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Prenatal Mood Disturbance Predicts Sleep Problems in Infancy and Toddlerhood

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Abstract

Background—Experimental animal data link prenatal stress with sleep disturbance in offspring, but the link in humans is unclear.

Aims—To investigate the link between prenatal maternal anxiety and depression and infant sleep disturbance from 6 to 30 months of age.

Study Design—Longitudinal prospective study of a large birth cohort from pregnancy to 30 months. Questionnaire measures of anxiety and depression were completed by mothers at 18 and 32 weeks gestation and at 8 weeks and 8 months postpartum

Subjects—The ALSPAC cohort, a prospective community study of women in the UK who have been followed since pregnancy.

Outcome measures—Measures of total sleep time, number of awakenings, and broadly defined sleep problems were available on children at ages 6, 18, and 30 months.

Results—Reliable measures of total sleep time, nighttime awakenings, and sleep problems were identified at 6, 18, and 30 months. Higher levels of prenatal maternal anxiety and depression predicted more sleep problems at 18 and 30 months, after controlling for postnatal mood and obstetric and psychosocial covariates; the association was not restricted to clinical extremes. No link with total sleep time was observed.

Conclusions—Mood disturbance in pregnancy has persisting effects on sleep problems in the child, a finding that is consistent with experimental animal research. The findings add to a growing

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literature showing that maternal prenatal stress, anxiety, and depression may have lasting effects on child development.

Keywords

prenatal anxiety and depression; sleep disturbance; community study

Sleep problems in young children are among the most common concerns reported to pediatricians; prevalence estimates suggest an affected rate of 20–30%(1–6). Sleep is thought to have important regulatory functions and, as such, is regarded as an index of healthy development. It is not surprising, therefore, that disturbance in sleep in early childhood is linked with a range of current and subsequent behavioral and physiological disturbances(7–10). Many clinical conditions are associated with sleep disturbance(11), and a number of psychosocial correlates have been identified(12), but the origins of sleep problems in most cases remain unclear.

Experimental animal research findings show that prenatal stress is a reliable predictor of sleep patterns(13), perhaps indexing a broader influence on circadian patterns and homeostatic regulation(14). In fact, sleep is one of many outcomes causally linked with prenatal stress in the animal literature(15–19). The hypothesized mechanism is that elevated prenatal exposure to glucocorticoids disrupts or perhaps programs the fetus' hypothalamic-pituitary-adrenal (HPA) axis and its diurnal pattern(20).

Human research also identifies prenatal anxiety or stress as a significant risk for behavioral/emotional and cognitive development in the child(21–25), but the extension to sleep problems has received very little attention(26). This study tests this hypothesis in a large prospective, longitudinal community sample. We investigated the degree to which prenatal maternal anxiety and depression predicted sleep problems in the infant and toddler, and the extent that this prenatal risk operates independently of other prenatal risks, obstetric outcome, and *post*natal mood disturbance.

METHODS

Subjects

The study is based on the Avon Longitudinal Study of Parents and Children (ALSPAC) a longitudinal, prospective study of women, their partners, and an index child(27). The study design included all pregnant women living in the geographical area of Avon, England who were to deliver their baby between 1 April 1991 and 31 December 1992. It was estimated that 85 – 90% of the eligible population took part. The average age of the women at pregnancy was 28 years (range 14 to 46 years). Approximately 45% of the women were expecting their first child; 6% of the women had 3 or more children. All data were collected via postal questionnaires (further information is available at http://www.alspac.bris.ac.uk).

The core ALSPAC sample consisted of 14,541 pregnancies that resulted in 14,676 known fetuses of which 14,062 were live births. In the current study, which included data through 30 months postpartum, sleep data were available on 11,490 at 6 months, 11,124 at 18 months, and 10,323 at 30 months; the sample was evenly divided between boys and girls at each age. Attrition analyses are reported below. The study was approved by the ethics committee at the University of Bristol and informed written consent was obtained.

Procedures and Measures

Sleep data were available from questionnaire assessments at 6, 18, and 30 months; data on predictors and covariates were included from measures collected at 18 and 32 weeks prenatal and 8 weeks and 8 months postnatal.

