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Maternal early exposure to violence, psychopathology, and child adaptive functioning: pre- and postnatal programming

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BACKGROUND: The pre- and postnatal programming mechanisms, timing, and direction of effects linking maternal early exposure to violence (MEEV), psychopathology, and child adaptive functioning are understudied. Thus, the following hypotheses were tested: (H1) higher pre- and postnatal maternal psychopathology will predict lower adaptive functioning, (H2) lower adaptive functioning will predict higher subsequent maternal psychopathology, (H3) cumulative effects of MEEV on maternal psychopathology and adaptive functioning will be observed, and (H4) higher MEEV will predict lower adaptive functioning via maternal psychopathology both pre- and postnatally.
METHODS: Prospective pregnancy cohort study including 1503 mother–child dyads with associations between MEEV, psychopathology, and child adaptive functioning examined using cross-lagged panel analysis. Assessment occurred in the third trimester and annually across the first four years of life.
RESULTS: Higher pre- and postnatal maternal psychopathology predicted lower child adaptive functioning at 12 and 24 months, respectively. MEEV predicted maternal psychopathology cumulatively and offered a repeated prediction of adaptive functioning across the first two years of the child's life, operating predominantly through maternal psychopathology during pregnancy. Child effects on mothers were not observed.
CONCLUSIONS: Like in socioemotional assessment, pediatric assessment of child adaptive functioning should consider the intergenerational transmission of MEEV.

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IMPACT:

- Associations between maternal early exposure to violence (MEEV), psychopathology, and child socioemotional development is well documented.
- Much less is known about the pre- and postnatal programming mechanisms, timing, and direction of effects between MEEV, maternal psychopathology, and child adaptive functioning.
- Findings suggest associations of both prenatal and postnatal maternal psychopathology with child adaptive functioning, though the effects of MEEV were more strongly operative through the prenatal pathway.
- Pediatric assessment and interventions surrounding adaptive functioning should consider the potential role of MEEV in shaping children's health and development, in addition to potential consequences of pre- and postnatal maternal mental health.

INTRODUCTION

Many mothers enter the reproductive stage of life having unresolved histories of violence exposure.[1] A recent study by Madigan and colleagues[2] discovered that 53% of mothers reported at least one traumatic experience, including exposure to violence when they were children. Research demonstrates that maternal early exposure to violence (MEEV) is associated with child health and development through biological, medical, and psychosocial mechanisms.[2–4] The pre- and postnatal programming mechanisms linking MEEV and child socioemotional development have been documented as operating through a variety of pathways (maternal stress physiology [e.g., endocrine and immune/inflammatory physiology], mother–child shared exposure to environmental stress, pregnancy and infant medical problems, and parenting behavior), with particular attention paid to maternal psychopathology and mental health problems.[4–12] The pathways of influence amongst these factors appear complex, characterized by intergenerational transmission, bidirectionality, and indirect effects.[13] However, the pre- and postnatal pathways linking MEEV, maternal psychopathology, and child adaptive functioning remain underexplored.[13–15] This area of research is of great interest to pediatricians and other clinicians who are regularly assessing developmental competencies among patients of varying levels of risk and social disadvantage.[16] To date, no studies have examined questions pertaining to timing and direction of effects across pregnancy and the postnatal period.

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Given the transactional nature of development,[17] it is possible that there are bidirectional effects between maternal psychopathology and adaptive functioning, as is the case with child socioemotional development.[18,19] Moreover, although increasing attention is being paid to maternal mental health and trauma within the pediatric care context, the possibility of intergenerational transmission of risk associated with MEEV on adaptive functioning via maternal psychopathology remains uncertain.[20,21] This is a major clinical research limitation. To provide optimal, trauma-informed care, providers must understand if and how MEEV influences the developmental trajectories of their patients.[16]

