Journal of Developmental Origins of Health and Disease

cambridge.org/doh

Intergenerational transmission of adverse childhood experiences via maternal depression and anxiety and moderation by child sex

N. Letourneau, D. Dewey, B. J. Kaplan, H. Ntanda, J. Novick, J. C. Thomas, A. J. Deane, B. Leung, K. Pon, G. F. Giesbrecht and the APrON Study Team

Department of Pediatrics, University of Calgary, Child Development Center, Calgary, AB, Canada

Original Article

Cite this article: Letourneau N, Dewey D, Kaplan BJ, Ntanda H, Novick J, Thomas JC, Deane AJ, Leung B, Pon K, Giesbrecht GF and the APrON Study Team. (2019) Intergenerational transmission of adverse childhood experiences via maternal depression and anxiety and moderation by child sex. *Journal of Developmental Origins of Health and Disease* **10**: 88–99. doi: 10.1017/S2040174418000648

Received: 6 October 2017
Revised: 3 July 2018
Accepted: 23 July 2018
First published online: 3 September 2018

Key words:
adverse; child behaviour; internalizing and externalizing behaviour; maternal anxiety; maternal depression

Address for correspondence:
G. F. Giesbrecht, Department of Pediatrics, University of Calgary, Child Development Center, #355, 3820–24 Ave, NW, Calgary, AB, Canada T3B 2X9.
E-mail: ggiesbre@ucalgary.ca

Abstract

Adverse childhood experiences (ACEs) of parents are associated with a variety of negative health outcomes in offspring. Little is known about the mechanisms by which ACEs are transmitted to the next generation. Given that maternal depression and anxiety are related to ACEs and negatively affect children's behaviour, these exposures may be pathways between maternal ACEs and child psychopathology. Child sex may modify these associations. Our objectives were to determine: (1) the association between ACEs and children's behaviour, (2) whether maternal symptoms of prenatal and postnatal depression and anxiety mediate the relationship between maternal ACEs and children's behaviour, and (3) whether these relationships are moderated by child sex. Pearson correlations and latent path analyses were undertaken using data from 907 children and their mothers enrolled the Alberta Pregnancy Outcomes and Nutrition study. Overall, maternal ACEs were associated with symptoms of anxiety and depression during the perinatal period, and externalizing problems in children. Furthermore, we observed indirect associations between maternal ACEs and children's internalizing and externalizing problems via maternal anxiety and depression. Sex differences were observed, with boys demonstrating greater vulnerability to the indirect effects of maternal ACEs via both anxiety and depression. Findings suggest that maternal mental health may be a mechanism by which maternal early life adversity is transmitted to children, especially boys. Further research is needed to determine if targeted interventions with women who have both high ACEs and mental health problems can prevent or ameliorate the effects of ACEs on children's behavioural psychopathology.

Introduction

Adverse childhood experiences (ACEs) are defined as a set of exposures to personal abuse, neglect and household dysfunction prior 18 years of age.[1] Based on the landmark work by Felitti and Anda,[1] a graded relationship has been observed between the number of ACEs experienced and adult health risk factors and problems. Extensive research has revealed the within-generation impact of ACEs on children and adults over the lifespan (for review, see McLaughlin[2]). Depression, inflammation and multiple metabolic risk markers such as obesity and diabetes are well-documented outcomes of exposure to ACEs.[3–6] Not only do ACEs significantly increase risk for a variety of mental health problems over the lifespan, they place women at increased risk for prenatal and postnatal depression and anxiety.[7–11] While prenatal and postnatal depression and anxiety have been linked to internalizing (e.g. anxiety, depression) and externalizing (e.g. aggression, hyperactivity) behavioural problems in their children,[12,13] growing evidence suggests that mothers' exposure to ACEs may also increase the child's risk for behavioural problems.[8,14] Understanding these associations may help guide intervention to address the steady increase in children's behavioural problems observed in first world countries,[15] that has recently placed these problems among the top five childhood disabilities.[16]

In contrast to the extensive evidence of within-generation impacts of ACEs on health, the intergenerational impact of ACEs on child outcomes has received less attention, with most studies examining associations between specific ACEs and children's behaviour, as opposed to using the omnibus ACE Questionnaire.[1] Indeed, the impacts of mothers' exposures to specific ACEs such as intimate partner violence and maltreatment[17–19] in childhood have well-documented consequences for children's mental health and behaviour. For example, in a sample of at-risk mothers ($n = 398$) recruited from community clinics, mothers' history of child abuse directly predicted socioemotional problems (e.g. difficultness) in their 6-month-

CAMBRIDGE UNIVERSITY PRESS

old infants, while household dysfunction indirectly predicted socioemotional problems.[8] Another study that utilized a measure of childhood adversity that overlapped with the ACE Questionnaire with the exception of questions about childhood physical and emotional neglect, found that more maternal ACEs were associated with more emotional health difficulties in 18-month-old infants. The only intergenerational study (that we could find) that utilized the ACE Questionnaire examined mothers ($n = 300$) who were also victims of intimate partner violence. When their children were 36 months of age, but not 12 or 24 months of age, maternal total ACE score was positively associated with more child behavioural problems, including anxiety, depression, aggressive behaviour, attention problems, and overall scores for internalizing and externalizing behavioural problems.[14] However, findings are limited by the concurrent exposure of children to both high-maternal ACE score and intimate partner violence. In summary, studies have not typically utilized broad exposure measures, like the ACE Questionnaire, to assess intergenerational impacts of maternal exposure on their children's behaviour and the one exception was confounded by concurrent exposed to violence. Thus, the intergenerational impact of ACES on children in community and normative samples remains to be explored.

Specific childhood adversities have also been linked to mental disorders in women, and especially to an increased risk for prenatal and postnatal depression and anxiety. Pregnancy increases the risk for depression and anxiety, which are often co-morbid during the perinatal period.[20–23] The prevalence of prenatal symptoms consistent with major depression is between 7 and 12%, with symptoms often persisting into the postnatal period resulting in rates of postpartum depression between 10 and 15%.[24–26] Findings are worse for anxiety, with prenatal rates between 18 and 25%[27] and postnatal rates between 13 and 40%.[28] A systematic review found a positive association between child sexual abuse and depressive symptoms in pregnancy, but an inconsistent association with postnatal depression.[9] For example, 25% of women exposed to child sexual abuse became depressed during pregnancy *v.* less than 2% of women not exposed.[7] Another systematic review identified that a history of abuse predicted both prenatal anxiety and depression.[10]

Connections between prenatal anxiety,[29,30] prenatal depression,[31–33] postnatal anxiety[28] and postnatal depression,[33,34] with children's behavioural problems are well-established. Prenatally, depression and anxiety activate the maternal hypothalamic–pituitary–adrenal (HPA) axis with resulting increases in cortisol that pass through the placenta to elevate the level of fetal cortisol concentration and dysregulate HPA axis development[35], which is a potential biological mechanism that increases risk of child behavioural problems.[11,36,37] Postnatally, mothers with depression characteristically demonstrate diminished sensitivity and responsiveness to infant needs,[38] as well as reduced affection during interactions with infants,[39] while anxious mothers are less likely to grant autonomy, and more likely to be withdrawn and disengaged.[40] These maternal behaviours are a potential psychosocial mechanism by which maternal postnatal anxiety and depression are linked to behavioural problems in children.[41–44] Taken together, prenatal biological and postnatal social processes may help explain why both externalizing and internalizing behavioural problems are more prevalent among children born to mothers who experience depression and anxiety and may be a mechanism by which maternal ACEs influence behavioural outcomes in the second generation.

A meta-analysis ($n = 21{,}709$ children) revealed that girls tend to experience more internalizing psychopathology and less externalizing psychopathology than boys.[45] Consistent with the Developmental Origins of Health and Disease (DOHaD) hypothesis,[46] differences in the prevalence and presentation of behavioural problems between boys and girls may be related to early life exposure to factors such as perinatal depression and anxiety.[47–50] Gendered behaviours are shaped by biological and sociocultural influences,[51] resulting in different patterns of behaviour for boys and girls.[45] Boys, compared with girls, display greater difficulty engaging with depressed mothers,[34] and are therefore more likely to demonstrate maladaptive behaviours and hampered emotional regulation.[52] The increased risk of girls demonstrating internalizing behavioural problems as a result of maternal depression may be explained by gender socialization during early childhood that encourages boys to be more aggressive and girls to be more passive than boys.[53,54] Whatever the reason, any examination of predictors of internalizing and externalizing behaviour needs to consider moderation by child sex.