Sleep problems—It is not feasible in large-scale community and epidemiological investigations to include laboratory (e.g., polysomnography) or other intensive observational (e.g., actigraphy, sleep logs) measures of sleep. Instead, as with other larger-scale studies, the current study assessed sleep according to parent report. There is no consensus on which measure of sleep is most appropriate, but there is considerable consensus on which types of items and sleep behaviors should be assessed. Following previous studies (5;28–31), we made several distinctions in measuring sleep. The first was the amount of sleep and the second was a specific measure of the frequency of awakening during the night. At 6, 18, and 30 months, parents were asked to report the number of hours their child typically slept during the day and night (i.e., wake-up and bedtime and naptimes) and the number of times the child awoke during the night (i.e., after sleep onset). We also included a third measurement distinction, a broad index of sleep problems. At 18 and 30 months, parents were asked to report if, during the past year, their child regularly refused to go to bed, woken very early, had difficulty going to sleep, had nightmares, continued to get up after being put to bed, woke in the night, or got up after only a few hours sleep. These are indicators of sleep problems widely used in the research literature(5;32). In the absence of a priori notions about how these sleep problems might co-occur or which particular forms of sleep problems would be linked with prenatal anxiety and depression, we collapsed the 7 indicators into a single problem scale (each items was rated as occurring ['1'] or not ['0']). Cronbach alphas of the "sleep problems" scale at 18 and 30 months were .75 and .72, respectively, suggesting that these items indexed a common underlying sleep problem construct. In the absence of clear diagnostic criteria, we used continuous ratings of total sleep time (computed as the sum of sleep time during the day and during the night), number of awakenings, and sleep problems (using the above 7-point scale); analyses of continuous data are complemented by analyses of clinical/extreme scores defined as 2 SD above the sample mean.

Maternal depression and anxiety—Maternal anxiety was measured using the anxiety items from the Crown-Crisp experiential index (CCEI), a validated self-rating inventory (33; 34); example items include "worry a lot" and "feeling strung up inside." Internal consistencies (Cronbach's alpha) exceeded .80 for each of the 4 assessments. There is no established clinical cut-off for this measure; we therefore identified as anxious mothers who scored in the top approximately 15% (based on scores at 18 weeks prenatal) for categorical analyses.. Maternal depression was assessed using the Edinburgh Postnatal Depression Scale (EPDS), a widely-used 10-item self-report questionnaire that has been shown to be valid in and outside the post-natal period (35;36). Internal consistencies of the EPDS exceeded .80 at each of the four assessments. The EPDS has an established cut-off score of 12 or more that identifies women at high risk for diagnosed depression with high sensitivity and specificity(29).

Covariates—Included as covariates were factors that have been shown to predict sleep problems or factors that covary with, or result from, maternal mood disturbance. Specifically, we included prenatal alcohol use (coded '0' if <1 units/day, '1' if >1 units/day), smoking (coded '0' if no tobacco in the 2 weeks prior to completing the prenatal questionnaire, '1' if any), maternal education (codings were particular to the UK educational setting, ranging from '0' for no qualifications to '5' for university degree), crowding (a continuous rating of the number of persons/room), the ratio of birth weight to gestational age, maternal age, and child sex. We also included *post*natal anxiety and depression assessed at 8 weeks and 8 months to test the hypothesis that there was a particular effect on sleep from mood in the prenatal period.

Alternative coding of the smoking and alcohol scales were not feasible because of the low level of use reported. In addition, the ratio of birthweight to gestational age optimized the prediction from the birthweight and gestational age variables.

Statistical analysis—Bivariate analyses linking maternal mood and sleep problems at each assessment are reported, followed by multivariate analyses that statistically control for prenatal and postnatal risk and obstetric outcome. Given the large sample size and our interest in assessing whether the findings might carry clinical significance, we present the main analyses using categorical data, using the cut-offs noted above. Our strategy for handling the multiple measures of anxiety and depression on the four occasions (two prenatal, two postnatal) was shaped by two considerations. The first was to demonstrate that there was something particular about anxiety and/or depression in pregnancy that could not be accounted for by postnatal mood disturbance. This was accomplished by including both anxiety and depression at two postnatal assessments, 8 weeks and 8 months. The second was to consider if there was an effect of timing of prenatal anxiety or depression, as implied by animal findings but as yet uncertain in human research. The central issue here is whether mood disturbance at one point in pregnancy is a significantly better predictor of outcome than mood disturbance at a different point in pregnancy. In the absence of a strong a priori hypothesis about timing in pregnancy (and the difficulty in inferring timing from the substantial continuity between pregnancy assessments) we included anxiety and depression at 18 and 32 weeks pregnancy in the initial analyses.