There are compelling arguments explicating the multilevel mechanisms that link both prenatal uterine environments[9] and postnatal psychosocial environments[22] with neurocognitive development. These frameworks formed the theoretical bases for our study considering the prenatal and postnatal effects of maternal psychopathology on adaptive functioning, in addition to the intergenerational consequences of MEEV on child adaptive functioning. For prenatal effects, the documented biological mechanisms (and uterine environments) that link MEEV to developmental differences in offspring maternal physiology (e.g., stress hormones, gut microbiome, immunity, and telomere biology), which correspond to neurophysiological correlates in children (e.g., structural and functional brain differences, HPA/ANS functioning). Postnatal influences are equally complex in the psychosocial realm. These behavioral mechanisms consider various aspects of mother–child relationships, including maternal sensitivity, attachment security, positive control, and scaffolding, on one end of the continuum, to harshness, punitive discipline practices, and even the experience of trauma such as abuse/neglect, in many instances.[2–6,13,15,17–19] It is important for studies to simultaneously include pre- and postnatal mechanisms, as the quality of the postnatal environment can potentially offset genetic risk in terms of stress physiology, as demonstrated in cross-fostering paradigms in rodent studies.[22] While extensively outlined in terms of offspring affective functioning, these mechanisms are less explicated in terms of general adaptive functioning.

Respective of these limitations and the recommendations to screen for and treat maternal perinatal psychopathology,[23] the present study aims to provide deeper insight into the associations between MEEV, pre- and postnatal psychopathology, and child adaptive functioning across the first four years of life. With maternal perinatal psychopathology being associated with other child outcomes beginning in pregnancy through numerous mechanisms,[9,24,25] a better understanding of the timing and direction of these effects will assist clinicians in screening and treatment of factors pertinent to maternal health and child adaptive functioning. Additionally, findings may inform treatment programs targeted towards the consequences of maternal psychopathology, thereby promoting the health of mothers and their families. Indeed, lower adaptive functioning in childhood is associated with poorer development in adulthood, making the understanding of this pathway relevant to later functionality across the life course.[26–28] To study this phenomenon, a developmental cascade model[29] was examined linking these factors using data from a prospective pregnancy cohort of mothers and their offspring from Shelby County, Tennessee. We hypothesized associations between MEEV and lower child adaptive functioning during the first four years, where this association would operate partially through higher levels of maternal psychopathology during pregnancy (prenatal programming) and postpartum (postnatal programming).

METHODS

Study design and population

Participating mother–child dyads came from the Conditions Affecting Neurocognitive Development and Learning in Early Childhood (CANDLE) Study; a prospective cohort that followed mothers from pregnancy to when children were roughly four years of age. The purpose of the CANDLE Study was to examine the prenatal and postnatal biological and psychosocial factors that impact children's mental health and development across the early years. Pregnant women were recruited and enrolled between 2006 and 2011 in Shelby County, Tennessee, and data were collected (4-year follow-up) until 2015. Maternal inclusion criteria 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) planned to deliver at one-of-five participating health care settings in Shelby County, and (7) was low medical-risk pregnancy (i.e., pregnancies that excluded all of the following: chronic hypertension requiring therapy or vascular disease requiring therapy; maternal red-cell alloimmunization except Rhesus (Rh) factor; hemoglobinopathy, including sickle-cell trait and severe iron-deficiency anemia (hemoglobin less than 9); insulin-dependent diabetes; appreciable renal or cardiopulmonary disease; prolapsed or ruptured membranes; oligohydramnios; complete placenta previa; endocrine disease; collagen disease (e.g., lupus erythematosus or scleroderma); active or chronic hepatitis; renal disease; pulmonary or heart disease requiring therapeutic medication or limitation of physical activity; major fetal anomaly (e.g., aneuploidy, major organ-system defect); and human immunodeficiency virus.)

Recruitment took place in a university medical group clinic and via community outreach. Written informed consent was obtained and any woman under the age of 18 required a legally authorized representative to co-sign the recruitment form. Of the 5228 women screened for eligibility, 3320 (63.5%) met inclusion criteria, and 1503 (45.3%) enrolled. The CONSORT flow diagram of study participation is presented in Supplemental Fig. 1 (online), including characteristics of those lost to follow-up. Mothers in the study had an average of 2.81 (SD = 1.88) pregnancies prior to the current pregnancy. Assessments took place during home and clinic visits in the third trimester and when children were 12, 24, 36, and 48 months of age. All procedures were approved by the University of Tennessee Health Science Center ethical review board.

