Research report

Maternal depression and psychiatric outcomes in adolescent offspring: A 13-year longitudinal study

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Abstract

Background: Maternal postnatal depression (PND) has been associated with adverse outcomes in young children, but an association with longer-term psychiatric disorder has not been demonstrated. We present the preliminary findings of a 13-year longitudinal study.
Methods: In the course of a prospective longitudinal study, we examined DSM-IV Axis I disorders in 13-year-old adolescents who had (*n*=53) or had not (*n*=41) been exposed to maternal PND. We also detailed the occurrence of depression in mothers throughout the 13-year follow-up period.
Results: Maternal PND was associated with higher rates of affective disorders in adolescent offspring. However, mothers who developed PND were also substantially more likely than those who did not to experience depression subsequently, a fact that contributed to the development of depressive disorder in offspring. Maternal PND was associated with increased risk for depression in adolescent offspring *only* if there had also been later episodes of maternal depression. In contrast, anxiety disorders in offspring were elevated in the maternal PND group regardless of the occurrence of subsequent maternal depression.
Limitations: Due to the modest sample size and consequently limited power, findings must be regarded as preliminary.
Conclusions: The particular association between early maternal depression and anxiety disorders in offspring was consistent with theories that emphasise the primacy of early environmental exposures. This position was not supported with respect to offspring depressive disorder, where overall duration of maternal depression was a significant factor. PND was associated with recurrent episodes of depression in the majority of cases, underlining the need for monitoring of this population beyond the postnatal period.
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1. Introduction

Maternal depression in the postnatal period has been associated with wide-ranging and persistent impairments in child functioning (Field, 1998, 1995; Hay et al., 2001; Murray and Cooper, 2003). In infancy, cognitive, neurological and regulatory disturbances have been observed (Field, 1995; Murray, 1992). In childhood, there appears

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to be a continuity of cognitive impairments, at least in the presence of other environmental risk factors (Hay et al., 2001; National Inst of Child Health and Human Development, 1999), and an emergence of risk factors for depressive disorder, including socioemotional disturbances (Essex et al., 2001; Murray et al., 1999) and depressogenic cognitions (Goodman and Gotlib, 2001; Murray et al., 2001). In addition, the presence of early maternal depression has been associated with alterations in cortisol secretion in children (Ashman et al., 2002; Essex et al., 2002) and adolescents (Halligan et al., 2004) that are consistent with biological risk for depression. Despite this apparent expression of risk for depression, the focus of research to date has been on outcomes in childhood, when depressive disorder is rare. Consequently, the emergence of offspring depression has not been explicitly examined in relation to maternal PND.

A number of processes may contribute to the observed links between PND and adverse child outcomes (Goodman and Gotlib, 1999, 2001; Murray and Cooper, 2003). Animal research has shown sustained biological and behavioural disturbances in offspring in association with disruptions in the early maternal environment, and has emphasised the primacy of environmental insults to the organism while development is ongoing (e.g., Anisman et al., 1998; Suomi, 1997; Weaver et al., 2004). As maternal PND is associated with disturbances in the provision of early care (Field, 1984; Murray et al., 1996), a pre-programming hypothesis may explain the transmission of disorder from mother to offspring; this holds that, as the environmental disturbances associated with maternal PND occur while the infant is maturing, they may shape developing cognitive and biological systems, with sustained consequences.

More recently, studies of early environmental factors have additionally identified a role for antenatal exposure; for example, maternal anxiety during pregnancy has been associated with biological and emotional disturbances in children, even after accounting for postnatal maternal disorder (Glover et al., 2004; O'Connor et al., 2002, 2003). The cause of such effects has yet to be explained, but likely mechanisms include foetal exposure to maternal stress hormones and placental insufficiency (Field et al., 2005; Gitau et al., 1998; Glover and O'Connor, 2002; O'Connor et al., 2005). As a significant proportion of cases of PND represent a continuation of antenatal disorder (Heron et al., 2004), antenatal factors are likely to contribute to any association between maternal PND and offspring disorder. Accounts that highlight the importance of the early postnatal environment and those that have emphasised antenatal exposure both hold that early maternal disorder is a more significant factor than later exposures in the development of offspring disorder.