RESULTS

Attrition analyses and missing data

In the analyses presented below we used mean substitution to replace missing data on covariates; in each case the percent of missing data was 10% or less. The exception was alcohol use; we did not use mean substitution because of the possibility that those who refused to complete that measure may have refused to answer because of high levels of use. Comparisons of results with and without missing covariate data yielded nearly identical results, indicating that bias associated with attrition on the covariates was minimal and that further investigation into optimizing methods for managing missing data was unnecessary.

It is possible that the children (and their mothers) for whom sleep data were available through 30 months differed from those children (and their mothers) for whom sleep data were unavailable. We examined those children who did not have valid sleep data at either 18 *or* 30 months (27.5% of sample) and compared them with children for whom sleep data were available (72.5% of sample) on key demographic and predictor variables collected in pregnancy and the early postnatal period. Analyses demonstrated that children for whom sleep data were unavailable had mothers who reported greater anxiety, depression, tobacco use, and crowding than children for whom sleep data were available. Those without valid sleep data also had lower birth weight for gestational age, education levels, and maternal age, compared with children for whom sleep data were available; the two groups did not differ on mother's alcohol use or child's sex. The magnitude of effect was small overall, but nevertheless significant in this large sample. The inclusion of these covariates in the main prediction analyses eliminates some of the bias in estimates associated with selective attrition. In addition, we include in the logistic regression a dummy variable identifying those cases for whom missing data were estimated; this procedure

Definition of sleep disturbance at 6, 18, and 30 months—Three measures of sleep were considered: a) Total time spent sleeping (day plus night), b) frequency count of awakenings in the night, and c) sleep problems scale. These measured overlapped modestly at

each time point, with correlations ranging from a high of .44 between number of awakenings and sleep problems at 18 months, to a low of −.22 between total sleep and sleep problems at 30 months. These indicators were therefore analyzed separately. Descriptive data for sleep measures and sample characteristics are given in Table 1.

Bivariate associations

Bivariate associations between anxiety and depression and the measures of sleep at 6, 18, and 30 months are reported in Table 2. To facilitate interpretation we report associations using the cut-off scores indicating clinical and more severe levels of anxiety and depression. Statistical tests on samples of this magnitude are significant even for trivial differences; however, it is clear that there are important and clinically meaningful effect sizes. Three patterns stand out. The first is that for no assessment period is there a meaningful link between prenatal maternal mood and the amount of total infant sleep. Second, starting at 18 months of age, there is a consistent link between pre- and postnatal maternal mood disturbance and the number of awakenings and total sleep problems, with effect sizes approximating 1/3 of a standard deviation. Third, the magnitude of effect was very similar for each of the measures of anxiety and depression in the prenatal and postnatal period. Multivariate analyses were then conducted to examine if prenatal maternal mood predicted sleep problems independent of postnatal mood and covariates.

Prediction of sleep outcomes from prenatal mood controlling for postnatal mood and covariates

Analyses predicting total sleep time from prenatal mood were unnecessary given the lack of significant bivariate associations. Analyses were then directed to the remaining two indicators of sleep, total number of awakenings and the sleep problem scale. Multivariate analyses showed that prenatal anxiety and depression did not predict the total number of awakenings at 18 or 30 months after postnatal anxiety and depression and the covariates were statistically controlled (all F's ≤ 1.01).

