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ORIGINAL CONTRIBUTION

Maternal postnatal mental health and offspring symptoms of ADHD at 8–9 years: pathways via parenting behavior

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Abstract

Exposure to maternal mental health problems during pregnancy and the first year of life has been associated with the development of ADHD. One pathway through which maternal mental health may influence children's outcomes is via its effects on parenting. This study aimed to investigate the mediating role of parenting behavior in the pathway between maternal postnatal distress and later symptoms of ADHD in the child. Biological mothers living with their children participating in the Longitudinal Study of Australian Children with data available from waves 1 (child age 3–12 months) and 5 (child age 8–9 years) were included in the current study ($n = 3456$). Postnatal distress was assessed by parent report at wave 1. Parenting warmth, hostility and consistency were assessed by parent report at wave 5. ADHD status at wave 5 was ascertained by parent report of the child having a diagnosis of ADHD/ADD or by elevated ADHD symptoms by both parent and teacher report. There was evidence of an indirect pathway from maternal postnatal distress to child ADHD at age 8–9 years via parenting hostility, but not through parenting warmth or consistency, even after accounting for concurrent maternal mental health. Our findings highlight the importance of early identification and intervention for maternal postnatal distress, as treatment may prevent mothers from developing hostile parenting practices and also disrupt the pathway to ADHD in their offspring.

Keywords Attention-deficit/hyperactivity disorder · Parenting · Postnatal distress · Sex

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is the most common neurodevelopmental disorder of childhood, affecting approximately 5% of children worldwide [1]. ADHD is characterized by developmentally inappropriate levels of inattention, hyperactivity and impulsivity that cause significant impairments in daily functioning [2]. Although ADHD is highly heritable (> 70%) [3], a number of environmental factors including prematurity [4] and maternal smoking during pregnancy [5] have been implicated in the etiology of ADHD. Exposure to maternal mental health problems during pregnancy and the first year of life have been associated with the development of ADHD [6]. One pathway through which maternal mental health may influence children's outcomes is via its effects on parenting. This pathway has been established for broad psychosocial adjustment [7, 8] but has yet to be examined for ADHD specifically.

Associations have been identified between maternal mental health during pregnancy (e.g., stress [9–11], anxiety [12, 13], antidepressant use [14, 15]) and ADHD symptoms in offspring. However, birth cohort studies have also demonstrated increased ADHD symptoms in children whose mothers experienced depression/anxiety in the postnatal period, independent of symptoms experienced during pregnancy [16–18]. For example, in an Australian birth cohort ($n = 3982$), Clavarino et al. [18] found that children whose mothers were anxious after pregnancy, but not during pregnancy, were five times more likely to experience persistent hyperactivity from 5 to 14 years of age than those whose mothers did not experience anxiety. Similarly in the National

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Longitudinal Survey of Children and Youth (NLSCY), a nationally representative Canadian sample ($n = 2946$), maternal postnatal depression was associated with twice the odds of experiencing a high persistent trajectory of hyperactivity symptoms [17].

Goodman and Gotlib [19] proposed three pathways through which postnatal depression may influence child psychosocial adjustment: shared genetic risk, impaired parenting, and stressful context (e.g., poverty). Of these three pathways, parenting is the most amenable to intervention therefore we decided to investigate this pathway, while taking into account some of the potential shared genetic risk by adjusting for maternal mental health difficulties during pregnancy (anxiety and depression, and antidepressant use) and stressful contexts (socio-economic position). Maternal mental health can influence the way in which mothers parent their children. For example, highly anxious mothers tend to be more overprotective and controlling [20], and mothers experiencing depression tend to be less consistent and may be withdrawn or overly intrusive [21].

