Parental Adverse Childhood Experiences and Offspring Development at 2 Years of Age

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abstract

OBJECTIVES: The study objective was to determine if maternal and paternal exposure to adverse childhood experiences (ACEs) have a significant association with negative offspring development at 24 months of age in a suburban pediatric primary care population.

METHODS: A retrospective cohort study was conducted of 311 mother-child and 122 father-child dyads who attended a large pediatric primary care practice. Children were born from October 2012 to June 2014, and data were collected at the 2-, 4-, and 24-month well-child visits. Multivariable Poisson regression with robust error variance was used to model the relationship between self-reported parental ACEs and the outcomes of suspected developmental delay at 24 months and eligibility for early intervention services.

RESULTS: For each additional maternal ACE, there was an 18% increase in the risk for a suspected developmental delay (relative risk: 1.18, 95% confidence interval: 1.08–1.29). A similar trend was observed for paternal ACEs (relative risk: 1.34, 95% confidence interval: 1.07–1.67). Three or more maternal ACEs (versus <3 ACEs) was associated with a significantly increased risk for a suspected developmental delay that affected multiple domains. Similar effects were observed for early intervention services.

CONCLUSIONS: Parental ACE exposures can negatively impact child development in multiple domains, including problem solving, communication, personal-social, and motor skills. Research is needed to elucidate the psychosocial and biological mechanisms of intergenerational risk. This research has implications for the value of parental ACE screening in the context of pediatric primary care.

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WHAT'S KNOWN ON THIS SUBJECT: There is a large literature that reveals the strong association between adverse childhood experiences and negative physical and psychological health. Little is currently known about the association between parental early life adversity and trauma and effects on offspring development.

WHAT THIS STUDY ADDS: Among patients in a large pediatric primary care practice, a significant association was observed between past parental early life adversity and an increased risk for delayed offspring development at 24 months of age. Effects were observed across multiple developmental domains.

ARTICLE

Adverse childhood experiences (ACEs) and, relatedly, toxic stress, have been identified as salient public health issues in pediatrics that have life-course consequences.[1,2] These early life exposures, including abuse, neglect, and household dysfunction, are reported by an estimated 59.4% of the US population, ranging from a prevalence of 29.1% for household substance abuse to 7.2% for having an incarcerated family member.[3] Increased exposure to ACEs is strongly associated with a myriad of poor outcomes, such as poor school readiness, dysregulated hypothalamic-pituitary-adrenal axis function, and premature death.[4–6] The disruptive nature of ACEs can be deleterious to normal development,[7] and the negative effects may span multiple generations.[8] However, there remains little known regarding the specific effects of parental early life adversity (parental ACEs) on offspring development.

As evidenced recently in diverse populations, maternal ACEs can have negative effects on early offspring social-emotional function.[9–12] At 6 months of age, infants had increased negative affectivity with greater cumulative maternal trauma.[10] In addition, maternal ACEs were associated with a greater risk for social-emotional problems at 6 and 18 months of age.[11,12] Although the findings are mixed, these intergenerational associations may be partially mediated by maternal psychopathology, such as elevated depressive symptoms.[9,11,12] Maternal ACEs increase the risk for offspring early life adversity,[13] potentially increasing developmental vulnerabilities linked to insecure attachment and/or maladaptive neurobiological responses.[14–19] Evidence also reveals that protective factors (eg, parental sensitivity) may interrupt these effects,[18] supporting a notion that parental early life resilience may extend to offspring. Research is needed to determine the

effects of both maternal and paternal ACEs on multiple domains of child development and the mechanisms responsible for promoting and preventing intergenerational transmission.

Although still uncommon in pediatrics, some primary care practices have begun to measure parental ACEs routinely in a transition toward trauma-informed care. These efforts have generated promising results regarding feasibility[20] but have been implemented with limited empirical evidence regarding intergenerational effects. As those working within pediatric practices search for and implement novel approaches to address family adversity, elucidating the developmental risks attributed to parental ACEs and the related mechanisms (eg, maternal depression) will inform prevention and mitigation strategies. We conducted this study to examine the relationship between parental ACEs and global child development at 24 months of age within the context of a suburban pediatric primary care setting. Increased parental ACEs were hypothesized to have a negative relationship with offspring development at 24 months of age. We further hypothesized that the effects of parental ACEs on child development are mediated in part by maternal depressive symptoms and attenuated by parental early life protective factors.

