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ORIGINAL CONTRIBUTION

Early life adversity and psychopathology in preschoolers: mechanisms and moderators

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Abstract

Developmental theories suggest that exposure to early life adversity (ELA) alters developing emotional response systems, predicting risk for psychopathology across the life span. The present study examines whether negative emotionality (NE), a trait-like measure of emotionality that develops during early childhood, mediates the association between ELA and psychopathology in a representative sample of 917 preschoolers ($M_{age} = 3.84$). Additionally, we explored whether cognitive control, which supports attentional focusing and inhibition and has been identified as a transdiagnostic protective factor, moderates the impact of heightened emotionality following adversity on psychopathology risk. We utilized parent report of adversity, psychopathology, and NE and parent report and task-based measures of cognitive control. Structural equation modeling of cross-sectional data revealed that NE partially mediated the link between ELA and psychopathology symptoms. Moreover, parent-reported cognitive control buffered this link such that the effect of ELA on psychopathology through NE was stronger in children with low versus high cognitive control. These results identify elevated NE as one mechanism linking ELA and psychopathology, specifically among children with poorer top-down control, informing our understanding of key risk and protective factors among adversity-exposed children.

Keywords Adversity · Psychopathology · Temperament · Cognitive control

Introduction

Early life adversity (ELA), conceptualized as the absence of expected inputs (e.g., neglect) or presence of unanticipated threats (e.g., maltreatment, community violence) that require significant adaptation by the child [1], is a well-known risk factor for adverse physical and mental health outcomes across the life span. Developmental theories suggest that common neurobiological and psychological mechanisms explain the diverse consequences of adversity exposure [2]. The present study examines the role of one mechanism, heightened emotionality, in mediating the link between ELA and psychopathology. Given the wide-ranging negative outcomes of ELA, it is crucial to identify protective factors that prevent adverse outcomes in children exposed to adversity. Accordingly, we explored whether better cognitive control alters the pathway from adversity through emotionality to psychopathology, ultimately decreasing risk for psychological distress following ELA. Because early childhood is a period of heightened sensitivity to experience [3], we examined pathways linking ELA to psychopathology during this critical stage.

Adversity and psychopathology

A wealth of research links adversity exposure to elevated risk for subsequent psychopathology [2]. Exposure to adversity during the early years of life is thought to alter biological and psychological development, setting the stage for increased rates of internalizing and externalizing psychopathology throughout development [4, 5]. Developmental psychopathology theories suggest that ELA alters still developing

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emotion response systems, shaping the way individuals react to their environment and regulate these responses as they grow [6, 7]. Theories of temperament highlight the dimensions of emotionality (i.e., an individual's characteristic emotional responses) and cognitive control (i.e., the ability to control and shift attention and self-regulate) as two evolutionary systems that develop early in life and contribute to later behavior and risk for psychopathology [8]. Because these traits develop across early childhood, are influenced by experience, including adverse experiences, and jointly shape psychopathology risk, we sought to explore the impacts of ELA and the mediating and moderating role of emotionality and cognitive control during the preschool period.

Mediating role of negative emotionality

Models of adversity and psychopathology suggest that alterations in emotional response may be one key pathway linking ELA to diverse psychopathological outcomes [9, 10]. Repeated exposure to threatening or unsupportive contexts may cause young children to experience chronically elevated negative emotions and difficulties in emotion regulation. Supporting this association, previous studies of preschool and early school-aged children have found that exposure to unsupportive parenting or parental conflict predicts increased emotional reactivity, which in turn is associated with increased internalizing and externalizing symptoms [11, 12]. Further, maltreatment is associated with increased negative affect [13] in school-aged children, and heightened emotional reactivity has been found to mediate the association between maltreatment and psychopathology in older children [14]. A recent meta-analysis identifies emotion dysregulation as a mechanism linking adversity to psychopathology across later childhood and adolescence [15]. In the present study, we examined the mediating role of negative emotionality (NE), a temperamental form of emotional reactivity characterized by the tendency to experience anger, fear, and sadness and having difficulty being soothed [8]. NE is a transdiagnostic risk factor that is measurable as early as infancy, becomes increasingly stable across time and context during childhood, and is associated with more difficulty regulating negative emotions [16]. Past studies suggest that, in children, a history of maltreatment predicts higher NE [17] and positive parenting predicts decreases in NE [18]. However, current research still largely considers NE an unmalleable risk factor, and studies to date have not examined whether elevations in NE following adversity mediate the link between ELA and psychopathology. Given the early development of NE and implications of heightened NE on risk for many forms of psychopathology across the life span, we sought to explore the mechanistic role of NE during the preschool years when NE is still developing and most sensitive to both detrimental environmental effects and potential intervention.