Numerous studies have indicated that the parenting styles employed by parents of children with ADHD differ from parenting styles used by parents of children who do not have ADHD [22–25]. The key parenting behaviors of interest in the current study are parenting hostility (parenting behaviors that are over-controlling or negative and rigid enforcement of rules/expectations [26]), warmth (affectionate, engaged and responsive parenting behaviors [27]), and consistency (the consistent application of consequences for child behavior [26]). Associations between negative aspects of parenting (e.g., hostility) and ADHD have consistently been reported in both clinical and community studies and in both children and adolescents [28]. In contrast, findings for parental warmth have been mixed. In a clinical sample of adolescents with ADHD ($n = 337$), mothers reported less parental warmth and greater overprotection and control than mothers of non-ADHD controls ($n = 223$) [22]. However, in a community sample of parents (94% mothers) of 6–8-year-old children with ($n = 179$) and without ($n = 212$) ADHD, there were no differences in parental warmth, although parents of children with ADHD were more hostile and less consistent than parents of non-ADHD controls [23]. Bhide et al. [23] also examined whether the child's gender influenced the parenting styles employed by parents of children with ADHD. Parents of boys and girls with ADHD were more hostile, however, only parents of girls with ADHD were less consistent than parents of non-ADHD control girls.

There has been some research examining whether parenting behavior mediates the relationship between maternal mental health and child psychiatric symptoms. In the NLSCY cohort ($n = 6048$), Elgar et al. [7] found that the relationship between maternal depressive symptoms and child externalizing problems in adolescence (mean age 12.6 years, SD = 1.7 years) was mediated through low parental warmth and parental rejection. Similarly, in a nationally representative sample of Australian children ($n = 2200$) aged 6–7 years, Giallo et al. [8] found that parental hostility mediated the relationship between maternal postnatal distress and emotional/behavioral difficulties. Both of these studies used broad measures of child psychosocial adjustment. Thus, it is not clear if the same relationships would exist between maternal postnatal mental health, parenting and ADHD.

Aims

Using data from a nationally representative sample of Australian children and their families, this study aimed to examine the longitudinal relationships between maternal postnatal distress, parenting behavior at 8–9 years and ADHD (both parent report of diagnosis and clinically significant symptoms) at 8–9 years. The aims of the study were twofold.

1. To investigate the mediating role of parenting behavior (i.e., warmth, hostility, and consistency) in the pathway between maternal postnatal distress and a later parent-reported diagnosis of ADHD, controlling for concurrent maternal distress. We hypothesized that postnatal distress would be associated with lower parenting warmth and consistency, and higher parenting hostility 8–9 years later, and that these behaviors in turn would be associated with a parent-reported diagnosis and clinically significant symptoms of ADHD in the child.
2. To determine whether there were model differences by the child's sex, socio-economic position (SEP), mental health problems during pregnancy, or antidepressant use during pregnancy. We predicted that the strength of the relationships between maternal distress, parenting and ADHD would be stronger for boys than girls, stronger in families from a low socio-economic background, and stronger for mothers experiencing mental health difficulties or using antidepressant medication during pregnancy.

Method

Study design and sample

Data were drawn from waves 1 and 5 of the Longitudinal Study of Australian Children (LSAC) infant cohort. The study was approved by the Australian Institute of Family Studies (AIFS) Ethics Committee. Detailed information about the study design is provided elsewhere [29]. Briefly, a two-stage sample design was used. First, approximately 10% of all postcodes were selected, then a number of children

proportional to population size was randomly selected from each postcode using the Medicare database. At wave 1, 5107 infants aged 3–12 months and their families were recruited into the study (64% response rate). Children from non-English speaking families and those living in rental properties were under-represented, whilst children with more highly educated parents were over-represented. Follow-up occurred every 2 years when the children were aged 2–3 years (wave 2), 4–5 years (wave 3), 6–7 years (wave 4) and 8–9 years (wave 5). At wave 5, the total number of participants was 4085, representing a retention rate of 80%.

The inclusion criteria for the present study were biological mothers who were living with their children and had data available at waves 1 and 5.

Measures

Psychological distress was assessed at waves 1 (first year postpartum) and 5 (when the child was 8–9 years) using the Kessler-6 (K6) [30]. Mothers reported on the extent to which they experienced symptoms of distress (e.g., nervous, sad) in the last 4 weeks. The six items were rated on a scale ranging from 0—none of the time to 4—all or most of the time, and summed to derive a total score. The K6 has been used in population-based studies due to its brevity, strong psychometric properties, and ability to screen for mood and anxiety disorders against Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV) criteria [31]. Cronbach's α for the current sample at waves 1 and 5 were 0.82 and 0.85, respectively.