METHODS

Study Design and Patient Population

This was a retrospective cohort study of parent-child dyads within a private pediatric primary care practice in the Portland, Oregon, metropolitan area. The study leveraged clinical data collected within the practice. The practice comprises 2 sites and 27 pediatricians who care for nearly 38 000 patients. Pediatricians asked mothers and fathers to

recall their exposure to ACEs and early life protective factors at the 4-month well-child visit and used the information to build parent rapport and generate referrals. Patients who remained active in the practice until the age of 24 months provided outcomes to examine the epidemiologic association between parental ACEs and offspring development at 24 months of age. Parental ACE screening was implemented with 8 physicians at 1 site and subsequently spread to all practitioners by 2015; the screening procedure is described in detail elsewhere.[20]

There were 1822 patients in the practice born during the study period of October 2012 to June 2014. The study cohort included 546 patients whose parent(s) were assessed for ACEs and 91.5% of whom were from 1 site, obviating the need to account for site-level effects in study analyses. Inclusion criteria were administrations of both the parental ACE measure and the 24-month child developmental screen. There were 92 mothers and 357 fathers in the study cohort who did not have an ACE measure because they were not present at the visit or provided incomplete data. Among those families screened for ACEs, 363 patients remained active in the practice and were screened for developmental delays at the age of 24 months. The sample included 311 mother-child dyads and 122 father-child dyads; parent type was not indicated in 30 records. There were 100 patients whose mother and father both completed the ACE measure.

Data Sources

Patient (child) sociodemographic characteristics and developmental screens and parental ACE and depression (maternal only) measures were abstracted from electronic medical records. All children in the study cohort turned 24 months

of age by June 2016. The study was approved by the Cincinnati Children's Hospital Medical Center Institutional Review Board.

Exposure and Mediator Variables

ACEs Scale Questionnaire

The ACE questionnaire was administered to parents at the 4-month well-child care visit and was used to identify past parental adversity and trauma. The ACE questionnaire comprises 10 items that are used to measure forms of abuse (physical, sexual, and emotional), neglect (physical and emotional), and household dysfunction (parents divorced or separated, mother treated violently, household member substance abuse, mental illness, or incarceration). The total score (range: 0–10 ACEs) was used as a continuous variable and was also dichotomized at multiple cutoffs (1–3), as in previous research.[5,6] The measure has good to excellent test-retest reliability and good internal consistency (Cronbach's α: 0.88).[21,22]

Resilience Questionnaire

The Southern Kennebec Healthy Start Resilience Questionnaire is a 14-item measure of early life resilience that is complementary to the ACE measure.[23] The measure includes protective characteristics related to the individual, family, and community that were present during childhood (eg, someone in my family cared about how I was doing in school). Higher scores indicate a greater degree of parental early childhood supports, and scores range from 0 to 14. Scores <13 (the first quartile of the distribution) were classified as low. Data from the current study revealed good internal consistency (Cronbach's α of 0.85), similar to findings in past research.[24]

Edinburgh Postnatal Depression Scale

The Edinburgh Postnatal Depression Scale (EPDS)[25] is a well-validated

10-item inventory that was administered at the 2-month well-child care visit to screen for elevated maternal depressive symptoms. Higher scores are associated with elevated depressive symptoms. We treated the score (range: 0–30) as a continuous variable in the mediation analysis. A clinical threshold ≥10 was used to indicate high risk for major or minor depression.[26,27]

Outcome Variables

Ages and Stages Questionnaire: Edition 3

The Ages and Stages Questionnaire: Edition 3 (ASQ-III)[28] was used to measure child developmental status at 24 months of age. The ASQ-III is a standardized screening tool completed by parents and contains 25 items to assess the developmental domains of communication, fine and gross motor skills, personal-social function, and problem solving. The ASQ-III has strong validity and reliability as a screening measure of developmental delay.[11] We dichotomized the ASQ-III domain scores as suspected delay versus normative. As in clinical practice, we defined a suspected delay (ie, requires further evaluation) as scores below the established cutoff (2 SDs below the mean) in any domain or within the monitor range (1–2 SDs below the mean) of at least 2 domains. Domain-specific analyses were performed by using the criteria of a score below the established cutoff or in the monitor range.

