individual merely to membership of a category – which I would regard as 'racism'.

It is then claimed that 'a public health approach' to discrimination is likely to be more effective in decreasing rates of mental illness than intervention at a health service level. But of what would such an approach consist, and how long would it be before its effects could be seen in a reduced prevalence of disorder? Regrettably, the causes of most mental disorders remain unknown and although large resources have been spent throughout the world on 'primary prevention', any positive results have been modest in the extreme.

If, as Sashidharan (1993) has argued, research should focus on 'power disparities in a predominantly racist society', it would be very likely to show that the majority of such differences have nothing to do with racism, as Chakraborty and McKenzie partly admit. Yet, if representatives of the majority were to propose that the emphasis should be moved away from the White–non-White difference, this would be used to prove how 'racist' they really were. It is a double-blind situation.

The authors call for acknowledgement of institutional racism in psychiatry, but the work they have quoted in support of this view consists only of allegations and not of evidence. Unfortunately, in the current climate of political correctness, there is a lack of serious scientific debate on the subject. Their call for longitudinal research into a possible link between racial discrimination and mental illness should certainly be supported.

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Authors' reply: Our paper was the first in the *British Journal of Psychiatry* that attempted to answer a simple question that many UK psychiatrists have been asked by their ethnic minority patients – does racial discrimination cause mental illness? (Chakraborty & McKenzie, 2002).

Patients know that the rates of psychosis, for instance in Black Caribbeans in the Caribbean, is the same as for White British people in the UK, but that the rates of psychosis in Black Caribbeans in the UK is markedly higher. There has been no plausible biological hypothesis to explain this and all the evidence, including the genetic evidence, points to a social aetiology (Sharpley *et al*, 2001).

With specific reference to Dr Eagle's comments: although there is no evidence whatsoever of a biological cause or of increased vulnerability in ethnic minority groups, there is cross-sectional evidence of an association between experiencing racial discrimination and both psychotic and non-psychotic illness in ethnic minority groups in the UK. There is also longitudinal evidence of a link between experiencing discrimination and the development of psychotic symptoms in The Netherlands and these associations cannot be explained by other known risk factors (Chakraborty & McKenzie, 2002).

We do not invoke charges of political incorrectness. We invoke scientific logic and scientific equipoise. Given the available information and the resurgence of social causation theories of psychosis, it is difficult not to come to the conclusion that racial discrimination is a practical area of investigation.

Dr Eagles is wrong in his assumptions about the paper by Boydell *et al* (2001). Movement within the London wards that were surveyed was very limited and could not explain the results.

Professor Freeman is correct to cite the high rates of depression in some developing countries and we would support his call for more research in this area. He may not be aware of the methodological flaws in the work of the Manchester group which make their findings very difficult to interpret (McKenzie, 1999).

Qualitative and quantitative research formats are complementary and offer different types of information. They are both scientific techniques, if used appropriately.

Racism is an experience that depends on context. We do hope that we have misunderstood Professor Freeman's suggestion which seems to be to try to establish some sort of league table of distress across different times or continents – this would be a bizarre idea. Phenotypic differences that we mention in our paper are not limited to skin colour and, of course, we accept that discrimination against many different White groups has been rife in the UK. We note the high rates of mental illness in some of these groups, such as the Irish.

Racism remains a major cause of the perpetuation of socio-economic differences between minority groups and ethnic majority groups in the UK and all of those working in the area, including governments, agree on this.

Most ethnic minorities in the UK are not first-generation immigrants, they were born in the UK. The majority of first-generation immigrants were asked to come to the UK to work during post-war labour shortages. Only a minority were fleeing persecution. Immigrants to the UK have always put more into the country than they have taken out. Professor Freeman's comments on the stress hypothesis are thus misinformed.

We agree with Professor Freeman that the ethnic density findings need much more detailed work to help make sense of the situation. In this regard, we point to the fact that qualitative methods are of particular use in investigating complex social systems.

We understand Professor Freeman's call for individualised care. However, we would feel better able to support him if the call was actually for individual choice of different models of care. There are some people to whom race, ethnicity and culture are very important; ignoring this or taking a 'colour-blind' approach offers them a poor service.

Professor Freeman states that there is a lack of serious debate on issues of racism in psychiatry and institutional racism. It is difficult to sustain such an argument. Although these issues rarely reach mainstream journals, there has been debate on this subject for decades in the UK, mainland Europe and the USA and there is a rich literature on these subjects (for a UK perspective see Bhui, 2002). Our modest editorial was an attempt to push the work forward and to link the literature to an outline service response.