ADHD status (yes/no)

At wave 5 the primary caregiver (in most cases, the child's mother) was asked "Does the study child have attention-deficit disorder or attention-deficit/hyperactivity disorder?" If the parent responded "yes", the child was classified as having a parent-reported diagnosis of ADHD. We have used this case definition in a previous study using LSAC data [6] and found that children with a parent-reported diagnosis of ADHD were very similar to those with clinically elevated ADHD symptoms on the Strengths and Difficulties Questionnaire (SDQ).

Clinically significant inattention and hyperactivity symptoms (yes/no)

In addition to parent report of ADHD diagnosis, we used mother and teacher report on the SDQ hyperactivity/inattention subscale [32] to create an alternative ADHD case definition. The SDQ is a well-validated measure of behavioral and emotional problems for children aged 4–16 years. Five items assessing hyperactivity/inattention were rated on a 3-point scale ranging from 0—not true to 2—certainly true and summed, with higher scores indicating more difficulties. Cronbach's α for the current sample was 0.81 for mother report and 0.89 for teacher report. Children who score in the clinical range (≥ 6) on this subscale have been found to be more likely to meet criteria for ADHD using DSM-IV [33] criteria, with an odds ratio of 17.9 [34]. Scores of ≥ 6 on both mother and teacher-reported SDQ hyperactivity/inattention subscale was considered clinically significant.

Parenting warmth was assessed at wave 5 using five items from the Child Rearing Questionnaire [35]. Mothers indicated how often they expressed warmth and affection toward their child (e.g., How often do you express affection by hugging, kissing and holding this child?) on a 5-point scale ranging from 1—never/almost never to 5—always/almost always. Scores were summed, with higher scores indicating greater warmth ($\alpha = 0.90$).

Hostile parenting was measured at wave 5 using four items from the NLSCY [36]. Mothers rated how often they experienced anger and irritability during interactions with their child (e.g., "How often are you angry when you punish this child?") on a 5-point scale ranging from 1—never/almost never to 5—all the time. Items were summed with higher scores indicating greater anger and irritability during interactions with their child ($\alpha = 0.72$).

Parenting consistency was assessed at wave 5 using six items from the NLSCY [36]. Mothers rated how often they apply consistent age-appropriate rules, expectations and consequences to their child (e.g., "When you give this child an instruction or make a request to do something, how often do you make sure that he/she does it?") on a 5-point scale ranging from 1—never/almost never to 5—all the time. Items were summed with higher scores indicating greater consistency in applying rules and consequences during interactions with their child ($\alpha = 0.70$).

Socio-economic position (SEP) was assessed at wave 1 and is a composite variable derived by ranking each family's relative SEP based on combined household parental income, education and occupational prestige [37]. Families with a standardized score $\leq$ 25th percentile were classified as 'low', whilst those $>$ 75th percentile were 'high' SEP, and the remainder 'medium' SEP.

Anxiety/stress/depression and use of antidepressant medication during pregnancy

Mothers were asked to indicate (yes or no) if they had problems with stress, anxiety or depression during their pregnancy with the study child at wave 1. They were also asked to indicate if they had taken antidepressant medication at any point during the pregnancy.

Data analysis

Path analysis using Mplus Version 7.11 [38] was conducted to test the hypothesized model using parent-reported ADHD diagnosis as the outcome and was repeated using clinically significant ADHD symptoms as the outcome (see Fig. 1). Weighted least squares estimation (WLSMV) was used to test all models, and assessed using the Chi square test (χ^2), and other practical fit indices including the Tucker-Lewis Index (TLI), the Comparative Fit Index (CFI), and Root Mean Square Error of Approximation (RMSEA). Indices for the TLI and CFI should exceed 0.90 for an acceptable fit [39], and values close to or below 0.05 for the RMSEA were considered acceptable [40]. The direct, indirect, and total effects using the product of coefficients approach [41] were estimated in Mplus, along with the bootstrap option using 1000 draws to obtain bootstrap confidence intervals for the indirect effects. The standardized estimates are reported. Evidence of mediation (i.e., an intervening variable transmits the effect of an independent variable to a dependent variable) is provided when the confidence interval for the estimate for the indirect effect does not cross zero and is significant (p value is provided in Mplus).

