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Pathogenesis of white matter lesions in Alzheimer's disease and depression

SIR: O'Brien *et al* (1996) highlight the associations between deep white matter lesions (DWML) and depression and between periventricular lucencies (PVL) and Alzheimer's disease. Although the contribution of vascular risk factors to these associations was closely examined, the influence of cerebrovascular disease in the pathogenesis of both white matter lesions remains unclear. Firstly, it is striking that vascular risk factors were significantly more common in depressed subjects than in those with Alzheimer's disease. However, notwithstanding the fact that the association between DWML and depression still existed after controlling for these risk factors, it is possible that other vascular risk factors may not have been taken into account. Those subjects with depression who had a past history of transient ischaemic attacks do not appear to have been excluded from the sample; it is possible that such episodes may have contributed to the development of DWML. The role of 'silent' infarcts may also be important, given their association with radiological changes and disruption of frontal connections (Meyer *et al*, 1995). A role for DWML as a risk factor for depression is put forward; again, a vascular contribution may be important in view of 'pre-stroke depression' found to be a possible risk factor for completed stroke (Colantonio *et al*, 1992).

The authors found no evidence of an association between PVL (which they suggest may involve other pathophysiological mechanisms) and vascular risk factors. Using both linear and volumetric measures, Schmidt (1992) found PVL to be significantly more common in vascular dementia than probable Alzheimer's disease. Furthermore, such lesions have also been shown to predict later development of clinically apparent cerebrovascular disease (Lopez *et al*, 1992). Thus, the role of cerebrovascular disease in the development of PVL remains open to speculation.

Before a more definitive statement about the role of vascular risk factors in the pathogenesis of white matter lesions can be made, prospective clinicopathological studies (including the use of *in vivo* neuroimaging) are needed to allow a better understanding of the relative contributions of vascular and non-vascular factors to such lesions in depression and Alzheimer's disease.

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Drug induced psychosis

SIR: We agree with the main conclusion of Poole & Brabbins (1996) that the use of the term "drug induced psychosis" may cause misunderstanding and should be discontinued. We would nevertheless like to draw the readers' attention to what seems to be a misunderstanding of our research on cannabis and schizophrenia (Andréasson *et al*, 1987, 1989; Allebeck *et al*, 1993).

The aim of our studies was not to elucidate the concept of "drug induced psychosis", but to assess the role of cannabis as one of several risk factors for schizophrenia. Poole & Brabbins are incorrect in saying that we did not take account of confounding factors that might be related to the exposure (cannabis use) as well as the outcome (schizophrenia). A number of potential confounders were analysed first by stratified analysis and then in a logistic model. The relative risk of schizophrenia among cannabis users decreased in these analyses, indicating that some of the association could be explained by these factors. Even in the logistic model, simultaneously controlling for a number of background factors, the relative risk for schizophrenia was significantly increased among high consumers of cannabis as compared to non-users. Additional analyses (Andréasson *et al*, 1989) showed that cannabis use indeed preceded the onset of schizophrenic symptoms and that other indicators of mental disturbances, which could act as precursors of both

cannabis use and schizophrenia, were not present. Similar results were obtained in a longitudinal study based on patients treated in Stockholm County (Allebeck *et al*, 1993).

Thus, although our research did not address the specific issue of "drug induced psychosis", as defined by Poole & Brabbins, we believe there is evidence that cannabis use may increase the risk of psychosis, in particular schizophrenia, beyond the immediate effect of intoxication or flashback.

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Ultra-rapid mood cycling

SIR: Kramlinger & Post (1996) intrigued me when they made reference to the potential contribution of prior sexual abuse to ultra-rapid mood cycling. Two further 'ultra-rapid' cases may be of interest. The first is a woman under intensive community based treatment and requiring frequently fine tuned polypharmacy for many years. An attempt to improve her chronically disabled state with a major revision and reduction of treatment was followed by over a year of chaos with mood cycling as often as every three days, depressions lasting many weeks, marked suicidality and apparent gestures of same. In the midst of this, many years into treatment, the patient surprised everyone by disclosing sexual abuse, and subsequently during the brief good spells took up an evening class and used public transport for the first time in a decade. The mood cycles continue but her functioning is undoubtedly improved. The second patient disclosed sexual abuse far earlier in her symptoms, but while her depressions seem typical, her rapid swings into the subjective complaint of 'being high' is vague and at times unconvincing. The striking thing about these two people, apart from the rapidity of their mood swings however is that both cling to a biological cause to their problems. It may well be that this is appropriate insight and a success of treatment, but I am struck that both high and low mood states prevent proper communication and may confer secondary gain. Furthermore might a diagnosis confer a form of personal identity seemingly lacking in survivors of abuse, and the hyperarousal following trauma respond partly to the majority of treatments employed which tend to reduce arousal and awareness?

While not suggesting that treatments should or could be withheld, I welcome Kramlinger & Post flagging the wider aetiological possibilities of this challenging disorder.

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No right to a Mental Health Review Tribunal?

SIR: The Mental Health Act (MHA) 1983 allows all detained patients, on sections providing for detention of longer than 72 hours, the right to appeal to a mental health review tribunal (MHRT) against their detention. Or so I believed. I have recently become aware that there are certain individuals who, when admitted to hospital under section 3 of the MHA 1983, are not entitled to a MHRT.

A patient at the Reaside Clinic in Birmingham detained under sections 37 and 41 of the MHA 1983 was granted a conditional discharge by a MHRT. The necessary conditions were met and the patient was discharged into the community. With such a discharge the Home Secretary may recall the patient to hospital at any time. The responsible medical officer (RMO) does not have the power to recall the patient, but can advise the Home Secretary to do so.

The patient's condition subsequently deteriorated in the community necessitating compulsory readmission to hospital under section 3 of the MHA 1983. It was hoped that detention under section 3 would allow greater clinical flexibility than recall by the Home Secretary, as such recall can lead to lengthy negotiations with the Home Office prior to release.

The patient objected to his detention under section 3, and therefore applied to the MHRT but the application was refused. The Department of Health has provided me with a legal opinion which