Measures

Covariates. All pathways are adjusted for maternal age and race, child sex, household income, birth weight, and preterm status measured in days.

Maternal early exposure to violence. During the third trimester of pregnancy, mothers reported on their childhood exposure to violence using the Traumatic Life Events Questionnaire (TLEQ).[24] The TLEQ is a widely used, 23-item, self-report measure[30,31] of participant experience of traumatic events across 22 pre-determined categories of trauma and one "other" category.[31] The TLEQ has high reliability and validity, including high content validity with a broad range of measurements for traumatic events.[30] Three of the 20 TLEQ items assessed within the CANDLE study ascertained traumas experienced specifically during childhood related to violence and threat: "physically punished growing up", "witnessed violence growing up", and "sexual molestation before 13th birthday". Each type of childhood trauma was added to create a count of types of exposure in childhood (coded 0–3) as has been done elsewhere.[32]

Maternal psychopathology symptoms. Mothers reported on their own symptoms of psychopathology (e.g., depression, anxiety, hostility, paranoia, etc.) by completing the Global Severity Index (GSI) of the Brief Symptom Inventory (BSI)[33] during the third trimester of pregnancy and when children were 12, 24, 36, and 48 months. The BSI is a shorter, suitable alternative to the Symptom Checklist-90-Revised and has strong reliability and validity,[33] as was the case in this sample ($\alpha = 0.94–0.96$).

Child adaptive functioning. When children were 12, 24, 36, and 48 months, mothers completed the Scales of Independent Behavior-Revised (SIB-R).[34] The SIB-R is a checklist-based questionnaire that measures adaptive and maladaptive behavior in the areas of motor skills, personal living skills, social interaction and communication skills, and community living skills from birth to 80 years of age.[34] The scale is widely used and has been validated for infant and child populations.[34–36] Internal consistency in the current study was good ($\alpha = 0.87–0.92$).

Statistical analysis and missing data

A cross-lagged panel analysis was conducted to examine associations amongst MEEV, maternal psychopathology, and child adaptive

