Correspondence

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Left frontal activation

We read with considerable interest the paper by Shergill *et al* (2004) about the temporal course of brain activity associated with auditory verbal hallucinations. The researchers used functional magnetic resonance imaging to reveal those brain regions activated before, during and after such hallucinations (the occurrence of which was indicated by patients pressing a button). They concluded that activation of the left inferior frontal gyrus some 9 seconds prior to button pressing supports the theory that hallucinations originate in brain areas involved in the generation of 'inner speech'. Given the importance of this question for future paradigm development, we wish to offer constructive comment.

There is a difficulty associated with the experimental method as described. Because no control condition was included (in which, for example, subjects might self-initiate button presses, unrelated to the timing of hallucinations) we cannot ascertain whether the frontal activation was attributable to the auditory verbal hallucinations or the procedure of button pressing itself; this problem emerged in the interpretation of an earlier, similar study (McGuire *et al*, 1993; Krams *et al*, 1996). In healthy individuals we have observed that the left frontal cortex also activates 9 seconds prior to simple, self-initiated button pressing (Hunter *et al*, 2004). Obviously, in healthy individuals this has no relationship to auditory verbal hallucinations (it is a feature of the temporal evolution of normal voluntary motor behaviour). During such behaviour, maximal frontal activity is seen in the middle and inferior frontal gyri (9 s prior to button pressing). The temporal sequence of frontal activation observed by Shergill *et al* (2004) could be related to the hallucinations or be attributed to the self-initiation of motor action (button pressing). This methodological consideration radically constrains the authors' conclusions. The techniques of functional neuroimaging are complex and unfamiliar to most general readers. We hope that the concern we raise is helpful in elucidating the methodological issues inherent in studies such as these.

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Confounding factors for depression in adults with mild learning disability

The point prevalence of a major depressive illness in people with learning disability is between 2 and 7%, which means that depression can be twice as common in this group as in the general population (Prasher, 1999).

Collishaw *et al* (2004) present strong evidence for directing strategies of primary prevention towards socio-economic deprivation and ill health in people with mild learning disabilities. However, these results should be viewed with caution as the study did not control for certain important factors. Certain groups of people with learning disability are shown to be at a risk of developing a depressive illness, for example those with Down's syndrome, fragile-X syndrome or epilepsy (Prasher, 1999). Down's syndrome and fragile-X syndrome are among the most common genetic causes of learning disabilities, and epilepsy is 10 times more common in people with mild learning disability than in the general population (Bird, 1997).

This implies that factors other than socio-economic deprivation could have contributed to the depressed mood in those with mild learning disability.

Bird, J. (1997) Epilepsy and learning disabilities. In *Seminars in the Psychiatry of Learning Disabilities* (ed. O. Russell), pp. 223–244. London: Gaskell.

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Authors' reply: We investigated the extent to which adult social adversity and ill health contributed to an elevated risk for depressed mood among adults with mild learning disability (Collinshaw *et al*, 2004). The study used data from the 1958 National Child Development Study (NCDS), a nationally representative cohort followed from birth to age 43 years.

Dr Feroz-Nainar makes the point that epilepsy, fragile-X syndrome and Down's syndrome are among the biological/genetic causes and correlates of learning disabilities and raises the question whether these factors contributed to the higher rate of depressed affect associated with mild learning disability.

A previous report on the NCDS birth cohort confirms that epilepsy and other neurological abnormalities were indeed more common for individuals with mild learning disabilities than for controls. However, the majority of individuals with mild learning disability had no known neuro-epileptic abnormalities and mild learning disability was more commonly associated with childhood social and family adversity (Maughan *et al*, 1999).

To investigate the possibility that group differences in depressed affect were due to biological factors such as epilepsy in those with mild learning disabilities, we re-analysed the statistical models

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reported in our recent paper (Collishaw *et al*, 2004). Controlling for childhood epilepsy/neurological problems did not reduce group differences in adult depressed affect (model adjusted only for gender: OR=2.84, 95% CI 1.7–4.9, $P<0.001$; model adjusted for gender and childhood neurological problems/epilepsy: OR=2.79, 95% CI 1.6–4.8, $P<0.001$). This is in contrast to the partial mediating effect of controlling for childhood social adversity (Maughan *et al*, 1999; Collishaw *et al*, 2004) and the almost complete mediating effect of additional controls for adult ill health and adult social adversity (Collishaw *et al*, 2004).

We cannot rule out completely the possibility that some other unmeasured third factor is confounded with social adversity and could explain our findings. We also acknowledge that specific biological factors may be of particular importance for understanding affective problems in some individuals with mild learning disability. Nevertheless, when assessed in an unselected general population cohort such as the NCDS, social factors and adult health do appear to have an important contribution to depressed mood among people with mild learning disability.

