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Special Issue Article

The Future of Developmental Psychopathology: Honoring the Contributions of Dante Cicchetti

The pernicious role of stress on intergenerational continuity of psychopathology

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Abstract

Development and Psychopathology has been a premier resource for understanding stressful childhood experiences and the intergenerational continuity of psychopathology. Building on that tradition, we examined the unique and joint influences of maternal stress on children's effortful control (age 7) and externalizing behavior (age 11) as transmitted via genetics, the prenatal environment, and the postnatal environment. The sample included $N = 561$ adopted children and their biological and adoptive parents. Path models identified a direct effect of biological mother life stress on children's effortful control ($\beta = -.08$) and an indirect effect of her life stress on child externalizing behavior via effortful control ($\beta = .52$), but no main or indirect effects of biological parent psychopathology, prenatal stress, or adoptive mother adverse childhood experiences (ACES). Adoptive mother ACES amplified the association between biological mother life stress and child effortful control ($\beta = -.08$), externalizing behavior ($\beta = 1.41$), and the indirect effect via effortful control, strengthening associations when adoptive mothers reported average or high ACES during their own childhoods. Results suggest that novel study designs are needed to enhance the understanding of how life stress gets "under the skin" to affect psychopathology in the offspring of adults who have experienced stress.

Keywords: adoption study; externalizing; genetic; intergenerational; stress

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Under the editorial leadership of Dante Cicchetti, this journal has been a premier resource for information about the psychological and biological consequences of interpersonal and economic stress on development and psychopathology (e.g., Cicchetti & Walker, 2001). Over the past 40 years, Cicchetti and colleagues have made groundbreaking advances regarding the consequences of stress, documenting pathways of intergenerational transmission and demonstrating the cumulative effects of child maltreatment on psychopathology and neurocognitive development and the importance of multi-level analytic approaches (e.g., Cicchetti & Rogosch, 2001; Cicchetti, 1984; Cicchetti & Toth, 2016; Cowell et al., 2015). Using a novel prospective parent-offspring research design, the current study honors and builds upon the foundational work that Cicchetti established for the field of development and psychopathology to longitudinally examine intergenerational pathways underlying stress-psychopathology associations. Drawing from the work of Cicchetti and others (e.g., Bowers & Yehuda, 2016), in this manuscript we define maternal "stress" as inclusive of stressful or traumatic interpersonal experiences (e.g., adverse family relationships, including maltreatment or living with parents with psychopathology or substance misuse) and stressful economic experiences (e.g., not having enough to eat, clothing, or housing). Intergenerational transmission of stress is measured in this manuscript as associations between interpersonal or economic stress experienced in one generation and neurocognitive development (effortful control) and externalizing outcomes in the next generation.

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Intergenerational transmission of stress

Stress can induce enduring and widespread effects in an individual long after the stressor is removed via epigenetic, neuroendocrine, or neuroanatomical mechanisms (Yehuda & Lehrner, 2018). Moreover, stress not only affects an individual throughout their own lifetime but can affect the well-being of subsequent generations. Multiple studies have shown that interpersonal or economic stress in one generation can compromise the well-being of their biological children decades later (Folger et al., 2017; Hammen et al., 2012; Lünnemann et al., 2019; McEwen & McEwen, 2017; Roseboom et al., 2006). These human-based research findings build upon non-human animal studies that have

mechanistically documented the negative impacts of parental stress on offspring behavior and health (e.g., Sproul Bassett et al., 2020; Suomi & Levine, 1998), underscoring the urgency for understanding the mechanisms that underlie cross-generational transmission of stress in human populations. The study of intergenerational transmission of stress is especially timely given findings from a recent poll, indicating that 27% of adults reported that most days they are so stressed they cannot function, and close to 76% of respondents reported experiencing health impacts due to stress in the prior month (https://www.apa.org/news/press/releases/stress/2022/concerned-future-inflation). What are the long-term consequences for children when significant portions of adults report stressful lives? This is an urgent question for the field to address with an increased pace; here we provide one example of a study that provides one window of insight into this question.

Explanations for intergenerational associations between exposure to stress in one generation and psychopathology in offspring have been attributed to postnatal caregiving environments, genetics, and intrauterine effects that impact the stress physiology of the developing fetus (Bowers & Yehuda, 2016; McEwen & McEwen, 2017). Stress exposure in each of these three pathways is examined in the current study, with a synopsis of the research underpinning each pathway's connection to child psychopathology described below.

Postnatal caregiving environment: rearing parents' own exposure to stress in childhood

Rearing parents' own childhood stress and trauma is one potential mechanism of intergenerational transmission, with documented associations with psychopathology in offspring. For example, higher total problems and greater odds of clinical problems were noted in children whose parents reported that they had experienced childhood trauma themselves (e.g., physical abuse before age 18 years, or sexual abuse before 13 years) relative to parents who did not report trauma (Bush et al., 2023). Much of the recent research in this area has examined parental exposure to stress via adverse childhood experiences, often measured with the Adverse Childhood Experiences (ACES) questionnaire (Felitti et al., 1998). A recent scoping review of parental ACES identified 68 studies that focused on the association between parental ACES and their child's outcomes, identifying consistent associations between parental ACES and outcomes such as child externalizing behavior (Zhang et al., 2023). For example, in a large panel study included in the review, children of parents with a history of four or more ACES (versus three or fewer ACES) had more behavior problems and higher odds of a diagnosis of attention deficit hyperactivity disorder (Schickedanz et al., 2018). A limitation of this body of research is that associations between rearing parents' ACES and their child's psychopathology could also result from shared genes or from prenatal exposures shared by the mother and child. This limitation is addressed by the current parent-offspring adoption study and discussed in more detail in the sections that follow.

Heritable aspects of stress-psychopathology linkages

Twin studies show that in addition to rearing environmental transmission mechanisms noted in the prior section, there are also heritable contributions to stress exposure, and to its association with psychopathology (see Afifi et al., 2010, for a review). For example, within a sample of adult twins and their siblings drawn from a large population-based sample selected based on childhood sexual abuse and physical abuse, the researchers found that a substantial portion (47%–60%) of the variance in recalled trauma exposure was attributable to genetic factors, suggesting a heritable aspect of trauma (Sartor et al., 2012). Further, the genetic covariance between high-risk trauma exposure and major depressive disorder was strong ($r = 0.89$), indicating that the genetic factors that contribute to high-risk trauma exposure also contribute to depression. By extension, genetic influences that lead to stress exposure in one generation may manifest as depression in their biological offspring (and vice versa), due to the shared genes associated with both stress exposure and depression. However, to our knowledge, such intergenerational work has not yet been conducted – a gap the current study attempts to fill.

Prenatal stress as a potential pathway for intergenerational transmission

There is also a growing body of research on associations between maternal stress during pregnancy and psychopathology in offspring (Bowers & Yehuda, 2016). For example, a study of nearly 2000 mother-child dyads showed that pregnancy stressful life events were associated with greater odds of clinical levels of problems in 4–6 year old offspring (Bush et al., 2023). In a related study, Ahmad et al. (2022) found that stressful life events during pregnancy were independently associated with both child executive functioning problems and externalizing problems in children ages 4–6 years old. In one of the largest prospective studies of the association between prenatal stress and child psychopathology to date ($n = 10{,}184$ mother–offspring pairs), mothers were asked about prenatal stress life events at 18-weeks gestation (MacKinnon et al., 2018). Subsequently, they rated their child's behavioral symptoms at multiple timepoints across childhood (age 6–16 years). Children whose mothers scored in the highest quartile of prenatal stress were more likely to belong to the high symptom trajectory for conduct disorder. Although the mechanisms whereby prenatal stress is connected to offspring psychopathology remain under investigation, research suggests that the prenatal uterine environment is a likely source of transmission via fetal programing (e.g., Conradt et al., 2018).

