LOCOCK, C. (1857) Discussion of paper by E. H. Sievking. Analysis of fifty two cases of epilepsy observed by the author. *Lancet, i,* 527.
MILLER, H. CRICHTON (1920) *Functional Nerve Disease.* London: Henry Frowde and Hodder and Stoughton.
MITCHELL, S. WEIR (1905) Some personal observations on the Civil War. *Transactions of the College of Physicians, Phila,* **27,** 88–94.

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Lithium augmentation in antidepressant-resistant patients

SIR: Austin *et al* (*Journal*, October 1991, **159,** 510–514) recently presented their meta-analysis of five placebo-controlled trials of lithium augmentation in depressed patients "resistant to a standard trial of an antidepressant". They concluded that lithium augmentation had significant efficacy in these patients. However, as the authors acknowledged, while the statistical procedure used to arrive at their conclusions may be 'elegant', its utility is limited by the quality of data being analysed.

Two issues deserve further discussion. The first relates to the duration of antidepressant medication before a patient can be considered treatment-refractory. The studies reviewed used antidepressants for a minimum of three weeks before adding lithium. However, there is evidence (Quitkin *et al*, 1984; Georgotas *et al*, 1986) that up to 25% of patients treated with an adequate dose of antidepressant medication will not respond until weeks four to six of treatment. Given that many of the patients used in the meta-analysis were prescribed lithium after only three weeks, one has to question whether clinical improvement was attributable to lithium or was, in fact, a delayed response to the primary antidepressant. Dr Austin *et al* state that "it is doubtful that many clinicians would in practice wait more than four weeks before changing to another treatment if no response is seen". However, if the evidence suggests that six weeks is required for an adequate trial of an antidepressant, then it is incumbent on the clinician to persist with that treatment rather than prematurely adding another medication with its own risks and side-effects.

The second issue pertains to the criteria used to define response to lithium augmentation. Three of the five studies used a $\geqslant$40–50% reduction in Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) score and a fourth study used a $\geqslant$2 point decrease on a 15-point nurses rating scale. Given that the mean HRSD entry scores for those studies ranged from 20–34, many patients fulfilling criteria for response could still have had scores consistent with mild to moderate levels of depression. Therefore, in these studies, response did not necessarily equate with remission of the depressive illness.

Given these considerations, we must await further studies to determine the value of lithium augmentation in the management of patients meeting current criteria for refractory depression (Guscott & Grof, 1991). At present, there are insufficient data to support the optimistic conclusions of Dr Austin and colleagues.

GEORGOTAS, A., MCCUE, R., HAPWORTH, W., *et al* (1986) Comparative efficacy and safety of MAOIs versus TCAs in treating depression in the elderly. *Biological Psychiatry,* **21,** 1155–1166.
GUSCOTT, R. & GROF, P. (1991) The clinical meaning of refractory depression: a review for the clinician. *American Journal of Psychiatry,* **148,** 695–704.
HAMILTON, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry,* **23,** 56–62.
QUITKIN, F. M., RABKIN, J. G., ROSS, D., *et al* (1984) Duration of antidepressant drug treatment. What is an adequate trial? *Archives of General Psychiatry,* **41,** 238–245.

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Schizophrenia among Afro-Caribbeans

SIR: In discussing the higher risk of diagnosed schizophrenia among the British-born African-Caribbean community, Wessely *et al* (*Journal*, December 1991, **159,** 795–801) state that the increase is not due to brief reactive psychosis as has apparently been suggested. As far as we know, no one has applied this argument to British-born patients of African–Caribbean origin. It was originally suggested to explain high rates of schizophrenia among migrants from the rural West Indies who were not found to have Schneider's first rank symptoms of schizophrenia (CATEGO S+) on project diagnosis (Littlewood & Lipsedge, 1981), and indeed the notion of *bouffée délirante* has been most typically developed in studies in peasant communities. Nevertheless, their use of the RDC minimum period of illness for schizophrenia (two weeks) would not include any patients satisfying our Jasperian criteria of 'reactivity' (*ibid*).