Early Intervention

A secondary child development outcome measure was the eligibility for early intervention (EI) services among children who had diagnostic evaluations (9–24 months) that indicated developmental delay. A universal release form was collected routinely in the practice, allowing follow-up on all EI referrals. There were 22 patients (7.1%) for whom EI eligibility was established; however,

additional evaluations may have occurred subsequent to the 24-month ASQ-III measure, precluding inclusion in the current study. Patients (n = 4) who were determined eligible for EI services because of a known physical condition at birth, including extreme prematurity and deafness, were excluded from the analysis of the EI outcome.

Statistical Analysis

Descriptive statistics were generated to summarize the parent-child demographic and risk characteristics of the study population according to maternal and paternal ACE exposure. χ^2 and Student's t tests were used to compare exposure groups (ACE ≥1 versus ACE = 0) by categorical and continuous outcome variables, respectively. Multivariable Poisson regression with robust error variance was used to model the dichotomous outcome of suspected developmental delay; this procedure provides a reliable estimate of relative risk (RR) for nonrare outcomes.[29] The following measures were evaluated for inclusion in the multivariable models on the basis of $P \leq .10$ or identification as a potential confounder: child sex, race, insurance type, and prematurity. Interaction terms (eg, ACE*Resilience Scores) were included in the models to test for hypothesized effect modification by parental early life protective factors. The analyses were performed in SAS version 9.4 (SAS Institute, Inc, Cary, NC).

Structural equation modeling performed in Mplus 7.0 was used to evaluate mediation by maternal depressive symptoms. To examine mediation in the context of continuous mediator (EPDS) and dichotomous outcome, we used maximum likelihood estimation with robust SEs. Missing EPDS values (17.4%) were handled by using the maximum likelihood parameter estimation.

RESULTS

Sample Characteristics

The sample of 363 unique patients was predominantly white (53.4%); however, 72 (19.8%) did not disclose race in the medical record. Nonwhite patients were largely Hispanic (59.8%). Public insurance was used by 30.9% of the sample, and the rate of prematurity was 8.3%. A suspected developmental delay was identified at age 24 months among 18.7% of patients, and 5.8% of patients were determined eligible for EI services.

The 1276 patients without parental ACE measure(s) relative to the 546 with ACE measure(s) were more likely to be white (33.0% versus 18.7%, $P < .01$) and have a private payer source (38.0% versus 28.6%, $P < .01$). The 183 patients who were excluded from the study sample because of loss-to-follow-up at 24 months were more likely to be nonwhite (35.0% versus 26.7%, $P < .01$) and have a public payer source (48.0% versus 52.0%, $P < .01$); the differences were nonsignificant for parental ACEs and all other variables examined.

Parental ACE Exposure

Among the 311 mothers and the 122 fathers in the study sample, 47.9% and 47.5% reported at least 1 ACE, respectively. These proportions were 19.3% and 19.7% for ≥2 ACEs and 12.5% and 10.7% for ≥3 ACEs. The presence of at least 1 parental ACE was associated with significantly different distributions of parent-child factors, including increased maternal depressive symptoms, decreased parental early life protective factors (Resilience Score), increased white race, and increased child eligibility for EI services (Table 1). The distributions of ACE types by parent are shown in Fig 1.

Suspected Developmental Delay

For each additional maternal ACE, there was an 18% increase in the risk for a suspected developmental delay (RR: 1.18, 95% confidence interval [CI]: 1.08–1.29). Relative to a simple linear parameter for total maternal ACEs, a natural cubic spline function had marginally better model fit and further suggested a dose response (Fig 2). A similar trend was observed for each additional paternal ACE (RR: 1.34, 95% CI: 1.07–1.67); however, the limited sample size precluded further assessment of dose response. All risk estimates were adjusted for child sex, preterm birth, and payer source. Child race was excluded in favor of more parsimonious models because this variable was missing from nearly 20% of records, was nonsignificant when available data were included in the models, and was relatively homogeneous.