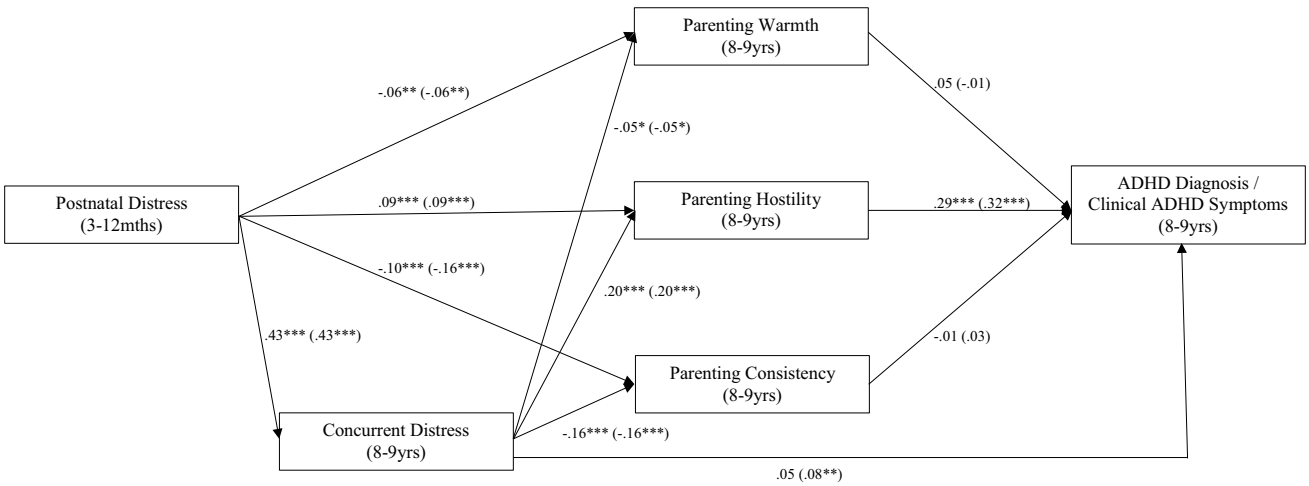
Next, multi-group analyses were conducted to test whether the models differed significantly by the child's sex, SEP, maternal mental health problems, or maternal anti-depressant use during pregnancy. For each analysis, a model with all parameters freely estimated between groups (unconstrained model) was statistically compared to a model with all parameters forced to be equal between the groups (constrained model). The Chi square difference test for the WLSMV estimation method was conducted using the DIFFTEST option available in Mplus Version 7.11 [38]. A significant Chi square difference test indicates that there are significant differences in one or more model pathways between the groups. To identify which pathways were significantly different, a series of contrasts in order of smallest to largest difference in path estimates were conducted, and the Chi square difference results were assessed. A Bonferroni adjusted alpha level was used given that multiple contrasts were conducted.

Results

Sample characteristics

Of the 5107 families recruited into the study, 59 (1.1%) mothers did not meet the inclusion criteria (biological mothers living with their children), and 764 (15.0%) were lost to attrition. A further 29 (0.6%) cases had missing data on parent-reported ADHD diagnosis, and 799 (15.7%) cases had more than 30% missing data across the variables of interest and so were excluded. The final sample for analysis consisted of 3456 mothers and their children (see Figure S1). Mothers excluded from the analyses were more likely to be born outside Australia, be from non-English speaking and Aboriginal or Torres Strait Islander backgrounds, live in a single parent household, have lower educational attainment and be of lower SEP compared to mothers in the final sample (all $p < 0.001$).

