hallucinations. Almost half of our patients had sleep disturbance in contrast to 8–23% reported by Deutsch and Rovner (1991). There were many disturbances of biological function in our dementia patients, jeopardizing the patients and burdening the caregivers. However, methodological variations in these studies would warrant a cautious approach while comparing the reported BPSD symptoms across different setups and cultures.

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Risperidone and tardive dyskinesia

Dear Editor

In their longitudinal study of elderly psychotic inpatients treated with risperidone, Davidson *et al.* (2000) assessed the relative effect of this 'atypical' antipsychotic on the occurrence of tardive dyskinesia (TD) by comparing the incidence of TD observed in their sample with the incidence reported by Jeste *et al.* (1995) in their study of elderly psychiatric outpatients treated with conventional antipsychotics. Davidson *et al.* (2000) identified six new cases of TD during a 1-year follow-up period among 139 patients who were free of TD symptoms at baseline; thus, their estimated 1-year risk (cumulative incidence) was 6/139 = 4.3%. Since the estimated 1-year risk reported by Jeste *et al.* (1995) was 26%, the authors concluded that the risk of TD is much lower in patients treated with risperidone than in patients treated with conventional antipsychotics. Although there is evidence from other studies to support the relatively low risk of TD in users of atypical antipsychotics (Beasley *et al.*, 1999; Jeste *et al.*, 1999), the results of this study should not be used to quantify the relative effect of risperidone on TD risk. There are four reasons for our position.

First, Davidson *et al.* (2000) and Jeste *et al.* (1995) used different methods for detecting new cases of TD. While Jeste *et al.* used the abnormal involuntary movement scale (AIMS) (Guy, 1976), which is comprised of seven anatomical item scores, Davidson *et al.*, used five items from the extrapyramidal symptom rating scale (ESRS) (Chouinard *et al.*, 1980), which do not reflect distinct anatomical components. Given the diagnostic criteria used in these two studies, it is possible that the threshold for detecting new TD cases was higher in the Davidson *et al.* study; therefore, the estimated TD risk would be artificially lower.

Second, the 1-year risk estimate of 4.3% is biased downward (i.e. an underestimate of the true risk) because a large proportion of the subjects at risk in this study were not followed for at least a year. Although this bias was apparently not recognized by the authors, they also conducted a Kaplan–Meier (survival) analysis of TD incidence, which takes into consideration different durations of follow-up. According to the text (p. 511), the estimated 1-year risk of TD was $1-0.866=13.4\%$, which is appreciably larger than the 4.3% estimate reported in the abstract and discussion.

Third, the TD risk is estimated very imprecisely in this study because of the small number of incident cases observed. Unfortunately, we cannot assess the precision from Davidson *et al.*'s paper because they did not provide a confidence interval or enough information for the reader to compute a confidence interval around the risk estimate of 13.4%.

Fourth and most important, the populations in these two studies differed appreciably with respect to the distribution of at least one important TD risk factor: duration of previous antipsychotic exposure at baseline. While the median duration of such exposure was only 21 days in the Jeste *et al.* study, the median duration was many years in the Davidson *et al.* study (since the mean duration of their current hospitalization was 17 years; see Table 1). Because the incidence rate of TD is much higher in the first few years after starting antipsychotic treatment than in subsequent years (Morgenstern and Glazer, 1993; Glazer *et al.*, 1993), duration of exposure will strongly confound (bias) the TD-risk contrast between the two populations. Thus, the true difference in 1-year risk, controlling for confounding, should be much less than the difference reported by Davidson *et al.*

In summary, a study of risperidone users only should not be used to quantify the effect on TD incidence of risperidone relative to conventional antipsychotics. The major limitations of relying on a comparison group obtained from another study are methodologic differences in the detection of outcome events and confounding that cannot be controlled by the investigator.

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