontrollable worry (10 items): difficulty focusing, feeling keyed up or on edge, irritability, low energy, memory problems, muscle soreness or fatigue, restlessness, and sleep difficulties (both falling and staying asleep). Responses for each item were coded on a scale from 0 (*never*) to 1 (*worries for more days than not or most days*; ω = .890 and .900 at W1 and W3, respectively). MDD severity was assessed on a scale of 0 (*lowest depression*) to 7 (*highest depression*). The measurement focused on MDD symptoms associated with depressed mood and anhedonia (7 items): appetite changes, difficulty concentrating, fatigue, loss of interest in most activities, low self-esteem, sleep disturbances, and thoughts of death (ω = .960 and .970). PD severity was measured from 0 (*lowest panic score*) to 10 (*highest panic score*). It assessed symptoms experienced during panic attacks or spells (10 items): presence of spell/attack when frightened, at least one attack in the past year, spell/attack for no reason, attack occurred when not in danger or during the center of attention, chest/stomach pain, heart-pounding, hot flashes/chills, tightness/discomfort, trembling/shaking, and a sense of unreality (ω = .900 and .890).

Additionally, AUD severity was assessed using the Alcoholism Screening Test (AST; Selzer, 1971) on a scale of 0 (*lowest severity*) to 5 (*highest severity*). Participants reported alcohol-related issues, including a strong urge to consume alcohol, emotional problems from using alcohol, excessive drinking time, increased tolerance to its effects, and emotional issues stemming from using alcohol. Each item's responses were coded as 0 (*no*) or 1 (*yes*) (ω = 0.760 and 0.790). SUD severity was assessed on a scale of 0 (*lowest severity*) to 7 (*highest severity*). Participants disclosed problems linked to using substances (cocaine/crack, heroin, inhalants, LSD/other hallucinogen, marijuana/hashish, nerve pills, prescription painkillers, sedatives, stimulants), encompassing using larger amounts than intended, adverse effects of using substances during school/work, use increased odds to get hurt, use contributed to emotional issues, strong desire to use, substance use took excessive time, and increased tolerance to its effects (Turiano et al., 2012). Each item's responses on the SUD scales were coded as 0 (*no*) or 1 (*yes*) (ω = .890 and .820).

2.4. Data preprocessing

As a preprocessing step, the W2 daily diary indices of stress reactivity/appraisal were averaged across all eight days, aggregated across participants, and merged by participant's unique identifier with the W1 and W3 panel data that comprised symptom severity using the *dplyr R* package (Wickham et al., 2023). Next, using the *mice R* package (van Buuren and Groothuis-Oudshoorn, 2011), missing data (present in 13.1 % of the total data set) was managed using multiple imputation; the gold standard approach for our data set was assumed to be missing at random (Lee and Shi, 2021). Before performing the structural equation modeling (SEM) mediation analyses, the data underwent screening to assess univariate and multivariate normality, outliers, and multicollinearity. Mahalanobis distance analysis revealed the absence of outliers. Analysis of skewness and kurtosis coefficients indicated no significant breach of

univariate and multivariate normality assumptions. No signs of multicollinearity were observed (all variance inflation factor values were <1.1). Last, based on best practices (Guenole and Brown, 2014), we determined that the psychopathology severity scales showed adequate levels of measurement invariance across W1 and W3 (online supplemental materials (OSM) Tables S1 to S5).

2.5. Data analyses

To test the fit of the SEM model using the *lavaan R* package (Rosseel, 2012), we employed the chi-square (χ^2) statistic (Hu and Bentler, 1999), model degrees of freedom, and its related degrees of freedom and probability (*p*) values (Kline, 2015). CFI values within the range of 0.9 to 1.0 signified a satisfactory fit (Bentler, 1990). Regarding the RMSEA, values under 0.10 denoted an acceptable fit (Steiger, 1990). SEM mediation analyses were carried out using the product-of-coefficients approach of indirect effect for the coefficients of W1 childhood maternal/paternal abuse predicting W2 daily stress reactivity/appraisal (*a* path) and W2 daily stress reactivity/appraisal predicting W3 disorder symptom severity (*b* path). We presented unstandardized regression coefficients (β) and *p*-values and applied bootstrapping with 1000 resampling iterations with robust maximum likelihood estimators to obtain standard errors (SE; refer to Figs. 1 and 2 for generic example schematic diagrams; Cheung and Lau, 2008).

The mediation effect size quantifies the fraction of the indirect effect (a * b) to the total effect (c = a * b + c'; Preacher and Kelley, 2011). It was indicated as the percentage of variance in how much the focal mediator explained the distinctive associations between childhood maltreatment from maternal or paternal figures and adulthood psychopathology severity. In all models, W1 outcome variables were adjusted, such as including W1 MDD severity when predicting W3 MDD severity. We refrained from adjusting for W1 daily stress reactivity/risk appraisal, as established principles of causal inference methodologies caution that such control at baseline could potentially introduce bias into estimating total effects by inadvertently obstructing a portion of the causal influence through the mediator (D'Onofrio et al., 2020; Rosenbaum, 1984). Further, we conducted a moderated mediation SEM analysis to test how W1 maternal and paternal abuse might interact to predict W3 disorder severity through W2 daily stress reactivity/appraisal (Fig. 2). The *indprod* function of *semTools* (Jorgensen et al., 2022) was used to create a latent interaction term between W1 maternal and paternal abuse. We calculated Cohen's *d* using the formula $d = 2t /$

