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RESEARCH ARTICLE

Neural Sensitivity to Peer Feedback and Depressive Symptoms: Moderation by Executive Function

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ABSTRACT

Theories of adolescent development suggest that elevated neural sensitivity to social evaluation confers tradeoffs for adolescents' wellbeing, promoting adaptation to changing social contexts but increasing risk for emotional distress and depression. This study investigated whether the association between neural processing of peer feedback and depressive symptoms depends on teacher-reported executive function (EF) ability in adolescent girls. Girls showed activation to negative and positive peer feedback in regions implicated in social–emotional processing that interacted with EF to predict depressive symptoms. Specifically, activation predicted more depression in youth with poorer EF but less depression in youth with better EF, suggesting that the impact of increased social sensitivity may depend on youths' ability to regulate this sensitivity in adaptive ways.

1 | Introduction

Adolescence is a period of rapid biological, psychological, and social development during which youth become more independent and learn to navigate an increasingly complex social environment. Although adolescence is an important stage in neural and social growth, it is also a time of elevated risk for depressive symptoms (Rudolph 2009), particularly for girls (Hankin and Abramson 2001). Adolescent girls who are reactive to the effects of peer evaluation may be at especially high risk for depression (Rudolph and Conley 2005). At the same time, adolescence is an important period for the development of executive function (EF), which may allow youth to channel social sensitivity in adaptive ways that promote adjustment in a complex social world. The present study utilized a multi-method approach to explore whether neural sensitivity, as measured by activation during a social feedback task, and EF interact to predict depressive symptoms in adolescent girls. This study extends prior research by examining how in the moment reactivity to realistic negative and positive social cues predicts depressive symptoms and provides a novel perspective on the possible tradeoffs of neural sensitivity that may be contingent on adolescents' self-regulatory ability, as reflected in their EF skills.

1.1 | Social Reorientation Adolescence

Humans possess a fundamental need for belonging and social connectedness that is critical to survival and wellbeing (Baumeister and Leary 1995). These social belonging needs may be

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especially salient during adolescence, a time of heightened peer connectedness, particularly for girls. Social reorientation theories of adolescent development suggest that as youth become more embedded within their social networks, peer evaluation becomes more meaningful and motivating (Nelson et al. 2016). Heightened sensitivity to peer evaluation in adolescence is evident at both the psychological and neural levels. At the psychological level, adolescents report more concerns about peer perceptions, and they ascribe more value to the perspectives of their peers than do children or adults (O'Brien and Bierman 1988; Steinberg and Morris 2001), with girls showing particularly high levels of social-evaluative concerns (Rudolph and Conley 2005). They also place higher importance on fitting in and are more likely than adults to change their opinions and behavior to conform to group expectations (Knoll et al. 2015). At the neural level, adolescents show heightened activation to social feedback in regions associated with threat and reward processing, such as the amygdala, ventral striatum (VS), and ventromedial prefrontal cortex (vmPFC), as well as those involved in salience detection and self-focused thought (e.g., anterior cingulate cortex [ACC], insula, medial prefrontal cortex [mPFC]; Cacioppo et al. 2013; Eisenberger et al. 2003; Masten et al. 2009) and perspective taking (e.g., temporoparietal junction [TPJ]; Schmälzle et al. 2017). In particular, girls compared to boys show stronger increases in activation from childhood to adolescence when anticipating peer evaluation (Guyer et al. 2009).

Heightened sensitivity to social feedback during adolescence is reflected in attunement to both negative and positive cues. With regard to negative evaluation, adolescents report lower mood following rejection than do adults (Sebastian et al. 2010) and show greater amygdala reactivity to faces indicating threat (Casey et al. 2008) than do children. Similarly, in response to rejection, older (relative to younger) adolescents evidence greater activation in regions such as the amygdala, sub-ACC, and mPFC (Gunther Moor et al. 2010; Silk et al. 2013; Vijayakumar et al. 2017). With regard to positive evaluation, adolescents are more willing to take risks to receive positive social feedback than are adults (Gardner and Steinberg 2005) and show greater activity in regions including the amygdala, mPFC, ACC, and insula than children or adults when anticipating (Casey et al. 2008; Silverman et al. 2015) or receiving (Crone and Dahl 2012; Galvan et al. 2006) rewards. Further, adolescents show greater reactivity in the VS and vmPFC when making risky decisions in a simulated driving task in the context of peers but not when alone (Chein et al. 2011), highlighting the role of social monitoring in guiding reward-seeking and reward-sensitivity in adolescents.

1.2 | Tradeoffs of Sensitivity to Social Evaluation

Heightened sensitivity to social evaluation may promote sensitivity to context and confer tradeoffs for adolescents' social and emotional wellbeing. On the one hand, heightened attunement to social evaluation may lead to rumination and negative self-perception and has been proposed as one factor that contributes to elevated risk for depression among adolescents (Silk et al. 2013). On the other hand, social reorientation theories suggest that increased sensitivity to peer feedback allows adolescents to navigate their complex social world and establish supportive peer relationships (Nelson et al. 2016), promoting healthy social engagement (Crone et al. 2020) and social–emotional wellbeing (Telzer 2016). Empirical findings support these tradeoffs, with research suggesting that elevated reactivity to rejection in neural regions involved in salience detection and mentalizing predicts both higher levels of depressive symptoms (Jankowski et al. 2018; Masten et al. 2011; Rudolph et al. 2016; Silk et al. 2013) and more prosocial behavior (Tousignant et al. 2018). Further, elevated striatum reactivity to correctly anticipating positive peer evaluation is associated with more depressive symptoms in adolescents, suggesting that sensitivity to both negative and positive social feedback has implications for adolescent wellbeing (Quarmley et al. 2019).