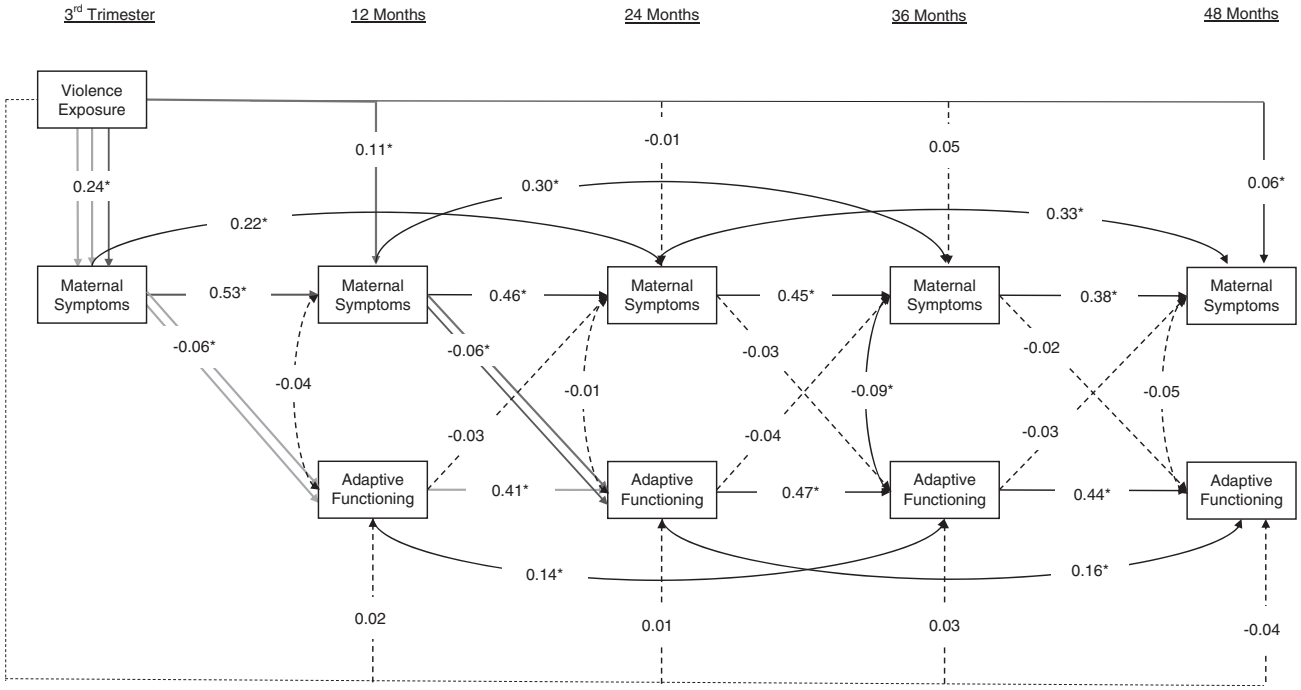

Fig. 1 Cross lagged panel model with standardized estimates linking maternal exposure to violence, maternal psychopathology, and child adaptive functioning.

functioning. The cascade covered the period from pregnancy to when children were 4 years old. Models permit the examination of directional associations between variables controlling for previous levels (i.e., if maternal psychopathology predicts adaptive functioning at a subsequent time point or vice-versa). 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 > 0.80.[37] Our model tested four hypotheses: (H1) higher pre- and postnatal maternal psychopathology would predict lower child adaptive functioning at subsequent time points, (H2) lower adaptive functioning would predict higher postnatal maternal psychopathology at subsequent time points, (H3) there would be cumulative (i.e., repeated) effects of MEEV on maternal psychopathology and adaptive functioning over time (stated differently, there will be a unique effect of MEEV across waves, even after accounting for significant effects at earlier time points; see research by Wade and colleagues[38]) and (H4) higher MEEV would predict lower adaptive functioning via indirect associations through maternal psychopathology.

As is the case in longitudinal cohorts,[39–42] various forms of attrition took place (see Supplemental File). Little's Test of Missing Completely at Random was significant, χ^2 (721) = 1043.40, $p < 0.001$, indicating that the null hypothesis of MCAR was rejected. Versus those retained, mothers with missing postnatal follow-up data were more likely to be Black ($n = 111$, 78.2% vs. $n = 879$, 64.6%, χ^2 (721) = 1056.40, $p = 0.005$) and not have completed high school at the time of pregnancy ($n = 115$, 81.0%, vs. $n = 778$, 57.2%, χ^2 (721) = 1056.40, $p = 0.005$). In settings where MCAR is violated, Graham[43] recommends handling missing data with Full Information Maximum Likelihood supplemented by auxiliary variables that predict missingness. Using this procedure, models were fit using a benchmark analytic sample of either 1503 (full sample), 1474 (full sample, minus 29 mothers who identified as neither Black nor White), 1361 (mothers who participated in third-trimester visits, minus two participants who had incomplete responses), and 1157 (those who participated in the last follow-up visit). Results were substantively the same across all analyses, suggesting minimal bias associated with missing data. Consistent with the recommendation of Graham,[43] we employed the full sample (minus 29 mothers who fell into race categories other than Black or White. Note, models including families who were neither Black nor White had a convergence problem when accounting for the "other" race category as a covariate, likely due to the small residual categories.) Models presented in this paper are $N = 1474$ and were fit in Mplus 8.0 using the robust (MLR) estimator. Standardized estimates are reported, and unstandardized estimates are presented in the Supplemental File.

RESULTS

The hypothesized cross-lagged model was fit to the data, estimating associations amongst MEEV, pre- and postnatal maternal psychopathology symptoms, and child adaptive functioning (Fig. 1). Based on modification and fit indices, *t*-1 and *t*-2 autocorrelations were included. This model fit the data well, χ^2 (14) = 87.60, $p < 0.001$, RMSEA = 0.060, (90% CI = 0.048, 0.072), CFI = 0.976, SRMR = 0.018.

