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Declaration of interest

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Cognitive impairment in bipolar disorder

We write to correct some misconceptions evident in the recent editorial by Ferrier & Thompson (2002). Previously, we reported impairment in accuracy measures on recognition memory tasks and increased latencies on executive tasks in patients with bipolar disorder in remission (Rubinsztein *et al*, 2000). Ferrier & Thompson (2002) argue that the cognitive impairment observed in our study may have been confounded by the effects of 'residual' symptoms. As yet there is no generally accepted 'cut-off' for what constitutes remission. We devised rigorous criteria to define remission based on a patient's own view of his or her illness, that of their psychiatrist and a structured interview. We excluded patients with scores of $\geqslant 8$ on both the Hamilton Rating Scale for Depression (HRSD) and Young Mania Scale (YMS). These rating scales were devised to rate symptom severity in patients with an affective disorder and not for use in normal control subjects. Our average reported score on the HRSD was 2.1 (s.e.m.=0.5) and on the YMS it was 0.8 (s.e.m.=0.4). Thus, very few residual symptoms were evident and these scores certainly do not support any concern that patients had residual depression or mania.

Although the rationale for using such scales in controls is dubious, for the sake of argument we have reanalysed our data reported in Rubinsztein *et al* (2000) using a partial correlation analysis, as in Clark *et al* (2002), to control for differences observed on the HRSD (we did not rate control subjects using the mania scale) on the tests that showed significant impairment by analysis of variance (ANOVA). We still find significant impairment on both the visual recognition memory tasks and on latency measures from the one-touch Tower of London planning task (see Table 1).

These findings suggest that there are trait impairments in accuracy of visual recognition memory and slower responses on a planning task in bipolar remission. Importantly, impairments of memory and learning have been consistently observed in a number of other recent studies where rigorous diagnostic criteria for remission were applied (e.g. Van Gorp *et al*, 1998; Krabbendam *et al*, 2000; Cavanagh *et al*, 2002) as well as in a recent unpublished study (L. Clark, personal communication, 2002) that showed that verbal recall was still impaired following partial correlation for residual symptoms. The presence of significant impairments on executive tasks in bipolar remission has been more variable and may depend on clinical factors or the specific neuropsychological test paradigm employed. The precise functional significance of the cognitive impairment in bipolar remission needs to be examined further but may well impact on response to psychological and drug treatments. Cognitive symptoms could in fact be among the most sensitive indicators of incomplete remission.

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Antenatal anxiety, parenting and behavioural/emotional problems in children

O'Connor *et al* (2002) report the effects of antenatal anxiety on behavioural/emotional problems in 4-year-old children. Their analysis of the Avon Longitudinal Study of Parents and Children (ALSPAC), a longitudinal, prospective study of women, their partners and an index child (Golding *et al*, 2001) takes into account a number of important covariates, including postnatal anxiety, gestational age, birth weight, and socio-economic status. They have not, however, included any measures of parenting. This is of concern because there is now a substantial body of evidence to indicate a clear association between parenting and child emotional and behavioural problems. For example, there are now a number of empirically validated models depicting the developmental progression for conduct and behaviour problems. These show a clear association between parenting practices characterised by harsh and inconsistent discipline, little positive parental involvement with the child, poor monitoring and supervision, and behaviour and conduct problems in early childhood (Patterson *et al*, 1989). Indeed, work using structural equation models showed that parenting and family interaction variables accounted

Table 1 Results of partial correlation analysis on tests in which ANOVAs were significant

	Dependent variable	Partial correlation coefficients	*P*
Pattern recognition memory	Proportion correct	0.41	0.02
Spatial recognition memory	Proportion correct	0.31	0.07
Delayed matching to sample	Proportion correct	0.35	0.04
One-touch Tower of London	Response time	−0.42	0.02
Affective shifting task	Response time	0.04	0.81

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for 30–40% of the variance in child antisocial behaviour (Patterson *et al*, 1989).