1.3 | Moderating Role of Executive Function

The role of heightened neural sensitivity to social evaluation in predicting adolescent adjustment may vary depending on youths' ability to channel this sensitivity via top-down regulatory control processes. The present study examined these regulatory abilities in terms of teacher-reported adolescents' EF skills in everyday life. EF, a heterogeneous construct that includes the ability to maintain goals in working memory, shift attention, and inhibit distraction by irrelevant information (Banich 2009; Friedman and Miyake 2017), has consistently been associated with lower levels of depressive symptoms in adolescence (Muris and Ollendick 2005; Snyder 2013). Past research suggests that teacher-reported EF significantly predicts self- and parent-report (Andersen et al. 2024) and EF task performance (Nin et al. 2022; Weiss 2023) and buffers adolescent girls against the depressogenic effects of exposure to peer stressors (Agoston and Rudolph 2016). Further, inhibitory control, a key dimension of EF, moderates the effect of social avoidance goals (conceptualized as the tendency to avoid negative peer evaluation) on depressive symptoms, such that social avoidance predicts more depressive symptoms in girls with low but not high inhibitory control (Rudolph et al. 2013). Additionally, several studies reveal that cognitive control ability moderates the link between negative emotionality, a trait characterized by sensitivity to negative stimuli, and symptoms of depression, such that negative emotionality is more strongly associated with depressive and internalizing symptoms in youth with low relative to high self- and teacher-reported cognitive control (Muris et al. 2007; Rudolph et al. 2017; Vasey et al. 2013). However, high social avoidance (Llewellyn and Rudolph 2014; Rudolph et al. 2013) and psychological and neural sensitivity to social exclusion (Rudolph et al. 2020) predict particularly low levels of depressive symptoms in girls with strong regulatory control and supportive relationships, highlighting the potential benefits afforded by social attunement. Collectively, theory and research suggest that neural sensitivity to social cues may amplify or dampen risk for depression contingent on adolescent girls' EF ability.

1.4 | Overview of the Present Study

The present study sought to examine tradeoffs in the association between heightened neural sensitivity to social feedback and depressive symptoms that are dependent on youths' EF. Neural sensitivity was measured during an ecologically valid social feedback task in which adolescents expressed preferences and

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believed they would receive feedback about whether their preferences conformed to those of peers (Davis et al. 2022; Rudolph et al. 2021). EF was measured via teacher-reported EF difficulties in everyday life. Depressive symptoms were assessed using a semi-structured clinical interview. Gender differences in depression (Hankin and Abramson 2001) emerge during mid-adolescence and have been attributed to a number of biological (Martel 2013), psychological (Charbonneau et al. 2009), and social (Jose and Ratcliffe 2004) factors. Compared to adolescent boys, adolescent girls experience more interpersonal stress (Hankin et al. 2007) and show a stronger link between sensitivity to negative peer evaluation (i.e., social avoidance motivation) and depression (Rudolph et al. 2013). Although girls tend to show better EF than same-age boys (Grissom and Reyes 2019; Weis et al. 2013), the negative consequences of worse EF may be more pronounced in adolescent girls, who are expected to show more maturity and behavioral control than boys (Crick and Zahn-Waxler 2003). Because girls have more exposure and reactivity to peer evaluation and are at greater risk for depression in adolescence, we chose to focus this study on mid-adolescent girls.

2 | Methods

2.1 | Participants and Procedures

Participants were 38 adolescent girls (M_{age} = 17.06, SD = 0.53, range: 15.86–17.73) who completed fMRI tasks and a diagnostic interview in the summer following 10th or 11th grade. Participants were drawn from a longitudinal study in which their teachers reported on their EF when youth were in sixth and seventh grade. We contacted all girls who had completed the final wave of data collection from the longitudinal study and had not previously participated in a neuroimaging study in the lab. Of those contacted, 109 were ineligible (due to having moved away, having braces, being claustrophobic, or having a learning disorder that would inhibit their ability to complete tasks), 32 declined to participate, and 13 were contacted but not scheduled before we reached a sample size of 40. Two participants were excluded from analyses due to excessive movement during the scan or issues with fMRI data collection, leaving a final sample of 38 girls. Girls were from multiple self-identified racial backgrounds (81.58% White, 13.16% African American, 5.26% multi-racial or other) and parent-reported yearly family incomes (13.16% < $30,000; 23.68% $30,000–60,000; 15.79% $60,000–90,000; 44.74% > $90,000; 2.6% unknown). Graduate students and post-baccalaureate research staff administered all procedures and were trained and supervised on the diagnostic interviews by a clinical psychologist. Participants were compensated $50 for the completion of the scan and $25 for the completion of the diagnostic interview. All procedures were approved by the university's Institutional Review Board.

2.2 | Measures

2.2.1 | Social Feedback Task

Adolescents completed a social feedback task (Davis et al. 2022; Rudolph et al. 2021) while undergoing fMRI. This task included 60 trials in which adolescents chose between 2 options from a variety of domains (e.g., extracurricular activities, music genres) and then received feedback ostensibly indicating whether or not other teens who had completed the task agreed or disagreed with their choice. They saw either a thumbs up (indicating that the majority of other teens agreed with their choice), a thumbs down (indicating that the majority disagreed), or a thumb pointing to the side (indicating that roughly half of other teens agreed and half disagreed). In reality, feedback was randomly generated so that adolescents saw 20 trials each of positive (thumbs up), negative (thumbs down), and neutral (sideways thumb) feedback. Feedback remained on the screen for 2 s (Figure 1).

2.2.2 | fMRI Data Acquisition and Analysis

The fMRI data were collected using a 3 Tesla Siemens Trio MRI scanner. The task included T2*-weighted echoplanar images [slice thickness = 3 mm; 38 slices; slice gap = 1.4 mm; TR = 2 s; TE = 25 ms; matrix = 92 × 92; FOV = 230 mm; voxel size 2.5 × 2.5 × 3 mm^3]. Structural scans consisted of a T2-weighted, matched-bandwidth, high-resolution anatomical scan (TR = 4 s; TE = 64 ms; matrix = 192 × 192; FOV = 230; slice thickness = 3 mm; 38 slices) and a T1* magnetization-prepared rapid-acquisition gradient echo (MPRAGE; TR = 1.9 s; TE = 2.3 ms; matrix = 256 × 256; FOV = 230; slice thickness = 1 mm; 192 slices).

