Adverse effects of maternal antenatal anxiety on children: causal effect or developmental continuum?[†]

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There is much literature on the effects on the developing brain of adverse events in pregnancy and the sensitive period postpartum, both in humans and in animals (Perry & Pollard, 1998). The study published by O'Connor et al (2002, this issue) contributes to this by suggesting that maternal antenatal anxiety increases the risk of behavioural problems in early childhood. They suggest that this could be due to the direct effect of maternal anxiety and stress on foetal brain development. This study also contributes to the longstanding and increasing evidence base that suggests that maternal mental ill health is related to childhood difficulties (Murray et al, 1996).

THE NEURODEVELOPMENTAL CONTINUUM

In the different field of early life predictors of adult psychiatric disorder, there is now substantial evidence linking adverse events during pregnancy and childbirth to the development of schizophrenia (Isohanni et al, 2000) and affective disorder in later life (O'Keane, 2000). Two recent volumes of the British Journal of Psychiatry contain sections devoted to papers on developmental psychopathology (Harrington, 2001a,b). These can be seen to unite all three areas into a developmental continuum from prenatal and early postpartum life, through childhood and into adult life.

Developmental psychiatry, developmental psychopathology and neurodevelopmental psychiatry emphasise the complex interplay between multiple risk and protective factors. Such factors include genetic, neuroendocrine, environmental and psychosocial factors, with adverse events contributing during pregnancy,

[†]See pp. 502–508, this issue.

delivery, the neonatal period and during childhood. Individual risk factors are seldom seen as causal but rather that they contribute to a dynamic, interactive process over time (Harrington, 2001b).

SCORES ON SELF-REPORT QUESTIONNAIRES OR DISORDER AND INTERVENTION?

Most of the studies in the literature of the effects of adverse events on the developing brain describe stressors that are obviously either unusual or traumatic, such as extreme stress, violence or privation (Rutter et al, 2001). Studies of the effects of maternal mental ill health on children and of the prenatal and obstetric events associated with adult mental health problems describe categorical events that can be clearly defined, for example physical illnesses in pregnancy, obstetric complications or outcome such as schizophrenia. The Avon Longitudinal Study of Parents and Children (ALSPAC) used in O'Connor et al's (2002) study is different in that it uses the results of postal questionnaires on anxiety and depression from a very large cohort of women who delivered a baby in 1991-1992, at two points during pregnancy and four points during the 3 years following delivery. The behaviour of the child delivered in that year is then assessed at 4 years by means of a self-report scale completed by the mother. Both the emotional state of the mothers in pregnancy and the postpartum years and the behavioural state of the children at 4 years represent a continuum from normality to pathology. For the purposes of further statistical analysis, a cut-off point was decided arbitrarily on all of these measures, above which it was thought likely that these women and their children might be suffering from clinically significant conditions. The authors are careful to emphasise that a cut-off score on a self-report scale is not equivalent to a clinical diagnosis. They also acknowledge that self-report scales have limitations, in that anxious mothers might be more likely to express concern about their children. However, the authors believe that their statistical analyses allow them to conclude that not only was antenatal anxiety related to childhood disturbance and that the mothers who suffered most antenatal anxiety were those with the most disturbed children at 4 years, but also that the effects of antenatal anxiety were independent of the effects of postnatal anxiety and of depression. They argue that this supports the hypothesis that a neuroendocrine process is adversely affecting the infant's brain during pregnancy.

Many of the women who are most anxious during pregnancy are likely to be so as the result of stress and adversity. It is difficult to imagine that housing problems, problems at work, or with families and partners, and even violence, will stop at the point of delivery. The only causes of anxiety likely to be cured by childbirth are those relating to the pregnancy itself. We already know that selfreport scales of anxiety and depression rise during pregnancy and fall following delivery (Green & Murray, 1994), presumably because many items reflect the common discomforts and concerns of late pregnancy.

