Nigeria (Lambo, 1964). They hint that such collaboration is "undesirable on moral grounds". This authoritative censure is made in connection with a reference to our co-operative relationship with traditional healers in a different culture area, that of the Northwest Coast Indians of North America. The therapeutic ceremonials described (Jilek & Todd, 1974) have nothing to do with witches, witchcraft or sorcery. The witch-doctors referred to in our article endeavour to assist, by means of a culture-congenial traditional psychotherapy and sociotherapy, those North American Indians who under the impact of rapid socio-cultural change are showing symptoms of anomic depression and anxiety.

While working with tribal societies in three continents, I have had the experience that friendly contacts and monitoring collaboration with traditional practitioners who have an established role in their community is more beneficial to the patient than ignoring or condemning them. Such collaboration becomes a necessity in the planning of comprehensive primary health and mental health care in a developing country with limited professional manpower resources (Jilek, 1985).

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Depression in Schizophrenia

SIR: The paper by Elk *et al* (*Journal*, August 1986, **149**, 228–229), looking at rates of depressive symptoms in schizophrenic patients from three "racial" groups, raises a number of issues.

One might question results based on small numbers (groups of 19, 15 and 22 patients) but, more importantly, is it methodologically valid to compare three such disparate groups without considering other variables? No information is given in the paper about patients' social circumstances or how typical they are of patient populations in the three "racial" groups. Referees and readers of the *Journal* may not know that black, coloured and white patients in South Africa by law have to be nursed in separate wards, usually separate hospitals, and that black and coloured people have extremely limited access to medical care.

A further point is that the reasons for comparing these three groups are not explained; is it because they were thought to be biologically different or were from different social backgrounds? Referees should ensure that the assumptions underlying a research project are clarified for the readership in either the introduction or the discussion.

Finally, there are many who would suggest that at the present time, papers from South Africa should not be published by British journals. It would be helpful to have some editorial comment on this issue.

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Macrocytosis and Cognitive Decline in Down's Syndrome

SIR: Welfare & Hewitt (*Journal*, April 1986, **148**, 482–483) suggest that in Down's syndrome cognitive decline and macrocytosis may be related. The causal relationship they suggest is unlikely. While red blood cell (RBC) membranes may age more rapidly in patients with Down's syndrome, this would lead to these RBC becoming smaller rather than larger. The macrocytosis seen in patients with accelerated ageing of RBCs due to thalassaemia or haemolysis is caused by the large size of the reticulocytes produced in response to the RBC loss (Beard, 1978).

Welfare & Hewitt state that in their Down's syndrome patients "there was no evidence of vitamin B12 or folate deficiency to account for this further increase" in RBC size. Eastham & Jancar (1983) state that Down's syndrome patients with macrocytosis were "not being treated with anticonvulsants and were not anaemic". It is well recognised that functionally significant folate deficiency in association with macrocytosis may be present in patients without megaloblastic anaemia being apparent (Botez &

Reynolds, 1979). I would suggest that macrocytosis and cognitive decline in Down's syndrome is likely to be related to undetected folate vitamin deficiency consequent on institutional nutrition, complicated by the gastrointestinal malabsorbtion that some Down's syndrome patients have.

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Dependence on Pseudoephedrine

SIR: Sympathomimetic amines are a major ingredient in proprietary medications for the treatment of upper respiratory tract infections. We describe a patient dependent on pseudoephedrine.

Case report: The patient first presented at the age of 21, with symptoms of depression. This responded poorly to treatment, and continued for 12 years. In the eleventh year she was convicted of the theft of a medical prescription pad and for making a prescription for herself for Actifed. She subsequently admitted taking between 50 and 300 ml/day of this preparation — the recommended dosage is 30 ml/day. She said that she took it because it "gave her a lift". A year later she began to describe psychotic symptoms, which have lasted for four years. She had auditory and visual hallucinations and passivity feelings. These symptoms fluctuated and were variable in content; she showed none of the negative symptoms of schizophrenia. Although it was not possible to make a diagnosis of schizophrenia, she clearly suffered from depression, had an unstable personality, and had abused Actifed. Treatment included both oral and depot phenothiazines in addition to supportive psychotherapy, but her compliance was poor.

The present report is important because it shows that dependence and possibly psychosis can occur with over-the-counter preparations. Each 5 ml of Actifed contains 30 mg of pseudoephedrine hydrochloride and 1.25 mg of triprolidine hydrochloride. Dependence on amphetamines and other sympathomimetic drugs is well known. There is, however, only one report of dependence on pseudoephedrine (Diaz *et al*, 1979). Visual hallucinations on attempted withdrawal are described in that report. Paranoid psychosis after abuse of Actifed has also been described (Leighton, 1982). The patient's fluctuating psychosis is thus in accordance with previous findings.

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Admission Rates and Lithium Therapy

SIR: Dickson & Kendell (1986) recorded admissions for mania and psychotic depression in Edinburgh over the years 1970–1981 and found a rise which they could not explain. They had expected a fall, because the use of long-term lithium therapy had increased ten-fold during the same period, and they felt that their findings cast doubt on the efficacy of lithium prophylaxis in ordinary clinical practice. Such a conclusion might have far-reaching consequences for patients with recurrent manic-depressive illness, and Dickson & Kendell's analyses merit close scrutiny.

It seems a dubious procedure to draw conclusions about the efficacy of a treatment given to a limited number of patients from admission rates for a much larger number. Not all manic-depressive patients receive prophylactic lithium treatment: it is given only to those with frequent recurrences and is started only after the patients have had several episodes. So even if lithium treatment were 100% effective, it could be expected to prevent only a fraction of the admissions for mania and depression.

Even so, a fall in the admission rate, albeit a small one, would be expected if lithium treatment was the only factor influencing admissions. It obviously was not; powerful forces with an opposite effect must have been at work. Dickson & Kendell examined some factors, such as change in diagnostic fashions or admission thresholds, but were unable to account for the rise. One could think of several others.

Be that as it may, the fact remains that the admission rate for mania and depression showed a pronounced rise, and the rise must have been caused by something. The moderate effect of lithium may