The fMRI data were preprocessed using SPM8 (Wellcome Department of Cognitive Neurology). Functional data were spatially realigned to the mean and coregistered to the structural MPRAGE, which was then segmented into cerebrospinal fluid, gray matter, and white matter. Structural and functional images were then transformed into standardized stereotactic space as defined by the Montreal Neurological Institute. Normalized functional data were smoothed using an 8 mm Guassian kernel, full-width-at-half-maximum, and filtered using high-pass temporal filtering with a cutoff of 128 s to remove low-frequency drift in the data.

For each participant's data, a general linear model (GLM) was created using regressors that corresponded to each phase (i.e., *Decision, Feedback Anticipation, Feedback Receipt*) of the task and an unweighted regressor for trials with excessive head motion (over 2.5 mm overall motion). Inter-trial intervals and trials in which participants did not respond were not explicitly modeled and are therefore included in the implicit baseline. The present study focused on activation to feedback receipt based on valence. To model feedback type, separate regressors were created for positive (20 trials), negative (20 trials), and neutral (20 trials) feedback. Parameter estimates resulting from the GLM were then used to create linear contrasts.

2.2.3 | Executive Function

Teachers completed the Behavior Rating Inventory of Executive Function (BRIEF; Gioia et al. 2000) when participants were in sixth (M = 1.25, SD = 0.36; range = 1–2.38; α = 0.97) and seventh (M = 1.20, SD = 0.28; range = 1–2.28; α = 0.96) grades. A subset of 40 items (see Agoston and Rudolph 2016) was selected to assess working memory (e.g., "Has trouble with chores or tasks that have more than one step"), planning (e.g., "Does not plan ahead for school assignments"), shifting (e.g., "Tries the same approach

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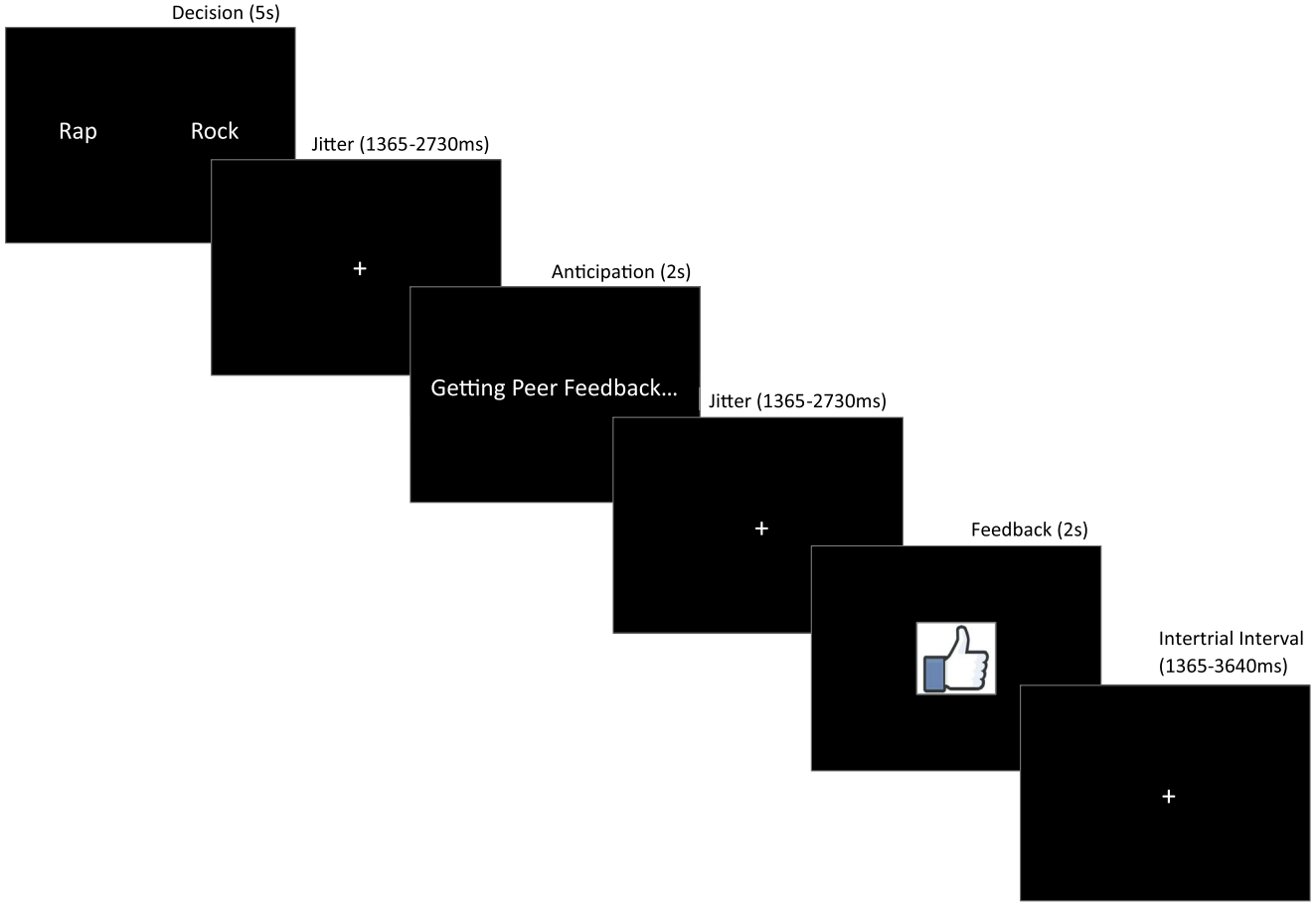

FIGURE 1 | Social feedback task design.

to a problem over and over, even when it does not work"), and inhibition (e.g., "Has trouble putting the brakes on her actions"). BRIEF scores reflect stable EF difficulties in everyday life; scores have high ecological validity (Toplak et al. 2013) and show strong rank order stability (Friedman et al. 2016), internal consistency, and inter-rater reliability (Gioia and Isquith 2004). Construct validity has been supported in clinical and community samples (Gioia et al. 2000). Sixth and seventh grade BRIEF scores were quite stable over time in this sample ($r = 0.64$, $p < 0.001$) and thus were standardized by grade and averaged to create a composite score of EF for each participant.