Moderating role of cognitive control

Although ELA is associated with diverse negative health outcomes, only a minority of youth who have experience ELA go on to develop psychopathology [6, 19], highlighting the importance of understanding protective factors that may promote healthy development following adversity exposure. One transdiagnostic protective factor that develops early in life and predicts decreased risk for psychopathology is cognitive control, a broad, multifaceted construct that includes the ability to focus attention, inhibit distraction, and organize behavior toward goals [20, 21]. Two largely separate bodies of literature have studied cognitive control across development. The first approach relies on self- or informant report of trait-like effortful control, which reflects cognitive control abilities in everyday life [22]. The second measures performance on a variety of neuropsychological tasks of executive function (EF), which are thought to tap different overlapping dimensions of cognitive control [20]. These measures of cognitive control overlap conceptually [23, 24], although correlations between them tend to be small to moderate [25, 26]. Cognitive control exhibits a protracted development; however, previous work suggests that better parent-reported [27, 28] and task-based [29] cognitive control predicts lower psychopathology even in preschoolers.

This protective role of cognitive control may be especially relevant among adversity-exposed youth, who are at elevated risk for psychopathology. Past research suggests that better performance on inhibition and attentional switching tasks predicts less psychopathology among preschoolers experiencing homelessness [30] and task-based cognitive control dampens the effect of maltreatment on externalizing symptoms among preschoolers in foster care [31]. Additionally, cognitive control buffers the impact of community adversity [32] and poverty [33] on depression in adolescents, highlighting its potential protective role for these youth.

One way that cognitive control may promote better adjustment following ELA is by supporting better top-down control of heightened emotionality, decreasing risk for psychopathology. Several studies support the idea that better cognitive control buffers the effect of elevated NE on psychopathology risk in school-aged children [34, 35]. In this study, we assessed whether cognitive control moderates the link from adversity to psychopathology through NE in preschoolers such that this pathway would be stronger in youth with low relative to high cognitive control.

Study overview

While evidence suggests that ELA predicts altered emotion processing [12, 36] and risk for psychopathology [5] in early childhood, less work has examined mechanistic paths linking ELA to adverse outcomes during the preschool years. Central to developmental models of the 'embodiment' of adversity is the hypothesis that early childhood represents a sensitive period during which emotion response systems are still developing and therefore more easily altered by environmental exposure [37, 38]. Accordingly, this study investigated the novel hypothesis that NE, an aspect of trait emotionality that emerges as a potent, stable predictor of psychopathology during early childhood, may be shaped by ELA *and* represent a mechanism through which adversity exerts lifelong effects on psychological wellbeing. Further, we examine whether cognitive control buffers the path from ELA to psychopathology through heightened emotionality in early childhood when cognitive control is still developing and may represent a targetable protective factor among adversity-exposed young children. To assess adversity, we used caregiver report on a semi-structured interview and self-report measures, which allowed us to capture a range of adversity exposure in the home and community. Negative emotionality and effortful control, a trait-like dimension of cognitive control, were assessed via caregiver report. We additionally operationalized cognitive control using two common EF tasks completed by a subset of children to examine both parent report and task-based assessments of cognitive control. Psychopathology was measured using a semi-structured interview designed specifically to measure symptoms of psychological distress among preschool-aged children. We hypothesized that: (1) adversity would predict more psychopathology, and that this association would be partially explained by elevated NE; and (2) that cognitive control would moderate this link, such that the mediation from adversity to psychopathology through NE would be attenuated in youth with better cognitive control.