The demographic characteristics for the final sample at wave 1 are presented in Table 1. Majority of mothers were born in Australia, had completed high school, were in part-time employment, and headed a two-parent family. Approximately 3% of women ($n = 101$) reported that their



Note: *$p < .05$, ** $p < .01$, ***$p < .001$; Model for clinical ADHD symptoms in brackets; Residual variances between parenting behaviors are correlated

Fig. 1 Standardized parameter estimates for the hypothesized models for ADHD diagnosis and clinical ADHD symptoms

Table 1 Demographic characteristics for the final sample of mothers and children with and without a parent-reported diagnosis of ADHD

	Child without ADHD diagnosis ($n=3355$)	Child with ADHD diagnosis ($n=101$)	Total ($N=3456$)
Maternal characteristics			
Age, mean (SD)	31.7 (5.0)	30.9 (5.9)	31.6 (5.0)
Country of birth—Australia, *n* (%)	2705 (80.6)	86 (85.1)	2791 (80.8)
Language other than English, *n* (%)	376 (11.2)	7 (6.9)	383 (11.1)
Aboriginal or Torres Strait Islander, *n* (%)	52 (1.5)	3 (3.0)	55 (1.6)
Education level—year 12 and above, *n* (%)	2469 (73.6)	60 (59.4)	2529 (73.2)
Employment status, *n* (%)			
Work part-time (1–34 h/week)	2721 (81.1)	74 (73.3)	2795 (80.9)
Work full-time (35 + h/week)	643 (18.9)	27 (26.7)	661 (19.1)
Mental health problems during pregnancy, *n* (%)	547 (16.5)	35 (34.7)	582 (17.1)
Infant and family characteristics			
Study child's age—wave 1 (months), *M* (*SD*)	8.8 (2.6)	8.6 (2.7)	8.7 (2.6)
Study child's age—wave 5 (years), *M* (SD)	8.4 (0.5)	8.4 (0.5)	8.4 (0.5)
Study child's gender—male, *n* (%)	1746 (52.0)	80 (79.2)	1826 (52.8)
Number of children in household, *M* (SD)	1.9 (1.0)	2.0 (1.3)	1.9 (1.0)
Family structure—two-parent family, *n* (%)	3161 (94.1)	89 (88.1)	3250 (94.0)
Socio-economic position, *n* (%)			
Low	600 (17.9)	28 (27.7)	628 (18.2)
Middle	1770 (52.8)	49 (48.5)	1819 (52.6)
High	985 (29.4)	24 (23.8)	1009 (29.2)

child had a diagnosis of ADHD. The proportion of children in the clinical range on the parent- and teacher-reported SDQ hyperactivity/inattention subscale was approximately 19% ($n=661$) and 18% ($n=526$), respectively. The proportion of children with clinical ADHD symptoms by both parent and teacher report, the measure used in our analyses, was 7.8% ($n=269$). Significantly more boys than girls had a parent-reported diagnosis of ADHD or clinical ADHD symptoms ($p<0.001$).

Compared to women without a child with ADHD, significantly more women with children with ADHD reported mental health problems during pregnancy ($p<0.001$), were from a low SEP ($p=0.037$), did not complete high school ($p=0.003$), and headed a single parent household ($p=0.018$).

Descriptive statistics

After excluding cases who were not biological mothers living with their children at waves 1 and 5, cases with missing data on ADHD status, and cases with more than 30% of data missing across variables of interest, there was less than 1.5% missing data across all variables, and these were imputed using the Full Information Maximum Likelihood (FIML) option in Mplus [38]. Descriptive statistics are presented in Table 2. Correlations among the study variables were modest (for hostility) or low (all other variables) and statistically significant except for the relationships between parenting warmth and parent-reported ADHD diagnosis.