Stress transmission pathways in women

Stress-psychopathology linkages may be especially pronounced in females. For example, analyses using the National Comorbidity Survey Replication–Adolescent Supplement study indicated that interpersonal trauma was associated with externalizing problems in females but not males (Carliner et al., 2017). Similarly, a separate study found that adolescent externalizing symptoms were associated with exposure to violence and with future post-traumatic stress disorder symptoms in females only (Haller & Chassin, 2012).

Considered together with the aforementioned twin studies, this work underscores stress-externalizing behavior linkages in women, with evidence of a shared genetic etiology of the two constructs. This the possibility that stress experienced by biological mothers may be a reliable indicator of psychopathology in offspring, in part via genetic transmission pathways.

Study design limitations in prior research on intergenerational transmission of stress

As summarized above, explanations for intergenerational associations between exposure to stress in one generation and psychopathology in offspring have been attributed to maternal ACES, genetics, and intrauterine effects that impact the stress

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physiology of the developing fetus (Bowers & Yehuda, 2016; McEwen & McEwen, 2017). However, there are methodological limitations to our understanding of how these three potential sources of intergenerational continuity operate separately and together. These limitations are partly because most of the prior research has focused on children reared with their biological parents. In such designs, the same individual (the biological mother) is a potential source of intergenerational transmission to the child be it via genetic influences or via the prenatal and postnatal environment that she provides to the child. To address these limitations, the current study uses data from a prospective adoption study of children who were placed with adoptive families around the time of birth to advance the understanding of intergenerational transmission of stress effects on child externalizing behavior. In this type of design, because data from the child's adoptive parents (who provide the child's rearing environment) and their biological mother (who provides genetic material and the prenatal environment) are both included, we can gain a unique perspective on the intergenerational transmission of stress and how its effects may get "under the skin" to influence offspring psychopathology via biological and environmental pathways and their interplay.

Effortful control as an early mechanism linking parental stress with externalizing behavior in offspring

We can also use the prospective parent-offspring study design to examine mediators of associations between maternal stress exposure and children's later externalizing problems. We focus this manuscript on children's effortful control as a plausible mechanism because it is heritable (e.g., Ganiban et al., 2021), is predicted by parental interpersonal and economic stress (Lee et al., 2019; Taylor et al., 2018), and is associated with subsequent child externalizing problems in childhood and adolescence (e.g., Yang et al., 2022). For example, specific to parental stress and children's effortful control, a study of war veterans and non-veterans found that the children of war veterans performed more poorly in late adolescence on an effortful control task that contained war-related words compared to the children of non-veterans (Motta et al., 1997). This finding suggests that parents' own traumatic experiences may be transmitted to their children by impacting their child's effortful control. As such, it is plausible that effortful control could serve as one step in the pathway from parental stress to children's externalizing behavior.

The current study

Using data from a prospective parent-offspring adoption design, we examined whether and how stress experienced by one generation affected the externalizing behavior in their children, often decades later. This type of adoption design is uniquely suited to disentangle intergenerational stress influences originating from the rearing environment from influences resulting from genetic and/or prenatal sources of stress because the children are raised by adoptive parents who are not their biological relatives. Moreover, as the children in this study were placed with adoptive families within a few weeks after birth and the biological parents did not have a role in rearing the child, associations between the adoptee and their biological parent are assumed to be due to genetic, prenatal, and/or other biological influences. We focus on the intergenerational outcome of child externalizing problems measured at child age 11, while considering children's effortful control at age 7 as a potential mediator of intergenerational transmission effects. We measure interpersonal and economic stress in biological mothers to form our genetic indicator, recalled ACES (which includes interpersonal and economic stress) in adoptive mothers to form our rearing environment measure, and prenatal stress in biological mothers to form our prenatal variable.

All measures of biological and adoptive mother stress in the current study assessed stress exposure before the birth of the child and/or external to the child's postnatal rearing environment. This approach was intentional to better understand how parental stress experiences *outside of the parenting context* may affect a child's risk for externalizing behavior. Conceptually, this approach fills a gap in the field by distinguishing stressful events that the offspring did not directly experience (e.g., events that occurred in the lives of the rearing parents before the child was born or in the lives of the biological parents outside of the pregnancy period), from events experienced specifically during the prenatal period.

Using this design, we aimed to advance the understanding of how stress gets "under the skin" to influence the intergenerational transmission of psychopathology by testing three study hypotheses. First, we expected that adoptive mothers' recalled ACES, biological mothers' life stress, and biological mothers' stress experienced during pregnancy would each contribute unique variance to children's externalizing behavior at child age 11. By including all three predictors, stress transmitted via the child's rearing environment, genetics, and the prenatal environment can each be examined. Second, based on prior research linking genetic, prenatal, and postnatal environmental pathways of exposure to parental stress to offspring effortful control, we hypothesized that any associations identified in hypothesis 1 would be mediated by children's effortful control at age 7. Third, we hypothesized that the effects of one source of maternal stress would amplify the effects of the other sources of maternal stress, such that the effects of biological mother stress during her own lifetime or specific to the pregnancy period would be amplified by adoptive mothers' ACES experienced during her own childhood.

Method

Participants

Study participants included 561 linked sets of adopted children and their adoptive and biological parents. Participants were drawn from the Early Growth and Development Study (EGDS; Leve et al., 2019). Recruitment into EGDS occurred from 2003 to 2010 through 45 adoption agencies across the United States. Eligibility criteria were (a) domestic adoption placement, (b) adoption within the first 3 months of birth ($M = 5.58$ days, $SD = 11.32$ days), (c) adoption placement with a nonrelative, (d) birth and adoptive parent reading level of at least eighth grade, and (e) no major infant medical conditions. The adoptee and their adoptive and biological parents were recruited in infancy and followed longitudinally. Data collected during infancy, middle childhood, and adolescence are included in the current study. Approximately half of the children (57%) are male assigned at birth ($n = 321$ males, $n = 240$ females). Most children are White (55.3%), 19.6% are multiracial, 13.2% are Black or African American, 10.9% are Hispanic or Latinx, < 1% are Asian, < 1% are Native Hawaiian or Pacific Islander, < 1% are American Indian, and < 1% are of unknown ethnicity/not reported.

At study enrollment (infancy), the median total household income for adoptive families was over $100,000, and the median educational attainment was at least a 4-year college degree for both adoptive mothers and fathers. Most adoptive mothers were

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non-Latine White (91.8%); others were Black/African American 3.9%, Hispanic/Latine 2.0%, Multiethnic 0.9%, or Other 1.4%. Most adoptive fathers were also non-Latine White (90.4%); others were Black/African American 4.9%, Hispanic/Latine 1.6%, Multiethnic 1.1%, or Other 2.0%. Adoptive parents' mean age at the time of adoptee birth was 37.4 years ($SD = 5.6$ years) for adoptive mothers and 38.3 years ($SD = 5.8$ years) for adoptive fathers.

For biological mothers, the median total household income at the time of study enrollment was less than $15,000 and the median educational attainment was at least a high school degree. In approximately one-third of the families (37.4%), the child's biological father was identified and consented to participate in the study. For participating biological fathers, the median total household income at the time of the adoptee's birth was between $15,000 and $25,000 and the median educational attainment was at least a high school degree. Most biological mothers and biological fathers were non-Latine White (70.1% and 69.9%, respectively); others were Black/African American 13.3% and 11.5%, Hispanic/Latine 6.7% and 9.6%, Multiethnic 4.9% and 4.8%, or Other 5.0% and 4.2%, respectively. Biological mothers averaged 24.4 years old ($SD = 6.0$ years) and biological fathers averaged 26.1 years old ($SD = 7.8$ years) at the time of the child's birth.

