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Is diazepam an antidepressant?

SIR: Like many other trainee psychiatrists, no doubt, we read the paper by Tiller *et al* (*Journal*, October 1989, **155**, 483–489) with much interest. The title, 'Is diazepam an antidepressant?', intrigued us. However, a careful reading of the article showed that the study was not originally intended to investigate the antidepressant activity of diazepam, but that of the antidepressant moclobemide. Diazepam was chosen as a placebo. Against predictions, the authors found that there was a significantly better improvement in the Hamilton Rating Scale for Depression (HRSD) scores in the diazepam group compared with the moclobemide group after one and four weeks of treatment. As a result, they concluded that diazepam was a better antidepressant than moclobemide in atypical depression.

There are two points here. Firstly, atypical depression is not a descriptive diagnostic concept in DSM–III and in ICD–9. The HRSD contains items rating not only for depression but for anxiety as well. The authors rightly pointed out that the patients might in fact be suffering from an anxiety state instead of depression. The rapid improvement in the HRSD scores (within one week) in the diazepam group is certainly more suggestive of an anxiety state responding to the anxiolytic properties of the benzodiazepine.

Secondly, and more importantly, we think that what this article really describes is a failed drug trial of moclobemide. The title seems to be an afterthought, given that the main focus was on moclobemide rather than diazepam. We wonder about the ethics of suggesting the use of diazepam as an antidepressant founded on these unclear results. Certainly trainees should not decide to start prescribing diazepam in depression on the basis of this curious study.

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SIR: I read the paper by Tiller *et al* (*Journal*, October 1989, **155**, 483–489) with a sense of *déjà vu*. Alprazolam, a 1,4-benzodiazepine like diazepam, was originally credited with antidepressant properties, but the methodology of most such studies is suspect (O'Shea, 1989). Later studies failed to support a primary role for alprazolam in the treatment of depression.

The major flaws in most such research are as follows. Firstly, the benzodiazepines often improve 'depression scores' faster than true antidepressants in the first weeks of a study, but the antidepressant then produces a longer-lasting and superior effect (O'Shea, 1989).

Secondly, the Hamilton Rating Scale for Depression (Hamilton, 1960) is not a diagnostic instrument. The effect mentioned above is due to anxiolysis, because up to 8 of the 21 items in this instrument measure anxiety (O'Shea, 1989).

Thirdly, the definitions of depression have been flawed. Studies which used neurovegetative signs (Goldberg *et al*, 1986) or reduced REM latencies (Rush *et al*, 1985) failed to demonstrate an antidepressant use for alprazolam.

For these reasons, as well as the potential for dependence, it may be premature to use diazepam as a replacement for antidepressants.

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