Racial discrimination and mental illness

Chakraborty & McKenzie (2002) ask: ‘Does racial discrimination cause mental illness?’ In raising criticisms of their paper, one might raise 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 aetiology?

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.

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
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.


Farmer et al (2002) examined whether self-reports of neuroticism and extraversion represent enduring vulnerabilities to depression. While their findings make a valuable contribution, their conclusions regarding neuroticism warrant additional consideration. They found variables reflecting past and current depression were significant predictors of neuroticism and interpreted this as inconsistent with their hypothesis that neuroticism would ‘exhibit trait-like qualities’ and would not be ‘substantially influenced by alteration in mood-state’ (p. 118). Farmer et al’s findings did not directly address this hypothesis because they did not use a longitudinal design needed to observe fluctuations in mood-state. None the less, several longitudinal studies indicate that neuroticism is affective-state dependent. For example, Hirschfeld et al (1983) found that patients in remission from depression reported lower levels of neuroticism than they originally reported when depressed. Findings of this nature have been used to argue that neuroticism and related traits are only contaminants of depression. More recently, investigators (e.g. Santor et al, 1997) studying samples of patients have noted that absolute changes in depression-related traits are associated with changes in mood (i.e. affective-state dependent), but that there is also a consistency in the rank of patients with regard to their scores on these measures (i.e. relative stability). Findings such as these have been interpreted as indicating that depression-related personality traits have both state-like and trait-like properties.

Given the large association between neuroticism and depression, Farmer et al suggested that neuroticism may be largely ‘a proxy measure for present or past depression’ (p. 121) and questioned whether neuroticism reflects a vulnerability for depression. Neuroticism refers to a tendency to experience negative affect, so this high degree of overlap is not surprising. More importantly, longitudinal studies (Hirschfeld et al, 1989; Krueger et al, 1996) have found that high premorbid neuroticism is positively associated with the development of depression.

In summary, Farmer et al’s conclusion that neuroticism does not measure a vulnerability to depression and primarily reflects symptoms of depression is not warranted. Self-reports of neuroticism prospectively predict depression. Longitudinal studies support Farmer et al’s conclusion that neuroticism is strongly associated with a person’s current affective state. However, such studies also suggest that neuroticism is likely to have trait-like properties in addition to the state-like properties noted by Farmer et al.


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Authors’ reply: We are pleased to respond to the comments of Drs Hodgins & Ellenbogen and Dr McWilliams and are grateful for their interest in our work.

Hodgins and Ellenbogen suggest that an absence of a difference in mean scores for neuroticism (N) for never-depressed siblings of probands with depression and never-depressed siblings of healthy controls can be interpreted as showing that the siblings of probands with depression have not inherited the vulnerability for the disorders. However, this is missing the point. We hypothesised that scores represent a genetically influenced trait that underpins the risk of developing depression in the presence of precipitating factors such as adverse life events. If N were such a trait, then it would be expected that all first-degree relatives of probands with depression who share an average 50% of their genes with their relative with depression, would have higher mean scores than subjects without such a genetic relationship to a proband with depression, irrespective of affective status. Our failure to detect a difference for N scores is not due to lack of power since we have shown differences between the relatives of probands with depression and controls for other personality measures (Farmer et al, 2003) such as the Harm Avoidance Scale of the Temperament and Character Inventory (Cloninger et al, 1993).

Both Hodgins & Ellenbogen and McWilliams rightly point out that longitudinal studies may help to disentangle the relationship between N and depression. However, even longitudinal studies can fail to answer the issue of what came first, the chicken of neuroticism or the egg of depression. For example, it is now well recognised that depressive symptoms occur in children and adolescents as well as in adults. Consequently, in order to demonstrate that N scores represent an underlying vulnerability to depression and are not merely a proxy measure of depressive symptoms, it is necessary to show that elevated N scores occur in the absence of significant depressive symptoms at the first point of measurement. To our knowledge, no longitudinal study to date has shown this.

