

unipolar disorder developed dementia (MMSE < 24) compared with 3.4% of age-matched controls (Kessing, 1998; Kessing *et al*, 1999). Even within a younger sample of psychiatric patients (approximate mean age 50 years), Kessing *et al* (1999) reported that people with bipolar disorder had the highest risk of receiving a diagnosis of dementia, followed by those with unipolar affective disorder, schizophrenia and neuroses. Thus, if affective disorders do increase both the risk of dementia and the likelihood of receiving lithium treatment, then owing to the sampling method used by Dunn *et al* one could expect to find more lithium treatment among elderly people with dementia. Dunn *et al* discussed this alternative explanation of their findings as a 'reverse causation' possibility.

Dr Terao mentions the possible effects of lithium on GSK-3 beta. We recently investigated the effects of lithium on the transcriptional regulation of GSK-3 beta and found a significant reduction of its expression in primary cultures of rat hippocampal neurons as well as a reduction in regional intracerebral expression in lithium-treated adult rats and in leukocytes of elderly patients undergoing chronic lithium therapy for bipolar disorder (details available from the authors). These observations suggest a mechanism for GSK-3 beta inhibition by lithium, which may influence the formation of both amyloid plaques and neurofibrillary tangles, the two neuropathological hallmarks of Alzheimer's disease.

We think that it is important to investigate further the potential protective effect of lithium in Alzheimer's disease, as this could represent a low-cost universally available strategy to reduce the prevalence.

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Mortality and electroconvulsive therapy

Munk-Olsen *et al* (2007) reported that the mortality rate from natural causes was lower for patients undergoing electroconvulsive therapy (ECT) than for other psychiatric in-patients. The lower relative risk was particularly significant for mortality linked to respiratory disease (RR=0.67, 95% CI 0.55–0.95) and a trend was founded for cardiovascular disease (RR=0.85, 95% CI 0.70–1.03). The authors concluded that this decreased risk of mortality from natural causes is unlikely to be the result of a selection bias. They based this statement on: (a) the absence of absolute contraindications to ECT in the international guidelines; and (b) the concordant findings of previous studies.

At variance with this statement, clinical practice suggests that psychiatrists are generally reluctant to consider ECT in patients with medical illness, and are more likely to ask for the opinion of a colleague in such a case (e.g. anaesthetist, cardiologist) (Benbow & Shah, 2002). Thus, patients with severe medical illness could be less likely to be treated by ECT. Furthermore, identification of cardiovascular diseases or pulmonary disorders, as well as physical examination and standard laboratory tests are part of a systematic screening procedure before ECT. This practice improves the diagnosis and the treatment of medical comorbidities. Indeed, the absence of such preliminary medical examination led to a high level of cardiac complications after ECT in the past (Gerring & Shields, 1982).

Accordingly current guidelines emphasize the importance of identifying and carefully managing patients with risk factors before, during and after ECT, as well as assessing the risks associated with anaesthesia (National Institute for Clinical Excellence, 2003). Patients receiving ECT are therefore not representative of all psychiatric in-patients. The careful assessment and treatment of their physical comorbidities contrasts with the increased rate of untreated physical illness in psychiatric patients, mostly because of inadequate somatic care in psychiatric units (Rasanen *et al*, 2006). Therefore, the observed diminution of mortality from natural causes in patients with ECT is more likely to be related to appropriate medical assessment and treatment than to a direct effect of ECT on physical health.

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In an era that has seen ECT being opposed for political not clinical reasons, it was heartening to see an article on ECT addressing the very important issue of mortality. The study of Munk-Olsen *et al* (2007) is based on the Danish registry system which is acclaimed for its reliability, but certain issues need further clarification. It would have been relevant to know the total number of patients who received ECT and the total number of ECT treatments received by patients over the study period. Furthermore, the results could be better understood if information regarding physical comorbidity and the age of patients at the time of ECT had been provided. These variables can have a strong influence on mortality rates. In addition, as the study included only in-patients it is likely that the sample included patients who were severely ill. Also, the results show that inclusion of 'days since last ECT treatment' in the analysis causes the relative risk of mortality from natural causes of patients 'discharged within the past 8–30 days' to rise.

The relative risk of mortality from natural causes is also highest within 7 days of last ECT (RR=2.11), which is similar to the trend seen in deaths due to unnatural causes, especially suicide. Both these figures go against the conclusion of the authors that the mortality from natural causes is lower with ECT. It must also be noted that the relative risk of dying by suicide after

ECT is 1.20, which is not significant but which the authors refer to as 'a marginally significant trend', and 'significantly increased suicide rate'. The finding that the risk from suicide is highest in the first 7 days after discharge and ECT is based on a small sample ($n=6$). Although the authors concede that admission status and time since discharge are important confounders in the analysis of suicide in patients with affective disorders, the statistical analysis does not consider these factors when calculating the relative risk of suicide after ECT. The authors discuss in some length the lack of a selection bias of patients with poor physical health. However, it is likely that patients with very poor physical health are not given ECT and this introduces a selection bias. Also, given the bias that occurs as patients at high risk for suicide are given ECT preferentially, this calls into question the validity of the conclusions. Further, it would have been very useful if the authors could have compared the death rates with those in the general population. This study provides several good research questions which need to be pursued further.