There are other factors which have been implicated in the transmission of depression from mother to offspring which do not attach particular significance to experiences early in development. The broader literature on maternal depression has indicated associations with both family disturbances and wider environmental adversity (Cummings and Davies, 1994; Goodman and Gotlib, 1999); to the extent that PND is associated with higher rates of subsequent depression in mothers (Cooper and Murray, 1995; Wisner et al., 2002), any association between maternal and child disorder may be a function of ongoing environmental disturbances. Transmission of risk for disorder also operates via genetic factors and heritability has been estimated at approximately 37% for depressive disorder (Sullivan et al., 2000). Although recurrence of depression appears to be an indicator of higher genetic liability, genetic studies have generally not considered the timing of parental depression to be of particular significance.

The current research examines psychiatric outcomes in adolescence in relation to maternal PND; we present data on a prospective longitudinal sample of mothers and their children who have been studied from the early postnatal period. Previous research with the current sample has shown cognitive, emotional and biological disturbances in the offspring consistent with risk for depression (Murray and Cooper, 2003); observations that these disturbances are particularly related to maternal PND, rather than to maternal depression occurring later in development or to overall exposure to maternal depression, have broadly supported a role for environmental pre-programming in the intergenerational transmission of risk for disorder. If this position is correct, then early life exposure to maternal depression should also be associated with higher rates of psychiatric disorder than maternal depression occurring at other times.

The question of the impact of timing of exposure to maternal depression on offspring vulnerability to disorder is complicated to address in practical terms; as already noted, individuals exposed to maternal depression early in development are also likely to be exposed to subsequent episodes of maternal depression. The careful documentation of the occurrence of maternal depression and offspring disorder over an extended period (13 years) in our longitudinal sample provides a significant opportunity, despite the modest sample size and consequently limited power. As such, we have conducted a preliminary exploration of the impact of postnatal versus later occurring maternal depression on psychopathology in adolescent offspring, while also taking account of possible

confounding factors, including the occurrence of negative life events and marital conflict, and adolescent pubertal status. We also report data on the overall rates of depressive disorder in mothers during the course of this 13-year study.

2. Methods

Participants were part of a prospective longitudinal study of the development of children of postnatally depressed and well women (Murray, 1992). The sample was originally recruited at two months postpartum, with further assessments made when the child was 18 months, 5 and 8 years old, in addition to the current, 13-year follow-up. Initial recruitment was through screening a community sample ($N = 702$) of primiparous mothers of healthy, full-term infants at 6 weeks postpartum using the Edinburgh Postnatal Depression Scale (EPDS) (Cox et al., 1987). Women scoring over 12 on the EPDS were interviewed; 61 women who met Research Diagnostic Criteria (Spitzer et al., 1978) for depressive disorder were identified, 58 of whom were recruited for the study. Forty-two mothers without PND were randomly selected from the same postnatal population as a comparison group. Fifty-three (91.4%) PND group and 41 (97.6%) comparison group families were retained at 13 years. Informed consent was obtained prior to participation in this ethically approved study.

2.1. Maternal measures

2.1.1. Mental state

Maternal depression was assessed using the Structured Clinical Interview for DSM-IV (Spitzer et al., 1995). As clinical interviews were conducted at all assessments, detailed longitudinal information relating to maternal mental state was available. This information was used to establish the total number of months that mothers were depressed through the course of the study and, hence, total offspring exposure to maternal depression; and it was also used to identify a group of children with late exposure, defined as maternal depression occurring after the child was 5 years of age. As the developing brain reaches 90% of its adult size by 5 years, with relatively slow growth thereafter (Giedd et al., 1996), researchers have frequently considered this an appropriate cut-off for studies examining the timing of developmental influences.