2.2.4 | Depressive Symptoms

To assess depressive symptoms, participants completed a semi-structured diagnostic interview within 3 weeks of their scan. The diagnostic interview was adapted from the Mini-International Neuropsychiatric Interview for Children and Adolescents (MINI-Kid), a structured clinical interview, to include more detailed follow-up questions regarding symptom description, frequency and duration, and contexts in which symptoms manifested. To reflect continuous variability in symptoms, rating criteria were changed from yes/no to a 0–2 scale (0 = symptom not present, 1 = subthreshold symptom, 2 = clinically significant symptom; for a similar approach, see Agoston and Rudolph 2016). Participants provided information regarding symptoms of several disorders, including major depressive disorder (MDD) and persistent depressive disorder (PDD; formerly dysthymia). Symptoms of MDD and PDD were summed to create a score of overall depressive symptoms. Symptom ratings were assigned through consultation with a clinical psychologist and other trained interviewers. Audiotapes of a quarter of interviews were coded by an independent coder. Inter-rater reliability was high for symptom scores of MDD and PDD (intraclass correlation coefficient = 0.94). We utilized a continuous score in order to capture the full range of variability in depressive symptoms (M = 4.89, SD = 6.22, range = 0–20). However, when considering rates of diagnosis based on the MINI, five girls met DSM criteria for MDD and one met criteria for PDD; an additional eight girls had moderate subthreshold MDD or PDD symptoms (i.e., at least one symptom present at clinically significant levels and moderate impairment). In total, 15.79% of our sample had a depressive disorder diagnosis, and another 21.05% experienced at least moderately impairing subthreshold symptoms, in line with national rates for this age and gender (Substance Abuse and Mental Health Services Administration [SAMHSA] 2020).

2.3 | Analysis Plan

To identify regions showing heightened activation to the receipt of peer feedback, we used whole-brain independent sample *t*-tests to explore patterns of activation in response to receiving negative and positive (vs. neutral) feedback.[1] Monte Carlo simulations using the 3dClustSim tool in AFNI (Cox et al. 2017) were

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used to determine the minimum cluster size necessary in each contrast for a voxel-wise threshold of $p < 0.005$ and a family-wise error rate of $p = 0.05$. To explore how neural activation to feedback receipt interacted with EF scores to explain variability in depressive symptoms, we extracted activation from each region surviving cluster-size correction and entered them into separate hierarchical linear regressions predicting depressive symptoms. Neural activation and EF scores were standardized, and their interaction was calculated by multiplying the standardized terms. Neural activation and EF were entered in the first step of the regressions, and their interaction was entered in the second step. Significant interactions were probed by examining simple slopes across levels (−1 SD, −0.5 SD, mean, 0.5 SD, 1 SD) of EF (Aiken and West 1991; McCabe et al. 2018).[2]

3 | Results

3.1 | Negative Feedback Receipt × EF Predicting Depressive Symptoms

Girls showed more activation to the receipt of negative versus neutral feedback in the mPFC, bilateral inferior frontal gyrus (IFG), and left TPJ, and less activation to negative versus neutral feedback in several frontal and temporal regions, including bilateral dorsolateral PFC and bilateral inferior temporal gyrus, as well as bilateral cerebellum (Table 1). Regression analyses revealed significantly positive left IFG × EF and right IFG × EF interactions and a marginally significant positive left TPJ × EF interaction predicting depressive symptoms (Table 2, Figure 2). Examining simple slopes at 1 SD above and below the mean of EF revealed that more left IFG activation predicted significantly higher depressive symptoms in girls with low EF ($b = 4.68$, 95% confidence interval [CI] = [0.88, 8.48]), but significantly lower depressive symptoms in girls with high EF ($b = -3.09$, CI = [−6.08, −0.1]). Similarly, more right IFG activation predicted significantly higher depressive symptoms in girls with low EF ($b = 3.88$, CI = [1.28, 6.47]), but nonsignificantly lower depressive symptoms in girls with high EF ($b = -1.19$, CI = [−3.90, 1.51]). More left TPJ activation predicted nonsignificantly higher depressive symptoms in girls with low EF ($b = 4.34$, CI = [−0.36, 9.04]) but nonsignificantly lower depressive symptoms in girls with high EF ($b = -1.45$, CI = [−4.20, 1.31]).

3.2 | Positive Feedback Receipt × EF Predicting Depressive Symptoms

Girls showed more activation to the receipt of positive versus neutral feedback in the mPFC, left IFG, and cuneus, and less activation to positive versus neutral feedback in bilateral inferior temporal gyrus and right inferior parietal lobule (Table 3). Regression analyses revealed a significantly positive cuneus × EF interaction and a positive left IFG × EF interaction that did not survive false discovery rate correction (Table 4, Figure 3). Examining simple slopes at 1 SD above and below the mean of EF revealed that more activation in the cuneus predicted significantly higher depressive symptoms in girls with low EF ($b = 3.36$, CI = [0.38, 6.33]) but significantly lower depressive symptoms in girls with high EF ($b = -2.97$, CI = [−5.55, −0.39]).

TABLE 1 | Regions showing significant activation in the negative feedback > neutral feedback contrast.

Region label	*k*	*t*	*x*	*y*	*z*
Activation (negative > neutral)					
mPFC	2131	8.421	−9	62	19
Left IFG (posterior orbitalis)	999	8.851	−30	23	−8
Right IFG (posterior orbitalis)	261	6.401	33	20	−20
Left TPJ	118	4.324	−63	−52	22
Activation (neutral > negative)					
Left inferior temporal gyrus	356	7.240	−51	−61	−5
Right inferior temporal gyrus	619	6.292	54	−58	−11
Right middle occipital gyrus	3229	5.485	36	−73	34
Right ACC	169	5.254	6	20	−2
Left cerebellum	74	4.953	−27	−37	−41
Left middle frontal gyrus	93	4.937	−42	41	28
Right cerebellum	95	4.895	18	−46	−50
Right Rolandic operculum	223	4.725	42	2	19
Left caudate	81	4.443	−18	29	4
Right cerebellum	56	4.351	18	−70	−44
Right dlPFC	97	4.117	36	47	31
Left Rolandic operculum	99	3.597	−60	−7	13

Note: k refers to the number of voxels in each cluster; *t* refers to the peak activation in each cluster; *x*, *y*, and *z* refer to MNI coordinates.

