disorder was not associated with higher ('psychotic') scores on the neurotic-psychotic continuum at the time of the index admission. Thus position on the continuum did predict poor outcome, but not by identifying individuals who went on to chronic psychosis (Duggan *et al*, 1989). Perhaps it was the Australian group's lack of such a continuum score that led to their failure to replicate our findings of poorer outcome among the more endogenous/psychotic depressives.

The absence of any patients in the Australian series who developed schizophrenia-like psychoses is suprising, as these have been a consistent feature of other follow-up studies of depression (Bebbington, 1982). Perhaps their cases were less severe. Alternatively, any patients who developed schizophrenia may have remained hidden among their 12 patients who refused interview or could not be traced. In our series, in which 99% of patients were traced, some of those who were most difficult to trace turned out to have chronic psychosis.

We are puzzled by the fact that our data appears to have undergone an optimistic transformation during its journey to the antipodes. We found that between 25% and 30% of patients remained always incapacitated or died by suicide; Dr Kiloh *et al* (Table IV, p. 755) report our figure as 19%. We found that one-third of our patients fell into our very poor outcome group; they report our figure as 25%. The detail of their own analysis is confusing, for they report 17% always incapacitated or dead from suicide, but then say that only 11% fell into the very poor outcome group, despite using our definition in which the former is a subset of the latter. We also wonder whether the figures in Table III (p. 754) are strictly comparable, as the Australian group describe their own as based on "proportions readmitted" rather than on survival analysis.

These differences apart, we are intrigued by the possibility that it is better to have a severe depressive illness in New South Wales than in London, particularly since the Australian endogenous patients were much older and therefore one might have expected them to fare worse. Dr Kiloh *et al* suggest that more effective treatment may be the key and point out that 10% of their whole series received lithium. We have re-examined our own casenotes and found that 20% of the London series received lithium. Perhaps we in London should recommend emigration to our patients!

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... Or not?

SIR: We read with interest the papers by Lee & Murray (*Journal*, December 1988, **153**, 741–751) from London and Kiloh *et al* (*Journal*, December 1988, **153**, 752–757) from Sydney. The London paper focuses on shortcomings in previous studies on the outcome of depression and attempts to redress them. The Sydney group use a longitudinal study in a continuing attempt to validate the neurotic-endogenous classification.

A major problem with the London study is the selection of the initial cohort. We feel that the cohort is more likely to have included atypical cases. Firstly, only 17% of the subjects were from the catchment area. A significant proportion of the remainder were undoubtedly referrals for second opinion, and therefore more likely to have been atypical or severe cases. The authors attempt to counter this criticism using anecdotal evidence, but do not provide any data to rule out sample bias. The better outcome in the referred group may have been related to more aggressive treatment and follow-up. Secondly, at follow-up the sample had only four unipolar depressive patients. We are not questioning the fact that 'complications' may be an intrinsic part of depression. However, a cohort of 89 in which 3 patients have leucotomy for bulimia nervosa and 18 go on to develop other psychotic disorders is likely to have been biased towards severe or atypical cases. It would be useful to compare the rate of 'complications' between patients whose index admission was their first with those whose index admission was not their first. It would have been better to exclude patients over the age of 65 altogether, rather than include only two.

The Sydney paper makes no comment about admission patterns and possible sampling bias. Their cohort was not made up of consecutive admissions. Their follow-up was incomplete, in that of the 145 patients only 92% were traced and 72% of the survivors interviewed. Twelve refused to co-operate or could not be traced. In London more than half of the severely disabled group had lost contact with the psychiatric services. This suggests that the poor outcome

group are more difficult to follow up, and may have constituted a significant proportion of the 12 who were not followed up in Sydney. In both the endogenous and the neurotic group 17% were always incapacitated by their illness or committed suicide. Yet when the London criteria were applied, only 9% and 12% in the respective groups had a very poor outcome. The poor outcome group in Sydney, therefore, appears to have been underestimated.

It is interesting to note that both groups have come up with findings that contradict their previously firmly-held but opposed beliefs about the endogenous-neurotic dichotomy. Both groups used a neurotic-endogenous classification for the cohort. When the Sydney group reclassified their cohort using DSM–III criteria they found that their sample covered the entire range of depressive illness from major depressive illness to adjustment disorder. The same spread of diagnoses may apply to the London cohort. This highlights the heterogeneity of both the samples, and casts doubts on the conclusions they make. It would, therefore, be a pity if these papers stimulated more wasteful research into the validation of the endogenous and neurotic categories.

Both studies clearly show us that a significant proportion of depressed patients do poorly in the long term. The findings of two major studies conducted in the pre-treatment era (Lundquist, 1945; Rennie, 1942) are broadly similar to those of the present studies. We wonder why it is that, almost 50 years later, with innumerable treatment methods at our disposal, we are still faced with such a dismal outcome for depression. Are we not using these therapeutic tools appropriately, or are they of no use in ameliorating the poor long-term outcome? It is unfortunate that neither of the current studies have attempted to correlate outcome with treatment. There is clearly a need for further long-term prospective studies into the outcome of depression and the effect of different treatment methods, both short and long-term, on prognosis. We hope the findings of these studies will stimulate further research into this rather neglected and difficult area.

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AIDS hypochondriasis

SIR: Todd (*Journal*, February, 1989, **154**, 253–255) describes five cases of unwarranted concerns about AIDS, "which cover a wide range of psychopathology". In several of the non-psychotic cases, there are similarities with cases of primary hypochondriasis (DSM–III–R; American Psychiatric Association, 1987), a diagnosis which has not been considered. Examination of these similarities may improve the understanding of the processes involved in such cases.

In a cognitive-behavioural formulation of primary hypochondriasis, it has been suggested that the central feature is the persistent misinterpretation of innocuous bodily sensations as indications of physical illness (Salkovskis & Warwick, 1986; Warwick & Salkovskis, 1989). As in these examples, the bodily sensations involved may be the result of a wide range of conditions, such as anxiety states, trivial physical disorders, or normal bodily variations. The resultant erroneous perception of threat to health inevitably leads to anxiety. This anxiety will be associated with a variety of other clinical features, such as avoidance, reassurance seeking, and guilt, and may resemble phobic or obsessive-compulsive states. In this view, the misinterpretation is the primary problem, and the other clinical features should be regarded as secondary phemonena, rather than "symptomatic of an underlying psychiatric disorder".

Consideration of hypochondriasis along cognitive-behavioural lines also has important implications for treatment, which are clearly illustrated by the results of Dr Todd's cases 3 and 5. Clinical experience suggests that some patients who are labelled 'poorly motivated' in fact experience high levels of anxiety, which may make them reluctant to carry out exposure treatments. It is likely that successful treatment of such cases will need to include *direct* attention to the beliefs and misinterpretations responsible for this anxiety, along with treatment directed at *secondary* symptoms. A cognitive approach may help such patients to correctly attribute their sensations and hence facilitate exposure and response prevention. Dr Todd rightly makes the point that these cases often do not respond to simple reassurance, yet case 3 "required repeated reassurance between outpatients appointments". In both hypochondriasis and obsessive-compulsive disorder it is recognised that the provision of repeated reassurance can reinforce and prolong the patient's fears (Warwick & Salkovskis, 1985). Reassurance can take the form of unnecessary physical consultations, examinations,