Methods

Participants and procedures

Participants included 917 children ($M_{age} = 3.84$, 49.2% female, 50.8% male), who with a primary caregiver were enrolled in the Duke Preschool Anxiety Study [39], which recruited a representative sample of preschool-aged children (i.e., ages two to five) from primary care clinics in Durham County, North Carolina. Although the study sample was over-selected for the presence of anxiety symptoms, analysis of the full sample of 917 includes sample weights so that conclusions can be drawn about the population of children in the diverse southeastern region from which they were drawn. A subset of the original sample ($n = 330$ with usable data) was recruited to participate in a laboratory visit that included EF tasks. Additional details about recruitment procedures for all phases of the study have been detailed elsewhere [39] and in the supplement. Race and ethnicity data were collected in accordance with the National Institutes of Health policy and based on US census categories, although we acknowledge that this approach is an imperfect operationalization of lived experience and advocate caution in interpreting effects of race and ethnicity characterized in this way. Parents of children in the sample predominantly identified their children as non-White (55.8%) and 29.5% reported family incomes below $30,000 per year (see Supplement for additional demographic detail). Primary caregivers provided written consent and children provided verbal assent for all study procedures. All study procedures were conducted in accordance with the ethical standards of the Declaration of Helsinki and the American Psychological Association and were approved by the university's Institutional Review Board.

Measures

See supplement for additional information about measure composition and reliability and validity.

Adversity

Caregivers reported on their child's exposure to adversity using two self-report measures, the Conflict Tactics Scale, second edition (CTS-2; [40]), and the Conflict Tactics Scale-Parent Child (CTS-PC; [41]), and semi-structured interview, the Preschool Age Psychiatric Assessment (PAPA; [42]). The CTS-2 assesses exposure to intimate partner violence between caregivers. The CTS-PC assesses physical and sexual abuse and neglect. Finally, the PAPA includes questions assessing children's exposure to abuse, neglect, family and neighborhood violence, and other potentially traumatic events. A sum score was calculated reflecting the total number of unique adverse experiences across all measures. In this sample, 52.5% of participants were exposed to at least one threatening experience (34.7% experienced physical abuse, 2.9% experienced sexual abuse, and 33.1% experienced family or neighborhood violence) and 10.4% experienced neglect.

Negative emotionality

NE was assessed via caregiver report on the very short form of either the Early Childhood Behavior Questionnaire (ECBQ; [43]) or the Childhood Behavior Questionnaire (CBQ; [15, 44]), depending on child age. Mean scores were

calculated of the NE scale and the low soothability subscale for each participant.

Effortful control

Effortful control was assessed via caregiver report on the very short form of the ECBQ or CBQ depending on child age. Mean scores of the effortful control scale were calculated for each participant.

Executive function

EF was measured via two tasks, the Day–Night Stroop (response inhibition; [45]) and Dimensional Change Card Sort (DCCS, rule switching; [46]). Hierarchical linear modeling was used to create slope scores reflecting sustained accuracy across the Day–Night Stroop and across the post-switch phase of the Dimensional Change Card Sort. These scores were standardized and averaged to create a more robust composite score that reflects common EF shared across these two tasks for each participant [47], although patterns were similar when considering each task alone (see Supplemental Table S1).

Psychopathology

Psychopathology was measured via caregiver report on the PAPA [42]. During the PAPA interview, caregivers reported on children's symptoms of psychological disorders over the previous 3 months as defined by the Diagnostic and Statistical Manual (DSM-IV; American Psychiatric Association, 2000). Symptoms were summed across disorders to calculate an overall psychopathology score for each participant. Additionally, we tested the robustness of effects by exploring whether results were similar for internalizing and externalizing symptoms separately (see Supplement).