2.1.2. Parental conflict

In addition to supplying basic information about their marital status, mothers completed 10-point rating scales on perceived and felt criticism as a measure of parental conflict (Hooley and Teasdale, 1989).[1]

2.2. Adolescent measures

2.2.1. Mental state

Diagnostic interviews were conducted at 8 and 13 years using the affective and behavioural disorder modules of the Kiddie Schedule for Affective Disorders and Schizophrenia (Kaufman et al., 1997). At 8 years, only current disorder was assessed as we believed that children would not be sufficiently reliable in reporting past difficulties. However, at 13 years, we asked about problems occurring since the previous interview. Thus, the overall period of assessment of offspring diagnoses was 8–13 years.

All diagnostic interviews were carried out by a trained, highly experienced clinician who was blind to maternal PND status. All interviews were also reviewed by a clinically experienced team to ensure that the best possible standards of diagnosis were maintained. While mothers were asked to confirm child reports at 8 and 13 years, the emphasis was placed on information derived directly from the child in order to minimize the potential for contamination of child diagnoses by maternal symptoms.

2.2.2. Puberty

Tanner stage (Tanner, 1966) was obtained using adolescent self-report guided by standard line drawings (Netherton et al., 2004).

2.2.3. Life events

Adolescents were administered the Life Events Schedule (LES; Goodyer et al., 2000), a valid and reliable interview assessment of the occurrence of a range of undesirable life events and difficulties. Mothers also completed a questionnaire measure of significant loss events experienced by offspring over the course of their life (EXITS questionnaire; Goodyer and Altham, 1991a,b), with endorsement of an event being followed by a rating of associated distress. In combination, the LES and EXITS provide a detailed assessment of life events in the past 12 months, as well as a broad assessment of significant events over the life course.

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[1] Mothers also completed the Dyadic Adjustment Scale (Spanier, 1979), but this proved problematic for those who were separated from their partner as many items did not apply or had a different meaning. Thus, we employed the marital criticism ratings as an alternative index of parental conflict, as these applied to separated and non-separated couples alike; criticism scores correlated $r = -0.56$ with satisfaction scores on the Dyadic Adjustment Scale.

3. Results

Sample characteristics are presented in Table 1. The sample comprised white, primarily middle class, families, consistent with the Cambridge UK population from which it was drawn. The adolescents in the PND and the comparison group were similar in terms of background characteristics, including age, pubertal development (Tanner stage), gender distribution, socioeconomic status and parental marital status. Comparable numbers of adverse life events were reported in each group (see Table 1). However, the groups did differ in terms of other concurrent stressors; a higher proportion of adolescents in the PND group had been exposed to a recent episode of maternal depression, and there was a trend for mothers in the PND group to report more parental conflict than comparison group mothers (see Table 1).

Diagnostic outcomes are reported in Table 2. Logistic regression indicated that, compared to adolescents of comparison group mothers, significantly more adolescents in the PND group had experienced psychiatric disorder by 13 years (odds ratio (OR)=2.75, 95% CI=1.07–7.05; Wald=4.43, $df=1$, $P=0.035$).

Depressive disorder was just beginning to emerge in our sample at 13 years; the earliest onset of depression

Table 1
Sample characteristics

	No PND $n=41$	PND $N=53$	Statistics
Maternal characteristics			
Social classes I, II and III non-manual	65.9%	63.6%	$\chi^2(1)=0.05$
Percent separated from child's father	12.2%	22.6%	$\chi^2(1)=1.70$
Percent reporting recent depression	2.4%	22.6%	$\chi^2(1)=$ 7.92**
Total study months depressed (months: S.D.)[a]	2.76 (4.43)	21.51 (16.2)	$t(61.8)=$ −8.04***
Reported parental conflict (mean 0–10: S.D.)	4.45 (2.32)	5.35 (2.26)	$t(90)=$ −1.88*
Child characteristics			
Proportion of boys	51.2%	47.3%	$\chi^2(1)=0.15$
Age at assessment (months: S.D.)	160.8 (3.8)	159.9 (1.7)	$t(92)=1.46$
Tanner stage (mean 1–5: S.D.)	3.3 (0.9)	3.3 (0.6)	Z $(N=91)=$ −0.33
Number of adverse life events (mean: S.D.)[a]	2.3 (1.5)	2.2 (1.7)	Z $(N=94)=$ −0.43
Number of loss events (mean: S.D.)[b]	1.3 (1.1)	1.3 (1.2)	Z $(N=94)=$ −0.08

[+]$P<0.10$, **$P<0.01$, ***$P<.001$; PND: maternal postnatal depression.

