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Intergenerational Transmission of Maternal Childhood Adversity and Depression on Children's Internalizing Problems

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Abstract

Objective: Childhood trauma exacts a lasting toll on one's own mental health and the health of one's offspring; however, limited research has examined the pathways through which this *intergenerational transmission* occurs. This study aimed to identify the transactions and mechanisms that link maternal early life trauma, maternal depressive symptoms, and children's internalizing symptoms.

Method: A pregnancy cohort of N = 1462 mothers (66% Black, 32% White, 2% Other race) reported their childhood trauma exposure and depressive symptoms during pregnancy. Maternal depressive and children's internalizing symptoms were measured repeatedly when offspring were 12, 24, 36, and 48–60 months of age. A path model tested the transactional associations between maternal and child symptomatology and mediation of maternal childhood trauma on offspring symptoms via maternal depressive symptoms.

Results: Mothers' childhood trauma history was related to greater prenatal and postnatal (12 and 24 months) maternal depressive symptoms, which were prospectively associated with offspring internalizing problems at 36 and 48–60 months. Child-directed effects on maternal depressive symptoms were not observed. The association of maternal trauma on children's internalizing at 36 months was mediated by maternal depressive symptoms at 24 months.

Limitations: Assessments of the key study variables were provided by mothers. Childhood trauma was evaluated retrospectively.

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Conclusion: Women's experiences of adversity in childhood have persistent and cumulative effects on their depression during the transition to parenthood, which is associated with risk for children's internalizing. Given the two-generation influence of maternal childhood trauma exposure, attending to its impact may protect both caregivers and their children.

Keywords

Adverse childhood experiences; intergenerational trauma transmission; prenatal programming; maternal depression; child internalizing

Exposure to traumatic and stressful early life experiences occur with alarming frequency. In a nationally representative sample, approximately one-quarter of adults experienced three or more adversities in childhood (Mersky et al., 2021), with some evidence that individuals who identify as Black or female are more frequently exposed (Merrick et al., 2018). The negative sequelae of childhood traumas span multiple domains of health (i.e., physical, social, emotional; Hughes et al., 2017), extend across the lifespan (Herzog and Schmahl, 2018), and may be transmitted from one generation to the next (Plant et al., 2018). This latter point, referring to the *intergenerational transmission of trauma*, describes the risk conferred by mothers' own experiences of adversity on their children's emotional and behavioral problems. In a growing research program, mothers' exposure to childhood traumatic events (CTE; e.g., physical, sexual, or emotional abuse; neglect; exposure to domestic violence) has been associated with a range of socioemotional, behavioral, and neurodevelopmental outcomes among offspring (Plant et al., 2018). Following this consistent documentation of *which* child outcomes are influenced by maternal early life adversity, empirical investigations have turned to examining *how* such associations emerge. The mechanistic pathways that link maternal CTE to child outcomes are complex and likely interactive, including biological factors (Jones et al., 2019), negative life events (Negriff et al., 2020), attachment styles (Cooke et al., 2019), and maternal mental health (Browne et al., in press; Doi et al., 2020). The present study focuses on the potential mediating role of maternal depressive symptoms, given the high prevalence of such symptomatology among women who have experienced early trauma (Letourneau et al., 2019). For example, a meta-analysis of more than 16 epidemiological studies found that adults who experienced childhood maltreatment or abuse were twice as likely to develop persistent and recurrent depression (Nanni et al., 2012).

Depressive symptoms among mothers, in turn, serve as a risk factor for children's emotional problems, particularly internalizing symptoms (Goodman, 2020). Evidence for these separate pathways (i.e., maternal childhood trauma to maternal depressive symptoms; maternal depressive symptoms to children's internalizing problems) has prompted tests (albeit limited) of comprehensive mediation models. In a study of mother-child dyads, Letourneau and colleagues found that maternal perinatal depressive symptoms mediated the relation between maternal adverse childhood experiences (ACEs) and internalizing behavior when children were 2 years of age (Letourneau et al., 2019). Though compelling, the Letourneau study recruited a sample of predominantly White, university-educated, Canadian mothers with higher incomes. Studies that explore these relations in racially and ethnically diverse samples and/or those with lower socioeconomic status are essential yet limited,