TABLE 1 Study Sample Characteristics by Parental ACE Exposure

	All n = 311	Maternal ACEs ≥1 (n = 149)	Maternal ACEs 0 (n = 162)	P^a	All n = 122	Paternal ACEs ≥1 (n = 58)	Paternal ACEs 0 (n = 64)	P^a
Parental characteristics								
Payer, %				.22				.60
Public	29.6	32.9	26.5		23.8	25.7	21.9	
Private	70.4	67.1	73.5		76.2	74.1	78.1	
Depressive symptoms[b], %				<.01				—
High	4.2	7.4	1.2		—	—	—	
Low	78.5	76.5	80.3		—	—	—	
Missing	17.4	16.1	18.5		—	—	—	
Early life resilience[c], %				<.01				.03
Low	20.6	30.2	11.7		31.2	39.7	23.4	
High	76.2	65.1	86.4		66.4	55.2	76.6	
Missing	3.2	4.7	1.9		2.5	5.2	0	
Child characteristics								
Age (mo), mean ± SD	4.2 ± 0.5	4.2 ± 0.5	4.2 ± 0.5	.57	4.2 ± 0.5	4.1 ± 0.3	4.3 ± 0.5	.16
Race, %				.01				.01
White	54.7	59.7	50.0		51.6	65.5	39.1	
Nonwhite	25.4	18.8	31.5		27.1	19.0	34.4	
Missing	19.9	21.5	18.5		21.3	15.5	26.6	
Sex, %				.93				.47
Female	48.6	48.3	48.8		48.4	51.7	45.3	
Male	51.4	51.7	51.2		51.6	48.3	54.7	
Gestational age at birth, %				.08				.10
<37 wk	7.4	4.7	9.9		9.8	5.2	14.1	
≥37 wk	84.9	87.3	82.7		85.3	89.7	81.3	
Missing	7.7	8.1	7.4		4.9	5.2	4.7	
Developmental risk[d], %				.10				.33
Elevated (assess further)	19.0	22.8	15.4		15.6	19.0	12.5	
Normative	81.0	77.2	84.6		84.4	81.0	87.5	
EI eligibility[e], %				.03				.62
Yes	5.8	8.7	3.1		2.5	1.7	3.1	
No	93.9	90.6	96.9		97.5	98.3	96.9	
Missing	0.3	0.7	0		0	0	0	

—, not applicable

[a] P values were derived from χ² tests (categorical variables) or t tests performed on nonmissing data.

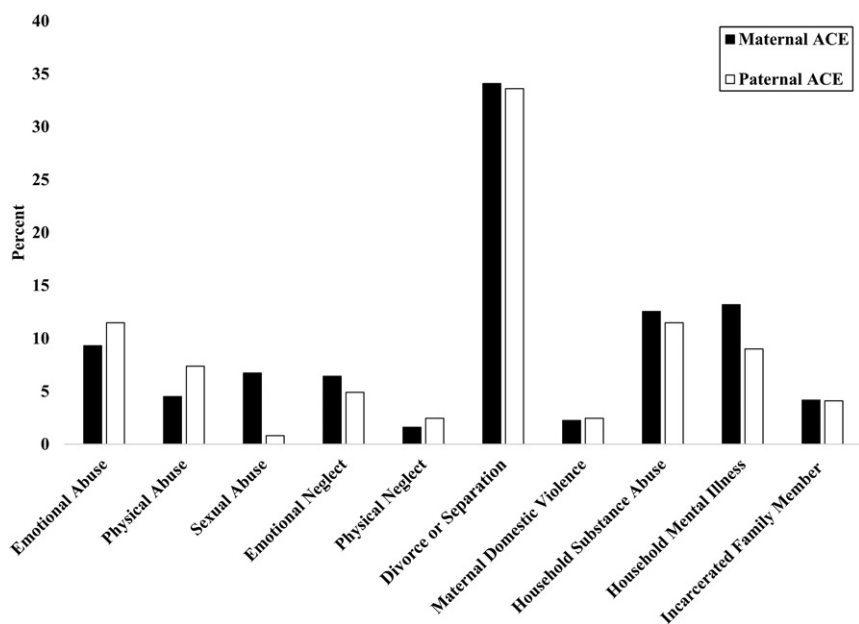[b] High depressive symptoms were defined as a score ≥10 on the EPDS.

[c] Low protective factors determined from total score on resilience inventory <13 (25th percentile of distribution).

