learning disability are not a homogenous group, neither are people with Down's syndrome. The chromosomal origin of Down's syndrome is an important variable which every study of people with Down's syndrome must account for.

Psychiatric disorders of people with a learning disability should no longer be studied as a "group phenomenon", and every effort must be made to compare their psychopathology based on aetiological/syndrome groups. Problems inherent in the study of behaviours of heterogenous groups in developmental psychiatry are well documented in a recent article by O'Brien (1992).

Another important source of error in both studies was that the psychiatric diagnoses were made without due consideration to the observation that this population has a higher risk of developing insidious medical illnesses, such as sensory impairment and thyroid dysfunction. These impairments can present as a psychiatric symptom leading to errors in clinical diagnosis. There is no mention of the nature of, and figures for, coexisting medical conditions in either of the studies.

In the second article an attempt is made to equate "regression in adaptive functioning" in the elderly Down's syndrome subjects with the "development of Alzheimer's disease". Not all elderly people who lose skills have dementia. Without a full medical screen, in particular for hypothyroidism and sensory loss, both of which will lead to loss of skills and subsequent regression, a probable diagnosis of Alzheimer's disease is untenable.

In the first article there is a statement that "Heaton-Ward (1977) had found a prevalence of 3.4% ... similar to that of 32.2% found by Reid (1972*a*,*b*)". Review of the papers by Reid (1972*a*,*b*) shows the actual percentage to be only 3.2%.

We recommended that no psychiatric diagnosis should be made for people with Down's syndrome/learning disability without a full medical screen first, as these people are at higher risk for developing related psychiatric conditions. The authors of both articles deserve commendation for their industry and contribution to the literature but, in view of the flaws in the studies, the conclusions that they have arrived at should be accepted with reservation.

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AUTHORS' REPLY: We wish to thank Drs Prasher & Krishnan for the interest that has been shown in our two studies (Collacott *et al*, *Journal*, November 1992, **161**, 671–674; Collacott, *Journal*, November 1992, **161**, 675–679) which investigated different psychiatric aspects of people with Down's syndrome. While they highlight one error, clearly typographical in origin, we are puzzled by some of their criticisms.

The plethora of characteristics typical of Down's syndrome renders the diagnosis essentially a clinical one. The overwhelming majority of cases of Down's syndrome are due to trisomy 21 (93–96%). Hence, in studies of a large size, such as ours, the chromosomal origin of the syndrome loses relevance. Additionally, cases of mental illness and Alzheimer's neuropathology have been reported in translocation and mosaic Down's syndrome individuals (Crapper *et al*, 1975; Lai & Williams, 1989; Reid & Aungle, 1974; Reid *et al*, 1978). Conversely, there is no evidence to suggest that such individuals sustain a different pattern of mental disorders than those with trisomy 21.

The study of psychopathology in groups of people with specific causes for their learning disability clearly provides a different kind of information to that gained from studying a heterogeneous group of people with learning disability from diverse causes. The former may provide information that becomes relevant to clinical practice and the assessment and treatment of individuals, whereas the latter may delineate the extent of problems and needs in whole populations, and may have an influence on service planning. Our study has tried to address the former issue, rather than the latter, by studying people with Down's syndrome. For the purpose of describing rates of psychiatric disorders, a comparison control group is, of course, necessary due to the lack of standard diagnostic criteria for psychiatric illness in people with learning disabilities.

Regrettably, we must disagree with the statement that in people with Down's syndrome/learning disabilities, the first step in making a psychiatric diagnosis is a "full medical screen". In clinical practice, psychiatric diagnosis requires the taking of a full history, under the usual standard headings (from both patient and collaterally, from an informant), a

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mental state examination and, where indicated, a physical examination or selective physical examination, and special investigations. This applies as much to people with Down's syndrome as it does to the general population.

We hope our papers help to establish that people with Down's syndrome experience increased rates of certain psychiatric disorders, and that the pattern of adaptive behaviour in these individuals changes with age. There is, of course, a distinction between descriptive psychopathology by which a psychiatric diagnosis is made, and aetiological factors which contribute to the described illness. Having established different rates of psychiatric disorders, the next stage is to determine the relevant aetiological factors. The view of Drs Prasher & Krishnan, that sensory impairment and medical illness account for the altered rates of psychiatric disorders, is speculative. We look forward with great anticipation to the publication of results from the Birmingham Down's syndrome study to support their hypothesis.

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Combination treatment of depression

SIR: The question of how to treat depressed patients who have failed to respond to a first antidepressant agent is of great clinical importance. Seth *et al* (*Journal*, October 1992, **161**, 562–565) describe the successful treatment of such patients by the addition of a selective serotonin reuptake inhibitor (SSRI) to the tricyclic antidepressant; a potentially more straightforward manoeuvre than lithium augmentation or changing to a monoamine oxidase inhibitor. Points by subsequent correspondents are well made (Cowen & Power, *Journal*, February, 1993, **162**, 266–267) but, given the potential hazards of combining drugs, may fail to address one critical issue – would *changing* to an SSRI be equally as effective as adding one? One influential study (Nolen *et al*, 1988) found no benefit in changing to fluvoxamine for patients who were tricyclic antidepressant non-responders, and is frequently cited to dismiss the strategy. However, a number of other authors have found that a clinically significant proportion of tricyclic non-responders do respond to a subsequent SSRI given alone (Lingjaerde *et al*, 1983; Delgado *et al*, 1988; Beasley *et al*, 1990; White *et al*, 1990). Admittedly these have been open studies but I would argue that it is premature to dismiss changing to another non-monoamine oxidase inhibitor antidepressant in favour of combination drug treatment on current evidence. Further research is required to determine the best 'second-step' treatment for patients failing to respond to an adequate trial of a tricyclic antidepressant drug.

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Dysfunctional attitudes and Beck's cognitive theory of depression

SIR: I would like to comment on Brittlebank *et al*'s article (*Journal*, January 1993, **162**, 118–121) on autobiographical memory in depression. They compared a measure of autobiographical memory to the Dysfunctional Attitudes Scale (DAS; Weissman, 1979) with respect to: (a) whether scores on each measure fell as depression scores decreased (i.e., whether the variables behaved as state or trait markers); and (b) whether scores on each measure predicted depression at three- and seven-month follow-up. The findings concerning autobiographical memory are interesting

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