Abbreviations: ACC, anterior cingulate cortex; dlPFC, dorsolateral prefrontal cortex; IFG, inferior frontal gyrus; mPFC, medial prefrontal cortex; TPJ, temporoparietal junction.

4 | Discussion

Sensitivity to cues signaling social status and belonging is key to human survival and is especially elevated among adolescent girls (Rudolph and Conley 2005). Being attuned to social cues may lead to excessive interpersonal concern and is associated with enhanced risk for the development of depression (Silk et al. 2013). However, when directed toward prosocial aims, this sensitivity may promote better adjustment among adolescents. The present study identified patterns of neural reactivity to negative and positive social evaluation in adolescent girls and explored how EF modulated the effect of neural reactivity to feedback on depressive symptoms. These initial results suggest that heightened reactivity to social feedback may be risky among youth with poorer EF but potentially protective among those with better EF.

4.1 | Tradeoffs of Neural Sensitivity to Feedback

In the context of negative (vs. neutral) feedback, girls showed elevated activation in the mPFC, bilateral IFG, and left TPJ.

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TABLE 2 | Neural activation to negative feedback and executive function difficulties predicting depressive symptoms.

	mPFC			Left IFG			Right IFG			Left TPJ		
Predictors	***ß***	***t***	***p* (FDR)**	***ß***	***t***	***p* (FDR)**	***ß***	***t***	***p* (FDR)**	***ß***	***t***	***p (FDR)***
Neural activation	−0.01	−0.07	0.94 (0.94)	0.12	0.73	0.47 (0.63)	0.21	1.40	0.17 (0.46)	0.22	1.23	0.23 (0.46)
EF difficulties	0.37	1.75	0.09 (0.09)	0.55	2.88	0.007 (0.03)	0.33	2.17	0.04 (0.07)	0.31	1.89	0.07 (0.09)
Neural activation × EF	0.27	1.29	0.21 (0.21)	0.53	2.86	0.007 (0.02)	0.41	2.74	0.01 (0.02)	0.36	1.92	0.06 (0.08)

Abbreviations: FDR, false discovery rate corrected *p*-value; IFG, inferior frontal gyrus; mPFC, medial prefrontal cortex; *p*, uncorrected *p*-value; TPJ, temporoparietal junction.

TABLE 3 | Regions showing significant activation in the positive feedback > neutral feedback contrast.

Region label	***k***	***t***	***x***	***y***	***z***
Activation (positive > neutral)					
mPFC	96	3.974	−6	59	22
Left IFG (posterior orbitalis)	80	3.933	−27	17	−17
Right cuneus	51	4.965	21	−97	16
Activation (neutral > positive)					
Right inferior temporal gyrus	400	6.099	54	−61	−11
Right inferior parietal lobule	230	5.076	36	−49	55
Left inferior temporal gyrus	186	4.757	−51	−49	−20

Note: k refers to the number of voxels in each cluster; *t* refers to the peak activation in each cluster; *x*, *y*, and *z* refer to MNI coordinates.
Abbreviations: IFG, inferior frontal gyrus; mPFC, medial prefrontal cortex.

As predicted, results revealed significant bilateral IFG × EF interactions and a marginally significant TPJ × EF interaction predicting depressive symptoms, such that activation to negative feedback in these regions predicted more depression in the context of poorer EF, an effect that was buffered or even reversed in the context of better EF. Our findings build on past research that has found elevated IFG activation during emotion regulation (Frank et al. 2014; Goldin et al. 2008) and elevated TPJ activation in the context of mentalizing and negative peer evaluation (Bolling et al. 2011). The present findings may indicate that greater activation in these regions during negative feedback may be adaptive when coupled with effective EF, perhaps by supporting emotion regulation and perspective-taking that allow youth to flexibly incorporate peer feedback to guide future behavior and avoid social isolation and subsequent depression. In contrast, greater activation in these regions may confer risk for depression in those with poorer regulatory ability by triggering excessive rumination about negative social evaluation. Interestingly, when considering neural activation to positive feedback, we observed a similarly patterned interaction between left IFG activation and EF that did not survive multiple comparison correction. It would be interesting for future studies to consider whether activation in this region to peer evaluation of both negative and positive valence may alter risk for depressive symptoms depending on adolescent girls' regulatory ability.

In the context of positive (vs. neutral) feedback, girls showed elevated activation in the mPFC, left IFG, and cuneus. Further, there was a significant cuneus × EF interaction predicting depressive symptoms, such that activation to positive feedback in this region predicted more depression in the context of poorer EF, but less depression in the context of better EF. Although most research on the cuneus has focused on its role in visual processing, our finding of greater activation to positive (vs. neutral) social feedback builds on a few studies that reveal lower resting state activation (Yuan et al. 2022) and lower activation during recall of happy memories (Foland-Ross et al. 2014) among depressed relative to nondepressed individuals and potentially implicates atypical cuneus responsivity to reward in depression risk.