Hypothesis 1 Higher levels of pre- and postnatal maternal psychopathology symptoms predicted significantly lower child adaptive functioning (Fig. 1). Specifically, prenatal maternal psychopathology predicted lower adaptive functioning when children were 12 months of age, while maternal psychopathology at 12 months predicted lower adaptive functioning at 24 months of age, even after accounting for stability in adaptive functioning.

Hypothesis 2 There was no evidence that children's adaptive functioning predicted later levels of maternal psychopathology. Thus, the transmission of effects in the present analysis flow from mother to child, and not the obverse.

Hypothesis 3 MEEV offered a cumulative prediction of maternal psychopathology at pregnancy, 12, and 48 months, even when controlling for previous levels (*t*-1 and *t*-2) of these variables (Fig. 1). Based on the standardized coefficients (interpretable in SD units), a one SD increment in violence exposure corresponded to psychopathology scores that were one-quarter standard deviation units higher ($\beta = 0.24$, medium effect size). Even after accounting for this effect, additional exposures to violence corresponded to further increments in psychopathology at 12 months and 48 months postpartum (small effect sizes). Thus, the differences between mothers in levels of psychopathology, as a function of early exposure to violence, get larger and grow across the first four years of a child's life

Hypothesis 4 Higher levels of MEEV were associated with higher pre- and postnatal maternal psychopathology, which predicted lower child adaptive functioning at 12 and 24 months, respectively. Four indirect effects were tested that connect these variables (Fig. 1 and Table 1). In addition to the simple mediation paths, an effect from MEEV to persistent maternal

Table 1. Descriptive statistics of CANDLE Study Birth Cohort.

Characteristic	Value, no. (%) $N = 1503$
Maternal race	
Black	990 (65.9)
White	482 (32.1)
Other/missing	31 (2.1)
Household income (pregnancy)	
$0–$4999	191 (12.7)
$5000–$9999	105 (7.0)
$10,000–$14,999	97 (6.5)
$15,000–$19,999	100 (6.7)
$20,000–$24,999	106 (7.5)
$25,000–$34,999	155 (10.3)
$35,000–$44,999	109 (7.3)
$45,000–$54,999	106 (7.1)
$55,000–$64,999	80 (5.3)
$65,000–$74,999	85 (5.7)
$75,000 or higher	234 (15.6)
Missing/unknown	135 (9.0)
Maternal education (pregnancy)	
<High school	184 (12.2)
High school diploma	709 (47.2)
Technical school	138 (9.2)
College degree	299 (19.9)
Graduate/professional degree	171 (11.4)
Missing/unknown	2 (0.0)
Child gender	
Male	676 (50.0)
Female	677 (50.0)
Missing/unknown	8 (0.1)
Maternal exposure to trauma[a]	
0 Types	857 (63.0)
1 Type	343 (25.2)
2 Types	122 (9.0)
3 Types	39 (2.9)
Variable	**Mean (SD)**
Maternal psychopathology[b]	
Pregnancy	50.51 (9.23)
1 Year	47.50 (10.33)
2 Years	46.35 (10.42)
3 Years	46.58 (10.55)
4 Years	46.55 (10.62)
Child milestones[c]	
1 Year	111.83 (11.95)
2 Years	111.14 (17.71)
3 Years	110.38 (15.78)
4 Years	106.68 (15.92)

[a]Maternal trauma exposure (during childhood) reported by mothers during pregnancy.
[b]Maternal reports of Global Severity Index on the Brief Symptom Inventory.
[c]Maternal reports of child developmental milestones on the Scales of Independent Behavior—Revised.

psychopathology from pregnancy to postpartum, to lower child adaptive functioning at 24 months was tested. Another multi-step indirect effect connected MEEV, to prenatal psychopathology, to child adaptive functioning, where stability was considered from 12 to 24 months.