Social support has long been established as a buffer to maternal perinatal depression[55,56] and anxiety[57] and as protecting children from the influence of postnatal depression[55,58,59] and anxiety.[57] Low social support is associated with prenatal anxiety[60] and partner support is associated with a lower risk of co-morbid postnatal anxiety and depression.[61] In contrast, low-income has also been implicated in postpartum depressive symptoms[56] and difficulties with finances and non-white ethnicity have been implicated in co-morbid postnatal depression and anxiety.[61] Thus, it is important to consider the socioeconomic and interpersonal influences on the intergenerational transmission of ACEs to children's behavioural problems.

In summary, there is a paucity of research examining the intergenerational relationship between maternal ACEs and children's behavioural outcomes, especially considering the potential mediating mechanisms of maternal symptoms of depression and anxiety or moderation by child biological sex. Thus, our objectives were to determine: (1) the association between ACEs and 2-year-old children's behaviour, (2) whether maternal symptoms of prenatal and postnatal depression and anxiety mediate the relationship between maternal ACEs and 2-year-old children's behaviour, and (3) whether the relationships observed in addressing objective 2 are moderated by child sex.

Method

Study design and sample

The data are from the Alberta Pregnancy Outcomes and Nutrition (APrON) longitudinal cohort study designed to examine the relationship between prenatal nutrition, maternal mental health, and child neurodevelopment and mental health. A detailed description of the APrON study design and methods has been previously published.[62] Briefly, a community sample of pregnant women residing in Alberta, Canada was recruited from maternity care and ultrasound clinics. Women were eligible if they had a gestational age less than 27 weeks and were 16 years of age or older. Women were not eligible if they were unable to complete questionnaires in English or were planning to move out of the study region within the next 6 months. Participants provided informed consent before participation in the study. The study was approved by both the University of Calgary Conjoint Health

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press

Research Ethics Board (ID E22101/REB14-1702) and the University of Alberta Health Research Ethics Biomedical Panel (Pro00002954).

Measures

Data were collected in each trimester of pregnancy and 3-, 6-, 12-, and 24-months postnatal. Data collection occurred between May 2009 and February 2015. Figure 1 presents the number of participants who provided data at each time point. The analytic sample was constrained to the sample of 907 with complete data on the ACE Questionnaire.

Exposure

Maternal ACEs

The ACEs Questionnaire[1] is a 10-item self-report measure that examines cumulative adverse experiences from birth to 18 years of age. ACE items assess five aspects of child maltreatment including physical, verbal or sexual abuse, and physical or emotional neglect. An additional five items assess family factors including domestic violence, marital dissolution, parental addiction, mental illness or incarceration. Responses are scored as '0 = no' or '1 = yes' with total scores ranging from 0 to 10, and higher scores indicating a greater accumulation of ACEs. Murphy *et al.*[63] demonstrated excellent convergent validity and internal consistency ($\alpha = 0.88$) of the ACE Questionnaire. In the current study, total ACEs ranged from 0 to 9. Because scores above 4 occurred infrequently, and in keeping with previous studies (e.g. Anda *et al.*[64]), scores above 4 were recoded to a value of 4 so that the final ACEs scores ranged from 0 to 4.

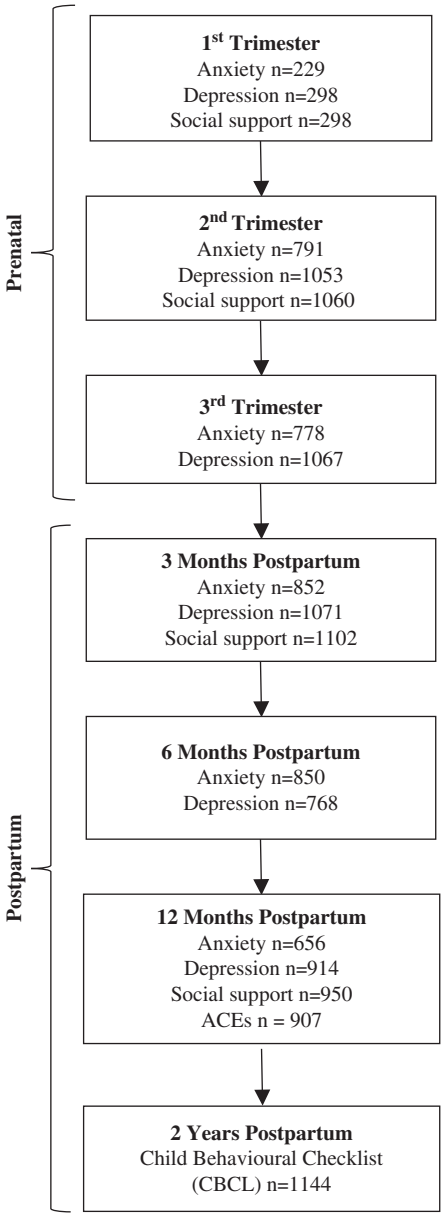

Fig. 1. Flow diagram for study time points and measurements. ACEs, adverse childhood experiences.

Mediators

Measures of maternal anxiety and depression were administered to mothers in each trimester of pregnancy and at 3-, 6-, and 12-months *postpartum*. These repeated measures were used to construct pre- and postnatal latent variables for anxiety and depression.

Maternal depressive symptoms

The Edinburgh Depression Scale (EDS)[65] is a 10-item, likert-type scale with total scores that can range from 0 to 30, and higher scores indicating more severe symptoms of depression. The EDS correlates well with other measures of depression[66] and has excellent internal consistency, Cronbach's $\alpha = 0.87$.[67]

Maternal anxiety symptoms

The Symptoms Checklist-90-Revised Questionnaire[68] is a 90-item self-administered questionnaire measuring psychological problems and symptoms of psychopathology. For this study, only the 10-item anxiety subscale was administered. Scores across items are summed and can range from 0 to 40, with higher scores indicating greater distress. Research supports the convergent validity[69] and internal consistency (Cronbach's $\alpha = 0.81$)[70] of this anxiety subscale.

Outcome measures

Child internalizing and externalizing behaviour

The Child Behaviour Checklist (CBCL) for children ages 1.5–5[71] is a 100-item parent report questionnaire measuring parent perceptions of their child behaviour within the preceding 2 months. The CBCL examines internalizing problems (emotionally reactive, anxious/depressed, somatic complaints and withdrawn), and externalizing problems (aggressive behaviour, attention problems). Internalizing scores can range from 0 to 72 and externalizing scores from 0 to 48, with higher scores indicating greater problematic child behaviours. The CBCL has excellent convergent validity[71] and displays high internal consistency($\alpha = 0.87$ and 0.89 for the internalizing and externalizing problem scales, respectively).[72] Maternal report of children's behaviour was measured at 2 years of age.

Moderator measure

Child sex was based on maternal report at birth.

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press

Covariates

Socioeconomic status and sociodemographics

Variables for maternal self-reported ethnicity, education and household annual income were obtained at the first study visit (first or second trimester). These variables were standardized before inclusion in all models. Child age at the 2 year assessment was also included in all models.

Maternal social support

Maternal perception of social support received during pregnancy and at 3-, and 12-months *postpartum* was assessed with the Social Support Questionnaire,[73] a four-item measure of emotional, affirmational, informational and instrumental support available. Responses were scored as 'Yes = 1' or 'No = 0' and summed. Thus, total scores ranged from 0 to 4, with higher scores indicating higher levels of support from family, friends and the community. The internal consistency for this measure is strong ($\alpha = 0.81$).[73]

Data analyses

Descriptive statistics were used to summarize the demographic characteristics of women and children who participated in the 2-year follow-up and all model variables. To address objective 1, Pearson correlations were conducted. To address objective 2 latent path analyses were conducted in Mplus Version 8[74] to test the indirect (mediation) effects of maternal ACEs on child internalizing and externalizing behaviours via maternal anxiety and depression. Latent path analysis maximized the use of the available longitudinal repeated measures and enabled the examination of mediation and moderation.[75] Mediation models were estimated using maximum likelihood and tests of indirect effects were evaluated using bias corrected 95% bootstrapped confidence intervals (CI) with 5000 resamples. This approach accounts for the non-normality of the indirect effects.[76] Indirect effects are supported when the CI does not contain 0. All results reported are standardized estimates and included the following covariates selected *a priori* and collected at time of behavioural assessment: maternal education, ethnicity and social support, household income, and child age and biological sex (when not included as a moderator as in objective 3). To address objective 3, multi-group analyses were conducted to determine whether there were differences between males and females with regard to the effects of maternal ACEs on child behaviour. The Satorra–Bentler scaled χ^2 difference test[1] (as described at www.statmodel.com/chidiff.shtml) was used to determine whether the indirect effects differed by child sex. Significant differences between models constrained to be the same for both sexes and models that were estimated freely for each sex provide evidence of moderation. Goodness of fit for models was assessed using the following criteria: χ^2 test > 0.05, Tucker–Lewis Index (TLI) $\geqslant$ 0.90, root mean square error of approximation (RMSEA) close to or below 0.05, and standardized root mean square residual (SRMR) < 0.05.[77,78] Although the χ^2 test is not a sensitive measure of model fit with large samples,[79] we report it for full disclosure.

