

## Course and Recurrence of Postnatal Depression Evidence for the Specificity of the Diagnostic Concept

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**Background.** It is unclear whether the causative factors of non-psychotic postnatal depression are the same as those of depression at other times.

**Method.** The course and recurrence of postnatal depression was studied in two groups of primiparous women experiencing an index episode of postnatal depression: those for whom the mood disorder had arisen *de novo* ( $n = 34$ ), and those for whom it was a recurrence of previous affective disturbance ( $n = 21$ ). The mental state of these two groups, together with a psychiatrically well control group ( $n = 40$ ), was studied for five years.

**Results.** It was found that those for whom the index episode was a recurrence of depression were at raised risk of further non-postpartum episodes but not postpartum episodes, and that those for whom the index episode had arisen *de novo* were at raised risk for further episodes of postnatal depression but not for non-postpartum episodes.

**Conclusion.** These findings suggest a specific nosologic reference for the concept of postnatal depression.

A number of epidemiologic studies have been conducted of the nature, prevalence, and course of non-psychotic postnatal depression (Kumar & Robson, 1984; Watson *et al*, 1984; Nott, 1987; Cooper *et al*, 1988; Carothers & Murray, 1990; O'Hara *et al*, 1990; Cox *et al*, 1993). It has consistently been found that around 10% of women experience an episode of depression in the first weeks after delivery, that the symptom profile of these episodes is the same as that of depressive disorders occurring at other times, and, generally, that the great majority of these depressions resolve spontaneously within three to six months (Cooper *et al*, 1991). The causative factors which have emerged from these studies as important are the same as those found to be associated with the onset of depression at other times, for example marital discord, previous history of depression, and the occurrence of adverse life events (Cooper *et al*, 1991).

In view of the absence of distinctive features, the nosologic status of postnatal depression has been questioned (Watson *et al*, 1984; Cooper & Stein, 1989; Whiffen, 1992). Recently, however, it has been argued that this scepticism might have arisen because account has not been taken in previous research of the possibility that there are two populations of women who become depressed postnatally: one for whom the depression is specifically related to the emotional demands of motherhood, and one for whom the birth is either unrelated to the depression or is a non-specific stressor (Murray, 1989). If there were, indeed, two such populations, one of these might well have distinctive features which

would have been obscured by the failure of previous work to draw the proper distinctions. This argument has never been empirically tested. One way to do so is to examine the recurrence of postpartum depression: if the postpartum period does represent a specific vulnerability for certain women, one would expect such women, having had one postpartum depression, to be at raised risk of the recurrence of depression after a subsequent delivery. Previous studies have found that women who experience an episode of postnatal depression are at risk of later depression (Uddenberg & Engleson, 1978; Wolkind *et al*, 1980; Philipps & O'Hara, 1991). However, to date no study has investigated whether these women are at particular risk of recurrent postpartum depressive episodes; nor has any study distinguished different kinds of postpartum depressive disorder.

The present study investigated whether there are two distinct populations of women experiencing depression postnatally by examining the course and recurrence of postpartum depression. Two groups of primiparous depressed women were studied: those for whom the postpartum episode constituted a recurrence of previous depression, and those for whom the postpartum episode arose *de novo*. It can be hypothesised that, as compared with those with a previous history of depression, in those for whom the postpartum episode was a first onset, the depression was more likely to have arisen as a specific response to childbirth, and, therefore, that such women would be at particular risk of depression after a subsequent delivery.

### Method

Women presenting on the postnatal wards of the Cambridge maternity hospital during the period February 1986 to February 1988 were invited to participate in a study of the experience of motherhood and infant development (Murray, 1992). They were included if they met the following criteria: primiparous, aged 20–40 years, married or cohabiting, having had a 37–42 week pregnancy, intending to be the infant's primary carer, and intending to be resident in Cambridge for the next 18 months. In addition, the infant was required to have a birth weight of at least 2.5 kg, to show no gross congenital abnormality, and not to have required admission to the special-care baby unit. A total of 702 women fulfilled these criteria, only nine (1.3%) of whom refused to take part. Basic demographic information, method of delivery, sex, gestation, and birth weight of the infant were recorded for all 702 women, along with details of any previous psychiatric history.

