Maternal History of Childhood Abuse and Risk of Asthma and Allergy in 2-Year-Old Children

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ABSTRACT

Objectives: Exposure to child abuse (CA) is associated with an increased risk of developing asthma and allergies; it is unknown if that risk is present across generations. This study investigated if 2-year-old children born to mothers with a history of CA were at an increased risk of receiving a diagnosis of asthma or allergies.

Methods: Data from 1,551 participants were collected as part of the All Our Babies (AOB) study, a prospective pregnancy cohort. During pregnancy, each woman provided information about her own history of CA, and at 24 months postpartum, she provided information about her child's medical diagnoses. Symptoms of maternal depression and anxiety were assessed during pregnancy and at 24 months postpartum.

Results: Unadjusted models showed that compared to children born to mothers without a history of CA, 2-year-old children born to mothers with a history of CA were more likely to have had a diagnosis of asthma (7.4% vs 4.2%, $p = .016$) or allergy (15.6% vs 9.2%, $p < .001$). Maternal symptoms of depression assessed in late pregnancy and symptoms of depression and anxiety at 24 months postpartum were significant mediators of the relationship between maternal CA and 2-year-old asthma diagnosis. Maternal symptoms of depression and anxiety assessed in late pregnancy were also significant mediators of the relationship between maternal CA and 2-year-old allergy diagnosis.

Conclusions: The results indicate that maternal exposure to CA is associated with increased risk of asthma and allergy in their 2-year-old children; symptoms of maternal depression and anxiety were identified as pathways linking the variables.

Key words: pregnancy, postpartum, maternal child abuse, asthma, allergy.

INTRODUCTION

Social and psychological factors have been associated with the onset and progression of asthma and allergies. Specifically, cross-sectional and prospective studies have demonstrated relationships between exposure to childhood abuse (CA) and increased prevalence and severity of asthma (1–7) and allergy (8,9) both in children and adults. The relationship between CA and higher prevalence of immunological disorders such as asthma and allergy is theorized to stem from psychological and physiological changes that occur as a result of abuse. For example, CA is associated with an increased prevalence of depression and anxiety (10,11), and a greater propensity to abuse substances (e.g., smoking) (12), each of which have been implicated in the subsequent development of asthma and allergy (13,14). Childhood abuse has also been associated with heightened physiological stress responding and alterations in immune system activity (13,15), which have been identified as plausible pathways for increasing risk of asthma and allergy (13,14).

Maternal Childhood Abuse and Risk of Asthma and Allergy in Children

A number of the psychological and behavioral variables that are elevated in women who have experienced CA have also been linked with an increased risk of asthma and allergy in children. For example, pregnant women with a history of CA are more likely to report symptoms of depression and anxiety than those without (16,17); in turn, symptoms of maternal depression and anxiety experienced during pregnancy and the postpartum period have

AOB = All Our Babies, **CA** = child abuse, **CEA** = child emotional abuse, **CI** = confidence interval, **CPA** = child physical abuse, **CSA** = child sexual abuse, **DV** = dependent variable, **IV** = independent variable, **M** = Mean, **PE** = point estimate, **SD** = standard deviation, **SE** = standard error

SDC Supplemental Content

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been associated with an increased risk of asthma and atopic diseases in children (18–22). Health practices also differ between pregnant women with a history of CA and those without, such that exposure to CA has been linked with an increased propensity to abuse substances during pregnancy, such as smoking (23). Substance use in pregnancy has been associated with worse child health outcomes; specifically, maternal smoking during pregnancy is associated with an increased risk of a child developing asthma (24,25).

In-utero and early life experiences influence the development of immune system structure and its functional responses (26). The psychological and physiological changes that result from exposure to CA could potentially alter maternal endocrine and immune system function, subsequently influencing fetal growth and development of the fetal immune system (13,14,27); these changes could serve as biologically plausible mechanisms that increase risk of asthma and allergy in a developing child. A small body of literature suggests that maternal CA is associated with worse child health, such that maternal history of CA has been associated with an increased risk of infants being born preterm, having lower birth weight, and experiencing more medical problems in the first year of life compared to infants born to mothers without a history of CA (28–31). Maternal interpersonal trauma experienced both in childhood and adulthood has been associated with higher child immunoglobulin E levels at birth, suggesting that interpersonal trauma may be associated with the development of an atopic phenotype (32). However, to our knowledge, no studies have examined potential relationships between maternal CA and an increased prevalence of child asthma and allergy.

Objectives of the Current Study

The current study examined if maternal CA was associated with an increased risk of diagnosis of asthma or allergy in 2-year-old children. We also examined potential maternal variables that may account for the associations between maternal CA and 2-year-old children's diagnoses, including maternal mental health (i.e., antenatal and postpartum symptoms of anxiety and depression) and smoking during pregnancy. We hypothesized that (1) a positive history of maternal CA would be associated with an increased risk of child diagnoses of asthma or allergy at 24 months postpartum; (2) women who reported CA would report higher symptoms of depression and anxiety and be more likely to report smoking in pregnancy; and (3) differences in mental health variables and smoking would mediate the relationship between maternal CA and child asthma and allergies.