As they point out, our current interpretations involve a complex interplay between the biological and the political: simple 'misdiagnosis', with its implications for better education of psychiatrists, is

unlikely to be the single explanation (Littlewood, 1992).

LITTLEWOOD, R. (1992) Psychiatric diagnosis and racial bias: empirical and interpretative approaches. *Social Science and Medicine*, **34**, 141–149.

— & LIPSEDGE, M. (1981) Acute psychotic reactions in Caribbean-born patients. *Psychological Medicine*, *II*, 308–318.

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SIR: The debate concerning the excess risk of schizophrenia among the Afro-Caribbean population of Great Britain has generated considerable research interest. The recent papers by Eagles (*Journal*, December 1991, **159**, 783–789), and by Wessely *et al* (*Journal*, December 1991, **159**, 795–801), were typical in both supporting the notion of an increased risk, and in stressing biological factors. However, the report from Jamaica, by Hickling (*Journal*, December 1991, **159**, 817–821) showed an admission rate for schizophrenia five to six times lower than the rate reported for Afro-Caribbeans in the UK. Thus, despite accumulating data, we seem to be no nearer an explanation.

One problem seems to be that discussion of this research remains concentrated around the issues of a theoretical viral aetiology, neurodevelopmental abnormalities, and obstetric complications. Thus, O'Callaghan *et al* (1991) found an increased risk of schizophrenia for those in their fifth month of foetal development during the 1957 influenza epidemic. The authors suggested that this supported the evidence for "aberrant foetal brain development in the pathogenesis of schizophrenia". Yet their own figures showed no overall increase in subsequent schizophrenia for the year in question, compared with the four control years, which hardly accords with our expectations of a causal agent. Many other reviews of schizophrenia, while comprehensive in analysis, do tend to focus narrowly on biological data, ignoring the more difficult problems of social research. In particular, it seems that not enough attention has been paid to the pathoplastic, as opposed to the pathogenic, effects of brain abnormalities.

Yet a key area of Afro-Caribbean studies is the problem of quantifying the true presence of schizophrenia. As Glover (1989) has pointed out, if symptoms are more florid in this group, "then a higher proportion of Caribbean schizophrenics would reach the level required for diagnosis by standardized instruments", and fewer would have "subclinical illness". Yet there have been no *community* surveys of schizophrenia and ethnicity in this country; all the reports rely on the data of admission and/or presentation to hospital. Such community studies are, of course, notoriously difficult to carry out, but vital in an area of such social controversy and aetiological importance. In addition, we have an extremely uncertain census base from 1981, and the increased numbers of black males in prisons, and under Mental Health Act Sections, seem to point to a behavioural prominence that may well be distorting the admission figures. Given the community care policies of today, and the increased pressure on acute psychiatry beds (especially in the inner-city areas with high proportions of Afro-Caribbean residents), is it not likely that such individuals will have more obvious admission needs? A further vital issue is the notion of cross-cultural validity in terms of the instruments used. Dr Wessely *et al* admitted in their study that these were not established, and agreed that other biases in their control group might have been present.

As a clinician working among a significant Afro-Caribbean population, it is quite clear to me that there is an excess of admissions, both first and subsequent, for schizophrenia in this group. However, it is also clear that they seem to have much less family support, to be more often technically homeless, and in a number of instances seem to have preferentially ended up in the British health care system. This is despite their families residing in North America, Africa or the Caribbean. Seven out of twelve current African or Caribbean in-patients (on our 20-bed acute ward) fit this concept, and five have a schizophrenic illness. Several individuals have, literally, been sent to Britain for care, by relatives aware of the limited resources available for psychotic individuals in the Third World, or unable to cope with the expenses liable in America. The patterns of migration in the post-war period have also been much more complex than the simple model of West Indians coming to Britain, and I would suggest that there may well have been a secondary selective process.

Overall, the Afro-Caribbean debate seems to be worryingly unresolved. This is due to an underemphasis on the less medical, less 'hard', and more difficult to fund areas of research. Yet if we do not balance out such research needs we are at risk of creating a top-heavy viral/genetic theory that has no foundation in social facts. Whatever the quality of such biological research, and it seems to me in general to be first class, this will nevertheless create a distrust of psychiatrists among ethnic groups