which significantly detracts from the inclusivity and generalizability of intergenerational research on maternal adversity and mother/child emotional symptomatology (Henrich et al., 2010). Given racial disparities in adversity-related perinatal health outcomes and the provision of maternal child healthcare services (Lorch and Enlow, 2016), and the need for broad representation of all groups in psychological science (Roberts et al., 2020), it is essential to test these associations in diverse samples. Moreover, extant mechanistic research has generally been methodologically limited by cross-sectional designs and/or single assessments of maternal and child mental health during the early postpartum period (Doi et al., 2020).

Notably, research examining the consequences of maternal depression largely focuses on unidirectional parent-to-child effects, omitting consideration of an essential component of the processes through which parent and child symptoms develop and maintain over time: child-to-parent effects (Paschall and Mastergeorge, 2016). Transactional frameworks capture these dynamic reciprocal processes by modeling how parents and children influence (and are influenced by) each other in a continuous process of development (Paschall and Mastergeorge, 2016). In this way, developmental outcomes are not reduced to a sole cause (i.e., individual or environmental context), but are the product of continuous, interdependent interactions between the child and the family context (Sameroff, 2000). One longitudinal study of ethnically diverse, low-income children and their mothers observed transactional relations such that heightened levels of maternal depressive symptoms at 18 months were positively associated with internalizing symptoms among children at 4 years of age, while heightened levels of children's internalizing at 18 months were associated with greater maternal depressive symptoms at 4 years (Roubinov et al., 2019). Despite the logical appeal and widespread acceptance of transactional models, such approaches continue to be underutilized when compared to models of parent-directed effects (Paschall and Mastergeorge, 2016). Moreover, prior transactional research has not examined the bidirectionality between maternal-child symptoms that may emerge from a backdrop of mothers' CTE.

The present study examined longer-term direct and indirect associations among maternal CTE, maternal depressive symptoms, and children's internalizing symptoms collected from pregnancy through age 5 in a longitudinal cohort of socioeconomically and racially diverse women and their offspring. In order to address the gaps in prior research, the current study tested the following four hypotheses:

1. Higher levels of prenatal and postpartum maternal depressive symptoms would predict higher child internalizing symptoms at subsequent timepoints.
2. Higher levels of child internalizing symptoms would predict higher maternal depressive symptoms at subsequent timepoints.
3. Mothers' experiences of CTE would exert repeated, cumulative effects on maternal depressive symptoms over time such that maternal CTE could predict outcomes for mothers and children at multiple points in time, even when controlling for previous associations.

4. Maternal depressive symptoms would mediate the effect of mothers' CTE on offspring internalizing symptoms.

Method

Participants.

The sample included mothers and their biological children who participated in the Conditions Affecting Neurocognitive Development and Learning in Early Childhood (CANDLE) Study, a prospective study of the effects of environmental exposures on maternal and child health among families in Shelby County, Tennessee (LeWinn et al., 2020). Women were recruited prenatally and followed during their offspring's childhood years; the present study includes data from follow-up assessments through child age 5. Criteria for inclusion in the study were: (1) Shelby County resident, (2) between 16 and 28 weeks gestation, (3) between 16 and 40 years of age, (4) could speak and understand English, (5) had a singleton pregnancy, (6) low medical-risk pregnancy, and (7) planned to deliver at one-of-five participating health care settings in Shelby County. The Institutional Review Board at the University of Tennessee Health Science Center approved all study procedures prior to recruitment and data collection.

There were 5,228 women who were screened for eligibility. Of the 3,320 (63.5%) who met inclusion, 1,503 (45.3%) enrolled in the study. Sample descriptives for participants who were included in the present analyses are displayed in Table 1.

Procedures

Women were recruited from university medical group clinics and through community outreach. For any woman under 18 years old, a legally authorized representative co-signed the written informed consent. Prenatal data were collected from women during a third trimester clinic visit (when women were between 27 and 42 weeks pregnant). Subsequent study assessments were conducted in the homes or during children's clinic visits: 12 months (clinic visit), 24 months (clinic or home visit), 36 month (clinic visit), and 48–60 months (clinic visit).