METHODS

Participants

Data were collected as part of the All Our Babies (AOB) study, a prospective pregnancy cohort study established in 2008. Between 2008 and 2010 more than 3,000 pregnant women were recruited from the Calgary, Alberta area with the goal of investigating determinants of maternal and infant health outcomes and health care use. Participants included in this secondary analysis of the data were 1,551 women who gave birth to singletons and provided data about their own child abuse history and their child's asthma and allergy diagnoses. Overall, participants were highly educated, with 78% of the women having completed a university, college, trade level education, or higher. Most (82%) of the sample reported that they were white; the remaining sample was diverse, reporting that they were Chinese (3.9%), South Asian (2.6%), South American (1.9%), Filipino (2.0%), South East Asian (1.2%), African North American (1.4%), Arab (0.8%), Japanese (0.4%), West Asian (0.2%), First Nations (0.2%), or "other" (2.6%). Almost all women reported that they "currently had a partner" at baseline (99.2%) and approximately half were pregnant with their first child (49.1%). Characteristics of the sample are reported in Table 1.

Measures

Demographic Variables

Demographic information, including maternal education, income, race, and relationship status, was collected via a self-report questionnaire. Maternal education was dichotomized as completion of "university, college, or trade school" or higher (e.g., graduate school), compared with completion of "some" trade, college, or university, or high school, or less than high school. Annual income was categorized according to the low income cutoff for a family of four in the city of Calgary (<$40,000) and the median income in Calgary at that time (approximately $80,000), yielding three categories of less than $40,000, $40,000-$79,999, and ≥$80,000 (33). Race was classified as white or "other." Relationship status was categorized as married or common law compared to single or "other."

Maternal Childhood Abuse

A detailed questionnaire assessing childhood events was administered (16). Questions assessing interpersonal violence experienced in childhood were created specifically for the AOB study. Types of interpersonal violence assessed were derived from national and international classification and previous research studies, in additional to discussion with content experts in the field of child maltreatment (34–36). Questions assessed exposure to, the timing of, and perpetrator of different types of abuse, including physical, sexual, and emotional abuse. Specifically, participants were asked, "Have you ever experienced sexual abuse?," "Have you ever experienced physical abuse?," and "Have you ever experienced emotional abuse (including psychological or verbal?" Affirmative answers were followed by questions about the perpetrator ("By whom?") and timing of the abuse, including the questions: "How old you were you when this started?," "Has it stopped?," and "If yes, how old were you when it stopped?" Any abuse that occurred before the age of 18 was classified as having occurred in childhood. As exposure to any childhood sexual (CSA), physical (CPA), and emotional abuse (CEA) could be associated with a risk of asthma/allergies, we created a measure of any CA classified dichotomously as any physical, sexual, or emotional abuse that occurred when individuals were younger than 18 years (yes/no). We also examined associations with specific types of CA.

Child Asthma and Allergies

Information about child medical diagnoses were obtained from maternal report at the 2-year follow-up visit. In line with previous epidemiological research methodology, women were asked, "Has a health care provider ever told you that your child has (1) allergies or (2) asthma?" (7,37). Affirmative answers were followed up with specific questions, including "Please specify your child's allergy." A diagnosis of allergy or asthma was each coded dichotomously (yes/no). Parents who responded affirmatively that their child had a diagnosis of asthma or allergy were also asked if their child took

TABLE 1. Characteristics of the Sample According to Abuse Reported During Childhood

Variable	All Participants (N = 1,551) M (SD) or n (%)	No Self-Reported Child Abuse (n = 1,307) M (SD) or n (%)	Any Self-Reported Child Abuse (n = 244) M (SD) or n (%)
Age at baseline, M (SD), years	31.4 (4.3)	31.5 (4.3)	31.1 (4.9)
University graduate, yes	1211 (78.1)	1041 (80.2)	170 (70.0)***
Annual income, $			
0–39,999	85 (5.7)	68 (5.4)	17 (7.2)
40,000–79,999	321 (21.6)	263 (21.1)	58 (24.6)
>80,000	1078 (72.6)	917 (73.5)	161 (68.2)
Race, white	1271 (81.9)	1061 (81.7)	210 (86.8)
Current partner, yes	1531 (99.4)	1291 (99.5)	240 (99.2)
Child's sex, boy	800 (51.6)	677 (52.1)	123 (51.2)
Parity, first child	760 (49.0)	637 (49.3)	123 (51.0)
Preterm, yes	98 (6.3)	84 (6.6)	14 (6.0)
Pregnancy variables, M (SD)			
Smoking, days/wk	0.33 (1.41)	0.31 (1.36)	0.48 (1.64)
Depressive symptoms	4.9 (4.3)	4.7 (4.2)	6.2 (4.5)***
Anxiety symptoms	32.0 (8.9)	31.7 (8.7)	33.8 (9.5)***
Postpartum variables, M (SD)			
Breastfeeding, weeks	36.4 (17.6)	36.7 (17.5)	34.7 (18.2)
Depressive symptoms	7.7 (7.0)	7.2 (6.5)	10.6 (8.7)***
Anxiety symptoms	30.5 (8.4)	30.1 (8.2)	32.7 (9.1)***

M (SD) = mean (standard deviation).