Relationships between maternal postnatal distress, parenting and parent-reported ADHD diagnosis and clinical ADHD symptoms

The hypothesized models for parent-reported ADHD diagnosis and clinical ADHD symptoms were an excellent fit to the data, χ^2 (1, $N=3456$) = 1.39, $p=0.239$, CFI = 1.00, TLI = 1.00, RMSEA = 0.01 (90% CI 0.00–0.05) and χ^2 (1, $N=3456$) = 1.8, $p=0.277$, CFI = 1.00, TLI = 1.00, RMSEA = 0.01 (90% CI 0.00–0.05), respectively. The proportion of the variance in parent-reported ADHD diagnosis and clinical ADHD symptoms accounted for by the models were 9% ($R^2=0.09$) and 11% ($R^2=0.11$), respectively. Both models accounted for 6% of the variance in parenting hostility and 5% of the variance in parenting consistency ($R^2=0.05$), 1% of the variance in parenting warmth ($R^2=0.01$), and 19% of the variance in concurrent distress ($R^2=0.19$).

Figure 1 presents the standardized parameter estimates for the models of parent-reported ADHD diagnosis and clinical ADHD symptoms, with the estimates for clinical ADHD symptoms presented in brackets. High postnatal distress (wave 1) and concurrent distress (wave 5) were significantly associated with high parenting hostility, and

Table 2 Descriptive statistics and correlations among the study variables

	Variables	1	2	3	4	5	6	7
1	Parent-reported ADHD diagnosis (wave 5)	–						
2	Clinical ADHD symptoms (wave 5)	228.93***[a]	–					
3	Postnatal distress (wave 1)	0.05**	0.07***	–				
4	Concurrent distress (wave 5)	0.06**	0.09***	0.43***	–			
5	Parenting warmth (wave 5)	− 0.02	− 0.06***	− 0.09***	− 0.08***	–		
6	Parenting hostility (wave 5)	0.14***	0.21***	0.17***	0.23***	− 0.30***	–	
7	Parenting consistency (wave 5)	− 0.06**	− 0.07***	− 0.18***	− 0.21***	0.19***	− 0.42***	–
	M	–	–	3.39	2.83	26.59	7.40	25.19
	SD	–	–	3.31	3.29	3.27	2.22	3.43
	Skewness	–	–	1.63	2.05	− 0.91	0.77	− 0.74

*$p < 0.05$, **$p < 0.01$, ***$p < 0.00$

[a]Chi square value

low parenting warmth and consistency when the child was aged 8–9 years (wave 5). High parenting hostility was in turn associated with a parent-reported diagnosis of ADHD and clinical ADHD symptoms, while concurrent distress, parenting warmth and consistency were not associated with parent-reported ADHD diagnosis or clinical ADHD symptoms.

The total indirect effects of postnatal distress on parent-reported ADHD diagnosis (total indirect effect 0.07 (lower 2.5% CI 0.03, upper 2.5% CI 0.10), $t = 3.85$, $p < 0.001$) and clinical ADHD symptoms (total indirect 0.08, (Lower 2.5% CI 0.05, upper 2.5% CI 0.12), $t = 6.80$, $p < 0.001$) were small but significant. The specific indirect pathways are provided in Table 3. The strongest indirect pathways were via parenting hostility and concurrent distress at wave 5. The indirect pathways via parenting warmth and consistency were not significant.

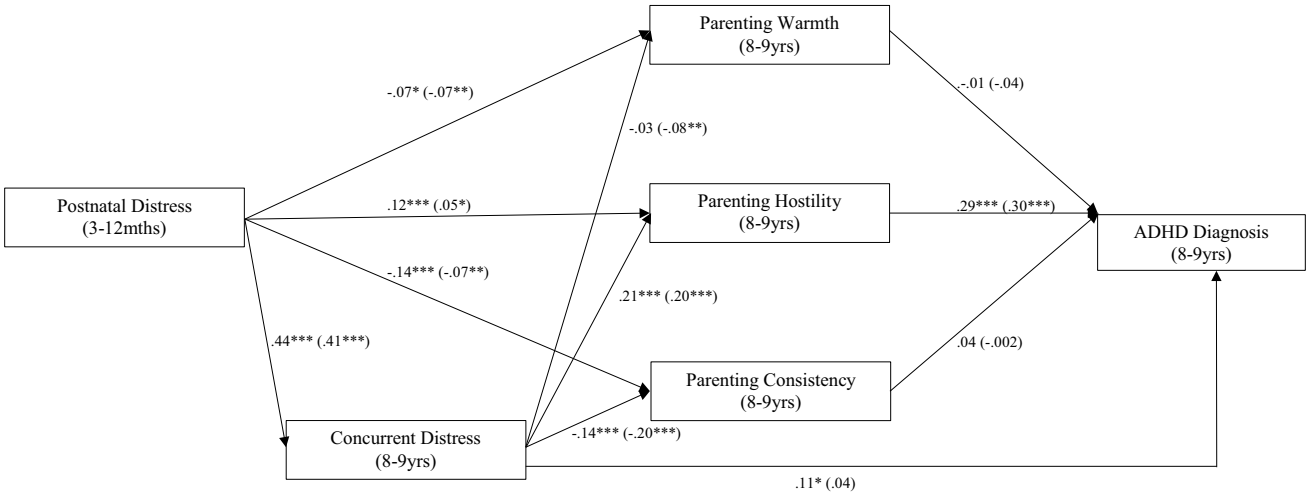
Testing for group differences by child's sex, socio-economic position, mental health problems during pregnancy, and use of anti-depressant medication during pregnancy