No one can deny the need for more research but one must always balance the need for research with the problems with delay and the likely positive outcomes. Public health approaches have wide-based outcomes which must always be kept in mind when analysing their impacts. For instance, a public health policy aimed at reducing

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racist attacks, reducing institutional racism, improving schooling, supporting the family, decreasing the number of Black children in care and offering people skills to deal appropriately with discrimination, could have such a positive impact on society that it would be a reasonable initiative for psychiatrists to support, even if there was only a modest direct decrease in the rates of mental illness in ethnic minority groups. However, we are aware that there is a danger that protracted, scientific attention given to empirical questions might overshadow consideration of the more important error which is the implication that socio-moral tenets can be appropriately derived from science. Concepts such as liberty, justice and freedom from discrimination are neither determined nor justified by scientific results but flow from constitutional and moral principles. Science has a role in social policy but, in this regard, it is less in defining rights and more in developing methods for achieving rights. Cost–benefit analysis of public health measures based on rights is a moral discourse. Given the difficulty in raising research funds for research into racism, waiting for the evidence, or opposing such initiatives because of the lack of 'evidence', is not a zero-sum game. We have no initiative to decrease the rates of mental illness in ethnic minority groups in the UK. Is it not time that we did?

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Neuroticism and depression

Farmer *et al* (2002) draw conclusions that we believe are not supported by the results of their study. The study compared probands with depression and their siblings, with healthy probands with no history of depression and their siblings. Between two-thirds and three-quarters of the participants were women. The groups did not differ in age that varied from 36.2 to 39.1 years.

The absence of a difference in mean scores for neuroticism for the never-depressed siblings of both the healthy probands and those with depression was interpreted to suggest that this scale does not measure a genetically influenced trait for depression. This finding, however, can be interpreted otherwise. As the siblings of the probands with depression were in their mid-thirties and had not, as yet, experienced a depression, it is reasonable to assume that they have passed the age of risk for a first episode (Burke *et al*, 1990) and therefore may not have inherited a vulnerability for the disorder. The finding that the siblings of the probands with depression obtained scores for the trait of neuroticism similar to those obtained by siblings of the healthy probands, who presumably are not genetically vulnerable to depression, could be interpreted to suggest that neuroticism is necessary for depression. As long as there is no way to determine whether an individual carries the genes associated with depression, a cross-sectional study of adults cannot ascertain whether neuroticism reflects a part of the genetic vulnerability for depression. A prospective, longitudinal investigation in which the trait, or an age-appropriate proxy for the trait, is measured before the onset of symptoms could untangle the relationship between neuroticism and depression. Studies comparing monozygotic and dizygotic twins could also address the issue, as did Kendler and colleagues (Kendler *et al*, 1993) who reported that the genetic liability for major depression largely overlapped with that for neuroticism. The finding that neuroticism scores are positively correlated with symptoms of depression and with severe life events does not address the aetiological question.

It is important to determine whether the trait of neuroticism reflects a part of the genetic vulnerability for depression. It is also important to identify the factors that exacerbate the inherited vulnerability and lead to depression, in order to design prevention programmes for children at risk for depressive disorders. Among parents with a major affective disorder, neuroticism may have more influence on the development of their offspring than does the severity of their disorder. In a prospective study of the children of parents with bipolar disorder, we have found that neuroticism is associated with high levels of negative life events, low levels of psychosocial functioning and with poor parenting, which in turn are associated with the children's level of psychosocial functioning and symptoms (Hodgins *et al*, 2002; further details available from the author upon request). By contrast, none of the indices of the severity of the parents' disorder is associated with psychosocial functioning or symptoms of the offspring. These findings suggest that neuroticism, rather than the disorder, influences parental behaviours that impact on the mental health of the offspring.

In light of the above considerations, we believe that Farmer *et al*'s 'Clinical implication' that 'Neuroticism reflects subclinical or residual symptoms of depression' is misleading. Among adult patients, symptoms of depression do appear to be associated with scores for neuroticism, as has been reported previously (Sauer *et al*, 1997). Whether or not the trait of neuroticism also represents a risk factor for depression, however, is not known. The cross-sectional study reported by Farmer *et al* (2002) does not address this important question. Available data suggest that the trait of neuroticism may play a critical role in the development of depressive disorders, conferring an inherited vulnerability and leading to parental behaviours associated with impaired functioning among the offspring.

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