by the statement that "Neuropsychological and neuroimaging studies have not shown robust differences between DLB and AD subjects". There is considerable evidence from published data to suggest that there is a clear difference in the neuropsychological profile between dementia with Lewy bodies (DLB) and Alzheimer's disease (AD) and that this could contribute to making the differential diagnosis.

The literature on the neuropsychological profile of DLB has been extensively reviewed by Salmon & Galasko (1996). They summarise earlier work by Hansen's group which showed greater deficits in attention, verbal fluency and visuospatial processing in nine pathologically confirmed Lewy body variant patients compared with 10 AD patients, as well as data from the Newcastle Group, using the CANTAB, a computerised battery of tests. In the latter study DLB patients were more impaired on delayed matching to sample, conditional learning paired associate tasks and spatial working memory. This difference in visuospatial ability was thought to reflect greater dysfunction of frontal lobe structures. The visuospatial differences have recently been shown to be of sufficient magnitude to be detected even by tests commonly used in clinical practice, e.g. on the CAMCOG (Walker *et al*, 1997*a*) and the Clock Face Test (Gnanalingham *et al*, 1997), suggesting those differences could be utilised to distinguish DLB from other dementias in a clinical setting.

As for neuroimaging studies, both perfusion studies (Albin *et al*, 1996) and dopamine receptor imaging (Walker *et al*, 1997*b*) have shown significant differences in the two conditions, suggesting that those might be a further aid to differentiate the two conditions.

Albin, R. L., Minoshima, M. D., D'Amato, B. S., *et al* (1996) Fluorodeoxyglucose positron emission tomography in diffuse Lewy body disease. *Neurology*, **47**, 462–466.

Gnanalingham, K. K., Byrne, E. J., Thornton, A., *et al* (1997) Motor and cognitive function in Lewy body dementia: comparison with Alzheimer's and Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry*, **62**, 243–252.

Miller, B. L. (1997) Clinical advances in degenerative dementias. *British Journal of Psychiatry*, **171**, 1–3.

Salmon, D. P. & Galasko, D. (1996) Neuropsychological aspects of Lewy body dementia. In *Dementia with Lewy Bodies* (eds R. Perry, I. McKeith & E. Perry), pp. 99–113. Cambridge: Cambridge University Press.

Walker, Z., Allen, R. L., Shergill, S. S., *et al* (1997*a*) Neuropsychological performance in Lewy body dementia and Alzheimer's disease. *British Journal of Psychiatry*, **170**, 156–158.

—, Costa, D. C., Jansen, A. G., *et al* (1997*b*) Dementia with Lewy bodies: a study of post synaptic dopaminergic receptors with iodine-123 iodobenzamide single-photon emission tomography. *European Journal of Nuclear Medicine*, **24**, 609–614.

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Tests of 'dissociation' and mood disorder

Sir: Nijenhuis *et al* (1997) maintain that what Fahy (1988) and Merskey (1992) regard as the misdiagnosis of some cases of bipolar disorder as dissociative conditions (particularly multiple personality disorder) permits the hypothesis that, if we are right, instruments measuring dissociative pathology should give high scores, both in patients with supposed dissociative disorders and in those with bipolar affective disorder. Their claim is invalid. Patients with bipolar disorder may be misdiagnosed in at least two ways. First, as in the double consciousness cases, the existing natural phenomena are simply misread for honest, but antiquated reasons. Second, patients with bipolar disorder may be educated into producing the desired states that are to be labelled dissociative. It was not to be expected that patients with bipolar disorder would match the dissociative disorder group on these scales unless the former had been indoctrinated. Nijenhuis *et al* have compared un-indoctrinated subjects with others whom they consider to be dissociative and have obtained a number of highly significant statistical results which are predictable, but not for the reason they suppose. They comparison that has been offered is worthless.

Fahy, T. A. (1988) The diagnosis of multiple personality disorder. A critical review. *British Journal of Psychiatry*, **153**, 597–606.

Merskey, H. (1992) The manufacture of personalities. The production of multiple personality disorder. *British Journal of Psychiatry*, **160**, 327–340.

Nijenhuis, E. R. S., Spinhoven, P., Van Dyck, R., *et al* (1997) Dissociative pathology discriminates between bipolar mood disorder and dissociative disorder (letter). *British Journal of Psychiatry*, **170**, 581.

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Large same-year effects: fact or artefact?

Sir: Kessler *et al* (1996) report inordinately large odds ratios for 'same-year' effects for major depressive disorder (MDD) and comorbid disorders (see Table 4). An examination of the estimation method indicates that the large odds are likely to be an artefact.

The authors used survival models to estimate time-lagged, same-year, and time-trend effects (Table 4). The three time-dependent covariates were: (a) same-year – z_1=1 if MDD and prior disorder occurred in the same year, 0 otherwise; (b) time-lagged – z_2=1 if prior disorder occurred one or more years prior to onset of MDD, 0 otherwise; (c) time-trend – x=number of years since onset of prior disorder.

The estimation of the odds ratio is similar to that of the Mantel–Haenszel approach with two-by-two tables defined at each event (MDD) time (Crowley, 1975, 1980). For z_1 this table would be:

		MDD	
		Yes	No
Other disorder and MDD in same year	Yes	A	B
	No	C	D

By definition, cell B in the table is always 0; no subject can be both 'no' for MDD and 'yes' for other disorder and MDD in the same year. This 'structural zero' inflates the odds ratio for the 'same-year' effect, which could account for the high odds ratios in Table 4.

An approach that avoids this artefact (although still problematic, as indicated below) would be to include two binary-time dependent variables: z_3=1 if time since prior disorder is less than one year, 0 otherwise; z_4=1 if time since prior disorder is greater than one year, 0 otherwise.