At six weeks postpartum, the mothers were sent the Edinburgh Postnatal Depression Scale (EPDS) (Cox *et al.*, 1987). A total of 674 (97%) returned the questionnaire, but 28 expressed inability or unwillingness to continue in the study. Of the remaining 646 (92% of the original sample), all those with an EPDS score of at least 13 were interviewed at 2–3 months postpartum by either a psychiatrist or a psychologist using the Standardised Psychiatric Interview (SPI) (Goldberg *et al.*, 1970), to identify minor definite and major (probable and definite) episodes of depression (according to the Research Diagnostic Criteria (RDC) of Spitzer *et al.*, 1978). Additional items on weight loss and appetite were incorporated into the SPI for conformity with the RDC. Finally, after every alternate index case was identified, and then, for the last six months of recruitment, after every case, a potential control was selected randomly from those who had a low EPDS score, no previous history of depression, and the same sex of infant as the index mother. These women were also interviewed with the SPI and the SADS-L (Endicott & Spitzer, 1978) to ensure that there had been no depression before or since delivery.

These procedures yielded 40 women in whom postnatal depression arose *de novo*, 21 women in whom the postnatal depression was a recurrence of previous affective disturbance, and a psychiatrically well control group of 42. Three women with *de novo* onsets were either unable or unwilling to participate in the study, and a further five women (three from the *de novo* onset group and two from the control group) were lost to follow-up over the subsequent five years. This left a final sample of 34 women

for whom the index episode had arisen *de novo*, 21 for whom it was a recurrence, and 40 controls.

In the course of assessing the effect of postnatal depression on child outcome, the mothers and children were assessed when the children were aged 18 months and five years. At each of these points, detailed mental state assessments were made with the SADS-L.

### Results

The control group and the two depression groups did not differ in age, social class, maternal education, and marital status. The two depression groups did not differ in the severity of the mood disturbance itself at the initial two-month postpartum assessment.

#### Initial course

The histogram in Fig. 1 shows the duration of the episode of postnatal depression for the two groups of women identified as depressed postnatally (up to 18 months postpartum). It can be seen that those for whom the postnatal depression constituted a *de novo* onset had a significantly shorter episode of mood disturbance (Mann-Whitney '*U*' = 211;  $P < 0.005$ ) than those for whom the postpartum disorder was not the first episode. One woman from each of the two depression groups remained depressed for the entire 18-month postpartum period.

During the first 18 months after delivery, four (i.e. 9.5%) of those among the control group experienced an episode of depression (RDC major or definite minor).

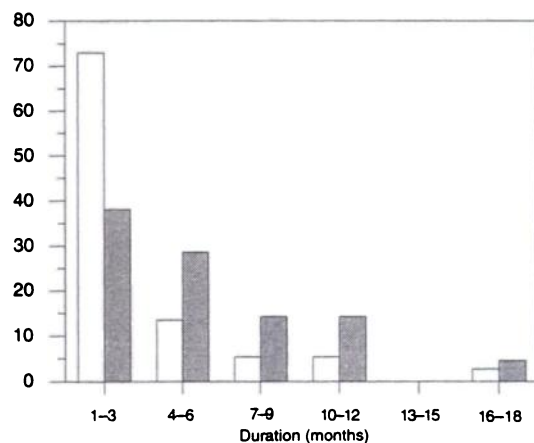


Fig. 1 Duration of index episode of postnatal depression. □, *de novo* episode; ■ recurrent episode.

Table 1  
Recurrence of depression

	First onset PPD	Subsequent onset PPD	Controls
<i>n</i>	19/34 (56%)	14/21 (67%)	14/40 (35%)

PPD: postpartum depression.

Of those women who had had postnatal depression, four had been prescribed antidepressant medication by their general practitioner at some time during the 18-month period. None had been referred to a psychiatrist.

#### Subsequent course

For the control group and the two postnatally depressed groups, mental state was examined over the three-and-a-half-year period from 18 months to five years after the index delivery. Table 1 shows the number and proportion within each group who experienced an episode of depression during this three-and-a-half-year period. It can be seen that 35% of the control group suffered an episode of depression (RDC major or definite minor), as compared with 60% (i.e., 33/55) of those who had been depressed postnatally ( $\chi^2=4.83$ ; d.f. = 1;  $P<0.05$ ). Of those for whom the index episode was a first onset, 56% had a subsequent episode; and of those for whom the index episode was a recurrence, 67% had a subsequent episode. These rates are not significantly different from one another.

Ten per cent of both the postnatally depressed groups had been prescribed antidepressant medication at some time between the 18-month and five-year assessments. None had been referred to a psychiatrist.

#### Subsequent non-postpartum depression

The figures cited above (derived from Table 1) do not exclude depressive episodes which may have followed deliveries after the index one. Of the 95 women, 73 had had at least one subsequent delivery (85% of the control group, 81% of those for whom the index episode had been a recurrence, and 65% of those for whom the index episode had arisen *de novo*;  $\chi^2=4.51$ ; d.f. = 2;  $P>0.10$ ). This permitted an analysis of the rate of subsequent postpartum and non-postpartum depression within each of the three groups. A later episode of depression was coded as postpartum if its onset was within 12 weeks of a delivery. Table 2 shows the number of women who experienced a non-postpartum episode of depression

Table 2  
Recurrence of non-postpartum depression

	First onset PPD	Subsequent onset PPD	Controls
<i>n</i>	13/34 (38%)	13/21 (62%)	10/40 (25%)

PPD: postpartum depression.

