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Psychopathological syndromes and familial morbid risk of psychosis

Sir: I wish to comment on the article by Van Os *et al* (1997). Before using the term "broad schizoaffective psychosis" the authors should consider the possibility that their original group of schizoaffective psychosis is already too broadly defined. Of the 150 patients with psychosis they diagnosed 65 as schizoaffective, giving a figure of 43% of consecutively admitted patients. When Brockington & Leff (1976) looked at the diagnosis of schizoaffective psychosis in what would seem to be an identical setting, in 222 consecutively admitted patients they found an incidence of only eight cases diagnosed according to the same Research Diagnostic Criteria (RDC). An almost tenfold increase in the diagnosis needs an explanation.

The RDC acknowledges that schizoaffective disorders could be either variants of affective disorder or schizophrenia, and in fact lays down criteria to further subdivide schizoaffective psychosis into predominantly depressive or manic and predominantly schizophrenic. This distinction is important as Brockington *et al* (1980) have shown that schizomania is a variant of bipolar disorder and that schizodepressive disorder is often closely related to schizophrenia (Brockington *et al*, 1978). As no subdivision has been made this should compromise the results. It is possible therefore that the group of schizoaffective disorder which the authors compared with schizophrenia already had a large number of schizophrenics, hence the findings that schizophrenia familial risk was higher in this group. It would certainly be helpful if this information were available.

Adams, R. & Taylor, M. A. (1976) Catatonia, a prospective clinical study. *Archives of General Psychiatry*, **33**, 579–581.

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Van Os, J., Marcelis, M. Sham, P., *et al* (1997) Psychopathological syndromes and familial morbid risk of psychosis. *British Journal of Psychiatry*, **170**, 241–246.

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Authors' reply: The likely reason for the higher rate of RDC schizoaffective disorder in our patients is that diagnoses were not only based on cross-sectional, but also on longitudinal (lifetime) psychopathological data. 'Pure' affective and schizophrenic states will become rarer with time as significant proportions of such patients will experience schizophrenic and affective symptoms respectively.

We agree that separate examination of schizodepressive and schizomanic cases would have been interesting and possibly revealing. The sample size, however, was too small to allow for such analyses.

While these diagnostic issues may be important, we would like to stress that the main findings of our paper involved effects that were independent of proband diagnosis.

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Treatment, outcome and predictors of response in elderly depressed in-patients

Sir: Heeren *et al* (1997) reported a naturalistic prospective study of treatment, outcome and predictors of response in elderly depressed in-patients in The Netherlands. Their findings showed a low percentage of full recovery (33–45%), with a high rate of partial recovery (43–66%), compared with previous studies. They concluded that a combination of inadequate treatment and therapeutic nihilism played a major role. Similar findings are paralleled in the adult service with failure to treat resistant depression adequately (Bridges *et al*, 1995).

However, there may be other reasons for such a low response in this study. The sample consisted of a wide variety of affective disorders, ranging from adjustment disorder to organic depression. Indeed, the study claimed to exclude patients with 'dementia'. How this was achieved is not clear and notably patients with Mini-Mental State Examination scores as low as five were recorded, with no evidence to suggest that this was a reversible cognitive impairment at follow-up. Thus this group of patients was an exceedingly heterogeneous mix, making it difficult to compare outcomes with previous studies.

If one assumes that the poor response is due to undertreatment, then this could be confirmed by comparing the two subgroups of patients, those treated with 'classic' antidepressants and 'modern' antidepressants. Undertreatment occurred much more frequently in the former group (82 *v.* 36%) and if their hypothesis is correct, should show a poorer prognosis. No comment was made regarding these data and it would be interesting to analyse this further.

Bridges, P. K., Hodgkiss, A. D. & Malizia, A. L. (1995) Practical management of treatment-resistant affective disorders. *British Journal of Psychiatry*, **170**, 436–440.

Heeren, T. J., Derksen, P., Heycop Ten Ham, B. F. v., *et al* (1997) Treatment, outcome and predictors of response in elderly depressed in-patients. *British Journal of Psychiatry*, **170**, 436–440.

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Authors' reply: We thank Pinner & Bouman for their critical comments and we agree with them that probably not only elderly depressives are the victims of therapeutic reticence. The first point concerns the heterogeneity of the population. The purpose of our study was to include as much as possible a 'normal' clinical population of elderly depressives and not a highly selected sample as usually occurs in clinical trials. Inclusion and exclusion were based on DSM–III–R criteria and not on the scores of the rating scales (these were used as measures of severity), so it could occur that one patient diagnosed with a recurrent major depressive episode had initially a score of five points on the MMSE. Although, unusual for a depressed patient, several factors such as motivation or physical illness may explain such a low score.

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The authors ask whether the hypothesis of undertreatment has been tested by comparing the data of patients who received a modern antidepressant with those who were treated with a classic antidepressant (36 *v.* 82% sub-optimal dose). The design of the study with outcome measures only at discharge did not permit such an analysis because all patients who had more than one treatment usually received both a modern and a classic antidepressant, but analysis of subgroups in the sample may give an answer in the future.

