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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.

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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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