Correspondence

EDITED BY KHALIDA ISMAIL

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Problem substance use and schizophrenia

McCreaidie et al. (2002) report on the problem of use of drugs and alcohol by people with schizophrenia. This is an excellent study as the pattern of use by patients is compared with controls from the general population, although the findings — that problem use is greater among patients — are unsurprising. It is impressive that the study included tobacco use, often disregarded as a ‘problem’ drug despite the obvious financial implications for patients surviving on state benefits. Previous studies quoted in the paper indicate that patients with schizophrenia often have been smoking for many years prior to the onset of the illness.

We are very interested to know whether the data collected for the study show that particular groups of patients appear to be more at risk from problem substance use, in order to focus efforts on helping them. Our experience is that admission to a psychiatric ward leads to increased tobacco use, and patients who have given up smoking recommence and continue smoking post-discharge, despite anti-smoking strategies. Also, we would like to know whether the study shows, or the authors know of, cultures that may be at lesser risk for developing problem use, accepting that numbers of ethnic minorities in the study sample may be small.


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Author’s reply: Bates & Rutherford raise some interesting points. I am impressed by their experience that admission to a psychiatric ward leads to increased tobacco use; this is certainly worthy of more-detailed study.

Betel use and schizophrenia

The Scottish Comorbidity Study Group has highlighted again the problem of greater use of drugs and alcohol, and especially tobacco, among patients with schizophrenia (McCreaidie et al., 2002). An underrecognised comorbidity, however, especially in developing countries, is that of chewing betel nut (Areca catechu), along with the betel leaf (Piper betle) and lime.

In a preliminary study conducted in the North Colombo Teaching Hospital, Sri Lanka, we observed that a higher proportion of patients with schizophrenia chewed betel compared with control subjects. The frequency of chewing betel was also higher among the patients with schizophrenia. A recent study from Micronesia (Sullivan et al., 2000) has shown that betel chewing may in fact have a beneficial effect on patients with schizophrenia in terms of reducing both positive and negative symptoms. They postulate that the muscarinic agonist action of the betel nut alkaloid, arecoline, may provide an explanation.

However, betel chewing is an important risk factor for oro-pharyngeal carcinoma, and contributes significantly to oral health-related morbidity and mortality (Trivedy et al., 2002). Thus, the dual diagnosis of schizophrenia and betel chewing should not be missed, and services to address this problem should receive priority in many developing countries.


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ECT and old age psychiatry

The editorial on electroconvulsive therapy (ECT) by Eranti & McLaughlin (2003) describes the current status of ECT as a treatment option. They have noted that the use of ECT is declining and also highlight that in a study from Edinburgh (Glen & Scott, 1999) a reduction in the number of recipients aged 18–65 was noted. While discussing the future of the ECT clinic, they raise concerns about how this reduced use could lessen clinical interest and how this could, in turn, affect future psychiatric trainees with respect to obtaining experience in ECT. They rightly highlight the need for this treatment option to be readily available for our patient group.

In this context, I want to report some of the findings from a study my colleagues and I presented as a poster at the annual meeting of the Royal College of Psychiatrists in Edinburgh in July 2000. The study looked at trends in ECT usage in the busy ECT clinic at the Royal London (St Clement’s) Hospital. It was a retrospective, chart-based study, covering a 3-year period during 1996–1999. Demographic and clinical data, which included response to ECT, were noted. There was a reduction in the number of patients who received ECT from 25 (171 total ECT episodes) in the year 1996/1997 to 12 (113 ECT episodes) in the year 1998/1999. Of the patients who received ECT, 70% were women and about 65% of the sample were aged above 60 years. A good response was noted in 45% of patients and, of this group, 70% were aged more than 60 years. The most common indication was depression and most of the findings of the practice of
ECT at the unit were in keeping with national trends reported by the Department of Health (1999). Over this 3-year period, consultant groups in the unit remained largely unchanged.

Concluding from this study, I feel that ECT is more commonly used in treating older people with depression. Availability of newer antidepressants and other treatment modalities, as highlighted by Eranti & McLoughlin (2003), could be some of the reasons why there is a decline in the number of patients under 65 who receive ECT. Furthermore, the limited response to ECT in the subjects of our study could be due to the fact that these patients had been treatment-resistant. On the other hand, in the case of older people suffering from severe depression, there are other factors that tilt the treatment options towards ECT. Factors such as physical frailty, propensity to develop side-effects from antidepressants, and the serious effects of dehydration and weight loss (as a result of severe depression) make it imperative that depression is controlled rapidly.

I feel that in the future, it will be old age psychiatrists who will be using ECT more commonly as a treatment option for depression. Old age psychiatrists could take a leading role in ensuring that psychiatric trainees have the opportunity to obtain experience in ECT. The effective (albeit reduced) use of ECT resulting in good clinical outcomes will ensure that clinical interest in this treatment modality is maintained.


