Maternal Adverse Childhood Experiences and Infant Development

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abstract

OBJECTIVES: To examine the prenatal and postnatal mechanisms by which maternal adverse childhood experiences (ACEs) predict the early development of their offspring, specifically via biological (maternal health risk in pregnancy, infant health risk at birth) and psychosocial risk (maternal stress during and after pregnancy, as well as hostile behavior in early infancy).

METHODS: Participants were 1994 women (mean age = 31 years) and their infants, who were recruited in pregnancy as part of a prospective longitudinal cohort from 2008 to 2010. Pregnant women completed self-report questionnaires in pregnancy and postpartum related to psychosocial risk and a questionnaire about hostile behavior when their infant was 4 months of age. Health risk in pregnancy and infant health risk at birth were obtained from health records. Mothers completed the Ages and Stages Questionnaire when infants were 12 months of age.

RESULTS: Path analysis revealed that the association between maternal ACEs and infant development outcomes at 12 months operated through 2 indirect pathways: biological health risk (pregnancy health risk and infant health risk at birth) and psychosocial risk (maternal psychosocial risk in pregnancy and maternal hostile behavior in infancy).

CONCLUSIONS: Psychosocial risks in pregnancy, but not in early infancy, contribute to the transmission of vulnerability from maternal ACEs to child development outcomes in infancy via maternal behavior. Maternal health risk in pregnancy indirectly confers risk from maternal ACEs to child development outcomes at 12 months of age through infant health risk. Maternal health and psychosocial well-being in pregnancy may be key targets for intervention.

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WHAT'S KNOWN ON THIS SUBJECT: Experiencing adversity in childhood is associated with negative health and mental health sequelae into adulthood. Less is known about the mechanisms by which maternal childhood adversity is transmitted to offspring and its impacts on their health and well-being.

WHAT THIS STUDY ADDS: Maternal early childhood adversity cascades across generations, conferring risk for poor offspring developmental health. Both biological and psychosocial risk in pregnancy, as well as maternal hostile behavior in the postpartum period, link maternal childhood adversity to poor offspring development.

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Exposure to adversity in childhood, including maltreatment, caregiver mental illness, and poverty, can initiate a negative developmental trajectory of poor physical and mental health outcomes into adulthood.[1–6] With 12.5% of children in the United States having experienced 4 or more adverse childhood experiences (ACEs)[7] and the known detrimental impact of toxic stress on the developing brain,[8] exposure to ACEs is a major public health concern. A new and emerging line of research is an examination of the far-reaching intergenerational consequences of ACEs, transmitted from mother to offspring.[9–12] The authors of this research suggest that children of mothers who have been exposed to ACEs are at increased risk of a multitude of poor health and developmental outcomes,[13] including delayed achievement of developmental milestones and increased likelihood of parent-child relationship difficulties in infancy.[11,14] Despite these associations, research examining the mechanisms by which the transmission of the consequences of maternal ACEs to child outcomes occurs is severely lacking. Understanding the antecedents and mechanisms that lead to the intergenerational transmission of risks can facilitate the development of preventive interventions that aim to break continuities of risk across generations.

The proposed mechanisms of transmission of early adversity have been hypothesized across different theoretical models; however, scientific research is needed to explicate the contribution of these diverse pathways. The developmental origin of health and disease (DOHaD) theory purports a primarily biological mechanism of disease transmission during pregnancy,[15–19] including exposure to psychosocial risk (eg, prenatal depression, anxiety, and stress[20–25]). However, according to the bioecological model of child development, the role of proximal processes in the postpartum environment are also important for understanding mechanisms of transmission from maternal ACEs to child development outcomes. Proximal processes are patterns of reciprocal interactions among persons, objects, and symbols in the child's immediate environment, which serve as the mechanisms that drive the association between the child's predisposing characteristics and developmental outcomes.[26] Maternal psychosocial risk[9] and hostile maternal behavior[27] have been identified as 2 critical proximal processes.[28] Thus, with the current study, we aim to examine both biological and proximal influences of child development, commensurate with developmental theorizing.

In using a prospective longitudinal cohort of 1994 mothers and their infants, our overall aim with the current study was to investigate the biological and psychosocial pathways by which maternal ACEs are associated with child development outcomes at 12 months of age. Novel contributions include the multimethod examination of biological and psychosocial mediators, the use of cumulative risk indicators, and the examination of predictors in both the pre- and postpartum periods. Two hypotheses guided the current investigation. First, on the basis of the DOHaD theory, it was hypothesized that maternal exposure to ACEs would influence development indirectly via a biological pathway, specifically maternal pregnancy health risk and infant health risk at birth. Second, on the basis of the bioecological theory, it was hypothesized that distal risks (ie, maternal ACEs) would predict infant outcomes via proximal mechanisms, namely maternal psychosocial risk in early childhood and parenting behavior, with parenting behavior being the sole direct influence.