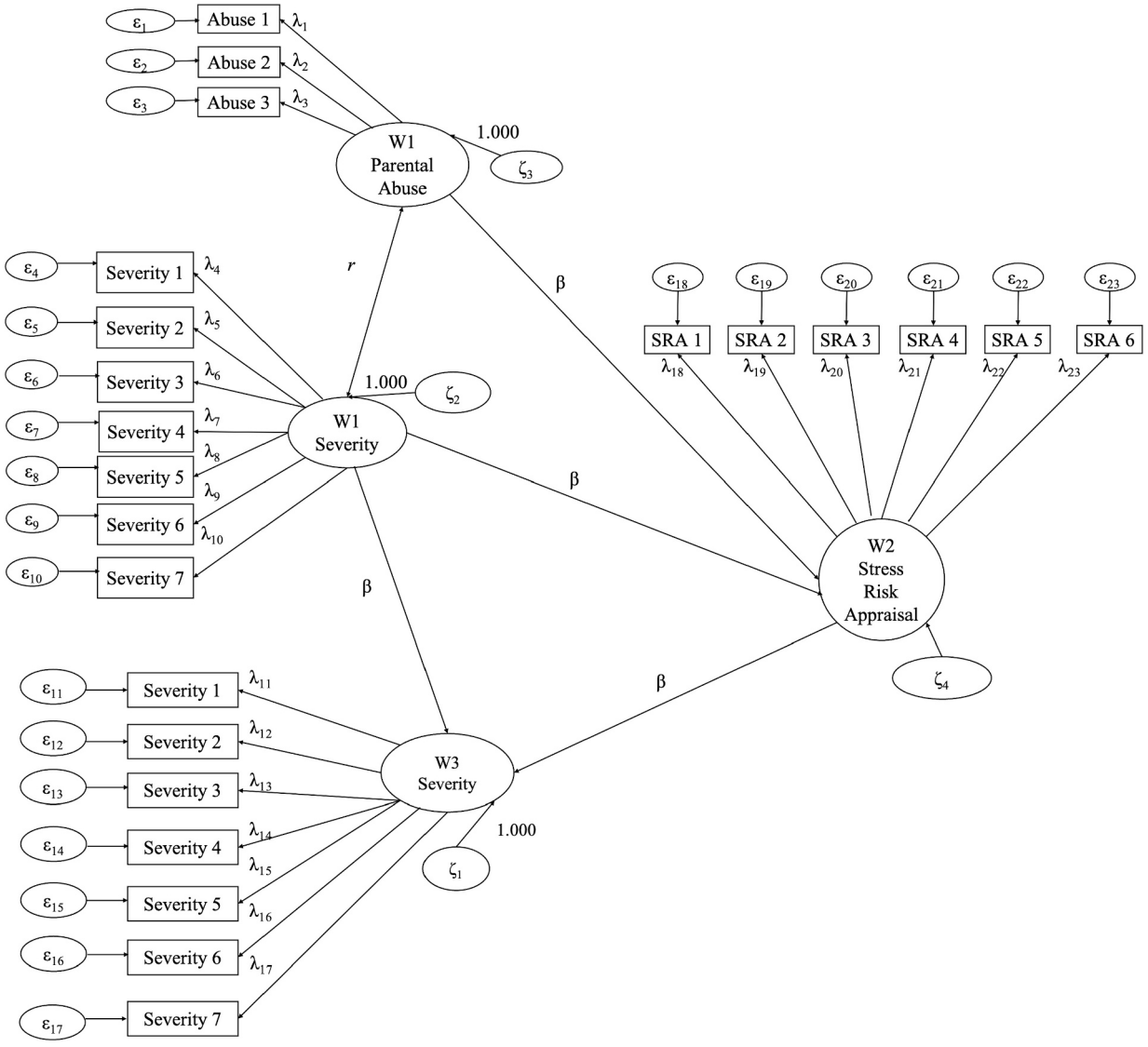

Fig. 1. Generic example diagram for non-moderated mediation analysis.
ε, residual error variance; λ, latent factor loading; ζ, residual latent variance; β, unstandardized regression estimate; *r*, latent correlation; W1, wave 1; W2, wave 2; W3, wave 3.

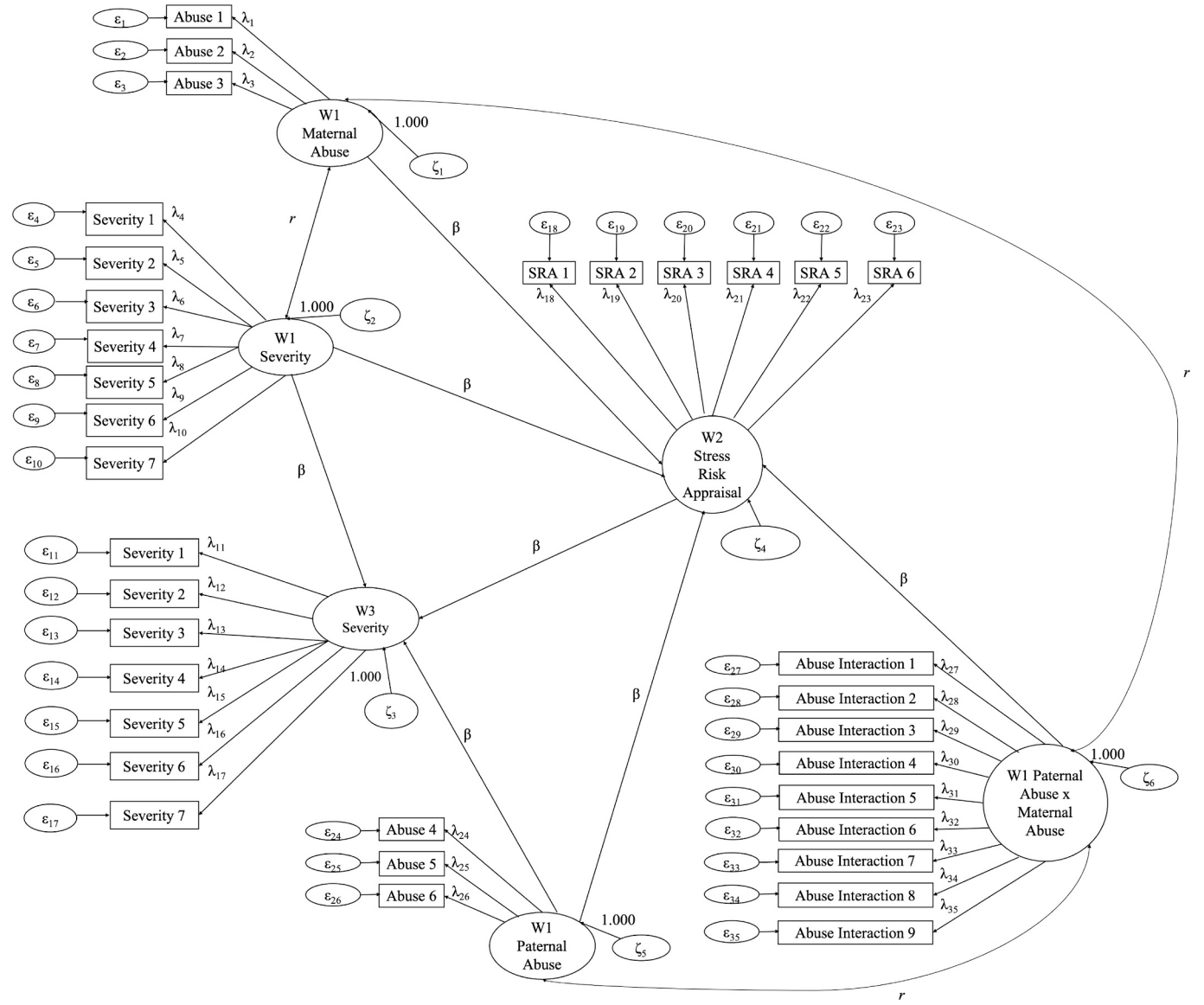

Fig. 2. Generic example diagram for moderated mediation analysis.
ε, residual error variance; λ, latent factor loading; ζ, residual latent variance; β, unstandardized regression estimate; *r*, latent correlation; W1, wave 1; W2, wave 2; W3, wave 3.

$\sqrt{(df)}$, such that t was the t-value of the parameter estimate and df referred to the model degrees of freedom (Lakens, 2013). Further, given the large sample size and examination of multiple outcomes, we only regarded p-values of <.01 as statistically significant as an alpha correction method (Simes, 1986).