* $p < .05$, ** $p < .01$, *** $p < .001$.

Complete data were not available for all participants for each variable. The percentages reported refer to the available data for the category. Depressive symptoms assessed with the Edinburgh Postnatal Depression Scale in pregnancy and the Center for Epidemiological Scales Depression Scale at 2 years postpartum; anxiety symptoms assessed with the Spielberger State Anxiety Scale in pregnancy and at 2 years postpartum.

a prescription medication and, if so, to specify the type of prescription medication taken. If a participant indicated that her child took a prescription medication for asthma or allergy (e.g., albuterol, salbutamol), this was noted and separate variables were created to indicate diagnosis plus medication use (yes/no) for each condition.

Childhood asthma before the age of 5 is difficult to diagnose; however, parental report of asthma medication use has been shown to be a reliable and valid measure of childhood asthma (38,39). Although research suggests that self-report of asthma and allergy symptoms can be influenced by mental health, such that higher symptoms of depression and anxiety are associated with higher self-reported symptoms, this finding does not seem to translate into clinician diagnoses (40). This means that whereas mothers with mental health problems may report more symptoms of asthma/allergy in their children, they are unlikely to report a higher number of actual diagnoses.

Maternal Depression

The *Edinburgh Postnatal Depression Scale* (*EPDS*) was used to measure depressive symptoms in the third trimester of pregnancy. The EPDS is a 10-item self-report questionnaire used to assess symptoms of depression in pregnancy and the postpartum period (41,42). Items were rated on a 4-point scale to produce a summative score ranging from 0 to 30, with higher scores indicating more depressive symptoms. The EPDS has been validated against interview schedules and other self-report instruments (43). It has well-documented reliability and validity (44). Cronbach alpha in the sample was 0.85.

The *Center for Epidemiological Scales Depression Scale* was used to assess maternal depressive symptoms at 24 months postpartum. The Center for Epidemiological Scales Depression Scale is a short self-report scale designed to measure depressive symptoms in the general population. Higher scores are indicative of more symptoms of depression (45). The scale has demonstrated reliability and validity in the general population and in women assessed during the postpartum period (46). Cronbach alpha in the sample was 0.88.

Maternal Anxiety

The *State Subscale of State-Trait Anxiety Inventory* (*STAI-S*) was used to assess antenatal and postpartum symptoms of anxiety. The STAI-S is a self-report instrument measuring state anxiety, defined as "subjective, consciously perceived feelings of tension and apprehension and heightened autonomic nervous system activity" (47). The STAI-S consists of 20 items rated on a 4-point intensity scale based on "how you feel right now." The scale has been validated against clinical interviews during the perinatal period and has acceptable sensitivity, specificity, and predictive validity in the identification of anxiety (48). Cronbach alpha was 0.92 in pregnancy and at the 24-month follow-up period.

Smoking

Maternal smoking was assessed in the third trimester via a single item: "Once you knew you were pregnant, how many days per week have you smoked cigarettes (on average)?" Responses ranged from 0 to 7 days/wk.

Birth Outcome Data

Information about gestational age and infant's sex was verified via the electronic medical record. Preterm birth was defined as delivery at less than 37 weeks' gestation.

Breast Feeding

At 12 months postpartum, participants were queried as to their breastfeeding history. Breastfeeding was quantified in number of weeks, with a range of 0 to 52 (women who reported breastfeeding at the 12-month follow-up were classified as 52 weeks). Approximately 80% of the participants (n = 1,232) who provided information about CA history and child asthma and allergy also provided data about breastfeeding history.

Procedure

Participants were recruited from primary care offices, community posters, word of mouth, and through a city-wide single-provider public health laboratory service (Calgary Laboratory Services) in Calgary, Alberta, Canada. A detailed description of the study methods and sample characteristics has been previously published (49).