Two of the four tested indirect effects were statistically significant, in addition to the total indirect effect from MEEV to adaptive functioning at 24 months. First, the prenatal programming effect was statistically significant, suggesting that maternal exposure to violence is associated with child adaptive functioning through higher levels of psychopathology during pregnancy. The simple postnatal programming effect was not statistically significant, though both pathways comprising this indirect effect were statistically significant. That said, the pathway from maternal exposure to violence, to prenatal maternal psychopathology, to postnatal psychopathology, to adaptive functioning at 24 months was statistically significant. Lastly, the effect from MEEV, to prenatal symptoms, to adaptive functioning at 12 and 24 months was not significant. Thus, the hypothesis of intergenerational effects of maternal exposure to violence on child adaptive functioning via psychopathology was supported, but only for pathways where maternal prenatal psychopathology was considered. Note, the total indirect effect from maternal trauma to adaptive functioning at 24 months via all possible pathways was significant, providing strong support for transmission of maternal trauma exposure to violence to child adaptive functioning, overall. All fully standardized indirect effects (Table 1) were small.

Clinically speaking, additional interpretation of results can be garnered from examining unstandardized coefficients (Supplemental file). Note, the SIB-R has a mean of 100 and SD of 15, like all standard score measures. Every additional type of violence exposure a mother reports corresponds to milestones that are 0.23 standardized scale points lower at 12-months, and up to 0.70 ($0.23 + 0.47$) points lower by 24 months, via maternal psychopathology. Thus, children of mothers who reported all three types of violence would have estimated standardized scores that are 2.13 units lower (i.e., 3*[−0.70]) than children of mothers who report no violence. Similar interpretations can inform the clinical significance of direct effects of maternal mental health (measured as a *t*-score, which means a mean of 50 and SD of 10) on child adaptive functioning. Compared to a statistically "average" mother, children of mothers who reported high levels of psychopathology (2 SDs above the mean, or a *t*-score of 70) would have adaptive functioning scores that were 1.54 points lower at 12 months of age (20*−0.077), and 2.10 points lower at 24 months of age (3.64 points lower total). For a child who is otherwise functioning well, this difference would likely not convey substantial weight. However, for a child who is in the sub-clinical range, these effects could potentially put children into the clinical range of functioning.

Covariates

The model-based associations between covariates and outcome variables will be discussed (see Supplemental file). Compared to Black mothers, White mothers reported lower levels of early exposure to violence. Higher levels of income were also associated with lower levels of early exposure to violence. In terms of maternal psychopathology, white mothers reported higher levels during pregnancy and at 12 months postnatal compared to Black mothers. Income was inversely related to psychopathology during pregnancy and again at 12 months. For child adaptive functioning, White mothers reported that their children had lower levels of adaptive functioning compared to black mothers at 12, 24, and 36 months. Preterm status and child sex were associated with child adaptive functioning at 12 months, where children who were more preterm and males (vs. females) had lower adaptive functioning, respectively.

DISCUSSION

The present study identified a developmental cascade linking maternal early exposure to violence (MEEV), perinatal maternal psychopathology, and child adaptive functioning across early childhood. Three-out-of-four research hypotheses were supported, and results are largely consistent with both prenatal and postnatal programming hypotheses. That is, greater maternal psychopathology during prenatal and postnatal periods predicted lower child adaptive functioning over time (H1), effects of MEEV on mother–child dyads were cumulatively observed from pregnancy to four years of life (H3), and MEEV in childhood predicted lower adaptive functioning in children via maternal psychopathology (H4). Contrary to our expectations, which were based upon evidence of transactional effects between maternal and child depression during this age period,[44] we did not find evidence for child effects on mothers (H2). Findings contribute to the extent and sizable prenatal and postnatal programming literature linking MEEV, maternal psychopathology, and child socioemotional functioning.[4–7,9,12] This study of infant and child adaptive functioning expands the scope of pediatric outcomes linked to MEEV and psychopathology, while also delineating multiple potential direct and indirect pathways through which adversity and stress appear to disrupt typical development during gestation and after birth. Understanding the factors associated with optimal child adaptive functioning informs interventions targeting a wider scope of developmental outcomes that consider timing and mechanisms of repeated effects, in addition to maternal early life experiences. Our findings suggest that early intervention during pregnancy for mothers with experiences of violence and psychopathology could be beneficial to both mothers and their children across numerous domains of development and time.