METHODS

Participants

The current investigation is part of a larger prospective pregnancy cohort (All Our Babies and All Our Families Study),[29] in which the aim is to examine the determinants of maternal and infant health outcomes and health care use. From May 2008 to December 2010, more than 3000 pregnant women were recruited from health care and laboratory offices in Calgary, Alberta, Canada. Inclusion criteria were (1) being <25 weeks' gestational age, (2) maternal age ≥18 years, (3) receiving prenatal care from health care and laboratory offices through Calgary Laboratory Services, and (4) being fluent in English. Approximately 84% of approached women in pregnancy agreed to participate. Participants included in this secondary analysis of the data were 1994 women (2909 eligible) who provided data about their history of ACEs before age 18 when their children were 36 months of age. As is typical in longitudinal studies,[30,31] there was 30% attrition over the course of the study, with mothers who had a higher income and higher level of education being more likely to remain in the study. Further information on attrition in the All Our Families cohort is reported elsewhere.[32]

Procedure

Participants completed questionnaires in pregnancy (<25 and 35 weeks), as well as in the postnatal period at 4, 12, 24, and 36 months of age. Time points used in the current study include <25 weeks' gestation, 4 months, 12 months, and the infant age of 36 months for the ACEs measurement. At <25 weeks' gestation, women completed self-reported surveys on their experiences during pregnancy

and, with consent, their surveys were linked to the electronic health records that were completed during the hospital admission for labor and delivery. Additional information on recruitment, eligibility, data collection, and questionnaires used in the study is described in detail elsewhere.[29,33] The study was approved by the institutional review board at the University of Calgary, and informed consent was obtained from all participants. Covariates included maternal age (in years), child age (in months), child sex (male = 1, female = 2), and maternal socioeconomic risk, which is described below.

Measures

Maternal ACEs

Mothers were asked to report on their ACEs by using a detailed questionnaire based on the original ACEs questionnaire.[4] This information was gathered when the child was 36 months of age and was retrospective in nature. Questions were used to assess exposure to adversity experienced before age 18, including physical, sexual, and emotional abuse, as well as the presence or absence of family dysfunction, by using 11 questions that mapped on to 8 categories of adversity (ie, emotional abuse, physical abuse, sexual abuse, exposure to familial substance abuse, mental illness, domestic violence, incarceration, and separation/divorce). In a method similar to that of previous studies,[4,6,34] mothers were categorized as having 0, 1, 2, 3, or 4 or more ACEs with a range from 0 to 4.

Pregnancy Health Risk

An antepartum risk assessment score[35] was completed by the responsible health care professional (eg, physician, nurse, or midwife) at birth. The Antepartum Risk Score is based on a 39-item questionnaire that is used to evaluate the medical risk of women giving birth[36] and includes prepregnancy risk factors, past obstetrical risk factors, problems in the current pregnancy, and other risk factors. In the current sample, 67.6% of women had 0 to 2 risks, 29.6% had 3 to 6 pregnancy risks, and 2.8% had more than 6 risks.

Pregnancy Psychosocial Risk

A 5-item cumulative outcome variable was constructed on the basis of maternal responses to questionnaires at <25 weeks' gestation. The items included in the maternal pregnancy stress index were derived as follows: (1) social support was based on a score of <70 on the Medical Outcomes Study Social Support Scale, which has been used in the literature to identify inadequate levels of social support[37]; (2) history of mental health difficulty was assigned to the risk category if a woman endorsed having had a mental health difficulty in the past by using a 1-question self-report; (3) stress was based on a score of >20 (1 SD above the mean) on the Perceived Stress Scale[38]; (4) anxiety was assigned to the risk category if a woman endorsed a score of 40 or higher on the Spielberger State Anxiety Scale, a cutoff that has been used to identify clinically significant levels of anxiety[37]; and (5) depression was assigned to the risk category if a woman endorsed a score of 10 or higher on the Edinburgh Postnatal Depression Scale, which is the cutoff score identified by the American Academy of Pediatrics for significant risk of postnatal depression.[39]

Postpartum Psychosocial Risk

The same methods for maternal postpartum stress score were employed at 4 months postpartum; however, only the social support, stress, anxiety, and depression indicators were included.