Our study is in keeping with a growing literature showing that N has considerable state-dependent as well as trait-like properties. Despite this, there remains a cherished belief that the measure does indeed represent a trait underlying the vulnerability to depression. We have not demonstrated such properties for the scale in our Cardiff study.

Declaration of interest

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Diffuse muscle pain with quetiapine

We report the case of a 28-year-old female out-patient with bipolar disorder, whose symptomatology was well-controlled with lithium carbonate 1200 mg (orally) (0.9 mEq/l plasma levels) and risperidone 1–2 mg (orally) daily. The patient had been treated for several years in our department and the course of her illness was well-known; it showed that only lithium was both effective and well-tolerated (topiramate was not effective and carbamazepine caused a rash) and only in coadministration with low doses of risperidone.

However, the use of risperidone caused a large increase in prolactin levels (above 2000 µU/l, with normal values below 500 µU/l) and amenorrhoea (the rest of the hormonal investigation and brain magnetic resonance imaging were normal). The patient was put on 5 mg olanzapine (orally), but she did not tolerate this agent because it made her feel ‘confused’ and ‘tired’. She was then put on 200 mg quetiapine (orally). Within 24 h the patient manifested diffuse muscle pains and headache. She reported that her legs were stiff and she had pain in her knee joints. Neurological examination was normal, as were blood and biochemical tests including creatine phosphokinase. Vital signs were normal. No extrapyramidal signs or symptoms (especially akathisia) were present. The pain persisted for 5 more days and the patient demanded that quetiapine be discontinued. The pain disappeared within the first 48 h of shifting back to risperidone, which was according to the wishes of the patient. Six months passed and the patient is still free from symptoms.

To our knowledge, this is the first report of this kind of adverse effect related to quetiapine. Various other antipsychotics, including haloperidol and olanzapine, are reported to cause muscle pain and rigidity because of rhabdomyolysis, but the current case had no laboratory or clinical findings related to rhabdomyolysis.

Adjunctive fluvoxamine with clozapine

We read with interest the article by Williams et al (2002). The authors summarise treatment options for patients resistant to clozapine monotherapy. However, in the section on combining antidepressants with clozapine, several issues deserve more attention. The authors disagree with utilisation of adjunctive antidepressants to reduce the cost of clozapine treatment. In our recent study (Lu et al, 2000), addition of 50 mg/day fluvoxamine to low-dose (100 mg/day) clozapine could raise the mean plasma clozapine level to over 400 ng/ml to achieve suitable therapeutic ranges. Therefore, concomitant fluvoxamine can reduce clozapine doses and, consequently, costs (Armstrong & Cozza, 2001).

Interestingly, this pharmacokinetic interaction is more pronounced in patients with high cytochrome P450 1A2 activity and at low clozapine plasma concentrations (Olesen & Linnet, 2000). This phenomenon could therefore be used to narrow down the wide interindividual variation in blood clozapine concentrations. Several open trials also demonstrated that coadministration of fluvoxamine could augment clozapine efficacy and curtail plasma norclozapine:clozapine ratios (Wetzelm et al, 1998; Lu et al, 2000). Norclozapine has been suggested to be more toxic than its parent compound. Although addition of fluvoxamine to low-dose clozapine was well-tolerated in our pilot study (Lu et al, 2000), further studies are warranted to substantiate its safety and efficacy.

Olanzapine-induced tardive dyskinesia

Tardive dyskinesia is a serious and common motor side-effect of treatment with traditional neuroleptics, with an unknown pathophysiological basis. It affects 20–30% of patients on long-term neuroleptic therapy, with elderly patients being at higher risk (American Psychiatric Association, 1994).

Olanzapine is an atypical antipsychotic agent with a reported lack of propensity to cause tardive dyskinesia (Beasley et al, 1999). Recently, it has been suggested that olanzapine can improve tardive dyskinesia in some patients (Littrell et al, 1998; Jaffe & Simpson, 1999). Other authors, however, have shown that the prolonged use of olanzapine can instead be associated with tardive dyskinesia/dystonia (Ananth & Kenan, 1999; Dunayevich & Strakowski, 1999). Here we report the case of a patient who experienced tardive dyskinesia after only a few months of treatment with olanzapine.

