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

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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

requiring our closest concern, and among other professional researchers (e.g. sociologists, anthropologists) with whom we have traditionally cooperated. Careful social and community research must now be the real priority in the issue of schizophrenia and Afro-Caribbeans.

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SIR: We read with interest the papers by Castle *et al* (*Journal*, December 1991, **159**, 790–794) and Wessely *et al* (*Journal*, December 1991, **159**, 795–801) and note their finding of higher rates of operationally defined schizophrenia among Afro-Caribbeans in the UK. In discussing possible explanations for these findings, Dr Wessely *et al* suggested that the prevalence of schizophrenia in this group could be increased either directly or indirectly by forms of social adversity such as unemployment, inadequate housing, low social class, the experience of racism and other forms of social deprivation.

The puzzling thing about these hypotheses to account for the higher rates of major mental illness is that the same factors are associated with an increased risk of non-psychotic disorders such as depression, anxiety, and functional somatic symptoms (Goldberg & Huxley, 1992). Yet the British evidence is that Afro-Caribbean patients are less likely to receive this diagnosis from general practitioners (GPs) than white British attenders. Gillam *et al* (1989), reviewing over 67 000 GP consultations in Brent, North London, found that GPs were less likely to diagnose psychosocial disorders in Afro-Caribbean attenders than in white British attenders. Similarly Johnson *et al* (1986) found from a community survey in the West Midlands that Afro-Caribbean respondents were much less likely than white British respondents to report having attended their GP with a psychological problem.

There are several possible interpretations of these apparently contradictory findings concerning schizophrenia and non-psychotic disorders. Firstly, the excess of schizophrenia might be due to biological not social factors, a consideration raised by Dr Wessely *et al*. Secondly, the excess of schizophrenia could be due to misdiagnosis, although the weight of evidence against this is increasing. Thirdly, the theories about the social precursors of schizophrenia might be correct yet operate differently with regard to non-psychotic disorders resulting in differing rates in the Afro-Caribbean population. Fourthly, Afro-Caribbean patients with non-psychotic disorders might not attend their GPs or the GP might not recognise these disorders. This could be due either to the mode of symptom presentation, or cultural differences in the nature of non-psychotic disorders (Helman, 1990). Lastly, it could be that Afro-Caribbean patients do not frame their distress in psychological terms because of the tremendous stigma attached to mental illness and the black community's discriminatory experiences of the mental health care system (Rack, 1982). Another aspect of discrimination which might impact upon the presentation and recognition of non-psychotic disorders is differential access to services and the unacceptability of having the stresses of living with discrimination redefined as neurotic illness (Rack, 1982).

Compared to the volume of work conducted into schizophrenia and the Afro-Caribbean community, relatively little work has been carried out in the UK on non-psychotic disorders such as anxiety, depression and somatic symptoms. If theories about the direct and indirect roles of social adversity in schizophrenia among Afro-Caribbeans in the UK are correct, then some explanation is required as to why it has been suggested that rates of non-psychotic disorders in primary care settings are lower, the opposite of what might be expected from work on vulnerability factors to non-psychotic disorders among the general population.

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SIR: Dr Eagles focuses interest on biological factors as possible causes of the excess of schizophrenia