

make the grave error of extrapolating from community estimates to the special population of the elderly in hostels and nursing homes.

Henderson, S., Andrews, G. & Hall, W. (2000)

Australia's mental health: an overview of the General Population Survey. *Australian and New Zealand Journal of Psychiatry*, **34**, 197–205.

Jorm, A. F. (2000) Does old age reduce the risk of anxiety and depression? A review of epidemiological studies across the adult life span. *Psychological Medicine*, **30**, 11–22.

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Antidepressants and suicide risk

Donovan *et al* (2000) make interesting points about deliberate self-harm (DSH) and antidepressant drugs, but their report as written is open to grave misinterpretation. Indeed, a reporter brought the article to my attention wanting to know why selective serotonin reuptake inhibitors (SSRIs) increased suicide risk relative to tricyclic antidepressants (TCAs).

A key problem with this cross-sectional, naturalistic study of DSH and antidepressant medications at emergency department presentation is that patients were not diagnosed. The authors write as if antidepressant medications are almost invariably prescribed to treat depression, yet clearly this is not always true. Even within mood disorders, patients may differ greatly in suicide risk. The authors found fragmentary evidence that patients on SSRIs may have been relatively treatment-resistant.

Moreover, SSRIs are prescribed for a growing spectrum of psychiatric illnesses beyond depression. The authors hint at the multiplicity of indications, mentioning enuresis as an indication (presumably for TCAs). Astoundingly, however, they never mention borderline personality disorder (BPD). Patients with BPD, known for their frequent parasuicidal gestures (Davis *et al*, 1999), are more likely to receive SSRIs than TCAs: partly because of their safety in overdose, partly for their benefit for impulsivity independent of mood disorder. Hence BPD and other patients at higher risk for DSH may have received SSRIs rather than TCAs. The authors mention this briefly (“... the question of whether patients prescribed TCAs were similar in terms of DSH risk to

those prescribed SSRIs”, p. 553) but fail to emphasise how crucial this issue is. (Neither do they mention substance misuse, a further risk factor for self-destructive behaviour.) Given this likely diagnostic and prescriptive imbalance, it is unsurprising that more suicidal patients presenting at emergency departments were taking SSRIs.

In summary, without knowing that equivalent patient populations were receiving the two classes of medications, we cannot compare their effect on suicide risk.

Davis, T., Gunderson, J. G. & Myers, M. (1999)

Borderline personality disorder. In *The Harvard Medical School Guide to Suicide Assessment and Intervention* (ed. D. G. Jacobs), pp. 311–331. San Francisco, CA: Jossey-Bass.

Donovan, S., Clayton, A., Beeharry, M., et al (2000)

Deliberate self-harm and antidepressant drugs. Investigation of a possible link. *British Journal of Psychiatry*, **177**, 551–556.

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Cognitive therapy and social functioning in chronic depression

We clinicians constantly encounter patients with major depression in partial remission. They are no longer acutely depressed but continue to present with substantial functional impairment (Paykel *et al*, 1995). For treatment-resistant depression, only one pharmacological intervention can be recommended today with reasonable evidence, namely lithium augmentation (Austin *et al*, 1991; Aronson *et al*, 1996), but this may not be the answer for those with low-grade residual depression.

Scott *et al* (2000) demonstrated that cognitive therapy can help these people. Critically appraising their article in our evidence-based psychiatry case conference, however, it was very difficult for us to appreciate the substantive significance of this improvement, because only means and standard deviations of scores on the Social Adjustment Scale were reported. Analyses based on these data can show whether or not the treatment is better than the control condition, but cannot show how much better it is – a crucial piece of information for both patients and clinicians. We therefore resorted to the normative data for this scale (Bothwell & Weissman, 1977).

Calculation based on the means and standard deviations under the assumption of a normal distribution showed that, at week 20, 68% of patients with residual depression reached the 95% range of the control subjects when treated with clinical management plus cognitive therapy, whereas only 45% did so when treated with clinical management only. This translates into a ‘number needed to treat’ of 4.4 (95% CI 2.6–12.6).

This is an impressive figure. By adding 16 sessions of cognitive therapy to usual care, we can achieve social remission in one additional patient out of four, compared with continued standard care only. The original authors had concluded, “In patients showing only partial response to antidepressants, the addition of CT produced modest improvement in social and psychological functioning”. We find that the improvement was more than modest and would be clinically meaningful.

Aronson, R., Offman, H. J., Joffe, R. T., et al (1996)

Triiodothyronine augmentation in the treatment of refractory depression: a meta-analysis. *Archives of General Psychiatry*, **53**, 842–848.

Austin, M.-P. V., Souza, F. G. M. & Goodwin, G. M. (1991)

Lithium augmentation in antidepressant-resistant patients. A quantitative analysis. *British Journal of Psychiatry*, **159**, 510–514.

Bothwell, S. & Weissman, M. M. (1977)

Social impairments four years after an acute depressive episode. *American Journal of Orthopsychiatry*, **47**, 231–237.

Paykel, E. S., Ramana, R., Cooper, Z., et al (1995)

Residual symptoms after partial remission: an important outcome in depression. *Psychological Medicine*, **25**, 1171–1180.

Scott, J., Teasdale, J. D., Paykel, E. S., et al (2000)

Effects of cognitive therapy on psychological symptoms and social functioning in residual depression. *British Journal of Psychiatry*, **177**, 440–446.

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Author's reply: I am a strong advocate of the use of cognitive therapy in chronic and residual depressive disorders. I am therefore the last to disagree with the comments of Ito and colleagues that there is real benefit in providing psychosocial treatments to individuals with residual depressive symptoms. My comment on social functioning was not meant to underestimate the benefits, but paid heed to two factors. First, although individuals who received cognitive therapy undoubtedly