A 62-year-old housewife with an unremarkable past medical history, sought out-patient treatment in June 2000 for anxiety, insomnia, difficulty thinking and concentrating, and frequent episodes of aggressive behaviour. She was evaluated by neurologists, and was submitted to routine biochemical investigations (unremarkable), a computerised tomography scan (normal), and the Mini-Mental State Examination (24/30). Olanzapine (10 mg/ day) was started and this was the sole medication continued thereafter. The patient soon experienced a subjective improvement. Three to four months later she noticed slight involuntary movements of the tongue and jaw. Despite these symptoms, she continued taking olanzapine until it was eventually stopped 1.5 years later (December 2001).

She was admitted to our hospital in March 2002. On examination, she displayed signs of tardive dyskinesia.

Declaration of interest

None. Funding was received from the National Science Council and the National Health Research Institutes, Taiwan.

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A 62-year-old housewife with an unremarkable past medical history, sought out-patient treatment in June 2000 for anxiety, insomnia, difficulty thinking and concentrating, and frequent episodes of aggressive behaviour. She was evaluated by neurologists, and was submitted to routine biochemical investigations (unremarkable), a computerised tomography scan (normal), and the Mini-Mental State Examination (24/30). Olanzapine (10 mg/day) was started and this was the sole medication continued thereafter. The patient soon experienced a subjective improvement. Three to four months later she noticed slight involuntary movements of the tongue and jaw. Despite these symptoms, she continued taking olanzapine until it was eventually stopped 1.5 years later (December 2001).

She was admitted to our hospital in March 2002. On examination, she displayed signs of tardive dyskinesia.
movements of the tongue and jaw, grinning, and mild choreic movements in the upper limbs. Extensive biochemical, neuro-psychological and imaging work-up was negative. A diagnosis of drug-induced tardive dyskinesia was thus made, other causes of dyskinesia excluded and therapy with vitamin E, lorazepam and tiapride initiated.

In this case, the tardive dyskinesia was most likely a result of olanzapine administration. The age of the patient may have favoured the early appearance of involuntary movements after initiation of the therapy, even though olanzapine has been claimed to carry a low risk for tardive dyskinesia and other extrapyramidal symptoms (Beasley et al, 1999).

As olanzapine is increasingly being used in elderly subjects for behavioural disturbances and/or insomnia in the absence of psychosis, our report underlines the need for a careful assessment for tardive dyskinesia and other movement disorders in patients (and in particular elderly patients) taking this atypical neuroleptic.

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One hundred years ago

The unconscious mind.
To the Editors of The Lancet

Sirs,—In a short account of Sir F. Treves’s address at Liverpool I observe that the two principal points mentioned both refer to a subject that is coming more to the front every day. I allude to the power of the mind over the body. He speaks with the greatest appreciation of the value of symptoms, pointing out that in diseases generally (specially naming appendicitis) they are nature’s effort to cure the disease. In short, he fully recognises the value of the vis medicatrix naturae, or as “nature” in this connexion is a pure fiction, we may say the unconscious purposive action of the organism or more briefly, and more accurately, “the unconscious mind.” The second point alluded to is that in a hospital patients should not know where the operating theatre is or when they are to be operated on. This is because of the depressing effect the conscious mind, dwelling on these points, has on the body, influencing, indeed, to some extent the operation itself. This address therefore gives two capital illustrations of the effect of the unconscious mind and conscious mind on the body in disease—a subject I am most anxious to see developed scientifically by the profession and no longer left to be exploited by quacks.

I am, Sirs, yours faithfully,
A. T. Schofield, M.D. Brux.
Harley-street, W., Oct 13th, 1902

REFERENCE

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