Multiple group analyses revealed that the model pathways differed for both parent-reported ADHD diagnosis and clinical ADHD symptoms by child sex but not SEP, maternal mental health problems or anti-depressant use during pregnancy. The Chi square difference test results are presented in Table S1. Given that a similar pattern of results was obtained for both outcomes, we only report on the model differences between males and females using parent-reported clinical ADHD symptoms. Figure 2 presents the standardized parameter estimates for males and females, with estimates for females in brackets. Follow-up tests were conducted to identify which model pathways were significantly different by sex. At an alpha level of 0.05, there was a significant difference in the pathway from postnatal distress to parenting hostility by sex ($p = 0.022$), where the relationship

Table 3 Standardized parameter estimates of specific indirect pathways from maternal postnatal distress to parent-reported ADHD diagnosis and clinical ADHD symptoms

Specific indirect pathways	ADHD diagnosis Indirect effect estimate (lower 2.5% CI, upper 2.5% CI)	Clinical ADHD symptoms Indirect effect estimate (lower 2.5% CI, upper 2.5% CI)
Postnatal distress → parenting warmth → ADHD	− 0.003 (− 0.01, 0.002)	0.001 (− 0.004, 0.005)
Postnatal distress → parenting hostility → ADHD	0.03 (0.01, 0.04)***	0.03 (0.02, 0.04)***
Postnatal distress → parenting consistency → ADHD	< 0.001 (− 0.01, 0.01)	− 0.004 (− 0.01, 0.004)
Postnatal distress → concurrent distress → ADHD	0.02 (− 0.01, 0.05)	0.03 (0.01, 0.06)*
Postnatal distress → concurrent distress → parenting warmth → ADHD	− 0.001 (− 0.004, 0.001)	< 0.001 (− 0.001, 0.002)
Postnatal distress → concurrent distress → parenting hostility → ADHD	0.03 (0.02, 0.03)***	0.03 (0.02, 0.04)***
Postnatal distress → concurrent distress → parenting consistency → ADHD	− 0.001 (− 0.01, 0.01)	− 0.002 (− 0.01, 0.002)

*$p < 0.05$, ***$p < 0.001$



Note: *$p < .05$, ** $p < .01$, ***$p < .001$; Model for females in brackets; Residual variances between parenting behaviors are correlated

Fig. 2 Standardized parameter estimates for the hypothesized clinical ADHD symptoms model for males and females

was stronger for boys than girls. However, at a Bonferroni adjusted alpha level of 0.003 to account for multiple contrasts, the difference was not significant. Finally, the proportion of the variance in clinical ADHD symptoms was 11% for both boys and girls ($R^2 = 0.11$).

Discussion

In this large, population-based sample, there is evidence that the relationship between maternal postnatal distress and child ADHD at age 8–9 years may be mediated through parenting hostility even after accounting for concurrent maternal psychological distress. This relationship was identified both when using parent-reported ADHD diagnosis and clinically elevated ADHD symptoms as the outcome variable.