Data analytic plan

To examine the pathways from adversity to psychopathology, we conducted moderated mediation analyses (Fig. 1) using the lavaan package in R [48]. First, we explored bivariate correlations between all variables and examined whether NE[1] mediates the link between ELA and total psychopathology symptoms. Next, we ran moderated mediation analyses to examine whether this association is moderated by EC. We used full information maximum likelihood estimation to account for missing data and added sampling weights to weight the data back to the original representative sample. We also used moderated mediation analyses to examine the moderating effect of EF in the subset of participants who completed the laboratory visit. Because this sample was drawn from a case control study based on the presence of an anxiety diagnoses, analyses were also run separately in participants with and without any anxiety diagnosis. Because adversity exposure is not even distributed across race, socioeconomic status, and gender, we initially conducted analyses without these demographic controls to assess the full impact of ELA. We then examined whether patterns of findings hold controlling for potential demographic confounders, namely participant age, gender, race, parent education, and family income. In all analyses, adversity, NE, effortful control, and EF were standardized, and interactions were computed by multiplying standardized variables. Exploratory follow-up analyses examined whether patterns of moderated mediation would differ when considering two well-validated [9, 14] dimensions of adversity, threat and deprivation, separately; results are presented in the Supplement.

[1] We also re-ran all analyses using the low soothability subscale of NE as the mediator in order to explore whether observed effects were attributable specifically to the heightened emotional reactivity dimension of NE that is captured by low soothability. Results were similar and maintained the same significance patterns using either NE or low soothability; however, because the low soothability subscale includes only two items, and in line with several studies exploring interactions between NE and cognitive control, we chose to present results using the full NE scale.

Results

Bivariate correlations

Table 1 presents descriptive statistics and bivariate correlations among the variables.

Adversity predicting psychopathology through NE

We first examined whether NE would mediate the association between exposure to adversity and overall symptoms of psychopathology. We observed a significant effect of adversity exposure ($b = 0.12$, SE = 0.03, $\beta = 0.11$, $z = 3.43$, $p = 0.001$) on NE and significant effects of adversity ($b = 2.44$, SE = 0.21, $\beta = 0.35$, $z = 11.78$, $p < 0.001$) and NE ($b = 1.98$, SE = 0.20, $\beta = 0.29$, $z = 9.74$, $p < 0.001$) on psychopathology. Moreover, we found a significant indirect effect of adversity on psychopathology through NE ($b = 0.23$, SE = 0.07, $\beta = 0.03$, $z = 3.24$, $p = 0.001$). All results held when controlling for potential confounders.

Moderation by effortful control

We next examined whether the links between adversity, NE, and psychopathology would differ based on children's level of effortful control in a moderated mediation

