The unrepresentative nature of the pre-senile vascular dementia group is acknowledged by the authors, and patients with mixed Alzheimer's and vascular pathology are also likely to be included in this vascular category. To date it is unclear as to the degree to which the two conditions coexist. As it is apparent that the Alzheimer's disease group may also be unrepresentative, the question begs to be asked, what groups are actually being compared? The overall suggestion that pre-senile Alzheimer's disease and vascular dementia have a similar prognosis needs to be taken in the context of these limitations and highlights the need for neuropathological studies in this area.

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Snowden, J. S., Neary, D. & Mann, D. M. A. (1996) Fronto-temporal lobar degeneration. In *Fronto-Temporal Dementia, Progressive Aphasia, Semantic Dementia*, pp. 1–227. Edinburgh: Churchill Livingstone.

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Psychotherapy and developmental disability

I welcome Holmes' (2000) editorial outlining the application of psychotherapies in psychiatry and across the sub-specialities. He advocates the inclusion of the broad range of psychotherapeutic treatments in all psychiatric practice and the development of a research and evidence base for the work. It is of concern, however, that in spite of his reference to learning disability as a psychiatric speciality in his introduction, he has effectively excluded people with learning disability by failing to include those with a developmental disability in his list of those benefiting from psychotherapeutic techniques.

On account of the neglect of developmental disability by psychotherapy, the Institute for Disability and Psychotherapy has been founded in order to provide, train in and research effective treatments for people with developmental disability and thus include them in health care. Each method of therapy cited in the editorial from analytic to family therapy is relevant and applicable to the patient group in my practice. Common themes in the experience of their lives are abuse and rejection (Sinason, 1992). The behavioural and psychological manifestations and the effects on personality of these problems in a person with developmental disability have the potential to give invaluable insights into the development of personality and the treatment of personality disorders in the population without developmental disability.

I hope that practitioners of the psychotherapies will have the courage to embrace all that working with people with developmental disability has to offer and to include rather than further compound the exclusion of people from the provision of effective care and treatment.

Holmes, J. (2000) Fitting the biopsychosocial jigsaw together. *British Journal of Psychiatry*, **177**, 93–94.

Sinason, V. (1992) *Mental Handicap and the Human Condition*. London: Free Association Books.

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Ethnic differences in forensic hospitalisation

It is sobering to note that Coid *et al* (2000) have once again found that variations in compulsory hospitalisation cannot be entirely attributed to racial bias, as some would lead us to believe. This was a large multi-centre study that did not limit itself to inner-city areas. The authors must be congratulated on their courage in challenging a popular and attractive myth and at the same time suggesting that services should be culture-sensitive.

Regarding their question of whether (predominantly White) forensic psychiatrists actively select White people with personality disorders as more suitable for treatments such as psychotherapy in secure setting, the answer may lie in the fact that maybe White people do have a greater chance of having a personality disorder (and thus meriting treatment) than the Black or Asian population. There is a study currently taking place at the Institute of Psychiatry and Broadmoor Hospital which is looking at Black patients with personality disorders, and the results should be most interesting. The Asian people in this study show a less than expected degree of morbidity, personality disorder, substance use and previous conviction, in spite of sharing the same socio-economic disadvantage, which is consistent with current knowledge. To paraphrase Freud, maybe a cigar is just a cigar.

Coid, J., Kahtan, N., Gault, S., *et al* (2000) Ethnic differences in admissions to secure forensic psychiatry services. *British Journal of Psychiatry*, **177**, 241–247.

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Stigmatisation: classifying drug and alcohol misuse as mental illness

Crisp *et al*'s (2000) article aims "to determine opinions of the British adult population concerning those with mental illnesses as baseline data for a campaign to combat stigmatisation". Specifically, the authors go on to list the disorders investigated: severe depression, panic attacks, schizophrenia, dementia, eating disorders, alcoholism and drug addiction.

I was surprised by the way in which alcoholism and drug addiction were grouped under the label of mental illness as if this was a commonly accepted truth within the scientific community.

The literature on drug and alcohol use and addiction suggests that these phenomena have to be seen as a complex interaction between a variety of factors, including psychosocial ones (McMurran, 1994). Similarly, views on drug use (and also mental illness) may change over time and are also the result of socio-political and historical contexts (Foucault, 1967; Levine, 1979). Treating drug and alcohol addiction as mental illness is an indication of the way mental illnesses are currently defined by the American Psychiatric Association (Cooksey & Brown, 1998) and should perhaps not be accepted all too readily as truth.

I recognise the psychiatric community's need to categorise mental illnesses. However, by classifying drug and alcohol users as suffering from mental illness, a

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position for which no evidence is given, this research, however well intended, may be stigmatising in itself.

Cooksey, E. C. & Brown, P. (1998) Spinning on its axes: DSM and the social construction of psychiatric diagnosis. *International Journal of Health Services*, **28**, 525–554.

Crisp, A. H., Gelder, M. G., Rix, S., *et al* (2000) Stigmatisation of people with mental illnesses. *British Journal of Psychiatry*, **177**, 4–7.

Foucault, M. (1967) *Madness and Civilisation: A History of Insanity in the Age of Reason*. London: Tavistock.