[1]Note that model comparisons were made using Maximum Likelihood Estimation (MLE) with robust standard errors, which does not allow for use of bootstrapping; however, all confidence intervals reported are using MLE with bias-correction and bootstrapping.

Results

Descriptive findings

The mean age of mothers was 32.2 years. Most were married or in a common-law relationship, had some post-secondary education, had family income $\geqslant$ $70,000, were born in Canada, and were first time mothers (see Table 1). The mean age of children was 24.3 months, and more were male. Table 2 provides descriptive data on responses to the ACE Questionnaire and Table 3 provides descriptive data on the other predictors and outcomes. Missing data were less than 16.5% across all variables.

Because exposure information was available from only 907 mothers, we conducted comparisons between this sample and the remaining members of the cohort for which other data were available ($n = 237$). Women included in the analysis were more likely to be married (97.5 *v.* 94.5%, χ^2 (1) = 4.8, $P = 0.03$) and White (86.9 *v.* 81.9%, χ^2 (1) = 3.9, $P = 0.05$), but did not differ on education, annual household income, number of previous children, immigration to Canada, age, anxiety, depression or social support (all P's > 0.05).

Objective 1: association between maternal ACES and child behaviour.

Table 4 presents the bivariate correlations between the child behavioural outcomes and ACEs total and individual items scores, revealing that only externalizing behaviours are significantly correlated ($r = 0.08$) with ACEs total scores. One ACE item, on emotional neglect, was significantly correlated with internalizing behaviour ($r = 0.10$), while two ACE items, on emotional neglect ($r = 0.10$) and sexual abuse ($r = 0.09$), were significantly correlated with externalizing behaviour.

Objective 2: mediation by maternal depression and anxiety

Prenatal mediation of externalizing behaviour

The prenatal mediator model for child externalizing behaviours had good fit (see Table 5), accounting for 10.1% of variance in externalizing behaviour at 2 years. Unique indirect effects of maternal ACEs on child externalizing behaviours were not supported for either depression, 95% CI [−0.001–0.055], or anxiety, 95% CI [−0.010–0.085]. However, the 95% CI for the combined effects of prenatal depression and anxiety, [0.017–0.089], indicated that depression and anxiety together are associated with the effects of maternal ACEs on children's externalizing behaviours. Maternal exposure to ACEs was positively associated with both prenatal depression and anxiety, which were in turn positively associated with the child externalizing behaviour. Child sex was not reliably associated with externalizing behaviour, 95% CI [−0.022–0.101], indicating no overall difference in externalizing behaviour for boys and girls.

Prenatal mediation of internalizing behavior

The model for child internalizing behaviour had good fit (see Table 5), accounting for 10.9% of variance in internalizing behaviour at 2 years. A unique indirect effect of maternal ACEs on child internalizing behaviours was supported for anxiety, 95% CI [0.022–0.143], but not for depression, 95% CI [−0.033–0.020]. Maternal exposure to ACEs was positively associated with maternal prenatal anxiety, which was positively associated with child internalizing behaviour. There was also a combined indirect effect of prenatal depression and anxiety, 95% CI [0.030–0.116].

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press

Table 1. Socio-demographics of study participants ($n = 907$)

Variables	Frequency (%)	Male [n (%)]	Female [n (%)]
Marital status			
Single	23 (2.54)	13 (2.69)	10 (2.36)
Married	878 (96.8)	467 (96.49)	411 (97.16)
Missing	6 (0.66)	4 (0.83)	2 (0.47)
Educational level			
Below university degree	681 (75.08)	360 (74.38)	321 (75.89)
University degree	219 (24.15)	120 (24.79)	99 (23.40)
Missing	7 (0.77)	4 (0.83)	3 (0.71)
Annual household income			
Less than $70,000	142 (15.66)	88 (18.18)	54 (12.77)
$70,000 or more	754 (83.13)	389 (80.37)	365 (86.29)
Missing	11 (1.21)	7 (1.45)	4 (0.95)
Number of previous children			
None	535 (58.99)	286 (59.09)	249 (58.87)
1 or more	365 (40.24)	194 (40.08)	171 (40.43)
Missing	7 (0.77)	4 (0.83)	3 (0.71)
Born in Canada			
No	157 (17.31)	88 (18.18)	69 (16.31)
Yes	742 (81.81)	394 (81.40)	348 (82.27)
Missing	8 (0.88)	2 (0.41)	6 (1.42)
Infant sex			
Male	484 (53.36)	–	–
Female	423 (46.64)	–	–
Missing	0	–	–
Ethnicity			
Caucasian	788 (86.88)	419 (86.57)	369 (87.23)
Non-Caucasian	119 (13.12)	65 (13.43)	54 (12.77)
Missing	0	0	0
	Mean [SD]	Mean [SD]	Mean [SD]
Maternal age (years)	32.16 [4.03]	32.20 [4.07]	32.12 [3.98]
Child age (months)	24.28 [1.12]	24.27 [1.10]	24.29 [1.13]

Table 2. Adverse childhood experiences (ACE) Questionnaire scores ($n = 907$)

Variables	Frequency	Percentage
Total ACE Questionnaire score		
0	499	55.02
1	188	20.73
2	98	10.80
3	56	6.17
4	27	2.98
5	17	1.87
6	13	1.43
7	6	0.66
8	3	0.33
ACE Questionnaire items		
Emotional abuse		
No	797	87.87
Yes	110	12.13
Physical abuse		
No	848	93.50
Yes	59	6.50
Sexual abuse		
No	804	88.64
Yes	103	11.36
Emotional neglect		
No	804	88.64
Yes	103	11.36
Physical neglect		
No	891	98.24
Yes	16	1.76
Parental separation		
No	773	85.23
Yes	134	14.77
Battered mother		
No	773	85.23
Yes	134	14.77
Substance abuse		
No	771	85.01
Yes	136	14.99
Mental illness		
No	722	79.60
Yes	185	20.40
Criminal behavior		
No	889	98.02
Yes	18	1.98

Child sex was associated with internalizing behaviour, 95% CI [−0.149 to −0.025], indicating that boys had lower internalizing scores than girls.

Postnatal mediation of externalizing behavior

The postnatal model for child externalizing behaviour had good fit (see Table 5), accounting for 10.4% of variance in externalizing

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press

behaviour at 2 years. A unique indirect effect of maternal ACEs on child externalizing behaviour was supported for depression, 95% CI [0.007–0.076], but not for anxiety, 95% CI [−0.003–0.050]. Maternal exposure to ACEs was positively associated with maternal postnatal depression, which was positively associated with the child externalizing behaviour. There was also a combined indirect effect of postnatal depression and anxiety, 95% CI [0.010–0.078].

Table 3. Descriptive data on predictors and outcomes ($n = 907$)

Variables	Mean [SD]	Range
Prenatal social support	14.63 [2.28]	3–16
Postnatal social support	14.83 [2.09]	4–16
Anxiety symptoms 1st trimester	0.25 [0.31]	0–2.3
Anxiety symptoms 2nd trimester	0.22 [0.30]	0–1.6
Anxiety symptoms 3rd trimester	0.24 [0.33]	0–2.8
Anxiety symptoms 3 months pp	0.18 [0.30]	0–2.4
Anxiety symptoms 6 months pp	0.18 [0.29]	0–2.8
Anxiety symptoms 12 months pp	0.18 [0.30]	0–2.1
Depression symptoms 1st trimester	5.36 [3.93]	0–22
Depression symptoms 2nd trimester	4.65 [3.75]	0–17
Depression symptoms 3rd trimester	4.95 [3.76]	0–20
Depressive symptoms 3 months pp	4.64 [3.77]	0–19
Depressive symptoms 6 months pp	2.88 [3.03]	0–20
Depressive symptoms 12 months pp	3.96 [3.80]	0–21
Child internalizing behaviour (2 years)	4.80 [3.92]	0–24
Child externalizing behaviour (2 years)	9.07 [6.41]	0–37

pp, *postpartum*.