3. Results

3.1. W1 childhood maternal abuse predicting W3 disorder severity via W2 daily stress reactivity

All mediation models within this set of analyses showed acceptable-to-good fit with various adulthood psychopathology severity outcomes (Table 2). Higher childhood maternal abuse significantly predicted increased daily stress reactivity for all examined outcomes: GAD (d = 0.847), MDD (d = 1.230), PD (d = 0.925), AUD (d = 1.613), and SUD (d = 1.032; all p values < .001). Except for AUD (d = 0.606, p = .018) and SUD (d = 0.359, p = .030), greater daily stress reactivity, in turn, significantly predicted stronger psychopathology severity for other examined outcomes: GAD (d = 0.699), MDD (d = 1.302), and PD (d = 0.723; all p values < .001). Except for AUD (d = 0.573, p = .025) and SUD (d = 0.341, p = .039), the indirect effects of higher daily stress reactivity mediating the path between increased childhood maternal abuse predicting elevated adulthood psychopathology severity were also significant for other examined outcomes: GAD (d = 0.528), MDD (d = 0.888), and PD (d = 0.569; all p values < .001). Increased daily stress reactivity accounted for 83.0%, 24.5%, and 33.0% of more childhood maternal abuse predicting increased adulthood psychopathology severity for W3 GAD, MDD, and PD, respectively. Hypothesis 1A was, thus, partially supported.

3.2. W1 childhood paternal abuse predicting W3 disorder severity via W2 daily stress reactivity

All mediation models within this set of analyses fit well with various adulthood psychopathology severity outcomes (Table 3). Higher childhood paternal abuse significantly predicted increased daily stress reactivity for all examined outcomes: GAD (d = 0.753), MDD (d = 0.717), PD

Table 2
Wave 1 (W1) Childhood maternal abuse predicting Wave 3 (W3) mental disorder severity via Wave 2 (W2) daily stress reactivity.

	GAD		MDD		Panic disorder		AUD		SUD	
	β	(SE)	β	(SE)	β	(SE)	β	(SE)	β	(SE)
Factor loadings										
W1 Emotional abuse	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W1 Physical abuse	0.848*	(0.057)	0.846*	(0.076)	0.762*	(0.053)	0.887*	(0.033)	0.859*	(0.039)
W1 Severe physical abuse	0.457*	(0.035)	0.579*	(0.059)	0.421*	(0.034)	0.481*	(0.025)	0.475*	(0.027)
W2 Daily SR	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W2 Daily NA	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W3 Symptom item 1	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W3 Symptom item 2	1.418*	(0.180)	0.409*	(0.000)	−0.005	(0.006)	0.595*	(0.105)	1.038*	(0.082)
W3 Symptom item 3	2.003*	(0.240)	0.794*	(0.076)	0.168*	(0.025)	0.713*	(0.094)	0.125*	(0.023)
W3 Symptom item 4	1.857*	(0.239)	0.956*	(0.059)	0.342*	(0.037)	0.790*	(0.113)	0.067*	(0.017)
W3 Symptom item 5	1.554*	(0.215)	0.744*	(0.000)	1.066*	(0.047)	–	–	0.032*	(0.012)
W3 Symptom item 6	1.295*	(0.179)	0.769*	(0.000)	0.772*	(0.046)	–	–	0.050*	(0.015)
W3 Symptom item 7	2.246*	(0.293)	1.325*	(0.000)	0.781*	(0.047)	–	–	0.069*	(0.017)
W3 Symptom item 8	1.873*	(0.257)	–	–	0.539*	(0.040)	–	–	–	–
W3 Symptom item 9	1.640*	(0.219)	–	–	0.722*	(0.048)	–	–	–	–
W3 Symptom item 10	1.571*	(0.212)	–	–	0.368*	(0.037)	–	–	–	–
W1 Symptom item 1	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W1 Symptom item 2	1.390*	(0.149)	0.426*	(0.017)	0.318	(0.040)	0.670*	(0.092)	1.063*	(0.120)
W1 Symptom item 3	1.360*	(0.157)	0.799*	(0.044)	0.231*	(0.027)	0.532*	(0.082)	0.597*	(0.102)
W1 Symptom item 4	1.204*	(0.144)	0.978*	(0.035)	0.375*	(0.033)	0.581*	(0.080)	0.351*	(0.075)
W1 Symptom item 5	1.065*	(0.133)	0.698*	(0.046)	1.092*	(0.042)	–	–	0.166*	(0.047)
W1 Symptom item 6	0.984*	(0.125)	0.693*	(0.044)	0.850*	(0.041)	–	–	0.155*	(0.047)
W1 Symptom item 7	1.322*	(0.163)	1.619*	(0.080)	0.727*	(0.041)	–	–	0.267*	(0.066)
W1 Symptom item 8	1.435*	(0.166)	–	–	0.707*	(0.040)	–	–	–	–
W1 Symptom item 9	1.183*	(0.136)	–	–	0.818*	(0.041)	–	–	–	–
W1 Symptom item 10	1.137*	(0.134)	–	–	0.429*	(0.033)	–	–	–	–
Regression estimates										
W1 Abuse → W2 Daily SRC	0.242*	(0.035)	0.340*	(0.064)	0.244*	(0.032)	0.194*	(0.031)	0.193*	(0.031)
W2 Daily SRC → W3 Symptoms	0.013*	(0.002)	0.016*	(0.003)	0.031*	(0.005)	0.007	(0.003)	0.011	(0.005)
W1 Abuse → W3 Symptoms	0.001	(0.002)	0.028*	(0.009)	0.015	(0.008)	−0.003	(0.004)	−0.006	(0.008)
W1 Symptoms → W3 Symptoms	0.280*	(0.050)	0.194*	(0.041)	0.304*	(0.029)	0.306*	(0.053)	0.837*	(0.130)
Indirect effect	0.003*	(0.001)	0.006*	(0.001)	0.008*	(0.002)	0.001	(0.001)	0.002	(0.001)
Total effect	0.004	(0.002)	0.034*	(0.009)	0.023	(0.007)	−0.002	(0.004)	−0.004	(0.008)

Note. GAD, generalized anxiety disorder; MDD, major depressive disorder; AUD, alcohol use disorder; SUD, substance use disorder; SR, stress reactivity; NA, negative affect; SRC, stress reactivity latent composite score; β, unstandardized regression or factor loading estimate; SE, standard error of β. For GAD severity, items (in order of presentation) were worry-linked feelings of being keyed up/restless, trouble falling asleep, difficulty staying asleep, trouble concentrating, trouble remembering, low on energy, tired quickly, muscle aches, and interference with life. For MDD severity, items were depressed mood linked to symptoms of anhedonia, low energy, loss/increased appetite, difficulty falling asleep, trouble concentrating, feeling worthless, and thinking a lot about death. For PD severity, items were having panic spells/attacks, spells/attacks occurred for no reason, the number of panic attacks, attacks occurred during danger/being the center of attention, heart pounds during attacks, chest pain/tightness, sweating, trembling/shaking, hot flashes/chills, and feelings of unreality. For AUD severity, items were a strong urge to consume alcohol, emotional problems from using alcohol, excessive drinking time, increased tolerance to its effects, and emotional issues stemming from using alcohol. For SUD severity, using larger amounts than intended, adverse effects of using substances during school/work, use increased odds of getting hurt, use contributed to emotional issues, a strong desire to use, substance use took excessive time, and increased tolerance to its effects. All mediation models within this set of analyses showed acceptable-to-good fit with various adulthood psychopathology severity outcomes: GAD ($\chi^2(271) = 751.133$, $p < .001$, CFI = .962, RMSEA = .023), MDD ($\chi^2(148) = 733.355$, $p < .001$, CFI = .975, RMSEA = .033), PD ($\chi^2(270) = 1397.298$, $p < .001$, CFI = .921, RMSEA = .037), AUD ($\chi^2(61) = 186.941$, $p < .001$, CFI = .960, RMSEA = .024), and SUD ($\chi^2(147) = 326.828$, $p < .001$, CFI = .910, RMSEA = .026).