This secondary analysis used data from the baseline questionnaire conducted in the second trimester, the second questionnaire collected in the third trimester, and follow-up questionnaires collected when the child was approximately 12 and 24 months of age. The questionnaire collected in the second trimester assessed demographic characteristics, including maternal age, race, education, household income, relationship status, and parity. The questionnaire collected during the third trimester of pregnancy (all between 34 and 36 weeks' gestation) asked about abuse experienced before 18 years of age, symptoms of maternal depression and anxiety, and smoking. The questionnaire collected when the child was 12 months of age asked about breastfeeding history. The questionnaire collected when the child was 24 months of age asked about child asthma and allergy as well as symptoms of maternal depression and anxiety. The study protocol was approved by the Institutional Review Board at the University of Calgary.

Initially, women who agreed to participate in the AOB cohort were asked to complete questionnaires twice during pregnancy and at 4 months postpartum. In total, 2,969 participants completed all three questionnaires. In January 2011, the research team recognized there was an opportunity to continue to follow-up participants and their children from the AOB study, and 12- and 24-month follow-up questionnaires were developed. Not all women recruited into AOB were eligible for the 12-month follow-up because their children were older than 13 months by the time funding and ethics approval were secured. Women whose child was 12 months (±1 month) at the time the 12-month follow-up study began and who had agreed to be contacted for future research were invited to participate (n = 1,573). For the present study, participants who completed questionnaires in the third trimester of pregnancy (when maternal child abuse was assessed), and at 24 months postpartum (when child asthma and allergy was assessed; n =1,551) were included in the study.

Statistical Analyses

Statistical analyses were performed using SPSS statistical software package (IBM SPSS Statistics, Version 22; Chicago, IL). χ^2 tests were used to identify if maternal sexual, physical, or emotional abuse and a combined CA score were associated with risk for diagnosis of asthma or allergies in children. Independent-samples t tests for continuous variables and χ^2 tests for dichotomous variables were used to test demographic, psychological, and smoking differences by maternal child abuse status and diagnosis of asthma or allergies. All data were entered continuously when assessing potential mediation.

Logistic regression was used to determine if maternal CA was associated with risk of asthma and allergies after adjusting for potential confounds. Covariates were chosen based on known associations with child asthma/allergy and included maternal education and household income (50,51), child's sex (51,52), parity (53), and maternal race (51,54). Covariates were held constant in all adjusted models. We also conducted an additional analysis adjusting for the potential influence of preterm birth (28,29). Analytic sample size varied slightly because a small number of women were missing data for at least one covariate (1.6%) or a mediator variable (between 0% and 2.5%).

Mediation Analyses

Next, potential mediators of the associations between maternal CA and asthma and allergies were investigated using a multivariate test of mediation and the macro PROCESS (55). Although longitudinal studies such as this one cannot conclusively investigate mediation, they can provide an indication as to whether variables related to maternal CA and child asthma/allergy are associated in a way consistent with mediation. Potential mediators that were tested included maternal symptoms of depression and anxiety (20) and maternal smoking (25,56) assessed in the third trimester of pregnancy and symptoms of depression and anxiety assessed at 24 months postpartum.

The mediation analyses examined the indirect effects of independent variables (IV; e.g., maternal CA) on a mediator variable such as depressive symptoms (path a), the mediating variable on one of the dependent variables (DV; path b) of asthma or allergies and the IV on the DV without the inclusion of mediators (path c). Finally, the relationship between the IV on the DV was considered after the mediator was included in the model (path c'). The products of coefficients (paths $a \times b$) were calculated using a bootstrapping procedure (n = 10,000 bootstrap resamples), which yielded a point estimate for the indirect or mediating effect. An example is seen in Figure 1. Mediators were considered significant if the confidence interval

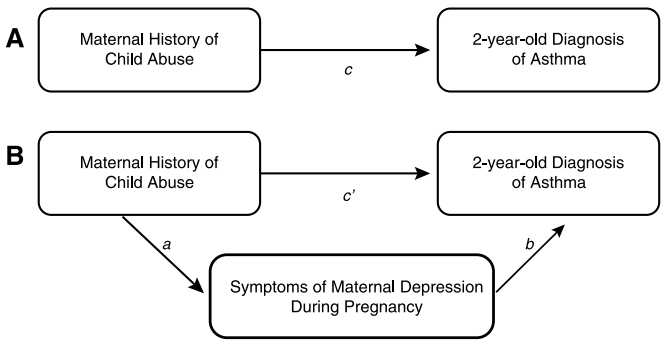

FIGURE 1. An example mediation model testing the relationship between maternal history of child abuse and 2-year-old children's diagnosis of asthma through symptoms of maternal depression during pregnancy.

(CI) around the point estimate did not cross zero. According to newer assumptions of mediation, the IV and DV do not need to be statistically related to investigate potential mediation (55).

RESULTS

Maternal CA History and Associations With Covariates and Mediators

In total, 244 women (15.9%) reported experiencing any physical, sexual, or emotional abuse in childhood. Eighty-nine women reported experiencing CPA (5.7%), 122 reported CSA (7.9%), and 151 reported CEA (9.7%). In the total sample, 150 women (9.7%) reported experiencing one type of CA, 71 (4.6%) reported two types of CA, and 23 (1.5%) reported having experienced all three.