Postnatal mediation of internalizing behaviour

The postnatal model for child internalizing behaviour had good fit (see Table 5), accounting for 10.3% of variance in internalizing behaviour at 2 years. A unique indirect effect of maternal ACEs on child internalizing behaviour was supported for depression, 95% CI [0.001–0.063], but not for anxiety, 95% CI [−0.018–0.066]. Maternal exposure to ACEs was positively associated with maternal postnatal depression, which was also positively associated with children's internalizing behaviour. There was also a combined indirect effect of depression and anxiety, 95% CI [0.015–0.080].

Post-hoc analyses of mediator models

The patterns of results observed for mediator models raised further questions that we addressed in *post-hoc* analyses. Specifically, the model for prenatal exposures and child externalizing behaviour found that neither prenatal anxiety nor depression had unique effects, although they did have combined effects. Therefore, we ran two additional models with prenatal anxiety and depression as separate mediators to determine if either had individual effects when the other was not included in the model. The models for prenatal anxiety and depression were both supported, 95% CI [0.018–0.090] and [0.014–0.059], respectively, suggesting that although prenatal anxiety and depression are not unique mediators of maternal ACEs on child externalizing behaviour, they each contribute to transmitting the effects of maternal ACEs to child externalizing behaviour, with considerable overlap in their effects.

In addition, for child internalizing behaviour, we ran an additional model with the two previously identified mediators, prenatal anxiety and postnatal depression, to determine if either was a unique mediator after controlling for the other. The indirect effects of ACEs via postnatal depression was

Table 4. Correlations between adverse childhood experiences (ACEs) and child behaviour ($n = 907$)

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. ACEs Total	–												
2. Internalizing	0.04	–											
3. Externalizing	0.08*	0.61*	–										
4. Emotional abuse	0.56*	0.00	0.01	–									
5. Physical abuse	0.42*	0.05	0.03	0.54*	–								
6. Sexual abuse	0.44*	0.05	0.09*	0.18*	0.12*	–							
7. Emotional neglect	0.49*	0.10*	0.10*	0.42*	0.33*	0.17*	–						
8. Physical neglect	0.24*	0.01	0.01	0.15*	0.20*	0.21*	0.24*	–					
9. Parental separation	0.54*	0.04	0.00	0.22*	0.23*	0.20*	0.18*	0.20*	–				
10. Battered mother	0.27*	−0.01	0.00	0.27*	0.20*	0.11*	0.11*	0.13*	0.23*	–			
11. Substance abuse	0.53*	0.00	0.03	0.26*	0.16*	0.12*	0.13*	0.15*	0.24*	0.17*	–		
12. Mental illness	0.62*	0.00	0.03	0.29*	0.20*	0.16*	0.20*	0.16*	0.21*	0.16*	0.24*	–	
13. Incarceration	0.22*	0.00	0.03	0.07*	0.15*	0.10*	0.02	0.16*	0.12*	0.12*	0.18*	0.12*	–

*P-value <0.05.

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press

Table 5. Overall indirect effects of maternal adverse childhood experiences (ACEs) on externalizing and internalizing behaviour

Model	95% bias corrected CI for indirect effect	Model Fit
Prenatal Exposures		
ACEs → Depression → Externalizing	-0.001 – 0.055	Chi-Sq(41) = 43.4, $P = 0.37$
ACEs → Anxiety → Externalizing	-0.010 – 0.085	RMSEA=0.008 [0.000, 0.025] TLI: = 0.99 SRMR = 0.023
ACEs → Depression → Internalizing	-0.033 – 0.020	Chi-Sq(41) = 40.3, $P = 0.50$
ACEs → Anxiety → Internalizing	0.022 – 0.143	RMSEA=0.000 [0.000, 0.022] TLI = 1.00 SRMR = 0.023
Postnatal Exposures		
ACEs → Depression → Externalizing	0.007 – 0.076	Chi-Sq(41)=65.1, $P = 0.01$
ACEs → Anxiety → Externalizing	-0.033 – 0.050	RMSEA=0.026 [0.013, 0.037] TLI = 0.98 SRMR = 0.018
ACEs → Depression → Internalizing	0.001 – 0.063	Chi-Sq(41) = 67.1, $P = 0.006$
ACEs → Anxiety → Internalizing	-0.018 – 0.066	RMSEA=0.027 [0.014, 0.038] TLI = 0.98 SRMR = 0.019

CI = confidence interval; RMSEA = root mean square error of approximation which is a parsimony-adjusted index in which values closer to 0 represent a good fit and cut-off representing good fit is RMSEA < 0.08; TLI = Tucker–Lewis Index. A TLI of 0.95, indicates the model of interest improves the fit by 95% relative to the null model. Cut-off for good fit is TLI ⩾ 0.95; SRMR = standardized root mean square residual which is the square-root of the difference between the residuals of the sample covariance matrix and the hypothesized model. Cut-off for good fit is SRMR <0.08. Covariates included were: maternal education, ethnicity, social support, household annual income, and child age and sex.

supported, 95% CI [0.003–0.065], but the pathway via prenatal anxiety was not [− 0.008–0.088], suggesting that postnatal depression uniquely contributes to the effects of maternal ACEs on child internalizing behaviour even after accounting for the effects of prenatal anxiety.

Objective 3: moderation by child sex.

In multi-group analyses, all four mediation models had significantly better model fit when child sex was included as a grouping factor, suggesting that the mediator models may be moderated by child sex: prenatal model for externalizing (unconstrained $\chi^2(78) = 81.4$; constrained $\chi^2(108) = 251.8$; $\chi^2_{diff}(30) = 154.9$, $P < 0.001$); prenatal model for internalizing (unconstrained $\chi^2(78) = 72.4$; constrained $\chi^2(108) = 247.9$; $\chi^2_{diff}(30) = 158.7$, $P < 0.001$); postnatal model for externalizing models (unconstrained $\chi^2(78) = 88.4$; constrained $\chi^2(108) = 269.5$; $\chi^2_{diff}(30) = 160.6$, $P < 0.001$); and postnatal model for internalizing (unconstrained $\chi^2(78) = 88.4$; constrained $\chi^2(108) = 269.5$; $\chi^2_{diff}(30) = 160.6$, $P < 0.001$). Accordingly, all mediator models were re-estimated with child sex as a grouping factor. Model fit for all models was excellent, RMSEA = 0.03 [0.01, 0.05], TLI = 0.97–1.0, SRMR = 0.03–0.04, with the exception of χ^2, which had $P < 0.05$ for the postnatal models.

Prenatal model of externalizing behaviour

Similar to what was observed for the model without grouping by sex, all 95% CIs for the indirect effects of ACEs on externalizing behaviour included 0: girls via prenatal depression [− 0.006–0.067], and prenatal anxiety, [− 0.018–0.107], and boys via prenatal depression, [− 0.026–0.067], and prenatal anxiety, [− 0.017–0.163]. However, when the effects of the two mediators were combined, the model did support a moderated mediator effect for boys 95% CI [0.018–0.144], but not for girls, [− 0.003–0.099].

Prenatal model of internalizing behavior

The 95% CI for the unique indirect effect of ACEs on internalizing behaviour via prenatal depression was not supported for girls [− 0.036–0.029] or boys [− 0.079–0.025]. In contrast, the 95% CI for prenatal anxiety supported a unique mediating effect of anxiety for boys [0.035–0.265], but not girls [− 0.001–0.140]. As shown in Fig. 2, maternal exposure to ACEs was associated with increased prenatal anxiety, which in turn was related to increased internalizing behaviour in boys. The combined indirect effects of prenatal anxiety and depression on internalizing behaviour were significant for both girls [0.002–0.111] and boys [0.045–0.209].