Measures

Maternal childhood traumatic events (CTE).—Three items from Traumatic Life Events Questionnaire (TLEQ) were used to assess women's exposure to CTE. The TLEQ is a 23-item measure that evaluates lifetime exposure to traumatic events across varied stressor types (Kubany et al., 2000). Prior research has found the measure to have high reliability and validity (Peirce et al., 2009). Three of the items on the TLEQ are specific to childhood traumas and thus, were used for the current study: 1) physically punished growing up, 2) witnessed violence growing up, and 3) sexual molestation before 13th birthday. As in prior published studies using the CANDLE cohort, affirmative responses were summed to create a count of CTE (0–3) (Adgent et al., 2019; Ahmad et al., 2021; Pilkay et al., 2020; Shih et al., 2020; Slopen et al., 2018; Steine et al., 2020).

Maternal depressive symptoms.—At all five study timepoints (prenatal; 12, 24, 36, and 48–60 months postpartum), mothers completed the Brief Symptom Inventory (BSI). The BSI is one of the most widely used and accepted measures of psychiatric symptoms that was adapted from the longer Symptom Checklist-90-R (Derogatis and Melisaratos, 1983). It has good construct validity, concurrent validity, internal consistency, and test-retest reliability (Derogatis & Fitzpatrick; Urbán et al., 2014). The present study used the Depression subscale from the BSI, which evaluates the severity of six symptoms during the prior 7 days on a 5-point Likert scale (0 = never to 5 = almost always). Higher scores indicate greater depressive symptoms.

Child internalizing symptoms.—Offspring internalizing symptoms were evaluated with two different measures in accordance with children's age/developmental stage. At 12 and 24 months, mother's reported on child internalizing symptoms using the 8-item Internalizing subscale of the Brief Infant Toddler Social Emotional Assessment (BITSEA; Briggs-Gowan et al., 2004). Example items include "cries or hangs onto you when you try to leave," "seems very unhappy, sad, depressed, or withdrawn," and "worries a lot or is very serious." At 36 and 48–60 months, mothers completed the 36-item Internalizing subscale of the Child Behavior Checklist (Achenbach and Rescorla, 2000). The CBCL evaluates a number of the same internalizing symptoms as the BITSEA (e.g., "unhappy, sad, or depressed," "worries," "withdrawn, doesn't get involved with others," "gets too upset when separate from parents"), as well as other developmentally-appropriate internalizing symptoms that may emerge in later toddlerhood (e.g., "doesn't answer when people talk to him/her," "acts too young for age").

Covariates.—All models were adjusted for child sex (0 = female, 1 = male), maternal race (0 = Black and Other Race, 1 = White), and household income adjusted for family size when children were 12 months old (see Table 1). Note, we did not also control for education in models since the correlation between income and education in this sample was very high ($r = 0.70$, $p < 0.001$).

Data Analysis

Cross-lagged panel model (CLPM) analysis was used to examine the association among maternal CTE, maternal depressive symptoms, and child internalizing symptoms when mothers were in their third trimester of pregnancy, and when offspring were 12, 24, 36, and 46–60 months old.[1] Models of this type permit simultaneous examination of autoregressive paths within variables over time and directional associations between variables controlling for previous levels (i.e., the association of maternal depression at time 1 to child internalizing at time 2 and vice versa). We also included paths from maternal CTE to maternal depression and child internalizing at every time point. In addition to the standard autocorrelations among repeated measures, pathways were also permitted linking observations across two time points (e.g., 24 months maternal depression with 3rd trimester