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Lithium augmentation in treatment-refractory unipolar depression

Stimpson et al (2002) have taken an ‘all or nothing’ approach to evaluating randomised controlled trials (RCTs) for their systematic review. Their rigorous procedures eliminated over 98% of the 919 RCTs considered (although we note that the flow chart in Fig. 1 appears to ‘lose’ 166 of them without explanation). As a consequence, they have provided a matchless summary of the very best evidence about intervention for treatment-refractory unipolar depression but have left undescrived the very large quantity of remaining levels of evidence.

In 1999 Bauer and Dompfer identified 11 placebo-controlled studies of lithium augmentation. As always, the trials were of varying quality; nevertheless, they concluded (using the three studies of highest quality, two of which were used by Stimpson et al) that there is ‘firm evidence’ in favour of lithium as an augmentation strategy for treatment-refractory unipolar depression, with a number needed to treat of 3.7. They supported their conclusion by performing a separate analysis adding a further six studies (that used either lower doses or shorter duration of lithium augmentation) and found a similar, indeed slightly stronger, effect size (Bauer & Dompfer, 1999).

We note that there have been no studies of lithium augmentation against placebo for treatment-resistant unipolar depression that are of a suitable quality for a systematic review in the approximately 3-year period between the acceptance dates of the two papers cited above. We suggest that many clinicians now consider the weight of evidence (at many levels) supporting the use of lithium as an augmentation strategy for treatment-refractory unipolar depression sufficiently compelling. Thus, it is unusual for our service dedicated to treatment-resistant depression to receive referrals of patients not yet tried on lithium. Although further and better RCTs of lithium augmentation would be welcome (even Bauer & Dompfer identified only 234 subjects studied), many would feel that other questions now have more clinical salience. Pressing examples might include whether psychological treatments are effective in these patients, how they compare with lithium augmentation, and how olanzapine augmentation (for which a large body of evidence is emerging; see Dube et al, 2002) compares with both.


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Authors’ reply: According to Drs Lee and Cleare ‘many clinicians’ regard the current evidence for lithium augmentation in treatment-refractory depression as ‘compelling’. They are correct in repeating one of the principles of evidence-based medicine, that all levels of evidence need to be taken into account when making clinical decisions.

Previous systematic reviews of this area have included patients who have had ≤3 weeks’ treatment with an antidepressant or who have bipolar disorder. We do not think that many UK psychiatrists would consider lithium augmentation in unipolar depression that had not responded to an antidepressant for only 3 weeks. For patients with bipolar disorder, most UK psychiatrists, we think, would in any case be treating with lithium or another mood-stabiliser. Our inclusion criteria, which were set before the review started, were based therefore upon sensible and pragmatic clinical considerations.

We too were surprised and shocked by the lack of randomised evidence to support lithium augmentation; but it is also important to remember that lithium may well be effective, even though the evidence to support its use is extremely weak.

Lithium has a number of potentially serious side-effects, even at normal therapeutic doses (Bell et al, 1993). When we discuss the advantages and disadvantages of lithium with our patients we are unable to provide them with much more than clinical anecdote in its favour. We certainly have no idea from empirical research about the severity of depression for which lithium augmentation might be effective.

We have a collective responsibility to our patients to provide them with good-quality research evidence to justify the treatments we recommend. As a profession we need to address areas of uncertainty
such as this using well-designed RCTs that will inform clinical practice.

**Declaration of interest**

G.L. has received payments for lectures from the pharmaceutical industry.


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Getting closer to suicide prevention

We would like to offer a slightly different perspective from De Leo (2002) on the progress of suicide prevention. There is no argument against suicide representing a complex set of variables. The general method of science, however, is to analyse phenomena in order to find the most simple explanation – the principle formulated by William of Occam in the early 14th century. In the medical paradigm, death results from a disease process. Studying people with heart attacks led to the identification of atherosclerosis as the underlying disease process for the vast majority. Treating myocardial infarctions is important. The development of various approaches to prevention and treatment of atherosclerosis was, however, prevented more premature deaths from heart attacks. Why must one conclude that suicide is a more complicated medical problem than myocardial infarction?

A fundamental discovery was made in the late 1950s (Robins et al, 1959): the majority of suicides were committed by people with clinical depression. This finding has been replicated over and over again and we believe that many, like us, have concluded that this connection has been replicated enough to be proven. We have also presented evidence that suicides occur infrequently in people with depression taking antidepressant medication (Isacsson et al, 1994).