Postnatal model of externalizing behaviour

The 95% CI for the indirect effect of ACEs on externalizing behaviour via postnatal depression was supported for boys, [0.002–0.113], but not girls [− 0.009–0.110]. There was no support for postnatal anxiety as a unique mediator for either girls [− 0.087–0.043], or boys, [− 0.048–0.084]. Furthermore, the combined indirect effect of postnatal anxiety and depression on externalizing was not significant for girls [− 0.036–0.076], but was significant for boys [0.011–0.111]. As shown in Fig. 3, maternal exposure to ACEs was associated with increased postnatal depression, which was related to increased externalizing behaviour in boys. Although the individual path from maternal depressive symptoms to externalizing behaviour in boys was only marginally significant ($\beta = 0.23$, $P = 0.069$), the indirect effect was significant for boys, as described above.

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press

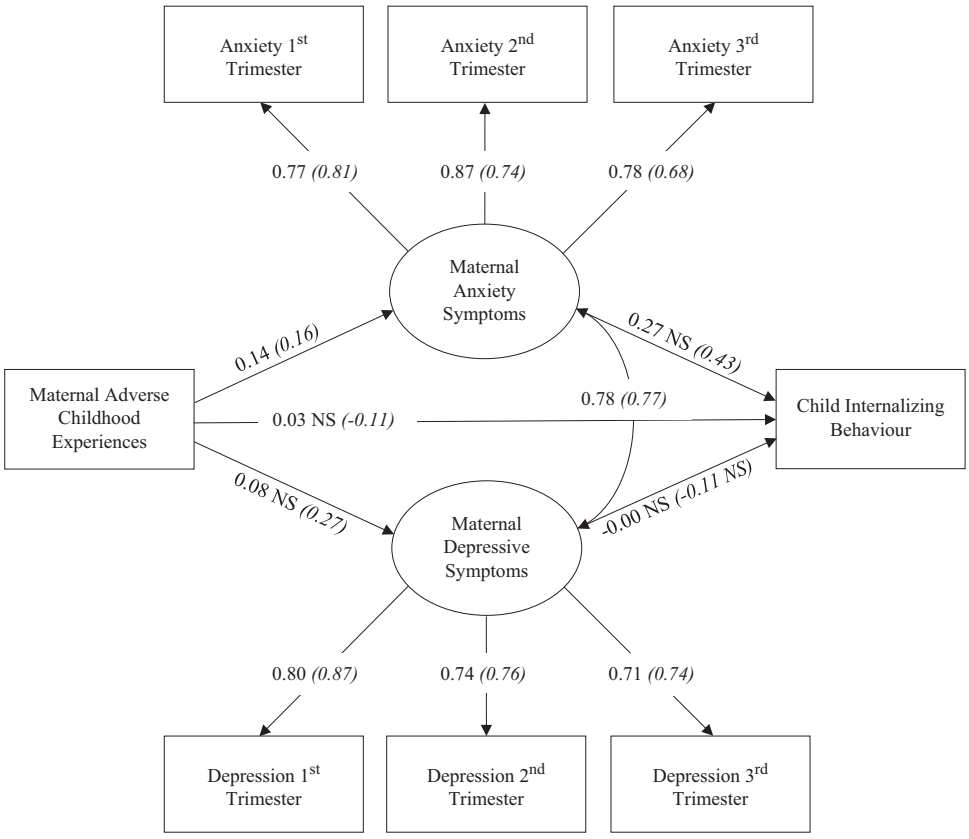

Fig. 2. Standardized parameter estimates for the moderated mediator model for child internalizing behaviour. Parameter estimates are for girls and boys (*in brackets*). Non-significant associations are designated as NS, otherwise all associations were significant at $P < 0.05$. The curved line with double-headed arrows represents the correlation between latent variables. For simplicity, covariates were not included in the figure but were included in the models: maternal education, ethnicity, and social support, household income, and infant age at behavioural assessment.

Postnatal model of internalizing behaviour

None of the moderated mediator models for internalizing behaviour was supported, despite the fact that when children were not grouped by sex there was a significant indirect effect for depression. The indirect effects of postnatal anxiety and depression for girls were, [−0.066–0.068] and [−0.006–0.100], and for boys [−0.011–0.136] and [−0.016–0.096], respectively. However, the combined indirect effect of anxiety and depression was significant for boys, 95% CI [0.025–0.128], but not for girls, 95% CI [−0.029–0.072]. Similar to other models, ACEs were associated with increased postnatal anxiety and depression, which subsequently were associated with increased internalizing behaviour in boys.

Discussion

This study builds on emerging research examining the intergenerational effects of ACEs and mechanisms related to behavioural psychopathology. The objectives of this study were to examine first, the impact of maternal ACEs on child behaviour at 2 years of age, second, maternal depression and anxiety as mediators, and third, the potential for moderation of associations by child sex. Direct associations were observed between total maternal ACE scores and children's externalizing behaviours. Overall, we observed a positive relationship between the number of maternal ACEs and both children's externalizing and internalizing behavioural problems via both maternal anxiety and depression. Even after controlling for relevant social and socio-demographic characteristics, mothers' exposure to ACEs increased their symptoms of depression and anxiety during the prenatal and postnatal periods, which consequently increased the expression of children's behavioural problems. Exposure timing effects and sex differences were observed.

ACEs predicted externalizing behaviour only in boys. The only unique mediator was postnatal depression, however, the combined effects of maternal anxiety and depression, both in the prenatal and postnatal periods, were significant mediators of ACEs on externalizing behaviour in boys. For internalizing behaviour, again the findings were primarily for boys. The only unique mediator was prenatal anxiety. The combination of prenatal anxiety and depression mediated the effects of ACEs on internalizing behaviour for both girls and boys and the combination of postnatal anxiety and depression also mediated the effects of ACEs on internalizing behaviour in boys. The findings also suggest a relatively greater impact of prenatal and postnatal anxiety and depression for boys compared to girls in both internalizing and externalizing behaviours. Effects for girls were observed only for internalizing behaviour and only when the effects of prenatal depression and anxiety were combined. Other research suggests that effects of early adversity on girls, particularly in internalizing behaviours such as anxiety, may be observable at later ages in boys.[80]

While our findings agree with other studies showing maternal ACEs increased the propensity for child behavioural

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press

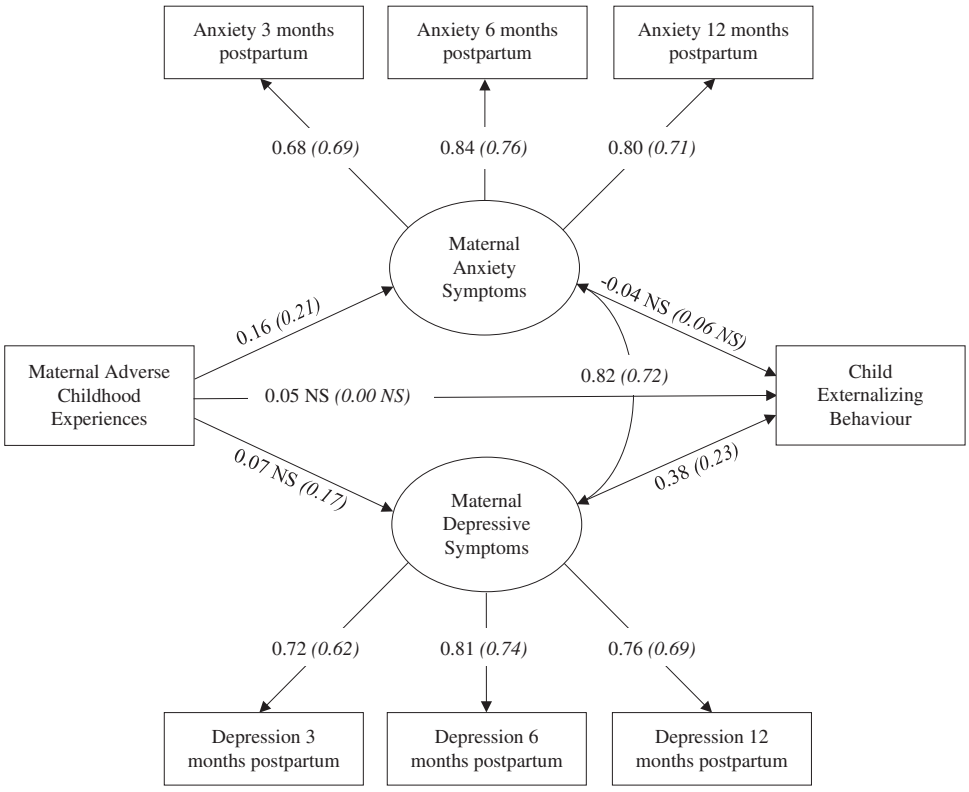

Fig. 3. Standardized parameter estimates for the moderated mediator model for child externalizing behaviour. Parameter estimates are for girls and boys (*in brackets*). Non-significant associations are designated as NS, otherwise all associations were significant at $P < 0.05$, with the exception of the pathway from maternal depressive symptoms to externalizing behaviour for boys for which $P = 0.07$. The curved line with double-headed arrows represents the correlation between latent variables. For simplicity, covariates were not included in the figure but were included in the models: maternal education, ethnicity, and social support, household income, and infant age at behavioural assessment.