[1]Prior to running CLPM, we attempted to fit a random-intercept cross-lagged panel model (RI-CLPM) in accordance with prevailing recommendations.(Hamaker et al., 2015) All attempts were unsuccessful and led to model non-convergence due to negative variance components. Consultation with statistical experts advised a less complex model (i.e., CLPM; Personal communication with Linda Muthén, 2020)

maternal depression, 36 months maternal depression with 12 months maternal depression, 48–60 months maternal depression with 24 months maternal depression). Such "time minus two timepoints" correlations were included to account for the high stability in our repeated measures of maternal depression and child internalizing. Model fit was evaluated using a) root mean squared error of the approximation (RMSEA; cutoff value < 0.06), b) comparative fit index (CFI; cutoff value > 0.95), c) standardized root mean square residual (SRMR < .08), d) Tucker-Lewis index (TLI; cutoff value > 0.95), and e) chi-square with degrees of freedom and *p* (Hu and Bentler, 1999; West, Taylor, & Wu, 2012). Maternal race, child sex, and income were included as covariates in all adjusted models, and scores were standardized given that different measures of child internalizing problems were used in the present analyses (BITSEA at the 12 and 24 month assessments and CBCL at the 36 and 48 month assessments, as described above). All analyses were conducted in Mplus version 8.1 (Muthén, L.K. & Muthén, B.O., 1998) with maximum likelihood estimation with robust standard errors (MLR) as our estimation method since it is robust to non-normality. Standardized estimates are reported. Effect sizes were evaluated as small ($\beta < 0.1$), medium ($\beta \in [0.1, 0.5]$, or large ($\beta \geq 0.5$) (Cohen, 1988).

Missing data.—In accordance with the recommended standard missing data handling procedures (Graham, 2009), hypothesis testing was carried out with full information maximum likelihood (FIML) procedures supplemented by auxiliary variables. Of the full sample N = 1,503 women recruited in the study, the present analytic sample of N = 1,462 women included those with a living child who provided data during the prenatal assessment and at least one postnatal visit. Women who were missing all postnatal data were excluded. Women who were retained did not differ from those who were excluded on the basis of race ($p = 0.41$), exposure to childhood trauma ($p = 0.69$), or prenatal depressive symptoms ($p = 0.57$). Simulation studies for complex mediation suggest that our sample size is larger than the minimum sample size required (N>640) to detect small indirect effects with power > .80 (Thoemmes, MacKinnon, & Reiser, 2010).

Results

As shown in Table 1, over one-third of mothers reported exposure to at least one type of traumatic event during childhood: 25% experienced one type, 9% experienced two types, and 2.8% experienced all three types of traumatic events. Zero-order correlations are provided in Table 2.

Fit indices for the cross-lagged model presented in Figure 1 indicated a good fit of the model to the data (*RMSEA* = 0.05 (90% CI = 0.04, 0.06), *CFI* = 0.95, *SRMR* = .04; *TLI* = 0.80; $\chi^2(72) = 1518$, $p = 0.00$). Although the *TLI* was below the recommended cut-off, the *RMSEA*, *CFI*, and *SRMR* of the current model suggest good fit. Prior research states that a combination of *RMSEA* < .06 and *SRMR* < .08 indicates good model fit (Hu & Bentler, 1999); the present model meets these criteria. Autoregressive paths for both maternal depression and child internalizing indicated significant stability from the prenatal period through child age 5 across both constructs (all *p*s < 0.05). Below, we organize our primary results by hypothesis.

Hypothesis 1: Evidence of transactional associations emerged at two timepoints during the early childhood period. Higher levels of maternal depressive symptoms at 24 months were associated with higher child internalizing symptoms at 36 months (β = 0.097, *SE* = 0.037, $p < 0.01$) and similarly, maternal depressive symptoms at 36 months were positively associated with child internalizing at 48–60 months (β = 0.126, *SE* = 0.05, $p < 0.01$), reflecting small and medium sized effects, respectively. Notably, such relations emerged even after accounting for the stability of children's internalizing symptoms and the concurrent relations between the residuals of the maternal depressive symptoms and children's internalizing symptoms.

Hypothesis 2: The paths representing child-directed effects were not significant from infancy through early childhood. Thus, there was no evidence that children's internalizing symptoms predicted maternal depressive symptoms during the study period after adjusting for prior relations across time.

Hypothesis 3: Mothers' exposure to childhood trauma was associated with maternal depressive symptoms during the perinatal through early toddlerhood periods. More specifically, maternal CTE was associated with depressive symptoms during the third trimester of pregnancy (β = 0.194, *SE* = 0.42, $p < 0.01$), and when children were 12 months (β = 0.138, *SE* = 0.04, $p < 0.01$) and 24 months old (β = 0.131, *SE* = 0.05, $p < 0.01$), reflecting medium sized effects. After accounting for these prior relations and the stability of depression, mothers' childhood trauma was not significantly associated with maternal depressive symptoms when children were between 36 and 60 months of age.