Compared with mothers who did not report experiencing any abuse during childhood, women with a positive history of CA reported having less education. Women with a positive CA history also reported higher symptoms of depression and anxiety in pregnancy and the postpartum. There was a trend suggesting that women with a history of CA smoked more days per week on average than women without ($p = .083$). Data are presented in Table 1.

Child Asthma/Allergy and Associations with Covariates and Mediators

At the 2-year follow-up point, 69 mothers (4.4%) reported that their children have had a diagnosis of asthma, 66 (96%) of those reported that their child also had a prescribed medication for treatment of asthma. Removal of the three cases that had an asthma diagnosis without medications did not significantly alter any of the main findings. Due to the small number of children with a diagnosis of asthma without a prescribed medication, we included all cases in our final analyses.

Additionally, 158 mothers (10.2%) reported that their child has had a diagnosis of an allergy. Of those with a diagnosis of an allergy, 29 (18.5%) reported that their child had a prescribed medication. Reported allergies varied; most were food allergies (e.g., dairy, eggs, soy, seafood, peanuts, etc.; $n = 69$) followed by eczema ($n = 13$), animal dander ($n = 11$), antibiotics (e.g., penicillin, $n = 11$), and hay fever ($n = 4$). There was also one reported allergy to each of the following: lavender, dust, latex, and codeine. The remaining participants did not provide specific information about the specific allergy of which their child had a diagnosis ($n = 45$). Analyses were conducted to determine if the results differed when examining (1) the entire sample that were reported to have an allergy or (2) only the subgroup of children whose mothers reported they have had a diagnosis of an allergy and also listed the specific allergy type. With and without covariates in the model, maternal CA remained significantly associated with child allergy diagnosis (ps $< .05$) whether using (1) the entire group of children with a reported allergy or (2) only those children for whom we had data about their specific allergy. Given the consistency of findings, we retained the entire sample for further analysis.

Finally, we investigated how many mothers reported that their child have had a diagnosis of both allergy and asthma. Only 16 children had received both diagnoses; given this small number, we examined each outcome separately.

Children with asthma were more likely to be male, born preterm, and their mothers reported shorter breastfeeding duration. Mothers of children with asthma reported higher symptoms of depression and anxiety in pregnancy and the postpartum period; they were also more likely to report smoking in pregnancy. Children with allergies had mothers who reported shorter breastfeeding duration. In addition, mothers of children with allergies reported higher symptoms of depression and anxiety in pregnancy and higher symptoms of depression in the postpartum period. Data are presented in Table 2.

Maternal CA and Child Asthma

In unadjusted models, among children of women who reported any CA, the prevalence of asthma was higher (7.4% of children of women exposed had asthma vs 4.2% of children of women unexposed; $p = .016$). When examining by subtypes of abuse, maternal experience of CSA (8.2% of children of women exposed had asthma vs 4.3% of children of women unexposed; $p = .042$) was associated with asthma, whereas CPA and CEA were not (ps $> .35$). After adjusting for covariates (maternal education, annual household income, maternal race, parity, and child's sex), the relationship between any maternal CA and asthma remained significant ($p = .044$), whereas the relationship between maternal CSA and asthma was not ($p = .071$). Unadjusted and adjusted models are presented in Table 3. When we included preterm birth in the models, relationships between maternal CA (odds ratio [OR] = 1.79, $p = .061$) and maternal CSA (OR = 1.99, $p = .073$) and asthma were both attenuated.

Potential Mediators of the Relationship Between Maternal CA and Child Asthma

We next examined potential mediators of the relationship between maternal CA and child asthma. Individual mediators were tested in separate models and covariates (maternal education, annual household income, maternal race, parity, and child's sex) were controlled for in each analysis. After adjusting for covariates, symptoms of depression in pregnancy and depression and anxiety in the postpartum emerged as significant indirect pathways in the relationships between maternal CA and childhood asthma. Additionally, symptoms of depression and anxiety in pregnancy and depression in the postpartum emerged as significant indirect pathways in the relationships between

TABLE 2. Characteristics of the Sample According to Diagnosis of Asthma and Allergies in Child at Age 2

