the success of these interventions might be inflated by drug defaulters among the controls. It is important to recall that many have argued that neuroleptics themselves serve to delay relapse (Englehart *et al*, 1967) and that, to quote Macmillan *et al* (*Journal*, February 1986, **148**, 128–133), "relapse rates were lower on... medication than placebo, *but however assessed, outcome at this early stage was poor for many patients*" (our italics).

Clearly, we have no cause for complacency, and both environmental and biological research needs to be actively encouraged and pursued.

MAX BIRCHWOOD
JOANNE SMITH

All Saints Hospital
Lodge Road
Birmingham B18 5SD

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A Comparative Trial of Amitriptyline and Fluoxetine

SIR: The comparative trial of amitriptyline and fluoxetine in depressed out-patients by Young *et al* (*Journal*, September 1987, **151**, 337–340) found no significant difference in outcome except for a slight difference, discounted by the authors, on the Beck Scale for Depression (BSD). Improvement in BSD scores commenced in the first week, and six-week improvement is described as moderate. I may not be alone in being baffled by the conclusions drawn from these results: "Efficacy was similar in the two groups"; "Overall both drugs proved to be effective antidepressants over the six-week trial..."; "The apparent efficacy of fluoxetine..."; "... both drugs proved equally efficacious"; "... the absolute usefulness of both drugs can only be assumed".

With the possible exception of the last of these statements, none of them (*pace* the pharmaceutical industry) is warranted by the evidence presented. Perhaps the most convincing result was that amitriptyline gave patients dry mouths and made them fat; fluoxetine made them sick. This seems a good example of the "me-too" variety of drug trial. A placebo group was omitted on ethical grounds. Similar grounds might have been invoked for the omission of the conclusions.

T. J. FAHY

Department of Psychiatry
Regional Hospital
University College Galway

Tuberous Sclerosis and Psychosis

SIR: Dennis & Hunt (*Journal*, September 1987, **150**, 413–414) have recently drawn attention to two case reports in which tuberous sclerosis in associated with psychotic symptomatology (Lawlor & Maurer, *Journal*, March 1987, **150**, 396–397; Clarke *et al*, *Journal*, May 1987, **150**, 702–703). In addition, they refer to their own study of ninety children with tuberous sclerosis, over half of whom demonstrated psychotic behaviours (predominantly autistic or hyperkinetic) (Hunt & Dennis, 1987). They propose that profound language delay, severe impairment of social interactions, hyperkinesis and sleep disturbance constitute a behavioural phenotype. They believe that this occurs so frequently as to be included within the diagnostic criteria for tuberous sclerosis.

Within the field of mental handicap, there are few specific organic disorders which are associated with specific psychiatric disorders. Exceptions might include organic mental states in Down's syndrome, self-mutilation in Lesch-Nyhan and Cornelia de Lange syndromes, autistic-like symptomatology in Rett's syndrome, and perhaps a few others.

The problem of associating tuberous sclerosis with autistic symptoms is of interest, but difficult, as it raises our uncertainty over the aetiology of autism itself, although this is presumably the result of some organic lesion. The association really resolves itself around two questions: is tuberous sclerosis *directly* associated with autistic symptoms? Or does it frequently give rise to degrees of mental handicap, which are themselves frequently associated with autistic symptoms?

The authors need to show that, having controlled for degrees of mental handicap, patients with tuberous sclerosis are more likely to develop autistic symptoms than other groups of brain-damaged individuals. Unless this is done, the suggestion that autistic symptoms are specifically applicable to individuals with tuberous sclerosis, any more than to patients with, say, hydrocephalus, may be entirely spurious.

RICHARD A. COLLACOTT

Leicester Frith Hospital
Groby Road
Leicester LE3 9QF

Reference

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Comparative Trial of a New Antidepressant

SIR: Few would argue with the point raised by Dunn (*Journal*, August 1987, **151**, 269) and Thompson (*Journal*, November 1987, **151**, 702–703) that a comparison of a new antidepressant with a placebo to establish that some efficacy exists is desirable. Clearly, ethical issues are a problem, and there is an extensive literature highlighting these (Klerman, 1986; Rickels, 1986). Practical issues are also a factor, and in a current clinical trial involving placebo control an eminent colleague withdrew his involvement because he felt that every patient should receive an active compound.

Studies previously carried out comparing fluoxetine, a tricyclic antidepressant, and a placebo (Stark & Hardison, 1985; Colin & Wilcox, 1985) confirmed that fluoxetine was more effective than placebo.

My study (Levine *et al*, *Journal*, May 1987, **150**, 653–655) was carried out to establish further evidence of efficacy and to compare the occurrence of side-effects with those of imipramine. The numbers involved allowed for any significant difference to be discriminated. A minimum score on the HRSD of 17 permitted less severe cases to be included. The suggestion by Thompson that 75 mg of a tricyclic antidepressant is "well accepted to be inadequate" is surely an idiosyncratic view, and is at variance with established practice over almost 30 years. Perhaps the as yet unpublished paper quoted in his letter will inform us otherwise.

The need for new antidepressants clearly exists when present compounds are only marginally more effective than placebo at any dose level, and future research will hopefully not be influenced by the "overcrowded market".

SIDNEY LEVINE

Oldham & District General Hospital
Rochdale Road
Oldham OL1 2JH

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Depressed Mood After Stroke

SIR: For Wade *et al* (*Journal*, August 1987, **151**, 200–205) to compare their finding that 20% of their stroke patients were definitely depressed and 10% probably depressed with Bergmann's (1982) reported rate of 4.4% for depressive illness and neurosis in 360 elderly women is of doubtful relevance. Reported rates of depression in community studies of the elderly vary enormously, and one could equally well argue on the basis of comparison with the work of Zung (1967) or Stenback (1979) that stroke protects against depression, as both these studies can be interpreted as demonstrating depression in well over 40% of elderly subjects. It is possible to quote studies which purport to show rates of depression in old age which vary from 48% right down to 1%. None of them help to interpret the findings of Wade *et al*, whose study is flawed by the lack of an age-matched control group, and the use of an instrument which was not developed in order to measure depression in stroke victims with an mean age of 70 but to measure the severity of the depressive syndrome in a group of patients already diagnosed as depressed whose mean age was in the mid-forties (Snaith *et al*, 1971).

DAVID AMES

The Royal Free Hospital
Pond Street
London NW3 2QG

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SIR: We thank Ames for stressing that the Wakefield Self-Assessment Depression Inventory was not developed for use with elderly people, and for pointing out that we did not have an age-matched control population. We discussed both points in the original paper, although we acknowledge that we did not refer to the population studies he mentioned.