Infant Health Risk

A 5-item cumulative variable was constructed on the basis of information provided in the health record. The items included birth weight <2500 g, being born with a congenital anomaly, being admitted to the NICU, having an Apgar score <7, and have a gestational age <37 weeks.

Maternal Hostile Behavior

Maternal hostile behavior has been identified as a proximal predictor of child development outcomes and was thus included as a predictor. Four items from the parental hostile-reactive behavior subscale from the Parental Cognitions and Conduct Toward the Infant Scale[40] were used. This scale is a self-report measure of parental perceptions and behavioral tendencies toward their infant. Mothers rated their behavior on a 0 to 10 scale for each item, in which 0 represented "not at all what I think" and 10 represented "exactly what I think."

Maternal Socioeconomic Risk

A 4-item cumulative measure of maternal socioeconomic risk was obtained in pregnancy. The items included in the maternal socioeconomic risk index were derived as follows: (1) marital status was assigned to the risk category if a woman was single; (2) immigrant status was assigned to the risk category if a woman was not born in Canada; (3) education was assigned to the risk category if a woman reported that she had a high school education or less; and (4) income was assigned to the risk category if a woman reported that her household family income was <$40 000 per year.

Child Development Outcome

The Ages and Stages Questionnaire, Third Edition (ASQ-3)[41] is a parent-report screening questionnaire designed to screen for developmental delays in 5 domains of child

development, which include the communication, gross motor, fine motor, problem solving, and personal-social domains. Scores on each of the 5 domains of child development range from 0 to 60, and higher scores indicate better development.

Statistical Analysis

We used path analysis to test the direct and indirect effects of maternal ACEs on infant development at 12 months of age. Analyses were conducted by using MPlus 7.4[42] and SPSS version 22.0 (IBM SPSS Statistics, IBM Corporation, Armonk, NY).[43] Path analysis was performed with full information maximum likelihood estimation to include all participants with missing data in the analysis. The intermediary pathways were modeled at the same time, and within-time covariance terms were controlled. We also tested the indirect pathway between maternal psychosocial risk and child development outcomes via maternal hostility, as well as testing the indirect path between maternal physical health risk in pregnancy and child development outcomes via infant health risk at birth. The outcome variable "infant development outcome" at 12 months of age was operationalized as a latent variable within the model and was constructed on the basis of the 5 domains on the ASQ-3, including the communication, gross motor, fine motor, problem solving, and personal-social domains. Model goodness of fit was assessed by using the root mean square error of approximation (<0.05 indicates good fit) and Comparative Fit Index (>0.95 indicates good fit).[44]

RESULTS

Model Variable Correlations

Sociodemographic characteristics are presented in Table 1. Variable descriptions are shown in Table 2, and the bivariate associations between variables can be found in Table 3. Associations between all variables and covariates (maternal age, child sex, child age, and maternal socioeconomic risk) are also found in Table 3.

TABLE 1 Study Characteristics

Variable	Characteristics	*N* (%)
Maternal ethnicity	White	1631 (81.8)
	Black/African American	24 (1.2)
	Indigenous	9 (0.5)
	Asian	156 (7.9)
	Hispanic	31 (1.6)
	Multiracial/other	132 (6.7)
	Missing	31 (1.6)
Child sex	Female	938 (47)
	Male	1019 (51.1)
	Missing	11(0.6)
Maternal education	Some elementary school or high school	46 (2.3)
	Graduated high school	116 (5.8)
	Some college or university	245 (12.3)
	Graduated college or university	1250 (62.7)
	Some graduate school	51 (2.6)
	Completed graduate school	275 (13.8)
	Missing	11 (0.6)
Household income	$39 999 or less	117 (5.9)
	$49000–$79 999	406 (20.4)
	$80 000 or more	1394 (69.9)
	Missing	77 (3.9)
		Mean (SD)
Maternal age, y		31 (4)

TABLE 2 Descriptions of the Variables in the Study

Variable	Mean (SD) or Range
Adverse child experiences total score	1.41 (1.44) 0%–37.5% 1%–23.3% 2%–13.7% 4+%–14.8%
Pregnancy health risk	1.92 (1.99) 0–13
Pregnancy psychosocial risk	0.81 (1.15) 0–5
Communication	48.65 (10.80) 10–60
Gross motor	45.88 (15.88) 0–60
Fine motor	52.83 (7.73) 10–60
Problem solving	47.77 (11.18) 0–60
Personal-social	45.40 (11.39) 5–60
Maternal hostile behavior	2.73 (3.99) 0–30
Postpartum psychosocial risk	0.47 (0.94) 0–4
Maternal socioeconomic risk	0.33 (0.60) 0–3