Variable	Asthma M (SD) or *n* (%)		Allergies M (SD) or *n* (%)	
	No	Yes	No	Yes
Age at baseline, M (SD), years	31.4 (4.4)	30.9 (4.2)	31.5 (4.4)	30.8 (4.4)
University graduate, yes	1163 (78.5)	48 (70.3)	1081 (77.8)	130 (80.9)
Annual income, $				
0–39,999	80 (5.6)	5 (7.9)	74 (5.5)	11 (7.3)
40,000–79,999	310 (21.8)	11 (17.5)	287 (21.5)	34 (22.7)
>80,000	1031 (72.6)	47 (74.6)	973 (72.9)	105 (70.0)
Race, white	1214 (82.1)	57 (83.8)	1143 (82.4)	30 (80.2)
Current partner, yes	1465 (99.4)	66 (98.6)	1377 (99.5)	154 (98.1)
Child's sex, boy	753 (51.3)	47 (68.5)**	715 (51.8)	85 (54.7)
Parity, first child	725 (49.6)	35 (54.1)	678 (49.5)	82 (51.9)
Preterm, yes	88 (6.3)	10 (15.3)**	90 (6.9)	8 (5.1)
Pregnancy, M (SD)				
Depressive symptoms	4.9 (4.3)	6.6 (4.8)**	4.9 (4.3)	5.8 (4.6) *
Anxiety symptoms	31.9 (8.8)	34.6 (9.3)*	31.9 (8.8)	33.5 (9.0)*
Smoking, days/wk	0.31 (1.37)	0.71 (2.09)	0.32 (1.38)	0.43 (1.58)
Postpartum, M (SD)				
Breastfeeding, weeks	36.8 (17.5)**	29.4 (18.2)	36.8 (17.5)	33.5 (18.1)*
Depressive symptoms	7.6 (6.8)	10.7 (9.3)**	7.6 (6.8)	9.2 (8.4)**
Anxiety symptoms	30.4 (8.3)	32.7 (9.2)**	30.5 (8.3)	30.9 (9.2)

M (SD) = mean (standard deviation).

Significance between group differences (e.g., children with asthma versus those without) are indicated with * $p < .05$ and ** $p < .01$. Depressive symptoms assessed with the Edinburgh Postnatal Depression Scale in pregnancy and the Center for Epidemiological Scales Depression Scale at 2 years postpartum; Anxiety symptoms assessed with the Spielberger State Anxiety Scale in pregnancy and at 2 years postpartum.

maternal CSA and childhood asthma. In other words, the direct effects of maternal CA and maternal CSA on asthma (path *c* to path *c*') were significantly reduced after accounting for differences in depression and anxiety in pregnancy and at 24 months postpartum (all *p*s > .061). Prenatal smoking did not emerge as a significant mediator in any models. Parallel mediation results are presented in Table S1, Supplemental Digital Content 1, http://links.lww.com/PSYMED/A338.

Maternal CA and Child Allergies

In unadjusted models, children of women who reported a history of any type of CA had higher prevalence of allergies (15.6% of children of women exposed had allergies vs

TABLE 3. Maternal Abuse During Childhood and Adolescence and Risk of Asthma and Allergies in Children at Age 2

Outcome	Type of Maternal Childhood Abuse	Unadjusted OR (95% CI)	Adjusted OR[a] (95% CI)
Child asthma			
	Any child abuse	1.96 (1.13–3.42)*	1.85 (1.02–3.35)*
	Child sexual abuse	2.10 (1.05–4.22)*	1.95 (0.92–4.11)
Child allergies			
	Any child abuse	1.83 (1.23–2.71)**	2.05 (1.36–3.08)**
	Child sexual abuse	1.84 (1.11–3.07)*	2.00 (1.18–3.40)**
	Child emotional abuse	2.00 (1.27–3.17)**	2.22 (1.38–3.57)**

* $p < .05$ and ** $p < .01$.

[a] Model adjusted for maternal education, annual household income, maternal race, parity, and child's sex.

9.2% of children of women unexposed; $p < .001$). When specific types of CA were examined, prevalence of allergies was elevated among children of women who reported CSA (16.4% of children of women exposed had allergies vs 9.6% of children of women unexposed; $p = .019$) and CEA (17.2% of children of women exposed had allergies vs 9.4% of children of women unexposed; $p = .003$). Child physical abuse was unrelated to allergy diagnosis ($p = .98$). After adjusting for covariates (maternal education, annual household income, maternal race, parity, and child's sex), relationships between experiencing any maternal CA, CSA, and CEA and child allergy diagnosis remained significant. Unadjusted and adjusted models are presented in Table 3. When we included preterm birth in the models, relationships between maternal CA (OR = 2.07, $p < .001$), maternal CSA (OR = 2.00, $p = .010$), maternal CEA (OR = 2.24, $p < .001$), and child allergy all remained significant.

Potential Mediators of the Relationship Between Maternal CA and Child Allergies

In mediation models, after adjusting for covariates (maternal education, annual household income, maternal race, parity, and child's sex), symptoms of depression and anxiety in pregnancy showed significant indirect pathways between maternal CA, CSA, and CEA and allergies. Depressive symptoms 24 months postpartum also significantly mediated the relationship between maternal CSA and allergies. In all cases, direct effects (path *c*) between maternal CA, CSA, and CEA and child allergies remained significant after mediators were included in the model ($ps < .026$), suggesting that other variables also influence these relationships. Postpartum anxiety and prenatal smoking did not emerge as mediators in any of the models. Parallel mediation results are presented in Table S2, Supplemental Digital Content 1, http://links.lww.com/PSYMED/A338.