Hypothesis 4: Maternal childhood trauma exposure predicted her depressive symptoms during the postpartum period, which in turn, predicted internalizing symptoms in her offspring. We tested three indirect effects suggested by the significant paths in Figure 1. The effect of maternal childhood trauma on offspring internalizing at 36 months was significantly mediated by maternal depressive symptoms at 24 months (β = 0.013, 95% CI 0.001 to 0.025, $p = 0.04$), a small effect. Two additional serial mediation paths were not significant: 1. Maternal CTE to maternal depressive symptoms from 12 to 24 months to children's internalizing symptoms at 36 months (β = 0.006, 95% CI 0.0 to 0.012, $p = 0.052$) and 2. Maternal childhood trauma to maternal depressive symptoms from 24 to 36 months to children's internalizing symptoms at 48–60 months (β = 0.008, 95% CI −0.002 to 0.018, $p = 0.10$).

Discussion

An individual with a history of traumatic experiences in childhood may endure effects of such exposures across their lifespan and potentially transmit such impacts to subsequent generations. However, studies rarely consider the complexities of such relations, including how caregiver and offspring mental health are reciprocally related over time. Integrating intergenerational transmission of trauma and parent-child transactional frameworks, the current study examined how risk initiated by mothers' early CTE generated subsequent reciprocal associations between maternal and child mental health. In a large, prospective, longitudinal cohort study of predominantly Black and White families, mothers' childhood

trauma was associated with maternal depressive symptoms from the perinatal period through 24 months postpartum, but it did not continue to add predictive value to maternal depressive symptoms in later toddlerhood. During toddlerhood, maternal depressive symptoms were associated with children's internalizing symptoms, however child-directed effects of internalizing on subsequent maternal depressive symptoms were not found. A mechanistic pathway was observed whereby the association of maternal childhood trauma on child internalizing at 36 months operated through maternal depressive symptoms at 24 months. This study represents a rigorous test of intergenerational transmission and directionality of effects, as all pathways controlled for previous levels of the predictors and outcome variables and all directions and timings of association were considered from pregnancy to 60 months postpartum.

In the present study, heightened levels of depressive symptoms were observed among mothers with early trauma exposure, confirming prior empirical associations (Choi et al., 2019). A unique feature of the current research is its intensive, repeated measures longitudinal design, which provides for a nuanced exploration of timing. Illustratively, we observed persistent, cumulative effects of maternal CTE on prenatal, 12, and 24 month postnatal maternal depressive symptoms, but no significant relations between CTE and maternal depression when children were 36 through 60 months of age. In general, the perinatal and infancy periods place women at risk for the development of depressive symptoms given normative bodily changes, physical discomfort, identity/role changes, and lack of sleep (Davis and Narayan, 2020). Women with a history of early adversity exposure may be particularly vulnerable because infant and child distress may cue her own early trauma, exacerbating the stressors that typically emerge during this time (Davis and Narayan, 2020). Originating in attachment theory (Bowlby, 2008), the metaphors "ghosts in the nursery" (Fraiberg et al., 1975) and "voices from the past" (Bernard et al., 2012) describe the ways in which mothers' own experiences of abuse, neglect, or other childhood trauma may powerfully influence her adjustment during the transition to parenthood. For mothers in current study, it appears that the legacy of CTE exerts a particularly pronounced effect during pregnancy and in offspring's first two years of life compared to later in childhood.