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Prevalence of dementia

We thank Dr Varghese (2005) for his letter regarding our article (Shaji *et al*, 2005). Lack of education is a potent predictor of poor performance across many items of the Chinese version of the Mini-Mental State Examination (MMSE; Katzman *et al*, 1988). There was no significant difference between total MMSE scores of those who were illiterate and those who were literate in the pilot study conducted with the Malayalam adaptation of the MMSE. Hence it was decided to use the same score for both groups.

We identified 55 cases of dementia among 327 people who scored at or below the cut-off on the MMSE. The one case identified from the 10% of the negatively screened population was counted as one among the ten cases in the negatively screened population of 1607 (i.e. 65 cases in 1934 people aged 65 years and above).

The assessment of risk factors based on retrospective accounts of the carers and an inadequate number of controls for calculating the odds ratios can be considered methodological limitations of the study. The prevalence of dementia increases proportionately with age ($\chi^2=40.29$, d.f.=5, $P<0.001$). This χ^2 value was not given in the text. The number of patients with Alzheimer's disease was 30. The error in the article is regretted.

Katzman, R., Zhang, M.Y., Ouang-Ya-Ou, *et al* (1988) A Chinese version of the Mini-Mental State Examination; impact of illiteracy in a Shanghai dementia survey. *Journal of Clinical Epidemiology*, **41**, 971–978.

Shaji, S., Bose, S. & Verghese, A. (2005) Prevalence of dementia in an urban population in Kerala, India. *British Journal of Psychiatry*, **186**, 136–140.

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Measures for mental health outcomes

I was very interested to read the article by Salvi *et al* (2005) on choosing the measure for mental health outcome assessments. Readers might be interested in a comparison of the Camberwell Assessment of Need Short Appraisal Schedule (CANSAS; Phelan *et al*, 1995) and Health of the Nation Outcome Scale (HoNOS; Wing *et al*, 1998) scores. One thousand pairs of HoNOS and CANSAS scores were recorded by four trainees and myself. Figure 1 shows the means with standard errors of the HoNOS values associated with each CANSAS score.

The higher CANSAS scores (13–22) were not encountered very often and accounted for only 3.5% of scores. The large standard errors are because some of the CANSAS scores occurred infrequently.

HoNOS and CANSAS scores are related in the lower CANSAS range of 1–8, the most common range, accounting for 79% of the scores. Up to a CANSAS score of 12 ($n=955$) there is a reasonably close correlation with the HoNOS scores. The Spearman coefficient is 0.564, indicating that the correlation is significant at the 0.01 level (two-tailed).

The use of CANSAS is becoming established in Lothian mental health services. CANSAS is very useful as a needs assessment tool for individual patients. Its face

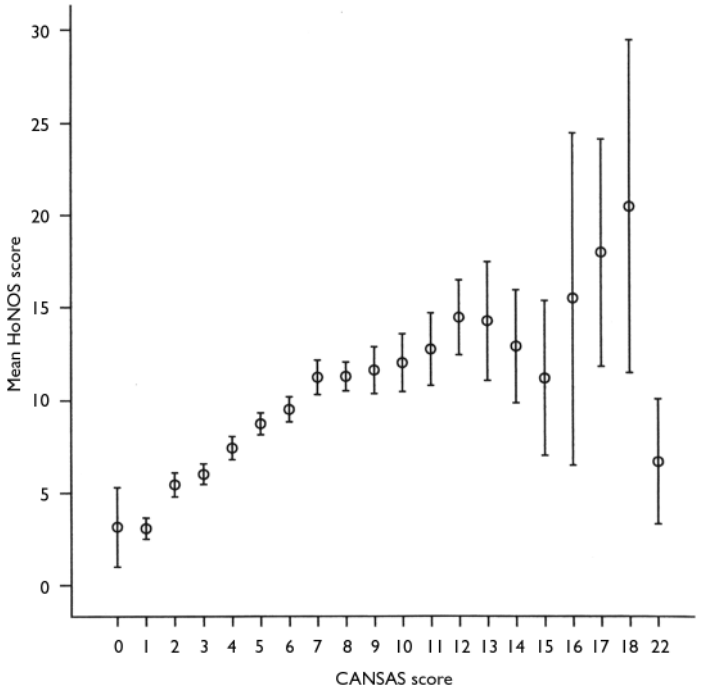

Fig. 1 Comparison of 1000 pairs of CANSAS and HoNOS scores. Bars represent two standard errors above and below the mean. CANSAS, Camberwell Assessment of Need Short Appraisal Schedule; HoNOS, Health of the Nation Outcome Scale.

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