* Statistically significant at $p < .01$ (two-tailed).

($d = 0.782$), AUD ($d = 1.229$), and SUD ($d = 0.795$; all p values $< .001$). Except for AUD ($d = 0.607$, $p = .018$) and SUD ($d = 0.392$, $p = .017$), greater daily stress reactivity, in turn, significantly predicted stronger psychopathology severity for other examined outcomes: GAD ($d = 0.705$), MDD ($d = 0.979$), and PD ($d = 0.739$; all p values $< .001$). Except for AUD ($d = 0.546$, $p = .033$) and SUD ($d = 0.354$, $p = .032$), the indirect effects of daily stress reactivity mediating the path between childhood paternal abuse predicting psychopathology severity were also significant for other examined outcomes: GAD ($d = 0.510$), MDD ($d = 0.595$), and PD ($d = 0.542$; all p values $< .001$). Elevated daily stress reactivity accounted for 56.0%, 19.5%, and 44.9% of greater childhood paternal abuse predicting higher adulthood psychopathology severity for W3 GAD, MDD, and PD, respectively. Hypothesis 1B was, therefore, partially supported.

3.3. W1 childhood maternal abuse predicting W3 disorder severity via W2 daily stress risk appraisal

All mediation models within this set of analyses fit well with various adulthood psychopathology severity outcomes (Table 4). Higher childhood maternal abuse significantly predicted increased daily stress risk appraisal for all examined outcomes: GAD ($d = 0.539$), MDD ($d = 0.724$), PD ($d = 0.530$), AUD ($d = 0.841$), and SUD ($d = 0.624$; all p values $< .001$). Except for AUD ($d = 0.358$, $p = .056$) and SUD ($d = 0.362$, $p = .016$), greater daily stress risk appraisal, in turn, significantly predicted stronger psychopathology severity for other examined outcomes: GAD ($d = 0.420$), MDD ($d = 0.667$), PD ($d = 0.572$; all p values $< .001$). Except for AUD ($d = 0.328$, $p = .080$) and SUD ($d = 0.321$, $p = .016$), the indirect effects of higher daily stress risk appraisal mediating the path between increased childhood maternal abuse predicting stronger psychopathology severity were also significant for other examined outcomes: GAD ($d = 0.347$), MDD ($d = 0.517$), and PD ($d = 0.434$; all p values $< .001$). Increased daily stress risk appraisal accounted for 72.9%, 25.2%, and 47.5% of the childhood maternal abuse predicting adulthood psychopathology severity for W3 GAD, MDD, and PD, respectively. Hypothesis 2A was, hence, partially supported.

Table 3
Wave 1 (W1) Childhood paternal abuse predicting Wave 3 (W3) mental disorder severity via Wave 2 (W2) daily stress reactivity.

	GAD		MDD		Panic disorder		AUD		SUD	
	β	(SE)	β	(SE)	β	(SE)	β	(SE)	β	(SE)
Factor loadings										
W1 Emotional abuse	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W1 Physical abuse	0.693*	(0.063)	0.912*	(0.075)	0.647*	(0.065)	1.076*	(0.039)	1.083*	(0.041)
W1 Severe physical abuse	0.424*	(0.045)	0.643*	(0.063)	0.389*	(0.043)	0.649*	(0.031)	0.665*	(0.033)
W2 Daily SR	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W2 Daily NA	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W3 Symptom item 1	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W3 Symptom item 2	1.396*	(0.174)	0.402*	(0.022)	0.326*	(0.045)	0.600*	(0.106)	1.037*	(0.085)
W3 Symptom item 3	1.979*	(0.233)	0.786*	(0.046)	0.175*	(0.025)	0.704*	(0.094)	0.124*	(0.023)
W3 Symptom item 4	1.821*	(0.231)	0.960*	(0.028)	0.341*	(0.036)	0.788*	(0.113)	0.065*	(0.017)
W3 Symptom item 5	1.552*	(0.211)	0.756*	(0.042)	1.083*	(0.048)	–	–	0.032*	(0.012)
W3 Symptom item 6	1.282*	(0.176)	0.756*	(0.045)	0.780*	(0.047)	–	–	0.049*	(0.014)
W3 Symptom item 7	2.221*	(0.288)	1.340*	(0.095)	0.788*	(0.048)	–	–	0.069*	(0.017)
W3 Symptom item 8	1.838*	(0.253)	–	–	0.538*	(0.040)	–	–	–	–
W3 Symptom item 9	1.626*	(0.216)	–	–	0.725*	(0.048)	–	–	–	–
W3 Symptom item 10	1.528*	(0.207)	–	–	0.358*	(0.037)	–	–	–	–
W1 Symptom item 1	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W1 Symptom item 2	1.317*	(0.142)	0.420*	(0.018)	0.003*	(0.010)	0.643*	(0.089)	1.056*	(0.125)
W1 Symptom item 3	1.331*	(0.151)	0.806*	(0.044)	0.220*	(0.027)	0.527*	(0.081)	0.643*	(0.106)
W1 Symptom item 4	1.168*	(0.138)	0.968*	(0.037)	0.374*	(0.033)	0.583*	(0.079)	0.348*	(0.075)
W1 Symptom item 5	1.036*	(0.127)	0.695*	(0.047)	1.063*	(0.042)	–	–	0.183*	(0.053)
W1 Symptom item 6	0.960*	(0.121)	0.671*	(0.045)	0.836*	(0.041)	–	–	0.159*	(0.048)
W1 Symptom item 7	1.282*	(0.155)	1.616*	(0.082)	0.721*	(0.041)	–	–	0.279*	(0.069)
W1 Symptom item 8	1.392*	(0.159)	–	–	0.711*	(0.040)	–	–	–	–
W1 Symptom item 9	1.137*	(0.128)	–	–	0.813*	(0.041)	–	–	–	–
W1 Symptom item 10	1.119*	(0.131)	–	–	0.446*	(0.034)	–	–	–	–
Regression estimates										
W1 Abuse → W2 Daily SRC	0.219*	(0.035)	0.196*	(0.045)	0.208*	(0.032)	0.167*	(0.035)	0.167*	(0.035)
W2 Daily SRC → W3 Symptoms	0.013*	(0.002)	0.020*	(0.003)	0.031*	(0.005)	0.007	(0.003)	0.012	(0.005)
W1 Abuse → W3 Symptoms	0.002	(0.003)	0.016	(0.008)	0.008	(0.007)	−0.004	(0.005)	−0.013	(0.009)
W1 Symptoms → W3 Symptoms	0.271*	(0.049)	0.209*	(0.041)	0.317*	(0.029)	0.304*	(0.053)	0.857*	(0.131)
Indirect effect	0.003*	(0.001)	0.004*	(0.001)	0.006*	(0.001)	0.001	(0.001)	0.002	(0.001)
Total effect	0.005	(0.003)	0.020*	(0.008)	0.014	(0.007)	−0.003	(0.005)	−0.011	(0.009)