Transactional paths revealed that maternal depressive symptoms at 24 and 36 months were prospectively associated with children's internalizing problems at the subsequent timepoint (36 and 48–60 months, respectively). However, maternal depression measured during pregnancy or the earlier postpartum periods (12 and 24 months) was not associated with children's later internalizing symptoms. Exposure to perinatal maternal depression has been strongly implicated in the development of offspring behavior problems (Goodman, 2020), thus the lack of significant predictive associations for this time period in the current study is surprising, though not without precedent. In a nationally representative sample, children exposed to maternal depression between the ages of 2 to 3 years and 4 to 5 years were at greater odds for developing behavior problems, however risk was not elevated among children exposed to maternal depression during the first postpartum year (Naicker et al., 2012). It may be the case children experience greater negative effects from cumulative exposure to maternal depression, which would be reflected by an association with later maternal depression (when children are 3 to 5 years of age) that persists from earlier postpartum depression (when children are 1 to 2 years of age; Halligan et al., 2007). While

such findings may suggest a substantive rationale for our pattern of findings, potential methodological reasons may also play a role. In particular, it is harder to discriminate or identify internalizing problems among younger children who have less developed verbal skills compared to when children are older such that there may be limitations in the measurement of this construct at earlier ages.

Similarly surprising was the lack of child-directed effects of early internalizing problems on subsequent maternal depressive symptoms. In other words, maternal depressive symptoms in the current study increased risk for offspring maladjustment, but evidence for the opposing relation was not observed. Although reciprocal relations are less commonly observed during infancy (Hanington et al., 2010), emerging evidence of bidirectionality between maternal depression and children's behavior problems has been found in prior longitudinal studies of older children (Roubinov et al., 2019). Notably, prior transactional models of maternal and child mental health have not attended to mothers' experiences of adversity during the childhood period and it is possible that a relatively stable factor, such as the influence of CTE, could operate as confound (Dora and Baydar, 2020). Our inclusion of maternal childhood adversity in our predictive models may thus explain the small/nonsignificant reciprocal relations found in our models. Depressive disorders among adults with and without exposure to childhood abuse have also been suggested to represent two distinct subgroups of depression given differences in neurobiological underpinnings (Teicher and Samson, 2013) and responsiveness to treatment (Nanni et al., 2012). For women with a history of childhood trauma, it may be the case that factors other than their children's behavior problems exert a more marked impact on depressive symptoms, compared to women without such a history. However, it is also possible that significant associations from children's behavior problems to maternal depression did not emerge due to a lack of statistical power.

Broadening and strengthening prior research that has examined these associations among samples with higher socioeconomic status and using cross-sectional designs (Plant et al., 2018), we observed an indirect pathway of maternal CTE on child internalizing problems via a pathway of maternal depressive symptoms in our socioeconomically and racially diverse sample. Results of this explanatory pathway in the present longitudinal study suggests specificity in the timing of such effects: Maternal CTE was associated with maternal depressive symptoms at 24 months postpartum, which was associated with internalizing when offspring were 36 months of age. Thus, maternal depression during offspring's early years of life appeared to serve as a mechanism linking a mother's own childhood trauma exposure to her child's internalizing problems. However, there was no evidence that mothers' childhood trauma influenced children's behavior problems through a mechanism of maternal mental health in pregnancy. It is interesting to speculate why we observed postnatal (but not prenatal) programming effects. Some studies of fetal programming suggest a stronger influence of prenatal anxiety (Glover, 2011) and prenatal stress (Bush et al., 2021) than prenatal depression on children's development, thus our focus on the latter may have precluded significant findings. Moreover, behavior problems among older offspring appear to be more strongly influenced by depression exposure during the postpartum and later childhood periods rather than prenatal exposure (Hay et al., 2008). Finally, other findings from this cohort indicate that maternal CTE predicts the level and rise of placental

Corticotrophin-Releasing Hormone (pCRH; Steine et al., 2020) a hormone only detectable only during pregnancy that is associated with both risk for maternal postpartum depression (Glynn and Sandman, 2014) and children's neurodevelopmental outcomes (Howland et al., 2016). It may be that effects of maternal CTE on maternal and child mental health are operating, at least in part, through effects on maternal biology during pregnancy. This is an area ripe for further study.