problems,[11,14] our novel examination of sex differences revealed that maternal postnatal depression or anxiety did not mediate the relationships between maternal ACEs and internalizing problems among girls (although there was an effect for prenatal anxiety and depression). This is in contrast to other research showing that maternal postnatal anxiety and depression are associated with internalizing symptoms in girls.[81] Taken together, the findings suggest that maternal depression and anxiety may be important predictors of internalizing behaviour in girls but they do not appear to be involved in transmitting the effects of maternal ACEs to girls. Nevertheless, our finding that *prenatal* depression and anxiety together do mediate the effect of ACEs on internalizing in girls suggests that the timing of exposure may be an important determinant of outcome in girls. This did not appear to be the case for boys, where both prenatal and postnatal depression and anxiety were involved in transmitting the effects of ACEs to both internalizing and externalizing behaviour. In the context of exposure to ACEs, it appears that boys are vulnerable to both internalizing and externalizing psychopathology via prenatal and postnatal distress, while girls appear vulnerable in the prenatal period only, and only with respect to internalizing psychopathology. In general, these findings support the premise that boys are more vulnerable than girls to the intergenerational transmission of ACEs via maternal distress.[82]

The transmission of maternal ACEs to childhood vulnerability for developmental psychopathology may stem from sustained perturbations of stress regulatory systems that persist from early life into childhood and adulthood.[11] Childhood is a developmentally salient period,[83] during which cumulative exposure to adversities may participate in developmental programming of biological,[2] emotional[84] and social[85] responses to stress. For some women, such developmental programming persists into the childbearing years where its effects predispose women to develop depression or anxiety during the major life changes that occur as a consequence of childrearing. Depression and anxiety may operate together to affect the growing fetus via biological mechanisms[86] or the growing child via alterations in caregiver–child interaction,[42] with sex differences in vulnerability as proposed by the DOHaD hypothesis.[46] So, for both girls and boys, whose mothers were exposed to ACEs, prenatal exposures to the biological substrates of maternal distress connotes vulnerability to behavioural psychopathology. Postnatally, the observation that infant girls display greater responsiveness to social stimuli than boys (e.g. more eye contact and orientation to faces and voices),[87] suggests a potential protective mechanism for girls by improving the quality of caregiver–child relationship.

Our findings corroborate previous research which demonstrated that maternal depression during the prenatal and postnatal periods negatively impacts children's behavioural outcomes[31,88,89] and extends this understanding to include the effects of maternal anxiety. The findings also broaden our understanding of the intergenerational transmission of maternal

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press

ACEs, specifically via the effect of maternal depression and anxiety on child behaviour, paying special attention to sex differences associated with perinatal exposures. These findings are also in line with previous work which demonstrated increased externalizing behaviours among boys as a result of prenatal and postnatal maternal distress.[90]

Strengths and limitations

The prospective nature of our study design and sample size are significant strengths that allow us to evaluate the plausibility of causal pathways from maternal early life adversity to offspring behaviour and to control for relevant covariates while testing for sex differences. To our knowledge, the findings are the first to formally test indirect (mediator) effects of ACEs on children's behaviour via maternal depression and anxiety experienced in the prenatal or postpartum periods. Moreover, research to date utilized high-risk samples (e.g. mothers affected by concurrent or recent intimate partner violence),[14] assessed more limited conceptions of behaviour (e.g. socioemotional functioning),[8,11] used less rigorous measures of behaviour,[8,11] and did not examine the moderating influence of sex differences. Our study employed a community sample, rigorous, well-regarded measures of both internalizing and externalizing behaviours, and a well-designed examination of sex differences.

Nevertheless, several limitations constrain interpretation and generalizability of the findings. First, the sample represents a relatively low-risk sociodemographic population. Mothers were predominantly White, university educated, and had high income. The generalizability of these findings may therefore be limited to middle and upper class families. Second, the measurement of maternal ACEs is retrospective and prone to recall bias; however, evaluations of repeated administrations of the ACE Questionnaire reveal it to be reliable.[91,92] Third, our measures may have common source variance owing to the fact that mothers self-reported on their ACEs, self-reported on their depression and anxiety, and reported on their children's behaviour. Child behaviour assessment relied exclusively on maternal report, and others may have different perceptions of behaviour problems in the children. The APrON data set does not possess information on mental health history which would have been ideal to consider in modelling. Fourth, despite temporal ordering of the exposure, mediator and outcome variables, these data remain correlational and therefore cannot conclusively establish causality. Finally, despite the inclusion of relevant covariates, residual confounding may persist.

Conclusion

This study provides new evidence regarding the role of maternal mental health in the intergenerational transmission of maternal ACEs on internalizing and externalizing behaviour in 2-year-old boys and girls. The study shows that adverse experiences before the age of 18 are associated with increased symptoms of maternal depression and anxiety during both the prenatal and postnatal periods, and this increase in symptoms is associated with increased symptoms of behavioural problems in children, especially in boys. Overall, the current study suggests that children of mothers who experienced ACEs may be at heightened risk for developmental psychopathology. Mental health interventions for mothers with a history of ACEs represents an important future research objective because it has the potential to prevent or reduce the intergenerational cascade that perpetuates cycles of stress.

Supplementary material. To view supplementary material for this article, please visit https://doi.org/10.1017/S2040174418000648

Acknowledgements. The authors gratefully acknowledge the participants of the APrON study and the members of the APrON study team whose individual members are B.J. Kaplan, C.J. Field, D. Dewey, R.C. Bell, F.P. Bernier, M. Cantell, L.M. Casey, M. Eliasziw, A. Farmer, L. Gagnon, G.F. Giesbrecht, L. Goonewardene, D.W. Johnston, L. Kooistra, N. Letourneau, D.P. Manca, J.W. Martin, L.J. McCargar, M. O'Beirne, V.J. Pop and N. Singhal.

Financial support. This work was supported by Alberta Innovates – Health Solutions (AIHS), the Alberta Children's Hospital Foundation, and the Alberta Children's Hospital Chair in Parent-Infant Mental Health awarded to the first author.

Conflicts of interest. All of the authors declare that they have no conflicts of interest.

Ethical standards. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national guidelines on human experimentation, the Canadian Tri-Council Policy Statement on the Ethical Conduct for Research Involving Humans, and with the Helsinki Declaration of 1975, as revised in 2008, and has been approved by the institutional committees at the University of Calgary and the University of Alberta.

References

1. Felitti VJ, Anda RF, Nordenberg D, *et al*. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *Am J Prev Med*. 1998; 14, 245–258.
2. McLaughlin KA. Future directions in childhood adversity and youth psychopathology. *J Clin Child Adolesc Psychol*. 2016; 45, 361–382.
3. Danese A, Moffitt TE, Harrington H, *et al*. Adverse childhood experiences and adult risk factors for age-related disease: depression, inflammation, and clustering of metabolic risk markers. *Arch Pediatr Adolesc Med*. 2009; 163, 1135–1143.
4. McDonald S, Tough S. Alberta Adverse Childhood Experiences Survey 2013 Report, 2014. Alberta Centre for Child, Family and Community Services, Calgary, AB.
5. Anda RF, Felitti VJ, Bremner JD, *et al*. The enduring effects of abuse and related adverse experiences in childhood. *Eur Arch Psychiatry Clin Neurosci*. 2006; 256, 174–186.
6. Schilling EA, Aseltine RH, Gore S. Adverse childhood experiences and mental health in young adults: a longitudinal survey. *BMC Public Health*. 2007; 7, 30.
7. Leeners B, Rath W, Block E, Görres G, Tschudin S. Risk factors for unfavorable pregnancy outcome in women with adverse childhood experiences. *J Perinat Med*. 2014; 42, 171–178.
8. McDonnell CG, Valentino K. Intergenerational effects of childhood trauma: evaluating pathways among maternal ACEs, perinatal depressive symptoms, and infant outcomes. *Child Maltreat*. 2016; 21, 317–326.
9. Wosu AC, Gelaye B, Williams MA. History of childhood sexual abuse and risk of prenatal and postpartum depression or depressive symptoms: an epidemiologic review. *Arch Womens Ment Health*. 2015; 18, 659–671.
10. Biaggi A, Conroy S, Pawlby S, Pariante CM. Identifying the women at risk of antenatal anxiety and depression: a systematic review. *J Affect Disord*. 2016; 191, 62–77.
11. Madigan S, Wade M, Plamondon A, Maguire JL, Jenkins JM. Maternal adverse childhood experience and infant health: biomedical and psychosocial risks as intermediary mechanisms. *J Pediatr*. 2017; 1, 1–9.
12. Thomas JC, Letourneau N, Bryce CI, Campbell TS, Giesbrecht GF. Biological embedding of perinatal social relationships in infant stress reactivity. *Dev Psychobiol*. 2017; 59, 425–435.
13. Letourneau N, Dennis C, Linder J, Cosic N. The effect of perinatal depression treatment for mothers on parenting and child development: a systematic review. *Depress Anxiety*. 2017; 34, 928–966.