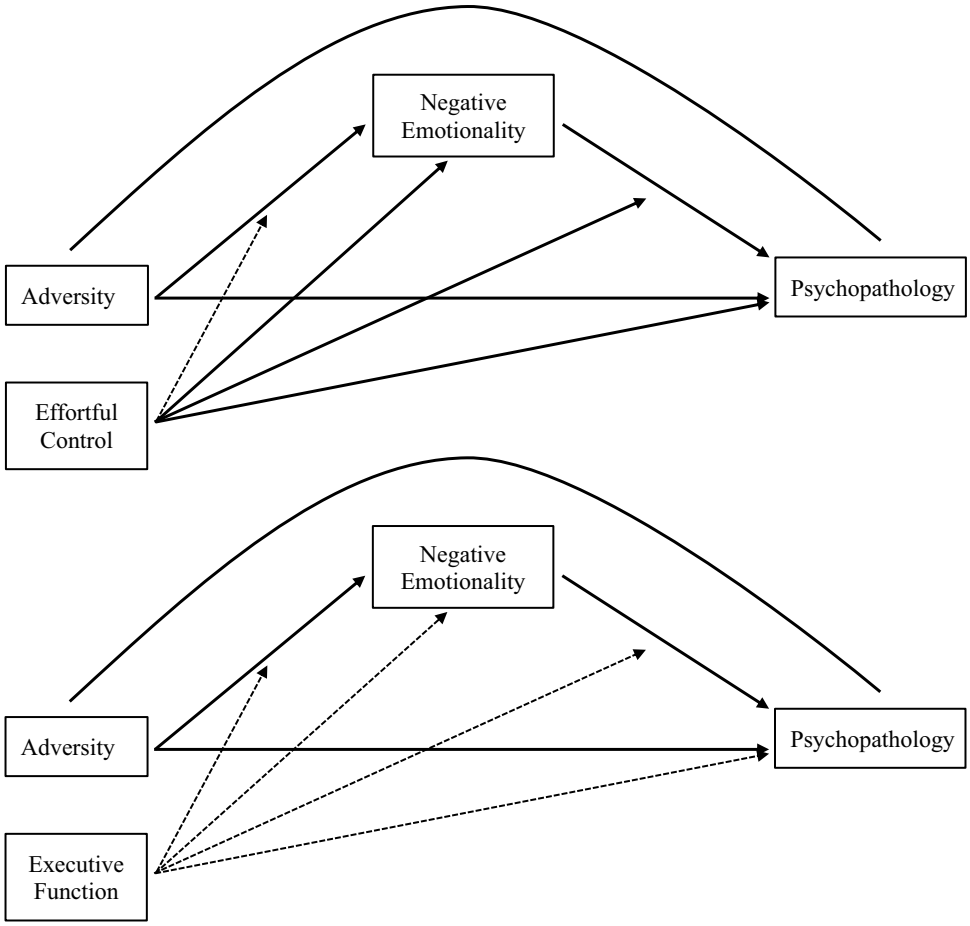

Fig. 1 Pathway from early life adversity to total psychopathology symptoms is mediated by negative emotionality and moderated by effortful control (top), but not executive function (bottom). Significant paths are indicated with solid lines; non-significant paths are indicated with dashed lines

Note. Significant paths are indicated with solid lines; non-significant paths are indicated with dashed lines.

Table 1 Descriptive statistics and bivariate correlations among the variables

Variable	*M*	SD	Range		1	2	3	4	5
1. Cumulative early life adversity	3.93	1.94	1–12	–					
2. Negative emotionality	3.95	1.02	1.42–6.58	0.20**	–		–	–	–
3. Effortful control	5.01	0.82	1.09–7	−0.01	0.19**		–	–	–
4. Day–night stroop slope score	−0.09	0.04	−23–−0.03	0.01	−0.05		0.01	–	–
5. Card sort slope score	−0.02	0.01	−0.04–0.004	−0.08	0.04		0.09	0.21*	–
6. Total psychopathology symptoms	11.23	8.01	0–46	0.35**	0.38**		−0.16**	0.01	0.01

*$p < .05$. **$p < .001$

analysis. In the first step, we observed significant main effects of adversity and effortful control on NE, but no adversity–effortful control interaction. In the second step, we observed significant main effects of adversity, NE, and effortful control on psychopathology and a significant NE–effortful control interaction on psychopathology (Table 2). The conditional indirect effect of adversity on psychopathology through NE was significant at low ($b = 0.39$, SE = 0.10, $\beta = 0.06$, $z = 3.78$, $p < 0.001$) and high ($b = 0.25$, SE = 0.07, $\beta = 0.03$, $z = 3.47$, $p = 0.001$) levels of effortful control. However, simple slope analyses revealed that the direct effect of NE on psychopathology was stronger at low ($b = 3.81$, $t = 10.48$, $p < 0.001$) relative to high ($b = 2.55$, $t = 7.38$, $p < 0.001$) effortful control (Fig. 2). Model fit statistics indicated a generally good fit (CFI = 0.93; GFI = 0.99; RMSEA = 0.11; SRMR = 0.03). Results remained when controlling for potential confounders.