Procedure

Participants were assessed with in-person, web-based, and/or phone interviews from infancy to adolescence. For the current study, we used data collected between 3 and 18-months postpartum from the biological parents (T1), and from child age 6–7 years old (T2; $M_{age} = 6.79$ years, $SD = 0.48$) and child age 11 years old (T3; $M_{age} = 11.40$ years, $SD = 0.54$) from the adoptive parents. In addition, the retrospective ACES data were collected from biological and adoptive mothers about their own childhoods approximately 15 years after the adoptee was born. All research activities were approved by the institutional review boards of the participating institutions. All adult participants consented to participation in the research activities and children who were 7 years and older provided assent.

Measures

Adoptive mother recalled adverse childhood experiences (ACES)

Adoptive mothers retrospectively reported on their own adverse childhood experiences using the Adverse Childhood Experiences (ACES) questionnaire (Felitti et al., 1998). They self-reported whether they had experienced 10 types of ACES comprising emotional, physical, and sexual abuse, emotional and physical neglect, and familial dysfunction during their own childhood (before age 18). Responses were binary (0 = did not occur; 1 = did occur) and summed to produce a total ACES score with a possible range of 0–10, with higher scores indicating more adverse childhood experiences. Inter-item reliability in this sample was acceptable ($\alpha = .64$). Means and standard deviations for all measures are included in Table 1.

Biological mother prenatal stress (T1 measure)

Biological mother prenatal stress was assessed as a standardized mean composite of prenatal stressors occurring during pregnancy as coded from medical records, and retrospective report of anxiety symptoms experienced during pregnancy. For the former, prenatal care and delivery medical records were coded for endorsement of 16 stressors and traumatic events experienced by the biological mother during pregnancy (e.g., stressful living conditions, loss of family member or close friend, physical abuse). Codes were binary (0 = did not occur; 1 = did occur) and summed to produce a total prenatal stressor score with a possible range of 0–16. Inter-item reliability in this sample was acceptable ($\alpha = .71$). Pregnancy anxiety symptoms were assessed with a subset of items from the Beck Anxiety Inventory (BAI; Beck et al., 1988). Biological mothers indicated whether they experienced worry during their pregnancy (0 = no; 1 = yes) and reported on the severity of 4 symptoms (e.g., heart pounding) using a 4-point Likert scale (0 = not at all, 3 = severely). Responses were summed to produce a total prenatal anxiety score with a possible range of 0–13. The inter-item reliability for the BAI symptoms scores in this sample was acceptable ($\alpha = .78$). Scores on the prenatal stressors sum and the BAI pregnancy anxiety symptoms were correlated ($r = .15$, $p = .003$); the two measures were standardized and a mean composite used in analyses.

Biological mother life stress (T1 measure)

We created a composite measure of biological mother life stress that was designed to capture trauma and life stress that biological mothers experienced during their own childhood (before age 18) and again after placement of the adoptee. Accordingly, any associations identified between this composite measure and the adoptee's psychopathology would include biological transmission mechanisms passed from mother to child via genetic (including epigenetic) mechanisms and would not be indicative of postnatal or prenatal exposures for the child. The composite measure included three measures collected at 18-months postpartum (material needs, household income below the U.S. federal poverty level, and negative life events) and one measure regarding retrospective self-report of ACES collected approximately 15 years post-partum. Material needs were assessed with a 6-item subscale of the Financial Satisfaction Questionnaire (Conger et al., 1992; 1994), indicating whether the participant had the financial resources to meet their needs. The scale includes 6 items (e.g., having enough money to afford food) rated on a 5-point Likert scale, where higher scores indicated greater neediness. The material needs subscale had acceptable inter-item reliability in this sample ($\alpha = .87$). Participants self-reported their total household income, which was subsequently binary coded (0 = falls above, 1 = falls below) to indicate whether they were living below the U. S. federal poverty level based on household size and composition at the time of data collection. Negative life events were assessed using a standard checklist (Dohrenwend et al., 1978), where participants indicated whether they experienced 32 events deemed stressful or deleterious (e.g., a close friend or family member passing) in the past year. Responses were binary (0 = did not occur; 1 = did occur) and summed to produce a negative life events total score with a possible range of 0–32. Inter-item reliability in this sample was acceptable ($\alpha = .75$). Parallel to the adoptive mother ACES measurement, biological mothers responded to the Adverse Childhood Experiences questionnaire (Felitti et al., 1998) and indicated retrospecively whether they experienced 10 types of ACEs comprising emotional, physical, and sexual abuse, emotional and physical neglect, and familial dysfunction during their own childhood (before age 18). Responses were binary (0 = did not occur; 1 = did occur) and summed to produce a total ACES score with a possible range of 0–10. Inter-item reliability in this sample was acceptable ($\alpha = .84$). The four measures in the biological mother life stress composite were all significantly inter-correlated (r's ranged from .13 to .27),

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Table 1. Means, standard deviations, and correlation matrices of study variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Adoptive mother ACES	–								
2. Biological mother prenatal stress T1	.03	–							
3. Biological mother life stress T1	.18**	.21***	–						
4. Child effortful control T2	−.04	−.02	−.10*	–					
5. Adolescent EXT T3	.03	.01	.13*	−.35***	–				
6. Biological parent psychopathology	.12*	.19***	.39***	.00	.06	–			
7. Child sex assigned at birth	−.06	−.05	−.02	.21***	−.12*	−.01	–		
8. Biological mother race/ethnicity	.06	−.13**	−.01	.02	−.01	−.18**	−.01	–	
9. Adoption openness	.04	.07	−.05	.04	.06	.05	−.08	−.23***	–
M	1.60	−0.01	0.00	4.96	51.72	0.00	0.43	0.29	0.00
(SD)	(1.91)	(0.82)	(0.70)	(0.52)	(9.93)	(1.32)	(0.50)	(0.46)	(0.95)

Note. ACES = adverse childhood experiences. EXT = externalizing behavior. Child sex assigned at birth dummy coded 0 = male, 1 = female. Biological mother race/ethnicity dummy coded 0 = non-Latine White, 1 = other. *$p < .05$, **$p < .001$, ***$p < .001$.

with one exception: the correlation between household income below the U.S. federal poverty level and ACES was nonsignificant, ($r = .06$, $p = .415$). The four life stress measures were standardized, and a mean composite score used in the analyses.

Child effortful control (T2)

Child effortful control was assessed at T2 via adoptive parent report on the effortful control subscale of the Children's Behavior Questionnaire - Very Short Form (Putnam & Rothbart, 2006). Each adoptive parent rated 12 items (e.g., *can wait before entering into new activities if s/he is asked to; can easily stop an activity when s/he is told "no"*) on a 7-point Likert scale, where higher scores indicate greater effortful control. To maximize the sample size, T2 assessments of child effortful control were selected when children were 7 years old. If age 7 data were unavailable, then age 6 data were selected. Adoptive mother and father reports were correlated ($r = .46$, $p < .001$) and a mean composite score was computed. Inter-item reliability in this sample was acceptable ($\alpha = .70$).

