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The impact of maternal depression in pregnancy on early child development

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Objective Postpartum depression in mothers is associated with developmental problems in their children. Many women who are depressed following childbirth are also depressed during pregnancy. The aim of this study was to examine the associations between maternal depressive symptoms during pregnancy and child development at 18 months of age.

Design A prospective cohort study, Avon Longitudinal Study of Parents and Children.

Setting The former county of Avon, southwest England.

Population All pregnant women in the defined area with delivery dates between April 1991 and December 1992, 9244 women and their children.

Methods Data were collected antenatally, at 18 and 32 weeks of gestation and at 8 weeks and 8 months postnatally, through postal questionnaires, including a self-report measure of depression (Edinburgh Postnatal Depression Scale [EPDS]). By the time their child was 18 months old, women completed five further questionnaires about their children's health and development.

Main outcome measure Child development at 18 months using a modified Denver Developmental Screening Test (modified DDST).

Results Applying the standard 12/13 cutoff, 1565 (14%) women were depressed antenatally but not at either time-points postnatally. Employing the modified DDST, 893 (9%) children were developmentally delayed at 18 months of age. Persistent depression (EPDS ≥ 10 at both time-points) is associated with developmental delay (adjusted OR 1.34, 95% CI 1.11–1.62). Applying the 12/13 and 14/15 cutoffs gave similar results. After further adjustment for postnatal depression, the effect sizes were slightly attenuated.

Conclusions These findings highlight the importance of depression in pregnancy. Some effects on child development attributed to postpartum depression are caused in part by depressive symptoms during pregnancy.

Keywords ALSPAC, antenatal depression, child development, pregnancy, postnatal depression.

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Background

It is widely recognised that depression is common during the postpartum period and that maternal postnatal depression can have a detrimental effect on child development (the term 'depression' and 'depressed' is used as a short-hand for those women who scored above the specified cutoff on the Edinburgh Postnatal Depression Scale [EPDS]. The EPDS is not a diagnostic tool and the women had not been assessed formally, therefore they had not been diagnosed as clinically depressed).[1–7] Investigations of the inter-relationships between maternal wellbeing and child development have mostly been conducted after childbirth with few studies collecting data prospectively, starting during pregnancy.[8–10] There is increasing evidence that the mother's mood during pregnancy is important. For example, uterine blood flow has been observed to change in association with anxiety during pregnancy,[11–15] and antenatal anxiety in late pregnancy is independently associated with children's behavioural/emotional problems at 4 years of age.[16] The children of women in socio-economically deprived families, who are at risk of depression antenatally, are at greater risk of developmental delay at 2 years of age.[17] The strongest predictor of postnatal depression is depression in pregnancy; however, antenatal depression may be more common than postnatal depression.[18,19] Although severity and chronicity of maternal depression are related to increased developmental problems in their children,[20–22] less is known about the importance of timing of

the exposure to maternal depression, in particular whether pregnancy is a sensitive period.

Closing the gap in the evidence about associations between parental depression and child health outcomes has important implications for both families and healthcare practitioners. The aim of this study was to assess the association between maternal depression in pregnancy and child development. Although fathers' mood influences child development,[23] the focus of this paper is on depressive symptoms in mothers. We hypothesised that antenatal depression scores have an impact on early childhood development that is independent of postnatal depression. This was tested, first, by investigating the association between maternal antenatal depression and a measure of child development and second, by examining the independent effects of antenatal depression. Data used to test this hypothesis were derived from the Avon Longitudinal Study of Parents and Children (ALSPAC),[24] a large community sample in England that has been followed prospectively since early pregnancy.

Methods

Samples

The ALSPAC study design included all pregnant women living in the geographical area of Avon, England, who expected to deliver their baby between April 1991 and December 1992, resulting in 14 062 live births. Recruitment, dropout and other methodologies have been described elsewhere.[24] Baseline data, including socio-demographic and family details, were collected from pregnant woman and their partners at 18 and 32 weeks of gestation using postal questionnaires. Those women who did not complete the antenatal questionnaires at both 18 and 32 weeks of gestation were excluded from these analyses, as were those children from multiple births.