This paper describes a continuum of antenatal anxiety related to a continuum of reported disturbance in children. To suggest in their conclusions that intervention programmes targeted specifically anxious mothers in pregnancy might reduce maternal anxiety and have protective effects for children seems to be a case of the statistical tail wagging the clinical dog. This paper, like another recent paper on antenatal depression from the same ALSPAC (Evans et al, 2001), moves from the cautious language of dimensions and continuous variables to the language of disorder and treatment in the conclusion. It is a utopian fantasy to imagine that there will ever be sufficient therapists or intervention programmes to treat all anxious women during pregnancy. It is unlikely, even if such resources existed, that they would be able to address the complex difficulties that often underlie such problems. Conclusions such as these are likely only to increase the expectations and subsequent disappointments of both women and their clinicians and to induce further worry and anxiety. It could also lead some clinicians to treat these women with psychotropic medication. If, as this paper suggests, the foetal brain is so sensitive, can we afford to be sanguine about the use of anti-depressants in pregnancy despite the lack of evidence so far of their harmful effect?

BLAMING MOTHERS – AN ANTHROPOLOGICAL PERSPECTIVE

Since the beginning of time, and across all cultures, child-bearing women have been subject to proscriptions and prohibitions of their activities. Many of these might appear to be quaint culture-specific rituals. Nonetheless, vestiges of these proscriptions and prohibitions persist in our own society and are reflected in many of the modern rituals and language of childbirth. Whatever the local meaning and cultural attributions of these behaviours, they are all open to a modern interpretation of protecting the mother and her developing infant from undue emotional stress, infection, malnutrition and a premature return to potentially stressful activities. At best, these expectations can be seen to provide a supportive and protective structure in which the woman can safely raise her child, both in utero and following delivery. At worst they can induce guilt when things go wrong, when a birth is complicated, or when the infant dies or develops problems. The mother will blame herself for failing to do those things that she was supposed to do or doing those things that she was not supposed to do. The tendency for societies and science to attribute adverse outcome to maternal

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fault increases this tendency to self-blame. Most perinatal psychiatrists will be familiar with the phenomenon of distraught mothers blaming themselves for a variety of childhood ills, because of early ambivalence to the pregnancy, working too hard, drinking alcohol on occasion or these and any of the other disapproved behaviours of pregnancy.

The modern Western pregnant woman must not drink more than four cups of coffee a day, drink alcohol, smoke cigarettes, change cat litter trays, eat soft cheese, uncooked eggs or packaged salads or go into the lambing sheds. They should not work too hard or too long, nor at night or be ambivalent about their pregnancies. Now it seems they must not become anxious either.

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DECLARATION OF INTEREST

None.

REFERENCES

Evans, J., Heron, J., Francomb, H., et al (2001) Cohort study of depressed mood during pregnancy and after childbirth. *BMJ*, **323**, 257–260. Green, J. M. & Murray, D. (1994) The use of the Edinburgh Postnatal Depression Scale in research to explore the relationship between antenatal and postnatal dysphoria. In *Perinatal Psychiatry: Use and Abuse of the Edinburgh Postnatal Depression Scale* (eds J. L. Cox & J. M. Holden), pp. 180–198. London: Gaskell.

Harrington, R. (2001a) Causal processes in development and psychopathology. *British Journal of Psychiatry*, **179**, 93–94.

____(2001b) Developmental continuities and discontinuities. British Journal of Psychiatry, 179, 189–190.

Isohanni, M., Jones, P., Kemppainen, L., et al (2000) Childhood and adolescent predictors of schizophrenia in the Northern Finland 1966 Birth Cohort – a descriptive life-span model. European Archives of Psychiatry and Clinical Neuroscience, **250**, 311–319.

Murray, L., Fiori-Cawley, A. & Hooper, R. (1996) The impact of postnatal depression and associated adversity on early mother—infant interactions and later infant outcome. *Child Development*, **67**, 2512–2526.

O'Connor, T. G., Heron, J., Golding, J., et al (2002) Maternal antenatal anxiety and children's behavioural/emotional problems at 4 years. Report from the Avon Longitudinal Study of Parents and Children. British Journal of Psychiatry, 180, 502–508.

O'Keane, V. (2000) Evolving model of depression as an expression of multiple interacting risk factors. *British Journal of Psychiatry*, **177**, 482–483.

Perry, B. & Pollard, R. (1998) Homeostasis, stress, trauma & adaptation: A neurodevelopmental view of childhood. *Child and Adolescent Psychiatric Clinics of North America*, **7**, 33–51.

Rutter, M. L., Kreppner, J. M. & O'Connor, T. G. (2001) Specificity and heterogeneity in children's responses to profound institutional privation. *British Journal of Psychiatry*, 179, 97–103.