However, there was strong and robust evidence that prenatal mood disturbance, using the average of the two prenatal assessments, predicted total sleep problems at both 18 and 30 months of age independent of postnatal mood and the obstetric and psychosocial covariates. Table 3 indicates that prenatal anxiety and depression predicted sleep problems at 18 months of age, independent of postnatal anxiety and depression (at 8 weeks and 8 months), and the covariates noted above. An identical pattern was observed for sleep problems at 30 months (Table 4), with the consistent magnitude of effect implying no meaningful diminution of effect in the sample, at least across this 12-month period. Of the other variables in the models, only younger maternal age demonstrated a consistent independent relation to sleep problems at both 18 and 30 months ($B = -.04$, $SE = .009$, $p < .000$ and $B = -.05$, $SE = .011$, $p < .000$ for 18 and 30 months, respectively). Indicators of social class also predicted sleep problems, although the specific index that was significant differed at the two assessments. Specifically, lower maternal education predicted more sleep problems at 18 months only ($B = -.11$, $SE = .04$, $p < .007$); greater crowding predicted more sleep problems at 30 months only ($B = -.27$, $SE = .09$, $p < .002$). The regression analysis also shows that postnatal mood disturbance predicted sleep problems at 18 and 30 months of age, implying that there are likely several sets of processes by which maternal mood disturbance may influence infant sleep.

Supplementary analyses

Several kinds of supplementary analyses confirmed the robustness of the link between prenatal mood and infant sleep problems. For example, a similar pattern of findings was obtained when we examined more severe sleep problems, defined as a score of 2 standard deviations above the mean, and applied logistic regression analysis. Odds ratios (or effect sizes) for the prenatal

mood disturbance variables in this analysis were small but highly significant ($p<.005$; not tabled; available from the first author). Also, consistent with the nearly identical bivariate associations for the 18- and 32-week gestation measures reported in Table 2, there was no reliable evidence of a timing effect of mood disturbance in pregnancy. That is, individual measures of depression and anxiety at 18 and 32 weeks gestation did not improve prediction of sleep outcomes over the averaged measure at 18 and 32 weeks.

The moderate stability of sleep problems at 18 and 30 months ($r = .45$) suggested that the persistence of effect from prenatal mood disturbance obtained at the latter assessment might have resulted entirely from its association with, or establishment of, problems at 18 months. That was not entirely supported by a regression analysis; thus, when we added sleep problems at 18 months into the regression model predicting sleep problems at 30 months (as well as the postnatal mood and covariates listed above), prenatal anxiety and depression continued to predict sleep problems at 30 months ($B = .03$, $SE = .01$, $p < .02$ and $B = .026$, $SE = .01$, $p < .08$ for prenatal anxiety and depression, respectively).

The final supplementary analysis examined if there was robust evidence that certain sleep problems were more consistently predicted by prenatal mood disturbance than others (of the seven assessed); there was no evidence that this was the case (details available from the first author).

DISCUSSION

Sleep problems and problems in rhythmicity are multiply determined, and are known to be influenced by several factors including in utero exposures of several types(37), such as prematurity(38), and feeding schedule(39). Findings from this large-scale community study indicate that variation in sleep difficulties at 18 and 30 months is associated with prenatal maternal anxiety and depression. This finding was robust, that is, evident at successive assessments, and evident even after controlling for obstetric outcome and psychosocial risk and *multiple* measures of *post*natal depression and anxiety. The findings support considerable animal evidence and extend limited human data showing that prenatal factors may have persisting influences on sleep in the child.

Before considering the scientific and clinical implications of the work, we first note some limitations of the study. Assessing sleep using maternal report is an established methodology, particularly for large-scale studies, and the measure used here assessed sleep behaviors that are widely assessed in survey studies; however, this measure may not index the same kinds or degree of problems as laboratory measures. In the absence of a priori hypotheses about particular forms of sleep problems, we defined a broad sleep problem phenotype. We cannot determine whether or not children who exhibited problems on this scale are at risk for discrete sleep disorders and parasomnias, but the prediction of the most widely reported sleep problems means that these findings carry clinical meaning. Reliance on maternal report also raises concern about rater bias inflating the link between prenatal mood and infant sleep; however, the inclusion of multiple *post*natal measures of depression and anxiety substantially undermines this explanation. Also, we did not have a direct index of the putative mediating mechanism implied by the animal work, namely, HPA axis functioning. Accordingly, this findings needs to be followed up with smaller laboratory investigation that incorporates biological mechanisms. Set against these limitations are several important strengths of the study, including a prospective longitudinal design, a large community sampling frame, and multiple measures of maternal mood and infant/toddler sleep.