4.2 | Contributions, Limitations, and Future Directions

This study makes several novel contributions. Past studies suggest that variability in reactivity to social cues may be a marker of risk for psychopathology (e.g., Caouette and Guyer 2014; Jankowski et al. 2018). However, these studies reveal mixed and sometimes contradictory results, with some linking depression to hyper-reactivity (Rudolph et al. 2016) and others finding evidence of hypo-reactivity (Forbes et al. 2010; Spielberg et al. 2014) in the same regions. Yet other research suggests that sensitivity to social cues reflects differential susceptibility to context that may confer both advantages and disadvantages contingent on the social environment in which adolescents live (Rudolph et al. 2020). To further explore these potential tradeoffs of social sensitivity, this study examined whether the implications of neural activation to social threat and reward might differ as a result of EF. Findings suggest that the impact of activation across several social–emotional processing regions on depressive symptoms depends on self-regulatory ability. This novel finding adds to theory and prior research by highlighting the importance of considering neural activation to social evaluation within the context of youths' ability to regulate this sensitivity. At the same time, there are likely multidirectional associations between neural sensitivity, EF, and depression, and further longitudinal research is needed to elucidate transactions between sensitivity to feedback, regulatory control, and depressive symptoms across development in larger samples.

A key strength of this study was the use of a multi-method, multi-informant design that included neural, informant-report, and interview-based measures. This study leveraged a neuroimaging paradigm to examine in the moment reactivity to the receipt of

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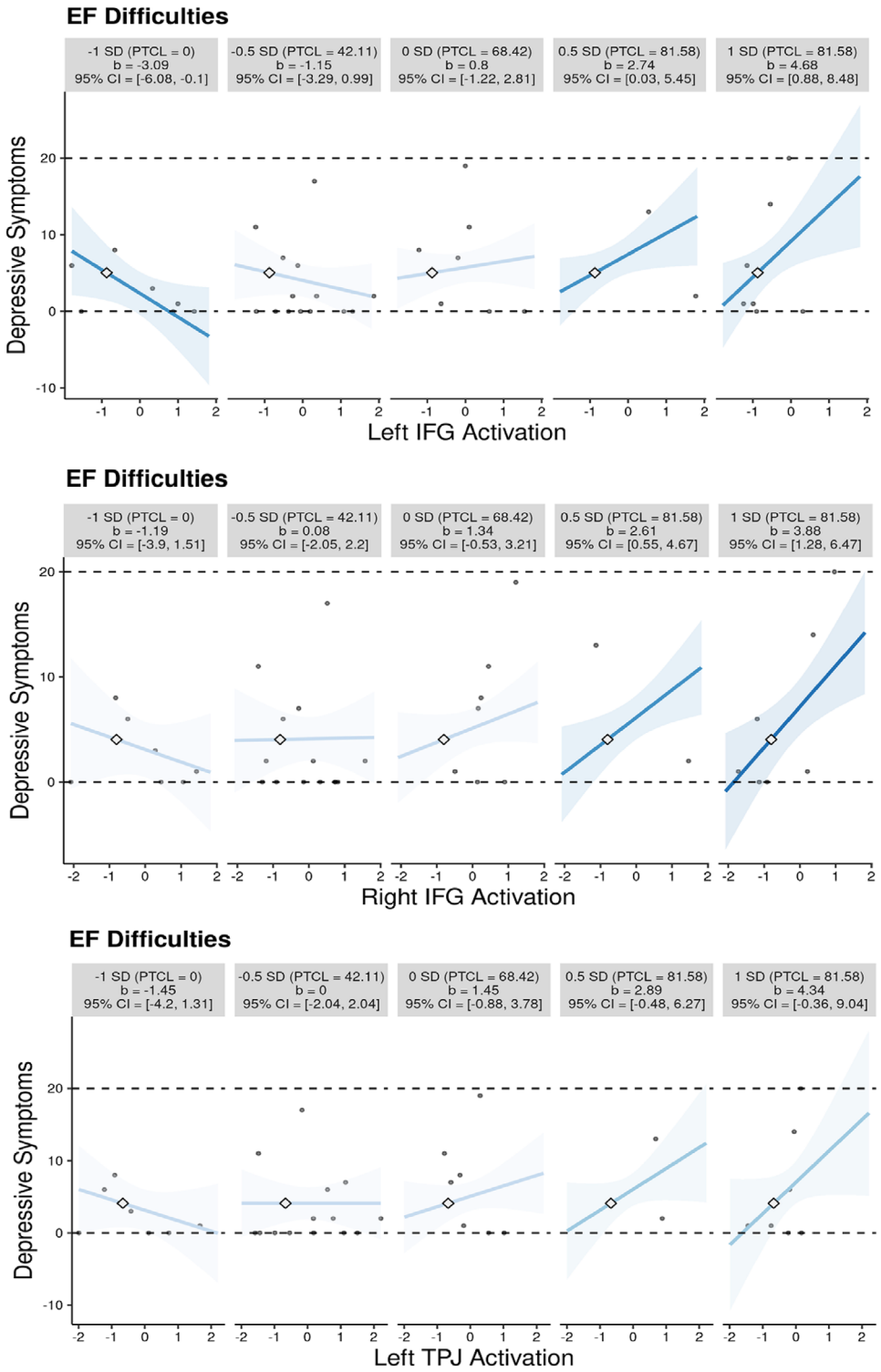

FIGURE 2 | Neural activation to negative (vs. neutral) feedback and executive function difficulties predicting depressive symptoms. CI, confidence interval; EF, executive function; IFG, inferior frontal gyrus; PTCL, percentile; SD, standard deviation; TPJ, temporoparietal junction.

peer feedback similar to that adolescents encounter and react to in person and online in their daily lives (Magis-Weinberg et al. 2021). The design allowed us to explore sensitivity to both negative and positive feedback within the same task and with minimal deception, contributing to a literature base that has largely focused on the role of threat sensitivity in the development of depression. Interestingly, our findings suggest that activation to negative and positive peer feedback may function similarly in adolescent girls, promoting sensitivity to social cues that may shape risk for, or protection against, depressive symptoms depending on youths' ability to channel this sensitivity. EF was assessed via teacher-report over the course of 2 years, providing an indicator that is more reliable and taps into girls' EF in daily life situations during which they may encounter peer stressors. Finally, the semi-structured interview allowed us to assess depressive symptoms in a way that is less influenced by current mood than

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TABLE 4 | Neural activation to positive feedback and executive function difficulties predicting depressive symptoms.

	mPFC			Left IFG			Cuneus		
Predictors	***ß***	***t***	***p* (FDR)**	***ß***	***t***	***p* (FDR)**	***ß***	***t***	***p* (FDR)**
Neural activation	−0.13	−0.77	0.45 (0.67)	−0.16	−1.01	0.32 (0.67)	0.02	0.13	0.90 (0.90)
EF difficulties	0.22	1.33	0.19 (0.19)	0.24	1.50	0.14 (0.19)	0.47	2.68	0.01 (0.03)
Neural activation × EF	0.14	0.79	0.43 (0.43)	0.28	1.76	0.09 (0.13)	0.54	3.31	0.002 (0.006)

Abbreviations: FDR, false discovery rate corrected *p*-value; IFG, inferior frontal gyrus; mPFC, medial prefrontal cortex; *p*, uncorrected *p*-value.