[a] Measured using the Life Events Schedule.
[b] Measured using the EXITS Questionnaire.

Table 2
Prevalence of psychiatric diagnoses (current or past) reported by postnatal depression versus comparison group

	Comparison group, $n=41$	PND group, $n=55$
	n (%)	n (%)
Any Axis I diagnosis	8 (19.5)	22 (40.0)
Male	3 (14.3)	9 (34.6)
Female	5 (25.0)	13 (44.8)
Anxiety disorder	4 (9.8)	15 (27.8)
Male	1 (4.8)	5 (20.0)
Female	3 (15.0)	10 (34.5)
Depressive disorder	3 (7.3)	12 (22.6)
Male	0 (0)	3 (12.0)
Female	3 (15.0)	9 (32.1)
Behavioural disorder	2 (4.9)	4 (7.4)
Male	2 (9.5)	3 (11.5)
Female	0 (0)	1 (3.6)

PND: postnatal depression.

was 11 years of age, and the mean age of onset for those who had experienced a depressive episode was 12 years 8 months. The prediction of lifetime depressive disorder in adolescents by the presence or absence of maternal PND was examined using logistic regression, with gender as a second predictor due to higher rates of depressive disorder in girls compared to boys (see Table 2). Results indicated a trend for an association between maternal PND and adolescent depression: more than three times the rate of depressive disorder was observed in adolescents who had been exposed to maternal PND than in those who had not been exposed (OR=3.78, 95% CI=0.96–14.9; Wald=3.60, $df=1$, $P=0.058$). There was also a significant gender effect (OR=4.85, 95% CI=1.24–19.0; Wald=5.16, $df=1$, $P=0.023$).

We repeated analyses of depressive disorder in offspring controlling for other likely contributors to depression, namely exposure to adverse life events and the occurrence of parental conflict (rating of mutual criticism).[2] As our two measures of adverse life events (EXITS and LES) overlapped in the events that they examined, two separate regressions were run including each of these measures in turn; only EXITS scores proved to be associated with adolescent depression, therefore this analysis is presented.[3] The results indicated that, after taking account of the presence of loss events (OR=2.09, 95% CI=1.02–4.25; Wald=4.11, $df=1$, $P=0.043$),

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[2] We chose to examine parental conflict versus divorce as prior research has suggested that the former may be more pertinent to offspring psychological adjustment. However, the current findings were not altered by controlling for parental divorce versus reported conflict.

[3] The findings with respect to maternal PND were essentially the same whether the LES, the EXITS questionnaire or both measures were included in the regression.

maternal partner conflict (OR = 0.98, 95% CI = 0.76–1.28; Wald = 0.01, df = 1, ns) and gender (OR = 5.49, 95% CI = 1.24–24.3; Wald = 5.03, df = 1, P = 0.025), maternal PND remained a marginally significant predictor of offspring depressive disorder (OR = 3.86, 95% CI = 0.90–16.5; Wald = 3.30, df = 1, P = 0.069). We also examined whether adolescent life stress interacted with the presence of maternal PND to trigger depression in vulnerable individuals. This did not appear to be the case; when the above regression analysis was repeated, adding the interaction between PND and loss events or parental conflict, the interaction terms did not improve the fit of the model and were not significant predictors of adolescent depression (P's > 0.40).