Strengths and Limitations

Strengths of the current study include its prospective, longitudinal design and repeated measures that allow for examination of both timing and directionality of associations among maternal childhood trauma, maternal depressive symptoms, and child internalizing problems during a salient period of child development. However, there are a number of limitations that must also be considered. Assessments of the three primary study constructs (maternal CTE, maternal depressive symptoms, child internalizing problems) were provided by mothers, introducing depression-related negative perceptual biases that may contribute to overreporting of child adjustment problems (Chilcoat and Breslau, 1997). Future research would be strengthened by evaluations of child behavior by other informants (e.g., preschool teacher, other primary caregivers) and/or observational measures, although this is not typical in studies of young child internalizing and often not feasible to collect within large cohorts. Although depressive and internalizing symptoms were assessed in a prospective manner, maternal CTE was evaluated retrospectively. Questions have been raised about the validity of retrospective reports, however prospective and retrospective reports of adversity show moderate agreement and the latter is more strongly predictive of subjectively measured adult outcomes than the former (Reuben et al., 2016). Heritability is a mechanism for the transmission of depression from parents to offspring (Goodman, 2020), though we did not measure genetic factors in the present study. Statistically significant mother-child paths are likely partly due to heritability and partly due to environmental influences. Finally, the present study focused on maternal depression given its prevalence during the childbearing years and its well-established associations with children's internalizing symptoms (Goodman, 2020). Yet, empirical studies suggest parents' symptoms of posttraumatic stress may be a stronger mechanism linking parental history of childhood trauma and offspring adjustment than parental depressive symptoms (Narayan et al., 2021). Future research should examine a broad range of maternal distress and/or psychopathology when evaluating pathways of intergenerational transmission.

Conclusions

Findings from this study support the position that the psychological scars of childhood trauma may not be bound within a single generation. A comprehensive understanding of the intergenerational transmission of trauma necessitates consideration of the timing and mechanisms of such effects. Results of the present study support a model in which mothers with a history of childhood trauma experienced heightened depressive symptoms from the prenatal period through early toddlerhood; maternal depressive symptoms, in turn, were associated with offspring internalizing problems in later toddlerhood and early childhood. The effect of mothers' early trauma to children's internalizing at 36 months operated via a pathway of maternal depressive symptoms at 24 months. Awareness that

maternal depression and its sequelae may have roots in women's exposure to early adverse experiences is important for trauma-informed prevention and intervention efforts that support mothers during the transition to parenthood. Specifically, comprehensive models of care that support maternal mental health in concert with child mental health and parent-child relationship quality (e.g., Healthy Steps; Piotrowski, Talavera, & Mayer, 2009) are essential (yet underutilized) evidence-based models (Hagan et al., 2017). That said, meaningful improvements to population health will also necessitate socioeconomic and policy changes that redress the longstanding systemic inequities that contribute substantially to adverse exposures and their mental health sequelae.

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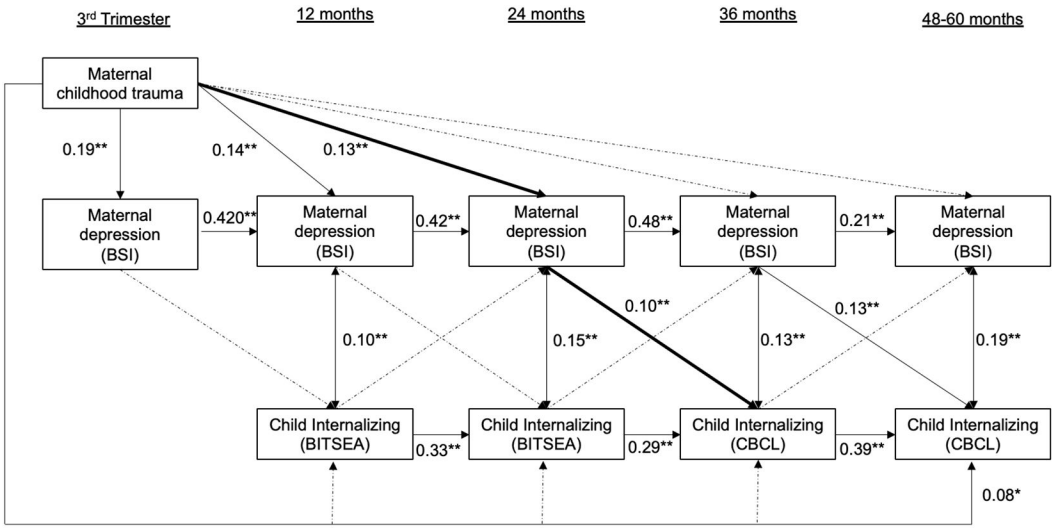
Highlights