14. Fredland N, McFarlane J, Symes L, Maddoux J. Exploring the association of maternal adverse childhood experiences with maternal health and child behavior following intimate partner violence. *J Womens Health*. 2017; 27, 65–71.
15. Atladottir HO, Gyllenberg D, Langridge A, *et al*. The increasing prevalence of reported diagnoses of childhood psychiatric disorders: a descriptive multinational comparison. *Eur Child Adolesc Psychiatry*. 2014; 24, 173–183.
16. Halfon N, Houtrow A, Larson K, Newacheck PW. The changing landscape of disability in childhood. *Future Child*. 2012; 22, 13–42.
17. Renner LM, Slack KS. Intimate partner violence and child maltreatment: understanding intra-and intergenerational connections. *Child Abuse Negl*. 2006; 30, 599–617.
18. McFarlane J, Symes L, Binder BK, Maddoux J, Paulson R. Maternal-child dyads of functioning: the intergenerational impact of violence against women on children. *Matern Child Health J*. 2014; 18, 2236–2243.
19. Stith SM, Rosen KH, Middleton KA, *et al*. The intergenerational transmission of spouse abuse: A meta-analysis. *J Marriage Fam*. 2000; 62, 640–654.
20. Kingston D, Tough S, Whitfield H. Prenatal and postpartum maternal psychological distress and infant development: a systematic review. *Child Psychiatry Hum Dev*. 2012; 43, 683–714.
21. Kingston D, Tough S. Prenatal and postnatal maternal mental health and school-age child development: a systematic review. *Matern Child Health J*. 2014; 18, 1728–1741.
22. Falah-Hassani K, Shiri R, Dennis C-L. The prevalence of antenatal and postnatal co-morbid anxiety and depression: a meta-analysis. *Psychol Med*. 2017; 47, 2041–2053.
23. Fricchione G. Generalized anxiety disorder. *N Engl J Med*. 2004; 351, 675–682.
24. Campbell SB, Cohn JF, Murray L, Cooper PJ. The timing and chronicity of postpartum depression: implications for infant development. In *Postpartum Depression and Child Development* (eds. Murray L, Cooper P), 1997, pp. 165–197. Guilford Press: New York, NY.
25. Lee AM, Lam SK, SMSM Lau, *et al*. Prevalence, course, and risk factors for antenatal anxiety and depression. *Obstet Gynecol*. 2007; 110, 1102–1112.
26. Muñoz RF, Le H-N, Ippen CG, *et al*. Prevention of postpartum depression in low-income women: development of the Mamás y Bebés/Mothers and Babies Course. *Cogn Behav Pract*. 2007; 14, 70–83.
27. Falah-Hassani K, Shiri R, Dennis C-L. The prevalence of antenatal and postnatal co-morbid anxiety and depression: a meta-analysis. *Psychol Med*. 2017; 47, 2041–2053.
28. Field T. Postpartum anxiety prevalence, predictors and effects on child development: a review. *J Psychiatry Psychiatr Disord*. 2017; 1, 86–102.
29. O'connor TG, Heron J, Golding J, Beveridge M, Glover V. Maternal antenatal anxiety and children's behavioural/emotional problems at 4 years. *Br J Psychiatry*. 2002; 180, 502–508.
30. Van den Bergh B, al. e. Antenatal maternal anxiety and stress and the neurobehavioural development of the fetus and child: links and possible mechanisms. A review. Neurosci Biobehav Rev. 2005; 29, 237–258.
31. Field T, Diego M, Hernandez-Reif M. Prenatal depression effects on the fetus and newborn: a review. *Infant Behav Dev*. 2006; 29, 445–455.
32. Field T. Prenatal depression effects on early development: a review. *Infant Behav Dev*. 2011; 34, 1–14.
33. Giallo R, Woolhouse H, Gartland D, Hiscock H, Brown S. The emotional–behavioural functioning of children exposed to maternal depressive symptoms across pregnancy and early childhood: a prospective Australian pregnancy cohort study. *Eur Child Adolesc Psychiatry*. 2015; 24, 1233–1244.
34. Grace SL, Evindar A, Stewart D. The effect of postpartum depression on child cognitive development and behavior: a review and critical analysis of the literature. *Arch Womens Ment Health*. 2003; 6, 263–274.
35. Giesbrecht G, Letourneau N, Campbell T. Sexually dimorphic and interactive effects of prenatal maternal cortisol and psychological distress on infant cortisol reactivity. *Dev Psychopathol*. 2017; 29, 805–818.
36. Waters CS, Hay DF, Simmonds JR, van Goozen SH. Antenatal depression and children's developmental outcomes: potential mechanisms and treatment options. *Eur Child Adolesc Psychiatry*. 2014; 23, 957–971.
37. Thomas JC, Magel C, Tomfohr-Madsen L, *et al*. Adverse childhood experiences and HPA axis function in pregnant women. *Horm Behav*. 2018; 102, 10–22.
38. Moehler E, Brunner R, Wiebel A, Reck C, Resch F. Maternal depressive symptoms in the postnatal period are associated with long-term impairment of mother–child bonding. *Arch Womens Ment Health*. 2006; 9, 273–278.
39. Field T. Postpartum depression effects on early interactions, parenting, and safety practices: a review. *Infant Behav Dev*. 2010; 33, 1–6.
40. Schrock M, Woodruff-Borden J. Parent-child interactions in anxious families. *Child Fam Behav Ther*. 2010; 32, 291–310.
41. Letourneau N, Tramonte L, Willms JD. Maternal depression, family functioning and children's longitudinal development. *J Pediatr Nurs*. 2012; 28, 223–234.
42. Letourneau N, Dennis C, Benzies K, *et al*. Postpartum depression is a family affair: addressing the impact on mothers, fathers, and children. *Issues Ment Health Nurs*. 2012; 33, 445–457.
43. Field T. Postpartum depression effects on early interactions, parenting and safety practices: a review. *Infant Behav Dev*. 2011; 33, 1–6.
44. Pemberton CK, Neiderhiser JM, Leve LD, *et al*. Influence of parental depressive symptoms on adopted toddler behaviors: an emerging developmental cascade of genetic and environmental effects. *Dev Psychopathol*. 2010; 22, 803–818.
45. Chaplin T, Aldao A. Gender differences in emotion expression in children: a meta-analytic review. *Psychol Bull*. 2013; 139, 735–765.
46. Wadhwa PD, Buss C, Entringer S, Swanson JM. Developmental Origins of Health and Disease: brief history of the approach and current focus on epigenetic mechanisms. *Seminars in Reproductive Medicine*, 2009; 27, 358.
47. Arnett AB, Pennington BF, Willcutt EG, DeFries JC, Olson RK. Sex differences in ADHD symptom severity. *J Child Psychol Psychiatry* 2014; 56, 632–639.
48. Glover V, Hill J. Sex differences in the programming effects of prenatal stress on psychopathology and stress responses: an evolutionary perspective. *Physiol Behav*. 2012; 106, 736–740.
49. Trapolini T, McMahon CA, Ungerer JA. The effect of maternal depression and marital adjustment on young children's internalizing and externalizing behaviour problems. *Child Care Health Dev*. 2007; 33, 794–803.
50. Chaplin TM, Aldao A. Gender differences in emotion expression in children: a meta-analytic review. *Psychol Bull*. 2013; 139, 735–765.
51. Leman PJ, Tenenbaum HR. Practising gender: children's relationships and the development of gendered behaviour and beliefs. *Br J Dev Psychol*. 2011; 29, 153–157.
52. Carter AS, Garrity-Rokous FE, Chazan-Cohen R, Little C, Briggs-Gowan MJ. Maternal depression and comorbidity: predicting early parenting, attachment security, and toddler social-emotional problems and competencies. *J Am Acad Child Adolesc Psychiatry*. 2001; 40, 18–26.
53. Goodman S, Rouse M, Copnnell A, *et al*. Maternal depression and child psychopathology: a meta-analytic review. *Clin Child Fam Psychol Rev*. 2011; 14, 1–27.
54. Goodman SH, Gotlib IH. *Children of Depressed Parents: Mechanisms of Risk and Implications for Treatment*. 2002. American Psychological Association: Washington, DC.
55. Fleming A, Klein E, Corter C. The effects of a social support group on depression, maternal attitudes and behavior in new mothers. *J Child Psychol Psychiatry*. 1992; 33, 685–698.
56. Coburn SS, Gonzales N, Luecken L, Crnic K. Multiple domains of stress predict postpartum depressive symptoms in low-income Mexican American women: the moderating effect of social support. *Arch Womens Ment Health*. 2016; 19, 1009–1018.
57. Meritesacker B, Bade U, Haverkock A, Pauli-Pott U. Predicting maternal reactivity/sensitivity: the role of infant emotionality, maternal depressiveness/anxiety, and social support. [References] *Infant Ment Health J*. 2004; 25, 47–61.
58. Herwig J, Wirtz M, Bengal J. Depression, partnership, social support, and parenting: interaction of maternal factors with behavioral problems of the child. *J Affect Disord*. 2004; 80, 199–208.
59. Cutrona C, Troutman B. Social support, infant temperament, and parenting self-efficacy: a mediational model of postpartum depression. *Child Dev*. 1986; 57, 1507–1518.