Relation of ACEs to Infant Development Through Prenatal and Postnatal Health and Psychosocial Risk Variables

The pathways between maternal ACEs and infant development at 12 months of age, which were identified by using path analysis while controlling for maternal age, child age, child sex, and socioeconomic risk, were tested. In Fig 1, we present the individual paths this model comprises (standardized scores). Model fit for this model was determined to be excellent (Comparative Fit Index = 0.99, root mean square error of approximation = 0.02). Indirect effects for the association between ACEs and the infant development outcome were examined via maternal pregnancy health risk, infant health risk, maternal psychosocial risk in pregnancy, maternal postpartum psychosocial risk, and maternal hostile behavior. Post-hoc analyses were used to examine whether there was an indirect effect for the association between ACEs and infant development via socioeconomic risk.

TABLE 3 Pearson Correlations Among Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Maternal age	1													
2. Child sex	0.01	1												
3. Child age	0.04	0.03	1											
4. ACEs	−0.05*	0.02	0.004	1										
5. Pregnancy health risk	0.33**	−0.001	0.07*	0.07**	1									
6. Pregnancy psychosocial risk	0.01	0.03	0.07**	0.24**	0.13**	1								
7. Postpartum psychosocial risk	0.03	−0.02	0.07*	0.19**	0.12**	0.49**	1							
8. Infant health risk	0.00	−0.04	0.16**	−0.01	0.31**	0.06*	0.06*	1						
9. Maternal hostile behavior	−0.10**	−0.06	0.02	0.10**	−0.02	0.22**	0.30**	0.06	1					
10. ASQ-3: communication	0.002	0.15**	−0.010	0.02	−0.07*	−0.09*	−0.05	−0.14**	−0.10**	1				
11. ASQ-3: gross motor	−0.08*	−0.04	−0.007	0.02	−0.05	−0.06	−0.01	−0.12**	−0.07*	0.23**	1			
12. ASQ-3: fine motor	−0.02	−0.06*	−0.03	−0.01	−0.10**	−0.10**	−0.07*	−0.15**	−0.08*	0.31**	0.18**	1		
13. ASQ-3: problem solving	−0.03	0.05	0.05	0.03	−0.09**	−0.08**	−0.07*	−0.12**	−0.13**	0.41**	0.22**	0.41**	1	
14. ASQ-3: personal-social	0.01	0.09**	−0.02	0.01	−0.09**	−0.14**	−0.07*	−0.11**	−0.13**	0.47**	0.22**	0.43**	0.48**	
15. Maternal socioeconomic risk	−0.09**	0.01	0.07*	0.06*	0.06*	0.17**	0.13**	0.03	0.03	−0.01	0.07*	−0.11*	−0.12**	−0.07*

Asterisks indicate statistical significance.

* $P < .05$;

** $P < .01$.

There was no significant indirect effect via socioeconomic risk on infant development.

Bootstrapping was used to test the significance of indirect effects with 5000 bootstrap samples, which were considered significant when the 95% confidence interval (CI) did not include 0.[45] Results revealed that higher maternal ACEs were indirectly associated with lower offspring development scores through heightened psychosocial risk in pregnancy (β = −.03, P = .004, 95% CI:−0.06 to −0.01), but not through maternal pregnancy health risk (β = −.01, P = .14, 95% CI: −0.01 to 0.002), maternal hostile behavior (β = −.01, P = .23, 95% CI: −0.01 to 0.003), infant health risk at birth (β = .01, P = .10, 95% CI: −0.001 to 0.02), or maternal psychosocial risk in the postpartum period (β = .003, P = .43, 95% CI: −0.004 to 0.01).

Maternal psychosocial risk in pregnancy was associated with the infant development outcome at 12 months via maternal hostile behavior at 4 months of age (β = −.03, P = .004, 95% CI: −0.06 to −0.01). Furthermore, the indirect path from maternal ACEs to the infant development outcome via maternal psychosocial risk in pregnancy and subsequent maternal hostility in the postpartum period was also significant (β = −.01, P = .01, 95% CI: −0.01 to 0.002). Maternal health risk in pregnancy was associated with the infant development outcome at 12 months via infant health risk at birth (β = −.06, P < .001, 95% CI: −0.09 to −0.03). A second indirect path from maternal ACEs to the infant development outcome was also statistically significant via maternal pregnancy health risk and infant health risk at birth (β = −.01, P = .02, 95% CI: −0.01 to −0.001). The direct effect from ACEs to the child development outcome at 12 months of age was not significant (P > .05), and thus the effects in the current model operated exclusively through the biological health and prenatal stress pathways.