Levine, H. G. (1979) The discovery of addiction: changing conceptions of habitual drunkenness in America. *Journal of Studies on Alcohol*, **15**, 493–506.

McMurran, M. (1994) *The Psychology of Addiction*. London: Taylor and Francis.

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Olanzapine and pancreatitis

Two patients with chronic schizophrenia were admitted with acute pancreatitis. Olanzapine was used by both patients and may represent a further example of drug-induced pancreatitis. Olanzapine is a thienobenzodiazepine similar in structure to clozapine; clozapine has been associated with the induction of acute pancreatitis (Frankenburg & Kando, 1992; Gatto *et al*, 1998).

A 34-year-old man with chronic schizophrenia was admitted with abdominal pain. Two years previously he had had acute pancreatitis, but continued to drink alcohol (8 units/day). The patient was taking 20 mg olanzapine daily. Glasgow criteria and computed tomography classified the episode as severe pancreatitis. The patient developed respiratory failure and required artificial ventilation for 11 days. A full recovery was made.

A 29-year-old man with chronic schizophrenia was admitted with abdominal pain. He had a past history of acute pancreatitis, having had an attack 8 years previously. The patient drank at least 15 units of alcohol per week. Regular medication was sulpiride and olanzapine 20 mg daily. Diagnosis was made by computed tomography. The patient made an uncomplicated recovery. The same patient was readmitted 4 months later with another attack of acute pancreatitis – he had continued to take olanzapine and to drink alcohol.

Although both patients were regular consumers of alcohol, a known risk factor for the development of acute pancreatitis, we felt it unusual that two patients on olanzapine should present within such a short period of time. Clozapine has been associated with pancreatitis and has a similar structure to olanzapine; one might therefore suspect that olanzapine might cause pancreatitis. The evidence for olanzapine inducing pancreatitis is strengthened by the fact that in one of the patients a further episode occurred after rechallenge with the drug. It is possible that olanzapine acts in a synergistic fashion with alcohol in the pathogenesis of the disease. We propose that olanzapine should be used with caution with patients who drink alcohol on a regular basis or who have a previous history of pancreatitis.

Frankenburg, F. & Kando, J. (1992) Eosinophilia, clozapine and pancreatitis. *Lancet*, **340**, 251.

Gatto, E., Castronuovo, A. & Uribe Roca, M. (1998) Clozapine and pancreatitis. *Clinical Neuropharmacology*, **21**, 203.

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Antidepressant augmentation with low-dose olanzapine in obsessive–compulsive disorder

We read with interest the article on obsessive–compulsive disorder (OCD) and delusions, by O'Dwyer & Mark (2000). The suggestion of a continuum of pathological beliefs in OCD from 'none' to 'delusional intensity' suggests the possibility of another continuum of mental disorders from OCD to psychosis.

The authors presented five cases of OCD associated with delusional beliefs. They concluded that these patients are best considered within an OCD management plan. Moreover, they do not recommend use of long-term antipsychotic medication since they consider that such patients are unlikely to respond. However, none of the five patients they reported upon was treated with low doses of an atypical antipsychotic in order to augment the action of serotonin-specific antidepressants, the preferred pharmacotherapy in OCD.

Recently, we have successfully treated a man in his 50s who presented with a 7-year history of typical OCD. His problem was suitable for treatment using exposure and ritual prevention, combined with an antidepressant. He was referred for behavioural psychotherapy at the specialist unit based at the Maudsley Hospital. Although motivated to try this treatment he later found it too difficult to continue with this approach. He therefore remained under the care of his general practitioner (GP) who treated him with fluoxetine 20 mg daily, on which he had only a very slight improvement in his symptoms. When we saw him at the request of his GP he was asking for relief from anxiety symptoms. Small doses of thioridazine produced unacceptable side-effects, so olanzapine 2.5 mg was substituted. After approximately 4 weeks his symptoms were almost completely gone, and at a follow-up appointment he stated that for the first time after 7 years of rituals and obsessions, he felt 90% better.

Initially, our choice of olanzapine was determined by the patient's need for anxiolysis. However, there is emerging evidence that olanzapine may augment the action of fluoxetine in the treatment of individuals with OCD (Weiss *et al*, 1999). Another possibility is that olanzapine was having a direct action on psychotic phenomena. Whatever the case, we would suggest that contrary to the recommendations for management offered by O'Dwyer & Marks (2000) some treatment-resistant cases of OCD might respond to a therapeutic trial with low doses of an atypical antipsychotic in addition to a serotonin-specific antidepressant as usually recommended.

O'Dwyer, A. M. & Marks, I. (2000) Obsessive–compulsive disorder and delusions revisited. *British Journal of Psychiatry*, **176**, 281–284.

Weiss, E. L., Potenza, M. N., McDougle, C. J., *et al* (1999) Olanzapine addition in obsessive–compulsive disorder refractory to selective serotonin reuptake inhibitors: an open-label case series. *Journal of Clinical Psychiatry*, **60**, 524–527.

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Reboxetine-induced spontaneous ejaculation

Delayed or absent ejaculation is a common side-effect of antidepressant drugs. Reboxetine, a selective noradrenaline reuptake inhibitor, is known for its lack of sexual side-effects. We report a case of reboxetine-induced spontaneous ejaculation.

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