Table 2 Predicting psychopathology from adversity, NE, and cognitive control

Predictors	Effortful control			Executive function		
	b (SE)	*β*	*z*	*b* (SE)	*β*	*z*
Predicting NE						
Adversity	0.13 (0.03)	0.13	4.04**	0.28 (0.05)	0.31	5.85**
Cognitive control	0.22 (0.03)	0.24	6.83**	0.01 (0.05)	0.01	0.20
Adversity × cognitive control	0.02 (0.03)	0.03	0.75	−0.05 (0.05)	−0.05	−0.89
Predicting psychopathology						
Adversity	2.26 (0.20)	0.32	11.23**	2.25 (0.41)	0.26	5.51**
NE	2.41 (0.20)	0.35	11.99**	4.20 (0.47)	0.44	8.88**
Cognitive control	−1.74 (0.21)	−0.27	−8.20**	−0.10 (0.44)	−0.01	−0.23
NE × cognitive control	−0.54 (0.19)	−0.09	−2.83*	0.51 (0.46)	0.06	1.12

*$p < .01$. **$p < .001$

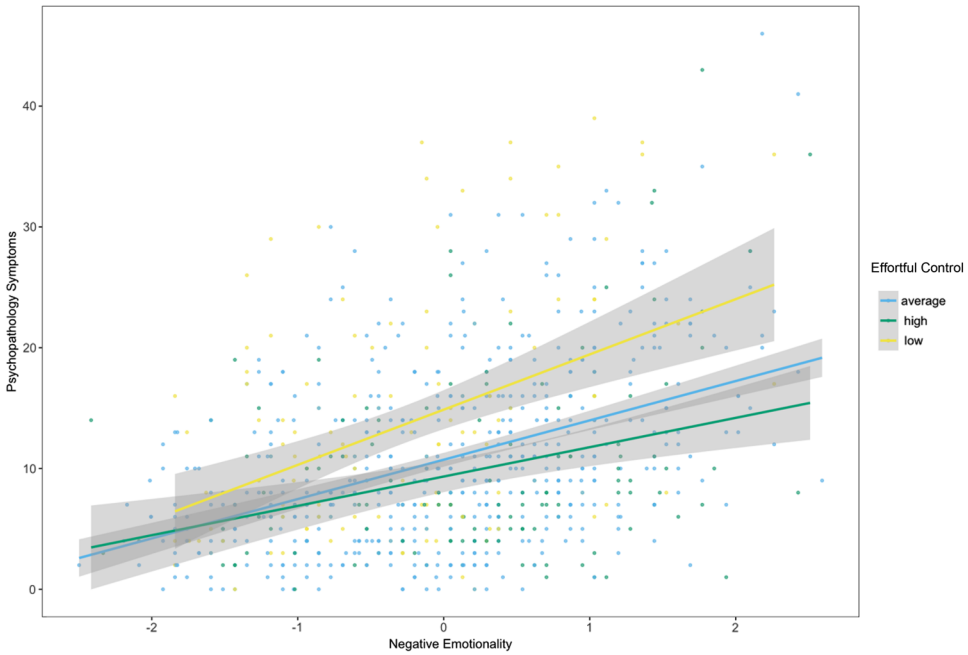

Fig. 2 The effect of negative emotionality on total psychopathology symptoms is moderated by effortful control

Moderation by executive function

We next examined whether the links between adversity, NE, and psychopathology would differ based on children's level of EF in a moderated mediation analysis in the subset of 330 children who completed the laboratory visit. In the first step, we observed a significant main effect of adversity on NE, but no significant main effect of EF or adversity–EF interaction. In the second step, we observed significant main effects of adversity and NE, but no significant main effect of EF or NE–EF interaction (Table 2). The model fit the data well (CFI = 1.00; GFI = 0.99; RMSEA = 0.00; SRMR = 0.01). We re-ran moderated mediation analyses in the subsample with ($n = 168$) and without ($n = 162$) an anxiety diagnosis. In each case, the overall pattern of results was largely the same. Results held when controlling for potential confounders.

