CORRESPONDENCE

Depressive Disorder in Primary Care

SIR: Blacker & Clare (*Journal*, June 1987, **150**, 737–751) state that "only a proportion, probably less than 10%, of depressed patients in primary care are referred to psychiatric services".

A large proportion of the psychiatric literature is devoted to research into biological aspects of depressive illness and to clinical trials of antidepressants. Fifteen to twenty years ago, patients with moderate to severe depression were often directly referred to a psychiatrist before antidepressant treatment had been prescribed. During that time, and under those conditions, much pioneering work was carried out into the biochemistry of depressive illness. The situation today is somewhat different: patients are often referred to specialist research units, often within the university setting, only after a general practitioner has failed to obtain an adequate response with one or more antidepressants for periods of one to two months. Since Katona et al (1986) have pointed out that previous antidepressant treatment can affect biological variates for up to three weeks after withdrawal from the drug, researchers are investigating not the biological characteristics of depressive illness but the biological legacy of previous antidepressant treatments. In addition, they are also looking at a highly selected group of patients who are often half-way through their self-limiting episode.

The majority, if not all, of new antidepressant drug trials are carried out under the supervision of a psychiatrist in either an in or out-patient setting. Conclusions are therefore drawn from a population of patients who may have psychotic features or suicidal ideation and, as described above, are by definition often relatively treatment-resistant. While this traditional testing ground provides a stringent test of a new antidepressant, I feel that putative antidepressants should be tested in the general practice arena where one finds the full spectrum of depressive disorders.

I concede that such trials would be difficult to carry out. Indeed, my own preliminary survey has indicated a marked disinclination of general practitioners to test new drugs. The possibility of destroying the doctor/family relationship at the first sign of an adverse reaction was the most common reason.

It should be emphasised, as Blacker & Clare have done, that the situation may be somewhat different in the USA where one may bypass the general practitioner and contact a mental health professional directly. For this reason, biological and clinical trial data obtained in the USA in the last ten years or so may be more relevant than that obtained in the UK. Perhaps closer collaboration between psychiatrists and general practitioners and their respective Royal Colleges could once again ensure that pioneering research is carried out in the UK.

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Reference

 KATONA, C. L. E., THEODOROU, A. E., DAVIES, S. L., YAMAGUCHI, Y., TUNNICLIFFE, C. A., HALE, A. S., HORTON, R. W., KELLY, J. S.
& PAYKEL, E. S. (1986) Platelet binding and neuroendocrine responses in depression. In *The Biology of Depression* (ed. J. F. W. Deakin). London: Gaskell.

Khat-induced Paranoid Psychosis

SIR: As my article of 1945 was perhaps the first on the subject of khat-induced psychosis (Carothers, 1945), and since the course of the illness in the two cases there described differed markedly in one respect from that described by Dhadphale & Arap Mengech (*Journal*, June 1987, **150**, 876), I would like to add a comment to this discussion. The latter writers saw no relapses in any of their 15 patients, whereas one of my patients suffered two relapses requiring readmission within a year, and the other had three such relapses within 13 months. In explanation of this discrepancy, I can only surmise that by the time of the later article some supervision after discharge was maintained in Kenya: a supervision which was not available there in 1945.

However this may be, it seemed to me then (and does so still) that, since the great majority of khat chewers suffer no obvious ill-effects, these florid psychoses occur in people who are mentally vulnerable and who do require some organised support when discharged from hospital.

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Reference

CAROTHERS, J. C. (1945) Miraa as a cause of insanity. East African Medical Journal, January, 4-6.

HIV and Informed Consent

SIR: I read Davies's letter (*Journal*, June 1987, **150**, 881–882) with interest. As the natural history of human immunodeficiency virus (HIV) infection unfolds, so the neuropsychiatric complications are recognised with increasing frequency. Psychosis may

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