Animal investigations have demonstrated that early experiences influence the development (e.g., establishment) of the 24-hour diurnal pattern to the HPA system(40). Parallel evidence

in the human is limited, although several possible prenatal risks for the child's sleep have been suggested(41–43). Findings from the current study build on the animal work and extend previous human studies. Our use of a conservative analytic strategy (e.g., covarying multiple measures of *post*natal maternal mood) provided particular leverage in testing the hypothesis that there is something particular about prenatal anxiety and depression that confers risk for sleep disturbance in the child. The finding that this association persisted across two occasions of measurement implies that the effect is not transient; quite how enduring the effect is requires further follow-up study.

Further human research that incorporates a direct index of the mediating mechanism implied in animal work, namely the action of glucocorticoids, is important for at least two reasons. First, several studies report a link between the establishment of a diurnal pattern in cortisol and sleeping through the night in infancy(44). The measure of sleep disturbance used in this study was somewhat broad, but consistent with, and incorporated signs of, a delayed onset of consolidated sleep at 18 and 30 months. It may be that exposure to prenatal maternal anxiety/depression and the implied associated increased exposure to glucocorticoids(45) disrupts the onset of a normal diurnal pattern and a normal sleep cycle in the child. Further research along these lines also needs to consider how this process may be coordinated with the development of the suprachiasmic nucleus, a key contributor to circadian rhythmicity. Second, it remains to be seen if the sleep problems observed here can account for the reported behavioral and emotional and cognitive problems previously linked with prenatal maternal anxiety, presumably also through disturbance in the HPA axis. Research of that kind might provide one etiological explanation for why sleep and behavioral disturbances covary across development (8;46;47). Research into prenatal mood disturbance and sleep problems may also contribute to the growing interest in the links between sleep quality and general health outcomes, and the possibility that there may be fetal origins of a variety of health outcomes(48).

The current findings do little to resolve lingering questions about timing

Alongside these positive findings, several non-findings may also be of some interest. It is an interesting developmental point that, at a period at which sleep is not yet consolidated in all infants, there was no link with prenatal mood. Variation in sleep "problems" (e.g., awakening in the night) at that early period are not uncommon and may reflect normative variation in the consolidation of sleep. Also, in later assessments, which took place long after the expected onset of consolidated sleep, the amount of sleep remained strikingly unrelated to prenatal mood, and was non-significant even in a sample of many thousand children. These observations provide important clues to clarifying which sleep phenotypes may be most profitable to study in further research.

One intriguing hypothesis that needs testing is that prenatal intervention to reduce anxiety and depression may have beneficial carry-over effects for the baby, including sleep. Evidence-based psychological interventions may carry special advantages for women in pregnancy given the concerns about the use of drug treatment in pregnant women(49). Studies of sleep-disturbed children often find evidence of co-occurring maternal depression(50) or relationship difficulties(51); significantly, the treatment of these disturbances have been shown to improve the young child's sleep(52). Research on the effects of prenatal treatment of maternal mood disturbance on infant/toddler sleep is needed before the clinical implications of the current results can be fully understood.

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Table 1

Descriptive statistics for demographic and sleep variables.

	Mean	S.D.	%	Sample size
Smoking: yes/any			17.6	10,626
Crowding	1.63	0.64		10,419
Alcohol: > 1 unit/day			1.3	10,360
Maternal education				9962
CSE			13.1	
Vocational			10.0	
O level			37.5	
A level			25.0	
University degree			14.5	
Female sex			48.7	11,011
Maternal age	28.55	4.70		10,336
Maternal prenatal anxiety				
18 weeks	4.80	3.49		9905
32 weeks	5.00	3.55		9901
Maternal prenatal depression				
18 weeks	6.73	4.70		9905
32 weeks	6.85	4.99		10,150
Total sleep				
6 months	12.80	2.13		11,490
18 months	12.78	1.21		11,124
30 months	11.96	1.13		10,323
Number of awakenings				
6 months	0.67	1.34		11,490
18 months	0.81	1.09		11,124
30 months	0.76	1.06		10,323
Sleep problems				
18 months	2.26	1.92		11,124
30 months	3.11	1.99		10,323