Measures

Exposures

The exposure of maternal depression was assessed using the EPDS.[25] Although developed as a screening tool for depression following childbirth, this scale has been validated during pregnancy as well as outside the postpartum period.[26,27] The EPDS is a widely used 10-item self-rating questionnaire on which women rate their feelings over the previous 7 days, giving a score ranging from 0 to 30.[27] The EPDS has been used in many studies of depression in childbearing women, most typically in the first year postpartum. Women completed the EPDS at 18 and 32 weeks of gestation and then again at 8 weeks and 8 months postpartum. Women were categorised into three 'antenatal depression' groups: those with a score below the EPDS cutoff indicating the absence of depression and those with EPDS scores at or above the cutoff indicating depression either at 18 *or* 32 weeks (once), or at 18 *and* 32 weeks (twice); the latter as a measure of more persistent depression. *A priori*, to reflect the continuous nature of the data, there were three stages of the analysis undertaken. Each stage applied a different cutoff on the EPDS: 9/10, 12/13 and 14/15; 12/13 is the standard cutoff.

Post hoc, another method was also applied in which women were categorised at both 18 and 32 weeks of gestation into four groups depending on their EPDS scores (0–9, 10–12, 13–14 and 15–30) and data were scored as given in Table 1, creating an ordinal scale from 0 to 6. This method combined both levels of intensity (EPDS scores) and persistence (depressed once, twice or at both time-points during pregnancy) and aimed to better characterise any dose–response relationship, or threshold effect, between persistent antenatal depression and developmental delay.

Outcomes

A modification of the Denver Developmental Screening Test (modified DDST) was used as the child outcome. This is a screening questionnaire designed to identify cognitive and behavioural problems in preschool children. The data are presented as age norms. The more items a child fails to perform passed by 90% of his/her peers, the more likely the child manifests a significant developmental deviation. The items used in the screening test were from the Denver II that has been shown to be predictive of developmental delay.[28] It had been adapted for parental report through discussions with focus groups and piloting, using the same cohort as this study and found to significantly relate to the Griffiths Scales classification[29] for general development.[30] Parents completed a questionnaire at 18 months postpartum. The developmentally delayed group were those who failed two or more items, which was the case for 10% of the ALSPAC cohort.

Data analysis

Descriptive statistics and frequencies were used to examine the characteristics of the study population. The three cutoffs

Table 1. Categories allocated to each woman according to their antenatal EPDS scores. A woman's category at each time-point was then summed to indicate the severity and persistence of her depression

EPDS score at 18 weeks of gestation	EPDS score at 32 weeks of gestation			
	EPDS 0–9	EPDS 10–12	EPDS 13–14	EPDS ≥ 15
EPDS 0–9	0	1	2	3
EPDS 10–12	1	2	3	4
EPDS 13–14	2	3	4	5
EPDS ≥ 15	3	4	5	6

Category 0, EPDS 0–9; category 1, EPDS 10–12; category 2, EPDS 13–14; category 3, EPDS ≥ 15.