Adolescent externalizing behavior (T3)

At T3, adolescent externalizing behavior was assessed via adoptive parent reports on the age 6 - 18 version of the Child Behavior Checklist (Achenbach & Rescorla, 2001). Adoptive parents rated externalizing behavior across 35 items comprising the rule-breaking behavior and aggressive behavior subscales (e.g., *Argues a lot*) on a 3-point Likert scale (not true, true, very true), where higher scores indicate greater externalizing behavior. The T3 assessments of adolescent externalizing behavior were selected when children were 11 years old. Adoptive mother and father reports were correlated ($r = .68$, $p < .001$), and a mean composite score was computed. Inter-item reliability in this sample was acceptable ($\alpha = .91$).

Covariates

Biological parent psychopathology

Biological parent psychopathology symptoms were included as a traditional proxy for heritable risk of child externalizing behaviors, as prior research has shown linkages between general broad-band psychopathology in one generation and externalizing behavior in their offspring (Zhou et al., 2023) and that these familial associations reflect genetic attributes (Caspi et al., 2023). A composite score was created using principal components analysis based on the biological mothers' and fathers': (1) number of lifetime diagnoses assessed via the Diagnostic Interview Schedule (DIS; Robins et al., 1981) and Composite International Diagnostic Interview (CIDI; Kessler & Üstün, 2004), (2) number of symptoms endorsed via the DIS and CIDI, (3) age of onset at each disorder assessed via DIS and CIDI, and (4) proportion of first-degree relatives endorsing the same class of problems. To disentangle genetic from prenatal influences, the genetic risk indicator (e.g., the diagnosis score and/or symptom count scores) was coded as absent if the onset and/or symptoms of a given disorder occurred only during pregnancy. Missing values were handled in the principal component analysis. Data from biological mothers and fathers were aggregated, and a mean score was used when data from both parents were present ($r = .25$, $p < .001$). See Marceau et al., 2019 for detailed rationale and methodology for composite formation.

Child sex assigned at birth

Child sex assigned at birth was included (0 = male; 1 = female), given sex differences in child effortful control and adolescent externalizing behavior.

Biological mother race/ethnicity

Biological mother race/ethnicity was dummy coded (0 = non-Latine White status and 1 = an endorsement of any other racial/ethnic category).

Adoption openness

We controlled for the level of openness in the adoption at 18 months to account for the effect of contact with and knowledge about their adoption counterpart. Adoption openness was assessed as a standardized composite of biological mothers' and adoptive parents' self-reported perceptions of the level of openness in the adoption (Ge et al., 2008). Main effects and interactions with biological mother prenatal stress and life stress were included in the models, to ascertain whether adoption openness impacted the association between the primary biological mother predictor variables and the child outcomes.

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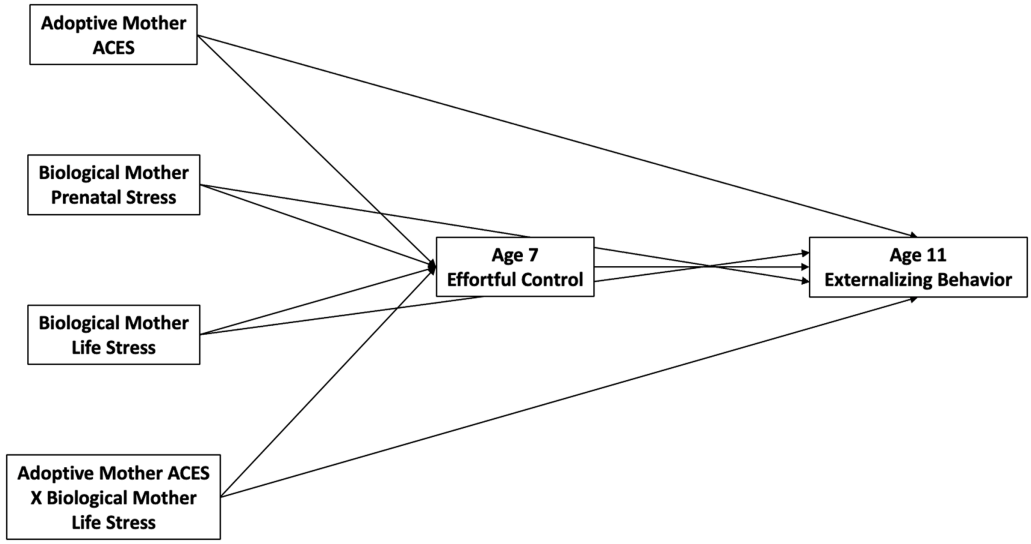

Figure 1. Moderated mediation model testing the effects of adoptive mother ACES, biological mother prenatal stress, and biological mother life stress on adolescent externalizing behavior through effortful control with conditional effects of biological mother life stress across levels of adoptive mother ACES.

Analytic plan

Missing data patterns were evaluated using the *misty* package in R. Among 561 cases, there were 32 different missing data patterns for variables in the analysis covariance matrix. There were 245 (43.67%) complete and 316 (56.33%) partial data cases. Specifically, 283 cases had adoptive mother ACES data, 561 had biological mother prenatal stress data, 522 had biological mother life stress data, 470 had child effortful control data, and 407 had child externalizing behavior data. Data were missing completely at random [Little's MCAR χ^2 (197) = 193.12, $p = .565$]. Mediation analyses were conducted in Mplus version 8.6 (Muthén & Muthén, 1998-2020) using full information maximum likelihood to account for missing data. Model 1, the baseline bias-corrected bootstrap mediation model, examined the total effects of adoptive mother ACES, biological mother prenatal stress, and biological mother life stress on T3 adolescent externalizing behaviors (hypothesis 1) and whether child effortful control at T2 mediated these associations (hypothesis 2). Models were based on 5000 bootstrap resamples. Bias-corrected bootstrap methods generate estimates of indirect effects that account for nonnormality of the sampling distribution of the indirect effect. Significant effects were indicated when the 95% confidence interval for the indirect effect point estimate did not include zero (MacKinnon et al., 2004). Model 2 (see Fig. 1), the bias-corrected bootstrap moderated mediation model, added the interaction of biological mother life stress and adoptive mother ACES at T1 as a predictor of both T2 child effortful control and T3 adolescent externalizing behavior. A loop plot was generated to examine the significance of the indirect effect of biological mother life stress on adolescent externalizing behavior across values of adoptive mother ACES. This approach was repeated for the interaction of biological mother prenatal stress and adoptive mother ACES. Unstandardized results are reported.

Results

Means, standard deviations, and correlations are reported in Table 1. Biological mother prenatal stress was positively associated with her life stress and with biological parent psychopathology ($r = .21$ and $.19$, $p < .001$, respectively). Biological mother life stress was negatively associated with child effortful control and positively associated with adolescent externalizing behavior ($r = -.10$, $p = .029$ and $r = .13$, $p = .014$, respectively); it was also positively associated with biological parent psychopathology ($r = .39$, $p < .001$). Unexpectedly, adoptive mother ACES were positively associated with both biological mother life stress and biological parent psychopathology ($r = .18$, $p = .004$ and $r = .12$, $p = .043$, respectively). Child effortful control was negatively associated with adolescent externalizing behavior ($r = -.35$, $p < .001$). Girls exhibited higher effortful control and lower externalizing behavior than boys ($r = .21$, $p < .001$ and $r = -.12$, $p = .012$, respectively). Finally, biological mothers from racial/ethnic minoritized backgrounds exhibited less prenatal stress and lower biological parent psychopathology than their non-Latine White counterparts ($r = -.13$, $p = .003$ and $r = -.18$, $p < .001$, respectively) and reported lower rates of adoption openness ($r = -.23$, $p < .001$).