TABLE 2

Infant Sleep Measures at 6, 18, and 30 months by Pre- and Post-Natal Maternal Mood

	6 months		18 months			30 months		
	Total Sleep mean (SD)	**# of Awakenings mean (SD)**	**Total Sleep mean (SD)**	**# of Awakenings mean (SD)**	**Sleep Problems mean (SD)**	**Total Sleep mean (SD)**	**# of Awakenings mean (SD)**	**Sleep Problems mean (SD)**
Antenatal maternal mood								
18 week anxiety								
No	12.80 (2.09)	0.66 (1.22)	12.78 (1.19)	0.78 (1.06)*	2.16 (1.87)*	11.96 (1.11)	0.74 (1.00)*	3.02 (1.97)*
Yes	12.75 (2.26)	0.66 (1.28)	12.71 (1.34)	0.93 (1.21)	2.91 (2.10)	11.90 (1.23)	0.88 (1.18)	3.80 (1.99)
18 week depression								
No	12.80 (2.07)	0.66 (1.22)	12.78 (1.19)	0.78 (1.06)*	2.16 (1.87)*	11.95 (1.11)	0.74 (1.00)*	3.03 (1.98)*
Yes	12.79 (2.35)	0.67 (1.29)	12.74 (1.31)	0.94 (1.21)	2.82 (2.08)	11.96 (1.12)	0.88 (1.15)	3.70 (1.98)
32 week anxiety								
No	12.80 (2.07)	0.66 (1.22)	12.78 (1.18)	0.78 (1.06)*	2.16 (1.87)*	11.96 (1.11)	0.74 (1.00)*	3.01 (1.97)*
Yes	12.70 (2.35)	0.72 (1.35)	12.70 (1.32)	0.97 (1.30)	2.81 (2.17)	11.94 (1.23)	0.86 (1.17)	3.76 (2.04)
32 week depression								
No	12.80 (2.08)	0.66 (1.22)	12.78 (1.18)	0.78 (1.07)*	2.13 (1.86)*	11.96 (1.10)	0.74 (0.99)*	3.00 (1.97)*
Yes	12.71 (2.33)	0.71 (1.31)	12.74 (1.31)	0.95 (1.22)	2.84 (2.10)	11.95 (1.23)	0.86 (1.17)	3.75 (2.03)
Postnatal maternal mood								
8 week anxiety								
No	12.81 (2.10)*	0.67 (1.27)	12.78 (1.20)	0.78 (1.07)*	2.19 (1.90)*	11.96 (1.11)*	0.74 (1.01)*	3.05 (1.97)*
Yes	12.54 (2.28)	0.72 (1.33)	12.66 (1.23)	1.09 (1.43)	2.93 (2.05)	11.82 (1.24)	0.88 (1.17)	3.90 (2.01)
8 week depression								
No	12.81 (2.08)*	0.67 (1.25)	12.79 (1.19)*	0.78 (1.08)*	2.19 (1.90)*	11.96 (1.11)*	0.74 (1.00)*	3.03 (1.97)*
Yes	12.60 (2.42)	0.66 (1.22)	12.64 (1.33)	1.09 (1.26)	2.79 (2.04)	11.86 (1.23)	0.85 (1.14)	3.80 (2.04)
8 month anxiety								
No	12.82 (2.09)	0.65 (1.22)*	12.78 (1.20)	0.78 (1.06)*	2.18 (1.88)*	11.96 (1.11)*	0.74 (1.00)*	3.04 (1.98)*
Yes	12.64 (2.33)	0.81 (1.42)	12.67 (1.33)	1.09 (1.40)	2.91 (2.11)	11.82 (1.24)	0.91 (1.14)	3.77 (2.03)
8 month depression								
No	12.83 (2.08)*	0.65 (1.21)*	12.78 (1.19)	0.78 (1.07)*	2.18 (1.88)*	11.96 (1.10)*	0.74 (1.01)*	3.04 (1.98)*
Yes	12.51 (2.37)	0.82 (1.42)	12.69 (1.36)	0.99 (1.30)	2.86 (2.10)	11.84 (1.26)	0.91 (1.13)	3.72 (2.05)

Note: SD indicates standard deviation.

* Mean difference between the depressed/nondepressed or anxious/nonanxious groups is significant at $p < 005$.