on the women's EPDS scores (9/10, 12/13 and 14/15) were applied at three different stages of the analysis. Bivariable tests of association and multivariable regression analyses were undertaken for each stage to quantify the individual relationships between maternal depression, potential confounding factors and developmental delay. Potential confounding factors entered into the analysis included demographic details for both parents, previous maternal depression, maternal anxiety, paternal depression and anxiety, life events in the previous year, gestation, gender and ethnicity of child, feeding method, postnatal mood of parents and life events postnatally (Table 2). Individual unadjusted odds ratios were calculated for each potential confounding and explanatory variable. For the multivariable analysis, because of the complex interrelationship between antenatal depression and child development, a conceptual framework was used to describe the hierarchical relationships between risk factors (Table 3).[31] This framework uses findings from previous research and guides the decision about which variables to include in the analysis. A multivariable regression analysis was undertaken applying the steps of the conceptual framework one by one starting with step 1. Following the inclusion of each step, variables were removed if, individually, they had minimal impact on the relationship between women with antenatal depression and developmental delay (less than 5% change in the odds ratio).[32] Those variables remaining in the model were carried forward and included together with the variables in the next step of the framework. Each variable was then individually re-entered at the end. The evidence for any negative effect of young maternal age on developmental delay is conflicting, therefore maternal age was retained.[33] This may also be a proxy variable for some other factor that has not been, or cannot be, measured. Due to missing data for the exposure and for potential confounding variables, a sensitivity analysis was undertaken using imputed data. In the multivariable models of our primary analyses, missing data on confounding variables resulted in a loss of almost 10% of the sample when the fully adjusted models were derived. To avoid any potential bias that might result when incorporating these confounding variables into the model, a missing data imputation technique was employed (missing imputation for chained equations)[34] using the procedure in STATA known as ice.[35] Imputation was restricted to confounding variables (no imputation of the outcome variables was performed). The results from the regression using imputed data were similar to those using unimputed data. This suggests that the missing data were not substantially biasing the findings and therefore only the analysis using the unimputed data is reported. Data

Table 2. Details of the potential confounding factors that were included in the conceptual framework and analysis

Potential confounding factors	Not depressed women EPDS < 13 (n = 8799), n (%)*	Depressed women EPDS ≥ 14 (n = 2299), n (%)*
Socio-economic		
Primiparous women	3911 (44.5)	922 (40.1)
Owned/mortgaged housing	6943 (78.9)	1417 (61.6)
Overcrowding	1272 (14.5)	591 (25.7)
No use of car	1749 (19.9)	771 (33.5)
No smoking first trimester	7035 (79.9)	1464 (63.7)
No alcohol first trimester	3938 (44.8)	1038 (45.2)
Parental		
Mother less than 20 years	234 (2.7)	146 (6.4)
Father less than 20 years	52 (0.6)	53 (2.6)
Mother: attained O'levels/equivalent	3091 (35.3)	802 (35.1)
Father unemployed	495 (6.4)	233 (12.6)
Have partner	8372 (98.4)	2040 (95.6)
Preconceptional/pregnancy		
Mother: previous depression	463 (5.3)	454 (19.7)
Mother: anxious	920 (10.5)	1562 (67.8)
Father: depressed	185 (2.7)	135 (8.2)
Father: anxious	274 (3.1)	118 (5.1)
<3 life events in past year	2135 (24.5)	189 (8.4)
Birth		
Preterm	462 (5.3)	150 (6.5)
Ever breastfed	6163 (77.9)	1344 (70.3)
Male child	4510 (51.3)	1194 (51.9)
Ethnicity: white	8545 (97.1)	2183 (94.9)
Postnatal		
Mother: no low mood	7176 (81.5)	1282 (55.9)
Father: no low mood	4378 (49.8)	876 (38.1)
<3 life events in past year	2456 (31.1)	1052 (54.7)

*These percentages represent the percentage response for the total number of answers for that particular question.

Table 3. Conceptual hierarchical framework of risk factors for child development

Step	Label for steps	
		Antenatal factors
1	Socio-economic	Parity, housing tenure, overcrowding, use of car, mother's smoking habit, mother's alcohol intake
2	Parental	Mother's age, partner's age, mother's education, partner's employment, have partner
3	Preconceptional/ pregnancy	Previous maternal depression, maternal anxiety, life events in previous year, paternal depression and anxiety
		Postnatal factors
4	Birth	Gestation, breastfeeding, gender, ethnicity of child
5	Postnatal	Postnatal mood of parents, life events postnatally

Modified from Victora *et al.*[31]

analysis was performed using STATA (STATA version 8) (Stata Corporation, College Station, TX, USA).

Ethics approval for the study was obtained from the ALSPAC Law and Ethics Committee and the Local Research Ethics Committees.