Note. GAD, generalized anxiety disorder; MDD, major depressive disorder; AUD, alcohol use disorder; SUD, substance use disorder; SR, stress reactivity; NA, negative affect; SRC, latent composite score of stress reactivity; β, unstandardized regression or factor loading estimate; SE, standard error of β. For GAD severity, items (in order of presentation) were worry-linked feelings of being keyed up/restless, trouble falling asleep, difficulty staying asleep, trouble concentrating, trouble remembering, low on energy, tired quickly, muscle aches, and interference with life. For MDD severity, items were depressed mood linked to symptoms of anhedonia, low energy, loss/increased appetite, difficulty falling asleep, trouble concentrating, feeling worthless, and thinking a lot about death. For PD severity, items were having panic spells/attacks, spells/attacks occurred for no reason, the number of panic attacks, attacks occurred during danger/being the center of attention, heart pounds during attacks, chest pain/tightness, sweating, trembling/shaking, hot flashes/chills, and feelings of unreality. For AUD severity, items were a strong urge to consume alcohol, emotional problems from using alcohol, excessive drinking time, increased tolerance to its effects, and emotional issues stemming from using alcohol. For SUD severity, using larger amounts than intended, adverse effects of using substances during school/work, use increased odds of getting hurt, use contributed to emotional issues, a strong desire to use, substance use took excessive time, and increased tolerance to its effects. All mediation models within this set of analyses showed good fit with various adulthood psychopathology severity outcomes: GAD ($\chi^2(271) = 836.322$, $p < .001$, CFI = .941, RMSEA = .025), MDD ($\chi^2(148) = 777.398$, $p < .001$, CFI = .972, RMSEA = .035), PD ($\chi^2(270) = 1614.457$, $p < .001$, CFI = .907, RMSEA = .040), AUD ($\chi^2(61) = 165.952$, $p < .001$, CFI = .970, RMSEA = .021), and SUD ($\chi^2(147) = 326.137$, $p < .001$, CFI = .908, RMSEA = .026).

* Statistically significant at $p < .01$ (two-tailed).

3.4. W1 childhood paternal abuse predicting W3 disorder severity via W2 daily stress risk appraisal

All mediation models within this set of analyses fit well with various adulthood psychopathology severity outcomes (Table 5). Higher childhood paternal abuse significantly predicted increased daily stress risk appraisal for all examined outcomes: GAD (d = 0.464), MDD (d = 0.607), PD (d = 0.410), AUD (d = 0.601), and SUD (d = 0.452; all p values < .001). Except for AUD (d = 0.372, p = .047), greater daily stress risk appraisal, in turn, significantly predicted stronger psychopathology severity for other examined outcomes: GAD (d = 0.408), MDD (d = 0.672), PD (d = 0.587), and SUD (d = 0.383; all p values < .004). Except for AUD (d = 0.307, p = .101) and SUD (d = 0.305, p = .022), the indirect effects of heightened daily stress risk appraisal mediating the path between higher childhood paternal abuse predicting greater psychopathology severity were also significant for other examined outcomes: GAD (d = 0.333), MDD (d = 0.490), PD (d = 0.370; all p values < .001). Increased daily stress risk appraisal accounted for 43.6%, 24.6%, and 52.1% of higher childhood paternal abuse predicting stronger psychopathology severity for W3 GAD, MDD, and PD, respectively. Hypothesis 2B was, thus, partially supported.

3.5. Exploratory moderated mediation analyses

The latent interaction between W1 childhood maternal and paternal abuse did not significantly moderate the pathways of maternal or paternal abuse predicting any W3 disorder severity through both W2 daily stress reactivity (Tables S6) and stress risk appraisal (Tables S7). In addition, sensitivity analyses were conducted by adding baseline age to all examined models. All patterns of findings remained similar even after adjusting for baseline age.

4. Discussion

Partially supporting our hypotheses, daily stress reactivity and risk appraisal mediated 18-year longitudinal associations between childhood maternal and paternal abuse predicting adulthood GAD, MDD, and PD, but not AUD and SUD symptom severity. Our study extended other

Table 4
Wave 1 (W1) Childhood maternal abuse predicting Wave 3 (W3) mental disorder severity via Wave 2 (W2) daily stress risk appraisal.

	GAD		MDD		Panic disorder		AUD		SUD	
	β	(SE)	β	(SE)	β	(SE)	β	(SE)	β	(SE)
Factor loadings										
W1 Emotional abuse	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W1 Physical abuse	0.874*	(0.059)	0.869*	(0.080)	0.788*	(0.055)	0.927*	(0.035)	0.889*	(0.042)
W1 Severe physical abuse	0.474*	(0.036)	0.590*	(0.061)	0.440*	(0.035)	0.503*	(0.026)	0.493*	(0.028)
W2 Daily Stress Risk Appraisal 1	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W2 Daily Stress Risk Appraisal 2	0.497*	(0.064)	0.468*	(0.084)	0.498*	(0.065)	0.493*	(0.055)	0.481*	(0.056)
W2 Daily Stress Risk Appraisal 3	0.874*	(0.105)	0.770*	(0.121)	0.986*	(0.117)	0.816*	(0.085)	0.819*	(0.086)
W2 Daily Stress Risk Appraisal 4	0.647*	(0.090)	0.584*	(0.107)	0.692*	(0.096)	0.722*	(0.081)	0.714*	(0.081)
W2 Daily Stress Risk Appraisal 5	0.479*	(0.064)	0.419*	(0.073)	0.456*	(0.064)	0.441*	(0.055)	0.435*	(0.055)
W2 Daily Stress Risk Appraisal 6	0.802*	(0.098)	0.717*	(0.107)	0.796*	(0.096)	0.681*	(0.069)	0.684*	(0.071)
W3 Symptom item 1	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W3 Symptom item 2	1.427*	(0.180)	0.404*	(0.022)	−0.005	(0.006)	0.584*	(0.105)	0.889*	(0.042)
W3 Symptom item 3	1.988*	(0.242)	0.783*	(0.045)	0.167*	(0.024)	0.674*	(0.093)	0.493*	(0.028)
W3 Symptom item 4	1.851*	(0.241)	0.951*	(0.026)	0.348*	(0.037)	0.760*	(0.113)	1.000	(0.000)
W3 Symptom item 5	1.530*	(0.212)	0.744*	(0.041)	1.057*	(0.047)	–	–	0.481*	(0.056)
W3 Symptom item 6	1.279*	(0.178)	0.770*	(0.043)	0.771*	(0.046)	–	–	0.819*	(0.086)
W3 Symptom item 7	2.200*	(0.288)	1.352*	(0.091)	0.781*	(0.047)	–	–	0.714*	(0.081)
W3 Symptom item 8	1.863*	(0.256)	–	–	0.538*	(0.040)	–	–	–	–
W3 Symptom item 9	1.628*	(0.217)	–	–	0.716*	(0.048)	–	–	–	–
W3 Symptom item 10	1.575*	(0.212)	–	–	0.372*	(0.038)	–	–	–	–
W1 Symptom item 1	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W1 Symptom item 2	1.388*	(0.148)	0.424*	(0.017)	0.005*	(0.010)	0.665*	(0.091)	1.066*	(0.121)
W1 Symptom item 3	1.360*	(0.157)	0.806*	(0.044)	0.223*	(0.027)	0.526*	(0.082)	0.600*	(0.102)
W1 Symptom item 4	1.206*	(0.144)	0.981*	(0.035)	0.372*	(0.033)	0.576*	(0.080)	0.355*	(0.075)
W1 Symptom item 5	1.067*	(0.133)	0.708*	(0.046)	1.072*	(0.043)	–	–	0.166*	(0.048)
W1 Symptom item 6	0.988*	(0.125)	0.697*	(0.044)	0.845*	(0.042)	–	–	0.156*	(0.047)
W1 Symptom item 7	1.322*	(0.163)	1.635*	(0.080)	0.729*	(0.042)	–	–	0.268*	(0.067)
W1 Symptom item 8	1.436*	(0.166)	–	–	0.713*	(0.041)	–	–	–	–
W1 Symptom item 9	1.182*	(0.136)	–	–	0.817*	(0.042)	–	–	–	–
W1 Symptom item 10	1.138*	(0.134)	–	–	0.448*	(0.034)	–	–	–	–
Regression estimates										
W1 Abuse → W2 Daily SRA	0.061*	(0.012)	0.080*	(0.024)	0.054*	(0.011)	0.050*	(0.011)	0.053*	(0.011)
W2 Daily SRA → W3 Symptoms	0.048*	(0.012)	0.081*	(0.023)	0.207*	(0.038)	0.022	(0.011)	0.071*	(0.026)
W1 Abuse → W3 Symptoms	0.001	(0.000)	0.028*	(0.009)	0.012	(0.008)	−0.003	(0.004)	−0.009	(0.009)
W1 Symptoms → W3 Symptoms	0.283*	(0.059)	0.200*	(0.041)	0.315*	(0.029)	0.313*	(0.054)	0.839*	(0.130)
Indirect effect	0.003*	(0.036)	0.007*	(0.002)	0.011*	(0.003)	0.001	(0.001)	0.004*	(0.002)
Total effect	0.004	(0.000)	0.035*	(0.009)	0.023*	(0.007)	−0.002	(0.004)	−0.006	(0.008)