Munk-Olsen, T., Laursen, T. M., Videbech, P., et al (2007) All-cause mortality among recipients of electroconvulsive therapy. Register-based cohort study. *British Journal of Psychiatry*, **190**, 435–439.

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Authors' reply: Both Le Strat & Gorwood and Bharadwaj & Grover comment on the finding of a decrease in mortality in ECT-treated patients. In Denmark, all psychiatric patients are given a thorough medical assessment prior to any somatic treatment. This is partly because of the well-known cardiac contraindications for the use of tricyclic antidepressants which were widely used during the study period from 1976 to 2000, as the selective serotonin reuptake inhibitors (SSRIs) were only available in the latter part of the period described. Furthermore, SSRIs were generally considered less effective than tricyclic antidepressants or ECT in patients with

severe depression. Accordingly, ECT was often used in patients with contraindications for tricyclic antidepressants. We are aware that this notion is at variance with several British guidelines (e.g. National Institute for Clinical Excellence, 2003) but it is in accordance with Danish and American Psychiatric Association guidelines, which state that the only contraindications to ECT are cerebral and other aneurysms. In Denmark, a preponderance of patients with medical illness is thus found among ECT-treated patients compared with those treated with tricyclic antidepressants and we therefore maintain our conclusion.

Drs Bharadwaj and Grover point out that admission status and time since discharge are important confounders. We fully agree and have hence adjusted for these variables in the analysis. The variables in Table 3 on risk of suicide in ECT recipients were mutually adjusted but this was not mentioned specifically in the footnote.

The number of patients dying by suicide in the first week after ECT discontinuation was small, and therefore our results should be interpreted with caution, as we mention in the discussion. Electroconvulsive therapy is often administered to patients who are assessed to be suicidal and we acknowledge that this could introduce selection bias (confounding by indication), which we also mention in our paper. These are the reasons why we concluded that: 'the increased suicide rate among ECT patients shortly after treatment is probably a result of bias' and we therefore disagree that the validity of the study is questionable regarding suicide rates after ECT.

A more in-depth description of the ECT patients can be found in a paper based on the same data (Munk-Olsen *et al*, 2006).

Munk-Olsen, T., Laursen, T. M., Videbech, P., et al (2006) Electroconvulsive therapy: predictors and trends in utilization from 1976 to 2000. *Journal of ECT*, **22**, 127–132.

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Measuring stigma

King *et al* (2007) frequently state that their stigma scale is measuring 'the stigma of mental illness' but, when closely scrutinised, it measures nothing other than stigmatisation perceived by users in out-patient, in-patient and crisis settings. There is no evidence that this is an objective assessment of stigmatisation. Users' perception of stigma is affected by their mental state, depression, persecutory delusions or hallucinations. These symptoms can help to exaggerate the estimate of social stigmatisation (including rejection and discrimination) and hence the assessment is by no means an accurate measure. Measurements of more objective perceptions of stigmatisation can only be obtained from users in remission.

The reported negative correlation between self-esteem and perceived stigma can be confounded by high rates of both low self-esteem (e.g. Axford & Jerrom, 1986; Barrowclough *et al*, 2003; Blairy *et al*, 2004) and persecutory ideation and depressive cognition, including 'self-stigmatisation' in people with mental illness. Indeed, low self-esteem is a common symptom in psychiatric conditions such as depressive disorders, in which people can perceive more rejection and discrimination than warranted. Overemphasis on this correlation can divert attention from the fact that the correlation has to do more with people's mental state than objective level of social stigmatisation.

An instrument can only be called 'standardised' if it is shown to be both reliable and valid. This instrument is not validated and so cannot be called standardised, on the basis of mere test-retest reliability. The correlation between the stigma scale and self-esteem scale is not an indication of validity of the instrument and although King *et al* admit this, they end up referring to their instrument as 'standardised' and to the correlation as 'concurrent validity'.

A wide range of people with diverging diagnoses and mental states were recruited by King *et al* but there was no randomisation and no exclusion criteria. Even the 'perceived stigmatisation' cannot be attributed to a particular category of patients with a given diagnosis, or at least to psychiatric users in general, owing to lack of randomisation and inclusion of arbitrary proportions of participants with different diagnoses. This is likely to cause problems