Undertreatment, however, is not only low-dose treatment but also the tendency not to set a next step in the treatment strategy when incomplete recovery occurs.

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BSE and human prion disease

Sir: In his review of our current understanding of the possible relationship between bovine spongiform encephalopathy (BSE) and human prion diseases Harrison (1997) appears reasonably certain that "all potentially infectious material has been banned from the food chain" as a result of the 1989 Specified Offals Ban. I fear that his confidence on this point may be misplaced.

The Specified Offals Ban excludes organs which contain significant amounts of central nervous system tissue ante mortem, principally brain and spinal cord. However, the slaughter process itself, consisting as it does of significant brain trauma from a 'bolt', may result in embolisation of central nervous system tissue to other parts of the body around the time of death. This was graphically demonstrated by Garland *et al* (1996*a*), who found macroscopic quantities of brain tissue (confirmed histologically) in "the main pulmonary artery of 2.5–5% of cattle after slaughter" – the largest embolus found was 14 cm in length! Such brain embolisation is a long-recognised complication of both open- and closed-head trauma in humans (McMillan, 1956).

Despite a claim by the Ministry of Agriculture, Fisheries and Food (MAFF) that "there is . . . no evidence that human lungs present a source of human exposure to bovine brain tissue in the UK" (Taylor, 1996), bovine lungs are legally permitted in certain meat products "sold for consumption without further cooking", such as salami and patés (MAFF, 1984). In addition, the British Veterinary Association, in a fax cited by Garland (1996*b*), confirmed that "it is perfectly legal for bovine lungs to enter the human food chain", and that "lungs can legally be sold in their fresh state".

Given Garland *et al*'s findings, our still-limited understanding of the risk to humans of the ingestion of prion-containing bovine tissue and the possible prevalence of currently incubating 'new-variant' Creutzfeldt–Jacob disease (Cousens *et al*, 1997), we should ensure that *no* part of any BSE-infected animal enters the human food chain, rather than merely excluding central nervous system tissue, as at present.

Cousens, S. N., Vynnycyk, E., Zeidler, M., *et al* (1997) Predicting the CJD epidemic in humans. *Nature*, **385**, 197–198.

Garland, T., Bauer, N. & Bailey, M. (1996*a*) Brain emboli in the lungs of cattle after stunning. *Lancet*, **348**, 610.

Garland, T. (1996*b*) Brain emboli in the lungs of cattle. *Lancet*, **348**, 749.

Harrison, P. J. (1997) BSE and human prion disease. *British Journal of Psychiatry*, **170**, 293–300.

Ministry of Agriculture, Fisheries and Food (1984) The Meat Products and Spreadable Fish Products Regulations, Statutory Instrument No. 1984. No 1566.

McMillan, J. B. (1956) Embolism of cerebral tissue in lungs following severe head injury. *American Journal of Pathology*, **32**, 405–415.

Taylor, K. C. (1996) Brain emboli in the lungs of cattle. *Lancet*, **348**, 749.

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Risk assessment and clinical risk management

Sir: Reed (1997) states that risk factors for future violence are summative, and that the co-existence of mental illness, psychopathic disorder and substance misuse may present a very significantly increased risk. This is not necessarily so. A study of patients discharged from a special hospital (Jones *et al*, 1994) found that, specifically in the case of patients classified as psychopathically disordered, co-morbidity in the form of mental illness or substance misuse significantly reduced the risk of further convictions for serious criminal offences after discharge.

We hypothesised that the existence of a psychotic illness, or substance misuse, could provide a focus for treatment, and a specific risk factor which could be reduced by treatment. In contrast, 'pure' psychopaths, for whom no definitive treatment may be available, and in whom improvement may be harder to quantify, are likely to represent a higher future risk to others.

Historical data, by its very nature, remain unchanged by the therapeutic process. Risk, in contrast, is dynamic, and accurate risk assessment must be able to modify actuarial consideration in the light of treatment response. Risk assessment is a complex procedure, relying crucially upon a detailed clinical understanding of an individual patient. General principles must always be considered only as a starting point for assessment, and factors which in general increase risk may, if amenable to therapeutic interventions, paradoxically serve to reduce that risk, if their impact on future behaviour can be altered.

Reed, J. (1997) Risk assessment and clinical risk management: the lessons from recent enquiries. *British Journal of Psychiatry*, **170** (suppl. 32), 4–7.

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Serotonin: 5-HT$_{2A}$ receptor occupancy *in vivo* and response to the new antipsychotics olanzapine and sertindole

Sir: Up to a third of patients do not respond to typical antipsychotics which potently block-dopamine type 2 (D_2) receptors. This treatment is often associated with distressing extrapyramidal side-effects. The novel atypical antipsychotic drugs clozapine, risperidone, sertindole and olanzapine appear relatively free from extrapyramidal side-effects. It has been suggested that this is secondary to 5-HT$_{2A}$ antagonism. *In vitro* and indirect evidence suggests that the ratio of 5-HT$_{2A}$ to D_2 blockade may be crucial to the clinical profile of atypical antipsychotics (Kapur & Remington, 1996).

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