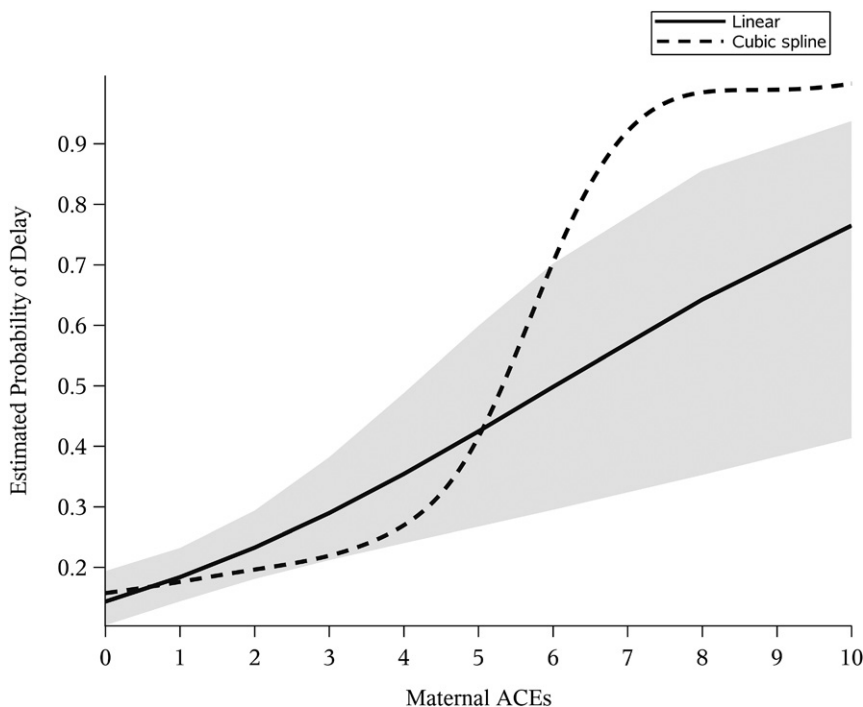[d] Suspected developmental delay as determined from the 24-mo ASQ-III.

[e] Evaluation performed by EI services; eligibility due to reasons other than physical conditions known to increase the likelihood for a developmental delay.

FIGURE 1
Distributions of parental ACE types. The distribution of ACE types was similar among mothers and fathers, with the marked exception of sexual abuse (reported more by mothers) and physical abuse (reported more by fathers). Experiencing parental divorce or separation was the most common ACE.

FIGURE 2
Relationship between maternal total ACE score and risk for suspected developmental delay. A higher number of maternal ACEs was associated with an increased probability of a suspected developmental delay. Both linear and spline (natural cubic) models indicated a dose-response effect; model fit was marginally improved with the spline. The gray band represents the 95% CI for the linear fit model.

Maternal ACEs ≥2 or ≥3 were associated with significant increases in the risk for offspring suspected developmental delay (Table 2); effects remained after adjusting for covariates and confounders. Mothers with a reported ACE exposure of ≥3 (versus <3) were 2.23 times more likely to have children with a suspected developmental delay. Note that conditioning on preterm birth resulted in negligible changes to effect estimates. Although the paternal sample was smaller, and the effect estimate was more variable, ≥2 paternal ACEs (versus 0–1 ACEs) was associated with a nearly 4 times higher risk of suspected developmental delay (Table 2). However, there was no effect on child developmental risk when comparing paternal ACEs of ≥3 to ACEs of <3.

As maternal ACE exposure increased, there was an increase in the domains of development affected (Table 3). Maternal ACE exposure was associated with a significantly increased risk for suspected developmental delay in the domain of problem solving. An exposure of ≥3 ACEs (versus <3 ACEs) was associated with a significantly increased risk for suspected developmental delay in the domains of communication and gross and fine motor skills. The limited sample size precluded domain-specific analyses for the paternal sample.

Although the interaction between maternal ACEs and maternal early-life Resilience Score was nonsignificant ($P = .564$), an interesting trend was observed in which maternal ACE exposure had a larger estimated effect on offspring risk for developmental delay when mothers reported low (RR: 1.20, 95% CI: 1.07–1.34) versus high (RR: 1.11, 95% CI: 0.88–1.40) protective factors.