A handful of studies have linked MEEV and psychopathology during the peripartum period,[45] as well as perinatal maternal psychopathology and developmental outcomes during the first years of life.[9,13,46–52] The psychobiological mechanisms linking maternal psychopathology and child outcomes are extremely complex, including shared genetic risk and gene-environment correlation, prenatal programming effects via intrauterine environmental influences and pregnancy complications, as well as postnatal environmental processes, which include parenting and associated epigenetic processes, gene-environment interactions, and family-wide influences, as outlined in both human and animal studies.[4,8,9,22,53] While we are left to speculate on these mechanisms in the present study for the outcome of adaptive functioning in children, our intensive and longitudinal design reveals that this cascade is dynamic and cumulative, operating between mothers and children, repeatedly and across development. Clinically speaking, consideration of the effects of maternal history and psychopathology on child adaptive functioning should acknowledge that these effects are not static at the time of birth but persist and resonate over time.

In the current study, the influence of MEEV on child adaptive functioning appears to operate most strongly through prenatal (vs. postnatal) psychopathology. At face value, this finding would align most strongly with arguments pertaining to the prenatal origins of health and disease. That said, we are cautious to not over (or under) interpret differential mechanisms as a function of the reproductive phase (prenatal vs. postnatal) since both indirect effects were near the threshold of statistical significance. As always, replication studies of adaptive functioning that specifically consider intergenerational transmission via prenatal and postnatal mechanisms are warranted. In terms of timing, some studies suggest the relationship between maternal psychopathology and poorer child cognitive outcomes is greatest during the first year of life, but not during pregnancy or during childhood.[50,54] Other research suggests that chronic maternal depression, present during and beyond the child's first year of life, disrupts children's cognitive development, whereas time-limited maternal depression does not.[48,55] One of the main contributions of the present study is the intensive and repeated longitudinal design that delineates the pattern, direction, and timing of effects across development. Our findings imply similar contributions of maternal psychopathology during pregnancy and at 12 months, suggesting each period of exposure is uniquely important for children's adaptive functioning, though MEEV may be more implicated in the prenatal period.

Importantly, maternal psychopathology scores when children reached the age of four were contextualized by the number of types of childhood violence mothers experienced, with each increment corresponding to a higher maternal psychopathology score. Consistent with the "developmental cascade" framework, findings suggest that clinicians move away from considering maternal adversity and psychopathology as singular (albeit important) risk factors, at least in relation to child adaptive functioning assessed here. Rather, they are critical starting points in a multifaceted sequence that conveys pre- and postnatal risk, across layers of organizations, and throughout periods of early development. These findings further support the need to implement screening for maternal mental health and MEEV during pregnancy,[23] although efforts to reduce trauma-related mental health issues prior to conception may be even more advantageous.

A novel aspect of the current study is the investigation of the direction of effects between MEEV, maternal psychopathology, and child adaptive functioning across time. Prior research has illustrated a bidirectional relationship between perinatal maternal psychopathology and child socioemotional development.[7,44] Similar findings were not found for child adaptive functioning on later maternal psychopathology. That is, the direction of influence flowed from MEEV to maternal psychopathology to child adaptive functioning. Taken with previous research, this may suggest that child emotional and behavioral functioning provide a greater influence on maternal mental health than their adaptive functioning (at least in a sample of children without severe neurodevelopmental disorders). This pattern is in accordance with studies that suggest disruptive, hostile, aggressive, and otherwise negative child behaviors are particularly important influences on parents.[19] These difficult-to-manage behaviors, and the impact on parenting, may be one of the greater influences on caregiver stress.[56] Future longitudinal cohort studies examining the conditions under which child adaptive functioning influences maternal psychopathology, such as in the presence of other risk factors, are warranted. Additionally, socioeconomic status was widely represented, and majority of the current sample was Black (64.6%; Table 2). Prior research has focused primarily on White mothers and children, as well as families with middle to high socioeconomic status.[6] Expansion of these demographics enhances the generalizability of this putative cascade. Future studies that explore nuances in these differential timing effects are warranted, including moderator analysis whereby factors that increase maternal susceptibility to prenatal distress as a function of life circumstances are identified.