It seems likely that parenting practices would not be adequately controlled for through the use of a socio-economic covariate owing to the fact that, although parenting practices are influenced by social and cultural factors such as class (Hoff *et al*, 2002), one of the most extensive epidemiological studies of childhood psychiatric disorders showed that social class was a poor predictor of child adjustment (Rutter *et al*, 1975).

It seems likely that parenting exerts an independent effect on child outcomes such as emotional and behavioural adjustment. The ALSPAC data contain a number of measures of parenting, including, for example, a standardised instrument measuring parenting practices during toddlerhood. It would be useful if further analysis of this data-set were undertaken to establish whether these important findings are maintained when parenting is included in the model.

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Author's reply: A recent study from our group indicated that antenatal anxiety is associated with a significantly increased risk of behavioural/emotional problems in young children (O'Connor *et al*, 2002). The study was based on the ALSPAC cohort, a prospective, longitudinal study of women followed since pregnancy. Analyses indicated that antenatal anxiety at 32 weeks' gestation was associated with an approximately 2-fold increase in behavioural/emotional problems in boys and girls at age 4 years; these associations were observed after accounting for key antenatal, obstetric and psychosocial risks, and postnatal anxiety and depression. The findings are important in providing the strongest evidence to date that the substantial evidence for long-term effects of antenatal stress/anxiety found in numerous animal investigations (e.g. Schneider & Moore, 2000) may extend to humans.

In our paper, the focus was on whether or not the antenatal environment had a role in the development of behavioural/emotional problems, an issue with substantial implications for our understanding of development, as well as for prevention and public health. Dr Barlow's letter helps draw attention to a separate research base linking behavioural/emotional problems in children with postnatal factors, particularly parent–child relationship quality. Although there remain some controversial matters in that field of research, especially concerning causal mechanisms (see O'Connor, 2002), parent–child relationship quality is certainly a robust predictor of children's psychological development. Given the multiple-risk nature of development and psychopathology, we would agree with Dr Barlow that there is a need to bring together findings from different lines of research and to revise our models and theories that consider multiple levels of risk. Indeed, there are a number of directions for this research to pursue, including the consideration of how postnatal experiences such as parent–child relationship quality moderate the effects of antenatal anxiety/stress and how the role of genetic factors may explain individual differences in response to antenatal anxiety/stress. Research along these lines is underway. Because it has tracked women intensively since pregnancy and has continued to collect information on a wide range of biological and psychosocial variables, the ALSPAC study is an especially important resource for studies of this kind.

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Brain weight in suicide revisited

In their excellent paper Hamilton & McMahon (2002) examined brain weight in suicide victims of all ages to see whether it was higher than in a control group. They attempted to replicate and reinterpret our findings (Salib & Tadros, 2000) reported in an elderly sample. The authors, quite rightly, looked at brain weight in cases and controls adjusted for body mass index (BMI), having collected additional data about body weight and height, data which Salib & Tadros (2000) were not able to collect and which was already accepted as a major limitation in the latter study.

Hamilton & McMahon (2002) did not find any significant difference between brain weight adjusted for BMI in cases and controls. However, brain weight was significantly higher in those dying by hanging than in those dying by overdose.

I would like to make one or two comments which may help to explain the difference in the findings of the two studies. In Hamilton & McMahon's study, the mean age is 38.5 years (for cases and controls) compared with 72 years in the study by Salib & Tadros (2000). Also, the mean brain weight for Hamilton & McMahon's control group was 1449 g compared with 1238 g in the sample reported by Salib & Tadros (2000). Hamilton & McMahon (2002) included only 6% of subjects aged over 60. The method of selection of the control group in their sample is different from that used by Salib & Tadros (2000) – the latter study included only controls who died naturally and not accidentally. Hamilton & McMahon (2002) were not able to replicate our findings in an elderly sample but were careful in their comparison of the findings by taking into account the differences in some basic parameters in the two studies.

It is interesting to note that another recently published paper (Balazic & Marušič,

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