Results

Demographic characteristics

Complete antenatal EPDS data (18 and 32 weeks of gestation) were available for 11 098 women, of whom 4833 (44%) were expecting their first child. Women with incomplete antenatal EPDS data were excluded from the study. There were few demographic differences between these women and those retained in the analysis. The main differences were that, compared with the women who were excluded, a higher percent of included women had a partner, a lower percent achieved O'level or equivalent qualifications or had partners who were unemployed (Table 4).

Child developmental outcomes

Parental report on the modified DDST ($n = 10\ 125$) identified 893 (9%) children who were developmentally delayed. The children were mainly White British (97.7%), 612 (5.5%) were preterm, 5704 (51.4%) were male children and 7505 (76.4%) ever breastfed. There were just 36 (0.4%) children whose parents reported that their child was usually unwell.

Antenatal depression

Using the standard 12/13 cutoff, 8262 (74.4%) women were not depressed either antenatally or postnatally, 1565 (14.1%) women were depressed on at least one occasion antenatally but at neither postnatally. Five hundred and thirty-seven (4.8%) women were depressed on at least one of the two measurement occasions postnatally but at neither antenatally (Table 5). There were just 156 (1.4%) women who were persistently depressed antenatally and postnatally.

When combined with the modified DDST data, of the 11 098 women with complete EPDS scores, antenatal data were available on 9244 (83%) women and children.

Multivariable analysis

For the first stage of the analysis, complete case analyses for the EPDS 9/10 cutoff were applied. Those women whose EPDS scores were <10 at both 18 and 32 weeks of gestation formed the baseline comparison group. The final model provided adjusted estimates of the odds of developmental delay of tobacco smoked in the first trimester, mothers' age and life events at 8 months. The odds ratio for developmental delay associated with antenatal depression at both time-points was 1.24 (95% CI 1.04–1.49) and when adjusted for smoking, maternal age and life events was 1.34 (95% CI 1.11–1.62).

For the second stage, the EPDS 12/13 cutoff was applied and the results followed a similar pattern, with a 50% increase in the odds of developmental delay (adjusted odds ratio 1.50; 95% CI 1.15–1.96) (Table 6). For the third stage, applying the 14/15 cutoffs gave similar results, although with wider and nonsignificant confidence intervals. For each cutoff, after further adjustment for postnatal depression, the effect sizes were similar to those of the unadjusted models for each cutoff (Table 6).

Applying the conceptual framework, paternal depression and antenatal anxiety were included in the analysis. However, as they both had minimal impact on the relationship between antenatal depression and the odds of developmental delay, they were removed from the model.

Post hoc analysis

In the *post hoc* analysis, an ordinal scale combining both intensity (EPDS scores) and persistence (above EPDS cutoff at both 18 and 32 weeks of gestation) of symptoms was applied. The odds ratios and 95% CIs show a consistent pattern between the multivariable models without and with adjustment (Figure 1). There is a suggestion of a threshold

Table 4. Demographic descriptions for the women who were retained in the analysis and those who were excluded from the study

	Included women ($n = 11\ 098$), *n* (%)	Excluded women ($n = 3370$), *n* (%)
Primiparous [missing data for variable]	4833 (44.3) [180 (1.6)]	971 (47.3.) [1317 (39.1)]
Average age (SD)	28 (4.8)	27 (5.3)
Age in years (range)	15–44	15–44
Achieved O'levels/equivalent [missing data for variable]	6002 (54.4) [60 (0.5)]	892 (63.2) [1958 (58.1)]
No partner [missing data for variable]	227 (2.1) [459 (4.1)]	63 (5.7) [2267 (67.3)]
Partner unemployed [missing data for variable]	728 (7.4) [1228 (11.1)]	215 (14.1) [1840 (54.6)]

Each variable have missing data and therefore percentages are presented for both missing data and for the variable in question. For the variable itself (e.g. no partner) what is taken as 100% = 11 098 (or 3370)—missing data. For 'partner unemployed', the data for no partner are excluded from the missing data figures.