Logistic regression also indicated a higher rate of anxiety disorders in the adolescent offspring of mothers who had had PND relative to comparison group adolescents (OR = 3.56, 95% CI = 1.08–11.71; Wald = 4.36, df = 1, P = 0.037) (see Table 2). The anxiety disorders detected were specific phobias (n = 14), social phobia (n = 1), separation anxiety disorder (n = 2), obsessive compulsive disorder (n = 4) and generalized anxiety disorder (n = 3). There was no significant association between PND exposure and the presence of behavioural disorders (OR = 1.56, 95% CI = 0.27–8.96; Wald = 0.62, df = 1, ns); however, the actual frequency of behavioural disorders in the current sample was very low (6 cases by 13 years, see Table 2).

3.1. Ongoing maternal depression

Mothers who became depressed in the postnatal period were also more likely to have subsequent episodes of depression than mothers who were not depressed postnatally. Over the 13 years of study, the median number of depressive episodes for the comparison group was 0 (range 0–3), as compared to a median of 3 in the PND group (range 1–11). Indeed, 83.6% of mothers in the PND group reported a further episode of depressive disorder subsequent to their postnatal episode. Thus, even excluding the depression occurring in the postnatal period, PND group mothers spent significantly more study months depressed than comparison group mothers, a mean of 16.2 months (S.D. = 15.9, range 0–66 months) versus 2.8 months (S.D. = 4.4 months, range 0–16 months).

Logistic regression was used to examine the contribution of total number of study months of maternal depression ("total maternal depression") to depressive disorder in PND group offspring. Total maternal depression significantly predicted the occurrence of depression in PND group adolescents (OR = 1.05, 95% CI = 1.01–1.10; Wald = 6.10, df = 1, P = 0.014); those who became

depressed had mothers with a mean of 32.6 months (S.D. = 21.5) of depression through the course of the study (including the postnatal episode), compared to 18.2 months (S.D. = 12.9) in non-depressed adolescents. In the light of this positive association, we further examined the impact of "total maternal depression" on our previously reported PND effect. Binary logistic regression indicated that the effect of maternal PND on adolescent depression (OR = 3.71) reported earlier was effectively eliminated by the inclusion of total months of maternal depression in the regression model (OR = 1.26, 95% CI = 0.24–6.62; Wald = 0.08, df = 1, ns), while the effect of total months of maternal depression was itself retained (OR = 1.05, 95% CI = 1.01–1.10; Wald = 5.89, df = 1, P = 0.015). In contrast, total maternal depression did not predict of the occurrence of anxiety disorders in PND group adolescents (OR = 0.99, 95% CI = 0.96–1.04; Wald = 0.002, df = 1, ns).

The above analyses indicate a significant cumulative impact of maternal depression on offspring depressive disorder; however, the extent to which maternal PND is confounded with the subsequent occurrence of maternal depression in the current sample makes the interpretation of these observations difficult. In an attempt to further separate the effects of early versus later maternal depression, we conducted exploratory analyses specifically investigating the effects of "late" maternal depression (i.e. occurring after the child was aged 5 years). Thus, we examined whether PND contributed to offspring disorder once late maternal depression was accounted for by dividing the sample into four groups:
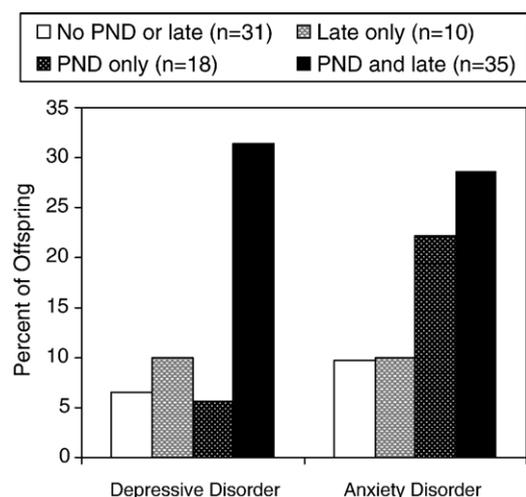

Fig. 1. Rates of depressive and anxiety disorders in offspring measured from 8 to 13 years reported according to the presence or absence of postnatal depression and/or "late" depression in mothers. PND: maternal postnatal depression.

