so the problem is solved by suggesting there is an "ethnopharmacological basis" for the *zombi*, implying the colonial psychiatrist got it right every time.

I was merely saying we have to "examine more closely" the so-called culture-bound syndromes (*Journal*, July 1985, **147**, 93) and pointed out evidence for and against "voodoo death". This is nothing to do with *zombis*, *vodun* or even Haiti. It is one of the terms bequeathed to us by European psychiatrists in Africa and Australia, and is perhaps more properly termed *psychogenic death*.

As to Haiti, Douyon and Wade Davis (1983) do not claim that tetrodotoxin causes people to be "raised in a comatose trance from the grave and forced to toil as a slave". Wade Davis' evidence is that intraperitoneal injections of local poisons into rats produce "catalepsy", and similar symptoms have been noted in diguatera and tetrodotoxin poisoning (as found in the consumption of the Japanese delicacy *Fuga pardalis*).

The term *zombi* (and the cognate *jumbi* as it is called in rural Trinidad where I carried out my fieldwork in traditional medicine) has at different times had a variety of meanings: god (*power*, *orisha*), possessed initiate, soul or body, twin, skin disease, mildew and mushroom, let alone reanimated corpse (Thompson 1958, Deren 1970). My own informants offered related ambiguous and binary notions including lesbian (*zami*). *Zombi* is probably derived from Amerindiam *zemi* (twin soul) and is an indigenous psychological term, a rich, complex and subtle web of explanations of personal identity, selfhood, autonomy, action, causality and power. Mircea Eliade has gone so far as to say the term refers to mystical power in general, "the sacred". Interestingly, given the intensive fieldwork in remote areas of Haiti by numerous social anthropologists previously eg Métraux 1959), none came across the mysterious slave corpse beloved of the European imagination.

Apart from questions of scholarship and sensationalism, it may be relevant to emphasise again the dangers of scientism: reducing social patterns and belief to a single unitary biological cause, its "basis" as de Pauw terms it. Social meanings are ascribed to the phenomena of the natural world, whether they are stars, sexes or the dead. Mars (1947) found that one claimed *zombi* had neurosyphilis.

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Phobic Neuroses in India: A Cross-Cultural Comparison

DEAR SIR,

We read with interest Drs. Raguram and Bhide's account of phobic neuroses in India (*Journal*, November 1985, 147, 557–560). While commenting on the lack of Indian studies specifically on this subject, they fail to mention an earlier report by us (Chambers *et al*, 1980) on phobic neuroses from the same institute. Apart from a clinical analysis of 39 phobic subjects seen during the first three years (1976–1979) of the eight years spanning Raguram and Bhide's study, we had carried out a cross-cultural Indo-British comparison of phobic neuroses.

The Indian part of our study was a similar retrospective case record survey of 39 patients (31 male and 8 female) diagnosed as having phobic neuroses (ICD-9 criteria) at the National Institute of Mental Health and Neurosciences (NIMHANS), India. The British part of the study involved analysis of case records of a group of 30 similarly diagnosed phobic patients at the Maudsley Hospital, London, UK. The Maudsley comparison group was drawn randomly from a series of 268 phobic patients treated at this hospital between 1972–1976, and matched for age and sex with the Indian group. One of the investigators (J.C.) analysed the case records in both hospitals.

We found that males (79%) predominated in the Indian group as compared to the British group (21%) confirming Raguram and Bhide's finding of a male preponderance in the Indian group. The median ages of presentation, duration of symptoms, social class and marital status did not significantly differ between the two groups.

Thirty-six percent of the Indian sample were graduates as compared to 11% of the British sample ($P<0.001$) suggesting that education promotes help-seeking behaviour for phobic patients in the Indian context.

The British sample contained significantly more patients with agoraphobia (Indian group: 27.5%; British group: 60%, $P<0.05$) and social phobias

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(Indian group: 7.5%; British group: 35.5%, $P<0.05$) while phobias for sudden death (Indian group: 30%; British group: 10%, $P<0.05$) were more frequent in the Indian group. Associated somatic symptoms were significantly more frequent in the Indian sample, in keeping with earlier observations of the predominance of somatisation in non-western cultures (Carstairs & Kapur, 1976). Precipitating psychosocial stressors were more frequent in the Indian group (57.5%) as compared to the British group (40%; $P<0.05$). Family history of phobic illness was significantly less frequent in the Indian group (2.5%) as compared to the British group (9%; $P<0.05$).

Clearly, the clinical patterns of phobic neuroses in western and non-western clinic populations are not essentially similar as Drs Raguram and Bhide appear to conclude. Our data indicate important differences even after careful matching of the two groups. While these differences may indeed be due to a variety of psychosocial factors, more field studies are needed before venturing fanciful culture-based explorations.

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Neuroleptic Malignant Syndrome and Lethal Catatonia

DEAR SIR,

In their review of the neuroleptic malignant syndrome (*Journal*, January 1986, **148**, 47–51) Abbott and Loizou correctly point out that it is clinically identical to Stauder's lethal catatonia; however, in common with previous writers on this subject they do not suggest how the two conditions may be distinguished. Lethal catatonia has been recognised for over a century as a rare and dramatic cause of sudden death in mental patients, but in recent decades this diagnosis has suffered a curious eclipse. As described by the clinicians of the pre-neuroleptic era it typically presented with a prodromal phase of personality change, hyperactivity, affective disturbance, delusions, hallucinations, confusion and catatonia. Since the introduction of neuroleptic drugs in the 1950s, patients presenting with such florid psychotic symptoms will have received a major tranquillizer as a matter of course, and any subsequent hyperpyrexia may well have been attributed to the drug rather than the original disorder.

Is it then possible that all cases of neuroleptic malignant syndrome are in fact nothing more than the old lethal catatonia in spurious association with new and commonly-used drugs? The existing literature is not very helpful on this question: examination of published cases of neuroleptic malignant syndrome shows that many closely resemble the classic clinical picture of lethal catatonia (for example, the case described by Abbot and Loizou), but the uneven quality of description limits the conclusions that may be drawn by such comparisons (Peele & Von Loetzen 1973). Taken alone, however, these case reports cannot be regarded as adequate evidence for a separate neuroleptic-related condition. More convincing in this respect are descriptions of the syndrome in non-psychotic cases (Burke *et al*, 1981); cases where clinical course has been related to the kinetics of the implicated drug (Allan & White, 1972); and relapses of the condition following re-exposure to neuroleptics (Coons *et al*, 1982). Reports of typical cases in association with other dopamine-depleting drugs and with the withdrawal of dopamine enhancers such as levodopa also support the existence of a disorder secondary to iatrogenic disturbance of central dopaminergic transmission. However, it remains likely that the 'neuroleptic malignant syndrome' as we currently perceive it is a hybrid of this iatrogenic disorder and mis-diagnosed lethal catatonia.

If this clinical syndrome is indeed due to a disorder of dopaminergic function, then it is of some interest that one form of the disorder appears to have existed long before the introduction of major tranquillizers. In follow-up studies of the survivors of lethal catatonia there is a variable but persistant association with subsequent functional psychosis, usually described as schizophrenia (Arnold & Stepan, 1952). If the idiopathic form of this syndrome is associated with an increased risk of future schizophreniform psychosis, and the iatrogenic form with exposure to dopamine-blocking drugs, then the pre-neuroleptic accounts of lethal catatonia may represent important historical evidence in support of the dopaminergic theory of schizophrenia. This is an intriguing possibility and it deserves further study. However, it will first be necessary for us to