Note. GAD, generalized anxiety disorder; MDD, major depressive disorder; AUD, alcohol use disorder; SUD, substance use disorder; SRA, latent composite score of stress risk appraisal; NA, negative affect; β, unstandardized regression or factor loading estimate; SE, standard error of β. For GAD severity, items (in order of presentation) were worry-linked feelings of being keyed up/restless, trouble falling asleep, difficulty staying asleep, trouble concentrating, trouble remembering, low on energy, tired quickly, muscle aches, and interference with life. For MDD severity, items were depressed mood linked to symptoms of anhedonia, low energy, loss/increased appetite, difficulty falling asleep, trouble concentrating, feeling worthless, and thinking a lot about death. For PD severity, items were having panic spells/attacks, spells/attacks occurred for no reason, the number of panic attacks, attacks occurred during danger/being the center of attention, heart pounds during attacks, chest pain/tightness, sweating, trembling/shaking, hot flashes/chills, and feelings of unreality. For AUD severity, items were a strong urge to consume alcohol, emotional problems from using alcohol, excessive drinking time, increased tolerance to its effects, and emotional issues stemming from using alcohol. For SUD severity, using larger amounts than intended, adverse effects of using substances during school/work, use increased odds of getting hurt, use contributed to emotional issues, a strong desire to use, substance use took excessive time, and increased tolerance to its effects. All mediation models within this set of analyses showed good fit with various adulthood psychopathology severity outcomes: GAD ($\chi^2(372) = 680.120$, $p < .001$, CFI = .961, RMSEA = .017), MDD ($\chi^2(225) = 496.041$, $p < .001$, CFI = .989, RMSEA = .021), PD ($\chi^2(371) = 1204.994$, $p < .001$, CFI = .930, RMSEA = .029), AUD ($\chi^2(114) = 228.776$, $p < .001$, CFI = .950, RMSEA = .020), and SUD ($\chi^2(224) = 417.872$, $p < .001$, CFI = .967, RMSEA = .018).

* Statistically significant at $p < .01$ (two-tailed).

EMA evidence that childhood abuse predicted future daily stressors (Baker et al., 2020) and stress reactivity (Cristobal-Narvaez et al., 2016). Increased EMA-indexed heightened emotional reactivity to stressors among childhood trauma-exposed persons, in turn, foreshadowed the emergence of more anxiety, depressive, and psychosis symptoms (van Nierop et al., 2018). We proffered plausible ideas to explain these observations in hypothesis-generating ways that future empirical studies should evaluate to advance clinical psychological science.

Why did daily stress reactivity and appraisal mediate the 18-year pathways between childhood maternal and paternal abuse predicting GAD, MDD, and PD severity? These results concurred with biopsychosocial theories. Potential biological mechanisms include dysregulated stress hormones (Otte et al., 2005), such as catecholamines and glucocorticoid-signaling, released during challenging situations (van Zuiden et al., 2013). Prospective investigations are necessary to test these conjectures.

Regarding psychosocial processes, childhood maltreatment might, over time, contribute to issues with social-cognitive skills, such as perspective-taking, as these processes typically take shape via daily interactions with primary caregivers (Weijers et al., 2018). In addition, early encounters with emotional and physical abuse could prompt the formation of an involuntary defeat strategy (Sloman and Taylor, 2016), marked by a psychobiological response to threats designed to minimize the prospect of future harm. Although this response can be beneficial in certain situations, it can also trigger stress mechanisms that ultimately harm long-term mood and well-being (Shapero et al., 2014). The stress and coping theory (Folkman and Moskowitz, 2004) also likely underpinned our findings, offering insight into how individuals evaluate and manage a spectrum of stressful situations, encompassing everyday challenges and substantial life events. Abused individuals might resort to detrimental habits and lifestyles, such as emotional avoidance and physical inactivity, to deal with the repercussions of traumatic

Table 5
Wave 1 (W1) Childhood paternal abuse predicting Wave 3 (W3) mental disorder severity via Wave 2 (W2) daily stress risk appraisal.