Table 5. Women's depression scores during pregnancy (18 and 32 weeks of gestation) and postnatally (8 weeks and 8 months) using the EPDS and the standard 12/13 cutoff (n = 11 098)

Antenatally	PN EPDS <13, *n* (%)*,***	PN EPDS at 8 weeks, *n* (%)*,***	PN EPDS at 8 months, *n* (%)*,***	PN EPDS at 8 weeks and 8 months, *n* (%)*,***	Total, *n* (%)**
AN EPDS < 13**	8262 (74.4)	247	215	75	8799 (79.3)
AN EPDS at 18 weeks**	518	69	55	33	675 (6.1)
AN EPDS at 32 weeks**	603	88	73	81	845 (7.6)
AN EPDS at 18 and 32 weeks**	444	103	76	156	779 (7)
Total	9827 (88.5)	507 (4.6)	419 (3.8)	345 (3.1)	11 098 (100)

*Percentages given relative to total sample.
**AN EPDS: antenatal EPDS scores.
***PN EPDS: postnatal EPDS scores.

effect between the groups who scored 3 and 4 on the ordinal scale, indicating an underlying effect of antenatal depression, although none of the results are statistically significant and the wide confidence intervals arise from the small numbers involved, especially in the severe and persistent depression groups.

Discussion

Summary of the study findings

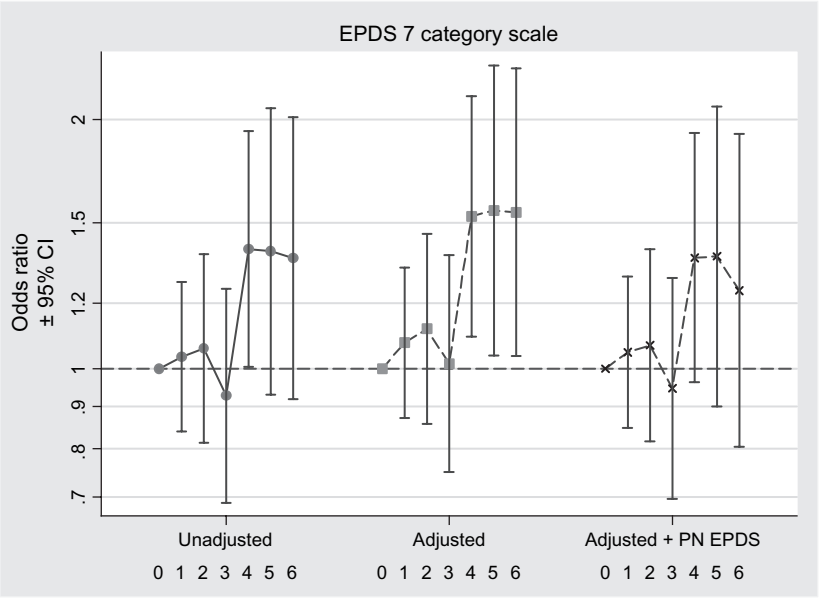
This study set out to test the hypothesis that antenatal depression has an adverse impact on early childhood development. This was tested, first, by investigating the association between women's antenatal depression and a child development measure and second, by examining the effects of antenatal depression independent of postnatal depression. Applying the modified DDST at 18 months of age as a measure of development, an association was found between persistent depression during pregnancy and developmental delay with a 50% increase in the odds of developmental delay associated with persistent antenatal depression. While the presence of postnatal depression had a modifying effect on the odds, we found evidence of an independent and statistically significant 34% increase in the odds of developmental delay when the standard 12/13 cutoff on the EPDS was used to indicate depression.