Discussion

Theories of adversity suggest that exposure to adverse environments taxes developing emotion and stress response systems, increasing risk for psychopathology [4, 9]. This study builds on past research by examining whether the pathway from adversity to psychopathology is mediated by trait negative emotionality in very young children. Further, this is the first study to our knowledge to test the moderating role of cognitive control in early childhood, which is imperative because this is the period during which effortful control first develops. We observed links between adversity and transdiagnostic psychopathology symptoms in early childhood to be mediated cross-sectionally by emotionality, particularly for children with poorer

effortful control. These findings extend previous literature using a sample that is representative of preschoolers in primary care clinics in the southeastern USA.

Emotionality linking adversity to psychopathology

In the present study, we found that trait NE, the tendency in young children to experience negative emotions and have difficulty being soothed, partially mediated the concurrent link between ELA and symptoms of psychopathology transdiagnostically in young children. Although NE has historically been considered a trait that stabilizes early in childhood [15], past studies suggest that supportive parenting predicts decreases in NE [18]. Our findings add to this literature by suggesting that increased NE may present one mechanism through which adversity increases risk for psychopathology in children. This finding illustrates the power of early adversity to shape risk in periods of peak developmental plasticity and demonstrates that the antecedents of preschool psychopathology are similar to those in later childhood. Exploring how changes in environment (e.g., through intervention) alter trajectories to psychopathology through NE is an important next step in understanding this pathway.

Although this study explored pathways linking total adversity experience to psychopathology outcomes, there may be important differences in the mechanisms through which different experiences of adversity shape psychopathology risk. One prominent model suggests that experiences characterized by the presence of threat (e.g., maltreatment, violence exposure) increase emotional reactivity, while experiences characterized by deprivation (e.g., neglect, lack of cognitive stimulation) inhibit cognitive development [9]. In our sample, threatening experiences more strongly predicted NE (see Supplement) as would be expected from this theory, although future research more thoroughly probing different experiences in young children is needed. Additionally, because our study relied on parent report, we could not measure the child's interpretation of adverse experiences or their perspective on the impact of their experiences. While it is challenging to assess such complex cognitions in preschool-aged children, interpretations of events may provide unique predictive information and are thus worth considering in future research [49–51].

Moderation by cognitive control

Although ELA predicts elevated risk for adverse health outcomes, many youth exposed to adversity develop effective coping strategies protective against the development of psychological disorders. In the present study, we examined whether two metrics of cognitive control, trait effortful control and EF task performance, would moderate the link from adversity to psychopathology through heightened NE in early childhood. In line with past research in older children, results suggest that effortful control moderates the association between higher NE linked to adversity exposure and overall psychopathology. Children with enhanced cognitive control may be better able to regulate heightened emotional reactivity, allowing them to recover from negative affective states more easily, potentially buffering them from psychopathology. This interactive effect was observed despite a somewhat unanticipated small positive correlation between NE and effortful control. While a positive association between reactivity and regulation is potentially counterintuitive, it is possible that in young children, heightened fearfulness and sadness is developmentally appropriate and not reflective of regulatory failure. As both indices were reported by caregivers, it may also be that children's heightened NE led to more internalized distress that was not observed by parents as reflecting dysregulation, and that shared measure variance led to some of the observed overlap. Interestingly, we did not see a similar buffering pattern when we measured cognitive control using task-based measures of EF. It is possible that while temperamental control is well developed even in preschoolers, the multifaceted abilities required to complete EF tasks are just emerging during this stage and not yet able to help children regulate their emotionality. Trait effortful control reflects broader regulatory ability in everyday life, while task-based EF reflects the ability to use top-down control in a controlled task environment, and correlations between these measures tend to be in the low–moderate range [26]. Still the correlation in this study was very low ($rs = 0.01$–0.09, see Table 1) and accuracy on the EF tasks was relatively low (accuracy 66.84–67.96%), suggesting that preschoolers may not be able to leverage self-regulatory ability to complete these tasks. While EF tasks have been used previously to assess EF in a preschoolers, the skills required to successfully complete these tasks are myriad (e.g., following directions, remembering task goals) and task performance may reflect more than just pure EF ability. This hypothesis is supported by the lack of association between this measure and symptoms of psychopathology in this sample (but see [31]). Future research should examine when and in what contexts EF may be protective for ELA-exposed children. There is also a body of literature examining the impacts of ELA on EF which generally finds deleterious effects of ELA [52–54], although recent findings suggest that ELA may be linked with improvements in some dimensions of EF, such as cognitive flexibility [55]. We focused on total cognitive control, which includes the influences of biological and psychological factors, but it is relevant to consider how ELA-linked variability in cognitive control might interact with emotionality to predict psychopathology risk.