EI Eligibility

A maternal exposure of ≥2 ACEs (versus 0–1 ACEs) was also associated with a significantly increased risk for offspring EI eligibility (diagnosed delay) by 24 months of age (RR: 3.33, 95% CI: 1.36–5.15). A similar effect was not

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TABLE 2 Adjusted Risk for Suspected Developmental Delay

	RR (95% CI)	
	Maternal (n = 311)[a]	Paternal (n = 122)[b]
ACE[c]		
≥1	1.25 (0.77–2.00)	2.47 (1.09–5.57)**
<1 (Ref)	—	—
≥2	1.78 (1.11–2.91)**	3.96 (1.45–10.83)***
<2 (Ref)	—	—
≥3	2.23 (1.37–3.63)***	0.82 (0.12–5.72)
<3 (Ref)	—	—
Child sex		
Female	0.41 (0.21–0.71)***	0.33 (0.14–0.78)**
Male (Ref)	—	—
Payer source		
Public	1.67 (1.05–2.67)**	0.87 (0.37–2.03)
Private (Ref)	—	—
Gestational age at birth		
<37 wk	1.70 (0.89–3.24)	7.76 (3.12–19.33)***
≥37 wk (Ref)	—	—

Ref, reference category. —, not applicable.
[a] n = 24 (7.7%) were excluded from analysis because of missing data on preterm birth.
[b] n = 6 (4.9%) were excluded from analysis because of missing data on preterm birth.
[c] Each cutoff-specific RR was adjusted for the other variables in the model. The RRs shown for child sex, payer source, and gestational age were derived from the model that used an ACE risk cutoff of ≥2.
** $P < .05$;
*** $P < .01$.

TABLE 3 Domain-Specific Developmental Risk (<1 SD Below Mean) by Maternal ACE Exposure

	Maternal ACEs		RR (95% CI)
	≥1 (n = 149), n (%)	<1 (n = 162), n (%)	
Communication	24 (16.3)	18 (11.1)	1.47 (0.83–2.60)
Gross motor	20 (13.5)	17 (10.6)	1.28 (0.70–2.35)
Fine motor	18 (12.1)	16 (9.9)	1.22 (0.65–2.31)
Problem solving	17 (11.6)	8 (5.0)	2.31 (1.03–5.20)**
Personal-social	19 (12.9)	17 (10.6)	1.22 (0.66–2.26)
	≥2 (n = 60), n (%)	<2 (n = 251), n (%)	
Communication	12 (20.3)	30 (12.0)	1.69 (0.92–3.11)*
Gross motor	12 (20.0)	25 (10.0)	1.99 (1.06–3.73)**
Fine motor	9 (15.0)	25 (10.0)	1.51 (0.74–3.06)
Problem solving	11 (18.3)	14 (5.7)	3.23 (1.55–6.76)***
Personal-social	9 (15.0)	27 (10.9)	1.38 (0.68–2.77)
	≥3 (n = 39), n (%)	<3 (n = 272), n (%)	
Communication	10 (26.3)	32 (11.8)	2.23 (1.19–4.16)**
Gross motor	9 (23.1)	28 (10.4)	2.23 (1.14–4.36)**
Fine motor	8 (20.5)	26 (9.6)	2.15 (1.05–4.40)**
Problem solving	6 (15.4)	19 (7.1)	2.17 (0.92–5.10)*
Personal-social	8 (20.5)	28 (10.4)	1.97 (0.97–4.01)*

There were n = 2, n = 2, n = 4, and n = 3 records missing domain-specific scores for communication, gross motor, problem solving, and personal-social, respectively.
* $P < .1$;
** $P < .05$;
*** $P < .01$.

observed for paternal ACEs and EI eligibility, but this may have been due in part to power limitations (RR: 1.59, 95% CI: 0.17–14.6).

Maternal Depression

The 2-month maternal measure of depressive symptoms was significantly associated with maternal ACE exposure ($P = .025$). However, the indirect effect of maternal depressive symptoms was nonsignificant ($P = .78$) in the relationship between maternal ACE exposure and offspring suspected developmental delay.