neither PND nor late maternal depression (n=31), late depression without PND (n=10), PND without late depression (n=18) and PND with late depression (n=35). Logistic regression was used to examine the prediction of offspring depressive disorder by the dummy coded maternal depression category variable, with gender as a second predictor. The four-group categorical variable was a significant predictor of offspring depression (Wald=7.82, df=3, P=0.050), as was gender (Wald=7.80, df=1, P=0.032). Indicator contrasts compared each of the three maternal depression groups to the comparison group (i.e. no PND or late depression); the results demonstrated that the presence of *both* PND and late maternal depression was associated with significantly elevated rates of offspring depression (contrast Wald=4.86, df=1, P=0.027), but the presence of either PND alone or late depression alone was not (P's>0.82; see Fig. 1). By contrast, logistic regression did not reveal the four-group maternal variable to be a significant predictor of offspring anxiety disorder (Wald=4.45, df=3, ns). As illustrated in Fig. 1, the occurrence of offspring anxiety was elevated in association with maternal PND regardless of the presence or absence of late maternal depression.[4]

4. Discussion

We found that adolescents exposed to maternal PND show elevated rates of affective disorder by 13 years of age; to our knowledge, this is the first longitudinal study to specifically examine the association between maternal PND and adolescent psychopathology. However, mothers who developed PND also experienced more depression subsequent to the postnatal period than mothers who were not depressed postnatally; and when this subsequently occurring maternal depression was taken into account, specific effects of maternal depression in the postnatal period on adolescent depression were not observed. In contrast, for adolescent anxiety disorder, there appeared to be a specific risk associated with postnatal maternal depression.

The fact that there was no association between PND and increased risk for depression in offspring in the absence of later maternal depression argues against the primacy of disturbances that occur early in develop-

ment, at least for the intergenerational transmission of risk for depression. Our results are similarly problematic for accounts that emphasize the significance of possible intrauterine disturbances and those that focus on early postnatal effects, neither position being consistent with a cumulative effect of maternal depression. An exploratory analysis examining the presence of maternal PND, late depression or both, suggested that early exposure may make offspring particularly vulnerable to later exposure; larger samples, in which total exposure to maternal depression is controlled across groups, are required to test this possibility. Nevertheless, our data are inconsistent with the hypothesis that early life exposure alone contributes significantly to the transmission of depressive disorder from mother to offspring.

Our observations support the findings of Hammen and Brennan (2003), whose cohort study indicated that severity and chronicity of maternal depression, but not timing, were significant determinants of risk for depression in offspring. As severity and recurrence of depressive disorder are associated with a strong genetic component (Kendler et al., 1993, 2001), it is likely that genetic factors contributed to the association observed in the current study between depression in adolescent offspring and prolonged maternal depression. An ongoing environmental effect is also possible; offspring may directly model the manifest cognitive and behavioural patterns of their parent, or maternal depression may contribute to, or else reflect, generalized environmental adversity (Kim-Cohen et al., 2005). Earlier research also suggests a further consideration; namely, that the association between maternal and offspring depression may be bidirectional, with offspring disorder exacerbating maternal symptoms as well as the converse (Elgar et al., 2003, 2004). While a bidirectional effect could explain the association between late maternal depression and offspring depressive disorder, the fact that anxiety disorders in offspring were not similarly associated with late maternal depression in the current study is problematic for such an account.

The association between ongoing exposure to maternal depression and depression in offspring is of interest in the light of earlier findings from the current sample. In particular, a number of child outcomes that are consistent with risk for depression have been found to be associated with maternal PND and *have not* been accounted for by the cumulative effects of maternal depression; these include elevated basal cortisol (Halligan et al., 2004), socio-emotional disturbances (Murray et al., 1999, 2006) and a negative cognitive style (Murray et al., 2001). This divergence in the factors that are associated with, on the one hand, the presumed

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[4] The use of cut-off scores can be problematic as results may vary depending on the point selected. As such, we verified that our findings were also replicated using cut-points on either side of the 5-year cut-off selected. Furthermore, our exploratory categorical analyses tend to confirm the conclusions indicated by the equivalent analyses using the continuous variable of total study months of maternal depression as a predictor.

expression of vulnerability for disorder and, on the other, the actual development of depression remains to be explained. Possible accounts include previously observed, relatively subtle disturbances being non-pathological, the presence of an interaction between early-expressed risk and ongoing exposure to maternal depression, or the emergence of an overriding genetic vulnerability.