Mediation model

Prior to examining moderation effects, a baseline bias-corrected bootstrap mediation model was tested to examine the main effects of the predictors, and to determine whether T2 effortful control mediated the associations of adoptive mother ACES, biological mother prenatal stress, and biological mother life stress with T3 adolescent externalizing behavior. Biological mother life stress was negatively associated with child effortful control ($\beta = -0.08$, {95% CI, −0.16, −0.001}, SE = 0.04, $p = .048$), and child sex assigned at birth was positively associated, such that girls exhibited greater effortful control than boys ($\beta = 0.22$, {95% CI, 0.13, 0.32}, SE = 0.05, $p < .001$). In turn, child effortful control was negatively associated with adolescent externalizing behaviors ($\beta = -6.27$, {95% CI, −8.32, −4.01}, SE = 1.08, $p < .001$). The total effect of biological mother life stress on adolescent externalizing behavior was positive and significant ($\beta = 1.84$, {95% CI, 0.13, 3.63}, SE = 0.89, $p = .039$); the total effects of adoptive mother ACES ($\beta = 0.07$, {95% CI, −0.55, 0.71}, SE = 0.32, $p = .824$) and biological mother prenatal stress were nonsignificant ($\beta = -0.33$, {95% CI, −1.51, 0.69}, SE = 0.56, $p = .559$). After adjusting for the effect of child effortful control, the direct effect of biological mother life stress on adolescent externalizing behaviors reduced in magnitude relative to the total effect and was nonsignificant ($\beta = 1.32$, {95% CI, −0.36, 3.01}, SE = 0.87, $p = .127$). Finally, the indirect effect of biological mother life stress on adolescent externalizing behavior through child effortful control was significant, evidenced by the

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Table 2. Adoptive mother ACES, biological mother prenatal stress, and biological mother life stress predicting adolescent externalizing behavior, mediated by child effortful control

Outcome	β (*SE*)	CI
Child effortful control T2		
Adoptive mother ACES (a^1)	−0.01 (0.02)	[−0.04, 0.02]
Biological mother prenatal stress (a^2)	0.01 (0.03)	[−0.05, 0.06]
Biological mother life stress (a^3)	**−0.08 (0.04)***	**[−0.16, −0.001]**
Biological parent psychopathology	0.02 (0.02)	[−0.02, 0.06]
Child sex assigned at birth	**0.22 (0.05)*****	**[0.13, 0.32]**
Biological mother race/ethnicity	0.05 (0.05)	[−0.06, 0.16]
Adoption openness	0.03 (0.03)	[−0.03, 0.08]
Biological mother prenatal stress X openness	0.01 (0.03)	[−0.05, 0.08]
Biological mother life stress X openness	0.01 (0.04)	[−0.07, 0.08]
Adolescent externalizing behaviors T3		
Adoptive mother ACES (c'^1)	0.00 (0.30)	[−0.58, 0.60]
Biological mother prenatal stress (c'^2)	−0.29 (0.55)	[−1.42, 0.74]
Biological mother life stress (c'^3)	1.32 (0.87)	[−0.36, 3.01]
Child effortful control (b)	**−6.27 (1.08)*****	**[−8.32, −4.01]**
Biological parent psychopathology	0.05 (0.40)	[−0.72, 0.83]
Child sex assigned at birth	−1.00 (1.00)	[−3.01, 0.92]
Biological mother race/ethnicity	0.28 (1.10)	[−1.93, 2.41]
Adoption openness	0.64 (0.50)	[−0.34, 1.60]
Biological mother prenatal stress X openness	0.31 (0.66)	[−1.01, 1.59]
Biological mother life stress X openness	−0.04 (0.80)	[−1.67, 1.48]
Total effects		
Adoptive mother ACES-EXT (c^1)	0.07 (0.32)	[−0.55, 0.71]
Biological mother prenatal stress-EXT (c^2)	−0.33 (0.56)	[−1.51, 0.69]
Biological mother life stress-EXT (c^3)	**1.84 (0.89)***	**[0.13, 3.63]**
Indirect effects		
Adoptive mother ACES-EC-EXT	0.07 (0.10)	[−0.13, 0.28]
Biological mother prenatal stress - EC-EXT	−0.04 (0.18)	[−0.40, 0.34]
Biological mother life stress-EC-EXT	**0.52 (0.29)**$^+$	**[0.02, 1.17]**

Note. Unstandardized coefficients are reported. CI = 95% bias-corrected bootstrap confidence interval. ACES = adverse childhood experiences. EXT = externalizing behavior. EC = effortful control. $^+p < .10$, $^*p < .05$, $^{***}p < .001$. Estimates with confidence intervals that do not contain zero, indicating significant effects, bolded.

bias-corrected bootstrap confidence interval that does not contain 0 ($\beta = 0.52$, {95% CI, 0.02, 1.17}, SE = 0.29 $p = .073$). Unstandardized coefficients for the full model are reported in Table 2.

Moderated mediation model

Next, to test the third study hypothesis, interaction effects between biological mother life stress and adoptive mother ACES were tested. Adoptive mother ACES significantly moderated the path from biological mother life stress to child effortful control ($\beta = -0.08$, {95% CI, −0.13, −0.03}, SE = 0.02, $p = .002$). Simple slopes analysis indicated that associations between biological mother life stress and child effortful control were significant and negative when adoptive mother ACES were average ($\beta = -0.09$, {95% CI, −0.18, −0.01}, SE = 0.04, $p = .023$) and high (1 *SD* above the mean; $\beta = -0.24$, {95% CI, −0.37, −0.11}, SE = 0.07, $p < .001$) but nonsignificant at low levels of adoptive mother ACES ($\beta = 0.05$, {95% CI, −0.06, 0.17}, SE = 0.06, $p = .380$), suggesting an environmental amplification of genetic risk. In addition, child sex assigned at birth was significantly associated with child effortful control, such that girls exhibited greater effortful control than boys ($\beta = 0.23$, {95% CI, 0.14, 0.33}, SE = 0.05, $p < .001$). In turn, child effortful control was negatively associated with adolescent externalizing behavior ($\beta = -5.53$, {95% CI, −7.65, −3.18}, SE = 1.13, $p < .001$).

The conditional indirect effect of biological mother life stress through child effortful control on child externalizing behavior was significant when adoptive mother ACES were average ($\beta = 0.52$, {95% CI, 0.09, 1.10}, SE = 0.26, $p = .041$) and high (1 *SD* above the mean; $\beta = 1.32$, {95% CI, 0.58, 2.30}, SE = 0.44, $p = .002$) but nonsignificant at low levels of adoptive mother ACES ($\beta = -0.28$, {95% CI, −1.00, 0.34}, SE = 0.33, $p = .393$), indicating that child effortful control significantly mediated associations between