Thus, in spite of the ‘extreme complexity’ of the phenomenon of suicide, a simple and testable hypothesis can be stated: depression is a necessary cause of most suicides. Based on this proposition, it has been suggested that effective suicide prevention must focus on improving identification and treatment of depression in the population (Isacsson, 2000). When we look at the declining suicide rates over the past decade or so, we see a great deal of support for that theory. Since the introduction of the new generation of antidepressants during the past 10–15 years, the use of antidepressants has increased up to 3-fold. Concurrently, suicide rates have decreased considerably in many Western countries (e.g. Joyce, 2001). It appears to us that we are getting closer to suicide prevention.

We believe that a lack of focus on depression as the basic disease leading to suicide is most likely the reason why the current decline in suicide rates ‘seems reasonably unrelated to the existence of any national plan’.

**Declaration of interest**

Both authors have delivered lectures at scientific meetings sponsored by pharmaceutical companies.


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**Author’s reply:** There is little doubt that depression has a major role in suicide, being identifiable in approximately 50% of cases (Andersen et al, 2001). For this reason, depression is a target in all the national plans that I am aware of.

The role of depression in suicide has been well known since antiquity (Van Hooff, 2000) and this understanding has been largely responsible for the decline in a punitive attitude towards those exhibiting suicidal behaviour since the Enlightenment. Consequently, the ‘fundamental discovery’ at the end of the 1950s of the role of affective disorders in suicide was far from revolutionary. It is worth remembering that in the vast majority of cases, fortunately, depression does not culminate in suicide. The relative risk for suicide across the life span has been recently revised downwards (see, for example, Bostwick & Pankratz, 2000). In addition, a significant percentage of patients who die by suicide appear to have been adequately treated (25% in the experience of Andersen et al, 2001). A World Health Organization (1998) technical report has pointed out that optimal treatment of clinical depression would have little impact on global suicide rates, leaving the field open to speculations around more powerful factors in suicide prevention. In any case, the ‘medical paradigm’ is, in my view, only one of many possible perspectives, and needs to be integrated with other disciplines. Clearly, it is not the different prevalence of depression among countries that helps to explain the enormous diversity in rates of suicide that I mentioned in my editorial. Religious, cultural and social factors play very relevant roles in suicidal behaviour. It is in this light that the World Health Organization has correctly endorsed an ecological model, to help both understand and prevent/intervene in suicidal behaviours.

I am aware that Isacsson and Rich, through their research, strongly support the role of the newer antidepressants in preventing suicide. But others are a bit more hesitant in accepting this hypothesis (see, for example, Van Praag, 2002), and maybe lithium has shown more consistent (and convincing) effects, so far, on suicidal behaviour (Tondo et al, 2001).

With regard to the comments about a possible overemphasis on the complexities of suicidal behaviour, I am afraid that the philosopher Albert Camus, if he came back to life, would die again on hearing that!


Concepts of social capital

McKenzie et al (2002) illustrate how emerging conceptions of social capital can help psychiatric researchers study links between social context and the prevalence, course and outcome of psychiatric conditions. Two further considerations deserve a place in this discussion. First, the premise that social capital is "a property of groups rather than of individuals" (McKenzie et al, 2002: p. 280) does not enjoy an unqualified consensus. Work by Princeton sociologist Alejandro Portes (1998) summarises the case against insisting that social capital be treated as a group attribute. A more individualist approach draws attention to the important distinction between the social relationships that allow a person to make claims on resources held by others and the resources themselves. A family's struggle to find a job for a recently hospitalised relative may be eased somewhat when they live in a community with trusting social relationships, but this effect is more limited in a resource-poor community. (For example, Portes (2000) found that alleged effects of social capital on the academic achievement of immigrant children in the USA are drastically reduced when proper controls are used for parental socio-economic status.)

Second, McKenzie et al note that high social capital may be found in bad groups, such as the Mafia, and in homogeneous groups that restrict the freedom of members or exclude outsiders and minorities. This analysis of negative consequences can be expanded by an individual-oriented discussion of a dilemma familiar to clinicians working with socially marginal populations. Individuals may indulge in apparently irrational spending sprees to buy food, drugs or alcohol for companions because these allow them to make future claims for reciprocity when times are lean (Dordick, 1997). The resulting obligations may make it difficult for even a highly motivated person to enter (or re-enter) the social mainstream because he or she is vulnerable to criticism for breaking ranks with compatriots (Bourgois, 1995) or to claims on cash resources saved to facilitate an exit (for eviction, a new apartment, etc.). Programmes serving these populations need to devise strategies to help patients manage this dynamic aspect of social capital, even as they focus on recovery.


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Author's reply: The problem with the emerging concept of social capital is that it is in danger of trying to be all things to all people. Dr Walkup is correct to point to the view of Portes and others that social capital can be individual. I do not think that this approach is particularly useful. Social capital is not a thing, it is a way of trying to describe a number of social processes. It is a theory that helps us understand what is happening in a society. Although there may be analogous processes occurring at group and individual levels, conceptualising them as the same thing is problematic.