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press

60. Norbeck JS, Anderson NJ. Life stress, social support, and anxiety in mid- and late-pregnancy among low income women. *Res Nurs Health*. 1989; 12, 281–287.
61. Falah-Hassani K, Shiri R, Dennis C-L. Prevalence and risk factors for comorbid postpartum depressive symptomatology and anxiety. *J Affect Disord*. 2016; 198, 142–147.
62. Kaplan BJ, Giesbrecht GF, Leung BM, *et al*. The Alberta Pregnancy Outcomes and Nutrition (APrON) cohort study: rationale and methods. *Matern Child Nutr*. 2014; 10, 44–60.
63. Murphy A, Steele M, Dube S, *et al*. Adverse Childhood Experiences (ACES) questionnaire: and Adult Attachment Interview (AAI): implications for parent child relationships. *Child Abuse Negl*. 2014; 38, 224–233.
64. Anda RF, Felitti VJ, Bremner JD, *et al*. The enduring effects of abuse and related adverse experiences in childhood. *Eur Arch Psychiatry Clin Neurosci*. 2006; 256, 174–186.
65. Cox JL, Holden J, Sagovsky R. Detection of postnatal depression. Development of the 10-item Edinburgh Postnatal Depression Scale. *Br J Psychiatry*. 1987; 150, 782–786.
66. Brouwers EP, van Baar AL, Pop VJ. Does the Edinburgh Postnatal Depression Scale measure anxiety? *J Psychosom Res*. 2001; 51, 659–663.
67. Huizink AC, Mulder EJ, de Medina PGR, Visser GH, Buitelaar JK. Is pregnancy anxiety a distinctive syndrome? *Early Hum Dev*. 2004; 79, 81–91.
68. Derogatis L. *Symptom Checklist-90-R: Administration, Scoring, and Procedure Manual*. 1994. NCS Pearson Inc.: Minneapolis, MN.
69. Dinning WD, Evans RG. Discriminant and convergent validity of the SCL-90 in psychiatric inpatients. *J Pers Assess*. 1977; 41, 304–310.
70. Vallejo MA, Jordán CM, Díaz MI, Comeche MI, Ortega J. Psychological assessment via the internet: a reliability and validity study of online (vs paper-and-pencil) versions of the General Health Questionnaire-28 (GHQ-28) and the Symptoms Check-List-90-Revised (SCL-90-R). *J Med Internet Res*. 2007; 9, 1–22.
71. Achenbach TM, Rescorla LA. *Manual for the ASEBA Preschool Forms and Profiles*. 2010. University of Vermont: Burlington, VT.
72. Kristensen S, Henriksen TB, Bilenberg N. The Child Behavior Checklist for Ages 1.5–5 (CBCL/1½–5): assessment and analysis of parent-and caregiver-reported problems in a population-based sample of Danish preschool children. *Nord J Psychiatry*. 2010; 64, 203–209.
73. Michalos AC. Essays on the Quality of Life (vol. 19), 2003. Springer Science & Business Media: New York City, NY.
74. Mplus Version 7.3 [computer program], 1988–2015. Los Angeles, CA: Muthén & Muthén.
75. Rabe-Hesketh S, Skrondal A. *Generalized Latent Variable Modeling: Multilevel, Longitudinal, and Structural Equation Models*. 2004. Chapman and Hall/CRC: London, UK.
76. Hayes AF. *Introduction to Mediation, Moderation, and Conditional Process Analysis: A Regression-Based Approach*. 2013. Guilford Press: New York.
77. Bentler PM. Comparative fit indexes in structural models. *Psychol Bull*. 1990; 107, 238.
78. Lt Hu, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Struct Equ Modeling*. 1999; 6, 1–55.
79. Bearden WO, Sharma S, Teel JE. Sample size effects on chi square and other statistics used in evaluating causal models. *J Mark Res*. 1982; 19, 425–430.
80. Roza SJ, Hofstra MB, van der Ende J, Verhulst FC. Stable prediction of mood and anxiety disorders based on behavioral and emotional problems in childhood: a 14-year follow-up during childhood, adolescence, and young adulthood. *Am J Psychiatry*. 2003; 160, 2116–2121.
81. Essex MJ, Klein MH, Cho E, Kraemer HC. Exposure to maternal depression and marital conflict: gender differences in children's later mental health symptoms. *J Am Acad Child Adolesc Psychiatry*. 2003; 42, 728–737.
82. Schore AN. All our sons: the developmental neurobiology and neuroendocrinology of boys at risk. *Infant Ment Health J*. 2017; 38, 15–52.
83. Holzman C, Eyster J, Tiedje LB, *et al*. A life course perspective on depressive symptoms in mid-pregnancy. *Matern Child Health J*. 2006; 10, 127–183.
84. Cloitre M, Stovall-McClough C, Zorbas P, Charuvastra A. Attachment organization, emotion regulation, and expectations of support in a clinical sample of women with childhood abuse histories. *J Trauma Stress*. 2008; 21, 282–289.
85. Al Odhayani A, Watson WJ, Watson L. Behavioural consequences of child abuse. *Can Fam Physician*. 2013; 59, 831–836.
86. Giesbrecht G, Poole J, Letourneau N, Campbell T, Kaplan B. The buffering effect of social support on HPA axis function during pregnancy. *Psychosom Med*, 2013; 75, 856–862.
87. Alexander GM, Wilcox T. Sex differences in early infancy. *Child Dev Perspect*. 2012; 6, 400–406.
88. Avan B, Richter LM, Ramchandani PG, Norris SA, Stein A. Maternal postnatal depression and children's growth and behaviour during the early years of life: exploring the interaction between physical and mental health. *Arch Dis Child*. 2010; 95, 690–695.
89. Choe DE, Olson SL, Sameroff AJ. Effects of early maternal distress and parenting on the development of children's self-regulation and externalizing behavior. *Dev Psychopathol*. 2013; 25, 437–453.
90. Arnett AB, Pennington BF, Willcutt EG, DeFries JC, Olson RK. Sex differences in ADHD symptom severity. *J Child Psychol Psychiatry*. 2015; 56, 632–639.
91. Dube SR, Williamson DF, Thompson T, Felitti VJ, Anda RF. Assessing the reliability of retrospective reports of adverse childhood experiences among adult HMO members attending a primary care clinic. *Child Abuse Negl*. 2004; 28, 729–737.
92. Hardt J, Rutter M. Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. *J Child Psychol Psychiatry*. 2004; 45, 260–273.

https://doi.org/10.1017/S2040174418000648 Published online by Cambridge University Press