	GAD		MDD		Panic disorder		AUD		SUD	
	β	(SE)	B	(SE)	β	(SE)	β	(SE)	β	(SE)
Factor loadings										
W1 Emotional abuse	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W1 Physical abuse	0.725*	(0.065)	0.707*	(0.069)	0.715*	(0.068)	1.107*	(0.041)	1.106*	(0.044)
W1 Severe physical abuse	0.444*	(0.046)	0.452*	(0.048)	0.430*	(0.044)	0.668*	(0.032)	0.682*	(0.034)
W2 Daily Stress Risk Appraisal 1	1.000	(0.000)	1.000	(0.000)	1.000	(0.000)	1.000	(0.000)	1.000	(0.000)
W2 Daily Stress Risk Appraisal 2	0.503*	(0.065)	0.436*	(0.061)	0.493*	(0.065)	0.495*	(0.055)	0.484*	(0.055)
W2 Daily Stress Risk Appraisal 3	0.846*	(0.104)	0.710*	(0.092)	0.957*	(0.114)	0.802*	(0.083)	0.806*	(0.085)
W2 Daily Stress Risk Appraisal 4	0.669*	(0.091)	0.604*	(0.085)	0.703*	(0.097)	0.748*	(0.083)	0.741*	(0.083)
W2 Daily Stress Risk Appraisal 5	0.478*	(0.063)	0.391*	(0.055)	0.454*	(0.064)	0.444*	(0.055)	0.438*	(0.055)
W2 Daily Stress Risk Appraisal 6	0.795*	(0.098)	0.681*	(0.086)	0.789*	(0.096)	0.677*	(0.068)	0.681*	(0.070)
W3 Symptom item 1	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W3 Symptom item 2	1.405*	(0.174)	0.407*	(0.012)	0.325*	(0.045)	0.588*	(0.105)	1.039*	(0.086)
W3 Symptom item 3	1.964*	(0.235)	0.769*	(0.027)	0.175*	(0.025)	0.664*	(0.094)	0.123*	(0.023)
W3 Symptom item 4	1.814*	(0.232)	0.923*	(0.018)	0.345*	(0.036)	0.758*	(0.113)	0.064*	(0.017)
W3 Symptom item 5	1.528*	(0.208)	0.738*	(0.026)	1.076*	(0.048)	–	–	0.032*	(0.012)
W3 Symptom item 6	1.266*	(0.176)	0.675*	(0.029)	0.778*	(0.047)	–	–	0.045*	(0.013)
W3 Symptom item 7	2.176*	(0.283)	1.350*	(0.057)	0.788*	(0.047)	–	–	0.067*	(0.017)
W3 Symptom item 8	1.827*	(0.251)	–	–	0.537*	(0.040)	–	–	–	–
W3 Symptom item 9	1.614*	(0.213)	–	–	0.719*	(0.048)	–	–	–	–
W3 Symptom item 10	1.531*	(0.207)	–	–	0.361*	(0.037)	–	–	–	–
W1 Symptom item 1	1.000	—	1.000	—	1.000	—	1.000	—	1.000	—
W1 Symptom item 2	1.316*	(0.142)	0.421*	(0.012)	0.003	(0.010)	0.638*	(0.088)	1.059*	(0.125)
W1 Symptom item 3	1.333*	(0.151)	0.753*	(0.030)	0.221*	(0.027)	0.521*	(0.081)	0.648*	(0.106)
W1 Symptom item 4	1.172*	(0.139)	0.975*	(0.020)	0.373*	(0.033)	0.578*	(0.079)	0.351*	(0.076)
W1 Symptom item 5	1.041*	(0.128)	0.748*	(0.027)	1.062*	(0.042)	–	–	0.185*	(0.054)
W1 Symptom item 6	0.967*	(0.121)	0.667*	(0.028)	0.836*	(0.041)	–	–	0.160*	(0.049)
W1 Symptom item 7	1.283*	(0.156)	1.641*	(0.049)	0.722*	(0.041)	–	–	0.282*	(0.069)
W1 Symptom item 8	1.394*	(0.159)	–	–	0.711*	(0.040)	–	–	–	–
W1 Symptom item 9	1.137*	(0.128)	–	–	0.813*	(0.041)	–	–	–	–
W1 Symptom item 10	1.122*	(0.131)	–	–	0.446*	(0.034)	–	–	–	–
Regression estimates										
W1 Abuse → W2 Daily SRA	0.048*	(0.011)	0.053*	(0.012)	0.038*	(0.010)	0.037*	(0.012)	0.040*	(0.012)
W2 Daily SRA → W3 Symptoms	0.047*	(0.012)	0.091*	(0.018)	0.205*	(0.036)	0.022	(0.011)	0.075	(0.026)
W1 Abuse → W3 Symptoms	0.003	(0.003)	0.015*	(0.005)	0.007	(0.007)	−0.004	(0.005)	−0.015	(0.010)
W1 Symptoms → W3 Symptoms	0.274*	(0.049)	0.266*	(0.027)	0.318*	(0.029)	0.311*	(0.053)	0.859*	(0.132)
Indirect effect	0.002*	(0.001)	0.005*	(0.001)	0.008*	(0.002)	0.001	(0.001)	0.003	(0.001)
Total effect	0.005	(0.003)	0.020*	(0.005)	0.015	(0.007)	−0.003	(0.005)	−0.012	(0.010)

Note. GAD, generalized anxiety disorder; MDD, major depressive disorder; AUD, alcohol use disorder; SUD, substance use disorder; SRA, latent composite score of stress risk appraisal; NA, negative affect; β, unstandardized regression or factor loading estimate; SE, standard error of β. For GAD severity, items (in order of presentation) were worry-linked feelings of being keyed up/restless, trouble falling asleep, difficulty staying asleep, trouble concentrating, trouble remembering, low on energy, tired quickly, muscle aches, and interference with life. For MDD severity, items were depressed mood linked to symptoms of anhedonia, low energy, loss/increased appetite, difficulty falling asleep, trouble concentrating, feeling worthless, and thinking a lot about death. For PD severity, items were having panic spells/attacks, spells/attacks occurred for no reason, the number of panic attacks, attacks occurred during danger/being the center of attention, heart pounds during attacks, chest pain/tightness, sweating, trembling/shaking, hot flashes/chills, and feelings of unreality. For AUD severity, items were a strong urge to consume alcohol, emotional problems from using alcohol, excessive drinking time, increased tolerance to its effects, and emotional issues stemming from using alcohol. For SUD severity, using larger amounts than intended, adverse effects of using substances during school/work, use increased odds of getting hurt, use contributed to emotional issues, a strong desire to use, substance use took excessive time, and increased tolerance to its effects. All mediation models within this set of analyses showed good fit with various adulthood psychopathology severity outcomes: GAD ($\chi^2(372) = 778.783$, $p < .001$, CFI = .965, RMSEA = .020), MDD ($\chi^2(225) = 559.426$, $p < .001$, CFI = .984, RMSEA = .023), PD ($\chi^2(371) = 1399.774$, $p < .001$, CFI = .914, RMSEA = .032), AUD ($\chi^2(114) = 234.847$, $p < .001$, CFI = .965, RMSEA = .018), and SUD ($\chi^2(224) = 433.677$, $p < .001$, CFI = .906, RMSEA = .023).

* Statistically significant at $p < .01$ (two-tailed).

experiences, potentially leading to mental health issues across long periods. Relatedly, more encounters with childhood parental abuse could heighten individuals' susceptibility to future stress exposure, thereby elevating the likelihood of developing future mental health issues (cf. *sensitization hypothesis*; Heim and Nemeroff, 2001; Heim et al., 2000). Future longitudinal mediational investigations should evaluate these ideas.

The lack of connection between maternal and paternal abuse and higher adulthood AUD/SUD severity via daily stress reactivity and risk appraisal suggests that these mechanisms may not account for relationships between childhood abuse and AUD/SUD severity. Specifically, although higher abuse was linked to greater stress reactivity and worse appraisals, there was no indirect effect on AUD/SUD severity. However, greater stress reactivity at W2 might lead to more drinking or substance use, which could have accounted for the increased symptom severity. Future prospective research designs that assess stress-related drinking and substance use could examine this possibility.