Our observation of an association between maternal PND and anxiety disorders in offspring is broadly consistent with earlier research, which has indicated elevated rates of anxiety disorders in association with parental depression as it occurs more generally (Nomura et al., 2002). However, as noted above, our findings were also consistent with early life exposure being of particular significance for the development of anxiety in offspring, with later maternal depression not having a significant impact; thus, in this case, a pre-programming model is broadly supported. Child development may be affected by both prenatal maternal disorder, presumably via impact on the intrauterine environment, and postnatal depression, which impacts on the quality of early mother–infant interactions. In the absence of data relating to the presence of prenatal disorder in our sample, we cannot distinguish between these two possibilities: as pre- and postnatal depression frequently co-occur, an influence of both seems likely.

Substantial comorbidity exists between anxiety and depression, and certain anxiety disorders (particularly GAD) have a shared genetic liability with depression (Hettema et al., 2001); the prepubescent onset of anxiety diagnoses may predispose to the emergence of depressive symptoms later in development (Silberg et al., 2001). In our sample, 60% of adolescents who became depressed had also been diagnosed with an anxiety disorder, compared with only 11.4% of non-depressed adolescents. Our observation that these disorders show somewhat different associations with maternal depression is perhaps surprising in the light of this co-morbidity. Prior research has suggested that shared genetic liability may be shaped by different environmental influences to produce anxiety (specifically GAD) versus depressive disorders (Kendler et al., 1992). However, other explanations may also account for our divergent findings. Anxiety disorders tend to emerge at a younger age than depression, as was the case in the current study; anxiety disorders were already prevalent at the 8-year diagnostic interview, while depressive disorders were only evident at 11 years or later. As such, it is possible that our observation of a direct association between maternal PND and offspring anxiety disorders, but not offspring depression, is due to

a diminishing influence of early life exposures as the child becomes older. Although exploratory analyses[5] did not suggest age-related variation in the strength of the association between maternal PND and offspring rates of anxiety disorders, further examination of this issue is warranted.

The significant effect of ongoing maternal depression on risk for depression, but not anxiety, in offspring may also be explained by the developmental progression from anxiety to depression requiring additional liability. Thus, in their review of childhood internalising disorders, Kovacs and Devlin (1998) suggest that anxiety and depression are characterised by similar basic underlying pathologies, such as poor emotional regulation, but that the chronological progression from anxiety to depression requires the activation of further biological or cognitive vulnerabilities. Furthermore, anxiety disorders themselves appear to show a degree of developmental progression and, in the current sample, those pathologies that have typically been more closely linked to depression (such as GAD) were only just becoming apparent by 13 years. Limitations of power dictated that we examine anxiety disorders as a group in the current study; however, the separate consideration of individual disorders could potentially have yielded instances where the pattern of results follows that observed for depression.

We found no association between the occurrence of behavioural disorders and the presence of maternal PND, a surprising result given that, in the current sample, exposure to maternal depression predicted higher levels of externalizing symptoms at 5 years and 8 years (Morrell and Murray, 2003). Indeed, numerous earlier studies have highlighted an association between maternal depression and behavioural problems in offspring, which appears to be attributable both to the quality of environment associated with maternal depression and to comorbidity between depression and antisocial behaviours in the mother (Kim-Cohen et al., 2005). The current null findings may simply reflect a lack of power; we note that rates were slightly higher in PND versus control group adolescents (7.3% versus 4.9%), albeit with very small overall rates of disorder (6 cases in total). Our results may also be explained by characteristics of the sample, which was primarily middle class and lacking in gross environmental or familial adversity. An absence of generalized environmental adversity may have protected against the emergence of overt behavioural disorder.