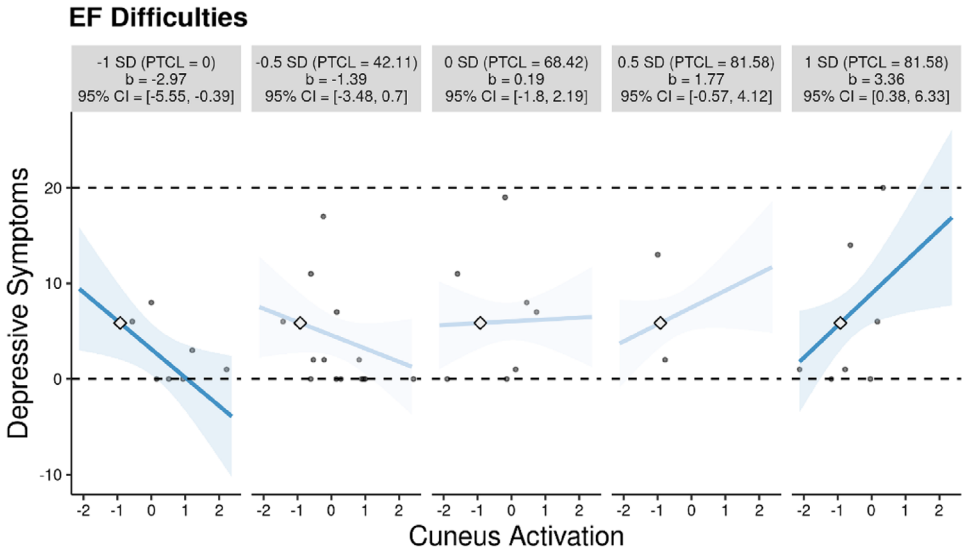

FIGURE 3 | Neural activation to positive (vs. neutral) feedback and executive function difficulties predicting depressive symptoms. CI, confidence interval; EF, executive function; PTCL, percentile; SD, standard deviation.

self-report measures (Summerfeldt et al. 2020). Using neuroimaging tasks and interview measures allowed us to avoid effects of reporting bias, although it will be important to examine overlap with other measures of social sensitivity, cognitive control, and depression.

Despite these strengths, there are limitations of the present study. Notable among these is the fact that the sample was small, and future studies that replicate this pattern of findings in a larger group of youth are essential. Additionally, we focused on girls because they tend to experience more social stress (Hankin et al. 2007) and stress-linked depression risk (Rudolph et al. 2013) than boys, despite showing better EF than same-age boys (Grissom and Reyes 2019). However, adolescence is also a period of heightened peer importance and social stress for boys (Östberg et al. 2018) and gender minority youth (Reisner et al. 2015), and research on these samples is vital for understanding how the impact of social sensitivity and EF may depend on gender socialization during this stage.

In this study, EF was assessed via a teacher-report measure completed approximately 3 years before neural processing of social feedback and depressive symptoms, which were assessed concurrently. Although EF tends to be stable from childhood into adolescence (Ahmed et al. 2019; Snyder 2013) and adulthood (Casey et al. 2011; Friedman et al. 2016) and was stable across the 2 years it was assessed in this study, a more proximal measure of EF could better capture girls' regulatory ability during the mid-adolescent period of interest. Moreover, although teacher-reported EF is associated with reports from other informants (Andersen et al. 2024) and task-based measures (Nin et al. 2022; Weiss 2023), it will be important to examine self- and other-report and task-based measures of EF in the same study, as each could provide unique predictive value (Friedman and Gustavson 2022; Toplak et al. 2013).

With regard to study design, the use of concurrent measures of neural sensitivity and depression limits our ability to draw conclusions about predictive associations. Sensitivity to social feedback likely develops as a result of learning and past experience and may be shaped by self-regulatory ability, highlighting the importance of longitudinal measurement of these constructs. Exploring how associations among neural processing of social feedback, EF, and depression risk shift across development will inform models of neurobiological development and clarify sensitive periods during which the regulation of social sensitivity may be particularly impactful. Despite using a strong control of neutral feedback, it is possible that adolescents differ in their perception of neutral feedback; this would be worth probing in future research. Finally, although we found that EF may buffer against depressogenic

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effects of heightened neural sensitivity to valenced social evaluation, future studies that include positive outcomes, such as social connectedness or quality of life, would additionally clarify whether sensitivity may promote positive adjustment in youth with higher EF and are essential to fully probe the potential tradeoffs of elevated social sensitivity in adolescence.

5 | Conclusion

This study sought to examine how neural sensitivity to social evaluation confers tradeoffs for adolescent girls' wellbeing. Findings suggest partially overlapping patterns of neural activation to negative and positive peer feedback that augment or diminish risk for depressive symptoms contingent upon EF ability. This study is the first to examine how the effect of neural responses to social cues may differ as a function of regulatory control, highlighting the role of neural sensitivity to social feedback as a susceptibility factor that can function as a risk *or* protective factor depending on youths' other tendencies. Results further indicate that bolstering EF may represent one way to channel adolescents' social attunement to enhance social–emotional adjustment and promote wellbeing during this critical developmental stage.