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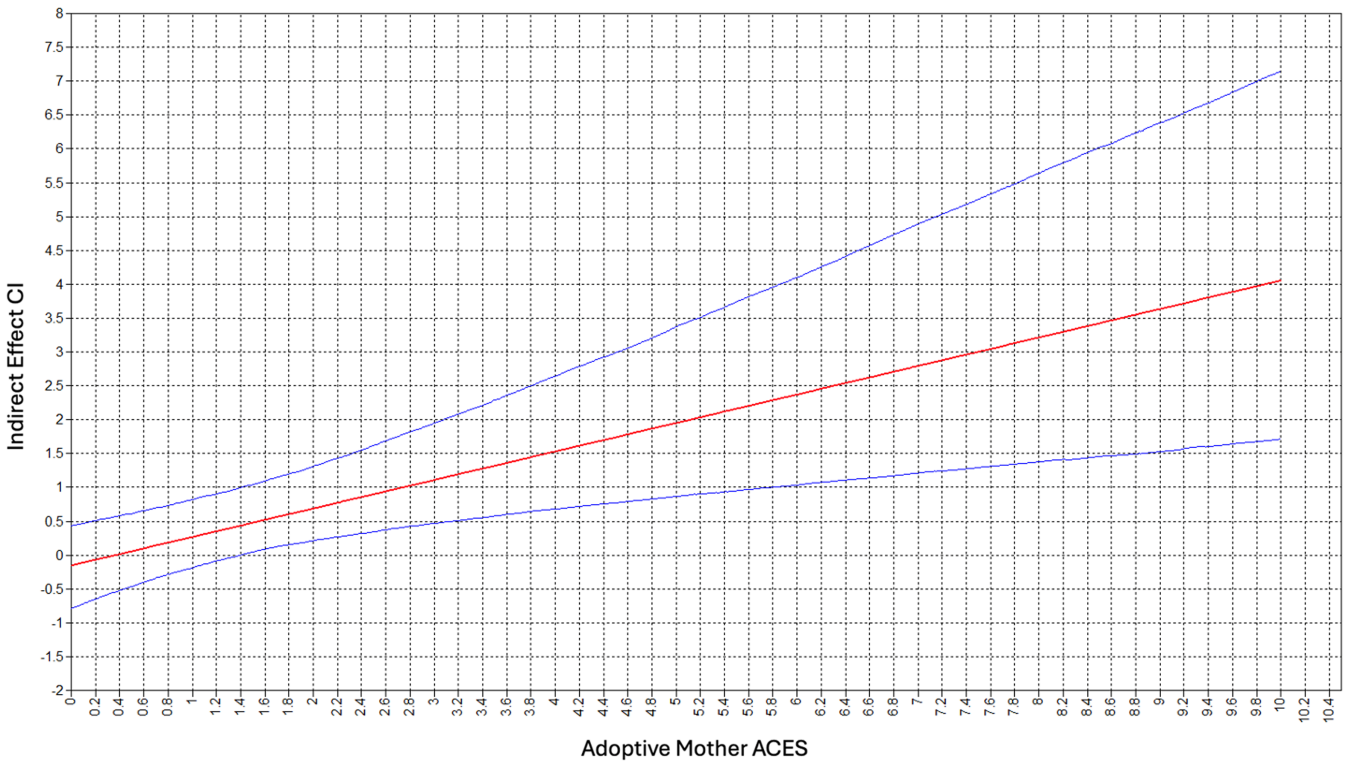

Figure 2. Loop plot of bias-corrected boot strap confidence intervals for the indirect effect of biological mother life stress as a function of adoptive mother ACES.

biological mother life stress and adolescent externalizing behaviors only when adoptive mother ACES were average or high, but not low. Moreover, the loop plot of bias-corrected bootstrap confidence intervals for the indirect effect indicated that significant mediation occurred when adoptive mothers reported more than 1.4 ACES (see Fig. 2). Finally, adoptive mother ACES significantly moderated the path from biological mother life stress to adolescent externalizing behavior ($\beta = 1.41$, {95% CI, 0.50, 2.39}, SE = 0.49, $p = .004$), such that the direct effect of biological mother life stress on adolescent externalizing behavior was only significant at high levels of adoptive mother ACES ($\beta = 4.30$, {95% CI, 1.85, 6.96}, SE = 1.33, $p = .001$) and nonsignificant at low ($\beta = -1.09$, {95% CI, −3.31, 1.33}, SE = 1.19, $p = .356$) and average levels ($\beta = 1.60$, {95% CI, −0.08, 3.21}, SE = 0.84, $p = .056$), indicating that the effect of biological mother life stress on adolescent externalizing behavior persists above and beyond the indirect effect through child effortful control when adoptive mother ACES were high. Unstandardized coefficients for the full model are reported in Table 3.

Adoptive mother ACES were also tested as a moderator of paths from biological mother prenatal stress to child effortful control and adolescent externalizing behavior, yielding nonsignificant results ($\beta = -0.03$, {95% CI, −0.07, 0.01}, SE = 0.02, $p = .116$ and $\beta = -0.13$, {95% CI, −0.85, 0.56}, $SE = 0.36$, $p = .712$, respectively).

Sensitivity analysis

The above models were recomputed using only the subsample of biological mothers who were age 18.75 years or older at the birth of the adoptee (83% of the sample; $n = 463$) to rule out the possibility that the biological mother life stress composite score was confounded with direct prenatal stress exposures that occurred before age 18 as reported on the ACES, for women who began their pregnancy before age 18. Results of these analyses replicated those with the full sample and are reported in Supplementary Table 1 and 2.

Discussion

We examined intergenerational transmission pathways of maternal stress to child externalizing behavior with a parent-offspring adoption sample. Building on the foundational work on intergenerational pathways of trauma and psychopathology led by Cicchetti and colleagues, this approach enabled genetic, prenatal, and rearing mother stress to be modeled separately and interactively to test whether maternal stress exposure in one domain amplified the intergenerational effects of maternal stress in another domain. Children's effortful control was examined as a hypothesized mediator of linkages between parental stress and child externalizing behavior. The study hypotheses were partially supported, with significant main and indirect effects of biological mother life stress (representing genetic influences) on children's effortful control and externalizing behavior, but no main or mediated effects of prenatal stress or biological parent psychopathology on either child outcome. Moreover, adoptive mother ACES amplified the direct and mediated associations of biological mother life stress and child outcomes, strengthening associations when adoptive mothers reported that they had experienced more ACES during their own childhood. The results suggest that the ways in which exposure to stress embeds itself under the skin to precipitate lower effortful control and more externalizing problems in offspring is quite nuanced, as all of the predictors in the current study examined maternal stress experiences outside of the context of the child's postnatal rearing environment. The

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Table 3. Moderated mediation: adoptive mother aces, biological mother prenatal stress, and biological mother life stress predicting age 11 externalizing behavior, mediated by child effortful control, with adoptive mother ACES as a moderator

Outcome	AH β (*SE*)	AH CI
Child effortful control T2		
Adoptive mother ACES (a^1)	0.00 (0.02)	[−0.03, 0.03]
Biological mother prenatal stress (a^2)	0.01 (0.03)	[−0.05, 0.06]
Biological mother life stress (a^3)	0.03 (0.05)	[−0.08, 0.13]
Biological life stress X AM ACES (a^4)	**−0.08 (0.02)****	**[−0.13, −0.03]**
Biological parent psychopathology	0.02 (0.02)	[−0.02, 0.06]
Child sex assigned at birth	**0.23 (0.05)*****	**[0.14, 0.33]**
Biological mother race/ethnicity	0.03 (0.05)	[−0.08, 0.14]
Adoption openness	0.01 (0.03)	[−0.04, 0.07]
Biological mother prenatal stress X openness	0.01 (0.03)	[−0.05, 0.07]
Biological mother life stress X openness	−0.02 (0.04)	[−0.10, 0.07]
Adolescent externalizing behaviors T3		
Adoptive mother ACES (c'^1)	−0.22 (0.32)	[−0.83, 0.41]
Biological mother prenatal stress (c'^2)	−0.28 (0.54)	[−1.40, 0.74]
Biological mother life stress (c'^3)	−0.66 (1.08)	[−2.71, 1.54]
BM life stress X AM ACES (c'^4)	**1.41 (0.49)****	**[0.50, 2.39]**
Child effortful control (b)	**−5.53 (1.13)*****	**[−7.65, −3.18]**
Biological parent psychopathology	0.01 (0.39)	[−0.77, 0.76]
Child sex assigned at birth	−1.35 (1.02)	[−3.38, 0.62]
Biological mother race/ethnicity	0.53 (1.13)	[−1.70, 2.74]
Adoption openness	0.83 (0.51)	[−0.13, 1.86]
Biological mother prenatal stress X openness	0.37 (0.67)	[−0.96, 1.70]
Biological mother life stress X openness	0.47 (0.85)	[−1.24, 2.12]
Total effects		
Adoptive mother ACES-EXT (c^1)	−0.21 (0.34)	[−0.88, 0.45]
Biological mother prenatal stress-EXT (c^2)	−0.32 (0.55)	[−1.45, 0.70]
Biological mother life stress-EXT (c^3)	−0.82 (1.06)	[−2.78, 1.39]
BM life stress X AM ACES (c^4)	**1.83 (0.51)*****	**[0.86, 2.85]**
Conditional indirect effects		
BM life stress-EC-EXT, Low AM ACES	−0.28 (0.33)	[−1.00, 0.34]
BM life stress-EC-EXT, Avg AM ACES	**0.52 (0.26)***	**[0.09, 1.10]**
BM life stress-EC-EXT, High AM ACES	**1.32 (0.44)****	**[0.58, 2.30]**
Conditional direct effects		
BM life stress-EXT, Low AM ACES	−1.09 (1.19)	[−3.31, 1.33]
BM life stress-EXT, Avg AM ACES	1.60 (0.84)	[−0.08, 3.21]
BM life stress-EXT, High AM ACES	**4.30 (1.33)****	**[1.85, 6.96]**