However, postnatal depression may be either a factor on the causal pathway between antenatal depression and child development, in which case it would be inappropriate to adjust for its effects, or it may act as an independent influence on child development, in which case it would be appropriate to adjust for its effects. This study set out to examine specific effects of antenatal depression on child development, independent of postnatal depression, rather than to investigate any potential link between the two. The observed threshold effect is difficult to explain by confounding factors; moreover,

Table 6. Unadjusted and adjusted odds ratios of the relationship between antenatal depression and developmental delay

Variables	Unadjusted		Adjusted*		Adjusted* + PN depression**	
	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
Stage 1						
EPDS ≥ 10 at 18 or 32 weeks	0.99 (0.84–1.19)	0.996	1.06 (0. 88–1.27)	0.557	1.11 (0.85–1.23)	0.819
EPDS ≥ 10 at 18 and 32 weeks	1.24 (1.04–1.49)	0.023	1.34 (1.11–1.62)	0.003	1.23 (1.00–1.51)	0.047
Stage 2						
EPDS ≥ 12 at 18 or 32 weeks	1.04 (0.86–1.29)	0.641	1.14 (0.92–1.40)	0.231	1.07 (0.86–1.33)	0.530
EPDS ≥ 12 at 18 and 32 weeks	1.35 (1.04–1.76)	0.025	1.50 (1.15–1.96)	0.003	1.34 (1.01–1.78)	0.043
Stage 3						
EPDS ≥ 14 at 18 or 32 weeks	1.16 (0.92–1.47)	0.218	1.26 (0.99–1.60)	0.065	1.16 (0.90–1.49)	0.245
EPDS ≥ 14 at 18 and 32 weeks	1.34 (0.91–1.98)	0.140	1.49 (1.00–2.20)	0.050	1.27 (0.84–1.92)	0.254

PN, postnatal.
*Adjusted for tobacco used in first 3/12 of pregnancy, mothers' age, life events at 8 months.
**Adjusted for postnatal depression as well.



0 = No depressive symptoms.
6 = Greatest intensity and persistence of symptoms.
*Adjusted for tobacco used in first trimester of pregnancy, mothers' age and life events at 8 months.

Figure 1. Association of antenatal depression (using an ordinal scale) and developmental delay (*post hoc* analysis). Results for the unadjusted analyses, adjusted for confounding factors* and further adjusted for postnatal depression (PN EPDS) are illustrated.

adjustment for suspected confounders did not markedly change the estimates for the effect of cumulative exposure to antenatal depression. So, although not statistically significant, the consistent association between antenatal depression and child development seems credible. While the link between antenatal and postnatal depression is widely recognised,[8,9,18] aspects of both antenatal and postnatal depression in mothers are likely to be important in relation to early child development. Any persistent depression during pregnancy has the potential to be an important risk factor for developmental delay in childhood. On the basis of the threshold effect, these findings suggest that, for those women who reach this threshold during pregnancy, there is a risk of developmental delay in early childhood.

Strengths and limitations

Strengths

This study uses a large prospective, longitudinal design and recruited a large community sample that is broadly representative of the UK population when compared with the 1991 census.[24] Many potential confounding variables were controlled for during the process of the analysis. However, all but three were removed by the confounder selection strategy for the final model. The evidence for an adverse affect of young maternal age on child development is conflicting and therefore maternal age was included.[33] It may also be a proxy variable for some other factor that has not been, or cannot be, measured. Nevertheless, there remains a possibility of residual confounding of some unmeasured variables, in particular mother–child interaction, a measure of which was not available for these analyses. This has been shown to be related to both maternal depression and lead to poor cognitive development in children.[22,36]

A conceptual framework was used to manage the complex hierarchical inter-relationship between antenatal depression and child development and to describe the relationships between risk factors. The framework guided the multivariable analysis and was thus not based on purely statistical association.[31]

To reflect the continuous nature of the data and to explore the possibility of a dose–response effect between antenatal depression and developmental delay, separate stages of the analysis used different cutoffs on the EPDS: 9/10, 12/13 and 14/15. Using these three cutoffs do not necessarily deal with the problem of misclassification of cases but rather shifts the balance of false positives (women classified as having depression but who are not depressed) and false negatives (women classified as not having depression but who are depressed) by a particular cutoff. A *post hoc* analysis tried to address this by creating an ordinal scale combining both intensity and persistence of symptoms. Analysing the data in this way allowed us to explore the possibility of a dose–response relationship to be investigated further. However, underlying this method is the assumption that the association with developmental delay is the same for women in each category on the ordinal scale.