Overall, findings from the current study reveal that cumulative indices of biological and psychosocial risk from pregnancy and the postnatal period serve as mechanisms between maternal ACEs and child development outcomes at 12 months of age. In this article, we make 3 novel contributions to advance the literature. First, maternal ACEs were indirectly associated with poor child developmental outcomes via 2 distinct intermediary pathways: biological and psychosocial. Importantly, with these findings, we support the existence of both biological and environmental mechanisms of intergenerational transmission of risk. Second, we used cumulative indicators of maternal health and psychosocial risk in pregnancy and in the postpartum period, rather than using single

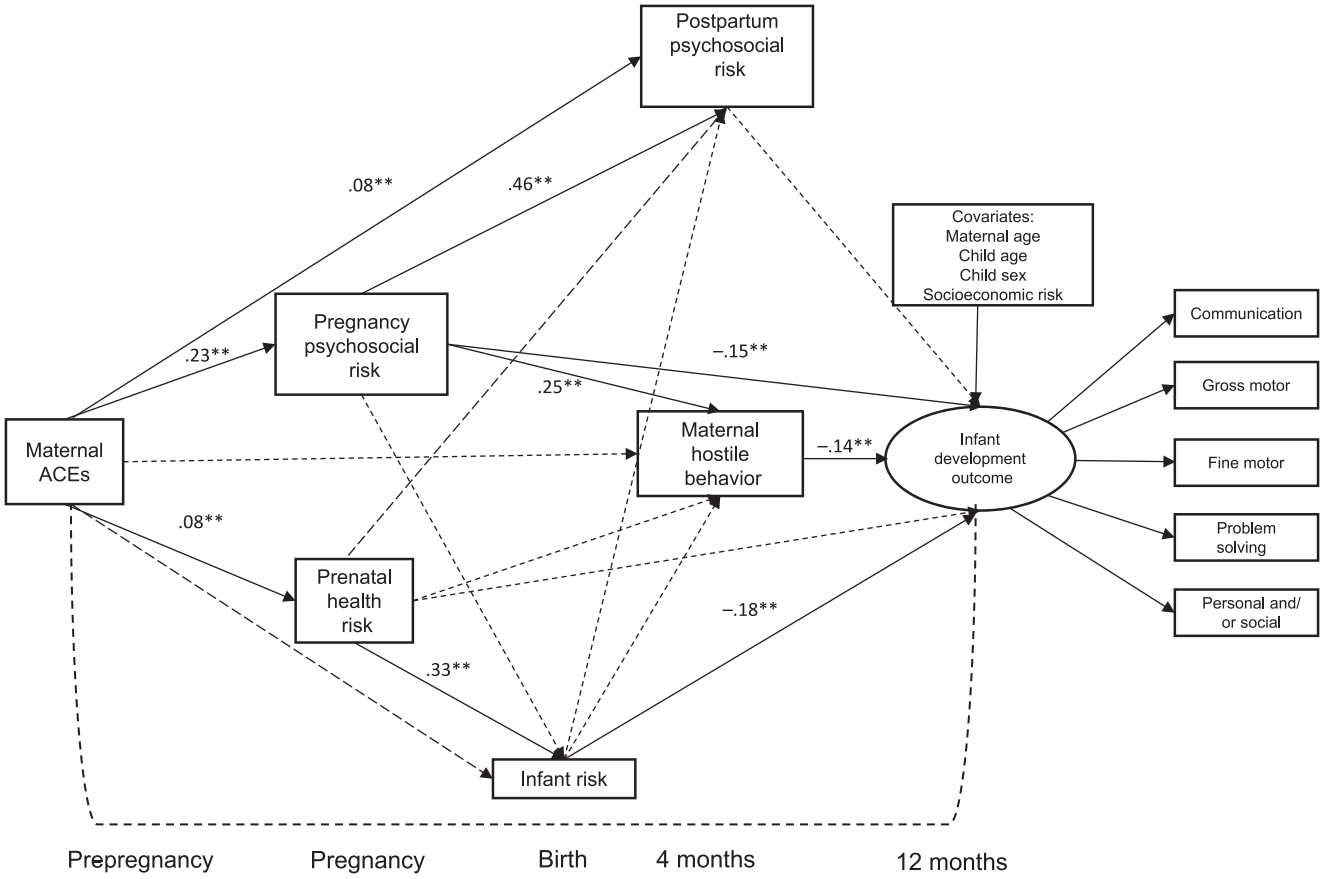

FIGURE 1

Transmission of maternal ACEs to infant development. Maternal ACEs predicting infant development at 12 months of age via biological health and psychosocial pathways are shown (standardized coefficients are presented). Solid lines indicate paths; dashed lines indicate nonsignificant paths (* $P < .05$; ** $P < .01$). A total of 12.2% of the variance was accounted for in the current model.

indicators such as maternal depression or infant birth weight that have been used in past research.[11] Lastly, maternal pregnancy stress mediated transmission in pregnancy, whereas maternal behavior is a partial mediator in the postnatal period. Each of these findings will be discussed in turn below.

DISCUSSION

Our results are supportive of the idea that there is an indirect biological pathway that accounted for the association between maternal ACEs and child development outcomes at 12 months of age. Specifically, mothers who experienced more adversity in childhood experienced more health risks in pregnancy and, in turn, had infants who were born with more infant health risks, which were associated with poorer developmental outcomes at 12 months. Thus, maternal early adversity has downstream consequences that perpetuate risks for the next generation. With these innovative findings, we support the biological intergenerational transmission framework[19] suggesting that transmission of maternal maltreatment occurs via biophysical and behavioral mechanisms during pregnancy, which influence fetal and infant development.

We also found support for a psychosocial pathway in explaining the association between maternal ACEs and infant developmental outcome. With our findings, we suggest that maternal stress in pregnancy had a significant influence on infant development, whereas postnatal maternal stress did not, even after controlling for socioeconomic factors. This finding may be an indicator that pregnancy is a more sensitive period, throughout which psychosocial risks are expressed in physiologic and behavioral responses that influence infant development outcomes. Furthermore, in partial support of our hypothesis, maternal hostile behavior partially mediated the association between maternal psychosocial difficulties in pregnancy and the infant development outcome, whereby maternal stress in pregnancy continued to be directly predictive of the infant development outcome. Overall, these findings are in line with both the DOHaD and bioecological theories, which leads