One of the most striking observations in this prospective longitudinal study is the rate at which women who

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[5] Results not reported in the manuscript due to small cell sizes.

develop postnatal depression are subject to the recurrence of depressive disorder. By the time adolescents were 13 years of age, 84% of postnatally depressed women in the current sample had reported subsequent episodes of depressive disorder. Thus, our findings reinforce the prevailing view that postnatal depression should not be afforded special diagnostic status, but rather primarily occurs in women who are vulnerable to depression (Cooper et al., 1988). Furthermore, our results highlight the fact that although it is of theoretical interest to consider whether early exposure to maternal depression is of particular significance for child development, in practice such early exposure usually occurs in the context of more persistent maternal difficulties.

The current results must be viewed as preliminary in the light of several limitations. First, the high rate of recurrence of maternal depression in the PND group, as discussed above, limited our ability to address the issue of timing of maternal disorder. Second, the small sample size meant that we had limited power to detect differences in rates of disorder in offspring; consequently any null findings presented in the current study must be viewed with caution. Third, several possible confounding factors were not examined in the current research. In particular, we did not measure either maternal prenatal depression or anxiety, or rates of paternal disorder, both of which are likely to have occurred more frequently in the PND group (Goodman, 2004; Heron et al., 2004). As both maternal prenatal disorder and paternal postnatal depression have been linked to child disorder (Brennan et al., 2002; Nomura et al., 2001; O'Connor et al., 2003; Ramchandani et al., 2005), they may have contributed to the association between maternal PND and offspring disorder that we observed. In addition, we did not examine the impact of comorbid maternal diagnoses in the current study; there were 14 cases of maternal anxiety disorder in the course of the study in this community sample, and one mother was diagnosed with an eating disorder. Thus, although the presence of comorbid disorders may have contributed to psychopathology in offspring, this issue could not be addressed in the current sample where the predominant psychopathology in mothers was depressive disorder.

Finally, the impact of treatment seeking on maternal and offspring outcomes was not examined in the current study; relatively low rates of treatment seeking combined with variability in the type and timing of treatment preclude a meaningful analysis of treatment effects. Nevertheless, the effect of maternal treatment intervention is of interest. Assuming that transmission is not wholly genetic, our association between total months of maternal depression and rates of depressive disorder in offspring suggests that successful intervention for maternal disorder has the potential to have a positive effect on offspring outcomes. Few studies to date have examined this issue. We have previously studied the impact of treatment for maternal depression in the postnatal period on outcomes in infancy; although a significant acceleration in the rate of recovery was achieved with psychological intervention, associated improvements in infant outcomes were limited and were not sustained through to childhood (Cooper et al., 2003; Murray et al., 2003). Weissman et al. (2006) examined the impact of pharmacological intervention with depressed mothers of older children and adolescents, and reported significantly lower rates of psychopathology in offspring in cases where maternal disorder remitted within 3 months of treatment onset compared to cases where remission did not occur within 3 months. Overall, there is support for a policy of aggressive treatment of maternal depression, but also for longer term monitoring for the recurrence of maternal disorder and repeated intervention.

5. Conclusion

Despite low actual frequencies of disorder in our sample of 13 year olds, the current findings of elevated rates of affective disorders in association with maternal PND are striking. Further study of the current sample as the adolescents move into adulthood may prove illuminating; not only are rates of depressive disorder likely to increase, but risk for disorder may also be more purely expressed as the proximal environmental impact of ongoing maternal disturbance begins to diminish. The current findings did not support the proposition that early environmental exposure alone is sufficient to raise the risk for adolescent depression: depressive disorder in adolescents was more significantly related to the chronicity or recurrence of maternal depression. In contrast, anxiety disorders were more prevalent in association with maternal PND regardless of subsequent maternal depression. Our research highlights the fact that, regardless of the underlying mechanisms, maternal depression in the postnatal period is associated with ongoing maternal difficulties and the development of child disorder. As such, the presence of PND could identify for possible long-term monitoring and clinical intervention a subgroup of mothers and children who are at risk by virtue of ongoing maternal vulnerability to depression.

Acknowledgements

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