Variable z_3, which measures within-year effect, does not result in a structural zero. A subject may have a prior disorder for less than a year but not have MDD when evaluated at another subject's event time. In other words, whereas z_1 requires that the onsets of the two disorders *within an individual* occur in the same year, z_3 does not.

To illustrate the two approaches, a simulation study was run. The generated data consisted of 1000 observations from an exponential distribution. The prevalence of MDD and prior disorder were set at 20%. Distributions were chosen so that the MDD odds ratio for all years including

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same-year was 1.0. The simulation was repeated 20 times. The average same-year odds ratio for z_3 was 1.01 (s.d. 0.29), as would be expected. In contrast, the average same-year odds ratio for z_1 was 5.46 (s.d. 1.60), reflecting the spurious increase.

While the z_3 approach does not generate an artificially large odds ratio, it has no advantage over the one used by Kessler *et al*, with respect to the indeterminate temporal order between same-year events. In the US National Comorbidity Survey, as in other studies with similar diagnostic interviews, the time of onset of disorders was recorded as subjects' age *in years*, a method that results in ties. Survival analysis with time-dependent covariates deals with chronologically ordered events, with the dependent variable occurring after the independent variable(s). The approach using z_3 also assumes a time order where none can be determined. Indeed, no statistical approach can correct for the lack of information regarding which disorder preceded the other. An unbiased approach to modelling tied data is to censor cases with same-year onsets just prior to the year in which the two events occur.

Kessler, R. C., Nelson, C. B., McGonagle, K. A., *et al* (1996) Comorbidity of DSM-III-R major depressive disorder in the general population: results from the US National Comorbidity Survey. *British Journal of Psychiatry*, **168** (supplement 30), 17–30.

Crowley, J. (1975) *Estimation of Relative Risk in Survival Studies*. Department of Statistics University of Wisconsin, Technical Report #423.

— **(1980)** *Some Extensions of the Logrank Test*. Wisconsin Clinical Cancer Center, Biostatistics, Depts. of Human Oncology and Statistics, University of Wisconsin-Madison, Technical Report #10.

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Author's reply: Professors Peterson & Breslau are incorrect in thinking that the cross-sectional odds ratios reported by Kessler *et al* (1996) are artefactually inflated. Their confusion is due to a lack of clarity in our paper about the specification in Model 2. This model used the predictors described by Peterson & Breslau as z_3 and z_4, not z_1 and z_2. As Peterson & Breslau note, there is no bias in this approach. We regret the confusion and appreciate this opportunity for clarification.

Peterson & Breslau also stated that bias in estimating the effect of z_4 can be avoided by censoring same-year onset cases. This, too, is incorrect. The fact that none of the excluded person-years has a prior history of the predictor will lead to bias in the estimated effect of z_4. However, a slightly different approach will yield an unbiased estimate: to censor all person-years with a value of one on z_3.

It is noteworthy that the estimated effect of z_4 in this unbiased approach is equivalent to the estimate in our Model 2, as both methods compare z_4 person-years with the other non-z_3 person-years. The censoring method does this by excluding the z_3 person-years, while Model 2 does it by introducing a control variable for the z_3 person-years. We prefer Model 2 for two reasons. First, unlike the censoring method, it allows the magnitude of the lagged and cross-sectional odds ratio to be compared directly. Second, in multivariate models, where more than one time-varying predictor is considered at a time, it allows direct comparison of nested models, which is impossible in the censoring approach due to the fact that the number of excluded person-years varies with the number of predictors.

Finally, it might be useful to comment on the concern raised by Peterson & Breslau that the cross-sectional logit associated with z_3 does not have a clear causal interpretation. This is correct. In the absence of confounding variables, the cross-sectional association between a predictor and an outcome can be due either to an effect of predictor on outcome, an effect of outcome on predictor, or both. It is possible to use the method of instrumental variables to obtain separate estimates of these two effects if an appropriate instrument variable exists (Angrist *et al*, 1996). However, this method yields inconsistent estimates unless the instrument variable is strongly related to the outcome (Bound, *et al* 1995). This is seldom the case. As a result, it is usually preferable to estimate the cross-sectional effect of predictor on outcome using a recursive specification. This is what we did in our report. This coefficient can generally be interpreted as an upper-bound estimate of the short-term effect of predictor on outcome when one has reason to believe that the reciprocal effects between predictor and outcome have the same sign, making it a useful statistic despite the fact that it is biased.

Angrist, I. D., Imbens, G. W. & Rubin D. B. (1996) Identification of causal effects using instrumental variables. *Journal of the American Statistical Association*, **91**, 444–455.

Bound, J., Jaeger, D. A. & Baker R. M. (1995) Problems with instrumental variable estimation when the correlation between the instruments and the endogenous explanatory variable is weak. *Journal of the American Statistical Association*, **90**, 443–450.

Kessler, R. C., Nelson, C. B., McGonagle, K. A., *et al* (1996) Comorbidity of DSM-III-R major depressive disorder in the general population: Results from the US National Comorbidity Survey. *British Journal of Psychiatry*, **168** (supplement 30), 17–30

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One hundred years ago

The Lunacy Act, 1890, and its amendments

Your correspondent, "Formerly a County Asylum Superintendent," I am glad to see, suggests as a remedy for the present evils the *abolition of lunacy certificates*. I have long maintained that the lunacy certificate should be abolished as constituting a grievous interference with the liberty of the subject in obtaining medical treatment. That a person suffering from disease of the brain must obtain, or have obtained, the authorisation of a magistrate before going to a sanatorium for treatment would be grotesquely absurd if it did not cause such serious evil. The public would rebel at once if the lawyers demanded that a man with a