Exploratory Analyses

A recent systematic review and meta-analysis concluded that breastfeeding was protective against the development of asthma (longer duration associated with less risk), with the strongest effects observed from the ages of 0 to 2 years (57). Results linking breastfeeding to allergies have been less conclusive (58). We therefore examined if breastfeeding influenced the relationship between maternal CA and child asthma or allergy.

There were no differences in weeks of reported breastfeeding between women with CA and women without CA history (M(SD) = 34.70 (18.20) vs M (SD) = 36.72 (17.50); $p = .14$). Results of logistic regression analyses showed that longer breastfeeding duration was associated with lower risk of asthma ($B = -.02$, SE = .01, $\chi^2 = 8.98$, $p = .003$) and allergy ($B = -.01$, SE = .01, $\chi^2 = 3.97$, $p = .046$) in 2-year-old children. After adjusting for covariates (maternal education, annual household income maternal race, parity, child's sex, and breastfeeding duration), maternal CA remained significantly associated with asthma [adjusted OR = 2.17, $p = .020$] and allergy [adjusted OR = 2.19, $p < .001$]. We investigated if breastfeeding duration was a significant mediator in the relationship between any of the CA variables and asthma or allergies and found that it was not in either case ($ps > .05$).

DISCUSSION

In this community sample of perinatal women and their 2-year old children, we found that maternal report of having experienced CA was associated with increased risk of having a 2-year-old child with a diagnosis of asthma or allergy. There were indirect pathways linking maternal CA and child asthma through symptoms of depression in pregnancy and depression and anxiety during the postpartum period. Symptoms of depression and anxiety in pregnancy were also identified as indirect pathways in the relationship between maternal CA and 2-year-old allergies. To our knowledge, this is the first paper showing a relationship between maternal history of CA and child diagnoses of asthma and allergy. Results also extend findings linking mental health symptoms in pregnancy to an increased risk of child allergy.

In line with previous findings, we observed that women with a CA history reported higher symptoms of depression and anxiety in pregnancy and the postpartum period (10,11). Maternal depressive symptoms in pregnancy have been associated with increased risk of wheeze and asthma in infants and children (59,60); similarly, we observed that symptoms of depression in pregnancy mediated the relationship between maternal CA and child asthma. High prenatal anxiety has also been associated with an increased prevalence of asthma in school-aged children (61). We also observed that maternal prenatal anxiety was a mediator of the relationship between maternal CSA and child asthma and maternal CA and child allergy. Changes in maternal physiology associated with CA and elevated symptoms of depression and anxiety may include alterations in the hypothalamic-pituitary-axis (HPA),sympathetic-adrenal-medullary system, and the immune system (62–65); these alterations have the potential to influence the developing fetal immune system and should be examined as candidate mediators in future studies (66).

Infant HPA and immune system development continues after birth and is influenced by postnatal variables (67). In our sample, postpartum depressive symptoms mediated the relationship between both maternal CA (any type and sexual) and asthma and maternal CSA and allergy. Maternal depression has been linked to child asthma severity through parental negativity, negative parenting behaviors, harsher and more punitive parenting, and low maternal responsivity (68–71). Since exposure to maternal depression and CA are themselves a documented risk factor for asthma, it could be that exposure to maternal behaviors

stemming from CA history results in an increased likelihood of developing asthma (1–6,8,9) due to changes in infant/child HPA responding and subsequent immunological shifts (66,72).

Work by our group has shown that trajectories of depression and anxiety are relatively stable throughout pregnancy and up to 1 year postpartum (73). Possibly, prenatal and postnatal psychological distress work together, such that biologically primed systems coupled with postnatal exposures such as repeated exposure to environmental influences (e.g., maternal postpartum depression) increase risk for the development of asthma and allergy (74). Future studies with larger sample sizes should examine potential interactions between maternal antenatal and postnatal mental health in the prediction of child asthma/allergy. Also notable is that maternal depression and anxiety are highly correlated (73). It is unclear why symptoms of depression and anxiety emerged as mediators in some models but not others, but it is possible that the underlying construct of maternal psychological distress drove associations, and a broader definition of maternal distress may have captured more variance than investigating depression and anxiety separately.