DISCUSSION

The study revealed evidence that both maternal and paternal ACEs are predictors of negative developmental outcomes in the next generation and can undermine parental mental health. As the number of maternal and paternal ACEs increased, there were corresponding significant increases in the risk for suspected offspring developmental delays. An accumulation of at least 3 maternal ACEs and 2 paternal ACEs were markers of more substantial risk to offspring development. The observed thresholds were similar to known effects of direct ACE exposure on multiple outcomes, including hypothalamic-pituitary-adrenal axis dysregulation, poor school readiness, depressive symptoms, and mortality.[4–6,30]

The current study builds on a fledgling literature largely limited to maternal ACEs and offspring social-emotional outcomes. The authors of 1 study demonstrated that a maternal history of physical abuse predicted an increased risk for offspring internalizing behaviors at 3 years.[31] The study was restricted to maternal ACEs of abuse. The authors of similar studies have examined the intergenerational effects of cumulative maternal ACEs and reported that the incidence of more ACEs was related to offspring emotional dysfunction at 6 and 18 months of age.[11,12] Although not measured in our study, the strength of parental ACE effects may be intensified by maternal physiologic stress response during pregnancy.[10] Evidence also reveals that these maternal stressors may program offspring developmental risk through epigenetic and related neurobiological responses.[15,17,19] With our findings, we contribute to a growing trauma focus in pediatrics by identifying parental ACEs as a predictor of negative offspring development across multiple domains, including communication,

motor skills, problem solving, and personal-social skills.

Contrary to the findings of other studies,[9,11,12,31] we did not show a mediated effect of parental ACEs on child development through maternal postpartum depression. Maternal depressive symptoms may affect offspring internalizing behaviors through unresponsive parenting (undermining attachment), revealing a cascade of risks subsequent to early maternal ACEs.[31] Although the null indirect effect of depression was unexpected, previous researchers have reported <24% of similar intergenerational effects attributed to the maternal depression pathway.[9] Further research is needed to elucidate pathways (psychosocial and/or biological) that underlie the association between parental ACEs and offspring development. Better knowledge of mechanisms will inform risk-stratification methods and more targeted intervention. Our lack of an indirect effect may have been due to the unique, suburban population evaluated and/or the absence of other key measures of parenting behavior and psychopathology not captured through a single depression screen.

The feasibility of integrating parental ACE measures into pediatric primary care has recently been demonstrated in the extant study population.[20] This work is grounded in the notion that intergenerational pathways can be interrupted by promoting family resilience and positive parenting behaviors. In our study, the findings were at most suggestive that parental early life resilience buffered the observed effects of parental ACEs on offspring development; however, we did not measure current resilience, a construct more germane to

pediatric-based intervention. This is important because new evidence reveals that bolstering parental sensitivity among other positive factors may mitigate developmental and health risks originating from adverse early environments.[18,32]

This study had several strengths, such as the inclusion of paternal and maternal ACE exposure data, the use of reliable and validated inventories, and the use of a large, pediatric primary care study population, supporting the generalizability of our findings to other suburban practices. There were also several limitations to this study. First, the analysis was conditioned on patients who remained in the practice at 24 months of age, creating a potential selection bias. Although the differences were small, patients who remained active were more likely to be white and use a private payer source. Second, the primary developmental outcomes were collected from a screening instrument and potentially overestimated prevalence. However, the inclusion of diagnostic outcomes from EI evaluations supported the observed effects from screening (ASQ-III) data. Third, previous research from this cohort reveals that although item-level ACE measures are feasible to collect, the total score may be underreported compared with aggregate-level ACE reporting (ie, identifying only a total ACE score).[20]

CONCLUSIONS

Parental early life adversity can be deleterious to normal offspring development, potentially having life-course implications. Therefore,

parental ACE measures may be relevant clinical assessment data in pediatric primary care. Specifically, the added knowledge of parental trauma history will facilitate the adoption of 2-generation approaches inherent to the increasingly recognized ecobiodevelopmental framework in pediatrics.[1,2,33] High parental ACEs could signal the need for early family-based interventions to mitigate developmental risks. Strategies need to be multifaceted and include addressing current adversity, family resiliency, and parenting behaviors.[13] Examples include collaboration with home visiting programs, behavioral health integration to better support at-risk children, and parental screening for mental health risks.[2,34,35] Future researchers should focus on expanding our understating of intergenerational mechanisms and the development and implementation of pediatric-based intervention strategies.

ABBREVIATIONS

ACE: adverse childhood experience
ASQ-III: Ages and Stages Questionnaire, Edition 3
CI: confidence interval
EI: early intervention
EPDS: Edinburgh Postnatal Depression Scale
RR: relative risk

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