us to suggest that the transmission of maternal adversity to child development outcomes must be considered from both physiologic and environmental perspectives.[26] Both the prenatal and postnatal environments play a role in the transmission of maternal abuse to child development outcomes and provide windows for intervention to improve infant development outcomes.

With the current study, we extend the literature by simultaneously examining biological health risk in pregnancy, infant health risks, maternal stress in pregnancy, and maternal behavior as predictive mechanisms of the transmission of maternal ACEs to infant development outcomes, accounting for 12.2% of the variance in child developmental outcomes at 12 months. Researchers examining the transmission of emotional and physical health problems in infancy have found similar effect sizes,[12] whereas those examining behavior problems in older children have found that such transmission accounts for ∼25% of the variance.[9] Thus, we suggest that as children develop more complex abilities and behaviors, more variance may be accounted for by the transmission of health problems in infancy.

Findings from the current study should be interpreted in the context of some limitations. The majority of the mothers who participated in the study were well educated and had high household family incomes, reducing the generalizability of the findings to populations with extreme sociodemographic risks. Additionally, as is typical in longitudinal cohort studies,[30,31] we experienced an attrition range of 69% to 81%, which was based on the eligible population and follow-up time point.

CONCLUSIONS

The developmental cascade from maternal ACEs to child development outcomes appears to begin prenatally, revealing the need for early intervention and support for women with exposure to early adversity before and during pregnancy. Second, program leaders and decision-makers can improve on strategies for identification and support for women at risk, including programs that assist in early identification of children who have experienced adversity, to mitigate risk throughout their lifetime. Mothers who have a maltreatment history may demonstrate less sensitive parenting behaviors and increased hostility with their children,[46] and as such, interventions that normalize help seeking behavior for all parents may attract women at risk so that they can better understand barriers to maternal sensitivity and develop behaviors that have the potential to enhance the developmental outcomes of their children.[47] The authors of future research should examine whether different types, severity, and chronicity of exposure to ACEs play a role in the transmission from mothers to their offspring.

ABBREVIATIONS

ACE: adverse childhood experience
ASQ-3: Ages and Stages Questionnaire, Third Edition
CI: confidence interval
DOHaD: developmental origin of health and disease

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