A small body of literature suggests that associations between types of child abuse may differentially affect adult physical health outcomes (75). Taken together, the studies suggest that recurrent CSA and CPA may be more associated with adult health outcomes than CEA (76–78). In line with these findings, our data showed that maternal CSA was associated with an increased risk for both child asthma and allergy. In contrast, we did not see associations between CPA and child health outcomes; however, this could have been due to the fact that the prevalence of CPA in our sample was relatively low. Additionally, we observed that maternal CEA was associated with increased child allergy. A recent meta-analysis concluded that CEA is associated with significant increases in adult mental health problems (79), a finding also observed in this study. In our sample, the relationship between CEA and child allergy was mediated by increased symptoms of depression and anxiety in pregnancy. To the extent that maternal mental health accounts for relationships between maternal CA and child health outcomes, CA types that deleteriously affect maternal mental health may increase risk.

In line with previous studies, children in our sample who were born preterm were more likely to have a diagnosis of asthma a 2 years of age (80). Notably, in light of the attenuation of the associations between maternal CA/CSA and child asthma when prematurity was included in the models, it is possible that differences in gestational age may serve as a mediator of this relationship. As our paper was mainly focused on maternal mediators or associations, we did not focus explore this question, but it is an area open for future exploration.

Another plausible explanation for the findings is that maternal CA and subsequent symptoms of depression and anxiety that are often associated with CA may have increased the prevalence of maternal asthma or allergy, relationships that have been demonstrated in previous studies (81). In turn, increased diagnosis of maternal asthma or allergy may have served as a mechanism that increased the risk of child asthma/allergy (82,83). Future studies should examine the potential for maternal diagnosis of asthma or allergy to serve as a pathway linking maternal CA and child asthma and allergy.

Although smoking during pregnancy was associated with an increased risk of child asthma in our sample, it did not emerge as a mediator. Additionally, shorter duration of breastfeeding was associated with an increased risk of a child receiving a diagnosis of asthma or allergy, but our findings showed that the relationship between maternal CA and child asthma or allergy were not accounted for by breastfeeding duration (57,58). Mean weeks of breastfeeding reported by women in our sample was high relative to World Health Organization–recommended guidelines, and it is possible that in samples with lower breastfeeding durations, additional relationships may have emerged (84).

Limitations and Future Directions

Findings from this study should be interpreted in light of several limitations. First, diagnosis of asthma in young children is difficult, as young children often experience wheezing and cough that is not directly attributable to asthma (85). Many children do not receive a definitive diagnosis before the age of 5 to 6 years (86). More confidence in the findings would be possible with outcomes documented by physician examination, coupled with testing for asthma (e.g., methacholine challenge) and allergy (skin-prick or serologic). However, such assessments are rarely practical in epidemiological investigations. Additionally, most of the participants in our study also had a prescribed medication for treatment of asthma, suggesting that their respiratory problems were severe in nature and therefore less likely to remit (85). Nevertheless, future studies should assess if the relationship between maternal CA history and child diagnosis of asthma remains when children are older than 5 years. Another limitation is that we did not have data from mothers regarding their own history of asthma and allergies, and it is possible that maternal diagnoses could have accounted for observed associations (82,83). Additionally, health outcomes beyond those assessed in this cohort may be associated with maternal history of CA, and a more comprehensive assessment in future studies would be helpful.

Mothers self-reported their abuse history using a scale that was not validated, and we did not have objective confirmation of the reports. Furthermore, the questions asked may not have adequately captured abuse that victims themselves may not have recognized as abusive. Serious and

repeated child abuse is less likely to be underreported than less severe abuse (87); however, it is still possible that CA reported in this sample underestimates the true prevalence in the cohort (87–89). Evidence increasingly suggests that there is a dose-response relationship between CA and physical health outcomes in adults (75,90), and research investigating relationships between maternal CA and child outcomes should also examine the possibility that higher frequency and severity of maternal abuse may increase risk of asthma/allergy in children. Finally, we did not have an assessment of current abuse of the children in the study, which has been associated with child asthma/allergy.

We assessed anxiety using the state measure of the STAI. Given our hypothesis that longer-term mental health conditions contribute to associations between maternal CA and child asthma/allergy, it would have been preferable to assess trait anxiety. That being said, the STAI-S has been shown to be highly stable from pregnancy through the first year postpartum (73), and is comparable to STAI-Trait in pregnancy (48). Finally, we acknowledge that this sample is unique in that they are highly educated, have a high annual income, are primarily white, and most were in stable relationships. Additionally, the rates of reported smoking in the sample were low. Given the links between a history of CA and lower socioeconomic profiles with regard to education, income, and higher levels of substance use, this sample may be uniquely resilient and not necessarily representative of the broader population (91). Future studies should examine if these findings hold in more socioeconomically and racially diverse samples.

CONCLUSIONS

Relationships between early life events and experiences on individual's later life health outcomes have been well recognized in the literature. Despite limitations of this study, we observed that maternal history of CA was associated with an increased risk of child asthma and allergies at 2 years of age. Findings from this study along with the previous research provide further evidence consistent with the view that adverse early environments affect health outcomes in the next generation of children (92). Maternal adverse childhood experiences may widen inequalities in the development and health status of their children.

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