Developmental considerations

The present findings align with theories of adversity which posit that exposure very early in life alters emotional response systems in ways that heighten emotional sensitivity across the life span, potentially explaining the greater impact of earlier versus later adversity exposure on psychopathology risk [3]. Importantly, however, the present findings suggest that this risk may be partially mitigated among young children with relatively stronger regulatory ability. These results thus identify a protective factor that may be leveraged by interventions aimed to prevent the development of psychopathology among adversity-exposed young children. Although research on interventions directly targeting cognitive control is mixed, meta-analytic findings suggest that younger children benefit more from these interventions [56]. Further, children with poorer EF tend to benefit more from EF training [57], suggesting such interventions may be especially important among young children with compromised self-regulatory ability as a result of adversity exposure.

Strengths, limitations, and future directions

The present study examined developmental factors shaping risk for psychopathology in preschool. A key strength of this study was the use of a large and representative sample, which allows us to better draw externally valid and reliable conclusions regarding how adversity shapes developmental processes. Additionally, the current study included intensive interview-based measures of adversity and psychopathology. Utilizing a structured interview in which trained staff probed for the context of adversity exposure and ascertained details of symptom presentation, duration, and frequency likely decreased the extent to which results are affected by recall bias. Use of caregiver report is standard in studies of such young children and allowed us to characterize participants' behaviors and experiences based on the knowledge of the person who spends the most time with the child. However, incorporating additional measures, such as observation of child behavior and parent–child relationships could improve our measurement of the constructs used here. While the amount of variance in psychopathology accounted for by the moderated mediation models was reasonably meaningful ($R^2 = 0.28–0.33$), utilizing additional metrics may clarify how impactful these mechanistic factors are in the development of psychopathology in young children. Our study is also limited by its reliance on concurrent assessment of adversity, NE and cognitive control, and psychopathology. While it is important to establish potential pathways linking adversity to outcomes within early childhood, longitudinal studies will be necessary to test mediational pathways through which ELA predicts changes in NE that in turn predict changes in psychopathology risk and to examine multidirectional links between these constructs. Finally, we were only able to assess EF in a subsample ($n = 330$) of the original 917 participants. While this sample is still well powered to examine moderated mediation for medium-to-large effects, it is possibly underpowered to identify a small moderating effect.

Conclusion

The present study explored how ELA shapes developmental processes associated with psychopathology risk in a large representative sample of preschool-aged children. Findings identify elevated NE as one mechanism linking ELA and psychopathology, specifically among children with poorer effortful control. These results represent an initial exploration into the role of heightened emotionality in mediating the adverse effects of ELA in very young children. Further, findings suggest that as in older children, regulatory control may be an important protective factor that can mitigate the effect of heightened reactivity. These results inform our understanding of developmental risks young children experience following adversity exposure and highlight ways to identify and intervene with adversity-exposed youth.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00787-023-02295-5.

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Author contributions MMD and MAS designed research question and analytic plan, MDD, EN, and MAS conducted data analysis, and MMD wrote main manuscript text. WEC, BS, and HLE oversaw data collection and entry. All authors reviewed and contributed to the manuscript.

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Data availability Summary data and analysis code are available from the corresponding author by request.

Declarations

Conflict of interest The authors have no other competing interests to declare that are relevant to the content of this article.

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