Theories of causation argue that causes at different levels are often governed by different rules and need different methods of investigation. An example would be the effects of smoking on health. This can be investigated at a number of levels; there would be the cellular level (the effects of nicotine on the cell), the individual level (physical and psychological effects of smoking and addiction) and the group level (what increases smoking levels in one group compared with another).

One would not try to employ the concept of cellular biology to investigate groups of people and one would not try to use group or systems approaches to investigate the individual. Moreover, the factors that increase the level of smoking in a group may not be the same as those that increase an individual's risk of smoking-related disease.

Given that group social processes are likely to affect health in different ways from individual processes, it would not seem helpful to consider social capital as a single entity that works at both levels. A choice has to be made and the choice of the majority is to conceive of social capital as operating at an ecological or group level and to consider effects at an individual level as social networks.

Dr Walkup is correct to point to the differences between the social relationships that allow a person to call on resources, and the resources themselves. However, the theory of social capital as an ecological variable does allow for this. Bonding and bridging social capital describe factors at the community level, but the concept of vertical social capital attempts to describe the ability of a community to facilitate access to resources from those in power.

Clearly, in our individualised world our interventions tend towards helping people decrease their risk of illness and their risk of relapse, and improve their participation in the world. The exciting difference about ecological conceptualisations is that they are about how society decreases the risk of illness and relapse of its population and how society facilitates the participation of the individual. These approaches aim for the same outcome but they are not the same thing and will need different conceptualisations, investigations and interventions.

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Vulnerable individuals and the Human Rights Act

With reference to the recent editorial on the Human Rights Act and mental health legislation (Bindman et al, 2003), the 'steady trickle' of human rights cases rather than a flood is not surprising when considered in context of the history of UK human rights.
Since 1965 UK organisations and individuals have had the right to petition under the European Convention on Human Rights (ECHR). This was then made binding on the British Government when we joined the European Union in 1973, under Article 189. Since then, British courts have had to take into account the ECHR in their decisions and judgements. So since 1973 we have been subject to the influence of the ECHR, which is nearly identical to the Human Rights Act.

What is new in the Human Rights Act 1998? There are no new rights but, as Bindman et al stated, it is easier to pursue alleged injustices. However, a major difference is frequently overlooked – only a directly affected individual can pursue legal challenges. Under the ECHR anyone with sufficient interest (i.e. pressure groups or interest groups) could petition. In the Human Rights Act this has been limited to individual ‘victims’ only. Potentially, this leaves some vulnerable individuals, such as those with mental health problems or learning disabilities, disenfranchised under the Human Rights Act, having still to rely upon the ECHR to protect them.


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**One hundred years ago**

**London County Asylum, Claybury (Report for the year ended March 31st, 1902)**

The average number of patients resident during the year was 2431, comprising 1015 males and 1416 females. The admissions during the year amounted to 426 – viz., 131 males and 295 females. Of these 364 were first admissions. Dr. Robert Jones, the medical superintendent, states in his report that the general character of the admissions was unsatisfactory as regards prospect of recovery. 38 per cent. of the admissions were over 60 years of age and over 16 per cent. of the males were suffering from general paralysis. 14 per cent. of the males and 9 per cent. of the females were admitted suffering from alcoholic insanity, “although as a predisposing cause the percentage is probably higher.” It is interesting to notice, adds Dr. Jones, that the two classes which furnished the greatest number of male admissions were described as “clerks” and “persons of no occupation.” The number of patients discharged as recovered during the year amounted to 148, comprising 52 males and 96 females, or 6·1 per cent. of the average number resident. The deaths during the year amounted to 201, or 8·27 per cent. as calculated on the same basis. “Asylum dysentery attacked 40 males and 81 females, and was responsible for 21 deaths, or over 10 per cent. of the total deaths.” Death was due to cancer of the stomach in six cases, renal disease in seven cases, epilepsy in eight cases, pneumonia in 14 cases, senile decay in 15 cases, colitis in 21 cases, cardiac disease in 24 cases, pulmonary and other forms of tuberculosis in 25 cases, general paralysis of the insane in 50 cases, and other causes in the rest. Two patients who were pregnant upon admission were safely delivered. There has been, with the exception of colitis, no outbreak of zymotic disease during the year. The Commissioners in Lunacy state in their report that the wards were in excellent order, that the day-rooms were comfortable and cheerful, that the dormitories were clean and well aired, and that the medical case-books and records were very well kept. The sub-committee of management states in its report that owing to the drought the crops and farm produce showed a considerable falling off during the year. The sum of £9320 was spent during the year upon improvements, alterations, and repairs.

**REFERENCE**

Lancet, 6 December 1902, p. 1572.

Researched by Henry Rolin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey
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