Table 3

Predictors of sleep problems at 18 months

	B (S.E.)	OR (95% CI)	Wald
Pregnancy/obstetric risks			
Birthweight for gestational age	− 0.002 (0.002)	1.00 (0.99–1.003)	0.79
Smoking			0.01
Yes/any	− 0.01 (0.08)	0.99 (0.85–1.16)	
Alcohol			0.002
> 1 unit/day	− 0.01 (0.25)	0.97 (0.60–1.62)	
Psychosocial factors			
Crowding	0.04 (0.05)	1.05 (0.95–1.15)	0.85
Educational attainment			44.24
Vocational	− 0.08 (0.11)	0.93 (0.74–1.16)	
O level	− 0.33 (0.09)	0.72 (0.60–0.86)	
A level	− 0.47 (0.10)	0.62 (0.51–0.76)	
Degree	− 0.64 (0.12)	0.53 (0.41–0.66)	
Female	− 0.04 (0.06)	0.96 (0.86–1.07)	0.55
Maternal age	− 0.03 (0.01)	0.97 (0.96–0.99)	18.45
Prenatal mood			
Anxiety			
18 weeks			13.58
Yes	0.39 (0.11)	1.47 (1.20–1.80)	
32 weeks			0.56
Yes	− 0.08 (0.11)	0.92 (0.75–1.14)	
Depression			
18 weeks			0.50
Yes	0.07 (0.10)	1.08 (0.88–1.31)	
32 weeks			13.14
Yes	0.36 (0.10)	1.43 (1.18–1.73)	
Postnatal mood			
Anxiety			
8 weeks			6.77
Yes	0.35 (0.14)	1.43 (1.09–1.86)	
8 months			1.88
Yes	0.18 (0.13)	1.19 (0.93–1.54)	
Depression			
8 weeks			0.03
Yes	0.02 (0.12)	1.02 (0.81–1.29)	
8 months			0.71
Yes	0.11 (0.13)	1.11 (0.87–1.43)	

For educational attainment, minimal education is the control condition. $p < 0.01$, $p < 0.001$; $n = 7458$.

Table 4

Predicting sleep problems at 30 months

	B (S.E.)	OR (95% CI)	Wald
Pregnancy/obstetric risks			
Birthweight for gestational age	− 0.01 (0.002)	1.00 (0.99–1.00)	3.83
Smoking			19.50
Yes/any	− 0.34 (0.08)	1.41 (1.21–1.64)	
Alcohol			0.08
> 1 unit/day	0.07 (0.25)	1.07 (0.66–1.76)	
Psychosocial factors			
Crowding	− 0.10 (0.05)	0.91 (0.83–1.002)	3.67
Educational attainment			26.19
Vocational	0.16 (0.12)	1.18 (0.93–1.48)	
O level	− 0.17 (0.09)	0.84 (0.70–1.02)	
A level	− 0.26 (0.10)	0.78 (0.64–0.95)	
Degree	− 0.38 (0.12)	0.69 (0.54–0.86)	
Female	− 0.11 (0.06)	0.90 (0.81–1.01)	3.49
Maternal age	− 0.02 (0.01)	0.98 (0.97–0.99)	11.12
Prenatal mood			
Anxiety			
18 weeks			8.79
Yes	0.33 (0.11)	1.39 (1.20–1.80)	
32 weeks			0.05
Yes	0.03 (0.11)	1.03 (0.83–1.27)	
Depression			
18 weeks			0.004
Yes	0.01 (0.11)	1.01 (0.81–1.25)	
32 weeks			12.57
Yes	0.36 (0.10)	1.43 (1.18–1.76)	
Postnatal mood			
Anxiety			
8 weeks			3.60
Yes	0.26 (0.14)	1.30 (0.99–1.71)	
8 months			1.79
Yes	0.18 (0.13)	1.20 (0.92–1.56)	
Depression			
8 weeks			2.64
Yes	0.20 (0.12)	1.22 (0.96–1.54)	
8 months			0.39
Yes	0.08 (0.13)	1.09 (0.84–1.41)	

For educational attainment, minimal education is the control condition. $p < 0.05$, $p < 0.01$, $p < 0.001$; $n = 6829$.