Note. Unstandardized coefficients are reported. CI = 95% bias-corrected bootstrap confidence interval. ACES = adverse childhood experiences. BM = biological mother. AM = adoptive mother. EC = effortful control. EXT = externalizing behavior. *$p < .05$, **$p < .01$, ***$p < .001$. Estimates with confidence intervals that do not contain zero, indicating significant effects, bolded.

complex and multidimensional nature of intergenerational stress transmission is discussed in the sections that follow.

Genetic pathways: transmission from biological mother life stress but not biological parent psychopathology

We found that biological mother's life stress was associated with the adoptee's effortful control (age 7) and externalizing behavior (age 11). As the biological mother did not parent the adoptee, these associations are consistent with a genetic transmission pathway linking stress in one generation to externalizing problems in offspring. We did not find an association between biological parent psychopathology and child externalizing behavior, which is a more traditional approach to measuring genetic influences on child psychopathology in adoption designs. Rather, our construct of biological mother life stress was the singular indicator that showed

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associations with adoptee executive control and externalizing behavior (moderated by adoptive mother ACES, as discussed below).

The current study was not designed to ascertain why biological mother life stress was a potent marker of intergenerational genetic influences and biological parent psychopathology was not, but we pose a few possibilities. First, it may be that exposure to stress or trauma leaves a biological trace on the individual that is transmitted to the next generation via epigenetic processes, even if the individual who experienced the stress has not experienced psychopathology, themself. Perhaps it is these "under the skin" markers of stress that are transmitted to the next generation, via genes passed from parent to child. An increasing body of work suggests that epigenetic processes may be at play in the intergenerational transmission of stress and trauma via the effects of early-life stress on DNA methylation (e.g., Conradt et al., 2018). Although the most robust literature on epigenetic processes comes from non-human animal studies, a recent review by Nöthling et al. (2020) identified a variety of epigenetic mediators on a common pathway between childhood trauma and psychiatric disorders. They noted, however, that longitudinal studies and more consistency in methodological approach are needed to disentangle cause and effect associations. Similarly, a scoping review of the evidence for intergenerational epigenetic transmission of stress and trauma in humans found that 19 of 22 studies reported differential DNA methylation in offspring of women exposed to stress and trauma (Zhou & Ryan, 2023). Thus, it is plausible that exposure to stress in one generation affects gene expression in their offspring—resulting in associations with externalizing behavior.

Alternatively, a person's stressful life experiences may represent bidirectional processes related to genetic tendencies. For example, individuals with lower effortful control may be genetically predisposed to risk-taking contexts that expose them to more adverse or traumatic events (e.g., accidents, substance abuse). Prior research indicates that executive functioning deficits are associated with exposure to greater stress (Adamis & Olatunji, 2024), lending some credence to this possibility.

Finally, drawing from the stress-generation hypothesis (Hammen, 2006), there is empirical support for the notion that some individuals generate or perpetuate stressful experiences in their lives, as a function of their psychopathology. This explanation is somewhat controversial for accounting for the intergenerational continuity of psychopathology, in that it puts the onus on the individual, rather than on the structural and relational context in which they reside. Nonetheless, stress-generation effects have been widely replicated in clinical, community, child, adolescent, and adult samples (see Hammen, 2006, and Liu & Alloy, 2010, for reviews), forecasting a pernicious cycle of recurring psychopathology and stress that could explain the association between biological mother life stress and externalizing behavior in the adoptee.

The moderating role of rearing mother ACES

Although ACES reported by adoptive mothers showed an intriguing moderating effect, amplifying the association between biological mothers' life stress on children's effortful control and externalizing behavior, the current study falls short of identifying the mechanism underlying how maternal ACES cause this exacerbation. Clearly, mothers' ACES that occurred well before the child was born cannot have a direct, proximal effect on child psychopathology; an intermediary mechanism must be at play. Further, because adoptive mothers in this study are not genetically related to the adoptee, specific genes shared between mother and child are not the source of the amplification. There is no evidence in this study for selective placement into adoptive homes due to biological parents choosing to place their child with adoptive parents who share characteristics with themselves (Leve et al., 2019), and because levels of openness in the adoption were incorporated into the analyses and did not appear to contaminate the adoption study premise of separation of genetic and rearing environmental influences, several possibilities for this association remain. First, prior studies on the association between exposure to stress and parenting behaviors suggest that parenting practices are adversely affected by trauma (e.g., Rowell & Neal-Barnett, 2022; Siverns & Morgan, 2019). Thus, it is likely that there are unmeasured parenting variables in the current study that are the more proximal moderators of the biological parent-child externalizing behavior association. Similarly, prior research reviews show clear linkages between exposure to childhood trauma and adult mental health (Hales et al., 2023; McKay et al., 2021; Xiao et al., 2023), which could then undermine parenting quality and skills. Connecting both of these aspects from the prior literature, a systematic review of the effects of parental ACES on parenting and child psychopathology found a direct association between parental ACES and parenting, and between parental ACES and child externalizing symptoms (Rowell & Neal-Barnett, 2022). Maternal anxiety and depressive symptoms, emotional availability, and attachment were identified as mediators of the association between parental ACES and child externalizing symptoms. Future studies should incorporate aspects of the current rearing environment, such as rearing parent psychopathology and emotional availability, to further distill how ACES perpetuate intergenerational continuity via impacts on rearing parent mental health and parenting behaviors.

A second possible explanation for the moderating role of adoptive mother ACES draws upon limitations related to the retrospective self-reported nature of ACES. The literature on childhood trauma indicates that there is often low consistency between prospective data and retrospective recalls of trauma. For example, a systematic review and meta-analysis found poor agreement between prospective and retrospective measures of childhood maltreatment. Specifically, 52% of individuals with prospective observations of childhood maltreatment did not retrospectively report it, and likewise, 56% of individuals retrospectively reporting childhood maltreatment did not have prospective evidence of childhood maltreatment (Baldwin et al., 2019). Thus, adoptive mothers might mis-recall events from their own childhoods; their self-reported ACES could instead reflect their current well-being, which may be negatively impacted by their child's current externalizing behavior (which is also reported by the mother). Indeed, adoption studies have identified evocative effects emanating from genetic predispositions in the child that evoke specific parenting behaviors (O'Connor et al., 1998).