Intriguingly, the mediation effect sizes predicting GAD, MDD, and PD severity were stronger when the predictor was baseline childhood maternal abuse (24.5–83.0%) than paternal abuse (19.5–56.0%). Such observations were concordant with evidence that the enduring impact and intricate interplay between children and their mothers, compared to fathers, persisted long into adulthood (Rosenthal and Kobak, 2010). Maternal abuse may pose a more direct risk for the development of adult psychopathology compared to paternal abuse, possibly influenced by variations in the frequency of interaction with each parent. Recent findings illustrated that emotion dysregulation was a mediator in the connection between childhood maternal abuse and depressive symptoms in adulthood, whereas paternal abuse was a direct predictor (Moretti and Craig, 2013). Likewise, several recent studies have noted that childhood abuse by mothers, as opposed to fathers, was linked to decreased psychological well-being, elevated psychopathological risk,

and increased distress (Kong and Martire, 2019; VanMeter et al., 2021). Additional inquiry is required to elucidate the mechanisms through which interactions and abuse by mothers, as opposed to fathers, can impact enduring mental health outcomes.

Additionally, in our moderated mediation analyses, childhood maternal and paternal abuse did not substantially interact to predict any mental disorder severity outcome. Nonetheless, future studies should still explore if, in the context of a two-parent household, the abusive effect from one parent might be buffered by affection, care, and warmth from another (Ng et al., 2024). On that note, measures capturing constructs of parental affection (e.g., Bartek et al., 2021) should be included in the analyses while examining potential mechanisms of the adverse effects of childhood abuse on psychopathology in adulthood.

4.1. Limitations and strengths

The current study exhibited certain limitations. First, the measures relied on self-reporting and could be influenced by respondent bias (e.g., underreporting alcohol or substance use). Retrospective childhood abuse self-reports may be influenced by individuals' future experiences, potentially introducing bias, given the limited agreement between prospective and retrospective assessments of childhood abuse (Baldwin et al., 2019). Considering the baseline sample's average age of 45, it is worth noting that the assessment of anxiety, mood, and substance use disorders took place about 18 years or more following the occurrence of childhood maltreatment, well beyond the typical age of onset for these conditions (Solmi et al., 2022). Longitudinal measures and designs are thus needed in future studies (Danese, 2020). Relatedly, no examined variables were isolated or singular occurrences. For instance, the mediator (daily stress reactivity/appraisal) is probably persistent. Thus, although abuse, stress reactivity/appraisal, and psychopathology were assessed at various waves, it does not imply a sequential origin for them. This issue is compounded by shared method variance by using the same informant for all three waves, possibly resulting in evident reporting bias. Second, confounding covariates linked to selection bias in the childhood abuse variable, such as behavioral genetics and parental psychopathology, might alter results and should also be included in future studies. Third, subsequent replication endeavors should incorporate assessments aligned with DSM-5 criteria since the present study deployed assessments consistent with the DSM-III-R criteria. Fourth, since the sample was mostly White, it was not representative of the U.S. population. Future studies should recruit diverse samples.

Despite its limitations, the present study had several notable strengths. To begin, we employed a longitudinal approach across three measurement waves spanning 18 years. Secondly, all the assessments employed produced scores that were both psychometrically reliable and valid. Thirdly, our study investigated the role of a novel mediator indexed by EMAs, daily stress reactivity and risk appraisal, in potentially explaining how maternal and paternal childhood abuse may predict the symptom severity of GAD, MDD, PD, AUD, and SUD in adulthood. We underscore the significance of examining plausible mechanisms in the link between childhood parental maltreatment and adult psychopathology, contributing to the body of research on the mental health consequences of childhood parental abuse. Last, our findings remained similar after adjusting for baseline age, which might imply that the odds of misremembering childhood memories and related issues (e.g., unwillingness to report abuse) were not confounding variables.

4.2. Conclusions

To summarize, daily stress reactivity and risk appraisal functioned as mediators in the 18-year longitudinal relations between childhood maternal and paternal abuse, forecasting adulthood GAD, MDD, and PD, but not AUD and SUD symptom severity. Although child abuse is an established risk factor for psychopathology, its observed long-term effects on daily stress reactivity/risk appraisal are more novel. Clinicians must thus acquire knowledge about and identify childhood maltreatment experiences and develop optimal strategies to respond to diverse perceived stressors for preventing and treating patients who have endured them (Jones et al., 2020). Broadly, our research underscores the imperative need for ongoing assessment and intervention efforts in trauma-informed care across inpatient and outpatient treatment contexts (Bendall et al., 2021). More specifically, our study suggested that EMA indices of stress reactivity/appraisal might provide better measurement-based care and routine outcome monitoring than retrospective measures for adult clients with child maltreatment experiences undergoing psychotherapies (Lutz et al., 2021). Cognitive-behavioral therapies such as cognitive processing therapy and prolonged exposure therapy could remedy the adverse long-term effects of childhood maltreatment (Carpenter et al., 2018; Toth and Manly, 2018). Overall, our study indicates that early adulthood among persons who experienced childhood parental abuse represents a crucial period of vulnerability for long-term mental illness and a critical timeframe for targeted intervention strategies, primarily by reducing stress reactivity/risk appraisal.

Scientific interdisciplinarity and/or collaboration

Our endeavor can guide personalized approaches to education, diagnosis, prevention, and intervention (Hayes et al., 2019; Reber, Canning, & Harackiewicz, 2018; van Os, Delespaul, Wigman, Myin-Germeys, & Wichers, 2013).

Diversity and representation in clinical science

Our sample used a predominantly White, non-Hispanic sample. Nonetheless, we acknowledged this as a limitation in the discussion of the paper.

Open and transparent practices and methodological rigor

Data can be accessed via the Inter-university Consortium for Political and Social Research (ICPSR), which hosts the Midlife Development in the United States (MIDUS) Series (https://www.icpsr.umich.edu/web/ICPSR/series/203). Analytic scripts in R can be made available upon reasonable request.

Funding sources

The data used in this publication were made available by the Data Archive on the University of Wisconsin at Madison Institute on Aging, 1300 University Avenue, 2245 MSC, Madison, Wisconsin 53706-1532. Since 1995, the MIDUS study has been funded by the following: John D. and Catherine T. MacArthur Foundation Research Network; National Institute on Aging (P01-AG020166); National Institute on Aging (U19-AG051426). Dr. Zainal received funding support from the National University of Singapore (NUS) Presidential Young Professorship Start-Up Fund. Dr. Van Doren was supported by the National Institute on Drug Abuse (T32DA007250). The original investigators and funding agency are not responsible for the analyses or interpretations presented here.

CRediT authorship contribution statement

Nur Hani Zainal: Writing – review & editing, Writing – original draft, Visualization, Validation, Formal analysis, Data curation, Conceptualization. **Chui Pin Soh:** Writing – review & editing, Validation, Data curation. **Natalia Van Doren:** Writing – review & editing, Validation, Supervision, Conceptualization.

Declaration of competing interest

None of the authors have any conflict of interest.

Acknowledgments

Our study received institutional review board (IRB) approval from universities that participated in the Midlife Development in the United States (MIDUS) project. Informed consent was obtained from participants per IRB requirements at Harvard University, Georgetown University, the University of California at Los Angeles, and the University of Wisconsin at Madison. Since this study used a publicly available dataset, it was exempt from IRB approval.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2024.04.068.

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