Correspondence

EDITED BY KHALIDA ISMAIL

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Racial discrimination and mental illness

Chakraborty & McKenzie (2002) ask: ‘Does racial discrimination cause mental illness?’ In raising criticisms of their paper, one might risk allegations of political incorrectness, but hopefully readers will feel that science is a more important consideration.

The question that they pose is, to my mind, a simplistic one which is likely to give rise to a simplistic answer. To ask ‘does smoking cause physical illness?’ would give rise to the answer that it causes some physical illnesses and not others. The same relationship is likely between racial discrimination and mental illness.

That racial discrimination, like other aspects of social adversity, gives rise to an increased risk of depression is something that all psychiatrists almost certainly find entirely plausible. That it might cause schizophrenia, on the other hand, is surely much more contentious. Psychosocial stressors can undoubtedly precipitate relapse, but I know of no good evidence that such stressors can cause schizophrenia. Ethnic differences exist with regard to the epidemiology of multiple sclerosis (e.g. Warren et al, 1996) but it would be regarded as absurd to invoke racial discrimination as a causative (or indeed a protective) factor. Is it politically incorrect to suggest that different ethnic groups may be biologically predisposed to different levels of risk with regard to developing illnesses which have predominantly biological aetologies?

Finally, in quoting the work of Boydell et al (2001), the authors may be confusing cause and effect. The fact that the incidence of schizophrenia is increased among ethnic minority groups living in London wards which have a lower percentage of ethnic minority inhabitants, may indicate that schizophrenia can give rise to people moving away from their families and their communities of origin.


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Drs Chakraborty and McKenzie (2002) seek to answer the question, ‘Does racial discrimination cause mental illness?’, but in doing so they raise further concerns. They refer to high community prevalence rates of depression in the UK, compared with the countries of origin of minority groups, but very high rates have been reported in indigenous populations from Uganda, the Himalayas and the Indian subcontinent. Further reliable studies would be desirable, but this is not a fashionable field for research. In Manchester, Shaw et al (1999) found no difference in rates of common mental disorders between the White and African–Caribbean populations.

When the authors suggest that social and service-related risk factors ‘may be better studied using qualitative’ rather than ‘quantitative epidemiological approaches’, this should provoke serious disquiet. If attempts at scientific measurement are to be discarded, what will be put in their place? The accusation that, for example, ‘this work is racist’ is qualitative enough, but how can its truth be demonstrated or compared with others?

The statement that racism is ‘widespread in the UK’ is not helpful in itself. Is it worse than in Rwanda or Sri Lanka? And does ‘phenotypic difference’ refer only to skin colour? The all-White Jewish population of Europe in the 1940s was not notably exempt from racism – a fact rarely mentioned in this literature. If ‘some believe’ that minor hostile incidents have a greater impact on health than racist attacks, they have not demonstrated this to be so. Similarly, ‘paranoia’ cannot, by definition, represent a healthy coping strategy, since it is separated from reality.

It is argued that ‘racism produces and perpetuates socio-economic difference’. This may be true to some extent, but most socio-economic difference is unrelated to race. Pre-World War 2, Britain contained only minuscule numbers of non-Whites, yet was rigidly affected by social difference and advantage. Race merely adds an additional factor.

When the question is examined in terms of ‘stress’, it is usually assumed that this only applies to the host society. Yet the reason people migrate is primarily to escape the stress of their original home. This may take such forms as desperate poverty, corrupt government, climatic disasters, civil strife, absence of essential services, etc. Is it more stressful to live in a ‘racist’ welfare state or to die in the street of a monoracial African or Asian country?

Two authors are quoted who reported that African and Caribbean patients with psychosis in Britain were more likely to attribute their problems to racism, but in the absence of any comment, it is not clear what we are to make of this.

The relationship between the proportion of ethnic minorities in a local population and their prevalence of mental disorder is said to reflect ‘complex interactions between exposure to discrimination, social support, socio-economic factors and social capital’. In other words, just about everything except the kitchen sink. How can any meaningful relationship between factors possibly be extracted from this melange?

A relationship is then suggested between community-level racist attitudes and mental illness in American minority groups, but the only evidence cited is for all-cause mortality, which is totally different and largely unrelated.

Fernando (1991) is quoted as arguing that the European emphasis on an individualised pathology renders psychiatry a racist institution. But in fact, the opposite is more likely to be true. Considering each patient more as an individual respects his/her unique situation, whereas emphasis on ‘race and culture’ tends to reduce the
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