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Effects of schizophrenia on patients' relatives

Tennakoon *et al* (2000) stated that their study is 'one of the first' to investigate burden among caregivers of people with first-episode psychosis. Their paper was published 13 years after our paper which covered much the same ground (The Scottish Schizophrenia Research Group, 1987) and which they did not mention.

We found, using the General Health Questionnaire (GHQ), that 24 (77%) of 31 main caregivers were categorised as 'psychiatric cases'; this compares with 12% of caregivers in the Tennakoon *et al* study, which also used the GHQ. However, the relatives in our study were interviewed during the first week of the patients' first admission to hospital, and before the patients received antipsychotic medication. Tennakoon *et al*'s patients could have been ill for up to 2 years and received up to 12 weeks of antipsychotic drugs – hardly 'first-episode'.

In our 5-year follow-up study (The Scottish Schizophrenia Research Group, 1992), 14 of the 19 relatives who were still living with the patient were reassessed; 6 (43%) were still categorised as 'cases', using the GHQ. We concluded that a patient's illness had a considerable and continuing effect on his or her relatives.

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Over-representation of Black people in secure psychiatric facilities

I read with great interest Lelliott *et al*'s (2001) survey of patients from an inner-London health authority in medium secure psychiatric care. In particular, the authors set out to compare Black and White patients and found statistical differences which they dismiss. The scores on the Health of the Nation Outcome Scales and compound variables of clinical behaviour and social function differed between Black and White patients, but Lelliott *et al* comment that these may not be clinically significant.

If statistically significant findings on scales used in the study need not be explained, then the instruments cannot be considered valid to address the third aim of the study, to compare Black and White patients. Black patients were significantly less morbid on a number of clinical, social and behavioural variables, including affective symptoms, activities of daily living, problems with living skills, relationship problems and other mental or behavioural problems of self-harm and overactive and aggressive behaviour. Why should Black patients with less severe psychopathology or aggressive behaviour continue to find themselves in medium secure units?

There are two possible explanations. One is non-engagement with treatment options in less secure environments. It is known that Black patients are more likely to abscond from in-patient units (Falkowski *et al*, 1990) and that they are increasingly dissatisfied with each consecutive in-patient admission (Parkman *et al*, 1997).

An alternative explanation is that Black patients are perceived to be more dangerous despite lower ratings of psychopathology (Cope, 1990; Lewis *et al*, 1990). Lelliott *et al* unfortunately dismiss important findings as clinically insignificant. These very findings warrant further research and exploration and such work may well deliver a better understanding of why Black people are over-represented in secure psychiatric facilities.

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Evidence-based psychiatry

Lawrie *et al* (2001) have touched upon the core problem of evidence-based psychiatry by raising the most relevant questions. Although the evidence-based medicine movement began in 1992 (Sackett *et al*, 2000), it was not until the Royal College of Psychiatrists introduced a Critical Review Paper to the MRCPsych Part II examination in 1999 that clinicians suddenly realised the problems of not knowing enough about critical appraisal of scientific papers.

Brown & Wilkinson (2000) assert, "Psychiatrists should be able to evaluate published literature both in terms of its scientific validity and its clinical relevance". Why – to be able to practise evidence-based psychiatry, or to help trainees pass their exam? In a Scottish survey Lawrie *et al* (2000) discovered that senior psychiatrists found the time required to search and appraise the literature as the greatest barrier to practising evidence-based psychiatry. Would they be able to practise better if they had enough time, for instance 60 minutes per day? I do not know how to search for the best evidence to answer this question. I have recently read the recommended books (Brown & Wilkinson, 2000; Sackett *et al*, 2000), and I have also attended a few evidence-based medicine workshops. When my patients and trainees ask real-life clinical questions, I often get lost. Is this the beginning of my ageing-related cognitive impairment? Or is it just because I am such a busy clinician that I do not have time and need to take evidence-based psychiatry seriously? But how can we help our trainees? What about our own revalidation? We cannot really fudge the issue any more if we want to remain effective trainers.

