Finally, we look forward to reading Blacker and Clare's findings on general practice depression, and hope that the differences—as well as the similarities—between the studies will help elucidate the nature of these disorders.

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Depression in School Phobia

DEAR SIR,

We read the paper by Kolvin, Berney & Bhute (Journal, October 1984, 145, 347–357) with interest and disagree with many of its conclusions which serve only to obfuscate the diagnostic precision of depression in childhood. The authors demonstrate circular reasoning when they conclude that their criteria are different from Weinberg's but similar to the RDC. We wish to point out that the Weinberg criteria are based on the RDC.

The key items that Kolvin and colleagues put forward account for 5 of the Weinberg criteria. We wonder what has happened to the others, in particular, concentration problems and childrens' school performance, as both these are important cognitive variables. In Brumback's paper (1977), depressed children did in fact, endorse 6 or 7 of the 10 Weinberg criteria and they also used a semistructured closed-ended interview with good interrater reliability. There is no indication about those parameters by Kolvin and his colleagues.

Unfortunately, this paper by Kolvin et al has some serious problems with regard to its aims. The first is quite reasonable, to identify subgroups in a population of children with school phobia using clinical and statistical methods. However, to identify a depressive subtype and to then develop a criterion for depression in the absence of a control group is totally invalid. As the authors observed all of these children studied may represent a spectrum of severity of depressive illness and not distinct diagnostic groups.

Finally, to conclude that depression has a multifactoral origin obscures the finding that many of these children have an underlying substrate of right cerebral dysfunction and that the dependent variable in 80% of the cases is a positive family history as reviewed by Brumback et al, 1980. Further nosology will be dependent upon the discovery of state-independent biological markers and state-dependent biological correlates.

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DEAR SIR.

Kolvin et al (Journal, October 1984 145, 347–357) addressed the issue of depression in school refusal. But liberal use of $(1) \chi^2$ procedures and (2) principal components analysis probably inflated the discriminant validity of depression measures and resulted in spurious association of these with other variables studied.

(1) In testing the discriminatory efficacy of five compound depressive indices, Kolvin et al sought that point which maximally discriminated depressed and non-depressed subjects. This "best cut" procedure works to exploit chance data fluctuation (Cronbach, 1949). Implicit in the approach are S-1 (where S=number of scores in a range) comparisons, though only the most advantageous is reported. The result is to increase Type I and per experiment error rates. For example, the chance probability of Kolvin et al finding a significant difference on the 21-item Total Global Depression Score is approximately $21-1 \times .05 = 1.00$; on the 15-item Global Score it is about $14-1 \times .05 = .65$; on the "11 items-cut-of 5+" it is around 11-1 x .05 = .50.

Also the "best cut" approach probably inflated artifactually the discriminant efficacy (rates of sensitivity and specificity) of the variables in question. This is serious, given the authors' own admission that most of the indices were not highly sensitive in any case. A preferable approach would have been to make the cuts on theoretical grounds a priori, or to use the median split.

(2) In two principal component analyses, Kolvin et al met none of the prerequisites ensuring valid factor extraction (Wade, 1978).

- (a) A recommended *minimum* ratio of subjects to variables is 5:1. As the ratio moves towards unity, factor analytic results become increasingly meaningless. Kolvin *et al* performed analyses with approximately 2 and 3.6 subjects per variable.
- (b) The recommended *minimum* number of subjects for any factor analysis is 100. Kolvin *et al* used 50 participants in one analysis, 47 in the other.
- (c) Correlation matrices should be tested for psychometric adequacy prior to factoring. Kolvin et al reported no data on matrix adequacy.
- (d) Determination of number of interpretable factors is problematic. In their second analysis, Kolvin *et al.* interpreted two factors without reporting criteria for doing so.
- (e) Given that factor analysis imposes as well as extracts structure, some measure of replicability is necessary. Kolvin *et al* might wisely have subjected the data to an alternative factor extraction technique and tested for congruence.
- (f) Kolvin and coleagues' criterion factor loading of .2 is extremely low. Explaining only $.2^2=4\%$ of the variance, it is substantively meagre and statistically vulnerable to chance data fluctation. Moreover, in the text itself, Kolvin *et al* interpreted variables with loadings as low as -.13 (Unreality index) and -.15 (Recent obsessionality).

Many of these shortcomings are serious even in isolation. Combined, they severely undermine conclusions drawn by Kolvin et al.

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Folate Deficiency in Dementia DEAR SIR.

The report by Hancock and colleagues (Journal, October, 1985, 147, 404–407) is a further confirmation of widespread malnutrition in the elderly mentally ill including those in institutions (Thomas et al, 1982) and those in the community (Shaw et al, 1984).

Their findings of the common occurrence of deficiencies in vitamins C, B₂ and B₆ prompt us to

report the results of a survey of folate levels in patients with dementia.

Seventeen male and 28 female demented patients who were electively admitted into the dementia research bed at St George's Hospital were studied. Their ages ranged between 67 and 96 years (mean 78.7 years). These patients were admitted for one week mostly from the community to carry out a large battery of clinical and biological investigations including cognitive (orientation test), behavioural rating scales and 14 sub-tests of cortical function. Biological investigations included haematology and biochemistry profile. Diagnosis of dementia was confirmed by computerised tomography. Seven male (41%) and nine female patients (32%) were found to have folate deficiency (serum folate < 2.5 ng/ml). There was no association between folate deficiency and classification of dementia and the degree of cognitive and cortical dysfunction. Folate deficient patients however were significantly older than those with normal foliate levels P < 0.03. There was a significant association between folate deficiency and increased erythrocyte sedimentation rate (ESR) and increased thyroxine levels (T4). Interestingly, only one folate-deficient patient had increased mean corpuscular volume (MCV). There was no association between folate deficiency and the presence of physical illness and any concurrent prescribed medication.

We have previously reported the common occurrence of folate deficiency (serum and red blood cell folate) in drug-free depressive and lithium treated patients (Abou-Saleh, 1985). More recently Coppen and his colleagues (to be published) have explored the usefulness of daily physiological supplements of folate (200 µg) in the management of 75 patients on prophylactic lithium and in the context of a double-blind placebo-controlled trial. During the trial, patients with the highest plasma folate concentrations showed a significant reduction in their affective morbidity and patients who had their plasma folate increased to 13 ng/ml or above had a 40% reduction in their affective morbidity.

The implications of these results for nutritional hypotheses for the psychoses have been discussed (Abou-Saleh & Coppen, 1986). It is conceivable that supplements of folic acid and other vitamins should enhance the recovery of the elderly mentally ill both in institutions and in the community.

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