Limitations

Despite the overall size of the cohort because of the relatively small number of women with severe and persistent depression, our analysis has limited power to detect modest but clinically important effects. The modified DDST is a binary outcome and, by the nature of any population, there were few women who experienced more severe depression that further limits the power.

Maternal low mood may make completion of the modified DDST difficult:[37] women who successfully completed the questions relating to their child's development may be less likely to experience depression than those nonrespondents. However, this would probably result in an *underestimation* of any association. Moreover, the identification of those women with depression was undertaken prior to the analysis of the outcome. Typically, within large-scale studies, most of the data relied on maternal (or parental) report. There is always the potential for some effect of reporter bias, e.g. depressed mothers not recognising their children's abilities. However, the findings *are* broadly consistent with an earlier study that looked at associations between women at risk of antenatal depression and child development in a socio-economically deprived population, independent of postpartum depression, where child development was assessed using a trained assessor, not by parental completion as in this study.[17] A sensitivity analysis was undertaken imputing missing data, which suggested missing data were not biasing the results.

Generalisability of the study findings

This was a large community-based population sample that was followed-up for 18 months. The prevalence estimates of probable depression, as assessed by a screening tool both antenatally and postpartum, are similar to those reported in other studies.[5,22,38] Comparison is difficult because few studies have been undertaken on *prospectively* collected antenatal depression data. Those that have, found no association with antenatal depression and preschool children's development after adjusting for postpartum depression.[2,6] However, the number of women with antenatal depression in these studies was small. As mentioned above, the findings *are* broadly consistent with an earlier study that looked at associations between women at risk of antenatal depression and child development in a socio-economically deprived population, independent of postpartum depression.[17] Our results also fit with other studies that reported associations between postpartum depression and child development[5,8,22] and, using the ALSPAC data, between antenatal anxiety and children's emotional and behavioural problems.[16]

Our findings are also consistent with studies on rodents and nonhuman primates, which indicate that maternal anxiety during pregnancy can influence the developing fetus, resulting in delay in motor and cognitive development and impaired adaptation to stressful situations.[39] Moreover, studies have shown a link between antenatal anxiety and cognitive, behavioural and emotional problems in children, after adjusting for postpartum factors.[13] These results support a fetal programming hypothesis whereby biological systems adapt to environmental input during sensitive periods of development, e.g. the fetal period;[11] anxiety experienced by the mother has a direct influence on the development of the hypothalamic–pituitary–adrenal axis in the fetus.[12] Chronic maternal distress compromises the normal regulations of hormonal activity during pregnancy. Additionally, children of depressed mothers may inherit directly a vulnerability to depression.[40] It has also been suggested that the magnitude of the long-term effects of antenatal maternal anxiety in the child could be substantial.[41]

Postpartum depression is a known risk factor for behavioural/emotional problems in children, but it is less clear whether postnatal depression leads to cognitive delay in children unless it is prolonged or severe.[36] When postpartum depression was included in these analyses, persistent antenatal depression remained an important risk factor for developmental delay and this is likely to be magnified for those who might be diagnosed as clinically depressed. This implies that this association is unlikely to be fully explained by raised postnatal depression in those women who experienced antenatal depression. Therefore, some effects on child development attributed to postpartum depression may be caused in part by depression in pregnancy.

Implications of the study

These findings add to the growing body of research, suggesting that maternal psychological wellbeing during pregnancy has important consequences for child development. Women who are between 15 and 44 years form a group in which depression is the leading cause of disease burden worldwide.[42] Therefore, obstetricians, midwives and GPs can play an active role in assessing and identifying maternal depression.[43–45] Family and personal histories of depression and postpartum depression are known risk factors, and therefore, the use of these by healthcare professionals may be helpful in identifying women at risk. This is highly relevant for practice because maternal mental health problems are associated with the health of women's partners, their children's health, development and use of health services.[46,47]

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