(RDC major or definite minor). It can be seen that the rate of such depression among the controls was 25%; among those for whom the index episode arose *de novo*, it was 38%. However, among those for whom the index depression was a recurrence, the rate for later non-postpartum depression was 62%. This distribution of proportions showed a significant variation among the groups ( $\chi^2=7.97$ ; d.f. = 2;  $P<0.05$ ).

#### Subsequent postpartum depression

Table 3 shows the number of women who had an episode of depression between 18 months and five years after the index delivery at a time classed as postpartum (as defined above). The rates differed significantly for the three groups: 12% of the control had such an episode, as did 18% of those for whom the index episode of depression had been a recurrence. However, of those for whom the index episode had been a first onset, 41% had a later postnatal depression ( $\chi^2=6.87$ ; d.f. = 2;  $P<0.05$ ).

An attempt was made among those who had another delivery to compare the risk of an episode of depression postpartum with the risk of an episode at some other time. This was done separately for the two depression groups. To avoid contamination with the index episode, the time frame considered was the three-and-a-half-year period from 18 months to five years after the index delivery. For each group, the rate of depression (RDC major or definite minor) arising within three months of a delivery was compared with the rate in the last three-month period considered (i.e. 57–60 months), where this period

Table 3  
Recurrence of postpartum depression

	First onset PPD	Subsequent onset PPD	Controls
<i>n</i>	9/22 (41%)	3/17 (18%)	4/34 (12%)

PPD: postpartum depression.

did not represent a postpartum period. The rate of depression during these two periods was very similar for the 17 women for whom the index depression had not constituted a first onset of disorder: thus, of the five episodes identified, three had arisen in a postpartum phase and two in the comparison phase. The findings for the 22 women for whom the index episode had arisen *de novo* were very different: thus, of the 11 episodes identified (accounted for by nine women), nine had arisen in a postpartum phase and two in the comparison phase.

### Discussion

It has been suggested that the population of women who develop non-psychotic depression after childbirth subsumes two distinct subgroups: those for whom the experience of having a child constitutes a specific causative factor, and those for whom the birth is not a specific stressor. In the current study, this suggestion was examined by studying the course and recurrence of postnatal depression in women who experienced depression for the first time postnatally, and in women for whom the postpartum depression was a recurrence of mood disorder. It was hypothesised that, if childbirth constituted a specific vulnerability for the former group, these women would be at increased risk of later postpartum depressive episodes. This was found to be the case: among those who had another delivery, the rate of postpartum depression was much higher for the group in whom the index episode had been a first onset than among those for whom the original postpartum depression had constituted a recurrence of depression.

The two depression subgroups differed in the duration of the index postpartum depressive episode. Those for whom the depression was a first onset had a much shorter duration of disorder than those for whom the depression was a recurrence. The reason for this is unclear. However, it is possible that the specific vulnerability of these women involves issues which arise in the context of the relationship demands of early infancy, and that these issues no longer obtain with the end of the phase of primary intersubjectivity (Trevarthen, 1979).

The short duration and a specific vulnerability to postnatal depression are consistent with a biological cause, as is the fact that many instances of postnatal depression follow a phase of severe maternity 'blues'. However, despite extensive research into steroid hormones in women during the puerperium, no firm evidence has emerged linking these hormones to the development of postnatal depression (Harris, 1994). It has been suggested that in a small subgroup of those experiencing postnatal depression there might

be a thyroid disorder (Harris, 1993). This possibility merits detailed attention in view of its important therapeutic implications. However, Harris notes that this account is not necessarily at variance with a psychological formulation of the ontogenesis of postnatal depression such as the one outlined above, as the thyroid dysfunction could be secondary to immunologic changes brought about by stress. Clearly, detailed prospective work is required to establish the relationship among the relevant biological, psychological, and social factors involved.

Whether the principal cause is biological or intrapsychic, the findings of the current study suggest that the notion of postnatal depression might well have a specific nosologic reference. A further line of supportive evidence comes from the finding that, as compared with the infants of those for whom the postpartum episode was a recurrence of depression, the infants of those for whom the postpartum episode was a first onset are significantly more likely to perform poorly on cognitive tasks at nine months of age, and at 18 months are more susceptible to the adverse effect of low social class and male sex on cognitive and language development (Murray, 1992). A number of further lines of evidence need to be explored. In particular, it is important to establish whether specific causative factors operate for this group, as well as whether they have a specific response to treatment.

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