Lawrie *et al* (2001) have raised the question, 'Whose responsibility?' Clinicians intimidated by the practical concepts of evidence-based psychiatry need to respond by expressing their difficulties and asking for time and resources to guide them through its complexities. Easy access to summaries of evidence may be the short-term solution, but the science of evidence-based psychiatry has to be mastered to continue practising the art of medicine. It's *our* responsibility.

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Lawrie, S. M., Scott, A. I. F. & Sharpe, M. C. (2000) Evidence-based psychiatry – do psychiatrists want it and can they do it? *Health Bulletin*, **58**, 25–33.

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Reintroduction of clozapine after diagnosis of lymphoma

The atypical antipsychotic clozapine has been shown to be of value in some patients with treatment-resistant schizophrenia. The drug is now only used with careful blood monitoring after fatalities were noted in the early 1970s when the drug was initially released. Alvir *et al* (1993) estimate the incidence of agranulocytosis to be about 0.9% at 1 year. The following case demonstrates that a patient may suffer blood dyscrasias for reasons other than the known effects of clozapine and that the drug can be successfully reintroduced with a coexistent haematological malignancy.

A patient with a history of treatment-resistant schizophrenia was started on clozapine. After several months she developed asymptomatic agranulocytosis. On admission, investigations were normal apart from a bone marrow biopsy which showed agranulocytosis and mild myeloblastic changes attributed to an acute drug effect. Clozapine was ceased and short-term treatment with granulocyte colony-stimulating factor appeared to be successful.

The patient's mental state deteriorated after treatment with chlorpromazine and quetiapine. During her subsequent psychiatric admission, fevers were noted and a further general hospital admission was arranged. She was found to have severe hypercalcaemia and hyperphosphataemia and reported bone pain. Bone marrow aspirate revealed a diffuse large B-cell lymphoma, which was treated with intensive combination chemotherapy over three cycles.

The patient was initially managed with haloperidol and diazepam. Relatively large doses of these medications were used to provide sedation during the initial phases of chemotherapy. After discussion with the patient, her relatives and the treating haematology team, it was decided to reintroduce clozapine seeking better antipsychotic control. The drug was restored with good effect and continued, despite very significant neutropenia secondary to the chemotherapy.

The case illustrates that clozapine can be ceased because of suspicions that it has lead to agranulocytosis while an underlying more sinister cause is not immediately detected. Subsequently, the drug was reintroduced with good antipsychotic effect in a patient who was severely medically ill.

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Stigma and ineffective legislation

Further to Haightat (2001), I would like to point out that the impact of stigmatisation is what it was when Goffman was developing his thoughts on stigma.

The impact of the the Human Genome Project potentially reveals boundless information that is stigmatising to both individual and family, in terms of employment, education and insurance. Although Haightat refers to legislation in this area to prevent such discrimination, existing law provides little confidence in these burgeoning areas. The UK Disability Discrimination Act 1995, specifically referring to physical or

mental impairment, provides little but deficiencies in the setting of discrimination against a propensity of developing a disorder in the future. Discrimination against such individuals would not be deemed unlawful. Amendments regarding this issue were discussed in both Houses of Parliament but not implemented (Nuffield Council on Bioethics, 1998). In contrast, a number of states in the USA have prohibited the use of information about employees' genetic characteristics by employers (Yesley, 1997).

In insurance, clients who have undergone genetic tests will be required to inform insurance companies of these. Proposals to avoid the unfair use of genetic information by insurance companies were announced in November 1998. Such a scheme relies on a voluntary rather than legal framework between the Department of Trade and Industry and the Human Genetics Advisory Committee (Clarke, 1995). This may do little to allay fears that an essentially profit-making business is being expected voluntarily to operate an ethical code of practice.

We may find that genetics provides a potential source of stigmatisation. As yet, the UK has few legal safeguards in place to protect individuals, who increasingly will have to manage this information carefully, rather as the 'discreditable' Goffman wrote of. Rather than legislation providing 'institutional support', the present situation serves only to propagate, in the public eye, a vision of a 'genetic underclass' (Clarke, 1995). An underclass where the stigmatising scars are invisible but their devastating effects on individual freedom are all too apparent.

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