- Maternal childhood trauma was persistently associated with her depressive symptoms
- Maternal depressive symptoms prospectively predicted children's internalizing symptoms
- Children's internalizing symptoms did not predict subsequent maternal depression
- Maternal depressive symptoms mediated the impact of maternal trauma on internalizing
- Childhood trauma may exert a two-generation impact on caregivers and their children



*p<.05, **p<.01

Notes. Model adjusts for race/ethnicity, child sex, and income at all timepoints. Solid lines denote significant paths and dashed lines denote non-significant paths. Bold paths indicate significant mediated effect. All pathways linking observations across two time points (i.e., 24 months maternal depression with 3rd trimester maternal depression; 36 months child internalizing with 12 months child internalizing) were included and significant (p < .01), however they are not depicted to reduce visual clutter.

Figure 1.

Cross lagged panel model with standardized estimates linking maternal exposure to childhood trauma, maternal depressive symptoms, and children's internalizing symptoms.

Table 1.

Sample demographics and descriptive statistics (n = 1462)

Maternal race/ethnicity (n, %)	
Black	961 (65.8%)
White	472 (32.3%)
Other race (Asian, American Indian/Alaska Native, Native Hawaiian/Pacific Islander or Other)	27 (1.8%)
Child sex (n, %)	
Male	736 (50.3%)
Household income (*M, SD*)	$20,567 ($18,863)
Maternal childhood traumatic event types (n, %)	
0 traumatic event types	852 (63.0%)
1 traumatic event types	341 (25.2%)
2 traumatic event types	122 (9.0%)
3 traumatic event types	38 (2.8%)
Maternal depressive symptoms (*M, SD*)	
3rd trimester of pregnancy	0.24 (0.4)
1 year	0.21 (0.2)
2 years	0.20 (0.4)
3 years	0.21 (0.5)
4–5 years	0.19 (0.4)
Children's internalizing symptoms (*M, SD*)[a]	
12 months	2.01 (1.6)
24 months	2.20 (1.7)
36 months	6.71 (6.2)
48–60 months	6.21 (6.1)

Note.

[a]Children's internalizing symptoms were measured by the BITSEA at 12 and 24 months and the CBCL at 3 and 4–5 years.

Table 2.

Zero-order correlations among study variables

	1	2	3	4	5	6	7	8	9	10	11	12
1. Maternal childhood traumatic exposures	--											
2. Prenatal maternal depressive symptoms	.19***	--										
3. 12 month maternal depressive symptoms	.19***	.51***	--									
4. 24 month maternal depressive symptoms	.15***	.44***	.45***	--								
5. 36 month maternal depressive symptoms	.14***	.38***	.41***	.55***	--							
6. 48–60 month maternal depressive symptoms	.16***	.40***	.45***	.50***	.41***	--						
7. 12 month child internalizing symptoms	.19***	.10**	.16***	.08*	.004	.10**	--					
8. 24 month child internalizing symptoms	.07*	.11***	.10***	.21***	.11***	.11***	.36***	--				
9. 36 month child internalizing symptoms	.07*	.11***	.20***	.17***	.24***	.14***	.27***	.37***	--			
10. 48–60 month child internalizing symptoms	.10**	.17***	.23***	.26***	.22***	.35***	.21***	.32***	.48***	--		
11. Child sex	−.03	−.04	−.04	.01	.008	.01	−.02	.03	−.03	−.003	--	
12. Household income at 12 months	−.19***	−.11***	−.09**	−.09**	−.08*	−.10**	−.25***	−.21***	−.13***	−.09**	−.03	--
13. Maternal race/ethnicity	−.09**	−.01	.03	−.02	−.01	−.02	−.21***	−.17***	−.004	−.01	−.03	.50***

Notes. Child sex coded as 0 = Female, 1 = Male. Maternal race coded as 0 = Black or Other Race, 1 = White.

* $p < .05$,

** $p < .01$,

*** $p < .001$