In the current study, the unexpected positive correlation between both the biological mother life stress and psychopathology constructs with ACES in adoptive parents further suggests that evocative processes may be at play. In a post-hoc analysis, we examined the correlation between biological mothers' ACES (from the biological mother life stress construct) and adoptive mothers' ACES and identified a similarly positive and significant association ($r = .32$ $p < .001$). In the absence of selective placement, the primary explanation for associations between a measure in the biological parent and a measure of the adoptive parent is via a genetically evoked pathway from biological parent, to adoptee, to an evoked response in the rearing parent (e.g., Ge et al., 1996). The notion that children's behavior might impact their rearing parents'

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ACES memories is a very speculative albeit intriguing possibility. It highlights the possibility that the child plays an evocative role in their own psychopathology via influences on their caregivers, and also points to cognitive interventions and mindfulness strategies for caregivers as possible effective intervention strategies. Intervention strategies such as Acceptance and Commitment Therapy and mindfulness approaches have shown promise in helping parents consider traumatic events from their own childhood and move forward in healthy ways (Boyd et al., 2018; Laifer et al., 2017; Ruiz, 2010).

Prenatal stress

The lack of associations between our measure of prenatal stress and child effortful control and externalizing behavior was counter to the study hypotheses. Although there is some prior evidence and theory to suggest we would find such associations, most of the prior research on prenatal effects on children's psychopathology is confounded by the use of biologically reared samples of children. As such, genetic and postnatal effects are difficult to disentangle from prenatal-specific influences. The adoption design separates postnatal influences from prenatal and genetic influences, and measures prenatal and genetic influences separately, but it isn't optimal for also isolating genetic from prenatal influences. In addition, our measure of prenatal stress may not have been as comprehensive as is needed to detect associations, and/or it may have under-classified stressful events within the medical records element of our prenatal stress construct. Studies that leverage data from assisted reproductive technologies (ART) and include individuals who participated in ART with egg or embryo donation, or pregnancy surrogacy, are especially clever ways to isolate prenatal influences from the mother's genetic influences (Harold et al., 2012), including the examination of maternal stress effects during pregnancy on offspring outcomes (Rice et al., 2010). However, studies using this type of innovative research design are rare and require replication using complementary research designs.

Limitations

Several study limitations are discussed in the preceding sections of the discussion, but additional caveats are worth noting here. First, due in part to sample size limitations, the current study focused on biological parent life stress, prenatal stress, and rearing parent ACES only in mothers. However, biological and rearing fathers also play important roles in child development. For example, as noted by Bowers and Yehuda (2016), paternal stress may directly affect offspring via epigenetic modifications in sperm. In addition, fathers can indirectly impact offspring psychopathology via their effects on the mother, the couple relationship, and fathers' direct interactions with the child (Jansen et al., 2023). Likely, paternal stress is transmitted by a combination of both biologically driven direct effects (i.e., the genes he contributes to the child; effects of stress on his sperm), direct rearing environment effects (e.g., his interaction with the child in the context of the rearing environment), and indirect effects (e.g., his relationship with the mother pre-conception, during the prenatal period, and postnatally) that impact the mother's own stress, well-being, and parenting abilities. As noted in a recent review (Jansen et al., 2023), there is a growing body of evidence on father's pre-conception, prenatal, and postnatal role on children's health, including their neurodevelopment and physical health, yet fathers' contributions to children's development and psychopathology remain a significantly understudied area that warrants additional attention.

A second limitation of the current study is the self-report nature of most variables included in the models. Although we attempted to minimize potential within-rater bias with strategies such as using an aggregate parent rating of children's effortful control and externalizing problems (i.e., data from both rearing parents were aggregated to create a composite score), incorporating medical record coding into the index of prenatal stress, and using both biological parent and adoptive parent data, the study lacked data on child self-report or observationally coded effortful control and externalizing behavior. The lack of inclusion of a measure of child-reported life stress or ACES is another limitation of the current study. Similarly, having biophysiological measures of stress such as cortisol or other neuroendocrine functioning in parents and children would advance the understanding of the biological stress mechanisms and the extent to which children's biophysiology is a reflection of genetic, prenatal, and rearing environmental transmission. Further, this study examined externalizing outcomes, but the effects of stress tend to lack specificity in terms of predicting externalizing versus internalizing outcomes (McMahon et al., 2003). Future research could incorporate additional measures of children's problems beyond externalizing behavior, and measures of prosocial behavior.

Clinical insights would be strengthened if more children in the current study showed externalizing behaviors at or above the clinical threshold; approximately 11.5% of children in the current study showed clinically elevated externalizing problems. Longer-term follow-up into later adolescence would also provide a more comprehensive examination of whether the moderated biological parent stress-child externalizing behavior association sustains.

Future directions and recommendations for the field

The ways in which biological (e.g., genetic) and rearing environmental influences work together to shape intergenerational transmission are complex and our ability to identify malleable mechanisms of their intergenerational transmission is still in its early stages. Yet, evidence for the harmful, sustained, and intergenerational effects of stress is indisputable. We identified several specific variables or approaches that we recommend incorporating in future research and clinical directions earlier in this discussion. In this final section of the manuscript, we advocate for three directions that the field of developmental psychopathology can take to advance the understanding of how stress gets under the skin to affect the intergenerational continuity of psychopathology.

First, we urge researchers in the field of development and psychopathology to conduct more widespread and more rigorous measurement of family stress. Such strategies could include the incorporation of brief screeners such as the ACES in studies that may have a heavy assessment burden and/or where stress is not a primary focus of the research, or, the inclusion of biophysiological measures of stress response in studies that are for directly focused on stress mechanisms. Where possible, a prospective measurement approach should be incorporated, and both interpersonal and economic stress measured. For example, studies that prospectively measure ACES before a person is parenting and then again after they are parenting could help disentangle whether child evocative effects are operating to influence adults' retrospective recollections of their own childhood trauma.

Second, we need to leverage creative study designs that can help the field disentangle genetic, prenatal, and postnatal influences of stress intergenerationally. In addition to parent-offspring adoption and ART designs already mentioned, sibling and family studies

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and case/control studies where one individual in a family is exposed to a stressor and another unexposed are innovative approaches that can be leveraged. For example, traumatic events that a mother experienced during one pregnancy but not another pregnancy can be a useful method for isolating the effects of prenatal stress, and postnatal stressors that one sibling experiences and another does not experience can help disentangle rearing environmental influences. As most children are raised by their biological parent(s), these types of approaches provide an important means of disentangling prenatal, genetic, and postnatal stress transmission mechanisms.

Third, the field must more proactively translate findings from basic science studies such as the current study to help inform the selection of prevention and intervention targets. There is unambiguous evidence that families are experiencing stress at high levels, especially since the onset of the COVID-19 pandemic, and that stress effects are transmitted intergenerationally to offspring. By partnering with clinicians, practitioners, and program developers, those involved in leading basic science studies of stress can begin conversations about mechanisms of action that can be targeted to reduce the likelihood of harmful effects of stress on generations to come.

In conclusion, researchers of development and psychopathology have a social and professional imperative to better understand intergenerational stress-psychopathology pathways and to translate that knowledge to develop targeted preventive interventions. We hope that readers of this manuscript will have gained a deeper understanding of the complex pathways to intergenerational transmission of stress and feel inspired to leverage some of the innovative development and psychopathology tools and methods put forward in this journal to develop creative study designs, robust measures, and novel prevention approaches to lead the field into its next steps to promote child well-being in the context of stress exposure.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579424000191.

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