Previous research has demonstrated that postnatal distress influences child psychosocial functioning through the impact it has on parenting behaviors [7, 8]. Our work has extended this finding by specifically examining whether there is an indirect relationship between postnatal distress and ADHD via parenting behavior. Consistent with Giallo et al. [8] findings regarding emotional/behavioral problems we found that parenting hostility, but not warmth or consistency, mediated the relationship between postnatal distress and ADHD. Our results differ from Elgar et al. [7] who reported that low parental warmth and parental rejection mediated the relationship between postnatal depressive symptoms and child externalizing behavior. It may be that postnatal depressive symptoms have more impact on parental warmth than the broader concept of postnatal distress used in the current study. Further, prior research in community samples of children with ADHD has found a limited association between parental warmth and ADHD [23]. The finding that parenting hostility was most strongly associated with ADHD is consistent with the literature that demonstrates negative parenting behaviors are observed more commonly for children with ADHD than non-ADHD controls [22–25].

Our findings indicate that mothers who experience postnatal distress may employ more hostile parenting behaviors which may in turn increase the risk of ADHD in their child. However, it is important to note that only a small amount of the variation in outcome was explained by the models. Furthermore, we only examined the relationship in one direction. There is likely to be a transactional relationship between parenting practices and child ADHD symptoms, whereby the child's and parent's behavior influence each other. It is possible that the child displayed challenging behaviors in infancy (e.g., colic or sleep problems) which contributed to the mother's postnatal distress [42, 43]. Additionally, the child's ADHD symptoms may induce hostile parenting practices [28, 44]. Future research should take a developmental approach to investigating the interplay between parental mental health, parenting, child behavior and ADHD symptoms by measuring all of these variables at multiple time points from infancy through to adolescence.

The study had a number of key strengths. It is a large prospective community based study, cohort retention was high, and we used two complementary outcome measures. Limitations of the study include that ADHD diagnosis was by parent report rather than clinically confirmed. However, our results were consistent for children with parent-reported diagnosis and for children with clinically elevated ADHD symptoms as assessed by combined parent and teacher reports. Our measure of parenting was through self-report of behaviors which may be subject to a social desirability

response bias. Our measure of maternal depression, anxiety and stress during pregnancy was self-reported and retrospective using a single item. We were unable to determine if the role of parenting in child behavior differs according to child age. There was some selection bias in the sample, with an over-representation of children with more highly educated parents and an under-representation of children from non-English speaking backgrounds and those living in rental properties. Further, selective attrition occurred over the course of the study with mothers from lower socio-economic backgrounds, born outside Australia, and from non-English speaking and Aboriginal or Torres Strait Islander backgrounds less likely to remain in the study. This likely resulted in better outcomes than one may see in the general population, although whilst we measured symptoms, rather than using a diagnostic instrument, the prevalence of clinically significant ADHD symptoms in this study was very similar to the prevalence rate of 7.4% identified in the most recent Australian national survey of youth mental health [45]. This study was only able to examine the relationship between maternal mental health and parenting, indeed there is a lack of information about paternal–child relationships in the broader literature. Future research should seek to examine whether paternal mental health and parenting practices are related to child's ADHD symptoms. Finally, although this study focused on specific aspects of maternal mental health and parenting, there are other factors worth consideration for inclusion in models that seek to understand the contribution of early life stress and adversity to the development of ADHD including smoking during pregnancy, stressful life events, and partner conflict and violence.

In conclusion, this study has demonstrated that the pathway from maternal postnatal distress to ADHD symptoms in 8–9-year-old children may be mediated through parenting hostility. This relationship holds even when accounting for concurrent maternal distress. Our findings highlight the importance of early identification and intervention for mothers experiencing postnatal distress, as treatment of these problems may prevent mothers from developing hostile parenting practices and disrupt the pathway to ADHD. Early intervention should include teaching parents effective strategies to manage stressful parent–child interactions rather than using hostile parenting behaviors. Our results also demonstrate the need for child health professionals to enquire about women's mental health during pregnancy and the postnatal period. If clinicians are aware of these issues, they will be better equipped to monitor the emergence of ADHD symptoms in children.

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Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states there is no conflict of interest.

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