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Ethics Statement

This study was conducted in accordance with the ethical standards of the American Psychological Association and was approved by the Institutional Review Board of the University of Illinois at Urbana-Champaign. Caregivers provided written consent, and adolescents provided written assent prior to study participation.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Endnotes

[1] We also examined activation to negative and positive (vs. neutral) feedback as part of a larger study from which this sample was drawn; these results have previously been reported (Davis et al., 2022). In the present study, we examined activation specifically in the subset of participants who had BRIEF EF data in order to explore interactive effects of activation and EF on depressive symptoms.

[2] In order to control for multiple comparisons of activation drawn from the same contrast (negative vs. neutral feedback or positive vs. neutral feedback), we also calculated false discovery rate corrected p-values for all coefficients (presented in Tables 2 and 4).

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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

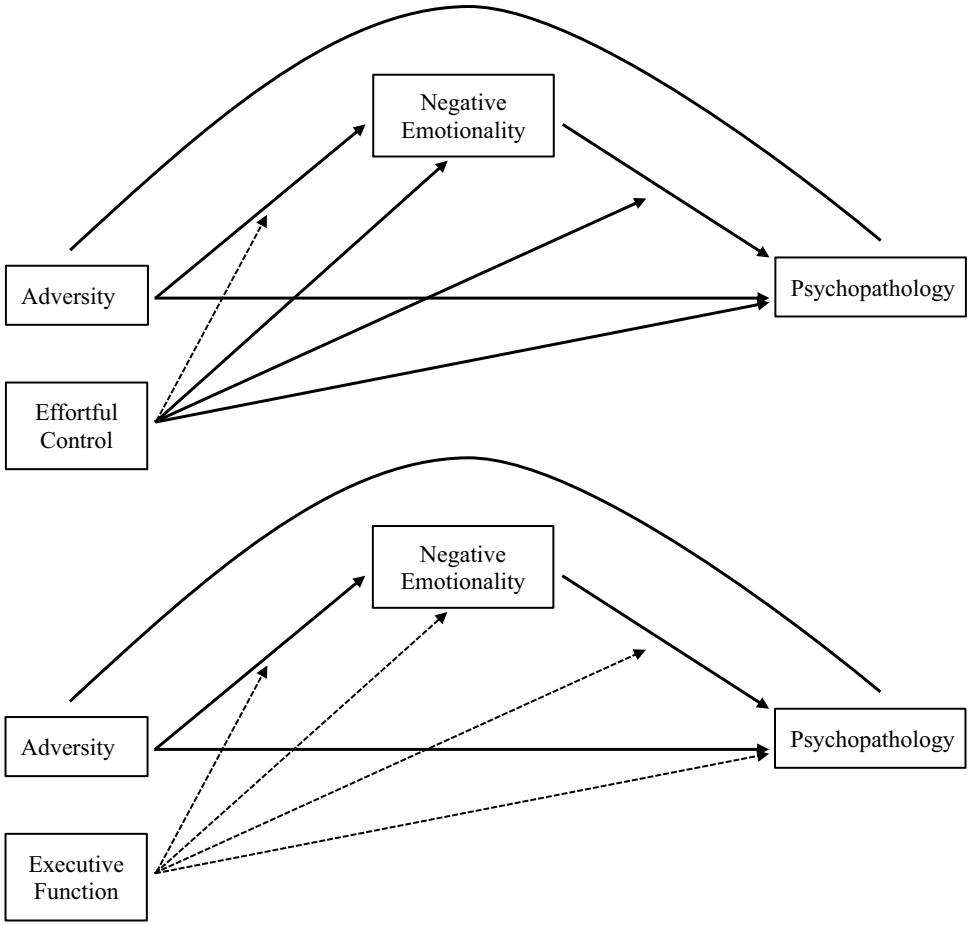

Fig. 1 Pathway from early life adversity to total psychopathology symptoms is mediated by negative emotionality and moderated by effortful control (top), but not executive function (bottom). Significant paths are indicated with solid lines; non-significant paths are indicated with dashed lines

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

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

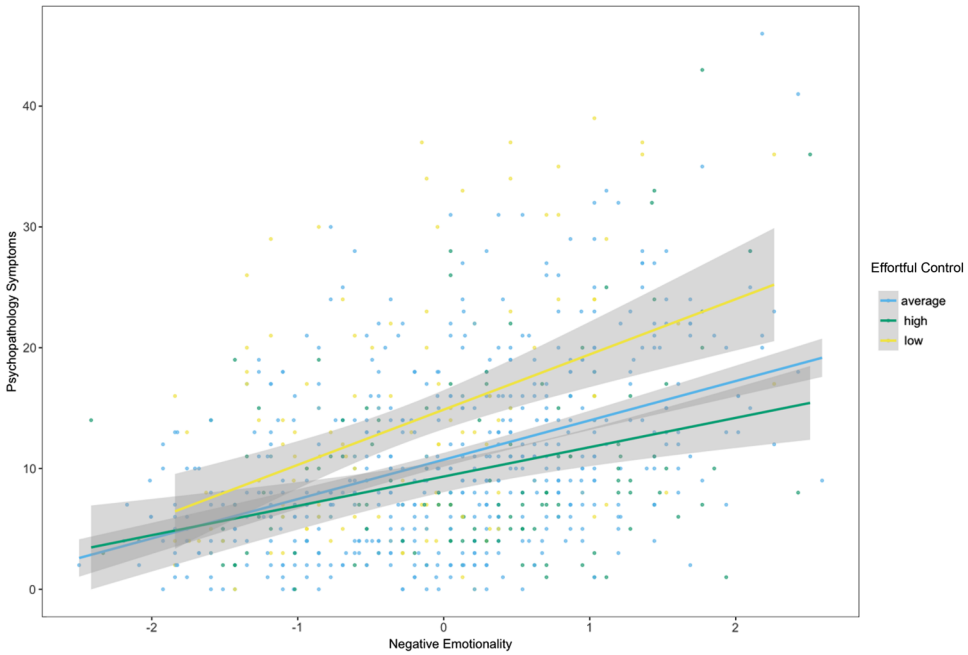

Fig. 2 The effect of negative emotionality on total psychopathology symptoms is moderated by effortful control

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.

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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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