This study has several strengths including its sizable birth cohort research design, facilitating prospective measurements of maternal psychopathology before and after birth, and the consideration of both timing and direction of influences. This study also had some limitations. First, the current study had a single-informant design which has the potential to inflate correlation between variables. Therefore, further research should consider a multi-informant research design to confirm the results of the present study. Second, while the pregnancy cohort was a prospective design, mothers were asked to recall their own experiences of violence when they were children. Although the recall of major adverse events is thought to have limited recall bias and be accurate over a span of years,[57] it is possible that this recall tendency was influenced by their own psychological health and well-being at the time of reporting. Third, our study did not

Table 2. Estimates of indirect effects reported in fully standardized indirect effect size (SD units).

Indirect pathway	ES	95% CI	*p*
$Trauma_{Preg.}$[a] → $Symptoms_{Preg.}$[b] → AF_{12m}[c]	−0.015	(−0.029, 0.000)	0.048
Sum of indirect (for all paths leading → AF_{24m})	−0.020	(−0.036, −0.005)	0.010
$Trauma_{Preg.}$ → $Symptoms_{12m}$ → AF_{24m}	−0.006	(−0.013, 0.001)	0.074
$Trauma_{Preg.}$ → $Symptoms_{Preg.}$ → $Symptoms_{12m}$ → AF_{24m}	−0.008	(−0.015, 0.000)	0.041
$Trauma_{Preg.}$ → $Symptoms_{Preg.}$ → AF_{12m} → AF_{24m}	−0.006	(−0.012, 0.000)	0.051

[a]Maternal exposure to trauma during childhood, reported by mothers during pregnancy.
[b]Maternal reports of Global Severity Index on the Brief Symptom Inventory.
[c]Maternal reports of child Adaptive Functioning on the Scales of Independent Behavior—Revised.

assess maternal lifetime history of psychopathology. While our study did repeatedly assess maternal psychopathology, additional assessment of lifetime prevalence would have been beneficial to isolating prenatal programming from shared genetics. Fourth, our study did not collect data on service utilization, thus, it is unclear how health and social services intersect with the outlined patterns. Therapeutic services for mother–child dyads affected by violence have been outlined elsewhere.[58] Fifth, our findings must be considered in the context of selective attrition of higher risk participants, which can potentially bias estimates even if a missing data analysis technique is employed.[43] Finally, our study does not directly consider the multiple intermediary mechanisms linking MEEV, maternal psychopathology, and child adaptive functioning. Future studies with multi-method assessment protocols can shine light on the psychobiological pathways connecting these important variables.

CONCLUSION

There is a complex and dynamic developmental cascade linking MEEV to prenatal/postnatal maternal psychopathology and, subsequently, to children's adaptive functioning. This cascade is characterized by cumulative effects (i.e., associations that recur over time) that begin during gestation and occur across early childhood. Based on these findings, support of child cognitive, motor, and social development should incorporate an awareness of maternal violence exposure and mental health during pregnancy and postpartum, and their repeated contributions to adaptive functioning, given that these effects repeatedly manifest and accumulate as early as gestation. If replicated, findings from this study, specifically the timing and direction of effects, may enhance screening efforts during pregnancy and targeting of interventions aimed at improving child adaptive functioning and maternal psychopathology.

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D.T.B.: Conceptualization/design, acquisition of data, data analysis and interpretation, drafting/revising article, and approval of the final version. K.Z.L.: Conceptualization, acquisition of data, revising the article, and approval of the final version. S.S.M.: Data analysis and interpretation, drafting/revising article, and approval of the final version. F.T.: Acquisition of data, revising article, and approval of the final version. N.R.B.: Conceptualization, acquisition of data, revising the article, and approval of the final version.

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